

Metronidazole-induced Cerebellar Ataxia: A Rare Presentation

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ABSTRACT

Metronidazole is an antibiotic and an antiprotozoal drug very commonly used in our day-to-day practice. It is generally well tolerated and common vomiting, abdominal cramps, and metallic taste. We are presenting a rare case of metronidazole-induced cerebellar ataxia in a patient who received the drug for a relatively shorter duration. The neurological features usually occur when the drug dose exceeds 2 gm/day for a prolonged period. Peripheral neuropathy, dizziness, encephalopathy, seizures, and optic neuropathy can be seen. Cerebellar ataxia is a rare and serious side effect of this drug.

Keywords: Ataxia, Cerebellum, Dentate nucleus, Metronidazole.

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INTRODUCTION

Metronidazole is a 5-nitroimidazole that is *in vitro* activity against a wide range of anaerobic protozoan parasites and anaerobic bacteria.¹ It is clinically effective against trichomoniasis, amebiasis, and giardiasis. It exhibits antibacterial activity against all anaerobic cocci, including the genus *bacteroides* and anaerobic gram-negative bacilli.

Common side effects include headache, nausea, dry mouth, and metallic taste. Occasionally, vomiting, diarrhea, and abdominal pain occur. Dysuria, cystitis, and pelvic pressure have also been reported. Vertigo, dizziness, very rarely, encephalopathy, convulsions, incoordination, and ataxia are neurotoxic effects that justify drug discontinuation.²

Neurological features usually appear when the drug is used for a long time at doses above 2 gm/day. In cases of cerebellar ataxia due to metronidazole, the dosage usually ranges from 25 gm to 1080 gm, and the therapeutic duration ranges from 5 to 730 days.³ Here, we present an interesting case of cerebellar ataxia with a far lower dose of metronidazole which is 6 gm.

CASE DESCRIPTION

A 24-year-old male with no prior comorbidities presented with sudden onset speech abnormality and difficulty in walking with pain in bilateral lower limbs for 2 days. There was a history of liver abscess and for which he has been taking treatment in the form of metronidazole for the last 45 days. There was no history of similar complaints in the past. There was no history of substance or alcohol abuse. On examination, the patient was conscious and well-oriented. The language was scanning speech. There was locomotor ataxia. The deep tendon reflexes were normal.

Brain magnetic resonance imaging (MRI) shows changes in signal intensity, with T2-weighted-fluid-attenuated inversion recovery (T2/FLAIR) hyperintensities seen in the bilateral caudate nuclei of the cerebellum and the inferior colliculus of the dorsal pons correlating with metronidazole toxicity (Fig. 1).

The patient was treated conservatively and improved over the next 5 days. At the time of discharge, his speech was normal. The patient was followed up 20 days later and found that all of his neurological defects had recovered.

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DISCUSSION

Our patient took metronidazole over 45 days and showed neurotoxic symptoms such as cerebellar ataxia. Brain MRI showed lesions characteristic of metronidazole toxicity. The literature suggests that the dose and duration of metronidazole, which induces neurotoxicity in previously reported cases, were much higher than in the current case.

Ahmed et al. reported a case of nausea, vomiting, dizziness, confusion, ataxia, and peripheral neuropathy while taking about 35 gm of metronidazole. MRI showed symmetrical signal abnormalities in the cerebellum.⁴

Chandak et al. reported a 45-year-old case who received about 62 gm of metronidazole for 26 days for a liver abscess with cerebellar symptoms.⁵

The exact mechanism is unknown, but metronidazole and its toxic metabolites are said to bind to ribonucleic acid in sensitive human neurons, causing reversible axonal swelling.⁶ Other theories, such as interstitial edema, ischemia, and Purkinje cell damage, have also been proposed. There are no specific tests to confirm the toxicity of metronidazole. Diagnosis is based on the

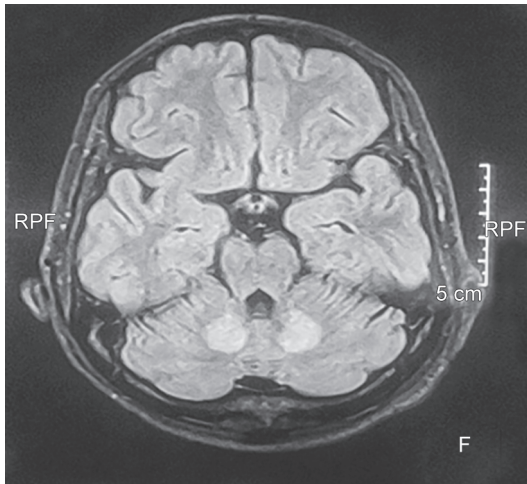


Fig. 1: T2/FLAIR hyperintensities in the bilateral dentate nucleus

history of metronidazole use and the exclusion of other causes of cerebellar ataxia. Brain MRI shows hyperintensities in the bilateral caudate nuclei of the cerebellum and the inferior colliculus of the dorsal pons.

The diagnosis is confirmed by a history of metronidazole use, MRI findings, and reversibility after drug discontinuation. The most common mimics are demyelinating diseases, infections, and Wernicke encephalopathy.

CONCLUSION

Patients taking metronidazole should be watched for cerebellar ataxia, seizures, paresthesia, and neurotoxicity. On the development of such side effects the problematic drug should be discontinued. A complete reversal is usually observed.

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