JMSCR Vol||05||Issue||11||Page 29940-29942||November 2017

www.jmscr.igmpublication.org
Impact Factor 5.84
Index Copernicus Value: 71.58
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: https://dx.doi.org/10.18535/jmscr/v5i11.21

IGM Publication
Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Dimethoate Poisoning Induced Toxic Cardiomyopathy – Case Report

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Abstract
Organophosphates are commonly used as insecticides and are most common suicidal agents in developing countries. Poisoning is common in young adults, farmers and accounts for 35% to 40% of all suicidal deaths in India. Acute self poisoning with Dimethoate has human case fatality three fold higher than poisoning with chlorpyrifos and other pesticides. Clinical presentation of dimethoate is distinct from other OPC compounds with the presentation of severe hypotension and shock which is irreversible and leads to toxic cardiomyopathy with a low ejection fraction.
Keywords: *Dimethoate,Toxic cardiomyopathy.*

Introduction

Organophosphates are extensively used as agricultural insecticides, and some of those compounds serve as nerve agents for chemical warfare[1]. They act as effective inhibitors of acetylcholinesterase. Liver is the organ where activation and detoxification of organophosphates occurs, however they're eliminated through kidneys[2]. OP compounds can be divided into types: diethyl (e.g. Chlorpyrifos, diazinon, parathion, phorate and dochlofenthion) and dimethyl (e.g. Dimethoate, dichlorvos, fenitrothion, malathion and fenthion).

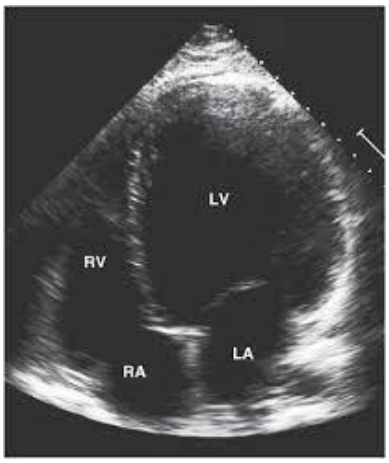
Clinical presentations are categorised as muscarinic and nicotinic which includes bradycardia, hypotension, tachycardia, increased salivation, vomiting, lacrimation, sweating, diarrhea, abdominal pain, fecal and urinary incontinence[3]. CNS manifestations includes anxiety, restleness, convulsions, miosis, coma, cheynes stokes breathing, respiration and cardiac failure[4]. Chronic OPC poisoning induces delayed neuropathy and seen in farmers[5].

Case Report

A 22 yr old male patient presented to casualty with alleged H/O consumption of Dimethoate 30% poison of about a hundred ml mixed in alcohol at round 2.00 pm on 21/4/17 in his farm. He was found unconscious and was taken initially to local GH and was referred to RMMCH at 5.30 pm. O/E Patient was unconscious, not responding to painful stimuli. VITALS: GCS – E1V1M1, PR: 116 b/min, BP:? SPO2: 45%, RR: 42 /mt, T: 98.4 f. NO SLUDGE, Breath smelled of OPC. CVS: S1S2+, sinus tachycardia, RS: NVBS, B/L AE+,

crepts +all over lung fields, P/A : SOFT, BS +, CNS: GCS 3/15, PUPIL B/L 3mm, RTL, No focal deficits.

Immediately stomach wash was given. Ryles tube was in situ to prevent aspiration. He was immediately intubated and treated with Inj. Atropine at a rate of 16 mg/hr. Pralidoxime 2 g stat followed by 500 mg Tds was given. Inotropes was started and titrated accordingly. Inspite of maximal inotropes support, there was refractory hypotension. Hence to find the cause for persistent hypotension and tachycardia, screening echo was done which showed dilatation of all chambers of heart, global akinesia of LV, poor systolic function with EF of 12% suggestive of Toxic Cardiomyopathy.



Then patient went in for VT (ventricular tachycardia) following sudden cardiac arrest.

As per as ACLS protocol, CPR was carried out, but despite effective resuscitative measures he couldn't be revived.

Investigations

RBS	84 mg/dl
SR.Cholinesterase	1855 IU/L,
S.Na	146
S.K	4.4
S.cl	98 mmol/l
S.urea	24 mg/dl
S.creat	0.6 mg/dl
Total count	7,800 cells/cu.mm

ECG

1. HR 120 b/min, Normal axis, ST elevation in AVR, ST depression in 2, 3 , AVF , V3-V6
2. Ventricular tachycardia.

Discussion

Dimethoate causing persistent hypotension: Acute self poisoning with dimethoate has a human case fatality three fold higher than other OPC compounds. Many pts usually present with hypotension that progresses to shock and death within 12-48 hrs of ingestion. This intractable hypotension was due to very high plasma conc. Of OPC which was due to low lipid solubility of dimethoate[6]. Possible pathophysiological mechanisms was due to cardiotoxic effect on peripheral vasodilatation causing a distributive shock with decreased systemic vascular resistance[7].

Persistent hypotension leading to tachycardia: The possible mechanisms of cardio toxicity are related to sympathetic and parasympathetic over activity, hypoxemia, acidosis, electrolyte de–rangements and a direct toxic effect of the compounds on the myocardium. Sinus tachycardia was due to nicotinic effect of OPC compounds .

Toxic exposure leads to arrythmias or respiratory arrest: As tachycardia was persistent, in conjunction with the exposure of toxins cardiomyopathy occured causing LV dysfunction. Abnormalities of autonomic nervous system and Renin angiotensin aldosterone axis appears to promote the occurrence of ventricular arrythmias.

Dimethoate poisoning causing death: Over 40% of patients die from dimethoate poisoning with SBP <80 mmHg compared to 5% of pts from chlorpyriphos poisoning[8].

Conclusion

In this case study, young patient with persistent hypotension which was intractable and irreversible in spite of double inotropic support with ECHO featuring dilatation of all four chambers with global akinesia of LV and EF of 12%. Hence we concluded that cardiomyopathy is

the cause for cardiac complication and the probable etiology might be due to toxin (OPC) induced. We report this case for its rareity. Physicians, specially in rural India should be aware of varied manifestation of OPC and must be prepared to treat such complications if need arises.

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