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# Development of extensive inferior vena cava thrombosis due to the ligation of a large mesenteric-caval shunt during liver transplantation: A case report



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#### ABSTRACT

INSTRUCTION: Inferior vena cava (IVC) thrombosis can be a life-threatening complication after liver transplantation (LT). Although this complication is usually related to technical problems associated with vascular anastomosis, we report a case of IVC thrombosis which developed from a ligated large mesenteric-caval shunt.

PRESENTATION OF CASE: A 35-year-old man underwent LT from a brain-dead donor for primary sclerosing cholangitis. Enhanced computed tomography (CT) before LT showed a huge collateral vessel of the inferior mesenteric vein (IMV) draining into the infra-renal IVC directly. To obtain sufficient portal vein (PV) flow, the dilated IMV collateral was ligated. A routine Doppler ultrasound study on post-operative day 1 showed thrombus inside the infra-hepatic IVC. Enhanced CT showed that this thrombus originated from a ligated collateral vessel of the IMV and extended into the IVC. He was hemodynamically stable and liver function was consistently stable. The size of IVC thrombus slowly reduced and he is currently in good condition without any symptoms.

DISCUSSION: To obtain adequate PV flow, ligation of a major PSS at the time of LT has been suggested. However, where it should be occluded has not been discussed. We should occlude a mesenteric-caval shunt not only at the upper side, but at the IVC side, based on findings from the current case.

CONCLUSION: To obtain appropriate PV flow toward a liver graft, occlusion of portosystemic shunts during LT is recommended. However, the position of ligation should be carefully considered to avoid extension of thrombus to major vessels.

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### 1. Introduction

Extensive venous collateral formation is one of the characteristics of portal hypertension [1]. When the shunts are large and well established, spontaneous closure may be delayed or not occur even after liver transplantation (LT). In addition, under conditions of increased hepatic graft resistance, including rejection, ischemic injury, and portal hypertension, reopening of shunts has been

Abbreviations: CT, computed tomography; DDLT, deceased donor liver transplantation; IMV, inferior mesenteric vein; IVC, inferior vena cava; LT, liver transplantation; POD, post-operative day; POY, post-operative year; PSS, portosystemic shunt; PV, portal vein.

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described [1]. Moreover, appropriate portal vein (PV) flow toward the liver graft at transplantation is important for successful LT. Therefore, many authors have recommended occlusion of spontaneous portosystemic shunts (PSSs) at the time of LT [2,3]. However, as a note of caution, we report a case of LT complicated by extensive inferior vena cava (IVC) thrombosis caused by ligation of a large PSS.

Postoperative IVC thrombosis is rare, but can be a lethal complication in an LT patient because of deteriorating liver and kidney function, unstable cardiac output because of decreased venous return, pulmonary embolism, and lower extremity edema [4,5]. This complication is usually related to technical problems associated with vascular anastomosis [4,5]. There are no previous case reports of IVC thrombosis that developed from a ligated inferior-mesenteric-caval shunt.

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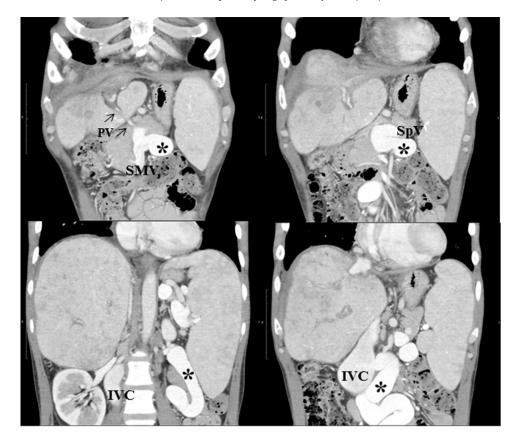
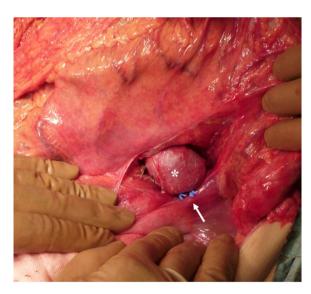


Fig. 1. Preoperative coronal computed tomography scan at 4months prior to liver transplantation. The PV trunk (arrows) was sclerotic and narrowed. A huge mesenteric-caval shunt (\*) originating from the inferior mesenteric vein directly drained into the infra-renal IVC. PV, portal vein; SMV, superior mesenteric vein; SpV, splenic vein; IVC, inferior vena cava.

# 2. Presentation of case

A 32-year-old man was referred for LT for end-stage liver cirrhosis secondary to primary sclerosing cholangitis. Because he had no suitable living donor candidates, he had been on the deceased donor liver transplantation (DDLT) list for more than 3 years. The patient's decompensated features included jaundice, esophageal varices, and repeated episodes of hepatic encephalopathy. During his waiting period, his collateral vessels including an inferior-mesenteric-caval shunt, considerably developed. An enhanced computed tomography (CT) scan at 4 months prior to his DDLT showed a huge mesenteric-caval shunt originating from the inferior mesenteric vein (IMV) and directly draining into the infra-renal IVC (Fig. 1).

He underwent DDLT at the age of 35. The patient's Model for End-Stage Liver Disease score at the time of LT was 25. A whole liver graft was obtained from a 52-year-old female brain-dead donor. Total hepatectomy of the recipient was performed uneventfully. Reconstruction of hepatic veins was performed using the piggyback technique. Because of the unexpected short supra-hepatic IVC of the graft, hepatic vein plasty and a longitudinal incision of the IVC were performed on the back table. After implantation, graft hepatic veins were reconstructed with end-to-side cavocavostomy. Due to the narrowed PV of the recipient, PV reconstruction was performed using a donor iliac vein interpositional graft. In addition to this PV interposition vein graft reconstruction, the dilated IMV collateral was ligated at the level of ligament of Treitz to obtain sufficient portal flow (Fig. 2). Hepatic arterial reconstruction was performed between the proper hepatic artery from the recipient and the common hepatic artery of the graft. Biliary reconstruction was performed by hepaticojejunostomy with a 5 Fr external stent



**Fig. 2.** Intraoperative findings of the recipient. Ligation (arrow) of the dilated IMV-caval shunt (\*).

tube. The cold ischemic, warm ischemic, and total operative times were 69,937, and 1002 min, respectively. Blood loss was 12,039 ml.

A standard immunosuppression protocol using calcineurin inhibitor, mycophenolate mofetil, and steroid was applied. Immediate graft function was excellent, but a routine Doppler ultrasound study on post-operative day (POD) 1 showed thrombus inside the infra-hepatic IVC (Fig. 3A). A CT scan on POD 1 showed that this thrombus originated from the location of the ligated collateral ves-

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