Case 18543

Eurorad

“Clearing the haze”: A case report of acute hepatic encephalopathy

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ISSN: 1563-4086
Section: Neuroradiology
Area of Interest: Abdomen Paediatric
Procedure: Diagnostic procedure
Imaging Technique: CT
Special Focus: Metastases Obstetrics Case Type:
Clinical Case
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Patient: 62 years, female

Clinical History:

A 62-year-old female patient, who is a known case of non-alcoholic steatohepatitis (NASH) related cirrhosis of the liver, presented in the emergency department with a sudden dip in sensorium levels and was found in a gasping state in the early morning. She had hypothyroidism as her comorbidity. MRI Brain was requested to rule out hypoxic brain injury or posterior circulation stroke.

Imaging Findings:

Axial FLAIR (Figure 1a) and T2WI (Figure 1b) show symmetrical gyriform hyperintensities predominantly involving bilateral cingulate gyri, insular cortices and bilateral fronto-temporo-parietal lobes with sparing of bilateral occipital lobes. There is a gyriform restriction of diffusion seen involving these areas on DWI (Figure 2a) and corresponding ADC maps (Figure 2b). Relative sparing of bilateral peri-rolandic regions is also seen as shown on DWI (Figure 3a) and ADC (Figure 3b) images. Axial T1WI (Figures 4a and 4b) shows hyperintense signal intensity involving bilateral globus pallidi and anterior aspects of the bilateral cerebral peduncles of the midbrain, which is suggestive of manganese deposition.

Discussion:

Background

Hepatic encephalopathy (HE) can be because of acute fulminant hepatic failure, or as a chronic process in patients with hepatocellular dysfunction that leads to portosystemic shunting. Acute HE can be rapidly fatal, whereas chronic HE is usually a more indolent process causing neuropsychiatric symptoms [1,2]. Cerebral oedema caused by increased intracellular osmolytes is postulated as the pathophysiologic basis underlying both the acute and chronic forms of HE [3].

Clinical Perspective

The most common clinical presentation in these patients is confusion or coma precipitated by gastrointestinal bleeding, acute superimposed hepatitis, or concomitant infection in a previously asymptomatic patient with cirrhosis [4]. The neurologic manifestations are mainly due to shunting of blood arising from the portal venous bed into the systemic circulation, which are reversible once the liver function abnormality or precipitating factor has been corrected.

Imaging Perspective

Patients with symptoms of acute hepatic encephalopathy are evaluated by CT or MR imaging to exclude haemorrhage or infarction.

Conventional MRI may reveal hyperintense signal in the globus pallidus, subthalamic region and midbrain on T1-weighted images, and may also show diffuse cortical oedema and hyperintensity on T2-weighted images, with sparing of the peri-rolandic and occipital regions. Both FLAIR and DWI severity have strong correlations with plasma ammonia levels.

The metabolic abnormalities correlate with clinical severity and revert after treatment. A combination of characteristic MR imaging features and an MR imaging scoring system in patients with an elevated PAL helps in predicting the clinical severity.

In patients with acute hepatic encephalopathy, MR imaging can show characteristic regions of involvement on FLAIR or DWI, determine the reversibility of lesions, and whether the extent of MR imaging involvement on FLAIR or DWI (MR imaging severity) correlates with either the PAL, the initial clinical severity (West Haven criteria), or the clinical outcome severity [5].

Our patient had symmetrical T2W and FLAIR hyperintensities with diffusion restriction involving bilateral cingulate gyri, bilateral temporo-parietal lobes and insular cortices with sparing of the peri-rolandic region and bilateral occipital lobes. Symmetrical T1 hyperintensity was seen involving globus pallidi and midbrain, which could be attributed to manganese deposition. Blood ammonia levels were elevated.

Outcome

The patient was managed as a case of encephalopathy due to hyperammonemia. However, she had a poor prognosis, had a cardiac arrest, and succumbed to her illness.

Imaging can help us to rule out acute events such as haemorrhage or infarction and guide us to narrow our differentials in patients with encephalopathy.

Take Home Message

Acute hepatic encephalopathy should be considered in the setting of cirrhosis of liver presenting with altered sensorium.

All patient data have been completely anonymised throughout the entire manuscript and related files.

Differential Diagnosis List: Hypoxic brain insult, Hypoglycemic encephalopathy, Acute hepatic encephalopathy, Carbon monoxide poisoning, Sporadic Creutzfeldt–Jakob disease (CJD)

Final Diagnosis: Acute hepatic encephalopathy

References:

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Ferenci P, Lockwood A, Mullen K, Tarter R, Weissenborn K, Blei AT (2002) Hepatic encephalopathy--definition, nomenclature, diagnosis, and quantification: final report of the working party at the 11th World Congresses of Gastroenterology, Vienna, 1998. Hepatology 35(3):716-21. doi: 10.1053/jhep.2002.31250. (PMID:11870389)
Häussinger D, Kircheis G, Fischer R, Schliess F, vom Dahl S (2000) Hepatic encephalopathy in chronic liver disease: a clinical manifestation of astrocyte swelling and low-grade cerebral edema? J Hepatol 32(6):1035-8. doi: 10.1016/s0168-8278(00)80110-5. (PMID: 10898326)
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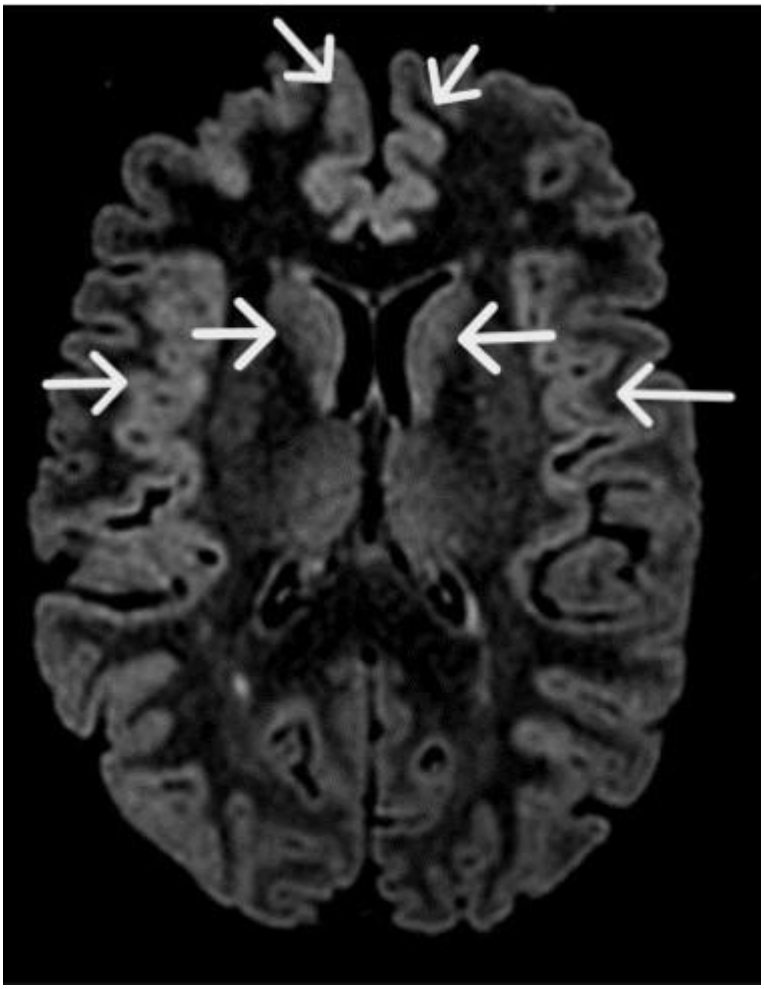
McKinney AM, Lohman BD, Sarikaya B, Uhlmann E, Spanbauer J, Singewald T, Brace JR (2010) Acute hepatic encephalopathy: diffusion-weighted and fluid-attenuated inversion recovery findings, and correlation with plasma ammonia level and clinical outcome. AJNR Am J Neuroradiol 31(8):1471-9. doi: 10.3174/ajnr.A2112. (PMID: 20448015)

Figure 1

a



Description: Plain MRI Brain, FLAIR axial (1a) and T2WI axial (1b) show symmetrical gyriform hyperintensities involving predominantly bilateral cingulate gyri, insular cortices and bilateral fronto-temporo-parietal lobes and central grey matter nuclei. **Origin:** © Department of Radiodiagnosis, Command Hospital (Western Command), Panchkula, Haryana, India

b

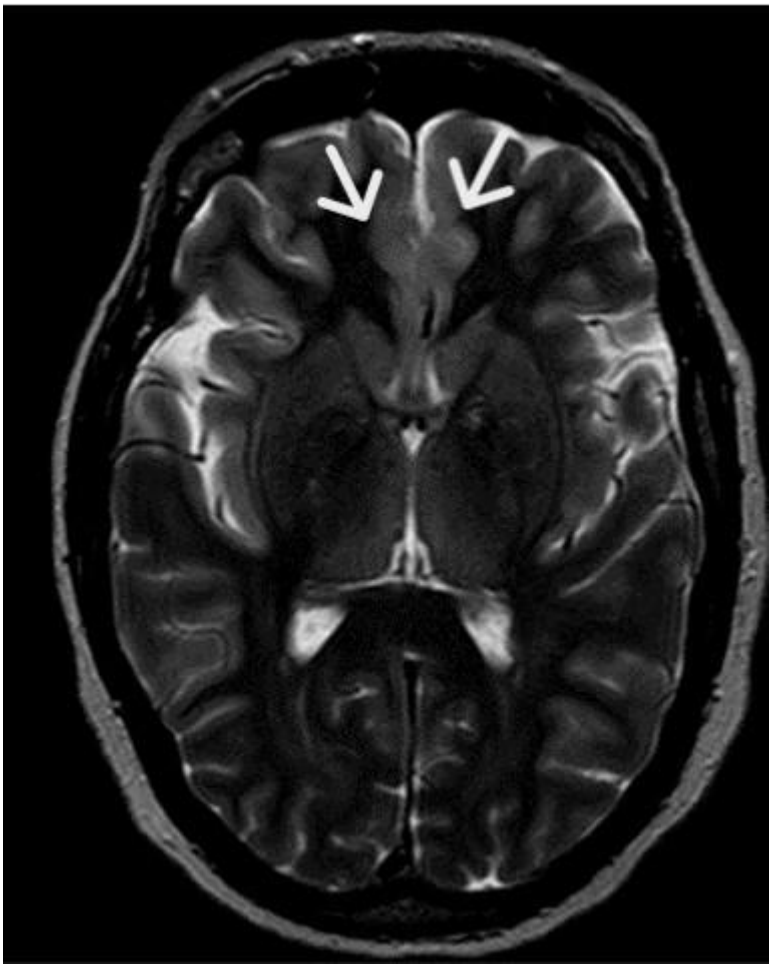


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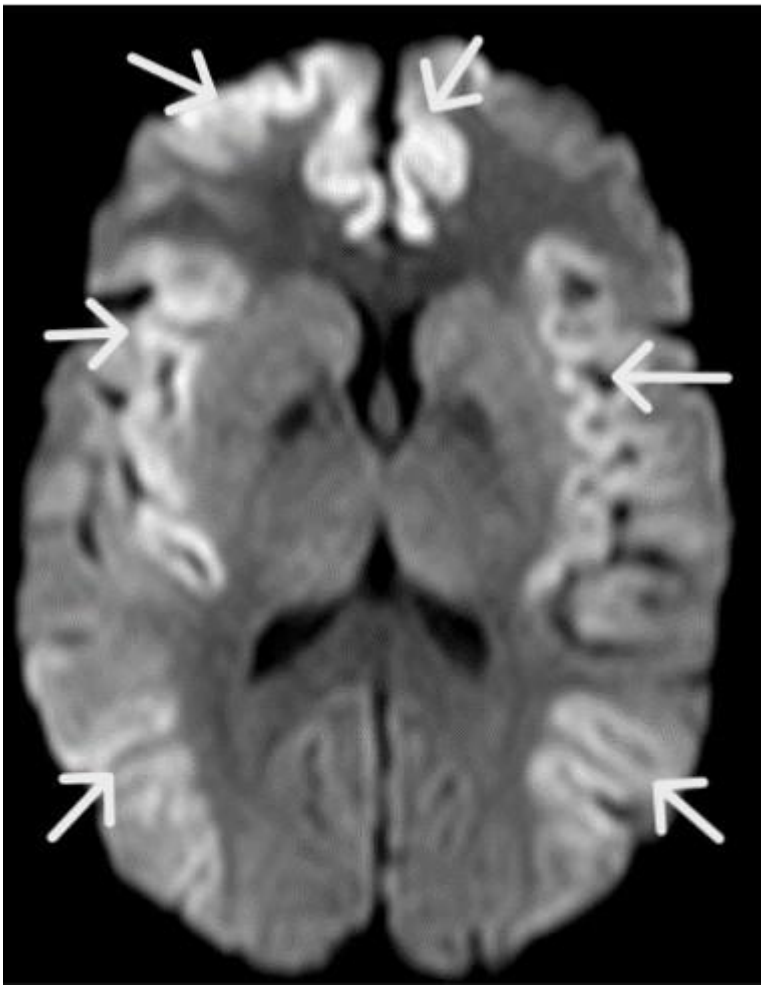
Figure 2

a



Description: DWI (2a and 3a) and corresponding ADC maps (2b and 3b) show gyriform restriction of diffusion involving bilateral cingulate gyri, insular cortices and bilateral fronto-temporo-parietal lobes with sparing of bilateral peri-rolandic regions. **Origin:** © Department of Radiodiagnosis, Command Hospital (Western Command), Panchkula, Haryana, India

b

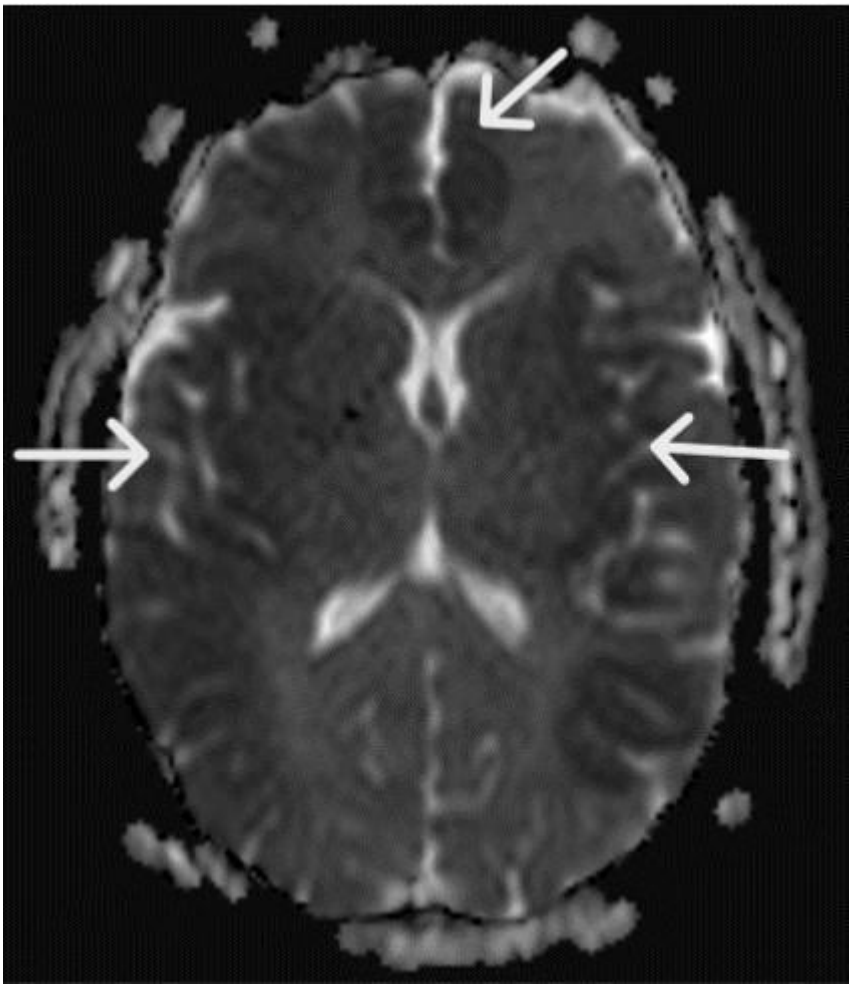


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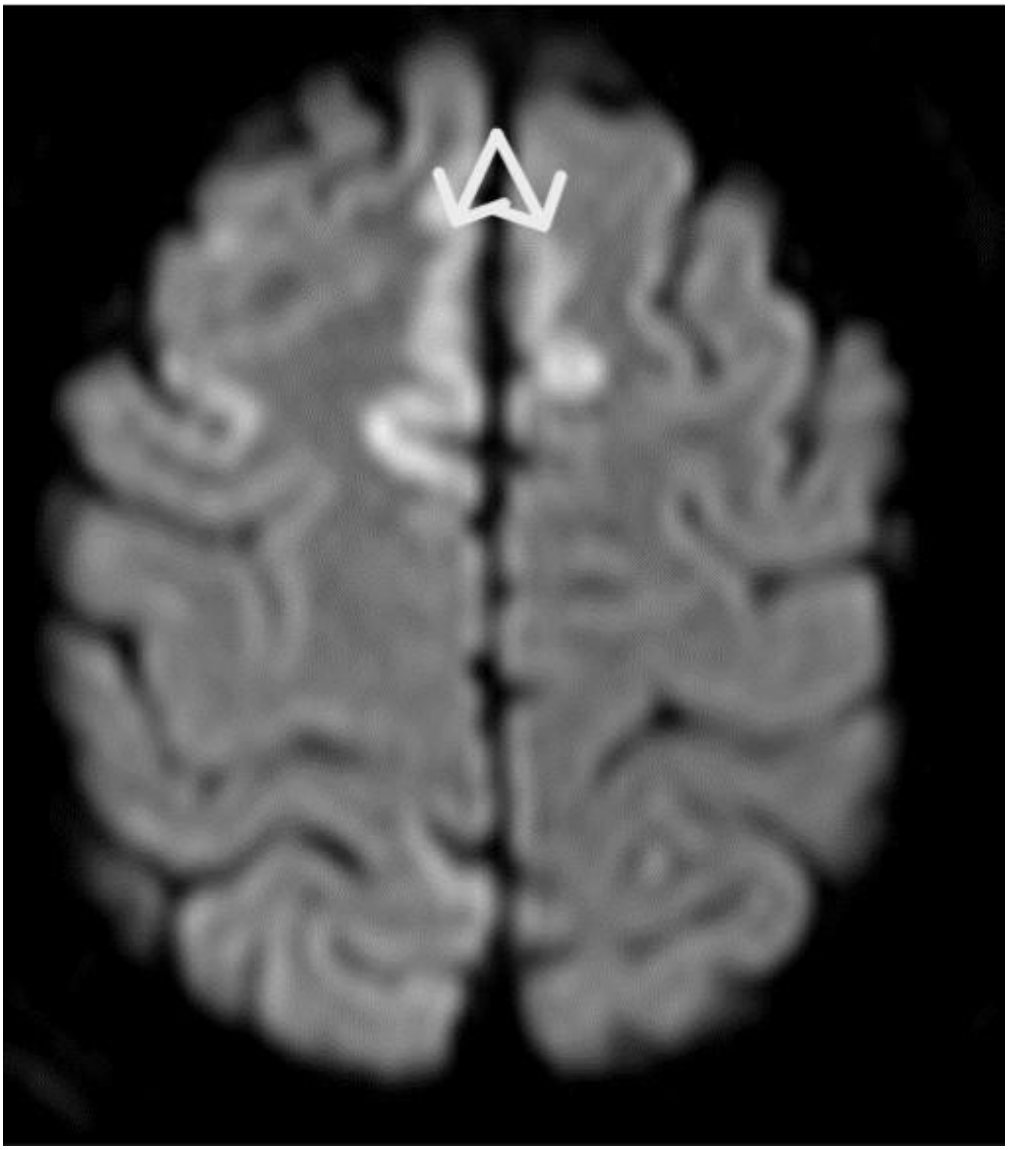
Figure 3

a



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b

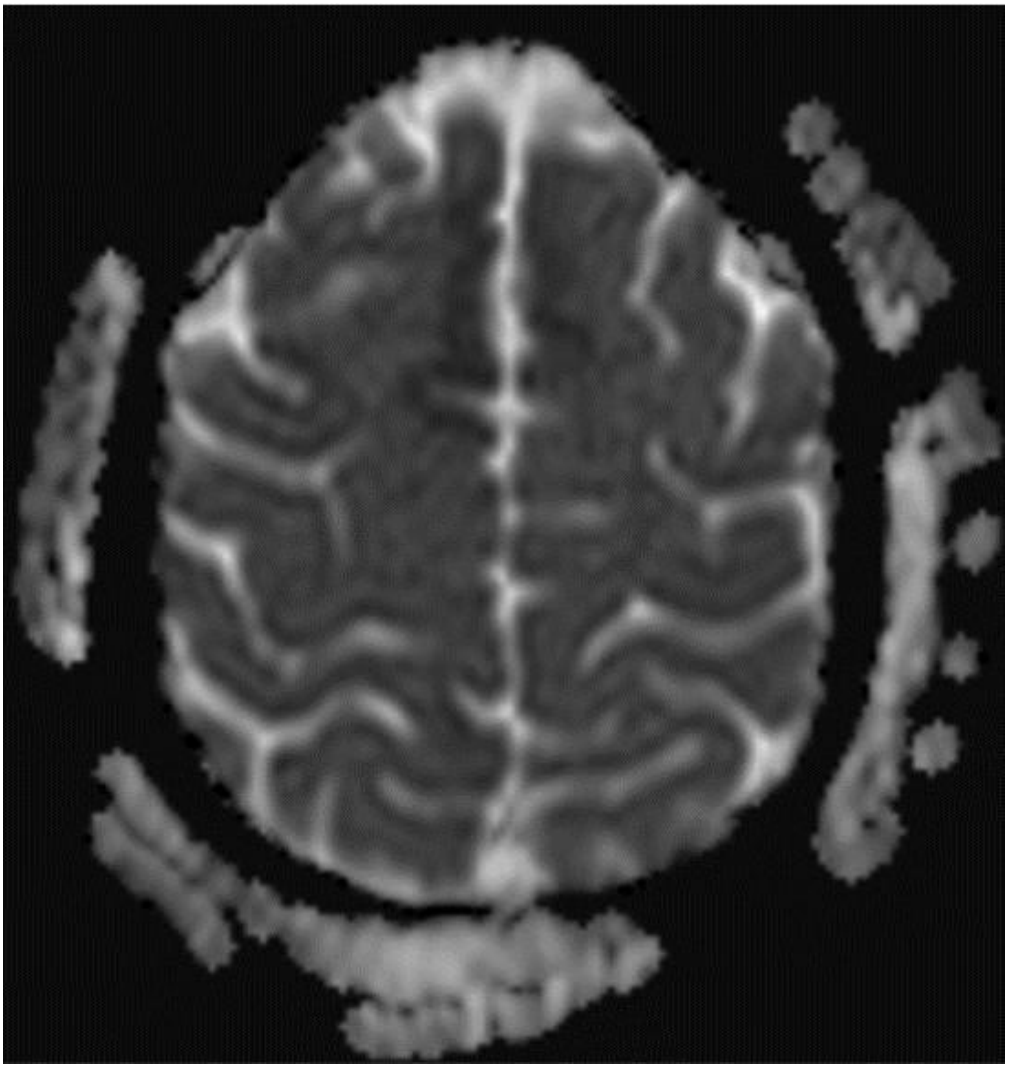

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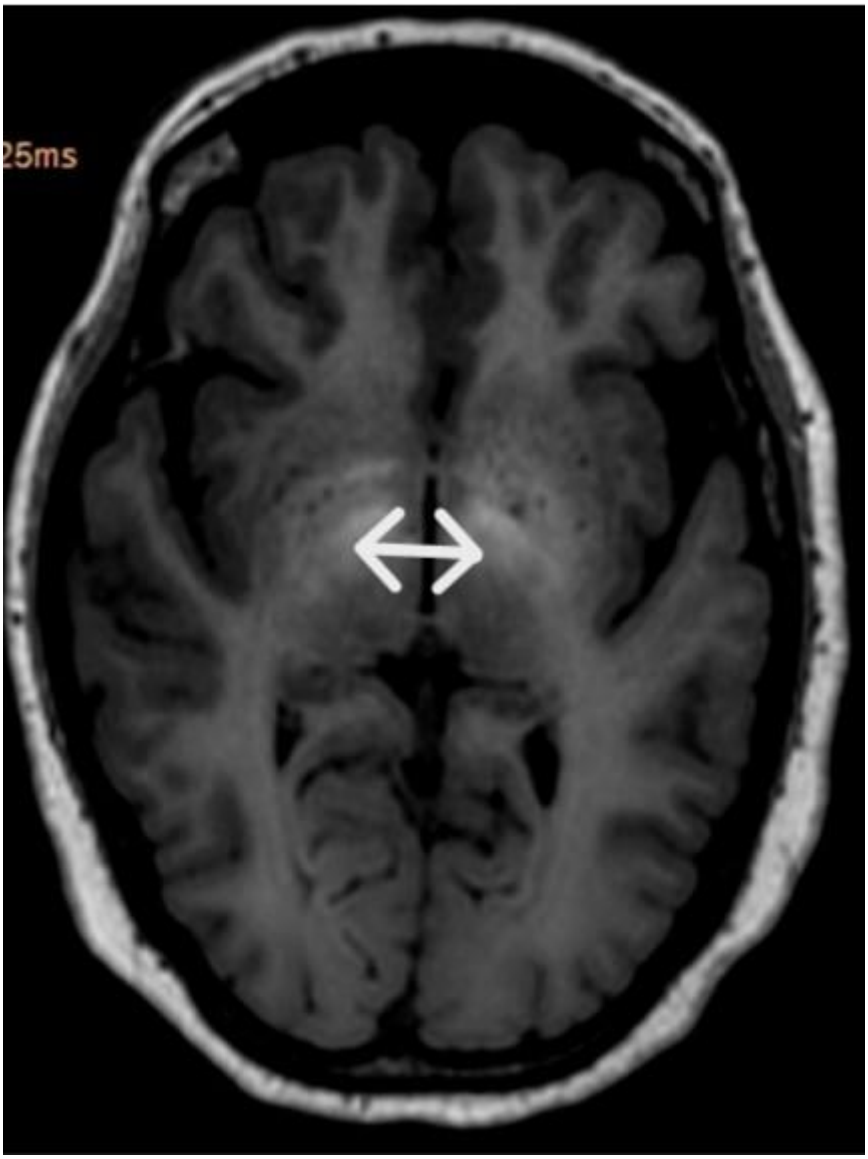
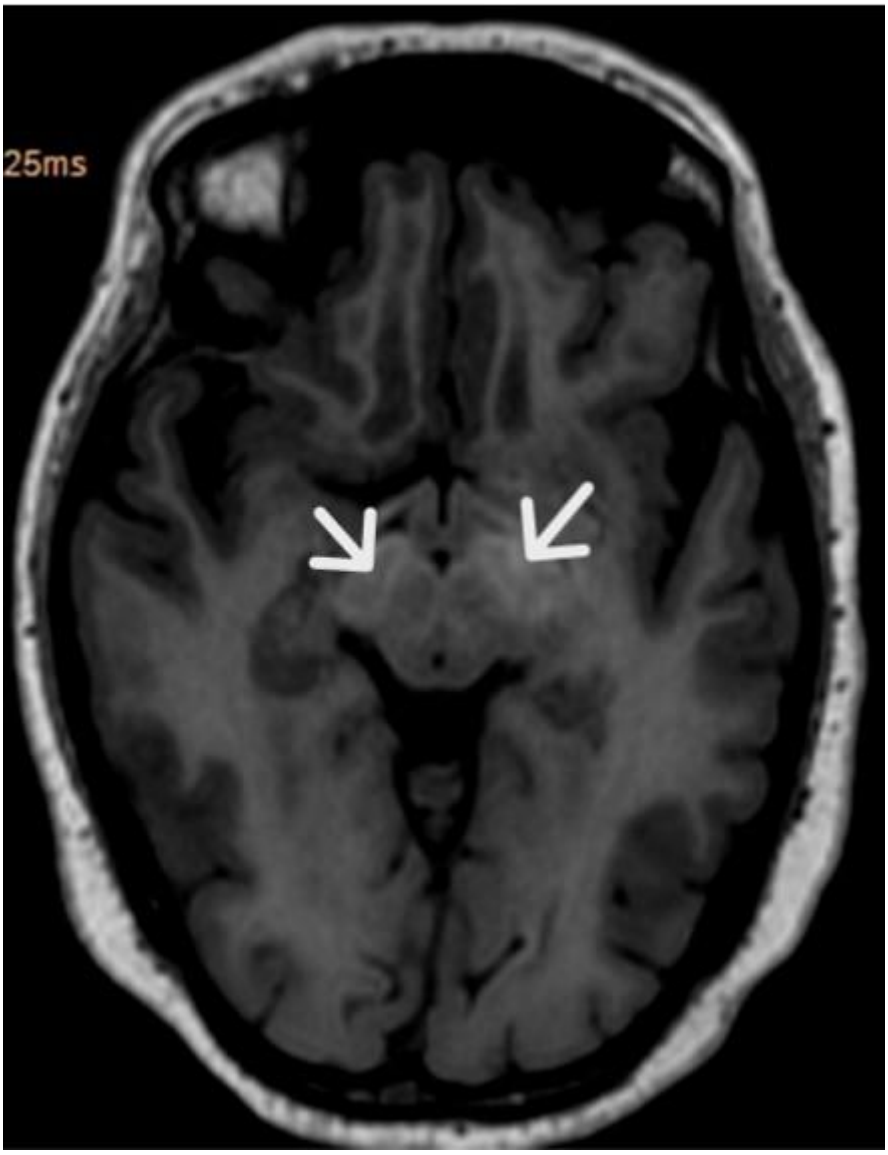
Figure 4

a



Description: Axial T1WI (4a and 4b) shows hyperintensity involving bilateral globus pallidi and anterior aspects of bilateral cerebral peduncles of the midbrain, likely due to manganese deposition. **Origin:** © Department of Radiodiagnosis, Command Hospital (Western Command), Panchkula, Haryana, India

b



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