



# Over Tipping: Understanding Cerebral Edema as a Complication of TIPS in a Cirrhotic Patient

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## Abstract

Transjugular Intrahepatic Portosystemic Shunt (TIPS) is an established treatment for complications of decompensated liver cirrhosis causing portal hypertension. Hepatic encephalopathy is a frequent complication of TIPS; however, cerebral edema, which is more commonly seen in acute liver failure, has rarely been reported in chronic liver failure or post-TIPS. There are multiple proposed mechanisms that may contribute to cerebral edema after TIPS placement, but few established risk factors or treatment options exist. Here, we report a case of a patient with NASH related cirrhosis who underwent TIPS to control recurrent variceal bleeding and subsequently developed cerebral edema.

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## Introduction

In patients with chronic liver disease, Transjugular Intrahepatic Portosystemic Shunt (TIPS) is often used to treat complications of portal hypertension by decreasing the portosystemic gradient. However, the benefits of TIPS must be weighed against the most common post-procedural complication of hepatic encephalopathy and a related rare but deadly complication - cerebral edema. Here, we report a case of a patient with Non-Alcoholic Steato Hepatitis (NASH) related cirrhosis who underwent TIPS to control recurrent variceal bleeding and subsequently developed cerebral edema.

## Case report

The patient is a 56-year-old female with NASH cirrhosis without a history of encephalopathy and not on lactulose or rifaximin who presented with melena and hematemesis. Labs were notable for hemoglobin 11.4 g/dL, platelets 66 K/uL, bilirubin 1.3 mg/dL, INR 1.3, AST 58 U/L, and ALT 43 U/L. An echocardiogram showed an ejection fraction of 55-60% and no right heart dysfunction. Upper endoscopy demonstrated grade 2 esophageal varices, which were banded, as well as grade 1 gastric varices. Given persistent bleeding, the decision was made to proceed with TIPS. The Hepatic Portosystemic Gradient (HPVG) was



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reduced from 26 mmHg to 6 mmHg. 1 hour after the procedure, the patient's ammonia was noted to be 260ug/dL. 24 hours later, the patient had deterioration in mental status and ammonia was 670 ug/dL. The patient returned to the interventional suite for TIPS revision. The revised HPVG was 11 mmHg and the patient was started on continuous veno-venous hemofiltration. The patient then developed myoclonic seizures and subsequent computed tomography of her head revealed cerebral edema. She was treated with hypertonic saline, mannitol, bolt placement, and complete embolization of her TIPS. Serum ammonia downtrended, but neurologic function remained significantly impaired.

### Discussion

TIPS remains a common solution for patients with refractory gastrointestinal bleeding secondary to liver disease. Although cerebral edema can be seen in patients with acute liver failure, it is rarely reported in chronic liver failure patients or cirrhotic patients who undergo TIPS. This complication was first reported in four patients in 1997 [1], but its mechanism and how to prevent it remain unclear. This case report adds to existing evidence that cerebral edema can occur in patients with cirrhosis after undergoing TIPS. Although guidelines [2], recommend reducing the HPVG to  $< 12$  mmHg or  $\geq 20\%$  from baseline, there are no studies that examine the relationship between post-TIPS HPVG and risk for cerebral edema. In our patient, and those reported in prior case reports, the final HPVG was between 2 and 7 mmHg while the change in HPVG was between 69%-77% [3, 4]. Furthermore, ammonia levels were checked only after a significant change in baseline mental status. Given that hyperammonemia causing accumulation of astrocytic glutamine and subsequent dysfunction of ion transporters involved in cellular

volume regulation is hypothesized to lead to cerebral edema [5], there may be a role in implementing a protocol to monitor serial ammonia levels before and after TIPS. Further research into goal HPVG, the maximal percentage change in HPVG, serial ammonia levels, and cellular mechanisms of cerebral edema post TIPS could help prevent future patients from suffering this devastating complication.

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