Clinical Medicine and Health Research Journal (CMHRJ)
Volume 05, Issue 02, March - April, 2025
Page No. 1248-1252,

Case Report

Is Fatty Liver the Last Nail in The Coffin for Central Pontine Myelinolysis? A Case Report and Literature Review

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Received: 10 March, 2025 : Accepted: 10 April, 2025 : Published: 17 April 2025

Abstract:

We report a case of a 45-year-old male admitted with recurrent falls, melaena, abdominal pain, vomiting, deranged electrolytes, and a low heamoglobin. He has a complex past medical history, including a lung abscess, alcohol abuse, a history of falls, cholecystitis, duodenal ulcer, duodenitis, epilepsy, folate deficiency, an ankle fracture, gallstones, a history of anoxic brain injury, and pancreatitis. His medications include lamotrigine, losartan, quetiapine, omeprazole, and venlafaxine MR. The patient's history reveals excessive alcohol intake, malnutrition, and smoking. Ultrasound showed the presence of diffuse fatty liver and gallstones. He developed left-sided weakness, which was not present before. MRI of the head revealed a classic trident appearance in the pons, consistent with post-motor demyelination/pontine myelinolysis. It was noted that a few days prior to the MRI, the patient had been treated for mild hyponatraemia, and 10 days before, he had been noted to have mild hypernatremia. In this case report, we demonstrate how fatty liver can be associated with an increased risk of central pontine myelinolysis (CPM).

Key words: fatty liver, central pontine myelinolysis, malnutrition,

Introduction

The liver appears to play an important role in the causation of central pontine myelinolysis (CPM). For instance, CPM has been reported in association with liver transplants, liver cirrhosis, and chronic alcohol intake (1). Other risk factors for CPM, which can directly or indirectly affect the liver, include malnutrition, malignancy, hypertriglyceridemia, and hyperglycemia (2,3). CPM is known to be related to osmotic demyelination that occurs in the brain with the rapid correction of hyponatremia (4). However, in recent years, CPM has also been reported in cases of normonatraemia and hypernatraemia (5). Even meticulous correction of hyponatremia in high-risk patients, like chronic alcoholics, can also precipitate the onset of CPM (6,7). This raises the possibility that other factors may play a crucial role in the pathogenesis of CPM. Importantly, a case report has shown that acute liver dysfunction can occur with CPM in the absence of alcohol intake, malnutrition, and with normal plasma sodium levels. Rarely, CPM has also been reported in association with Wilson's disease and abnormal liver enzymes (8,9). In a case series of eight patients, it was shown that CPM was associated with adrenal insufficiency, alcohol use, kidney and liver failure, immunocompromised states, uncontrolled diabetes, electrolyte imbalances, high triglycerides, and the use of antidepressants (10). Liu et al. showed that chronic use of venlafaxine can also be associated with CPM in individuals without a history of alcohol use, malnutrition, or liver disease (11). In systematic review that included 541 patients with CPM, showed most clinical presentation was encephalopathy, favourable outcome in 51.9% and death was in 24.8%. Importantly, the systematic review showed patients with liver transplant have combined rate of death and disability of 77.4% (12). It is plausible to suggest that both direct and indirect factors are associated with CPM in particular liver diseases.

Case report

We report a case of a 45-year-old male admitted with recurrent falls, melaena, abdominal pain, vomiting, deranged electrolytes, and low hemoglobin (Hb). He has a complex medical history, including lung abscess, alcohol abuse, falls risk, cholecystitis, duodenal ulcer, duodenitis, epilepsy, folate deficiency, ankle fracture, gallstones, a history of anoxic brain injury, and pancreatitis. His medications include lamotrigine 100 mg BD, losartan 100 mg OD, quetiapine 100 mg AM and 200 mg EV, omeprazole 20 mg BD, and venlafaxine MR. He lives alone in a flat, smokes approximately 10-15 cigarettes/day since his teenage years, and drinks half a bottle of vodka per day. His blood investigations are shown in Table 1. Other investigations included a liver ultrasound, which showed the presence of diffuse fatty liver and gallstones, but no liver cirrhosis. He also received a CT scan of the chest, abdomen, and pelvis due to weight loss, which revealed colitis, gallstones,

and no evidence of cancer. Over the past two weeks, he has noted worsening weakness on his left side, which was not present before. He has also experienced numbness and tingling sensations in both hands and feet. CT head imaging showed no intra- or extra-axial hemorrhage, age-related brain involution changes, and deep white matter ischemia with a few old lacunar infarcts. No mass lesion was seen, and the skull appeared unremarkable. MRI of the head showed the pons with a classic trident appearance, consistent with post-motor demyelination/pontine myelinolysis (Figure 1). It was noted that a few days prior to the MRI, the patient had been treated for mild hyponatremia, and 10 days before that, he was also noted to be in mild hypernatremia (Figure 2). His ECG showed normal sinus rhythm, and he was started on normal saline to prevent dehydration and melaena. Later, he received a blood transfusion. Endoscopy revealed oesophagitis, a 2 cm hiatus hernia, and mild duodenitis, but no active bleeding or fresh blood was noted. A few days later, the patient made a full recovery, was seen by a physiotherapist and occupational therapist, and was discharged home.This case report demonstrates how fatty liver can be associated with an increased risk of central pontine myelinolysis (CPM).

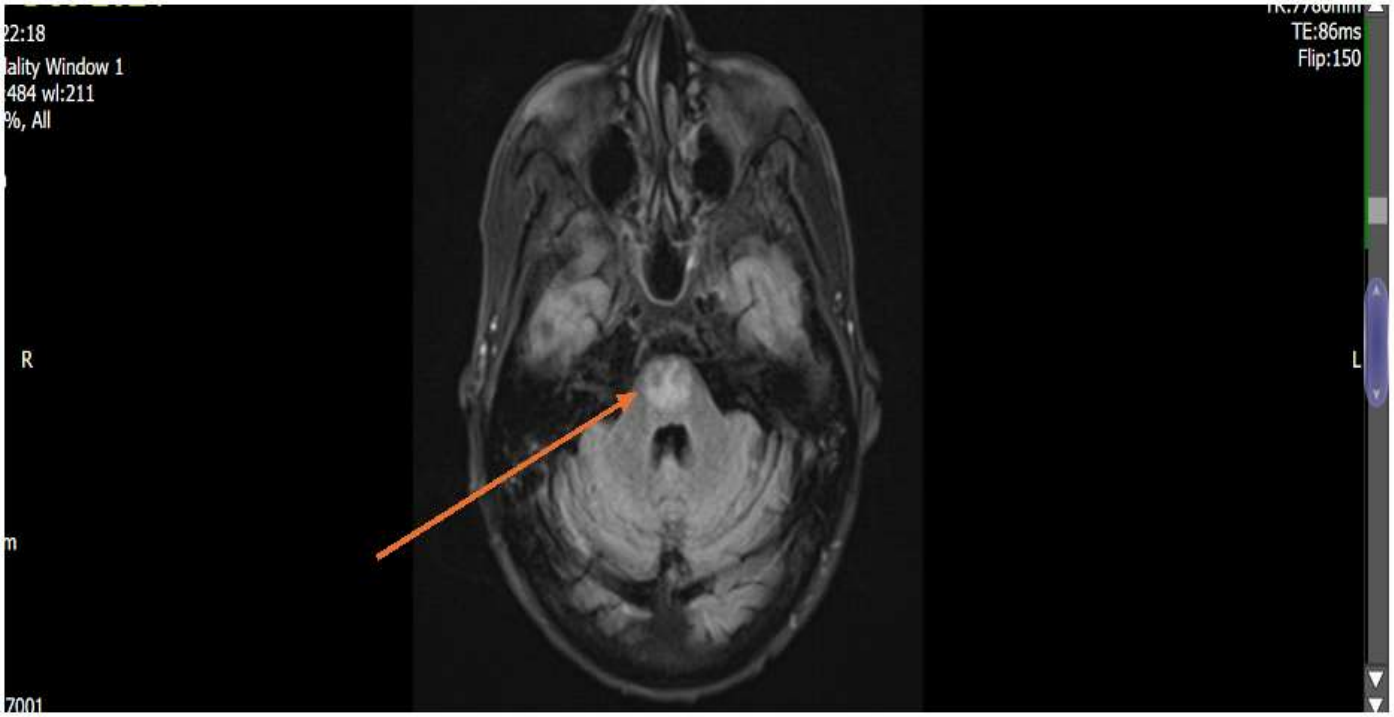

Figure 1 The pons reveals a classic trident appearance in keeping with post pontine myelinosis

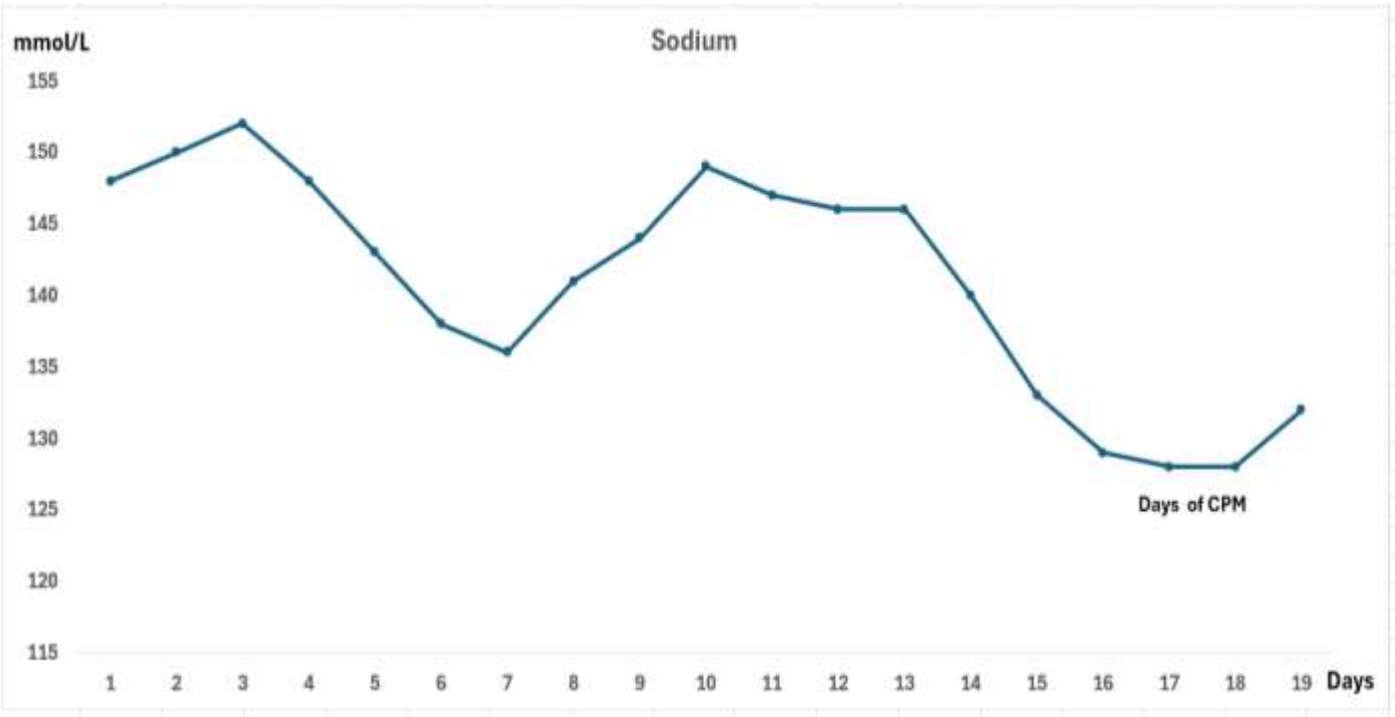

Figure 2 shows the level of plasma sodium. Mild fluctuations in hypernatremia were noted before the onset of mild hyponatremia, at which point the patient reported weakness on the left side, and an MRI revealed the presence of central pontine myelinolysis (CPM)

Table 1: List of summary of investigations performed

Test	Value and normal reference range
Hb	84 (130-170 g/L)
White cell count (WCC) Platelets	6.5 (3.7-11.1 6.5 x10*9/L) 466 (150-450 466 x10*9/L)
Urea	4.2 mmol/L (2.5-7.5)
Creatinine Glomerular filtration rate	52 mmol/L (reference range 64-104) 59 ml/min (> 90)
TSH	6.11 pmol/L (0.34-5.6)
FT4	10.1 pmol/L (7.7-15.1)
CRP	6.4 mg/L (0-6)
HbA1c	5.2%
ALT	12 (1-49)
GGT	112 (0-54)
Alkaline phosphatase	100 (30-130)
PH	7.44
Lactate	1 mmol/L (< 2)
Glucose	7.5 mmol /L
Faecal Elastase	<15 ug/g [Reference Range = 200 - 0]

Discussion:

Fatty liver has been associated with CPM in diabetic Wistar rats (13). Endo et al. reported an association between CPM and fatty liver cirrhosis due to alcohol (14). Current evidence from the literature does not show a direct association between fatty liver and CPM. However, in our patient, there are several risk factors for CPM, and we propose that the presence of fatty liver may exacerbate the onset of CPM symptoms. For instance, our patient has a history of chronic alcohol intake, malnutrition, and immunocompromise (1-6). He also experienced a period of hypernatremia and was later treated for mild hyponatremia (4,5). Importantly, his medication regimen included quetiapine and venlafaxine, both of which have been linked to CPM (11,15). Brain injury has also been associated with CPM, and our patient has a known history of anoxic brain injury (16). Fatty liver is an important risk factor for stroke. For instance, the ten-year incidence rates of stroke with fatty liver was found to be 8.36% in south Korea (17). Xu et al showed that in 742 participants and follow up of 2.9 years , non-alcoholic fatty liver disease (NAFLD)was associated with significant recurrent stroke or TIA (18). Wang et al, showed in systematic review and meta-analysis that NAFLD was associated with increased risk of stroke (19). It is worth noting that, despite his heavy alcohol consumption, we have not established whether he developed liver cirrhosis. Although fatty liver disease and CPM are not directly related, they may share some indirect associations. For example, fatty liver can lead to cirrhosis, which may be associated with hyponatremia. Alcohol abuse can also increase the risk of cirrhosis and hyponatremia. Individuals with fatty liver disease, particularly in the context of malnutrition and alcohol abuse, may be more vulnerable to rapid electrolyte shifts, especially during hospitalization or treatment, which could increase the risk of CPM. In this sense, fatty liver could potentially act as a contributing factor or the "last straw" in individuals who already have other risk factors for CPM.(Figure 3).

Conclusion

Fatty liver disease is not typically considered a direct cause of CPM. However, in patients with liver disease or other conditions like chronic alcoholism, malnutrition and electrolyte disturbances, fatty liver may play a role in triggering conditions that predispose individuals to CPM. Further research is needed to determine whether fatty liver is involved in the pathogenesis of CPM.

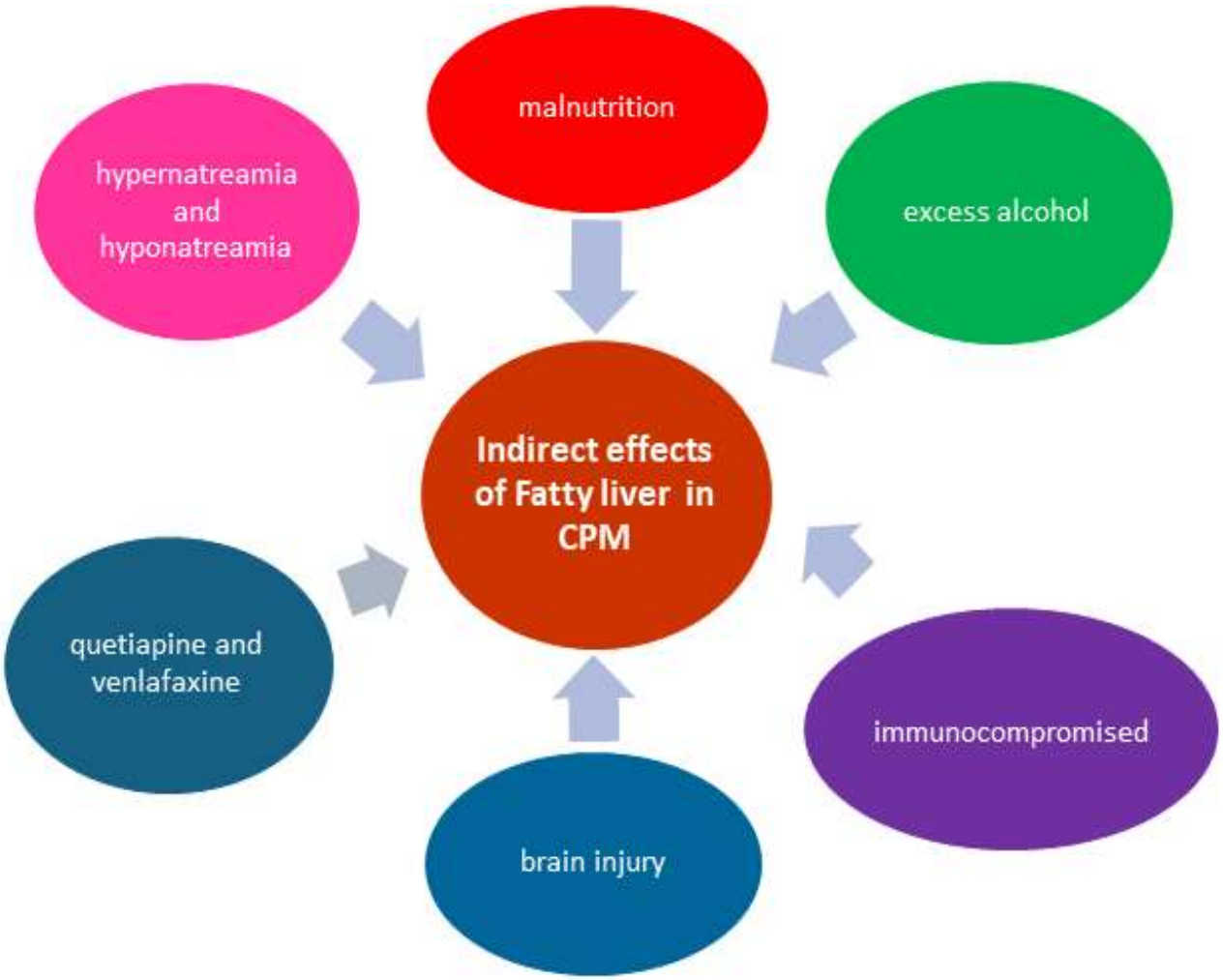

Figure 3: **Summary of factors reported in the history of patients linked with central pontine myelinolysis (CPM). Fatty liver, in the presence of these factors, may be the final contributor to the association with CPM.**

Acknowledgments

We are grateful for all the team in the stroke ward (ward 7) in Milton Keynes University Hospital for their unlimited cooperation.

Funding Statement

None to declare.

Conflict of Interest

None to declare.

Informed Consent

Informed consent for publication of this case report was obtained from the patient.

Author Contributions

Conception and design: all authors. Administrative support: all authors. Provision of study materials or patients: all authors. Collection and assembly of data: all authors. Data analysis and interpretations, manuscript writing, and final approval of manuscript: all authors.

Data Availability

The authors declare that data supporting the findings of this study are available within the article.

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