

Case Report

A DELAYED NEUROLOGICAL COMPLICATION OF PYRETHROID POISONING

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ABSTRACT

Insecticide poisoning is being encountered more frequently in the present day emergency department of hospitals in rural India. Hence the complications of such poisonings are also on the rise. In this case report, a rare complication of pyrethroid poisoning is brought to limelight. In general delayed neurological complications due to insecticide poisoning are caused commonly by organophosphates with lower motor neuron signs in the form of delayed neuropathy. We report a case of pyrethroid poisoning induced delayed weakness, that too with upper motor neuron signs.

Keywords: Pyrethroid Poisoning, Delayed Neuropathy, UMN form of Neuropathy

INTRODUCTION

Insecticide poisoning can cause acute cholinergic crisis, intermediate syndrome and delayed neurological complications (Singh *et al.*, 2000). Delayed neuropathy is a well-recognized complication of organophosphate poisoning (Singh *et al.*, 2004). But pyrethroid induced neuropathy is not reported in literatures. Here we describe a 30-year old male with delayed type of neurological complication in the form of paraplegia with UMN signs.

CASES

A 30-year old male with a suicidal intent consumed approximately 100ml of Karate poison (Lambda cyhalothrin) which is a pyrethroid compound was admitted to hospital. He initially had some of the muscarinic and nicotinic symptoms (salivation, diaphoresis, fasciculation and tachypnoea) of pyrethroid poisoning for which he was treated with supportive measures. Later he developed respiratory failure for which he was intubated and treated with assisted mechanical ventilation. After 24 days of hospital stay, patient was discharged from the hospital without any residual complications. His neurological examination was also within normal limits at the time of discharge.

After 45 days of discharge, this patient was brought again by relatives for weakness of both lower limbs. To start with, he had pain in the right lower limb. After 2 days, he felt the same limb to be tight and weak to such an extent that he had to carry on his routine work only with support. After the onset of weakness in his right lower limb, he also developed pain in his left lower limb and a day later, felt weakness and tightness of left lower limb. There was no disturbance in higher mental function, cranial nerve, sensory, autonomic and cerebellar function both in history as well in examination. Patient was bedridden with 1-2/5 power in both lower limbs with slightly more distal weakness than proximal weakness. Both lower limbs were hypertonic with exaggerated lower limb deep tendon reflexes and patellar clonus. His upper limb examination was perfectly alright.

Routine blood investigations were not significant. MRI spinal cord did not reveal any spinal cord compression. CT- Brain & CSF analysis was normal. Nerve conduction study showed no 'F' waves in tibial & peroneal nerves on both sides. Sciatic, tibial & peroneal (motor) nerves on both sides CMAP was not obtained.

During the 2nd hospital admission, patient was treated with inj. Methyl prednisolone 500mg iv BID & other supportive measures. By the 5th day of hospital admission, patient felt subjective improvement of

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symptoms. By 10th day, patient was able to stand and walk with support. No adverse reactions were seen during the treatment. He was discharged from the hospital with 4/5 power in both lower limbs. On follow up his weakness improved completely.

DISCUSSION

Insecticide poisoning can cause acute cholinergic crisis, intermediate syndrome and delayed neurological complications (Singh *et al* 200). Pyrethroid poisoning also may cause similar initial symptoms with lesser intensity (Senanayake *et al* 1992). Delayed neuropathy is a well-recognized complication of organophosphate poisoning (Jhonson *et al* 1975). But reports of pyrethroid poisoning induced neurological weakness is not present in literature. Through this case we are reporting a delayed neurological weakness caused by pyrethroid poisoning (Lamda cyalothrin) that too with UMN signs.

In our case, when the patient presented with neurological weakness after 2 months of pyrethroid poisoning, we made a provisional diagnosis of paraplegia for evaluation. Patient had obvious UMN signs such as hypertonia, exaggerated tendon reflexes with patellar clonus in both lower limbs. So we thought spinal cord compression as one of the differential diagnosis along with delayed neuropathy of pyrethroid poisoning. We did MRI of spinal cord to rule out cord compression which was normal. We did nerve conduction study to see the pattern of neuropathy, which was in favour of diffuse motor axonal neuropathy. Even central cause of paraplegia was thought as one of the differential diagnosis, which was ruled out by taking CT brain.

We started the patient on steroid (Inj.Methylprednisolone 500mg iv BID for 7 days) in view of suspected cord compression with edema effect along with other supportive measures (IVFluids, Inj ranitidine, and Inj B1B6B12 etc). Patient showed improvement for the above mentioned management, hence steroid was continued until 7th day, then tapered and stopped. We actually do not know whether steroid caused improvement of neurological weakness, though it appears to be. We need further studies to support this finding.

The UMN signs which appeared along with weakness, is difficult to explain. There are case reports, which state about UMN signs in delayed neuropathy of organophosphate poisoning (Lotti *et al* 2005). In general pyrethroid poison cause hyperexcitation and irritability of nervous system. That is why seizure is a conspicuous manifestation of pyrethroid poisoning. Same mechanism may be the cause for UMN signs such as hyperreflexia in this patient.

Conclusion

In conclusion cases of paraplegia with UMN signs needs prompt evaluation for the cause even in patients with history of recent insecticide poisoning. However if no cause is found we can consider delayed neuropathy of poisoning as reported in this case. Through this case we are first time reporting pyrethroid compound induced delayed neuropathy with unusual UMN signs. Pyrethroid induced delayed weakness in this case is a diagnosis of exclusion. We need further case reports to support this observation.

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