An Ignored Iatrogen

Adegboyega O. Olayode, Lindsay C. Northam, Temple Brannan. Internal Medicine, Creighton University Medical Center, Omaha, NE, United States. (Control ID: 1323226)

Learning Objective 1: Recognize the clinical features of serotonin syndrome
Learning Objective 2: Manage serotonin syndrome

Case: A 53 year old male who had been previously seen at two medical facilities was transferred due to findings of fever, tachycardia, elevated white blood cell count (WBC) with bandemia and elevated creatine phosphokinase (CPK). Preliminary diagnoses from the referring facilities included sepsis and rhabdomyolysis. Prior to transfer he was started on IV fluids and given a 2mg dose of IV lorazepam for agitation. History revealed anxiety, agitation, restlessness, diaphoresis and a mild non-productive cough. On careful review of his outpatient medications, it was observed that he was simultaneously taking three serotonergic agents: bupropion, citalopram and tramadol.

On initial assessment of vital signs he was febrile, tachycardic and had a blood pressure of 104/68. Physical examination revealed resting tremors and hyperreflexia predominant in the lower extremities. Initial lab data revealed a WBC of 16.7 with 19% bands and a CPK of 1496. Investigative procedures revealed no demonstrable foci of infection. Urine drug screen was negative. Serotonergic agents were held at the time of admission and IV fluids were continued. The patient was placed on continuous cardiac monitoring and treated symptomatically with IV lorazepam for tremor and agitation. Symptom resolution, including cessation of tremor and agitation, occurred quickly in less than 24 hours and was accompanied by normalization of white blood cell count and significant decrease in CPK level.

Discussion: It has been reported that over 85% of physicians are unaware of serotonin syndrome as a clinical diagnosis. This case illustrates how easily it may be misdiagnosed and highlights the importance of taking a detailed history including the use of prescription drugs, over-the-counter medications and illicit substances along with performing a thorough physical examination. Serotonin syndrome commonly presents as a triad of mental status changes (agitation, restlessness, delirium, disorientation), autonomic dysfunction (diaphoresis, tachycardia, hyperthermia, BP instability, vomiting, diarrhea) and neurologic abnormalities (tremor, hyperreflexia, myoclonus, muscle rigidity, bilateral Babinski sign).

The diagnosis of serotonin syndrome is a clinical diagnosis. Serum serotonin concentrations don’t correlate with clinical findings and no lab tests confirm the diagnosis. Nevertheless it can be associated with some non-specific lab findings namely: leukocytosis, elevated CPK, transaminitis, metabolic acidosis and these can be used to monitor for potential complications. The Hunter Toxicity Criteria Decision Rules represent the most accurate diagnostic criteria for it. To fulfill the Hunter Criteria a patient must have taken a serotonergic agent and have ONE of the following: spontaneous clonus, inducible clonus plus agitation or diaphoresis, ocular clonus plus agitation or diaphoresis, tremor and hyperreflexia, hypertonia, temperature above 38°C plus ocular clonus or inducible clonus. In mild cases, discontinuation of inciting medications, supportive care, and sedation with benzodiazepines is generally sufficient. Moderately ill patients require more aggressive treatment of autonomic instability and possibly treatment with a serotonin antagonist (cyproheptadine). Hyperthermic patients are critically ill and often require paralysis and endotracheal intubation.
Post Transfusion Purpura Versus Heparin Induced Thrombocytopenia: A Diagnostic Dilemma

Subhraleena Das¹, Sujith Cherian¹, Wasim A. Hamarneh¹, Ehtesham Ul Haq¹ ¹ Internal Medicine, SUNY Upstate Medical University, Syracuse, NY, United States. (Control ID: 1334621)

Learning Objective 1: Recognize the complexity of sudden onset thrombocytopenia in an inpatient setting.
Learning Objective 2: Distinguish between post transfusion purpura (PTP) and heparin induced thrombocytopenia (HIT) when faced with the dilemma.

Case: A 59 year-old African American man with history of hypertension and severe peripheral vascular disease was admitted for evaluation of fever. The patient was just recently discharged 7 days prior to the admission, during which he underwent a left below knee amputation, which was complicated with severe bleeding, requiring 2 units of packed red cells (PRBC) transfusion. During the present admission, review of systems was negative. Physical examination was benign except for a temperature of 101.5 degrees F. Labs revealed anemia with a hematocrit of 25, for which 2 units of PRBC was given. Over the next day, platelet counts plummeted from 160,000 to 4,000. All heparin products were discontinued empirically. However, HIT antibody was negative. PTP was considered, given the previous history of blood transfusion, and he was started on intravenous immunoglobulin (IVIG). A positive human platelet alloantigen (HPA) antibody confirmed the diagnosis. Platelet counts subsequently improved. After a week, the patient developed right upper extremity pain and was a Doppler ultrasound confirmed the diagnosis of basilic vein thrombosis, and blood counts at the time also revealed a rapid drop in platelet count raising a suspicion for HIT. HIT antibody was positive and the diagnosis of HIT was confirmed with serotonin release assay (SRA). Patient was started on argatroban infusion thereafter and discharged subsequently on warfarin.

Discussion: PTP and HIT are two immune syndromes causing extreme thrombocytopenia, but with marked differences in pathogenesis and treatment. PTP is characterized by marked thrombocytopenia <15 Gpt/L, with platelet alloantibodies (most commonly anti HPA-1a antibody) following human platelet alloantigen incompatible blood product transfusion which may be complicated by severe hemorrhagic complications. Treatment options include mainly IVIG therapy. HIT is associated with platelet nadir between 20 to 150 Gpt/L, usually with generation of antibodies to heparin and platelet factor 4 (PF4) antigen complexes, and thrombotic complications. Treatment is discontinuation of all heparin products with initiation of danaparoid products or recombinant hirudin. Delayed onset HIT can occur as late as even 3 months after heparin products are discontinued. Our case is unique with the presence of both HPA 1a antibody and HIT antibodies, which has been reported only once before, emphasizing the importance of making the right diagnosis due to the difference in treatment of both the conditions.
“Bath Salts”: A New High, Not Found in the Hygiene Aisle

William A. Hammond11 Internal Medicine, Dartmouth-Hitchcock Medical Center, Lebanon, NH, United States. (Control ID: 1333709)

Learning Objective 1: Recognize “bath salts” as new designer drugs of abuse undetectable by current readily available screening techniques

Learning Objective 2: Recognize signs of intoxication and adverse effects of bath salt use and appropriately advise at risk patient populations of these adverse effects.

Case: A 32 year old man with chronic low back pain (LBP) and a remote history of opioid and other substance abuse presented to an Emergency Department (ED) with new onset LBP after lifting a washing machine. Despite being febrile to 38.8 degrees Celsius, he was diagnosed with mechanical injury and discharged with opioids, benzodiazepine, and prednisone. Over the ensuing 24 hours, he experienced worsening fever, systemic symptoms, and new onset left-sided facial droop. His fiancée returned him to the ED for re-evaluation. He quickly became septic with decreased mental state and was transferred to a tertiary care facility after tracheal intubation. Just prior to his dramatic decline, he admitted to recently injecting “bath salts”, a legal synthetic amphetamine. Exam on transfer revealed temperature of 38 degrees Celsius, hyperdynamic pulses, multiple skin pustules, bilateral wrist track marks, and involuntary neck flexion with flexion of the knees. Lumbar puncture returned frank pus, and subsequent magnetic resonance imaging of the spine and brain revealed a large psoas abscess with extension into the epidural space, ventriculomegaly with ventriculitis, and bilateral pontine infarcts. He was treated with intravenous antibiotics, as well as rapid extraventricular drain placement to remove purulent fluid and reduce intracranial pressure. Cultures returned with methacillin sensitive Staphylococcus aureus. After multiple surgical drainages, he made a surprising recovery to discharge to a local rehabilitation facility.

Discussion: “Bath salts” are new synthetic amphetamine-like substances that have been legally sold, prior to October 2011, in smoke shops with the intent of abuse. These products were sold legally with the label “not for human consumption” under clever labels such as “bath salts”, “plant food”, or “insect repellant”. Manufacturers circumvent the law by creating substances with slightly altered structure or side chain so that the substances do not require Drug Enforcement Agency monitoring. The most common substances are synthetic cathinones, most commonly methylenedioxypyrovalerone (MDPV), however numerous other substances have been produced and sold similarly. The substances are typically consumed orally or via inhalation, however they can also be taken intravenously, such as the patient in the clinical presentation. The effects are similar to those of amphetamines, producing euphoria, hyperactivity, and hypersexuality, however negative effects including hallucinations, paranoia, and seizures. Public media reports of emotional lability with injury to self and others, as well as suicidality among teenage and young adult users have been increasing. Currently, there are no readily available screening tests for bath salts, so the diagnosis must be made on clinical suspicion along with elimination of other substances as the causative agent in an intoxicated patient.
Bowel Beyond Borders: A Massive Paraesophageal Hernia Mimicking A Pulmonary Embolism

Michael Farbaniec1, James Lamberg1, Ethan Kuperman11 Internal Medicine, Penn State University Hershey Medical Center, Hershey, PA, United States. (Control ID: 1339107)

Learning Objective 1: Distinguish between the different types of hiatal/paraesophageal hernias and recognize less common presenting symptoms.

Learning Objective 2: Understand the management of paraesophageal hernias.

Case: An 87-year-old woman with gastroesophageal reflux disease but no history of cardiopulmonary disease presented with a fracture of her right femoral neck after a mechanical fall. The night of her admission, she became acutely short of breath, tachycardic, and developed increased oxygen requirements. Chest auscultation revealed decreased breath sounds along the left lung field but occasional bowel sounds could be heard in the bases and midlung fields bilaterally. With initial suspicion for pulmonary embolism, computed tomography of her chest was performed. No pulmonary embolism was identified but a 7x10cm type IV paraesophageal hernia was revealed, which included the entirety of her stomach and pancreas, portions of the duodenum, the splenic flexure of the colon, and associated vasculature. She remained stable after conservative treatment and was taken to surgery the following day for a right hip hemiarthroplasty. The procedure was uncomplicated and she was extubated successfully. After extubation, her oxygen requirements had diminished. Cardiothoracic surgery was consulted for management of her large paraesophageal hernia. The patient decided to decline any further surgical intervention at this time and remained asymptomatic upon discharge.

Discussion: Hiatal hernias are classified into four different types. Type I classifies sliding hernias and accounts for 95% of hiatal hernias while II, III, and IV are collectively known as paraesophageal hernias. Type II classifies herniation of the stomach through the hiatus while the gastroesophageal junction remains below the diaphragm. Type III is classified by the gastroesophageal junction herniating through the diaphragm. Type IV is classified by any other intraabdominal organs besides the stomach herniating through the diaphragm. Most common symptoms include reflux and vomiting but about 3% may develop dyspnea. Fewer than 8% of paraesophageal hernias involve viscera beyond the stomach. The small bowel and colon are often involved, but only five previous cases have reported pancreatic involvement. This patient’s unique hernia is the only documented paraesophageal hernia to contain the entirety of the pancreas as well as being one of the largest currently documented in the literature.

Management of these hernias has been controversial. A majority of past publications suggest correction of all paraesophageal hernias regardless of symptomatology. However, large database studies show a mortality rate of 1.4% in elective repair but only 1.1% developed acute symptoms requiring emergent surgery. Indications for elective surgery include failed medical management of reflux or persistent vomiting. Indications for emergent surgery include volvulus, obstruction, bleeding, and respiratory failure. In this case, the patient’s dyspnea resolved after her surgery and she remained asymptomatic. This did not make her a candidate for elective surgical repair.
Mystery Case

Asa Z. Oxner¹, Jeffrey William¹¹ Internal Medicine, Beth Israel Deaconess Medical Center, Boston, MA, United States. (Control ID: 1334637)

Case: A 63 year old man with non-Hodgkin’s lymphoma presented to his community hospital with two weeks of neck stiffness, body/joint aches, and fevers to 103F with rigor.