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CARDIOTOXICITY OF ACUTE ORGANOPHOSPHATE POISONING

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Abstract

To study the cardiotoxicity of acute organophosphate (OP), poisoning, this study was designed to evaluate 46patients who presented to the Benha poisoning control unit over a 14 months period from the 1st of March, 2007 to 30 April, 2008, with acute OP poisoning and discus their associated cardiac complication and electrocadiographical (ECG) abnormalities. The serum level of cholinesterase (AChE) was significantly lower than the normal value. At the same time serum creatinine kinase (CK-MB) and cardiac trophonin I (CTnI) levels were significantly elevated at the time of admition indicating the presence some degree of cardiac injury. ECG changes confirmed the presence of cardiac injury. These was sinus tachycardia (34.78%) which the most common ECG abnormality, sinus bradycardia occurred in 9 patients (19.56%), hypertension developed in 6 patients (14.04%) and hypotension in 6 patients (13.04%). OP induced impaired cardiac conductivity in form of prolongation of the QTc interval (32.61%) and prolonged PR interval (8.70%) and increased cardiac excitability in the form of extracystole (6.52%), ventricular tachycardia (2.17%) and atrial fibrillation (4.35%) and also induced myocardial cell injury manifested by elevated ST segment (15.22%). Cardiac trophonin I (CTnI) level is indicated for diagnosis of cardiac injury due to OP poising when the patient is seen 3 days after intoxication. Most of cardiac complications associated with organophosphate occur during the first few hours after exposure. Sympathatic, parasymathatic over activity hypoxemia, acidosis and electrolyte derangements and a direct toxic effect of the OP on the myocardium are major predisposing factors for the development of these complications. The cardiac complications and ECG abnormalities all returned to normal before the patients were discharged. Initial com-

plete ECG is recommended and should be obtained immediately in the Poison Control Unit in patients with acute OP or poisoning.