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Case Report

Unusual presentations of acute kidney injury and neurologic complications due to snake bite

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Abstract

Introduction: Vascularity of kidneys is very high, so these organs are potentially susceptible to be affected with toxins including snake venom. Hypersensitivity to snake venous could cause some neurological problem.

Case Report: We present a 14-year-old boy with acute kidney injury (AKI) due to snake bite. After a few days, kidney failure with hematuria was developed. His serum creatinine level rose to 3 mg/dl and following 2 weeks gradually and decreased to normal level without any special treatment except for anti-venom, which was not prescribed inappropriate time (this type of AKI is not reported previously). He had seizure attacks, which were according to magnetic resonance imaging due to posterior reversible encephalopathy syndrome (PRES) (This neurologic complication has been seen in other kidney injuries but up to now it was not reported in snake bite victims).

Conclusion: Sanke venom could cause PRES due to AKI and seizure could be one of the most important complications in snake bite.

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Introduction

Vascularity of kidneys is very high, so these organs are potentially susceptible to be affected with toxins, including snake venom. Renal involvement has so many different features such as proteinuria, hematuria and renal failure.[1] Renal damage may be the reason of hypovolemia and hypoperfusion, thrombotic microangiopathy, rhabdomyolysis and of venom cytotoxicity on the renal tubules.[2] Glomerular lesions are not seen frequently, but there are rare reports of proliferative and rapidly progressive glomerulonephritis.[3] A direct cytotoxic effect on the kidney may occur. However, it is still not documented with other studies. Hypersensitivity to venomous or anti-venomous protein occasionally causes acute renal failure.[4]

Case Report

A 14-year-old boy presented due to snake bite. He had abdominal pain and was admitted to infectious disease ward. His initial vital signs and lab exams were normal, and there were not any petechiae or purpura. He had not any previous history of renal disease. A peripheral blood smear was normal. Creatine kinase level was normal, and there was not any evidence of hemolysis. Fever was low grade (38 °C) and the blood pressure was 150/80 mmHg. Urine examination revealed dysmorphic red blood cells (RBCs) (without casts) so glomerulonephritis was considered.

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Creatinine level rose to more than 3 mg/dl. Four vials of anti-venom were infused. Lab examination results were as below: Urine volume: 2800 cc/24 h, protein in urine: +3 (1530 mg) erythrocyte sedimentation rate: 80 mm, prothrombin time (PT): 15 s, international normalized ratio: 1.5, platelet: 210000, hepatitis B core antibody: (Negative), hepatitis B surface antigen: (Negative), Cr: 2.7 mg/dl, urea: 239 mg/dl, white blood cell (WBC): 10900, albumin: 2.8 g/dl, anti-nuclear antibodies: (Negative), antistreptolysin O (ASO): 250 u, antineutrophil cytoplasmic antibody: (Negative), C3 and C4: decreased. Kidney biopsy was planned, but it was not allowed by patient's parent. His urea and creatinine level got better during next days. 7 days thereafter an episode of tonic colonic seizure happened. Magnetic resonance imaging (MRI) were ordered. Bilateral hyper-signal areas in the occipital region were detected (Figure 1).

According to MRI, posterior reversible encephalopathy syndrome (PRES) was considered, and sodium-valproate started. He was discharged with normal serum creatinine and urea level (0.7 and 34 mg/dl respectively). 3 weeks after discharge, there was not any evidence of edema. Creatinine and urea level again was normal. Urine analysis revealed mild hematuria (6-8 RBCs).

Discussion

Usual systemic manifestations of snake bite are hemolysis, rhabdomyolysis, hypotension and shock which may lead to renal damage and may be kidney failure.[5]

The renal histology mainly consists of acute tubular necrosis and acute interstitial nephritis, while glomerular changes are rare.[1] Abdominal pain is a common feature. The presence of abdominal pain and its severity is correlated with the severity of coagulopathy, neurotoxicity, kidney involvement.[6] It was detected in our patient.

Acute kidney injury (AKI) developed in 31.0% of patients with snake bite, leading to mortality in 39.1% patients. Factors associated with AKI are a bite to hospital time, hypotension, albuminuria, prolonged bleeding time, prolonged PT, low hemoglobin and a high total bilirubin,[7] which

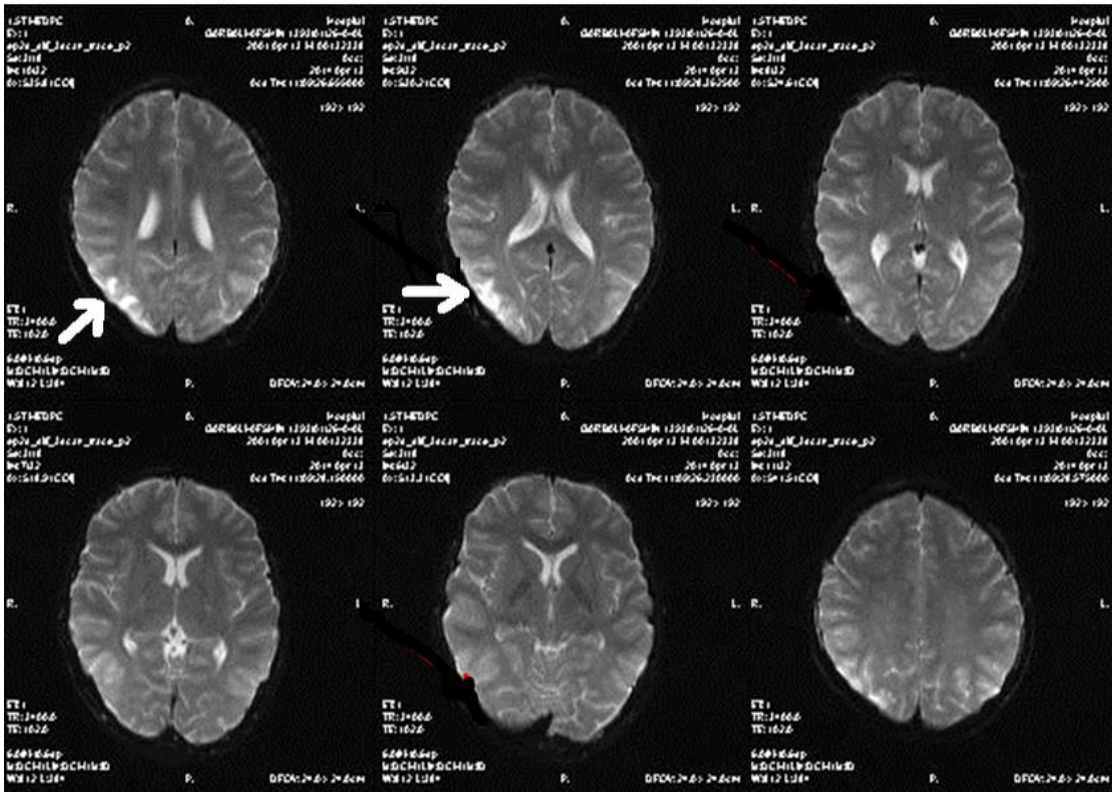

Figure 1. Hypersignal T2 area in both occipptial regions

they were not detected in the presented boy. Hypotension and shock may result from a snake bite. The presence of rhabdomyolysis also reported frequently, which is documented with elevated creatine phosphokinase and detection of myoglobine in urine, which was not seen in studied patient. Renal function of this patient got better after 14 days and 2 weeks after discharge his kidney function was good. Neurotoxicity is another complication, characterized initially by ptosis, diplopia, and bulbar palsy and respiratory paralysis but in literature review we could not find PRES due to snake bite. PRES is a clinic-radiographic entity, which has similar findings on neuroimaging and symptom are headache, vision loss, altered mentation, and seizures.[8,9]

The contributory factors were uncontrolled hypertension, severe hypoproteinemia, persistent hypocalcemia, hemolytic uremic syndrome, cyclosporine toxicity, lupus nephritis, and pulse methylprednisolone.[10] None of the above mentioned kidney involvements was not seen in this patient. Because of dysmorphic RBCs, hypertension, subnephrotic proteinuria, a kind of acute glomerulonephritis was suspected. Glomerulonephritis is reported very rarely due to snake bite[1] and it is interesting that kidney function improved in a short period of time.

Conflict of Interests

Authors have no conflict of interest.

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