

Case Report

Wasp Sting-Induced Acute Kidney Injury – A Case Report

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ABSTRACT

A 22-year old male student presented with a 3 weeks history of multiple wasp stings and subsequent progressive generalized body swelling, reduction in urine output and passage of coke colored urine. At presentation, he had anasarca, jaundice but not pale. Admitting blood pressure was 160/90mmhg. Bedside urinalysis revealed protein (3+), blood (2+) and urobilinogen (3+). The admitting serum biochemistry showed markedly elevated urea of and creatinine. A diagnosis of Wasp-induced acute kidney injury most likely pigment nephropathy from intravascular haemolysis was made. The patient had 5 sessions of intermittent hemodialysis and made full clinical and renal recovery. This presentation highlights a rare case of acute kidney injury from multiple wasp stings.

Keywords: Wasp sting, acute kidney injury, rhabdomyolysis, intravascular haemolysis, acute tubular

CASE PRESENTATION

Mr A.J. is a 22 year old male student who was referred from a private hospital to our emergency unit on account of three weeks history of multiple wasp stings, and two weeks history of progressive generalised body swelling associated with reduction in urine output and passage of coke coloured urine.

The patient sustained multiple wasp stings, more than 20 stings involving mostly the face, upper limbs and trunk while working in the farm, this was immediately followed by the development of severe pain at the site of the stings. He also developed

transient erythematous skin rash with accompanying intense pruritus and swelling of the face, lips and tongue and features suggestive of acute urticaria with angioedema which resolved after interventions at a nearby peripheral health centre.

A week later, he developed progressive bilateral lower limb swelling, facial swelling and abdominal swelling. He had no associated cough, difficulty in breathing, orthopnoea or paroxysmal nocturnal dyspnoea.

At about the same time he noticed a reduction in his urine output, estimated daily urine output was about

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400mls, it was frothy and coke-coloured, but no frank (macroscopic) haematuria. He had no pruritus, hiccups or recurrent vomiting. There was no altered sleep pattern, irrational talk or loss of consciousness. He had no fever or sore throat prior to the incident and onset of symptoms, and no history of chronic ingestion of non-steroidal anti-inflammatory drugs or herbal concoctions.

He is not a previously known diabetic or hypertensive but the blood pressure was elevated in the course of this illness. He had no history of acute kidney disease, and no previous blood transfusions. His genotype is AA.

The father was hypertensive but no family history of diabetes or chronic kidney disease. He does not smoke cigarettes nor take alcoholic beverages, and he had no history of illicit drug use.

Since the onset of the illness, he had been to a private hospital where he was given some medications that made the initial swelling to regress and was referred to our emergency unit on account of significantly elevated deranged serum electrolytes, urea and creatinine.

General physical examination revealed a young man with anasarca, he was not pale. He was icteric but afebrile with an axillary temperature of 36.8°C. There was no lymphadenopathy or digital clubbing. Cardiovascular examination revealed a pulse rate of 80 beats/min which was regular and of good volume. The blood pressure was 160/90mmhg, the jugular venous pressure was not elevated, the apex beat was localised at the 5th left intercostal space, mid-clavicular line and it was not heaving. The 1st and 2nd heart sounds were heard with no pericardial friction rub or additional sounds. The respiratory examination revealed a respiratory rate of 26 cycles per minute and fine bibasalcraepitations. His abdomen was distended but moved with respiration, he had no areas of tenderness or palpable organomegaly but had ascites demonstrable by shifting dullness. The nervous system examination was essentially normal with no asterixis.

A diagnosis of acute kidney injury precipitated by multiple wasp stings.

The results of the investigations are summarised below as in tables 1, and 2:

- **Full Blood Count:** Packed Cell Volume- 34%, haemoglobin-11.2g/dl, white cell count- $4.9 \times 10^9/l$, neutrophils-66%, lymphocytes-30%, others- 4%, platelet count- $252 \times 10^9/l$, reticulocyte count- >2.5%, peripheral blood film revealed polychromasia and nucleated red blood cells.
- **Liver function test:** Alanine transaminase- 41 IU/L (0-15 IU/L), Aspartate transaminase- 49 IU/L (0-20 IU/L), Alkaline phosphatase- 72 IU/L (9-35 IU/L), Total bilirubin- 76 $\mu\text{mol/l}$, Conjugated bilirubin- <20 $\mu\text{mol/l}$, Total protein- 66 g/l (60 – 80 g/l), Albumin- 36 g/l (35 – 50 g/l)
- **Urine Microscopy, Culture and Sensitivity:** Appearance- coke colored, Pus cells: 0 – 3/hpf, Red blood cells: 0 – 1/hpf, Epithelial cells, Casts: granular casts (2+), Culture: yielded no growth.
- **Hepatitis-B Surface Antigen screening:** Negative
- **Anti-Hepatitis-C Virus screening:** Negative
- **Human Immunodeficiency Virus screening:** Negative
- **Abdomino-pelvic ultrasound scan:** Both kidneys are at the upper limit of normal in size (Right-122mm \times 29mm, Left- 126mm \times 52mm), they both showed increased cortical echoes and poor cortico-medullary differentiation.
- **At Presentation:** Calcium-2.02mmol/l, Phosphate-2.4mmol/l, Urate-0.9mmol/l, Total Protein-66g/l, Albumin-36g/l
- **3 months post-discharge:** Calcium- 2.35mmol/l, Phosphate-1.0mmol/l, Urate- 0.45mmol/l, Total Protein-68g/l, Albumin-40g/l

SUMMARY OF TREATMENT

Table 3 shows the fluid input and output of the patient. He remained oliguric and subsequently had 5 sessions of haemodialysis with markedly reduced fluid retention, serum biochemistry and increased urine output. He was discharged home on the 14th day of admission. At one-month post discharge, he had no evidence of fluid retention, the blood pressure was 110/80mmhg and his dipstick urinalysis

revealed a trace of protein while serum creatinine was 112µmol/l.

At his follow-up clinic visit three months after discharge all clinical and biochemical parameters were within normal limits.

Table 1: Serial serum electrolytes, urea and creatinine

Date	Sodium (mmol/l)	Potassium (mmol/l)	Urea (mmol/l)	Creatinine (µmol/l)
At presentation	130	5.3	23.4	1157
Post 1 st dialysis session	127	4.4	29.0	948
Post 3 rd dialysis session	129	3.6	16.7	570
Post 4 th dialysis session	129	3.6	14.7	230
Post 5 th dialysis	133	3.4	12.4	140
1 month post discharge	128	3.0	6.0	112
3 months post discharge	136	3.6	4.8	98

Table 2: Serial urinalysis

Date	Protein	Blood	Specific gravity	pH	Urobilinogen
Day 1	3+	2+	1.015	5.0	3+
Day 5	2+	2+	1.020	5.0	2+
Day 10	2+	1+	1.020	6.0	1+
Day 14	1+	Negative	1.025	5.5	Negative
1 month post discharge	Trace	Negative	1.025	5.0	Negative
3 months post discharge	Negative	Negative	1.030	5.5	Negative

Table 3: Fluid input and output chart

Date	Input (mls)	Output (mls)
Day 1	500	150
Day 2	1350	200
Day 3 (1 st session of haemodialysis)	1620	270
Day 5 (2 nd session of haemodialysis)	1790	570
Day 7 (3 rd session of haemodialysis)	1800	1200
Day 8	1750	3500
Day 10	3200	2800
Day 12 (5 th session of haemodialysis)	3000	2500

DISCUSSION

Wasps, hornets and yellow-jackets belong to the family Vespidae and the order Hymenoptera.^{1,2} Other medically important insects belonging to the hymenopterid order include the families of Apidae (bees) and Formicidae (ants).² Wasp sting is an occupational hazard in the tropics particularly among farmers, like in this patient.² The clinical manifestations of wasp stings are diverse, ranging from mild local allergic reactions to severe systemic reactions and death. This depends on the patient's sensitivity to the venom and the number of stings.³ The clinical symptoms resulting from wasp stings can be classified into allergic and toxic reactions.³ Allergic reactions may involve dermal symptoms such as erythema, pruritus, urticaria, and angioedema which this patient developed shortly after being stung. Other allergic reactions include respiratory distress from laryngeal edema and bronchial constriction as well as cardiovascular collapse from anaphylactic shock and cardiac arrest.^{5,6} Localized allergic reactions are usually self-limiting, lasting 2-3 days.³ Toxic reactions resulting from direct effects of the venom include acute kidney injury (AKI), rhabdomyolysis, acute pancreatitis, hepatic dysfunction (centrilobular necrosis), and cardiac dysfunction (toxic myocarditis, myocardial infarction, subendocardial necrosis). Others include toxic encephalopathy, stroke, intravascular hemolysis, disseminated intravascular coagulopathy and thrombocytopenia.^{3,7} These toxic reactions usually occur after multiple stings in excess of 20–200.⁸ The patient presented had stings in excess of 20. Wasp venom contains a complex admixture of biologically active peptides, enzymes, and amines. These include phospholipase A2, hyaluronidase, chemotactic peptides, mast cell– degranulating peptide (peptide 401) histamine, serotonin, catecholamines, mastoparan, kinins, apamine, acetylcholine, antigen 5 and neurotoxic cynines amongst others.^{2,8} Phospholipase A2 (PLA2) is believed to initiate inflammation by triggering the release of arachidonic acid from phospholipids in the cell membrane which leads to the production of inflammatory eicosanoids.⁹ In addition, PLA2 has a

direct cytotoxic effect on striated muscle cells, red blood cells and mast cells.^{3,10} Hyaluronidase in the venom causes breakdown of connective tissues, thereby facilitating the spread of the venom while histamine increases vascular permeability and apamine is neurotoxic.²

Victims of multiple wasp stings usually develop a syndrome rhabdomyolysis, intravascular haemolysis, AKI and hepatic dysfunction.¹¹ This highlighted patient had features of intravascular hemolysis, deranged liver enzymes and also presented with established acute kidney injury. Rhabdomyolysis could not be confirmed in our patient because the serum creatine phosphokinase and urinary myoglobin could not be done due to logistics.

Acute kidney injury is a well-established complication of multiple wasp stings.⁸ Several case series estimated its incidence to be 54–84.5%.¹² It is a potentially life-threatening phenomenon, the mortality could be as high as 25%.¹²

The mechanism of wasp sting-induced AKI is multiple. These include acute tubular necrosis (ATN), pigment nephropathy from rhabdomyolysis (myoglobinuria) and intravascular haemolysis (haemoglobinuria), renal hypoperfusion from hypotension induced by anaphylactic shock, direct nephrotoxicity, acute interstitial nephritis (AIN), and in rare instances acute cortical necrosis and thrombotic microangiopathy.^{1,2,6,7} However, ATN from rhabdomyolysis or intravascular hemolysis appears to be the most frequently encountered mechanism of renal injury. Both myoglobin and haemoglobin are freely filtered by the glomeruli, however when they are present in high concentrations, especially in the setting of volume depletion and renal vasoconstriction, they form ferrihematin precipitates which interact with Tamm-Horsfall protein to form obstructive intratubular pigment casts at the level of the distal tubules, this process is enhanced by acidic urine. In addition, myoglobin exerts direct toxic effect on the proximal tubular epithelial cells.^{2,13} Moreover, both myoglobin and haemoglobin are also potent inhibitors of nitric oxide and can trigger intra-renal vasoconstriction and ischaemia.⁸ For our patient, pigment induced

nephropathy from intravascular hemolysis may be the most likely cause of AKI. The patient passed coke-colored urine, had reticulocytosis with polychromasia on the peripheral blood film examination, unconjugated hyperbilirubinemia, and significant urobilinogen (3+) on the dipstick urinalysis and haematuria.

Acute interstitial nephritis is mediated by a delayed type III hypersensitivity reaction to the venom with immune complex deposition and subsequent activation of the complement system and ensuing interstitial inflammation.¹²

Following multiple wasp stings, timely and prompt intervention is key in preventing AKI. The management strategy include aggressive hydration with intravenous normal saline to prevent hypovolaemia, renal hypoperfusion and to maintain urine output > 100 mls/hr; and urinary alkalinization with sodium bicarbonate to prevent intratubular cast formation.¹⁴ These measures could not be instituted comprehensively in our patient, because he presented late with established AKI and fluid retention. Once overt kidney failure sets in, renal replacement therapy is the treatment of choice.^{8,12} Our patient had five sessions of intermittent hemodialysis which significantly improved the urine output and renal biochemistry. Other interventions that may improve patient outcomes include plasmapheresis and exchange transfusion. They reduce the burden of circulating venom and inflammatory mediators. Steroids may be used to treat allergic interstitial nephritis. Renal biopsy is indicated to diagnose AIN in order to institute early steroid therapy.¹⁴ Other indications for renal biopsy include non-response to supportive measures or delayed renal recovery.¹¹ The highlighted case improved with the institution of renal replacement therapy forestalling the need for a biopsy.

The prognosis is good but it is dependent on the time interval between the stings and intervention at the hospital.^{6,8,12} In the largest case series on wasp-induced AKI by Zhang *et al.*¹² 60 out of the 75 patients studied (80%) survived with complete recovery of the renal function while 7 (9.3%) died, and 8 (10.7%) progressed to chronic kidney disease. In the report, the time to complete renal recovery

ranged from 29 – 41 days.¹² Our patient recovered his full kidney function about 90 days after institution of therapy, this might be explained by the delayed presentation to the hospital, about 3 weeks after the wasp stings, significant envenomation (>20 stings) and the mode of renal replacement therapy of intermittent hemodialysis instead of continuous renal replacement therapy.

CONCLUSION

Acute kidney injury is a well-recognized complication of multiple wasp stings and is commonly due to pigment induced acute tubular necrosis from rhabdomyolysis and hemolysis. There is no specific anti-venom for wasp sting, hence, treatment is largely supportive, such as intensive hydration, alkaline diuresis, and correction of dyselectrolytaemia. Renal replacement therapy is indicated when AKI is unresponsive to conservative treatment.

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