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Letters to the Editor

Rhabdomyolysis due to multiple honey bee stings

Sir,

Recently, we were involved in the management of a patient who presented to us with renal failure due to venom-induced rhabdomyolysis as a result of multiple bee stings. Severe allergic reactions subsequent to hymenoptera sting is a well-documented emergency.[1] Optic neuritis, vasculitis, generalized polyneuropathy, myasthenia gravis and rhabdomyolysis are some of the unusual reactions following a sting. We report such an unusual case.

A 43 year old South Indian male was exposed to multiple bee stings while rescuing some school girls who had been on a trekking expedition. The school girls were discharged after first aid and none suffered from any major systemic reactions. On admission the patient complained of tightness over the throat, face and swelling over the site of sting. Clinical examination revealed severe angioedema of face, periorbital region, neck and arms. He had sustained 1,600-1,650 sting marks on the face, arms, chest, trunk and tongue. Barbed stings present on the skin were removed. Blood pressure was 120/70 mmHg. Systemic examination was essentially normal. After 16 hours of admission he complained of generalized aches and pains, and passed 200 ml of cola-coloured urine. Thereafter he remained oliguric for 2 days. After one week he complained of partial deafness. Two honey bees were extracted from his right external ear and were sent for identification. They were identified as Indian rock bees or great honey bees of the order Hymenoptera, family Apidae, genus *Apis*, species *dorsata*.

Investigations showed Hb 14.5 g/dl, blood urea 24.9 mmol/l (3.3-6.6 mmol/l), serum creatinine 388.96 μmol/l (44.2-132.6 μmol/l), sodium 142 mmol/l, potassium 5.6 mmol/l, creatine kinase 1,721 IU/l (0-195 IU/l) and lactic acid dehydrogenase 1,200 IU (230-560 IU). Urinalysis was normal.

Skeletal muscle biopsy was taken from right deltoid muscle, which showed features of muscle damage, with loss of striations and swelling of fibres. Prominent inflammatory reaction was present.

Acute renal failure was managed by mannitol, sodium bicarbonate and optimal intravenous fluids. The cutaneous local reaction was treated by antihistamines, glucocorticoids and local applications. The patient made an uneventful recovery with muscle enzymes and renal function becoming normal in 3 and 5 weeks, respectively.

Each year in the United States twice as many people die as a result of stings by hymenoptera insects as from poisonous snakes.[2] There are three distinct genera of hymenoptera capable of causing sting reactions, namely, Apidae (various species of bee), Vespidae (hornet, yellow jackets and wasps) and Solenopsis (fire ants). The bees have barbed stingers that remain in skin after a sting. The stingers have to be removed to prevent further envenomation from the attached gland. We removed 103 stings from our patient.

Acute renal failure following rhabdomyolysis has been reported due to hornet,[3,4] vespid wasps[5] and honey bee.[2] The muscle necrosis is due to the presence in the venom of toxic amines and peptides, such as phospholipases, histamines, polypeptidases, serotonin and kinins. Acute renal failure can also occur due to the direct nephrotoxicity of the venom. The generalized myalgia, cola-coloured urine, raised muscle enzyme and skeletal muscle biopsy favours rhabdomyolysis as the cause of acute renal failure in our patient.

Successful management of rhabdomyolysis depends upon early diagnosis and therefore a high index of clinical suspicion. The mainstay for prevention and treatment of myoglobunuric acute renal failure is sodium bicarbonate. The object is to maintain an alkaline urine and prevent dissociation of myoglobin to its nephrotoxic metabolite ferrihemate. Diuretics, notably, mannitol, have a role by promoting diuresis thereby diluting the nephrotoxic substance and flushing through blocked renal tubules.[6] Our patient was managed on the above principles, resulting in diuresis and resolution of renal failure.

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Response to dietary restrictions in migraine: a comparison of results in children and adults

Sir,

The role of various kinds of food as specific provocative agents in inducing attacks in migraineurs is well documented.[1-3] We wish to report a marked difference in the frequency of this factor between children and adults, as indicated by positive responses to cessation of intake of the particular dietary factor implicated. We are not aware of this difference having been emphasized previously.

A total of 248 children aged 7-16 (males 104) and 216 adults (males 68) aged 20-54 years, suffering from migraine with or without aurae were seen after 4 weeks, and subsequently followed at monthly intervals over a total period of 12 weeks, whilst suspected food items were

Table I Results of dietary restriction

	Adults	*Children*
Cessation of attacks	14	76
Reduction >80%	22	86
Reduction >50%	38	42
No response	142	44
Total	216	248

eliminated from their diet. In those with favourable responses, chocolate was the chief provocative dietetic factor in 90% of both groups, cheese in 30% (with some 20% as an additional factor), and citrus fruit in some 10% again with occasional double sensitivity. Alcohol was responsible in 8% of the adults, with isolated instances only implicating bananas and beans. No other form of prophylactic therapy was administered concomitantly. Results were recorded as either cessation of attacks, reduction of over 80%, over 50%, or as no response (Table I), and are summarized as favourable in 82% of children as against 34% of adults.

We have no clear explanation for this disparity, beyond suggesting that in adults, factors additional to food allergy, such as psychological stress or perhaps hormonal influences may also play a prominent role in the precipitation of attacks. Sex differences seemed not to be involved in the outcome.

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Barium carbonate, hypokalaemic paralysis and trismus

Sir,

Barium carbonate, a rodenticide being used in this part of the world, causes hypokalaemic paralysis.[1] I report a patient who developed trismus, which is a hitherto unreported manifestation of this poisoning. It was associated with hypokalaemia and flaccid quadriparesis.

A 28 year old man was admitted with a history of ingestion of barium carbonate followed by vomiting. About 12 hours later, the patient developed gradually progressive quadriparesis with difficulty in speech and swallowing. Examination revealed a bulbar palsy and flaccid quadriparesis; the lower limbs were affected more severely than the upper. Blood pressure was 110/70 mmHg.

About 20 hours after ingestion, the patient developed trismus. There were no involuntary movements or convulsions. The neck muscles were not involved. The patient could not talk. Cranial nerve examination revealed only bulbar palsy. Percussion myotonia was absent. There was no sensory deficit on the face. Four hours later, the trismus disappeared along with recovery of the weakness of the limbs. The patient made an uneventful complete recovery. During his stay in hospital, he was given parenteral potassium chloride, magnesium sulphate and supportive treatment, such as intravenous fluids and supplemental oxygen.

Investigations revealed that haemoglobin, blood urea, sugar, creatinine and sodium were within normal limits. Serum potassium was 2.14 mmol/l at the time of weakness. Electrocardiogram showed T-wave inversion in inferior leads and V_1-V_3. Chest X-ray was normal.

Barium salts block potassium channels and thereby reduce potassium efflux from the muscle; potassium uptake by muscle, mediated by the sodium potassium pump continues and hypokalaemia results.[1] An increased sodium conductance may also result in hypokalaemia.[2] The hypokalaemia causes a flaccid quadriparesis.

Myotonia is a transient uncontrollable muscle tension during voluntary muscle contraction. The tension is caused by inability of skeletal muscle to relax normally. Bryant and Lipicky[3] found chloride conductance to be decreased in myotonia congenita. More recently, abnormalities in ion channels have been found in one form of myotonia congenita, myotonic dystrophy and in several forms of periodic paralysis.[4] There is current evidence to suggest that potassium-sensitive periodic paralysis and myotonic disorders are the result of single base-pair changes in the α-subunit of the skeletal muscle sodium channel gene.[4]

Although myotonia of jaw muscles has not been reported with barium carbonate poisoning, there is a reference to eyelid myotonia with hypokalaemic periodic paralysis.[5] In fact, until 1985, there were only two cases of hypokalaemic periodic paralysis – familial primary or thyrotoxic in which myotonia was described.[6,7] I surmise that trismus may be a manifestation of localized myotonia due to secondary periodic paralysis (hypokalaemic) as a result of barium carbonate, since both myotonia and periodic paralysis are dependent on ion-sensitive channels (which may have a genetic predisposition).

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