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CMV Hepatitis in an Immunocompetent Young Pregnant Female: A Rare Cause for Maternal Mortality

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Abstract: ***Cytomegalovirus is one of the TORCH infections. TORCH infections classically comprise toxoplasmosis, rubella, cytomegalovirus, herpesvirus, hepatitis viruses, human immunodeficiency virus, Treponema pallidum and other infections, such as varicella, parvovirus B19, and enteroviruses. Cytomegalovirus can be transmitted from mother to child in utero, intrapartum or during breastfeeding. Here we arepresenting the case of immunocompetent young pregnant female with 18 weeks period of gestation who presented in hospital with complaints of fever, vomiting, yellowish discoloration of eyes and urine with altered sensorium. She was being evaluated as a case of acute onset hepatitis with coagulopathy with hepatic encephalopathy (fulminant hepatic failure). While other viral markers were negative, IgM CMV serology came out to be positive. In spite of prompt proper management, she deteriorated further and succumbed to the illness. It stresses the importance that one should keep high degree of suspicion of CMV in immunocompetent individual as a rare cause of fulminant hepatitis in pregnancy.***

Keywords: Cytomegalovirus, TORCH, immunocompetent, pregnancy, viral fulminant hepatitis.

1. Introduction

Cytomegalovirus (CMV) is a DNA virus of Herpesviridae family. The two known main sources of primary CMV infection during pregnancy are through sexual activity and contact with young children. Primary infection occurs in approximately 1 to 4% of pregnancies, and is mostly asymptomatic in immunocompetent adults. However, primary infection may manifest as a mild mononucleosis or flu - like syndrome with persistent fever, malaise, fatigue and lymphadenopathy. CMV can be transmitted from mother - to - child in utero, intrapartum, or during breastfeeding. It is among the TORCH infections (toxoplasmosis, other infections including syphilis, rubella, CMV, and herpes simplex virus). [1]

Positive CMV IgM results indicate a recent infection (primary, reactivation, or reinfection). Primary CMV infection during pregnancy poses a 30% to 40% risk of intrauterine transmission and adverse outcome is more likely when infection occurs within the first half of gestation. [2]

Clinical manifestations (i. e., flu - like syndrome, fever) and abnormal laboratory findings (i. e., lymphocytes >or=40%, elevated aminotransferases) may suggest the presence of primary CMV infection and should prompt subsequent virological investigations. [3]

2. Description

22 - year - old female, G2P1L1 with 18 weeks of period of gestation had complaints of fever, vomiting, yellowish discoloration of eyes and urine for 10 days. She also had altered sensorium for 1 day. Fever was documented, around 101 - degree Fahrenheit, associated with chills and rigor, myalgia, arthralgia, headache, not associated with rash or retroorbital pain and relieved on medication. She had vomiting for 10 days which wasnon - projectile, non - bilious, non - foul smelling, not containing blood, initially containing food later containing water. History of easy fatiguability and loss of appetite were also present. There was no history of pain abdomen, bleeding from any site, burning micturition, generalised itching. No history of leaking or bleeding per vaginum. There was no history of drug intake, unknown substance abuse, neck pain or stiffness, abnormal body movement, blood transfusions, multiple sexual partners. There was no history of tuberculosis, diabetes or hypertension.

The patient was following up for routine antenatal check - up in government hospital. She was referred to medicine department at a higher centre in view of deranged liver function test. HBsAg came out to be weakly positive by rapid card test, but Elisa test for HBsAg came negative.

On examination, the patient had altered sensorium with pulse rate of 116 beats per minute, blood pressure of 104/60mmHg, saturation of 99% on room air, respiratory rate of 18 per minute. She had icterus with dry tongue.

On per abdominal examination, abdomen was distended without tenderness, guarding, rigidity, fluid thrill or shifting dullness. Liver span was 10centimetres. GCS was E2V3M5. Asterixis couldn't be assessed as patient was not following commands. There was no neck rigidity. Bilateral plantar

were extensors. Bilateral pupils were normal size and reactive to light. Cardiovascular and respiratory system did not reveal any abnormality.

She was being evaluated for acute Viral Hepatitis with hepatic encephalopathy with possible aetiology like malaria or leptospirosis or sepsis or autoimmune hepatitis or Pregnancy Related liver diseases (Acute Fatty Liver of Pregnancy or Intrahepatic cholestasis of pregnancy or HELLP Syndrome), or Wilson's Disease etc.

Routine investigations revealed - haemoglobin level of 9.2 gm/dL, total leukocyte count 10670/mm^3 with 57% neutrophils and 35% lymphocytes, Platelet count 2.09 lakhs/mm^3. The blood urea nitrogen and serum creatinine were 10 mg/dL and 0.3 mg/ dL respectively. Liver enzyme panel were as follows - AST 7400 IU/ml, ALT 2680 IU/ml, ALP 163 IU/ml, total protein 6 g/dL, albumin 2.9 g/dL, total bilirubin 11.3 mg/dL and direct bilirubin 7.7mg/dL. Serum electrolytes showed sodium at 143 meq/L and potassium at 3.2 meq/L. Her procalcitonin and lactate dehydrogenase were 0.6 and 393 respectively. Coagulation profile showed Prothrombin time, INR, aPTT and D Dimer as 70.1 s, 5.73, 40 and 7470respectively. On further evaluation malaria rapid antigen test was twice negative. There were no toxic granules. HIV and Hepatitis A, B, C and E were non - reactive. Investigations ruled out leptospirosis, malaria. Ultrasonography of whole abdomen showed single live intra uterine foetus with preserved cardiac activity and liver was 11 cm with normal size, echo texture but gall bladder was partially distended with echogenic sludge at body and neck with pericholecystic fluid. Rapid antigen test for COVID - 19 was negative.

So, provisional diagnosis of Acute Viral Hepatitis with coagulopathy with encephalopathy (Fulminant Hepatic Failure) was kept. Transfusions of fresh frozen plasma were given in view of coagulopathy. But patient continue to deteriorate and was intubated on day 3 of hospital admission in view of poor GCS (5). On day 3 of intubation patient developed fever due to Ventilator associated pneumonia. She was started empirically on ceftriaxone and metronidazole. Antibiotics were upgraded to piperacillin - tazobactam and later to meropenem. Other symptomatic treatment were also given. Further 20 FFP and 1PCV transfusions were given due to coagulopathy and fall in Hb. There was no frank bleed from any site and even Ryle's tube lavage was clear. On ophthalmic fundoscopy, impending papilledema was noticed. Patient had several episodes of hypoglycaemia despite being on continuous dextrose infusion. Conservative management was advised from gynae department. **Rubella IgG, CMV IgM, Herpes IgM, Herpes IgG came out to be positive and rest of the viral markers were negative.** Patient developed bleeding from endotracheal tube, this could be secondary to DIC. Eventually patient went into sudden cardiac arrest but couldn't be revived and succumbed to illness.

3. Discussion

The cytomegalovirus (CMV) is an infectious disease that's a member of the herpes family. While most pregnant women who become infected with CMV will recover on their own, they can transmit the virus to their baby in the womb. [4] Primary infection with cytomegalovirus in pregnancy may manifest as a mild mononucleosis or flu - like syndrome with persistent fever, malaise, fatigue. [1]CMV pregnancy is pretty rare, with only 4 in 100 pregnant women being infected. Those with weaker immune systems who become infected with the CMV virus can experience more serious symptoms that affect the eyes, lungs, liver, and stomach, among other organs. Sometimes, the CMV virus can also cause mononucleosis or hepatitis as in our case. [4]fulminant hepatitis sec to CMV is less thought and often missed Primary CMV infection during pregnancy poses a 30% to 40% risk of intrauterine transmission and adverse outcome is more likely when infection occurs within the first half of gestation. [2]

After ruling out common infectious causes of hepatitis during pregnancy, the threshold for suspicion of infections with other etiological agent like CMV, herpes simplex virus, Epstein Barr virus, Leptospira etc should be kept in mind in the pregnant women with fever and other systemic clinical manifestations of hepatitis.

Early recognition and prompt management is critical in managing such cases in order to reduce the fatal event like maternal mortality.

This case was highlighted as having succumbed to low immunity due to viral CMV hepatitis in a HIV negative case. Literature mentions bad prognosis in immunocompromised host. On thorough literature review we found globally it's very rarely reported in journals. To best of our knowledge only one case has been reported. [6]However due to resource limitation we could not do CMV DNA levels.

4. Conclusion

Clinical manifestations (i. e., flu - like syndrome, fever) and abnormal laboratory findings (i. e., lymphocytes >or=40%, elevated aminotransferases) may suggest the presence of primary CMV infection and should prompt subsequent virological investigations. [3]CMV in pregnancy is pretty rare, still a high suspicion of CMV should be kept in mind.

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