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Medicine

KEY WORDS:

COMPLICATIONS, MORTALITY AND SCORING SYSTEMS IN LIVER CIRRHOSIS

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Introduction

Cirrhosis named by Laennec in 1826 means orange or twany in Greek (1). Many forms of liver injuries are marked by fibrosis. This response to liver injury is potentially reversible. In contrast, cirrhosis is not a reversible process (2). Cirrhosis is defined by the World Health Organization (WHO) as a diffuse process characterized by fibrosis and the conversion of normal liver architecture into structurally abnormal nodules (3,4). The progression of liver injury to cirrhosis may occur over weeks to years. Chronic liver diseases and cirrhosis result in 26,000-35,000 deaths each year in the United States (US). Cirrhosis is the 9th leading cause of death in the US and is responsible for 1.2% of all US deaths (5). Over 2 lakh people lose their lives every year in India due to liver disease. While around 20,000 people require liver transplantation, only 2,000 transplantations are being done in India in a year.

Cirrhosis can be classified as follows: 1) alcoholic; 2) cryptogenic or post hepatic; 3) biliary; 4) cardiac; 5) metabolic 6) inherited and 7) drug related (6). Chronic liver Disease caused by steatohepatitis (alcohol or obesity) or chronic viral hepatitis leads to morphological changes in the liver. These changes could be attributed to four processes: 1) cell damage and degeneration; 2) cell death and necrosis; 3) liver regeneration; and 4) fibrogenesis. Cirrhosis is the consequence and final stage of various Chronic liver disease (7) Associated to this phenomenon, in cirrhotic patients, increased intrahepatic vascular resistances leads to portal hypertension and its complications, namely gastrointestinal (GI) bleeding from varices and/or ascites. Moreover, major functions of the liver are also impaired such as immunological function with increased infection sensibility and several perturbations in anabolism and catabolism liver function. Unfortunately, there are no correlations between morphological changes and the severity of functional impairment. Nevertheless, put together, all these perturbations, often asymptomatic when cirrhosis is 'compensated', become symptomatic when the cirrhosis is 'decompensated'.

Natural history of the disease could be progressive, with a slow decrease of liver function but without the potential for full recovery leading to end-stage of cirrhosis. End-stage of cirrhosis is characterised by chronic decompensation of the liver. At which point, the only definitive treatment is liver transplantation (Ltx). Patients with CLD may have acute decompensation (AD) that is usually precipitated by an event that represents a direct or indirect hepatic insult. For example, indirect insult could be infection or extra-hepatic surgery. Direct insult could be new viral hepatitis infection (like virus Delta or E), viral hepatitis reactivation, or hepatotoxic drug misuse. In case of AD, partial or full recovery to the original liver function level is assumed after treatment. The clinical presentation of cirrhosis is variable depending on the aetiology and whether hepatocellular or portal hypertension predominates.6 However, severe liver injury may also be present without any clinical signs (compensated). The diagnosis of cirrhosis is based on the clinical features, laboratory investigations, histology and radiologically. A number of prognostic scoring systems such as the Child Pugh and the MELD Na score which incorporate important biochemical parameters such as serum creatinine, serum bilirubin, prothrombin time, serum albumin and relevant clinical parameters such as the degree of ascites and stage of encephalopathy(8), have been established in the past. However, there has been limited research on the prognostic significance of these individual parameters and their association with mortality in patients with decompensated liver cirrhosis. The profile of cirrhosis may vary with different age and ethnic groups, geographical, social and etiological factors. Hence we undertook this short study in our hospital.

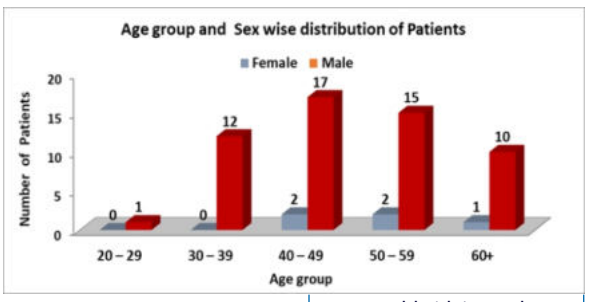
MATERIALS AND METHODS

The study was a prospective, cross sectional, comparative study conducted among 60 patients with liver cirrhosis fulfilling the proposed inclusion and exclusion criteria, admitted at MGM Medical College and Hospital, Navi Mumbai. Patients between 18 to 70 years were included in the study. The case of clinical cirrhosis was defined as a patient having at least one clinical sign of hepatocellular failure and one of portal hypertension along with at least three ultrasound (USG) findings suggestive of cirrhosis of liver. The diagnosis of alcoholic cirrhosis was made on the basis of history of any form of alcohol consumption >80g/dl in men and > 40g/dl in women for 10yrs. Spontaneous Bacterial Peritonitis (SBP) was considered if the ascitic fluid analysis showed one of the following: • Total leukocyte count (TLC): >500 cells/µml. Total polymorphonuclear (PMN) count: >250 cells µml. Ascitic fluid culture positive. All relevant blood investigations haematological and biochemical like Complete blood count, liver function tests, renal function tests, serum electrolytes, fasting and post prandial blood sugar, serum ammonia, prothrombin time/INR, viral markers were sent. Prognosis was measured by Child Turcot Pugh scores, MELD Na scores.

RESULTS

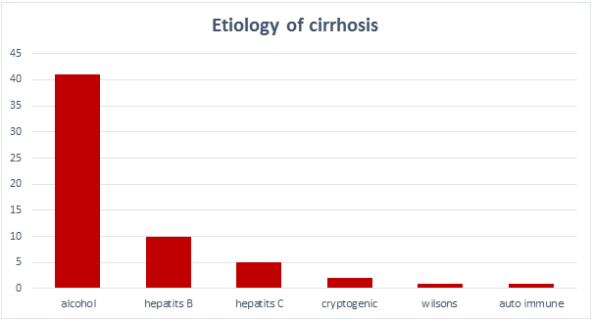
Age and sex wise distribution (graph 1)

Mean age of patients in the study was 48.62±11.36 years with the youngest patient being 22 years old and the oldest being 69 years old. Majority of the patients were in the 40-49 years age group. Most of the patients in the study group were males (91.67%).



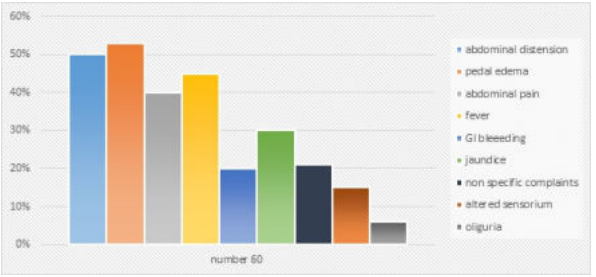
Aetiology of liver cirrhosis (graph 2)

Alcohol was the predominant aetiology of liver cirrhosis in 41/60 (68.33%), followed by Hepatitis B in 10/60 (16.67%), followed by Hepatitis C in 5/60 (8.33%), cryptogenic liver cirrhosis in 2/60 (3.33%), autoimmune hepatitis and Wilson's disease in 1/60 (1.67%).



Presenting complaints (graph 3)

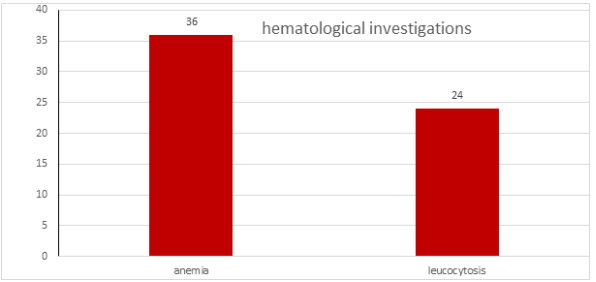
Abdominal distension was present in 50% patients followed by abdominal pain in was present in 40%, swelling in bilateral feet in 32/60(53%), fever in 27/60(45%), jaundice in 18/60(30%), other nonspecific complaints in 13/60(21%), GI bleeding manifestations in the form of hematemesis and Malena in 12/60(20%), altered sensorium in 9/60(15%) and oliguria in 4/60(6%) of all patients.



Abnormal blood investigations

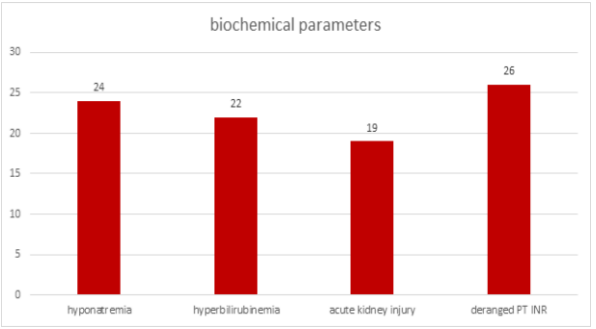
Haematological abnormalities (graph 4)

Anemia was present in 36/60 (60%) patients. Leucocytosis was present in 24/60 (40%) patients.



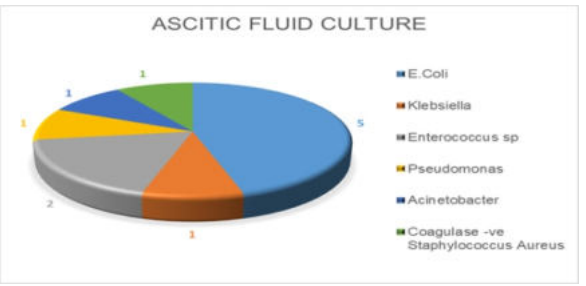
Abnormal biochemical parameters (graph 5)

Hyponatremia was present in 26/60 (40%) patients. Hyperbilirubinemia was present in 22/60 (36%) patients. Acute kidney injury was present in 19/60 (31%) patients. Coagulopathy in the form of deranged prothrombin time and INR was present in 26/60 (43.33%) patients.



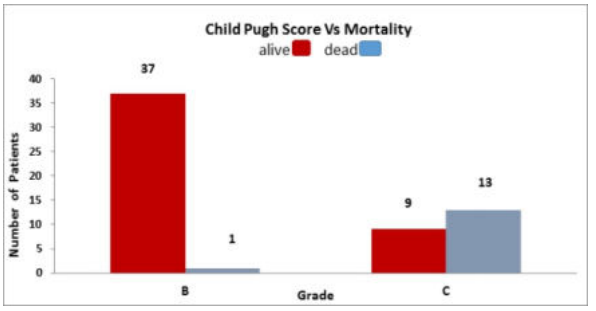
SBP and ascitic fluid culture (graph 6)

11/60 patients had SBP, .coli was the predominant organism found on culture in 5/11 (45.45%) patients (10 culture positive SBP and 1 Bacterascites), followed by Enterococcus species found in 2/11 (18.18%) patients followed by Klebsiella, Pseudomonas, Coagulase negative staphylococcus aureus found in 1/11 (9.09%) each.



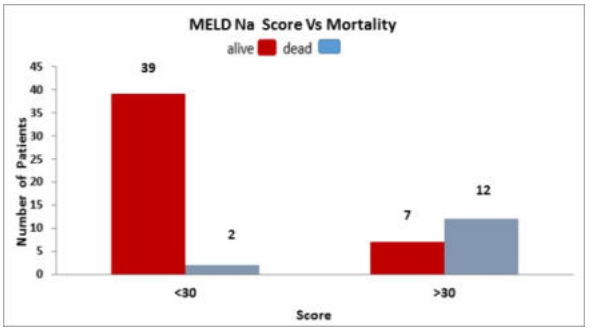
Child pugh score and mortality (graph 7)

Child Pugh score was grade C in 22/60 (36.67%) patients and 13/22 (59.09%) patients expired whereas it was grade B in 38/60 (63.33%) patients of which 1/38 (0.031%) patient expired. Mortality was significantly lower in patients classified as Child Pugh grade B as compared to Child Pugh grade C.



MELD Na score and mortality (graph 8)

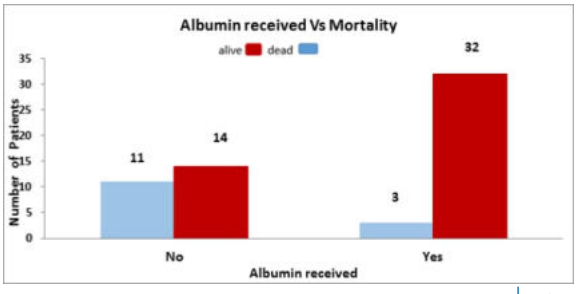
MELD Na score was > 30 in 19/60 (31.67%) of all patients. MELD Na score was < 30 in 41/60 (68.33%) of all patients. Mortality was significantly lower in patients with MELD Na score < 30 as compared to patients with score > 30.



Albumin received and mortality (graph 9)

25/60 (41.67%) patients did not receive albumin as compared to 35/60 (58.33%).

Mortality was statistically lower in patients who received albumin as compared to patients who did not receive albumin.



Aetiology

Literature shows that in India most cases of cirrhosis are due to alcohol and viral hepatitis B and C. Alcohol was the predominant aetiology of liver cirrhosis in our study (68.33%) followed by post necrotic liver cirrhosis (25%). This was in concordance with similar other studies conducted by Kavita Paul et al and Nakul Kadam et al.

Symptoms and signs:

It is a well-known fact that most patients present late i.e when decompensated. Great variation in symptoms and signs has been reported in different studies. In our study apart from abdominal distension, abdominal pain (53%), swelling in b/l feet (53%), fever (45%), jaundice (30%), altered sensorium (21%) and GI bleeding (9%) were the common presenting complaints. Syed VA et al., in his study found that the most common presenting symptoms were UGI bleeding (75%) followed by pain in abdomen (65%). Minhas et al., reported fever (54%), pain in abdomen (57%) as the common presenting complaints.

Mortality

Our study revealed that variables such as Child Pugh Grade C, MELD Na score > 30, anemia, leukocytosis, coagulopathy or deranged PT INR, hyperbilirubinemia, hyponatremia, AKI, hepatic encephalopathy, refractory Ascites, hepatorenal Syndrome and absence of albumin transfusion during hospitalization were significantly associated with mortality.

Conclusion

Cirrhosis of liver is a major health problem in India and affects males in the most productive years. Alcohol abuse is the major cause of cirrhosis in India that is entirely preventable through proper education and legislation. Patients present in advanced stage of disease with complications. However, limitation of the study was that patients were not followed up for long and the effect of the various treatment options were not recorded.

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