Acta Scientific Neurology (ISSN: 2582-1121)

Volume 5 Issue 9 September 2022

Case Report

Delayed Myelopathy and Neuropathy Develop After Organophosphate Poisoning

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Received: June 23, 2022

Published: August 05, 2022

Abstract

We report a case of accidental ingestion of organophosphate (OP) resulting acute OP poisoning shows delayed neuropathy and myelopathy in follow up.

Keywords: Delayed; Myelopathy; Neuropathy; Organophosphate

Introduction

Organophosphates poisoning is very common in rural area of India. It occurs due to accidental or suicidal ingestion of organophosphates compound (like parathion, malathion, chlorpyrifos, diazinon, fenthion). It also occurs through inhalation and absorption through skin. It causes cholinergic, nicotinic and delayed polyneuropathy. We described a case of accidently taken insecticides (organophosphates) presenting with breathlessness, loss of consciousness. Quadriparesis develop as a delayed complication due to myelopathy and neuropathy.

A variety of neurological manifestations of organophosphate poisoning have been reported in the literature; some of these manifestations like encephalopathy. Seizures can occur acutely at the time of exposure. Intermediate syndromes are reported few days after exposure and they manifest as bulbar, proximal and respiratory weakness. Delayed manifestations occur 2-8 weeks after initial exposure and are typically characterized by development of a polyneuropathy often referred to us organophosphate induced delayed neuropathy (OPIDN) [1]. Occurrence of delayed myelopathy following organophosphate exposure is a very rare phenomenon, only few such cases had been reported in the literature till date [2,3]. We report a case of delayed myeloneuropathy following accidental exposure to organophosphate compound.

Case Report

A female patient of aged 28 years was admitted in our Intensive care unit (ICU) with an episode of breathlessness and loss of consciousness. At the time of admission she was unconscious showing vital signs of pulse rate 130 per minute BP 140/90 mm Hg, respiratory rate 18 per minute, Glasgow coma scale was E2V2M4, pin point pupil, plenty of oral secretion coming out of mouth, on chest auscultation bilateral crepts were present. Cardiovascular system found to be normal, on neurological examination deep tendon reflex absent, plantar reflex bilateral extensor. Investigation at time of admission shows normal blood count, liver function test, kidney function test, normal electrolytes, CT, MRI scan, CSF analysis all are normal. Ryle's tube inserted and aspiration was done at that time. Empirically antibiotics were started and bolus atropine of 3 mg, followed by infusion at the rate of 5mg/hr and the dose was

titrated as per her clinical response and signs of atropinisation. Atropine treatment response was good after 5 days. Arterial blood gas (ABG), Serum procalcitonin (PCT) at the time of admission was normal. On 6th day ET aspirate shows the growth of A. baumanii, after getting AST (antibiotic sensitivity test), antibiotics based on AST was started. No significant improvement in power in all the limbs was noted at day 7. GCS 15/15, vitals within normal limit no need of ventilatory support. Vitamin B12, homocystine, CPK levels all are in normal limit. On day 10 methyl prednisolone was also started, patient start improving. After 22 days in ICU patient was discharged from ICU.

Post discharge manifestations

10 days after discharge from the hospital patient shows weakness and numbness in both upper and lower limb. On physical examination patient was fully oriented Glasgow coma scale 15/15. Power in upper limb 5/5 and in lower limb it was 3/5. Deep tendon reflexes were exaggerated while bilateral plantar reflex is extensor. Blood investigation, liver function test, kidney function test all are within normal limit. MRI shows long segment linear area of signal intensity alterations within the cord in ventral aspects extending from C3 to D5 levels. On nerve conduction study findings were as follows.

Sensory NCS

Right and left median and ulnar sensory nerve action potential are of prolonged peak latency, normal amplitude and conduction velocity.

Right and left Sural sensory nerve action potential are of normal peak latency amplitude and conduction velocity.

Motor NCS

Right and left medial compound motor action potentials are of normal distal latency reduced amplitude and conduction velocity.

Right and left Ulnar compound motor action potential is of normal distal latency, amplitude and conduction velocity.

Right and Left Common Peroneal Nerve and Posterior Tibial Nerve compound motor action potential are of normal latency, amplitude and conduction velocity.

So, in this case patient shows both myelopathy and neuropathy in follow up of organophosphorous poisoning case.

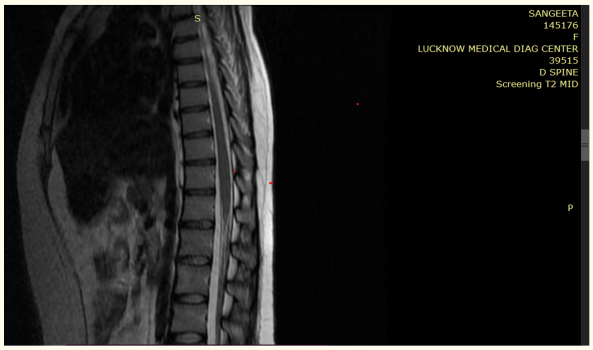

Figure 1: MRI spine.

Discussion

Organophosphates insecticides poisoning is common modes of suicides in developing country. The clinical features of OP poisoning are as follows

Cholinergic symptoms

Increased secretions like salivation, urination, seizures, miosis and bradycardia

Nicotinic symptoms

Fasciculation, muscle weakness, respiratory paralysis. Intermediate syndromes: Develop 3, 4 days after ingestion, characterized by weakness of ocular muscle, neck, bulbar, proximal limb.

Delayed poly neuropathy

This occurs 2, 4 week after ingestion of large doses of organophosphorous poisoning weakness of distal muscles of legs and small muscles of hand. At the onset Electrophysiological changes causes reduced nerve conduction velocity reduced amplitude of muscle action potential and decrease latencies. Neuropathy target esterases initiate neuropathy by inhibiting carboxy esterases.

Reduced levels of plasma cholinesterase confirm diagnosis of OP poisoning and levels decreased till 7 weeks in case of organo phosphorous poisoniong. Ryle's tube aspiration is done. Atropine

given which antagonizes muscarinic receptor mediated action; atropine is given at a dose of 2-3 mg loading dose repeated every 5 to 10 minutes till the signs of atropinization occur.

Organophosphorous poisoning shows delayed neuropathy after 2-3 weeks of ingestion. It occurs due to inhibition of neuropathy target esterases.

Our patient present with delayed neuropathy and myelopathy at 3 weeks after discharge. This was concluded by MRI and nerve conduction studies. Pathogenesis associated with organophosphate poisoning induced delayed myelopathy and neuropathy involves inhibition and phosphorylation of neuropathy target esterases present in brain, peripheral nerves and spinal cord [4].

Ventral motor horn neurons of spinal cord gray matter and dorsal root ganglion shows chromatolysis and neuronal necrosis in chicks exposed to di isopropylflurophosphates (organophosphorous) [5].

In Srilanka females using gingili oil contains tricresyl phosphate shows paralysis of distal limb muscles with subsequent development of pyramidal tract sign showing neuropathy/myelopathy [6].

Conclusion

Organophosphate poisoning shows the symptoms of immediate cholinergic syndrome, which relieves on medication. Delayed manifestation of neuropathy and myelopathy developed as late sequel in follow up of patient. So, we have to think about these rare complications in organophosphorous poisoning cases.

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