Case Report

A case of organophosphate poisoning with intermediate syndrome and acute pancreatitis – A rare complication

Nidhi Kaeley, Nagasubramanyam Vempalli, Bharat B. Bhardwaj, Biswaranjan Samal

Department of Emergency Medicine, All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India

Abstract

Organophosphate (OP) poisoning is one of the serious occupational hazards worldwide and easily accessible pesticides for suicidal poisoning. It is associated with high mortality and morbidity. OP poisoning is characterized by three main syndromes – cholinergic syndrome, intermediate syndrome and syndrome of delayed polyneuropathy. Other rare complications of OP poisoning are arrhythmias, pancreatitis and hepatic dysfunction. We present 46-year-old male patient with history of OP poisoning, who developed features of intermediate syndrome and pancreatitis. The patient was immediately intubated and managed on mechanical ventilation. Patient was given symptomatic treatment and recovered completely on day 14 of illness.

Keywords: Intermediate syndrome, organophosphate poisoning, pancreatitis

Introduction

Poisoning is one of the serious public health problems worldwide. It ranks 45th in total death in the world. The highest incidence has been reported from exposure of household agents (44.1%) followed by drugs (18.8%) and agricultural pesticides (12.8%). Organophosphate (OP) poisoning is one of the most common causes of poisoning in developing countries like India.[1,2]

Signs and symptoms of OP poisoning are categorized into acute (minutes to 24 hours), delayed (24 hours to 2 weeks) and late (beyond 2 weeks) onset. Acute onset manifestations are caused due to nicotinic and muscarinic receptor action. The muscarinic symptoms are salivation, lacrimation, urination, defecation, gastric cramps, emesis, bradycardia, hypotension, miosis and bronchospasm. The nicotinic symptoms are weakness, fasciculation, cramps and paralysis. The central nervous system symptoms are anxiety, restlessness, convulsions and respiratory depression. The delayed onset nicotinic symptoms are intermediate syndrome whereas muscarinic symptoms are cholinergic symptoms in the form of bradycardia, miosis, and salivation. Coma and extrapyramidal symptoms can occur as delayed onset. The late-onset symptom is peripheral neuropathy.[3] Intermediate syndrome (IMS) usually occurs after 2-4 days after oral exposure of OP poisoning. It is seen in around 20% of the cases of OP poisoning. The characteristic manifestations of IMS are paralysis of muscles of respiration and peripheral limb paralysis. The treatment of IMS involves only mechanical ventilation for 7-15 days and sometimes 21 days.[4] One of the rare complications of OP poisoning is acute pancreatitis.[5] Our patient presented with OP poisoning induced acute pancreatitis and IMS. This case report highlights this rare complication of OP poisoning which if not detected early can increase the risk of mortality.

Address for correspondence: Dr. Nagasubramanyam Vempalli, Department of Emergency Medicine, All India Institute of Medical Sciences, Rishikesh, India.
E-mail: purplehostile@gmail.com

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Case Presentation

A 46-year-old male patient presented to the emergency department of tertiary care hospital of Uttrakhand with alleged history of intake of around 50 ml of organophosphorus insecticide. He had past history of psychiatric illness but was not on any treatment for the same. He had consumed poison around six hours prior to hospitalization. He presented with complaints of altered sensorium, increased sweating and oral secretions. On examination, his GCS was E3V2M6, blood pressure was 110/60 mm Hg, pulse rate was 56 beats per minute and his pupils were pinpoint. Respiratory system examination revealed crepitation in bilateral axillary and mammary region. The rest of his examination was unremarkable. Immediately on arrival, gastric lavage was done. Loading dose of atropine given till clearance of oral respiratory secretions and later on maintenance dose was started. Injection pralidoxime was given as 30 mg/kg of loading dose followed by 10 mg/kg as continuous infusion. On day three of illness, patient developed shortness of breath, difficulty in holding neck and sitting up from supine position. He also complained of abdominal pain in epigastric region associated with three episodes of non-projectile vomiting. The patient was immediately intubated and started on mechanical ventilation.

On examination, his abdomen was soft and tender in the epigastric region with normal bowel sounds. Investigation on day three was suggestive of leukocytosis (total leukocyte count 27206/mm3) with predominant neutrophils (88%), raised lipase (560 Units/L), transaminitis (ALT-380U/L and AST- 198U/L) and normal electrolytes and kidney function tests. An ultrasound abdomen and CT abdomen was suggestive of pancreatitis. [Figures 1 and 2]

A diagnosis of OP poisoning with intermediate syndrome with pancreatitis was made. Nasogastric tube was inserted and patient was kept nil per oral. There was no nasogastric tube aspirate for 24 hours. Thereafter, patient was started on nasogastric tube feeding which he tolerated. Intravenous atropine was gradually tapered. Patient was weaned off from the ventilator over seven days gradually. After extubation, the patient was shifted to general medicine ward where psychiatry consultation was done. He was discharged in stable condition on antidepressants.

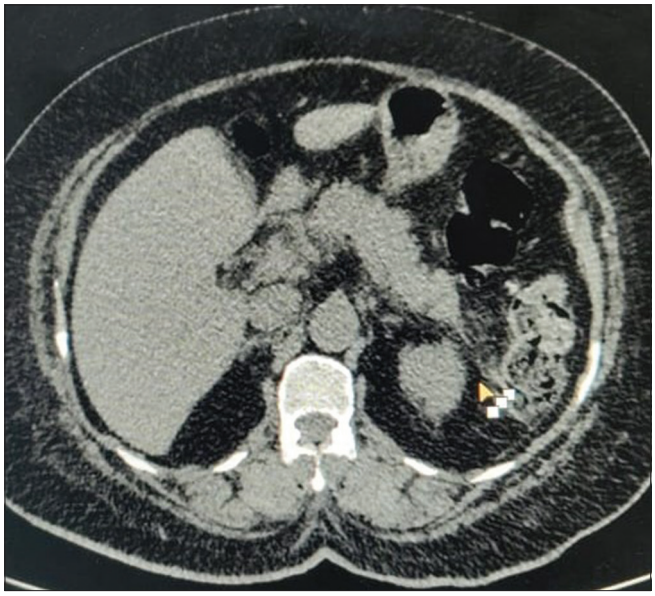

Figure 1: Non contrast CT at the level of pancreas, shows diffusely bulky pancreatic body and tail region with mild peripancreatic fat stranding (Yellow arrow head)

Discussion

OP poisoning is one of the most common poisonings seen in Emergency Medicine departments in developing countries, requiring intensive monitoring and urgent intervention. It presents as diverse symptoms and signs in the form of muscarinic, nicotinic and central nervous system symptoms. SLUDGE (salivation, lacrimation, urination, defecation, gastric cramps and emesis) symptoms are the most common symptoms of OP poisoning. The diagnosis of OP poisoning is mainly done clinically based on nicotinic and muscarinic signs and symptoms. However, one of the screening tools to diagnose OP poisoning is measurement of acetylcholinesterase and butyrylcholinesterase/plasma cholinesterase levels. Acute pancreatitis has been reported in the past in approximately 12% patients with OP poisoning. OP induced pancreatitis is an unusual presentation of poisoning. This manifestation is usually camouflaged by systemic toxicity of the poisoning.[6,7] The suggested underlying mechanism is excessive cholinergic stimulation of the pancreas by OP compounds. The contraction of sphincter of Oddi leads to increased internal pressure inside the pancreatic duct. The excessive acetylcholine occludes the ampulla of Vater and pancreatic duct leading to stimulation of pancreatic acinar cells, causing acute interstitial pancreatitis.[8]

Raised amylase levels not only occur in patients of OP poisoning induced acute pancreatitis but also seen in patients with interstitial

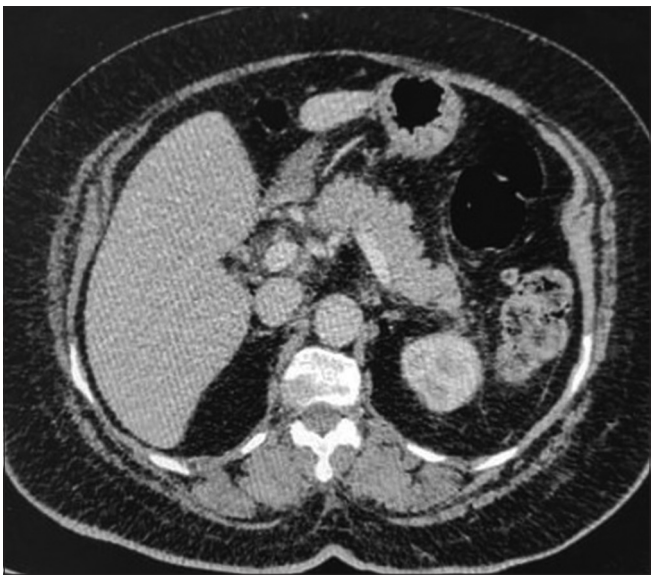

Figure 2: Post contrast imaging shows no obvious areas of non-enhancement within the pancreatic parenchyma

ischemia, enteritis and hypersalivation. According to previous studies, acute pancreatitis is seen in 5.7% to 29% patients with OP poisoning. Thus in cases of OP poisoning, diagnosis of acute pancreatitis should be confirmed by CT scan.[9]

OP poisoning induced pancreatitis often runs a subclinical course, thus not much data is available on the same. Marsh *et al.* have reported a case of 24-year-old farmer who developed OP-induced pancreatitis which persisted for 6 months.[10]

It has been observed that OP induced pancreatitis usually improves within 72 hours whereas complicated pancreatitis improves within 3-5 days.[11] However, our patient recovered within ten days of OP poisoning exposure. S Singh *et al.* did a study from June 2001 to June 2005 to estimate the incidence of hyperamylasemia and acute pancreatitis in patients with OP poisoning. They observed that there was no correlation between the nature of compound, severity of disease and amylase levels. They also concluded that though hyperamylasemia is common in patients with OP poisoning, acute pancreatitis rarely occurs.[6] Another dreaded complication which took place in our patient was intermediate syndrome. It occurs 24-96 hours after the exposure of the OP poisoning. It is characterized by weakness of head and neck, proximal limb weakness and respiratory muscle paralysis. It occurs as a result of excess of acetylcholine at the neuromuscular junction. The treatment involves only mechanical ventilation.[12]

This case report is important from primary physician point of view. Acute pancreatitis is one of the under investigated and underreported complications of organophosphorus poisoning. Thus, amylase, lipase levels should be done in patients with OP poisoning to avoid missing this complication especially in critically ill patients with op poisoning. This will aid the primary as well as the emergency physicians to timely diagnose acute pancreatitis in these patients, thus preventing significant mortality.

Conclusion

OP poisoning is associated with 3-25% mortality rate. Our patient had two serious complications pancreatitis and intermediate syndrome. Mortality rate associated with OP induced acute pancreatitis is around 5-10%. Thus, timely and appropriate management of patients with OP poisoning induced pancreatitis by acute care physicians and primary care physicians can prevent significant mortality and morbidity in these patients.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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