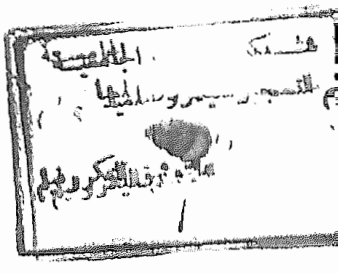
THE INCIDENCE OF PORTAL COLOPATHY IN PATIENTS WITH PORTAL HYPERTENSION

THESIS

SUBMITTED FOR PARTIAL FULFILMENT
FOR MASTER DEGREE IN
MEDICINE



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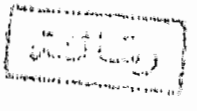

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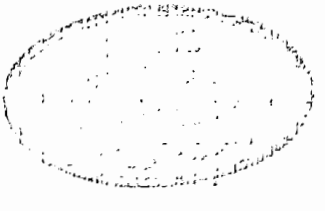

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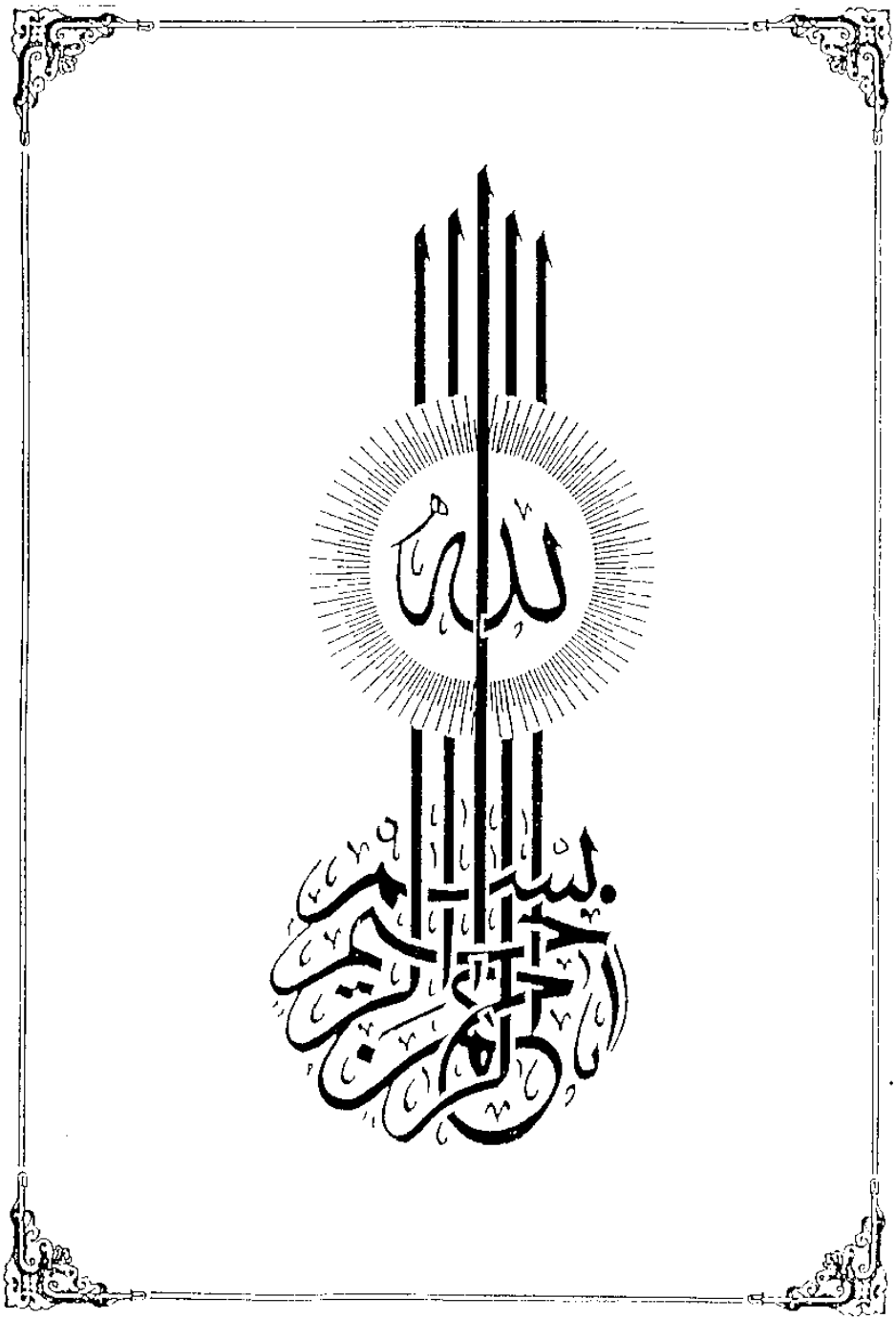

DEDICATION

TO MY BELOVED PARENTS FOR LOVE, CARE AND ENCOURAGEMENT

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INTRODUCTION
AND AIM OF THE WORK

Introduction:

Portal hypertension is a condition characterized by prolonged elevation of portal venous pressure. The clinical features result principally from portal venous congestion and collateral vessel formation. The most important collaterals are those forming gastro-oesophageal varices, which can cause severe upper gastrointestinal bleeding.

Other collaterals include colorectal varices, which can cause lower gastrointestinal bleeding that often mistaken for haemorrhoids.

Congestive gastropathy is a well recognized consequence of portal hypertension and may cause upper gastrointestinal bleeding ***(Mc Cormack et al., 1985)***. It is visualized "endoscopically" as a mosaic pattern of gastric mucosa with or without red spots in antrum, in addition angiodysplasia like lesion, oedema, petechiae and erosions are found ***(Rabinovitz et al., 1990)***. Such lesions have been associated with acute and recurrent gastrointestinal bleeding.

A recent study suggests that similar lesions may be found in the proximal small bowel of patients with portal hypertension. Cherry red spots with dilated mucosal vasculature are described in the duodenum and jejunum of cirrhotic patients with congestive gastropathy *(Thiruvengadam and Gostout, 1989)*.

Other very recent study suggests that lesions resembling the vascular ectasias and telangiectasias noted in portal gastropathy may be found in the colons of cirrhotic patients and it has been described as portal (congestive) colopathy *(Kozareck et al., 1991)*.

Aim of the work:

In the present work, we aimed to study and define the incidence of portal (congestive) colopathy and vascular abnormalities of colon in patients with portal hypertension.

REVIEW OF LITERATURE

I. PORTAL HYPERTENSION

Portal haemodynamics in portal hypertension:

I. Portal venous flow:

Normally, the portal blood flow is about 1000-1200 ml/min. The flow is probably stream lined rather than turbulent. In presence of any block to the portal circulation, the portal pressure rises above the normal range to maintain the blood flow ***(Sedywick and Poulantzas, 1967)***.

When the resistance produced by the block in the system becomes greater than the pressure gradient, the blood flow and velocity inevitably diminish with resulting circulatory stasis. This is decompensated portal hypertension. Stasis is the essential element in the genesis of clinical manifestation. At this stage, circulatory activity can be maintained only by collateral circulation ***(Sedywick and Poulantzas, 1967)***.

As the portal venous pressure is lowered by the development of collaterals, the portal hypertension is maintained by the increased blood flow in the portal system, which becomes hyperdynamic.

The increased flow is achieved by the raised cardiac output and splanchnic vasodilatation. The increased flow refers to total portal flow (hepatic and collaterals), the actual portal flow reaching the liver is of course reduced ***(Sherlock, 1993)***. The hyperdynamic portal circulation is part of the generalized vasodilatation of liver failure. The nature of the splanchnic vasodilator is unknown. Ammonia and endotoxin are unlikely ***(Mehta et al., 1990)***. Glucagon although increases azygos blood flow but has little effect on systemic vascular resistance ***(Lee et al., 1988)***. Ritanserin, 5-hydroxy tryptamine inhibitor reduces portal pressure in cirrhotic patients ***(Vorobioff et al., 1989)***.

II. Hepatic arterial flow:

In man, indirect evidence obtained after death suggests increased hepatic arterial flow in cirrhosis, as the hepatic artery gets larger caliber in cirrhotics than in normal patients ***(Hales et al., 1959)***.

III. Total hepatic blood flow:

Most investigators agree that there is a substantial decrease of blood flow through the liver *(Sherlock, 1993)*.

IV. Circulatory stasis in the portal system:

The velocity of portal blood flow diminishes in portal hypertension as evidenced by prolongation of the portal circulation time *(Castell et al., 1969)*.

V. Development of collateral circulation:

Porto-systemic anastomoses are normally small but enlarge greatly with obstruction to carry the portal blood into the systemic veins. Portal collaterals include two types; "hepatopetal collaterals" which develop in presence of extrahepatic blockage of portal vein and "hepatofugal collaterals" which develop in presence of intrahepatic obstacle shunting blood from the abdominal viscera around the intrahepatic obstacle into the systemic veins *(Rappaport, 1987)*.

1. Hepatofugal collaterals:

Normally, 100% of the portal vein blood flow can be recovered from the hepatic vein, whereas in cirrhosis only 13% is obtained, the remainder enters collateral channels.