A 40-year-old woman was stung by a scorpion on her left foot. Sharp burning pain at the bite site was followed by a high-grade fever, severe breathlessness, oliguria, and altered sensorium over a period of hours. She was rushed in an unconscious state to a critical care center where blood pressure was 90/60, and she was diagnosed with acute pulmonary edema, myocarditis, and acute renal failure (venom-induced multiorgan failure). After endotracheal intubation, she received low-dose aspirin, enalapril, and supplemental intravenous fluids.

Eight hours later, on recovery of consciousness, she reported poor vision in both eyes. On examination 1 week later, visual acuity was light perception in both eyes. Anterior segments were unremarkable, including pupillary reactions. A dilated fundus examination was normal in both eyes. Limb ataxia, dysdiadochokinesis, poor tandem walking, and staccato speech could be elicited. Otherwise the findings from neurological examination appeared to be normal.
Brain MRI revealed restricted diffusion in the medial occipital (Fig. 1) and occipitotemporal lobes and cerebellum bilaterally. Results of a coagulation profile, C3 and C4 complement, homocysteine, protein C and S, VDRL, antinuclear antibody (ANA), and antiphospholipid antibody were negative. Cerebrospinal fluid analysis and a color Doppler study of both carotid and vertebral arteries showed normal results.

A diagnosis of cerebral and cerebellar infarction was made. No direct treatment occurred. Over 10 days, there was gradual improvement so that the patient was able to walk without support. At the 6-month follow up, visual acuity was light perception in both eyes, the only neurologic deficit.

Scorpion bites affect the central nervous system in three ways: altered consciousness, seizures, and infarctions (1). Cerebral and/or cerebellar infarctions have been reported (2-7), with numerous mechanisms advanced to explain them: 1) an acute rise in blood pressure during the autonomic storm that ruptures unprotected or diseased vessels (2); 2) toxic myocarditis that precipitates arrhythmias that give rise to embolic stroke (2); 3) hypercoagulability (3); 4) disseminated intravascular coagulation (4); 5) vasculitis caused by venom (5); and 6) hypotension caused by myocarditis, parasympathetic overactivity, and dehydration (6).

Bilateral optic neuropathy (6), transient ophthalmoplegia (8), transient blindness (9), and myelopathy (10) have been documented. We believe that this is the first description of persistent (and severe) cerebral visual loss after a scorpion bite. We cannot be certain of the mechanism, although hypotension seems likely.

Sabyasachi Sengupta, DO

Praveen Dhanapal, MBBS

Ravilla D. Ravindran, MS, DO

Nirmala Devi, DO

Department of Neuro-Ophthalmology
REFERENCES


