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Acute Kidney Injury and Intermediate Syndrome Following Organophosphorus Poisoning: A Case Report

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Abstract

Organophosphate (OP) poisoning is an important cause of acute poisonings in several regions of India and leads to serious complications like cholinergic crisis, respiratory failure, and intermediate syndrome. Renal dysfunction is relatively uncommon and less described in such cases. We describe here a case of a 46-year-old gentleman admitted with severe OP poisoning associated with respiratory failure necessitating mechanical ventilation, intermediate syndrome, hemodynamic instability, rhabdomyolysis, and acute kidney injury (AKI) leading to hemodialysis. The initial presentation was with altered sensorium, hypoxemia, tachycardia, tachypnea, and metabolic acidosis with type 1 respiratory failure. The patient received atropine drip, pralidoxime, and mechanical ventilation. In addition, the patient during his hospital stay had episodes of hypotension necessitating pressor support, cardiopulmonary arrest necessitating resuscitation, and proximal muscle weakness, which is suggestive of intermediate syndrome. Creatinine progressively increased, urine output decreased, creatine phosphokinase (CPK) levels were found to be very high at 38,710 U/L, and there was refractory hyperkalemia requiring intermittent dialysis. AKI in this patient is thought to be multifactorial and could be related to hemodynamic instability, infection, rhabdomyolysis, and toxin-induced nephrotoxicity. Renal function subsequently improved after dialysis. He was also treated for nosocomial infections. He was successfully treated and was closely followed up till full clinical recovery was achieved. This case highlights the importance of monitoring renal function, electrolytes, and markers of muscle injury in patients with severe OP poisoning.

Categories: Other, Internal Medicine, Nephrology
Keywords: acute kidney injury, cholinergic toxicity, hemodialysis, intermediate syndrome, organophosphorus poisoning, rhabdomyolysis

Introduction

The use of organophosphate (OP) pesticides for poisoning remains an important cause of poisoning in India. OPs inhibit the activity of acetylcholinesterase (AChE), resulting in the accumulation of acetylcholine on muscarinic and nicotinic receptors and, consequently, the manifestation of cholinergic symptoms. Complications of poisoning include respiratory failure, cardiovascular effects, intermediate syndrome (IMS), and delayed neurotoxicity [1,2].

IMS is a neurologic condition that occurs due to OP poisoning. This disease usually occurs 24 to 96 hours after the subsidence of the acute cholinergic crisis. IMS is distinct from OP-induced delayed polyneuropathy (OPIDN), which occurs later. IMS consists mainly of muscle weakness involving the proximal muscles and neck flexors, respiratory muscle dysfunction, and dysfunction of motor cranial nerves. IMS has been proposed to occur due to an abnormality in the neuromuscular junction, specifically postsynaptic transmission, though the exact pathophysiology is not yet clear [2,3].

Renal toxicity in association with OP poisoning has also been reported, but there may be various mechanisms for this condition to develop, including direct nephrotoxicity, hemodynamic effects, rhabdomyolysis, oxidative stress, and dehydration [4-6].

The frequency of acute kidney injury (AKI) and IMS among patients suffering from OP poisoning varies across studies. In a nationwide retrospective study among OP poisoned patients, the hazard of AKI was 6.17 times greater than that of the control group [6]. In another study, AKI was observed in 22.3% of 300 OP patients [7]. Regarding the occurrence of IMS, the incidence of IMS was 17.6% in 176 cholinergic insecticide patients [8].

In this case report, we present a case of OP poisoning complicated with IMS, rhabdomyolysis, and AKI with the need for renal replacement therapy. The hospital stay was complicated by infection with carbapenem-resistant non-lactose-fermenting gram-negative bacilli that resulted in an extended hospital stay. With targeted treatment and supportive care, the patient achieved clinical recovery.

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Case Presentation

A 46-year-old man, known hypertensive and diabetic, was brought to the emergency department by his relatives in a semiconscious state approximately two hours after an alleged history of ingestion of an OP pesticide. There was a history of two to three episodes of non-bilious vomiting and diarrhea. An empty Parryfos bottle (Monocrotophos 36% SL) was reportedly found next to him, and his family stated that he had been under the influence of alcohol before the incident. There was no reported history of fever, headache, or seizures. Classic cholinergic signs, including increased secretions, miosis, and diarrhea, were noted.

He was initially taken to a local hospital, where gastric lavage was performed and stat doses of atropine 4 mg and pralidoxime (2 amp) were administered. He was then referred to our hospital for further management. On arrival, his Glasgow Coma Scale score was E2M3V2 (7/15). He was restless and drowsy, with oxygen saturation of 78% on room air, heart rate of 142 beats/minute, and respiratory rate of 32 breaths/minute. Arterial blood gas analysis showed metabolic acidosis with type 1 respiratory failure. In view of depressed consciousness and respiratory failure, he was intubated and mechanically ventilated.

On examination, bilateral conducted breath sounds were present; the remainder of the systemic examination was reported as unremarkable. Initial laboratory investigations showed leukocytosis with a normal renal profile, and the initial chest radiograph had a right-side homogenous opacity. An atropine infusion was started according to the treatment protocol. Empirical antibiotics were administered because aspiration was suspected, after cultures were obtained.

On day 1 of hospitalization, he was responding slowly to the treatment. He was continued on injection atropine infusion and IV antibiotics.

On day 2 of hospitalization, the patient developed sudden hypotension that was not responsive to fluid administration and required escalating vasopressor/inotropic support. Antibiotic therapy escalated to injection meropenem. Repeating renal parameters showed an increase in serum creatinine, although urine output remained adequate during the initial period. Medication doses were adjusted according to renal function.

On day 3, he developed sudden bradycardia followed by cardiopulmonary arrest. Return of circulation was achieved after two cycles of cardiopulmonary resuscitation.

On day 4, muscle power was less than 2/5, with prominent proximal muscle weakness, which was assessed after stopping sedation. Critical illness neuropathy was a confounder. In the clinical context of OP poisoning and the timing of neuromuscular weakness, IMS was diagnosed. Atropine was continued according to the treatment protocol. Subsequently, his sensorium and muscle power improved, but poor neck control was noted, and he could not be weaned off ventilatory support.

On day 8, a serial rise in potassium levels was noted, and he was started on round-the-clock potassium-binding resins, injection calcium gluconate for myocardial membrane stabilization, and salbutamol nebulization. Persistent rise despite treatment was noted from day 10 of hospital stay.

By day 12, improvement in sensorium and neck control was noted. A trial of extubation was attempted but was unsuccessful, and tracheostomy was performed after obtaining consent because prolonged ventilatory support was anticipated.

On day 13, serial monitoring demonstrated a progressive rise in serum creatinine over the preceding days, accompanied by a reduction in urine output. Serial relevant laboratory findings are summarized in Table *1*.

Day	Creatinine (0.7-1.3 mg/dL)	Urea (15-40 mg/dL)	Potassium (3.5-5 mmol/L)
0	1.3	-	4
2	1.6	-	-
3	1.6	-	-
4	-	-	3.4
5	3.6	104	3.6
6	3.2	-	4.1
7	3.5	129.3	4.6
8	4	154.4	6.5
9	4.8	163	5.7
10	5.4	140	6.4
11	5.9	137	5.7
12	6.7	144.7	5.6
13	7.2	-	6
14	7.1	153.2	4.8
15	5.4	-	4.6
16	5.7	159	4.7
17	4	122.9	4.3
18	3.9	-	4.1
19	3.8	129.7	4.6
20	4.6	125.8	4.2
21	4.4	133.8	3.8
22	3.6	-	3.3
24	3	75	3.3
25	-	-	3.3
27	-	-	3.6
28	2.4	56	4.8
29	-	-	-
30	2.6	-	-
Follow-up 1	1.5	29	4.5
Follow-up 2	1.5	-	4.9
Follow-up 3	0.9	-	-

TABLE 1: Serial serum creatinine, urea and potassium during hospitalization and follow-up.

Creatine phosphokinase (CPK) was subsequently measured at a higher-level laboratory and was markedly elevated at 38,710 U/L, supporting significant rhabdomyolysis. Despite hydration, renal function continued to deteriorate. The patient developed refractory hyperkalemia, and the need for emergency dialysis was discussed with the patient's relatives. Hemodialysis was initiated after obtaining consent; he underwent three sessions of dialysis over three days.

Possible contributors to the AKI were evaluated, including hemodynamic kidney injury related to shock,

infection, possible direct or toxin-associated renal injury, and rhabdomyolysis. Infectious evaluation later identified a carbapenem-resistant Enterobacterales isolate from the endotracheal aspirate, while ultrasonography demonstrated findings suggestive of left-sided pyelonephritis, shown in Figure *1*, but urine and blood cultures had not grown. Given persistent fever and the clinical context, infection was considered a potential contributor to the patient's renal dysfunction.

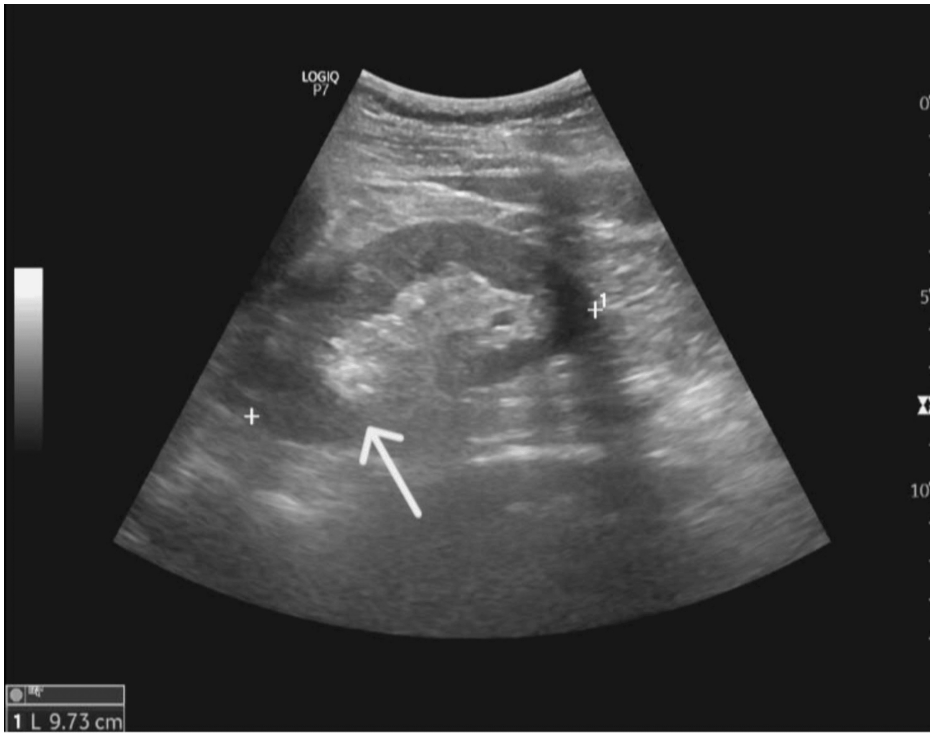


FIGURE 1: Ultrasonography of the left kidney with arrow pointing towards peri-nephric fat stranding

The patient remained under close monitoring and received further supportive care. After approximately two weeks of intensive care, he was transferred to the high-dependency unit, where he had persistent fever spikes, and an evaluation was localized to left pyelonephritis. He was started on injection polymyxin B therapy at a renally adjusted dose, which was continued for a total of five days and then stopped based on the culture and susceptibility results. He was subsequently shifted to the ward on day 22, as his clinical parameters improved.

The patient later requested discharge and was discharged at the request after psychiatric counselling, as the ingestion was intentional due to family discord. Tracheostomy care was explained to his relatives, and outpatient follow-up was arranged. He was followed weekly for one month. The tracheostomy tube was progressively downsized and was closed after one month. At one-month follow-up, the patient was clinically well, ambulatory without support, and had no reported symptoms attributable to renal dysfunction. Follow-up creatinine was in an improving trend with the last follow-up creatinine of 0.9 mg/dL. The clinical course is summarized in Table *2*.

Hospital day	Clinical events
Day 0	3 pm: Found at home in a semi-conscious state. 4:40 pm: Taken to local hospital - Gastric lavage followed by inj. atropine 4 mg and inj. pralidoxime 2-amp stat was given. 5:00 pm: Reached our hospital with GCS of 7/15- intubated, started on inj. atropine infusion, empirical IV antibiotics and admitted to ICU for further management.
Day 1	He was continued on inj. atropine and IV antibiotics.
Day 2	Developed hypotension, not responsive to fluids; escalating vasopressor/inotropic support; creatinine began to rise.
Day 3	Bradycardia and cardiopulmonary arrest; ROSC was achieved after 2 cycles of CPR.
Day 4	Marked proximal muscle weakness (<2/5); intermediate syndrome diagnosed. Atropine continued.
Day 8	Serial rise in potassium levels was noted and correction was started as per protocol.
Day 12	Improved sensorium and neck control; failed trial of extubation; tracheostomy performed after consent.
Day 13	Progressive AKI with oliguria; CPK 38710 U/L; refractory hyperkalaemia; creatinine 7.2 mg/dL, potassium 6 mmol/L; emergency haemodialysis initiated.
Day 16	Clinical and renal improvement; transfer out of ICU; had persistent fever spikes - diagnosed to have left pyelonephritis - inj. polymyxin B x 5 days.
Day 22	Shifted to ward.
Day 30	Corking trial of tracheostomy tube was done - tolerating well. Planned for removal on OPD basis as patient requested for discharge.
Post-discharge follow-up	Creatinine in improving trend. Vitally stable. Ambulating without support. Tracheostomy tube removed after a month of follow-up.

TABLE 2: Clinical timeline of the hospital course and follow-up

GCS: Glasgow Coma Scale, OPD: Outpatient Department, inj.: Injection, CPR: Cardiopulmonary Resuscitation, ROSC: Return of Spontaneous Circulation, AKI: Acute Kidney Injury, CPK : Creatine Phosphokinase, amp: Ampoule

Discussion

Toxicity from OP poisoning is due mainly to AChE inhibition with consequent cholinergic overstimulation. Manifestations can include gastrointestinal symptoms, increased secretions, bronchospasm, altered level of consciousness, cardiovascular instability, muscle weakness, respiratory failure, and ultimately death [1]. In cases of severe OP poisoning, acute cholinergic syndrome can precede IMS, which includes neuromuscular weakness involving proximal muscles, neck flexors, respiratory muscles, and motor cranial nerves [2].

In the current case, initial toxicity was manifested by loss of consciousness, hypoxemia, respiratory failure requiring intubation, and later cardiovascular instability. IMS occurred in this patient four days after OP poisoning and was manifest by marked weakness of proximal muscles. Ventilator support eventually required tracheostomy.

The neuromuscular examination of our patient was first limited by the fact that the patient was being managed under mechanical ventilation and sedation. However, after stopping the sedation in order to assess the fitness of the patient for extubation, muscle weakness (Grade 2/5 on Medical Research Council (MRC) scale) was observed. Taking into account the clinical picture and the development of the illness in the patient following OP poisoning, there was suspicion of the development of IMS in our patient.

There have also been some reports of renal problems after poisoning by OPs, but their frequency and exact mechanisms remain unclear. Possible mechanisms suggested include tubular necrosis, oxidative stress, hypovolemia, and renal damage resulting from systemic issues [4,5]. For this case, there was no obvious single cause for the AKI. Vasopressor requirement due to early hemodynamic instability, presumed infection/aspiration, significant rhabdomyolysis, and the possibility of renal injury due to the toxins were all possible factors.

The high CPK level of 38,710 U/L was a very important finding and indicates that rhabdomyolysis was possibly one of the causes of the renal injury [9,10]. Rhabdomyolysis results in AKI due to pigment injury to the tubules, especially in critically ill patients with prolonged immobility, muscle injury, and systemic

toxicity [9,10]. Since the available information about the individual factors cannot confirm their exact contribution to the problem, the AKI in this case was multifactorial in nature.

The progressive increase in creatinine, decrease in urine output, and development of refractory hyperkalemia necessitated hemodialysis. Thereafter, there was improvement in renal functions and the patient had a good clinical course with follow-up. It has been previously documented that AKI and acute renal failure can occur after OP poisoning, and in some cases, the need for renal replacement therapy was seen [5]. The population-based data have shown an increased incidence of AKI after OP poisoning [6].

It underscores the significance of evaluating renal function in patients with severe OP poisoning, especially when hemodynamic instability is present, when the patient is in a critical condition for a long duration, or when there is evidence of rhabdomyolysis. Serial evaluation of urine output, serum creatinine, electrolyte status, and muscle injury markers can help in early diagnosis of renal complications.

There are few limitations of this case. We were unable to measure the cholinesterase levels, and CPK level reports were only available on the 13th day of admission. Samples were sent to a nearby tertiary care center for analysis. Hence, the exact timing of rhabdomyolysis and its impact on AKI could not be determined.

Conclusions

Complications of severe OP toxicity include IMS, respiratory failure, rhabdomyolysis, and acute renal failure. In patients with severe toxicity, regular monitoring of kidney function, output of urine, electrolyte imbalance, and muscle damage is required, especially in cases of hemodynamic instability. Prompt detection and treatment of such complications, as well as the use of renal replacement therapies where necessary, may lead to favorable results.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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