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Portal stenosis in liver transplantation. Treatment by endoluminal prosthesis

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Sir: Portal vein stenosis, with the accompanying technical difficulties, may occur after liver transplantation and is usually detected either in the early postoperative period, or later on in association with inflammatory reactions in the hepatic hilum. It is a subject seldom referred to in the literature [1, 6] but one that can lead to significant disturbances in hepatic function, particularly when the stenosis is severe.

A 29-year-old man with cirrhosis secondary to hepatitis C virus (Child B), developed a biliary leak in the immediate postoperative period. The leak originated from a choledochocholedochostomy that had been repaired on the 18th postoperative day. The repair converted the anastomosis to a Roux-en-Y hepaticojejunostomy. Three days later, after the accidental extraction of the biliary stent, a new leak was observed and a reanastomosis was performed with a new stent. The patient was discharged from the hospital on the 39th postoperative day with good hepatic function.

One year later, a stenosis of the hepaticojejunostomy with hepatolithiasis was diagnosed. A stone was removed from the hepatic duct and a new anastomosis was performed. During a routine analysis 30 months after liver transplantation, a moderate alteration in the coagulation values was detected (prothrombin activity 60 %), while the other hepatic parameters remained near-normal (AST 35 U, ALT 36 U, bilirubin 1.6 mg/dl, alkaline phosphatase 405 IU, GGT 120 IU). At the same time, endoscopy showed esophageal varices. Hepatic arteriography was performed and demonstrated a severe and rather long (3-cm) portal vein stenosis with major collateral circulation through the coronary vein (Fig. 1 A), something that has not been reported in earlier studies.

Transparietohepatic measurement of the preanastomotic portal vein pressure showed a value of 35 mm Hg. Therefore, a series of progressive dilatations with angioplasty balloons, ranging in diameter from 10 to 14 mm, was carried out. The dilatation obtained was limited (20 % of the portal diameter), and although the pressure decreased to 9 mm Hg, most of the collateral circulation through the coronary vein persisted (Fig. 1 B). Given these circumstances, we decided to perform a new angioplasty and to place a 12-mm "Wall stent" prosthesis (Schneider, Zurich), which enabled us to obtain an almost complete dilatation of the stenosis with disappearance of the collateral circulation through the coronary vein (Fig. 1 C, D). At the end of the procedure, the intrahepatic path was embolized with coils and gel foam. Measurement of portal pressure immediately after the procedure failed to show any decrease. In the 2 weeks that followed, the patient had fever that was successfully treated with

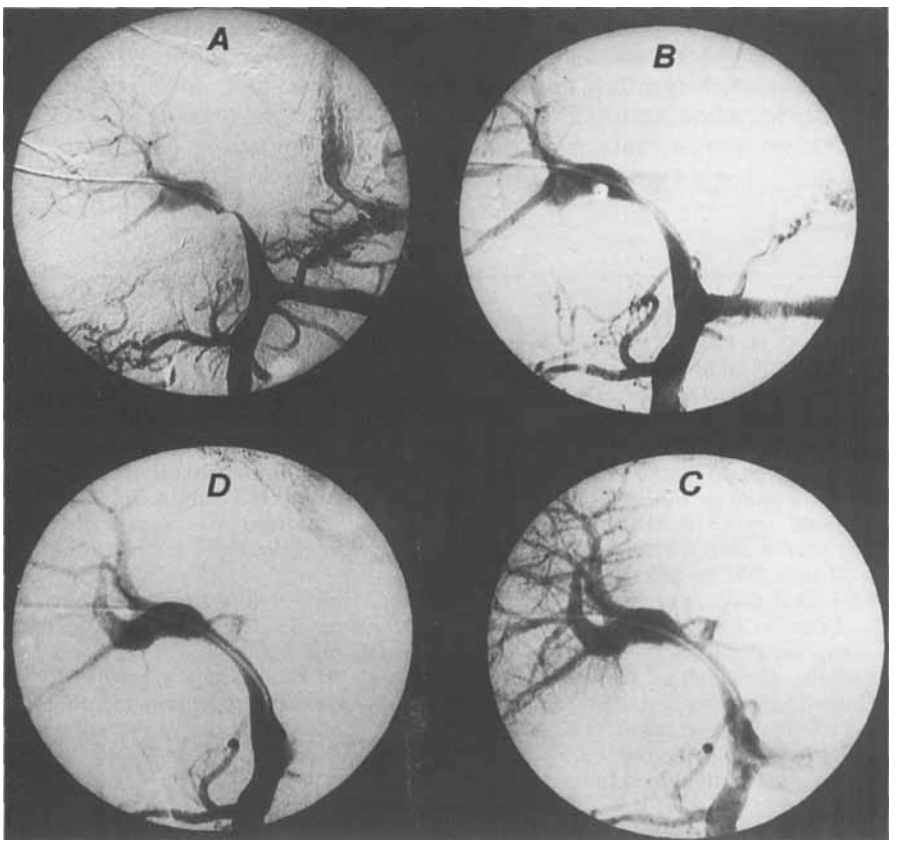

Fig. 1 A–D Portography: **A** transhepatic, demonstrating a severe stenosis of the portal vein; **B** after dilatation with a 14-mm balloon; **C, D** after placement of a "Wall stent" prosthesis

antibiotics. Hepatic function became normal and the esophageal varices disappeared.

Now, 1 year after the angioplasty, the patient has normal hepatic values. The Doppler ultrasound results are normal and endoscopy shows no varices. Portal vascularization, which has been studied by arterial portography, shows an increase in the diameter of the stenotic portion compared to its size immediately after placement of the prosthesis.

In the present case, portal stenosis was detected in a patient 30 months after liver transplantation, something not previously reported. The characteristics and timing of this complication suggest that it may be related to the inflammatory processes and operations that were involved in repairing the bile leaks. Clinically, the complication was demonstrated by alterations in the coagulation data, slight changes in hepatic tests, and the appearance of esophageal varices resulting from portal hypertension.

The portal thrombosis rate is low in large series, ranging from 1 % to 2.6 % [2–5]. However, this is seldom referred to in the literature [1, 6]. The series reported by Azoulay et al. is the most important of these series [1]. They also describe an original treatment with a minimally invasive angioplasty technique.

In our case, angioplasty was clearly insufficient, and for that reason we decided to place a "Wall stent" prosthesis. With this technique an almost complete resolution of the portal stenosis was achieved, with the disappearance of the collateral circulation and esophageal varices. This is the first case of portal stenosis after liver transplantation that was successfully treated with this kind of vascular prosthesis.

With regard to the route used for the angioplasty, percutaneous transhepatic puncture can, in theory, lead to more complications than mesenteric access, as described by Azoulay et al. [1]. An advantage, however, is that general anesthesia can be avoided and the patient can be treated in the interventional radiology unit. Nonetheless, transhepatic access has frequently been used in our patients without major complications since the intrahepatic path is embolized at the end of the procedure.

We conclude that the use of "Wall stent" vascular prostheses should be considered in cases such as the one described here when angioplasty does not suffice and always as an alternative to reconstructive surgery.

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