Study the effect of helicobacter pylori infection on portal hypertensive gastropathy.

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بسم الله الرحمن الرحيم

"سنريهم عاياتنا في الآفاق وفي
أنفسهم حتي يتبين لهم انه الحق أو
لم يكفء بربك أنه علي كل شئ
شهيد "

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Abbreviations

C.L.O.	**:**	**Campylobacter like organism**
G.R.P.	**:**	**Gastrin-releasing peptide**
H.V.P.G.	**:**	**Hepatic venous pressure gradient**
I.C.U.	**:**	**Idiopathic chronic urticaria**
I.T.P.	**:**	**Idiopathic Thrombocytopenic Purpura**
M.A.L.T.	**:**	**Mucosa associated lymphoid tissue**
M.D.	**:**	**Menetrier's disease**
M.H.C.	**:**	**Major Histocompatibility Complex**
M.R.A.	**:**	**Magnetic resonance angiography**
N.O.	**:**	**Nitric oxide**
N.U.D.	**:**	**Non ulcer dyspepsia**
P.C.R.	**:**	**polymerase chain reaction**
P.H.G.	**:**	**Portal hypertensive gastropathy**
P.U.D.	**:**	**Peptic ulcer disease**
R.B.C.	**:**	**Ranitidine bismuth citrate**
S.P.E.C.T.	**:**	**Single photon emission CT**
v.W.F.	**:**	**von Willebrand factor**
W.H.V.P	**:**	**wedged hepatic venous pressure**

Study the effect of helicobacter pylori infection on portal hypertensive gastropathy.

Helicobacter pylori infection is probably the most common chronic infection, and the human populations throughout the world are affected. Its incidence increases with age, and occurs earlier and with increased rates in developing countries and in lower socioeconomic groups. ***(de Boer et al.,1995- Graham et al.,1992-Soll et al.,1996)***

Helicobacter pylori infection causes chronic inflammation of the inner lining of the stomach (gastritis) and is the most common cause of ulcers worldwide .(***Unge et al.,1997- van der Hulst et al.,1996).***One every six patient with helicobacter pylori infection will develop ulcers of the duodenum or stomach Helicobacter pylori also is associated with stomach cancer.

Portal hypertension is a common disease that affect the stomach .It is mostly caused by liver cirrhosis .Portal hypertension may be defined as a portal pressure gradient of 12 mm Hg or greater and is often associated with varices , gastropathy and ascites.

Gastric mucosal lesions are common in individuals with portal hypertension and can be an important cause of blood loss even in the absence of oesophogeal or gastric varices which is generally slow and insidious but can be massive and occasionally fatal***.(Iwao T et al .,1992)***

It has been estimated that non variceal bleeding from portal hypertension gastropathy is responsible for up to 30%of all cases of gastrointestinal bleeding in patients with portal hypertension***.(Balan KK et al.,1999)***

Since its original description by Smart and Riger in 1991 (***Smart HL et al.,1991)*** portal hypertensive gastropathy has been recognized as a distinct pathologic entity characterized by mucosal hyperemia and dilated

submucosal vessels in the stomach caused by chronic portal hypertension. (***Zhy HH et al.,1996)***

So it is clearly necessary to know how the two mentioned important causes of gastric lesions act together and to know to what extent helicobacter pylori infection affect portal hypertensive gastropathy.

Many studies were done to estimate this relationship ,some of them mentioned that helicobacter pylori infection is not essential factor that worsen portal hypertensive gastropathy state***.(Bahnacy A et al.,1997- Balan KK et al.,1997),***while others mentioned that helicobacter pylori infection is a major risk factor in worsening (up to peptic ulcer disease) of portal hypertensive gastropathy***.(Dore MP et al.,2004- McCormack TT, et al.,1985- P. Kamalaporn J et al.,200 5).***

Aim of the Work

The aim of this study is to study the effect of helicobacter pylori infection on portal hypertensive gastropathy.

Patients and Methods:

Choice of the Patients:

This study will entail thirty patients, chosen from Ain Shams University Hospitals, with portal hypertension, fifteen of them with helicobacter pylori infection proved by gastric biobsy and/or clue test, the other fifteen patients are not infected with helicobacter pylori.

All Patients will be subjected to the following studies:

1.Clinical assessment :

Full history taking and complete clinical examination.

2.Laboratory investigations will include:

-C.B.C.

- ALT, AST.

-S. Albumin.

-S. Bilirubin.

-P.T.

3.Ultrasonographic examination of the abdomen:

To prove portal hypertension and measure portal vein diameter.

4.Upper G.I.T. endoscopy :

To assess the state of the stomach.

5.Gastric biopsies from the antrum and corporus will be taken to exclude presence or absence of helicobacter pylori and to assess the degree of portal gastropathy.

Chapter 1

Anatomy of the portal venous system

The portal system includes all the veins draining the abdominal part of the digestive tube (except the lower anal canal), spleen, pancreas, and gall bladder from these viscera blood is conveyed by the portal vein to the liver, where it ramifies like an artery, ending in the sinusoids, from which blood again converges to reach the inferior vena cava via the hepatic veins. The blood therefore passes through two sets of exchange vessels; capillaries of the drained viscera and hepatic sinusoids ***(Williams et al.,1989).***

Anatomically, the portal vein measures approximately 6 to 8cm long and 12mm in diameter ***(Schiff, 1993)***. The portal vein is formed by the union of the superior mesenteric vein and the splenic vein just posterior to the head of the pancreas at about the level of the second lumbar vertebra. It extends slightly to the right of the midline for a distance of 5.5-8cm to the portal hepatic. The portal vein has a segmental intra-hepatic distribution, accompanying the hepatic artery ***(Sherlock and Dooley, 2002)***.

T*ributaries of the portal vein:*

Splenic, superior mesenteric, left gastric, right gastric, paraumbilical and cystic ***(Williams et al.,1989).***

The splenic veins: (5-15 channels) originate at the splenic hilum and join near the tail of pancreas with the short gastric vessels to form the main splenic vein. It receives numerous tributaries from the head of the pancreas, the left gastro-epiploic vein enters it near the spleen. The inferior mesenteric vein bringing blood from the left part of the colon and recturn ***(Sherlock and Dooley, 2002)***.

The superior mesenteric vein: is formed by tributaries from the small intestine, colon and the head of the pancreas, and irregularly from the stomach via the right gastroepiploic vein ***(Sherlock and Dooley, 2002)***. It ends behind the head of the pancreas at the level of the second lumbar vertebra, by joining the splenic vein at a right angle to form the portal vein ***(Williams et al.,1989).***

The left gastric vein: it receives tributaries from both gastric surfaces and the esophageal vein. It ends in the portal vein at upper border of the superior part of the duodenum. ***(last, 1984).***

The paraumbilical vein: They connect veins of the anterior abdominal wail with the portal vein. The best developed one, begins at the umbilicus and runs in or o ligamentum teres in the falciform fold to end in the left branch of the portal vein ***(Williams et al.,1989).***

The right gastric vein: It receives the prepylotic vein ***(Romanes, 1981).***

The cystic vein: Drains the gall bladder ***(Williams et al.,1989).***

Physiology of the portal system:

The viscera and the liver receive 30% of the cardiac output, about 1500ml via the celiac, superior and inferior mesenteric arteries. The major function of the splanchnic circulation is to support the broad ranges of activities associated with the function of the digestive system ***(Guyton,1996).*** The reduction of any of the general haemodynamic factors including cardiac output, arterial pressure, gastrointestinal blood flow ***(Park and Jacobson, 1987).***

Portal vein flow is about 900-1200ml/min., hepatic arterial flow about 500-700ml/min. the portal venous pressure is normally about 5-10mmHg, only a little higher than that of the inferior vena cava. Little is known

about the factors controlling splanchnic and thus portal blood flow; the increase after meals is proportional to the increase in splanchnic arterial flow ***(Weatherall et al., 1996).***

Portal oxygen content: The fating arterio-portal oxygen difference is only 1.9 volumes percent (range 0.4-3.3 volumes percent) and the portal vein contributes 40ml/min or 72% of the total oxygen supply to the liver.

During digestion, the arterio-venous oxygen difference increases due to increased intestinal utilization.

Hepatic blood flow and velocity:

The hepatic blood flow in humans at resting condition represents 25-30% of cardiac output. About 75% of this are supplied by the portal vein; the hepatic artery supplies the remaining 25%. Portal blood flow through the hepatic sinusoids in close contact with the hepatocytes and then enters the hepatic vein to drain into the inferior vena cava. The arterial blood, often supplying the structural elements of the liver, empties into hepatic sinusoids, where it is mixed with the portal blood ***(Jenkins and Billing, 1985).***

Chapter 2

Portal hypertension

Definition:

Portal hypertension may be defined as an increase in resting portal venous pressure above 12mmHg, but it may rise as high as 50mmHg. It results from increased resistance to blood flow and/ or an increase in portal flow (Weatherall et al., 1996).

Pathogenesis of portal hypertension:

Portal pressure is determined by portal venous blood flow and resistance of portal venous inflow to the liver ***(Granger et al., 1980)***. The relationship is expressed mathematically as: P= Q × R, where P is the change in pressure along a vessel, Q is the flow and R is the resistance to that flow. Thus, increase in flow or resistance are translated into increase in pressure and changes in both have a multiplicative effect ***(Mahl and Groszmann, 1990).***

When the normal compensatory haemostatic mechanisms are inadequate, portal hypertension results as a consequence of pathological increase in either portal venous inflow (forward hypothesis) or resistance (backword hypothesis) ***(Witte, 1983).***

Etiology of portal hypertension

I- Extra hepatic obstruction of portal vein and its tributaries:

a) Portal vein thrombosis:

Infections: umbilical infection with or without catheterization of the umbilical vein may be responsible in neonates. Acute appendicitis and peritonitis are causative in older children. Neonatal dehydration and infection may be responsible ***(Sherlock and Dooley, 2002).*** Ulcerative colitis can be complicated by portal vein block.

Postoperative: The portal and splenic vein commonly block after splenectomy, especially when preoperatively, the patient had a normal platelet count. It is especially likely in patients with myeloid metaplasia ***(Sherlock and Dooley, 2002).***

Hypercoagulable state:

This is a frequent cause of portal vein thrombosis in adults. It is commonly due to myeloproliferative disorder which may be latent ***(Valla et al., 1988).***

Trauma:

Portal vein injury rarely follow a vehicle accidents or stabbing. Laceration of the portal vein is 50% fatal and ligation may be the only method to control bleeding ***(Sherlock and Dooley, 2002).***

Invasion and compression:

The classic example is hepatocellular carcinoma. Carcinoma of the pancreas, usually of the body, and of other adjacent organs may lead to portal vein block. Chronic pancreatitis is frequently associated with splenic vein obstruction ***(Sherlock and Dooley, 2002).***

Congenital:

Congenital obstruction can be produced anywhere along the right and left vitelline veins from which the portal vein develops. The portal vein may be absent with visceral venous return passing to systemic veins, particularly the inferior vena cava ***(Morse et. al., 1986).***

Cirrhosis:

The prevalence of portal vein thrombosis complicating cirrhosis is very low. Invasion by hepatocellular carcinoma is the most frequent cause. Post-splenectomy thrombocytosis is another aetiological factor ***(Okuda et al., 1985).***