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Organophosphorus Poisoning Presenting as Pulmonary Thromboembolism

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Abstract

In situ pulmonary artery thrombosis (PAT) in adults is a potentially life-threatening disorder rarely recognized ante mortem. The literature pertaining to PAT is limited to isolated clinical case reports or small descriptive pathological series stating a close association with concurrent morbidity such as thrombophilia, pneumonia or pulmonary arterial flow disturbance.

A rare case of extensive in situ central pulmonary artery thrombosis in organophosphorous poisoning is presented. We report a case of 37-year-old woman with history of consumption of organophosphorous compound (mahaphos). She presented with vomiting, drowsiness, poor response to oral commands, fasciculations and shallow breathing. She was admitted and treated in ICU for 20 days wherein her general condition improved. She developed respiratory distress and collapsed all of a sudden. On autopsy, she had a large immobile wall-adherent thrombus located in the pulmonary trunk extending into the right pulmonary artery. Left pulmonary artery showed emboli and right coronary artery showed a thrombus attached to the arterial wall. Histopathology revealed multiple areas of infarction in lung and wide spread toxic injury to hepatocytes, spleen and kidney.

We conclude that op compound poisoning caused wide spread endothelial damage resulting in pulmonary and coronary thrombosis leading to cardiac arrest.

Key Words

Organophosphorus (OP), pulmonary artery, coronary artery thrombosis.

Introduction

Organophosphorus (OP) poisoning is the commonest mode of suicidal deaths in rural part of India. Easy availability and accessibility to OP compounds is the reason for the increased incidence of its acute poisoning in these parts with agricultural background.

In our intensive care unit (ICU) which is a multidisciplinary unit, OP compound poisoning forms a significant number of total medical admissions. Last year, of total 462 medical cases there have been 116 OP admissions with Case fatality rate of 16%. We report a case of sudden cardiac arrest in a patient recovering from OP compound poisoning. The cause of death was due to known but less discussed manifestation of OP compound toxicity.

Case Report

A 37 year old lady was brought to our casualty with h/o consumption of OP compound (mahaphos) presenting with flaccidity, drowsiness, poor response to verbal commands, diaphoresis, generalised fasciculations, ptosis, tachypnea and pinpoint pupils. Respiration was shallow with inadequate respiratory attempts and oxygen saturation (SPO2) was 88% with 10 litres of oxygen with Hudson mask. Her heart rate was 120 beats /min and blood pressure was 150/110 mm Hg. Systemic examination revealed no other significant abnormality. After decontamination, gastric lavage through Ryles tube and urinary catheterization, atropine 5 mg was given iv every 5 minutes till atropinisation and atropine infusion later on. Pralidoxime (PAM) was started at 0.5g/hr. After explaining to the patient's attendants about the need for ventilatory support, patient was shifted to ICU for further management. Patient was intubated with 7.5 mm internal diameter portex tube and put on Pressure cycled synchronised intermittent mandatory ventilation (P-SIMV) mode of ventilation. Vitals and input output chart was monitored. Graded compression stocking was used for deep vein thrombosis (DVT) prophylaxis. Investigations revealed no significant abnormality other than decreased pseudocholinesterase levels (3813 U/l). Chest and passive limb physiotherapy was started. She was tracheostomised on 3rd day. Weaning started on 5th day. Atropine infusion tapered and stopped. She was put on T-piece on 12th day. Though patient had good muscle power and respiratory efforts, she desaturated on removal of supplemental oxygen and had recurrent episodes of dyspnoea. So she was observed in ICU for 8 more days during which she was ambulant. She was due to be shifted out of ICU when she suddenly developed restlessness and became tachypneic and collapsed. Her heart rate was 120beats/ min, pulse was feeble, BP not recordable and later she developed bradycardia and cardiac arrest. Cardiopulmonary resuscitation (CPR) was started. After 30 minutes of resuscitation she could not be revived. Clinically, cause of death was thought to be due to DVT and thromboembolism as other causes of sudden death like ventricular arrhythmias, myocardial infarction and hypoxia due to tracheostomy tube block were ruled out. On autopsy, she had a large immobile wall-adherent thrombus located in the pulmonary trunk extending into the right pulmonary artery. Left pulmonary artery showed emboli and right coronary artery showed a thrombus attached to the arterial wall(fig 1). Histopathology revealed multiple areas of infarction in lung(fig 2) and wide spread toxic injury to hepatocytes(fig 3), spleen and kidney(fig 4).

Fig. 1: Gross photograph showing pulmonary artery with wall adherent thrombus

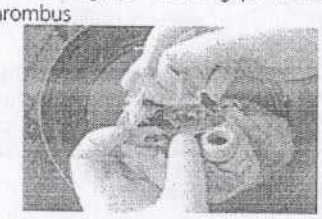


Fig. 2: Microphotograph showing pulmonary infarction (H&E,X100)

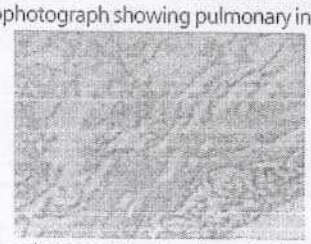


Fig. 3: Microphotograph showing feathery degeneration of hepatocytes (H&E,X100)

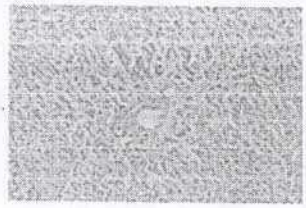
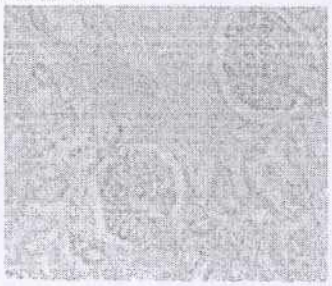


Fig 4: Microphotograph showing acute tubular necrosis with macrophages (H&E, x40).



Discussion

Signs and symptoms of organophosphate poisoning can be divided into 3 broad categories, including (1) muscarinic effects, (2) nicotinic effects, and (3) CNS effects.

The known but less discussed manifestation includes aberration of clotting mechanism. It manifests as a biphasic reaction consisting of coagulation followed by prolongation of clotting time. Increased coagulation is associated with increased prothrombin activity and consumption secondary to increased factor VII[1]. Jastrzebski et al[2] presented a case of suicidal poisoning with organophosphate pesticide, associated by acute activation of blood coagulation where heparin treatment efficiently inhibited this activation whereas Zieman et al[3] contradicted and reported that OP compounds caused thrombocytopenia and decreased fibrinogen, plasminogen and antithrombin III levels necessary for coagulation thereby causing hypocoagulation. George A Petrioanu[4] reported that in OP intoxication, hypercoagulability was seen in the sympathomimetic phase (in our case), due to massive release of catecholamines from the adrenals whereas hypocoagulability in the vagal phase, shown by the PTT-prolongation due to influence on platelet function or inhibition of clotting factors.

In our case, there was a coronary thrombus and a primary pulmonary artery thrombus (PPAT) which is rare. In fact, in situ pulmonary artery thrombosis in adults is considered potentially life-threatening and rarely recognized antemortem. The literature pertaining to PPAT is limited to isolated clinical case reports or small descriptive pathological series stating a close association with concurrent morbidity such as thrombophilia, pneumonia or pulmonary arterial flow disturbance[5,6].

Case reports suggest association of PPAT with pulmonary hypertension either primary[7] or secondary to pulmonary artery hemangiosarcoma[8], and Eisenmenger's syndrome[9]. Steroid responsive nephrotic syndrome[10], severe acute respiratory syndrome[11], tetralogy of fallot[12], thrombangitis obliterans[13], antiphosholipid syndrome[14] and pulmonary arteritis[15] associated PPAT have also been reported. OP poisoning with PPAT has not been reported till date. The course of acute massive PAT is serious and may result in sudden death. PAT may also cause subacute cor pulmonale or have a more chronic fatal course in cases with missed diagnosis. Unfortunately due to its presumably inconstant and non-specific clinical symptoms, ante mortem diagnosis is difficult and the true incidence of this under diagnosed disorder is unknown.

In our case, initial diagnosis of sudden death was thought to be due to DVT and thromboembolism as the patient was clinically stable throughout her stay in ICU. But, autopsy revealed PPAT and coronary thrombosis as cause of death.

Conclusion

As OP compounds cause wide spread endothelial toxic injury and hypercoaguable or hypocoaguable states which can be potentially life threatening, we conclude that monitoring the hematologic parameters and coagulation profile should be considered.

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