BEYOND THE CHOLINERGIC CRISIS: NON-OLIGURIC ACUTE KIDNEY INJURY IN ORGANOPHOSPHATE POISONING

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ABSTRACT

Organophosphate (OP) poisoning is a common toxicological emergency, primarily presenting with cholinergic manifestations. Acute kidney injury (AKI) is an uncommon but clinically important complication of OP poisoning, usually attributed to secondary factors such as hypotension, dehydration, rhabdomyolysis, or sepsis. However, intrinsic renal injury may occur even in the absence of these classical risk factors. We report a case of a 24-year-old male who developed non-oliguric acute kidney injury following intentional ingestion of an organophosphate compound containing chlorpyrifos 50.0% and cypermethrin 5.0%. The patient presented with features of cholinergic excess and markedly reduced plasma cholinesterase levels. He remained hemodynamically stable during hospitalization without persistent hypotension or need for renal replacement therapy. Laboratory investigations showed a transient rise in serum creatinine, peaking on the second day and returning to baseline with supportive management. Serum creatine kinase levels were normal, and urine output was preserved throughout the hospital stay. This case suggests the possibility of intrinsic tubular injury as a mechanism of AKI in organophosphate poisoning. Routine monitoring of renal function may help in early detection of renal involvement even in clinically stable patients. Awareness of non-oliguric AKI as a potential complication of organophosphate toxicity is important for timely supportive management.

KEYWORDS

Acute kidney injury, cholinesterase inhibition, non-oliguric AKI, organophosphate poisoning, tubular toxicity

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INTRODUCTION

Organophosphate (OP) compounds are widely used pesticides and are an important cause of both accidental and intentional poisoning, particularly in agricultural regions.[1,2] These compounds inhibit acetylcholinesterase, leading to accumulation of acetylcholine at synaptic junctions and resulting in excessive stimulation of muscarinic and nicotinic receptors.[3,4] Clinically, organophosphate poisoning commonly presents with cholinergic manifestations such as nausea, vomiting, salivation, respiratory distress and neurological symptoms.

Although neurological and respiratory complications are well recognized, renal involvement in organophosphate poisoning is relatively uncommon. Acute kidney injury (AKI) has been reported as a complication of OP toxicity and is usually attributed to secondary factors such as hypotension, dehydration, rhabdomyolysis, sepsis, or pigment nephropathy.[5,6] However, emerging evidence suggests that intrinsic renal injury may occur even in the absence of persistent hemodynamic instability or classical risk factors.

Non-oliguric AKI may be particularly under-recognized in patients with organophosphate poisoning because patients may remain clinically stable despite biochemical evidence of renal dysfunction. Therefore, routine monitoring of renal function may be important for early detection of renal involvement. The pathophysiology of OP-induced AKI is thought to be multifactorial, involving cholinergic overstimulation, renal vascular effects, and possible direct tubular toxicity mediated by oxidative stress mechanisms.

CASE REPORT

A 24-year-old male college student with a history of depressive disorder, not on pharmacological treatment, presented to the emergency department approximately four hours after ingesting 40–50 mL of an insecticide containing chlorpyrifos 50.0% and cypermethrin 5.0% in a suicide attempt. He experienced 2–3 episodes of watery vomiting and excessive salivation but denied diarrhea, abdominal pain, seizures, or altered sensorium. There was no history of recent medication, alcohol, or tobacco use. He had no prior renal or chronic medical conditions.

On examination, he was anxious but fully conscious and oriented (Glasgow Coma Scale E4V5M6). Pupils were bilaterally constricted and reactive to light. Vital signs were: blood pressure 90/60 mmHg, heart rate 58 beats per minute, respiratory rate 26 breaths per minute, temperature 98.7°F and oxygen saturation 94.0% on room air. There were no signs of dehydration or shock. Respiratory examination revealed decreased basal air entry with fine crepitations, while cardiovascular and abdominal examinations were unremarkable.

Initial management included gastric lavage followed by intravenous administration of atropine and pralidoxime. The patient was admitted to the intensive care unit for monitoring and supportive care, continuing atropine and pralidoxime infusion. He remained hemodynamically stable and did not require dialysis or mechanical ventilation.

Laboratory investigations revealed a transient rise in serum creatinine from 1.43 mg/dL on day 1 to 2.39 mg/dL on day 2, which normalized to 0.98 mg/dL by day 4. Blood urea remained stable (27 mg/dL on day 1 to 21 mg/dL on day 4), and sodium and potassium levels were within normal ranges. Urine output was maintained at approximately 1 mL/kg/hour, consistent with non-oliguric acute kidney injury (Table 1).

Arterial blood gas analysis showed a mild lactate elevation (3.6 mmol/L) on admission with normal pH and bicarbonate, which normalized over the next two days, indicating reversible metabolic changes (Table 2).

Baseline hematological parameters, liver function tests, and creatine kinase levels were within normal limits. Plasma cholinesterase was markedly reduced (0.34 U/mL), confirming organophosphate exposure. Hemoglobin was 15.6 g/dL, total leukocyte count was 12,620/µL with neutrophil predominance, and

Table 1: Day-wise renal function and electrolytes

Parameter	Normal range	Day 1	Day 2	Day 3	Day 4
Serum creatinine (mg/dL)	0.72–1.18	1.43	2.39	1.9	0.98
Blood urea (mg/dL)	17–43	27	26	18	21
Sodium (mmol/L)	135–145	142	135	137	136
Potassium (mmol/L)	3.5–5.0	3.6	3.8	4.2	4.2

Table 2: Arterial blood gas trends				
Parameter	**Normal range**	**Day 1**	**Day 2**	**Day 3**
pH	7.35–7.45	7.40	7.42	7.37
Bicarbonate (mmol/L)	22–26	22.3	25.1	22.8
Lactate (mmol/L)	0.5–2.2	3.6	1.0	0.8
Anion gap (mmol/L)	8–12	10.8	6.6	7.5

Table 3: Baseline hematological, biochemical and urine investigations		
Parameter	**Normal range**	**Value**
Creatine kinase total (IU/L)	25–175	157
Serum cholinesterase (U/mL)	4.65–10.44	0.34
Hemoglobin (g/dL)	13–18	15.6
Total leukocyte count (/µL)	4000–11000	12620
Neutrophil/lymphocyte percentage (%)	40–70 / 20–45	74 / 21
Platelet count (/µL)	150,000–450,000	291,000
Total bilirubin (mg/dL)	0.2–1.0	1.55
Direct bilirubin (mg/dL)	<0.3	0.28
Aspartate aminotransferase (U/L)	<40	15.3
Alanine aminotransferase (U/L)	10–40	11.6
Alkaline phosphatase (U/L)	30–120	99.8
Urine albumin	Nil	Nil
Red blood cells in urine (per high power field)	0–2	0
Pus cells in urine (per high power field)	0–2	0
Casts in urine	Nil	Nil
Urine glucose	Nil	Nil

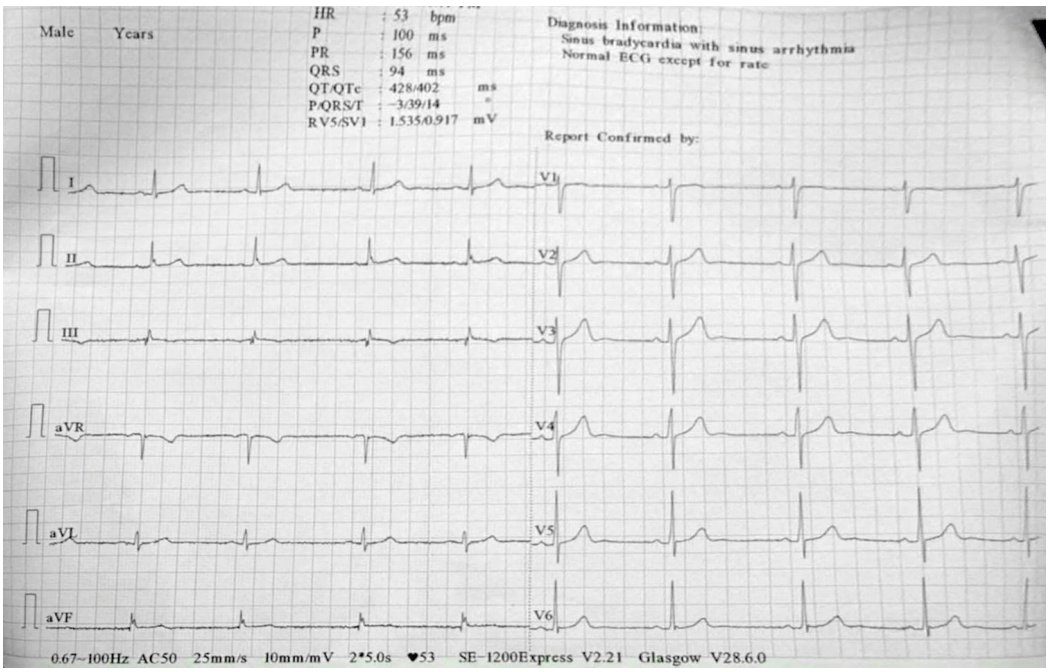

Fig. 1: Electrocardiogram showing sinus bradycardia. **Legend:** A 12-lead electrocardiogram demonstrating sinus bradycardia with a heart rate of approximately 55 beats per minute. PR interval, QRS duration, and QTc are within normal limits. No significant ST-segment or T-wave abnormalities are noted.

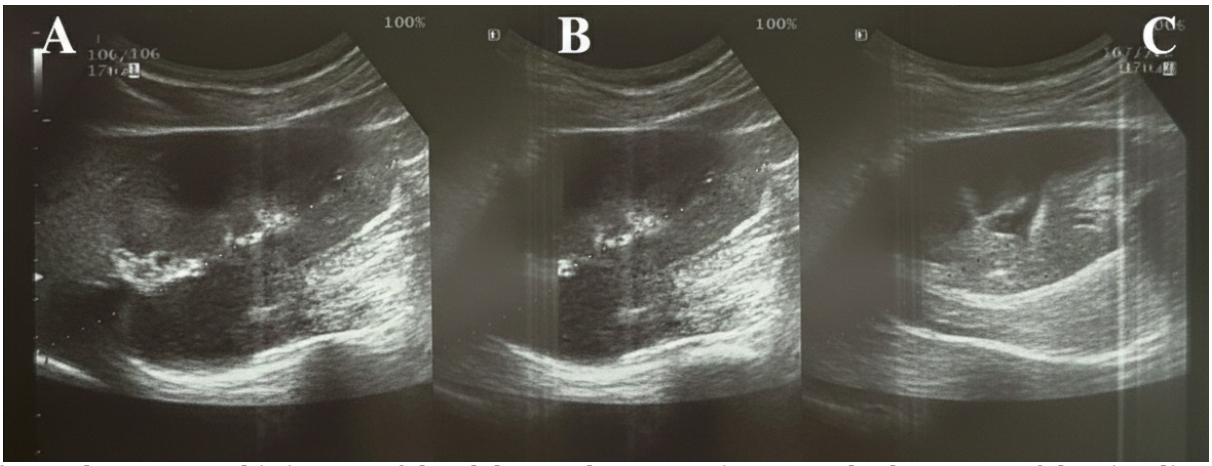

Fig. 2: Ultrasonographic images of the abdomen demonstrating normal echotexture of the visualized abdominal organs with no focal lesions or abnormal fluid collections. **Legend:** (A) Normal liver and gallbladder appearance, (B) Normal spleen and pancreas with preserved echotexture, (C) Bilateral kidneys showing normal size and corticomedullary differentiation

platelet count was 291,000/µL. Urinalysis revealed no proteinuria, hematuria, or casts (Table 3). Electrocardiogram on admission showed sinus bradycardia, which resolved during hospitalization (Fig. 1). Abdominal ultrasonography demonstrated normal kidneys with preserved corticomedullary differentiation and no abnormal findings (Fig. 2A, B, and C).

DISCUSSION

AKI is an uncommon but clinically significant complication of organophosphate (OP) poisoning.[1-3] Classical mechanisms of renal involvement in OP poisoning include hypotension, dehydration, rhabdomyolysis, sepsis, and pigment nephropathy.[1,2] However, in some patients, AKI may develop despite preserved hemodynamics and urine output, suggesting the possibility of intrinsic renal injury.[4-6]

In the present case, the patient developed non-oliguric AKI with a transient rise in serum creatinine that peaked on the second day and subsequently normalized with supportive management. Urine output was maintained at approximately 1 mL/kg/hour throughout hospitalization. Although the initial blood pressure was 90/60 mmHg, there were no clinical features of shock or persistent hypoperfusion, and the blood pressure stabilized without vasopressor support. There was no persistent hypotension or clinical evidence of circulatory shock during the hospital stay. Serum creatine kinase levels were within normal limits, excluding rhabdomyolysis as a cause of renal injury.

Similar cases of AKI following organophosphate exposure have been reported in the literature. Cavari *et al*[5] described organophosphate poisoning–induced acute renal failure in the absence of obvious pre-renal causes. Veeranki *et al*[7] reported severe and recurrent AKI following dichlorvos exposure, suggesting possible intrinsic renal injury. These reports support the possibility that renal dysfunction in organophosphate poisoning may occur independently of classical risk factors.[6,8] The occurrence of acute renal failure in organophosphate poisoning has also been described in other reports, including cases managed with haemodialysis and studies describing clinical features and prognosis of poisoned patients.[9-11]

The pathophysiology of OP-induced AKI is likely multifactorial. Organophosphate compounds inhibit acetylcholinesterase, resulting in excessive accumulation of acetylcholine and persistent stimulation of muscarinic and nicotinic receptors, including those present in renal vasculature.[3,4] This cholinergic overstimulation may lead to renal vasoconstriction, medullary hypoxia, and subsequent tubular injury. Additionally, experimental studies suggest that chlorpyrifos may induce oxidative stress and mitochondrial dysfunction in renal tubular cells, leading to cellular injury.[5,6] These mechanisms may explain the development of reversible non-oliguric AKI observed in this patient.

Measurement of urinary indices such as fractional excretion of sodium (FENa) could have further supported the diagnosis of intrinsic tubular injury. However, this parameter was not assessed in the present case, which is a major limitation of this report. Nevertheless, preserved urine output, absence of persistent hypotension, normal creatine kinase levels,

and normalization of serum creatinine after supportive care suggest a transient intrinsic renal insult rather than pre-renal azotemia.

From a clinical perspective, this case highlights the importance of routine renal function monitoring in patients with organophosphate poisoning, even when hemodynamic status appears stable. Early recognition of renal involvement, close observation of urine output, and avoidance of nephrotoxic drugs are important for preventing progression of renal injury. Early supportive care may result in complete recovery, as seen in our patient. This case adds insight into the spectrum of organophosphate toxicity, showing that non-oliguric AKI due to possible direct tubular toxicity may occur even in the absence of classical risk factors.

In conclusion, AKI is an uncommon but important complication of organophosphate poisoning, even in patients with stable hemodynamics and preserved urine output. Non-oliguric AKI may occur due to possible intrinsic tubular injury independent of hypotension, dehydration, or rhabdomyolysis. Early recognition through close monitoring of renal function and urine output is essential for timely supportive management. Awareness of this potential complication may help clinicians identify renal involvement at an early stage and improve patient outcomes. Although further studies are needed to better understand the exact mechanism, clinicians should remain vigilant for renal dysfunction in organophosphate poisoning.

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Ethical consideration: Written informed consent was obtained from the patient for publication of this case report and for the use of relevant clinical information. Ethical approval was obtained in accordance with the institutional publication policy of Nepal Medical College Teaching Hospital. Patient confidentiality was strictly maintained, and no identifying information has been included in this manuscript. The study was conducted in compliance with standard ethical guidelines for case report publication.

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