Case Report

**Metronidazole induced Reversible Cerebellar Ataxia in a patient of multiple liver abscesses: a case report**

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**Abstract**

Metronidazole is a frequently used antibiotic for the treatment of anaerobic bacterial and protozoal infections. The drug has few adverse reactions, most commonly nausea, dry mouth, vomiting, and diarrhea. Neurologic toxicity is rare and has included peripheral neuropathy, headache, dizziness, syncope, vertigo, and confusion. Cerebellar toxicity is a reported, although very unusual, manifestation of metronidazole therapy. We are reporting a case of this very rare side effect, i.e. Reversible Cerebellar toxicity, in a patient who was on prolonged metronidazole therapy for multiple liver abscesses.

**Keywords:** Metronidazole, Cerebellar Ataxia, infections

1. Introduction

Cerebellar toxicity is a rare adverse event in patients treated with metronidazole. While proposed mechanism was high cumulative dose in most patients (25-1080 grams), our patient in spite of taking about 75 grams had normal serum Metronidazole levels. MRI has proven to be the most important diagnostic tool.

2. Case report

A 50-year old male was admitted in surgery in January 2013 with complaints of fever and right hypochondriac pain, diagnosed as a case of multiple liver abscesses and was managed initially by intravenous ciprofloxacin and metronidazole given for 7 days along with drainage of abscess. He improved and was discharged on oral metronidazole 800mg thrice a day for 7 days. The patient did not return for follow up, but continued medication for next 20 days. In February 2013, the patient presented to medicine outpatient with 3 days history of slurring of speech, incoordination, progressive unsteadiness of gait and subsequent inability to walk. On neurologic examination, he was alert and oriented to person, place, and time. His speech was dysarthric. Cranial nerve examination revealed horizontal nystagmus of 2-3 beats on lateral gaze; there was no vertical nystagmus. Pupils were equally reactive to light. Cerebellar examination revealed that heel-to-shin maneuvers, finger-to-nose testing were abnormal. The patient’s muscle strength was normal and tone in all four limbs was decreased. Deep tendon reflexes were physiologically active and equal. His stance was wide-based; he could not stand with his feet together, he felt unsteady and was unable to walk without support. His sensory examination was normal. The Romberg’s sign could not be elicited because of unsteadiness. MRI Brain revealed abnormal T2 as well as FLAIR hyper intensities in dentate nuclei and deep cerebellar parenchyma as well as splenium of corpus callosum, no mass, hemorrhage or herniation was seen. The serum Metronidazole levels were found to be in normal range.

After discontinuation of metronidazole therapy, the patient started regaining ambulatory function. He was able to walk with support in 4 days and without support in 10 days. A follow up MRI after 8 weeks was found absolutely normal with resolution of hyper intensities of dentate nuclei.
Axial Flair Images reveal Bilateral Symmetric semilunar hyper intensities involving Dentate Nuclei and Deep Cerebellar Parenchyma which also reveals restricted diffusion.

Figure 2: Follow Up Image after 8 weeks

Subsequent image revealed complete resolution of the signal abnormalities.

2. Discussion

Cerebellar toxicity is a rare adverse event in patients treated with metronidazole. While proposed mechanism was high cumulative dose in most patients (25-1080 grams), our patient inspite of taking about 75 grams had normal serum Metronidazole levels. MRI has proven to be the most important diagnostic tool. All the patients show improvement clinically as well as on radioimaging completely after discontinuation of metronidazole therapy. The exact mechanism by which Metronidazole causes reversible cerebellar ataxia and dentate nuclei changes is unclear. Axonal swelling due to localized vasogenic edema, rather ischemia or demyelination, is a possible mechanism. High doses of metronidazole in rats have also been shown to induce lesions in the cerebellum; alterations were qualitatively and topographically comparable to central nervous system lesions induced by thiamine deficiency in rats and in Wernicke’s encephalopathy in humans. Studies in both dogs and mice have found Purkinje cell lesions and carbon-labeled metronidazole detected in the cerebellum respectively. In our patient, as the serum metronidazole level was absolutely normal, a possible mechanism may be idiosyncratic reaction to metronidazole. In summary, Cerebellar toxicity should be considered in any patient who presents with ataxia and/or dysarthria and is receiving prolonged therapy with metronidazole. MRI should be performed for definitive diagnosis and metronidazole should be immediately discontinued. Further studies are needed to define the pathogenesis of this unusual event.

References