

Clinical Vignette Competition

2023



UTAH ACP RESIDENTS & FELLOWS COMMITTEE

Emily Signor, MD – Chair

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FALL CLINICAL VIGNETTE PROGRAM | THURSDAY, NOVEMBER 2, 2023

University of Utah | School of Medicine | Internal Medicine Grand Rounds

12:00 PM	WELCOME & OPENING REMARKS Residents & Fellows Committee	JUDGES Aaron Crosby, MD Rick Rose, MD John Gerstenberger, MD
12:10 PM	PRESENTATIONS	
	A Rare Case of Acute Liver Injury Presents a Diagnostic Dilemma <i>Presented by: Deborah Furman, MD [PGY2]</i>	Pg. 3-4
	Help, I've Stopped Cloza-peeing! <i>Presented by: Ellenor Chi [MS4]</i>	Pg. 13
	Barbeque with a side of Escherichia coli <i>Presented by: Elisha Eggink [MS4]</i>	Pg. 15
	Orange You Glad I Came In? <i>Presented by: Caroline Wang-Crocker [MS4]</i>	Pg. 17
	Acute liver injury in a 19-year-old woman <i>Presented by: Jacob Winter [MS4]</i>	Pg. 18
12:50 PM	ANNOUNCE RUNNERS-UP AND 1ST PLACE	
1:00 PM	CLOSING COMMENTS Residents & Fellows Committee	

UTAH ACP RESIDENTS & FELLOWS COMMITTEE | MISSION STATEMENT

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

- 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.
- Foster educational Opportunities for Internal Medicine Residents.**
 - Encourage participation in local and national ACP – ASLIM 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.
- 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.
- Identify public policy concerns of residents.**
 - Monitor local and national health policy and how it relates to Internal Medicine and residency training.
- Encourage an interest in community service.**
 - Identify ways associates can become involved with community service in Utah.

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MULTIFACETED CASUES | NEWSHA SEDGHI MSIII

Case Presentation: A 29-year-old male with epilepsy presented with a three-day history of vomiting and acute-on-chronic nausea. The emesis was non-bloody and non-bilious, accompanied by an inability to tolerate oral intake and subjective fevers. Additionally, the patient reported several months of preceding symptoms, including occasional diarrhea, easy bruising, and bleeding gums.

On arrival to the ED, vital signs were notable for fever (38.5°C) and tachycardia (108 bpm). Examination revealed a pallor, scattered ecchymoses, gingivitis, and mild epigastric abdominal pain on light palpation. Laboratory results showed a low WBC (1.43 k/uL), ANC (0.4 k/uL), and Hgb (8.3 g/dL). Abdominal and head CT scans were unremarkable. The patient was admitted for neutropenic fever and evaluation of new bicytopenia.

Differential Diagnosis: Diagnoses considered included marrow failure due to nutritional deficiencies, malignancies (leukemia, myelodysplastic syndrome, lymphoma), infections (HIV, parvovirus, EBV), medication side effects (lamotrigine), and autoimmune diseases (SLE). Extensive testing revealed deficiencies in copper, iron, thiamine, vitamin B12, A, D, and B6. A celiac panel showed low IgA levels but negative tTG antibodies. Further testing showed immunoglobulin levels were low in all classes.

Treatment: The patient received copper, iron, thiamine, vitamin B12, and multivitamin repletion. His ANC recovered began to recover, and he remained afebrile. No source of infection was found. He was discharged home. Follow-up lab work one week later showed continued improvement of leukopenia and anemia. The patient was referred to Immunology and is undergoing evaluation for Common Variable Immunodeficiency (CVID).

Discussion: The patient's presentation of profound bicytopenia was presumed secondary to his multiple micro- and macro- nutrient deficiencies. Copper deficiency in particular is known to cause neutropenia and anemia. The underlying cause of these nutritional deficiencies remains unclear. The suspected etiology was malabsorption due to CVID. GI complications are common in CVID, affecting 10-20% of patients with this disease. Potential complications include small intestinal bacterial overgrowth (SIBO), CVID enteropathy, or chronic giardiasis, all of which can cause malabsorption. SIBO in particular is underdiagnosed and aligns with the patient's symptoms.

Conclusion: This case emphasizes the importance of testing for nutritional deficiencies in cytopenic patients, recognizing clinical signs of micronutrient deficiencies like gingivitis, easy bruising, diarrhea, and nausea. It also underscores the significance of considering immunodeficiencies as a potential, albeit uncommon, cause of malabsorption.

Identification & Chief Complaint: 65-year-old male with jaundice

History: Patient is a 65-year-old male with a history of hyperlipidemia and osteoarthritis who presents with jaundice, diarrhea, and nausea. He recently took a six-week course of terbinafine for onychomycosis which he discontinued one week after jaundice appeared. He was briefly hospitalized at an OSH with elevated LFTs and bilirubin and was discharged after these began to normalize. He reports persistent nausea, vomiting, and diarrhea since symptom onset. He has noted that his urine is dark. Since stopping terbinafine he reports no improvement in symptoms.

Physical Abnormalities: Alert and oriented. Marked jaundice with scleral icterus. Abdominal exam revealed no organomegaly. Neurological exam without abnormalities.

Lab Results: Elevated bilirubin (23.7 mg/dL), Alk phos (861 unit/L), AST (430 unit/L) and ALT (649 unit/L) as well as leukocytosis (30.0 K/mcl), thrombocytopenia (61 K/mcl) and monocytosis of 63%. INR 1.2. Viral testing and autoimmune serologies negative. Abdominal US and MRCP were negative. WBC climbed precipitously over the following days, reaching the 100K; peripheral smear revealed 10% promyelocytes. Subsequent bone marrow biopsy and flow cytometry revealed highly proliferative acute myeloid leukemia with monocytic differentiation. Liver biopsy revealed diffuse sinusoidal involvement with monocytic infiltrate; features compatible with myeloid sarcoma.

Differential Diagnosis: DILI secondary to terbinafine, vanishing bile duct syndrome, autoimmune hepatitis, acute viral hepatitis, malignancy, Budd-Chiari syndrome, cholangitis, and choledocholithiasis.

Case Presentation: After pathology confirmed AML, the patient was transferred to the bone marrow transplant unit for induction treatment with hydroxyurea, cytarabine, cladribine, and ventoclax. His condition rapidly declined, and he developed acute kidney injury secondary to tumor lysis syndrome, acute liver failure secondary to infiltrative myeloid sarcoma, diffuse pulmonary alveolar hemorrhage due to thrombocytopenia, acute hypoxemic respiratory failure requiring intubation, and septic shock secondary to line associated bacteremia. The decision was made to transition to comfort care and the patient died 17 days after initial admission.

Discussion & Conclusion: Acute liver injury is a common presentation in the hospital setting. Our patient highlights the utility of a stepwise approach with eventual liver biopsy when the diagnosis remains obscure. Although DILI is considered in many patients, it is important to maintain a broad differential including malignancy. In this patient, without

known AML and with normal synthetic function, our approach led to the diagnosis of myeloproliferative neoplasm with extramedullary progression. Myeloid sarcoma is rare, occurring in 3-5% of AML patients. It typically involves the bones, lymph nodes, and soft tissues, with the liver involved in as few as 4% of such cases¹. Our case of hepatic myeloid sarcoma is unusual in the literature as the sinusoidal involvement was diffuse without notable mass. Hepatic involvement is particularly problematic as infiltration of the hepatic sinusoids contributes to multisystem organ failure and leads to unpredictable metabolism and toxicity of chemotherapeutics. This case highlights the need for expedited workup in patients with acute liver injury even with intact synthetic function.

1. Tomasian A, Sandrasegaran K, Elsayes KM et-al. Hematologic malignancies of the liver: spectrum of disease. *Radiographics*. 2015;35 (1): 71-86. doi:10.1148/rg.351130008

Case Presentation:

We present a newly described benign, soft tissue tumor found within the back muscles of a 66-year-old male with a past medical history of HTN, HLD, OSA. After a diagnosis of malignant spindle cell neoplasm was made, the case was referred to the Sarcoma Department and the mass was excised.

Intraoperatively, it appeared to be well encapsulated and was sitting in between the muscle bellies. On gross examination, the tumor was a well-circumscribed, heterogeneous mass, measuring 5.8 x 3.5 x 2 cm, tan-brown to white-yellow cut surfaces with focal areas of hemorrhage and without definitive necrosis.

Lab Results:

H&E sections show a well-circumscribed mass composed of relatively uniform, bland spindle cells within a variably myxoid-to-collagenous stroma, scattered lymphoplasmacytic infiltrates, and prominent and complex vascular pattern, some with hyalinized walls and fibrin depositions. Immunohistochemical stains were performed; the neoplastic cells showed EMA immunoreactivity with retained Rb nuclear staining, but negative for CD34, Pan-cytokeratin, MUC4, SMA, Desmin, and S100 protein. UCSF500 cancer gene panel test was performed, which revealed a pathogenic translocation involving AHRR, the gene encoding the aryl hydrocarbon receptor repressor, and NCOA2, the gene encoding the nuclear receptor coactivator

2. Overall, the clinical, histological, and immunohistochemical findings are most consistent with angiofibroma of soft tissue (AFST).

Differential Diagnosis:

Presumed diagnosis was malignant neoplasm until biopsy and pathology was obtained. Based on vascular pattern and stroma, the possibility of a myxoid liposarcoma was considered but lack of lipogenic differentiation argues against it. Therefore, to further characterize the lesion and confirm the diagnosis UCSF500 cancer gene panel was performed as noted above.

Discussion/Conclusion:

Genetic testing is an essential component of accurate diagnosis as the appearance of these lesions can mimic other, more harmful neoplasms. AFSTs often contain a unique NCOA2 fusion gene as well as other identifiable positive markers which include desmin, CD34, α -SMA, and epithelial membrane antigen. Immunohistochemical analysis

frequently shows the AHRR-NCOA2 driver mutation which is thought to be associated with t(5;8)(p15;q13), also commonly found in AFSTs. In combination, these immunohistochemical findings can help result in accurate diagnosis of angiofibroma of soft tissue in all the locations it can be found in. The accurate diagnosis of this tumor is essential to prevent additional unnecessary treatments and aggressive surgeries, and as mentioned above, this can be achieved by proper genetic testing for the presence of markers including the NCOA2 fusion gene, CD34, α -SMA, and epithelial membrane antigen.

History:

A 21 year-old woman with history of type 1 diabetes mellitus complicated by recurrent diabetic ketoacidosis, chronic pancreatitis, and hypertriglyceridemia presented with acute epigastric pain, nausea, and vomiting. She had several episodes of non-bloody, non-bilious emesis on the day of presentation. The severity of the epigastric pain was 10/10. She reported adhering to her insulin regimen of 45 U Glargine at night and 15 U Lispro with meals.

Physical Abnormalities:

Vital signs were notable for a heartrate of 96 and respiratory rate of 16. She was alert and oriented. She had normal work of breathing and no tachypnea. Her abdomen was soft, nondistended, and tender in the epigastrium.

Lab Results:

- Venous blood gas showed pH of 7.34, pCO₂ 37.5 mm/Hg, HCO₃⁻ 19.6 mmol/L, sodium 138 mmol/L, potassium 3.8 mmol/L, glucose 332 mg/dL, and lactate 1.70 mmol/L. The blood was turbid.
- A CMP demonstrated sodium 133 mmol/L, potassium 3.7 mmol/L, chloride 100 mmol/L, CO₂ < 5 mmol/L, normal creatinine, anion gap of > 28 mmol/L.
- Lipase was 341 (ULN 78 U/L). Triglycerides exceeded the limit of detection (> 6,400 mg/dL).
- Urinalysis was notable for glucose > 1000 and ketones > 160.

CT abdomen and pelvis with contrast demonstrated acute on chronic pancreatitis.

Discussion and Diagnosis:

This patient presented with acute on chronic pancreatitis with an equivocal diagnosis of DKA. She had ketonuria and hyperglycemia, but the CMP and VBG values for bicarbonate were markedly discordant (< 5 on CMP vs. 19.6 on VBG). This discrepancy was attributed to profound lipemia (TG > 6,400 mg/dL), which caused turbidity that interfered with the spectrophotometry assay used to measure CO₂ in the CMP, a phenomenon known as “pseudohypobicarbonatemia” (1). While ultracentrifugation can clarify a sample by separating lipoproteins from plasma, it risks the escape of CO₂ from the sample to the atmosphere, again leading to falsely low bicarbonate measurements. In contrast, a blood gas analyzer directly measures pH and pCO₂ using electrodes, neither of which are affected by lipemia. Bicarbonate in the sample is then calculated using the Henderson-Hasselbalch equation: $\text{pH} = \text{pK}_a + \log_{10}\left(\frac{[\text{A}^-]}{[\text{HA}]}\right)$.

Altogether, this patient had acute on chronic pancreatitis with hypertriglyceridemia and only mild diabetic ketoacidosis. This case illustrates how calculated bicarbonate on a VBG can in some cases be more accurate than the measured value on a chemistry panel.

Reference:

1. Varghese V, Griener D, Qu Q, Velez JCQ. Pseudohypobicarbonatemia in severe hypertriglyceridemia. *AJKD* 2020 76(4) P601-603

Identification/History: A 57-year-old female with a history of metastatic breast cancer with bony metastasis, pathologic fractures, and bisphosphonate-induced jaw necrosis presented with nausea and vomiting. She is currently not receiving systemic cancer therapy. She had a recent intramedullary nail placement following a pathologic hip fracture when walking downhill. She notes mild confusion during this time.

Physical exam/laboratory Abnormalities: The patient is an alert but fatigued-appearing woman of a stated age, with tearful affect, and no focal neurologic deficits. Abdominal, cardiac, and pulmonary exams are unremarkable. Labs are remarkable for Calcium:13.1 Creatinine: 0.60, PTH: 10, Vit. D25OH: 16, Vit. D 1,25 Dihydroxy: <5. Albumin 3.7 PTHrP: <2.

Ddx: The differential diagnosis for hypercalcemia includes hypercalcemia of malignancy, primary hypercalcemia, milk-alkali syndrome, adrenal insufficiency, Vit. D intoxication and hyperthyroidism.

Case presentation: The patient's labs were consistent with hypercalcemia of malignancy. Given the patient history of bisphosphate-induced jaw necrosis, endocrinology was consulted for the management of hypercalcemia. Bisphosphonates were initially avoided, given the absolute contraindication. The patient's treatment was limited to intravenous fluids, calcitonin, and cinacalcet. Calcium initially improved. However, progress slowed after a few days and remained >12. She initiated PARP therapy inpatient with the hope that it could control her calcium in addition to IV fluids.

Discussion: In addition to treating an underlying cause, the mainstay of treatment of hypercalcemia is fluids, bisphosphonates, zoledronic acid, and calcitonin. As this patient has a history of bisphosphate-induced necrosis, treatment with zoledronic acid or additional bisphosphonates. As seen in this patient, treatment with calcitonin loses effect over the course of about three days due to an effect known as tachyphylaxis.

Conclusion: This patient continued treatment with IV fluids and calcitonin with inadequate control of calcium. A multidisciplinary risk/benefits discussion was had between the patient, OMFS, endocrine, and oncology. Zoledronate was initiated, and the patient was discharged six weeks later with calcium levels of 8.3. The plan is to continue taking zoledronate along with calcitonin and IV fluids for calcium control for the next 4-6 weeks, at which point adequate control of underlying malignancy is anticipated.

Case Presentation: We present the case of a 44-year-old female who came to the ED for general fatigue, weight loss, and night sweats for the past year. She reported a past medical history of uterine prolapse with subsequent hysterectomy but has had overall limited interaction with the medical system due to her nomadic living situation. Most recently has travelled over multiple southwestern states. She has had previous evaluation in medical centers in different states where she traveled, but was lost to follow up when she moved.

Workup: Her work up was notably for CT scan 9 months ago with evidence of abdominal lymph node swelling. Initial work up in the ED was remarkable for elevated ESR at 103 and CRP of 7.5, otherwise initial labs and vital signs were within normal limits. On admission she was evaluated for possible chronic infection, given travel history and living situation, versus malignancy. She had extensive laboratory studies done that were all normal. CT abdomen and pelvis was performed that was largely unremarkable except for left common iliac lymphadenopathy with some evidence of central necrosis. Needle biopsy of the lymph node had histological features consistent with a metastatic carcinoma of either renal or gynecologic origin. Given positive PAX8 staining and lack of any evidence of renal mass or lesion it was presumed to be the latter with an unknown primary. She was later seen by gynecologic oncology and had an exploratory laparotomy with bilateral salpingoophorectomy with tumor debulking. Operative findings were notable for almost 20 sub centimeter punctate round lesions on the top third of the vaginal apex, some with central ulceration and confirmed metastatic clear cell adenocarcinoma on frozen pathology.

Discussion: Clear cell adenocarcinoma of gynecologic structures is rare accounting for 4-9% of cases of cervical adenocarcinoma and even less for primary vaginal lesion. Diagnosis is usually reliant on pathologic examination and is often difficult requiring the use of immunohistochemistry. This case illustrates the importance of thorough evaluation when pretest probability is high. This otherwise healthy patient had classic B symptoms and a significantly elevated ESR that suggested chronic disease with only minor abnormalities on imaging. Pathology was essential in diagnosis as the only evidence of malignancy was her necrotic lymph nodes. On tumor board review her case was discussed with no consensus of primary lesion, being either vaginal or uterine tube in origin. The difficulty in ascertaining a definite primary despite pathological and surgical diagnostics suggests that there is more to be learned and study regarding gynecological clear cell adenocarcinoma. This case also highlights the importance of adequate follow up for high-risk patients and the dangers of siloed healthcare.

References: Tantitamit T, Hamontri S, Rangsiratanakul L. "Clear cell adenocarcinoma of the cervix in second generation young women who are without maternal exposure to diethylstilbestrol: A case report." *Gynecol Oncol Rep.* 2017 Feb 22;20:34-36. doi: 10.1016/j.jgore.2017.02.008.

ID: An 81-year-old female with past medical history of OSA on CPAP, and paroxysmal atrial fibrillation presented with chest pain and shortness of breath

CC chest pain and shortness of breath

History: This patient had a history of paroxysmal atrial fibrillation previously treated with wide antral circumferential ablation and pulmonary vein isolation that was unsuccessful. She then underwent direct current cardioversion 4 days prior to presentation. She had been feeling well until the procedure but noted worsening fatigue, dyspnea on exertion as well as some shortness of breath for the few days after the procedure. She began to have substernal chest pain which prompted her presentation.

PE: Physical exam remarkable for a woman of stated age in moderate distress. Cardiac exam revealed tachycardia with regular rhythm, and no murmurs noted.

Labs: Troponin was elevated to 0.35 ng/ml, serial ekg were negative for ischemic changes. Complete blood count and chemistry panel were normal. Cardiac catheterization revealed minimal coronary atherosclerosis and normal LVEDP. Echocardiogram revealed a newly reduced LVEF of 20% (previously >50%), and hypercontractile basal segments and akinetic mid and apical segments.

DDX: Initially ACS was suspected, in addition to other causes of chest pain such as PE, pericarditis, myocarditis, and new heart failure. The patient's echocardiographic findings were diagnostic of stress induced cardiomyopathy. This diagnosis generally uses the modified Mayo Clinic criteria. The four criteria that need to be met include transient left ventricular dysfunction, absence of obstructive coronary disease, new electrocardiographic abnormalities or modest elevation in cardiac troponin and finally the absence of pheochromocytoma or myocarditis [1].

Treatment: While stress induced cardiomyopathy is generally reversible there are many serious acute complications to consider. Acute hypotension or shock is present in around 5-10% of patients, and new heart failure is present in around 30% of patients [2]. These conditions are managed as in any other patient population. Patients should expect recovery of systolic function in 1-4 weeks [3]. This patient responded well to medical management with losartan and metoprolol and was able to follow up in clinic with some recovered function.

Conclusion: While classically associated with an emotional stressor, stress induced cardiomyopathy has also been associated with physical stressors as well. There are several other documented case reports of stress induced cardiomyopathy as a rare but

serious complication of DCCV with important clinical implications for diagnosis, treatment and prognosis [4].

1. Prasad A, Lerman A, Rihal CS. Apical ballooning syndrome (Tako-Tsubo or stress cardiomyopathy): a mimic of acute myocardial infarction. *Am Heart J.* 2008 Mar;155(3):408-17. doi: 10.1016/j.ahj.2007.11.008. Epub 2008 Jan 31. PMID: 18294473.
2. Vallabhajosyula S, Barsness GW, Herrmann J, Anavekar NS, Gulati R, Prasad A. Comparison of Complications and In-Hospital Mortality in Takotsubo (Apical Ballooning/Stress) Cardiomyopathy Versus Acute Myocardial Infarction. *Am J Cardiol.* 2020 Oct 1;132:29-35. doi: 10.1016/j.amjcard.2020.07.015. Epub 2020 Jul 13. PMID: 32762963.
3. Sharkey SW, Lesser JR, Zenovich AG, Maron MS, Lindberg J, Longe TF, Maron BJ. Acute and reversible cardiomyopathy provoked by stress in women from the United States. *Circulation.* 2005 Feb 1;111(4):472-9. doi: 10.1161/01.CIR.0000153801.51470.EB. PMID: 15687136.
4. Zaghlol R, Hritani R, O'Donoghue S. Shock begets shock: A case of direct current cardioversion-induced takotsubo cardiomyopathy. *HeartRhythm Case Rep.* 2019 Mar 12;5(6):310-313. doi: 10.1016/j.hrcr.2019.02.013. PMID: 31285987; PMCID: PMC6587052. Sep;132(3):809-16. doi: 10.1378/chest.07-0608. Epub 2007 Jun 15. PMID: 17573507.

Case Presentation: Patient is a 37-year-old male with history of IV drug use, paranoid schizophrenia retriared on clozapine despite prior clozapine-related myocarditis due to limited treatment options, and major depression with suicide attempts presenting with one week of progressive weakness and fatigue. He is experiencing shortness of breath, dysphonia, minimal urine output, constipation, and myalgias. He is now unable to ambulate without support. Patient reports no IV drug use within one year, previously used heroin and meth. He is adherent with medications but has taken 1600 mg ibuprofen daily for myalgias.

Physical Abnormalities: Tachycardia to 100s. Muffled voice and stertor. Diffuse musculoskeletal tenderness to palpation with peripheral pitting edema.

Lab Results: CK 22000, Cr 10.96, BUN 103, phosphate 8, anion gap 17, lactate 1.6, AST 335, ALT 316, Troponin-I 0.05. FeNa 4.1%. Clozapine and metabolite levels within normal ranges. Myositis panel negative. HIV and HepB negative with positive HepC Ab. Imaging was unremarkable.

Case Resolution: Clozapine was immediately held given history of clozapine-induced myocarditis. For dysphonia and stertor, direct laryngoscopy was pursued and notable for only oropharyngeal edema and obstruction. He was treated with fluid resuscitation for AKI secondary to pigment-induced ATN from rhabdomyolysis. Patient developed worsening respiratory distress, with imaging findings suggestive of pulmonary edema and volume overload. He also became symptomatically uremic, requiring three sessions of hemodialysis. With continued fluid resuscitation, his renal function returned, though eventually developing post-ATN diuresis. All other workup was negative. CK downtrended to normal levels, and transaminitis concurrently resolved. However, off antipsychotics, he began to experience hallucinations. Ultimately, he was restarted on low-dose clozapine per recommendations from psychiatry.

Discussion: Clozapine-related rhabdomyolysis is a rare and poorly understood adverse effect without established management guidelines.¹ Treatment should include prompt cessation of clozapine and supportive care. Notably, this patient presented dysphonia and upper airway obstruction, suspected to be from oropharyngeal and esophageal skeletal muscle involvement and potential dystonic reaction. Levels of clozapine and metabolite were normal in this patient and do not correlate with risk for adverse effects. This patient was at risk given history of medication-induced myocarditis. Despite this, his treatment-resistant schizophrenia still necessitated reinitiation of clozapine.

Conclusion: Providers should be cognizant of atypical manifestations of clozapine adverse effects. Normal drug levels do not preclude abnormal side effects. Despite these risks, it is often necessary to continue antipsychotic medications in the setting of refractory psychiatric disease.

References:

1. Béchar, Laurent, et al. "Management of rhabdomyolysis in a patient treated with Clozapine: a case report and clinical recommendations." *Clinical Psychopharmacology and Neuroscience* 20.1 (2022): 194.

CASE PRESENTATION: 37-year-old male with a past medical history of severe erosive polyarticular gout was hospitalized with diabetic ketoacidosis following a COVID-19 infection. Patient reported mild symptoms during the infection, did not receive treatment, and was not immunized against SARS-CoV-2. Patient consulted an ophthalmologist for blurry vision one week prior to ICU admission on 10/06/2021 for fulminant DKA. He was diagnosed with Type 1 diabetes and discharged on glargine and lispro.

The patient reported an 80-pound weight loss during insulin treatment. A few months later, he was hospitalized with severe gout. Despite prednisone treatment, the patient did not require insulin for blood glucose control. He was discharged on metformin only with a diagnosis of Type 2 diabetes.

He presented to our endocrinology clinic 14 months later taking metformin 500mg twice daily. The patient had regained 30 pounds. He had no complications of diabetes including retinopathy, nephropathy, neuropathy, and macrovascular disease. The review of systems and physical exam were unremarkable.

LAB RESULTS: HbA1c levels since time of DKA were: 11.5% (11/2021), 5.4% (3/2022), 4.7% (7/2022), and 4.8% (2/2023). At follow-up on 2/8/23, lab results revealed an elevated c-peptide of 9.5 ng/mL, negative Type I diabetes antibodies (GAD, zinc transporter, IA-2, and islet cell cytoplasmic), and an albumin creatinine ratio of 4 mg/g.

DIFFERENTIAL DIAGNOSIS: The initial diagnosis was Type 1 diabetes based on severe DKA at time of presentation. His second hospitalization increased clinical suspicion for Type 2 diabetes as he no longer required insulin. Normalization of HbA1c to <5.6% supports the current diagnosis of viral-induced transient fulminant Type 1 diabetes.

DISCUSSION: The dramatic reduction in HbA1c from 11.5% to 5.4% over a four-month period is an atypical response to insulin and metformin. Since the patient stopped requiring insulin, in conjunction with negative pancreatic autoantibodies, the diagnosis of Type 1 diabetes is unlikely. Though elevation of C-peptide level suggests insulin resistance, the normalization of HbA1c does not support a diagnosis of Type 2 diabetes. The proximity of DKA to a COVID-19 infection suggests a potential inflammatory-mediated cause of transient loss in beta cell function. Long-term follow up is imperative in patients with new onset diabetes associated with COVID-19 infection.

CONCLUSION: COVID-19 is known to cause systemic inflammation. This case highlights the potential risk of COVID-19 induced pancreatitis and blood glucose dysregulation. Coordinated global efforts are currently underway to understand the transient versus permanent nature of COVID-19 induced beta cell damage.

Identification/Hospital Course:

A 48-year-old female with a history of bipolar disorder, hypothyroidism, and chronic low back pain presented with three days of nausea, vomiting, and diarrhea after eating at a BBQ restaurant. In the ED, she was found to have neutrophil predominate leukocytosis, normocytic anemia, and an AKI with a creatinine of 1.27 (baseline 0.6). Abdominal CT scan showed diffusely abnormal kidneys with decreased attenuation of the cortices bilaterally. Following admission, GI pathogen panel was positive for shiga toxin-producing *E. coli*. Labs were consistent with hemolytic anemia with an elevated LDH, low haptoglobin, worsening renal function (creatinine of 6.57), and blood smear with occasional schistocytes. ADAMSTS13 negative.

Patient then acutely decompensated with the development of altered mental status overnight. She had been taking oxycodone for chronic back pain.

Physical Exam/Laboratory Abnormalities:

Patient was unable to answer questions or follow commands. She was not oriented to self, place, time, or situation. No focal neurological deficits. Head CT without evidence of an intracranial process.

Differential Diagnosis:

The differential diagnosis for her altered mental status included uremic encephalopathy, oxycodone toxicity secondary to AKI, infection, CNS involvement of HUS, seizure, hypothyroidism, and electrolyte abnormalities.

Case Conclusion:

Given her diagnosis of STEC-HUS, uremic encephalopathy was the leading diagnosis. However, she did not improve with dialysis. She was then started on PLEX to decrease complement levels. An hour into initiation of PLEX, the patient had a seizure. LP was unrevealing. Brain MRI negative. Neurology and hematology were consulted and the patient was diagnosed with STEC-HUS with CNS involvement. She was treated with PLEX, eculizumab, antiepileptics, and dialysis. After a prolonged hospital course, she had renal and neurologic improvement and was transferred to rehabilitation for recovery.

Discussion:

Hemolytic uremic syndrome is a rare condition that typically presents with a triad of thrombocytopenia, hemolytic anemia, and AKI, often preceded by bloody diarrhea and vomiting. HUS with CNS involvement occurs in about 20-50% of cases, often with

symptoms of altered mental status and seizures¹. Typical neuroimaging findings include symmetrical involvement of the basal ganglia with predilection for the dorsolateral aspect of the lentiform nucleus¹. While few cases have been reported, CNS symptoms generally rapidly improve with treatment of the underlying HUS.

1. Mansour MA, et al. Hemolytic uremic syndrome with central nervous system manifestations, a case report and literature review. *Radiol Case Rep.* 2023 Apr 18;18(6):2268-2273. doi: 10.1016/j.radcr.2023.02.035. PMID: 37128253; PMCID: PMC10147953.

Identification: A 58-year-old male with a history of metastatic pelvic fibrosarcoma and T2DM presented with intermittent, asymptomatic, nighttime hypoglycemia for the last week.

Chief Complaint: Hypoglycemia

History: A 58-year-old male with a history of metastatic pelvic fibrosarcoma and T2DM presented with intermittent hypoglycemia for the last week. Blood glucoses at home were in the 50s for which the patient was drinking orange juice. Of note, he endorsed early satiety with poor PO intake. He stopped taking metformin for a week prior to admission. He had persistent hypoglycemia which prompted further work up. He was initially treated with a D10 drip but remained hypoglycemic when attempting to wean him off dextrose.

Physical Abnormalities: On exam, he appeared cachectic, diaphoretic, and lethargic.

Lab Results: CBC was notable for mild leukocytosis with WBC 12k. CMP was unremarkable. Further work up was as follows: Sulfonylureas panel was negative. Insulin level, c-peptide, pro-insulin, and beta hydroxybutyrate were at the low end of normal. AM cortisol was within normal limits. Insulin growth factor-1 (IGF-1) and IGF-2 were low; however, the ratio of IGF-2 to IGF-1 was 3.5.

Differential Diagnosis: Differential diagnosis included sepsis, adverse effects of sulfonylureas, surreptitious insulin use, insulinoma, Dodge-Potter Syndrome and adrenal insufficiency. He had a negative sulfonylurea panel. With normal insulin level, c-peptide and beta-hydroxybutyrate, surreptitious insulin use and insulinoma were unlikely. The patient had normal chemistries and AM cortisol which argued against adrenal insufficiency. Doege-Potter was the presumed diagnosis given his IGF-2 to IGF-1 ratio was greater than 3. To establish a diagnosis, the patient would need a ratio greater than 10.

Treatment: Definitive treatment of Doege-Potter Syndrome involves treatment of the primary cancer; however, this patient did not have further treatment options. Treatment for hypoglycemia would then entail use of high-dose glucocorticoids which help reduce the production of IGF-2 from the tumor. Ultimately, this patient benefited from high-dose glucocorticoids despite representing with worsening hypoglycemia. This was related to progression of disease as well as early satiety caused by a large left thoracic mass causing mass effect on the GE junction.

Conclusion: Doege-Potter Syndrome is a rare paraneoplastic syndrome found in solitary fibrous tumors and occurs in less than 5% of these patients. In Doege-Potter Syndrome, the tumor overproduces IGF-2 which binds to insulin receptors causing internalization of glucose. Definitive treatment involves treating the underlying malignancy, as corticosteroids for hypoglycemia prevention.

Chief Complaint: “Bloating”

History: A 19 year-old woman with history of asthma and EVALI presented with four days of progressive abdominal fullness, nausea, vomiting, and scleral icterus. She denied abdominal pain. Her emesis was non-bloody and non-bilious. She endorsed fevers, lower extremity edema, and a malodorous vaginal discharge. She denied dysuria and dyspareunia.

She drank at least one six-pack of beer daily, vaped tobacco daily, but denied other substance use. She owned a tattoo gun and tattooed herself and her friends. She had been sexually active with two male partners in the last year and did not use barrier protection. She did not take any medications nor supplements.

Physical Abnormalities: Vital signs were notable for heartrate of 130. She was jaundiced with scleral icterus. Her abdomen was distended, nontender, and soft, with a positive fluid wave. Genitourinary exam was unremarkable.

Lab Results: A CBC demonstrated microcytic anemia (MCV 65.4). BMP showed hyponatremia to 128, hypokalemia to 3.0, and normal creatinine. Liver function tests showed ALT 123, AST 290, Alk Phos 354, total bilirubin 11.2, albumin 2.9. INR was 2.0.

CT abdomen and pelvis with contrast revealed large volume ascites and a markedly abnormal liver concerning for extensive hepatic necrosis.

Differential Diagnosis and Further Workup: The patient’s urethral swab was positive for chlamydia, which raised concern for pelvic inflammatory disease and Fitz-Hugh-Curtis syndrome. However, this would not cause diffuse necrosis of the liver parenchyma and the patient did not have abdominal tenderness or dyspareunia. A1AT deficiency was considered given her history of asthma and EVALI, but her A1AT levels were normal. She had risk factors for HBV and HCV in amateur tattooing and unprotected intercourse, but a hepatitis viral panel was negative.

Unexpectedly, CMV IgM was above the limit of detection, plasma viral load was elevated (>11,000), and IgG was negative. Additional workup was unrevealing, including ferritin, HSV, HIV, ceruloplasmin, and an autoimmune hepatitis panel. Ascites fluid was negative for spontaneous bacterial peritonitis.

Conclusion: This patient’s presentation is most consistent with acute CMV hepatitis, which is remarkably rare in immunocompetent individuals (45 cases reported to date; PMID: [33604261](#)). Alcohol use likely primed her liver for injury in the context of CMV infection. She was treated with ganciclovir and doxycycline for acute CMV infection and chlamydia, respectively. On follow up, she had seroconverted her CMV IgG and had a decreasing CMV viral load.

Chief Complaint: sudden single episode of a seizure

Case Presentation: A 22-year-old female patient presents to the clinic due to a sudden episode of seizure one month ago. Denies any previous seizure. While getting out of bed, she felt herself convulsing and fell to the floor. Complains of memory loss after the episode and no symptoms leading to the seizure or afterwards. Medical history is significant for hypothyroidism, anxiety and depression, and PANDAS and PANS. She abruptly stopped taking Lexapro a month ago because she stated her depression and anxiety had become more manageable. She contacted her primary care provider, which he recommended to discontinue taking her birth control, and that mold toxicity may be something to look out for. Overall, the patient feels fine, but she seemed fatigue and had slow speech.

History: History of hypothyroidism and taking levothyroxine, history of anxiety and depression which patient was previously taking Lexapro but abruptly discontinued due to symptoms being more manageable. Patient discontinued birth control after the seizure. History of PANDAS and PANS.

Physical Abnormalities: Overall speech was slow. General physical appearance of patient looked fatigued. Patient's blood pressure was 116/80 and heart rate was 51. Due to bradycardia and overall health concerns and EKG was performed in house and only significant for sinus bradycardia.

Lab Results: TSH, T3, T4 all within normal rages

Differential Diagnosis: Single episode of seizure that may potentially have different causes. Can consider PANDAS and PANS history and mold toxicity exposure leading to seizure, abrupt discontinuation of Lexapro, or other neurological imbalances.

Discussion: Research has found that people with PANDAS and PANS may have some immunodeficiency, which makes fighting off infections even more difficult. And especially considering mold toxicity that may have contributed to her random onset of a seizure. Although rare, it is also important to consider abrupt discontinuation of Lexapro during the same time period of when this patient's seizure came about. There is a myriad of culprits to this patient experiencing a single episode of a sudden seizure, and that is why it is vital to monitor and look at all the aspects that may contribute to this condition.

Conclusion: Since this patient was seen in an outpatient clinic, EKG was preformed, which demonstrated sinus bradycardia, and thyroid levels came back within normal range. This patient was advised to get an MRI and follow up with a neurologist for further