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Case Report Open Access

Pulmonary Haemorrhage due to Hump-Nosed Viper Bite; Excellent Response to Methyl Prednisolone-Case Report and Review of Literature

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Abstract

Background: Hump-nosed viper bite, the commonest venomous snake bite in Sri Lanka, is associated with significant morbidity. Specific anti-venom is not available for hump-nosed viper envenomation which is usually managed with supportive treatment. Pulmonary hemorrhage is an unusual manifestation of hump-nosed viper bite. Here we present a case of hump-nosed viper envenomation which complicated by pulmonary hemorrhage and was successfully treated with systemic steroids. To the best of our knowledge, it has not been reported in the literature before.

Case Presentation: A previously healthy 55-year-old man presented to the local hospital 18 hours after a hump-nosed viper bite. He developed bilateral severe pulmonary hemorrhages, evidenced by rapid desaturation which needed intubation and mechanical ventilation, bleeding from the endotracheal tube and bilateral alveolar shadows in a chest X-ray. He had no other bleeding manifestations. Because of the life-threatening situation, he was treated with methylprednisolone pulse therapy. There was a rapid improvement of hypoxia with a resolution of X-ray changes. He was successfully weaned off from the ventilation after 24 hours.

Conclusion: This case highlights the importance of suspecting pulmonary hemorrhage in a patient who develops desaturation and alveolar shadow following hump-nosed viper bite even in the absence of other bleeding manifestation. Early and timely treatment with systemic steroid can be lifesaving in such patients.

Keywords: Hump-nosed viper; Pulmonary hemorrhage; Methyl-prednisolone

Abbreviations: HNV: Hump-nosed Viper; CSTH: Colombo South Teaching Hospital; MAHA: Micro Angiopathic Haemolytic Anaemia

Introduction

Hump-nosed Viper (HNV) bite is one of the medically important envenomation in the Indian subcontinent which causes significant morbidity and mortality [1]. It can cause a variety of clinical manifestations such as local inflammation, blistering and systemic envenomation such as hematological manifestations and acute kidney injury [2].

Although coagulopathy is reported frequently pulmonary hemorrhage is an unusual manifestation of hump-nosed viper bite [3,4]. Here we report a patient who developed pulmonary hemorrhage which was successfully treated with systemic steroids. This highlights the importance of suspecting pulmonary hemorrhage in a patient who de-saturate with alveolar shadows in chest X-ray despite the absence of overt coagulopathy, and the rapid response to methylprednisolone.

Case Presentation

A 55-year-old previously healthy man from the western province of Sri Lanka was admitted to Colombo South Teaching Hospital (CSTH) 18 hours after an HNV bite. He was initially managed in a peripheral hospital and later transferred as he was anuric for 8 hours. He also had vomiting and loose stools. Fang marks were seen on the fifth left toe with pain, minimal swelling and two blisters on the dorsum of the foot. He was conscious and rational with a pulse rate of 100 bpm, blood pressure 150/100 mmHg and oxygen saturation of 98%. There was no bleeding tendency or neurological manifestations. Bedside whole blood clotting time was less than 20 minutes on admission to the peripheral hospital and at 18 hours when seen at the CSTH. Polyvalent antivenom was not given as it is ineffective in neutralizing HNV toxicity and carry a high risk of side effects.

Initial investigations revealed, hemoglobin 13.2 g/dl, white cells 13.2 × 109/L, platelets 68 × 10^9/L, serum sodium 143 mmol/L, serum potassium 4.2 mmol/L and serum creatinine 3.2 mg/dl. On the 2nd day, hematological investigations revealed, hemoglobin 10.5 g/dl, white blood cells 14.1 × 10^9/L and platelets 58 × 10^9/L, whole blood clotting time >20 minutes, PT/INR 1.7 (reference range: <1.1) and APTT 48 seconds (30-40s). Total bilirubin-62.14 µmol/L (5-21) with direct bilirubin 10.08 µmol/L (<3.4), serum alanine aminotransferase (ALT) 171 U/L (10-40), serum Aspartate Aminotransferase (AST) 808 U/L (10-35), Creatine kinase (CK) 750 U/L (15-105), serum Lactate Dehydrogenase (LDH) 2370 (230-460) and serum creatinine was 409 µmol/L (70-120). Blood picture revealed fragmented red cells and thrombocytopenia suggestive of Microangiopathic Haemolytic Anaemia (MAHA). In view of Thrombotic Microangiopathy (TMA), he was transfused with fresh frozen plasma with the improvement of

INR and APTT. The patient was commenced on hemodialysis due to Acute Kidney Injury (AKI).

On the 3rd day, he became tachypneic with de-saturation and blood gases revealed PH 7.21, PCO_2 45 mmHg, PO_2 31 mmHg HCO_3 12.4. He was intubated and started on mechanical ventilation. Bleeding through the endotracheal tube was noted but there was no bleeding from elsewhere. Chest X-ray revealed bilateral alveolar shadowing suggestive of pulmonary hemorrhages (Figure 1). At this time his platelet count was 56×10^9/L, INR 1.1, APTT 40 seconds, thrombo-elastometry showed only a deficiency of platelets. Due to the life-threatening nature of the situation, he was commenced on intravenous methylprednisolone 1 g pulse therapy daily along with FFP and platelet transfusions. There was a rapid improvement of hypoxia with the resolution of chest X-ray changes during the next 48 hours. We discontinued steroid therapy after three days as there was no further bleeding and chest X-ray changes were resolving (Figure 2).

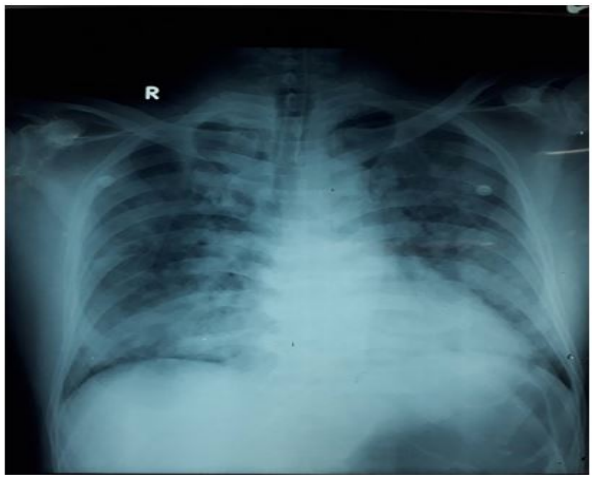

Figure 1: Bilateral alveolar shadowing suggestive of pulmonary hemorrhages.

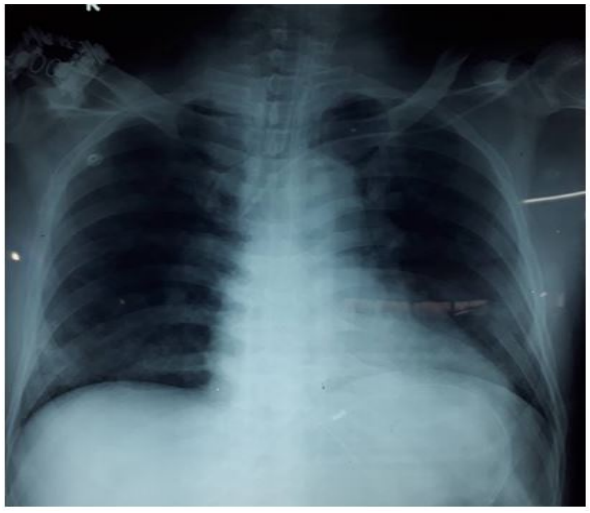

Figure 2: Resolution of pulmonary hemorrhage after steroid therapy.

Because of, persistent TMA as evidenced by a further drop in hemoglobin (8 mg/dl) and platelets (28×10^9/L) plasmapheresis was commenced and continued for 6 cycles. Despite the effective treatment of TMA, the patient went on to develop dry gangrene of toes on both feet (Figure 3). Renal function did not improve and required long term maintenance hemodialysis. He underwent renal transplantation eleven months after the incident due to end-stage renal disease.

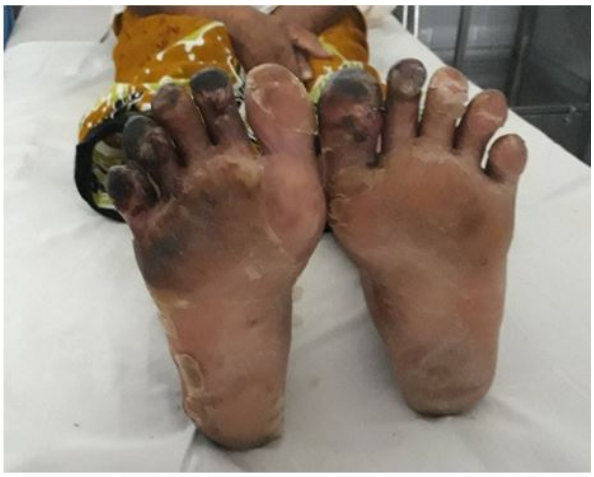

Figure 3: Dry gangrene of toes on both feet.

Discussion

The commonest cause of snake bite envenoming in Sri Lanka is due to the hump-nosed viper [1,2]. Three species of HNV of the genus Hypnale are found in Sri Lanka; *Hypnale hypnale, H. nepa* and *H. Zara* [4]. Previously regarded as a mildly poisonous snake, hump-nosed viper is now known to cause significant systemic toxicity and fatalities. Maduwage et al. demonstrated potent cytotoxic, weak procoagulant, neurotoxic, myotoxic and phospholipase A2 activities in all three Hypnale venoms *in vitro* [5]. Clinical manifestations of envenomation are the pain, local swelling, occasionally hemorrhagic blisters at the bitten site, coagulopathy including TMA, and AKI. Coagulopathy is reported in 3.8% of cases which occurs within the first 24 hours [6].

Identification of the snake by the victim or by medical personnel is the commonest means of diagnosing the respective snake. When the snake is unidentified visually, a syndromic diagnosis can be made in the clinical setting by applying clinical features into a validated algorithm [7]. Mild local swelling, blister formation, and renal failure were the key clinical features in this patient favoring HNV bite. Twenty-minute blood clotting time can be normal in the first 24 hours. The absence of neurological features helped to differentiate from toxicity due to Russell viper bite. Later development of coagulopathy and TMA further confirmed the HMN toxicity.

The fact that this patient's pulmonary hemorrhage occurred with mild coagulopathy suggests that it is due to a direct effect of venom rather than due to coagulopathy. Studies in mice have shown that hump-nosed venom can induce macroscopic pulmonary congestion, edema, gross hemorrhages and petechial hemorrhages [2]. *H. hypnale* causes petechial hemorrhages which were observed microscopically with a minimum amount of venom [8]. It is known that

metalloproteinases found in the snake venom can induce the release of inflammatory mediators such as cytokines which intensify the inflammatory response. Metalloproteinases comprise a series of zinc-dependent enzymes, of varying molecular mass, which plays a key role in the hemorrhagic effects by acting directly on the capillary basement membrane and endothelial cells [9].

Although a single case of death due to acute kidney injury and coagulopathy leading to death following *H. zara* envenoming is reported in the literature, we did not find any reported cases of pulmonary hemorrhage due to hump-nosed viper bite. Almost all cases of pulmonary hemorrhage have been reported for Bothrops species bites which are endemic to Central and South America. Bothrops venom has both procoagulant and anticoagulant properties and pulmonary hemorrhage is secondary to anticoagulant properties [10]. There was only one case of a fatal outcome due to pulmonary hemorrhage following Russel's viper bite reported from Sri Lanka [11]. This patient continued to bleed into the lungs despite normalization of clotting parameters and anti-venom therapy. In a large case series of Russell's viper bites described (336 patients) by Kularatne et al. [12], although 77% of patients had evidence of coagulopathy as demonstrated by a nonclotting 20-min whole-blood clotting test, none showed frank pulmonary hemorrhage. Disseminated intravascular coagulopathy was observed only in seven patients (2%).

Pulmonary hemorrhage is a rare complication of snake bite [13]. This is the first case reported from humped-nose viper bite, which was diagnosed based on the clinical criterion. Furthermore, other common instances where IV methylprednisolone is given are pulmonary hemorrhages due to vasculitis, connective tissue disorders, and leptospirosis etc. Our experience in using methylprednisolone in severe leptospirosis with pulmonary hemorrhages encouraged us to use it in this patient with an excellent response.

Conclusion

Pulmonary hemorrhages with HNV bite are extremely rare and occur due to direct toxicity rather than coagulopathy. It is associated with increased mortality and morbidity. Early consideration of pulmonary hemorrhages in a hypoxic patient led to treatment with steroids with a successful outcome and early weaning off from the mechanical ventilation.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contribution

All the authors were involved in the management of the patient. As wrote the first draft. KW and JP revised it. All authors have read and approved the final manuscript.

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