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Case Report

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Cerebral infarction following snake bite: observation of a case at the Sourô Sanou University Hospital in Bobo-Dioulasso

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ABSTRACT

Snake bites are a public health problem in developing countries. Neurovascular complications are rare. We report a case of cerebral infarction at the Sourô Sanou University Hospital Center in Bobo-Dioulasso complicating a snake bite. The patient was a 44-year-old left-handed man with no cardiovascular risk factors, presenting with left hemiplegia and expressive aphasia two days after a snake bite (reported to be a viper). Cerebral computed tomography revealed a well-systematized right temporo-parietal cortico-subcortical hypodensity in the deep and superficial territory of the right middle cerebral artery (MCA). He received polyvalent antivenom from Vins Bioproducts Ltd and symptomatic treatment from the first day. The outcome was unfavorable, and the patient died on the eighth day after the bite. Cerebral infarction is a rare complication of snake bite envenomation. However, it is responsible for high morbidity and mortality in our context.

Keywords: Cerebral infarction, envenomation, ophidian, Bobo-Dioulasso, Burkina Faso

INTRODUCTION

Snake bite envenomation remains a major public health challenge in tropical areas, particularly in West Africa. According to the WHO, approximately 5.4 million bites occur worldwide each year, of which 1.8 to 2.7 million result in clinical envenomation, causing between 81.410 and 137.880 deaths annually, as well as permanent disability in many cases.[1,2]

In Burkina Faso, this burden is particularly heavy, especially in rural areas. A recent study in the Cascades region (2016-2021) showed that 42% of patients hospitalized for envenomation had complications, and that delayed access to care, lack of antivenom, and harmful traditional practices (tourniquets, incisions) were strongly associated with these complications.[3] However, access to antivenom has improved recently. In 2023, the government subsidized antivenom serums, reducing their cost to 2.000 CFA francs, a major step forward for the rural population.[4] At the same time, traditional medicine continues to play a central role. An ethnobotanical study conducted in 2025 documents the use of plants by traditional practitioners to neutralize venom. More than 60% of them report effective remedies, and nearly half refer patients to hospitals when their treatment alone is not sufficient.[5]

Despite this progress, serious neurological complications such as strokes remain poorly documented. At the Sourô Sanou University Hospital in Bobo-Dioulasso, two recently reported cases of cerebral hemorrhage following envenomation demonstrate the lethal and disabling potential of these rare forms.[6]

In this context, it is essential to raise awareness of atypical neurovascular forms (ischemic or hemorrhagic) and the need to improve care (diagnosis, antivenom, intensive care), especially in an under-resourced health system. We present here a case of cerebral infarction following a viper bite at the Sourô Sanou University Hospital Center, illustrating one of the most severe and little-known complications in Burkina Faso.

CASE

The patient is a 44-year-old male farmer, independent, left-handed, residing in a rural area (Seguéré, Bama department, Houet province, Guiriko region); with no particular medical history or cardiovascular risk factors, who was bitten by a snake on his right thumb on November 2, 2025, at around 6 p.m. while working in the fields. The snake was described as a viper (killed after the bite and not available for identification). The bite immediately caused local swelling at the site. At the local health facility, he received one ampoule of Vins Bioproducts Ltd's Africa polyvalent antivenom upon arrival, approximately 8 hours after the bite, symptomatic treatment with hydrocortisone 200mg/6 hours, paracetamol 1g/8h, vascular filling with isotonic saline solution 500ml/24h, probabilistic dual antibiotic therapy consisting of ceftriaxone 2g/24h and metronidazole 500mg/8h, and nursing care.

The following day, his condition deteriorated rapidly with the onset of motor deficit in the left side of his body and loss of speech. He was referred to the medical center with a surgical unit in Dandé. There, he received a second dose of polyvalent antivenom from Vins Bioproducts Ltd, the same symptomatic treatment, and was evacuated to the emergency department of the Souro Sanou University Hospital. He was transferred to the neurology department 24 hours after his admission to the emergency department.

The clinical examination carried out three days after the onset of symptoms found that he was in WHO stage III general condition, with normal consciousness and correct hemodynamic parameters: blood pressure of 120/70 mmHg, temperature of 37.5 degrees Celsius, pulse of 70 beats/min, respiratory rate of 20 cycles/min, and peripheral oxygen saturation of 96% in ambient air. The locoregional examination revealed inflammatory swelling of the right thumb with traces of hooks. The neurological examination noted proportional flaccid left hemiplegia affecting the face with motor strength rated at 0/5 in the limbs, abolished osteotendinous reflexes on the left side of the body but present and normal on the right, an indifferent plantar reflex on the left and a flexed reflex on the right, expressive aphasia, left hemineglect, and left lateral homonymous hemianopia, resulting in a National Institutes of Health Stroke Scale (NIHSS) score of 18.

Laboratory tests showed hyperleukocytosis with 12.370 cells/mm³, predominantly neutrophils (73.3%), hemoglobin level of 13.6 g/dl, and platelets at 158.000 cells/mm³. Blood ionogram, urea, and creatinine levels were normal. CRP was not performed; TP/TCA-INR, D-dimers, fibrinogen, and creatine kinase were not performed (blood was uncoagulable after several attempts at blood sampling without a tourniquet).

In the absence of magnetic resonance imaging, which was unavailable in our setting, a cerebral computed tomography performed 24 hours after the onset of the deficit revealed heterogeneous cortico-subcortical hypodensity in the right fronto-parieto-temporal region, well systematized in the superficial and deep territory of the right middle cerebral artery (MCA), with hemorrhagic transformation, a mass effect on neighboring structures, and subfalcial involvement. Figure shows cerebral computed tomography images of the patient's cerebral parenchymal lesions.

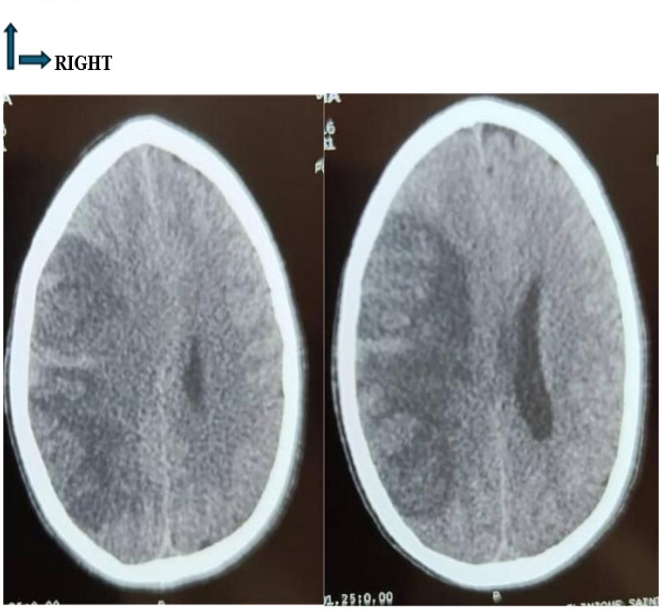

Figure. Cerebral computed tomography images of the patient's lesions

Heterogeneous cortico-subcortical hypodensity in the right fronto-parieto-temporal region, well localized in the superficial and deep territory of the right middle cerebral artery, with hemorrhagic transformation, mass effect on neighboring structures, and subfalcine involvement

In the neurology department, he received an antiplatelet agent based on acetylsalicylic acid 100mg/day, enoxaparin 0.4 ml SC/24 hours, continuation of probabilistic dual antibiotic therapy, paracetamol 1 g/8 hours, isotonic saline solution 500/24 hours, and mannitol 20% 100 ml/8 hours for 48 hours.

The outcome was marked on day 8 after the bite by the patient's death from cardiorespiratory arrest.

DISCUSSION

Cerebral infarction secondary to snake bite envenomation remains a rare but potentially fatal complication. Our patient, a 44-year-old man with no history of cardiovascular disease, fits the profile described in several series in which victims of post-venom stroke are generally young, healthy adults, mainly exposed for agricultural reasons.[7,8] In these studies, male predominance and rural context are regularly reported as exposure factors.

Our patient's profile is consistent with observations from cases published in Asia and Africa, where post-viper bites infarcts occur in young subjects, often without comorbidities.[7,9] This similarity reinforces the hypothesis of a direct causal link between the venom and the neurovascular event, rather than a stroke linked to classic risk factors.

Although the species has not been formally identified by a professional, those around the patient describe it as a viper. The species most commonly found in the geographical context of Burkina Faso is a viper of the genus Echis or Bitis. Viperids are known for their hemotoxic venoms, rich in metalloproteinases and snaclecs responsible for platelet activation and a paradoxical prothrombotic state.[10,11] These mechanisms are well documented in the literature as being capable of inducing cerebral arterial thrombosis, even in the presence of consumptive coagulopathy.[10,11] The cases of ischemic stroke after viper bites described by Vasconez-Gonzalez et al.[7] clearly illustrate this process.

The 48-hour delay between the bite and the onset of neurological deficit is comparable to that reported in series of post-viperid strokes, where strokes usually occur between 24 and 72 hours after envenomation.[7,10] This delay corresponds to the time required for the activation of coagulation cascades, the formation of microthrombi, and their migration to the cerebral territory, particularly to the branches of the MCA.

Our patient had severe coagulopathy ("uncoagulable blood"), a classic marker of severe viper venom envenomation.[10,11] However, the progression to ischemic infarction despite this apparent incoagulability confirms the well-described pathophysiological paradox: local platelet activation with systemic consumption, creating an environment conducive to arterial thrombosis.[10,11] Several authors have reported sylvian infarcts in patients with similar coagulopathy.[11]

Viper venoms contain several bioactive components such as metalloproteinases, serine proteases, and C-type lectin-like proteins (snaclecs), which play a central role in venom-induced coagulopathy. These toxins can activate platelets and coagulation factors, triggering a prothrombotic cascade that promotes the formation of microthrombi in the arterial circulation. Paradoxically, this occurs in the context of systemic consumption of coagulation factors and fibrinogen depletion, a condition often referred to as venom-induced consumptive coagulopathy. This dual mechanism explains why arterial thrombosis, including cerebral infarction, may occur despite apparent incoagulability of blood samples.

In addition to coagulation disturbances, viper venoms can directly damage the vascular endothelium. Metalloproteinases are known to induce endothelial injury, disruption of the vascular basement membrane, and inflammatory responses. This endothelial dysfunction promotes platelet aggregation and localized thrombus formation, creating conditions similar to a vasculitic process within cerebral vessels.

Secondary systemic factors may further contribute to cerebral ischemia after envenomation. Hypotension, dehydration, systemic inflammatory response, or microvascular dysfunction can aggravate cerebral hypoperfusion. In patients with extensive MCA territory involvement, these factors may accelerate infarct progression and worsen neurological outcomes.

The cerebral computed tomography scan showing an infarction in the territory of the right MCA is consistent with the most frequent descriptions in the literature, where post-envenomation infarcts often affect the sylvian territories, sometimes in the form of multiple infarcts.[7,9,11] The lack of MRI is a common limitation in African settings, as highlighted in African venom series where advanced imaging is rarely available.[3,15,16] Nevertheless, the clinical and cerebral computed tomography findings allow a reasonable diagnosis to be made.

Treatment is based on the early administration of antivenom, ideally within the first 6 hours according to WHO recommendations.[16] In our case, a prolonged delay between the bite and the first dose of antivenom was noted, a situation that is common in rural areas and described as a factor for poor prognosis.[15,16] Studies show that even with antivenom, some patients develop strokes or die, particularly in the absence of adequate biological monitoring or imaging.[7,15,16] Our patient's situation illustrates the structural limitations encountered in West Africa: unavailability of hemostasis testing, lack of MRI, inability to consider thrombolysis or neurointensive monitoring; all factors that may have contributed to the unfavorable outcome.

The administration of antiplatelet therapy and prophylactic anticoagulation in this patient despite the presence of coagulopathy may appear paradoxical. However, several reports of viper envenomation describe thrombotic complications resulting from platelet activation and microvascular thrombosis. In this context, low-dose acetylsalicylic acid and prophylactic enoxaparin were introduced to limit further thrombotic extension, particularly given the large territorial infarction. This therapeutic approach remains controversial but has been described in similar cases where the thrombotic component of venom-induced coagulopathy is suspected.

The fatal outcome observed is consistent with the trend reported in several cases of post-viperid stroke, where mortality remains high, often exceeding 30-40% depending on the series.[7,13,15] Systemic complications, uncontrolled coagulopathy, delays in treatment, and the lack of specialized intensive care are the major determinants of mortality.[7,15,16]

Contribution of This Case

This case contributes several important elements to African and international literature:

- Confirmation that ischemic infarction following viper envenomation can occur in West Africa, a region where publications on neurological complications are rare.
- Illustration of the "consumptive coagulopathy+arterial thrombosis" paradox, a mechanism that is increasingly recognized but still under-documented in tropical countries.
- Highlighting of diagnostic and therapeutic challenges in resource-limited areas.
- Importance of strengthening policies to combat envenomation (rapid access to antivenom, imaging, staff training).

CONCLUSION

Cerebral infarction is a rare but severe complication of viper envenomation. Early recognition of neurovascular complications and rapid access to antivenom and specialized care are essential to improve outcomes, particularly in resource-limited settings.

ETHICAL DECLARATIONS

Informed Consent

Written informed consent was obtained from the patient included in this report. Signed consent forms are retained by the authors and are available upon request.

Peer Review Process

This report underwent external peer review.

Conflict of Interest

The authors declare no conflicts of interest.

Financial Disclosure

This case report did not receive any financial support.

Author Contributions

Study Design: ZA; Data Collection: ZA; Manuscript Writing and Editing: ZA; Data Collection: SAA; Clinical Follow-up: SAA; Therapeutic Monitoring: SAA; Manuscript Review: OPV, LDL, NC, MA; Editing and Validation: OPV, LDL, NC, MA.

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