PORTAL VEIN THROMBOSIS: THROMBECTOMY AND PORTA-CAVAL ANASTOMOSIS WITH THE USE OF A GRAFT

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Approximately 13% of patients with liver cirrhosis and portal hypertension will develop thrombotic occlusion in the portal vein (3). With the portal vein occluded, relief of portal hypertension by porta-caval shunt is therby impossible. Under ordinary circumstances, if portal vein thrombosis is demonstrated by splenoportography, a shunt operation utilizing the portal vein is not embarked upon, and alternative operative procedures should be considered.

The purpose of this report is to describe a case of portal hypertension with portal vein thrombosis, in which thrombectomy was done to establish patency of the portal vein prior to portal-caval anastomosis with the use of a prosthetic graft. Previous splenectomy precluded the use of the splenic vein for spleno-renal anastomosis.

CASE REPORT

The patient, a Malay watchman, 35 years of age, was admitted to the Singapore General Hospital in April, 1960, for hemetemesis and melena. In 1945. he was treated for malaria. and at that time was noticed to have splenomegaly.

In 1957, he had his first attack of hemetemesis and melena, and was treated by blood transfusion. The following year he developed hypersplenism. Laboratory investigations revealed a Hb. of 30% leucocyte 1600/c.mm., platelet 10,000/c.mm. Bone marrow biopsy showed normal cellularity, serum protein was 6.7g.% (albumen being 3.5g%) Barium swallow showed esophageal varices. On December 1958, splenectomy was carried out for his hypersplenism. At operation, the liver was found to be slightly enlarged but not grossly abnormal. Histologically, the spleen showed congestion. Splenectomy subsequently corrected the hematologic abnormalities associated with hypersplenism.

He was well until August 1959. when he had a second episode of bleeding. He was again transfused. In May 1960, he had his third bleed, and this led to his present admission. It was then decided that a porta-caval shunt should be done in an effort to prevent further hemorrhage.

On 6.6.60, under general anesthesia, a right thoraco-abdominal incision was made. The right lobe of the liver was mobilized by dividing the triangular ligament. Through an incision on the posterior parietal peritoneum, an adequate length of the inferior vena cava was mobilized. The portal vein in the free edge of the lesser omentum was exposed and dissected free but was found to be thrombosed. The alternative procedure of spleno-renal anastomosis was not possible because of previous splenectomy. The portal vein was transected as it entered the liver. No blood flowed out of its obliterated lumen. The thrombus was peeled out of the portal vein as a firm plug which extended from the liver to the still patent superior mesenteric vein. The intima of the portal vein beneath this thrombotic plug was smooth and glistening and there were no fibrous attachments between the vessel wall and the occluding thrombus. A good flow of blood gushed from the now patent portal vein which was easily controlled with an occluding vascular clamp. Despite radical mobilization of both the cava and the portal vein stump and the head of the pancreas. The portal vein and vena cava could not be approximated sufficiently for anastomosis. A teflon vascular graft $\frac{1}{2}''$ diameter and $1\frac{3}{4}''$ long was used to bridge the gap and to complete the anastomosis. When the anastomosis was completed blood was felt flowing from the portal vein into the inferior vena cava. At the same time, the grossly distended tributaries of the portal vein were found to have diminished in size and pressure. The wound was closed and the right pleural cavity drained.

The first two post-operative days were stormy, featured by shock and oliguria. With blood transfusion and vasopressor drugs, he responded. and the subsequent convalescence was uneventful. No anticoagulant was used. A month after his operation, test for the patency of his shunt was carried out according to the method we have previously described (2). This test depends on the oral intake of ammonium citrate. and the serial estimation of blood ammonia levels. In normal people, the ammonium citrate passing through the liver is converted to urea. Hence a low flat curve is obtained. If a shunt is patent, the blood by-passes the liver and a corresponding high level of blood ammonia results. Our graph suggests a patent shunt. Barium swallow repeated six months after operation showed persistence of the esophageal varices. The patient remained well for almost a

year after his operation, when he was readmitted for hemetemesis. He was transfused. The bleeding stopped spontaneously.

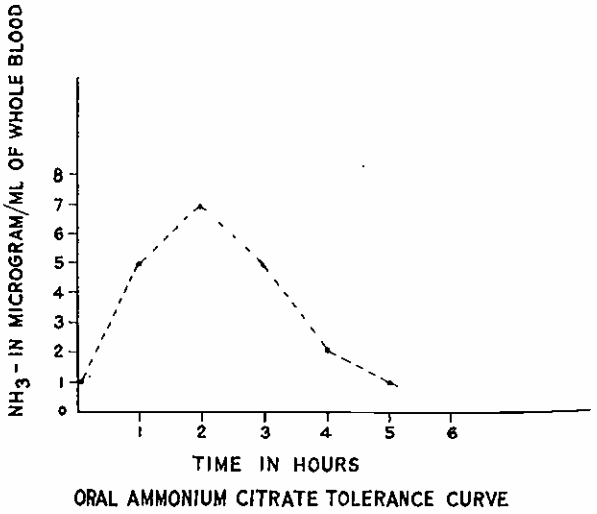

Fig. 1. Oral ammonium citrate tolerance curve which shows a peak in the blood ammonia level about two hours after the intake of ammonia citrate. Curve suggests patency of the shunt.

DISCUSSION

Portal vein thrombosis occurs in approximately 13% of patients with cirrhosis and portal hypertension (4). The exact cause of the thrombosis is not clear, but it seems reasonable to suppose that thrombosis is the result of venous stasis in the portal vein due to increased intrahepatic pressure. McIndoe (7) thought that the portal obstruction in cirrhosis resulted from the derangement of the intrahepatic portal veins by proliferation of the hyperplastic liver cells. Such a balance, and indeed the reversal of flow in the portal vein has been shown to be the result of intrahepatic arteriovenous shunts that occur in cirrhosis (3,8,12). Thus in the cirrhotic, blood within the portal vein may actually flow away from the liver. When the pressure in the portal bed is equalled by the intrahepatic pressure (portal), the blood in the portal vein may come to a standstill, and thus predisposes to thrombosis. In fact, Kelsey et al have shown that cirrhosis is the commonest cause of portal vein thrombosis (5).

In cases of portal hypertension, it is customary to visualize the portal venous tree by means of spleno-portography. In cases where portal vein thrombosis is demonstrated, splenorenal shunt or other alternative operative procedures are employed, although there are some workers in this field who would regard splenorenal shunt as the operation of choice (6). However this case report shows that even in the presence of portal vein thrombosis, portacaval shunt should not necessarily be excluded from consideration. It also emphasizes the fact that splenectomy alone is an inadequate operation for hemetemesis of portal hypertension. If splenectomy is done, it should be done with splenorenal shunt.

Hunt(4) in his discussion of portal vein thrombosis described one case in which he disobliterated the thrombus in the portal vein and did a direct end-to-side portacaval shunt. We have performed such portal venous thrombectomy on 4 previous occasions. On the other hand, the use of a graft in shunt operations is very uncommon. Reynolds (9) described two cases in which he used the azygos vein to bridge the gap between the portal vein and the inferior vena cava. One of the patients died in the second postoperative week, and autopsy showed the venous graft to be patent. The second patient was discharged from the hospital, well. Autogenous veins, preserved arteries, and prosthetics have all been evaluated experimentally in the portal venous system (1,11,13) with generally encouraging results. In a few patients (10, 14) similar reference is made to their use in patients requiring portal-venous shunts.

Although a bridging prosthetic shunt in the portal venous system may remain patent, the low and varying pressures will necessarily make their use unappealing if it can possibly be avoided. In the case under discussion the necessity for prior portal vein thrombectomy plus prosthetic grafting undoubtedly represented a double jeopardy. The fact that the patient subsequently bled again is certainly suggestive that his shunt has closed. His subsequent course should indicate whether this be so. Vein, preserved arterial, and prosthetic grafts have all been used experimentally in the portal venous system (1,11,13).

SUMMARY

This paper discusses a case of portal vein thrombosis in a cirrhotic patient. It is mentioned that cirrhoisis is the commonest cause of portal vein thrombosis.

The report describes the use of a teflon graft in porta-caval shunt.

ACKNOWLEDGMENT

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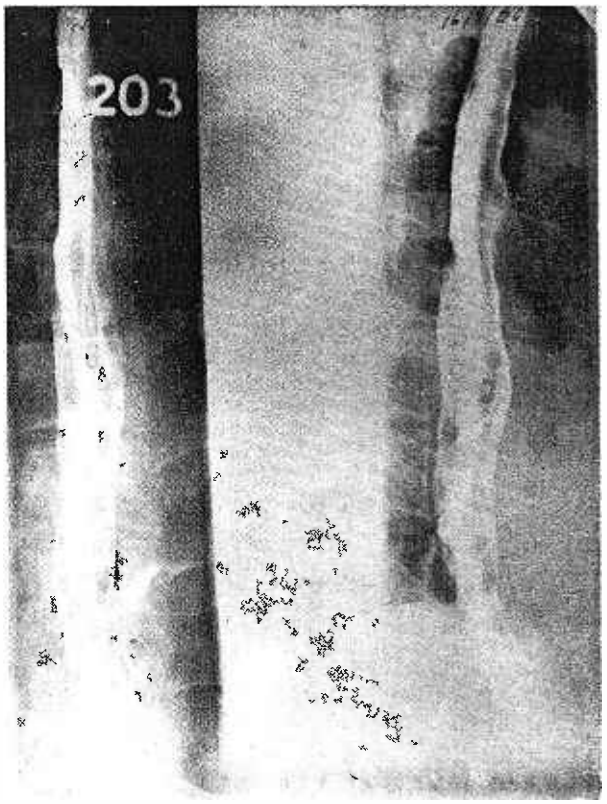

Fig. 2a.

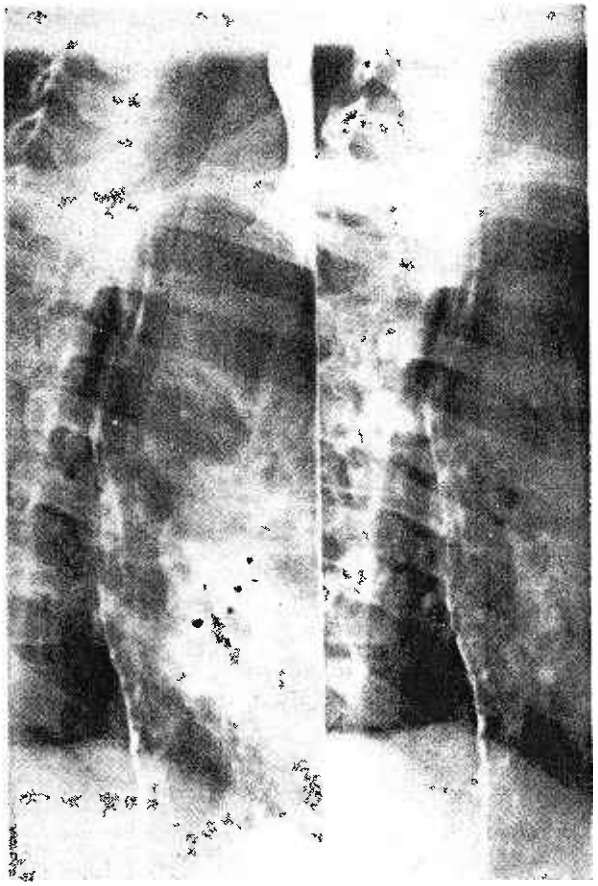

Fig. 2b.

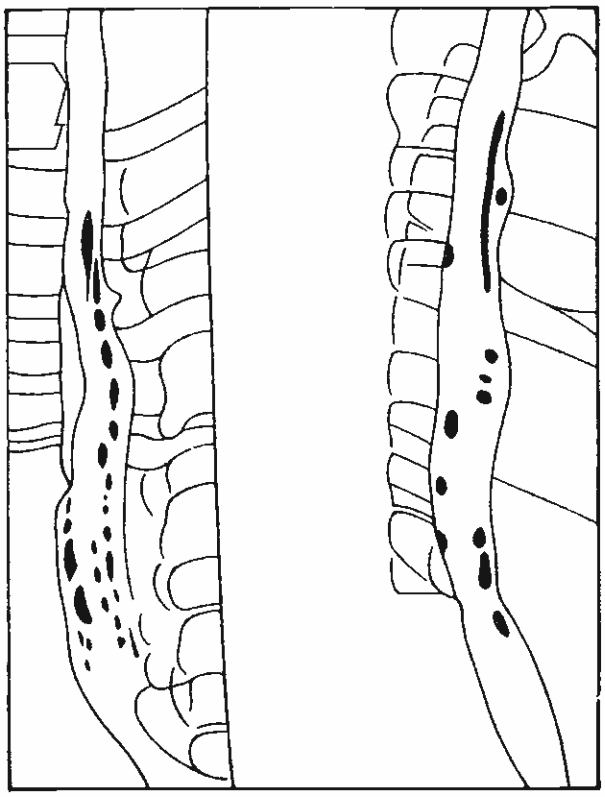

Fig. 2a. Pre-operative barium swallow which shows extensive esophageal varices.

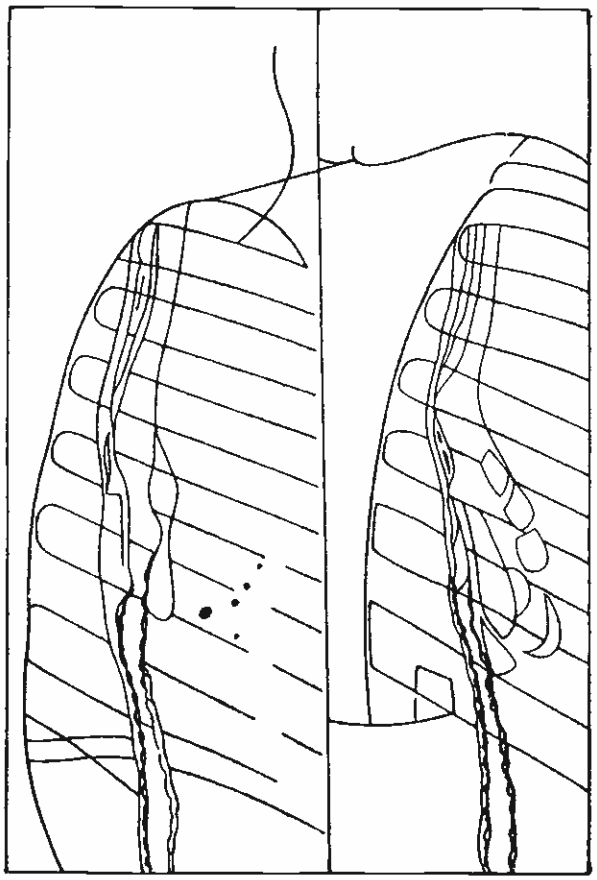

Fig. 2b. Post-operative barium swallow six months after shows the persistence of the esophageal varices.

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