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(CASE REPORT)

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Bane toxic left the heart hypoxic

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Abstract

Introduction: Acute myocardial infarction as a result of acute organophosphorous poisoning has been reported but is exceptionally rare. We present a patient with consumption of Chlorpyriphos 50% who developed acute myocardial infarction with cardiogenic shock.

Case report: A 45 year old female patient was brought to our Emergency Department with alleged history of consumption of Chlorpyriphos 50%, 2 hours prior to presentation. She had brown colored vomiting, altered sensorium. On arrival she was tachycardic, hypotensive and she had tachypnea. Initial resuscitation was done, she was started on Atropine and Pralidoxime. Crystalloid fluids bolus was given for hypotension but patient did not respond for the same hence she was started on vasopressors. Initial ECG in the ED revealed non sustained ventricular tachycardia followed by ST segment elevation in Anterior chest leads. We diagnosed it as Acute ST-Elevation myocardial infarction with cardiogenic shock. She was thrombolysed with streptokinase. But she expired after 24 hours of thrombolysis.

Conclusion: Acute myocardial infarction is a rare association with Organophosphorous poisoning. Though prognosis with organophosphorous poisoning is good with appropriate and timely treatment but when associated with acute MI, there is increased risk of mortality.

Keywords: Organo-phosphorous poisoning; Acute myocardial infarction; Stemi; Chlorpyrifos; Insecticide

1. Introduction

Chlorpyrifos 50% is a insecticide of Organo phosphorus and Synthetic pyrethroids group. Organophosphate are the insecticides most commonly associated with systemic illness. This case is very unusual and presented because of it's rarity.

2. Case report

A 45 yrs old female with suicidal intention had consumed chlorpyrifos 50%, presented after 5 hours of consumption to our hospital.She had 4 episodes of vomiting, dark brown, watery, non blood tinged, non bile stained, followed by altered sensorium. patient was referred from a government hospital where she received 20 cc of atropine, 2gm of pralidoxime. There was no history of seizures, no breathlessness, chest pain. patient had no comorbidities.

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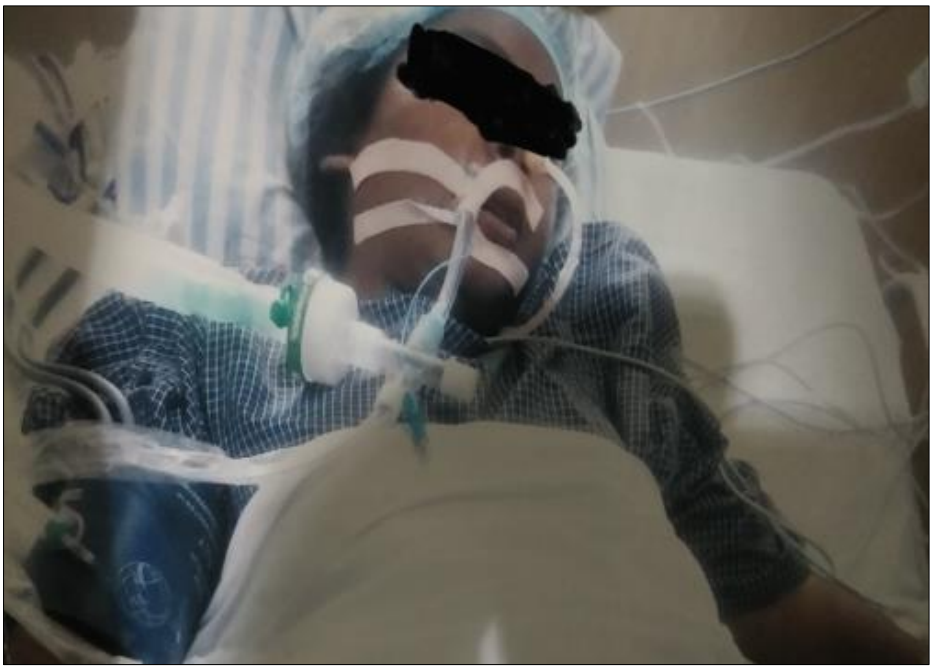

Figure 1 Picture of patient x

2.1. Vital signs

HR-118/min, RR-16/min, BP-80/50 mmhg, SPO_2-70% with 15% Liters of O_2, GRBS-192 mg/dl. ecg-sinus tachycardia, ST elevation in lead II, III, AVF, v4-v6.systemic examination:-RS-bilateral air entry present, CNS-GCS E2V1M5, b/l pupils 5mm dilated sluggish reacting to light.CVS-S1S2 heard. Per abdomen-was soft.

2.2. Investigations

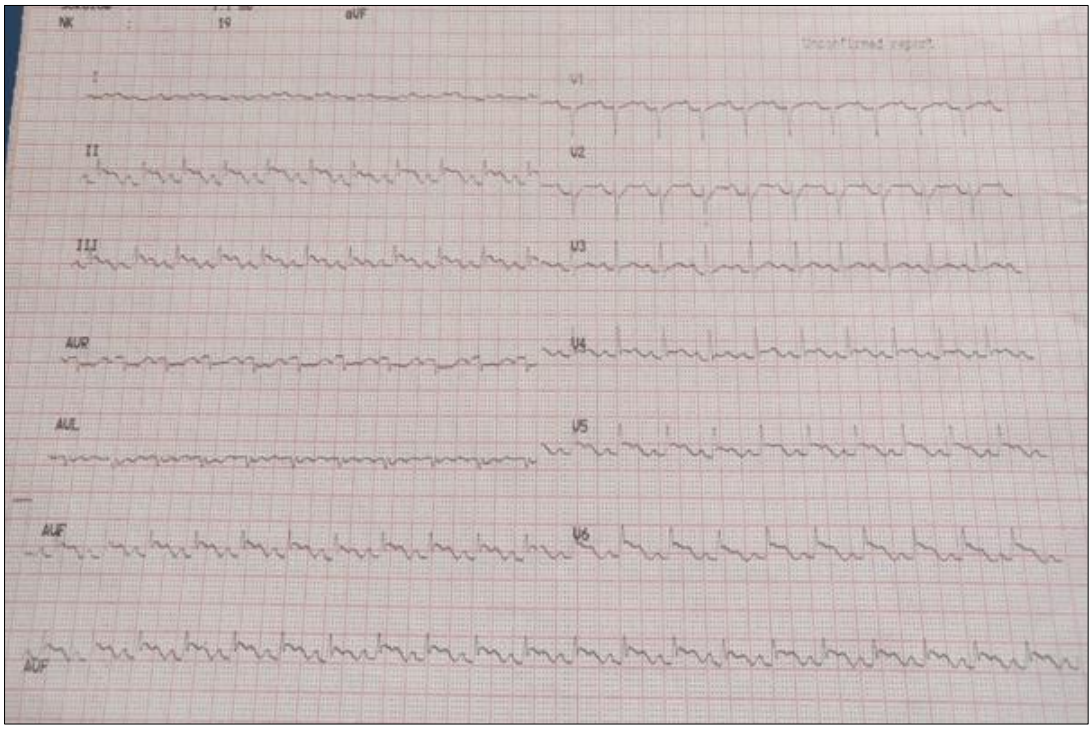

Figure 2 Image of ECG prethrombolysis

Troponin I-3.79ng/dl, ckmb-13.8 ng/dl. abg-lactic acidosis, Tc-17400.S.pseudocholinesterase-450 IU.2D echo-severe global hypokinesia (ant wall thinned and akinetic) with ejection fraction of 30%.Ct brain-normal.rest all blood investigations were found to be normal

3. Intervention

3.1. Treatment

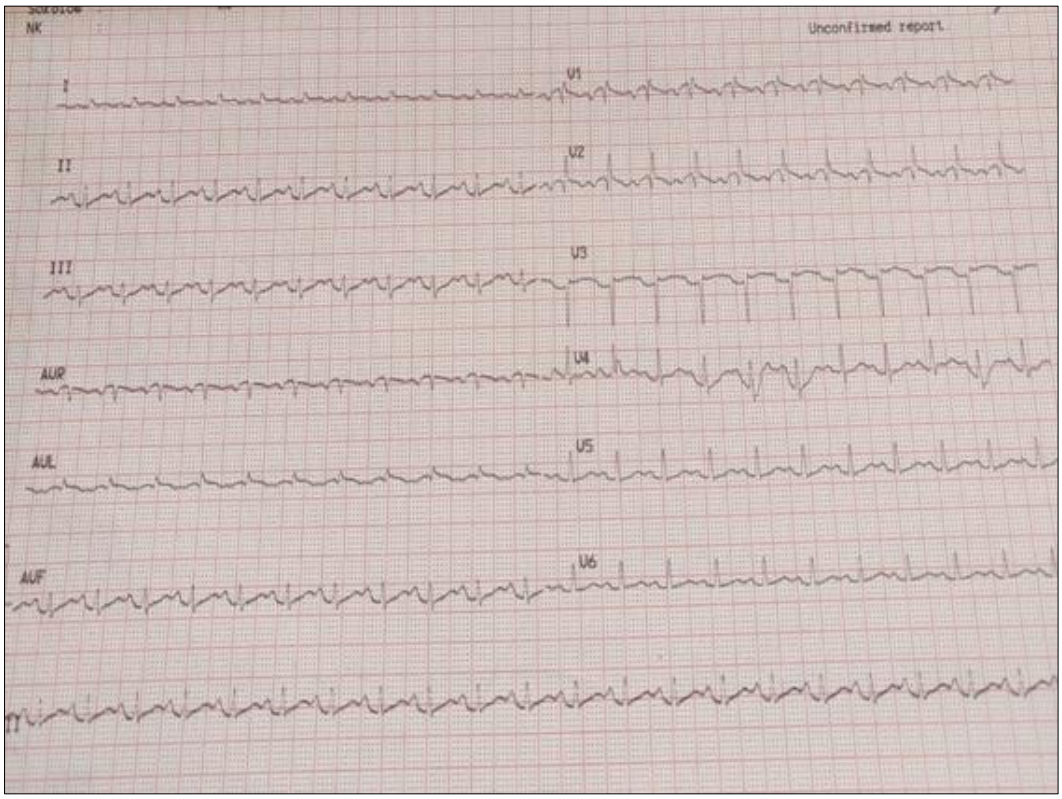

Figure 3 Image ECG post thrombolysis

Gastric lavage was given.pt was intubated in view of hemodynamic instability, IV fluids normal saline 1ooo ml bolus iv, inj atropine iv @5ml/hr, inj pralidoxime iv @20ml/hr, inj ondansetron 4 mg iv. inj magnesium suphate 4gm in 100 ml ns over 20 min. inj noradrenaline @5ml/hr. patient was shifted to Eicu and thrombolysed with inj streptokinase 1.5 million IU stat. inj piperacillin and tazobactum, inj dobutamine @8ml per hr was started iv, 4 fresh frozen plasma was transfused.

4. Discussion

Organophosphates inhibit enzyme cholinesterase which causes accumulation of acetylcholine in neuromuscular junction.they bind irreversibly by phosphorylation.treatment has to be started before the aging starts.aging is a term describing the permanent irreversible binding of organophosphorous compound to cholinesterase.once aging sets in antidote cannot be regenerated cholinesterase(4).OP poisoning are associated with the cardiac complications and most of them occur during the first few hours after exposure.. Possible mechanism of cardiac toxicity after OPP may include phase 1, a brief period of increased sympathetic tone; phase 2 a prolonged period of parasympathetic activity; and phase 3 in which Q-T prolongation followed by torsade de pointes ventricular tachycardia and then ventricular fibrillation occur.This parasympathetic over activity plays a major role in the coronary artery spasm which is an important factor in pathogenesis of myocardial infarction[1]. Also, pesticides release increased the amount of catecholamines and other vasoactive amines (histamines and neutral proteases) that penetrate the collagen matrix of plaque causing erosions and rupture which can lead to myocardial injury[2]. These inflammatory mediators can cause coronary thrombosis, as well as spasm leading to myocardial infarction.

But the exact pathophysiology behind organophosphorus compound leading to cardiac manifestations is not clearly understood. Apart from the direct toxic effect of the organophosphorus compounds on myocardium, over activity of cholinergic or nicotinic receptors, the increase in sympathetic and/or parasympathetic activity, hypoxemia, electrolyte abnormalities and acidosis have been hypothesized to play a role in the damage caused to myocardium along with the high dose of atropine[3].

5. Conclusion

Acute myocardial infarction is a rare association with Organophosphorous poisoning. Though prognosis with organophosphorous poisoning is good with appropriate and timely treatment but when associated with acute MI, there is increased risk of mortality.if we already have a history that patient is known case of ishemic heart disease we can start infusion rather than bolus doses of atropine injection.

Recommendations

Monitoring of cardiac enzymes

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from patient party who were included in the study.

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