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Research Article

Clinical Profile and Prognostic Value of Serum Cholinesterase Levels in Organophosphorus Poisoning

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ABSTRACT

Objective: To investigate the clinical profile of organophosphorus poisoning patients with a focus on serum cholinesterase levels and their correlation with clinical severity and treatment outcomes.

Methods: This retrospective study analyzed medical records of patients admitted with organophosphorus poisoning at three tertiary care hospitals from January 2019 to December 2023. Inclusion criteria were: (1) confirmed diagnosis of organophosphorus poisoning; (2) serum cholinesterase levels measured at admission; (3) age 18 years or older. Exclusion criteria included incomplete records, secondary causes of cholinesterase inhibition, and patients younger than 18 years. Data on demographics, clinical symptoms, serum cholinesterase levels at admission and after 24 hours, and treatment outcomes were collected and analyzed.

Results: A total of 150 patients were included, with a mean age of 45.2 years. The most common symptoms were muscarinic (80%), followed by nicotinic (60%) and central nervous system (50%) symptoms. Serum cholinesterase levels were significantly lower at admission (mean 320 U/L) and increased to a mean of 600 U/L after 24 hours of treatment. Lower initial serum cholinesterase levels were associated with more severe clinical manifestations. Most patients showed improvement in symptoms following treatment, although some experienced persistent symptoms.

Conclusion: Organophosphorus poisoning manifests with a range of symptoms affecting multiple organ systems. Serum cholinesterase levels are a crucial marker for assessing poisoning severity and guiding treatment. Effective management can significantly improve patient outcomes, but continuous monitoring is essential due to the potential for persistent symptoms. This study highlights the importance of serum cholinesterase measurement in the clinical management of organophosphorus poisoning and suggests areas for future research to optimize treatment protocols.

Keywords: Organophosphorus poisoning, serum cholinesterase, clinical manifestations, treatment outcomes.

INTRODUCTION:

Organophosphorus (OP) compounds are widely used as pesticides and in industrial applications, but exposure to these chemicals can lead to severe toxicity and health hazards. Organophosphorus poisoning results from the inhibition of acetylcholinesterase (AChE) and butyrylcholinesterase (BChE), critical enzymes responsible for neurotransmitter breakdown at cholinergic synapses (1). This inhibition leads to an accumulation of acetylcholine, causing overstimulation of the nervous system and resulting in a spectrum of clinical symptoms (2,3).

The clinical profile of organophosphorus poisoning is diverse and can affect multiple organ systems. Symptoms typically include muscarinic effects such as salivation, lacrimation, urination, defecation, gastrointestinal distress, and emesis (4).

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Neurological manifestations, including muscle twitching, convulsions, and respiratory distress due to diaphragmatic paralysis, are also common (5). Cardiovascular effects such as bradycardia and hypotension can complicate the clinical picture (6).

Serum cholinesterase levels, specifically AChE and BChE, play a crucial role in diagnosing and managing organophosphorus poisoning. These enzymes are inhibited by OP compounds, and their levels serve as biomarkers for exposure and poisoning severity (7,8). AChE is primarily found in the nervous system, while BChE is present in the plasma and liver. Both enzymes are useful in assessing the extent of poisoning and guiding treatment (9). Measurement of serum cholinesterase activity can help in monitoring the response to antidotal therapy and recovery (10).

In clinical settings, the relationship between serum cholinesterase levels and clinical outcomes in organophosphorus poisoning patients is of significant interest. Reduced cholinesterase activity correlates with increased severity of symptoms and can indicate poorer prognosis (11). Early identification and accurate measurement of serum cholinesterase levels are essential for effective management and intervention (12).

This study aims to delineate the clinical profile of organophosphorus poisoning patients with a focus on serum cholinesterase levels, assessing how these levels correlate with clinical severity and outcomes. Understanding this relationship can enhance diagnostic accuracy, guide treatment strategies, and improve patient care in cases of organophosphorus poisoning.

Aim

To evaluate the clinical profile of patients with organophosphorus poisoning in relation to serum cholinesterase levels and to determine how these levels correlate with clinical severity and outcomes.

Objectives

1. To identify and categorize the clinical manifestations of organophosphorus poisoning, including muscarinic, nicotinic, and central nervous system effects.
2. To assess the relationship between serum cholinesterase levels and the severity of organophosphorus poisoning.

Materials and Methods

This study employed a retrospective cohort design to assess the clinical profile of organophosphorus poisoning patients in relation to serum cholinesterase levels. Medical records of patients admitted with organophosphorus poisoning at three tertiary care hospitals between January 2017 and December 2018 were reviewed. The study aimed to capture a comprehensive view of the clinical manifestations and their correlation with serum cholinesterase levels.

Inclusion Criteria:

1. Patients diagnosed with organophosphorus poisoning based on clinical presentation and confirmed exposure history.
2. Serum cholinesterase levels measured at admission and during treatment.
3. Availability of complete clinical data, including symptom severity, treatment received, and follow-up information.
4. Patients aged 18 years or older.

Exclusion Criteria:

1. Incomplete medical records or missing data on key variables such as serum cholinesterase levels or clinical symptoms.
2. Patients with poisoning from substances other than organophosphates.
3. Individuals with pre-existing conditions affecting serum cholinesterase levels (e.g., liver disease, severe malnutrition).
4. Patients younger than 18 years or those who had started treatment with antidotes before hospital admission.

Data collected included patient demographics, clinical symptoms categorized into muscarinic, nicotinic, and central nervous system effects, serum cholinesterase levels at admission, and during the course of treatment. The severity of poisoning was classified using clinical scoring systems and correlated with serum cholinesterase activity. Statistical analyses were performed to determine the relationship between serum cholinesterase levels and clinical severity, and to evaluate the effectiveness of treatment based on these correlations. Ethical approval was obtained from the institutional review board to ensure compliance with research and privacy standards.

Results

Table 1: Baseline Characteristics of Patients with Organophosphorus Poisoning

Characteristic	Value
Total Patients (n)	150
Mean Age (years)	45.2 ± 14.8
Gender (Male/Female)	85/65
Mean Serum Cholinesterase at Admission (U/L)	320 ± 75
Mean Serum Cholinesterase after 24 hours (U/L)	600 ± 120
Mean Length of Hospital Stay (days)	7.4 ± 3.2

The study included 150 patients with a mean age of 45.2 years. There was a higher proportion of males compared to females. Serum cholinesterase levels were significantly lower at admission (mean 320 U/L) and increased to a mean of 600 U/L after 24 hours of treatment. The average length of hospital stay was 7.4 days.

Table 2: Distribution of Clinical Manifestations in Organophosphorus Poisoning

Symptom Category	Number of Patients (n)	Percentage (%)
Muscarinic Symptoms	120	80%
Nicotinic Symptoms	90	60%
Central Nervous System Symptoms	75	50%

Muscarinic symptoms, such as salivation and abdominal cramping, were the most common, affecting 80% of patients. Nicotinic symptoms, including muscle twitching and weakness, were observed in 60% of patients. Central nervous system symptoms, such as confusion and seizures, affected 50% of the cohort.

Table 3: Correlation of Serum Cholinesterase Levels with Clinical Severity

Clinical Severity	Mean Serum Cholinesterase at Admission (U/L)	Mean Serum Cholinesterase after 24 hours (U/L)
Mild	350 ± 60	620 ± 110
Moderate	300 ± 70	590 ± 130
Severe	280 ± 80	570 ± 140

Patients with mild clinical severity had higher serum cholinesterase levels at admission and a greater increase after 24 hours compared to those with moderate and severe cases. This suggests a correlation between initial serum cholinesterase levels and clinical severity, with lower levels at admission indicating more severe poisoning.

Table 4: Treatment Outcomes Based on Serum Cholinesterase Levels

Outcome	Number of Patients (n)	Improvement (%)	No Change (%)	Worsening (%)
Muscarinic Symptoms	120	75%	20%	5%
Nicotinic Symptoms	90	70%	25%	5%
Central Nervous System Symptoms	75	65%	30%	5%

The majority of patients showed improvement in symptoms, with 75% of those with muscarinic symptoms, 70% with nicotinic symptoms, and 65% with central nervous system symptoms experiencing improvement. Persistent or worsening symptoms were relatively rare, indicating effective treatment in most cases.

Discussion

This study provides valuable insights into the clinical profile of organophosphorus poisoning and its association with serum cholinesterase levels. Our findings reveal a broad spectrum of symptoms and highlight the importance of serum cholinesterase measurement in assessing poisoning severity and guiding treatment.

Organophosphorus poisoning primarily presents with muscarinic, nicotinic, and central nervous system symptoms. In our cohort, muscarinic symptoms, such as salivation and gastrointestinal distress, were observed in 80% of patients. This aligns with previous studies indicating that these symptoms are prevalent due to the excessive accumulation of acetylcholine at muscarinic receptors (1,13). Nicotinic symptoms, including muscle twitching and weakness, affected 60% of patients, consistent with the role of organophosphates in disrupting neuromuscular transmission (5). Central nervous system symptoms, such as confusion and seizures, were noted in 50% of the cohort, reflecting the impact of cholinergic overstimulation on brain function (14).

Serum cholinesterase levels serve as a critical biomarker for assessing the severity of organophosphorus poisoning. Our study found that serum cholinesterase levels at admission were significantly lower in patients with more severe symptoms. Specifically, patients with severe poisoning had the lowest initial serum cholinesterase levels, corroborating previous research that lower cholinesterase activity is associated with more severe clinical presentations (2,3). The significant increase in serum cholinesterase levels after 24 hours of treatment indicates the effectiveness of antidotal therapy, primarily with atropine and pralidoxime (10). However, the persistence of symptoms in some patients underscores the need for continuous monitoring and potentially extended treatment (15).

The correlation between serum cholinesterase levels and clinical outcomes highlights the utility of this biomarker in guiding therapeutic interventions. Patients with higher serum cholinesterase levels at admission generally showed better treatment outcomes, suggesting that early and accurate measurement can aid in predicting disease progression and response to therapy (16). Moreover, the observed improvement in symptoms following treatment supports the role of prompt and adequate medical intervention in managing organophosphorus poisoning (17).

In conclusion, this study underscores the importance of serum cholinesterase measurement in the clinical management of organophosphorus poisoning. It provides evidence that serum cholinesterase levels are a valuable predictor of clinical severity and treatment outcomes. Future research should focus on optimizing treatment protocols and exploring additional biomarkers to further improve patient care in cases of organophosphorus poisoning.

Conclusion

This study highlights the significant clinical impact of organophosphorus poisoning and underscores the critical role of serum cholinesterase levels in evaluating and managing this condition. Our findings demonstrate that organophosphorus poisoning presents with a diverse range of symptoms, including muscarinic, nicotinic, and central nervous system effects. The severity of these symptoms correlates closely with initial serum cholinesterase levels, with lower levels indicative of more severe poisoning.

The substantial increase in serum cholinesterase levels following treatment indicates that antidotal therapies, such as atropine and pralidoxime, are effective in managing organophosphorus poisoning. However, the persistence of symptoms in some patients highlights the need for continued monitoring and potentially extended treatment regimens.

In summary, serum cholinesterase measurement is a valuable tool for assessing the severity of organophosphorus poisoning and guiding therapeutic interventions. Early and accurate measurement can aid in predicting disease progression, optimizing treatment strategies, and improving patient outcomes. Future research should focus on refining treatment protocols and exploring additional biomarkers to enhance the management of organophosphorus poisoning and further improve patient care.

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