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Acute Fulminant Hepatitis Complicating an Acute Amoebic Dysentery?

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ABSTRACT

A two and a half year old boy presented for 3 days history of high-grade fever and watery diarrhea followed by a progressing hypoactivity and lethargy. On presentation, he was lethargic, cooperative only with his mother, having a rigid and a tender abdomen. Laboratory workup revealed an acute amoebic dysentery associated with an acute fulminant hepatitis; no other etiologies were found. Only medical treatment was applied and the boy recovered with no sequelae and a subsequent normal liver function.

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INTRODUCTION

Acute fulminant hepatitis is a severe condition caused by massive necrosis or severe impairment of hepatocytes (1). It is considered one of the most challenging GI emergencies encountered in clinical practice.

In adults, encephalopathy is a main diagnostic feature but in children, biochemical evidence of an acute liver injury (less than 8 weeks in duration) with absence of a chronic liver disease and a coagulopathy (INR >1.5 with clinical encephalopathy or INR> 2 without encephalopathy) are major criteria for diagnosis (1).

In many cases, no identifiable cause was found, however, the following etiologies can be considered as the origin of acute fulminant hepatitis:

- Infectious diseases: Hepatitis viruses as Hepatitis A Virus (HAV), Hepatitis B Virus (HBV) on the top of the list; Herpes Simplex Virus (HSV), parvovirus B19 and adenovirus; enterovirus can be the cause in infants less than seven months of age. For other viruses, such as Hepatitis C Virus (HCV), Cytomegalovirus (CMV), Epstein-Barr Virus (EBV), Human Herpesvirus 6 (HHV6) and human immunodeficiency viruses (HIV), there is less certainty that the presence of the virus can be the cause for acute liver failure (2).

-Immune dysregulation: check for Autoimmune markers: antinuclear antibody (ANA), anti-smooth muscle antibody (SMA), and/or liver-kidney microsomal antibody (LKM), an acute viral infection such as EBV can result in a hemophagocytic lymphohistiocytosis(3)(4), neonatal hemochromatosis.

- Metabolic disorders: galactosemia, type I tyrosinemia, mitochondrial diseases, fructose intolerance, urea cycle disorders, Wilson disease... (5)

- Drugs and toxins: mainly Acetaminophen in paediatric population: an acute single ingestion of a dose greater than 100 mg/kg or the Chronic ingestion of >90 mg/kg per day for more than one day can be toxic (6), herbal medications and toxic mushrooms.

-Hypoperfusion: ischemic hepatitis can lead to acute liver failure due to neonatal shock, sepsis, cardiac dysfunction, Budd-Chiari syndrome (hepatic vein thrombosis), veno-occlusive disease, or the use of vaso-constricting drugs such as cocaine and methamphetamine.

Acute fulminant hepatitis is a rapidly evolving clinical condition usually presenting with ascites, jaundice, seizure and encephalopathy (7). Its treatment is a must as the high risk of progression to permanent liver failure and later on to liver transplant is common mainly if the causative agent is unknown, but once the origin is well defined, a targeted therapy will stop the progression to liver failure (7).

Case report:

We report a case of a previously healthy two and a half year old boy who presented to the emergency department for 3 days with history of high-grade fever and profuse watery diarrhea that was once bloody. One day prior to presentation, those symptoms resolved and the boy became hypoactive, lethargic with decrease in appetite. He had no vomiting, no other major complaints and had a normal urine output.

As past medical history, he is a well growing child with unremarkable neonatal history, negative past surgical and family history, well followed in the clinic with updated vaccination.

On presentation: the boy was pale, hypoactive, reacting and cooperative only with his mother. Vital signs were within normal range: Blood pressure: 90/60, Heart rate: 97, Oxygen saturation on room air: 98%, afebrile.

On examination: no signs of dehydration with normal capillary refill (2 seconds), normal oral mucosa and tympanic membranes, good bilateral air entry on lung auscultation with normal heart sounds. The abdomen was non distended but rigid with diffuse tenderness on palpation, normal genitalia for age, no edema of lower limbs, no skin rash, no meningeal signs.

Urgent US abdomen and pelvis was done to r/o any intussusception or intestinal obstruction and was normal.

Laboratory workup done on admission showed the following results:

WBC: 13000, Neutrophils: 84%, platelets: 285000, CRP: 94 mg/L, SGPT: 4555 U/L, SGOT: 7865 U/L, bilirubin (T,d): 2.2/1.5 mg/dl, albumin: 35.8 g/L, INR: 7.44, with otherwise normal electrolytes, GGT and alkaline phosphatase.

An acute fulminant hepatitis was diagnosed.

Vitamin K was given immediately at a dose of 5 mg with fresh frozen plasma (10cc/kg), empiric antibiotic: cefotaxime (150mg/kg/day) was also started.

Stool analysis showed *Entamoeba histolytica* cysts, trophozoites with RBCs and WBCs, so metronidazole (35mg/kg/day) was added.

Daily laboratory workup including SGPT, SGOT, INR and albumin was done and showed an increase in SGPT to 8470 U/L and SGOT to 15060 U/L on the next day then a progressive amelioration in those values. To note that albumin, glycaemia and platelets were always within normal range.

Clinically, the child's activity was better day after day with normalization of bowel movements and appetite.

Full workup searching for the causative event was done and turned to be negative including all of the following tests:

- Viral etiologies leading to an acute fulminant hepatitis (HAV, HBC, HCV, hepatitis D virus (HDV), hepatitis E virus (HEV), parvovirus, EBV, CMV, HSV1, HSV2, toxoplasmosis, widal and wright serologies)

- Autoimmune diseases (anti LKM antibodies, anti-smooth muscle antibodies, ANA)

- Metabolic diseases (ceruloplasmin level, alpha1 antitrypsin level, ferritin was high early in the disease (3312) then it decreased to normal level (64)).

- There was no history of overconsumption of Acetaminophen and serum dosage was normal.

Concerning the imaging: US abdomen performed on admission was normal, Doppler US of liver vessels was also normal.

Follow up tests showed a remarkable amelioration in the previously affected functions: INR: 1.24, SGPT: 119 U/L, SGOT: 56 U/L, bilirubin (T,d): 0.7/0.2.

As a conclusion, this is a case of an acute fulminant hepatitis associated to an *Entamoeba histolytica* infection. It recuperated on pharmacotherapy without reaching a liver transplant.

DISCUSSION

Acute fulminant hepatitis is a complex, rapidly progressive clinical syndrome that is the final common pathway for many disparate conditions, some known and others yet to be identified (8). The initial care of patients with acute liver failure depends on prompt recognition of the condition and early detection of etiology as the mortality of acute liver failure is as high as 40–50% (9).

Concerning the *Entamoeba histolytica* or a related species parasitic infection, the lumen of the gastrointestinal tract is the main target and causes few symptoms or sequelae (1). Clinical amoebiasis generally has a subacute onset. The severity can range from mild diarrhea to severe dysentery, producing abdominal pain, diarrhea and bloody stools. In some cases, it can even progress to a fulminant amoebic colitis that may lead to bowel necrosis then perforation, and subsequently peritonitis. Toxic megacolon and liver abscesses are rare complications (10).

In our case, the acute liver failure was of indeterminate cause and the patient was only on supportive treatment, which contradict with most of previous publications concerning the acute liver failure of unknown etiology. Only the acute amoebic dysentery was present as a cause and its amelioration on therapy was accompanied by a resolution of the liver failure. So can we consider acute liver failure as a complication for acute amoebic dysentery?

CONCLUSION

Acute liver failure is a life threatening disorder in pediatric population where etiologies and management different than that in adult age group. The proportion of acute liver failure with unknown cause is high, about 50% which may reveal that many causes can be attributed as an etiology. Can acute amebic dysentery be one of them? More cases are needed to be reported so we can generalize this hypothesis.

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