To the Editors:

Severe multi-organ dysfunction following multiple wasp \((\text{Vespa affinis})\) stings

The wasp \((\text{Vespa affinis})\) is common and widely distributed in Sri Lanka. Wasp venom is known to contain factors that release histamine, enzymes, haemolysins, neurotoxins and vasodilators [1]. Case reports with severe multi-organ dysfunction after wasp stings are few in literature. We report two patients who developed severe multi-organ dysfunction following multiple wasp bites.

Case report 1

A 38-year old previously healthy male was admitted with about 70 wasp stings in September 2000. He felt intense pain and developed swelling of the body immediately. He passed red coloured urine just after the stings and became unconscious 4 h later. He progressively became oliguric with marked swelling of the body and repeated vomiting over the next 2 days. Examination on day 3 revealed deep icterus, conjunctival pallor, oedema of the body, a blood pressure of 170/110 mmHg, a pulse rate of 106/min, myalgia and tenderness in the abdomen. On the day 4 he developed hepatic flaps and became drowsy and dyspnoeic. The oliguria persisted.

His serum creatinine was 340 \(\mu \text{mol/L}\), blood urea 18.4 \(\mu \text{mol/L}\), total serum bilirubin 213 \(\mu \text{mol/L}\) (direct 165 \(\mu \text{mol/L}\)), serum alkaline phosphatase 674 u/L, serum alanine aminotransferase 1778 u/L, serum aspartate aminotransferase 1,773 u/L, serum potassium 5.8 mmol/L, serum amylase 529 u/L, Hb\% 120 g/L, WBC 18 x 10^9/L with neutrophils 86% and platelet count 138 x 10^9 L. Albumin, pus cells and red cells were detected in the urine. The electrocardiogram (ECG) showed antero-lateral non-Q wave myocardial infarction.

He was managed with peritoneal dialysis and liver failure regimen. Peritoneal dialysis was continued for 24 days. Repeated ultrasound examinations showed oedematous kidneys persisting up to the 40th day and the serum creatinine level remained elevated for 50 days. The liver biochemistry reversed gradually. Subsequently, he developed mild hypertension (BP 150/100 mmHg) lasting for 5 months despite normal renal function.

Case report 2

A man aged 57 years was stung by 50 to 60 wasps while working in the home garden in October 2001. He was conscious and had generalised oedema, severe myalgia and a blood pressure of 100/80 mmHg. On the first day, he passed 200 ml of dark urine. The blood urea was 9 mmol/L and serum potassium 4.3 mmol/L. Myoglobinuria was detected. On the second day, his urine output increased to 700 mL, but a 12 lead ECG showed tall peaked T-waves suggesting hyperkalaemia. Peritoneal dialysis was commenced but he died from ventricular fibrillation. At necropsy, multiple sting marks were observed on the scalp, chest, back and arms.

The skeletal muscles were dark red in colour, the lungs were oedematous and the heart showed an occluded right coronary and anterior descending artery. There was no histological evidence of infarction. The kidneys were normal in size but the cut surface showed indistinct cortico-medullary demarcation. The brain and liver were normal. Histology of the kidney showed acute tubular necrosis.

Very often wasp stings are ignored or treated with home remedies. It is not uncommon to see deaths in wasp stings due to anaphylaxis and persistent shock especially among elderly and victims with concurrent illnesses.

Acute myocardial infarction following wasp stings was reported in two patients in 1972 and one of them was young and healthy [2]. Severe hypotension due to anaphylaxis could be attributed as the causation of acute cardiac ischaemia, but other factors such as direct vascular effect of venom need to be excluded. Isolated cases where systemic manifestations included syndromes that mimicked myasthenia gravis, reversible optic neuropathy and mastocytosis are reported [3–6].

The patients reported here had acute life threatening multi-systemic involvement that included myocardial infarction, acute hepatorenal failure and pancreatitis. Severe muscle damage producing myoglobinuria with hypotension and hypovolaemia might have contributed to the renal damage.

References
To the Editors:

A case of self-limiting Coomb’s negative haemolytic anaemia following dengue shock syndrome

In addition to dengue haemorrhagic fever and dengue shock syndrome, involvement of other organ systems has been reported in dengue fever. Hepatic dysfunction and neurological manifestations are common [1,2]. We report a patient who developed a self-limiting Coomb’s negative haemolytic anaemia following dengue shock syndrome.

A 27-year old woman who was clinically diagnosed to have dengue fever developed shock (dengue shock syndrome) 12 h after admission to hospital. She was transferred to the intensive care unit and resuscitated. On the sixth day after admission dengue IgM antibodies became positive. She recovered and her haemoglobin was 12 g/dL and platelet count was 140 x 10^9/L on the seventh day. During the next 2 days she became increasingly pale without evidence of haemorrhage and developed icterus. The haemoglobin had dropped to 6 g/dL. The following investigations were done: platelet count 205 x 10^9/L, blood picture normochromic normocytic red cells with marked red cell agglutination, reticulocyte count 8.2%, white cell count 6.4 x 10^9/L, total bilirubin 168.9 µmol/L (indirect fraction 100 µmol/L), ESR 98 mm in the first hour, direct and indirect Coomb’s tests repeatedly negative. Her prothrombin time/INR, partial thromboplastin time, C-reactive protein and D-dimer levels were normal. Liver enzymes were mildly elevated (SGPT=56 U/L and SGOT=94 U/L). Mycoplasma pneumoniae, Epstein-Barr IgM and IgG antibodies, antinuclear factor and anti-dsDNA antibodies were negative. Chest x-ray, abdominal ultrasound scan, blood urea, serum electrolytes and serum creatinine were also normal.

She recovered spontaneously and her haemoglobin was 10.2 g/dL when she was discharged from the hospital 6 days after haemolysis was first detected. Two weeks after admission dengue antibodies showed an IgG titre of more than 2560, confirming recent secondary dengue infection. At follow up she was asymptomatic and had normal haematological and biochemical parameters.

This patient had a self-limiting haemolytic anaemia 6 days after dengue shock syndrome. An extensive literature survey did not reveal previous reports of such an association. We have excluded, as far as possible, other likely causes of haemolysis. The mechanism of the haemolytic anaemia is not clear. Cold-type autoimmune haemolytic anaemia is a recognised complication of certain infections, characterised by destruction of antibody-coated red blood cells. The mechanism that initiates production of autoantibodies remains unclear. Regulatory cytokines are thought to play an important role, and activation of immunoregulatory T lymphocyte subsets has been observed in dengue infection [3]. In our patient, an immune mechanism was considered because of delay between the infection and onset of haemolysis. Furthermore, her blood film was suggestive of a cold-type autoimmune haemolysis. However, the Coomb’s test was repeatedly negative. Coomb’s negative autoimmune haemolytic anaemia is known to occur when haemolysis is caused solely by IgA antibodies [4]. We checked only IgM and IgG antibodies and had no facilities to test for IgA antibodies.

References
