Clinics and Research in Hepatology and Gastroenterology (2015) xxx, xxx–xxx

Available online at ScienceDirect www.sciencedirect.com

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ORIGINAL ARTICLE

Evaluation of mid- and long-term efficacy of shunt limiting for hepatic myelopathy after transjugular intrahepatic portosystemic shunt

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Summary

Background: Hepatic myelopathy (HM) is a rare condition caused by severe liver dysfunction, and may be secondary to transjugular intrahepatic portosystemic shunt (TIPS). This study aimed to evaluate the mid- and long-term clinical efficacy of TIPS reduction (TIPSR) for treatment of HM secondary to TIPS.

Methods: Patients who underwent TIPS ($n = 1325$) for severe portal hypertension between August 2002 and August 2013 at the Affiliated Beijing Millennium Monument Hospital, Capital Medical University (Beijing, China) were reviewed. During follow-up, 22 patients were diagnosed with HM, and 12 underwent TIPSR. Patients were evaluated using the Barthel index (daily activities), the Lovette's Six Classification (lower extremity muscle strength), and the Fugl-Meyer assessment (FMA; lower extremity activity). Hepatic encephalopathy grade was used to assess the severity of clinical symptoms.

Results: TIPSR did not affect portal vein pressure (31.6 ± 6.2 vs. 33.3 ± 7.9 mmHg, $P = 0.55$). Blood ammonia levels were 77.9 ± 17.9 mmol/L before TIPSR and 77.9 ± 14.8, 73.5 ± 21.5, 59.5 ± 14.5, and 52.0 ± 16.5 mmol/L at 1, 3, 6, and 12 months ($P < 0.05$ for 6 and 12 months vs. baseline). The Barthel index was improved 6 months after TIPSR (42.1 ± 10.5 vs. 45.0 ± 8.8, $P < 0.05$), while FMA was improved 3 months after TIPSR only (24.6 ± 3.2 vs. 25.5 ± 3.2, $P < 0.05$). Lovette's Six Classification was improved 12 months after TIPSR (2.1 ± 0.7 vs. 2.8 ± 0.9,

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http://dx.doi.org/10.1016/j.clinre.2015.11.004

Please cite this article in press as: Zhao H, et al. Evaluation of mid- and long-term efficacy of shunt limiting for hepatic myelopathy after transjugular intrahepatic portosystemic shunt. Clin Res Hepatol Gastroenterol (2015), http://dx.doi.org/10.1016/j.clinre.2015.11.004

$P < 0.05$). After TIPS, hepatic encephalopathy grade was I ($n = 3$), II ($n = 6$), III ($n = 2$), or IV ($n = 1$), and was I ($n = 8$), II ($n = 1$), or III ($n = 1$) at 6 months.
Conclusion: TIPSR can improve the mid- and long-term symptoms of HM secondary to TIPS.

Introduction

Hepatic myelopathy (HM) is a rare condition caused by severe liver dysfunction, occurring in 2—4% of liver diseases. Age at onset is 11—69 years old and males are predominantly affected [1—3]. HM may occur in acute and chronic liver failure, end-stage cirrhosis, and after large diameter portosystemic shunt surgery. The limbs show slowly progressive symmetric spastic paralysis without sensory loss [2]. The disease is slowly progressive, but the patient will ultimately become wheelchair-bound. HM may appear after transjugular intrahepatic portosystemic shunt (TIPS) used to manage portal hypertension in cirrhotic patients [4]. Hepatic encephalopathy (HE) almost always precedes the appearance of HM as both diseases share the same mechanisms [4,5]. HE and HM are associated with disorganized proliferation of astrocytes [4]. Conservative treatments are usually successful for HE, but not for HM [6].

HM has a poor prognosis, and patients with HM often die from liver failure, sepsis, hepatorenal syndrome, and other complications [7]. There is no accepted treatment for HM. Timely and effective surgical treatments such as liver transplantation could be a final therapeutic approach for HM [3,7—10], but treatments are controversial [11,12]. Occurrence of TIPS-associated HM may be associated with large-diameter shunts through which intestinal toxins enter directly into the systemic circulation without being detoxified in the liver, causing injuries to the spinal cord [2,13].

Therefore, TIPS may be improved by placing a reducing stent in the shunt (TIPS reduction [TIPSR]) to reduce the shunt diameter and the amount of intestinal bacterial toxins that enters the systemic circulation while preserving the shunt. This study aimed to evaluate the mid- and long-term clinical efficacy of TIPSR for the treatment of HM after TIPS.

Materials and methods

Patients

The charts of the patients who underwent TIPS ($n = 1325$) for severe portal hypertension between August 2002 and August 2013 at the Affiliated Beijing Millennium Monument Hospital, Capital Medical University (Beijing, China) were reviewed. Consequently, 22 (1.66%, 22/1325) patients were identified as having developed HM after TIPS. These patients were diagnosed by the same team of neurologists. Data were retrieved and retrospectively analyzed. Twelve patients underwent TIPSR, and all 12 were included in the present study. The present study was approved by the local ethics committee. The need for individual consent was waived by the committee.

Patients were diagnosed with myelopathy and damaged corticospinal tract after consultation with neurologists. Diagnostic criteria included:

- history of chronic liver disease and formation of a natural collateral circulation or portosystemic stent;
- progressive spastic paraplegia without obvious atrophy and shallow sensory dysfunction;
- recurrent or transient HE;
- liver dysfunction, normal cerebrospinal fluid, and serum blue copper protein, without pigmented corneal ring;
- without spinal cord space-occupying mass, multiple sclerosis, or hepatolenticular degeneration [3,14,15].

Pre-TIPSR imaging and laboratory

The twelve patients underwent preoperative magnetic resonance imaging (MRI), computed tomography (CT), and ultrasound. Imaging suggested cirrhosis, portal hypertension, esophageal and/or gastric varices, ascites, and significant liver atrophy (Fig. 1).

TIPSR approach

The technique for TIPSR is almost the same as for TIPS. Patients underwent TIPSR using a new stent. The circulation was entered via the right jugular vein using a catheter. The guide wire was placed into the intrahepatic vein or inferior vena cava, and then into the TIPS. The absence of tributary blood flow was confirmed by angiography. Portal blood pressure was measured. The reducing stent was then placed, with the small diameter end in the distributary vessel and the large diameter end in the vena cava or inferior hepatic vein. The aim of the procedure was to increase the portosystemic gradient. Stents with a diameter of 12 mm (12×80 mm ev3 stent, ev3 Endovascular Inc., Plymouth, MN, USA), 10 mm (7—10×40 mm ev3 Protégé Rx taper stent, or 10×80 mm eV3 stent, ev3 Endovascular Inc., Plymouth, MN, USA), and 8 mm (6—8×40 mm eV3 Protégé Rx taper stent, ev3 Endovascular Inc., Plymouth, MN, USA) (Fig. 2) were placed inside the previous stent. A Siemens digital subtraction angiography system (Siemens AXIOM ArtisdTA, Siemens, Erlangen, Germany) was used to measure the inner diameter of the shunt.

Blood pressure was consistently measured using a pigtail catheter placed in the main portal vein through the TIPS channel. The catheter was connected to a 3-way valve. The perpendicular end of the valve was connected to the intravenous infusion catheter to make a right angle for the pressure test. Sterile water was injected through the third

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