

CASE REPORT

Asian wasp envenomation and acute renal failure: a report of two cases

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ABSTRACT: Acute renal failure is an unusual complication of wasp stings. We report two cases of renal failure after multiple wasp stings (*Vespa affinis*). Both patients had evidence of intravascular haemolysis, hepatic dysfunction, oligo-anuria and azotaemia and required dialysis. The first patient had severe hemolysis, rhabdomyolysis, pigment and venom nephropathy and died on the 8th day in hospital. The second patient, who recovered completely in 3 weeks time with steroid and antihistaminic therapy, had interstitial nephritis. Although acute renal failure after wasp stings is typically caused by acute tubular necrosis (ATN) in the setting of haemolysis or rhabdomyolysis, in some patients, acute renal failure may result from a direct nephrotoxic effect or acute interstitial nephritis from a hypersensitivity reaction.

KEYWORDS: Wasp envenomation, rhabdomyolysis, interstitial nephritis, acute renal failure, Nepal.

INTRODUCTION

In the Pokhara Valley, Nepal, there are many unpublished cases of wasp poisoning which take a heavy death toll annually. Wasp stings usually cause local allergic reactions but can sometimes lead to intravascular haemolysis, rhabdomyolysis, thrombocytopenia, acute tubular necrosis, acute hepatic injury (1) and even myocardial infarction (2) in addition to various respiratory and neurological (3) manifestations.

Death from wasp envenomation is a rare event and results from acute renal failure (ARF) involving various mechanisms. Although ARF after wasp stings is typically caused by ATN in the setting of haemolysis or rhabdomyolysis, in some patients, renal failure may result from a direct nephrotoxicity of wasp venom or acute interstitial nephritis from a hypersensitivity reaction. We here we report two cases of acute renal failure after wasp stings (*Vespa affinis*).

CASE 1

A 55-year-old farmer who had been collecting fodder from a jungle was admitted with dyspnea, hoarse voice and myalgia within 8 hours of being attacked by several wasps. The patient was given intravenous saline, oxygen, salbutamol (β_2 -adrenergic receptor agonist), chlorpheniramine (antihistamine), cyproheptadine (antihistamine), prednisolone (corticosteroid) and ranitidine (histamine H₂-receptor antagonist). He also received fluid, mannitol and furosemide. By the next morning he had haemoptysis and had produced 400 ml of dark urine. On examination, the patient was drowsy, pale, icteric and cyanosed and had approximately one hundred and fifty red and swollen sting marks all over the body. Systemic examination revealed polyphonic wheezes and crepitations at the base of the right lung. Investigations are shown in Table 1. The onset of the oliguric phase was at 12 hours postenvenomation. Chest radiograph showed right basal consolidation. He died on the 8th day following admission despite aggressive therapy with medication, blood transfusions, assisted ventilation, and 16 cycles of dialysis.

CASE 2

A 40-year-old forest guard was attacked by a swarm of wasps. He presented with approximately twenty-five

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sting marks in exposed areas of face, throat, hands and legs [Fig.1]. The patient was treated in the primary health centre with rubbing of saliva and papaya slices over the sting marks and referred to teaching hospital almost 24 hours after being stung. He developed anuria and had not passed any urine the previous night. On examination, the patient had a rapid pulse, unrecordable blood pressure, icterus, an urticarial rash in exposed parts of the face, legs and hands, facial puffiness, and a swollen left ankle and right knee joint. The rest of the physical examination was unremarkable. Investigations are shown in Table 1. He was treated with oxygen, salbutamol, chlorpheniramine, cyproheptadine, prednisolone and ranitidine. His hepatic and renal function improved gradually with fluid challenge, furosemide, mannitol, bicarbonate infusion, dopamine, and 12 cycles of dialysis in 3 weeks' time.

OBSERVATION

DISCUSSION

Wasp stings are well-known causes of toxic and hypersensitivity reactions. Direct toxicity is rare, but has been reported in cases when a very large amount of venom is injected. Immediate hypersensitivity reactions, such as bronchospasm in the first case and an urticarial rash in the second, are known to occur. In our



Figure 1: Case 2: Multiple stings & swollen joints.

Table 1: Pertinent lab results.

	Case 1		Case 2		Normal Range
	1	2	1	2	
Day (since admission)	1	2	1	2	
Blood Count					
Hemoglobin	9.8		11.2		12.5- 14.5g%
WBC	11800		12480		4000-11000 cells/cm ²
Eosinophils			37%		1-5%
Reticulocytes			4%		0.2-2%
Prothrombin Time (sec)	78	20	28	14	12.5
Urinalysis					
Specific Gravity	1.028		1.016		1.002-1.018
Albumin	+		+		nil
WBC	6-8		2-4		1-2/hpf
RBC	10-14		1-2		nil/hpf
Hb	+				nil
Urinary Myoglobin	728		6		0-5 ng/mL
Culture	-		-		sterile
Renal Failure Index		3.00		2.98	
Urinary Na ⁺		50		42	50-250 mEq/L/day
FE Na ⁺		2.65		2.90	

	Case 1		Case 2		Normal Range
	1	2	1	2	
Day (since admission)	1	2	1	2	
Blood Gases					
pH	7.28				7.35-7.45
PaO ₂	88				75-100mmHg
PaCO ₂	38				35-45mmHg
HCO ₃ ⁻	22				22-28mEq/L
General Chemistry					
K ⁺	6.4	7.2	5.6	4.2	3.5-5mEq/L
Ca ²⁺	8.8	8.2	9.6	10.5	8.5-10.5mg%
PO ₄ ²⁻	4.2	4.6	4.2	4.6	3.0-4.5mg%
Urea	112	224	76	55	0-20mg%
Creatinine	5.6	13.9	3.6	2.4	0.5-1.5mg%
Bilirubin Indirect	3.4		2.5		0.4-0.8mg%
Bilirubin Direct	0.6		0.7		0.2-0.4mg%
Creatine Kinase		8400		90	<17-167U/L
LDH		4500		340	240-420 U/L
AST	1260	140	1188	78	0-40U/L
Serum Albumin	1.8		3.2		3.5-5.0g%

report, both reactions responded to steroids and antihistaminics. The second patient had swollen joints indicating serum sickness-like reaction in a sensitized individual.

Wasp venom contains toxic melittin, apamine, phospholipases A1, hyaluronidase, acid phosphatase, histamine, and degranulating peptide mastoparan (4). These components have direct and indirect cytotoxic (hepatic, renal and myocyte membrane), hemolytic, neurotoxic and vasoactive properties, which can cause intravascular haemolysis and rhabdomyolysis (5, 6).

Wasp venom can cause ARF by several mechanisms, which include ATN, acute interstitial nephritis, pigment nephropathy resulting from rhabdomyolysis (myoglobinuria) or intravascular haemolysis (haemoglobinuria) and hypotension caused by an anaphylactic reaction (7, 8).

Previously rhabdomyolysis and renal ischemia were thought to be main causes of nephropathy. Sakhuja et al had postulated that direct toxic injury could be one of the possible mechanisms of ARF following wasp poisoning (9).

Many cases of rhabdomyolysis-associated ARF have been published, but those due to wasp stings are rare. The wasp venom has deleterious effect on renal tubules and glomeruli (albuminuria, haematuria and ARF), red blood cells (haemolysis, reticulocytosis, unconjugated hyperbilirubinaemia), muscles (rhabdomyolysis, elevated creatinine phosphokinase and lactate dehydrogenase, myoglobinuria) and liver (elevated transaminases, hypoalbuminaemia and prolonged prothrombin time) (10). Kularatne et al had described similar multi-organ failure with high mortality following wasp poisoning owing to direct toxic effect (11).

In the first case we presented, the patient had myalgia, indicating muscle injury as evidenced by elevated CPK, LDH and AST and myoglobinuria (728 ng/ml). He also had intravascular haemolysis and haemoglobinuria. Toxic pigments might have caused nephropathy resulting in ARF. The alternative mechanism of ARF postulated was direct nephrotoxicity by massive wasp venom.

Zhang R et al. (12) reported for the first time that acute tubulointerstitial nephritis could lead to ARF in wasp sting cases. In the second case we present, the patient had eosinophiluria, indicating interstitial nephritis. He recovered fully with the mentioned treatment. He did not report taking any medication which might have had nephrotoxic side-effects, and no other causes of ATN could be found. Ultrasound abdomen was unremarkable. Kidney biopsy revealed proximal peritubular necrosis and eosinophilic

infiltration. Hence it can be hypothesized that the ATN was caused by a hypersensitivity reaction to the wasp venom.

CONCLUSION

Wasp stings pose a great environmental hazard in Nepal and early recognition of anaphylactic shock, hepatic or renal dysfunction, rhabdomyolysis or haemolysis and rapid transport to hospital are essential steps of management to avoid fatalities. ARF due to toxic or pigment nephropathy and tubulointerstitial nephritis should be considered in any oliguric and azotemic patient following wasp attack.

COMPETING INTEREST

The authors declare that they have no competing interests.

AUTHOR'S CONTRIBUTION

The second author KG managed the patients with dialysis and conceived the idea for the case report.

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Keka Mukherjee worked with the first author in the medical ward and searched the bibliography for this article. She graduated in December 2006 from Kathmandu University. She is interested in renal diseases.