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The Unseen Victim of Maternal Envenomation: Delayed Presentation, Placental Abruptio, and Fetal Demise at 36 Weeks Gestation Following Echis Ocellatus Bite in a 40 Year Old Multiparous Woman- A Case Report

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KEYWORDS:

Echis ocellatus; snakebite envenomation; pregnancy; placental abruption; intrauterine fetal demise; delayed presentation; maternal complications.

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ABSTRACT

Background: Snakebite envenomation during pregnancy is a rare but potentially life-threatening obstetric emergency, threatening both maternal and fetal survival. Evidence regarding fetal complications following Echis ocellatus envenomation remains limited.

We report the case of a 40-year-old multiparous woman at 36 weeks of gestation following a delayed presentation after an Echis ocellatus bite. Her clinical course was complicated by placental abruption and intrauterine fetal demise.

Conclusion: This case underscores the devastating consequences of snakebite envenomation in late pregnancy when venom-induced coagulopathy may compromise placental integrity and fetal viability and highlights the importance of prompt presentation, early assessment, and multidisciplinary management. Increased awareness among pregnant women and healthcare professionals, particularly in snakebite-endemic regions, is essential to facilitate timely intervention and improve maternal and fetal outcomes.

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INTRODUCTION

Snakebite envenoming remains a neglected tropical disease and an important cause of preventable morbidity and mortality in rural and resource-constrained communities. Globally, an estimated 5.4 million snakebites occur annually, with 1.8–2.7 million resulting in clinical envenoming and up to 137,880 deaths. [1]Sub-Saharan Africa bears a disproportionate burden, particularly among populations with limited access to timely healthcare and effective antivenom. In West Africa, Echis ocellatus (carpet viper) is a major cause of severe envenoming, producing venom-induced consumption coagulopathy, systemic haemorrhage, shock and tissue injury.[2,3]

Pregnancy introduces an additional dimension to snakebite envenoming because the systematic effects of venom toxicity may compromise both maternal and fetal survival. Reported complications include maternal haemorrhage, disseminated or venom-induced coagulopathy, hypotension, preterm labour, placental abruption, intrauterine fetal death and neonatal mortality.[4,5] Placental separation may result from venom-associated coagulopathy, haemorrhage and microvascular injury, while fetal compromise may also arise from maternal hypovolaemia, hypoxia and impaired uteroplacental perfusion.[6] Although uncommon, fetal loss following snakebite has been documented, particularly when presentation and administration of effective antivenom are delayed.[5,7]

Studies from the major referral snakeenvenoming centre, the snakebite Treatment and Research Hospital, Kaltungo, Gombe, Gombe state, Nigeria has substantiated the clinical burdenof E. ocellatus in North-eastern Nigeria, where the significant case volumes, delayed hospital presentation and inadequate access to effective antivenom are important determinants of adverse outcomes.[2,8] More recently, a six-year study of 77 pregnant women treated at the Snakebite Treatment and Research Hospital, Kaltungo, found that 64% were bitten by carpet vipers and that one-quarter presented with vaginal bleeding, underscoring the distinctive obstetric consequences of envenoming in this population.[9]

We report a 40-year-old multiparous woman at 36 weeks' gestation who presented late following an E. ocellatus bite complicated by placental abruption and fetal demise. This case highlights the “unseen victim” of maternal envenoming—the fetus—and emphasizes the need for early recognition, prompt antivenom therapy, obstetric surveillance and multidisciplinary management of snakebite during late pregnancy.

CASE PRESENTATION

The patient is a 40 year old multigravid woman at 36 weeks gestational age who was bitten by a carpet viper in the left hand. She first sought for herbal help within her locality before presentation at our obstetrics triage 48 hours after the bite with 2/7 history of snake bite with 2 day history of swollen left hand, vaginal bleeding which began 12 hours prior to presentation with associated perception of absent fetal movement 2 hours before presentation. There was no history of abdominal trauma, she is not a known hypertensive, no previous history of abruptio placenta, or collapse. However there is associated history of dizziness.

On examination: she was pale, afebrile, anicteric, acyanosed, well hydrated PR=weak and thready, BP=70/50mmHg, SPO2=96% in room air, swollen left hand.

Abdomen: was tonically contracted and tender, cephalic presented, longitudinal lie, Fetal Heart was absent.

Vaginal examination revealed: Normal female external genitalia, perineum stained with altered blood, no active bleeding, cervical dilatation of 3cm, uneffaced, moderate consistency, central and station -1.

She was managed as a case of Abruptio placenta with intrauterine fetal death complicated by haemorrhagic shock and Acute Kidney Injury in a background hematotoxic snakebite envenomation.

She was resuscitated by A, B, C, …….. Approach, two wide bore cannulas were inserted and intravenous fluid Normal saline 1L fast was commenced and blood samples taken for urgent lab investigations.

Packed cell volume=20(36-48)%, WBC=8.6(4-11) x 10^9/L, Platelets 360(150-450) x10^9/L, Neutrophils 72(55-75)%, Eosinophils 02(1-4)%, Basophils 0(0-1)%, Monocytes 8(2-8)%, Lymphocytes 28(20-40)%, Anisocytosis+, Microcytosis +, Target cells+, Hypochromasia+, LFT: AST 33 up to 45U/L, ALP 310<270U/L, Total Bilirubin 4.7<17Umol/L, Conjugated Bilirubin 3.2<8umol/L, Total Protein 56g/L, Albumin 27g/L.

Urinalysis: Negative for protein, Glucose, Ketones, Leukocytes, Nitrites and Bilirubin, Urobilinogen Normal, P.H 6, Blood ++, EUCR: Sodium 139 (135-145) mmol/L, Potassium 4.4(3.5-5.5) mmol/L, Chloride 96(95-110)mmol/L, Bicarbonate24(20-30)mmol/L, Urea 9.0 (2.5-5.8) mmol/L, Creatinine 315(44-132) umol/L

Viral screening: HBsAg, HCV and Retroviral screening were negative

Clotting profile: PT control: 14(9-14) sec, PT test : >120

PTTK/APTT Control 35(29-35) sec, PTTK/APTT test: >240

Abdominopelvic Ultrasound: confirmed fetal demise with evidence of retro-placental clots. No intramural mass or organomegaly seen.

She progressed from latent to active phase of labour and had vaginal delivery of a macerated still birth 24 hours later with evidence of retro-placental clots, and developed primary postpartum haemorrhage which was managed with Uterotonics and blood products (transfused a total of 3 units of fresh whole blood, 2 units of Fresh Frozen Plasma and 2 units of packed cells). She was co-managed with the Haematology and Nephrology teams.

DISCUSSION

The clinical findings strongly support severe haemotoxic Echis ocellatus envenomation complicated by placental abruption, haemorrhagic shock, acute kidney injury and intrauterine fetal demise (IUFD). The markedly prolonged PT (>120 s) and APTT (>240 s), together with vaginal bleeding and retroplacental clots, are characteristic of venom-induced consumptive coagulopathy (VICC), a major manifestation of E. ocellatus envenoming. The severe anaemia (PCV 20%), hypotension (70/50 mmHg) and elevated creatinine (315 µmol/L) indicate significant systemic consequences of delayed treatment.

Pregnancy increases the clinical consequences of maternal envenomation because fetal survival depends on adequate maternal circulation and an intact uteroplacental interface. VICC-associated placental haemorrhage and abruption, compounded by maternal hypovolaemia and reduced uteroplacental perfusion, can produce acute fetal hypoxia and death. Placental abruption is recognised as an important mechanism of fetal loss following haemotoxic snakebite. The retro placental clots demonstrated ultra-sonographically and at delivery therefore provide a plausible mechanism for the IUFD in this patient.

The approximately 48-hour delay, preceded by seeking herbal treatment, is particularly important. A previous series of pregnant women with E. ocellatus envenomation reported fetal loss in a woman with delayed presentation, whereas timely administration of effective antivenom was associated with favourable maternal and fetal outcomes. More broadly, pregnancy-associated snakebite has been associated with substantial fetal loss, although the available evidence remains predominantly observational.

The patient's subsequent postpartum haemorrhage further illustrates the persistence of haemostatic disturbance after envenomation. Her management with blood and blood products, alongside multidisciplinary care, was an appropriate medical intervention for severe haemorrhage and organ dysfunction. However, specific antivenom remains the cornerstone of treatment, as effective E. ocellatus antivenoms can rapidly restore blood coagulability and reduce spontaneous bleeding. Evidence also indicates that antivenom can be used when clinically indicated during pregnancy and that maternal stabilisation is central to improving fetal outcomes.

This case therefore reinforces the need to treat snakebite in pregnancy as a maternal–fetal emergency, with immediate coagulation assessment, haemodynamic resuscitation, species-appropriate antivenom, blood-product support, renal monitoring and serial fetal surveillance.

CONCLUSION

Maternal Echis ocellatus envenomation in late pregnancy can cause severe coagulopathy, haemorrhage, placental abruption and fetal demise. Delayed presentation worsens outcomes. Early antivenom administration, prompt correction of anaemia and coagulopathy, serial laboratory monitoring, and coordinated multi-speciality maternal–fetal management are essential to improve survival.

PATIENT CONSENT

Written informed consent was obtained from the patient for publication of this case and the accompanying data. Patient identifiers have been removed.

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