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Letter to the Editor

Concurrent acute coronary syndrome and ischemic stroke following multiple bee stings

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Local and systemic reactions after bee stings are common, but there are few reports of severe complications, such as acute coronary syndrome, acute renal failure and stroke [1].

A 64-year-old man was stung by approximately 200 bees on his face, neck, chest and upper extremities. The stingers were removed in the emergency department and he was treated with promethazine 25 mg i.v., methylprednisolone 125 mg i.v., meperidine 25 mg i.v., pantoprazole 40 mg i.v., ondansetron 8 mg i.v. He had itching but no other symptoms. Sixteen hours later he developed left side weakness and substernal chest pain. He was treated with t-PA (0.9 mg/kg) 2 h after the onset of his neurological symptoms and was transferred to our hospital. He had a left cranial nerve seven palsy, left hemiparesis, loss of pain and proprioception on the left, hyperreflexia, and a positive Babinski's sign on the left side.

Complete blood counts showed decreased RBCs, hemoglobin, hematocrit and platelet count on admission which returned to normal on hospital day two. A comprehensive metabolic panel CMP was normal except for an elevated AST which returned to normal on hospital day four. His urinalysis showed hematuria and proteinuria. Brain magnetic resonance imaging showed a large right middle cerebral artery ischemic stroke and 2–3 mm midline shift to the left side (Fig. 1). Cardiac enzymes were increased on admission; CK was 1,496 IU/L (26–308 IU/L), CKMB was 29.2 ng/mL (0.1–4.9 ng/mL), and troponin I was 1.48 ng/mL (0.01–0.03 ng/mL). ECG showed normal sinus rhythm and no ST segment or T wave changes. His transthoracic echocardiogram revealed no regional wall motion abnormalities. Left ventricular systolic function was normal, and mild diastolic dysfunction was present. Carotid Doppler ultrasound did not identify any stenosis. Follow-up ECGs were unchanged; cardiac enzymes decreased to within normal limits over a week. His initial NIH stroke score scale was 14. Twenty-four hours after the onset of his neurological symptoms the NIH stroke scale score was four; it was zero after seven days. Aspirin and simvastatin were started, and the patient was transferred to a rehabilitation facility.

To review the literature, we searched the PubMed database in English using the MeSH terms Bees or Bee venoms combined with Stroke; or Kidney failure, acute; or Acute coronary syndrome; or Coronary artery disease; or Arrhythmias, cardiac.

Reactions following bee stings can cause significant morbidity and mortality; the mechanism for injury includes both toxic effects and severe allergic reactions (Fig. 2) [2–5].

We found three case reports of ischemic stroke [6,8,9]. These patients had typical stroke syndromes which developed between 2 h and two days after the stings; they also had injuries in the CNS outside the area causing the primary deficit. These patients were treated with steroids ($n=2$), antihistamines ($n=3$), anticholinergics ($n=1$), antiemetic drugs ($n=1$), aspirin ($n=1$), and heparin ($n=1$) but did not receive thrombolytic therapy. None of them recovered fully. Three case reports describe hemorrhagic strokes [7,10,11] which developed between 2 and 24 h after bee stings and caused significant neurological deficits. Two patients died [11] and the other one remained paralyzed [10]. Three cases had other neurological complications (Table 1) [12,13,15], including an acute brachial plexopathy, acute disseminated encephalopathy and parkinsonism, which developed between 30 min and 10 days after stings. One patient had a persistent facial asymmetry and weakness [14] which developed in childhood. Two patients recovered [12,13], one patient improved [15], and one patient remained symptomatic [14]. Therefore, neurological deficits after bee stings are heterogeneous. They can be caused by an allergic reaction or a direct toxic effect by neurotoxins and mediators released from damaged cells. The prognosis depends on the mechanism, the patient's age, and preexisting conditions but is often poor, especially with hemorrhage.

Cardiac complications after bee stings occurred in two groups Table 2. The first group includes patients presenting with arrhythmia and/or other ECG changes following bee stings. These patients had T

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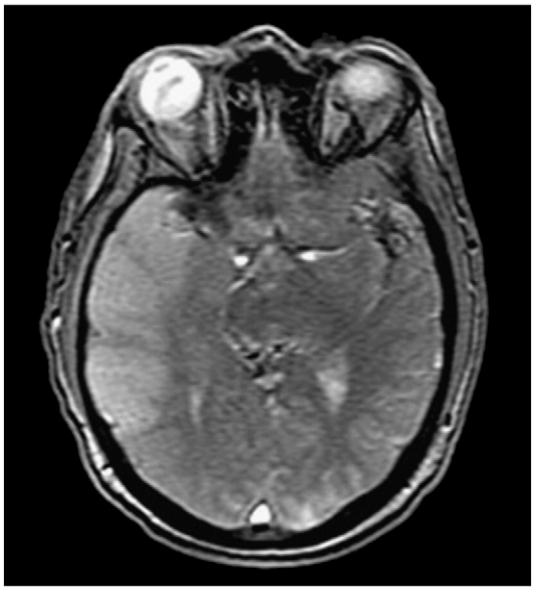

Fig. 1. Brain MRI. Large right MCA territory ischemic stroke and degree of mass effect and 2–3 mm midline shift to the left.

wave inversion [24], atrial flutter [25], atrial fibrillation with rapid ventricular response [25] and ventricular fibrillation [26]. These patients developed symptoms and ECG changes from minutes to several hours after bee stings. The second group included patients presenting with acute coronary syndrome following bee stings. These patients had typical chest pain that developed within 60 min in two patients and after 60 min in six patients. Three patients had normal coronary arteries [16,17,19]. Five patients had significant coronary occlusion(s) and were successfully treated with stenting [18,20–23]. Four recovered [18,20,22,23], and one had an apical thrombus during follow up [21]. The pathogenesis of these events likely involves both vasospasm and acute thrombosis.

We found twenty-one cases of kidney injury following bee stings [27–41] (Table 3). The onset of symptoms varied from immediately after the bee stings to ten days. Thirteen patients had rhabdomyolysis and hemolysis with subsequent acute tubular necrosis. Three patients had a nephrotic syndrome [27,35,37]. One patient had anaphylactic shock [36]; one had serum sickness [33]. The patients with nephrotic syndrome [27,35,37] did not require hemodialysis or peritoneal dialysis. Nine patients with the other syndromes required hemodialysis [28,29,31,34,39], and four patients required peritoneal dialysis [33,36,38,40]. Seventeen patients recovered during follow-up [27–29,32–41], one patient retained moderate renal insufficiency [31], and three died [29,30,33]. The patients with acute tubular necrosis strongly support the pathogenetic sequence of cell injury releasing cellular constituents, especially hemoglobin, which cause delayed renal injury.

In summary, some patients with bee stings have delayed cardiovascular events with acute coronary syndrome and/ or stroke. These probably reflect direct toxic reactions and not allergic reactions. These patients are potentially candidates for short term anticoagulant therapy at the time of the stings and for thrombolytic therapy if they develop acute CNS ischemic injury after the stings.

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The authors of this manuscript have certified that they comply with the Principles of Ethical Publishing in the International Journal of Cardiology [42].

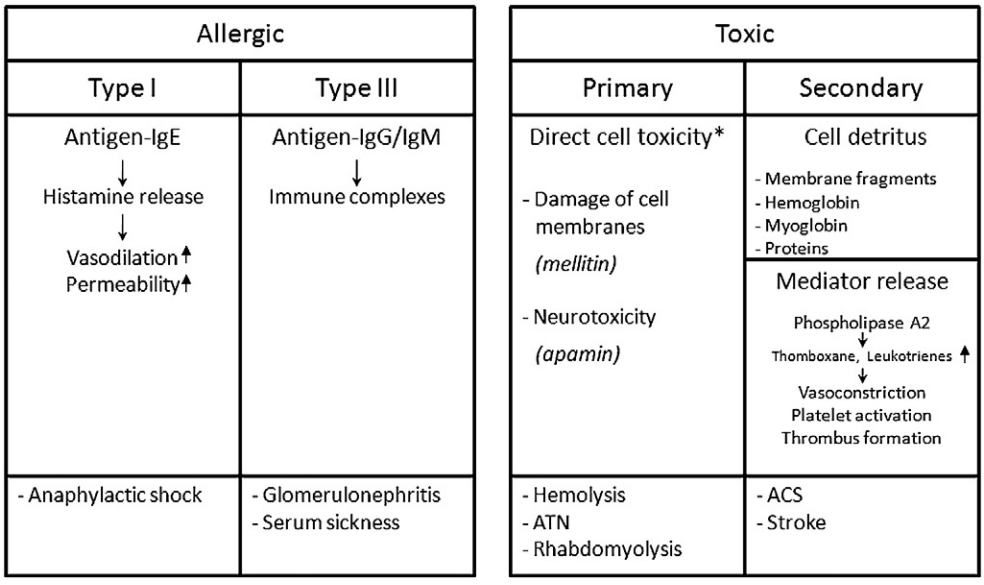

Fig. 2. Effects of bee venom.