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Metronidazole-Induced Encephalopathy Mimicking Hepatic Encephalopathy in a Patient With Residual Liver Abscess: A Case Report.

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ABSTRACT

Background

Metronidazole is commonly used for anaerobic and protozoal infections, including liver abscess. Though it's a relatively safe drug, it can rarely cause neurotoxicity leading to metronidazole-induced encephalopathy (MIE). This entity is clinically important because it may present with acute or subacute altered sensorium and may closely mimic hepatic encephalopathy, This is particularly more likely to occur in patients with underlying liver disease or in cases with intra-abdominal infection.

Case Report

We report a 45 years old man with a recent liver abscess which was managed with percutaneous drainage and prolonged metronidazole therapy. Patient developed progressive anorexia followed by confusion and irritability without focal neurological deficits. Given the clinical context, hepatic encephalopathy, central nervous system infection, metabolic causes and drug-induced toxicity were considered as differential diagnosis and a work-up was initiated. Magnetic Resonance Imaging (MRI) demonstrated a characteristic bilateral symmetric pattern involving cerebral white matter and the corpus callosum with additional symmetric signal changes in the cerebellar dentate nuclei and brainstem-related structures. These imaging findings were consistent with toxic encephalopathy highly suggestive of MIE. Metronidazole was promptly discontinued and supportive care was provided. concurrent measures for possible hepatic encephalopathy were continued during the acute phase. The patient improved steadily after withdrawal of metronidazole and was discharged with complete clinical recovery.

Conclusion

This case underscores the need for heightened awareness of MIE in patients receiving prolonged metronidazole particularly when co-existing hepatic comorbidity creates diagnostic ambiguity. Recognition of the typical symmetric MRI pattern and early cessation of the offending agent are key to preventing unnecessary investigations and achieving favorable neurological outcomes.

Keywords: Liver Abscess, Metronidazole-induced Encephalopathy, Magnetic Resonance Imaging, Dentate Nuclei.

INTRODUCTION

Metronidazole is a nitroimidazole antimicrobial which is known to be effective against anaerobic bacteria and protozoa.[1] It is commonly used in cases of intra-abdominal infections and liver abscess. Common adverse effects are usually related gastrointestinal symptoms. Neurotoxicity is uncommon but clinically important because it may present abruptly and mimic other causes of encephalopathy particulalry in patients with concurrent hepatic illness.[2]

Metronidazole-induced encephalopathy (MIE) has distinctive neuroimaging features. On magnetic resonance imaging it mainly manifests as bilateral symmetric T2/FLAIR hyperintense lesions involving the cerebellar dentate nuclei and brainstem structures, with additional involvement of the corpus callosum and cerebral white matter.[3] Early recognition and withdrawal of metronidazole are central to management and are associated with improvement in most patients.

CASE REPORT

A 45-year-old man, a farmer by occupation, had been diagnosed with liver abscess approximately one to two months prior to this admission. He had a history of chronic alcohol use (reported intermittent use over approximately 20 years) and long-term tobacco exposure (smoking for about 20 years and chewing tobacco for about 15 years). For hepatic abscess he had undergone percutaneous pigtail drainage and was treated with antibiotics including metronidazole for a prolonged period.

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He subsequently developed anorexia for around 20 days and presented to the hospital with altered sensorium. On initial evaluation, he was conscious but disoriented and irritable, with no clear focal neurological deficit. Laboratory investigations showed microcytic anemia with hemoglobin 11.6 g/dL and low mean corpuscular volume (71 fL), with total leukocyte count 9,450/µL and platelets 279,000/µL. Electrolytes demonstrated mild hyponatremia (serum sodium 136 mmol/L) and hypokalemia (serum potassium 3.0 mmol/L), with chloride 104 mmol/L. Liver biochemistry was not markedly deranged (total bilirubin 0.8 mg/dL, direct bilirubin 0.1 mg/dL, alanine aminotransferase 25.9 U/L, aspartate aminotransferase 27.2 U/L, alkaline phosphatase 105.07 U/L), with serum albumin 3.6 g/dL and globulin 4.6 g/dL (A/G ratio 0.78).

Abdominal and pelvic ultrasonography demonstrated mild hepatomegaly (liver size approximately 155 mm). Two residual liver abscess cavities were seen on ultrasound. A right lobe lesion in segment VIII measuring approximately 59 × 66 × 68 mm (estimated volume was found to be approximately 133 cc) with the tip of a pigtail catheter within it and a left lobe lesion measuring approximately 37 × 44 × 37 mm (estimated volume of 32 cc), both described as minimally liquefied. Mild right-sided pleural effusion was also noted.

Given the altered sensorium in the context of liver abscess and ongoing antimicrobial therapy, differentials considered included hepatic encephalopathy, infectious meningo-encephalitis, metabolic encephalopathy and drug-induced neurotoxicity. A lumbar puncture was performed and cerebrospinal fluid was sent for analysis.

MRI of the brain (plain and contrast) revealed bilateral symmetric confluent areas showing restricted diffusion with corresponding low apparent diffusion coefficient values and T2/FLAIR hyperintensity involving the bilateral frontoparietal-occipital deep white matter and the entire corpus callosum. Additional bilateral symmetric T2/FLAIR hyperintense signal was seen in the diffusion-weighted images without corresponding low ADC (suggestive of T2 shine-through) involving the bilateral dentate nuclei, bilateral vestibular nuclei and bilateral inferior colliculi. The remainder of the cerebral parenchyma was described as normal with no mass effect, hemorrhage or midline shift. These imaging features were highly suggestive of toxic encephalopathy, most likely metronidazole-induced encephalopathy [Fig 1].

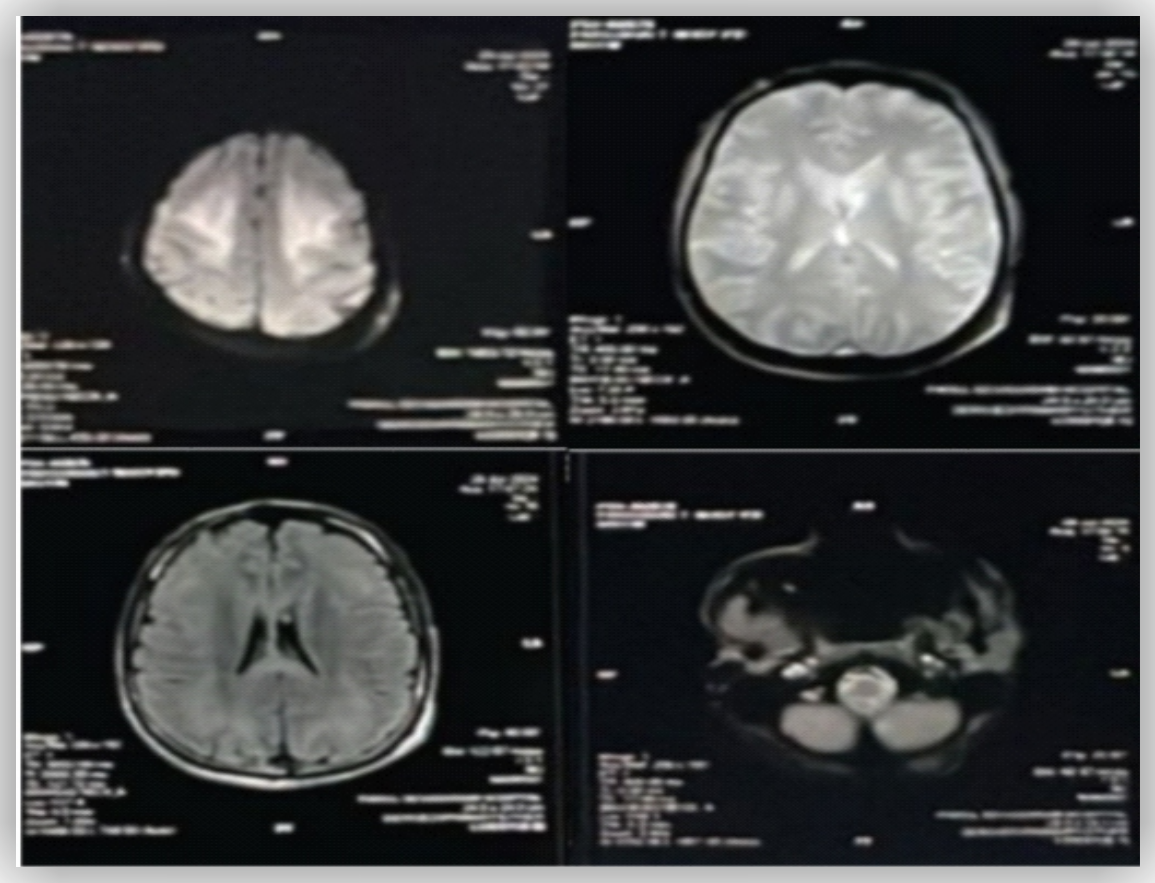

Image 1: MRI brain (DWI/ADC and T2/FLAIR) demonstrating bilateral, symmetrical confluent diffusion restriction with low ADC and T2/FLAIR hyperintensity involving the frontoparietal-occipital deep white matter and entire corpus callosum, with additional symmetric T2/FLAIR hyperintensity (T2 shine-through) in the dentate nuclei, vestibular nuclei, and inferior colliculi.

On the basis of imaging finding the diagnosis of metronidazole induced neurotoxicity was made and metronidazole was discontinued. The patient received supportive care with close neurological monitoring, correction of electrolyte abnormalities and ongoing management of residual liver abscess with alternative antimicrobial strategy. Given the elevated ammonia and overlap with hepatic encephalopathy, concurrent supportive measures directed at hepatic encephalopathy were also continued during the acute phase. Over subsequent days his sensorium improved and at discharge he was documented to have normal sensorium and stable vitals.

DISCUSSION

Metronidazole is widely used for anaerobic and protozoal infections (including intra-abdominal infections such as liver abscess) and is also sometimes used in hepatic encephalopathy; however, central neurotoxicity remains under-recognized and can clinically mimic hepatic encephalopathy, sepsis-associated encephalopathy, or metabolic causes. The mechanism of metronidazole neurotoxicity is not fully established and proposed explanations include reversible myelin edema, axonal swelling and metabolic or mitochondrial vulnerability within selectively susceptible neural tracts. While these hypotheses remain incompletely proven the consistent anatomic predilection on MRI across reports supports a toxic-metabolic injury pattern rather than vascular pathology.

In our patient the history of prolonged metronidazole exposure for liver abscess followed by onset of altered sensorium along with the characteristic MRI pattern made metronidazole-induced encephalopathy a key diagnostic consideration over alternative etiologies.[4]

Clinically, metronidazole-induced encephalopathy most often manifests with cerebellar signs (dysarthria, gait ataxia, and limb incoordination). However other neurological manifestations such as altered mental status, seizures, vertigo have also been reported. Importantly metronidazole toxicity has been described across a wide range of cumulative doses and durations. Although many patients have substantial cumulative exposure, clinically significant encephalopathy has also occurred at comparatively low total doses. This indicates that there is no strict dose threshold for occurrence of MIE.[5]

Neuroimaging, particularly MRI, plays an important role in the diagnosis because the radiological features are often distinctive and can help separate drug toxicity from other diagnoses. Classic findings are bilateral, symmetric T2/FLAIR hyperintensities which most frequently involve the cerebellar dentate nuclei. The other commonly affected regions include the splenium of the corpus callosum and brainstem structures including midbrain, pons and medulla. Kim E et al described the typical distribution and imaging behavior of metronidazole-related lesions and recommended that symmetric involvement of these predilection sites should immediately prompt review of nitroimidazole exposure.[6] In our case, the MRI impression favoured toxic encephalopathy related to metronidazole which was also consistent with the clinical timeline.

Management is primarily prompt discontinuation of metronidazole with supportive care and aggressive evaluation and treatment of coexisting contributors such as hepatic encephalopathy, electrolyte imbalances and ongoing infection. Most published cases demonstrate substantial clinical improvement within days to two weeks after stopping the drug that is reported to result in partial or complete radiologic resolution on follow-up imaging. The reversibility of this entity has been recognized since early descriptions such as the report by Ahmed A et al which emphasized reversible MRI abnormalities with metronidazole-induced encephalopathy.[7] Subsequent reports and series have broadened the spectrum including 5 nitroimidazole –associated encephalo-neuropathy described by Chacko J et al.[8] Similarly case reports such as those by Agarwal et al[9] and more recent case-based discussions including Chaudhari et al[10] collectively underscores the fact that this diagnosis should remain on the differential whenever compatible MRI findings appear in a patient receiving metronidazole.

In patients with liver dysfunction or systemic illness the diagnosis can be challenging because hepatic encephalopathy and infection-related encephalopathy may coexist in these cases. However, a compatible MRI pattern in the appropriate exposure window should be treated as strong evidence for drug toxicity. Given the potential for rapid reversibility with withdrawal clinicians should have a high index of suspicion and low threshold to stop metronidazole when new neurological symptoms occur.

CONCLUSION

Metronidazole-induced encephalopathy should be considered in any patient receiving prolonged metronidazole therapy who develops acute or subacute altered mental status or cerebellar signs. Typical bilateral symmetric MRI abnormalities involving the dentate nuclei, brainstem structures, corpus callosum and cerebral white matter can strongly support the diagnosis. Prompt withdrawal of metronidazole and supportive management are key and may result in full clinical recovery.

Conflict Of Interest : None

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REFERENCES

1. Brogden RN, Heel RC, Speight TM, Avery GS. Metronidazole in anaerobic infections: a review of its activity, pharmacokinetics and therapeutic use. Drugs. 1978 Nov;16(5):387-417. doi: 10.2165/00003495-197816050-00002. PMID: 363399.
2. Quickfall D, Daneman N, Dmytriw AA, Juurlink DN. Metronidazole-induced neurotoxicity. CMAJ. 2021 Oct 25;193(42):E1630. doi: 10.1503/cmaj.201671. PMID: 34697097; PMCID: PMC8562982.
3. 5. Patel L, Batchala P, Almardawi R, Morales R, Raghavan P. Acute metronidazole-induced neurotoxicity: an update on MRI findings. Clin Radiol. 2020 Mar;75(3):202-208. doi: 10.1016/j.crad.2019.11.002. Epub 2019 Dec 16. PMID: 31858989.
4. Villagrán Sancho D, Villar Rodríguez C, Luque Ambrosiani AC, Baena-Palomino P. Metronidazole-induced toxicity of the central and peripheral nervous system. BMJ Case Rep. 2025 Jun 26;18(6):e264904. doi: 10.1136/bcr-2025-264904. PMID: 40571418.
5. Lala VG, Bobat B, Haagensen M, Kathan P, Mahomed A. Metronidazole-induced encephalopathy. SA J Radiol. 2021 Mar 18;25(1):2016. doi: 10.4102/sajr.v25i1.2016. PMID: 33824746; PMCID: PMC8008077.
6. Kim E, Na DG, Kim EY, et al. MR imaging of metronidazole-induced encephalopathy: Lesion distribution and diffusion-weighted imaging findings. Am J Neuroradiol. 2007. October 1;28(9):1652–1658. 10.3174/ajnr.A0655
7. Ahmed A, Loes DJ, Bressler EL. Reversible magnetic resonance imaging findings in metronidazole-induced encephalopathy. Neurology. 1995 Mar;45(3 Pt 1):588-9. doi: 10.1212/wnl.45.3.588. PMID: 7898724.
8. Chacko J, Pramod K, Sinha S, Saini J, Mahadevan A, Bharath RD, Bindu PS, Yasha TC, Taly AB. Clinical, neuroimaging and pathological features of 5-nitroimidazole-induced encephalo-neuropathy in two patients: Insights into possible pathogenesis. Neurol India. 2011 Sep-Oct;59(5):743-7. doi: 10.4103/0028-3886.86552. PMID: 22019662.

9. Agarwal A, Kanekar S, Sabat S, et al. Metronidazole-induced cerebellar toxicity. Neurol Int. 2016;8(1):4–6. 10.4081/ni.2016.6365

10. Chaudhari DM, Renjen PN, Hasan U, Goyal N, Ahmad K. Metronidazole-Induced Encephalopathy (MIE). Cureus. 2022 Dec 30;14(12):e33133. doi: 10.7759/cureus.33133. PMID: 36721588; PMCID: PMC9884464.

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Author Contribution :
UP: Contributed to patient management, data collection, and manuscript drafting. **MM**: Was involved in data compilation and literature review, and edited the manuscript. **PP**: Performed data interpretation and critical revision of the manuscript. **RP**: Supervised the study and approved the final manuscript for submission.

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