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RESEARCH ARTICLE

CLINICAL PROFILE OF ACUTE VIRAL HEPATITIS IN A TERTIARY CARE HOSPITAL OF ODISHA

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ABSTRACT

Objectives: To describe the etiological and clinical profile of viral hepatitis and its outcome in a tertiary care hospital of Odisha.
Methods: 71 children with jaundice were selected from dept. of Paediatrics, SCB MCH, Cuttack. Clinical examination, LFT & tests of viral markers of these children were done.
Results: Most common age group of presentation was 5-10 years. Most of the children were male (61%). Majority (71%) were from rural areas. The most common signs & symptoms were jaundice (97.1%), fever (88.7%), nausea & vomiting (92,9%), splenomegaly (47.8%), ascites (19.7%), encephalopathy (22.7%) & GI bleed (9.8%). The common cause of viral hepatitis were hepatitis A (67.6%), hepatitis B (11.2%), hepatitis C(1.4%), hepatitis E (4.2%). No marker was detected in 14% of cases. The highest value of bilirubin total & direct were 44.6mg%, 13.6mg% respectively in the cohort. SGPT level was more than four times in all cases with highest 9350IU/L. PT was prolonged in 26.8% cases. 20(28.1%) cases which developed fulminant hepatic failure (FHF) were admitted to ICU out of which 9 patients recovered and 11 patients (16.71) died. Out of the 11 patients who died 9 patients (75%) had no viral marker. FHF due to hepatitis A singly is more common & 2 cases of Sickle cell disease with hepatitis A developed FHF more rapidly. 2 cases of hepatitis E developed FHF & recovered completely.
Conclusion:-There is need of hepatitis A vaccination to young children, which will reduce the risk of infection in adolescents and adults through a herd immunity effect. Hepatitis B can be prevented by Universal immunization. Hepatitis C can be prevented by proper screening of blood and blood Products.

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INTRODUCTION

Acute viral hepatitis is a systemic infection affecting the liver predominantly. It continues to be a common medical problem in both developing & developed countries. This disorder is caused by at least five pathogenic hepatotropic viruses recognized to date that is Hepatitis A, B, C, D, E (HAV, HBV, HCV, HDV and HEV respectively). Hepatitis G (GBV) and transfusion transmissible virus (TTV) often infect liver as a co-infection with another hepatotrophic virus and may produce acute or chronic viraemia but rarely produce hepatocellular injury on their own (Kliegman, 2008). More than 90% patients with acute viral hepatitis recover without any hepatic dysfunction (Sherlock, 2002).

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Nearly 1-2% of cases progress to chronic hepatitis (HBV, HCV) leading to chronic liver failure, 6 months after the icteric phase of acute viral hepatitis. 1-2% of cases of viral hepatitis develop massive liver cell necrosis resulting in fulminant hepatic failure. There is a paucity of studies on acute viral hepatitis in children. There is a need to understand the aetiology, clinical manifeststions, complications and outcome of acute viral hepatitis

The study was conducted with the following objectives

- To determine the etiological profile of acute viral hepatitis in patients attending the paediatric department of SCB medical college Odisha
- To study the different clinical manifestations, different complications & outcome of acute viral hepatitis

MATERIALS AND METHODS

71 children with jaundice were selected from the indoor pediatrics department of SCB MCH and SVPPGIP, Cuttack over a period of 3 years. The cases were diagnosed on the basis of meticulous clinical history and examination, relevant routine as well as special liver function test and test for viral markers in all selected cases. Exclusion criteria were family history of liver diseases, malarial hepatopathy, hepatitis due to toxins, hemolytic anaemia, leptospira cases, obstructive jaundice, neonatal jaundice(viral markers negative) and physiological jaundice. *Investigation like LFT, CBC, PT, LIVER VIRAL MARKERS, USG of abdomen were done.*

RESULTS

Most common age group of presentation was 5-10 years. Most of the children in study were males (61%) & the females constituted only 38%. Male to female ratio is 1.6:1. Out of 71 patients majority (71%) were from rural area. The most common symptoms were Jaundice (97.1%), fever (88.7%), nausea & vomiting (92.9%), anorexia(94.3%) and myalgia (87.3%). (Fig. 1). Common signs of viral hepatitis were hepatomegaly (95.7%), splenomegaly (47.8%), ascitis(19.7%), encephalopathy (22.7%) & G.I. bleed (9.8%) (Table II).

Table 1. Showing Outcome of different types of Hepatitis

Viral hepatitis	total	FHF	death	Complete recovery	Chronic hepatitis
HAV	49	7	2	47	0
HBV	8	2	1	3	4
HCV (thalasemia)	1	0	0	0	1
HEV	3	2	0	3	0
Non(A-E)	10	9	9	1	0

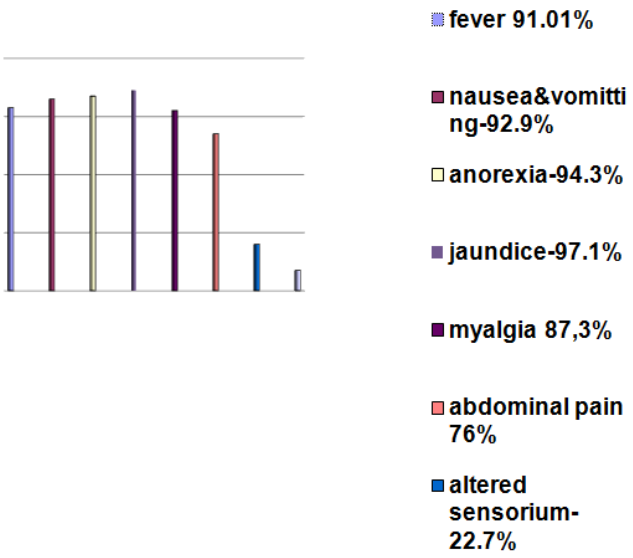

Figure 1. (Clinical symtoms)

The common virus causing viral hepatitis were Hepatitis A(HAV-67.6%), Hepatitis B(HBV-11.2%), Hepatitis C(HCV-1.4%) and hepatitis E(4.2%). No markers were found in 14% cases. The highest value of bilirubin total & direct seen in the series were 44.6mg%, 13.6mg% in the same patient. The highest SGPT value was 9350 IU/l. PT was prolonged in 26.8%. All patients got supportive treatment. 20(28.1%) cases developed fulminant hepatic failure (FHF) and were admitted to ICU out of which 9 patients recovered and 11 patients (16.71) died.

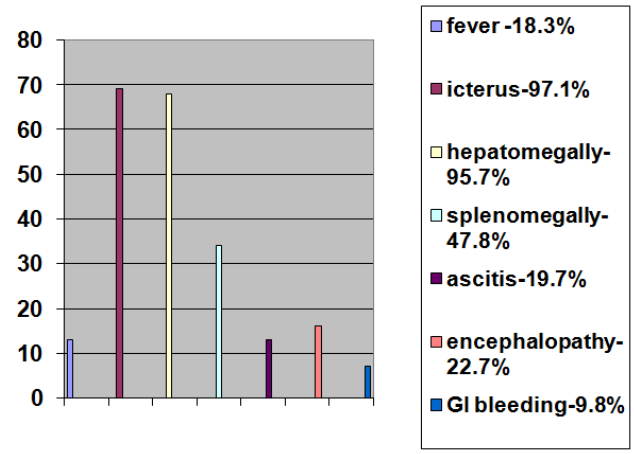

Figure 2. Showing Clinical Signs

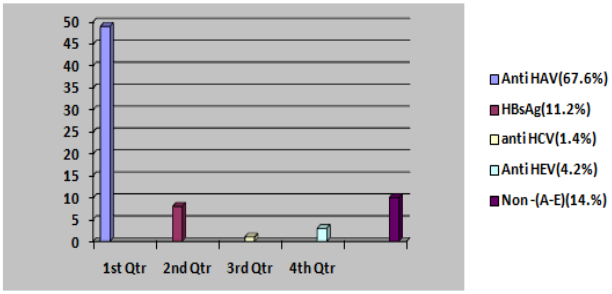

Figure 3. Showing Viral Markers

Out of total death 9 patients (75%) had no viral marker. FHF due to hepatitis A singly is more common & 2 cases of Sickle cell disease with hepatitis A developed Fulminant Hepatic Failure more rapidly. 2 cases of hepatitis E developed Fulminant Hepatic Failure & recovered completely. In 2 patients of hepatitis A infection having hepatic encephalopathy, exchange transfusion was done, one improved and the other died.

DISCUSSION

Most common infection in our study was due to HAV infection (http://www.merck.com/mmpe/sec03/ch027/ch027b.html). We had more cases coming from rural areas, as most of the urban patients are coming early & are being treated on OPD basis, and people are becoming conscious about vaccination & sanitation. Most of the cases are male, as males are given more importance than females. The most common age group of presentation in hepatitis A was between 5-10 years, probably due to their eating habits from unhygienic roadside places. It is more commonly seen in lower socioeconomic communities especially in rural area where sanitation and hygienic conditions are poor (Battegay, Battegay, 1995). HBV infection was found in newborns of mothers infected with HBV (HBeAg positive). Similarly one study showed that the risk of perinatal or vertical transmission is highest (90%) with HBeAg positive mother, whereas infants of mothers who are HBsAg positive but HBeAg negative have a lower risk(10-15%)[5]. Almost 90% of neonates exposed to the virus become chronic carrier[6]. We had one patient of thalassemia having HCV infection, due to frequent blood transfusion which developed in to chronic

hepatitis. According to Lai *et al*. (Lai, 1993), no of thalassemic patients acquiring HCV through tranfusion was as high as 60%, among them recovery was seen in 20% cases, chronic hepatitis in 80% cases and cirrhosis in 11% cases. We had 3 cases of HEV infection. 10 cases had no viral markers, may be due to non A-E hepatitis. Upto 50% of cases of fulminant hepatitis B involve HDV co-infection. Fulminant hepatitis with HAV is rare, but may be more likely in people with preexisting liver disorders (http://www.merck.com/mmpe/sec03/ch027/ ch027b. html). Fulminant Hepatic Failure due to HAV is more common where there was combined infection with HAV and HEV (Arora, 1996). We found 2 cases of hepatitis A with sickle cell disease (SCD) developing fulminant hepatic failure. One case died within 24 hours of admission. Another case underwent double volume exchange transfusion and survived. So SCD may be a risk factor in development of Fulminant Hepatic Failure due to HAV infection (Renge, 2002). But in our centre we found cases of hepatitis A singly, not associated with other hepatitis or preexisting liver disorder developing Fulminant Hepatic Failure. Mortality from hepatitis A is very low in children and about 0.1% is under 14years old (http//www.who.int/csr/disease/hepatitis/whocdscsredc2007/en/ index.html;accessed). But in our centre it is more than 2%. Countries with transitional and intermediate HAV endemicity (e.g. Eastern Asia) have experienced the "epidemiological shift" due to last 20 years (Jacobsen, 2004). The "epidemiological shift" leads to an increased risk of potentially large hepatitis A epidemics (Bell, 2002). Similarly out of three HEV cases two developed FHF singly & recovered completely. We found 10 cases having no viral markers, probably there may be some other viruses causing hepatitis in children.

Conclusion

Food borne hepatitis can be prevented by maintenance of good hygiene. Epidemiological shift in Hepatitis A has resulted in a reduced prevalence of naturally acquired HAV antibodies and an increased susceptibility in adults leading to more symptomatic and more severe cases of Hepatitis A. There is a need of hepatitis A vaccination to young children, which will reduce the risk of infection in adolescents and adults through herd immunity[13]. Hepatitis B infection carries more risk of fulminant hepatitis or development of chronic hepatitis or hepatic carcinoma. This can be prevented by universal immunization with Hepatitis B & safe injection practices. Hepatitis C can be prevented by proper screening of blood & blood products in blood banks before transfusion. Double volume exchange transfusion may be a modality of treatment in those who can afford in encephalopathy cases.

Probably there may be some other viruses causing hepatitis in children as we found 10 cases having no viral markers but investigation showed features of hepatitis.

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