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### Metronidazole Neurotoxicity--Not Your Usual Suspect

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## ACP Michigan Chapter Scientific Meeting 2021

Oral# 13

Category: Clinical Vignette

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### **Metronidazole Neurotoxicity--Not Your Usual Suspect**

An 85-year-old female with past medical history notable for chronic lymphocytic leukemia and Stage III Merckel cell carcinoma presented to our hospital for evaluation of generalized weakness of a few weeks duration. Notably, she had a recent hospital admission for E. coli bacteremia in the setting of hepatic abscesses that underwent ultrasound guided drainage. She was discharged to inpatient rehabilitation, and ultimately home, with a planned six-week course of ceftriaxone and metronidazole, in addition to oral vancomycin for C. difficile infection.

Upon readmission, she was fatigued and had progressive lower extremity weakness. She denied any bowel or bladder incontinence or saddle anesthesia. She was admitted to the Internal Medicine service with Oncology and Neurology consultations. Prior antibiotics were continued.

Noncontrast CT head showed no hemorrhage. MRI of the lumbar spine showed degenerative changes, without intradural enhancement. Due to the lack of a conclusive diagnosis thus far, EMG was recommended, which showed signs of lumbosacral neuropathy and no evidence of Guillain Barre Syndrome.

She then developed facial twitching and concern for seizure-like activity. Repeat CT head was negative, and she was started on levetiracetam. MRI brain showed abnormal signal intensity with diffusion restriction of the lenticular nucleus bilaterally and symmetrically, which can be associated with transient edema from metronidazole toxicity. MRA was unremarkable. Metronidazole was discontinued and the patient did not have any further seizure activity.

This case highlights an interesting, and uncommon toxicity of metronidazole. The frequency of metronidazole induced encephalopathy (MIE) is unknown. Peripheral neuropathy is common, but the entity can also result in dizziness, ataxia, confusion, encephalopathy and seizures. It can be visualized on MRI and is characterized by bilateral, symmetric T2 hyperintense lesions that resolve after cessation of the drug. High clinical suspicion must be present to warrant neuroimaging to secure the diagnosis.