Journal of Clinical and Translational Hepatology **2026**
DOI: 10.14218/JCTH.2026.00044

Illuminating and Instructive Clinical Case

Occlusion of Paraspinal Vein Shunt Alleviated Post-TIPS Hepatic Myelopathy in a Patient with Cirrhosis

Yuhong Suo[1#], Lixue Xu[2#], Xinyan Zhao[1], Yu Wang[1*], Fuliang He[1*] and Jidong Jia[1]

[1]Liver Research Center, Beijing Friendship Hospital, Capital Medical University; State Key Laboratory of Digestive Health; National Clinical Research Center of Digestive Diseases, Beijing, China; [2]Department of Radiology, Beijing Friendship Hospital, Capital Medical University; State Key Laboratory of Digestive Health; National Clinical Research Center of Digestive Diseases, Beijing, China

Received: January 13, 2026 | **Revised:** March 12, 2026 | **Accepted:** March 19, 2026 | **Published online:** May 14, 2026

Abstract

Hepatic myelopathy, a rare neurological complication of decompensated chronic liver disease, profoundly impairs quality of life. While liver transplantation represents the only curative treatment for hepatic myelopathy, we report a case in which progressive and severe spastic paraparesis was markedly improved following embolization of a paraspinal vein shunt.

Citation of this article: Suo Y, Xu L, Zhao X, Wang Y, He F, Jia J. Occlusion of Paraspinal Vein Shunt Alleviated Post-TIPS Hepatic Myelopathy in a Patient with Cirrhosis. J Clin Transl Hepatol 2026. doi: 10.14218/JCTH.2026.00044.

Introduction

Hepatic myelopathy (HM), also known as portosystemic shunt myelopathy, is a rare neurological complication of chronic liver disease characterized by progressive spastic paraparesis of the lower limbs without significant sensory impairment.[1,2] Its pathogenesis remains poorly understood. Apart from early liver transplantation, HM is currently considered incurable.[3,4] Accumulating evidence indicates that HM is strongly associated with portosystemic shunting, whether spontaneous or iatrogenic in origin.[3,5] However, the specific shunt responsible remains unclear. Here, we report a case in which a previously unrecognized paraspinal vein shunt (PVS) appears to play a pivotal role in the pathogenesis of HM.

Case presentation

A 42-year-old man with alcoholic- and hepatitis B-related cirrhosis presented with progressive lower limb weakness impairing ambulation and recurrent hepatic encephalopathy (HE). Four years prior, he had undergone transjugular intrahepatic portosystemic shunt (TIPS) placement for recurrent esophageal variceal bleeding secondary to portal hypertension, with the portal pressure gradient (PPG) decreasing from 25 to 9 mmHg. Four years following TIPS, clinical examination revealed progressive worsening of spastic paraplegia affecting both lower limbs, with preserved sensory and sphincter function; the patient was unable to ambulate independently and experienced recurrent episodes of delirium. Given his four-year history of sustained alcohol abstinence, alcohol-related neurological disorders were considered an unlikely etiology. Laboratory findings were notable for pancytopenia (white blood cell count 2,380 cells/mm^3, hemoglobin 10.1 g/dL, platelet count 45,000 cells/mm^3), hypoalbuminemia (3.1 g/dL), hyperbilirubinemia (total 3.52 mg/dL, direct 0.93 mg/dL), prolonged INR (1.4), and hyperammonemia (122 mcg/dL). Levels of vitamin B_{12} (469 pg/mL), ceruloplasmin (0.35 g/L), and 24-h urinary copper (34 μg/24 h) were within normal limits, thereby excluding Wilson disease and vitamin B_{12} deficiency as potential etiologies. Cranial magnetic resonance imaging (MRI) indicated changes compatible with metabolic encephalopathy. MRI of the entire spine showed degenerative changes only; spinal canal stenosis and compressive lesions were ruled out by the neurologist. Importantly, no intrinsic spinal cord abnormalities were identified. Abdominal imaging confirmed a patent TIPS, evidence of cirrhosis with portal hypertension and splenomegaly, and multiple portosystemic collaterals, notably a 1.18-cm-diameter gastro-renal shunt (GRS). Aggressive therapy with lactulose and rifaximin successfully normalized blood ammonia levels; however, this biochemical response was not accompanied by improvement in the patient's gait. Based on these findings, a diagnosis of HM was established. Attempted embolization of the GRS for recurrent HE led to the incidental identification of a previously unreported PVS, with the left renal vein communicating directly with the paraspinal vein and subsequently draining into the ascending lumbar vein (Fig. 1A–D). The GRS embolization procedure is shown in Figure 1E. Despite successful GRS embolization with a residual PPG of 10 mmHg, no improvement in gait function was observed. We hypothesized that the varicose paraspinal vein compressed the spinal cord, thereby contributing to the patient's gait impairment, and proceeded to embolize the PVS (PPG remained at 10 mmHg) (Fig. 1E). Post-embolization angiography demonstrated sig-

Keywords: Hepatic myelopathy; Paraspinal vein shunt; Gastro-renal shunt; Transjugular intrahepatic portosystemic shunt; Case report.

[#]Contributed equally to this work.

[*]**Correspondence to:** Fuliang He and Yu Wang, Liver Research Center, Beijing Friendship Hospital, Capital Medical University, 95 Yong-an Road, Xicheng District, Beijing 100050, China. ORCID: https://orcid.org/0000-0003-0057-9182 (YW). Tel: +86-13521681366 (FH) and +86-13311100797 (YW). Fax: +86-10-63138435 (FH) and +86-10-63138435 (YW). E-mail: hefuliangjiayuan@163.com (FH) and wangyuliver@ccmu.edu.cn (YW).

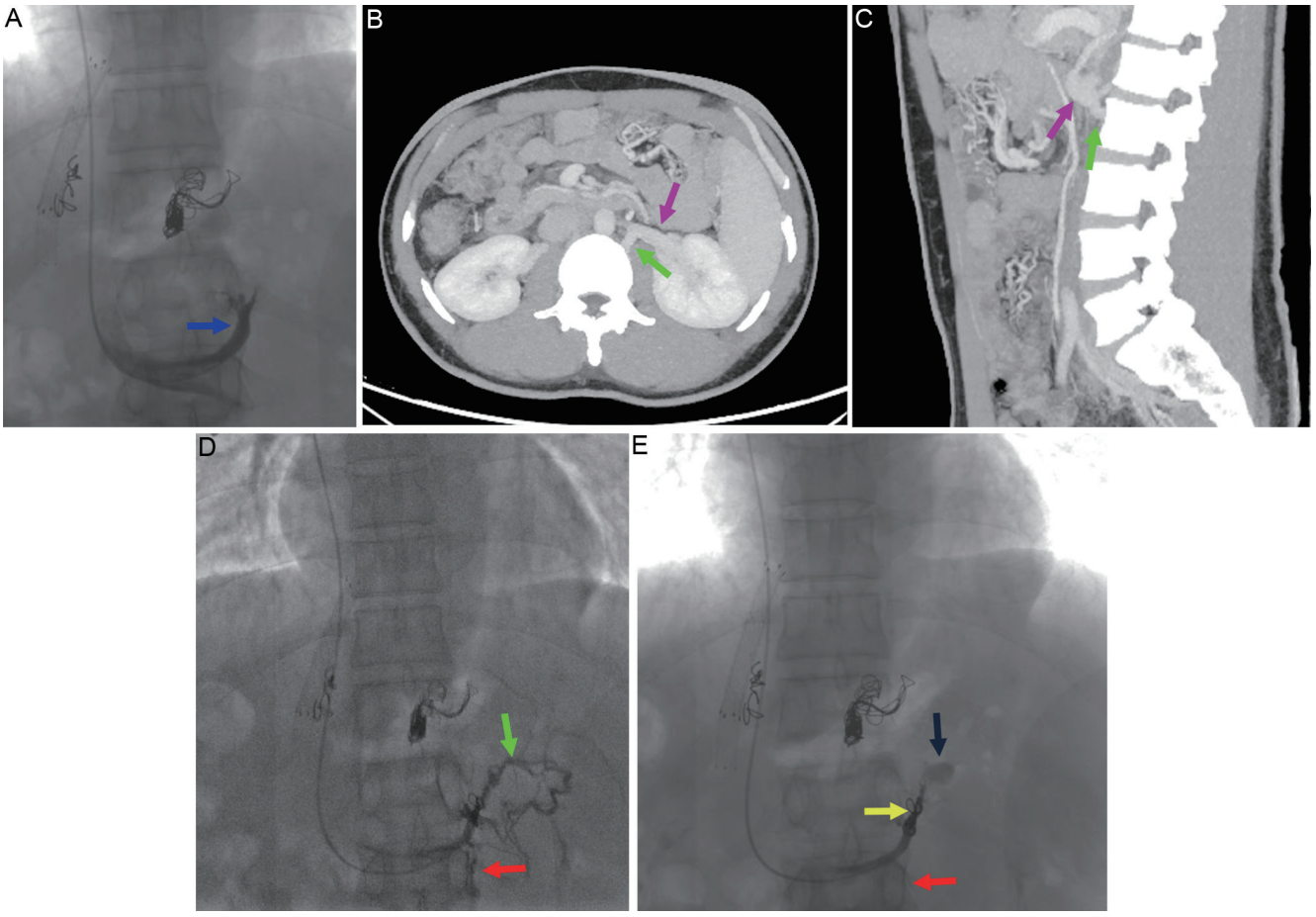

Fig. 1. Imaging and angiography findings of the patient. The blue arrow indicates the GRS; the green arrow indicates the PVS; the violet arrow indicates the left renal vein; the red arrow indicates the paraspinal vein; the yellow arrow indicates the embolization of the GRS; the black arrow indicates the embolization of the PVS. PVS, paraspinal vein shunt; GRS, gastro-renal shunt.

nificant reduction in venous varicosities compared to baseline (Fig. 1E). Remarkably, lower limb muscle strength improved at the 1-month postoperative follow-up. Subsequent assessments at 2, 3, 6, and 12 months, conducted via video-based assessments or outpatient visits, demonstrated sustained functional recovery. Currently, the patient maintains independent ambulation with a daily walking capacity of 3–4 miles (Supplementary Video 1). Preoperatively, key muscle groups exhibited Medical Research Council (MRC) grades 0–1 (flaccid paralysis), improving to grades 4–5 post-procedure. Severe spasticity (Modified Ashworth Scale (MAS) grade 3–4) decreased to mild spasticity (MAS grade 1+). Functional Ambulation Category (FAC) improved from 0 (non-ambulatory) to 5 (independent ambulation on all surfaces).

Discussion

Currently, understanding of HM remains limited,[6] necessitating urgent investigation into its pathogenesis and the development of novel therapeutic strategies.

Prior investigations have established strong associations between HM, overt hepatic failure, and spontaneous or iatrogenic portosystemic shunting,[7] which permits toxic substances to bypass hepatic metabolism. While liver transplantation represents the sole curative intervention for early-stage HM by restoring hepatic function, interventional radiology offers alternative approaches.[8] Huang *et al.* demonstrated HM alleviation following embolization of an inferior mesenteric venous shunt combined with partial splenic artery embolization.[9] Han *et al.* similarly reported concurrent resolution of HE and HM after paraumbilical vein embolization.[10] These interventions presumably exert benefit through dual mechanisms: elimination of toxin delivery to spinal motor pathways and amelioration of portal hypertension.

In the present case, GRS embolization alone failed to achieve clinical improvement, whereas PVS embolization resulted in substantial recovery of independent ambulation. This discrepancy suggests that the GRS may not constitute the sole upstream feeder to the PVS. Furthermore, despite embolization of GRS presumably reducing toxic insult to the spinal cord, no improvement in walking ability was observed. This finding indicates that venous hypertension, rather than toxicity, likely constitutes the predominant pathogenic mechanism in HM (Fig. 2). PVS embolization may have alleviated mechanical compression of the spinal cord by the varicose paraspinal vein, with resultant restoration of independent ambulation, providing empirical support for this hypothesis. These findings implicate the PVS as a critical final common pathway in HM pathogenesis.

This study is limited by its single-case design, precluding definitive conclusions regarding generalizability. The favorable outcome observed may be attributable to patient-specific

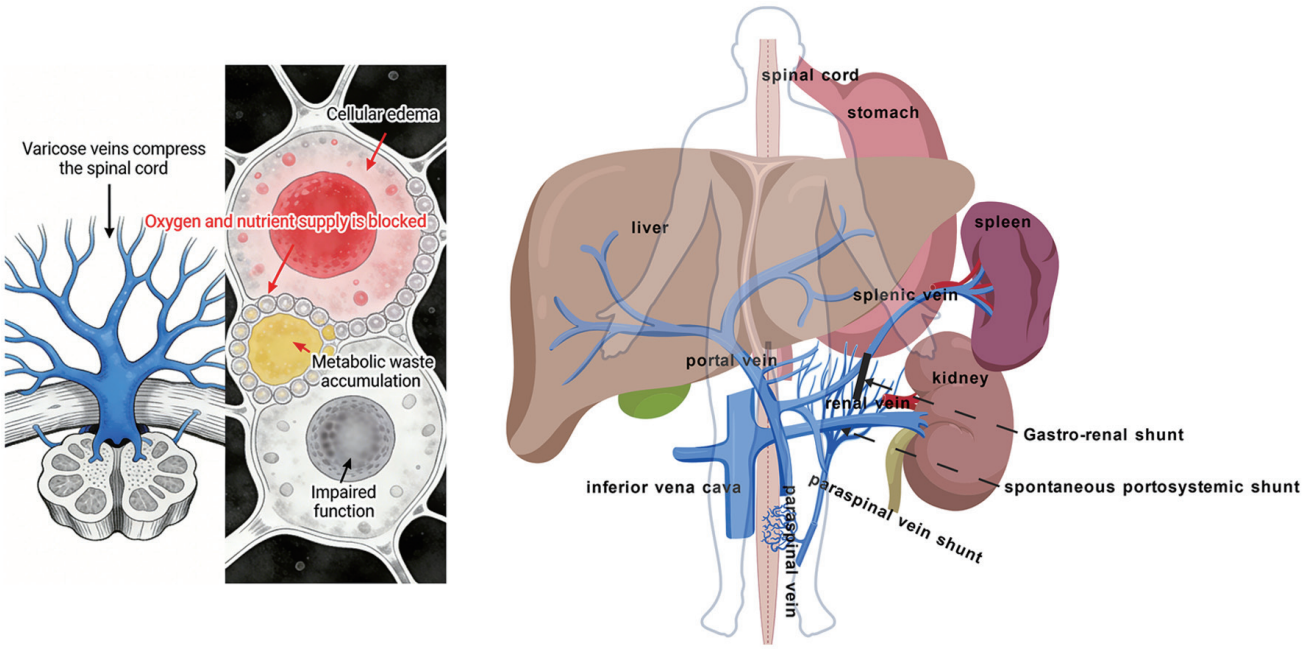

Fig. 2. Schematic illustration of the proposed pathogenic mechanism in HM. HM, hepatic myelopathy.

factors, distinct disease phenotypes, or unique anatomical configurations that may not translate to broader populations. Ongoing prospective enrollment is underway to substantiate these preliminary observations, establish the reproducibility of PVS embolization, and characterize its long-term therapeutic durability in HM.

Conclusions

Embolization of the PVS may interrupt the pathway through which venous compression mediates spinal cord injury. These findings offer novel insights into HM pathogenesis and suggest a promising interventional alternative for patients who are not candidates for liver transplantation.

Acknowledgments

The authors thank the patient and his family for agreeing to use their data for research purposes, specifically for the publication of this report.

Funding

This work was supported by the Beijing Municipal Administration of Hospitals Incubating Program (No. PX2023003) and the Capital's Funds for Health Improvement and Research (CFH 2022-2-1104).

Conflict of interest

JJ has been an Executive Associate Editor of *Journal of Clinical and Translational Hepatology* since 2013. The other authors have no conflict of interests related to this publication.

Author contributions

Study concept and design (YS, FH), acquisition of data (LX, XZ), analysis and interpretation of data (YS, LX), drafting of the manuscript (YS, LX), critical revision of the manuscript for important intellectual content (YW, FH, JJ), and study supervision (FH). All authors have made significant contributions to this study and have approved the final manuscript.

Ethical statement

The study was performed in accordance with the ethical standards of the institutions to which the authors are affiliated (No. 2022-P2-141-02) and with the Declaration of Helsinki (as revised in 2024). Written informed consent was obtained from the patient for publication of this case report.

Data sharing statement

The data used to support the findings of this study are available from the corresponding author at hefuliangjiayuan@163.com upon request.

References

[1] Acar S, Dinckan A, Akyildiz M. Liver transplantation in hepatic myelopathy. Hepatol Forum 2022;3(2):64–65. doi:10.14744/hf.2021.2021.0004, PMID:35783476.
[2] Nardone R, Orioli A, Höller Y, Lochner P, Kunz A, Golaszewski S, *et al*. Central motor and sensory conduction in patients with hepatic myelopathy. Spinal Cord 2014;52(6):439–443. doi:10.1038/sc.2014.61, PMID:24777163.
[3] Zhao H, Liu F, Yue Z, Wang L, Fan Z. Evaluation of mid- and long-term efficacy of shunt limiting for hepatic myelopathy after transjugular intrahepatic portosystemic shunt. Clin Res Hepatol Gastroenterol 2016;40(4):440–446. doi:10.1016/j.clinre.2015.11.004, PMID:26724169.
[4] Cui LB, Ren S, Xi YB, Zeng LL, Chen G, Liu K, *et al*. Motor Cortex Mapping in Patients With Hepatic Myelopathy After Transjugular Intrahepatic Portosystemic Shunt. Acad Radiol 2019;26(5):e38–e46. doi:10.1016/j.acra.2018.06.013, PMID:30076082.
[5] Philips CA, Kumar L, Augustine P. Partial splenic artery embolization for severe hepatic myelopathy in cirrhosis. Hepatology 2018;67(3):1169–1171. doi:10.1002/hep.29597, PMID:29059463.
[6] Li J, Wan S, Wen F, Li Q, Cui Y, Lu Z, *et al*. Liver Transplantation Reverses Hepatic Myelopathy in the Decompensated Phase of Cirrhosis: Case Report and Literature Review. J Clin Transl Hepatol 2024;12(4):436–442. doi:10.14218/JCTH.2023.00487, PMID:38638382.
[7] Nardone R, Höller Y, Storti M, Lochner P, Tezzon F, Golaszewski S, *et al*. Spinal cord involvement in patients with cirrhosis. World J Gastroenterol

2014;20(10):2578–2585. doi:10.3748/wjg.v20.i10.2578, PMID:24627593.
[8] Baccarani U, Zola E, Adani GL, Cavalletti M, Schiff S, Cagnin A, *et al*. Reversal of hepatic myelopathy after liver transplantation: fifteen plus one. Liver Transpl 2010;16(11):1336–1337. doi:10.1002/lt.22149, PMID:21031552.
[9] Liu F, Deng J, Li Z, Huang Z. Hepatic myelopathy improved by embolization of spontaneous portosystemic shunt and partial splenic artery embolization – Literature review. Rev Esp Enferm Dig 2026;118(2):119–120. doi:10.17235/reed.2025.11451/2025, PMID:41020951.
[10] Wang Z, Lv Y, Han G. Embolization of Patent Paraumbilical Vein for the Treatment of Recurrent Hepatic Encephalopathy and Hepatic Myelopathy. Clin Gastroenterol Hepatol 2025;23(11):A31–A32. doi:10.1016/j.cgh.2025.01.023, PMID:40107640.