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2026

CLINICAL VIGNETTES & RESEARCH COMPETITION

ABSTRACT BOOK

Oral Presentations • Clinical Vignettes • Research

November 14, 2026

Complejo Ferial de Puerto Rico

Ponce, Puerto Rico

2026 ACP PUERTO RICO CHAPTER

CLINICAL VIGNETTES & RESEARCH COMPETITION

Oral Presentations Abstracts

ABSTRACT O-01

Vogt-Koyanagi-Harada Syndrome in an Adult Patient: A Case Report

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Program: Ramón Betances Hospital-Mayaguez Medical Center

ABSTRACT

Vogt–Koyanagi–Harada (VKH) syndrome is a rare autoimmune disorder characterized by T-cell-mediated inflammation directed against melanocyte-containing tissues, predominantly involving the eyes, meninges, inner ear, skin, and hair. Ocular manifestations include bilateral granulomatous panuveitis with diffuse choroiditis and exudative retinal detachments, which may result in permanent visual impairment if treatment is delayed. We report a case of a 48-year-old woman with type 2 diabetes, hyperlipidemia, and class II obesity who presented with three weeks of frontal headache, ocular pain, progressive blurring of vision, and photopsia. Ophthalmic evaluation revealed bilateral multifocal exudative retinal detachments, choroidal folds, and disc edema. Fundus fluorescein angiography demonstrated early pinpoint hyperfluorescence with late leakage corresponding to areas of neurosensory detachment and optic disc leakage. Laboratory workup was notable for mildly elevated ESR and CRP; autoimmune serologies and infectious screening, including ANA, ANCA, and syphilis testing, were negative. Neuroimaging was unrevealing. Based on the revised international criteria, the constellation of bilateral granulomatous panuveitis-like posterior segment involvement and serous retinal detachments supported a diagnosis of Vogt–Koyanagi–Harada (VKH) disease, acute (stage II). Initial management with oral prednisone (40 mg daily tapered) produced partial response but relapsed during taper. Escalation to high-dose prednisone (70 mg daily) plus adalimumab resulted in rapid clinical improvement. However due to insurance denial of adalimumab, mycophenolate mofetil was initiated as a steroid-sparing immunosuppressant with good tolerance. The patient has remained symptom-free without recurrence for six months. This case highlights the importance of early recognition of VKH in patients with bilateral exudative detachments, as delayed diagnosis and treatment can result in permanent vision loss. It further highlights the diagnostic value of multimodal imaging in confirming VKH and demonstrates that combined corticosteroid and immunomodulatory strategies and incorporating both biologic and conventional steroid-sparing agents can achieve durable remission and preserve visual function, even when access to first-line biologic therapy is limited by insurance constraints.

ABSTRACT O-02

Diabetic Striatopathy in Severe Diabetic Ketoacidosis: A Rare Neurologic Manifestation of Hyperglycemia With Persistent Hemichorea

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Program: Centro Médico Menonita Cayey Program

ABSTRACT

Diabetic striatopathy is a rare neurologic complication of severe hyperglycemia characterized by chorea or hemiballismus associated with abnormalities of the basal ganglia on neuroimaging. It is classically described in older patients with poorly controlled type 2 diabetes and nonketotic hyperglycemia, while its occurrence in diabetic ketoacidosis is considerably less common. Neurologic manifestations generally improve following correction of the underlying metabolic disturbance; however, clinical and radiographic recovery may not occur simultaneously. We describe a patient with profound diabetic ketoacidosis and striatal abnormalities who developed persistent hemichorea despite successful correction of hyperglycemia and ketoacidosis.A 69-year-old woman with hypothyroidism, dyslipidemia, and schizophrenia, but no prior diagnosis of diabetes mellitus, was transferred from a nursing home after staff noted decreased oral intake followed by a seizure-like episode with gaze deviation. Capillary blood glucose was 578 mg/dL. During transport, she became progressively obtunded and required endotracheal intubation shortly after arrival. Laboratory evaluation revealed profound diabetic ketoacidosis with mixed hyperosmolar hyperglycemic syndrome, serum glucose 984 mg/dL, pH 7.096, bicarbonate <10 mmol/L, anion gap 42.5, large ketones, and HbA1c >14%, indicating substantial antecedent hyperglycemia despite the absence of a prior diabetes diagnosis. Corrected sodium was 153 mmol/L. Concurrent Enterococcus faecalis urinary tract infection, leukocytosis of 14.26 $\times 10^3/\mu L$, and lactate of 2.7 mmol/L suggested an infectious precipitant.Neuroimaging demonstrated interval bilateral basal ganglia abnormalities concerning for a metabolic insult, with diabetic striatopathy considered in the setting of profound hyperglycemia. Following correction of ketoacidosis and improvement in mental status, the patient was extubated and subsequently demonstrated prominent left-sided choreiform movements correlating with contralateral striatal involvement. Although metabolic parameters and glycemic control improved, the involuntary movements persisted beyond correction of the acute hyperglycemic state. They gradually diminished but remained inducible or recurrent during voluntary activity.Diabetic striatopathy represents an uncommon but important neurologic consequence of uncontrolled diabetes and is traditionally associated with nonketotic hyperglycemia. This case demonstrates that characteristic striatal abnormalities and choreiform movements may also accompany profound ketotic metabolic decompensation. Moreover, the neurologic phenotype may become apparent only after recovery from severe encephalopathy and may persist despite normalization of the inciting metabolic disturbance. The temporal dissociation between glycemic correction and neurologic recovery suggests that restoration of metabolic homeostasis does not immediately reverse established striatal dysfunction. Recognition of this clinicoradiologic syndrome is important to distinguish diabetic striatopathy from acute cerebrovascular disease and other causes of new-onset movement disorders.

ABSTRACT O-03

Atraumatic Escherichia coli Septic Arthritis of the Native Shoulder in an Immunosuppressed Patient With Rotator Cuff Arthropathy

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ABSTRACT

INTRODUCTIONSeptic arthritis of the shoulder is rare and most commonly caused by gram-positive organisms. Escherichia coli is an uncommon cause, typically associated with urinary tract infection, uncontrolled diabetes, retroperitoneal abscess, trauma, or recent procedures. Isolated E. coli infection of a native shoulder without these risk factors is rarely reported. We present a case of E. coli septic arthritis in an immunosuppressed patient with massive rotator cuff arthropathy and no identifiable distant infectious source or systemic manifestations.METHODSThis case report was conducted according to institutional policy, which determined that institutional review board approval was not required. The patient provided consent for publication.RESULTSA 70-year-old man presented with three days of atraumatic left shoulder pain. His history included chronic kidney disease, hypertension, remote poliomyelitis requiring long-term crutch-assisted ambulation, and membranous glomerulonephritis treated with mycophenolate mofetil. He denied fever, chills, diaphoresis, or urinary symptoms. Examination revealed severe pain with active and passive motion and a tender, nonerythematous, cystic mass over the superolateral deltoid. Radiographs showed superior humeral head migration and a reduced acromiohumeral interval.Synovial fluid was pink and turbid, with 104,600 WBC/µL, 38,000 RBC/µL, 87.6% polymorphonuclear cells, and no crystals. Culture grew E. coli resistant to ampicillin and ampicillin-sulbactam. Peripheral WBC count was 11.46 × 10^3/µL and C-reactive protein was 6.5. MRI demonstrated a large glenohumeral effusion communicating with the subacromial-subdeltoid bursa, glenohumeral arthritis consistent with rotator cuff arthropathy, and multifocal intramuscular edema without osteomyelitis. Operative irrigation and debridement identified approximately 40 mL of purulent glenohumeral fluid. Intraoperative cultures were negative after four days of antibiotics. The patient received piperacillin-tazobactam for 9 days followed by ceftriaxone for 21 days, completing four weeks of therapy. At 14-month follow-up, he had minimal pain, improved range of motion and 4/5 strength, without recurrent infection or reoperation.DISCUSSION AND CONCLUSIONFew cases of E. coli septic arthritis of the native shoulder have been reported, most involving trauma, urologic procedures, or distant infection. In this case, no urinary or systemic infection, recent surgery, prosthesis, penetrating injury, or documented bacteremia was identified. Chronic kidney disease, mycophenolate-associated immunosuppression, and glenohumeral osteoarthritis were potential risk factors. Communication between the glenohumeral joint and subacromial-subdeltoid bursa likely contributed to the deltoid mass and may have obscured the diagnosis as an exacerbation of chronic rotator cuff arthropathy. Markedly elevated synovial WBC count and neutrophil predominance supported septic arthritis despite modest systemic inflammatory markers and absent systemic symptoms. Early aspiration, organism identification, and operative debridement enabled targeted therapy and durable improvement. Limitations included absence of blood cultures, an unidentified source, and negative intraoperative cultures following antibiotics. This case emphasizes that E. coli should be considered in native shoulder septic arthritis, particularly in immunosuppressed patients, even without systemic manifestations or an identifiable infectious source.

ABSTRACT O-04

Not Mucor After All

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Program: Damas Hospital Program

ABSTRACT

BackgroundRapidly progressive necrotic lesions of the nose raise immediate concern for invasive fungal rhinosinusitis, particularly mucormycosis. However, a growing body of literature shows that hematologic malignancies can closely mimic invasive fungal rhinosinusitis, both clinically and radiographically, particularly extranodal NK/T-cell lymphoma, nasal type (ENKTL).Case PresentationA 67-year-old male developed, over five days, erythema and edema of the nasal skin with nasal discharge, hyposmia, and difficulty breathing. CT demonstrated pansinusitis, nasal soft-tissue swelling, upper-airway narrowing, and cervical lymphadenopathy. Despite broad-spectrum antibacterial therapy, the overlying skin rapidly progressed to crusting, ulceration, and necrosis, with new scalp lesions. Given the destructive evolution, poor antibacterial response, and new information about spelunking in Barbados a few months earlier, invasive fungal disease became a major concern, raising suspicion for environmentally acquired fungal infections, such as histoplasmosis or mucormycosis. Empiric antifungal therapy was started with fluconazole and escalated to amphotericin B for coverage against suspected mucormycosis. Our patient developed fever to 39.4°C, trilineage cytopenias, ferritin 17,605 ng/mL, triglycerides 384 mg/dL, and soluble interleukin-2 receptor 16,801 U/mL. These findings fulfilled HLH-2004 criteria and had an HScore of 168 (estimated 40–54% probability) consistent with hemophagocytic lymphohistiocytosis (HLH). For this reason, corticosteroid therapy was started in an attempt to control the cytokine storm. Diagnosis was particularly challenging and delayed given that cutaneous biopsies were not routinely obtained at our institution. Eventually biopsies from a scalp lesion and nasal lesion were obtained, and no fungal hyphae were seen; special stains were also negative for fungal organisms. Histoplasma urine antigen was negative, and tissue biopsies had no evidence of Histoplasma. Histopathologic and immunophenotypic evaluation demonstrated an atypical T-cell lymphoproliferative process. By the time tissue sampling was performed, the patient was already critically ill and unstable. Although a diagnosis of T-cell lymphoma was established, extensive necrosis limited the analysis and precluded further subclassification. The patient's course was complicated by respiratory failure, multiorgan dysfunction, and seizures. Given the rapid progression of disease, the patient's continued clinical deterioration, and the limitations of the definitive treatment available at our institution, the goals of care were transitioned to palliative care. Despite intensive multidisciplinary supportive care, the patient's condition continued to deteriorate, ultimately resulting in his death.ConclusionInvasive fungal rhinosinusitis was initially suspected, however biopsy ultimately demonstrated T-cell lymphoma. T-cell lymphoma should be considered in rapidly progressive necrotic sinonasal and cutaneous lesions, particularly when presumed infection fails to respond to appropriate antimicrobial therapy. Secondary HLH, as in this case, is still largely underdiagnosed worldwide. It often manifests as a critical illness with sepsis-like manifestations that are unresponsive to sepsis-directed therapy. In a review involving 661 critically ill adults with HLH, infections were the most common trigger (50), followed by cancers (28%). This case highlights the risk of diagnostic anchoring and demonstrates the importance of early tissue biopsy given how a T-cell lymphoproliferative disease can closely mimic invasive fungal infection.

ABSTRACT O-05

Beyond the Left Atrial Appendage: An Unusual Calcified Right Atrial Appendage Thrombus in Chronic Atrial Fibrillation

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ABSTRACT

Introduction: Right atrial appendage (RAA) thrombus is a rare and underrecognized manifestation of atrial thrombosis. In a series of 983 patients undergoing transesophageal echocardiography (TEE) for atrial fibrillation or flutter, RAA thrombus was identified in only 0.73%, compared with 9.3% for left atrial appendage (LAA) thrombus.Case: A 74-year-old man with longstanding persistent atrial fibrillation, pancytopenia, chronic kidney disease secondary to polycystic kidney disease, and gout was referred for TEE after transthoracic echocardiography demonstrated an incompletely characterized calcified right atrial structure. Several months earlier, he had experienced an isolated syncopal episode with an unrevealing cardiac and neurologic evaluation. He subsequently remained clinically stable without recurrent syncope, chest pain, dyspnea, or symptoms of heart failure.TEE demonstrated preserved biventricular systolic function and left atrial dilation. Despite longstanding atrial fibrillation, no LAA thrombus was identified. Evaluation of the right atrium revealed a 3.9 × 1.72 cm nonmobile, heavily calcified hyperechoic structure at the level of the RAA extending toward the superior vena cava. Based on its location, morphology, and clinical context, the finding was considered most consistent with a chronic organized and calcified RAA thrombus, rather than an atrial myxoma or other intracardiac tumor.Conclusion: This case highlights the rare occurrence of RAA thrombus in atrial fibrillation despite the absence of LAA thrombus. Right atrial masses can pose a diagnostic challenge, particularly when chronic organization and calcification mimic an intracardiac tumor. Careful assessment of the RAA and consideration of multimodality imaging may help establish the diagnosis and guide management.

ABSTRACT O-06

Defying The Odds: Serial Autologous Blood Patch Rescue Of A High-Grade Pleuropulmonary Fistula In Ventilated Ards

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Program: San Juan City Hospital Program

ABSTRACT

IntroductionPersistent pleuropulmonary fistula during mechanical ventilation creates a therapeutic paradox: positive pressure required to sustain gas exchange simultaneously maintains the pressure gradient driving the air leak. High-grade, multisegmental fistulas are particularly difficult when profound hypoxemia precludes surgery and anatomy limits endobronchial valve placement. Evidence supporting autologous blood patch (ABP) therapy in this setting remains limited, particularly for high-flow leaks. We describe durable closure of a refractory fistula losing up to 60% of delivered tidal volume using serial pleural and targeted endobronchial ABP in a mechanically ventilated patient with severe acute respiratory distress syndrome (ARDS).Case PresentationA 61-year-old man with 20% total-body-surface-area electrical burns and traumatic cervical injury developed severe ARDS requiring prolonged mechanical ventilation, recurrent pneumothoraces, progressive subcutaneous emphysema, and a continuous left-sided air leak estimated at 45–60% of delivered tidal volume, with PaO_2:FiO_2 persistently <120.Definitive management options were severely constrained. Profound hypoxemia prevented safe single-lung ventilation for surgical repair, while multisegmental fistula anatomy limited endobronchial valve placement. A salvage strategy was pursued with 100 mL pleural ABP, followed by serial bronchoscopic endobronchial ABP. Fogarty balloon occlusion sequentially localized contributing bronchopulmonary segments, followed by targeted instillation of 20–30 mL autologous blood per segment.Each intervention produced a reproducible reduction in air leak accompanied by improvement in oxygenation, with PaO_2:FiO_2 increasing from approximately 95–110 to 150–165. Despite ongoing positive-pressure ventilation, multisegmental disease, multidrug-resistant pneumonia with Klebsiella aerogenes, Stenotrophomonas maltophilia, and Pseudomonas aeruginosa, and severe hypoalbuminemia (2.2 g/dL), serial treatment ultimately achieved complete and durable cessation of the air leak. Respiratory function progressively recovered, and the patient was ultimately discharged to rehabilitation.DiscussionThe challenge was not simply sealing the fistula—it was achieving closure while the ventilation required for survival continuously opposed healing. When surgery was physiologically prohibitive and valve therapy anatomically unsuitable, serial ABP provided a minimally invasive salvage strategy. The repeated temporal association between each intervention, reduced air leak, and improved oxygenation, followed by eventual durable closure, supports a plausible therapeutic effect despite formidable predictors of impaired healing. Spontaneous closure and concurrent ventilator optimization remain potential contributors and preclude definitive causal attribution.ConclusionHigh-grade pleuropulmonary fistula during ventilated ARDS can leave clinicians without conventional definitive options. In this patient, serial pleural and Fogarty-guided endobronchial ABP was associated with reproducible physiologic improvement, durable fistula closure, respiratory recovery, and discharge to rehabilitation. This case supports further evaluation of targeted serial ABP as individualized salvage therapy for otherwise refractory persistent air leaks.

ABSTRACT O-07

Beyond Infection: High-Grade B-Cell Lymphoma Presenting As A Chronic Thigh Ulcer

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Program: Universidad Central del Caribe Program

ABSTRACT

BEYOND INFECTION: HIGH-GRADE B-CELL LYMPHOMA PRESENTING AS A CHRONIC THIGH ULCERIntroduction: Chronic ulcerative skin lesions may initially be attributed to infection, but persistence despite appropriate antimicrobial therapy and source control should prompt reconsideration. We present high-grade B-cell lymphoma presenting as a progressive ulcerative thigh lesion complicated and initially obscured by recurrent polymicrobial infection.Case Description: A 29-year-old man with no significant medical history developed a pustular lesion on the right medial thigh five to six months before presentation that progressively enlarged and developed foul-smelling purulent drainage. After 21 days of amoxicillin without improvement, he was admitted. He was afebrile and hemodynamically stable, with WBC 5.23×10^3/µL, ESR 15 mm/hr, and CRP 3.18 mg/dL. Examination revealed a 13-cm full-thickness ulcer with slough, eschar, surrounding erythema, and raised borders, with an adjacent 4-cm ulcer. Wound cultures grew Morganella morganii, Proteus penneri, and Proteus mirabilis. He received vancomycin and piperacillin-tazobactam, underwent wide excision and debridement, and antibiotics were narrowed to levofloxacin, and he was discharged after approximately one week. Pathology from the excised tissue was nondiagnostic, showing a poorly differentiated malignant neoplasm without definitive lineage.Approximately three weeks after discharge, the lesion worsened, prompting readmission with recurrent erythema, malodor, and purulent drainage. The ulcer had enlarged to approximately 20 cm with irregular, raised violaceous borders and extensive slough, yet he remained afebrile without leukocytosis. CT of the thigh demonstrated a large soft-tissue defect with phlegmonous changes and lymphadenopathy without a drainable abscess or osteomyelitis. Repeat cultures grew Providencia stuartii and Citrobacter sedlakii. Pyoderma gangrenosum was considered, prompting pulse methylprednisolone while autoimmune evaluation was pursued; ANA, rheumatoid factor, and anticardiolipin antibodies were negative. Staging CT revealed widespread lymphadenopathy, scattered sub-6-mm pulmonary nodules, splenomegaly with multiple splenic lesions up to 5.4 cm, and intra-abdominal soft-tissue nodules concerning for disseminated malignancy. Repeat deep tissue biopsies established the diagnosis of high-grade B-cell lymphoma, not otherwise specified, with diffuse large-cell features; tumor cells were CD20-positive and CD45-positive with a non-germinal-center phenotype (CD10-negative, BCL-6-positive, MUM1-positive). MYC was positive by immunohistochemistry, but FISH detected no MYC, BCL-2, or BCL-6 rearrangements. He subsequently received six cycles of rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone (R-CHOP), with marked regression and progressive healing of the ulcerative lesion.Discussion: This case demonstrates how malignancy may be obscured by secondary infection. Repeated polymicrobial cultures and phlegmonous changes supported antimicrobial therapy but reinforced diagnostic anchoring. Months of progression, recurrence despite antibiotics and debridement, violaceous raised borders, a limited systemic inflammatory response, absence of a drainable collection, and widespread lymphadenopathy were discordant with isolated infection. Although tissue was obtained during the first admission, the initial pathology was nondiagnostic, underscoring the need for repeat, adequate tissue sampling when initial pathology is inconclusive and the clinical course remains discordant. Persistent or recurrent ulcerative lesions despite appropriate antimicrobial therapy and source control warrant reassessment, recognizing that infection and malignancy can coexist.

ABSTRACT O-08

Unmasking Amyotrophic Lateral Sclerosis in an Elderly Patient Hospitalized for Pneumonia and Pulmonary Embolism

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ABSTRACT

Introduction Amyotrophic lateral sclerosis (ALS) is a progressive neurodegenerative disorder with an annual incidence of 1.5-2.5 cases/100,000 individuals globally (JAMA.2026.John Ravits, MD, Dominic Ferrey, MD,et al), that typically presents with gradually worsening muscle weakness involving the limbs or bulbar musculature. Both upper and lower motor neurons are affected, producing a combination of spasticity, hyperreflexia, muscle atrophy, and fasciculations. Median survival is approximately three to five years, with latestage disease characterized by respiratory muscle failure and death. Case Presentation An 84-year-old male with a history of chronic obstructive pulmonary disease and hypothyroidism was transferred from a community hospital following a diagnosis of pneumonia. He also reported several days of general malaise, cough, rhinorrhea, and sore throat. Due to this generalized weakness, he experienced a fall while nourishing his animals, sustaining trauma to the head, right shoulder, and right knee. Upon arrival to the emergency department, CT angiography revealed acute pulmonary embolism, prompting activation of the PERT protocol and admission to the intensive care unit. Thrombolysis with tPA was attempted but not completed due to guidewire perforation of a left pulmonary artery branch. After stabilization, the patient was transferred to the Internal Medicine ward to continued management and discharge coordination. During his stay, he was noted to have bilateral upper and lower extremity fasciculations as well as tongue fasciculations. Further history from his niece revealed progressive functional decline and generalized weakness over the past year. Neurologic evaluation demonstrated diminished reflexes with hyperreflexia at the right elbow. Given his chronic malnutrition, etiology was initially unclear and suspected to be multifactorial. Electrodiagnostic testing was recommended. Electrodiagnostic studies demonstrated evidence of active denervation including spontaneous activity and fasciculations in multiple muscle groups. Additionally, several muscles showed chronic neurogenic changes such as polyphasic potential and increased motor unit amplitude, consistent with ongoing reinnervation. The overall pattern reflected multifocal neuropathic involvement with reduced motor unit recruitment and decreased interference across examined regions. Study findings and patient's clinical presentation led to a diagnosis of ALS. Discussion This 84-year-old man with newly diagnosed ALS had experienced a year of progressively decreasing functionality and generalized weakness, significantly increasing his frailty. His hospitalization for pneumonia and pulmonary embolism triggered a detailed neurological examination, revealing fasciculations and objective weakness. The clinical picture raised suspicion for an underlying neuromuscular disorder that had likely contributed to reduce his mobility, patient's fall, and, in turn, increased thromboembolic risk. Patient's constellation of clinical findings supported by the electrodiagnostic results lead to in ALS as patient's leading comorbidity diagnosis. Conclusion Unexplained decline in function and progressive weakness over months requires a broad differential diagnosis encompassing cardiac, pulmonary, metabolic, and neuromuscular etiologies. Although cardiopulmonary diagnoses are common, clinicians must remain vigilant for neuromuscular conditions when symptoms do not fully align with a typical cardiac, pulmonary or musculoskeletal pathology. Careful physical examination and attention to atypical findings such as fasciculations are essential in identifying critical disorders like ALS, particularly when more common diagnoses have been excluded.

ABSTRACT O-09

B-Cell Aplasia and Complete Metabolic Response Following CD19-Directed CAR-T Cell Therapy: A Clinical Vignette from Puerto Rico

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Program: Auxilio Mutuo

ABSTRACT

INTRODUCTIONChimeric antigen receptor T-cell (CAR-T) therapy has transformed treatment of large B-cell lymphoma, but delivery requires specialized infrastructure and longitudinal care. We describe a patient with refractory diffuse large B-cell lymphoma (DLBCL) treated with axicabtagene ciloleucel (axi-cel; Yescarta) in Puerto Rico, highlighting response, toxicity management, and integrated cellular-therapy delivery.CASE DESCRIPTIONA 45-year-old man was diagnosed with stage IV high-grade DLBCL after presenting with a left cervical mass. Excisional biopsy demonstrated CD20, CD10, and BCL6 positivity, a Ki-67 proliferative index of approximately 100%, and C-MYC rearrangement by FISH; BCL2 and BCL6 rearrangements were not detected. Disease was stage IVA with an R-IPI of 2. He received R-CHOP followed by six cycles of DA-EPOCH-R but developed recurrent cervical lymphadenopathy at the end of induction chemotherapy, imaging confirming refractory disease. Radiation and systemic bridging therapy with obinutuzumab, GemOx, and glofitamab were subsequently administered during CAR-T evaluation.Leukapheresis was completed on April, 2026. Pre-CAR-T included an HCT-CI of 2, left ventricular ejection fraction of 50%. Following fludarabine/cyclophosphamide lymphodepletion, axi-cel (2.0 × 10^8 anti-CD19 CAR-positive viable T cells) was infused on April 27, 2026, without immediate complications. On day +4, he developed grade 1 cytokine release syndrome (CRS), resolved after one dose of tocilizumab. No immune effector cell-associated neurotoxicity syndrome (ICANS) was documented; serial ICE scores remained 10/10, normal EEG and no acute pathology on brain MRI.Day +49, neck CT showed reduction of the left cervical nodal mass, residual 2.8 × 1.8 × 3.3-cm lesion as a positive treatment response. Chest CT showed no suspicious mediastinal, or axillary lymphadenopathy and no definite recurrent or metastatic disease. On day +58, complete B-cell aplasia (CD19+ cells, 0%; absolute CD19 count, 0 cells/μL) with preserved T-cell counts (CD3, 1,612 cells/μL; CD4, 470 cells/μL). On day +98, FDG-PET/CT demonstrated no significant FDG-avid disease consistent with a complete remission. Residual soft-tissue fullness at the previously involved left level II cervical site had an SUVmax of 2.0 and Deauville score of 2, consistent with CMR. At day +100, he remained without recurrent CRS, ICANS, prolonged cytopenias, or major infectious complications. Examinations showed no palpable lymphadenopathy, no organomegaly and a nonfocal neurologic examination; ICE 10/10 with grade 0 CRS/ICANS. In 2024 Puerto Rico established the first CAR-T therapy program in the island at Hospital Auxilio Mutuo. This required the development of a multidisciplinary team including medical subspecialty, social services, apheresis, coordinators and nurses among others. Thus far the program has performed 22 CAR-T cell infusions on patients with Non-Hodgkin lymphoma (17), multiple myeloma (2) and Acute Lymphocytic Leukemia (3).DISCUSSIONThis case demonstrates successful local delivery of axi-cel for refractory DLBCL. Despite prior treatment failure, he achieved CMR near day +100, with day +58 B-cell aplasia and preserved T-cell counts; toxicity was limited to grade 1 CRS responsive to one dose of tocilizumab, without ICANS. Coordinated candidacy assessment, bridging treatment, lymphodepletion, toxicity surveillance, prophylaxis, and outpatient follow-up supported a favorable early outcome. Integrated local capability may broaden access while preserving the specialized monitoring and continuity essential to CAR-T therapy.

ABSTRACT O-10

When Rhabdomyolysis Does Not Resolve: Recognizing an Immune-Mediated Myopathy After Statin Exposure

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Program: University of Puerto Rico Program

ABSTRACT

Introduction:Statin-associated muscle injury generally improves after withdrawal of the offending medication. Persistent weakness and marked creatine kinase elevation despite medication discontinuation should prompt evaluation for an immune-mediated myopathy.Case Description:A 63-year-old woman with hypertension, dyslipidemia, and prior statin exposure presented with progressive proximal muscle weakness, myalgias, fatigue, and dysphagia. She had recently been hospitalized with rhabdomyolysis and a creatine kinase (CK) level of approximately 28,000 U/L. Despite discontinuation of lipid-lowering therapy and intravenous hydration, significant weakness and marked CK elevation persisted, prompting readmission and rheumatologic evaluation.Examination demonstrated symmetric proximal muscle weakness, most pronounced in the lower extremities, without heliotrope rash, Gottron papules, Raynaud phenomenon, sclerodactyly, or inflammatory arthritis. MRI of the pelvis and thigh demonstrated diffuse muscle edema compatible with myositis. Rheumatologic evaluation favored an inflammatory myopathy without clinical features of a defined connective tissue disease overlap syndrome.Because of persistent weakness, dysphagia, and severe hyperCKemia, she was treated with pulse-dose corticosteroids and intravenous immunoglobulin, followed by prednisone 60 mg daily. Dysphagia improved, allowing her to tolerate solid foods without choking or aspiration symptoms, while CK decreased to 4,857 U/L. Proximal lower-extremity strength remained 3/5 at that time. Subsequent muscle biopsy demonstrated active myopathy with necrotic and regenerating fibers, slight denervation atrophy, and patchy type 2B fiber atrophy. The pathology was considered compatible with recent rhabdomyolysis or early immune-mediated necrotizing myopathy, with recommendation for clinical and serologic correlation.Discussion:This case highlights the importance of reconsidering the diagnosis when presumed toxic rhabdomyolysis does not follow its expected course. Recognition of this pattern may shift management from supportive measures alone to immunosuppressive therapy. Muscle biopsy may demonstrate predominantly necrotic and regenerating fibers with limited inflammation and should be interpreted together with the clinical course and serologic evaluation. Persistent weakness and CK elevation after withdrawal of a suspected offending medication should prompt evaluation for an immune-mediated myopathy, as recognition may shift treatment from supportive measures alone to immunosuppressive therapy.

2026 ACP PUERTO RICO CHAPTER

CLINICAL VIGNETTES & RESEARCH COMPETITION

Clinical Vignettes Presentation Abstracts

ABSTRACT V-1

Recurrent Supraventricular Tachyarrhythmias Following Viral Respiratory Infections: A Case Report

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Program: Universidad de Guadalajara

ABSTRACT

Supraventricular tachyarrhythmias are recognized cardiovascular complications of acute infections; however,recurrent arrhythmic episodes occurring during separate viral respiratory illnesses are rarely reported. Wepresent the case of a 40 year old female with recurrent supraventricular tachyarrhythmias temporally associatedwith two distinct viral respiratory infections occurring five years apart.The patient initially experienced symptomatic tachyarrhythmia during a COVID-19 infection in 2021. Sheremained clinically stable until 2026, when she presented with palpitations and dizziness during an acuteInfluenza B infection. On arrival, she developed a narrow complex tachycardia with rates exceeding 200 beatsper minute that required adenosine administration for termination. Despite initial medical therapy with metoprololand flecainide, recurrent episodes persisted.Electrophysiologic study demonstrated dual atrioventricular nodal physiology consistent with atrioventricularnodal reentrant tachycardia (AVNRT), and successful slow pathway ablation was performed. Unexpectedly, thepatient continued to experience recurrent supraventricular tachyarrhythmias following ablation, raising concernfor additional atrial arrhythmic mechanisms. Echocardiography revealed newly reduced left ventricular systolicfunction with an ejection fraction of 40–45%, right ventricular systolic dysfunction, and a small localizedpericardial effusion. Cardiac biomarkers remained within normal limits.The temporal association between recurrent arrhythmic episodes and separate viral respiratory infectionssuggests a potential infection related trigger in a susceptible patient. This case highlights the diverseelectrophysiologic manifestations that may accompany acute viral illnesses and emphasizes the importance ofconsidering recurrent infection associated arrhythmias when evaluating patients with unexplainedtachyarrhythmias. Further investigation is needed to better understand the relationship between viral respiratoryinfections and recurrent supraventricular arrhythmias.

ABSTRACT V-2

Recurrent Diabetic Ketoacidosis Following Platelet Transfusion in a Patient With Newly Diagnosed Chronic Lymphocytic Leukemia

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Program: Damas Hospital Program

ABSTRACT

Diabetic ketoacidosis (DKA) is classically precipitated by infection, insulin deficiency, or medications. Platelet transfusions are associated with numerous immunologic and inflammatory complications, yet metabolic decompensation leading to DKA has not been previously described. We report a patient with recurrent DKA occurring reproducibly after platelet transfusions in the setting of newly diagnosed chronic lymphocytic leukemia (CLL).A 66 year old man with insulin treated type 2 diabetes mellitus (HbA1c 6.8%), coronary artery disease, hypertension, and hypothyroidism presented with severe thrombocytopenia and was diagnosed with CLL. During his hospitalization, he developed repeated episodes of DKA characterized by serum glucose >400 mg/dL, high anion gap metabolic acidosis, and ketosis. Each episode occurred within 12–24 hours of platelet transfusion and resolved with guideline directed treatment using intravenous insulin, aggressive fluid resuscitation, and electrolyte replacement. Corticosteroids were administered only during the initial hospitalization; however, subsequent DKA episodes followed additional platelet transfusions despite complete absence of steroid exposure. Serial evaluations excluded recurrent infection, myocardial ischemia, medication changes, or other recognized precipitants. After initiation of CLL directed therapy, thrombocytopenia improved, platelet transfusions were no longer required, and no further episodes of DKA occurred.The striking reproducibility of DKA following successive platelet transfusions, together with the absence of alternative triggers, suggests a previously unrecognized temporal association. Platelet products contain biologically active inflammatory mediators capable of inducing systemic cytokine release and transient insulin resistance, effects that may be amplified in patients with hematologic malignancies and underlying diabetes. Although causality cannot be established from a single observation, this case raises the possibility that platelet transfusion may serve as a metabolic stressor capable of precipitating DKA in susceptible individuals.This case highlights a potential transfusion associated trigger for DKA that has not been previously reported. Clinicians should consider platelet transfusion among the differential precipitants of unexplained DKA in high risk patients and maintain close post transfusion glucose surveillance. Recognition of this possible association may facilitate earlier diagnosis and treatment while further investigation clarifies its mechanism and clinical significance.

ABSTRACT V-3

Large Malignant Pericardial Effusion Without Tamponade: Hemodynamic Assessment Guiding Management

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Program: Not provided

ABSTRACT

Malignant pericardial effusion (MPE) is a life-threatening complication of advanced malignancy that frequently reflects metastatic disease and is associated with poor overall prognosis and limited survival. Although most commonly encountered in patients with lung and breast cancer, MPE may also occur in gastrointestinal malignancies, including pancreatic adenocarcinoma. Clinical management is particularly challenging in patients with concomitant pulmonary arterial hypertension (PAH) or connective tissue disorders such as systemic sclerosis, where dyspnea often has multiple competing etiologies and echocardiographic findings alone may not accurately reflect the degree of hemodynamic compromise. Furthermore, the presence of a large pericardial effusion does not invariably indicate cardiac tamponade, making invasive hemodynamic assessment an important diagnostic adjunct in selected patients. We present a case in which right heart catheterization (RHC) provided critical physiologic information that guided intervention and facilitated successful treatment with balloon pericardiostomy.71-year-old woman with stage IV metastatic pancreatic adenocarcinoma, systemic sclerosis, and pulmonary arterial hypertension presented with progressive dyspnea and worsening shortness of breath over several days. She denied chest pain or syncope. On presentation, she was hemodynamically stable without hypotension or clinical evidence of shock. Chest radiography demonstrated marked cardiomegaly with a classic “water bottle” cardiac silhouette, raising concern for a significant pericardial effusion. Transthoracic echocardiography confirmed a large circumferential pericardial effusion with partial right atrial collapse but no definitive evidence of cardiac tamponade. Given her advanced malignancy, multiple comorbidities, and limited life expectancy, her case was reviewed during a multidisciplinary cardiac conference. After discussion among cardiology, interventional cardiology, and the treating oncology team, a strategy incorporating invasive hemodynamic assessment followed by a more durable drainage procedure was selected rather than isolated pericardiocentesis to maximize symptomatic relief while minimizing recurrence.The patient underwent RHC, which demonstrated mild pulmonary hypertension with a mean pulmonary artery pressure of 24 mmHg, right ventricular pressure of 39/7 mmHg, right atrial pressure of 10 mmHg, pulmonary capillary wedge pressure of 19 mmHg, cardiac output of 5.1 L/min, and pulmonary vascular resistance of 1 Wood unit. Importantly, there was no equalization of diastolic pressures, confirming the absence of classic tamponade physiology despite the large pericardial effusion. These findings clarified that her symptoms were multifactorial and allowed safe progression to definitive intervention. Under combined fluoroscopic and echocardiographic guidance, subxiphoid percutaneous pericardial access was obtained. An 18-mm BARD True Dilatation balloon catheter was advanced over a super-stiff guidewire to perform balloon pericardiostomy, creating a controlled communication for continuous pericardial drainage. Approximately 500 mL of serous fluid was evacuated without procedural complications. The patient experienced immediate improvement in dyspnea, remained clinically stable throughout hospitalization, and was discharged home two days later. Management of malignant pericardial effusion should integrate prognosis, symptom burden, and hemodynamic assessment. This case demonstrates that even a large pericardial effusion may not represent cardiac tamponade, particularly in patients with pulmonary vascular disease or connective tissue disorders. Right heart catheterization provided essential physiologic confirmation that guided safe intervention, while balloon pericardiostomy offered durable symptom relief through a minimally invasive palliative approach with a lower likelihood of recurrence than isolated pericardiocentesis.

ABSTRACT V-4

Checkpoint to Cytokine Storm: Pembrolizumab-Associated HLH Presenting as Septic Shock

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Program: Not provided

ABSTRACT

Hemophagocytic lymphohistiocytosis (HLH) is a life-threatening hyperinflammatory syndrome characterized by immune dysregulation, cytopenias, and multiorgan involvement. Immune checkpoint inhibitors have emerged as a rare but important trigger of HLH-like syndromes, often mimicking sepsis and posing a diagnostic challenge.We present a 62-year-old man with lung adenocarcinoma (pathologic stage T3N0M0) status post lobectomy and adjuvant Cisplatin and Pemetrexed, transitioned to Pembrolizumab after treatment-limiting neuropathy. After three cycles of Pembrolizumab, he was admitted with several days of progressive dyspnea, malaise, weakness, and subjective fever. On presentation, he was hypotensive with elevated lactate and inflammatory markers, requiring vasopressor support for presumed septic shock. Extensive infectious workup, including blood cultures, respiratory testing, cerebrospinal fluid studies, and advanced molecular testing, remained negative. Imaging did not identify a clear infectious source.During hospitalization, the patient developed persistent high-grade fevers, worsening malaise, and progressive bicytopenia with leukopenia and severe thrombocytopenia (nadir 29,000/mcL). Laboratory evaluation revealed hyperferritinemia (4,106 ng/mL), hypertriglyceridemia (439 mg/dL), mild transaminitis, and elevated inflammatory markers. Fibrinogen remained within normal limits. Peripheral smear showed no schistocytes. Imaging demonstrated hepatosplenomegaly, further supporting a systemic inflammatory process. Although bone marrow biopsy and soluble CD25 testing were not performed, the constellation of fever, cytopenias, hyperferritinemia, hypertriglyceridemia, and organomegaly raised strong concern for HLH or an HLH-like syndrome.Given the absence of an identifiable infectious etiology and recent exposure to immune checkpoint inhibition, an immune-related adverse event was suspected. The patient was treated with high-dose corticosteroids, initially dexamethasone followed by methylprednisolone and transition to oral Prednisone. He demonstrated rapid clinical improvement, including resolution of fevers, recovery of blood counts, and overall symptomatic improvement without need for transfusion support.This case highlights a rare but critical complication of Pembrolizumab therapy presenting as an HLH-like syndrome. The presentation closely mimicked septic shock, emphasizing the importance of maintaining a high index of suspicion for HLH in patients with persistent fevers, cytopenias, and elevated inflammatory markers despite negative infectious evaluation. Early recognition and prompt initiation of immunosuppressive therapy are essential, as timely corticosteroid treatment can be life-saving and lead to rapid clinical recovery.Immune checkpoint inhibitor–associated HLH remains an under-recognized entity that may not fulfill classic diagnostic criteria, further complicating diagnosis. Increased awareness is necessary to differentiate this condition from sepsis and to guide appropriate management in oncology patients receiving immunotherapy.

ABSTRACT V-5

When Fatigability Misleads: Acute Cerebellar Stroke Masquerading as Myasthenia Gravis Exacerbation

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Program: Not provided

ABSTRACT

Background:Myasthenia gravis (MG) is an autoimmune disorder of the neuromuscular junction characterized by fluctuating skeletal muscle weakness and fatigability. Exacerbations are commonly triggered by infection and often present with worsening generalized, bulbar, or respiratory weakness. This expected presentation may lead clinicians to attribute new neurologic symptoms solely to MG, delaying recognition of alternative diagnoses. Distinguishing MG exacerbation from central nervous system pathology is essential when atypical findings are present.Case Presentation:A 64-year-old man with acetylcholine receptor antibody-positive MG treated with pyridostigmine, prednisone, and azathioprine presented with 12 hours of generalized weakness and vomiting. His medical history included type 2 diabetes mellitus, hyperlipidemia, and chronic oropharyngeal dysphagia. On admission, extremity weakness was consistent with his documented MG baseline. Initial laboratory studies and non-contrast head computed tomography (CT) were unremarkable. Chest imaging demonstrated a left lower lobe opacity concerning for pneumonia, and broad-spectrum antibiotics were initiated for presumed infection-triggered MG exacerbation. Respiratory parameters remained reassuring, including a negative inspiratory force of –60 cm H_2O, an appropriate single-breath count, and no evidence of respiratory compromise.Despite treatment, the patient developed progressive right upper extremity weakness and increasing difficulty maintaining head posture. Repeat neurologic examination demonstrated bilateral fatigability but also asymmetric weakness, more pronounced on the left, with hyperreflexia and sustained clonus in the left lower extremity. Cranial nerve examination remained unchanged from baseline without new ptosis, diplopia, or worsening bulbar dysfunction. Given these atypical findings, brain magnetic resonance imaging (MRI) was obtained, revealing a large left cerebellar lesion causing mass effect on the fourth ventricle and brainstem, along with a small acute-to-subacute left parieto-occipital infarct. Initial differential diagnosis included a posterior fossa mass versus hemorrhagic stroke. Following multidisciplinary review, imaging findings were determined to represent an acute ischemic cerebellar infarction with hemorrhagic conversion. The patient was managed conservatively in the intensive care unit with corticosteroids, serial neurologic examinations, and close radiographic monitoring. Neurosurgical intervention was not required, and he was discharged with outpatient neurology follow-up.Discussion:This case illustrates the importance of avoiding diagnostic anchoring in patients with established MG presenting with worsening weakness. Although infection is a common trigger for MG exacerbations, asymmetric weakness, hyperreflexia, clonus, and preserved respiratory function are atypical and should prompt evaluation for central nervous system pathology. Posterior fossa strokes, particularly those with hemorrhagic conversion, may initially present with subtle neurologic deficits but can rapidly become life-threatening because of edema, mass effect, and brainstem compression. Careful neurologic examination and timely neuroimaging were essential in establishing the correct diagnosis and preventing inappropriate escalation of MG-directed therapy.Conclusion:Patients with MG who present with worsening weakness require careful assessment beyond the assumption of disease exacerbation. The presence of focal neurologic deficits or upper motor neuron signs should prompt urgent neuroimaging, as early recognition of ischemic stroke with hemorrhagic conversion can substantially alter management and improve outcomes.

ABSTRACT V-6

When the Kidney Speaks Through Hypertension: A Case of Page Kidney

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ABSTRACT

Page kidney is a rare but reversible cause of secondary hypertension due to external renal compression by subcapsular or perinephric fluid collections, leading to activation of the renin-angiotensin-aldosterone system. It is often associated with trauma, surgery, or spontaneous bleeding, and diagnosis may be delayed due to nonspecific symptoms.Our patient is a 35-year-old male with a history of hypertension, polysubstance use disorder, and end-stage kidney disease (ESKD) on thrice-weekly intermittent hemodialysis who presented to the emergency department after a motor vehicle accident 2 days prior resulting in head trauma ad intracranial hemorrhage. On evaluation the patient complaints of headache and dizziness and was clinically euvolemic. Vital signs were remarkable for elevated blood pressure readings >200/100 mmHg requiring clevidipine infusion. Laboratory studies showed elevated renal function markers but no significant acid-base or electrolyte abnormalities. Head CT scan revealed a subdural hematoma but unremarkable cervical, chest and abdominal imaging. Available antihypertensive regimen were initiated and titrated to maximal doses, including losartan 100 mg daily, carvedilol 25 mg twice daily, nifedipine extended-release 60 mg twice daily, doxazosin 4 mg twice daily, hydralazine 100 mg three times daily, and clonidine 0.3 mg three times daily. Additionally, hemodialysis frequency was increased to daily with maximal ultrafiltration; however, the patient remained dependent on clevidipine infusion, despite being asymptomatic. Following 5 days of admission patient remained hypertensive but with sinus tachycardia, moreover, acute hemoglobin drop noted from a baseline 9.5mg/dl to 6.5mg/dl prompting further evaluation including abdominopelvic CT angiogram which revealed a large intra-abdominal hemorrhage due a grade 3 splenic laceration. The patient underwent exploratory laparoscopy with evacuation of approximately 6 liters of serosanguineous fluid and embolization of the splenic artery. Following the procedure, the clevidipine infusion and multiple oral antihypertensives were successfully discontinued.This case emphasizes the importance of considering Page Kidney in patients with kidney disease who present with persistent, treatment-resistant hypertension, especially in cases involving trauma or intra-abdominal bleeding. The normalization of blood pressure following the removal of the compressive hematoma supports the diagnosis. It underscores the need for early recognition and a high level of suspicion, particularly in ESKD, as prompt intervention can help prevent prolonged hypertensive episodes and potential end-organ damage.

ABSTRACT V-7

The Cardio-Biliary Reflex: A Case of Cope's Phenomenon in Acute Calculous Cholecystitis

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Program: Not provided

ABSTRACT

Cope's phenomenon, or the cardio-biliary reflex, is a rare condition in which gallbladder inflammation or distention triggers a vagally mediated autonomic response, causing transient bradyarrhythmias. These cardiac findings may mimic primary cardiac disease and lead to unnecessary evaluation or delayed recognition of biliary pathology. We present a case of acute calculous cholecystitis associated with transient sinus bradycardia that resolved after treatment of the underlying gallbladder disease.A 63-year-old female presented to the Emergency Department with one day of severe right upper quadrant pain radiating to the back, associated with fatigue, headache, nausea, and two episodes of non-bloody, non-bilious emesis. She reported intermittent similar episodes over the preceding year, with the current pain being significantly more severe. She denied fever, chills, jaundice, urinary symptoms, or bowel changes. On examination, she was in significant distress with marked right upper quadrant tenderness and a positive Murphy sign. ECG showed sinus bradycardia at 53 beats per minute. High-sensitivity troponin was 16 ng/L and within normal limits, with no evidence of acute myocardial injury. She also had uncontrolled hypertension, treated with nifedipine and intravenous enalapril. CT abdomen/pelvis and right upper quadrant ultrasound demonstrated acute calculous cholecystitis with a 2.0-cm gallstone impacted at the gallbladder neck. She received morphine, ketorolac, and acetaminophen and was kept nil per os. General Surgery initially recommended conservative management. Following analgesia and improvement of the inflammatory process, her abdominal pain improved, and repeat ECG showed resolution of bradycardia with normal sinus rhythm at 78 beats per minute. She subsequently underwent cholecystectomy for definitive treatment.Cope's phenomenon occurs when gallbladder inflammation or distention activates vagal afferent pathways, increasing parasympathetic activity and producing transient cardiac conduction abnormalities. Although sinus bradycardia is most common, atrioventricular block and other ECG changes have also been reported. This phenomenon should be considered in patients with acute right upper quadrant pain and unexplained bradycardia after acute coronary syndrome and primary conduction disorders have been excluded. In this patient, normal cardiac biomarkers, absence of ischemic ECG changes, and resolution of bradycardia with improvement of biliary symptoms supported a vagally mediated cardio-biliary reflex.Recognition of this rare association is important because cardiac manifestations may mimic acute coronary syndrome and lead to unnecessary interventions. Identifying the underlying biliary pathology allows appropriate treatment of the gallbladder while maintaining vigilance for potentially life-threatening cardiac conditions.

ABSTRACT V-8

Rings, Rash, and Relapse: Sequential Cerebral Toxoplasmosis and Ramsay Hunt Syndrome in Advanced AIDS

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Program: San Juan City Hospital Program

ABSTRACT

IntroductionMultiple ring-enhancing brain lesions often trigger concern for metastatic malignancy, yet the same pattern in an immunocompromised host may represent a reversible opportunistic infection. Cerebral toxoplasmosis occurs predominantly at CD4 counts below 100 cells/µL, whereas Ramsay Hunt syndrome has an estimated incidence of only 5 per 100,000 persons annually. We describe three admissions in a patient with newly diagnosed AIDS during which presumed brain metastases became cerebral toxoplasmosis, new cranial neuropathy revealed concurrent viral and bacterial infections, and subsequent radiographic progression required distinction among relapse, resistance, immune reconstitution inflammatory syndrome, and medication nonadherence.Case PresentationA previously independent 40-year-old man without known chronic conditions presented with three weeks of progressive dysarthria, gait instability, and left-sided weakness following a seizure-like episode. Brain magnetic resonance imaging demonstrated numerous heterogeneously ring-enhancing supratentorial lesions with central necrosis, extensive vasogenic edema, and mass effect, initially concerning for metastatic malignancy. Computed tomography of the chest, abdomen, and pelvis revealed no primary malignancy. Fourth-generation HIV testing was positive, with HIV-1 RNA 242,000 copies/mL and CD4 count 46 cells/µL. Strongly positive Toxoplasma gondii IgG shifted the leading diagnosis to cerebral toxoplasmosis. Empiric anti-Toxoplasma therapy, dexamethasone, and antiretroviral therapy were initiated without stereotactic brain biopsy. Clinical and radiographic improvement supported the presumptive diagnosis and permitted discharge. Two weeks later, he returned with right peripheral facial paralysis, vesicular external auditory canal lesions, marked edema, and purulent drainage, establishing simultaneous Ramsay Hunt syndrome and acute otitis externa. Cultures grew Pseudomonas putida, Enterococcus faecalis, and Bacillus species. Intravenous acyclovir, corticosteroids, and targeted antimicrobials produced clinical improvement. Five weeks later, he presented with positional dizziness, vomiting, and persistent contralateral otalgia. Repeat MRI demonstrated interval progression of multiple contrast-enhancing lesions involving the posterior frontal, right parietal, and bilateral occipital regions. Progressive toxoplasmosis, antimicrobial resistance, immune reconstitution inflammatory syndrome, and medication nonadherence were considered. Multidisciplinary evaluation favored nonadherence based on difficulty following the regimen, inconsistent follow-up, and limited understanding of treatment requirements.DiscussionEach admission required reframing rather than reflexively extending the preceding diagnosis. Previously unrecognized immunosuppression transformed presumed metastases into a treatable opportunistic infection; new facial paralysis identified simultaneous viral and polymicrobial otologic disease; and later radiographic progression demanded separation of biologic treatment failure from a modifiable barrier to treatment. More than 90% of appropriately treated patients with cerebral toxoplasmosis improve neurologically within 14 days, while toxoplasmosis-associated immune reconstitution inflammatory syndrome occurs in approximately 5% of cases. Initial response followed by progression, together with documented medication barriers, favored nonadherence over resistance or immune reconstitution.ConclusionIn advanced HIV, new neurologic findings warrant renewed diagnostic inquiry rather than automatic attribution to an established opportunistic infection. Across three admissions, evolving symptoms revealed overlapping parasitic, viral, and bacterial processes, while careful integration of host factors, imaging, and therapeutic response avoided an invasive brain biopsy. This case underscores that successful treatment requires not only diagnostic accuracy, but also early recognition and mitigation of barriers to adherence.

ABSTRACT V-9

The Postoperative Water-Soluble Contrast Challenge: A High-Value Diagnostic Sequence for Suspected Recurrent Small-Bowel Obstruction

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Program: San Juan City Hospital Program

ABSTRACT

Introduction: Distinguishing recurrent mechanical small-bowel obstruction from postoperative ileus is a high-stakes diagnostic challenge. During the early postoperative period, diffuse bowel dilation and delayed transit may resemble recurrent obstruction on computed tomography. Premature re-laparotomy exposes older adults to anesthesia-related complications, infection, aspiration, and critical care. When urgent surgical indications are absent, a water-soluble contrast challenge can provide dynamic information about intestinal patency and the likelihood of successful nonoperative management [1].**Case Description:** A 75-year-old man presented with complete mechanical small-bowel obstruction. An initial water-soluble contrast challenge failed to demonstrate colonic transit and supported urgent exploratory laparotomy with adhesiolysis. On postoperative day 3, he developed recurrent severe abdominal distension. Computed tomography demonstrated diffuse bowel dilation and a possible transition point but could not reliably distinguish recurrent mechanical obstruction from postoperative ileus.The patient remained hemodynamically stable, serial lactate levels remained normal, and repeated abdominal examinations did not demonstrate progressive findings mandating emergent re-exploration. The multidisciplinary team administered 90 mL of Gastrografin and obtained abdominal radiographs at 8 and 24 hours. Although contrast had not reached the colon by 24 hours, imaging demonstrated progressive distal migration without clinical deterioration. Abdominal radiographs and surgical examinations were therefore continued every 24 hours.On day 5 after contrast administration, radiographs demonstrated contrast within the cecum, coinciding with resolution of the abdominal distension and obstructive symptoms. These findings demonstrated intestinal patency and supported postoperative ileus rather than recurrent high-grade mechanical obstruction. He was managed with bowel rest, fluid and electrolyte optimization, and close monitoring. Oral intake was resumed, and he was discharged without reoperation on postoperative day 11.**Discussion:** Failure of contrast to reach the colon within 24 hours should raise concern for persistent obstruction, but early after surgery this finding should not be interpreted in isolation. In our patient, continued distal movement of contrast, improving symptoms, and reassuring serial examinations supported continued observation [1], [2]. The contrast study remained an adjunct to clinical judgment rather than a replacement for it. Hemodynamic instability, peritoneal signs, worsening metabolic markers, or imaging findings concerning for ischemia would have required urgent surgical reassessment.Water-soluble contrast is best viewed as a diagnostic and prognostic tool because its independent therapeutic benefit remains uncertain [3]. High Value Care came from combining bedside reassessment, laboratory monitoring, and serial abdominal radiographs to follow the clinical trajectory and avoid premature re-laparotomy. Although contrast protocols have been associated with shorter hospitalizations and lower costs [4], these savings were not measured in this patient. Observation for five days should not be applied routinely; it was reasonable because the patient remained stable, the contrast continued to advance, and direct surgical monitoring was maintained. This case shows that High Value Care can mean allowing postoperative physiology time to improve while remaining prepared to intervene if the patient deteriorates.

ABSTRACT V-10

"The Worst Shoulder Pain of My Life": Spontaneous Cervicothoracic Epidural Hematoma Presenting as Acute Paraplegia

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Program: Consortium Hospital Episcopal San Lucas

ABSTRACT

IntroductionSpontaneous spinal epidural hematoma (SSEH) is a rare neurosurgical emergency with an estimated annual incidence of approximately 0.1 per 100,000 persons. Early diagnosis is challenging because the initial presentation frequently mimics benign musculoskeletal or cerebrovascular disorders, while delayed recognition may result in irreversible spinal cord injury. We present an unusual case of spontaneous cervicothoracic epidural hematoma presenting initially as isolated atraumatic shoulder pain before rapidly progressing to complete paraplegia. This case emphasizes the importance of maintaining a broad differential diagnosis when evaluating severe shoulder pain accompanied by evolving neurologic symptoms.Case PresentationA 71-year-old woman with hypertension, type 2 diabetes mellitus, stage III chronic kidney disease, and a history of cardiac arrest presented to the emergency department complaining of “the worst shoulder pain of my life.” She denied trauma, falls, anticoagulant or antiplatelet use, recent spinal procedures, fever, or infectious symptoms. Within hours, she developed progressive bilateral lower-extremity paresthesias followed by complete paraplegia. Initial computed tomography of the brain and spine demonstrated no acute abnormalities. Given her rapidly progressive neurologic deficits, magnetic resonance imaging was obtained and revealed an extensive anterior epidural hematoma extending from C2 through T3, causing severe cervical spinal cord compression with associated intramedullary cord signal abnormality from C7 through T3. A smaller posterior thoracic epidural hematoma was also identified without significant thoracic cord compression. The patient underwent urgent C4–C7 decompressive laminectomies with evacuation of the hematoma approximately 48 hours after symptom onset. Her postoperative course was complicated by transient KDIGO stage 2 acute kidney injury and uncontrolled diabetes mellitus, both of which improved with appropriate medical management. Despite surgical decompression approximately 48 hours after symptom onset, the patient had no significant neurologic recovery and remained paraplegic at discharge. She was transferred to an inpatient rehabilitation facility for long-term rehabilitation. During follow-up several months later, she developed an unstageable sacral pressure ulcer secondary to persistent immobility, further highlighting the devastating long-term consequences of irreversible spinal cord injury.DiscussionSSEH should be considered in patients presenting with sudden atraumatic neck or shoulder pain followed by evolving neurologic deficits, even in the absence of trauma, anticoagulation, or coagulopathy. Pain frequently precedes motor deficits, making early diagnosis particularly challenging and increasing the risk of irreversible spinal cord injury. This case highlights three important clinical lessons. First, isolated severe shoulder pain may represent the earliest manifestation of impending spinal cord compression rather than primary musculoskeletal disease. Second, normal computed tomography does not exclude SSEH; magnetic resonance imaging remains the diagnostic modality of choice when neurologic deficits evolve. Finally, delayed diagnosis may result in permanent neurologic disability despite appropriate surgical decompression, emphasizing the critical importance of early recognition, prompt MRI, and urgent neurosurgical consultation to optimize neurologic outcomes.

ABSTRACT V-12

From 60% to 20%: Rapidly Progressive Cardiomyopathy Unmasking Systemic Sarcoidosis in an Elderly Patient

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Program: Universidad Central del Caribe Program

ABSTRACT

Introduction: Sarcoidosis is a multisystem granulomatous disease most commonly affecting the lungs and intrathoracic lymph nodes. Although it typically presents in young adults, it also occurs in patients older than 65 years, in whom its diagnosis may be delayed because its manifestations often mimic malignancy, infection, or ischemic cardiomyopathy. While cardiac involvement is clinically recognized in only approximately 5% of patients, it is a major cause of sarcoidosis-related mortality as delayed diagnosis may result in irreversible myocardial injury. We present an elderly patient whose rapidly progressive heart failure led to the diagnosis of systemic sarcoidosis with probable cardiac involvement.Case description: A 79-year-old man with prior coronary artery bypass grafting, bioprosthetic aortic valve replacement, and permanent pacemaker placement presented with two months of progressive dyspnea, hypoxemia, and bilateral lower extremity edema. Five weeks earlier, left heart catheterization had demonstrated non-obstructive coronary artery disease with a preserved left ventricular ejection fraction (LVEF) of 55–60%. On current admission, laboratory values were remarkable for elevated pro-BNP at 1930 pg/mL and echocardiography showed a reduced LVEF of 35–40%, consistent with acute decompensation of heart failure. Computed tomography of the chest and abdomen revealed bilateral peribronchial consolidations, partially calcified mediastinal lymph nodes, and innumerable hypodense hepatic and splenic lesions, raising concern for disseminated infection or metastatic malignancy. Despite empiric broad-spectrum antibiotics, repeated blood, urine, and sputum cultures remained negative, and the patient failed to improve clinically. Liver biopsy demonstrated well-formed non-caseating granulomas without evidence of malignancy, acid-fast bacilli, or fungal organisms, establishing biopsy-proven systemic sarcoidosis. Applying the Heart Rhythm Society consensus criteria, a diagnosis of probable cardiac sarcoidosis was established. Despite two weeks of high-dose intravenous corticosteroids and optimal medical therapy for heart failure, repeat echocardiography showed further deterioration to an LVEF of 20–25%. The patient experienced persistent hypoxemic respiratory failure, progressive functional decline, and refractory heart failure, ultimately transitioning to hospice care.Discussion:This case illustrates that rapidly progressive left ventricular dysfunction despite recently documented non-obstructive coronary arteries should prompt consideration of infiltrative cardiomyopathy, even in elderly patients with preexisting cardiovascular disease. In the setting of biopsy-proven extracardiac sarcoidosis, unexplained conduction disease, and rapidly progressive nonischemic cardiomyopathy, cardiac sarcoidosis should remain a key diagnostic consideration, particularly when advanced cardiac imaging, such as FDG-PET and CMR, is not feasible. Early recognition is critical, as timely immunosuppressive therapy may limit progression to irreversible myocardial fibrosis.

ABSTRACT V-13

Racing The Clock: Racing Anti-Mda5 Rapidly Progressive Interstitial Lung Disease From Puerto Rico To Bilateral Lung Transplantation

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ABSTRACT

IntroductionAnti-melanoma differentiation-associated gene 5 (anti-MDA5) dermatomyositis accounts for under 2% of idiopathic inflammatory myopathies, yet carries the highest pulmonary mortality of any myositis phenotype: rapidly progressive interstitial lung disease (RP-ILD) develops in 40–60% of affected patients and kills 33–67% within six months. Lung transplantation is the only definitive rescue for refractory disease, but fewer than 15 recipients have been reported worldwide. Data in Hispanic populations are essentially absent. Puerto Rico lacks a lung-transplant center and had no locally available pathway to bridge this patient to transplantation with ECMO.Case PresentationA 49-year-old Puerto Rican man presented with clinically amyopathic dermatomyositis, anti-MDA5 antibody positivity, marked hyperferritinemia of 4,687 ng/mL, and progressive exertional dyspnea requiring 3 L/min supplemental oxygen. High-resolution computed tomography demonstrated bibasilar consolidation and ground-glass opacities evolving into extensive pleuroparenchymal fibrosis with traction bronchiectasis. He was referred to a mainland U.S. transplant center and accepted for evaluation.One week before his scheduled flight, he was hospitalized with rapidly worsening dyspnea, cough, and pleuritic chest pain. Computed tomography excluded pulmonary embolism. An extensive infectious evaluation, including bronchoalveolar lavage, was unrevealing. He received high-dose corticosteroids and azathioprine; additional rescue immunosuppression was contraindicated by concern for infection and was unlikely to reverse established fibrosis within the imminent transplant window. Within days, his oxygen requirement escalated from 3 L/min nasal cannula to high-flow nasal cannula at 40 L/min and FiO_2 40%. ECMO bridging was unavailable locally, eliminating a potential strategy for extending his transplant window.Same-week coordination among pulmonary, rheumatology, transplant, case management, out-of-jurisdiction insurance, and air-medical services enabled air-ambulance transfer. He was listed within two days of arrival with a Composite Allocation Score of 85, documented seven days before transplantation, and underwent bilateral lung transplantation seven days after listing. At one month, he demonstrated improving functional status without evidence of acute rejection.DiscussionRP-ILD raises mortality nearly tenfold in anti-MDA5 disease, and ferritin above 1,600 ng/mL identifies the highest-risk phenotype—his value of 4,687 placed him near the ceiling of reported survivable disease at presentation. Two lessons emerge. First, referral must precede refractory failure because candidacy may be rapidly lost to infection, deconditioning, or invasive ventilatory support; his CAS of 85 quantified the narrow therapeutic margin. Second, geography is a modifiable variable. For Puerto Rico's 3.2 million residents, the absence of a transplant center and locally available ECMO-to-transplant pathway means that insurance authorization and air-medical logistics must operate on the disease's timeline, not the system's. Anticipatory referral and pre-negotiated transfer pathways are therapeutic interventions.ConclusionAnti-MDA5 RP-ILD converts a rare autoimmune diagnosis into a fatal pulmonary emergency within weeks. Anticipatory referral and coordinated interjurisdictional transfer enabled bilateral lung transplantation nine days after arrival at the transplant center. The race is against the disease and against the system.

ABSTRACT V-14

The Disappearing Lung Mass: A Heart Failure–Associated Pseudotumor

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Program: San Juan City Hospital Program

ABSTRACT

IntroductionPulmonary pseudotumor, also known as a phantom or vanishing tumor, is a localized accumulation of pleural fluid, typically within an interlobar fissure and most commonly associated with congestive heart failure. Its mass-like appearance may mimic pulmonary malignancy and prompt extensive diagnostic evaluation.Case PresentationA 70-year-old man with coronary artery disease status post percutaneous coronary intervention, hypertension, type 2 diabetes mellitus, dyslipidemia, and heart failure presented with six months of progressive dyspnea on exertion, 18 days of productive cough, subjective fever, and approximately 60 pounds of unintentional weight loss over several months. He denied tobacco use, hemoptysis, and night sweats. Given his constitutional and respiratory symptoms, pulmonary malignancy was a major concern.He was afebrile and hemodynamically stable, with an oxygen saturation of 94% on room air. Noncontrast chest computed tomography (CT) demonstrated a 71 × 39 mm hypodense pseudonodular lesion centered along the right oblique fissure with fluid attenuation (2 Hounsfield units). Additional findings included bilateral pleural and fissural pseudonodular thickening, a small left pleural effusion, right lower-lobe parahilar consolidation with air bronchograms, ground-glass opacities, and septal thickening. The overall presentation raised concern for an underlying pulmonary malignancy complicated by post-obstructive pneumonia, prompting antibiotic therapy and further malignancy evaluation. Bronchoscopy revealed no endobronchial mass or extrinsic compression, while bronchoalveolar lavage and bronchial brushing were unrevealing for malignancy. Left lower-lobe lavage demonstrated acute inflammation. The patient received ceftriaxone and doxycycline for suspected post-obstructive/community-acquired pneumonia, with clinical improvement.Concurrently, pro-B-type natriuretic peptide was markedly elevated at 4,063 pg/mL. Echocardiography demonstrated severe left ventricular systolic dysfunction with an ejection fraction of 30–35% and moderate mitral stenosis. The patient underwent treatment for concomitant heart failure, including intravenous furosemide and optimization of guideline-directed medical therapy.Repeat contrast-enhanced chest CT 11 days later demonstrated near-complete resolution of the 71 × 39 mm right fissural lesion, resolution of the pleural effusion, and marked improvement of the pleural and interstitial abnormalities. Persistent left lower-lobe tree-in-bud opacities and a small subpleural collection suggested a residual inflammatory component.DiscussionThe rapid regression of a large mass-like lesion initially concerning for malignancy, together with its fissural location, fluid attenuation (2 HU), markedly elevated pro-BNP, and severe left ventricular systolic dysfunction, supported the diagnosis of a heart-failure-associated pulmonary pseudotumor. Concurrent pneumonia complicated the radiographic presentation; however, persistence of inflammatory abnormalities despite resolution of the dominant fissural lesion suggests that cardiogenic and infectious processes likely coexisted. Pulmonary pseudotumor should be considered when a mass-like fissural opacity demonstrates fluid attenuation in a patient with evidence of cardiac dysfunction. Recognition of these features and short-interval reassessment following appropriate therapy may help avoid unnecessary invasive oncologic procedures.

ABSTRACT V-15

Atypical Cogan's Syndrome in a Hispanic young male with vitiligo: A case report

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Program: Consortium Hospital Episcopal San Lucas

ABSTRACT

Cogan's syndrome (CS) is a rare disorder of unknown etiology that is theorized to have an autoimmune origin and is also characterized by inflammatory ocular disease and sensorineural hearing loss (SNHL). No specific autoantibodies have been identified, and it remains a diagnosis of exclusion after infectious and other autoimmune causes have been ruled out. Management is usually challenging, requiring high-dose corticosteroids, and for refractory cases, immunomodulatory therapy is added for better outcomes. We present a 21-year-old Hispanic male with a past medical history of vitiligo who presents with a seven-month history of progressive audiovestibular symptoms, including imbalance, dizziness, nausea, tinnitus, and fluctuating SNHL. Initial otologic evaluation demonstrated right-sided SNHL with a 55 dB HL speech recognition threshold (SRT) on the audiometry report, requiring systemic corticosteroids and intratympanic steroid therapy. Despite therapy, right-sided SNHL worsened during corticosteroid tapering, reaching a 70 dB HL SRT and ultimately requiring a hearing aid and another course of high-dose systemic corticosteroids, with mild recovery of symptoms. During this period, the patient also developed ocular symptoms, including severe photophobia and bilateral ocular inflammation. Ophthalmologic evaluation revealed keratoconjunctivitis without evidence of interstitial keratitis on follow-up examinations. Extensive infectious and autoimmune workup was negative, including ANA, ANCA, RF, anti-CCP, anti-HSP70, HLA-B51. In addition, brain MRI and cardiac imaging were also unremarkable. Workup only revealed elevated levels of beta-2 globulin fraction, consistent with an inflammatory process. Seven months after initial presentation, during another corticosteroid taper, he developed worsening right-sided SNHL to 85 dB HL SRT, accompanied by new-onset left-sided hearing loss, marking progression to bilateral audiovestibular involvement. The patient was treated with a short course of high-dose methylprednisolone 500 mg intravenously followed by oral prednisone 30 mg daily, combined with azathioprine 150 mg daily. Resulting in near-complete recovery of left-sided hearing and significant improvement of right-sided hearing. The clinical course and response to immunosuppressive therapy supported a diagnosis of atypical Cogan syndrome. Our case demonstrates the challenges faced in diagnosing atypical CS. The limited awareness of CS and its variable presentation contributes frequently to diagnostic delay. The coexistence with vitiligo, an autoimmune disorder, raises the possibility of a shared immunologic mechanism that has not been previously reported. This case highlights the importance of early recognition and combination therapy of corticosteroids with immunosuppressants for preserving hearing function. A high index of suspicion should always be maintained for early recognition of atypical CS and appropriate multidisciplinary team evaluation.

ABSTRACT V-16

Beyond the Myxoma: When Preoperative Evaluation Changes the Surgical Plan

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ABSTRACT

INTRODUCTIONCardiac myxomas are rare primary cardiac tumors with potentially serious obstructive and embolic complications. Although surgical excision is definitive, evaluation should extend beyond characterization of the intracardiac mass, particularly in older patients with cardiovascular risk factors. We present an elderly patient with a left atrial myxoma in whom preoperative evaluation identified significant coronary artery disease, altering the surgical strategy, followed by a clinically significant postoperative conduction disturbance.CASE DESCRIPTIONAn 86-year-old woman with hypertension, type 2 diabetes mellitus, hypothyroidism, and a 10-pack-year smoking history was referred for evaluation of a newly identified left atrial mass on transthoracic echocardiography, initially concerning for myxoma versus thrombus. She reported intermittent exertional chest tightness. Transthoracic echocardiography demonstrated a left atrial mass attached to the interatrial septum, mild global hypokinesis, and a left ventricular ejection fraction of 40–45%. Transesophageal echocardiography further characterized a mobile, irregular mass arising from the interatrial septum, morphologically consistent with atrial myxoma, without mitral inflow obstruction or left atrial appendage thrombus. Given her age, cardiovascular risk factors, exertional symptoms, and anticipated cardiac surgery, coronary angiography was performed and demonstrated 70–80% stenosis of the mid-left anterior descending artery. She underwent left atrial mass resection with atrial septal repair and concomitant single vessel coronary artery bypass grafting using the left internal mammary artery to the left anterior descending artery. Histopathology confirmed cardiac myxoma. Postoperatively, temporary epicardial pacing was required. Following discontinuation of pacing, she developed symptomatic junctional bradycardia at <40 bpm accompanied by nausea, dizziness, and malaise. Persistent symptomatic bradyarrhythmia ultimately required permanent dual-chamber pacemaker implantation. DISCUSSIONThis case illustrates two important considerations in the management of cardiac myxoma. First, evaluation should not end with characterization of the intracardiac mass. In older patients with cardiovascular risk factors or ischemic symptoms, assessment for concomitant coronary artery disease may substantially alter operative planning. In this patient, detection of significant LAD disease allowed tumor resection and coronary revascularization to be performed during a single operation. Second, postoperative rhythm surveillance is important after myxoma resection. Surgical manipulation of atrial and septal tissue, local inflammation, perioperative ischemia, cardiopulmonary bypass, and underlying age-related conduction disease may contribute to postoperative conduction abnormalities. Although many are transient, persistent symptomatic bradyarrhythmias may require permanent pacing. This case emphasizes that management of cardiac myxoma requires a comprehensive approach extending from multimodality imaging and ischemic evaluation to postoperative rhythm surveillance. In appropriately selected patients, this strategy may identify clinically important concomitant disease, modify surgical management, and facilitate early recognition of postoperative complications.

ABSTRACT V-17

The Bleeding Behind the Hernia: An Overlooked Cause of Recurrent Hospitalization

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ABSTRACT

Background: Cameron lesions are rare linear gastric erosions or ulcerations associated with large hiatal hernias and represent an underrecognized cause of gastrointestinal bleeding. Hiatal hernias are identified in approximately 0.8–2.9% of patients undergoing upper gastrointestinal endoscopy, while Cameron lesions are believed to be significantly underdiagnosed due to their small size and difficult visualization. These lesions are classically associated with chronic occult blood loss and iron deficiency anemia; however, atypical presentations can result in delayed diagnosis and significant morbidity. Case Presentation: An 82-year-old female with a medical history of hypertension and rheumatoid arthritis presented with recurrent melena, coffee-ground emesis, and generalized weakness. Initial laboratory evaluation demonstrated profound anemia with hemoglobin levels as low as 4.0 g/dL, requiring multiple packed red blood cell transfusions. Initial EGD failed to identify a bleeding source. Despite supportive management, the patient experienced recurrent gastrointestinal bleeding requiring repeated hospitalizations, blood transfusions, and extensive diagnostic evaluation, including colonoscopy and computed tomography angiography, both of which were unrevealing. Given persistent overt bleeding and ongoing transfusion requirements, repeat EGD was performed and ultimately revealed Cameron ulcers within a previously unrecognized hiatal hernia. The patient was treated with high-dose proton pump inhibitor therapy, transfusion support, and outpatient referral for surgical evaluation. Conclusions: This case highlights an uncommon and potentially life-threatening presentation of Cameron lesions causing recurrent overt gastrointestinal bleeding and severe transfusion-dependent anemia in the absence of both a known hiatal hernia and the classic finding of iron deficiency anemia. Unlike the typical presentation, she had no evidence of iron deficiency anemia and no previously documented hiatal hernia. The absence of iron deficiency anemia was likely related to an acute overt bleeding pattern rather than the gradual chronic blood loss usually associated with Cameron lesions, with transfusion requirements potentially affecting iron parameters. Clinicians should maintain a high index of suspicion for Cameron lesions with recurrent gastrointestinal bleeding, even when traditional clinical features are absent. Early recognition may reduce repeated hospitalizations, invasive procedures, transfusion requirements, and overall healthcare utilization while improving patient outcomes.

ABSTRACT V-18

Silent but Catastrophic: Extensive Stanford Type A (DeBakey I) Aortic Dissection with Fatal Postoperative Complications

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ABSTRACT

Aortic Dissection is defined as a tear in the innermost layer of the aortic wall and is classified as Stanford type A or type B. Type A involves the ascending aorta regardless of the site of the primary intimal tear, whereas type B originates distal to the left subclavian artery. Although uncommon (approximately 5–6 per 100,000 ED visits), aortic dissection is a life-threatening condition requiring rapid diagnosis and intervention to improve survival.We present the case of a 70-year-old male with a history of hypertension who presented to the emergency department after his cardiologist reviewed a chest CTA concerning for aortic dissection. The patient was completely asymptomatic, denying chest pain, back pain, trauma, palpitations, dyspnea, nausea, vomiting, dizziness, headache, syncope, or other acute complaints. One week earlier, he experienced hypogastric pain and underwent CTA at an outside hospital because of concern for mesenteric ischemia in the setting of elevated lactate; however, no additional management was pursued. He denied tobacco, alcohol, or illicit drug use.Repeat CTA at our institution confirmed a Stanford type A aortic dissection with the intimal flap originating in the ascending aorta near the brachiocephalic artery and extending through the descending thoracic and abdominal aorta into the iliac arteries, right common femoral artery, and right superficial femoral artery. The patient underwent emergent surgical repair requiring more than eight hours of operative time and was initially stabilized postoperatively in critical condition.Several hours later, he developed acute cardiac tamponade followed by cardiac arrest before chest reopening. Return of spontaneous circulation was achieved, and the bleeding source was identified and corrected. Despite aggressive resuscitation and surgical intervention, he subsequently developed disseminated intravascular coagulation (DIC) and hemorrhagic shock, ultimately expiring in the ICU.This case highlights the catastrophic and unpredictable nature of acute Stanford type A aortic dissection, emphasizing that it may remain asymptomatic despite extensive disease. Early recognition, prompt imaging, and emergent surgical repair remain essential, although mortality remains high due to complications such as cardiac tamponade, massive hemorrhage, and DIC. If action had been taken earlier with the original CTA, the outcome might have been different.

ABSTRACT V-19

When Myelin Is Not the Culprit: Fulminant Anti-GD1a–Positive Acute Motor Axonal Neuropathy

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Program: Universidad Central del Caribe Program

ABSTRACT

IntroductionAcute motor axonal neuropathy (AMAN) is an uncommon Guillain-Barré syndrome (GBS) variant characterized by immune-mediated injury of the motor axolemma and nodes of Ranvier, with the potential for rapid and severe progression, making early diagnosis critical. AMAN accounts for approximately 3–5% of GBS cases in Western populations, although higher proportions have been reported in parts of Asia and Latin America. Because early cerebrospinal fluid (CSF) studies may be nondiagnostic, timely recognition depends on identifying a motor-selective phenotype before respiratory and autonomic failure emerge. We describe fulminant anti-GD1a–positive AMAN evolving from isolated foot drop to ventilator-dependent quadriparesis, with eventual restoration of neurologic function. Case PresentationA 35-year-old man with well-controlled HIV on antiretroviral therapy presented with acute left foot drop one week after a self-limited diarrheal illness. Within 48 hours, weakness progressed to both lower extremities and subsequently the upper extremities, while sensation and proprioception remained preserved and deep-tendon reflexes progressively diminished. Neurology was consulted for evaluation of rapidly progressive ascending flaccid paralysis. Brain and cervical-spine MRI and early lumbar puncture were unrevealing.During hospitalization, the patient developed flaccid quadriparesis, bilateral foot drop, ineffective cough, secretion retention, dysautonomia, and respiratory muscle failure requiring intubation, mechanical ventilation, and ICU-level care. IVIG was administered for five days. As profound motor dysfunction persisted with sensory sparing, progressive reflex loss, and respiratory/bulbar involvement, Neurology suspected an axonal-motor GBS variant. Targeted ganglioside testing subsequently demonstrated markedly elevated anti-GD1a IgG, providing strong serologic support for AMAN. Prolonged ventilatory dependence ultimately required tracheostomy and gastrostomy. Following multidisciplinary rehabilitation, the patient was weaned from mechanical ventilation, underwent tracheostomy and gastrostomy removal, resumed oral intake, and at one-year follow-up had achieved complete functional recovery.DiscussionThis case highlights AMAN as a diagnostic challenge when rapidly progressive neurologic deterioration occurs despite initially unrevealing studies, making recognition of the clinical phenotype critical for early identification. Early phenotype recognition can accelerate immunotherapy, anticipate respiratory and autonomic deterioration, and preserve appropriate expectations for meaningful neurologic recovery even in seemingly devastating disease. Although electrodiagnostic confirmation was unavailable, clinic-serologic concordance strongly supported AMAN.Most importantly, catastrophic axonal dysfunction does not necessarily predict irreversible disability: despite ventilator-dependent quadriparesis requiring tracheostomy and gastrostomy, this patient ultimately achieved complete functional recovery.

ABSTRACT V-20

Necrotic liver lesions and sepsis: a diagnostic pitfall revealing poorly differentiated neuroendocrine carcinoma

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ABSTRACT

Background: Poorly differentiated neuroendocrine carcinomas (PDNECs) are rare, aggressive malignancies with high proliferative indices and dismal prognosis. Extensive hepatic involvement with central tumor necrosis may closely mimic pyogenic liver abscess on imaging and clinical presentation, leading to delayed oncologic diagnosis. This case highlights the diagnostic challenge of distinguishing PDNEC from hepatic abscess and emphasizes the importance of early tissue sampling when clinical features are atypical for infection. Case presentation: A 67-year-old male with chronic tobacco use, substance use disorder, and prior treated tuberculosis presented with two weeks of abdominal pain, fever, nausea, anorexia, and rapid 30-pound weight loss. Examination revealed cachexia and massive tender hepatomegaly. Laboratory studies demonstrated leukocytosis, abnormal liver function tests, and hyperlactatemia. Abdominal computed tomography showed marked hepatomegaly with multiple centrally necrotic hepatic lesions, necrotic porta hepatis lymphadenopathy, and right portal vein thrombosis. Chest imaging revealed a calcified spiculated right upper lobe lesion. The patient was treated empirically for presumed pyogenic liver abscess with broad-spectrum antibiotics; however, blood cultures remained negative and symptoms persisted. Progressive abdominal pain prompted exploratory laparotomy, which revealed extensive hepatic malignancy with ascites and omental metastases without abscess. Pathology demonstrated a poorly differentiated carcinoma with neuroendocrine features. Immunohistochemistry was positive for cytokeratins AE1/AE3, 7, 19, and 20, SATB2, CD56, and INSM1, with Ki-67 approaching 100%, and negative for synaptophysin, TTF-1, HepPar-1, arginase-1, glypican-3, CDX2, and NKX3.1, supporting a PDNEC of probable gastrointestinal or pancreatobiliary origin. The patient's rapidly declining performance status precluded systemic chemotherapy. The course was complicated by malignant ascites, hepatic encephalopathy, acute kidney injury, and secondary bacterial peritonitis with multidrug-resistant Acinetobacter baumannii. Despite aggressive supportive care, the patient developed multiorgan failure and expired during hospitalization. Conclusions: This case underscores that centrally necrotic hepatic PDNECs can masquerade as pyogenic liver abscess. Portal vein thrombosis, persistently negative cultures, significant weight loss, and failure to improve with antimicrobial therapy should prompt early tissue diagnosis. Comprehensive immunohistochemical profiling is essential for confirming neuroendocrine differentiation and determining site of origin in these rare, rapidly fatal tumors.

ABSTRACT V-21

No Worm Left Unturned: Occult Strongyloidiasis Driving Salmonella Group C Bacteremia

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ABSTRACT

Introduction:Nontyphoidal Salmonella (NTS) bacteremia in an immunocompetent adult should prompt a search for a predisposing source. Chronic strongyloidiasis is an underrecognized facilitator of invasive enteric bacterial infection: enteric bacteria can be carried across the mucosa on migrating filariform larvae or escape through larva-induced ulcers, and even chronic infection without hyperinfection has been associated with extra-intestinal bacterial infection of enteric origin. We describe disseminated Salmonella serogroup C bacteremia that unmasked occult strongyloidiasis in an immunocompetent host.Case Description:A 75-year-old male veteran with atrial fibrillation, hypertension, type 2 diabetes, and prior cerebrovascular accidents presented to the emergency department with watery nonbloody diarrhea, vomiting, fever, and weakness. Presumed viral gastroenteritis was diagnosed and he was discharged. He returned two days later with unrelenting diarrhea and was admitted. Severe hypokalemia (1.8 mEq/L) prompted ICU transfer for cardiac monitoring and central venous potassium repletion.A history of consuming home-raised chicken raised suspicion for salmonellosis. Blood cultures drawn at the initial visit grew multidrug-susceptible Salmonella serogroup C, subsequently isolated from stool and urine, confirming disseminated infection. CT demonstrated biliary ductal dilatation. Stool ova-and-parasite examination identified Strongyloides species. Workup for immunocompromise—HIV, HTLV-I/II, complement levels, and immunoglobulins—was unremarkable, and there was no clinical or laboratory evidence of hyperinfection.He received two courses of ivermectin and four weeks of intravenous ceftriaxone with full clinical recovery. Follow-up stool studies cleared Strongyloides, and MRCP confirmed resolution of the biliary dilatation.Discussion:This case illustrates that chronic strongyloidiasis can predispose to disseminated NTS bacteremia in the absence of overt immunosuppression or hyperinfection. Filariform larvae transport enteric organisms into the bloodstream and disrupt the mucosal barrier, and chronic, non-hyperproliferative infection alone has been linked to invasive enteric bacterial infection, supporting screening in at-risk patients presenting with such infections. Helminth-driven shifts in the gut microbiome offer a plausible additional mechanism for impaired colonization resistance.Serogroup identification carried prognostic weight. Group C Salmonella was an independent predictor of endovascular infection among non-AIDS patients with nontyphoidal salmonellosis, and invasive NTS disease overall carries substantial risk of life-threatening complications, including mycotic aneurysm and endocarditis, with case-fatality near 15%. These data justify the extended intravenous course and heightened surveillance for endovascular seeding pursued here.Ivermectin remains first-line for strongyloidiasis, with documented cure by follow-up stool studies.ConclusionOccult strongyloidiasis can drive disseminated NTS bacteremia even in immunocompetent hosts. Clinicians should screen for Strongyloides in patients with invasive enteric bacterial infection who have epidemiologic risk factors, treat both pathogens concurrently, and remain vigilant for endovascular complications when serogroup C is isolated.

ABSTRACT V-22

Ventricular Fibrillation and Cardiac Arrest From Pre-Excited Atrial Fibrillation in Pregnancy: A Rare Presentation of Wolff-Parkinson-White Syndrome

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ABSTRACT

Wolff-Parkinson-White Syndrome affects 0.1-0.3% of the general population and may rarely present with pre-excited atrial fibrillation, an arrhythmia characterized by rapid conduction through an accessory pathway that may degenerate into ventricular fibrillation and sudden cardiac death. Although pregnancy may increase arrhythmia burden, ventricular fibrillation secondary to pre-excited atrial fibrillation during pregnancy remains exceptionally rare and represents a cardio-obstetric emergency.We describe the case of a 21-year-old woman at 36 weeks gestation who presented with palpitations, chest pain, and dyspnea. Electrocardiography demonstrated an irregular wide-complex tachyarrhythmia consistent with pre-excited atrial fibrillation. Despite initial hemodynamic stability, the patient rapidly deteriorated and developed cardiac arrest. Advanced cardiovascular life support was initiated. During resuscitation, the rhythm degenerated into ventricular fibrillation requiring defibrillation, followed by unstable supraventricular tachycardia requiring synchronized cardioversion with successful return of spontaneous circulation. Echocardiography demonstrated preserved left ventricular systolic function without structural abnormalities. Procainamide infusion was initiated while avoiding atrioventricular nodal blocking agents because of the risk of enhanced accessory pathway conduction. Following stabilization, the patient underwent cesarean delivery with favorable maternal and fetal outcomes. She was subsequently transitioned to flecainide and maintained sinus rhythm. At outpatient follow-up after delivery, electrophysiology study identified a left posterolateral accessory pathway responsible for atrioventricular reentrant tachycardia. Successful radiofrequency ablation of the accessory pathway was achieved.Pre-excited atrial fibrillation in pregnancy is a rare cardio-obstetric emergency with the potential for rapid degeneration into ventricular fibrillation and sudden cardiac death. Early diagnosis, appropriate therapy, and multidisciplinary management are critical for favorable outcomes. This case highlights the importance of prompt recognition and treatment of an accessory pathway with rapid ventricular response due to its life threatening implications as shown in our case, thus eliminating the arrhythmogenic substrate and reducing the risk of recurrent malignant arrhythmias and sudden cardiac death.

ABSTRACT V-23

Beyond the Rash: Fatal Toxic Epidermal Necrolysis Following Enfortumab Vedotin and Pembrolizumab

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ABSTRACT

BackgroundEnfortumab vedotin (EV) plus pembrolizumab is a standard first-line therapy for locally advanced or metastatic urothelial carcinoma. Although associated with cutaneous toxicity, Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are rare but potentially life-threatening complications. We report a fatal case of rapidly progressive TEN associated with EV and pembrolizumab therapy, characterized by a fulminant course and progression despite prompt discontinuation of therapy and treatment with systemic corticosteroids, intravenous immunoglobulin, and etanercept.Case PresentationA 71-year-old man with hypertension, diabetes mellitus, dyslipidemia, chronic kidney disease, and advanced squamous cell carcinoma of the bladder receiving treatment with enfortumab vedotin and pembrolizumab presented to the emergency department four days after his most recent infusion with a progressive diffuse morbilliform rash. The rash was associated with severe pruritus, pain, and mucosal symptoms, including oral swelling, dry oral mucosa and dysphagia. He was initially managed as severe chemotherapy related cutaneous toxicity with grade 3 mucositis and stomatitis.Despite discontinuation of both agents and initiation of systemic corticosteroids, the rash rapidly progressed to widespread epidermal sloughing involving approximately 30% of total body surface area, consistent with SJS/TEN overlap. At the time of diagnosis, the patient had a SCORTEN of 7, corresponding to an estimated mortality exceeding 95%. Following diagnosis of SJS/TEN, he received intravenous immunoglobulin and etanercept; however, the disease continued to progress.Cutaneous involvement expanded to approximately 40% TBSA with persistent desquamation and development of multiorgan dysfunction, including acute kidney injury, leukopenia, and altered mental status. Despite maximal immunomodulatory and supportive therapy, the patient's clinical condition continued to deteriorate, ultimately resulting in a fatal outcome.ConclusionThis case illustrates fulminant, treatment-refractory SJS/TEN in a patient receiving enfortumab vedotin and pembrolizumab. Despite early discontinuation and aggressive therapy with systemic corticosteroids, intravenous immunoglobulin, and TNF-α inhibition, the patient developed extensive epidermal detachment with a maximal SCORTEN score and rapid clinical deterioration. EV plus pembrolizumab associated with SJS/TEN may be rapidly fatal despite prompt drug discontinuation and immunosuppressive therapy.

ABSTRACT V-24

Brown Tumor Mimicking Metastatic Bone Disease in Severe Primary Hyperparathyroidism

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ABSTRACT

Parathyroid carcinoma is a rare cause of primary hyperparathyroidism. Severe hypercalcemia, markedly elevated parathyroid hormone (PTH), renal dysfunction, and skeletal disease should raise concern for malignancy. Brown tumors are osteolytic manifestations of prolonged hyperparathyroidism that may mimic metastatic disease or multiple myeloma. A 62-year-old man with hypertension and prior acute kidney injury presented with progressive generalized bone pain, weakness, and fatigue. Initial studies demonstrated calcium 18.03 mg/dL, creatinine 3.18 mg/dL, and alkaline phosphatase 163 U/L. Computed tomography revealed multiple lytic lesions of the lumbar spine, right iliac bone, and right acetabulum, concerning for metastatic disease or multiple myeloma. MRI confirmed a 2.4 cm L4 vertebral lesion. Myeloma evaluation, including serum and urine protein electrophoresis, immunoglobulins, and serum free light-chain ratio, was unrevealing. Further testing showed intact PTH 1,110.3 pg/mL, 24-hour urine calcium 330 mg, low-normal phosphorus, and normal 25-hydroxyvitamin D and 1,25-dihydroxyvitamin D levels. Ultrasound identified a 4.59 cm hypoechoic mass posterior to the left thyroid lobe, and sestamibi scintigraphy demonstrated a hyperfunctioning left parathyroid lesion. Right iliac biopsy showed bone and fibroconnective tissue with reparative changes, favoring a brown tumor. Hypercalcemia and renal function improved with intravenous hydration, pamidronate, calcitonin, and cinacalcet. The patient underwent left hemithyroidectomy and parathyroidectomy with intraoperative PTH monitoring. Final pathology confirmed parathyroid carcinoma with direct thyroid parenchymal invasion and lymphovascular invasion. This case highlights parathyroid carcinoma presenting as profound PTH-dependent hypercalcemia with multifocal brown tumors mimicking disseminated malignancy. In patients with lytic lesions and severe hypercalcemia, early PTH measurement is essential after plasma cell dyscrasia is excluded. Recognition of this biochemical pattern enables prompt localization, definitive surgery, and appropriate oncologic follow-up.

ABSTRACT V-25

When Hemolysis Outruns the Marrow: Severe DAT-Positive AIHA With Reticulocytopenia

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ABSTRACT

Autoimmune hemolytic anemia (AIHA) is characterized by immune-mediated red blood cell destruction. Reticulocytosis is generally expected during active hemolysis; however, reticulocytopenia may indicate inadequate marrow compensation and can contribute to profound anemia, hemoglobin instability, and transfusion dependence. We describe severe direct antiglobulin testing (DAT)-positive AIHA with marked reticulocytopenia in a young adult with hidrotic ectodermal dysplasia.A 22-year-old man with hidrotic ectodermal dysplasia presented with three days of fever, chills, malaise, night sweats, headache, dizziness, weakness, and dark urine. He denied cough, dyspnea, chest pain, palpitations, abdominal pain, gastrointestinal symptoms, dysuria, recent antibiotic exposure, tobacco use, and illicit drug use. He took no home medications. He was hemodynamically stable. Examination demonstrated facial flushing, hypodontia, and splenomegaly without lymphadenopathy or scleral icterus.Initial laboratory testing showed normocytic anemia with hemoglobin 7.9 g/dL, hematocrit 22.3%, and mean corpuscular volume 84.8 fL. Hemoglobin decreased to 6.5 g/dL on hospital day one, with a nadir of 6.4 g/dL. Hemolysis studies demonstrated elevated lactate dehydrogenase, initially 347 U/L and later 460 U/L; total bilirubin 1.9–2.0 mg/dL; and decreased haptoglobin. Despite severe anemia and biochemical evidence of hemolysis, the reticulocyte count was inappropriately low at 0.2%. Peripheral smear demonstrated left shift, atypical lymphocytes, and monocytes without schistocytes. Direct antiglobulin testing was positive for anti-human globulin, IgG, and C3d, supporting immune-mediated hemolysis with IgG and complement deposition. Erythropoietin was elevated at 126.89 mIU/mL. Vitamin B12, folate, and thyroid-stimulating hormone were normal.Evaluation for secondary causes was unrevealing. HIV, Epstein-Barr virus, cytomegalovirus, hepatitis studies, parvovirus B19, antinuclear antibody, double-stranded DNA, lupus panel, Sjögren antibodies, Scl-70, Smith antibody, and Smith/ribonucleoprotein antibody were negative. Blood and urine cultures showed no growth, and chest radiography showed no acute cardiopulmonary process. Immunoglobulin testing demonstrated elevated IgE at 5,511.5, IgG at 3,312, and IgA greater than 540, with normal IgM.Management required close coordination with the blood bank because compatible blood products were obtained through the Red Cross. The patient received six units of leukocyte-reduced packed red blood cells and was maintained in a warm environment. He was initially treated with IV Solumedrol 80 mg every eight hours, followed by prednisone 80 mg daily. Because of persistent hemoglobin instability despite transfusions and corticosteroids, rituximab was initiated after infectious causes were excluded. He received two inpatient doses and completed two outpatient doses.Before discharge, hemoglobin improved to 8.5 g/dL, reticulocyte count increased from 0.2% to 12.05%, and lactate dehydrogenase decreased to 245 U/L. At outpatient follow-up, hemoglobin increased to 11.9 g/dL and normalized to 13.6 g/dL. At the most recent follow-up, the patient was clinically well, with no documented recurrence of hemolysis.This case highlights severe DAT-positive AIHA with reticulocytopenia and substantial transfusion requirements. Prompt recognition of immune hemolysis, coordinated transfusion support, corticosteroids, rituximab, and continued evaluation for secondary causes were associated with sustained hematologic recovery without recurrence at the latest follow-up.

ABSTRACT V-26

Saving Sight and Skin: Integrated Multidisciplinary Care for Pembrolizumab-Induced SJS/TEN

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ABSTRACT

Immune checkpoint inhibitors (ICIs) have transformed oncologic therapy but introduced a spectrum of immune-related adverse events demanding coordinated, multidisciplinary internal medicine management. We present a case illustrating the diagnostic complexity and critical role of an integrated clinical approach when severe cutaneous toxicity arises in the oncology setting.A 59-year-old woman with triple-negative breast cancer, type 2 diabetes mellitus, chronic kidney disease, hypertension, and hypothyroidism developed a rapidly progressive mucocutaneous eruption following pembrolizumab-based therapy. She presented with painful oral and periocular lesions, cutaneous blistering and erosions involving approximately 20% body surface area, scattered tense and flaccid bullae, atypical targetoid lesions, and positive Nikolsky and Asboe-Hansen signs. Severe bilateral ocular involvement included conjunctival membranes, ectropion, lagophthalmos, and near-complete corneal epithelial defects. Histopathology demonstrated epithelial necrosis with re-epithelialization consistent with erythema multiforme/Stevens-Johnson syndrome (SJS). Direct immunofluorescence remained pending to exclude paraneoplastic pemphigus (PNP).The central diagnostic challenge was distinguishing ICI-associated SJS/toxic epidermal necrolysis (TEN) from PNP, entities with overlapping mucocutaneous features but distinct pathophysiology and management. A clinically striking feature was the disproportionately severe ocular and oral mucosal involvement relative to cutaneous disease, raising further diagnostic uncertainty. Pembrolizumab was identified as the most likely causative agent, though cyclophosphamide and doxorubicin exposure could not be fully excluded.Management required active collaboration across internal medicine, oncology, dermatology, ophthalmology, and nutrition. Bilateral amniotic membrane transplantation was performed to preserve ocular surface integrity. Systemic corticosteroids and multidisciplinary supportive care were initiated concurrently.This case highlights the diagnostic dilemma between SJS/TEN and PNP in patients with active malignancy and emphasizes that severe ocular and oral involvement may occur despite relatively limited cutaneous disease.

ABSTRACT V-27

Weakness, Hypercalcemia, and Renal Failure: A Diagnostic Approach To Non-PTH-Mediated Hypercalcemia

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ABSTRACT

Introduction:The most common cause of inpatient hypercalcemia is malignancy. Hypercalcemia with renal dysfunction, anemia, and back pain raises concern for multiple myeloma. However, these findings are nonspecific, and premature diagnostic closure may delay recognition of other causes of hypercalcemia.Case Description:A 62-year-old man with type 2 diabetes mellitus, hypertension, heart failure, and chronic kidney disease, (baseline creatinine of 1.84 mg/dL), presented with generalized weakness, malaise, dyspnea, orthopnea, dizziness, and imbalance of one week duration. His history was notable for poor medication adherence, recurrent emergency department visits for low back pain, and daily use of up to six Alka-Seltzer tablets for reflux. On admission, blood pressure was 187/92 mmHg, and pulmonary examination revealed bilateral rhonchi. Laboratory evaluation demonstrated acute kidney injury with creatinine 4.61 mg/dL, eGFR 14 mL/min/1.73 m^2, calcium level 13.5 mg/dL (corrected 13.3 mg/dL), phosphorus 5.68 mg/dL, and normocytic normochromic anemia with hemoglobin 11.6 g/dL. Urinalysis showed proteinuria and glucosuria. Renal ultrasonography demonstrated bilateral nephrolithiasis without hydronephrosis. Chest radiography revealed a pulmonary nodule.Hypercalcemia, renal failure, anemia, and back pain mimicked the CRAB features of multiple myeloma prompting evaluation for a plasma cell disorder. A bicarbonate level of 22 mmol/L ruled out calcium-alkali syndrome despite chronic antacid use. Given the pulmonary findings, evaluation for an occult malignancy was also pursued. Management included cautious isotonic saline hydration at 100–150 mL/hour with strict intake and output monitoring given underlying heart failure. Despite hydration, creatinine peaked at 5.29 mg/dL with rising pro-BNP and pulmonary congestion. Workup demonstrated PTH-independent hypercalcemia with suppressed intact PTH, elevated ionized calcium, low 25-OH vitamin D, and upper-limit 1,25-dihydroxyvitamin D. Urine calcium/creatinine ratio of 0.22 excluded familial hypocalciuric hypercalcemia. Serum Protein Electrophoresis and Urine Protein Electrophoresis were negative and a skeletal survey showed no lytic lesions, excluding multiple myeloma. The diagnostic focus shifted toward a possible paraneoplastic malignancy causing hypercalcemia. Chest CT revealed mediastinal, hilar, and bilateral axillary lymphadenopathy, and solid left upper lobe pulmonary nodules (up to 15mm). Suppressed PTH with high-normal calcitriol raised the suspicion for a dual PTH-rp and Vitamin D-mediated process, as seen with lymphoma, renal cell carcinoma, or certain lung malignancies.Discussion:This case illustrates the broad differential diagnosis of hypercalcemia complicated by acute kidney injury. Although the presence of hypercalcemia, renal dysfunction, anemia, and back pain appropriately raises concern for multiple myeloma, this presentation should guide further investigation rather than establish the diagnosis. Evaluation should include PTH status, monoclonal protein studies, medication exposure, and investigation of imaging abnormalities suggesting occult malignancy. It also highlights the therapeutic challenge of volume resuscitation for hypercalcemia and renal hypoperfusion in patients at increased risk for heart failure exacerbation.

ABSTRACT V-28

Diagnostic Challenge of Scintigraphy-Positive ATTR Cardiac Amyloidosis with Concurrent Monoclonal Gammopathy Suggesting Possible AL Amyloidosis

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ABSTRACT

Introduction:Cardiac amyloidosis is an infiltrative cardiomyopathy caused by extracellular deposition of misfolded proteins, most commonly transthyretin (ATTR) or immunoglobulin light chain (AL). It can cause restrictive cardiomyopathy, heart failure, arrhythmias, thromboembolic events, renal dysfunction, and elevated cardiac biomarkers. Recognition is critical because ATTR and AL amyloidosis require different diagnostic and therapeutic approaches.[1][2] This case illustrates the diagnostic complexity of suspected systemic amyloidosis in a critically ill patient with multiorgan failure, myocardial injury, and ischemic stroke.Case Presentation: A 66-year-old woman with hypertension, severe persistent asthma, and insulin-dependent type 2 diabetes mellitus presented with an acute asthma exacerbation and progressive dyspnea. She developed acute hypoxemic respiratory failure requiring BiPAP, severe acute kidney injury with creatinine 8.9 mg/dL and blood urea nitrogen 120 mg/dL, oliguria, hyperkalemia of 6.7 mmol/L, metabolic acidosis with pH 7.20 and bicarbonate 11.5 mmol/L, lactic acidosis of 6.57 mmol/L, marked anasarca, and pulmonary edema. Additional abnormalities included hyperglycemia of 341 mg/dL with proteinuria, normocytic anemia, and non-ST-elevation myocardial infarction with troponin 1,810 without ST-segment elevation.She received guideline directed medical therapy, consequently prior to management due to refractory volume overload, uremia, hyperkalemia, acidosis, and oliguria, a femoral dialysis catheter was placed and emergent hemodialysis initiated. After dialysis, creatinine improved to 5.84 mg/dL, potassium to 4.4 mmol/L, bicarbonate to 20.1 mmol/L, and blood urea nitrogen to 76 mg/dL. However, renal recovery did not occur, and she ultimately underwent four hemodialysis sessions.Her hospitalization was complicated by acute aphasia and dense left hemiparesis. Non-contrast head computed tomography demonstrated a hyperdense right middle cerebral artery sign, right middle cerebral artery territory ischemic infarction, and cerebral sulcal effacement without hemorrhage. Thrombolysis was withheld because of active bleeding from the right internal jugular insertion site, anticoagulation administration, severe uremia, and concern for platelet dysfunction. She was intubated for airway protection and subsequently managed with mechanical ventilation via tracheostomy.Further evaluation showed antinuclear antibody positivity with a speckled pattern at 1:640, negative anti-double-stranded DNA antibody, positive M-spike, elevated free kappa and lambda light chains, and beta-2 microglobulin 15.86. A cardiac amyloid scan was positive for ATTR cardiac amyloidosis. Her later course included severe thrombocytopenia with platelets 26, persistent normocytic anemia, pulmonary vascular congestion, bibasilar atelectasis, and poor neurologic recovery. Kidney biopsy was planned when clinically feasible to evaluate suspected amyloidosis or autoimmune nephropathy, however patient was clinically unstable.Discussion: This case illustrates the diagnostic complexity of suspected systemic amyloidosis presenting with multiorgan dysfunction. A positive cardiac scintigraphy scan strongly supports ATTR cardiac amyloidosis in the appropriate clinical setting; however, the coexistence of a monoclonal gammopathy and elevated free light chains prevents ATTR from being diagnosed noninvasively and necessitates exclusion of AL amyloidosis. In such patients, tissue biopsy with definitive amyloid typing is critical because misclassification may result in inappropriate or delayed disease-specific therapy. When clinically feasible, biopsy of an involved organ should be pursued to establish the amyloid subtype and guide management. Early collaboration among cardiology, nephrology, and hematology is essential, particularly in patients presenting with cardiac and renal dysfunction suggestive of systemic amyloidosis.

ABSTRACT V-29

Unmasking a Rare Autoimmune Disease of the Biliary System: IgG4-Related Sclerosing Cholangitis

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ABSTRACT

Immunoglobulin G4-related sclerosing cholangitis (IgG4-SC) is a rare autoimmune disease of the biliary tree, with an estimated prevalence of approximately 0.5 per 100,000, occurring predominantly in middle-aged and older men. It is characterized by biliary strictures resulting from IgG4-predominant subepithelial lymphoplasmacytic infiltration of the intrahepatic and extrahepatic bile ducts, often accompanied by storiform fibrosis and obliterative phlebitis on histology. IgG4-SC frequently mimics cholangiocarcinoma radiographically and is often confused with primary sclerosing cholangitis (PSC), though it is distinguished by elevated serum IgG4 levels, frequent concomitant autoimmune pancreatitis, and marked responsiveness to corticosteroid therapy. Misdiagnosis can lead patients toward unnecessary surgical evaluation. We present a case of a patient who required multiple hospital admissions and extensive gastroenterological workup to establish the correct diagnosis.A 69-year-old male with a history of hypertension presented to the emergency department with right upper quadrant abdominal pain shortly after discharge from another institution, where he had been diagnosed with acute cholangitis and undergone biliary stent placement for choledocholithiasis. On arrival, laboratory findings were notable for AST 44 U/L, ALT 74 U/L, and ALP 393 U/L, with a benign abdominal examination aside from right upper quadrant tenderness. Non-contrast abdominal CT demonstrated pericholecystic fluid and mild intrahepatic ductal dilation, prompting further evaluation with MRCP, which revealed nodular soft tissue foci within the extrahepatic bile ducts with moderate intrahepatic and mild extrahepatic ductal dilation, findings concerning for cholangiocarcinoma. The patient subsequently underwent ERCP, which showed post-sphincterotomy changes; cholangiography demonstrated an irregular, non-dilated extrahepatic bile duct with focal stenosis, without a discrete mass lesion. Brushings were negative for malignant cells. Given the diagnostic uncertainty, serum IgG4, antinuclear antibody (ANA), and antimitochondrial antibody (AMA) were obtained to evaluate for an underlying autoimmune biliary process. Results showed an elevated IgG4 level of 335 mg/dL, with positive ANA (1:320) and AMA (27.7 U/mL), consistent with a diagnosis of IgG4-SC. The patient was started on corticosteroid therapy, with subsequent clinical improvement, and completed an uncomplicated hospital course with a confirmed diagnosis and outpatient follow-up arranged for steroid tapering.IgG4-SC is a rare autoimmune disease that is frequently misdiagnosed and often confused with cholangiocarcinoma. This distinction is clinically critical, as the two entities are managed very differently: cholangiocarcinoma is treated with surgical resection, chemotherapy, or radiation depending on staging, whereas IgG4-SC is managed medically with corticosteroids as first-line therapy, often with dramatic clinical improvement. This case illustrates the diagnostic difficulty posed by the overlapping features of these two conditions, as our patient required multiple ERCP procedures and serologic workup before a definitive diagnosis could be established. Clinicians should maintain a high index of suspicion for IgG4-SC in patients presenting with biliary strictures of uncertain etiology, particularly when imaging raises concern for malignancy without a clearly identifiable mass, as early recognition can spare patients unnecessary surgical intervention and allow for timely, effective treatment.

ABSTRACT V-30

Amiodarone-Induced Severe Hypothyroidism Presenting as Recurrent Cardiac Tamponade with Impending Myxedema Coma

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ABSTRACT

Background: Amiodarone-induced hypothyroidism (AIH) is a recognized complication of chronic therapy resulting from iodine excess and disruption of thyroid hormone metabolism. Although pericardial effusions are a known manifestation of severe hypothyroidism, progression to recurrent cardiac tamponade remains rare. We present a case of severe AIH presenting with recurrent tamponade physiology requiring multiple pericardiocenteses and intensive care management.Case presentation: 51-year-old man with heart failure with reduced ejection fraction (EF 20–25%), atrial fibrillation on chronic amiodarone therapy, implantable cardioverter-defibrillator placement, coronary artery disease, and hypertension presented with progressive dyspnea on exertion, chest discomfort, and declining functional capacity over several months. Physical examination revealed muffled heart sounds and mild peripheral edema. Electrocardiography demonstrated low-voltage QRS complexes with electrical alternans, while chest radiography showed marked cardiomegaly with a “water-bottle” cardiac silhouette. Point-of-care ultrasound and transthoracic echocardiography identified a large circumferential pericardial effusion with right atrial and right ventricular diastolic collapse, consistent with cardiac tamponade physiology. Laboratory studies revealed severe primary hypothyroidism with TSH 143 μIU/mL and free T4 0.17 ng/dL. Initial infectious and inflammatory studies were unrevealing.Urgent pericardiocentesis evacuated approximately 700 mL of serosanguineous fluid. Despite initial improvement, the patient developed recurrent effusions with repeated episodes of tamponade physiology requiring three drainage procedures and removal of approximately 1.8 L of fluid. Extensive evaluation for malignant, autoimmune, and infectious etiologies was pursued. Amiodarone was discontinued, and levothyroxine therapy was initiated with cautious titration because of underlying cardiomyopathy and arrhythmia risk. Thyroid function progressively improved, with TSH decreasing from 143 to 54 μIU/mL and normalization of free T4. The patient ultimately achieved sustained hemodynamic stability without recurrence and was discharged with cardiology and endocrinology follow-up.Conclusion: Although AIH is relatively common, life-threatening tamponade secondary to severe hypothyroidism is exceptionally uncommon. This case emphasizes the importance of considering thyroid dysfunction in patients receiving chronic amiodarone who present with unexplained pericardial effusion, low-voltage electrocardiographic findings, or recurrent tamponade physiology. Early recognition, urgent drainage, and thyroid hormone replacement are essential to prevent hemodynamic collapse and other severe hypothyroid complications.

ABSTRACT V-31

When the Liver Silences the Esophagus: Progressive Dysphagia From Gastroesophageal Mass Effect of Massive Polycystic Liver Disease

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ABSTRACT

Autosomal Dominant Polycystic Kidney Disease (ADPKD) may present with Polycystic Liver Disease (PLD), which can lead to hepatomegaly causing mass effect on adjacent structures. Gastroesophageal compression resulting in dysphagia is an extremely uncommon presentation, with very few case reports available in the literature. We describe a case of severe PLD manifesting with symptoms of progressive dysphagia that was initially suspected to have primary esophageal etiology.A 64-year-old woman with ADPKD, PLD, and prior esophageal narrowing developed months of progressive dysphagia from solids to liquids, regurgitation, nausea, vomiting, and inability to maintain oral intake. The patient lost approximately 30 lb over four months, causing malnutrition, with a body mass index of 17.7 kg/m^2, requiring multiple hospitalizations. Radiological imaging using computed tomography and upper gastrointestinal contrast study showed distended, fluid-filled esophagus with severe distal narrowing at the gastroesophageal junction (GEJ). Esophagogastroduodenoscopy (EGD) revealed distal food bolus impaction, proximal esophageal dilatation, and traversable GEJ narrowing, with possible differential diagnoses of esophageal stricture, achalasia, and superior mesenteric artery syndrome. Magnetic resonance imaging detected severe cystic liver enlargement, causing hepatomegaly, abrupt esophageal cutoff at the GEJ, and stomach compression with lateral displacement. Following multidisciplinary evaluation, the patient underwent laparoscopic fenestration of four dominant hepatic cysts with drainage of about 3 liters of cystic fluid, providing temporary improvement in oral tolerance. Dysphagia recurred, and percutaneous aspiration was deferred due to extensive liver cysts burden and poor long term improvement. At present, the patient has a feeding jejunostomy for enteral nutrition and is under evaluation for liver transplantation.This case presents a unique mechanical complication of PLD that mimicked an esophageal primary pathology. Extrinsic etiology is suggested by the existence of a traversable stricture in the absence of a fixed intraluminal pathology, the evidence of anatomical mass effect, and transient response to liver cyst fenestration. Recurrence of dysphagia despite targeted therapy demonstrates the limitations of localized approaches, which cannot resolve the mass effect from persistent residual cyst burden. Therefore, severe nutritional compromise combined with persistent compressive symptoms may require long-term nutritional support and liver transplant evaluation.Severe PLD should be suspected as a possible underlying cause for idiopathic progressive dysphagia when no evident intraluminal cause is found on endoscopy, and there is a mass effect on the gastroesophageal junction on imaging studies. Recognition of this uncommon extrinsic cause can redirect care toward hepatobiliary intervention, nutrition optimization, and liver transplant assessment because in the case of severe or refractory PLD, liver transplantation is the only curative therapy when other treatments fail to provide durable relief.

ABSTRACT V-32

IgG4-Related Disease with Suspected ANCA-Associated Vasculitis in a Patient with Bronchiectasis and Alveolar Hemorrhage: A Diagnostic Challenge

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ABSTRACT

Introduction:IgG4-related disease (IgG4-RD), eosinophilic granulomatosis with polyangiitis (EGPA), and microscopic polyangiitis (MPA) are systemic inflammatory disorders with potentially overlapping pulmonary, serologic, and histopathologic manifestations. IgG4-positive plasma-cell infiltration may also occur in ANCA-associated vasculitis (AAV), complicating interpretation of tissue findings. Both IgG4-RD and AAV may involve the respiratory tract, kidneys, orbit, and upper airways, yet simultaneous histologic and serologic evidence remains uncommon and can obscure the dominant mechanism of organ injury. Distinguishing true coexistence from phenotypic overlaps is therefore essential because classification, organ surveillance, and therapeutic decisions may differ.Case Presentation:A 75-year-old woman with chronic bronchiectasis, recurrent pulmonary infiltrates, and chronic bronchitis underwent extensive evaluation for persistent inflammatory pulmonary disease. Bronchoscopies demonstrated chronic tracheobronchial inflammation and, subsequently, diffuse blood throughout the tracheobronchial tree concerning for alveolar hemorrhage. Bronchoalveolar lavage specimens showed predominant neutrophilic inflammation and were repeatedly negative for malignant cells, acid-fast bacilli, and fungal organisms. Infectious evaluation, including Aspergillus serologies, galactomannan, and Strongyloides IgG, was negative. Immunologic evaluation demonstrated markedly positive P-ANCA (>8.0 antibody index) with negative C-ANCA, raising concern for an ANCA-associated vasculitis phenotype. Total IgE was elevated to 911 IU/mL, with peripheral eosinophilia reaching 9.8% (0.55 $\times 10^3/\mu L$). Inflammatory markers were notable for an ESR of 130 mm/hr. Progressive microcytic anemia was also observed, with hemoglobin decreasing to 9.5 g/dL in the setting of pulmonary bleeding. Serum ACE, rheumatoid factors, anti-CCP, and double-stranded DNA antibodies were unrevealing. Lacrimal gland biopsy with immunohistochemistry demonstrated abundant IgG4-positive plasma cells and an IgG4/IgG ratio >90%, interpreted as most consistent with IgG4-related dacryoadenitis. The patient subsequently received four doses of rituximab, including two inpatient doses and two administered at home.Discussion:The combination of histologically supported IgG4-RD with P-ANCA positivity, eosinophilia, elevated IgE, bronchiectasis, pulmonary infiltrates, and suspected alveolar hemorrhage created a complex differential involving IgG4-RD with concomitant AAV. P-ANCA positivity alone does not establish MPO specificity or distinguish MPA from EGPA. Pulmonary hemorrhage and anemia suggested a small-vessel vasculitis process, whereas elevated IgE and eosinophilia raised consideration of EGPA. However, the peak absolute eosinophil count of 0.55 $\times 10^3/\mu L$ was modest, and the available findings did not independently establish EGPA. Conversely, tissue IgG4 enrichment supported IgG4-related dacryoadenitis but could not, by itself, determine whether the pulmonary disease represented IgG4-related lung involvement, a separate AAV, or overlapping immune phenomena. Classification criteria should support, rather than replace, clinicopathologic diagnosis. This distinction matters because IgG4-RD commonly responds to glucocorticoids or B-cell depletion, whereas AAV with alveolar hemorrhage may require urgent remission-induction therapy and closer renal and pulmonary monitoring. Rituximab provides B-cell depletion relevant to both disease processes. This case therefore emphasizes careful correlation of organ involvement, antigen-specific MPO/PR3 testing, complete histologic morphology, and longitudinal treatment response.Conclusion:Histologically supported IgG4-related dacryoadenitis accompanied by P-ANCA positivity, eosinophilia, pulmonary hemorrhage, and inflammatory airway disease raised concern for concomitant AAV without definitively establishing MPA or EGPA. Recognition of this uncommon diagnostic intersection can prevent overreliance on a single biomarker and guide appropriate multidisciplinary evaluation, organ surveillance, and immunosuppressive management.

ABSTRACT V-33

Beyond HELLP: Complement Mediated Atypical Hemolytic Uremic Syndrome and a Kidney-Saving Turnaround.

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ABSTRACT

Complement-mediated atypical hemolytic uremic syndrome (CM-aHUS) is a rare thrombotic microangiopathy caused by dysregulation of the alternative complement pathway, resulting in endothelial injury, microvascular thrombosis, and multiorgan dysfunction. It is characterized by thetriad of microangiopathic hemolytic anemia, thrombocytopenia, and acute kidney injury. In pregnancy and the postpartum period, CM-aHUS frequently overlaps with preeclampsia and HELLP syndrome, creating a significant diagnostic challenge and often delaying definitive therapy.A 23-year-old primigravid woman at 31 weeks of gestation with a history of Kawasaki disease was transferred to our institution following the acute onset of right upper quadrant abdominal pain and nausea. The rapid emergence of hypertension, severe thrombocytopenia, microangiopathic hemolytic anemia, transaminitis, and acute kidney injury raised strong suspicion for HELLP syndrome. Progressive clinical deterioration necessitated emergent cesarean delivery, after which the patient was admitted to the surgical intensive care unit for ongoing management of severe obstetric-associated multiorgan dysfunction. Despite delivery and aggressive supportive management, her postpartum course was complicated by progressive anasarca, bilateral pleural effusions, hypoxemic respiratory failure requiring noninvasive ventilatory support and worsening non-oliguric acute kidney injury. Seventy-two hours postpartum thrombocytopenia and transaminitis improved, but persistent microangiopathic hemolysisand progressive renal dysfunction requiring emergent hemodialysis were disproportionate to the expected recovery trajectory of HELLP syndrome, prompting evaluation for thrombotic thrombocytopenic purpura (TTP) and CM-aHUS. A low C3 level, normal C4, and preserved ADAMTS13 activity (50%) effectively excluded TTP and supported a diagnosis of CM-aHUS. Targeted complement inhibition with eculizumab was subsequently initiated, resulting in rapid hematologic stabilization, progressive renal recovery, sustained urine output improvement, and complete discontinuation of dialysis. Following clinical remission, continued outpatient monitoring and genetic evaluation allowed cessation of therapy without evidence of relapse.This case highlights the importance of considering CM-aHUS in patients with persistent thrombotic microangiopathy and severe renal dysfunction beyond the expected postpartum resolution of HELLP syndrome. Early recognition and prompt initiation of complement blockade was kidney-savingin this case, allowing complete resolution of kidney function and hemodialysis cessation in a new young mother, impacting significantly the quality of life of our patient and her new daughter.

ABSTRACT V-34

Severe Hypothyroid Decompensation Suggestive of the Myxedema Coma Spectrum Without Altered Mental Status

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ABSTRACT

Introduction:Myxedema coma is a rare, life-threatening manifestation of severe hypothyroidism classically characterized by altered mental status, hypothermia, bradycardia, and hemodynamic instability. However, frank coma is not a prerequisite, and reliance on neurologic decline for diagnosis may delay recognition and treatment. We describe a patient with severe hypothyroid decompensation and features suggestive of impending myxedema coma despite preserved cognition and cardiovascular stability.Case Presentation:A 61-year-old woman with hypothyroidism, coronary artery disease status post myocardial infarction and coronary artery bypass grafting, hypertension, and type 2 diabetes presented with five days of right upper quadrant pain, vomiting, and progressive edema, along with cold intolerance, generalized weakness, constipation, and facial swelling. She had discontinued levothyroxine approximately two months earlier. She was alert and fully oriented, with blood pressure 166/73 mmHg, heart rate 78 beats/min, respiratory rate 20 breaths/min, oxygen saturation 99% on room air, and temperature 37.1°C. Examination showed facial edema, bilateral non-pitting lower-extremity edema, and abdominal tenderness. Laboratory studies revealed TSH 139 mIU/L, free T4 0.28 ng/dL, sodium 134 mmol/L, estimated glomerular filtration rate 25–39 mL/min/1.73 m^2, total cholesterol 360 mg/dL, and LDL cholesterol 263 mg/dL. Computed tomography demonstrated diffuse soft-tissue edema consistent with anasarca. During hospitalization, her temperature decreased to 35.5°C while mental status and hemodynamics remained stable. Her Popoveniuc score was 35, below the threshold suggestive of increased risk for myxedema coma. She received intravenous levothyroxine with a 200-µg loading dose; liothyronine was withheld given her significant coronary disease and clinical stability. A single 125-mg dose of intravenous methylprednisolone was administered in the emergency department. She improved without requiring mechanical ventilation or vasopressor support and was discharged with endocrinology follow-up.Discussion:Myxedema coma is primarily a clinical diagnosis, and its terminology may obscure severe presentations occurring before frank neurologic collapse. Our patient had profound biochemical hypothyroidism, systemic myxedematous edema, gastrointestinal manifestations, renal dysfunction, mild hyponatremia, hypothermia, and medication nonadherence as a clear precipitating factor, yet remained cognitively and hemodynamically intact. Her Popoveniuc score of only 35 reflected the absence of central nervous system and cardiovascular dysfunction despite marked biochemical hypothyroidism and multisystem manifestations, illustrating a key limitation of relying on classic late-stage findings or a diagnostic score in isolation. Scoring systems should complement, not replace, clinical judgment. Recognizing and treating severe hypothyroidism before the onset of altered mental status, respiratory failure, or cardiovascular collapse may prevent progression to life-threatening decompensation.Conclusion:Severe hypothyroid decompensation should remain a diagnostic consideration even in the absence of altered mental status or hemodynamic instability. Profound hypothyroidism accompanied by multisystem manifestations and a recognizable precipitating factor warrants prompt recognition and thyroid hormone replacement, with close monitoring for progression toward myxedema coma.

ABSTRACT V-35

When Treatment Becomes the Limitation: Disseminated Mycobacterium Abscessus After Liver Transplantation Complicated by GVHD

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ABSTRACT

Early graft versus host disease (GVHD) after liver transplantation is a rare and potentially life-threatening complication. Disseminated Mycobacterium abscessus infection is also uncommon among solid organ transplant recipients and presents substantial therapeutic challenges. We describe a case in which cumulative antimicrobial toxicity and constrained immunosuppression, rather than a lack of active agents, became the dominant barriers to effective treatment.A 59-year-old man with cirrhosis due to metabolic dysfunction and alcohol associated liver disease underwent deceased donor liver transplantation. Within weeks, he developed a widespread desquamating rash initially concerning for Stevens Johnson syndrome. Skin biopsy confirmed early GVHD involving skin and gastrointestinal tract, supported by donor chimerism of 25%, decreasing to 12% and later <5%. His course was complicated by pancytopenia, malnutrition requiring enteral nutrition, encephalopathy, and progressive weakness. He subsequently developed painful nodular skin lesions across the extremities and trunk. Blood cultures and skin biopsies revealed disseminated M. abscessus. Susceptibility testing demonstrated susceptibility to amikacin; intermediate activity to imipenem, cefoxitin, and linezolid; and resistance to clarithromycin and fluoroquinolones. Subspecies identification and macrolide resistance mechanism were unavailable, limiting assessment of macrolide activity in regimen planning.Given clarithromycin resistance, a macrolide sparing multidrug regimen was initiated. Treatment options rapidly narrowed due to cumulative toxicity. Linezolid led to neuropathy and thrombocytopenia; dual beta lactam therapy worsened mental status; and tigecycline caused severe gastrointestinal intolerance, prompting substitution with omadacycline. A salvage combination of imipenem/cilastatin, bedaquiline, omadacycline, and clofazimine was selected. Amikacin was initially included but discontinued after audiologic testing showed moderate to severe ototoxicity. Immunosuppression was reduced when feasible to improve infection control, though further reduction was limited by GVHD and graft function considerations.Despite prolonged therapy, imaging demonstrated persistent and new lesions without evidence of microbiologic cure. Clinically, the patient remained stable enough for continued suppressive therapy, but with increasing treatment related toxicity and limited likelihood of eradication. Therapeutic expectations were revisited frequently, and the patient elected to proceed with ongoing therapy after shared decision making discussions.This case highlights the extraordinary complexity of treating disseminated M. abscessus in a profoundly immunocompromised liver transplant recipient with GVHD. Although susceptibility testing identified several potentially active agents, the ability to sustain an effective multidrug regimen was progressively restricted by neurotoxicity, hematologic toxicity, gastrointestinal intolerance, and ototoxicity. Concurrent GVHD further limited reduction of immunosuppression, while disseminated disease restricted opportunities for source control. In this setting, therapeutic feasibility was determined less by in vitro antimicrobial activity than by cumulative toxicity, inability to sufficiently reverse immunosuppression, and limited source control. Early recognition of these constraints is essential to guide toxicity surveillance, establish realistic therapeutic expectations, and incorporate shared decision making into the management of complex post-transplant infections.

ABSTRACT V-36

The Diagnosis Behind the Diagnosis: Mixed Connective Tissue Disease in Severe Heart Failure

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ABSTRACT

Introduction:Systemic rheumatologic diseases may evolve insidiously and remain unrecognized until multiorgan dysfunction develops. Their heterogeneous manifestations can be attributed to more common conditions, particularly when severe dysfunction of one organ system provides an apparently unifying diagnosis. Recognizing findings that do not fit the working diagnosis is therefore critical to avoiding diagnostic closure.Case Description:A 62-year-old man presented with one month of progressive dyspnea, profound weight loss, poor oral intake, dysphagia, muscle weakness, fatigue, and functional and cognitive decline. Because of profound weakness and encephalopathy, the initial history was obtained from family. He had recently been discharged from another facility after treatment for presumed pneumonia and possible scabies, but his symptoms continued to worsen. On presentation, he was cachectic and hypoxemic, requiring bilevel positive airway pressure.Initial evaluation revealed acute kidney injury, non-ST-segment elevation myocardial infarction, and B-type natriuretic peptide >5,000 pg/mL. Echocardiography demonstrated a newly reduced left ventricular ejection fraction of 30%-35%, moderate-to-severe concentric left ventricular hypertrophy, and a mild-to-moderate pericardial effusion. De novo heart failure with cardiorenal syndrome and cardiac cachexia initially appeared to unify his presentation.However, examination revealed swollen hands, sclerodactyly, Raynaud-type digital discoloration, and dark crusted ulcerative lesions. Creatine kinase was mildly elevated, and urinalysis demonstrated hematuria and proteinuria with intrinsic renal injury. Together with dysphagia and muscle weakness, these findings raised high suspicion for systemic autoimmune disease with possible vasculitic involvement.Given progressive multiorgan dysfunction, pulse-dose intravenous methylprednisolone and hemodialysis were initiated before serologic results became available. Subsequent testing revealed speckled antinuclear antibody >1:2,560, markedly elevated anti-RNP antibodies (>8.0 AI), and p-ANCA positivity at 1:20; Smith and anti-double-stranded DNA antibodies were negative with preserved complement levels. The clinical phenotype and anti-RNP positivity supported mixed connective tissue disease.Within 72 hours, encephalopathy, oral intake, respiratory status, and cutaneous findings improved. Respiratory support was discontinued, and hemodialysis was stopped after four sessions. Renal biopsy subsequently demonstrated glomerulosclerosis and hyaline vascular changes without active vasculitis.Discussion:The diagnostic challenge was not recognizing acute organ dysfunction but determining whether heart failure truly unified the presentation. Severe systolic dysfunction, cardiorenal syndrome, and cachexia initially provided a compelling explanation; however, discordant systemic findings prompted reconsideration of an isolated cardiac process. MCTD is diagnostically challenging because manifestations overlap with other connective tissue diseases and may emerge across multiple organ systems. Here, the clinical phenotype justified treatment before serologic confirmation, while subsequent anti-RNP positivity and overlapping systemic sclerosis- and myositis-like features established the diagnosis. Although renal involvement and low-titer p-ANCA raised concern for concomitant vasculitis, renal biopsy showed no active vasculitis.Conclusion:Limited access to rheumatologic care may allow systemic autoimmune disease to progress before specialty evaluation is obtained, leaving internists to recognize these disorders when multiorgan dysfunction is already present. This case underscores the importance of questioning an apparently unifying diagnosis when key findings remain unexplained. For internists practicing in resource-limited settings, early recognition of discordant rheumatologic features and timely initiation of appropriate evaluation and treatment may be critical to preventing irreversible organ damage.

ABSTRACT V-37

From Refractory Migraine to Facial Atrophy: Diagnosing Active Parry-Romberg Syndrome

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Program: Centro Médico Menonita Cayey Program

ABSTRACT

Keywords: Parry-Romberg syndrome; progressive hemifacial atrophy; refractory migraine; enophthalmos; neuroimaging; methotrexateIntroduction:Parry-Romberg syndrome (PRS), or progressive hemifacial atrophy, is a rare acquired neurocutaneous disorder with an incidence of roughly 1 in 700,000. This syndrome is characterized by progressive unilateral atrophy of facial skin, subcutaneous tissue, and muscle. Adult presentations require a high level of suspicion given the subtle initial neurologic manifestations, such as migraine-like headache, seizures, and trigeminal symptoms. Brain MRI findings include ipsilateral abnormalities, which may precede or accompany visible facial atrophy. We report an adult patient initially treated for chronic migraine whose progressive unilateral facial changes and serial neuroimaging led to recognition of active PRS.Case Description:54-year-old female with history of major depressive disorder, dyslipidemia, rheumatoid arthritis, hypothyroidism, nephrolithiasis, and bronchial asthma presented for evaluation of recurrent severe headaches. Patient had history of controlled childhood-onset headaches, until recurrence approximately 1–2 months before her initial neurology visit. She reported about 12 headache days monthly that were bifrontal with occipital radiation, pulsatile and pressure-like in quality, rated 10/10, and lasted up to one day. Associated symptoms included photophobia, phonophobia, nausea, allodynia, and visual phenomena described as floating colorful dots. Headaches worsened with exertion and improved in a dark, quiet room. Given this presentation, she was initially treated for migraine with galcanezumab-gnlm and rizatriptan benzoate. Initial head MRI demonstrated nonspecific frontal and parietal subcortical/deep white-matter hyperintensities and an age-indeterminate left thalamic microhemorrhage. Head CTA showed no significant findings or occlusions. Galcanezumab-gnlm was discontinued because of a wearing-off effect, and atogepant was titrated to 60 mg daily. Despite more than six months of therapy, she continued to have one to three severe headache days weekly. One year after initial visit, she reported progressive left facial numbness, smell changes, and perceived facial asymmetry. Examination demonstrated left enophthalmos, facial soft-tissue loss, and underlying cranial asymmetry without focal cranial nerve deficits.Repeat brain MRI revealed thinning of the left extracalvarial superficial soft tissues, left enophthalmos, mild hypoplasia of the left maxillary sinus, and slight volume loss of the left cerebral hemisphere at the high frontoparietal convexity. In the setting of progressive unilateral hemifacial atrophy, these findings supported PRS. Erenumab-aooe and ubrogepant replaced atogepant and rizatriptan benzoate, improving headache frequency to approximately once every other week. Also added were methotrexate 15 mg weekly, folic acid, and prednisone 40 mg daily for three months with taper. Rheumatology, neuro-ophthalmology, maxillofacial surgery, and neurosurgery referrals were placed.Conclusion:This case illustrates how PRS can be obscured by a common primary headache presentation. Persistent or treatment-refractory migraine-like headaches accompanied by progressive facial asymmetry, enophthalmos, sensory changes, or skull contour abnormalities should prompt diagnostic reassessment and targeted imaging. Serial examination and MRI were critical in recognizing this case of PRS and allowed initiation of immunosuppressive therapy during suspected active disease. This case underscores the importance of avoiding diagnostic anchoring and coordinating multidisciplinary care for rare disorders.

ABSTRACT V-38

The Mimic in the Irradiated Breast: Radiation-Associated Angiosarcoma With Epithelioid Features

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ABSTRACT

IntroductionRadiation-associated angiosarcoma of the breast (RAASB) is a rare and aggressive secondarymalignancy arising within previously irradiated tissue. In a nationwide population-based cohort of184,823 breast cancer patients treated with radiotherapy, only 209 developed RAASB or chest-wallangiosarcoma, corresponding to approximately 0.1%, or 1 case per 1,000 irradiated patients.Despite its rarity, RAASB is clinically important because of its infiltrative behavior, high localrecurrence rates, and poor long-term outcomes. Early manifestations may resemble benigninflammatory, infectious, or post-radiation changes, and conventional breast imaging may benonspecific or apparently benign.Case DescriptionAn 81-year-old woman with prior left breast invasive ductal carcinoma underwent breast-conservingsurgery with axillary lymph node dissection, adjuvant chemotherapy, breast irradiation, andendocrine therapy with tamoxifen. Approximately seven years after radiotherapy, she developederythematous rash-like changes in the previously irradiated breast. The process progressed despiteoral antibiotics, while mammography and ultrasonography were interpreted as BI-RADS 2 with focalmastitis. Within less than one month, the cutaneous disease advanced strikingly, with marked skinthickening, discoloration, multiple violaceous nodules, and ulceration. A dermatologic punch biopsywas subsequently reported as compatible with angiosarcoma. At specialist evaluation, the multifocalviolaceous morphology prompted immediate concern for angiosarcoma, with inflammatory breastcarcinoma considered less likely. She underwent left mastectomy with wide radical resection. Finalpathology demonstrated multifocal angiosarcoma with epithelioid features, with the largest focusmeasuring 6.5 cm and involving breast parenchyma, skin of all four quadrants, and axillary softtissue. The epithelioid morphology initially raised concern for recurrent invasive breast carcinoma;however, subtle vasoformative features at the tumor periphery prompted immunohistochemicalevaluation. CD31 and CD34 positivity supported endothelial differentiation and confirmedangiosarcoma. Although the tumor appeared macroscopically completely excised with ample grossmargins, microscopic examination demonstrated focal involvement of the upper-outer-quadrant skinmargin. Postoperative FDG PET/CT showed postoperative inflammatory/reactive uptake withoutregional or distant FDG-avid metastatic disease. Paclitaxel plus bevacizumab was planned but notinitiated before the patient later died during hospitalization for an acute myocardial infarction.DiscussionThis case is noteworthy not simply because RAASB is rare, but because it illustrates two importantdiagnostic pitfalls. Clinically, a predominantly cutaneous malignancy may resemble mastitis or otherbenign inflammatory processes and may remain occult or nonspecific on conventional breastimaging. Histologically, epithelioid angiosarcoma may mimic recurrent carcinoma, makingrecognition of vasoformative architecture and endothelial immunophenotyping essential. Thedramatic progression over less than one month, extensive multifocal involvement, and positivemicroscopic margin despite an aggressive grossly complete resection further underscore theinfiltrative biology of RAASB. New or progressive erythematous, violaceous, ecchymotic, nodular, orulcerative changes in a previously irradiated breast should therefore prompt a low threshold fortissue biopsy, even when imaging appears reassuring.

ABSTRACT V-39

Airway Attack: Disseminated Mpox with Pulmonary Involvement as an AIDS-Defining Diagnosis

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ABSTRACT

Introduction:Mpox is a viral infection that can cause severe and disseminated disease in individuals with advanced HIV infection. Although cutaneous manifestations are characteristic, diffuse organ involvement is extremely rare. We present a case of disseminated Mpox in an AIDS patient with pulmonary involvement.Case Presentation:A 36-year-old Hispanic man presented with a 2-month history of a progressively worsening painful and pruritic rash with centrifugal spread, evolving into pustules. The patient appeared frail with decreased muscle mass but was afebrile and hemodynamically stable. Skin examination was remarkable for erythematous, well-demarcated, tender ulcers with circumferential erythema and central umbilication, with widespread involvement and superimposed impetigo in the facial and genital areas. Lung examination was significant for a nonproductive cough and right basilar crackles. Laboratory evaluation revealed positive non-variola orthopox virus PCR, HIV-1, a CD4 count of 19 cells/μL, and a reactive RPR. Chest CT demonstrated multiple irregular bilateral pulmonary nodules with surrounding ground-glass opacities in a peribronchovascular distribution. Bronchoscopy revealed friable bronchial mucosa with multiple small, flat, protuberant lesions involving the respiratory tract throughout. Bronchoalveolar lavage and cytology revealed reactive bronchial cells with viral-like cytopathic changes, including intranuclear ground-glass inclusions. Malignancy and special staining were negative, and infectious workup for typical and atypical pathogens was unrevealing. The patient was diagnosed with disseminated Mpox with pulmonary involvement in the setting of AIDS. He was treated with tecovirimat and doxycycline for 14 days, bictegravir-emtricitabine-tenofovir, trimethoprim-sulfamethoxazole, and topical mupirocin. Lung biopsy was planned; however, the patient left against medical advice before the procedure and was subsequently lost to follow-up.Discussion:Prior reports have characterized Mpox respiratory involvement through indirect or non-bronchoscopic modalities as randomly distributed necrotic pulmonary nodules on CT, bronchopneumonia and acute lung injury on autopsy or biopsy histopathology, viral cytopathic changes on respiratory cytology, and plaque-like supraglottic lesions on flexible laryngoscopy, with isolated necrotic tracheal involvement also reported. To our knowledge, this is the first reported case of Mpox with bronchoscopically visualized multiple flat, protuberant, and ulcerated tracheal lesions. The endoscopic airway involvement observed in this patient therefore represents an unusual manifestation of disseminated Mpox in the setting of advanced HIV/AIDS. This case supports consideration of disseminated Mpox as an AIDS-defining condition and emphasizes the importance of including Mpox in the differential diagnosis of unusual pulmonary and airway lesions in severely immunocompromised patients.

ABSTRACT V-40

Hungry Bone Syndrome Presenting as Laryngospasm After Subtotal Parathyroidectomy

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ABSTRACT

Introduction: Hungry bone syndrome is a common complication following parathyroidectomy for severe secondary hyperparathyroidism in patients with end-stage renal disease, with reported incidences ranging from approximately 20% to 70% depending on the population studied and diagnostic criteria used. It results from rapid skeletal uptake of calcium and minerals after removal of hyperfunctioning parathyroid tissue and may cause profound, prolonged hypocalcemia. While neuromuscular manifestations are well recognized, progression to severe hypocalcemia causing acute laryngospasm, respiratory failure, and the need for mechanical ventilation is infrequently reported, making recognition of this complication critical in high-risk patients. Case Description: A 65-year-old woman with end-stage renal disease on hemodialysis, type 2 diabetes mellitus, and hypertension underwent elective subtotal parathyroidectomy for severe secondary hyperparathyroidism with a preoperative parathyroid hormone level exceeding 4,000 pg/mL. During the postoperative period, she developed severe hypocalcemia consistent with hungry bone syndrome, reaching a nadir total serum calcium of 6.0 mg/dL, corresponding to a corrected calcium of 6.4 mg/dL (albumin 3.5 g/dL), and an ionized calcium of 3.4 mg/dL. She subsequently developed acute laryngospasm with respiratory failure requiring endotracheal intubation and mechanical ventilation. Computed tomography of the neck showed no evidence of laryngeal edema, and computed tomography angiography excluded pulmonary embolism, supporting severe hypocalcemia as the most likely etiology of airway compromise. Management included continuous intravenous calcium infusion, high-dose oral calcium carbonate, calcitriol, vitamin D supplementation, adjustment of dialysate calcium concentration, serial electrolyte monitoring, and close nephrology follow-up. Surgical pathology demonstrated diffuse hyperplasia of all four parathyroid glands. Her hospitalization was additionally complicated by multidrug-resistant Escherichia coli bacteremia, which resolved following meropenem therapy. Following metabolic stabilization and renal replacement therapy optimization, she was successfully extubated without recurrence of airway compromise and showed progressive clinical improvement. She was discharged home in stable condition on optimized calcium and vitamin D supplementation with close nephrology follow-up. Discussion: This case highlights a rare and potentially life-threatening presentation of hungry bone syndrome following parathyroidectomy. Anticipation of profound postoperative calcium shifts, intensive electrolyte surveillance, and prompt multidisciplinary intervention are essential to prevent severe airway complications and optimize outcomes in dialysis-dependent patients.

ABSTRACT V-41

When Hypothyroidism Silences the Heart: Myxedema Coma Presenting With Acute Bradycardic Cardiac Tamponade

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Program: UCC Lcdo. Eduardo Artau

ABSTRACT

Myxedema coma is a rare and life threatening manifestation of severe hypothyroidism characterized by altered mental status, hypothermia, bradycardia, hypoventilation and multi-organ dysfrunction. Pericardial effusion is a recognized complication of severe hypothyroidism; however, progression to cardiac tamponade is exceedingly rare and may be difficult to recognize. Hypothyroidism-associated bradycardia can blunt the expected tachycardic response to tamponade, potentially masking a key clinical sign and delaying recognition of this reversible cause of hemodynamic collapse.This is the case of a 60-year-old man with untreated hypothyroidism, morbid obesity (body mass index of 49 kg/m^2) and a 48-pack-year smoking history presenting to the Emergency Room after collapse and syncope. Given his smoking history and respiratory symptoms, chronic obstructive pulmonary disease (COPD) exacerbation was initially suspected. He progressed to severe hypercapnic and hypoxemic respiratory failure, with arterial PCO_2 119 mmHg and PaO_2 35 mmHg, requiring endotracheal intubation and mechanical ventilation. Further evaluation revealed severe hypothyroidism with TSH 69 mIU/L, free T4 <0.1 ng/dL accompanied by hypothermia, hypoglycemia, cardiomegaly, pulmonary edema, hypotension, profound bradycardia, non-pitting edema, and macroglossia, supporting a diagnosis of myxedema coma. Chest Computed Tomography (CT) revealed findings concerning for superimposed pneumonia, which was considered the likely precipitating factor for myxedema coma. He received an intravenous loading dose of hydrocortisone 100 mg before an intravenous levothyroxine loading dose of 200 mcg, followed by hydrocortisone 50 mg every 6 hours and levothyroxine 150 mcg daily beginning on hospital day one. Despite aggressive therapy, the patient developed worsening hypotension and persistent profound bradycardia with a limited chronotropic response on hospital day two. Bedside echocardiography demonstrated a large pericardial effusion with findings concerning for cardiac tamponade. At the time of tamponade, he exhibited Beck's triad of hypotension, muffled heart sounds, and jugular venous distension. Emergent pericardiocentesis yielded approximately 600 mL of golden-yellow fluid, followed by rapid improvement in blood pressure from 90/60 to 143/82 mmHg. With continued thyroid hormone and corticosteroid therapy, his hemodynamics improved, and he subsequently became awake and able to follow commands. A small residual pericardial effusion gradually decreased with continued treatment of the underlying hypothyroidism. His prolonged intensive care course was later complicated by multidrug-resistant Acinetobacter baumannii bacteremia and pneumonia, leading to septic shock, hemodialysis, and ultimately death from complications of prolonged critical illness.This case highlights the diagnostic complexity of myxedema coma when severe respiratory and cardiovascular abnormalities coexist. The initial presentation mimicked COPD exacerbation; however. hypothermia, hypoglycemia, profound bradycardia, hypoventilation among others in light of severe hypothyroidism supported a diagnosis of myxedema coma. Importantly, worsening hypotension despite appropriate thyroid hormone and corticosteroid therapy prompted evaluation for an additional reversible cause. In this case, bedside echocardiography identified cardiac tamponade and was rapidly relieved by emergent pericardiocentesis, resulting in immediate hemodynamic improvement. Severe hypothyroidism-associated bradycardia and impaired chronotropic response may obscure the cardiovascular manifestations of tamponade. Cardiac tamponade should remain in the differential diagnosis of unexplained or worsening hypotension in patients with myxedema coma. Early bedside echocardiography may be critical for identifying this uncommon but potentially reversible complication.

ABSTRACT V-42

When a Contaminant Becomes a Pathogen: Invasive Bacillus Infection with Ocular and Endovascular Involvement

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Program: University of Puerto Rico Program

ABSTRACT

Introduction Bacillus species, although frequently dismissed as blood culture contaminants, can cause severe invasive disease in susceptible hosts, particularly those with intravascular devices. We present an unusual case of persistent Bacillus bacteremia associated with corneal abscess, septic pulmonary emboli, and tricuspid valve infective endocarditis. Case Description A 74-year-old man with type 2 diabetes mellitus and end-stage renal disease receiving hemodialysis through a tunneled catheter presented with right eye pain and purulent drainage. Examination revealed a large central corneal ulcer with corneal melting and a total corneal abscess involving a chronically blind eye. Laboratory evaluation demonstrated leukocytosis and elevated inflammatory markers, and he was admitted for sepsis. Initial blood cultures grew Staphylococcus epidermidis in four of five bottles and Bacillus species in one of five bottles. Given the patient's tunneled catheter and low culture burden of Bacillus, Staphylococcus epidermidis catheter-related bloodstream infection with contaminant Bacillus bacteremia was initially suspected. The patient received intravenous antibiotics and underwent catheter exchange; catheter tip cultures grew both organisms. Although Staphylococcus epidermidis bacteremia resolved, repeat blood cultures intermittently remained positive for Bacillus species over the following three weeks. Subsequent review revealed that the corneal abscess culture had also grown Bacillus species. Computed tomography of the chest demonstrated septic pulmonary emboli. Transthoracic echocardiography showed no vegetations. Transesophageal echocardiography, obtained following antimicrobial optimization to vancomycin and imipenem-cilastatin, revealed a 1.3-cm highly mobile vegetation involving the anterior and posterior tricuspid valve leaflets, confirming infective endocarditis. Blood cultures subsequently cleared, and the patient was discharged to complete six weeks of intravenous antimicrobial therapy. Discussion Bacillus species are frequently dismissed as blood culture contaminants but can cause severe invasive infection. In this case, intermittent single-bottle positivity and concurrent Staphylococcus epidermidis bacteremia initially obscured its clinical significance. Persistent recovery of Bacillus species and concordant growth from the corneal abscess ultimately supported true invasive infection. Clinicians should reconsider presumed contaminants when they persist or are isolated from multiple clinically relevant sites.

ABSTRACT V-43

Beyond the Kidney: Extensive Infected Urinoma Following Spontaneous Calyceal Rupture

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ABSTRACT

Introduction Spontaneous calyceal rupture is an uncommon complication of obstructive uropathy, most frequently associated with ureterolithiasis. Urinary extravasation may result in urinoma formation, which can become infected and rarely extend extensively throughout the retroperitoneum. Xanthogranulomatous pyelonephritis (XGP) is a chronic destructive inflammatory process associated with longstanding urinary obstruction and infection. We present a case of spontaneous calyceal rupture secondary to chronic obstructive ureterolithiasis complicated by a remarkably extensive infected urinoma, with subsequent histopathology revealing XGP. Case Presentation A 78-year-old woman with a history of hypertension, type 2 diabetes mellitus, hypothyroidism, and nephrolithiasis requiring lithotripsy presented after two months of progressively worsening left inguinal pain that migrated to the left lower quadrant and impaired ambulation. She denied fever, chills, nausea, vomiting, dysuria, hematuria, or trauma. She was afebrile and hemodynamically stable. Laboratory studies demonstrated neutrophilic leukocytosis, microcytic hypochromic anemia, thrombocytosis, pyuria, and bacteriuria. Computed tomography demonstrated a 2.0-cm obstructing proximal left ureteral calculus with severe hydronephrosis and spontaneous calyceal rupture, resulting in an extensive infected urinoma with perirenal and pararenal abscess formation extending along the left paracolic gutter, iliopsoas, and pelvis. Given her clinical stability, immediate nephrectomy was deferred. The patient was treated empirically with intravenous antibiotics. She underwent left percutaneous nephrostomy for urinary decompression and placement of two percutaneous pigtail drains into the retroperitoneal collection, yielding purulent fluid. Follow-up computed tomography demonstrated marked interval improvement following decompression and drainage. After clinical stabilization, she was discharged on oral antibiotic therapy. Approximately three months after her initial presentation, she underwent definitive laparoscopic radical nephrectomy of the chronically nonfunctioning kidney. Her postoperative course was uncomplicated, with preserved renal function. Histopathologic examination demonstrated xanthogranulomatous pyelonephritis. Discussion Spontaneous calyceal rupture is a rare complication of obstructive ureterolithiasis resulting from increased pressure within the collecting system and subsequent urinary extravasation. Although urinomas may remain localized, persistent urinary leakage and secondary infection can lead to extensive retroperitoneal abscess formation. This case is notable for the remarkable anatomic extent of the infected urinoma, involving the perirenal and pararenal spaces and extending along the paracolic gutter, iliopsoas, and pelvis. Despite this extensive disease, the patient remained afebrile and hemodynamically stable, demonstrating that severe complications of chronic obstructive uropathy may present indolently and without systemic toxicity. Histopathologic examination demonstrated XGP, a rare chronic inflammatory disorder characterized by destruction of renal parenchyma, typically associated with longstanding urinary obstruction and infection. The findings further demonstrate the chronic and destructive nature of the underlying renal disease. Management required a staged approach consisting of antimicrobial therapy, urinary decompression with percutaneous nephrostomy, and drainage of the infected collections, followed by delayed nephrectomy after control of the acute infectious process. Conclusion This case demonstrates the potential for obstructive ureterolithiasis to progress beyond the collecting system, resulting in calyceal rupture, extensive infected urinoma, and widespread retroperitoneal involvement. It also highlights that advanced disease may occur in the absence of systemic toxicity and that histopathology can reveal underlying chronic destructive renal disease contributing to irreversible renal damage.

ABSTRACT V-44

Diagnostic Discordance in Amebic Liver Abscess: When Cytopathology Outweights Serology

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ABSTRACT

BackgroundAmebic liver abscess (ALA), caused by Entamoeba histolytica, is the most common extraintestinal manifestation of amebiasis and should be considered in travelers returning from endemic regions who present with fever and right-upper-quadrant pain. Diagnosis is challenging when serology and stool studies are negative, as antibodies may be undetectable early in disease and stool microscopy is insensitive (New England Journal of Medicine, 2003). We report a seronegative ALA in a returning traveler, confirmed by aspirate cytopathology, presenting with a toxic septic picture, recurrent high-grade fever, and severe abdominal pain.Case PresentationA 36-year-old man with recurrent travel to Colombia presented with 3–5 days of nausea, bilious vomiting, altered bowel habits, and progressively severe, diffuse abdominal pain. He appeared acutely ill and toxic, and met sepsis criteria with tachycardia, leukocytosis (WBC 17.3 × 10^3/μL), elevated procalcitonin, and lactic acidemia, prompting ICU admission and resuscitation. Throughout admission he had recurrent episodes of high fever reflecting the ongoing hepatic infectious process, and severe abdominal pain worst in the epigastrium and right upper quadrant that required multimodal analgesia. A coexisting opioid use disorder was noted and briefly considered in the differential for his fevers, but the febrile course tracked with the hepatic collections and their drainage.InvestigationsCross-sectional imaging demonstrated multiple hepatic collections. Contrast-enhanced MRI showed four T2-hyperintense, T1-hypointense fluid collections with thick enhancing walls and adjacent parenchymal hyperemia (4.7 cm in segment 3, with additional lesions in segments 4A/8, 5, and 6), consistent with hepatic abscesses. Ultrasound confirmed multiple complex avascular hypoechoic lesions in both lobes with hepatomegaly. Percutaneous aspiration yielded thick "anchovy-paste" purulent material. Cytopathology revealed abundant acute inflammatory debris and cells with dense bubbly cytoplasm, small round nuclei, peripheral condensed chromatin, and central dot-like karyosomes — morphology consistent with amebic trophozoites. Notably, E. histolytica serology and serial stool ova-and-parasite examinations were negative, underscoring that seronegativity does not exclude ALA.Management and OutcomeStaged image-guided percutaneous catheter drainage was performed for the larger collections. Antimicrobial therapy comprised a nitroimidazole—initially metronidazole, transitioned to tinidazole after a metronidazole reaction—followed by the luminal agent paromomycin to eradicate intestinal colonization. Fevers and leukocytosis resolved, abdominal pain abated, and serial imaging demonstrated progressive reduction in abscess size. The patient was discharged in stable condition and remained asymptomatic at infectious-disease follow-up, with continued radiographic improvement.ConclusionALA can present with a toxic appearance, sepsis physiology, recurrent high fever, and severe abdominal pain in a returning traveler; coexisting substance use disorder may briefly confound the febrile differential but should not delay evaluation of an intra-abdominal source. This case also highlights a key diagnostic pitfall: although amebic serology is highly sensitive (>90%) in established disease, it is positive in only about 70–80% of patients at presentation because the antibody response lags symptom onset, leaving roughly 20–30% seronegative when tested early. A single negative serology does not exclude ALA; when suspicion persists, aspirate examination for trophozoites, E. histolytica antigen or PCR, and convalescent serology establish the diagnosis. Prompt recognition, nitroimidazole-plus-luminal therapy, and drainage of large collections yield excellent outomes

ABSTRACT V-45

Frozen Eyes, Failing Muscles, and a Faltering Heart: Ocular Symptoms as the First Manifestation of Pembrolizumab-Induced Triple-M Syndrome

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Program: Not provided

ABSTRACT

IntroductionImmune checkpoint inhibitors (ICIs) have transformed oncologic care but cause rare, fatal immune-related adverse events. The overlap of myocarditis, myositis, and myasthenia gravis (Triple-M syndrome) occurs in fewer than 1% of treated patients yet carries an in-hospital mortality of 40%–60%. Because early manifestations are often limited to ocular symptoms, patients frequently present first to non-oncologic settings. We describe a patient whose only initial finding was ophthalmoplegia with a negative ice pack test and who required mechanical ventilation nine days later.Case DescriptionA 77-year-old man with hypertension, type 2 diabetes, and recurrent urothelial carcinoma received pembrolizumab plus enfortumab vedotin. Eighteen days after the second cycle, he developed bilateral ptosis and diplopia, and the third cycle was withheld. Ophthalmologic evaluation documented complete ophthalmoplegia with negative forced ductions, positive fatigability, and a negative ice pack test. Fatigability localized the process to the neuromuscular junction rather than enfortumab-related neuropathy, and ICI-induced myasthenia gravis was suspected. Brain MRI was unremarkable, but the patient left against medical advice before evaluation was completed. Six days later he returned with unchanged ocular findings but new dysphagia, dysphonia, and respiratory difficulty and was admitted. Examination showed complete ptosis and ophthalmoplegia, fatigability, nasal speech, and neck flexion weakness (4/5). Negative inspiratory force was -35 cmH2O. Creatine kinase was 7,882 U/L and aldolase 46.7 U/L, with elevated transaminases and normal gamma-glutamyl transferase, localizing injury to muscle. Troponin T peaked at 1,767 ng/L, and echocardiography showed an ejection fraction of 45% to 50% with regional wall-motion abnormalities. Acetylcholine receptor antibodies later returned positive on all three assays (binding 6.59 nmol/L, blocking 47%, modulating 57%); chest CT showed no thymoma. ICI-induced Triple-M syndrome was diagnosed and pembrolizumab was permanently discontinued. Despite pyridostigmine and intravenous immunoglobulin, he required mechanical ventilation on hospital day 3, at which point high-dose corticosteroids were initiated. Ventilator-associated Proteus mirabilis pneumonia delayed escalation to rituximab. After the pneumonia was treated, plasma exchange, rituximab, and complement or interleukin-6 inhibition were offered but declined. He improved on corticosteroids and supportive care, was extubated after 15 days, and was discharged to rehabilitation with troponin T <22 ng/L and an ejection fraction of 45% to 50%.DiscussionThis case offers three lessons for internists. First, isolated ocular symptoms in a patient receiving an ICI are an emergency: this patient's ocular examination never changed, yet nine days after an evaluation at which it was the only abnormality he was intubated. The first encounter should include negative inspiratory force, creatine kinase, and troponin. Second, immunosuppression should not await antibody or electrodiagnostic confirmation, and a negative ice pack test must not lower suspicion. Third, myocarditis is the principal determinant of mortality and must be sought in every patient with suspected ICI-related myasthenia gravis or myositis, even without cardiac symptoms. Pneumonia both mimicked myasthenic worsening and delayed rituximab, illustrating how narrow the therapeutic window becomes once respiratory failure develops. Recognizing ocular symptoms as the earliest sign of Triple-M syndrome and acting immediately may be the only opportunity to treat before irreversible complications develop.

ABSTRACT V-46

When Infection and Malignancy Are Not the Answer: Recognizing Granulomatosis With Polyangiitis in Progressive Pulmonary Disease

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ABSTRACT

Introduction:Granulomatosis with polyangiitis (GPA) is a rare systemic vasculitis predominantly affecting the upper and lower respiratory tracts that may mimic infectious or malignant diseases. Published literature describing GPA in Puerto Rico is notably limited, with few reported cases and even less information from the southern region. We present a case from southern Puerto Rico in which an evolving multisystem presentation ultimately led to the diagnosis of GPA.Case Description:A 42-year-old nonsmoking man with hypertension and type 2 diabetes mellitus presented with one month of progressive dyspnea, cough, hemoptysis, pleuritic chest pain, fatigue, bilateral calf pain, and an unintentional 8-pound weight loss. He had previously received levofloxacin for a presumed respiratory infection without improvement. On admission, he was acutely ill, tachycardic, and hypoxemic. Laboratory studies demonstrated leukocytosis, normocytic anemia, thrombocytosis, elevated inflammatory markers, and preserved renal function. Chest radiography showed bilateral infiltrates and a nodular pulmonary opacity. He was admitted for presumed community-acquired pneumonia and received ceftriaxone and azithromycin, with subsequent broadening of antimicrobial coverage due to persistent symptoms and hypoxemia.Chest computed tomography demonstrated left lower-lobe consolidation, multiple pulmonary nodules, and a pleural effusion. Despite broad antimicrobial therapy, his condition deteriorated. He subsequently developed marked nasal swelling, bloody nasal secretions, epistaxis, gingival bleeding, conjunctival inflammation, migratory interphalangeal joint pain and swelling, lower-extremity petechiae, and persistent hemoptysis. Repeat chest imaging eight days later demonstrated rapid progression of pulmonary abnormalities, including a mass-like lesion, raising concern for malignancy given the associated weight loss.Bronchoscopy with bronchoalveolar lavage and cytology showed no evidence of malignancy or fungal infection, while extensive infectious testing was unrevealing. Progressive pulmonary disease despite antimicrobial therapy and evolving multisystem manifestations prompted evaluation for systemic vasculitis. Cytoplasmic antineutrophil cytoplasmic antibodies (C-ANCA) were positive at 1:160, and anti-proteinase-3 antibodies were markedly elevated (>8.0 units), strongly supporting GPA. High-dose corticosteroids were initiated with clinical improvement. The patient was discharged on prednisone and azathioprine, with planned rituximab therapy.Discussion:This case demonstrates how GPA can masquerade sequentially as pulmonary infection and malignancy. Pneumonia was initially supported by hypoxemia, leukocytosis, consolidation, and respiratory symptoms, while rapid radiographic progression, a mass-like lesion, and weight loss subsequently raised concern for malignancy. The diagnostic turning point was recognizing that pulmonary disease progressed despite broad antimicrobial therapy, infectious and malignant evaluations remained unrevealing, and manifestations accumulated across multiple organ systems. Preserved renal function should not exclude GPA when the respiratory and systemic phenotype is compelling. Early recognition is essential to enable timely immunosuppressive therapy, limit clinical deterioration, and reduce the risk of progressive and potentially irreversible organ damage.

ABSTRACT V-47

Beyond Classic Myositis: Profound Dysphagia and Diaphragmatic weakness in Severe Anti-Synthetase Syndrome

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Program: San Juan VA Medical Center

ABSTRACT

Anti-synthetase syndrome (ASyS) is a rare autoimmune condition characterized by inflammatory myopathy, interstitial lung disease, arthritis, Raynaud phenomenon, and the presence of anti–aminoacyl tRNA synthetase antibodies, most commonly anti–Jo 1. Although dysphagia and respiratory muscle involvement are recognized manifestations, severe pharyngoesophageal dysfunction and diaphragmatic weakness remain underreported and can lead to life threatening respiratory compromise. This case describes a severe presentation of ASyS marked by rapidly progressive myositis, profound dysphagia, and diaphragmatic dysfunction requiring urgent multimodal therapy. A 75 year old male with multiple cardiometabolic comorbidities presented with 12 days of worsening proximal muscle weakness, diffuse myalgias, inflammatory arthralgias, severe dysphagia, and progressive dyspnea. He experienced functional decline and necessitating assistance with basic activities. Examination revealed pronounced symmetric proximal weakness and impaired swallowing. Diaphragmatic ultrasound demonstrated significantly reduced excursion, suggesting respiratory muscle involvement. Laboratory evaluation showed markedly elevated muscle enzymes, including a creatine phosphokinase level of 6,796 U/L and aldolase greater than 150 U/L. Anti–Jo 1 antibodies were strongly positive, and infectious workup was negative. Muscle biopsy showed nonspecific myopathic changes consistent with inflammatory myositis. Given the rapid progression and concern for impending respiratory failure, high dose intravenous methylprednisolone and intravenous immunoglobulin (IVIG) were initiated. The patient exhibited substantial clinical improvement in strength, dyspnea, and swallowing within days. He was subsequently transitioned to daily corticosteroids and later high dose oral prednisone. Due to severity and serologic profile, rituximab induction therapy was administered, followed by mycophenolate mofetil titrated to 1,500 mg twice daily as long term immunosuppression. Monthly IVIG was recommended but later declined. Over outpatient follow up, muscle enzymes normalized, dysphagia resolved, and proximal strength significantly improved, though mild fatigue and residual weakness persisted. Months later, he developed digital ischemia affecting digits 2–3, prompting evaluation for vasculopathy manifestations and antiphospholipid syndrome. Despite intermittent medication nonadherence, overall disease stability was maintained under immunosuppressive therapy and prednisone tapering. This case highlights a severe phenotype of anti synthetase syndrome with debilitating myopathy, marked dysphagia, and diaphragmatic involvement. Early aggressive treatment with high dose steroids, IVIG, and rituximab facilitated rapid and durable improvement. Diaphragmatic ultrasound proved valuable in detecting respiratory muscle dysfunction, emphasizing the importance of noninvasive imaging in inflammatory myopathy assessment. The late onset of digital ischemia underscores potential vasculopathy complications in ASyS. Additionally, the course reinforces the critical role of sustained adherence to immunosuppressive therapy for long term disease control.

ABSTRACT V-48

Hemophagocytic Lymphohistiocytosis Secondary to HSV-1: An Atypical And Severe Presentation in an Immunocompetent Young Adult

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Program: University of Puerto Rico Program

ABSTRACT

Introduction: Hemophagocytic lymphohistiocytosis (HLH) is a life-threatening form of immune hyperactivation triggered by infections, malignancies, autoimmune diseases, or immunosuppressive states. While Epstein-Barr virus (EBV) and cytomegalovirus (CMV) are common infectious triggers in both the pediatric and adult population, rare presentations in immunocompetent young adults are seen due to Herpes Simplex Virus 1 (HSV-1). Given its high mortality when diagnosis and treatment are delayed, rapid clinical recognition and targeted interventions are essential.Case Description: A 23-year-old healthy woman presented with a 10-day history of sore throat and swelling, high-grade fever (42°C), and epigastric pain following a visit to a community river. She was transferred and admitted to a tertiary medical center after failing symptomatic management. A physical examination revealed hepatomegaly, abdominal tenderness, and anasarca. Initial laboratory studies revealed severe thrombocytopenia (11,000/uL), profound transaminitis (AST > 6,000 U/L, ALT > 2,000 U/L), predominantly direct hyperbilirubinemia (4.58 mg/dL), high ferritin levels (38,125 ng/mL), elevated lactate dehydrogenase (LDH) (>2,500 U/L) and hypertriglyceridemia (251 mg/dL). Neck CT revealed bilateral tonsillar phlegmonous process, while an Abdominal/Pelvis CT was remarkable for innumerable liver hypodense lesions, hepatosplenomegaly, and mild hepatic capsular enhancement.Multidisciplinary services were consulted and HLH was suspected based on the constellation of fever, cytopenia, hypertriglyceridemia, extreme ferritinemia, and acute hepatic failure. An extensive infectious work-up for viral and bacterial etiologies was performed, including EBV, dengue virus, CMV, HSV-1/2, and leptospira. Despite empiric bacterial and antiviral (acyclovir) therapy, the patient developed worsening anasarca and acute liver injury, resulting in acute respiratory failure requiring mechanical ventilation and ICU transfer.Immunosuppressive therapy was started with intravenous immunoglobulin (IVIG) and glucocorticoids. A bone marrow biopsy subsequently confirmed hemophagocytic lymphohistiocytosis. Following a 5-day course of IVIG, weekly etoposide was added to the antibiotic and antiviral regimen. During the second week of treatment, serum PCR confirmed HSV-1 viremia while alternative infectious etiologies were negative, supporting HSV-1 as the likely trigger of HLH. Combination therapy of etoposide and acyclovir resulted in the patient's significant improvement with resolution of anasarca, hyperbilirubinemia, and heightened inflammation. She was discharged with multidisciplinary services follow-up plan for serial HSV-1 quantitative PCR, liver enzymes, and inflammatory markers.Discussion: This case illustrates an atypical and severe presentation of HSV-1-induced HLH in an immunocompetent young adult. HLH may be associated with infectious etiologies, including viral infections such as EBV and CMV and bacterial infections, as well as autoimmune diseases and malignancies. However, this case highlights the importance of considering other causes in healthy young adults and how prompt, aggressive combination therapy with immunosuppressants and antivirals can improve inflammation, particularly when these patients reach critically ill status and are at their most vulnerable state. Integrating clinical manifestations with objective findings can facilitate accurate diagnosis, even when the underlying etiology is rare.

ABSTRACT V-49

A Fall Too Small for Two Fractures

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Program: Centro Medico Episcopal San Lucas Transitional Year Residency Program

ABSTRACT

Femoral neck fractures in young adults are uncommon and typically follow high-energy trauma. Bilateral fractures after low-energy trauma should raise concern for underlying metabolic bone disease. Patients with end-stage renal disease (ESRD) are susceptible to mineral and bone abnormalities, including renal osteodystrophy and secondary hyperparathyroidism, which can profoundly compromise skeletal integrity. We present a young dialysis-dependent patient with bilateral displaced femoral neck fractures following a low-energy fall, highlighting a severe skeletal manifestation of longstanding mineral and bone disease. A 40-year-old woman with ESRD on maintenance hemodialysis, hypertension, diabetes mellitus, hypothyroidism, anemia, and parathyroid-associated metabolic bone disease presented after slipping in her bathroom and falling into a seated position, followed by inability to ambulate. She reported right lower-extremity discomfort for approximately one week preceding the fall. Examination demonstrated shortening and external rotation of the right lower extremity with painful limitation of motion. Pelvic radiography revealed bilateral transverse femoral neck fractures with superolateral displacement of the distal fracture fragments. Metabolic evaluation during the week of presentation demonstrated severe secondary hyperparathyroidism with intact parathyroid hormone >2,000 pg/mL and hyperphosphatemia of 6.6 mg/dL, while serum calcium remained approximately 9.0–9.3 mg/dL. Alkaline phosphatase had been markedly elevated at 787 U/L three months earlier. Her perioperative course was complicated by severe hyperkalemia reaching 7.0 mmol/L, requiring potassium-lowering therapy and additional hemodialysis before definitive surgical management. Following medical optimization, bilateral hip hemiarthroplasty was performed, with bone specimens obtained from both femoral heads. Histopathologic examination demonstrated renal osteodystrophy bilaterally. Hyperkalemia resolved with hemodialysis, and she was discharged with physical therapy, home-care services, and continued maintenance hemodialysis. At one-month follow-up, she was ambulating with a cane, reported minimal pain, and had no limitations in activities of daily living. This case demonstrates profound skeletal fragility associated with mineral and bone disease in a young patient with dialysis-dependent ESRD. Bilateral fractures after minimal trauma, intact parathyroid hormone >2,000 pg/mL, hyperphosphatemia, previously elevated alkaline phosphatase, and biopsy-confirmed renal osteodystrophy demonstrate severe secondary hyperparathyroidism with compromised skeletal integrity. Her preceding lower-extremity discomfort raises concern for preexisting skeletal compromise, although an insufficiency fracture before the fall cannot be established retrospectively. The case also demonstrates the internist's role beyond identifying the underlying metabolic disorder: severe hyperkalemia required urgent correction and additional hemodialysis before definitive orthopedic intervention could safely proceed. In young adults, fractures disproportionate to the mechanism of injury should prompt evaluation for secondary causes of skeletal fragility. In patients with ESRD, recognition and management of mineral and bone abnormalities and secondary hyperparathyroidism are essential to identifying patients at increased fracture risk and potentially preventing devastating skeletal complications.

ABSTRACT V-50

When pneumonia is blood: bronchoscopy uncovers diffuse alveolar hemorrhage in hematologic malignancy

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Program: San Juan City Hospital Program

ABSTRACT

IntroductionDiffuse alveolar hemorrhage (DAH) is a life-threatening pulmonary complication of hematologic malignancy, with reported mortality approaching 50–80% and exceeding 90% in patients requiring mechanical ventilation. Recognition is challenging because diffuse infiltrates, hypoxemia, fever, and anemia frequently mimic pneumonia or acute respiratory distress syndrome, while hemoptysis may be absent in up to one-third of patients. We describe DAH initially treated as severe pneumonia in a patient with acute leukemia and place the case within an institutional experience demonstrating the devastating mortality associated with this complication.Case PresentationA middle-aged woman with mixed-phenotype acute leukemia undergoing induction therapy developed rapidly progressive hypoxemic respiratory failure with diffuse bilateral pulmonary infiltrates. Severe pneumonia was initially suspected, and broad-spectrum antimicrobial therapy was initiated. Profound thrombocytopenia accompanied her hematologic malignancy; however, hemoptysis was absent, obscuring an alternative pulmonary hemorrhagic process.Despite antimicrobial therapy, oxygen requirements progressively increased without meaningful clinical improvement. The discordance between persistent respiratory deterioration and the presumed infectious diagnosis prompted flexible bronchoscopy.Sequential bronchoalveolar lavage aliquots became progressively more hemorrhagic, establishing DAH. Microbiologic studies were unrevealing, shifting the therapeutic focus away from isolated infection and toward pulmonary hemorrhage. Corticosteroids and supportive management were initiated; however, respiratory failure progressed to multiorgan dysfunction, and the patient ultimately died.The severity of this outcome reflected a broader institutional pattern. Among 10 patients with hematologic malignancies—including AML, ALL, APL, and mixed-phenotype acute leukemia—who developed DAH at our institution, nine died during hospitalization, corresponding to 90% in-hospital mortality. Bronchoscopy was frequently performed only after several days of progressive respiratory deterioration and empiric antimicrobial therapy.DiscussionThe diagnostic challenge in DAH is not always the absence of clues, but the overlap of those clues with infection. In immunocompromised patients, bilateral infiltrates and worsening hypoxemia appropriately trigger antimicrobial therapy; however, failure to improve should prompt reconsideration of the working diagnosis, particularly when thrombocytopenia or falling hemoglobin increases the probability of pulmonary hemorrhage.Our institutional experience demonstrates the extreme mortality accompanying DAH in hematologic malignancy but does not establish delayed bronchoscopy as the cause of mortality. Rather, the frequent delay in diagnostic BAL identifies an actionable opportunity for earlier diagnostic consideration. Sequentially hemorrhagic BAL can rapidly distinguish DAH from competing pulmonary processes while simultaneously permitting microbiologic evaluation in a population in whom infection and hemorrhage may coexist.ConclusionDAH should remain an early consideration in patients with hematologic malignancy who develop unexplained hypoxemia and diffuse pulmonary infiltrates, even without hemoptysis. Our institutional experience, with 90% in-hospital mortality among 10 affected patients, underscores the severity of this complication. Earlier consideration of bronchoscopy with sequential BAL may provide critical diagnostic clarity before progressive respiratory failure limits therapeutic options.

ABSTRACT V-51

A Preventable Relapse: Antiretroviral Therapy Gaps in a Gastrostomy-Fed Patient

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Program: University of Puerto Rico Program

ABSTRACT

Antiretroviral therapy administered via gastrostomy tube poses unique challenges, including limited formulation options, potential drug interactions with enteral feeds, and reliance on caregivers for consistent dosing and solution preparation. Many patients also frequently lack routine infectious disease follow-up once they become homebound, leaving virologic failure undetected until it presents as an opportunistic infection. We present a case in which these gaps converged, resulting in unrecognized multiclass antiretroviral resistance and a fatal relapse of cerebral toxoplasmosis.A 50-year-old bedridden, nonverbal male patient with past medical history of seizures, human immunodeficiency virus on antiretroviral therapy, remote history of cerebral toxoplasmosis 2014 with recurrence and cerebrovascular accident in 2015 and residual right hemiparesis, was transferred to our institution for suspected recurrence of cerebral toxoplasmosis. Patient presented with fever, lethargy, and new left-sided weakness for one week for which he was taken to an outside hospital. Imaging showed innumerable ring-enhancing brain lesions with edema and midline shift suggestive of cerebral toxoplasmosis. He had not seen an infectious disease specialist in years and his HIV was followed by his primary care physician. His CD4 count and viral load had not been checked in over two years. Patient has been receiving Raltegravir/Emtricitabine/Tenofovir via gastrostomy tube since 2020 for all nutrition and medication administration. Patient's mother reports she had not missed a single antiretroviral dose since HIV diagnosis. Admission laboratory testing revealed a CD4 count of 19 and viral load exceeding 500,000 copies/mL, indicating virologic failure despite reported home adherence to his antiretroviral regimen. While hospitalized, patient improved on empiric antitoxoplasmosis therapy, but genotypic resistance testing later revealed mutations conferring resistance to his entire nucleoside backbone and to the integrase inhibitor class, while non-nucleoside and protease inhibitors remained fully active. His regimen was changed to a non-nucleoside/protease inhibitor (Etravirine/Darunavir), at hospital, but his hospitalization was complicated by aspiration pneumonia and septic shock, and he died before the revised regimen could take effect.This case highlights how administration of antiretroviral therapy through a gastrostomy tube, when not specifically reviewed for crushability, formulation compatibility, and interaction with enteral feeds, can result in unrecognized subtherapeutic dosing and progressive resistance. As of today, there are only a handful of case reports studying efficacy and bioavailability of antiretrovirals administered via enteral tube feedings and there are no comprehensive pharmacokinetic studies published. Compounding this risk, homebound and dependent patients with HIV are especially prone to falling out of infectious disease follow-up, since routine CD4 count and viral load monitoring often depends on specialist visits rather than primary care alone. By the time this patient's virologic failure became clinically apparent, resistance had already developed across two drug classes, leaving few effective options once his infection recurred. Clinicians managing HIV in patients with gastrostomy tubes should confirm that antiretroviral formulations are appropriate for enteral administration, and should ensure scheduled infectious disease follow-up and periodic virologic monitoring are maintained even when patients are homebound, in order to detect treatment failure before resistance accumulates and opportunistic disease recurs.

ABSTRACT V-52

Not Every NSTEMI Is Atherosclerotic: Spontaneous Coronary Artery Dissection in a 76-Year-Old Woman

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ABSTRACT

Intruduction Spontaneous coronary artery disection (SCAD) is a nonatherosclerotic, nontraumatic, and noniatrogenic cause of acute coronary syndrome characterized by the spontaneous separation of the layers of an epicardial coronary artery wall. However, unlike conventional acute coronary syndromes, myocardial injury in SCAD results primarily from coronary obstruction caused by intramural hematoma or intimal disruption rather than atherosclerotic plaque rupture and thrombosis. SCAD may account for up to 35% of MIs in women ≤50. Although SCAD is classically associated with younger women, it should remain in the differential diagnosis of ACS in older women, including those with traditional cardiovascular risk factors.Case PresentationOur patient is a 76-year-old female with a past medical history of osteoporosis, hypertension, hyperlipidemia and trigeminal neuralgia that presents to the ER for ongoing oppressive chest pain, rated 10/10, weakness on the left arm diaphoresis and shortness of breath. The pain persisted despite nitroglycerine and aspirin administration. She denies alcohol, tobacco or illicit drug use. Family history is significant for father passing away during sleep of unknown cause at the age of 42 and mother with coronary artery disease requiring CABG. She had elevated high sensitivity troponin-I (67,216.3 pg/mL), cholesterol (286 mg/dL) and LDL (206 mg/dL). She had a working diagosis of NSTEMI and managed with DAPT, high intensity statins and sublingual nitroglycenrin. The patient was taken to cath lab for LHC and was found to have SCAD involving the distal obtuse marginal branch with preserved distal flow and no significant atherosclerotic obstructive coronary disease. The patient was discharged home after resolution of symptoms, treated with DAPT and conservative management, avoiding PCI. DiscussionA 76-year-old woman with hypertension, severe hyperlipidemia, and a clinical NSTEMI phenotype would naturally be presumed to have atherosclerotic ACS, yet angiography revealed spontaneous coronary artery dissection. The patient was older that the typical age of SCAD and she had no known fibromuscular disorders. Although SCAD frequently presents as an acute myocardial infarction, its nonatherosclerotic pathophysiology has important diagnostic and therapeutic implications compared with conventional plaque-mediated ACS. It is important to raise awareness of this condition as it is often overlooked, especially in cases such as this one with other atherosclerotic risk factors. Distinguishing SCAD-related myocardial infarction from atherosclerotic ACS is critical because management differs substantially and applying standard atherosclerotic ACS management to SCAD can be actively harmful. SCAD management shifts focus from aggressive antithrombotic/revascularization strategies toward conservative management, because the vast majority lesions heal spontaneously. There is also higher level of complications after PCI in patients with SCAD when compared to NSTEMI. High intensity statins are not recommended unless there is coexisting dyslipidemia. Regarding DAPT, there is no consensus on the approach for management in SCAD and ranges from DAPT to aspirin alone. Most importantly, once SCAD is identified, systemic anticoagulation is generally discontinued in the absence of another indication, and fibrinolysis should be avoided because of the potential for extension of intramural hemorrhage. Finally, this patient should also be screened for other conditions such as FMD and arteriopathies.

ABSTRACT V-53

A Bladder Under Pressure: Spontaneous Extraperitoneal Rupture of a Bladder Diverticulum Causing Dialysis-Requiring Acute Kidney Injury

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Program: University of Puerto Rico Program

ABSTRACT

Introduction Spontaneous (idiopathic) rupture of the urinary bladder (SRUB) is a rare, potentially fatal event. In the largest systematic review, rupture was intraperitoneal in approximately 89% of cases, usually at the dome. Extraperitoneal rupture through a bladder diverticulum is exceptionally uncommon, with only isolated reports in literature. We describe spontaneous extraperitoneal rupture of a bladder diverticulum producing extensive retroperitoneal urinary extravasation and severe acute kidney injury (AKI) requiring temporary hemodialysis. Case Presentation A 62-year-old man with no known medical conditions presented with five days of progressive lower abdominal pain following an episode of urinary retention, without recent abdominopelvic trauma. Further history revealed prolonged anabolic-androgenic steroid use intermittently for approximately 35 years. Laboratory evaluation was notable for marked leukocytosis (17.24×10^3/µL) and mild normocytic anemia (hemoglobin 11.2 g/dL). Renal indices revealed severe azotemia (creatinine 11 mg/dL, BUN 125 mg/dL) with hyperkalemia, metabolic acidosis, hyperphosphatemia, and hyponatremia. Urinalysis demonstrated hematuria, pyuria, and bacteriuria with positive nitrites and leukocyte esterase, indicating concurrent urinary tract infection. Diagnosis was established by CT cystography, which demonstrated a 0.6 × 0.6-cm defect in the left posterolateral bladder wall with extensive extraperitoneal contrast extravasation into the retroperitoneum and left thigh. Multiple bladder diverticula and moderate bilateral hydroureter were also identified, suggesting chronic outlet obstruction as the underlying substrate. Initial management consisted of Foley catheter placement for maximal bladder decompression and urinary diversion, with hemodialysis on three occasions until intrinsic renal function recovered. The hospital course was complicated by Proteus mirabilis urinary tract infection and bacteremia, managed with broad-spectrum intravenous antibiotics. Serial imaging tracked the evolving retroperitoneal collection: a large retroperitoneal urinoma/hematoma without active hemorrhage and persistent diverticular rupture on repeat cystography. Given the absence of active bleeding and the extraperitoneal nature of the injury, management remained conservative with prolonged catheter drainage rather than operative repair. In outpatient setting, follow-up CT cystography ultimately demonstrated complete resolution of contrast extravasation, confirming healing of the bladder wall defect. Persistent detrusor hypertrophy, trabeculation, bilateral hydroureter, and probable prostatomegaly implicated longstanding bladder outlet obstruction as the underlying etiology; outpatient urologic evaluation for benign prostatic hyperplasia and bladder outlet obstruction was arranged. At nephrology follow-up, renal function had improved but remained impaired (creatinine 2.17 mg/dL; estimated glomerular filtration rate 33 mL/min/$1.73m^2$), consistent with chronic kidney disease stage 3b. Discussion Chronic bladder outlet obstruction produces elevated intravesical pressure, detrusor hypertrophy, trabeculation, and acquired diverticula, which are structurally weak zones prone to rupture during acute retention. This case illustrates a rare extraperitoneal SRUB through a diverticulum with retroperitoneal extension and profound renal dysfunction. Although the patient reported prolonged anabolic-androgenic steroid use, its contribution remains speculative. Conclusion Spontaneous bladder rupture should be considered in patients with abdominal pain, urinary retention, severe AKI, and imaging evidence of chronic outlet obstruction, even without trauma. CT cystography is essential for diagnosis and monitoring of healing, while early urinary decompression may avert further renal and infectious complications.

ABSTRACT V-54

Beyond the Prescription: Social Determinants of Health in a Hyperosmolar Hyperglycemic Crisis

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ABSTRACT

IntroductionSocial determinants of health (SDOH) can profoundly influence a patient's ability to implement medical recommendations after hospital discharge. This becomes particularly important when new functional limitations transfer responsibility for complex medical care from the patient to a caregiver. We present a case of severe hyperosmolar hyperglycemic state (HHS) in which limited caregiver literacy, inadequate preparation for insulin administration, and a challenging transition from hospital to home contributed to interruption of essential diabetes therapy.Case DescriptionA 71-year-old man with recently diagnosed type 2 diabetes mellitus requiring insulin therapy and a recent ischemic stroke presented to the emergency department after he was found unresponsive at home. Following his stroke, he became bedbound and dependent on his wife for activities of daily living and medication administration. He was discharged home five days before presentation after a three-week hospitalization with prescriptions for insulin glargine 60 units daily, insulin lispro 12 units twice daily, statin and dual antiplatelet therapy.Due to his functional limitations, the patient was unable to self-administer insulin and relied on his wife as his primary caregiver. His wife had limited literacy and reported that she did not receive hands-on instruction regarding insulin administration before discharge. Consequently, the patient received no insulin after returning home. His wife subsequently noticed increased urinary frequency. On the day of presentation, during his first home-care nursing visit since discharge, he was found unresponsive and markedly tachypneic, with a respiratory rate of 48 breaths/min and a glucometer reading "High," prompting transport to the emergency department by emergency medical services.Initial laboratory evaluation revealed a serum glucose of 969 mg/dL, serum osmolality of 364 mOsm/kg, sodium of 165 mEq/L, creatinine of 3.14 mg/dL, and lactate of 15–17 mmol/L. His clinical course was complicated by shock and acute hypoxemic respiratory failure requiring intubation, mechanical ventilation, vasopressor support, intravenous fluids, and continuous insulin infusion. Chest imaging demonstrated lower-lobe consolidation and tree-in-bud opacities concerning for aspiration-related infection, representing an additional potential precipitant. His presentation was consistent with severe HHS complicated by acute kidney injury, shock, and respiratory failure.DiscussionThis case illustrates how SDOH and functional limitations can determine whether an otherwise appropriate treatment plan can be safely implemented after discharge. Following his stroke, the patient's new dependence shifted responsibility for insulin administration to a caregiver with limited literacy who reported not receiving practical insulin training. Although aspiration-related infection likely contributed to his metabolic decompensation, interruption of insulin therapy represented an important and potentially modifiable precipitant. This case highlights an opportunity to strengthen transitions of care by ensuring that patients and caregivers receive individualized education before discharge and demonstrate their ability to safely administer prescribed therapies. Literacy-sensitive education, hands-on demonstration, teach-back, assessment of caregiver readiness, and timely coordination of home-health services may help identify and address barriers before patients return home. Safe transitions of care require not only prescribing appropriate therapy, but also confirming that patients or caregivers have the knowledge, skills, and resources necessary to carry it out.

ABSTRACT V-55

Beyond the Tumor: Extensive Venous Thrombosis in Malignant SVC Syndrome

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Program: San Juan VA Medical Center

ABSTRACT

Superior vena cava (SVC) syndrome is a well-recognized complication of thoracic malignancy that is often attributed to direct tumor compression. However, concomitant extensive venous thrombosis may substantially worsen venous obstruction and represents a potentially treatable contributor to refractory symptoms. We present the case of a progressive SVC syndrome in a patient with dual malignancies whose symptoms persisted despite cancer-directed therapy and was ultimately found with extensive bilateral upper-extremity and central venous thrombosis requiring endovascular intervention.A 48 year-old-male with stage IIIC lung adenocarcinoma and a mesenteric neuroendocrine tumor was referred from radiation oncology for progressive dyspnea on exertion, facial and neck congestion, and right arm edema extending to the chest and face. He had multiple prior emergency department visits for similar symptoms and had been diagnosed with SVC syndrome secondary to a right upper lobe mass seen on arterial phase CT angiography. He was treated with carboplatin and pemetrexed and received radiation therapy, but his symptoms showed minimal improvement. On arrival he had facial plethora, prominent chest wall collaterals, and right upper extremity edema. Duplex ultrasonography of the right upper extremity was performed due to swelling and demonstrated thrombosis of the right axillary, subclavian, and internal jugular veins. He was started on corticosteroids and was admitted to the ICU for airway monitoring.Catheter-directed venography revealed a more extensive vascular process than initially appreciated, demonstrating marked tumor-mediated SVC compression with extensive bilateral thrombosis involving the internal jugular, brachial, axillary veins, SVC, and left innominate vein. Additional thrombus extended around the right internal jugular infusion port. Endovascular therapy consisted of local tissue plasminogen activator infiltration, rheolytic mechanical thrombectomy, balloon maceration and venoplasty, and manual aspiration. Central venous flow was reestablished, although a residual mural thrombus persisted given the clot burden and device limitations. The patient was transitioned from intravenous heparin to therapeutic enoxaparin and then to apixaban. His edema and dyspnea improved, and he was discharged to continue oncologic care.This vignette highlights a practical internal medicine approach when malignant SVC syndrome is slow to respond to tumor-directed therapy. Contrast-enhanced CT is commonly used for initial evaluation of SVC syndrome. However, the extent of venous thrombosis may be incompletely characterized on an arterial-phase study. In patients with active malignancies, indwelling central venous devices, or symptoms refractory to tumor-directed therapy, considering both arterial and venous-phase imaging may help identify a concomitant thrombotic component. Recognizing and treating this reversible contributor can be critical when symptoms persist despite appropriate oncologic therapy.

ABSTRACT V-56

The Anergy Trap: Active Pulmonary and Extrapulmonary Tuberculosis Presenting with an Unclassified Mediastinal Mass and False-Negative IGRAs

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Program: University of Puerto Rico Program

ABSTRACT

Introduction: Mycobacterium tuberculosis (TB) remains a formidable health challenge. According to the CDC, TB maintains a global incidence rate of 131 cases per 100,000 population, translating to roughly 10.7 million new cases annually. The United States and Puerto Rico are classified as low-incidence regions, reporting rates of 3.0 and 0.7 cases per 100,000 residents, respectively. While TB primarily manifests as a pulmonary disease, cervical tuberculous lymphadenitis (CTL), medically termed scrofula, is the most prevalent extrapulmonary manifestation. Because CTL can clinically mimic other chronic infections or malignancies like lymphoma or Nocardia, establishing a definitive diagnosis remains challenging. Despite its low incidence in Puerto Rico, TB warrants strong consideration in the differential diagnosis of chronic cervical lymphadenitis, even when screening tools yield negative results. Notably, interferon-gamma release assays (IGRAs) demonstrate a false-negative rate of 10% to 18% in extrapulmonary cases. We present a compelling case of an IGRA-negative geriatric patient who developed scrofula and subsequently manifested active pulmonary tuberculosis. Case Presentation: An 82-year-old previously healthy female presented with a 6-month history of progressive, bilateral suppurative cervical lymphadenopathy. Her illness began in January 2026 with a left pre-auricular lesion, initially biopsied as actinic keratosis without malignancy. The lesion slowly progressed into a necrotic abscess. Consecutive fine-needle aspirations (FNAs) demonstrated granulomatous inflammation and suppurative abscess formation. By June 2026, the lesions advanced bilaterally with persistent suppuration despite oral amoxicillin-clavulanate and clindamycin. Repeat FNA biopsies revealed extracellular acid-fast bacilli (AFB) with negative fungal stains and no malignancy. An initial IGRA was negative, raising suspicion for nontuberculous mycobacteria or Nocardia. The patient subsequently developed a productive cough, anorexia, and a 15-lb weight loss. She was admitted for inpatient workup. Serologic testing for HIV, RPR, viral hepatitis, and respiratory viral panels were negative. Repeated inpatient IGRA and PPD skin tests were also negative. Neck and chest CT revealed extensive necrotic cervical, supraclavicular, axillary, and mediastinal lymphadenopathy, alongside an active pulmonary process and an anterior mediastinal mass measuring 4.4 x 5.4 x 5.3 cm. Differential diagnosis for this mediastinal mass included thymoma, malignancy, or tuberculoma. Interventional radiology was consulted for a core biopsy of the mass and lymph nodes, but the procedure was postponed because her sputum test returned positive for AFB; PCR assays confirmed Mycobacterium tuberculosis. The patient was placed in airborne precautions and started on RIPE (rifampin, isoniazid, pyrazinamide, ethambutol) plus pyridoxine. She's currently tolerating treatment. Discussion: This case highlights the challenges of diagnosing CTL, particularly within a low-incidence region, as it can mimic bacterial abscesses, atypical mycobacteria, Nocardia, or lymphoma. While IGRAs possess high specificity, sensitivity is heavily compromised in geriatric populations due to immunosenescence. Relying entirely on these immunologic markers can delay vital airborne isolation and treatment. Tuberculosis should therefore remain in the differential diagnosis of chronic head and neck lymphadenopathy unresponsible to antibiotics, even when screening tests are negative. Sputum PCR testing remains paramount to avoid diagnostic and treatment delays.

ABSTRACT V-57

From Chest Pain to Hemorrhagic Pancreatitis and Splenic Infarction: A Diagnosis That Would Not Settle

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Program: Centro Médico Menonita Cayey Program

ABSTRACT

Introduction: Vascular complications are common in acute pancreatitis. Arterial disease as a cause, however, is rarely considered. One such lesion is celiac artery dissection, which usually coexists with dissection of a neighboring visceral artery and is most often found incidentally on computed tomography obtained for another indication. Pancreatitis as its presenting manifestation is rarely described. Case presentation: A 49-year-old woman presented to another institution following an episode of sudden central chest pain at rest lasting one hour, and was admitted with a diagnosis of non-ST-elevation myocardial infarction (NSTEMI). Aspirin, prescribed after an NSTEMI in 2023, had been stopped at home for intolerance; it was restarted with enoxaparin for the current event. She was transferred to our institution on hospital day 6 for catheterization. On arrival her blood pressure was 71/40 mmHg. Catheterization later that morning showed patent coronary arteries. Sinus tachycardia began that afternoon and persisted, reaching 122 beats per minute on day 7, when she developed progressive abdominal pain and hemoglobin fell from 13.5 to 7.5 g/dL. Computed tomography (CT) of the abdomen and pelvis showed acute hemorrhagic pancreatitis, splenic artery and vein thrombosis, more than 50% splenic infarction and hemoperitoneum. Aspirin and enoxaparin were discontinued; she was transferred to the intensive care unit and received four units of packed red blood cells and two of fresh frozen plasma. On day 8 amylase and lipase were normal (56 and 58 U/L, respectively); she met two of three revised Atlanta criteria on pain and imaging. Anticoagulation was recommended for the presumed thrombosis but declined because she was bleeding. Repeat CT on day 10 reclassified the lesion as splenic artery dissection, unchanged on CT angiography on day 11. Mesenteric angiography on day 14 showed a dissection originating within the celiac artery and extending into the splenic artery, with preserved antegrade flow and no extravasation or pseudoaneurysm. Stenting was not attempted because the dissection arose near the hepatic artery origin. The spleen was preserved and anticoagulation withheld throughout. Clopidogrel was started on day 17, and she was discharged on day 21 with a hemoglobin of 12.8 g/dL. Discussion: The lesion responsible for the pancreatitis and splenic infarction was identified only at mesenteric angiography on day 14, after three cross-sectional studies had not localized it. A likely explanation is that a dissection in a small-caliber artery lying within its own bed of inflammation and hemorrhage is easily obscured, and repeating the same modality did not resolve it. The initial interpretation carried a therapeutic consequence: anticoagulation was recommended for the presumed thrombosis and deferred only because she was bleeding. Had the dissection been recognized from the outset, that question would have been framed differently. Localization also determined what could be offered, since a dissection arising beside the hepatic artery origin could not be safely stented. We therefore suggest that in hemorrhagic pancreatitis without a conventional cause, particularly with disproportionate splenic infarction or non-diagnostic enzymes, dedicated vascular imaging should be obtained early and catheter angiography considered when cross-sectional studies are discordant or inconclusive.

ABSTRACT V-58

Beyond Papilledema: A Reversible Cause of Bilateral Optic Disc Edema From Severe Multinutrient Deficiency in Autism Spectrum Disorder

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ABSTRACT

Bilateral optic disc edema is an ophthalmologic emergency that typically prompts evaluation for elevated intracranial pressure, inflammatory optic neuropathies, demyelinating disease, or intracranial mass lesions. Nutritional optic neuropathy is an uncommon but potentially reversible cause that may be overlooked, particularly in patients with restrictive eating behaviors. This case highlights the importance of avoiding premature diagnostic closure and demonstrates how obtaining a detailed dietary history can lead to the identification of a vision-threatening yet treatable condition. A 24-year-old man with autism spectrum disorder presented with a 10 -day history of progressively worsening bilateral eye pain, headaches, and rapidly declining vision. He was initially diagnosed with blepharitis and treated with topical antibiotics without improvement. Neuro-ophthalmologic examination demonstrated visual acuity of 20/400 in the right eye and 20/65 in the left, normal intraocular pressures (12 mmHg bilaterally), no relative afferent pupillary defect, and 360° bilateral optic disc edema. Funduscopic examination revealed diffuse whitish retinal lesions throughout the mid-peripheral retina bilaterally. Optical coherence tomography (OCT) demonstrated marked retinal nerve fiber layer thickening (235 µm OD, 263 µm OS). Further history revealed a profoundly restrictive diet related to sensory aversions from autism spectrum disorder. For several years, he consumed fast-food chicken nuggets at nearly every meal with virtually no fruits, vegetables, dairy products, or vitamin supplementation. He denied recent illness, toxin exposure, gastrointestinal symptoms, or medications associated with intracranial hypertension. MRI of the brain and orbits with MR venography demonstrated no intracranial abnormalities, optic nerve enhancement, or cerebral venous sinus thrombosis. Cerebrospinal fluid analysis was normal. Extensive infectious, inflammatory, and autoimmune evaluation, including ANA, MOG antibody, ACE level, Lyme serology, syphilis testing, and QuantiFERON-TB, was unrevealing. Laboratory studies demonstrated profound deficiencies of vitamin A (3), vitamin D (6 ng/mL), vitamin B12 (186 pg/mL), vitamin B6 (2.3), and gamma vitamin E (0.6). High-dose micronutrient replacement, including vitamin A supplementation, was initiated. One week later, visual acuity improved to 20/20 bilaterally with persistent but improved optic disc edema. Repeat OCT demonstrated decreasing retinal nerve fiber layer thickness (215 µm OD, 240 µm OS), consistent with early structural recovery. This case illustrates the diagnostic complexity of bilateral optic disc edema and the importance of systematic clinical reasoning. Although initial evaluation appropriately prioritized elevated intracranial pressure, cerebral venous sinus thrombosis, inflammatory optic neuritis, and demyelinating disease, unrevealing neuroimaging, cerebrospinal fluid studies, and extensive laboratory testing prompted consideration of less common etiologies. A comprehensive dietary history proved to be the critical diagnostic clue, identifying severe multinutrient deficiency in a patient with autism spectrum disorder and longstanding restrictive eating behaviors. Rapid visual recovery following vitamin replacement supported the diagnosos and highlighted its reversibility. Nutritional optic neuropathy should remain in the differential diagnosis of bilateral optic disc edema after common neurologic, inflammatory, and infectious causes have been excluded. Early recognition through careful dietary assessment and prompt micronutrient replacement can preserve vision, prevent permanent neurologic sequelae, and avoid unnecessary diagnostic testing.

ABSTRACT V-59

Not Every Acute Kidney Injury in Multiple Myeloma Is Disease Progression: A Diagnostic Challenge of Drug-Induced Thrombotic Microangiopathy, Tumor Lysis Syndrome, and Secondary Plasma Cell Leukemia

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ABSTRACT

IntroductionThrombotic microangiopathy (TMA) is a life-threatening syndrome characterized by microangiopathic hemolytic anemia, thrombocytopenia, and end-organ injury. Drug-induced TMA is uncommon and particularly challenging to recognize in patients with hematologic malignancies, in whom acute kidney injury (AKI) and cytopenias have multiple competing etiologies. Carfilzomib, a proteasome inhibitor used in relapsed multiple myeloma (MM), has been associated with TMA, with evidence suggesting complement activation in its pathogenesis.[1] We describe a patient with relapsed standard-risk MM who developed multiple concurrent complications shortly after initiating a new treatment regimen, creating a complex diagnostic challenge.Case PresentationA 51-year-old woman with standard-risk IgG kappa MM, previously treated with autologous stem cell transplantation, developed biochemical relapse and was started on isatuximab and carfilzomib. She subsequently presented with malaise, right facial paresthesias, and progressive renal dysfunction. Laboratory evaluation revealed severe AKI (creatinine 4.29 mg/dL), thrombocytopenia (52 × 10^3/µL), anemia, hyperkalemia, hyperuricemia, and hypocalcemia. Coagulation studies were normal, direct antiglobulin testing was negative, ADAMTS13 activity exceeded 100%, and soluble C5b-9 was markedly elevated (899.6 ng/mL). Peripheral smear demonstrated schistocytes and circulating plasma cells. Peripheral blood flow cytometry demonstrated 20% plasma cells, while bone marrow biopsy revealed >90% plasma cell replacement with plasmablastic features. FISH analysis showed standard-risk cytogenetics without conventional high-risk abnormalities.AssessmentCoombs-negative hemolytic anemia, thrombocytopenia, AKI, schistocytosis, preserved ADAMTS13 activity, and elevated soluble C5b-9 supported TMA, with carfilzomib identified as the suspected culprit requiring its discontinuation. Concurrent hyperuricemia, hyperkalemia, hypocalcemia, and AKI supported TLS, while 20% circulating plasma cells confirmed transformation to sPCL. The patient received plasmapheresis, rasburicase, and chemotherapy. Due to inadequate response to plasmapheresis, ravulizumab, a terminal complement inhibitor, was initiated. Subsequent suppression of CH50 supported effective complement blockade. Chemotherapy and ravulizumab achieved TMA resolution and disease control.DiscussionThis case highlights the rare coexistence of three life-threatening processes: drug-induced TMA, TLS, and transformation of standard-risk MM to sPCL. Their overlapping renal, hematologic, and metabolic manifestations create substantial potential for diagnostic anchoring. Preserved ADAMTS13 activity, schistocytosis, and terminal complement activation supported TMA, while circulating plasma cells revealed concurrent malignant progression. Notably, progression to sPCL occurred despite the absence of conventional high-risk cytogenetic abnormalities, demonstrating that standard-risk cytogenetics do not preclude aggressive disease evolution. Timely recognition of these concurrent diagnoses allowed all three conditions to be addressed simultaneously.ConclusionIn patients with MM presenting with AKI and cytopenias, multiple etiologies, including treatment toxicity, metabolic complications, and disease progression, may coexist. This case demonstrates the importance of avoiding diagnostic anchoring and maintaining a broad differential when multiple life-threatening processes collide. Reference:Blasco, M., Martínez-Roca, A., Rodríguez-Lobato, L. G., Garcia-Herrera, A., Rosiñol, L., Castro, P., & Palomo, M. (2021). Complement as the enabler of carfilzomib-induced thrombotic microangiopathy. British Journal of Haematology, 193(1), 181-187.

ABSTRACT V-60

Autoimmune limbic encephalitis with concurrent systolic heart failure and left ventricular thrombus

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ABSTRACT

IntroductionPersistent amnesia and behavioral change after seizure control may signal autoimmune limbic encephalitis rather than an isolated postictal state. Coexisting heart failure and ventricular thrombosis add diagnostic and therapeutic complexity.Case DescriptionA 30-year-old woman with remote childhood seizures presented with recurrent adult seizures despite levetiracetam. She was afebrile, without meningismus or focal deficits. Admission metabolic testing and head computed tomography were unrevealing; magnetic resonance imaging (MRI) on hospital day 3 was normal. Levetiracetam was increased to 1,500 mg twice daily. Overt seizures were no longer reported, but disorientation, repetitive speech, insomnia, behavioral withdrawal, and recent-memory impairment persisted.Cerebrospinal fluid (CSF) contained 7 leukocytes/µL, all mononuclear, with reported normal glucose and protein and negative bacterial culture. On day 6, marked tachycardia, hypoxemia, hypotension, and oliguria prompted intensive care transfer. Bilateral pulmonary consolidations and effusions accompanied respiratory failure requiring mechanical ventilation. Empiric acyclovir and antibiotics were administered, and intravenous methylprednisolone 500 mg twice daily was given for five days after specialist evaluation.Cardiac evaluation documented systolic heart failure with a reported ejection fraction of approximately 30% and left ventricular thrombus. Rivaroxaban, candesartan, a beta-blocker, and spironolactone were instituted. These cardiac findings preceded intravenous immunoglobulin therapy.She was extubated by day 14. CSF N-methyl-D-aspartate receptor antibodies were documented as negative by day 15. Repeat MRI on day 16 demonstrated mild bilateral hippocampal and parahippocampal signal abnormalities; peri-ictal changes remained in the imaging differential. Neurology diagnosed autoimmune limbic encephalitis using the persistent limbic syndrome, CSF pleocytosis, imaging, and assessment of alternatives. Intravenous immunoglobulin, 2 g/kg over five days, was added for residual cognitive and behavioral symptoms. Infectious Disease discontinued acyclovir after reassessment and herpes simplex virus polymerase chain reaction was subsequently reported negative. By day 21, orientation to person, place, and time had improved, and discharge was planned. Long-term cognitive and cardiac outcomes were unavailable.DiscussionThis case illustrates a clinical diagnosis within the Graus framework despite initially normal imaging and negative testing for one neuronal antibody. Persistent amnesia and behavioral dysfunction after seizure control warranted serial neurological reassessment, while CSF and bilateral mesial temporal findings supported the diagnosis. Concurrent ventricular dysfunction and thrombus required separate treatment and consideration of infusion volume and thromboembolic risk during immunotherapy. Their coexistence did not establish autoimmune cardiomyopathy. However, further research and investigation should focus on limbic symptoms while independently evaluating major cardiac abnormalities and comparing them to systemic deterioration related to dysautonomia.

ABSTRACT V-61

Beyond Tachycardia: Dilated Cardiomyopathy as a Consequence of Prolonged Untreated Thyrotoxicosis

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Program: Centro Médico Menonita Cayey Program

ABSTRACT

Introduction:Thyroid hormone is a master regulator of cellular energetics, and sustained excess imposes a profound metabolic burden across virtually every organ system. Nowhere is this physiologic strain more dramatic than in the heart. Reduced systemic vascular resistance, activation of the renin–angiotensin–aldosterone system, and expansion of circulating volume drive a hyperdynamic state in which cardiac output may rise by as much as 300%. Initially adaptive, this chronic hyperkinetic, volume-loaded state promotes eccentric ventricular remodeling; when prolonged metabolic and hemodynamic demands exceed compensatory reserve, contractility deteriorates, transforming high-output physiology into dilated cardiomyopathy and systolic heart failure. Thyrotoxic dilated cardiomyopathy develops in approximately 1% of patients. Severe thyrotoxicosis may extend beyond the heart, producing gastrointestinal and neuropsychiatric manifestations and ultimately profound multisystem decompensation.Case PresentationA 55-year-old woman with hypertension presented to the emergency department febrile, disheveled, acutely disoriented, and delusional, with persistent diarrhea and an irregularly irregular rhythm. Laboratory evaluation revealed free T4 >6.99 ng/dL, total T4 >24.90 µg/dL, free T3 7.18 pg/mL, and free T4 index 16.19. Thyroid storm was supported clinically by a Burch-Wartofsky Point Scale of 55, prompting treatment with propylthiouracil, hydrocortisone, propranolol, and cholestyramine. Further evaluation revealed atrial fibrillation, and echocardiography demonstrated severe dilated cardiomyopathy with an ejection fraction of 20%, biatrial enlargement, and regurgitation across all four cardiac valves, consistent with functional regurgitation secondary to chamber and annular dilation. Coronary angiography demonstrated no obstructive coronary artery disease (40% mid-left anterior descending stenosis), supporting a nonischemic cardiomyopathy attributable to thyrotoxicosis, likely compounded by atrial fibrillation–mediated tachycardia. Subsequent review of her medical record revealed a nuclear thyroid uptake study performed three months before presentation demonstrating markedly elevated 75% uptake; despite this documented hyperthyroid state, she had received neither antithyroid therapy nor beta-blockade. She was initiated on guideline-directed medical therapy, referred for a wearable cardioverter-defibrillator, evaluated by electrophysiology for rhythm management, and scheduled for endocrinology follow-up.DiscussionAdvanced end-organ manifestations of untreated thyrotoxicosis, including severe dilated cardiomyopathy, atrial fibrillation, and psychosis occurring concurrently, are now uncommon where effective antithyroid therapy is readily available. Although thyrotoxic cardiomyopathy is potentially reversible, recovery is not guaranteed and appears strongly influenced by disease duration and the extent of ventricular remodeling. Importantly, the abnormal uptake study three months before presentation establishes only the minimum documented duration of thyrotoxicosis, not its onset; cumulative exposure to excess thyroid hormone may have been substantially longer. Longer untreated disease duration, severe systolic dysfunction, and ventricular dilation have been associated with incomplete recovery. This case underscores how delayed treatment may allow an initially compensatory hyperdynamic state to progress to potentially irreversible structural cardiac disease and profound multisystem decompensation. Prompt restoration of euthyroidism is essential to maximize the likelihood of recovery.

ABSTRACT V-62

Not Every Painless Jaundice Is Malignancy: Lemmel Syndrome in an Elderly Man with Metastatic Prostate Cancer

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Program: Not provided

ABSTRACT

Lemmel Syndrome is a rare cause of obstructive jaundice in which a duodenal diverticulum that arises 2-3 cm from the ampulla of Vater, a periampullary duodenal diverticulum, causes mechanical compression of the distal common bile duct (CBD), causing dilatation of the biliary and pancreatic ducts and a biliary obstruction in the absence of choledocholithiasis, a tumor, or other common causes.An 89-year-old male with type 2 diabetes mellitus, hypertension, incomplete C6 tetraplegia, prior cholecystectomy, and metastatic prostate cancer presented to the ER due to a 3-day history of generalized pruritus and jaundice along with abnormal liver function tests. He denied fever, abdominal pain, unwanted weight loss, melena, hematochezia, and stool color change. Physical examination showed scleral icterus with a nondistended, soft, and nontender abdomen.Laboratory studies showed a total bilirubin of 11.8 mg/dL with direct predominance of 7.8 mg/dL, ALP of 390 U/L, GGT of 893 U/L, AST of 62 U/L, and ALT of 106 U/L, with an R-factor of 0.8 consistent with a cholestatic pattern. Lipase and coagulation studies were normal. CA 19-9 was <5 U/mL, and CEA was 3.8 ng/mL. Abdominal ultrasound and a contrast-enhanced abdominopelvic CT revealed a CBD measuring between 8-9 mm with no intrahepatic ductal dilation or hepatic lesions. Given the initial imaging results and the patient's clinical presentation, the Gastroenterology service was consulted, and an MRCP was recommended. MRCP demonstrated mild central intrahepatic biliary dilation, fusiform dilation of the CBD to 1cm cranial to the pancreas, and a 2.2 x 2.2cm periampullary duodenal diverticulum abutting the distal CBD with abrupt ductal cutoff at that level, no CBD filling defects, and a normal-caliber pancreatic duct.ERCP the following day identified an intradiverticular papilla with a normal-appearing major papilla that was successfully cannulated. A 4mm biliary sphincterotomy was performed due to the suspicion of Lemmel Syndrome. Complete opacification of the biliary tree showed no stones or strictures and mild ductal dilation attributable to the patient's prior cholecystectomy. Total bilirubin decreased post-sphincterotomy to 10.8 mg/dL on post-procedure day 0 before peaking to 19.6 mg/dL on post-procedure day 3, which can be consistent with post-sphincterotomy papillary edema. By day 4 post-ERCP, total bilirubin decreased to 13.6 mg/dL, with improvement in ALP (395 to 312 U/L), AST (68 to 38 U/L), and ALT (115 to 61 U/L).Periampullary duodenal diverticula are reported in around 23% of patients, with only around 5% causing clinical symptoms of obstructive jaundice through diverticular compression of the distal CBD or chronic papillary inflammation. This case highlights numerous points. First, ultrasound and CT may be falsely reassuring, and biochemical evidence of cholestasis with non-dilated ducts should prompt MRCP rather than reassurance. Second, painless jaundice of cholestatic pattern in an elderly patient with known malignancy invites anchoring toward metastatic obstruction, and normal tumor markers with normal-caliber ducts should prompt reconsideration. Third, a transient rise in bilirubin after sphincterotomy should not be mistaken for treatment failure.

ABSTRACT V-63

“When the Obvious Diagnosis Is Not the Answer: Fulminant Wilson Disease Masquerading as Acetaminophen Toxicity”

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Program: University of Puerto Rico Program

ABSTRACT

INTRODUCTION: Acute liver failure is a rare, a potentially reversible condition resulting in severe liver impairment and rapid clinical deterioration in patients without pre-existing liver disease. Although acetaminophen toxicity is the most common cause of ALF, diagnostic anchoring on an apparent toxic exposure can delay recognition of alternative etiologies. Wilson disease is an uncommon cause of ALF, accounting for approximately 3% of cases, but its fulminant presentation carries an extremely poor prognosis without timely liver transplantation. CASE PRESENTATION: A 23-year-old woman with a history of drug-induced psychosis, depression, anxiety, and multiple psychiatric hospitalizations presented following a suicide attempt involving the ingestion of approximately 10 acetaminophen tablets. Initial laboratory evaluation revealed elevated aminotransferases without evidence of acute liver failure. Within 24 hours, however, she rapidly deteriorated, developing fulminant acute liver failure manifested by severe coagulopathy (INR 7), altered mental status, recurrent hypoglycemia, hypoalbuminemia, and marked elevations in AST, ALT, and total bilirubin with a disproportionately low alkaline phosphatase. Intravenous N-acetylcysteine was initiated, but hepatic synthetic function continued to deteriorate. Despite an apparent toxicologic etiology, a comprehensive evaluation for alternative causes of acute liver failure was pursued including negative viral hepatitis serologies, antinuclear antibody, anti-smooth muscle antibody, and alpha-1 antitrypsin testing. Abdominal ultrasound demonstrated no biliary obstruction. As the clinical course progressed, several findings became increasingly discordant with isolated acetaminophen toxicity, including persistent disproportionate hyperbilirubinemia relative to alkaline phosphatase, severe thrombocytopenia and low ceruloplasmin. Given her young age and neuropsychiatric history, these findings prompted evaluation for Wilson disease. Ophthalmologic examination revealed bilateral Kayser-Fleischer rings, providing a critical diagnostic clue. Although serum copper was low and 24-hour urinary copper excretion was nondiagnostic, the combination of Kayser-Fleischer rings, low ceruloplasmin, disproportionately low alkaline phosphatase relative to bilirubin, thrombocytopenia, and rapidly progressive acute liver failure remained highly concerning for fulminant Wilson disease. Urgent liver transplant evaluation and disease specific therapy, including chelation, were pursued. However, the patient’s liver synthetic function improved, and she ultimately left the hospital against medical advice before the diagnostic evaluation and definitive treatment could be completed. CONCLUSION: This case highlights the importance of avoiding premature diagnostic closure in acute liver failure. Although an apparent acetaminophen overdose initially provided a plausible etiology, the patient's rapid clinical deterioration and atypical biochemical and hematologic findings prompted further investigation. A complete ALF workup should be pursued even when a suspected cause is identified, particularly when the clinical course is disproportionate to the presumed etiology. In this patient, low alkaline phosphatase relative to bilirubin, thrombocytopenia, low ceruloplasmin, and Kayser-Fleischer rings provided critical clues to underlying fulminant Wilson disease despite nondiagnostic copper studies. Early recognition of these findings is essential because fulminant Wilson disease requires urgent liver transplant evaluation.

ABSTRACT V-64

Dissecting the Real Problem: When NSTEMI Is Not the Answer

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ABSTRACT

BackgroundAcute type A aortic dissection (ATAAD) is a life-threatening cardiovascular emergency that may present with atypical symptoms and mimic acute coronary syndromes. Early recognition is critical because delays in diagnosis and surgical intervention are associated with significant morbidity and mortality.Case PresentationA 59-year-old woman with a history of hypertension, active tobacco use, and migraines initially presented to the emergency department with lower-extremity weakness, malaise, and mild disorientation. She was discharged, but her symptoms progressed, and she subsequently developed chest pain with persistent leg weakness. She therefore returned to the emergency department. On physical examination, she was found to have a wide pulse pressure (140/65 mmHg), a normal heart rate, and a grade IV diastolic murmur. Electrocardiography demonstrated normal sinus rhythm with nonspecific ST-segment changes. Serial troponin I values were 0.320, 0.269, and 0.243 ng/mL (reference range, 0–0.12 ng/mL), raising concern for non-ST-segment elevation myocardial infarction (NSTEMI). Invasive coronary angiography was planned following head CT, which ruled out a cerebrovascular event.However, bedside transthoracic echocardiography demonstrated severe aortic regurgitation and an aortic intimal flap. Computed tomography performed with a pulmonary embolism protocol raised suspicion for acute aortic dissection. She was transferred to our institution, where emergent computed tomography angiography confirmed a Stanford type A aortic dissection extending from the ascending aorta through the aortic arch and descending thoracic aorta into the abdominal aorta and iliac arteries. The dissection also involved the left renal artery, resulting in impaired renal perfusion.Upon arrival at our institution, she remained hypertensive. Intravenous labetalol was initiated to control blood pressure and heart rate. Cardiothoracic surgery was urgently consulted for surgical repair. Transesophageal echocardiography confirmed a Stanford type A aortic dissection originating near the right sinotubular junction and extending into the aortic arch and descending thoracic aorta, with severe aortic regurgitation and preserved left ventricular systolic function (left ventricular ejection fraction 55–60%). The patient underwent a Bentall procedure with replacement of the aortic root, ascending aorta, and aortic valve using a mechanical valved conduit. The procedure included reimplantation of the coronary arteries and was performed with deep hypothermic circulatory arrest.OutcomePostoperatively, the patient remained hemodynamically stable without significant bleeding or coagulopathy. She recovered well following surgery and was started on warfarin anticoagulation for her mechanical valve.ConclusionThis case highlights the diagnostic challenge of ATAAD presenting with elevated troponin and chest pain in the setting of neurologic symptoms, initially raising concern for NSTEMI. Identifying severe aortic regurgitation and an intimal flap on bedside echocardiography was pivotal in establishing the diagnosis and enabling prompt definitive management. Maintaining a high index of suspicion for acute aortic dissection in patients with chest pain accompanied by neurologic or systemic symptoms is essential, particularly when clinical findings are not fully consistent with an isolated acute coronary syndrome.

ABSTRACT V-65

Beyond DKA: Posterior Reversible Encephalopathy Syndrome in Severe Metabolic Crisis

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ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) is an acute neurologic disorder characterized by vasogenic cerebral edema, predominantly affecting the parieto-occipital regions. It is commonly associated with severe hypertension, renal dysfunction, autoimmune disease, eclampsia, and immunosuppressive therapies. PRES associated with diabetic ketoacidosis (DKA) is rare and may be difficult to recognize because neurologic manifestations can overlap with those of severe metabolic derangement. We present a case of PRES occurring during severe DKA in a young adult with poorly controlled type 1 diabetes mellitus.Case Presentation:A 22-year-old man with type 1 diabetes mellitus and multiple previous admissions for DKA presented with influenza-like symptoms, severe respiratory distress, and altered mental status. Laboratory evaluation revealed profound metabolic acidosis with bicarbonate of 4 mEq/L and severe hyperglycemia with glucose of approximately 1,800 mg/dL, consistent with severe DKA. His neurologic and respiratory status deteriorated, requiring endotracheal intubation and mechanical ventilation.His hospital course was complicated by influenza pneumonia, severe metabolic abnormalities, marked blood pressure fluctuations, and persistent encephalopathy despite treatment of DKA. Following initial extubation, persistent neurologic dysfunction prompted further investigation. Brain magnetic resonance imaging demonstrated vasogenic edema involving the bilateral parieto-occipital regions with extension into the frontal lobes, consistent with PRES. Neurologic evaluation supported the diagnosis, with severe metabolic instability and blood pressure variability considered likely precipitating factors. The patient subsequently experienced neurologic deterioration requiring re-intubation.Management included supportive care, careful blood pressure control, correction of metabolic abnormalities, and treatment of associated infectious and respiratory complications. His neurologic status gradually improved, and he was successfully liberated from mechanical ventilation. Further assessment identified significant insulin nonadherence associated with inadequate diabetes education and limited family support, contributing to recurrent DKA.Discussion:PRES is a rare but potentially reversible complication of DKA. Proposed mechanisms include cerebral autoregulatory failure, endothelial dysfunction, blood-brain barrier disruption, and subsequent vasogenic edema. Severe metabolic abnormalities, systemic inflammation, osmotic shifts, and blood pressure variability may contribute to endothelial injury during DKA. Recognition is challenging because encephalopathy may initially be attributed to the metabolic crisis itself. Persistent or worsening neurologic dysfunction despite metabolic correction should prompt evaluation for alternative neurologic complications. MRI is the preferred imaging modality for identifying characteristic vasogenic edema.Conclusion:PRES should be considered in patients with severe DKA who develop persistent encephalopathy or neurologic deterioration despite appropriate metabolic correction. Early recognition, neuroimaging, blood pressure management, correction of precipitating factors, and supportive care are essential to reduce the risk of permanent neurologic injury. Comprehensive diabetes education, adherence support, family engagement, and multidisciplinary follow-up are also critical for preventing recurrent DKA and its potentially life-threatening complications.

ABSTRACT V-66

From Pontine Stroke to Prion Disease: A Diagnostic Evolution

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Program: Universidad Central del Caribe Program

ABSTRACT

Introduction:Rapid neurological decline is a neurological emergency encompassing vascular, infectious, autoimmune, and neurodegenerative etiologies. Creutzfeldt–Jakob disease (CJD) is a rare and fatal prion disorder characterized by rapidly progressive cognitive and neurological deterioration. We present a case in which an established acute pontine infarct complicated recognition of an evolving prion disease.Case Presentation:A 67-year-old man with coronary artery disease, hypertension, hyperlipidemia, and type 2 diabetes presented with left-sided weakness, dizziness, and imbalance. Non-contrast head CT showed a small left pontine hypodensity consistent with lacunar infarction. He was managed for acute ischemic stroke with clinical improvement and discharged home. Within 2–3 weeks, he developed rapidly progressive cognitive and functional decline, hallucinations, severe insomnia, dysphagia, gait instability, recurrent falls, and myoclonus, progressing to mutism and dependence in activities of daily living. Brain MRI subsequently showed bilateral symmetric cortical restricted diffusion involving the frontal, parietal, and temporal lobes with cortical ribboning. CSF studies were unremarkable, and an extensive infectious evaluation was negative. The discordance between the initial focal infarct and subsequent diffuse neurological deterioration prompted reconsideration of the diagnosis. Given the characteristic MRI findings and rapid clinical progression, prion disease became a leading consideration. CSF testing revealed positive RT-QuIC with markedly elevated 14-3-3 and tau proteins, confirming sporadic CJD. Despite supportive and empiric immunomodulatory therapy, he continued to deteriorate. Following multidisciplinary discussions regarding his poor prognosis and goals of care, he was discharged home with hospice services.Discussion:This case demonstrates how a plausible initial diagnosis can complicate recognition of a second, rapidly progressive neurological process. Although the patient initially improved following a documented pontine infarct, his subsequent global neurological decline was inconsistent with the expected trajectory of a small lacunar stroke. The evolution from a focal pontine lesion to bilateral cortical ribboning provided a critical diagnostic clue and shifted the diagnostic framework toward prion disease. The unremarkable CSF and negative infectious evaluation further supported consideration of a non-inflammatory neurodegenerative process. RT-QuIC subsequently established the diagnosis, allowing clinicians to provide prognostic counseling and align care with the patient's goals. Recognition of the diagnosis therefore had implications beyond diagnostic certainty, facilitating timely transition to goal-concordant hospice care at home.Conclusion:CJD should remain in the differential of rapidly progressive neurological decline, even after an established cerebrovascular event. Recognizing when a patient's clinical trajectory no longer fits the initial diagnosis is essential to avoiding premature closure. In this case, serial MRI findings and prion-specific CSF testing ultimately established the diagnosis of CJD after the decline was initially attributed to stroke. Establishing the diagnosis provided prognostic clarity and allowed timely goals-of-care discussions, ultimately facilitating discharge home with hospice services.

ABSTRACT V-67

Red Flags, Not Red Eyes: Carotid-Cavernous Fistula Masquerading as Refractory Orbital Cellulitis

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Program: University of Puerto Rico Program

ABSTRACT

Introduction:Dural carotid-cavernous fistulas (CCFs) are rare arteriovenous communications that frequently present with nonspecific ocular findings, mimicking more common orbital pathologies. Clinical presentation varies depending on the fistula's flow and venous drainage pattern. While high-flow CCFs may present with the classic triad of pulsatile proptosis, ocular bruit, and conjunctival chemosis, low-flow dural fistulas can have a more subtle and progressive course. Because delayed recognition can cause irreversible vision loss, maintaining high clinical suspicion in refractory orbital syndromes is critical.Case Presentation:A 71-year-old man with uncontrolled hypertension, coronary artery disease, stage 4 chronic kidney disease (CKD) secondary to autosomal dominant polycystic kidney disease (ADPKD), and hypothyroidism presented with several months of progressive right eye redness and intermittent diplopia, followed by two weeks of worsening periorbital edema refractory to outpatient oral antibiotics. Ophthalmologic examination revealed right-sided proptosis, hemorrhagic chemosis with corkscrew conjunctival injection, borderline elevated intraocular pressure, and ophthalmoplegia with preserved visual acuity. Serum inflammatory markers and leukocyte counts were normal, and renal function was impaired.Non-contrast computed tomography of the orbits demonstrated right preseptal soft-tissue edema and fat stranding. Presumed to have preseptal cellulitis with potential orbital extension, the patient was admitted for broad-spectrum intravenous vancomycin, ceftriaxone, and metronidazole. Despite therapy, chemosis and extraocular muscle restriction worsened. Non-contrast orbital magnetic resonance imaging demonstrated mild extraocular muscle enlargement and marked dilatation of the right superior ophthalmic vein, raising suspicion for a vascular etiology. Antibiotics were discontinued. Subsequent digital subtraction angiography confirmed a low-flow dural CCF supplied by pterygopalatine branches of the maxillary artery and cavernous branches of the internal carotid artery, with retrograde ophthalmic venous drainage (Barrow type D fistula). The patient underwent two uncomplicated transvenous embolization procedures via the superior ophthalmic vein with significant symptomatic resolution and preserved vision under multidisciplinary follow-up.Discussion:This case illustrates a low-flow dural CCF masquerading as antibiotic-refractory pseudo-cellulitis. The absence of systemic inflammation, progression despite broad-spectrum antimicrobials, and characteristic vascular signs (corkscrew conjunctival vessels and asymmetric proptosis) were pivotal in prompting diagnostic reassessment.Furthermore, this case underscores high-value diagnostic reasoning in renal impairment. Severe CKD precluded routine contrast administration; however, identifying superior ophthalmic vein dilation on non-contrast orbital MRI served as the critical non-invasive clue that justified definitive angiography. Finally, while ADPKD is classically associated with intracranial aneurysms and vascular fragility, its co-occurrence with dural arteriovenous fistulas remains exceedingly rare. Clinicians must consider vascular etiologies in patients with presumed orbital infections who fail standard antimicrobial regimens to prevent permanent visual morbidity.

ABSTRACT V-68

Missed Endovascular Window, Medical Success: Apixaban in Symptomatic May Thurner Syndrome

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ABSTRACT

May-Thurner Syndrome (MTS) is the anatomic compression of the left common iliac vein by the right common iliac artery, predisposing to extensive left-sided iliofemoral DVT. While imaging-detected left iliac vein compression is common in asymptomatic cohorts, the true burden of symptomatic MTS is uncertain and likely underrecognized. Some reviews cite about 2% to 5% of DVTs as MTS-related, though robust population estimates are lacking. We present an 87-year-old man with extensive left iliofemoral DVT in the setting of MTS, in whom correct anticoagulant dosing and coordinated specialty care led to clinical improvement without immediate decompression.An 87-year-old man with type 2 diabetes and hyperlipidemia, ambulatory at baseline, developed progressive left leg swelling and pain over ten days. He was discharged from another ED on apixaban, then presented to the VA with worsening edema. Medication reconciliation revealed incorrect dosing: he had been taking two pills once daily due to access barriers. He was afebrile and hemodynamically stable. Exam showed left leg erythema, warmth, and +3 pitting edema with intact distal pulses. Labs demonstrated stable counts and renal function, without coagulopathy or major electrolyte disturbances. CT abdomen and pelvis with IV contrast demonstrated thrombosis of the deep femoral, common femoral, external, and internal iliac veins, secondary to left common iliac vein compression by the right common iliac artery, measuring about 4 mm at the compressed segment versus 12 mm peripherally. This is consistent with May-Thurner physiology. He was started on therapeutic intravenous unfractionated heparin. Interventional Radiology assessed the case as outside the window for catheter-directed thrombolysis or mechanical thrombectomy, and Vascular Surgery advised no acute inpatient intervention, continuation of therapeutic anticoagulation, graduated compression stockings, and outpatient follow up. Symptoms improved during hospitalization. He was transitioned to the standard apixaban DVT regimen with compression stockings and close follow up. At one-month surgical reevaluation, the team documented reduced pitting edema, intact perfusion, and no ulcers or phlegmasia. The DVT was assessed as chronic, and the plan was to continue apixaban and compression with surveillance. Hematology and Oncology concluded that anatomic left common iliac vein compression was the dominant driver and recommended ongoing direct oral anticoagulant therapy since decompression was not pursued and marked improvement was seen on apixaban.Unilateral, extensive left-sided iliofemoral DVT should prompt early consideration of MTS and rapid engagement of endovascular teams while the intervention window remains open, even though many patients will ultimately be managed medically. Apparent DOAC failure can reflect access and dosing errors rather than pharmacologic resistance. Accurate initiation of the apixaban loading phase and patient education are foundational when invasive options are no longer timely, even in MTS. The paradox of MTS is that radiologic compression is frequent in asymptomatic patients, while symptomatic cases are uncommon and often missed, which underscores the need for clinical context and targeted imaging

ABSTRACT V-69

Isolated Acute Ileal Diverticulitis: An Unexpected Cause of Abdominal Pain

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Program: San Juan VA Medical Center

ABSTRACT

Introduction Small bowel diverticula are rare, more often located in the duodenum, and mainly asymptomatic. When present, it is most often found in elderly males. Although uncommon, small bowel diverticula can rise complications including acute diverticulitis, which can mimic of other diseases. Here we present a case of an elderly male with acute ileal diverticulitis as the cause of abdominal pain. Case Presentation A 90-year-old man with history of chronic constipation and severe colonic universal diverticulosis with previous bouts of acute diverticulitis, presents to our Emergency Department complaining of subjective fever and right lower quadrant abdominal pain since the day before evaluation. Other symptoms were denied. Physical exam revealed normal vital signs and tenderness on deep palpation in the right lower quadrant without signs of peritonitis. Laboratories showed neutrophilic leukocytosis, and normal hemoglobin, platelets, renal function and liver enzymes. Abdominopelvic CT scan with oral contrast was remarkable for diverticulosis of the terminal ileum with moderate wall thickening and surrounding inflammation consistent with acute ileal diverticulitis. The appendix was normal. The patient was discharged home with a 10-day course of oral antibiotics with complete resolution of his symptoms. Discussion Diverticula of the small bowel are relatively uncommon, affecting 0.07 to 2% of the population. Although it can affect any part of the small bowel, the terminal ileum is the least common location4. Acute ileal diverticulitis is a rare cause of right lower quadrant abdominal pain, which can mimic other more prevalent disorders such as acute appendicitis and Crohn’s disease. Physicians should be aware of this uncommon manifestation of small bowel diverticulosis and should consider it in the differential diagnosis of right lower quadrant abdominal pain.

ABSTRACT V-70

A Syrinx of Unfortunate Events: An Elusive Case of Syringohydromyelia and Spinal Cord Compression

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ABSTRACT

Background: Spinal cord compression is a neurologic emergency in which delayed recognition can result in irreversible disability. Diagnosis remains challenging because early symptoms are often nonspecific, pain may precede objective neurologic deficits, and examination alone cannot reliably exclude an evolving myelopathy. Diagnostic recognition becomes particularly difficult when competing infectious, cardiopulmonary, or metabolic illness dominates the clinical presentation. Syringohydromyelia poses a similar challenge, with an insidious course that may progress from subtle constitutional symptoms or focal findings to profound motor, sensory, sphincter, and autonomic dysfunction. When access to definitive spinal imaging and neurosurgical expertise is limited, these overlapping diagnostic challenges may further narrow the opportunity for neurologic recovery. Case Presentation: A 63-year-old woman presented with left-sided neck pain following 2–3 weeks of fatigue, dyspnea, myalgias, difficulty voiding, and imbalance. Initial neurologic examination showed intact sensation and 5/5 strength. Evaluation revealed bilateral pulmonary emboli, multifocal pneumonia, metabolic acidosis, acute kidney injury, hyperglycemia, and rhabdomyolysis, providing competing explanations for her presentation.On the following day, acute asymmetric weakness and sensory deficits raised concern for ischemic stroke, prompting stat head CT scan and transfer to the ICU. CT was unrevealing, with no acute intracranial abnormalities. On the third day, she progressed to quadriparesis, urinary retention, loss of perianal sensation, reduced sphincter tone, and a thoracic sensory level, redirecting localization to the spinal cord. Differential diagnoses included compressive myelopathy, transverse myelitis, and anterior spinal artery ischemia.Cervical and thoracic spine MRI was recommended but delayed until day 10 by hemodynamic and respiratory instability and patient intolerance. MRI ultimately demonstrated multilevel cervical, thoracic, and lumbar disease with cervical cord compression, compressive myelopathy, and extensive syringohydromyelia. Urgent neurosurgical evaluation and transfer were pursued; however, physiologic instability, prolonged neurologic deficits, local surgical limitations, case complexity, and transfer barriers precluded definitive intervention. Her course was further complicated by recurrent infections and sepsis superimposed on dysautonomia, contributing to mixed distributive and neurogenic shock, respiratory failure requiring intubation, and multiorgan dysfunction. Profound neurologic deficits persisted, and she died on hospital day 27 with refractory shock, sepsis with multiorgan failure, and subacute spinal cord compression among the certified causes of death.Discussion: This case highlights how progressive spinal cord disease may be obscured by an initially reassuring examination and competing life-threatening diagnoses. An initially reassuring examination may reflect an early stage of an evolving myelopathy, making serial examination and dynamic localization particularly important. Acute asymmetric weakness reasonably prompted evaluation for cerebral ischemia; subsequent quadriparesis, a thoracic sensory level, urinary retention, sphincter dysfunction, and dysautonomia increasingly supported spinal cord involvement. This evolution highlights the importance of reconsidering localization as the neurologic phenotype changes and recognizing urinary and autonomic dysfunction as potential spinal warning signs. When myelopathy emerges, magnetic resonance imaging provides definitive cord evaluation beyond the limitations of computed tomography. In critically ill patients, however, physiologic instability, imaging feasibility, institutional capabilities, and transfer logistics may complicate both diagnosis and intervention, emphasizing the value of early, parallel consideration of definitive imaging, specialty consultation, and transfer when feasible.

ABSTRACT V-71

When Fat Blocks the Airway: An Obstructing Endobronchial Lipoma

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Program: San Juan City Hospital Program

ABSTRACT

IntroductionEndobronchial lipomas are rare benign tumors, accounting for only a tiny fraction of pulmonary neoplasms, and typically arise within the first three subdivisions of the tracheobronchial tree. Despite benign histology, their endoluminal growth can produce progressive bronchial obstruction, atelectasis, recurrent post-obstructive infection, and irreversible distal lung injury. Because symptoms are non-specific, diagnosis is frequently delayed. This case highlights the value of integrating characteristic CT and bronchoscopic findings to recognize an endobronchial lipoma and relieve obstruction before permanent parenchymal damage occurs.Case DescriptionA 68-year-old woman was evaluated for an obstructing right upper lobe (RUL) endobronchial lesion. Contrast-enhanced chest CT demonstrated a low-attenuation, fat-containing mass within the RUL bronchus with associated RUL atelectasis, and no enlarged mediastinal lymph nodes or pleural effusion. Fat attenuation on CT is a well-recognized, near-diagnostic feature of endobronchial lipoma.Flexible bronchoscopy under general anesthesia revealed a well-circumscribed, smooth, yellow, non-friable mass completely obstructing the RUL bronchial orifice. The bronchoscope could not be advanced beyond the lesion, preventing inspection of the distal RUL bronchial tree and documenting substantial airway compromise. The predominantly endoluminal configuration and low-vascularity appearance supported an endoscopic resection attempt.Endoscopic transection at the lesion's base immediately restored RUL bronchial patency. Mild procedural bleeding was controlled with cold saline, topical epinephrine, and tranexamic acid, and final inspection confirmed complete recanalization without residual obstruction or active bleeding. The retrieved specimen measured 26 x 15 x 3 mm in aggregate. Histopathology demonstrated mature adipose tissue consistent with endobronchial lipoma, without malignancy. The patient tolerated the procedure without major immediate complications and did not require surgical lung resection.DiscussionEndobronchial lipoma should be considered when CT shows a non-enhancing, fat-attenuation endoluminal mass, particularly with post-obstructive atelectasis or recurrent pneumonia. Definitive diagnosis requires histopathology, as imaging and bronchoscopic morphology alone cannot fully exclude malignancy.Management has shifted decisively toward bronchoscopy: in contemporary literature, the majority of endobronchial lipomas are managed bronchoscopically, and recurrence rates are low following both bronchoscopic and surgical resection. Bronchoscopic resection is now considered the first-line treatment for appropriately selected lesions, with endoscopic techniques proving both diagnostic and therapeutic when there is no severe distal bronchiectasis. Surgery remains indicated for extrabronchial extension, irreversible distal lung destruction, persistent concern for malignancy, or technical limitations preventing adequate endoscopic treatment.This case illustrates how concordant imaging and bronchoscopic assessment can establish the diagnosis and relieve airway obstruction in a single session, sparing the patient a lung resection. Follow-up is needed to confirm radiographic resolution of atelectasis and durable airway patency.

ABSTRACT V-72

Beyond the Algorithm: Goals-of-Care Decision-Making in Refractory Sustained Monomorphic Ventricular Tachycardia

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ABSTRACT

Introduction: Sustained monomorphic ventricular tachycardia (SMVT) is VT lasting> 30 s or requiring intervention, arising from scar-related reentry, triggered activity, abnormal automatism, or functional reentry. Acute management includes cardioversion and antiarrhythmic therapy; deep sedation bridges refractory cases, yet airway compromise may require intubation. ICD is Class I for secondary prevention in patients with meaningful life expectancy, yet active electrical storm is a contraindication to implantation.Case: A 69-year-old male with ischemic cardiomyopathy (EF <20%), CAD, prior Afib ablation, aortic valve replacement, pulmonary hypertension, CKD, HIV, and squamous cell carcinoma on chemotherapy with limited life expectancy presented in SMVT with hemodynamic compromise (BP 87/65, HR 160). Initially, VT failed to respond to pharmacological therapy with amiodarone boluses, lidocaine, adenosine, and esmolol. His DNR/DNI directive was reaffirmed after goals-of-care discussion. Deep sedation was precluded not by DNR status, but by refusal of intubation. Antiarrhythmic infusion continued in the CCU, with eventual restoration of sinus rhythm and transition to oral amiodarone, mexiletine, and carvedilol.Discussion: Two palliative interventions defined management. First, deep sedation was limited by refusal of intubation, illustrating that code status does not prohibit treatment, but preferences can restrict interventions. Second, ICD implantation was not pursued: the patient was not a candidate given limited life expectancy, end-of-life preferences, and active electrical storm. Catheter ablation was similarly declined. This case underscores the need for proactive collaboration between cardiology and palliative medicine to ensure shared decision-making guides management at every critical junction.Conclusion: In patients with poor prognosis who are not candidates for, or decline, advanced therapy, VT storm management centers on antiarrhythmic optimization rather than escalation to sedation or device-based interventions. When intubation is refused, sedation-based strategies are limited, making pharmacologic control the primary bridge to stability. ICD implantation should be excluded when it poses a risk of repeated shocks without meaningful benefit, given the patient's directives. This case highlights that VT storm management in this population must be guided by goals of care, integrating palliative principles at every decision point.

ABSTRACT V-73

Hidden in Plain Sight: Recognizing the Aslanger Pattern in Acute Coronary Occlusion

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ABSTRACT

The Aslanger pattern is an electrocardiographic manifestation of acute coronary occlusion that may be overlooked because it does not fulfill conventional ST-elevation myocardial infarction (STEMI) criteria. Despite its subtle appearance, recognition is essential, as it is frequently observed in patients with multivessel coronary artery disease and has been associated with worse clinical outcomes when misdiagnosed.We present a 76-year-old man with diabetes mellitus, hypertension, hyperlipidemia, stage 3 chronic kidney disease, obstructive sleep apnea, and known three-vessel coronary artery disease who presented to the emergency department with persistent chest pain radiating to his back and left arm, accompanied by dyspnea. Symptoms began while preparing coffee in the morning. Sublingual nitroglycerin failed to relieve his chest pain. Upon arrival to the emergency department, he was tachycardic, with persistent chest pain, and with no signs of heart failure.Initial electrocardiography (ECG) demonstrated a 1 mm ST-segment elevation in lead III, with concomitant ST-segment depression in V4–V6 with positive T-waves and ST-segment in V1 greater than V2, consistent with the Aslanger pattern. High-sensitivity troponin was 91 ng/L at 0 hours, rising to 398 ng/L at 1 hour. Although conventional STEMI criteria were not met, the emergency department consulted cardiology approximately 2 hours after the initial electrocardiogram due to elevated troponin levels and persistent ischemic symptoms despite being on intravenous nitroglycerin. Upon cardiology evaluation, the Aslanger pattern was recognized, and due to ongoing chest pain, a STEMI code was activated. Emergent coronary angiography revealed a heavily calcified 95% distal right coronary artery stenosis, in addition to 60% distal left main and 70% proximal left anterior descending artery stenoses. Intravascular ultrasound confirmed the severe calcified distal right coronary artery lesion. The lesion was successfully treated with a drug-eluting stent. His chest pain resolved following revascularization, and he was discharged 48 hours later in stable condition.The Aslanger pattern is a diagnostic challenge because its ST-segment abnormalities may be subtle and fall outside conventional STEMI criteria. The pattern reflects the interaction of an inferior injury current with opposing electrical forces from diffuse subendocardial ischemia, often in the setting of multivessel coronary artery disease. Partial vector cancellation can attenuate ST-segment elevation in leads II and aVF while preserving elevation in lead III, an appearance that can mask acute coronary occlusion and may contribute to misclassification as a non-ST-elevation myocardial infarction.In this patient, persistent ischemic symptoms and elevated troponin levels prompted cardiology consultation despite the absence of conventional STEMI criteria on ECG. Recognition of the characteristic pattern led to immediate STEMI activation, with emergent angiography demonstrating a critical, heavily calcified distal right coronary artery lesion. This case illustrates the importance of integrating the clinical presentation with subtle electrocardiographic findings when evaluating patients with suspected acute coronary syndrome, as recognition of the Aslanger pattern may help prevent diagnostic delay and facilitate timely reperfusion.

ABSTRACT V-74

More Than Deconditioning: Unmasking Seronegative Immune-Mediated Necrotizing Myopathy in an Older Adult

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ABSTRACT

Immune-mediated necrotizing myopathy (IMNM) is an idiopathic inflammatory myopathy characterized by severe proximal muscle weakness, markedly elevated creatine kinase (CK), and myofiber necrosis with minimal inflammatory infiltrates on muscle biopsy. IMNM is classified as anti–3-hydroxy-3-methylglutaryl-coenzyme A reductase (HMGCR) positive, anti–signal recognition particle (SRP) positive, or seronegative. Seronegative IMNM remains poorly characterized and presents a particular diagnostic challenge in older adults whose comorbidities and baseline functional limitations may obscure progressive weakness.A 76-year-old Latino man presented with fever and bilateral lower-extremity edema following exchange of a chronic indwelling urinary catheter. His medical history included hypertension, type 2 diabetes mellitus, prostate cancer treated with radiation and prostatectomy, chronic statin exposure, and longstanding functional impairment requiring assistance with activities of daily living. He was admitted for a catheter-associated urinary tract infection. Initial evaluation was additionally notable for newly elevated aminotransferases without other evidence of hepatic dysfunction.Further history revealed progressive difficulty entering vehicles, washing his hair, and brushing his teeth, as well as chronic stable dysphagia. Examination demonstrated proximal lower-extremity weakness and myalgias. Serum CK exceeded 22,000 U/L, suggesting that the aminotransferase elevations originated from skeletal muscle injury. The differential diagnosis included toxic statin myopathy, infection-associated rhabdomyolysis, and an idiopathic inflammatory myopathy. Magnetic resonance imaging of the thighs with short tau inversion recovery sequences demonstrated diffuse bilateral muscle edema and enhancement. Muscle biopsy revealed widespread necrotic fibers, myophagocytosis, and regenerating fibers with minimal inflammatory infiltrates. Testing for anti-HMGCR and anti-SRP antibodies was negative, supporting a diagnosis of seronegative IMNM. Persistent CK elevation despite statin discontinuation further favored an immune-mediated process over self-limited toxic statin myopathy. High-dose corticosteroid therapy produced a rapid decline in CK and aminotransferase levels, accompanied by improvement in myalgias and lower-extremity edema. Proximal weakness persisted, however, complicating assessment of his functional recovery against a background of chronic disability.Seronegative IMNM should be considered in patients with marked CK elevation, progressive proximal weakness, and characteristic biopsy findings despite negative myositis-specific antibodies. In older adults with limited baseline mobility, new weakness may be misattributed to infection, aging, or deconditioning. Chronic statin exposure can further obscure the diagnosis because toxic statin myopathy and IMNM share clinical and biochemical features. Disproportionate difficulty performing routine activities and otherwise unexplained aminotransferase elevation should prompt evaluation for skeletal muscle injury. Recognizing these subtle changes and distinguishing toxic, infectious, and immune-mediated etiologies are essential because delayed immunosuppressive therapy may contribute to irreversible muscle damage and persistent disability.

ABSTRACT V-75

When Metastatic Breast Cancer Presents as Acute Pancreatitis

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ABSTRACT

Pancreatic metastases are uncommon, comprising under 3-5% of pancreatic malignancies. Renal cell carcinoma predominates. Breast carcinoma is an infrequent source; difficult to distinguish radiographically from primary pancreatic and presentation as acute pancreatitis is particularly unusual unless obstruction takes place. This distinction is clinically consequential because treatment differs substantially between metastatic breast carcinoma and a new pancreatic primary, making tissue sampling crucial. We describe a young woman with metastatic HER2-positive breast carcinoma presenting with acute pancreatitis and a pancreatic head lesion initially suspected to represent a second primary malignancy. A 35-year-old woman with hormone receptor-negative, HER2-positive invasive ductal carcinoma of the left breast (pT2 pN3a, 11 of 17 nodes, extranodal extension) diagnosed two years earlier underwent bilateral mastectomy and adjuvant trastuzumab and pertuzumab after declining chemotherapy and radiotherapy. She subsequently developed hepatic and nodal metastatic disease and resumed HER2-directed therapy seven months before admission. She presented with five days of epigastric pain radiating to the back with nausea. Examination revealed epigastric tenderness without jaundice. Lipase was 569 U/L and alkaline phosphatase 298 U/L, meeting the Revised Atlanta criteria for acute pancreatitis. Ultrasonography (US) showed no cholelithiasis, and she denied alcohol use. A prior similar episode had been imaged at an outside emergency department, showing pancreatic enlargement read as pancreatitis versus neoplasm. Cross-sectional imaging six months earlier demonstrated a normal pancreas. Magnetic resonance cholangiopancreatography (MRCP) revealed a double-duct sign with tapering at the pancreatic head without a discrete mass. Subsequent contrast-enhanced magnetic resonance imaging (MRI) demonstrated a 3.9-cm hypoenhancing pancreatic head mass concerning for primary pancreatic adenocarcinoma or cholangiocarcinoma. As bilirubin progressively increased, lipase down trended and endoscopic retrograde cholangiopancreatography (ERCP) demonstrated a distal biliary stricture treated with sphincterotomy and stenting. Biliary brushings were negative for malignancy. Given persistent concern for malignant obstruction, endoscopic ultrasound-guided core biopsy was obtained. Histopathology demonstrated carcinoma positive for CK7 and GATA3 on two independent blocks, matching the immunophenotype of her previous breast carcinoma and establishing pancreatic metastasis. Staging demonstrated additional hepatic, pulmonary, osseous, and peritoneal metastatic disease, prompting redirection of systemic therapy while currently hospitalized. This case illustrates the diagnostic challenge of a new pancreatic lesion in a patient with known malignancy. Acute pancreatitis and subsequent biliary obstruction initially suggested a new pancreatic primary, while imaging could not reliably distinguish primary pancreatic malignancy from metastatic disease. Negative biliary brushings did not exclude malignancy, and definitive diagnosis required core biopsy with lineage-specific immunohistochemistry, which ultimately answered the question. Establishing the lesion as metastatic breast carcinoma fundamentally altered staging and systemic treatment considerations leading to adequate ERBB2-amplified based chemotherapy with trastuzumab emantasine, deruxtecan or tucatinib. Had the presumed pancreatic primary kept in the working diagnosis, the patient would have been treated with gemticitabine or Folfirinox based chemotherapy. For internists, diagnostic anchoring on a second primary or conversely assuming every new lesion represents known metastatic disease can lead to inappropriate treatment. A new pancreatic mass in a patient with established malignancy requires integration of oncologic history, imaging, clinical progression, and, when management would differ, tissue diagnosis before redirecting therapy.

ABSTRACT V-76

Early-Onset Intra-Abdominal Malignant Peripheral Nerve Sheath Tumor in a Young Adult with Neurofibromatosis Type 1 and a Novel Germline Variant

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ABSTRACT

Introduction: Patients with neurofibromatosis type 1 (NF1) face a lifetime malignant peripheral nerve sheath tumor (MPNST) risk of 8–13%, with most tumors arising from pre-existing plexiform neurofibromas. What's less common is seeing this transformation in a young adult, particularly one carrying a germline NF1 variant that hasn't been reported before — a scenario that raises real questions about how molecular findings should shape both diagnosis and management.Case Description:A 22-year-old male with a known history of NF1 came in with severe abdominal pain in early April 2025. Imaging — a CT of the abdomen and pelvis obtained April 23 2025— showed a large intra-abdominopelvic mass, and he went on to exploratory laparotomy with resection on May 1.st, 2025 Unfortunately, follow-up imaging on May 27th, 2025 showed substantial residual disease. On pathology, the tumor was a high-grade MPNST: negative for S100, SOX10, cytokeratin AE1/AE3, and smooth muscle actin, with a notably elevated Ki-67 proliferation index. Molecular testing turned up two findings of note — a novel germline NF1 nonsense variant, c.2875C>T (p.Gln959*), and a somatic CDKN2A copy number loss. Given the unusual pathology and molecular results, the case was presented at our institution's multidisciplinary tumor board to guide further management.Discussion: Two things make this case worth reporting. First, intra-abdominal MPNST at this age, in the setting of an NF1 variant not previously described, is genuinely rare — most malignant transformations in NF1 patients occur later and in more typical anatomic locations. Second, the combination of findings here reinforces a practical point: new or escalating abdominal pain in someone with NF1 shouldn't be dismissed as routine, and when malignancy is confirmed, molecular profiling (not just histology) is what ultimately guides prognosis and opens the door to targeted therapy. Tumor board discussion of this case underscored the value of a multidisciplinary approach — particularly given the residual disease and the novel variant — in weighing options such as adjuvant therapy, surveillance intensity, and eligibility for targeted or investigational approaches.

ABSTRACT V-77

When Chronic Pain Has Another Face: Overlooked Crowned Dens Syndrome in a Patient with Fibromyalgia

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ABSTRACT

Crowned dens syndrome (CDS) is an uncommon but clinically significant manifestation of calcium pyrophosphate dihydrate (CPPD) deposition disease, characterized by acute or subacute onset of severe upper neck pain, restricted cervical motion, and elevated inflammatory markers. CDS results from the deposition of CPPD in the ligaments surrounding the odontoid process producing a characteristic "crown-like" calcification on cervical CT scan. Although chondrocalcinosis is common in CPPD, axial involvement as CDS is rare, representing approximately 5% of acute CPPD presentations. The clinical presentation of CDS often mimics other conditions such as meningitis, polymyalgia rheumatica, or giant cell arteritis, leading to frequent misdiagnosis and unnecessary invasive procedures. Awareness of CDS is particularly important in patients with diffuse chondrocalcinosis and chronic pain syndromes where overlapping symptoms may obscure the diagnosis.A 63-year-old male with a medical history of fibromyalgia presented to the Emergency Department complaining of worsening myalgia and sudden onset neck pain, accompanied by fever and chills. He reported that his diffuse pain had progressively worsened over the past month despite adherence to his prescribed pain medical regimen. During this period, he experienced a fibromyalgia flare, which was further managed with intramuscular steroid and anti-inflammatory injections. These interventions provided temporary symptomatic relief; however, the symptoms recurred the following day. Physical examination revealed limited range of motion in cervical spine due to pain and bilateral paraspinal tenderness upon palpation. Laboratory tests showed mild leukocytosis, normocytic-normochromic anemia, and elevated inflammatory markers. Rheumatologic work up was negative. Multiple x-rays demonstrated significant chondrocalcinosis in the left shoulder, bilateral knee and wrist. Neck CT scan without contrast revealed moderate disc space narrowing, mild multilevel facet joint arthropathy, and mineralization within the soft tissues surrounding the odontoid process, findings consistent with Crowned dens syndrome. The patient was started on oral steroid therapy, after which he reported rapid significant clinical improvement within the next days. Crowned dens syndrome can become a diagnostic challenge particularly in patients with chronic pain syndromes. CDS remains under-recognized, and only a minority of patients with CPPD and neck pain are formally diagnosed. The rarity of CDS, combined with its nonspecific symptoms and overlap with other rheumatologic and neurologic disorders, contribute to frequent misdiagnosis. In patients with chronic pain syndromes, the acute inflammatory features of CDS may be attributed to their underlying conditions further delaying recognition. CT imaging is the gold standard for diagnosis. Prompt recognition is crucial, as CDS responds rapidly to nonsteroidal anti-inflammatory drugs or corticosteroids, with most patients experiencing dramatic improvement. Increased awareness among clinicians is essential to avoid unnecessary procedures and to ensure timely effective management. This case underscores the importance of heightened clinical suspicion in patients presenting with acute sudden neck pain, especially with predisposing factors such as diffuse chondrocalcinosis in the presence of other chronic pain disorders, in order to provide the most appropriate medical management.

ABSTRACT V-78

When Granulomas Mislead: Pulmonary Tuberculosis After a Diagnosis of Sarcoidosis

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Program: Universidad Central del Caribe Progrm

ABSTRACT

Introduction: Sarcoidosis and tuberculosis (TB) are both granulomatous diseases that overlap heavily in their clinical picture, imaging findings, and biopsy appearance. Both can cause fevers, night sweats, enlarged lymph nodes, and lung abnormalities. The key diagnostic trap is that noncaseating granulomas, the biopsy finding classically taught for sarcoidosis, also occur in TB and no histologic feature is unique to sarcoidosis. Biopsy alone therefore cannot prove sarcoidosis, it remains a diagnosis of exclusion. This matters because sarcoidosis is treated with corticosteroids, which suppress the immune system and can activate an unrecognized TB infection. For this reason, TB must be actively ruled out with sputum smear, culture, and molecular testing before steroids are started.Case Presentation: A 23-year-old woman with Scimitar syndrome and pulmonary hypertension presented with two months of productive cough, night sweats, and subjective fevers, without weight loss, travel, or sick contacts. She was febrile with normal oxygen levels and decreased breath sounds over the left upper lung. Her workup began with an enlarged neck lymph node biopsied as reactive lymphadenitis. CT and PET imaging then showed highly active lesions in the mediastinum and left lung apex. A lung biopsy showed chronic inflammation with noncaseating granulomas and giant cells without malignancy; stains for acid-fast bacteria, fungi, and other bacteria were negative. This nonspecific pattern was interpreted as favoring sarcoidosis, though the same pattern is seen in tuberculosis.Before any steroids were given, repeat imaging showed a new cavity and consolidation at the left lung apex, a finding that argues against sarcoidosis and is typical of tuberculosis yet mycobacterial testing was not repeated. Sarcoidosis was diagnosed and prednisone started, later switched to methylprednisolone for intolerance. Follow-up CT then showed a large thick-walled cavity, a second smaller cavity, and numerous small nodules, highly concerning for tuberculosis. Tuberculin skin testing was strongly positive, three sputum samples were smear-positive, and molecular testing confirmed rifampin-susceptible M. tuberculosis. HIV testing was negative. Tuberculosis treatment was started, cultures turned negative by week five. Steroids were tapered, and she was discharged about one month after admission on a shortened rifamycin-based regimen supported by updated guidelines.Discussion: This case highlights the diagnostic challenge created by the substantial overlap between sarcoidosis and TB and, more importantly, the risk of anchoring on sarcoidosis before mycobacterial infection has been adequately excluded. The initial finding of noncaseating granulomas, together with negative acid-fast staining, favored sarcoidosis but did not definitively exclude TB. The development of cavitary disease before corticosteroid initiation was an important discordant finding that warranted renewed mycobacterial evaluation. The subsequent confirmation of active tuberculosis after corticosteroid exposure raises the possibility of initially undetected infection, reactivation of latent disease, or coexistence of both. This case emphasizes that a diagnosis of sarcoidosis should be continually reassessed when the clinical or radiographic course deviates from the expected pattern. Negative tissue staining and noncaseating granulomas should not provide false reassurance when suspicion for tuberculosis persists.

ABSTRACT V-79

Two Faces of Creutzfeldt-Jakob Disease: A Comparative Analysis of Atypical Presentations

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Program: Universidad Guadalajara

ABSTRACT

Creutzfeldt-Jakob disease (CJD) is a rare, fatal prion disorder with a mean age of onset of 60 years, typically characterized by myoclonus and rapidly progressive cognitive decline. We report two cases in which atypical initial presentations delayed recognition of CJD. Clinical presentations, diagnostic considerations, and management approaches are compared, highlighting the challenges of diagnosing CJD when its initial features mimic other neurological conditions.A 75-year-old woman with hypothyroidism, hypertension, and dyslipidemia presented with recurrent right-sided focal seizures, initially attributed to hydroxyzine started shortly before symptom onset. Brain MRI demonstrated left frontoparietal diffusion restriction, and levetiracetam was initiated, resulting in temporary seizure control. She was readmitted two weeks later with worsening cognition, and autoimmune encephalitis was suspected. Antiseizure medications, high-dose methylprednisolone, and IVIG failed to control her seizures, which progressed to status epilepticus with facial twitching, gaze deviation, and hemibody tonic-clonic movements; anti-LGI1 and anti-NMDA antibodies were negative. Repeat MRI showed diffusion restriction extending across the cortex, and over two months she declined from full independence to a bedridden, nonverbal, PEG-dependent state. CSF studies confirmed CJD: RT-QuIC was positive, total tau exceeded 1,149 pg/mL, and 14-3-3 gamma protein exceeded 1,999 AU/mL. She died four months after initial presentation. An 81-year-old woman with hypothyroidism and Alzheimer's disease had remained functional with supervision, dancing with family at a holiday gathering about a month before presentation. She then abruptly refused to get out of bed, with no preceding new medication, trauma, vaccination, infection, or travel. She was admitted for encephalopathy, with a differential of metabolic derangement, encephalitis, and atypical infection. Initial MRI demonstrated clear cortical diffusion abnormalities involving the left frontal, parietal, occipital and temporal cortices. Over the following weeks she developed profound deterioration in cognition, language, coordination, mobility, vision, and swallowing, with cortical thumb posturing, choreiform movements, and myoclonus on exam. Repeat MRI revealed cortical ribboning, and EEG presented periodic sharp-wave complexes. Infectious, autoimmune, and metabolic evaluation was negative, and empiric ceftriaxone and acyclovir did not help. She progressed to a bedridden, nonverbal state requiring enteral nutrition. Antemortem CSF RT-QuIC returned positive two months into her illness, with total tau exceeding 13,000 pg/mL, and hospice care was initiated. She died five months after initial presentation. Postmortem Western blot and immunohistochemical analysis confirmed CJD; genetic testing could not be performed because of tissue limitations. The two patients reached the same diagnosis by different routes. The first underwent evaluation for a seizure disorder, initially attributed to medication and later to autoimmune encephalitis; the second was evaluated for metabolic encephalopathy and infection following an abrupt clinical decline. In each case the initial diagnosis failed to explain the continued decline, and it was the evolving cortical MRI findings, confirmed by CSF RT-QuIC, that pointed toward CJD. RT-QuIC has markedly improved antemortem detection of prion disease and was central to diagnosis in both patients. These cases suggest CJD deserves earlier consideration when a patient's course does not track with the presumed diagnosis, since timely recognition allows prognostic counseling and goals of care planning.

ABSTRACT V-80

When infection precipitates paralysis: Thyrotoxic Periodic Paralysis as the first presentation of hyperthyroidism

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ABSTRACT

IntroductionThyrotoxic periodic paralysis (TPP) is an uncommon but potentially life-threatening manifestation of thyrotoxicosis characterized by acute muscle weakness and hypokalemia resulting from intracellular potassium redistribution. TPP predominantly affects young men, particularly those of Asian descent, but can occur in other ethnic groups. Because clinical features of hyperthyroidism may be subtle, TPP can initially mimic primary neurologic or infectious disorders. Early recognition is essential, as severe hypokalemia may result in cardiac complications while the paralysis itself is rapidly reversible.Case DescriptionA 24-year-old Hispanic man with no significant medical history presented with rapidly progressive generalized weakness, bilateral lower-extremity numbness, and inability to ambulate. Two days earlier, he had developed fever and chills and reported cleaning his swimming pool barefoot, raising concern for leptospirosis. An outside-hospital Leptospira IgM was positive, and he was transferred for persistent severe hypokalemia and neurologic symptoms. On presentation, potassium was 1.8 mmol/L with preserved renal function. Electrocardiography demonstrated flattened T waves and prominent U waves. Neurologic examination revealed diffuse, symmetric weakness, greater in the lower extremities, with decreased reflexes, prompting evaluation for Guillain-Barré syndrome.Upon further questioning, the patient reported approximately 18–20 pounds of unintentional weight loss over the preceding two months and a family history of thyroid disease. These findings, together with the profound hypokalemia, prompted evaluation for an underlying thyroid disorder. Thyroid studies demonstrated a markedly suppressed TSH of 0.005 mIU/L and elevated free T4 of 3.36 ng/dL, consistent with overt thyrotoxicosis. He denied previous episodes of similar weakness or associations with exertion, high-carbohydrate meals, or alcohol. Following potassium replacement, serum potassium normalized and his weakness and numbness completely resolved, favoring TPP over a primary neurologic process.A 24-hour radioactive iodine uptake study subsequently demonstrated markedly increased uptake of 60.4% with diffuse, relatively homogeneous distribution and no hyper-functioning nodules, findings consistent with Graves disease.DiscussionTPP results from a rapid intracellular shift of potassium driven by increased Na^+/K^+-ATPase activity and enhanced β-adrenergic sensitivity rather than true potassium depletion. Acute physiologic stress, including infection and fever, may precipitate an attack or unmask previously unrecognized thyrotoxicosis. In this case, the infectious exposure and positive Leptospira IgM, together with neurologic findings suggestive of Guillain-Barré syndrome, initially complicated the diagnostic evaluation. However, the profound hypokalemia and subtle historical clues to thyrotoxicosis redirected the workup. This case illustrates that acute hypokalemic paralysis should prompt consideration of TPP even in patients without a known thyroid disorder or prominent classic symptoms of hyperthyroidism. Thyroid function testing should therefore be considered in unexplained hypokalemic paralysis, even when an infectious or neurologic process initially appears more plausible. Prompt recognition allows rapid reversal of paralysis and identification and treatment of the underlying thyrotoxicosis to prevent recurrence.

ABSTRACT V-81

Behind a Mask of Madness: A Post-Thyroidectomy Myxedema Madness Misdiagnosed as Schizophrenia

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ABSTRACT

Introduction: Thyroid hormone deficiency exerts profound neuroendocrine effects that can present as severe psychiatric disturbances, historically described as "Myxedema Madness". Hypothyroidism-induced neuropsychiatric symptoms range from treatment-resistant depression and psychomotor slowing to frank psychosis, visual and auditory hallucinations, and active suicidal ideation. Myxedema madness stands as a prototypical late clinical presentation of unmonitored hypothyroidism, routinely escaping early detection for years or decades following thyroid resection or untreated hypothyroidism due to the insidious onset of neuroendocrine failure and diagnostic misattribution.Case Summary: A 33-year-old male with a reported history of schizophrenia, chronic heavy smoking, and alcohol use disorder presented to the emergency department after a ground level fall that occurred while under the influence of Fentanyl. X rays studies were negative for fractures and Internal Medicine was consulted for medical clearance before admission to the Psychiatric ward in the setting of active suicidal ideation, as well as, visual and auditory hallucinations, presumed initially to represent an exacerbation of his underlying schizophrenia. At that point, one to one observation had been initiated at the level of the ED. Upon further evaluation, the patient exhibited depressive symptoms, including anhedonia, self-guilt, fatigue, low energy, and marked psychomotor retardation. He also reported a longstanding history of constipation and cold intolerance. On examination, he appeared disheveled and lethargic, with slowed speech, bilateral periorbital edema, diffuse muscle tenderness, and a flushed appearance. Vital signs were notable for bradycardia and bradypnea. These findings prompted further evaluation for an underlying medical etiology which eventually yielded a thyroid-stimulating hormone level of 134 mIU/L and an undetectable level of free T4, consistent with profound untreated primary hypothyroidism. Clinical signs suggested an impending myxedema coma for which treatment was initiated with intravenous levothyroxine and glucocorticoids. In response, the patient demonstrated a progressive clinical improvement in the next 24 hours, with resolution of bradycardia and bradypnea, improvement in lethargy and psychomotor retardation, and cessation of his active suicidal ideation. Further investigation revealed a history of a complete thyroidectomy more than 10 years ago for an unspecified thyroid disease, after which he was lost to medical follow up. He retrospectively reported a perceived temporal association between that surgery and the subsequent deterioration of his psychosocial and psychiatric functioning.Discussion: This case highlights how in patients with established psychiatric conditions or substance abuse disorder clinicians frequently suffer from diagnostic overshadowing, prematurely attributing chronic insidious neuroendocrine failure and acute organic neuro-metabolic collapse to primary psychiatric diagnosis, progression or relapse. This prolonged diagnostic delay converts a readily reversible hormone deficiency into a fatal trajectory, as unmonitored long-term hypothyroidism carries up to a 60% mortality rate once end-organ decompensation ensues. We emphasize the importance of a more attentive and rigorous stance in the process of excluding organic causes regarding patients whose main diagnostic concern remains primary neuropsychiatric conditions, in the inpatient, as well as, in the outpatient setting.

ABSTRACT V-82

A Paradoxical Reaction: Dermatomyositis after Adalimumab

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ABSTRACT

Introduction:Tumor necrosis factor-alpha (TNF-α) inhibitors are commonly used to treat rheumatoid arthritis and other inflammatory diseases. While mostly effective, drug-induced autoimmune manifestations, including inflammatory myopathies, have been reported. Dermatomyositis (DM) is characterized by distinctive cutaneous manifestations and proximal muscle weakness. We present a case of clinically significant dermatomyositis developing shortly after discontinuation of adalimumab in a patient with previously diagnosed amyopathic dermatomyositis and rheumatoid arthritis. Case Presentation:A 33-year-old woman with a history of seropositive rheumatoid arthritis and amyopathic dermatomyositis presented with progressively worsening symmetric proximal muscle weakness and dysphagia, which began approximately one month prior to admission. She reported persistent dysphagia, fatigue, and the development of new cutaneous lesions over the preceding 2 to 3 weeks. The patient had been maintained on a low-dose prednisone regimen that was recently tapered and reported approximately two weeks of nonadherence to methotrexate and hydroxychloroquine. Notably, she had received adalimumab for eight months, with the last dose administered 2 to 3 weeks before symptom onset. Physical examination revealed a hoarse voice, hair thinning, violaceous erythema involving the posterior neck and scalp, photosensitive malar erythema sparing the nasolabial folds, and violaceous papules over the metacarpophalangeal joints consistent with Gottron papules. Nail pitting and onycholysis were also observed. Initial laboratory studies demonstrated normocytic anemia (hemoglobin 9.3 g/dL), markedly elevated erythrocyte sedimentation rate (ESR, 140 mm/hr) and C-reactive protein (CRP, 36.4 mg/L), and elevated lactate dehydrogenase (LDH, 300 U/L). Creatine phosphokinase (CPK) and thyroid-stimulating hormone (TSH) levels were within normal limits. Myositis serology was notable for a positive anti-MDA5 antibody. Antinuclear antibody (ANA) was positive at 1:1280 with a homogeneous pattern, increased from a previous 1:640 speckled pattern, while anti-double-stranded DNA (anti-dsDNA) remained negative and complement levels were normal. High-resolution chest computed tomography (CT) showed no evidence of interstitial lung disease. Based on the clinical presentation, there was an initial concern for a myopathic dermatomyositis flare. The patient was treated with methylprednisolone 500 mg intravenously daily for three days, followed by prednisone 1 mg/kg daily, in addition to methotrexate and folic acid. Adalimumab was permanently discontinued. The temporal association between symptom onset and adalimumab therapy supported a diagnosis of TNF-α inhibitor–induced dermatomyositis rather than idiopathic inflammatory myopathy. Following corticosteroid therapy, the patient demonstrated rapid improvement in muscle strength, cutaneous findings, and dysphagia, with full strength and oral intake restored at discharge. The rapid clinical recovery following corticosteroid initiation was consistent with patterns described in drug-induced dermatomyositis and contrasted with the slower treatment response typical of primary disease. Conclusion:This case demonstrates the complexity of distinguishing primary dermatomyositis from TNF-α inhibitor-associated autoimmune disease in patients with preexisting rheumatologic conditions. The temporal relationship to adalimumab exposure, evolving autoantibody profile, and rapid clinical improvement following corticosteroid therapy support a possible drug-induced mechanism. Clinicians ought to remain vigilant for new autoimmune manifestations in patients receiving TNF-α inhibitors and consider discontinuing the medication when appropriate.

ABSTRACT V-83

It's Not the B12: Hypocellular Acute Myeloid Leukemia Masquerading as Nutritional Pancytopenia in a 34-Year-Old

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ABSTRACT

Low serum vitamin B12 offers a treatable explanation for macrocytic anemia and pancytopenia. Yet serum total-B12 can be falsely low without true deficiency, making methylmalonic acid (MMA) and homocysteine necessary for confirmation. When cytopenias are severe or progressive and the metabolites are normal, the reassuring nutritional label becomes a diagnostic trap. This case follows a young man whose low B12 nearly anchored the workup away from leukemia.A 34-year-old man with hypothyroidism was referred for progressive leukopenia and thrombocytopenia. He reported self-limited gingival bleeding but denied fever, weight loss, or infection. Empiric corticosteroids for presumed immune cytopenias produced no improvement. Examination disclosed a malar eruption sparing the nasolabial folds and intermittent arthralgia, prompting a parallel autoimmune evaluation, as lupus can cause both a malar rash and multilineage cytopenias. The complete blood count (CBC) showed pancytopenia with only mild macrocytosis: white cells 2.27 × 10^3/µL, hemoglobin 11.5 g/dL, platelets 51 × 10^3/µL, mean corpuscular volume 95-98 fL. Serum B12 was persistently low (197 and 214 pg/mL), and cyanocobalamin was begun. The workup then unraveled each benign hypothesis: MMA and homocysteine were normal, excluding functional B12 deficiency; transferrin saturation (68%) and ferritin (542 ng/mL) excluded iron deficiency, the high ferritin instead hinting at ineffective hematopoiesis; anti-intrinsic-factor and anti-parietal-cell antibodies were negative, arguing against pernicious anemia; antinuclear, anti-double-stranded DNA, and anti-Smith antibodies were negative with normal complement. Critically, the reticulocyte production index was only 1.9 despite worsening anemia, and counts continued to fall on replacement, culminating in profound neutropenia (absolute neutrophil count ≈70/µL). With every peripheral explanation exhausted and the marrow failing to respond, bone marrow biopsy was performed. The marrow was the diagnostic pivot: paradoxically hypocellular (20-30%) yet infiltrated by approximately 75% blasts, with megaloblastoid erythroid changes mimicking the very B12 deficiency invoked. Flow cytometry confirmed acute myeloid leukemia (AML) with 72.4% CD13/CD33/CD117+ blasts. Molecular profiling identified IDH1 (R132C), STAG2, and IKZF1 mutations with a KMT2A (11q23.3) abnormality; FLT3, NPM1, and CEBPA were wild-type: a European LeukemiaNet 2022 adverse-risk genotype. The patient was transferred for definitive therapy.Hypocellular AML accounts for only 5-7% of de novo AML and typically affects older adults, making this presentation unusual. Its defining paradox is partly molecular: hypocellular AML harbors fewer proliferation-associated and NPM1/FLT3-ITD mutations, and leukemic pancytopenia arises less from crowding-out than from a differentiation block at the stem-to-progenitor transition and niche remodeling, so residual normal stem cells persist but cannot mature. The genotype also carries therapeutic weight: IDH1 R132C generates the oncometabolite 2-hydroxyglutarate, enforcing an epigenetic differentiation block that is targeted with ivosidenib. STAG2 and non-MLLT3 KMT2A lesions define secondary-ontogeny, adverse-risk disease.We present this case to enforce that low serum B12 must never be accepted as the sole cause of severe or progressive pancytopenia without biochemical confirmation. Three discordances should force marrow examination: cytopenias out of proportion to macrocytosis, counts that worsen despite replacement, and an inadequate reticulocyte response. Normal MMA and homocysteine do not merely refine a nutritional diagnosis: they redirect the entire investigation toward early recognition of an aggressive but treatable malignancy.

ABSTRACT V-11

When Aggressive CLL Masquerades as Richter Transformation

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Program: San Juan City Hospital Program

ABSTRACT

IntroductionRichter transformation (RT) of chronic lymphocytic leukemia/small lymphocytic lymphoma (CLL/SLL) most often manifests as diffuse large B-cell lymphoma. Rapid deterioration, B symptoms, adenopathy, cytopenias, elevated lactate dehydrogenase (LDH), and high fluorodeoxyglucose (FDG) avidity raise suspicion but are nonspecific. Histologically aggressive ("accelerated") CLL/SLL (A-CLL/SLL) and secondary hemophagocytic lymphohistiocytosis (HLH) can mimic RT. We describe their simultaneous convergence producing an RT-like phenotype.Case DescriptionA 70-year-old man with multiply treated CLL/SLL (prior ibrutinib, venetoclax, rituximab, and acalabrutinib) presented with four weeks of fatigue, weakness, anorexia, 10-lb weight loss, drenching night sweats, and diffuse adenopathy. Laboratory evaluation revealed leukocytosis, anemia, severe thrombocytopenia, hyperuricemia, elevated LDH, and acute kidney injury consistent with spontaneous tumor lysis syndrome. Flow cytometry demonstrated prolymphocytic progression (≥30% prolymphocytes); molecular/cytogenetic studies revealed unmutated IGHV, ATM deletion, biallelic del(13q14.3), CDKN2A loss, and a complex karyotype.Whole body FDG-PET/CT demonstrated bulky hypermetabolic lymphadenopathy above and below the diaphragm with extranodal involvement of the salivary glands, Waldeyer ring, liver, spleen, and bone marrow (Deauville 5). An index left axillary node had an SUVmax of 17.13, raising significant concern for RT. Concurrently, he developed fever with unrevealing infectious workup, hepatosplenomegaly, marked hyperferritinemia, hypertriglyceridemia, elevated sIL-2R, and hypofibrinogenemia, yielding an HScore of 253 (>99% probability of HLH) and fulfilling the optimized HLH inflammatory index.Clinical instability and severe thrombocytopenia delayed tissue diagnosis, therefore, malignancy directed therapy was initiated with split dose rituximab and dexamethasone, followed by pirtobrutinib and obinutuzumab with venetoclax (PVO). Ferritin decreased substantially and LDH to normal parameters with significant clinical improvement. Subsequent excisional biopsy of the left axillary node demonstrated accelerated CLL/SLL with Ki-67 of 60–70%, meeting Giné criteria but falling short of RT, without confluent sheets of large cells. Bone marrow biopsy showed approximately 80% involvement by histologically aggressive CLL/SLL. He subsequently completed cycle 1 of PVO, remained clinically stable, and was discharged for outpatient management.DiscussionThis simultaneous convergence of accelerated CLL/SLL and secondary HLH reproduced an RT phenotype. Striking FDG avidity cannot establish transformation, excisional biopsy of the highest uptake lesion remains definitive for diagnosis. Accelerated CLL/SLL is a biologically aggressive gray zone entity enriched for high risk genetics and inferior survival. Malignancy associated HLH carries a poor prognosis and is frequently undertreated. The tension between confirmation and treating an unstable patient was resolved by guideline concordant empiric therapy while tissue was pending, with treatment of the malignant driver serving as definitive HLH therapy. In CLL with disproportionate inflammation and cytopenias, secondary HLH should be considered concurrently rather than attributing everything to transformation.ConclusionAccelerated CLL/SLL and secondary HLH can occur simultaneously and closely mimic Richter transformation clinically and radiographically. Recognizing this overlap, obtaining tissue from the most FDG-avid accessible lesion, and treating both the hyperinflammatory state and its malignant driver are critical to establishing the correct diagnosis and guiding therapy. In this case, a pirtobrutinib-based strategy with obinutuzumab and venetoclax was associated with marked clinical and biochemical improvement, while tissue diagnosis ultimately established accelerated CLL/SLL rather than Richter transformation.

ABSTRACT V-84

Persistent SARS-CoV-2 Infection During Epcoritamab Therapy for Relapsed Diffuse Large B-Cell Lymphoma Successfully Treated with Remdesivir

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ABSTRACT

IntroductionPersistent or prolonged SARS-CoV-2 infection, although not a formalized definition, could be described as the ongoing viral replication beyond 30 days (Machkovech HM et al., Lancet Infect Dis. 2024). Prolonged SARS-CoV-2 infection has been described primarily in immunocompromised patients with impaired humoral immunity (Lee CY et al.Cancer Discov. 2022). Patients receiving B-cell-depleting therapies are at particular risk for persistent COVID infection; most cases reported involve anti-CD20 monoclonal antibodies such as rituximab or obinutuzumab (Little et al., 2026). However, persistent infection associated with bispecific antibody therapy such as Epcoritamab, a CD3xCD20 antibody, is far less characterized. We describe a case of rapid clinical improvement of Epcoritamab-Associated persistent SARS-CoV-2 infection in a patient with Diffuse Large B-cell Lymphoma (DLBCL) following Remdesivir Therapy. Case DescriptionA 76-year-old male with relapsed DLBCL on Epcoritamab presented initially to the ER with weakness, fatigue, fevers, hypogeusia, and anorexia. COVID-19 was confirmed by PCR testing, and was sent home with supportive management. One month later, the patient was admitted due to lack of resolution of symptoms. Repeat PCR and antigen tests remained positive for COVID-19 infection, suggesting ongoing viral replication. Laboratory evaluation revealed leukocytosis with monocytosis. Chest X-ray showed no new pulmonary infiltrates. Given persistent symptoms, and B-cell depletion from Epcoritamb therapy, persistent infection was favored over a post-infectious syndrome. Despite no current guidelines established, a 4-day course of intravenous remdesivir was administered. Administration of antiviral therapy showed rapid and complete resolution of symptoms, supporting active viral replication as the driver of illness.DiscussionThis case contributes to the limited literature on persistent SARS-CoV-2 infection in patients treated with bispecific B-cell-engaging antibodies. It highlights the importance of recognizing persistent infection in individuals with impaired humoral immunity, particularly those receiving B-cell-depleting therapies. These therapies can impair viral clearance and prolong viral shedding, increasing the risk of chronic or relapsing infection. In patients receiving bispecific antibody therapy, prolonged or recurrent COVID-19 symptoms accompanied by sustained test positivity in PCR and antigen testing values should raise suspicion for persistent SARS-CoV-2 infection.Antiviral therapy is traditionally reserved for acute COVID-19 infection. However, in the setting of therapy-induced immunosuppression, antiviral treatment may remain beneficial regardless of symptom duration or apparent disease acuity. As demonstrated in this case, delayed intervention with remdesivir was associated with rapid clinical improvement, suggesting that ongoing viral replication remained the primary driver of symptoms. This case underscores the need for a high index of suspicion for persistent SARS-CoV-2 infection among patients receiving bispecific therapies for hematologic malignancies and provides additional evidence supporting the potential role of antiviral therapy in this under-recognized clinical scenario. Finally, it highlights the need for standardized definitions, diagnostic criteria, and treatment strategies to guide the identification and management of similar cases in the future.

ABSTRACT V-85

Ventricular Septal Rupture Following Vasospastic Myocardial Infarction: A Rare Mechanical Complication of Severe Coronary Vasospasm

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ABSTRACT

Ventricular septal rupture (VSR) is a rare but devastating mechanical complication of acute myocardial infarction, occurring in fewer than 0.3% of patients. Although coronary vasospasm is a recognized cause of myocardial infarction, progression to transmural necrosis and subsequent mechanical complications such as VSR is exceedingly uncommon. We present a rare case of post-infarction VSR associated with severe coronary vasospasm, highlighting a potentially catastrophic manifestation of vasospastic coronary disease.A 72-year-old woman with hypertension, coronary artery disease, and rheumatoid arthritis presented with persistent exertional chest pain associated with nausea, diaphoresis, and fatigue despite optimal medical therapy. Coronary angiography was performed, followed by percutaneous coronary intervention (PCI) to the left anterior descending artery (LAD). Forty-eight hours after discharge, she returned with recurrent chest pain and palpitations. Electrocardiography demonstrated atrial fibrillation with rapid ventricular response and ST-segment elevation in the lateral leads. Repeat coronary angiography revealed a patent LAD stent with severe vasospasm proximal and distal to the stent, which resolved following nitroglycerin administration.Three days later, she developed progressive dyspnea and cough. Physical examination revealed a new grade IV/VI holosystolic murmur with a palpable thrill and radiation throughout the precordium and to the back. Despite an initially unrevealing transthoracic echocardiogram, the marked discordance between the physical examination and imaging findings prompted transesophageal echocardiography, which demonstrated an apical VSR measuring approximately 1.18 cm with a significant left-to-right shunt (Qp/Qs >2). Cardiac computed tomography confirmed an apical muscular ventricular septal defect measuring approximately 1.2 × 1.0 cm.Given her frailty and prohibitive surgical risk, a multidisciplinary Heart Team determined that surgical repair was not feasible. She was transferred for structural heart intervention and underwent successful percutaneous VSR closure with an Amplatzer septal occluder. Despite successful device deployment and defect closure, her subsequent clinical course was complicated by further deterioration, and she ultimately died.Severe coronary vasospasm, although often reversible, can result in myocardial infarction with catastrophic mechanical complications such as VSR. This case emphasizes the diagnostic importance of new physical examination findings and the role of multimodality imaging when initial studies are inconclusive. Early recognition and multidisciplinary Heart Team evaluation are essential to guide timely intervention in this rare, high-mortality complication.

ABSTRACT V-86

When Preeclampsia Is Not The Whole Story: Maternal Cardiovascular Collapse With Thyrotoxicosis

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ABSTRACT

Introduction:Thyroid storm during pregnancy is rare but potentially fatal and may closely resemble severe preeclampsia. Pulmonary edema, tachycardia, hypertension, altered hemodynamics, and multiorgan dysfunction can occur in both conditions, creating a diagnostic challenge when they coexist. Failure to recognize a concurrent endocrine emergency may delay appropriate treatment.Case Description:A 31-year-old woman with Graves disease, chronic hypertension, and medication nonadherence presented with fatigue, cough, progressive edema, and abdominal distention. Initial evaluation demonstrated sinus tachycardia, a B-type natriuretic peptide level of 4,516 pg/mL, and a suppressed thyroid-stimulating hormone level of 0.005 mIU/L. Transthoracic echocardiography showed preserved cardiac structure and systolic function without significant abnormalities.Further evaluation unexpectedly revealed a previously unrecognized 30-week pregnancy complicated by intrauterine fetal demise. Severe preeclampsia with pulmonary edema was diagnosed, and the patient underwent emergent cesarean delivery followed by intensive care admission requiring mechanical ventilation.Persistent cardiovascular instability and biochemical evidence of hyperthyroidism prompted consideration of thyroid storm. Her Burch-Wartofsky score was 45, strongly supporting the diagnosis, and treatment with propylthiouracil, hydrocortisone, propranolol, and iodine was initiated. Despite therapy, she developed atrial fibrillation with rapid ventricular response requiring intravenous rate control. Her hospitalization was further complicated by bilateral pulmonary emboli, for which systemic anticoagulation was initiated.Discussion:This case demonstrates how severe preeclampsia and thyroid storm can coexist and produce overlapping manifestations of maternal cardiovascular decompensation. Although pulmonary edema and tachycardia may initially be attributed to hypertensive disease of pregnancy, marked biochemical hyperthyroidism and persistent hyperadrenergic features should prompt consideration of an additional endocrine process. A normal left ventricular ejection fraction does not exclude severe cardiopulmonary consequences of either disorder.The principal teaching point is that pregnancy-related cardiovascular collapse should not automatically be attributed to a single obstetric diagnosis. In patients with known or suspected thyroid disease, unexplained tachyarrhythmia, pulmonary edema, or disproportionate cardiovascular instability should prompt urgent evaluation for thyroid storm and early multidisciplinary management.

ABSTRACT V-87

From Stress to Tamponade: Hemorrhagic Pericardial Effusion During Recovery From Takotsubo Syndrome

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Program: Universidad Central del Caribe Program

ABSTRACT

IntroductionTakotsubo syndrome (TS), or stress-induced cardiomyopathy, is a transient ventricular dysfunction thought to involve catecholamine excess after emotional or physical stress and frequently mimics acute coronary syndrome. Although small, asymptomatic pericardial effusions are detected by cardiac magnetic resonance imaging in up to 43% of patients, clinically significant involvement is uncommon, and hemorrhagic tamponade without identified ventricular rupture is exceptionally rare. We describe a rapidly evolving bloody pericardial effusion with concurrent pleural effusion during recovery from TS.Case PresentationA 60-year-old woman with hyperlipidemia presented with excruciating substernal chest pain, dyspnea, nausea, and diaphoresis, though hemodynamically stable. Electrocardiography showed no findings suggestive of ischemia, and initial troponins were elevated at 4.84 ng/mL. She was treated for non-ST-elevation acute coronary syndrome with dual antiplatelet therapy and enoxaparin. Initial transthoracic echocardiography (TTE) demonstrated a left ventricular ejection fraction (LVEF) of 30–35%, akinesis of the apical and midventricular segments, hyperdynamic basal segments, and no pericardial effusion. A coronary angiography revealed angiographically normal normal coronary arteries, supporting TS.Five days after admission, radionuclide ventriculography (MUGA scan) demonstrated improving systolic function with an LVEF of 48%, but incidentally suggested a new pericardial effusion. Repeat TTE confirmed a moderate-to-large effusion without hemodynamic compromise. Within hours, the patient developed new-onset hypotension to 88/64 mm Hg. Repeat bedside TTE showed an enlarging effusion with right ventricular diastolic collapse. Emergent pericardiocentesis under fluoroscopic and echocardiographic guidance removed 350 mL of bloody fluid, followed by hemodynamic recovery. Bilateral pleural effusions subsequently developed, requiring thoracentesis. Enoxaparin was reduced to prophylactic dosing; high-dose aspirin and colchicine were started for suspected pericarditis. Computed tomographic angiography showed no pulmonary embolism, thoracic mass, or lymphadenopathy; rheumatologic evaluation was unrevealing, and no ventricular rupture was identified. By hospital day 12, LVEF had recovered to 55–60% with only a small residual effusion. At one month, ventricular wall motion was normal and the pericardial effusion had resolved.DiscussionThis case demonstrates an uncommon progression from TS to inflammatory pericardial disease, hemorrhagic effusion, and tamponade despite improving ventricular function. Although small imaging-detected effusions are relatively common, hemodynamically significant effusions are unusual. Despite recovery of systolic function on imaging modalities, some patients may suffer persistent symptoms, largely attributable to the residual functional and structural myocardial abnormalities, including impaired myocardial energetics and myocardial inflammation. Hemorrhagic effusion should prompt urgent evaluation for ventricular free-wall rupture and alternative etiologies. When no rupture is identified, recent evidence suggests that inflammatory pericardial injury, potentially amplified by antithrombotic exposure is a plausible mechanism, but additional data is required to support it. In conclusion, apparent ventricular recovery in TS should not create false reassurance. New hypotension or systemic symptoms during recovery should prompt immediate repeat echocardiography, and indications for continued antithrombotic therapy should be reassessed once obstructive coronary disease has been ruled out.

2026 ACP PUERTO RICO CHAPTER

CLINICAL VIGNETTES & RESEARCH COMPETITION

Research Presentations Abstracts

ABSTRACT R-01

Monoclonal Gammopathies Among Puerto Rican Populations: A Systematic Review of Prevalence, Incidence, and Outcomes

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ABSTRACT

Background: Monoclonal gammopathy of undetermined significance (MGUS) is a recognized premalignant precursor to multiple myeloma (MM). Although racial and ethnic disparities in the prevalence, incidence, and progression of monoclonal gammopathies have been documented, Hispanic populations remain understudied. The current literature does not clarify whether epidemiologic patterns observed in U.S. populations are applicable to Puerto Rico. The Puerto Rican population possesses a distinct demographic profile compared to mainland Hispanic groups commonly included in research. To date, this area has received limited systematic evaluation. Methods: A systematic search was conducted to identify studies reporting prevalence, incidence, risk factors, progression, or survival outcomes of monoclonal gammopathies (MGUS, smoldering myeloma, multiple myeloma, Waldenström macroglobulinemia, AL amyloidosis) in Puerto Rican populations. Studies that focused exclusively on mainland Hispanic subgroups without Puerto Rico-specific data, case reports lacking confirmed Puerto Rico residency, and non-peer-reviewed sources were excluded. Of 32 records screened, 3 met the inclusion criteria. All included studies utilized data from the Puerto Rico Central Cancer Registry (PRCCR) or its associated Health Insurance and Linkage Database (PRCCR-HILD). Results: Among 1,183 patients with MM identified through PRCCR-HILD, 117 (9.9%) had a documented MGUS diagnosis prior to MM. After adjustment for sex, age, comorbidity index, and insurance, prior MGUS was associated with a 53% reduction in MM-specific mortality risk (SHR, 0.47; 95% CI, 0.32–0.70). This association was also observed among males, adults aged 40–69 years, individuals with a comorbidity index of 0–1, and Medicare beneficiaries. Over a 26-year registry comparison (1987–2013), MM accounted for approximately 72% of deaths among Puerto Rican patients with MM, a proportion similar to that observed in U.S. Surveillance, Epidemiology, and End Results (SEER) program populations. MM-specific mortality declined by 1.8% annually in Puerto Rico, compared with 2.1% and 5.2% annual declines in non-Hispanic SEER populations before and after 2003, respectively, and 3.0% annually among Hispanic SEER patients. From 2001 to 2019, MM incidence increased significantly in Puerto Rico across all age and sex groups (APC, 2.6–6.3%), with the greatest increase among females younger than 65 years (APC, 6.3%). Mortality did not significantly change across age or sex groups. Puerto Rico exhibited the lowest 5-year overall survival among males younger than 65 years (54.6%) and males 65 years or older (34.5%), but not among females. Conclusion: Data regarding monoclonal gammopathies in Puerto Rico remain limited. Although prior MGUS diagnosis was associated with substantially lower MM-specific mortality, only 9.9% of patients with MM had a documented preceding MGUS diagnosis, and no population-based study has directly measured MGUS prevalence on the island. MM incidence has increased across age and sex groups without significant improvement in mortality, which contrasts with declining mortality trends in U.S. populations. These findings highlight a critical gap in understanding the burden, detection, and outcomes of monoclonal gammopathies in Puerto Rico. For internists, these results emphasize the importance of considering plasma-cell disorders in patients presenting with unexplained anemia, renal dysfunction, hypercalcemia, or bone pain. Prospective, population-based studies are required to determine MGUS prevalence and progression in Puerto Rico.

ABSTRACT R-02

PCI Outcomes Under a Remote/On-Call Cardiothoracic Surgical Backup Model

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ABSTRACT

Background: Puerto Rico faces a persistent shortage of on-site cardiothoracic (CT) surgeons, raising the question of whether percutaneous coronary intervention (PCI) can be performed safely at centers relying on a remote/on-call surgical-backup model rather than in-house surgical coverage.Methods: We conducted a retrospective single-center cohort study of 2,396 consecutive cardiac catheterization procedures performed by a single interventional cardiologist under a formal remote/on-call CT-surgical backup arrangement. Procedure and outcome data were abstracted from narrative cardiac catheterization reports, with complication fields coded as absent unless explicitly documented in the procedure note. The primary analysis cohort comprised 425 PCI procedures (17.7% of the total), including chronic total occlusion (CTO), multivessel, and primary STEMI cases. The primary endpoint was a composite of intraprocedural CT-surgeon contact, transfer-protocol activation, emergency CABG, or emergency transfer for surgical rescue. Secondary endpoints included procedural success and 11 individually adjudicated complications. Exact (Clopper-Pearson) confidence intervals were used for zero-event endpoints and Wilson intervals for other proportions. Seven pre-specified exploratory subgroup comparisons were tested against three outcomes (21 Fisher's exact tests in total), with a Bonferroni correction (α=0.00238) applied to control the family-wise error rate.Results: No PCI case (0/425) triggered any component of the primary escalation composite (upper bound of exact 95% CI, ≤0.86–0.87% for each component). Procedural success was 99.3% (422/425; 95% CI 97.9–99.8%). Any documented complication occurred in 9.4% of cases (40/425; 95% CI 7.0–12.6%), and the composite primary outcome (any complication or incomplete procedural success) was met in 9.6% (41/425; 95% CI 7.2–12.8%). Vessel-related events accounted for most complications: coronary dissection occurred in 4.0% (17/424; 95% CI 2.5–6.3%), no-reflow phenomenon in 4.3% (18/422; 95% CI 2.7–6.6%), acute vessel closure in 0.5% (2/423; 95% CI 0.1–1.7%), and vascular access-site complications in 0.2% (1/423; 95% CI 0–1.3%) together roughly 87% of all complication events. Non-vessel events were less frequent: arrhythmia requiring intervention and cardiac arrest during/after the procedure each occurred in 1.2% (5/422; 95% CI 0.5–2.7%), while coronary perforation, cardiac tamponade, procedural stroke, major bleeding, and acute kidney injury each occurred in 0% of cases (upper bound of 95% CI ≤0.9%). Patients selected for PCI were more often male than those undergoing diagnostic-only catheterization (70.6% vs 53.8%; $p<0.0001$) and marginally older (median 67 vs 66 years; $p=0.029$). Of 21 exploratory subgroup tests, 7 were nominally significant ($p<0.05$); only 2 survived Bonferroni correction: CTO-PCI was associated with a lower full-procedural-success rate ($p=0.0006$), and reduced ejection fraction (<40%) was associated with a higher composite-outcome rate ($p=0.0023$).Conclusions: At this single, experienced center, PCI including complex subsets was performed with high procedural success and without any instance of surgical escalation under a formal remote/on-call CT-surgical backup model, consistent with contemporary expert-consensus positions that on-site surgical backup may not be required for selected PCI in appropriately resourced settings. These findings are hypothesis-generating, derived from a single non-randomized center without a comparator arm, and require prospective, multicenter validation before informing practice or policy.

ABSTRACT R-03

Histopathologic Features and CD123 Expression in Lupus Panniculitis: A Retrospective Puerto Rican Case Series

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Program: UCC Lcdo. Eduardo Artau

ABSTRACT

IntroductionLupus panniculitis (LP), also known as lupus erythematosus profundus, is an uncommon deep cutaneous manifestation of chronic cutaneous lupus erythematosus characterized by inflammation of the subcutaneous adipose tissue. Because its clinical and histopathologic features can overlap with subcutaneous panniculitis-like T-cell lymphoma and other inflammatory panniculitides, distinguishing LP from these mimickers may be diagnostically challenging and clinically consequential. Clinicopathologic data on LP in Hispanic and Caribbean populations remain limited, particularly in Puerto Rican patients. This study therefore aimed to characterize the demographic, anatomic, and histopathologic features of LP in a Puerto Rican cohort and to evaluate CD123 immunohistochemical expression as a potential diagnostic adjunct in a subset of cases.MethodsWe conducted an Institutional Review Board–approved retrospective descriptive clinicopathologic study of biopsy-confirmed lupus panniculitis cases diagnosed at a dermatopathology center in Puerto Rico between 2015 and 2023. Demographic characteristics, anatomic distribution, biopsy features, and histopathologic findings were reviewed for each case. Evaluated histopathologic features included the pattern of panniculitis, interface change, hyaline fat necrosis, mucin deposition, perivascular lymphocytic inflammation, calcification, membranocystic change, lymphoid follicles, and plasmacytoid dendritic-cell clusters. CD123 immunohistochemistry was performed in selected cases. Categorical variables were summarized as frequencies and percentages, whereas continuous variables were described using measures of central tendency and dispersion. CD123 expression was quantified to assess its utility in distinguishing inflammatory lymphoid infiltrates from malignant conditions (Chen et al., 2018).ResultsTwenty-nine biopsy-confirmed cases of LP were included. The cohort was predominantly female (93%), with a mean age of 49 years. The extremities were the most frequently affected anatomic locations. All cases demonstrated lobular lymphocytic panniculitis. Hyaline fat necrosis, mucin deposition, and perivascular lymphocytic inflammation were each identified in more than 90% of evaluable specimens. Interface change was present in 53% of cases. CD123 immunohistochemistry was performed in 13 cases, all of which demonstrated CD123-positive plasmacytoid dendritic cells.ConclusionThis retrospective series characterizes the histopathologic spectrum of LP in a predominantly female Puerto Rican cohort. Lobular lymphocytic panniculitis was the most consistent finding, whereas interface change was present in only approximately half of the cases, indicating that its absence does not infer exclusion in LP. CD123 expression was frequent and supports the presence of plasmacytoid dendritic cells within LP, suggesting that CD123 may serve as a supportive immunohistochemical adjunct in appropriately selected cases. However, its diagnostic specificity cannot be established without direct comparison with histopathologic mimickers, including subcutaneous panniculitis-like T-cell lymphoma. These findings contribute to the limited clinicopathologic literature on LP in Hispanic and Caribbean populations.

ABSTRACT R-04

Beyond Shingles Prevention: Varicella-Zoster Vaccination and Reduced Alzheimer's Disease Burden in a Puerto Rican Cohort

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ABSTRACT

Background: Alzheimer's disease (AD) affects approximately 11% of adults aged ≥65 years, with a comparable prevalence in Puerto Rico (12.5%). Emerging evidence suggests that neuroinflammation and latent viral reactivation may contribute to AD pathogenesis. Varicella-zoster virus (VZV), a neurotropic herpesvirus, has been implicated in central nervous system inflammation and vascular injury. However, Caribbean Hispanic populations remain underrepresented in this area of research. We examined whether VZV (zoster) vaccination is associated with AD diagnosis and cognitive outcomes in older Puerto Rican adultsMethods: We conducted a cross-sectional analysis of 91 adults from the Association-Gut-Microbiome-AD cohort. Participants were categorized by zoster vaccination status (vaccinated n=43; unvaccinated n=48). AD diagnosis was determined from clinical records, and cognitive status was assessed using the Montreal Cognitive Assessment (MoCA), Clinical Dementia Rating (CDR), and Geriatric Depression Scale (GDS). Group differences were evaluated using chi-square, Fisher's exact, and t-tests, as appropriateResults: AD was present in 56 participants (61.5%). Vaccinated individuals had a significantly lower prevalence of AD compared to unvaccinated individuals (27.9% vs 91.7%, $p<0.001$), corresponding to markedly reduced odds of AD (OR≈0.04).Vaccinated participants also demonstrated less severe cognitive impairment, with lower rates of moderate-to-severe impairment on MoCA (37.2% vs 68.1%, $p=0.008$) and a higher proportion without dementia on CDR (69.8% vs 12.8%, $p<0.001$). However, vaccinated individuals were younger and had higher educational attainment, and unvaccinated participants exhibited greater cognitive severity and higher use of dementia-related medications, suggesting important baseline differences between groups. No significant differences were observed in GDS scores or APOE genotype distribution.Conclusions: Zoster vaccination was associated with lower AD prevalence and less severe cognitive impairment in this cohort. However, given the cross-sectional design and substantial baseline differences between groups, these findings may reflect confounding, including age, education, and disease severity, as well as potential reverse causation and healthcare utilization bias.These results highlight the need for longitudinal studies with careful temporal characterization of vaccination exposure to determine whether VZV vaccination may influence Alzheimer's disease risk or progression.

ABSTRACT R-05

The Kidneys Can Take It: A Retrospective Analysis of Renal Outcomes after Chest CT Angiography in a Puerto Rican Cohort

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ABSTRACT

Introduction: Concern about contrast-associated acute kidney injury (AKI) is rooted in studies from the 1990s and 2000s that lacked control groups. More recent, better-controlled research has found that intravenous contrast adds little or no risk of AKI, dialysis, or death, even in patients with reduced kidney function. This evidence has led to radiology and nephrology societies now advising against withholding contrast on the basis of kidney function when life-threatening diagnoses are suspected. Nevertheless, prioritizing perceived kidney protection over timely diagnosis, persists among clinicians and can delay chest CT angiography (CTA) for pulmonary embolism or aortic dissection. We hypothesized that AKI after chest CTA is uncommon and is associated primarily with underlying acute illness rather than with contrast exposure.Methods: We conducted a record review retrospective cohort study at Centro Médico Episcopal San Lucas in Puerto Rico. All chest CTA examinations performed between October 2025 and June 2026 were identified by CPT code. Patients receiving maintenance hemodialysis before CTA and those without serum creatinine measured both before and within 72 hours after CTA were excluded. The primary outcome was AKI within 72 hours by KDIGO creatinine criteria; the secondary outcome was percent change in creatinine at 24, 48, and 72 hours. Comparisons were analyzed with chi-square or Fisher's exact tests, Mann–Whitney U tests, and multivariable logistic regression.Results: Of 400 chest CTA examinations reviewed, 218 patients were included (61.9% female; mean age 66.8 ± 16.4 years; median baseline creatinine 0.95 mg/dL). AKI occurred in 25 patients (11.5%; 95% confidence interval [CI] 7.9–16.4%): 56% (14/25) of those patients without pre-existing AKI and 44% (11/25) of those with AKI before CTA. A total of 47 patients had documented AKI present before CTA, 76.6% of which, showed resolving AKI during the 72hrs after chest CTA was performed. Overall, median creatinine decreased at 24 hours (−8.4%), 48 hours (−6.9%), and 72 hours (−9.6%) after CTA. Patients who developed AKI had lower baseline creatinine clearance (median 40.6 vs. 62.6 mL/min, P = 0.028). In multivariable analysis, sepsis or septic shock (adjusted OR [aOR] 9.02, P < 0.001), pre-existing AKI (aOR 3.43, P = 0.018), and diuretic exposure within 24 hours (aOR 3.06, P = 0.029) independently predicted AKI; documented hypotension, clinical evidence of dehydration, and acute decompensated heart failure did not.Conclusion: Serum creatinine declined on average after chest CTA, and most patients with pre-existing AKI did not worsen after contrast. AKI after CTA was associated with markers of acute illness, particularly sepsis. Concern about contrast-associated kidney injury should not delay chest CTA when time-sensitive diagnoses are suspected, furthermore, the risk of not doing a procedure may cause more harm.

ABSTRACT R-06

Bridging the Gap in HFpEF Care: Real-World GLP-1 Receptor Agonist Utilization in Puerto Rico

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ABSTRACT

Introduction: Heart failure with preserved ejection fraction (HFpEF) is increasingly recognized as a cardiometabolic syndrome associated with obesity, systemic inflammation, and impaired functional status. Emerging evidence from contemporary heart failure trials supports glucagon-like peptide-1 receptor agonists (GLP-1 RAs) as a potential obesity-targeted therapeutic strategy in HFpEF. However, real-world utilization in Hispanic populations remains poorly characterized, with limited available data from Puerto Rico.Methods: We conducted a retrospective cross-sectional study of adults with HFpEF (left ventricular ejection fraction ≥50%) and body mass index ≥25 kg/m^2 from two tertiary-care hospitals in northwestern Puerto Rico between 2025 and 2026. Cardiometabolic comorbidities, obesity severity, New York Heart Association (NYHA) functional class, and GLP-1 RA utilization were evaluated. Descriptive analysis was used to characterize eligibility and utilization patterns.Results: A total of 1000 patients were evaluated. Overweight or obesity was identified in 64.7% of patients. Hypertension, diabetes mellitus, and dyslipidemia were highly prevalent, occurring in 93.7%, 66.3%, and 64.3% of patients with available data, respectively. Most patients with documented NYHA status were classified as class II or III, indicating clinically meaningful functional limitation. Despite this high cardiometabolic burden, GLP-1 RA utilization remained low at 4.7% (95% CI: 2.3%-7.1%). Semaglutide represented the predominant prescribed agent, with minimal use of alternative incretin-based therapies.Conclusions: GLP-1 receptor agonists remain markedly underutilized in obesity-associated HFpEF despite emerging evidence supporting clinical benefit. This study provides among the first real-world Puerto Rican data evaluating GLP-1 RA utilization and patient perceptions in HFpEF and identifies a significant evidence-to-practice gap in cardiometabolic heart failure care. These findings underscore the need for improved provider awareness and equitable access to obesity-targeted therapies in Puerto Rico.

ABSTRACT R-07

Factors Associated with Colonoscopy Timing, Colonoscopic Findings, and Clinical Outcomes: A Single-Center Study in Puerto Rico

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ABSTRACT

Background: Optimal timing of colonoscopy in hospitalized patients remains debated across specialties around the world, particularly regarding its effect on diagnostic yield and clinical outcomes. This study aimed to evaluate whether timing of colonoscopy was associated with differences in patient characteristics, colonoscopic findings, and short-term clinical outcomes among 508 hospitalized patients undergoing colonoscopy. Secondary aims were to identify clinical factors associated with time to colonoscopy and to compare findings between early (<48 hours) and delayed (≥48 hours) colonoscopy.Methods: We conducted a retrospective single-center study of 508 hospitalized patients undergoing colonoscopy. Baseline demographics, comorbidities, procedural characteristics, and clinical outcomes were compared between early and delayed colonoscopy groups. Spearman correlation and multivariable linear regression were used to identify predictors of time to colonoscopy, and independent-samples t tests and chi-square/Fisher's exact tests were used to compare continuous and categorical outcomes, respectively.Results: Length of hospital stay differed significantly according to bowel preparation quality ($p<0.001$). Patients with good bowel preparation had the shortest mean hospital stay (7.54±5.80 days), while those with fair (10.16±8.87 days) and poor (10.08±7.55 days) preparation had longer stays, though the effect size was small ($\eta^2=0.033$). Time from admission to colonoscopy did not differ significantly by bowel preparation quality ($p=0.336$). Higher heart rate ($\rho=0.143$, $p=0.001$) and older age ($\rho=0.115$, $p=0.010$) were weakly but significantly correlated with longer time to colonoscopy, while lower initial hemoglobin showed a moderate, significant correlation with longer delays ($\rho=-0.317$, $p<0.001$). Patients who underwent delayed colonoscopy had a greater burden of comorbid illness, including coronary artery disease, chronic kidney disease, and diabetes mellitus (all $p<0.05$), and were more likely to require blood transfusion ($p<0.001$). Diverticulosis was more common among patients undergoing delayed colonoscopy (53.0% vs. 37.7%, $p=0.004$), while colitis/ischemia was identified more frequently among those undergoing early colonoscopy (14.9% vs. 8.6%, $p=0.049$). No significant differences in rebleeding, repeat colonoscopy, 30-day readmission, or in-hospital mortality were observed between early and delayed colonoscopy groups or according to transfusion status.Conclusion: Colonoscopy timing was associated with differences in comorbidity burden, transfusion requirements, and specific colonoscopic findings, but not with short-term clinical outcomes such as rebleeding, readmission, or mortality. Lower initial hemoglobin was the strongest correlate of delayed colonoscopy, suggesting that clinical acuity and hemodynamic status, rather than bowel preparation quality, drive procedural timing decisions in practice. These findings suggest that although earlier colonoscopy may not by itself improve major clinical outcomes, patient-specific factors should continue to guide individualized timing decisions in hospitalized patients undergoing this procedure.

ABSTRACT R-08

Evaluation of ChatGPT and Claude for Hip Fracture Detection on Radiographs: A Sensitivity Focused Study

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ABSTRACT

Artificial intelligence (AI) has increasingly been integrated into healthcare, including radiographic interpretation. Most existing orthopedic AI systems are specialized tools, while general purpose multimodal large language models (LLMs) remain less extensively studied. A clinically important setting in which these models could potentially assist is the evaluation of geriatric hip fractures, where radiographs are the recommended first-line imaging modality and subtle fractures may be missed. Approximately a sensitivity of 26.7% and 22% have been reported in LLMs such as ChatGPT4.0 and Claude Sonnet 3.5, respectively without including subtrochanteric fractures. The purpose of this study was to evaluate newer versions of ChatGPT and Claude for identification of fracture presence and anatomical location on hip radiographs, compare AI-generated management recommendations with treatment provided by the clinical team, and determine whether osteopenia, osteoporosis, or osteoarthritis influenced fracture detection sensitivity. A retrospective review was performed at a single institution from January 1, 2022, through March 30, 2025, for hip fractures diagnosed with ICD-10 codes S72.0, S72.14, and S72.2. Cases were excluded due to incomplete medical records, patients transferred from another institution, pathological fractures, polytraumatic patients, non-operative management and periprosthetic fracture. Fracture presence and location were established by the final radiology report. However, when the radiologist did not identify a fracture, the orthopedic surgeon's operative diagnosis was used as the reference standard. De-identified radiographic images were uploaded to ChatGPT 5.5 and Claude – Fable 5 with a standardized prompt through all the logged cases. Out of 261 hip fracture cases, a total of 217 cases met inclusion criteria. The radiologist identified 206 fractures (sensitivity 94.9%), ChatGPT identified 200 (92.2%), and Claude identified 215 (99.1%) (Table 1). There was no statistically significant difference between ChatGPT and the radiologist for fracture presence ($p=.286$), whereas Claude differed significantly from the radiologist ($p=.022$). For fracture location, ChatGPT demonstrated 72.8% ($n=158$) overall agreement with the radiologist and moderate agreement by Cohen's κ ($\kappa=0.512$, $p<.001$), compared with 52.5% ($n=114$) overall agreement and slight agreement for Claude ($\kappa=0.152$, $p=.007$) (Table 2). ChatGPT recommended the correct surgical procedure in 152 cases (70.0%) compared with 121 cases (55.8%) for Claude. Agreement with the surgical procedure performed was moderate for ChatGPT ($\kappa=0.495$, $p<.001$) and slight for Claude ($\kappa=0.194$, $p<.001$). Across osteoporosis, osteoarthritis, and osteopenia subgroups, ChatGPT sensitivity ranged from 92.3% to 94.9% and Claude from 98.0% to 100%, suggesting that these bone health comorbidities did not meaningfully influence fracture detection sensitivity. In conclusion, Both LLMs demonstrated high sensitivity for hip fracture detection, with Claude achieving the highest sensitivity but lower agreement in fracture localization, while ChatGPT demonstrated better localization agreement despite lower detection sensitivity. Moderate to slight agreement in treatment recommendations further demonstrates that current AI models cannot substitute orthopedic surgeon evaluation. These findings suggest that AI may complement hip fracture evaluation, still its performance remains task dependent and requires orthopedic surgeon interpretation and clinical judgment.

ABSTRACT R-09

Polysaccharide Peptide Suppresses Akt Activation in Association With Time-Dependent Cytotoxicity in A172 Glioblastoma Cells

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ABSTRACT

Introduction: Glioblastoma (GBM) is an aggressive primary brain malignancy characterized by rapid progression, therapeutic resistance, and poor clinical outcomes. Aberrant activation of the phosphoinositide 3-kinase (PI3K)/Akt signaling pathway contributes to GBM cell survival and treatment resistance, making Akt an important target for therapeutic investigation. Polysaccharide peptide (PSP), a bioactive compound derived from Coriolus versicolor, has demonstrated antiproliferative properties; however, its effects on survival signaling in glioblastoma remain poorly characterized. This study investigated whether the time-dependent cytotoxic effects of PSP in human A172 glioblastoma cells are associated with altered Akt activation. Methods: Human A172 glioblastoma cells were treated with PSP at concentrations ranging from 500 to 4,000 µg/mL for 6, 9, and 12 days. Cell viability was assessed by MTT assay, and half-maximal inhibitory concentrations (IC50) were calculated at each treatment interval. To investigate a potential molecular mechanism associated with PSP-mediated cytotoxicity, phosphorylated Akt (p-Akt) protein expression was evaluated by Western blot analysis in PSP-treated cells and corresponding time-matched controls. Western blot experiments were performed in three independent replicates (N=3). Results: PSP produced a progressive time-dependent increase in cytotoxicity in A172 glioblastoma cells. The calculated IC50 decreased from 3,679 µg/mL at Day 6 to 2,080 µg/mL at Day 9 and 710 µg/mL at Day 12, representing an approximately 81% reduction in the PSP concentration required to achieve 50% inhibition between Days 6 and 12. Western blot analysis demonstrated a reproducible trend toward significantly reduced p-Akt protein levels (approximately 0.6-0.2 fold expression) in PSP-treated cells compared with their respective time-matched controls at Days 6, 9, and 12 across three independent experiments. Conclusion: Prolonged PSP exposure progressively enhances cytotoxicity in A172 glioblastoma cells and is accompanied by reduced Akt phosphorylation. The concurrent decline in PSP IC50 and p-Akt levels suggests that attenuation of Akt-mediated prosurvival signaling may contribute to the cytotoxic effects of PSP in glioblastoma cells. These findings provide preliminary mechanistic evidence supporting further investigation of PSP as a potential adjunctive therapeutic candidate for GBM. IRB approval was not required for this study as it does not involve human subjects.