RESEARCH PAPER

Atypical Organophosphate Poisoning Masquerading as Acute Flaccid Quadriparesis Following Gastroenteritis: A Diagnostic Challenge in the Emergency Department

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ABSTRACT

Background Organophosphate (OP) poisoning remains a significant cause of morbidity and mortality, particularly in agricultural regions of developing countries. While the classical cholinergic toxidrome is well recognized, atypical presentations that lack the hallmark features can pose considerable diagnostic challenges. This is especially true when the initial symptoms closely mimic common medical conditions such as gastroenteritis or electrolyte disturbances.

Case Presentation: We report the case of a 40-year-old male farmer who presented to the emergency department with a two-day history of profuse watery diarrhea, progressive quadriparesis, bilateral ptosis, and respiratory failure. The initial clinical picture, taken together with a recent history of snakebite and documented hypokalemia at the referring hospital, directed the diagnostic workup toward hypokalemic paralysis, neurotoxic snake envenomation, and neuromuscular disorders. The absence of classic organophosphate features, including the characteristic garlic-like odor, excessive secretions, bradycardia, and initial miosis, significantly delayed the diagnosis. Serial clinical re-examination revealed progressive miosis (pupil size 1.5 mm, sluggishly reactive), which, combined with the constellation of diarrhea, muscle weakness, and respiratory failure, raised suspicion for OP poisoning. The diagnosis was confirmed by markedly reduced serum pseudocholinesterase levels (103 U/L) and positive toxicological analysis of gastric aspirate. The patient's family subsequently discovered an empty container of an organophosphate-pyrethroid combination (Ethion + Cypermethrin) at the patient's residence. Treatment with intravenous atropine and pralidoxime infusions led to significant clinical improvement.

Conclusion: This case underscores the importance of maintaining a broad differential diagnosis and the critical role of serial clinical re-examination in emergency presentations of acute flaccid paralysis. Organophosphate poisoning should remain in the differential diagnosis of any patient presenting with unexplained quadriparesis, respiratory failure, and gastrointestinal symptoms, even when classical cholinergic features are absent.

Keywords: Organophosphate poisoning; atypical presentation; acute flaccid paralysis; quadriparesis; diagnostic challenge; cholinesterase; emergency medicine

How to cite this article: Anas T C, Pawar A, Udapudi S V, Hatti M A, Lakshmi M, Haneef H, Aboobacker S M, Khamarunnisa K, Unais A M, Jishnu K S., Atypical Organophosphate Poisoning Masquerading As Acute Flaccid Quadriparesis Following Gastroenteritis: A Diagnostic Challenge in the Emergency Department. Int J Drug Deliv Technol. 2026;16(43s): 860-867; Doi: 10.25258/Ijddt.16.43s.87

Source of support: Nil.

Conflict of interest: None

Introduction

Organophosphate (OP) compounds remain among the most commonly encountered causes of poisoning in agricultural communities across the developing world, accounting for significant morbidity and mortality in South and Southeast Asia [1,7]. The World Health Organization estimates that approximately three million cases of pesticide poisoning occur worldwide each year, with organophosphates constituting a substantial proportion of these exposures. In India, OP poisoning is

one of the leading causes of toxicological emergencies, particularly among rural agrarian populations who have easy access to these compounds [3].

The underlying pathophysiology of OP poisoning involves irreversible inhibition of acetylcholinesterase at synaptic junctions, leading to an accumulation of acetylcholine at muscarinic receptors found in parasympathetic end organs, nicotinic receptors at neuromuscular junctions and autonomic ganglia, and within central nervous system synapses [1,2]. The classical presentation, commonly referred to as the cholinergic toxidrome, is typically characterized by the SLUDGE/DUMBELS mnemonic: Diarrhea, Urination, Miosis, Bronchospasm and Bradycardia, Emesis, Lacrimation, and Salivation. When these features are present in their entirety, the clinical picture is relatively straightforward to recognize [3].

However, atypical presentations of OP poisoning, in which classical cholinergic features are absent, delayed, or overshadowed by predominant nicotinic or central manifestations, pose a formidable diagnostic challenge [2,4]. In such cases, the clinical picture may closely mimic a variety of neuromuscular disorders, including Guillain-Barré syndrome, myasthenia gravis, hypokalemic periodic paralysis, botulism, or neurotoxic snake envenomation. This can lead to delayed diagnosis and potentially fatal outcomes [5,6].

We present a diagnostically challenging case of a 40-year-old male farmer who arrived at the emergency department with acute flaccid quadriparesis and respiratory failure following an episode of gastroenteritis. The absence of characteristic organophosphate features, a misleading history of recent snakebite, and initial laboratory findings of hypokalemia led to a prolonged diagnostic odyssey before serial clinical re-examination ultimately uncovered the true diagnosis of organophosphate poisoning.

2. CASE PRESENTATION

2.1 Patient Demographics and Presenting Complaint

A 40-year-old male farmer, residing in Vidyanagar, Devlatti, Karnataka, India, was brought to the Emergency Department of Jawaharlal Nehru Medical College, Belagavi, on the 23rd of June 2025. He presented with a history of multiple episodes of loose stools and decreased responsiveness lasting two days, along with an inability to open his eyes and bilateral upper and lower limb weakness that had developed over the preceding day.

2.2 History of Presenting Illness

The patient had been apparently well until two days before presentation, when he developed sudden-onset profuse watery diarrhea amounting to more than ten episodes per day. The stools were watery in consistency, non-foul smelling, and contained no blood. He also experienced one episode of projectile vomiting containing food particles, which was non-bilious and non-bloody.

Approximately twelve hours after the onset of these gastrointestinal symptoms, the patient developed progressive weakness involving all four limbs. The weakness was acute in onset, symmetrical, and progressive, affecting both upper and lower extremities simultaneously. This was accompanied by an inability to speak (motor aphasia with preserved comprehension) and bilateral ptosis. Over the following hours, there was a progressive deterioration in his level of responsiveness. There was no history of abdominal pain, fever, reduced urine output, consumption of food from outside sources, recent travel, or similar episodes among family members. No history of head injury was elicited.

2.3 Past Medical History

Of particular significance, the patient had sustained a snakebite on the 16th of June 2025, one week before his current presentation, for which he was taken to a local hospital and managed with injection tetanus toxoid, injection hydrocortisone, and injection chlorpheniramine (Avil). He had been discharged on the same day. There was no known history of diabetes mellitus, hypertension, connective tissue disorders, or thyroid disease. He was not taking any regular medications. The patient reported a history of alcohol consumption spanning the previous six years, with his last intake being five days before admission. No known drug allergies were reported.

2.4 Pre-hospital Course

The patient was initially brought to a local hospital following the onset of his gastrointestinal symptoms. On the second day of admission at the referring facility, he developed bilateral ptosis, bilateral upper and lower limb weakness, breathlessness, and a further decrease in responsiveness. Investigations at the referring hospital revealed hypokalemia (serum potassium 2.50 mmol/L) and hyperglycemia (blood glucose 300 mg/dL). He was treated with intravenous antibiotics, potassium correction, and intravenous fluids. The patient was subsequently transferred to our Emergency Department with supplemental oxygen, ongoing potassium correction, and a Foley's catheter in situ containing 250 mL of urine in the collection bag.

2.5 Emergency Department Assessment

2.5.1 Primary Survey

On arrival in the Emergency Department, the primary survey revealed the following findings:

Parameter	Findings
Airway	Drowsy but arousable; unable to speak; absent neck lifting; no pooling of secretions
Breathing	Respiratory rate: 30 cycles/min; SpO_2: 80% on 15 L/min O_2 via face mask; bilaterally equal air entry with conducted sounds; bilateral crepitations present
Circulation	Heart rate: 90 bpm; Blood pressure: 130/80 mmHg; peripheral pulses palpable in all limbs
Disability	GCS: E1V1M6 (8/15); Pupils: 2.0 mm, bilaterally equal and reactive to light; weakness of bilateral upper and lower limbs; bilateral ptosis present
Exposure	GRBS: 161 mg/dL; Temperature: 97.5°F; no external injuries or bite marks identified

Given the severity of the respiratory failure, with oxygen saturation of just 80% despite 15 L/min of supplemental oxygen and a respiratory rate of 30 breaths per minute, the patient was immediately intubated via the endotracheal route and placed on mechanical ventilation.

2.5.2 Point-of-Care Ultrasound (POCUS)

Bedside echocardiography revealed an ejection fraction of 55 to 60% with no regional wall motion abnormalities. The inferior vena cava diameter measured 1.2 cm, consistent with euvolemic status.

2.5.3 General and Systemic Examination

The patient was a middle-aged male of moderate build and nutrition. He was drowsy but arousable and capable of obeying simple commands. There was no pallor, icterus, clubbing, cyanosis, or lymphadenopathy. No muscle wasting or neurocutaneous markers were observed, and no bite marks or signs of active bleeding were present.

Cardiovascular examination revealed normal S1 and S2 heart sounds with no murmurs. Respiratory examination showed bilateral equal chest rise and air entry with bilateral diffuse crepitations. The abdomen was soft, non-tender, with no organomegaly and normal bowel sounds.

2.6 Neurological Examination

The neurological examination formed the cornerstone of the diagnostic workup and yielded the following findings.

Higher Mental Functions: The patient was right-handed, drowsy, arousable, and able to obey simple commands. Formal assessment of memory, language, and cranial nerve function was limited by his clinical state. Bilateral ptosis was a prominent finding.

Motor System: The attitude of the limbs was in extension. Hypotonia was present bilaterally in both upper and lower limbs. Power was graded 3/5 on the Medical Research Council (MRC) scale in all four limbs symmetrically.

Table 1: Motor Power Assessment (MRC Grading)

	Right	Left
Upper Limb	3/5	3/5
Lower Limb	3/5	3/5

Table 2: Deep Tendon Reflexes

Reflex	Right	Left
Biceps	Absent	Absent
Triceps	Absent	Absent
Supinator	Absent	Absent
Knee	Absent	Absent
Ankle	Absent	Absent

Deep tendon reflexes were universally absent (areflexia) in all four limbs. Plantar responses were bilaterally mute. At the time of initial assessment, the pupils measured 2.0 mm, were bilaterally equal, and were reactive to light. Speech assessment revealed motor aphasia with intact comprehension. Pain sensation was preserved; however, temperature, vibration, joint position sense, and cortical sensations could not be formally evaluated given the patient's clinical state. There were no signs of cerebellar dysfunction or meningeal irritation, as neck stiffness, Kernig's sign, and Brudzinski's sign were all absent.

3. INVESTIGATIONS

3.1 Laboratory Investigations

Table 3: Hematological Parameters

Parameter	22/06/2025 (Referring Hospital)	23/06/2025 (Our Center)
Hemoglobin	8.2 g/dL	11.7 g/dL
Hematocrit	–	38.9%
MCV	74.1 fL	79.7 fL
MCH	22.7 pg	24.0 pg
MCHC	30.7 g/dL	30.1 g/dL
RBC Count	3.6 million/µL	4.8 million/µL

RDW	–	23.0%
Reticulocyte Count	–	2.8%
WBC Count	11,800/µL	16,300/µL
Platelets	–	2,58,000/µL
Neutrophils	90%	94%
Lymphocytes	10%	2%

Table 4: Biochemical Parameters

Parameter	22/06/2025	23/06/2025
Total Bilirubin / Direct	0.53 / 0.91 mg/dL	0.98 / 0.63 mg/dL
SGOT / SGPT	134 / 56 IU/L	48 / 35 IU/L
ALP	50.0 IU/L	48 IU/L
Albumin	3.80 g/dL	4.0 g/dL
Sodium	134 mmol/L	139 mmol/L
Potassium	2.50 mmol/L	3.90 mmol/L
Chloride	108 mmol/L	106 mmol/L
Serum Creatinine	0.90 mg/dL	–
Blood Urea	30.8 mg/dL	–
Blood Glucose	300 mg/dL	161 mg/dL

The coagulation profile showed a prothrombin time (test) of 15.5 seconds against a control of 12.5 seconds, with an INR of 1.21. The whole blood clotting time was 20 minutes and showed normal clotting.

Table 5: Serial Arterial Blood Gas Analysis

Parameter	23/06 12:10 PM (15L O_2 Face Mask)	24/06 7:00 AM (VC Mode, FiO_2 60%)	24/06 5:00 PM (SIMV-VC, FiO_2 35%)
pH	7.270	7.385	7.359
PaO_2 (mmHg)	44.5	180.5	202.6
$PaCO_2$ (mmHg)	29.1	25.7	34.4
HCO_3^- (mmol/L)	13.1	15.0	18.9
Base Excess (mmol/L)	−12.6	−8.5	−5.8
SpO_2 (%)	70.7	99.5	99.6
Hemoglobin (g/dL)	9.38	8.10	10.50
Lactate (mmol/L)	0.38	0.48	0.60
Glucose (mg/dL)	100.5	71.1	101

The initial arterial blood gas analysis, obtained while the patient was receiving 15 L/min of oxygen via face mask, demonstrated type I respiratory failure accompanied by metabolic acidosis (pH 7.270, PaO_2 44.5 mmHg, HCO_3^- 13.1 mmol/L, base excess of minus 12.6 mmol/L). Notably, the lactate level was normal at 0.38 mmol/L, suggesting a non-anion gap metabolic acidosis most likely secondary to bicarbonate losses from the diarrhea. Serial arterial blood gas analyses showed progressive improvement following intubation and the initiation of mechanical ventilation.

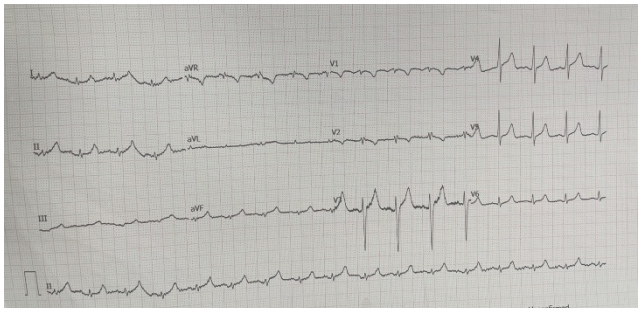
Table 6: Key Diagnostic Investigations

Investigation	Value	Reference Range
Serum Potassium (23/06)	3.90 mmol/L	3.5 to 5.0 mmol/L
Serum Pseudocholinesterase	**103 U/L**	5,320 to 12,920 U/L
Creatine Phosphokinase (CPK)	1,303 U/L	39 to 308 U/L
Gastric Aspirate Toxicology	**Positive for OP compound**	Negative

3.2 Electrocardiogram (ECG):

The 12-lead ECG revealed normal sinus rhythm with a heart rate of approximately 99 beats per minute. Low-voltage QRS complexes were noted. The QRS duration was 70 ms, QT/QTcBaz was 346/444 ms, and the PR interval was 126 ms. The tracing was classified as borderline. The random blood sugar at the time of ECG recording was 135 mg/dL. Importantly, there was no bradycardia, a finding that initially argued against typical organophosphate poisoning.

Figure: 12-lead ECG showing normal sinus rhythm at



99 bpm with low-voltage QRS complexes. No bradycardia. QRS duration 70 ms, QT/QTcBaz 346/444 ms, PR interval 126 ms

3.3 Imaging Studies

Chest X-ray:

The chest radiograph demonstrated bilateral diffuse opacities, consistent with aspiration pneumonitis and possibly pulmonary edema in the clinical context.

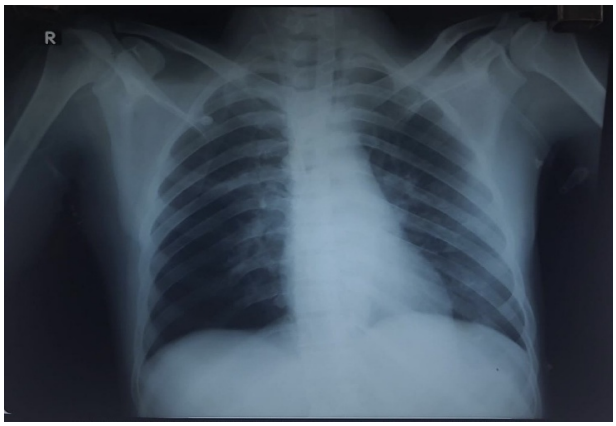


Figure: Chest radiograph demonstrating bilateral diffuse opacities consistent with aspiration pneumonitis in the setting of decreased consciousness and vomiting.

MRI Brain (Plain):

A brain MRI was performed on 24/06/2025 at KLE Dr. Sampatkumar S. Shivanagi Cancer Hospital, Belagavi. The imaging revealed a normal cortical grey matter and white matter signal intensity pattern, unremarkable bilateral hippocampi, normal ventricles with no midline shift, and normal brainstem and cerebellar signal intensities. Incidental findings included T2 hyperintense mucosal thickening of the left maxillary sinus, suggestive of sinusitis and a polyp. The overall impression was that there was no significant diagnostic pathology in the brain parenchyma.

MRI Spine:

MRI of the spine was performed to rule out spinal cord pathology as a cause of quadriparesis. The findings were unremarkable.

4. CLINICAL COURSE AND MANAGEMENT

4.1 Initial Management

On arrival, the patient was immediately intubated in view of his respiratory failure and placed on mechanical ventilation. Given the history of snakebite one week earlier, a whole blood clotting time (WBCT20) test was performed, which showed normal clotting at 20 minutes, effectively ruling out viperine envenomation with coagulopathy. Initial management consisted of intravenous potassium correction (pending laboratory confirmation), intravenous antibiotics for suspected aspiration pneumonia, intravenous fluid resuscitation, and a fentanyl infusion for sedation and ventilator synchrony.

4.2 Diagnostic Evolution

The initial differential diagnosis included hypokalemic paralysis secondary to acute gastroenteritis, neurotoxic snake envenomation in light of the recent snakebite history, botulism, myasthenia gravis, and organophosphate compound poisoning. When the serum potassium level returned within normal limits at 3.90 mmol/L following correction, and the CPK was found to be elevated at 1,303 U/L, hypokalemic paralysis became a less convincing explanation on its own, prompting a broader reconsideration of the differential diagnosis.

MRI studies of the brain and spine were performed to rule out central nervous system pathology and returned unremarkable results. An atropine-neostigmine challenge test was administered but yielded no improvement in ptosis or muscle power, making myasthenia gravis considerably less likely.

4.3 The Diagnostic Breakthrough

With the patient's condition failing to improve despite aggressive supportive care, a systematic re-examination was undertaken along with a refocused effort at history-taking. This proved to be the turning point. On re-examination, progressive miosis was identified, with pupil size now measuring 1.5 mm, reduced from the initial 2.0 mm, and the pupils were now only sluggishly reactive to light. This evolving sign, when viewed alongside the constellation of profuse diarrhea, progressive muscle weakness, respiratory failure, and now the progressive miosis, raised a strong clinical suspicion for organophosphate poisoning.

Serum pseudocholinesterase was urgently sent and returned markedly reduced at 103 U/L (reference range: 5,320 to 12,920 U/L), confirming the clinical suspicion. A sample of gastric aspirate was sent for toxicological analysis and returned positive for organophosphate compounds. Following this, the patient's family members, upon further prompting and detailed questioning, discovered an empty bottle of an organophosphate-pyrethroid combination insecticide (Ethion + Cypermethrin) at the patient's residence, providing definitive confirmation of the exposure.

4.4 Definitive Treatment and Outcome

Once the diagnosis of OP poisoning was confirmed, the patient was commenced on an intravenous atropine infusion, titrated to clinical endpoints of drying of

secretions and mydriasis, along with an intravenous pralidoxime (2-PAM) infusion as oxime therapy to reactivate acetylcholinesterase before irreversible aging of the enzyme could occur. The patient demonstrated significant clinical improvement with this specific antidotal therapy, showing progressive recovery of muscle power, resolution of ptosis, and improvement in respiratory parameters, eventually allowing successful weaning from mechanical ventilation.

5. DISCUSSION

This case serves as a powerful example of an atypical presentation of organophosphate poisoning that posed a genuine diagnostic challenge in the emergency department setting. Several features of this case are particularly instructive and merit careful documentation and discussion.

5.1 Atypical Features

The classical presentation of organophosphate poisoning involves a readily recognizable cholinergic toxidrome [1,3]. In our patient, however, several hallmark features were conspicuously absent or delayed at the time of initial presentation. The characteristic garlic-like or petroleum-like odor commonly associated with OP compounds was not detected at any point during the initial evaluation. The heart rate was 90 bpm, which was neither bradycardic nor significantly altered from normal. The initial pupil size was 2.0 mm and reactive, with miosis only becoming apparent on serial examination as the pupils progressively constricted to 1.5 mm with sluggish reactivity. Excessive secretions, including salivation, lacrimation, and bronchorrhea, were not prominently present. Instead, the dominant presenting features were gastrointestinal in nature (diarrhea and vomiting), followed by nicotinic manifestations (muscle weakness, ptosis, and respiratory failure), which effectively overshadowed whatever muscarinic features may have been present [2,4].

5.2 The Temporal Sequence: Muscarinic to Nicotinic Progression

The clinical evolution of this case is entirely consistent with the known pathophysiology of OP poisoning, in which muscarinic effects (diarrhea and vomiting) typically precede nicotinic effects (muscle weakness, fasciculations, and respiratory failure) [1,2]. In our patient, the gastrointestinal symptoms preceded the onset of quadriparesis by approximately twelve hours. Looking back, this temporal sequence is characteristic of OP poisoning, but at the time of presentation it was attributed to acute gastroenteritis with secondary hypokalemic paralysis.

The specific compound involved, Ethion combined with Cypermethrin, is a combination organophosphate-pyrethroid insecticide that is commonly available in Indian agricultural settings. Ethion, the organophosphate component, is a moderately toxic compound classified as WHO Class II, and it works by inhibiting acetylcholinesterase [1]. Cypermethrin, the pyrethroid component, acts on voltage-gated sodium channels and may contribute to neurotoxicity through an entirely different mechanism, which could potentially explain some of the atypical features observed in this case.

5.3 Diagnostic Confounders

Several factors contributed to the diagnostic delay in this case. The history of snakebite one week before presentation introduced a significant diagnostic confounder, directing initial suspicion toward delayed neurotoxic envenomation. The documented hypokalemia (2.50 mmol/L) at the referring hospital provided a plausible explanation for the muscle weakness, particularly given the profuse diarrhea. Additionally, the absence of any reported history of pesticide exposure or suicidal intent initially pointed away from a toxicological etiology. It was only after the family discovered the empty insecticide container at the patient's residence that the exposure was confirmed, raising the possibility of either accidental occupational exposure given his occupation as a farmer, or deliberate self-harm [7,10].

5.4 Differential Diagnosis of Acute Flaccid Quadriparesis

The presentation of acute flaccid quadriparesis with areflexia, ptosis, and respiratory failure requires consideration of a broad differential diagnosis [3,4]. Hypokalemic periodic paralysis typically presents with episodic weakness in association with low serum potassium, and correction of hypokalemia usually results in rapid improvement. In our case, normalization of potassium did not resolve the weakness. Neurotoxic snake envenomation, particularly by elapid species, can produce descending paralysis with ptosis and respiratory failure; however, the WBCT20 was normal and the temporal profile did not match typical envenomation kinetics. Guillain-Barré syndrome presents with ascending weakness and areflexia but typically follows an infection by one to four weeks and demonstrates albuminocytological dissociation in the cerebrospinal fluid. Myasthenia gravis presents with fatigable weakness and ptosis, but the negative atropine-neostigmine challenge argued strongly against this diagnosis. Botulism produces descending paralysis with

bulbar involvement but is typically associated with contaminated food consumption and fixed mydriasis rather than miosis [5,6,12].

5.5 The Role of Serial Re-examination

This case powerfully illustrates the critical importance of serial clinical re-examination in the emergency department, particularly when a patient's condition fails to improve as expected [3,11]. The evolving miosis, progressing from 2.0 mm reactive pupils at initial assessment to 1.5 mm sluggishly reactive pupils on subsequent examination, was the pivotal clinical finding that redirected the entire diagnostic workup toward organophosphate poisoning. This finding, combined with the triad of diarrhea, muscle weakness, and respiratory failure, prompted the targeted investigations that ultimately confirmed the diagnosis.

5.6 Diagnostic Value of Serum Pseudocholinesterase

Serum pseudocholinesterase (butyrylcholinesterase) serves as a readily available and reliable biomarker for OP exposure [1,8]. In our patient, the level was profoundly reduced at 103 U/L, representing approximately 1 to 2% of the lower limit of the normal range, and confirming significant cholinesterase inhibition. While erythrocyte acetylcholinesterase (true cholinesterase) is a more specific marker, serum pseudocholinesterase is far more widely available and correlates well with clinical severity [9]. The markedly suppressed level in our patient was entirely consistent with significant OP exposure and provided strong justification for the initiation of specific antidotal therapy.

5.7 Clinical Implications and Lessons Learned

This case offers several important lessons for emergency physicians. First, toxidromes cannot be reliably excluded on the basis of absent classical features alone; atypical presentations are more common than generally appreciated and must always be considered [2,4]. Second, in agricultural communities, organophosphate exposure should always remain in the differential diagnosis of unexplained acute flaccid paralysis, regardless of whether a toxicological history is initially forthcoming [7,10]. Third, serial clinical re-examination is an invaluable diagnostic tool that can reveal evolving signs not apparent at the time of initial presentation. Fourth, a systematic approach to the differential diagnosis of acute quadriparesis, with stepwise exclusion of competing diagnoses, will ultimately lead to the correct diagnosis even in the most challenging of cases [3,12].

6. CONCLUSION

We present a diagnostically challenging case of organophosphate poisoning in a 40-year-old male farmer who arrived at the emergency department with acute flaccid quadriparesis, bilateral ptosis, and respiratory failure following a prodromal phase of profuse diarrhea. The absence of classical cholinergic features, a misleading history of recent snakebite, and initial findings of hypokalemia directed the diagnostic workup away from the true etiology. It was only through diligent serial clinical re-examination, which uncovered progressive miosis, that the correct diagnosis was suspected and subsequently confirmed by markedly reduced serum pseudocholinesterase levels and positive gastric aspirate toxicology. This case underscores the fact that organophosphate poisoning should be considered in the differential diagnosis of any patient presenting with acute flaccid paralysis and respiratory failure, particularly in agricultural settings, even when classical cholinergic toxidrome features are entirely absent. Vigilant serial clinical re-examination remains an indispensable diagnostic tool in the emergency department.

DECLARATIONS

Ethical Approval and Consent

Written informed consent was obtained from the patient and his family for publication of this case report and any accompanying images. Patient identifying information has been anonymized to protect privacy.

Conflicts of Interest

The authors declare no conflicts of interest.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Author Contributions

Dr. Anas TC was involved in the clinical management of the patient, conceptualization of the case report, data collection, literature review, and manuscript preparation.

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