

ORAL CLINICAL VIGNETTES - 2017

1. **Dr. Sayed Mohammad Hosseini**
GBMC
Integrative Toxicity: A Case of Unexpected Methotrexate Affect
2. **Dr. Fahid Alghanim**
Johns Hopkins/Bayview
Denture Paste: A Culprit in Zinc Toxicity and Resulting Copper Deficiency
3. **Dr. Joanna Wang**
Johns Hopkins/Broadway
Treatment of Severe Recurrent Gastrointestinal Bleeding with Bevacizumab in a Patient with Hereditary Hemorrhagic Telangiectasia-Like Phenotype
4. **Dr. Sarah Ahmad**
MedStar Harbor Hospital
Autoimmune Myelofibrosis in the Setting of IGG4-Related Disease
5. **Dr. Chisom Ikeji**
MedStar Health Internal Medicine
Death by Misadventure with Designer Drug N-Ethylpentylone
6. **Dr. Atul Matta**
St. Agnes Hospital
Why is My Patient Hypoxic?
7. **Dr. Harleen Kaur Dehal**
Sinai Hospital of Baltimore
Thyrotoxic Periodic Paralysis
8. **Dr. Andrew Mark**
UMMC
The AKA That Wasn't: The Kidney Wrongly Accused
9. **Dr. Matthew Evans**
UMMC/VA
Vibrio Vulnificus Infection in an Immunocompromised Patient Following Raw Oyster Ingestion
10. **Dr. Robert H. Wauters**
Walter Reed
Just Dance – Anaphylaxis at a Wedding

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Integrative toxicity: a case of unexpected methotrexate affect

Introduction:

Methotrexate (MTX) is one of the most widely used disease-modifying antirheumatic drugs for the treatment of rheumatoid arthritis (RA). While low dose MTX (LD-MTX) acts mainly as an anti-inflammatory drug by increasing tissue adenosine levels besides other mechanisms, high doses (HD-MTX) has anti-proliferative cytotoxic action with different toxicity profile and adverse effects. LD-MTX toxicities and adverse effects are rarely if ever life-threatening. Medical conditions that impair clearance may, however, result in sustained serum levels of the drug that induce toxicity.

We present a 60 y.o. Male, w/ a PMH of diabetes, borderline HTN, rheumatoid arthritis, and celiac disease who was evaluated for fatigue, abdominal pain and was found to have acute kidney injury and pancytopenia. Six months prior to the admission he had been diagnosed with rheumatoid arthritis and had been started on LD-MTX. Over the week before the admission, he noticed multiple ulcers in his mouth and throat and an erythematous, non-pruritic maculopapular rash on his neck, trunk, arms and legs. It spared his feet and hands. Lab evaluation showed a mild pancytopenia (WBC:2.39, Hb:10 and PLT: 138) with a reticulocyte count of 1.17. His calcium was 12.2 and his creatinine was 2.7.

Careful medication reconciliation revealed that the patient had been started on 50,000 units of D3 daily (not ergocalciferol) on the recommendation of a relative who was an integrative medicine specialist. He also continued NSAIDS in the face of hypervitaminosis D, hypercalcemia, and apparent volume depletion. The accumulated MTX likely exacerbated renal function with a resulting MTX level on admission of 1.1 micromole/l, consistent with a dose 100 times expected. The patient was started on leucovorin and vigorous IVF until resolution.

Conclusion:

Although NSAIDS are a known co-factor in HD-MTX toxicity, they are usually well tolerated in LD- MTX therapy in rheumatoid arthritis. As 38 % of Americans report using complementary and alternative medications, our case demonstrates the potential for these therapies to have complex interactions. Performing a detailed reconciliation of integrative therapies and anticipating potential interactions with traditional therapies may be crucial in recognizing toxicity.

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Denture Paste: A Culprit in Zinc Toxicity and Resulting Copper Deficiency

Copper deficiency is a rare condition that results in hematological and neurological complications; it can mimic vitamin B-12 deficiency and causes sub-acute combined degeneration.

A 65-year-old electrician with a history of diffuse atherosclerotic disease resulting in ischemic cardiomyopathy and abdominal aortic aneurysm with endovascular repair presented to our facility with a sub-acute onset of severe weakness in his legs and an unintentional weight loss of 30 lbs. He was hospitalized in August 2016 for frequent falls and reported the ability to ambulate independently 2 months prior to presentation. His leg weakness progressively worsened and, as a result, he trialed assisted ambulation with a cane, which he quickly replaced with a walker. He also reported numbness/tingling in his hands and feet, perianal numbness, and new onset stool incontinence. His labs were notable for a normocytic anemia since August 2014 and leukopenia since December 2015. He had undergone CT of the chest, abdomen, and pelvis, which did not reveal an occult malignancy, and subsequently a CT of the thoracolumbar spine that was negative for any osseous lytic or sclerotic lesions. His most recent screening colonoscopy was unrevealing. Neurological consult was obtained and his physical exam was notable for decreased lower extremity bulk with diffuse 4/5 muscle strength in his bilateral lower extremities. He also exhibited severely diminished proprioception in his distal upper and lower extremities and a dependent deficit of fine and crude touch along his arms and legs and a small area perianally. His reflexes were diffusely diminished aside from 3+ and symmetric patellar reflexes and up-going plantar reflexes. There was a high suspicion for myelopathy, which was confirmed with MRI of the spine showing dorsal column T2-weighted hyper-intensity consistent with sub-acute combined degeneration. Laboratory studies were consistent with copper deficiency at 3 mcg/dL and low ceruloplasmin level at 2 mg/dL. 24-hour urine copper was normal at 6 mcg/L. Ophthalmology consult was obtained and there was no evidence of Kayser-Fleischer rings, but he did have left optic neuropathy. His Vitamin B-12 level was normal at 615 pg/mL. His HIV and syphilis screens were negative. Vitamin E level was normal. Thus, the patient was diagnosed with copper deficiency. The etiology of copper deficiency was not entirely clear, but was thought to be related to his 35-year use of a zinc containing denture cream and resultant relative zinc excess. The patient was treated with copper repletion and cessation of zinc-containing denture cream use. His cytopenias have since resolved and his neurological deficits have greatly improved.

This case illustrates the importance of including copper deficiency in the differential diagnosis of subacute combined degeneration. Early recognition of copper deficiency could eliminate further neurological compromise and improve quality of life.

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Title: Treatment of severe recurrent gastrointestinal bleeding with bevacizumab in a patient with a hereditary hemorrhagic telangiectasia-like phenotype

History of present illness: A 77-year-old man with a history of hypertension and mild aortic stenosis was transferred from an outside hospital for a 15-month history of recurrent melena. He had been admitted nine times to outside hospitals with severe anemia and melena requiring transfusions. Multiple upper endoscopies and colonoscopies revealed arteriovenous malformations (AVMs) of the proximal small bowel, treated with argon plasma coagulation (APC). On admission, he endorsed a two-year history of mild epistaxis, which was also present in one first degree relative. His blood pressure was 118/50, SpO₂ 99% on room air. His exam was notable for telangiectasias of the cheeks, chest, lips, and pharynx, and a grade II/VI systolic ejection murmur heard at the left upper sternal border. His labs were notable for iron deficiency anemia with hemoglobin 9.1 g/dL.

Clinical course: Over the next three months, he presented four times, spending only weeks between admissions. He underwent a total of seven upper endoscopies, colonoscopies, video capsule endoscopies, and double-balloon enteroscopies, revealing innumerable small bowel AVMs treated with repeated APC. Contrast echocardiogram revealed mild aortic stenosis without intracardiac or pulmonary shunt. His von Willebrand factor multimer level was normal and genetic testing for HHT was negative. However, his vascular endothelial growth factor (VEGF) level was elevated. CT angiography of the head, chest, and abdomen, and liver duplex ultrasonography did not show AVMs. Empiric combination therapy with aminocaproic acid, estrogen, octreotide, and tranexamic acid resulted in only transient improvement with continued transfusion requirement. Given his elevated VEGF level, he was started on bevacizumab infusions which were continued outpatient. Ultimately, his bleeding resolved and he has been without readmission for over five months.

Discussion: Hereditary hemorrhagic telangiectasia (HHT), or Osler-Weber-Rendu syndrome, is an autosomal dominant disorder characterized by mucocutaneous and visceral vascular malformations. Classically, individuals present with epistaxis, gastrointestinal (GI) bleeding, and iron deficiency anemia (IDA). The majority of patients with HHT present in the second decade of life, and over 90% of patients present by age 40. However, HHT is a heterogeneous condition displaying age-related penetrance, and is underdiagnosed in the general population.¹ Mutations in ACVRL1, ENG, and SMAD4 are usually implicated in HHT, but 15% of patients with suspected HHT have negative genetic testing.¹ There are reports of patients with an HHT-like phenotype who do not meet the Curacao consensus criteria. Other potential markers of disease include elevated levels of VEGF and other angiogenesis-related factors.² Prior case reports have shown the efficacy of bevacizumab, an inhibitor of VEGF-A, in treating severe epistaxis and GI hemorrhage associated with HHT. These patients experienced resolution of their bleeding with anti-VEGF therapy, and withdrawal of therapy was often followed by return of bleeding.³⁻⁶ This case illustrates the importance of understanding the pathophysiology of angiogenesis in HHT and demonstrates the potential benefit of bevacizumab therapy in patients who present with an HHT-like phenotype and elevated VEGF.

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AUTOIMMUNE MYELOFIBROSIS IN THE SETTING OF IGG4-RELATED DISEASE

Ahmad, S. MD, Eshetu, N. MD, Karrikeneth, A. MD, Williams, R. MD, FACP, Reed, J. MSIII, Canacevic, S. MSIII

Introduction: Autoimmune myelofibrosis (AIMF) is a distinct pathological entity characterized by diffuse bone marrow fibrosis and an overall benign clinical course. A variety of benign and malignant diseases can cause myelofibrosis including myeloproliferative diseases such as primary myelofibrosis (PMF). We present a case of a young man that presented with significant pancytopenia with evidence of systemic IgG4-related sclerosing disease.

Case Presentation: A 27-year old male with PMH of hypertension presents with a rash and yellow sclera for 1 month. He endorses fatigue, pruritus, darkening of his urine and 100-pound unintentional weight loss over the past 7 months. No recent travel history, illnesses, or illicit drug use. Physical exam was notable for obesity, a petechial rash on the extremities, and scleral icterus. Initial laboratory revealed leukopenia, anemia, transaminitis and hyperbilirubinemia. US Abdomen was negative for gallstones or obstruction. CT Abdomen showed mild intrahepatic biliary duct dilatation, without obstruction and acute pancreatitis with pseudocyst. MRCP showed tapering of common duct. CEA and CA 19-9 were negative. Acute hepatitis panel, Anti-mitochondrial Ab, Anti-smooth muscle Ab, liver/kidney microsome Ab, screen for autoimmune disease, JAK2 mutation were all within normal limits. Albumin to Globulin ratio was low. Immunoglobulin panel showed elevation of IgG4, 4 times the upper limit of normal. Biopsy of liver showed moderate inflammation of portal tracts with fibrosis and many IgG4 plasma cells, consistent with IgG4-related autoimmune sclerosing cholangitis. A diagnosis of AIMF was made, given the extensive myelofibrosis in the bone marrow biopsy and related autoimmune cholangitis from IgG4 related disease.

Discussion: AIMF is an under recognized cause of bone marrow fibrosis. Secondary AIMF is associated with an established autoimmune disorder such as systemic lupus erythematosus (SLE) while primary AIMF occurs in patients without a well-characterized autoimmune disorder but who have autoantibodies (such as IgG4). There have been limited cases of AIMF and the pathogenesis is largely unknown. Production of fibrogenic cytokines (such as transforming growth factor- β and substance P) by inflammatory cells is thought to be the cause. The primary treatment of AIMF is corticosteroids and for some cases immunosuppressive drugs such as methotrexate. Our patient was started on a trial of steroids, which lead to complete resolution of his pancytopenia, transaminitis, and hyperbilirubinemia.

Conclusion: AIMF, a rare but well-established clinicopathologic entity, can cause myelofibrosis in patients presenting with cytopenias and responds well to systemic steroids. While the spectrum of clinical presentation is broad, IgG4-related disease can be possibly linked to primary AIMF.

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DEATH BY MISADVENTURE WITH DESIGNER N-ETHYLPENTYLONE

N-Ethylpentylone is a cathinone derivative shown to have psychostimulant effects and has become newly available as a cheap substitute for stimulants such as methamphetamine and cocaine. Very little is known in regards to its clinical effects making management and predicting clinical prognosis difficult.

A 21-year-old man was brought to the hospital unresponsive after suffering a cardiac arrest in the field. His girlfriend reported that he left the house to smoke marijuana and came back behaving very strangely. Police arrived to his home after a disturbance call and it was reported that he was running and screaming, attempted to enter a marked police car, and then started banging on the door of a neighbor's home yelling, "Help! Call the police!" He was uncooperative, which resulted in a physical struggle requiring three officers to subdue him. Medics described him as agitated and diaphoretic with hot skin. 5mg of Haldol was effective in calming him but then he went into cardiac arrest shortly thereafter. CPR was done for 3 minutes with return of spontaneous circulation. Upon arrival to the hospital, the patient the was intubated, vital signs showed hypotension and tachycardia, and EKG showed sinus rhythm with premature atrial complexes and ST depression in inferior leads. On physical exam, he was noted to have multiple abrasions on his face, sluggish pupillary reflexes, negative vestibulo-ocular reflex, and myoclonus at right lower extremity. Clinical laboratory analysis revealed hyperkalemia, hypoglycemia, rhabdomyolysis, liver failure, renal injury, elevated troponin, pH 6.80 and lactic acid of 28. Tox screen was positive for cannabinoid and an ethanol level of 12. CT head showed no abnormalities; however, MRI of the brain showed bilateral restricted diffusion in the posterior parietal and occipital regions. Treatment was initiated with hypothermia cooling measures, intravenous fluids, and bicarbonate. EEG showed very low amplitude delta range background rhythm consistent with very severe encephalopathy. On day two, he was started on continuous renal replacement therapy due to renal failure and oliguria. Despite supportive treatment he developed severe hypocalcaemia, hypotension, leukopenia and thrombocytopenia. On day four, his hemodynamic status worsened, he became severely hypothermic and he went into cardiac arrest. CPR was performed for 30 minutes unsuccessfully. Autopsy findings sited the cause of death to be intoxication with N-Ethylpentylone.

This case is a rare example of unexpected death in relation to N-ethylpentylone drug. It demonstrates pathophysiological changes resulting from drug toxicity and provides information of the behavioral effects, clinical presentation, and sequelae of multiorgan failure.

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Why is My Patient Hypoxic ?

Atul Matta, M.D., Kimberly O'dell, MS4, Ammer Z. Bekele, M.D.

Introduction: Chronic hypoxia is the most common cause of secondary polycythemia. However, hypoxia unrelated to cardiopulmonary disease is rarely seen as a presenting complaint in primary polycythemia or polycythemia vera (PV). Here we present a case of PV who presented with hypoxic respiratory failure likely due to increased blood viscosity.

Case: A 59-year-old male presented complaining of progressive dyspnea for one month. His medical history was significant for hypertension, diastolic heart failure, and chronic kidney disease. He was a former smoker. Physical examination was significant for blood pressure 162/110 mmHg, oxygen saturation 86% on room air, tachypnea and coarse crackles at lung bases. Laboratory evaluation showed hemoglobin 21.2 g/dl, hematocrit 67.6%, normal leukocyte and platelet counts. Brain natriuretic peptide level was 111pg/ml and D-dimer 1.13mg/L. Chest x-ray was unremarkable. Polycythemia was thought to be secondary to chronic hypoxia; however, erythropoietin levels were ordered to be able to differentiate between primary and secondary causes.

Acute coronary syndrome, decompensated heart failure, and pulmonary embolism (PE) were considered to be possible etiologies. The patient was empirically treated enoxaparin, nitroglycerin paste, and furosemide. Lung ventilation perfusion scan came back as low probability for PE. The patient's respiratory status continued to deteriorate; he became more tachypneic, diaphoretic and subsequently required noninvasive mechanical ventilation. At this point hyperviscosity secondary to polycythemia was suspected. He was admitted to the ICU and emergent phlebotomy was performed. A total of 600ml of blood was phlebotomized over a period of three days. The patient's symptoms significantly improved and he was weaned to 2 liters of oxygen via nasal cannula. Erythropoietin levels came back low. JAK2V617F mutation assay was ordered and was positive. The patient was discharged with home oxygen and advised to follow up with a hematologist.

Discussion: Polycythemia vera is overproduction of red blood cells, platelets, and granulocytes by the bone marrow due to an activating point mutation in the tyrosine kinase, JAK2. This mutation most commonly results in a valine to phenylalanine substitution at residue 617. Constitutive JAK2 signaling decreases progenitor cell dependence upon erythropoietin and other growth factors for proliferation, thus cell counts are high while erythropoietin levels remain low. This is a point of differentiation between primary and secondary polycythemia. The resulting elevated hematocrit may lead to increased viscosity of the blood. In addition thrombocytosis and abnormal platelet function, due to morphologic abnormalities, portend a proclivity for thrombosis and bleeding. PV is uncommon and tends to have onset in the sixth and seventh decades of life. Symptoms are a result of abnormal blood flow. Patients commonly exhibit plethora, headache, dizziness, hypertension, and gastrointestinal symptoms. Pruritus may occur often after a hot shower due to release of histamine from basophils. Up to a quarter of the patients present with venous thromboembolism, myocardial infarction, or stroke. Hypoxia is generally considered to be the cause of secondary polycythemia; however, it can also be a manifestation of PV. In the past, studies have demonstrated that patients with PV with no coexisting cardiorespiratory disease had lower arterial oxygen tension and arterial oxygen saturation compared to normal subjects. This was attributed to decreased diffusion capacity that could result from an alteration in pulmonary vasculature due to unrecognized thromboembolism. Phlebotomy is the mainstay of treatment and delays progression to myelofibrosis. Aspirin and hydroxyurea as adjunctive therapies have been shown to reduce the incidence of thrombosis. Median survival is approximately 13 years with treatment compared to death within month from bleeding or thrombosis if left untreated.

Conclusion: Although rare, hypoxia could be a primary manifestation of PV likely secondary to decreased diffusion resulting from alteration in the pulmonary vasculature due to increased blood viscosity.

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Thyrotoxic Periodic Paralysis

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INTRODUCTION: Thyrotoxic periodic paralysis (TPP) is an uncommon presentation of hyperthyroidism. Though the prevalence of hyperthyroidism is much higher in women, men seem to be more likely to present with TPP with a reported male to female ratio 20.5:1. Higher prevalence has also been seen in Asian ethnicities. Though quite rare in the US in the past, this is becoming more frequently reported due to increasing migration and cultural mixing, making the recognition of this condition critical due to the widely different treatment approach as compared to other neurological causes of muscular weakness.

CASE PRESENTATION: Our patient was a 45-year old African American man with no prior diagnosed significant comorbidities, who presented to the emergency department (ED) with the chief complaint of weakness that started the morning prior to admission. Questioning revealed that the patient had suffered from similar sudden episodes in the past, usually triggered by prolonged inactivity, that would resolve spontaneously. Other associated symptoms included anxiety, nervousness and increased diaphoresis. His exam was notable for global hair loss, no thyromegaly and an initial motor exam consistent with a power of 2/5 in all muscle groups. This however, improved to 3 to 4-/5 over the course of a few hours. Initial labs showed severe hypokalemia (1.7mmol/L) and hypomagnesemia (1.3mg/dL) with EKG showing T wave inversion and U waves. CPK was elevated at 41,156 units/L with a normal renal function (creatinine 0.52mg/dL). Hence, aggressive IV magnesium and potassium supplementation was started, with complete reversal of paralysis. Due to a suspicion for hyperthyroidism and, in turn, thyrotoxic periodic paralysis (TPP), a TSH was sent, which was undetectable (<0.008 U/mL). Thyroid stimulating immunoglobulin, levels of free T3 and T4 were elevated (142, 8.8 pg/mL and 3.33 ng/dL respectively), supporting a diagnosis of Grave's disease. Hence, the diagnosis of TPP was established, and propranolol as well as methimazole were started.

CONCLUSION: TPP remains a lesser known albeit important cause of sudden onset weakness. Though it is more prevalent in males of Asian descent, it is becoming exceedingly more common in the western world due to population migration and admixture. Diagnosis of TPP can be easily missed, which can have grave repercussions especially if it associated with electrocardiac complications. Potassium supplementation remains the mainstay of treatment along with maintenance of a euthyroid state preventing recurrences.

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THE AKI THAT WASN'T: THE KIDNEY WRONGLY ACCUSED

A healthy 24 year-old man presents to the emergency department (ED) one morning complaining of acute abdominal pain with associated nausea, vomiting, and anorexia after an episode of difficult micturition the previous night. He denied any trauma. Past medical history was significant for primary enuresis until the age of 12, which resolved after an unknown abdominal surgery. On physical examination the patient was noted to have difficulty finding a comfortable position and he had supra-pubic fullness and tenderness with mild abdominal distention. An indwelling urinary catheter was placed and it immediately drained 1000 mL of translucent yellow fluid.

Initial laboratory tests revealed apparent AKI with serum creatinine of 1.88 mg/dL. This dropped to 0.9 mg/dL over the next twenty-four hours. A computerized tomography (CT) scan of his abdomen and pelvis identified large-volume ascites. His abdominal pain resolved and he tolerated oral intake by the day after admission, so he was discharged with a therapeutic paracentesis scheduled for two days hence. That procedure yielded 2000 mL of slightly turbid yellow fluid. The serum-ascites fluid gradient was greater than three, consistent with a transudative process. The procedure was otherwise uneventful and the patient returned home.

Three days later, the patient returned to the ED with a presentation similar to that of his initial admission. A comprehensive metabolic panel and complete blood count revealed a serum creatinine of 11.44 mg/dL and hemoglobin of 18.1 g/dL. Urinalysis was notable for 500mg/dL of proteinuria. An indwelling urinary catheter was again placed and this produced 1400 mL of pale-yellow urine. Aggressive intravenous fluid resuscitation led to a rapid drop in serum creatinine to 0.87 mg/dL over the next 36 hours. Fluoroscopic cystourethrogram revealed contrast extravasation from the bladder. Laparotomic surgical repair of his ruptured bladder was completed uneventfully. A cystoscopy done five days later revealed prominent trabeculae. Retrospective review of the CT scan from his initial admission showed that the sagittal images were very suggestive of an interruption of the well-demarcated contour of the bladder.

Spontaneous bladder rupture can masquerade as renal pseudo-failure, and should be considered in the setting of elevated creatinine and bladder dysfunction before embarking on an evaluation of AKI. Imaging plays an important role in making this uncommon but important diagnosis.

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VIBRIO VULNIFICUS INFECTION IN AN IMMUNOCOMPROMISED PATIENT FOLLOWING RAW OYSTER INGESTION. Evans MC, MD.
The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Vibrio vulnificus is a species of gram-negative, motile, comma-shaped bacteria typically associated with three types of infections: acute diarrheal illness, necrotizing wound infections typically associated with exposure to contaminated marine water, and bacteremia often presenting as septic shock, particularly in patients with chronic liver disease or those that are otherwise immunocompromised.

A 73-year-old man prescribed mycophenolate mofetil for a history of pulmonary fibrosis presented with a three-day history of nausea, vomiting, and diarrhea as well as a bilateral lower extremity rash. He was febrile to 39.0°C, tachycardic with a heart rate of 120 beats per minute, and hypotensive with a systolic blood pressure of 70mmHg. Physical examination was notable for weak peripheral pulses, mottling of his bilateral upper extremities and numerous bullae noted on his bilateral lower extremities.

Initial basic metabolic panel was notable for serum bicarbonate of 12 meq/L and serum creatinine of 3.03 mg/dL (from a baseline of 0.9 mg/dL). Serum calcium was 6.5 mg/dL and serum phosphate was elevated at 11.2mg/dL. Serum lactate level was 13.0 mmol/L. Upon further questioning, his family noted that five days prior to admission he had consumed raw oysters near his home along the Eastern Shore of Maryland, about two days prior to the onset of symptoms. Subsequently two out of two blood cultures returned positive for gram-negative rods which later speciated as *V. vulnificus*. He had initially been started on empiric broad-spectrum antibiotics with vancomycin and piperacillin-tazobactam, which were narrowed upon speciation of blood cultures to cefepime and doxycycline. His sedation was weaned to assess his neurologic status and he was noted to have absent brainstem reflexes. Vasopressors were discontinued at the request of his family and he expired approximately 12 hours after admission.

V. vulnificus bacteremia can be rapidly fatal, with mortality estimated at greater than 35%. The diagnosis should be considered in critically ill immunocompromised patients presenting with recent raw seafood ingestion or exposure to warm marine environments, particularly those presented with lower extremity bullae.

201

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JUST DANCE – ANAPHYLAXIS AT A WEDDING
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INTRODUCTION: Food-dependent exercise induced anaphylaxis (FDEIAn) is a rare subtype of anaphylaxis that occurs only when exertion takes place within a few hours of ingestion of certain foods, usually wheat. The exact mechanism of FDEIAn is unknown. Co-factors such as NSAID use, alcohol consumption, perimenstruation, and pollen exposure have been associated with reported cases. We describe a case of FDEIAn to crab in a healthy 32-year-old woman.

CASE: A 32-year-old woman presented with diffuse urticaria that started after vigorous dancing during a wedding. Symptoms quickly progressed to throat pruritus, coughing, and glossitis. Before dancing, the patient consumed crab cakes, steak, vegetables, one glass of wine, and one cocktail. She denied any use of NSAIDs. EMS was called and the patient was treated with intramuscular epinephrine, diphenhydramine, and corticosteroids during transport to emergency department. The patient's symptoms resolved in four hours. During allergy consultation, detailed history revealed that since this episode she had strictly avoided crab, but had tolerated all other foods and exercise without recurrent symptoms. She denied history of atopic disease and physical exam was normal. Skin prick testing was negative for IgE-mediated response to crab, lobster, or shrimp and patient successfully passed graded oral food challenge to crab. Subsequent oral food-exercise challenge (OFEC) was scheduled. During incremental crab OFEC, the patient developed urticaria on her face. She was treated with cetirizine with resolution of urticaria and no development of other symptoms. The patient's clinical history and evaluation confirmed a diagnosis of FDEIAn to crab.

DISCUSSION: FDEIAn is a challenging diagnosis due to delayed symptoms, tolerance of food consumed without exertion, and occasional negative specific IgE testing. Augmenting factors such as NSAID use, alcohol, perimenstruation, aero-allergens, and extremes of temperature are thought to contribute to symptom development. Proposed mechanisms suggest that exercise induces physiologic changes that enhance absorption of the undigested allergen. Treatment is centered on education, avoidance of identified trigger food prior to exercise, and prescription of epinephrine auto-injector. FDEIAn is a rare but potentially fatal condition and an important consideration for primary care physicians evaluating patients who have experienced anaphylaxis without clear cause. Thorough clinical history and appropriate testing to make the diagnosis can lead to life saving preventative interventions.