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A Case of Non-Operative Management of Atraumatic Splenic Hemorrhage Due to Snakebite Venom-Induced Consumption Coagulopathy

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Male, 62
Final Diagnosis: Splenic hemorrhage
Symptoms: Hypotension • syncope
Medication: —
Clinical Procedure: Splenic artery angio-embolization
Specialty: Critical Care Medicine

Objective: Rare disease

Background: Snakebite envenoming results from injection of a mixture different toxins following snakebite. Coagulopathy and life-threatening hemorrhage can occur, or venom-induced consumption coagulopathy (VICC). A rare case is presented of spontaneous splenic hemorrhage due to VICC that was successfully treated by non-surgical splenic artery embolization.

Case Report: A 62-year-old man was admitted to the emergency department after an episode of dizziness and loss of consciousness following a snakebite. He was transferred to our hospital with hypotension and an abnormal blood coagulation test. On admission, he was hypotensive, with reduced hemoglobin and hematocrit levels, but did not complain of abdominal pain. The occult source of bleeding was identified by abdominal computed tomography (CT) as splenic hemorrhage. Treatment began with the administration of antivenom and blood transfusion. Splenic artery angio-embolization was performed to control the bleeding and was without complication.

Conclusions: Snakebite envenoming associated with VICC is a serious and life-threatening condition. Because of the possibility of associated occult bleeding from internal organs or blood vessels, imaging studies should be performed as soon as possible. For patients who are hemodynamically stabilized and have atraumatic hemorrhage from the spleen, non-operative treatment using angio-embolization may be performed with intensive monitoring and follow-up.

MeSH Keywords: Blood Coagulation Disorders • Embolization, Therapeutic • Snake Venoms • Splenic Diseases

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Background

Snakebite envenoming results from injection of a mixture different toxins following snakebite, and includes coagulopathy and life-threatening hemorrhage, or venom-induced consumption coagulopathy (VICC). Worldwide, VICC occurs as a result of bites by vipers, most Australasian elapid snakes, and a few species of colubrids [1]. VICC results from the combined effects of toxins, which include phospholipase A_2 (PLA_2), snake venom serine protease (SVSP), and snake venom metalloprotease (SVMP) [2,3]. Bleeding may be mild and involve localized bleeding from the site of the snakebite, but can be systemic and life-threatening and include gastrointestinal and intracranial hemorrhage [2,3].

Currently, there have been few previously reported cases of spontaneous atraumatic liver or splenic hemorrhage due to snakebite [4–6]. In most previously reported cases, inaccessibility of the splenic artery prevented arterial embolization, and if arterial embolization was performed, the patients were hemodynamically unstable and required splenectomy [4–6]. When surgery was performed, mechanical ventilation and continuous hemodialysis were required due to complications from surgery [5,6].

A case is presented of a patient who was treated with antivenom for snakebite at another hospital, but who subsequently developed delayed VICC with splenic hemorrhage, who was successfully treated without complications by splenic artery embolization, blood transfusion, and additional antivenom therapy.

Case Report

A 62-year-old man with a history of hypertension, benign prostate hypertrophy, and right hip joint arthroplasty performed three years previously, was admitted to the emergency department of another hospital with dizziness followed by loss of consciousness during breakfast. He had been bitten by a snake on his left index finger three days previously and was treated with antivenom. On admission, his blood pressure was 70/50 mmHg. His complete blood cell count (CBC) parameters included a white blood cell (WBC) count of 10,700 per µl, hemoglobin (Hb) of 12.0 g/dL, hematocrit (Hct) of 35%, and a platelet count of 136,000 per µl.

He was transferred to the emergency department of our hospital due to hypotension. On admission, he was alert with a blood pressure of 92/58 mmHg, heart rate of 59 beats/min, a respiratory rate of 16/min, and a body temperature of 36.1°C. The patient had no complaints, other than pain in the left index finger. Physical examination showed swelling and a focal hemorrhagic bulla on the left index finger (Figure 1). There was

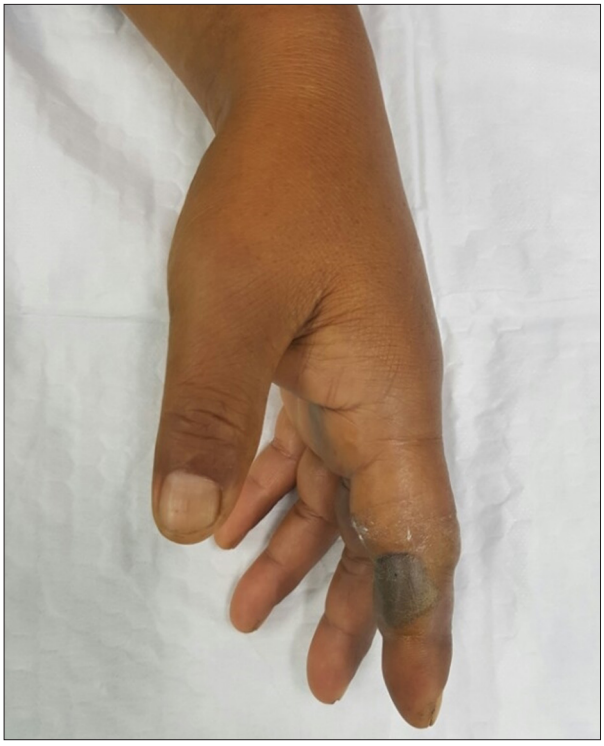

Figure 1. Swelling of the left index finger and a hemorrhagic skin bulla were seen around the snakebite wound.

no systemic subcutaneous hemorrhage, bleeding of the gums, or purpura. There was no abdominal tenderness, and the respiratory and heart sounds were normal. The initial electrocardiogram (ECG) showed normal sinus rhythm. There were no abnormalities on plain film chest X-ray.

On this admission, the CBC parameters included a WBC count of 8,760 per µl, Hb of 9.7 g/dL, Hct of 29.1%, and a platelet count of 125,000 per µl. Routine biochemistry tests were in the normal range, including serum electrolytes, aspartate aminotransferase (AST), alanine aminotransferase (ALT), creatinine, and creatinine kinase (CK). Blood coagulation tests showed a prolonged prothrombin time (PT) of 26.2 seconds (normal range, 11.6–15.5 seconds), an increased international normalized ratio (INR) of 2.37 (normal range, 0.87–1.24), a normal activated partial thromboplastin time (aPTT) of 39.2 seconds (normal range, 28.0–45.0 seconds) and an increased D-dimer level >20 µg/ml (normal range, 0–0.5 µg/ml). Reticulocytes were normal at 1.9%, the antithrombin III level was reduced to 56%, fibrinogen was reduced at <60 mg/dL, and the level of fibrin degradation products (FDPs) was increased 14.7 µg/ml.

Continuous monitoring of the patient's vital signs showed intermittent hypotension and reduced Hb and Hct measurements. Gastric lavage was performed using a nasogastric tube and a digital rectal examination excluded gastrointestinal bleeding. Continuous infusion of normal saline began, and two units

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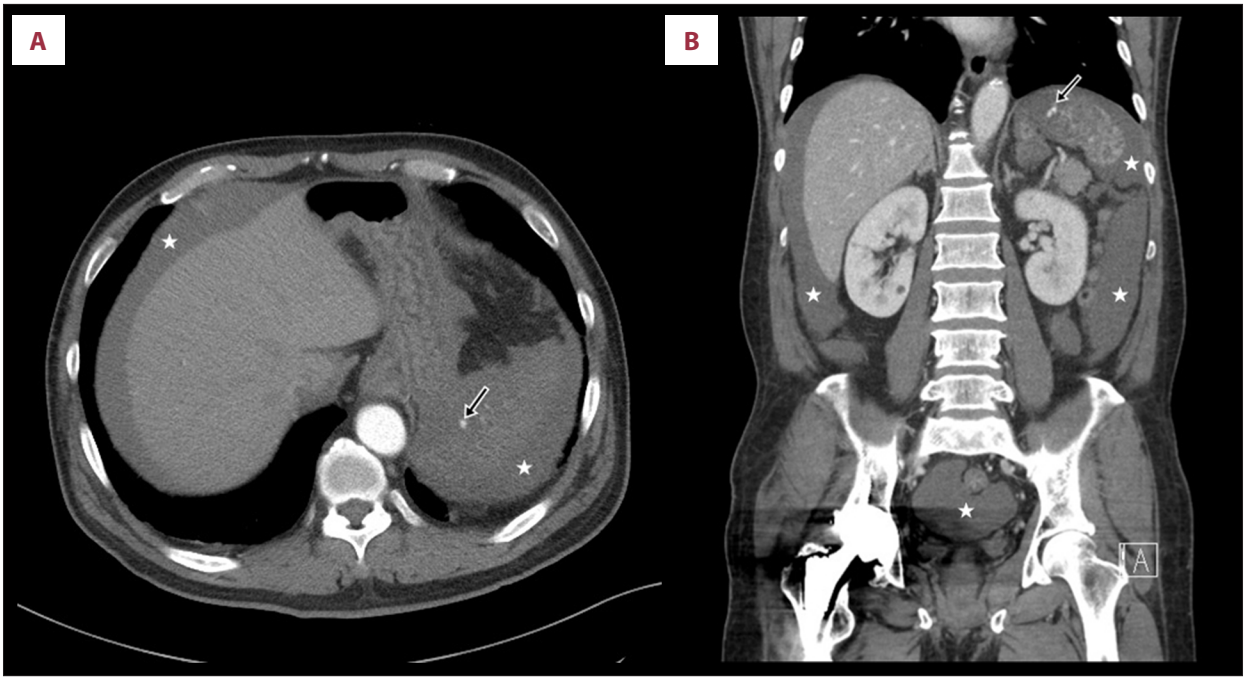

Figure 2. Contrast-enhanced computed tomography (CT) scan of the abdomen performed in the emergency department. Splenic arterial hemorrhage (arrow) and hemoperitoneum (white star) are shown in the axial (**A**) and coronal (**B**) images.

of packed red blood cells were transfused to correct his anemia (Hb 9.7 g/dL). Antivenom was administered (freeze-dried Agkistrodon antivenom 6000 U).

Three hours after admission to our emergency department, the patient complained of sudden abdominal pain, and mild epigastric tenderness was found on abdominal examination. Abdominal computed tomography (CT) was performed to determine the cause of his abdominal pain. Abdominal CT showed splenic arterial bleeding and hemoperitoneum (Figure 2). At this stage, his blood pressure was 99/61 mmHg, his pulse rate was 56 beats/minute, and his follow-up CBC parameters included a Hb 6.7 of g/dL, Hct of 19.2%, and a platelet count of 89,000 µl, which were reduced when compared with the findings on initial admission to the emergency department.

Three units packed red blood cells and three units of fresh frozen plasma were transfused. Urgent angiography with splenic arterial embolization was performed using Histoacryl/Lipiodol mixture (Figure 3). The patient was then admitted to the intensive care unit (ICU) for close observation. Packed red blood cells, fresh frozen plasma, and platelets were transfused until the next day when the patient's vital signs stabilized. Antivenom was administered until the third hospitalization day and coagulation function was normalized. On the fourth hospital day, a follow-up abdominal CT scan was performed, which confirmed that there were no signs of active bleeding from the splenic artery, and the hemoperitoneum was slightly decreased (Figure 4). After the CT scan, the patient was transferred to the general hospital ward and was discharged on the eighth hospital day without any complications.

Discussion

Snake venom is a complex natural toxin comprising proteins and metallic ions, carbohydrates, hexanes, amines, and lipids [7]. Snake venom contains enzymatic protein toxins that include phospholipase A_2 (PLA_2), snake venom serine protease (SVSP), and snake venom metalloprotease (SVMP), L-aminoacid oxidase, and phosphoesterase [2]. The venom also contains C-type lectin receptor, and disintegrin coagulation factors that act on the vascular endothelium, resulting in vessel wall injury, impaired platelet aggregation, and increased capillary permeability and bleeding [2].

Spontaneous and atraumatic splenic hemorrhage is a potentially life-threatening condition that can be associated with infection, bleeding disorders, and malignancy [8]. In the present case, there was no recent trauma, and treatment was successfully performed with non-surgical management. Unlike previously reported cases [5,6], splenectomy and histology of the spleen were not performed. Therefore, it was not possible to exclude other causes of splenic hemorrhage, such as the presence of tumors or infection. Also, even though we could not exclude vascular injury, the history of snakebite was considered to be the cause of splenic hemorrhage or venom-induced consumption coagulopathy (VICC) due to snakebite envenoming.

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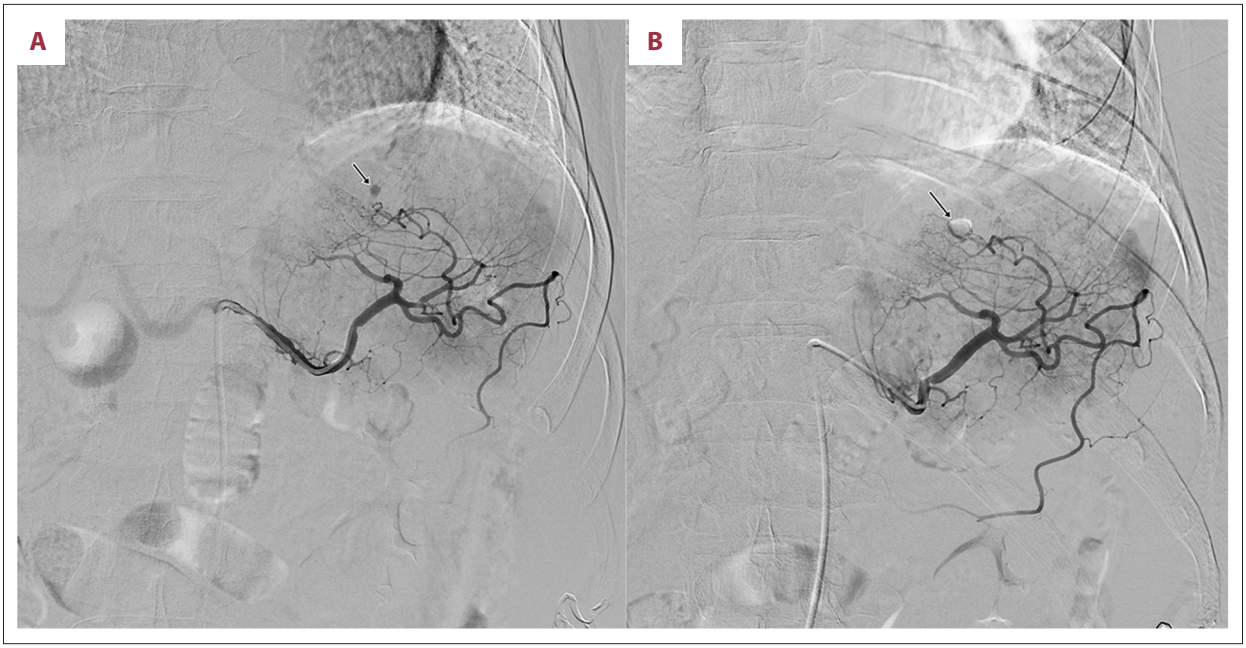

Figure 3. Splenic arterial angiography images. (**A**) Splenic arterial angiography shows active hemorrhage of a branch of the splenic artery (arrow). (**B**) Splenic arterial angiography performed after embolization using Histoacryl/Lipiodol mixture and selection of two branches of the eighth segment of the hepatic artery. No localized contrast leak is noted.

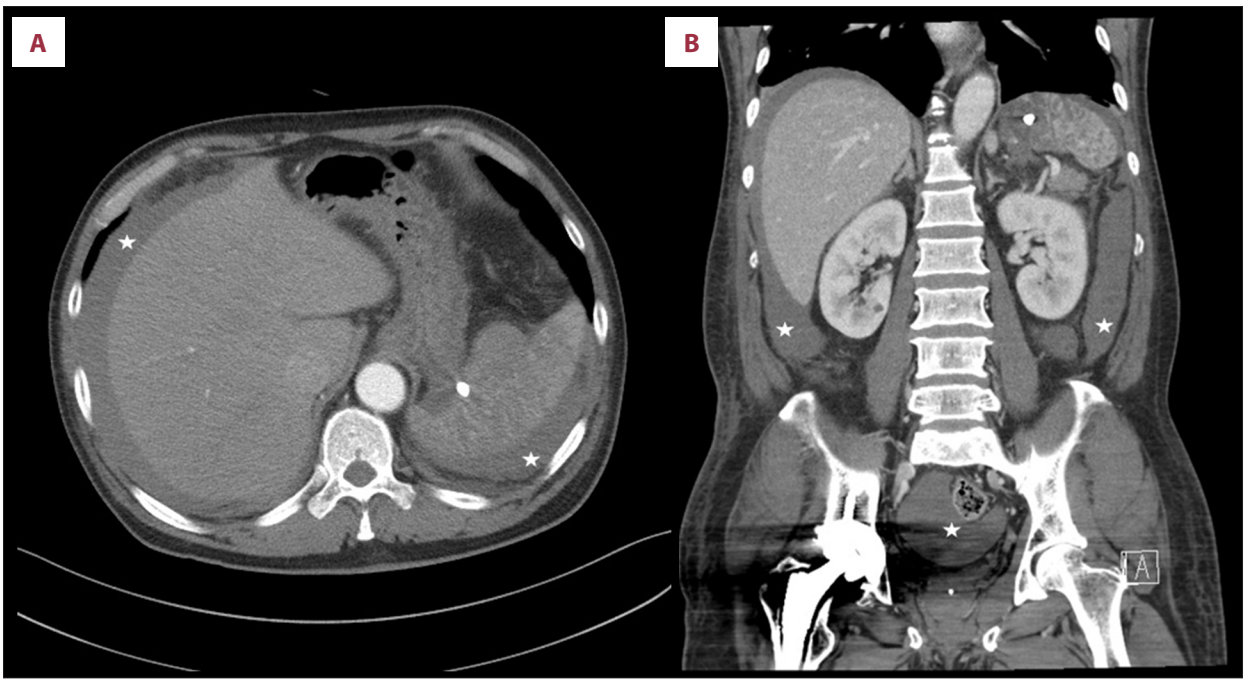

Figure 4. Contrast-enhanced computed tomography (CT) of the abdomen three days after angio-embolization. Contrast-enhanced axial (**A**) and coronal (**B**) abdominal computed tomography (CT) scans show no evidence of splenic artery hemorrhage and slightly reduced hemoperitoneum (white star).

The presence of coagulopathy was confirmed from the findings of the blood tests, including a prolonged prothrombin time (PT) and increased international normalized ratio (INR), reduced fibrinogen, and increased levels of D-dimer and fibrin degradation products (FDPs). In this case, antivenom had been administered at a previous hospital, but VICC and splenic hemorrhage could not be prevented. This may have been because the dose of antivenom was insufficient or the antivenom could

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have combined with the venom, reducing its potency and resulting in delayed symptoms of toxicity [9].

When VICC results in organ hemorrhage, treatment for VICC and organ hemorrhage should be performed simultaneously. In the treatment of VICC, antivenom should be given to prevent consumptive coagulopathy, although the timing of treatment remains controversial [10]. Also, once the toxin is combined with the antivenom, it is helpful to transfuse fresh frozen plasma to replace the deficient coagulation factors [10]. However, there are no guidelines regarding the management of atraumatic splenic rupture and how it should differ from the management of traumatic splenic injury [11]. Renzulli et al. [8] recommended that patients with atraumatic splenic rupture with a non-malignant etiology may be treated with organ-preserving surgery, and the non-surgical approach of transcatheter arterial embolization.

The trend for the management of patients with solid organ injury continues to favor a non-surgical approach using a conservative management technique. Splenic embolization is increasingly used for both stable patients and for patients who do not respond to fluid and blood replacement. For patients with uncontrolled bleeding from rupture of the spleen and severe hemodynamic instability, splenectomy is the treatment of choice [8,11,12]. However, non-operative treatment can be a safe option if appropriate diagnostic evaluation has been undertaken, and close monitoring and surgical supervision are used in selected hemodynamically stable patients [8,11,12]. Although non-operative treatment was reported to be unsuccessful in previously reported cases, based on our experience, splenic hemorrhage due to snakebite envenoming can be treated with angio-embolization in addition to antivenom therapy and blood transfusion. After antivenom therapy and procedures for hemostasis, close observation is required, as additional complications may develop. Follow-up tests and imaging studies to assess coagulopathy or re-bleeding should be performed to evaluate treatment response or delayed VICC.

The clinical presentation of atraumatic, spontaneous splenic hemorrhage is similar to traumatic splenic injury and includes classical signs and symptoms such as left upper quadrant pain, abdominal guarding on examination, and hemodynamic instability [13,14]. However, in this case, the patient presented with hypotension, the patient did not initially complain of abdominal pain, and symptoms of abdominal pain developed during later examinations. In this case, the combination of blood tests that showed decreased levels of coagulation factors, and imaging using abdominal computed tomography (CT) confirmed bleeding from the spleen. The patient was treated with emergency transfusion, and additional antivenom treatment was initiated. Although gastrointestinal lavage and digital rectal examination were initially used to identify the source of bleeding, focused assessment sonography for trauma (FAST) and CT scan were not initially performed.

For the patient who presents with hypotension and coagulopathy following a snakebite, the diagnosis of spontaneous atraumatic intra-abdominal hemorrhage can be challenging as there is no associated trauma and the symptoms are nonspecific and subtle. However, delayed diagnosis may be life-threatening. In a patient with a snakebite who presents with nonspecific symptoms that include abdominal pain and dizziness with hypotension, the physician should be alert to the possibility of occult intra-abdominal hemorrhage secondary to snakebite envenoming and VICC. As this case has shown, the definitive diagnosis of the site of occult hemorrhage can be determined using CT, and serial hemoglobin levels should be measured.

Conclusions

This case has shown that in atraumatic splenic hemorrhage followed by snakebite envenoming, non-operative treatment using angio-embolization of the splenic artery may be performed. This procedure should be followed by close monitoring of patients who respond to initial resuscitation or who are hemodynamically stable because the patient might still be at risk of delayed coagulopathy and occult hemorrhage.

Conflict of interest

None.

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