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HEPATIC MYELOPATHY AS A RARE BUT REVERSIBLE NEUROLOGICAL COMPLICATION POST TRANSJUGULAR INTRAHEPATIC PORTOSYSTEMIC SHUNT (TIPS) INSERTION IN LIVER CIRRHOSIS

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ABSTRACT

Hepatic myelopathy is a rare but underrecognized complication of chronic liver disease characterized by pure motor, symmetric and spastic paraparesis without sphincter involvement. We report the case of a 39-year-old female with Child Pugh A alcohol-related cirrhosis presenting with progressive spastic paraparesis 2 months after transjugular intrahepatic portosystemic shunt (TIPS) placement for recurrent oesophageal variceal bleed. TIPS occlusion achieved partial neurological recovery but significant functional improvement. She had no further presentations of decompensation with variceal bleeding or hepatic encephalopathy. Early recognition and multidisciplinary input are essential in preserving functional outcome, especially when liver transplant is not indicated.

KEYWORDS

Hepatic myelopathy, liver cirrhosis, portal hypertension, transjugular intrahepatic portosystemic shunt

LEARNING POINTS

- The onset of symmetric, pure motor spastic paraparesis following a transjugular intrahepatic portosystemic shunt procedure in cirrhosis should prompt clinical suspicion for hepatic myelopathy.
- Hepatic myelopathy can be reversible with timely shunt occlusion/modification, especially when a liver transplant is not indicated.
- Recognizing liver cirrhosis as a systemic syndrome and adopting early multi-disciplinary input improve patient outcomes.

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INTRODUCTION

Hepatic myelopathy is a rare but debilitating neurological complication of liver cirrhosis, manifested as progressive, symmetric spastic paraparesis with minimal sensory or sphincter involvement[1]. It occurs in the context of portocaval shunts, either spontaneous or iatrogenic via the placement of a transjugular intrahepatic portosystemic shunt (TIPS), with a reported incidence of up to 7.1% in the post TIPS population[2].

CASE DESCRIPTION

A 39-year-old woman with a history of Child-Pugh A alcohol-related cirrhosis presented to the outpatient physiotherapy clinic in October 2024 with a spastic gait. She reported weekly falls due to impaired balance, progressive right lower limb weakness and spasms in the preceding months.

In August 2024, the patient received a pre-emptive TIPS after having five hospital admissions for recurrent oesophageal variceal bleeding despite repeated endoscopic band ligation and maximal medical therapy. The hepatic venous pressure gradient was satisfactorily reduced from 20 mmHg to 9 mmHg. Her neurological history was significant for alcohol withdrawal seizures and craniotomy for a traumatic subdural haemorrhage with no residual neurological deficits. Relevant medications included carvedilol, lactulose, rifaximin, spironolactone and acamprosate.

The patient was admitted to the neurology unit for further workup. The neurological examination revealed the following:

- Spasticity in all limbs, more pronounced in the lower limbs and on the right;
- Brisk reflexes throughout the upper and lower limbs, with spreading ankle jerks and a positive crossed adductor sign on the right;
- Positive Hoffman's sign bilaterally; plantar reflex was extensor on the right and flexor on the left;
- Power was preserved except for mild right hip flexion, knee flexion and great toe extension weakness (grade 4/5);
- Sensation to fine touch, temperature and vibration was globally reduced on the right lower limb;
- No dysmetria and limb or gait ataxia was observed.

At this stage, the patient had compensated Child Pugh A 5 disease and relatively unremarkable biochemical parameters: haemoglobin 124 g/l, platelets 202 $\times 10^9$/l, international normalized ratio 1.3, creatinine 56 µmol/l, estimated glomerular filtration rate >90 ml/min/1.73m^2, albumin 42 g/l, bilirubin 14 µmol/l, alanine aminotransferase 33 U/l, gamma-glutamyl transferase 330 U/l, alkaline phosphatase 113 U/l, and ammonia 62 µmol/l (upper normal limit 53). An extensive myelopathy screen was unrevealing: vitamin B12 340 pmol/l, vitamin E 27 µmol/l, plasma copper 13 µmol/l, negative antineutrophil cytoplasmic antibody and antinuclear antibody serology,

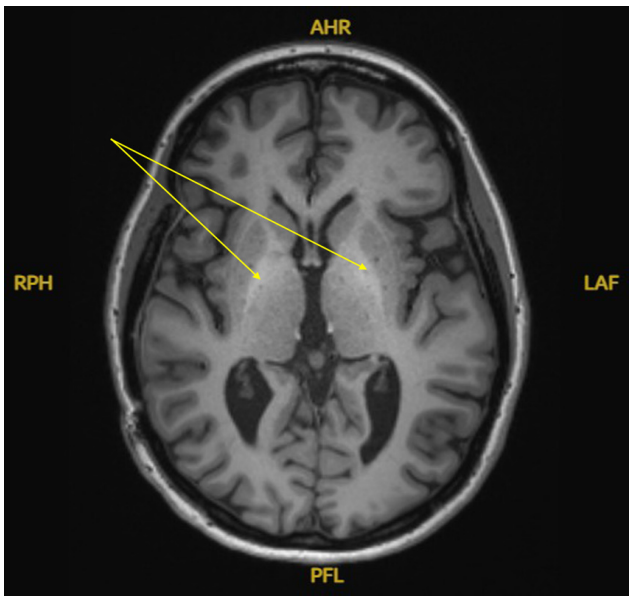


Figure 1. Magnetic resonance imaging of the brain: T1 sequence axial view, of of a 39-year-old woman at time of presentation with spastic paraparesis, demonstrating bilateral hyper-intensity in the globus pallidus (yellow arrows) due to manganese deposition, a typical sign of hepatic encephalopathy.

negative neuronal antibody screen(Hu, Yo, Ri, CV2/CRMP6, Zic4, GAD65, Tr, Amphiphysin, SOX1), negative syphilis enzyme immunoassay total antibody assay, human immunodeficiency antigen and antibody, hepatitis C antibody, and hepatitis surface antigen. Thyroid function was normal. Magnetic resonance imaging (MRI) of the brain revealed encephalomalacia in the right temporal lobe with smooth postoperative thickening of the dura, and T1 signal hyper-intensity in the globus pallidus bilaterally *(Fig. 1)*. MRI of the spine revealed right paracentral disc protrusion at the level of C5-C6 with mild canal narrowing with a normal cord signal throughout.

The case was reviewed in a multidisciplinary team meeting involving hepatologists, neurologists and interventional radiologists. A presumptive diagnosis of hepatic myelopathy was made given the temporal association with TIPS and the absence of other structural or metabolic. Baclofen 5 mg was initiated twice daily and up titrated for symptomatic spasticity. As the patient was not a liver transplant candidate due to ongoing alcohol use, a decision was made to occlude the TIPS to prevent progressive neurological and functional deterioration. Via a transjugular approach, remnant gastric portosystemic varices were shown to originate via the left gastric vein and were embolized using 10 ml of 3% polidocanol (Aethoxysclerol foam, Kreussler Pharma, Wiesbaden, Germany) followed by two 0.035" coils *(Fig. 2)*. Following this, a 14 mm Amplatzer plug was placed into the TIPS to permanently occlude it.

Four months after TIPS embolization, the patient's exercise tolerance improved from 2km to 4km. She regained the ability to independently sit-to-stand, which she was unable to do so unaided prior to TIPS embolization. She

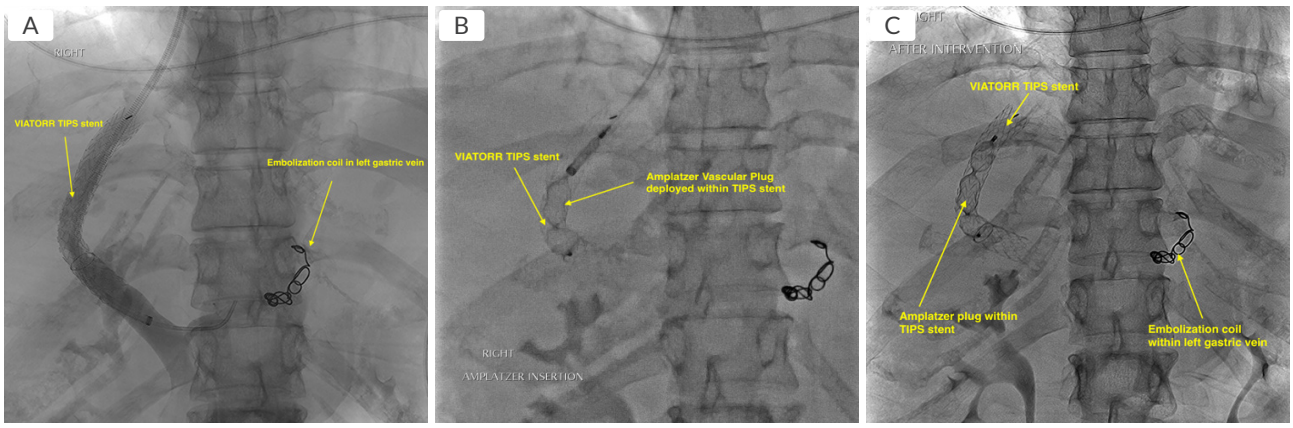


Figure 2. A) Access of the left gastric vein via a right transjugular approach through the TIPS. Post embolization of the left gastric vein with a combination of aethoxysklerol foam and tornado coils. B) Embolization of TIPS with a 14mm Amplatzer Vascular Plug. C) Successfully deployed Amplatzer plug at the end of intervention.
Abbreviation: TIPS, transjugular intrahepatic portosystemic shunt.

only experienced one fall since the procedure, owing to tripping over in new pair of shoes. A serial neurological exam confirmed improved lower limb power and spasticity. However, upper motor neuron signs including brisk reflexes and upgoing plantar responses persisted. She had not had further presentations of decompensation with variceal bleeding.

DISCUSSION

The pathophysiology of hepatic myopathy is thought to be multifactorial. Proposed mechanisms include neuronal damage by ammonia and other nitrogenous toxins such as indoles, mercaptans and fatty acids bypassing hepatic clearance, nutritional deficiencies and immune-mediated effects[3]. Post-mortem studies have shown symmetrical demyelination of the pyramidal tract, preferentially affecting the thoracic spinal cord than the cervical spinal cord[4]. This is in keeping with the clinical findings of upper motor neuron signs with a lower limb predominance.

The diagnosis of hepatic myelopathy remains clinical, following the exclusion of other possible aetiologies with multidisciplinary input. Differentials are broad and a thorough history of environmental exposure, substance use, malignancy and family history should be gathered, with particular focus on infective, inflammatory, autoimmune, paraneoplastic, metabolic, toxic, neurodegenerative and hereditary aetiologies such as motor neuron disease, multiple sclerosis, subacute degeneration of the cord secondary to B12 deficiency, hereditary spastic paraparesis, and vascular causes[3]. Despite histopathological findings of demyelination in late-stage hepatic myelopathy, MRI of the spine is frequently unremarkable, as was the case with our patient. Nevertheless, MRI of the brain with signs of hepatic encephalopathy favours the diagnosis of hepatic myelopathy[4-6]. Neurophysiologic testing can be valuable in detecting early hepatic myelopathy whereby a prolonged central motor conduction time in the lower limbs is supportive of corticospinal tract demyelination with preserved axons[1].

The clinical trajectory of hepatic myelopathy and its response to treatment are poorly understood and limited to case reports and series. None of the conservative management strategies such as ammonia-lowering agents, dietary supplementation, protein restriction, and oral nitrates show consistent benefit in delaying or reversing the progression of functional disability[7]. Retrospective data derived from a cohort of 12 patients with hepatic myelopathy who underwent TIPS reduction demonstrated improvement in strength and functional indices at 1 and 3 months. Unfortunately, neither sustained benefits nor full recovery was noted beyond this period, possibly owing to the progression of the underlying liver disease[8]. Liver transplantation is considered the definitive treatment; however, neurological recovery varies depending on the severity of initial liver dysfunction and the extent of myelopathy at the time of transplantation[9,10]. Emerging therapies such as faecal microbiota transplantation showed promise in individual case reports but require further prospective investigation and validation[10].

CONCLUSION

Hepatic myelopathy is a rare but debilitating complication of chronic liver disease. In patients with cirrhosis and portal hypertension, the onset of new, symmetrical, pure motor spastic paraparesis should prompt clinical suspicion for hepatic myelopathy to facilitate early diagnosis and potentially improve outcomes. This case underscores the importance of a multidisciplinary approach in the timely recognition of hepatic myelopathy and highlights shunt modification as a therapeutic consideration when liver transplantation is not indicated.

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