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شبكة المعلومات الجامعية

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التوثيق الالكتروني والميكروفيلم

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التوثيق الإلكتروني والميكروفيلم

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بالرسالة صفحات لم ترد بالأصل

COMPARATIVE STUDY BETWEEN SCLEROTHERAPY AND BAND LIGATION FOR ESOPHAGEAL VARICES IN PATIENTS WITH PORTAL HYPERTENSION

THESIS

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By

Amr Aly Abd El-Moety Soliman

MBBCh (Alex), MM (Alex)

FACULTY OF MEDICINE
UNIVERSITY OF ALEXANDRIA

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Supervisors

Prof. Dr. RAFIK ABBAS ZAHER

Professor of Internal Medicine
Faculty of Medicine
Alexandria University

Prof. Dr. MOHAMED YOUSRY TAHER RASHED

Professor of Internal Medicine
Faculty of Medicine
Alexandria University

Prof. Dr. EL-SAID HASSAN IBRAHIM

Professor of Internal Medicine
Faculty of Medicine
Alexandria University

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Introduction

PORTAL VENOUS BLOOD FLOW

Portal blood flow accounts for about three-fourths of the total blood entering the liver. About four-fifths of this flow originates from the intestine and the stomach and the remaining one-fifth originates from the spleen and pancreas. Flow studies suggested that the blood flow in the portal vein is streamlined rather than turbulent and the flow from the splenic vein and the superior mesenteric vein often do not mix in the main stem of the portal vein. Moreover, blood from the spleen drains particularly in the left lobe, whereas blood from the intestine (mesenteric) goes predominantly to the right lobe of the liver.(1)

Meanwhile, at any given moment, the level of the portal blood flow is the result of a whole set of a rapidly changing inter-related blood flows and resistances in a larger number of vascular beds in the hepatic arterial, portal venous, hepatic venous and splanchnic vascular beds.(2)

Several factors exert control over the blood flows and resistances in these vascular beds including intrinsic regulatory, nervous and humoral mechanisms.(3)

1- The Intrinsic Regulatory Mechanisms:

Intrinsic regulatory mechanisms are secondary to changes in the transmural pressure gradient (myogenic mechanism)[4] or in oxygen supply to the hepatocytes and gut (metabolic mechanism).[5] In contrast to hepatic and splanchnic arterial vascular beds, the portal vasculature does not exhibit any of the classical signs of intrinsic regulation of blood flow, there is no pressure-flow autoregulation, reactive hyperemia or functional hyperamia. This reflects the fact that the portal venous inflow is principally determined by the outflow from the intestine and the spleen.[3]

Moreover, one form of intrinsic autoregulation, peculiar to the liver is the "reciprocity of flow" between the hepatic arterial and portal circuits tending to maintain a constant blood flow to the liver despite the pressure gradient. Therefore, reduction of flow in the portal system decreases the hepatic arterial resistance and increases the arterial inflow whereas reduction of the arterial inflow lowers the portal inflow resistance but to a lesser extent.[4] However, this phenomenon is inadequate to compensate fully for an obstruction in one inflow circuit.[3] Furthermore, an elevated hepatic venous resistance i.e outflow resistance, tends to increase hepatic arterial

vascular resistance and reduces portal vascular resistance.[4]

2- Nervous Mechanisms:

The hepatic arterial and splanchnic beds have both alpha and beta adreno receptors, while the portal venous vascular bed contains mainly alpha receptors, since the beta adrenoreceptors are not present in adequate amounts to modulate the portal vasoconstrictor effect by alpha receptors.[6] As might be expected in a vital organ as the liver, the vasoconstrictor stimuli e.g baro-reflexes, exert relatively weak influences at physiological conditions.[7]

Vagal stimulation exerts neurogenic dilator tone in the splanchnic bed as well as in the control of the caliber of the liver sinusoids increasing the number of perfused ones.[3]

3- Humoral Mechanisms:

Many vasodilator substances released from the intestine and the spleen such as dopamine (at low dose), bradykinin, autocoids, prostaglandins, gastrointestinal hormones as well as increased plasma osmolality after absorption from the intestine have influences on the hepatic arterial and splanchnic beds. Glucagon has a unique spectrum of actions; vasodilation plus the antagonsim of vasoconstriction, which ensures a maintained hepatic blood flow in stress states.[7]

Both hepatic arterial and portal venous vascular beds respond by vasoconstriction to nor-epinephrine. Acetyl choline constricts hepatic sinusoids while angiotensin reduces total liver blood flow presumably a combination of hepatic arterial and splanchnic vasoconstriction.[7]

PORTAL VENOUS BLOOD FLOW IN PORTAL HYPERTENSION

According to Ohm's law, the blood pressure (P) in any vascular system is the product of the blood flow (F) to the system and the vascular resistance (R) that impedes this flow and is defined by the equation

$P = F \times R$[8]

Therefore, portal venous pressure is the result of the interplay between portal venous blood flow and vascular resistance offered to that flow.[9] Nevertheless, the amount of blood entering the liver through the portal vein in portal hypertension depends not only on the splanchnic resistance but also on the division of the resulting splanchnic flow between the portal vein and portal systemic collaterals.[10]

In evaluating portal hypertension, two dissimilar haemodynamic theories have been advanced to define portal and splanchnic haemodynamics. While some