Guillain-Barre Syndrome Due to Organophosphate Compound Poison

D Rajasekaran*, G Subbaraghavalu**, P Jayapandian***

Abstract
Acute manifestations of Organophosphate Compound (OPC) poison are due to effect cholinergic excess. Others are intermediate syndrome [IMS], organophosphate induced delayed neuropathy [OPIND]9, chronic organophosphate induced neuropsychiatric disorder [COPIND].5,6 All these manifestation have specific period of occurrence and duration. There are very sparse reports of toxic demyelination due to OPC poisoning. We report a case of Guillain-Barre Syndrome (GBS) due to toxic demyelination following OPC poison.

Introduction
The spectrum of manifestations with OPC are due to excess cholinergic activity in acute toxicity, intermediate syndrome [IMS], organophosphate induced delayed neuropathy [OPIND]9, chronic organophosphate induced neuropsychiatric disorder [COPIND].5,6 Each manifestations have specific period of occurrence. Delayed manifestations of OPC poisoning are IMS or OPIND. But very rarely it may as acute demyelinating polyneuropathy which can occur alone without any other manifestations.

Case
A 25 years old male was admitted in toxicology ward with OPC poison [chlorpyrifos]. He was treated with stomach wash, atropine, pralidoxime, 2 gm iv bolus followed by 500 mg hourly infusion was given for 6 days. He developed respiratory difficulty and was put on ventilator for respiratory support.

Examination of the patient revealed, conscious, oriented, afebrile with stable vitals. His respiratory examination revealed bilateral wheeze and crepitations. All other system examinations were normal. His biochemical values were normal including arterial blood gas analysis, His ECG. Chest X-ray, were normal except serum cholinesterase level which was very low first three days which increased gradually and was normal when the patient was transferred to the ward (from 126 on day one to 10958 before transfer). He was treated with antibiotics, ventilator, supportive measures and monitoring. He was successfully weaned from ventilator without any complications after 16 days of OPC ingestion and was transferred to medical ward for tracheotomy care.

On 26th day of OPC ingestion, he developed acute onset of weakness of lower limbs which ascended up in over 24-48 hours. His neurological examinations revealed a hypotonic weakness of all four limbs with normal plantar response and absent deep tendon reflexes [DTR]. Repeat biochemical values including creatinine phosphokinase were normal. CSF study showed, acellur smear, suger-45 mgs, protein-2.6 gms which was highly elevated, indicating a gross albmino-cytological dissociation. Repeat CSF study showed the same values. His nerve conduction study showed, an axonal and demyelinating neuropathy of both limbs, the features which were indicative of GBS. He was treated with pulse methyl prenisolone 1gm followed by plasmapheresis. His motor weakness improved over 10 to 15 days and was discharged. He is on regular follow up with us and has fully recovered.

Discussion
In OPC poison, there is spectrum of delayed neurological manifestations like IMS,OPIND,9 COPIND.5,6 Each one has specific features and period of occurrence from ingestion of OPC. IMS present in 1-4 days after ingestion with weakness of proximal muscles, neck muscles, extracranial muscles and involvement of cranial nerve. There is no sensory involvement. Possible mechanisms are prolonged downregulation of Ach receptors, inadequate oxime therapy and reversible subclinical changes in Neuro Muscular Junction. EMG shows a tetanic fade.

OPIND, manifests 2-3 weeks after ingestion of OPC as weakness of distal muscle, with or without sensory involvement. Phosphorylation or aging of neuropathy target esterase is postulated mechanisms.EMG shows denervation potenciation. COPIND may occur between 4-40 days as extra pyramidal and psychiatric manifestations. Possible mechanism is inhibition of cholinergic neurons in striatum.

Clinical features and investigations of our patient strongly indicated that GBS that he had manifested as a sequale of OPC poisoning which is possibly toxin induced delayed demyelination.

To the best of our knowledge, there are very few case reports of GBS in sequale of OPC poisoning. Hence we report this case.

References

*Prof and Chief of Internal Medicine, **Assistant Professor, ***Post graduate, Institute of Internal Medicine, Madras Medical College, Chennai – 600 003.

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