Mesenterico-left intrahepatic portal vein shunt : original technique to treat symptomatic extrahepatic portal hypertension

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Abstract

Mesenterico-left intrahepatic portal vein shunt : original technique to treat symptomatic extrahepatic portal hypertension.

Objective **: Revascularization of the intrahepatic portal system as decompressive surgery for chronic extrahepatic portal hypertension.**

Summary background data **: In patients with extrahepatic portal hypertension (portal trunk thrombosis in presence of a normal liver), shunt surgery is indicated when patient is bleeding from varices at a site not accessible for the endoscopist. Although surgical portal decompression is an efficient procedure, there is a risk of depriving the liver from the splanchnic venous flow and a risk of developing porto-systemic shunt related side effects.**

Method **: A shunt was created between the superior mesenteric vein and the umbilical portion of the left portal vein. This technique allows to bypass the thrombosed portion of the portal vein but avoiding dissection of the cavernoma in the liver hilum and related risk of intraoperative hemorrhage.**

Results **: The procedure was successfully performed in one adult patient considered unshuntable in view of classic surgical procedures and in whom sclerotherapy was unsuccessful. This operation achieved an effective decompression of the splanchnic venous system.**

Conclusion **: Rerouting the venous splanchnic flow through the liver was possible. It had the major physiological advantage of restoring the normal hepatic vascularization. It also avoided putting the patient at risk of developing porto-systemic shunt related side effects. This option should be considered when shunt procedures are indicated in patients with extrahepatic portal hypertension.** (Acta gastroenterol. belg., **1998**, 61, **13-16**).

Keywords : portal hypertension, extrahepatic, portal vein thrombosis, surgical technique, mesenterico-portal bypass.

Introduction

Portal or splanchnic vein thrombosis is not an uncommon complication of chronic pancreatitis or after regional surgery (1-3). The incurred extrahepatic portal hypertension in the presence of normal liver function is said to be extremely well tolerated (2, 3). However, evolution frequently leads to recurrent gastro-intestinal bleeding, usually treated by sclerotherapy (2, 4, 5) or by performing porto-systemic bypasses (6, 7), and a 5 to 15% death rate due to recurrent bleeding is reported, including death of "untreatable" patients (4, 8-10).

We describe a case where endoscopic treatment was ineffective and where conventional surgical shunting was not possible. In this patient, the extrahepatic portal hypertension was relieved using an original surgical technique (11, 12) connecting the superior mesenteric vein to the intrahepatic patent portal vein ; this procedure also had the major physiological advantage of restoring the portal hepatic flow.

Case report

In 1976, the patient, then aged 45, underwent a hepatico-jejunostomy (Roux-en-Y loop) because of a bile duct stenosis due to idiopathic chronic pancreatitis. Two years later, a splenopancreatectomy had to be performed. Both operations were uneventful, but the patient required insulinotherapy from 1982. When admitted in November 1994 in a peripheral hospital for a first episode of melaena and anemia, an upper and lower gastro-intestinal tract endoscopy revealed grade 2 oesophageal varices ; there was no direct evidence of oesophageal variceal bleeding, but endoscopic sclerotherapy was started. Abdominal computed tomography showed mild ascites. Liver function tests and liver histology were normal.

In January 1995, he was referred to our centre because of recurrent melaena, hemochezia and severe anemia (Hb : 7.6 g/dl). There was no history of liver disease, excessive alcohol intake, hematemesis or peptic disease. Physical examination was unremarkable except for pallor. Blood examination showed a microcytic anemia. An upper GI endoscopy confirmed the presence of non bleeding oesophageal varices, also showed duodenal varices, but no blood in spite of active hemochezia. Minutious colonoscopy revealed rectal varices among 30 cm, some vascular spiders and a colonic mucosa with slight pseudonodular pattern : histological examination indicated venous stasis and capillar congestion. Catheterism showed low wedged and free hepatic vein pressures, without gradient (6 and 3 mmHg). Liver histology was normal. Color Doppler ultrasonography and spiral CT-scan during arterial portography concluded for a thrombosis of the mesenterico-portal confluence and the splenic vein. However, the intrahepatic portal branches as well as the distal portion of the portal trunk and its bifurcation remained patent. Enlarged varicose veins coursing

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along the Roux-en-Y loop provided most of the cavernomatous hepatopetal flow. Endoscopic ligation of oesophageal varices did not succeed to cease the bleeding as shown by seven hemorrhagic episodes during the following four months. It was assumed that the source of bleeding was the congested hepaticojejunostomy. This hypothesis was confirmed by a SPECT labelled erythrocyte scintigraphy during one episode of recurrent bleeding.

Whilst hospitalized, the patient developed pulmonary emboli originating from a femoro-popliteal vein thrombosis. Heparinotherapy was started but had to be stopped because of recurrent hemochezia ; an inferior vena cave umbrella was then positioned.

In order to prevent further bleeding, shunting had been discussed, but several factors seemed against it : the previous splenectomy, the fact that all the major tributaries of the portal system were not visualized at angiography and Doppler US, and the position of the umbrella which made access to inferior vena cave much difficult. The patient had therefore been initially considered "unshuntable". As bleeding had recurred, explorative laparotomy was performed in order to explore directly the mesenteric venous system (April 1995).

Operative technique

A catheter was positioned in an ileal vein ; this allowed both pressure gradient measurement (atrium/mesenteric vein) and direct venography which showed a patent distal superior mesenteric vein. Then the umbilical vein was re-canalized and a catheter positioned in the left intrahepatic portal vein. This allowed the exclusion of any intrahepatic block by recording absence of pressure gradient between intrahepatic portal branches and right atrium. Venography through this latter catheter also confirmed left portal vein patency and anatomy. The umbilical remnant was then further dissected, towards the distal part of the left portal vein and the branches for segments III and IV, allowing excellent exposure of the "umbilical portion" of the left portal vein over 3 to 4 cm length. A small Saltinsky clamp allowed good control of the left portal vein and its branches (lateral clampage). The umbilical remnant was divided at its junction with the left portal vein, and this venotomy then longitudinally extended on the ventral aspect of the left portal vein. A 10 mm reinforced Goretex prosthetic graft was anastomosed end-to-side to this vein, then brought in front of the duodenum and then through the transverse mesocolon towards the superior mesenteric vein, where it was implanted terminolaterally (Fig. 1).

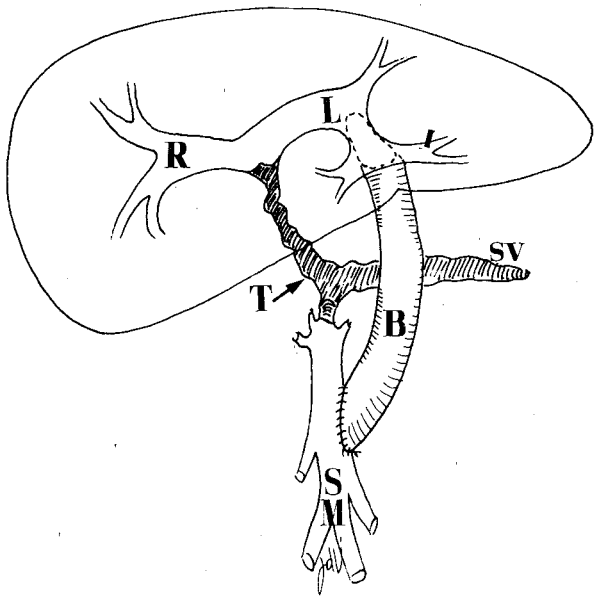

Fig. 1. — Direct bypass from the superior mesenteric vein (SM) to the distal part of the left portal vein (L). (R : right portal vein. Dark area : thrombosed portal trunk (T) and splenic vein (SV)).

The portosystemic pressure gradient (44 mmHg before bypass) was reduced to normal (10 mmHg) values. Intraoperative venography confirmed full restoration of the mesenteric flow to the liver.

The postoperative course was uneventful. The patient was followed clinically and using Doppler ultrasound regularly, confirming shunt patency. To date, over 16 months later, gastro-intestinal hemorrhage has not recurred. Rectoscopy performed 5 months postoperatively showed disappearance of the rectal varices.

Discussion

Many patients appear to tolerate extrahepatic portal hypertension very well and remain asymptomatic. This is probably due to the fact that liver function is normal in most patients. However, recurrent gastro-intestinal bleeding of varying severity is the more important event in the clinical course and outcome of these patients. When variceal bleeding occurs, endoscopic sclerotherapy is the first treatment of choice for several teams (4, 13), while others propose porto-systemic shunts with similar results (6, 7). However, this latter therapy is impossible for numerous "unshuntable" cases where splenic and mesenteric veins are involved in the thrombotic process. Sugiura procedure or more radical procedures such as oesophagogastrectomy or even hepatic and multivisceral transplantation have even been proposed to treat patients who were either "non-responding" to either sclerotherapy or variceal ligature, and/or "unshuntable" symptomatic patients (8, 13-15).

In this case, extrahepatic portal hypertension was diagnosed after bleeding from unusual variceal sites. Endoscopy did not succed to stop the bleeding and the patient was initially considered as "unshuntable" because of the anatomical condition. However, as intrahepatic branches of the portal vein remained patent, we examined the possibility of bypassing the thrombosed venous segments. Finally, we decided to perform a laparotomy for direct exploration of the splanchnic venous system. The successful outcome of

this direct exploration should be taken into account when patients are said to be "unshuntable" according to classic imaging.

The original mesenterico-left portal vein bypass was first developed by our team in 1991 for treatment of portal vein thrombosis occurring after paediatric orthotopic liver transplantation (11, 12). As suggested by this case report, it can also be applied to non-transplanted patients presenting extrahepatic portal vein thrombosis. In the setting of extrahepatic portal hypertension, it is nearly always the rule (at least in our pediatric personal experience) that intrahepatic portal branches remain patent and that liver histology is normal. Normal liver has a low resistance to portal venous flow ; thus, it was assumed that splanchnic venous pressure will normalize after bypassing the obstructed portal vein and revascularization of the intrahepatic portal system.

In 1992, Chen *et al.* proposed a similar bypass using the splenic vein ; however, the disadvantages are the necessity of performing a splenectomy and the fact that the technique is useless when the splenic vein is thrombosed (16). In December 1995, Silvestri *et al.* proposed another operation (17), bypassing from the superior mesenteric vein directly to the patent portion of the portal trunk ; they mentioned a very difficult exposure of the portal vein because of the tissue reactions to prior operations. This experience confirms that, because of the cavernomatous transformation of the liver hilum, direct access to the portal trunk should be avoided, especially in case of previous hilar operations. Using the extrahilar approach allows one to dissect out an intact anatomical area without difficulty, and avoiding the risk of major bleeding.

According to MacDougall *et al.*, portal vascular resistance is the dominant factor maintaining raised portal pressure (5). Using this original technique in patients with an histologically normal liver, the resistant portal venous segment is bypassed. The intrahepatic portal system is revascularized and the physiological portal hepatic flow is restored ; it allows the normalization of portal pressure and hemodynamics with progressive regression of submucosal gastrointestinal varices. In our case, an intraoperative decrease of the atrio-mesenteric pressure gradient and selective venography confirmed immediate restoration of a physiological splanchnic venous flow towards the liver. The good clinical evolution with absence of recurrent gastrointestinal bleeding and disappearance of varices, illustrates that this technique allowed an adequate diversion of splanchnic venous blood flow with subsequent decompression of the splanchnic venous hypertension.

This case report confirms that direct hepatic portal revascularization is possible ; comparing the outcome after various other therapeutic programs should give impetus to consider this new therapeutic option. Because the liver is normal, patients with extrahepatic portal hypertension have a better prognosis than patients with cirrhosis ; however, a 5 to 15% mortality rate is reported (9, 10) as well as significant related morbidity (hepatic function deterioration with age, hypertensive gastropathy, gastro-intestinal bleeding, pulmonary hypertension, biliary obstruction, coagulation abnormalities, ascites) (4, 5, 8, 9, 14, 18-20). After shunt surgery in patients with normal liver, the risk of developing encephalopathy or technical failure is reported to be low (6, 7, 21), but shunts tend to deprive the liver of the splanchnic flow. The discovery, some years ago, that portal diversion can promote several physiological consequences regarding cell structure and function or regeneration is thus probably of major concern for the long term prognosis of the "shunted" patients (22, 23).

In conclusion, we consider that this technique should be considered whenever symptomatic patients with extrahepatic portal hypertension require shunt surgery, with the limitation that the liver must be histologically normal and that intrahepatic branches of the portal vein must be patent. As suggested by our case, some of the conventionally "unshuntable" patients may also benefit from a direct approach to the intact intrahepatic portal system. The re-routing of the portal flow into the normal liver is the most physiological diversion that can be achieved, and it allows one to rule out any risk of post-shunt encephalopathy. These major advantages imply that this intervention could be indicated earlier in the evolution of portal hypertension of extrahepatic origin ; an earlier and hemodynamically adequate operation could eventually prevent the thrombotic process from extending to all major veins. Wider experience and further studies are necessary to provide evidence that preventive treatment could eventually be considered.

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