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Case Report

Guillain Barre Syndrome Presenting as Delayed Complication of Organophosphorus Poisoning

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Abstract

Organophosphorus (OP) poisoning is an important cause of suicidal poisoning in India. It is known to cause acute, intermediate and delayed complications depending upon the duration between the exposure to OP compounds and onset of symptoms. Delayed complications include intermediate syndrome, peripheral neuropathy named as organophosphates induced delayed neuropathy (OPIDN) or chronic organophosphorus induced neuropsychiatric disease (COPIND). We present here a case of late complication of OP poisoning- a demyelinating neuropathy, Guillain-Barre syndrome which was distinct from usual presentation of OPIDN.

Keywords: *GBS; Intermediate Syndrome; OPIDN*

Introduction

Organophosphorus (OP) compounds are commonly used pesticides globally [1]. India being predominantly agricultural country, easy availability of these compounds and less cost make OP compounds very commonly used poisons with suicidal intent. It is known to cause acute and delayed complications. These manifestation occur at specific time-line [2]. Delayed complications of OP compounds are intermediate syndrome, organophosphates induced delayed neuropathy (OPIDN) or chronic organophosphorus induced neuropsychiatric disease (COPIND). We present here a case of one of the delayed complications of OP poisoning.

Case Report

38 years male, farmer by occupation, non-diabetic, non-hypertensive, chronic alcoholic and tobacco chewer, was admitted with history of alleged consumption of Organophosphorus compound [Chlorpyrifos]. On examination patient was conscious, oriented. He had bradycardia, fasciculations and pinpoint pupils. His rest general and systemic examination revealed no abnormality. Investigations revealed significantly decreased serum cholinesterase levels (342 U/L) at the day of admission and rest all other investigations including Complete Blood Count, Liver Function Tests and Kidney Function Tests were normal. He was treated under standard lines with Gastric lavage, atropine infusion and pralidoxime. He required atropine for almost 10 days. He was discharged on 14th day.

Seven days later patient again presented with complains of sudden onset weakness in lower limbs which ascended in upper limbs in next 48 hours. There was no e/o bladder/bowel involvement. Detailed neurological examination revealed normal nutrition, hypotonia and weakness in all 4 limbs. Power was grade 2 in all the muscles of upper limb proximally and grade 4 distally and grade 1 in all the

muscles of lower limb proximally and grade 3 in muscles of ankle joints. There was no e/o foot drop or wrist drop. Weakness was more pronounced in proximal muscles as compared to distal muscles. He had generalised areflexia and flexor plantar reflex. There was no involvement of respiratory muscles. Sensory system examination revealed no abnormality.

Repeat cholinesterase levels were 2120 U/L which were on lower side of normal but improved from previous report and rest all blood investigations were normal.

Electromyography/Nerve conduction study (EMG/NCV) was suggestive of acute axonal motor polyneuropathy with sensory conduction minimally affected. There was e/o conduction block and prolonged latencies, decreased conduction velocities, and prolonged F response s/o demyelination. CSF study revealed acellular fluid with raised CSF proteins (275.5 mg/dl) which was highly elevated suggestive of albumino-cytological dissociation. Considering rapid onset and progression, predominant proximal involvement, albumino-cytological dissociation and EMG/NCV findings possibility of GBS was kept and plasmapheresis was initiated. There was objective improvement in the power of upper limbs and deep tendon reflexes of upper limbs showed up after 5th cycle of plasmapheresis. In total 7 cycles of plasmapheresis were given and after this there was significant improvement in the power. It was grade 4 in upper limb and grade 3 in lower limb. Deep tendon reflexes of upper limb appeared but were depressed and that of lower limb only knee was present bilaterally. Patient was discharged on 10th day. He came for follow-up after 15 days and further recovery was noted on his upper as well as lower limbs.

Discussion

In India, OP compounds are one of the most commonly used agents for suicidal poisoning. These are cheap and easily available pesticide. OP poisoning have many neurological manifestations which can be acute, intermediate or delayed [3-5]. All these manifestations occur at a very specific time period [6]. Acute neurological symptoms occur immediately after poisoning and have predominantly cholinergic symptoms which is referred as cholinergic syndrome or type I paralysis. Intermediate syndrome or Type II paralysis presents in 1 - 4 days after history of ingestion of poison which presents as neck muscles weakness and extraocular muscle weakness with involvement of cranial nerves. It occurs because neuro-muscular dysfunction. OPIDN or type III paralysis usually occurs 1 - 3 weeks following exposure. It is subacute onset and progresses slowly over couple of weeks. It starts with tingling sensation in lower limbs and calf pain followed by distal weakness to start with in all 4 extremities which may progress proximally [6]. In contrast to this usual presentation, our patient also had delayed complication that occurred 3 weeks after exposure to OP compound. But it had acute onset, rapid progression, predominantly proximal involvement and generalised areflexia. Even Cerebro spinal Fluid examination and EMG/NCS features are consistent with GBS. Organophosphate-induced delayed polyneuropathy is considered to be due to inhibition of an enzyme called neuropathy target esterase although exact ethology is not known.

Conclusion

To conclude, GBS is a rare presentation of OP poisoning. It is important to recognise GBS because with plasmapheresis or IV immunoglobulin, there are fair chances of recovery of this delayed complication which is associated with significant morbidity and mortality.

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