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Case Study RRJOMST

Concurrent Acute Fatty LIVER of Pregnancy, Acute Hepatitis, And Viral Meningitis in An Advanced Maternal Age Pregnancy a Rare Triple Presentation

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Abstract

Background: *Acute fatty liver of pregnancy (AFLP) is a sometimes lethal, but very seldom, chronic hepatic disease during late pregnancy. The presence of AFLP with other severe diseases raises the level of diagnostic and therapeutic complexity to a higher level.* ***Case Presentation:*** *We present the case of a 45 years old multigravita and 26 weeks of gestation with a distinct triad of AFLP, acute viral hepatitis (cytomegalovirus [CMV] and herpes simplex virus [HSV]) and viral meningoencephalitis. The symptoms that were presented by the patient were fever, vomiting, jaundice, altered mental status, and progressive hepatic decompensation. The work carried out in the laboratory showed thrombocytopenia, coagulopathy, hyperbilirubinemia, and high transaminases. CMV and HSV TORCH serology were positive and the cerebrospinal fluid examination revealed pleocytosis. Abdominal ultrasound showed grade 3 fatty liver and neuroimaging showed subacute ischemic injury. Management and Outcome: Multidisciplinary team started management which was inclusive of hepatoprotective agents, antivirals, broad-spectrum antimicrobials, corticosteroids, and supportive therapy. The patient exhibited progressive liver dysfunction in spite of intense treatment.* ***Conclusion:*** *What is unusual about this case is that it is a combination of AFLP, viral hepatitis, and viral meningoencephalitis in pregnancy, a combination never described to our knowledge in the literature. It reiterates the importance of high clinical suspicion, early diagnosis and multidisciplinary care in order to enhance the maternal and fetal outcomes in the case of this complicated clinical setting.*

Keywords: Acute fatty liver of pregnancy, acute viral hepatitis, AFLP, cytomegalovirus infection, herpes simplex virus, pregnancy-related liver disease

INTRODUCTION

Acute fatty liver of pregnancy (AFLP) is a fulminant metabolic syndrome of late pregnancy with an estimated prevalence of 1 in 7,000-20,000 pregnancies [1–3]. Diagnosed methodically by diffused microvesicles steatosis and clinically by extraordinary hepatic decompensation [4, 5], AFLP should be recognized instantly and obstetrically intruded. Acute viral hepatitis is a more common hepatic condition but rarely occurs concomitantly with AFLP and their combined appearance gives rise to a consequent challenge since they share common biochemical derangements. Viral meningitis in pregnancy is rare [6]; however, it adds a new dimension of complexity as it can involve maternal neurological deficits, a systemic inflammatory load, and transplacental transgression. The syngradic appearance of all three entities - AFLP, acute hepatitis and viral meningitis manifestation in a pregnancy of advanced maternal age is extremely unusual, and as such, has no precedence in the indexed literature [7, 8]. Such a triad with absent

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precedent is evidence of why careful clinical suspicion, a prompt multidisciplinary approach, and a personalized care plan with a surgical intervention are needed to reduce the severity of threats to the mother and the foetus.

CASE PRESENTATION

A 45-year-old multigravid female at 6 months amenorrhea, presented with a history of fever of 7 days, vomiting of 2 days and altered sensorium of 1 day. Fever was acute in nature and showed intermittency with low grade, and was accompanied by several symptoms such as chills, rigors, malaise, and night sweats. This was partially alleviated by antipyretics and re-occurred after 4-6 hours. There were no rashes, and arthralgia, or other localizing signs were not reported. She had four bouts of vomiting that occurred over the past two days before admission without abdominal pains or diarrhoea; however, the vomiting contained partially digested food. The day prior to presentation she experienced acute confusion, which culminated in a decreased level of responsiveness. On arrival, she was disoriented to time, place and person and could only respond to painful stimuli. No focal neurological impairments were found. She did not have medial history, drug use, or relevant family history of any significance. On examination pulse, blood pressure and SpO2 were 102bpm,148/90mmhg,98% on room air respectively. General evaluation indicated that the patient was deeply jaundiced and her face looked pale. Respiratory and cardiovascular systems were normal. The abdomen was grossly distended nontender and 26 weeks gestation with foetal heart sound present. Serial studies informed refinement of diagnosis. The initial lab analysis revealed severe thrombocytopenia (39,000/microL), leucocytosis (17,000/microL), and chronic anaemia (haemoglobin 9.0 g/dL). Systemic decompensation was also indicated by her electrolyte abnormalities (Na 124 mmol/L, K 3.2 mmol/L, and Cl 89.6 mmol/L) and coagulopathy (INR 2.2). A significant amount of hepatocellular dysfunction was found in the liver enzymes obtained upon admission and retaken on the seventh day of stay. These enzymes showed elevated transaminases (SGOT 371-636 U/L; SGPT 127-555 U/L), elevated bilirubin (7.0-17.0 mg/dL), and maintained alkaline phosphatase, which did not indicate cholestasis. Enteric fever, bacterial septicaemia, malaria, and parasite infections were excluded by microbiological testing (negative Widal, sterile cultures, unimpressive peripheral smear). IgG 7.91 g/L; ANA, ASMA, and autoimmune screen negative, ruling out autoimmune hepatitis. In comparison, TORCH serology was strongly positive in CMV IgM (231) and HSV IgM (2.78) signifying recent viral infection. Cerebrospinal fluid showed pleocytosis (721/mm 3, protein 386 mg/dL), which added support to the diagnosis of viral meningoencephalitis. Neuroimaging also demonstrated subacute ischemic injury in the right cerebellum, whereas ultrasonographic abdominal indicated a grade 3 fatty liver, which is associated with advanced hepatic dysfunction. The absence of isolated hepatic encephalopathy (normal ammonia 17 μmol/L) and low procalcitonin (0.83 ng/mL) improved the probability that the patient did not have a bacterial sepsis infection. Taken together these results prove acute viral hepatitis meningoencephalitis, which leads to hepatic failure, in line with rare, reported cases. The management of the patient was stepped up as the clinical demands changed. Original treatment comprised haloperidol/promethazine and risperidone to treat agitation, pantoprazole to protect the stomach, ondansetron to against nausea or vomiting, ceftriaxone to treat an infection, N- acetylcysteine to offer protection against hepatotoxicity and low-molecular- weight heparin as thromboprophylaxis. Dexamethasone was used to decrease the level of cerebral oedema, as well as aspirin and clotrimazole as obstetric assistants. Broad-spectrum antimicrobials sequently were initiated as neurological and infectious parameters deteriorated: meropenem, piperacillin-tazobactam, and acyclovir. New drugs included labetalol as antihypertensive, clindamycin as an anaerobe agent and nifedipine as tocolytic agent. A multidisciplinary approach was used to target a multitude of hepatic, infectious, neurological, cardiovascular and obstetric complications (Figure 1 & Table 1).

Table 1. Swansea criteria applied to the present case.

Criterion	Present in Case?	Details from Case
Vomiting	Yes	Reported vomiting over two days prior to admission.

Abdominal pain	yes	Abdominal examination was non-tender.
Polydipsia/polyuria	No	No mention of increased thirst or urination.
Encephalopathy	Yes	Altered sensorium, disorientation to time, place, and person.
Elevated bilirubin (>14 umol/L or >0.8 mg/dL)	Yes	Bilirubin elevated to 17 mg/dL.
Hypoglycemia (<4 mmol/L or <72 mg/dL)	Yes	Glucose levels not specified.
Elevated uric acid (>340 umol/L)	no	Uric acid not mentioned in laboratory results.
Leukocytosis (>11 x10^9/L)	Yes	White cell count: 17,000/microL.
Ascites or bright liver on ultrasound	Yes	Grade 3 fatty liver confirmed by ultrasound.
Elevated transaminases (AST or ALT >42 IU/L)	Yes	SGOT: 371–636 U/L; SGPT: 127–555 U/L.
Elevated ammonia (>47 umol/L)	No	Ammonia level normal (17 umol/L).
Renal impairment (creatinine >150 umol/L)	no	Renal function values not mentioned.
Coagulopathy (PT >14 s or APTT >34 s or INR >1.5)	Yes	INR elevated to 2.2.
Microvesicular steatosis on liver biopsy	Not performed	Liver biopsy not done.

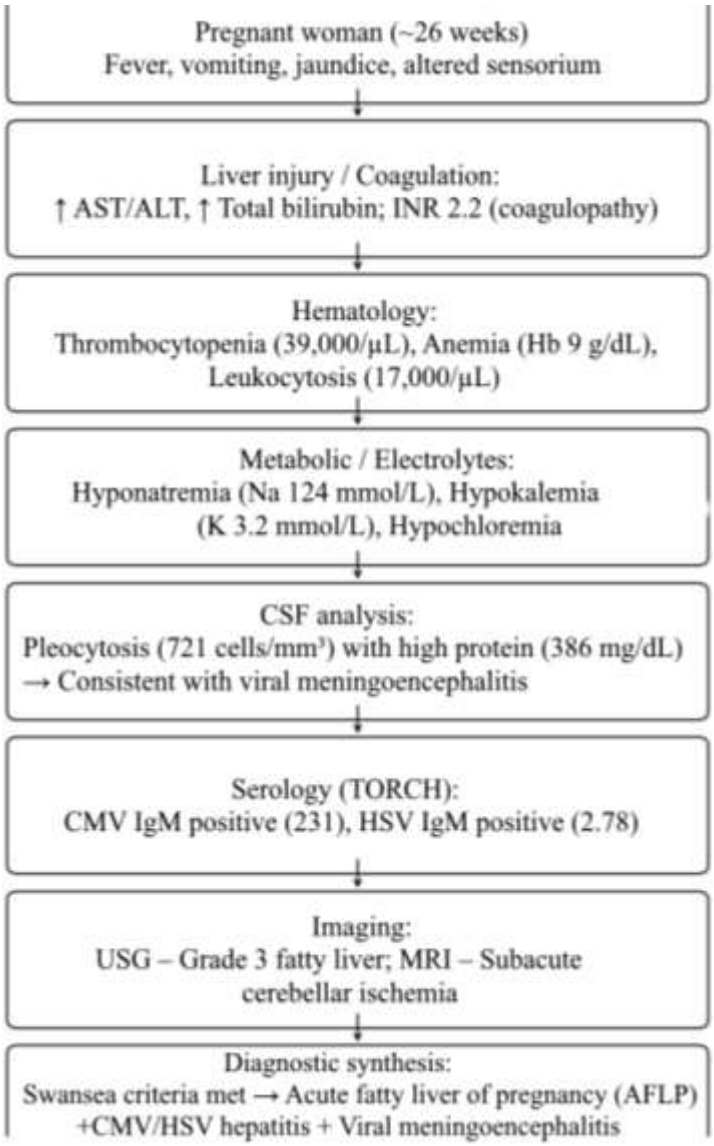

Figure 1. Clinical course/outcome: Progressive hapatic fallure; Intrauterine fetal demise (IUFD).

Total Criteria Met: 9 /14

According to the Swansea Criteria, a diagnosis of AFLP is supported when 6 or more features are present, which is met in this case.

DISCUSSION

An unusual and to the best of our knowledge this is a previously unreported case presentation of a woman of advanced maternal age, who presented with the co-existence of acute fatty liver of pregnancy (AFLP), acute viral hepatitis (CMV and HSV) and viral meningoencephalitis. Each condition is challenging in its own right as a condition to treat and diagnose; when they happen together in one patient the complexity of the challenge is compounded tremendously. AFLP is a rare and formidable liver disorder with a rate of 1 in 7000-15000 pregnancies and a maternal death rate of nearly 18% unless diagnosed and managed promptly [9, 10]. Its pathogenesis is closely associated with dysfunction of mitochondrial 3-hydroxyacyl-CoA-dehydrogenase 3-hydroxyacyl-CoA-dehydrogenase (LCHAD), typically in foetal form, though not always, leading to maternal hepatic microvesicular steatosis and thus to eventual poly-organ dysfunction [1–11].Incorporating features of vomiting, encephalopathy, leucocytosis, elevated transaminases, coagulopathy, hypoglycemia and characteristic imaging finding, the Swansea criteria allow clinical diagnosis in the absence of invasive liver biopsy [12]. The risk of AFLP in this case was supported by the constellation of biochemical aberrations, coagulopathy, and ultrasonographic grade 3 fatty liver which nested the clinical cutoff of AFLP. Other possible causes including viral pathogens, such as CMV and HSV, are relatively rare [13, 14] but increasingly considered as the potential mimics or concomitant contributors to hepatic failure during pregnancy. MV-induced hepatic failure has previously been misreported as AFLP, and HSV hepatitis is notorious in pregnancy [15] due to its fulminant polymerase-chain-reaction as such, it has entered the differential diagnosis of AFLP as well as HELLP in pregnancy. The highly positive CMV and HSV IgM titres obtained in our patient, together with the pleocytosis and cerebral imaging depicting subacute cerebellar ischemia indicated overlapping viral hepatitis and meningoencephalitis. This triple-pathology synergy, i.e. AFLP, viral hepatitis, and viral meningoencephalitis, to our best knowledge, has not been described previously in indexed obstetric literature [16]. Although hepatic encephalopathy is a known effect of AFLP, the presence of CSF pleocytosis and brain lesions described with ischemia by MRI indicated viral neuroinvasion and Cerebro vasculopathy. Viral meningoencephalitis with ischemic sequelae is not frequently recorded in pregnancy and poses an added complication of maternal morbidity. The neurological worsening in this case was consequently multifactorial-representing hepatic metabolic encephalopathy, viral neurotropism and inflammatory vasculopathy. Treatment-wise, the patient required quick maternal stabilization, treatment of coagulopathy, administration of broad-spectrum antibiotics, and administration of targeted antiviral substances with acyclovir against HSV. N-acetylcysteine was also used as a hepatoprotective agent and the corticosteroids deployed to treat the cerebral oedema. Despite overall AFLP standard protocols regarding facilitated delivery to eliminate the pregnancy burdening effect and enhance the efficacy of maternal outcomes, in this case, the viral infection load necessitated multispecialty coordination between obstetrics, hepatology, neurology, infectious diseases, and newborn medicine. Despite these aggressive interventions, the patient experienced progressive liver failure, illustrating the daunting nature of this combination. Limitations of this report include that there was no confirmation of foetal LCHAD deficiency by genetic testing. Nevertheless, it highlights the importance of general diagnostic awareness of pregnant women with hepatic dysfunction and neurological dysfunction. This case demonstrates that although AFLP should be considered as a primary aetiology of late-gestation hepatogenous liver failure, it is imperative to be wary of associated viral aetiologies, like CMV and HSV, which can dynamically shift management when neurological compromise is involved. The convergence of rare yet a severe situation reflects the need of early suspicion, prompt multidisciplinary coordination, and customized therapeutic approach. Early identification and combined treatment are key to the maximum health of mothers and foetuses in such unprecedented and life-threatening situations while in this case neonatal outcome was iufd and mother was admitted in intensive care unit for further management and resulted in partially improved lifestyle. The patient showed advancing liver failure despite these aggressive steps, illustrating the combination's threatening characteristics. This example shows that despite AFLP should be regarded as the main cause of late-gestation hepatogenous liver failure, it is important to be aware regarding

associated viral aetiologies which include CMV and HSV, since they can change strategies when there is neurological compromise. The combination of uncommon yet serious circumstances highlight the necessity of early detection, timely interdisciplinary collaboration, and a tailored treatment strategy. For women and foetuses to have the best possible health in such extraordinary and probably fatal circumstances, early detection and simultaneous treatment are essential.

CONCLUSION

This case describes a rare and complex presentation of acute fatty liver of pregnancy occurring alongside CMV- and HSV-related viral hepatitis and viral meningoencephalitis. The overlapping symptoms led to rapid clinical deterioration, highlighting the diagnostic and therapeutic challenges in late-pregnancy liver failure. Despite timely multidisciplinary care, the disease progressed with poor fetal outcome. This report emphasizes the importance of early suspicion, thorough evaluation for concurrent viral infections, and coordinated multidisciplinary management when hepatic and neurological dysfunction coexist in pregnancy.

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