

Diagnostic Dilemmas in Obstetric Medical Emergencies: A Case Series

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ABSTRACT

The differentiation among hepatic, hypertensive, and gastrointestinal disorders during pregnancy poses significant diagnostic challenges due to overlapping clinical manifestations and biochemical abnormalities. Conditions such as Intrahepatic Cholestasis of Pregnancy (IHCP), preeclampsia, HELLP syndrome, Acute Fatty Liver of Pregnancy (AFLP), and gallbladder disease share common symptoms—including pruritus, hypertension, right upper quadrant pain, and elevated liver enzymes—complicating timely and accurate diagnosis. This case series highlights the diagnostic dilemmas encountered across four distinct clinical presentations: (1) IHCP presenting with pruritus and elevated liver enzymes, initially raising suspicion for AFLP and viral hepatitis; (2) Preeclampsia with markedly elevated alkaline phosphatase, requiring differentiation from HELLP syndrome; (3) Gallbladder calculi mimicking IHCP and hypertensive disorders due to biochemical abnormalities and localised pain; and (4) An isolated 14-fold increase in alkaline phosphatase, which proved heuristically complex. A systematic, multidisciplinary approach—integrating detailed clinical assessment, laboratory investigations, and imaging—was essential for precise diagnosis and optimal management. Emphasising careful clinical judgment, the exclusion of severe conditions, and tailored interventions, this case series underscores the need for clear diagnostic algorithms and management guidelines to improve maternal and foetal outcomes in overlapping obstetric hepatobiliary and hypertensive disorders.

Keywords: Acute fatty liver of pregnancy, Intrahepatic cholestasis of pregnancy, Preeclampsia

INTRODUCTION

The convergence of hepatic, hypertensive, and gastrointestinal disorders during pregnancy presents a considerable diagnostic challenge for obstetricians. Many conditions that affect pregnant women share overlapping biochemical markers and clinical features, necessitating careful differentiation for appropriate management [1]. Among these, IHCP, hypertensive disorders such as preeclampsia and HELLP syndrome, and hepatobiliary conditions like cholelithiasis are characterised by similar laboratory abnormalities, including elevated liver function tests and hyperbilirubinaemia. These biochemical derangements, combined with symptoms such as pruritus, hypertension, and right upper quadrant pain, complicate diagnosis and often result in delayed or inappropriate management [2].

The IHCP is a pregnancy-specific liver disorder, typically presenting in the third trimester with pruritus and elevated bile acids and liver enzymes. Although primarily affecting the mother, it carries significant foetal risks—including stillbirth and preterm delivery—making timely diagnosis and intervention essential. Diagnostic difficulty arises from the need to distinguish it from AFLP and viral hepatitis, which may present with similar biochemical findings but require entirely different management strategies [3]. Similarly, hypertensive disorders such as preeclampsia and HELLP syndrome may present with elevated liver enzymes, thrombocytopenia, and hypertension, leading to diagnostic uncertainty. Differentiation between preeclampsia and HELLP syndrome is crucial, as haemolysis and severe thrombocytopenia may necessitate urgent delivery to prevent maternal and foetal complications [4].

Furthermore, gastrointestinal conditions such as gallbladder disease add to the diagnostic complexity. Gallbladder pathology, particularly cholelithiasis, may mimic obstetric disorders because of overlapping symptoms—especially upper abdominal pain and biochemical evidence of hepatic dysfunction. Determining whether

conservative or surgical management is appropriate requires a nuanced, multidisciplinary approach [5].

CASE SERIES

Case 1: Intrahepatic Cholestasis of Pregnancy (IHCP) and Hepatobiliary Dysfunction

A 29-year-old primigravida at 30 weeks and three days of gestation presented with generalised pruritus without rash [Table/Fig-1], one of the characteristic symptoms of IHCP. The itching, which had worsened over the past two weeks, was most severe on the palms and soles. There was no associated jaundice, nausea, or vomiting, and no relevant drug or family history. Clinical examination revealed no abdominal tenderness or hepatosplenomegaly. Initial investigations showed elevated total bilirubin (3.2 mg/dL), alkaline phosphatase (396.36 IU/L), serum bile acids (23.57 μ mol/L), and Serum Glutamic Pyruvic Transaminase (SGPT) (88.94 IU/L). Viral hepatitis markers were negative, and coagulation studies were within normal limits. Obstetric ultrasound showed normal amniotic fluid and foetal growth, and an abdominal ultrasound revealed no biliary obstruction. Although the presentation was classical for IHCP, the elevated liver function tests necessitated exclusion of AFLP and viral hepatitis, as both may present similarly. However, the absence of systemic manifestations such as hypoglycaemia, coagulopathy, or maternal distress supported the diagnosis of IHCP rather than AFLP. The patient was started on Ursodeoxycholic Acid (UDCA) 300 mg twice daily until six weeks postpartum and was monitored closely for foetal wellbeing. Considering the increased risk of stillbirth in IHCP, induction was planned at 38 weeks. However, she went into spontaneous labour and underwent vacuum-assisted delivery without complications. Liver function tests and serum bile acids [Table/Fig-2] normalised eight weeks postpartum.



[Table/Fig-1]: Pruritus.

Test	Total bilirubin (mg/dL)	Alkaline phosphatase (IU/L)	Serum bile acid (mmol/L)	SGPT (IU/L)
Patient value	0.8	221	9.8	10
Normal value	0.1-1.2	44-147	up to 10	7-56

[Table/Fig-2]: Blood investigations six weeks postpartum.

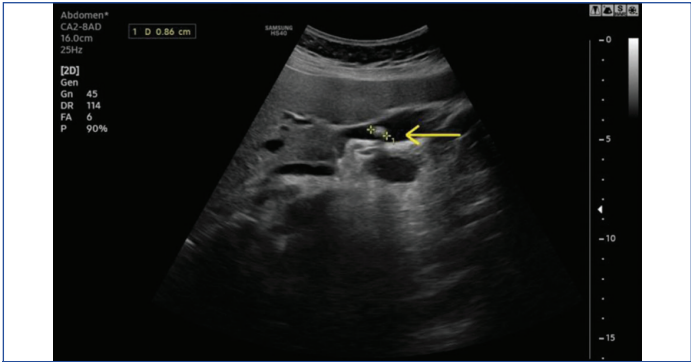
Case 2: Preeclampsia vs HELLP Syndrome vs Acute Fatty Liver of Pregnancy (AFLP)

A 24-year-old G2P1L1 at 35 weeks and four days of gestation, with a previous lower segment caesarean section, presented with persistent headache and mild pedal oedema for five days. She was newly diagnosed with hypertension, with a blood pressure of 150/100 mmHg. Laboratory evaluation revealed alkaline phosphatase of 577 IU/L, haemoglobin of 10.4 g/dL, random blood sugar of 113 mg/dL, prothrombin time of 15.3 seconds, APTT of 30 seconds, and WBC count of 10,600 cells/mm³. Foetal assessment showed mild heart rate variability but no immediate distress. Given the raised alkaline phosphatase and hypertension, differentiation between preeclampsia and HELLP syndrome was crucial. Although hypertension suggested preeclampsia, the absence of haemolysis and thrombocytopenia excluded HELLP syndrome. AFLP was also considered, but the absence of hypoglycaemia and coagulopathy made it unlikely. The patient was started on antihypertensive therapy, but her blood pressure continued to rise, and she developed imminent symptoms within 12 hours of admission. An emergency caesarean section was performed at 35 weeks and five days. Both mother and infant were discharged in stable condition.

Case 3: Third Trimester Pregnancy and Gallbladder Calculi

A 24-year-old G2P1L1 at 28 weeks of gestation presented to the emergency department with a six-day history of right upper quadrant pain. The pain was aggravated by meals and partially relieved by intravenous Paracetamol (1 g). She reported nausea but had no jaundice, fever, or vomiting. Laboratory investigations showed a total leukocyte count of 17,800/cumm, Serum Glutamic Oxaloacetic Transaminase (SGOT) of 19 IU/L, SGPT of 17 IU/L, total bilirubin of 0.6 mg/dL, and alkaline phosphatase of 233 IU/L. Obstetric ultrasound revealed normal foetal parameters, and abdominal ultrasound confirmed gallbladder calculi without cholecystitis [Table/Fig-3].

Given the biochemical abnormalities, IHCP and preeclampsia were initially considered. However, localised tenderness and ultrasound

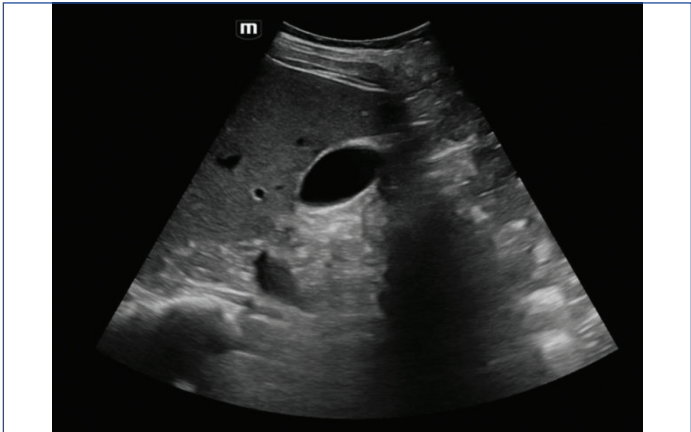


[Table/Fig-3]: GB calculi.

evidence of gallstones supported a diagnosis of cholelithiasis. As there was no acute cholecystitis or biliary obstruction, conservative management was initiated, including dietary modification (high-fibre, low-fat diet) and analgesia with oral Paracetamol 500 mg twice daily for two days. The pregnancy progressed uneventfully to 38 weeks, when she went into spontaneous labour and delivered a healthy infant. Elective postpartum cholecystectomy was planned.

Case 4: Isolated 16-fold Rise in Alkaline Phosphatase

A 22-year-old primigravida at 38 weeks of gestation presented with pruritus over the abdomen for three days. She had no other complaints. An abdominal ultrasound showed no hepatobiliary abnormality [Table/Fig-4].



[Table/Fig-4]: Normal hepatobiliary system.

Laboratory findings included total bilirubin of 0.51 mg/dL, markedly elevated alkaline phosphatase of 1697 IU/L, and bile acid level of 4.5 µmol/L. Alkaline phosphatase electrophoresis confirmed an osteal origin for the enzyme elevation. The patient went into spontaneous labour. Due to grade 3 meconium-stained liquor during latent labour and evidence of foetal distress, an emergency lower segment caesarean section was performed. She delivered a healthy infant. Alkaline phosphatase levels normalised six weeks postpartum.

DISCUSSION

Obstetric hepatobiliary and hypertensive disorders present significant diagnostic dilemmas due to the substantial overlap in clinical manifestations and biochemical abnormalities. Physiological changes in pregnancy can mimic pathological conditions, making the interpretation of liver function tests and hypertensive markers particularly challenging [6]. This case series highlights four such conditions—IHCP, preeclampsia, and gallbladder calculi—each presenting initially with signs suggestive of multiple differential diagnoses.

IHCP, although well documented, is often misdiagnosed, especially in the absence of jaundice. Geenes V and Williamson C reported that up to 20% of IHCP cases were initially diagnosed as viral hepatitis or drug-induced liver injury, leading to delays in management and

increased maternal anxiety [7]. Similarly, in the first case, elevated liver enzymes raised suspicion for AFLP; however, the absence of hypoglycaemia and coagulopathy favoured a diagnosis of IHCP. While bile acid estimation is the definitive diagnostic test, it is frequently unavailable in developing countries, compelling clinicians to rely on clinical symptoms, ultrasound findings, and trends in liver enzyme elevation [2].

Differentiating preeclampsia from hepatic dysfunction is equally challenging. Hypertensive disorders of pregnancy can mimic AFLP and HELLP syndrome due to overlapping biochemical abnormalities. In the second case, markedly elevated alkaline phosphatase levels raised concerns for hepatic pathology; however, the absence of haemolysis and thrombocytopenia excluded HELLP syndrome, and stable coagulation parameters made AFLP unlikely.

Gallbladder disease can also be confused with preeclampsia or IHCP because right upper quadrant pain and mild elevations in liver enzymes are shared features. A cohort study reported that nearly 30% of pregnancy-associated gallbladder disease was initially misdiagnosed as preeclampsia owing to these biochemical similarities [5]. In the third case, IHCP was considered initially, but localised tenderness and ultrasound findings confirmed gallbladder calculi.

Isolated elevation of alkaline phosphatase in pregnancy frequently poses clinical uncertainty. A progressive rise in ALP, particularly during the second and third trimesters, is primarily attributed to increased placental enzyme production. The placental isoenzyme significantly contributes to maternal serum ALP, often exceeding non pregnant reference values [8]. Differential diagnosis should include hepatobiliary disorders such as IHCP, preeclampsia-associated hepatic dysfunction, and less common bone diseases such as Paget's disease or osteomalacia. Elevated ALP has also been sporadically associated with placental pathology, including infarction and insufficiency, although such associations are uncommon in asymptomatic patients.

Several studies support the benign nature of ALP elevation during pregnancy, and case reports indicate no consistent correlation between markedly elevated ALP and adverse perinatal outcomes [9,10]. However, a recent study by Zhang B et al., demonstrated an association between elevated ALP levels and adverse maternal and foetal outcomes [11]. The absence of standardised trimester-specific reference ranges complicates interpretation and underscores the

need for further research. Clinical management of isolated ALP elevation remains conservative; in most cases, reassurance and routine monitoring are sufficient.

CONCLUSION(S)

Careful clinical assessment is crucial for distinguishing among IHCP, preeclampsia, and hepatobiliary disease. Multidisciplinary management facilitates timely and accurate diagnosis, minimises delays, and optimises maternal and foetal outcomes. Improved diagnostic algorithms are needed to reduce misclassification and enhance patient management.

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