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Study Of Non-Invasive Predictor For Esophageal Varices In Chronic Liver Disease Patients

[1]Dr. Hemant Kumar Mahur, [2]Dr. Ranjeet Morya, [3]Dr. Roshan Singhadia, [4]Dr. Vivek Kumar Yadav, [5]Dr. Oshin Raj Kumawat

[1]Senior Professor, [2]Senior Resident , [3,4,5]Junior Resident,
Department of General Medicine,
RNT Medical College and Hospital, Udaipur , Rajasthan

***Corresponding Author:**
Dr. Ranjeet Morya
Senior Resident, General Medicine, RVRS Medical College , Bhilwara, Rajasthan

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Abstract

Background: Chronic liver disease (CLD) is a significant global health burden, leading to fibrosis, cirrhosis, and complications such as portal hypertension and esophageal varices (EV). Esophagogastroduodenoscopy (EGD) remains the gold standard for EV diagnosis but is invasive and costly. This study evaluates non-invasive predictors for EV, aiming to reduce unnecessary endoscopies.

Methods: A total of 120 CLD patients from RNT Medical College, Udaipur, were analyzed. Patients were categorized into two groups: Group A (CLD with EV) and Group B (CLD without EV). Clinical, biochemical, and imaging parameters, including Aspartate Aminotransferase to Platelet Ratio Index (APRI) and Platelet Count/Spleen Diameter (PC/SD) ratio, were assessed. Statistical analyses included chi-square, t-tests, and receiver operating characteristic (ROC) curves.

Results: The mean age was 43.91±8.20 years, with males comprising 75.83%. Alcohol-related liver disease was the most common etiology (49.17%). EV prevalence was 71.66%. Patients with EV had significantly lower platelet counts and PC/SD ratios but higher AST levels, spleen diameters, and APRI scores ($p<0.01$). ROC analysis showed the highest diagnostic accuracy for PC/SD ratio (AUC 0.96, sensitivity 90.7%, specificity 97.06%) and APRI (AUC 0.95, sensitivity 89.5%, specificity 88.4%).

Conclusion: EV is common in CLD, with non-invasive markers such as APRI and PC/SD ratio demonstrating high diagnostic accuracy. These parameters can aid in risk stratification, reducing the need for endoscopy and improving patient outcomes. Implementing non-invasive screening can enhance CLD management and prevent life-threatening complications.

Keywords: Chronic liver disease, esophageal varices, portal hypertension, APRI, platelet count/spleen diameter ratio, non-invasive markers, endoscopy, cirrhosis, fibrosis, risk stratification.

Introduction

Chronic liver disease (CLD) is a major global health concern, leading to progressive liver damage, fibrosis, and cirrhosis. Underdeveloped countries are disproportionately affected due to unawareness, lack of facilities, and poverty. CLD progresses slowly, resulting in complications such as portal hypertension, ascites, hepatorenal syndrome, hepatic encephalopathy, and spontaneous bacterial peritonitis.[1]

Portal hypertension, defined as a hepatic venous pressure gradient (HVPG) of ≥5 mmHg, occurs due to obstruction at different levels of the portal venous system. When HVPG exceeds 12 mmHg, acute

variceal bleeding becomes a major concern, significantly increasing mortality above 20 mmHg.[2] Approximately 50% of portal hypertension patients develop esophageal varices (EV), with a high risk of bleeding linked to variceal size. The risk of rebleeding after an initial variceal hemorrhage is 60-70% within 24 months, with the highest risk occurring shortly after the first bleed.[3]

The American Association for the Study of Liver Disease and the Baveno IV Consensus recommend screening all cirrhotic patients for EV upon diagnosis. The prevalence of varices in cirrhotic patients is 60-80%, with a bleeding risk of 25-35%.[4] Despite hospital treatment, mortality from variceal bleeding remains approximately 20%.[5] Endoscopy is the gold standard for EV diagnosis but is invasive, costly, and associated with low patient compliance.

To address these challenges, non-invasive methods have been explored to predict EV, improving cost-benefit ratios and reducing unnecessary endoscopies. Ideal non-invasive predictors should have high negative (NPV) and positive predictive values (PPV).[6] Various parameters, including spleen diameter, platelet count, portal vein diameter, platelet count/spleen diameter ratio (PC/SD), Fibrosis-4 score (FIB-4), aspartate aminotransferase-to-platelet ratio index (APRI), and aspartate aminotransferase/alanine aminotransferase ratio (AAR), have shown promise.[7]

APRI is widely used to assess liver fibrosis severity, with scores ≥1.0 indicating significant liver damage. The PC/SD ratio is another crucial indicator, with lower values suggesting increased portal hypertension and a higher risk of EV. These non-invasive markers can effectively stratify patient risk and guide clinical decisions.[8]

Material And Methods

Data was collected from patients attending the outpatient and inpatient Department of Medicine, RNT Medical College, and associated hospitals in Udaipur, after obtaining informed written consent. Patients with chronic liver disease (CLD) with or without esophageal varices (EVs) meeting the following criteria:

Inclusion Criteria: 1. Written informed consent.

2. Adults >18 years of both sexes diagnosed with CLD of any etiology.

Exclusion Criteria: Key complications of liver disease include prior interventions for portal hypertension (such as portosystemic shunts), hepatocellular carcinoma, portal vein thrombosis, and intra-abdominal or extra-hepatic malignancies.

Study Procedure

Patients were categorized into two groups: Group A (CLD patients with EVs, with or without a history of bleeding) and Group B (CLD patients without EVs). All patients underwent history assessment, clinical examination, laboratory tests (CBC, liver function, HCV Ab, HBs Ag), abdominal ultrasound (spleen and portal vein diameter), and upper GI endoscopy (EVs presence, grading, and risk signs). Predictive markers analyzed included the APRI score and the platelet count/spleen diameter (PC/SD) ratio.

Statistical Analyses: Data were analyzed using SPSS v15, with $\chi 2$ tests for qualitative data and t-tests/F-tests for quantitative comparisons. A p-value <0.05 was considered significant, and ROC curves assessed diagnostic accuracy. Chronic liver disease (CLD) is a progressive condition leading to fibrosis and cirrhosis, caused by toxins, alcohol, infections, autoimmune disorders, or genetic/metabolic factors.

Results And Observation

Table 1: Distribution of cases according to etiology

Etiology	No. of Patients	Percentage
Alcoholic	59	49.17
Hepatitis B	20	16.67

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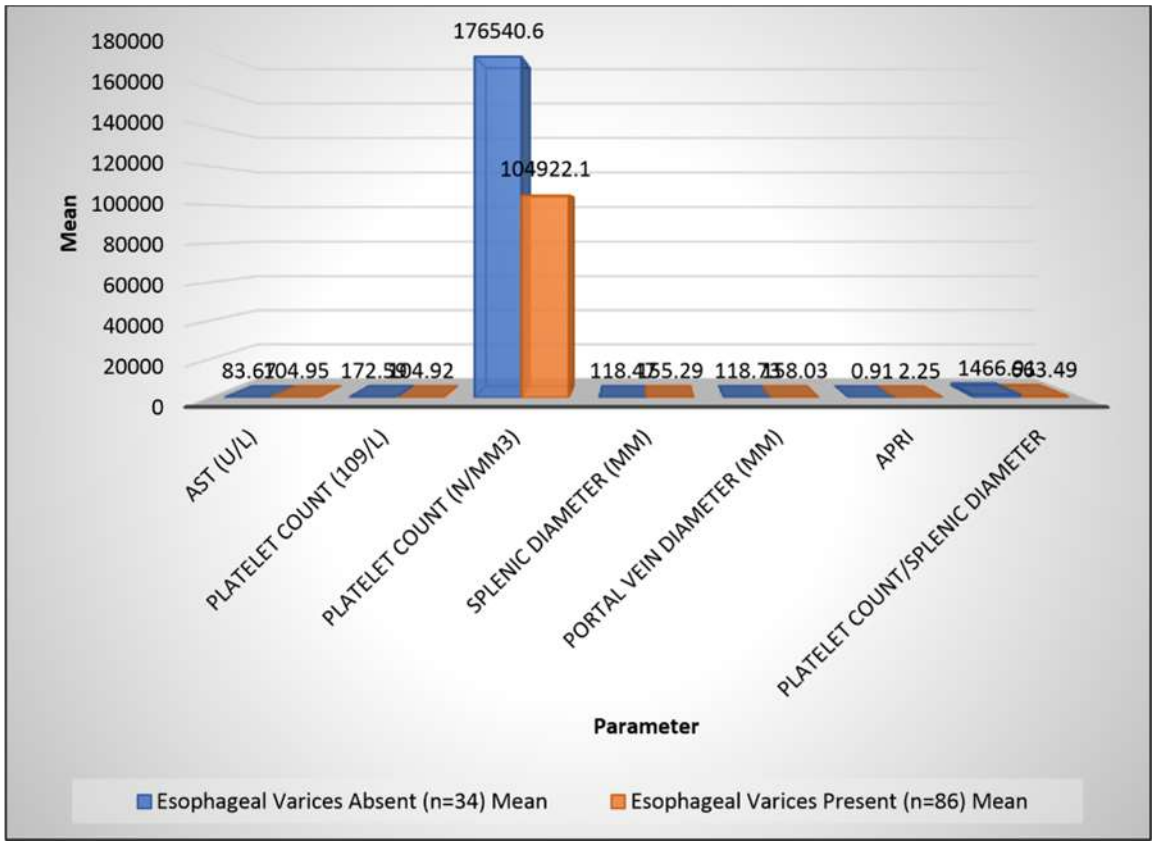
Hepatitis C	12	10.00
Auto Immune	4	3.33
Wilson Disease	3	2.50
Alcoholic + Hepatitis C	2	1.67
Hepatitis B + Alcoholic	1	0.83
Other	19	15.83
Total	**120**	**100.00**

The study of 120 patients found a mean age of 43.91±8.20 years, with most (55%) aged 41-55. Males comprised 75.83%. Esophageal varices were present in 71.66% (Group A), while 28.33% (Group B) had none. Alcohol-related liver disease was the leading cause (49.17%), followed by Hepatitis B (16.67%) and Hepatitis C (10%). Autoimmune, Wilson disease, and mixed etiologies were less common. Additionally, 15.83% had an unknown cause but were diagnosed with chronic liver disease. The findings highlight the predominance of alcohol-related liver disease and male patients, with varices affecting most cases.

Figure 1: Comparison of esophageal varices with Hepatological and Haematological Markers



The study analyzed 120 patients, with 67.5% having ascites, 69.17% melena, 26.67% hematemesis, and 25% pedal edema. The mean AST level was 98.92±43.24 U/L, and the mean platelet count was 124.09×10⁹/L. Splenic

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and portal vein diameters averaged 144.85±23.77 mm and 146.9±27.02 mm, respectively. The APRI score was 1.875±0.92, and the platelet count/splenic diameter ratio was 891.12±480.39. Patients with esophageal varices had higher AST levels, larger spleens and portal veins, and higher APRI scores ($p<0.01$), while their platelet counts and platelet count/splenic diameter ratios were significantly lower ($p<0.0001$). These findings suggest that esophageal varices are linked to more severe liver disease and could serve as key indicators for early diagnosis and risk assessment.

Table 2: Spearman Rho Analysis on varices with Hepatological and Haematological Markers.

Spearman Rho Analysis	Varices	
	r-value	p-value
AST (U/L)	0.27	<0.001
Platelet Count (10^9/L)	-0.58	<0.001
Platelet Count (N/mm^3)	-0.62	<0.001
Splenic Diameter (mm)	0.68	<0.001
Portal Vein Diameter (mm)	0.71	<0.001
APRI	0.71	<0.001
Platelet count/splenic diameter	-0.72	<0.001

The study of 120 patients found 67.5% had ascites, 69.17% melena, 26.67% hematemesis, and 25% pedal edema. Mean AST was 98.92±43.24 U/L, and mean platelet count was 124.09×10^9/L. Splenic and portal vein diameters averaged 144.85±23.77 mm and 146.9±27.02 mm, respectively. APRI score was 1.875±0.92, and the platelet count/splenic diameter ratio was 891.12±480.39. Platelet counts declined with increasing esophageal varices severity ($p<0.001$). Portal hypertension was linked to lower platelet counts. Spearman-Rho analysis showed AST and APRI positively correlated with varices, while platelet counts and platelet count/splenic diameter ratio had strong negative correlations ($p<0.001$). Splenic and portal vein diameters were also strongly associated with varices, highlighting their role in risk assessment.

Table 3: ROC analysis to predict Varice

ROC Analysis	AST	Platelet Count (10^9/ L)	Platelet Count (N/mm^3)	Splenic Diameter (mm)	Portal Vein Diameter (mm)	APRI	Platelet count/ splenic diameter
Area	0.675	0.876	0.898	0.93	0.957	0.957	0.96
Cutoff	85.5	137.25	134250	137	141	1.23	976.3
P-Value	0.003	0.0001	<0.0001	<0.0001	<0.001	<0.001	<0.001
Sensitivity	70.9	83.72	80.2	82.6	90.7	89.5	90.7

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Specificity	70.59	85.29	91.18	82.35	91.18	88.4	97.06
Accuracy	70.83	84.17	83.3	82.5	90.83	89.5	92.5

ROC analysis showed the highest diagnostic accuracy for the platelet count/splenic diameter ratio (AUC 0.96, sensitivity 90.7%, specificity 97.06%) at a 976.3 cutoff. APRI followed (AUC 0.95, sensitivity 89.5%, specificity 88.4%) at 1.23. Platelet count (AUC 0.89), portal vein diameter (AUC 0.957), and splenic diameter (AUC 0.93) also had strong diagnostic value. AST had the lowest AUC (0.675). All parameters were statistically significant ($p < 0.05$).

Discussion

Esophageal varices (EV) are a severe complication of chronic liver disease, with 20-40% experiencing variceal bleeding. While endoscopy is essential for diagnosis, its invasiveness and cost highlight the need for non-invasive predictors.[9]

In this study, alcohol was the most common etiology of chronic liver disease (49.17%), followed by Hepatitis B (16.67%) and Hepatitis C (10%). Other causes included autoimmune disease (3.33%), Wilson disease (2.50%), and mixed etiologies (2.50%). Additionally, 15.83% of cases had an unidentified etiology. These findings align with previous studies. **Cherian et al.**[10] reported alcohol as the leading cause (42.4%), followed by Hepatitis B (15.3%) and Hepatitis C (10%). **Sarangapani et al.**[11] similarly found alcohol to be the major contributor, affecting 62 cases, with Hepatitis B in 23 patients and splenomegaly in 42. **Duah A et al**.[12] noted alcohol was responsible for 32.89% of cirrhosis cases, rising to 39.61% with HBV/HCV.

This study found an average AST level of 98.92±43.24 U/L, platelet count of 124.09×10⁹/L (SD 63.09), splenic diameter of 144.85±23.77 mm, and portal vein diameter of 146.9±27.02 mm. The mean APRI score was 1.875±0.92, and the platelet count/splenic diameter (PC/SD) ratio averaged 891.12±480.39. Patients with esophageal varices had significantly higher AST levels, splenic and portal vein diameters, and APRI scores, while showing lower platelet counts and PC/SD ratios ($p<0.0001$).

Anbukumar T et al.[13] reported similar findings, with platelet counts decreasing and spleen diameters increasing as variceal grade progressed ($p<0.0001$). Their study also confirmed a significant decline in PC/SD ratios from Grade 1 to Grade 3 varices, reinforcing its predictive value for varices. **Cherian J V et al.**[10] found that patients with large varices (Grade III-IV) had lower platelet counts (78,000/µl vs. 104,500/µl; $p = 0.001$) and larger spleen diameters (180 mm vs. 155 mm; $p = 0.001$). Portal vein diameter was slightly larger (14 mm vs. 13 mm; $p = 0.005$), and the platelet count/spleen diameter ratio was lower (462.50 vs. 699.33; $p = 0.001$), highlighting its predictive value for variceal severity.

Similarly, Rapelly S S et al.[14] found that the APRI score was greater than 1.5 in 94.5% (n = 103) of patients, with the remaining 5.5% having scores between 0.5 and 1.5. The mean APRI score was 2.32 ± 0.41, with no patients having a score below 0.5. The analysis of variance test showed a p-value of 0.01, indicating statistical significance.

Conclusion

Chronic liver disease (CLD), primarily due to alcohol consumption, is common in males aged 41-55 and often leads to complications like ascites, melena, and esophageal varices. Many patients have grade 2 or 3 varices, with a high risk of life-threatening upper gastrointestinal (UGI) bleeding from variceal rupture. While endoscopy is the standard diagnostic tool, its invasiveness and cost limit patient compliance. This study highlights non-invasive predictors such as platelet count, spleen diameter, portal vein diameter, APRI score, and PC/SD ratio. A high APRI score and low PC/SD ratio strongly correlate with severe varices. Early detection using these markers can enable timely intervention, reducing complications and improving patient outcomes. Implementing non-invasive screening can enhance CLD management and prevent critical events.

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