

Review began 06/19/2026
Review ended 10/12/2026
Published 10/19/2026

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DOI: 10.7759/cureus.118185

Intrahepatic Portosystemic Shunt as a Cause of Recurrent Encephalopathy in a Non-cirrhotic Patient: Successful Combined Transhepatic and Transjugular Embolization

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Abstract

Intrahepatic portosystemic shunts (IPSS) are rare vascular anomalies that can cause recurrent encephalopathy in non-cirrhotic patients. We present a case of a female with recurrent episodes of delirium, in whom initial laboratory tests showed hyperammonemia with preserved hepatic function. Cross-sectional imaging revealed a left-sided IPSS connecting distal branches of the left portal vein to the left hepatic vein. Given her persistent neurocognitive symptoms despite optimal medical therapy, endovascular closure was performed using a dual-access strategy: transhepatic coil embolization of the portal afferent branches, followed by transjugular deployment of an Amplatzer plug in the venous outflow. After the procedure, the patient experienced full resolution of symptoms, with no recurrent neurological episodes during follow-up. This case highlights the importance of considering IPSS as a reversible cause of unexplained encephalopathy and supports the efficacy of combined-access embolization.

Categories: Radiology

Keywords: embolization, encephalopathy, intrahepatic vascular anomaly, non-cirrhotic liver, portosystemic shunt

Introduction

Intrahepatic portosystemic shunts (IPSS) are uncommon vascular communications between branches of the portal and hepatic venous systems [1]. Although most portosystemic shunts develop secondary to cirrhosis, a small proportion occur in non-cirrhotic patients as congenital or spontaneous venous malformations [2]. By diverting portal blood away from hepatic metabolism, these shunts allow ammonia and other gut-derived neurotoxins to enter the systemic circulation, potentially causing encephalopathy despite preserved hepatic function [3]. These lesions may remain subclinical, subsequently presenting with neurocognitive dysfunction secondary to portosystemic encephalopathy [3]. As liver biochemistry is frequently normal in this setting, the diagnosis is easily delayed or misattributed to primary neurological or psychiatric disorders. Imaging plays a central role in recognizing and characterizing these anomalies, and endovascular therapy has become the preferred treatment in symptomatic cases.

The Park classification describes four morphological types of IPSS: a single large communication between the right portal vein and the inferior vena cava (type I); localized peripheral communications between portal and hepatic venous branches within one hepatic segment (type II); an aneurysmal communication between portal and hepatic veins (type III); and multiple diffuse communications involving both hepatic lobes (type IV) [4]. Anatomical characterization guides procedural planning. We present a non-cirrhotic patient with recurrent encephalopathy and an IPSS involving multiple portal feeding branches and a shared hepatic venous outflow. This configuration prompted combined transhepatic and transjugular access to enable selective embolization of the portal inflow and occlusion of the venous outflow.

Case Presentation

A 74-year-old female arrived with marked alteration in her level of consciousness, with a history of recurrent episodes of delirium characterized by fluctuating attention, disorientation, and episodic confusion. Her family denied fever, constitutional symptoms, or exposure to neurotoxic substances. Head CT ruled out acute intracranial pathology, showing only diffuse cortical atrophy and chronic microangiopathic white matter changes. Laboratory evaluation showed marked hyperammonemia, whereas liver enzymes, bilirubin, coagulation parameters, and renal function were all within normal limits (Table 1). This biochemical discordance raised suspicion for a non-cirrhotic etiology of encephalopathy.

How to cite this article

Duarte N, Vasquez Perdomo A, Olarte Bermudez J, et al. (October 19, 2026) Intrahepatic Portosystemic Shunt as a Cause of Recurrent Encephalopathy in a Non-cirrhotic Patient: Successful Combined Transhepatic and Transjugular Embolization. Cureus 18(10): e118185. DOI 10.7759/cureus.118185

Parameter	Pre-procedure value	Post-procedure value	Reference range	Units
Serum ammonia	221.20	56.50	18.70 - 86.90	µg/dL
Aspartate aminotransferase	28.00	28.00	0.00 - 35.00	U/L
Alanine aminotransferase	12.00	16.00	0.00 - 35.00	U/L
Total bilirubin	0.79	0.61	0.30 - 1.00	mg/dL
International normalized ratio	1.1	0.97	0.8 - 1.1	-
Creatinine	0.88	0.85	0.55 - 1.02	mg/dL

TABLE 1: Initial laboratory findings.
U/L: units per liter; mg/dL: milligrams per deciliter; µg/dL: micrograms per deciliter.

Acute intracranial pathology was initially considered; however, head CT showed no acute findings. Marked hyperammonemia with preserved liver biochemical and coagulation parameters prompted further evaluation for a non-cirrhotic cause of encephalopathy. Contrast-enhanced CT demonstrated dilated intrahepatic venous structures forming a left IPSS, with distal branches of the left portal vein draining directly into the hepatic veins (Figure 1). As neurocognitive symptoms persisted despite optimized medical management, the patient was referred for definitive endovascular therapy.



FIGURE 1: Computed tomography images of the abdomen in the axial (A), coronal (B), and sagittal (C and D) planes showing a cluster of dilated venous structures in segments III and IV of the liver secondary to a communication between the left intrahepatic branch of the portal vein (blue arrow) and the hepatic venous drainage (white arrows), forming an intrahepatic portosystemic shunt. The shunt drains into the left hepatic vein (red arrow in C), while its portal inflow arises from branches of the left portal vein (yellow arrow in D), and the afferent flow to this shunt is through left intrahepatic portal vein branches (yellow arrow in D).

Embolization was performed under general anesthesia using combined transjugular and transhepatic access. Right internal jugular venous access enabled a combined dual-access strategy. Right internal jugular venous access allowed selective catheterization of the left hepatic vein, while a left transparietohepatic puncture provided direct access to the left portal venous system. Portal venography confirmed at least three distal left portal vein branches converging toward the left hepatic vein, consistent with a complex IPSS (Figure 2).

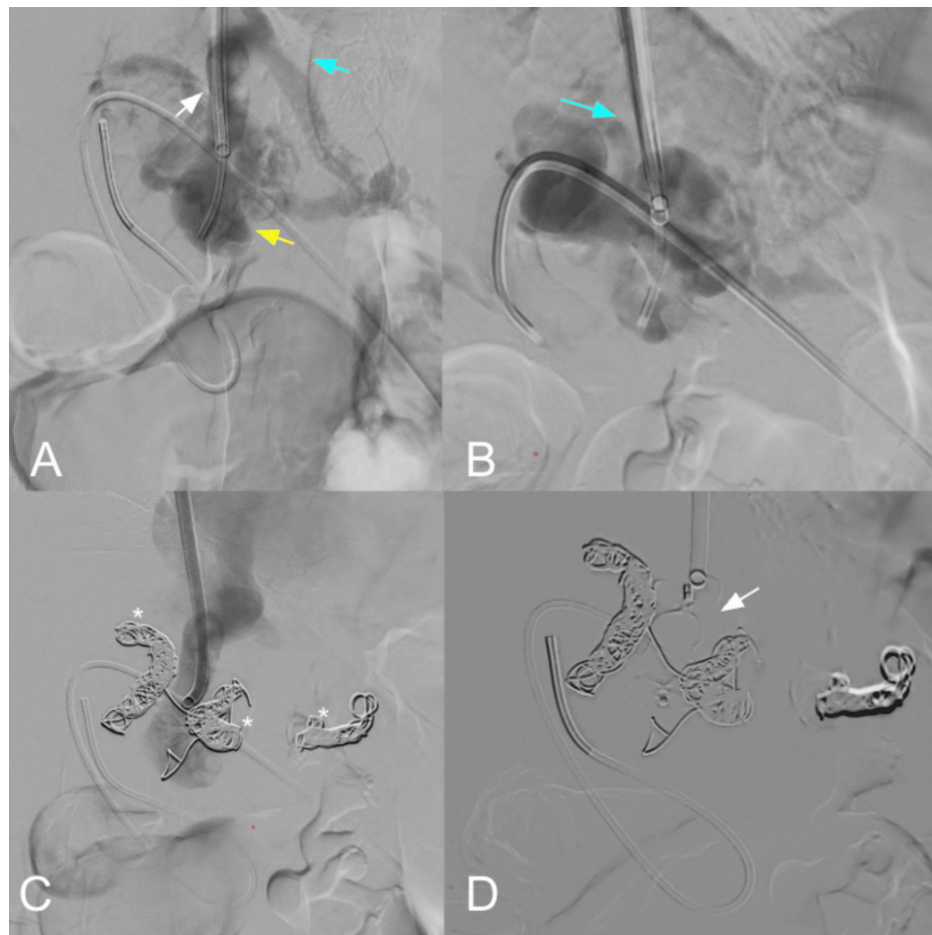


FIGURE 2: Angiography images through transjugular and transhepatic vascular access showing a left intrahepatic portosystemic shunt with multiple dilated veins between the left intrahepatic branch of the portal vein (yellow arrow in A) and the middle (white arrow) and left hepatic veins (blue arrow). Image C shows multiple coils occluding the afferent branches of the left portal vein to the portosystemic shunt (white asterisk) deployed through the transhepatic portal access. Image D shows occlusion achieved by placing an Amplatzer plug through the transjugular sheath (white arrow).

Through the transhepatic approach, each portal afferent branch was superselectively catheterized and embolized with multiple coils until complete vascular occlusion was achieved. Subsequently, through the transjugular access, an Amplatzer vascular plug was deployed in the left hepatic vein, at the level of the aneurysmal sac associated with the shunt and the distal portion of the left hepatic vein, to occlude the venous outflow. Completion venography demonstrated satisfactory exclusion of the shunt.

Hemostasis of the transparietohepatic tract was achieved using gelatin sponge embolization, and the jugular access was closed with manual compression. No procedural or immediate post-procedural complications occurred. During follow-up, the patient remained asymptomatic, showed normalization of ammonia levels, and experienced no further episodes of disorientation or neurological dysfunction, suggesting that her prior dissociative episodes likely corresponded to transient ammonia elevations related to the shunt.

At 12 months, contrast-enhanced abdominal MRI demonstrated post-embolization changes with susceptibility artifacts in hepatic segments II and III. Liver morphology and signal intensity remained normal, and no additional shunt-related abnormalities were reported.

Discussion

Hepatic encephalopathy (HE) is a brain dysfunction classified into three types: type A, due to acute liver

failure; type B, secondary to non-cirrhotic portosystemic shunts; and type C, associated with cirrhosis [1]. Therefore, non-cirrhotic portosystemic shunts occur when portal blood bypasses the liver through a shunt, which is usually congenital, spontaneous, or acquired after trauma, surgery, or liver biopsy. From a pathophysiological standpoint, shunts lead to neurotoxin accumulation despite preserved hepatic function, and patients may remain asymptomatic for years, presenting later in life with fluctuating cognitive impairment, confusion, or overt encephalopathy that can mimic psychiatric disorders.

Portosystemic shunts present diverse morphological variants, including single prominent tubular tracts, aneurysmal-type communications, and multifocal peripheral patterns. The morphological characteristics of the shunt are clinically relevant, as they influence both symptom severity and therapeutic planning [5]. On the other hand, assessment of hyperammonemia is essential to evaluate its contribution to neuroinflammation, neurotransmitter dysregulation, and hemodynamic alterations. Nevertheless, serum ammonia levels do not consistently correlate with the severity of neurological manifestations [2,3]. In this context, cross-sectional imaging plays a key role in diagnosis, providing precise anatomical delineation and classification of the shunt. Among imaging modalities, knowing the shunt's size, location, and anatomical complexity is critical for treatment planning. The diagnosis is typically established using contrast-enhanced CT or MRI, including magnetic resonance portography. Large shunts, generally defined as those measuring ≥ 8 mm in diameter, are considered suitable candidates for embolization using various endovascular techniques [5,6].

Previous reports describe similar presentations of IPSS in older adults without cirrhosis. Brader and Kim reported a 73-year-old male with confusion, ataxia, and hyperammonemia despite treatment with lactulose and rifaximin. Transhepatic embolization using two Amplatzer vascular plugs and four coils resulted in complete resolution of his symptoms [7]. Bayona Molano et al. described two patients with noncirrhotic IPSS and encephalopathy. Particularly relevant to our case was a 72-year-old male with three communications between the left portal vein and the left hepatic vein, which were treated with Amplatzer vascular plugs through a transjugular approach. His encephalopathy resolved, ammonia levels normalized, and Doppler ultrasonography showed no shunt recurrence at six months [8].

In these patients, cross-sectional imaging and angiography are essential to identify the shunt anatomy, define the portal inflow and systemic venous outflow, and guide treatment planning. Reported therapeutic approaches include transcatheter embolization using coils, vascular plugs, detachable balloons, or combined devices, with most cases showing clinical improvement or resolution of encephalopathy after successful shunt occlusion [9,10]. Similarly, our patient had multiple left portal venous afferents; however, the associated aneurysmal venous component was treated using combined transjugular and transhepatic access with coils and a vascular plug.

Treatment of HE type B requires combined medical and procedural strategies. Although medical therapy with ammonia-lowering strategies such as lactulose and rifaximin remains an important initial approach, shunt occlusion should be considered in selected symptomatic patients with large or anatomically significant portosystemic communications, particularly when neurological symptoms persist or recur despite optimized medical management [11].

Interventional radiology techniques, such as endovascular shunt closure, have emerged as a safe and effective option [2,3,12,13]. Usually, the most frequently used method combines Amplatzer vascular plugs with coils, enabling precise placement and controlled deployment. In the present case, dual-access approaches (transhepatic and transjugular) are pivotal for assessing simultaneous treatment of both the portal inflow and venous outflow, reducing the risk of residual or recurrent shunting [5]. This strategy also carries fewer risks than alternative embolization techniques or surgical occlusion [2,3,11].

Potential complications of endovascular shunt closure include device migration, portal or mesenteric venous thrombosis, and new-onset portal hypertension following occlusion. These risks highlight the importance of careful anatomical and hemodynamic assessment before closure and clinical and Doppler ultrasound surveillance afterward [13]. For this reason, it is important to recognize IPSS as a reversible cause of HE in non-cirrhotic patients. Endovascular intervention can achieve full symptom resolution, prevent recurrent neurological deterioration, and significantly improve quality of life.

Conclusions

IPSS represent an uncommon entity and may be misinterpreted as a primary neurological or psychiatric condition. However, they constitute an important and reversible cause of recurrent encephalopathy in non-cirrhotic patients. For this reason, this case highlights the role of cross-sectional imaging in establishing the diagnosis and guiding management. Endovascular treatment using a combined transhepatic and transjugular approach allows precise control while minimizing the risk of residual shunting. Successful embolization can lead to complete symptom resolution, emphasizing that timely recognition and intervention may prevent further neurological impairment and significantly improve patient quality of life.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Comité Corporativo de Ética en Investigación de la Fundación Santa Fe de Bogotá (Corporate Research Ethics Committee of Fundación Santa Fe de Bogotá) issued approval CCEI-19260-2026.

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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