

Kidney Failure Due to Abdominal Compartment Syndrome Following Snakebite

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Treatment of snakebite complications is challenging, as it is difficult to distinguish what kind of antivenins should be used. Kidney failure as a result of rhabdomyolysis or hemolysis may happen due to accumulated fluids that increase the pressure in the abdomen. This case report describes acute kidney failure probably due to intra-abdominal hypertension following an unknown bite.

Keywords. snakebite, acute kidney injury, abdominal compartment syndrome, intra-abdominal pressure

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INTRODUCTION

Worldwide, only about 15% of the more than 3000 species of snakes are considered dangerous to humans.¹ Mortality rate of snakebite is fairly low, but treatment of its complications is an issue, as it is difficult to distinguish what kind of antivenins should be used.² Kidney failure of patients with snakebites is related to rhabdomyolysis or hemolysis. Accumulated fluids increase the pressure in the abdomen. Elevation of the intra-abdominal pressure (IAP) causes some physiologic sequels such as kidney failure by reduction of glomerular filtration rate and develops oliguria or anuria. We describe a diagnosis of acute kidney failure probably due to intra-abdominal hypertension following an unknown bite, which was followed and treated for generalized edema.

CASE REPORT

A 60-year-old man was admitted to a local hospital because his left thumb was bitten while working in his garden. The patient had a good health condition prior to this unknown bite, which gradually became edematous and painful. After 3 hours, swelling started developing and progressing to his arm, and a bulla was gradually apparent. Two days later, edema spread to the left part of his body with ecchymosis appearing on his skin. At this time, the patient was hospitalized in a local

hospital, and edema progressed to his face and all other parts of his body by the 3rd day.

On the 4th day, he complained about 2 episodes of fever and chills, dyspnea, cough, rib pain, and flank pain, and 1 episode of hemoptysis. Due to severe dyspnea, he could not even lie down on his bed. Conservative treatment with no clear diagnosis was done. The laboratory tests and paraclinic evaluations results are shown in Table 1.

On the 7th day, the patient was released from the local hospital and was admitted to the Department of Clinical Toxicology at our referral hospital. In the emergency room, he had diffused edema and tightness of skin. He also complained about severe constipation, decreasing urinary volume over a period of 2 days and some grade of dyspnea. He has had a history of hypertension and benign prostatic hypertrophy. He indicated using tamsulosin, amlodipine, and multivitamin. He would live with his wife and work as a farmer and a car mechanic. No history of smoking, alcohol consumptions, or recreational drugs use was documented.

Examination of vital signs showed a blood pressure of 130/80 mm Hg, pulse rate of 90 per minutes, body temperature of 37.5°C, respiratory rate of 16 per minutes, and saturated oxygen pressure of 98% in room air. His consciousness was intact. Tight skin and nonpitting edema with tenderness and a few ecchymosis were detected all

Table 1. Test Results of First Admission to a Local Hospital

Parameter	Days From Snake Bite				
	2	3	4	5	6
Leukocyte count, × 10 ⁹ /L	13.3	13.3	16.3	14.0	14.7
Hemoglobin, g/dL	17.7	17.7	15.3	13.1	11.6
Platelet count, × 10 ⁹ /L	97	89	109	860	...
Blood glucose, mg/dL	164
Blood urea, mg/dL	29	...	53	...	42
Serum creatinine, mg/dL	1.4	...	1.4	...	2.0
Serum sodium, mg/dL	139	...	137	...	126
Serum potassium, mg/dL	5	...	4.2	...	4
Creatine phosphokinase-MB, IU/L	101	105
Creatine phosphokinase, IU/L	95	395
Partial thromboplastin time, sec	15	28	28
Prothrombin time, sec	13.8	12.9	12.7
International normalized ratio	1.30	1.20	1.07
Lactate dehydrogenase, mg/dL	730	756
pH	7.35	7.35	7.3
Carbon dioxide pressure, mm Hg	51	51	42
Bicarbonate, mg/dL	28	28.2	20.7

over his body. On physical examination, bulla on the left thumb, left axillary lymphadenopathy, fine rales in the bases of both lungs, a systolic ejection murmur (grade 2/6), and a mild tenderness in the lower abdominal quadrants without rebound were all positive findings.

In the toxicology service, 10 vials of Razi polyvalent antivenin were infused. A chest radiography revealed basilar effusion, with no evidence of pneumonia or basilar atelectasis of the left lung. The result of analysis of pleural effusion was transudate and urinalysis was normal, as shown in Table 2.

No sign of compartment syndrome was evident, and elevation of the left hand and the testis were advised by a consultant surgeon. Spiral and high-resolution computed tomography of the lung showed bilateral pleural effusion, infiltration of the right middle and right lower lobes, and basilar atelectasis of the left lung. Ipratropium and seroflu spray and intravenous meropenem and ciprofloxacin were administered. Echocardiography showed an ejection fraction of 55%, moderate tricuspid regurgitation, no pericardial effusion, and pulmonary arterial pressure of 50 mm Hg. Ultrasonography of the abdomen was normal. Amount of creatine phosphokinase, reticulocyte count, level of bilirubin, and serum haptoglobin were within normal ranges. Peripheral blood smear showed no schistocyte.

On the 9th day, after initiation of antibiotic

therapy, fever and sputum stopped and dyspnea alleviated. The results of blood culture and urine culture came back negative. During this time, creatinine and blood urea nitrogen were continuously rising and urine volume was decreasing. Finally, the patient became anuric.

Results were negative for antineutrophil antibody; anti-double-stranded-DNA; complements C3, C4, and CH50, hepatitis B surface antigen, hepatitis C antibody, antineutrophil cytoplasmic antibodies, anticardiolipin antibodies, human immunodeficiency virus antibody, and Coombs direct and indirect tests. Urine was stained by Hansel stain and Wright stain for possibility of interstitial nephritis, which showed that eosinophilia was not present. There was not any clinical or paraclinical sign of collagen disorder, but the patient still had some degrees of dyspnea and abdominal pain with very tight skin.

On the 12th day of post-bite, we decided to check IAP in order to rule out intra-abdominal hypertension (IAH) every 6 hours, which was 14 mm Hg. At the same time, necessity of hemodialysis and kidney biopsy was discussed with the patient to inform him about development of kidney failure and its suitable treatment, which he did not accept. After day 15th, texture of his skin changed, the urine output increased, creatinine level significantly decreased, and IAP gradually reduced from 14 mm Hg to 5 mm Hg, in 5 days. The patient was discharged on the 16th day with a creatinine of 4

Table 2. Test Results in Referral Center

Parameter	Days From Snake Bite																			
	7	8	9	10	11	12	13	14	15	16	17	18	19	20						
Leukocyte count, × 10 ⁹ /L	9.7	9.4	8.8	8.7	8.1	6.0	4.8	1.0						
Hemoglobin, g/dL	9.3	8.1	8.7	9.3	9.4	8.1	8.3						
Platelet count, × 10 ⁹ /L	95	101	168	176	176	211	206						
Blood urea, mg/dL	96	121	141	156	172	208	200	168	161	126	103	...						
Serum creatinine, mg/dL	5.5	7.3	7.5	...	8.5	10.2	12.4	14.1	15.8	16.5	17	5.9	4.9	4.2						
Serum sodium, mg/dL	131	133	132	...	133	134	134	129	135	134	140	142	140	...						
Serum potassium, mg/dL	3.8	3.7	3.8	...	4.3	4.4	4.2	4.5	4	4.5	4.9	5	4.3	...						
Serum calcium, mg/dL	8.1	8.2	8.7						
Serum phosphorus, mg/dL	5.4	6.2	5.4						
Creatine phosphokinase-MB, IU/L	14	20	9						
Creatine phosphokinase, IU/L	229	179	73	...	37	42	76						
Partial thromboplastin time, sec	37.7	34	36.5	32						
Prothrombin time, sec	13.1	13.5	13.9	14	13.4						
International normalized ratio	1.16	1.23	1.30	1.21						
Serum protein, mg/dL	5.9	6.1						
Lactate dehydrogenase, mg/dL	930	837	991	932	872	...	665						
pH	7.48	7.41	7.47	...	7.38	7.43	7.33	...	7.35	7.43	7.42	...						
Carbon dioxide pressure, mm Hg	33.4	44.2	34.3	...	36.1	34.6	35.4	34.8	30.1	37.5						
Bicarbonate, mg/dL	25.1	23	24.9	...	21.4	21.4	18.4	18.8	19.6	24.2						
Urinalysis	pH, 6.5; specific gravity, 1028; blood, 2+; leukocyte, 9 to 11 per HPF; erythrocyte, 20 to 25 per HPF; no cast																			
Urine volume, mL	400	300	200	150	-	-	50	20	1500	2600	3200	3500	4000	...						

mg/dL. A week later, the patient was in a good condition and stated that a large snake was found and killed in his garden. His laboratory tests showed a hemoglobin level of 12.3 g/dL; serum creatinine of 1.1 mg/dL, blood urea nitrogen of 16.5 mg/dL, serum sodium of 141 mg/dL, and normal urinalysis.

DISCUSSION

Snake venoms are complex mixtures of enzymes. Hemorrhaging venoms are harmful components that promote vascular leakage and cause both local and systemic bleeding. Proteolytic enzymes cause local tissue necrosis, affect the coagulation pathway at various steps, and impair organ function. The most important aspect of prehospital care of snake bitten patients is supportive care (airway, breathing, and circulation) and antivenin administration.³ The best results are achieved when the antivenin is initiated within 24 hours.⁴

Accumulated fluids increase the pressure in the abdomen. Once the IAP progresses to more than 12 mm Hg, it is defined as IAH, a syndrome which is found in 30% to 50% of the patients.⁵ Unnoticed IAH may develop to organ failure, which is referred to as abdominal compartment syndrome.⁵ Intra-abdominal hypertension and abdominal compartment syndrome cause some physiologic sequels by elevation of IAP.⁶ In the kidney, elevation of IAP causes compression of renal veins and parenchyma and reduction of cardiac output to the kidneys and renal congestion, which reduces glomerular filtration rate and develops oliguria and anuria.^{6,7}

Kidney failure of patients with snakebites is related to rhabdomyolysis or hemolysis.⁸ Although rhabdomyolysis has been named as the most common cause of renal insufficiency for snakebite victims, this diagnosis was not confirmed in our patient.

Theoretically, some components of snake venom may be responsible for capillary leakage and developing IAH and abdominal compartment syndrome. Even though we had some time delay to

measure IAP, the reduced level of IAP and clinical and paraclinical factors of our cases encourage us to consider IAH as a cause of acute kidney failure. To support of this idea, we recommend that IAP be measured for snakebite victims as soon as suspected.

CONFLICT OF INTEREST

None declared.

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