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Assessment of acute hepatic failure in children with encephalopathy

[1] Dr. Umesh Kumar, [*2] Dr. Manoj Kumar Ram [3] Bankey Bihari Singh

[1, 2] Senior Resident, Department of Pediartics, Anugrah Narayan Magadh Medical College and Hospital, Gaya, Bihar, India
[3] HOD, Department of Pediatrics, Anugrah Narayan Magadh Medical College and Hospital Gaya, Bihar, India

Abstract
Hepatic encephalopathy can occur in those with acute or chronic liver disease [3]. Episodes can be triggered by infections, GI bleeding, constipation, electrolyte problems, or certain medications [4]. The underlying mechanism is believed to involve the build up of ammonia in the blood, a substance that is normally removed by the liver.
This study is planned in, Anugrah narayan magadh medical college and hospital. Total 30 children's with Children with increased liver encephalopathy were admitted in the study. The neurological examination was performed in all patients. On the basis of history and clinical examination, staging of hepatic encephalopathy was done.
Acute hepatic failure is a disease with high mortality rate. This is more so if bleeding manifestations are there. Hepatitis A virus Is a common agent of Acute hepatic Failure in children while hepatitis B has become less common. many reasons of Acute hepatic failure still to be established largely due to scarcity of new diagnostic modalities. Facility for liver transplantation is still out of reach to a majority of population and where there it is available, it is unaffordable for the most. Therefore focus should be in proper implementation of prevention such as vaccination and society education.

Keywords: hepatic encephalopathy, liver disease, children, acute hepatic failure

Introduction
Hepatic encephalopathy (HE) is an altered level of consciousness as a result of liver failure [2]. Onset may be gradual or sudden [2]. Other symptoms may include movement problems, changes in mood, or changes in personality [2]. In the advanced stages it can result in a coma [3].
Hepatic encephalopathy can occur in those with acute or chronic liver disease [3]. Episodes can be triggered by infections, GI bleeding, constipation, electrolyte problems, or certain medications [4]. The underlying mechanism is believed to involve the build up of ammonia in the blood, a substance that is normally removed by the liver [2]. The diagnosis is typically made after ruling out other potential causes [2]. It may be supported by blood ammonia levels, an electroencephalogram, or a CT scan of the brain [3].
Hepatic encephalopathy is possibly reversible with treatment [1]. This typically involves supportive care and addressing the triggers of the event [3]. Lactulose is frequently used to decrease ammonia levels [1]. Certain antibiotics and probiotics are other potential options [1]. A liver transplant may improve outcomes in those with severe disease [1].
More than 40% of people with cirrhosis develop hepatic encephalopathy [5]. More than half of those with cirrhosis and significant HE live less than a year [1]. In those who are able to get a liver transplant, the risk of death is less than 30% over the subsequent five years [1]. The condition has been described since at least 1860 [1].
The mildest form of hepatic encephalopathy is difficult to detect clinically, but may be demonstrated on neuropsychological testing. It is experienced as forgetfulness, mild confusion, and irritability. The first stage of hepatic encephalopathy is characterised by an inverted sleep-wake pattern (sleeping by day, being awake at night). The second stage is marked by lethargy and personality changes. The third stage is marked by worsened confusion. The fourth stage is marked by a progression to coma [3].
More severe forms of hepatic encephalopathy lead to a worsening level of consciousness, from lethargy to somnolence and eventually coma. In the intermediate stages, a characteristic jerking movement of the limbs is observed (asterixis, "liver flap" due to its flapping character); this disappears as the somnolence worsens. There is disorientation and amnesia, and uninhibited behaviour may occur. In the third stage, neurological examination may reveal clonus and positive Babinski sign. Coma and seizures represent the most advanced stage; cerebral oedema (swelling of the brain tissue) leads to death [3].
Encephalopathy often occurs together with other symptoms and signs of liver failure. These may include jaundice (yellow discolouration of the skin and the whites of the eyes), ascites (fluid accumulation in the abdominal cavity), and peripheral edema (swelling of the legs due to fluid build-up in the skin). The tendon reflexes may be exaggerated, and the plantar reflex may be abnormal, namely extending rather than flexing (Babinski's sign) in severe encephalopathy. A particular smell (foetor hepaticus) may be detected [6].
N a small proportion of cases, the encephalopathy is caused directly by liver failure; this is more likely in acute liver failure. More commonly, especially in chronic liver disease, hepatic encephalopathy is triggered by an additional cause, and identifying these triggers can be important to treat the episode effectively [3].

Table 1

Type	Causes
Excessive nitrogen load	Consumption of large amounts of protein, gastrointestinal bleeding e.g. from esophagealvarices (blood is high in protein, which is reabsorbed from the bowel), kidney failure (inability to excrete nitrogen-containing waste products such as urea), constipation
Electrolyte or metabolic disturbance	Hyponatraemia (low sodium level in the blood) and hypokalaemia (low potassium levels)—these are both common in those taking diuretics, often used for the treatment of ascites; furthermore alkalosis (decreased acid level), hypoxia (insufficient oxygen levels), dehydration
Drugs and medications	Sedatives such as benzodiazepines (often used to suppress alcohol withdrawal or anxiety disorder), narcotics (used as painkillers or drugs of abuse), antipsychotics, alcohol intoxication
Infection	Pneumonia, urinary tract infection, spontaneous bacterial peritonitis, other infections
Others	Surgery, progression of the liver disease, additional cause for liver damage (e.g. alcoholic hepatitis, hepatitis A)
Unknown	In 20–30% of cases, no clear cause for an attack can be found

Hepatic encephalopathy may also occur after the creation of a transjugular intrahepatic portosystemic shunt (TIPS). This is used in the treatment of refractory ascites, bleeding from oesophageal varices and hepatorenal syndrome [8, 9]. TIPS-related encephalopathy occurs in about 30% of cases, with the risk being higher in those with previous episodes of encephalopathy, higher age, female sex and liver disease due to causes other than alcohol [7].

It is necessary to evaluate the clinical spectrum and markers influencing the prognosis of patients with hepatic encephalopathy. There is ainsufficiency of data about the field of manifestation and prognosis of children with hepatic encephalopathy especially in developing countries. Therefore the study was tried at a tertiary level centre to analyse the clinical and biochemical spectrum of manifestation, prognosis and markers determining the prognosis in children with acute hepatic encephalopathy.

Methodology

This study is planned in Anugrah narayan magadh medical college and hospital. Total 30 children's with Children with increased liver encephalopathy were admitted in the study. All patients were informed consents. The aim and the objective of the study were conveyed to patients.

The neurological examination was performed in all patients. On the basis of history and clinical examination, staging of hepatic encephalopathy was done.

In every patient tests was done which involved blood cell counts, peripheral blood smear for malarial parasite, liver function tests [Serum Asparatateaminotras ferase, Alanine aminotransferase, Direct and Total Bilirubin, Serum Albumin, Prothrombin time (PT), INR, Serum electrolytes, Blood urea, Serum creatinine and blood culture. Tests for Viral markers was done which included hepatitis B surface antigen (HBsAg) and antibodies against hepatitis A virus and hepatitis C virus (HCV).

The end result was determined in form of survival (improvement and successful discharge after the encephalopathy has been resolved), death and leave against medical advice (LAMA).

Results & Discussion

Total 30 cases were studied in the present study. There are 20 male and 10 females out of the enrolled study patients.

Table 2: Study of different parameters

Parameters	Mean	Standard Deviation (SD)
Icterus (duration)	12.5	16.2
Declined level of consciousness (duration)	1.80	1.85
Hemoglobin	8.6	2.2
TLC	1.86	0.65
Platelet count	1.95	1.21
PT	52.00	31.5
INR	4.30	1.9
SGPT	890	101
SGOT	742	80.5
Alkaline Phosphatase	850	48.5
Bilirubin (serum)		
Total	16.3	9.2
Direct	7.9	6.2
Proteins(serum)		
Total	6.1	1.1
Albumin	3.6	1.2
Sodium	128.5	7.5
Potassium	4.1	0.9
Blood Urea	58.2	40.5
Serum Creatinine	1.3	0.9

Table 3: Displaying course on the basis of Encephalopathy's stage

Stage	Expire	Survival	Lama
One (n=7)	0	5	0
Two(n=5)	2	2	0
Three (n=10)	4	5	3
Four (n=10)	6	0	3

Acute hepatic failure is life-threatening condition of varied causes. Clinical scenario of it involves icterus, coagulation abnormality and encephalopathy. Aetiology of it is varied in nature. Common agents are viruses and drugs. Hepatitis A and Hepatitis E are common cause of Hepatic encephalopathy in underdeveloped nations [10-12]. Hepatitis B virus is not a common agent nowadays, probably due to vast coverage of hepatitis B vaccination [13, 14]. Evaluation of etiology could not be possible in most (75%) of patients due to scarcity of diagnostic modalities. Still it has been mentioned that etiology is not clear in 45-50% of acute hepatic failure even after detailed investigations [15]. These cases known as 'cryptogenic' cases [16] may be due to metabolic disorders or unknown viral agents

Seventy percent of study patients were expired or taken leave against medical advice therefore, survived were thirty percent. This proportion is similar to evidences from other institutions where there is absence of facility for liver transplantation [15, 17]. In majority of cases, there were markedly increased PT (INR > 4), which is well known factor to assess prognosis and very long prothrombin time is linked with high mortality [18]. At INR >4, there is the indication for the liver transplantation, especially in small children. Chances of Survival after liver transplantation is 60% to 80% which depends on patient's conditions and facilities and infrastructure which are available [17-18]. The study evaluated the indicators which influences end result of liver failure. Higher the stage of encephalopathy poorer the outcome. Hemorrhage was common in expired. Coagulation derangement is a common picture associated with Acute hepatic failure. There was not an important difference in serum bilirubin ranges, hepatic enzymes and Prothrombin time between survived and expired. This may be due to small sample size. In a from Turkey based study, stage of encephalopathy, serum bilirubin levels were significantly greater in expired patients [15]. In this study, Sodium levels were lesser in expired patients as compared to those who survived, which may be explained by syndrome of inappropriate secretion of antidiuretic hormone.

Conclusion

Acute hepatic failure is a disease with high mortality rate. This is more so if bleeding manifestations are there. Hepatitis A virus is a common agent of Acute hepatic Failure in children while hepatitis B has become less common. many reasons of Acute hepatic failure still to be established largely due to scarcity of new diagnostic modalities. Facility for liver transplantation is still out of reach to a majority of population and where there it is available, it is unaffordable for the most. Therefore focus should be in proper implementation of prevention such as vaccination and society education.

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