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The Superior Vena Caval Syndrome During Orthotopic Liver Transplantation: A Complication of LeVeen Shunt and Venovenous Bypass

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A CASE of the superior vena caval (SVC) syndrome that was precipitated by venovenous bypass during orthotopic liver transplantation (OLT) in a patient with a nonfunctioning LeVeen shunt is presented.

CASE PRESENTATION

A 58-year-old man with the diagnosis of cryptogenic cirrhosis was scheduled for OLT. A LeVeen shunt had been implanted for 2 years, but was no longer functioning. Physical examination was significant for decreased breath sounds on the left and prominent collateral veins on the chest wall. Anesthesia was induced with thiopental and fentanyl, and was maintained with isoflurane, fentanyl, lorazepam, and pancuronium. A femoral and a radial arterial catheter were placed, and two IV catheters (8.5 F) were inserted, one in the right antecubital vein and another in the left internal jugular vein. Insertion of an oximetric pulmonary artery catheter was attempted through the internal jugular veins, but it could not be advanced more than 20 cm on either side. An intraoperative venogram showed a severe narrowing of the SVC at the junction with the right subclavian vein, and several collateral veins draining caudally were seen. The collateral venous flow was considered adequate because the patient had been asymptomatic, and the infusion pressure was less than 150 mm Hg during a short trial infusion of a blood mixture (400 mL/min) via each catheter. An oximetric pulmonary artery catheter was inserted through the right femoral vein. To maintain blood flow through the inferior vena cava (IVC), a "piggy-back procedure" was considered to be the technique of choice. This technique involves transection of the hepatic veins instead of the IVC, thereby preserving the integrity of the IVC and maintaining venous return to the heart even without the use of venovenous bypass.[1] During the dissection of the hepatic hilum, however, it was felt that venovenous bypass would be a safer technique because of the presence of severe portal hypertension and the potential for severe decrease in preload with inadvertent compression of IVC during a piggy-back procedure. Venovenous bypass (portal and femoral to axillary) began with a flow rate of 2 L/min, and the patient was hemodynamically stable: right atrial pressure (RAP) was 10 mm Hg and pulmonary capillary wedge pressure (PCWP) was 13 mm Hg. Twenty minutes after the onset of the bypass, however, before clamping of the IVC and hepatectomy, edema and cyanosis of the head were noted. SVC pressure (SVCP) measured at this time by an internal jugular venous catheter was 40 mm Hg, RAP 8 mm Hg, and mean arterial pressure (MAP) 80 mm Hg. To reduce the venous congestion, the following maneuvers were performed: the patient was placed in a reversed Trendelenburg position, fluids were given only via a newly inserted femoral IV catheter (8.5 F), 500 mL of blood was drained via the left internal jugular vein, dexamethasone (8 mg) and furosemide (40 mg) were administered, and positive end-expiratory pressure was removed. The bypass was terminated, and the procedure was completed using the piggy-back technique. The venous congestion improved over the next 30 minutes, when SVCP decreased to 25 mm Hg. Reperfusion of the grafted liver did not change MAP. Transient increases in RAP to 9 mm Hg, and SVCP to 37 mm Hg, returned to previous levels within 15 minutes. Postoperatively, the patient regained consciousness within 24 hours, and the trachea was extubated on the second day. The SVCP remained at 25 mm Hg, and he was discharged from the intensive care unit on the third day without neurologic deficit.

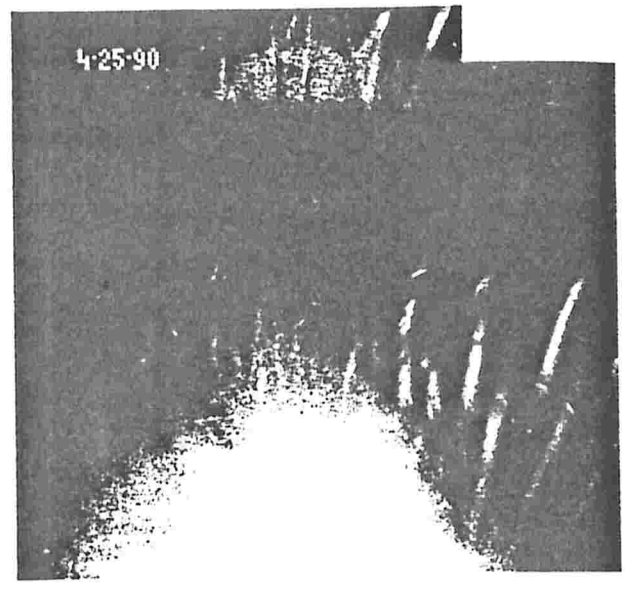

Fig 1. Intraoperative venogram of the superior vena cava.

DISCUSSION

LeVeen shunt has been reported to cause central venous thrombosis in 23% of patients.[2] In patients with recurrent ascites after LeVeen shunt placement, however, total and partial occlusion of the SVC is seen in 53% and 17% of cases, respectively.[3] Thus, the SVC syndrome may occur more frequently than is commonly believed. Patients with partial obstruction may be asymptomatic preoperatively, but a full-blown SVC syndrome may develop during OLT

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with the additional load of blood flow from venovenous bypass. In this case, drainage of SVC blood was erroneously assumed to be adequate because the patient had been asymptomatic, and collateral vessels appeared to be satisfactory on the venogram. Retrospectively, the SVC was noted to be completely obstructed and, therefore, cross-clamping of the IVC could have been fatal. The optimal care of these patients should include preoperative venography to evaluate the patency of the SVC. Intraoperatively, monitoring of SVCP by a jugular venous catheter may be helpful in a suspected case. The piggy-back technique can be a life-saving procedure in patients with the SVC syndrome. When the piggy-back technique is impossible, portal and femoral venous blood should be bypassed directly to the right atrium before cross-clamping of the IVC.

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