


Acute Coronary Syndrome After Insecticide Spraying: A Suspected Association with Organophosphate Exposure

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ABSTRACT

Introduction: Organophosphate is one of the most commonly used insecticides to control vector-borne illnesses. Both organophosphate and biodiesel (commonly used as solvents for outdoor space spraying methods) intoxications have cardiovascular manifestations. **Case**: A 58-year-old male with a sudden onset of shortness of breath after spraying insecticides 1 hour before admission. On examination, there were no SLUDGEM (salivation, lacrimation, urination, defecation, gastrointestinal distress, emesis, miosis) findings. Bibasilar rales were present on auscultation, and a 12-lead ECG showed ST elevation in leads V1-V6. There was no preceding chest pain. The patient received oxygen, antiplatelet loading, and insulin therapy for hyperglycemia and was prepared for primary PCI. Diagnostic coronary angiography revealed a mid-LAD total occlusion, and a drug-eluting stent was implanted with final TIMI 3 flow. **Discussion**: Dyspnea has various causes, including organophosphate (OP) poisoning, which may result in cholinergic syndrome, respiratory dysfunction, and life-threatening cardiovascular complications. Early recognition through rapid diagnostic evaluation and timely interventions such as atropine, oximes, and reperfusion therapy in STEMI are essential to improve survival and outcomes. **Conclusion**: Early recognition and prompt revascularization are critical in STEMI to reduce infarct size and improve outcomes. Organophosphate exposure may be associated with cardiovascular effects, and clinicians should consider acute coronary occlusion in patients with respiratory complaints after insecticide exposure, even in the absence of classical cholinergic signs.

Keywords: Case report, insecticides spraying, organophosphate, STEMI.

ABSTRAK

Pendahuluan: Organofosfat adalah salah satu insektisida yang paling sering digunakan untuk mengendalikan vektor/nyamuk. Baik keracunan organofosfat maupun biodiesel (bahan pelarut yang sering digunakan pada metode penyemprotan di luar ruang) dapat memiliki manifestasi kardiovaskular. **Kasus**: Seorang laki-laki berusia 58 tahun datang dengan keluhan sesak napas mendadak setelah melakukan penyemprotan insektisida 1 jam sebelum masuk rumah sakit. Pada pemeriksaan tidak ditemukan gejala SLUDGEM (*salivation, lacrimation, urination, defecation, gastrointestinal distress, emesis, miosis*). Didapatkan ronki pada kedua lapang bawah paru, dan EKG 12 sadapan menunjukkan elevasi ST pada V1–V6. Tidak ada nyeri dada sebelumnya. Pasien mendapat oksigen, *loading* antiplatelet, serta terapi *insulin* untuk hiperglikemia dan dipersiapkan untuk PCI primer. Angiografi koroner menunjukkan oklusi total LAD segmen tengah, dan pemasangan *drug-eluting stent* menghasilkan aliran TIMI 3 akhir. **Diskusi**: Dispnea memiliki berbagai penyebab, termasuk keracunan organofosfat (OP), yang dapat menyebabkan sindrom kolinergik, gangguan pernapasan, dan komplikasi kardiovaskular yang mengancam nyawa. Pengenalan dini melalui evaluasi diagnostik cepat dan intervensi tepat waktu seperti atropin, oksim, dan terapi reperfusi pada STEMI sangat penting untuk meningkatkan angka kelangsungan hidup dan hasil pengobatan. **Simpulan**: Pengenalan dini dan revaskularisasi segera sangat penting pada pasien STEMI untuk mengurangi luas infark dan memperbaiki luaran atau *outcome*. Paparan terhadap organofosfat berkaitan dengan efek kardiovaskular, dan dokter klinisi perlu mempertimbangkan oklusi koroner akut pada pasien dengan keluhan respirasi pasca-penyemprotan insektisida, meskipun tanda tanda kolinergik klasik. **Hari Adityo Nugroho, Istan Irmansyah Irsan, Mardani Cahyono. Sindrom Koroner Akut Setelah Penyemprotan Insektisida: Hubungan yang Diduga dengan Paparan Organofosfat.**

Kata Kunci: Laporan kasus, penyemprotan insektisida, organofosfat, STEMI.

INTRODUCTION

Acute organophosphate poisoning can cause cardiac problems after a few hours of exposure, despite numerous studies showing that these compounds aren't highly hazardous. In a 2017 study conducted in Egypt, 48 adult with acute organophosphate intoxication and were included. These patients did not have a history of cardiac disease, weren't cointoxicated,

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had not received medical treatment for acute organophosphate poisoning in any medical center prior to admission without a documented medical report, did not have any pre-existing chronic diseases. They exhibited abnormal, ischemic, and arrhythmic ECGs in 81,25%, 66,7%, and 43,6% cases, respectively.[1] In a research by Kiss and Fazekas, myocardial infarction was seen in five out of 168 cases of organophosphate poisoning.[2] Even at low doses, patients may experience coronary vasoconstriction due to acetylcholine (Ach) accumulation caused by organophosphate poisoning (anticholinesterase).[3] Even though previous research and studies have produced inconsistent results, it's crucial to rule out the most critical differential diagnoses that has the best prognosis and result.

CASE

A 58 year-old male came to Dr. Iskak Tulungagung General Hospital with the chief complaint of sudden shortness of breath after spraying insecticides (suspected malathion or other organophosphate group) 1 hour before admission. He has history of diabetes mellitus, taking metformin daily, but no history of hypertension, smoking, or cardiac disease.

The patient appeared moderately ill and fully alert, blood pressure 132/70 mmHg, pulse 90 x/min, respiration 24 x/min, axillary temperature 36.7°C, peripheral O_2 saturation 100% with NRBM oxygen (non-rebreathing mask), and random blood sugar 537 mg/dL. On physical examination, pupils were isocoric (3mm) with positive light reflexes; no neurological deficit; no SLUDGEM (salivation, lacrimation, urination, defecation, gastrointestinal distress, emesis, miosis) symptoms; but rales were found on chest auscultation. A 12-lead ECG examination showed ST elevation in leads V1–V6 **(Figure 1).**

Acute heart failure, acute coronary syndrome, and organophosphate poisoning were the top three differential diagnosis. The patient was treated and observed in the resuscitation area. The patient was given oxygen with non-rebreather mask (NRBM) and crystalloid fluids, double antiplatelet loading dose (320 mg aspirin and 300 mg clopidogrel), insulin bolus 0.1 U/kgBW followed by drip 0.1 U/kgBW/hour, urinary catheter, and prepared for primary PCI (percutaneous coronary intervention).

Diagnostic coronary angiography (DCA) showed total occlusion in the middle left anterior descending (LAD) artery, with insignificant stenosis in the proximal LAD, distal left circumflex artery (LCX), and proximal-mid right coronary artery (RCA). Primary PCI at the mid-LAD was conducted using a drug-eluting stent (DES) BuMA **(Figure 2).**

The patient was treated for 4 days in the intensive cardiovascular care unit (ICVCU), and 1 day in the ward. The patient was discharged with medication (aspirin, clopidogrel, atorvastatin, ramipril, bisoprolol, and long-acting insulin).

DISCUSSION

Three categories for the differential diagnosis of dyspnea are: acute, acute on chronic, and chronic causes. Various diseases, ranging from non-urgent to life-threatening, can cause dyspnea; covering many different systems such as psychological, cardiac, metabolic, infectious, neuromuscular, traumatic, and hematologic disorders.[4] Rapid diagnostic evaluation in the emergency department (ED) is crucial to identify potentially fatal causes of dyspnea. Radiography, laboratory testing, and electrocardiography may assist in differentiating diagnoses and rule out some possible diagnoses.[5]

Diagnosis of acute organophosphate (OP) poisoning was based on the presence of the following criteria: (I) history of exposure to or contact with organophosphates within the last 24 hours; (II) characteristic clinical signs and symptoms of OP; (III) improvement in signs and symptoms after treatment with atropine and oximes; (IV) definition of responsible organophosphate agents; and (V) decreased serum cholinesterase activity.[6] Although there was a limitation in the serum cholinesterase level, we are still unable to rule out OP poisoning because the patient's main complaint was dyspnea following exposure to OP insecticides (the patient sprayed insecticides, suspected malathion,

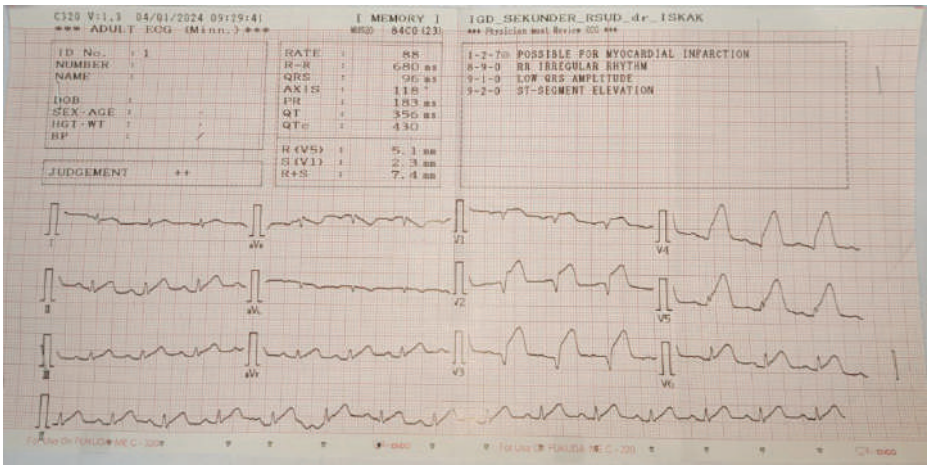

Figure 1. Patient ECG, sinus rhythm, 88 bpm, ST elevation in V1–V6.

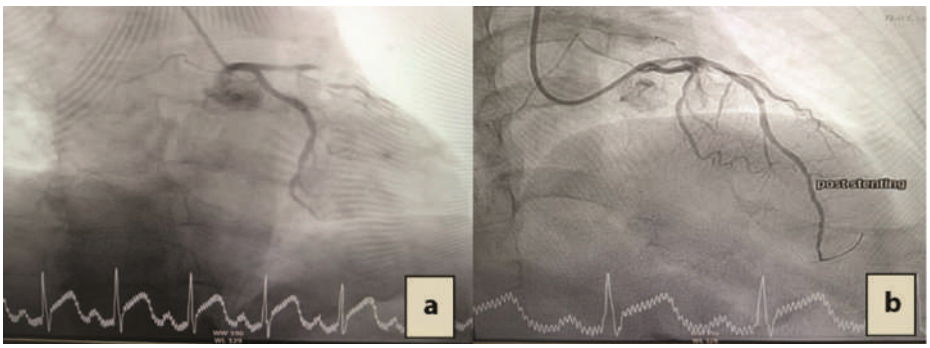

Figure 2. (a) Total occlusion in mid LAD, 30% stenosis in proximal LAD, 30% stenosis in LCX and 30%-40% stenosis in RCA. (b) Primary PCI at mid LAD with thrombolysis in myocardial infarction (TIMI) flow 3.


for about 30 minutes prior to symptoms appearing, despite no history of chest pain or shortness of breath provoked by activity). The patient's primary complaint was dyspnea after exposure to organophosphate (OP) insecticides (patient sprayed insecticides for approximately 30 minutes before symptoms developed); there was no history of either chest pain or dyspnea on activity. The goal of emergency management should be to relieve symptoms and avoid complications as well as to perform all necessary diagnostic testing to rule out any other options.

Clinical features in acute OP poisoning have traditionally been approached from three different aspects: (1) the effects of OP on muscarinic, nicotinic, and central nervous system (CNS) receptors, which can produce a variety of symptoms and signs; (2) the timing of the onset of symptoms, such as SLUDGEM (salivation, lacrimation, urination, defecation, gastrointestinal distress, emesis, and miosis); (3) an organ-specific approach focused on the neurologic, respiratory, metabolic, or cardiovascular effects of OP.[7]

OP insecticides and nerve agents are designed to inhibit acetylcholinesterase (AChE), crucial for regulating the neurotransmitter acetylcholine. While some OP compounds like malathion and parathion are poor direct AChE inhibitors, they generate oxon metabolites upon metabolism, which strongly bind to cholinesterase enzymes.[8] Reduced AChE levels lead to acetylcholine accumulation, causing cholinergic syndrome characterized by SLUDGEM symptoms. Severe cases may result in extended neuromuscular depolarization, muscle twitches, tachycardia, and respiratory dysfunction, potentially leading to paralysis or death.[9] Ventricular dysrhythmias, occurring days after admission, may result from direct myocardial damage, including interstitial inflammation, myocarditis, or patchy pericarditis. Prolongation of QT interval and occurrences of *Torsades de pointes* are reported. Cardiac manifestations of organophosphorus insecticide poisoning include bradycardia, tachycardia, ventricular arrhythmias (including *Torsades de pointes* and ventricular fibrillation), asystole, and various ECG changes such as ST-segment changes, peaked T waves, AV block, and QT interval prolongation.[10] Bradycardia (more frequent than tachycardia) was observed in 45.8% patients in the study of El-Sheikh, *et al.*, while tachycardia occurred in 25% in the same study,[1] consistent with study of Akdur, *et al.*, 11.1 and 5.6%, respectively.[11] Hypotension was noted in 29.2% cases, potentially due to intravascular volume depletion and negative cardiac effects of Ops.[1] Ischemic findings on ECG, such as inverted T-waves and depressed ST segments, were more common than arrhythmic findings. Early recognition and adequate treatment of these complications are crucial for preventing adverse outcomes.[1] Diesel exhaust (DE) is associated with adverse cardiovascular effects. Specifically, DE inhalation has been shown to promote myocardial ischemia, inhibit endogenous fibrinolytic capacity, impair the regulation of vascular tone, increase thrombus formation, activate platelets, and induce vasoconstriction.[12]

Organophosphate exposure can cause cardiovascular symptoms ranging from ECG abnormalities or lethal arrhythmias to cardiorespiratory arrest. Within a few hours of exposure, OP poisoning has been linked to serious heart problems, such as myocardial infarction. There are 3 phases of cardiotoxicity after OP poisonings, the first phase was a brief period of increased sympathetic tone, the second one was a prolonged period of parasympathetic activity and the third phase in which QT prolongation is followed by ventricular tachycardia, polymorphous ventricular tachycardia *(torsade de pointes)*, and then ventricular fibrillation. ST elevation was suggestive of transmural myocardial ischemia and T-wave inversion was suggestive of subendocardial myocardial ischemia.[1] Following a suspected OP exposure, a thorough history taking, physical examinations, serial ECGs, and cardiac troponin measurements can assist in ruling out the cause. Coronary angiography can be utilized as a diagnostic and therapeutic method. to rule out acute coronary thrombosis.

STEMI (ST-elevation myocardial infarction) is a severe form of coronary artery disease, requires prompt reperfusion to salvage myocardial tissue and reduce infarct size. Rapid diagnosis within 10 minutes of initial medical contact is crucial for initiating appropriate treatment, emphasizing early transfer to facilities capable of performing primary percutaneous coronary intervention (PCI).[13] New treatment strategies targeting inflammation aim to further reduce the risk of recurrent cardiovascular events.[14] Post-STEMI in-hospital mortality has significantly decreased to under 10% due to advancements in care and widespread adoption of early reperfusion strategies.[15] While primary PCI is preferred over fibrinolysis, the choice depends on factors such as treatment initiation time and patient presentation, with efforts to reduce "door to balloon time" showing significant improvements in contemporary practice.[13] Reducing mortality and improving patient outcomes in acute STEMI is mostly achieved by early reperfusion with PCI. Early intervention reduces cardiac damage and increases survival rates considerably (preferably within 90 minutes of initial medical contact). Reperfusion delays are linked to increased death rates, especially when symptoms start more than an hour before treatment begins. Rapid treatment plans and prehospital 12-lead ECG are two examples of strategies created to improve early detection and prompt action for STEMI patients. Reducing mortality in patients with ACS requires establishing an all-encompassing healthcare system centered on establishing early reperfusion in prehospital and emergency department settings.[16,17] The physical component of quality of life (QoL) significantly improved in study participants who had survived acute coronary syndrome (ACS) between one and six months after percutaneous coronary intervention (PCI), indicating that PCI-assisted revascularization improves patients' physical health.[18]

CONCLUSION

Organophosphate poisoning may result in cardiovascular symptoms. The main goals of treatment should be symptom relief, further investigation to look for evidence of organophosphate exposure, and rule out other possible differential diagnoses. A high suspicion of acute coronary occlusion should be referred to the cath lab as quickly as possible.

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