ISSN: 1607-8322, e-ISSN: 2220-5799 Anaesthesia, Pain & Intensive Care
Vol 26(4); August 2022 DOI: 10.35975/apic.v26i4.1964

CASE REPORT INTENSIVE CARE

Delayed onset intermediate syndrome after organophosphate poisoning

Ahmed H. Ayoub[1], Mohamed I. Soliman[2], Mohamed A. Hussein[3], Mohammed T. Rageh[4]

Author affiliations:

1. Ahmed Hany Ayoub, MD, Lecturer of Anesthesia and Intensive Care, Ain Shams university, Cairo, Egypt. E-mail: ahmedayoub2610@gmail.com; ORCID: {0000-0001-8691-015X}
2. Mohamed Ibrahim Soliman, MSc, EDAIC, Assistant Lecturer of Anesthesia and Intensive Care, Ain Shams university, Cairo, Egypt. E-mail: dr.dokdok.2020@gmail.com; ORCID: {0000-0002-7674-3949}
3. Mohamed Ahmed Hussein, MSc, EDAIC, Assistant Lecturer of Anesthesia and Intensive Care, Ain Shams university, Cairo, Egypt. E-mail: doctor.mido2000@gmail.com; ORCID: {0000-0002-7957-5354}
4. Mohammed Taher Rageh, MSc, EDAIC, Anesthesia and Intensive Care, Al-Azhar university, Cairo, Egypt. E-mail: mohammedtaher112@gmail.com; ORCID: {0000-0002-7305-6107}

Correspondence: Dr. Ahmed Mahmoud Hany Ayoub, MD; E-mail: ahmedayoub2610@gmail.com; Tel: 00201011354434.

Abstract

Intermediate syndrome is not an uncommon condition, it may occur following exposure to organophosphates, either accidental or suicidal. Picture of intermediate syndrome can develop within 24 to 96 h. We report the case of a 41-year-old male patient, who had a picture of intermediate syndrome, occurring with a delayed onset, than the usual, after exposure to an insecticide solution containing organophosphates. We stress that the patients who have recovered from initial acute cholinergic crisis, should be closely observed for the development of intermediate syndrome.

Abbreviations: ER - emergency room; GCS - Glasgow Coma Scale; ICU - intensive care unit; IMS - Intermediate syndrome

Key words: Adult; Atropine / administration & dosage; Atropine / therapeutic use; Critical Care; Humans; Insecticides / poisoning; Intermediate syndrome; Male; Muscarinic Antagonists / administration & dosage; Muscarinic Antagonists / therapeutic use; Organophosphorus Compounds; Poisoning / diagnosis; Poisoning / drug therapy; Pralidoxime Compounds / administration & dosage; Respiration, Artificial / methods

Citation: Ayoub AH, Soliman MI, Hussein MA, Rageh MT. Delayed onset intermediate syndrome after organophosphate poisoning. Anaesth. pain intensive care 2022;26(4):551-553; **DOI:** 10.35975/apic.v26i4.1964

Received: June 02, 2022; **Reviewed:** July 11,2022; **Accepted:** July 11,2022

1. Introduction

Acute organophosphorus poisoning is an important cause of morbidity and mortality in the developing countries.[1] In acute phase, clinical signs of acute cholinergic crises include excessive salivation, lacrimation and sweating. In addition, a picture of pulmonary edema, bradycardia or tachycardia, muscle weakness with fasciculations, miosis and altered mental status could occur.[2] Intermediate syndrome can occur after 24-96 h of intensive cholinergic crisis. It is characterized by acute respiratory muscle paralysis that may lead to respiratory failure requiring mechanical ventilation. The etiology of intermediate syndrome is unknown. It may be due to prolonged overstimulation of cholinergic receptors leading to dysfunction of the neuromuscular junction and desensitization to pre- and post-synaptic acetylcholine receptors.[3] We report a case who had a delayed presentation of intermediate syndrome after being exposed to an insecticide ingestion.

2. Case report

A 41-year-old patient with no past medical history, was brought to the emergency room (ER). He had been found unconscious with an insecticide bottle beside him.

In ER, his Glasgow Coma Scale (GCS) score was 4/15 (E1M2V1); additionally, frothy secretions were noticed coming from his mouth. His BP was 80/45 mmHg, HR was 120 beats/min and his pupils were pinpoint. Therefore, the patient was immediately intubated, received 3 mg atropine, fluid resuscitation started with 500 ml of normal saline followed by maintenance infusion. Nasogastric tube was passed and gastric wash was started. The patient was shifted directly to the intensive care unit (ICU).

In ICU, the patient was kept sedated, and connected to the mechanical ventilator. Physical examination showed pinpoint pupils and a lot of frothy secretions were noticed coming from the tracheal tube. At that time his BP was 130/70 mmHg and HR was 60 beats/min. His arterial blood gas (ABG) analysis showed severe metabolic acidosis (pH 7.04, PCO_2 4.1 kPa, HCO_3 14 mmol/L, lactate 10 mmol/L). He received another 3 mg atropine and 1 gm pralidoxime; fluid resuscitation was continued. Within one day, the patient's conscious level, arterial blood gases and the heart rate improved. The secretions from the endotracheal tube ceased to flow. Thus, the patient was extubated and commenced on low flow oxygen. The patient remained fully conscious, vitally stable and he was discharged to the ward on the second day. After 5 days, while the patient was in the ward, he developed bilateral upper and lower limb weakness along with difficulty in swallowing. A CT brain was ordered, which showed no acute insult. After 3 more days, he developed disturbance of his conscious level; GCS was 6/15 (E3M1V2), his ABG showed pH 7.1, PCO_2 9.3 kPa, PO_2 6.1 kPa, HCO_3 20.5 mmol/L, and lactate 2 mmol/L. This picture indicated type-2 respiratory failure. Moreover, his pupils were noticed to be pinpoint, and frothy salivation again started. His BP was 120/60 mmHg and HR was 130/min. So, the patient was re-admitted to the ICU, re-intubated and connected to mechanical ventilator. Shortly after intubation, his conscious level improved; however, the patient was quadriplegic and was unable to move his neck. Therefore, another CT brain was repeated which showed no acute insult. Neurology consultant's opinion was requested and he recommended to do a CSF analysis to rule out CNS infection, which was done and showed normal values. Additionally, an MRI scan of the brain and cervical spine were done to rule out any central cause of quadriplegia; all were without any focal lesion. Therefore, the patient was diagnosed as a case of 'intermediate syndrome of organophosphate poisoning'. Subsequently, He received atropine 2 mg IV as a loading dose, then 1 mg/h and pralidoxime 2 gm IV then 1 gm IV every 6 h with targeted HR around 100 beats/min.

Within 2 days from the second ICU admission, the patient's conscious level and muscle power improved. Therefore, he was extubated on the third day. After extubation; the patient was conscious, oriented, vitally stable with good muscle power. He was commenced on atropine 1 mg every 6 h; however, pralidoxime was stopped. 3 days later, the patient was discharged to the ward on the same regimen. He was discharged home after a period of 9 more days.

A follow-up at our OPD found him perfectly healthy.

3. Discussion

Intermediate syndrome (IMS) was reported as a common sequel following organophosphate poisoning,[4,5] which could occur 2-4 days after the initial exposure.[6] It can occur soon after the recovery from acute cholinergic crisis or present with a delayed neuropathy.[7] Clinical manifestations of IMS include muscular weakness mainly of the neck flexors and the proximal limb muscles associated with cranial nerve(s) palsy of variable degrees.[6,8] It could lead to mortality due to respiratory failure, resulting from the respiratory muscles paralysis.[6] IMS results from a neuromuscular junction dysfunction that could be due to cholinergic overstimulation by excess acetylcholine on the nicotinic receptors;[9] although the exact etiology for this condition remain unclear.[10]

In our case, the onset of IMS was late as the patient developed respiratory failure after 9 days of initial presentation with cholinergic crisis (after ingestion of insecticide), and 7 days after ICU discharge, although the patient was free of symptoms on the second day of the initial ICU admission. Alongside with IMS, the patient showed features of relapsing muscarinic manifestations. The management of IMS in the second ICU admission consisted of ventilatory support through mechanical ventilation and appropriate pharmacological treatment with atropine and pralidoxime.

We report this case as it shows that even with initial improvement and adequate management of organophosphate poisoning the patient could develop IMS; and the IMS onset could be delayed beyond the typical range when the risk is wrongly considered to be absent. Such consideration could lead to mortality, if the patient is not properly followed in the medical ward, or if he has been discharged home.

4. Conclusion

Proper follow-up for the organophosphate patients after relieved acute cholinergic crisis symptoms should be considered during hospital stay; and deranged patient orientation or any neurological symptoms taking place after the patient was discharged home, should warn the relatives to seek immediate medical help. We need more studies regarding IMS risk factors, the cause and

predictors of IMS. Perhaps. The IMS could be associated with muscarinic signs.

5. Conflicts of interest

No conflicts of interest declared by the authors.

6. Authors' contribution

AHA: Concept, Manuscript editing and supervision.

MIS, MAH, MTR: Manuscript drafting, software.

7. References

1. Aygun D. Diagnosis in an acute organophosphate poisoning: report of three interesting cases and review of the literature. Eur J Emerg Med. 2004; 11:55-58. [PubMed] DOI: 10.1097/00063110-200402000-00012
2. Aiuto LA, Pavlakis SG, Boxer RA. Life-threatening organophosphate-induced delayed polyneuropathy in a child after accidental chlorpyrifos ingestion. J Pediatr. 1993; 122:658-660. [PubMed] DOI: 10.1016/s0022-3476(05)83560-7
3. Abdollahi M, Karami-Mohajeri S. A comprehensive review on experimental and clinical findings in intermediate syndrome caused by organophosphate poisoning. Toxicol Appl Pharmacol 2012; 258: 309-314. [PubMed] DOI: 10.1016/j.taap.2011.11.014
4. Uprety A, Pantha B, Karki L, Nepal SP, Khadka M. Prevalence of Intermediate Syndrome among Admitted Patients with Organophosphorous Poisoning in a Tertiary Care Hospital. JNMA J Nepal Med Assoc. 2019 Sep-Oct;57(219):340-343. doi: 10.31729/jnma.4569. [PubMed] PMCID: PMC7580432 DOI: 10.31729/jnma.4569
5. Leon SF,Pradilla G, Vesga E. Neurological effects of organophosphorus pesticides. BMJ 1996; 313:690-691. [PubMed] PMCID: PMC2351988 DOI: 10.1136/bmj.313.7058.690c
6. Karalliedde L, Baker D, Marrs TC. Organophosphate-Induced Intermediate Syndrome. Toxicol Rev 2006; 25:1–14. [PubMed] DOI: 10.2165/00139709-200625010-00001
7. Singh S, Sharma N. Neurological syndromes following organophosphate poisoning. Neurol India. 2000 Dec;48(4):308-313. [PubMed] [Free full text]
8. Senanayake N, Karalliedde L. Neurotoxic effects of organophosphorus insecticides. An intermediate syndrome. N Engl J Med. 1987 Mar 26;316(13):761-763. [PubMed] DOI: 10.1056/NEJM198703263161301
9. De Bleecker J, Van den Neucker K, Colardyn F. Intermediate syndrome in organophosphorus poisoning: a prospective study. Crit Care Med. 1993;21:1706–1711. [PubMed] DOI: 10.1097/00003246-199311000-00020
10. Yang CC, Deng JF. Intermediate syndrome following organophosphate insecticide poisoning. J Chin Med Assoc. 2007 Nov;70(11):467-472. doi: 10.1016/S1726-4901(08)70043-1. [PubMed] DOI: 10.1016/S1726-4901(08)70043-1

