

Rhabdomyolysis and Acute Renal Failure after Poisonous Snake (*Agkistrodon halys blomhoffii*) Bite

Key words: continuous hemofiltration, myoglobin

Most victims of mamushi (*Agkistrodon halys blomhoffii*) bites do not develop severe symptoms. Fatal cases are relatively rare (1). We describe an elderly patient who developed rhabdomyolysis and acute renal failure after a mamushi bite treated with continuous hemofiltration (CHF) and hemodialysis.

An 80-year-old woman was brought to hospital because of left arm pain and swelling. The day before admission, while she was farming, she was bitten on the left forefinger by a mamushi. Afterwards, her left arm became swollen and urine color turned dark brown. On admission, blood pressure was 164/88 mmHg, pulse was 80 beats/min, and temperature was 37.4°C. Physical examination was normal except for two bite marks on the left forefinger and marked left arm swelling. Laboratory parameters showed leukocyte count 20,600/ μ l, platelet count 285,000/ μ l, and fibrin/fibrinogen degradation products (FDP) of 9.3 μ g/ml. Aspartate aminotransferase, alanine aminotransferase, lactate dehydrogenase, and creatine kinase levels were 1,488 IU/l, 383 IU/l, 3,516 IU/l, and 47,509 IU/l, respectively. Blood urea nitrogen, serum creatinine, potassium, and bicarbonate levels were 35.6 mg/dl, 1.01 mg/dl, 4.6 mEq/l, and 17.3 mEq/l, respectively. The patient was oliguric. Rhabdomyolysis and acute renal failure caused by mamushi bite were diagnosed. Ten mg of cefaranthin and 1 g of cefazolin per day were administered intravenously. Antivenom was not administered. Hydration and intravenous furosemide administration failed to increase her urine output. On day 2, creatine kinase and serum creatinine rose to 92,568 IU/l and 2.33 mg/dl, respectively. Serum and urine myoglobin were 104,000 ng/ml and over 250,000 ng/ml, respectively. We performed twenty hours of CHF using 1.0 m² polyacrylonitrile membrane (APF-10S; Asahi Medical, Tokyo) for renal replacement therapy and myoglobin removal. Myoglobin level of filtered fluid (13,360 ml of total volume) was 40,000 ng/ml. On day 3, we changed the extracorporeal modality to intermittent hemodialysis and continued it for two weeks. On day 17, the patient entered a diuretic phase. Despite the further increase of FDP, definite disseminated intravascular coagulation (DIC) was not observed. She made a complete recovery. Renal biopsy was not performed because of her high age.

Chugh mentioned that the pathology of renal failure after snake bite is acute tubular necrosis or cortical necrosis (2). Possible factors contributing to renal injury include nephrotoxicity of venom, hypotension, intravascular hemolysis, myoglobinuria, DIC, and hypersensitivity to venomous or antivenomous protein (2). In the present case, any direct effect of venom on the kidney was unclear. However, renal failure was probably caused in part by rhabdomyolysis.

The mechanisms of myoglobinuric renal injury are considered to be renal vasoconstriction, intraluminal cast formation, and direct heme protein-induced cytotoxicity (3). Myoglobin has a molecular weight of 17,000 and is poorly cleared by dialysis. In the present case, twenty hours of CHF could remove approximately 530 mg of myoglobin. Hemofiltration is probably useful for treatment of myoglobinuric renal injury (4).

In addition to specific antivenom administration, maintenance of the extracellular volume, adequate hydration, and alkalization of urine are important in victims of snake bite (5). Antivenom therapy in mamushi bite patients is controversial. Kimoto et al (1) showed a favorable outcome using a minimal dose of antivenom, methylprednisolone, and cefaranthin in mamushi bite victims. However, allergic reactions to antivenom are known to occur in about 15% of patients (2). Further studies are needed to determine whether routine antivenom use is indicated for mamushi bite victims.

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