Case Report

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Internal Medicine Section

Case Report: Organophosphorus Poisoning-induced Intermediate Extrapyramidal Syndrome

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ABSTRACT

Organophosphate (OP) compounds are one of the most common agents used for deliberate self-harm in developing countries, including India. OP compounds inhibit the enzyme acetylcholinesterase, which is responsible for hydrolysing the neurotransmitter acetylcholine in both the central and Peripheral Nervous Systems (PNS). Acute cholinergic crises are the most common clinical presentation of OP poisoning. While physicians mostly encounter acute and delayed complications, there have been rare documented cases in the litreature of an intermediate syndrome presenting with extrapyramidal symptoms such as tremors, rigidity, and dystonia. In this report, the authors present a case of a 64-year-old male patient who presented to the Emergency Department (ED) with a history of consuming an unknown quantity of OP-Phorate (11.2%). The initial manifestations included vomiting, sweating, difficulty in breathing, and a low level of consciousness. The patient was managed with atropine, pralidoxime, and mechanical ventilation. He was extubated but later reintubated due to respiratory failure. Eventually, a neurological examination revealed rigidity in all four limbs. The patient was diagnosed with an extrapyramidal type of intermediate syndrome and treated with oral amantadine and trihexyphenidyl. After further hospital monitoring, the patient was extubated and discharged with intact neurological function.

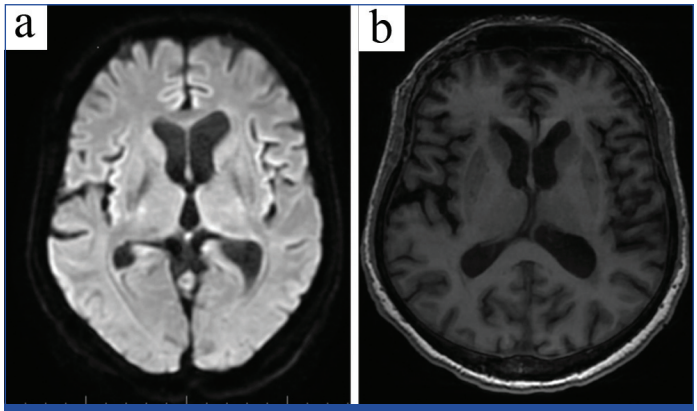
Keywords: Acute pesticide poisoning, Atropine, Extrapyramidal signs, Suicide

CASE REPORT

A 64-year-old elderly male patient presented to the ED with a history of consuming an unknown quantity of OP-phorate (11.2%) with the intent of suicide. After two hours of consumption, initial manifestations included vomiting and sweating, followed by difficulty in breathing and a low level of consciousness. The patient was initially taken to a local hospital where two litres of Ryle's Tube (RT) wash were given, followed by stat doses of atropine and pralidoxime. Cholinesterase levels were measured at 815 U/L (normal range: 4000 U/L-11000 U/L). Due to a low Glasgow Coma Scale (GCS) score (E2V4M5), the patient was intubated and then referred to a tertiary centre for intensive care.

Upon arrival at the ED, the patient had an endotracheal tube in place. His vital signs were as follows: Pulse Rate (PR) of 112 bpm, Blood Pressure (BP) of 124/76 mmHg, SpO_2 of 98%, and Respiratory Rate (RR) of 24 cpm. The patient's GCS score was E2VTM5, and his pupils were bilaterally sluggishly reactive at 3 mm. He was initiated on atropine (12 mg/hr) and pralidoxime (500 mg/hr) infusions and admitted to the emergency Intensive Care Unit (ICU). He remained hemodynamically stable, his sensorium improved, and he was extubated on day 3, eventually transitioning to the Intensive Therapy Unit (ITU). On day 4, the patient became drowsy, developed fasciculations, experienced loose stools, and had respiratory distress characterised by abdomino-thoracic breathing. As a result, he was reintubated and continued on atropine and pralidoxime infusions. On day 6, the patient regained consciousness but was unable to move his upper and lower limbs. On day 7, he developed a fever of 100.4°F, prompting the sending of cultures and initiation of empirical antibiotics.

On day 8, the patient exhibited persistent perioral fasciculations along with poor neck holding. A neurological examination revealed rigidity in all four limbs and poor neck holding. By day 10, the patient displayed characteristic cogwheel rigidity, a mask-like face, and continued fasciculations. The patient was diagnosed with an extrapyramidal type of intermediate syndrome. Magnetic Resonance Imaging (MRI) was conducted to rule out other causes of Extrapyramidal Syndrome (EPS), which yielded normal results [Table/Fig-1]. Alongside supportive care, the treatment regimen was supplemented with oral Amantadine 100 mg twice daily, Trihexypynidine 2 mg three times daily, and Cindopa.



[Table/Fig-1]: Magnetic Resonance Imaging (MRI) of Brain showing normal findings: a) Normal Diffusion Weighted Imaging (DWI); and b) axial T1.

By day 12, the patient's vitals were stable, and there was an improvement in rigidity. On day 13, the patient regained the ability to move all four limbs and hold their neck. Extubation took place on day 15, with normal vital signs and a GCS score of 15/15 during further observation in the Intensive Care Unit (ICU). The patient was then transferred to the ward; however, he developed hoarseness of voice and persistent cough. A video laryngoscopy revealed left vocal cord palsy as a result of prolonged intubation.

To address this, adrenaline nebulisation and oral steroids (prednisolone 1 mg/kg/day) were initiated and tapered over the next two weeks. The patient was monitored in the ward for four days and eventually discharged with complete resolution of rigidity and other EPS features. During a follow-up after two weeks, the patient reported experiencing cough while swallowing liquids but was able to tolerate a solid and semisolid diet. Further evaluation through Fiberoptic laryngoscopy indicated well-compensated left vocal cord palsy, while fiberoptic endoscopy revealed grade 6 aspiration with liquid feeds [1]. As part of rehabilitation, swallow exercise therapy was initiated, and the patient

was advised to gradually introduce soft feeds orally while continuing with Ryle's Tube (RT) feeds.

DISCUSSION

OP pesticide poisoning most commonly occurs from deliberate self-harm, but accidental exposures can also happen in industrial and agricultural settings. Intoxication commonly occurs through oral, inhalational, and transcutaneous routes, and rarely through parenteral routes such as intramuscular, intravenous, or subcutaneous [2]. The acute cholinergic crisis caused by the inhibition of synaptic acetylcholinesterase is a common manifestation of OP poisoning. Intermediate syndrome, delayed neuropathy, and cranial nerve palsies have also been described [3]. Although extrapyramidal symptoms associated with OP poisoning are rare, they were first reported by Joubert J et al., in 1984 [4]. Subsequently, a few more case reports have been published [5-7].

OPs are used as medications, insecticides, and nerve agents. They act by irreversibly binding to the acetylcholinesterase enzyme, preventing the breakdown of acetylcholine. High levels of acetylcholine in the synaptic clefts of the Central Nervous System (CNS) and PNS result in initial excess stimulation and later blockade of synaptic transmission. The onset of clinical manifestations depends on the route of exposure, poison load, chemical nature, and rate of metabolism of the compound [3].

A litreature search revealed a few case reports of extrapyramidal manifestations (such as dystonia, rest tremor, cogwheel rigidity, and choreoathetosis) after acute OP poisoning. Padma DM et al., described a case report of a patient who inhaled the chlorpyrifos compound. The patient initially presented with headache, dizziness, and nausea, but later developed extrapyramidal manifestations on day 8 [5]. Senanayake N and Shanmugaratnam PS reported six patients with extrapyramidal manifestations, including dystonia, tremors, rigidity, and choreoathetosis, following poisoning with the OP insecticide fenthion [6]. Another case report by Sarkar S et al., described a 12-year-old girl with fenthion poisoning and extrapyramidal involvement, such as tremors, rigidity, abnormal muscle tone, and loss of speech. The symptoms improved by day 17 of treatment [8]. A prospective observational study on extrapyramidal symptoms in OP poisoning conducted by Reji KK et al., showed that extrapyramidal features were observed in the second week following OP exposure and resolved in 1-2 weeks. Extrapyramidal features are associated with increased morbidity due to the risk of infections and the duration of ventilation, but they are not associated with mortality [9].

The pathophysiology of extrapyramidal syndrome remains unclear. EPS in OP poisoning may result from an imbalance between dopamine and acetylcholine in the basal ganglia and substantia nigra [10]. The basal ganglia, being rich in neurotransmitters, mitochondria, and blood supply compared to other brain areas, are more susceptible to toxins, metabolic abnormalities, and vascular insults [11]. MRI findings in OP-induced extrapyramidal syndrome may reveal hyperintensities in the basal ganglia or appear normal. Goel D et al., reported a case of a 21-year-old female with insecticide ingestion and extrapyramidal manifestations; the MRI showed symmetrical hyperintensities in the Putamen and Caudate nucleus [12]. Kalyanam B et al., reported a case of a 30-year-old female with insecticide ingestion and extrapyramidal syndrome; the MRI brain showed no abnormalities in the brain parenchyma [13]. Therefore, this case report highlights the importance of raising awareness and knowledge about intermediate syndrome among primary, emergency, and critical care physicians. This awareness facilitates early identification and appropriate management to prevent morbidity and mortality in such patients.

CONCLUSION(S)

Extrapyramidal syndrome is an uncommon manifestation of organophosphorus poisoning. Manifestations such as tremors, dystonia, and rigidity occur during the second week following OP exposure and may be masked by the critical condition of the patient. A precise clinical examination helps in timely diagnosis and management, resulting in a better outcome.

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