Case Report

T-wave Electrocardiographic Changes in Organophosphorous Compound Poisoning

Savitha Vijayakumar*#, M. Mahesh*, Sanoop Kovoorayyathu Sahadevan*, S.Mamatha**

ABSTRACT

In India, primarily an agrarian economy, various pesticides are easily available. Organophosphorous pesticide poisoning resulting from suicidal consumption is one of the major public health issues. Various electrocardiographic changes ranging from sinus tachycardia to ST elevation and T wave inversions have been reported in literature. In many cases ECG changes are usually nonspecific and unpredictable.We hereby present a case of acute organophosporous poisoning where the T wave ECG changes closely paralleled the clinical severity of intermediate syndrome.

Keywords: cholinomimetics; electrocardiogram; toxic; myocarditis; pesticide; poisoning

INTRODUCTION

Organophosporous (OP) insecticides are used extensively in horticulture and agriculture in India. Due to easy availability, poisoning with these agents has become very common. Cardiac complications such as pulmonary edema often accompany poisoning with these compounds, which may be serious and often fatal.[1] Various electrocardiographic changes ranging from sinus tachycardia to ST elevation and T wave inversions have been reported in literature. In many cases the ECG changes are usually nonspecific and unpredictable and do not correlate with the clinical severity of OP compound poisoning. We hereby present a case of acute OP poisoning wherein the T wave ECG changes closely paralleled the clinical severity of intermediate syndrome in OP compound poisoning.

CASE REPORT

A 35 year old male presented to the Emergency Department with history of consumption of OP compound allegedly with suicidal intention. He had consumed about 200 ml of Dimethoate along about five hours prior to arrival. On examination, pulse rate was 70 beats pm and rhythm was regular. B.P - 110/70mm/Hg, Respiratory Rate was -19/min, SpO2-95% at room air. Auscultation of chest revealed a few bilateral crepitations. The heart sounds were normal. Central Nervous System examination was normal except for pinpoint pupils.

The Patient was admitted to ICU, was treated as per hospital protocol with atropine, pralidoxime, and antibiotics. Supportive care was instituted. Investigations revealed Hb-12.6Gm/dl, TC-14, 620/mm3, there was neutrophilic leukocytosis. The plasma pseudocholinesterase was reduced at 1891 IU/L (Normal 4000 -11000 IU/L),Chest X Ray was normal.

The initial ECG was within normal limits **(Fig1)**. On the second day, the patient's condition worsened with breathlessness and neck muscle weakness.He was intubated and connected to ventilator. At around the same time, T-wave inversions were noted on the cardiac monitor. The 12 lead ECG done at this time showed T wave inversions in lead II, III and aVF. **(Fig 2)** The cardiac enzymes CKMB , Trop T were normal. Bedside echocardiography did not reveal any Regional Wall Motion Abnormality .The T wave ECG changes persisted **(Fig 3)** till extubation on day 10. On the eleventh day the ECG was again within normal limits with reversion of inverted T-waves back to normal upright pattern **(Fig 4)**

#(Author for correspondence): E Mail: dr.savitha2014@gmail.com
*Department of Medicine, JSS Medical College and Hospital, JSS University, Mysore. Karnataka State. India
**Department of OBG, JSS Medical College and Hospital, JSS University, Mysore. Karnataka State. India

Fig 1: ECG on admission. Within Normal Limits

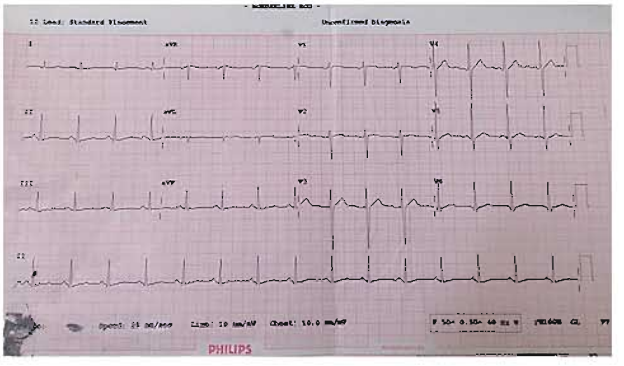


Fig 2: ECG on day two at the time of onset of intermediate syndrome. T wave inversions in lead II, III and aVF

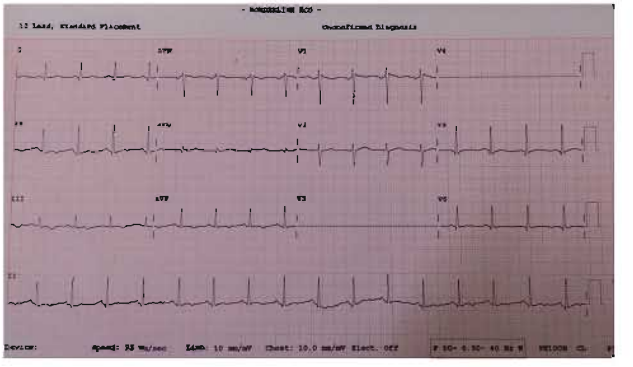


Fig 3: ECG on day ten. T-wave inversions persisting

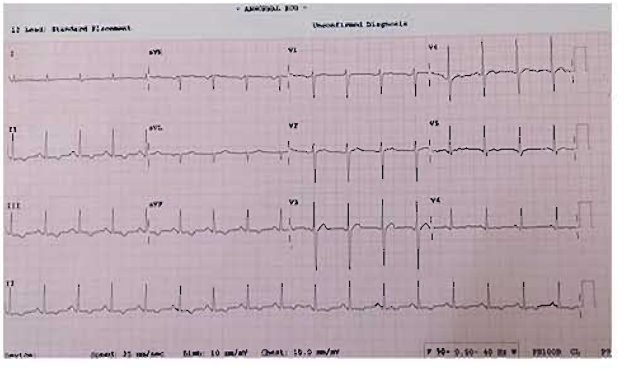
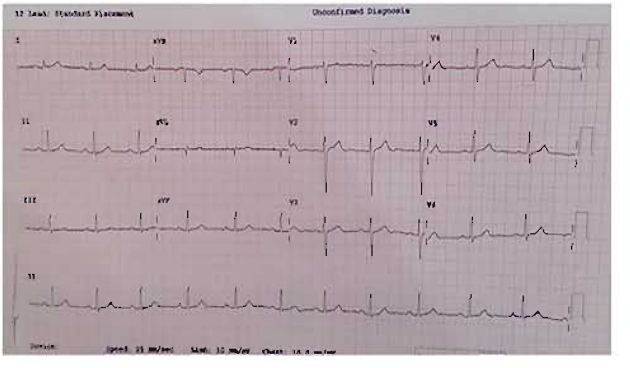


Fig 4: ECG after extubation. T-waves in II III aVF are again upright



Thereafter the patient was shifted to general ward and he was discharged after two days in a healthy condition

DISCUSSION

In India and other predominantly agricultural countries, pesticides are easily available, and organophosphorus poisoning is a major health issue. Organophosphorus poisoning presents with respiratory and gastrointestinal symptoms such as dyspnea, wheeze, cough, diarrhea, vomiting, and abdominal pain. (SLUDGE Symptoms-Salivation, Lacrimation, Urinary incontinence, Diarrhea and Emesis) Nervous system involvement leads to restlessness, tremor, ataxia, convulsions and later coma. The well known intermediate syndrome occurs in some patients due to persistent cholinesterase inhibition leading to muscle weakness and paralysis through hyperstimulation of muscarinic and nicotinic acetylcholine receptors.

Cardiac manifestations are range from arrhythmias to myocarditis and very rarely infarction.Early in the course, tachycardia is present, due to acetylcholine stimulation of nicotinic receptors, followed by bradycardia, secondary to muscarinic receptor stimulation. In severe poisonings, advanced AV block, bradyarrythmias and asystole may occur[1,2]

QTc interval prolongation followed by ventricular tachyarrhythmias , including Torsades de pointes can also occur. A rare feature is acute myocardial infarction due to coronary spasm induced by parasympathetic hyperactivity and direct toxic effect of pesticide on the myocardium.[3]

The various electrocardiographic abnormalities may thus range from sinus tachycardia to ST elevation. However the most common abnormalities are sinus bradycardia, prolonged QT interval and ST

elevation.[4-6] The ECG changes are unpredictable and often change over the time course of the poisoning.

The mechanism by which organophosphorus compounds and carbamates induce cardiotoxicity is still unknown. Ludomirsky described three phases of cardiac toxicity after organophosphorus poisoning, with the first phase being a brief period of increased sympathetic activity, the second being a prolonged period of parasympathetic activity, and the third involving Q-T prolongation followed by torsade de pointes ventricular tachycardia and then ventricular fibrillation.[7] There have been reports of sudden death occurring many days after clinical stabilization, presumably due to ventricular fibrillation.[7,8] The predisposing factors for the development of these complications are sympathetic and parasympathetic hyperstimulation, acidosis, hypoxemia, electrolyte abnormalities, and the direct toxic effect of the OP compound.[9]

In our patient significant T wave inversions were noted in inferior leads. There was no history of prior cardiac disease or risk factors for Ischemic Heart Disease such as hypertension or diabetes, alcohol abuse and tobacco usage. The cardiac enzymes were normal and a follow up Tread Mill Test was normal. We surmise that the T-wave changes were due to the effect of the toxin itself.

The notable feature in this case was that the ECG changes closely paralleled the clinical severity of intermediate syndrome in OP compound poisoning.

CONCLUSION

Physicians should be familiar with the various cardiac complications and ECG features of organophosphorus poisoning and a careful monitoring of ECG changes should be practiced.

ACKNOWLEDGEMENTS

We thank Dr BS Jayaraj Pulmonologist, Dr Ashok P, Assistant Professor of Medicine and Dr Ranganath Konganur ,Senior Resident in Medicine for their help in management of the patient.

CONFLICTS OF INTEREST

Declared none

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