

QT prolongation due to organophosphate poisoning

AYC Siu, LCH Tsoi, WCB Lo, CCH Chung

Organophosphate poisoning is a life-threatening condition. The toxicity is due to the irreversible inactivation of cholinesterase. Apart from the enhanced muscarinic and nicotinic activities, cardiac toxicity is also possible. We present a case of organophosphate poisoning in which the electrocardiogram showed QT prolongation. The literature related to the topic is also reviewed. (*Hong Kong j.emerg.med.* 2000;7:234-235)

Keywords: Organophosphate poisoning, QT prolongation

Introduction

Organophosphate is a serious poisoning and the major effect is due to inactivation of cholinesterase. The toxic insecticide will overdrive the cholinergic receptors with manifestation of muscarinic and nicotinic effect, e.g. hypersecretion and muscle fasciculation. Cardiac toxicity can present with bradycardia and hypotension. But tachycardia and other tachyarrhythmias are also possible. Prolonged QT has also previously documented. Besides the clinical assessment, serum cholinesterase is considered the gold standard in the assessment of the severity and the prognosis of this poisoning. However, the result may not be available immediately in most of the emergency departments. The use of electrocardiographic features in the assessment may therefore play a role.

Case History

An 80-year-old lady with history of diabetes mellitus and hypertension was found collapsed at home. An

empty bottle of insecticide (Phosphothionate) was found nearby and she was suspected to have taken it. But the amount and the time of ingestion were not known. Her relative also recalled that she had expressed the wish to die recently. She was brought to our department for initial resuscitation. On arrival, she was unconscious. Her pupils were 2 mm, fixed on right side and deformed on left side due to previous eye surgery. There was no muscle fasciculation noted but the muscle tone was reduced in all limbs. The oral cavity was full of secretion.

The initial electrocardiogram showed sinus tachycardia with rate of 100/min. (Figure 1) QT interval was 0.48 sec (QTc=0.62 sec. Normal <0.42 sec). Continuous ECG monitoring did not detect further arrhythmia. Chest radiograph taken after intubation was normal.

On arrival, she was immediately intubated using rapid sequence induction and 2 gm of pralidoxime was given intravenously over 30 minutes. Atropine was not given initially as the duty officer was worried that degeneration into overwhelming arrhythmia may occur in the presence of QT prolongation. After initial stabilisation, she was transferred to the intensive care unit of a nearby hospital which provided critical care support for our hospital. However, she died 2 days after admission despite intensive care management. The serum cholinesterase was requested for, but the result was not available as the specimen was haemolysed.

Correspondence to:

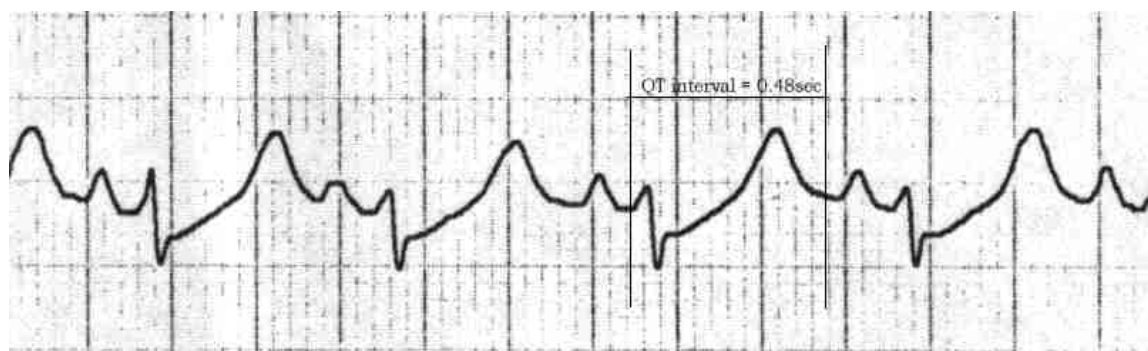
Siu Yuet Chung, Axel, MBChB(CUHK), FRCS(Edin)

North District Hospital, Accident and Emergency Department, 9 Po Kin Road, Fanling, N.T., Hong Kong
Email: axel@hknet.com

Tsoi Chun Hing, Ludwig, MBChB(CUHK), MRCP(UK)

Lo Chi Bui, William, MBBS(HK), MRCP(UK), FRCS(Edin)

Chung Chin Hung, Charles, MBBS(HK), FRCS(Glasg)



Bazett's formula: $QTc = QT / \text{square root of the RR interval}$
 $= 0.48 \text{ sec} / \sqrt{0.6}$
 $= 0.62 \text{ sec}$

Figure 1. The initial rhythm strip of lead II showed sinus tachycardia with prolonged QT interval.

Discussion

Organophosphate poisoning is always considered a life-threatening condition. The toxic insecticide binds to cholinesterase and irreversibly inhibits it. The clinical manifestation is mainly related to the enhanced muscarinic and nicotinic activities. The commonest presenting signs include constricted pupils, hypersalivation, abdominal pain, depressed conscious level and muscle fasciculation, etc.¹ Most of the fatality results from respiratory failure.²

The cardiac manifestation of organophosphate included hypotension and bradycardia. Tachycardia was not uncommon due to the stimulation of pre-ganglionic nicotinic receptors. Other dysrhythmia was also noticed. A retrospective study with 46 patients poisoned with organophosphate, 67% of them were found to have QT prolongation. ST segment changes were detected in 41% and prolonged PR interval in only 9%.³

Traditionally, serum cholinesterase has been used to assess the severity of intoxication. But the result is not usually available within minutes. Researchers have also started to question the accuracy of using the serum cholinesterase as the indicators.^{4,5} A major retrospective study conducted in Taiwan confirmed the relationship of QT prolongation with the severity of organophosphate in terms of the incidence of respiratory failure and mortality.⁶ The severity of poisoning indicated by the presence of

QT prolongation also shows correlation with the serum cholinesterase level. Our case also demonstrated the presence of QT prolongation and this in fact had predicted a grave prognosis. Unfortunately, the serum cholinesterase level was not available for comparison.

In the absence of immediate availability of serum cholinesterase level, electrocardiogram can serve as an important early prognostic indicator for assessment of intoxication in the emergency department.

References

1. Tafuri J, Roberts J. Organophosphate poisoning. *Ann Emerg Med* 1987;16(2):193-202.
2. Zwiener RJ, Ginsbrug CM. Organophosphate and carbamate poisoning in infants and children. *Pediatrics* 1988;81(5):121-6.
3. Saadeh AM, Farsakh NA, al-Ali MK. Cardiac manifestation of acute carbamate and organophosphate poisoning. *Heart* 1997;77(5):461-4.
4. Nouira S, Abroug F, Elatrous S, et al. Prognostic value of serum cholinesterase in organophosphate poisoning. *Chest* 1994;106(6):1811-4.
5. Bardin PG, Van eeden SF. Organophosphate poisoning: grading severity and comparing treatment between atropine and glycopyrrolate. *Crit Care Med* 1990;18(9):956-60.
6. Chuang FR, Fang SW, Lin JL, et al. QTc prolongation indicates a poor prognosis in patients with organophosphate poisoning. *Am J Emerg Med* 1996;14(5):451-3.