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A Rare Case of Immune Thrombocytopenia After Unintentional Acetaminophen Overdose
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Mentor: Jill Zabih
Program: Internal Medicine
Type: Case Report

Background: Acetaminophen is a common anti-pyretic and analgesic medication that is often included in several over-the-counter medications. Despite its easy accessibility, acetaminophen can be dangerous in large quantities leading to acute liver failure. Immune thrombocytopenic purpura is a rare, but documented, adverse effect of acetaminophen toxicity.

Case: A 40-year-old woman with no past medical history presented to the emergency room with new hematemesis and tooth pain. She was found to have a platelet count of 3000/µL without anemia or leukopenia. Peripheral blood smear confirmed thrombocytopenia without schistocytes (figure 1). Further history from the patient revealed that she had unintentionally overdosed on 4500mg of acetaminophen over the past day to treat her tooth pain. She unknowingly ingested more acetaminophen in the prior days from over-the-counter cold medications. The patient was diagnosed with drug induced thrombocytopenia and was started on high dose dexamethasone and N-acetylcysteine. She had slow improvement in her platelet count over the next two weeks and discharged home without any severe bleeding events. Patient consent was obtained to use the case for educational purposes.

Conclusion: Drug induced thrombocytopenia is a rare cause of thrombocytopenia but often leads to platelet nadir of less than 20,000/µL. Quinine, trimethoprim-sulfamethoxazole, and GPIIb/IIIa inhibitors are some of the most common drugs implicated in the development of drug induced immune thrombocytopenia, although many other medications have been documented including acetaminophen. Given the temporal relationship between acetaminophen toxicity and severe thrombocytopenia, it was suspected that acetaminophen overdose was likely the causative agent leading to drug induced thrombocytopenia.

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Spontaneous Coronary Artery Dissection (SCAD) in a Turner Syndrome Patient: The Abstract
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Mentor: Jeffery Levisman
Program: Cardiology
Type: Case Report

Background: Turner Syndrome (TS) is a rare genetic condition in females which causes numerous phenotypic cardiovascular manifestations. Spontaneous coronary artery dissection (SCAD) is a nonatherosclerotic etiology of acute coronary syndrome (ACS) which frequently affects younger women. Despite the shared demographic there is a scarcity of literature on the overlap of TS and SCAD.

Case: A 31-year-old African American female with TS, obesity, and Type-2 Diabetes presented with 8/10 chest pain over 10-12 hours. She has no cardiac history. Chest CT with contrast showed bronchitis but was negative for pulmonary embolism or aortic pathology. Troponins were negative. Electrocardiogram showed sinus rhythm with a mild j-point and ST elevation in leads V1 and V2 with poor r-wave progression across the precordial leads. The patient continued experiencing chest pressure, now rated 4/10. Her laboratory values were significant for a mild leukocytosis but were otherwise normal. TEE showed EF of 30-35% with anteroseptal, apex and distal inferoseptum segmental wall motion abnormalities, ruling out Takusubos. Interventional cardiology then performed left heart catheterization. Mid left anterior descending artery SCAD was diagnosed, confirmed by intravascular ultrasound pullback recording. No coronary artery disease was noted. No percutaneous intervention was performed. Twenty-four-hour ICU observation and medical management