Original Article

Frequency of peptic ulcer in patients having decompensated cirrhosis of liver.

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ABSTRACT

Introduction Hepatitis B and C are spreading like an endemic disease in developing countries like Pakistan, due to many reasons. The late diagnosis of HCV and HBV infection has resulted in increased number of patients with decompensated liver disease. One of the common complications of cirrhosis is upper GI bleed caused by peptic ulceration in UK. Local data shows peptic ulceration was the second commonest of the lesions causing upper GI bleed after esophegeal varices. Present study was conducted to determine the frequency of peptic ulcer in patients having decompensated cirrhosis of liver presenting with upper GI bleed, also to emphasize the importance of primary prophylaxis with proton pump inhibitors for prevention of peptic ulcer in these patients.

Study design: Descriptive study.

Setting: MU-III Allied Hospital Faisalabad.

Materials and Methods: Hundred consecutive patients having decompensated cirrhosis of liver were selected according to pre designed proforma and endoscopy was performed to determine the site of bleeding, from Jun to November 2007.

Results: This study showed peptic ulcer as the second most important cause of upper GI bleed (34%) after esophegeal varices (57%), also decompensate cirrhotics have increased incidence of peptic ulceration (34%) as compared to general population (8.3%). Also significant relationship between source of upper GI bleed and serum albumin level in patients having decompensated cirrhosis of liver. (P value = .019) was found.

Conclusions: There is definitely an increased frequency of bleeding peptic ulcer in patients having decompensated liver cirrhosis as compared to general population necessitating the need of primary prophylaxis of peptic ulcer with proton pump inhibitor in decompensated cirrhotics.

Key Words: Upper GI bleed, proton pump inhibitor, peptic ulcer, esophegeal varices, HCV, HBV and decompensated cirrhosis.

INTRODUCTION

A peptic ulcer is a disruption of mucosal integrity and may occur any where between lower esophagus to jejunum [1,2]. Its prevalence in general population is 10% of all adults [2].

Decompensation refers to if any of jaundice, ascities, hypoalbuminemia or encephalopathy is present in patient having cirrhosis of liver [3].

Specific chronic disorders have been associated with peptic ulcer disease and one of the strong associations is cirrhosis of liver [1,4,5,6,7,8,9]. Peptic ulceration is more common in cirrhotics as compared to general population [10,11].

Uncomplicated peptic ulcer causes epigastria pain and less commonly nausea vomiting and weight loss[2]. Acute bleeding ulcer causes haematemesis and malena[2] and is one of the most common emergencies in UK [2] as well as in Pakistan [13].

Upper GI bleed either from peptic ulcer or esophegeal varices is a major complication of cirrhosis [14,11].

Viral infections are spreading like the fire of jungle due to poor hygienic conditions, making of tattoo marks, use of non-sterile syringes, lack of screening facilities for blood transfusion, increase in the number of addicts and ignorance among the masses [15,16]. The late diagnosis of HCV and HBV infection has resulted in an increased pool of patients with decompensated liver disease. Because of high frequency of peptic ulcer in all chronic ailments particularly chronic liver disease, [10,1,4,5,6,7,8,9] the

study was conducted to emphasize the need of primary prophylaxis with proton pump inhibitors in decompensate cirrhotic in an attempt to decrease the mortality, morbidity and hospital admission as far as upper GI bleed due to peptic ulcer as a cause is concerned.

Research work in this particular aspect of study is lacking both nationally and internationally.

Data Collection

The study was performed on 100 patients admitted in medical unit III of Allied hospital Faisalabad, sorted out by inclusion, exclusion criteria and according to the attached Proforma.

Patients were diagnosed as decompensated cirrhosis of liver on basis of history (jaundice, abdominal distension, and encephalopathy) laboratory investigations (serum billirubin, serum albumin, and prothrombin time) and abdominal ultrasound (to document ascities). Then endoscopy was performed to document the source of bleeding.

Data Analysis Procedure

The study was analyzed on SPSS-Ver-10 for windows. Chi-Square statistics were applied on different variables to study significance. In the study variable of interest were age, gender, serum billirubin, serum albumin, encephalopathy, prothrombin time, ascities and source of upper GI bleed. Among these, source upper GI bleed is dependent variable and remaining are independent variables. Level of significance was P = 0.05.

RESULTS

In this study 100 patients having decompensated cirrhosis of liver presenting with upper GI bleed were included. Among which 34% were having peptic ulcer as compared to varices 57% and only 9% were having non ulcer non variceal lesion as a cause of upper GI bleed (Figure 1)

Cross tabulation of various variables in the study to the etiological distribution of upper GI bleed in decompensated cirrhotic population was done to determine the relation of upper GI bleed with sex, age, serum billirubin, serum albumin, amount of ascities, prolongation of prothrombin time and grade of encephalopathy.

This study showed a significant relationship between source of upper GI bleed and serum albumin level in patients having decompensated cirrhosis of liver. (P value = .019). (Table 3). The relation of upper GI bleed with remaining variables proved insignificant.

Fig. 1:
Frequency of Various Etiologies of Upper GI

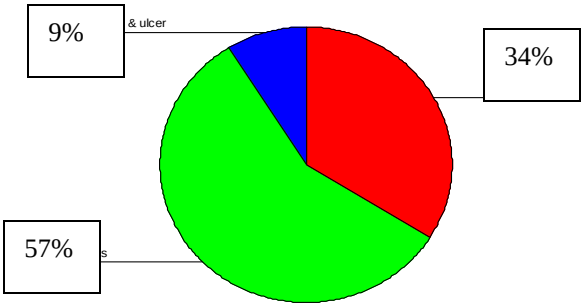

Table-1: Gender.

Parameter & Frequency Gender	Source of Bleed Ulcer	Varices	Non ulcer non variceal	Total
Male 56%	20%	34%	2%	56%
Female 44%	14%	23%	7%	44%
Total 100	34%	57%	9%	100

Degree of freedom = 2
P Value = .101

Table 2: Age.

Parameter & Frequency Age (year)	Source of Bleed Ulcer	Varices	Non ulcer non variceal	Total
<40 41%	8%	27%	6%	41%
41-60 43%	20%	21%	2%	43%
>60 16%	6%	9%	1%	16%
Total 100	34%	57%	9%	100

Degree of freedom = 4
P Value = .087

Table 3: Serum Albumin.

Parameter & Frequency Serum Albumin (mg/dl)	Source of Bleed Ulcer	Varices	Non ulcer non variceal	Total
>35 12%	5%	3%	4%	12%
28-35 59%	20%	36%	3%	59%
<28 29%	9%	18%	2%	29%
Total 100	34%	57%	9%	100

Degree of freedom = 4
P value = .019

Table-4: Serum Billirubin.

Parameter & Frequency Serum Billirubin (mg/dl)	Source of Bleed Ulcer	Varices	Non ulcer non variceal	Total
1-2mg/dl > normal 17%	6% 2%	9%		17%
2.1-3mg/dl > normal 20%	5%	14%	1%	20%
>3mg/dl > normal 63%	23%	34%	6%	63%
Total 100	34%	57%	9%	100

Degree of freedom = 4
P value = .761

Table-5: Encephalopathy

Parameter & Frequency Encephalopathy	Source of Bleed Ulcer	Varices	Non ulcer non variceal	Total
Non 48%	14% 6%	28%		48%
Mild to Moderate 35%	14% 3%	18%		35%
Severe 17%	6% 0%	11%		17%
Total 100	34% 9%	57%		100

Degree of freedom = 4
P value = .512

Table 6: Prothrombin Time

Parameter & Frequency Prothrombin Time	Source of Bleed Ulcer	Varices	Non ulcer non variceal	Total
3 Sec. prolonged 50%	14%	29%	7%	50%
3-6 Sec. prolonged 27%	12%	15%	0%	27%
>6 Sec. prolonged 23%	8%	13%	2%	23%
Total 100	34%	57%	9%	100

Degree of freedom = 4
P value = .263

Table-7: Ascities

Parameter & Frequency Ascities	Source of Bleed Ulcer	Varices	Non ulcer non variceal	Total
Non 26%	10%	15%	1%	26%
Mild to Moderate 67%	22%	38%	7%	67%
Severe 7%	2%	4%	1%	7%
Total 100	34%	57%	9%	100

Degree of freedom = 4
P value = .447

DISCUSSION

The epidemiology of various causes of upper G.I. bleeding has been changing in recent years[17,18]. Variations in disease pattern from time to time require the need for periodic studies to define the changing etiological distribution for continuous medical education and learning.

Viral infections causing cirrhosis are spreading in developing countries like Pakistan due to poor hygienic conditions, making of tattoo marks, use of non-sterile syringes, lack of screening facilities for blood transfusion, increase in the number of addicts

and ignorance among the masses [15,16]. HBV and HCV induced liver disease has markedly increased during the last decade [19].

The unchecked transmission and late diagnosis of HCV and HBV infection has resulted in an increased pool of patients with decompensated cirrhosis. Also the increase in number of patients in recent year with upper GI bleed with peptic ulcer as source propelled us to select this topic for study. This study was conducted to determine the frequency of peptic ulcer in patients having decompensated cirrhosis of liver and also to collect sufficient evidence for recommendation of prophylactic use of proton pump inhibitors to prevent peptic ulcer in decompensated cirrhosis.

There are some studies conducted in Pakistan which are comparable to current study as for as its first objective (frequency of peptic ulcer in decompensated cirrhosis of liver) is concerned.

Current study is comparable to the study conducted in Mayo hospital Lahore Pakistan in which the frequency of peptic ulcer in cirrhotic patients was found to be 32% as compared to varices which were almost double[20]. The result of the presented study showed, frequency of peptic ulcer 34% and of varices 57%, as a source of upper GI bleed in sample population of decompensated cirrhotics.

Present study is also comparable to the study conducted by Javed Iqbal Farooqi and Rukhsana Javed Farooqi, mentioning that half of the patients with non-variceal acute upper GI bleed are having peptic ulcer disease[21]. Present study showed the same distribution of peptic ulcer and esophegeal varices in its sample population.

Current study is also comparable to the western published data indicating increased frequency of peptic ulcer in patients having cirrhosis[1,2,5,6,7,8,9]compared to general population [10,11]. In Pakistan frequency of peptic ulcer in general population is 8.3%[28] while it is much increased in cirrhotics (34%) as shown in present study.

Factors increasing the likelihood of development of peptic ulcer in cirrhotic population, as mentioned in foreign data, include the male gender[22], seropositivity for H. pylori[23,2,49,25], advanced cirrhosis as indicated by child's grade[9], greater average gastric ph [26] and grade of portal hypertensive gestropathy [6]. In present study male gender seemed to be having statistically insignificant relation with development of peptic ulcer in cirrhosis (P-value .101). Among the features of decompensation serum albumin seemed to be having statistically significant relation with development of peptic ulcer (P-value .019), this relation is comparable to the increased risk of peptic ulcer in advanced cirrhosis as indicated by child's grade[9] (mentioned in western data).

Unfortunately local as well as foreign data about the second objective of study (prophylactic use of proton pump inhibitors for development of peptic ulcer in patients having decompensated cirrhosis) is lacking. However, some proved facts are summarized here.

Specific chronic disorders like cirrhosis of liver have been associated with peptic ulcer disease [1,4,5,6,7,8,9]. Peptic ulceration is more common in cirrhotics as compared to general population [10,11]. Upper GI bleed either from peptic ulcer or esophegeal varices is a major complication of cirrhosis [14,11]. Upper GI bleed in turn is a major contributor of precipitating hepatic encephalopathy [27]. Bleeding peptic ulcer is a major cause of mortality in cirrhotic patients [4]. Acute bleeding peptic ulcer occurs in more then 15% of cirrhotic [25]. So increased frequency and morbidity of peptic ulcer in patient having decompensated cirrhosis of liver is a proven fact [1,14,4,5,7,8,9,10].

Also there is an inadequate response of H_2 receptor blockers in prevention or treatment of peptic ulcer in cirrhosis [7], while long term use of proton pump inhibitors is safe in cirrhosis [6].

All these proven facts of increased frequency of peptic ulcer and increased morbidity and mortality due to bleeding peptic ulcer in cirrhotics emphasize the importance of prophylactic use of proton pump inhibitors for peptic ulcer in decompensated cirrhotics.

LIMITATION OF THE STUDY:

Number of patients in this study is very small nevertheless it may be taken as an ignition to conduct more research work for recommendation of prophylactic use of proton pump inhibitors for peptic ulcer in decompensated cirrhotics.

CONCLUSION

There is an increased frequency of peptic ulcer in decompensated cirrhosis of liver as compared to general population. And it is the second most common cause of upper GI bleed in decompensated cirrhotic

population. Peptic ulcer is causing more then half cases of non variceal upper GI bleed in cirrhotic patients. Prophylactic medication for peptic ulcer in decompensated cirrhotics can not only decrease morbidity but also decrease hospital admissions hence health budget expenditures.

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