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Scorpion Sting - A Rare Cause of Stroke in the Young

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Abstract

Two cases of hemiplegia following scorpion sting are described. The infarction was haemorrhagic in one and ischaemic in the other. The pathophysiology of this rare manifestation of scorpion sting is discussed.

Key words -
Stroke in the young,
Scorpion sting,
Venous thrombosis

Scorpion sting is a common problem in rural India. Majority of cases recover and death though rare, is mainly due to pulmonary oedema [1], [2]. A variety of complications following scorpion sting are described, however, reports of neurological complications are rare [3], [4]. "Stroke in young" is a diagnostic dilemma. Recent reviews do not make any mention of scorpion sting as a cause [5]. The authors in this communication report two young patients who developed stroke following scorpion sting.

Patient 1:

A 28 year old man was stung by a scorpion on the left little toe. Fifteen minutes later, he developed vomiting and inability to talk and walk. He complained of headache and indicated his needs by gesture. Examination by the local doctor revealed signs of peripheral circulatory failure, dysphasia and right hemiparesis. He was given intravenous fluids and dexamethasone and injection xylocaine locally and then was transferred to NIMHANS, Bangalore the next day. On examination, he was alert and responded to verbal commands by unintelligible monosyllables. He obeyed simple commands and exhibited motor perseveration. He had right hemiparesis along with weakness of right side of face and tongue. Corneal, abdominal and cremasteric reflexes were normal but gag and palatal reflexes were absent on both sides. Muscles stretch reflexes were brisk bilaterally and plantar response was extensor on both sides. There were no sensory deficits or signs of meningeal irritation. His systemic examination was unremarkable.

The following investigations revealed normal or negative results. Total and differential leukocyte count ESR, platelet count, bleeding, clotting and prothrombin time, blood urea and sugar, serum GOT, GPT, bilirubin, cholesterol, triglycerides, creatinine, uric acid, VDRL, LE cell phenomenon and Rheumatoid factor, total proteins and electro-phoresis, X-rays of chest and skull and electrocardiogram. His haemoglobin was 10.6 gm%. CSF analysis revealed xanthochromic fluid, 12 leukocytes/cmm (lymphocytes and polymorphs), 640 RBCs/ cmm, 48 mg% glucose and 150 mg% protein. Arch - aortogram and four vessel studies did not reveal any abnormality. He made partial recovery over a period of 15 days.

Patient 2:

A 10 year old boy had scorpion sting over scalp. A few minutes later he developed vomiting and profuse sweating. Vomiting stopped shortly but sweating continued. Two days later he was seen at a peripheral hospital, and was found to have features of pulmonary oedema. He received digoxin, frusemide and made a dramatic recovery. A day later he reported pain and swelling of right leg and fever. On the 9th day of scorpion sting, he suddenly developed aphasia and right hemiparesis. There was no history of convulsion, headache or visual impairment. His past health was unremarkable. There was no family history of premature atherosclerosis or stroke. On examination he was afebrile, but had tender swelling of right calf, soft hepatomegaly of 1 cm and short systolic murmur over left parasternal region. Peripheral vessels were normally felt and there was no bruit over carotid vessels. He had global aphasia and right hemiparesis (1-2/5 in lower limb and 0- 1/5 in upper limb) but visual and sensory functions were grossly normal. Muscle stretch reflexes were brisk on right side and plantar response was extensor. Ocular fundi were normal and there were no signs of meningeal irritation.

The following laboratory test were negative or normal. Urine, haemoglobin, ESR, total and differential leukocyte count, platelet count, bleeding, clotting and prothrombin time, blood urea and glucose, serum cretinine, electrolytes, cholesterol, total proteins, bilirubin, calcium, phosphorus, alkaline phosphatase, Rheumatoid factor and LE cell phenomenon. However, serum GOT-70 U/L (normal < 30), GPT-113 U/L (normal <30), creatine Kinase 770 U/L (normal <170) and LDH-2062 U/L (normal <450) and triglycerides 515 mg%, (normal < 150) were raised. While serum GOT, GPT, LDH and CK improved within two weeks, triglycerides remained high. CT scan showed hypodense lesion, conforming to anterior and middle cerebral artery territory on the left side, with mass effect. Electrocardiogram and echo-cardiogram were normal. Patient received oral glycerol, parenteral dexamethasone and intravenous heparin (1500 IU/6th hourly) for two weeks. He made quick recovery. He could comprehend well but could not express. Motor power improved (upper limb 3/5 and lower limb 4/5) and he could walk unaided with mild spastic gait at six weeks.

Discussion

Both the patients were young, had acute onset of neurological deficit followed by rapid but partial recovery. There were no predisposing factors for stroke except for elevated triglycerides in case 2. The paralysis was noted within a few minutes after the bite in Case 1. However, contrary to most of the

reported cases it was delayed by nine days in case 2. Case 1 had haemorrhagic infarction, as evident by xanthochromic CSF, and case 2 had ischaemic infarct on CT. There was no bleeding diathesis or abnormalities of bleeding, clotting or prothrombin time. Both the patients had transient hypotension at the onset and case 2, in addition, had thrombophlebitis of leg veins, remote from the site of scorpion sting. Cardiac examination and EKG were normal in both and Echo cardiogram was also unremarkable in case 2. Case 1 received only steroid therapy as there was evidence of haemorrhagic CSF but case 2 was given steroids along with heparin for two weeks.

There are two morphological types of scorpions - black and brown. Black scorpion's sting is more harmful. Nearly 600 species of scorpions are recognized of which Buthus tumulus is the common Indian scorpion. The poison of scorpion contains hemolysins, agglutinins, ferments, coagulins, lecithin, cholesterin and neurotoxin. The latter acts on respiratory and vasomotor centers, end plates of striated and non-striated muscles and nerve terminals but cannot directly explain the stroke like manifestation [3]. The authors could find only eight case reports of hemiplegia following scorpion sting [6], [7], [8], [9], [10], [11], [12], [13]. Of them, majority of cases were children and had variable recovery. One of these cases [8] had also evidence of popliteal artery thrombosis and gangrene. In the present report one patient was an adult while the other had evidence of leg vein thrombosis.

The exact pathogenesis of stroke following scorpion sting is obscure. Sithadevi and her colleague [14] based on autopsy features of fatal cases and elegant experimental work in animals postulated that disseminated intravascular coagulation followed by bleeding tendency could account for the complications. They observed that administration of heparin could reverse the abnormal coagulation parameters and prevent the mortality. However, similar work by Gajalakshmi [15] contradicted the above observation. Myocarditis with or without arrhythmia causing hypotension and border-zone infarction and hyper-coagulability due to coagulins in venom causing cerebral arterial or venous thrombosis or peripheral venous thrombosis and paradoxical embolism through patent foramen ovale are other possible mechanisms. In both the cases there was transient hypotension but the clinical feature and CT Scan findings did not corroborate border zone infarction. There were no features of disseminated intravascular coagulation or bleeding diathesis. It is likely that hypertriglycerdemia partly increased the risk of stroke in Case 2. Our cases differed form the cases reported earlier in
(1) both carotid and the vertebro basilar systems were involved,
(2) infarction was haemorrhagic in case 1 as evident by xanthochromic CSF and
(3) there was a time lag between the scorpion sting and stroke in Case 2.

Hypercoagulability of blood perhaps could be yet another factor in cerebral infarction, as evident by simultaneous peripheral venous and cerebral arterial thrombosis in case 2. Lack of facilities to estimate various coagulation parameters prevented us from confirming the same.

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