CASE REPORT

Acute Renal Failure and Posterior Reversible Encephalopathy Syndrome Following Multiple Wasp Stings: A Case Report

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SUMMARY
Wasp stings can present in various ways, ranging from mild self-limiting illness to severe multi organ failure with a potentially fatal outcome. We report a case of multiple wasp stings leading to acute renal failure needing prolonged dialysis support and posterior reversible encephalopathy syndrome.

KEY WORDS:
Wasp stings, Acute renal failure, Posterior reversible encephalopathy syndrome

INTRODUCTION
Wasp stings, a case report

Wasp stings, Acute renal failure, Posterior reversible encephalopathy syndrome

CASE REPORT

A 29-year old lady, previously healthy, was stung by multiple wasps. She presented to us 6 hours post event complaining of headache, giddiness and pain at the sites of sting. There was no pruritis, edema, rash, shortness of breath or nausea and vomiting. On examination, she was alert and had generalized sting marks over her limbs and body. She was afebrile. BP 143/76mmHg, heart rate 88/minute, with oxygen saturation of 97% on room air. There was no abnormality on cardiovascular, respiratory and abdominal examination.

Renal profile on presentation showed Sodium 138mmol/L, Potassium 3.0mmol/L, Chloride 102mmol/L, Urea 3.8mmol/L, Creatinine 77µmol/L. Haemoglobin was 11.9g/dL, white cell count (WCC) 14,100/µL, platelet counts 415,000/µL. 12-lead electrocardiogram showed sinus tachycardia and chest radiograph was normal. She was given intravenous hydrocortisone and chlorpheniramine for prevention of anaphylaxis and tramadol for pain.

She became anuric after admission. Renal profile 20 hours post event showed Sodium 128mmol/L, Potassium 4.4mmol/L, Chloride 101mmol/L, Urea 16.7mmol/L, Creatinine 256 µmol/L. She also developed metabolic acidosis with bicarbonate level of 15.1 mmol/L, base excess -12.7 mmol/L, pH 7.26, and pCO2 of 29mmHg. A repeat full blood count showed a drastic drop in haemoglobin to 7.7g/dL, WCC 24,800/µL, and platelet count 292,000/µL. There was no evidence of active bleeding. LDH was 7748 U/L, bilirubin level 98 µmol/L (mixed direct and indirect bilirubinemia). Her liver enzymes were markedly raised with AST 2496 and ALT 708. Her CK was 62,784 U/L, and urine was positive for myoglobin, red blood cells 120/µL, but negative for casts.

She required intubation 28 hours post event for worsening facial edema with occasional stridors, as well as worsening gas exchange. A repeat chest radiograph showed increased vascular markings. She remained anuric despite intravenous fluids and intravenous frusemide boluses. Hemodialysis was started 31 hours post event. This was subsequently converted to CVVH for 4 days in view of the presence of rhabdomyolysis and hemolysis. She remained dialysis-dependant for a total of 6 weeks throughout her 7 weeks stay.

Her CKP level reduced gradually to 10,483 U/L 4 days post admission. Unfortunately, no repeat LDH level was available, but her bilirubin levels and liver enzymes normalized around 2 weeks post event. She required packed cell transfusion initially but her hemoglobin level subsequently remained stable.

About a month after the primary event, when she was showing signs of recovery, her platelet counts dropped from 145,000/µL to 84,000/µL. Coagulation profile was normal. Her haemoglobin level also dropped >2g/dL within 2 days. There was no documented fever, evidence of sepsis or bleeding but new spontaneous bruises were noted over her limbs. At the same time, her BP rose to 160-190 systolic and 88-104 diastolic, requiring increasing doses of anti hypertensive. She had an episode of witnessed loss of consciousness with urinary incontinence. No jerking movements were observed and there was no documented visual abnormality. Blood sugar during that time and fundoscopy were normal. An urgent CT brain showed symmetrical bilateral white matter hypodensities at the posterior circulation which was suggestive of posterior reversible encephalopathy syndrome (PRES) (fig 1). On the same afternoon, her BP normalized and her symptoms resolved spontaneously. Opinion was sought from a neurologist thereafter, who felt that the clinical and
radiological features of this patient, together with the rapid recovery were consistent with a diagnosis of PRES.

Her urine output increased on day 23 of admission and HD was stopped 6 weeks after the primary event. Patient made a good recovery and creatinine on discharge was 319 µmol/L. Her renal function continued to improve on follow up, creatinine level reduced to 117 µmol/L 15 weeks after the primary event and her BP also normalized and all antihypertensives were stopped. She was last seen 31 weeks post event and her creatinine level was 79 µmol/L.

**DISCUSSION**

Wasp venom contains 13 different types of antigen, which are responsible for causing local reaction, and subsequently, through cellular and humoral response, causes systemic reaction.

There have been few reported cases of anaphylaxis after wasp stings which presented with facial edema, wheezes or stridor, and hypotension. Our patient had occasional stridor and worsening facial edema before intubation. These symptoms are probably due to hypersensitivity.

Acute renal failure (ARF) is rarely reported as one of the complications following multiple wasp stings. The exact cause for the development of acute renal failure in only some individuals is still poorly understood; however it can be associated with acute tubular necrosis (ATN) due to the toxin, rhabdomyolysis, hemolysis or disseminated intravascular coagulopathy.

For our patient, no renal biopsy was done to determine the exact cause of her acute renal failure. There was no documented hypotension throughout the admission. She had features of rhabdomyolysis and hemolysis. These, in addition to direct toxin effect causing ATN are the most likely causes of ARF in her.

To our knowledge, there have been no reported cases, so far, of associations between developments of PRES with wasp stings. Our patient had a few episodes of high BP (160-190/88-104 mmHg) which might have contributed to PRES. In view of her low platelets, bruising, a drop in haemoglobin levels, neurological involvement and spontaneous recovery, this raises the possibility of a diagnosis of thrombotic thrombocytopenic purpura (TTP). Peripheral blood film was not sent. However, her rapid spontaneous recovery within the same day makes the diagnosis of TTP unlikely.

There have been reported associations between TTP following multiple wasp stings. Jennifer R Ashley et al in South Med J. 2003; 96(6) described a case of a 40-year old woman who, following honeybee sting, developed fever, headache, microangiopathic haemolytic anemia, thrombocytopenia and was diagnosed as having TTP. However, the time of development was within 1 week of bee sting, but for our patient, the onset of symptoms suggestive of TTP was 1 month after the stings. Moreover, our patient did not have fever during that period.

Diagnosis of PRES is based on a combination of appropriate clinical features and imaging appearance. These clinical features include headaches, altered mental state, seizures, visual disturbances and in the majority (but not all) of cases, reasonably rapid reversibility upon treatment of the cause. It is often caused by or associated with uncontrolled hypertension. The imaging features are that of symmetric changes of vasogenic oedema predominantly in the posterior (ie. parieto-occipital) subcortical regions of the brain. Our patient had clinical features (altered mentation, a possible seizure, hypertension and rapid reversibility) and radiological features consistent with a diagnosis of PRES. The only other neurological syndrome that could potentially fit would be Acute Acute Disseminated Encephalomyelitis (ADEM), but this is less likely because of the rapid reversibility (ADEM usually takes longer to resolve) and that the CT appearance would be less symmetrical in ADEM.
In summary, prompt recognition of the possible complications following wasp stings is essential in preventing fatal outcomes. Rhabdomyolysis and hemolysis can occur resulting in renal failure. Adequate hydration is necessary with close monitoring of the renal function. Prolonged dialysis support may be needed but good renal outcome can be expected in those who survive the acute event.

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REFERENCES


ABSTRACT

Why Coronary Artery Bypass Surgery is still The Optimal Treatment Strategy for Left Main Stem Disease: An Evidence-Based Review with A Malaysian Surgical Perspective

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Left main stem (LMS) coronary artery disease (CAD) remains an important risk factor for increased mortality and morbidity at all stages of diagnosis and treatment of coronary artery disease.

Significant flow limiting stenosis usually results in low-tolerance angina and has prognostic implications. Historically, coronary artery bypass grafting (CABG) has been the treatment of choice for LMS revascularisation, but advances in percutaneous coronary intervention (PCI) have challenged this surgery-only paradigm. This article is a surgical appraisal of the current evidence regarding the optimal revascularisation strategy for LMS disease in terms of safety, efficacy and durability.

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