

Acute Atrial Fibrillation Induced by Organophosphorus Poisoning: Case Report

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Abstract

Organophosphorus poisoning (OPP) remains a major clinical and public health problem in developing countries. OP compounds act as irreversible cholinesterase inhibitors. Toxicity of these compounds leads to cholinergic excess symptoms through the muscarinic, nicotinic, and central nervous system receptors. Cardiac complications include cardiac arrest, pulmonary oedema, and arrhythmia. Rarely, myocardial infarction has also been reported. Cardiac injury is a strong predictor of death in these patients. We are reporting a case of OP poisoning in a child recently admitted to our facility with acute onset of atrial fibrillation which reverted to sinus rhythm by direct current (DC) cardio version and detoxification of OP component.

Keywords: Organophosphorus poisoning; Cardiac toxicity; Atrial fibrillation

Case Report

A 12-year-old child with no past medical or surgical was referred our medical facility complaining of irregular palpitations following accidental exposure to OP compound in a farm. On clinical examination, his pulse was 120 beats/minute, irregular, blood pressure of 100/60 mmHg, with bilateral pinpoint pupils and fasciculations, were noted in calf muscles. Auscultation revealed bilateral chest crepitations with no other adventitious heart sounds or murmurs. Chest radiography showed normal sized cardiac shadow with mild pulmonary congestion. Electrocardiographic (ECG) revealed atrial fibrillation with a rapid ventricular response. A two-dimensional echocardiography was done showing a normal study in respect to valvular morphology and ventricular functions. Routine laboratory findings demonstrated adequate hemoglobin, normoglycemia with normal serum electrolytes, and total leukocytic count. Renal, liver, and thyroid function tests were within normal limit. The patient started supportive measures including intravenous atropine and pralidoxime in a toxicology unit and reverted to normal sinus rhythm by direct current cardio version. He was discharged home safely after a two days follow-up.

Discussion

OP compounds phosphorylate the active site of acetyl cholinesterase, inactivation of the enzyme causes accumulation of receptors causing widespread clinical symptoms like nausea, vomiting, diarrhea, urinary incontinence, blurring of vision, salivation, lacrimation, broncorrhoea, bradycardia, hypotension, muscle paralysis, fasciculation, confusion, seizures, coma and respiratory failure [4]. Cardiac complications may vary from asymptomatic ECG abnormalities such as sinus tachycardia, sinus bradycardia,

acetylcholine which excessively stimulates nicotinic and muscarinic receptors causing widespread clinical symptoms like nausea, vomiting, diarrhea, urinary incontinence, blurring of vision, salivation, lacrimation, broncorrhoea, bradycardia, hypotension, muscle paralysis, fasciculation, confusion, seizures, coma and respiratory failure [4]. Cardiac complications may vary from asymptomatic ECG abnormalities such as sinus tachycardia, sinus bradycardia, atrioventricular block, prolonged Q-Tc interval, and ST-T changes to life-threatening complications such as cardiac arrhythmias, hypertension, hypotension, myocardial ischemia, and noncardiogenic pulmonary edema [5,6]. Paul and Bhattacharyya [7] described ECG abnormalities in OPP and concluded that prolonged Q-Tc interval was the most common ECG abnormality, found in 67 patients (62.6%) and followed by sinus tachycardia in 36 patients (33.6%) and sinus bradycardia in 33 patients (30.8%). Elevation of ST segment was seen in 27 patients (25.2%) with T-wave inversion in 21 patients (19.6%). First-degree heart block (P-R interval >0.20 s) occurred in 9 cases (8.4%). Atrial fibrillation was seen in 5 patients (4.6%). Ventricular tachycardia occurred in 6 cases (5.6%) and ventricular premature complexes were noted in 3 patients (2.8%). Management includes supportive measures: These include decontamination where clothes are removed off the patient and the patient are thoroughly cleaned gently with soap and water. Eyes are irrigated with normal saline. Gastric decontamination is done by frequent gastric lavage. Oxygen support (ventilatory support may be required in cases of severe intoxication causing bronchorrhea induced bronchospasm or respiratory muscle paralysis), intravenous fluids, and maintaining electrolyte balance [8]. The mainstay of treatment is atropine, pralidoxime, and benzodiazepines (for anxiety, restlessness and seizure control).

Conclusion

Cardiac complications of OPP can be associated with a wide variety of manifestations. Acute atrial fibrillation though it is a rarity, yet it can complicate a setting of OPP. So, careful patient's monitoring is required by treating physicians. Citation: Abdelnaby MH (2018) Acute Atrial Fibrillation Induced by Organophosphorus Poisoning: Case Report. J Clin Toxicol 8: 370. doi: 10.4172/2161-0495.1000370

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References

- 1. Maheshwari M, Chaudhary S (2017) Acute atrial fibrillation complicating organophosphorus poisoning. Heart views 18: 96-99.
- Organization WH (2000) Epidemiology of pesticide poisoning: harmonized collection of data on human pesticide exposures. International Programme on Chemical Safety.
- 3. Yamashita M, Tanaka J, Ando Y (1997) Human mortality in organophosphate poisonings. Vet Hum Toxicol 39: 84-85.
- 4. Yang CC, Deng JF (2007) Intermediate syndrome following organophosphate insecticide poisoning. J Chin Med Assoc 70: 467-472.
- 5. Karki P, Ansari J, Bhandary S, Koirala S (2004) Cardiac and electrocardiographical manifestations of acute organophosphate poisoning. Singapore Med J 45: 385-389.
- Yurumez Y, Yavuz Y, Saglam H, Durukan P, Ozkan S, et al. (2009) Electrocardiographic findings of acute organophosphate poisoning. J Emerg Med 36: 39-42.
- 7. Paul UK, Bhattacharyya AK (2012) ECG manifestations in acute organophosphorus poisoning. J Indian Med Assoc 110: 107-108.
- 8. Rawal G, Yadav S, Kumar R (2016) Organophosphorus poisoning: A case report with review of literature. Indian J Immunol Resp Med 1: 20-22.