Myocardial Infarction following Organophosphorus Compound Poisoning

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Abstract

We report a 22 year old male who was admitted to our hospital with alleged history of consumption of monocrotophos poison and had presented with chest pain. His electrocardiogram (ECG) had showed ST segment elevation myocardial infarction and troponins were elevated. He also had low cholinesterase levels and was treated with pralidoxime and atropine and his condition improved. Cardiac catheterization showed patent coronaries. Acute coronary syndrome is a rare manifestation of organophosphorus compound (OPC) poisoning. The current case and subsequent review of literature tells us the need for close cardiac monitoring of all patients with OPC poisoning.

Introduction

OPC poisoning is very common in India where farmers form a significant proportion of the population who commonly use it as insecticides. OPC poisoning can cause cholinergic symptoms like salivation, lacrimation, urination and defecation. Nicotinic symptoms like neck muscle weakness, ocular weakness, proximal muscle weakness and respiratory muscle weakness can occur as a part of intermediate syndrome. ECG changes like transient ST-T wave changes, QT prolongation, atrial and ventricular arrhythmias can occur[1]. Few cases of myocardial infarction (MI) after OPC poisoning has been reported. We report a young man who developed myocardial infarction after OPC (monocrotophos) poisoning.

Case

22 year old young man got admitted in our toxicology ward with alleged history of consumption of 15 ml of monocrotophos poison in his house. He was initially taken to the nearby private hospital where gastric lavage and activated charcoal was given. He had presented to the hospital with complaints of chest pain. Chest pain was left sided and diffuse and 8/10 in intensity. He also had shortness of breath at the time of presentation. No palpitation or syncope was noted. He also had increased salivation. Review of system was negative for other complaints. He had no significant past medical history. He was a nonsmoker and did not drink alcohol. He was not allergic to any medications. Physical examination revealed moderately built male. Cardiopulmonary examination was clinically normal. Abdomen was soft and he had bilateral constricted pupils on neurological examination.

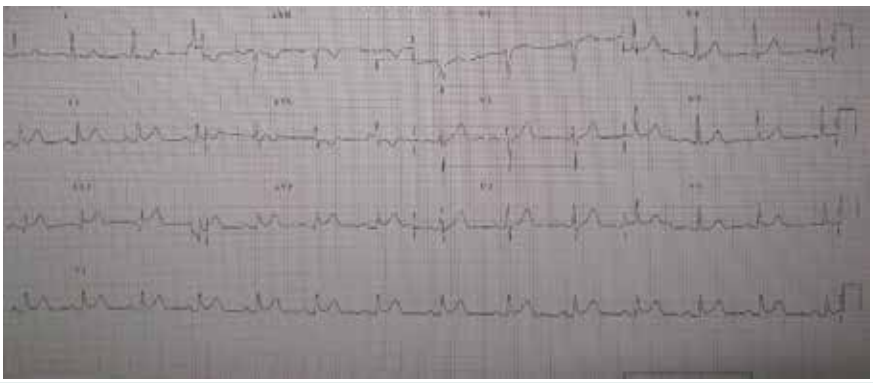

Fig. 1: ECG of the patient showing ST-elevation in lead 2, 3 and AVF— Inferior wall myocardial infarction

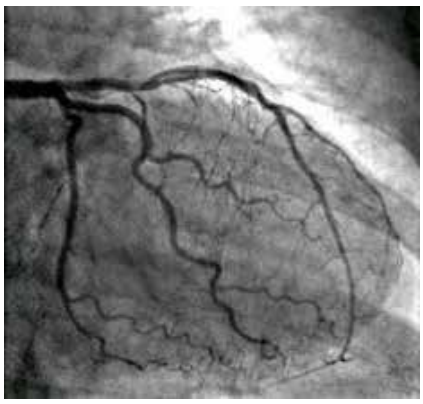

Fig. 2: Coronary angiogram of the patient which reveals patent coronary vessels

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ECG which was taken revealed ST elevation in leads II, III, AVF (Figure-1).His vitals were stable. He was then referred to the government general hospital, Chennai. In our center, serum CPK-MB, troponins were immediately done which were elevated. Echocardiogram was done which showed regional wall motion abnormality in the inferior wall of the left ventricle. Serum cholinesterase levels were 1172 IU/dl which is low. Serum homocysteine levels, PT/INR, APTT, antithrombin, lupus anticoagulant and anticardiolipin antibodies were within normal limits. On the next day serum pro-NT BNP levels was done which was elevated. Patient was treated with pralidoxime, atropine, anticoagulant and antiplatelet drugs. Following this treatment, the patient's serum cholinesterase levels improved, chest pain recovered. Coronary angiogram (Figure 2) was done the next day which was found to be normal. Patient's medical condition improved and he was discharged.

Discussion

Cardiac complications often accompany poisoning with OPC. These may be serious and often fatal, being represented by cardiac arrhythmias, electrocardiographic abnormalities and conduction defects, as well as MI, a rarely reported complication of OPC poisoning. The extent and pathogenesis of cardiac toxicity from these compounds is not yet clearly defined. In literature we had few cases of MI occurring after OPC poisoning. Lionte C et al[1] reported a 57 year old woman who developed anteroseptal MI and succumbed to death. Kiss Z et al[2] reviewed 168 cases of OPC poisonings with special respect to frequent arrhythmias. In five patients a transient picture of MI was seen. Dayton S.B et al[3] reported increased risk of MI among farm women exposed to pesticides. A rare case of MI due to parathion poisoning was reported by Yajneesh kidiyoor et al.[4] The affected patient was a farmer from rural India who had succumbed to the complications of MI. Madhu Pankaj[5] et al reported a 30 year old male who had taken chlorpyrifos and had presented with anterior wall myocardial infarction. Edibe Karasu[6] et al also reported a 52 year old patient who had presented with inferior wall myocardial infarction after parathion ingestion. In patients with angiographically smooth coronary arteries, acetylcholine has been reported to produce both vasodilation and constriction. The development of vasoconstriction is likely to be an abnormality of endothelial function that precedes atherosclerosis or an early marker of atherosclerosis not detectable by angiography. This is a likely mechanism in our patient. Coronary vasoconstriction response in isolated perfused heart mediated by M 3 receptor has been reported in rats. The cardiovascular manifestations also reflect mixed effects on the autonomic nervous system. Increased sympathetic tone is often initially present and most patients manifest as sinus tachycardia and sometimes hypertension. As toxicity becomes more severe, bradycardia with a prolonged PR interval and atrio-ventricular blocks of various degrees occur because of excessive parasympathetic tone and possibly because of reduced coronary blood flow.

Conclusion

Cardiac complications often accompany poisoning with OPC, particularly during the first few hours. Hypoxemia, acidosis, and electrolyte derangements are major predisposing factors. Close monitoring in intensive or coronary care facilities with administration of antidotes in adequate doses early in the course of the illness will improve the outcome.

Conflict of interest

The authors of the paper declare that there is no conflict of interests involved regarding the publication of this paper.

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