# The Management of Hypersplenism in Portal Hypertension due to Liver Cirrhosis

Vaidya P.L., and
Hagop D. Yacoubian*

WE wish to present three cases of hypersplenism associated with liver cirrhosis and portal hypertension managed at Salmaniya Medical Center during the past 17 months.

## CASE PRESENTATIONS

### Case No. 1

A.A.N., a 16 years old Bahraini male was admitted to Salmaniya Medical Center with splenomegaly, pancytopenia and left upper abdominal pain of 11 years duration. Patient was a known case of sickle-cell trait with reduced G 6 PD activity and mild jaundice. His abdomen was grossly distended due to a huge spleen. He was noted to have gynaecomastia and bilateral small testes.

*Pertinent Laboratory Data :* Haemoglobin was 10.3gm% WBC count 2900/cu mms and platelets 42000/cu mms. Liver profile showed total proteins of 6.8 gms% (A/G. 3.6/3.2). Total Serum Bilirubin was 4.0 mgs% (conjugated 2.8 mgs%, non-conjugated 1.2 mgs%) Alkaline phosphatase : 340 I Us/Litre (normal range 25 — 80), S G P T : 37 I Us/Litre (normal range 0 — 16).

Bone marrow revealed normoblastic hyperplasia. Barium swallow demonstrated oesophageal and gastric varices.

A diagnosis of hypersplenism was made and splenectomy and liver biopsy were performed. Portal pressure was 30 cms of water. The liver biopsy showed post-necrotic cirrhosis. The post-operative course was complicated by fever of unknown origin for one week. Platelet count shot up rapidly to 520,000/cu mms within 9 days post-operatively and the WBC count went upto 10300/cu mms.

The patient was heparinized prophylactically from the 9th post-operative day on up to the 24th post-operative day, when the platelet count dipped below 500,000 and heparin was discontinued. He was discharged on the 30th post-operative day. When seen in the outpatient clinics on the 50th post-operative day he was well, with a platelet count of 300,000/cu mms, WBC count of 12,700/cu mms and Haemoglobin of 12.7 gms%.

### Case No. 2

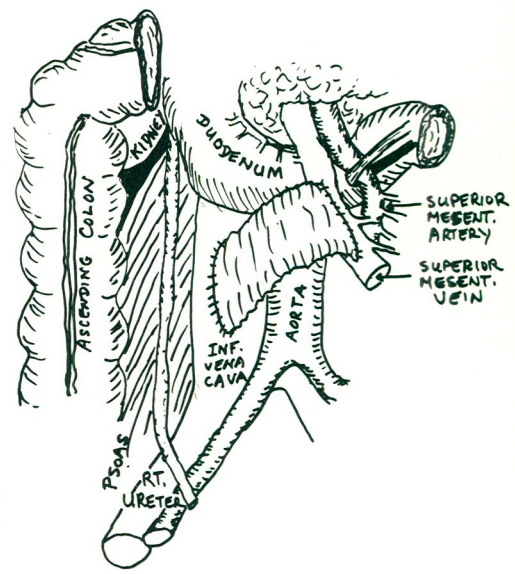
S.H.T., a 43 years old Bahraini female was admitted to Salmaniya Medical Center for investigation of giddiness and burning in the epigastrium of three months duration. She was found to have an enlarged spleen reaching six cms below the left costal margin; liver was not enlarged. She had been admitted in the past for anaemia and blood transfusions. She was a known sickler with reduced G6 PD activity and Hb SA.

Laboratory studies revealed pancytopenia, with haemolglobin of 5 gms%, WBC count of 2600/cu mms, and platelets of 50,000/cu mms; RBC count was 2.4 x 10⁶/cu mms. Liver studies showed total proteins of 7.8 gms % (A/G : 3.4/4.2). Serum Bilirubin : total 1.1 mgs % (conjugated 0.8 mgs %). Alkaline phosphatase : 118 I Us/Litre, S.G.P.T. : 19 I Us/Litre.

Bone marrow revealed a normoblastic picture with iron deficiency. Barium swallow showed oesophageal varices in the lower third of the oesophagus.

In view of the moderate hypersplenism and only moderately enlarged spleen not causing physical discomfort, it was decided to perform a mesocaval shunt to control the portal hypertension hoping that this would also control the hypersplenism by decreasing the size of the spleen. A 20mm diameter dacron graft was interposed between the inferior venacava and the superior mesenteric vein (Fig. 1). Pre-shunt portal pressure was

*Figure 1 The crimped dacron graft is shown in place between the inferior venacava and the superior mesenteric vein. The anastomoses are end to side at both ends.*




* The Department of Surgery,
Salmaniya Medical Center,
Ministry of Health,
State of Bahrain

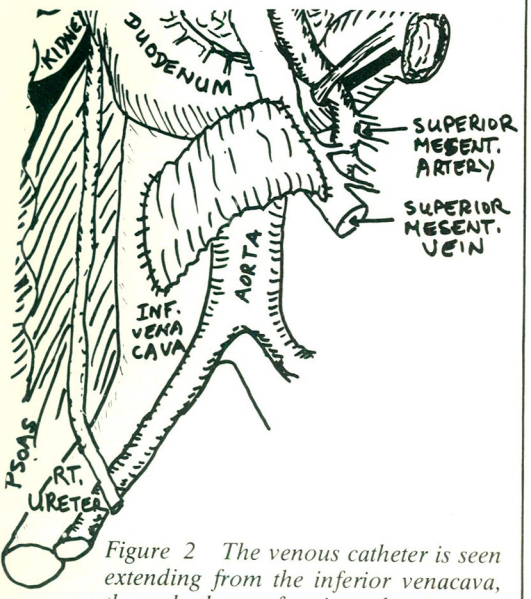



*Figure 2 The venous catheter is seen extending from the inferior venacava, through the graftm into the superior mesenteric vein. The latter is well visualized by the dye. The study indicates patency of the shunt.*

34 cms of water; this dropped to 13 cms of water after the shunt. Wedge biopsy of the liver showed portal cirrhosis. On the first post-operative day the spleen was no longer palpable. On the 3rd post-operative day the splenomegaly recurred but was less than pre-operatively never reaching more than three to four cms below the costal margin. Graft patency was confirmed by an inferior venacavogram (Fig. 2). There was, however, no improvement in the pancytopencia up to four months post operatively. Her Haemoglobin was 12.2 mgs%, WBC count 2300/cu mms, platelets 96000/cu mms and RBC 4.2 x $10^6$. Splenectomy was suggested but she and her family refused to accept further surgery, She is still being followed in surgical clinics.

**Case No. 3**

R.E.E., a 22 years old Bahraini male was admitted to Salmaniya Medical center for melaena of 2 days duration. On admission he had splenomegaly and was discovered to have gastro-oesophageal varices and moderate hypersplenism with WBC count of 2600/cu mms and platelet count of 60,000/cu mms. The spleen was markedly enlarged, reaching the iliac crest and causing physical discomfort. There was a history of jaundice in childhood.

Laboratory investigations revealed a haemoglobin of 14 gms% PCV of 43%, RBC count of 3.88 x $10^6$/cu mms. Liver studies showed total plasma proteins of 7.7 gms% (AG : 4.8/2.9). Serum Bilirubin was less than 1 mgs%; Alkaline phosphatase was 7 I Us/Litre and S.G.P.T. was 32 I Us/Litre, prothrombim time was 14 seconds (85% activity).

Splenectomy and spleno-renal shunt were performed. Portal pressure before the shunt was 33 cms of water but dropped only to 31 cms of water after the shunt. This was ascribed to kinking of the splenic vein. Therefore a mesocaval shunt was performed with an 18 cms dacron graft. This decreased the portal pressure from 31 cms of water to 18. A liver biopsy was performed. It revealed post-necrotic cirrhosis.

The post-operative course was uneventful, platelets shot up to 750,000/cu mms by the 17th post-operative day prophylatic heparinization was instituted and continued till the 24th post-operative day, when the platelet count dropped to 386,000/cu mms. WBC count rose to above 60,000/cu mms but gradually subsided to normal levels.

He was discharged from the hospital on the 37th post-operative day and followed-up in the out-patient clinics. He was readmitted three months later for an acute abdomen due to partial small bowel obstruction, which responded to conservative management. A barium meal, done 11 months post-operatively showed disappearance of the oesophageal varices. Haemoglobin, at this time was 13.3 gms%, PCV 40% and platelets 222,000/cu mms. The patient was feeling very well, had no complaints and indicated that his strength, energy and well-being were much better than they had been for many years.

**DISCUSSION**

Congestive splenomegaly is frequently associated with portal hypertension and in many instances there is sufficient sequestration of peripheral blood elements in the enlarged spleen to result in hypersplenism. The condition is known as Banti's Syndrome and would fall in the group of secondary hypersplenism. There is no true correlation between portal hypertension and splenic size but there is a significant correlation between spelenic size and hypersplenism. A white cell count of less than 4000/cu mms, a platelet count of less than 100,000/cu mms and persistent anaemia not associated with bleeding are considered suggestive of hypersplenism. While the incidence of splenomegaly in portal hypertension varies between 30 and 70% (4), the incidence of hypersplenism in portal hypertension is only 15 to 20% (7). Most of these are in the group with significantly enlarged spleens. Although splenomegaly appears to be a prerequisite for the development of functional hypersplenism in patients with cirrhosis, the mechanism for enlargement of the spleen is not clear. Portal hypertension, circulatory stasis and congestion have traditionally been considered as important factors. However, splenomegaly cannot be explained solely on the basis of portal hypertension and circulatory congestion. Splenomegaly may be absent in chronic and severe cases of portal hypertension and is not regularly reduced by porto-systemic shunting and relief of

congestion in the splanchnic vascular bed. The actual mechanism of hypersplenism is also not very clear. Splenic humoral factors, interfering with bone marrow release of blood cells, have been implicated but this remains conjectural. The depression of blood count has been related to hypervolaemia, disproportionate increase in plasma volume and haemodilution (5). Decreased red cell life has also been implicated (6). The best explanation still provided is based on pooling and sequestration of blood elements in the enlarged spleen (1).

The cytopaenias associated with secondary hypersplenism are effectively corrected by splenectomy. It has been stated that porto-systemic shunting has a beneficial effect on hypersplenism. This apparent benefit is manifested by a reduction in size of the spleen improvement in the cytopaenias. Felix 1974 reported 277 patients with portal hypertension, liver cirrhosis and oesophageal varices (7). 189 out of the 277 had splenomegaly (68%); 41 of these patients had hypersplenism (21%). Of 14% with hypersplenism treated by therapeutic shunt eight improved (57%). Felix also pointed out that new instances of hypersplenism occured in the group treated by medical therapy alone. None of the 172 surgically treated patients developed hypersplenism subsequent to shunt operation when the latter was absent before shunt. The observation suggest that reduction in portal pressure may reverse established hypersplenism and that it will definitely prevent its appearance if not present before shunting. Since the congestion, resulting from portal hypertension, is presumably the cause of splenomegaly and the secondary hypersplenism the question then arises as to whether portal decompression alone will effect a reverals of the syndrome, or whether splenectomy is required. Felix's results indicate that only 50% of patients with this particular complication will show improvement after adequate shunting.

Two of our patients had marked improvement in their pancytopenias after splenectomy alone (Patient No. 1) and splenectomy with porto-systemic shunting (Patient No. 3). Patient No. 2 who had only shunting showed no improvement. Granted this is a very small group from which no valid conclusions can be drawn but the results support the general impression in the literature, namely, that in severe splenic pancytopenia splenectomy is the treatment of choice (3) and porto-systemic shunting should be added only when indicated for control of bleeding from oesophageal varices. Splenectomy alone, though effective in correcting hyper-splenism, is of no significant benefit in portal hypertension and the control of varices. It is known that portal hypertension plays a compensatory role in cirrhosis by helping to maintain blood flow through the liver. It has also been unequivocally established that portosystemic shunting adds further injury by diverting portal and hepatic artery flow from the liver. Its use should, therefore, be limited to the treatment of the life threatening complication of bleeding varices. It does not seem justifiable to subject a patient who has not bled to the hepatocellular insult of shunting when the beneficial effect of the latter on hypersplenism is doubtful. Figure 3 summarizes the consensus on procedures indicated for the various situations in portal hypertension that require surgical intervention. Discussion of the most desirable type of porto-systemic shunt is beyond the scope of this presentation and we wish not to be-labour this point. However, when shunting is indicated in conjunction with splenectomy, it is very tempting to resort to a spleno-renal anastemosis. Though this has proven to provide adequate portal decompression in most cases, technical problems associated with splenic vein kinking, inadequate size and length of the vein, the need to interrupt renal flow temporarily have reduced its usefulness and attractiveness. Meso-canal shunting is being more and more universally accepted as the procedure of choice because of its simplicity and ease of performance.

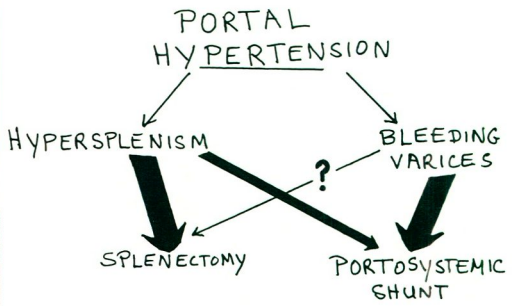



*Figure 3 This schema summarizes the modalities of surgical management in hypersplenism and bleeding oesophageal varices. The procedure of choice in hypersplenism is splenectomy while in bleeding varices it is porto-systemic shunting. The latter may have limited value in the management of hypersplenism. Splenectomy has no place in the treatment of bleeding varices.*

Post-operative thrombocytosis is always seen after splenectomy but the phenomenon is temporary and the platelet count will return to normal in few weeks. We feel it is important to institute prophylactic heparinization for counts above 500,000 to prevent deep vein thrombosis and pulmonary embolism. The WBC count may also, on occasion, rise to very high levels. Both rises are due to bone marrow hyperactivity in response to consumption by the spleen, of formed elements in blood. Following splenectomy it takes time for the bone marrow to adjust to the decreased demand and slow down the output of platelets, white cells and red cells.

## SUMMARY

Splenectomy is the treatment of choice in controlling hypersplenism complicating liver cirrhosis and portal hypertension. It has the added benefit of eliminating the physical discomfort associated with the very large spleens that many of these patients have. Porto-systemic shunting alone seems to have limited value in the control of hypersplenism but should be added to splenectomy when control of portal hypertension is indicated for bleeding varices.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*A view of the distinguished guests present on the occasion of the Bahrain Medical Society Research Awards held at the Regency Intercontinental on 10th December, 1981.*