Editorial

CURRENT CONCEPTS IN PORTAL HYPERTENSION

Considerable progress has been made during the last two decades in visualisation of portal vein, yet the management of Oesophageal varices and cirrhosis remains unsatisfactory.

Portal hypertension is usually classified into sinusoidal and intrahepatic types (Sherlock 1974). In presinusoidal type, obstruction is due to proliferation of Kupffer and other cells and is associated with relatively normal hepato-cellular function. The intrahepatic portal hypertension on the other hand is associated with hepato-cellular disease and hamorrhage and liver failure is more frequent.

Presinusoidal portal hypertension could be due to extrahepatic obstruction of portal vein such as neonatal sepsis, pyclephlebitis, tumours and thrombosis while intra-hepatic obstruction is the result of the lesions in the portal zones within the sinusoids of the liver. This may occur in reticuloendothelial diseases, sarcoidosis and congenital hepatic fibrosis (Sherlock 1978). Primary biliary cirrhosis may also present as intra-hepatic portal hypertension (Zeegan et al., 1969; Kew et al., 1971). Inorganic arsenic has been known to cause injury to intra-hepatic portal veins leading to fibrosis and portal hypertension (Morris et al., 1974). Similarly inhalation of sprays containing Copper (Pimental and Menzes, 1977) and exposure to the vapour of vinyle-chloride (Thomas et al., 1975) leads to fibrosis and sclerosis of portal venules. Portal hypertension associated with malaria (Butler et al., 1973) and Felty's syndrome (Blendis et al., 1970) is probably due to lymphocytic infiltration of sinusoids in portal zone. Many patients who were thought to be suffering from idiopathic portal hypertension have intrahepatic portal venous sclerosis.

Intrahepatic portal hypertension occurs in cirrhosis and obstruction to portal venous system is at all levels. In Budd-chiari syndrome obstruction occurs at post sinusoidal level (Tavill et al., 1975).

Portal venous pressure seems to play an important role in bleeding from Oesophageal varices. Gastro-Oesophageal reflux does not seem to be an important factor in bleeding, as biopsies of Oesophageal mucosa at the time of bleeding do not show any change (Orlaff and Thomas, 1963).

Upper gastro-intestinal bleeding in patients with cirrhosis may occur from duodenal ulcer, gastric erosions and Mallory-Weis syndrome (Sherlock 1978). Endoscopy will indicate the source of bleeding in a large number of patients if performed within 8 hours of bleeding (Mitchell and Jewell, 1977). Bleeding from Oesophageal varices was common in patients with cirrhosis without hepatic failure, usually associated with ingestion of analgesics while in patients with decompensated liver acute mucosal ulcer were more frequently seen (Sherlock 1978). If the source of bleeding is not visualised, measurement of wedge hepatic venous pressure is indicated.

The management of bleeding Oesophageal varices consists in administration of blood, Vitamin K_1 injections, Oral neomycin and avoidance of salt containing infusions and sedatives (Sherlock 1978). Cimetidine is recommended routinely in these patients to reduce gastric acidity (Sherlock 1978). Vasopression is useful where general measures fail to control the bleeding and is given as 20 units in 100 ml glucose over 10 minutes or it can be given as continuous infusion of 40 units over 60 minutes. Infusion of vasopressin is supposed to provide prolonged drop in portal pressure and cause less substernal discomfort and abdominal pain (Thomford et al., 1975). Continuous intravenous vasopressin infusion seems to be the method of choice but has only limited value and does not seem to improve prognosis.

Sengstaken compression tube has produced cessation of bleeding when meticulous care was taken in positioning the tube and a fourth lumen which is now available helps in pharyngeal aspiration (Andersen and Agger, 1976). The Sengstaken tube is associated with numerous complications and is useful in limited situations as an emergency measure prior to surgery.

Percutaneous transhepatic obliteration of oesophageal varices has been recently used by injecting human thrombin and gelatin foam (Scott et al., 1976). This method has been used in patients with decompensated liver where surgery has not been possible.

The aim of surgery in portal hypertension is to reduce the portal pressure. The porto-caval shunt does prevent bleeding from oesophageal varices but the improvement in long term survival is not significant (Conn 1974). The incidence of encephalopathy in these shunts is higher (Mutchnick et al., 1974) and there is continuous deterioration in hepatocellular function. The mortality of emergency porto-caval shunts remains high (Orloff et al., 1975). Mesocaval shunt with Dacron 'H' graft is currently in vogue and is suitable in low risk patients with low mortality and lesser risk of severe encephalopathy (Darpanas et al., 1975). Selective shunts such as splenorenal shunt has an advantage of decompressing the portal system and maintaining the portal venous

blood flow. The incidence of encephalopathy and deterioration in hepatic function is less pronounced in selective shunt as compared to mesocaval shunt (Galambos et al., 1976) although the mortality in selective shunt is higher.

References

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