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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.

https://doi.org/10.32873/unmc.dc.gmerj.4.1.033

Spontaneous Coronary Artery Dissection (SCAD) in a Turner Syndrome Patient: The Abstract
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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 inferoseptal 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
with ticagrelor, aspirin, atorvastatin and metoprolol tartrate daily were initiated, and was subsequently discharged with follow-up catheterization. Patient consent was obtained to use this case for educational purposes.

**Conclusion:** SCAD is a rare cause of ACS in young women. TS predisposes patients to a variety of arteriopathies and may include SCAD. SCAD should be considered in a TS patient presenting with ACS. ■

https://doi.org/10.32873/unmc.dc.gmerj.4.1.034

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**Minocycline Induced Pauci-Immune Glomerulonephritis**

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**Mentor:** Ketki Tendulkar

**Program:** Nephrology

**Type:** Case Report

**Background:** Minocycline has been implicated in various autoimmune syndromes. Here we present a case of likely minocycline induced pauci-immune glomerulonephritis.

**Case:** A 74 year old male presented to rheumatology clinic for follow-up of his difficult to manage rheumatoid arthritis (RA). He had previous adverse effects and contraindications to several other disease-modifying anti-rheumatic drugs and hence was started on minocycline a year and a half ago. At that visit, he had an acute kidney injury with creatinine increase to 1.9 mg/dL (baseline 1.0 mg/dL), and urinalysis showed 53 RBCs, 8 WBCs, and 100 mg/dL protein. Urine protein/creatinine ratio (UPC) was 2.46. Complements were normal. ANA, hepatitis B and C were negative. Proteinase-3 antibodies and c-ANCA were negative. However, p-ANCA was positive at 1:640 with myeloperoxidase (MPO) antibody positive at 8 antibody index. Following these results, minocycline was stopped and renal biopsy was scheduled. Renal histology showed pauci-immune necrotizing glomerulonephritis. He was initiated on a monthly prednisone taper and received two doses of rituximab 1 g two weeks apart. With monthly follow-up his creatinine improved to 1.7 mg/dL and UPC had decreased to 1.97. He was continued on steroid taper and will receive further rituximab doses in the next few months.

Minocycline has been linked to cases of autoimmune hepatitis, drug-induced lupus, serum sickness, and vasculitis. One possible explanation is that minocycline is oxidized by MPO and its reactive metabolites then alter MPO, stimulating p-ANCA production. Most cases of minocycline-induced syndromes were successfully treated with cessation of the drug and a steroid taper. Minocycline induced autoimmune syndromes are important to recognize as treatment relies heavily on discontinuation of the drug. To our knowledge, this is the first reported case of minocycline-induced pauci-immune glomerulonephritis in a patient with RA.

**Conclusion:** Minocycline can cause p-ANCA pauci-immune glomerulonephritis and must be considered as treatment relies heavily on discontinuation of the drug. ■

https://doi.org/10.32873/unmc.dc.gmerj.4.1.036

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**Prisma MRI and MEG Leading to a Successful Frontal Lesionectomy in a Medically Refractory Epilepsy Patient**

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**Mentor:** Aditya Vuppala

**Program:** Neurological Sciences & Neurosurgery

**Type:** Case Report

**Background:** A well-defined focal lesion on brain imaging in medically refractory frontal lobe epilepsy (MRFLE) patients who undergo epilepsy surgery is associated with a better chance of achieving seizure freedom compared to patients who have no identifiable lesion. High resolution Prisma MRI can uncover lesions that cannot be identified with conventional 3T MRI, but the utility of this technology for presurgical evaluation in such patients has yet to be formally evaluated.

**Case:** A 22-year-old male with MRFLE underwent presurgical evaluation including 3T MRI which revealed a left mesial frontal lesion and scalp EEG that captured seizures emanating from the left frontal region. Magnetoencephalography (MEG) demonstrated multiple left anterior frontal dipoles that were significantly more anterior to the 3T MRI was performed. Using these data, an intracranial stereo EEG (sEEG) was performed which captured multiple seizures arising from the left anterior frontal region. Prisma MRI, which later became available, revealed a lesion extending from the left anterior frontal cortex to frontal horn of the lateral ventricle not previously identified on 3T MRI. Retrospectively, this lesion was concordant with MEG dipoles and seizure onset zone identified on sEEG. The lesionectomy performed using these data has resulted in seizure freedom for over 10 months to date. Patient consent was obtained to use this case for educational purposes.

**Conclusion:** This case describes the successful evaluation and surgical treatment of a patient with MRFLE following the inclusion of Prisma MRI in the presurgical assessment and planning. We are identifying similar cases to further study the utility of this imaging modality in presurgical evaluation for MRFLE. ■

https://doi.org/10.32873/unmc.dc.gmerj.4.1.036