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## Ligation of huge spontaneous porto-systemic collaterals to avoid portal inflow steal in adult living donor liver transplantation: A case-report



Mohamed Elshobary<sup>a</sup>, Ahmed Shehta<sup>a,\*</sup>, Tarek Salah<sup>a</sup>, Ahmed Mohamed Sultan<sup>a</sup>, Usama Shiha<sup>b</sup>, Ahmed Nabieh Elghawalby<sup>a</sup>, Ahmed Monier<sup>a</sup>, Mohamed Elsadany<sup>a</sup>, AmrYassen<sup>a</sup>, Omar Fathy<sup>a</sup>, Mohamed Abdel Wahab<sup>a</sup>

<sup>a</sup> Liver Transplantation Unit, Gastroenterology Surgical Centre, College of Medicine, Mansoura University, Egypt

<sup>b</sup> Radiology department, Gastroenterology Surgical Centre, Mansoura University, Egypt

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## ABSTRACT

**INTRODUCTION:** In adult living donor liver transplantation (LDLT), maintenance of adequate portal inflow is essential for the graft regeneration. Portal inflow steal (PFS) may occur due to presence of huge spontaneous porto-systemic collaterals. A surgical procedure to increase the portal inflow is rarely necessary in adult LDLT.

**PRESENTATION:** A 52 years male patient with end-stage liver disease due to chronic hepatitis C virus infection. Preoperative portography showed marked attenuated portal vein and its two main branches, patent tortuous splenic vein, multiple splenic hilar collaterals, and large lieno-renal collateral. He received a right hemi-liver graft from his nephew. Exploration revealed markedly cirrhotic liver, moderate splenomegaly with multiple collaterals and large lieno-renal collateral. Upon dissection of the hepato-duodenal ligament, a well-developed portal vein could be identified with a small mural thrombus.

The recipient portal vein stump was anastomosed, in end to end fashion, to the graft portal vein. Doppler US showed reduced portal vein flow, so ligation of the huge lieno-renal collateral that allows steal of the portal inflow. After ligation of the lieno-renal collateral, improvement of the portal vein flow was observed in Doppler US.

**DISCUSSION:** There is no accepted algorithm for managing spontaneous lieno-renal shunts before, during, or after liver transplantation, and evidence for efficacy of treatments remains limited. We report a case of surgical interruption of spontaneous huge porto-systemic collateral to prevent PFS during adult LDLT. **CONCLUSION:** Complete interruption of large collateral vessels might be needed as a part of adult LDLT procedure to avoid devastating postoperative PFS.

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### 1. Introduction

Living donor liver transplantation (LDLT) has been a useful therapeutic option for end-stage liver disease because of the shortage of deceased donor liver grafts [1].

In adult LDLT, maintenance of adequate portal inflow is essential for the graft regeneration. Portal inflow steal (PFS) may occur

**Abbreviations:** LDLT, living donor liver transplantation; PFS, portal inflow steal; DDLT, deceased donor liver transplantation; MELD, model for end stage liver disease; US, ultrasonography; CT, computed tomography; GRWR, graft weight to recipient weight ratio; TIPS, transjugular intrahepatic porto-systemic shunt.

\* Corresponding author at: Gastroenterology Surgical center, Gehan Street, Mansoura, Postal code: 35516, Egypt.

E-mail addresses: [ahmedshehta@mans.edu.eg](mailto:ahmedshehta@mans.edu.eg), [dr.ahmedshehta@yahoo.com](mailto:dr.ahmedshehta@yahoo.com) (A. Shehta).

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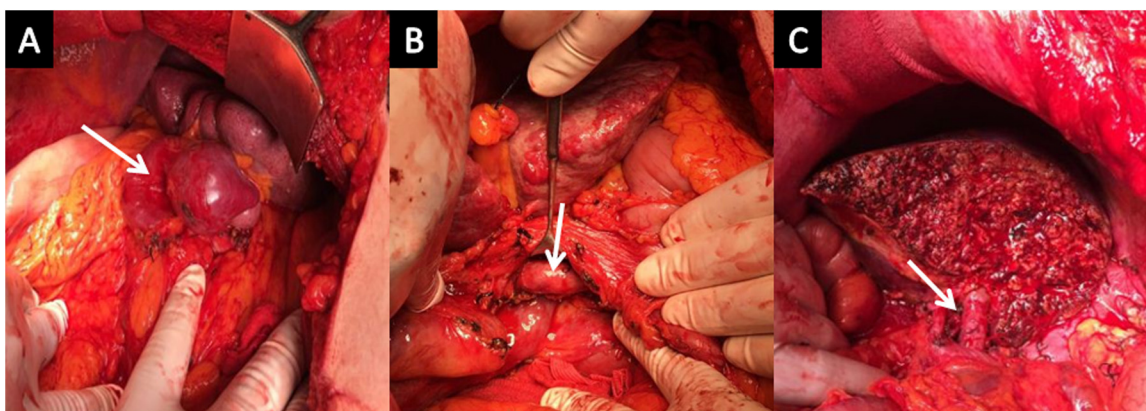
due to presence of huge spontaneous porto-systemic collaterals. If the portal inflow to the liver graft is inadequate after adult LDLT, through persistent spontaneous porto-systemic collaterals, post-transplant impairment of the graft regeneration and eventually graft failure would occur [2].

A surgical procedure to increase the portal inflow is rarely necessary in adult LDLT. Unlike pediatric LDLT where a ligation of the porto-systemic collaterals is sometimes necessary to maximize the portal inflow to a relatively large graft [3], and deceased donor liver transplantation (DDLTL) where a sufficient liver vascular bed is usually present [4].

We report a case of prophylactic surgical interruption of spontaneous huge porto-systemic collateral to prevent PFS during adult LDLT procedure. This work has been reported in line with the SCARE criteria [5].



**Fig. 1.** Preoperative CT portography showing markedly attenuated portal vein (thin arrow) with huge lieno-renal collateral (thick arrow).



**Fig. 2.** Operative photos showing; A- Huge lieno-renal collateral (arrow), B- After dissection of hepatoduodenal ligament and exposure of healthy portal vein (arrow), C- After completion of graft implantation (arrow at portal vein anastomosis).

## 2. Case presentation

52 years male patient with end-stage liver disease due to chronic hepatitis C virus infection. He His preoperative Child-Pugh score was 7 (class B), and his model for end stage liver disease (MELD) was 13. He was planned for living donor liver transplantation.

Preoperative abdominal ultrasonography (US) showed marked cirrhotic liver with no ascites, moderate enlargement of the spleen, and dilated splenic vein with multiple hilar collaterals. Doppeler US showed marked attenuated portal vein along its course (5 mm) with absent flow inside and large lieno-renal shunt siphoning the portal circulation.

Preoperative triphasic abdominal computed tomography (CT) showed shrunken cirrhotic liver, marked attenuated portal vein, moderate enlarged uniform spleen, and dilated splenic vein with multiple hilar collaterals. CT portography showed marked attenuated portal vein and its two main branches, dilated patent tortuous splenic vein, normal patent superior mesenteric vein, multiple dilated splenic hilar collaterals, and large lieno-renal collateral (Fig. 1).

He received an ABO identical right hemi-liver graft without the middle hepatic vein from his nephew. The actual graft weight was 983 g and graft weight to recipient weight ratio (GRWR) was 1.06.

Exploration of the patient revealed markedly cirrhotic liver, moderate splenomegaly with multiple collaterals and large lieno-renal collateral. Upon dissection of the hepato-duodenal ligament, a well-developed portal vein could be identified with a small mural

thrombus, unlike the preoperative CT findings (Fig. 2). Thrombectomy was done, and the forward flow from the portal vein stump was evaluated before the implantation of the graft and found to be sufficient for the reperfusion of the graft.

The graft had a single dominant right hepatic vein, and was anastomosed to the recipient right hepatic vein with venoplasty.

The recipient portal vein stump was anastomosed, in end to end fashion, to the graft portal vein. Doppler US showed reduced portal vein flow (52 cm/s). The decision was to ligate the huge lieno-renal collateral that allowed steal of the portal inflow. After ligation of the lieno-renal collateral, improvement of the portal vein flow was observed in Doppler US (92 cm/sec).

Then arterial reconstruction was done between the recipient right hepatic artery and graft right hepatic artery. Biliary reconstruction was done between the recipient right and left hepatic ducts to the graft right anterior and posterior hepatic ducts, respectively. Completion Doppler US showed sound anastomoses and adequate portal inflow.

The postoperative course was uneventful. Regular follow-up Doppler US showed adequate portal blood flow and he was discharged 15 days after transplantation. 6 month after liver transplantation, the patient is doing well with normal liver function tests (serum albumin: 4.5 gm/dL, serum bilirubin: 0.8 mg/dL, serum alanine aminotransferase: 46 IU/ml, serum aspartate aminotransferase: 38 IU/ml, serum international normalized ratio: 1, serum alkaline phosphatase: 5 KAU), and adequate Doppler US examination. Follow-up triphasic abdominal CT showed normal liver graft,

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