COMPARATIVE STUDY OF DIFFERENT LINES OF MANAGEMENT OF BLEEDING OESOPHAGEAL VARICES IN PATIENTS WITH BILHARZIAL HEPATOSPLENIC SYNDROME

THESIS

Submitted in Partial Fulfilment for the
Master Degree of General Surgery

By
ALI SOLIMAN THABET
M. B. B. Ch.

Supervised by

Prof. Dr. REFAAT KAMEL
Prof. of General Surgery

Faculty of Medicine
Ain Shams University

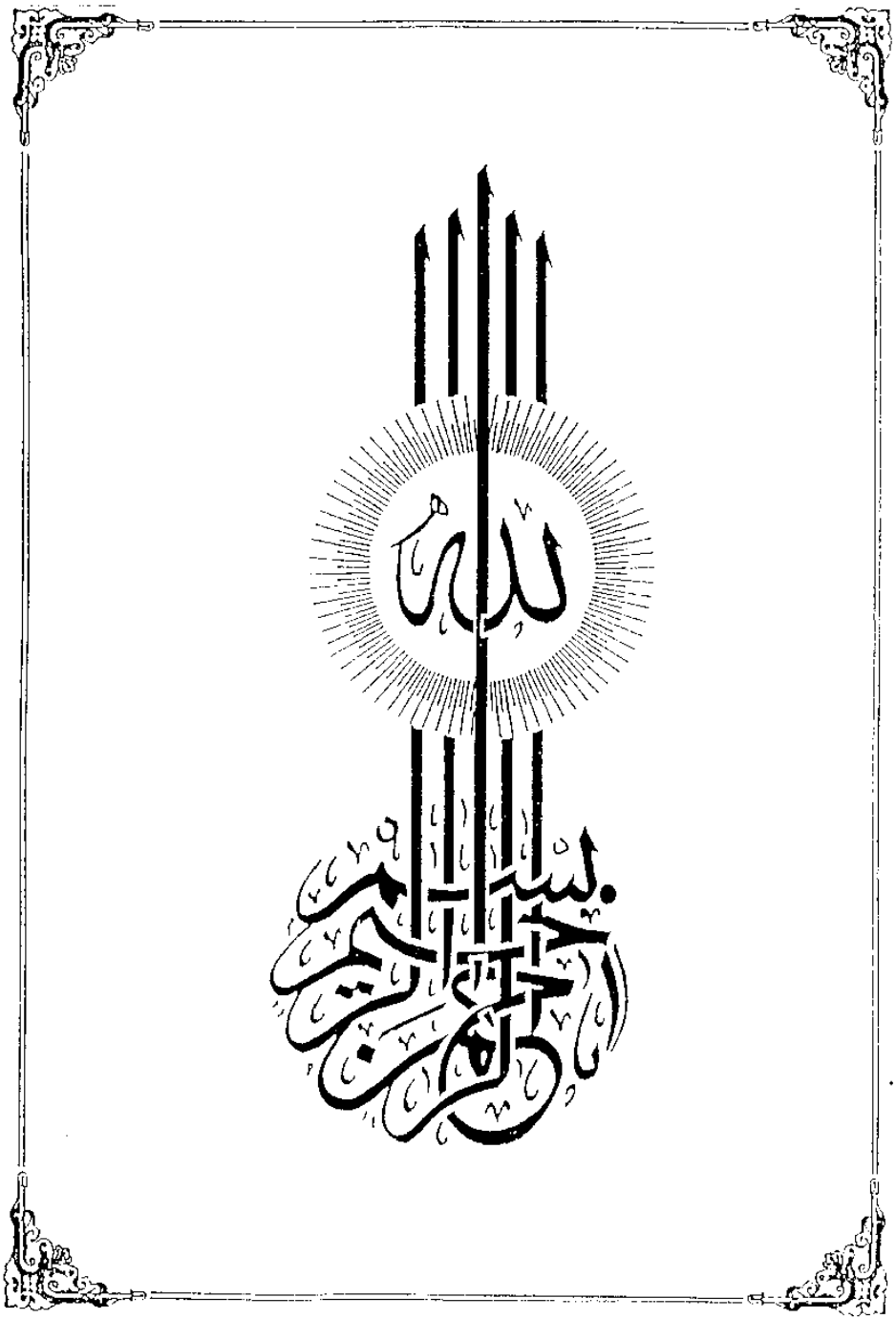
1981

ACKNOWLEDGEMENT

I wish to express my sincere thanks and deepest gratitude to professor Dr. REFAAT KAMEL for his constant encouragement, supervision and revision of this work.

Also, I wish to thank all my colleagues, for their help in performing this work.

- - - -



C O N T E N T S

- - -

INTRODUCTION AND AIM OF WORK

INTRODUCTION

Bleeding from oesophageal varices in the most lethal form of gastrointestinal haemorrhage. In patients with hepatic fibrosis, varix rupture is one of the several serious complications responsible for the high mortality rate of this disease. The major unsolved problem in the management of portal hypertension concerns the emergency treatment of bleeding oesophageal varices, and there are disadvantages in all current therapeutic methods.

The ideal procedure considering mortality and effective control of bleeding, however, still remains in doubt.

This work aims to assess the different methods in current use in the management of bleeding oesophageal varices.

- - -

REVIEW OF LITERATURE

Anatomy of the portal circulation

The portal vein is formed by the union of the splenic and superior mesenteric vein at the level of second lumbar vertebra behind the neck of the pancreas.

It runs for 8 - 9 cm to the hilum of the liver, where it divides into lobar branches.

The coronary vein usually enters the portal vein on its anteromedial aspect just cephalad to the margin of the pancreas; In 25% of cases, the coronary vein joins the splenic vein.

Other small venous tributaries from the pancreas and duodenum are less constant.

The inferior mesenteric vein generally drains into the splenic vein several centimeters to the left of the junction with the superior mesenteric vein; not uncommonly, it empties directly into the superior

mesenteric vein.

In the hepatoduodenal ligament, the portal vein lies dorsal and slightly medial to the common Bile duct. A large lymph node is often encountered lateral to the vein (Du plessis 1975).

The Collateral Circulation

These communications are of great importance especially in cases where the portal vein is obstructed as in cases of cirrhosis of the liver.

(1) The most important communication is at the lower end of the oesephagus and the cardia of the stomach. Where the veins of stomach draining to theportal communicate with the oesephageal veins to the azygos and vena cava.

(11) Around the umbilicus: Veins pass along the falciform ligament to the umbilicus, Connecting the veins of the liver draining to the portal system with the veins round the umbilicus

namely epigastric veins which are systemic. Enlargement of these may produce a bunch of veins radiating from the umbilicus, which is called the caput medusae.

(111) At the lower end of the rectum:

Three arteries supply the rectum, and the accompying veins drain partly to the systemic and partly to the portal circulation.

The superior haemorrhoidal vein becomes the inferior mesenteric which is portal.

The middle and inferior haemorrhoidal veins empty into the hypogastric which is systemic.

In portal hypertension the veins of the rectum becomes dilated to form haemarrhoid, but this is rare.

(IV) At the Back of the colon:

In front of the kidney small vessels unite with

the vessels of the peritoneum and colon draining to portal circulation with the vessels of the kidney draining into the systemic circulation.

(V) Bare area of the liver:

Small vessels unite the diapharagmatic veins draining to the systemic circulation with the liver veins draining to the portal circulation.

HIDAYAT (1962) reported that the oesophag-ogastric varices are the most important because of their great liability to produce haemorrhage. They fall into the following groups:

1- Submucous group of veins in the lower third of the oesophagus and the proximal part ofthe stomach.

They follow a longitudinal course raising the mucous membrane in three or four parallel ridges which may be seen in oesophagoscopy or by barium swallow.

These veins are those more subjected to the formation of sacculation and varices.

11- Extrinsic group of veins develop in late cases and are lying in the retroperitoneal tissue outside the oesophagus and stomach. They form a complex network of dilated channels.

This set is beneficial since it help in nature's attempt at shunting the portal blood into the caval system without carrying by itself any danger.

111- Communicating veins which exist between the two previous sets and connect them by short tributories which pass through the wall of the stomach and oesophagus.

Aetiology of portal hypertension

Rains (1975), described the causes of portal

hypertension according to the site of obstruction:

1- Prehepatic: About 20% of patients belong to this group. The obstruction arises in one of two ways. The first is congenital absence or abnormality of the portal vein, the second is thrombosis of the portal vein due to extension of the normal obliterative process of the umbilical vein or may be due to sluggish circulation in the portal vein.

11- Intrahepatic: account for nearly 80% of all cases. The cause is liver cirrhosis or Bilharzial liver fibrosis which account for 80% cases of portal hypertension in EGYPT (Kamel,1965).

111- Post hepatic: very rare. It may be caused by constrictive pericarditis and tricuspid valvular incompetence, and it is also a component of Budd-Chiari syndrome which is a veno

occlusive disease affecting the hepatic veins.

The Basic lesion is increased resistance to the portal flow whatever the aetiological factor.

Kamel (1965), stated that hepatic bilharziasis is mainly a vascular affection and more than two thirds of these patients get portal hypertension.

He also reported that the main cause of portal hypertension is a presinsoidal obstruction of the portal veins tributaries, so that the intrasplenic pressure is high while the wedged hepatic pressure is normal.

Pathology of Bilharzial Hepatosplenic Syndrome:

Mousa (1964), described the pathological changes of hepatic bilharziasis.

He stated that these changes pass through 2

stages: an early infiltrative stage in which ova are impacted in the main or fine portal tracts, usually around portal tributaries and excite a histocytic and eosinoplilic reaction but the liver parenchymal cells show no signs of degeneration, necrosis or regeneration. In the second stage of advanced fibrosis, in addition to these changes, there is thickening of the portal tracts with mild cloudy swelling and wasting of the liver cells at the periphery of the lobules.

It is apparent that Bilharzial hepatic fibrosis attains two characteristics which differentiate it from liver cirrhosis: the first one being a presinsoidal lesion and the second is that the parenchyma is spared and shows no signs of degeneration or regeneration.

The lobular pattern of the liver may show some distortion by fibrous tissue. This may be fine or coarse nodularity depending on whether the smaller or larger portal tracts are mainly involved and this in