CASE REPORT

Acute kidney injury after massive attack of Africanised bees

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Accepted 18 February 2014

SUMMARY

Acute kidney injury (AKI) is a well-documented complication of massive attack by Africanised bees and can be observed 48–72 h after the accident. We report a case of Africanised bees attack followed by severe and lethal AKI. A 56-year-old man was admitted to emergency department after a massive attack of Africanised bees (>1000 bee stings). He was unconscious, presenting with hypotension and tachycardia. Mechanical ventilation, volume expansion and care for anaphylaxis were instituted. The patient was transferred to the intensive care unit (ICU) and after 48 h he developed rhabdomyolysis, oliguria, increased creatinine levels, hyperkalaemia and refractory acidosis. A diagnosis of AKI secondary to rhabdomyolysis and shock was made. The patient was treated with a prolonged course of haemodialysis. However, he progressed to refractory shock and died 5 days after admission.

BACKGROUND

Africanised bees (*Apis mellifera scutellata*) are an extremely aggressive bee species, which have the habit of performing massive attacks to victims.^{1–9} Acute kidney injury (AKI) is a well-documented complication of these attacks and can occur when there are more 500 bee stings and is observed 48–72 h after the accident. There are nearly 45 cases reported in the literature.^{1–5} The pathophysiology of renal failure is related to direct nephrotoxicity from bee venom, and this includes rhabdomyolysis, haemolysis and renal ischaemia by anaphylaxis, hypovolaemia or low cardiac output.^{8,9}

We report the case of a patient who developed AKI after a massive attack of Africanised bees (about 1000 bee stings), progressing to severe rhabdomyolysis.

CASE PRESENTATION

A 56-year-old man was admitted to emergency department after a massive attack of Africanised bees (>1000 bee stings) in a rural area in Sao Paulo, Brazil. His medical history included hypertension and alcoholism. On admission, the patient was unconscious, hypoxic (PaO₂=71 mm Hg and SatO₂=83%), hypotensive (90×60 mm Hg) and tachycardic (126 rpm). Widespread erythematous and papules were found on his body.

INVESTIGATIONS

On admission, laboratory measurements revealed normal or little altered parameters: creatine kinase (CK) 790 U/L (30–135 U/L), serum creatine (Cr)

0.9 mg/dL (0.9–1.4 mg/dL), blood urea nitrogen (BUN) 32 mg/dL (35–51 mg/dL), potassium 3.5 mmol/L (3.6–5.0 mmol/L), sodium 147 mmol/L (137–145 mmol/L), calcium 7.5 mg/dL (8.4–10.2 mg/dL), phosphorus 4.6 mg/dL (2.5–4.5 mg/dL), aminotransferase (AST) 216 U/L (9–52 U/L), lactate dehydrogenase (LDH) 624 U/L (50–245 U/L), uric acid (UA) 541 mmol/L (90–420 mmol/L), total bilirubin 1.1 mg/dL (0.2–1.3 mg/dL), glucose 96.0 mg/dL (<100 mg/dL) and C reactive protein (CRP) 0.6 mg/dL (<1.0). Haematological tests showed haemoglobin as 12.4 g% (12–16 g%), and white cell count was 9000/mm³. Urinalysis showed pH 5.0, density 1035, protein 1+, and haematuria was negative. An X-ray of the chest did not show any evidence of acute respiratory distress syndrome.

TREATMENT

On admission, the patient was treated with intravenous fluid (after 3 L of crystalloids blood pressure was 140×90 mm Hg), epinephrine 1 mg intravenous and glucocorticosteroids. We proceeded with tracheal intubation due to unconsciousness and bladder catheterisation (urine output was 100 mL) and the patient was transferred to intensive care unit.

OUTCOME AND FOLLOW-UP

After 48 h, the patient developed shock, severe rhabdomyolysis, oliguria, increased creatinine levels, hyperkalaemia and refractory acidosis. Urinalysis showed pH 5.0, density 1020, haeme pigment 2+, haematuria (2+), without pyuria and protein 1+. Fraction excretion of sodium (Na) and urinary Na were high (1.9% and 44 mEq/L, respectively). Intravenous fluids were started, as well as sodium bicarbonate and vasoactive drugs (norepinephrine) were provided for urinary alkalinisation. The patient required 0.9 µg/kg/min to maintain mean blood pressure of 71 mm Hg. On the same day, forced diuresis with furosemide (60 mg/day) was introduced. However, he did not show any improvement in renal function and he was initiated on haemodialysis (72 h after admission). The patient was treated with three prolonged haemodialysis sessions (10 h); however, he progressed to refractory shock and died 5 days after admission. Table 1 shows laboratory and clinical results during follow-up.

Creatine (0.9–1.4 mg/dL, creatine kinase (CK) (30–135 U/L), blood urea nitrogen (BUN) (35–51 mg/dL), potassium (K) (3.5–5.1 mmol/L), lactate dehydrogenase (LDH) (50–245 U/L), aspartate aminotransferase (AST) (9–52 U/L), sodium (Na) (135–145 mmol/L), phosphorus (P) (2.5–4.5 mg/dL),

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To cite: Bridi RA, Balbi AL, Neves PM, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-201381

Table 1 Main clinical parameters and laboratory results on admission and during follow-up

	Time since admission						
	Admission	12 h	24 h	48 h	72 h*	96 h	120 h
Urine output 24 h (mL)	100		900	1900	400		200
Fluid balance (mL)	(+) 5000		(+) 3524	(+) 4043	(+) 900		1
Laboratory examinations							
Creatine (mg/dL)	0.9		1.3	2.7	4.2	6.5	6.9
Creatine kCK	790		1458	7600	11 400	14 000	6800
BUN (mg/dL)	21	42	92	111	122	128	131
K (mmol/L)	3.5	4.7	4.8	5.4	8.0	5.3	4.5
LDH (U/L)	624	680	1863	3639	4815	4311	
AST (U/L)	216	359	9	489	559	419	133
Na (mmol/L)	147	145	138	139	137	137	146
	4.6			4.9	6.2		7.3
CaT (mg/dL)	7.5	8.8	9.6		8.9	8.9	8.9
CRP	0.6	4	5	25.3	49.2	49.2	50.3

*Haemodialysis was started.

AST, aminotransferase; BUN, blood urea nitrogen; CaT, total serum calcium; CRP, C reactive protein; K, potassium; LDH, lactate dehydrogenase; NA, sodium.

total serum calcium (CaT) (8.4–10.2 mmol/L), magnesium (Mg) (1.5–2.5 mmol/L) and C reactive protein (CRP) (<0.5 mg/dL).

DISCUSSION

By the year 1956, the European bees (*Apis mellifera mellifera* and *Apis mellifera ligustica*) were the only species of bees found in Brazil. In that year, African bees (*Apis mellifera scutellata*), which have aggressive behaviour and higher honey production, were brought into Brazil. An escape of their queens following an accident in 1957 led to the mixture of these bees with European bees. This has resulted in the formation of more aggressive Africanised bees, which are extremely aggressive and have the habit of performing massive attacks to victims.^{1–4} Since then, nearly 45 cases of accidents have been reported in the literature.^{1–7} Africanised bee attack can result in a simple local inflammatory reaction to anaphylactic shock.

The pathophysiology of AKI secondary to stings by Africanised bees is a complex process. There may be a combination of direct nephrotoxicity, rhabdomyolysis with subsequent myoglobinuria and hypovolaemia following anaphylaxis or low-cardiac output.^{8–9}

The bee venom contains peptides such as melittin and phospholipase A which can provoke rhabdomyolysis and haemolysis. They have cytolytic effects that act on the phospholipid membrane of blood cells, muscles and the vascular endothelium, producing proinflammatory substances and hepatocellular lesions.^{1–4}

Our patient developed severe rhabdomyolysis and this diagnosis was confirmed by laboratory examinations. Rhabdomyolysis is a condition in which damaged skeletal muscle tissue disintegrates rapidly, and products of damaged muscle cells such as myoglobin, LDH, AST and ALT are released into the bloodstream.⁹ CK levels are the most sensitive indicator of myocyte injury in rhabdomyolysis. CK rises within 12 h of the onset of muscle injury, peaks in 1–3 days and declines 3–5 days after the cessation of muscle injury. CK levels of 5000 U/L or greater can be associated with AKI.² The patient in this case reached CK levels of 14 000 U/L.

The treatment of massive attack of Africanised bees consists of general measures for anaphylaxis such as antihistamines, corticosteroids and non-opioid analgesics. There is

controversy about whether the stings should be removed.^{9–11} It is estimated that about two-third of the venom remain deposited on the stings. For this reason, some authors affirm that we should remove the stings to avoid further venom injection.^{1–2–11} There are no differences between the methods used to remove the stings ('shaving or extracting'). However, one other article affirms that there is no evidence for removing the stings, since most of the venom is injected into the victim within 20 s of a sting.¹⁰ In our patient's case, stings were removed by shaving.

The management of rhabdomyolysis includes life support and measures to preserve renal function. Previous studies have shown that early aggressive fluid replacement, use of bicarbonate and forced diuresis can be beneficial in reducing the occurrence of AKI.^{1–5} Bicarbonate minimises intratubular haeme pigment deposition and behaves as a free radical scavenger, leading to decreased cell injury.^{5–9} This patient received vigorous hydration immediately, with bicarbonate and forced diuresis on the day after admission; however, he did not show any improvement in renal function and developed oliguria, refractory metabolic acidosis and hyperkalaemia, and dialysis was indicated.

In a recent review, Mejia-Velez *et al*⁷ evaluated 43 cases of AKI secondary to stings of Africanised bees. The authors observed that the need for dialysis occurred in 86% of patients and haemodialysis and peritoneal dialysis showed similar efficacy.^{2–4–7–9}

In experimental studies, the estimated median lethal dose of venom was equivalent to 19 stings per kilogram of weight.⁸ Our patient weighed 60 kg and suffered about 1000 bites, totalling 16.6 bites per pound of weight, near lethal dose. There are reports that, in adults, a rate greater than 500 bites would be related to a worse prognosis.^{2–3–8–9}

Massive attack of Africanised bees should be considered as a medical emergency, and the medical community should become conscious of the need for rapid referral of these victims to high-complexity health centres since severe AKI can be part of the natural history of this accident. Measures for prevention of AKI should be initiated immediately and when severe AKI is established, dialysis treatment must be indicated in order to prevent complications and reduce mortality.^{10–11}

Learning points

- ▶ Accidents involving massive attack of Africanised bees should be considered as a medical emergency.
- ▶ Acute kidney injury (AKI) is a well-documented complication of massive attack of Africanised bees and can be observed 48–72 h after the accident.
- ▶ The pathophysiology of AKI secondary to stings by Africanised bees is a complex process. There may be a combination of direct nephrotoxicity, rhabdomyolysis and renal ischaemia by anaphylaxis, hypovolaemia or low cardiac output.
- ▶ If AKI becomes refractory to clinical measures, dialysis treatment must be started in order to prevent complications and reduce mortality.

Contributors PMN and RAB acquired the data and drafted the article. ALB and DP revised the manuscript for important intellectual content.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Almeida RAMB, Olivo TET, Mendes RP, *et al.* Africanized honeybee stings: how to treat them? *Rev Soc Bras Med Trop* 2011;44:755–61.
- 2 Daher EF, Oliveira RA, Silva LSV, *et al.* Insuficiência renal aguda por picada de abelhas: relato de casos. *Rev Soc Bras Med Trop* 2009;42:209–12.
- 3 Bresolin NL, Carvalho FLC, Goes JE, *et al.* Acute renal failure following of massive attack by Africanized bee stings. *Pediatr Nephrol* 2002;17:625–7.
- 4 Diaz-Sanches CL, Lifshitz-Guinzberg A, Ignacio-Ibarra G, *et al.* Survival after massive (>2000) Africanized honeybee stings. *Arch Intern Med* 1998;158:925–7.
- 5 Gabriel DP, Barsante RC, Silva VS, *et al.* Severe acute renal failure after massive attack of Africanized bees. *Nephrol Dial Transplant* 2004;19:2680.
- 6 Silva GAR, Pires KL, Soares DCS, *et al.* RRH: Envenoming syndrome due to 200 stings from Africanized honeybees. *Rev Inst Med Trop Sao Paulo* 2013;55:61–4.
- 7 Mejia-Velez G. Acute renal failure due to multiple stings by Africanized bees. Report on 43 cases. *Nefrologia* 2010;30:531–8.
- 8 Quan D. North American poisonous bites and stings. *Crit Care Clin* 2012;28:633–59.
- 9 Grisoto LSD, Mendes GE, Castro I, *et al.* Mechanisms of bee venom-induced acute renal failure. *Toxicon* 2006;48:44–54.
- 10 Vetter RS, Visscher PK, Camazine S. Mass envenomation by honey bees and wasps. *West J Med* 1999;170:223–7.
- 11 Abdulkader RC, Bárbaro KC, Barros EJ, *et al.* Nephrotoxicity of insect and spider venom in Latin America. *Semin Nephrol* 2008;28:373–82.

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