DOI: https://doi.org/10.53350/pjmhs22165446

ORIGINAL ARTICLE

Frequency of Portal Gastropathy in Child Pugh Class A, B, C in Chronic Liver Disease

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ABSTRACT

Liver disease progresses through four stages of inflammation, fibrosis, cirrhosis leading to hepatocellular carcinoma and liver failure.

Aims: To check the frequency of portal gastro-pathy in child Pugh class A, B, C in chronic liver disease.

Study Design: Cross sectional study.

Methodology: Patients (n=246) with chronic liver disease were included and classified based on the Child-Pugh classification and grouped into category A (good hepatic function), category B (moderately impaired hepatic function and category C (advanced hepatic dysfunction). Findings suggestive of portal gastropathy were noted in the endoscopy reports and were classified as mild or severe according to McCormack criteria. ALT, platelets, Haemoglobin and INR levels were also recorded.

Statistical analysis: Data was analyzed using SPSS version 26. Results were presented as frequency and percentage. Age was presented as mean± SD.

Results: Out of total 246 patients 155 (63%) were males and 91 (37%) were females. Mean age of the patients was 45.1 ± 10.1 years. 142 (57.7%) patients were of child PUGH class A, 66 (26.8%) were of class B and 38 (15.4%) were of class C. Frequency of portal gastropathy was 62.2% (n=153). In our study 57.7% patients in child PUGH class A, 69.6% of Child PUGH class B and 65.7% in PUGH class C were positive with PHG findings.

Conclusion: It was concluded that Portal hypertensive gastropathy was not significantly related to the Child Pugh classification of liver disease. However, an increase in ALT levels depicted the severity of liver damage.

Keywords: Child PUGH Classification, Chronic Liver Disease, Portal Hypertension and Portal Hypertensive Gastro-pathy.

INTRODUCTION

Every year in Pakistan, many people die due to liver disease caused by chronic hepatitis. Liver disease progresses through four stages of inflammation, fibrosis, cirrhosis leading to hepatocellular carcinoma and liver failure. Hepatitis is Among the top most reported diseases in Pakistan[1]. According to a survey seropositivity of chronic liver disease in Pakistan is 45.7% and 37.7% of cases are reported with cirrhosis[2]. Major cause leading to CLD include hepatitis A, B, C, D, E, infections, viruses, alcohol abuse etc[3]. Progressive deterioration in liver functions is ultimately seen in patients with CLD. Patients usually present with nonspecific symptoms which include fatigue, loss of appetite, sudden weight loss etc.

Portal hypertension, ascites, bleeding from varices, hepatic encephalopathy are among the widely noted complications associated with liver cirrhosis. Portal vein pressure gradient greater than 5mm hg is labelled as portal hypertension[4]. The gold standard for diagnosing this condition is to measure the hepatic venous pressure gradient[5]. The most common effect of this hypertension is observed on the gastric mucosa[4]. These Lesions in the gastric mucosa are termed as portal gastropathy. The exact mechanism behind its formation is unknown but various theories have been proposed which include alteration in local blood pressure, coagulation and hemodynamic factors and circulatory mediators in the gastric mucosa[6]. Clinically it usually manifests as gastrointestinal haemorrhage.

Portal gastropathy in Different populations worldwide have different prevalence in relation to CLD patients which ranges from 16 – 100%[7]. This widely different statistics may be due to lack of uniformity in patient selection, variations in the observer's perceptions and understandings etc. Severity of Portal gastropathy is directly related to the risk of gastric hemorrhage[8]. Rising disease burden and Scarcity of literature in Pakistani population regarding chronic liver diseases, its complications and management forced us to perform this study in our clinical setup. **Objectives:** To check the frequency of portal gastro-pathy in child Pugh class A, B, C in chronic liver disease.

METHODOLOGY

Present study was a descriptive cross sectional study. Informed consent was taken from the patients or guardians prior to enrolling the patients into the study. A Sample size of 246 was calculated to meet the objectives of our study ie power of test 80% with a confidence interval of 95% and alpha of 0.05 using frequency of portal hypertensive gastropathy as 80% in patients with chronic liver disease[9]. Non probability consecutive sampling technique was used. Patients who were diagnosed with chronic liver disease based on the history, clinical examination, radiological and laboratory investigations were included in the study. Those having any other ongoing respiratory, cardiac comorbid, unwilling to participate, taking drugs (NSAIDS, PPI) and CLD of unknown aetiology were excluded from the study.

Patients with chronic liver disease were classified based on the Child-Pugh classification and grouped into category A (good hepatic function), category B (moderately impaired hepatic function and category C (advanced hepatic dysfunction)[10]. Findings suggestive of portal gastropathy were noted in the endoscopy reports and were classified as mild or severe according to McCormack criteria[11]. ALT, platelets, Haemoglobin and INR levels were also recorded.

Statistical Analysis: Data was analyzed using SPSS version 26.0. Mean and SD were calculated for variables such as Age, mean HB, platelet count, ALT and INR. Percentage and Frequency was calculated for variables (categorical) such as gender, patients having portal gastropathy etc. Data Normality was assessed using Shapiro wilk test, which showed a parametric distribution of data. Chi square test was used to compare the frequency of portal gastropathy in CLD patients grouped on the basis of child PUGH classification. One way ANOVA was used to compare Hb, platelets, ALT and INR among Child PUGH class A, B and C. Independent samples T test was used to compare Hb, platelets, ALT and INR among patients with and without portal gastropathy. p value of ≤0.05 was considered to be significant.

RESULTS

Out of total 246 patients 155 (63%) were male and 91 (37%) were females. Mean age of the patients was 45.1 ± 10.1 years. Age range of the patients included n the study fell between 22 – 75 years. Basic characteristics of the study population were listed in table-1.

Table-1: Basic characteristics of study population (n=246)

Variables	Mean ± SD	Minimum	Maximum
Hb (g/dl)	9.01 ± 0.76	7.10	10.80
Platelets (x10^9/l)	133.67 ± 19.77	101.00	214.00
ALT (IU/L)	71.52 ± 14.08	46.00	109.00
INR	1.43 ± 0.13	1.09	1..82

142 (57.7%) patients were of child PUGH class A, 66 (26.8%) were of class B and 38 (15.4%) were of class C. Frequency of portal gastropathy was 62.2% (n=153) in our study population. 37.8% (n=93) patients with chronic liver disease presented with no signs of portal gastropathy. 58.5% (n=144) patients were having mild portal gastropathy changes and 3.7% (9) presented with severe changes as shown in Fig-1.

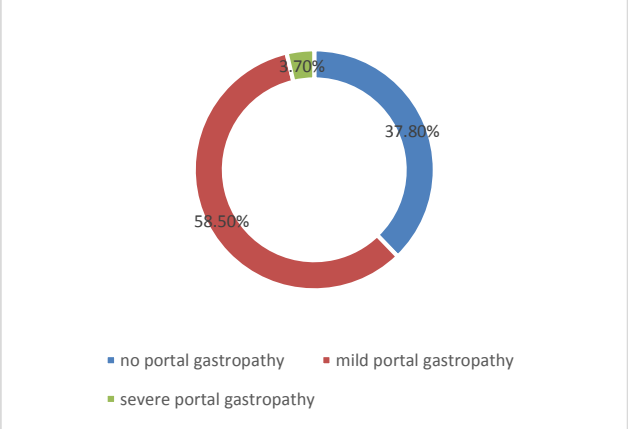

Fig-1: Frequency of portal gastropathy in CLD patients

Table 2: Distribution of portal gastropathy among male and female patients showed no significant difference.

Table-2: Distribution of portal gastropathy among male and female patients.				
Gender	No PG	Mild PG	Severe PG	p value
Male	55	93	7	0.4
Female	38	51	2	
Total	93	144	9	

Frequency of PG among CLD patients categorized on the basis of child PUGH class A, B and C was shown in Fig-2. Class A had the highest frequency of mild gastropathy but these differences were not statistically significant (p=0.2).

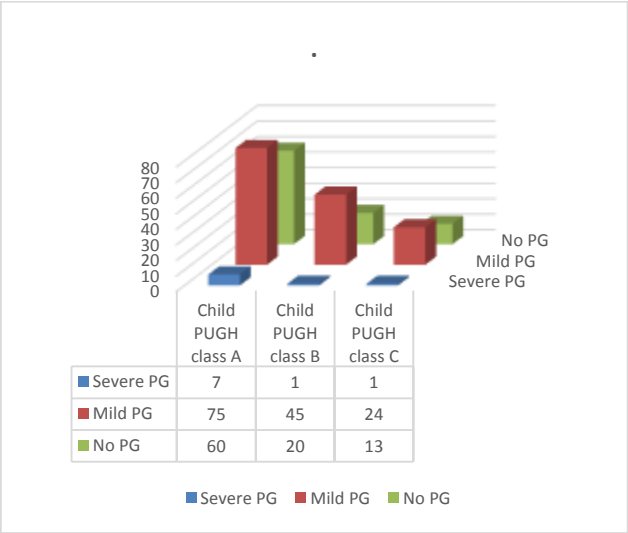

Fig-2: Frequency of portal gastro-pathy according to Child pugh Score

Laboratory parameters (Hb, platelet count, ALT and INR) were compared between child PUH classes A, B and C as shown in Table-3. ALT showed a statistically significant increase as the disease progressed from class A to class C showing deterioration in the liver functions. Hb, platelets and INR did not show any significant difference among three classes.

Table-3: Comparison of Laboratory Parameters among Child PUGH Classes

Parameter	Child Pugh class A	Child Pugh class B	Child Pugh class C	p value
Hb (g/dl)	9.09 ± 0.7	8.9 ± 0.7	8.8 ± 0.7	0.15
Platelets (x10^9/l)	134.8 ± 20.9	131.2 ± 15.7	133.3 ± 21.4	0.47
ALT (IU/L)	67.5 ± 11.8	71.1 ± 13.4	86.8 ± 12.0	0.001
INR	1.42 ± 0.13	1.44 ± 0.14	1.46 ± 0.12	0.40

Post hoc Tukeys comparison for ALT (at subset for alpha to be 0.5) among groups showed that there was a significant difference between groups A and C (p=0.001), B and C (p=0.001).

Insignificant differences were observed in laboratory parameters of CLD patients presenting with or without signs of portal gastropathy as depicted by Table-4.

Table-4: Comparison of Laboratory parameters among CLD patients with and without Portal Gastro-pathy

Parameter	Without portal gastro-pathy	With portal gastro-pathy	P-value
Hb (g/dl)	9.11 ± 0.72	8.95 ± 0.77	0.24
Platelets (x10^9/l)	132.80 ± 17.15	134.20 ± 21.25	0.27
ALT (IU/L)	71.47±14.9	71.54 ±13.5	0.26
INR	1.43 0.13	1.44 ± 0.13	0.71

DISCUSSION

On endoscopy of patients with liver cirrhosis various lesions of the gastric mucosa are observed. These changes in the morphology and architecture of gastric mucosa are termed as portal hypertensive gastropathy. In our study the prevalence of portal gastropathy was 62.2 %. Pratap et al in his study reported 66.6% prevalence of portal hypertensive gastropathy[12], out of which 80.6% had mild and 19.4% had severe condition. Marrache et al in his study concluded that 45 (62%) patients had portal hypertensive gastropathy out of which 41 patients had mild and only 4 had severe gastropathy[13]. Saleem et al in his study conducted in Lahore revealed that frequency of PHG in patients having hepatitis C induced liver cirrhosis is as high as 97.5% with Severe gastropathy observed in 61.6% of the patients[14], which was significantly higher than the results of our study.

Abbasi et al reported a 79.2% prevalence of PHG in cirrhosis[15]. He also concluded that there was a significant effect of raised portal vein pressure on esophageal mucosa, similar to that observed in the gastric mucosa. Primignani et al in a study of 373 patients with liver cirrhosis concluded that 80.2% had PHG[9]. 29% – 57% of the patients with portal hypertension usually have mild PHG, where as prevalence of severe PHG varies from 9%-46%[16]. In comparison to above mentioned statistics, our study revealed a lower prevalence of severe PHG (3.7%).

PHG is not an isolated phenomenon occurring in the gastric region. It is usually associated with an underlying liver cirrhosis leading to increased portal vein pressure. In our study 57.7% patients in child PUGH class A, 69.6% of Child PUGH class B and 65.7% in PUGH class C were positive with PHG findings. Contrary to our results Tiwari et al in his study reported that the highest prevalence of PHG was found in Child PUGH class C (35.9%)[12]. Sarin et al reported that Child PUGH class C Had 87% prevalence of PHG and class A had the least ie 13%[17]. Kumar et al reported that on multivariate analysis only Child PUGH class C is associated with presence of PG[18]. Sungkar et al in his study also showed association of portal gastropathy and child Pugh classification of liver disease. He revealed that PHG was 66.7% in class A, 96% in class B and 95.2% in class C. in this study they

further concluded that severity of portal gastropathy increased with increasing severity of the liver disease[19].

When a comparison was drawn among various laboratory features in 3 classes of Child Pugh scoring, only ALT showed a significant rise as the severity of liver disease increased. No significant difference was observed in laboratory parameters between patients with and without portal gastropathy. Evaluation of serum ALT levels remain an easily accessible test to establish liver damage in patients routinely suspected for any underlying liver disease. Laura et al however concluded that ALT has only moderate diagnostic accuracy in determining liver damage[20]. Jialing et al however showed a positive correlation between liver stiffness/ fibrosis and ALT levels[21].

No significant association between Hb, platelets, ALT, INR levels and PHG severity could be established. Although Our institute is a tertiary care hospital receiving patients from various regions of country, but being a single centre study is the limitation of our study. No liver biopsy was carried out to diagnose the extent of liver damage.

Limitations: Our study had limitations like financial constraints, lack of resources, genetic workup and short duration of study.

CONCLUSION

It was concluded that prevalence of portal gastro-pathy was 62.2 % in patients with chronic liver disease in our population. Portal hypertensive gastropathy was not significantly related to the Child Pugh classification of liver disease. However, an increase in ALT levels depicted the severity of liver damage.

Authors' Contribution:

AH, SAAG &NKN: Conceptualized the study, analyzed the data, and formulated the initial draft.

SHT&IR: Contributed to the proof reading.

RS & TL: Collected data.

Acknowledgements: I am thankful to Allah and all my colleagues for their help.

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