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Comprehensive Clinical Assessment of Chronic Liver Disease Patients with Portal Hypertension

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ABSTRACT

Background: There are comparatively few research on chronic liver disease in India, despite the substantial burden of liver illnesses. This is a cross-sectional observational study conducted at a single centre. We looked at 100 chronic liver disease patients who were admitted to our ward or who came to the Medicine Emergency/OPD. The study sought to determine the severity of the disease (Child-Pugh classification), the laboratory parameters, and the clinical profile of patients with Chronic Liver Disease (CLD) who exhibit portal hypertension symptoms (in terms of clinical presentation). Finding out the number of cases of Portal Vein Thrombosis (PVT) linked to Chronic Liver Disease with Portal Hypertension was the secondary goal.

Methods: This was a cross-sectional, observational study done in the Department of Medicine at Lady Hardinge Medical College and its associated hospitals in New Delhi from November 2016 to March 2018. The study's inclusion criteria encompassed all patients with Chronic Liver Disease and Portal Hypertension, regardless of aetiology.

Results: In our study of 100 CLD cases, the mean age was 46.72 years, with 82 males and 18 females. Alcohol was the most common cause, especially in males. Hepatitis B (29%) was the second most common cause, particularly in females, while Hepatitis C was the third. Most cases had significant alcohol intake, and 92 had ascites. Hepatic encephalopathy (HE) was the primary cause of hospitalization, with PVT linked to severe HE.

Conclusion: The study has concluded that PVT in patients with chronic liver disease (CLD) is associated with more severe liver dysfunction, as indicated by higher levels of AST, ammonia, and aPTT, along with lower albumin levels.

Key-words: Chronic Liver Disease (CLD), Portal Hypertension, Portal Vein Thrombosis (PVT), Hepatic Encephalopathy (HE) and Child-Pugh Classification

INTRODUCTION

The liver plays a crucial role in metabolic balance, receiving 25% of cardiac output. Its dual blood supply from the hepatic artery (25-30%) and portal vein (70-75%) supports detoxification, protein synthesis, and metabolism [1-3].

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Blood from both sources mixes in hepatic sinusoids before systemic circulation. This vascular arrangement ensures efficient nutrient processing, detoxification, and immune function.

CLD involves progressive liver damage, leading to cirrhosis, marked by fibrosis and regenerative nodules [2,4]. Cirrhosis disrupts liver function, causing complications like portal hypertension, ascites, gastrointestinal bleeding, splenomegaly, and hepatic encephalopathy [5,6]. It is a major global health issue, ranking as the fourth leading cause of death in central Europe and responsible for over a million deaths annually [7]. Common causes include hepatitis C, alcohol

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misuse, and non-alcoholic fatty liver disease (NAFLD) in developed nations, while hepatitis B dominates in Asia and Africa [8].

The progression of CLD varies by etiology and patient factors. Alcohol-related liver disease results from chronic alcohol consumption leading to hepatocyte damage, while viral hepatitis-induced CLD is driven by chronic infection and immune-mediated injury. NAFLD is increasingly recognized as a major contributor, especially in individuals with obesity and metabolic syndrome. Genetic predisposition, environmental exposure, and lifestyle factors all influence disease progression and severity.

Cirrhosis progresses silently, with symptoms appearing late. Early signs include jaundice, fatigue, pruritus, nausea, and abdominal distension [7]. Portal hypertension results in severe consequences such as variceal haemorrhage, ascites, and hepatic encephalopathy [8]. Fibrosis results from hepatic stellate cell activation, reducing liver function [11,12]. Progressive fibrosis leads to increased intrahepatic vascular resistance, impairing blood flow and further exacerbating portal hypertension. This cascade of events ultimately results in the development of severe complications requiring urgent medical intervention.

Decompensated cirrhosis manifests with ascites, spontaneous bacterial peritonitis (SBP), and hepatorenal syndrome (HRS) [8,9]. Hepatic encephalopathy results from ammonia buildup, leading to cognitive dysfunction [10]. Jaundice indicates bilirubin accumulation, signaling liver failure. The systemic effects of cirrhosis extend beyond the liver, impacting renal function, coagulation pathways, and cardiovascular health. The hyperdynamic circulation seen in cirrhotic patients contributes to further disease complications.

With the rising prevalence of CLD, early screening and management are crucial. Lifestyle changes, antiviral therapy, and interventions for alcohol-related liver disease can slow progression. Advances in antifibrotic therapy offer hope in targeting fibrosis pathways. Emerging treatments, such as hepatic regenerative therapies, hold promise in reversing fibrosis and restoring liver function. Timely diagnosis and intervention improve survival and reduce complications [12].

Future research must focus on refining non-invasive diagnostic tools, identifying novel therapeutic targets, and optimizing management strategies for CLD. As the burden of liver disease continues to grow, multidisciplinary approaches involving hepatologists, nutritionists, and public health specialists will be key in improving patient outcomes.

MATERIALS AND METHODS

Research Design- This study was a cross-sectional, observational research conducted at the Department of Medicine, Lady Hardinge Medical College and Associated Hospitals, New Delhi. It aimed to analyze the clinical profile of patients diagnosed with CLD and portal hypertension. The study focused on the clinical presentation, and disease severity using the Child-Pugh classification, and laboratory parameters in these patients. A minimum of 100 patients, who met the inclusion criteria, were enrolled and assessed for the presence of PVT using ultrasonographic color Doppler imaging.

Inclusion and Exclusion Criteria- The study included patients diagnosed with chronic liver disease and portal hypertension, irrespective of the aetiology. These patients were either referred to the outpatient department, emergency department, or hospitalised during the study period from November 2016 to March 2018.

The exclusion criteria were defined as patients with chronic liver disease who had concurrent malignancies other than hepatocellular carcinoma, those diagnosed with Budd-Chiari syndrome, individuals who had undergone recent abdominal surgery (within the last three months), or those with a history of abdominal trauma. These criteria ensured that only relevant cases of CLD with portal hypertension, without confounding factors, were included in the study.

Procedure- The study followed a systematic approach, starting with the enrolment of patients who met the inclusion criteria. Clinical assessments were performed, including detailed history taking, physical examination, and laboratory tests such as complete blood count, liver function tests, renal function tests, serum electrolytes, viral markers, and others. Radiological assessments involved ultrasonography to measure portal vein diameter, portal vein flow, and splenic vein diameter, and color Doppler imaging was used to detect portal vein

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thrombosis. Upper gastrointestinal endoscopy was performed to identify any esophageal varices or other findings related to portal hypertension. Disease severity was determined using the Child-Pugh classification. The presence or absence of portal vein thrombosis was confirmed using USG Doppler.

Outcome Assessment- The primary outcome was the percentage of patients with chronic liver disease and portal hypertension who also had associated portal vein thrombosis, as diagnosed by ultrasonographic color Doppler. The secondary outcome assessed the differences in the clinical presentation between patients with portal vein thrombosis and those without it. This included comparing presenting complaints such as gastrointestinal bleeding, ascites, abdominal pain, jaundice, and hepatic encephalopathy. These outcome variables were analysed to identify any significant differences in the clinical and laboratory parameters between the two groups and to assess the impact of portal vein thrombosis on disease presentation and progression.

Statistical Analysis- The SPSS software package was used for statistical analyses. Categorical variables were expressed as frequencies (percentages). The continuous variable (age) was analysed using a normality test. All quantitative data were expressed as mean±standard deviation (SD).

RESULTS

Table 1 summarizes the laboratory parameters of the study population. The mean hemoglobin (Hb) level was 9.29 g/dL (SD=2.19), with a median of 9.1 g/dL, suggesting lower Hb levels were common. The total leukocyte count (TLC) had a mean of 11,764.1 cells/µL (SD=6,263.63), with a median of 10,150 cells/µL, reflecting variability in the immune response. Platelet counts averaged 1.97 lakh/µL (SD=1.05), highlighting thrombocytopenia.

Total bilirubin had a mean of 5.4 mg/dL (SD=5.7), with a median of 3.2 mg/dL. Direct bilirubin averaged 2.5 mg/dL (SD=3.1), while indirect bilirubin was 3.9 mg/dL. Elevated AST (94 IU/L) and ALT (58 IU/L) indicated liver damage, while ALP levels (124 IU/L) were relatively lower. Hypoalbuminemia was evident, with total protein at 6.1 g/dL, albumin at 2.64 g/dL, and globulin at 3.57 g/dL.

Kidney function markers, including urea (46.15 mg/dL) and creatinine (1.37 mg/dL), showed moderate elevation. Ammonia levels averaged 69.31 µmol/L, suggesting hepatic encephalopathy risk. Prothrombin time was prolonged (20.64 sec), with an INR of 1.89, indicating coagulopathy. Portal vein (14.77 mm diameter, 11.92 cm/s flow velocity) and splenic vein (10.87 mm diameter) measurements reflected mild portal hypertension.

Table 1: Summary of the laboratory parameters

Parameter	Mean	Standard deviation	Median	Mode
Hb(g/dL)	9.29	2.19	9.1	7.2
TLC (Numerical/µL)	11764.1	6263.63	10150	9200
Platelet counts (Number of cells in Lakh/µL)	1.97	1.05	1.70	1.80
Total bilirubin (mg/dL)	5.4	5.7	3.2	3.6
Direct Bilirubin (mg/dL)	2.5	3.1	1.5	1.2
Indirect Bilirubin (mg/dL)	3.9	9.5	1.9	2
AST(IU/L)	94	60	82	66
ALT(IU/L)	58	45	42.5	42
ALP (IU/L)	124	105	100	92

Total protein (g/dL)	6.1	0.94	6.2	6.2
Albumin(g/dL)	2.64	0.67	2.6	2.8
Globulin(g/dL)	3.57	0.87	3.4	3
Urea(mg/dL)	46.15	38.90	32.5	22
Creatinine(mg/dL)	1.37	0.93	1	0.9
Sodium(meql/L)	135.29	6.74	135	133
Potassium(meq/L)	3.933	0.68	3.8	3.6
Serum Ammonia (µmol/L)	69.31	18.30	68	92
PT (seconds)	20.64	7.18	18.6	16.2
aPPT(seconds)	49.12	10.51	47.8	48
INR	1.89	0.713	1.7	1.8
Portal vein diameter (mm)	14.77	1.85	15	14
Portal Vein Flow Velocity (cm/s)	11.92	3.23	12	12
Splenic Vein Diameter (mm)	10.87	1.49	11	11

Table 2 compares patients with PVT to those without it. No significant differences were found in age (52 vs 46 years, p=0.10) or gender distribution (p=0.094). Hemoglobin was lower in PVT patients (8.0 g/dL vs 9.4 g/dL, p=0.063), indicating anemia. Platelet counts were also lower (1.76 lakh/µL vs 2.00 lakh/µL, p=0.47), suggesting thrombocytopenia. Blood urea (59.18 mg/dL vs 44.53 mg/dL, p=0.24) and serum creatinine (1.60 mg/dL vs 1.34 mg/dL, p=0.38) were slightly elevated in PVT cases. AST was significantly higher in the PVT group (162.09 IU/L vs 86.11 IU/L, p=0.010), indicating greater liver injury. ALT was also elevated (95.72 IU/L vs 54.1 IU/L, p=0.20), though not statistically significant. Albumin was lower (2.21 g/dL vs 2.7 g/dL, p=0.015), suggesting more severe liver dysfunction.

Prothrombin time was prolonged (23.37 sec vs 20.31 sec, p=0.07), and aPTT was significantly longer (58.85 sec vs 47.92 sec, p=0.0009), reflecting coagulopathy. Ammonia levels were higher (82.27 µmol/L vs 67.7 µmol/L, p=0.006), indicating a greater risk of hepatic encephalopathy in PVT patients.

Table 2: Comparison of PVT and Non-PVT Cases

Parameter	Cases with PVT	Cases without PVT	p-value
Mean Age (years)	52	46	0.10
Gender (frequency) Male Female	 7 4	 75 14	 0.094
Mean Hb (g/dL)	8.0	9.4	0.063
Mean TLC (Number of cells/µL)	12936.36	11619.21	0.41
Mean Platelet counts (Number of cells (in Lakh)/µL)	1.76	2.00	0.47
Mean Bloodure a(mg/dL)	59.18	44.53	0.24
Mean Serum creatin in (mg/dL)	1.60	1.34	0.38

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Mean Total bilirubin(mg/dL)	6.127	5.32	0.67
Mean AST(IU/L)	162.09	86.11	0.01
Mean ALT(IU/L)	95.72	54.1	0.20
Mean ALP(IU/L)	107.18	126.2	0.94
Mean Albumin (g/dL)	2.21	2.7	0.015
Mean Globulin (g/dL)	3.46	3.58	0.66
Mean PT (seconds)	23.37	20.31	0.07
Mean a PTT (seconds)	58.85	47.92	0.0009
Mean INR	2.22	1.85	0.10
Mean S.NH3(µmol/L)	82.27	67.7	0.006

Table 3 compares the clinical presentations of CLD patients with and without PVT. Among the clinical manifestations, gastrointestinal bleeding (UGI bleeding) showed a statistically significant difference, with more cases in the PVT group (9 cases vs 22 cases, p=0.001). This finding suggests that PVT may be associated with a higher likelihood of variceal bleeding due to the increased portal pressure. Ascites, a common complication of portal hypertension, did not show a significant difference between the two groups (p=0.30), indicating that ascites may not be strongly associated with the presence of PVT in this cohort. Hepatorenal syndrome (HRS) was more common in the PVT group (5 cases vs 21 cases), though this difference did not reach statistical significance (p=0.121), suggesting that renal dysfunction may not be a direct consequence of PVT in this study. Spontaneous bacterial peritonitis (SBP) also showed no significant difference (p=0.66), indicating that PVT does not seem to increase the incidence of infections in the abdominal cavity.

The presence of hepatic encephalopathy (HE) was notably more frequent in patients with PVT, with more severe grades observed. The PVT group had a higher number of patients with HE grades III (3 cases vs 24 cases) and HE grade IV (6 cases vs 9 cases), while the non-PVT group had more cases of HE grade I (23 cases) and grade II (30 cases). This suggests that PVT is associated with more severe hepatic encephalopathy in patients with chronic liver disease. Esophageal varices of higher grades (III) were more common in the PVT group (9 cases vs 37 cases), though the comparison of variceal grades did not reach statistical significance in this study.

Table 3: Table shows a different clinical presentation in cases of CLD with and without PVT

Clinical Presentation	Number of Cases with PVT	Number of cases Without PVT	p-value
Ascites	11	81	0.30
UGI bleed	9	22	0.001
HRS	5	21	0.12
SBP	2	14	0.66
HEgrade0	0	3	-
HE I	1	23	-
HE II	1	30	-
HE III	3	24	-
HEIV	6	9	-
Esophageal varices Grade I	0	13	-
Grade II	2	39	-
Grade III	9	37	-

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DISCUSSION

The study aimed to analyze the clinical profile of CLD patients with Portal Hypertension (PH) and the prevalence of PVT [13]. Among 100 patients, the mean age was 46.72 years, with a male predominance (82%). Alcohol was the leading cause of CLD (71%), followed by Hepatitis B (29%) and Hepatitis C (26%) [14-17]. The coexistence of alcohol use and viral hepatitis was observed in multiple cases, indicating a compounded risk factor for disease progression.

Hepatic encephalopathy (HE) was the most frequent complication (97%), with PVT patients experiencing more severe grades [12,13]. Ascites was present in 92% of cases, reinforcing its role as a hallmark of advanced liver disease. Upper gastrointestinal bleeding (UGIB) was noted in 31% of patients, significantly higher among those with PVT (81.82%), suggesting a strong correlation between PVT and variceal hemorrhage [15,16]. Hepatorenal syndrome (HRS) and spontaneous bacterial peritonitis (SBP) were observed in 26% and 16% of cases, respectively, consistent with global trends [9].

Comparative analysis with previous studies highlighted regional variations in CLD etiology and outcomes. While alcohol was the dominant cause in our cohort, Hepatitis B was more prevalent in females, aligning with studies indicating gender-based differences in disease distribution [9,10]. PVT prevalence (11%) was lower than in some international studies but consistent with others, emphasizing the need for further investigation into genetic and environmental influences [13-17].

Our study underscores the impact of PVT on CLD progression, with affected patients exhibiting higher rates of hepatic decompensation. Table 4 provides a comparative analysis of key clinical parameters between PVT and non-PVT patients, demonstrating significant differences in liver function markers. Patients with PVT showed more severe liver dysfunction, characterized by higher levels of AST, ammonia, and aPTT, along with lower albumin levels, reinforcing the association between PVT and liver damage. This finding aligns with previous research suggesting that PVT exacerbates portal hypertension and hepatic dysfunction.

Table 4: Comparison with previous studies: etiology and clinical profile

Parameters	Our study	Sharma *et al.* [9]	Mukherjee *et al.* [10]	Michitaka *et al.* [11]	Méndez-Sánchez *et al.* [12]
Number of cases	100	178	13014	33379	1486
Age (in years, Mean ±SD)	46.72±11.04	51.2±8.9	42.8±14.4	-	-
Male (%)	82%	69.7%	73%	62.4%	48.93%
Female (%)	18%	30.3%	27%	37.6%	51.07%
Prevalence of Alcohol (%)	45%*	62.9%	34.3%	13.6%	39.5%
Prevalence of Hepatitis B (%)	29%	10.1%	33.3%	13.9%	5%
Prevalence of Hepatitis C (%)	26%	<27%	21.6%	60.9%	36.6%
Prevalence of diabetes mellitus	11%	-	11.7%	1.2%	-
Prevalence of Ascites (%)	92%	89.8%	-	-	-
Prevalence of HE (%)	97%	69.7%	-	-	-
Prevalence of SBP (%)	16%	24.7%	-	-	-
Prevalence of HRS (%)	26%	-	-	-	-
Prevalence of UGIB led (%)	31%	27%	-	-	-

**Seventy-one patients consumed alcohol, of which 16 additionally had concurrent Hepatitis B and 16 had concurrent Hepatitis C infections.*

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Hepatic encephalopathy was the most frequent complication in our cohort, with a notable severity in PVT patients. This may be due to worsened detoxification ability in the presence of thrombosis, leading to increased ammonia levels and subsequent neurological impairment. Similarly, UGIB was more prevalent in the PVT group, highlighting the increased risk of variceal rupture in these patients. These findings emphasize the importance of regular screening and early intervention in high-risk CLD patients.

Ascites was present in most patients, confirming its role as a major consequence of portal hypertension. Table 5 highlights the distribution of complications observed in PVT versus non-PVT patients, reinforcing the higher severity of disease in those with thrombosis. The presence of SBP, though less frequent, highlights the risk of bacterial infections in cirrhotic patients with ascites. HRS, observed in over a quarter of patients, indicates the systemic impact of CLD on renal function, further complicating disease management.

Table 5: Chronic liver disease accompanied by portal vein thrombosis: a comparative analysis with worldwide research

	Our Study	**Lankarani *et al.*** [13]	**Amitrano *et al.*** [14]	**Borjas-Almaguer *et al.*** [15]	**Zampino *et al.*** [16]
Total patients	100	219	701	169	130
Patients with PVT Male Female	11(11%) 7(63.64%) 4(36.36%)	35(15.9%) 26(74.28%) 9(25.71%)	79(11.2%) 47(59.4%) 32(40.6%)	13(7.6%) 8(61.5%) 5(38.5%)	19(14.16%) 8(42.1%) 11(57.89%)
Prevalence of Alcohol in Patients with PVT	5(45.45%)	<28%	11(13.8%)	6(46.2%)	1(5%)
Prevalence of Hepatitis Bin patients with PVT	4(36.36%)	11(31.4%)	9(11.3%)	Combined 1 (7.7%)	4(21%)
Prevalence of Hepatitis C in patients with PVT	2(18.18%)	-	36(45.5%)		7(36%)
Child Pugh class A	0	3(8%)	7(10%)	4(30.8%)	13(68.4%)
Child Pugh Class B	0	20(57.1%)	41(51.9%)	3(23%)	6(31.5%)
Child Pugh Class C	11(100%)	12(34.28%)	31(39.1%)	6(46.2%)	0
UGI bleed	9(81.82%)	-	-	8(61.5%)	-
SBP	2(18.18%)	-	-	2(15.4%)	-
HE	11(100%)	-	-	4(30.8%)	-

All data has been presented as numerical values (percentages).

Our findings contribute to the growing body of knowledge on CLD and PVT in North India. While consistent with global research, the study reveals regional differences in disease patterns and outcomes. However, limitations such as a small sample size, single-center design, and absence of long-term follow-up warrant further multicenter studies to validate these findings and explore targeted interventions for improved patient outcomes [17].

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CONCLUSIONS

The study concluded that portal vein thrombosis in chronic liver disease is linked to severe liver dysfunction, evidenced by higher AST, ammonia, and a PTT, along with lower albumin levels. PVT contributes to liver injury, coagulopathy, and hepatic encephalopathy. Hepatic encephalopathy was the predominant manifestation, succeeded by ascites. The majority of patients arrived late in a decompensated condition, with more than 90% classified as Child-Pugh class B or C. Alcohol was the predominant cause of chronic liver disease in males, whereas hepatitis B was the most prevalent in females. Overall, alcohol was the primary etiology, followed by hepatitis B and C. Diabetes Mellitus was associated with 11% of cases, with no hypertension cases reported. PVT affected CLD severity, with an incidence of 11%, most common in alcohol-related CLD. Most PVT cases had advanced CLD, reinforcing its link to disease severity.

CONTRIBUTION OF AUTHORS

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