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Acute Ischemic Stroke Following Snakebite: A Case Report

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ABSTRACT

Snakebite envenomation is associated with a wide spectrum of complications, ranging from local tissue damage to life-threatening systemic effects. This case report describes a rare presentation of acute ischemic stroke in a 77-year-old man following envenomation by a suspected *Montivipera raddei albicornuta* in northwestern Iran. The patient initially presented with right lower limb cellulitis, thrombocytopenia, coagulopathy, and acute kidney injury. Within six hours, he developed left-sided hemineglect, hemiparesis, hemifacial weakness, dysarthria, and a decreased level of consciousness. Neuroimaging revealed a non-hemorrhagic infarct localized to the right parietal lobe within the distribution of the middle cerebral artery. This case underscores the potential for thrombotic events, including ischemic stroke, in the context of venom-induced disseminated intravascular coagulation (DIC). It also highlights the need to reassess the efficacy and dosing strategies of available antivenoms and to establish protocols for early identification of thrombotic events following envenomation in high-risk snakebite patients.

Introduction

Snakebite envenomation remains a critical yet under-recognized public health issue worldwide. Globally, snakebites result in an estimated 2.7 million envenomations and up to 138,000 deaths annually, with significant regional variation in species distribution and venom toxicity [1]. In Iran, venomous snakes such as *Vipera lebetina*, *Echis carinatus*, and *Macrovipera lebetina obtusa* are endemic to the northern and western provinces, contributing to hundreds of envenomation cases each year [2]. Herein, we report a case of a 77-year-old Iranian man who developed disseminated intravascular coagulation (DIC) and acute ischemic stroke within six hours of admission following a snakebite from a presumed *Montivipera raddei albicornuta*.

Case presentation

A 77-year-old male from a rural area in Zanjan

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Province, Iran, presented to the emergency department after being bitten on his right lower leg by a yellow-colored snake approximately one meter in length. Based on regional epidemiology, the bite was presumed to be from *Montivipera raddei albicornuta* ("Zanjan rattlesnake"). The patient had no known past medical history. Upon admission, he complained of severe pain, swelling, and ecchymosis localized to the right leg (Figure 1). Initial physical examination revealed significant edema, erythema, warmth, and local tenderness. Vital signs were stable.

The presence of significant coagulopathy, including elevated D-dimer level, prolonged PT/INR, and thrombocytopenia (Table 1), raised strong suspicion for venom-induced disseminated intravascular coagulation (DIC). Furthermore, the progressive normalization of these parameters following antivenom administration strongly supports the diagnosis of secondary DIC triggered by envenomation. The patient also developed acute kidney injury (AKI), likely attributable to volume depletion and direct nephrotoxic effects of the venom, which resolved with intravenous hydration.

Empiric antibiotic therapy with ciprofloxacin 400 mg IV every 12 hours and clindamycin 600 mg IV every 8 hours was initiated to treat suspected cellulitis of the right leg. Color Doppler ultrasound of the right lower limb showed no evidence of thrombosis in the proximal two-thirds; the distal portion could not be visualized due to edema. In accordance with the national Iranian protocol for snakebite management, 35 vials of polyvalent antivenom serum were administered.

Within six hours of the snakebite, the patient developed acute neurological symptoms, including left-sided hemineglect, hemiparesis, hemifacial weakness, dysarthria, and decreased level of consciousness (lethargy). The National Institutes of Health Stroke Scale (NIHSS) score was 15, indicating a moderate to severe stroke. A non-contrast brain CT scan revealed a hypodense area consistent with an acute ischemic infarct localized to the right parietal lobe within the distribution of the middle cerebral artery. He was transferred to the Stroke Care Unit and monitored for two days.

Due to persistent leukocytosis and progression of leg swelling, antibiotics were escalated to meropenem 1 g IV every 12 hours and vancomycin 1 g IV daily for broader coverage. Neurologically, the patient gradually improved with partial recovery of speech and motor function. On hospital day 7, he was fully conscious and oriented, with residual left-sided weakness but improved strength (NIHSS score of 10). Coagulation parameters normalized, and renal

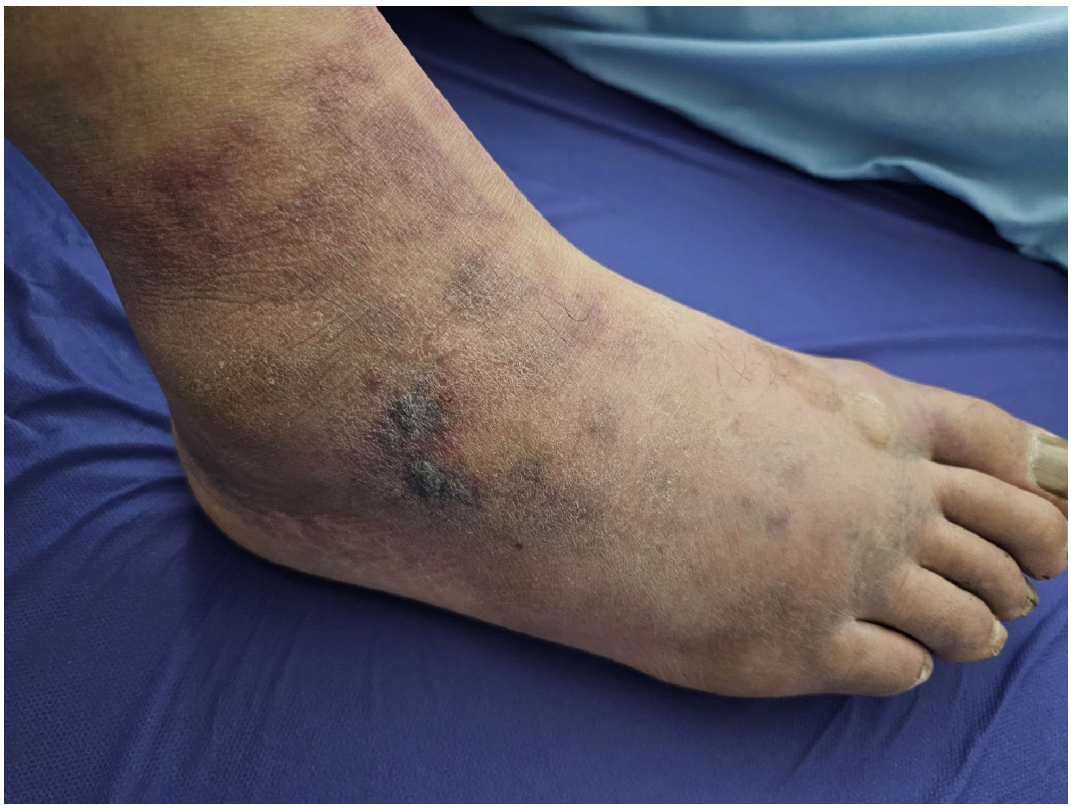

Fig. 1. Right lower limb following snakebite

Table 1. Serial laboratory findings from following snakebite envenomation

Parameter (Unit)	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
Hematology							
WBC (×10³/μL)	16.7	12.7	7.6	9.9	8.3	9.2	6.4
Neutrophils (%)	87.5	83.3	83.5	80.2	79.7	77.8	54.6
Hemoglobin (g/dL)	22.5	20.2	18.8	15.8	15.8	15.3	14.4
Hematocrit (%)	64.3	59.9	55.8	45.8	48.3	44.7	43.8
MCV (fL)	85.7	85.6	87.2	84.8	86.3	84.0	84.23
Platelets (×10³/μL)	30	82	93	98	117	141	193
Coagulation Profile							
PT (sec)	26.0	19.4	15.7	13.0	13.8	13.1	13.0
PTT (sec)	29.0	30.0	30.0	40.0	52.0	33.1	34.0
INR	2.4	2.2	1.27	1.0	1.27	1.02	1.00
D-dimer (μg/mL)	73.0						
Renal Function							
Creatinine (mg/dL)	1.1	1.7	1.4	0.9	0.8	1.0	0.8
BUN (mg/dL)	22.6	30.0	45.0	47.0	40.0	30.0	12.1
Biochemistry & Others at admission							
Calcium (mg/dL)	8.7	Phosphate (mg/dL)	4.4	CRP (mg/L)	49.0	ESR (mm/hr)	46.0
Reticulocyte (%)	0.4	LDH (U/L)	770	Total Bilirubin (mg/dL)	0.8	Direct Bilirubin (mg/dL)	0.2
AST (U/L)	24.3	ALT (U/L)	16.7	ALP (U/L)	182	CPK (U/L)	134

function remained stable. The patient was discharged with recommendations for outpatient neurology follow-up and physical rehabilitation.

Discussion

Cerebral infarction and hemorrhage are primary contributors to mortality in cases of viper envenomation, with both hemotoxic and vasculotoxic components of the venom playing a role in these life-threatening outcomes [3]. Our patient developed an acute non-hemorrhagic cerebral infarction within six hours of envenomation, coinciding with laboratory evidence of disseminated intravascular coagulation (DIC). The pathophysiology of ischemic stroke following viper envenomation is multifactorial, involving coagulopathy, hemoconcentration, and vascular injury [4].

Viper venom contains metalloproteinases that directly activate coagulation factors, leading to massive thrombin generation and venom-induced consumption coagulopathy (VICC). This process depletes fibrinogen and platelets while producing markedly elevated D-dimer levels, reflecting systemic fibrinolysis. Concurrently, fluid loss and endothelial permeability triggered by venom-induced capillary leak may cause hemoconcentration and hyperviscosity, further predisposing patients to thrombosis. Additionally, viper hemorrhagins exert direct cytotoxic effects on vascular endothelium, promoting endothelial dysfunction, exposure of subendothelial collagen, and platelet aggregation—conditions conducive to cerebral thrombus formation.

Leukocytosis and rhabdomyolysis have also been significantly associated with coagulopathy, and both hemolysis and rhabdomyolysis are known risk factors for developing DIC, further reinforcing the multifactorial nature of venom-induced thrombotic complications [5]. These intersecting mechanisms contribute to the development of cerebral ischemia, even in patients without traditional vascular risk factors [6]. Similar cases of acute ischemic stroke occurring in the setting of venom-induced DIC have been reported in the literature, further supporting a causal link between coagulopathy and cerebrovascular events following snake envenomation [7–11].

Treatment with antivenom plays a crucial role in preventing and potentially reversing neurological complications associated with snakebite envenomation [3]. The Iranian polyvalent antivenom, produced by the Razi Vaccine and Serum Research Institute, is an equine-derived immunoglobulin preparation formulated to neutralize the venoms of five clinically significant venomous snakes in Iran: *Macrovipera lebetina*, *Montivipera raddei*, *Echis carinatus*, *Pseudocerastes persicus*, and *Gloydius halys caucasicus* [12].

Although the patient received antivenom therapy promptly upon admission, the subsequent development of ischemic stroke suggests that the administered dose may have been insufficient or that the efficacy of the antivenom itself may be limited—

an issue highlighted in other studies [13]. This case adds to the growing literature on rare thrombotic complications of viper bites and underscores the need for antivenom optimization and early recognition of thrombotic events in envenomated patients.

Ethical Considerations

Ethics approval

This study was approved by Zanjan University of Medical Sciences, Zanjan, Iran. Written informed consent was obtained from the patient for participation in the study and the rights of the subject were protected.

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Conflict of Interests

The authors have no conflict of interest to declare.

Authors' Contribution

All authors contributed to the preparation of data and finalization of this article.

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