# Acute renal failure due to multiple bee stings – case reports

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# Summary

This report deals with a father and his son who developed acute renal failure following multiple bee stings. The renal lesion in these patients appears to be due to rhabdomyolysis caused by the bee venom. The other mechanisms are also discussed. The need for clinicians to be aware of acute renal failure as a complication of bee stings is stressed.

Key words: Bee venom, toxicity, hypersensitivity, rhabdomyolysis, kidney failure, acute.

### Introduction

Bee stings are common in the tropics. The anaphylactic reaction due to bee stings and its management has been well described earlier. <sup>1-3</sup> But little has been mentioned about acute renal failure as a complication of bee stings. <sup>4,5</sup> We describe a father and his son who developed acute renal failure following multiple bee stings. Both needed peritoneal dialysis to tide over the acute crisis.

## Case Reports

A 70-year old man and his 40-year old son were attacked by a swarm of bees while working in their rubber-plantation. Both suffered dozens of stings over the face, neck, upper torso and upper limbs. The son was stung substantially worse for having heroically shielded his father.

On admission, both were in pain, with oedema and erythema over the stung areas, from where many sting-apparatus were carefully removed intact. Vital signs were unremarkable and at no time was either of them hypotensive. Urine outputs over the first twelve hours were satisfactory. Both then produced dark brown urine which did not contain red blood cells or haemoglobin. Their muscle aches and tenderness with raised creatinine phosphokinase (CPK) levels suggested rhabdomyolysis with myglobinuria.

After the first 12 hours, urine output progressively fell, accompanied by rising blood urea. The classical sequence of acute renal failure with oliguria followed by a polyuric phase before normalisation was observed in both patients who were successfully treated with temporary peritoneal dialysis. Subsequent follow-up up to five months after the stings demonstrated normal renal function. Table 1 details some features of the clinical course in both patients.

Table 1 Clinical course

Characteristic (upper limit of normal values and units in brackets where applicable).			Father	Son
1.	Admission blood urea	(7.8 nmol/l)	13.0	11.7
2.	Peak blood urea	(7.8 nmol/l)	61.0	57.2
3.	Serum creatinine	(176 micromole/L)	253	644
4.	Creatinine phosphokinase	(195 units/l)	6459	3359
5.	Average hourly urine output in 1st. 12 hours	(ml/hour)	100	51
6.	Lowest urine output in oliguric phase before dialysis	(ml/day)	200	160
7.	Peak urine output in polyuric phase after dialysis	(ml/day)	4200	5250
8.	Interval between admission and maximal diuresis	(days)	19	15
9.	Total time of temporary peritoneal dialysis	(hours)	147	94
10.	Blood urea at follow-up at five months	(7.8. nmol/l)	6.8	5.2

### Discussion

The exact mechanism of acute renal failure due to bee stings is not known. The venom contains several active substances including mellitin which has both haemolytic and vasoactive properties. Thus it appears that the venom induces rhabdomyolysis leading to acute renal failure in these patients. The markedly raised creatinine phosphokinase levels in our patients support this hypothesis. Moreover our patients were never hypotensive at any stage which virtually ruled out renal ischemia as the cause of their problems.

In a hypersensitive patient a single sting may cause death from anaphylactic shock or may cause other non-fatal complications, including serum sickness-like reaction, delayed skin eruptions, haematuria and short-lived proteinuria.<sup>3</sup> It is not certain whether acute renal failure in bee stings is entirely dose dependent, as shown in the case of a woman who only had minor symptoms of anaphylaxis without renal impairment when she was stung by 65 bees.<sup>6</sup>

Whatever the pathophysiology it is necessary for clinicians to be aware of the possibility of acute renal failure as a complication of bee stings. Very often, these patients are admitted for observation. A simple check on their urine output, proteinuria and renal profile may go a long way in the early detection and management of this otherwise fatal complication.

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