

Surgical treatment of a large portopulmonary venous anastomosis in a patient with portal vein thrombosis



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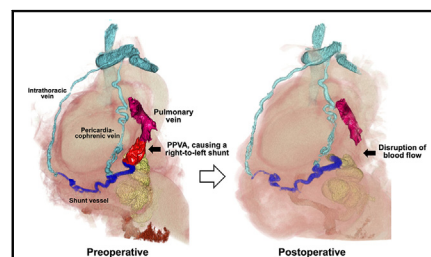
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The left lateral view of the preoperative 3-dimensional image. The red vessel shows portopulmonary venous anastomosis, causing a right-to-left shunt.

CENTRAL MESSAGE

Surgical intervention can be considered a safe and effective option for treating large portopulmonary venous anastomosis.

Video clip is available online.

Portal hypertension is associated with developing collateral vessels in the portal and systemic circulations. A portopulmonary venous anastomosis (PPVA) is a rare collateral blood vessel that causes a right-to-left shunt, and can range from 2 to 7 mm in diameter.¹

We describe a large PPVA coexisting with gastric varices and portal vein thrombosis caused by liver cirrhosis due to nonalcoholic steatohepatitis. Despite the risk of systemic embolization during endoscopic treatment for gastric varices using embolic materials and portal vein endovascular thrombectomy, prior surgical intervention for the PPVA allowed both procedures to be safely performed.

The Ethical Committee of Chiba University Hospital determined that ethical review was not required for the case report (#HK202306-03; June 7, 2023). Written informed consent for publication of this case report was obtained from the patient.

CLINICAL SUMMARY

A 47-year-old man with gastric varices caused by liver cirrhosis due to nonalcoholic steatohepatitis was admitted after hematemesis. The patient had undergone endoscopic treatments for gastric varices 6 times before, and after

that, he was under outpatient observation at this hospital, and no bleeding or thrombosis occurred.

Upper gastrointestinal endoscopy revealed that hemostasis had occurred in the gastric varices. Enhanced computed tomography showed portal vein thrombosis, PPVA, and port-pericardiacophrenic and port-intrathoracic venous shunts on the left side (Figure 1). PPVA was varicose, and the maximum diameter was 15 mm. A blood test revealed the model for end-stage liver disease score of 6 and platelet counts $>100,000/\mu\text{L}$. However, he had pancytopenia associated with hypersplenism due to portal hypertension. Considering the absence of ascites and encephalopathy, the Child-Pugh score was 5.

Although endoscopic treatment was indicated to prevent rebleeding from the gastric varices, a large PPVA introduced the risk of systemic embolism, which may cause cerebral infarction, during endoscopic treatment using embolic materials. Portal vein thrombosis was also an indication for endovascular thrombectomy; however, this treatment was deemed to have the same risk. Because liver function was preserved, surgical intervention for PPVA was planned in advance. We choose an open chest approach instead of a minimally invasive approach, such as thoracoscopic surgery, to handle any complications, including

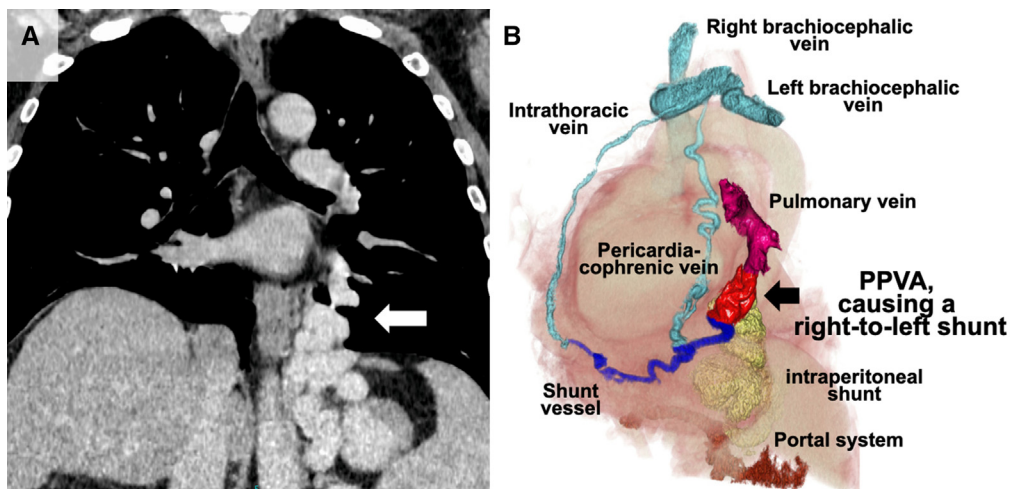


FIGURE 1. Enhanced chest computed tomography. A, Preoperative image shows a portopulmonary venous anastomosis, which flows from the left inferior diaphragmatic vein to the left inferior pulmonary vein (*white arrow*). B, The left lateral view of the 3-dimensional image. The *yellow* vessel demonstrates an intraperitoneal shunt from the portal system (*brown*). The portopulmonary venous anastomosis (*red*) flows into the inferior pulmonary vein (*pink*), causing a *right-to-left* shunt. The shunt vessel on the diaphragm (*blue*) flows into a pericardiacophrenic and intrathoracic vein, reaching the right atrium through the left brachiocephalic vein and superior vena cava (*light blue*).

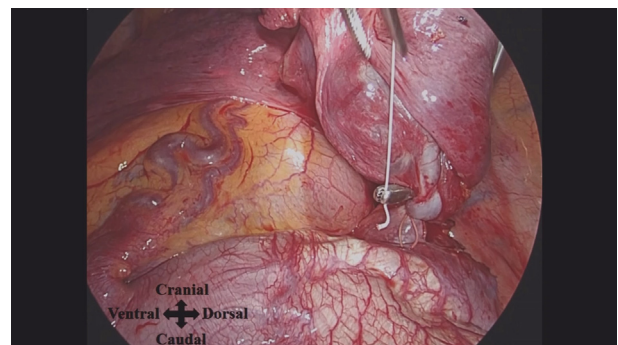
bleeding. Due to the fragility of the abnormal vessel and the risk of bleeding, we decided not to treat the nonsystemic port-pericardiacophrenic and port-intrathoracic venous shunts. We also decided not to perform a surgical devascularization for the varicose veins because the procedure would be more invasive because it requires a laparotomy and a thoracotomy.

Surgical treatment was performed under single-lung ventilation in the right lateral decubitus position, and a 12-cm skin incision was made along the left sixth intercostal space (*Video 1*). The collateral vessels were exposed by incision of the pulmonary ligament and mediastinal pleura, and the left inferior pulmonary vein was secured. There were 2 collateral vessels: 1 flowing through the lung and the other flowing directly into the inferior pulmonary vein (*Figure 2*); each vessel was doubly ligated.

After his general condition had improved, the patient was scheduled to undergo endoscopic treatment for the gastric varices. However rebleeding from the gastric varices occurred on postoperative day 8, and an endoscopic injection of embolic materials was performed. The patient underwent transjugular intrahepatic portosystemic shunt procedure and endovascular thrombectomy for portal vein thrombosis on postoperative day 27. A balloon catheter was used to dilate the short circuit between the portal and hepatic veins in this procedure. A stent was implanted to maintain the short circuit to prevent PPVA recurrence, and the clot was subsequently retrieved. The patient underwent additional gastric variceal embolization on postoperative day 32 and was discharged on postoperative day 37. The postdischarge course was uneventful, without recurrence of gastric varices, portal vein thrombosis, or PPVA for more than 6 months after the series of treatments.

DISCUSSION

Portal hypertension is caused by factors such as liver cirrhosis or hepatic vascular abnormalities and can lead to collateral vessel development in the portal and systemic circulations. Most collateral circulation is formed with veins from the stomach and esophagus. PPVA, causing a right-to-left shunt, is a relatively infrequent collateral blood vessel and the incidence of PPVA in patients with portal



VIDEO 1. Three-dimensional image of preoperative enhanced chest computed tomography shows a portopulmonary venous anastomosis (*red*), which flows from the left inferior diaphragmatic vein to the left inferior pulmonary vein (*pink*). The shunt vessel on the diaphragm (*blue*) flows into a pericardiacophrenic and intrathoracic vein, reaching the right atrium through the left brachiocephalic vein and superior vena cava (*light blue*). In surgical treatment, the collateral vessels were exposed by incision of the pulmonary ligament and mediastinal pleura, and the left inferior pulmonary vein was secured. There were 2 collateral vessels: 1 flowing through the lung and the other directly into the inferior pulmonary vein; each vessel was doubly ligated. Postoperative 3-dimensional image shows disruption of blood flow in the portopulmonary venous anastomosis. Video available at: [https://www.jtcvs.org/article/S2666-2507\(23\)00470-4/fulltext](https://www.jtcvs.org/article/S2666-2507(23)00470-4/fulltext).

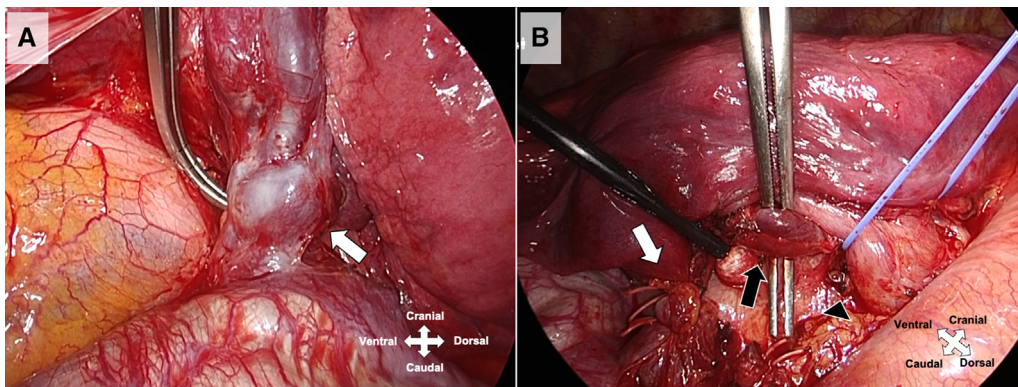


FIGURE 2. Intraoperative findings. Two collateral vessels were recognized. A, A collateral vessel (white arrow) on the caudal side flowing through the lung into the inferior pulmonary vein. B, A collateral vessel (black arrow) on the cranial side flowing directly into the inferior pulmonary vein (black arrowhead). The inferior pulmonary vein was encircled with blue tape. The white arrow indicates a collateral vessel on the caudal side.

hypertension has been reported to range from 8% to 18%.²⁻⁴ To the best of our knowledge, a small number of reports have targeted PPVA, and no articles have described surgical treatment for these vessels.

In the treatment of PPVA, surgery was performed in the present case because portal vein thrombosis prohibited the selection of percutaneous transhepatic endovascular treatment, whereas an alternative strategy of transeptal left atrial puncture (the Brockenbrough procedure) was high risk. In addition, the endovascular and endoscopic approach carried a risk of systemic embolism caused by the deviation of coils or embolic materials because of the large shunt vessels in the present case. Before surgery, the risk of postoperative bleeding from ligated vessels was considered; however, no complications occurred. Postoperative deterioration of portal hypertension caused rebleeding of the gastric varices, but the bleeding was manageable.

Finally, endovascular treatment was performed to prevent the recurrence of PPVA, and the patient had no recurrence or symptoms for more than 6 months. Postoperative enhanced computed tomography showed the disappearance of blood flow in the PPVA. This result suggests that surgical

intervention can be considered an effective option for treating large PPVA to perform gastric varix or portal thrombosis treatments.

Conflict of Interest Statement

The authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

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