ORIGINAL ARTICLE

COMPARING CLINICAL DIAGNOSIS AND LABORATORY DIAGNOSIS IN PAEDIATRIC PATIENTS OF ACUTE VIRAL HEPATITIS IN AHMEDABAD, GUJARAT

Ekta A Dalal[1], Gaurav Vishal[2]

Author's Affiliations: [1]Associate Professor Department of Pediatrics, GMERS Medical College Gandhinagar, Gujarat; [2]Consultant Pediatrician, Gujarat
Correspondence: Dr. Ekta A Dalal Email: drektadalal@gmail.com

ABSTRACT

Introduction: During the last 30 years, Paediatric hepatology has been transformed by advances in our understanding and management of a wide spectrum of liver diseases. Globally however, viral hepatitis remains one of the most important causes of liver disease in children. Typical symptoms of acute hepatitis are fatigue, anorexia, nausea, and vomiting. Very high aminotransferase values (>1000 U/L) and hyperbilirubinemia are often observed. The objective of the study was to compare the clinical diagnosis and laboratory diagnosis.
Methodology: The current study was conducted in Paediatric department of tertiary care hospital of Ahmedabad, Gujarat, India. It was a record based cross sectional study. Complete data of basic socio demographic profile, symptoms, signs, all laboratory investigations, outcome of patients were obtained and analysed.
Results: There were total 150 cases of symptoms of acute viral hepatitis fulfilling inclusion criteria in Paediatric department. It was observed that first rise in cases was seen in the month of June and another peak was seen in the month of September. After September, there was rapid fall in total number of cases. Yellow sclera (94.67%) and yellow colour urine (93.33%) were most common symptoms reported by patients, followed by fever (88.67%) & loss of appetite (73.33%). Icterus (94.67%) and Subcostal Tenderness (66.67%) were most common sign.
Conclusion: Clinical diagnosis corresponds laboratory investigations and whenever laboratory facility is not accessible, further management should be started at the earliest as per clinical diagnosis of viral hepatitis in children to prevent mortality. Majority of the patients had bilirubin below 10 mg/dl 126 patients (84%), while 12 (8%), patients had bilirubin up to 15, 7 (4.67%) between 15-20 mg/dl. Mean Serum bilirubin was 7.28 mg/dl.

Keywords: Hepatitis, SGPT, S. Bilirubin, Icterus

INTRODUCTION

During the last 30 years, Paediatric hepatology has been transformed by advances in our understanding and management of a wide spectrum of liver diseases. Globally however, viral hepatitis remains one of the most important causes of liver disease in children. Major advances in this field include the implementation of hepatitis B virus (HBV) and hepatitis A virus (HAV) vaccination, definition of the immunologic phases of HBV infection, and the development of treatments for chronic HBV infection. Advances in virology, epidemiology, and therapeutics have led to considerable progress in understanding and managing hepatitis infection in childhood.[1,2,3]

In developing countries, where routine HAV vaccination is not available, HAV infection is still common and is a leading cause of acute liver failure (ALF). This is in stark contrast to developed countries where the infection risk is now low. HBV infection remains a global public health problem. Although vaccination has reduced the prevalence of chronic hepatitis B infection in many countries, it is still high in areas such as Western Africa and Asia. Human immunodeficiency virus (HIV) coinfection has added to the complexity of managing these children. Although less common than HAV and HBV infection, increasing numbers of children with HCV are presenting for treatment.[4] Viral pathogens are the main cause of hepatitis in Asian children. Of these viruses, HBV infection remains a major health hazard in Asia because of its high prevalence and the severe complications of chronic infection. This problem persists in children even in the era of HBV vaccination.

HAV (South and South-East Asia) and HEV (Central and South-East Asia) are endemic in parts of Asia. HAV and HEV are transmitted through the fecal-oral route and transmission is significantly associated with poor hygiene.[5,6] HAV and HEV infections are generally mild and self-limiting diseases in immune-competent children, although rarely ALF may occur. Typical symptoms of acute hepatitis are fatigue, anorexia, nausea, and vomiting. Very high aminotransferase values

(>1000 U/L) and hyperbilirubinemia are often observed. Severe cases of acute hepatitis may progress rapidly to acute liver failure, marked by poor hepatic synthetic function.[7] Acute liver injury caused by the hepatotropic viruses manifests in 3 main functional liver biochemical profiles. These serve as an important guide to supportive care and monitoring in the acute phase of the infection for all viruses. As a reflection of *cytopathic injury* to the hepatocytes, there is a rise in serum levels of alanine aminotransferase (ALT) and aspartate aminotransferase (AST). Rapidly falling aminotransferase levels can predict a poor outcome, particularly if their decline occurs in conjunction with a rising bilirubin level and a prolonged prothrombin time; this combination of findings usually indicates that massive hepatic injury has occurred.

Cholestasis, defined by elevated serum conjugated bilirubin levels, results from abnormal bile flow at the canalicular and cellular level due to hepatocyte damage and inflammatory mediators. Elevation of serum alkaline phosphatase (ALP), 5'-nucleotidase, γ-glutamyl transpeptidase (GGT), and urobilinogen all mark cholestasis. Improvement tends to parallel the acute hepatitis phase. The most important marker of liver injury is altered *synthetic function*. Abnormal liver synthetic function is a marker of liver failure and is an indication for prompt referral to a transplant center. Serial assessment is necessary because liver dysfunction does not progress linearly. Synthetic dysfunction is reflected by a combination of abnormal protein synthesis (prolonged prothrombin time, high international normalized ratio [INR], low serum albumin levels), metabolic disturbances (hypoglycemia, lactic acidosis, hyperammonemia), poor clearance of medications dependent on liver function, and altered sensorium with increased deep tendon reflexes. The objective of the study was to compare the clinical diagnosis and laboratory diagnosis.

METHODOLOGY

The current study was conducted in Paediatric department of tertiary care hospital of Ahmedabad, Gujarat, India. It was a record based cross sectional study. Records of all patients admitted in paediatric ward during the study period of October 2011 to September 2013 with symptoms of acute viral hepatitis, not having chronic hepatitis, obstructive jaundice, portal hypertension, underlying liver disease, poisoning, toxin or drug induced hepatitis and patients with complete data were enrolled in the study.

Patients age less than one month or patients with incomplete data were excluded from the study. Records of Patients having incomplete data were excluded.

Permission of Institutional Ethical Committee and hospital administration were taken to obtain the data and conduct the study. Confidentiality of data was maintained at all level.

Complete data of basic socio demographic profile, symptoms, signs, all laboratory investigations, outcome of patients were obtained.

Data analysis: Data was entered in Microsoft excel and analyse in excel itself.

RESULTS

There were total 150 cases of symptoms of acute viral hepatitis fulfilling inclusion criteria in Paediatric department.

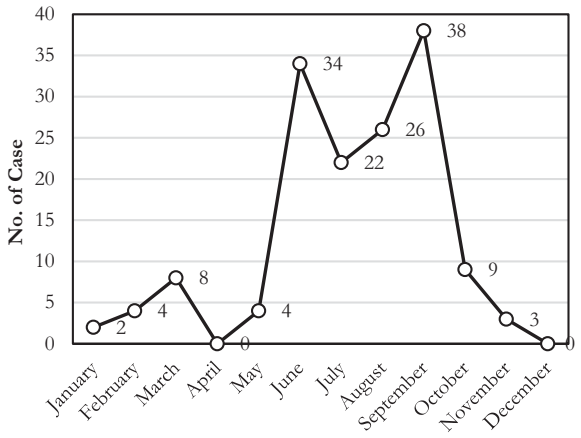

Figure 1: Trend of Hepatitis infection

Figure 1 shows trend of Hepatitis infection during whole year. It was observed that first rise in cases was seen in the month of June and another peak was seen in the month of September. After September, there was rapid fall in total number of cases.

Yellow sclera (94.67%) and yellow colour urine (93.33%) were most common symptoms reported by patients, followed by fever (88.67%) & loss of appetite (73.33%). Icterus (94.67%) and Subcostal Tenderness (66.67%) were most common sign.

Table 1 shows that Yellow sclera, yellow urine, Fever, Loss of Appetite and Nausea/Vomiting were common symptoms. It was observed that out of 150 study participants, 142 (94.67%) of patients were having yellow sclera, 140 (93.33%) were having yellow sclera and 133 (88.67%) were having fever. Table also shows that Icterus and subcostal tenderness were most common signs in study participants.

Table 1: Sign and Symptoms wise distribution of study participants (N=150)

Symptoms	No. (%)
Loss of Appetite	110 (73.33)

Nausea/Vomiting	90 (60.0)
Abdominal Pain	78 (52.0)
Abdominal Distension	42 (28.0)
Yellow Sclera	142 (94.67)
Yellow Urine	140 (93.33)
Fever	133 (88.67)
Seizures	12 (8.0)
Signs	**No. (%)**
Icterus	142 (94.67)
Subcostal Tenderness	100 (66.67)
GI Haemorrage	46 (30.67)
Splenomegaly	24 (16.0)
Ascites	22 (14.67)
Pruritus	22 (14.67)

Majority of the patients had bilirubin below 10 mg/dl 126 patients (84%), while 12 (8%), patients had bilirubin up to 15, 7 (4.67%) between 15-20 mg/dl. Mean Serum bilirubin was 7.28 mg/dl. Considering SGPT, total 15 (10%) of patients were having SGPT of 0-500 IU/L. Total 22 (14.7%) and 48 (32%) patients were having SGPT of 1001 to 1500 IU/L and 1501 to 2000 IU/L.

Table 3 shows that maximum number of deaths occur due to Hepatitis A and children less than 5 years of age were major culprit.

Table 2: Important Laboratory investigations of study participants

Lab Investigations	No. (%)
S. Bilirubin (mg/dl)	
0-5	66 (44.0)
5.1-10	60 (40.0)
10.1-15	12 (8.0)
15.1-20	7 (4.7)
20.1-25	3 (2.0)
>25	2 (1.3)
SGPT (IU/L)	
0-500	15 (10.0)
501-1000	18 (12.0)
1001-1500	22 (14.7)
1501-2000	48 (32.0)
2001-2500	28 (18.7)
2501-3000	10 (6.7)
3001-3500	2 (1.3)
3501-4000	3 (2)
>4000	4 (2.7)

Table 3: Incidence of fulminant hepatic failure in relation to age distribution and mortality

Age Group	Hepatitis A (%)	Hepatitis E (%)	Hepatitis A & E (%)	Hepatitis B (%)	Other (%)	Total (%)
< 5	54 (85.71)	4 (6.35)	2 (3.17)	3 (4.76)	4 (6.35)	63 (100)
5-10	28 (58.33)	12 (25.0)	6 (12.50)	2 (4.17)	3 (6.25)	48 (100)
10 – 16	6 (20.00)	20 (66.67)	4 (13.33)	0	2 (6.67)	30 (100)

DISCUSSION

There were total 150 cases admitted in the hospital during the study period. Trend of Hepatitis infection during whole year shows two peaks first in June and another September. After September, there was rapid fall in total number of cases.

Based on data from 28 studies, irregular, cyclical patterns were observed for acute viral hepatitis, with most prominent peaks were shown in spring and summer for hepatitis A; B; C and E in some of the countries subjects. These findings have been explained by some authors as due to summer travel to an endemic area, swimming habits of the population in hot months, increase sexual contact, tattoo, poor hygiene and environmental sanitation, and food habits (feco-oral transmission of viral hepatitis).[8,9]

HAV and HEV are communicable by way of the fecal-oral. On the other hand, HBV, HCV are contagious by blood and blood products, also may be transmitted by sexual intercourse and household exposure to an infected contact, exposure to multiple partners and perinatal exposure, particularly for hepatitis C, but the efficiency of transmission in these settings appears to be low.

Hepatitis A virus is an extremely stable virus and can survive for 12 weeks to 10 months in water.[10,11] This stability accounts for the frequent occurrence of waterborne and shellfish-transmitted outbreaks.[12,13] In this regard, the virus is relatively resistant to heat or chemical inactivation and this situation allow the dissemination of HAV infection. Therefore, disruption of sanitation and water supplies was the most likely contributing factor for the seasonal occurrence of hepatitis A and E.

In our study it was observed that in all patients having two or more symptoms and signs suggestive of Hepatitis, laboratory markers are raised considerably. Clinical management of Hepatitis is simple if started early. Early treatment also halts the progression of disease and prevents further complication. Thus, whenever laboratory facility is not accessible, further management should be started at the earliest as per clinical diagnosis of viral hepatitis in children to prevent complications and mortality.

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