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Paediatrics

A STUDY OF INCIDENCE OF FULMINANT HEPATIC FAILURE IN PAEDIATRIC AGE GROUP

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ABSTRACT Fulminant hepatic failure is a life threatening condition which is characterised by rapid impairment of hepatic function in a patient without pre-existing liver disease. Sudden detoriation of hepatic function results in jaundice, coagulopathy,encephalopathy and multi organ failure. We reported 198 cases of jaundice out of which 6 cases were diagnosed with fulminant hepatic failure with most common etiology being acute viral hepatits. **AIMS AND OBJECTIVE:** To study the incidence and etiology of fulminant hepatic failure in children between 6 month to 18 yrs of age belonging to both rural and urban areas with sign and symptoms suggesting of liver disease and fulfilling diagnostic criteria of FULMINANT HEPATIC FAILURE. **MATERIAL AND METHOD:** A detailed history, clinical examination and investigation was dne of each and every patient. Hematological investigation was done through SYSMEX XS 800i. **RESULT:** In the present study which was conducted on 198 patients, the incidence of fulminant hepatic failure was 3%.The incidence was more common in male population as compared to female population,ie:66.6 percent population studies were male as compared to 33.3 percent female population. The age group which was more involved was 6-12 years ie 50% cases were seen in the age group 6-12 years as compared to other. The most common cause of fulminant hepatic failure was Hepatitis A as compared to other etiologies like Hepatitis B,C, Drug induced hepatitis etc. In this study the incidence was more common in rural population as compared to urban population. **CONCLUSION:** FHF has a low incidence in paediatric age group. Acute liver failure in children is a hetrogenous condition with varied etiology. Early detection of liver disease and management of the case by a multi disciplinary team is the mainstay to decrease the mortality from FHF.

KEYWORDS : Jaundice, Fulminant Hepatic Failure, Encepalopathy, Coagulopathy

INTRODUCTION

Hepatic failure is a syndrome that occurs as a consequence of severe hepatocyte dysfunction.

Fulminant hepatic failure implies absence of any pre existing liver disease.

Acute liver failure is a life threatening illness caused by rapid detoriation in hepatocellular function due to hepatocellular injury or death.

Hepatic failure leads to a multi organ response characterised by hepatic encephalopathy, coagulopathy, cerebral oedema, abnormalities in metabolic pathways, susceptible to infection and hemodynamic disturbance.

Fulminant hepatic failure is defined on the basis of encephalopathy within 8 weeks of the first symptom of illness without any previous history of underlying liver disease[1].

Fulminant hepatic failure is subcategorised on the basis of development of hepatic encephalopathy as

Hyper acute liver failure in which the encephalopathy occurs within 7 days of occurrence of jaundice.

Acute liver failure in which encephalopathy develops during 8-28 days after onset of jaundice and,

Sub acute liver failure in which encephalopathy develops during 5-12 weeks after onset of jaundice.

There are studies which have proved that those patients with the rapid onset of encephalopathy have the best chances of recovery.

Rivera-Panera and colleagues[3] have found that children surviving Fulminant Hepatic Failure without transplant were admitted to the hospital sooner after the onset of illness (mean 8 days) then those non-survivors (mean 22 days).

The above definition is not adequate in case of children as it is difficult to assess the early stages of encephalopathy and encephalopathy may not be apparent in infants until the terminal stages of ALF has reached.[2]

Due to this difficulty in finding an adequate definition of ALF in children the Pediatric Acute Liver Failure Study Group (PALFSG) has come out with a new definition[1].

It is characterised by

A. Biochemical evidence of Acute liver injury with no evidence of Chronic liver disease.
B. Hepatic based coagulopathy not corrected by Vit.K administration with Prothrombin time (PT)>15sec or International normalised ratio (INR)>1.5 sec with any grade of encephalopathy or INR>2.0 without encephalopathy[(1)].

The PALFSG ALSO DEVELOPED a scale to assess encephalopathy in children. (Table 1). As the patho-physiology of liver failure becomes better understood, definitions should reflect disease mechanisms ratherthan clinical descriptions.

Table 1. Stages Of Hepatic Encephalopathy

Stage	Clinical manifestations	Asterixis/ reflexes	Neurologic signs	EEG
I	Confused, mood changes	Absent/ normal	Tremor, apraxia	Diffuse slowing
II	Drowsy, decreased inhibition	Present/ hyper-reflexic	Dysarthria, ataxia	Abnormal general slowing
III	Stupor,Sleepy but Arousable	Present/ Babinski sign	Muscle rigidity	Triphasic waves
IV	Coma	Absent	Decerebrate or Decorticate	Very slow delta activity

In infants and young children it is difficult to determine on the basis of behavioral or mental status changes whether such a patient is manifesting early signs of encephalopathy; therefore, the early signs for infants and toddlers should include irritability, poor feeding, listlessness, seizures (often due to hypoglycemia) and loss of normal infantile reflexes.

Pathology

Patchy or confluent, massive necrosis of hepatocyte is commonly found on liver biopsy.

Collapse of the reticulin framework of the liver may occur as a result of multilobular or bridging necrosis.

Centrilobular necrosis is characteristic in cases of drug intoxication or circulatory shock.[4-7]

Pathophysiology

Due to hepatocyte injury,the metabolic functions of liver are impaired, because of this patients develop abnormal glucose homeostasis, increased lactate production, impaired synthesis of coagulation factors and reduced capacity to eliminate drugs, toxins and bilirubin. As a result, patients develop coagulopathy, hypoglycemia and acidosis, all of which increase the risk of gastrointestinal bleeding, seizures and myocardial dysfunction. Bacterial and fungal infections often complicate ALF. Bacteria may enter the systemic circulation from the gut as a result of impaired liver macrophage cell function or in association with the insertion of catheters and endotracheal tubes. Depressed production of complement and acute-phase reactants may contribute to decreased response to infection. The combination of a decreased integrity of the immune system, exposure to antibiotics and insertion of catheters increases the risk of fungal infection.[4-7]

MATERIAL AND METHODS

A detailed case history was taken and a thorough clinical examination was done in each case. The findings were recorded in a specially prepared performa.

The cases were subjected to following investigations –Hb, TLC, DLC, ESR, Hepatic Enzymes-SGPT, SGOT, Serum Alkaline Phosphatase, Serum Bilirubin-Direct and Indirect, RBS, Renal function-S.Creatinine, Serum Electrolytes (Na+,K+,Ca++,p04-),Coagulation Profile-PT,APTT,Viral Studies-HBsAg, IgM anti HBcAg, Anti HAV IgM, Anti HCV antibody, CMV,EBV and Herpes virus, Toxin and drug level in serum whenever indicated

RESULT

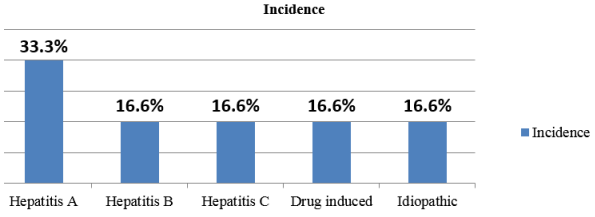
Table -I) Incidence Of FHF

Total Patient	Number of FHF pt.	Incidence
198	6	3

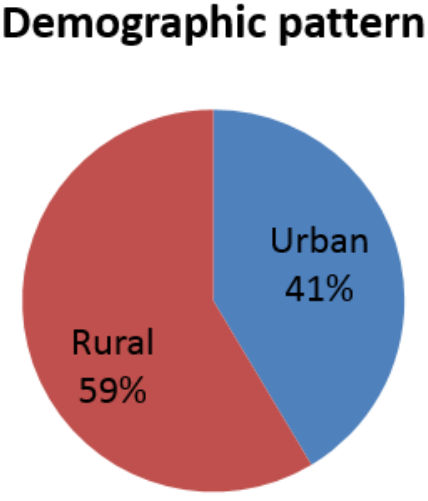
Table-II) Age Incidence Of FHF

Age group	No. Of patients	Percentage
Below 6 years	1	16.6%
6 – 12 years	3	50%
13 - 18 years	2	33.3%
Total patients	6	100%

(Table-III) Etiology Of FHF



(Table-IV)Demographic Pattern of jaundice patients



DISCUSSION

We have studied 198 Jaundice patients, admitted in paediatrics department out of which 6 were diagnosed as cases of acute fulminant hepatic failure.

Most of the patients (50%) were in 6-12 year age group. **Arora NK, Mathur P(2003)[8-10]**, reported that the disease affects the young adolecent predominantly.In the present study male:female ratio was 2:1,results were comparable with **Nanda SK, Gulati S(1996)[8],** drug induced FHF was in 16.6%,where as paracetamol overdose was prominent cause of FHF, **Gagan .k.sood(2010)[11].**

No cause was found in 16.6% cases,the observation for idiopathic FHF was similar to **Samanta T, Ganguly S et al(2007)[12]**. The etiology of liver failure depends on the age and geographical location also.

Acute infectious hepatitis due to the hepatitis A,B,C or E virus is the most common cause of acute liver failure in indian children responsible for 50 to 70% of cases[8].

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CONCLUSION

FHF has a low incidence in paediatric age group. In our study incidence rate was 3%, 50 percent patients were of 6-12 years of age group. Acute liver failure in children is a heterogeneous condition with varied aetiology. Early detection of liver disease and management of the case by a multi disciplinary team is the mainstay to decrease the mortality from FHF. Our result showed that there is significant co-relation between knowledge, attitude and behaviour on the occurrence of jaundice and its complications, this calls for increasing awareness on all aspects of jaundice to minimize the complications.

It is concluded that there is need to increase the public awareness by different educational programs regarding aetiology of hepatitis and its prevention by immunization for hepatitis A and B,use of safe injection, safe water and hygienic Sanitary habits.

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