COLLEGE OF HEALTH SCIENCES

DEPARTMENT OF INTERNAL MEDICINE

Incidences of gastroesophageal varices composite outcomes of cirrhotic and non-cirrhotic adult patients with portal hypertension in Addis Ababa: A Comparative Longitudinal Institution Based Study

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ADDIS ABABA UNIVERSITY

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Abbreviation and acronym

CPH Cirrhotic Portal Hypertension

GI Gastrointestinal

NCPH Non-Cirrhotic Portal Hypertension

OS Overall survival

PH Portal Hypertension

Portal vein thrombosis (PVT) vein thrombosis

TASH Tikur Anbessa Specialized hospital

WHO World Health Organization

Abstarct

Background: Gastroesophageal varices (GEV) are common in portal hypertension. While their incidence and outcomes may differ between cirrhotic and non-cirrhotic patients, comparative data, particularly from Ethiopia, are limited.

Objective: To compare the incidences of GEV composite outcomes among cirrhotic vs. non-cirrhotic patients with portal hypertension in selected hospitals in Addis Ababa Ethiopia.

Methods: This retrospective longitudinal study was conducted in Tikur Anbessa Specialized Hospital a tertiary referral center in Addis Ababa, including data from December 2021 to December 2024. An equal number of patients with cirrhotic and non-cirrhotic portal hypertension (PH) were included. Data were extracted from medical charts and electronic records. Descriptive statistics summarized baseline characteristics, while Chi-square and independent t-tests were used to compare clinical features and composite outcomes between groups. A p-value < 0.05 was considered statistically significant.

Results: A total of 160 patients diagnosed with portal hypertension were included in the study, comprising 77(48.1%, 95% CI: 40.4%-55.9%) patients with Non Cirrhotic Portal Hypertension (NCPHT) and 83(51.8%, 95% CI: 44.1%–59.6%) with Cirrhotic Portal Hypertension (CPHT). The mean age was 36.95 ± 13.34 years, with a predominance of male patients. Non-cirrhotic patients were significantly younger. Smoking, alcohol use, ascites, spontaneous bacterial peritonitis, AST, and ALP levels were significantly higher in the CPHT group. Schistosomiasis was the leading cause of NCPHT, while hepatitis B was the most common etiology of CPHT. The composite outcome was initially observed in 76.3% of patients, decreasing to 61.3% at one year and 8.8% by the end of the follow-up period. There was no significant difference in outcomes between the CPHT and NCPHT groups at baseline (79.5% vs. 72.7%) or at two years (6% vs. 6.5%), with both groups showing comparable improvement over time.

Conclusion and recommendations: Despite distinct baseline characteristics and aetiologies, cirrhotic and non-cirrhotic portal hypertension patients had comparable composite outcomes. Early detection and management of gastroesophageal varices are essential in both groups. Further studies are needed to guide long-term care.

Keywords: Cirrhotic portal Hypertension, Non-cirrhotic portal hypertension, oesophageal varices, Ethiopia

1. Introduction

1.1 Background

Portal hypertension (PH) is a common clinical condition caused by elevated portal pressure, which is defined as a hepatic venous pressure gradient of more than 5 mm/hg. It is primarily caused by intrahepatic resistance to blood flow and obliteration of the portal vein, which results in increased pressure [1] The elevated pressure leads to the development of portosystemic collateral and predisposition varices and other complications like splenomegaly and hyperkinetic circulatory system [2]

Portal hypertension can develop on the background non-cirrhotic liver, or cirrhotic liver disease [3]. Non-cirrhotic portal hypertension (NCPH) encompasses vascular and parenchymal liver diseases, which result in portal hypertension and its complications in the absence of cirrhosis[4.5]. The spectrum of NCPH is diverse and is characterized by obliteration of the portal venous system at different levels that is affecting the prehepatic, intrahepatic (without cirrhosis), or posthepatic vasculature, leading to increased portal pressure [6]. In some instances the cause of portal hypertension is not apparent even after exclusion of cirrhosis and obstruction of the portal system with liver biopsy and imaging respectively and with no evidence for the presence of other known portal hypertension causes This entity is called Idiopathic non-cirrhotic portal hypertension which is mentioned as part of NCPH in different literatures with different incidence all over the world (35)

Overall, cirrhotic liver disease is the most common cause of PH globally, and non-cirrhotic PH accounts for less than 10% of the cases [7]. Compared to the Western countries, non-cirrhotic PH is relatively common in developing countries including African countries and India. This might be related to the differences in the etiologies across the regions [5]. Schistosomiasis and portal vein thrombosis are the main cause of non-cirrhotic PH in developing countries[8].

Gastroesophageal variceal (GEV) is considered one of the major fatal complications in patients with hepatic cirrhosis and portal hypertension [9]. It appeared in approximately 50% of patients with cirrhosis. Despite an improvement in treatment strategy, variceal bleeding is still associated with a 6-week mortality rate of 12–26% in cirrhotic patients. Untreated, the risk of recurrent

bleeding is also high. In non-cirrhotic patients, variceal bleeding is also the most common clinical feature [10].

Although previous studies in Ethiopia, including those from Gondar University Hospital and St. Paul's Millennium Medical College, have reported a high prevalence of GEV among cirrhotic patients (52% and 80%, respectively), these findings are limited to cirrhosis-related GEV. There is a significant gap in the literature regarding the burden and characteristics of GEV in patients with NCPH[11]. Schistosomiasis, one of the neglected tropical diseases, remains highly prevalent in Ethiopia, affecting millions of people [18]. In parallel, there have been an increasing number of patients evaluated in haematology units for various conditions such as myeloproliferative neoplasms and both inherited and acquired thrombophilia. These trends suggest a growing burden of NCPH and its major complication, GEV. Despite this, data on the incidence and outcomes of GEV in the context of NCPH remain limited in Ethiopia. Therefore, this study aims to compare the incidence and related outcomes of GEV using a longitudinal study design.

1.2 Statement of the problem

Esophageal varices are the major cause of mortality and morbidity among patients with PH [12]. In cirrhotic patients, nearly 50% of patients with GEV experience bleeding at some point, and 10% to 20% of them die in the six weeks following the episode [13]. Among NCPH patients, about 75 % of these patients had GEV at the initial endoscopy [14]. Mortality due to variceal bleeding is significantly lower in NCPH than that observed in cirrhotic patients, likely because of a preserved liver function, about 3% at 6 weeks[15].

Non-cirrhotic portal hypertension may develop esophageal varices, with differing rates of bleeding and outcomes compared to cirrhotic patients [16]. The prevalence of esophageal varices among NCPH patients is not well-defined, but studies suggest it varies depending on the underlying etiologies such as schistosomiasis [17]. In Ethiopia, for example, around 37.5 million people are at a high risk of developing schistosomiasis, and 5.01 million people have the disease, so we expected a large number of patients with NCPH [18].

Previously, a majority of studies investigating therapy, etiologies, and prognosis focused on gastro-esophageal varices associated with cirrhotic portal hypertension [19] NCPH-related

varices can be challenging due to late presentation because of the absence of cirrhosis and the complexity of the underlying vascular pathology. Effective treatment and preventive strategies for variceal bleeding in NCPH patients require a clear understanding of the risk factors and progression of varices in this population. Hence the current study will assess the composite incidences of GEV among NCPH compared to patients with GEV related to cirrhotic portal hypertension patients among adults in selected hospitals of Addis Ababa.

1.3 Significance of the study

Understanding the incidence of GEV composite outcomes in patients with portal hypertension on the background of the cirrhotic and non-cirrhotic liver is essential to provide insights into disease progression and risk stratification. For cirrhotic patients, early detection and intervention can prevent fatal bleeding and decompensation, while in non-cirrhotic patients, managing variceal bleeding and underlying conditions optimizes outcomes. Understanding these elements allows for tailored treatment approaches, better resource management, and improved survival and quality of life across both patient populations. Currently, in the absence of large studies in patients with NCPH, current guidelines on vascular liver diseases suggest managing this complication according to the guidelines of cirrhotic portal hypertension [20]. Therefore, the finding of this study helps to develop early intervention, screening, and management guidelines for GEV among these patients. Furthermore, the study findings help the healthcare systems in resource planning, ensuring that endoscopic services are available to high risk patients for early variceal identification and prophylaxis.

2. Literature review

2.1 Gastroesophageal varices among cirrhotic with portal hypertension

The magnitude of the composite gastrointestinal incidences includes the magnitude of new varices, increasing in size, bleeding of the GEV, and death-related GEV differed across the studies. In one clinical review prevalence varies from around 30% in compensated to 60% in decompensated cirrhosis. The incidence rate of new varices is 9% per year and the progression rate from small to medium/large varices is 10% per year. The rate of bleeding from small esophageal varices is around 10% at 2 years while for medium/large varices it increases to 30% also depending on the Child-Pugh score. Without secondary prophylaxis, after an initial episode of bleeding, the 6-week risk of re-bleeding is 15–20% and increases to 60% within the first year. The 6-week mortality rate is 20–25%, mainly due to recurrence of bleeding (40%) and the development of liver complications. Gastric varices are less frequent (prevalence 10–20%); and bleed less frequently than esophageal varices (25% vs. 64%) but usually more severely with a 6-week mortality rate of 45%[21].

A study was conducted in Beijing among 206 cirrhotic portal hypertension patients on the incidence of GEV during the follow-up period. Notably, during the 3 years follow-up period, recurrence of esophageal varices occurred as high as 93.5% (143/153) patients, whereas a considerably low non-recurrence rate at 6.5% (10/153) was observed after endoscopic eradication esophageal varices. The most common etiology of cirrhosis in this study are Hepatitis B virus, alcoholic liver disease, and Hepatitis C virus in decreasing order. The high Child-Pugh score, large peri-ECVs, PFVs, and EVL, were independent risk factors identified to correlate with the recurrence of esophageal varices following the endoscopic treatment for esophageal varices eradication [22].

In a study conducted in Italy that evaluated the incidence and natural history of small esophageal varices in cirrhotic patients among 206 cirrhotic patients over 3-year periods, the rate of incidence of esophageal varices (EV) was 5% at 1 year and 28% at 3 years. The rate of EV progression was at 1 year and 31% at 3 years. Post-alcoholic origin of cirrhosis, Child-Pugh's class (B or C), and the finding of red wale marks at first examination were predictors for the variceal progression. The two-year risk of bleeding from EV was higher in patients with small varices upon enrolment than in those without varices 12% vs. 2% [23] In another similar study conducted in France but conducted to assess the incidence of large EV in 84 patients with

cirrhosis, 26% patients had large esophageal varices at one year and 48% at two years. The initial size of the esophageal varices a high initial Child-Pugh score and a smaller improvement in Child-Pugh score during the study were an independent risk factor [24].

A study was conducted to determine EV in 149 patients with liver cirrhosis attending a major tertiary hospital in Ghana. In this study, 12 (8.16%) patients were in class A, 64 (43.54%) in class B and 71 (48.3%) in class C at presentation. On UGIE, 135 (90.60%) had varices and fourteen patients (9.40%) had no varices. One hundred and eleven of the varices (82.22%) were large varices and the rest (17.78%) were small varices[25]

In Nigeria, 75 of thePatients had EV at endoscopy with 88.3% having grade 2 or 3 varices while 73.3% had moderate/large varices. Thirty-five percent of the varices had “red signs” with “red whale” markings as the predominant red sign. Gastric varices were seen in 12.5%. Advanced age, ascites, shrunken liver span, and low platelet count as independent predictors of EV [26].

In Ethiopia, studies were conducted to assess the magnitude of GEV among cirrhotic patients. In a study conducted in Northern Ethiopia, The prevalence of GEVH was found to be 52% among cirrhotic patients. The cause of CLD was not identified in more than 40% of patients. Higher grades of (F2 and F3), not taking beta blockers, duration of illness low platelet number (less than 50,000/µl) were significantly associated with increased risk of GEV [27].In another study conducted at **Saint paul Millenium Medical college Hospital in Addis Ababa**, 80% (n=50) of the patients have esophageal varices. The commonest etiology of cirrhosis is HBV infection (54.8%), followed by alcohol-related (14.5%), HCV (12.9%), drugs, and autoimmune causes (4.8%). All patients with Child class C stage liver disease have esophageal varices[11].

2.1 Gastroesophageal varices among non-cirrhotic with portal hypertension

In this retrospective observational study conducted in Singapore, they determined the magnitude of GEV among 75 patients who fulfilled the diagnostic criteria for NCPH. The most common etiologies were extra hepatic portal vein (PV) obstruction (EHPVO) (39%) and myeloproliferative disorders (MPD) (32%), while Idiopathic non-cirrhotic portal hypertension (INCPH) (5%) and Nodular regenerative hyperplasia (NRH) (7%) were uncommon. The most common presentation was upper gastrointestinal bleeding from the rupture of varices, which was observed in 32% of the cohort. Using Gastroscopy, Gastroesophageal varices were identified in 66%, with EV seen in 49% and gastric varices in 36% [28].

In Spain, a study was conducted to determine the magnitude and management of variceal bleeding among 178 patients with chronic non-cirrhotic, non-tumoral PVT. The usually recommended strategy for endoscopic screening and management of varices is the same as in cirrhosis. However, the efficacy of this policy in patients with PVT is unknown. The Median follow-up was 49 months. Variceal bleeding was the initial manifestation in 27 (15%) patients. Initial endoscopy in the remaining 151 patients showed no varices in 52 (34%), small EV in 28 (19%), large esophageal varices (LEVs) in 60 (40%), and gastric varices without LEVs in 11 (7%). In patients without varices, the probability of developing them was 2%, 22%, and 22% at 1, 3, and 5 years, respectively. In those with small EV, growth to LEVs was observed in 13%, 40%, and 54% at 1, 3, and 5 years, respectively. In patients with LEVs on primary prophylaxis, the probability of bleeding was 9%, 20%, and 32% at 1, 3, and 5 years, respectively [29].

In Italy, a study was conducted to describe the clinical presentation and the outcomes of 89 patients with non-cirrhotic PH with 77 patients with Child A cirrhosis. The prevalence of small (low risk) GEV was 41.6%(37/89) and large GEV 19.1%(17/89) among NCPH patients. In the comparative groups of patients with Child A cirrhosis, the prevalence of small GEV was 51.9%(40/77) and large GEV was 15.6%(12/77). The prevalence of varices and variceal bleeding was similar between the two groups at initial presentation. However during followup non-cirrhotic portal hypertension had higher rate of variceal progression and had more frequent bleeding than in patients with cirrhosis independently of the size of varices at the first endoscopy [30].

In Egypt, a hospital-based cross-sectional study was conducted at a tertiary hospital in Alexandria governorate. The most common causes were schistosomiasis periportal fibrosis (48%) followed by portal vein thrombosis (28%), Myeloproliferative diseases (12%), Budd-Chiari syndrome (8%), then Sarcoidosis (4%) [31]. In northern Ethiopia, a total of 55 patients with Hepatosplenic schistosomiasis (HSS) were evaluated. Most patients (70%) presented with upper gastrointestinal bleeding and severe anemia. Upper gastrointestinal endoscopy was performed in 31 patients (56%), in whom LEVs with red signs were the most common finding [32]

3. Objective

3.1General objective

To assess the incidences of EV composite outcomes among adults with cirrhotic and non-cirrhotic portal hypertensive patients in Tikur Anbessa hospital in Addis Ababa, Ethiopia (December 2021 – December 2024)

3.2 Specific objective

To determine the incidence of EV composite outcomes among adult cirrhotic patients with portal hypertension

To determine the incidence of EV composite outcomes among adult non-cirrhotic patients with portal hypertension

To compare the incidence of EV composite outcomes among cirrhotic and non-cirrhotic portal hypertensive adult patients

4. Methods

4.1 Study setting and period

The study was conducted at Tikur Anbessa Specialised Hospital (TASH), which is one of the largest hospitals in the country. Patients who followed from December 2021 to December 2024, were included in the analysis

4.2 Study design

This is an institution-based comparative retrospective longitudinal study design.

4.3. Population

4.3.1. Source population

All adult patients diagnosed with portal hypertension who had follow up at the selected hospital**4.3.2. Study population**

All adult patients with cirrhotic vs non-cirrhotic portal hypertensive patients who fulfill the inclusion criteria

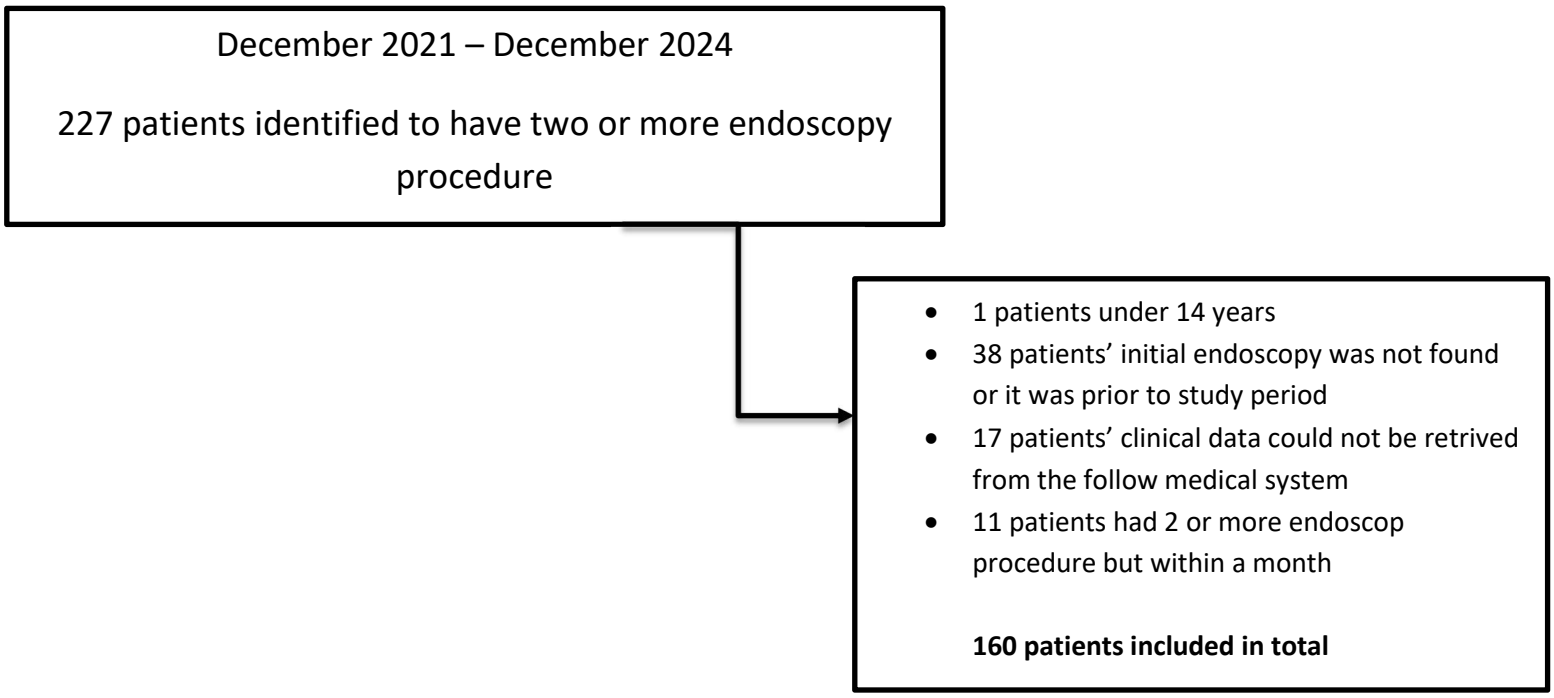
4.4 Sample size determinations, and sampling procedures

5.4.1 Sample size

The sample size will be calculated using the sample size determination using the following formula at a 95% confidence interval ($Za_{1/2}$=1.96), the margin of error(d)= 5%, the proportion of targeted esophageal varies among cirrhotic patients with portal hypertension (p= 80%) from a study conducted Saint Paul's Hospital Millennium Medical College [11].

$$\text{N} = \frac{p(1-p)(Za/2)^2}{(d)^2} = \frac{0.8(1-0.8)(1.96)^2}{(0.05)^2} = 246$$

The total sample size after 10% non-response (incomplete data) the final sample size will be 270. Due to the logistical challenges of managing data from a large patient population, a total of 160 patients were included in the study, with a 1:1 ratio between non-cirrhotic and cirrhotic portal hypertension patients.



4.5 Sampling techniques

All patients in the study period having met the inclusion critereas were included

4.6 Data collection tool and techniques

A structured data collecting sheet was be adopted from the previous study and comments of advisers was incorporated by reviewing the medical record. The variables of the questionnaires contained sociodemographic variables, clinical history, endoscopic feature vicarial bleeding (VB), oesophageal variceal and esophageal bleeding episodes. A general practitioner was assigned for the data collection. Episodes of GEV during the follow-up period was collected yearly. The principal investigator will be continuously supervising the data collectors. The data was collected in the Kobo data collection toolbox.

4.7 Eligibility criteria

4.7.1. Inclusion criteria

- Patients who had portal hypertension
- Patients should have endoscopic either for gastroesophageal varices screening or interventions;
- Adult age groups (Age >14 years).

4.7.2. Exclusion criteria

- Patients have no endoscopic evaluation for GEV at the study site
- Patients have no documented data on GEV;
- Had no follow-up for at least two years or referred to another center before two-year

4.8. Study variables

4.8.1 Dependent variable

- The composite of gastroesophageal varices related variables. These include **new gastroesophageal varices, progression from small, medium, or large, bleeding from EV, or death [33]. Deaths within 6 weeks after a bleeding episode were classified as bleeding-related deaths [34]**

4.8.2. Independent variables

- **Demographic variables:** Age, Gender, Residency, Comorbidity;
- **Lab findings at initial visits:** Platelet Count, International Normalized Ratio (INR), Bilirubin, Albumin, Creatinine and Hemoglobin, viral hepatitis, markers, HIV tests

  Cause of portal hypertension: Alcohol-related, viral cause, autoimmune cause, and other
- **Endoscopic finding:** Variceal Size and presence of Red Signs (Red Wale Markings);
- **Others:** Presences of ascites, spontaneous bacterial peritonitis, alcoholic history;
- **Use of Medications:** Beta Blockers, use of Anticoagulants or Antiplatelets

4.9. Data quality control

Pre-test was conducted among 10% of the total sample to check clarity of the tool and amend accordingly. The data collector was trained and the data collection process continuously monitored. The collected data was checked for completeness consistency and clarity daily .The primary investigator was supervising on sit.

4.9. Data entry and analysis

Data was be checked for its completeness exported, cleaned, and exported to be analyzed by SPSS version 25. The generated data was compiled by proportion, frequency tables, charts, and graphs. Descriptive statistics summarized baseline characteristics. To compare clinical features and composite outcomes between groups, chi-square tests were used for categorical variables,

while independent t-tests were used for continuous variables. A p-value < 0.05 was considered statistically significant.

4.10. Operational definition

Gastroesophageal Visceral bleeding: - refers to bleeding in the esophagus or stomach, especially in patients with portal hypertension, where dilated blood vessels/varices/ in the esophagus and or stomach rupture, leading to significant bleeding.

Cirrhotic portal hypertension: - a condition where increased portal vein pressure results from , significant fibrosis and scarring /liver cirrhosis/ . This is defined here by the abnormal liver synthetic tests with ultrasound and/or Fibroscan evidence of cirrhosis or advanced fibrosis respectively or histologic findings consistent liver cirrhosis on liver biopsy if done.

Non-cirrhotic portal hypertension: - refers to elevated blood pressure in the portal vein system that is due to the portal vein, hepatic veins, or liver sinusoids, often without significant liver scarring. This refers to causes of portal hypertension where cirrhosis is excluded based on the aforementioned criteria.

Portal hypertension: The presence of splenomegaly and esophageal varices /other systemic collaterals and /or findings on abdominal ultrasound with Doppler study suggesting portal hypertension such as Ascites, splenomegaly, nodular liver, portal flow mean velocity <12 cm/second, flow reversal on the portal vein, splenorenal collateral, portal vein diameter >13mm, no or decreased respiratory variation in splenic and superior mesenteric vein diameter

Outcomes of esophageal varices. range from potentially life-threatening complications due to acute bleeding to chronic issues that impact the quality of life. It also include progression from small to large varices , the presence of red signs(marking the thinnin of variceal wall and increasing the risk of bleeding) and 6 weeks mortality primary endpoint to assess the impact of therapies for acute VH

Outcome of endoscopic procedure for visceral bleeding:- The outcome of an endoscopic procedure for visceral bleeding includes stopping active bleeding , stabilising the patient and decreasing the burden of recurrent bleeding.Some patients may require admission to the critical care unit for airway protection, management of CV compromise, and in ideal setup treatments like as TIPS.

4.11. Ethical consideration

Ethical clearance and approval letters were be obtained from AAU, Internal Medicine Ethical Review Committees to conduct the research. A support letter was written from the department to the respective study area. After obtaining permission data was collected. Confidentiality was assured for each chart or medical records data reviewed. Specific patient identifiers were not included in the publication or presentation. The data collected was used only for the study and the ethical approval provided.

4.12. Dissemination of the result

The findings of the study will be presented and submitted to AAU, department of internal medicine and, efforts will be made to present it at scientific conferences. The paper will also be published in peer-reviewed open-access journals

5. Results

5.1 Clinical and Laboratory Characteristics

A total of 160 portal hypertensive patients were included, with **77**(48.1%, 95% CI: 40.4%-55.9%) non-cirrhotic and **83**(51.8%, 95% CI: 44.1%–59.6%) cirrhotic. The meidan age was 34 [IQR:27-45] and 96 (60%) were male. Non-cirrhotic patients were significantly younger, with 51.9% aged 16–30 years versus 20.5% of cirrhotic patients (p = 0.001).

A history of smoking was significantly associated with cirrhosis, with 53.8% of cirrhotic patients reporting smoking compared to only 15.6% of non-cirrhotic patients (p = 0.001). Similarly, alcohol use was markedly more common among cirrhotic patients (55.8%) than among non-cirrhotic patients (15.8%), a difference that was also statistically significant (p = 0.001). However, there were no significant differences between the two groups in terms of sex distribution (p = 0.301) or marital status.

Table 1 Demographic and Clinical Characteristics Comparison Between Cirrhotic and Non-Cirrhotic Portal Hypertension (PHT) Patients Status

Variables	Total (%)	Noncirrhotic PHT	Cirrhotic PHT	P-value
Sex				0.301
Female	64(40.0)	34(44.2)	30(36.1)	
Male	96(60.0)	43(55.8)	53(63.9)	
Age groups				
14-30	57(35.0)	40(51.9)	17(20.5)	0.001
31-45	64(40.0)	26(33.8)	38(45.8)	
46-60	30(18.8)	8(10.4)	22(26.5)	
>60	9(5.60)	3(3.9)	6(7.2)	
Adress				0.07
Addis Ababa	97(60.6)	41(53.2)	56(67.5)	
Other Regions	63(39.4)	36(46.8)	27(32.5)	
Smoking history (n=71)				0.001
Yes	26(37.1)	5(15.6)	21(53.8)	
Never	45(64.3)	27(84.4)	18(46.2)	
Alcoholic history (n=71)				0.001
Yes	30(42.3)	6(15.8)	24(55.8)	
No	51(71.8)	32(84.2)	19(44.2)	

5.2 Laboratory and Clinical Characteristics

Table 2, presents a comparative analysis of the clinical and laboratory features between cirrhotic and non-cirrhotic portal hypertension (PHT) patients. The presence of diabetes or hypertension was more common in cirrhotic patients (14.2%) than in non-cirrhotic patients (5.2%), with borderline statistical significance ($p = 0.05$).

Ascites and spontaneous bacterial peritonitis (SBP) were significantly more frequent among cirrhotic patients (90.4% and 36.1%, respectively) compared to non-cirrhotics (55.5% and 10.4%, respectively; $p = 0.001$ for both). Hepatitis B or C positivity was markedly higher in the cirrhotic group (41.0%) compared to the non-cirrhotic group (2.6%) ($p = 0.001$).

Cirrhotic patients had significantly higher mean AST, ALP, and bilirubin levels than non-cirrhotic, indicating more pronounced hepatic dysfunction. There were no significant differences in INR, albumin, renal function (creatinine), or complete blood count parameters (WBC, platelet count, hemoglobin) between the two groups. Specifically, in patients with portal vein thrombosis (PVT) associated with myeloproliferative neoplasms (MPN), the median platelet count was 435,000 (IQR, 41,500–570,000).

Table 2 Clinical and Laboratory Characteristics Comparison of Cirrhotic and Non-Cirrhotic Portal Hypertension (PHT) Patients.

Variables	Total (%)	Non cirrhotic PHT(N=77)	Cirrhotic PTH(N=83)	P-value
Comorbidities	29(18.1)			
Diabetes /hypertesion	16(10.0)	4.0(5.20)	12(14.20)	0.05
Renal disease	7.0(4.4)	4.0(5.20)	3.0(3.60)	0.62
Others	10(6.3)	5(6.5)	5(6.0)	0.92
Clinical Presentation				
Ascites	118(73.8)	43(55.8)	75(90.4)	0.001
SBP	38(23.8)	8(10.4)	30(36.1)	0.001
Drugs Patient taking				
Beta-blocker	158(98.8)	77(100)	81(97.6)	0.17
Anticoagulant	26(16.3)	14(18.2)	12(14.5)	0.53
Viral markers				
Hep B/C positive Both Hep B and C Postive	31(19.4) 3	2(2.6) 0	29(41.0) 3	0.001
Live enzymes and functions				
AST, mean(SD)(n=136)	48.37(52.3)	35.53(31.13)	60.83(65.3)	0.005
ALT, mean (SD)(n=136)	33.14(26.98)	29.14(14.89)	37.56(34.56)	0.06
ALP, mean(SD)(n=125)	125.4(88.28)	105.45(55.14)	144.45(108.17)	0.02
INR, mean(SD)(n=29)	1.43(0.39)	1.40(0.33)	1.45(0.46)	0.70
Billirubun,mean(SD)(n=78)	1.42(1.41)	1.10(0.81)	1.74(1.85)	0.04

Albumin, mean (SD)(n=26)	3.26(0.77)	3.27(0.88)	3.29(0.76)	0.94
Renal Function test				
Creatinine, mean(n=95)	0.76(0.28	0.76(0.34)	0.75(0.33)	0.94
Complete blood cells				
WBC,(SD)(*10^3)	5.54.14(5.02)	4.78(3.88)	5.96(5.84)	0.121
PLT, mean(SD),(*10^3)	119.88(119.2)	103.71(113.14)	133.86(104.28)	0.11
Hgb, mean,(SD)	10.77(3.89)	10.74(3.16)	10.70(3.56)	0.94

5.3 Etiology of the cirrhotic and noncirrhotic PHT

The bar graph depicts the distribution of portal hypertension etiologies, categorized into cirrhotic, non-cirrhotic, and other structural/secondary causes. Among the cirrhotic causes, Hepatitis B virus (HBV) emerged as the most common etiology, representing 38.6% of cases. In the non-cirrhotic category, schistosomiasis was the predominant cause, accounting for 67.5%**,** making it the leading cause of portal hypertension overall in this population.

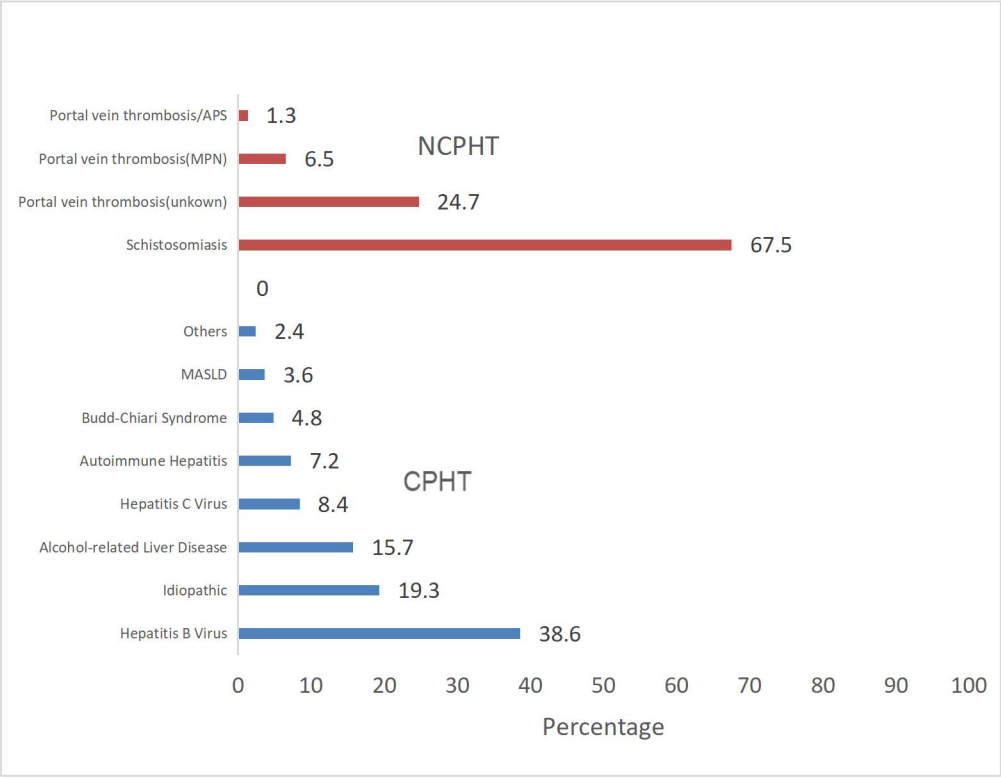


Figure 1:Etiology of Cirrhotic and Non-Cirrhotic Portal Hypertension (PHT) Patients.

5.3 Gastroesophageal varices and associated complications

At baseline, the majority of patients in both the non-cirrhotic portal hypertension (NCPHT) and cirrhotic portal hypertension (CPHT) groups presented with Grade III gastroesophageal varices (GEV) (87.0% vs 77.1%, respectively), while a smaller proportion had Grade I or II varices. Over time, there was a notable trend toward improvement in GEV grading in both groups. By the first year, most patients had shifted to Grade I (NCPHT: 46.8%, CPHT: 48.2%), and by the second year, this further increased to 73.3% in the NCPHT group and 77.4% in the CPHT group. However, the differences in GEV grading between the groups were not statistically significant at any time point (initial: $P = 0.26$; second year: $P = 0.33$; third year: $P = 0.84$).

Similarly, the prevalence of upper gastrointestinal (UGI) bleeding was high at the initial visit in both groups (72.7% in NCPHT vs 79.5% in CPHT), but declined progressively over time. By the first year, bleeding occurred in 64.9% of NCPHT and 56.6% of CPHT patients, and by the second year, it further decreased to 21.7% and 12.0%, respectively. Despite this overall reduction in bleeding events, there was no statistically significant difference between the groups at any time point ($P = 0.98$, 0.28, and 0.72, respectively).

Regarding the presence of red signs on endoscopy, the initial prevalence was similarly high in both groups (81.8% in NCPHT vs 81.9% in CPHT). This declined to 41.6% and 48.2% by the first year, and by the second year, only a few patients had red signs (5.0% in NCPHT vs 5.9% in CPHT). The differences in red sign prevalence between the groups were not statistically significant throughout the follow-up ($P = 0.33$, 0.39, and 0.83, respectively).

Table 3 Comparison of GEV Grading, UGI Bleeding, and Red Signs Between NCPHT and CPHT Patients at Baseline and After Two Years

Variables		Initial visit		P-value (x^2)	1st year		P-value (x^2)	2nd year		P value (x^2)
		NCPHT	CPHT		NCPHT	CPHT		NCPHT	CPHT	
GEV grading				0.26			0.33			0.84
	I	3(3.9)	6(7.2)		36(46.8)	40(48.2)		44(73.3)	41(77.4)	
	II	7(9.1)	13(15.7)		25(32.5)	19(22.9)		9(15.0)	6(11.3)	
	III	67(87.0)	64(77.1)		16(20.8)	24(28.9)		7(11.7)	6(11.3)	
UGI bleeding										
	Yes	56(72.7)	66(79.5)	0.98	50(64.9)	47(56.6)	0.28	13(21.7)	10(12.0)	0.72
	No	21(27.3)	17(20.5)		27(35.1)	36(43.4)		47(78.3)	43(51.8)	
Red signs										
	Yes	63(81.8)	68(81.9)	0.33	32(41.6)	40(48.2)	0.39	3(5.0)	3(5.9)	0.83
	No	14(18.2)	15(18.1)		45(58.4)	43(51.8)		57(95)	48(94.1)	

5.4 Composite outcome

A total of 160 patients were evaluated at the initial visit and followed for two years. By the second year, 113 patients remained under follow-up. The composite outcome, defined as any of progression in gastroesophageal varices (GEV), variceal bleeding, or mortality, was observed in 76.3% (n=122) at the initial visit, 61.3% (n=98) in the first year, and decreased to 8.8% (n=10) by the second year. Mortality was recorded only in the second year, with 3 deaths. Among 9 patients with low-risk (Grade I) GEV at baseline, 7 progressed to Grade II/III, and 6 experienced bleeding in the first year (77.8% composite outcome), with no further worsening in the second year. The deterioration occurred while being on treatment for underlying disease. Subsequtly 3 of the 9 patients had no sign of varices at the end of the second year but the rest were lost to followup

Initially, the composite outcome was higher in the CPHT group than in NCPHT (79.5% vs. 72.7%,p=0.31). By the first year, it was higher in NCPHT (66.5% vs. 56.6%, p=0.21), but by the second year, both groups showed similar improvement (6.0% vs. 6.5%, P=0.79). Mortality included 1 NCPHT and 2 CPHT cases. The site of death was one in hospital and others were out of hopsital (information retrieved via phone call). The presumed cause of death was attributed to portal hypertension related complication.

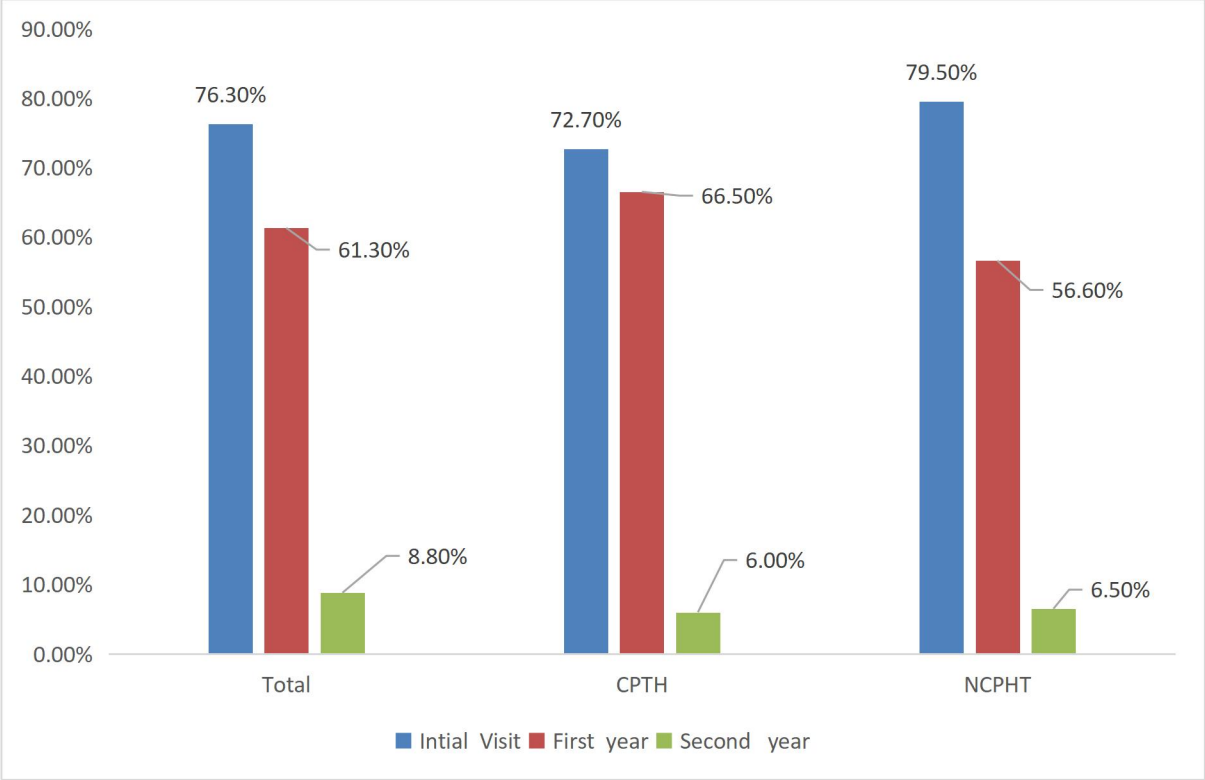


Figure 2 Overall Composite Outcome of Cirrhotic and Non-Cirrhotic Portal Hypertension (PHT) Patients.

6. Discussion

This study aimed to compare the incidence of composite GEV-related complications—including variceal grading, upper gastrointestinal bleeding, and 6-week mortality after bleeding—between non-cirrhotic and cirrhotic portal hypertension patients in tertiary hospitals in Addis Ababa, Ethiopia. The overall composite outcome was observed in 76.3% of patients at the initial visit, decreasing to 61.3% after one year and further to 8.8% by the end of the follow-up period. Comparison between CPHT and NCPHT groups showed no significant difference at baseline (79.5% vs 72.7%). Although both groups demonstrated a significant decline in complications over time—with rates reaching 6% and 6.5%, respectively, after two years -there was no statistically significant difference between the groups throughout the follow-up period.

The study observed a significant decline in the composite outcome (variceal progression, bleeding, and mortality), from 76.3% at baseline to 8.8% by the end of follow-up, demonstrating the effectiveness of standardized interventions such as endoscopic eradication, non-selective beta-blockers (NSBBs), and secondary prophylaxis in managing PH [21,22]. This notable improvement reflects the substantial impact of implementing evidence-based strategies in routine care, especially in resource-constrained settings where PH-related complications remain a major cause of morbidity and mortality. The **steepest reduction** was seen within the second year (61.3% to 8.8%) emphasizing the critical importance of early and aggressive intervention in altering the natural history of PH and preventing irreversible decompensation. This finding reinforces global evidence indicating that prompt initiation of endoscopic ligation and pharmacologic therapy can significantly reduce the risk of variceal bleeding and death, regardless of the underlying etiology [22–24]. Nevertheless, the residual risk of approximately 8% at follow-up highlights the persistent vulnerability of these patients and the importance of long-term monitoring. Even among those who receive optimal care, complications such as variceal recurrence, delayed bleeding, or late hepatic decompensation may still occur, necessitating continuous surveillance and individualized follow-up strategies [25,27]. These results suggest that while current therapies are highly effective in mitigating short- and medium-term risks, PH remains a chronic and evolving condition requiring sustained management. Most importantly successful treatment of underlying etiology is also essential. Management of underlying viral hepatitis , autoimmune hepatitis or schistosomiasis and recanalization of

thrombosis might have significantly contributed to the better control of the portal hypertension, reversing fibrosis or “recompensation”.

Notably, no significant differences were observed between cirrhotic (CPHT) and non-cirrhotic portal hypertension (NCPHT) groups at baseline (79.5% vs. 72.7%) or during follow-up (6% vs. 6.5% at 2 years). This finding challenges the traditional view that cirrhosis necessarily predicts worse outcomes and instead underscores that the presence of portal hypertension itself, rather than cirrhosis per se, is the primary determinant of complications when both groups receive standardized care [21,28,30]. The convergence in clinical outcomes likely reflects the shared pathophysiologic basis of elevated portal pressure and collateral formation, which are directly targeted by PH-directed therapies such as NSBBs and endoscopic interventions. For example, patients with NCPHT due to high-risk etiologies such as portal vein thrombosis or schistosomiasis, which are particularly prevalent in countries like Egypt [31] and Ethiopia [32], can experience variceal progression and bleeding risks comparable to those with cirrhosis, particularly when untreated or undertreated. Regional studies from China (Beijing) [22], Italy [23,30], and France [24] have similarly demonstrated that aggressive and protocolized management of portal hypertension, irrespective of etiology, can significantly reduce the incidence of life-threatening complications. For instance, studies from China have shown that endoscopic ligation is effective in reducing bleeding risk in patients with HBV-related cirrhosis [22], while trials conducted in Egypt and Ethiopia revealed comparable benefits in cases of schistosomiasis-associated PH [31,32]. These findings further support the concept that the pathophysiologic consequences of portal hypertension are amenable to intervention, even in the absence of underlying cirrhosis, provided that systematic and timely care is administered.

7. Strengths and Limitations

The study employs a comparative retrospective longitudinal design, allowing for the assessment outcomes over time, which enhances the validity of incidence comparisons between cirrhotic and NCPHT groups. It uses a well-defined composite outcome, which includes new or progressive gastroesophageal varices, bleeding episodes, and death, providing a comprehensive evaluation of clinically significant portal hypertension-related events. To ensure consistency, efficiency, and reliability in data collection, the study adopts a structured data collection sheet and uses Kobo Toolbox. Notably, this is the first study conducted in Ethiopia to assess portal hypertension

complications, offering a deeper understanding of disease progression and improving the generalizability of findings to similar populations.

The study has several limitations. First, its retrospective design may be subject to biases such as incomplete or inaccurate medical records, which can affect the accuracy of data collection. Second, the study is conducted at a single center in Ethiopia, which may limit the generalizability of the findings to other regions or countries with different healthcare systems and patient populations. Additionally, as the study focuses on a specific cohort of cirrhotic and non-cirrhotic portal hypertension patients, the results may not apply to patients with other forms of liver disease or portal hypertension. Furthermore, the study does not account for potential confounding variables such as variations in treatment regimens, socioeconomic factors, or lifestyle factors, which may influence the progression of portal hypertension and its complications. Lastly, the absence of prospective follow-up data could limit the ability to establish causality between certain interventions and outcomes over time.

8. Conclusion and recommendations

8.1 Conclusions

The study demonstrates that standardized interventions, such as endoscopic intervention for varices, use of appropriate NSBBs, and secondary prophylaxis, significantly reduce the composite outcome of variceal progression, bleeding, and mortality in patients with PH during the follow-up periods. Furthermore, no significant differences were found between cirrhotic and non-cirrhotic PH groups, suggesting that portal hypertension, rather than cirrhosis alone, is the primary driver of complications. The study's findings are consistent with global evidence showing that early and aggressive management of PH, regardless of its etiology, can significantly reduce bleeding risks and improve long-term outcomes.

1.2 Recommendations

First, early intervention strategies, including endoscopic ligation and pharmacotherapy with non-selective beta-blockers (NSBBs), are crucial in managing portal hypertension (PH) and preventing severe complications, particularly within the initial years after diagnosis. Second, despite significant reductions in risks, continuous surveillance and individualized follow-up care remain essential to monitor residual risks such as variceal recurrence and late hepatic decompensation, even after initial positive treatment responses. Third, the study suggests that cirrhosis may not always predict worse outcomes in patients with PH, highlighting the need for

further research into the role of PH itself in driving complications, particularly in non-cirrhotic patients with high-risk etiologies like portal vein thrombosis or schistosomiasis. Fourth, given the regional prevalence of such conditions, tailored interventions targeting these high-risk populations should be prioritized, with ongoing collaboration between healthcare systems to address unique challenges. Finally, continued multicenter and international studies focusing on PH management in both cirrhotic and non-cirrhotic patients are necessary to build a stronger evidence base and refine treatment protocols, ultimately leading to more nuanced guidelines for managing PH across diverse patient populations and regions.

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Annex

Questionnaire

This extraction sheet is designed to assess the incidences of GEV among patients with and without chaotic portal hypertension among adult medical patients who had follow-ups in selected hospitals in Addis Ababa, Ethiopia.

	Variables	Response	Skip
1. Sociodemographic variables			
1.	Age (year)	_____	
2.	Sex	1. Male 2. Female	
3.	Marital status	1. Single 2. Married 3. Widowed 4. Divorced	
4.	Residence	1. Addis Ababa 2. Out of Addis Ababa	
5.	Height (in cm)	_____	
6.	Wight (in kg)	_____	
7.	Smoking history	1. Current smoker 2. Previous smoker 3. Never smoker 4. Not documented	
8.	Alcoholic history	1. Current drinker 2. Previous drinker 3. Never drinker 4. Not documented	
III.	**Admission diagnosis and comorbidities**		
9.	Cause portal hypertesion	1. Non -cirrhotc portal hypertesion 2. Non-cirrhotic portal hypertesion	
10.	Specific etiologies for portal hypertension	1. Viral etiology 2. Alcoholic 3. Autoimmune 4. Infections such as schistosomiasis 5. Neoplasm (e,p MPN) 6. Vascular anomalies 7. Portal thrombosis 8. Idiopathic 9. Others specify _____	
11.	Comorbidities	1. Cardiac disease 2. Renal disease	

			3. Diabetes 4. Chronic lung disease 5. HIV and its complications 6. Others: specify ____	
12.	Alcoholic history		1. Current drinker 2. Former drinker 3. Never drinker 4. Not documented	
13.	Ascites		1. Yes 2. No	
14.	History SBP		1. Yes 2. No	
15.	Use of BB		1. Yes 2. No	
16.	Use of anticoagulant		1. Yes 2. No	
III.	**Laboratory investigations**			
13.	Live and renal function test		1. AST_____ 2. ALT:_____ 3. ALP:_____- 4. INR:_____ 5. Billirubin:_____ 6. Creatiniene :_____-	
14.	Initial Complete blood cell count		1. WBC:_____ 2. PLT :_____ 3. HGB:_____	
14.	Viral marker		1. HBV 2. HCV 3. HIV test	
IV.	**Gastroesophageal varices**			
17.	First year GEV(initia l visit)	Is there GEV	1. Yes 2. No	
		History UIB	1. Yes 2. No	
		Grade if GEV	1. Grade I 2. Grade II 3. Grade III	
		Red signs	1. Yes 2. No	
		Death with 6wk of GIB	1. Yes 2. No	
18.	2nd year	Is there GEV	1. Yes 2. No	
		History of bleeding	1. Yes 2. No	
		Grade if UGEV	1. Grade I 2. Grade II	

			3. Grade III	
		Red signs	1. Yes 2. No	
		If the pts had a history of GEV, change of size/grade	1. Yes 2. No	
		Death within 6wks of UGIB	1. Yes 2. No	
19	3rd year follow	Is there GEV	1. Yes 2. No	
		History of bleeding	1. Yes 2. No	
		Grade if GEV	1. Grade I 2. Grade II 3. Grade III	
		Red signs	1. Yes 2. No	
		If the pts had a history of GEV, change of size/grade	1. Yes 2. No	
		Death within 6wks of UGIB	1. Yes 2. No	