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992
958

Successful Reconstruction of Late Portal Vein Stenosis After Hepatic Transplantation

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• **Stenosis of the portal vein anastomosis occurred in three pediatric patients seven to 42 months after transplantation. Dominant symptoms were those of portal hypertension and hypersplenism. Diagnosis was made by angiography. Successful surgical reconstruction was possible in all three patients.**
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Biliary tract complications and thrombosis of the hepatic artery are the most common technical problems observed following orthotopic liver transplantation (OLT).[1,2] In contrast, portal vein (PV) complications are unusual; however, they are associated with high morbidity and mortality.[3-6] Three pediatric patients presented 7, 10, and 42 months after OLT with PV stenoses that were successfully treated by excision of the stenotic segment and reanastomosis.

PATIENTS AND METHODS

The condition of the PV, hepatic artery, and vena cava is determined routinely with ultrasonography in all liver transplant recipients. Between March 1980 and January 1986, three of 184 children who survived more than three months after transplantation had evidence of PV stenosis. The diagnosis was confirmed by selective angiography. In one patient (case 3), venous pressures were measured across the stenosis after transhepatic catheterization of the PV. The following parameters were reviewed: age, indication for transplantation, time interval between OLT and diagnosis of PV stenosis, symptoms, physical and laboratory findings at the time of diagnosis, angiograms, the type of technique for secondary reconstruction, and clinical outcome. The technique for liver transplantation has been previously reported.[7]

RESULTS

The Table summarizes the profile of these three patients and their clinical features at the time PV stenosis was diagnosed. Two of the three patients were symptomatic. The other patient (case 2) had no overt symptoms, although she had mild hepatic dysfunction. On further evaluation, she was found to have splenomegaly and sonographic findings of hepatofugal flow. Because of this, she underwent angiographic evaluation. The cause of hepatic dysfunction was from a stricture of the common bile duct.

Angiographic Findings

All three patients underwent selective celiac and superior mesenteric angiography. In each case, stenosis of the PV anastomosis was identified (Fig 1, left).

Two patients (cases 1 and 3) underwent further evaluation by transhepatic catheterization of the PV to determine if there was a significant pressure gradient across the anastomotic stenosis. Successful catheterization of the PV was possible in both cases. However, in only one patient could the catheter be advanced across the stenosis to measure a gradient (Fig 2). In the other patient, transhepatic portal venography clearly demonstrated a marked stricture even though a gradient could not be measured (Fig 3).

In two patients (cases 1 and 3), evidence of portal hypertension was found as manifested by gastroesophageal varices (Fig 2). In the third patient (case 2), no varices were seen despite the presence of a severe stenosis (Fig 1, left). This last patient underwent follow-up angiography after operative revision of the PV anastomosis, documenting a widely patent PV (Fig 1, right).

Operative Findings

Operation was performed through the same subcostal incision that had been used for the transplantation. In one patient (case 3), the choledochojejunostomy was not taken down because mobilization of the Roux-en-Y allowed adequate exposure of the PV. In the other two cases, the choledochojejunostomy was taken down to facilitate the exposure. Both of these patients (cases 1 and 2) had biliary strictures.

Stenosis of the PV in all three patients was at the anastomoses. The gross pathologic findings were similar in each case. The veins were phlebosclerotic, with focal calcification and foreign-body reaction at the site of the

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Summary of Profile and Clinical Features at Time of Diagnosis of Portal Vein Stenosis					
Case No./Age, y	Indication for Orthotopic Liver Transplantation	Time From Orthotopic Liver Transplantation, mo	Clinical Symptoms	Physical Findings	Laboratory Findings
1/10	Familial cholestasis	7	Variceal hemorrhage	Splenomegaly and ascites	Pancytopenia
2/6	Biliary atresia	10	Asymptomatic	Splenomegaly	Leukopenia
3/6	Biliary hypoplasia	42	Fatigue	Splenomegaly	Thrombocytopenia and leukopenia

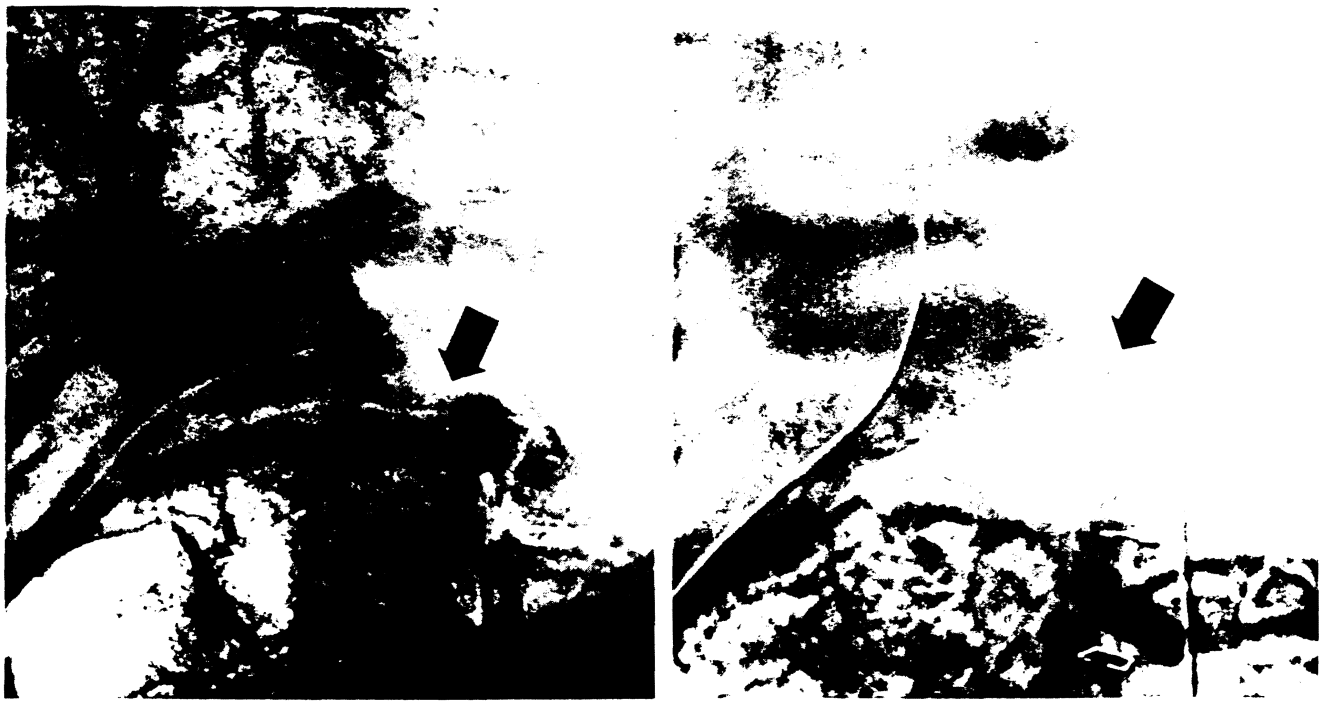

Fig 1.—Case 2. Left, Portal vein anastomotic stenosis (arrow) demonstrated on venous phase of superior mesenteric arteriogram. Poststenotic dilatation of portal vein is evident. Right, Widely patent portal vein anastomosis (arrow) demonstrated on venous phase of superior mesenteric arteriogram performed six days after portal vein revision. Decrease in previously seen poststenotic dilatation is shown.

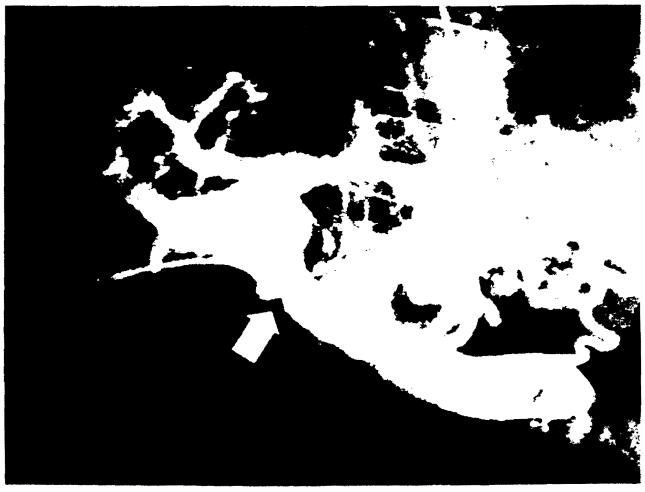

Fig 2.—Case 3. Portal vein anastomotic stenosis (arrow) demonstrated on transhepatic portogram. Prominent gastric varices are seen. Pressure in splenic vein was 29 mm Hg, and pressure in intrahepatic portal vein was 12 mm Hg.

anastomoses. In one patient (case 1), a recent clot was found in the donor side of the PV that extended into the hilum. Histologic examination of this specimen showed mural thrombosis with recanalization. Thrombectomy was performed.

In each case the anastomosis was excised by removing a ring of vein measuring 1 to 2 cm long after obtaining proximal and distal vascular control. In all three cases the length of the vein was adequate for allowing reapproximation of the two ends without tension. The anastomoses were performed end-to-end with 7-0 polypropylene suture. Systemic heparinization was not used. All patients recovered successfully and were discharged in excellent condition. The spleen in all three patients was noted to have decreased in size at the time of last follow-up.

COMMENT

Portal vein thrombosis after OLT occurs more frequently in pediatric patients. Symptoms of portal hypertension are the primary manifestations. Lerut et al[3] reported seven cases of PV thrombosis following OLT, with an overall incidence of 1.8%. Five of the seven patients developed variceal hemorrhage.

Previous portosystemic shunt and partial or complete thrombosis of the portal system requiring thrombectomy

Fig 3.—Case 1. Marked stenosis of portal vein anastomosis demonstrated on transhepatic portogram (arrow). Catheter could not be advanced across stenosis to measure pressure gradient.

at the time of transplantation are thought to be contributing factors to the development of postoperative PV complications. However, not all patients demonstrate an obvious cause. Of the seven cases reported by Lerut, only three had contributing factors. One patient had a portacaval shunt, one had a thrombosed PV, and one had hypoplasia of the PV. None of the three patients described herein had any contributing cause for the development of PV stenosis following transplantation. However, dense scarring in the portal area may have contributed; two of the three recipients also had a stricture at the site of bile duct anastomosis. The best possibility is that technical errors were made at the time of the original transplantation. The fact that secondary repair was possible without vein grafts might suggest that the cuffs were originally made too long, which consequently caused kinking or distortion.

In all three cases, the diagnosis was suspected by physical examination and confirmed by angiography. Evidence of a recent clot in the portal vein extending into the hilum of the liver was found in one patient (case 1). This clot was not demonstrated by angiographic studies performed three and eight days before revision and may have been contributed to by the instrumentation. Pathologic findings of focal calcification, thickening, and fibrosis in the area of the anastomotic suture line were common to all cases.

The status of the extrahepatic PV should be evaluated in patients after transplantation with recurring symptoms of portal hypertension, hypersplenism, or both. Ultrasonography can be performed for screening, but definitive diagnosis requires angiography. Successful surgical correction of PV anastomotic stenosis can be achieved with little morbidity. The possibility of balloon dilatation of the strictures after transhepatic PV catheterization was not seriously considered for these patients, but this may be a viable nonoperative option for the future.

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