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CASE REPORT

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Acute splenic hematoma: A rare complication of snake bite

Niranjan Thapa[1] | **Shiva K. Regmi**[1] | **Sunil Basukala**[2] | **Sandip Paudel**[1] | **Oshan Shrestha**[1] | **Bipin Mehta**[1] | **K. C. Manoj**[1] | **Kaushal K. Singh**[1]

[1]Nepalese Army Institute of Health Sciences, Kathmandu, Nepal

[2]Department of Surgery, Nepalese Army Institute of Health Sciences, Kathmandu, Nepal

Correspondence
Shiva K. Regmi, Nepalese Army Institute of Health Sciences, Kathmandu, Nepal.
Email: shivkeshab6@gmail.com

Key Clinical Message

Splenic hematoma secondary to snake bite is a potential complication due to snake envenomation and poses a significant risk to the health of the patients. Although relatively rare, this complication once diagnosed, should be initiated with timely anti-venom administration and supportive care. Clinicians must be aware of any signs of hematological abnormalities in snakebite patients, as the development of splenic hematoma can have serious implications for patient outcomes. Awareness of this potential complication and multidisciplinary collaboration among medical teams are crucial to ensuring effective management and optimal patient care in these clinical scenarios. Understanding this concern can improve patient prognosis and advance the overall approach to snakebite management in healthcare settings.

KEYWORDS

anti-venom, hematological abnormalities, snake bite, splenic hematoma, venom-induced consumptive coagulopathy

1 | INTRODUCTION

According to the World Health Organization, snakebite remains a neglected public health concern in numerous tropical and subtropical regions.[1] The World Health Organization has designated snakebite envenomation (SBE) as a high priority neglected tropical illness.[2] Rural areas are more likely to experience SBE-related fatalities, impairments, and socioeconomic effects.[3,4] Elapid bites mostly cause neurotoxic consequences, while viper snake bites primarily cause hemotoxic effects.[5] Hemocoagulation failure: venom-induced consumption coagulopathy (VICC), most frequently with defibrination and consequent potential bleeding and organ hematoma forms, is one of the snakebites envenoming manifestations.[6] Worldwide, VICC occurs due to bites by vipers, most Australasian elapid snakes, and a few species of colubrid.[7] VICC results from the combined effects of toxins, which include phospholipase A2, snake venom serine protease, and snake venom metalloprotease.[8,9] Splenic rupture because of VICC is a relatively rare complication. Due to this organ's fragility, splenic rupture is frequently brought on by trauma. Atraumatic, non-traumatic, or spontaneous splenic rupture, which is less common due to a variety of factors, can also occur without traumatic injury.[10] Non-traumatic splenic rupture and bleeding after snakebite has been described so far in three cases, of which only in one case initially intact spleen with massive hemoperitoneum and splenectomy necessity has occurred.[11,12] Hence, we report this unusual case of spleen laceration to a depth of approximately 4.2 cm involving 25%–50% of the spleen and rupture of the splenic capsule

along peri splenic hematoma following Russell viper bite. We believe the importance of this paper will bring an effective medical prospect in treating non-traumatic splenic rupture among medical practitioners.

2 | CASE REPORT

A 59-year-old known hypertensive male under medication was brought to the emergency department after a history of snake bite and sustained a bite mark on the anterior aspect of his left leg while cutting grass during the evening. He was immediately transported to a nearby hospital, upon admission to the hospital, the patient was initially hemodynamically stable. The patient's party had brought a photograph of Russell's viper.

On local examination, no visible bite marks or local pain were observed at the site of the bite. As a therapeutic measure to counteract the effects of the snakebite, one vial of anti-venom was administered, and the patient's vital signs were closely monitored.

Following a stable condition during the initial 2 days, the patient was discharged from the hospital. However, after 6 h of discharge, he presented with a sudden onset of epigastric pain and multiple episodes of nonbilious, nonbloody vomiting. There was no recent specific history of illness or trauma. Recognizing the deterioration of his health condition, the medical team referred him to a more specialized facility for comprehensive evaluation and treatment.

Upon admission to our hospital (Shree Birendra Hospital), the patient's condition significantly deteriorated, evident by signs of notable distress. A thorough physical examination revealed the patient was in an anxious state, ill-looking, and dehydrated with a blood pressure of 110/70 mmHg, a pulse rate of 118 beats per minute, a respiratory rate of 20 breaths per minute, and a body temperature of 37.3°C. During the Systemic examination, the abdomen was found to be soft, with tenderness localized to the left hypochondriac region, without distention. Bowel sounds were present. Heart sounds and lung fields were unchanged, and the patient showed grossly intact neurological function.

2.1 | Diagnostic assessments

During admission, results for various blood parameters with normal range are shown in Table 1.

A focused ultrasonography examination revealed peri splenic hematoma and the presence of intraperitoneal fluid accumulation in perihepatic, peri splenic, bilateral paracolic gutters, and pelvis.

CECT of abdomen showed laceration in the spleen with 25%–50% hypo enhancing area within; rupture of the splenic capsule with peri splenic hematoma—feature suggestive of AAST grade III splenic injury. No obvious active contrast extravasation is seen in Figure 1.

CT scan of abdomen with angiography showed splenic hematoma with capsular rupture and moderate to marked

TABLE 1 Results for various blood parameters with normal range.

S. No.	Test	Result	Normal range
1	Complete blood count (CBC)	11,765 cells per μL	4000–11,000 cells per μL
2	Hemoglobin (Hb)	5.4 gm/dL	M: 13–17 gm/dL F: 12–15 gm/dL
3	Hematocrit (Hct)	21.3%	M: 41% to 50% F: 36% to 48%
4	Platelet	115,000 cells per μL	1,50,000-4,00,000 cells per μL
5	Aspartate aminotransferase (AST)	17.8 U/L	0.0–35.0 U/L
6	Alanine aminotransferase (ALT)	11.2 U/L	0.0–45.0 U/L
7	Creatinine	1.18 mg/dL	0.7–1.30 mg/dL
8	Creatinine kinase (CK)	45 U/L	22 to 198 U/L
9	Prothrombin time (PT)	28.6 s	10–14 s
10	International Normalized Ratio (INR)	3.32	0.87–1.24
11	activated Partial Thromboplastin Time (aPTT)	42.7 s	28.0–45.0 s
12	D-dimer	23 μg/mL	0–0.5 μg/mL
13	Reticulocyte count	2.2%	0.5% to 2.5%
14	Antithrombin III	53%	80% to 120%
15	Fibrinogen	56 mg/dL	200–400 mg/dL

hemoperitoneum with moderate bilateral pleural effusion with passive basal atelectasis. Figure 2.

2.2 | Treatment

The patient was initially assessed and treated in the emergency room. He was placed on nil per oral, and intravenous administration of normal saline was used to manage fluids. The pain was addressed with the administration of hyoscine butyl bromide, accompanied by pantoprazole to manage any potential gastric issues. Intravenous antibiotics, specifically meropenem and teicoplanin were administered for infection. Subsequently, surgical consultation was done for epigastric pain and vomiting and advised for CECT of the abdomen and transferred to the surgical intensive care unit. Initially, one unit of packed red blood cells and two units of fresh frozen plasma transfusion were done with hemoglobin monitoring every 6 h. The next day, another unit of packed red blood cell transfusion was done until the patient's vital signs stabilized. Antivenom therapy was administered until the fourth day of hospitalization, leading to the normalization of coagulation function. On the fifth day of hospitalization, a follow-up abdominal CT scan was performed, which revealed no active bleeding from the splenic artery, with a marked reduction in the hemoperitoneum. After the CT scan, the patient was transferred to the general hospital ward and ultimately discharged on the ninth day of hospitalization, having experienced an uncomplicated recovery.

3 | DISCUSSION

VICC is the most common, also the most important systemic effect of snake envenomation worldwide. Vipers and Elapids, particularly Australian Elapids have been linked with VICC.[13] While blunt abdominal trauma remains the leading cause of splenic injury, non-traumatic splenic injury can also occur due to coagulation disorders or splenic neoplasia.[14] However, coagulation disorder secondary to snake envenomation leading to splenic hematoma is relatively rare and not adequately reported in the literature.

The snake venom components acting on the coagulation system are classified according to the part of the coagulation pathway they act upon. They include factor V activators, factor X activators, prothrombin activators, and fibrinogenases.[15] These venom components result in varying degrees of coagulopathy some of which recover without a sequel, while others can progress to bleeding or even major hemorrhage having fatal consequences.[16]

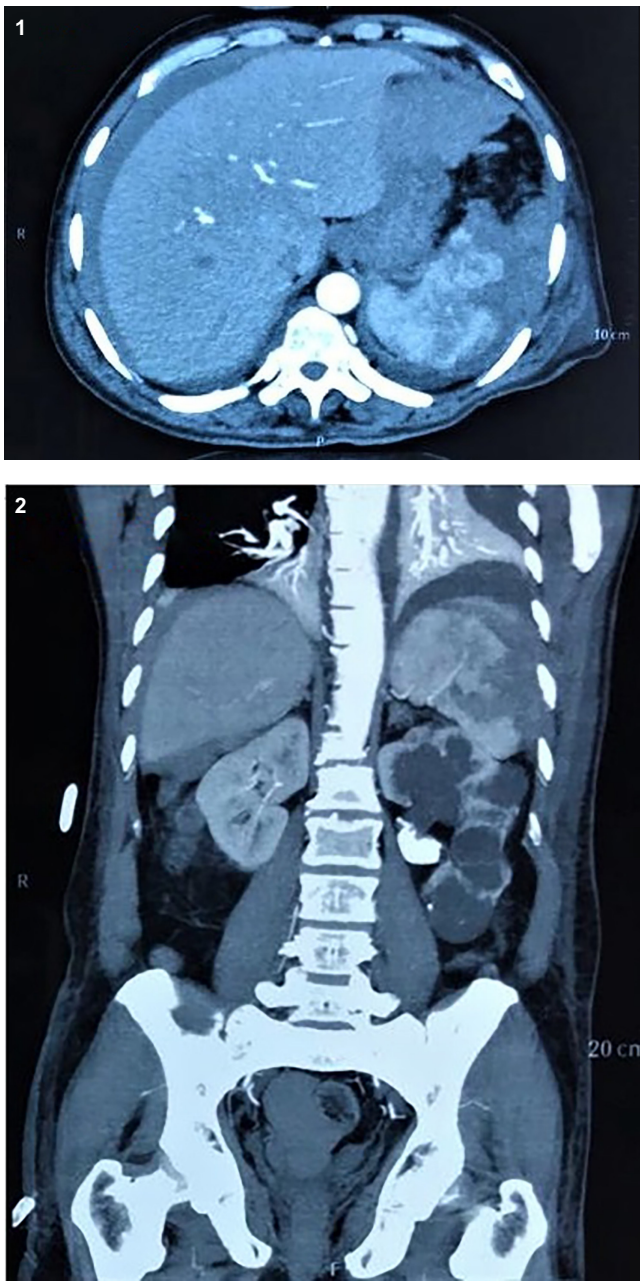

FIGURE 1 Abdominal computed tomography reveals splenic laceration, ruptured capsule, and peri-splenic hematoma (approx. 7.2 cm) (1) associated with intraperitoneal hemorrhage (2).

Splenic hematoma is a rare complication of consumption coagulopathy in snake bites.[17]

Existing knowledge suggests that in certain snakebites, the components of toxins such as metalloproteases, serine proteases, and phospholipase A2 might affect the permeability of the vessels. This in turn may lead to a bleed in the spleen, increasing intrasplenic pressure and eventually a ruptured spleen.[18]

Patients of splenic injury usually present with pain in the left hypochondria, pleuritic chest pain, tachycardia, or features of hypotension. The left hypochondriac

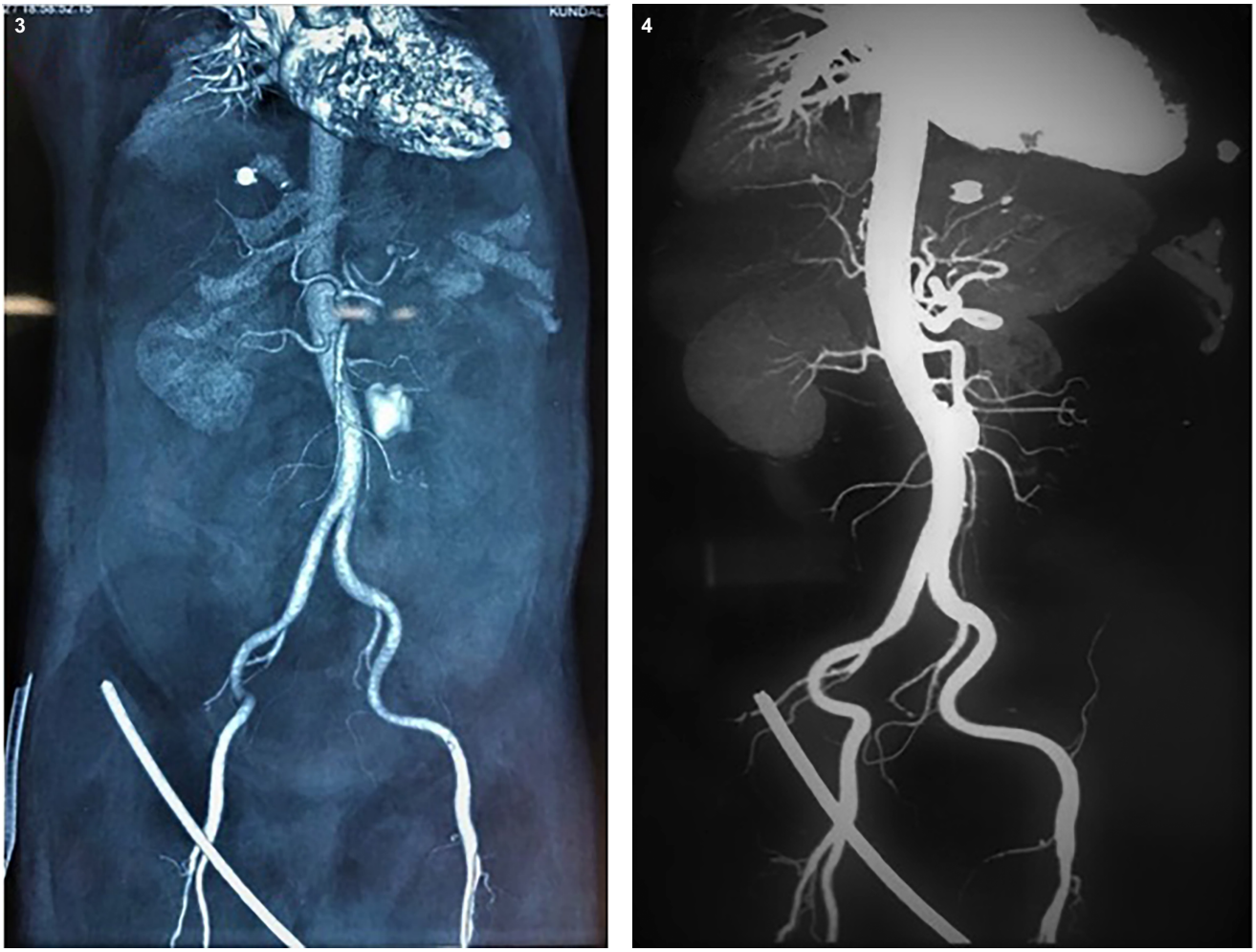

FIGURE 2 Abdominal CT angiography showing splenic hematoma measuring 6.8 × 5.5 × 4.5 cm, accompanied by capsular rupture and moderate to marked hemoperitoneum.

pain usually refers to the left shoulder in approximately 20% of the patients, called Kehr's sign.[19] Our patient was presented with abdominal pain, headache, diaphoresis, and nausea. Upon examination, tenderness was found localized in the abdominal region suggesting abdominal pathology. However, localization of injury was not possible with vague clinical features and examination findings alone, and further tests were conducted.

Blood coagulation profile plays an important role in the diagnosis and monitoring of patients with VICC. Assessment of fibrinogen levels, D-dimer levels, Prothrombin time (PT), and International Normalized Ratio (INR), are important in the diagnosis of coagulopathies. D-dimer (DD), is the primary enzymatic degradation product of fibrin and is found markedly elevated in consumptive coagulopathies, thus helping differentiate it from other types of coagulopathies.[15] As fibrinogen is consumed by the fibrinogenase component of the venom, their levels are found low or even absent. Alongside, PT and INR are found markedly elevated. The blood profile of our patient demonstrated increased PT, INR, decreased fibrinogen levels, and markedly elevated d-dimer levels suggesting consumptive coagulopathy resulting from envenomation.

Computed tomography (CT) scan is the mainstay of diagnosis of intra-abdominal injuries and grading of splenic injuries is based on the AAST grading system.[20,21] A CT scan of the abdomen in our patient revealed spleen laceration of depth approximately 4.2 cm involving 25%–50% of the spleen and rupture of the splenic capsule along with hyperdense collection in the peri splenic region suggestive of hematoma. Upon consideration of radiological findings, a diagnosis of AAST grade IV spleen injury was made.

When VICC results in organ hemorrhage, treatment should be initiated simultaneously for both. VICC should be managed with anti-venoms, which facilitate the initiation of fresh frozen plasma therapy to replenish lost coagulation factors.[22] In our case, administration of antivenom was done continuously for 4 days until the condition of the patient improved.

On the other hand, surgery is the most employed treatment for splenic injury. However, the evidence of Overwhelming Post splenectomy Infection in 20% of splenectomy cases for hematological disorders has brought to light the importance of preservation of the spleen.[23] Thus, non-operative management (NOM) is now the standard of treatment in cases of splenic injuries. NOM involves bed rest and a mild diet for patients with minor injuries, but injuries of higher grades require angioembolization for control of hemorrhage.[24] Our patient had radiographic features suggestive of grade III splenic injury; still, conservative management was found successful in our case. Continuous infusion of packed red blood cells and fresh frozen plasma with a strict evaluation of vitals was done. Eventually, the vitals of the patient improved and upon follow-up CT abdomen on the fifth day, no active bleeding was observed from the splenic artery.

4 | CONCLUSION

This case highlights the potential severity and complications associated with envenomation. Snake bites can lead to various systemic effects, including hematological disturbances, which in rare cases can result in the development of splenic hematoma. Timely administration of anti-venom and supportive care is essential to neutralize the venom's effects and prevent the risk of life-threatening complications.

AUTHOR CONTRIBUTIONS

Niranjan Thapa: Conceptualization; formal analysis; investigation; methodology. **Shiva K. Regmi:** Data curation; methodology; project administration; resources. **Sunil Basukala:** Conceptualization; formal analysis. **Sandip Paudel:** Conceptualization; methodology. **Oshan Shrestha:** Conceptualization; methodology; resources. **Bipin Mehta:** Conceptualization; data curation; formal analysis. **K. C. Manoj:** Conceptualization; data curation; formal analysis; resources. **Kaushal K. Singh:** Conceptualization; formal analysis; investigation.

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CONFLICT OF INTEREST STATEMENT

No conflict of interest.

DATA AVAILABILITY STATEMENT

All the findings are present within the manuscript.

CONSENT

Written informed consent was obtained from the patient to publish this report by the journal's patient consent policy.

ORCID

Niranjan Thapa https://orcid.org/0000-0001-9043-3368
Shiva K. Regmi https://orcid.org/0009-0008-7556-4392
Sandip Paudel https://orcid.org/0009-0002-2996-1043
Oshan Shrestha https://orcid.org/0000-0002-8655-9168

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