A Cross Sectional Study of Chronic Liver Disease with Reference to Portal Hypertension and Endoscopic Finding at Down Town Hospital, Guwahati, Assam

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ABSTRACT

BACKGROUND

Cirrhosis is a progressive clinical condition associated with considerable mortality and morbidity. It leads to a wide spectrum of characteristic clinical manifestations, mainly attributable to hepatic insufficiency and portal hypertension.[1] In cirrhosis, primary diagnostic test for evaluation of upper-gastro-intestinal bleeding (UGIB) is endoscopy.[2] The present study attempts to find out different clinical patterns of the chronic liver disease (CLD) with portal hypertension along with the endoscopic profile of the patients.

METHODS

It was a cross sectional study conducted in Down Town Hospital, Guwahati, Assam.

RESULTS

A cross sectional study was conducted at Downtown Hospital, Guwahati, Assam on patients diagnosed with chronic liver disease from 01 January 2017 to 31 January 2018. Male predominance was observed in this study with 84 % males and 16 % females. Male to female ratio was 5.25 : 1. Maximum patients (58 %) were observed in the age group of 40 to 60 years followed by 30 % in the age group of 60 to 80 years. The average age was 53.6 years. Most common aetiological factor was alcohol (66 %) followed by non-alcoholic steatohepatitis (NASH) (16 %). Other aetiologies were hepatitis-B, hepatitis-C and cryptogenic. 88 % cases were recorded in model for end stage liver disease (MELD) score range of 10 to 29. Ascites was noted in 88 % cases, of which 20 % had grade-1 and grade-2, 48 % had grade-3. Splenomegaly was noted in 70 % cases. Child-Pugh class-C consisted of 26 % cases (13) with grade-III varices followed by 18 % cases (9) with grade-II varices while Child-Pugh Class-B had 24 % cases (12) and 8 % cases (4) with grade-III and grade-II varices respectively. Child-Pugh class-A had all the cases with grade-I varices.

CONCLUSIONS

Alcoholism was the leading cause for cirrhosis followed by hepatitis-B, hepatitis-C, NASH and cryptogenic. Class-C had maximum number of cases with grade-III varices followed by class-B. MELD score ranged between 10 - 29 in majority. Severe anaemia was noted in 10 % cases. Thrombocytopenia a non-invasive indicator of oesophageal varices was noted in 70 % with different grades of oesophageal varices.

KEYWORDS

Endoscopy, Portal Hypertension, Chronic Liver Disease

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BACKGROUND

Cirrhosis is a progressive and severe clinical condition associated with considerable mortality and morbidity. It leads to a wide spectrum of characteristic clinical manifestations, mainly attributable to hepatic insufficiency and portal hypertension.[1] Major complications include ascites, gastro-intestinal bleeding, hepatic encephalopathy, renal failure, bacterial infections and coagulopathy. Alcohol abuse and viral hepatitis are the most common causes of cirrhosis, although non-alcoholic fatty liver disease is emerging as an increasingly important cause.[2] Alcohol is consumed widely in most parts of the world and has long been identified as a major risk factor for all liver diseases.[3] Non-alcoholic fatty liver diseases (NAFLD) are now being increasingly recognized as a major health burden. It has been documented that a number of hepatic steatosis cases can lead to chronic liver disease.[4,5,6] Other causes include autoimmune hepatitis, sclerosing cholangitis, primary biliary cirrhosis, hemochromatosis, Wilson's disease, veno-occlusive disease, drugs.

Rupture of gastroesophageal varices as a result of portal hypertension is a severe and frequent complication of cirrhosis.[7] Gastrointestinal Bleeding is to be a major cause of chronic liver disease related hospital admission and mortality throughout the world ranging from 0.9 % to 26.5 %.[8,9] Upper gastrointestinal bleeding includes haemorrhage originating from the oesophagus to the ligament of Treitz. Peptic ulcer bleeding causes more than 60 percent of cases of upper gastrointestinal bleeding, whereas oesophageal varices attribute approximately 6 percent. Other aetiologies include arteriovenous malformations, Mallory-Weiss tear, gastritis, duodenitis, and malignancy.[10] Patients can be stratified as having either variceal or non-variceal sources of upper gastro-intestinal haemorrhage as the two have different treatment algorithms and prognosis. The primary diagnostic test for evaluation of upper gastro-intestinal bleeding is endoscopy.[11,12]

As the number of patients of chronic liver disease with portal hypertension and related morbidity and mortality is increasing day by day in this part of the country, the present study attempts to find out different clinical patterns of the CLD with portal hypertension along with the endoscopic profile of the patients.

METHODS

This is a cross sectional study. 50 cases were selected by using non-random (convenience) sampling technique from outdoor and indoor patients in Down Town hospital, Guwahati for a period of one year from January 2017 to January 2018.

Justification of Sample Size

Minimum sample size was calculated using the formula

$$n = \{Z^2\ p\ (1-p)\} / d^2$$

Where,

N is sample size

Z is standard normal variate corresponding to confidence level.

P is expected prevalence

D is precision or margin of error corresponding to effect size.

Minimum sample size was calculated to be 101 with confidence level of 95 % and precision of 5 %. Expected prevalence was taken to be 7 % as determined by reviewing previous studies. Patients who were eligible according to the inclusion/exclusion criteria and willing to participate in the study by giving written informed consent were included in the study. Many patients were not eligible to participate owing to the previous endoscopic interventions. With our best efforts to enrol maximum patients in the study, we were able to include only 50 subjects due to small number of patient flow in this setup, with the aim to complete the study within the given duration of time.

Inclusion Criteria

- Both male and female patients with varying grades of CLD with age more than 18 years.

Exclusion Criteria

- Patients without clinical features of portal hypertension.
- Patients who have undergone TIPS (Trans jugular intrahepatic portosystemic shunt.)
- Already diagnosed case of portal hypertension that has undergone endoscopic intervention.

A detailed history was noted and patients were evaluated clinically. Patients enrolled in the study were allotted identification number to protect their identity. Proper written consent was taken.

Blood was collected by venepuncture for blood investigations in these patients which included haemoglobin (Hb), total leukocyte count (TLC), differential count (DC), erythrocyte sedimentation rate (ESR), platelets, random blood sugar (RBS), creatinine, ammonia, liver function tests (LFT) with protein fraction. Prothrombin time and International normalized ratio (INR) were calculated.

Portal hypertension was diagnosed based on clinical examination findings including presence of ascites, splenomegaly and decreased platelet count in blood examination. Ultrasonography (USG) and doppler features such as portal vein dilation, portal vein occlusion and dilated intra-abdominal collaterals, splenomegaly were also considered. Child-Turcotte-Pugh (CTP), model for end stage liver disease criteria were used to assess the severity of liver disease. Patients will be divided into class a, class B, class C according to the criteria.

Endoscopic evaluation in the form of upper gastro-intestinal endoscopy was done to evaluate presence or absence of oesophageal varices with different grades and portal hypertensive gastropathy.

Data Collection Methods

For primary data - History taking and physical examination.

Tools

- Direct observations, interview schedule, protocols, tests, examination of medical records, and collection of writing samples.
- Child Pugh Score, MELD to assess severity and prognosis of liver disease.
- Modified Paquet classification for grading oesophageal varices of upper gastro-intestinal endoscopy.

Statistical Methods

Data has been analysed using descriptive statistics. Results of continuous measurement were presented as mean with standard deviation and results of categorical measurements were presented in number (%). Spearman's correlation test was used for correlation and Fisher's exact test was used for association. Data was presented in bar diagram, pie diagram, charts, table formats, for ease of understanding and interpretation. Epi Info was used for the analysis of the data and Microsoft Word and Excel have been used to generate charts, diagrams, graphs, tables etc.

RESULTS

A total of 50 patients fulfilling the inclusion/exclusion criteria were studied in this analysis. Out of 50 cases, 42 cases were male 8 cases were female out total of 50 cases i.e. 84 % cases were males and 16 % were females. In this study, the age incidence ranged from 20 years to 80 years. Chronic liver disease was seen predominantly in older age group with 88 % of patients > 40 years, of which 58 % were in between 41 and 60 years of age. Average age was 53.68 ± 12.08 years. Maximum number (29) of cases were seen in age group of 40 to 60 years of which 27 were males and 2 were females.

For males, maximum number cases were in the age group of 40 to 60 years, whereas for females it was seen in the age group of 60 to 80 years. Least number of cases were seen in age group of 20 to 40 years for males and females, and both combined. Alcohol was the most common aetiology accounting to 66 % of the cases followed by NASH which was 16 % of the cases. Hepatitis B and hepatitis C were accounted for 6 % and 4 % cases respectively. In 8 % cases the aetiology was cryptogenic.

Out of 50 cases, 45 (90 %) had abdominal distension followed by 44 (88 %) had pallor. Oedema was seen in 43 cases (86 %) whereas icterus was noted in 41 cases (82 %). Altered sleep was noted in 32 cases (64 %) while gastro-intestinal (GI) bleeding was seen in 25 (50 %). Other findings included fever and vomiting in 11 cases (22 %) each, while constipation was noted in 24 cases (48 %). Diarrhoea was seen in 3 cases (6 %). Signs of liver failure were noted in 46 cases (92 %). Splenomegaly was observed in 35 cases (70 %).

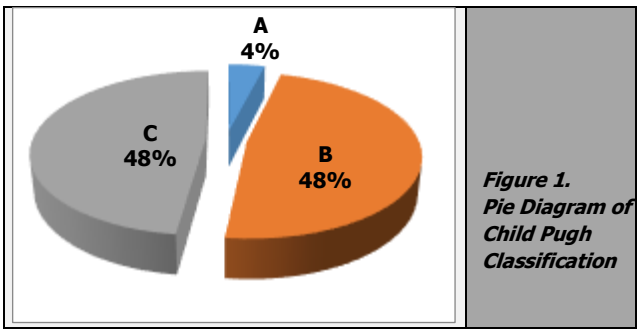

Figure 1. Pie Diagram of Child Pugh Classification

Distribution of cases according to Child Pugh Classification, 4 % patients were seen in Class A. Majority of the patients were seen in Class B and Class C which was 48 % each. In relation to sex and distribution, we found class B and Class C had equal number of cases i.e. 24 each. Class B had more males (21) compared to Class C (20); in contrast Class C had more females (4) compared to Class B (3). Class A had equal number of males and females (1).

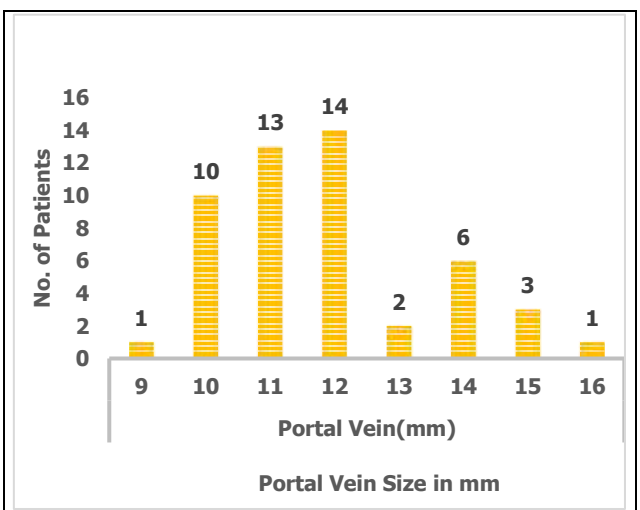

Figure 2. Portal Diameter

Maximum number of cases (17) was recorded Class C in age group of 41 to 60 years followed by Class B (10) in the same age group. No cases were recorded in Class A with age group of 20 to 40 years and 61 to 80 years. Out of 6 cases in age group of 20 to 40 years, 5 cases were seen in Class B. Out of 15 cases in age group of 61 to 80 years, 9 cases were seen in Class B. Total number of cases in Class A were 2, Class B and Class C represented 24 cases each.

Distribution of cases according to MELD score. 33 cases were recorded with MELD score within the range of 10 to 19. 11 cases were seen in the range of 20 - 29. 3 cases were recorded in the range 30 to 39, 2 cases were recorded in the range less than 9. Only 1 case was recorded with MELD score more than 40. 12 % cases did not have ascites, 20 % had grade 1 or mild ascites, 20 % cases had grade 2 or moderate and 48 % cases had grade 3 or severe ascites.

Maximum number of cases i.e., 14 cases (28 %) had portal vein diameter of 12 mm followed by 11 mm in 13 cases (26 %). Minimum diameter of 9 mm and maximum diameter of 16 mm was noted 1 case each. In the study majority of the cases were recorded with grade 3 varices (50 %), followed by 26 % with grade 2 varices. Grade 1

accounted for 20 % cases while 4 % cases did not have varices. In the relation and distribution to grade of varies, grade 3 varices were noted in both males (22) and females (3). Least number of cases had no varices in males and females i.e. 1 each.8 males and 2 females had grade 1 varices. 11 males and 2 females had grade 2 varices.

In relation to age distribution and grades of varices maximum number of cases were in age group of 41 to 60 years having grade 3 varices followed by 8 cases having grade 2 varices in same age group. Most of the cases in age group 61 to 80 years (7 cases) were recorded to have grade 3 varices followed by 4 cases having grade 1 varices. 3 cases in this age group had grade 2 varices. 1 case had no varices in this age group. Similar to this only 1 case had no varices in the age group 41 to 60 years. Majority of the cases (4) in age group 20 - 40 years had grade 3 varices followed by 2 cases with grade 2 varices. No patient in this age group was noted with grade 1 or no varices.

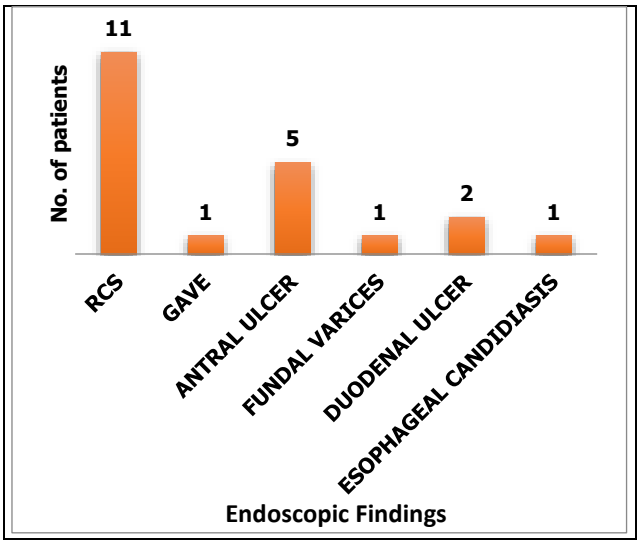

Figure 3. Endoscopic Examination

All cases with child A had grade 1 varices. Maximum number of cases (12) cases with child B had grade 3 varices followed by 7 cases which had grade 1 varices. 4 cases with child B had grade 2 varices and 1 case had no varices. In case of child C maximum cases (13) had grade 3 varices followed by 9 cases which had grade 2 varices, 1 case had grade 1 varices and 1 case was noted without varices.

Study shows that cases with no varices were equally distributed with 1 case each in platelet count range of 100000 to 150000 and 150000 to 200000. Maximum cases (5) of grade I varices were noted in the range 100000 to 150000 and 1 case in the range more than 200000. Majority of cases with grade II varices were seen in range of 50000 to 100000. Most of the cases (10) of grade III varices were noted in range 50000 to 100000 with just 1 case in range less than 50000.

Endoscopic finding in addition to varices in various cases revelled. Red colour sign (RCS) in 11 cases followed by antral ulcer which was seen in 5 cases. Gastric antral vascular ectasia (GAVE), fundal varices, oesophageal candidiasis were in 1 case each while duodenal ulcer was noted in 2 cases.

	Mean	SD	Min	Max	Percentiles		
					Q1	Median	Q3
Age	53.68	12.08	20	79	46.75	55	62.5
Pulse /min	84.22	9.45	66	112	78	84	88.25
BP (mm of Hg) systolic	125.12	15.72	100	160	110	130	132.5
BP (mm of Hg) diastolic	77.76	9.07	60	90	70	80	90
Haemoglobin	8.87	2.18	4.4	15.5	7.525	8.75	10.4
TC	7478.00	3928.77	1500	20800	4175	6750	9525
Neutrophils	66.32	12.02	39	88	58	68	76
Lymphocytes	25.76	12.68	8	62	17.25	23.5	33.5
Monocytes	5.50	2.59	1	12	3	5	7.25
Platelets	136740	56857.79	35000	280000	94500	130000	168500
ESR (mm/hr.)	58.52	30.98	5	135	35.75	50	70
RBS (mg/dl)	131.90	48.70	74	276	94.75	121	153.25
S. creatinine (mg/dl)	1.40	1.11	0.51	5.9	0.8075	1	1.365
Total bilirubin (mg/dl)	5.32	8.05	0.62	37.88	1.4825	2.35	5.8475
Direct bilirubin	3.86	7.72	0.1	37.3	0.6	1.15	3.15
Indirect bilirubin	1.44	1.19	0.1	5.6	0.6	1.1	1.825
Total protein(g/dl)	6.82	1.03	4	9.6	6.325	6.85	7.525
S. albumin (g/dl)	2.91	0.62	1.6	4.5	2.5	2.9	3.3
S. globulin (g/dl)	3.91	0.85	1.97	6.17	3.405	3.955	4.39
SGOT (units/L)	103.00	103.28	22	549	43.75	67	119.75
SGPT (units/L)	55.96	47.67	7	225	28.75	40	65.75
Alkaline phosphatase	159.94	77.40	46	393	95.25	140	208.5
GGT	167.76	151.13	13	607	42.75	131	239
INR	1.67	0.70	0.94	5.44	1.2775	1.48	1.8475
S. ammonia	56.84	78.97	9	459	19	31.5	52.5

Table 1. Descriptive Statistics of the Study Variables

		GI Bleed		Total
		Present	Absent	
Varices	No Varices	1	1	2
	Grade 1	4	6	10
	Grade 2	7	6	13
	Grade 3	13	12	25
Platelet count	< 50000	1	1	2
	50000 - 100000	12	6	18
	100000 - 150000	7	8	15
	➢ 150000	5	10	15

Table 2. Distribution of Number of Cases of GI Bleeding with Grade of Varices and Platelet Count

ESR-Erythrocyte Sedimentation Rate; RBS-Random Blood Sugar; SGOT-Serum Glutamic Oxaloacetic Transaminase; SGPT-Serum Glutamic Pyruvic Transaminase; GGT-Gamma-Glutamyl Transferase; INR-International Normalised Ratio.

Severe anaemia with haemoglobin < 6 mg/dL was seen in 5 cases (10 %). The mean haemoglobin value was 8.87 ± 2.18 mg/dL. Total leukocyte count (TC) > 11000/cumm was noted in 8 cases (16 %). The mean total leukocyte count was 7478 ± 3928.77/cumm. Serum creatinine > 1.25 mg/dL was seen in 14 cases (28 %) The mean serum creatinine was 1.40 ± 1.1mg/dL. Serum total bilirubin > 3 mg/dL was observed in 20 cases (40 %). Albumin level < 3.5 g/dL was noted in 41 cases (82 %). The mean albumin level was 2.91 ± 0.62. Mean platelet count was 136740 ± 56857.79/cumm with 35 cases (70 %) with platelet count < 150000 cumm. Mean INR was noted as 1.67 ± 0.70. Serum ammonia was noted with mean value 56.84 ± 78.97 umol/L with 20 cases (40 %) having value > 35 umol/L.

Most of the cases of GI bleeding i.e., 13 (52 %) out of 25 were noted in patients with grade 3 varices followed 7 cases (28 %) in grade 2 varices and 4 cases (16 %) in grade 1 varices. Maximum cases of GI bleeding i.e. 12 (48 %) out of 25 were noted in patients with platelet count in the range 50000 - 100000 followed by 7 cases (28 %) in range of 100000 -150000 and 5 cases (20 %) in range more than 150000.13 cases (52 %) were noted with platelet count less than 100000. Oesophageal varices were correlated with various study variables like platelet count, total bilirubin and serum albumin using Spearman's correlation test. Platelet count and serum albumin had negative correlation

coefficient while it was positive with total bilirubin level. Correlation between these variables was statistically non-significant. Association between GI bleeding and platelet count was statistically non-significant using Fisher's exact test.

		Oesophageal Varices				Total	Fisher's Exact Test
		No Varices	1	2	3		P Value
Signs of liver cell failure	Absent	1 25.00 %	2 50.00 %	1 25.00 %	0 0.00 %	4 100.00 %	0.02*
	Present	1 2.20 %	8 17.40 %	12 26.10 %	25 54.30 %	46 100.00 %	
Ascites (grade)	0	0 0.00 %	5 83.30 %	0 0.00 %	1 16.70 %	6 100.00 %	0.007*
	1	1 10.00 %	0 0.00 %	3 30.00 %	6 60.00 %	10 100.00 %	
	2	1 10.00 %	1 10.00 %	1 10.00 %	7 70.00 %	10 100.00 %	
	3	0 0.00 %	4 16.70 %	9 37.50 %	11 45.80 %	24 100.00 %	

Table 3. Association between Oesophageal Varices and Various Study Variables

*p < 0.05 statistically significant
P > 0.05 Non-Significant, NS

Association of oesophageal varices was calculated with various study variables using Fisher's exact test. Association was statistically significant with signs of liver cell failure and ascites. Distribution of cases in various grades by West Haven criteria of hepatic encephalopathy, maximum 23 cases (46 %) were seen in grade 0 followed by 14 cases (28 %) with grade 1. Grade 2 and grade 3 consisted of 5 cases (10 %) each. Least number of cases was noted in grade 4 which was 3 (6 %). Diabetes mellitus was the most common co-morbid condition seen in 15 cases followed hypertension and hypothyroidism in 6 cases. Other co-morbidities included nephrolithiasis, chronic kidney disease, hyperuricemia and bronchial asthma.

DISCUSSION

A total of 50 patients of age group > 18 years, who were diagnosed as chronic liver disease were studied for 12 months in down town hospital, Guwahati, Assam. Portal Hypertension was studied in these patients with the help of history, clinical examination, radiological and laboratory investigations and upper GI endoscopy. Portal hypertension is the major cause of severe complications and death in patients with cirrhosis.[13]

Development of oesophageal varices is one of the major complications of portal hypertension (PHT). A major cause of PHT-related morbidity and mortality is the development of variceal haemorrhage, which occurs in 25 - 40 % of patients.[14] Out of 50 patients which were included in this study, 84 % (42) were male and 16 % (8) were female with male to female ratio of 5.25 : 1.This predominance of male patients is comparable to study done by Uppalapati S, et al.[15] and Sharma S, et al.[16] in which it was 90 % and 86 % respectively. In this study, 88 % of the patients were more than 40 years of age, of which 30 % were more than 60 years of age. Maximum number of patients (58 %) was in age group of 40 to 60 years. Remaining 12 % were in age group of 20 to 40 years of age. This is usually due to compensated cirrhosis remaining undiagnosed for long period of time. Decompensated cirrhosis often presents late after most of complications have developed.

Maximum number of male cases (27) was in the age group of 41 - 60 years and females (5) the age group of 61 - 80 years. Least number of cases was noted in age group 20 - 40 years for both males (5) and females (1). The youngest patient was a 20 years old female and the eldest patient was a 79 years old male. Average age was 53.6 years in this study compared to 40.5 years in the study done by Devrajani et al.[17] and 39 years in study done by Uppalapati S et al.[15]

In our study causes of cirrhosis included alcoholism, viral hepatitis, NASH related and cryptogenic cirrhosis. Of these in 66 % patients, alcohol was the aetiological factor followed by NASH in 16 % patients. This was similar to the study done by Choudhary et al.[18] in which 69.86 % of the total subjects the cause was alcohol, followed by NAFLD (19.17 %). Chandra et al.[19] in his study stated that alcohol is the most common culprit for CLD in Central India. In the present study abdominal distension was most common presentation followed by pallor (88 %) and oedema (86 %). Icterus was present in significant number of cases (82 %). Other major presenting complaints were altered sleep (64 %) and GI bleeding (50 %). Fever (22 %) and other GI symptoms like vomiting (22 %), diarrhoea (6 %), and constipation (48 %) were also noted. Signs of liver cell failure were present in large number of cases (92 %). In the study by Kaji et al.[20] abdominal distension was the most common presenting complaint followed by icterus and oedema over feet with pallor (88 %). Pal et al.[21] in his study revealed abdominal distension in 52 % of patients followed by icterus in 40 % and GI bleeding in 24 %, which is almost similar to the findings of present study.

The Child-Pugh score is a reliable predictor of survival in many liver diseases and predicts the likelihood of major complications of cirrhosis. This classification scheme was used to assess prognosis in cirrhosis.[22] In the present study, majority of the patients (96 %) were seen in Class B and Class C. Out of which 48 % belonged to Class C and Class B each and 4 % belonged to Class A which was similar to studies by Pal et al.[21] which found 51 % of patients belonged to Class B followed by 35 % in Class C and only 14 % to Class A and Chandra[19] et al. which showed 23 (38.33 %) presenting in Class C and 26 (43.33 %) presenting in Class B. Only 11 (18.33 %) patients presenting in Class a Child Pugh score.

Maximum patients, 34 % were seen in Class C in the age group of 41 - 60 years followed by 20 % in Class B in the same age group. Most males (21) were observed in Class B, most females (4) were observed in Class C. Equal number of males and females (1) were observed in Class A. Model for end-stage liver disease (MELD) is now used for assessing prognosis. In this study 88 % cases were recorded in MELD score range of 10 to 29. Bhattacharya M[23] et al. showed that 83 % patients had MELD score in the range 10 - 29 which was similar to our study.

Ultrasonography abdomen showed ascites in 88 % patients, splenomegaly in 70 % patients as compared to

studies by Kaji et al.[20] and Uppalapati S et al.[15] which reported ascites in 80 % and 93.3 % patients, splenomegaly in 70 % and 76.6 % patients respectively. Study by Bhattacharya M et al. [23] noted ascites in 76.6 % and splenomegaly in 82.8 % patients. Out of 88 % patients who had ascites, 48 % cases had grade 3 or severe ascites, 20 % cases had grade 2 or moderate and 20 % had grade 1 or mild ascites. Maximum number of cases i.e. 14 cases (28 %) had portal vein diameter 12 mm. Prihatini et al. demonstrated in their study that portal vein size 1.2-cm (12 mm) as non-invasive parameter to detect oesophageal varices in cirrhotic patients. Maximum diameter of portal vein was 16 mm and minimum was 9 mm.

Uppalapati S et al.[15] in his study found that out of 30 cases, 70 % had oesophageal varices. Sarwar et al.[24] and Chalasani et al.[25] in their studies found oesophageal varices in 64.3 % and 70 % respectively. Study done by Pal J et al.[21] found 78 % cases had oesophageal varices. Kaji et al.[20] stated that incidence of oesophageal varices in patients with PHT was 95 %. In present study prevalence of oesophageal varices was 96 % similar to above studies.

Based on modified Paquet classification for oesophageal varices on endoscopy, in this study population, 20 % of the cases had grade I varices, 26 % had grade II varices and 50 % cases were noted to have grade III varices. This was in contrast to study done by Uppalapati S, et al.[15] which reported incidence of grade I varices to be 50 % followed by grade II and grade III of 10 % in each grade. Among the patients with varices 45.61 % had grade II oesophageal varices and 17.54 % had grade I oesophageal varices. 36.85 % had large (grade III and IV) varices in the study done by Choudhary et al.[18] The study by Kaji et al.[20] showed asymptomatic oesophageal varices were found in 80 % of patients, 20 % had grade I, 26 % had grade II and 34 % had grade III oesophageal varices. These findings were comparable to the present study. In majority of the cases, grade III varices were observed in both males (22) and females (3) and grade II varices were noted in 11 males and 2 females. 8 males and 2 females had grade I varices. Majority of the cases were recorded with grade III varices in all age groups of which maximum cases (14) were noted in age group 41 - 60 years, followed by 7 cases in age group of 61 - 80 years. Age group of 20 - 40 years consisted 4 cases of grade III varices and 2 cases of grade II varices. The variations found in the present study can be attributed to the late presentation of the cases and exclusion criteria of excluding patients with prior endoscopic interventions.

In our study Child Pugh Class C had 26 % cases (13) with grade III varices followed by 18 % cases (9) with grade II varices. Child Pugh Class B had 24 % cases (12) and 8 % cases (4) with grade III and grade II varices respectively. All the cases of Child Pugh Class A had grade I varices. Our findings were similar to studies by Thapa et al.[26] and Sumon SM et al.[27] Thapa et al.[26] in their study observed that patient with high grade of Child Pugh's score had higher grade of oesophageal varices. Sumon SM et al.[27] noted in their study that Child Pugh's scores B and C was associated with higher grade of varices. This was contrary to findings in the study by Flores-Rendon AR et al.[28] which did not show relationship between oesophageal varices and Child Pugh score.

Choudhary et al.[18] recorded that among the 16 patient who did not have varices 93.75 % (15/16) had platelet count more than 150000 whereas among those who had varices 85.96 % (49/57) had platelet count less than 150000. Majority of patient with large varices (grade III and IV) i.e. 66.67 % (14/21) had platelet count less than 100000. Gill et al. reported that platelet counts 100000 are a reliable marker for predicting oesophageal varices in cirrhotic patients. In our study 70 % (35) cases had platelet count less than 150000 of which 17 cases had grade III varices, 9 cases had grade II varices and 8 cases had grade I varices and varices were absent in 1 case. 30 % (15) cases had platelet count more than 150000 of which 8 cases had grade III varices 4 cases had grade II varices, 2 cases had grade I varices and in 1 case varices were absent.

In the study by Uppalapati S, et al.[15] out of 30 cases, 16 cases had other significant endoscopic findings like portal hypertensive gastropathy, erosive gastropathy and red colour sign. In our study portal hypertensive gastropathy (PHG) was seen in 92 % cases of which 4 % was severe PHG. Other findings were red colour sign (RCS), gastric antral vascular ectasia (GAVE), antral ulcer, fundal varices, duodenal ulcer and oesophageal candidiasis. RCS was associated with grade III varices in all cases.

Compared with findings by Bhattacharya M, et al.[23] our patients had similar Hb % (8.87 versus 8.69 gm/dl) and low platelet counts (1.30 ± 0.52 vs 1.36 ± 0.56 lakh/cumm). Serum creatinine was found to be similar in both studies (1.40 ± 1.1 versus 1.37 ± 2.10). Mean albumin levels were low and comparable in both studies (2.91 ± 0.62 versus 2.69+ 0.64 mg/dl). Our study noted total bilirubin level higher (5.32 ± 8.05 versus 4.25±7.83 mg/dl) while INR was lower (1.67 ± 0.70 versus 1.74 ± 0.76). The mean total leukocyte count was comparable in both studies (7478 ± 3928.77/cumm versus 7643.9 + 6161.6). Serum ammonia was noted with mean value 56.84 ± 78.97 umol/L with 20 cases (40 %) having value > 35 umol/L.

Serum total bilirubin > 3 mg/dL was observed in 20 cases (40 %) compared to 26.2 % as noted by Chandra et al.[19] Albumin level < 3.5g/dL was noted in 41 cases (82 %) similar to 79.3 % in study done by Chandra et al.[19]

Majority of cases of GI bleeding were noted in grade II and grade III which was 28 % and 52 % respectively. Grade 1 varices consisted of 6 % cases. Similar findings were noted in study by Nevens F et al.[29] and Erdogan A et al.[30] Both noted increased risk of bleeding with higher grade of oesophageal varices. Maximum cases of GI bleeding i.e. 12 (48 %) out of 25 were noted in patients with platelet count in the range 50000 - 100000 followed by 7 cases (28 %) in range of 100000 - 150000 and 5 cases (20 %) in range more than 150000. 13 cases (52 %) were noted with platelet count less than 100000. Goh et al. found thrombocytopenia, splenomegaly, and presence of ascites as predictors of the presence of bleeding. Oesophageal varices were correlated with various study variables like platelet count, total bilirubin and serum albumin. Platelet count and serum albumin had negative correlation coefficient. Total bilirubin had positive correlation coefficient. It was statistically non-significant. (P - value 0.92, 0.88 and 0.43 respectively).

Association of oesophageal varices was calculated with various study variables using Fisher's exact test. Association was statistically significant with signs of liver cell failure and ascites. This can be attributed to ascites, signs of liver cell failure and presence of oesophageal varices which mark decompensated liver disease with portal hypertension.[31] Association between GI bleeding and platelet count was statistically non-significant in our study.

Patients were distributed in various grades by using West Haven criteria of grading hepatic encephalopathy. Most of the cases (46 %) were noted in grade 0 followed by 28 %, 10 %, 10 % and 6 % in grade 1, grade 2, grade 3 and grade 4 respectively. 54 % cases had hepatic encephalopathy which was higher compared to studies done by Devrajani BR, et al.[17] Bhattacharya M, et al.[23] and Shah H[32] et al. which was 24 %, 21.6 % and 6.9 % cases respectively.

Diabetes mellitus was the most common co-morbid condition followed by hypertension and hypothyroidism. Other co-morbidities included chronic kidney disease, hyperuricemia, nephrolithiasis and bronchial asthma.

In this study we found that alcoholism was the major etiological factor of cirrhosis which was seen in 66 % of our cases. This is a modifiable factor and an avoidable one. Most of the studies worldwide have reported it as a leading etiological factor for development of cirrhosis. Majority of the patients belonged to child B and child C class. This implies that the patients were in decompensated stage. Most of the cases (96 %) had oesophageal varices of which maximum number of cases (50 %) had grade III varices. In conclusion, regardless of severity of signs and symptoms patient should be subjected to endoscopic evaluation for early diagnosis and management of portal hypertension to prevent further complications and reduction of overall morbidity and mortality in cirrhotic patients.

CONCLUSIONS

Chronic liver disease is a commonly encountered disease, presenting with variety of sign and symptoms. Presentation may vary owing to the time of presentation and depending upon associated complications. Middle aged males are most commonly affected. Male predominance is noted as compared to females. Portal hypertension leads to other complications like ascites, oesophageal varices and splenomegaly. Most common presentation was abdominal distension due to development of ascites. Pallor, oedema and icterus were other common findings. Splenomegaly was seen in 70 % cases. Gastrointestinal bleeding was noted in 50 % cases. Majority of the cases with bleeding had grade III varices. Alcoholism was the leading cause of cirrhosis in the present study. Other causes included hepatitis B, hepatitis C, NASH and cryptogenic. Majority of the cases were noted in child B and child C class. Class C had maximum number of cases with grade III varices followed by Class B. MELD score was in the range of 10 - 29 in most of the cases. Oesophageal varices were present in 96 % of the cases, of which 50 % cases had grade III varices, majority of which were seen in older patients. Other endoscopic findings included red colour sign, antral ulcer, gastric antral vascular ectasia (GAVE), fundal varices, oesophageal candidiasis, and duodenal ulcer. Severe anaemia was noted in 10 % cases and hypoalbuminaemia was seen in majority of the cases. Thrombocytopenia has been considered as non-invasive indicator of oesophageal varices in many studies. Thrombocytopenia was noted in 70 % with different grades of oesophageal varices. Diabetes mellitus was the most common co-morbidity noted followed by hypertension and hypothyroidism.

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REFERENCES

[1] Afdhal NH. The natural history of hepatitis C. Seminars in Liver Disease 2004;24(Suppl 2):3-8.
[2] Heidelbaugh JJ, Bruderly M. Cirrhosis and chronic liver failure: part I. Diagnosis and evaluation. American Family Physician 2006;74(5):756-762.
[3] Rush B. An inquiry into the effects of ardent spirits upon the human body and mind: with an account of the means of preventing, and of the remedies for curing them. 8th edn. Reprint. Exeter (NH): Richardson 1785.
[4] Agarwal AK, Jain V, Singla S, et al. Prevalence of non-alcoholic fatty liver disease and its correlation with coronary risk factors in patients with type 2 diabetes. J Assoc Physicians India 2011;59:351-354.
[5] Cheah WL, Lee PY, Chang CT, et al. Prevalence of ultrasound diagnosed non-alcoholic fatty liver disease among rural indigenous community of Sarawak and its association with biochemical and anthropometric measures. Southeast Asian J Trop Med Public Health 2013;44(2):309-317.
[6] Baffy G, Brunt EM, Caldwell SH. Hepatocellular carcinoma in non-alcoholic fatty liver disease: an emerging menace. J Hepatol 2012;56(6):1384-1391.
[7] Garcia-Pagan JC, De Gottardi A, Bosch J. Review article: the modern management of portal hypertension--primary and secondary prophylaxis of variceal bleeding in cirrhotic patients. Aliment Pharmacol Ther 2008;28(2):178-186.
[8] Tammaro L, Di Paolo MC, Zullo A, et al. Endoscopic findings in patients with upper gastrointestinal bleeding clinically classified into three risk groups prior to endoscopy. World J Gastroenterol 2008;14(32):5046-5050.
[9] Stanley AJ. Update on risk scoring systems for patients with upper gastrointestinal haemorrhage. World Journal of Gastroenterology 2012;18(22):2739-2744.
[10] Wilkins T, Khan N, Nabh A, et al. Diagnosis and management of upper gastrointestinal bleeding. Am Fam Physician 2012;85(5):469-476.
[11] Anand D, Gupta R, Dhir M, et al. Clinical and endoscopic profile of patients with upper gastrointestinal bleeding

at tertiary care centre of North India. Journal of Digestive Endoscopy 2014;5(4):139-143.
[12] Garcia-Tsao G, Sanyal AJ, Grace ND, et al. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. American Journal of Gastroenterology 2007;102(12):922-938.
[13] Al-Busafi SA, Ghali P, Wong P, et al. Endoscopic management of portal hypertension. Article ID 747095, International Journal of Hepatology 2012;2012:12.
[14] Sanyal AJ, Bosch J, Blei A, et al. Portal hypertension and its complications. Gastroenterology 2008;134(6):1715-1728.
[15] Uppalapati S, Lokesh S. Correlation of portal vein diameter with the presence of oesophageal varices in chronic liver disease: a prospective study. International Journal of Advances in Medicine 2018;5(4):859-864.
[16] Sharma SK, Aggarwal R. Prediction of large esophageal varices in patients with cirrhosis of the liver using clinical, laboratory and imaging parameters. Journal of Gastroenterology and Hepatology 2007;22(11):1909-1915.
[17] Devrajani BR, Soomro AA, Attra KM. Variceal bleeding and its dependence on portal vein size in liver cirrhotic patients. World J Medical Sciences 2009;4(1):50-53.
[18] Choudhary DRJ, Barupal DRKG. A study of correlation of portal vein diameter and spleen size with gastro-esophageal varices in portal hypertension - a cross sectional observational study. Indian Journal of Applied Research 2018;8(10):72-75.
[19] Chandra KP, Singh SN, Pawah AK. Study of clinico-etiological profile and the complication pattern in patients with chronic liver disease at tertiary care centre. International Journal of Contemporary Medical Research 2016;3(6):1668-1670.
[20] Kaji BC, Yagnesh BR, Nitin P, et al. A study of clinical profile of 50 patients with portal hypertension and to evaluate role of non-invasive predictor of esophageal varices. Indian Journal of Clinical Practice 2012;22(9):454-457.
[21] Pal J, Dasgupta S, Agarwal V, et al. Clinical profile of chronic liver diseases in a tertiary care center in Kolkata. J Association of Physicians of India 2003;51:1173-1174.
[22] Bacon BR. Cirrhosis and its complications. Harrison's Principles of Internal Medicine. 19th edn. McGraw-Hill Education 2015: p. 2058-2067.
[23] Bhattacharyya M, Barman NN, Goswami B. Clinical profile of cirrhosis of liver in a tertiary care hospital of Assam, North East India. IOSR Journal of Dental and Medical Sciences 2016;15(1):21-27.
[24] Sarwar S, Khan AA, Alam A, et al. Non-endoscopic prediction of presence of esophageal varices in cirrhosis. J Coll Physicians Surg Pak 2005;15(9):528-531.
[25] Chalasani N, Imperiale TF, Ismail A, et al. Predictors of large esophageal varices in patients with cirrhosis. The American Journal of Gastroenterology 1999;94(11):3285-3291.
[26] Thapa P, Maharjan D, Tamang T, et al. Clinical correlation between Child Pugh's score and oesophageal varices in upper gastrointestinal endoscopy in cirrhotic patient. Journal of Kathmandu Medical College 2017;4(4):135-139.
[27] Sumon SM, Sutradhar SR, Chowdhury M, et al. Relation of different grades of esophageal varices with Child-Pugh classes in cirrhosis of liver. Mymensingh Medical Journal 2013;22(1):37-41.
[28] Flores-Rendón ÁR, González-González JA, García-Compean D, et al. Model for end stage of liver disease (MELD) is better than the Child-Pugh score for predicting in-hospital mortality related to esophageal variceal bleeding. Ann Hepatol 2008;7(3):230-234.
[29] Nevens F, Bustami R, Scheys I, et al. Variceal pressure is a factor predicting the risk of a first variceal bleeding: a prospective cohort study in cirrhotic patients. Hepatology 1998;27(1):15-19.
[30] Erdogan MA, Benli AR, Acmali SB, et al. Predictive value of mean platelet volume in variceal bleeding due to cirrhotic portal hypertension. Euroasian Journal of Hepatogastroenterology 2017;7(1):6-10.
[31] Melato M, Sasso F, Zanconati F. Liver cirrhosis and liver cancer. A study of their relationship in 2563 autopsies. Zentralbl Pathol 1993;139(1):25-30.
[32] Shah H, Manohar TP. Clinical endoscopic parameters of upper gastrointestinal bleeding. Int J Com Health and Med Res 2016;2(1):8-13.