Guillain-Barre Syndrome: A Rare Complication of Organophosphate Poisoning

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ABSTRACT

Organophosphate poisoning is common following accidental or suicidal ingestion. Cases have been reported with different neurological consequences including acute cholinergic excess, intermediate syndrome (IMS), organophosphate-induced delayed neuropathy (OPIND), and organophosphate-induced chronic neuropsychiatric disorder (COPIND). Cases of Guillain-Barre syndrome (GBS) have also been reported as a consequence of delayed toxic effects of organophosphate poisoning. Here, we report a case of a 17-year male with accidental organophosphate ingestion, who developed acute onset of neuropathy and subsequently was diagnosed as GBS. The patient was treated with plasmapheresis and recovered successfully.

Key Words: *Guillain-barre syndrome, Organophosphate poisoning, Plasmapheresis*.

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INTRODUCTION

Organophosphate poisoning is frequently seen in clinical practice. In this part of the word, self-poisoning with organophosphate insecticides is a common clinical scenario.[1] Toxicity of organophosphate is being noted in farm workers, using it as insecticide and defoliant[2], and in factory workers where this chemical is prepared. It has many neurological complications including acute and delayed onset neuropathies.[3,4] Guillain-Barre syndrome (GBS) is a rare complication of organophosphate poisoning.[5] Here, we report a case of accidental organophosphate (Typhon) ingestion. The patient developed flaccid paralysis with areflexia, and was diagnosed as GBS. He was successfully treated with plasmapheresis. Though rare, GBS should be suspected in acute settings of organophosphate poisoning as early recognition and timely management may prevent morbidity and mortality associated with this treatable condition.

CASE REPORT

A 17-year male, student of class 9, with no known comorbids, presented through Emergency Department (ED) with accidental ingestion of insecticide (Typhon) two hours ago.

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Following chemical ingestion, he had two episodes of vomiting containing food particles taken at the last meal. It was non bilious and had no blood in it. Vomiting was associated with retching. There was no history of loose motions, excessive urination, salivation, lacrimation, tremor, drowsiness or seizure. On examination, the Glasgow coma scale (GCS) was 15/15. Pupils were 2 mm in size with bilaterally equal reaction to light. His pulse was 120 beats/minute, blood pressure (BP) 120/70 mmHg, and respiratory rate 16 breaths/minute. On systemic examination, the abdomen was soft and non-tender with exaggerated gut sounds. Chest was clear and cardiovascular examination was unremarkable. On the initial assessment of motor system, power was 5/5 in all muscles of both upper and lower limbs. Deep tendon reflexes were 1+ over all joints of both upper and lower limbs and bilateral down-going planters. Gastric lavage was done in ED and red blood cell (RBC) cholinesterase levels were <800 U/L. He was admitted in the intensive care unit (ICU) and treatment with pralidoxime and atropine was started. The next day, he complained of inability to move his lower limbs while upper limbs, were mobile. On examination, there was bilateral symmetric lower motor neuron type weakness in both lower limbs with power reduced to 3/5 in all muscle groups of both lower limbs and diminished deep tendon reflexes (even on reinforcement) over all joints of lower limbs. Upper limb examination showed weak hand grip, while reflexes were elicitable on reinforcement. The next day, weakness ascended to involve upper limbs bilaterally along with worsening of weakness in both lower limbs. The examination revealed power of 2/5 in all muscle groups of all 4 limbs with absent deep tendon reflexes. The nerve conduction studies (NCS) showed normal right median nerve amplitude, distal latency, conduction velocity, and F-wave latency. The left

median motor nerve showed low amplitude, normal distal latency, normal conduction velocity, and F-wave latency. Both sided ulnar motor nerves had low amplitude, normal distal latencies, normal conduction velocities, and normal F-wave latency. The left peroneal nerve had low amplitude, normal distal latency, normal conduction velocities, and normal F-wave latency. The left posterior tibial nerve had normal amplitude, normal distal latency and normal conduction velocity, and normal F-wave latency. Bilateral sural, ulnar sensory nerves and median sensory nerves had normal amplitude, peak latencies and conduction velocities. NCS suggested motor axonal generalised neuropathy with no sensory involvement.

Cerebrospinal fluid (CSF) examination showed albumin-cytologic dissociation (Table I). Blood sugar was 176 mg/dl at the time of CSF sampling. A diagnosis of GBS was made and treatment started with plasmapheresis. Improvement in powers of all limbs with return of reflexes was noted following 3rd session of plasmapheresis. On further sessions, the patient recovered successfully with the return of compete power and reflexes in all muscles groups of all four limbs. He was walking on the follow-up visit.

Table I: Cerebrospinal fluid analysis.

Tests	Result
Sugar	95 mg/dl
Protein	764 mg/dl
WBC/µL	2
RBC/µL	1780
Color	Reddish
Appearance	Turbid

DISCUSSION

Organophosphate poisoning has signs and symptoms consistent with cholinergic excess in acute settings with multi-organ involvement. Different neurological complications have been reported in association with organophosphate poisoning including acute, intermediate and delayed neuropathies and polyneuropathies. Each complication is noted at specific time following poisoning. Therefore, accurate observation of neurological sequelae in terms of time (when it develops first following poisoning) and pattern of muscular weakness is crucial for correct diagnosis and timely management. Intermediate syndrome develops in 1-4 days of chemical ingestion and is characterised by proximal muscles weakness. It also affects neck muscles, eye muscles and cranial nerves, but no sensory involvement is seen. Other complication of organophosphate poisoning is organophosphate-induced delayed neuropathy/polyneuropathy (OPIND),[3] noted to develop in 2-3 weeks after the poisoning.

It causes distal muscle weakness in the setting of polyneuropathy; while it also affects central nervous system in the setting of neuropathy. Sensory involvement may occur.[6] Some patients may experience cramping muscle pain in the lower limbs, distal numbness and paresthesias, followed by progressive weakness, depression of deep tendon reflexes in the lower limbs and, in severe cases, in the upper limbs.[7] Electrophysiological pattern for this complication shows axonal polyneuropathy/neuropathy. Pyramidal tract dysfunction can also be observed.[4]

Another pattern of nervous system involvement is chronic organophosphate poisoning-induced neuropsychiatric disorder (COPIND).[3] This disorder is rarely seen. The mechanism for this complication is not fully established, but data suggest that exposure to chemical in large toxic doses cause acute necrotic neuronal cell death in the brain; whereas, sublethal doses result in apoptotic neuronal death resulting in neuropsychiatric effects associated with organophosphate poisoning.[8] Timeframe for COPIND is 4 to 14 days.

GBS, following ingestion of organophosphate, is very rarely seen.[5] Pathogenesis is supposed to be a direct toxic effect of the poison on nerves. It is a neurological syndrome characterised by inflammatory polyradiculoneuropathy. It causes acute-onset lower motor neuron type of ascending paralysis with symmetric involvement of both lower limbs and diminished reflexes, which ascends gradually to affect upper body musculature including upper limbs, respiratory and cardiac muscles. Though it is supposed to be demyelinating disease of peripheral nerves, many variants have been identified. It is a potentially treatable cause of acute flaccid paralysis,[9] which if not treated timely, can result in mortality secondary to respiratory muscle and cardiac muscles involvement and morbidity due to residual limb weakness. Early recognition and treatment of GBS are crucial for a better long-term prognosis.

In our case, we suspected GBS on clinical grounds and performed NCS and CSF analyses, which were consistent with the clinical diagnosis. Plasmapheresis was started and the patient responded well with complete recovery.

CONFLICT OF INTEREST:
The authors declared no conflict of interest.

PATIENT'S CONSENT:
Informed consent was taken from parents of the patient regarding the publication of the case report.

AUTHOR'S CONTRIBUTION:
SB: Written the original paper including the whole case report, introduction and discussion.
SK: Helped in literature search and case writing.
IA: Reviewed the case and made important corrections in the presentation.

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