شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

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جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

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بالرسالة صفحات لم ترد بالاصل

ELECTIVE FIBEROPTIC ENDOSCOPIC INJECTION SCLEROTHERAPY AS COMPARED WITH BAND LIGATION FOR LARGE ESOPHAGEAL VARICES

Thesis

Submitted to the Faculty of Medicine,
University of Alexandria,
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MASTER OF INTERNAL MEDICINE

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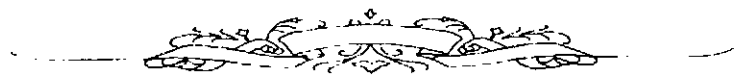
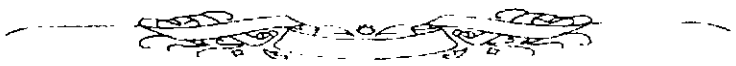


INTRODUCTION



PORTAL HYPERTENSION

Portal hypertension is a condition in which there is persistent increased pressure in the portal venous system above the normal range of 5-10 mmHg.(1) Complications of portal hypertension are observed only when the portal hepatic venous gradient is above 12 mmHg. This value therefore, defines what is known as "clinically significant portal hypertension".(2)

CLASSIFICATION AND ETIOLOGY OF PORTAL HYPERTENSION:

Portal hypertension is, practically, classified into two main groups; (a) pre-sinusoidal (extra- or intra-hepatic) and (b) A big general group of hepatic causes. The pre-sinusoidal form is associated with relatively normal hepato-cellular function. Consequently, if patients with this type suffer a haemorrhage from esophageal varices, liver failure is a rare consequence. In contrast, the hepatic type is associated with hepato-cellular disease; patients with this type following haemorrhage frequently develop liver cell failure.(3)

I- Pre-sinusoidal Causes:(3)

1- Extra-hepatic type.

* Portal vein obstruction.

* Splenic vein obstruction.

* Increased splenic flow: (i) Arterio-venous fistula.

(ii) Idiopathic tropical splenomegaly.

2- Intra-hepatic type.

* Schistosomiasis.

* Early primary biliary cirrhosis.

* Chronic active hepatitis.

* Myelo-proliferative diseases.

* Congenital hepatic fibrosis.

* Sarcoidosis.

* Toxins, vinyl chloride, arsenic, copper.

* Hepato-portal sclerosis.

II- Hepatic causes:[3]

1- Intra-hepatic type.

* Cirrhosis.

* Non-cirrhotic nodules.

* Acute alcoholic hepatitis.

* Cytotoxic drugs.

* Vitamin A intoxication.

2- Post-sinusoidal type.

* Hepatic venous obstruction (Budd-Chiari syndrome).

* Veno-occlusive disease.

* Alcoholic central hyaline sclerosis.

* Inferior vena cava obstruction.

* Rise in right atrial pressure e.g. constrictive pericarditis.

PATHOPHYSIOLOGY OF PORTAL HYPERTENSION:

Normally, the portahepatic circulation is highly compliant and accommodates large variations in blood flow with minor changes in pressure.[4] Portal pressure is determined by the inter-relationship between portal blood flow and the vascular resistance that opposes that flow.[5]

Portal hypertension results from the pathological increase in portal venous resistance (backward flow mechanism) and/or flow (forward flow mechanism); the most common pathology being an increased resistance to flow. As a consequence, portasystemic collaterals develop to decompress the portal system. However, despite these communications, the portal pressure remains elevated by the development of hyperdynamic splanchnic and systemic circulations which maintain enhanced portal venous flow (forward flow mechanism). Thus, it seems that the interplay between increased portal vascular resistance and blood flow is responsible for this condition.[5]

A- Increased portal vascular resistance:

The main factor influencing portal vascular resistance is the radius of the vessel which can be influenced by passive changes (e.g. dilation in response to an increased pressure and blood flow, contraction in response to reduced blood volume) or actively by factors that modify the contractile state of vascular smooth muscle. The length of the vessel is another factor that contributes significantly to the collateral resistance and finally blood viscosity change due to changes in hematocrite and plasma protein concentrations, especially after gastro-intestinal bleeding.[2]

The liver is the main site of resistance to portal blood flow. Pathological conditions which cause an increase in the portal vascular resistance may occur at the pre-sinusoidal, sinusoidal or post-sinusoidal levels.[5] The resulting haemodynamic state is characterised by an elevated portal pressure, an increase in splanchnic resistance and decreased portal blood flow and total hepatic blood flow. One function of splanchnic vasoconstriction in the face of an increased resistance to portal blood flow is to provide the liver with a means to regulate its venous inflow. Also, increases in portal venous resistance would, also, divert more blood through porta-systemic collaterals adding to the decreased portal blood flow.[2]

In liver cirrhosis, it has been proposed that the primary site of increased portal vascular resistance is post-sinusoidal as the hepatic venous radicles are more vulnerable to compression from expanding regenerative nodules than the portal tributaries.[6] Meanwhile, accumulation of collagen within the Disse space in the cirrhotic liver results in a reduction in the sinusoidal diameter and therefore will increase portal vascular resistance[7] with a significant correlation between the amount of collagen deposited and the intrahepatic sinusoidal pressure.[8] Another concept was that the enhanced intra-hepatic sinusoidal pressure may result from hepatocyte enlargement primarily as a consequence of excessive intracellular swelling or fat accumulation.[9] Furthermore, myofibroblasts (i.e activated stellate cells) are localised in fibrous bands, where, under the influence of endothelin or other mediators, their contraction could alter hepatic architecture at the microscopic or macroscopic level, and thus contribute to abnormal blood flow patterns and increased resistance to flow.[10]

Contrary to cirrhosis, hepatic schistosomiasis represents the prototype of pre-sinusoidal veno-occlusive syndrome; leading to diminution of portal vascular bed and the increased resistance to flow of blood. Schistosome eggs embolized and impacted in the peripheral portal venules of the liver excite an immunological reaction of the delayed cell-mediated type (granuloma) which ends with periportal fibrosis.[11,12] Also, the intravascular lesions including endothelial proliferation, subintimal thickness, phelbosclerosis and thrombophlebitis in the intrahepatic branches of the portal vein may contribute to the increased portal venous resistance.[11,13] Meanwhile, a spastic element of the portal vein as a response to various vasoactive agents may add to the elevation of portal pressure in schistosomal hepatic fibrosis (SHF).[14] Nevertheless, there is an evidence for "sinusoidal" hypertension in SHF which has been attributed to the hypertrophy and dilatation of intrahepatic arterial tree with increased arterial blood flowing into a reduced sinusoidal bed due to prominent collapse of hepatic parenchyma and septal fibrosis,[15] and collagen deposition in the perisinusoidal (Dissë) space.[16,17]

B- Splanchnic vasodilation and hyperdynamic circulation:

Increased portal venous inflow is the result of a marked arteriolar vasodilation in splanchnic organs draining into the portal veins, and contributing to maintenance and aggravation of the portal hypertensive syndrome.[18] This is evidenced by an elevated splanchnic blood inflow; a reduced splanchnic arteriolar resistance;[19] a shortened mean transit time of labelled albumin injected into the splanchnic circulation;.[20] a decrease in arteriovenous oxygen difference between the splanchnic and systemic circulations;[21] and increased splenic blood flow.[22]