AN UNCOMMON PRESENTATION OF OPC (ORGANOPHOSPHATE COMPOUND) POISONED PATIENT WITH VENTILATORY SUPPORT

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Summary

An 18 years old girl with history of ingestion of unquantified amount of insecticide was admitted in the intensive care unit with unconsciousness, hurried respiration. It was diagnosed as a case of organo-phosphate compound poisoning and treated accordingly. After the crisis over, the patient developed respiratory failure. The patient had no fever, no change in X-ray chest or blood count. It is now recognized as a complication of organo-phosphate compound poisoning named Intermediate Syndrome. Manifestations in this particular case is that only the respiratory muscles were involved and repeated respiratory insufficiency developed which was overcome by positive pressure ventilation.

Key words: Intermediate Syndrome; Organo-phosphate; Intoxication, Poisoning.

Introduction

Organo-phosphate compound poisoning is one of the commonest type of poisoning in our country. Intermediate Syndrome is a delayed onset of muscle weakness and paralysis following an episode of acute cholinergic crisis due to organo-phosphate compound poisoning. It is so named because it can occur between 1-4 days after resolution of acute cholinergic crisis and the onset of organo phosphate induced delayed neuropathy which has been reported to occur 2-3 weeks after the resolution of acute toxidrome [l].

Case Report

A female patient, 18 years old, with history of ingestion of un-quantified amount of insecticide only one hour back was admitted in the Intensive Care Unit (lCU). Vomiting had occurred three times after ingestion of insecticide. She was unconscious with hurried respirations. On examination, pulse-96/minute, blood pressure-80/60 mm of Hg, respiratory rate-36/minute, pupil pinpointed bilaterally, Glasgow Coma Score (GCS)-8/15, ronchi all over the chest, oxygen saturation on pulse oxymetry-90%, Arterial Bood Gas (ABG) analysis-severe metabolic acidosis.

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All cloths belonging to the patient were changed. Whole body was sponged with water. Oxygenation was performed through nasal prongs with 3-4 liter oxygen/minute. Gastric lavage was done with three liters of normal saline. Activated charcoal 50 gm was given twice via ryles tube four hourly interval. Dopamine infusion 5-10 µg/kg/minute was used. Intravenous (IV) Sodium Bicarbonate 100 mili equivalent was given. IV Atropne-3 ampoules stat dose and then doubling the each subsequent dose every 15 minutes intervals were given till target end points of Atropinization achieved (After 7 hours of IV Atropine started). Then maintenance dose of Atropine infusion at 3-5 mg/hour was continued. Pralidoxime IV 500 mg 8 hourly daily for 3 days were used. After 48 hours of maintenance dose of Atropine, vital signs of the patient became stable, patient was alert and responding on command. In the mean time, bed side monitor showed gradually lowering of oxygen saturation from 99% to 80%. Arterial Blood Gas (ABG) analysis suggested type-l respiratory failure. Patient complained of difficulty in breathing and sweating over the face and whole over the body. Electrolyte level in blood was normal. Blood count was within normal limit. X-ray chest showed no abnormal findings. Endotracheal intubation quickly was done. Mechanical ventilation ensured on controlled mode (CMV) and continued for 20 hours and then turned into Synchronized Intermittent Mandatory Ventilation (SIMV) as patient's condition permitted. SIMV was maintained for 10 hours. During the period of SIMV mode, self extubation was happened by the patient. As the patient's condition was not deteriorated, we continued oxygenation by face mask. After 9 hours patient became dyspnoec. Endotracheal intubation gently performed and mechanical ventilation ensured on controlled mode and continued for 18 hours. Weaning was tried. CMV was changed into SIMV mode. Mechanical ventilation was made off. Oxygenation was continued via T- piece with oxygen flow 2L/minute for 24 hours. After then, patient's oxygen saturation started to fall and patient's breathing pattern was changed. Artificial ventilation was put in through previously having endotracheal tube and continued for 36 hours.

Afterwards as patient's condition allowed, weaning from ventilation started. The patient kept on T- piece with oxygen flow 2L1minute for next 24 hours. Extubation was done. Patient was on spontaneous breathing. Oxygenation by face mask with oxygen flow 5L/minute for another 24 hours was maintained. During the period of stay in ICU, nutrition was maintained via ryles tube. Next day, patient was transferred to High Dependency Unit (HDU) for monitoring. Finally, after 13 days of intoxication patient was discharged from the hospital with no residual symptoms.

Discussion

The features obvious in the presenting case of Intermediate Syndrome following organo-phosphate compound poisoning are:

i) The only involvement of respiratory muscles.

ii) Respiratory insufficiency developed was overcome by artificial ventilation. But after great pause of normal spontaneous breathing varying from 9 to 24 hours, the patient developed respiratory distress 3 times and at each time by the help of mechanical ventilation distress was relieved.

These are the atypical manifestations observed in our present analysis.

Intermediate Syndrome first termed by Wadia et al. in 1974 as type-II paralysis. This terminology was later changed by Senanayake and Karalliedde in 1987 to Intermediate Syndrome [1,2]. It develops 12-96 hours after organo-phosphate compound poisoning. It reflects a prolonged action of acetylcholine on the nicotinic receptors. The basis of Intermediate Syndrome is that nicotinic transmission requires inhibition of at least 80% of the synaptic acetylcholine esterase unlike the muscarinic synapses and the nerve endings where acetylcholine esterase can be easily inhibited and the syndrome occurs only in severe poisoning. So this syndrome is also known as the nicotinic Syndrome. In this syndrome, characteristically muscles of neck, proximal limbs and the eyes, bulbar and respiratory groups are effected [3]. Cranial nerves palsies are common. The sensory function characteristically remains normal and recovery is evident in 4-8 days. In this case, respiratory muscle groups were affected causing respiratory failure but other groups of muscle have not been at all involved.

In this case report, although confirmatory test like are red blood cell choline esterase level was not determined, but majority of clinical features of complications of organo-phosphate compound intoxication described in literatures were observed [4,5]. Despite a-high incidence, the path physiology that underlies Intermediate Syndrome remains unclear. Previously proposed mechanisms of Intermediate Syndrome include different susceptibility of various cholinergic: receptors, muscle necrosis, prolonged acetylcholine esterase inhibition, inadequate oxime therapy, down regulation or desensitization of post synaptic acetylcholine release and oxidative stress related myopathy [6]. The Pralidoxime used in this case was 500 mg 8 hourly for 3 days. But the required dose should be 1 gm 8 hourly. The inadequate oxime therapy seen in, may be one of the reasons behind Intermediate Syndrome [6]. Oxidative stress related myopathy may be an important factor for inducing respiratory muscles weakness several times [6].

Intermediate Syndrome occurs in 20-60% of patients with severe organo-phosphate compound poisoning [7, 8]. The conclusions derived from salient experimental and clinical studies are that Intermediate Syndrome relates to the severity of poisoning, not the specific organo-phosphate compound and to prolonged inhibition of acetylcholine esterase activity of the erythrocytes, brain and muscle end plate with pre and post synaptic impairment of neuromuscular transmission. Some authors propose that poor regulation of acetylcholine receptors could explain the syndrome and neuro physiological findings [9]. Binding of the drug to the active site of acetylcholine esterase may also result in “ageing”; i. e. the chemical bond between the drug and acetylcholine esterase becomes progressively resistant to deactivators like Pralidoxime. If ageing occurs, the same acetylcholine esterase cannot be reactivated anymore and the physiologic activity at the site will be restored only by the synthesis of new enzyme. This process occurs in both tissue and plasma at varying rates and full restoration may take up to six weeks in untreated patients. The ageing process is variable and related to the type of poison. Some drugs become bound to the acetylcholine esterase within minutes and others take longer. This idea and view correlates with manifestation of repeated respiratory muscle weakness following resolution by mechanical ventilation in our present study.

In another study, the patients who had a low GCS on admission with very low level of acetylcholine esterase indicating severe exposure developed Intermediate Syndrome [10]. If patients survive first 24 hours following severe poisoning, they could go on to develop Intermediate Syndrome and perhaps a centrally mediated phenomenon. It is possible the Intermediate Syndrome is not a single entity, but a continuum in severe poisoning. In the case studied, patient was unconscious, hypotensive, GCS was low-7/15. The manifestation of type-1 respiratory failure after cholinergic crisis was evident and the repetition of same occurrence can be justified by the study [10].

Conclusion

Intermediate Syndrome is an important of organo-phosphate compound poisoning. It should be recognized and treated adequately. It is unclear which patients will develop this condition. Many cases are not diagnosed until significant respiratory insufficiency has occurred. It can be a major cause of organo-phosphate induced morbidity and mortality. Close monitoring of patients with organo-phosphate compound poisoning is the only way to detect Intermediate Syndrome and must be continued after the resolution of respiratory failure too.

Disclosure

All the authors declared no competing interest.

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