SUBACUTE HEPATIC FAILURE- A CLINICAL PROFILE

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ABSTRACT

A combined experience of 37 cases of subacute hepatic failure encountered in five major gastroenterology centres over a period of ten years is discussed. Majority (65%) were males with average age of 38 years. Maximum (54%) were in 5th decade. Jaundice (100%), abdominal distention (38.7%), swelling feet (64%), fever (54%), abdominal pain (54%), exhaustion (78.3%) were the major presenting features. Jaundice and ascites were present in all cases. Pedal oedema (78.3%), hepatomegaly (54%), splenomegaly (32.4%) and encephalopathy (27%) were the other important clinical features. Hypoalbuminemia and prolonged prothrombin time were significant laboratory findings in addition to hyperbilirubinemia and elevated ALT and AST. Hbs Ag was detected in 46%. Major complications encountered were renal failure (48.7%), spontaneous bacterial peritonitis (43.2%), other infections (43.2%), encephalopathy (43.2%) and upper gastrointestinal bleed (22%). 54% died during stay in hospital. To conclude subacute hepatic failure is potentially fatal condition.

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KEY WORDS : Subacute hepatic failure; HBsAg, Jaundice.

Introduction

Subacute hepatic failure (SAHF) is now recognized as distinct disease with characteristics of progressive hepatic failure defined by clinical and biochemical criteria [1]. There has been lot of controversy regarding the time frame by which hepatic failure occurs as well as clinical features particularly presence of encephalopathy [2]. Acute liver failure is characterized by onset of encephalopathy within 8 weeks of appearance of jaundice where as chronic liver failure is defined as hepatic decompensation occurring 24 weeks after the onset of liver disease. SAHF is now characterized by gradual deterioration of hepatic function between 8 and 24 weeks of onset of jaundice.

Second controversy is regarding inclusion of encephalopathy in diagnostic criteria. As the encephalopathy was noted in only 16% of their cases by Tandon et al [1] and that also as a preterminal event they have removed it as a diagnostic criteria. Hence at present SAHF is taken as hepatic decompensation in the form of continuing hepatocellular dysfunction and development of ascites between 8 and 24 weeks from onset of jaundice without previous history of liver disease. Based on these criteria a combined experience of 37 cases of SAHF encountered in five major gastroenterology centres of Armed Forces is discussed.

Material and Methods

The study pertains to 37 cases of SAHF. All the patients satisfied the diagnostic criteria as mentioned above. None of these had past history of liver disease. Detailed history and physical examination findings were recorded. Patients with pre-existing liver disease, alcohol intake, chronic renal failure, sonographic evidence of extrahepatic biliary obstruction and endoscopic evidence of oesophageal varices were excluded from the study. Investigations consisted of complete blood count, blood sugar, urea, creatinine, ALT, AST, alkaline phosphatase, total proteins, albumin, prothrombin time and Hbs Ag. Patients were also subjected to skiagram of chest, ultrasonography abdomen and upper gastrointestinal endoscopy. Ascitic fluid was collected for physical, biochemical and bacteriological examination. The patients were treated conservatively and symptomatically. All these patients were followed up every month for one year.

Results

There were 24 (64.8%) males and 13 (35.2%) females in present study with an average age of 37 years. However maximum number of patients were from 5th decade (54%). Clinical features of these patients are shown in Table 1. Jaundice and ascites were present in all cases being the mandatory criteria for diagnosis. Prodromal symptoms of acute hepatitis like severe anorexia were present in 83.7% of cases. UGI bleed was not a major presenting feature and present only in 16.2% of patients. All these patients had erosive gastritis on endoscopy. Extreme degree of fatigue and exhaustion was another prominent presenting feature present in 70.2% of patients. Cholestatic features in the form of itching was present in 35.1% of cases. Ascitis was accompanied by pedal oedema in 78.3% of patients. Splenomegaly was not a very common finding (32.4%) whereas hepatomegaly by clinical exam/ultrasonography was detected in 54%. The patients who had splenomegaly did not have oesophageal/gastric varices on upper gastrointestinal endoscopy. Again encephalopathy which has been

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considered as an essential criteria for diagnosis of SAHF by some workers was present at the onset in only 27% of cases in our study. This encephalopathy was grade I to II. Preterminally 43.2% had encephalopathy.

TABLE 1
Clinical features in 37 cases of subacute hepatic failure

Feature	Number	Percentage
Jaundice	37	100
Ascitis	37	100
Pedal oedema	29	78.3
Hepatomegaly	20	54
Splenomegaly	12	32.4
Encephalopathy	10	27
Spider naevi	6	16.2
Exhaustion	26	70.2
Anorexia	31	83.7
Gastrointestinal bleed	6	16.2
Abdominal pain	20	54
Itching	13	35.1

Biochemical profile of these patients is depicted in Table 2. Severe hepatic damage as shown by hyperbilirubinemia, raised ALT, AST, hypoalbuminemia and prolonged prothrombin time were significant findings. Alkaline phosphatase was only marginally elevated. 48.7% patients had biochemical evidence of renal failure with mean urea of 96 mg/dl and serum creatinine of 3.4 mg/dl, Hbs Ag was detected in 46% patients. Other viral markers were not studied. Liver biopsy was not performed in any patient.

TABLE 2
Biochemical investigations in 37 cases of subacute hepatic failure

Investigation	Value
Serum bilirubin	13.7 ± 11.3 mg/dl
AST	271.2 ± 111.4 IU/L
ALT	309.7 ± 189.6 IU/L
Alkaline phosphatase	19.2 ± 13.0 KA units/dl
Serum proteins	6.1 ± 0.9 G/dl
Serum albumin	2.9 ± 1.3 G/dl
Blood urea	96.1 ± 69.6 mg/dl
Creatinine	3.4 ± 1.4 mg/dl
Prothrombin time (seconds more than control)	8.6 ± 5.3
HBsAg positive	17 (46%)

Major complications (Table 3) encountered were renal failure and infections occurring in 48.7% and 86.4% of cases. Majority of infections were in the form of respiratory infections and spontaneous bacterial peritonitis. Non variceal GI bleeding and encephalopathy occurred in 21.6% and 43.2% of patients respectively. All patients were treated conservatively and symptomatically. 54% of patient died during hospital stay. Only 16.3% were alive on follow up where as 29.7% were lost to follow up.

Discussion

Fulminant hepatic failure and chronic hepatic failure are well established and defined entities. Subacute hepatic failure has now come to be recognized as separate entity. Presently continuing hepatocellular damage (clinical and biochemical) persisting after 8

TABLE 3
Complications and outcome in hospital of 37 cases of subacute hepatic failure

Complication	Number	Percentage
Renal failure	18	48.7
Spontaneous bacterial peritonitis	16	43.2
Respiratory infections	16	43.2
GI Bleed	8	21.6
Encephalopathy	16	43.2
Death in hospital	20	54

weeks of onset of jaundice and development of ascitis after 8 weeks but before 24 weeks of onset of jaundice are taken as the diagnostic criteria. However there should be no past history of liver disease. The presence of encephalopathy is not considered essential for the diagnosis. We have taken diagnostic criteria suggested by Delhi group for the present study [1].

Majority of our cases belonged to 5th and 6th decade. This fact and other clinical features as found in our study are similar to other studies [1,3,4]. Liver function tests indicated severe hepatocellular damage as evidenced by hypoalbuminemia and prolonged prothrombin time. In spite of this bleeding manifestations were not prominent. Virus B viral hepatitis was the major aetiological cause of SAHF. As the other viral markers were not studied we are not sure how much virus A, C, D or E have contributed to SAHF. There was no history of intake of hepatotoxic drugs in any case.

Mortality due to this condition is very high. In our study in hospital mortality was 54%. As many as 29.7% of our cases were lost to follow up. Mohapatra et al [4] have noted high mortality to the tune of 84.6% in their cases. Of the various complications infection and renal failure were the most prominent. The infective complications were much more common in our study as compared to Mohapatra's study [4]. To conclude, the SAHF is a distinct but rare entity, viral hepatitis is the most important cause. Mortality due to this condition is high and there is no satisfactory form of treatment available.

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