

# PORTAL HYPERTENSIVE GASTROPATHY IN PATIENTS WITH LIVER CIRRHOSIS IN THI-QAR HOSPITAL

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## ABSTRACT:

### Background:

Portal hypertensive gastropathy (PHG) refers to changes in the mucosa of the stomach in patients with liver cirrhosis, it may present with a wide variety of manifestations ranging from asymptomatic to severe upper GIT bleeding.

### Patient and method:

50 patients with histological proven liver cirrhosis involved in this study .35patients (70%) were male while 15 patients(30%) were female. OGD was done for them which revealed 46% of them were affected with PHG irrespective to the cause of liver cirrhosis. all kinds of liver cirrhosis was taken in the study apart from those who took NSAIDs in the last two weeks to exclude other causes of erosive gastritis.

### Results:-

PHG was found in 46% of the patients , it range from gastric fold edema in 12% to ,erosive hemorrhagic gastritis in 16% erythematous mucosa in 24%, petechiae in 30% to snake skin appearance in 36%.

### Conclusion:

PHG is well known complication of liver cirrhosis that can be asymptomatic or cause other manifestations from dyspepsia to severe upper GIT bleeding that must be treated.

## INTRODUCTION

Portal Hypertensive gastropathy:

Refers to changes in the mucosa of the stomach in patients with portal hypertension. Liver cirrhosis is the most common cause (1). These mucosal changes include friability of the mucosa and the presence of ecstasic blood vessels at the surface (2). Patients with (PHG) may uncommonly manifest itself as haematemesis or melena, more commonly as upper gastrointestinal bleeding, such as esophageal varices and gastric varices(3). On endoscopic evaluation of the stomach some time the condition shows a characteristic mosaic or "snake-skin" appearance of the stomach(3)and(4)

### Pathogenesis:

Several studies have found that patients with portal hypertension develop increased blood flow to the stomach (5). The physiological findings that correlate with worsening portal hypertensive gastropathy include an increased portal venous pressure gradient and decreased hepatic blood flow(6). Biopsies of the stomach in patients with portal hypertensive gastropathy show ecstasic (or dilated) blood vessels, evidence of bleeding by means of red blood cells in the lamina propria, and edema in the stomach wall(7) . nitric oxide also play a role in blood vessel dilatation .

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## Clinical Findings:

Most patients with portal hypertensive gastropathy have either a stable or improving course in the appearance of the gastropathy on endoscope (3). However, according to retrospective data, roughly one in seven patients with portal hypertensive gastropathy will develop bleeding (either acute or chronic) attributable to the gastropathy(8). Patients with chronic bleeding will usually come to the attention of the medical system because of anemia(7) The usual appearance of portal hypertensive gastropathy on endoscope is a mosaic-like or reticular pattern in the mucosa. Red spots may or be present(9). The pattern is usually seen throughout the stomach. A similar pattern can be seen with a related condition called gastric antral vascular ectasia (GAVE)(2), or watermelon stomach. However, in GAVE, the ecstatic blood vessels are more commonly found in the antrum or lower part.

## Treatment:

### Medications:

Beta-blockers, which reduce portal pressures. Non-selective beta blockers (such as propranolol and nadolol have been used to decrease the pressure of the portal vein in patients with esophageal varices(10)and, and have been shown to regress (PHG). Anti-fibrinolytic medications such as tranexamic acid have also been used in case reports of patients with(PHG)(11),(12)and(13). These medications work by stabilizing deposits of fibrin at sites that ordinarily would bleed....

Finally, octreotide(14), an analogue of somatostatin that leads to vasoconstriction of the portal circulation, can be used for active bleeding due to (PHG). Sucralfate(15), a coating medication has also been used, but evidence.

### Procedural:

Endoscopic therapy by a fiber-optic camera into the stomach. Argon plasma coagulation(16) and electrocautery have both been used to stop bleeding from ecstatic vessels, and an attempt to

obliterate the vessels have limited utility .

Transjugular intrahepatic portosystemic shunt procedures or TIPS(17) involve decompressing the portal vein by shunting a portal venule to a lower pressure systemic venule, under guidance with fluoroscopy. The literature reports suggest both regression of(PHG) on endoscopic images .

Finally, cryotherapy(18) involves the use of pressurized carbon dioxide administered through the endoscope to freeze and destroy tissue in a focal area.

Cirrhosis of the liver:

Is a chronic disease that causes cell destruction and fibrosis (scarring) of hepatic tissue. Fibrosis alters normal liver structure and vasculature, impairing blood and lymph flow and resulting in hepatic insufficiency and hypertension in the portal vein. Complications include hyponatremia, water retention, bleeding esophageal varices coagulopathy(19),spontaneous bacterial peritonitis,and hepatic encephalopathy.

Cirrhosis is a potentially life-threatening condition that occurs when scarring damages the liver. When chronic diseases cause the liver to become permanently injured and scar. The loss of normal liver tissue slows that processing of nutrients, hormones, drugs, and toxins by the liver. Also, the production of proteins and other substances made by the liver is suppressed. People with cirrhosis often have few symptoms at first. The person may experience fatigue, weakness, and exhaustion. Loss of appetite is usual, often with nausea and weight loss. As liver function declines, water may accumulate in the legs and the abdomen (ascites). A decrease in proteins needed for blood clotting makes it easy for the person to bruise, bleeding or infection. In the later stages of cirrhosis, jaundice (yellow skin) may occur, caused by the buildup of bile pigment that is passed by the liver into the intestine. The liver of a person with cirrhosis also has trouble removing toxins, which may build. The doctor often can diagnose cirrhosis from the patient's symptoms and from laboratory tests.one of

these tests is liver biopsy in which tissue scarring is seen, the severity of this lesion and its associated outcome, chronic gastrointestinal bleeding, has been correlated with the degree of portal hypertension. While increased gastric blood has been documented in patients with portal hypertensive gastropathy(14). Further supporting this association with portal hypertension is the presence of esophageal varices(15,16).

Histological, we see on low power examination villiform architecture with expansion of lamina propria by fibro muscular stroma and subepithelial capillary proliferation c/w reactive gastropathy(20). On high power exam, in addition there are several ectatic capillaries without hyalinization of the wall or micro-thrombi within the lumen. These findings are c/w (PHG). If within the lumen of these dilated capillaries, the pathologist can identify hyalinization or thrombus formation then the diagnosis of GAVE or gastric antral vascular ectasia must be considered (21).

## **MATERIAL & METHODS:**

Is a cross sectional descriptive epidemiological study extended from the 1<sup>st</sup> week of June 2004 till the end of June 2005, performed in alhussain teaching hospital .

In which 50 patients were included, all of them was diagnosed to have liver cirrhosis histologically proven and they were in continuous therapy including H2 blockers, steroids , Vitamin K, isosorbide dinitrate.

All of them subjected to upper endoscopy which is part of the study

Investigations were performed to each of them to prove the diagnosis and to know the degree of hepatic dysfunction.

All types of liver cirrhosis were included in the study. The examination was done at every day of the week when a case of liver cirrhosis to be available.

Olympus type endoscopy PQ20 and pantax endoscopy was used with the facility of

xylocaine spray ,using modified dose of medazolam and fentanyl to facilitate the procedure of endoscopy.

The results of the endoscopy were classified according to the type of liver cirrhosis and degree of gastric involvement as portal hypertensive gastropath.

PHG lesions range from mild (petechiae ) ,focal erythematous mucosa, diffuse mucosal erosion(erosive hemorrhagic gastritis), through mosaic pattern(snake skin appearance).

We exclude all patients that received NSAID in the previous 4 weeks and those patients who took B blocker in the past 2 weeks to exclude the other causes of erosions and all patients that received any kind of drugs that can alter the vascular picture of the mucosa.

15 patients(30%) had a history of esophageal veins sclerotherapy for upper GIT bleeding.

Epidemiological analysis was used to assess the positive findings of the results by using tabular presentations of data, their percentages, prevalence of the portal hypertension gastropathy through manual calculation.

## **RESULTS:**

From the studied population 35 (70%) were male and 15(30%) were female regardless the cause of liver cirrhosis.

There age was range from 10 years up to 60 years age mean 43 + - 6 stander deviation.

PHG was found in 46% from the total number of liver cirrhotic populations undergo endoscopic examination.

Some of our patients have fundal varices which are found in 10 % although some times not accompanied by esophageal varices.

## **DISCUSSION**

PHG prevalence in our study is 46 % which is different from other studies like Robert J Fontana from Michigan University because we use a wide exclusion range from sclerotherapy to many drug intake like NSAIDS.

Classification is highly different from one study to the other although old classification had used which did not give you any idea about the current state of the patient it was range from (mosaic like pattern, red point lesion, cherry red spots to black –brown spots) this was mentioned in the gastroenterology club in Dec 1997.

Snake skin appearance which is now called mosaic pattern is the most common findings in many studies as K W Burak, S SLEE and Kumar et al.

PHG can cause acute and severe or chronic bleeding from the petechial spots or gastritis which is also shown in other studies like Nelson Garcia et al. In our study erosive gastritis was 16 % while in Nelson it was 15 -20 % which is equal in both studies.

Liver cirrhosis is more in male due to the more prevalence of the causes of liver cirrhosis in male than female like haemochromatosis, viral hepatitis and alcohol intake.

### **Limitations of the study:**

- 1- Refusal rate for endoscopic examination was very high.
- 2- Political situation instability in the governorate at the time of the study.
- 3- Exclusion criteria that limit the number of studied population.

## **CONCLUSION**

From the previous results we can conclude that :

- 1) liver cirrhosis in male more than female.
- 2) viral cause for liver cirrhosis is more common in Iraq than the other countries.
- 3) PHG is a well known complication of liver cirrhosis (45%)??? which is usually ignored in most of the endoscopy sessions.
- 4) mosaic pattern (snake skin appearance ) is the most common finding during endoscopy although more benign finding than it is the edema of the folds or erythematous mucosa.
- 5) erosive gastritis present in 16 % which is more than in the general population.
- 6) many patients have more than one finding in the same session.

## **RECOMMENDATIONS:**

- 1) Application of hepatitis B surface antigen, hepatitis viral antibody screening test for the whole risk groups.
- 2) Special attention for those with specific predisposing disease who are considered as a main cause of liver cirrhosis.
- 3) Public education.

**TABLES:**

Table 1-  
Demographic charters of studied population:

Age	Male	female	Total
10-35	5 (10)	2 (4)	7 (14)
36-49	27 (54)	12 (24)	39 (78)
50 & above	3 (6)	1 (2)	4 (8)
total	35 (70)	15 (30)	50 (100)

Table 2-  
Liver cirrhosis according to the causes

Causes	No. of the patients	Percentage
<b>Cryptogenic</b>	16	32%
<b>Viral hepatitis B</b>	14	28%
<b>Viral hepatitis C</b>	12	24%
<b>Alcoholics</b>	2	4%
<b>Autoimmune hepatitis</b>	1	2%
<b>Wilson''s disease</b>	1	2%
<b>Primary biliary cirrhosis</b>	2	4%
<b>Haemocromatosis</b>	2	4%
<b>Total</b>	50	100

Table 3:  
Gastric picture in liver cirrhotic patients according to the endoscopic findings:

Endoscopic findings	No. of the patients	No.(%)
Gastric folds odema	4	8%
Snake skin appearance	17	34%
Petechiae	13	26%
Erythematous mucosa	11	22%
Erosive heamorrhagic gastritis	5	10%
total	50	100%

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## اعتلال المعدة الناشئ من ارتفاع الضغط البوابي للمرضى المصابين بتشمع الكبد

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### الخلاصة:

اعتلال المعدة الناشئ من ارتفاع الضغط البوابي يشير إلى التغيرات الحاصلة في بطانة المعدة للمرضى المصابين بتشمع الكبد والتي يمكن أن تظهر بأعراض مختلفة تتباين من كونها غير محسوسة وطفيفة إلى نزف حاد أو مزمن من المعدة وبالتالي من الجهاز الهضمي العلوي.

المرضى والطرق المستخدمة:

أجريت الدراسة على خمسون مريض تم تشخيصهم سريريا ومختبريا ونسجيا لإصابتهم بتشمع الكبد. ٣٥ (٧٠%) منهم ذكور و١٥ (٣٠%) كانوا من الإناث. اجري لهم فحص المعدة بالناظور وتبين إصابة ٤٦% منهم باعتلال المعدة بغض النظر عن أسباب التشمع الكبدي، تم فحص جميع المرضى ما عدا أولئك الذين تناولوا العقاقير الغير استرويدية في الأسبوعين السابقين للفحص لاحتمال إصابتهم بالتهاب بطانة المعدة.

### النتائج:

وجد إصابة ٤٦% من المرضى باعتلال المعدة. تتراوح هذه الإصابة ما بين وذمة بطانة المعدة في ١٢% من المرضى، التهاب بطانة المعدة النازفة ١٦%، احمرار بطانة المعدة ٢٤%، بقع نزفية طفيفة ٣٠% أو مظهر جلد الأفعى ٣٦% ويمكن أن نجد خليط من أكثر من مظهر في نفس المريض.

### النتائج:

اعتلال بطانة المعدة هي من المضاعفات المعروفة والمميزة بعد الإصابة بمرضى تشمع الكبد، يمكن أن تظهر علاماتها على المرضى ما بين الغير مرئية، سوء الهضم، ألم في المعدة أو نزف من الجهاز الهضمي العلوي مع حالة الصدمة.

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