Original Research Article

The study of clinical profile and aetiology of portal hypertension amongst adults attending OPD in tertiary care hospital

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Abstract

Background: Portal hypertension (PH) is defined as when the portal venous system pressure exceeds 10 mm Hg. Very few studies have been done in the present field practice area, with this view the present study was carried out. **Methodology:** It was a prospective, observational study carried out on patients admitted in/and referred to the tertiary care hospital and attending the General Medicine OPD. **Results:** The study comprised of 202 patients with portal hypertension as per clinical, laboratory and radiological criteria. It was a prospective, observational study. Clinical profile, aetiology and complication of portal hypertension was studied. The mean age of presentation of portal hypertension was 45.8+_13.92. Abdominal distension due to ascites was the commonest presenting complaint seen in 162 (80.20%) patients. Most common aetiology amongst cirrhotic patients was found to be ALD in 128(63.36%) followed by cryptogenic liver cirrhosis 36 (17.82%),post viral 18 (8.91%),4 (1.98%) in each wilson disease and budd chiary syndrome, autoimmune in 3 (1.48%),congenital hepatic fibrosis 1 (0.49%) and multifactorial in 2 (0.99%) patients. Most common aetiology amongst non cirrhotic patients was found to be Extra hepatic portal vein obstruction (PVT) 3 (1.49%). Other aetiologies were Budd NCPF (Non-Cirrhosis) 1 (0.50%), post viral 1 (0.50%), Caroli's disease 1 (0.50%). **Conclusion:** Liver cirrhosis was the most common cause of portal hypertension. In portal hypertension with liver cirrhosis, Alcoholic liver disease was the most common aetiology, with rare causes being cryptogenic liver cirrhosis, post-viral, Budd Chiary syndrome, Wilson's disease, autoimmune, Congenital hepatic fibrosis. In patients with Portal hypertension without cirrhosis most had EHPVO, some patients had post viral chronic active hepatitis, NCPF and caroli's disease.

Key Words: Portal hypertension.

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INTRODUCTION

Portal hypertension (PH) is defined as when the portal venous system pressure exceeds 10 mm Hg.[1] Portal hypertension is characterised by the increase in porto-systemic pressure gradient in any portion of the portal venous system[2]. Portal hypertension is the main complication of liver cirrhosis.[2,3] In 2007 in United States, liver cirrhosis accounted for 30,000 deaths; making it the 12th leading cause of death in the US, this was in accordance with the National Institute on Alcohol Abuse and Alcoholism (NIAAA).[4] In western countries, cirrhosis of the liver accounts for more than 90% cases of portal hypertension.[5] It is reported that in India, extra hepatic portal venous obstruction (EHPVO) is responsible for about one third cases of adults and more than half of the

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cases in children as a cause of portal hypertension.[5] The causes for portal hypertension are

1. Pre hepatic PH [normal wedged hepatic venous pressure (WHVP), free hepatic venous pressure (FHVP) with normal hepatic venous pressure gradient (HVPG)],
2. Hepatic PH (increased WHVP, normal FHVP, and increased HVPG) and
3. Post hepatic PH (increased WHVP and FHVP and normal HVPG)[6]

The difference between cirrhotic and non-cirrhotic portal hypertension is that cirrhotic PH is associated with an elevated hepatic venous pressure gradient (HVPG) predominantly due to raised sinusoidal resistance, while in the non-cirrhotic PHT (NCPH), HVPG is normal or only mildly elevated and is significantly lower than portal vein (PV) pressure.[7] Non-cirrhotic portal hypertension comprises diseases of the liver manifesting with portal hypertension due to intra-hepatic or pre-hepatic lesions in the absence of cirrhosis. The common cause of non-cirrhotic intra-hepatic portal hypertension worldwide is noted to be due to non-cirrhotic portal fibrosis (NCPF) or idiopathic portal hypertension (IPH).[8] Development of gastrointestinal variceal bleeding, which is a direct consequence of portal hypertension is noted to be the major cause of cirrhosis related morbidity and mortality.[9]Acute gastrointestinal haemorrhage from variceal rupture in the oesophagus or stomach often is a medical emergency, with 6-week mortality rates approaching 30% in those with severe liver disease.[10] Ascites is also reported to occur in severe cases of cirrhosis and portal hypertension. The complications of ascites include hepatorenal syndrome and spontaneous bacterial peritonitis.

MATERIAL AND METHODS

The study was initiated after obtaining the permission from the Institutional Ethics Committee of tertiary care hospital

Study Population: Study included the patients admitted in/and referred to the tertiary care hospital and attending the General Medicine OPD.

Study Site: The study was conducted in tertiary care hospital

Study Design: It was a prospective, observational study.

Study period: The study was conducted over a period of 2 years from the day of obtaining the permission of the Institutional Ethics Committee.

Study Selection Criteria

Inclusion Criteria

Patients who presented with signs and symptoms suggestive of portal hypertension (Hematemesis, Malena, Splenomegaly, Ascites)

Patients admitted for portal hypertension as diagnosed by esophageal varices on OGDscopy with or without portal vein thrombosis due to any cause and or SAAG >1.1 of Ascitic fluid.

- Patients willing to give written consent.

Exclusion Criteria

- Children below the age of 12 years.

Sample Size: Sample size includes all those patients who are attending General Medicine OPD and admitted as inpatient in tertiary care hospital during the study period and fulfilling all the inclusion and exclusion criteria.

Study procedure: The study was initiated after obtaining the approval from the Ethics committee of tertiary care hospital. The patients were selected based on the inclusion and exclusion criteria of the study protocol. The patients diagnosed with portal hypertension at tertiary care hospital and giving a signed informed consent were enrolled in the study. A detailed clinical history including the demographic details, past records (focusing on the quantity and duration of alcohol intake), along with clinical examination of vital signs with signs of liver cell failure - pulse, temperature, blood pressure and general examination for findings of pallor, oedema, icterus, clubbing, cyanosis, lymphadenopathy, chalky white nails, parotid size, dilated veins of abdomen, spider naevi, fetor hepaticus, KF ring, palmer erythema, Dupuytren's contracture and systemic examination including respiratory, cardiovascular and central nervous system was done in each patient. The results were interpreted as per the history, systemic examination and clinical examination which were tabulated and compared. In all patients investigations to confirm portal hypertension were done like OGD scopy, and/or SAAG. All patients were underwent basic investigations like Hb-CBC, LFT, Sr. creatinine, ascitic fluid examination, USG abdomen. Investigations for etiology of cirrhosis were done like HBsAg, HCV, ANA (in non-alcoholics), Portal vein Doppler and liver biopsy (wherever indicated).

RESULTS

The mean age pt presentation was 45.80+_13.92. Maximum patients (32.18%) are from 41-50 years of age group. Gender distribution has shown male predominance with 165 (81.68%) of patients and remaining 37 (18.32%) were females. It was found to be statistically significant. The commonest presenting complaints was abdominal distension in 162 (80.20%) of patients followed by pedal oedema in 104 (51.49%). Other complaints were GI blood loss in 34 (16.83%), jaundice in 50 (24.75%), altered sensorium in 13 (6.44%), fever in 21 (10.40%) and other complaints 87 (43.07%). Maximum number of patients was suffering from diabetes 17 (8.42%), followed by tuberculosis 13 (6.44%), hypertension 12 (5.94%),

pulmonary embolism 1 (0.5%), chronic kidney disease 1 (0.5%) and other conditions 12 (5.94%). Total 130 patients had history of alcoholism. Significant alcohol intake was reported by 116 (89.23%) of patients and non significant intake was reported by 14 (10.77%). Alcohol intake was found to be statistically significant. Pallor was present in 144 (71.29%) and there were 46 (22.77%) patients who were reported with splenomegaly both were found to be statistically significant. Cirrhosis was present in 196 (97.03%) of patients and absent in 6 patients (2.97%) Statistical significant association was found between cirrhosis and portal hypertension. Most common aetiology amongst cirrhotic patients was found to be Alcoholic liver disease in 128(63.36%) followed by cryptogenic liver cirrhosis 36 (17.82%),post viral 18 (8.91%),4 (1.98%) in each wilson disease and Budd - Chiari syndrome, autoimmune in 3 (1.48%),congenital hepatic fibrosis 1 (0.49%) and multifactorial in 2 (0.99%) patients. Most common aetiology amongst non cirrhotic patients was found to be Extra hepatic portal vein obstruction (PVT) 3 (1.49%). Other aetiologies wereNCPF (Non-Cirrhosis) 1 (0.50%), post viral 1 (0.50%), Caroli's disease 1 (0.50%).

DISCUSSION

Considering the age factor results match with the study results by Goel *et al*[11] where mean age of presentation was 46 years, Kaji BC *et al* [12], where mean age of presentation was 41 ± 10.9 years, Nayak J *et al*[13], in which maximum patients were found between 3rd and 4th decades. (Nayak J *et al*) and Maskey R study in Nepal where the mean age of the patients was 49.06 +/- 11.27 years. In present study Abdominal distension was reported in 162 (80.2%) patients and was the most common presenting complaint. Abdominal distension was the most common specific presenting complaint followed by jaundice and edema over feet in the study of Kaji *et al*.[12].In the study of Nayak J *et al*[13], presenting complaints were engorged abdominal vein in 14% patients, swelling of feet in 21% patients, hematemesis in 62% patients. In our study 14 (6.93%) patients presented with malena,5 (2.47%) presented with hematemesis and 7 (3.46%) presented with features of both malena and hematemesis, in rest of the 168 (83.16%) patients there were no such bleeding manifestations seen. However in our study the number of patients with oesophageal varices were 117 (57.92%) out of which 17 (14.52%) presented with bleeding manifestations and 100 (85.47%) patients were having asymptomatic varices. Asymptomatic esophageal varices were found in 80% of patients. Hematemesis was present in 62% and malena in 56 % in the study by Nayak J *et al*[13], the bleeding manifestations were higher as compared to our study. In our study 13 (6.43 %) patients have tuberculosis. Out of these patients, 6 (2.97%) had abdominal, 4 (1.98%) had pulmonary, 1 had disseminated tuberulosis (pulmonary and abdominal), 1 (0.49%) patient of Tubercular meningitis and 1 (0.49%) had pott's spine. Thus in our patients extra pulmonary TB was more common. In a Korean study by Thulstrup *et al* 31% patients with cirrhosis had extra pulmonary tuberculosis as compared to 12 % in non cirrhotic control group. Higher incidence of tuberculosis in liver cirrhosis patients could be due to fact that, liver cirrhosis leads to malnutrition specially in alcoholic liver cirrhosis and also an immunodeficient state. In our study out of the 130 patients who consumed alcohol 116 (89.23%) showed significant alcohol intake (60-80 gm of alcohol per day consumption for males and> 20 gm of alcohol per day for females for > 10 years). There were 29% cases of cirrhosis attributed to alcohol in Goel A *et al*[11] As per study by Kaji *et al*[12] in 52% cases cirrhosis attributed to alcohol. In our study the number of patients with anaemia (male-Hb<12 and females Hb<10) were 55 (27.23%). So anaemia was found to be a common presenting sign in portal hypertension. In our study alcoholic liver cirrhosis was leading aetiology of liver cirrhosis, in this population of patients anaemia is contributed by, nutritional factors like Vitamin B12, iron, folate deficiency. Anaemia due to liver cirrhosis is multifactorial like hypersplenism, blood loss due to vatical bleeding and in severe liver cirrhosis there is spur cell anaemia. The number of patients with pancytopenia were 32 (15.84 %).Presence of pancytopenia in these patients was suggestive of hypersplenism. Anemia was reported in 88% patients with portal hypertension in a study by Kaji BC *et al*[12], 2012 and 85% cases in Nayak *et al*[13] These reports were more or less similar to our study. While noting the aetiology of portal hypertension, in our study, out of the causes of portal hypertension with cirrhosis, there were 128 (63.36%) reported with ALD, 36 (17.82%) patients with cryptogenic liver cirrhosis, 1 (9.40%) patients with post-viral, 4 (1.98%) patients had Budd Chiary syndrome, 4 (1.98%) had Wilson's disease, 3 (1.49%) patients were autoimmune, 1 (0.49%) patient had 1 (0.49%) had Congenital hepatic fibrosis. 2 (0.99%) patients had multifactorial causes like alcoholic liver disease with viral hepatitis. All 4 (1.98%) diagnosed Budd-Chiary patients in our study had liver cirrhosis. However liver cirrhosis is seen in only advanced cases of Budd Chiary and is a late complication, As all our cases had liver cirrhosis due to advanced disease they were classified under sinusoidal (cirrhotic) causes and not as post-sinusoidal cause or vascular cause in our study. Caudate lobe hypertrophy was observed in 2 patients. In the study by Goel *et al*[11] 4 % patients had budd-chiary syndrome.

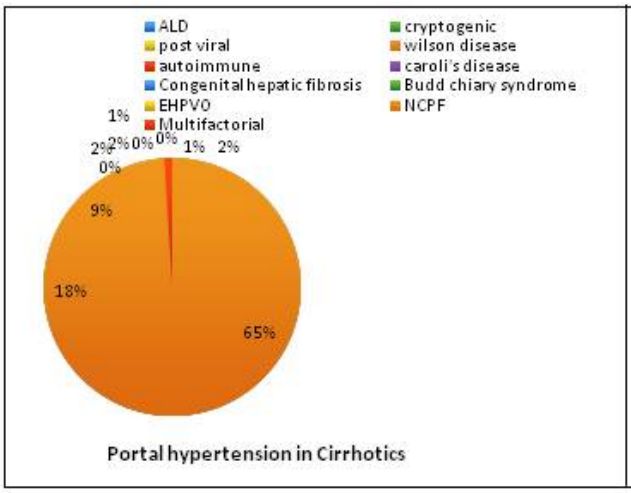

Figure 1

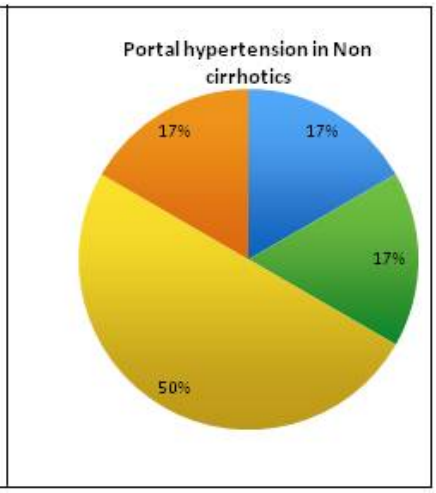

Figure 2

LIMITATIONS

Liver biopsy was not done. Portal vein diameter was not measured SAAG and Child-Pugh score was not calculated.

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