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General Medicine

A CASE OF ACUTE LIVER FAILURE DUE TO HEPATITIS E

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ABSTRACT We report the case of a 20 year old male who came to the hospital because of jaundice and a fever. His symptoms were associated with significant liver impairment and a necroinflammatory pattern due to viral hepatitis B although he had no relevant medical history. His symptoms developed rapidly until death. We present the factors that may have influenced his progression to fulminant liver failure as described in the literature.

KEYWORDS : Fulminant hepatic failure, jaundice, encephalopathy

INTRODUCTION

Fulminant hepatic failure is a severe and acute injury which presents infrequently. It is probably related to or induced by an exaggerated immune response to viral hepatitis, or as described in most of the series, to acetaminophen poisoning and acute liver necrosis.

It is typically described as encephalopathy occurring after a prolonged time (INR> 1.5). Depending on the amount of time of evolution between symptoms and the development of encephalopathy, it is classified into either hyperacute, acute or subacute types.

With this article, we want to put constraints in the early diagnosis of the etiology of liver failure, as well as its proper evolution.

CASE REPORT

We report a case of 20 years old male name Naushad, chronic tobacco chewer, belonging to lower socio-economic class admitted with c/o - high grade intermittent fever with chills and rigor, associated with dry cough, abdominal pain, loss of appetite since 1 month. He presented to hospital with complain of diffuse abdominal pain and subjective fever. Physical examination shows following information-: blood pressure-100/70 mm of hg, heart rate 110/min, blood gas saturation-98% on fio2-0.21, respiratory rate- 18 per minutes, no shortness of breath, no jugular venous distension, painful right upper quadrant palpation.

The patient was admitted for clinical observation because of the high risk of liver failure and the need to study possible liver disease. Fluid resuscitation was started, and studies of liver disease began with a viral profile (HBsAg, HVC, HVA, IgM Hep E, IgG, M Varicella zoster), an immunological profile (Anas - ASMA), and levels of ceruloplasmin and copper.

The patient and family were interviewed and did not refer to any history of alcoholism, so this source was not considered likely. Similarly they did not mention any ingestion of homeopathic medicines or excessive use of acetaminophen. During the first three days in the hospital, the patient remained hemodynamically stable without signs of hepatic encephalopathy, but with worsening of liver function. Tests for HEV Ag were frankly positive. A hepatitis virus panel of tests for HBeAg, Anti HBeAg, Anti-Hepatitis B core total antibodies, and Anti-Hepatitis D antibodies was requested. Treatment began with a 9-16 mg/kg dose of ribavirin.[3] His liver biopsy was s/o-multifocal confluent hepatic necrosis with regenerating nodules and bridging hepatic necrosis.

On the fourth day the patient suffered overall deterioration to grade II hepatic encephalopathy. Management included was metronidazole, lactulose (to counter encephalopathy), N- acetyl cysteine to manage liver disease.Arterial blood gases showed an elevated anion gap which was related to a high level of high metabolic acidosis due to liver failure (PH- 7.47, PCO2- 22, PO2- 96, HCO3- 10.9, SO2- 98 %) .

Patient was treated with bicarbonate infusion, . Patient was shifted to ICU for further management. Patient was given inotropic support and on the 5 th days he developed grade IV hepatic encephalopathy. Orotracheal intubation became necessary to protect respiration.

A CT scan of his brain was taken (Figure 1) appeared normal. The fundus of the eyes was without papilloedema, but he was given bolus of mannitol in view of the possibility of intracranial hypertension.

There was no clinical improvement. Patient was developed upper gastrointestinal bleeding for which 15 cc plasma plus vitamin k and inj. Terlipressin was given stat. Patient went to cardiac arrest, the cause of death was fulminant hepatic failure secondary to hepatitis E infection. Evolution of test results are shown in Table 1.

Table – 1 Test Results

Test name	Au-gust 31/8	Sept-3/9	Sept-5/9	Sept-11/9	Sept-13/9
ALT(Alanine transferase) (U/L)	726	1275	1078	365	191
AST(Aspartat transferase) (U/L)	974	1165	704	422	166
Total bilirubin (mg/dl)	1.40	4.05	9.74	20.13	19.84
S. albumin (gm/dl)	3.1	2.6	2.2	2.3	2.7
S.globulin (gm/dl)	2.5	2.2	4.4	5	5
A/G ratio	1.24	1.18	0.5	0.46	0.54
Prothrombin Time(sec)	15.3	24	27.1	59.4	40
INR	1.17	2.07	2.43	6.54	3.98
PLATELET COUNT (/CU MM)	220	154	150	214	146
S.Creat(mmo l/l)	0.58	0.66	0.65	1.29	1.13
BUN(mmol/l)	23.5	21.4	17.1	15	12.8
Sodium(mE Q /L)	136	130	142	145	153
Potassium(m EQ/LIT)	3.9	5.2	4.1	3.8	4.5
Random Blood Sugar	117 MG/DL	112	100	86	93

His NCCT BRAIN PAIN suggestive of no bleeding or ischemia as shown in Figure 1.

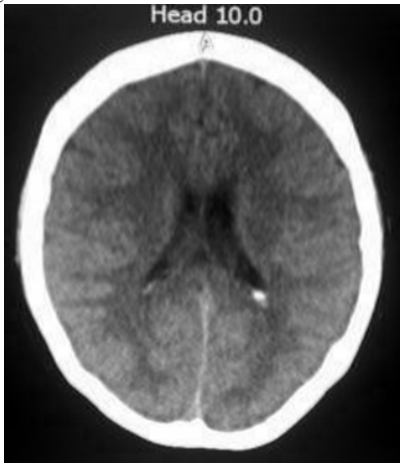

Fig.1 Simple Skull CAT Scan With No Findings Consistent With Evident Bleeding Or Ischemia. No Cerebral Edema.

DISCUSSION

Hepatitis E is a viral disease caused by infection of hepatitis E virus.The virus has 4 genotypes 1,2,3,4.GENOTYPES 1 and 2 found in human.The virus has 3 modes of transmission.
1) Ingestion of undercooked meet.
2) Transfusion of infected blood products
3) Vertical transmission from pregnant women to baby.

It has an incubation period of 2 to 10 weeks.

Fulminant liver failure due to HEV is rare, but it has a high mortality rate and poor prognosis for the patient. Another theory involves alteration of endothelial nitric oxide (ONe). Its normal function is vasodilation for hemodynamic control. When its levels are decreased and nitric oxide synthase (ONS) increases, it causes a change in vasodilation and considerably increases toxic free radical production resulting in damage to the endothelium. In addition, it causes massive activation of caspases leading to large scale apoptosis (20). In fulminant liver failure, Kupffer cells in the sinusoids produce nitric oxide allowing for increased vascular permeability and thus increased concentrations of macrophages in the liver. Furthermore, there is less expression of endothelial nitric oxide (eNO) in the bile ducts, endothelial cells and lymphocytes. There are various classifications based on the time between the onset of jaundice and development of encephalopathy which also provide clues about the cause of the disease.in our patient time interval was seven days which makes this a case of acute or fulminant liver failure.

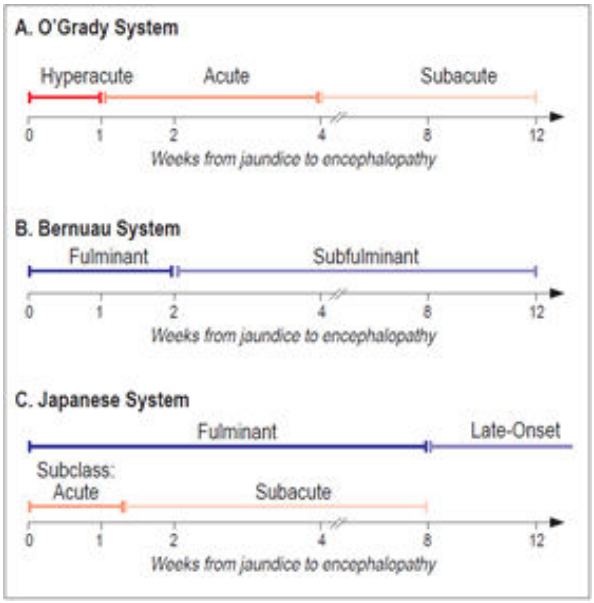

Fig.2 Classification systems for acute liver failure. Modified from the New England Journal of Medicine 2013; 369: 2525-34 [1]

Cerebral edema occurs most in the acute presentation of fulminant liver failure and is the leading cause of mortality For patients with Grades 3 and 4 of hepatic encephalopathy orotracheal intubation is, as in our patient, probably necessary. pressure <40 mm Hg for more than two hours). Mannitol IV bolus at doses of 0.25 to 1.0 g/kg (33°C-35°C) may be beneficial for reducing splanchnic production of ammonia, restoring autoregulation of cerebral hemodynamics and decreasing oxidative metabolism in the brain.

WEST HAVEN GRADING FOR HEPATIC ENCEPHALOPATHY

Grade- 1- Altered Sleep Pattern, Decreased Attention Span
Grade- 2- Asterixis, Lethargy, Decrease DTR
Grade-3 Somnolence to Semi Stupor, Disoriented To Time, Place, Person, Hyperactive DTR
Grade 4- COMA [2]

N acetyl cysteine is use for hepatic acetaminophen poisoning, but it is also used for hepatic encephalopathy to resolve. The recommended dose is 150 mg/kg via IV bolus for 15 to 60 minutes followed by maintenance dose of 5.12 mg/kg/hr for 4 hours and then 6.25 mg/kg/hr. The prophylactic use of antibiotics is not indicated except in case of refractory hypotension, SIRS.[1]

LIVER TRANSPLANTATION

Transplantation is the only treatment option for some specific causes of acute liver failure. The risk is higher among older recipients and among those who received grafts from donors whose ABO blood group is different from that of the recipient.[1]

CONCLUSIONS

The case described in this study is that of a young male with no history of liver disease, no known risk factors for liver disease, no known ingestion of toxic substances but who developed a clinical picture of acute fulminant liver failure. The clinical course was related to hepatitis E infection whether it was acute or relapse, Neither can we rule out other etiologies because studies that would allow this require prior authorization and referral.With this case, we have tried to place this important issue in its medical context.

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