A STUDY ON A COMPARISON OF THE EFFECT OF SCLEROTHERAPY AND BETA-BLOCKERS ON THE DIAMETER OF THE PORTAL VEIN IN CASES OF PORTAL HYPERTENSION

THESIS

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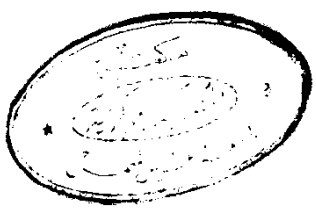
MOHAMED OSAMA EL-SHARKAWY

M.B., B. Ch.



Supervised By

Prof. Dr. YEHIA MOHRAN

Prof. of General Medicine

Ain Shams University

Dr. OMAIMA EL-LAMEI

Ass. Prof. of General Medicine

Ain Shams University

Dr. ZAKARIA MOHRAN

Lecturer of Tropical Medicine

Ain Shams University

FACULTY OF MEDICINE, AIN SHAMS UNIVERSITY

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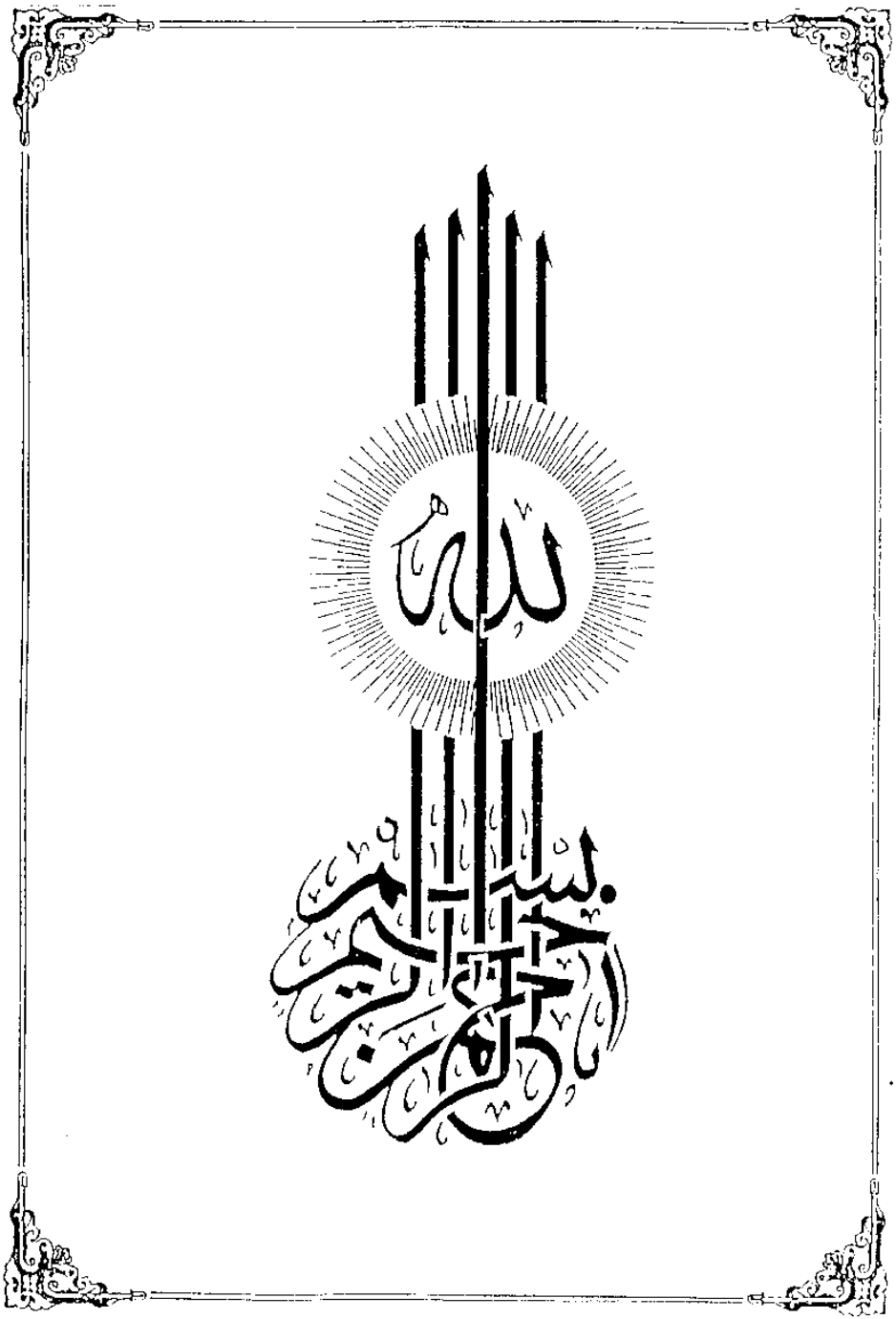

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INTRODUCTION
and
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INTRODUCTION AND AIM OF THE WORK

Patients suffering from portal hypertension are very common in Egypt, which is without doubt one of the most serious ailments present in our country, and several complications can occur including life threatening haemorrhage.

Many patients may suffer from schistosomiasis or have had a past attack of viral hepatitis and go on leading a normal life, yet once portal hypertension develops (with its sequelae of oesophageal varices and haemorrhage), the situation becomes different completely .

During the last few years, many methods have been studied for the treatment of bleeding oesophageal varices. Beginning from the various surgical shunts to the medical lowering of portal hypertension by B-blockers and the technique of injection sclerotherapy, many workers have reported various results in a trial to lower the portal pressure and thus guard against subsequent haemorrhage .

The study of the portal system has also passed through several stages ; from strictly invasive techniques (with their serious drawbacks) such as : the estimation of the intrasplenic pressure (Atkinson and Sherlock , 1954) ,

followed by more sophisticated procedures such as wedged hepatic venous pressure (Myers and Taylor, 1951) and the transhepatic measurement of the portal pressure (Okuda et al., 1974) to the non-invasive ones such as ultrasonography and CT scanning . Indeed, the use of non-invasive techniques in such precarious patients are now very much recommended where abdominal ultrasonography has recently been shown to be useful for screening for portal hypertension (Bolondi et al., 1982) .

The recent randomized trials (Macdougal et al.,1982; Terblanche et al., 1983) have favoured sclerotherapy over the established medical regimen, for managing variceal bleeding of portal hypertension .

The idea behind this work began to crystallize after seeing and knowing the size of the problem of portal hypertension in our country and the huge number of patients suffering from portal hypertension who bled spontaneously or after some procedure .

Really, this work had the aim of studying the effect of various methods of treatment of portal hypertension (sclerotherapy and B-blockers on the portal system in general and the diameter of the portal vein in particular using an ultrasound which is a non-invasive technique and tried to compare between the results before and after each method .

REVIEW OF LITERATURE

CHAPTER ONE

An Introduction to Anatomy of Portal System

The liver receives its major blood supply from the hepatic arteries, branches of the common hepatic artery, and the portal vein. Under pathological conditions, the liver may receive arterial supplies from hypertrophied phrenic arteries, but normally such tributaries are not important (Richardson and Withrington, 1981).

The portal system is a system of veins which drains blood from the abdominal part of the alimentary tract (except the lowest part of the rectum and the anal canal), the spleen, the pancreas and the gall bladder. The main veins which are responsible for the formation of this system are the portal vein, the splenic vein, the superior and inferior mesentric veins (Sherlock, 1981).

The tributaries of the portal vein discharge their blood into the sinusoids of the liver where it is only separated from the liver cells by a single layer of phagocytic endothelial cells. The portal vein carries the

products of digestion of carbohydrates and proteins to the liver and contains in its various tributaries and branches up to one third of the total volume of blood in the body (Romanes, 1975). The portal vein contributes 72% of the total oxygen supply to the liver (Sherlock, 1981).

In spite of anatomical variations in the various branches of the portal system, the portal vein itself usually begins at the level of the second lumbar vertebra (posterior to the junction of body and head of pancreas) at the union of the splenic and the superior mesentric veins.

It then ascends posterior to the superior part of the duodenum and behind the bile duct and the hepatic artery, where it receives a variable number of small veins. It ends at the porta hepatis by dividing into two branches, one to each of the corresponding lobes of the liver. The right branch is usually joined by the cystic vein before its enterance into the liver. The left branch gives branches to caudate and quadrate lobes and is also connected to a fibrous cord, the ligamentum teres, which is a remenant of the obliterated left umbilical vein running in the free border of the falciform ligament (Last, 1973).

The small paraumbilical veins run together with the ligamentum teres and connect the portal vein with veins around the umbilicus. These may become prominent in cases of portal hypertension. A second fibrous cord, the ligamentum venosum is a vestige of the obliterated ductus venosus and connects the inferior vena cava with the left portal vein (Davies and Coupland, 1969).

The splenic vein begins by five or six tributaries issuing from the spleen. Such tributaries are then joined by the short gastric veins to form a single vessel. It then descends to the right (across the posterior abdominal wall) where it receives numerous short tributaries from the pancreas. It usually receives the inferior mesentric vein at a right angle and after that unites with the superior mesentric vein to form the portal vein (Warmick and Williams, 1975).

The superior mesentric vein is very variable , having from ten to twenty five tributaries. It collects blood from the small intestine, the caecum, the descending and transverse part of the colon. It usually begins in the right iliac fossa by the union of its numerous tributaries (jejunal veins , ilial veins, ileocolic vein, right colic vein, middle colic vein, right gastroepiploic

vein and pancreaticodudenal vein) and ascends in the mesentry until the neck of the pancreas to meet the splenic vein (Gardner et al., 1975) .

The inferior mesentric drains blood from the rectum, the sigmoid and the descending parts of the colon. Starting as the superior rectal vein in the rectum it continues upwards and ends in the medial third of the splenic vein but may sometimes enter the junction of the splenic and superior mesentric veins (Romanes, 1969).

The Portal Blood Flow :

The normal portal blood flow is about 1200 ml/minute (25 per cent of the cardiac output) and work done on experimental animals has shown that this flow is stream-lined rather than turbulent, although some crossing of the blood stream does occur (Richardson and Withrington,1981).

Three quaters of the portal venous blood is derived from the mesentric circulation and one quarter from the splenic circulation. The mesentric blood flow tends to reach the right hepatic lobe while splenic blood pass to the left lobe (Groszmann et al., 1972; Kashiwagi et al., 1974) .

Normal portal haemodynamics depend on the relationship between the blood flow, the pressure to the flow and resistance to flow (Beker, 1976).

An increase in the vascular resistance in the portal system is the most common cause for portal hypertension.

Site localization for obstruction of blood flow :

1. Prehepatic lesions of the splenic and/or portal veins.
2. Intrahepatic, presinusoidal lesions which obstruct the blood flow through the terminal branches of the portal vein.
3. Parasinusoidal and postsinusoidal lesions which obstruct the blood through sinusoids and hepatic vein tributaries.
4. Posthepatic lesions of the major hepatic veins (Jeffries, 1979).

Any permanent increase in resistance to portal blood flow produces a dilatation of the vascular bed reflected by:

1. Decrease in the velocity of portal flow resulting in prolongation of portal circulation time.

2. Congestion of the organs drained by the portal vein, principally the gastrointestinal tract and fundamentally the spleen resulting in congestive splenomegaly (Beker, 1976).

The conditions which cause massive splenomegaly, in which splenic blood flow accounts for 10 to 20 per cent, and sometimes for as much as half of the cardiac output may result in an increase in the splanchnic blood flow. Such conditions include tropical splenomegaly, Felty's syndrome, chronic lymphatic leukaemia, polycythemia rubra vera and vascular disorders of the spleen, and the associated portal hypertension is often predominantly of the presinusoidal type. No obstruction can be found in the portal vessels radiologically and bleeding may occur from oesophagogastric varices (Garnett et al., 1969).

Embryology of The Portal Vein :

The portal vein is derived from the omphalomesenteric vein which brings blood from the yolk sac and intestine to the liver. The omphalic portion of the vein regresses with disappearance of the yolk sac. With the growth of the intestine, the mesenteric portion persists and become the tributaries of the portal vein.

Its stem is formed by the omphalomesenteric trunk arranged in a figure of 8 around the first and third portions of the duodenum. Its spiral course is formed by dropping out of the posterior (right) limb of the 8 below and the anterior (left) limb of the 8 above (Rappaport, 1975) .

Anomalies of The Portal Vein:

Anomalies of the portal vein are rare but have been reported and may result in portal hypertension which can be fatal. Snavely and Breakell (1954) reported the case of a 13 year old patient presenting with an enlarged spleen, elevated intrasplenic pressure and severe haematemesis which turned out to be fatal. The post mortem showed two separate portal veins with marked narrowing at the points of junction of the splenic and the superior mesentric veins to the portal vein.

A prepancreatic portal vein (lying anterior to the pancreas) associated with biliary stresia was found in a patient with portal hypertension and jaundice since birth (Renner and Child, 1963).

Other rare anomalies of the portal vein include agenesis of the vein, a bifid vein, valves present in