

Study of Acute Viral Hepatitis With Special Reference To Fulminant Hepatic Failure



Medical Science

KEYWORDS : Fulminant Hepatic Failure, acute viral hepatitis

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ABSTRACT

Objective: This study was conducted to observe clinical profile of acute viral hepatitis (AVH) and fulminant hepatic failure (FHF), laboratory investigations helping in establishing of diagnosis and prognosis and outcome.

Method: Retrospective study of 200 patients suspected of having AVH/FHF admitted during period of January 2011 to January 2016 at Sola Civil hospital, Ahmedabad, were included.

Result: 20% patients of AVH developed FHF, Most common causative organism was HAV in 47% patients. Mortality in patients having FHF was 55%.

Conclusion: FHF is a leading cause of death in children admitted for Acute viral hepatitis. Mortality rates are high due to rapid progression. Early laboratory diagnosis helps to determine prognosis and improving outcome.

INTRODUCTION:

Viral hepatitis is one of the major health problem in developing countries. Hepatotropic viruses are designated hepatitis A, B, C, D, E, and G viruses. Many other viruses can cause hepatitis including herpes simplex virus (HSV), cytomegalovirus (CMV), Epstein-Barr virus, varicella-zoster virus, human immunodeficiency (HIV), rubella, adenoviruses, enteroviruses, parvovirus B 19, and arboviruses.^{1,2,3}

HAV appears to cause most cases of hepatitis in children.^{1,2}

Fulminant hepatic failure is a complication of viral hepatitis, and is one of the leading causes of death in hospitalized children with viral hepatitis in India. It is a clinical syndrome resulting from massive necrosis of hepatocytes or from severe functional impairment of hepatocytes in a patient who does not have a pre-existing liver disease. It is associated with very high mortality rate of 70 to 80%.

MATERIALS AND METHODS

All the patients admitted in the pediatric wards who were suspected of having acute viral hepatitis and/or fulminant hepatic encephalopathy were included in the study.

All the patients of chronic hepatitis, chronic liver disease, cirrhosis with portal hypertension, obstructive jaundice and neonatal jaundice were excluded from the study.

Clinical examination including a detailed neurological examination, triage scoring, Glasgow coma scale and encephalopathy grading were recorded at the time of admission. Investigations were done as recorded in the

proforma. Treatment was instituted as early as possible. Daily follow-up examination was done.

RESULTS:

Out of 200 patients of acute viral hepatitis, 61(30.50%) patients were in the 0 to 3 years of age group, 71(35.50%) patients were in the 4 to 6 years of age group, 43(21.50%) patients were in the 7 to 9 years of age group and 25(12.50%) patients were in the 10 to 12 years of age group. 126(63%) patients were male.

Table 1 : Incidence of causative agent in acute viral hepatitis patients

Agent	Causative	Total no. of Patients (n=200)	Incidence
HAV		94	47.00%

HBV		7	3.50%
HCV		1	0.50%
HEV		16	8.50%
+ HEV	HAV	15	7.50%
Unidentified	Uni-	67	33.00%

HAV infection was the most common viral infection with 94(47%) patients having HAV infection. Causative agent for 67(33%) patients remained unidentified. (Table 1)

40 patients out of 200 patients with Acute Viral Hepatitis, developed fulminant hepatic failure with hepatic encephalopathy. Out of these 40 patients, 19(47.50%) had HAV infection, 3(7.50%) had HBV infection, 2(5.00%) had HEV infection, 2(5.00%) had HAV+HEV infection and 14(35.00%) had unidentified viral infection. None of the patients having HCV infection developed fulminant hepatic failure.

Out of 40 patients with fulminant hepatic failure 22(55.00%) patients expired. Out of these 22 patients 8 had HAV infection, 2 had HBV infection, 2 had HEV infection, 1 had HAV+ HEV infection and 9 had unidentified viral infection.

Out of 40 patients with fulminant hepatic failure, 14(35.00%) patients had SGPT levels of 0 to 500 IU/L out of which 7(50.00%) mortalities were there. (Table 2)

Table 2 : Distribution of SGPT level and mortality in fulminant hepatic failure patients

SGPT Level (IU/L)	Total No. of Patients (n=40)	Mortality
0 to 500	14 (35.00%)	7(50.00%)
501 to 1000	7(17.50%)	5(71.43%)
1001 to 1500	9(22.50%)	4(44.45%)
1501 to 2000	2(5.00%)	2(100%)
2001 to 2500	1(2.50%)	1(100%)
2501 to 3000	1(2.50%)	0(0%)
3001 to 3500	1(2.50%)	0(0%)
3501 to 4000	2(5.00%)	2(100%)
More than 4000	3(7.50%)	1(33.33%)

Out of 40 patients with fulminant hepatic failure, 28 patients developed gastrointestinal bleeding, 15 patients developed renal failure, 9 patients developed seizures and 6 patients developed hypoglycaemia. Mortality in patients who developed complication ranged from 65% to 84%. 8 patients did not develop any complication and in this group of patients there was no mortality.

(Table3)

Table 3 : Distribution of complication and mortality in fulminant hepatic failure patients

Complications	Total No. of Patients (n=40)	Mortality
Hypoglycemia	6(15.00%)	5(83.33%)
Seizures	9(22.50%)	6(66.67%)
Gastrointestinal bleeding	28(70.00%)	21(75.00%)
Renal failure	15(37.50%)	10(66.67%)
No complication	8(20.00%)	0(0%)

DISCUSSION:

In the present study, hypoglycemia was associated with high mortality (83.33%). In the study of Srivastava et al⁴, which showed that blood glucose level less than 45mg/dl was associated with poor outcome. So seizures, gastrointestinal bleeding and hypoglycemia were associated with high mortality in patients of fulminant hepatic failure.

According to Comparison in the present study, HAV was found in 47.50% patients, which was comparable with studies of Poddar et al⁵ (53.97%) bender et al⁶ (33.33%). HBV was found in 7.50% patients, HCV was not found in any patient. HEV was found in 5.00%. Combined HAV+HEV infection was present in 5.00% patients.

CONCLUSION:

Incidence of acute viral hepatitis was maximum in age group 3 to 6 (35.50%). Most common symptoms were jaundice, fever, anorexia, nausea. HAV was most common cause. Maximum mortality was associated with prothrombin time 41-50 seconds.

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