Case Report

Organophosphate Poisoning-Induced Acute Pancreatitis: A Peculiar Case Report

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Organophosphate Poisoning-Induced Acute Pancreatitis: A Peculiar Case Report

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Key clinical message: Acute pancreatitis is an uncommon side effect of organophosphate poisoning that is brought on by the pancreas' stimulation of exocrine secretion and the sphincter of Oddi's constriction. Increased exocrine pancreatic fluid output results in increased pressure within the pancreatic duct, which in turn causes pancreatitis due to organophosphate poisoning.

Abstract: Background: Rarely, organophosphate poisoning can lead to acute pancreatitis. Subclinical signs of acute pancreatitis may be present in organophosphorous poisoning patients. **Case Presentation:** A 41-year-old African male farmer was brought to the emergency room nine hours after it was thought that he had consumed an unknown amount of an organophosphate pesticide poison. He was brought to the clinic at 11:30 p.m. by his neighbors due to a history of semi-consciousness. He was groggy (unable to move or think normally) and had been vomiting progressively. He had a habit of ingesting unknown substances from his wife's house. He was immediately given normal saline for stomach lavage when he arrived. His oxygen saturation rose to 96% after four liters of oxygen were administered to him every minute via a nasal cannula. The dose of atropine was increased to 4 mg/hr and titrated to keep the patient's heart rate between 100 and 140 beats per minute. **Conclusion:** Increased exocrine pancreatic fluid output results in increased pressure within the pancreatic ducts, which in turn causes pancreatitis due to organophosphate poisoning. The most uncommon side effect of organophosphate poisoning is acute pancreatitis.

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1. Introduction

Organophosphate insecticides are commonly used in both domestic and agricultural settings. Poisoning from organophosphates is a concern that can seriously cause morbidity and mortality, particularly in developing countries [1]. Pesticides with organophosphates can cause severe poisoning when used orally. The intoxications caused by organophosphate pesticides can vary in intensity depending on whether they are absorbed through the skin, mucous membranes, conjunctiva, or inhalation [2]. Increased exocrine pancreatic fluid output results in increased pressure within the pancreatic duct, which in turn causes pancreatitis due to organophosphate poisoning. Typically, organophosphate exposure does not cause severe acute pancreatitis [3]. Hypersalivation, stomach discomfort, nausea, vomiting, diarrhea, muscular fasciculations, bradycardia, and hypotension are some of the clinical signs and symptoms of organophosphate poisoning. Seizures, respiratory failure, shock, and death might occur in some instances [4]. An elevated level of serum lipase or amylase is often used to make the diagnosis of acute pancreatitis [5]. The treatment of organophosphate poisoning involves hemodynamic stabilization using intravenous fluid resuscitation, airway control, oxygen supply, a reduction in poison absorption, and removal of the poison from the body [6]. This case study shows the effects of organophosphate poisoning, which led to severe pancreatitis, on an adult man who was damaged by a pesticide that includes them.

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2. Case Presentation

2.1. Patient Information

A 41-year-old African male farmer was brought to the emergency room nine hours after it was thought he had consumed an unknown amount of an organophosphate pesticide poison. He was groggy (unable to move or think normally) and had been vomiting progressively. He had no known co-morbid conditions, such as diabetes mellitus, hypertension, or renal diseases. There was no history of fever, headache, seizures, drug overdose, or use of illegal drugs. Additionally, he had no family history of chronic diseases. He had been on omeprazole for 2 years and had a 2- year history of peptic ulcer disease. He had a habit of ingesting unknown substances from his second wife's house. His second wife told him, "You will never see your other wife, but today you will die here while you see my eye,". She told him, "I will wish you a hellfire life for the cruelty you have done on the earth." He told to his neighbor before admitting all what happened. The patient smelled like garlic, which was a sign that he had been poisoned.

At the time of admission, he exhibited foaming at the lips, rolling of the eyes, incontinence of the urine, increased bronchial secretions, weakness, excruciating stomach pain, vomiting, and loss of feces. He had two wives, and one of them made the decision to have him killed because she envied him when he went to see his other wife. She then sprayed the snack with an organophosphate insecticide called parathion that would poison it. After two hours of eating the snack from his envious wife, he began vomiting and passed out. He was taken to the emergency room at 11:30 p.m. by his neighbors due to a history of semi-consciousness after being poisoned at 9:30 a.m.

2.2. Clinical Findings

His vital signs at the time of his arrival in the emergency room showed that he had a body temperature of 33.8°C, tachypnea with a respiratory rate of 22 breaths per minute, tachycardia with a heart rate of 102 beats per minute, oxygen saturation on room air of 90%, and normal hemodynamic parameters with a blood pressure of 128/62 mmHg (Table 1). His laboratory examinations at the time of his emergency department visit revealed a white blood cell count of 12,140 cells/mm3. The serum amylase level was high at 483 U/l, and serum lipase was at 397 U/l. An increase in white blood cells, serum amylase, and serum lipase are the key diagnostic indicators for acute pancreatitis, an inflammatory sickness with considerable pancreatic damage discovered in a laboratory evaluation. Lab research uncovered inflammation coupled with significant pancreatic dysfunction. Analysis of the arterial blood gases did not show any anomalies.

Table 1. Laboratory tests to detect acute pancreatitis from the time of admission till the time of discharge.

Laboratory investigation	Day 1	Day 2	Day 3	Normal Range
White blood cell count	12,140 cells/mm3	11,040 c/mm3	9,870 cells/mm3	4,500–11,000 cells/mm3
Serum amylase level	483 U/l	386 U/l	142 U/l	40-140 U/l
Serum lipase level	397 U/l	196U/l	158U/l	0-160 U/l

Laboratory investigations of the patient revealed: blood urea nitrogen of 25 mg/dL, potassium of 3.6 mmol/L, sodium of 153 mEq/L, fasting blood glucose of 118 mg/dL, higher density lipoprotein of 63 mg/dL, lower density lipoprotein of 143 mg/dL, triglycerides of 245 mg/dL, aspartate aminotransferase level of 58 units/L, an alanine aminotransferase level of 87 units/L, an erythrocyte sedimentation rate of 16 mm/hour, hemoglobin of 12.5 mg/dL, hematocrit of 45%, and serum creatinine of 1.4 mg/dL, neutrophils of 67%, and lymphocytes of 27% (Table 2).

Table 2. Blood chemistry from the time of admission till the time of discharge.

Blood Chemistry	Day 1	Day 2	Day 3	Normal Range
BUN	25 mg/dL	21 mg/L	19 mg/L	6-20 mg/dL
Potassium	3.6 mmol/L	3.3 mmol/L	4.2 mmol/L	3.6-5.2 mmol/L

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Sodium	153 mEq/L	149 mEq/L	137 mEq/L	135-145 mEq/L
FBS	118 mg/dL	121 mg/dL	109 mg/dL	100-126 mg/dL
HDL	63 mg/dL	61 mg/dL	54 mg/dL	40-60 mg/dL
LDL	143 mg/dL	117 mg/dL	110 mg/dL	100-129 mg/dL
Triglycerides	245 mg/dL	198 mg/dL	134 mg/dL	< 150 mg/dL
AST	58 units/L	43 units/L	36 units/L	0 - 35 units/L
ALT	87 units/L	66 units/L	29 units/L	0 - 35 units/L
ESR	16 mm/hour	19 mm/hour	12 mm/hour	0 - 20 mm/hr
Hemoglobin	12.5 mg/dL	13.7 mg/dL	14.9 mg/dL	13.8-17.2 mg/dL
Hematocrit	45%	42%	39%	41-50%
Serum creatinine	1.4 mg/dL	1.1 mg/dL	0.8 mg/dL	0.7-1.3 mg/dL
Neutrophils	67%	56%	61%	55-70%
Lymphocytes	27%	31%	39%	20-40%

ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; BUN: Blood urea nitrogen; ESR: Erythrocyte sedimentation rate; FBS: Fasting blood glucose; HDL: Higher density lipoprotein; LDL: Lower density lipoprotein.

Upon admission to the emergency room, his physical examination revealed generalized sweating, cold extremities, fecal incontinence, increased muscular tension, spotted lower limbs, and vomitus around the mouth. A sleepy patient with dyspnea, diaphoresis, drooling, and many mouth ulcers was discovered during a medical examination. He had bilaterally contracted pupils that responded slowly to light. Radiography of the chest and electrocardiography were both normal. His chest auscultation revealed bilateral vesicular breath sounds and upper abdominal discomfort. The patient's eye-opening was 1/4 (no eye-opening), the motor response was 3/6 (flexion to pain), and the verbal response was 3/5 (inappropriate words), all according to the Glasgow Coma Scale, which measures the level of consciousness in this patient. With a corrected QT interval of 0.85 seconds, the electrocardiogram (ECG) values were normal (normal value: 0.6–1.2 seconds). Both abdominal computed tomography and abdominal ultrasound found no evidence of pancreatic inflammation or gallstones. By using magnetic resonance imaging, the gallbladder and pancreas were shown to be normal. The results of an endoscopic ultrasonography showed that the bile and pancreatic ducts are not blocked.

2.3. Therapeutic Intervention

As soon as he was admitted, he had stomach lavage and suctioning, as well as rapid decontamination with normal saline. His oxygen saturation rose to 96% when oxygen was administered to him at a rate of four liters per minute via a nasal cannula. On the first day, 10 mg of intravenous diazepam was given to suppress muscular fasciculations. Immediately after arrival, a bolus dose of 8 mg of atropine was administered. The patient's heart rate was maintained between 100 and 140 beats per minute by titrating the atropine dosage to 4 mg/hr.

The patient received 100 mL of 0.9% normal saline solution within one hour, along with 300 mg of pralidoxime three times daily as part of the first course of therapy. After administering a total of 4.3 g of pralidoxime, the dosage was gradually lowered and terminated at the conclusion of the third day. Dialysis was performed on the third day to eliminate dialyzable toxins and lessen severe metabolic abnormalities in order to treat acute pancreatitis. The patient had continuous injections of omeprazole, 40 mg once a day for three days, to treat acute pancreatitis. To relieve the mild discomfort caused by acute pancreatitis, he was given 500 mg of paracetamol orally as needed. The patient made a full recovery in 60 hours after receiving acute pancreatitis therapy.

2.4. Outcome and follow-up

Over the course of the following three days, he displayed a noticeable recovery from organophosphate pesticide poisoning symptoms, with the exception of severe pancreatitis.

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Omeprazole 40 mg was prescribed for the patient, to be taken twice daily for 14 days. He recommended continuing his follow-up as before monthly.

3. **Discussion**

Insecticide poisoning from organophosphates is particularly prevalent in underdeveloped nations [7]. Acetylcholinesterase, which hydrolyzes acetylcholine, is inhibited by organophosphate-poisoning substances. The acetylcholinesterase enzyme is irreversibly inhibited in the usual toxidrome of acute organophosphate poisoning, leading to acetylcholine buildup at muscarinic and nicotinic synapses and causes cholinergic crisis [8]. Organophosphate poisoning can affect the body's ability to maintain a healthy level of blood sugar through a number of different mechanisms, such as physiological stress, oxidative stress, paraoxonase inhibition, nitrosative stress, pancreatitis, inhibition of cholinesterase, stimulation of the adrenal gland, and changes in the liver's tryptophan metabolism [9]. Acute organophosphate poisoning frequently results in cardiac or hemodynamic abnormalities such as hypotension, bradycardia, or tachycardia. These abnormalities can be brought on by a number of mechanisms, including autonomic disturbances (caused by overstimulation of muscarinic and/or nicotinic acetylcholine receptors), the effects of hypovolemia or hypoxia, peripheral vasodilatation, and direct myocardial stimulation [10].

Acute pancreatitis is an uncommon side effect of organophosphate poisoning that is brought on by the pancreas' stimulation of exocrine secretion and the sphincter of Oddi's constriction, which increases the pancreatic duct's internal pressure. Clinically, severe organophosphate poisoning is mostly brought on by respiratory failure. Bronchoconstriction, paralysis of the respiratory muscles, and injury to the medullary respiratory centers are all factors that contribute to respiratory failure [11]. The systemic symptoms of organophosphate poisoning, such as nausea and vomiting, which are also frequent symptoms of acute pancreatitis, may obscure the diagnosis of acute pancreatitis [12]. In this study, the patient developed low urine output, increased bronchial secretions, foaming at the mouth and lacrimation, weakness, excruciating stomach pain, vomiting, drooping eyes, and loose feces.

There are three main symptoms that develop from organophosphate poisoning: (i) Dysregulation of cholinergic transmission: Acute muscarinic, nicotinic, and central nervous system-related poisoning symptoms typically appear one to two hours after exposure [13]. (ii) Intermediate Syndrome: Although it can occur up to a week later, the illness usually manifests itself 1-4 days after exposure to the organophosphate poisoning toxin. It results from neuropathic target esterase inhibition. Neck flexion weakness is the first symptom that develops into respiratory muscle weakness and respiratory failure [14]. (iii) Organophosphate induced delayed neuropathy, commonly known as delayed paralysis syndrome, is a rare delayed consequence of acute organophosphate poisoning. After 10 to 21 days of exposure, a distal ascending neuropathy manifest. Motor sluggishness and paresthesias are frequent [15].

When compared to the three main syndromes caused by organophosphate, the patient, in this case reported having acute cholinergic syndrome, which included muscarinic symptoms like salivation, lacrimation, bronchorrhea, gastric upset, and emesis; nicotinic symptoms like abdominal cramping, weakness, and tachycardia; central nervous system symptoms like confusion and the inability to move or think normally; and intermediate symptoms like muscle weakness, loss of consciousness, and restlessness. The third delayed toxicity, known as organophosphate poisoning induced delayed polyneuropathy, which causes paresthesia or numbness over the legs, ataxia, neuropathy, or respiratory failure, is not present in this case because the patient recovered from organophosphate pesticide poisoning within three days of being admitted.

Excessive cholinergic activation inside the pancreas and ductular hypertension may be the pathogenetic processes of acute pancreatitis [16]. The following criteria are used to determine whether a person has acute organophosphate poisoning: (1) a history of pesticide exposure; (2) distinctive clinical symptoms of organophosphate poisoning; (3) clinical improvement after receiving atropine and oxime; and (4) lowered serum acetylcholinesterase activity [17]. An uncommon side

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effect of compound poisoning with organophosphates is pancreatitis. The pancreatic effects of organophosphates often go away in 72 hours and are gone completely in 4-5 days [18].

The patient in this study recovered fully from acute pancreatitis after two days and twelve hours of therapy, as opposed to 72 hours or 4-5 days, because he did not have any coexisting conditions and because the amount of organophosphate he drank was moderate because he did so with a snack. His quick recovery from pancreatitis and prompt transport to the hospital when it was suspected he had been poisoned was both due to this reason.

Hepatic dysfunction, extrapyramidal characteristics, pancreatitis, and cardiac arrhythmia are a few more documented side effects of organophosphate poisoning. Acute pancreatitis brought on by organophosphate poisoning is an uncommon complication [19]. When a patient exhibits poisoning symptoms after ingesting organophosphate poisoning, it is important to check that they have a patent airway, breathing, and circulation. The medications utilized for specialized treatments include atropine, which has an antimuscarinic action, and pralidoxime, an enzyme reactivator. Acetylcholine is only competitively antagonistic to atropine at muscarinic receptors [20].

3.1. Limitations of the study

Due to the difficulty in obtaining chemicals to assess the severity of organophosphate pesticide poisoning, serum pseudocholinesterase and cholinesterase level laboratory studies were not performed. He consumed an unknown quantity, which made it hard to determine the seriousness and intricacy of his condition when he arrived.

4. Conclusion

Only a small number of cases of severe necrotizing acute pancreatitis caused by organophosphate pesticide poisoning have been documented. The overstimulation of cholinergic receptors caused by acetylcholinesterase inhibition, which results in increased intraductal pressure and excessive pancreatic enzyme release, is thought to be the mechanism of organophosphate poisoning and insecticide-induced pancreatitis. Keeping the airway open and giving cardiac support are the first steps in treating organophosphate poisoning. Gastric lavage and activated charcoal must be used if a hazardous chemical is consumed orally.

4.1. Recommendations

The government should investigate the best ways to prevent poisoning, such as pesticide control and limiting the open sale of organophosphate poison in all stores and requiring that only authorized entities sell it.

Particularly in developing countries, people who work in agricultural fields should get instruction from the government on the dangers of organophosphate poisoning.

The author advised all individuals to refrain from acts of cruelty, such as putting poison in their food or drink or killing someone for problems that might be resolved amicably.

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