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Case Report

Coexistence of acute fatty liver of pregnancy, gestational diabetes insipidus, and preeclampsia leading to severe hypernatremia and perinatal mortality: a case report

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ABSTRACT

Acute fatty liver of pregnancy (AFLP) and gestational diabetes insipidus (GDI) are rare but life-threatening obstetric complications. AFLP is linked to hepatic dysfunction, which can impair the breakdown of placental vasopressinase, leading to GDI and eventually hypernatremia. The coexistence of AFLP with GDI can be further worsened by placental dysfunction associated with preeclampsia, due to the disruption of the placental barrier that allows fetal vasopressin to enter the maternal circulation. This rare confluence of AFLP, GDI, and preeclampsia is scarcely reported and presents significant diagnostic and management challenges. We report a 25-year-old G2P1 woman, at 36 weeks of pregnancy, presenting with fever, jaundice, altered mentation, and hypertension. Laboratory tests showed hypernatremia (195 mEq/l), low urine osmolality, and abnormal kidney and liver function tests. Swansea criteria confirmed AFLP. Sodium was gradually corrected at a rate of 6-8 mEq/l per day, and vaginal delivery was planned. A live baby weighing 2.08 kg was delivered but died from respiratory failure on day two. Maternal recovery was complete by day 11 postpartum. This case highlights the interaction between AFLP, preeclampsia, and GDI. It emphasizes the importance of careful sodium correction and increased suspicion of vasopressinase dysfunction in cases of unexplained hypernatremia.

Keywords: Acute fatty liver of pregnancy, Preeclampsia, Gestational diabetes insipidus, Transient diabetes insipidus, Hypernatremia

INTRODUCTION

Acute fatty liver of pregnancy (AFLP) is a rare but life-threatening complication of late pregnancy or the early postpartum period, with an incidence of five cases per 100,000 pregnancies.[1] Although microvesicular hepatic steatosis with progressive worsening of liver function remains the characteristic feature, it is diagnosed by the Swansea criteria.[2] Patients are labeled positive for AFLP if at least six out of 14 criteria listed in the Swansea criteria are met, in the absence of other causes of liver insufficiency.[2,3] The presenting symptoms are often vague, including nausea, vomiting, and abdominal pain, but can progress to cause liver failure, coagulation abnormalities, and encephalopathy.[2] Thus, early diagnosis and early delivery are both known to be associated with decreased morbidity and mortality in both fetus and mother.[4]

GDI is caused by the increased activity of placental vasopressinase, resulting from either overproduction or decreased degradation.[5] It is a rare complication of pregnancy with an incidence of four cases per 100,000 pregnancies.[6] Increased vasopressinase or placental leucine aminopeptidase (P-LAP) activity results in decreased activity of antidiuretic hormone (ADH), leading to polydipsia, polyuria, reduced urine osmolality, and hypernatremia. It commonly occurs during the second or third trimester and typically resolves spontaneously within

4-6 weeks postpartum, with an unlikely recurrence in future pregnancies.[7] The diagnosis is based on clinical evaluation, serum sodium level, and serum and urine osmolality.[5]

The association between preeclampsia and GDI is likely due to leakage of fetal ADH into the maternal circulation due to broken P-LAP functioning at the level of the placenta. While these conditions are well-documented in the literature in isolation, a confluence of these conditions remains sparsely described. We report a case of AFLP with preeclampsia and GDI, culminating in extreme hypernatremia and perinatal mortality, emphasizing pathophysiological interplay and clinical lessons.

CASE REPORT

A 25-year-old woman, gravida 2, para 1 (G2P1), at 36 weeks of pregnancy, presented without antenatal care, complaining of a three-day history of fever, vomiting, jaundice, and confusion. Examination showed hypertension (150/96 mmHg), a fever of 101°F, icterus, and disorientation with a Glasgow coma scale score of ten.[8] Fetal bradycardia (100 bpm) and meconium-stained liquor were observed. Lab tests revealed hypernatremia, increased serum osmolality, abnormal liver and kidney function tests, polyuria, and decreased urine osmolality (Table 1). Ultrasonography (USG) revealed changes suggestive of grade 1 fatty liver (Figure 1).

Table 1: Laboratory parameters of the patient.

Parameter (Unit)	Patients value	Normal range
Serum sodium levels (mEq/l)	195	135-145
Serum osmolality (mOsm/kg)	325	275-295
Serum aspartate aminotransferase (AST) (IU/l)	245	<40 IU/L
Serum alanine aminotransferase (ALT) (IU/l)	210	<40 IU/L
Serum total bilirubin (mg/dl)	6.2	0.3-1
Serum creatinine (mg/dl)	2.4	0.5-1.2
Blood urea nitrogen (mg/dl)	68	6-20
Urine output (ml/day)	3500	800-2000
Urine osmolality (mOsm/kg)	150	275-900

Seven out of 14 criteria of Swansea were met, marking the patient AFLP positive. Other causes of liver dysfunction, such as viral hepatitis, sepsis, and hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome, were excluded. GDI was confirmed via assessment of vasopressinase activity.

Management involved gradually correcting serum sodium levels with a 5% dextrose infusion and fluid supplementation, aiming for a correction of 6-8 mEq/l per day. Delivery was scheduled because the trio of conditions is known to resolve spontaneously after delivery. Vaginal delivery was chosen due to unstable coagulation status and lower anesthesia risks compared to cesarean section. A live baby weighing 2.08 kg was born with APGAR scores of 1, 4, and 7. The neonate experienced respiratory distress and died on day two. Maternal sodium levels normalized by day four, and liver and kidney function tests fully recovered by day 11.

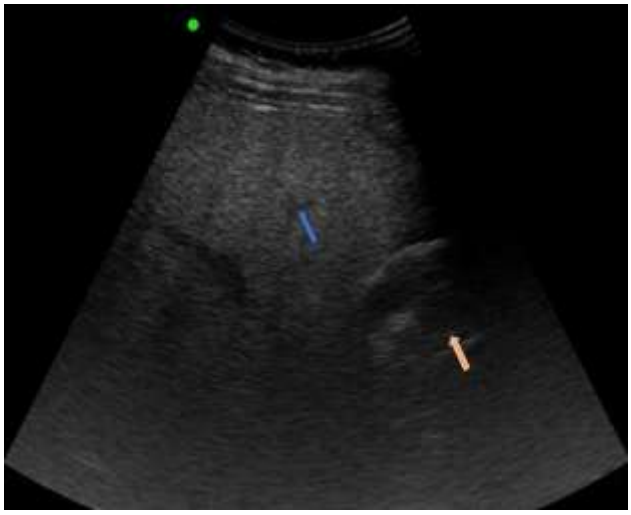

Figure 1: Ultrasonography of diffuse heteroechogenicity and mild fatty changes in the liver parenchyma (blue arrow) and right kidney (orange arrow).

DISCUSSION

This case uniquely highlights the interplay of AFLP, preeclampsia, and GDI. The patient presented with nonspecific symptoms like nausea, vomiting, and fever, but the presence of jaundice with a state of confusion stood out. Further examination and laboratory investigations cleared up the confusion surrounding the diagnosis. Swansea criteria confirmed AFLP, and urine osmolality pointed towards GDI.[3] Shallow trophoblast invasion in preeclampsia likely caused vasopressinase dysfunction, exacerbating hypernatremia beyond typical AFLP manifestations. Reduced placental exchange and insufficient fetal blood oxidation secondary to maternal dehydration might have caused the fetal bradycardia. Though the approach with early diagnosis and treatment has yielded significantly improved maternal and fetal outcomes in patients with AFLP, the fetal outcome remains poorer as compared to maternal outcomes.[1] Neonatal demise underscores fetal vulnerability to maternal dyselectrolytemia and acidosis.[2]

The common causes of AFLP include raised estrogen, abnormalities of fatty acid metabolism, and mitochondrial

dysfunction.[9] On microscopic examination, microvesicular steatosis remains the characteristic finding of AFLP, which causes hepatic dysfunction resulting in altered clearance of vasopressinase, thereby resulting in lowered ADH levels.

Differential diagnosis includes other liver insufficiencies such as HELLP syndrome. While some recent literature suggests seeing AFLP as part of the spectrum of preeclampsia and HELLP syndrome, our case was diagnosed with AFLP given the absence of signs of hemolysis.[2,10] Liver biopsy was avoided due to its highly invasive nature. Other conditions that can cause decreased urine osmolality with hypernatremia include central or nephrogenic diabetes insipidus. Administration of DDAVP (1-deamino-8-D-arginine vasopressin) can be used to rule out nephrogenic diabetes insipidus.[11] In our case, hypernatremia resolved quickly after delivery, and the patient's fluid balance and urine output were normalized with a 5% dextrose infusion and oral water intake; therefore, DDAVP was not administered.

Management of hypernatremia in GDI is centered around replenishing lost free water and aiming for a gradual correction of the serum sodium level of less than 12 mEq/L per day. It is essential not to attempt rapid correction, as this can lead to cerebral edema due to the movement of water from the serum into the brain cells. Serum sodium level, rate of correction, and urine output should be monitored closely during the acute phase of correction. Precipitation of seizures is a sign of cerebral edema and indicates a faster-than-desired rate of correction.[12]

CONCLUSION

In our case, the patient presented with nausea, vomiting, and fever, and was diagnosed with AFLP, GDI, and preeclampsia, which highlights the need for a high level of suspicion and thorough investigation even when symptoms are non-specific. This unique combination of AFLP, GDI, and preeclampsia requires prompt diagnosis and early intervention for a favorable outcome. It involves early delivery along with correction of electrolyte imbalances. The trio usually resolves spontaneously after delivery. Serum sodium levels were corrected gradually to prevent osmotic demyelination and cerebral edema. Perinatal mortality emphasizes fetal vulnerability to maternal dyselectrolytemia. To our knowledge, information regarding the diagnosis, management, and prognosis of cases involving AFLP, GDI, and preeclampsia together is very limited.

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