Short Communication

Cardiac Vitals: Not to be Missed in the Intensive Management of Organophosphorus Compound Poisoning

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ABSTRACT

Background - The importance of cardiac complications in organophosphorus compound (OPC) poisoning are not adequately appreciated by treating physicians. Most of such complications occur during the initial hours following exposure. Once the condition is recognized, immediate transfer to intensive care unit must be undertaken where appropriate resuscitative and monitoring facilities are available.

Aims and Objectives - To study the clinical profile of OPC poisoning with particular reference to the cardiovascular system.

Materials and Methods - This is a prospective study in which 22 patients presenting to the emergency department with deliberate or accidental ingestion of OPC were included as subjects. Data regarding clinical and biochemical profiles were recorded as per pre-structured proforma. In addition to routine investigations, CPK-MB levels and electrocardiogram were recorded in all these patients.

Results – Of the 22 cases, males (15, i.e., 68.18%) outnumbered females (7, i.e., 31.82%). Majority were in the age group 15–34 years (15, i.e., 68.18%). Miosis was present in19 cases (86.36%), frothing from the mouth in 17 (77.27%), and diarrhoea in 13 (59.09%). Leucocytosis was observed in 9 (40.91%), deranged renal profile in 2 (9%), elevated liver enzymes in 3 (14%), and raised CPK-MB levels in 7 (31.82%). ECG changes were present in 20 cases (90.90%). Mortality was 3 (13.64%).

Conclusion – OP poisoning is more common in males, and that too in the most productive age group. In the absence of reliable history, diagnosis can be made by the typical clinical profile associated with OP poisoning (cholinergic syndrome). OP poisoning can involve kidneys, heart, and liver in addition to its well known effects on the lungs and the central nervous system. Recognizing early cardiac abnormalities may avert death by timely admission to intensive care unit with resuscitative and monitoring facilities.

Key Words: Organophosphorus compound; Pesticide; Cholinergic syndrome; Creatine phosphokinase-MB; CPK-MB

Introduction

The global problem of pesticide poisoning is a widespread scourge that affects most countries, and is especially rampant in India.[1] Deliberate self harm is a major public health problem and a cause of premature mortality worldwide. A vast number of pesticides, particularly insecticides are being consumed with suicidal intent. Pesticides are the commonest poisons consumed in the tropics causing high mortality.[2] Following accidental or suicidal exposure, these anticholinesterases lead to three well defined neurological syndromes: initial life threatening acute cholinergic crisis, intermediate syndrome in which cranial nerve palsies, proximal muscle weakness and respiratory muscle weakness occur, for which ventilator support may be needed, and lastly, delayed organophosphate-induced polyneuropathy.[3]

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Biochemical changes are related to the severity of the organ damage and can thus help in the early diagnosis of complications. Aside from the classical cholinergic syndrome (with muscarinic and nicotinic symptoms), an array of cardiac abnormalities are also often seen which are serious and may often be fatal.[4] A delay in noticing these cardiac changes may lead to serious deleterious effects and death. Cardiac complications are potentially preventable if they are recognised early and treated promptly. The extent, frequency, and pathogenesis of the cardiac toxicity from these compounds have not been clearly defined. The mortality rate can however be considerably reduced following intensive management.[3]

Materials and Methods

This prospective study comprising 22 patients was carried out in GR Medical College, Gwalior (Madhya Pradesh) during 2009–2010. Subjects presenting in the emergency department with a history of organophosphorus compound (OPC) ingestion and characteristic signs and symptoms of cholinergic excess were included in the study. Those with a history of ingestion of OPC but having no clinical or biochemical evidence of poisoning were excluded. All the selected patients were subjected to a thorough clinical examination. Blood was drawn for biochemistry, including creatinine phosphokinase-MB (CPK-MB) levels. An electrocardiogram was recorded in all these patients. Management was mainly supportive with atropine and oximes as antidotes. Data collected were analyzed for demographic profile, clinical profile, biochemical profile, and the final outcome.

Results

A total of 22 patients were enrolled as subjects. The intention of consuming the OP compound was suicidal in all cases. Males (15, i.e., 68.18%) outnumbered females (7, i.e., 31.82%). Majority were in the age group of 15–34 years (15, i.e., 68.18%). Miosis was present in19 (86.36%), frothing from the mouth in 17 (77.27%), and diarrhoea in 13 cases (59.09%). Leucocytosis was observed in 9 cases (40.91%), while deranged renal profile was seen in 2 (9%), elevated liver enzymes in 3 (14%), and raised CPK-MB levels were noted in 7 (31.82%). ECG changes were present in 20 cases (90.90%). Mortality was 3 (13.64%).

Discussion

Miosis was present in 19 cases (86.36%), and frothing from the mouth in 17 (77.27%). These two clinical features should at once prompt a clinician to suspect a case of organophosphorus compound (OPC) poisoning in the absence of history. Deranged renal profile in 2 cases (9%) and elevated liver enzymes in 3 (14%) point towards the severity of poisoning, which should decide the line of treatment (supportive measures), duration of stay, and is important for follow-up after discharge.

Organophosphorus pesticide self-poisoning is an important clinical problem in developing countries. Unfortunately there are no universally accepted guidelines regarding the use of atropine, oximes and diazepam in the management of OPC poisoning even today. Since respiratory failure is the major cause for mortality, careful monitoring, appropriate management and early recognition of respiratory distress may decrease the mortality rate among these patients.[5] Equally important, though often neglected are the cardiac complications. These complications are often encountered in OPC poisoning, particularly during the first few hours. Hypoxaemia, acidosis, and electrolyte abnormalities are the major predisposing factors. Potentially lethal cardiac complications are common in patients with acute OPC poisoning and should not be missed.[7] The mechanism by which OPCs induce cardiotoxicity is still uncertain.[8] Intensive supportive treatment in intensive care facilitiy with administration of atropine in adequate doses early in the course of the illness will reduce the incidence of mortality.

Possible mechanisms of OPC-induced cardiac complications include sympathetic and parasympathetic overactivity, hypoxaemia, acidosis, electrolyte derangement, and a direct toxic effect on the cardiovascular system. In addition to acute cardiac manifestations, delayed complications (cardiac asystole) several days after exposure have been reported.[9]

In our study, elevated CPK-MB levels were present in 7 cases (31.82%). ECG abnormalities were noticed in 20 cases (90.9%). Various observed abnormalities included bradycardia, tachycardia, T wave inversion, QTc prolongation and ST segment elevation. Myocardial involvement as a result of direct cardiac toxicity could be one of the factors responsible for elevation of cardiac enzymes and electrocardiographic changes. Commonest ECG abnormality was QTc prolongation (8 cases, i.e., 36.36%).

ECG recording and CPK-MB levels are very important tools to detect early cardiac abnormalities in the absence of symptoms. The incidence of frequent cardiac complications and even delayed complications emphasizes the

need for continuous cardiac monitoring and frequent follow-up after discharge.[9,10] Drug-induced long-QT syndrome is characterized by a prolonged corrected QT interval (QTc) and increased risk of developing polymorphic ventricular tachycardia or tosades de pointes. Potassium levels should be maintained in the high normal range, and all QT prolonging agents must be promptly discontinued.[11,12]

Conclusion

Organophosphorus compound (OPC) poisoning can be readily recognized by the typical clinical features, particularly miosis and frothing from the mouth, in addition to other muscarinic and nicotinic symptoms characteristic of cholinergic excess. Respiratory and neurological syndromes are also well known. In addition to monitoring these aspects, cardiac monitoring is also important as it could contribute significantly to further reduction of mortality.

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