Haemolytic–uraemic syndrome following a scorpion sting

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Introduction

Microangiopathic haemolytic anaemia, thrombocytopenia, and acute renal failure are characteristic of haemolytic–uraemic syndrome (HUS) [1]. Although it is more commonly described in children, it also occurs in adults, particularly following an episode of diarrhoea caused by Escherichia coli 0157:H7 but also following ingestion of drugs and malignancies, pregnancy, systemic disorders, AIDS, organ transplantation, and other glomerulopathies [1]. Although some scorpion venoms contain haemolysins, endotheliolysins, and neurotoxins [2], to our knowledge this is the first reported case of HUS following a scorpion sting.

Case

A 28-year-old man presented in December 1997 with a 24-h history of right leg pain, vomiting, diarrhoea, agitation, restlessness and oliguria following a scorpion sting 2 days previously. The sting puncture was sucked and ammonia was applied locally without antivenom treatment. On examination he was flushed, had an ecchymotic and necrotic area with local oedema on the right ankle (6 × 10 cm), his consciousness was impaired, vision blurred; temperature 38°C, blood pressure (BP) 40/10 mmHg, and pulse 120/min. Fine inspiratory rales were audible. Peripheral cyanosis and increased lacrimation and salivation were present. CVP was 5 cm H₂O. Haematology revealed Hb 10.4 g/dl, WBC 16 × 10⁹/l, platelets 77 × 10⁹/l, blood smear showed fragmented erythrocytes and burr cells; platelets were rarely seen. Urinalysis showed 4+ proteinuria and microscopic haematuria. Prothrombin time and partial thromboplastin time were 12 and 33 s respectively. Fibrinogen and fibrin split products were elevated to 1300 mg/dl and 2000 ng/dl respectively. Serum creatinine was 7.7 mg/dl, blood urea nitrogen 93 mg/dl, electrolytes were normal, aspartate aminotransferase 121 IU/l, alanine aminotransferase 44 IU/l, lactic dehydrogenase 1442 IU/l, creatinine phosphokinase 1400 U/day for 5 days), and dopamine infusion were given. BP was labile, ranging from 90/60 to 240/160 mmHg on day 1; later, he became hypertensive, responding to a Ca²⁺ channel blocking agent and vasodilators. He was well by day 10.

Discussion

Haemolytic–uraemic syndrome was first described in 1955 and is now widely recognised [1]. Although the pathogenesis in most cases is unclear, endothelial cell injury, with subsequent vasoconstriction, platelet activation, unbalanced prostacyclin/thromboxane ratio, and decreased release of endothelium-derived relaxing factor may play a role in the pathogenesis of HUS [1,3]. Any agent causing endothelial-derived relaxing factor may play a role in the pathogenesis of HUS [1,3]. Any agent causing endothelial injury is presumably able to trigger HUS.

Glands in the terminal segments of scorpions produce venom which is injected into the victim by a stinger located on the tip of the tail [2]. Of about 650 scorpion species, about 10 occur in Turkey [4]. Eighty-five Scorpion stings were recorded by RSH National Poison Center of Turkey in 1996; 20 of these were severe. The most common species in our region are Euscorpius carpathicus, E. germanus cliciensis and E.italicus mingrelious [4]. Non-lethal species of scorpions cause only local tissue reaction, while that of...
others is primarily neurotoxic. The latter type contains several components, including haemolysins, endothelio-
olysins, and neurotoxins, and typically produces aching
pain and numbness radiating from the site of injury,
lymphadenitis, ascending motor paralysis, convulsions
and coma. Release of catecholamines may results
in tachycardia, arrhythmias, and hypertension.
Myocarditis, pancreatitis, and cardiomyopathy have
been reported. Venoms contain numerous toxic poly-
peptides displaying various pharmacological activities.
These toxins interact with ion channels of excitable
membranes [5].

Our observation suggests that scorpion stings should
be added to the ever-growing list of causes of the
haemolytic–uraemic syndrome.

References
Am 1995; 42: 1505–1529
2. Wallace JF. Disorders caused by venoms, bites, and stings. In:
Isselbacher KJ, ed. Harrison’s Principles of Internal Medicine,
3. Thompson CE, Damon LE, Ries CA, Linker CA. Thrombotic
microangiopathies in the 1980s: clinical features, response to
treatment, and the impact of the human immunodeficiency virus
5. Inisan AG, Meunier S, Fedelli O et al. Structure–activity relation-
ship study of a scorpion toxin with high affinity for apamin-
sensitive potassium channels by means of the solution structure

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