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# Case Report on The Mystery Behind Rising Alkaline Phosphatase: A Fatal Hepatic Catastrophe

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## Abstract

**Objective:** To report a rare case of extreme Alkaline Phosphatase (ALP) elevation in pregnancy associated with a hepatic mass and Budd-Chiari Syndrome (BCS), leading to fatal maternal and intrauterine outcomes.

**Case report:** A 36-year-old multigravida at 26+5 weeks presented with jaundice, abdominal pain, and nausea. Labs showed ALP 1037 U/L and coagulopathy. Doppler ultrasound revealed hepatic masses and absent hepatic vein flow; BCS was confirmed on CT. Despite supportive care, the patient deteriorated, leading to intrauterine fetal demise and postpartum multi-organ failure.

**Conclusion:** While mild ALP elevation in pregnancy is typically physiological, levels exceeding 1000 IU/L warrant urgent evaluation for hepatic vascular pathologies such as BCS. This case underscores the importance of early Doppler imaging, multidisciplinary management, and the urgent need for standardized protocols to guide diagnosis and treatment of vascular liver disorders in pregnancy.

## Introduction

Alkaline Phosphatase (ALP) exists as tissue-specific isoforms, with placental ALP (P-ALP) emerging as the dominant circulating form after 20 weeks of gestation. Approximately 85-90% of pregnant women exhibit a 2-4 old ALP increase, with levels exceeding 300 IU/L in 95% of third-trimester pregnancies [1].

Significant elevations beyond this expected range frequently indicate pathological processes. We describe a pregnant patient who exhibited severe ALP elevation along with a hepatic mass and rapid clinical decompensation, culminating in fatal outcomes. This case illustrates the diagnostic complexities of pregnancy-related liver disease. It emphasizes the importance of prompt evaluation when ALP surpasses 1000 IU/L, a critical threshold for vascular pathologies such as Budd-Chiari Syndrome (BCS) [2].

The patient's presentation was notable for several alarming features: the extreme ALP elevation, a heterogeneous liver mass causing vascular compression, and concurrent coagulopathy. These findings contrasted sharply with more common pregnancy-related liver conditions, such as intrahepatic cholestasis or HELLP syndrome, which typically show more modest enzyme elevations [3].

This case highlights three critical imperatives. Firstly, the diagnostic challenge is of extreme ALP elevation of more than 1000

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IU/L in pregnancy. Secondly, the grave prognostic implications of concomitant hepatic masses, and lastly, there is the unmet need for standardized vascular liver disorder protocols. Our patient's rapid clinical deterioration - progressing to hepatic failure within days, mirrors contemporary data showing 78-82% mortality when pregnancy-associated BCS diagnosis exceeds 14 days [4].

## Case presentation

A 36-year-old woman, gravida 6 para 5+0, at 26 weeks and 5 days of gestation, presented to Dow University Hospital with a two-month history of worsening right upper quadrant abdominal pain, jaundice, and nausea. She had been receiving treatment at a secondary care center for the past ten days; however, due to progressive jaundice and discomfort, she was referred to our tertiary care facility. Her obstetric history was notable for five previous uncomplicated vaginal deliveries, the most recent three years ago.

On initial evaluation, she appeared visibly jaundiced and mildly tachycardic with a pulse of 112 bpm. Her blood pressure was 125/70 mmHg, respiratory rate 16 breaths/min, and oxygen saturation was 99% on room air. Abdominal examination revealed a uterine size corresponding to 27 weeks, with identifiable fetal parts and audible fetal heart tones. Obstetric ultrasound confirmed a single viable fetus with growth parameters corresponding to 28+2 weeks and adequate amniotic fluid volume, with no detectable anomalies. A Doppler ultrasound of the liver demonstrated hepatomegaly with lobulated contours and heterogeneous echotexture. Multiple lesions of varying echogenicity were observed across all liver segments, the largest being 4.5×3.7 cm in the left lobe and 4.3×3.4 cm in segment VI of the right lobe. Hepatic veins were not visualized, and the intrahepatic IVC was notably compressed. Although the portal vein remained patent with hepatopetal flow, mild ascites and cholelithiasis were present. These sonographic findings raised a strong suspicion of Budd-Chiari Syndrome (BCS), prompting further diagnostic workup.

Initial labs revealed anemia (Hb 9 g/dL), a platelet count of $134\times10^9$/L, and a WBC count of $7.9\times10^9$/L. Liver function tests were significantly abnormal, with total bilirubin 6.23 mg/dL, Alanine Aminotransferase (ALT) 36 U/L, Aspartate Aminotransferase (AST) 124 U/L, ALP 1037 U/L, and Gamma-Glutamyl Transferase (GGT) 69 U/L. Coagulation studies showed Prothrombin Time (PT) of 13.5 seconds and International Normalised Ratio (INR) of 1.27. Viral serologies for hepatitis A, B, C, and E were negative.

Despite supportive care, her clinical condition worsened. On day four of admission, a triphasic contrast-enhanced computed tomography scan confirmed Budd-Chiari Syndrome. The liver appeared enlarged and lobulated, with heterogeneous enhancement showing the classical "flip-flop" appearance. Numerous regenerating nodules were present, and a hemorrhagic lesion was identified in segment IVb. The intrahepatic IVC appeared slit-like, and multiple para-aortic and pre-aortic lymph nodes were noted, including one mass encasing the left renal vessels, raising suspicion of a possible lymphoproliferative or infiltrative malignancy. A biopsy was considered but deferred due to coagulopathy.

Her labs reflected the clinical decline, as exhibited in Tables 1 and 2. Coagulopathy worsened dramatically, with PT rising from 15.5 seconds on July 26 to 52 seconds by July 30, and INR increasing from 1.48 to 5.46. Fresh Frozen Plasma (FFP) was administered both before and after delivery, which led to a partial improvement (PT 31.4 seconds, INR 3.17).

**Table 1:** Serial Complete Blood Counts of the patient.

| Date | HB (g/dL) | RBC ($x10^{12}$/L) | HCT (%) | MCV (fL) | MCH (pg) | MCHC (g/dL) | WBC ($x10^9$L) | PLT ($x10^9$L) | NEUT% | LYMPH% | MONO% | EOS% | BASO% | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 24-Jul-24 | 9 | 3.3 | 29 | 87 | 27 | 31 | 7.9 | 134 | 79 | 14 | 6 | 1 | 0 | Normachromic aminecytosis Low platelets Clinical camulation advised |
| 25-Jul-24 | 8.7 | 3.2 | 28 | 86 | 27 | 31 | 8.7 | 126 | 79 | 13 | 7 | 1 | 0 | Normochromic anisocytosis Low platelets Clinical comulation advised |
| 26-Jul-24 | 8.6 | 3.2 | 28 | 87 | 27 | 31 | 7.1 | 131 | 84 | 10 | 5 | 1 | 0 | Normochromic anisocytosis Low plateles Clinical comulation advised |
| 27-Jul-24 | 8.7 | 3.2 | 27 | 84 | 27 | 33 | 6.8 | 120 | 78 | 14 | 7 | 1 | 0 | Normochemic anisocytesis Low platelets Clinical complation advised |
| 28-Jul-24 | 10.1 | 3.8 | 32 | 85 | 27 | 31 | 8.5 | 114 | 81 | 9 | 9 | 1 | 0 | Normochromic Low complation platelets advised Clinical |
| 29-Jul-24 | 9.7 | 3.7 | 32 | 86 | 26 | 31 | 10 | 110 | 83 | 8 | 8 | 1 | 0 | Nermochromic Lew platelets Clinical complation advised |
| 30-Jul-24 | 11.6 | 4.4 | 37 | 85 | 27 | 31 | 10.1 | 157 | 81 | 10 | 9 | 0 | 0 | Normochromic misecytosis |
| 31-Jul-24 | 6.1 | 2.3 | 19 | 82 | 27 | 33 | 12.2 | 171 | 83 | 8 | 9 | 0 | 0 | Hypechnomic anisocytosis Neutrophila |
| 01-Aug-24 | 4.7 | 1.6 | 15 | 91 | 29 | 32 | 21.3 | 45 | 70 | 12 | 0 | 1 | 0 | Levkocythroblank picture Severe anemia infection |

**Table 2:** Serial Liver Function Tests of the patient.

| | Total Bilirubin | Direct Bilirubin | Indirect Bilirubin | ALT (SGPT) | AST (SGOT) | ALP | GGT | |
|---|---|---|---|---|---|---|---|---|
| Date | *(0.2-1.2 mg/dL)* | (0-0.3 mg/dL)* | *(0.25-0.9 mg/dL)* | *(<34 U/L)* | *(<31 U/L)* | *(42-98 U/L) | *(<38 U/L)* | Remarks |
| 24-Jul-24 | 6.23↑ | 5.17↑ | 1.06↑ | 36↑ | 124↑ | 1037↑ | 69↑ | Cholestatic hepatocellular injury. |
| 26-Jul-24 | 5.73↑ | 4.90↑ | 0.83↑ | 37↑ | 124↑ | 1213↑ | 96↑ | Persistent cholestasis |
| 27-Jul-24 | 6.04↑ | 5.08↑ | 0.96↑ | 63↑ | 320↑ | 1455↑ | 144↑ | Worsening hepatocelular damage. |
| 28-Jul-24 | 9.50↑ | 7.85↑ | 1.65↑ | 103↑ | 553↑ | 1474↑ | 146↑ | Severe mixed injury |
| 29-Jul-24 | 9.69↑ | 7.91↑ | 1.78↑ | 103↑ | 411↑ | 1281↑ | 123↑ | Rechecked: critical elevation |
| 30-Jul-24 | 8.56↑ | 7.36↑ | 1.20↑ | 131↑ | 484↑ | 1022↑ | 90↑ | Ongoing severe dysfunction |
| 31-Jul-24 | 8.20↑ | 7.15↑ | 1.05↑ | 172↑ | 639↑ | 924↑ | 75↑ | Acute liver injury. |
| 1-Aug-24 | 8.79↑ | 7.32↑ | 1.47↑ | 2273↑ | >6000↑ | 1910↑ | 156↑ | Critical AST/ALT (acute failure?) |

By August 1, her liver enzymes were alarmingly elevated, as seen in Table 2. Troponin I levels were significantly raised at 19560.04 pg/mL, indicating possible myocardial damage, which may have contributed to her deteriorating status. Given the rapid progression toward multiorgan dysfunction and worsening coagulopathy, the decision was made to induce labor in hopes of stabilizing the mother or at least alleviating physiological stress. Signs of imminent maternal collapse further justified this course of action. Labor was initiated with an intracervical Foley catheter and oral Prostin.

Sadly, the patient delivered a stillborn male fetus weighing approximately 1 kg. Postpartum, her condition deteriorated acutely with worsening tachycardia and hypoxia, necessitating cardiopulmonary resuscitation. Despite a transient return of spontaneous circulation, she continued to decline and passed away the following day from multi-organ failure.

## Discussion

The case report revolves around a pregnant woman with a rare presentation of BCS. The patient showed classic symptoms of liver dysfunction, including jaundice, upper abdominal pain, and worsening coagulopathy. Despite intensive medical management, her condition kept deteriorating, leading to both intrauterine fetal as well as maternal demise. The case highlights the challenges of managing hepatic vascular disorders in pregnancy and sheds light on the importance of early diagnosis and multidisciplinary care.

Elevated levels of ALP during pregnancy pertain to multiple etiologies that can be physiological as well as pathological. The pathological situations can be isolated or accompanied by some condition, depending on the isoenzyme of ALP. Numerous cases of elevated ALP levels during pregnancy have been reported previously, but very few studies have shown its association with BCS secondary to a hepatic mass [5-7]. A multidisciplinary approach was found to be common in most of these studies, as high levels of ALP during pregnancy are shown to be complicated by hypertension, gestational diabetes, and mainly preterm labor [8].

Our findings align with previous cases reporting abnormal ALP levels in pregnancy-associated hepatic conditions. Our patient required close monitoring of liver function, thromboprophylaxis, and management for complications like low platelets, anemia, and ascites, similar to another case. Both patients required transfusions during delivery, but in our case, there was maternal as well as intrauterine demise due to worsening liver function, while the second patient, a 21-year-old, delivered a preterm live baby at 31 weeks after fetal distress, requiring NICU care [7]. According to a study, therapies targeting BCS may improve fertility and pregnancy outcomes [8]. This is highlighted in a study that describes a pregnant woman with BCS secondary to inherited thrombophilia. A multidisciplinary approach was acquired for her that resulted in a safe delivery at 36 weeks of gestation, followed by successful liver transplantation [5]. In contrast, there is a study where elevated ALP levels later got complicated with intrahepatic cholestasis of pregnancy and preeclampsia [9]. Another study highlights raised ALP levels secondary to placental damage [10].

The differences between our study and prior literature are most probably due to the differences in the study population. Moreover, genetic and environmental factors, such as limited access to specialized maternal-fetal care, could be a possible reason behind regional differences in clinical outcomes. Furthermore, our case was also complicated by the mass in the liver leading to BCS, while in other cases, BCS was secondary to other conditions like hypercoagulability or myeloproliferative disorder, followed by thrombosis.

Moreover, BCS could have also led to hepatic congestion, leading to hepatic ischemia and necrosis, causing further deterioration of liver function. Additionally, increased ALP and bilirubin levels in our patient also suggest cholestatic liver dysfunction due to hormonal changes during pregnancy. Furthermore, the worsening coagulopathy (INR rising to 5.4) is also highly suggestive of hepatic synthetic failure. This can be considered to explain the postpartum hemodynamic instability and eventual maternal mortality.

## Conclusion

The case underscores the importance of serial liver function assessment and fetal surveillance. The strength of our study is that it presents a rare case of elevated levels of ALP with BCS due to a hepatic mass, with detailed clinical and laboratory monitoring. Nonetheless, these weaknesses were overcome by employing serial ultrasound assessments and multidisciplinary management, ensuring detailed monitoring.

This also raises the need for increased awareness of such hepatic complications in pregnancy for early identification and intervention. The study also discovers an alternative for Obstetricians working at resource-limited facilities, they should

consider hepatic Doppler studies when faced with such persistent liver enzyme derangement. Further studies can be done to explore genetic predispositions and evaluate the role of early anticoagulation therapy in avoiding severe maternal-fetal outcomes.

**Author declarations**

**Conflicts of interest statement**

The Authors declare no conflict of interest

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