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Acute Organophosphorus Poisoning Complicated with Acute Coronary Syndrome (Nstemi) and right Atrial Thrombus: A case Report

KEYWORDS

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ABSTRACT *We are reporting a case of 28yr old male with alleged history of consumption OP poisoning (Chlorpyriphos 20%) who presented to ER with cholinergic symptoms of vomiting and constricted pupil, although his chest was clear with no signs of aspiration and hemodynamically stable and maintained consciousness. He was started with standard regime of treatment of OP Poisoning, but developed respiratory distress, hypoxemia and fresh ECG changes after 30hrs of admission to hospital suggestive of acute coronary syndrome/NSTEMI. Patient recovered with intensive care and supportive treatment. Organophosphorus poisoning is amongst the most commonly consumed poison due to its easy availability in Indian clinical setting. Complications may be acute or late. It mainly involves respiratory, cardiac and neurological. Cardiac complications are potentially lethal and understanding them and clinical suspicion in any susceptible individual can change the morbidity and mortality outcome. Cardiac complications can be due to treatment itself or due to compounds causing rhythms disorders to ischemic abnormalities. Hence, it is important to suspect cardiac ischemia as an early complication although rare, in case patient develops the symptoms for effective management.*

Introduction:
Organophosphorus (OP) pesticide poisoning is a major clinical and public health problem across the world including much of rural Asia[1]. It accounts for as much as 80% of pesticide-related hospital admissions[2]. Many studies estimate that OP pesticides are responsible for nearly two-third of self-harm deaths, a total of 200,000 a year[3]. Hospitals in rural areas should handle the impact of this problem with a case fatality of 15–30%[4]. The possible mechanisms of cardiac toxicity are related to sympathetic and parasympathetic over-activity, hypoxemia, acidosis, electrolyte derangements and a direct toxic effect of the compounds on myocardium[5]. On the other hand, the use of atropine as the antidote for OP poisoning itself may induce lethal arrhythmias[6].

The widespread use of organophosphates and carbamates as agricultural insecticides has increased the likelihood of poisoning with these compounds. Although poisoning can result from occupational exposure or accidental ingestion, in most cases there is suicidal intent, particularly in developing countries where the highest incidence of morbidity and mortality from this cause occurs.[7]

Case Report:
A 28yr male farmer presented with consumption of OP compound, Chlorpyriphos 20% about 20-30ml after 4hrs after ingestion with suicidal intent. He was conscious and oriented and provided the history of consumption. His main complaints were excessive salivation and uneasiness. He was hemodynamically stable on admission. Immediate gastric lavage was done at ER and was started with atropine and was admitted for further management. After 30hrs of admission patient went unconscious and developed hypoxemia with sPO_2 65%. Immediately patient was intubated and supportive ventilation was initiated. Initially ECG on admission was with in normal limit (Fig: 1& 2), but after the acute episode of unconsciousness and unexplained shock, the ECG showed ST segment changes as in (FIG: 3& 4). Immediate serum marker TROP-I quantitative estimation was done, which was 0.26µg/L (cut off: 0.01µg/L) suggestive of acute coronary event i.e. NSTEMI (ACS). Patient was started with Antianginals and LMWH, Tab. Aspirin+clopidogrel+atorvastatin (75mg/75mg/40mg) after initial loading dose by Ryles tube as he was unconscious and intubated. Patient had no prior cardiac illness or any risks for cardiac disease. All the necessary supportive measures were taken care along with Atropine infusion and PAM. Patient recovered on Day 7 after this event and a 2D-ECHO cardiography was done when patient was hemodynamically stable and extubated. The 2D- ECHO report was as follows:

Normal chamber dimension/concentric LVH, Trace TR, DDF I/IV, No RWMA, Thrombus of Size 18*2cm seen in RA, with EF: 55-60%.

Other routine investigations were within normal limit.
This report although suggested a cardiac thrombus, the primary cardiac event suggested ischemia (NSTEMI). For further evaluation the patient was advised to go for Cardiac MRI. Due to non-compliance of the patient, we could not proceed with this specified investigation. TROP-I was repeated after 12 days of primary event showed 0.02ng/mL (ref range: <0.1ng/mL), and CPK-MB level was 141.8 U/L (ref range: <24 U/L).

The patient was discharged completely stable with proper counselling and was put on antiplatelet drugs, and was advised follow-up after 15 days for repeat 2D-ECHO and if possible Cardiac MRI for any further changes in thrombus size or position.

Discussion:
The mechanism by which organophosphates and carba-

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mates cause cardiac manifestations is still an uncertainty. Various factors affect the cardiac outcome of any patient with OP poisoning. Although treatment with atropine by itself might cause cardiac complications, one might be vigilant to know what exactly the presentation of cardiac complication is. The mechanisms explained by Ludomirsky et.al[8] in 3 phases,

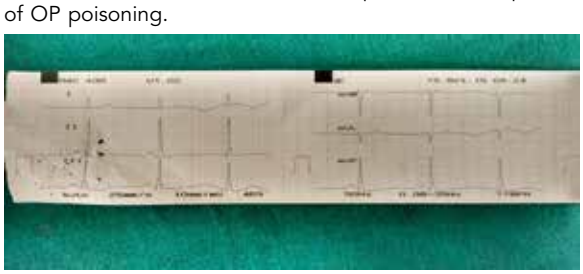
A brief period of increased sympathetic tone,
A prolonged period of parasympathetic activity,

Q-T prolongation is followed by Torsades de pointes ventricular tachycardia, and then ventricular fibrillation.

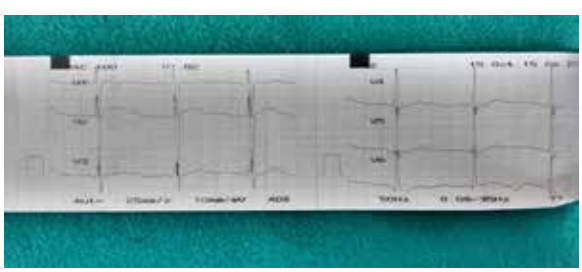
The possible mechanisms of cardiac toxicity are related to sympathetic and parasympathetic over-activity, hypoxemia, acidosis, electrolyte derangements and a direct toxic effect of the compounds on myocardium.[5] There is a role of increased parasympathetic and sympathetic over activity in myocardial damage. Although there is very less literature being reported on acute ischemic changes in such patients of poisoning, it's becoming more evident that one should always look for life threatening complications in a well improving patient. It has been postulated that parasympathetic over activity plays a major role in coronary artery spasm and coronary artery spasm has been induced with acetylcholine.

CONCLUSION:

We are reporting this case because, acute coronary syndrome in a patient with no history or risk factors for any cardiac illness, was only due to acute OP poisoning and not a co incidental event and was eventually found out to be a cardiac thrombus (RA Thrombus). Hence it is advocated not to overlook the cardiac complications in a patient of OP poisoning.



(FIG: 1)



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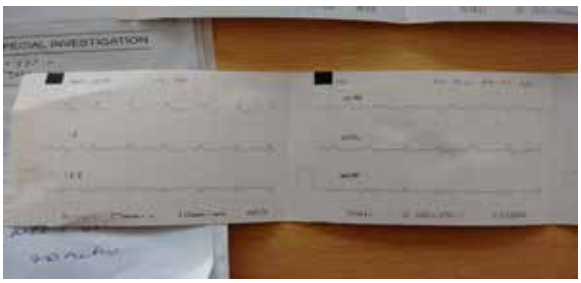

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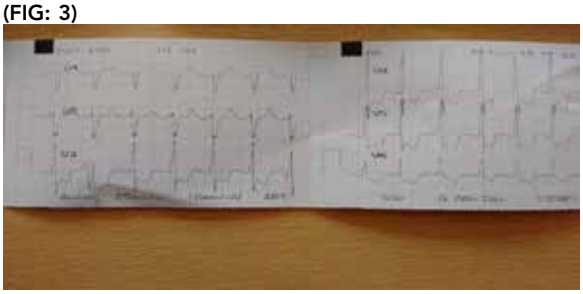

(FIG: 4)

REFERENCE

1. International Programme on Chemical Safety, World Health Organization (WHO). Epidemiology of pesticide poisoning: harmonized collection of data on human pesticide exposure in selected countries. Geneva, Switzerland: WHO Press; 2004. 2. Linden CH, Burns MJ, Mycyk MB. Poisoning, drug overdose and envenomation. In: Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, et al., editors. Harrison's Principles of Internal Medicine. 17th ed. New York: McGrawHill; 2008. p.2741-8. 3. Eddleston M. Patterns and problems of deliberate selfpoisoning in the developing world. QJM 2000;93:715-31. 4. World Health Organization (WHO), United Nations Environment Programme. Public Health Impacts of Pesticides Used in Agriculture. Geneva, Switzerland: WHO Press; 1990. 5. Taylor P. Anticholinesterase agents. In: Brunton LL, Lazo JS, Parker KL, editors. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York: McGraw-Hill; 2006. p.201-16. 6. Worek F, Kleine A, Falke K, Szinicz L. Arrhythmias in organophosphate poisoning: effect of atropine and bispyridinium oximes. Arch Int Pharmacodyn Ther 1995;329:418-35. 7. Jeyaratnam J. Acute pesticide poisoning: a major global health problem. World Health Stat Q 1990;43:139-44. 8. Ludomirsky A. Klein H. Sarelli P, Becker B. Hoffman S. Taitelman U, et.al. Q-T prolongation and polymorphous (torsade de pointes) ventricular arrhythmia associated with organophosphorus insecticide poisoning. Am J Cardiol 1982:49:1654-8.