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Acute Fulminant Hepatic Failure due to Hepatitis A Virus: A Case Report

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Abstract

Fulminant liver failure (ALF) is a severe and acute injury which presents infrequently. Hepatitis A virus (HAV) occurs commonly in resource -poor regions like Sudan. It is one of the commonest cause of acute hepatitis in Sudan, but rarely progressed to ALF. A 19 year old young man presented with convulsion and loss of consciousness, He was diagnosed as fulminant hepatic failure due to hepatitis A virus infection. The patient was recovered within two weeks demonstrating a rare consequence of the ALF.

Keywords: Acute hepatic failure ALF; Hepatitis A virus; Hepatic encephalopathy; Liver transplantation

Introduction

Fulminant Hepatic Failure (FHF) or acute liver failure (ALF) is defined as "the rapid development of hepatocellular dysfunction, specifically coagulopathy and mental status changes (encephalopathy) in a patient without known prior liver disease [1]. The time interval of onset of symptoms like jaundice and the appearance of encephalopathy led to several definitions of AFL [2]. Requirements to define ALF are coagulation disturbance determined by prolongation of International Normalized Ratio (INR) usually ≥1.5, or a prolongation of Prothrombin Time (PT) [3]. The other feature is clinical manifestations of hepatic encephalopathy [4]. Less than 1% of patients with acute HAV will develop ALF, and several co-factors will affect its evolution [5]. ALF due to HAV are more common in elder patients group and it has a worse prognosis [6]. This case describes a patient with full recovery from ALF due to HAV.

Case Presentation

A 19 year old man from Eldamar (northern Sudan) brought to the hospital after he developed one attack of convulsion lasts about five minutes aborted spontaneously followed by loss of consciousness, his condition actually started six days prior to admission with low grade fever, loss of appetite, epigastric and right upper quadrant abdominal pain, vomiting three times small amount proceed by nausea not projectile, he was seen in primary health care center and treated as simple malaria. Patient is not alcoholic or smoker, no history of paracetamol or aspirin overdose, no other drugs abuse or blood transfusion, no other medical or psychiatric illnesses history, no family history of similar condition or chronic disease. On physical examination patient looks ill, toxic, deeply jaundice, confused, not oriented in time, place or person, GCS was 8/15, in decorticate position (flexed upper limb, extended hip and knee and flexed ankle, rigidity, hyper-reflexia). There were signs of hepatic encephalopathy grade 4, BP 95/60, pulse rate 120/min regular, respiratory rate 20/min, oxygen saturation 93%, FiO_2 0.21, temperature 38,5 C, fundoscopy showed papilledema. No signs of chronic liver disease, in abdominal examination liver is palpable 3 cm below the costal margin at mid clavicular line and liver span is 14 cm, in cardiopulmonary system examination there was nothing abnormal detected. Laboratory results at admission (day 0) are listed in the Table 1 below, abdominal Ultrasonography showed features of acute hepatitis and portal vein was normal, chest x ray, ECG were normal

Diagnostic challenge

Other screening tests for other causes of acute hepatitis like immunological profile and the level of serum ceuroplasmin and serum copper is not available. Brain imaging (CT, MRI) also not available

Therapeutic intervention

After stabilization, patient was admitted to the Intensive Care Unit (ICU) and the following were done; elevation of the head to 30 degree, nasogastric tube feeding, Dextrose 10% 500ml 3 times per day (tds), Lactulose syrup 10ml tds, enteral Metronidazole tabs 500mg tds (Rifaximin was not available), Mannitol 60 mg bolus dose over 30 minutes and then 15 g/day for 3 days, broad spectrum antibiotic(ceftazidime 1 g iv tds), fresh frozen plasma 3 units and Vitamin k injection 10 mg once/day for three days, vitamin B complex injection once. Patient was improved dramatically clinically and laboratory as shown in Table 2. Patient discharged after 8 days from the ICU, and admitted to the ward for another 4 days for follow up, then discharge on good condition and had frequent visits to our clinic for follow up, also he had been seen in the ophthalmology clinic for slit lamp examination for Kayser–Fleischer rings and sunflower cataract and examination were normal. At his last check up all of his liver enzymes were within normal limits and there were no complains.

Discussion

Our patient presented with a typical features of fulminant hepatic failure according to the definition by the American Association for the Study of Liver Disease (AASLD), which includes evidence of coagulopathy (INR ≥1.5) and altered sensorium (encephalopathy) without pre-existing cirrhosis or liver disease, with duration of symptoms of less than 26 weeks [7].

According to the United States ALF Group Registry statistics, drug-induced liver injury (DILI), including acetaminophen and

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Table 1: Laboratory results at admission (day 0).

HB	12.8 g/dl
TWBCS	16.0 x 10^3
Platelets	280 x 10^9
total bilirubin	19.4mg/dl
direct bilirubin	15 mg/dl
alanine aminotransferase (ALT)	2100 u/l
aspartate transaminase (AST)	1110 u/l
PT	35
INR	3.5
blood glucose	75 mg/dl
blood urea nitrogen (BUN)	21mg/dl
Serum creatinine	0.9 mg/dl
Serum.sodium	132 mg/dl
Serum.potassium	4 mg/dl
Serum.Calcium	9.6 mg/dl
Serum.Chloride	99,1 mg/dl
BFFM	Negative
ICT for malaria	Negative
Anti-HAV IgM	positive
Anti-HCV IgM	negative
Anti-HBV IgM	negative
AgHBs	negative
Serum albumin g/dl	3.8
Serum globulin g/dl	2.9
Lactate dehydrogenase u/L	350

idiosyncratic, is responsible for more than 50% of cases, indeterminate causes (14%), hepatitis B virus (7.7%), autoimmune hepatitis (5.9%), followed by less common causes including ischemia, Wilson's disease, Budd-Chiari syndrome, and pregnancy-related liver failure (e.g. HELLP syndrome) [8,9]. In Sudan, seronegative hepatitis is the commonest cause of ALF at rate of 38%, HBV is the second common cause at a rate of 22%, other causes included severe *Plasmodium falciparum* malaria, hepatitis E virus (HEV) and idiosyncratic drug reactions, and the mortality rate was high at 84% [10].

HAV is a rare cause of ALF worldwide, with a high mortality rate [11]. The infrequently progression of HAV to ALF are poorly understood. Underlying host factors such as age and even minor pre-existing liver damage may play a role [12]. In contrast, there is some evidence that cytolytic T cells may play an important role in the pathogenesis of HAV infection and in determining the course [13].

Hepatic encephalopathy is an essential manifestation of ALF it tends to fluctuate and may progress from a trivial lack of awareness to deep coma [14]. The diagnosis is clinical and requires the exclusion of other causes of neurological disturbance. The course of HE is dictated by the outcome and phenotype of liver failure, and usually parallels the evolution of other parameters of liver function. Additional factors that may worsen the neurological outcome are the coexistence of infection or presence of inflammation without sepsis alongside the presence of other organ failure [15].

Table 2: Patient was improved dramatically clinically and laboratory.

	Day 1	Day 2	Day 3	Day 7	Day 10
Vital signs	stable	Stable	Stable	stable	Stable
GCS	9/15	9/15	10/15	14/15	15/15
Hepatic encephalopathy grade	4	3	(normal)	(normal)	(normal)
PT(seconds)	19.2	17.2	16.5	13	12.5
INR	3	2.4	1.7	1.5	1.4
Total bilirubin mg/dL	16	13	11,2	6.5	2.9
Direct bilirubin mg/dL	11	7.5	6	4.5	1.9
TWBCS count x 10^3	15,	10.0	5.0	4.3	4.4
Serum albumin g/dL	4	3.6	3.6	3.8	3.9
BUN mg/dL	20	18	16.4	13	9.7
Creatinine mg/dL	0.9	1.0	0.9	0.9	0.8
Serum sodium mEq/l	137	136	136	130	133
Serum potassium mEq/l	4.1	4.0	3.8	4.0	3.9
Serum calcium mg/dL	9.8	9.6	9.3	9.5	9.6
ALT u/l	1500	1100	8700	500	200
AST u/l	990	900	600	330	150
ALP u/l	200	190	150	110	70

Coagulopathy is another essential diagnostic component of ALF and has a significant prognostic value, but it is not translated into increase risk of bleeding, actually recent analysis suggested that most patients have normal coagulation state, despite prolongation of measured INR or PT. This is related to significant increases in endogenous heparinoids, procoagulant micro particles, von Wille brand factor and factor VIII, reduced pro- and anticoagulant factors and release of "younger" more reactive platelets in patients with ALF [16,17].

It is important in-patient with ALF to identify the etiology, because it is an important indicator for prognosis and the treatment strategy. Treatment goal is to achieve metabolic and hemodynamic stability. Patients with acute liver failure are at increased risk for hypoglycemia, which can be prevented by an intravenous glucose infusion. Patients with acute liver failure have high energy expenditure and protein catabolism, requiring nutritional support to preserve muscle bulk and immune function [18,19].

Liver transplantation is a treatment option for some specific causes of ALF, but such treatment is not universally available, and less than 10% of liver transplantations are performed in patients with ALF [20].

One of the most important differential diagnosis of ALF in Sudan is malaria. According to Sudan malaria diagnosis and treatment protocol 2017 by the Sudanese federal ministry of health, almost 75% of population in Sudan are at risk for malaria, and in 2015, the reported malaria cases represent 8.7% and 12.2% of the total outpatient attendance and hospital admissions, respectively. And it state that health care providers should regard a patient as having severe malaria if he or she has one or more of the clinical or laboratory findings of severe malaria . Many diseases on its early stages are misdiagnosed as malaria, especially in resource poor peripheral areas in Sudan where

most of laboratory investigations are not available, as our patient.

Conclusion

The case described in this report is of a young man, with no past medical history of chronic diseases or psychiatric illnesses, and no risk factors for liver disease, who was diagnosed as malaria six days prior to admission, presented with convulsion, loss of consciousness, high grade fever diagnosed as fulminant hepatic failure due to hepatitis A virus infection, the patient dramatically improved within a short period, and discharged on a good condition.

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