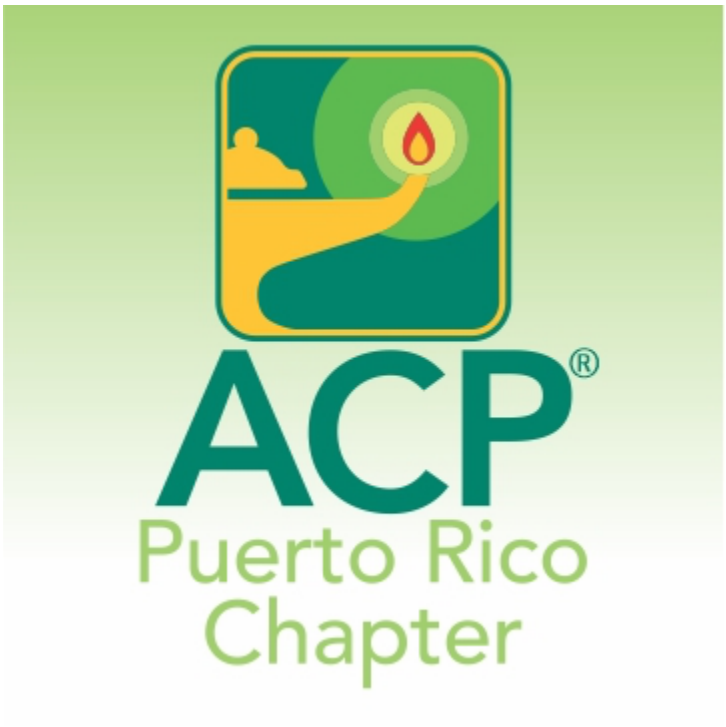

2025 Clinical Vignettes & Research Competition
November 8th, 2025
Embassy Suites by Hilton, Isla Verde PR

ORAL PRESENTATIONS

8:00 AM	**Accelerated Myasthenic Crisis: Bulbar-Onset Myasthenia Gravis Unmasked by Thymoma**	**Jorge Sánchez, MD** Universidad Central del Caribe Program
8:12 AM	**Post-nephrectomy Chyluria**	**José Vargas, MD** VA Caribbean Healthcare System
8:24 AM	**Atypical Adult-Onset Moyamoya Disease Presenting as CNS Vasculitis: A Diagnostic Challeng**	**Paola Alicea, MD** University of Puerto Rico Program
8:36 AM	**Crack in the White Matter: Multiple Sclerosis Masquerading as Toxic Leukoencephalopathy**	**Belissa López, MD** San Juan City Hospital Program
8:48 AM	**Malaria and Epstein Barr Virus Coinfection in a Returned Traveler from the Dominican Republic: Diagnostic Challenges and Successful Treatment with Hydroxychloroquine and Doxycycline**	**Cristian Rosa, MD** Mayagüez Medical Center Program
9:00 AM	**Medical Students Dilemma**	
9:30 AM	**Break**	
10:00 AM	**Hemodynamic Redistribution and Emergent Gastrointestinal Angioectasias After Pulmonary AVM Embolization in Osler-Weber-Rendu Syndrome: A Case Report**	**Diana Moni Febles, MD** Centro Médico Menonita Cayey Program
10:12 AM	**Yellow Plaques in the Colon: Strongyloides Stercoralis–Associated Eosinophilic Colitis After Hurricane Flood Exposure**	**Alexandra Rodil, MD** Damas Hospital Program
10:24 AM	**Life-Saving Rituximab in Severe Paraneoplastic Dermatomyositis with Multi-Organ Involvement**	**Kevin Vargas Feliciano, MD** Mayagüez Medical Center Program
10:36 AM	**Blinded by Leukemia: A Rare Case of Myeloid Sarcoma of the Orbit as the Presenting Sign of Chronic Myelomonocytic Leukemia**	**Zydnia Piñeiro, MD** San Juan City Hospital Program
10:48 AM	**Fatal Disseminated Intravascular Coagulation Secondary to Macrophage Activation Syndrome in a Patient with Adult-Onset Still's Disease**	**Yadiris Vázquez, MD** University of Puerto Rico Program
11:00 AM	**Doctor's Dilemma ® Competition Semi Finals I**	
11:30 PM	**Doctor's Dilemma ® Competition Semi Finals I**	
12:00 PM	**Lunch**	
1:00 PM	**2026 Doctor's ® Dilemma Finals**	
1:30 PM	**Awards**	

CLINICAL VIGNETTE POSTER PRESENTATIONS

V-01- Reversible Hypokalemia-Induced Nephrogenic Diabetes Insipidus in a Critically Ill Patient
Kimberly Medina Morales, MD- University of Medicine and Health Sciences St. Kitts

V-02- Histoplasmosis Unleashed: Disseminated Infection in an Adult living with HIV from Puerto Rico
Luis Colón Berrios, MD - Universidad Central del Caribe Program

V-03- Stung at the Heart: Bee Venom–Induced Immunologic Myocardial Infarction — A Rare Case of Kounis Syndrome
Gonzalo Martínez Ruiz, MD- Universidad Central del Caribe Program

V-04- Spontaneous Tumor Lysis leading to Diagnosis of Aggressive Lymphoma after Non-diagnostic Pulmonary Biopsies
Antonio Raimundi, MD- San Juan City Hospital Program

V-05- TP53-Mutated Myelodysplastic Syndrome in a Young Adult: A Rare Case Defying Epidemiologic Patterns
Gamaliel Monge-Ruiz, MD- San Juan City Hospital Program

V-06- Severe Multisystem Complications Following Bariatric Surgery: A Case of Protein-Losing Enteropathy with Neurologic and Cardiac Involvement
Josué Andino Vega, MD- University of Puerto Rico Progam

V-07- Azathioprine Toxicity Triggered by Allopurinol: A Case of Pancytopenia in Systemic Lupus Erythematosus with Gout
Johnathan Pagán Busigo, MD- VA Caribbean Healthcare System

V-08- Pulmonary Isolation of Shewanella Putrefaciens in a Puerto Rican Patient with a Right Middle Lobe Lung Mass
Alexandra Rodil, MD- Damas Hospital Program

V-09- High Atrioventricular Block After Fontan Surgery in Tricuspid Atresia: Epicardial Pacemaker as Lifesaving Therapy Degree
Radamés Revilla-Orellano, MD- University of Puerto Rico Program

V-10- Genetic Insights into CYB5R Deficiency and Methemoglobinemia in the Puerto Rican Population
Kevin Rodríguez, MD- VA Caribbean Healthcare Program

V-11- "When Lifesaving Therapy Turns Toxic: Pembrolizumab-Induced Necrotizing Glomerulonephritis"
Carolina Gaud Rodríguez, MD- San Juan City Hospital Program

V-12- "Silent Leak, Dramatic Consequence: Pancreaticopleural Fistula Post-ERCP"
Carolina Gaud Rodríguez, MD – San Juan City Hospital Program

V-13- A Silent Spiral: Fatal Thrombotic Microangiopathy in a Patient with Acute Pancreatitis
Sherley Báez, MD- Universidad Central del Caribe Program

V-14- Beyond the Skin: A Case of Disseminated Nocardiosis with Pulmonary Manifestations
William Cortes Figueroa, MD- VA Caribbean Healthcare System

V-15- A Single Stripe, a Startling Clue: Ischemic Colitis in a Healthy Adult
Andrea Pacheco Diaz, MD- VA Caribbean Healthcare System

V-16- "When Thyroid Cancer Strikes the Brain: Hemorrhagic Metastases Revealing a Hidden Threat"
Zydnia Piñeiro, MD- San Juan City Hospital

V-17- Not all Psychosis is Mental: A Case of Hyperactive Delirium Due To Myxedema Coma
Raúl Ríos, MD- VA Caribbean Healthcare System

V-18- Dyspnea In The Postpartum Period: A Rare Cause of Bilateral Empyema Due To S. Aureus
Betzaida Torres, MD- Rotatory Internship Program Auxilio Mutuo Hospital

V-19- A Race Against Time: Spontaneous Tumor Lysis Syndrome as the First Manifestation of Ovarian Adenocarcinoma
Madeline Guerrero González, MD- Mayagüez Medical Center Program

V-20- "It's just a pancreatitis"...Think again!!! Severe Acute Necrotizing Pancreatitis Complicated by Disconnected Duct Syndrome
Edlinn Gener, MD- San Juan City Hospital Program

V-21- A Vein of Malignancy: Multivisceral Resection for IVC Leiomyosarcoma
Marielle Bernard, MD- Consortium Hospital Episcopal San Lucas

V-22- Checkpoint Chaos: A Nivolumab-Associated Immune Event
Daphne Jorge Bezares, MD- VA Caribbean Healthcare System

V-23- Beyond the Gut: Case of Disseminated Histoplasmosis Associated with TNF-Alpha Inhibitor Therapy in the Southern Area of Puerto Rico
Wanda Cubero Cruz,MD- Consortium Hospital Episcopal San Lucas

V-24- When 'Wellness' Turns Harmful: Herbalife®-Associated Drug-Induced Autoimmune-Like Hepatitis in a Patient Pursuing a Healthier Lifestyle
Rafael Cummings López, MD- VA Caribbean Healthcare System

V-25- When the Treatment Feels Like the Disease: A Unique Case of Cefepime-Induced Encephalopathy
Melanie Santos Marrero, MD – University of Puerto Rico Program

V-26- Why Can't I See? The Diagnostic Dilemma of Post-Infectious Optic Neuritis in the Setting of Overlapping Features
José Roque Torres, MD- University of Puerto Rico Program

V-27- The Stroke That Runs in the Family: Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy
Cecilia Soler Llompart, MD- University of Puerto Rico Program

V-28- An Unexpected Uropathogen: Salmonella UTI With Systemic Spread In A Case Of Uncontrolled HIV Patient: A Rare Case Report
Natalia Guevara Rivera, MD- Intership Program Hospital Buen Samaritana

V-29- Amyloidosis Without Plasma Cell Dyscrasia? A Rare Case with Concurrent MDS
Ana Rivera Pagan, MD- Damas Hospital Program

V-30- Hemopericardium and Cardiac Tamponade in a Young Adult: A Cautionary Tale of Warfarin and Cannabis Interaction
Omar Rodríguez Pérez, MD- Mayagüez Medical Center Program

V-31- Metformin Mayhem: A Rare case of it's Toxicity and it's Unlikely Survival
Ludwig Rodríguez Beras, MD- Mayagüez Medical Center Program

V-32- Persistent Cushing Disease Successfully Managed with Mifepristone After Failed Surgery
Karen Sanabria, MD- Ponce Health Sciences University

V-33- Spontaneous Intramuscular Hemorrhage in Refractory Polymyositis: A Rare and Fatal Complication
Paola Alicea, MD- University of Puerto Rico Program

V-34- A Master of Disguise: A Case of 6-Mercaptopurine Overdose Presenting with Clinical Features Mimicking Both Diabetic Ketoacidosis and Thrombotic Thrombocytopenic Purpura
Denis Pérez Fausto, MD- Ponce Health Sciences University

V-35- When Common Pathogens Take Rare Routes: Possibly the Oldest Reported Case of Mycoplasma-Induced Tamponade
Milton Rivera, MD- Damas Hospital Program

V-36- Discordant HIT Testing in Critical Illness: Argatroban-Responsive Thrombosis Despite Negative Functional Assay
Julio Vega-Torres, MD VA Caribbean Healtcare System

V-37- Left-Lobe Predominant Hepatolithiasis in a Young Woman: A Rare Presentation in the Western Hemisphere
Diana Moni Febles, MD- Centro Médico Menonita Cayey Program

V**-38- Ceftriaxone-Induced Acute Generalized Exanthematous Pustulosis in a Patient with Osteomyelitis**
Alexandra Colón, MD- University of Puerto Rico Program

V-39- When Lupus Isn't the Only Culprit: Obstructive Uropathy from Fibroids in a Patient with Lupus Nephritis
Alexandra Colón, MD- University of Puerto Rico Program

V-40- The Heart's Silent Squeeze: A Young Woman's Journey from Liver Disease Misdiagnosis to Pericardial Cure.
María Reyes De Jesús, MD- Universidad Central del Caribe Program

V-41- Pneumonia-Induced Pandemonium: A Neuropsychiatric Complication Of Respiratory Infection
Belissa López, MD- San Juan City Hospital Program

V-42- "Fulminant Hepatic Failure in a Young Woman with Takayasu Arteritis: A Rare and Fatal Complication of Large Vessel Vasculitis"
Yadiris Vázquez, MD- University of Puerto Rico Program

V-43- Diabetes Ketoacidosis In A Patient On Pembrolizumab; A Case Report Of Inmune Checkpoint Inhibitor - Associated Endocrine Toxicity
Eden Ocana Vázquez, MD- Mayagüez Medical Center Program

V-44- Fatal Disseminated Staphylococcus epidermidis Infection in an Uninsured Patient With Untreated HIV/AIDS in Puerto Rico.
Leidiana González Guerrero, MD- San Juan City Hospital Program

V-45- Not So Forgotten After All: Lemmierre-like Syndrome in an Elderly Patient with a Vascular Access Device
Gretchen Abarca, MD- Damas Hospital Program

V-46- Unexpected Culprit: Epiglottitis Secondary to Candida parapsilosis in an Immunocompetent Host
Johnathan Pagan Busigo, MD- VA Caribbean Healthcare System

V-47- From Bump to Breakdown: A Rare Presentation of Leukocytoclastic Vasculitis
Natalia Canevaro Lugo, MD- San Juan City Hospital Program

V-48- Walking on an Empty Tank: Extreme Hemoglobin Tolerance in a Patient with Hereditary Hemorrhagic Telangiectasia
Carolina Machado De La Torre, MD- San Juan City Hospital Program

V-49- Fulminant Mucinous Adenocarcinoma of the Gallbladder in a Recent Migrant
Juan Santos-Rivera,MD- Ponce Health Science University

V-50- Severe Pulmonary Hemorrhage in Leptospirosis: A Case of Unexpected Recovery
Rafael Berrios Sierra, MD- VA Caribbean Healthcare System

V-51- Mediastinal Ectopic thyroid tissue: A Rare Cause of Chest Pain
Sebastián Martínez Carrión, MD- VA Caribbean Healthcare System

V-52- Old Drug, New Target: Rapid IgA Nephropathy Improved with Budesonide
Paola Manrique Pizarro, MD- Universidad Autónoma de Guadalajara

V-53- From Lungs to Electrolytes: The Triad of SIADH from Bacteremia and Empyema
Raúl Ríos, MD- VA Caribbean Healthcare System

V-54- Feathers and Fungi: Severe Pulmonary Histoplasmosis in a Patient with Idiopathic CD4+ Lymphocytopenia
Jefry Mejía, MD- Centro Médico Menonita Cayey Program

V-55- Unmasking HIV-Triggered Hemophagocytic Lymphohistiocytosis in an elderly woman presenting with pancytopenia: A Diagnostic Challenge
Yadriela Colón González, MD- Universidad Autónoma de Guadalajara

V-56- Marginal Zone Lymphoma with IgG Paraproteinemia: A rare case report
Alanis Rodríguez-Rosario, MD- University of Puert Rico Program

V-57- From ACS to Stable Ischemia: Management Dilemmas in Anomalous Left Main from the Right Coronary Cusp — A Three-Case Series
Julio Vega-Torres, MD- VA Caribbean Healthcare System

V-58- The Ace of Spades of Acute Coronary Syndrome: A Case of Yamaguchi Syndrome Mimicking NSTEMI
Bak Nin Choi Reina, MD- Universidad Central del Caribe Program

V-59- Idiopathic Aplastic Anemia in Three Young Adults with Environmental Exposure Histories
Héctor Rivera Jacquez, MD- Rotatory Internship Program Auxilio Mutuo Hospital

V-60- Unmasking the Culprit: Gliptin-Induced Bullous Pemphigoid in a Patient with Type 2 Diabetes Mellitus
Johanna Ortega, MD- Universidad del Central del Caribe Program

V-61- A Rare Duet: Simultaneous Onset of Reactive Arthritis and Erythema Nodosum
Edgar Solis Esquilín, MD- Universidad Central del Caribe Program

V-62- Myxofibrosarcoma in a Patient with Chronic Gout: A Diagnostic Challenge
Leilani Garayua-Cruz, MD- University of Puerto Rico Program

V-63- Rare Case of Terminal Ileum Adenocarcinoma Presenting as Cecal Bascule
Stephanie Cruz Del Valle, MD- Damas Hospital Program

V-64- Unusual Presentation of Extranodal Marginal Node Lymphoma in the Thyroid
Yvonne Suescun, MD- Rotatory Internship Program Auxilio Mutuo Hospital

V-65- Agitation and Psychosis—A Teratoma in Disguise: A Missed Diagnosis of Autoimmune Encephalitis
Ashley Vincenty Acosta, MD University of Puerto Rico Program

V-66- Primary CNS Lymphoma Presenting as Multifocal Brain Lesions in a Solid Organ Transplant Recipient
Marta Vázquez Villegas, MD- Rotatory Internship Program Auxilio Mutuo Hospital

V-67- Surviving the Unthinkable: Necrotizing Pancreatitis in the Setting of Diabetic Ketoacidosis
Jennifer Varela, MD- Rotatory Internship Hospital Auxilio Mutuo

V-68- Pulmonary Sequestration: The Role of Imaging and Surgical Intervention. A Case Report
Roberto Ortiz, MD- Rotatory Internship Hospital Auxilio Mutuo

RESEARCH POSTER PRESENTATIONS

R-01
Activation of STAT1/2 Signaling Pathways by Polysaccharide Peptide in Jurkat T-Cells Highlights a Novel Immunomodulatory Strategy Against HIV
Fabiola Santiago
Universidad Central del Caribe School of Medicine, Bayamón, PR

R-02
Implementing a Pharmacogenomic Driven Algorithm to Guide Antiplatelet Therapy Among Caribbean Hispanics: A Non-Randomized Clinical Trial
Edgardo González García
University of Puerto Rico School of Medicine, San Juan, PR

R-03
Adapt and Survive: Gram-Negative Pathogens Redefining Resistance: A Surveillance Study of Emerging Resistance Mechanisms of Gram-Negative Organisms in Puerto Rico from 2023 to 2025
Mariana Rolan Otero
University of Puerto Rico School of Medicine, San Juan, PR

R-04
From Outcomes to Action: Establishing Protocols in Acute Pancreatitis Management
Remy Rodríguez Chardón
Consortium Hospital Episcopal San Lucas, Ponce PR

R-05
Rehospitalization in Heart Failure: Comparative Impact of Sacubitril/Valsartan Vs. ACE Inhibitors/ARBs
Yomara Huertas Gómez
Consortium Hospital Episcopal San Lucas, Ponce, PR

R-06
Early Clues for Leptospirosis: A Retrospective Analysis at a Tertiary Center in Southern Puerto Rico to Improve Early Screening and Predict Disease Severity of Leptospirosis.
Marielisa Cabrera-Sánchez
University of Puerto Rico School of Medicine, San Juan PR

ORAL PRESENTATIONS ABSTRACTS

Accelerated Myasthenic Crisis: Bulbar-Onset Myasthenia Gravis Unmasked by Thymoma

Sánchez Romero, Jorge, MD; Vélez Figueroa, Andrea C., MD; Viera Maldonado, Carlos F., MD; Rodríguez Pérez, Sarahí, MD
Universidad Central del Caribe Program

Background: Myasthenia gravis (MG) with bulbar onset is a high-risk presentation of this disease associated with rapid neurological deterioration and myasthenic crisis. Thymoma is identified in up to 20% of MG patients and can influence both severity and treatment strategies.1,2
Case Presentation: We report the case of a 53-year-old Hispanic man with a history of hypertension and retinitis pigmentosa who developed an unusually rapid progression of bulbar myasthenia gravis (MG). Despite outpatient initiation of pyridostigmine, he presented within one week with worsening dysphagia and fatigable weakness. Upon admission, his pyridostigmine regimen was optimized; however, he progressed to myasthenic crisis requiring noninvasive positive pressure ventilation (NIPPV) in less than four weeks from initial symptoms. He was treated with a standard five-day course of intravenous immunoglobulin (IVIG), with minimal neurological improvement. Chest CT on admission revealed an anterior mediastinal mass highly suspicious for thymoma. In most patients with thymoma-associated MG, stabilization with immunotherapy allows for hospital discharge and elective outpatient coordination of thymectomy. In this case, however, the patient's symptoms remained severe and refractory despite aggressive inpatient management, necessitating the decision to proceed with radical thymectomy during the same hospitalization. He underwent median sternotomy with resection of the anterior mediastinal mass, partial left phrenic nerve resection, and lymph node dissection. The patient tolerated the procedure well, remained stable postoperatively, and notably avoided endotracheal intubation throughout his course.

Conclusion: This case highlights the importance of early recognition of bulbar-onset MG and coordinated multidisciplinary care. Notably, this patient progressed to crisis much sooner than the typical timeframe reported in large cohorts of thymoma-associated MG, where progression to crisis occurs at a mean of ~18 months compared to ~51 months in non-thymoma MG.3,4 Despite optimized medical therapy and IVIG, his neurological symptoms persisted, necessitating inpatient thymectomy—a departure from the usual practice of stabilizing and arranging surgery electively after discharge. The key learning point is that bulbar MG in the setting of thymoma can deteriorate rapidly and resist standard therapy, warranting heightened vigilance and timely consideration of surgical intervention during the same hospitalization.

Post-nephrectomy chyluria

Vargas, José, MD; Robles Franceschini, Mario J., MD; Manuel Figueroa, José E., MD; Castro Olmo, Coral A., MD
VA Caribbean Healthcare System Program

Introduction: Chyluria, the passage of chyle into urine, commonly associated with parasitic infections, is a rare complication in non-endemic regions. However, disruption of retroperitoneal lymphatics during renal surgery, particularly partial nephrectomy, can result in non-parasitic chyluria. The increasing frequency of nephron-sparing procedures may lead to more cases of this underrecognized entity. Clinical sequelae include malnutrition, weight loss, and immunosuppression.

Case Description: A 62-year-old male with a history of clear cell renal carcinoma (status post left open partial nephrectomy), atrial fibrillation (status post-ablation and permanent pacemaker placement for sick sinus syndrome), essential tremor, gastroesophageal reflux disease, opioid use disorder, presented with persistent chyluria. Two weeks after nephrectomy, he noted the onset of white, milky urine. Over four months, he experienced a 20-pound weight loss, raising concern for protein and fat malabsorption. Initial evaluations led to diagnosis and treatment of complicated urinary tract infections; however, urinalysis and clinical context in subsequent admissions were more consistent with chyluria. Imaging revealed a left perinephric fluid collection later identified as a lymphocele by left retrograde pyelogram.

Interventions included percutaneous drainage and sclerotherapy for the lymphocele, but chyluria persisted. Conservative management was initiated with a low-fat diet (<30% of total energy from fat, emphasizing medium-chain triglycerides). Subcutaneous octreotide was administered 50mcg every eight hours (later changed to 50mcg intravenous infusion over 3 minutes every eight hours due to patient preference, though optimal dosing is not established in the literature). Within 7 days, chyluria resolved, and the patient began regaining weight. No further invasive procedures were required.

Discussion: Post-surgical chyluria is a rare but increasingly recognized complication of nephron-sparing renal surgery, particularly with left-sided and extensive hilar dissection. Diagnosis relies on clinical suspicion, urinalysis for chylomicrons, and imaging to identify lymphatic-urinary fistula or lymphocele. Initial misdiagnosis as urinary tract infection is common due to the nonspecific presentation. The mainstay of management appears to be conservative: a low-fat diet supplemented with medium-chain triglycerides reduces lymphatic flow and promotes closure of the fistula. Octreotide, a somatostatin analogue, may further decrease lymphatic output and has shown efficacy in case series. Most cases resolve with dietary and medical therapy; persistent or refractory chyluria may require sclerotherapy or surgical intervention. This case highlights the importance of early recognition, nutritional support, and a stepwise approach to management. The patient's rapid response to low-fat diet and octreotide is consistent with published outcomes.

Atypical Adult-Onset Moyamoya Disease Presenting as CNS Vasculitis: A Diagnostic Challenge

Alicea, Paola, MD; Soler Llompart, Cecilia, MD; Sánchez Rodríguez, Luis, MD; Colón Roura, Alexandra, MD; Reimón López, Kevin, MD; Viera Rodríguez, Nichole, MD; Oyola Suárez, Andrea, MD; Vázquez Díaz, Yadiris, MD; Ocasio Meléndez, Ileana, MD
University of Puerto Rico Program

Introduction: Moyamoya disease (MMD) is a rare, progressive cerebrovascular disorder characterized by chronic occlusion of the intracranial internal carotid arteries and the development of compensatory collateral vessels. Its presentation can be highly variable, often mimicking other neurological conditions such as primary central nervous system (CNS) vasculitis or infectious meningoencephalitis, particularly in adults with complex comorbidities. We present the case of a patient whose initial presentation and subsequent clinical course highlighted the diagnostic challenges and management considerations in adult-onset MMD.

Case Presentation: A 40-year-old female with a medical history that includes hypertension, hypothyroidism, and type 1 diabetes mellitus was transferred from a peripheral hospital due to a suspicion of meningoencephalitis. Her initial presentation in the emergency department followed a vehicular accident, during which she reported a sudden onset of numbness and weakness on the right side of her body. The patient had previously experienced similar transient symptoms, characterized by intense headaches. During her hospitalization, she received empirical treatment for meningoencephalitis based on her ongoing neurological symptoms.

Two weeks into her stay, the patient exhibited disorientation and personality changes, notably disinhibition and apathy. Additionally, she experienced brief episodes of unresponsiveness, during which she lost bladder control. The neurology team suspected seizure activity, leading to an empirical treatment regimen with Levetiracetam. Her disorientation progressively worsened, culminating in an inability to identify time or place, recognize family members, or form coherent sentences. An MRA/MRV indicated multiple arterial abnormalities, including signal irregularity and segmental stenosis, predominantly affecting the right internal carotid artery, right ACA, and middle cerebral arteries. Further examinations revealed additional focal abnormalities on the left side and bilateral deep watershed infarcts, raising concerns for infectious vasculitis with secondary parenchymal infarctions. Consequently, she was transferred to the University District Hospital for more comprehensive evaluations by both the Neurology and Rheumatology teams, with a particular focus on the possibility of primary CNS vasculitis.

Physical examination revealed right nasolabial flattening and confirmed right-sided hemiparesis. Assessments indicated adequate fluency, yet limited comprehension, suggestive of non-expressive transcortical sensory aphasia. Laboratories were mostly unremarkable, except for an elevated glycated hemoglobin level (8.3). Following a negative lumbar puncture, an extensive autoimmune workup, and various imaging studies, a digital subtraction angiography (DSA) conducted revealed complete occlusion of the bilateral

internal carotid arteries, with collateral circulation facilitated through the posterior cerebral arteries, indicative of a stage 4 Moyamoya pattern.
The patient was managed with aspirin, high-dose statin therapy, and Levetiracetam, while an EEG demonstrated no epileptic activity. Her glycemic control was optimized utilizing a basal-bolus insulin regimen, given the constraints of poor social support and ongoing deficits, including right hemiparesis and limited comprehension. Discharge planning prioritized arrangements for social placement and rehabilitation services.

Conclusions: This case highlights the importance of considering MMD in the differential diagnosis of atypical cerebrovascular presentations in adults, particularly when initial symptoms mimic infectious or inflammatory CNS disorders. Early recognition and comprehensive vascular imaging are essential for accurate diagnosis and appropriate management. Multidisciplinary care is crucial for addressing both neurological deficits and comorbidities, particularly in patients with limited social support.

Crack in the White Matter: Multiple Sclerosis Masquerading as Toxic Leukoencephalopathy

López, Belissa, MD; Canevaro-Lugo, Natalia, MD; De la Rosa, Verónica, MD; Machado, Carolina, MD; López-Baquero, Rafael, MD; Ruiz-Ramos, Juan, MD
San Juan City Hospital Program

Introduction: White matter abnormalities in young adults with recurrent neurological symptoms often prompt evaluation for multiple sclerosis. However, toxic leukoencephalopathy due to neurotoxic exposures — including cocaine, heroin, and inhalants — can present with overlapping clinical and radiographic features. Misdiagnosis may result in inappropriate management, either withholding immunotherapy in MS or exposing patients with toxic etiologies to unnecessary immunosuppressive therapy.

The 2021 McDonald Criteria remain the standard for MS diagnosis, requiring evidence of dissemination in space(lesions in at least two typical CNS regions) and dissemination in time (clinical relapses, MRI progression, or CSF-specific oligoclonal bands). In patients with substance use history, attribution bias may obscure these diagnostic hallmarks.

We present a case of a woman with recurrent neurological symptoms whose history of cocaine use suggested toxic leukoencephalopathy, but whose clinical evolution, CSF analysis, and steroid responsiveness confirmed MS.

Case Presentation: A 39-year-old woman with asthma and hypothyroidism presented with recurrent episodes of left-sided weakness and numbness over seven years, sometimes associated with blurry vision. Her most recent relapse prompted hospital admission. Past history included intravenous cocaine use, last reported 10 months earlier following successful rehabilitation. She smoked fewer than one pack per day and denied alcohol use. On examination, sensation to light touch was diminished on the left, and gait was mildly ataxic.
MRI brain demonstrated confluent T2/FLAIR hyperintensities in the periventricular white matter, centrum semiovale, and splenium of the corpus callosum, with contrast enhancement in the left caudate and right parahippocampal gyrus. CSF analysis showed mild pleocytosis (21 WBC/μL), protein 50.8 mg/dL, and positive oligoclonal IgG bands with elevated IgG index, consistent with an inflammatory demyelinating process. Infectious studies were negative.
She was treated with high-dose intravenous methylprednisolone, with rapid improvement in motor and sensory deficits. She was discharged with referral for disease-modifying therapy initiation and remains under outpatient neurologic follow-up.

Discussion: This case highlights the diagnostic challenge of distinguishing MS from toxic leukoencephalopathy in patients with substance use history. Cocaine and other neurotoxins can produce symmetric, confluent white matter lesions in the corpus callosum and periventricular regions, overlapping with MS findings. However, toxic

leukoencephalopathy typically presents with encephalopathy, cognitive decline, or ataxia rather than relapsing focal deficits.

In our patient, diagnostic uncertainty arose from her history of cocaine use and the symmetric distribution of lesions. However, the relapsing-remitting clinical course, spinal cord involvement, positive CSF oligoclonal bands, and robust steroid responsiveness supported MS under the McDonald criteria.

Failure to recognize MS could have resulted in misclassification, missed initiation of disease-modifying therapy, and long-term neurological disability. Conversely, misdiagnosing toxic leukoencephalopathy as MS could expose patients to unnecessary immunotherapy. Avoiding these pitfalls requires a systematic approach to white matter disease that integrates history, radiology, CSF, and clinical response. Positive CSF oligoclonal bands, dissemination in time and space, and steroid responsiveness remain decisive discriminators favoring MS. Clinicians must remain vigilant against attribution bias, as timely diagnosis and initiation of disease-modifying therapy are critical to preventing irreversible disability.

Malaria and Epstein Barr Virus Coinfection in a Returned Traveler from the Dominican Republic: Diagnostic Challenges and Successful Treatment with Hydroxychloroquine and Doxycycline

Rosa, Cristian, MD
Mayaguez Medical Center Program

39-year-old female with a medical history of allergic rhinitis initially presented with persistent cyclical fevers, chills, and nausea since during winter season, following travel to the Dominican Republic. She visited the emergency department twice and was diagnosed with urinary tract infection on both occasions, receiving oral levofloxacin and trimethoprim-sulfamethoxazole. Despite therapy, her symptoms persisted, and she developed associated headache, inability to tolerate oral intake, and episodes of vomiting, while denying chest pain, palpitations, or sick contacts.

While inpatient, the patient developed acute hand edema which resolved sporadically. This in addition to eosinophilia, raised clinical suspicion for malaria. Further evaluation revealed coinfection with Epstein-Barr virus (EBV), as evidenced by laboratory confirmation of positive capsid immunoglobulin G (IgG) and immunoglobulin M (IgM) antibodies for EBV.In addition, malaria diagnosis was confirmed due to early Plasmodium-shaped species observed in the peripheral blood smear, consistent with standard diagnostic approaches using microscopy for identification of Plasmodium infection. Given her travel to a malaria-endemic region, prompt recognition and initiation of hydroxychloroquine and doxycycline resulted in clinical improvement. At three-month follow-up, the patient was stable with improved complete blood count (CBC) results. This case underscores the diagnostic challenge posed by non-specific symptoms and the potential for misdiagnosis in returning travelers, particularly in the context of coinfection. Accurate and timely laboratory-based diagnosis, as recommended by the Centers for Disease Control and Prevention, is essential to prevent severe complications. Awareness of the heterogeneous but persistent prevalence of malaria in the Dominican Republic, particularly outside urban and resort areas, is critical for clinicians evaluating febrile patients with relevant travel history. This case highlights the importance of considering both malaria and EBV infection in the differential diagnosis of fever in travelers and the need for vigilance to ensure optimal patient outcomes. Furthermore, it is essential for physicians to maintain a high index of suspicion for malaria in febrile travelers, and to provide comprehensive pre-travel education on malaria risk, chemoprophylaxis, and vector avoidance strategies. Enhanced physician awareness and patient education are critical interventions to reduce the burden of imported malaria in the era of globalization.

Hemodynamic Redistribution and Emergent Gastrointestinal Angioectasias After Pulmonary AVM Embolization in Osler-Weber-Rendu Syndrome: A Case Report

Moni Febles, Diana, MD; Reyes-Jiménez, Carmen, MD; Colón-Sierra, Raúl, MD; Martes Román, Izamar, MD; Rodriguez Pérez, Alexandra, MD
Centro Médico Menonita Cayey Program

Background: Osler-Weber-Rendu syndrome, or hereditary hemorrhagic telangiectasia (HHT), is an autosomal dominant vascular dysplasia characterized by mucocutaneous telangiectasias and visceral arteriovenous malformations (AVMs). Pulmonary AVMs, present in up to half of patients, are often treated with embolization to prevent complications. In patients with pulmonary hypertension, however, occluding a low-resistance shunt may alter venous hemodynamics and precipitate gastrointestinal (GI) vascular lesions.

Case Presentation: A 69-year-old woman with genetically confirmed HHT, pulmonary hypertension, Heart failure with preserved ejection fraction (HFpEF), chronic liver and kidney disease, and prior pulmonary AVM embolization presented with massive hematemesis and hemoptysis. Pre-embolization endoscopy had shown only minimal, non-bleeding duodenal angioectasias. After embolization, she developed multiple actively bleeding duodenal angioectasias and new gastric angioectasias requiring repeated endoscopic therapy. Echocardiography revealed increased pulmonary artery systolic pressure and new IVC dilation, consistent with worsening venous congestion. Despite aggressive management, she developed refractory shock and multi-organ failure, and ultimately expired.

Discussion:

This case highlights a rare complication of pulmonary AVM embolization in HHT with pulmonary hypertension: acute hemodynamic redistribution leading to rapid development of GI angioectasias and severe bleeding. Pulmonary AVMs act as low-resistance right-to-left shunts, and their closure increases pulmonary vascular resistance and central venous pressure. In patients with pulmonary hypertension, this added load can unmask right-sided dysfunction and transmit venous congestion retrograde into the hepatic and portal systems, precipitating GI vascular lesions similar to congestive gastropathy.

Conclusion:

In HHT patients with pulmonary hypertension, pulmonary AVM embolization may precipitate acute hemodynamic changes and GI vascular complications. Pre-procedural hemodynamic assessment and post-procedural GI surveillance are warranted in this high-risk population.

Yellow Plaques in the Colon: Strongyloides stercoralis–Associated Eosinophilic Colitis After Hurricane Flood Exposure

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Strongyloides stercoralis infection represents one of the most challenging parasitic diseases in clinical practice because of its unique capacity for lifelong persistence through autoinfection. Patients may remain asymptomatic for decades or present only with vague gastrointestinal complaints, making diagnosis difficult. This case describes a 63-year-old immunocompetent man from Ponce, who presented for evaluation with intermittent "belt-like" abdominal irritation and bloating. He denied diarrhea, melena, weight loss, fever, recent travel, or NSAID use. His past medical history included depression/anxiety, and osteoporosis; medications were clonazepam, zolpidem, and gabapentin. Notably, he reported prolonged environmental exposure to floodwater during Hurricane Maria rescue operations.

A prior colonoscopy in 2019 demonstrated diverticulosis with biopsies showing dense eosinophilic infiltrates of unclear significance. Surveillance colonoscopy performed on April 29, 2025, demonstrated diverticulosis and multiple yellowish plaques diffusely distributed throughout the colon, most prominent in the right and left segments. Targeted biopsies of the colon revealed mucosal eosinophilia without visible organisms on hematoxylin and eosin staining. Laboratory evaluation showed WBC 6.17 K/μL with eosinophils 11.2%, hemoglobin 13.4 g/dL, ESR 3 mm/h, CRP <0.5 mg/L, and negative ANA. Stool ova and parasite studies and Giardia antigen were negative, but Strongyloides IgG serology returned positive. The patient was treated with ivermectin 200 μg/kg PO.

The clinical importance of this case lies in its demonstration of an underrecognized colonoscopic phenotype—diffuse yellowish mucosal plaques associated with eosinophilic inflammation—that can mimic other gastrointestinal disorders and create diagnostic pitfalls. Colonic involvement by Strongyloides is rare and typically lacks distinctive features. In this patient, the diffuse plaques were initially indistinguishable from pseudomembranous colitis or primary eosinophilic colitis. Histology further complicated interpretation by showing only mucosal eosinophilia without visible parasites. Negative stool ova and parasite testing underscored the limited sensitivity of conventional diagnostic tools, emphasizing the need to integrate colonoscopic findings, serology, and clinical context.

Environmental and exposure history played a decisive role in raising suspicion. The patient's prolonged contact with floodwater provided a plausible infection route and critical epidemiologic clue that might otherwise be overlooked. Importantly, his symptoms were minimal—limited to intermittent abdominal discomfort and bloating—without diarrhea or systemic inflammation.

The most significant clinical implication is the potential for disastrous mismanagement if such cases are misdiagnosed as inflammatory bowel disease or primary eosinophilic colitis. Corticosteroids, often considered in those settings, can trigger Strongyloides hyperinfection syndrome, unleashing uncontrolled dissemination of larvae. Mortality rates

in hyperinfection exceed 60–80%, highlighting that exclusion of Strongyloides is mandatory before initiating immunosuppressive therapy in eosinophilic gastrointestinal disease of uncertain origin. Recognition of yellowish plaques as a potential visual marker of colonic strongyloidiasis, particularly in patients with environmental or geographic risk factors, can prevent catastrophic errors.

This case illustrates that colonic strongyloidiasis may present with a distinctive plaque-like endoscopic pattern and eosinophil-predominant inflammation, even in immunocompetent hosts. Its clinical importance lies in recognizing this underappreciated phenotype, and avoiding inappropriate corticosteroid therapy that could precipitate lethal hyperinfection. Timely diagnosis and treatment not only resolve symptoms but also avert catastrophic outcomes.

Life-Saving Rituximab in Severe Paraneoplastic Dermatomyositis with Multi-Organ Involvement

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Introduction: Dermatomyositis (DM) is an idiopathic inflammatory myopathy frequently associated with malignancy. Severe manifestations may affect the heart, lungs, and larynx, resulting in a poor prognosis. Although rituximab is not considered first-line therapy, it may be life-saving in refractory cases.

Case Presentation: A 60-year-old woman presented with proximal muscle weakness, cutaneous rashes, dysphonia, and dysphagia. Examination revealed heliotrope rash, V-sign, and shawl sign distribution rashes, Gottron's papules, nailfold changes, and a palpable left breast mass. Work-up demonstrated elevated inflammatory and muscle markers, diffuse myositis on MRI, and EMG-confirmed myopathy. Excision biopsy revealed invasive ductal carcinoma (ER-positive, PR-negative, HER2-negative). A myositis antibody panel, obtained after rituximab initiation, was negative for myositis-specific and myositis-associated antibodies.

Initial treatment with prednisone, methotrexate, and IVIG yielded minimal improvement. The patient developed acute respiratory failure requiring bilevel positive airway pressure (BiPAP) and new-onset heart failure with reduced ejection fraction (HFrEF <20%). Rituximab administration resulted in improved muscle strength, oxygen independence, and stabilization of cardiopulmonary function. A deltoid biopsy performed after the first rituximab infusion showed only chronic myopathic and denervation changes without active inflammation, consistent with treated or "burnt-out" DM. She tolerated a second rituximab dose and was discharged with a wearable cardioverter-defibrillator, guideline-directed HFrEF therapy, and ongoing immunosuppression. Anastrozole was started for breast cancer, with plans for continued antineoplastic therapy.

Discussion: Rituximab demonstrated efficacy in refractory, seronegative paraneoplastic dermatomyositis with multi-organ involvement. The negative antibody panel and biopsy findings likely resulted from prior treatment rather than the absence of true disease. This case highlights the potential of rituximab as a life-saving intervention, the impact of treatment timing on diagnostic accuracy, and the importance of multidisciplinary management in paraneoplastic DM.

Blinded by Leukemia: A Rare Case of Myeloid Sarcoma of the Orbit as the Presenting Sign of Chronic Myelomonocytic Leukemia

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Chronic myelomonocytic leukemia is a clonal hematopoietic stem cell disorder that combines features of both myelodysplastic and myeloproliferative neoplasms. While the disease typically presents with cytopenias, monocytosis, and constitutional symptoms, extramedullary involvement is rare and portends a poor prognosis. Myeloid sarcoma, a tumoral mass of immature myeloid cells occurring outside the bone marrow, is an uncommon but aggressive manifestation of CMML and is typically associated with transformation to acute myeloid leukemia. Orbital infiltration is exceedingly rare and can result in rapid and irreversible vision loss if not promptly identified. We report a unique case of CMML presenting with left eye ophthalmoplegia and blindness due to leukemic infiltration of the optic nerve.

A 70-year-old male with a history of myelodysplastic syndrome diagnosed in 2023, treated with Azacitidine and Venetoclax and prostate cancer in remission presented to the emergency department with acute left eye vision loss, retro-orbital pain, and mild dizziness. The patient reported a one-month history of progressive blurry vision that acutely worsened over two days.

On exam, he was alert and oriented with left eye ophthalmoplegia, a non-reactive dilated pupil, and no light perception. Extraocular movements were markedly limited, and the left orbit appeared proptotic with associated subconjunctival hemorrhage. The right eye was intact.

Initial imaging including head CT and CTA ruled out aneurysm or acute ischemia but showed a left temporal lobe hyperdensity. MRI of the brain and orbits revealed T2 hyperintensity, enlargement, and enhancement of the left optic nerve suggestive of leukemic infiltration. Ophthalmologic evaluation confirmed an infiltrative lesion of the optic nerve with subretinal hemorrhage and macular edema.

Bone marrow biopsy revealed persistent myelodysplasia with 7% blasts and molecular findings including TET2, ASXL1, and RUNX1 mutations. Next-generation sequencing was consistent with high-risk CMML. CSF analysis from lumbar puncture further confirmed the presence of leukemia.

The patient was initiated on cytoreduction with hydroxyurea and later received induction chemotherapy with cladribine, cytarabine, and venetoclax. He underwent three intrathecal methotrexate treatments and completed 10 fractions of external beam radiation therapy to the orbit. Ophthalmologic follow-up revealed partial improvement in extraocular movements, but vision in the affected eye was not recovered.

Orbital myeloid sarcoma is a rare manifestation of CMML, more commonly associated with AML, and may present with diplopia, proptosis, or visual loss. This case illustrates that rapidly progressive ocular symptoms, even without systemic blast crisis, warrant high suspicion and urgent imaging in patients with hematologic disorders. Diagnosis was supported by imaging, cytology, and the patient's clinical course, with TET2/ASXL1 mutations and >5% blasts indicating higher-risk disease with potential for AML transformation. CNS involvement in CMML is exceedingly rare and portends poor prognosis. Treatment typically involves aggressive chemotherapy and radiotherapy. Multidisciplinary collaboration across oncology, neurology, radiology, and ophthalmology is critical to timely management.

Orbital myeloid sarcoma in CMML is a rare but vision-threatening condition. Prompt recognition, imaging, histopathology, and combined systemic and local therapies are essential to stabilize progression. Clinicians should maintain vigilance for ocular symptoms in myeloid neoplasms, as early intervention may preserve function and prolong survival.

Fatal Disseminated Intravascular Coagulation Secondary to Macrophage Activation Syndrome in a Patient with Adult-Onset Still's Disease

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Adult-onset Still's disease (AOSD) is a systemic inflammatory disorder of unknown origin, typically affecting young adults. Diagnosis is primarily clinical and requires exclusion of infectious, neoplastic, and other rheumatic conditions. AOSD can lead to life-threatening complications, one of which is Macrophage Activation Syndrome (MAS), which occurs in up to 15% of AOSD patients and is regarded as the most severe complication, characterized by a high mortality rate.

A 22-year-old Asian male with no significant medical history presented with pleuritic chest pain, persistent febrile episodes, myalgia, sore throat, and odynophagia lasting for the past week. On physical examination, the patient appeared diaphoretic and had a distinct salmon-colored rash. Laboratory tests revealed leukocytosis and elevated inflammatory markers, including a ferritin level of 14,700. Renal function was preserved, and an electrocardiogram (EKG) showed diffuse ST elevation suggestive of pericarditis. Despite a thorough infectious workup—including blood cultures that returned negative—initial empirical treatment with antibiotics yielded no improvement. Tests for HIV, CMV, RPR, hepatitis panel, sexually transmitted diseases, leptospirosis, and dengue were all negative. During his hospitalization, new onset of transaminitis was noted, and an abdominal ultrasound revealed hepatomegaly. Although the patient did not exhibit arthritis, he fulfilled the Yamaguchi criteria for Adult-onset Still's disease, he met major criteria (fever ≥39 °C for >1 week, salmon-colored rash, leukocytosis) and minor criteria: (sore throat, hepatomegaly with abnormal liver function tests, negative ANA/RF, and serositis). Despite IVIG and immunosuppressive therapy, the patient experienced persistent fever, new-onset pancytopenia, elevated LDH/CPK, lactate, hypertriglyceridemia, and worsening liver function. These findings suggested the patient had developed Macrophage Activation Syndrome as a complication of AOSD. The rheumatology team decided to treat the patient with cyclosporine and IL-1 as second-line immunosuppressive agents. Unfortunately, the patient progressed to develop DIC secondary to MAS, leading to supportive treatment involving platelets, packed red blood cells (PRBC), fresh frozen plasma (FFP), and cryoprecipitate. Despite a multidisciplinary approach, the patient ultimately succumbed to multiorgan failure as a result of a flare of AOSD evolving into MAS.

This case underscores the diagnostic complexities associated with AOSD in young patients. MAS is a severe and life-threatening complication that can escalate to DIC, further increasing mortality risk. Distinguishing between MAS from a simple flare of Still's disease is crucial. Based on current literature and this case presentation, specific factors such as cytopenia, elevated LDH, and coagulopathy may help facilitate early detection of MAS. Extreme hyperferritinemia at the onset of Still's disease serves as a prognostic indicator for the development of MAS. Early recognition and prompt initiation of appropriate therapies, including steroids and immunosuppressants, are vital for improving outcomes and reducing mortality.

Clinical Vignettes Posters Abstracts

V-01 Reversible Hypokalemia-Induced Nephrogenic Diabetes Insipidus in a Critically Ill Patient

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Diabetes insipidus (DI) is a condition characterised by a large volume of diluted urine production and increased thirst. Nephrogenic DI is either congenital or acquired and includes chronic kidney diseases, metabolic conditions such as hypokalemia and hypercalcaemia; drugs such as lithium and demeclocycline; osmotic diuresis such as glucose and mannitol; amyloidosis; and myelomatosis. In this case, hypokalemia is the cause of nephrogenic DI. This case presentation discusses a 46-year-old male with a past medical history of hypertension, who was referred to Internal Medicine (IM) following surgical intervention for colostomy closure. The patient had initially undergone surgery due to perforated diverticulitis, complicated by extensive peritonitis and anastomotic leakage, requiring ICU admission for respiratory failure and sepsis management.

During his ICU stay, the patient was intubated, sedated and with vasopressor. During that time, the patient developed partial nephrogenic DI in the setting of severe hypokalemia, polyuria and hypernatremia. Initial laboratory findings revealed hypernatremia (Na 185 mEq/L), hypokalemia (K 3.3 mEq/L), and acute kidney injury (creatinine 1.1 → 1.8 mg/dL within 24 hours). Urine output exceeded 10 L/day. Urine osmolarity was unable to calculate since urine glucose levels were not available by the lab. After evaluating reversible causes of nephrogenic DI, nephrology recommended intravenous D5W (5% dextrose in water) at 300 mL/hr and aggressive potassium replacement to maintain K >3.5 mEq/L.
In this case, IV D5W was used to provide free water and gradually lower serum sodium while potassium was corrected in parallel. Over the following days, serum sodium trended down (185 → 165 mEq/L) as potassium normalized (>4.0 mEq/L). The following week, despite persistent polyuria (~20 L/day), the IM team stopped D5W, continued with electrolyte correction, and started free water replacement. These gradually stabilized sodium (144 mEq/L) with full resolution of partial nephrogenic DI.

This rare and reversible form of partial nephrogenic DI was resolved with potassium correction, highlighting the pathophysiological link between hypokalemia-induced downregulation of aquaporin-2 and reduced renal response to arginine vasopressin (AVP), which decreases the ability to concentrate urine. It is important to recognize reversible causes of nephrogenic DI such as electrolyte imbalances and differential diagnoses like Gitelman syndrome. The presentation serves as an educational tool to reinforce the evaluation and management of polyuria in critically ill patients, particularly in the context of hypokalemia.

V-02- Histoplasmosis Unleashed: Disseminated Infection in an Adult living with HIV from Puerto Rico

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Histoplasmosis is an endemic mycosis in Puerto Rico. In immunocompetent individuals, infection is often asymptomatic or limited to a mild, self-limited pulmonary illness. In contrast, disseminated histoplasmosis occurs when the infection spreads beyond the lungs, most commonly in immunocompromised patients, particularly those with advanced HIV/AIDS. Disseminated disease carries a significantly higher risk of morbidity and mortality, making early recognition and treatment essential.
A 32-year-old man with perinatally acquired HIV, diagnosed at age 13, presented with two weeks of night sweats, low grade fevers, progressive weight loss, malaise, dry cough, hemoptysis and watery diarrhea. His HIV history was notable for poor adherence to antiretroviral therapy over the preceding three years. On admission, physical examination revealed cachexia, and disseminated lymphadenopathy. Laboratory evaluation showed elevated liver enzymes, with no significant alteration on cell lines except for a CD4 count of 41.9 cells/µL and HIV viral load of 61,900 copies/mL. Chest radiography demonstrated diffuse reticulonodular infiltrates suggestive of a miliary pattern of infection. While abdomino pelvic CT showed evidence of reticuloendothelial system involvement including hepatomegaly, paraaortic and mesenteric lymphadenopathy, in addition to a 25mm right adrenal nodule. Given the endemic burden and radiographic findings, the initial differential diagnosis was broad, including Mycobacterium tuberculosis, for which empiric therapy was initiated. Due to the degree of immunocompromise, an extensive laboratory workup was performed to rule out alternative pathological scenarios, including the most common opportunistic infections seen in advanced HIV/AIDS. Once urine antigen testing for Histoplasma capsulatum returned positive (19ng//mL), tuberculosis therapy was discontinued and induction therapy with intravenous liposomal amphotericin B was initiated without delay. Bone marrow biopsy subsequently confirmed the diagnosis, demonstrating intracellular yeast forms consistent with Histoplasma capsulatum on histopathology. Antiretroviral therapy was already reinitiated 2 weeks before hospitalization and maintained throughout the antifungal course while inpatient. After completion of induction therapy, the patient was transitioned to oral itraconazole for consolidation and maintenance to complete a 12-month course, with clinical improvement.

This case illustrates the diagnostic complexity of systemic infections in immunocompromised patients in endemic regions like Puerto Rico, where tuberculosis, and endemic mycosis often overlap clinically and radiographically. Following IDSA recommendations, rapid antigen detection enabled timely initiation of amphotericin B induction therapy, while bone marrow histopathology provided definitive confirmation. Early discontinuation of empiric TB treatment, structured antifungal therapy amphotericin B followed by itraconazole), and timely antiretroviral management were central to patient recovery. In Puerto Rico, disseminated histoplasmosis must remain a key diagnostic consideration in HIV patients presenting with systemic symptoms. Differentiating localized

from disseminated infection underscores the importance of host immune status, and rapid diagnostic testing permits early initiation of targeted antifungal therapy with histopathologic confirmation.

V-03- Stung at the Heart: Bee Venom–Induced Immunologic Myocardial Infarction — A Rare Case of Kounis Syndrome

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Introduction:
Acute coronary syndromes (ACS) usually result from atherosclerotic plaque rupture or erosion, but not all myocardial infarctions result from obstructive disease. Kounis syndrome, or allergic ACS, represents the intersection of hypersensitivity reactions and myocardial ischemia. First described in 1991, this condition is mediated by inflammatory cells leading to coronary vasospasm or plaque destabilization. We describe a case of bee sting–induced Type I Kounis syndrome presenting as myocardial infarctions.
Case Presentation:

A 50-year-old female with hypertension and hypothyroidism presented with diffuse urticaria, facial edema, and severe substernal chest pain minutes after a bee sting. She was anxious and in distress, with diffuse wheezing and mild jugular venous distention. Laboratory revealed leukocytosis and progressive rise in high-sensitivity troponin (201>951>1836 ng/L). ECG showed T-wave inversions in anterior and inferior leads, and chest radiograph was remarkable for pulmonary edema. Transthoracic echocardiography revealed anterior wall hypokinesis and reduced systolic function, whereas coronary angiography showed normal coronaries.

Management:
She was treated for anaphylaxis with intramuscular epinephrine, corticosteroids, and antihistamines, alongside aspirin, enoxaparin, and nitroglycerin infusion for suspected ACS. After angiography confirmed normal coronaries, anticoagulation was discontinued, and therapy was refocused on vasospasm control with nitrates and calcium channel blockers. Her symptoms resolved within 48 hours and was discharged on vasospasm-directed therapy, ACE inhibitor, and epinephrine auto-injector. Allergy follow-up was arranged.

Discussion:
Kounis syndrome is an uncommon but clinically important cause of ACS in which allergic/hypersensitivity reactions trigger coronary vasospasm and myocardial ischemia. Mast cell mediators such as histamine, leukotrienes, and platelet-activating factor promote vasoconstriction, endothelial dysfunction, and platelet aggregation.

Three subtypes are recognized: Type I (vasospasm in normal arteries), Type II (plaque destabilization in atherosclerosis), and Type III (stent thrombosis). Our patient demonstrated Type I disease, with classic allergic manifestations, elevated biomarkers, ischemic changes, and normal angiography.

Current ACC/AHA and ESC guidelines lack recommendations for Kounis syndrome, leading clinicians to follow standard ACS protocols. This can result in unnecessary anticoagulation, invasive procedures, or prolonged dual antiplatelet therapy without addressing the underlaying mechanism.

Management requires balancing urgent treatment of anaphylaxis with strategies to relieve coronary vasospasm. Nitrates and calcium channel blockers are firstline, while β-blockers and morphine should be avoide. β-blockers can worsen coronary vasospasm by leaving α-adrenergic effects unopposed and may blunt the response to epinephrine, while morphine can trigger additional histamine release, aggravating vasodilation and allergic symptoms. Long-term care includes allergen avoidance, epinephrine auto-injector prescription, and referral for venom immunotherapy.

Conclusion:

This case illustrates Type I Kounis syndrome, where allergic mediator release causes coronary vasospasm and myocardial injury in structurally normal arteries. Despite being reported in up to 1% of allergic hospitalizations, the syndrome remains underrecognized. Major cardiology guidelines, including ACC/AHA, do not provide specific recommendations making it management challenging. Limited awareness can result in unnecessary and/or inappropriate treatments, delay of care and worsening patient outcomes. Prompt recognition allows us to provide adequate therapy while addressing life-threatening anaphylaxis. Long-term prevention is cornerstone in management, involving allergen avoidance, epinephrine auto-injector education, and referral for immunotherapy.

V-04- Spontaneous Tumor Lysis leading to diagnosis of Aggressive Lymphoma after Non-diagnostic Pulmonary Biopsies

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Tumor lysis syndrome (TLS) typically follows cytotoxic therapy for hematologic malignancies. Spontaneous TLS (sTLS) at presentation, especially with an acute respiratory failure and undiagnosed thoracic malignancy, is uncommon and requires time-sensitive management. We report a case of sTLS that unmasked an aggressive lymphoproliferative disorder after multiple non-diagnostic bronchoscopic procedures, with the diagnosis ultimately confirmed by repeat pleural fluid cytology.

This is a case of a middle-aged man presenting several weeks of weight loss, anorexia, and progressive dyspnea, rapidly deteriorating and developing an acute hypoxemic respiratory failure that required immediate endotracheal intubation and ICU admission upon arrival. Initial studies revealed sTLS with severe hyperuricemia (>30 mg/dL), hyperkalemia (7.8 mmol/L), hypocalcemia (4.9 mg/dL), and metabolic acidosis (pH 7.18; HCO_3^- 13 mmol/L), accompanied by an anuric acute kidney injury (AKI). He received rasburicase, high-dose dexamethasone, meticulous electrolyte control, and intermittent hemodialysis, with serum urate declining to 3.6 mg/dL and partial clinical stabilization. Cross-sectional imaging demonstrated a ~13-cm heterogeneous, partially necrotic right-lung mass with mediastinal lymphadenopathy, bilateral pleural effusions, and a small pericardial effusion.

Initial diagnostic attempts were unrevealing; bronchial washings and two broncho-alveolar lavages (BALs) were negative for malignancy. Transbronchial fine-needle biopsies (FNAs) of the right mass and 11R node and a small forceps biopsy were non-diagnostic, only necrotic tissue obtained. Early left-sided pleural fluid cytology was reported as negative for malignancy with reactive mesothelial cells and supportive immunostains. A subsequent left pleural tap showed lymphocyte-predominant effusion and that warranted flow cytometry. Within 48 hours, right pleural fluid cytology returned positive for malignancy, demonstrating a lymphoproliferative disorder, and repeat bilateral pleural fluids confirmed malignant lymphomatous effusions. With the new cytology, hematology service initiated expedited immunophenotyping (flow cytometry) and viral testing to define subtype and begin definitive therapy. The patient required multidisciplinary coordination while he remained on ventilatory and renal support, with ongoing large-volume pleural drainage.

Spontaneous TLS can be the presenting syndrome of aggressive lymphoma, even before therapy. Abrupt hyperuricemia with phosphate elevation, hypocalcemia, and AKI meeting Cairo–Bishop laboratory and clinical criteria, should trigger treatment before tissue confirmation. When early endobronchial/transbronchial sampling is non-diagnostic in a necrotic, bulky thoracic mass, clinicians should biopsy smart; either target non-necrotic areas via EBUS/IR or shift to pleural fluid cytology/flow, which may yield faster, and safer diagnosis in unstable patients. Teams must balance diagnostic yield against renal risk and respiratory instability. In this patient, immediate rasburicase and dialysis stabilized multiorgan failure, and created a window for high-yield sampling that ultimately established a lymphoproliferative malignancy.

V-05- TP53-Mutated Myelodysplastic Syndrome in a Young Adult: A Rare Case Defying Epidemiologic Patterns

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Myelodysplastic syndrome (MDS) is a heterogeneous group of clonal hematopoietic stem cell disorders characterized by myeloid dysplasia, ineffective hematopoiesis, and peripheral cytopenias affecting one or more cell lines. MDS predominantly affects older adults, with a median age at diagnosis of approximately 70 years, and is uncommon before age 50. Presentation in patients in their twenties is extremely rare. Population data show an overall prevalence of ~0.02% (~20 per 100,000), and cases under 30 are exceptionally uncommon, making diagnosis at age 23 extraordinary. Reporting a 23-year-old with TP53-mutated, MF-3 MDS who underwent haploidentical allogeneic hematopoietic stem cell transplantation (HSCT) with early hematologic recovery provides valuable insight into disease behavior and transplant outcomes in this underrepresented age group.

We present a 23-year-old previously healthy male with no significant family history who arrived at the emergency department with a 1-week history of dizziness, exertional dyspnea, gingival bleeding, and profound pancytopenia (Hgb 5.6 g/dL, ANC 494/μL, platelets 12,000/μL). Given concern for acute leukemia, a bone marrow biopsy was performed, revealing an MDS with 80–90% cellularity with extensive fibrosis (MF-3), megakaryocytic atypia, and 3–5% blasts. Next-generation sequencing identified a TP53 mutation.

Combination treatment was started on azacitidine plus venetoclax. After two cycles, repeat marrow showed variable cellularity with persistent dysplasia, 5–6% blasts, and a TP53 R282W mutation with a variant allele frequency of 25%. Given these high-risk features, he underwent haploidentical allogeneic HSCT from his sibling following myeloablative conditioning with fludarabine and busulfan. Graft-versus-host disease (GVHD) prophylaxis included tacrolimus, mycophenolate, and post-transplant cyclophosphamide.

Early post-transplant complications included grade 3 mucositis, mild gastrointestinal symptoms, grade 1 cytokine release syndrome, and an episode of aspiration pneumonia. Neutrophil engraftment was achieved by day +14, and he was discharged on day +16. At day +48, he reported good functional recovery, with stable blood counts, preserved renal and hepatic function, and no clinical or laboratory evidence of acute GVHD or disease relapse.

Despite adverse molecular and histopathologic features typically associated with poor outcomes, the patient achieved early hematologic recovery and remains in remission early post-transplant, likely due to his young age and absence of comorbidities. This case underscores the importance of prompt recognition and timely transplant referral in young patients with TP53-mutated MDS with high-grade fibrosis and provides valuable data regarding outcomes after haploidentical HSCT in this rare demographic.

V-06- Severe Multisystem Complications Following Bariatric Surgery: A Case of Protein-Losing Enteropathy with Neurologic and Cardiac Involvement

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Introduction:
Bariatric surgery is increasingly common and generally safe, but patients who miss regular postoperative follow-up can develop serious complications. Protein-losing enteropathy (PLE), severe malnutrition, and neurological complications are uncommon but potentially life-threatening. We describe a young woman who developed profound multisystem disease one year after Roux-en-Y gastric bypass (RYGB) performed abroad.

Case Presentation:
A 33-year-old Latina woman with a history of hypothyroidism and RYGB performed in Mexico one year earlier presented to the emergency department due to worsening fatigue and altered mental status. Physical examination findings were consistent with anasarca and Glasgow Coma Scale of 9/15. Laboratory findings revealed hypoalbuminemia, anemia, hypocalcemia, elevated thyroid- stimulating hormone, and low free T4 and T3 levels. She was also found to have a left leg deep vein thrombosis and features consistent with PLE.

During her hospital stay, her mental status worsened, and she experienced status epilepticus requiring intubation. Neurological evaluation raised concern for osmotic demyelination syndrome due to rapid electrolyte shifts in the setting of chronic diarrhea, malnutrition, and PLE. Endocrine workup revealed features of myxedema. Echocardiography identified a large pericardial effusion, putting her at risk for cardiac tamponade. Her course was complicated by infections with multidrug-resistant organisms, coagulopathy, and inability to wean from mechanical ventilation due to encephalopathy, eventually requiring a tracheostomy and a gastrostomy.

Management required coordination among neurology, cardiology, endocrinology, nephrology, nutrition, and infectious disease teams. Aggressive nutritional support and careful electrolyte monitoring were initiated. With multidisciplinary care, her condition gradually stabilized, and she was ultimately discharged home with tracheostomy, gastrostomy, wound care for a stage 4 sacral ulcer, optimized seizure and pain control, and nutritional supplementation.
Discussion:

Protein-losing enteropathy after bariatric surgery is rare, but it can lead to profound hypoalbuminemia and systemic complications. Neurological issues like osmotic demyelination are uncommon but have been reported in similar contexts, often triggered by rapid correction of sodium or other electrolytes. Cardiac involvement, such as pericardial effusion, can occur due to severe protein-calorie malnutrition. Adult cases of kwashiorkor after bariatric surgery have also been described, underscoring the importance of adequate protein intake and careful monitoring.

Conclusion:

Patients who undergo bariatric surgery, particularly without structured follow-up, can develop life-threatening multisystem complications. Early recognition of malnutrition, PLE, neurological dysfunction, and cardiac involvement is essential. Multidisciplinary care and vigilant monitoring are crucial to prevent irreversible outcomes and improve recovery in these high-risk patients.

V-07- Azathioprine Toxicity Triggered by Allopurinol: A Case of Pancytopenia in Systemic Lupus Erythematosus with Gout

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VA Caribbean Healthcare System Program

Systemic lupus erythematosus often requires long-term immunosuppressive therapy, with azathioprine frequently prescribed to achieve better disease control. Although generally well tolerated, azathioprine metabolism is highly influenced by concomitant medications. Allopurinol, a xanthine oxidase inhibitor widely used for gout management, can dramatically potentiate azathioprine toxicity, predisposing patients to life-threatening pancytopenia. Recognition of this drug–drug interaction is critical, yet it remains underappreciated in clinical practice.

We present the case of a 65-year-old male with moderate systemic lupus erythematosus (SLE) who had contraindications to standard therapies, including hydroxychloroquine (swelling) and mycophenolate (edema and gastrointestinal intolerance). Despite being serologically negative, the patient developed new and worsening prior discoid lesions affecting the hands and face. His regimen included oral prednisone and subcutaneous belimumab; however, prednisone tapering was limited by persistent disease activity and up-titration of dose by adverse steroid effects. Given these constraints, azathioprine was initiated at a low dose to improve disease control, despite his ongoing allopurinol therapy for gout. The patient initially tolerated azathioprine without complications. Two months later, he developed dizziness, headaches, and exertional dyspnea. Laboratory testing revealed pancytopenia, prompting hospital admission for transfusion and further evaluation. Rheumatology and hematology consultations concluded that the presentation was most consistent with azathioprine toxicity potentiated by concomitant allopurinol use. Allopurinol, a xanthine oxidase inhibitor, interferes with the metabolism of azathioprine's active metabolite, 6-mercaptopurine, leading to increased cytotoxicity. The patient received two units of packed red blood cells, and both medications were discontinued. Subsequently, his white blood cell, hemoglobin, and platelet counts returned to baseline.

This case underscores the importance of recognizing the potentially life-threatening interaction between azathioprine and allopurinol. Even at low doses, concomitant use can precipitate severe pancytopenia through impaired drug metabolism. Careful review of comorbid conditions and concurrent medications is essential before initiating immunosuppressive therapy in patients with complex autoimmune disease. Early recognition and prompt intervention can prevent serious morbidity and highlight the need for heightened vigilance in managing polypharmacy in SLE and gout.

V-08- Pulmonary Isolation of Shewanella putrefaciens in a Puerto Rican Patient With a Right Middle Lobe Lung Mass

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Shewanella putrefaciens is an oxidase-positive, Gram-negative marine bacillus rarely associated with human disease. Infections are uncommon and typically involve soft tissue infections, otitis, or bacteremia, usually following direct seawater exposure or seafood ingestion. Pulmonary involvement is exceedingly uncommon and represents a significant diagnostic challenge, particularly when presenting as a solitary lung mass mimicking malignancy. A 79-year-old Puerto Rican woman with a history of hypertension, type 2 diabetes mellitus, dyslipidemia, gout, and osteoarthritis was admitted for cardiac catheterization due to a non-ST-elevation myocardial infarction. Chest CT revealed a 7.6 cm right middle lobe (RML) lesion, well-circumscribed and without surrounding consolidation or cavitation. Given her 100 pack-year smoking history and the lesion's radiographic appearance, primary bronchogenic carcinoma or lymphoma was strongly suspected. On bronchoscopy, the RML mucosa appeared friable but showed no visible endobronchial lesions, obstruction, or purulent secretions. BAL and transbronchial biopsy specimens were obtained. Histopathology and cytology were negative for malignancy. Unexpectedly, BAL cultures grew S. putrefaciens. Blood cultures were negative, and no additional respiratory pathogens were identified. The patient denied raw seafood ingestion, humidifier use, or recreational/occupational seawater exposure. She reported living near the shoreline with recurrent flooding, notably during Hurricane María, but lacked traditional risk factors. She drank municipal tap water and had no recent travel history. Laboratory testing showed mild leukocytosis (WBC 12.3 × 10^3/uL), eosinophilia (9.1%), and stable renal function. She remained afebrile, hemodynamically stable, and without respiratory distress. She was discharged with outpatient pulmonary follow-up, and levofloxacin therapy was initiated. Pulmonary infections caused by S. putrefaciens are extremely rare, with fewer than a dozen cases reported worldwide. In Puerto Rico, only one case involving Shewanella algae has been described, in a neonatal patient in 2012. This case is unique due to its mass-forming presentation, which radiographically mimicked malignancy rather than the consolidative or cavitary patterns typically reported in bacterial infections. Most reported cases occur in patients with immunosuppression, chronic lung disease, or direct marine exposure; our patient lacked these risk factors. Clinicians should maintain a broad differential diagnosis when evaluating solitary pulmonary lesions, especially in high-risk smokers and flood-prone tropical regions. Early bronchoscopy with BAL and tissue sampling should be prioritized when imaging suggests malignancy, as rare infectious etiologies can mimic neoplasms. Awareness of atypical presentations of S. putrefaciens can prevent unnecessary invasive procedures, enable accurate diagnosis, and guide appropriate antimicrobial therapy.

V-09- High Degree Atrioventricular Block After Fontan Surgery in Tricuspid Atresia: Epicardial Pacemaker as Lifesaving Therapy

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Introduction:

Tricuspid atresia is a rare congenital heart defect characterized by the absence of the tricuspid valve, which disrupts direct communication between the atrium and right ventricle. Without surgical intervention, it leads to cyanosis and heart failure early in life. The Fontan procedure, which reroutes systemic venous blood directly to the pulmonary arteries, has significantly improved survival allowing patients to reach adulthood. Nevertheless, long-term complications remain a major concern including arrhythmias, such as intra-atrial reentrant tachycardia, especially in those with earlier atriopulmonary Fontan types. Sinus node dysfunction is common, often manifesting as sinus bradycardia or junctional rhythm. Late atrioventricular conduction disturbances, including complete heart block, can also occur although these are less frequently observed.

Pacing strategies in Fontan patients depend heavily on the specific anatomy. The transvenous pacing modality may suit a classic atriopulmonary Fontan but carries significant thromboembolic risk requiring lifelong anticoagulation. Contemporary lateral tunnel and extracardiac conduit Fontan anatomies generally preclude transvenous lead placement, making epicardial pacing the standard approach. Maintaining atrioventricular synchrony through atrial or dual-chamber pacing is crucial for optimal hemodynamics, while isolated ventricular pacing can be deleterious. Hybrid and leadless pacing technologies represent promising alternatives for difficult-to-manage Fontan patients.

Case Description:

We report the case of a 28-year-old female with a medical history significant for tricuspid atresia who underwent lateral extracardiac Fontan procedure at six years of age. She presented to the emergency department after loss of consciousness, preceded by lightheadedness, visual disturbances, and difficulty focusing. Loss of consciousness was accompanied by tonic-clonic movements lasting approximately 10 seconds. She reported having experienced similar self-limited episodes for which medical attention was not sought. On arrival, the patient was found in respiratory distress with dyspnea, diaphoresis, and chest discomfort. Electrocardiogram revealed a high degree atrioventricular block with a ventricular escape rhythm at around 30 bpm. Transvenous pacing was attempted, but unsuccessful due to the patient's cardiac anatomy. The rhythm then spontaneously converted to normal sinus rhythm. Cardiac CT demonstrated a patent extracardiac Fontan pathway and preserved ventricular function. Subsequently, an epicardial pacemaker was successfully implanted. The patient was discharged in stable condition.

Discussion:

Pacing in Fontan patients requires an individualized approach that accounts for surgical anatomy, arrhythmic burden, and long-term hemodynamic needs. Epicardial pacing remains the mainstay for most patients with modern extracardiac Fontan anatomy, while novel technologies and hybrid strategies continue to expand the therapeutic landscape for those with complex pacing requirements. This case highlights the importance of tailoring pacing decisions to the patient's Fontan anatomy and rhythm profile. Long-term follow-ups remain crucial to monitor for complications such as arrhythmias, ventricular dysfunction, and thromboembolism, with the goal of optimizing functional outcomes and quality of life in this challenging population.

V-10- Genetic Insights into CYB5R Deficiency and Methemoglobinemia in the Puerto Rican Population

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Methemoglobinemia is a rare and often underdiagnosed condition caused by cytochrome B5 reductase m(CYB5R) deficiency, an autosomal recessive disorder that impairs oxygen transport and results in chronic
yanosis. Specific genetic mutations linked to CYB5R deficiency have been identified in Puerto Ricans,
diagnosis, targeted treatments, and genetic counseling are essential for improving patient outcomes.

A 30-year-old male with a history of anxiety, depression, obstructive sleep apnea, insomnia, restless leg syndrome, and asthma developed progressive dyspnea both on exertion and at rest. Despite extensive
cardiopulmonary evaluations—including a sleep study, stress test, echocardiogram, and pulmonary function tests (PFTs)—no clear cause was identified. His exercise tolerance had declined since 2021, with occasional dizziness.

During a routine pulmonary evaluation, he experienced a vasovagal syncopal episode during an arterial blood gas (ABG) draw. Emergency department (ED) evaluation revealed hypoxemia (oxygen saturation 84–86%) unresponsive to supplemental oxygen, bilateral peripheral cyanosis, and elevated hemoglobin (18.1 g/dL). CT angiography ruled out pulmonary embolism, though increased bronchovascular markings were noted. A significantly elevated methemoglobin level (25%) suggested methemoglobinemia. Given the absence of known oxidizing agent exposures, congenital methemoglobinemia was suspected.
Genetic testing confirmed a homozygous recessive CYB5R3 (c.775C>T) mutation, consistent with congenital methemoglobinemia due to cytochrome B5 reductase deficiency. This mutation had not previously been classified as pathogenic, making this the first documented case. Treatment with riboflavin and vitamin C reduced methemoglobin levels (from 25% to 11.5%) and improved oxyhemoglobin saturation (from 72.8% to 87.4%).

This case highlights the importance of considering congenital causes of hypoxemia, particularly in populations with a higher prevalence, such as Puerto Ricans. Further research and screening programs are critical for the early identification and management of CYB5R deficiency.

V-11- "When Lifesaving Therapy Turns Toxic: Pembrolizumab-Induced Necrotizing Glomerulonephritis"

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Introduction

Immune checkpoint inhibitors (ICIs) have transformed the treatment of advanced malignancies, but they can also lead to immune-related adverse events (irAEs) across many organs. Kidney involvement is rare, affecting less than 1% of patients, and usually presents as acute interstitial nephritis. Pauci-immune necrotizing glomerulonephritis (GN) is uncommon, and pulmonary-renal syndrome associated with it is even rarer. We present the case of an older woman with Mullerian carcinosarcoma who developed fulminant ANCA-associated vasculitis with diffuse alveolar hemorrhage months after completing pembrolizumab therapy.

Case Presentation

An 81-year-old woman with a history of Mullerian carcinosarcoma, status post hysterectomy and bilateral salpingo-oophorectomy, had completed pembrolizumab therapy in November 2024. Her past history included hypertension and hypercholesterolemia.
Four months later, she presented with progressive oliguria and required acute hemodialysis. During a dialysis session, she suddenly developed dyspnea and hemoptysis. On exam, she was tachypneic and hypoxemic (PaO_2 47 mmHg on room air). Chest imaging revealed diffuse bilateral alveolar infiltrates consistent with pulmonary hemorrhage. Laboratory evaluation showed hemoglobin 5.4 g/dL, creatinine 7.45 mg/dL (baseline 0.80), and elevated inflammatory markers. Serologies were positive for c-ANCA (PR3) but negative for ANA, p-ANCA, and anti-GBM antibodies.
Renal biopsy confirmed pauci-immune necrotizing GN with crescents in more than 50% of glomeruli. A diagnosis of pembrolizumab-associated ANCA vasculitis was made. Despite treatment with high-dose intravenous methylprednisolone, plasmapheresis, and cyclophosphamide, her condition deteriorated with worsening pulmonary hemorrhage. After goals-of-care discussions, treatment was withdrawn, and she died shortly thereafter.

Discussion

This case illustrates one of the most severe renal irAEs associated with PD-1 inhibition. While interstitial nephritis is the most common ICI-related nephrotoxicity, pauci-immune GN is far less frequent and carries high morbidity and mortality. The presumed mechanism is loss of self-tolerance due to PD-1 blockade, leading to aberrant T-cell activation and autoantibody formation.
The clinical presentation can be dramatic. In ICI-treated patients, the combination of acute renal failure and sudden hemoptysis should immediately raise concern for pulmonary-

renal syndrome. Diffuse alveolar hemorrhage may be rapidly fatal without recognition and intervention. Biopsy remains the gold standard for diagnosis, though it may be challenging in unstable patients.

Management requires prompt discontinuation of the ICI, high-dose corticosteroids, and additional immunosuppression (rituximab or cyclophosphamide). Plasmapheresis is considered for patients with pulmonary hemorrhage. However, outcomes remain poor in fulminant cases, highlighting the importance of early suspicion and multidisciplinary care.

Conclusion

Pembrolizumab can rarely precipitate life-threatening ANCA-associated vasculitis with necrotizing GN and pulmonary hemorrhage. For internists, awareness of this possibility is critical. In ICI-treated patients, new hemoptysis or unexplained renal failure should prompt urgent evaluation for vasculitis. Early biopsy, rapid initiation of immunosuppression, and close collaboration with oncology, nephrology, and pulmonary teams are essential, though prognosis in advanced presentations remains guarded. Reporting such cases helps build awareness of these rare but devastating irAEs.

V-12- "Silent Leak, Dramatic Consequence: Pancreaticopleural Fistula Post-ERCP"

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Introduction

Pancreaticopleural fistula (PPF) is a rare complication of pancreatic disease and is infrequently reported after endoscopic retrograde cholangiopancreatography (ERCP). It results from leakage of pancreatic fluid into the pleural space through an abnormal ductal communication, typically manifesting as large or recurrent pleural effusions. Because patients often present with dyspnea rather than abdominal pain, diagnosis is easily delayed or missed.

Case Presentation

A 45-year-old man with a history of choledocholithiasis underwent ERCP with stone extraction and was discharged home the same morning. Later that evening, he returned to the emergency department with severe abdominal pain, nausea, tachycardia, tachypnea, and left-sided back discomfort. Laboratory evaluation and clinical presentation were consistent with acute pancreatitis.

Chest radiography obtained on admission demonstrated a moderate left pleural effusion. Thoracentesis was performed, and octreotide therapy was initiated. Despite these measures, the effusion rapidly recurred and progressed, ultimately requiring chest tube placement. Pleural fluid analysis revealed an exudative effusion with markedly elevated amylase (>1000 U/L; >3× serum upper limit) and lipase (>1000 U/L), strongly suggestive of a pancreatic origin.

Subsequent contrast-enhanced CT imaging confirmed extension of pancreatic fluid from the retroperitoneum through a small left diaphragmatic defect, consistent with pancreaticopleural fistula. During hospitalization, the patient underwent surgical drainage of associated abscesses. The diaphragmatic defect later closed spontaneously under conservative management, resulting in decreased pleural drainage and radiographic resolution on follow-up.

Discussion

This case illustrates how PPF can mimic a primary pulmonary process. In otherwise healthy middle-aged patients presenting with unexplained or recurrent pleural effusions, particularly left-sided, markedly elevated pleural fluid amylase should prompt consideration of this diagnosis. Imaging with CT or MRCP can confirm the fistulous tract and delineate anatomy.

Management requires an individualized approach. Medical therapy with octreotide decreases pancreatic secretions but achieves closure in only 16–33% of cases. Endoscopic stenting of the pancreatic duct is effective when feasible. Refractory cases or those complicated by infection often require surgical intervention. Our patient's course

was notable for spontaneous closure of the diaphragmatic defect, an outcome rarely described in the literature.
Fewer than 5% of PPF cases are reported as ERCP-related complications, with most occurring in the setting of chronic pancreatitis. This underscores both the rarity of our case and the importance of maintaining diagnostic suspicion in post-procedural patients with atypical respiratory presentations.

Conclusion

Pancreaticopleural fistula is an uncommon but clinically significant cause of pleural effusion. Recognition requires a high index of suspicion, especially when pleural amylase is elevated. Not every pleural effusion originates in the lungs, and early consideration of pancreatic sources can prevent misdiagnosis, expedite gastroenterology or surgical referral, and improve outcomes. This case highlights the importance of persistence, appropriate imaging, and multidisciplinary care in managing rare complications of ERCP.

V-13- A Silent Spiral: Fatal Thrombotic Microangiopathy in a Patient with Acute Pancreatitis

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Thrombotic thrombocytopenic purpura (TTP) is a rare, life-threatening thrombotic microangiopathy characterized by the classic pentad of thrombocytopenia, microangiopathic hemolytic anemia, neurological involvement, renal dysfunction, and fever. It may be triggered or complicated by sepsis, or pancreatitis, leading to rapid multi-organ failure. The following case highlights the rapid progression and poor prognosis of suspected TTP in the setting of acute pancreatitis and septic shock.

Case Presentation:
We present the case of a 53-year-old female with a history of schizophrenia, class III obesity, arterial hypertension and history of Guillain-Barré syndrome who was brought from her nursing home with three days of hypoactivity, poor oral intake, and altered mental status. On examination, she was obtunded, non-verbal, with abdominal tenderness, bibasilar crackles on auscultation, and peripheral edema. Initial laboratory findings revealed elevated pancreatic enzymes (lipase >2000 U/L), thrombocytopenia (69k/µL), acute kidney injury (creatinine rise from 1.03 to 2.04 mg/dL), and evidence of urinary tract infection with sepsis. After reviewing imaging studies and blood workup, she was diagnosed with acute pancreatitis, complicated urinary tract infection, and community-acquired pneumonia. She was admitted to the medical ward for broad-spectrum antibiotics, intravenous hydration, and bowel rest.

Despite management, the patient developed progressive thrombocytopenia, schistocytes on peripheral blood smear, elevated LDH, D-dimer, and fibrinogen, consistent with TTP. Hematology was consulted and recommended high-dose corticosteroids and urgent plasmapheresis. Within 48 hours, she deteriorated with acute respiratory distress syndrome (P/F ratio ~100), septic shock requiring multiple vasopressors, pulmonary hemorrhage, and worsening multi-organ failure. Despite mechanical ventilation, renal support, and escalation of intensive care, the patient's condition continued to decline, culminating in cardiac arrest and death.

This case illustrates the rapid progression and diagnostic challenges of TTP overlapping with sepsis in the setting of acute pancreatitis. The combination of thrombocytopenia, neurologic dysfunction, renal failure, and pancreatitis should raise a strong suspicion for TTP, warranting urgent recognition and prompt initiation of immunosuppressive therapy and plasmapheresis, the cornerstones of management. Clinicians should maintain a high index of suspicion in critically ill patients, as delay in diagnosis and treatment is associated with high mortality.

V-14- Beyond the Skin: A Case of Disseminated Nocardiosis with Pulmonary Manifestations

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Background:
Nocardiosis is a rare opportunistic infection caused by Nocardia species, gram-positive, filamentous, aerobic bacteria commonly found in soil and organic matter. It primarily affects immunocompromised individuals and often presents with nonspecific features that mimic other infectious and inflammatory conditions, leading to diagnostic delays. Clinically, nocardiosis most commonly presents as pulmonary infection but can also involve the skin, central nervous system, or dissemination to multiple organs. Early recognition and targeted therapy are essential to improving outcomes.

Case:
This is the case of an 87-year-old male with past medical history of DM II, CKD3a, interstitial lung disease, polymyalgia rheumatica, and Crohn's disease on chronic corticosteroid therapy who presented with a painful erythematous ulcer on the left thigh draining purulent fluid after failing outpatient doxycycline therapy. Empiric therapy with ceftaroline and cefepime showed no improvement. Further history revealed recent gardening activity. After one week, the wound culture grew Nocardia spp. During hospitalization, he developed a worsening cough, tachypnea, hypoxemia, and diffuse lung crackles. Blood labs were remarkable for leukocytosis and elevated inflammatory markers. Chest CT with IV contrast revealed bilateral ground-glass opacities in the upper lobes, raising concern for disseminated pulmonary nocardiosis. Bronchoscopy was recommended; however, due to the patient's respiratory deterioration and high risk for developing complications, he was not a candidate to tolerate the procedure. Brain MRI was done to evaluate the extension of the disease, and results were unremarkable, ruling out intracranial infectious disease. The patient was started on treatment for disseminated nocardiosis involving lungs and skin in the setting of an immunocompromised patient. He received induction therapy with intravenous trimethoprim-sulfamethoxazole (TMP-SMX) plus imipenem for three weeks, resulting in resolution of the skin ulcer and improvement in respiratory status. Afterward, the patient was discharged to a rehabilitation facility to continue long-term oral suppressive therapy with TMP-SMX and minocycline for 6 to 12 months.

Discussion/Conclusion:
This case underscores several critical challenges in the diagnosis and management of nocardiosis, particularly in elderly, immunosuppressed individuals who present initially with a skin infection with subsequent respiratory decompensation involving an opportunistic organism, which is rare to be observed in the population. Nocardiosis often

presents with nonspecific dermatologic manifestations that mimic other infectious or inflammatory conditions, contributing to diagnostic delays. As described in the review “Cutaneous nocardiosis: a great imitator,” such presentations—including nodular, lymphocutaneous, or subcutaneous infections—require a high index of suspicion and timely microbiologic evaluation to avoid misdiagnosis. This case highlights the diagnostic challenge of nocardiosis. Clinicians should maintain a high index of suspicion for immunocompromised patients with refractory skin lesions. Recognition of environmental exposures, patient risk factors, and atypical clinical presentations can guide earlier diagnosis and targeted therapy, reducing morbidity and mortality.

V-15- A Single Stripe, a Startling Clue: Ischemic Colitis in a Healthy Adult.

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Ischemic colitis is typically seen in older adults with vascular risk factors, but it can also occur in younger patients due to non-occlusive mechanisms. If untreated, can lead to bowel perforation, peritonitis, persistent bleeding among others. Medications such as triptans and NSAIDs have been implicated as potential contributors. Triptans, like sumatriptan, cause vasoconstriction that may extend to the mesenteric circulation, while NSAIDs impair mucosal defense and perfusion. This can predispose otherwise healthy individuals to ischemic injury of the colon. Here we present the case of a young male with an atypical presentation of ischemic colitis.

This is the case of a 30-year-old male with a history of migraine headaches treated with frequent sumatriptan use, and regular NSAID (diclofenac) consumption, who was referred to our hospital after being discharged from another emergency department where he had been treated empirically for gastritis. He initially presented with stabbing, progressive epigastric pain lasting several days. Initial imaging and laboratory evaluation were unremarkable, and he was discharged home. Two days later, he returned with recurrent severe abdominal pain, now accompanied by bright red blood per rectum. Due to ongoing hematochezia, a colonoscopy with biopsies was performed, revealing linear ulcerations from the splenic flexure to the descending colon—a classic “single-stripe sign”—. Biopsies were obtained and histology confirmed ischemic colitis. The patient was counseled to discontinue NSAID and sumatriptan use, and his hematochezia resolved. He was advised to follow up in the gastroenterology clinic for repeat evaluation and surveillance colonoscopy.

Ischemic colitis is rare in young adults and often linked to reversible, non-occlusive causes. In this case, frequent sumatriptan use and NSAID exposure acted synergistically to cause transient colonic hypoperfusion. This highlights the importance of considering lifestyle and medication-related factors in young patients with lower GI bleeding, even when imaging is unremarkable, to ensure timely diagnosis and prevent serious life-threatening complications.

V-16- “When Thyroid Cancer Strikes the Brain: Hemorrhagic Metastases Revealing a Hidden Threat"

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Brain metastasis from primary papillary thyroid carcinoma is an extremely rare event, occurring in less than 1% of patients with this malignancy. When it does occur, it is often many years after the initial diagnosis and treatment of the primary tumor. Presentation with hemorrhagic brain lesions is even more unusual and usually signals an aggressive course. Brain involvement in this context is clinically significant, as it is associated with acute neurological deterioration, high morbidity, and overall poor prognosis. Timely recognition and multidisciplinary management may lead to meaningful neurological recovery and improved quality of life.

We present the case of a 73-year-old female with a past medical history of hypertension, diabetes mellitus, and papillary thyroid carcinoma diagnosed more than 11 years prior, status post total thyroidectomy. She presented to the hospital with acute onset of disorientation and dysarthria. Given her initial presentation, age, and comorbidities, an initial workup for cerebrovascular accident was promptly initiated.

Neuroimaging revealed a hemorrhagic mass in the left middle frontal gyrus measuring 2.0 x 3.1 x 2.5 cm, associated with extensive vasogenic edema causing significant mass effect, effacement of the precentral sulcus, and compression of the lateral precentral gyrus. A second hemorrhagic lesion was identified in the left amygdala, measuring 1.7 x 2.0 x 2.0 cm, with surrounding edema and effacement of the temporal horn. Findings were consistent with hemorrhagic metastatic lesions.

Neurosurgery was consulted and craniotomy with decompression was performed. She was subsequently transferred to the Neuro-ICU for close postoperative monitoring. High-dose corticosteroids were initiated to reduce cerebral edema, and antiseizure prophylaxis was started. Thyroid function testing revealed a markedly elevated TSH of 80uIU/ml. Intravenous levothyroxine treatment initiated with transition to oral therapy. Left frontal lobe biopsy confirmed metastatic papillary thyroid carcinoma.

Following surgery and supportive care, the patient demonstrated gradual neurological improvement. Three weeks after the diagnosis, she was discharged home in a stable condition. At discharge, she was alert and oriented to person and place, able to follow simple commands, and exhibiting minimal dysarthria. Plans were made for continued outpatient follow-up with Endocrinology, Neurosurgery, Hematology-Oncology, and Physical Medicine and Rehabilitation services.

This case underscores several important clinical considerations. First, long-term surveillance is critical in patients with papillary thyroid carcinoma, even more than a decade after initial treatment, as late metastatic manifestations can occur. Second, clinicians must maintain a high index of suspicion when patients with a history of thyroid cancer present with acute neurological symptoms, as brain metastases, though rare, can present suddenly with hemorrhage and mass effect. Finally, early recognition and

aggressive multidisciplinary management, including surgical decompression, corticosteroids, thyroid hormone replacement, and rehabilitation, can result in meaningful recovery despite the poor prognosis associated with intracranial metastases.

Hemorrhagic brain metastases from papillary thyroid carcinoma are rare but life-threatening events. Prompt diagnosis and timely intervention provide the best opportunity for preserving neurological function and improving outcomes. This case illustrates how early multidisciplinary involvement can make a critical difference, reminding clinicians that one patient diagnosed and treated on time may represent a life saved.

V-17- Not all Psychosis is Mental: A Case of Hyperactive Delirium Due To Myxedema Coma

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VA Caribbean Healthcare System Program

Myxedema coma is a rare but life-threatening manifestation of severe hypothyroidism portrayed by changes in mental status, hypothermia, and multi-system organ dysfunction. Mostly seen in elderly females, especially when mixed by factors such as infections, cold exposure, medications, or discontinuation of thyroid hormone replacement therapy. Due to its nonspecific presentation and rarity, early diagnosis and prompt management are critical to reducing the associated high mortality rates. We present the case of an elderly man with waxing and waning disorientation episodes with acute delirium presenting early signs of Myxedema coma.

Case of 79-year-old male patient with pertinent medical history of Grave's Disease status post iodine ablation resulting in hypothyroidism treated with levothyroxine. The patient arrived to the emergency department due to new onset disorientation and abdominal distention for which was admitted to ward with volume depletion and associated hypovolemic hyponatremia of unclear chronicity. Patient initially arrived oriented in all spheres denying any acute complaint. During his second day at ward, the patient's condition rapidly progressed when during the night he started to have high oxygen demand and became severely agitated. Patient was uncooperative with physical examinations and with nursing staff for which was treated with sedatives after multiple reorientation attempts. He did not respond to treatment and oxygen demand continued to progress for which an aspiration event was suspected. Patient was transferred to our Intensive Care Unit with Non-invasive Mechanical Ventilation. TSH levels collected on admission were found markedly elevated at 85.260 uIU/mL and sodium levels at 121 mEq/L. Due to profound hypothermia, bradycardia, hypotension, electrolyte disturbances, hypoventilation, and altered mental status clinical diagnosis of myxedema coma was reached. Patient placed on low dose norepinephrine, IV hydrocortisone dose followed by IV levothyroxine. Once levothyroxine treatment was started, gradual clinical improvement was observed over several days, including reorientation, highlighting the efficacy of comprehensive and timely intervention during this diagnosis.
This case underscores the importance of maintaining high clinical suspicion for myxedema coma in patients with hypothyroidism and presenting with altered mental status. Some atypical features such as rapid progression of delirium with agitation rather than lethargy and concurrent hypovolemic hyponatremia can obscure diagnosis. Due to delirium, especially with agitation, using sedatives may worsen respiratory depression and precipitate further decompensation in hypothyroid patients. It should be suspected in patients who have unclear compliance to hypothyroidism treatment. Furthermore, it is usually associated with lethargy and hypoactivity, but there have been cases of psychosis. Early recognition and multidisciplinary management remain pivotal in improving outcomes for this critical condition.

V-18- Dyspnea in the postpartum period: a rare cause of bilateral empyema due to S. aureus

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A parapneumonic effusion is a pleural effusion formed adjacent to a pneumonia inside the pleural space, when infected by microorganisms it is known as empyema. Parapneumonic effusions within the postpartum period are uncommon, and even more rare is the development of empyema in a young healthy patient. Parapneumonic effusion incidence is around 32,000 US cases that occur yearly, with 20-30% needing surgery. However, no actual studies have quantified its incidence during postpartum. Roughly 5-7% of postpartum women develop infections within six weeks, but it's rarely empyema. The patient populations most at risk for developing empyema in the postpartum period due to S. aureus infection include women with the following characteristics: Pre-pregnancy diabetes, S. aureus colonization, postpartum women with skin or soft tissue infections, women with difficulty breastfeeding, mothers employed outside the home, and hospital transmission and community-acquired MRSA.

This is a case of a 31-year-old female who presented with dyspnea exacerbated when supine, back pain, fever, and bilateral leg edema nine days after a complicated C-section. Patient reported three prior "flu-like" episodes during late pregnancy that were poorly managed before delivery. Physical examination showed bilateral basilar crackles and whispered pectoriloquy.

Labs showed leukocytosis, anemia and elevated inflammatory markers. Serial Chest CT showed moderate-large right and moderate-sized left pleural effusions with associated subsegmental atelectasis of bilateral lower lobes. A diagnosis of bilateral loculated Empyema was made. Pleural fluid culture was positive for Methicillin-Susceptible Staphylococcus Aureus (MSSA). Chest U/S showed significant effusion and thoracentesis drained 500ml on the right side. At day 16th a right-sided chest tube was placed with intrapleural tPA and DNase that drained 950 mL, achieving radiographic resolution of the right effusion. She completed 24 days of IV cefazolin followed by 14 days of cefadroxil outpatient.

This case highlights that postpartum MSSA empyema is rare and requires early recognition to avoid complications. Intrapleural fibrinolysis provided effective, non-surgical resolution, avoiding surgical decortication and complications. It is essential to recognize, diagnose and treat this condition early. This case presentation will enable physicians to consider it within the differential diagnosis, thereby facilitating timely and accurate clinical decision-making, minimizing the need for invasive interventions, and ultimately contributing to a favorable patient outcome.

V-19- A Race Against Time: Spontaneous Tumor Lysis Syndrome as the First Manifestation of Ovarian Adenocarcinoma

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Mayaguez Medical Center Program

Tumor lysis syndrome (TLS) is an oncologic emergency most commonly seen after initiation of cytotoxic therapy in hematologic malignancies. Spontaneous TLS, occurring in the absence of treatment, is rare and even less frequently reported in solid tumors. Early recognition is critical due to its high mortality.

We report a diagnostically challenging case of spontaneous TLS in a 53-year-old woman with no prior medical history presenting to the emergency department with progressive abdominal distension, shortness of breath, and decreased urinary output. Physical examination revealed a tense, distended abdomen with preserved bowel sounds. Laboratory testing demonstrated acute kidney injury with a creatinine level of 4.09 mg/dL, along with hyperkalemia, hyperphosphatemia, hyperuricemia, and hypocalcemia. Imaging identified massive ascites and a large pelvic mass measuring 16.2 x 15.0 x 15.0 cm, but no hydronephrosis. Renal ultrasound showed normal kidney size and architecture. Given the severity of metabolic disturbances and absence of obstructive uropathy, spontaneous TLS was considered, supported by a high Cairo-Bishop score of 3. The patient received prompt treatment with aggressive intravenous hydration and rasburicase coordinated with hematology-oncology and nephrology teams. Despite initial concerns regarding potential dialysis, her condition improved significantly without the need for renal replacement therapy. Over the following five days, renal function normalized. Pathologic evaluation of the pelvic mass later confirmed ovarian adenocarcinoma.

This case illustrates spontaneous TLS as the initial presentation of an undiagnosed solid tumor. While TLS is well recognized in hematologic malignancies, its occurrence in solid tumors prior to therapy is rare. Clinicians should maintain a high index of suspicion in patients with bulky tumors, acute kidney injury, and metabolic derangements, as timely recognition and intervention can be lifesaving. Awareness of this possibility ensures prompt initiation of supportive measures and uric acid–lowering therapy, potentially preventing irreversible organ damage. To our knowledge, spontaneous TLS as the first manifestation of ovarian adenocarcinoma is exceedingly rare.

Spontaneous TLS can be the first manifestation of an occult malignancy. Early identification and management are crucial to optimize patient outcomes.

V-20- “It’s just a pancreatitis”……..Think again!!! Severe Acute Necrotizing Pancreatitis Complicated by Disconnected Duct Syndrome

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Pancreatitis is an inflammatory process involving the pancreas and extrapancreatic organs. Biliary disease is the most common cause of acute pancreatitis but alcohol, hypertriglyceridemia, medications, and idiopathic etiologies also play important roles. Premature activation of digestive enzymes and release of cytokines driven by autodigestion of pancreas and inflammation may involve surrounding organs. Disconnected duct syndrome is a complication in which necrosis causes disruption between viable segments of the main pancreatic duct and gastrointestinal tract.

This is a 47 year-old male with a past medical history of hypertension who was admitted with severe epigastric pain radiating to the back, associated with over 20 episodes of vomiting. He denied alcohol, smoking, or illicit drug use. No prior history of gallstones, hypertriglyceridemia, or abdominal surgery was reported.

On admission, he was afebrile but tachycardic. Laboratory evaluation revealed marked leukocytosis, elevated amylase and lipase with preserved renal and hepatic function. A CT of the abdomen demonstrated severe acute necrotizing pancreatitis with peripancreatic fat stranding, small ascites, and multiple acute peripancreatic fluid collections. No gallstones were identified.

During hospitalization, the patient developed coffee ground emesis concerning for an upper GI bleeding. Endoscopy was performed, and revealed a hiatal hernia, gastritis, and duodenitis, but no active bleeding source was identified. He was managed with IV hydration, electrolyte repletion, high-dose proton pump inhibitors, empiric antibiotics, and bowel rest.

Despite initial improvement, his course was complicated by enlarging peripancreatic collections. Perihepatic collection was managed with drainage and stent placement. Imaging confirmed disconnected pancreatic duct syndrome.

This case highlights a rare progression of an acute necrotizing pancreatitis in a patient without typical risk factors. The initial complication of suspected upper GI bleeding was clarified by endoscopy as reactive mucosal changes from vomiting and peripancreatic inflammation. The subsequent evolution into disconnected duct syndrome illustrates a challenging sequelae of a late complication of necrotizing pancreatitis.

V-21- A Vein of Malignancy: Multivisceral Resection for IVC Leiomyosarcoma

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Abstract

Leiomyosarcoma of the inferior vena cava is a malignant smooth-muscle tumor with poor prognosis and nonspecific clinical presentation. Symptoms range from asymptomatic incidental findings to vague abdominal pain. We report a 51-year-old woman with progressive right-sided abdominal pain and weight loss, found to have an incidental retroperitoneal mass ultimately diagnosed as IVC leiomyosarcoma. This report highlights the diagnostic challenges and surgical strategies required for curative treatment.

Introduction

Leiomyosarcomas constitute 10%–20% of soft-tissue sarcomas, and retroperitoneal origin is rare. IVC involvement is particularly aggressive and usually advanced at diagnosis because of slow growth and nonspecific symptoms. Imaging cannot reliably distinguish these tumors from other retroperitoneal masses, making histopathology with immunohistochemistry the diagnostic gold standard. Complete surgical excision with negative margins is the only proven curative therapy, but resection is technically demanding because tumors frequently involve adjacent viscera and major vessels.

Case Presentation

A 51-year-old woman with a strong family history of cancer presented with intractable right-upper and right-lower quadrant abdominal pain and unintentional weight loss. Examination revealed focal tenderness without organomegaly or edema. Contrast CT showed a hypervascular retroperitoneal mass near the porta hepatis with hemoperitoneum. Tumor markers (LDH, AFP, CEA, CA19-9, CA125) were normal. Endoscopic ultrasound–guided fine-needle aspiration confirmed sarcoma. Abdominal MRI demonstrated a slow-growing, vascular portocaval lesion present since 2019, suspicious for IVC leiomyosarcoma.

The patient underwent exploratory laparotomy with en bloc IVC tumor resection, pylorus-sparing pancreatoduodenectomy, cholecystectomy, and portal/aortocaval lymphadenectomy. Vascular continuity was restored using a prosthetic graft. Gross and microscopic examination confirmed high-grade IVC leiomyosarcoma infiltrating duodenum and pancreas. Resection margins were negative and lymph nodes uninvolved. Immunohistochemistry staining positive for Caldesmon.

Discussion

Primary IVC leiomyosarcoma represents <0.5% of all soft-tissue sarcomas and typically arises between the renal and hepatic veins. Common manifestations are abdominal pain, a palpable mass, and occasionally lower-limb edema, though many patients remain asymptomatic. Diagnosis is difficult because cross-sectional imaging and endoscopic ultrasound with FNA cannot always differentiate benign from malignant vascular lesions.

MRI best defines tumor extent and vascular involvement, which are crucial for operative planning.
These tumors respond poorly to chemotherapy or radiotherapy; radical en bloc resection with negative margins offers the only chance of cure and yields 5-year survival rates of 30%–50%. Surgical challenges include proximity to vital organs and vessels, often necessitating multivisceral resection and vascular reconstruction. The benefit of adjuvant therapy remains unclear. Our case is notable for slow growth over several years, absence of venous congestion, and successful complete resection with prosthetic IVC graft.

Conclusion

IVC leiomyosarcoma is a rare retroperitoneal malignancy that may present with subtle or atypical symptoms. Diagnosis requires high suspicion and multimodal imaging, confirmed by histopathology. Aggressive surgical resection with vascular reconstruction remains the cornerstone of curative treatment. Early recognition and multidisciplinary coordination are essential to optimize outcomes.
References

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V-22- Checkpoint Chaos: A Nivolumab-Associated Immune Event

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Nivolumab, a human monoclonal antibody used as an immune checkpoint inhibitor (ICI) targeting programmed cell death receptor 1 (PD-1), has become a cornerstone in the treatment of various malignancies. However, its use can lead to immune-related adverse events (irAEs), including rare but potentially severe immune-mediated hepatitis. We present the case of a 85-year-old male with obstructive esophageal squamous cell carcinoma stage III on palliative immunotherapy with nivolumab, who developed immune mediated adverse reaction following nivolumab treatment. Patient presented to ER due to a fall from his bed in which he hit his head. During routine laboratories, patient presented with marked elevated liver enzymes with hepatocellular injury (AST 2535 U/L; ALT 2449 U/L; Alkaline phosphatase 215 U/L, Bili 0.34 mg/dL). A comprehensive workup, including autoimmune and infectious panels, was negative, effectively ruling out autoimmune hepatitis and viral etiologies. Imaging studies revealed gallbladder wall thickening and trace pericholecystic fluid on ultrasound, and mild hepatomegaly on a non-contrast CT scan. However, gastrointestinal consultation deemed acalculous cholecystitis unlikely as the source of hepatic injury. As patient with hepatocellular injury with enzymes >20 times upper limit of normal, in addition to recent administration of nivolumab medication, scenario raised concern for possibility of PDL-1 induced hepatitis. As this condition poses an increased risk for life threatening adverse effects, management included discontinuation of immunotherapy and the prompt initiation of high-dose corticosteroids. After treatment initiation, patient with significant clinical improvement and normalization of liver function tests. This case underscores the importance of early recognition and management of immune-mediated hepatitis, a rare but potentially life-threatening complication of ICI therapy. As the use of ICIs like nivolumab continues to expand across various malignancies, awareness of irAEs is critical. The incidence of immune-mediated hepatitis with PD-1 inhibitors like nivolumab is estimated to be between 1% and 5%. Hepatotoxicity may present with non-specific symptoms or be discovered incidentally through routine labs, as in this patient. Timely diagnosis is often challenging, requiring exclusion of infectious, autoimmune, and biliary causes. Prompt discontinuation of the offending agent and initiation of corticosteroid therapy are essential to prevent further hepatic injury. This case emphasizes the need for regular monitoring, multidisciplinary evaluation, and a high index of suspicion to ensure patient safety during immunotherapy.

V-23- Beyond the Gut: Case of Disseminated Histoplasmosis Associated with TNF-Alpha Inhibitor Therapy in the Southern Area of Puerto Rico

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Histoplasmosis is an opportunistic fungal infection that can present as a self-limited or asymptomatic infection in immunocompetent individuals. However, it can manifest as a severe or disseminated infection in immunosuppressed patients. In patients with Crohn's disease, particularly those receiving immunosuppressive therapy such as corticosteroids, TNF inhibitors, or other biologics, the risk of infections, including fungal infections like histoplasmosis, is increased. This report discusses the case of a young male on active treatment with Adalimumab (a TNF-alpha inhibitor) for Crohn's disease, who was eventually diagnosed with disseminated histoplasmosis.

A 31-year-old male with a past medical history of Crohn’s disease, who was undergoing workup for suspected lymphoma, presented to the emergency room with abdominal pain, weight loss, general malaise, and decreased hemoglobin levels. An abdominal CT scan revealed splenomegaly with emphysematous changes. The patient was admitted for inpatient management and underwent a spleen biopsy, which revealed acute inflammation and the presence of fungal organisms. There was no history of recent travel or exposure to caves, but there was reported occupational exposure to gardening. The patient's extensive inpatient course was notable for significant clinical deterioration, including altered mental status. Further workup confirmed disseminated histoplasmosis. A course of anti-fungal treatment led to an improvement in symptoms. The patient was eventually discharged with oral anti-fungal therapy prescribed for a minimum of 12 months, along with close outpatient follow-up.

The clinical presentation of histoplasmosis in Crohn's disease varies depending on the extent of the infection. Disseminated histoplasmosis is especially concerning for immunocompromised individuals. This case highlights the importance of symptom recognition in patients on immunosuppressive therapy and the complications that can occur. Larger-scale studies could help in prompt screening, diagnosis, and treatment, especially in regions like Puerto Rico, where data on disseminated histoplasmosis remains limited.

V-24- When 'Wellness' Turns Harmful: Herbalife®-Associated Drug-Induced Autoimmune-Like Hepatitis in a Patient Pursuing a Healthier Lifestyle

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Introduction:

Drug-induced liver injury (DILI) remains a leading cause of acute liver dysfunction and is increasingly attributed to herbal and dietary supplements (HDS). A distinct phenotype, drug-induced autoimmune-like hepatitis (DI-ALH), presents with autoantibody positivity, often mimicking autoimmune hepatitis (AIH). Unlike AIH, DI-ALH typically resolves with withdrawal of the offending agent and rarely requires long-term immunosuppression. Distinguishing these entities is critical to avoid unnecessary treatment and improve outcomes.

Case Description:

A 45-year-old man with dyslipidemia, anxiety, and depression presented asymptomatically for routine follow-up. Having recently quit smoking, he embraced regular exercise and began Herbalife® supplements to support his "healthier" lifestyle. His prior medications, atorvastatin and ezetimibe, had been discontinued months earlier. Laboratory results revealed new findings of elevated transaminases. He denied alcohol or recent infections, and physical examination was unremarkable. Viral hepatitis, HIV, and antimitochondrial antibody testing were negative. Autoimmune testing demonstrated a positive antinuclear antibody (ANA) with a speckled pattern and elevated ferritin. Careful history-taking identified ongoing Herbalife® use as the most likely etiology. Herbalife® was discontinued, statins remained withheld, and follow-up testing six weeks later showed complete biochemical recovery.

Discussion:

This case illustrates the clinical features of DI-ALH: a clear temporal relationship to Herbalife® use, autoimmune serologies in the absence of other causes, and resolution after discontinuation of the supplement. Misclassification as idiopathic AIH could have led to unnecessary corticosteroid therapy. The case also highlights how patients motivated to improve their health may unknowingly place themselves at risk when using over-the-counter supplements marketed as safe. Careful history-taking was critical for uncovering supplement use in an otherwise asymptomatic patient, and early recognition likely prevented progression to more severe liver injury.

Conclusion:

Herbalife® products can trigger DI-ALH even in individuals pursuing a healthier lifestyle. Early recognition through thoughtful history-taking and patient education about the risks of "natural" supplements is essential to prevent misdiagnosis, avoid unnecessary immunosuppression, and support recovery.

V-25- When the Treatment Feels Like the Disease: A Unique Case of Cefepime-Induced Encephalopathy

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Introduction:

Cefepime-induced encephalopathy (CIE) typically develops within four to five days of starting the antibiotic and is often reversible within three days of discontinuation. The risk is increased in patients with renal impairment. During presentation, evaluation for other causes of encephalopathy may delay recognition of the insult and timely management, including discontinuation of cefepime. Herein, we present an atypical case of a patient with disorientation, confusion, and aphasia, ultimately leading to the diagnosis of CIE.

Case presentation:

A 39-year-old female with systemic lupus erythematosus (SLE) complicated by lupus nephritis presented with two days of disorientation and confusion. On evaluation, had resolution of altered mental status, but persistent diarrhea. Physical examination revealed an oriented, conversant patient in no acute distress and mild abdominal distention. Laboratory studies revealed acute kidney injury, elevated anti-double-stranded DNA (1:160), and decreased complement 3 levels. The abdominopelvic CT scan was unremarkable. The patient was admitted with a working diagnosis of suspected lupus exacerbation, possibly triggered by an infectious gastrointestinal process, and was started on high-dose glucocorticoids and intravenous broad-spectrum antibiotics, including cefepime. During the hospitalization, she subsequently developed an acute onset of altered mental status, characterized by disorientation, confusion, and aphasia. The differential diagnosis included neuropsychiatric lupus, steroid-induced altered mental status, and drug-related encephalopathy. Urgent brain MRI was unremarkable, effectively ruling out neuropsychiatric SLE. Given the temporal association with cefepime initiation, cefepime-induced encephalopathy was suspected. The antibiotic was promptly discontinued, and remarkably, within 24 hours, the patient's mental status returned to baseline; she was fully oriented and verbal. For the remainder of her hospitalization, her neurological examination remained completely normal, highlighting both the reversibility of CIE and the importance of considering medication-induced causes of encephalopathy in complex patients with SLE.

Conclusion:

This case underscores the importance of early recognition of cefepime-induced encephalopathy, even in relatively young patients. The presence of comorbid conditions such as active lupus nephritis, chronic diarrhea, and immunosuppressive therapy initially suggested alternative explanations for her altered mental status, delaying consideration of drug-related toxicity. Maintaining a high index of suspicion and promptly discontinuing cefepime are essential to avoid unnecessary interventions and ensure timely recovery.

V-26- Why Can't I See?: The Diagnostic Dilemma of Post-Infectious Optic Neuritis in the Setting of Overlapping Features

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Introduction:
Post-infectious optic neuritis (ON) is a rare, immune-mediated optic nerve inflammation that presents as acute vision loss shortly after a viral infection. It usually presents with bilateral involvement, marked optic disc edema, and severe nadir acuity, but without direct optic nerve invasion and with steroid responsiveness. The differential diagnoses include Multiple Sclerosis-associated ON, neuromyelitis optica, infectious optic neuropathy, papilledema due to increased intracranial pressure, giant cell arteritis (GCA), and many more. Post-infectious ON is essentially a diagnosis of exclusion, which complicates the diagnosis in patients with multiple comorbidities and an elaborate clinical presentation that increases risk for other etiologies of vision loss.

Case Presentation:
A 58 year-old female with a past medical history of DMT2 with diabetic retinopathy and bilateral cataracts presented to our institution with a four-day history of bilateral blurry vision that rapidly progressed to complete right-eye vision loss, and an associated right sided temporal headache. The patient had a recent upper respiratory illness (URI) that resolved one week prior to ocular symptom onset. No neurological deficits were identified. Ophthalmologic examination was remarkable for severely decreased bilateral vision but worse in the right eye, normal intraocular pressure, limited extraocular movements, optic nerve swelling of the right eye, and chronic diabetic retinopathy changes bilaterally. Laboratory studies revealed mildly elevated inflammatory markers. Lumbar puncture (LP) with evidence of pleocytosis. Neuroimaging with no intracranial pathologies or white matter lesions, but with findings of right sided orbital perineuritis. Initially, GCA was high in the differential diagnosis for which the patient underwent a steroid pulse with significant left sided vision recovery, but no right sided improvement. Consequently, a right sided optic nerve sheath biopsy was performed that showed fibroconnective tissue. Additional autoimmune workup was remarkable for a positive Anti-MOG antibody. The patient was diagnosed with post-viral ON, transitioned to oral steroid therapy, and discharged home. During follow up, the patient presented with significant improvement in bilateral vision with almost complete resolution of her symptoms.

Discussion:
This case highlights the diagnostic dilemma of post-infectious ON in a patient with multiple ophthalmologic comorbidities and overlapping features of alternative diagnoses. The acute unilateral onset of profound vision loss with an associated temporal headache and elevated inflammatory markers, raised concern for GCA. Additionally, the patient’s history of diabetic retinopathy and cataracts complicated the diagnosis. However, the history of a recent URI, absence of demyelinating lesions on imaging, lack of infectious pathogens on LP, positive anti-MOG antibody, and steroid responsiveness supported our final diagnosis. In this case, a prompt diagnosis along with early steroid treatment likely preserved the patient's vision with near-complete recovery. This emphasizes the importance of maintaining a broad differential diagnosis, including post-infectious ON and other rare etiologies, as timely intervention can prevent permanent disability.

V-27- The Stroke That Runs in the Family: Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy

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Introduction: Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL) is a rare, hereditary small-vessel disease caused by mutations in the NOTCH3 gene on chromosome 19. It typically presents with recurrent ischemic strokes, migraine with aura, and progressive cognitive decline. Characteristic neuroimaging findings include white matter hyperintensities and lacunar infarcts localized in the anterior temporal lobes and external capsules. Currently, no curative therapy exists. We present a case of a middle-aged woman with characteristic MRI findings and high clinical suspicion of CADASIL.

Case Report: A 78-year-old female with hypertension, hypercholesterolemia, chronic kidney disease, insulin dependent diabetes mellitus, coronary artery disease, and a left frontal meningioma was admitted to the internal medicine ward under the clinical impression of ischemic cerebral vascular accident (CVA) associated with involuntary movements and loss of consciousness. Neurological examination was remarkable for global aphasia. Laboratory findings revealed leukocytosis, stable hemoglobin and platelets, without major coagulopathies. Chemistry panel demonstrated acute kidney injury, hypernatremia, and metabolic acidosis. Neuroimaging was remarkable for anterior frontal meningioma measuring 1.4 x 1.8 x 2.1 cm with associated vasogenic edema and acute ischemic infarct of left thalamus and posterior limb of internal capsule, extensive chronic lacunar infarcts and scattered microhemorrhages. Follow up imaging with additional findings of lacunar strokes, bilateral, diffuse white matter hyperintensities, and cerebral microbleeds. These radiological features, in addition to a family history of a daughter with confirmed NOTCH3 mutation, made CADASIL our main differential diagnosis. In view of recent acute ischemic infarct, despite being on clopidogrel 75mg daily she was switched to aspirin 81mg daily for secondary stroke prevention and placed on a high dose statin. Levetiracetam 500 mg twice daily was initiated in view of structural etiology of seizures. Neurosurgery reccomended steroid therapy for vasogenic edema and no surgical management. Echocardiogram with no evidence of thrombus or valvulopathies. EEG was suggestive of encephalopathy. MRA without findings of aneurysm or arteriovenous malformations. The patient was discharged with physical therapy and scheduled for outpatient follow-up in the vascular neurology clinics for genetic testing.

Conclusion: CADASIL is the most common hereditary small-vessel disease of the brain. It is often silent in its early stages, progressive in its course, and frequently mistaken for other conditions. In this case, the family history provided a critical clue: the patient's daughter, once a vibrant woman, had suffered multiple strokes and was now living with dementia. In adults presenting recurrent ischemic events—particularly with such family history—CADASIL should be considered. MRI findings frequently suggest the diagnosis before genetic testing confirms it. Early recognition is essential, not for curative therapy, but for appropriate counseling, preventive strategies, and long-term management.

V-28- An unexpected uropathogen: Salmonella UTI with systemic spread in a case of uncontrolled HIV patient: A rare case report

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We do not have a residency program on record for you.

Urinary tract infections are common among immunocompromised patients, but Salmonella species are rarely implicated as uropathogens. They account for less than 0.07% of all urinary tract infections in the general population and may lead to severe systemic infections, including bacteremia. We present a case of a 59-year-old man with a past medical history of untreated AIDS since 2011 and anal cancer who was diagnosed with COVID-19 and bacteremia secondary to Salmonella UTI. The patient was brought to the emergency room with multiple episodes of watery diarrhea, hypotension, tachycardia, generalized malaise, weakness, chills, nausea and anorexia for a month. His social history is remarkable for poor hygiene and living alone without access to electricity or potable water. A complete lab workup was obtained to further investigate the patient's symptoms including urinalysis and urine cultures, which identified Salmonella species as a causative agent. The infection progressed to bacteremia, as confirmed by blood cultures. Initial laboratories showed pancytopenia with an ANC of 1,404 cells/µL; with further evaluation, the diagnosis of HIV was reached. In accordance with established clinical guidelines, screening for opportunistic infections was performed prior to the initiation of antiretroviral therapy and trimethoprim-sulfamethoxazole. This case report highlights a rare but serious presentation of UTI due to Salmonella in an immunocompromised host and underlying the importance of considering a broader spectrum of organisms in high-risk patients and recognizing the potential for systemic spread.

V-29- Amyloidosis Without Plasma Cell Dyscrasia? A Rare Case with Concurrent MDS

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Introduction:

Clinically, Myelodysplastic syndrome (MDS) and amyloidosis are well-known as distinct entities and their coexistence is extremely rare. MDS is characterized by ineffective hematopoiesis, morphologic dysplasia in myeloid cell lineages, and a risk of progression to leukemia. On other hand, amyloidosis is defined by extracellular deposition of misfolded, insoluble protein fibrils, being the most common forms: immunoglobulin light chain (AL) and transthyretin (ATTR) amyloidosis. AL-amyloidosis results from a clonal plasma cell disorder producing amyloidogenic light chains, depositing in any tissue and organs, leading to progressive organ dysfunction. The clinical manifestations and prognosis depend on the amyloid type and the organs affected. This case report presents a rare manifestation of isolated renal amyloidosis with evidence of MDS on bone marrow biopsy (BMB), and aims to enhance connections to continue developing a better clinical decision taking and management since its therapeutic challenges.

Case Presentation:

A 72-year-old female underwent a renal biopsy after large albuminuria, severely increased on routine examination. Pathology reports detection of AL-amyloidosis deposits, monoclonal lambda light chain related. Cardiology evaluation with Gated Dobutamine Myocardial Perfusion Scan (SPECT) showed evidence of mild global left ventricular dysfunction while pyrophosphate heart scan is without evidence of ATTR-positive cardiac amyloidosis, raise further interrogation. During further workup all hematologic cell lines were within normal limits; kappa/lambda light chain free ratio in random urine was normal; Urine protein electrophoresis (UPEP) didn't detect monoclonal immunoglobulin. Nonetheless serum protein electrophoresis (SPEP) is noticeable for marked proteins suggestive of possible monoclonal protein, though immunofixation failed to identify immunoglobulin.

Given these finding, the patient was referred to hematology-oncology for further evaluation with BMB, to find plasma cell dyscrasia. Interesting finding of MDS with increased blast and amyloidosis on bone marrow were found and no abnormal plasma cell proliferation was identified. Next- generation sequencing (NGS) failed to identify a specific entity in her condition.

Discussion:

Amyloidosis and MDS are two different entities with a different pathological mechanism and in this case have been found in the same patient. BMB did not find any abnormal plasma cells but she was diagnosed with MDS. Management is challenging, as therapeutic options may be limited by bone marrow dysfunction, treatment toxicity and if plasma cell dyscrasia is not confirm then treatment will not be effective in reducing amyloid deposits. We will continue close observation for plasma cell dyscrasia and a bone marrow aspiration and biopsy repetition will be considered.

Conclusion:

The co-existence of MDS and AL-amyloidosis is extremely rare and have important diagnosis and therapeutic challenges. Amyloidosis is most commonly associated with plasma cell dyscrasias such as multiple myeloma or monoclonal gammopathy of undetermined significance, not with MDS. In this case, renal amyloidosis was confirmed while on bone marrow biopsy plasma cell dyscrasia evidence was absent. This atypical overlap complicates management, as therapies are directed differently. Treatment for plasma cell disorders may not reduce excessive amyloid deposit, while MDS treatment options are limited due to marrow dysfunction and potential toxicity. Greater awareness of such rare associations may improve diagnostic accuracy and guide future therapeutic strategies.

V-30- Hemopericardium and Cardiac Tamponade in a Young Adult: A Cautionary Tale of Warfarin and Cannabis Interaction

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Introduction: Warfarin remains the mainstay of anticoagulation in patients with mechanical heart valves, but its narrow therapeutic window and numerous drug interactions pose significant risks. The increasing prevalence of cannabis use adds a new dimension to anticoagulation management, with potential for serious adverse events.

Case Description: A 36-year-old male with a history of type 2 diabetes mellitus and mechanical mitral valve replacement one month prior who is taking warfarin 5 mg daily, presented with acute onset shortness of breath, fatigue, and chest pain of one day duration. He reported daily marijuana use but denied other substance use or recent medication changes. On arrival he was hypoactive, tachycardic and hypotensive, with jugular venous distension and distant heart sounds. Bedside echocardiogram revealed a large pericardial effusion with tamponade. Laboratory studies showed a markedly elevated INR of 9.0. Emergent pericardiocentesis was performed, draining 2 liters of dark blood. The patient subsequently required a surgical pericardial window for recurrent effusion. Further review implicated a pharmacokinetic interaction between warfarin and cannabis, likely via CYP2C9 inhibition, as the cause of supratherapeutic anticoagulation and hemopericardium.

Discussion: This case highlights the potential for significant drug interactions between warfarin and cannabis, resulting in life-threatening bleeding complications. As cannabis use becomes more widespread, clinicians must be vigilant in obtaining thorough substance use histories and monitoring INR closely in patients on warfarin. This case underscores the importance of patient education regarding the risks of combining warfarin with substances that may alter its metabolism. Awareness of such interactions is critical to prevent adverse outcomes in anticoagulated patients.

V-31- Metformin Mayhem: A Rare case of it's Toxicity and it's Unlikely Survival

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Metformin-associated lactic acidosis (MALA) is a serious toxicity that can occur in patients either from an acute overdose of metformin or from underlying conditions that impair metformin elimination, such as kidney injury or tissue hypoxia. Risk factors include kidney dysfunction, liver disease, alcohol use, heart failure, and a history of lactic acidosis. Mortality rates can be as high as 36% underscoring the severity of this condition.

In this case we present a 72-year-old female with a medical history of type 2 diabetes mellitus, dyslipidemia, hypothyroidism, psoriasis, and coronary artery disease who presented to the emergency department with epigastric pain, multiple episodes of vomiting, loose stools, and shortness of breath. She reported recent use of moderate amounts of NSAIDs for left leg pain. On physical examination, the patient was alert and oriented but appeared acutely ill, with dry mucous membranes, muscle weakness, and overall distress.
Initial laboratory results revealed leukocytosis, high anion gap metabolic acidosis with a pH of 7.01, elevated lactate levels of up to 14.4, acute renal failure, urine analysis and further urine culture confirming a urinary tract infection. Given her known use of metformin and significant renal dysfunction, the patient was diagnosed with acute renal failure and high anion gap metabolic acidosis secondary to MALA and urosepsis.

Due to the high risk of cardiac arrest, the patient underwent rapid sequence intubation for airway protection, received aggressive IV fluid resuscitation, IV sodium bicarbonate replacement, and was started on IV antibiotics. Consultations with Nephrology and General Surgery services were made for urgent hemodialysis catheter placement, and an emergent hemodialysis session was performed.
The patient had a prolonged hospitalization, complicated by upper gastrointestinal bleeding and heparin-induced thrombocytopenia, both of which were successfully managed. She was eventually liberated from mechanical ventilation, transferred to the general ward, and discharged home to continue outpatient hemodialysis.

This case illustrates the importance of a thorough medication history and the identification of metformin use when evaluating high anion gap metabolic acidosis. It also emphasizes how timely intervention, including hemodialysis and management of underlying conditions, can substantially improve survival outcomes.

V-32- Persistent Cushing Disease Successfully Managed with Mifepristone After Failed Surgery

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Cushing disease (CD), caused by an ACTH-secreting pituitary adenoma, is typically managed with transsphenoidal surgery (TSS). Surgical failure and disease persistence remain common challenges, necessitating alternative therapies. We present a case of persistent CD after TSS, successfully managed with mifepristone, a glucocorticoid receptor antagonist, with substantial metabolic improvement and careful monitoring of treatment-related complications.

A 56-year-old woman with obesity, type 2 diabetes mellitus (T2DM), hypertension, and hyperlipidemia presented for worsening hyperglycemia (HbA1c 8.6%), osteopenia and Cushingoid features including central adiposity, dorsocervical fat pad, and violaceous striae. Initial work-up demonstrated inadequate cortisol suppression after a 1-mg overnight dexamethasone suppression test (cortisol 2.15 µg/dL). Twenty-four–hour urinary free cortisol was elevated (64.1 µg/24h; ref <50), with detectable plasma ACTH (20 pg/mL), consistent with ACTH-dependent Cushing syndrome. Pituitary MRI showed a partial empty sella without discrete adenoma. Bilateral inferior petrosal sinus sampling confirmed a central ACTH source unconfirmatory lateralization results. The patient underwent TSS without postoperative complications. Postoperatively, serum cortisol remained >18 µg/dL without adrenal insufficiency, consistent with persistent disease. Therapeutic options including re-operation, radiation, and medical therapy were discussed. Given her poorly controlled T2DM and metabolic complications, mifepristone was initiated. On therapy, the patient experienced marked metabolic benefit. HbA1c improved from 8.6% pre-treatment to 6.1% within 12 months. Continuous glucose monitoring revealed time-in-range >70%. Patient weight and lipids also improved significantly. She tolerated mifepristone up to 900 mg daily. During dose titration, notable adverse effects included hypokalemia (3.1 mmol/L) secondary to unopposed mineralocorticoid receptor activation, successfully corrected with spironolactone. Additionally, she developed subclinical hypothyroidism (TSH 7–8 mIU/L & low-normal free T4), requiring close follow-up to distinguish from cortisol withdrawal–related symptoms. No evidence of adrenal insufficiency was observed.

This case illustrates three key teaching points in the management of Cushing disease. Despite gold standard therapy, persistent CD is possible, underscoring the importance of individualized management strategies. Second, mifepristone offers an effective option for refractory CD, particularly in patients with diabetes, obesity, and difficult to control cardiometabolic complications as demonstrated in recent results from the CATALYST trial. Third, therapy requires vigilant monitoring for complications such as hypokalemia and thyroid dysfunction, which can mimic or confound symptoms of cortisol withdrawal. In summary, mifepristone provided meaningful glycemic and metabolic stabilization in a patient with persistent CD after failed surgery, nonsurgical candidates or patient preference from pharmacological therapy. Recognition of adverse effects and proactive management allowed for safe, sustained therapy. This case emphasizes the importance of tailoring treatment strategies to individual comorbidities and highlights the expanding role of glucocorticoid receptor blockade in refractory CD.

V-33- Spontaneous Intramuscular Hemorrhage in Refractory Polymyositis: A Rare and Fatal Complication

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Polymyositis is a rare, chronic inflammatory myopathy that mainly affects proximal skeletal muscles. It is marked by elevated creatine kinase (CK) levels and signs of myositis on electromyography (EMG) and biopsy, without the distinctive features of other idiopathic inflammatory myopathies. Refractory cases may indicate a more aggressive or atypical form of the disease, complicating management. In this report, we describe a case of refractory polymyositis that ultimately caused several complications, including spontaneous intramuscular hemorrhage, a rare but serious complication more often linked to dermatomyositis.

A 63-year-old female with a history of hypertension and type 2 diabetes presented with three months of progressive proximal muscle weakness in both her upper and lower extremities, accompanied by dysphagia and dysarthria. She reported no fever, chills, headaches, chest pain, nausea, vomiting, abdominal pain, diarrhea, dysuria, skin lesions, or any other associated symptoms. Upon examination, weakness was observed in the neck flexors and in the proximal muscles of the upper and lower extremities. Laboratory tests revealed elevated levels of CK, liver enzymes, and CRP, with no cytopenias and stable renal function. Rheumatology, PMR, and speech therapy services were consulted. Electromyography during admission indicated findings consistent with inflammatory myopathy, particularly polymyositis. A comprehensive myositis panel yielded negative. An MRI of the most affected thigh showed findings consistent with myopathy. CT imaging of the chest, abdomen, and pelvis revealed no signs of solid organ malignancy. She was started on aggressive immunosuppressive therapy, including IV solumedrol 80 mg daily, methotrexate 15 mg subcutaneously weekly, rituximab 1 g, and IVIG. Initially, she responded well to treatment, as demonstrated by a decrease in CPK levels. However, her muscle weakness later progressed, raising concerns about impending respiratory failure, which required intubation and transfer to the ICU. Her clinical course was further complicated by recurrent pulmonary embolism and pneumonia. Serologic tests for hypercoagulability were negative. Despite ongoing biochemical improvement, indicated by decreasing CPK levels, the patient showed minimal clinical progress with the current immunosuppressive therapy. She then experienced an acute drop in hemoglobin. A CTA of the abdomen, pelvis, and extremities revealed multiple deep hematomas, including a 5.4 x 5.2 x 6.6 cm hematoma in the anterolateral chest wall, a 2.7 x 3.7 x 3.0 cm hematoma in the posterior shoulder, a 4.7 x 3.3 x 3.9 cm hematoma behind the right scapula, and a large left-sided deep hematoma measuring 5.2 x 5.2 x 29.4 cm in the lower extremity. No evidence of active bleeding was seen on imaging. Immediate intervention was not feasible due to the patient being hemodynamically unstable and on vasopressors. Subsequently, the patient expired before further interventions could be performed.

Spontaneous intramuscular hemorrhage, although usually linked to dermatomyositis, may also complicate refractory polymyositis. Clinicians should recognize this rare, potentially fatal complication in patients receiving aggressive immunosuppression to enable prompt diagnosis and management.

V-34- A Master of Disguise: A Case of 6-Mercaptopurine Overdose Presenting with Clinical Features Mimicking Both Diabetic Ketoacidosis and Thrombotic Thrombocytopenic Purpura

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Inflammatory bowel disease (IBD) is a chronic relapsing condition that includes Crohn's disease and ulcerative colitis (UC). Thiopurines like 6-mercaptopurine (6-MP) remain important therapies for remission, their use is limited by dose-dependent complications including myelosuppression, hepatotoxicity, and gastrointestinal intolerance. Toxicity is influenced by variation in metabolism and can be amplified during periods of metabolic stress. Thiopurine toxicity can present with features mimicking hematologic or metabolic emergencies, creating significant diagnostic challenges.

This is the case of a 66-year-old male Veteran with a history of UC since 1990, type 2 diabetes, CKD, bipolar II disorder, and obstructive sleep apnea. He was admitted to the intensive care unit with lethargy and abdominal discomfort. Laboratories revealed hyperglycemia (glucose 628 mg/dL) with anion gap metabolic acidosis (β-hydroxybutyrate 5.29), consistent with DKA. Additionally, demonstrated acute hepatocellular injury, anemia, thrombocytopenia, elevated LDH, and low haptoglobin. Despite preserved renal function and a low reticulocyte count, his PLASMIC score was 6, raising strong suspicion for thrombotic thrombocytopenic purpura (TTP). He underwent two sessions of plasmapheresis and received rituximab, while his UC medications were held due to concern for worsening cytopenias in the setting of thrombocytopenia. Although his laboratories briefly improved, ADAMTS13 activity returned as normal, excluding acquired TTP. Following improvement after medication cessation, evaluation revealed markedly elevated 6-MP metabolites. Chart review confirmed non-adherence to prior instructions to reduce 6-MP, and metabolic stress likely contributed to toxic accumulation. While 6-MP does not directly cause DKA, prior reports suggest it can induce hypoglycemia and metabolic acidosis that mimic DKA, particularly in patients with diabetes or impaired glucose regulation. In this case, true DKA was compounded by thiopurine toxicity, and the overlapping findings of cytopenias, hemolysis, and acidosis created a presentation nearly indistinguishable from TTP and DKA until metabolite testing clarified the diagnosis. The patient improved with discontinuation of 6-MP and was discharged on mesalamine with outpatient follow-up.

This case underscores the diagnostic challenges of drug toxicity in patients with IBD. The clinical picture was compelling for TTP, yet distinguishing features such as a low reticulocyte count, preserved renal function, and normal ADAMTS13 activity argued against it. Similarly, while the patient appeared to present with DKA, thiopurine toxicity provided a more unifying explanation by amplifying metabolic stress and creating features that overlapped with both hematologic and metabolic emergencies. Studies suggest 6-MP may impair gluconeogenesis, deplete glycogen stores, and increase skeletal muscle glucose uptake, promoting hypoglycemia and metabolic stress. Thiopurine toxicity masqueraded as two emergencies, and because the patient's initial laboratory profile fit the clinical

picture of TTP and DKA, empiric therapy was initiated given the high mortality risk of TTP. Highlighting the importance of considering medication toxicity in patients on long-term immunosuppressive therapy who develop cytopenia or metabolic crises. Careful monitoring, adherence to dosing instructions, and recognition of atypical features are essential to prevent morbidity. Even in patients with well-controlled disease, appropriate management of their medications is crucial so that toxicity does not worsen comorbidities or delay diagnosis and treatment particularly when patients taking 6-MP present with acute myelosuppression or liver injury.

V-35- When Common Pathogens Take Rare Routes: Possibly the Oldest Reported Case of Mycoplasma-Induced Tamponade

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Abstract:
Cardiac tamponade caused by Mycoplasma pneumoniae is a rare clinical condition, particularly in elderly individuals. We present the case of a 92-year-old Puerto Rican female who developed pericardial tamponade secondary to M. pneumoniae infection. This report emphasizes the importance of broad diagnostic considerations for pericardial effusions and highlights the curative potential of timely drainage and targeted antimicrobial therapy.

Introduction:
Pericardial effusion, which can lead to tamponade, is a life-threatening emergency often associated with malignancy, autoimmune diseases, or bacterial infections. Mycoplasma pneumoniae is an atypical respiratory pathogen that is rarely associated with pericardial tamponade, particularly in geriatric patients. To our knowledge, this case represents one of the oldest patients reported with tamponade caused by this pathogen.

Case Presentation:
A 92-year-old woman with several comorbidities, including atrial fibrillation, hypertension, hypothyroidism, dyslipidemia, polymyalgia rheumatica, and a history of breast and colon cancer, presented with progressive dyspnea, epigastric pressure-like pain, and oliguria. She was initially admitted for decompensated heart failure, and imaging revealed a large pericardial effusion.
Within 48 hours, she developed hemodynamic compromise consistent with cardiac tamponade, characterized by hypotension, jugular venous distension, and muffled heart sounds. Echocardiography confirmed a large effusion with diastolic chamber collapse. An emergent pericardiocentesis evacuated 450 mL of serosanguinous fluid, resulting in a rapid improvement. A pericardial drain was placed, and the patient was monitored in the intensive care unit with serial echocardiography.

Pericardial fluid analysis revealed an exudative effusion based on Light's criteria. Cytology was negative for malignancy, and differential counts showed monocytosis. Serology for M. pneumoniae returned positive, with no other pathogens identified. The patient was treated with azithromycin and colchicine, resulting in clinical recovery and complete resolution of the effusion on follow-up imaging.

Discussion:
Mycoplasma pneumoniae is a rare but treatable cause of pericardial effusion and tamponade. In elderly patients, effusions are more commonly attributed to malignancy or renal dysfunction, making infectious causes easily overlooked. The uniqueness of this case lies not only in the patient's advanced age but also in the coexistence of multiple comorbidities that initially obscured the diagnosis.

Fewer than 20 cases of M. pneumoniae–associated tamponade have been described in the literature, with most occurring in younger or immunocompromised patients. Prompt diagnosis requires the exclusion of alternative etiologies and confirmatory serologic testing. First-line treatment involves macrolide antibiotics, while adjunctive colchicine helps reduce the risk of recurrence. In this case, timely pericardiocentesis, combined with targeted antimicrobial and anti-inflammatory therapy, resulted in a favorable outcome.

Conclusion:

This case underscores the necessity of considering M. pneumoniae in the differential diagnosis of pericardial effusion and tamponade, even in very elderly patients. Early recognition, urgent pericardial drainage, and appropriate antimicrobial therapy can be lifesaving. Our report contributes to the limited literature on M. pneumoniae-related tamponade in geriatric populations, reinforcing its treatable nature.

V-36- Discordant HIT Testing in Critical Illness: Argatroban-Responsive Thrombosis Despite Negative Functional Assay

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Heparin-induced thrombocytopenia (HIT) is a potentially lethal, immune-mediated complication of heparin therapy characterized by platelet activation, thrombocytopenia, and thrombosis. Diagnosis is challenging in critically ill patients, where thrombocytopenia is multifactorial and laboratory tests may yield discordant or inconclusive results. Discordance between immunoassays (PF4 antibodies) and functional assays (serotonin-release assay [SRA]) is increasingly recognized, complicating confirmation of HIT. We present a case of severe HIT with a positive PF4 test and negative SRA, underscoring the primacy of clinical probability when laboratory findings are discordant.

An 88-year-old male with severe aortic stenosis and heart failure with preserved ejection fraction (HFpEF) was admitted for chest pain and acute decompensated heart failure. Baseline platelet count was 126,000/μL. He received prophylactic subcutaneous heparin for 7 days. On day 13, he developed altered mental status, acute liver failure, purpuric lesions, severe thrombocytopenia (nadir 25,000/μL; >80% decline), and abnormal coagulation parameters (INR 2.44, PT 27.3, and PTT 42.4). Peripheral smear revealed no schistocytes. DIC, hemolysis, and active bleeding were excluded. An extensive workup ruled out other causes of thrombocytopenia. Anti-PF4 IgG ELISA was positive (O.D. 0.650, positive if >0.5), whereas the SRA was negative (2%, negative if <20%). Venous Doppler of the portal vein and proximal lower extremities confirmed extensive thrombosis. The 4Ts score was 6 (high probability). Given the high clinical probability and progressive thrombosis, subtherapeutic argatroban at 0.1 μg/kg/min was initiated. Follow-up Doppler ultrasound after 7 days of anticoagulation showed resolution of the portal and iliac thromboses, as well as a decreased clot burden in the femoral vein. The patient was transitioned to fondaparinux before being transferred to the ward. During his ICU stay, he remained chronically ill but demonstrated gradual recovery, with improving platelet counts and liver function.

Discordance between PF4 immunoassays and functional assays is most pronounced in postoperative and ICU patients. In this setting, treatment decisions must be guided primarily by clinical probability, since confirmatory assays are often delayed. When results are available, a positive PF4 IgG in the context of high clinical suspicion and documented thrombosis should not be dismissed solely because of a negative SRA. Critically ill patients with multiorgan failure may require dose adjustments; this case supports prior reports that argatroban can be safely titrated in such patients. It is among the first to demonstrate that even subtherapeutic dosing may promote regression of established thrombosis. Long-term considerations include avoiding future heparin exposure and determining whether patients with discordant test results warrant prolonged anticoagulation, as recommended in expert guidance.

HIT should remain a clinical diagnosis supported—but not excluded—by laboratory testing. In high-risk patients, a strongly positive PF4 IgG test with compatible clinical features warrants treatment despite a negative functional assay. Our case highlights that subtherapeutic argatroban may not only prevent but also reduce thrombotic burden in critically ill patients, reinforcing the need for further research to clarify optimal dosing strategies and anticoagulation duration in discordant HIT testing.

V-37- Left-Lobe Predominant Hepatolithiasis in a Young Woman: A Rare Presentation in the Western Hemisphere

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Abstract

Hepatolithiasis, defined as the presence of calculi within the intrahepatic bile ducts proximal to the hepatic confluence, is common in East Asia but rare in Western populations (<1% of biliary stones) and particularly uncommon in young adults. In Asia, it is often linked to recurrent infections, parasitic infestations, and malnutrition, whereas in the West it is more commonly associated with biliary strictures, congenital anomalies, or prior surgery. Its rarity, along with nonspecific symptoms and variable imaging findings, frequently delays diagnosis and complicates management.

This is the case of a 34-year-old female who presented to our institution after experiencing recurrent episodes of right upper quadrant pain with associated nausea and vomiting over several years. She had previously undergone an extensive evaluation, including serologies, imaging studies, and ultimately cholecystectomy; however, her symptoms persisted and no definitive diagnosis had been established. During her most recent admission, she presented with scleral icterus, right upper quadrant tenderness, and laboratory findings consistent with cholestatic liver injury (ALP 290 U/L, total bilirubin 3.2 mg/dL), along with elevated transaminases and leukocytosis, raising concern for biliary obstruction. ERCP demonstrated dilated left-lobe ducts containing stones; smaller stones and sludge were removed, but an impacted pigment stone could not be retrieved due to ductal narrowing. Cholangioscopy and EUS performed during this admission confirmed the diagnosis of hepatolithiasis. She improved with antibiotics, intravenous fluids, and supportive care, and was discharged with referral for consideration of left hepatectomy.

Discussion & Conclusion

This case illustrates how hepatolithiasis, though rare in Western populations, can affect even young adults and remain undiagnosed despite years of symptoms and extensive evaluation. To our knowledge, no prior cases have been described from Puerto Rico, which makes this report an important reminder to consider the disease outside of its traditionally recognized regions. Chronic hepatolithiasis carries a lifetime cholangiocarcinoma risk of up to 10%, particularly when stones persist or inflammation is ongoing. While endoscopic therapy can provide partial relief, surgical resection is often required for localized disease to achieve complete clearance, control symptoms, and lower the risk of complications such as recurrent cholangitis or malignant transformation. Early recognition, timely referral for surgery when indicated, and long-term follow-up remain essential for improving outcomes.

V-38- Ceftriaxone-Induced Acute Generalized Exanthematous Pustulosis in a Patient with Osteomyelitis

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Acute generalized exanthematous pustulosis (AGEP) is a rare cutaneous reaction that is frequently triggered by antibiotics, particularly beta-lactams and macrolides. This condition is characterized by a rapid onset, typically occurring within hours to a few days, presenting as widespread erythema and the eruption of numerous small, sterile, non-follicular pustules on an erythematous base. It is often accompanied by fever and leukocytosis. Histopathological examination typically reveals subcorneal or intraepidermal pustules with a neutrophilic infiltrate.

A 60-year-old male with a medical history of hypertension, human immunodeficiency virus (HIV), progressive myeloencephalopathic leukodystrophy, and hyperlipidemia was admitted to the hospital due to persistent low back pain that had begun one month prior. He had initially sought treatment at another hospital where a magnetic resonance imaging (MRI) scan indicated concerns for diskitis and osteomyelitis at the L3-L4 vertebrae, along with an associated epidural abscess. Although he had been experiencing symptoms earlier, his pain worsened, leading to an inability to move or walk, prompting his visit to the hospital. Upon evaluation, the patient was awake and in no acute distress. His physical examination revealed bitemporal muscle wasting and diminished strength (3/5) in hip flexion of both lower extremities. Laboratory tests indicated leukocytosis, elevated inflammatory markers, and blood cultures that were positive for E. coli. The cause of the diskitis/osteomyelitis and the source of hematogenous spread remained uncertain despite additional questioning. The patient was initially treated with Ceftriaxone and Vancomycin, showing a clinical response. However, three days after starting antibiotic therapy, he developed a low-grade fever, tachycardia, and a widespread, non-desquamating, confluent erythematous rash with satellite lesions. These lesions were most prominent in the axillary folds, inguinal area, and abdomen, and were also present on both arms, although mucosal areas were spared. Consequently, antibiotic therapy was paused. Dermatology service was consulted, and a skin biopsy was performed, which revealed a mixed cellular infiltrate containing neutrophils and eosinophils. The differential favored acute generalized exanthematous pustulosis (AGEP), most likely related to cephalosporin therapy, as vancomycin is less commonly implicated than other ß-lactams. The patient was started on supportive topical corticosteroid therapy, and his lesions progressed to a desquamating rash, which resolved within one to two weeks.

This case highlights the importance of maintaining a high index of suspicion for acute generalized exanthematous pustulosis (AGEP) in patients presenting with new-onset rash following antibiotic initiation, particularly beta-lactams. Prompt recognition and discontinuation of the offending agent, along with supportive care, are essential for resolution. Clinicians should remain vigilant for drug-induced cutaneous reactions, especially in complex patients with multiple comorbidities and recent antibiotic exposure.

V-39- When Lupus Isn't the Only Culprit: Obstructive Uropathy from Fibroids in a Patient with Lupus Nephritis

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Patients with systemic lupus erythematosus (SLE), particularly those with lupus nephritis, are at increased risk for complex renal complications due to both autoimmune activity and comorbid conditions. Obstructive uropathy is an uncommon but important contributor to acute kidney injury in this population, particularly when secondary to structural abnormalities such as uterine fibroids. This case highlights the diagnostic and management challenges of acute-on-chronic renal dysfunction in a patient with SLE, complicated by obstructive uropathy from a significantly enlarged myomatous uterus.

A 41-year-old female with SLE with lupus nephritis, anti-phospholipid syndrome, cardiac valvulopathy, and hypertension presented with two weeks of worsening bilateral leg swelling. Upon evaluation, she was calm, afebrile, and in mild discomfort. She mentioned developing urinary difficulty for a week with decreased volume, shortness of breath, back pain, vomiting, fever, and fatigue. Physical exam was remarkable for a mildly tender abdomen with a palpable mass, a Foley catheter with poor urine output, and +3 pitting edema in bilateral lower extremities up to the knees. Laboratory tests showed significant electrolyte disturbances, including hyperkalemia, azotemia, hyperphosphatemia, and decreased levels of central bicarbonate, with impaired renal function and elevated creatinine. Urinalysis revealed proteinuria, hematuria, and pyuria, with no cellular casts, and a urine protein-to-creatinine ratio of 0.8, consistent with previous visits. Complement levels (C3 and C4) were decreased, with elevated anti-dsDNA and increased inflammatory markers. Abdominopelvic CT with intravenous (IV) contrast showed a uterus that measured at least 22 centimeters (cm) long by 10cm by 6cm AP by 13.6cm transversely with multiple myomas with calcifications causing anterior displacement of the urinary bladder and compression of bilateral ureters with progressive hydro-ureteral nephrosis. Given the patient's clinical scenario, she was experiencing moderate SLE disease activity, consistent with previous visits, with multiple factors contributing to worsening renal dysfunction, including obstructive uropathy. Bilateral nephrostomies were placed, resulting in marked improvement in renal function. She was discharged for outpatient gynecology follow-up. However, she was readmitted to our institution due to worsened renal function and SLE flare, presenting as cytopenias, serositis of the pancreas, pleural effusions, ascites, nephrotic range proteinuria, and hypocomplementemia. Multiple infections and worsening of underlying comorbidities complicated hospitalization. She underwent bilateral uterine artery embolization with minimal improvement in the size of the myomas. OBGYN service performed a total abdominal hysterectomy with bilateral salpingo-oophorectomy. She continued inpatient treatment with significant improvement in renal function that returned to baseline. Eventually, the bilateral nephrostomies were removed successfully, and stable renal function parameters were maintained. She was discharged with follow-up care in Rheumatology clinic.

In patients with complex autoimmune and thrombotic comorbidities, such as systemic lupus erythematosus with lupus nephritis and antiphospholipid syndrome, prompt identification and surgical resolution of obstructive uropathy are essential to mitigate renal deterioration and systemic complications. This case illustrates the critical role of timely intervention and emphasizes the necessity of coordinated, multidisciplinary management involving Obstetrics-Gynecology, Rheumatology, Nephrology and Internal Medicine to achieve optimal clinical outcomes.

V-40- The Heart's Silent Squeeze: A Young Woman's Journey from Liver Disease Misdiagnosis to Pericardial Cure.

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Introduction

Ascites is most frequently attributed to liver cirrhosis, representing the leading cause of pathologic peritoneal fluid accumulation. However, it is essential to consider cardiac etiologies, particularly in cases where clinical, laboratory, or imaging findings do not align with typical hepatic pathology. Constrictive pericarditis represents an uncommon and frequently underrecognized cause of disease that can closely resemble chronic liver disease. We describe a case of recurrent ascites that, after an exhaustive diagnostic evaluation, was ultimately attributed to underlying constrictive pericarditis.

Case Presentation

A 40-year-old woman with a prior history of seizures experienced a prolonged diagnostic journey after presenting with progressive abdominal pain, ascites, anemia, and elevated CA-125. Over subsequent months, she developed worsening bilateral lower extremity edema, initially attributed to lymphedema. Despite symptomatic management, she experienced recurrent ascites necessitating multiple hospital admissions with frequent therapeutic paracenteses and thoracenteses.

Upon re-evaluation by a gastroenterologist, the patient was found critically ill with marked abdominal distension, shortness of bread, and required a walker for mobility. Laboratory investigations revealed a cholestatic liver enzyme pattern characterized by elevated alkaline phosphatase and normal liver enzymes, without thrombocytopenia. Although chronic liver disease was considered, the biochemical profile was inconsistent with cirrhosis. Diagnostic paracentesis demonstrated a serum-ascites albumin gradient (SAAG) > 1.1 g/dL with high ascitic protein concentration, supporting a diagnosis of cardiac ascites rather than portal hypertension from cirrhosis.

Concurrent symptoms of dyspnea and peripheral edema prompted cardiology referral. Transthoracic echocardiography revealed mild and nonspecific pericardial changes, insufficient to establish a diagnosis. Subsequent invasive cardiac catheterization showed ventricular interdependence, respirophasic discordance, elevated LVEDP, and a classic square-root sign stating constrictive pericarditis. In addition, a cardiac MRI confirmed constrictive pericarditis with characteristic of pericardial thickening and impaired diastolic filling. The patient underwent surgical pericardiectomy, resulting in marked clinical improvement: resolution of ascites, normalization of alkaline phosphatase, and correction of anemia. Since her surgery, she has remained under regular gastroenterology follow ups without ascitic recurrence.

Discussion

• Constrictive pericarditis can present with predominant right heart failure symptoms, including ascites and peripheral edema, mimicking hepatic or lymphatic diseases.

• Elevated CA-125, although often associated with malignancy, can be raised in benign serosal inflammation, including pericarditis-related ascites, reflecting mesothelial cell activation.
• Ascitic fluid analysis with high SAAG > 1.1 g/dL and elevated protein suggests a cardiac origin, differentiating from cirrhosis-related low protein ascites.

• Cardiac MRI and catheterization remain gold standards for confirming constrictive pericarditis.
• Early surgical pericardiectomy is curative in most cases, preventing progression to myocardial atrophy and chronic heart failure.

Conclusion

This case exemplifies constrictive pericarditis as a rare but treatable cause of recurrent ascites with systemic manifestations that can closely mimic chronic liver disease. In refractory ascites, physicians should suspect cardiac causes when hepatic findings are incongruent and ascitic fluid analysis suggests a cardiac origin. Multimodal evaluation with advanced cardiac imaging and cardiac catheterization is essential for diagnosis. Surgical pericardiectomy can reversed symptoms and laboratories abnormalities, while increased awareness of constrictive pericarditis reduces diagnostic delays, prevents unnecessary interventions, and improves patient prognosis.

V-41- Pneumonia-Induced Pandemonium: A Neuropsychiatric Conplication Of Respiratory Infection

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Introduction

Catatonia is a complex neuropsychiatric syndrome characterized by profound psychomotor dysfunction. It is commonly associated with chronic psychiatric disorders but can also arise from medical or neurologic conditions. Untreated cases have a mortality rate of 10% to 20%, necessitating early diagnosis and prompt intervention that includes addressing both psychiatric and medical contributors. Among medical triggers, atypical pneumonia is particularly significant, as systemic inflammation, hypoxia, and metabolic stress can precipitate catatonic states. Conversely, immobility, malnutrition, and immunosuppression increases susceptibility to respiratory infections, creating a bidirectional relationship that complicates management. Recognizing this interplay is critical for optimizing patient outcomes.

Case Presentation

A 52-year-old male with schizophrenia and untreated hepatitis C was transferred from a psychiatric hospital to the emergency department due to altered mental status, hypoactivity, dry cough, and fever of 8 days' duration.

On arrival, the patient appeared profoundly hypoactive, with postural rigidity, fixed gaze, mutism, and autonomic instability. A clinical diagnosis of catatonia was suspected. The Bush-Francis Catatonia Rating Scale (BFCRS) yielded a score of 25, supporting the diagnosis. His psychiatric medications, including risperidone, depakote, and trazodone, had been reduced due to concerns about sedation, but his condition worsened.

Laboratory studies revealed leukocytosis, elevated creatine phosphokinase (CPK), and raised inflammatory markers, including procalcitonin, erythrocyte sedimentation rate, and C-reactive protein. A chest X-ray demonstrated bilateral patchy opacities and bronchial wall thickening, while a CT scan revealed bilateral ground-glass opacities and consolidations, suggesting infection. Serologic testing confirmed Mycoplasma pneumoniae infection.
A lorazepam challenge test resulted in a 40% reduction in catatonic signs, confirming the diagnosis. Within 72 hours, his symptoms progressively improved, including restored verbal responsiveness and decreased rigidity.

Empiric ceftriaxone and azithromycin were initiated, once Mycoplasma pneumoniae pneumonia was identify was tao, alongside intravenous fluids and aspiration precautions. Over four days, the BFCRS score improved from 22 to 5, with resolution of catatonic features. His respiratory status also improved, and he was transferred back to psychiatric care once stabilized.

Discussion

This case exemplifies the bidirectional relationship between pneumonia and catatonia, where infection-driven neuroinflammation, cytokine dysregulation, and hypoxia can precipitate catatonic symptoms, while catatonia itself increases the risk of pulmonary complications. Mycoplasma pneumoniae has been linked to neuropsychiatric syndromes through cytokine overproduction, blood-brain barrier disruption, and potential autoimmune mechanisms. Distinguishing catatonia from neuroleptic malignant syndrome (NMS) is critical, as catatonia responds to benzodiazepines, while NMS requires dopamine restoration. Healthcare disparities often delay pneumonia recognition in psychiatric patients, emphasizing the need for respiratory monitoring in psychiatric settings. Multidisciplinary collaboration is essential to improving outcomes, and integrated medical-psychiatric care can help prevent complications in this vulnerable population.

Conclusion

This case underscores the need for early screening protocols to identify respiratory infections in patients with severe mental illness, particularly those at risk for immobility or aspiration. Timely antibiotic therapy, benzodiazepine treatment, and supportive care are essential for improving outcomes. Multidisciplinary collaboration between psychiatrists, pulmonologists, and infectious disease specialists is critical to managing these complex cases effectively. Addressing healthcare disparities through integrated care can reduce complications and improve patient survival.

V-42- "Fulminant Hepatic Failure in a Young Woman with Takayasu Arteritis: A Rare and Fatal Complication of Large Vessel Vasculitis "

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Takayasu Arteritis (TAK) is a large-vessel vasculitis primarily affecting the aorta and its major branches, most commonly seen in women under 40. While its etiology is unclear, autoimmune mechanisms are strongly implicated. Typical features include limb claudication, diminished pulses, vascular bruits, blood pressure discrepancies, and hypertension. Complications may involve aortic aneurysm, regurgitation, stroke, myocardial infarction, and critical limb ischemia, but liver failure is not typical. Here, we present a case of a young woman with TAK who developed fulminant liver failure without clear precipitating factors.

A 28-year-old female with a known history of Takayasu's arteritis was admitted due to uncontrolled bilateral uveitis. During her inpatient stay, she developed febrile peak episodes that were found to be associated with transaminitis. She reported episodes of chills, fatigue, nonspecific abdominal pain, and anorexia. She denied nausea, vomiting, pruritus, changes in urine color, melena, or hematochezia. The patient denied any toxic habits, drug exposure, or use of immunosuppressive medications in the past year. On a physical exam, the patient was remarkable for sclera icterus with anasarca and had a benign abdominal exam. Rheumatologic workup with elevated inflammatory markers and elevated fibrinogen (38k) with preserved liver function. However, the initial workup revealed mildly elevated liver enzymes. An autoimmune workup, including ANA, complement levels, IgE, IgA, IgG, P-ANCA, C-ANCA, IgG4, ceruloplasmin, rheumatoid factor, and alpha-1 antitrypsin tests, all returned negative results. The patient was treated empirically for herpes with Acyclovir and given broad-spectrum antibiotics; however, repeated infectious workup came out negative, including blood and urine cultures, tests for HIV, CMV, EBV, RPR, hepatitis panel, and HSV/VZV. Autoimmune hepatitis was considered in the differential diagnosis; however, tests for anti-smooth muscle and anti-mitochondrial antibodies were negative. As a diagnosis of exclusion, there was concern that the patient's condition was secondary to known rheumatologic issues related to Takayasu's arteritis affecting hepatic circulation due to improvement with immunosuppressive and steroid therapy. It was suggested that the etiology of the patient's condition, which progressed with hypotension, caused acute liver injury. Despite initial stabilization, the patient's condition deteriorated. She experienced worsening transaminitis (AST 13,831, ALT 5,913), hyperbilirubinemia, thrombocytopenia, rising INR, and hepatic encephalopathy, which were consistent with fulminant liver failure. Unfortunately, the patient passed away despite further intervention.

This case illustrates the diagnostic complexity associated with fulminant liver failure in a young patient diagnosed with Takayasu's arteritis, where, despite thorough evaluation, no

infectious, toxic, or typical autoimmune causes were identified. Notably, fulminant hepatic failure in this scenario may serve as the initial or sole manifestation of large-vessel vasculitis, potentially occurring in the absence of apparent vascular symptoms. This highlights the crucial need to consider hepatic failure in the differential diagnosis of unexplained transaminitis in patients with Takayasu's arteritis, as early recognition, careful monitoring, and a multidisciplinary approach may enhance patient outcomes.

V-43- Diabetes Ketoacidosis in a patient on Pembrolizumab; A case report of Inmune Checkpoint Inhibitor - Associated endocrine toxicity

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Pembrolizumab is a humanized monoclonal IgG 4 kappa antibody binds to PD 1 receptor on lymphocytes and prevent the inhibition of the immune system checkpoint from several types of tumors that express PD L 1 which inhibited the T cell function. This medication enhances the
immune response and is the treatment of choice for many malignancies It has been associated with the development of anti glutamic acid decarboxylase (anti GAD). This class of drug has been reported with elevated risk for colitis, pneumonitis, liver, kidney toxicity and endocrinopathies.

This is the case of a 72-year-old female with past medical history of hypertension and endometrial and liver malignancy who came to the emergency department presenting with polyuria, polydipsia, nausea, confusion, dry mouth and shortness of breath. Pembrolizumab was recently started two days prior to the onset of symptoms. Laboratories findings showed hyperglycemia of 664 mg/dl, pH 7.125, moderate ketones, bicarbonate level of 6 mmol/L, meeting the criteria for diabetic ketoacidosis (DKA). This patient had no prior history of diabetes mellitus and denied any hypoglycemic event during her lifetime. TSH 0.11 UIU/ml, Free T4 1.9 ng/dl, Free T3 2.5 ng/dl, normal FSH. Amylase of 156 U/L (28-100), lipase 258 U/L (13-60) HGB A1C 6.7 % and positive anti GAD antibodies. The patient was admitted to the intensive care unit and was placed on insulin drip, was given aggressive intravenous volume resuscitation and correction of electrolytes. The patient was successfully treated for DKA which responded adequately and was discharged home on insulin basal-bolus regimen.

DKA has been described with the use of Pembrolizumab on 0.1% of cases, making this a very rare presentation. This case highlights the importance of keeping in mind medication side effects in order to diagnose and treat accordingly and identify reversible causes. This patient was able to continue Pembrolizumab along with the addition of an insulin regimen.

V-44- Fatal Disseminated Staphylococcus epidermidis Infection in an Uninsured Patient With Untreated HIV/AIDS in Puerto Rico.

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Introduction

Staphylococcus epidermidis, typically considered a skin commensal or contaminant, can act as an opportunistic pathogen in immunocompromised hosts. It is increasingly implicated in serious infections, including bacteremia and end-organ dissemination, especially in those with chronic illness or barriers to care. This case illustrates the consequences of delayed HIV treatment and lack of insurance following relocation, culminating in fatal disseminated S. epidermidis infection.

Case Presentation

A 34-year-old male with a past medical history of HIV (diagnosed in 2015), pancolitis, and peptic ulcer disease presented with a one-week history of pleuritic chest pain, dyspnea, productive cough, fevers, and fatigue. He had been non-adherent to antiretroviral therapy since 2023 after losing insurance coverage upon relocating to Puerto Rico.

On admission, he had moderate respiratory distress. Chest CT revealed extensive right lung consolidation with moderate pleural effusion and debris in the right mainstem bronchus. He met criteria for type 1 acute hypoxemic respiratory failure ($PaO_2/FiO_2 = 285$), requiring high-flow nasal cannula at 40 L/min, FiO_2 30%. PSAI/PORT score was class III.

Initial labs showed ALC 522, elevated ESR (110), and procalcitonin (16.8 ng/mL). CD4 count was 44 cells/μL, confirming AIDS. Blood cultures were positive for Streptococcus pneumoniae (4/4 bottles), and pleural fluid later grew Staphylococcus haemolyticus. He was started on cefepime, doxycycline, steroids, and later vancomycin and TMP/SMX.

On hospital day 6, a new CT scan revealed a moderate-to-large right pneumothorax, patchy bilateral opacities, and cystic changes. A chest tube was placed. Repeat blood cultures taken on hospital day 11 grew Staphylococcus epidermidis. Despite vancomycin initiation, the patient deteriorated rapidly, developing multiorgan failure. He died on hospital day 13 due to septic shock and respiratory failure.

Discussion

This case underscores the potential for coagulase-negative staphylococci (CoNS), particularly Staphylococcus epidermidis, to cause life-threatening infection in severely immunocompromised patients. Though often dismissed as contaminants, S. epidermidis should not be underestimated when repeatedly isolated in high-risk hosts.

The patient’s pleural fluid culture notably grew Staphylococcus haemolyticus, while a subsequent blood culture yielded Staphylococcus epidermidis. These species are both members of the CoNS group and are commonly associated with skin flora. Differentiating between them in clinical cultures can be challenging due to their similar colony morphology, overlapping biochemical characteristics, and potential limitations in identification methods, especially when advanced molecular diagnostics are unavailable. Misidentification can lead to underestimation of pathogenic potential and inappropriate antimicrobial therapy.

The patient’s loss of insurance following relocation delayed both antiretroviral therapy and access to preventive care, compounding his immunosuppression and increasing his vulnerability to bacterial superinfections. The progression from severe CAP due to S. pneumoniae to pleural co-infection, pneumothorax, and disseminated CoNS bacteremia illustrates the compounding risks in medically marginalized populations.

Despite broad-spectrum antibiotics and intensive care, the patient's trajectory was ultimately fatal, highlighting how systemic healthcare barriers and advanced immunosuppression create a lethal synergy in HIV/AIDS patients.

V-45- Not So Forgotten After All: Lemmierre-like Syndrome in an Elderly Patient with a Vascular Access Device

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Introduction: Once labeled the "forgotten disease", Lemierre's syndrome (LSP) may not be that uncommon after all. Classically, LS is characterized by history of recent oropharyngeal infection, bacteremia, internal jugular vein thrombophlebitis (IJVT) and, metastatic septic emboli, mainly due to Fusobacterium necrophorum. Traditionally it is seen in younger, immunocompetent individuals following pharyngitis or tonsillitis.

Recently, reports have described atypical variants known as Lemierre-like syndrome (LLS). These include alternative pathogens and non-oropharyngeal sources of infection. Here we report an unusual case of LLS in an older adult, where the infectious source was not a preceding pharyngeal illness but rather a Mediport associated infection caused by methicillin-sensitive Staphylococcus aureus (MSSA). The mortality rate of LLS caused by this pathogen is approximately 16%. Timely diagnosis, appropriate antibiotic therapy, and infection source control remain foundation of treatment, while the role of anticoagulation as adjuvant therapy continues to be controversial. This case highlights the importance of considering LLS in vulnerable patient populations with indwelling central venous catheters (CVCs), where MSSA can act as primary pathogen.

Case: An 82-year-old male with past medical history of type 2 diabetes mellitus, hypertension, prostate cancer treated with resection, and right neck angiosarcoma previously managed with radiotherapy and currently on chemotherapy, presented with fever, chills, and generalized rash over the right neck.

On arrival, patient was tachycardic (113 bmp) but other wise stable. Labs showed leukocytosis (14.27 x 10^3/µL), acute kidney injury (Cr 1.45 mg/dL), and urinalysis positive for ESBL-producing organism. He was started on aztreonam and vancomycin.

Despite therapy, he developed poor appetite, malaise, persistent leukocytosis, and elevated inflammatory markers (CRP 12.44 mg/dL, ESR>140mm). Blood cultures grew MSSA; ertapenem was started, and aztreonam discontinued. His mediport was removed, yielding purulent drainage, confirming the source.
Patient eventually developed left arm edema and erythema. Vascular studies revealed thrombosis of the left internal jugular, subclavian, axillary and cephalic veins. Anticoagulation with apixaban was initiated for extensive thrombosis, to be continued for 6 months as per vascular surgery recommendations.

TEE showed no evidence of endocarditis. After 17 days on adequate therapy follow-up blood culture came back negative. Patient was discharged home with anticoagulation with apixaban and antibiotic therapy with vancomycin and ertapenem to complete 5 weeks

since negative blood cultures.

Discussion: Although classically seen in young patients with Fusobacterium, LLS has been increasingly reported in older adults, often linked to S. aureus and vascular access device. The rise of antibiotic resistance has also contributed to the growing number of these cases. S. aureus mediated LLS carries higher mortality than classic LS. Management depends on timely recognition, antibiotics, and source control. Persistent bacteremia should prompt evaluation for intravascular sources. The role of anticoagulation remains controversial but was given in our case due to extensive thrombosis.

This case expands the spectrum of LLS and stresses the need to consider the disease in older adults with vascular decides and unexplained persistent bacteremia with IJVT.

V-46- Unexpected Culprit: Epiglottitis Secondary to Candida parapsilosis in an Immunocompetent Host

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Epiglottitis is a serious and potentially life-threatening infection of the supraglottic airway, most commonly caused by bacterial pathogens such as Haemophilus influenzae type b, Streptococcus pneumoniae, and Staphylococcus aureus. Fungal involvement is exceptionally uncommon and typically arises in patients with impaired immunity, prolonged antibiotic exposure, or the presence of indwelling medical devices. While Candida albicans has, on rare occasions, been identified as a causative agent, Candida parapsilosis has been scarcely reported, particularly in individuals without underlying immunodeficiency. We describe an unusual case of epiglottitis caused by Candida parapsilosis in an otherwise healthy adult, highlighting the importance of considering uncommon pathogens when evaluating airway infections.

An 83-year-old male presented to the emergency department with a two-day history of shortness of breath, hoarseness, nasal congestion, and cough. On examination, he was noted to have bilateral wheezing and was treated with bronchodilators and intravenous corticosteroids, though bronchospasm persisted. Following reevaluation, his wheezing resolved, and he was admitted with a working diagnosis of acute bronchitis for further management with scheduled respiratory therapies. Shortly after arrival at the ward, however, he developed recurrent wheezing accompanied by new-onset stridor. Despite additional treatments with bronchodilators, intravenous corticosteroids, and two doses of racemic epinephrine, his stridor persisted, and he became tachypneic with abdominal respirations. Given impending respiratory failure, he was intubated and transferred to the intensive care unit for advanced management. A CT scan of the neck demonstrated supraglottic edema involving adjacent airway structures, raising concern for epiglottitis. Empiric ceftriaxone and corticosteroids were initiated to aid with inflammation. During his ICU course, the patient experienced an acute episode of refractory hypotension. Repeat blood cultures were obtained during the event and subsequently grew yeast. Antifungal therapy with micafungin was started, and further workup for underlying immunodeficiency, including HIV and immunoglobulin levels, was ordered and came back negative. Final cultures identified Candida parapsilosis. Infectious diseases consultation determined that Candida was the likely cause of the patient's presentation despite the absence of immunosuppression. He completed a course of micafungin with clinical improvement.

This case illustrates an unusual presentation of epiglottitis due to Candida parapsilosis in an immunocompetent host. While bacterial pathogens remain the primary culprits, clinicians should be aware that fungal organisms, although rare, can also present in patients without traditional risk factors for immunosuppression. Early recognition, appropriate antimicrobial coverage, and consideration of atypical pathogens are essential in guiding management, particularly when patients fail to improve with standard therapy. This report expands the spectrum of organisms associated with epiglottitis and highlights the importance of maintaining a broad differential diagnosis in cases of acute airway compromise.

V-47- From Bump to Breakdown: A Rare Presentation of Leukocytoclastic Vasculitis

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Introduction

Leukocytoclastic vasculitis (LCV) is a small vessel vasculitis most frequently associated with infections, medications, autoimmune conditions, or malignancies. Mechanical trauma has also been reported as a potential precipitant. Trauma-induced LCV is exceptionally rare, but its recognition is of clinical importance. Histopathologically, LCV is defined by neutrophilic infiltration of vascular walls, leukocytoclasia, fibrinoid necrosis, and extravasation of erythrocytes. Clinically, it often presents as palpable purpura, usually on the lower extremities. The overall incidence of biopsy confirmed LCV has been estimated at approximately 45 cases per million people per year. The rarity of trauma-related LCV poses diagnostic challenges, especially in patients with comorbidities that compromise vascular health, such as diabetes mellitus or antiphospholipid syndrome (APS). In such individuals, post-traumatic necrosis is often misattributed to infection or ischemia, delaying accurate diagnosis and treatment. By contributing to the limited evidence base, this case emphasizes the importance of early histopathologic evaluation following minor trauma in high-risk populations, enabling timely recognition and management.

Case Presentation

A 45-year-old Hispanic woman with uncontrolled type 2 diabetes mellitus, APS on chronic warfarin therapy, hypertension, chronic microcytic anemia, and a remote cerebrovascular accident presented with severe pain and discoloration of the left great toe. Symptoms began acutely after blunt trauma sustained when she struck her foot against a metal bedpost. On examination, distal toe necrosis was evident, accompanied by erythema, edema, and marked tenderness.

Initial vitals revealed low-grade fever and tachycardia. Laboratory testing showed leukocytosis, elevated inflammatory markers, and a supratherapeutic INR of 6.93. Imaging revealed a comminuted distal phalanx fracture but no abscess or fluid collection. Arterial Doppler showed no stenosis, and venous Doppler excluded deep vein thrombosis. Empiric vancomycin and piperacillin-tazobactam were initiated for presumed soft tissue infection, given her diabetic status, but blood and wound cultures remained negative.

Despite broad-spectrum antibiotics, necrosis progressed. Surgical amputation of the left first toe was required. Histopathologic analysis revealed findings consistent with LCV. Postoperatively, the patient was managed with a multidisciplinary team. Hydroxychloroquine was initiated for vasculitic pathology in the setting of APS, while anticoagulation was resumed. Her diabetes regimen was optimized with basal-bolus

insulin. Recovery was favorable, with no further tissue loss.

Discussion

This case illustrates the diagnostic and therapeutic complexities of trauma-induced LCV, particularly in the presence of significant comorbidities. Clinically, the presentation mimicked infection or ischemia, both common in diabetic and APS patients. The lack of microbiologic growth and progression despite antibiotics prompted histologic evaluation, which confirmed vasculitis.

In this patient, APS likely contributed endothelial dysfunction and hypercoagulability, while poorly controlled diabetes impaired neutrophil function and wound healing. Together, these factors amplified vascular injury, resulting in fulminant necrosis after minor trauma. Early biopsy was crucial in establishing the diagnosis, guiding appropriate therapy, and preventing further compromise.

This report adds to the sparse literature on trauma as a trigger for LCV and reinforces the need for clinicians to maintain vigilance when necrosis progresses despite adequate antimicrobial coverage. Histopathologic confirmation, comorbidity management, and early specialist input are key to improving outcomes in these rare cases.

V-48- Walking on an Empty Tank: Extreme Hemoglobin Tolerance in a Patient with Hereditary Hemorrhagic Telangiectasia

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Introduction

Hereditary hemorrhagic telangiectasia is a rare autosomal dominant vascular disorder marked by recurrent epistaxis, mucocutaneous telangiectasias, and visceral arteriovenous malformations. Chronic bleeding frequently causes iron-deficiency anemia and transfusion dependence. Transfusion guidelines typically recommend intervention at hemoglobin <7 g/dL, with values <5 g/dL considered life-threatening due to tissue hypoxia and risk of cardiovascular collapse. Survival at hemoglobin values below 3 g/dL is exceedingly rare, and preserved functionality at these levels has scarcely been described. We present a 62-year-old HHT patient who maintained full-time physical employment at profoundly low Hb levels, illustrating extraordinary physiologic adaptation to chronic anemia and challenging rigid transfusion thresholds.

Case Presentation

A 62-year-old man with known HHT presented to the emergency department with generalized malaise. He reported recurrent hospitalizations for epistaxis and anemia, requiring transfusions every 4–6 weeks. Despite profoundly low hemoglobin, he performed occupational duties and daily activities, only experiencing fatigue when hemoglobin dropped below 2.5 g/dL. He denied dyspnea, chest pain, syncope, or neurologic symptoms. On presentation, he was alert, conversant, and in no acute distress. Vital signs were stable and physical examination revealed pallor and mucocutaneous telangiectasias on the lips and tongue. Laboratory evaluation revealed hemoglobin of 1.9 g/dL (range on prior admissions 1.8–2.2 g/dL), hematocrit 7%, and normocytic indices (MCV 86 fL). Reticulocyte count was low-normal. Ferritin was markedly elevated (>1500 ng/mL) with basophilic stippling on peripheral smear. Lactate dehydrogenase and bilirubin were within normal limits, excluding acute hemolysis. Chest radiograph was normal. The patient received four units of packed red blood cells, raising his hemoglobin to 7.4 g/dL. He tolerated the transfusion well and was discharged with outpatient hematology follow-up and continuation of iron chelation therapy.

Conclusion

This case highlights remarkable tolerance to extreme anemia in HHT, where chronic blood loss often necessitates transfusion. Our patient maintained functional capacity despite Hb <2.0 g/dL, challenging typical decompensation at Hb <5 g/dL. This paradox is explained by physiologic adaptation: gradual Hb decline allows compensatory mechanisms like increased cardiac output, plasma volume expansion, vital organ perfusion redistribution, and elevated 2,3-diphosphoglycerate levels, shifting the oxyhemoglobin dissociation curve for enhanced oxygen unloading. Microvascular density and oxygen extraction support tissue survival. Clinically, this case underscores rigid transfusion threshold limitations.

While current guidelines (Carson et al., 2023) recommend a restrictive Hb threshold (<7 g/dL) for most hospitalized patients, HHT guidelines (Faughnan et al., 2020) offer limited specific guidance on transfusion, emphasizing individualized, symptom-based care in chronic disorders. While exceptional, transfusion dependence carries risks: hemochromatosis, alloimmunization, and infections, requiring monitoring and chelation. Our patient's preserved function at work, unlike previous symptomatic reports, highlights unique chronic adaptation. This demonstrates extraordinary physiologic resilience, challenging conventional thresholds and emphasizing individualized, symptom-guided management in HHT and chronic blood loss. Clinicians must recognize such adaptation, monitoring for long-term transfusion complications.

V-49- Fulminant Mucinous Adenocarcinoma of the Gallbladder in a Recent Migrant

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Introduction:

Mucinous adenocarcinoma of the gallbladder is a pathologic variant defined by abundant extracellular mucin and associated with aggressive clinical behavior. Unlike more common forms of gallbladder adenocarcinoma, it often lacks distinguishing clinical features and can present with atypical laboratory findings. This case illustrates an unusual constellation of preserved liver function, isolated carcinoembryonic antigen elevation, and an exceptionally fulminant clinical course in a recent migrant, highlighting the diagnostic complexity and systemic barriers that shaped the outcome.

Case Description:

A fifty-year-old Venezuelan man with no significant medical history presented with progressive abdominal distension, lower extremity edema, and constitutional symptoms. He had recently migrated to the United States after prolonged travel through Central America. Examination revealed tense ascites and peripheral edema. Laboratory evaluation showed markedly elevated carcinoembryonic antigen with normal liver function tests, ceruloplasmin, autoimmune panel, and CA 19-9. Ascitic fluid demonstrated a low serum-ascites albumin gradient, and cytology confirmed malignancy.

Computed tomography revealed a large gallbladder mass with hepatic invasion and peritoneal spread. Biopsy established mucinous adenocarcinoma with more than fifty percent extracellular mucin. The disease was staged as IV and considered unresectable. Outpatient chemotherapy was recommended, but initiation was delayed due to lack of insurance.

Two weeks later he came back with worsening ascites and melena. Endoscopy identified duodenal obstruction requiring stenting. His condition deteriorated rapidly with acute kidney injury requiring dialysis, sepsis, and spontaneous bacterial peritonitis. Despite intensive supportive care, he died within forty-eight hours of readmission.

Discussion:

The clinical rarity of this case lies not only in the histologic subtype but in the discordant presentation: a strikingly elevated carcinoembryonic antigen with otherwise normal hepatic profile and tumor markers, malignant ascites without portal hypertension, and a fulminant clinical course leading to death within weeks of diagnosis. These features distinguish it from more typical gallbladder cancers, which usually present with abnormal liver function tests or obstructive jaundice. The patient's preserved baseline status contrasted sharply with his rapid decline, underscoring the biological aggressiveness of mucinous histology.

In addition, social determinants played a decisive role. Recent migration, interrupted healthcare access, and lack of insurance delayed systemic therapy, compounding the poor prognosis of this disease.

Conclusion:

Mucinous adenocarcinoma of the gallbladder is clinically elusive, presenting with atypical laboratory findings and an unusually aggressive trajectory. This case demonstrates that even in the absence of hepatic dysfunction, malignant ascites with isolated tumor marker elevation should raise suspicion for advanced biliary malignancy. The patient's rapid deterioration highlights the lethal synergy between aggressive tumor biology and systemic barriers to care. Early recognition of these clinical red flags and heightened awareness of cancer risks in migrant populations are critical for timely intervention.

V-50- Severe Pulmonary Hemorrhage in Leptospirosis: A Case of Unexpected Recovery

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Introduction:
Leptospirosis can range from a mild illness to severe disease with multi-organ involvement. Pulmonary complications such as diffuse alveolar hemorrhage and ARDS are uncommon but carry high mortality.

Case Description:
An 84-year-old male with chronic kidney disease stage 3a, hypertension, type 2 diabetes, and coronary artery disease s/p CABG (2006) presented with three days of watery diarrhea, malaise, fever, and lower back pain. He denied cough, dyspnea, dysuria, abdominal pain, or vomiting. Social history was notable for chronic alcohol abuse, a 40–pack-year smoking history, and residence in a rural area where he works on a farm with multiple chickens and goats.

On examination, he appeared acutely ill but alert, without petechiae, jaundice, or orthostatic changes. Laboratory studies revealed leukocytosis (9,100/mm^3 with 4 bands), thrombocytopenia (90,000/mm^3), worsening renal function consistent AKI stage III (BUN 67.2 mg/dL, creatinine 4.91 mg/dL) and normal total bilirubin (0.33 mg/dL), with preserved transaminases. Procalcitonin remarkable at 1.96 ng/mL, lactic acid 1.9 mmol/L, and proBNP markedly elevated at 29,379 pg/mL (from 534). Chest X-ray showed increased parenchymal markings suggestive of pulmonary edema. Given the presence of reported flue-like symptoms, thrombocytopenia and with exposure to mosquitoes, a preliminary diagnosis of dengue was considered. Differential diagnoses included leptospirosis, vasculitis, and viral infections. An infectious workup was initiated, including stool cultures, C. difficile PCR Toxin B, Shiga toxin assay, H. pylori testing, respiratory viral panel, and serologies for dengue and leptospirosis.

During the first hospital night, he developed acute respiratory distress along with hypoxia requiring BiPAP and was transferred to the ICU, where he progressed to septic shock requiring vasopressors. Chest CT later showed bilateral pleural effusions and patchy infiltrates. His course was complicated by worsening thrombocytopenia (21,000/mm^3), rising bilirubin (3.8mg/dL), worening renal function (BUN 106.4mg/dL and creatinine 6.96 mg/dL) and progressive leukocytosis. He subsequently required intubation and bronchoscopy confirmed diffuse alveolar hemorrhage. The patient was initially started on intravenous doxycycline and empirical broad-spectrum coverage with meropenem due to hemodynamic instability. Due to worsening respiratory status and evidence of alveolar hemorrhage, and moderate ARDS intravenous corticosteroids were initiated. Also, patient underwent electrical cardioversion for hemodynamically unstable atrial flutter and successfully converted to normal sinus rhythm. Leptospira DNA testing on 6th day of admission returned positive, confirming the diagnosis of Leptospirosis associated Severe

Pulmonary Hemorrhagic Syndrome (SPHS) and antibiotics were narrowed to ceftriaxone.

The patient's renal function and platelet count gradually recovered, vasopressors were discontinued, and he was extubated on day 9 of admission. Oxygen supplementation was weaned off, and he was discharged in stable condition.

Discussion:

This case underscores the potential severity of leptospirosis, which can progress to diffuse alveolar hemorrhage, ARDS, septic shock, and multi-organ dysfunction. The absence of early jaundice and the overlap with other differential diagnoses delayed timely recognition, emphasizing the need for a high index of suspicion in endemic settings. Although the role of corticosteroids in pulmonary hemorrhage remains debated, in this case, their use in combination with prompt antimicrobial therapy and intensive supportive care contributed to a favorable outcome.

V-51- Mediastinal Ectopic thyroid tissue: A Rare Cause of Chest Pain

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Ectopic thyroid tissue (ETT) is the abnormal location of thyroid tissue outside its typical anatomical site in the neck. ETT most often arises from a developmental anomaly during embryologic thyroid descent; the exact cause remains unclear. Most patients with thyroid ectopia are asymptomatic; however, when symptomatic, they may present with obstructive complaints or hypothyroidism, depending on ectopic tissue size, anatomical relationships, or concurrent thyroid disease. On CT, ETT is densely iodine-rich (typically 70 ± 10 HU on non-contrast scans) due to intracellular iodine accumulation. Treatment is generally surgical in symptomatic patients. Mediastinal ectopic thyroid tissue, a rare subtype, represents ~1% of all ETT cases. The differential diagnosis of mediastinal masses is broad and includes thymoma, germ cell tumors, lymphoma, neurogenic tumors, benign cysts, lipoma, esophageal lesions, paragangliomas, metastases, and mediastinal ETT. We present the case of a 56-year-old male who presented to the emergency department with atypical chest pain and incidental findings of a mediastinal mass.

56-year-old male patient with a history of End Stage Renal Disease on Hemodialysis and Coronary Artery Disease status post percutaneous coronary intervention (2024) who presented to the Emergency Department due to acute dyspnea at rest and intermittent sharp chest pain radiating to the scapula, worsened by chest expansion. Physical examination was remarkable for hypoxemia and tachypnea. ECG revealed no acute ischemic changes. Based on presenting symptoms, a work-up was performed to rule out a cardiopulmonary emergency. CT angiography revealed no evidence of pulmonary embolism; however, it did show an incidental finding of a well-circumscribed 5.9x2.6x4.8cm solid mass in the anterior mediastinum with punctate calcifications within it. A subsequent PET CT Scan did not reveal hypermetabolic lesions; however, it demonstrated a mediastinal mass with a high-density lesion, as indicated by Hounsfield Units (HU) of 75. Core needle biopsy suggested benign thyroid parenchyma with focal hyperplastic changes, consistent with ectopic thyroid tissue. Thyroid function test revealed TSH within normal limits. A cardiothoracic surgery consult was consulted; the patient is undergoing evaluation for management.
Mediastinal ectopic thyroid tissue, although rare and usually asymptomatic, should be considered in patients presenting with acute symptoms such as dyspnea and chest pain, as it may mimic cardiopulmonary emergencies. This case highlights the diagnostic challenges of incidental findings during emergent evaluations and underscores the importance of considering ectopic thyroid in the differential diagnosis of mediastinal masses. Given its rarity, no standardized guidelines exist for management, particularly in euthyroid patients with compressive symptoms, posing significant challenges in therapeutic decision-making.

V-52- Old Drug, New Target: Rapid IgA Nephropathy Improved with Budesonide

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IgA Nephropathy (IgAN), characterized by hematuria often following respiratory or gastrointestinal infections, can progress to proteinuria, hypertension, chronic kidney disease, and fulminant renal failure. Although it may occur at any age, diagnosis is most common in the second and third decades of life. Because its clinical features overlap with those of other glomerular diseases, IgAN can be challenging to recognize and requires a kidney biopsy for confirmation.

46-year-old woman with hypertension, gout, neuropathy, dyslipidemia, idiopathic intracranial hypertension status post ventriculoperitoneal shunt, and epilepsy. While living in Florida, she was diagnosed with IgAN after presenting with hematuria and proteinuria. A left kidney biopsy showed segmental glomerulosclerosis, mild interstitial fibrosis, tubular atrophy, and no mesangial or endocapillary hypercellularity or crescents. She was treated with high-dose prednisone, losartan, and dapagliflozin for six months. During this time, she developed prednisone-related side effects, including moon face and purple striae, without improvement in proteinuria or hematuria.

After relocating to Puerto Rico, she was evaluated in the nephrology clinic. Laboratory studies showed nephritic-range proteinuria of 2,536 mg/day, microscopic hematuria, and a decreased eGFR of 65. Complete blood count revealed microcytic anemia (hemoglobin 7.7 g/dL, hematocrit 26%), and iron studies confirmed iron deficiency. Given the lack of response to prior therapy, targeted-release budesonide was initiated in February 2025. By August 2025, her hematuria had resolved, proteinuria decreased to 66 mg/day, and eGFR improved to 80. She tolerated treatment well without recurrence of corticosteroid-related side effects.

IgAN results from the deposition of galactose-deficient IgA1 (Gd-IgA1)–containing immune complexes in the glomerular mesangium, which trigger inflammation and injury. The gut-associated lymphoid tissue, particularly Peyer's patches in the distal ileum, is a major source of Gd-IgA1. Targeted-release budesonide is a steroid also used in inflammatory bowel disease, is coated to release its effect only in the ileocecal area. This targeted action reduces the production of Gd-IgA1 while limiting side effects in the rest of the body. In contrast, newer drugs such as endothelin A receptor antagonists act by improving kidney blood flow and protecting podocytes, rather than addressing the underlying immune problem.

This case highlights the importance of early diagnosis through hematuria detection, renal biopsy, and thorough clinical evaluation, including assessment of proteinuria, renal function, and risk factors for disease progression. This patient improvement with budesonide demonstrates the value of early biopsy confirmation and early initiation of targeted therapy. Management should be individualized, with strict blood pressure control,

reduction of proteinuria using renin–angiotensin–aldosterone system inhibitors, and, in selected cases, immunosuppressive treatment.

Recently, novel therapies targeting the mucosal immune system, such as budesonide in combination with SGLT2 inhibitors, have demonstrated excellent outcomes in slowing disease progression, particularly when started early reinforcing the importance of an early diagnosis. Integrating clinical expertise with latest evidence may enable earlier intervention, reduce the risk of irreversible kidney damage, and improve long-term prognosis.

V-53- From Lungs to Electrolytes: The Triad of SIADH from Bacteremia and Empyema

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Community-acquired Pneumonia (CAP) remains a significant cause of morbidity and mortality among elderly patients. Streptococcus pneumoniae is the most common bacterial pathogen associated with CAP and can lead to many complications including sepsis, respiratory failure, and pleural empyema. Hyponatremia during an infectious process is something underdiagnosed as being a metabolic complication with etiology in many cases could be due to the syndrome of inappropriate antidiuretic hormone secretion (SIADH). We present a clinical scenario of a male patient with severe CAP complicated by acute hypoxemic respiratory failure (ARF), empyema, and bacteremia that developed into SIADH.

A 64-year-old man with past medical history of hypertension, asthma, presented to the emergency department with two days of chest pain and a productive cough. Upon arrival, he was afebrile, tachypneic, slightly hypotensive, and borderline hypoxemic with a nasal cannula at 4 liters. Laboratory evaluation revealed marked leukocytosis, lactatemia, and hypoxemia requiring Bilevel Positive Airway Pressure (BiPAP). Chest radiograph (CXR) showed dense consolidation of the left lower lobe. The patient was admitted to the intensive care unit with severe sepsis and Type 1 ARF. Intravenous Ceftriaxone and Azithromycin were initiated, pending culture results.

Patient once stabilized was sent to the Internal Medicine ward where Blood cultures grew Streptococcus pneumoniae resistant to Trimethoprim/Sulfamethoxazole, Ceftriaxone and Fluoroquinolones; patient was started on Vancomycin. Despite antibiotic therapy, he developed persistent shortness of breath. New laboratories showed CRP >300 mg/L with improved leukocytosis. Repeated CXR revealed worsening opacities with follow up Chest Computer Tomography (CT) revealing a large partially loculated left sided pleural effusion with signs concerning for empyema. Ultrasound-guided thoracentesis showed a purulent fluid, pH 7.15, LDH > 2500 confirming our diagnosis. A chest tube was then inserted by Interventional Radiology with drainage of yellow, cloudy fluid. Patient transitioned to Ampicillin/Sulbactam to cover for anaerobes. Once chest tube was placed, the patient developed marked hyponatremia (Na^+ 125 mmol/L) with calculated serum osmolality 357 mOsm/kg with inappropriately high urine osmolality, consistent with SIADH related hyponatremia. Patient started on Urea for hyponatremia management. Follow up imaging demonstrated poor improvement of the loculated pleural effusion still present and intrapleural fibrinolysis was performed resulting in marked improvement. Chest tube then removed 12 days later. The patient slowly improved with resolution of sepsis and stabilization of sodium levels. He was successfully weaned from oxygen supplementation, and discharged to complete intravenous antibiotics for 42 days.

This case demonstrates the potential for Streptococcus pneumoniae to cause severe systemic complications in older adults. The rapid progression from sepsis due to CAP to

ARF, and the development of pleural empyema highlights the importance of early detection. Early imaging, prompt drainage, and intrapleural therapy for source control are essential in patients with persistent fever and dyspnea despite antibiotics. Electrolytes should be carefully monitored as hyponatremia due to SIADH is an underrecognized complication of pneumonia, but even more due to empyema. Overall, this case emphasizes the importance of early recognition of severe CAP complications, including SIADH and a multidisciplinary approach that includes antimicrobial therapy, ventilatory support, pleural intervention, and electrolyte management.

V-54- Feathers and Fungi: Severe Pulmonary Histoplasmosis in a Patient with Idiopathic CD4+ Lymphocytopenia

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We report a case where severe pulmonary histoplasmosis unveiled Idiopathic CD4+ Lymphocytopenia (ICL), a primary immunodeficiency with fewer than 500 reported cases worldwide. Histoplasmosis is a well-known but under-recognized endemic mycosis in Puerto Rico, with an annual incidence of 1-2 cases per 100,000 population, classically associated with cave outbreaks and immunocompromised populations. To our knowledge, this represents the first reported case of ICL-associated histoplasmosis in Puerto Rico, emphasizing the importance of maintaining broad differential diagnoses for opportunistic infections in seemingly immunocompetent hosts.

A 69-year-old woman from rural Puerto Rico with osteoarthritis presented with progressive dyspnea, fever (39.2°C), and hypoxemic respiratory failure requiring noninvasive positive-pressure ventilation. Physical exam revealed diffuse pulmonary crackles and lower extremity edema. Chest CT demonstrated diffuse bilateral ground-glass opacities consistent with pulmonary edema and superimposed infectious process. Despite initial improvement with broad-spectrum antibiotics, she deteriorated rapidly, requiring mechanical ventilation and ICU care.

Initial workup including cultures and serology were negative. The patient had no history of recent cave visits, however further inquiry revealed recent pigeon exposure at home, prompting Histoplasma urine antigen testing, which returned positive. Despite negative HIV immunology, flow cytometry revealed profound CD4+ lymphopenia (99 cells/μL), consistent with idiopathic CD4+ lymphocytopenia, establishing ICL-associated severe pulmonary histoplasmosis as the leading diagnosis.

Treatment with liposomal Amphotericin B was initiated with planned transition to itraconazole. Despite initial improvement, the patient developed progressive multi-organ failure and expired.

This vignette highlights two critical lessons: (1) severe histoplasmosis in HIV-negative patients warrants evaluation for alternative immunodeficiencies, particularly ICL; and (2) bird exposure remains an underrecognized risk factor for endemic mycoses in Puerto Rico.

V-55- Unmasking HIV-Triggered Hemophagocytic Lymphohistiocytosis in an elderly woman presenting with pancytopenia: A Diagnostic Challenge

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HIV prevalence in Puerto Rico is approximately 0.55%, yet atypical presentations and rare complications such as hemophagocytic lymphohistiocytosis (HLH) secondary to HIV alone remain underreported. This rarity poses diagnostic challenges in endemic regions with limited awareness. We present a rare case of HLH in the setting of undiagnosed HIV.

A 62-year-old female presented with a one-week history of mild abdominal pain, malaise, and intermittent fever. Past medical history significant for chronic leukopenia and chronic hypochromic microcytic anemia. She denied chills, nausea, vomiting, diarrhea, dysuria, or hematuria. Initial physical examination was unremarkable: no distress, no jaundice or scleral icterus, soft nontender abdomen, and normal cardiopulmonary findings. Initial laboratory results revealed pancytopenia (WBC 2.15 x10^3/µL, hemoglobin 8.6 g/dL, platelets 87 x10^3/µL) with bandemia (1.6%), elevated acute phase reactants (CRP 8.9 mg/dL, ferritin 9670 ng/mL), triglycerides 261 mg/dL, fibrinogen 239 mg/dL, and yeast on urine analysis. Abdominal ultrasound showed hepatomegaly (17.1 cm). Admitted for suspected dengue-like syndrome, she received IV fluids, fluconazole, doxycycline, and cefepime.

Despite partial response to empiric treatment, deterioration continued, prompting antibiotic escalation to linezolid and meropenem. Extensive infectious workup, including blood cultures, dengue, leptospira, influenza, mycoplasma, and SARS-CoV-2 serologies, were negative. Due to the laboratory and clinical symptomatology, HIV testing was ordered and returned positive. AIDS was confirmed with a CD4+ count of 11 cells/µL and a viral load of 444,000 copies/mL. The HScore was used to assess the probability of HLH differentiated from similar presenting conditions like sepsis, hematologic malignancies, or other inflammatory syndromes; HScore yielded 267 points with a >99% probability of hemophagocytic syndrome (169 points being optimal cut off). Patient was discharged on day 18 with resolution of initial symptoms demonstrating no active bacterial or fungal infections. She was transitioned to oral prophylaxis for opportunistic infections (trimethoprim-sulfamethoxazole, azithromycin, fluconazole) and referred to an HIV outpatient clinic for antiretroviral therapy.

This case highlights underreported atypical HIV presentations without concomitant infections, compounded by cognitive biases like anchoring on endemic infections and premature diagnostic closure. Pathophysiologically, untreated HIV can trigger HLH via cytokine storm and immune dysregulation, even rarely as the sole cause. In HIV-endemic areas, clinicians should maintain a low threshold for early HIV testing and HLH assessment using tools such as the HScore, especially in patients with persistent fever, pancytopenia, elevated inflammatory markers, hypertriglyceridemia, organomegaly, and potential hemophagocytosis. Timely recognition can mitigate diagnostic delays and improve outcomes.

V-56- Marginal Zone Lymphoma with IgG Paraproteinemia: A rare case report

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Introduction

Marginal zone B-cell lymphoma (MZL) is a rare, slow-growing non-Hodgkin lymphoma (NHL) that originates from post-germinal center B-cells and can involve the bone marrow. It may present as asymptomatic, but can later cause fatigue, fever, weight loss, and signs of anemia or thrombocytopenia as the disease progresses. MZL accounts for about 5–10% of NHL cases in the U.S. and may lead to cytopenias, infections, autoimmune disorders, secondary cancers, and in rare cases organ infiltration. Diagnosis is established with blood studies, bone marrow biopsy, imaging (PET/CT), and flow cytometry. Typically, treatment includes rituximab, chemotherapy, or radiation, all associated with potential side effects such as immunosuppression, infertility and infection.

Case Description

An 86-year-old male with hypertension, prior NHL treated with chemoradiation, and prostate cancer treated with radiotherapy and chemotherapy was referred for incidental detection of IgG kappa monoclonal paraproteinemia. Laboratory evaluation revealed an M-protein spike and an elevated kappa/lambda light chain ratio. Imaging showed no hypermetabolic lesions; however, bone marrow biopsy confirmed infiltration by a small B-cell lymphoma consistent with MZL, with nodular lymphoid infiltrates.
Immunophenotyping showed negativity for CD10, CD5, and CD11c. Given the absence of symptoms or radiographic disease, the patient was managed conservatively with active surveillance.
During follow-up, he developed profound thrombocytopenia and normocytic anemia in the setting of influenza infection. Hospitalization was required for platelet transfusions and evaluation for immune thrombocytopenia and hemolytic anemia. Due to persistent cytopenias, repeat bone marrow biopsy was performed, again showing small B-cell lymphoma with CD20 and CD138 expression. The patient was started on weekly rituximab for four weeks, which led to progressive normalization of platelet counts and hemoglobin levels. He remains in clinical and hematologic remission under surveillance, with maintenance rituximab considered for relapse.

Discussion

Here we present a case of an incidental finding of an abnormal M-spike, increased IgG kappa type monoclonal paraproteinemia, and a BM biopsy positive for MZL with nodular lymphoid infiltrate. Although M-spikes are most commonly associated with monoclonal gammopathy of undetermined significance (MGUS) or multiple myeloma, a subset of patients with MZL can produce monoclonal paraproteins, predominantly of the IgM isotype, followed by IgG. In addition, the presence of monoclonal paraprotein has shown to have prognostic relevance. According to a 2023 retrospective study, patients who had M-protein at the time of diagnosis had an inferior progression free survival when compared to patients who did not have M-protein. However, most published studies and case reports have focused on patients with IgM paraproteinemia. To our knowledge, this is the first

reported case specifically detailing IgG paraproteinemia in a patient with MZL. This underscores the importance of routinely assessing paraproteinemia in patients with MZL to help build a more comprehensive understanding of its potential prognostic implications. This case highlights a rare association between IgG paraproteinemia and MZL, underscoring the potential prognostic value of paraprotein screening in lymphomas beyond the commonly studied IgM subtype.

V-57- From ACS to Stable Ischemia: Management Dilemmas in Anomalous Left Main from the Right Coronary Cusp — A Three-Case Series

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Anomalous origin of the left main coronary artery (LMCA) from the right coronary cusp is a rare congenital finding associated with variable ischemic risk, sudden cardiac death, and complex clinical decision-making. While prior reports have focused on anatomical course classification (e.g., benign vs malignant), real-world presentations often involve discordance between symptoms, functional testing, and coronary anatomy. Additional factors—such as age, ventricular function, and coexisting atherosclerosis—further complicate individualized care.

We present three adult patients with anomalous LMCA arising from the right coronary cusp, each with distinct ischemic presentations: (1) a 59-year-old male with NSTEMI and heart failure with reduced ejection fraction (EF 20%), found to have severe three-vessel coronary artery disease (CAD); (2) a 66-year-old female with exertional angina and anterior ischemia on myocardial perfusion imaging (MPI), with 50% proximal LAD stenosis; (3) a 62-year-old female with dyspnea and mixed chest pain, with anterolateral ischemia on MPI and non-obstructive CAD. Coronary CTA confirmed anomalous LMCA origin from the right coronary cusp in all three patients, each demonstrating varying grades of interarterial course. Cases were discussed with a cardiothoracic specialist, currently under evaluation for surgical repair versus reimplantation. Management decisions remain individualized and unresolved, as discussed with patient and surgery services.

This series highlights the clinical and diagnostic variability associated with anomalous LMCA from the right coronary cusp. Unlike prior reports emphasizing anatomical classification, our cases illustrate the challenges of integrating ischemic symptoms, noninvasive testing, and coronary imaging to guide therapy. The lack of functional-anatomic concordance complicates risk stratification and management planning. Given the rarity of this anomaly, shared decision-making is often hindered by limited clinical experience and evidence. This series underscores the importance of combining functional and anatomical modalities when anatomical anomalies coexist with variable ischemic profiles.

Anomalous LMCA from the right coronary cusp can present across a wide ischemic spectrum. In the absence of definitive anatomical or functional triggers for intervention, a multidisciplinary and individualized approach remains essential.

V-58- The Ace of Spades of Acute Coronary Syndrome: A Case of Yamaguchi Syndrome Mimicking NSTEMI

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Apical hypertrophic cardiomyopathy (ApHCM), or Yamaguchi syndrome, is a rare variant of hypertrophic cardiomyopathy characterized by localized thickening of the left ventricular apex and the classic “Ace-of-Spades” ventricular contour on imaging. Although first described in Japanese populations, it can rarely present in other ethnicities and often mimics acute coronary syndrome (ACS) with chest pain, elevated cardiac biomarkers, and ischemic ECG changes, leading to potential misdiagnosis. We report the case of a 49-year-old Hispanic male with hypertension who presented with substernal chest pain radiating to the back, diaphoresis, deep T-wave inversions in V3, V4, and elevated high sensitivity troponin levels, prompting a diagnosis of non-ST-elevation myocardial infarction and initiation of ACS protocol. Coronary angiography revealed no obstructive disease, while ventriculography and echocardiography demonstrated apical hypertrophy with preserved ejection fraction and spade-like morphology, confirming ApHCM. Troponin elevation was attributed to apical wall stress rather than ischemia. The patient was transitioned to beta-blocker therapy with outpatient cardiology follow-up, and no complications arose during hospitalization. This case emphasizes the need to consider ApHCM in ACS presentations with non-obstructive coronaries to prevent unnecessary interventions and to guide appropriate management.

V-59- Idiopathic Aplastic Anemia in Three Young Adults with Environmental Exposure Histories

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Introduction:

Aplastic anemia (AA) is a rare bone marrow failure disorder characterized by pancytopenia and hypocellular marrow. This immune dysregulation is thought to be T-cell–mediated destruction of hematopoietic progenitors and hematopoietic stem cell transplantation (HSCT) remains the only curative therapy. Herein, we consider three patients presented with classic AA signs (petechiae, bruising, and pancytopenia) with environmental exposure to pesticides, solvents, paints, and glues.

Case Presentation:

A 39-year-old male with a 15-year history of agricultural work involving paraquat, glyphosate, and other herbicide exposure presented with petechiae, purpura, fatigue, and dyspnea. He had no recent infectious symptoms or travel. His viral panel (HIV, Parvovirus B19, CMV and EBV) was negative. There was no history of autoimmune disease. Bone marrow biopsy revealed hypocellularity, an abnormal T-cell population, and dysregulated myeloid maturation. For treatment he underwent sibling-donor transplant with horse AntiThymoGlobulin(ATG) and high-dose-cyclophosphamide. Sadly, he died from unrelated trauma.

A 30-year-old male with 12-year history as a hairstylist was initially admitted with petechiae and thrombocytopenia thought to be due to dengue. As other blood cell lines declined, a bone marrow biopsy confirmed aplastic anemia. The patient background included childhood exposure to chemicals, paints and secondhand smoke, and most recently cypermethrin. History of substance use includes using marijuana, opioids, amphetamines, and anabolic steroids. Viral testing was negative. Family history was notable for colon cancer (maternal grandfather) and leukemia (paternal uncle). Treatment consists of haploidentical HSCT with rabbit ATG, fludarabine, cyclophosphamide, and Total-Body-Irradiation.

A 27-year-old female pharmacy technician presented with progressive easy bruising following a flu-like illness with fever, sore throat, productive cough, and no gastrointestinal or urinary symptoms. The patient was nulligravid, not sexually active or on hormone therapy and denied any history of drug use, pets, autoimmune disease, or family history of malignancy. Most recent travel was to the northeast of the USA with no illnesses reported afterward. Occupational exposure included chemical compounding in her pharmacy and paints. Viral testing was negative. Treatment consisted of haploidentical HSCT with rabbit ATG, fludarabine, cyclophosphamide, and Total-Body-Irradiation.

Discussion:

All three patients were under 40 years old and tested negative for common viral etiologies and underwent successful allogeneic marrow transplantation from first-degree relatives. None had known autoimmune conditions or genetic disorders predisposing to bone

marrow failure. Each patient had potential environmental exposures, including pesticides, solvents, and pharmacologic agents.

The pathophysiology is thought to be T-cell–mediated destruction of hematopoietic progenitors and the states that autoantibodies targeting moesin (~46% prevalence) and DRS1 (~38% in HLA DR15+ patients) have mechanistic evidence for inflammatory or cytotoxic potential in vitro, suggesting they may amplify marrow suppression. Anti-kinectin antibodies destroy hematopoietic cells via TNF-α secretion through an ERK1/2-Dependent Pathway. The literature has shown pesticides and other occupational chemicals to potentially act on these mechanisms. resulting in hematopoietic suppression in mice.

Herein, idiopathic AA occurring in the context of environmental exposures is described and the efficacy of tailored HSCT protocols, the importance of gathering exposure histories and awareness of novel antibodies that may serve as biomarkers and potential treatment options is highlighted.

V-60- Unmasking the Culprit: Gliptin-Induced Bullous Pemphigoid in a Patient with Type 2 Diabetes Mellitus

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Introduction:
Bullous pemphigoid (BP) is a chronic autoimmune blistering disorder, primarily affecting older adults, that presents with tense bullae and erosions on the skin. Due to its variable presentation, BP can mimic other dermatologic conditions such as eczema, contact dermatitis, or cellulitis, often leading to delayed or incorrect diagnoses in primary care. An increasingly recognized cause of drug-induced BP is the use of dipeptidyl peptidase-4 (DPP-4) inhibitors (commonly known as gliptins), a class of oral antidiabetic agents frequently prescribed for the management of type 2 diabetes mellitus. Awareness of this association is essential for early identification and appropriate treatment.

Case Description:
We present the case of a 52-year-old obese female with multiple comorbidities, including type 2 diabetes mellitus, hypertension, and hyperlipidemia. Her diabetes had been managed with Jentadueto (linagliptin/metformin) for several years. She developed recurrent pruritic, blistering skin eruptions on her extremities and trunk. Initially, her primary care provider attributed the lesions to common skin conditions and treated her empirically with topical corticosteroids, oral antibiotics, and antihistamines without significant improvement.
As the condition worsened, dermatologic evaluation was pursued. Physical exam revealed tense bullae and erosions on the bilateral upper and lower extremities and trunk. A comprehensive review of her medication history raised suspicion for DPP-4 inhibitor–induced BP. She was prescribed topical betamethasone and oral antihistamines, and discontinuation of Jentadueto was recommended. BP antigen 1 and 2 serologies were ordered.

A skin biopsy performed at that time confirmed the diagnosis of medication-induced bullous pemphigoid. Jentadueto was discontinued, and the patient was transitioned to Synjardy (empagliflozin/metformin) for glycemic management. Subsequent follow-up demonstrated near-complete resolution of skin lesions and symptoms.

Discussion:
This case highlights the importance of recognizing medication-induced BP in patients with type 2 diabetes on gliptin therapy. Because primary care providers are often the first to evaluate new skin complaints, a high index of suspicion is necessary. Delays in diagnosis can lead to patient morbidity, unnecessary treatments, and prolonged disease course.

Conclusion:

In diabetic patients receiving DPP-4 inhibitors, new-onset blistering skin lesions should prompt evaluation for bullous pemphigoid. Early recognition, discontinuation of the offending agent, and interdisciplinary collaboration can lead to prompt resolution and improved patient outcomes.

V-61- A Rare Duet: Simultaneous Onset of Reactive Arthritis and Erythema Nodosum

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Reactive arthritis (ReA) is an inflammatory spondyloarthropathy most commonly preceded by gastrointestinal or genitourinary infection. While cutaneous manifestations such as keratoderma blennorrhagicum or circinate balanitis are characteristic, the concomitant association with erythema nodosum (EN) can be seen as well, especially after a bacterial infection. This report presents the case of a patient who developed ReA and EN simultaneously, an association that is documented in clinical literature.

We present the case of a 24-year-old female patient who came to the emergency department with an erythematous rash on both legs after returning from a trip to the Dominican Republic. She reported experiencing a cough during the trip and completed a 3-day course of azithromycin, which resolved the symptoms. Eight days before admission, the patient began experiencing episodes of dry cough. The following day, she developed an erythematous rash on both legs. The next day, she noticed swelling of the tonsils and had difficulty swallowing. A subjective fever was also reported. Later that evening, she visited another emergency department, but a definitive diagnosis was not established. A urinalysis revealed evidence of a urinary tract infection (UTI), for which ciprofloxacin was initiated. Dexamethasone was also prescribed and partially relieved her symptoms. On the day of admission to our facility, the patient reported articular pain in the ankles, knees, hips, and cervical spine. She denied chest pain, shortness of breath, palpitations, abdominal pain, diarrhea, nausea, vomiting, suprapubic pain, and dysuria. On physical examination, there were erythematous lesions on both legs, which were extremely tender to palpation. Warm, erythematous effusions with crepitus were noted in the left knee and right ankle. Laboratory results on admission showed marked leukocytosis with neutrophilic predominance. Urinalysis confirmed a UTI. A pregnancy test and COVID-19 molecular test were negative. Syphilis serology, Neisseria gonorrhea, and Chlamydia trachomatis were non-reactive. ESR and CRP were elevated. CPK, magnesium, phosphorus, and uric acid levels were within normal limits. Chest X-Ray revealed no hilar adenopathy or any acute cardiopulmonary process. Ciprofloxacin and steroid therapy were started. During hospitalization, leukocytosis gradually resolved, and blood and synovial cultures were negative after five days. By the fifth day, the patient reported improvement in pain. Rheumatology and Dermatology services evaluated the patient and determined that the skin lesions were erythema nodosum, and that articular symptoms corresponded to reactive arthritis. She was discharged with systemic and topical steroids and scheduled outpatient follow-up.

This case highlights that after an infectious process, EN and ReA can have a simultaneous delayed presentation. Clinicians should consider ReA in the differential diagnosis of EN, particularly when accompanied by articular symptoms. It is important to identify infectious causes and rule out rheumatologic conditions, through history, physical exam, and laboratory tests, to better understand the etiology that could explain these delayed immunologic manifestations. Recognizing this association is crucial to avoid diagnostic delay and ensure appropriate management.

V-62- Myxofibrosarcoma in a Patient with Chronic Gout: A Diagnostic Challenge

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Introduction:

Myxofibrosarcoma (MFS) is a rare, aggressive malignancy that commonly involves the extremities of elderly patients. It grows in an infiltrative pattern along fascial planes and carries a high risk of local recurrence and distant metastasis. Because of its nonspecific clinical and radiographic features, MFS can mimic other conditions and delay diagnosis. Chronic tophaceous gout, characterized by deposition of monosodium urate crystals forming subcutaneous tophi, may mimic neoplastic processes both clinically and radiographically. This case highlights a patient with longstanding gout whose MFS was initially misdiagnosed as a gouty tophus, underscoring the importance of vigilance in differentiating soft tissue masses.

Case Description:

We present the case of a 60-year-old male with a 20-year history of gout, managed with febuxostat, colchicine, and diclofenac. He developed an enlarging lesion at the right elbow in 2019, which ulcerated and drained a “ground meat-like" material and became warm to the touch. Only plain radiographs were obtained before excision, revealing a nonspecific soft tissue lesion. Initial pathology suggested gout, but further review identified high-grade MFS in a background of gouty tophi. The largest nodule measured 7.8 × 5.5 × 3.8 cm.

Staging MRI demonstrated enhancing nodular collections in the posterior elbow and regional metastases in the distal upper arm. PET-CT revealed several intensely FDG-avid right axillary lymph nodes, as well as soft tissue densities in the right elbow and proximal right upper extremity indicative of biopsy-proven malignancy with local extension. The patient underwent a right forequarter amputation with axillary lymph node dissection; tumor was present in 10 out of 15 regional lymph nodes, although margins were negative. One month later, biopsy of a lung nodule confirmed undifferentiated high-grade sarcoma. Despite two cycles of doxorubicin, he developed septic shock and died shortly after.

Discussion and Conclusion:

This case illustrates the diagnostic challenges of distinguishing gouty tophi from MFS. Both can present as soft tissue masses and cause bone erosion or changes in nearby bone structures, leading to delays in management. Sarcomas, including MFS, have been reported in association with gouty tophi, possibly due to inflammatory stress and the formation of reactive oxygen species (ROS).

Atypical or enlarging lesions in patients with gout should not automatically be assumed benign. Radiographs alone lack specificity; a stepwise approach including ultrasonography, MRI, and early tissue sampling is critical. Ultrasound-guided fine needle aspiration (FNA) provides minimal discomfort for the patient, confirms diagnosis, and facilitates timely oncologic referral.

In conclusion, MFS can masquerade as gouty tophi, leading to delayed recognition and adverse outcomes. Clinicians should maintain suspicion for malignancy in atypical cases and pursue advanced imaging and biopsy when indicated to ensure accurate diagnosis and timely management.

V-63- Rare Case of Terminal Ileum Adenocarcinoma Presenting As Cecal Bascule

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Cecal bascule is an uncommon presentation of Cecal volvulus, characterized by folding of the Cecum anteriorly and superiorly upon itself, without axial twist. It can result in large bowel obstruction, ischemia, perforation and sepsis. Cecal bascule is a surgical emergency that has a 1-2% incidence of all Cecal volvulus cases, mostly seen in the females less than 60 years old and pregnant women.

An 87-year-old female patient with past medical history of hypertension, AAA, CAD and left extremity PAD presented to the Emergency Department complaining of black and bloody stools, dizziness and weakness of 6 days evolution. Physical examination was remarkable for a soft, depressible, non-distended abdomen, non-tenderness to palpation and normoactive bowel sounds. Radiologic imaging at the time of admission was non-diagnostic. On day 10 after admission patient started to complain of constipation with severe abdominal pain. Physical examination was remarkable for a distended abdomen with hyperactive bowel sounds, severe tenderness to palpation and hyper tympanic sounds in the epigastric area. Radiologic imaging was diagnostic of Cecal bascule. Emergent exploratory laparotomy with Right hemicolectomy with ileo-colonic anastomosis was performed. During the surgery Cecum was observed severely distended and ischemic, located in the epigastric area of the abdomen. Incidentally a hard, concentric, ill-defined mass was noted at the terminal ileum, which served as the lead point for Cecal bascule development. In addition, multiple liver lesions were noted. Biopsies were sent for pathology evaluation, which came back positive for metastatic terminal ileum adenocarcinoma. The procedure was well tolerated by the patient and then transferred to the ICU. On the first day after the procedure patient started to present hypotension with tachycardic episodes. Despite aggressive multidisciplinary treatment, patient expired on the third day after procedure.

Cecal Bascule in an elderly frail patient, should raise high suspicious for malignancy. It is a rare presentation of large bowel obstruction that can lead to serious complications if not promptly diagnosed and managed.

V-64- Unusual Presentation of Extranodal Marginal Node Lymphoma in the Thyroid

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IntroductionLymphomas exist as Hodgkin and Non-Hodgkin(NHL) types. NHL has different subtypes derived from clonal B, T, or NK cells. Marginal-zone lymphomas originate from post-germinal center B-cells and can present as an extranodal marginal-zone lymphoma(EMZL) of mucosa-associated lymphoid tissue(MALT). These lymphomas express B-cell markers such as CD19, CD20, and CD22, but lack CD5, CD10, and CD23. EMZL represents an indolent form of NHL with the ability to differentiate into the marginal zone. It arises in the stomach, salivary glands, lungs, and ocular adnexa. However, thyroid involvement is uncommon.Case SummaryA 66-year-old male with hypothyroidism presented with an incidental thyroid mass. Fine-needle-aspiration(FNA) on 01/23/24 suggested chronic thyroiditis. On 02/26/24 he went to the OR for a right hemithyroidectomy. The surgeon found a large, friable mass attached to surrounding structures. Pathologic evaluation revealed EMZL positive for CD20, CD23 and dendritic cells. Margins were disrupted with lymphoma involvement of perithyroidal adipose tissue. PET-CT scan on 4/23/25 did not show evidence of malignancy outside of the surgical bed, making him Ann-Arbor Stage-1aE. He was treated with field radiation therapy. Unlike most EMZL cases, this patient had no infectious predisposing factors. Discussion NHL pathogenesis involves the accumulation of genetic alterations in proto-oncogenes and tumor suppressor genes which are generally linked to chronic antigenic stimulation(CAS) by infections or autoimmune processes. It's unusual for EMZL to present in thyroid and absence of CAS. The diagnosis was performed through cytology, immunohistochemistry, and genetic analysis of the surgical biopsy sample after FNA failed detection of lymphoma. Management options for non-gastric EMZL include surgery, radiotherapy, and chemotherapy. The patient had good response to treatment and remains in remission.ConclusionThyroid EMZL is a rare and often overlooked on cytology and definitive diagnosis requires histopathology and immunophenotyping. This case underscores the importance of surgical biopsy in suspicious thyroid lesions.

V-65- Agitation and Psychosis—A Teratoma in Disguise: A Missed Diagnosis of Autoimmune Encephalitis

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University of Puerto Rico Program

INTRODUCTION:

Autoimmune encephalitis is the third most common cause of encephalitis after viral etiologies. In young females, ovarian teratoma–associated anti-NMDA receptor encephalitis is particularly likely, as tumor-derived antibodies target neuronal surface antigens. Early manifestations—agitation, psychosis, and catatonia—can mimic drug intoxication. Failure to maintain a broad differential or initiate timely evaluation can delay diagnosis and worsen outcomes.

CASE

A 24-year-old Hispanic female presented with bilateral temporal headaches that progressed over five days to dizziness and disorientation. She was initially treated with morphine for pain, after which she developed erratic, aggressive, and non-communicative behavior. These symptoms were attributed to opioid effects following a positive toxicology screen after morphine administration. Two days later, her mother left against medical advice and sought care at a supra-tertiary hospital.

On evaluation, the patient was suspected of having encephalitis. Examination revealed negative Brudzinski and Kernig signs, catatonia, intermittent tremors, involuntary movements, and hyperreflexia in the right lower extremity; she was awake but not engaging or following commands. Labs showed leukocytosis with neutrophilia and reactive thrombocytosis. Lumbar puncture revealed clear CSF with 144 WBCs (98.7% mononuclear), glucose 57 mg/dL, protein 55.5 mg/dL, negative Gram stain, and opening pressure 39 cmH_2O. Brain CT and MRI were unremarkable. Autoimmune workup including anti-NMDA receptor antibodies were sent, and empirical antibiotics and antivirals were initiated.

The following day, she deteriorated neurologically and developed status epilepticus, requiring intubation, ICU transfer, and anticonvulsant therapy. Subsequent imaging identified a right ovarian teratoma, later confirmed by pathology, and anti-NMDA receptor antibodies returned positive, establishing the diagnosis. Management included high-dose methylprednisolone followed by prednisone, Rituximab and surgical removal of the teratoma. She continues to receive care for seizure control and neurologic optimization.

DISCUSSION:

Rapid-onset psychiatric symptoms with neurological signs, including catatonia or seizures, can mimic drug intoxication, risking misdiagnosis without thorough history and examination. This case underscores the importance of a broad differential for altered mental status, including meningitis, viral or autoimmune encephalitis, and metabolic causes. Negative imaging may falsely support a drug-related hypothesis; however, up to

50% of autoimmune encephalitis cases can present with normal MRI. Viral encephalitis may also trigger secondary autoimmune processes in up to 30% of patients. Autoimmune encephalitis predominantly affects young adult females, highlighting the need to consider it early. Early recognition and systematic evaluation could have prevented complications such as status epilepticus and ICU admission. This case emphasizes the need for vigilance and timely workup in young patients with acute neuropsychiatric symptoms.

CONCLUSION:

Rapid-onset neuropsychiatric symptoms in this young female were initially misattributed to drug intoxication, delaying recognition of anti-NMDA receptor encephalitis and leading to ICU admission for status epilepticus. Internists, as frontline physicians, must maintain a high index of suspicion in young females with acute psychiatric and neurological symptoms, avoiding premature closure on presumed intoxication and considering autoimmune encephalitis, where early recognition can be lifesaving.

V-66- Primary CNS Lymphoma Presenting as Multifocal Brain Lesions in a Solid Organ Transplant Recipient

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Primary central nervous system lymphoma (PCNSL) is a rare, aggressive extranodal non Hodgkin lymphoma with a predilection for immunocompromised individuals, including solid organ transplant recipients. Its clinical manifestations are often nonspecific, ranging from neurocognitive decline to focal neurological deficits, making early diagnosis challenging. This case describes a liver transplant recipient who presented with progressive neurological symptoms and was ultimately diagnosed with aggressive multifocal PCNSL.

This is the case of a 72-year-old male with a past medical history of liver transplantation (five years prior), type 2 diabetes mellitus, and depression. He had no known drug allergies, denied alcohol and tobacco use, and consumed one cup of coffee daily. The patient was brought to the Emergency Department by his daughter, who reported several weeks of gait disturbance, intermittent disorientation, and generalized weakness. Initial workup, including neuroimaging, revealed multiple intracranial lesions concerning for metastatic disease. MRI confirmed multifocal brain lesions. A systemic metastatic evaluation was negative.

Neurosurgery was consulted, and given the accessibility of a frontal lesion, the patient underwent a craniotomy with resection of the mass. The procedure was uncomplicated, and the patient remained neurologically intact postoperatively. However, additional non-surgical lesions were identified, including those in the brainstem. During hospitalization, he developed acute decline in mental status. The differential included metabolic encephalopathy and progression of the nonresectable brainstem lesion. A follow up CT scan revealed expected postoperative changes with mild edema but no hemorrhage, hydrocephalus, or acute ischemia. Pathological examination of the resected specimen demonstrated aggressive primary CNS lymphoma.

Oncology consultation determined that, given the multifocal involvement and overall prognosis, no further aggressive interventions were appropriate. The patient's daughter and caregiver were engaged in detailed goals-of-care discussions, ultimately electing for comfort-focused management. The patient already had a DNR/DNI order in place. Staples were removed at the bedside without complication, and he was discharged with recommendations for outpatient follow-up with oncology, internal medicine, and neurology, alongside palliative and hospice care.

This case illustrates the diagnostic complexity of multifocal brain lesions in patients with prior solid organ transplantation. In such populations, differential diagnoses extend beyond metastatic disease and include opportunistic infections, posttransplant lymphoproliferative disorders, and primary CNS lymphoma. Histopathologic confirmation is essential to establish the diagnosis and guide management. While high dose methotrexate based chemotherapy and whole brain radiotherapy remain standard treatments for PCNSL, therapeutic options may be limited in patients with multiple

comorbidities and poor functional reserve. In this case, the patient's immunosuppressed state and multifocal involvement contributed to the decision against further aggressive therapy.

In conclusion, PCNSL should remain a key consideration in transplant recipients presenting with multifocal brain lesions. This case underscores the importance of multidisciplinary collaboration and timely pathology confirmation, while also highlighting the critical role of patient decision making and palliative care in managing advanced neurooncological disease.

V-67- Surviving the Unthinkable: Necrotizing Pancreatitis in the Setting of Diabetic Ketoacidosis

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Acute pancreatitis (AP) is an uncommon but serious complication of diabetic ketoacidosis (DKA), most often mediated by severe hypertriglyceridemia. Population-based studies show that individuals with diabetes have a 1.7-fold higher risk of AP than non-diabetics, and those with prior hyperglycemic crises such as DKA have more than a six-fold increase in risk. Conversely, AP itself confers a two-fold increase in subsequent diabetes, underscoring a bidirectional relationship. Although hypertriglyceridemia accounts for only 1–4% of AP cases in the general population, it becomes a leading cause in DKA, with reported triglyceride levels frequently exceeding 1,000 mg/dL and sometimes >2,000 mg/dL in severe cases. Necrotizing pancreatitis remains rare in this setting—reported in <2% of published series—but carries a mortality rate of 15–30%, rising further with infected collections or multi-organ failure. We report a catastrophic case of necrotizing pancreatitis complicating DKA in the setting of severe hypertriglyceridemia to highlight this high-risk intersection and its management implications.

A 38-year-old woman with type 2 diabetes mellitus (BMI 35 kg/m^2) on sulfonylurea, untreated hyperlipidemia, hypertension, and hypothyroidism presented with five days of worsening abdominal pain, nausea, and vomiting. On arrival she was tachypneic with Kussmaul respirations and rapidly deteriorated, requiring intubation for acute respiratory distress syndrome. Laboratory evaluation revealed severe metabolic acidosis (HCO_3^- 18.1 mmol/L; anion gap 23.2; marked ketonemia), triglycerides >2,625 mg/dL, lipase 7,406 U/L, amylase 563 U/L, leukocytosis 18,000/µL, profound hypocalcemia (4.0 mg/dL), and coagulopathy (INR 11.0). Contrast-enhanced CT demonstrated extensive peripancreatic collections and diffuse nodular peritoneal infiltration consistent with necrotizing pancreatitis. Her ICU course was complicated by abdominal compartment syndrome (intra-abdominal pressure 22 mmHg), necessitating emergent decompressive laparotomy with temporary open abdomen, acute kidney injury requiring hemodialysis, paralytic ileus, and segmental pulmonary embolism. With aggressive metabolic resuscitation, surgical and anticoagulation interventions, and multidisciplinary critical care, she gradually improved and was discharged home.

This case exemplifies the “perfect storm” where DKA, severe hypertriglyceridemia, and pancreatitis amplify each other’s pathophysiology. Insulin deficiency and counter-regulatory hormones accelerate lipolysis, producing extreme triglyceride elevations; free fatty acids generated within pancreatic capillaries then trigger and perpetuate acinar cell injury. Once necrotizing pancreatitis develops, complications such as abdominal compartment syndrome, renal failure, and thromboembolism drive mortality rates into the 20–30% range. Current guidelines recommend a high index of suspicion for AP in any DKA patient with abdominal pain and hypertriglyceridemia, prompt imaging, aggressive insulin and fluid therapy to lower triglycerides, and early multidisciplinary involvement. Adjunctive therapies such as early plasmapheresis may be considered in refractory or very severe cases based on emerging evidence. Our patient’s survival underscores the importance of timely recognition, protocol-driven care, and coordinated critical care teams. Early recognition of this triad and rapid, standardized management may avert multi-organ failure and reduce mortality.

V-68- Pulmonary Sequestration: The Role of Imaging and Surgical Intervention. A Case Report

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Pulmonary sequestration is a congenital lung abnormality where a portion of lung tissue develops abnormally and lacks connection with the bronchial tree. It receives its blood supply from the systemic circulation rather than the pulmonary arteries. The affected lung tissue is abnormally perfused but unventilated and non-functioning(1). Incidence is around 0.15% to 6.4% of all congenital lung malformations making this a rare diagnosis.

A 30-year-old woman presented to ED with productive cough and dyspnea. Labs showed Leukocytosis. Chest X-Ray and CT showed right lower lobe consolidation with cavitations(2,3). Initially, the diagnosis of Pneumonia with Abscess was made. She was discharged with Levofloxacin 750mg POqdx7d, Mucinex, and outpatient follow-up with Pneumologist. Three months later, patient returned with a recurrent episode presenting same symptoms. Empirical antibiotics were started (Piperacillin-tazobactam 3.3gIVq6h, Linezolid 600mg/300mlIVq12h, Cefepime 2gIVq8h, and Metronidazole 500mg/100mlIVq6h). Interventional Radiology was consulted for a Intrathoracic Pleural Catheter placement. Successful U/S-guided placement of right chest tube drained 200ml fluid which showed a Glucose of 4mg/dL, consistent with Empyema. Chest fluid culture did not grow any organism, but Sputum culture grew Pseudomonas aeruginosa with broad susceptibility, and completing clinical criteria for Sepsis. A CT scan with IV contrast revealed an aberrant connection between the descending thoracic aorta and a segment of the right lower lobe(4) suspecting a pulmonary sequestration. Cardiothoracic surgery was consulted for a decortication procedure. Open thoracotomy revealed a fibrino-purulent empyema encasing all the right lung, with massive atelectasis secondary to intrabronchial secretions. This finding was the reason of the poor lung insufflation. Additionally, a 5mm diameter arterial supply from left descending thoracic aorta, is seen crossing over the vertebral bodies and supplying the right lower lobe(4). This confirmed the presence of an intralobar sequestration with the lesion located within the normal lobe. Statistically, 75% of blood supply from pulmonary sequestrations originate from the thoracic or abdominal aorta, and the intralobar variety accounts for 75% of all cases2,4,5. Following surgical intervention, the patient improved dramatically with resolution of leukocytosis after two days, and antibiotic regimen was continued. On day 11, Empyema cultures showed late growth of gram-negative Achromobacter xylosoxidans with broad susceptibility including the antibiotics the patient was already on. The patient remained symptom-free, with O2Sat 98%, and no complications. Chest tube was removed successfully, and patient was discharged. A ChestX-ray was done on outpatient follow-up showing a healed scar without cavitations and good lung expansion(5).

This case illustrates the presentation of pulmonary sequestration, highlighting the important use of imaging, and the roll of cardiothoracic surgery. Although rare, early recognition of this defect is crucial due to its potential for rapid progression into Sepsis. For complete resolution and to preserve normal lung tissue function, prompt surgical management is essential.3

Research Abstracts

R-01

Activation of STAT1/2 Signaling Pathways by Polysaccharide Peptide in Jurkat T-Cells Highlights a Novel Immunomodulatory Strategy Against HIV

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Despite major advances in antiretroviral therapy (ART), HIV remains a global health challenge. At the end of 2024, an estimated 40.8 million people were living with HIV, yet only 31.6 million (≈77%) had access to ART, leaving nearly one in four untreated. Furthermore, about 13% of people with HIV worldwide, roughly 5.3 million individuals, remain undiagnosed (corresponding to 1/8 individuals unaware of their HIV status). In Puerto Rico, historical reports indicated that 1 in 11 people living with HIV were unaware of their infection, underscoring persistent gaps in diagnosis and care. These limitations highlight the need for adjunctive therapies that can complement ART by enhancing antiviral immunity and preventive strategies. Polysaccharide peptide (PSP), derived from Coriolus versicolor, has shown immunomodulatory and antiviral effects. In our previous work, PSP restricted HIV-1 entry by ~73% in THP-1 monocytes, implicating its role in innate immunity. Building on these findings, we now explore PSP's effects on adaptive immunity, focusing on STAT1/2 and PKR pathways in Jurkat T-cells to determine its future as a potent, non-toxic, anti-HIV therapeutic treatment in combination to current antiretroviral therapies. Methods: Jurkat T-cells were treated with PSP (50–1000 μg/mL) for 3 and 6 days. Cell viability was assessed by MTT assay. Protein expression and phosphorylation of STAT1, STAT2, and PKR were analyzed by Western blot, and transcript levels by RT-qPCR. Cell viability data were analyzed by non-linear regression (dose–response, variable slope) in GraphPad Prism 10.0. Western blot and RT-qPCR data were analyzed by one-way ANOVA with Tukey's post hoc test under Gaussian distribution and equal variance assumptions. Results represent mean ± SEM from ≥3 independent experiments, with significance set at $p < 0.05$.

Results:
PSP induced robust, dose-dependent STAT signaling without significant toxicity. STAT1 and STAT2 phosphorylation increased (+1.3 to +3.3-fold across doses) at both 3 and 6 days, with the strongest activation at higher concentrations. These protein changes were mirrored by transcript upregulation (+1.4–1.6-fold at day 3; +2.1–+2.3-fold at day 6), confirming regulation at both transcriptional and post-translational levels. PKR expression was also enhanced (+1.2 to +2.8-fold) at mRNA and protein levels, indicating activation of an antiviral sensor pathway. Collectively, PSP consistently upregulated STAT1/2 and PKR in a dose- and time-dependent manner. Conclusion: PSP significantly enhances STAT1, STAT2, and PKR signaling in Jurkat T-cells, underscoring its role in activating adaptive immune responses. Together with prior evidence of HIV-1 restriction in THP-1 cells, these findings highlight PSP as a promising adjunctive immunomodulator with therapeutic potential against HIV.

R-02

Implementing a pharmacogenomic driven algorithm to guide antiplatelet therapy among Caribbean Hispanics: a non-randomized clinical trial

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This study aimed to determine whether using a genotype-guided clinical decision support (CDS) algorithm for selecting oral antiplatelet therapy could reduce the rate of major adverse cardiovascular and cerebrovascular events (MACCEs) in Caribbean Hispanic patients six months after undergoing percutaneous coronary intervention (PCI). Conducted as an open-label, multicenter, non-randomized clinical trial, the study involved 300 Caribbean Hispanic patients—both male and female—who were on clopidogrel and had received PCI due to acute coronary syndromes, stable ischemic heart disease, or documented extracardiac vascular conditions. The trial took place across eight public and private secondary and tertiary care hospitals in Puerto Rico. Participants were divided equally into two groups: a standard-of-care (SoC) group and a genotype-guided pharmacogenetic (PGx-CDS) group. Stratification was done using risk scores derived from a CDS algorithm that incorporated individual platelet function, genetic profiles, and clinical and demographic data to guide treatment decisions. In the PGx-CDS group, patients with a high-risk score (≥2) were recommended ticagrelor, while others remained on or were de-escalated to clopidogrel. This intervention occurred within 3–5 days post-PCI, and medication adherence was also assessed. The primary outcome was the rate of MACCEs, while secondary outcomes included bleeding episodes. Kaplan-Meier survival analysis and Cox proportional hazards models were used to examine associations between event-free time and predictors such as treatment group and risk scores. Although the PGx-CDS group showed a lower MACCE rate (8.7%) compared to the SoC group (10.7%), the difference was not statistically significant overall ($p=0.56$; HR=0.56). However, among patients with high-risk scores, the genotype-guided approach significantly reduced the incidence of MACCEs six months after coronary stenting (adjusted HR=0.104; $p<0.0001$). The findings suggest that the PGx-CDS algorithm may offer a significant clinical benefit in high-risk patients, though its effectiveness appears limited in lower-risk groups.

R-03

"Adapt and Survive: Gram-Negative Pathogens Redefining Resistance" A Surveillance Study of Emerging Resistance Mechanisms of Gram-Negative Organisms in Puerto Rico from 2023 to 2025

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Introduction:
Antimicrobial resistance is a significant global public health concern with multidrug-resistant organisms causing approximately 4.95 million associated deaths and 1.27 million directly related deaths in 2019. Projections indicate that by 2050, antimicrobial resistance could be responsible for approximately 8.22 million deaths. Some studies suggest that the highest antimicrobial resistance mortality rates for 2050 are expected in South Asia and in Latin America and Caribbean regions. Previous surveillance studies done in Puerto Rico, have identified Klebsiella Producing Carbapenemase (KPC) and IMP-18 beta-lactamase. Puerto Rico has experienced increasing rates of these pathogens and now is suspected to have cases involving all major classes of beta-lactamases. Nonetheless data has remained limited in our region and evaluation of antimicrobial resistance trends involving multidrug resistant gram negatives is essential.

Methods:
To describe the epidemiological and molecular evolution of antimicrobial resistance in carbapenem-resistant gram-negative organisms in Puerto Rico from January 2023 to April 2025. Retrospective descriptive analysis using surveillance data submitted voluntarily by clinical laboratories across Puerto Rico to the Department of Health. Isolates from hospitalized patients were characterized by species, infection source, and beta-lactamase gene presence. Demographics, geographic distribution, and monthly trends were also reviewed. Molecular resistance mechanisms were confirmed via PCR.

Results:
A total of 692 carbapenem-resistant gram-negative isolates were reported over the study period. Klebsiella pneumoniae consistently represented the majority of isolates annually, with 60.8% in 2023, 65% in 2024, and 64.5% so far in 2025, followed by Pseudomonas aeruginosa and Enterobacter cloacae. The predominant resistance mechanism throughout the reported timeframe was Klebsiella Producing Carbapenemase (KPC), followed by New Delhi metallo-beta-lactamase (NDM). During 2023, OXA-48-like beta-lactamases were also present. By 2024, there was an increase in NDM isolates and OXA-24/40 beta-lactamases emerged. In 2025, NDM continued to rise, and new metallo-beta-lactamases were reported, including VIM and IMP. The most common source of infection was the urinary tract, followed by stool, wound, and sputum. The elderly population represented the most affected demographic group.

Conclusion:

This study demonstrates a progressive rise in antimicrobial resistance across Puerto Rico. While KPC remains the predominant beta-lactamase, the emergence of NDM, OXA-24/40, VIM, and IMP suggest expanding resistance mechanisms, with NDM and VIM reported here for the first time in the region. Given the voluntary nature of reporting, the true burden of multidrug-resistant organisms is likely underestimated. These findings highlight the importance of mandatory reporting of multidrug-resistant organisms to the Department of Health to track resistance patterns and strengthen infection control strategies. Carbapenem-resistant gram-negative infections pose a major challenge due to very limited treatment options. Combined efforts between primary providers and infectious disease specialists are essential to implement targeted antibiotic therapies, enhance antimicrobial stewardship, and mitigate the growing antimicrobial resistance crisis.

R-04

From Outcomes to Action: Establishing Protocols in Acute Pancreatitis Management

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Acute pancreatitis (AP) is a frequent cause of hospitalization, affecting ~40/100,000 annually, with mortality <1% in mild cases but up to 15–30% in moderate–severe disease. Despite American College of Gastroenterology (ACG) guidelines, variation in inpatient management persists. This study describes AP admissions at our institution and evaluates relationships between fluid rate, diet initiation, and length of stay (LOS).

Methods: A retrospective review of San Lucas Hospital EHR (2021–2024) identified 465 AP admissions; 302 met inclusion criteria (≥18 years, ≥2/3 Revised Atlanta criteria, complete records). Exclusions included non-AP diagnoses, non-admissions, and incomplete records. Variables included demographics, BMI, etiology, CT-based severity, surgical interventions, antibiotic use, ICU admission, and inpatient management parameters. Data were entered into REDCap and analyzed with IBM SPSS. Primary outcomes were LOS and correlations with early diet initiation and fluid rate.

Results:
The mean LOS was approximately 6 days. Gallstones were the most frequent etiology (69% female, $p<0.001$), followed by alcohol-induced AP (93% male, $p<0.001$). Of 22 severe cases, 59% (n=13) were necrotizing. Cholecystectomy was performed in 71% (n=88) of gallstone cases. Mild AP was more common in females (56%), whereas moderate–severe cases predominated in males (65–68%, $p<0.05$). Other etiologies included drug-induced, post-ERCP, autoimmune, cancer-related, and idiopathic pancreatitis. In necrotizing AP, 92.3% (n=12) received antibiotics; however, only 69.2% (n=9) were guideline-concordant. Early diet initiation (~1.4 days) was significantly associated with shorter LOS ($p<0.001$). Most patients received moderate ER boluses (~1.5 L) followed by lower inpatient fluid rates, correlating with improved LOS. Data were insufficient to determine outcome differences between RL and NSS.

Discussion:
Gallstones remain the primary etiology of AP, especially among females, while alcohol-related disease predominates in males. Early enteral feeding within 24–36 hours consistently correlated with reduced LOS, underscoring the importance of early nutrition in line with ACG guidance. Suboptimal antibiotic adherence in necrotizing AP highlights an opportunity for stewardship interventions, as inappropriate selection or unnecessary use may increase resistance risk without improving outcomes. While moderate fluid resuscitation volumes were associated with better LOS, our cohort lacked statistical power to compare RL versus NSS;

however, recent evidence favors RL for its anti-inflammatory profile and potential to reduce disease progression. The idiopathic category, possibly influenced by medications, post-ERCP complications, or incomplete histories, warrants more thorough diagnostic evaluation to reduce recurrence risk.

Conclusions:
A substantial proportion of AP cases were acute, first-time presentations, often in obese patients. Gallstones and alcohol remain leading etiologies, with distinct gender patterns. Standardized inpatient protocols incorporating moderate IVF (~1.5 L bolus, then controlled rates), early diet initiation, and targeted antibiotic use may improve outcomes and reduce LOS. Although our data cannot confirm superiority of RL over NSS, existing literature supports RL's benefits. Prospective trials, including ongoing studies like WATERLAND, are needed to refine fluid strategies, validate feeding protocols, and reduce readmissions, particularly in patients with modifiable risk factors such as smoking, alcohol use, and hypertriglyceridemia. Implementing these evidence-based strategies directly enhances patient safety, reduces complications, and standardizes care across our institution.

R-05

Rehospitalization in Heart Failure: Comparative Impact of Sacubritral/Valsartan Vs. ACE Inhibitors/ARBs

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Introduction:
By 2030, 8.5 million Americans over 20 are predicted to have heart failure (HF). Heart failure is a leading cause of hospitalization in older adults, with high 30-day readmission rates significantly contributing to morbidity, mortality, and healthcare costs. While angiotensin-converting enzyme inhibitors (ACEi) and angiotensin receptor blockers (ARBs) have long been the standard therapy for HF with reduced ejection fraction (HFrEF), recent data suggests their effectiveness has plateaued. Angiotensin receptor-neprilysin inhibitors (ARNIs), such as sacubitril/valsartan (Entresto), have demonstrated superior outcomes in clinical trials. However, their real-world impact on readmission rates remains underexplored. We aim to evaluate the factors associated with heart failure readmission and compare the impact of Entresto versus ACEi/ARBs to guide therapy decisions and improve patient outcomes.

Methods:
We conducted a retrospective cohort study of patients admitted with a primary diagnosis of congestive heart failure (CHF) to San Lucas Hospital between 2022-2025. Electronic health records were reviewed to identify initial admissions and future readmissions for CHF. Data included discharge medication (ACEi/ARB or Entresto), number of readmissions, length of stay (LOS) during admission and readmission, and days between admissions. We also evaluated patients who were initially discharged on ACEi/ARB but then were changed to Entresto after readmission. Demographic, clinical, and laboratory data were also collected. The data was analyzed using t-tests, p-values <0.05 considered significant. IRB approval was obtained.

Results: Among 355 patiens (250 ACEi/ARB; 105 Entresto), readmission rates were 40.4% vs 37.1% ($p=0.596$; risk factor=0.884). Median LOS was 5 vs 7.5 days for the initial admissions and 7 vs 6 days for readmissions, respectively. Patients that switched from ACEi/ARB to Entresto had fewer mean readmissions (2.64 vs 3.14; $p=0.534$) and longer time between admissions (538 vs 463 days; $p=0.283$).

Conclusion:
Our findings, although not statiscically significant, indicate that patients on Entresto evidently showed fewer readmissions and longer time between hospitalization compared to those on ACEi or ARBs highlighting clinical relevance. This could benefit patients by having a better quality of life and improved symptoms control in patients with HFrEF. Moreover, it can also reduce healthcare utilization and cost. These findings, when supported by more research, may encourage the earlier use of Entresto in suitable patients, particularly those with a high risk of readmission.

R-06

Early Clues for Leptospirosis: A Retrospective Analysis at a Tertiary Center in Southern Puerto Rico to Improve Early Screening and Predict Disease Severity of Leptospirosis.

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Leptospirosis is a zoonotic disease caused by pathogenic spirochetes of the genus Leptospira. Infected individuals may present with a spectrum of clinical manifestations ranging from asymptomatic infection to severe, potentially fatal illness. The disease is often underdiagnosed and misdiagnosed due to its nonspecific symptoms, which can mimic other febrile illnesses such as dengue and tropical hemorrhagic fevers. This study aims to identify symptoms, vital signs, and laboratory parameters that may help facilitate early recognition of leptospirosis and provide prognostic indicators to estimate disease severity.

We conducted a retrospective analysis of the electronic medical records from 94 adult patients with suspected leptospirosis that visited the emergency room of Saint Luke's Hospital at Ponce, Puerto Rico, from 2019 to 2024. A total of 10 medical records were excluded from this study due to significant lack of information. The variables that were studied from laboratory confirmed (serologic and molecular) positive cases of leptospirosis include: presenting symptoms, vital signs, laboratory parameters, disease severity and outcome. IBM SPSS was used for statistical analysis, which included descriptive analysis, Chi-Squares and T-Test for Mean comparisons. IRB Approval was obtained (Protocol # 2504257511).

We found that among suspected cases of leptospirosis, 39% (n= 34) tested positive. Among these, 32% (n= 11) had symptoms associated with severe disease. While we did not find any specific symptoms associated with laboratory confirmed cases of leptospirosis, tachycardia was a vital sign associated with this disease. Additionally, we found several abnormal laboratory values associated with serologic and molecular confirmed cases of leptospirosis including anemia in females, thrombocytopenia, neutrophilia, hypocalcemia, azotemia, elevated liver enzymes, hyperbilirubinemia (direct and indirect), lactatemia, rhabdomyolysis, and elevated inflammatory markers. Altered urine laboratory values include proteinuria, elevated urine urobilirubin, and presence of WBC. Severe leptospirosis was associated with moderate thrombocytopenia, mild hypocalcemia and direct hyperbilirubinemia when compared to mild/moderate disease severity.

Although a small sample size limits our study, it highlights the challenges of diagnosing leptospirosis and disease severity. While no specific presenting symptoms were found to be associated with this illness, we found that the presence of tachycardia and several laboratory abnormalities may help diagnose leptospirosis accurately. These findings may contribute to the development of standardized clinical scoring systems for improving diagnosis and assessing disease severity in suspected leptospirosis cases.