**RESEARCH ARTICLE**

# Hepatic Encephalopathy & Liver Failure in Pediatric Patient with Hepatitis A

Sharma's Dakhel AbdulHassan

*Department of Pediatrics, Lecturer, FIBMS Pediatrics, College of Medicine- Al-Qadissiyah University/Iraq*

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**ABSTRACT**

**Background**; Symptomatic hepatitis A (HA) infection generally only requires caring. The prodromal symptoms incline to recover at the start of jaundice clinically. "fulminant hepatic failure (FHF)" is supposed to be an unusual impediment, occurring merely in 0.14–0.35% of admitted cases. "Hepatic encephalopathy (HE)" is a well-defined variety of neuropsychiatric oddities in those having dysfunction of the liver after omission of brain disease.

**Patients and methods**; Diagnosis of hepatitis A patient after clinical suspicion made, patient send for virology screen in central health lab for virology using ELIZA.

**Results:** All positive cases clinically have "Hepatitis A" involved in the revision, those in turn documented by virology screen, total patient were 357 proved by clinical and lab, five of them had a clinical finding of encephalopathy and liver failure from those four dead and only one had recovery.

**Keywords:** Acute liver failure, Fulminant hepatic failure, Hepatitis A, Hepatitis A virus, Hepatic encephalopathy.

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## INTRODUCTION

Indicative hepatitis A (HA) infection commonly requires caring. The initial symptoms incline to recover at the start of jaundice clinically. "FHF" is held to be a bizarre impediment, stirring only in ~0.14-0.35% of admitted cases. On the other hand, an escalating incidence much-admired in approximately northern European countries somewhere up to ~20% of gear of fulminant viral hepatitis is payable to HA infection. This experience equivalents the perpetually added delayed exposure to HA and the enlarged severity of the illness as soon as contracted in late life. The consequence of increasing FHF is most excellent monitored by assays of a coagulation factor, with factor V levels "being the most part favored" and the prothrombin time (PT). Survival of up to 67% be inflicted with been gotten with therapeutic management, in malevolence of the co-existence of such complications as brain edema, renal and respiratory catastrophe and the metabolic sequelae of Acute liver failure (ALF)[1] is definite as the inception of coagulopathy, jaundice and encephalopathy in 8 weeks, in the nonexistence of aforementioned hepatic disease.[2] In attendance are numerous reasons for ALF: vascular abnormalities, metabolic diseases, drug-induced, and viral hepatitis, which account for ALF in kids in frequent areas of the world. HAV is the main reason for pediatric ALF. Although there is a suggestion of a definite genetic character of viruses present in ALF patients,[3] this explains some HAV patients existing with ALF, while most patients had a complete recovery, remains recognized.

In ALF, extensive hepatocellular cell death occur, leading not merely for encephalopathy, coagulopathy and jaundice, nonetheless also for indicative lab results, as prominently higher alanine aminotransferase (ALT), contrary to raises of aspartate aminotransferase (AST) present underneath lesser severity illnesses, where cell death is not so extensive. The widely held of procedures provoking ALF are momentary, so on caring management is needed if the child is capable of surviving longer time enough for the liver to recover functionally.

ALF is a really uncommon impediment of HAV, occurring in 0.1–0.4% of the infected children.[4]

Despite development in new techniques, ALF death rate is more than ~80%, and solitary management is transplantation of liver, which strengthens the significance of the preventive measures.

HE is definite as a variety of neuropsychiatric oddities in those having hepatic dysfunction, afterward brain disease exclusion.[5-7] HE is regarded as personality alterations, intellectual weakening, and a depressed consciousness.[8] A

**Author for Correspondence:dr_sh_d@yahoo.com, shaimaa.dakhel@qu.edu.iq*

chief precondition for HE is deviation of portal blood into the systemic circulation.[9]

Around ~30% of cases die at the end-stage liver disease complaining from momentous encephalopathy, impending coma.[10]

Despite more than one hundred years of study, the HE pathogenesis is quiet not completely understood. This imitates the restriction of study the patient's brain with "HE" in vivo. Furthermost data available were derivative from investigational representations of HE, that's away from picture-perfect. The utmost public submissions comprise the neurotoxins role, compromised neurotransmission because of alterations of metabolism in ALF, modifications in the energy metabolism of the brain, systemic inflammatory response, and modifications of the blood-brain barrier.[11–13]

An underlying mechanism that's believed includes an accumulation of blood ammonia, a material that's usually excreted through the liver. A judgment is classically taken after the exclusion, further probable reasons.11 It might be reinforced through measuring blood ammonia, brain EEG, or a brain CT scan.[14]

Ammonia was a well-known neurotoxin linked to HE. The main source of ammonia is the intestine. It is created via bacteria of colon that catabolize of nitrogenous sources (for example blood of GI bleeding) and via enterocytes from glutamine.[15] Well, the liver drives out nearly whole the portal vein ammonia, altering it to glutamine and avoiding its entrance to the circulation. The ammonia increment in advanced hepatic disease is a result of blood shunting around a liver and of hepatic function impairment. Wasting of muscle, communal existence in those children likewise might contribute; meanwhile, muscle is a vital location for extrahepatic ammonia removal.

HE is probably reversible by giving suitable treatment.[13] This characteristically includes addressing the triggers of the event and supportive care.[14] To decrease ammonia levels, lactulose is frequently used. Probiotics & Certain antibiotics are additional choices. A hepatic transfer can advance consequences in individuals with the grave disease.[13]

**Treatment**

Folks with rigorous HE (advanced stages 3 and 4) have gamble of airway obstruction awaited to decrement in defending reflexes (as the gag reflex). It could result in the arrest of the respiration. Transporting kids to care units, such as an intensive care unit, is mandatory & airway frequently intubated to preclude lethal obstacles (e.g., aspiration or respiratory failure).[16,17] The settlement of a nasogastric tube licenses the innocent direction of medication and nutrients.[14]

If HE develops in ALF, even in minor stages "grade 1–2", it designates that hepatic transplantation might be requisite, & transmission to a professional center is counseled.17

**"Lactulose/lactitol"**

Lactulose\lactitol are disaccharides which don't absorb via the gastrointestinal tract. They are believed to diminish ammonia generation via bacteria, resulting in nonabsorbable ammonia through transforming it to ammonium ions and rise bowel content transit via the intestine. Quantities up to ~15-30 mL are giving 3 times\day; the consequence is intended to be ~35 soft stools\ day, or a pH stool of <6.0.14,16,18,19 Lactulose may be administered via enema, particularly in severe encephalopathy.[18] Frequently, enemas of phosphate are used. It can dismiss constipation, 1 of the reasons of HE, and rise bowel transit.[14] they are valuable in treating HE, and are the optional 1$^{ST}$-line management.[14,20] Lactulose doesn't arrive on the scene to be other useful than lactitol for treating relations with hepatic encephalopathy.[20] An adverse effect of them consists of the probability of bloating, flatulence, diarrhea, and nausea.[20] In ALF, it is indistinguishable whether lactulose is beneficial. The potential part makes happen of bloating might restrict hepatic transplantation if required.[17]

**"Antibiotics"**

Individuals with a continuing disease, rifaximin may be added to lactulose as 2$^{nd}$ line therapy if lactulose can't be tolerated or ineffective.[13] It is an antibiotic from the rifamycin class, which is nonabsorbable. It is believed to act in a comparable way to additional drugs but deprived of the side effects linked to metronidazole or neomycin.[19]

The neomycin antibiotics & metronidazole are other antibiotics used to cure HE.[11] The point that ammonia & unwanted products are produced & changed through bacteria of the intestine, and massacre these bacteria would diminish the production of the product. "Neomycin" had been preferred for its lowest absorption from the intestine. Anon studies displayed that it was undeniably absorbed once administered by the oral route, with resulting complications. "Metronidazole" is less frequently administered due to more extended usage could lead to nerve damage, as well as gastrointestinal side effects.[14]

**"L-ornithine and L-aspartate" [LOLA]**

They LOLA lowers ammonia level in a people's blood.[21] Actual weak data from clinical trials indicate that LOLA may assist those with HE LOLA descents ammonia levels through the increment of urea invention through the urea cycle a eliminates ammonia by spinning it into the neutral urea substance. LOLA might be mutual with "lactulose and/or rifaximin" if these deserted are ineffectual at symptoms control.[14]

## PATIENTS & METHODS

Data collected from the central health lab for virology in Al-Diwaniyah city-Iraq. Diagnosis of hepatitis A patient after clinical susbtion made, patient send for virology screen in central health lab for virology using ELIZA. Frankly, not all patients send to this center because many doctors depend on clinical diagnosis, in addition to 1º health centers, central hospital and outpatient clinics also do an investigation, which not reported in the center of virology. From fixed fact, many people in the rural areas treat HA by traditional methods without reaching the center or doing any investigation these last groups not reported in our data (missed).

## RESULTS

Total cases screened for HA were 483, positive cases were 357 (74%), Figure 1.

For those who are positive by test, a female was 170 cases (48%), a male was 187 cases (52%) Figure 2.

In our study, patients from urban areas 305 (85%), while rural area contributes to 52 cases (15%) Figure 3.

Patient was divided into two age group 1-5years old (234) 66% in the 2nd age group 5–15 years old (123) 34% Figure 4.

Over the year of the total study cases were 357, the higher rate of infection in a hot climate, as showing in Figure 5 and 6.

The higher percent of infection in the following months (May, June, July, August, September) that's mean more rate of infection in the summer Figure 6.

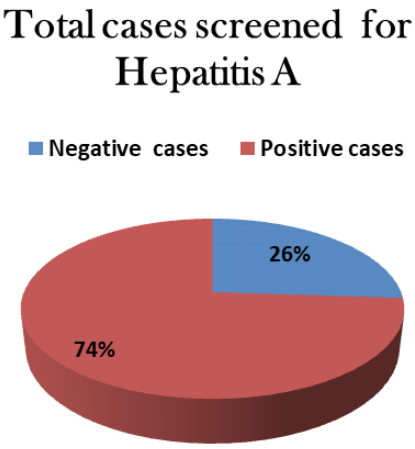


**Figure 1:** Total cases screened for hepatitis A

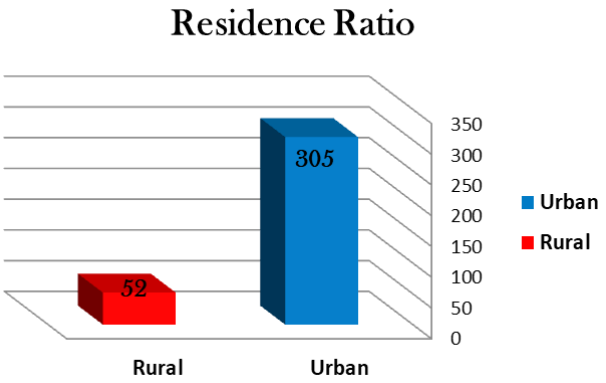


**Figure 3:** Residence ratio

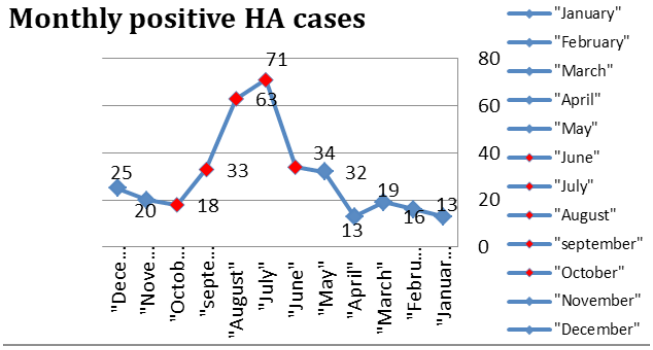


**Figure 5:** Positive monthly HA cases

Total patient were 357 proved by clinical and lab,5 of them (1.4%) (1 male 11years, 1 male 9 years, 1 male 7 years, 1 female 5 years and 1 male 11 years "who survive") had clinical finding of encephalopathy and liver failure from those four dead and only one had recovery Figure 7.

## DISCUSSION

A vital role of the liver is to make toxic materials in the body harmless. These constituents may be made via the body (ammonia), once the liver is damaged, these poisons can accumulate in the blood and affect the job of the nervous system. The outcome may be HE and LF

HE can happen abruptly, and the patient may come to be ill very rapidly as the result of infectious cause as hepatitis. In our study, hepatic encephalopathy and liver failure occur in

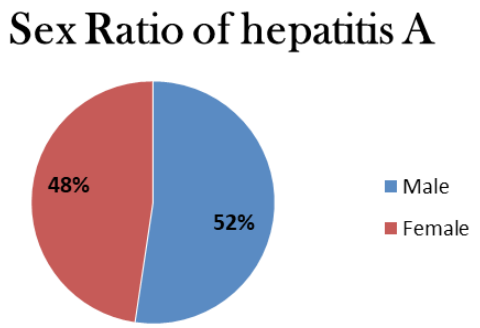


**Figure 2:** Sex ratio of hepatitis A

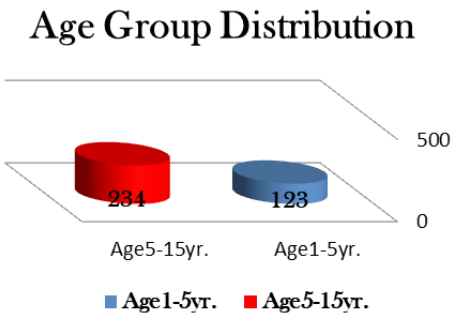


**Figure 4:** Age group distribution

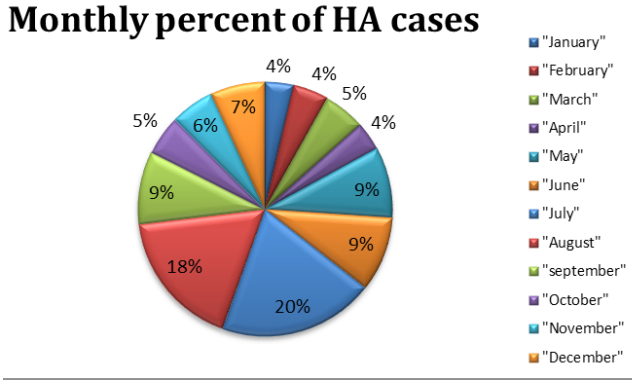


**Figure 6:** Percent monthly of HA cases

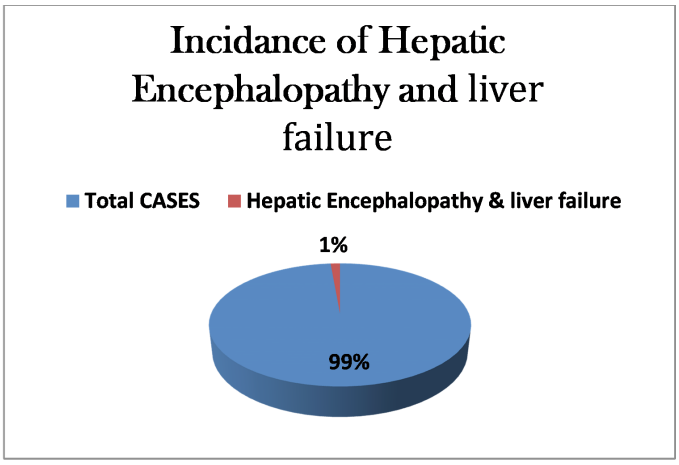


**Figure 7:** Incidence of Hepatic Encephalopathy & liver failure

1.4% of total cases of HA, while in France The occurrence of HAV-convinced ALF in kids is not well-known as the utmost offspring with symptomatic HAV aren't admitted nor notified to health authorities. HAV infection is the chief reason of FLF in kids sent to specific institutes "the main pediatric liver transplantation in France".[22-24]

The cause of the evolution of liver to damage and eventually advancement to FLF in a group of kids with HAV infection is unclear. The virulence, along with the quantity of inoculum of virus and Host factors, might be essential. Whether their genetic contextual could possibly influence these kids to extensive forms of hepatitis A is unknown.

Our study showing that prevalence of HA in male slightly higher than female so there is no sex factor predisposition, in addition to urban contributing 85%; rural 15% that's most likely due to cases in rural area not reported as in city because many patients not ask medical advice or doing investigation.

Tow third of patients from the age group (1-5years), one third age group (5-15yr.), so disease more frequently in age < 5year. But more sever in age >5 years. as in In Germany, 681 hepatitis A cases were testified to the Robert Koch Institute (RKI) in 2014.[25]

Nearly about 1% of HAV infections end to ALF,[26] while in our study, 1.4% develops ALF. Young kids mostly belong to patients group with unobvious or subclinical disease & had no jaundice or clinical symptoms[27] this fact also met in our study. In compare, the disease is further high severity in the older age group, where symptoms are occurring in ~70%. In general, HAV-associated ALF had a survival of ~69%; the lasting 31% necessitate urgent hepatic transplant or die.[28] In our study also disease more sever in the older age group where those with ALF 4 of them (80%) age >5 years and male; One case <5year. & female; death occurred in 80% and 20% survive.

The explanations why HAV infection may advancement once in a while to ALF are understood. Fundamental crowd issues, for example, age plus minor pre-existing hepatic impairment, might play a title role.[29,30] Furthermore, viral issues in addition to viral load and a greater proportion of changeovers in the 5′ untranslated area of the genome of the virus are moreover believed to raise the probability of a FHF sequence.[31,32] Inquiries to become aware of a perfect discrepancy in gene sequences among infected children with FHF courses of HAV and those having an inconsequential course didn't head to clear fallout.[33] In distinction, at hand is particular proof that "cytolytic T cells" had a vital role in HAV pathogenesis and in defining disease course.[34] The decreasing frequencies of HAV in developed nations make it unlikely that unlimited works should be occupied to object this problem in the nearby future.

With HAV, as with various other causes of ALF, carefulness is mostly supportive, and if rescue is doubtful, hepatic transplantation must be suggested.[35] Studies and data for consequences afterward hepatic transplantation doesn't exist.

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