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Case Report

Delayed Neurological Manifestations Seen After Organophosphate Poisoning

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Abstract

Introduction: The incidence of organophosphate poisoning is very common in developing countries like india. As suicidal or accidental ingestion of organophosphorous poisoning case are seen in emergency department. And we also face some neurological manifestations like neuropathy and myelopathy in follow up of these patients.

Case Presentation: A Female patient of 28 years ingest organophosphate accidently. Managed conservatively in icu on ventilator and discharge. But in follow up after 10 days of discharge after recovery from organophosphorous poisoning had power in upper limb 5/5 and in lower limb 3/5. Deep tendon reflexes were exaggerated while bilateral plantar reflex is extensor. 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 velocity also shows abnormal pattern. So we manifestated myelopathy And neuropathy like neurological manifestations.

Conclusions: We observe neurological manifestations in follow up of the Patient which were admitted in intensive care as a case of organophosphate poisoning.

Keywords: Organophosphorous Poisoning; Myelopathy; Neuropathy

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 5 mg/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.

After consultation with neurologist methyl prednisolone was started with initial dose of 10 mg once a day, thereafter the dose was increased to 20 mg. along with Vitamin B12 1000 µg day[1]. Limb physiotherapy and nutritional supplement including protein rich diet was advised. Family was counseled for long term out come and patient was in regularly visited in follow-up CCU clinic.

Figure of MRI spine

Figure 1

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.

Reduced levels of plasma cholinesterase confirm diagnosis of OP poisoning and levels decreased till 7 weeks in case of organo phosphorous poisoning. 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]. Neuropathy target esterases also known as patatin-like phospholipase domain containing protein 6 (PNPLA 6) which is located on human chromosome 19p13.2. Neuropathy target esterase is a serine hydrolases with phospholipase B activity. It is a protein appears to involved in axonal maintenance in neuron. One theory proposed that by inhibiting only the catalytic site of neuropathy target esterase does not cause organophosphate induced neurological manifestation for it chemical modification of protein is also required. So it results from combine loss of physiological function and pathological function of neuropathy target esterase to produces organophosphate poisoning induced delayed myelopathy and neuropathy.

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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