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CASE REPORT

A FATAL CASE OF SADDLE PULMONARY EMBOLISM: IS IT DUE TO HUMP-NOSED VIPER ENVENOMATION?

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ABSTRACT

Hump-nosed viper (HNV) bites are a common occurrence in Sri Lanka, and systemic envenomation followed by acute renal failure (ARF) and Micro Angiopathic Haemorrhagic Anaemia (MAHA) are well-documented complications. But pulmonary thromboembolism has never been reported. A 56-year-old previously healthy gentleman presented following an HNV bite with the complaint of pain at the site. On examination, fang marks were observed over the left 4th toe with local reaction. His initial clotting profile was unremarkable, but he got deranged on the third day, complicated with low urine output. While he was on peritoneal dialysis on the 9th day, he suddenly developed shortness of breath and expired. The autopsy revealed local necrosis over the bite site, indwelling saddle pulmonary thrombus and bilateral deep vein thrombosis. There was macroscopic and microscopic evidence of ARF. The histology of the lungs was suggestive of pulmonary hypertension. Effects of HNV bites range from local features to systemic envenomation like venom induced coagulopathy, thrombotic microangiopathy and ARF. Venom contains mild procoagulant and phospholipase activity, giving rise to MAHA. But deep vein thrombosis followed by pulmonary embolism (PE) has never been reported for HNV bites. The etiology of PE could be due to the shared effects of undiagnosed pulmonary hypertension, prolonged bed rest and venom induced coagulopathy. Systemic envenomation is not uncommon with HNV bites complicated with ARF and MAHA. Since PE has not been documented earlier as a complication, it is suggested that the possible association with future research, though there were a few other risk factors involved.

Keywords: *Hump nosed viper bites; saddle pulmonary embolism; systemic envenomation*

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INTRODUCTION

Hump-nosed viper (HNV) or *Hypnale hypnale* bites are common in Sri Lanka, especially in rural areas throughout Sri Lanka. It has been found that the venom of the hump-nosed viper contains procoagulant enzymes and phospholipases, which can give rise to a variety of systemic effects such as venom-induced coagulopathy, acute renal failure (ARF), and microangiopathic haemorrhagic anaemia (MAHA)[1]. These three complications are commonly observed following envenomation and are well-documented in clinical literature. However, pulmonary thromboembolism (PE), especially saddle pulmonary embolism, has never been reported with HNV bites.

Pulmonary embolism occurs when the pulmonary arteries are occluded by an indwelling blood clot, leading to respiratory and cardiovascular collapse. It is often related to risk factors such as prolonged immobility, venous stasis, hypercoagulable states, trauma, etc.[2]. Here we present a potential novel complication, saddle pulmonary embolism, in a case of HNV envenomation that has not been reported previously.

CASE HISTORY

A 56-year-old, previously healthy man with no significant medical history presented with a hump-nosed viper bite on the left foot. He complained of pain at the site of the bite, and on examination, fang marks and local erythema were observed. The initial clotting profile revealed no significant abnormalities, and there were no signs of systemic envenomation. However, by day three, the patient's clotting profile got deranged, with prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT), suggestive of venom-induced coagulopathy[3].

In addition to coagulopathy, the patient developed oliguria, leading to the diagnosis of acute renal failure (ARF). Peritoneal dialysis was initiated on day seven of admission. By the ninth day, the patient suddenly developed shortness of breath and respiratory distress. Despite intensive efforts to resuscitate, the patient unfortunately expired. An autopsy was performed, and it revealed several concerning findings. Local necrosis at the bite site (Fig. 1) was noted, and an indwelling saddle pulmonary embolism (Fig. 2) was observed, obstructing the pulmonary arteries. Bilateral deep vein thrombosis (DVT) was present in the lower extremities (Fig. 3). Histological examination of the lungs revealed evidence of pulmonary hypertension (Fig. 4), while the kidneys showed changes consistent with ARF.

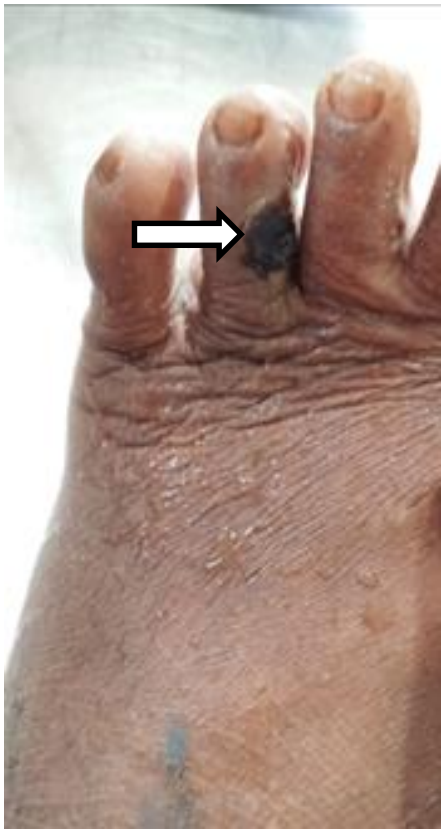

Figure 1: Local necrosis over the bite site on the left foot (white arrow)

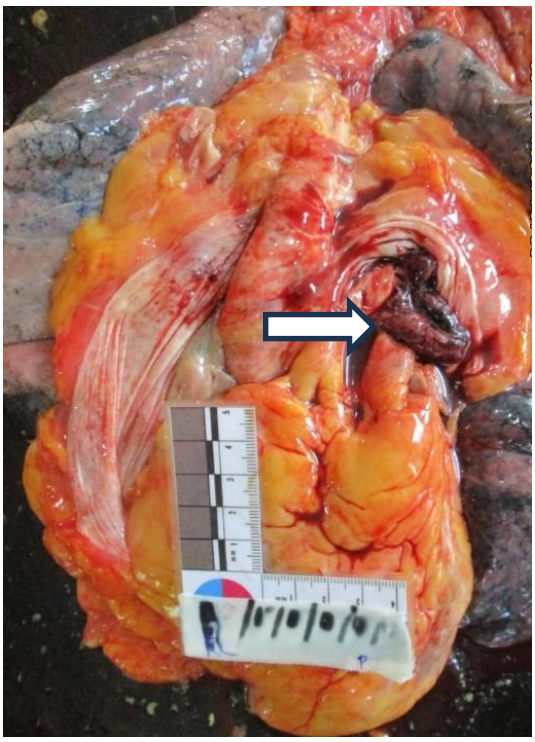

Figure 2: Indwelling saddle pulmonary thrombus obstructing pulmonary arteries (white arrow)

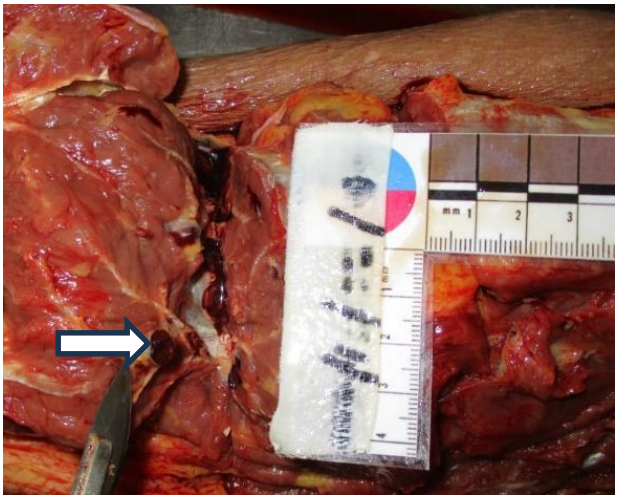

Figure 3: Deep vein thrombosis over one of the lower limbs (white arrow)

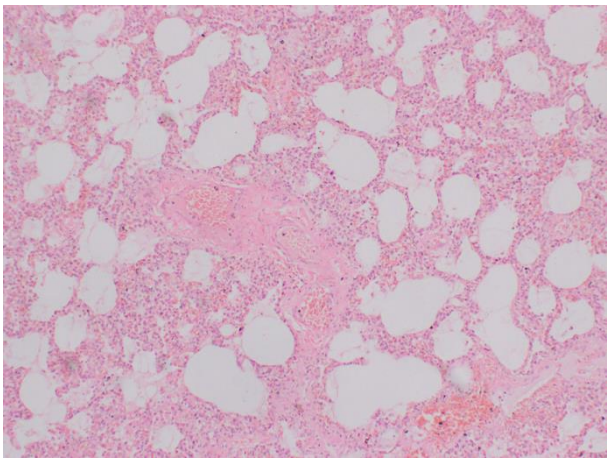

Figure 4: Photomicrograph of lung tissue showing vascular remodelling (H&E ×10)

DISCUSSION

ARF, MAHA, and venom-induced coagulopathy are well documented complications of the systemic effects of HNV bites. These complications are related to the venom's procoagulant properties, which cause widespread microvascular injury, thrombosis, and hemolysis[4]. However, the occurrence of pulmonary thromboembolism, especially saddle pulmonary embolism, has not been reported in the literature.

The development of PE in this patient can be attributed to a combination of factors. First, venom-induced coagulopathy may have contributed to the formation of microthrombi, particularly in the deep veins. The prolonged bed rest necessitated by peritoneal dialysis for ARF likely promoted venous stasis, further increasing the risk of thrombosis[5]. Additionally, histological evidence of pulmonary hypertension may have predisposed to the development of PE due to turbulent flow within and endothelial damage[6]. The presence of bilateral deep vein thrombosis (DVT) and saddle pulmonary embolism suggests that the clot formation in the lower extremities eventually migrated to the pulmonary arteries, blocking blood flow and resulting in fatal respiratory and cardiovascular collapse.

Moreover, the possibility of undiagnosed underlying clotting disorders cannot be excluded in the development of thromboembolic complications. The patient may have had a pre-existing clotting disorder, such as a mild deficiency in natural anticoagulants (e.g., Protein C or S deficiency), which could have predisposed him to thrombosis when combined with the procoagulant effects of the snake venom[7].

Another contributing factor could be the impact of renal failure on coagulation. ARF often results in altered haemostasis due to changes in platelet function, clotting factor levels, and the accumulation of waste products that interfere with coagulation. The patient's renal impairment could have exacerbated the effects of venom-induced coagulopathy, leading to a greater predisposition for thrombus formation. In addition, dialysis itself may have influenced coagulation by altering levels of various coagulation factors like tissue factor and fibrinogen[8].

CONCLUSION

This case represents the first documented case of saddle pulmonary embolism in a patient with hump-nosed viper envenomation. While systemic envenomation with complications such as acute renal failure and microangiopathic haemorrhagic anaemia is well recognised, the development of pulmonary embolism in this patient suggests the need for further research into this potential complication. The causes of pulmonary

embolism may include a combination of a multitude of factors such as venom-induced coagulopathy, prolonged immobility due to ARF and dialysis, undiagnosed clotting disorders, renal failure impacting coagulation, etc.

The authors recommend that clinicians remain vigilant for thromboembolic complications, including pulmonary embolism, in patients with systemic envenomation, particularly in those with prolonged bed rest or other risk factors. Further studies are recommended to better understand the pathophysiology of thromboembolic events in snakebite envenomation, which may be helpful in the development of preventive strategies and improved management for such patients.

ACKNOWLEDGMENTS

None.

CONFLICTS OF INTEREST

The authors declared no conflicts of interest.

ETHICAL ISSUES

The presented case was conducted for medico-legal purposes, and the findings were used for academic purposes, according to the institutional guidelines without divulging the identity of the individual.

SOURCES OF SUPPORT

None.

AUTHOR CONTRIBUTIONS

PRCW: Conception and design of the work; the acquisition, analysis, and interpretation of data for the work; drafting the work and revising it critically for important intellectual content; and final approval of the version to be published. **PRR:** Conception and design of the work; the acquisition, analysis, and interpretation of data for the work; drafting the work and revising it critically for important intellectual content; and final approval of the version to be published.

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