

IS – 31

Cardiotoxicity of organophosphates and metal phosphides

Ashish Bhalla

Department of Internal medicine, Post Graduate Institute of Medical Education and Research, Chandigarh India

Organophosphate poisoning (OPP) with suicidal intent is a major cause for concern in developing countries¹. These poisonings result in cholinergic and nicotinic signs and direct toxic effects on several organs. Abnormalities on electrocardiography (ECG) may occur in patients with acute OPP and vary from nonspecific to fatal ventricular arrhythmias. Patients who die following an apparent clinical recovery may have myocardial involvement, including myocardial necrosis and toxic myocarditis. The exact time interval from exposure to cardiac complications is unpredictable and it is not possible to predict which patients will develop cardiac abnormalities.

The cardiovascular effects of OPs reflect the net result of excitatory and inhibitory actions of accumulated acetylcholine (ACh) at ganglionic, medullary, vasomotor, and cardiac centers. These effects are further compounded by hypoxia, local release of catecholamines, and disturbances of ion transport. The three phases of cardiotoxicity are described as an initial intense sympathetic phase, a second prolonged parasympathetic phase usually accompanied by hypoxemia, which often manifesting as ST-T changes, A-V conduction disturbances which can degenerate to VF. The third phase of QT prolongation followed by TdP and VF. Various ECG changes have been described and sinus tachycardia is the commonest abnormality. This occurs as a result of intense sympathetic stimulation, nicotinic stimulation of ganglionic sites by excess ACh, atropine administration, and dehydration. Ventricular arrhythmias are also frequently observed. Symptomatic and supportive management is indicated. Phosphine and metal (Aluminium) phosphide are primarily cytotoxic causing mitochondrial respiratory chain inhibition in cells. Almost all patients develop nausea, vomiting, retrosternal burning, and epigastric discomfort after ingestion. Severe and refractory hypotension, tachypnoea and shock invariably develops within 30 minutes to 2 hours of exposure. Prolonged hypotension and tissue hypoxia results in diffuse cellular damage in various organs manifesting as multi-organ dysfunction.

Sweating and tachycardia are commonly observed due to sympathetic storm. Cardiac dysrhythmias are a hallmark of metal phosphide poisoning, and are typically fatal. Several electrocardiographic abnormalities have been reported. Commonest arrhythmias are atrial flutter, atrial fibrillation, ventricular tachycardia and ventricular fibrillation. Echocardiography reveals hypo or akinesia of the left ventricle (LV), LV dilatation and systolic dysfunction.

Magnesium sulfate and Amiodarone are indicated for prevention as well as management of arrhythmia, supportive management is usually helpful.