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## A rare case report of organophosphorus poisoning induced bidirectional ventricular tachycardia

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### Abstract

Bidirectional ventricular tachycardia is a rare form of ventricular arrhythmia, characterized by changing QRS axis of 180 degrees. Digitalis toxicity is considered as commonest cause of Bidirectional ventricular tachycardia; other causes include aconite toxicity, myocarditis, myocardial infarction, metastatic cardiac tumors and cardiac channelopathies. We describe a case of bidirectional ventricular tachycardia in a 40 year old male patient with Organophosphorus poisoning.

**Keywords:** organophosphorus poisoning, Bidirectional ventricular tachycardia

### Introduction

Organophosphates have been used as insecticides worldwide for more than 50 years. The use of these agents has declined in the last 10 to 20 years, in part due to the development of carbamate insecticides, which are associated with similar toxicities [1].

Medical applications of organophosphates and carbamates include reversal of neuromuscular blockade (neostigmine, pyridostigmine, edrophonium) and treatment of glaucoma, myasthenia gravis, and Alzheimer disease.

Worldwide, an estimated 3,000,000 people are exposed to organophosphate or carbamate agents each year, with up to 300,000 fatalities [2, 3]. Toxicity generally results from accidental or intentional ingestion of, or exposure to, agricultural pesticides [2, 4]. Other potential causes of organophosphate or carbamate toxicity include ingestion of contaminated fruit, flour, or cooking oil, and wearing contaminated clothing [4, 5]. We present a case of life-threatening bidirectional ventricular tachyarrhythmia that occurred after the ingestion of Organophosphorus.

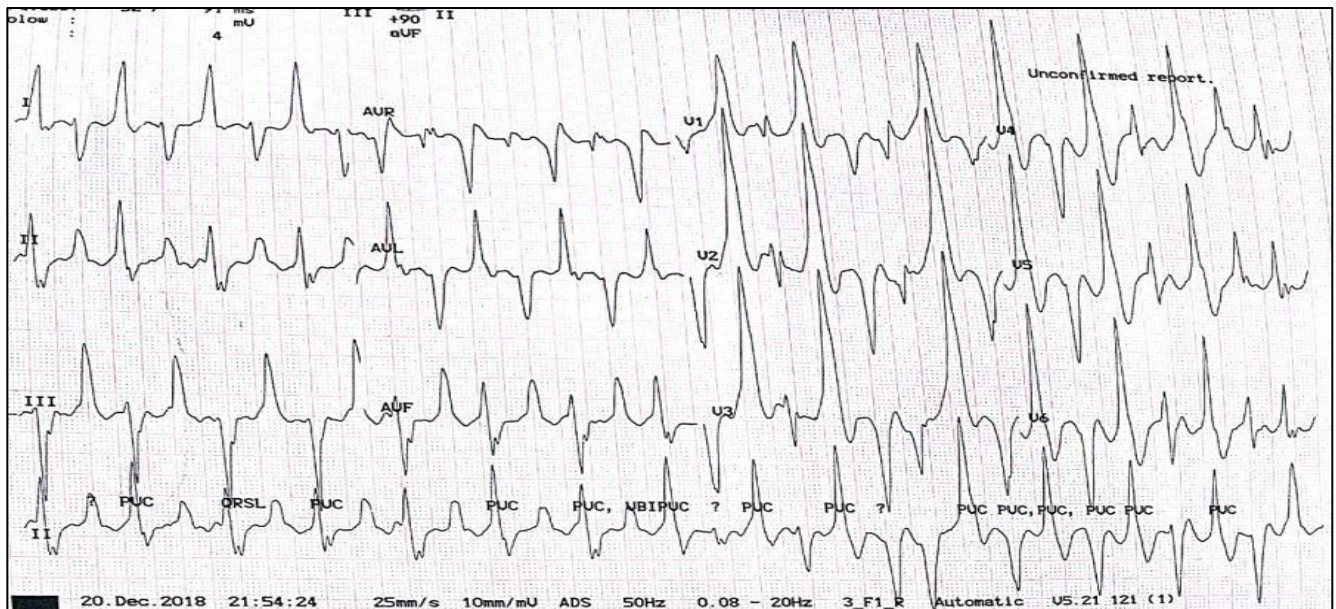
### Case Report

A 40-year-old male who was admitted to our hospital with alleged history of Organophosphorus poisoning. Patient was agitated with diaphoresis, lacrimation, vomiting with pin point pupil. On examination, his blood pressure was 80/40 mm Hg, with heart rate of 140 /minute, saturation was 90% at room air. On Neurology examination his GCS was E2V1M5 and bilateral pupils were pin point. Routine laboratory investigation revealed- Hb 15 g/dl, TLC 16000/mm<sup>3</sup> and platelets were 324000/mm<sup>3</sup>. PTI 11.3, INR 1.04, Blood Urea 37mg/dl, Serum Creatinine 0.7mg/dl, Na 141 mmol/L, potassium was 3.52mmol/l. LFT were normal. ABG severe acidosis - PH 6.8, PCO<sub>2</sub> 98, PO<sub>2</sub> 225, k 2.6, lactate 5.9, HCO<sub>3</sub> 17, SPO<sub>2</sub> 99. Routine cardiac biomarkers such as CPK-MB and Trop T was normal (0.05), BNP 5, D Dimer 188. 2D echocardiography revealed normal ejection fraction with no regional wall motion abnormality, no valvular pathology.

Cholinesterase levels were 160 U/L (Normal 5320 to 12920)

Electrocardiography revealed bidirectional ventricular tachycardia (QRS 150ms), with a heart rate of 180 beats/min (Fig 1).

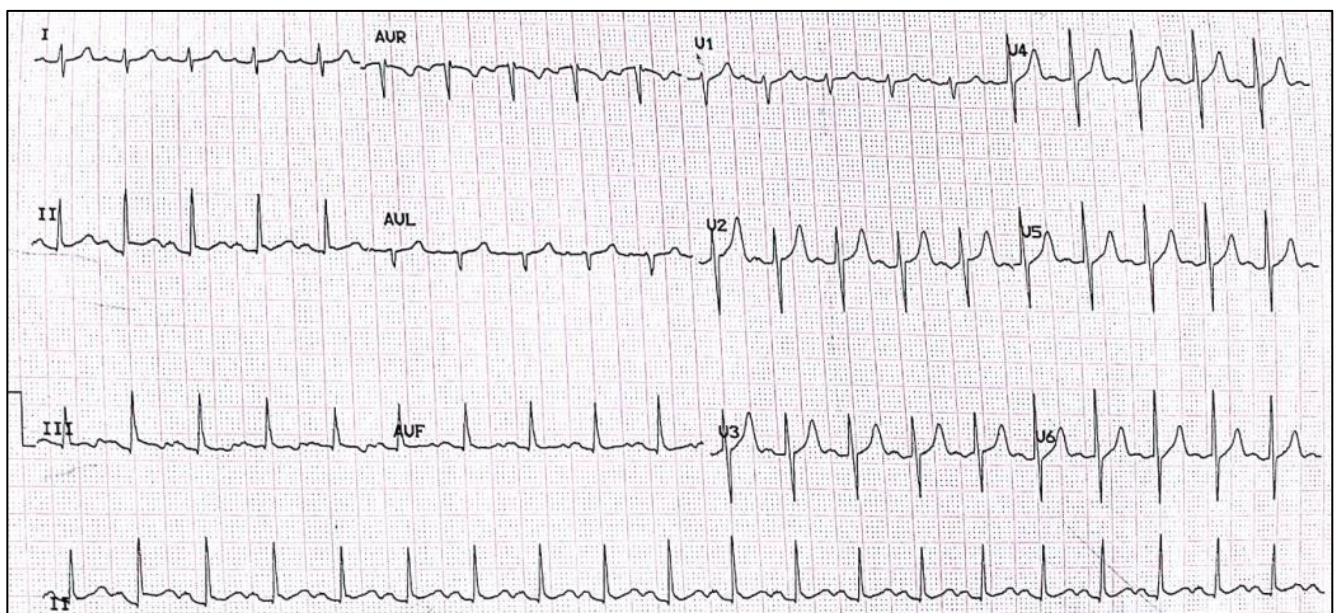
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**Fig 1:** Electrocardiogram showing Bidirectional ventricular tachycardia following organophosphorus poisoning.

There was no past history of Diabetes mellitus, Hypertension, Ischemic heart disease or any other structural heart disease. There was no family history ventricular tachycardia or Sudden Cardiac Death.

Patient was put on mechanical ventilation. Intravenous fluids started and patient was defibrillated with 200J. Following defibrillation complete resolution of tachycardia occurred as shown in figure 2.



**Fig 2:** complete resolution of ventricular tachycardia following defibrillation with 200 J.

Then patient was started on intravenous Atropine infusion and norepinephrine infusion to maintain MAP >70 mm Hg. Electrolytes were kept in normal range. But next day patient developed acute kidney injury with severe metabolic acidosis and planned for Continuous Renal Replacement Therapy. But patient developed cardiac arrest and expired.

### Discussion

Cardiac manifestations are observed in about two-thirds of patients with Organophosphorus poisoning. Common electrocardiographic findings are QTc prolongation, ST-T segment changes and T wave abnormalities [6].

Other cardiac manifestations include sinus bradycardia or tachycardia, hypotension or hypertension, supraventricular

and ventricular arrhythmias and ventricular premature complexes and non-cardiogenic pulmonary edema [7].

Death due to cardiac causes in Organophosphorus poisoning occurs either due to arrhythmias or severe and refractory hypotension [8] Although shock is primarily vasodilatory [8, 9]. circumferential endocardial ischemia with cardiogenic shock leading to death has also been reported with Malathion poisoning [10].

Necropsy of patients who died following Organophosphorus poisoning has revealed cardiac discoloration or blotchiness, patchy pericarditis, auricular thrombus and right ventricular hypertrophy and dilatation. Myocardial interstitial edema, vascular congestion, patchy interstitial inflammation, mural thrombus and patchy myocarditis were the histological findings. Organophosphorus poisoning presenting as cardiac

arrest <sup>[11]</sup> and late onset, prolonged asystole 12-day following poisoning <sup>[12]</sup> have also been described.

Bidirectional ventricular tachycardia is a rare arrhythmia characterized by beat-to-beat change in QRS axis on electrocardiogram. Bidirectional VT usually associated with digoxin toxicity, myocarditis, myocardial infarction <sup>[13]</sup>, metastatic cardiac tumour <sup>[14]</sup>, herbal aconite poisoning and cardiac channelopathies i.e. catecholaminergic polymorphic Ventricular tachycardia and Anderson-Tawil syndrome <sup>[15]</sup>

### Conclusion

Our case is rare because there are no case reports of Organophosphorus poisoning presenting with Bidirectional ventricular tachycardia.

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