Scorpion Sting Presenting as Subarachnoid Hemorrhage in an Elderly Male

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Introduction

Scorpion sting is an acute life-threatening, time-limiting medical emergency and a major public health problem in many underdeveloped tropical countries, especially Sahelian Africa, South India, Middle East, Mexico, and South Latin America. Out of 1500 scorpion species, 50 are dangerous to humans. Scorpion sting causes a wide range of clinical presentations from severe local skin reactions to neurological, respiratory and cardiovascular collapse. Alpha-receptors stimulation by the toxin results in hypertension, tachycardia, myocardial dysfunction, pulmonary edema. Raised angiotensin I levels have also been documented, which facilitates the sympathetic outflow through conversion to angiotensin II. Acute rise in blood pressure due to sympathetic stimulation, rupture of unprotected perforating arteries, intracerebral hemorrhage and cerebral infarction are reported in scorpion stings. Subarachnoid hemorrhage has been reported in cases of acquired vascular defects due to hypertension, age and smoking related atherosclerosis.

Case Report

A 60 year old man presented with severe headache, vomiting, altered sensorium with pain and paraesthesias at local site following scorpion sting one day back. On admission patient was febrile (101°F) with altered mental function (GCS 13: E3M6V4). He had tachycardia (pulse 98/min), hypertension (220/120 mm of Hg), respiratory rate 20/minute and spO2 96% without oxygen. Neck rigidity was present with positive Kernig’s sign. Pupils were normal size and normally reactive to light. Extensor plantar response was elicited bilaterally. There was no abnormality on other systemic examination. Patient was a chronic smoker and had no significant illness in past. Complete blood count and routine biochemistry were unremarkable except for a mildly raised total leukocyte count (12,000/cmm) and mildly deranged liver function test (SGOT/SGPT: 94/62 units). Tests of coagulopathy, urine examination, ECG, Chest X-Ray and ultrasound abdomen were within normal limits. Long standing hypertension was ruled out by normal ECG, fundus, lipid profile, echocardiography and previous medical records. CT scan head showed evidence of subarachnoid hemorrhage. MRI brain with angiography did not show any evidence of aneurysm. The patient was managed conservatively with supportive and symptomatic care, central venous pressure monitoring, decongestives (mannitol and glycerol), analgesics, calcium channel blocker (nimodipine), prazosin, anticonvulsants (phenytoin), antibiotics, intravenous fluids. Patient responded to treatment and was discharged in stable condition on tenth post admission day.

Discussion

Scorpion venom is a water-soluble, antigenic, heterogeneous mixture, as demonstrated on electrophoresis studies. Scorpion venoms are species-specific complex mixtures of short neurotoxic proteins. The venom is composed of varying concentrations of neurotoxin, cardiotoxin, nephrotoxin, hemolytic toxin, phosphodiesterases, phospholipases, hyaluronidases, glycosaminoglycans, histamine, serotonin, tryptophan and cytokine releasers. The primary targets of scorpion
venom are voltage-dependent ion channels, of which sodium channels are the best studied. Venom toxins alter these channels, leading to prolonged neuronal activity. The long-chain polypeptide neurotoxin causes stabilization of voltage-dependent sodium channels in the open position, leading to continuous, prolonged, repetitive firing of the somatic, sympathetic and parasympathetic neurons. This repetitive firing result in autonomic and neuromuscular overexcitation symptoms and it prevents normal nerve impulse transmissions. Furthermore, it results in release of excessive neurotransmitters such as epinephrine, norepinephrine, acetylcholine, glutamate and aspartate. Meanwhile, the short polypeptide neurotoxin blocks the potassium channels. Signs of autonomic nervous system involvement include sympathetic overactivity in the form of hyperthermia, tachypnea, tachycardia, hypertension, arrhythmia, pulmonary edema, diaphoresis, piloerection and parasympathetic involvement in the form of bradycardia, hypotension, salivation, rhinorrhea, bronchoconstriction, loss of bowel and bladder control. Cerebral infarction, cerebral thrombosis, acute hypertensive encephalopathy and cerebral hemorrhage have been described with a variety of Buthidae scorpion envenomations.

In our patient, subarachnoid hemorrhage can be best explained by accelerated hypertension due to sympathetic overactivity following scorpion sting which led to rupture of subclinical aneurysmal vessel wall which was weakened by age and smoking related atherosclerotic process. Treatment with prazosin, if initiated early, may prevent many cerebrovascular manifestations of scorpion sting.8

References