BACTERIAL PERITONITIS FOLLOWING OESOPHAGEAL INJECTION SCLEROTHERAPY FOR VARICEAL HAEMORRHAGE

Thesis

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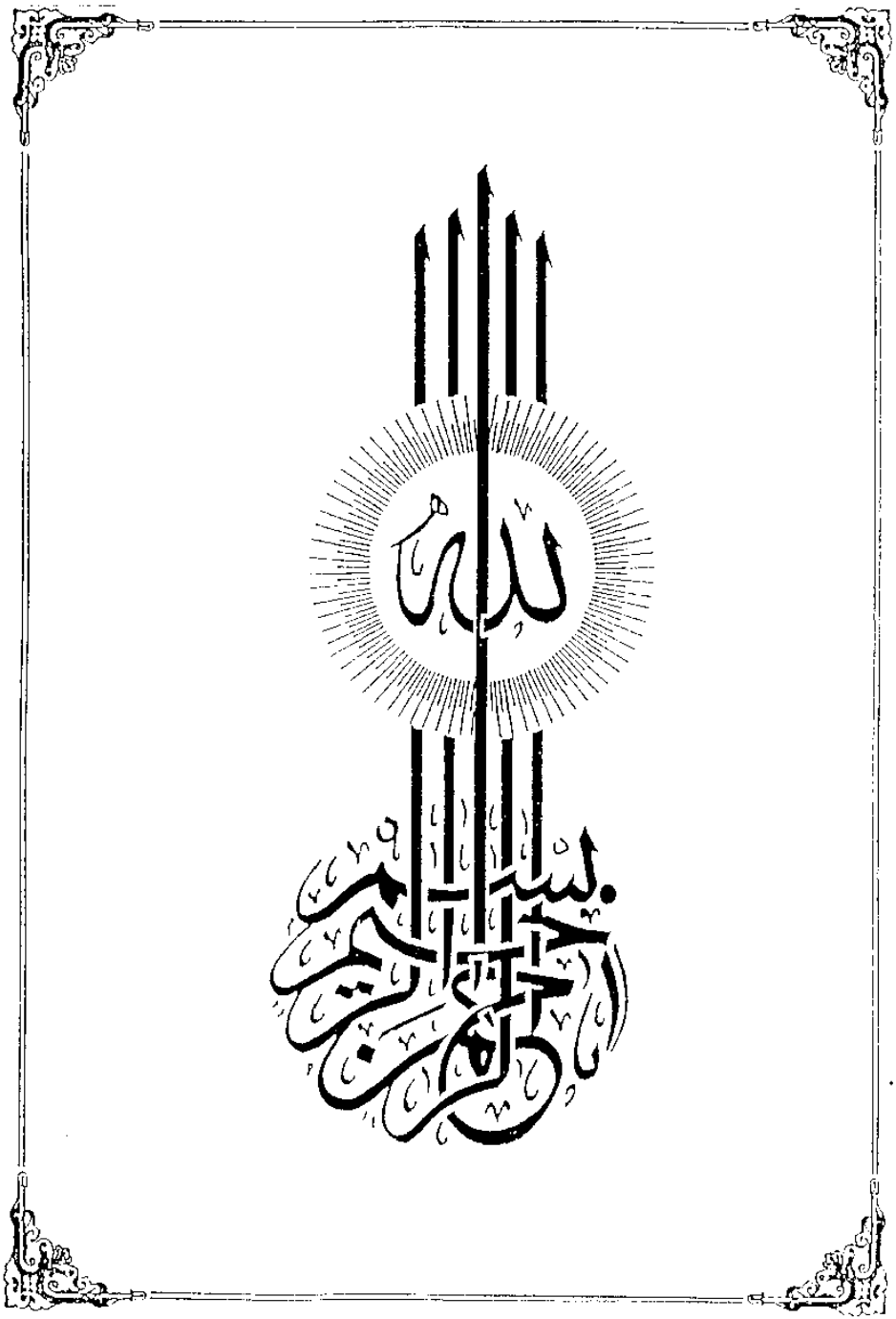

TO MY FATHER

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INTRODUCTION :

Oesophageal varices, because of their tendency to cause massive gastrointestinal haemorrhage, are the more relevant part of the portosystemic collateral circulation that develops in response to the chronic increase in the portal pressure in patients with cirrhosis and portal hypertension. Although the mechanism of variceal bleeding is not fully understood, increased portal and variceal pressures are thought to be of major importance (Garcia-Tsao et al., 1985).

Oesophageal varices constitute one of the major and most serious sequalae of schistosomiasis in Egypt. Variceal haemorrhage continues to be a frustrating entity for physicians to treat.

Endoscopic injection sclerotherapy has become the principal method for treatment of oesophageal variceal haemorrhage (Tam et al., 1990). The procedure is frequently associated with bacteraemia (Brayko et al., 1985). Bacterial peritonitis has recently been reported as a complication of sclerotherapy (Barnett and Elta, 1987).

This can be explained by possible haematogenous seeding of susceptible ascites, microperforations of the oesophagus resulting from injection sclerotherapy and defective immune response with decreased opsonization activity in patients with cirrhosis and ascites (Schembre and Bjorkman, 1991). Lai et al. (1986) proposed that distension of the bowel by air-feeding during sclerotherapy, in addition to gastro-intestinal bleeding, may increase the intestinal permeability of enteric bacteria, thus resulting in bacterial peritonitis.

Spontaneous bacterial peritonitis is also a well recognized compliction of severe liver disease with ascites, regardless of variceal bleeding.

AIM OF THE WORK :

The aim of this work is to evaluate the incidence of bacterial peritonitis following injection sclerotherapy in patients presenting with bleeding oesophageal varices and ascites and, also, to revise the incidence of spontaneous bacterial peritonitis in patients with liver cirrhosis and ascites on medical treatment.

REVIEW OF LITERATURE

PORTAL HYPERTENSION

Definition :

Portal hypertension is defined as an increase of portal pressure above the normal range of 2-6 mm Hg. The development of oesophageal varices as a part of the portal systemic collateral network and -of course- the occurrence of variceal haemorrhage requires portal pressure to exceed the threshold of 12 mm Hg (Garcia-Tsao et al., 1985).

Pathogenesis :

Two theories have been developed to explain the pathogenesis of chronic portal venous hypertension :

a. Increased blood flow : increased portal vein blood flow in some patients with tropical splenomegaly, hepatic arterial-portal and splenic arterial venous fistula appears to cause portal hypertension only when the vascular resistance in the liver has increased.

b. Increased resistance : an increase in vascular resistance is the most common cause of portal hypertension. The portal venous system lacks valves, and any increase in pressure is transmitted quantitatively to structures on

the intestinal side of obstruction.

(Zakim and Boyer, 1982)

Sherlock (1989) classified portal hypertension into two main groups :

I. Presinusoidal . Extrahepatic
. Intrahepatic

II. Hepatic . Intrahepatic (sinusoidal)
. Post sinusoidal

I. PRESINUSOIDAL PORTAL HYPERTENSION

It is also classified into extra and intrahepatic subdivisions :

A. The extrahepatic presinusoidal P.H. :

It is usually due to obstruction some where along the course of the portal or splenic veins, the causes may be :

. **Infections :** abdominal infections, septicaemia, ulcerative colitis, Crohn's disease and portal pyaemia.

. **Post operative :** after splenectomy and biliary surgery.

. **Hypercoagulable state :** polycythaemia rubra vera, myeloproliferative diseases and paroxysmal nocturnal haemoglobinuria.

. **Carcinoma :** pancreatic and hepatocellular carcinoma.

. **Congenital abnormality :** can occur any where along the line of the right and left vitelline veins from which the portal vein develops.

. **Miscellaneous :** portal vein thrombosis has been associated with pregnancy, oral contraceptives, retroperitoneal fibrosis, haemangioma of the spleen, puerperium and thrombophlebitis migrans.

. **Idiopathic.**

(Sherlock, 1989)

B. The intrahepatic presinusoidal P.H. :

. **Schistosomiasis :** ova reach the minute-portal venous radicles in the portal tracts and cause granulomatous reaction with periportal fibrosis.

. Infilteration of the portal zones with the haemopiotic tissues in myeloproliferative diseases like; myelosclerosis, myeloid leukaemia, Hodgkin's disease and systemic mastocytosis.

. Primary biliary cirrhosis and sarcoidosis, due to portal zone infilteration and narrowing of the sinusoids.

. Congenital hepatic fibrosis.

. Felty's syndrome and chronic malaria, due to lymphocytic

infilteration of sinusoids and portal zone.

. Toxic fibrosis : Induced by injurious substances, e.g.

. Arsenic used in treatment of psoriasis.

. Sprays containing copper, vinyl chloride.

. Cytotoxic drugs (6-mercaptopurine, azathioprine and methotrexate) used as immunosuppressant with renal transplant can lead to presinusoidal fibrosis.

(Sherlock, 1989).

II. Hepatic portal hypertension (Sinusoidal & postsinusoidal)

Cirrhosis :

Hepatic cirrhosis is responsible for the majority of cases of portal hypertension (Shearman and Finalyson, 1982).

In liver cirrhosis the portal hypertension can be produced by the following mechanisms :

a. Sinusoidal narrowing resulting from; deposition of collagen in the space of Disse, hepatocyte swelling and contractile myofibrils in the space of Disse could also play a role (Sherlock, 1985). Also, regenerating nodules compress the sinusoids and hepatic veins, the nodules increase resistance to blood flow through the liver and are the main reason for portal hypertension (Shearman and Finalyson, 1982).

b. Arteriovenous anastomosis, which transmit the hepatic arterial pressure to the portal venous system. These are certainly present in cirrhotic liver. This is known as arteriolization of the liver (Sherlock, 1985).

Other causes :

1. Hepatic venous obstruction (Budd Chiari syndrome) :

   It is a post-sinusoidal intrahepatic P.H., the block is in the small hepatic venules until the entery of I.V.C. into right atrium and may be due to venous thrombosis, tumours, congenital web of I.V.C. or fibrous occlusion of the orifices of the main hepatic veins without vena caval involvement (Safouth et al., 1965).

2. Veno-occlusive diseases, it is due to thrombophlebitis of the minute hepatic venous radicles leading to intrahepatic post- sinusoidal P. H. (Sherlock, 1985).

3. Non-cirrhotic nodules, these nodules may be found in : Felty's syndrome, monoclonal gammopathy and hyperviscosity syndromes (Sherlock, 1989).

4. Portal nodular hyperplasia : The perihilar region is replaced by nodules (Sherlock, 1985).

5. Cystic liver disease (Shearman and Finalyson, 1982).