MATHEWS

Case Report

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Atrial Fibrillation: A Rare ECG Finding in Organophosphate Poisoning

Shivam Patel[1,*], Roshan Mathew[2], Reena Patel[3]

[1]*Assistant Professor, Department of Emergency Medicine, Pt. JNM Medical College, Raipur, India*

[2]*Senior Resident, Department of Emergency Medicine, AIIMS, New Delhi, India*

[3]*Department of transfusion Medicine, AIIMS Raipur, India*

ABSTRACT

Organophosphate (OP) poisoning is very common in developing countries. Organophosphate compounds irreversibly bind to cholinesterase receptors that lead to cholinergic excess symptoms through nicotinic, muscarinic and central nervous system receptors. OP compounds can lead to cardiac complications which include tachycardia, bradycardia, hypotension, arrhythmias, AV blocks, pulmonary edema and rarely myocardial infarction. We report a case of organophosphate poisoning associated with atrial fibrillation and case was managed with atropine, PAM, antiarrhythmic drugs and other conservative measures. Cardiac complications should be treated accordingly along with antidote and supportive measures in OP poisoning for good outcomes

Keywords: Atrial fibrillation, Emergency department, Organophosphate poisoning

INTRODUCTION

Cardiac complications are common in organophosphate (OP) poisoning including wide varieties of ECG changes. Cardiac complications that often accompany poisoning with these compounds may be serious and are often fatal. Since early recognization of abnormal rhythm in OP poisoning protects the patients against acquiring life-threatening arrhythmias [1]. We report a case here in which patient develops atrial fibrillation with fast ventricular rate following organophosphate poisoning.

CASE

We received a 42-year-old male at around 9.00 P.M. in our emergency department. With history of dyspnea, altered behaviour and frothy secretions from mouth with one episode of vomiting over past three hours. On primary survey his airway was patent. Saturation of 98% on room air, Respiratory rate of 28 per minute and bilateral coarse crepitations on auscultation. Pulse rate was around 140 per minute irregular and blood pressure was 150/90 mm Hg. There was sweating all over the body. We

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***Corresponding Author**

Shivam Patel

Assistant Professor, Department of Emergency Medicine, Pt. JNM Medical College, India.

Phone: +91 9810697593.

E-mail: patelshivam999@gmail.com

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felt a peculiar garlic odour nearby patient. So thinking of OP poisoning, On further probing history patient reveals that he had taken around 250 ml of chlorpyrifos.

ECG revealed atrial fibrillation with fast ventricular rate (Figure 1). Emergency blood investigations and ABG were within normal limits. Random blood sugar was 154 mg/dl. Urine tox, screen was negative. Bedside point of care ultrasound was done which shows bilateral B-profile in all lung zones with collapsible IVC, 2D-Echo was normal with no valvular abnormality and normal ventricular functions.

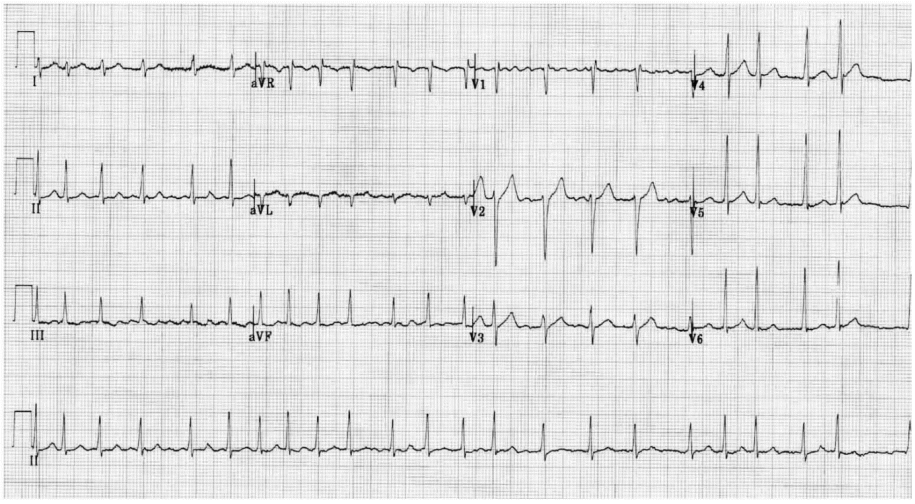

Figure 1. Atrial fibrillation with rate approx.140 on arrival of patient.

We asked patient and his relative that either patient has past history of any heart disease, HTN or any other comorbidities but they denied for any previous illness and there was no past history of palpitations. Patient was not on any medications.

So we take two large bore I.V. access and started intravenous fluids(Ringer lactate) at the rate of 150 ml per hour. RT lavage was done, we started bolus doses of atropine till atropinization. 2 gram PAM was given. And for atrial fibrillation with fast ventricular rate we give 5mg. of metoprolol I.V. bolus. After atropinization, we have started infusion of atropine but the irregular rhythm continues. So, we give 150 mg of amiodarone bolus over 15 minutes then infusion at the rate of 60 mg/kg has been started for next 6 hours and after that 30mg/kg for next 18 hours. After 16 hours of amiodarone and atropine infusion rhythm reverted to normal sinus rhythm(Figure 2). Patient was admitted in the ward for further monitoring and observation. After 6 days patient got discharged.

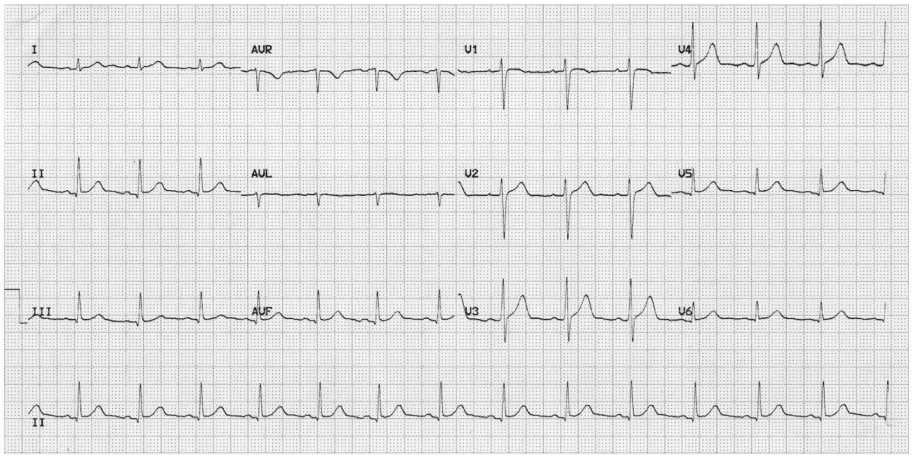

Figure 2. Normal sinus rhythm after antiarrhythmic treatment for atrial fibrillation.

DISCUSSION AND CONCLUSION

OP compounds inhibit acetyl cholinesterase, inhibition of the enzyme results in accumulation of acetylcholine which excessively stimulates nicotinic and muscarinic receptors causing widespread clinical symptoms like nausea, vomiting, diarrhea, urinary incontinence, blurring of vision, salivation, lacrimation, broncorrhoea, bradycardia, hypotension, muscle paralysis, fasciculation, confusion, seizures, coma and respiratory failure [2]. Wide variations of cardiac complications can occur from asymptomatic ECG abnormalities such as sinus tachycardia, sinus bradycardia, prolonged Q-Tc interval, AV blocks and ST-T changes to life-threatening complications such as hypotension, hypertension, cardiac arrhythmias, myocardial ischemia, and noncardiogenic pulmonary edema [3,4]. Paul and Bhattacharyya [5] described ECG abnormalities in organophosphorus poisoning and concluded that most common ECG abnormality was prolonged Q-Tc interval, found in 67 patients (62.6%) and followed by sinus tachycardia in 36 patients (33.6%) and sinus bradycardia in 33 patients (30.8%). ST segment elevation was found in 27 patients (25.2%) and inverted T-waves in 21 patients (19.6%). 9 cases(8.4%) were associated with First-degree heart block (P-R interval >0.20 s). Ventricular tachycardia occurred in 6 cases (5.6%) and ventricular premature complexes were noted in 3 patients (2.8%). Atrial fibrillation was seen in 5 patients (4.6%). Management includes supportive care: Decontamination where patients are cleaned with soap and water after removal of contaminated clothes. Irrigation of eyes with normal saline. Gastric lavage frequently for gastric decontamination. Respiratory support (through oxygen and mechanical ventilation in cases of severe intoxication causing bronchorrhea-induced bronchospasm or respiratory muscle paralysis), intravenous fluids, and maintaining electrolyte balance [6]. The mainstay of treatment is proper dose of atropine as antidote and treatment of other complications simultaneously.

Ludomirsky et al. described the mechanism in three phases. First phase is a brief period of sympathetic overactivity which causes tachycardia and hypertension. These are considered nicotinic effects and may be due to excessive release of catecholamines. In practice, tachycardia is usually observed as a result of fear and anxiety. In the second phase, parasympathetic overactivity predominates for a more prolong period which is characterized by cholinergic excess and causes bradycardia, hypotension along with ST-T changes, and life-threatening arrhythmias. The last and third phase is longer, usually associated with a prolonged QT interval and polymorphic ventricular tachycardia (torsades de pointes) that can result in sudden death. Hence, daily ECG recording should be warranted in every patient with OP poisoning so as to reduce cardiac mortality [7].

It is important to early recognize the cardiac complications of organophosphate poisoning. Atrial fibrillation although a rare cardiac complication in organophosphate poisoning can be associated with poor outcomes. Hence simultaneous treatment with anti-arrhythmias and antidote has been associated with good outcomes.

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