BREAKOUT
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CV 161 - 170
A TALE OF AUTOIMMUNITY: THYMOMA, THYMECTOMY AND LUPUS

Introduction: The thymus plays an integral role in immune system regulation, modulating the development, diversity, and selection of T-cells, thus preventing T-cell mediated autoimmunity. Rare cases of post-thymectomy autoimmune-related diseases, including lupus and pure red cell aplasia, have been reported in the literature, however such presentations are exceedingly rare.

Presentation: A 65-year-old African American man with transfusion-dependent anemia of unknown etiology presented with abdominal pain and was found to have a large 6 x 10 x 13cm right-sided, anterior mediastinal mass. CT-guided biopsy revealed thymoma which due to its large size was initially unresectable. Anti-nuclear and Acetylcholine receptor antibodies were negative. Chemotherapy (Cisplatin, Cyclophosphamide and Adriamycin) commenced with repeat CT demonstrating a reduction in thymoma size. The patient subsequently underwent a complete thymectomy and thymomectomy with histopathology revealing benign thymic tissue and noninvasive thymoma. Despite initial improvement, he remained severely anemic, dependent on transfusions and developed progressive leukopenia and thrombocytopenia. He underwent marrow biopsy, which revealed hypocellular marrow with difficult to identify erythroid precursors, suggesting erythroid hypoplasia. Repeat serologies demonstrated a positive ANA (1:160), with positive anti-Smith and ds-DNA antibodies. C3 and C4 complement levels were normal. He fulfilled the SLICC and EULAR criteria for diagnosis of SLE. Unfortunately, he declined treatment with hydroxychloroquine, and decided to continue with weekly RBC transfusions. He eventually succumbed due to severe infection in the setting of pancytopenia.

Discussion: The thymus’ role in development, differentiation, and maturation of T-cells and central tolerance via positive and negative selection is well recognized. Thymomas are the most common mediastinal mass in adults and are associated with autoimmune disease/paraneoplastic syndromes such as myasthenia gravis and lupus. The prevalence of thymoma/thymic hyperplasia-associated autoimmune disorders is as high as 30%, while it ranges from 7-9% in the general population. Prevalence of thymoma-associated is limited (2%), although its prevalence post-thymectomy is highly variable. Multiple theories on the pathogenesis of autoimmune disease associated with thymoma have been postulated, focusing on dysregulation of normal thymic function, in turn affecting positive and negative selection processes. The exact mechanism and pathogenesis of autoimmunity after thymectomy, its prevalence, and predisposition remains to be elucidated.

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CV 161

BREAKOUT ROOM 17

ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
ALLOPURINOL-INDUCED DRESS COMPLICATED BY ACUTE INTERSTITIAL NEPHRITIS AND ACUTE RENAL FAILURE

DRESS is a drug-induced reaction, characterized by cutaneous involvement and end-organ damage and a triad of rash, fever, and eosinophilia. Acute Interstitial Nephritis (AIN) is a known complication characterized by renal interstitial inflammation. Here, we present a case of allopurinol-induced DRESS complicated by AIN and renal failure. An 84-year-old woman with CAD, NASH, CKD, primary hyperparathyroidism, and hyperuricemia presented with progressive decline in serum creatinine and a diffuse, pruritic maculopapular rash. She reported usual health until one month prior and denied associated exacerbating/alleviating factors or new exposures. Vitals remained unremarkable. Examination revealed a chronically ill woman with lower extremity edema and a diffuse erythematous, maculopapular rash on her extremities, trunk, and back. Diagnostics were notable for acute on chronic kidney injury, eosinophilia, thrombocytopenia, elevated IgE, and eosinophiluria. Renal and abdominal ultrasound demonstrated echogenic kidneys, non-obstructing stones, and hepatic steatosis. Given elevated FIB4/APRI scores, elevated serum creatinine, and hyperuricemia, multimodal management with albumin and febuxostat were initiated with transition to midodrine/ octreotide. CT confirmed a cirrhotic liver and noted a filling defect in the IVC. cMREI demonstrated a lobulated inferior right intra-atrial mass with IVC extension. Outpatient records confirmed a one-month history of rising creatinine, eosinophilia, and IgE that was temporally associated with allopurinol use. Shave biopsy demonstrated spongiotic dermatitis with eosinophils. Regi-Scar score was elevated suggesting DRESS. Additional diagnostics were negative. Her course was complicated by MSSA bacteremia, precluding steroid use, and progression to ESRD. CT demonstrated HCC with extension into hepatic veins, IVC, and right atrium. Her pruritic rash resolved with continued allopurinol avoidance and topical triamcinolone.

DRESS is multi-organ hypersensitivity reaction characterized by T-cell mediated autoimmunity, in which certain drugs/metabolites trigger destruction of self-antigens. Allopurinol is a recognized culprit and is associated with HLA-B58-01, specifically in Asian and European populations, and rheumatology guidelines recommend screening prior to therapy. AIN is a known complication of DRESS and, as in our case, is associated with pre-existing renal disease in elderly individuals.

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Autoimmune Hepatitis (AIH) is a rare liver disease that is commonly diagnosed in females of reproductive age. While some AIH patients are asymptomatic, typical symptoms include fatigue, jaundice and abdominal discomfort\(^{[1]}\). AIH has variable etiologies including drug-induced AIH (DI-AIH)\(^{[2]}\). The most common drugs that lead to AIH are nitrofurantoin, minocycline, and alpha-methyldopa with a limited number of diclofenac cases. In this abstract, we present a rare case of diclofenac-induced AIH.

A 61-year-old man with history of osteoarthritis, hyperlipidemia and hypothyroidism, presented to his primary care clinic with episodic right upper quadrant discomfort for the past two months. There was no history of hepatitis or known triggers for his discomfort and his daily activities were not affected. He was recently started on oral diclofenac for osteoarthritis. Physical exam was unremarkable with no jaundice, abdominal tenderness or organomegaly. Biochemically, the liver panel was abnormal: AST 496, ALT 946, t. bilirubin 1.6 and alkaline phosphatase 162, with a normal biochemical panel prior to starting the diclofenac. Diclofenac was stopped and the patient was referred with additional lab work and imaging to a gastroenterologist. On additional testing he was found to be positive for anti-smooth muscle antibody although anti-nuclear antibody and anti-mitochondrial antibody were negative. CT scan of the abdomen and pelvis was unremarkable. He was diagnosed with autoimmune hepatitis and treated with prednisone 40mg daily. On subsequent follow-up after discontinuing diclofenac and commencement of prednisone, the liver panel normalized and the patient had resolution of his abdominal pain.

Discussion:

DI-AIH has an estimated prevalence of 11% amongst all AIH patients\(^{[3]}\). The diagnosis of DI-AIH is a diagnosis of exclusion with the need to rule out other causes of chronic liver disease in the presence of autoantibodies. There are two types, Type 1 and Type 2, characterized by anti-smooth muscle antibody and anti-liver and kidney antibody, respectively. Typically, liver biopsy shows interface hepatitis\(^{[4]}\). Treatment involves medication discontinuation and steroids, although some patients will experience treatment failure and progress to cirrhosis. In those patients who present with fatigue associated with abdominal discomfort or jaundice and deranged liver panel with a temporal relationship to a drug warrant evaluation for DI-AIH.
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( ) Clinical Research

Indicate your participation in research process (4 sentences or less): Dr. Han, Dr. O’Malley, and Dr. Honasoge admitted this patient to the University of Maryland Medical Center, and were directly involved in his diagnostic evaluation and care.

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FDG-PET/CT IN THE DIAGNOSIS OF ACUTE PERICARDITIS.
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The diagnosis of acute pericarditis is established by the presence of two of these criteria: pain typical of pericarditis, pericardial rub on auscultation, PR segment depression and/or ST segment elevation on electrocardiogram (ECG), and pericardial effusion.1 While current diagnostic approaches may be insensitive, fluorodeoxyglucose positron emission tomography/computed tomography (FDG-PET/CT) can localize inflammation at the anatomic level.2,3

A 58-year-old male with uncontrolled rheumatoid arthritis and coronary artery disease presented with sudden-onset chest pain and diffuse arthralgias. Chest pain was not positional, and pericardial rub could not be heard on exam. Serial troponins were negative. ECG revealed sinus tachycardia with diffuse J point elevation; no comparison was available. Contrast-enhanced CT showed a pericardial effusion. Subsequent FDG-PET/CT demonstrated diffusely increased FDG activity in the thickened parietal pericardium and aortic root. A diagnosis of acute pericarditis was established, and treatment with oral colchicine and prednisone resulted in significant clinical improvement.

In the absence of classic signs and symptoms, the diagnosis of acute pericarditis can be challenging. Although cardiovascular magnetic resonance imaging has been shown to be useful, it has limited application in the setting of renal dysfunction.4 FDG-PET/CT is a novel tool that can be effective not only when the diagnosis of acute pericarditis is equivocal, but also in identifying patients at high risk of relapse.5,6

References:

Program Director's Name: Susan Wolfsthal, MD
ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
SUMP SYNDROME AT REMNANT COMMON BILE DUCT (CBD) FOLLOWING A LIVE DONOR LIVER TRANSPLANT: A CASE REPORT

Introduction: Sump syndrome, a collection of digested food, debris, stones, bile, and bacteria in a poorly drained, distal bile duct reservoir, is a complication of Roux-en-Y hepaticojejunostomy (RYHJ). While most cases occur at the hepaticojejunostomy site, we report a rare case that occurred in the distal remnant CBD. The patient received a living donor liver transplant and presented with recurrent bacteremia and symptoms of acute cholangitis.

Case Presentation: A 38 y.o. woman with a history of ileocolonic Crohn’s disease in remission, end stage liver disease secondary to primary sclerosing cholangitis, history of living donor liver transplantation via RYHJ, and Klebsiella Pneumoniae bacteremia with no identified source 3 months ago, presented with sharp right upper quadrant (RUQ) abdominal pain, fevers to 101.2 F, and positive blood cultures for Klebsiella Pneumoniae. MRCP showed a biliary stricture in the remnant CBD, associated with new edema and surrounding infiltration; the diameter of the segment proximal to the stricture had been stable for 2 years (6 mm). Neither PTC and ERCP were pursued at that time. Three months later, the patient returned with 2 days of sharp epigastric and RUQ pain associated with emesis, fevers, chills, and poor appetite. Vital signs were stable. Physical examination was significant for severe tenderness over the epigastrum and RUQ. Liver function tests were at her baseline. Workup for Crohn’s disease flare and transplant rejection were negative. Repeated MRCP showed an increase in the diameter of the proximal segment of the remnant CBD from 6 to 9 mm along with persistent surrounding edema. ERCP demonstrated lower third of the CBD contained a single moderate stenosis 5 mm in length, which was successfully dilated with a 6 mm balloon. Pus was swept from the cystic duct. The hepaticojejunostomy anastomosis was intact, with no evidence of strictures or recurrence of PSC. Following successful sphincterotomy, patient’s abdominal pain, fever, and leukocytosis resolved. Blood cultures showed no growth. Patient was discharged with a diagnosis of sump syndrome and was maintained on sulfamethoxazole-trimethoprim at discharge.

Discussion: In patients with history of RYHJ who present with symptoms of acute cholangitis with normal liver function, the diagnosis of sump syndrome of the distal CBD remnant should be considered. Prompt ERCP for biliary sphincterotomy can help treat infection and provide appropriate source control.

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CV 165

BREAKOUT ROOM 17

ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
Mucormycosis in a Type 2 DM Patient Presenting with DKA

Mucormycosis is an invasive fungal infection, usually involving the nasal turbinates or alveoli. It typically presents in patients with underlying disease, such as in our case, who was a patient with type 2 diabetes mellitus (DM) who presented in diabetic ketoacidosis.

A 52-year-old male initially presented to the emergency department with 4-5 day history of right sided sinus congestion and facial pressure. He was prescribed pseudoephedrine, ibuprofen, and saline nasal spray and advised to follow up with his primary care provider next week. That visit resulted in a prescription for Amoxicillin Clavulanate for persistent symptoms. The patient returned to the ED one week later with nausea, vomiting, lightheadedness, and generalized weakness.

Vital signs during hospital admission were within normal limits other than elevated blood pressure. Physical examination was significant for photophobia and left infraorbital numbness. The patient had a high anion gap, low bicarbonate, and elevated beta-hydroxybutyrate, consistent with the diagnosis of diabetic ketoacidosis (DKA). The patient had a history of Type 2 DM and previous episodes of hyperosmolar hyperglycemic syndrome, but never with ketoacidosis. He reported being compliant to his insulin regimen prior to this episode.

The patient was started on antibiotics for persistent ongoing sinus infection. However, CT scan of face/sinus showed concerning findings for invasive fungal sinusitis, which was confirmed on endoscopic biopsy. IV amphotericin B along with saline spray, fluconazole spray, and amphotericin irrigation were initiated, accompanied by multiple debridements. Fungal cultures eventually grew Rhizopus arrhizus complex. The patient developed acute kidney injury, likely secondary to amphotericin B. The IV antifungal therapy was changed to micafungin and posaconazole. Repeat MRI scans with attention to the skull base showed extension into the orbital area, and exteneteration of the orbit was recommended. However, the patient refused surgery and desired continued conservative treatment with IV antifungal therapy indefinitely.

This case illustrates the complication of rhino-orbital mucormycosis in a Type 2 DM presenting with DKA. It signals the importance in determining a cause for DKA, such as Mucor infection in this case. With the high mortality rate of mucormycosis, early diagnosis and treatment is essential.

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Program Director’s Name: R. Dobbin Chow, MD

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ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
Hereditary hemochromatosis and exocrine pancreatic insufficiency

Introduction: Exocrine pancreatic insufficiency (EPI) is a well-known form of malabsorption, however association with hereditary hemochromatosis (HH) is a rare concurrence. We present a case of severe vitamin D deficiency, hypocalcemia and hypomagnesemia due to EPI and HH.

Case Report: A 44 year-old African American female with recent menopause onset and family history of liver cancer, presents with recurrent carpopedal spasms and perioral tightness associated with 2 days of diarrhea. Initial testing revealed hypocalcemia (ionized calcium 0.91 mmol/L, serum calcium 7.2 mg/dL) and hypomagnesemia (serum magnesium 0.7 mg/dL), severe vitamin D deficiency (vitamin D-25-OH of 3.8 ng/mL) and secondary hyperparathyroidism (PTH 181.2 pg/mL).

During the hospital course the patient developed acute on chronic transaminitis which resolved in the following days. Further workup revealed iron overload with ferritin levels of 3,789.7 ng/mL, iron saturation of 76.0%, hemoglobin 10 mg/dL and transferrin of 170 mg/dL. HFE genotypes C282Y/H63D/S65C were negative, however given iron overload, transaminitis, age of presentation and family history, HH was considered. Malabsorption workup revealed low vitamin A and severe exocrine pancreatic deficiency with fecal pancreatic elastase <50 mcg EL/g stool. After aggressive electrolyte supplementation the patient’s symptoms subsided and she was discharged on oral pancreatic enzymes, vitamin and mineral supplements, with a follow up on iron-quantification- MRI.

Conclusion: Despite a high incidence of pancreatic involvement in HH, malabsorption syndrome as a manifestation of EPI in HH has been described, although its concurrence is extremely rare. Severe symptomatic hypomagnesemia and hypocalcemia was associated with intestinal malabsorption of vitamin D due to EPI and HH. Deposition of iron in the liver impairs vitamin D activation, aggravating clinical manifestations of hypocalcemia and hypomagnesemia. Most cases of primary iron overload in African American population are phenotypically and genotypically distinct from whites of northern European descent. The mainstay in management of EPI is the administration of exogenous pancreatic enzymes, fat-soluble vitamins and other micronutrients. However if HH is the pivotal cause, phlebotomy is the most appropriate intervention to avoid further pancreatic and liver damage.

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CV 167

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ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
Immune mediated adverse event by combination therapy of Pembrolizumab and Axitinib
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Sinai Hospital of Baltimore

Introduction: Pembrolizumab is a humanized recombinant monoclonal immunoglobulin targeting the Programmed Death Ligand-1 (PDL1), and recently surpassed standard of care in overall survival for clear cell renal cell carcinoma (RCC) as seen in the CheckMate 214 trial. Studies combining Axitinib, a vascular endothelial growth factor receptor tyrosine kinase inhibitor, with immunotherapy such as pembrolizumab have been promising in patients with advanced RCC. Immune related adverse events (IRAE’s) are complications documented in temporal relation to administration of PDL-1 therapy. Within we present a case of an adverse outcome from axitinib and pembrolizumab immune mediated hepatic toxicity.

Case: A 72-year-old man with a medical history significant for hypertension, diabetes, and chronic kidney disease stage 3, presented to the Emergency Room (ER) with complaints of chest pain and hematuria. Imaging revealed a heterogenous right renal mass and biopsy was diagnostic of clear cell type RCC. He was also found to have extensive metastases to multiple organs including lung, liver and bone. The patient received 5 cycles of neoadjuvant Axitinib and Pembrolizumab before returning to the ER for confusion. Initial laboratory findings were remarkable for elevations in AST/ALT 176/127 U/L and ammonia at 42.8 μmol/dL. Liver enzymes continued to trend upwards and peaked at AST/ALT 360/261 U/L. Other causes were excluded with no antibodies for Hepatitis A, C, anti-smooth muscle, anti-mitochondrial, anti-nuclear and negative Hepatitis B surface antigen. For presumptive IRAE the patient first received stress-dose (120mg/24hr) glucocorticoids and was subsequently started on Mycophenolate-Mofetil as salvage therapy. Unfortunately, the hepatic dysfunction progressed, and the patient expired.

Lessons: IRAE in patients treated with checkpoint inhibitors have been well documented, and cases such as immune-related hepatitis can be fatal, as described in this case. PDL-1 combination therapy with Axitinib has been documented to lead to increased liver injury compared to using Pembrolizumab alone. Close monitoring of individuals on combination therapy is warranted as well as further clinical trials to determine best management options for management of hepatic injury related to IRAE.

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BREAKOUT ROOM 17

ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
Abstract
Introduction
Toxic epidermal necrolysis (TEN) is a rare, life threatening, mucocutaneous reaction with an annual incidence of 0.4-1.2 per million people. TEN involves skin detachment of >30% of the total body surface area (BSA); it carries a mortality of up to 50%.

TEN is almost always caused by medication within several weeks of starting the drug. Less commonly, TEN follows a viral or bacterial infection. The risk of TEN can be related to specific HLA alleles.

Pathogenesis involves a type IV hypersensitivity reaction with native killer and CD8+ T-cells releasing granulysin and perforin within epidermal bullae. Cellular apoptosis in keratinocytes and separation of epidermis from the dermis follows, leading to skin loss and, potentially, sepsis, multiorgan failure, shock and death.

Case: An 85 year-old Caucasian woman was hospitalized for pneumonia. During a month of subacute rehabilitation small, asymptomatic macules appeared on her back. Two weeks after spontaneous resolution of the rash, she was readmitted for confusion and a flu-like illness. A widespread rash which began as scaling and blisters without target formation was observed. Over 24 hours, the eruption extended to 65% of BSA, becoming confluent over the back and chest with areas of full thickness erosions/shaerings. The abdomen demonstrated atypical targetoid pink macules with red centers; confluent erythema was present on the anterior thighs and arms, and smaller erythematous patches were present on the lower legs, sparing the palms and soles. The rash was pruritic without pain. A gentle shear force applied to the skin separated the epidermis from the dermis (Nikolsky sign) in erythematous areas. She was afebrile, and hemodynamically stable without leukocytosis. The patient’s prior drug allergies included ciprofloxacin, mirtazapine, and omeprazole. The clinical differential diagnosis included TEN, acute generalized exanthematous pustulosis (AGEP), subacute cutaneous lupus or acute systemic lupus, atopic dermatitis, psoriasis, contact dermatitis and mycosis fungoides/Szery syndrome, pemphigus vulgaris and bullous pemphigoid. A skin biopsy demonstrated full thickness epidermal necrosis with negative direct immunofluorescence. While consistent with TEN or Stevens Johnson Syndrome, involvement of 65% BSA confirmed. SCORTEN was 3 (age>40, serum BUN>10 mmol/L, serum glucose level >14 mmol/L) suggesting 35% mortality. Following a 3 day course of intravenous solunemol with intensive wound care she ultimately recovered. Levofloxacin and loperamide were drugs started within the two weeks before the rash began and were the most likely triggers.

Conclusion
We present a case of drug-induced TEN. Notable features included: flu-like prodrome, explosive onset, and complete recovery. While this patient benefited from a course of systemic corticosteroids, other potential therapies include intravenous IgG, cyclosporine, and other immunosuppressives. Review of previously administered medications from multiple facilities was essential in determining likely culprit drugs, and preventing inadvertent drug re-exposure and recurrence of this life threatening condition.

CV 169
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Indicate your participation in research process (4 sentences or less): I performed the initial admission history and physical exam, wrote the case presentation, and designed the poster.

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