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Case Report

Portosystemic Encephalopathy in a Noncirrhotic Patient Treated by Vascular Plug Embolization of Mesoiliac Shunt

Abstract

Mesoiliac shunts in the absence of liver cirrhosis are rarely reported as a cause of hyperammonemia with encephalopathy. Here, we report the case of a 65-year-old female patient with no history of liver disease, who developed significant confusion. Workup showed hyperammonemia and encephalopathy due to spontaneous mesoiliac shunt with no imaging signs of portal hypertension. Liver biopsy showed no evidence of cirrhosis. The shunt was occluded using a vascular plug, resulting in complete resolution of symptoms with no recurrence at 8-year follow-up.

Keywords: *Embolization, hepatic encephalopathy, mesoiliac shunt, portal hypertension, portosystemic shunts, shunt occlusion*

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Introduction

Portosystemic shunts without liver disease could be potentially induced by abdominal trauma, prior surgery, and postnatal viral or hepatotoxic injuries. Congenital shunts are related to incomplete involution of the omphalomensenteric venous system.[1]

Case Report

A 65-year-old female patient presented to the emergency department with acute confusion, lethargy, dysarthria, nausea, and vomiting. Brain computed tomography (CT) showed no acute intracranial lesions. Serum ammonia level was elevated at 119 µmol/L (normal range: 11–32 µmol/L). There was slight elevation in transaminases (aspartate aminotransferase [AST] 59 U/L and alanine aminotransferase [ALT] 56 U/L) with normal total bilirubin (13 µmol/L).

Abdominal CT revealed a patent portal vein and an enlarged superior mesenteric vein with a 10-mm portosystemic shunt connecting the superior mesenteric vein to the right common iliac vein [Figures 1 and 2].

Through left common femoral vein access, the shunt was catheterized [Figure 3] with 65-cm long, 7-Fr sheath that was used for the deployment of Amplatzer Vascular Plug (18 mm × 14 mm) near the shunt inflow. This resulted in complete remission of symptoms and normalization of transaminases (AST 19 U/L and ALT 14 U/L) and ammonia level at 44 µmol/L without additional medical therapy. The patient remained free of symptoms after 8-year follow-up.

Discussion

Liver cirrhosis is the most common cause of hepatic encephalopathy (HE). It leads to portal-systemic venous shunts, in which the neurotoxic substances, such as ammonia, bypass the liver into the systemic circulation, affecting the brain.[2] However, there are various less frequent causes of high ammonia level not associated with liver disease such as high protein diet, constipation, and gastrointestinal bleeding.[2-5]

In our case, all the common causes of liver disease and causes of hyperammonemia in the absence of liver disease were excluded, and porto-systemic shunt was diagnosed on the basis of CT findings.

Interventional radiology treatment was first attempted by Nagino *et al*. in 1992 using fibrin glue to close a large shunt between the inferior mesenteric vein and the internal iliac vein; this offered amelioration of hypoproteinemia and reduction of serum ammonia levels without any complications.[3]

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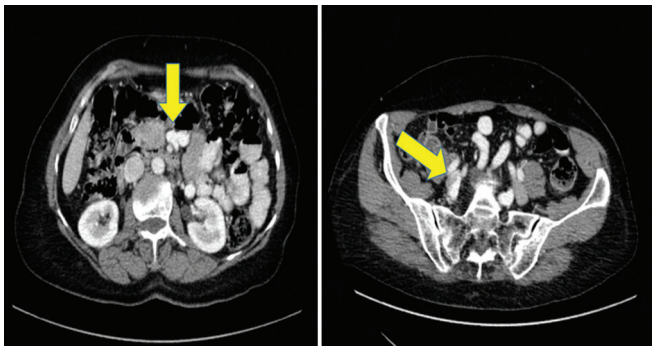

Figure 1: Computed tomography images of the mesoiliac shunt at inflow (superior mesenteric vein) and outflow (right common iliac vein)

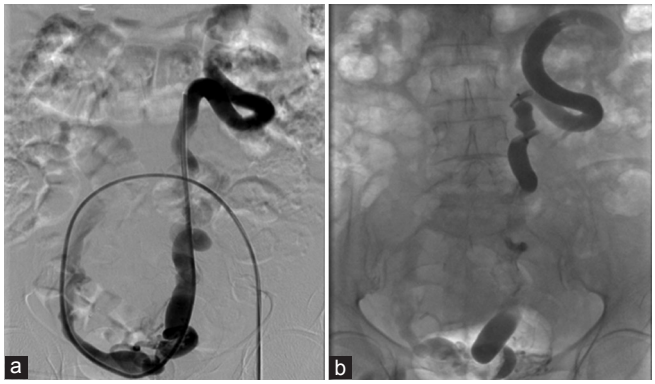

Figure 2: Angiographic images of the mesoiliac shunt before (a) and after intervention (b)

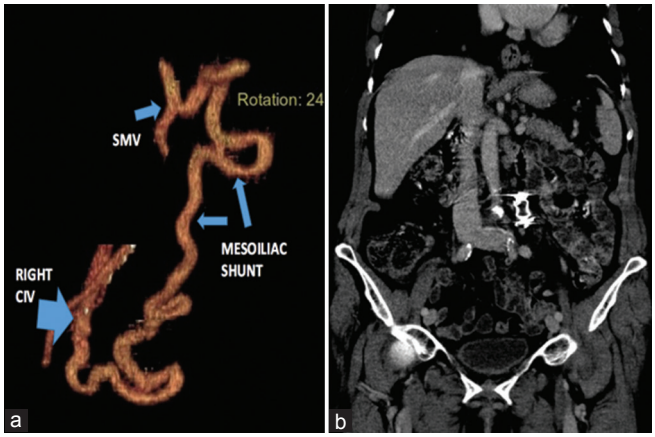

Figure 3: Three-dimensional reconstructed computed tomography image of the mesoiliac shunt (a) and postintervention coronal computed tomography image (b)

Other similar cases of noncirrhosis-related HE reported in literature were treated with percutaneous occlusion using coils to treat inferior mesenteric-caval shunt in patients with disturbed consciousness and Amplatzer plug for spontaneous spleno-renal shunt in patients presented with confusion and violent behavior. Plug and coils were used in combination to close inferior mesenteric-caval shunt in patients with relapsing confusion, lethargy, and dysartheria.[4,5]

Endovascular approaches represented an alternative treatment option to the usual surgical ligation.[6]

Conclusion

Extrahepatic portosystemic shunts are rare, but should be considered in patients presenting with altered consciousness and an elevated ammonia level in the absence of liver disease. In portosystemic shunts associated with encephalopathy, endovascular embolization may provide complete remission.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initial will not be published, and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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