CASE REPORT

Monitor lizard bite-induced acute kidney injury – a case report

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Abstract

Envenomations by venomous lizards are rare. Monitor lizard bite-induced acute kidney injury (AKI) is a previously unreported complication in humans. A 55-year-old female was bitten on her right leg during farming activity by a monitor lizard (Varanus bengalensis). The patient experienced severe local pain and bleeding from the wound, coagulopathy, hemolysis, rhabdomyolysis, sepsis, and AKI. Patient was treated with supportive care and peritoneal dialysis but succumbed to a sudden cardiac arrest. Post mortem kidney biopsy revealed pigment induced-acute tubular injury. AKI after monitor lizard envenomation is caused by acute tubular injury in the setting of intravascular hemolysis, rhabdomyolysis and sepsis. Coagulopathy and direct nephrotoxicity may be the other contributory factors in causing AKI.

Keywords

Acute kidney injury, envenomation, hemolysis, monitor lizard rhabdomyolysis

History

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Introduction

Monitor lizards are large-sized reptiles found throughout Asia, Africa, and Australasia, and occur as ground-dwelling, arboreal, and semi-aquatic forms.¹ The Bengal monitor (Varanus bengalensis) or common Indian monitor (Figure 1), is found widely distributed over South Asia. It is found in river valleys in eastern Iran, Afghanistan, Pakistan, India, Nepal, Sri Lanka, Bangladesh, and Burma. It is mainly terrestrial, often found in agricultural areas below 1500 m altitude.² It has been reported from North Indian states of Himachal Pradesh, Punjab, parts of Jammu & Kashmir, and Uttar Pradesh. Folk belief has it that they are venomous, and in Sri Lanka, their breath is believed to be poisonous.

Until a few years ago, the only two species (Gila Monster and the Mexican Beaded Lizard) of venomous lizards were known. Venom of Heloderma species contains a number of protein and nonprotein components including serotonin, a bradykinin-releasing substance, protease, hyaluronidase, helodermin, and gilatoxin.³ Recent work has revealed that Monitor and Iguana lizards produce venom capable of producing hypotension and coagulopathy.⁴ Envenomations by venomous lizards are rare. A few case reports of lizard envenomation by a Mexican beaded lizard (Heloderma horridum)⁵,⁶ and a Gila monster (Heloderma suspectum)⁷,⁸ have been published. Further, there is a case report of septic arthritis due to a Savannah Monitor lizard bite.⁹ We report an envenomation by a monitor lizard (Varanus bengalensis) that resulted in coagulopathy, intravascular hemolysis, rhabdomyolysis, and acute kidney injury (AKI).

Case report

A 55-year-old woman while working in her farms was bitten on her right leg by a monitor lizard locally called goh (Varanus bengalensis). She experienced severe local pain and had blood oozing from the wound. Patient had anxiety symptoms of nausea, diaphoresis, dizziness, and breathlessness. She was evaluated at local hospital where she was found to be hemodynamically stable [pulse rate 97/min, blood pressure (BP) 120/90 mm of Hg] with a respiratory rate (RR) of 14/min and had hematoma right leg. Patient was given injections of antihistaminic, hydrocortisone, tetanus toxoid, and referred to this hospital.

She arrived at this hospital 9 h after the bite. She had complaints of generalized body aches and had decreased urine output. There was no gross hematuria or bleeding from any other site. There was no history of vomiting, chest pain, fever, seizure, or altered sensorium. Patient was drowsy and had pulse rate 80/min, BP 120/80 mmHg, RR 12/min. There was bite mark with surrounding swelling and tenderness suggestive of cellulitis in the right leg below knee. No abnormality was found on systemic examination. Her emergency laboratory investigations were: Hemoglobin (Hb) 13.5 g/dL, total white blood cell (WBC) count 14,600/mm³, blood urea...
68 mg/dL, creatinine 1.1 mg/dL, sodium 137 meq/L, and potassium 4.6 meq/L. The results of investigations done on day 2 were: Hb 9.2 g/dL, total WBC count 20,850/mm³ with 85% neutrophils, platelets 190 x 10³/mm³, WBCT > 20 min, blood urea 181 mg/dL, creatinine 4.3 mg/dL, sodium 132 meq/L, potassium 6.7 meq/L, chloride 104 meq/L, calcium 8.8 mg/dL, phosphorous 4.8 mg/dL, uric acid 10.2 mg/dL, protein 6.6 g/dL, albumin 3.5 g/dL, bilirubin 1.1 mg/dL, aspartate aminotransferase 441 IU/L, alanine aminotransferase 156 IU/L, alkaline phosphatase 60 U/L, blood sugar 106 mg/dL, cholesterol 168 mg/dL, serum creatine phosphokinase 13,650 U/L (Normal range 5–190 U/L), and lactate dehydrogenase 1070 IU/L (normal 200–400 IU/L). Arterial blood gas revealed pH 7.20, paO₂ 80 mmHg, paCO₂ 20, HCO₃ 8 meq/L, SaO₂ 95%. Urinalysis showed a specific gravity 1.020, pH acidic, albumin 3+, RBCs 15–20 phf, WBCs 10–15 phf, urine sodium 25 mmol/L, and urine culture was sterile. Electrocardiogram was normal. No abnormality was found on chest X-ray.

The doctors in the emergency department were unsure of what to offer as a specific antidote to this reptile bite and decided to offer polyvalent anti-snake venom (ASV) which is against four major species of snakes: *Naja naja*, *Bungarus caeruleus*, *Daboia russelii*, and *Echis carinatus*, believed to be responsible for most snake bite deaths in India. In fact, patient received 200 mL of ASV. But as identity was confirmed by the witnesses and local wildlife authorities, no further ASV was administered. Patient was treated with intravenous fluids and broad spectrum antibiotics to cover gram positive, gram negative, and anaerobic bacteria. The investigations on day 2 suggested that patient had developed severe neutrophilic leukocytosis, intravascular hemolysis, rhabdomyolysis and AKI. Patient was given acute peritoneal dialysis (PD) by carrying out one hourly exchanges of PD fluid for 32 h. Patient had symptomatic improvement, WBCT normalized, azotemia (Figure 2) and urine output were improving. On day 3, patient succumbed to a sudden cardiac arrest after about 72 h of sustaining the bite. Patient was not subjected to autopsy but a post mortem kidney biopsy was carried out. The morphological features on kidney biopsy were consistent with acute tubular injury associated with pigment nephropathy (Figure 3).

**Discussion**

The monitors are carnivorous lizards who sometimes use their tongues like snakes do, to detect a prey by picking up scent particles while flicking it in and out. Unlike snakes, they do not inject their venom in the wound; venom merely flows into the wound. Luckily, attacks on people are uncommon for these reptiles are known to bite with tenacity. Monitor lizards tend to avoid confrontation and rather try to escape. Bites are only inflicted when they are manipulated or maintained in an inappropriate manner or if they are cornered and feel threatened.¹

Nine types of lizard toxins are shared with snakes, but some toxins are new and yet to be investigated. Unlike the hollow-fang delivery system of snakes, the venom of these lizards simply pools around their teeth and enters wounds when the prey is bitten.⁴ Furthermore, it is now thought that venom production had, actually, a single early origin for lizards and snake and that the common ancestor to all venomous species lived about 200 million years ago.
There is a wide variety of pathogenic and lethal bacteria have been demonstrated in the saliva of monitor lizards, and the wounds inflicted by this animal are often associated with sepsis and subsequent bacteremia. The saliva of the Komodo dragon (Varanus komodoensis) was found to contain 28 Gram-negative and 29 Gram-positive species of bacteria. The pathogenic bacteria like Escherichia coli and Pasteurella multocida, were found in the saliva of wild dragons but these species were not present in captive dragons. Indeed, prior to the discovery of the salivary toxins, bacteria were widely thought to be the sole cause of the rapid death observed in bitten prey. In a recent study done on the captive Komodo dragon (Varanus komodoensis), the oral flora was found to contain 39 aerobic and 21 anaerobic species of bacteria but no virulent species were isolated. This suggests that as with other carnivores, captive Komodo oral flora is simply reflective of the gut and skin flora of their recent meals and environment and is unlikely to cause rapid fatal infection.

Bites by hemotoxic snakes and myotoxic snakes are the common causes of renal involvement especially AKI. Therefore, AKI is often associated with hemorrhagic diathesis, intravascular hemolysis, and rhabdomyolysis. Renal pathological changes include mesangiolysis, glomerulonephritis, vasculitis, tubular necrosis, interstitial nephritis, and cortical necrosis. Tubular necrosis is an important pathological counterpart of AKI. Hemodynamic alterations induced by cytokines and vasoactive mediators leading to renal ischemia are important in the pathogenesis of AKI. Hemolysis, intravascular coagulation leading to glomerular microthrombi formation and rhabdomyolysis are important contributing factors. Direct nephrotoxicity can be induced by the venom through metalloproteases and phospholipase A2. Immunologic mechanism plays a minor role in the pathogenesis of the renal lesion. Though monitor lizards have recently been discovered to be venomous, there has never been a recorded death by one of them in the USA where they are popular pets. Our patient had severe leukocytosis, intravascular hemolysis, coagulopathy, and rhabdomyolysis. Kidney biopsy revealed acute tubular injury associated with pigment nephropathy. Our patient had coagulopathy but the fibrin microthrombi were not seen on histopathological examination. Therefore, from the clinical picture and morphological features seen on kidney biopsy, it can be concluded that the monitor lizard venom is capable of producing intravascular hemolysis, coagulopathy, and rhabdomyolysis. Particularly, the venom of a wild monitor can cause sepsis as was observed in this reported case. All these pathogenic mechanisms were responsible for causing the AKI. Further, as with other biological nephrotoxins, the direct nephrotoxicity of the lizard venom may have played a role in the AKI. All these pathogenic mechanisms are very similar to those seen with snakebite-related AKI.

Despite all supportive measures the outcome was not good in the reported case. The patient succumbed to a sudden cardiac arrest due to either electrolyte disturbances especially hyperkalemia or an acute coronary event. Though the laboratory reports 6 h before the demise showed hyperkalemia was corrected, but hyperkalemia from a transient or ongoing hemolysis and or rhabdomyolysis cannot be ruled out. Further, due to the lack of autopsy, coronary thrombosis cannot be excluded as a cause of death.

The complication of monitor lizard bite-induced AKI is a previously unreported complication in humans. It should be a warning to researchers who work with these animals or to laypersons that encounter these lizards in the wild or keep them as pets. No antivenom is commercially available and treatment is essentially a supportive care. Authors suggest that these cases should be hospitalized early and saline hydration and sodium bicarbonate administration for alkalization of urine are reasonable approach to prevent and treat the pigment-induced AKI.

**Declaration of interest**

All the authors declared no competing interests.

**References**