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# Role of splenectomy in the management of portal hypertension

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The problem of portal hypertension and its alarming complications is still attracting the attentions of surgeons and physicians all over the world. Portal hypertension usually follows obstruction to the portal blood flow somewhere along its course. The normal portal pressure varies between 10-15 cm water, and this is greatly raised in conditions which obstruct the portal blood flow, leading to a distinctive pattern of symptoms. Bilharzial hepatic fibrosis is the commonest cause of portal hypertension in Egypt, while in the U.S.A. portal hypertension is mainly caused by cirrhosis of the liver, and the most common form is that due to alcoholism. The possibility of portal hypertension should be always considered in any patient who presents with severe haematemesis and melena. Whatever the cause of portal hypertension, the clinical consequences are the same and these include: Ascites, Splenomegaly. The formation of systemic anastomotic channels particularly in the submucosa of the lower oesophagus (oesophageal varices), which may rupture leading to fatal haemorrhage, and Hepatic encephalopathy. Oesophageal varices occur in about 67% of patients with advanced cirrhosis, cause haematemesis in 25% of cases, and indeed are the principal cause of death in almost this same number. Varices may also develop in the anorectal region, thus a third to a half of patients with cirrhosis, develop haemorrhoids. The management of portal hypertension is directed mainly to oesophageal varices whether bleeding or silent. Acutely bleeding varices is an emergency situation that necessitates rapid action, resuscitation of the patient should be started as early as possible without delay. Initial steps usually consists of: intravenous infusion, blood transfusion, ice saline lavage of the stomach, measures to treat or prevent hepatic encephalopathy, and oesophagoscopy as soon as the patient become stable. Active measures to stop variceal bleeding include: A) Non operative measures: vasopressin therapy, balloon tamponade, injection sclerotherapy, and transhepatic embolization. Persistent bleeding at this time usually is an indication for surgery. Recently electrocautery and laser photocoagulation have been used with success to control bleeding varices. B) Emergency operative management: emergency portacaval shunt, variceal ligation, and oesophageal transection. Elective operative management of portal hypertension associated with non bleeding varices include, portasystemic shunts, or devascularization operations. Portasystemic shunts may be total shunt as, portacaval, central splenorenal and mesocaval shunt, or selective shunt as distal splenorenal and inokuchi shunt. De vascularization operations include: Tanner's

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operation, its modification by Abu-Zikry, and splenectomy and left gastric vessels ligation. Bilharzial hepatic fibrosis accounts for about 70% of all forms of cirrhosis in Egypt. Nutritional deficiencies are common contributing factors (Abdel Wahab, 1978). The effect of splenectomy on the level of portal venous pressure has been subjected to much controversy. In the presence of bilharzial hepatic fibrosis the spleen is commonly hugely enlarged, acting as a mechanical hinderence to the lungs and to the activity of the patient in general. Splenectomy obviates all these mechanical effects, it seems that removal of the spleen alone is to be of value in removal the ill-effects of hypersplenism, to relieve stitching pain of perisplenitis and protection against rupture of such a friable huge spleen (Abdel-Aleem, 1978). The operation of splenectomy is not enough for the treatment of haematemesis resulted from oesophageal varices, and it should be accompanied by dealing with the varices around the lower part of the oesophagus and upper part of the stomach. -However in non bleeders many authors claim definite value of splenectomy. (El Charabawi, 1979). Some authors state that removal of the spleen cuts away from the portal circulation an amount of blood which amount at least to one third of the portal blood, making it easier for the obstructed intrahepatic circulation to deal with the blood coming from the rest of the portal bed. If this explanation had been true the portal tension would shown a marked DROP after the removal of the spleen (Askar, 1962; Whipple, 1945; Edan, 1947; Sherlock, 1965; and Hachlas, 1958). Hassab, (1964), mentioned that splenectomy plays a definite role in minimizing the incidence of bleeding from oesophageal varices secondary to bilharzial hepatic fibrosis as it is followed by reduction of portal blood volume by -3-40% and this helping venous drainage of the oesophageal varicosities. Also Abdel Wahab et al., (1978), noticed decreased incidence of haematemesis following splenectomy and even when this occurred the attacks were minor and did not endanger the life of the patient. Some authors state that the spleen acts as arteriovenous fistulae and thus splenectomy resulted in decrease of portal blood pressure (Arab et al., 1970; Foda and Zaki, 1959; El-Gendy, 1978). Others concluded that the effect of splenectomy on portal tension is transient resulted in initial DROP of portal pressure which does not last long and it was found to be readjusted to its original preoperative level (Askar, 1962; Sherif, 1964; Way, 1981). Splenectomy is the treatment of choice for portal hypertension in case of isolated splenic vein occlusion. (Rintoul, 1978; Sobeih, 1982; Salam et al., 1973; Yale et al., 1971; Sutton et al., 1971).