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THE STUDY OF ELECTROCARDIOGRAPHIC FINDINGS IN PATIENTS WITH ORGANOPHOSPHATE POISONING AT A TERTIARY CARE HOSPITAL- A RETROSPECTIVE STUDY.

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ABSTRACT

INTRODUCTION: Cardiac manifestations that occur in a majority of patients with organophosphate (OP) poisoning may range from sinus tachycardia, to life-threatening complications including cardiogenic pulmonary edema and myocardial necrosis. In this study, we evaluated the various electrocardiographic manifestations in patients with OP poisoning.

METHODS: This retrospective-descriptive study was performed by reviewing the medical records from 350 patients poisoned with organophosphate admitted to GMC Jammu from January 2018 to October 2018. Patients with incomplete records were excluded from the study and remaining 150 patients were included. Histories of all patients were collected and ECG analysis was conducted including the rate, rhythm, ST-T abnormalities, conduction defects, and measurement of PR and QT intervals. Descriptive statistical analysis was conducted by SPSS software version18.

RESULTS: Of the total 150 patients (100 were male and 50 females) with OP poisoning that referred to the GMC Jammu, 63 patients presented ECG abnormalities. The mean age of the patients was 35.78 ± 12.91 years. Sinus tachycardia (38.1%) was the most common ECG abnormality, followed by non-specific ST-T changes (25.4%). Overall, mortality rate was 5% in 150 patients and all of the deceased patients presented with changes in ECG.

CONCLUSION: OP poisoning is associated with significant ECG abnormalities, especially tachycardia and non-specific ST-T changes.

INTRODUCTION

Acute organophosphate poisoning represents a major health problem in developing countries where indicates the need for a clinically based prognostic system organophosphate compounds are widely and easily available. Organophosphate insecticides inhibit both cholinesterase and pseudocholinesterase enzymatic activity and leads to cholinergic signs and symptoms [1].

The ease of availability of the organophosphate insecticides has resulted in a gradual increase in occupational, accidental, and suicidal poisoning mainly in developing countries (2). Accidental poisoning can occur after exposure through skin or inhalation and serious poisoning often follows suicidal ingestion (3). According to the World Health Organization (WHO), one million serious accidental and two million suicidal poisonings with organophosphate occur worldwide every year, and of these, approximately 200,000 died, mostly in developing countries (4).

OP compounds are phosphoric or phosphonic acid derivatives that act as irreversible cholinesterase inhibitors, causing a syndrome of cholinergic excess involving muscarinic, nicotinic, and central nervous system receptors (5, 6). The most common presenting signs of OP poisoning include constricted pupils, hypersalivation, abdominal pain, depressed level of consciousness, muscle fasciculation, etc. The most fatality results from respiratory failure (7).

MATERIALS AND METHODS

This retrospective-descriptive study was performed by reviewing the medical records of all patients with organophosphate poisoning admitted to GMC Jammu from January 2018 to October 2018.

INCLUSION

All patients of both genders with age more than 18 yrs with history of OP poisoning or evidence of exposure to OP compounds within the previous 24 hours with characteristic manifestations and improvement of the signs and symptoms of OP poisoning after administration of atropine were enrolled in this study.

EXCLUSION CRITERIA-

Patients with history of Atrioventricular arrhythmias, congestive cardiac failure, two or three degree heart blocks, ischemic heart diseases, or preexisting motor/sensory neuropathy were excluded from the study.

Overall, 350 patients with OP poisoning were admitted over a period of ten months. Eventually, 200 cases were excluded due to a past history significant for cardiac disease and lack of the registration of medical records. Age, sex, cause of poisoning, time elapsed between exposure and admission to the hospital, treatment duration, need for assisted ventilation, and outcome in terms of morbidity and mortality were recorded from the registration medical records.

During their hospital stay, electrocardiography (ECG) was carried out once daily on all patients in the general medical ward. ECG analysis was conducted for rate rhythm, ST/T abnormalities, conduction defects and measurement of P-R interval, R-R interval, Q-T interval, and QTc interval. QT interval was corrected according to the formula applied by Bazett . QTc was considered prolonged when it was longer than 0.41 second in men and longer than 0.42 second in women and QRS duration was measured. All this information was collected for each patient and then descriptive statistical analysis was conducted by SPSS version 18. Values were presented as frequency and mean ± standard deviation.

RESULTS

In our study out of 150 patients, 100(66.67%) were males, 50(33.33%) were females. Male: female ratio is 2:1. (Table 1).

Table 1 above shows; Sex Distribution

SEX	NUMBER (N)	PERCENTAGE (%)
MALE	100	66.67
FEMALE	50	33.33
TOTAL	150	100

In our study, the incidence of organophosphorus poisoning was more among the age group 41-50 years (26.7 %) (Table 2).

Table 2 shows; Age Distribution

AGE (YRS)	NUMBER (N)	PERCENTAGE (%)
18 -30	28	18.7
31-40	36	24
41-50	40	26.7
51-60	23	15.3
61-70	21	14
More than 80	2	1.3
TOTAL	150	100

ECG changes seen in our study are shown in (Table 3).

ECG CHANGES	NUMBER(N)	PERCENTAGE (%)
1.BRADYCARDIA	16	25.4
2.TACHYCARDIA	24	38.1
3.ST-T WAVE CHANGES	16	25.4
4.PROLONG QT INTERVAL	3	4.8
5.VENTRICULAR TACHYCARDIA	1	1.5
6.MORE THAN ONE FINDING	3	4.8
TOTAL	63	100

DISCUSSION

Organophosphate poisoning is always considered a life-threatening condition. The mechanism by which organophosphates induce cardiotoxicity is still uncertain. Cardiac toxicity after OP compounds poisoning may occur in three phases. Initially, there is a brief period of increased sympathetic activity characterized by tachycardia and hypertension. This is followed by a more prolonged period of extreme cholinergic activity during which bradycardia and hypotension along with ST-T changes and life-threatening rhythm disturbances can occur. A third longer phase is usually associated with a prolonged QT interval and polymorphic ventricular tachycardia that can result in sudden death (8, 9, 10).

In our study of 150 patients, the most common ECG finding was sinus tachycardia which is seen in 24(38.1%) patients. Similarly P Karki et al studied cardiac manifestations of OP manifestations and stated that cardiac complications usually occur during the first few hours after exposure and Sinus tachycardia was seen in 15 patients (40%) out of 37 patients. They explained that sinus tachycardia is due to nicotinic receptor stimulation on the heart and increased sympathetic tone [11, 12].

Sinus bradycardia is also seen. In our study of 150 patients with OP poisoning 16 (25.4%) patients developed sinus bradycardia. In a similar study done by Agarwal S et al. out of 121 patients of Organophosphorus poisoning Sinus Bradycardia is seen in 8 patients (6.6%). The mechanism being behind sinus bradycardia explained by Agarwal S et al. is that it's caused due to muscarinic receptor stimulation on the heart and also due to parasympathetic over activity [13]. The second most common ECG changes seen our study are ST-T changes seen in 16(25.4%) patients. Out of 150 patients among both males and females of OP poisoning the common ST-T changes noticed were T inversions (leads II, III,aVF) (III,V1) (V1-V3) , Tall T (V2-V3), ST elevation (I,aVL), ST depression. In a similar study done by Agarwal. S et al. out of 121 patients 9 (7.4 %) patients developed ST depression, 9 (7.4 %) patients developed T inversions in leads II, III, and aVF(13). In a study done by Morteza Rahbar Taromsari et al studied 100 patients in which 24 patients showed ST elevation [14].

In another study done by Gouda HS et al. showed out of 50 patients ,elevated ST segment is seen in 2 (4%),inverted T waves in 13 (26%), and conduction defects in 1 (2%) [15]

The mechanisms behind ST-T changes explained by both Agarwal .S et.al and Gouda HS et al are T inversions may be due to ischemia in sub endocardial myocardial tissue and Tall T may be due to prolonged period of parasympathetic activity, electrolyte abnormalities such as hyperkalaemia, hypokalaemia, hypomagnesemia and hypocalcaemia. ST elevation may be due to multiple mechanisms such as transient or transmural myocardial infarction, coronary vasospasm, hypoxemia, due to raised cardiac enzymes due to injury to myocardial tissue by organophosphorus poisoning [13,15].

Other ecg changes observed in our study is QT prolongation. Out of 150 patients 3 (4.8%) patients developed QT prolongation. In a similar study done by P Karki et al. out of 37 patients with OP poisoning described prolonged QTc interval in 14 (37.8%) patients[3]. QTc interval value range in between (>0.43-0.46 seconds) using Bazett's formula [13]. In a similar study done by P. Ravikumar et al. out of 100 patients QT prolongation was observed among 28 patients [16].

According to P Karki et al. P. Ravikumar et al. H.S Gouda et al., Lausdari .S et al. multiple mechanisms play role in QT prolongation in op poisoning.Malfunction of ion channels which leads to an intracellular excess of positively charged ions extends ventricular repolarisation and results QT interval prolongation.

Large QT dispersion (longest-shortest QT interval on any of the 12 lead ecg) is due to ischemic changes which may conceal the QT prolongation in affecting vascular area.Predisposing factors for QT prolongation and development of Torsade de pointes requires special care even in mild op poisoned patients of older age group, females, and patients with low LVEF, LVH, ischemia and dyselectrolytemia (hypomagnesemia and hypokalemia). QT prolongation also shows correlation with serum cholinesterase levels[17].QTc prolongation may also be due to unequal sympathetic stimulation of myocardial cells, interaction with potassium channels and sodium and calcium exchanger in myocardial cell membrane are probably responsible for occasional prolonged QTc interval .

CONCLUSION

Poisoning with OP compounds can produce significant ECG abnormalities, especially tachycardia and non-specific ST-T changes. Since these abnormalities can cause lethal arrhythmia and cardiac damage, careful observation of the electrocardiogram of the patients exposed to OP compounds is necessary, parallel to the appropriate medical treatment

CONFLICT OF INTEREST -NIL

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