Short Report

# Coagulopathy and fibrinolysis following the bite of a hump-nosed viper (*Hypnale hypnale*)

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The hump-nosed viper (*Hypnale hypnale*) is a moderately venomous snake. Most bites cause predominantly local effects such as pain, swelling and haemorrhagic blisters at the site of the bite (SELLAHEWA & KUMARARATNE, 1994). However, envenomation following its bite can cause systemic effects such as renal failure, and rarely can even be fatal (VARAGUNAM & PANABOKKE, 1970; SAWAI *et al.*, 1983). Coagulation defects following hump-nosed viper bites are extremely rare. There is only one case report of haemostatic dysfunction following the bite of a hump-nosed viper when there was no doubt about the identity of the snake (DE SILVA *et al.*, 1994).

We report the second case of abnormal bleeding tendency following a verified hump-nosed viper bite, where there was also *in vivo* evidence of excessive fibrinolytic activity.

## Case report

A 55 years old man from Ragama, Sri Lanka, was bitten on the hand by a hump-nosed viper (*H. hypnale*). The snake was killed and brought with the patient to hospital, where it was positively identified. The patient initially had pain and swelling at the site of the bite. The swelling spread proximally and, on admission to hospital half an hour after the bite, it had spread above the elbow. There was also oozing of blood from the site of the bite, but no bleeding from any other site was noted. There was no past or family history of an abnormal bleeding tendency, and the patient was not receiving any long-term medication. On examination, he was not pale or icteric. The cardiovascular, respiratory and nervous systems were clinically normal. The urine output was normal, and urine was not blood stained.

The clotting time was grossly abnormal. The blood did not clot even after 4 h (normally 2–10 min). The following investigations were done: bleeding time (Ivy method), 3 min (normally 2–7); serum fibrinogen, 0·28 g/L (normally 1·8–3·5); fibrinogen degradation products 6000 ng/mL (normally <500); platelet count 175 $\times$ $10^9$/L, haemoglobin 10·5 g/dL, white blood cell count 8·9 $\times$ $10^9$/L (neutrophils 77%, lymphocytes 21%, eosinophils 2%), blood urea 4·7 mmol/L, serum sodium 132 mmol/L, serum potassium 4·6 mmol/L, aspartate aminotransferase 4 iu/L, alanine aminotransferase 7 iu/L, urine (protein absent, deposit showed no red or white blood cells); erythrocyte sedimentation rate 4 mm in the first hour.

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At this stage the patient was given tetanus toxoid, and 10 ampoules of Serum Institute of India (SII) polyspecific antivenom intravenously. Neither this preparation nor the only other antivenom available in the country, Haffkine antivenom, has any known effect against hump-nosed viper venom. The clotting time about 2 h after antivenom infusion was still >30 min. When the clot finally appeared it dissolved rapidly. The patient was given another 10 ampoules of SII antivenom. By the third day in hospital he had developed cellulitis and a haemorrhagic blister at the site of the bite. This was treated surgically, and he was given crystalline penicillin, 2 megaunits, and cloxacillin, 500 mg, intravenously every 6 h. By the fourth day following the bite the clotting time had become normal (8 min), as had the prothrombin time (11 s; control 11·8; international normalized ratio = 0·93), activated partial thromboplastin time (26·1 s), and thrombin time (14 s). Oozing of blood from the site of the snake bite had stopped. During this period there was no evidence of bleeding from any other site. The patient recovered without further complication, and was discharged from the hospital on the ninth day following the snake bite.

## Discussion

One case of bleeding diathesis following hump-nosed viper bite has previously been reported in a 5 years old boy from Kandy, Sri Lanka (DE SILVA *et al.*, 1994). That patient had a severe coagulopathy with overt gastrointestinal bleeding and acute renal failure which needed dialysis; the authors also reported procoagulant, fibrinolytic and platelet aggregating activity of the venom *in vitro*.

Our patient developed coagulopathy, and evidence *in vivo* of excessive fibrinolysis. Unlike the previously reported case, he had no overt bleeding apart from oozing of blood from the site of the bite. Not suprisingly, the antivenom preparation we used did not seem to have any effect.

This case is further evidence that systemic envenomation does occur following hump-nosed viper bites, and such envenoming can be potentially life-threatening. We recommend, therefore, that haemostasis be investigated in all patients following bites of this species of snake and, as 27% of all snake bites in Sri Lanka are due to hump-nosed vipers (DE SILVA & RANASINGHE, 1983), a specific antivenom be developed.

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