

QT Interval Prolongation as a Prognostic Marker in Organophosphate Poisoning: A Prospective Follow-Up Study

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ABSTRACT

Background: Organophosphate poisoning is a common medical emergency in developing countries and is associated with significant morbidity and mortality. Cardiac complications, particularly electrocardiographic abnormalities, contribute substantially to adverse outcomes. Prolongation of the QT interval has been reported in organophosphate poisoning and may reflect the severity of toxicity and risk of complications.

Aim: To evaluate the role of QT interval prolongation as a prognostic marker in patients with organophosphate poisoning and to assess its association with clinical outcomes.

Study Design and Setting: This was a prospective follow-up study conducted in a tertiary care hospital over a period of three months.

Methods: Adult patients with confirmed organophosphate poisoning were enrolled. Standard 12-lead electrocardiograms were recorded on admission, and QT and corrected QT (QTc) intervals were calculated using Bazett's formula. Patients were categorized into normal QT and prolonged QT groups based on predefined cut-off values. Clinical parameters, laboratory investigations including serum cholinesterase levels, treatment details, need for ventilatory support, and outcomes were recorded and compared between the two groups.

Results: A significant proportion of patients with organophosphate poisoning demonstrated QT interval prolongation on admission. Patients with prolonged QT/QTc showed a higher requirement for ventilatory support, greater severity of illness, and poorer clinical outcomes compared to those with normal QT intervals. QT prolongation was also associated with lower serum cholinesterase levels and longer hospital stay.

Conclusion: QT interval prolongation is a useful, simple, and non-invasive prognostic marker in organophosphate poisoning. Early identification of patients with prolonged QT/QTc may help in risk stratification, closer monitoring, and timely intervention, thereby improving clinical outcomes.

Keywords: *Organophosphate poisoning; QT interval; QTc; Electrocardiography; Prognosis*

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Key Messages

QT interval prolongation is common in organophosphate poisoning and reflects disease severity. Patients with prolonged QT/QTc have worse clinical outcomes and higher need for intensive care support. Routine ECG assessment can aid early risk stratification and guide closer monitoring in these patients.

agricultural regions where these compounds are widely used as pesticides [1]. Intentional and accidental exposures contribute significantly to emergency department visits and hospital admissions, and the associated morbidity and mortality continue to be substantial despite advances in supportive care and antidotal therapy [2]. The clinical manifestations of organophosphate poisoning primarily result from inhibition of acetylcholinesterase, leading to accumulation of acetylcholine at synapses and neuromuscular junctions and producing a wide spectrum of muscarinic, nicotinic, and central nervous system effects [3].

INTRODUCTION

Organophosphate poisoning remains a major public health problem in many developing countries, particularly in

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In addition to the well-recognized respiratory and neurological complications, cardiovascular involvement plays an important role in determining patient outcomes. Various electrocardiographic abnormalities have been described in organophosphate poisoning, including sinus tachycardia, bradycardia, conduction disturbances, ST-T changes, and ventricular arrhythmias [4]. Among these, prolongation of the QT interval is of particular clinical importance because it reflects abnormalities in ventricular repolarization and is associated with an increased risk of malignant arrhythmias and sudden cardiac death [5].

The QT interval, when corrected for heart rate as QTc, serves as a readily available and non-invasive marker of electrical instability of the myocardium. Several factors in organophosphate poisoning, such as autonomic imbalance, electrolyte disturbances, hypoxia, and direct myocardial toxicity, may contribute to QT prolongation [6]. Prolonged QTc has been associated with worse outcomes in various acute medical conditions, but its prognostic significance in organophosphate poisoning requires further clarification, especially in routine clinical settings [7].

Early identification of patients at higher risk for complications is crucial for optimizing monitoring strategies and resource allocation in busy emergency and critical care units. Simple bedside tools that can aid in risk stratification are therefore of considerable clinical value. Electrocardiography is inexpensive, widely available, and easily repeatable, making it an attractive option for early prognostic assessment [8].

In this context, the present study was undertaken to evaluate the role of QT interval prolongation as a prognostic marker in patients with organophosphate poisoning. By comparing clinical course and outcomes between patients with normal and prolonged QT/QTc intervals, this study aims to assess whether early ECG findings can help predict disease severity and guide clinical management.

SUBJECTS AND METHODS

Study Design and Setting

This was a prospective follow-up study conducted over a period of three months in a tertiary care teaching hospital. The study was carried out in the emergency department and medical wards, where patients with acute organophosphate poisoning are routinely admitted and managed.

Study Population and Sample Size

Adult patients presenting with a history and clinical features suggestive of acute organophosphate poisoning were screened for inclusion. Patients with confirmed exposure to organophosphate compounds based on history, clinical examination, and supportive laboratory findings were enrolled in the study. All eligible patients admitted during the study period and fulfilling the inclusion criteria were included.

Inclusion Criteria

Adult patients aged 18 years and above.

Patients with confirmed or strongly suspected acute organophosphate poisoning.

Patients presenting within a clinically relevant time window after exposure.

Patients in whom a baseline electrocardiogram could be recorded on admission.

Exclusion Criteria

Patients with known pre-existing cardiac disease

Patients on drugs known to prolong the QT interval

Patients with mixed or unknown poisoning

Patients with significant electrolyte abnormalities or metabolic conditions known to affect the QT interval at presentation

Patients with a history of recent major surgery or significant comorbid illness influencing cardiac conduction

Clinical Assessment and Management

All patients underwent detailed clinical evaluation on admission, including assessment of vital signs and severity of poisoning. Standard treatment for organophosphate poisoning, including atropine and other supportive measures, was administered as per institutional protocol. The requirement for ventilatory support, duration of hospital stay, and in-hospital outcome were recorded for each patient.

Electrocardiographic Evaluation

A standard 12-lead electrocardiogram was recorded for all patients at the time of admission. The QT interval was measured manually, and the corrected QT interval (QTc) was calculated using Bazett's formula. QTc values greater than 450 ms in males and greater than 470 ms in females were considered prolonged. Based on QT/QTc values, patients were categorized into two groups: those with normal QT/QTc and those with prolonged QT/QTc. Patients with prolonged QT/QTc were monitored with serial electrocardiograms and continuous cardiac monitoring during their hospital stay.

Laboratory Investigations

Baseline laboratory investigations included serum cholinesterase levels, complete blood counts, renal and liver function tests, serum electrolytes, and arterial blood gas analysis where clinically indicated. These parameters were used to assess the severity of poisoning and to identify potential confounding factors affecting cardiac conduction.

Outcome Measures

The primary outcome measures were the need for ventilatory support and in-hospital outcome. Secondary outcome measures included duration of hospital stay and

association of QT/QTc prolongation with laboratory indicators of severity such as serum cholinesterase levels.

Statistical Analysis

Data were compiled and analyzed using appropriate statistical software. Continuous variables were expressed as mean and standard deviation, and categorical variables were expressed as frequencies and percentages. Comparisons between patients with normal and prolonged QT/QTc intervals were performed using suitable statistical tests. A p value of less than 0.05 was considered statistically significant.

RESULTS

A total of 80 patients with acute organophosphate poisoning were included in the study during the three-month study period. On admission electrocardiography, patients were categorized into two groups based on corrected QT interval values: those with normal QT/QTc and those with prolonged QT/QTc. QT/QTc prolongation was observed in a substantial proportion of patients, indicating frequent cardiac electrical involvement in organophosphate toxicity.

The study population consisted predominantly of young and middle-aged adults, with a slight male predominance. Common presenting features included excessive salivation, sweating, vomiting, respiratory distress, and altered sensorium. Serum cholinesterase levels were markedly reduced in most patients, with lower levels observed in those with prolonged QT/QTc intervals, suggesting greater severity of poisoning.

Patients with prolonged QT/QTc showed a higher requirement for ventilatory support and a longer duration of hospital stay compared to those with normal QT/QTc intervals. Adverse outcomes, including complications and mortality, were more frequent in the prolonged QT/QTc group. Overall, QT/QTc prolongation on admission ECG was associated with more severe clinical course and poorer outcomes.

Baseline demographic characteristics

Table 1: Demographic profile of patients with organophosphate poisoning (n = 80)

Table 1 shows the age and sex distribution of the study population.

Variable	Category	Number (n)	Percentage (%)
Age (years)	18–30	24	30.0
	31–40	22	27.5
	41–50	18	22.5
	>50	16	20.0
Sex	Male	46	57.5
	Female	34	42.5
Total		80	100.0

Distribution of QT/QTc interval status

Table 2: Distribution of patients based on QT/QTc interval on admission ECG

Table 2 describes the proportion of patients with normal and prolonged QT/QTc intervals.

QT/QTc status	Number (n)	Percentage (%)
Normal QT/QTc	38	47.5
Prolonged QT/QTc	42	52.5
Total	80	100.0

Clinical features at presentation

Table 3: Presenting clinical features among study participants

Table 3 summarizes the common presenting symptoms and signs.

Clinical feature	Number (n)	Percentage (%)
Excessive salivation	62	77.5
Vomiting	48	60.0
Sweating	44	55.0
Respiratory distress	36	45.0
Altered sensorium	28	35.0

Serum cholinesterase levels and QT/QTc status

Table 4: Serum cholinesterase levels in relation to QT/QTc interval status

Table 4 shows the distribution of serum cholinesterase levels in patients with normal and prolonged QT/QTc.

QT/QTc status	Mean serum cholinesterase (IU/L) $\pm$ SD
Normal QT/QTc	2850 $\pm$ 740
Prolonged QT/QTc	1980 $\pm$ 620

Requirement of ventilatory support

Table 5: Requirement of ventilatory support in relation to QT/QTc interval status

Table 5 compares the need for ventilatory support between the two groups.

QT/QTc status	Required ventilation n (%)	Not required n (%)	Total
Normal QT/QTc	8 (21.1)	30 (78.9)	38
Prolonged QT/QTc	24 (57.1)	18 (42.9)	42
Total	32	48	80

Duration of hospital stay

Table 6: Duration of hospital stay in patients with normal and prolonged QT/QTc

Table 6 shows the mean duration of hospital stay in the two groups.

QT/QTc status	Mean hospital stay (days) $\pm$ SD
Normal QT/QTc	5.2 $\pm$ 1.8
Prolonged QT/QTc	8.1 $\pm$ 2.6

Clinical outcome

Table 7: Clinical outcome in relation to QT/QTc interval status

Table 7 summarizes the outcomes in both groups.

Outcome	Normal QT/QTc n (%)	Prolonged QT/QTc n (%)
Recovered	35 (92.1)	34 (81.0)
Complications	2 (5.3)	5 (11.9)
Death	1 (2.6)	3 (7.1)
Total	38 (100.0)	42 (100.0)

Table 1 shows that among the 80 patients with organophosphate poisoning, the largest proportion belonged to the 18–30 years age group with 24 patients (30.0%), followed by the 31–40 years group with 22 patients (27.5%). Patients aged 41–50 years constituted 18 cases (22.5%), while those above 50 years accounted for 16 cases (20.0%). Males formed 46 cases (57.5%) and females 34 cases (42.5%), indicating a slight male predominance in the study population. **Table 2** demonstrates that QT/QTc prolongation was present in 42 patients (52.5%), while 38 patients (47.5%) had normal QT/QTc intervals on admission ECG. This indicates that more than half of the patients exhibited QT/QTc prolongation at presentation. **Table 3** shows that excessive salivation was the most common presenting feature, observed in 62 patients (77.5%), followed by vomiting in 48 patients (60.0%) and sweating in 44 patients (55.0%). Respiratory distress was present in 36 patients (45.0%), and altered sensorium was noted in 28 patients (35.0%), reflecting the typical cholinergic and systemic manifestations of organophosphate poisoning. **Table 4** indicates that the mean serum cholinesterase level in patients with normal QT/QTc was 2850 $\pm$ 740 IU/L, whereas patients with prolonged QT/QTc had a lower mean level of 1980 $\pm$ 620 IU/L. This suggests comparatively greater biochemical severity in patients with QT/QTc prolongation. **Table 5** shows that ventilatory

support was required in 24 out of 42 patients (57.1%) with prolonged QT/QTc, compared to 8 out of 38 patients (21.1%) with normal QT/QTc. Conversely, 30 patients (78.9%) in the normal QT/QTc group did not require ventilation, compared to 18 patients (42.9%) in the prolonged QT/QTc group, indicating a higher need for respiratory support in patients with QT/QTc prolongation. **Table 6** demonstrates that the mean duration of hospital stay was longer in patients with prolonged QT/QTc (8.1 $\pm$ 2.6 days) compared to those with normal QT/QTc (5.2 $\pm$ 1.8 days), showing increased hospitalization among patients with QT/QTc prolongation. **Table 7** shows that recovery was observed in 35 patients (92.1%) with normal QT/QTc and in 34 patients (81.0%) with prolonged QT/QTc. Complications occurred in 2 patients (5.3%) in the normal QT/QTc group and in 5 patients (11.9%) in the prolonged QT/QTc group. Mortality was recorded in 1 patient (2.6%) with normal QT/QTc and in 3 patients (7.1%) with prolonged QT/QTc, indicating poorer outcomes in the prolonged QT/QTc group.

DISCUSSION

Organophosphate poisoning continues to be a major cause of acute medical emergencies in many developing countries and is associated with significant morbidity and mortality. While respiratory failure and neurological complications are well-recognized contributors to poor outcomes, cardiovascular involvement plays a crucial role

in determining the clinical course [9]. Electrocardiographic abnormalities, particularly prolongation of the QT interval, reflect disturbances in myocardial repolarization and are known to predispose patients to serious arrhythmias and sudden cardiac events [10].

The findings of the present study indicate that QT/QTc prolongation is a common occurrence in patients with acute organophosphate poisoning and is associated with a more severe clinical course [11]. Patients with prolonged QT/QTc demonstrated greater physiological derangement, higher need for ventilatory support, longer hospitalization, and poorer overall outcomes compared to those with normal QT/QTc intervals. This supports the concept that QT/QTc prolongation is not merely an incidental ECG finding, but rather a marker of systemic toxicity and disease severity [12].

Several mechanisms may explain the occurrence of QT/QTc prolongation in organophosphate poisoning. Autonomic dysfunction resulting from excessive acetylcholine accumulation can lead to imbalance between sympathetic and parasympathetic activity, affecting cardiac conduction and repolarization [13]. In addition, hypoxia, electrolyte disturbances, and direct myocardial toxicity may further contribute to electrical instability of the myocardium. These factors together can create a substrate for repolarization abnormalities and increase the risk of malignant arrhythmias [14].

The association between lower serum cholinesterase levels and QT/QTc prolongation observed in this study further supports the link between biochemical severity of poisoning and cardiac electrical disturbances. Serum cholinesterase levels are widely used as a marker of exposure and severity in organophosphate toxicity, and their correlation with ECG abnormalities highlights the systemic nature of the toxic effects [15].

From a clinical perspective, early identification of patients at higher risk of complications is essential for optimizing monitoring strategies and resource utilization, especially in busy emergency and critical care settings [16]. Electrocardiography is inexpensive, widely available, and can be performed rapidly at the bedside. The presence of QT/QTc prolongation on admission ECG can therefore serve as a simple and practical tool for early risk stratification, prompting closer monitoring, early intensive care support, and more aggressive management when required [17].

Although arterial blood gas analysis, laboratory investigations, and clinical scoring systems provide valuable information, they may not always be immediately available or may require repeated sampling. In contrast, ECG can be repeated easily and non-invasively, allowing dynamic assessment of cardiac status during the course of illness. This makes QT/QTc monitoring particularly useful in settings with limited resources [18].

Despite its clinical relevance, QT/QTc prolongation should not be interpreted in isolation. Other factors such as pre-existing cardiac conditions, concurrent medications, and metabolic abnormalities can also influence QT duration [19]. Careful clinical correlation and comprehensive patient assessment remain essential. Nevertheless, the findings of this study reinforce the importance of incorporating routine ECG evaluation and QT/QTc assessment into the initial and ongoing evaluation of patients with organophosphate poisoning [20].

Overall, the study supports the role of QT/QTc prolongation as a valuable prognostic marker in organophosphate poisoning. Its use can aid early identification of high-risk patients, guide intensity of monitoring and treatment, and potentially contribute to improved clinical outcomes through timely intervention.

CONCLUSION

QT interval prolongation is a common electrocardiographic abnormality in patients with acute organophosphate poisoning and is associated with a more severe clinical course. Patients with prolonged QT/QTc intervals show higher requirements for ventilatory support, longer hospital stay, and poorer clinical outcomes compared to those with normal QT/QTc intervals. Routine assessment of QT/QTc on admission electrocardiography provides a simple, non-invasive, and cost-effective method for early risk stratification. Incorporation of QT/QTc monitoring into standard evaluation protocols for organophosphate poisoning may help identify high-risk patients early and guide closer monitoring and timely intensive care interventions.

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REFERENCES

1. Shadnia S, Okazi A, Akhlaghi N, Sasanian G, Abdollahi M. Prognostic value of long QT interval in acute and severe organophosphate poisoning. *J Med Toxicol.* 2009 Dec;5(4):196-9. doi: 10.1007/BF03178266. PMID: 19876851; PMCID: PMC3550412.
2. Akdur O, Durukan P, Ozkan S, Avsarogullari L, Vardar A, Kavalci C, Ikizceli I. Poisoning severity score, Glasgow coma scale, corrected QT interval in acute organophosphate poisoning. *Hum Exp Toxicol.* 2010 May;29(5):419-25. doi: 10.1177/0960327110364640. Epub 2010 Mar 4. PMID: 20203133.
3. Liu SH, Lin JL, Weng CH, Yang HY, Hsu CW, Chen KH, Huang WH, Yen TH. Heart rate-corrected QT interval helps predict mortality after intentional organophosphate poisoning. *PLoS One.* 2012;7(5):e36576. doi:

- 10.1371/journal.pone.0036576. Epub 2012 May 4. PMID: 22574184; PMCID: PMC3344908.
4. Pannu AK, Bhalla A, Vishnu RI, Garg S, Dhibar DP, Sharma N, Vijayvergiya R. Cardiac injury in organophosphate poisoning after acute ingestion. *Toxicol Res (Camb)*. 2021 Apr 26;10(3):446-452. doi: 10.1093/toxres/tfab036. PMID: 34141158; PMCID: PMC8201560.
5. Anand S, Singh S, Nahar Saikia U, Bhalla A, Paul Sharma Y, Singh D. Cardiac abnormalities in acute organophosphate poisoning. *Clin Toxicol (Phila)*. 2009 Mar;47(3):230-5. doi: 10.1080/15563650902724813. PMID: 19267290.
6. Baydin A, Aygun D, Yazici M, Karatas A, Deniz T, Yardan T. Is there a relationship between the blood cholinesterase and QTc interval in the patients with acute organophosphate poisoning? *Int J Clin Pract*. 2007 Jun;61(6):927-30. doi: 10.1111/j.1742-1241.2006.00931.x. PMID: 17504354.
7. Thandar SM, Naing KT, Sein MT. Serum High-sensitivity C-reactive Protein Level and Corrected QT Interval in Agricultural Workers in Myanmar Exposed to Chronic Occupational Organophosphate Pesticides. *J UOEH*. 2021;43(2):173-182. doi: 10.7888/juoeh.43.173. PMID: 34092762.
8. Grmec S, Mally S, Klemen P. Glasgow Coma Scale score and QTc interval in the prognosis of organophosphate poisoning. *Acad Emerg Med*. 2004 Sep;11(9):925-30. doi: 10.1197/j.aem.2004.03.018. PMID: 15347541.
9. Abraham S, Oz N, Sahar R, Kadar T. QTc prolongation and cardiac lesions following acute organophosphate poisoning in rats. *Proc West Pharmacol Soc*. 2001;44:185-6. PMID: 11793977.
10. Demirag K, Cankayali I, Eris O, Moral AR, Pehlivan M. The comparison of therapeutic effects of atropine and pralidoxime on cardiac signs in rats with experimental organophosphate poisoning. *Adv Ther*. 2005 Mar-Apr;22(2):79-86. doi: 10.1007/BF02849879. PMID: 16020398.
11. Yurumez Y, Yavuz Y, Saglam H, Durukan P, Ozkan S, Akdur O, Yucel M. Electrocardiographic findings of acute organophosphate poisoning. *J Emerg Med*. 2009 Jan;36(1):39-42. doi: 10.1016/j.jemermed.2007.08.063. Epub 2008 Mar 4. PMID: 18296005.
12. Lionte C, Sorodoc L, Petriş O, Sorodoc V. Modificări electrocardiografice în intoxicația acută cu pesticide organofosforice [Electrocardiographic changes in acute organophosphate poisoning]. *Rev Med Chir Soc Med Nat Iasi*. 2007 Oct-Dec;111(4):906-11. Romanian. PMID: 18389778.
13. Nel L, Hatherill M, Davies J, Andronikou S, Stirling J, Reynolds L, Argent A. Organophosphate poisoning complicated by a tachyarrhythmia and acute respiratory distress syndrome in a child. *J Paediatr Child Health*. 2002 Oct;38(5):530-2. doi: 10.1046/j.1440-1754.2002.00050.x. PMID: 12354276.
14. Chen YZ, Zhu M, Wang B. [A study about the clinical significance of cardiac QTc interval for predicting the mortality and prognostic of acute organophosphate poisoning]. *Zhonghua Wei Zhong Bing Ji Jiu Yi Xue*. 2013 Sep;25(9):558-60. Chinese. PMID: 24059427.
15. Gul EE, Can I, Kusumoto FM. Case report: an unusual heart rhythm associated with organophosphate poisoning. *Cardiovasc Toxicol*. 2012 Sep;12(3):263-5. doi: 10.1007/s12012-012-9165-z. PMID: 22528817.
16. Taira K, Aoyama Y, Kawamata M. Long QT and ST-T change associated with organophosphate exposure by aerial spray. *Environ Toxicol Pharmacol*. 2006 Jul;22(1):40-5. doi: 10.1016/j.etap.2005.11.008. Epub 2006 Jan 18. PMID: 21783684.
17. Chacko J, Elangovan A. Late onset, prolonged asystole following organophosphate poisoning: a case report. *J Med Toxicol*. 2010 Sep;6(3):311-4. doi: 10.1007/s13181-010-0095-5. PMID: 20532843; PMCID: PMC3550479.
18. Baydin A, Aygun D, Yazici M, Karatas A, Deniz T, Yardan T. Is there a relationship between the blood cholinesterase and QTc interval in the patients with acute organophosphate poisoning? *Int J Clin Pract*. 2007 Jun;61(6):927-30. doi: 10.1111/j.1742-1241.2006.00931.x. PMID: 17504354.
19. Wang MH, Tseng CD, Bair SY. Q-T interval prolongation and pleomorphic ventricular tachyarrhythmia ('Torsade de pointes') in organophosphate poisoning: report of a case. *Hum Exp Toxicol*. 1998 Oct;17(10):587-90. doi: 10.1177/096032719801701010. PMID: 9821023.
20. Finkelstein Y, Kushnir A, Raikhlin-Eisenkraft B, Taitelman U. Antidotal therapy of severe acute organophosphate poisoning: a multihospital study. *Neurotoxicol Teratol*. 1989 Nov-Dec;11(6):593-6. doi: 10.1016/0892-0362(89)90044-5. PMID: 2696878.