EVALUATION OF THE EFFECTIVENESS OF ENDOSCOPIC SCLEROTHERAPY OF OESOPHAGEAL VARICES BY ENDOSONOGRAPHY

Thesis
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قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا
عَلَّمْتَنَا إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ.

صَدَقَ اللهُ الْعَلِيُّ
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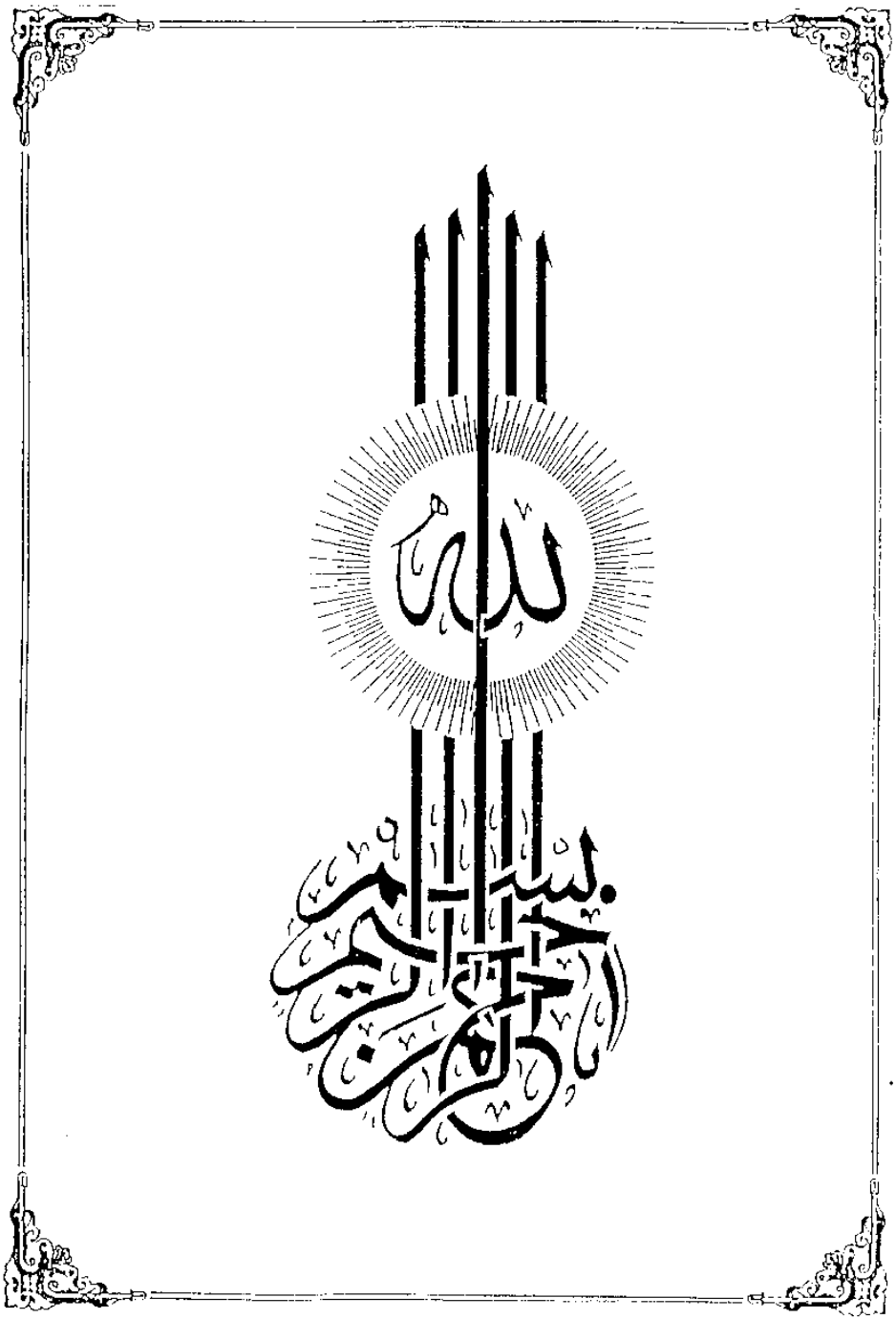

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INTRODUCTION & AIM OF THE WORK

INTRODUCTION

Oesophageal varices is a major medical problem. It develops in about 14-77 % of patients with cirrhosis depending upon the type of cirrhosis and the degree of portal hypertension ***(Sherlock, 1997).***

Haemorrhage from oesophageal varices is a catastrophic complication of portal hypertension requiring immediate and aggressive therapy. Despite available therapeutic options the mortality rate associated with acute variceal haemorrhage may approach 25-40 % ***(Bernuau & Rueff, 1985).***

Oesophageal sclerotherapy is the treatment of choice for bleeding oesophageal varices, it is effective for treating acute variceal bleeding as well as erradicating oesophageal varices for secondary prevention of bleeding ***(Arnon et al., 1993).*** It controls haemorrhage in 90-95 % of patients and enhance survival ***(Gregory, 1990).***

Endoscopic ultrasonography (E.U.S.) has an important role in the diagnosis of different oesophageal lesions. Concerning portal hypertension, by using (E.U.S.) before and after injection sclerotherapy, certain changes in the varices may be demonstrated. And because of the high resolution obtained close-up, this method allow visualization of intramural vessels in the lower oesophagus and upper part of the stomach ***(Ziegler et al., 1991).*** So it may be useful in assessing the effectiveness of sclerotherapy and revascularization after completion of a course of injection procedures ***(Yasuda et al., 1990) & (Tio et al., 1995)*** and to denote the end point of sclerotherapy.

AIM OF THE WORK

To assess the role of endoscopic ultrasonography (E.U.S.) in monitoring the success or inadequacy of sclerotherapy in the treatment of oesophageal varices.

REVIEW OF LITERATURE

ANATOMICAL BACKGROUND OF PORTAL TREE

The liver has a double blood supply, the hepatic artery, branch of the coeliac axis supplying arterial blood and the portal vein supplying venous blood from the abdominal part of the alimentary tract, spleen, pancreas and gall bladder. The liver drains its blood into the I.V.C. via the hepatic veins ***(Sherlock and Dooley, 1997).***

The main veins which are responsible for the formation of the portal system are : the portal vein, the splenic vein, the superior and inferior mesenteric veins ***(Sherlock and Dooley, 1997).***

The portal vein is formed by the union of superior mesenteric vein and the splenic vein at the level of the second lumber vertebra just posterior to the head of the pancreas and in front of the I.V.C. it is about 5.5-8 cm long and 1.2 cm in diameter and contains no valves in its larger branches ***(Sherlock and Dooley 1997).*** It passes upwards and slightly to the right at first behind the gastroduodenal artery and common bile duct separating it from the first part of the duodenum, then it lies in the free

margin of the lesser omentum behind the hepatic artery and common bile duct separated from the I.V.C. by the epiploic foramen. At the porta hepatis, it divides into right and left branches forming an angle of 90^0 with each other. The right branch receives the cystic vein, the left branch gives twigs to caudate and Quadrate lobes. It is joined by the paraumbilical veins and by the ligamentum teres (remnant of the left umbilical vein) which runs in the free edge of the falciform ligament. It is connected to the I.V.C. by the ligamentum venosum (a slender remnant of the ductus venosus of the fetus) ***(Warnick and Williams, 1975).***

The portal vein has a segmental intrahepatic distribution which is not consistent with the apparent separation of the liver into right and left lobes by the insertion of the falciform ligament ***(Sherlock and Dooley, 1997).***

Although there is no anastomosis between microscopic branches, large intercommunication at the level of the hepatic sinusoids exist ***(Rappaport, 1987).***

The superior mesenteric vein

Formed of tributaries from the small intestine, coecum, ascending and transverse colon and the head of pancreas and irregularly from the stomach via gastroepiploic vein. It ascends in the root of the mesentry until the neck of the pancreas to meet the splenic vein ***(Grander et al., 1975).*** It's diameter is about 0.78 cm in diameter ***(Rappaport, 1987).***

The splenic vein

(5-15 channels) originating at he splenic hilum are joined by the short gastric vessels near the tail of the pancreas to form the main splenic vein. It runs transversely into the body and the head of the pancreas in front and below the splenic artery receiving the gastroepiploic vein, pancreatic tributaries and inferior mesenteric vein at right angle to form the portal vein. ***(Rappaport, 1987).***

Inferior mesentric vein

Bringing blood from the left colon, rectum and upper part of the anal canal usually enters the medial third of the splenic vein ***(Sherlock, 1997).*** But it may sometimes enters the junction of the splenic and superior mesenteric vein ***(Rappaport, 1987).***

Tributaries of the portal vein :

1. The left gastric (coronary) vein:

Running along the lesser curvature at the stomach turn backward at the oesophageal opening of the diaphragm after receiving oesophageal tributaries (which with progressing cirrhosis of the liver it enlarges forming varices). It passes downwards and to the right behind the lesser sac to end in the portal vein ***(Rappaport, 1987).***

2. The right gastric vein

Runs along the lesser curvature from left to right ending in the portal vein. ***(Rappaport, 1987).***

3. The pancreaticoduodenal vein : joins the portal vein behind the superior part of the duodenum.

4. The cystic vein : from the gall bladder and joins the right branch of portal vein after its division in the portahepatis.

5. The paraumbilical veins : small veins passes along ligamentum teres to end in the left branch of portal vein.

PORTAL HYPERTENSION

Definition of Portal Hypertension

Portal hypertension is a clinical syndrome which is characterized by pathological increase in portal venous pressure and by the formation of porto-systemic collaterals that divert portal blood to systemic circulation ***(Bosch et al., 1989).*** The normal portal venous pressure in man is about 7mmHg ranging between 5-10mmHg ***(Reynolds, 1987).***

A complication of portal hypertension are only observed when the gradient between portal pressure and I.V.C. pressure is increased above a threshold value of about 12mmHg ***(Bosch et al., 1992).*** This value therefore defines what is known as clinically significant portal hypertension ***(Bosch et al., 1989 and Groszmann et al 1990).***

Pathophsiology of Portal Hypertension

Normally the portohepatic circulation is highly compliant and accommodates large variations in blood flow with minor changes in pressure. Portal pressure is determined

by the inter-relationship between portal blood flow and the vascular resistance that opposes that flow.

These parameters are mathematically represented by the following equation:-

$$P=QXR$$

where P = Perfusion pressure gradient in portal venous system, difference between portal & I.V.C. pressure.
Q = Portal blood flow,
R = Portal vascular resistance.

(Bosch et al., 1989b).

The normal portal blood flow is about 1000-1200 cc/minute and it is shown that this flow is streamlined rather than turbulent although some crossing of the blood stream does occur. ***(Richardson and Withrington 1981).***

The increased resistance can be located at any point along the splenoportal axis at the intrahepatic circulation or at the vascular resistance is thought to be located at the hepatic sinusoids, although pre-sinusoidal and hepatic vein obstruction have also been suggested ***(Shibayama and Nakata, 1985)*** increased hepatic vascular resistance