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RESEARCH ARTICLE
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FREQUENCY OF GASTRO ESOPHAGEAL VARICES IN CIRRHOTIC PATIENTS WITH SPLENOMEGALY

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Abstract

Background: Cirrhosis of the liver, particularly in the presence of splenomegaly, is a known risk factor for the development of gastroesophageal varices (GEVs), which are one of the major causes of upper gastrointestinal bleeding. Understanding the frequency of GEVs in cirrhotic patients with splenomegaly can help prioritize screening and management strategies.

Methods: A total of 225 cirrhotic patients with confirmed splenomegaly were included in this retrospective study. Endoscopic examination was performed to assess the presence and severity of GEVs. The study analyzed patient demographics, clinical characteristics, liver function tests, and splenic size measurements. The relationship between splenomegaly and the frequency of GEVs was evaluated using statistical methods.

Results: Of the 225 patients, 68% had gastroesophageal varices, with 20% presenting with large varices, associated with higher risks of bleeding. A significant correlation was observed between the size of the spleen and the presence of GEVs ($p < 0.05$). Additionally, patients with advanced cirrhosis (Child-Pugh class C) had a higher prevalence of varices (80%), compared to those with compensated cirrhosis (56%, $p < 0.01$).

Conclusion: This study confirms a high frequency of gastroesophageal varices in cirrhotic patients with splenomegaly, particularly those with advanced liver disease. The findings highlight the importance of screening for GEVs in these patients to prevent complications such as bleeding and rupture.

Keywords: Cirrhosis, Splenomegaly, Gastroesophageal Varices, Endoscopy, Liver Disease, Upper Gastrointestinal Bleeding.

Introduction

Cirrhosis is a chronic liver condition that results from long-term liver damage, characterized by progressive fibrosis, hepatocellular destruction, and a marked alteration in the structure of the liver [1]. One of the significant complications of cirrhosis is the development of gastroesophageal varices (GEVs), which are dilated veins in the esophagus and stomach, primarily due to portal hypertension [2]. The increased pressure in the portal venous system, stemming from liver fibrosis and cirrhosis, leads to the development of collateral circulation, including the formation of GEVs. These varices pose a serious risk because they are prone to rupture, leading to upper gastrointestinal bleeding, a life-threatening event that significantly impacts morbidity and mortality in cirrhotic patients [3].

The relationship between portal hypertension and gastroesophageal varices is well established, with variceal formation occurring in the majority of cirrhotic patients with significant portal hypertension [4]. Splenomegaly, or an enlarged spleen, is another hallmark of cirrhosis, and it often occurs as a consequence of the increased pressure in the portal venous system. The spleen's enlargement in these patients serves as an important marker for the degree of portal hypertension and the severity of cirrhosis [5]. As the spleen increases in size, it may reflect the extent of portal vein thrombosis, variceal formation, and hemodynamic changes that correlate with the development of gastroesophageal varices. While it is well known that splenomegaly is associated with portal hypertension, the frequency and severity of gastroesophageal varices in cirrhotic patients with splenomegaly are not always clear. The variability in the presence of GEVs within this patient group highlights the need for systematic studies to establish the prevalence and risk factors for varices in patients with cirrhosis and splenomegaly. Furthermore, while endoscopic screening remains the gold standard for detecting gastroesophageal varices, many patients may not be routinely screened due to limited access to resources or clinical guidelines that do not prioritize screening based on splenomegaly alone [5].

In this context, understanding the frequency of gastroesophageal varices in cirrhotic patients with splenomegaly becomes crucial, as this could help identify high-risk patients who require early screening and timely interventions to prevent variceal bleeding [6]. The role of splenic size in predicting the presence of GEVs, particularly in the absence of overt symptoms, is of significant clinical importance. Previous studies have shown a correlation between larger spleens and the increased presence of varices, yet the strength of this association and its impact on clinical outcomes remain underexplored [7]. Additionally, cirrhotic patients with advanced liver disease (such as those classified as Child-Pugh class B or C) may present with a higher incidence of GEVs, further emphasizing the need for targeted screening strategies. This study aims to investigate the frequency of gastroesophageal varices in cirrhotic patients with splenomegaly and examine the relationship between splenic size, Child-Pugh classification, and the development of GEVs [8]. By analyzing the prevalence of varices in these patients, the study seeks to provide a comprehensive understanding of the role of splenomegaly in GEV formation, and establish effective guidelines for early detection and preventive care in cirrhotic patients at risk for gastrointestinal bleeding. Given the high morbidity and mortality associated with variceal rupture, this study's findings could help in improving clinical management strategies and patient outcomes, particularly in cirrhotic patients with advanced disease and splenic enlargement [9].

Methodology

This was a retrospective cohort study conducted on 225 patients diagnosed with cirrhosis and confirmed splenomegaly. Patients were selected from the hospital database. All participants underwent clinical evaluation, including laboratory tests (liver function tests, complete blood count) and ultrasound imaging to measure splenic size. Only patients who had splenomegaly (defined as a spleen size greater than 13 cm) were included in the study.

Inclusion Criteria

- Patients with a confirmed diagnosis of cirrhosis (via clinical, radiological, and/or histological evidence).
- Presence of splenomegaly (spleen size > 13 cm).
- Age between 18 and 80 years.
- Available clinical and endoscopic data.

Exclusion Criteria

- Patients with malignant liver tumors or other systemic diseases.
- Previous history of variceal bleeding or esophageal variceal ligation.
- Patients who did not undergo endoscopic examination.

Data Collection

Data were collected from medical records, including demographic information (age, gender), clinical features (history of ascites, encephalopathy, etc.), splenic size, and endoscopic findings of gastroesophageal varices. Endoscopic findings were classified into no varices, small varices, and large varices based on the modified Paquet classification. The Child-Pugh classification was used to assess the severity of cirrhosis, and patients were categorized as Child-Pugh class A, B, or C.

Statistical Analysis

The relationship between splenomegaly and the presence of gastroesophageal varices was assessed using chi-square tests for categorical variables and t-tests for continuous variables. The correlation between spleen size and the presence of varices was evaluated using Pearson's correlation coefficient. Multivariate logistic regression was used to identify independent predictors of GEVs in cirrhotic patients. A p-value < 0.05 was considered statistically significant. All statistical analyses were performed using SPSS version 25.0.

Results

This table summarizes the demographics and clinical characteristics of the 225 cirrhotic patients included in the study. The mean age of participants was 55.6 ± 12.3 years, with 60% males. Splenic size was significantly larger in patients with gastroesophageal varices (GEVs) (17.1 ± 2.6 cm vs. 15.2 ± 2.3 cm, p=0.01). Among the Child-Pugh classifications, class C patients had a higher prevalence of varices (80%) compared to class A (56%) and class B (72%) (p=0.004). The table highlights the correlation between larger spleens and advanced liver disease in predicting variceal formation.

Table 1: Baseline Demographics and Clinical Characteristics

Characteristic	Total (n=225)	Varices Present (n=153)	Varices Absent (n=72)	p-value
Mean Age (Years)	55.6 ± 12.3	56.2 ± 11.5	54.3 ± 13.1	0.22
Male (%)	60%	62%	57%	0.49
Mean Spleen Size (cm)	16.3 ± 2.5	17.1 ± 2.6	15.2 ± 2.3	0.01
Child-Pugh Class A (%)	38%	30%	50%	0.04
Child-Pugh Class B (%)	41%	44%	35%	0.14
Child-Pugh Class C (%)	21%	26%	15%	0.07

This table shows the relationship between spleen size and the presence of GEVs. As splenic size increases, the prevalence of varices also rises. Patients with a spleen size of >18 cm had an 85% prevalence of GEVs, compared to 68% in patients with spleens between 16-18 cm, and 48% in patients with spleens between 13-15 cm ($p < 0.001$). This data indicates that larger spleens are strongly associated with a higher incidence of varices in cirrhotic patients.

Table 2: Frequency of Gastroesophageal Varices by Splenic Size

Spleen Size (cm)	Varices Present (%)	Varices Absent (%)	p-value
13-15 cm	48%	52%	0.04
16-18 cm	68%	32%	0.001
>18 cm	85%	15%	<0.001

This table assesses the association between cirrhosis severity (Child-Pugh classification) and the presence of GEVs. Patients with Child-Pugh class C cirrhosis exhibited the highest prevalence of varices (80%), followed by those in Child-Pugh class B (72%), and class A (56%) (p = 0.004). This confirms that advanced liver disease is a significant risk factor for variceal development in cirrhotic patients.

Table 3: Association of Cirrhosis Severity and Gastroesophageal Varices

Cirrhosis Severity	Varices Present (%)	Varices Absent (%)	p-value
Child-Pugh A	56%	44%	0.02
Child-Pugh B	72%	28%	0.01
Child-Pugh C	80%	20%	0.004

This table presents how Child-Pugh class and splenic size together influence variceal formation. For patients with spleen size 13-15 cm, Child-Pugh class A patients had the lowest prevalence of GEVs (45%), while class C had only 5% prevalence. In those with spleens larger than 18 cm, Child-Pugh class C patients had an 85% prevalence of varices, indicating a strong correlation between spleen size and cirrhosis severity for variceal formation.

Table 4: Distribution of Gastroesophageal Varices by Child-Pugh Class and Spleen Size

Spleen Size (cm)	Child-Pugh Class A (%)	Child-Pugh Class B (%)	Child-Pugh Class C (%)	p-value
13-15 cm	45%	50%	5%	0.02
16-18 cm	30%	55%	15%	0.01
>18 cm	20%	45%	35%	0.001

This table explores the link between platelet count and the presence of GEVs. A low platelet count (<100 $x10^3/\mu L$) was associated with a 75% prevalence of varices, compared to 60% in those with platelet counts between 100-150 $x10^3/\mu L$ and 42% in patients with platelet counts >150 $x10^3/\mu L$ (p=0.002). This suggests that thrombocytopenia is a marker of severe portal hypertension and is strongly associated with the presence of gastroesophageal varices.

Table 5: Association of Platelet Count and Presence of Gastroesophageal Varices

Platelet Count ($x10^3/\mu L$)	Varices Present (%)	Varices Absent (%)	p-value
>150	42%	58%	0.03
100-150	60%	40%	0.01
<100	75%	25%	0.002

This table shows the relationship between hepatic encephalopathy and variceal formation. Patients with Grade III-IV hepatic encephalopathy had an 85% prevalence of GEVs, significantly higher than those with Grade I-II encephalopathy (70%) and patients without encephalopathy (62%) (p=0.003).

Table 6: Incidence of Gastroesophageal Varices and Hepatic Encephalopathy

Hepatic Encephalopathy	Varices Present (%)	Varices Absent (%)	p-value
Absent	62%	38%	0.04
Grade I-II	70%	30%	0.02
Grade III-IV	85%	15%	0.003

Discussion

This study aimed to evaluate the frequency of gastroesophageal varices (GEVs) in cirrhotic patients with splenomegaly and assess their correlation with clinical factors such as Child-Pugh classification, splenic size, and platelet count. The findings confirm that splenomegaly is a significant risk factor for the development of GEVs in cirrhotic patients. A high prevalence of varices was observed in patients with splenomegaly, with 68% of the total cohort showing evidence of GEVs, and this prevalence increased significantly in patients with larger spleens (≥18 cm). These results suggest that splenomegaly is not just a clinical marker but also a reliable predictor of portal hypertension and variceal formation in cirrhosis, emphasizing its role in clinical practice for screening high-risk patients [10].

The study also highlighted the relationship between liver disease severity and variceal presence. Cirrhotic patients in Child-Pugh class C had a significantly higher prevalence of GEVs (80%) compared to those in Child-Pugh class A (56%) and Child-Pugh class B (72%), with a p-value of 0.004. This aligns with previous studies that have shown that patients with more advanced cirrhosis tend to develop more severe portal hypertension, leading to the formation of larger and more frequent varices. Patients with advanced liver disease are at greater risk of experiencing complications from variceal bleeding, which significantly impacts morbidity and mortality. Thus, the presence of splenomegaly can help identify high-risk patients who may require more frequent or earlier endoscopic screening to detect varices before they lead to bleeding events [11]. Moreover, the study observed that splenic size has a significant correlation with variceal presence, with patients whose spleens measured >18 cm exhibiting an 85% prevalence of varices. This finding suggests that as splenic size increases, the likelihood of developing GEVs also rises. This could be due to the degree of portal hypertension being proportional to splenic enlargement, which in turn promotes the formation of varices in the gastroesophageal region [12]. While this is an important clinical observation, further studies are needed to determine whether ultrasound-based spleen size measurements could serve as an accessible and non-invasive screening tool for GEVs in cirrhotic patients, especially in settings where endoscopic screening is not readily available.

The platelet count was another important factor in the study's findings. Patients with low platelet counts (<100 $x10^3/\mu L$) had a 75% prevalence of varices, which further supports the hypothesis that thrombocytopenia is a reflection of severe portal hypertension and may serve as an additional clinical indicator for variceal screening [13]. Thrombocytopenia in cirrhotic patients is often caused by hypersplenism due to splenomegaly, which leads to sequestration of platelets in the enlarged spleen. This phenomenon is closely related to the increased portal pressure, which further promotes the development of varices. Therefore, platelet count could serve as an additional risk stratification tool to determine which cirrhotic patients are at higher risk for variceal bleeding and would benefit from early endoscopic screening. A particularly striking finding in this study was the strong association between hepatic encephalopathy and the presence of GEVs [14]. Patients with Grade III-IV hepatic encephalopathy exhibited an 85% prevalence of varices, which is consistent with the notion that severe liver dysfunction increases the likelihood of developing varices. This is likely due to the extent of portal hypertension and reduced liver function, which in turn affect the hemodynamics of the portal venous system [15]. The development of encephalopathy often reflects significant hepatic decompensation, which is commonly accompanied by an increase in splenomegaly and a higher likelihood of variceal formation. Patients with hepatic encephalopathy may thus represent a high-risk subgroup that requires priority screening for varices to prevent catastrophic bleeding events [16].

Furthermore, this study provides important insights into the clinical management of cirrhotic patients with splenomegaly. Given the high prevalence of varices, particularly in those with larger spleens and advanced cirrhosis, early identification of GEVs through endoscopic screening is critical [17]. This approach is crucial for primary prevention of variceal bleeding and may involve the use of pharmacological interventions (such as beta-blockers) or endoscopic interventions (such as variceal ligation). Implementing a screening strategy based on splenic size and Child-Pugh classification may improve outcomes for cirrhotic patients, as it would allow for earlier detection of varices and more

proactive management of portal hypertension. While the findings are significant, this study has several limitations. First, it is a retrospective study, and thus selection bias may exist, especially in the choice of patients who underwent endoscopic screening. Secondly, while the study demonstrated a strong correlation between splenic size and variceal presence, the cross-sectional nature of the study does not allow for an evaluation of causality. Future prospective studies are needed to examine whether splenomegaly progression over time correlates with the development of varices. Additionally, longitudinal studies could help establish whether early screening based on splenomegaly and clinical markers like platelet count and hepatic encephalopathy lead to better patient outcomes in terms of variceal bleeding prevention [18].

Conclusion

This study confirms a high frequency of gastroesophageal varices in cirrhotic patients with splenomegaly. The findings underscore the importance of early screening for GEVs in these patients, especially those with advanced cirrhosis and larger spleen sizes, to prevent variceal rupture and associated bleeding complications.

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