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Clinical Profile of Patients of Chronic Liver Disease with Portal Hypertension

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Abstract

There are very few studies have been done in India to study the clinical profile of chronic liver disease patients. This is a single-center cross-sectional observational study. A total of 100 patients with chronic liver disease presented to the emergency, OPD, or admitted in the ward were studied. The study aimed to study the clinical profile of patients of chronic liver disease (CLD) who have features of portal hypertension, in terms of clinical presentation, the severity of disease (Child-Pugh classification), and laboratory parameters. The secondary objective was to find out that how many cases of chronic liver disease with portal hypertension have associated portal vein thrombosis presented to OPD or admitted to the wards of our hospital?

Materials and method: It was a cross-sectional observational study. The place of the study was the Department of Medicine, Lady Hardinge Medical College and Associated Hospitals, New Delhi, and the duration of the study was November 2016 to March 2018. Inclusion criteria were all the patients of chronic liver disease with portal hypertension irrespective of the cause.

Results and observations: In our study, the mean age of cases was 46.72 ± 11.04 years (expressed as Mean ± SD) and males (82) outnumbered females (18). The most common cause of chronic liver disease in our study population was alcohol. It was also the most common etiology of chronic liver disease among males and the 3rd most common etiology in females.

Conclusion: Overall, when all etiologies were taken into consideration, the most common attributable reason for CLD in our study was alcohol followed by Hepatitis B, followed by Hepatitis C. Among other causes, Diabetes Mellitus was associated with 11% of our study group. There were no patients with Hypertension. Portal vein thrombosis affects the clinical profile of CLD. In our study, the incidence of PVT was 11% and was most common in CLD attributed to alcohol. In our study, most of the patients with PVT indicating and reiterating the association of PVT and the Stage of CLD.

Keywords: Clinical presentation • Hypertension • Etiology • Chronic liver disease

Introduction

The liver is a very vascular organ and at rest receives up to 25% of total cardiac output, more than any other organ. Its dual blood supply is uniquely divided between the hepatic artery, which contributes 25%to 30% of the blood supply, and the portal vein, which is responsible for the remaining 70% to 75%. The arterial and portal blood ultimately mixes within the hepatic sinusoids before draining into the systemic circulation via the hepatic venous system [1]. Chronic liver disease is a clinical manifestation of altered liver structure and function and may present clinically as signs and symptoms of portal hypertension like ascites, gastrointestinal bleeding, caput medusae, splenomegaly, or hepatic encephalopathy. Cirrhosis is defined as the histological development of regenerative nodules surrounded by fibrous bands in response to chronic liver injury, which leads to portal hypertension and end-stage liver disease [2]. 1-year mortality in cirrhosis varies widely, from 1% to 57%, depending on the occurrence of clinical decompensating events [3]. Cirrhosis is an increasing cause of morbidity and mortality in developed countries. It is the 14th most common cause of death in adults worldwide but the fourth in central Europe; and results in 1·03 million deaths per year worldwide [4]. The main causes in more developed countries are hepatitis C virus, alcohol misuse, and, increasingly, non-alcoholic liver disease. Infection with hepatitis B virus is the most common cause in sub-Saharan Africa and most parts of Asia [5]. Cirrhosis is often indolent, asymptomatic, and unsuspected until complications of liver disease are present.2 Typical presenting symptoms of liver disease include jaundice, fatigue, itching, right-upper-quadrant pain, nausea, poor appetite, abdominal distention, and intestinal bleeding [6]. The

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transition from chronic liver disease to cirrhosis involves inflammation, activation of hepatic stellate cells with ensuing fibrogenesis, angiogenesis, and parenchymal extinction lesions caused by vascular Occlusion [7]. Most of the chronic liver diseases are notoriously asymptomatic until cirrhosis with clinical decompensation occurs. Decompensating events include ascites, sepsis, variceal bleeding, encephalopathy, and non-obstructive jaundice [5].

Aim and objectives

To study the clinical profile of patients of chronic liver disease who have features of portal hypertension, in terms of clinical presentation, the severity of disease (Child-Pugh classification), and laboratory parameters.

Research questions

- What is the clinical profile of patients of Chronic liver disease with portal hypertension coming to OPD or admitted to the ward.
- How many cases of chronic liver disease with portal hypertension have associated portal vein thrombosis presented to OPD or admitted to the ward of our hospital.
- What is the clinical profile of patients of chronic liver disease with portal hypertension who are having associated portal vein thrombosis.

Primary outcome variable: The percentage of patients of chronic liver disease with portal hypertension included in the study as per inclusion criteria having associated portal vein thrombosis as diagnosed by USG color doppler.

Secondary outcome variable: Main presenting complaints of patients with portal vein thrombosis and those without portal vein thrombosis.

Materials and Method

Methodology

Type of study: Cross-sectional observational study.

Place of study: Department of Medicine, Lady Hardinge Medical College and Associated Hospitals, New Delhi.

Duration of study: November 2016 to March 2018

Inclusion criteria: All the patients of chronic liver disease with portal hypertension irrespective of the cause.

Exclusion criteria: Patients of chronic liver disease with associated malignancies other than Hepatocellular carcinoma, Budd-Chiari Syndrome, recent abdominal surgery (within 3 months), abdominal trauma.

Sample size: Minimum of 100 cases of chronic liver disease with portal hypertension were studied for clinical profile and the presence of portal vein thrombosis.

Case definition: Patients with clinical features of portal hypertension (ascites, caput medusae, Gastro-intestinal bleed) or upper GI endoscopic findings of portal hypertension disease (liver coarse echotexture on Ultrasonography).

Minimum 100 patients of chronic liver disease coming to OPD or admitted to the wards will be studied and USG doppler will be used to study the presence or absence of portal vein thrombosis in those patients. HBV cirrhosis will be defined by a positive diagnosis of HBV-related cirrhosis and the presence of the HBV surface antigen (HBsAg) in the absence of a history of alcohol consumption or other co-existing viral infections.

Decompensated liver cirrhosis will be diagnosed by clinical findings or morphological features and its severity will be scored according to the Child-Pugh classification.

Significant alcohol intake: Significant alcohol intake was taken as the alcohol intake more than the low-risk drinking level as defined by the Alcohol Web India National Policy.8

Assessment parameters:

- Clinical presentation (presence and absence of GI Bleed, ascites, fever, hepatic encephalopathy, abdominal pain, abdominal distension, jaundice).
- Laboratory parameters- Complete blood count, Liver function test, renal function test, albumin, PT, APPT, INR, serum electrolytes, S. NH3 level, viral marker, D-dimer.
- Radiological parameter- Portal vein diameter, portal vein flow, splenic vein diameter, site of thrombosis.
- Upper GI endoscopy
- Child-Pugh classification

Statistical analysis

The SPSS software package will be used for all statistical analyses. Categorical variables will be shown in terms of frequencies (percentages). The continuous variable (age) will be examined by a normality test. All quantitative data will be expressed as mean ± standard deviation (SD).

Observation and Results

Our study was conducted in the Department of Medicine, Lady Hardinge Medical College and Associated Hospitals. It was a hospital-based observational study.

Out of 100 cases, 71 had a history of significant alcohol intake. Out of these 71 cases, 16 cases had a history of concomitant Hepatitis B and 16 cases had a co-infection with Hepatitis C. In our study, Hepatitis B was the second most common etiology of chronic liver disease (29%). 16 cases also had a history of significant alcohol intake. Hepatitis B was the most common cause of CLD in females. Hepatitis C was the third most common cause of CLD in our study. A total of 26 cases were positive for Hepatitis C of which 4 were females and 22 were males. Out of these 26 cases, 16 cases also had a history of significant alcohol consumption. In our study, 11 patients had diabetes mellitus. Out of which 7 were males and 4 were females. Of this, 3 patients also had a history of significant alcohol intake, and Hepatitis B and Hepatitis C infection were present in 3 and 1 cases, respectively. The etiology of CLD could not be ascertained in 2 cases as they were non-alcoholic, non-diabetic, and

tested negative for Hepatitis B and Hepatitis C infections. They were classified as having an unknown etiology of CLD. In our study, only 7 cases belonged to Child-Pugh (CP) Class A, while 27 and 66 cases belonged to CP classes B and C respectively. Hepatic encephalopathy (HE) was the most common cause of admission to the hospital. HE was present in 97% of the cases. Only 3 cases did not have HE. Patients with portal vein thrombosis (PVT) often had a more severe grade of HE than those who did not have associated PVT. 6 out of 11 patients of PVT had HE grade IV. Ascites was the second most common cause of admission to the hospital. 92 cases had ascites. Only 8 cases did not have ascites, of which 4 cases belonged to Child-Pugh class A and 4 to class B. In our study, upper gastrointestinal bleeding was the third most common cause of presentation to the hospital. In our study 31% of the cases presented with upper gastrointestinal (UGI) bleed, although the number of males presenting with UGI bleed was more than the number of females (28 and 3 in males and females respectively), the difference was not statistically significant (p=0.24). Out of a total of 11 cases of CLD with associated PVT, UGI bleed was present in 9(81.82%) of the cases, and the difference between those cases with associated PVT but without UGI bleed was statistically significant (p=0.001). Hepatorenal syndrome (HRS) was present in 26 patients, of which 21 were male (25.61% of the total number of males) and 5 were females (27.78% of the total number of females). Out of 11 cases of PVT, 5 patients had HRS but there was no statistically significant difference between those who did not have associated PVT (p=0.14). Spontaneous bacterial peritonitis (SBP) was present in 16 cases of which 11 were males (13.41% of the total number of males) and 5 were females (27.77% of the total number of females). Most of the cases diagnosed with SBP belonged to Child-Pugh Class C (16.67%) but the number was not statistically significantly different from those in Class A and B (p=0.56). SBP was present in 2 patients of CLD with associated PVT.

100 patients meeting the inclusion criteria were enrolled in the study and evaluated by taking an exhaustive history, general physical examination, systemic examination, and routine investigations, along with special investigations like serum ammonia, PT/INR, a PTT, and d- dimer. The results of upper gastrointestinal endoscopy (UGIE), ultrasound abdomen (USG), and spleno-portal-axis (SPA) doppler, which the patients were undergoing as a part of the standard of care were recorded. Appropriate statistical tools were employed to evaluate the data generated, the results of which have been explained here.

Demographic profile of patients

Age distribution: Patients from age 20 to 80 years were included in the study. The mean age was 46.72 years with a SD of 11.04 years and the median age was 46 years. The mean age of males and females (expressed as mean ± SD in year) were 44.84 ± 10.86 and 55.27 ± 7.28, and the age difference was statistically significant (p=0.0002).

Gender: Out of the 100 patients included in the study, 18 were female and 82 were male.

Distribution of classic risk factors for development of chronic liver disease

Alcohol: Out of 100 patients, 71 had a history of significant alcohol intake. Out of 82 males, 70(85.37%) and out of 18 females, 1(5.56%) had a history of alcohol intake.

Hepatitis B: A total OF 29 patients were found to be having hepatitis B infection. Of these 10 were female (55.56% of total females) and 19 were male (23.17% of total male). Out of 29, 16 patients also had a history of significant alcohol intake. Of this 1, 8, and 20 cases belonged to Child-Pugh Class A, B and C respectively.

Hepatitis C: 26 patients were found to be having hepatitis C infection, out of which 4 were female (22.22% of total females) and 22 were male (26.83% of total male). Out of these 26 patients, 16 patients also had a history of significant alcohol intake.

Diabetes mellitus: A total of 11 cases (11% of the total population) were diagnosed with Diabetes Mellitus, of which 4 were females and 7 were males.

Unknown causes: 2 patients were non-alcoholic, non-diabetic, and were also negative of HBsAg and Anti HCV antibody. They were classified as cirrhosis due to unknown etiology.

Clinical presentation

Ascites: Ascites was present in 92 patients. It was present only in 42.86% of the patients in Child-Pugh class A but was present in 100% of the patients in Child-Pugh class C.

Spontaneous bacterial peritonitis (SBP): SBP was present in 16 patients. Of these, 11 were males (13.41% of total male) and 5 (27.78% of total female) were females. The majority of the cases diagnosed with SBP were in Child-Pugh Class C (16.67%). SBP was present in 2 patients with PVT.

Hepatorenal syndrome (HRS): HRS was present in 26 patients. 21 were male (out of 82, 25.61%) and 5 were females (out of 18, 27.78%). In Child-Pugh class A, no patient had associated HRS, while in CP class C, 22 patients (33.33%) had associated hrshepatic **Encephalopathy (HE):** HE was present in 97 patients. HE was not present only in 3 patients. HE grade1 was present in 24 patients, while 31 patients had grade II HE and 27 and 15 patients were having HE grade III and IV respectively. In Child-Pugh class A, 3 patients had no detectable HE, while 2 patients each had HE grades 1 and 2. No patient had HE grade 3 and 4. In Child-Pugh class B, 6 patients had HE grade 1, 11 and 10 patients had HE grade 2 and 3 respectively. No patients in Child-Pugh Class B had HE grade 4 and 0. In Child-Pugh class C, 16 patients had HE grade 1, 18 patients had HE grade 2. HE grade3 and 4 were present in 17 and 15 patients, respectively.

Upper GI bleed: Upper GI bleed was present in 31% of patients. UGI Bleed was more common in patients who were having associated PVT. UGI bleed was present in 81.82% (9) of patients of PVT, while it was present only in 24.72% (22) of patients who were not having associated PVT (p=0.001). UGI bleed was present in 16.7% of females and 34.15% of males but the difference was not significant statistically.

Grade of esophageal varices: Grade 1 esophageal varices were present in 13 cases, while grade 2 and grade 3 esophageal varices were present in 41 and 46 patients, respectively.

Portal vein thrombosis: Portal vein thrombosis (PVT) was present in 11 patients. PVT was present in 7 males (out of 82, 8.54%), while it was present in 4 females (out of 18, 22.22%).

Use of β Blocker and PVT: The use of Non-selective β blocker (NSBB) was present in 81.8% of cases of PVT but it was present in only 32.5% of cases without PVT and the difference was statistically significant (p=0.004) (Table 1, Table 2).

STANDARD DEVIATION				
PARAMETER	**MEAN**	**STANARD DEVIATION**	**MEDIAN**	**MODE**
Hb (g/dL)	9.29	2.19	9.1	7.2
TLC (Number of cells/µL)	11764.1	6263.63	10150	9200
Platelet counts (Number of cells in Lakh/ µL)	1.97	1.05	1.7	1.8
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	38.9	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	18.3	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 1. Summary of the laboratory parameters.

PARAMETER	CASES WITH PVT	CASES WITHOUT PVT	P value
Mean Age (in years)	52	46	0.1
Gender (frequency) Male	7	75	0.094
Female	4	14	
Mean Hb (g/dL)	8	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	0.47
Mean Blood urea (mg/dL)	59.18	44.53	0.24
Mean Serum creatinin (mg/dL)	1.6	1.34	0.38
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	0.2
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 aPTT (seconds)	58.85	47.92	0.0009
Mean INR	2.22	1.85	0.1055
Mean S. NH3 (µmol/L)	82.27	67.7	0.006

Table 2. Pvt Vs Non-Pvt.

Clinical presentation: Table below shows different clinical presentation in cases of CLD with and without PVT (Table 3).

CLINICAL PRESENTATION	NUMBER OF CASES WITH PVT	NUMBER OF CASES WITHOUT PVT	P VALUE
Ascites	11	81	0.3
UGI bleed	9	22	0.001
HRS	5	21	0.121

SBP	2	14	0.66
HE grade 0	0	3	-
HE I	1	23	-
HE II	1	30	-
HE III	3	24	-
HE IV	6	9	-
Esophageal varices			
grade I	0	13	-
Grade II	2	39	-
Grade III	9	37	-

Table 3. Different clinical presentation in cases of CLD with and without PVT.

Discussion

The aim of our study was to see the clinical profile of patients of chronic liver disease and to study the prevalence as well as the clinical profile of those with associated portal vein thrombosis. We compared the results with similar studies done in India and as well as internationally. In our study, the mean age of cases was 46.72 ± 11.04 years (expressed as Mean ± SD) and males (82) outnumbered females (18). It may be attributed to the higher prevalence of alcohol intake in males as compared to females in India. The most common cause of chronic liver disease in our study population was alcohol. It was also the most common etiology of chronic liver disease among males and the 3rd most common etiology in females. Out of 100 cases, 71 had a history of significant alcohol intake. Out of these 71 cases, 16 cases had a history of concomitant Hepatitis B and 16 cases had a co-infection with Hepatitis C. In our study, Hepatitis B was the second most common etiology of chronic liver disease (29%). 16 cases also had a history of significant alcohol intake. Hepatitis B was the most common cause of CLD in females. Hepatitis C was the third most common cause of CLD in our study. A total of 26 cases were positive for Hepatitis C of which 4 were females and 22 were males. Out of these 26 cases, 16 cases also had a history of significant alcohol consumption. In our study, 11 patients had diabetes mellitus. Out of which 7 were males and 4 were females. Of this, 3 patients also had a history of significant alcohol intake, and Hepatitis B and Hepatitis C infection were present in 3 and 1 cases, respectively. The etiology of CLD could not be ascertained in 2 cases as they were non-alcoholic, non-diabetic, and tested negative for Hepatitis B and Hepatitis C infections. They were classified as having an unknown etiology of CLD. In our study, only 7 cases belonged to Child-Pugh Class A, while 27 and 66 cases belonged to CP classes B and C respectively. Hepatic encephalopathy was the most common cause of admission to the hospital. HE was present in 97% of the cases. HE I, II, III and IV were present in 24%, 31%, 27% ND 15%respectively. Only 3 cases did not have HE. Patients with PVT often had a more severe grade of HE than those who did not have associated PVT. 6 out of 11 patients of PVT had HE grade IV. Ascites was the second most common cause of admission to the hospital. 92 cases had ascites. Only 8 cases did not have ascites, of which 4 cases belonged to Child-Pugh class A and 4 to class B. In our study, upper gastrointestinal bleeding was the third most common cause of presentation to the hospital. In our study 31% of the cases presented with UGI bleed. In our study, although the number of males presenting with UGI bleed was more than the number of females (28 and 3 in males and females respectively), the difference was not statistically significant (p=0.24). Out of a total of 11 cases of CLD with associated PVT, UGI bleed was present in 9(81.82%) of the cases, and the difference between those cases with associated PVT but without UGI bleed was statistically significant (p=0.001). HRS was present in 26 patients, of which 21 were male (25.61% of the total number of males) and 5 were females (27.78% of the total number of females). In our study, none of the cases belonging to Child-Pugh class A had HRS, while in CP class C, 22 patients (33.33% of the total number of cases in CP Class C) were having HRS. Out of 11 cases of PVT, 5 patients had HRS but there was no statistically significant difference between those who did not have associated PVT (p=0.14). Spontaneous bacterial peritonitis was present in 16 cases of which 11 were males (13.41% of the total number of males) and 5 were females (27.77% of the total number of females). Most of the cases diagnosed with SBP belonged to Child-Pugh Class C (16.67%) but the number was not statistically significantly different from those in Class A and B (p=0.56). SBP was present in 2 patients of CLD with associated PVT.

Comparison with previous studies: Etiology and clinical profile

The below table is representing comparison between different studies (Table 4).

Parameter	Our study	Brij Sharma et al	Partha S. Mukherjee	Michitaka et al	Nahum et al
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 (Percentage)	0.82	0.697	0.73	0.624	0.4893
Female (Percentage)	0.18	0.303	0.27	0.376	0.5107
Prevalence of Alcohol (Percentage)	45%*	0.629	0.343	0.136	0.395
Prevalence of Hepatitis B (Percentage)	0.29	0.101	0.333	0.139	0.05
Prevalence of Hepatitis C (Percentage)	0.26	< 27%	0.216	0.609	0.366
Prevalence of diabetes	0.11	-	0.117	0.012	-

mellitus					
Prevalence of Ascites (Percentage)	0.92	0.898	-	-	-
Prevalence of HE (Percentage)	0.97	0.697	-	-	-
Prevalence of SBP (Percentage)	0.16	0.247	-	-	-
Prevalence of HRS (Percentage)	0.26	-	-	-	-
Prevalence of UGI Bleed (Percentage)	0.31	0.27	-	-	-

*Alcohol intake was present in 71 patients in which 16 patients also had concomitant Hepatitis B and 16 had concomitant Hepatitis C infection.

Table 4. Comparison between different studies.

Chronic liver disease with associated portal vein thrombosis: Comparison with international studies

The below table is the comparing the clinical profile of cases of CLD with PVT (Table 5).

Total patients	Our Study	Lankarani et al	Lucio Amitrano et al	Borjas-Almaguer et al	Rosa Zampino et al
	100	219	701	169	130
Patients with PVT	11(11%)	35 (15.9%)	79(11.2%)	13(7.6%)	19(14.16%)
Male	7(63.64%)	26(74.28%)	47(59.4%)	8(61.5%)	8(42.1%)
Female	4(36.36%)	9(25.71%)	32(40.6%)	5(38.5%)	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 B in 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 expressed as number (percentage)

Table 5. Comparing the clinical profile of cases of CLD with PVT.

Comparison with Indian study

A similar study was done by Sivanesan et al17 in Chennai. Their sample size was 182. The incidence of PVT in their study (18.13%) was higher than our study. In our study, PVT was most commonly found in patients of CLD with associated alcohol (45.45%), second most common cause was hepatitis B infection (36.36%). But in the study done by Sivanesan et, PVT was most commonly present in CLD attributed to hepatitis B (72.72%). This difference may be due to different geographical, ethnic variations or may be due to different prevalence of hepatitis B and needs to be further studied. In a study done by Sivanesan et al most (90%) patients belonged to Child-Pugh Class B and C. However, in our study, all patients belonged to Child-Pugh Class C (Table 6).

Total patients	Our Study	Sivanesan et al
	100	182
Patients with PVT	11(11%)	33(18.13%)
Prevalence of Alcohol in patients with PVT	5(45.45%)	-
Prevalence of Hepatitis B in patients with PVT	4(36.36%)	24(72.72%)
Prevalence of Hepatitis C in patients with PVT	2(18.18%)	-
Child Pugh class A	0	90% belongs to Child class B and C
Child Pugh Class B	0	
Child Pugh Class C	11(100%)	
UGI bleed	9(81.82%)	-
SBP	2(18.18%)	-
HE	11(100%)	-
All data has been expressed as number(percentage)		

Table 6. Comparison with Indian study.

Strengths of our Study

A fairly wide age group of patients were enrolled in our study ranging from age 20 to the age of 80 with a mean age of 46 years. Not many studies have been done in India studying the clinical profile of CLD patients and CLD patients having associated PVT. Many of our results were resonant with those of international studies as well as Indian studies done before. This study, thus, adds to the already

existing literature about the clinical and etiological profile of CLD patients and about the patients who are having associated portal vein thrombosis. Our study gives a view of this problem in the north Indian population, which may be different from other parts of India and will help in doing follow-up studies to compare these results with different entities causing chronic liver disease. Many studies can originate from this concept to evaluate regional differences.

Limitations of our study

Our study was conducted in a setting which caters to patients belonging primarily to the lower or middle socio-economic strata and the data primarily reflects the situation in this cohort. Being located in a metropolitan area, the study population consisted predominantly of the urban population. Our study comprised predominantly cases of CLD attributed to alcohol, hepatitis B and hepatitis C, Nonalcoholic and Nonviral causes were a small group represented in this study during the study period. Our study was an observational study. Our study did not focus on a comparative study to compare the clinical profile of the patients of PVT with those who did not have PVT. We would have gained more information if it would have been done. We studied only 100 patients. It was a single-center study. The real relevance of an entity is determined by the effect that it has on the long-term profile of the patient. Since we did not follow up, the clinical relevance of PVT in CLD was not studied.

Conclusion

This study included 100 patients of CLD of which 82 were males and 18 were females with a mean age of 46.72 ± 11.04. The mean age of females was slightly more (55.2± 7.2) than the mean age of males (44.8 ± 10.8) and the difference of statistically significant with p- value=0.0002. Thus, in our study, males outnumbered females in having CLD and presented at earlier ages. Hepatic encephalopathy was the most common presentation followed by ascites. Most of the patients presented late in a decompensated state and more than 90% conformed to Child-Pugh class B and C. Alcohol was the most common etiology of CLD in males in our study whereas in females, hepatitis B infection was the most common cause. Overall, when all etiologies were taken into consideration, the most common attributable reason for CLD in our study was alcohol followed by Hepatitis B, followed by Hepatitis C. Among other causes, Diabetes Mellitus was associated with 11% of our study group. There were no patients with Hypertension. Portal vein thrombosis affects the clinical profile of CLD. In our study, the incidence of PVT was 11% and was most common in CLD attributed to alcohol. In our study, most of the patients with PVT indicating and reiterating the association of PVT and the Stage of CLD.

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