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Management of Severe Scorpion Sting at a Rural Hospital

Severe scorpion sting is a life threatening accident not uncommon in Western Maharashtra [1]. Scorpion venom delays closing of sodium neuronal channel resulting in liberation of endogenous catecholamines into circulation, manifesting as “autonomic storm” [2]. In the past, upto 30% fatality due to refractory heart failure due to *Mesobuthus tamulus* envenoming has been reported. Since the advent of prazosin, a postsynaptic alpha blocker, the fatality due to refractory heart failure has reduced to less than 4-8% in pediatric age group [3].

Cottage Hospital is a public health institute situated on Mumbai-Goa highway. The mono specific scorpion antivenom against the *Mesobuthus tumulus* is prepared at Haffkine Institute, Mumbai has been available at our Institution since 2010. We retrospectively analyzed details of 12 children who suffered autonomic storm due to scorpion sting (**Table I**). Of these, six (2 males) had received scorpion antivenom plus prazosin and the remaining (3 males) received only scorpion antivenom. All gave history of scorpion sting and relatives brought the killed specimen.

Alpha receptor stimulation plays important role in the pathogenesis of acute pulmonary edema. Prazosin, by blocking alpha receptors corrects the abnormal hemodynamic, and metabolic effects of circulating catecholamines [3,4]. Patients treated with prazosin alone are reported to recover in 10-24 hours, till the venom is metabolized by body. In such situation victims needs close monitoring in intensive care [8]. 10-20% of children, irrespective of oral prazosin, were found to develop tachycardia, hypotension and pulmonary edema [8]. Scorpion antivenom neutralizes the circulating venom and it has no action in reverseing the effects of already raised catecholamine and tissue-bound venom

TABLE I CHARACTERISTICS OF CHILDREN WITH AUTONOMIC STORM (N=12)

Treatment	Anti-scorpion venom (AScV)	AScV + Prazosin
Age (y)	9 (7-12)	8.8 (3-13)
Time between sting to hospitalization (hours)	2.41 (1-3.5)	1.5 (1-2.5)
Blood pressure (mm Hg)		
Systolic	140 (80-190)	123 (90-170)
Diastolic	80 (60-100)	80 (70-100)
Pulse rate (per min.)	89 (68-110)	89 (84-100)
AScV dose* ((1.2)vial)	2.33 ((2-5)vial)	1.33
Pulmonary edema	3	0
Recovery	3	6
Time for recovery (h)	7.1 (4-8)	3.75 (2-5)

*1 vial=10 mL; Values in mean (range); AScv-Anti-Scorpion venom.

[5]. Cold extremities occur due to alpha receptor stimulation as a result of vasoconstriction, and delay the venom absorption in circulation from site of sting, which acts as depot. Simultaneous use of oral prazosin, which antagonizes the catecholamine actions and improves the peripheral circulation and rapid absorption of venom in circulation that becomes accessible to already circulating antivenom, thus recovery is shortened in prazosin plus antivenom group as compared to scorpion anti venom group alone [5]. Rapid recovery of victims treated with prazosin and antivenom prevent the extra load of these cases to intensive care unit, which is beyond the reach of poor people.

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Childhood Catatonia

Catatonia is a syndrome of motor dysregulation that is rarely recognized in pediatric age group with an estimated incidence of 0.16 million per year [1,2]. In any patient presenting with catatonia, neurological or other general medical conditions, neuroleptic induced side effects, substance intoxication or withdrawal should be ruled out before considering psychiatric etiology [3]. We report a rare case of childhood catatonia due to psychosis.

Master A, 11-year-old male, a fourth standard student, presented with abrupt onset and gradually progressive course of decreased interpersonal interaction and decreased self care for 3 months. He was evaluated in pediatrics, pediatric neurology and endocrinology OPD. Personal and developmental history was uneventful. There was no history of seizure, fever, drug use preceding onset of illness. Apart from BMI of 28, physical examination did not reveal any abnormality. Slit lamp examination did not reveal K-F ring. Hemogram, renal function tests, liver function tests, MRI brain, thyroid profile, serum insulin, fasting blood glucose, post-prandial blood glucose and serum cortisol did not reveal any abnormality. As no organic cause was found, he was referred to child and adolescent psychiatry OPD.

On mental state examination, patient fulfilled syndromal diagnosis of catatonia and had psychotic signs. He was not clinically depressed. He was diagnosed to have psychosis unspecified (F 29) and treated with risperidone (upto 4 mg/day) and lorazepam (upto 4 mg/day). He gradually improved over 8 weeks. Lorazepam was tapered and stopped. He was discharged on risperidone 4 mg/day. At 3 months follow up, he was mildly inactive as compared to his usual self but was doing well otherwise.

Dysregulation of γ -aminobutyric acid (GABA)-A,

glutamate, and dopamine systems are hypothesized to be involved in catatonia [4]. Deprivation, abuse and trauma can precipitate catatonia in paediatric patients without clear medical cause [5]. Acute management of catatonia involves lorazepam challenge test, identifying and correcting underlying medical cause, maintaining adequate nutrition, fluid and electrolyte balance, and avoiding postural immobility which may lead to complications like bed sores or muscle contracture [4]. Electroconvulsive treatment (ECT) is considered as the last choice [4]. As our patient responded well to pharmacotherapy, ECT was not necessitated.

Catatonia is poorly recognized in children and adolescents due to overshadowing by medical or neurological or pervasive developmental disorders [5]. Accurate diagnosis is important because catatonia responds readily to benzodiazepines and electroconvulsive therapy.

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