CASE STUDY

A 48-Year-Old Man With Severe Shortness of Breath

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Abstract

A 48-year-old man was found by his neighbor unconscious on the floor at his residence. Earlier in the day, his neighbor reported noticing the patient was becoming more short of breath and having some trouble speaking. The neighbor alerted EMS. Upon EMS arrival, the patient was tachypneic with a room air O2 saturation in the 60s. A LifeFlight helicopter responded to transfer this patient to a tertiary care center from his home in a rural farming community.

An unresponsive 48-year-old male farmer was taken by helicopter from his home in a rural farming area to a tertiary care hospital for respiratory distress. He was found unconscious at his residence on the floor by his neighbor. Earlier in the day, his neighbor reported noticing the patient had become more short of breath. The patient also showed signs of speech difficulty, including mumbling of words, followed by drooling. Eventually, he became unconscious, and the neighbor alerted emergency medical services (EMS).

Upon EMS arrival, the patient had profuse vomiting and required constant suctioning. He was tachypneic with a room air oxygen saturation in the 60s. EMS assisted his ventilation with a bag-valve-mask and noted that he was difficult to bag. On physical examination by the flight crew, he was profusely diaphoretic. His vital signs were as follows: heart rate of 113, blood pressure of 251/134, respiratory rate of 20, and oxygen saturation of 88% on 100% O_2. He had rhonchi and wheezed bilaterally. His abdomen was soft and nontender with hyperactive bowel sounds. He was incontinent of urine and stool. He was minimally responsive without localizing signs. The initial blood glucose was 228 mg/dL, and naloxone 2 mg IV push was administered with no significant response. A decision was made to perform rapid sequence intubation using midazolam and succinylcholine. Two intubation attempts were unsuccessful secondary to copious secretions despite suctioning. A bag-valve-mask with an oral airway and 2 rescuer techniques could only maintain oxygen saturations in the 70s. At that point, the flight crew elected to place a King LT-D Airway #5 (King Systems Corporation, Noblesville, IN). Placement was successful with good air exchange, equal chest expansion, and positive end-tidal CO_2 waveform. Oxygen saturations improved. The patient received rocuronium for continued paralysis and midazolam for continued sedation.

Upon arrival to emergency department (ED), the patient's vital signs were as follows: heart rate of 122, blood pressure of 201/131, respiratory rate of 26, end-tidal CO_2 of 35, and oxygen saturation of 94%. The King Airway was switched to an endotracheal tube using the GlideScope (Verathon Inc., Bothell, WA). Propofol infusion was initiated, and fentanyl was given for sedation. Laboratory workup showed electrolytes within normal limits except a potassium level of 2.5, anion gap of 17, and glucose of 263. He has a total bilirubin of 0.5, alkaline phosphatase of 127, alanine transaminase of 18, and aspartate transaminase of 20. A toxicology screen was negative. His white blood cell count was markedly elevated at 37.6. His urinalysis showed no signs of infection. A 12-lead echocardiogram showed sinus tachycardia (Fig. 1). His chest x-ray showed good endotracheal tube placement and mild atelectasis in the left base (Fig. 2). A computed tomographic chest scan was performed, which was negative for pulmonary embolism but showed left lower lobe atelectasis and possible consolidation. A computed tomographic head scan was negative for any acute process.

The patient was admitted to the medical intensive care unit (MICU) for an altered mental status and respiratory failure. In the MICU, he was quickly weaned off the ventilator. After he was extubated, he told the MICU staff that he had been exposed to pesticides that were determined to contain organophosphates (OPs). He was treated with intravenous antibiotics empirically for aspiration pneumonia. He remained afebrile with stable vital signs. He did have several days of minor recurrent left-sided epistaxis that did not require any acute intervention. He was discharged home in a stable condition after a 9-day hospital stay.

Discussion

OP compounds are a group of highly toxic and potent chemicals that are commonly applied in medicine, industry, and agriculture. OP insecticides are the most widely used pest control. Individuals handling these chemicals sometimes overlook using the necessary precautions to protect themselves. The

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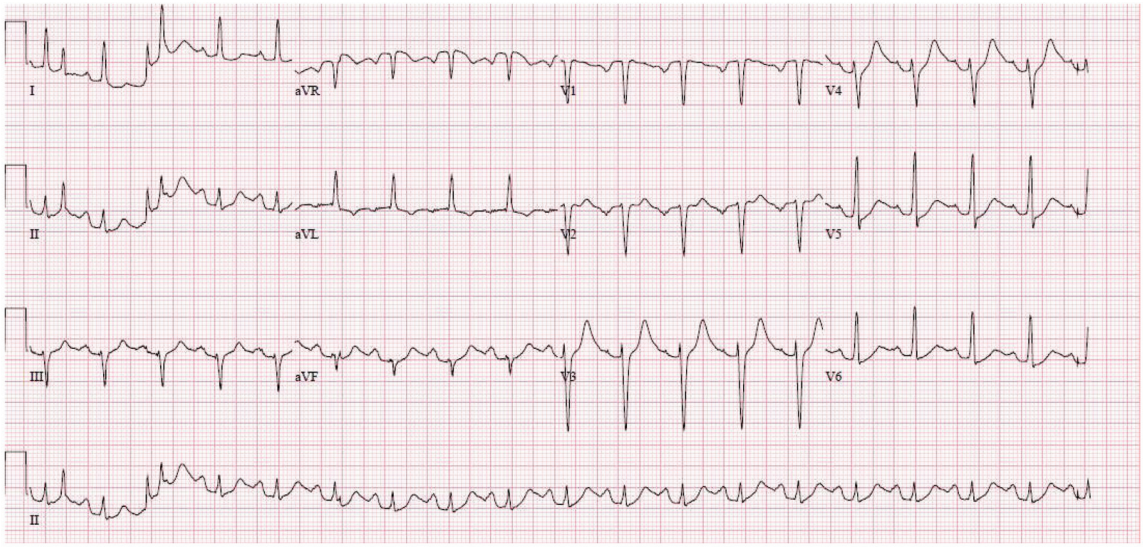
Figure 1. 12-lead EKG



prevalence of intoxication is especially high in developing countries. Clinicians in major metropolitan areas should be aware of its clinical presentation despite its primary usage in agriculture. OPs can also be developed as chemical warfare nerve agents such as soman, sarin, tabun, cyclosarin, and VX.[1] The routes of absorption include ingestion, inhalation, and skin penetration. Poisoning can be accidental or intentional such as suicides by the ingestion of pesticides. Exposure to pesticides is 1 of the most common causes of poisonings worldwide, with about 1 million cases each year resulting in several hundred thousand deaths, most of them in developing countries. Children account for up to 35% of OP-poisoned victims in some areas.[2] Depending on the specific compound, the amount, route, the duration since exposure, and the age and health status of the person exposed, its presentation may vary among patients. OPs disrupt the functioning of the cholinergic nervous system. It irreversibly inhibits acetylcholinesterase, which is involved in the breakdown of acetylcholine in the neuromuscular synapse. Aging, which is the permanent, irreversible binding of OPs to the organ, can occur, but this is highly variable with different agents and can range from minutes to days.[3] The inhibition of acetylcholinesterase by OPs leads to the accumulation of acetylcholine at synapses of the central and peripheral nervous systems and overstimulation of cholinergic receptors above normal physiological limits, precipitating a clinical cholinergic crisis.

Acetylcholinesterase inhibition causes excess acetylcholine and the classic toxidrome at 4 systems: the parasympathetic system (muscarinic), the sympathetic system, skeletal muscle (nicotinic), and the brain. Parasympathetic effects lead to SLUDGE (salivation, lacrimation, urination, defecation, gastrointestinal upset, and emesis) as well as bronchospasm, bronchorrhea, and bradycardia. Sympathetic response is a result of acetylcholine nicotinic receptors in sympathetic ganglia and adrenal medulla. Skeletal muscle effects can cause weakness, muscle fasciculations, and respiratory muscle paralysis leading to respiratory failure. Finally, the effect in the brain can present as delirium, seizures, and confusion. Most acutely poisoned patients are symptomatic within the first 8 hours and nearly all within the first 24 hours. Patients with OP-induced delayed neurotoxicity,[4] which is a neurodegenerative disorder, show a delayed onset of prolonged ataxia and upper motor neuron spasticity. This is a consequence of a single or repeated exposure to organophosphorus esters, which may also occur with fat-soluble compounds from the redistribution of adipose tissue. The neuropathological lesion is a central-peripheral distal axonopathy. It is caused by Wallerian-type degeneration, a chemical transection of the axon, followed by myelin degeneration of distal portions of the long and large-diameter tracts of the central nervous and peripheral nervous systems.[5] Depression leading to suicide can also be a result of its long-term exposure,[6] especially in the rural area.[7]

The diagnosis of OP poisoning is primarily based on history and the presence of a suggestive toxidrome. The onset of clinically significant oral or respiratory intoxication is usually within 3 hours. Meanwhile, dermal absorptions may take up to 12 hours. The laboratory red blood cell cholinesterase assays and reference laboratory testing may not help because of the lack of availability and timing in ED settings. Routine laboratory test abnormalities are mostly nondiagnostic but suggestive. Evidence of pancreatitis marked by hyperamylasemia,[8] hypoglycemia or hyperglycemia, leukocytosis,[9] and abnormal liver function can be detected by laboratory results. In severe cases, a chest radiograph may show pulmonary edema. Atrioventricular blocks, prolongation of QT interval, ventricular dysrhythmias, and torsades de pointes are common echocardiographic findings. A prolonged QTc interval correlates with severity and mortality in severe OP poisoning.