

Case Report

Wasp sting: An unusual fatal outcome

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ABSTRACT. Wasp stings are not uncommon especially in populations living in proximity of forested areas all over the world. Local manifestations following stings are common and unusually life threatening anaphylaxis may occur, requiring prompt treatment. Multi organ failure and acute renal failure following wasp stings are rare and histological evaluation suggest acute tubular necrosis secondary to hemolysis, rhabdomyolysis and direct venom toxicity. A rare complication of a patient following multiple wasp stings with disseminated intravascular coagulation, acute renal failure and thrombotic microangiopathy is presented.

Keywords: Acute renal failure, Thrombotic microangiopathy, Wasp sting

Introduction

Bees and wasps are associated with painful stings causing intense local reactions. Unusually they may result in anaphylaxis and death, often under reported due to under recognition.¹ Rarely they are associated with fulminant presentations characterized by anaphylaxis, rhabdomyolysis, hemolysis, multiorgan failure and disseminated intravascular coagulation which may occasionally be fatal.^{1,2}

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Case Report

A 22- year old woodcutter was stung by numerous (described by onlookers as 50-100) wasps while at work in a rural semi forested district in Himachal Pradesh, India. He was stung predominantly on the scalp, face, neck, upper limbs and trunk. Swelling of the face, upper arms and trunk was noticed and he did not have any features of anaphylaxis or hypotension. Hydrocortisone and pheniramine maleate were administered at a local hospital and 36 hours later the patient presented to our hospital for further management. He was anuric at presentation and a previous history of wasp bite could not be confirmed.

At presentation the patient was irritable and restless. His face and arms revealed multiple stings marks but only two stings could be recovered from his scalp. Moderate icterus was ob-

Table 1. Investigations along the course of the presentation

Investigation	Admission	Day 5	Day 7	Day 9
Hb (gm/dL)	16.3	7.4	7.5	10.1
TLC (/cu mm)	41500		29300	
Peripheral Blood Film (for schistocytes)	None	None	None	None
Platelet (/cu mm)	90000	29000	99000	90000
Prothrombin Time (Test/control secs)	20.3/12	16/12	15.5/12	14/12
Activated Partial Thromboplastin Time (Test/control secs)	120/30	56/28	29.5/29	39/28
D Dimer	3+			
BT	6 min			
CT	> 15 min			
Blood Urea (mg/dL)	165	165	58	110
Creatinine (mg/dL)	5.6	5.2	2.4	2.8
Aspartate Aminotransferase (5-50 U/L)	7295	3398		510
Alanine aminotransferase (5-50 U/L)	1753	822		290
LDH (230-400 U/L)	3136	20810		11470
CPK (24-195 U/L)	9560	96900		42450
Ig E (1-183 IU/mL)		735.4		
CRP (< 0.6 mg/dL)		9.25		

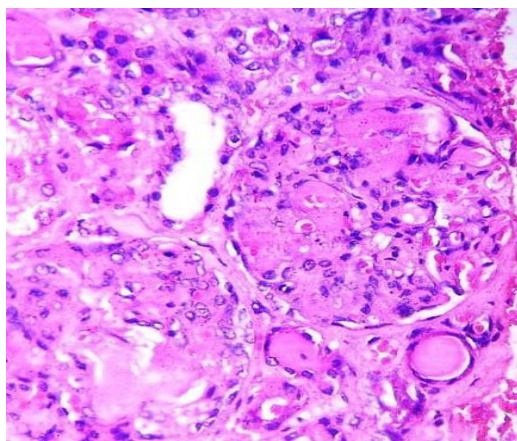


Figure 1. Photomicrograph showing glomerular capillaries plugged with numerous platelet microthrombi (H & E X 400)

served without any pedal edema. The swollen areas on the upper arms were tender but no local erythema or raised temperature was observed. Systemic examination revealed scattered ronchi and hepatomegaly. Hemodialysis was initiated immediately because of severe metabolic acidosis with hyperkalemia, Table 1. The Ig E levels were elevated; however, venom specific Ig E levels and tryptase were not available to confirm the result. The diagnosis included disseminated intravascular coagulation

(DIC) and rhabdomyolysis with acute renal failure (ARF) secondary to wasp envenomation. The possibility of thrombotic microangiopathy was looked for daily in view of persistent thrombocytopenia but no evidence was seen in closely scrutinized peripheral blood films. Plasmapheresis was withheld due to lack of convincing evidence on its utility. Anemia and coagulopathy was treated with blood, fresh frozen plasma, cryoprecipitate and platelet concentrate transfusions.

The patient remained anuric and developed multiorgan failure; including rhabdomyolysis, acute renal failure, acute respiratory distress syndrome and suspected hepatic necrosis and coagulopathy, Table 1. He died on the ninth hospital day. Post mortem biopsy from the liver, lung and spleen suggested hepatic, pulmonary and splenic congestion. Thrombotic microangiopathy with patchy cortical necrosis was seen on the renal biopsy, Figure 1.

Discussion

Hymenoptera insects include Apidae (bees) and Vespidae (wasps and hornets) and are frequently incriminated in insect bites in forested areas. While local reactions limited to pain and swelling are rarely dangerous, systemic aller-

gic reactions may be mild (local involvement), moderate (angioedema, asthma, abdominal pain) or severe (laryngeal edema, hypotension, loss of consciousness).¹

A fulminant multisystem response and multi organ failure is usually observed in an individual previously sensitized to the insect venom.¹

20 years Australian data found wasp sting fatality rate of 0.02% per million population per year, deaths occurring due to anaphylaxis within an hour of being stung.² Swedish Registry of 10 years duration found fatality incidence of 0.2 per million inhabitants and was unrelated to the number of wasp stings.³ While data on such fatalities is unavailable in India, it is likely to increase with encroachment into forested areas and poor garbage disposal. The aggressive, swarm like attack of wasps against intruders, as seen in this case, has been previously described in Himachal Pradesh, India with *Vespa magnifica*.⁴

Acute renal failure is rare with wasp bites and may be directly related to the insect venom causing acute interstitial nephritis or indirectly due to massive hemolysis and rhabdomyolysis induced tubular injury. Renal histopathology usually indicate acute tubular necrosis (ATN).⁵ Coagulopathy in these patients has been related to increased levels of anti thrombin⁶ and decreased levels of fibrinogen, high molecular weight kininogen, factors V and VIII.⁷ Hepatic failure due to centrilobular necrosis, portal triaditis and pericholangitis is seen on histopathology.⁸ On post mortem renal biopsy of our patient revealed thrombotic microangiopathy, rarely described in patients of wasp bite.⁹

The striking similarity of his clinical presentation to snake envenomation, particularly that seen with the Viperidae family, characterized by interaction with blood proteins, platelets, endothelial cells, coagulation cascade and fibrinolytic pathway was most unusual. DIC plays a major role in snake bite and the presence of fibrin thrombi in glomerular capillaries and renal microvasculature leads to microangiopathic hemolytic anemia, thrombocytopenia and cortical necrosis, very similar to our patient. ATN in snake bite is seen to a lesser extent than wasp bites probably due to lesser seve-

rity.¹⁰ Bee stings also unusually cause DIC and the role of mesothelial injury, thrombocyte/macrophage activation, leucotriene/bradykinin/cytokine release, immune complex deposition in small vessels and complement activation is postulated.¹¹ Previous exposure and sensitization to wasp venom could not be ruled out in our patient and was a possible cause of his fulminant presentation. Clinical predictors for anaphylaxis and serious reactions are elusive at the moment.¹²

Early diagnosis and management are crucial and include removal of the stings when possible¹³ and the use of intramuscular adrenaline (0.3 mg) followed by chlorphenaramine maleate and hydrocortisone for severe reactions.¹ Optimal hydration and alkaline diuresis reduces ischemic ATN and pigment induced tubular injury. Rapidly progressive renal failure and anuria warrant early and intensive dialytic support with reports on satisfactory response and recovery.⁵ Plasma exchange has been used with success in isolated case reports.¹⁴ Early presentation to centers with optimal facilities may reduce morbidity and mortality in patients following wasp bites.¹³ Furthermore patients with Hymenoptera allergy should be assessed for immunotherapy and instructed to carry adrenaline and use it in the event following a bee sting.²

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