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CASE REPORT

Occult Hemotoxic Envenomation presenting as Fulminant Septic Shock with Disseminated Intravascular Coagulation in a Young Male: A Diagnostic Challenge.

Sishir Siwakoti [1], M.B.B.S., M.D., Fellowship Critical Care Medicine, Laliteshwar Shah [1], M.B.B.S., Sarbagya Manandhar [2], M.B.B.S., Shristee Chudal [3], M.B.B.S., M.D., Fellowship Neurocritical Care.

[1] Department of Anesthesia and Critical Care, Nepal Medical College and Teaching Hospital, Attarkhel, Jorpati, Kathmandu, Nepal.
[2] Department of Internal Medicine, Nepal Medical College and Teaching Hospital, Attarkhel, Jorpati, Kathmandu, Nepal.
[3] Department of Anesthesia and Critical Care Medicine, HAMS Hospital, Kathmandu, Nepal.

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Corresponding author:
Dr. Sishir Siwakoti, M.B.B.S., M.D., Fellowship Critical Care Medicine
Lecturer, Department of Anesthesia and Critical Care, Nepal Medical College, Jorpati, Kathmandu, Nepal
Email: mesishir@gmail.com
Phone: +977-9843290433

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ABSTRACT

We report a case of a previously healthy 17-year male who presented with a sudden onset of fever, severe myalgia, altered sensorium, and rapidly progressive shock. Initial evaluation revealed severe metabolic acidosis, thrombocytopenia, acute kidney injury, and markedly elevated inflammatory markers, raising suspicion for fulminant septic shock with multiorgan dysfunction. Broad-spectrum antimicrobial therapy and intensive supportive care was initiated. Microbiological cultures from blood, urine, and cerebrospinal fluid were sterile.

The clinical course was further complicated by early development of purpura fulminans and laboratory evidence of disseminated intravascular coagulopathy. A diagnostic turning point occurred following the incidental identification of two punctate scabbed lesions over the lateral aspect of the foot, suggestive of a possible bite mark. In the context of sterile cultures, abrupt disease progression, and consumptive coagulopathy, occult snake bite with venom-induced consumptive coagulopathy was strongly considered.

The patient demonstrated gradual recovery with vasopressor and other organ support in ICU. This case highlights a rare but important diagnostic dilemma in which occult hemotoxic envenomation closely mimicked fulminant septic shock. In endemic regions, snakebite envenomation should be considered in patients presenting with hyperacute shock, coagulopathy, and multiorgan dysfunction, particularly when microbiological evidence of infection is lacking.

Keywords: *Septic shock, snakebite envenomation, venom-induced consumptive coagulopathy.*

INTRODUCTION

Acute shock with multiorgan dysfunction in an otherwise healthy adolescent is a medical emergency that most commonly raises suspicion of severe sepsis or septic shock.[1] These conditions are characterized by circulatory instability and organ dysfunction resulting from a dysregulated host response to infection and remain a major cause of morbidity and mortality in critical care settings.[1,2] Early recognition and prompt treatment are essential; however, several non-infectious conditions may closely mimic this presentation, creating significant diagnostic challenges in the absence of an identifiable source of infection.

Snakebite envenomation is an important yet frequently under-recognized cause of acute systemic illness in South Asia and continues to be a major public health problem in endemic regions.[3-5] Hemotoxic envenomation, particularly from viper species, may result in venom-induced consumptive coagulopathy (VICC), a distinct coagulopathic syndrome characterized by rapid depletion of clotting factors and clinical features that overlap substantially with disseminated intravascular coagulation (DIC), including thrombocytopenia, coagulopathy, shock, and multiorgan dysfunction.[6-8]

In endemic regions, the absence of a witnessed bite, clear history of envenomation, or prominent local signs may delay diagnosis and lead to misclassification as septic shock or other critical illnesses.[3,6] Occult hemotoxic envenomation presenting primarily with systemic manifestations remains particularly challenging to recognize. This case highlights the diagnostic dilemma of differentiating VICC from fulminant septic shock in a previously healthy adolescent presenting with rapidly progressive shock, disseminated intravascular coagulation-like features, and multiorgan dysfunction.

CASE PRESENTATION

A 17-years male previously healthy male from Kathmandu presented with an acute onset of severe generalized myalgia, arthralgia, and back pain, followed by chills, rigors, and undocumented fever. Later in the evening, he had multiple episodes of non-projectile vomiting. During the night, his family noted increasing restlessness, confusion, and inability to respond appropriately. By early morning, he was unable to stand or support himself, prompting transfer to a local hospital approximately 13 hours after symptom onset. There was no history of recent travel, animal exposure, or known insect or snake bite.

At the local hospital, he was noted to be drowsy but arousable (GCS 14/15), dehydrated, hypotensive, tachycardic, and hypoxic. Initial investigations revealed metabolic acidosis, elevated lactate, leukocytosis, thrombocytopenia, and acute kidney injury (Table 1). He received intravenous crystalloid resuscitation and was referred to our center.

Table 1. Clinical and key laboratory parameters during hospitalization

Parameter	Local Hospital	ICU Day 1	Day 2	Day 3	Day 4	Day 5
Blood Pressure (mm Hg)	80/50	70/40	90/60 (on nor-adrenaline and vasopressin)	90/60 (on nor-adrenaline)	100/60 (off vasopressor)	100/60
Heart Rate (bpm)	114	112	130	115	100	95
SpO_2 (%)	86 (RA)	81 (O_2)	93	92	92	94
Temperature (°F)	98	104	101	98	98	98
GCS	14	13	14	15	15	15
Lactate (mmol/L)	12	10.2	3.2	2.2	1.2	—
Creatinine (mg/dL)	2.3	3.29	3.06	2.31	1.30	0.93
Platelets (/µL)	120,500	105,000	70,000	60,000	90,000	Near normal
INR	1.2	—	4.07	—	1.78	—
AST (U/L)	58	130	—	—	967	573
ALT (U/L)	41	61	—	—	624	503
Total Bilirubin (mg/dL)	0.5	0.7	—	—	4.10	4.39

On arrival to our emergency department approximately 17 hours after symptom onset, the patient was in decompensated shock with severe global hypoperfusion. Blood pressure was 70/40 mmHg with a feeble pulse. He was tachypneic (38/min), hypoxic (SpO_2 81% on supplemental oxygen), febrile (104°F) and was disoriented (GCS 13/15). Capillary refill time was approximately 12 seconds with marked peripheral mottling. Few non-blanching petechiae were noted in bilateral lower limbs.

He was immediately admitted to the intensive care unit. Aggressive fluid resuscitation and vasopressor support (norepinephrine and vasopressin) were initiated. Empirical antimicrobial therapy included ceftriaxone 2 grams 12 hourly, vancomycin 1 gram 8 hourly, doxycycline 100 mg 12 hourly, acyclovir 800 mg 8 hourly and dexamethasone 8 mg 6 hourly to cover meningoencephalitis. Invasive monitoring was established. Point-of-care ultrasound showed an A-profile lung pattern with mild global left ventricular dysfunction. Laboratory findings confirmed worsening metabolic acidosis, acute kidney injury, thrombocytopenia, and markedly elevated inflammatory markers. Cerebrospinal fluid analysis was normal, leading to discontinuation of acyclovir. Within 24 hours, he developed rapidly progressive purpuric lesions over the extremities, consistent with purpura fulminans. DIC was confirmed clinically and biochemically. Given the severity of illness, antimicrobial therapy was escalated to meropenem and polymyxin, and hydrocortisone replaced dexamethasone. Despite initial instability, gradual improvement in perfusion and renal function was noted. By day 3, the patient regained full consciousness (GCS 15/15), vasopressor requirement decreased, and urine output improved significantly. However, all blood, urine, and cerebrospinal fluid cultures remained sterile.

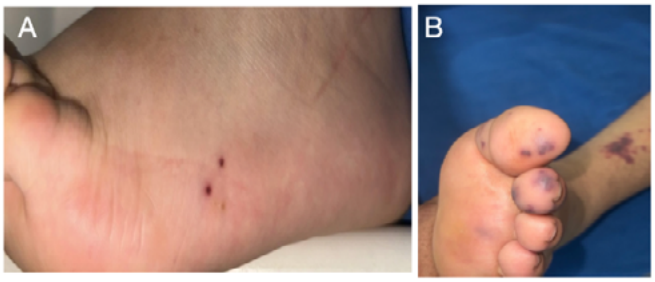


Figure 1. Panel A: Two closely spaced punctate lesions over the lateral aspect of the foot; **Panel B:** Purpuric lesions.

During routine nursing care, two closely spaced punctate scabbed lesions were identified over the lateral aspect of the left foot (Figure 1). These were distinct from surrounding purpuric lesions and raised suspicion of a possible bite mark. It was not associated with local swelling or gangrene. In the context of sterile cultures, hyperacute presentation, and consumptive coagulopathy, occult hemotoxic envenomation was strongly considered. Following this diagnostic reassessment, polymyxin was discontinued while other antimicrobial therapy was continued. The patient showed steady clinical recovery. As the bite marks was noticed on day 3 and patient started to get better from day 3 onwards. Hence antivenom wasn't administered in this patient. By day 4, vasopressors were discontinued. Echocardiography revealed global hypokinesia with a Left ventricular ejection fraction of 30%. Liver dysfunction became evident with elevated bilirubin and transaminases, likely secondary to systemic insult. The patient continued to improve and was transferred out of ICU on day 5. He was discharged on day 8 in stable condition with follow-up planned for cardiac dysfunction.

DISCUSSION

This case describes a rapidly progressive illness in a previously healthy adolescent presenting with acute shock, multiorgan dysfunction, and severe coagulopathy. The initial working diagnosis was septic shock, supported by hypotension, tachycardia, lactic acidosis, and altered mental status. However, the absence of microbiological growth and the atypical clinical evolution prompted reconsideration of alternative etiologies, particularly VICC.[6,8]

Sepsis remains the most common cause of distributive shock in critically ill patients. However, sterile blood, urine, and cerebrospinal fluid cultures, along with the hyperacute onset of illness within hours and absence of an identifiable infectious focus, made classical bacterial sepsis less likely.[1]

A major diagnostic challenge in this case was distinguishing sepsis-induced DIC from VICC. Both conditions share overlapping features, including thrombocytopenia, elevated D-dimer (>10µg/mL), prolonged coagulation parameters, and multiorgan dysfunction.[9] However, VICC results from procoagulant toxins in viper venom, leading to rapid depletion of clotting factors and a distinct consumptive pattern.[6,7,8] Unlike sepsis-associated DIC, VICC typically has a hyperacute onset and may show faster recovery with supportive management.[6] The marked hypofibrinogenemia (100mg/dL), elevated INR, and early purpura fulminans in this case were more consistent with a toxin-mediated process.[8,9]

Snakebite envenomation remains an under-recognized cause of acute coagulopathy in South Asia. Pit viper species can cause minimal or absent local signs while producing severe systemic toxicity, including VICC, acute kidney injury, and shock.[3,5,7] The absence of a definite bite history highlights the diagnostic challenge, particularly in urban settings. Similar reports describe systemic manifestations preceding recognition of envenomation.[3,5]

The patient also developed features consistent with septic cardiomyopathy, including reduced ejection fraction, global hypokinesia, and elevated NT-proBNP (7,500 pg/mL). Septic cardiomyopathy is well described in severe sepsis and is attributed to cytokine-mediated myocardial suppression and mitochondrial dysfunction.[2] However, similar cardiac dysfunction has been reported in systemic envenomation, likely due to direct venom toxicity and inflammatory mediator release, making clinical distinction difficult.[2,7]

Purpura fulminans in this case reflected severe dermal vascular thrombosis associated with DIC. While commonly

associated with bacterial sepsis, it has also been described in toxin-mediated consumptive coagulopathies.[9] The rapid progression of non-blanching purpura along with laboratory evidence of coagulation failure strongly supported a systemic consumptive process.[8,9] Acute kidney injury further supported severe systemic involvement. Viper envenomation may cause renal dysfunction through hypotension, hemolysis, pigment nephropathy, and direct nephrotoxicity.[7] These mechanisms overlap significantly with septic shock, complicating early differentiation between infectious and toxin-mediated etiologies.[1,7]

A key diagnostic pivot occurred when two punctate scabbed lesions were identified on the left foot during routine examination. In the setting of sterile cultures, rapid coagulopathy, and multiorgan dysfunction, this raised suspicion of occult envenomation. Green pit vipers are known to inhabit the Kathmandu region and may present with subtle local signs.[10] Systemic envenomation can trigger a severe inflammatory response with cytokine release, explaining fever, shock, and markedly elevated procalcitonin (>100 ng/mL), closely mimicking sepsis. VICC remains the hallmark feature, producing laboratory and clinical features identical to DIC.[6,7,8] Multiorgan dysfunction, including AKI and myocardial depression, is well documented in viper envenomation and further supports this pathophysiology.[3,7] However, there was no direct evidence of bite. Lack of local tissue destruction and no other tissue signs is an argument against hemotoxic envenomation.[3]

This case highlights the importance of maintaining a broad differential diagnosis in patients presenting with undifferentiated shock and coagulopathy. Although sepsis is most common, envenomation should be considered in endemic regions, particularly when disease progression is hyperacute and cultures remain sterile.[1,3,5] Differentiating VICC from sepsis-induced DIC is clinically critical, as management strategies differ significantly.[6,8,9]

CONCLUSION

This case illustrates a rare diagnostic dilemma where occult hemotoxic envenomation closely mimicked fulminant septic shock. Early recognition of VICC, particularly in endemic regions, and careful reassessment of atypical shock presentations are essential. Comprehensive clinical evaluation which includes meticulous head to toe examinations combined with laboratory and imaging correlation remains central to accurate diagnosis and timely management.[3,6,8]

AUTHOR CONTRIBUTIONS

SS: Manuscript drafting, literature review, manuscript revision and final submission; **LS**: Manuscript drafting, literature review, discussion; **SM**: Detailed history taking, case presentation and discussion; **SC**: Critical revision, drafting of manuscript.

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CONFLICTS OF INTEREST

None

CONSENT

Written informed consent was obtained from the patient's father for publication of this case report and any accompanying clinical information and images.

USE OF GENERATIVE AI

Artificial intelligence (AI)-assisted tools were used for language refinement and editing. All clinical information, interpretations, analyses, and conclusions were reviewed and approved by the authors. The authors take full responsibility for the content of this manuscript.

ORCID IDs

Sishir Siwakoti https://orcid.org/0009-0001-1000-5409

Laliteshwar Shah https://orcid.org/0009-0003-4442-6898

Sarbagya Manandhar https://orcid.org/0000-0002-1731-8299

Shristee Chudal https://orcid.org/0009-0002-4262-7306

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