Clinico-pathological profile of children with liver disease in Uttaranchal

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Abstract

The present study was conducted to find out the etiology and clinical profile of children with liver disease as seen at a tertiary care teaching hospital in Uttaranchal. The study included 117 patients with ages ranging between 1.5 months to 18 years. Loss of appetite was found to be the predominant presenting feature (95.7%) followed by nausea and vomiting (90.6%). Twenty four (20.5%) cases had cirrhosis of liver with portal hypertension. Acute hepatitis was seen in 28 (23.9%) cases. Fifteen (12.8%) cases had hepatic failure out of whom 9 died. Chronic hepatitis was present in 11(9.4%) cases. On the whole hepatotropic viruses were responsible for 43 (36.8%) cases of liver disease. A total of 13 (11.1%) deaths occurred. Hepatic failure with encephalopathy was the leading cause of death.

Introduction

Uttaranchal is a state where medical facilities are often not easily accessible to a large part of the population because of the difficult hilly terrain. Moreover, clinicians often do not recognize the presence of underlying liver disease delaying precise documentation of the disorder which can lead to subsequent delay in the initiation of effective therapies.

Currently there are no available data on prevalence and profile of liver disease in this region. The present study was, therefore, conducted to find out the etiology and clinical profile of children with liver disease as seen at a tertiary care teaching hospital in Uttaranchal.

Material and Methods

The study was conducted prospectively and retrospectively on children with liver disease admitted in the Pediatric ward of a tertiary care teaching hospital between January 1998 to September 2002. Serological markers for viral hepatitis had been sent where indicated. All the patients had been subjected to liver biopsy or histo-pathological examination of resected specimen.

Results

The study included 117 patients with ages ranging between 1.5 months to 18 years (Table- I). Mean age of presentation was 8.59 ± 3.99 years. There was an obvious male preponderance, the male female ratio being1.34:1. Loss of appetite was observed to be the predominant presenting feature (95.7% cases) and was followed by nausea and vomiting (90.6% cases), hepatomegaly (75.2% cases), icterus (53.9% cases), abdominal pain (51.3% cases),

fever (45.3% cases), gastro-intestinal bleed (34.2% cases), ascites (24.8% cases), varices (24.8% cases), pruritus (15.4% cases), and altered sensorium (8.6% cases).

Histo-pathological diagnosis of cases is shown in Table-II. Twenty four (20.5) cases had cirrhosis of liver with portal hypertension. Acute hepatitis was seen in 28 (23.9) cases. All patients of hepatitis had icterus and elevated transaminases. Serological evidence of hepatitis A infection was present in 10 (35.7%), hepatitis B in 6 (21.4%), hepatitis E in 3 (10.7%), hepatitis C in 2 (7.1%), and both hepatitis A and B in 2 (7.1%) patients with acute hepatitis. In the remaining 5 cases no viral marker was detected. Fifteen patients had hepatic failure (of whom 9 died). All these patients had icterus, 10 (66.7%) had encephalopathy, 6 (40.0%) had gastrointestinal hemorrhage, and 3 (20.0%) had ascites. Of the patients of hepatic failure, 3 (20.0%) had serological evidence of hepatitis B alone, 4 (26.7%) of hepatitis A, 3 (20.0%) of both hepatitis B and D, and 2 (13.3%) of hepatitis E. The remaining 3 cases had no positive markers. Liver biopsy showed evidence of submassive to massive necrosis. Elevated bilirubin was seen in all these cases while elevation of transaminases was present in 10 (66.7%) cases. Chronic hepatitis was present in 11 patients, 4 (36.4%) of whom had icterus, 2 (18.2%) had ascites and 1 (9.1%) had gastro-intestinal hemorrhage. Five (45.5%) of them tested positive for hepatitis B and 3 (27.3%) for hepatitis C. Six (54.6%) of these patients had elevated bilirubin while 8 (72.7%) had elevated transaminases.

On the whole hepatotropic viruses were responsible for 43 (36.8%) cases, hepatitis B for 19 (16.2%) cases and hepatitis

Table: Age and Sex Distribution of Patients

Age-Group (yrs.)	Male (%)	Female (%)
0 – 3	06 (09.0)	04 (08.0)
3 – 6	11 (16.4)	06 (12.0)
6 – 9	13 (19.4)	14 (28.0)
9 – 12	22 (32.8)	18 (36.0)
12 -15	11 (16.4)	08 (16.0)
15 -18	04 (06.0)	00 (00.0)
Total	67	50

Table 2: Histo-pathological diagnosis of cases

Pathology	Number of cases(%)
Acute hepatitis	28 (23.9%)
Cirrhosis	24 (20.5%)
Submassive-massive necrosis	15 (12.8%)
Chronic hepatitis	11 (09.4%)
Hydatid didease	8 (06.8%)
Wilson’s disease	5 (04.3%)
Biliary aresia	5 (04.3%)

Liver abscess	4 (03.4%)
Tubercular granulomas	4 (03.4%)
Glycogen storage disease	4(02.6%)
Lipid storage disease	3 (03.4%)
Fatty change	3 (03.4%)
Leishmaniasis	2 (01.7%)
Biliary hamartoma	1 (00.9%)

A for 16 (13.7%) cases of liver disease. Hydatid disease accounted for 6.8% cases. Four patients had liver abscess, (2 each were due to amoebic and pyaemic infection).

A total of 13 (11.1%) deaths occurred. Hepatic failure with encephalopathy was the leading cause of death (9 cases). One each of the remaining 4 deaths occurred due to extra-hepatic portal hypertension with cavernomatous malformation, chronic hepatitis with portal hypertension and hypersplenism, Wilson's disease, and disseminated tuberculosis.

Discussion

Anorexia, fatigue and scleral icterus, are the common presenting features of liver disorders. Cholestasis may lead to complaints of pruritus and particularly dark and foamy urine. The alteration in colour is due to choleuria (bile pigment in urine) and foaminess is due to presence of bile salts. Bile salts are detergent molecules that lower the superficial tension of solutions causing visible foaminess. Most common physical findings are hepatomegaly and icterus. Causes of liver disease include hepatitis (viral, autoimmune, toxic, pharmacologic), liver disease associated with chronic inflammatory bowel disease, sclerosing cholangitis, parasitic infections, toxins and pharmacologic remedies, malignancies, Wilson's disease, fatty liver, occlusion of hepatic veins etc. [1]. In the present study a considerable bulk (36.8% cases) of liver disease could be attributed to viral infections.

In an Indian study only 21.6% of children with chronic hepatitis were symptomatic, with icterus observed in 12%, features of decompensation such as ascites in 7% and gastrointestinal bleed in 5%. A high index for suspicion is therefore needed. Elevated alanine aminotrans-ferase (ALT) was observed in 76% of subjects. Seventy-four percent of the mothers had evidence of past or present HBV infection i.e., almost two-thirds of the children might have acquired their HBV infection perinataly [2] In the present study, however, a higher percentage of cases of chronic hepatitis had detectable icterus(36.4%) and ascites (18.2%).

In another Indian study on 54 children with acute viral hepatitis, 59.3% patients were found to have Hepatitis A, 33.3% Non A Non B, 3.7% Hepatitis B and 3.7% concurrent Hepatitis A and B infection. It was not possible to distinguish the etiological agents on the basis of the clinical and biochemical profile. Fulminant hepatitis was documented in 8 (14.8%) cases [3]. In the present study hepatitis A infection was present in 10 (35.7%), hepatitis B in 6 (21.4%), hepatitis E in 3 (10.7%), hepatitis C in 2 (7.1%), and both hepatitis A and B in 2 (7.1%) patients with acute hepatitis.

In southern Bengal, hepatotropic viruses were reported to be the predominant cause of acute liver failure in children. Of these, the hepatitis viruses A and E transmitted via the enteric route dominated (24 of 30 cases). Prodrome, decreased liver span, ascites, cerebral oedema, coagulopathy, renal failure, spontaneous bacterial peritonitis, signs and symptoms of clinical sepsis (corroborated by laboratory data), severe hypoalbuminaemia and electrolyte imbalance were significantly more in patients who died. The mean age, prothrombin time, serum bilirubin level and stage of encephalopathy differed significantly between survivors and non-survivors [4]. A study was conducted in a tertiary care centre in Northern India to document the demographic and clinical characteristics, natural course, and causative profile of patients with fulminant hepatic failure. In 62% of patients the serological evidence of HAV, HBV, or HDV infection was lacking, and none of them had ingested hepatotoxins. FHF was presumed to be caused by non-

A, non-B virus(es) infection. Sixty six% patients died, and approximately 75% of those who died did so within 72 hours of hospitalization [5].

Timely prevention, early detection and treatment can go a long way in reducing the morbidity and mortality due to liver disease. Vaccination against Hepatitis B and A can help in reducing the burden of liver disease considerably.

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