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Dedication

To My ….Parents

To My Brothers and Sisters :

Samah ,Mostafa , Mansour and Esraa ..

Mohamed Abobakr

إلَــى والِــدَايْ

. . .

لَـعـلّـي لَـمْ أُخـيِـبْ
أمـلَـكُم ا . . . .

مُـحَمّدْ . .

بسم الله الرحمن الرحيم

التغيرات النسيجية في الغشاء المخاطي المبطن للجهاز الهضمي في حالات ارتفاع ضغط الوريد البابي

بحث مقدم من

الطبيب / محمد أبوبكر عثمان

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LIST OFABBERVIATIONS

AVM:arterio-venous malformation

CSPH : clinical significant portal hypertension .

CT : computed tomography

ET-1 :endothelin – 1

GAVE : gastric antral vascular ectasia .

GI : gastrointestinal

GIT : gastrointestinal tract .

GEJ :gastroesophegeal junction

GMBF : gastric mucosal blood flow .

GOV : gastro-esophegealvarices

GVE :gasreic vascular ectasia

HbsAg :hepatits B surface antigen .

HbcAb :hepatits B core antibody

HCC : hepatocellular carcinoma

HCVAb :hepatits C virus antibody .

HVPG : hepatic venous portal gradient .

IGV : isolated gastric varices

IVC : inferior vena cava

LGV : left gastric vien .

MRI : magnetic resonance imaging

MLP : mosaic like pattern .

NIEC : north Italian endoscopic club

NO : nitric oxide

NOS : nitric oxide Synthase

PC :prothrombine concentration .

PHG: portal hypertensive gastropathy .

PH : portal hypertension

PF : portal fibrosis .

PV : portal vien .

RBC : red blood corpuscles .

SD : standard deviation .

SMV: superior mesenteric vien .

SV : splenic vien .

SBE :subacute bacterial endocarditis .

TIPSS :transjugular intrahepatic porto-systemic shunt

TNF : tumor necrosis factor

WHO :world health organization

WHVP :wedge hepatic venous pressure

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Morphological Changes ofGastrointestinal Mucosa in Cases of Portal Hypertension

An Essay
Submitted for partial Fulfillment of The
Requirement of Master Degree in
General Surgery

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INTRODUCTION AND AIM OF THE WORK

An increasing body of evidence made many authors since the 70's to suggest that portal hypertension induces definite vascular and microvascular changes in the gastric mucosa. These changes are likely basis of the unique functional and morphologic fetures of portal hypertensive gastric mucosa that may predispose it to severe damage or haemorrhage **(Tarnuwaski et al., 1987),**

Although several problems exist regarding the diagnostic criteria and clinical significance of this condition (confusion in terminology, lack of uniformity in the endoscopic description, absence of distinctive histological features and lack of correlation between the endoscopic and hislologic appearance of the mucosa in patients with portal hypertension), portal hypertensive gastropathy is now recognised as a distinct entity **(Viggiano and Gostout, 1992).**

Portal hypertensive gastropathy (PHG) is a term used to describe the endoscopic appearance of gastric mucosa with a characteristic mosaic-like pattern with or without red spots **(Thulvath and Yoo, 2002).**

The development of portal hypertensive gastropathy has important clinical implications; it is the second most common

cause of upper gastrointestinal bleeding (after esophageal varices) in patients with portal hypertension. Once the Initial episode of bleeding from portal hypertensive gastropathy has occurred, therisk of recurrent bleeding during the next 2 years is as high 75%. Although some patients may have acute, even life-Ithreatening haematemesis, others may have chronic or intermittent low-grade bleeding that result in severe iron-deficiency anemia **(Chang et al., 2000).**

This study aimed to asses morphological changes of gastrointestinal mucosa in cases of portal hypertension.

PORTAL HYPERTENSION

Historical perspective:

Gilbert and Villaret first coined the term portal hypertension in 1906, recognizing the association of ascites and cirrhosis. Banti, in 1894 had postulated that splenomegaly was the antecedent event leading to gastrointestinal bleeding, anemia, hepatomegaly, cirrhosis and ascites, but Mclndoe, in 1928 and McMichael, in 1931 showed that splenomegaly was the result of portal hypertension. Hemodynamic studies in the 1930s and 1940s, **(Thompson et al., 1937, Biadley et al., 1945),** continued through to the present time, led to a better understanding of the pathophysiology of portal hypertension, to definition of the **CSPH**, and to guidelines for necessary pressure reduction to prevent bleeding **(Li and Henderson, 2001).**

Surgical management has evolved over the last century. The first portocavai shunt was performed by Eck in 1877 and in 1893; Pavlov demonstrated that total portal diversion leads to liver failure and encephalopathy. Banti, in 1894 popularized splenectomy, while Morison and Talma tried omentopexy. Vidal in 1903 performed the first portocavai shunt in man, and porto-systemic shunt was reintroduced by Whipple in 1945. While bleeding control was good, liver failure was accelerated.

In the 1960s selective shunts were introduced by Warren, 1967 **(Li and Henderson, 2001).**

Non-shunt surgical procedures were introduced by **Tanner, (1950).** In Egypt, **Hassab, (1967)** reported an extensiveexperience with gastric devascularization in the treatment of Schistosomiasis patients with portal hypertension. In the 1980's the ultimate surgical management of portal hypertension became a clinical reality with liver transplantation **(Starz et al., 1989).**

Less invasive method of treating portal hypertension was sclerotherapy, first introduced by Crafoord and Frenckner in 1939, refined in the 1970s with flexible endoscopy**(Johnston et al., 1973 and Paqueretal, 1978),** and modified in the 1990s by band ligation of varices **(Steigman et al., 1992).** Pharmacotherapies were introduced in the 1980s to reduce portal hypertension **(Lebrec et al, 1980).** Finally, in the last decade radioiogically placed intrahepatic portocaval shunts are being used to decompress portal hypertension **(Rossle et al., 1994) & (Li and Henderson, 2001).**

ANATOMY OF THE PORTAL SYSTEM

The portal system includes all the veins which drain the blood from the abdominal part of the digestive tract (except lower part of the anal canal) and from the spleen, pancreas, and gallbladder. From these viscera the blood is conveyed to the liver by the portal vein. In the liver this vein ramifies like an artery and ends in capillary-like vessels termed sinusoids **(Williams et al., 1989).**

EXTRA-HEPATIC PORTAL VENOUS SYSTEM:

The veins which constitute the portal system are the superior mesenteric, inferior mesenteric, splenic and portal, the main channel entering the liver, arid their tributaries **(Anson, 1979).**

PORTAL VEIN

The portal vein is formed behind the neck of the pancreas by the confluence of its roots namely , the splenic vien and the superior mesenteric veins. It is about 7-10 cm long and 0.8 - 1.4 cm in diameter and is without valves in adults **(Skandalakis et al, 2004).**

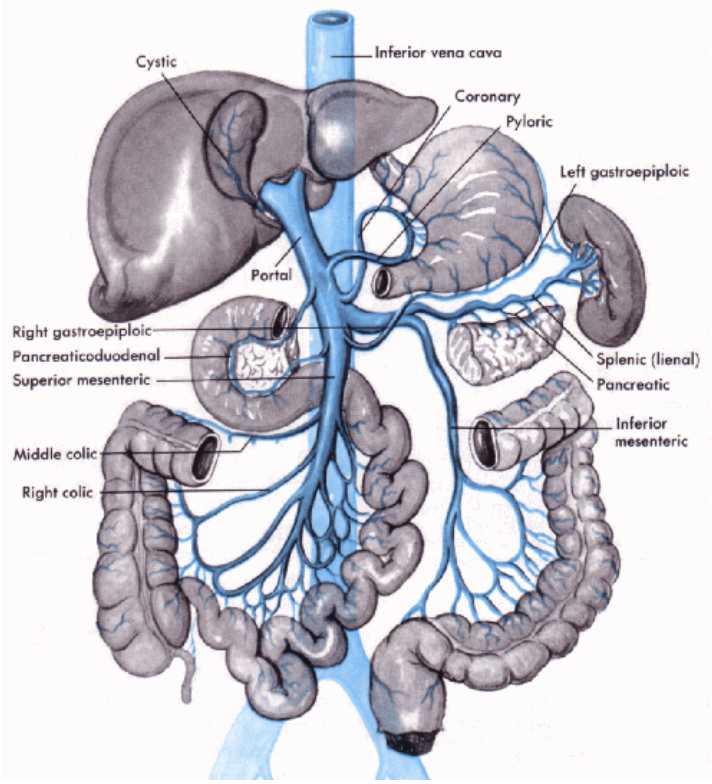

Figure 1 : Anatomy of portal vien .

The portal trunk passes upward, behind the first part of the duodenum, crosses in front of inferior vena cava and enters the hepatoduodenal ligament, just anterior to foramen of Winslow, in a plane dorsal to the common bile duct and hepatic artery; the former lies to the right of the latter. At the porta hepatis, the portal vein divides into two lobar branches before entering the liver. The right branch of the portal vein enters the right lobe of the liver, but before doing so generally receives the cystic vein. The left branch, longer and smaller in caliber, is joined by the ligamentum teres (obliterated remnants of left umbilical vein)