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Case Report

A complicated case of delayed onset intermediate syndrome post organophosphorus poisoning: A rare case report

Navtej Singh[1], Tarun Arora[1], Ankit Chamoli[1]

[1]Department of General Medicine, Bhagat Phool Singh Government Medical College for Women, Khanpur Kalan, Haryana, India.

ABSTRACT

Herein, we present a case of 44-year-old male who landed in our emergency department with altered mental status with difficulty in breathing after 14 days of consumption of chlorpyrifos. Organophosphorus (OP) poisoning is one of the most common causes of poisoning encountered in rural India. There are reported cases of its acute and sub-acute atypical presentation but very few of delayed onset intermediate syndrome. By means of this case report, we want to emphasize on the delayed complications post OP poisoning and importance of treating physician's awareness to recognize and treat such complications.

Keywords: Intermediate syndrome, Organophosphorus, Poisoning

INTRODUCTION

Organophosphorus (OP) pesticides are widely used in agriculture. These are the derivatives of phosphoric acid and not uncommonly used in suicidal attempts because of easy availability and wide spread use.[1] OP causes inhibition of Acetylcholinesterase (AchE) leading to the excessive accumulation of acetylcholine resulting in cholinergic signs and symptom.[2] Three clinical phases of OP poisoning are, (i) early cholinergic crisis, (ii) the intermediate syndrome (IMS), and (iii) organophosphorus induced delayed polyneuropathy.[3] The IMS is rare and commonly underdiagnosed.[4] This syndrome usually occurs 24–96 h following OP poisoning and presents with weakness of different muscle groups.[5] We report one such complicated case of delayed onset rather than usual IMS of OP poisoning.

CASE REPORT

A 44-year-old male presented to us in emergency department (ED) in altered sensorium with oxygen saturation 55% in room air, blood pressure 60/40 mm of Hg, pulse rate 80/min. Patient was immediately intubated with an endotracheal tube and put on mechanical ventilator. The oxygen saturation was raised to 99% post intubation; air entry was bilateral equal without any added breath sounds. One liter of intravenous fluid was rushed as initial resuscitation with improved blood pressure transiently to 110/60 mm of Hg. On examination pulse was regular, pupils were bilateral pin point, GCS $E_2V_TM_2$. Routine blood investigations, ECG, and urgent CT brain were done. Rest of the examination was unremarkable. No history of fever, abnormal body movements, head trauma, and vomiting was present. However, patient's attendants gave history of consumption of chlorpyrifos by the patient 14 days back as a suicide attempt for which he was admitted in nearby hospital and improved symptomatically and was discharged after 5 days to only return in our ED. Hence, provisional diagnosis of delayed onset IMS was made. For further management patient was shifted to the intensive care unit. Investigations showed blood sugar 166 mg%, blood urea 32%, normal serum electrolytes, and normal blood counts. Chest X-ray and CT brain were normal. ECG showed sinus tachycardia and non-specific ST-T changes. Patient was started on atropine, pralidoxime, and supportive management. Meanwhile patient developed hypotension during hospitalization and was started on inotropic support but hypotension did not respond. Despite all attempts patient's clinically condition did not improve and expired during ICU stay.

DISCUSSION

Every year pesticides are responsible for around 1–5 million cases of poisoning.[6] Among these OP poisoning is the most common one. It commonly occurs through ingestion, inhalation and skin absorption.[7] The very first description

***Corresponding author:** Ankit Chamoli, Department of General Medicine, Bhagat Phool Singh Government Medical College for Women, Khanpur Kalan, Haryana, India. ankitchamoli93@gmail.com

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of IMS was given by Wadia *et al.*[8] It occurs between acute cholinergic crisis and late polyneuropathy phase. In IMS there is hyperstimulation by excessive acetylcholine which results in neuromuscular paralysis and its effect may last up to many 18 days.[9] The different muscle groups involved in it are neck, limbs, bulbar, and respiratory. This syndrome causes death due to respiratory tract infections, pulmonary edema, airway obstruction, and respiratory failure. This patient developed respiratory failure 2 weeks after ingestion of OP which is quite late than its typical presentation. It is a very important complication which leads to morbidity and mortality. Delayed onset IMS might be because of larger consumption and longer duration of storage of OP in the patient's body. Longer duration is required for recovery as OP binds irreversibly to AchE.[10] If respiratory failure occurs, mechanical ventilatory support is required with other supportive measures. Role of oximes is still doubtful although used extensively in acute and delayed complications of OP poisoning. The reason for reporting this case is this uncommon complication which could be easily missed and might go undiagnosed. Early identification and intervention could decrease morbidity and mortality.

CONCLUSION

One of the major clinical problems faced in rural India is these poisoning cases which when complicated pose a challenge for diagnosis and management. Neurological complication of OP poisoning is not so common therefore proper follow-up of patient is required to identify such complications and treat accordingly. We all should be aware of these syndromes and their unusual presentation. Supportive measures are the mainstay of these complication.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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