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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.

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 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.

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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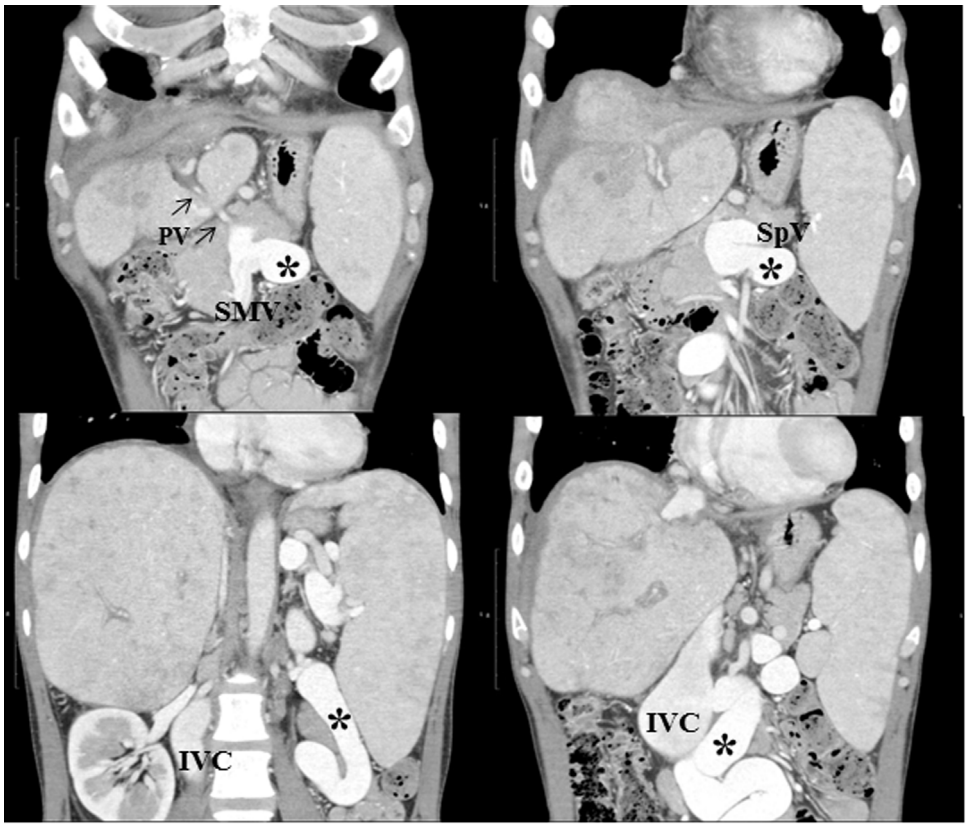

Fig. 1. Preoperative coronal computed tomography scan at 4months prior to liver transplantation. The PV trunk (arrows) was sclerotic and narrowed. A huge mesenteric-caval shunt (*) originating from the inferior mesenteric vein directly drained into the infra-renal IVC. PV, portal vein; SMV, superior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

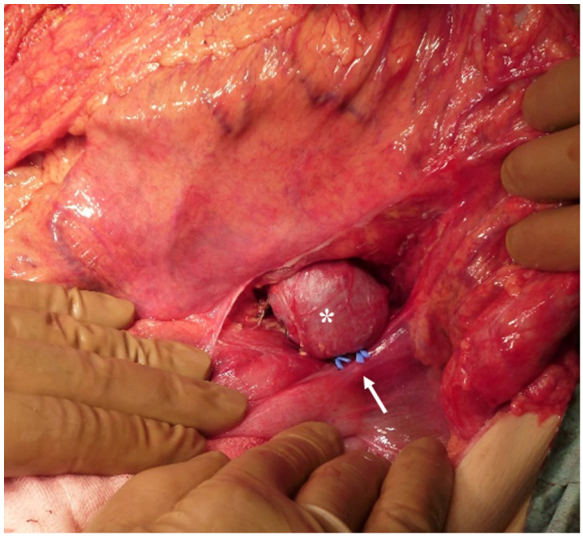

Fig. 2. Intraoperative findings of the recipient. Ligation (arrow) of the dilated IMV-caval shunt (*).

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-

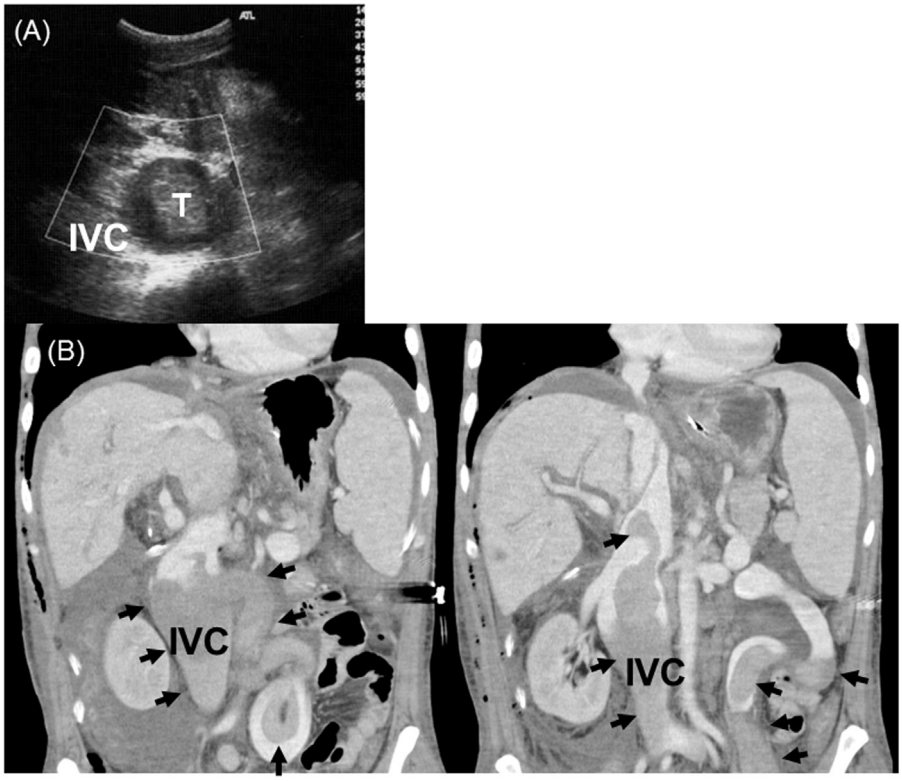

Fig. 3. Diagnosis of IVC thrombosis at postoperative day 1. (A) An ultrasound study showed thrombus (T) inside the infra-hepatic IVC. (B) A contrast-enhanced abdominal computed tomography scan showed a thrombus in the IVC that developed from a thrombus in the collateral vessel of the IMV (arrows). IVC, inferior vena cava.

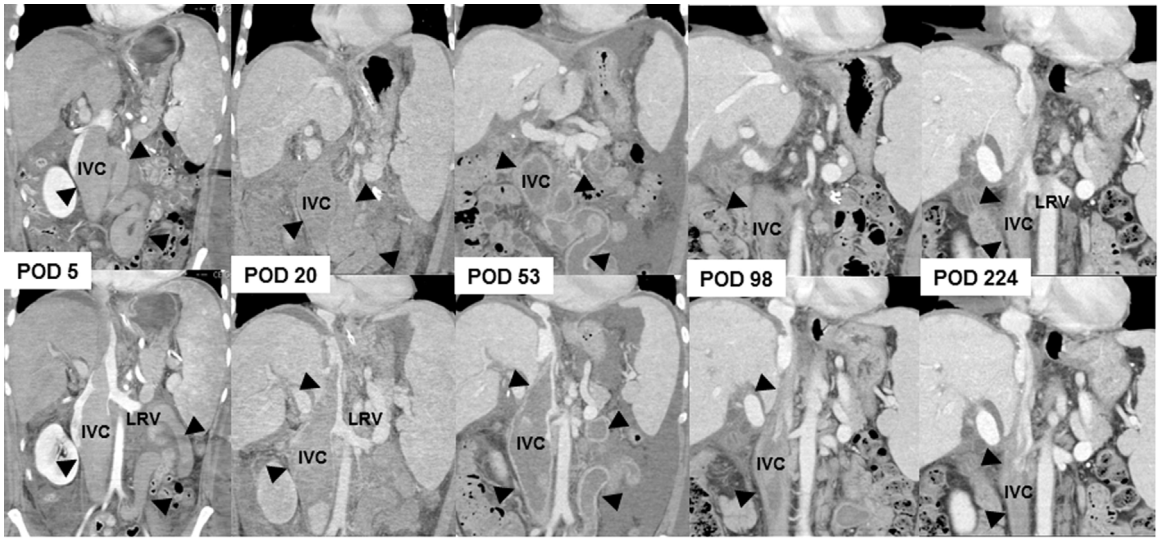

Fig. 4. Gradual resolution of IVC thrombus. The IVC thrombus (arrow heads) was slowly minimized with anticoagulant therapy, although it still remained. Hepatic and renal veins were consistently patent. IVC, inferior vena cava; LRV, left renal vein; POD, post-operative day.

sel of the IMV (Fig. 3B). The thrombus extended to the intrahepatic IVC, but bilateral renal veins and graft hepatic veins were not disturbed. A hypercoagulable workup, including protein S, protein C, and anti-phospholipid antibody, revealed no abnormalities in thrombophilia tests in this patient. Because the thrombus extended toward the intrahepatic IVC, total hepatic vascular exclusion might be required for surgical removal of this thrombus, which could cause liver graft damage, and was considered too challenging in this case. The thrombus was thought to be too large to remove completely with interventional radiology. Fortunately, the patient was hemodynamically stable and his liver function normalized. We eventually selected anticoagulation therapy, including intravenous urokinase, heparin, and antithrombin-III administration. Despite meticulous anticoagulation therapy (a bolus of 300,000 IU of urokinase daily until POD 13 and intravenous continuous infusion of heparin until POD 46), the IVC thrombus could not be dissolved. However, his general condition gradually recovered. For pulmonary embolism prophylaxis, an IVC filter was initially considered, but we concluded that there was no space for its implantation. Therefore, to avoid pulmonary embolism, he was restricted to bed rest for 1 month after DDLT, but physiotherapy was effective for recovering his activities of daily living. He was discharged from hospital

on POD 77. Currently (2 years after LT), he is in good condition with satisfactory liver and renal function. The size of IVC thrombus was slowly reduced with long-time warfarin administration, but complete dissolve of the thrombus was not achieved so far (Fig. 4).

3. Discussion

Collateral pathways in patients with portal hypertension vary [6]. Mesenteric collateral vessels may arise from the superior mesenteric vein and IMV, and may ultimately drain into the IVC via the retroperitoneal or pelvic veins. Retroperitoneal shunts generally develop from the mesenteric veins and drain into renal veins or directly into the IVC [6]. Mesenteric-caval shunts, mesenteric-renal shunts, or mesenteric-iliac shunts can be defined according to the receiving vein [6]. These shunts often have a large diameter and increase the risk of hepatic encephalopathy.

In addition, if a large PSS persists after LT, PV perfusion can become considerably compromised, especially if the diameter of the collateral vessel is larger than that of the PV. Under such conditions, any factor that raises intrahepatic portal resistance, including acute cellular rejection, graft ischemic damage, and vascular complications, can trigger diversion of PV flow. Consequently, a PV flow steal phenomenon can be aggravated, resulting in graft dysfunction or even graft failure despite no stenosis of anastomosis. Therefore, ligation of a major PSS, including a mesenteric-caval shunt, at the time of LT has been suggested [2,3], but where it should be occluded has not been discussed. A case with extensive IVC thrombosis caused by ligation of a major PSS has not been previously reported. Because the infrahepatic IVC was much dilated, the blood flow of the IVC was disturbed possibly by the liver or side-to-side anastomosis which may contribute to the development of devastating IVC thrombus in our case. But the position of large collateral ligation should be carefully considered. 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.

We recognize that treatment for IVC thrombosis selected in our case are arguable. Some might insist that surgical thrombectomy and shunt ligation at the confluence between IVC and shunt was possible when the IVC thrombus was noticed. In this case, total hepatectomy might be required for complete removal of IVC thrombus which was considered too challenging. Although we eventually employed anticoagulation therapy, surgical treatment might have been a better option.

4. Conclusion

In conclusion, ligating a large portosystemic shunt is necessary to obtain adequate portal flow during LT. However, we experienced unexpected consequences of PSS ligation in the form of life-threatening complication. This case serves as a note of caution when addressing a major PSS in LT. To avoid extension of thrombosis to major vessels, the position of large collateral ligation should be carefully considered.

Conflict of interest

We have no disclosure and financial support. We declare no conflict of interest.

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Ethical approval

This case report is written based on institutional ethical committee.

Informed consent

Written informed consent was obtained from the patient for publication of this Case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Author contributions

HK participated in surgical procedure and postoperative treatment of the patient, and draft the report. YO contributed surgical procedure and postoperative treatment. MI and YI treated this patient as hepatology. KS advised on the treatment for this patient. YO participated in surgery and took care of the patient, and supervised this report. All authors read and approved the final manuscript.

Guarantor

The guarantor of this manuscript is Hideya Kamei, corresponding author.

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