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A Case Report of Angioedema

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Angioedema is characterized by localized and self-limiting edema of the subcutaneous and submucosal tissue. Hereditary angioedema is rarely manifested in Korea. Factor XII mutation was recently found to be associated with subtype of hereditary angioedema occurring predominantly in female. A 32 years old female presented with recurrent angioedema since first occurrence of 7 months ago. She was shown to have swelling of both cheeks and lips, and abdominal pain. She had been taking oral contraceptive for 2 years and diagnosed of diabetes 7 months ago, however, not taking medication for the treatment of diabetes. Family history of angioedema was absent. C1 esterase inhibitor level and its activity, C4 level was normal range. Although, she was treated with antihistamines and systemic glucocorticoid for 2 weeks, her angioedema symptoms were not controlled. She was admitted and treated with danazol, tranexamic acid along with intravenous corticosteroid and intramuscular epinephrine. Oral hypoglycemic agent and antibiotics were also added to treat her diabetes and urinary tract infection. When her symptoms were controlled, she was discharged and has been maintaining with antihistamine, danazol and tranexamic acid without recurrence of angioedema. Factor XII mutation analysis was undertaken to find a missense mutation because her angioedema was suspected to be associated factor XII mutation. However, mutation was not found. We report an angioedema case with unknown mechanism, which was successfully managed with danazol, tranexamic acid and supportive care.

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Acute kidney injury and systemic rhabdomyolysis following repeated bee envenomation

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Introduction: Acute kidney injury and rhabdomyolysis are rare complications by bee sting. Interestingly, this case presents sudden induction of systemic rhabdomyolysis and acute renal failure after repeated bee envenomation more than decades. Bee envenomation in local upper extremity induced systemic rhabdomyolysis and acute renal failure in absence of anaphylaxis. **Case report:** A 70-year-old male with no past medical, allergic and trauma history was presented with hematuria, oliguria and myalgia after bee envenomation in neck and arms. He was hemodynamically stable and initial lab findings revealed high anion gap metabolic acidosis and acute kidney injury (Blood nitrogen urea/Creatinine 65/5.2mg/dL), hypocalcemia (ionized calcium 0.81mmol/L), hyperuricemia (uric acid 13.8mg/dL) and Creatinine Kinase> 14,000IU/L, Myoglobin> 20,000IU/L. Urine analysis identified proteinuria (3+), hematuria (RBC 10-19/HPF), pyuria (WBC 5-9/HPF), urine sodium 108mmol/L, potassium 21mmol/L, chloride 90mmol/L, Cr 89mmol/L, and osmolality 299mosm/kg. Urine myoglobin was elevated (9659 ng/mL). Kidney biopsy reported acute tubular necrosis with renal parenchyma preserved and some eosinophilic casts in parts of distal tubule without immune complex staining. Even though venom was injected in local areas of neck and upper extremity, it generated systemic rhabdomyolysis involving multiple muscles; both upper and lower extremities and abdominal wall (Figure 1). **Discussion:** Bee venom can induce acute hypersensitivity such as anaphylaxis and delayed reaction in form of serum sickness. After two weeks of asymptomatic period, he presented typical serum sickness like symptoms such as myalgia, proteinuria that is improved with steroid application. Both bee venom specific IgE and IgG were elevated and complement was normal without immune complex formation, implying it is IgE mediated delayed reaction. Systemic rhabdomyolysis is secondary to myotoxicity of venom and acute kidney injury appears mainly due to rhabdomyolysis and toxic effects of venom. After a month of supportive care and hemolytic dialysis, renal recovery was validated enough to be sustained without renal replacement therapy.