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CASE REPORT

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A Call for sting treatment protocol: Case report of a 3 year old with massive bee sting resulting in acute kidney injury

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Abstract*:* Acute Kidney Injury in children following bee sting envenomation is rare and survival is hinged on early recognition and prompt appropriate management. This report is aimed at raising awareness among healthcare workers, of one of the systemic effects of massive bee sting and the need to develop sting treatment protocol. A three year old preschooler was attacked by a swarm of bees, receiving over 150stings. Initial clinical features were allergic response involving the head, face and tongue for which he had first aid in a primary healthcare facility and thereafter sent home. Few hours later he developed clinical features of intravascular haemolysis, rhabdomyolysis, hypertension and acute renal failure. Laboratory findings were in keeping with clinical presentation. Despite supportive management, serum biochemistry worsened necessitating haemodialysis. Massive bee sting envenomation can cause systemic reactions and organ dysfunctions. To improve the overall clinical outcome, sting treatment protocol is advocated.

Key Words: Bee sting envenomation, acute kidney injury, sting treatment protocol, rhabdomyolysis, myoglobinuria, haemodialysis.

Introduction

Very little is known about massive envenomation by hymenoptera (bee), especially among children. Hymenoptera envenomation may result in systemic damage but with early commencement of standard care, chances of survival are increased. However in the absence of treatment protocol, appropriate management and survival is unlikely. There is a need to raise awareness among healthcare providers on the need to develop bee-sting treatment protocol that can be initiated rapidly at all levels of health care service. We report a case of a three year old boy who developed acute kidney injury following massive bee sting envenomation.

Case

A three year old boy presented to the Emergency Paediatric Unit (EPU) of our facility 22 hours after attack by a swarm of bees along a bush path. The bees covered his head and face, with some entering his mouth. He was rescued by a passerby 30minutes later and immediately taken to a primary health care centre (PHC) where over 150 stingers were immediately removed. The affected sites were washed with antiseptics and oral analgesics, antacids and antibiotics prescribed, and then sent home.
Eight hours later he developed swelling of the head and face and for 22 hours post the incident, he did not pass urine. There was no history of jaundice, difficulty in breathing, vomiting, diarrhoea, or loss of consciousness. He had no prior history of hospital admissions and no family history suggestive of atopy, kidney disease, or hypertension.

On admission, he was in painful distress and there was massive oedema involving the scalp, tongue and face with inability to open both eyes. There were 155 stings marks with tender ecchymotic lesions on the affected areas. He was not in respiratory distress, not jaundiced but mildly pale. He had sinus tachycardia (pulse rate 100/min), systolic hypertension (BP- 100/60 mmHg) and tachypnoea of 40cycles/min. There were no palpably enlarged organs. Anthropometry was within normal limits. While on admission he had one episode of generalized tonic clonic convulsion, controlled with paraldehyde. He also had an episode of epistaxis with estimated blood loss of 100mls. The scalp lesions ulcerated and he developed a fever but the swollen head, face and tongue gradually reduced in size.

Initial laboratory work up revealed: packed cell volume (PCV) 33%, total leukocyte count 8.3 $x10^9$/l with left shift. Platelets were initially normal. There was increased prothrombin time (PT) and Kaolin Cephalin Clotting Time (KCCT): - 17 and 38 seconds respectively (control: 14 and 35 seconds). The initial serum biochemistry showed deranged serum urea and serum creatinine (Table 1). Urinalysis revealed proteinuria and haema-

turia. Ultrasonography showed normal kidneys and pelvi-calyceal system. Clinically, he was assessed to have a systemic inflammatory reaction to bee sting venom-rhabdomyolysis and acute kidney injury.
Resuscitation at the EPU included challenging the kidneys with intravenous fluid and thereafter he passed 30mls of coca-coloured urine (maintenance fluid intake was based on daily requirement); intravenous hydrocortisone, antihistamine, tetanus toxoid (intramuscular), analgesic and antibiotics. Nasogastric tube was inserted for feeding and urethral catheter for continuous urinary output monitoring.

Despite adequate diuresis and improving blood pressure, serum biochemistry worsened with rapidly increasing levels of serum urea, creatinine and worsening acidosis (Table 1); PCV dropped to 20.1% and platelet count dropped from 249 x 10^9/L to 59 x 10^9/L. At admission, he had an estimated glomerular filtration rate (GFR) of 25.3 ml/min/1.73m^2 which worsened over days to 11.1ml/min/1.73m^2. He had haemodialysis with intradialysis blood transfusion and subsequent improvement in serum biochemistry (Table 1) and general condition. However, despite counselling, the parents declined continuation of management due to financial constraints and discharged against medical advice.

Table 1 shows worsening serum biochemistry (days 1, 5 and 10). Values seen on the day 12 are post haemodialysis

Table 1: Serum biochemistry results for days 1, 5, 10 and 12

Parameters	Day 1	Day 5	Day 10	Day 12
Urea (mmol/l)	27.1	6.5	43.8	40.0
Sodium(mmol/l)	137.0	120.0	131.0	133.0
Potassium(mmol/l)	4.8	2.9	2.9	4.3
Chloride(mmol/l)	100.0	83.0	94.0	93.0
Bicarbonate(mmol/l)	22.0	15.0	11.0	16.0
Creatinine (µmol/l)	165.0	353.0	377.0	236.0

Discussion

This case demonstrates that bee envenomation can cause severe systemic effects other than the commonly reported anaphylaxis. Bees, unlike wasps, leave stings behind which release large amounts of venom, 50–140 mcg/sting[1] leading to a spectrum of reactions from mild local allergic reactions to large local reactions and in some cases systemic toxic reaction.[1] Systemic toxic reactions, such as acute kidney injury, hepatic damage, neurological deficit, disseminated intravascular coagulopathy, hypotension, bronchoconstriction and even death, are seen in cases of massive bee envenomation.[1]

The clinical presentation of this patient can be explained by the effect of the biochemical components of bee venom [2, 3] The peptides and phospholipids contained in the venom actively cause destruction of red blood cells, release of histamine and other components by mast cells, causing pain and provoking haemolysis and rhabdomyolysis. The sudden rise of our patient's serum creatinine suggesting rhabdomyolysis and fall in haemoglobin with haemoglobinuria can be explained by this mechanism of bee sting envenomation. An assay of the creatine phosphokinase would have further reinforced the evidence, however financial constraints prevented that. Serial blood chemistry studies and serum levels of haemoglobin and myoglobin performed over a period of hours to days will reveal whether toxic venom effects have occurred. The renal damage, as seen in this report, could be as a result of either direct nephrotoxicity due to toxin,[4] hypotension leading to ischemic tubular necrosis[5] or nephropathy due to haemoglobinuria and myoglobinuria.[6] Direct toxin mediated cellular damage in massive envenomation has also been found to cause disseminated intravascular coagulopathy, cardiovascular abnormalities,[7] hepatic damage[8] and neurological deficits of different degrees.[9,10]

The management of massive bee envenomation is mainly supportive with no specific anti-venom being available[1,3]. Rapid removal of stingers is advocated as 90% of the venom sac contents are delivered in 20 seconds with delivery completed in one minute[11]. Delay in removal leads to increased weal size and increase in envenomation.[12] This was the case in our patient as the stingers were removed almost an hour after the sting. The cumulative dose of the multiple stings account for the morbidity and mortality.[2]

Early treatment with steroids and antihistamines, copious saline hydration for intravenous volume replacement and sodium bicarbonate for urine alkalization is advised.[3,4,13,14,15] Treatment of acute kidney injury following bee stings with haemodialysis, hemofiltration or peritoneal dialysis has been reported,[13,14] although exchange blood transfusion or plasmapharesis is equally effective because it directly reduces the circulating mediators of inflammation caused by the venom.[15] In this patient, supportive measures and haemodialysis, in addition to aggressive hydration and intensive wound care formed the basis for therapy. Patients who develop respiratory arrest require ventilator support while vasopressors can be used to provide vascular support. The patient's renal function started improving after commencing haemodialysis but further monitoring could not be done because parents requested discontinuation of management on account of financial constraints.

Literature search shows as few as 30-50 stings in children carries grave prognosis while very sensitive individuals may develop fatal anaphylaxis even after just one bite.[3,16] Our patient had over 150 stings and survived. The time between the accident and medical treatment, and the prompt removal of stingers seem to be important in determining our patient's prognosis.[3, 4,15, 17] Prognosis is also improved when systemic toxic effect is anticipated, with anticipatory management instituted.

Conclusion

In conclusion, multiple hymenoptera envenomation is not an innocuous condition, yet manifestation can be delayed and can be grave resulting in deadly consequences. Survival is hinged on early commencement of appropriate management. There is therefore a need to develop sting treatment protocols and increase public education. Meanwhile, the need for anticipatory management cannot be overemphasized.

Authors' contribution
All the authors participated in the conception, literature search, preparation, reviewing and writing of the manuscript.
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