CORRELATION OF ULTRASONOGRAPHIC FINDINGS WITH ENDOSCOPY IN PORTAL HYPERTENSION IN EGYPTIAN CIRRHOTIC PATIENTS

THESIS

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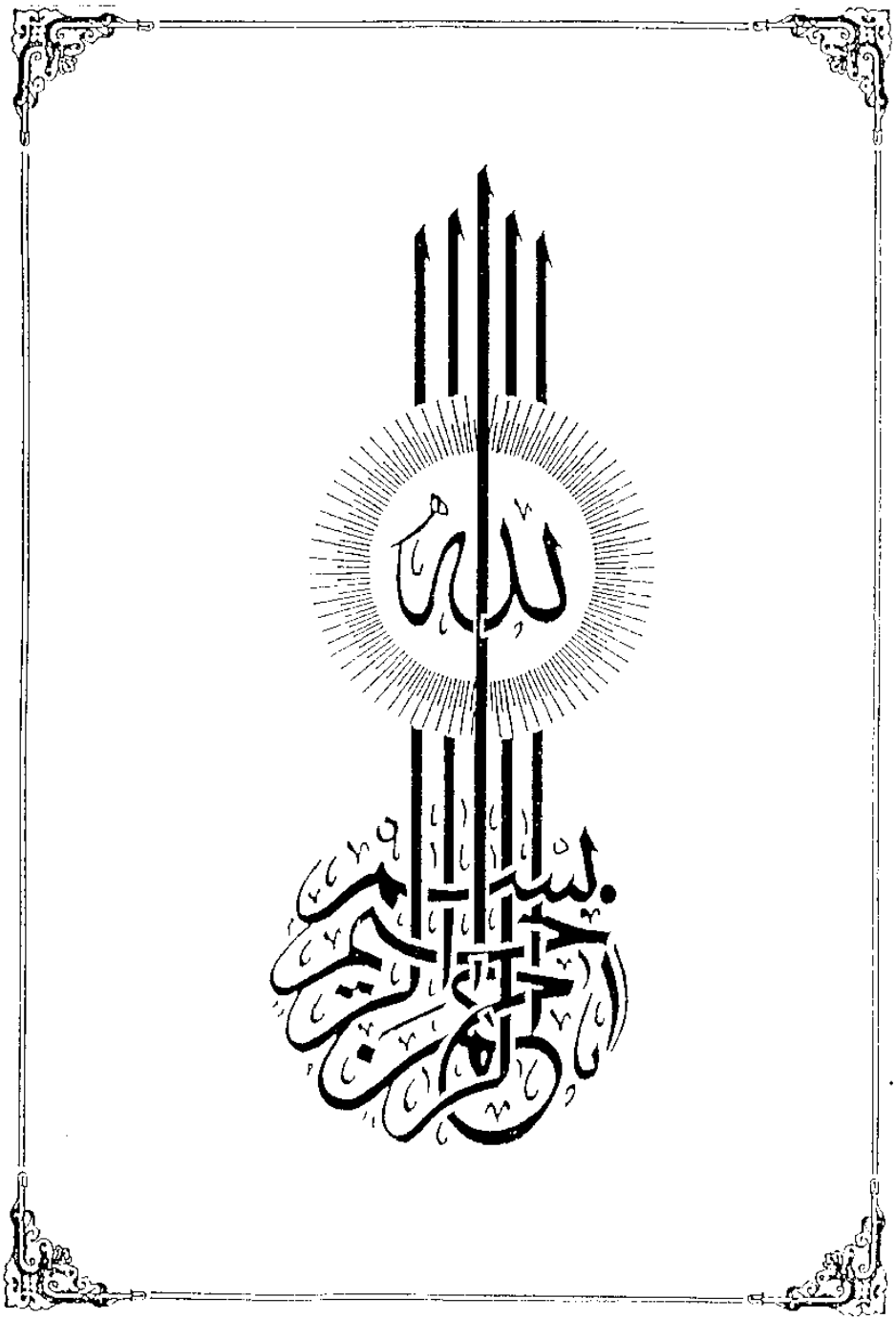

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Contents

INTRODUCTION AND AIM OF THE WORK

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One of the most serious health problems in our country is portal hypertension. Once it develops in a patient (whether following schistosomal infection, attack of viral hepatitis or other causes) it should be dealt with cautiously and all its sequele should be regarded. Hence many techniques have been applied to diagnose and assess the severity of this condition, however most of these techniques had serious drawbacks.

The usefulness of endoscopy in the diagnosis of oesophageal varices is undoubted. More recently, however, ultrasonography has been suggested as a simpler and first line of investigation in detecting all the collaterals due to portal hypertension (Bolondi et al., 1982).

Being safe, rapid and non invasive it is very tempting to try to utilize ultrasonography in various fields of diagnosis.

The aim of this work is to correlate the results of both methods and to study their relative merits in the assessment of portal hypertension. Endoscopic results of oesophageal varices will be compared with the ultrasonographic findings in portal hypertension. This comparison is an attempt to establish certain criteria by ultrasonography, which could prove useful in the diagnosis of portal hypertension in Egypt.

REVIEW OF LITERATURE

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ANATOMY OF THE PORTAL VENOUS SYSTEM

The portal system is a system of veins which drains blood from the abdominal part of the alimentary tract, 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 mesenteric veins (Sherlock, 1981).

Although there are 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 head of the pancreas) at the union of the splenic and the superior mesenteric veins. It then ascends 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 entrance into the liver. The left branch gives branches to the caudate and quadrate lobes and is also connected to a fibrous cord, the ligamentum teres which is a remnant of obliterated left umbilical vein. (It runs 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 the 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 length of the portal vein ranges from 6 to 8 cms., its diameter is about 1.2 cm. and it has no valves (Reynolds, 1969).

The valveless portal vein is an afferent nutrient vessel of the liver and in this sense is an arterial channel (Rappoport, 1975).

Tributaries of the portal vein are:

(1) Splenic.
(2) Superior mesenteric.
(3) Left gastric.
(4) Right gastric.
(5) Para umbilical.
(6) Cystic.

The most troublesome tributatry of the portal vein is the left gastric (coronary vein). It runs upwards along the lesser curvature of the stomach where it receives some oesophageal veins. With progressing cirrhosis of the liver these enlarge to form varices, that may rupture to produce fatal haemorrhage (Rappoport, 1975).

The splenic vein begins by five or six tributaries issuing from the spleen. Such tributaries are then joined by the short gastric

vessels 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 superior mesenteric vein at a right angle to form the portal vein (Warwick and Williams, 1975).

The superior mesenteric vein is very variable, having from ten to twenty five tributaries. It collects blood from the small intestine, the caecum, the ascending and transverse parts of the colon. It usually begins in the right iliac fossa by the union of its numerous tributaries and ascends in the mesentery until the neck of the pancreas to meet the splenic vein (Gardner et al., 1975).

The inferior mesenteric vein 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 mesenteric veins (Romanes, 1969).

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 a case of a 13years 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 mesenteric veins to the portal vein.

Leger (1962) stressed the point that the post natal obliterative process in the umbilical veins and the duct of aranitus may spread to the portal system. A number of small veins form in the process of by-passing the obliterated vascular area. A pre-pancreatic portal vein (lying anterior to the pancreas) associated with biliary atresia was found in a patient with portal hypertension and jaundice since birth. Another case of stenosis of the portal vein with cavernous transformation (which ended in fatal bleeding from oesophageal varices) was also commented on by the same authors (Renner and Child, 1963).

Other rare anomalies of the portal vein include agenesis of the vein, a bifid vein, valves present in the vein and direct drainage into the inferior vena cava (Rousselot and Burchell, 1969).

Collaterals of the Portal Vein:

When the portal circulation is obstructed whether within or outside the liver, a remarkable collateral circulation develops to carry the portal blood into the systemic veins (Sherlock, 1981).

Mcindoe (1928), stated that normally 100% of the portal vein blood flow can be recovered from the hepatic veins, whereas

in cirrhosis only 13% is obtained. The remainder enters collateral channels which form 4 main groups:

Group I:

Where protective epithelium adjoins absorptive epithelium:

(a) At the cardia of the stomach, where the left gastric (coronary) vein and the short gastric veins of the portal system anastmose with the intercostal, diaphragmooesophageal and azygos minor veins of the caval system. Deviation of blood into these vessels leads to varicosities.

(b) At the anus the superior haemorrhoidal vein of the portal anastmoses with the middle and inferior haemorrhoidal veins of the caval system. Deviation of blood into these channels may lead to haemorrhoides.

Group II:

In the falciform ligament through the paraumbilical veins.

Group III: where abdominal organs are in contact with retroperitoneal tissues or adherent to the abdominal wall.

Group IV:

Portal venous blood is carried to the left renal vein. This may be through blood entering directly from the splenic vein or via diaphragmatic, pancreatic, left adrenal or gastric veins. Collaterals running to the pulmonary veins have also been described.

In extrahepatic obstruction additional collaterals are found, for blood is trying to by pass the extrahepatic block. These enter the portal vein in the porta-hepatis beyond the block. These include veins at the "Venae comitantes of the portal vein and hepatic artery" Veins in the suspensory ligaments of the liver, diaphragmatic and omental veins. The larger oesophageal, gastric rectal, splenorenal and lumbar collaterals may also be prominent especially if the block is extensive, when the return of blood through the vena cava is necessary (Sherlock, 1981).

Another rare type of collaterals arises between the portal circulation and the pulmonary veins and such portopulmonary shunts can give rise to cyanosis (Ibrahim, 1968).

PORTAL HYPERTENSION

Normal Portal Pressure:

Sherlock (1981) stated that in normal man the portal pressure is about 7 mm mercury and most authors agree that it should not exceed 10 mm Hg (Davies, 1977). The range varies slightly from one author to another.

Mousa and El Garem in 1959, measured the intrasplenic pressure in more than fourty patients and stated that the upper normal level was 20 cm. saline and that pressures above this were considered as cases of portal hypertension. Asker (1960) recorded a range of 8-10 cm saline as measured at operation. Souidan (1962) reported a range of 17-20 cm saline and this is considered as normal.

Bearing technical errors in measurement, direct portal vein pressure at surgery over 30 cm of saline, intrasplenic pressure over 17 mm of Hg, and wedged hepatic vein pressure more than 4 mm of Hg above inferior vena caval pressure, are reliable indications of portal hypertension (Reynolds, 1975).

Measurement of Portal Pressure:

Measurement of portal pressure was first introduced by Rousselot in 1936 and then followed by Thompson et al. (1937).