
CLINICAL VIGNETTE COMPETITION

2025



Utah ACP Resident & Fellow Committee

David Chen MD—Chair

Adeline Browne MD—Co-Chair



Fall Clinical Vignette Program | Thurs, November 13, 2025

University of Utah School of Medicine | Internal Medicine Grand Rounds

12:00PM	Welcome & Opening Remarks <i>Resident & Fellow Committee</i>	Judges Stacy Johnson, MD Katie Lappe, MD
12:10PM	Presentations <i>Jackson Burton: Peripartum Cardiomyopathy in a Patient with a TTN Truncating Variant</i>	Pg 7
	<i>Kelsey Wartgow: Uncovering the Truth: A Full Body Rash</i>	Pg 65
	<i>Matt Glasgow: An unexpected side effect: The heartache of drug-induced lupus</i>	Pg 34
	<i>Lily Kreber: The Price of Complement Blockade: A Case of Mucor-Driven Vascular and Pleural Catastrophe</i>	Pg 55
12:50PM	Announce Runners-Up & 1st Place Winner	
12:55PM	Closing Comments <i>Resident & Fellow Committee</i>	

Utah ACP Resident & Fellow Committee | Mission Statement

To improve the professional and personal lives of Utah Residents and Fellows and encourage participation in the American College of Physicians.

1. Foster Internal Medicine Resident's interest in the ACP – ASIM.

- Encourage ACP associate membership and a lifelong interest in ACP – ASIM.
- Encourage representation on National and Local ACP subcommittees.

2. Foster educational Opportunities for Internal Medicine Residents.

- Encourage participation in local and national ACP – ASIM Associates Clinical Vignette and Research opportunities.
- Organize the local competitions. Provide information on board review courses. Publicize local and national educational opportunities. Work with residency programs to improve residency education.

3. Identify practice management issues for Internal Medicine Residents.

- Provide information for residents as they prepare to enter practice, such as practice opportunities and contract negotiation.

4. Identify public policy concerns of residents.

- Monitor local and national health policy and how it relates to Internal Medicine and residency training.

5. Encourage an interest in community service.

- Identify ways associates can become involved with community service in Utah.

Table of Contents

.....	1
Fall Clinical Vignette Program Thurs, November 13, 2025	2
Utah ACP Resident & Fellow Committee Mission Statement	2
#1 A perPLEXing case of thrombotic microangiopathy in pregnancy	5
#2 Peripartum Cardiomyopathy in a Patient with a TTN Truncating Variant	7
#3 “A Rash Decision: IgA Vasculitis During MSSA Bacteremia”	10
#4 Bringing the Diagnosis into Focus: Blurry Vision and Diffuse Lymphadenopathy Unmasking Primary HLH	12
#5 Endocarditis?! No, it’s not the cardia.....	14
#6 Cultural Barriers in Pain Assessment: A Case of an Indigenous Q’eqchi’ Patient with COVID-19.....	16
#7 The Heart Wants What it Wants (Recurrent ICD Pocket Infection)	17
#8 Acute Onset Meat Allergy vs Alpha-Gal Syndrome, a Historical Conundrum.....	19
#9 Toxicity as a Biomarker: Lessons from a Case of Severe irAEs in Melanoma	20
#10 From Loose Stool to Solid Diagnosis.....	22
#11 Anchored to Ovarian Cancer: A Case of Myeloid Sarcoma That Set Sail in a Different Direction	24
#12 Anaplastic in Appearance: A Rash with Hidden Depths	25
#13 An Uncommon Complication of a Common Condition	27
#14 Too Blue to Be True: A Case of Disproportionate Hypoxemia.....	28
#15 Recurrent Viremia and Immunosuppression Optimization in an EBV D+/R- Mismatch Kidney Transplant Patient.....	30
#16 Seizing the Diagnosis: A Rare Case of Fatal West Nile Virus Ventriculitis	32
#17 An unexpected side effect: the heartache of drug-induced lupus	34
#18 When Antihypertensives Leave a Mark: Amlodipine-Associated Schamberg Purpura	36
#19 A Shadow Amongst Pathogens: The Hidden Guest	38
#20 A Fine Line Between Infection and Intoxication: Valacyclovir Toxicity in End-Stage Renal Disease	40
#21 Starving the Mind: Poisoned Thoughts Masking Possible Autoimmune Encephalitis.....	41
#22 From murmurs to melena: a case of the overlooked heart-gut connection.....	44
#23 A Devastating and Fatal Case of Hemophagocytic Lymphohistiocytosis and Immune Reconstitution Inflammatory Syndrome in the Context of Untreated HIV.....	46
#24 When Ibuprofen Burns: A Rare Case of Drug-Induced Linear IgA Bullous Dermatitis	48
#25 A Difficult Case of Medicine Mimicry	50

#26	A Bleeding Misconception: When Anchoring Bias Delays Two Critical Diagnoses in an Anemic Patient	51
#27	The Tell-Tale Eye: How a Subtle Palsy Unveiled a Sellar Giant	53
#28	The Price of Complement Blockade: A Case of Mucor-Driven Vascular and Pleural Catastrophe...	55
#29	Abdominal pain, Fabry Disease, and a Rare Complication	57
#30	Beyond Viral Reactivation: EBV-Negative Methotrexate-Associated DLBCL in an Elderly Patient with Rheumatoid Arthritis	59
#31	Unmasking PTLD: The Challenges of Navigating Overlapping Pathology	61
#32	Metastatic Colorectal Cancer Presenting as Intractable Radicular Pain in a Young Patient Without Age-Appropriate Screening: A Clinical and Psychosocial Perspective	63
#33	Uncovering the Truth: A Full Body Rash	65
#34	Therapeutic Resection of Functional Adrenocortical Adenoma for Subclinical Cushing Syndrome	67
#35	Acute pancreatitis as primary driver of shock in the setting of LAD STEMI	69
#36	Blue Toe Syndrome: Manifestation of an often forgotten deadly illness	71
#37	Beyond Primary Hyperaldosteronism: Investigation of Refractory Hypertensive Hypokalemia	75
#38	Lupus Enterocolitis as an Initial Presentation of Systemic Lupus Erythematosus: A Diagnostic Challenge in Acute Abdominal Pain.....	76

#1 A perPLEXing case of thrombotic microangiopathy in pregnancy

Patient ID: 31 yo female at 29w5d gestation with PMH of spina bifida, neurogenic bladder requiring self-catheterizing, gastric reflux, major depressive disorder, obesity, who presents 29 weeks pregnant with severe thrombocytopenia.

Case presentation: A 31-year-old female was transferred to our hospital at 29w5d gestation for thrombocytopenia and preterm labor. Two days prior, she had presented to her local hospital for constipation, nausea, and suspected urinary tract infection (UTI). Laboratory findings were significant for thrombocytopenia of $25 \times 10^9/L$. Her cervical exam was found to be 4/90/-2. Betamethasone was given for fetal lung maturation given her preterm status, magnesium was started, and she was transferred for direct admission to labor and delivery at our hospital. On admission, cervical exam was unchanged and she reported intermittent suprapubic/abdominal pain and a mild headache. Scant vaginal bleeding was noted prior to transfer. She denied loss of fluid, changes in vision, or right upper quadrant pain. She had obtained most of her prenatal care in Mexico where her husband was living. The patient then had increasing abdominal pain concerning for contractions.

Vitals and Labs: Her vitals were notable for mild tachycardia to 115bpm, otherwise unremarkable. Repeat CBC was notable for hemoglobin of 10 g/dL, white blood cell of $18 \times 10^9/L$, and platelet count of $35 \times 10^9/L$. CMP showed normal electrolytes, hepatic, and renal function, however elevation in total bilirubin (1.7 mg/dL) and ALP (188 U/L) were noted. A urinalysis was cloudy with moderate leukocyte esterase, positive nitrites, rare bacteria, positive blood, WBC 83, RBCs 212. Her UPCR was 1311.

Differential at this time included: preeclampsia with severe features, HELLP, TTP, ITP, HUS, DIC.

Clinical Course: Hematology was consulted for thrombocytopenia. Further lab results suggested a low grade hemolytic process. While direct Coombs was negative with low fractionated bilirubin, LDH was elevated to 409 and haptoglobin was undetectable. Peripheral smear revealed moderate normocytic normochromic anemia, mild neutropenia with toxic changes, moderate thrombocytopenia, and occasional schistocytes at 3 per HPF. Her autoimmune, complement, and infectious labs were unremarkable beyond a positive lupus anticoagulant panel, and she was low in B12 and Zinc. A Shiga toxin and ADAMTS-13 testing were requested. PLASMIC score was 4-5 at this time (depending which platelet count was considered), suggesting intermediate risk for thrombotic thrombocytopenic purpura (TTP). On HD2, Shiga toxin came back negative and ADAMTS-13 panel revealed an activity $< 5\%$. **She was diagnosed with TTP.**

Treatment: She had emergent therapeutic plasma exchange (TPE) and was started on 1 mg/kg prednisone. Given her current pregnancy status, caplacizumab was not initiated. On HD3, the ADAMTS13 Inhibitor came back positive at a level of 0.8, supporting acquired TTP as opposed to congenital. She achieved a clinical response after 5 days of TPE and prednisone. Prednisone was continued until recovery of platelets. On HD 11, the patient had preterm premature rupture of membranes and proceeded to have an overall uncomplicated vaginal delivery. She was discharged on postpartum day 2 with a steroid taper plan and close follow up with hematology.

Discussion: TTP is a rare and deadly thrombotic microangiopathy (TMA) typical characterized by the pentad of fever, anemia (MAHA), thrombocytopenia, renal pathology, and neurologic manifestations. Mortality for untreated TTP is > 90%, and unfortunately, confirmatory testing with ADAMTS-13 can take several days. In our case, the rarity of schistocytes on peripheral blood smear and PLASMIC score with low-intermediate risk led to a lower suspicion for TTP initially, delaying early initiation of TPE. Notably, an absence of schistocytes occurs in up to 30% of cases due to obtaining a smear in early stages (Grall 2017; Decker 2021). This is an important reminder to repeat daily smears if clinical suspicion for TTP remains present. Lastly, while pregnancy is a known risk factor for TTP, there is an expanded differential for TMAs in pregnant patients. This case is a reminder to consider the full spectrum of differential diagnoses in pregnant patients.

References:

Grall M, Azoulay E, Galicier L, Provôt F, Wynckel A, Poullin P, Grange S, Halimi JM, Lautrette A, Delmas Y, Presne C, Hamidou M, Girault S, Pène F, Perez P, Kanouni T, Seguin A, Mousson C, Chauveau D, Ojeda-Uribe M, Barbay V, Veyradier A, Coppo P, Benhamou Y. Thrombotic thrombocytopenic purpura misdiagnosed as autoimmune cytopenia: Causes of diagnostic errors and consequence on outcome. Experience of the French thrombotic microangiopathies reference centre. *Am J Hematol.* 2017 Apr;92(4):381-387. doi: 10.1002/ajh.24665. Epub 2017 Feb 21. PMID: 28133771.

Decker P, Moulinet T, Revuz S, Perez P, Jaussaud R. Thrombotic Thrombocytopenic Purpura Without Schistocytes: Beware of Misdiagnosis. *Neurol Clin Pract.* 2021 Oct;11(5):e798-e800. doi: 10.1212/CPJ.0000000000001067. PMID: 34840915; PMCID: PMC8610503.



#2 Peripartum Cardiomyopathy in a Patient with a TTN Truncating Variant

Authors: Jackson S. Burton M.D., Konstantinos Karampinos M.D., Holly Andrews PA-C, Aditya Mehta M.D., Line Kemeyou M.D.

Introduction:

Peripartum cardiomyopathy is characterized by acute heart failure during pregnancy or in the immediate postpartum period. [1] Approximately 1 in 25 heart transplants performed in women in the United States are for peripartum cardiomyopathy. [2] It is uncommon that pregnancy unmasks a previously latent hereditary cardiomyopathy.

Case Description:

A 36-yo G2P2 female with known pathogenic TTN gene variant and familial history of dilated cardiomyopathy presents 7 weeks post-partum with 1 week of worsening dyspnea. She had an uncomplicated gestation and had normal surveillance transthoracic echocardiograms (TTEs) throughout her pregnancy and at four weeks postpartum. However, she experienced persistent postpartum bleeding after her elective C-section requiring D&C at 3 weeks, followed several weeks thereafter by re-presentation for endometritis, tubo-ovarian abscess, and septic pelvic thrombophlebitis requiring abdominal washout with TAH and bilateral salpingectomy. In the two weeks since her surgery, the patient was recovering well; she was seen in OB clinic 5 days prior to presentation and had no complaints. Over the subsequent days, however, the patient began to experience progressive activity intolerance and paroxysmal nocturnal dyspnea.

In the ED, the patient was afebrile, tachycardic (126 bpm), with elevated diastolic blood pressure (124/108 mm Hg), and saturating 98% on room air. Physical exam notable for mild bibasilar crackles, tachycardia, JVD mid-neck, and well-healing midline incision without erythema or drainage. Labs were significant for Hgb 11.7 (post-op nadir 7.1), WBC 8.3, D-Dimer 0.85, BNP 667, HS-Troponin 11, lactate 1.2, UA with 2+ protein. EKG showed sinus tachycardia and poor R wave progression unchanged from prior. Bedside TTE showed reduced left ventricular ejection fraction 30-35% with global LV dysfunction and without evidence of RV failure.

Following the preliminary workup, peripartum cardiomyopathy was considered the most likely diagnosis given the patient's family history, known pathogenic TTN mutation, and presentation with de novo acute HF rEF. Given the patient's positive D-dimer, sinus tachycardia, postpartum status, and recent surgeries, we also considered concurrent pulmonary embolism. With elevated diastolic blood pressure (>90 mm Hg) and isolated proteinuria, pre-eclampsia was also considered. Upon admission to the heart failure service, the patient was treated with IV diuretics and started on an ACE inhibitor. CT angiogram of the pulmonary arteries was obtained and revealed a small subsegmental pulmonary embolism, so enoxaparin was started. Repeat urinalysis was negative for protein and the patient's blood pressure improved with diuresis and enalapril. OB-GYN was consulted given the initial high diastolic blood pressure and proteinuria; there was low concern for pre-eclampsia given the negative repeat UA and the fact that the patient was >6 weeks postpartum. Over the next several days, diuresis was continued and all GDMT indicated for a breastfeeding patient was initiated (enalapril, metoprolol, spironolactone). On Day 5, the patient was discharged with nearly resolved symptoms for further follow-up outpatient in the heart failure clinic.

Discussion:

This case illustrates several interesting principles regarding the diagnosis and management of peripartum cardiomyopathy.

First, the differential diagnosis of the dyspneic postpartum patient is broad – postpartum bleeding, operative site infection, sepsis, pneumonia, pre-eclampsia, pulmonary embolism – and multiple ailments may occur concurrently in a patient with peripartum cardiomyopathy.

Second, genetic variants associated with hereditary cardiomyopathies have variable phenotypic penetrance; <30% of individuals with a pathogenic variant had clinical or subclinical cardiomyopathy in one large cross-sectional study. [3] Differences in the pathophysiologic mechanism resultant from various pathogenic TTN variants may explain their highly variable penetrance. [4] In our patient with the TTN truncating variant c.85090C>T (p.Arg28364*), a premature stop codon is introduced in the distal transcript resulting in toxic accumulation of the truncated protein within cardiomyocytes. Non-truncating variants and more proximal truncating variants, however, are more likely to exert phenotypic influence through haploinsufficiency, or insufficient production of the wild type protein, rather than accumulation of toxic products. Regardless of the mechanism of the underlying genetic abnormality, illness or physiologic stressors, such as pregnancy, can unmask previously phenotype-negative individuals by providing a “second hit”. Patients with peripartum cardiomyopathy and TTN truncating variants, such as ours, have lower rates of recovery to normal ejection fraction at 1 year than normal genotype patients and warrant close follow-up a heart failure specialist. [1]

Third, pregnant or lactating patients require special consideration for heart failure GDMT. During pregnancy, ACE-Is, ARBs, and MRAs are contraindicated, so therapies are restricted to beta blockers, vasodilators (hydralazine, nitrates), and loop diuretics. [5] For breast feeding patients, enalapril, metoprolol, and spironolactone are considered safe for use. For postpartum patients with severe LV dysfunction <35%, prophylactic anticoagulation can be considered as can wearable defibrillators to reduce the risk of sudden cardiac death.

References:

1. **Ware JS, Li J, Mazaika E, Yasso CM, DeSouza T, Cappola TP, et al; IMAC-2 and IPAC Investigators.** Shared genetic predisposition in peripartum and dilated cardiomyopathies. *N Engl J Med.* 2016;374(3):233-241. doi:10.1056/NEJMoa1505517 [PubMedAdvocate Health - Midwest](#)
2. **Elkayam U.** Clinical characteristics of peripartum cardiomyopathy in the United States: diagnosis, prognosis, and management. *J Am Coll Cardiol.* 2011;58(7):659-670. doi:10.1016/j.jacc.2011.03.047 [PubMed](#)
3. **Shah RA, Asatryan B, Sharaf Dabbagh G, et al; Genotype-First Approach Investigators.** Frequency, penetrance, and variable expressivity of dilated cardiomyopathy-associated putative pathogenic gene variants in UK Biobank participants. *Circulation.* 2022;146(2):110-124. doi:10.1161/CIRCULATIONAHA.121.058143 [PubMed](#)

4. **Huynh K.** Truncated titin proteins in the pathophysiology of DCM. *Nat Rev Cardiol.* 2022;19:6. doi:10.1038/s41569-021-00648-8 [Nature](#)
5. **McNamara DM, Arany Z; and Heart Institute, Keck School of Medicine group.** Peripartum cardiomyopathy: JACC state-of-the-art review. *J Am Coll Cardiol.* 2020;75:207-221. doi:10.1016/j.jacc.2019.11.014

#3 “A Rash Decision: IgA Vasculitis During MSSA Bacteremia”

Authors: Anila Mehta PGY2, Meenu Singh, MD

Case Description

A 52-year-old woman with history of extensive spinal surgeries, cerebral palsy, type 2 diabetes mellitus, hypertension, asthma presented with severe 10/10 acute on chronic low back pain for one day. She denied any inciting trauma or neurologic deficits. On admission, she was febrile (102.7 deg F), tachycardic (127 bpm) and tachypneic (RR 24/min). Physical examination revealed tenderness over the lumbar spine. Labs showed leukocytosis (WBC 17.7), elevated lactic acid at 2.5 and elevated CRP of 28.4. Initial workup was unremarkable with negative respiratory viral panel, urinalysis, CT chest and abdomen, MRI of thoracic and lumbar spine and echocardiogram. Blood cultures grew staphylococcus aureus in 2/2 bottles. Due to persistent severe back pain, MRI of entire spine was repeated which revealed acute L4-5 discitis-osteomyelitis and ventral epidural phlegmon with irregular abscess from T4-L5/S1. She was treated with cefazolin, later transitioned to nafcillin, resulting in resolution of sepsis physiology and improved back pain.

On hospital day 11, the patient developed a rapidly progressive, non-blanching, non-pruritic purpuric rash involving her lower extremities. The rash progressed to the upper extremities, shoulders, and lower abdomen. She denied any systemic symptoms aside from back pain. Labs showed normal renal function, urinalysis and an absence of eosinophilia. Differentials included cutaneous small-vessel vasculitis (CSVV), drug-induced hypersensitivity vasculitis and IgA vasculitis. Skin biopsy confirmed the diagnosis of IgA vasculitis.

Given active treatment for MSSA bacteremia and extensive epidural abscess, corticosteroids, which are first line therapy for IgA vasculitis, were avoided to prevent immunosuppression. Instead, she was treated with colchicine and topical steroids (clobetasol). With this regimen, the patient’s rash began to resolve within 2-3 days.

Discussion

Diagnostic uncertainty in this case arose from the overlapping clinical and histopathologic features of CSVV, drug-induced hypersensitivity vasculitis and IgA vasculitis. All can present with palpable purpura and leukocytoclastic vasculitis on biopsy. **Skin biopsy with direct immunofluorescence** is essential for identifying IgA deposition.

In addition to palpable purpura, IgA vasculitis typically presents with arthralgias, abdominal pain and renal involvement such as hematuria or proteinuria. However, up to 10-20% of adult cases present with only cutaneous findings, as in our patient, complicating timely diagnosis.

The main therapeutic challenge is that there is a lack of robust evidence guiding the optimal management of IgA vasculitis in adults with active infection and multiple comorbidities. **Another barrier is immunosuppressive therapy, especially corticosteroids, may worsen outcomes in infection-related vasculitis** by impairing host defense. Therefore, steroid-sparing agents such as colchicine and topical corticosteroids are preferred for skin-limited disease in the context of active infection.

In our patient case, colchicine was utilized and rash resolved within 2-3 days. Although evidence from clinical trials is limited, colchicine is generally safe, well tolerated and has shown benefit in cutaneous vasculitis through its anti-inflammatory effects. Other glucocorticoid-sparing medications do not cause clinically significant immunosuppression and can be used in the management of IgA vasculitis with minor manifestations include dapsone and hydroxychloroquine.

Conclusion

This case highlights the diagnostic and therapeutic challenges of IgA vasculitis in the setting of active infection. The absence of systemic involvement made diagnosis particularly difficult, emphasizing the importance of distinguishing between the different types of vasculitis. Our patient's rapid improvement with colchicine and topical corticosteroids illustrates the potential role of steroid-sparing therapies in safely managing skin-limited IgA vasculitis when immunosuppression is contraindicated.

#4 Bringing the Diagnosis into Focus: Blurry Vision and Diffuse Lymphadenopathy Unmasking Primary HLH

Christina Druskovich, MD, Department of Internal Medicine, University of Utah; Mackenzie Redmond, DO, Division of Respiratory, Critical Care, and Occupational Pulmonary Medicine, University of Utah; Jason Carr, MD, Department of Pulmonary and Critical Care Medicine, Intermountain Medical Center and Division of Respiratory, Critical Care, and Occupational Pulmonary Medicine, University of Utah

A 28-year-old female with past medical history of depression and PTSD presented with three days of blurry vision and several months of progressive fatigue, unintentional weight loss, and night sweats. She was admitted initially to the ICU due to toxic appearance and concern for a rapidly progressive clinical course.

Physical exam

On arrival to the ICU, she was tachycardic, tachypneic, and encephalopathic with hypotension which was responsive to fluids. Her exam was notable for pink conjunctivae, 20/50 bilateral visual acuity, 20/70 in the right eye, and unable to assess the left eye due to blurriness. Additionally, she had a hemorrhagic lesion on her upper lip, rhonchi in her right upper lung fields, and diffuse lymphadenopathy in the submental, submandibular, preauricular, cervical, inguinal, and popliteal areas.

Lab and imaging results

Her initial CBC was significant for WBC of 9.7k/ml, hemoglobin of 6.1 g/dl, platelets of 23 k/ml, BMP notable only for sodium of 123 meq/l. CT imaging revealed diffuse adenopathy in the neck and upper chest, splenomegaly, hepatomegaly, and multiple enlarged lymph nodes throughout the retroperitoneum. Other notable workup included positive cardiolipin and a CMV molecular assay that was detected but not quantifiable. Other infectious workup, including viral and zoonotic infections, were unremarkable. Rheumatologic workup was largely unremarkable with the exception of a ferritin of greater than 100,000 and soluble IL-2 receptor of 2,746.7 pg/mL. Transthoracic echocardiogram demonstrated an ejection fraction of 49% with mildly increased LV wall thickness with concentric hypertrophy and a small pericardial effusion.

Clinical Course

Her initial presentation was concerning for lymphoma with secondary hemophagocytic lymphohistiocytosis (HLH) given young age and diffuse lymphadenopathy paired with weight loss and night sweats. Ophthalmology was consulted to evaluate her vision and noted bilateral retinal hemorrhages and a large pre-retinal hemorrhage. Our differential also considered autoimmune etiologies such as systemic lupus erythematosus and antiphospholipid antibody syndrome. Rheumatology was consulted in addition to oncology. Core needle biopsy of affected lymph nodes and bone marrow biopsy were obtained in addition to significant serologic work up for autoimmune disease and to verify HLH.

The patient remained stable in the ICU and was transferred to the floor but ultimately transferred back to the ICU with progressive hypoxemia. Bone marrow biopsy was expedited due to concern for evolving clinical course. It demonstrated toxic vacuolation of neutrophils and atypical lymphocytes. Two lymph nodes biopsied prior to administration of steroids were without evidence of hematologic malignancy. At this point fibrinogen and triglycerides returned and were markedly elevated, further raising concern for secondary HLH in the setting of a new malignancy and negative infectious and autoimmune work up. The low titer CMV viremia was deemed to be reactivation in the setting of critical illness. The patient was given high dose intravenous dexamethasone and stabilized clinically prior to transfer to a BMT capable facility for ongoing care. Genetic testing from peripheral blood sample resulted as equivocal, with findings of a novel LYST (c4391A>G, p.Asn1464Ser) mutation of unknown significance. A myeloid malignancy mutation panel (blood sample) resulted in KRAS and KDM6A mutations, but no malignancy.

Treatment

After the lymph node biopsy, the patient was started on 20 mg dexamethasone daily. Facial and neck edema improved notably within 24 hours. After serious infection was ruled out, she was started on etoposide 150

mg/m² on D1, 4, 8, 11 and then weekly D18, 25, 32, 39, 46 and 53. She was continued on a 56-day dexamethasone taper. Once the steroid taper is finished, she will receive intrathecal chemotherapy with methotrexate 12 mg and hydrocortisone 15 mg each week, for a total of 4 treatments.

Conclusion

There are two types of HLH: secondary and primary. Primary HLH is a genetic disorder usually presenting in childhood, whereas secondary HLH is caused by an external trigger, such as infections, malignancies, and autoimmune disorders (termed macrophage activation syndrome). The majority of cases are due to secondary HLH. Our patient uniquely was diagnosed later in life and was found to have a LYST mutation, a potential cause of primary HLH. The current primary consideration for etiology of her primary HLH is incomplete penetrance of heterozygous LYST mutation versus Chediak-Higashi Syndrome.

HLH is rare, and diagnosis requires exclusion of more common causes of multisystem illness like autoimmune, infectious, or malignant etiologies. An interdisciplinary team with access to significant diagnostic resources was required to rapidly facilitate work up, safely monitor treatment, and identify a likely primary diagnosis emphasizing the role of large systems and teamwork in complex patient care. This case highlights the need for a broad differential when patients present with vague symptoms of fevers, blurry vision, and diffuse lymphadenopathy.

#5 Endocarditis?! No, it's not the cardia

Blake Findley, BS^a; Mark Sims, MD^b

^aUniversity of Utah School of Medicine

^bDivision of General Internal Medicine, Intermountain Health

Introduction: 48-year-old male with a history of culture-negative infective endocarditis with septic emboli to brain and lungs 2 years prior to admission presented to the ED with right sided chest pain, dyspnea, hemoptysis, and altered mental status. He reports two weeks of trouble focusing his vision, a productive cough with blood-tinged sputum, and a syncopal episode at work one day prior to admission.

Exam: Patient was tachycardic to 110 otherwise vitals were stable. He appeared uncomfortable and ill-appearing. He kept his eyes closed due to photophobia. Coarse breath sounds were noted bilaterally without wheezes or crackles. Diffusely weak with grip strength and plantarflexion weaker on the right side compared to the left. Disoriented to situation.

Labs and Imaging: Labs revealed a leukocytosis to 12.9 with a left shift. MRI brain showed 3 small ring-enhancing lesions on the right thalamus, right cerebellum, and left parietal lobe causing a mild right to left midline shift. CTA PE showed several nodules in both lungs and a RML infiltrate representing pneumonia vs pneumonitis. TTE was significant for a 1mm, mobile, hypodense lesion on the non-coronary cusp of the aortic valve. HIV was negative.

Clinical Course: The patient was admitted with a working diagnosis of endocarditis with septic emboli to the brain and lungs. Blood cultures were drawn. He was started on empiric treatment with ceftriaxone and vancomycin. Infectious disease was consulted. Blood cultures remained negative for the course of the hospitalization. Further history revealed that the patient worked as landscaper and with frequent cuts to his hands and arms. Due to the negative cultures and occupational exposure, the team had high suspicion for infection with an atypical organism. Antibiotics were broadened to vancomycin, cefepime, and metronidazole. Additional testing for *Coccidioides*, *Cryptococcus*, *Candida*, and HACEK organisms were ordered, all of which returned negative. A Karius test was ordered. The patient's neurologic status returned to baseline by hospital day 4. A TEE showed no evidence of endocarditis and a trivial PFO. Pulmonology was consulted for bronchoscopy with BAL and biopsy. Microbial cell-free DNA by sequencing (Karius) testing returned on hospital day 6 and was positive for *Nocardia paucivorans* and *Nocardia abscessus*.

Conclusion: The patient received a final diagnosis of *Nocardia* pneumonia with extrapulmonary CNS involvement. He was discharged with a PICC line and IV ceftriaxone and linezolid with PO trimethoprim-sulfamethoxazole. He completed a 12-month treatment course of PO trimethoprim-sulfamethoxazole after IV induction. Multiple repeat MRIs showed resolution of the prior ring enhancing lesions. Culture and biopsy results were negative and therefore susceptibilities were not available to tailor his antibiotic regimen.

Discussion: Nocardia is a challenging clinical diagnosis, and a high index of suspicion is needed to make an accurate diagnosis especially in immunocompetent hosts with disseminated disease. As in our case, Nocardia is difficult to identify in culture and biopsy samples. Molecular testing, such as Karius, has supplanted prior diagnostic tools and clinicians should be aware of its utility, limitations, and appropriate use.

Our patient was initially treated for culture-negative infective endocarditis (CNIE) with septic emboli. While this was consistent with his presentation, especially in the setting of his prior history of CNIE, CNIE is rare accounting for only 14.2% of endocarditis cases¹. Therefore, when cultures are negative, it is reasonable to consider other diagnoses. In this case, we suspect that the patient's prior presentation was not CNIE but rather disseminated nocardiosis which was inadequately treated resulting in recurrence for this hospitalization. Similar occurrences are described in the literature, reiterating the need for clinician awareness of this rare, but endemic infection.

Sources

1. McHugh J, Saleh OA. Updates in Culture-Negative Endocarditis. *Pathogens*. 2023 Aug 10;12(8):1027. doi: 10.3390/pathogens12081027. PMID: 37623987; PMCID: PMC10459830.
2. Traxler RM, Bell ME, Lasker B, Headd B, Shieh WJ, McQuiston JR. Updated Review on Nocardia Species: 2006-2021. *Clin Microbiol Rev*. 2022 Dec 21;35(4):e0002721. doi: 10.1128/cmr.00027-21. Epub 2022 Oct 31. PMID: 36314911; PMCID: PMC9769612.
3. Xue, Kun MMA; Zhang, Anling MMb; Liu, Shuyu MMA; Chen, Dawei MDa,* . Multiple brain abscesses caused by Nocardia farcinica infection after hand injury: A case report and literature review. *Medicine* 103(29):p e39019, July 19, 2024. | DOI: 10.1097/MD.00000000000039019

#6 Cultural Barriers in Pain Assessment: A Case of an Indigenous Q'eqchi' Patient with COVID-19

Daniel S. Barrera, Medical Student, Spencer Fox Eccles School of Medicine, Salt Lake City, UT; Andrew Chang, MD, Spencer Fox Eccles School of Medicine, Salt Lake City, UT.

A 19-year-old Indigenous Q'eqchi' man with an unknown history arrived at an emergency immigration shelter at the Mexican-American border for a health evaluation. He presented with a cough and a positive COVID-19 rapid test and was promptly moved to a well-ventilated tent in isolation for the rest of his evaluation. Through a Q'eqchi' interpreter, the patient shared symptoms of sore throat, tiredness and muscle aches. When assessing pain severity using a standardized 1-to-10 pain scale, the patient appeared confused by the question and responded, "What is that?" followed by, "Yes, it hurts." Despite an explicit explanation of how a pain scale worked, assessing the patient's pain was a challenge, ultimately leaving the severity of his pain an unquantified and unmanaged symptom.

This case demonstrates the critical importance of cultural humility in clinical practice. While Western medicine's objectivity and standardization are powerful tools, this can lead to communication breakdowns when cultural context is ignored. In the Q'eqchi' culture, for example, the concept of quantifying pain on a numerical scale is foreign and ineffective, as pain is primarily described qualitatively. This barrier extends beyond pain, highlighting a fundamental clash between Western medicine's quantitative framework and a more holistic, qualitative understanding of health. For instance, the distinction between specific pathogens and generalized 'illness' caused by spiritual or communal imbalance may not be relevant. Similarly, Western-defined diagnoses like depression may not accurately capture a community's understanding of sorrow or mental anguish.

For effective, patient-centered care, clinicians are urged to practice mindful communication. When encountering confusion, it is recommended to adopt a key phrase to engage in cultural humility: "It appears our standardized questions are not being understood. Interpreter, can you advise on a culturally relevant way to assess this concern?" This vignette demonstrates that providers must empower interpreters as cultural liaisons, not just translators, to bridge gaps in understanding and deliver effective, patient-centered care.

#7 The Heart Wants What it Wants (Recurrent ICD Pocket Infection)

Author: Kathy Ding, PGY-1

Case presentation:

S.C. is a 70 year old male with past medical history significant for ischemic cardiomyopathy, HFrEF (EF 43%), monomorphic ventricular tachycardia, and T2DM who presented for R-sided ICD infection. He initially had a L-sided ICD placed after sustained monomorphic ventricular tachycardia was captured on his Holter monitor, but the site gradually developed worsening wound dehiscence and drainage concerning for underlying infection. Because of this, he underwent explantation and reimplantation on the contralateral (right) side. A month and a half later, he presented to the wound care clinic and was found to have purulent drainage and wound dehiscence from the R-sided ICD site. He was started on doxycycline for two weeks and scheduled for device explantation once again. Almost two weeks later, he returned to the wound care clinic after playing an intense game of pickleball and was still found to have frank pus draining from the ICD site despite compliance with the antibiotic regimen. He was ultimately routed to the ED by his wound RN. Patient was admitted to the cardiology unit for further evaluation.

Physical Exam:

He was pleasant and sitting comfortably. His vitals were all within normal limits (afebrile, normotensive, not tachycardic). On physical exam, the patient was A&Ox3 and not in acute distress. He had bilateral wound dressings on the R and L side. When the R-sided dressing was removed, frank yellowish-green pus drained from his ICD site with mild surrounding erythema and tenderness to palpation. Otherwise, there were no other remarkable exam findings.

Lab/Imaging Results:

His lactate in the ED was 1.4 and WBC was 8.59. BMP was stable. Blood cultures had no growth to date. Superficial wound culture yielded moderate white blood cells and rare staphylococcus epidermidis.

Intra-operative wound cultures yielded no white blood cells and rare staphylococcus capitis. TTE showed reduced LVEF (43% ->33%) but otherwise demonstrated no evidence of vegetations or growths.

Treatment:

On admission, he was started on vancomycin and zosyn for broad coverage, but after ID was consulted, both were peeled off to optimize intra-operative culture results. He then underwent explanation of the R-sided ICD without complications. Post-procedure, he was given two more doses of vancomycin and was discharged with a 12-day regimen of linezolid (total 14-day course) with standard monitoring and close follow-up. As for the cardiac aspect, he was discharged with a life vest with plans for possible ICD reimplantation after a few months pending healing and resolution of his current infection.

Conclusion:

ICD pocket infection is quite rare at baseline with only 1-2% of patients developing ICD infections within the first 6 months of placement¹. This case is made more unique by the fact that the patient was not

immunocompromised or on any immunosuppressive medications. He also remained HDS during the entire duration of the recurrent infections and had negative wound cultures throughout as well. The case underscores the complex decision-making that goes into creating an optimal plan for this patient who is both at higher risk for sudden cardiac death given his history of monomorphic ventricular tachycardia and also has serious risks and potential complications associated with his recurrent ICD pocket infections.

References:

1. Prutkin JM, Reynolds MR, Bao H, Curtis JP, Al-Khatib SM, Aggarwal S, Uslan DZ. Rates of and factors associated with infection in 200 909 Medicare implantable cardioverter-defibrillator implants: results from the National Cardiovascular Data Registry. *Circulation*. 2014 Sep 23;130(13):1037-43. doi: 10.1161/CIRCULATIONAHA.114.009081. Epub 2014 Jul 31. PMID: 25081281.

#8 Acute Onset Meat Allergy vs Alpha-Gal Syndrome, a Historical Conundrum

Josh Hales, MD candidate 2028, Dr. Sonja Raam, MD, Spencer Fox Eccles School of Medicine at the University of Utah, Salt Lake City, UT

Adult-onset meat allergy, particularly Alpha-Gal Syndrome (AGS), is increasingly prevalent in North America. While distribution of the Lone Star Tick, and diagnostic challenges may make diagnosis difficult, a quality history can ensure it is not missed on the differential.

A 59-year-old, Spanish-speaking woman with a history of migraines with aura presented with three-year history of episodic abdominal pain, nausea, vomiting, and diarrhea. The patient initially presented 3 months prior where symptoms were attributed to migraine-related gastrointestinal symptoms. She was referred for endoscopy, which she deferred due to financial concerns. On follow-up, she reported persistent symptoms. A detailed review of symptoms and dietary history revealed symptom onset after consuming fatty meats such as beef, pork, and fish, with symptom onset around 1 hour after ingestion. She also described intermittent throat fullness, raising suspicion for an allergic etiology. Her social history was notable for frequent travel between Utah and Sinaloa, Mexico, a known domain for Lone Star ticks. She recalled a tick bite prior to symptom onset. Meat allergy and alpha-gal antibody testing were obtained, revealing positive mammalian meat-specific IgE but negative alpha-gal IgE.

Patient was offered referral to Gastroenterology and Allergy but declined further specialty evaluation and chose strict avoidance of mammalian meats, which remains her primary management strategy. Based on symptoms and history, AGS was considered the primary diagnosis, but this was complicated by a negative alpha-gal IgE test. However, the test could not exclude the diagnosis entirely, as some studies show alpha-gal IgE testing to have lower sensitivity in female patients.

This case emphasizes the diagnostic challenges with alpha-gal and the need for further research in the area. It further emphasizes the importance of detailed history taking. Upon reviewing past notes the use of a qualified Spanish interpreter was not identified. Details including a gastrointestinal review of symptoms and travel history were also omitted. These factors created a barrier in communication and limited earlier providers' ability to see the full clinical vignette which led to a delay in diagnosis. As AGS become more prevalent in North America, it is essential that physicians understand major symptoms and associated historical components for diagnosis.

#9 Toxicity as a Biomarker: Lessons from a Case of Severe irAEs in Melanoma

Case Narrative

A 63-year-old man initially presented to the Huntsman Cancer Institute in February 2025 with a biopsy-proven left axillary lymph node metastasis of BRAF V600E-positive melanoma. Baseline imaging showed no distant metastases. He received one cycle of pembrolizumab (400 mg), but restaging revealed multiple new osseous metastases, prompting escalation to ipilimumab (3 mg/kg) plus nivolumab (1 mg/kg).

Shortly after the first cycle, he developed grade 3 hypophysitis and thyroiditis, confirmed by MRI pituitary enlargement and endocrine testing showing central adrenal insufficiency and hyperthyroidism. He required high-dose corticosteroids and methimazole. Despite ongoing prednisone 80 mg daily, cycle 2 was administered due to rapid disease progression.

Soon after, he was hospitalized with persistent fever, cytopenias, ferritin >8,900 ng/mL, and elevated soluble IL-2 receptor—consistent with immune checkpoint inhibitor–induced hemophagocytic lymphohistiocytosis (HLH). He also developed grade 3 hepatitis (AST/ALT >900 U/L, total bilirubin 15.4 mg/dL), requiring addition of a second immunosuppressant mycophenolate mofetil, and a pulmonary embolism necessitating anticoagulation. Radiation therapy was delivered to painful osseous lesions.

Despite these multisystem immune-related adverse events (irAEs) and early discontinuation of combination therapy, repeat PET/CT in July 2025 demonstrated a metabolic response, with resolution of axillary nodal disease and decreased FDG uptake in osseous metastases.

Discussion

This patient's clinical course highlights the toxicity–efficacy paradox of immune checkpoint inhibitors (ICIs). The paradox refers to the observation that patients who develop irAEs are more likely to experience superior tumor responses and survival compared to those without such toxicities. The biological rationale is that as checkpoint blockade unleashes T-cell activation against tumor antigens, this same activation can cross-react with self-antigens, causing autoimmune-like toxicity (Weinmann et al. 2019).

Several studies support this phenomenon. In a large cohort of metastatic melanoma patients receiving ipilimumab plus nivolumab, development of hepatitis or hypophysitis correlated with significantly improved overall survival (Al Remawi H et al, 2025). Similarly, pituitary dysfunction has been associated with better survival outcomes in both melanoma and lung cancer patients (Kobayashi T et al., 2020). Long-term data from CheckMate-067 demonstrate that durable responses persist even when therapy is discontinued early due to toxicity (Wolchok et al., 2024).

Clinically, the paradox underscores the need for prompt recognition and treatment of irAEs while maintaining oncologic vigilance. Data suggest that early immunosuppression with corticosteroids or second-line agents such as mycophenolate does not negate long-term efficacy. On the other hand, another analysis of ipilimumab-induced hypophysitis found that patients treated with lower total doses of glucocorticoids had longer overall survival compared to those receiving high doses, suggesting that overly aggressive

immunosuppression may blunt efficacy (Faje AT et al., 2018). These data suggest prompt initiation of immunosuppression upon severe irAEs and tapering patients off as soon as they can tolerate is important in overcoming toxicities while maintaining clinical benefit.

In this case, a profound immune activation resulted in severe multisystem autoimmunity—hypophysitis, HLH, hepatitis, thyroiditis, and thromboembolic complications—yet coincided with measurable tumor control. The patient’s course exemplifies the toxicity–efficacy paradox and highlights the importance of balancing aggressive toxicity management with careful monitoring for durable oncologic benefit.

References

Weinmann SC, Pisetsky DS. Mechanisms of immune-related adverse events during the treatment of cancer with immune checkpoint inhibitors. *Rheumatology (Oxford)*. 2019;58(Suppl 7):vii59-vii67. doi:10.1093/rheumatology/kez308

Al Remawi H, Lindén M, Zhao Z, et al. Immune-related hepatitis and hypophysitis are associated with superior survival in melanoma patients treated with combined ipilimumab and nivolumab. *Oncoimmunology*. 2025;14(1):2543510. PMID: 40778883.

Kobayashi T, Iwama S, Yasuda Y, et al. Pituitary dysfunction induced by immune checkpoint inhibitors is associated with better overall survival in both malignant melanoma and non–small cell lung carcinoma: a prospective study. *J Immunother Cancer*. 2020;8(2):e000779. PMID: 32606047.

Wolchok J, Chiarion-Sileni V, et al. Ten-Year Survival Outcomes With Nivolumab Plus Ipilimumab in Advanced Melanoma: The CheckMate 067 Trial. *N Engl J Med*. 2024;391(12):1075-1086. doi:10.1056/NEJMoa2402625. PMID: 39052724

Faje AT, Lawrence D, Flaherty K, et al. High-dose glucocorticoids for the treatment of ipilimumab-induced hypophysitis is associated with reduced survival in patients with melanoma. *Cancer*. 2018;124(18):3706-3714. PMID: 29975414.

#10 From Loose Stool to Solid Diagnosis

Rachel Pernick, MS3 – The University of Utah School of Medicine Salt Lake City, UT Arindam Sharma, MD – Advanced Heart Failure and Transplant Cardiology, Intermountain St. George Regional Hospital Saint George, UT

Case Presentation

A 66-year-old male with PMHx of hypertension was referred to gastroenterology for evaluation of a 4-month history of intermittent diarrhea and bloating. Workup, including food allergy panel, testing for celiac disease, hydrogen breath test, stool studies, colonoscopy, and EGD, was unrevealing. Six months later, with persistent symptoms and 25lb unintentional weight loss since first seeking care, he was diagnosed with irritable bowel syndrome and advised to follow-up with a dietician. Two months later, upon routine visit with his primary care provider, he was noted to have a new murmur and sent for an echocardiogram. The echocardiogram showed torrential (5+) tricuspid regurgitation, moderate pulmonary valve (PV) stenosis, and moderate to severe PV regurgitation. The right ventricle was moderately dilated. The left ventricular ejection fraction was normal (64%) without left-sided valvular abnormalities. These significant isolated right-sided valvular abnormalities were concerning for carcinoid heart disease, prompting referral to cardiology. By this time he had developed pedal edema and exertional dyspnea and fatigue.

Physical Exam/Labs/Imaging

Physical exam in the cardiology clinic revealed elevated JVD to at least 15cm and a harsh 4/6 holosystolic murmur in the left 3rd and 4th intercostal spaces. The liver was palpable to at least 5cm below the costal margin. Lab testing revealed elevated proBNP at 1,719 pg/mL, serum serotonin at 2,884 ng/mL, chromogranin A at 3,835 ng/mL, and urinary 5-HIAA at >938 mg/day. CT scan of the abdomen and pelvis revealed bulky hepatic masses, the largest measuring 11.8 x 9.2 cm. Enlarged mesenteric lymph nodes were also noted. Cardiac MRI confirmed significant right-sided abnormalities.

Management

The patient was referred to Medical Oncology and underwent Dotatate PET/CT scan, which noted multiple large liver masses, a 4.5 x 2.4 cm soft tissue mass within the mesentery, and additionally noted lesions within the lungs and vertebral bodies. Liver biopsy showed metastatic well-differentiated neuroendocrine tumor. He subsequently initiated treatment with octreotide and was referred for pulmonary and tricuspid valve replacement.

Discussion

Diarrhea and GI upset are common clinical entities, and differential diagnosis is broad, including malabsorptive, secretory, inflammatory, and functional disorders. Initial workup typically includes labs and stool studies, but further workup is guided largely by clinical suspicion. A thorough history and physical examination can narrow the differential and guide further evaluation. Physical exam, therefore, can be critical to diagnosis. In this case, physical exam findings—the presence of a new murmur and massive hepatomegaly—

were critical in diagnosis. While diarrhea is a common problem, a new murmur in a patient with chronic diarrhea should prompt consideration of a rarer entity, carcinoid syndrome.

#11 Anchored to Ovarian Cancer: A Case of Myeloid Sarcoma That Set Sail in a Different Direction

Edward Compton, M.D.¹, James Willey, M.D.¹

¹Division of Internal Medicine, University of Utah, Salt Lake City, UT, USA

Case Presentation: A 19-year-old woman presenting to the emergency department with subacute onset of abdominal distension and diffuse abdominal pain. She has no pertinent past medical history. Associated symptoms include poor appetite, vomiting, shortness of breath, and decreased urine output. Her last menses was approximately 2 weeks before presentation. Eleven days prior to presentation, she was seen in an outpatient gynecology clinic with similar complaints. Pelvic ultrasound revealed a 2.1x2.2cm heterogeneous cystic structure in the right ovary and moderate echogenic fluid in the pelvis, which was thought to represent a ruptured hemorrhagic cyst with hemoperitoneum. The left ovary had a small, thick-walled cyst vs. corpus luteum.

Physical Exam: On presentation, the patient was afebrile, blood pressure was 150/113, with a heart rate of 97. She appeared healthy and physically comfortable but anxious. The exam was notable for decreased breath sounds in the lower posterior lung fields. The abdomen was significantly distended and firm, with pain on deep palpation, but without guarding or peritoneal signs.

Diagnostic Work-up: Notable for acute kidney injury with a serum creatinine of 2.28 mg/dL. LDH was elevated to 877 U/L with otherwise unremarkable serum ovarian tumor markers. CT abdomen and pelvis demonstrated large-volume ascites, diffuse thickening of the peritoneum, large ovaries, moderate right hydronephrosis with ureter dilation, and mild left hydronephrosis. MRI pelvis demonstrated complex cystic ovarian structures with extensive peritoneal carcinomatosis. Diagnostic and therapeutic paracentesis was performed on hospital day 1, removing 4L of milky-red fluid. Ascitic fluid studies indicated exudative ascites, with a total cell count of 34,230 cells/uL—flow cytometry of the ascitic fluid returned on hospital day 2, demonstrating atypical myeloid blasts consistent with acute myeloid leukemia/myeloid sarcoma.

Clinical Course/Treatment: On admission, given initial concern for a primary ovarian tumor, work-up was directed toward gynecologic malignancy, and Gynecology was consulted. The milky appearance of the patient's ascitic fluid prompted workup for a primary hematogenous malignancy with ascitic fluid flow cytometry. Overnight hospital day 1, the patient's MRI results were automatically released to the patient. Using ChatGPT to interpret the results, the patient self-diagnosed a late-stage ovarian tumor. This prompted a tense discussion with the cross-covering intern, who adeptly refocused the patient on the need for results before establishing a definitive diagnosis, which returned the following afternoon. Her acute kidney injury was likely due to obstructive uropathy in the setting of large-volume ascites. Her serum creatinine remained elevated despite paracentesis and IV fluids and on hospital day 2, a right percutaneous nephrostomy tube was placed. Based on flow cytometry results, she was transferred to oncology for chemotherapy.

Discussion: We present a case of acute myeloid leukemia/myeloid sarcoma with extensive peritoneal carcinomatosis and ascites, mimicking a primary ovarian tumor. It highlights the diagnostic error of premature closure due to anchoring bias and the ethical concerns of immediate patient results or "open notes" in the era of AI. Caution is necessary when interpreting a patient's current presentation based solely on imaging results without considering other potential diagnoses.

#12 Anaplastic in Appearance: A Rash with Hidden Depths

Annie Galt, MD, Internal Medicine/Pediatrics PGY-1 University of Utah Health

Case Description & Work-Up:

A 44-year-old man with hypertension, depression, and obstructive sleep apnea presented with a painful, erythematous rash involving more than 90% of his body. It is accompanied by skin sloughing, malaise, low-grade fevers, night sweats, and weight loss. He had a history of recurrent localized rashes over the past five years, with prior biopsies showing pityriasisiform dermatitis. These rashes had resolved uneventfully with topical steroid administration. He had no significant family history, no reported allergies, and lived in Wyoming. On examination, he had diffuse erythema of the face, neck, trunk, and extremities with trace scale on proximal extremities, palmoplantar hyperkeratosis, and fissuring. Right inguinal lymphadenopathy was palpable; otherwise, the physical exam was unremarkable.

The original workup consisted of two skin biopsies that showed nonspecific spongiosis. CT of the abdomen/pelvis revealed diffuse retroperitoneal, mediastinal, and inguinal lymphadenopathy. PET-CT demonstrated diffuse FDG-avid adenopathy with splenic and bone marrow uptake. Laboratory evaluation showed elevated LDH, ESR, and CRP, with negative HIV and Hepatitis C serologies, EBV DNA <1.54 log IU/mL. Peripheral flow cytometry and T-cell clonality studies were unremarkable. It was assumed to be idiopathic erythroderma with reactive lymphadenopathy at that time, and the patient was trialed on cyclosporin without much improvement. A lymph node biopsy was performed, and while pathology was pending, the patient was trialed on high-dose steroids, again without significant improvement. Eventually, the inguinal lymph node biopsy confirmed ALK-negative, CD30+ anaplastic large cell lymphoma (ALCL) without evidence of cutaneous lymphoma involvement. The patient was initiated on Brentuximab Vedotin – Cyclophosphamide, Doxorubicin, and Prednisone therapy.

Discussion:

Erythroderma, defined by erythema and scaling involving more than 90% of the skin surface, can arise from a variety of causes. Most commonly, it represents a generalization of pre-existing dermatoses such as psoriasis or atopic dermatitis. Still, it may also occur secondary to drug reactions, cutaneous T-cell lymphoma (CTCL), or other lymphoproliferative processes.¹ Identifying the underlying etiology is essential, as management and prognosis vary widely. We present a case of erythroderma secondary to ALK-negative anaplastic large cell lymphoma (ALCL) without cutaneous T-cell lymphoma, illustrating the diagnostic complexity and paraneoplastic manifestations of this T-cell lymphoma.

This case highlights a diagnostically challenging cause of erythroderma, characterized by non-specific skin biopsies and refractoriness to empiric therapies. This rare paraneoplastic manifestation of ALK-negative ALCL presented with severe erythroderma in the absence of cutaneous lymphoma involvement. While generalized dermatitis and drug-induced exanthems are common causes of erythroderma, malignancy-associated

etiologies should be considered when the rash is refractory to immunosuppressive therapy and accompanied by systemic findings such as lymphadenopathy and splenomegaly. Clinicians should have a low threshold to evaluate for the presence of these features, especially when these may be less clinically apparent in patients with a larger body habitus. ALK-negative ALCL is an aggressive subtype of T-cell lymphoma associated with poorer outcomes than ALK-positive ALCL. The absence of cutaneous infiltration supports a paraneoplastic inflammatory mechanism rather than direct tumor involvement. Paraneoplastic erythroderma is a less common clinical feature of this disease, and can make diagnosis challenging, as standard preparation of skin biopsies alone will not be able to diagnose the underlying etiology.

¹ Harper-Kirksey K. Erythroderma. *Life-Threatening Rashes*. 2018 Sep 12:265–77. doi: 10.1007/978-3-319-75623-3_19. PMID: PMC7139437.

#13 An Uncommon Complication of a Common Condition

Taylor Thompson, Spencer Fox Eccles School of Medicine

Case Description: A 28-year-old male with hyperthyroidism, previously diagnosed but not currently on medication, who presented to the ER after waking up that morning unable to move his legs and lower torso. He had no recent trauma or injury. The lower extremity weakness was equal in both legs and was associated with pain on movement. He had no deficits in his upper extremities.

Physical exam: Initial vital signs were notable for tachycardia. He was alert and oriented x3, and cranial nerves II-XII were grossly intact. His neurological exam was notable for $\frac{2}{5}$ hip flexion strength bilaterally, $\frac{2}{5}$ knee flexion strength bilaterally, intact patellar tendon reflexes, and no sensory deficits.

Labs/Imaging: Initial labs were notable for a potassium of 2.1, free T4 of 5.29, and undetectable TSH.

Clinical course: Given the patient's constellation of history, symptoms, and lab findings, a presumptive diagnosis of thyrotoxic periodic paralysis was made. The patient's potassium was carefully repleted intravenously. His lower extremity weakness improved with the normalization of his serum potassium levels. He was tested for thyroid-stimulating hormone receptor antibodies, which returned elevated. After two days of inpatient care, he was discharged home with plans for close endocrinology follow-up.

Treatment: The patient was diagnosed with Graves' disease and started on methimazole as well as propranolol for acute symptom management.

Conclusion: Thyrotoxic periodic paralysis is a rare complication of hyperthyroidism, occurring with very low incidence among affected patients and most commonly reported in Asian populations. Interestingly, although hyperthyroidism is more prevalent in women, over 95% of cases occur in men. The pathophysiology is not fully understood but involves altered electrolyte flow across skeletal muscle cells. Excess thyroid hormone directly activates and increases

beta-adrenergic stimulation of the sodium-potassium ATPase pump in skeletal muscle cells. This drives potassium into cells in exchange for sodium, causing hyperpolarization of the cell membrane and impaired depolarization, resulting in flaccid paralysis. If left untreated, this can progress to respiratory failure, cardiac arrhythmias, and even death. Because its clinical presentation can mimic other neuromuscular or metabolic disorders, prompt recognition is crucial. This underscores the importance of maintaining a broad differential and obtaining a thorough history when evaluating patients with common chronic conditions.

Source: Pompeo, Arsenio, et al. "Thyrotoxic hypokalemic periodic paralysis: An overlooked pathology in western countries." *European Journal of Internal Medicine*, vol. 18, no. 5, Sept. 2007, pp. 380–390, <https://doi.org/10.1016/j.ejim.2007.03.003>.

#14 Too Blue to Be True: A Case of Disproportionate Hypoxemia

Case Description:

A 25-year-old previously healthy man presented with fever, conjunctivitis, painful oral/genitourinary mucositis, and truncal rash after one week of upper respiratory symptoms. Respiratory PCR detected *Mycoplasma pneumoniae* and he was diagnosed with Mycoplasma-induced rash and mucositis (MIRM). He was admitted for management with corticosteroids, ocular care, and supportive therapy. On hospital day (HD) 11, he developed profound acute hypoxic respiratory failure, requiring maximal settings on high-flow nasal cannula to maintain SpO₂ >90%. Despite his respiratory decompensation, he remained hemodynamically stable with normal blood pressures.

Differential:

In a patient with mycoplasma pneumonia and MIRM who develops progressive hypoxemia, initial considerations included pneumonia progression, airway compromise due to mucositis progression, aspiration, acute respiratory distress syndrome, or pulmonary embolism (PE).

Treatment and Resolution:

CT angiography of the chest showed extensive bilateral pulmonary emboli, including a large central thrombus in the right pulmonary artery with evidence of right-heart strain and pulmonary infarct. Point-of-care echocardiography was positive for McConnell's sign, and cardiac biomarkers were elevated. Despite severe hypoxemia, large burden of thrombus, and right heart strain, his blood pressure remained preserved. This prompted concern for right-to-left intracardiac shunting. Catheter-directed thrombolysis was selected over mechanical thrombectomy to reduce the risk of intraoperative paradoxical embolus. This resulted in improvement in the patient's hypoxemia and cardiac biomarkers. He was anticoagulated with enoxaparin, then transitioned to a 3-month course of apixaban for provoked PE. Formal cardiac echo with contrast showed a large amount of shunting from right to left both at rest and with valsalva, confirming the presence of a patent foramen ovale (PFO). Following adequate improvement in his mucositis and conjunctivitis, he was discharged on HD 23 in stable condition.

Discussion:

This case illustrates several teaching points. First, acute infection and systemic inflammation (in this case, MIRM) may transiently heighten thrombotic risk, leading to venous thromboembolism even in young and otherwise healthy adults. Second, the presence of a PFO can significantly alter the clinical presentation PE. PFO is the most common structural cardiac abnormality, and while patients with PFO are often asymptomatic they are at risk for right to left intracardiac shunting. A large PE typically will present with shock and hypotension. However when a PFO is present, right-to-left intracardiac shunting can preserve left ventricular filling pressures and systemic perfusion while exacerbating hypoxemia. As such, PE with hypoxemia disproportionate to hemodynamic stability warrants evaluation for intracardiac shunt. In the case of suspected intracardiac shunt, special consideration should be given to advanced therapies such as

thrombectomy or thrombolysis, as the typical qualifying criteria of hypotension and may be absent. Lastly, it should be considered that mechanical thrombectomy carries a risk of embolizing the thrombus into arterial circulation through the PFO.

#15 Recurrent Viremia and Immunosuppression Optimization in an EBV D+/R- Mismatch Kidney Transplant Patient

Author: Kendon Holdaway, Spencer Fox Eccles School of Medicine (University of Utah)

Case: KH is a 27-year-old male who is status-post living, related-donor kidney transplant with a past medical history of end-stage renal disease secondary to congenital renal anomaly (oligomeganephronia) and secondary FSGS. The patient was transplanted at the Mayo Clinic on September 12, 2018, with donor-recipient workup including negative crossmatch, 11/12 antigen mismatch, and Epstein Barr mismatch (D+/R-). In addition to Simulect induction intraoperatively, the patient's immunosuppressant regimen includes tacrolimus, mycophenolate mofetil, and maintenance steroid protocol following a standard post-operative taper. On 3/15/19, approximately 6 months post-transplant, the patient presented to the Emergency Department with complaints of white tonsillar and pharyngeal exudate in addition to odynophagia and lymphadenopathy. After an extensive workup, the etiology was determined to be infective mononucleosis secondary to Epstein-Barr viral infection. Patient's work-up for secondary malignancy and post-transplant lymphoproliferative disorder was negative, and infection promptly resolved with temporary elimination of mycophenolate mofetil and doubling prednisone to 10 mg/day. Cautious reintroduction of mycophenolate in April 2019 quickly resulted in recurrent Epstein Barr viremia without patient-reported symptoms and ultimately led to successful IgG seroconversion on 12/26/19. However, despite this reduction in immunosuppressive burden and consequent adaptive immune response, the patient once again tested positive for recurrent Epstein Barr viremia in both 2020 and 2021.

Impact/Discussion: Epstein Barr Viremia is a common post-transplant complication, with occurrence in upwards of 67% of transplant patients¹. Even though upwards of 90% of the general population exhibits antibody protection against Epstein Barr Virus, Donor-Recipient mismatch potentiates high-risk for the EBV-naive recipient, with increased risk of infection, post-transplant lymphoproliferative disorder, consequent sequelae (reduction in immunosuppression, increased risk of rejection and organ failure, greater need for close monitoring)³⁻⁴. Despite this known risk, many transplant centers across the United States (and world) adhere to different guidelines regarding treatment of Epstein Barr Viremia².

Conclusion: EBV D+/R- mismatch remains a challenging complication of transplant in young patients, particularly demonstrated by this case of consistent, low-level EBV viremia after spontaneous seroconversion. This is further exacerbated by frequent transition of care between transplant centers, private practice, and academic centers.

This case highlights the challenge of balancing immunosuppressive burden while considering patient quality of life and long-term graft survival. Evidence-based, standardized care is needed to reduce variability of care and improve effective implementation in these patient populations.

Disclosure: The author is also the patient described in this case report and has provided informed consent for publication.

#16 Seizing the Diagnosis: A Rare Case of Fatal West Nile Virus Ventriculitis

Matthew J Cobler-Lichter, MD^{1,2}, Connie Chung, MD², Madison Bangert, MD³, Stephanie S Gelman, MD³, Peter E. Schloesser, MD⁴, Shawn Smith, MD², Kevin Meier, MD², Dean Roller, MD², Evan Gross, MD⁵, Kyle Hobbs, MD²

¹Department of Neurology, University of Utah, Salt Lake City, UT, USA; ²Department of Neurosciences, Intermountain Medical Center, 5121 Cottonwood Street, Murray, UT, 84107, USA; ³Division of Infectious Diseases, Intermountain Medical Center, Murray, Utah, USA; ⁴Department of Radiology, Intermountain Medical Center, Murray, Utah, USA; ⁵Department of Internal Medicine, University of Utah, Salt Lake City, UT, USA

Case

An 81 year-old male with chronic pain was admitted to the hospital with increasing weakness, nonbloody diarrhea, emesis, and fever the days prior. He presented afebrile, alert and oriented, with unremarkable labs. The following morning, he had two seizures and received lorazepam and levetiracetam load prior to intubation and transfer to a quaternary care hospital. He presented afebrile with WBC of 15, which normalized over the next few days. Upon arrival, he was not following commands and had slight withdrawal of extremities to pain. Initial EEG showed slow, poorly organized, poorly reactive background with no seizures or epileptiform discharges. MRI showed diffusion restriction in the lateral ventricles, prompting empiric treatment for presumed ventriculitis. Lumbar puncture was notable for glucose 54, protein 248, and WBC 124 with 48% lymphocytes. On day two of admission, the patient became persistently febrile to 39°C and intermittently tachypneic. HIV testing was negative. He tested positive for West Nile Virus (WNV) IgG/IgM in serum and WNV IgG in CSF, with positive WNV PCR. St. Louis Encephalitis Virus (SLEV) IgG was also positive in serum. IVIG and dexamethasone were started and antimicrobials discontinued. After two weeks of little improvement, he was extubated, transitioned to comfort measures, and died the next day.

Discussion

This appears to be only the third case reported in the literature of WNV causing ventriculitis (1) (2). Furthermore, WNV causes seizures in only 3-6 % of patients (3). Similar antigenic profiles of flaviviruses such as WNV and SLEV result in antibody cross-reactivity, which necessitates confirmatory testing. Plaque-reduction neutralization testing (PRNT) is the most specific test to detect antibodies to arboviruses, but is only available at a limited number of laboratories (4). The elderly are the most vulnerable to severe neuroinvasive WNV. Treatment remains limited to supportive therapy.

Citations

1. Shetty N, Batra A, Kim M. West Nile Virus Encephalitis Associated with Intraventricular Diffusion Restriction (P4.9-029). *Neurology*. 2019;92(15_supplement):P4.9-029.

2. Dammann C, Arar A, Klimenko M. Hydrocephalus due to Pyogenic Ventriculitis in West Nile Encephalitis *Journal of Hospital Medicine* 2022;Abstract E43.
3. Gould CV, Staples JE, Guagliardo SAJ, Martin SW, Lyons S, Hills SL, et al. West Nile Virus: A Review. *Jama*. 2025;334(7):618-28.
4. Miller JM, Binnicker MJ, Campbell S, Carroll KC, Chapin KC, Gonzalez MD, et al. Guide to Utilization of the Microbiology Laboratory for Diagnosis of Infectious Diseases: 2024 Update by the Infectious Diseases Society of America (IDSA) and the American Society for Microbiology (ASM). *Clinical Infectious Diseases*. 2024.



#17 An unexpected side effect: the heartache of drug-induced lupus

Matt Glasgow, MD and Alekses Clifton, MD; Department of Internal Medicine, University of Utah

Case description:

44-year-old female with history of peripheral spondylarthritis on infliximab, hyperlipidemia, and type 2 diabetes mellitus presented to urgent care with chest pain and cough 3-days after her second infliximab infusion. She was diagnosed with community-acquired pneumonia (CAP) and discharged with amoxicillin. The patient's symptoms worsened over the course of the following week, and she went to the emergency department. CT chest showed a left-sided, loculated pleural effusion with adjacent atelectasis and small pericardial effusion. The patient was admitted to the hospital for IV antibiotics for treatment of CAP. Echocardiogram demonstrated a small, free-flowing pericardial effusion. The patient was discharged on amoxicillin-clavulanic acid for 14 days with a plan for repeat CT in 4 weeks to monitor presumed parapneumonic effusion. One week after discharge, the patient had recurrence of chest pain exacerbated by exertion and supine positioning, with new progressive shortness of breath, orthopnea, fever, and a rash on her anterior chest.

She returned to the ED where vital signs were notable for tachycardia, and labs showed WBC 12, CRP 26.2, and ESR 49. Exam was notable a well-demarcated erythematous rash over the anterior chest that spared the clothing lines. ECG demonstrated sinus tachycardia with non-specific T-wave changes throughout all leads. CT chest showed slight improvement in left-pleural effusion without consolidations or opacities, but persistent loculations and small pericardial effusion. Point-of-care ultrasound demonstrated small pericardial effusion.

A broad autoimmune and infectious work-up demonstrated anti-double stranded DNA antibody titer of 1:640 and antinuclear antibody titer of >1:2560. The patient had undetectable anti-histone antibodies. Cardiac MRI showed a trace circumferential pericardial effusion with marked pericardial thickening and diastolic restraint of the LV free walls visually. Ultimately, the patient was diagnosed with constrictive pericarditis secondary to drug-induced lupus from infliximab. She was started on a 3–6-month course of colchicine, prednisone, and high dose ibuprofen.

After several days of treatment in the hospital, the patient had complete resolution of chest pain and tachycardia. Anti-infliximab antibodies (AIs) were detectable. Repeat cardiac MRI 5 months after discharge demonstrated complete resolution of constrictive physiology and pericardial thickening. At her follow-up appointment, the patient remained symptom-free with improving exercise tolerance.

Discussion:

Drug-induced systemic lupus erythematosus (DILE) is an uncommon, but documented side effect of TNF-alpha inhibitors such as infliximab [1]. This patient's presenting symptoms were related to severe pericarditis with constrictive physiology. However, DILE is more likely to present with isolated subacute cutaneous manifestations without systemic involvement compared with idiopathic SLE [2]. Furthermore, anti-histone antibody is classically elevated DILE and a vast majority will have a positive ANA titer at the time of diagnosis [2]. While this patient had a markedly elevated ANA titer, her anti-histone antibody was undetectable, and her anti-dsDNA antibody was positive. This pattern is more in line with idiopathic SLE, but TNF-alpha inhibitor-

related DILE has been shown to present with a positive anti-dsDNA antibody and negative anti-histone antibody more commonly than other medications implicated in DILE [1]. Lastly, AIAs are associated with lupus-like autoantibody production, but the correlation between AIAs and clinical DILE is not well established.

This patient's progressive dyspnea and orthopnea were key historical features that prompted us to consider constrictive or tamponade physiology, particularly in the setting of persistent tachycardia. The ACC emphasizes the importance of prompt evaluation for tamponade or constriction in the patients with pericarditis and evidence of heart failure [3]. This patient was diagnosed quickly after the development of symptomatic constriction which likely contributed to a favorable outcome.

The patient's pericarditis rapidly improved with triple therapy including colchicine, high-dose NSAIDs, and steroids. Steroids have moderate evidence in the treatment of pericarditis but may have a more robust role in the treatment of severe pericarditis related to a flare of an autoimmune condition such as SLE [3, 4].

References:

1. Beigel F, Schnitzler F, Paul Laubender R, Pfennig S, Weidinger M, Göke B, Seiderer J, Ochsenkühn T, Brand S. Formation of antinuclear and double-strand DNA antibodies and frequency of lupus-like syndrome in anti-TNF- α antibody-treated patients with inflammatory bowel disease. *Inflamm Bowel Dis*. 2011 Jan;17(1):91-8. doi: 10.1002/ibd.21362. PMID: 20564536.
2. Lowe GC, Henderson CL, Grau RH, Hansen CB, Sontheimer RD. A systematic review of drug-induced subacute cutaneous lupus erythematosus. *Br J Dermatol*. 2011 Mar;164(3):465-72. doi: 10.1111/j.1365-2133.2010.10110.x. Epub 2011 Feb 17. Erratum in: *Br J Dermatol*. 2014 Apr;170(4):999. Lowe, G [corrected to Lowe, G C]. PMID: 21039412.
3. Mazio M, Gaita F, LeWinter M. Evaluation and Treatment of Pericarditis: A Systematic Review. *JAMA*. 2015;314(14):1498–1506. doi:10.1001/jama.2015.12763
4. Wang, T, Klein, A, Cremer, P. et al. 2025 Concise Clinical Guidance: An ACC Expert Consensus Statement on the Diagnosis and Management of Pericarditis: A Report of the American College of Cardiology Solution Set Oversight Committee. *JACC*. null2025, 0 (0).

#18 When Antihypertensives Leave a Mark: Amlodipine-Associated Schamberg Purpura

Authors: Eliza Neal¹, Vidya Gopinath¹, Anne Cioletti¹

Affiliations: ¹University of Utah Health, 50 Medical Dr. North, Salt Lake City, Utah, 84132

Case description:

This patient was a 75-year-old female with a past medical history of hypertension, hyperlipidemia, anemia, and depression who contacted our primary care team over concerns for an eczema flare. She noted a month-long history of hyperpigmentation and occasional urticaria in her bilateral lower extremities, predominantly along her calves. She was seen by an external dermatology APRN first, who expressed concerns about possible drug reaction to amlodipine and prescribed triamcinolone. Upon evaluation by our team two weeks later, the patient had stopped taking amlodipine; however, she saw no appreciable difference using triamcinolone. At this visit, her vitals were normal, and her physical exam was unremarkable, with negative cardiopulmonary or abdominal findings. Her skin exam was notable for an ill-defined, hyperpigmented, dull purpuric rash with “Cayenne pepper” type macules in the bilateral shins (Figures 1,2). Lower leg edema was absent. E-consult to dermatology was placed, and amlodipine was preemptively replaced with verapamil for blood pressure control. The differential for these lesions included Schamberg’s capillaritis, other pigmented purpuric dermatoses (PPD), post-inflammatory hyperpigmentation from chronic venous stasis, or other cutaneous eruptions (scurvy, drug hypersensitivity, etc.).

Treatment and Trajectory: Per dermatology, this patient’s rash appeared most consistent with Schamberg’s capillaritis due to amlodipine use. They recommended primary control with leg swelling reduction (compression stockings, leg elevation, etc.). We ruled-out other cardiac or thrombotic reasons for leg swelling and continued with verapamil over amlodipine. Upon follow-up six months later, the patient’s blood pressure was controlled, and her rash had completely resolved, further cementing her diagnosis of Schamburg purpura due to amlodipine use. She has not seen a recurrence in this rash in the year since initial resolution.

Discussion: Schamberg disease (SD) is the most common type of PPD seen amongst children and adults. [1]. While overall incidence of SD and other PPD are low, the prevalence of these diseases is poorly understood, as eruptions generally have a benign course and are underreported. [2] SD is characterized by discrete, non-blanchable purpuric patches found commonly on the lower extremities. These purpura are caused by red blood cell extravasation from capillaries with subsequent hemosiderin deposition. While T-cell mediated immunity appears to be implicated in the pathophysiology of this disease, gravitational dependence, alcohol intake, venous stasis, and a history of diabetes or hypertension are considered aggravating factors. [1] A number of drugs have likewise been connected with the cutaneous eruption—these include common medications like aspirin, glipizide, hydralazine, nitroglycerin, and sildenafil [1]. Within the literature, there are three case reports that discuss similar episodes of SD after amlodipine use [3,4,5]. While SD can be chronic, with recurrent episodes of exacerbations and remissions [1], we saw no recurrence of amlodipine-associated SD in the literature. All patients saw full resolution of their symptoms with simple discontinuation of the offending medication. While SD represents a less common reaction to many of our most commonly prescribed

drugs, this case highlights the importance of early recognition of the disease and the utility of discontinuing offending medications to save patients from unnecessary skin biopsies or pharmacologic treatment.

Sources:

[1] Zaldivar Fujigaki JL, Anjum F. Schamberg Disease. [Updated 2023 Aug 8]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK560532/>

[2] Giovanna Aldonza Rios López, and Leonel Martín Pulido Gutiérrez. 2024. "Schamberg's Purpura: A Comprehensive Review of Clinical Manifestations, Pathophysiology, and Management Strategies". *International Journal of Medical Science and Clinical Research Studies* 4 (02):323-27. <https://doi.org/10.47191/ijmscrs/v4-i02-30>.

[3] Faria C, Henriques F, Leite J, Fernandes C. Purpura is Not Always Caused by the Anticoagulant. *Eur J Case Rep Intern Med*. 2017 Apr 27;4(3):000536. doi: 10.12890/2017_000536. PMID: 30755927; PMCID: PMC6346870.

[4] Schetz D, Kocić I. A new adverse drug reaction--Schamberg's disease caused by amlodipine administration--a case report. *Br J Clin Pharmacol*. 2015 Dec;80(6):1477-8. doi: 10.1111/bcp.12742. Epub 2015 Oct 27. PMID: 26256559; PMCID: PMC4693475.

[5] Singh G, Jain H, Lamichhane J, Gambhir HS. Amlodipine-Associated Schamberg Disease. *Am J Ther*. 2022 Nov-Dec 01;29(6):e663-e665. doi: 10.1097/MJT.0000000000001317. Epub 2021 Feb 9. PMID: 33590988.

#19 A Shadow Amongst Pathogens: The Hidden Guest

William Tang, PhD; Yu Xia, MD/PhD; Adeline Browne, MD, and Assistant Professor, University of Utah, Salt Lake City, UT

Case Presentation

A 57-year-old woman presented to the emergency department with two days of nitroglycerin-refractory chest pain and a new oxygen requirement of 10 L/min following two weeks of myalgias, nausea, diarrhea, dizziness, and vomiting. The chest pain was concerning for acute coronary syndrome, and she described the pain as similar to her typical reflux symptoms. An unremarkable electrocardiogram and serial troponin measurements ruled out acute coronary syndrome, and a normal B-type natriuretic peptide and volume findings argued against acute heart failure. Admission laboratory evaluation revealed the following: hemoglobin 18.6 g/dL, white blood count $15.7 \times 10^3/\mu\text{L}$ with reactive lymphocytosis, platelet count $48 \times 10^3/\mu\text{L}$, lactic acid 4.2 mmol/L, and alkaline phosphatase 196 U/L, findings consistent with hemoconcentration, lactic acidosis, profound thrombocytopenia, leukocytosis, and transaminitis. Urine cultures were positive for *E. coli*, and gastrointestinal pathogen testing was positive for norovirus. CT angiography ruled out pulmonary embolism and aortic dissection but showed bilateral pleural and pericardial effusions, interlobular septal thickening, and scattered ground-glass opacities. There was no evidence of hemolysis, as indicated by an elevated haptoglobin of 257 mg/dL and the absence of schistocytes that would support a microangiopathic hemolytic process contributing to her thrombocytopenia. However, leukocytosis with left shift and immunoblasts were noted. In part, her GI symptoms and septicemia could be attributed to the diagnoses of norovirus and *E. coli* urinary tract infection; however, her thrombocytopenia, pulmonary edema, and reactive lymphocytosis with immunoblasts could not be explained. Throughout her hospital course, the patient's oxygen requirements, acute kidney injury, and sepsis continued to improve with antibiotics and supportive care. Further history during her admission revealed that she worked as a campground host and routinely swept rodent droppings. Given this exposure, hantavirus infection-mediated cardiopulmonary syndrome was suspected, as her presentation included key features such as a gastrointestinal prodrome, hemoconcentration, severe thrombocytopenia, immunoblasts, pulmonary edema, and acute respiratory failure. Hantavirus infection was later confirmed by positive IgM and IgG serology. The patient was discharged with continued home oxygen needs.

Discussion

This case illustrates the diagnostic importance of environmental history and clinical pattern recognition in identifying rare infections. Hantavirus, a negative-sense single-stranded RNA virus of the *Hantaviridae* family, causes two major syndromes: hemorrhagic fever with renal syndrome (HFRS) and hantavirus cardiopulmonary syndrome (HCPS)¹. The most common pathogenic North American strain, Sin Nombre virus^{2,3}, can present as HCPS up to 39 days after exposure⁴. It is characterized by a 3-6 day prodrome⁴ consisting of fever, myalgia, and gastrointestinal symptoms followed by hypoxemic respiratory failure due to non-cardiogenic pulmonary edema⁵. The nonspecific presentation of HCPS makes early diagnosis difficult, particularly in cases with mild to moderate symptoms, as seen here, which may be underdiagnosed. High mortality rates and limited

therapeutic interventions necessitate early recognition of this syndrome, followed by appropriate supportive care and prevention of future exposures in endemic areas⁶.

Bibliography

1. Nichol ST, Spiropoulou CF, Morzunov S, et al. Genetic Identification of a Hantavirus Associated with an Outbreak of Acute Respiratory Illness. *Science*. 1993;262(5135):914–917. doi:doi:10.1126/science.8235615
2. MacNeil A, Nichol ST, Spiropoulou CF. Hantavirus pulmonary syndrome. *Virus Research*. 2011/12/01/2011;162(1):138–147. doi:<https://doi.org/10.1016/j.virusres.2011.09.017>
3. Levy DL. Hantavirus pulmonary syndrome. Outbreak of a new disease caused by a new virus. *Postgrad Med*. Mar 1995;97(3):127–30, 133–4, 139.
4. Vial PA, Valdivieso F, Mertz G, et al. Incubation period of hantavirus cardiopulmonary syndrome. *Emerg Infect Dis*. Aug 2006;12(8):1271–3. doi:10.3201/eid1208.051127
5. Moore RA, Griffen D. Hantavirus Pulmonary Syndrome. *StatPearls*. StatPearls Publishing Copyright © 2025, StatPearls Publishing LLC.; 2025.
6. Saavedra F, Díaz FE, Retamal-Díaz A, Covián C, González PA, Kalergis AM. Immune response during hantavirus diseases: implications for immunotherapies and vaccine design. *Immunology*. 2021;163(3):262–277. doi:<https://doi.org/10.1111/imm.13322>

#20 A Fine Line Between Infection and Intoxication: Valacyclovir Toxicity in End-Stage Renal Disease

Authors: Bassel Tekarli¹, Lara Hayes²

Affiliations: ¹MD Candidate, Spencer Fox Eccles School of Medicine, University of Utah; ²Assistant Professor, Hospital Medicine Section, Division of General Internal Medicine, University of Utah

Clinical Vignette:

A 76-year-old man with a history of end-stage renal disease on peritoneal dialysis presented to his family physician with a painful rash along his chest and back. Examination revealed clusters of small, fluid-filled vesicles on an erythematous base in a dermatomal distribution, consistent with herpes zoster. He was prescribed valacyclovir 500 mg BID for seven days. Two days later, he returned to the emergency department with progressive fatigue, blurry vision, dizziness, headache, and confusion. He was afebrile and without leukocytosis. Non-contrast CT of the head and neck showed no evidence of acute hemorrhage or dissection, and he was admitted for further evaluation.

On admission, the patient reported that his shingles-related pain had largely resolved and confirmed adherence to valacyclovir. On examination, cranial nerves were intact, and the rash was crusted with minimal tenderness. Urine and peritoneal studies were negative for infection, and no vitamin deficiencies were identified. He was admitted to the medicine service with a plan for lumbar puncture. On further questioning the day after admission, the patient admitted to doubling his initial dose of medication in addition to being on a twice-daily regimen that was already supratherapeutic for his renal function.

A diagnosis of drug-induced encephalopathy secondary to valacyclovir toxicity was made. Valacyclovir was discontinued, and an additional peritoneal dialysis exchange was performed to enhance drug clearance. Given his history of end-stage renal disease, the patient was counseled on the importance of medication dose adjustments and verifying prescriptions before use. His mental status returned to baseline by hospital day 2, and he was discharged in stable condition.

This case highlights the importance of differentiating herpes zoster encephalitis from valacyclovir toxicity in patients with altered mental status following shingles. The two pathologies share overlapping neurologic features, yet their management strategies are entirely opposite: antiviral escalation versus immediate discontinuation. Careful attention to dosing history, renal function, and the absence of infectious markers such as fever or leukocytosis can guide clinicians toward the correct diagnosis and appropriate intervention.

#21 Starving the Mind: Poisoned Thoughts Masking Possible Autoimmune Encephalitis

Dominique Piber¹, Zach Hart², Guillaume Lamotte³, Melissa Whipple², Roxanne Weiss²

¹Department of Psychiatry, University of Utah, Huntsman Mental Health Institute, Salt Lake City, UT;

²Department of General Medicine, University of Utah, Salt Lake City, UT; ³Department of Neurology, University of Utah, Salt Lake City, UT

Introduction

Subacute neuropsychiatric decline in older adults presents a major challenging diagnostic scenario in clinical medicine. When behavioral changes, nutritional compromise, and unexplained functional deterioration converge, the search for an underlying cause often spans multiple disciplines. This case illustrates the diagnostic journey of a patient whose progressive mental and physical decline defied initial explanation, highlighting the critical role of interdisciplinary collaboration in uncovering elusive etiologies.

Case Description

A 71-year-old man with a history of venous thromboembolism, benign prostatic hyperplasia, and gastroesophageal reflux disease presented with several weeks of progressive functional decline, dysphagia, weight loss, and behavioral changes. Prior to symptom onset, he was cognitively intact, physically active, and fully independent in all activities of daily living. Over several weeks, he developed progressive difficulty swallowing solids and liquids, resulting in an approximate 30-lb weight loss. Concurrently, he began exhibiting perseverative and paranoid thoughts centered on the belief that his family was poisoning him, further limiting his intake leading to worsening malnutrition. His family sought multiple evaluations in his rural community, including primary care and hospital visits, as well as consultations with neurology and oncology. Brain MRI and CT imaging of the head, chest, abdomen, and pelvis were unremarkable. As his condition continued to deteriorate, his care was transferred to a tertiary care center for further evaluation.

On admission, physical and neurological examinations were unremarkable, though the patient continued to endorse delusional fears of being poisoned. Laboratory evaluation revealed no metabolic, infectious, or vitamin abnormalities. EEG and repeat brain MRI with and without contrast were normal. CSF studies showed normal cell count and glucose, mildly elevated protein, and positive 14-3-3 protein, but negative RT-QuIC and dementia biomarkers. PET imaging demonstrated no metabolic abnormalities or evidence of occult malignancy. Serologic workup revealed positive ganglionic acetylcholine receptor $\alpha 3$ (AChR $\alpha 3$) antibodies at 50 pmol/L and myelin oligodendrocyte glycoprotein (MOG) antibodies at a titer of 1:10. As the clinical condition worsened — marked by decreased responsiveness and the need for enteral feeding — a multidisciplinary discussion among medicine, neurology, and psychiatry began. Upon re-evaluation of the PET scan, nuclear medicine noted hypometabolism of the occipital lobe and relatively increased activity in the cerebellum and brainstem, raising concern for autoimmune encephalitis.

The patient was treated with intravenous immunoglobulins and methylprednisolone, along with low-dose risperidone for symptom management. Over subsequent days, he demonstrated marked improvement—regaining alertness, ambulating with assistance, and, most notably, transitioning from tube feeds to oral intake. He was eventually transferred to inpatient rehabilitation, where he continued to make both cognitive and functional gains.

Discussion

Autoimmune encephalitis is a serious but potentially reversible disorder of the central nervous system characterized by subacute onset of psychiatric and neurological symptoms. The diagnosis can be challenging and requires a broad differential. Criteria for probable autoimmune encephalitis include: 1) rapid progression of symptoms within less than three months; 2) presence of new-onset seizures, CSF pleocytosis, or imaging findings compatible with inflammation; and 3) Exclusion of alternative causes. Although many cases are associated with identifiable autoantibodies, some remain seronegative or present with atypical antibody profiles. Early recognition and prompt initiation of immunotherapy are essential, as patients can experience substantial recovery with treatment, whereas delays or misdiagnosis may lead to irreversible decline and harm from inappropriate therapy.

This case underscores the diagnostic complexity of autoimmune encephalitis in the absence of classic antibody or imaging findings. The patient's positive AChR α 3 and MOG antibodies were initially perplexing, as their known clinical associations—autonomic dysfunction and demyelination—did not match his presentation. The combination of rapidly progressive neuropsychiatric symptoms, exclusion of alternative etiologies, subtle PET findings, with subsequent clinical improvement following immunotherapy supported a diagnosis of probable autoimmune encephalitis.

Importantly, this case demonstrates the essential value of multidisciplinary collaboration and diagnostic persistence in unraveling atypical presentations. Clinicians should maintain a high index of suspicion for autoimmune encephalitis in patients with subacute, unexplained neuropsychiatric decline, particularly when standard investigations are unrevealing. Timely immunotherapy and coordinated interdisciplinary care can lead to meaningful recovery and improved quality of life.

Conclusion

This case highlights the importance of maintaining clinical suspicion for autoimmune encephalitis in older adults presenting with subacute neuropsychiatric decline, even when classical diagnostic features are absent. The patient's atypical antibody profile and normal imaging initially obscured the diagnosis, but multidisciplinary reassessment and empiric immunotherapy led to substantial recovery. This underscores the necessity of cross-specialty collaboration and the value of revisiting diagnostic data in light of evolving clinical findings. Early recognition and prompt initiation of immunosuppressive therapy can be critical, emphasizing that diagnostic uncertainty should not delay treatment when autoimmune encephalitis is suspected.

Learning Points

- Maintain a broad differential for subacute neuropsychiatric decline in older adults, including autoimmune and paraneoplastic etiologies, even when initial imaging and CSF studies are unremarkable.

- Autoimmune encephalitis may present atypically and without classical antibody or imaging findings; clinical judgment and multidisciplinary input are essential to diagnosis.
- Positive but nonspecific serum antibodies (e.g., AChR α 3, MOG) should be interpreted cautiously within clinical context and not dismissed outright when the presentation suggests autoimmune pathology.
- Empiric immunotherapy can lead to significant improvement in suspected autoimmune encephalitis and should not be delayed when alternative causes have been reasonably excluded.

Ongoing follow-up is essential to assess treatment response, monitor for relapse, and reconsider the diagnosis if the clinical course deviates from expectations.

#22 From murmurs to melena: a case of the overlooked heart-gut connection

Matt Glasgow, MD, Evan Sweren, MD, and Danielle Babbel, MD

Department of Internal Medicine at the University of Utah

Case description:

An 85-year-old male with a history of IgA nephropathy status-post kidney transplant (in 1990) on chronic immunosuppression, tertiary hyperparathyroidism status-post partial parathyroidectomy, and heart failure with preserved ejection fraction (HFpEF) presented to the emergency department (ED) with two weeks of progressive shortness of breath, fatigue, and melena. The patient had not been taking any antiplatelets or anticoagulants.

While in the ED, vital signs were unremarkable. His labs showed a Hgb of 5.7 g/uL (baseline 15.0 about 3 months prior), platelet count 269 k/uL, creatinine 2.0 mg/dL (baseline around 1.20), BUN 67 mg/dL, and a positive fecal occult blood test. His exam was notable for a systolic murmur and pale appearance. The patient was admitted for a presumed upper gastrointestinal (GI) bleed. Upper endoscopy and colonoscopy demonstrated no obvious source of bleeding. The patient's symptoms abated with RBC and IV iron transfusions. He was discharged with GI follow-up. Over the next 5 months, the patient was hospitalized on six separate occasions for symptomatic anemia and melena. During one admission, he underwent push enteroscopy, which was unrevealing. Outpatient capsule endoscopy was attempted, but the capsule did not pass the pylorus and was nondiagnostic.

Ultimately, the patient presented again with melena and dizziness. He was found to have a Hgb 6.8 g/dL. He was transferred to our hospital for subspecialist coordination. Once admitted, TTE demonstrated severe AS with a peak transaortic valve gradient of 82 mmHg and a mean gradient of 50 mmHg. A von Willebrand factor (vWF) panel showed a vWF RCF/Ag ratio of 0.5 (normal: > 0.7), consistent with a diagnosis of acquired vWF syndrome (aVWS). Capsule endoscopy was repeated with endoscopic delivery of the capsule past the pylorus, which showed several small bowel angiodysplasias.

The patient was started on subcutaneous octreotide with stabilization in hemoglobin and resolution of melena over the course of his 14-day hospital admission. Interventional cardiology was consulted and performed a transcatheter aortic valve replacement (tAVR). The patient was discharged on subcutaneous octreotide with cardiology and GI follow-up. Discharge hemoglobin was 8.8 g/dL. One month after discharge, hemoglobin remained 8.8 g/dL and the patient had no recurrence of melena.

Discussion:

Heyde's syndrome is a rare and possibly underrecognized complication of severe AS in elderly patients that results from sheer force-mediated proteolysis of vWF multimers. This process precipitates aVWS, which promotes gastrointestinal angiodysplasia formation via lack of VEGF inhibition [1]. The combination of aVWS

and angiodysplasias results in persistent GI bleeding. This case represents a classic presentation of Heyde's syndrome and highlights challenges in diagnosis and treatment.

Diagnostic criteria for Heyde's syndrome are not well-established, but clinicians typically rely on the triad of severe AS, GI angiodysplasias, and aVWS. This patient's course was protracted with respect to diagnosis. Repeat video capsule endoscopy (VCE) and vWF testing confirmed the diagnosis. Small bowel angiodysplasias are notoriously difficult to visualize and VCE has emerged as the gold standard for diagnosis [2]. Additionally, vWF testing abnormalities are often transient and can be absent even in the presence of clinically significant vWF proteolysis. Interestingly, the vWF RCF/Ag ratio is inversely correlated with peak aortic valve gradient, suggesting that the severity of aVWS is directly correlated with the severity of AS [3]. This case emphasizes the importance of maintaining a high index of suspicion for Heyde's syndrome in patients with recurrent occult GI bleeding and severe AS even in the absence of vWF testing abnormalities and nondiagnostic endoscopy.

With respect to treatment, this patient experienced rapid resolution of GI bleeding and symptomatic anemia following tAVR. One meta-analysis found that patients with Heyde's syndrome who underwent AVR had a GI bleeding cessation rate of 73% and aVWS recovery rate of 86% [4]. Additionally, the patient was discharged on octreotide, which has been shown to resolve bleeding in up to 40% of patients with GI angiodysplasias [5]. Octreotide may play an important role in the medical management of Heyde's syndrome if tAVR is unable to be performed, but its utility is not well-established.

References:

1. Vincentelli A, Susen S, Le Tourneau T, Six I, Fabre O, Juthier F, Bauters A, Decoene C, Goudemand J, Prat A, Jude B. Acquired von Willebrand syndrome in aortic stenosis. *N Engl J Med.* 2003 Jul 24;349(4):343-9. doi: 10.1056/NEJMoa022831. PMID: 12878741.
2. Gerson, Lauren B MD, MSc, FACP¹; Fidler, Jeff L MD²; Cave, David R MD, PhD, FACP³; Leighton, Jonathan A MD, FACP⁴. ACG Clinical Guideline: Diagnosis and Management of Small Bowel Bleeding. *American Journal of Gastroenterology* 110(9):p 1265-1287, September 2015. | DOI: 10.1038/ajg.2015.246
3. Tamura T, Horiuchi H, Imai M, Tada T, Shiomi H, Kuroda M, Nishimura S, Takahashi Y, Yoshikawa Y, Tsujimura A, Amano M, Hayama Y, Imamura S, Onishi N, Tamaki Y, Enomoto S, Miyake M, Kondo H, Kaitani K, Izumi C, Kimura T, Nakagawa Y. Unexpectedly High Prevalence of Acquired von Willebrand Syndrome in Patients with Severe Aortic Stenosis as Evaluated with a Novel Large Multimer Index. *J Atheroscler Thromb.* 2015;22(11):1115-23. doi: 10.5551/jat.30809. Epub 2015 Aug 11. PMID: 26269004.
4. Goltstein LCMJ, Rooijackers MJP, Hoeks M, Li WWL, van Wely MH, Rodwell L, van Royen N, Drenth JPH, van Geenen EM. Effectiveness of aortic valve replacement in Heyde syndrome: a meta-analysis. *Eur Heart J.* 2023 Sep 1;44(33):3168-3177. doi: 10.1093/eurheartj/ehad340. PMID: 37555393; PMCID: PMC10471563.
5. Nardone G, Compare D, Scarpignato C, Rocco A. Long acting release-octreotide as "rescue" therapy to control angiodysplasia bleeding: A retrospective study of 98 cases. *Dig Liver Dis.* 2014 Aug;46(8):688-94. doi: 10.1016/j.dld.2014.04.011. Epub 2014 Jun 2. PMID: 24893688.

#23 A Devastating and Fatal Case of Hemophagocytic Lymphohistiocytosis and Immune Reconstitution Inflammatory Syndrome in the Context of Untreated HIV

CASE PRESENTATION

The patient is a 37 year-old male with a past medical history of untreated HIV, remote coccidiomycosis, and cognitive disability who presented to the ED with several months of nausea, vomiting, diarrhea, night sweats and altered mental status. Initial MRI of the brain and LP revealed no acute findings. Labs revealed elevated IL2r and ferritin, hypertriglyceridemia and bicytopenia. Patient continued to fever during hospitalization, and a presumptive diagnosis of hemophagocytic lymphohistiocytosis (HLH) was made, for which anakinra and dexamethasone were started. However, after transfer to the University of Utah hospital for HLH evaluation, these medications were discontinued due to uncertainty about the diagnosis.

Evidence was found of disseminated coccidiomycosis for which he was treated with dual antifungal therapy, but symptoms continued to worsen. He was also started on antiretroviral therapy (ART) for his previously untreated HIV. On the 11th day of his inpatient stay, he experienced clinical decompensation and encephalopathy prompting transfer to the ICU. Ferritin and IL2r increased dramatically, though came down after restarting dexamethasone and anakinra as suspicion re-grew for HLH contributing to the decompensation.

After improvement and transfer to the floor, he was discharged but returned six days later with altered mental status and hemodynamic instability. He did not respond to resuscitation, was transitioned to comfort care per family wishes, and passed away two hours later.

PHYSICAL EXAM ABNORMALITIES/LAB RESULTS

During his inpatient stay, the patient was found to have a high HIV viral load and very low CD4 count (26) and tested positive for CMV. Lymphadenopathy was noted, and a core needle biopsy confirmed additional coccidiomycosis. Lumbar puncture showed normal opening pressure and cell counts but was positive for cocci antibodies in the CSF. He also developed elevated transaminases, multiple electrolyte abnormalities, and severe lactic acidosis. Notably, he had persistently elevated ferritin (>4000), IL2r (>3500), hypertriglyceridemia, pancytopenia, and hepatosplenomegaly. Initial bone marrow biopsy lacked evidence of HIV infection, hemophagocytosis or evidence of malignancy. A repeat biopsy later revealed trace hemophagocytosis.

DIFFERENTIAL DIAGNOSIS

Initial diagnosis was HLH secondary to fungal and viral infection, supported by splenomegaly, elevated ferritin and IL2r, cytopenias, recurrent fevers and triglyceridemia. Throughout the hospital course, there was uncertainty about this diagnosis, due to initial lack of hemophagocytosis. The differential for his encephalopathy and decompensation shifted to favor uncontrolled HIV and associated opportunistic infections. However, no direct sources of CNS infection were found. After a repeat BMB showed hemophagocytosis, HLH was again presumed to be the final diagnosis, thought to be exacerbated by immune reconstitution inflammatory syndrome (IRIS) from initiation of ART therapy.

CONCLUSION

This case underscores the devastating consequences of untreated HIV and highlights the complex interplay between immunosuppression, opportunistic infection, and immune dysregulation.

The patient's profound immunodeficiency allowed for disseminated fungal and viral infections, which likely precipitated the hyperinflammatory cascade of HLH. Subsequent initiation of antiretroviral therapy may have further amplified this inflammatory response through immune reconstitution inflammatory syndrome (IRIS). Though the patient was treated as thoroughly as possible, these processes of immune dysregulation did not fully resolve and ultimately led to his death.

Earlier diagnosis and treatment of HIV—prior to the development of opportunistic infections and severe CD4 depletion—might have mitigated the risk of both HLH and IRIS. This case reinforces the critical importance of timely HIV detection and treatment as a global health priority.

#24 When Ibuprofen Burns: A Rare Case of Drug-Induced Linear IgA Bullous Dermatitis

Authors: Alan Zhang, MS3; Jeff Miller, MD

Case Presentation

A 42-year-old woman with no significant past medical history was transferred in for evaluation of a rapidly progressive blistering eruption. One week earlier, she developed vesicles in her nares that spread to her extremities and trunk, evolving into painful bullae. She was initially diagnosed with hand-foot-mouth disease at urgent care and treated with prednisone, without improvement. She subsequently developed a fever, diffuse skin pain involving nearly her entire body, and odynophagia, which prompted her ED visit. Notably, she reported taking several doses of ibuprofen daily since a week ago for a recent dental abscess.

Physical Examination

On arrival, she was febrile (38°C), tachycardic (128 bpm), and tachypneic (22/min). A thorough derm exam revealed widespread tense vesicles and bullae, notably many with annular configuration, involving the trunk, face, buttocks, and bilateral upper and lower extremities. Approximately 5–6% BSA was denuded, primarily across the back and

buttocks. Lesions were Nikolsky positive. No conjunctival, oral, or vaginal involvement was observed.

Laboratory & Imaging Findings

WBC $31 \times 10^3/\mu\text{L}$ (neutrophilia, eosinophilia); Hgb 9.0 g/dL; Creatinine 1.67 mg/dL; Lactate 2.8 mmol/L; Na 128 mmol/L.

Clinical Course

On admission, given the extent of skin denudation, she was managed with burn-unit level precautions, including sterile wound care, careful monitoring of fluid status, and vigilance for secondary infection. Skin biopsy was obtained and ibuprofen was listed as an allergy.

The dramatic extent of her blistering, coupled with the absence of mucositis, created a puzzling differential that included bullous pemphigoid, Stevens–Johnson syndrome, and

autoimmune blistering disorders. However, the annular configuration of her rashes strongly suggested linear IgA bullous dermatosis (LABD), an uncommon autoimmune blistering

disorder defined by linear IgA deposition at the dermal–epidermal junction. Over the following days her creatinine improved with resuscitation, but her hemoglobin continued to

decline (7.1 g/dL), necessitating a transfusion. On the third hospital day, her biopsy result confirmed LABD and pointing to ibuprofen as the most likely causative agent.

Treatment

The patient was initially treated with intravenous methylprednisolone, which provided partial control of disease activity. Once G6PD testing confirmed normal levels, dapsone was started for LABD specific treatment. She was later transitioned from IV to oral prednisone with a structured taper. Topical clobetasol was applied to the palms and soles. Denuded areas were covered with Vaseline and silver dressings. Her pain was aggressively managed with opioids and acetaminophen. Additional supportive measures included

compounded mouthwash for odynophagia, and iron supplementation with PRBC transfusion for symptomatic anemia. Close daily monitoring of hemoglobin and metabolic parameters guided therapy, particularly given her risk for dapsone-induced hemolysis. With this multidisciplinary approach, her systemic stability improved, pain decreased, and no new lesions emerged, signaling that her disease was under control.

Discussion

This case illustrates a rare case of ibuprofen-induced LABD, a rare autoimmune blistering disorder. Although vancomycin remains the prototypical culprit, NSAIDs such as naproxen has been reported as causative agents (Bouldin, 2011). However, there hasn't been any

reports of ibuprofen-associated LABD. Misdiagnosis is common early in the disease course due to its resemblance to viral exanthems or other bullous disorders. The patient's course emphasizes the importance of recognizing key dermatological patterns such as the annular patterned rash in this case, considering drug-induced etiologies in rapidly progressive

bullous eruptions, obtaining biopsy with DIF for definitive diagnosis, and providing burn-unit-style supportive care. Early dermatology input and transition to dapsone were key to her stabilization.

#25 A Difficult Case of Medicine Mimicry

Authors: Tess Hickey, MD; Taryn Young, MD

Learning objectives:

1. Formulate a differential diagnosis for a patient who presents with fever and polyarthralgia.
2. Recognize the clinical manifestations associated with calcium pyrophosphate crystal deposition (CPPD) disease.

Case:

An 80-year-old female with a history of pulmonary embolism, heart failure with preserved ejection fraction, calcium pyrophosphate deposition disease (CPPD), and C3-7 fusion presented to the hospital with fatigue, fevers, and arthralgias of the neck, back, and wrist limiting functioning.

Labs were remarkable for leukocytosis with white blood cells of 13, c-reactive protein of 22.6, erythrocyte sedimentation rate of 56, and Epstein-Barr Virus (EBV) VCA IgM Ag elevation. Creatine kinase was within normal limits. Computed tomography of the neck demonstrated prominent left-sided lymph nodes, cervical spondylosis, and foraminal narrowing. Physical exam was notable for cervical lymphadenopathy, pain on palpitation of deltoids, quadriceps, calves, and decreased strength in shoulders and hips bilaterally. Additional notable findings of right wrist edema and pain with active and passive range of motion. Work-up was notable for a mildly elevated rheumatoid factor with negative antinuclear antibody and anti-citrullinated peptide, normal thyroid stimulating hormone, negative human immunodeficiency virus, normal uric acid, and negative cytomegalovirus testing. EBV panel notable for elevated: EBV VCA IgM, VCA IgG, EBV Ab to nuclear Ag IgG, and early D Ag IgG. MRI C/T spine showed paraspinal enhancement with dorsal epidural/interspinous enhancement to C4 with associated posterior paraspinal edema to C7 with noted retro-dental and interspinous mineralization consistent with inflamed crystalline arthropathy. CT head dual energy with peri-odontoid and ventral epidural thickening and mineralization consistent with calcium pyrophosphate deposition though dorsal epidural soft tissue density without CPP deposition. Plastic surgery consulted given concern for right wrist septic arthritis and right wrist MRI concerning for flexor tenosynovitis with complex fluid collection. Rheumatology was consulted given findings consistent with CPPD and possible crowned dens syndrome. Rheumatology and plastic surgery felt findings most consistent with CPPD and crowned dens syndrome exacerbated by acute EBV infection given improvement on colchicine, down trending leukocytosis without antimicrobials, and clinical stability. She was started on Anakinra with significant improvement in symptoms and without need for joint aspiration or washout. The patient received four doses of anakinra and was discharged in improved condition with outpatient rheumatology follow-up.

Discussion:

Crowned dens syndrome is a rare manifestation of CPPD disease. It can be characterized by neck pain, shoulder stiffness, fever, elevated inflammatory markers, and CPPD around the atlanto-axial articulation and can mimic other diseases such as septic arthritis, meningitis, polymyalgia rheumatica, giant cell arteritis, and discitis. Crowned dens syndrome is misdiagnosed as meningitis up to 21.4% of the time.^[1] Crowned dens syndrome is an important diagnosis to keep on the differential since recognition could potentially prevent invasive diagnostic work-up of similarly presenting diagnoses. Furthermore, patients typically experience rapid improvement with appropriate therapy.

References:

1. Ledingham D, Cappelen-Smith C, Cordato D. Crowned dens syndrome. *Practical Neurology* 2018;18:57-59.

#26 A Bleeding Misconception: When Anchoring Bias Delays Two Critical Diagnoses in an Anemic Patient

Authors: Addie Netsanet, PGY1 University of Utah; Roshnee Raithatha, PGY2 University of Colorado; Julie Knoeckel, Associate Professor of Hospital Medicine Denver Health Medical Center

Introduction:

A 46-year-old woman with history of hemorrhoids and alcoholic cirrhosis presented to the ED after a ground-level fall and was admitted for acute-on-chronic anemia. She had 3 admissions in the previous 6 months for anemia requiring transfusions, which was attributed to hemorrhoidal bleeding and coagulopathy from cirrhosis.

Case Description:

Initial vitals were normal, and exam was notable for pallor. There was no blood on rectal exam. Labs showed leukocyte count of 24k/uL, hemoglobin 5.8g/dL, MCV 93, platelets 149k/uL, INR 1.35, total bilirubin 2.7mg/dL (direct 1.9), iron 60ug/dL, TIBC 352ug/dL, iron saturation 17%, reticulocyte count 2.91% (ULN 2.5%), ferritin 9.8ng/mL.

Her hemoglobin initially improved to 8.4g/dL with 2 units of pRBCs. However, 24 hours later her hemoglobin decreased to 6.5g/dL without any new evidence of bleeding. LDH was 481 U/L (ULN 220-250 U/L) and her haptoglobin, which was 16 mg/dL on admission, was undetectably low when repeated. Peripheral smear revealed anisocytosis and slight polychromasia. Direct antiglobulin test (DAT) was positive and the patient was started on high dose steroids for autoimmune hemolytic anemia

Subsequently, due to chronic, mild leukocytosis with a rise to 56.5 k/uL following steroid initiation, there was concern for leukemoid reaction versus hematologic malignancy. BCR-ABL1 fusion transcript was positively identified and bone marrow biopsy showed hypercellular marrow without increased blasts. She established with hematology for management of newly diagnosed chronic myelogenous leukemia.

Discussion:

This case illustrates the complexities of diagnosing recurrent anemia, particularly when multiple comorbidities obscure the underlying cause. In this patient, severe, refractory anemia resulted from warm autoimmune hemolytic anemia (wAIHA). While wAIHA is idiopathic in about 50% of cases, it can also be triggered by malignancies, infections, drugs, and autoimmune conditions. Although wAIHA is well-documented in association with chronic lymphocytic leukemia (CLL), its concurrence with chronic myelogenous leukemia (CML) is rare.

This case underscores the necessity of maintaining a broad differential for anemia, especially when clinical findings do not match the severity of the anemia. This patient had multiple hospitalizations for anemia that was misattributed to hemorrhoidal bleeding, but the severity of her anemia was

disproportionate her history of mild bleeding. Severe anemia that is disproportionate to the clinical presentation warrants a thorough workup, including evaluation for hemolysis and hematologic malignancies. Red flags, like worsening anemia despite transfusions, abnormalities in other cell lines, and an abnormal peripheral smear suggests that additional diagnoses should be considered.

#27 The Tell-Tale Eye: How a Subtle Palsy Unveiled a Sellar Giant

Authors: Alice Snelling, Morgan Williams MD, TJ Hartridge DO

Case presentation:

A 36-year-old male with history of migraines presented to the University of Utah ER with 1 day of sudden vision changes, 5 days of progressively worsening headache, and fatigue. He was at a festival two days ago, reports dehydration and taking various drugs. In the past, his migraines have been preceded by dehydration and decreased sleep. Regarding his double vision/blurry vision, patient reports difficulty focusing on objects, particularly when looking to the left. No photosensitivity. No difficulty walking, no weakness or numbness in his extremities, no other neurologic symptoms. Denies any other symptoms including chest pain, shortness of breath, vomiting, or diarrhea. Endorses minimal nausea. Does not take any medications other than Omeprazole.

Physical Exam

Afebrile, vital signs unremarkable. Of note, visual acuity was 20/16 bilaterally and neurological exam was significant for isolated left abducens palsy, no other focal neurological deficits.

Differential:

Differential diagnosis for progressively worsening headache, double vision, concurrent broad intoxication use, and left abducens palsy includes but is not limited to complex migraine, abscess/intracranial infection, idiopathic intracranial hypertension, neoplastic compression, cavernous sinus thrombosis, and traumatic injury.

Lab and Imaging results:

CBC showed mild anemia with a hemoglobin of 14, otherwise unremarkable. CMP was unremarkable. Given patient's abnormal neurologic exam CT imaging was obtained. CTA neck was unremarkable. CTA head showed pituitary lesion which involves the left cavernous sinus. Immediate follow up MRI was obtained for better characterization, that showed a heterogeneously enhancing sellar/suprasellar mass, most compatible with a macroadenoma with lateral displacement of the left cavernous carotid artery, likely explaining the left abducens nerve palsy.

Clinical course:

On day 1 patient was given a migraine cocktail while awaiting imaging results. Following CT/MRI results, patient was diagnosed with a pituitary macroadenoma and admitted to Neuro critical care under Neurosurgery. On day 2, he underwent a transsphenoidal approach for resection of a pituitary tumor (TSRPT). Due to elevated IGF-1 and remaining tumor tissue on imaging, patient underwent another TSRPT on day 3. Pt was discharged on day 7 with plans for close follow up.

Conclusion/discussion:

Abducens nerve palsy is an exceptionally rare clinical manifestation and presentation of a pituitary macroadenoma. Ocular nerve palsies themselves are uncommon, estimated to be present in 4.6% to 32% of pituitary macroadenoma cases [1]. Among these, the ocular motor nerve is most frequently affected due to its position within the cavernous sinus. In contrast, the abducens nerve is rarely involved in pituitary apoplexy due to the nerve's central location within the cavernous sinus. This case highlights the need for performing a thorough neurological exam, even in a busy setting such as the emergency room as pituitary apoplexies, including macroadenomas, can have very subtle physical exam findings.

Sources

1. Singh, A., Khurana, M., Pal, H., Azad, S., Sihag, R. K., & Kumar, B. (2022). Bilateral sixth cranial nerve palsy, the first presenting feature of hemorrhagic apoplexy of pituitary macroadenoma: A case report. *International journal of surgery case reports*, 98, 107522. <https://doi.org/10.1016/j.ijscr.2022.107522>



#28 The Price of Complement Blockade: A Case of Mucor-Driven Vascular and Pleural Catastrophe

Lily Kreber MD, Matthew Glasgow MD, Kyle Alexander MD, Dustin Anderson-Bell MD, Luis Vargas Buonfiglio MD

Case Description

A 45-year-old female with history of atypical hemolytic uremic syndrome (aHUS) treated with ravulizumab, stage 3b chronic kidney disease, chronic obstructive pulmonary disease, type I diabetes mellitus, and recent admissions for community acquired pneumonia and DKA presented to an outside emergency department (ED) complaining of left lower extremity pain and swelling. She had associated respiratory symptoms including dyspnea and progressive dry cough and was requiring 4 Lpm of oxygen from a baseline of 2 Lpm. She was found to have a large left deep vein thrombus (DVT) extending into the proximal iliac vein, resulting in transfer to our medical center for thrombectomy.

On arrival, exam was remarkable for 4+ left lower extremity edema that was tender to palpation and absent left sided breath sounds. Labs were significant for white blood cell count 15.0 k/uL, hemoglobin 7.2 g/dL, creatinine 1.92 mg/dL (baseline 2.0 mg/dL), BUN 45 mg/dL, albumin 2.1 g/dL, bicarbonate 18 mmol/L, fasting glucose 408 mg/dL, and Hgb A1c 16.3. CT angiogram of the chest demonstrated a large left-sided pleural effusion with near total collapse of the left lung and pulmonary artery (PA) pseudoaneurysm.

A left-sided chest tube was placed with frankly purulent output. Pleural fluid studies revealed a total nucleated cell count >100,000 per microliter (100% neutrophils), glucose 150 mg/dL, LDH >7,500 U/L, pH 6.53, adenose deaminase 182 U/L, and protein 3.6 g/dL. The patient was started empirically on vancomycin and piperacillin-tazobactam as well as intrapleural fibrinolytic therapy.

Treatment and Trajectory

Blood and pleural fluid cultures remained negative for aerobic, anaerobic, and acid-fast organisms through hospital day four, and therefore Infectious Disease was consulted. Sputum and serum testing for mucor was sent and empiric amphotericin B was initiated.

Unfortunately, she developed large volume hemoptysis necessitating intubation. Post-intubation bronchoscopy suggested fistulization between the pseudoaneurysm and bronchus so she subsequently underwent emergency embolization. Sputum mucor PCR returned positive and posaconazole and caspofungin were added to her regimen. The patient underwent a pneumonectomy of a completely consolidated left lung with extensive adhesions to the mediastinum and chest wall. Her airway was washed with amphotericin intraoperatively. She subsequently developed severe right ventricular failure, acute renal failure, and progressive neurologic deterioration with absence of brainstem reflexes. The patient's surrogate decision makers decided to transition to comfort care measures and the patient died shortly after.

Discussion

Ultimately, this immunocompromised patient with aHUS on a terminal complement inhibitor, asplenia, and type I diabetes had angioinvasive pulmonary mucormycosis with PA pseudoaneurysm formation following a subacute disease course with multiple hospitalizations for pneumonia and DKA. While the patient's uncontrolled diabetes and recent admission for diabetic ketoacidosis are both known risk factors for mucormycosis, the association between mucor infection and complement inhibition via razulizumab with asplenia is not well established. Ravulizumab functions as a terminal complement inhibitor by inhibiting cleavage of C5 into C5a (which contributes to neutrophil recruitment and chemotaxis) and C5b (which forms part of the membrane attack complex). In vitro studies and murine models suggest that the innate immune response to mucormycosis infection is dependent upon the alternative complement pathway which includes C5a and C5b as central mediators^{2,3}. Furthermore, the spleen contributes to phagocytosis of opsonized pathogens – another important aspect of the alternative complement pathway. Additionally, mucor infection in association with other hypocomplementemic states has been described in previous case reports⁴⁻⁶. It thus seems likely that our patient's complement inhibition and asplenic state left her particularly vulnerable to severe, invasive mucormycosis infection and such susceptibility should be considered for future patients and research.

References:

1. Multani A, Reveron-Thornton R, Garvert DW, Gomez CA, Montoya JG, Lui NS. Cut it out! Thoracic surgeon's approach to pulmonary mucormycosis and the role of surgical resection in survival. *Mycoses*. 2019 Oct;62(10):893-907. doi: 10.1111/myc.12954. Epub 2019 Aug 6. PMID: 31173415.
2. Granja, L. F. Z., Pinto, L., Almeida, C. A., Alviano, D. S., Da Silva, M. H., Ejzemberg, R., & Alviano, C. S. (2010). Spores of *Mucor ramosissimus*, *Mucor plumbeus* and *Mucor circinelloides* and their ability to activate human complement system in vitro. *Medical Mycology*, 48(2), 278-284.
3. Harpf, V., Rambach, G., Parth, N., Neurauter, M., Fleischer, V., Lackner, M., ... & Speth, C. (2023). Complement, but not platelets, plays a pivotal role in the outcome of mucormycosis In vivo. *Journal of Fungi*, 9(2), 162.
4. Sunseri, M., Walsh, B., Tsay, J., & Smith, R. (2019). Mucormycosis and candidemia following eculizumab treatment for atypical hemolytic uremic syndrome. *Chest*, 156(4), A1212.
5. Mok, C. C., Que, T. L., Tsui, E. Y. K., & Lam, W. Y. (2003, September). Mucormycosis in systemic lupus erythematosus. In *Seminars in arthritis and rheumatism* (Vol. 33, No. 2, pp. 115-124). WB Saunders.
6. Kusaba, G., Ohsawa, I., Ishii, M., Inoshita, H., Ohi, H., Horikoshi, S., ... & Tomino, Y. (2010). Evidence of immunopathological traces in mucormycosis: an autopsy case. *Clinical and experimental nephrology*, 14(4), 396-400.

#29 Abdominal pain, Fabry Disease, and a Rare Complication

Max Aycock, MD, Boomer Olsen, MD, Shalini Kasera, Adeline Browne, MD

Introduction

Fabry disease (FD) is an X-linked disorder due to a deficiency or lack of alpha-galactosidase A, leading to glycosphingolipid accumulation and damage to multiple organ systems. Evidence suggests that greater than two-thirds of females with FD experience symptoms related to the cardiovascular, neurological, renal, and gastrointestinal systems. Chronic abdominal pain occurs in 45-60% of females with FD.

Case Presentation

An 18-year-old female with a history of well-controlled type 1 diabetes and FD presented to the hospital with intractable abdominal pain, nausea, and vomiting. She had been treated with enzyme replacement therapy (i.e., Fabrazyme) since her diagnosis with FD at age 5. She was previously worked up for these symptoms with an esophagogastroduodenoscopy which showed patchy gastritis, and experienced mild symptomatic improvement after treatment with proton pump inhibitor and empiric pro-motility agents. When she returned to the hospital one week later with worsening of these symptoms, she was afebrile and had mild diffuse abdominal tenderness. Laboratory studies revealed a WBC of 19.93 K/uL, and ultrasound showed a small amount of free fluid in the pelvis, which was attributed to hemorrhagic cyst rupture. Laboratory and imaging studies, including a CTA abdomen, were unremarkable.

On hospital day three, she developed a fever, peritoneal signs, and lactic acidosis. Broad-spectrum antibiotics were started and repeat CTA abdomen was again negative for atherosclerotic disease but uncovered new pneumatosis. Surgical exploration revealed 80 cm of necrotic small bowel, which was resected, and purulent exudate in the peritoneal cavity. The patient subsequently developed intraabdominal abscess-like collections, which were drained by interventional radiology but did not grow bacteria.

Ultimately, histology of the resected bowel revealed ischemic injury and cystic spaces in the mesenteric vasculature, suggesting a pathological role of sphingolipid accumulation in the development of the ischemia and perforation. The patient was discharged twelve days postoperatively on a four-week course of ertapenem, tolerating an oral diet.

Discussion

Gastrointestinal manifestations of FD are typically characterized by nonspecific pain and diarrhea. Intestinal perforations have only been described in a small number of case reports, and to the best of our knowledge only in males not on enzyme replacement therapy. FD has an extremely heterogeneous presentation in females, but this case highlights how even rare complications may affect these patients. Mesenteric ischemia and/or intestinal perforation should be considered in any FD patient with unexplained abdominal pain and new lactic acidosis.

1. Germain, D. P. (2010). Fabry disease. *Orphanet Journal of Rare Diseases*, 5, 30. <https://doi.org/10.1186/1750-1172-5-30>
2. Hopkin, R. J., Laney, D., Kazemi, S., Ajroud-Driss, S., Bernstein, H. S., Cole, A. L., Kopp, J. B., Lippa, C. F., Ortiz, A., Parini, R., Ramaswami, U., Wilcox, W. R., & Germain, D. P. (2025). Fabry disease in females: Organ involvement and clinical outcomes compared with the general population. *Orphanet Journal of Rare Diseases*, 20, 433. <https://doi.org/10.1186/s13023-025-03922-x>
3. Germain DP, Fouilhoux A, Decramer S, et al. Consensus recommendations for diagnosis, management and treatment of Fabry disease in paediatric patients. *Clin Genet*. 2019;96(2):107-117. doi:10.1111/cge.13546
4. Zar-Kessler C, Karaa A, Sims KB, Clarke V, Kuo B. Understanding the gastrointestinal manifestations of Fabry disease: promoting prompt diagnosis. *Therap Adv Gastroenterol*. 2016;9(4):626-634. doi:10.1177/1756283X16642936
5. Jardine DL, Fitzpatrick MA, Troughton WD, Tie AB. Small bowel ischaemia in Fabry's disease. *J Gastroenterol Hepatol*. 1994;9(2):201-204. doi:10.1111/j.1440-1746.1994.tb01244.x
6. Bryan, A., Knauft, R. F., & Burns, W. A. (1977). Small bowel perforation in Fabry's disease. *Annals of Internal Medicine*, 86(3), 315–316. <https://doi.org/10.7326/0003-4819-86-3-315>

#30 Beyond Viral Reactivation: EBV-Negative Methotrexate-Associated DLBCL in an Elderly Patient with Rheumatoid Arthritis

Authors: Ayush V. Peddireddi, Emma K. Hughes, Madeline Foley

Case Description

An 84-year-old male with a history of newly diagnosed atrial fibrillation, heart failure with recovered ejection fraction (LVEF 70%), and rheumatoid arthritis on chronic methotrexate (MTX) presented to an outside emergency department with generalized weakness, worsening dyspnea, and dark, hard stools. Over the preceding year, he had experienced progressive functional decline with frequent falls and, during the prior five weeks, accelerated deterioration accompanied by 11-pound weight loss, early satiety, abdominal cramping, and urinary hesitancy. A CT of the abdomen and pelvis revealed a 6.5 cm pelvic mass with extensive mesenteric lymphadenopathy and segments of thickened small bowel. He was transferred for further evaluation, and CT-guided biopsy of a mesenteric lymph node demonstrated diffuse large B-cell lymphoma (DLBCL), activated B-cell (ABC) subtype with co-expression of BCL2 and MYC (double expressor phenotype).

A staging PET-CT confirmed stage IV disease with FDG-avid lymphadenopathy both above and below the diaphragm and extranodal involvement of bowel, mesenteric soft tissue, and pelvic structures, consistent with advanced disseminated disease. Methotrexate was discontinued upon admission; however, due to the aggressive appearance and extent of disease, the team elected to pursue systemic therapy rather than observation. After a goals-of-care discussion, the patient opted for treatment with curative intent. Given his history of heart failure, an anthracycline-containing regimen was deferred. He received a short course of pre-phase corticosteroids followed by a dose-reduced regimen of rituximab, cyclophosphamide, vincristine, and prednisone (R-CVP).

After undergoing cycle 1 of R-CVP inpatient with excellent tolerance, he was discharged to a subacute rehabilitation facility and tolerated R-CVP cycle 2 well. While mere monitoring was considered at this point, the decision was made to continue chemotherapy for a total of six cycles due to the patient's frailty and absence of EBV on biopsy. The patient was then discharged home with plans for repeat PET-CT staging prior to cycle 3.

Discussion

This case illustrates a rare presentation of methotrexate-associated lymphoproliferative disorder (MTX-LPD) manifesting as EBV-negative diffuse large B-cell lymphoma (DLBCL) with ABC subtype and double expressor phenotype in an elderly patient. MTX-LPD typically arises after several years of chronic immunosuppression, most often in patients with rheumatoid arthritis on long-term methotrexate therapy. The pathogenesis is usually linked to EBV reactivation secondary to impaired T-cell immune surveillance, leading to proliferation of EBV-infected B cells. Consequently, most MTX-LPDs are EBV-positive, and discontinuation of methotrexate

alone leads to spontaneous regression in up to 80% of cases, typically within several months¹. In contrast, EBV-negative MTX-LPD represents a less common and biologically distinct subset that behaves more like de novo DLBCL, showing limited potential for regression after methotrexate withdrawal. This distinction was particularly relevant in our patient, whose biopsy demonstrated EBV negativity, ABC molecular subtype, and BCL2/MYC co-expression, all features associated with more aggressive clinical behavior and poorer prognosis.

Given the patient's extensive extranodal disease involving bowel and mesenteric structures and his rapid clinical decline, the decision was made to initiate systemic therapy rather than observation, even after stopping methotrexate. Although R-CHOP remains the standard curative regimen for DLBCL, the patient's history of heart failure precluded anthracycline use. Therefore, a dose-reduced R-CVP regimen was selected to balance efficacy and tolerability in the context of frailty and cardiac comorbidity. This regimen is typically palliative in de novo DLBCL, but has demonstrated activity in MTX-LPD and anthracycline-ineligible patients. The patient's excellent tolerance of early cycles and stable clinical trajectory supported continuation for six total cycles. This case underscores the importance of recognizing MTX-LPD as a diagnostic possibility in patients on chronic immunosuppression who present with new lymphadenopathy or systemic symptoms, while emphasizing that EBV-negative cases may require full systemic chemotherapy akin to de novo DLBCL. It also highlights the clinical nuance required when balancing curative intent, treatment-related toxicity, and patient frailty in elderly individuals with aggressive lymphomas

Works Cited

1. Kurita D, Miyoshi H, Ichikawa A, et al. Methotrexate-associated Lymphoproliferative Disorders in Patients With Rheumatoid Arthritis: Clinicopathologic Features and Prognostic Factors. *Am J Surg Pathol*. 2019;43(7):869-884. doi:10.1097/PAS.0000000000001271

#31 Unmasking PTLD: The Challenges of Navigating Overlapping Pathology

Authors: Ayush Peddireddi, Matthew Glasgow, Evan Gross, Lily Kreber, Andrew Courtwright

Case Presentation

A 61-year-old male with advanced pulmonary silicosis underwent an uncomplicated bilateral lung transplant for which he was cytomegalovirus (CMV) donor and recipient negative (D-/R-) and Epstein Barr virus (EBV) donor positive, recipient negative (D+/R-). Five months post-transplant, he was hospitalized for dyspnea and bilateral pleural effusions.

Pleural fluid studies revealed a lymphocytic exudate. Flow cytometry and cytology were negative for malignant cells, but EBV DNA was detected in both pleural fluid and serum by PCR (log 2.88). Transbronchial biopsy suggested acute cellular rejection (ACR); however, treatment was deferred pending further diagnostic evaluation for post-transplant lymphoproliferative disease (PTLD).

PET imaging demonstrated multiple fluorodeoxyglucose (FDG)-avid lesions in subclavian lymph nodes, liver, and spleen. Core needle biopsy of a subclavian lymph node showed granulomatous changes consistent with the patient's known silicosis. Based on radiographic appearance, the hepatic and splenic lesions were also felt to be related to silica exposure. The patient received high-dose steroids for ACR and a single dose of rituximab for persistent EBV viremia (log 3.25).

One month later, he was readmitted with fatigue, abdominal bloating, and orthostatic hypotension. A CT of the abdomen and pelvis revealed interval enlargement of his hepatic and splenic lesions. Biopsy of a 1.8 cm liver lesion was **nondiagnostic**, showing only necrotic tissue with unremarkable flow cytometry. EBV viral load increased to log 4.31. Bone marrow biopsy was negative for malignancy. Given ongoing diagnostic ambiguity, the patient underwent repeat PET imaging which demonstrated new hypermetabolic lesions in multiple lymph nodes and enlarging hepatic, osseous, and pulmonary lesions.

A second liver biopsy of a new PET-avid lesion showed **atypical lymphoid proliferation with extensive necrosis**, staining positive for **CD19, CD79a, CD30, MUM1, and BCL2**, but **negative for CD20**. Retrospective hematopathology review of the initial liver biopsy identified variable CD20 staining within the necrosis. These findings established the diagnosis of EBV-positive, stage IV diffuse large B-cell lymphoma (DLBCL) secondary to PTLD, consistent with the monomorphic PTLD subtype per World Health Organization (WHO) classification.

The patient received four additional doses of rituximab, with EBV PCR decreasing to log 2.65. Several weeks later, new onset diarrhea prompted colonoscopy, which revealed erythematous, ulcerated mucosal; intraluminal biopsy demonstrated polymorphic PTLD. With concern for disease progression, the patient was transferred to a tertiary cancer center for re-staging and additional inpatient chemotherapy.

Discussion

This case underscores the importance of maintaining vigilance for **PTLD** in an **EBV D+/R- transplant recipient**, particularly when post-transplant complications overlap. Although the initial biopsies were

negative, the patient's donor/recipient mismatch and ongoing immunosuppression kept PTLD a key consideration, as both are strong risk factors for **EBV reactivation and lymphoproliferation**. The coexistence of **ACR** added diagnostic and therapeutic complexity; because **pulse-dose corticosteroids** are **T-cell-lytic** and can worsen **EBV replication** and **PTLD**, full ACR treatment was appropriately deferred until the diagnosis was clarified.

Further complicating the diagnosis was the patient's prior silica exposure, which resulted in FDG-avid lymph nodes in the absence of supportive PTLD testing. This overlap necessitated a broad infectious and neoplastic workup, including consideration of **de novo lymphoma**. Ultimately, diagnosis was confirmed only after repeat biopsy targeted a new lesion.

Finally, a notable feature was the **loss of CD20 expression** following **rituximab administration**. While the extent of the positive CD20 staining in the confirmatory biopsy was limited to one weakly positive lymphoid cell, re-examination of the initial biopsy demonstrated variable positivity.

In summary, while PTLD is a well-recognized complication of solid organ transplantation, this case exemplifies the diagnostic ambiguity that can arise in complex transplant recipients. The coexistence of rejection, granulomatous disease, and post-rituximab changes created multiple diagnostic challenges, underscoring the need to keep a high degree of suspicion when initial findings are inconclusive.

#32 Metastatic Colorectal Cancer Presenting as Intractable Radicular Pain in a Young Patient Without Age-Appropriate Screening: A Clinical and Psychosocial Perspective

Authors: Emma K. Hughes, Nicole Kummet, Madeline Foley

Case Presentation

A 49-year-old woman with fibromyalgia, chronic fatigue syndrome, and recent left L5-S1 laminoforaminotomy for radiculopathy presented to an outside emergency department with severe right-sided back pain. The pain was similar to prior radiculopathy but substantially more intense and refractory to her home medications. CT lumbar spine revealed abnormalities prompting further imaging, and CT chest/abdomen/pelvis demonstrated diffuse metastatic disease involving the chest, liver, pelvis, and lymph nodes. She was transferred for workup of metastatic cancer. MRI thoracolumbar spine showed T11-L1 osseous lesions without spinal cord compression. CT-guided biopsy of a hepatic lesion confirmed metastatic colorectal adenocarcinoma.

Her hospital course was complicated by bilateral pulmonary emboli in the setting of active GI bleed secondary to metastatic disease, requiring anticoagulation with worsening bleed.

Additionally during her hospitalization she developed multiple infections including pyelonephritis, pneumonia, and acute cholangitis secondary to metastatic liver lesions resulting in acute hepatic failure. Palliative radiation was initiated for gastrointestinal symptoms, with quick resolution of GI bleed. Radiation was terminated early with plans to start systemic chemotherapy; however, her rapid decline precluded initiation of treatment.

Pain Management

At presentation, the patient was treated with oxycodone, intravenous hydromorphone, ketorolac, pregabalin, and diazepam. She reported greatest benefit from ketorolac, suggesting a strong inflammatory component to her pain. Given the limitations of prolonged NSAID use, palliative care was consulted for comprehensive management.

Her final multimodal regimen included:

Methadone 2.5 mg Q12H

Hydromorphone 4 mg Q4H PRN

Tylenol 1000 mg TID

Meloxicam 15 mg daily

Nortriptyline 25 mg QHS

Pregabalin 150 mg BID

This approach targeted neuropathic, inflammatory, and nociceptive pain pathways while minimizing somnolence in accordance with the patient's goals. Low dose methadone provided opioid receptor activation and NMDA antagonism pregabalin and nortriptyline addressed neuropathic components via calcium and sodium channel modulation. NSAIDs were retained for osteolytic and inflammatory pain. Pain control improved substantially, allowing the patient to interact meaningfully with her family.

Clinical Course and Psychosocial Dynamics

Despite optimized pain management, her condition deteriorated rapidly due to recurrent infections, hepatic failure, and respiratory decline. Initially, she expressed a desire for aggressive treatment; however, through ongoing palliative discussions emphasizing prognosis and best/worst case scenario building, she and her family elected for home hospice. She was discharged with comfort measures and passed away two days later surrounded by loved ones.

The psychological shift from recovery after spinal surgery to terminal cancer within weeks was profound for this patient. The patient and family experienced distress, disbelief, and anticipatory grief. Early palliative involvement was critical in facilitating communication, aligning care with her evolving values, and supporting emotional adaptation during this transition.

Discussion

This case underscores several important considerations. First, metastatic cancer should remain within the differential for intractable pain, even in younger patients or those with plausible alternative explanations such as recurrent surgery or chronic pain syndromes. Overlapping symptoms from fibromyalgia and postoperative radiculopathy likely masked early manifestations of malignancy. The absence of age-appropriate colorectal cancer screening further delayed diagnosis until widespread metastasis.

Second, the patient's pain was multifactorial, arising from osseous lesions, nerve compression, and visceral involvement. Combining agents with complementary mechanisms of action in a multimodal approach optimized analgesia while minimizing opioid burden and sedation, aligning with palliative goals of preserving awareness and dignity.

Finally, this case highlights the emotional and existential impact of an abrupt transition from health to terminal illness in less than 30 days. Integrating palliative care early allowed for effective symptom control, psychosocial support, and shared decision-making consistent with the patient's values.



#33 Uncovering the Truth: A Full Body Rash

Author: Kelsey Wartgow, MD PGY-3 University of Utah Internal Medicine Resident

Case presentation:

This is a 44-year-old male with history of obesity, type 2 DM, hypertension, anxiety, depression, ADHD, OSA on CPAP who presented with a progressively painful full body rash. Additionally, the patient had an unclear but significant history of rashes in the previous 5 years and was followed by outside dermatologist but did not have a specific known diagnosis or treatment plan at the time of presentation. Several weeks prior to presentation, the patient started to experience reddening of his skin that spread diffusely head to toe with involvement of palms and soles. His skin began sloughing and he developed low grade fever, chills and malaise in the few days prior to presentation. He initially sought care at an outside hospital due to worsening skin pain and was prescribed a course of prednisone and ciprofloxacin. He re-presented due to lack of improvement in symptoms and was transferred to our hospital for higher level of care. Review of systems was otherwise unremarkable. He was taking multiple prescription medications including lisinopril and hydrochlorothiazide, but none were started close to the onset of this rash. He was not using any over the counter medications. He denied recent travel, sick contacts, sexual contacts, and substance use other than smoking 1 pack of cigarettes per day and use of nicotine pouches.

Physical exam:

Vitals signs were within normal limits except HR 101. There was bilateral conjunctival injection with clear discharge from eyes. There was full body erythema with shiny and taut skin consistent with erythroderma. Areas of sloughing were present without any notable blistering of the skin. There was no mucosal involvement. Bilateral lower extremities and scrotum had significant non-pitting edema.

Labs:

WBC 8k, Hb 11.3, Plt 460k. CMP within normal limits except elevated glucose. A1c 8.5%. CRP 21, ESR 102. HIV, Hep B/ C, RPR, chlamydia/ gonorrhea were all negative.

Clinical course:

Dermatology was consulted and performed skin biopsy. The highest suspicion for this case of erythroderma was a drug reaction (ACE-I or thiazide) vs atopic dermatitis after dermatopathology showed a nonspecific spongiotic dermatitis. There was no evidence of lymphocyte atypia. Oral cyclosporine was initiated and later transitioned to high dose prednisone (60 mg/day) along with BID topical steroids dressings. However, erythroderma was refractory to these interventions. On hospital day 7, he was noted to have right inguinal mass. Ultrasound demonstrated extensive inguinal lymphadenopathy. Subsequent CT abdomen/ pelvis and then PET scan revealed diffuse hypermetabolic lymphadenopathy. Upon concern for lymphoma, systemic steroids were discontinued to improve yield for biopsy. Surgical biopsy of a lymph node revealed ALK- negative anaplastic large cell lymphoma (ALCL). Multiple repeat biopsies had no evidence of direct infiltration of lymphoma. Ultimately, erythroderma was felt to be paraneoplastic and that improvement of the skin condition may take weeks to improve after starting appropriate treatment

of underlying lymphoma. He was transferred to hematology service where he began CHOEP chemotherapy. Currently, he is nearing cycle 2 of chemotherapy with now moderate improvement in erythroderma. He remains on topical steroids in addition to treating the underlying lymphoma.

Discussion:

Erythroderma is a severe and potentially life-threatening condition that presents as diffuse skin erythema and scaling involving $\geq 90\%$ body surface area. It should be thought of as a clinical sign rather than one specific disease entity, as it is the result of a wide range of cutaneous and systemic diseases including psoriasis, atopic and contact dermatitis, pityriasis rubra pilaris, drug reaction, GVHD, cutaneous T-cell lymphoma (including Sezary syndrome and mycosis fungoides) and paraneoplastic. In this case, erythroderma was distinguished as paraneoplastic from cutaneous T-cell lymphoma based on lack of direct lymphoma skin involvement. Like many other conditions, treatment of erythroderma consists of identifying and treating any underlying cause. Additional treatment aims at direct application of topical steroids and supportive care including aggressive pain control and emollient application. Severe complications of erythroderma include electrolyte derangements, temperature dysregulation, fluid losses and heart failure.

#34 Therapeutic Resection of Functional Adrenocortical Adenoma for Subclinical Cushing Syndrome

Krey Ramsey, OMS-II; Kalin Sorenson, OMS-III; Gursharan Lubana, OMS-II; Tyler Haberle, MD, Professor of Clinical Medicine; Rocky Vista University College of Osteopathic Medicine, Ivins, UT

Subclinical Cushing syndrome is characterized by mild autonomous cortisol secretion at levels that are clinically silent. However, even subtle cortisol elevations have been linked to metabolic and systemic symptoms, and no standardized management approach has been established.¹

A 61-year-old woman presented to an endocrinologist for evaluation of an incidentally discovered left adrenal mass found two months earlier during imaging for post-influenza hepatomegaly. Abdominal MRI showed a 3.0 cm heterogeneous lesion with microscopic fat, raising suspicion for a myelolipoma or collision tumor. She reported feeling unwell for over nine months, with progressive symptoms including morning anxiety improving by evening, intermittent hot flashes, depressed mood, palpitations, chest discomfort, tremors, decreased appetite, and unintentional 17-pound weight loss. Hormone replacement therapy for presumed menopausal symptoms had worsened her condition and was discontinued. Her history included hypertension, major depressive disorder, and benign ovarian tumor resection in 1987. Medications included triamterene– hydrochlorothiazide. She denied tobacco, alcohol, or illicit drug use and had recently stopped several dietary supplements. She appeared well and in no distress, with only a fine procedural tremor noted. Facial plethora, abdominal striae, dorsocervical fat pad, central adiposity, and muscle weakness were not noted on physical exam. Morning serum cortisol was slightly elevated (17 µg/dL). A 24-hour urinary cortisol was normal (19 µg/day), but the cortisol-to-creatinine ratio was elevated (40). A 1 mg dexamethasone suppression test showed elevated cortisol (5.3 µg/dL) with suppressed ACTH (<1.5 pg/mL). Repeat CT after three months showed mild interval growth to 3.2 cm and attenuation of 21 HU. Findings were consistent with a functional adrenocortical adenoma causing subclinical Cushing syndrome. The patient was started on ketoconazole while awaiting surgical evaluation and subsequently underwent laparoscopic left adrenalectomy without complications. Pathology revealed a 17 g, 3.0 ×

2.3 × 2.0 cm unencapsulated golden-yellow cortical nodule with central hemorrhage and no necrosis. Histology showed a low-grade adrenocortical adenoma with Ki-67 index of 3% and focal nuclear atypia; no Weiss criteria for malignancy were met. She was started on physiologic hydrocortisone replacement (20 mg AM, 10 mg PM) with a gradual taper. At postoperative follow-up, she reported marked improvement in mood, energy, and appetite. Her blood pressure stabilized at 140/80 mmHg, cortisol levels normalized, and morning anxiety, palpitations, and weight changes resolved.

This case illustrates how even mild, clinically silent cortisol excess can manifest with meaningful systemic symptoms and reversibility after adrenalectomy. It emphasizes the importance of maintaining thorough and individualized approach in patients with incidentally discovered adrenal lesions.

Reference

Rowe NE, Kumar R, Schieda N, Siddiqi F, McGregor T, McAlpine K, Violette P, Bathini V, Eng M, Izzard J. *Diagnosis, Management, and Follow-Up of the Incidentally Discovered Adrenal Mass: CUA Guideline Endorsed by the AUA*. J Urol. 2023 Oct;210(4):590–599. doi:10.1097/JU.0000000000003644. Epub 2023 Aug 9. PMID: 37556768.

#35 Acute pancreatitis as primary driver of shock in the setting of LAD STEMI

Austin Meyer MD, Department of Internal Medicine, University of Utah, Salt Lake City, UT.

Case Presentation:

67-year-old male without significant cardiac history, hypertension and active tobacco use called EMS during a road trip for severe epigastric abdominal pain which woke him from sleep and denied any chest pain at the time. EMS ECG with anterior ST elevations and was transferred to a tertiary care center and activation of the cath lab where he was loaded with aspirin and ticagrelor. He was found to have heavy thrombus burden in proximal LAD and required thrombectomy with stenting and transferred to CVICU in stable condition. HS-Troponin > 60k. His post-cath ECG was notable for anterolateral Q-waves but resolution of ST-elevations. Echo with decreased EF 35-40% and no LV thrombus. He continued to have severe abdominal pain but was otherwise alert, oriented with stable hemodynamics.

Overnight on hospital day two, physical exam notable for: alert only to self and pulling at lines becoming increasingly agitated. Blood pressures remained stable with tachycardia into 110s. Cool extremities. Differential diagnosis for acute onset encephalopathy included cardiogenic shock, septic shock, alcohol withdrawal and hospital acquired delirium. He was subsequently started on BAWs/CIWA protocol without improvement in clinical condition.

By mid-morning his exam was notable for exquisitely, diffusely tender and distended tympanitic abdomen but soft without guarding or rebound. Bilateral lower extremities were mottled and cool with worsening encephalopathy. CT abdominal imaging was obtained and notable for acute edematous pancreatitis and had concordant lipase of 2100. IV fluids with stepwise boluses of 500 cc were initiated cautiously given for fear of precipitating cardiogenic shock with new heart failure and moderately elevated LV end-diastolic pressure in his cath.

Unfortunately, by 1200, he decompensated rapidly with anuric renal failure and hypotension and promptly started on dobutamine. His abdominal exam remained significantly distended, tender but soft. He also became febrile to 39.3 and started on vancomycin and piperacillin/tazobactam given concern for septic shock from intraabdominal source. Over about 1 hour, he rapidly escalated to five vasopressor shock and was intubated for airway protection and hypoxemia. In the setting of profound abdominal distension and multi-organ failure, during intubation an indirect bladder pressure was obtained while paralyzed which yielded a pressure of 28 mmHg consistent with Grade IV primary abdominal compartment syndrome. General surgery was consulted and emergently performed bedside decompressive laparotomy with rapid improvement in urine output and hemodynamics. Severe necrotic pancreatitis, acalculous cholecystitis and small bowel ischemia were evident on visual inspection. Percutaneous cholecystostomy was performed and subsequently taken to the OR for partial small bowel resection. He returned to the SICU in critical but stable condition, ultimately extubated and weaned off vasopressors by hospital day 12. Unfortunately, his hospital course was complicated by persistent intrabdominal infection, complete heart block, and recurrent arterial thrombi of bilateral lower extremities. The patient ultimately expired on hospital day twenty-seven after a transition to comfort measures.

Discussion:

Given the lack of meaningful clinical status change following successful coronary reperfusion from an LAD STEMI, keeping a broad differential to include possibilities of septic and distributive shock were essential in identifying this patient's primary driver of critical illness. Given the hyperacute SIRS physiology secondary to acute pancreatitis, his intra-abdominal pressures were profoundly elevated requiring urgent surgical decompression which explains the lack of response to inotropy or vasopressor support.

Additionally, although intra-abdominal pressures ultimately confirmed the diagnosis, clinical recognition of a distended abdomen, cool extremities, skin mottling and multiorgan failure were needed to suspect ACS as the primary etiology of his shock physiology rather than any specific objective lab values or hemodynamic measurements. His lactate peaked at only 3.6 throughout this initial course.

Furthermore, provided his primary symptom was abdominal pain, our suspicion the SIRS from pancreatitis may have been the inciting event to a thrombotic LAD occlusion was raised but impossible to know for sure. Either way, early closure and focus on his cardiac pathophysiology likely lead to a slower recognition and treatment of his acute pancreatitis in the absence of a STEMI diagnosis.

Sources:

1. Popowicz P, Newman RK, Dominique E. Abdominal Compartment Syndrome. [Updated 2025 Sep 15]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK430932/>
2. Zarnescu NO, Dumitrascu I, Zarnescu EC, Costea R. Abdominal Compartment Syndrome in Acute Pancreatitis: A Narrative Review. *Diagnostics* (Basel). 2022 Dec 20;13(1):1. doi: 10.3390/diagnostics13010001. PMID: 36611293; PMCID: PMC9818265.

#36 Blue Toe Syndrome: Manifestation of an often forgotten deadly illness

Case Presentation:

76 yo M w/ PMHx of CKD 5, BPH, presented to ED after a ground level fall at home.

On evaluation, patient was afebrile with heart rate 100-110, severe hypertension with systolic blood pressure 190-210. Laboratory evaluation notable for acute renal failure and acute on chronic anemia with; chemistry notable for bicarbonate of 10, , creatinine 9.37, blood urea nitrogen of 104; CBC notable for hemoglobin 6.5 (baseline 8.0 – 8.5) with MCV of ~102, platelet of ~75 (baseline ~150-175). Pt was transferred to medicine for further workup of rapid progression of renal disease and initiation of dialysis.

Recent medical history was most notable for subacute rapid progression of renal disease over the three months prior to presentation, with outpatient renal biopsy notable for nonspecific findings including focal global glomerulosclerosis, moderate to severe interstitial fibrosis/tubular atrophy, and moderate severe arterial sclerosis; all of which were attributed to hypertension and liberal use of NSAIDs. He was scheduled to initiate peritoneal dialysis in the coming weeks.

Clinical Course:

Evaluation on admission notable for mild altered mental status, 2+ lower extremity edema, livedo reticularis of the bilateral lower extremities, and blue dusky discoloration of the distal plantar podagra bilaterally. Bilateral lower pedal pulses 2+ bilaterally.

Subjectively, he denied any recent bleeding episodes, vertigo, fevers, chills, cough/congestion, chest pain, or shortness of breath. Denied any subjective confusion, neurologic symptoms, or new or changing paresthesias. Patient was alert, oriented, and in no acute distress on evaluation. He reported that the noted lower extremity skin findings have appeared and resolved intermittently over the course of the last few months, and improve with warming. A warming blanket was placed and serial examination did indeed note improvement in livedo reticularis, and mild improvement in the distal dusky discoloration of the podagra.

Our leading hypothesis at time of presentation favored that subacute progression of renal disease & subsequent uremia drove rendered the patient susceptible to mechanical fall, with hypertension exacerbated by volume overload in the setting of renal disease. Laboratory abnormalities of the comprehensive metabolic panel and complete blood count were favored to be explained by acute on chronic renal failure.

On HOS-D1, pt began to exhibit mild altered mentation with decreased attention in conversation, increased confusion, favored to be due to uremic encephalopathy.

Nephrology was consulted for evaluation of progression of renal disease and initiation of dialysis. With recent renal biopsy results favoring hypertension and NSAID use as the most likely precipitating etiologies, repeat renal biopsy was deferred. Dermatology was consulted for evaluation of livedo reticularis with lower extremity distal cyanotic changes. A broad workup for autoimmune, cryoglobulinemic, and vasculitic etiologies was obtained. Dermatologic biopsy was also deferred. Further laboratory evaluation resulted and was concerning for ongoing hemolysis (total bilirubin 1.7 / direct 0.8, LDH ~350, haptoglobin undetectable).

On HOS-D2, Hematology was consulted for evaluation, and further workup was sent to assess for occult hematologic malignancies (SPEP/UPEP, flow cytometry), occult infection (respiratory viral panel, HBV/HCV, HIV), screening for DIC (fibrinogen, coags), further hemolysis workup (DAT). Peripheral smear was obtained and notable for 2-3+ schistocytes. The broad serologic workup as detailed above resulted largely negative; most notably with negative vasculitis serologies, negative cryoglobulins, and no evidence of active or occult infection. Despite initiation of dialysis on the day prior, the patient's mentation continued to decline, favored at that time to be hospital acquired delirium with delayed neurologic recovery from uremic encephalopathy.

Neurologic function continued to decline on HOS-D3; on AM examination pt was hypophonic and minimally reactive to stimuli, however without clear focal deficits. A brain attack was called; CT head and CTA H&N were without abnormalities.

Neurology was consulted and recommended MRI brain for further evaluation and workup. Notably, the patient's lower extremity dermatitis continued to improve with warming. Laboratory evidence of hemolysis improved on serial evaluation, with improvement in hypertension following HD sessions; the etiology of hemolysis was favored to be due to urgent hypertension in the setting of renal and microvascular disease. Goals of care discussions were held with family given the patient's rapid progression of disease without identifiable modifiable targets for treatment; following this discussion the patient's code status was transitioned to DNR/DNI. The patient's neurologic function continued to rapidly decline throughout the day, becoming unresponsive and obtunded with no response to verbal or noxious stimuli. The patient ultimately developed neurogenic respiratory failure, and in consultation with family at bedside, was transitioned to comfort measures in keeping with the patient's and family's wishes.

Autopsy was obtained to further investigate the patient's unexplained rapid progression of disease, and was notable for a four centimeter abdominal aortic aneurysm with extensive ulcerated atheromatous plaques throughout the large vessel vasculature. Extensive atheroemboli with cholesterol clefts were identified throughout the kidneys. Neuropathology resulted with severe arteriosclerosis and white matter rarefaction, most consistent with severe small vessel vascular disease affecting the CNS. Ultimately in consideration of the above autopsy results with clinical

Presentation: the unifying diagnosis most fitting with the patient's presentation and cause of death would be Spontaneous Cholesterol Embolization Syndrome due to the identified ulcerated large vessel atheromatous plaques.

Discussion:

Cholesterol Embolization Syndrome (CES) is a multisystem disorder caused by the embolization of cholesterol crystals from atherosclerotic plaques, most commonly following vascular interventions; in rare circumstances, it can occur spontaneously, termed the spontaneous Cholesterol Embolization Syndrome (sCES) [Ozcok 2019].

The typical constellation of findings involves progressive renal injury, lower extremity livedo reticularis, and often involves 'blue toe syndrome', an indicator of microvascular ischemia [Hirschmann 2009]. This presentation may overlap significantly with pathologies such as small vessel vasculitis, cryoglobulinemia, or various infections/malignancies (particularly viral and lymphomatous) which may frequently induce livedo reticularis [Sajjan 2015]. The presentation of blue toes in this context however is significant, and is thought to be more specific to microembolic events [Hirschmann 2009].

Management of sCES is primarily supportive: key pieces of management involve strict control of hypertension to minimize further pressure-driven embolization from ulcerated atheromatous plaques, use of statin and antiplatelet agents for plaque stabilization, and consideration of glucocorticoids for suppression of the inflammatory cascade driven by embolic events [Belenfant 1999, Yücel 2005]. There is minimal research available investigating optimal treatment strategies, and no current guideline-directed treatment for this condition currently exists; however, anecdotal reports and rare retrospective data support improved outcomes with directed supportive care [Belenfant 1999, Yücel 2005].

CES was briefly considered on our patient's initial presentation, but without prior angiographic or procedural intervention it was considered to be unlikely. Particularly given the improvement noted for the patient's livedo reticularis with warming, our initial differential favored cryoglobulinemic vs vasculitic etiologies driving the patient's presentation. Embolic etiologies were considered – but in the absence of fever, abnormal cardiac rhythms, hypoxemia, or typical dermatologic phenomena typically considered in embolic events of cardiac or venous origin (Janeway lesions, Osler nodes, Petechiae/Purpura, splinter hemorrhages), this was also considered to be unlikely. Our diagnostic error in this instance lay in failing to consider atypical embolic sources, with the abdominal aorta being a less commonly considered site for spontaneous embolic events.

Another important diagnostic aspect of this case lies in the consideration of renal biopsy, with the prior renal biopsy having resulted negative. As the patient reported that he had noted intermittent livedo reticularis prior to his presentation, we hypothesize that he had low-grade sCES causing his progressive renal dysfunction in the three months prior to his eventual admission. It may then be expected that evidence of cholesterol atheroemboli and histologic evidence of cholesterol clefts would be seen in the renal biopsy which occurred during the course of his disease. It is important to know that in this context renal biopsy has a sensitivity of approximately ~75% for cholesterol atheroemboli in the setting of acute disease, and is thought to be much lower in the subacute or chronic phase, the phase of disease in which our patient received his biopsy. If considering cholesterol embolization within the differential diagnosis, biopsy of skin lesions may be less morbid and of higher diagnostic yield, with an estimated sensitivity of 92% [Scolari 2010].

Shortly following this case, this writer identified a similar presentation for a patient with significant risk factors for vasculopathy including type 2 diabetes and renal disease, presenting with altered mental status, blue toe syndrome, acute renal failure, and livedo reticularis. Having so recently cared for the patient in our vignette above, we obtained a CT Angiogram of the abdomen to better delineate his vasculature, which identified a large ulcerated atheroma of the abdominal aorta, ultimately leading to a probable diagnosis of sCES. This exemplifies the importance of understanding and disseminating atypical patient presentations as seen in this vignette, to better expedite diagnosis and patient care in future cases.

References:

Ozkok A. Cholesterol-embolization syndrome: current perspectives. *Vasc Health Risk Manag.* 2019 Jul 8;15:209-220. doi: 10.2147/VHRM.S175150. PMID: 31371977; PMCID: PMC6626893.

Hirschmann JV, Raugi GJ. Blue (or purple) toe syndrome. *J Am Acad Dermatol.* 2009 Jan;60(1):1-20; quiz 21-2. doi: 10.1016/j.jaad.2008.09.038. PMID: 19103358.

Sajjan VV, Lunge S, Swamy MB, Pandit AM. Livedo reticularis: A review of the literature. *Indian Dermatol Online J.* 2015 Sep-Oct;6(5):315-21. doi: 10.4103/2229-5178.164493. PMID: 26500860; PMCID: PMC4594389.

Belenfant X, Meyrier A, Jacquot C. Supportive treatment improves survival in multivisceral cholesterol crystal embolism. *Am J Kidney Dis.* 1999 May;33(5):840-50. doi: 10.1016/s0272-6386(99)70415-4. PMID: 10213638.

Yücel AE, Kart-Köseoglu H, Demirhan B, Ozdemir FN. Cholesterol crystal embolization mimicking vasculitis: success with corticosteroid and cyclophosphamide therapy in two cases. *Rheumatol Int.* 2006 Mar;26(5):454-60. doi: 10.1007/s00296-005-0012-4. Epub 2005 Jul 16. PMID: 16025335.

Scolari F, Ravani P. Atheroembolic renal disease. *Lancet.* 2010 May 8;375(9726):1650-60. doi: 10.1016/S0140-6736(09)62073-0. Epub 2010 Apr 8. PMID: 20381857.

#37 Beyond Primary Hyperaldosteronism: Investigation of Refractory Hypertensive Hypokalemia

Authors: Jack Ruske MD, University of Utah; Nathan Allred MD, Intermountain Health

A 76-year-old male with past medical history of hypertension, anxiety, and parkinsonism presented to primary care clinic for follow up of a recent hospitalization. The week prior, he had run out of his chronic benzodiazepine and was found down with a principal complaint of chest pain and shortness of breath. He was hypertensive at 196/85 with a heart rate of 120-150. Symptoms improved with lorazepam. His potassium was 2.3, lactate 10.2, and troponin was negative. A withdrawal seizure was suspected; however, he continued to have hypertension and hypokalemia. He was admitted for five days and underwent a full cardiac and stroke workup that was negative. He was discharged with a refill for lorazepam and a new prescription for mirtazapine; there were no other changes to his medication regimen.

At follow up in primary care clinic he had resumed his previous regimen of medications and felt much better. Vitals were normal, exam at baseline, but labs showed persistent mild hypokalemia with a potassium of 3.1 mmol/L. He was not on any medications expected to cause hypokalemia, and it was decided to encourage potassium-rich foods and follow up labs in a month. At that next lab draw, his potassium had decreased to 2.2 mmol/L and he was referred to the ED. At this time, he complained of fatigue and lower extremity weakness. He was hypertensive in the 160s over 80s. EKG demonstrated sinus bradycardia, flattened T waves, and QTc 518. The patient was admitted to the hospital for further monitoring and investigation of his persistent hypokalemia and metabolic alkalosis.

During admission, the patient was aggressively repleted with potassium. He also had a very high urine output of 5 L/day with low urine osmolality. Given his presentation with severe treatment resistant hypokalemia, borderline elevated sodium, and hypertension, nephrology and endocrinology were consulted. The initial concern was for mineralocorticoid excess, and the patient was tested for both Cushing's disease and primary hyperaldosteronism. Early AM cortisol level was normal, and his renin and aldosterone levels returned undetectable. Further investigation was pursued for syndrome of apparent mineralocorticoid excess. At the suggestion of endocrinology, further discussion with the patient revealed that he had been eating 2 to 3 pounds of black licorice a week for the last 2 to 3 months.

For the next 5 days his BP was managed with repeated as needed doses of labetalol and he received aggressive potassium and magnesium repletion, requiring a total of 860 mEq PO potassium, 460 mEq IV potassium, and 18g magnesium. He was discharged on spironolactone 50 mg for 14 days, potassium 40 meq twice daily for 5 days and then 40 meq daily for 5 days. At primary care follow up, his blood pressure was found to be 89/55 with symptoms and a prerenal AKI. His potassium had normalized and his antihypertensive regimen modified.

In summary, licorice-induced pseudo aldosteronism, caused by glycyrrhizic acid, is rare in the general population but is likely underrecognized and can be severe, especially with chronic or excessive intake. Awareness and careful history-taking are essential in patients presenting with unexplained hypertension and hypokalemia. This case demonstrates the potential chronic effects of glycyrrhizic ingestion on blood pressure and the subacute dangers associated with increased intake.

#38 Lupus Enterocolitis as an Initial Presentation of Systemic Lupus Erythematosus: A Diagnostic Challenge in Acute Abdominal Pain

Authors: Maci Winn, PhD; Sarah E. Norton, MD, MSc-GH, MA; Claire Ciarkowski, MD, FACP, SFHM

Background:

Enterocolitis is an uncommon but potentially life-threatening gastrointestinal manifestation of systemic lupus erythematosus (SLE), presenting with nonspecific symptoms like abdominal pain, vomiting, and diarrhea that mimic infectious or inflammatory enteritis. Diagnosis is particularly challenging without a known autoimmune history. Early recognition and corticosteroid therapy are critical to prevent complications and improve outcomes.

Case

Presentation:

A 29-year-old female presented with a two-week history of progressive abdominal pain, nausea, vomiting, and non-bloody diarrhea. Two weeks earlier, she underwent laparoscopic right ovarian cystectomy and appendectomy for pelvic pain and nausea at an outside hospital, with initial symptom resolution. She subsequently developed persistent vomiting and diarrhea. Initial evaluation included edematous enteritis on abdominal computed tomography (CT), mild gastritis on esophagogastroduodenoscopy, and an unremarkable gastrointestinal pathogen panel, colonoscopy, and small bowel study. Histopathology of the ovarian cyst and appendix was benign. She was discharged after tolerating oral intake with antiemetics.

One day after discharge, she re-presented with worsening abdominal pain and numerous episodes of vomiting and diarrhea daily. She denied fever, chills, antibiotic use, travel, or sick contacts. Family history was non-contributory. Vital signs were stable, and physical exam revealed diffuse abdominal tenderness most prominent in the suprapubic and epigastric regions without rebound or guarding. No rash, oral ulcers, arthritis, or lymphadenopathy were noted. Labs revealed normal blood counts, non-anion gap metabolic acidosis, mild hyponatremia (Na 146 mmol/L), and proteinuria (urine protein-to-creatinine ratio 560 mg/g). Inflammatory markers were normal. Stool studies suggested osmotic diarrhea.

Repeat abdominal CT showed diffuse bowel wall thickening and submucosal edema involving the entire gastrointestinal tract, with moderate ascites, bladder wall thickening, and bilateral hydronephrosis attributed to extrinsic ureteral compression. Given concern for infectious peritonitis, she was empirically started on piperacillin-tazobactam. However, diagnostic paracentesis revealed no infection or malignancy.

Given her significant abdominal distension and recent surgery, surgical evaluation was pursued to rule out postoperative complications or acute abdomen. However, imaging and clinical findings did not suggest a surgical etiology, so conservative management continued. Despite supportive care, she developed worsening distension, new joint swelling, and superficial venous thromboses in bilateral upper extremities. Persistent gastrointestinal symptoms, submucosal edema on imaging, joint findings, and signs of coagulopathy prompted rheumatologic evaluation. Further workup revealed a markedly elevated antinuclear antibody titer (1:1280), low complement levels (C3 and C4), positive anti-Sjögren's syndrome-related antigen A (anti-SSA) antibodies, and lupus anticoagulant, fulfilling Systemic Lupus International Collaborating Clinics (SLICC) criteria for SLE.

Treatment and Outcome:

A diagnosis of lupus enterocolitis as the initial manifestation of SLE was established. The patient was started on high-dose corticosteroids, resulting in rapid symptom improvement. She was discharged on a prednisone taper with outpatient rheumatology and primary care follow-up.

Discussion:

This case highlights diagnostic challenges of lupus enterocolitis, especially without a prior SLE diagnosis. CT findings, including mucosal hyperenhancement, submucosal edema, mesenteric vessel engorgement (the 'comb sign'), and ascites, though non-specific, provide clues when correlated with serologic evidence of autoimmunity. Prompt diagnosis and immunosuppression are critical to prevent bowel ischemia, perforation, and morbidity. Early recognition may also prevent unnecessary interventions and imaging.