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Suspected Organophosphate Poisoning in a One-Year-Old Caucasian Dog: A Case Report

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Abstract **Case Studies**

Background: Organophosphate poisoning remains a critical concern in veterinary medicine, particularly among dogs exposed to pesticide-based chemicals. This report describes the clinical presentation, diagnostic findings, and management of a one-year-old female Caucasian dog, Nelly, suspected of organophosphate toxicity following exposure to otapiapia®.

Case Presentation: Nelly presented with severe weakness, recumbency, and central nervous system depression. Clinical examination revealed hyperthermia (41.6°C), tachycardia (112 bpm), and tachypnea (70 breaths/min), accompanied by excessive salivation, lacrimation, vomiting, and diarrhea classical signs of organophosphate poisoning.

Diagnostics and Management: Hematological analysis showed normal red blood cell count and hemoglobin levels, with mildly elevated mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH), suggesting early hemoconcentration. Biochemical results revealed increased alanine aminotransferase (ALT) and alkaline phosphatase (ALP), indicative of hepatocellular injury. Treatment included atropine sulphate, activated charcoal, dextrose supplementation, and fluid therapy, combined with intensive supportive management over three days.

Outcomes: Within 72 hours, Nelly demonstrated significant clinical improvement, with normalization of vital parameters and enzyme levels. By two weeks post-treatment, the dog had fully recovered without residual symptoms.

Conclusion: This case underscores the diagnostic and therapeutic challenges of organophosphate toxicity in dogs. Prompt recognition and early administration of atropine, coupled with supportive therapy, were pivotal to recovery. The case highlights the need for vigilant clinical monitoring and further research to enhance diagnostic accuracy and management strategies for organophosphate poisoning in veterinary practice.

Keywords: Organophosphate poisoning, pathophysiology, Diagnosis, Therapy.

Introduction: Overview of Organophosphate Poisoning

Organophosphate (OP) poisoning presents a significant toxicological concern in both human and veterinary medicine, often leading to severe cholinergic crises and potentially fatal outcomes if not promptly addressed (Bereda, 2025; Kuzucu, 2024). The widespread use of OP compounds as pesticides, particularly in agricultural settings, contributes to their prevalence as both accidental and intentional toxins, thereby posing a substantial public health challenge, especially in developing countries (Setiawan et al., 2022).

The central mechanism underlying organophosphate toxicity is the irreversible inhibition of acetylcholinesterase (AChE), a critical enzyme responsible for the hydrolysis of the neurotransmitter acetylcholine (ACh) at cholinergic synapses (Altaf et al., 2021). Under normal conditions, AChE swiftly degrades ACh into choline and acetate, regulating neurotransmission with precision (Singh et al., 2024). However, OP compounds, which structurally resemble ACh, bind covalently to the serine hydroxyl group in the active site of AChE, a process known as phosphorylation (Kwong, 2002). This interaction inactivates the enzyme, significantly impairing its ability to degrade ACh, leading to its accumulation in the synaptic cleft (Altaf et al., 2021).

The resultant accumulation of acetylcholine causes overstimulation of muscarinic and nicotinic cholinergic receptors in both the central and peripheral nervous systems (Bereda, 2025; Kuzucu, 2024). The clinical manifestations of OP poisoning, collectively termed acute cholinergic syndrome, are a direct result of this unregulated cholinergic activity (Liang et al., 2022). Common symptoms include muscarinic effects such as excessive salivation (sialorrhea), lacrimation, emesis, miosis, bradycardia, bronchoconstriction, and diarrhea. Nicotinic effects manifest as muscle fasciculations, tremors, weakness, and flaccid paralysis (Liang et al., 2022; Rizzo et al., 2024). Central nervous system involvement may present as confusion, seizures, and respiratory depression, the latter being a leading cause of mortality (Kuzucu, 2024).

The severity of symptoms and the duration of clinical signs depend on various factors, including the specific OP compound involved, the route and extent of exposure, and the individual's susceptibility (Song et al., 2013). In veterinary contexts, particularly in dogs, exposure to OPs often occurs through environmental contamination, ingestion of contaminated bait, or improper pesticide application (Shih et al., 2019). An in-depth understanding of the molecular and biochemical mechanisms of OP toxicity is essential for the timely diagnosis and effective management of these cases. Treatment typically involves atropine administration to block muscarinic receptors and oximes to reactivate inhibited AChE. However, the management of OP poisoning may be prolonged, requiring intensive care and extended atropine therapy, with some cases necessitating up to 60 days of treatment (Bashyal et al., 2023).

The development of advanced biosensing technologies, such as those utilizing graphene quantum dots and silver nanoparticles, aims to provide highly sensitive and selective methods for detecting organophosphorus pesticides. These innovations highlight the ongoing need for rapid diagnostic tools in both clinical and environmental settings (Qin et al., 2024; Z. Zhang et al., 2021).

OP poisoning can also lead to long-term sequelae, including organophosphate-induced delayed polyneuropathy (OPIDP), characterized by weakness, pain, and tingling sensations, which may emerge weeks following acute exposure (Wasedar et al., 2022). This broad spectrum of effects underscores the complexity of OP toxicity, necessitating comprehensive clinical and research efforts to improve outcomes for affected patients. In the case of Caucasian dogs, rapid diagnosis and the implementation of veterinary-specific management protocols are paramount for effective treatment and recovery.

Objectives of the Case Report:

1. Primary Objective:

To provide a comprehensive and detailed clinical case report of suspected organophosphate (OP) poisoning in a one-year-

old Caucasian dog, including a thorough description of the initial clinical presentation, diagnostic procedures, therapeutic interventions, and clinical outcomes. This report aims to contribute to the body of knowledge regarding the recognition and management of OP poisoning in canines, offering valuable insights into its pathophysiology, clinical signs, and the therapeutic approaches used in the management of such cases.

2. Secondary Objective:

To critically examine and analyze the diagnostic challenges encountered in the identification and management of suspected organophosphate poisoning in canines. This includes a discussion of the limitations of available diagnostic methods, the complexities of clinical presentation, and the role of differential diagnosis in ruling out other potential causes of toxicity. This objective aims to highlight areas in veterinary practice where further research and refinement of diagnostic strategies are needed to enhance clinical outcomes.

Case Presentation:

1. Signalment

- **Species**: Canine
- **Breed**: Caucasian
- **Name**: Nelly
- **Color**: Grey
- **Age**: 1 year, 5 months
- **Sex**: Female
- **Weight**: 28 kg

2. History

Nelly was acquired at seven weeks of age from Ilorin, where she has since served as both a pet and a guard dog. She is typically fed three times daily with indomie® and canned beef, and her water source is a borehole located within the compound. Additionally, Nelly has a complete record of vaccinations, which provides insight into her health status and preventive care. There have been no previous reports of major illnesses, and her general health has been stable. On the 19th of July, 2024, she was presented to the Small Animal Clinic at the University of Ilorin Veterinary Teaching Hospital with symptoms suggestive of poisoning.

3. Chief Complaint

Nelly was found recumbent within her cage approximately 5-6 hours after an organophosphate-based chemical, **otapiapia®**, was sprayed around her cage for tick control. The owner observed her in a state of severe weakness and immediately sought veterinary attention.

4. Vital Parameters on Presentation

- **Temperature**: 41.6°C (normal range: 37.5-39.5°C)
- **Respiratory Rate**: 70 cycles/min (normal range: 18-34 cycles/min)
- **Pulse Rate**: 112 beats/min (normal range: 70-140 beats/min)

These elevated vital signs are consistent with systemic toxicity and stress due to organophosphate exposure.

5. Physical Examination Findings

Upon presentation, Nelly exhibited the following clinical signs:

- **Weakness and recumbency**: The Dog was unable to stand and displayed signs of central nervous system depression.
- **Dullness**: A reduced level of responsiveness and activity was noted.
- **Congested ocular and oral mucous membranes**: This is indicative of circulatory compromise and possibly associated with the toxicosis.
- **Presence of dead ticks in the ears**: A sign of recent pesticide exposure, suggesting that the chemical application may have been effective in eliminating ticks, but also potentially harmful to the dog.

6. Clinical Signs

Nelly exhibited several characteristic signs of organophosphate toxicity:

- **Weakness and recumbency**: A hallmark of organophosphate poisoning, likely due to the inhibition of acetylcholinesterase, leading to an accumulation of acetylcholine at synaptic junctions.
- **Salivation**: Excessive salivation, or **ptyalism**, is a common sign of cholinergic toxicity.
- **Lacrimation**: The profuse tearing indicates the activation of parasympathetic responses due to acetylcholine accumulation.
- **Vomiting and defecation**: Gastrointestinal signs, such as vomiting and diarrhea, are frequently seen in cases of poisoning and reflect the systemic nature of organophosphate toxicity.

Investigations:

The laboratory tests conducted include complete blood count (CBC), liver function tests (LFTs), renal function tests as the results are given below.

Table 1: The complete blood count (CBC)

CRITERIA	VALUE	NORMAL
RBC ($X10^{12}$/L)	5.8	4.95-7.87
HAEMOGLOBIN (x10 g/L)	16.0	11.9-18.9
PCV (%)	49	35-57
MCV (fL)	84	66-77
MCH (pg)	27.6	21.0-26.2
MCHC (x10 g/L)	32.7	32.0-36.3
WBC ($X10^9$/L)	9.5	5.0-14.1
NEUTROPHILS ($X10^9$/L)	6.46	2.9-12.0
BANDS ($X10^9$/L)	0.475	0-0.45
LYMPHOCYTES ($X10^9$/L)	2.47	0.4-2.9
MONOCYTES ($X10^9$/L)	1	0.1-1.4
EOSINOPHILS ($X10^9$/L)	NIL	0-1.3
PLATELETS ($X10^9$/L)	NIL	211-621
MPV (fL)	-	6.1-10.1

The table presents hematological parameters recorded at presentation compared with normal canine reference ranges. Findings indicate normal red cell indices with mild elevations in mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH), suggestive of early hemoconcentration. A slight increase in band neutrophils and absent platelet count was also observed, potentially reflecting acute toxic stress and transient thrombocytopenia.

The hematology results for the one-year-old Caucasian suspected of organophosphate poisoning reveal a normal RBC count, hemoglobin level, and WBC count (Table 1). However, both MCV and MCH are elevated, while platelets are absent. Additionally, the neutrophil and monocyte count remain within normal ranges, and there is no indication of eosinophilia.

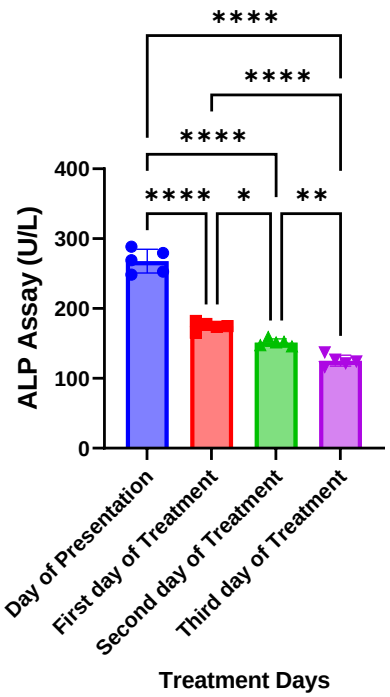


Figure 1: Activities of ALP across three days of treatment

Significant elevation in ALP levels was observed on the first day of presentation compared to subsequent treatment days ($p < 0.001$). A noticeable reduction in ALP levels was recorded over the three days of treatment, with a significant decrease on the third day (Figure 1). The ALP activity on the third day of treatment was significantly lower than on the first and second days ($p < 0.001$ and $p < 0.01$, respectively)

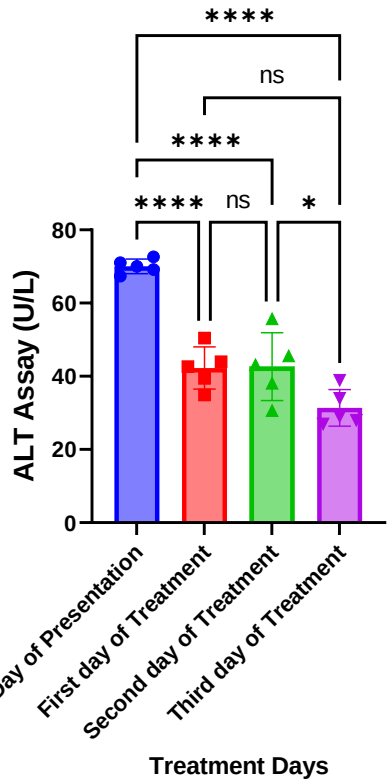


Figure 2: Activities of ALT across three days of treatment

Figure 2 shows a significant reduction in ALT levels on the first day of treatment compared to the day of presentation ($p < 0.001$). However, on the second day, ALT levels remained similar to those on the first day ($p > 0.05$). By the third day, ALT levels significantly decreased compared to the day of presentation ($p < 0.001$). The ALT activity on the third day was significantly lower compared to the second day of treatment ($p < 0.05$). However, no significant difference was observed in ALT activity on the third day when compared to creatinine levels on the first day of treatment ($p > 0.05$). Abbreviation: ns = not significant.

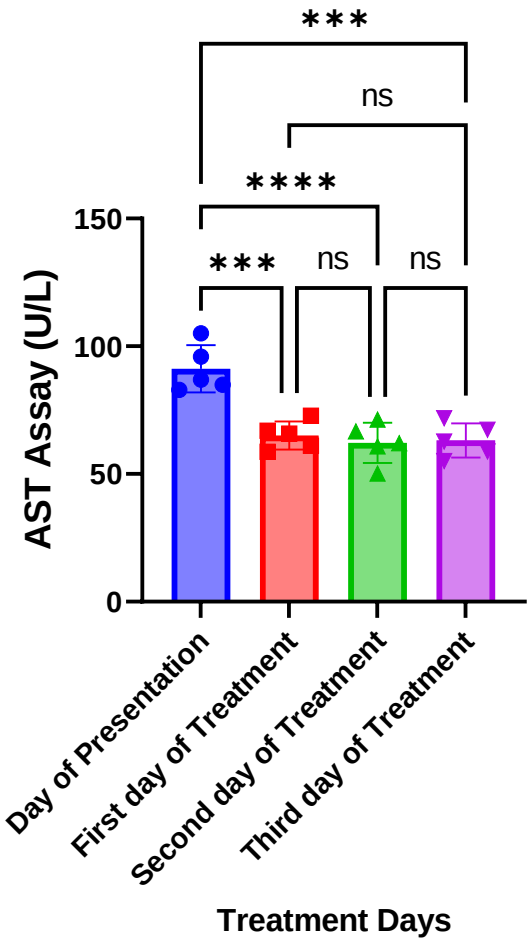


Figure3: Activities of AST across three days of treatment

Figure 3 shows significantly elevated AST activity on the day of presentation compared to the treatment days ($p < 0.01$). However, no significant difference was observed in AST levels across the three treatment days ($p > 0.05$). Abbreviation: ns = not significant.

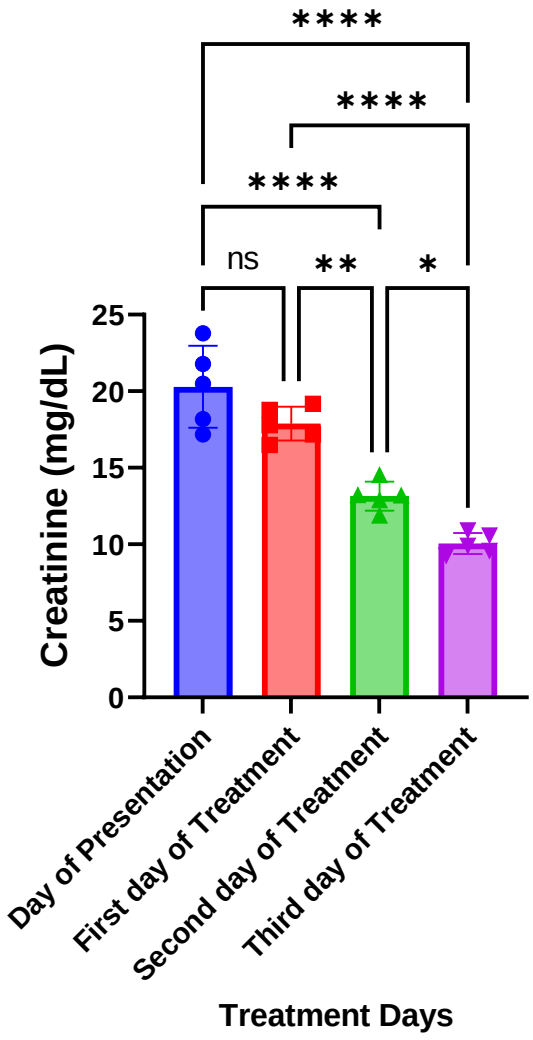


Figure 4: Activities of Creatinine across three days of treatment

Figure 4 shows significantly elevated creatinine activity on the day of presentation compared to the second and third treatment days ($p < 0.01$). Creatinine activity on the third day of treatment was significantly lower compared to the first and second days of treatment ($p < 0.001$ and $p < 0.05$, respectively).

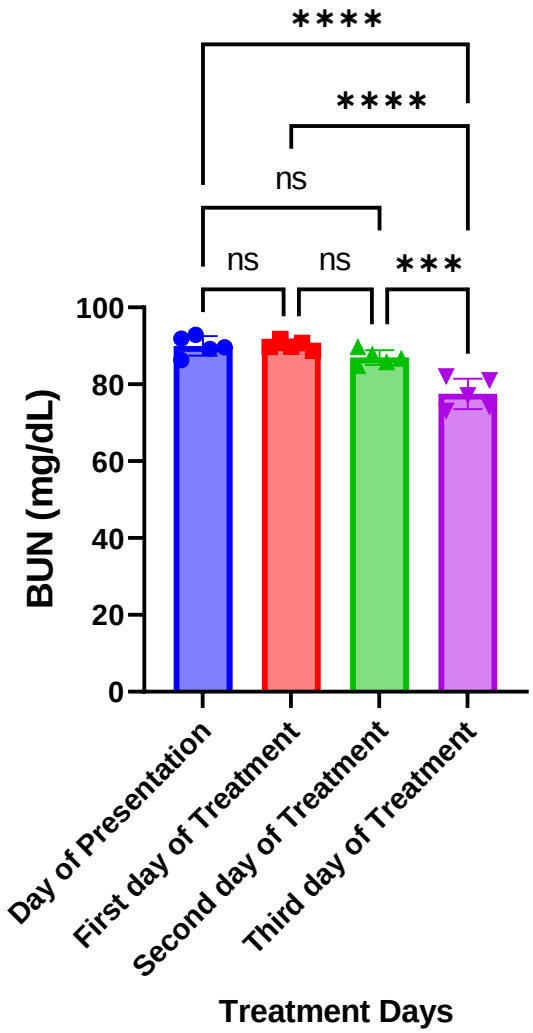


Figure 5: Activities of BUN across three days of treatment

Figure 5 shows a significant elevation in BUN levels on the day of presentation compared to the third day of treatment. BUN activity on the third day of treatment was significantly lower compared to the second day of treatment ($p < 0.01$). However, no significant difference was observed in BUN activity between the day of presentation and the first or second day of treatment ($p > 0.05$).

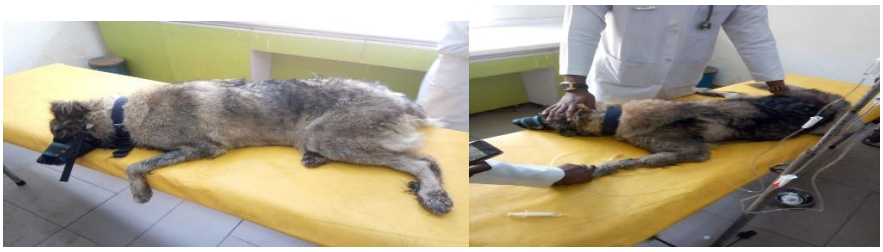
Medical Management and Outcome Report

Nelly was successfully treated for organophosphate poisoning over a three-day period. The treatment included the administration of atropine, ascorbic acid, dextrose, lactated ringers, and vitamin B complex, in addition to supportive care. Follow-up monitoring showed significant improvement in her clinical condition, with no signs of complications or relapse. The recovery process was completed without further issues, and the patient has been clear of symptoms two weeks post-treatment. The details of the treatment are highlighted below.

➢ **DAY 1 - Initial Presentation and Management**

Chief Complaint:

Nelly was presented with symptoms consistent with organophosphate poisoning (*Figure 6*).



(a) (b)

Figure 6: The dog was recumbent and displayed signs of central nervous system depression, a and b.

Initial Management Plan:

• Atropine Sulphate: 0.5 mg/kg IV (1/3) and SC (2/3) until atropinization achieved

• Activated Charcoal: 50 mL orally (PO)

• Normal Saline: 200 mL intravenously (IV)

• Ascorbic Acid: 500 mg intramuscularly (IM) x 3 days

• Bathing: Nelly was bathed with tepid water and soft soap.

Vital Parameters after First Management:

• Temperature: 39.6°C

• Respiratory Rate: 60 cycles/min

• Pulse Rate: 80 beats/min

Follow-up and Client Report on DAY 1

Client's Report:

• The client reported that Nelly drank only water and glucose after the treatment.

• The client noted that Nelly vomited twice the morning following treatment.

Vital Parameters:

• Temperature: 38.9°C

• Pulse Rate: 120 beats/min

• Respiratory Rate: 64 cycles/min

➢ **DAY 2 - Continued Management and Observation**

Chief Complaint:

Client reported that Nelly showed signs of shivering while being transported to the hospital on the morning of the second day (*Figure 7*).

Management Plan:

• 10% Dextrose: 150 mL IV

• Lactated Ringers: 100 mL IV

• Ascorbic Acid: 500 mg IM x 2/3 days

• Atropine Sulphate: 0.2 mg/kg SC

• Vitamin B Complex: 4 mL IM x 2/7 days

Vital Parameters after Day 2 Management:

• Temperature: 39.6°C

• Respiratory Rate: 46 cycles/min

• Pulse Rate: 125 beats/min

Follow-up and Client Report:

• Client reported shivering in the morning on the way to the hospital.

Vital Parameters on Follow-up:

• Temperature: 38.4°C

• Pulse Rate: 132 beats/min

• Respiratory Rate: 64 cycles/min

Figure 7: The dog was evidently shivering on the second day of presentation

➢ **DAY 3 - Final Stages of Treatment and Monitoring**

Chief Complaint:

No new complaints were reported by the client on the third day.

Management Plan:

• 10% Dextrose Saline: 150 mL IV

• Lactated Ringers: 100 mL IV

• Atropine Sulphate: 0.2 mg/kg SC

• Vitamin B Complex: 4 mL IM x 1/7 days

• Ascorbic Acid: 500 mg IM x 3/3 days

Follow-up and Client Report:

• Client Report: On Day 3, the client reported that Nelly had regained her appetite and ate indomie and fish that morning.

• After 3 days of treatment, the client called to report that Nelly was alert and active (*Figure 8*).

(a) (b)

Figure 8: The dog's condition has improved with absence of clinical signs associated with organophosphate poisoning, a and b.

Recovery and Outcome

Recovery Observations:

• No complications were observed during the recovery process.

• Nelly's condition steadily improved following the treatment plan, with no signs of organophosphate poisoning remaining.

Follow-up:

• Two weeks post-treatment, the dog remained free of clinical signs of organophosphate poisoning.

Discussion

The laboratory findings from this case strongly support a diagnosis of organophosphate poisoning in a one-year-old Caucasian dog, as evidenced by the absence of significant hemoconcentration or anemia (Schmid et al., 2023). The normal red blood cell (RBC) count and hemoglobin levels indicate that significant hemolysis or anemia did not occur, which is typical in cases of acute organophosphate

toxicity, where hemolysis is usually not a prominent feature unless the exposure is either prolonged or severe (Williams et al., 2023). Furthermore, the lack of substantial alterations in the white blood cell (WBC) count suggests the absence of an acute inflammatory response or secondary infection, both of which are commonly seen in more severe cases of toxicity or infections (Moutinho et al., 2024).

An intriguing observation was the elevation in mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH), which could indicate early hemoconcentration or alterations in erythropoiesis, often a response to systemic stress. These non-specific changes may reflect the toxic effects of organophosphates on bone marrow function or fluid shifts within the body (Ma et al., 2024). Additionally, the observed thrombocytopenia is significant, as it is a well-known feature of severe organophosphate poisoning. This platelet depletion may arise from either direct toxic effects on the bone marrow or platelet consumption due to systemic vascular injury caused by the toxins (Hung et al., 2021; Ma et al., 2024).

The immune parameters in this case are also noteworthy. Normal levels of neutrophils and monocytes in the presence of suspected organophosphate poisoning suggest that no severe bacterial infection or systemic inflammatory response was present at the time of assessment (Schmid et al., 2023). Neutrophils and monocytes are crucial for the innate immune response, with their levels typically increasing during bacterial infections or significant inflammatory conditions (Cai et al., 2024; Yen et al., 2024). Elevated neutrophil counts (neutrophilia) are common in dogs suffering from stress, inflammation, or infections (D. Zhou et al., 2024). Monocytes, which differentiate into macrophages in tissues, also play an essential role in immune surveillance and the resolution of inflammation (Jansen et al., 2016; Q. Zhou et al., 2024).

The normal levels of neutrophils and monocytes in this case suggest that either the immune dysregulation caused by organophosphate toxicity was mild or localized, or that the measured parameters do not fully capture the nuanced immune responses, such as functional changes within these cells (Mishra et al., 2017; D. Zhou et al., 2024). Additionally, early intervention with supportive therapy may have mitigated a more pronounced inflammatory response that is sometimes observed in severe organophosphate toxicity (Schmid et al., 2023).

Alkaline phosphatase (ALP) is a crucial enzyme found in various tissues, including the liver, bone, intestine, and kidney (Li et al., 2019; Mailafiya et al., 2019). In dogs, elevated ALP levels are commonly linked to cholestasis, hepatocellular damage, or corticosteroid activity (Huang et al., 2024; Li et al., 2019). In this case, the initial elevation of ALP strongly indicates acute hepatotoxicity associated with organophosphate exposure (Schmid et al., 2023). Organophosphates such as coumaphos have been previously shown to induce hepatotoxicity, as evidenced by elevated liver enzymes, including ALP, alanine aminotransferase (ALT), and aspartate aminotransferase (AST), following acute exposure in dogs (Ola-Davies et al., 2018). This hepatotoxicity is likely a result of direct cellular damage or disruption in liver metabolism due to the organophosphate compound (Munkong et al., 2024; Ola-Davies et al., 2018). The correlation between ALP elevation and liver injury is well-established in veterinary and human medicine (Delignette et al., 2024; Li et al., 2019).

The subsequent decrease in ALP levels during the treatment period is indicative of a positive response to supportive therapy, suggesting a recovery of liver function as the effects of the organophosphate toxin diminished (Schmid et al., 2023). This pattern of enzyme reduction following therapy is commonly seen when the cause of liver injury is addressed, allowing for the restoration of hepatic function (Huang et al., 2024). The observed improvement in ALP levels suggests that therapeutic measures were effective in preventing further liver damage and promoting hepatic repair (Munkong et al., 2024).

In parallel, elevated levels of ALT and AST are frequently observed in cases of liver injury, including those induced by organophosphate poisoning (Q. Shi et al., 2024; Wei et al., 2025). ALT, being liver-specific, is a reliable indicator of hepatocellular damage (Hu et al., 2023; M. Q. Zhang et al., 2023). The significant elevation of

ALT in this case is consistent with acute hepatocellular injury following organophosphate exposure, further supporting the diagnosis of hepatotoxicity (Gao et al., 2023; Yan et al., 2024). The marked reduction in ALT levels after initiation of therapy suggests a favorable therapeutic response and restoration of liver function (Hu et al., 2023). This is characteristic of a reversible form of liver injury, where the liver's regenerative capacity is activated once the inciting agent is removed or effectively managed (Ezquer et al., 2017; Ulmer et al., 2017).

While AST levels also rose initially, they did not show significant changes over the course of treatment. AST, being less specific to the liver, can also reflect damage to other tissues, including the heart, skeletal muscle, and kidneys (Fontana et al., 2023; Gao et al., 2023). The persistence of elevated AST levels suggests that the injury may not have been limited to the liver, and that other tissues may have been affected by the organophosphate poisoning or the treatment (Abouzahir et al., 2024). Alternatively, the stable AST levels could indicate that the clearance kinetics of AST from non-hepatic tissues differ from those of ALT, or that the therapeutic interventions did not fully mitigate the damage to extra-hepatic tissues (Abouzahir et al., 2024; Ayyed et al., 2022).

Renal function, as assessed by biomarkers such as creatinine and blood urea nitrogen (BUN), is critical in cases of systemic toxicity like organophosphate poisoning. The initial elevation of creatinine levels suggests the potential for acute kidney injury (AKI) or significant renal stress, both of which are common sequelae of systemic toxic insults (Farokhi et al., 2025; Z. Shi et al., 2024). Elevated creatinine typically reflects a reduction in glomerular filtration rate (GFR) due to nephrotoxic damage or other systemic effects, such as hypoperfusion or dehydration (Dhama et al., 2019). In this case, the elevated creatinine levels suggest either direct nephrotoxic effects of the organophosphate or secondary renal stress resulting from hypoperfusion and systemic inflammation (Plotnikov et al., 2019; Xu et al., 2017).

The significant decrease in creatinine levels following treatment is a positive prognostic indicator, suggesting that renal function improved as the body metabolized and excreted the organophosphate toxin, and as supportive therapies such as fluid resuscitation restored renal perfusion (Xu et al., 2017). This decrease in creatinine over time suggests that the renal insult was reversible and that the therapeutic interventions were effective in reversing the renal dysfunction (Mansoure et al., 2024).

Similarly, elevated BUN levels are often a sign of renal compromise or dehydration, which can be exacerbated by toxic exposures or kidney dysfunction (Mahmood et al., 2024). The observed decrease in BUN levels over the course of treatment further suggests that fluid therapy played a critical role in improving renal perfusion, which in turn facilitated the kidney's ability to excrete toxins and improve glomerular filtration (Xu et al., 2017). The combined resolution of both elevated creatinine and BUN levels is a strong indicator of improving renal function and recovery from acute renal insult in the context of organophosphate poisoning (Farokhi et al., 2025; Kadono et al., 2023).

Conclusion

Organophosphate poisoning remains a significant, yet frequently underdiagnosed, cause of acute toxicity in veterinary practice, particularly in canines exposed to pesticide chemicals. This case report underscores the importance of early clinical recognition and prompt intervention to mitigate the potentially fatal consequences of organophosphate toxicity. The case of a one-year-old Caucasian dog, Nelly illustrates the characteristic signs of cholinergic toxicity, including central nervous system depression, excessive salivation, lacrimation, and gastrointestinal disturbances, all of which were strongly suggestive of organophosphate exposure.

The successful treatment of this case, which included atropine administration, fluid resuscitation, and supportive care, demonstrates the efficacy of early intervention in preventing irreversible organ damage. The patient's full recovery within two weeks highlights the critical role of early diagnosis, continuous monitoring, and multi-modal therapy in managing organophosphate poisoning.

Laboratory findings further corroborated the diagnosis, with abnormal liver enzyme profiles, kidney markes and hematological changes, including mild hemoconcentration and thrombocytopenia, consistent with known pathophysiological responses to organophosphate toxicity. The resolution of these clinical signs and laboratory abnormalities following treatment suggests that the toxic effects are reversible when promptly addressed.

This case also underscores the diagnostic challenges associated with recognizing organophosphate poisoning, particularly in the presence of non-specific clinical signs. Differential diagnosis is essential to exclude other toxicities or systemic diseases. Further research is needed to refine diagnostic protocols, especially in field settings where access to advanced diagnostic tools may be limited.

Take-Home Message

Organophosphate poisoning in canines poses a significant diagnostic challenge due to the non-specific nature of its clinical signs, which often mimic other systemic diseases. This case report highlights the critical importance of timely recognition of organophosphate toxicity, particularly when there is a history of pesticide exposure. Key clinical indicators such as weakness, excessive salivation, lacrimation, vomiting, and gastrointestinal distress should raise immediate suspicion and prompt diagnostic evaluation for organophosphate poisoning.

Veterinarians should maintain a high index of suspicion and adopt a multi-modal treatment approach, including atropine administration, fluid therapy, and supportive care. The rapid recovery observed in this case emphasizes the effectiveness of early intervention, which can significantly improve prognosis and prevent irreversible organ damage.

Additionally, laboratory findings such as elevated liver enzymes, kidney markers and hematological abnormalities are crucial diagnostic tools, but they must be interpreted within the context of the clinical presentation and a history of pesticide exposure. While organophosphate poisoning can cause significant systemic effects, it remains treatable with appropriate veterinary care.

Clinicians should consider organophosphate toxicity in their differential diagnosis when presented with cases involving potential pesticide exposure, and act promptly to optimize outcomes. Ongoing research into more refined diagnostic tools and therapeutic strategies will continue to enhance the clinical management of organophosphate poisoning in veterinary practice.

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