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Case Report
Acute Liver Failure

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Acute Liver Failure in an Immunocompetent Host: A Diagnostic Dilemma

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Acute liver failure (ALF) is defined as the acute onset of hepatic dysfunction with a timing of 8-28 days between the onset of jaundice and hepatic encephalopathy. It is a medical emergency which needs prompt diagnosis and appropriate management with identification of the aetiological factors. An interesting case of acute liver failure due to cytomegalovirus (CMV) hepatitis in an immunocompetent, non-addict patient without any co-morbidities is presented here.The patient recovered with supportive treatment and did not require anti-viral therapy as he was not immune compromised. The importance of the case lies in the fact that viral hepatitis by CMV can present in immune-competent patients also and other treatable causes of acute liver failure should also be identified as several patients of ALF may need liver transplantation,

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Introduction

Acute liver failure causes sudden hepatic dysfunction without any pre-existing liver disease with the timing of 8-28 days between the presentation of jaundice and the development of hepatic encephalopathy. Coagulopathy and encephalopathy are common clinical features.

Drug-induced ALF occurs with acetaminophen, propylthiouracil, isoniazid, phenytoin, valproate etc. HAV, HEV, and HBV are common causes of ALF and other viruses such as EBV, CMV, and HSV may also be related. Wilson's disease, mushroom poisoning and autoimmune hepatitis may also be associated with ALF.

CMV-related ALF is not common and causes diagnostic dilemmas in an immune-competent host. Here, an interesting case of a 32-year male patient without any addictions and co-morbidities who presented with jaundice and hepatic encephalopathy is discussed. After ruling out all the common causes of ALF, a diagnosis of acute CMV infection was made by serology and the patient recovered with treatment. The importance lies in the fact that CMV may cause ALF even in the immune-competent host which needs a high index of suspicion and prompt symptomatic treatment results in recovery.

Case Report

32 years non-diabetic, non-hypertensive, non-addict single male presents with a 5-day history of nausea and vomiting with generalized weakness followed by yellowish discolouration of eyes and urine with itching and clay-coloured stool for the last 4 days with disorientation from day 10 of onset of icterus.

There was no history of medications, exposure to toxins, blood transfusion, sexual exposure, travel history, or any history of poor sanitary or oral hygiene or taking food from a community kitchen. There was no previous similar history in the patient or his family, nor any history of rash or any history suggestive of Musculo skeletal symptoms.

On examination, the patient was disoriented in time, place and person with mild pallor and icterus. There was mild hepatomegaly without any skin lesions, lymphadenopathy, or splenomegaly. CVS and respiratory system examination were unremarkable. The neck was supple with bilateral extensor plantar response and a flapping tremor was present.

A diagnosis of septic or metabolic encephalopathy was made as the patient's relatives gave an undocumented history of fever in the initial stage of presentation. The patient was put on 5% dextrose with Ryle's tube and catheterisation in a propped-up position with frequent monitoring of vitals and CBG. Injection PPI, ondansetron, ceftriaxone 2g iv bd APST were added with rifaximin 550 mg bd and syrup lactulose 15 ml thrice daily with a target of 2-3 semi-formed stools per day.

ABG revealed mild hypokalaemia with alkalosis without hypoxemia or hypercarbia and SPO2 was 92% with 2l/min oxygen therapy.

Blood was sent for CBC, peripheral blood smear, RBS, urea, creatinine, LFT, electrolytes, Procalcitonin, Hepatitis A and E virus IgM antibody, HbsAg, anti-HCV antibody, HIV 1 and 2 ab, EBVNA and IgM anti-VCA ab, HSV 1 and 2 DNA PCR, CRP, serum ammonia, IgM and IgG CMV ab, MP slide and dual antigen, IgM and IgG Dengue ab, typhi Dot M, Covid 19 RTPCR, blood for CS aerobic single hand, serum ceruloplasmin.

Urine RE and CS, Stool for RE and CS, Chest X-ray AP supine view, USG whole abdomen, ECG,2D echocardiography with colour doppler, CT brain plain, EEG and CSF study were done after ruling out papilledema.24-hour urinary copper was also sent.

CBC revealed mild anaemia with TLC of 12,000/mm3 without any toxic granules or thrombocytopenia. Urea was mildly elevated at 60 mg/dl with normal creatinine and there was mild hyponatremia and hypokalaemia. LFT revealed conjugated hyperbilirubinemia with conjugated bilirubin 20 mg/dl and unconjugated bilirubin 14 mg/dl. AST was elevated at 130 U/L and ALT was 198 U/L with an ALP of 280 U/L with a rise of GGT level of 72 U/L.There was mild hypoalbuminemia. Ceruloplasmin and urinary copper levels were normal. USG abdomen revealed gall bladder wall thickening and GB sludge and MRCP done did not reveal any additional abnormality IgM CMV ab was elevated at>0.9 S/Co(>0.8 positive)with normal CMV IgG levels. All other tests of fever profile were unremarkable including Scrub typhus IgM ab and Leptospira and brucella IgM ab. Chest x-ray revealed mild bilateral pleural effusion with normal echocardiography and CT brain.

Ammonia was elevated with EEG showing changes of encephalopathy. Urine and blood cultures were negative. INR was elevated at 1.7 which was repeated twice.

A diagnosis of acute liver failure due to CMV hepatitis was made with positive titres of IgM and as jaundice to encephalopathy timing was between 8-28 days with EEG and coagulopathy, ALF was diagnosed with the presence of hepatic flap and rise of ammonia levels. Wilson's disease was also excluded.

The patient started to improve from day 7 of admission with the gradual normalisation of TLC, INR, urea, sodium, and potassium. Bilirubin levels started to decrease along with liver enzymes. The patient started to eat clear liquids and was gradually put on a semisolid diet. IVF and intravenous medications were stopped, and repeat CMV IgG ab levels done after 5 weeks of presentation, showed the value of 1.96 S/Co(>0.7 Positive) proving primary CMV infection as initial IgG was negative. ANA Hep2, ANA profile, Autoimmune hepatitis profile and AFP were normal.

The patient was discharged 5 weeks after admission and sent to Gastroenterology OPD for further management where the patient is undergoing follow-up presently. A liver biopsy was refused by the patient at the initial stage.

Discussion

ALF with jaundice to encephalopathy timing between 8-28 days [1] has several aetiologies such as medications, viral infections, toxins, Wilson's disease etc. It presents with acute onset liver injury without pre-existing liver disease often needing liver transplantation. The combination of jaundice, encephalopathy with coagulopathy proves the diagnosis.

Apart from the common viral agents CMV infection may also cause ALF not only in immune-compromised patients but also in immune-competent hosts causing diagnostic dilemmas.

CMV, a common beta herpes virus causes opportunistic infections in organ transplant recipients with the highest rates in HSCT recipients. [2].

CMV hepatitis was first described in immune-competent patients by Lamb and Stern [3]. The disease presents with less than a 5-fold rise of AST and ALT with a rise of ALP and bilirubin. CMV-induced acute liver failure is extremely rare. [4,5]In immunocompetent hosts infections may range from self-limiting asymptomatic stage to hepatitis. It is transmitted by vertical transmission, via breast milk, blood transfusion, sexual route, and organ transplant [6,7,8,9]

CMV IgM can also be positive in secondary CMV infection, so CMV IgG test in 2 samples taken one month apart is used to diagnose primary CMV infection with the first sample being IgG negative and the second sample IgG positive proving recent infection as per CDC guidelines for CMV diagnosis.

The patient was diagnosed as per CDC (Centres for Disease Control and Prevention). As the patient improved and was non-immune compromised no anti-viral treatment was given. Intravenous corticosteroids in high doses can also be the most important risk factor in CMV liver failure.[10]

Conclusion

CMV infection can cause ALF in rare cases even in immunocompetent patients with serology proving the diagnosis as PCR testing is not available in all set up. The typical presentation of jaundice, encephalopathy with coagulation abnormality and serology proved the diagnosis after the exclusion of other causes. The importance of the case lies in the early diagnosis and treatment of an uncommon cause of ALF.

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