An Unusual Presentation of Vasculotoxic Snake Bite

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ABSTRACT
In India, viper bites are more common than any other poisonous snake bites. In these patients, the cardinal features are local pain, swelling and mild coagulation abnormalities; but gangrenous changes involving bilateral upper and lower limbs are uncommon. We are reporting a case of a 62-year-old man bitten by a Russell viper on the right thumb. He experienced local pain, swelling and later developed disseminated intravascular coagulopathy, pre-renal azotemia, sepsis, acute respiratory distress syndrome (ARDS) and peripheral gangrenous changes involving bilateral upper and lower limbs. He succumbed to his ailment 5 months after the bite. There have been very few cases of gangrenous changes involving distal part of extremities associated with snake bite.

Keywords: Adult respiratory distress syndrome, Gangrene, Whole blood clotting time, Anti-snake venom, Disseminated intravascular coagulopathy.


Source of support: Nil
Conflict of interest: None

CASE REPORT

A 62-year-old Indian male, gardener by occupation was brought to our casualty with history of snake bite on the palmar aspect at the base of the right thumb. The dead snake was identified as Daboia russelii (Russell Viper) (Fig. 1). There was no history of ptosis, diplopia, dysphagia, dysphonia or convulsions. There was no history suggestive of any connective tissue disorder.

On examination, his pulse was 96 beats per minute, blood pressure was 110/70 mm Hg. His peripheral pulsations were normal. Patient was conscious, oriented. Small bruising was noted at the site of bite which later progressed to pain and swelling of the right thumb and then the hand. Twenty minutes whole blood clotting time was normal at the time of admission.

However, after 3 hours, his 20 minutes whole blood clotting time prolonged for more than 1 hour. He was given antitetanus toxoid, analgesics and wound dressing was done. He received 100 IU of ASV and developed severe hypotension and bradycardia (blood pressure 70/40 mm Hg, pulse 50 beats per minute) and consequently was started on inotropic support, anti-histaminics and steroids.

The next day, he started complaining of abdominal pain, loose stools and reduced urine output. On per abdomen examination, there was diffuse tenderness. He received intravenous fluid. After 6 hours, his 20 minutes whole blood clotting time was still greater than 1 hour. He was again administered another 100 units of ASV.

On the third day, he developed breathlessness with tachypnea and tachycardia (pulse rate increased to 130 beats per minute). Patient was electively intubated and put on mechanical ventilator.

On the fourth day, he developed gangrenous changes in bilateral upper and lower limbs; initially in the right thumb, 2nd and 3rd finger and then over the left thumb and digits of the left hand extending up to the tip of metacarpals, both mid foot and digits of both lower limbs (Figs 2 and 3).

After 72 hours, patient recovered and was weaned off from mechanical support. His blood pressure and urine output improved. During this course, he received about 4 units of fresh frozen plasma.

His right hand showed improvement. He was advised amputation of his both legs and left hand followed by a reconstruction surgery with prosthesis. However, patient denied and was taken home. After 5 months, he succumbed to snake bite and its complications.

Laboratory investigations revealed: Hb—11.4 gm/dl, total leukocyte count: 10,400, platelets—1.06 lakhs/mm$^3$, 20 minutes white blood cells test (WBCT)—on admission 4 minutes and 10 seconds which increased to more than 1 hour after 3 hours, random blood sugar levels—82.1 mg/dl, serum creatinine: 1.2 mg/dl which progressed to 2.3 mg/dl and again came to 1.6 mg/dl suggestive of acute kidney injury, sepsicaemia, disseminated intravascular coagulopathy diagnosed on the basis of prolonged prothrombin time which was INR 1 and progressed to INR 1.8 and low platelet counts as shown in the Table 1. Chest X-ray showed fluffy shadows on days 3 (Figs 4 and 5). Electrocardiography was normal. Arterial blood
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Fig. 1: Russell’s viper (*Daboia russelli*)

Fig. 2: Developed acrocyanosis with gangrenous changes in bilateral upper limbs

Fig. 3: Developed acrocyanosis with gangrenous changes in bilateral lower limbs

Fig. 4: Chest X-ray showed bilaterally fluffy shadows

Fig. 5: Chest X-ray after 3 days of intubation showing resolving fluffy shadows

gas analysis revealed hypoxia with Murray’s score of 2.5. Color Doppler study for the arteries of both upper and lower limbs were normal.

Patient received 200 units of polyvalent anti-snake venom, higher antibiotics, systemic anticoagulation in the form of heparin, fresh frozen plasma and his renal functions improved on his own.

DISCUSSION

Snake bite remains a problem in the developing world. It has been estimated by the World Health Organization that there are 15 to 20 thousand deaths per year in India.\(^1\)

Peripheral gangrene is distal ischemic damage in more than two sites in the absence of major vascular disease. There is a high mortality rate with high frequency of multiple limb amputations in the survivors. Disseminated intravascular coagulopathy has been the cause of common pathway of its pathogenesis. It occurs due to spontaneous activation of factor V and factor X by procoagulants present in the venom. It has also been suggested that small dose of venom, as typically injected in humans, leads to continuous activation of fibrinogen, producing a fragile fibrin more susceptible to lysis than ordinary fibrin, leading to bleeding manifestations.
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<td>Prothrombin time (mins)</td>
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<td>Chest X-ray</td>
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**Table 1**: Serial laboratory investigations of the case.

BUN: Blood urea nitrogen; WNL: Within normal limit.

Toxic vasculitis due to action of the toxins on vascular endothelium is also a possible mechanism in turn leading to DIC and thrombocytopenia.\(^2\)

Most of the viper venoms act on the hematological system, particularly on capillary endothelium. Viper bite being primarily vasculotoxic, causes spontaneous hemorrhage, damages vascular endothelium and inhibits platelet aggregation. Procoagulant venom factors act at various points of clotting cascade, while fibrinolytic factors act directly by activating plasminogen. Systemic bleeding due to consumption coagulopathy and thrombocytopenia due to activation of procoagulants is the leading manifestation and cause of death in Russell’s viper systemic envenoming.

Local necrosis with viper bites often appears to be mainly ischemic, developing slowly over weeks and presenting like dry gangrene. Disseminated gangrene at sites apart from the local bitesite has been rarely reported with snakebites.\(^3,4\) Toxic vasculitis due to action of the toxins on vascular endothelium is also a possible mechanism in turn leading to DIC and thrombocytopenia.\(^5\) Another possibility includes inotropic drugs that are common in the intensive care unit (ICU).

Severe local complications, such as skin necrosis and digital gangrene appear to be more common with green pit viper bites. Skin necrosis is more common if blisters are present and if the bites are on fingers and toes.

Anti-snake venom remains the major treatment option for all patients with signs of systemic envenomation including coagulopathy, renal failure and extensive local swelling of the bite site. Early treatment in the 3 to 6 hours of systemic signs may be effective in reducing renal damage\(^6\) however, there is little evidence on its effectiveness to reverse gangrene.\(^7\)

**CONCLUSION**

Peripheral gangrene in more than two sites in the absence of a major vascular disease occurs in rare cases. Disseminated intravascular coagulation has been stated as the pathogenesis behind it. There is high mortality rate with high frequency of multiple limb amputations in the survivors.

This is a rare presentation of Russell viper, vasculotoxic snake bite with gangrenous changes in all four limbs due to disseminated intravascular coagulopathy with probable effect of vasopressors, acute respiratory distress syndrome, prerenal azotemia and septicemia. However, patient denied surgery for reconstruction of limbs, survived for 5 months with this morbidity.

**REFERENCES**