Myelopathy following Cypermethrin Poisoning

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Abstract
Cypermethrin is a synthetic pyrethroid and pyrimethrin analogue. Cypermethrin poisoning produces neurological manifestations due to its primary target on sodium channels.¹ Neuropathy following cypermethrin poisoning is common. But myelopathy is rare and not reported so far. We report a 17 year old healthy male who developed myelopathy following cypermethrin ingestion. The possible mechanism for the myelopathy in this patient is discussed.

Introduction
The spectrum of manifestations with cypermethrin poisoning are due to its direct action on sodium channels. Neurological manifestations which include neuropathy are common with this poisoning. We present the case of a young male who developed myelopathy after cypermethrin poisoning.

Case Report
17 year old male was admitted in an unconscious state following ingestion of unknown quantity of cypermethrin. On examination patient was unconscious, afibrile, blood pressure was 120/70 mm Hg, pulse rate was 84/min with no pallor, icterus, cyanosis, clubbing, oedema or lymphadenopathy. His pupils were 3 mm in size and equally reacting to light bilaterally. He was treated with stomach wash and supportive measures. He developed respiratory compromise and was put on the ventilator. His ECG, X-ray chest and blood investigations including biochemistry were normal. He was successfully weaned off from ventilator after 15 days.

On 21st day, patient developed difficulty in walking along with bladder involvement in the form of hesitancy of micturition. He had weakness of both lower limbs with power of MRC grade 3, preserved deep tendon reflexes and bilateral extensor plantar reflexes. Bilateral footdrop and high steppage gait were also noted. Sensory and cerebellar systems were normal. MRI (T2 Sagittal) Dorsal spine showed hyperintense lesion in D4-D10 cord segments (Figure 1).

MRI (T2 Axial) Dorsal spine showed hyperintense lesion in D7-D8 segments (Figure 2) in D9-D10 segments (Figure 3). CSF Analysis showed 5 cells/ml 100% lymphocytes, protein of 50 mg % and glucose of 62 mg %. Nerve conduction studies of both upper and lower limb showed features of axonal neuropathy. A detailed vasculitic work up including ANA was negative. A diagnosis of myeloneuropathy following cypermethrin poisoning was made. He was treated with pulse methylprednisolone which resulted in clinical improvement. On follow up after 6 months, his neurological status improved except for the foot drop. His repeat MRI Dorsal spine showed complete resolution of the lesion (Figure 4).

Fig. 1: MRI (T2 sagittal) dorsal spine showing hyperintense signals in D4-D10 cord segments

Fig. 2: MRI (T2 axial) dorsal spine showing hyperintense lesion in D7-D8 segment

Fig. 3: MRI (T2 axial) dorsal spine showing hyperintense lesion in D9-D10 segment

Fig. 4: MRI (T2 sagittal) dorsal spine showing complete resolution of the lesion

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Received: 20.04.2015; Revised: 29.07.2015; Accepted: 28.10.2015
Discussion

Delayed neuropathy and myelopathy are well documented after organophosphorus poisoning. Pyrethroids are a totally different group of man-made pesticides similar to the natural pesticide pyrethrum, which is produced by chrysanthemum flowers. They now constitute the majority of commercial household insecticides. Pyrethroids are axonic excitoxins, the toxic effects of which are mediated through prevention of closure of the voltage-gated sodium channels in the axonal membranes. Cypermethrin is a composite pyrethroid, broad spectrum, non cumulative insecticide and fast acting neurotoxin with great contact and stomach action and a moderately high toxicity to mammals. It is readily absorbed from gastrointestinal tract, may be absorbed by inhalation of dust and fine spray moistures and minimally through the intact skin. Early symptoms include excessive salivation, nausea and vomiting, labored breathing, coarse tremors, hypertension and bradycardia followed by hypotension, tachycardia and convulsions. Neuropathy following cypermethrin poisoning is common but myelopathy is not.

The primary target site of cypermethrin is the sodium channel in nerve membrane. It causes a long lasting prolongation of the sodium permeability of the nerve membrane during excitation leaving the axonal membrane depolarized, thereby producing paralysis. Since the mechanism responsible for nerve impulse generation and conduction are basically the same throughout the entire nervous system, we propose that pyrethroids may exert effects similar to that on the peripheral nervous system (PNS) on the central nervous system (CNS). It could also exert a distinct and reversible inflammatory effect on the CNS resulting in myelopathy. An interesting analogy is the CNS involvement described in patients with Guillain–Barre syndrome. Following autopsy, CNS inflammatory changes were noted distinct from primary demyelination seen in the PNS. Similarly only histopathological studies can throw light on the type of spinal cord changes in cypermethrin poisoning noted in our case.

The temporal profile of events and the total recovery of our patient strongly indicated that the myelopathy is toxin induced.

Conclusion

We feel that in all such instances of cypermethrin poisoning, one should look for evidence of myelopathy clinically and radiologically. To the best of our knowledge, this is the first documented evidence for a reversible myelopathy in cypermethrin poisoning.

References