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# Heart Failure with Preserved Ejection Fraction - A Rare and Late Complication of Organophosphorus Poisoning

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**Abstract:** ***Organophosphorus poisoningis a major medical and public-health problem across rural Asia. Here we highlight an unexpected and late cardiac complication of cardiomegaly and heart failure in a young patient post organophosphorus consumption.A 24-year-old female with no known comorbidities presented to ED 3 hours after consumption of insecticide, with symptoms typical of Organophosphorus poisoning and was managed on the lines of poisoning. Late during the hospital course, she developed sudden onset retrosternal heaviness. Serial ECGs showed non-specific ST changes with negative cardiac markers, increased cardiothoracic ratio on X-ray and raised NT-ProBNP. A Tc99m SISTAMIBI rest cardiac scan confirmed infero-basal myocardial ischemia and was managed medically.***

**Keywords:** Organophosphorus poisoning; cardiomyopathy; cardiomegaly; heart failure with preserved ejection fraction

## 1. Background

Organophosphorus is widely used as insecticide in agriculture and easily available in the market so that it is frequently used for suicide and homicide and is a major medical and public-health problem across the rural Asia.[1] Worldwide over 200,000 people die of acute organophosphate poisoning with mortality rates usually rising above 15%.[2] It acts by irreversibly inhibiting acetylcholinesterase leading to acetylcholine accumulation at neuromuscular junction, causing muscarinic and nicotinic symptoms.[3] The damage to myocardium causes arrhythmia, heart failure, cardiogenic shock and sudden cardiac arrest, however heart failure and cardiomegaly are rarely reported as late complications and although considered fatal in acute setting, often overlooked.[4]

Here we present a case of 24-year-old female with acute OP poisoning developing heart failure with preserved ejection fraction and cardiomegaly as a late complication of severe organophosphate

## 2. Case Report

A 24-year-old female allegedly consumed approximately 15-20 ml of Novan (Dichlorvos; OP pesticide) following which she had 3-4 episodes of vomiting, urine incontinence with nasal secretions and frothing from mouth and followed by altered sensorium irrelevant talk, drooling of saliva and decreased responsiveness to commands. She was brought to our hospital and diagnosed as Organophosphorus poisoning. On examination, she had a BP of 90/60, Heart Rate of 40/min, RR – 28/min, GCS of E3V3M5 and pupils were constricted, 2 mm in diameter bilaterally and sluggishly reactive to light. Point of care ABG was indicative of acute respiratory alkalosis with hypokalemia. ECG was suggestive of sinus bradycardia and normal cardiac enzymes. She was atropinized along with securing airway, breathing and blood pressure, and pralidoxime. She was started on atropine infusion. Acetylcholinesterase (AchE) levels were found very low and the patient was given 4 units of Fresh frozen plasma. Atropine was tapered. She improved symptomatically and AchE levels were restored to normal after 3 days. The patient developed sudden onset shortness of breath, tachycardia and retrosternal chest pain with sweating after atropine was stopped. ECG was indicative of sinus tachycardia with no S-T changes and Trop I levels were normal. Chest X ray revealed increased cardio thoracic ratio as compared to baseline. Subsequently her ECG showed T wave changes initially in inferior leads then followed by diffuse t wave inversions in all leads over a period of 2 days in spite normal oxygenation and electrolytes. She continued to have intermittent chest pain, which tend to relive spontaneously and lasting less than 30 minutes. Cardiac markers remained negative serially, NT Pro BNP was raised (4600 pg/ml). D- Dimer was found to be raised, but CTPA showed no signs of pulmonary thromboembolism. 2 D ECHO during the first 3 days

showed decreased contractility, which later came back to normal with mild apical hypokinesia, left ventricular end diastolic diameter 57mm, Left atrial volume index – 30ml/$m^2$ and EF of 55%. Cardiac perfusion scan was done which confirmed the findings of decreased endocardial perfusion in basal areas of the heart, confirming a diagnosis of subendocardial ischemia as a late complication of OP poisoning.

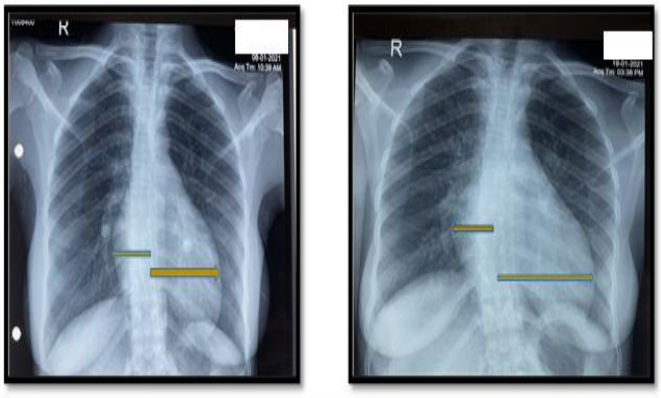

Fig 1. The X-ray on the left is the baseline X-ray with a CT ratio of 0.48 dated 08/01/2021 on 2nd day of OP poisoning. On the right, X-ray was taken on 19/01/2021 showing CT ratio of 0.60 corresponding to cardiomegaly. Both the X-ray are Chest PA view in adequate inspiratory effort.

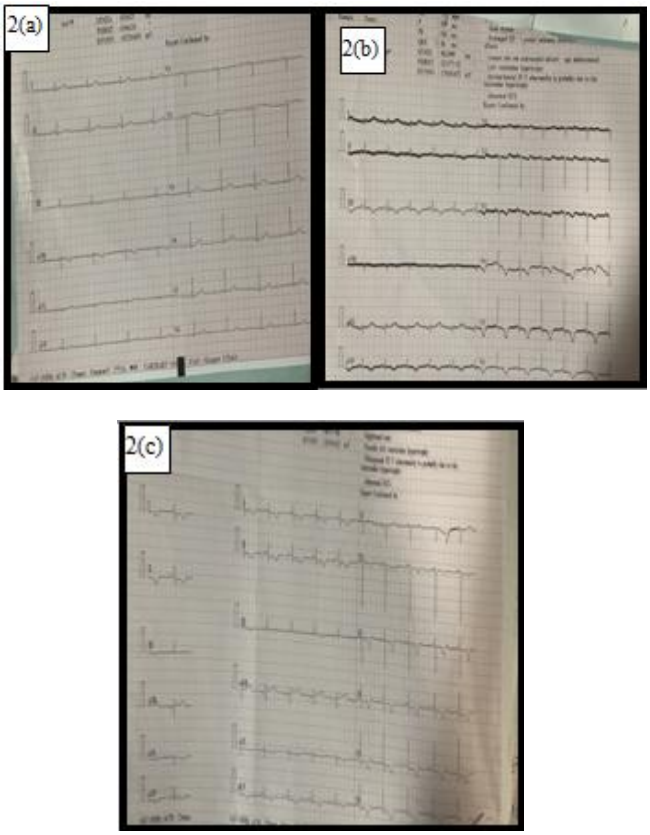


**Figure 2:** 2.a) Baseline ECG on the day of admission indicating sinus arrhythmia without any ST-T changes. 2.b) ECG on 11th day of admission shows T wave inversions in inferior leads. 2.c) ECG showing diffuse T stave inversions in all leads.

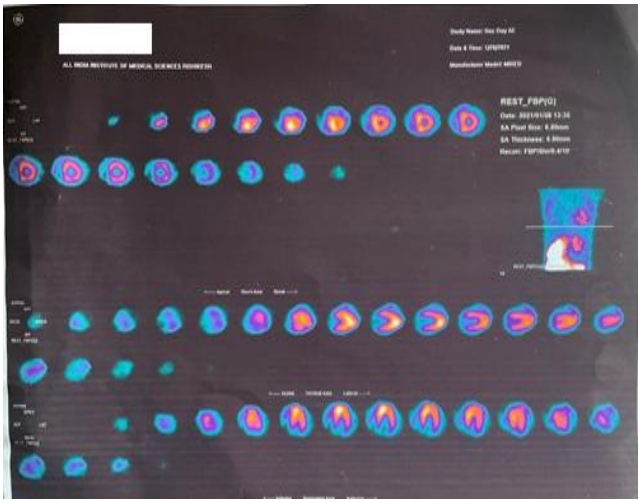

**Figure 3:** Cardiac perfusion scan of same patient 2 weeks after date of poisoning, illustrating slightly decreased perfusion at infero-basal areas of the cardiac tissue.

**Treatment and Follow Up**

The patient was managed with guideline directed medical therapy including antiplatelet, beta blockers, diuretic and ace inhibitors. Coronary angiography was planned. The patient improved symptomatically, but dyspnea NYHA grade 1 was reported on the follow up visit at 2nd week. Apex beat was still shifted laterally but no signs of congestion were present. Review 2D ECHO shows ejection fraction 60% with dilated chambers and no regional motion abnormality. ECG was suggestive of normal sinus rhythm and no ST T changes. The patient did not consent for coronary angiography as she had almost complete resolution of symptoms. She was planned for further follow up at 4th week.

## 3. Discussion

ECG manifestations including ST T changes, arrhythmias, and conduction disturbances are considered common in Organophosphorus and carbamate poisoning, heart failure and dilated cardiomyopathy is seldom reported. The commonly reported cardiac complications include – non-cardiogenic pulmonary edema (43%), ST-T changes (41%), cardiac arrhythmias (24%), and conduction defects (9%).[5] This further highlights the importance of identifying heart failure as a delayed manifestation of poisoning, as it affects the prognosis and further management. Possible mechanisms of cardio toxicity includes brief period of increased sympathetic tone, prolonged parasympathetic activity and QTc prolongation.[6] Kai-Xiang et al(2019) identified that heart failure with preserved ejection fraction was more common in patients with acute organophosphorus poisoning and its effects were correlating to not only the dosage but also the presentation time and solvent used for the poison. Similar results were supported by Ludomersky et al (1982).[7,8] The patient had heart failure with preserved ejection fraction as confirmed by raised NT Pro BNP levels and evidence of endocardial ischemia in perfusion scan and an ejection fraction of 58%. Other mechanisms in play include hypoxemia, acidosis, dyselectrolytemia and direct cardiac toxicity. Owing to the large quantity of poison consumption and Low acetylcholinesterase levels, increased parasympathetic activity could be the possible mechanism

of cardiac injury in this case. The initial few hours of poisoning have a high risk for fatal arrhythmias and myocardial infarction, but understanding and early detection of delayed effects myocardial damage might help in better prognostication of pesticide poisoning especially in developing nations.

Uniqueness of the case lies in the timing of an unexpected cardiac complication in an otherwise fairly common infirmity of rural India. Secondly, the diagnostic workup of heart failure and ischemic heart disease in an otherwise healthy and young woman with no risk factor. We would like to emphasize the importance of follow up for a medical condition which was once limited to acute management. Further study is needed for determination of exact cause of endocardial ischemia in OP poisoning.

## 4. Conclusion

OP poisoning is a medical emergency and it's not very uncommon, but the delayed complications are often overlooked. This case highlights the importance of close monitoring and follow up even after tiding over acute crisis. Identification of heart failure with preserved ejection fraction enhances our understanding regarding cardio toxic effects of pesticides and also correlates dosage of poison consumption with cardiac toxicity. It warrants high degree of suspicion and the need for rigorous cardiac monitoring and cardiac bio markers, once the patient develops shortness of breath, palpitations and any chest discomfort. Early detection and management of heart failure should be made integral of holistic approach towards OP poisoning management, which was once limited to atropine and oximes along with supportive care.

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