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CASE REPORT
ORGANOPHOSPHORUS POISONING LEADING TO RESPIRATORY FAILURE

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SUMMARY

Organic insecticide poisoning (suicidal, accidental or occupational exposure) is quite prevalent in the rural areas of Pakistan. Presentation is variable, ranging from GIT symptoms to severe neurological manifestations of paralysis and cardiovascular instability. Such patients need careful thorough assessment, vigilant monitoring and aggressive supportive management. We present a case of such poisoning who went into respiratory failure.

KEY WORDS: organophosphorus, manifestations, paralysis, neurological, anticholinergic, respiratory, oximes.

INTRODUCTION

Organophosphorus compounds constitute the main class of organic insecticides used globally for pest control and as such contribute to major illness (more than 80%) in majority of insecticide poisoning cases (1). In the rural areas such agents are frequently misused for committing suicide, homicide or their accidental over exposure leads to acute severe illness involving GIT, CNS, CVS & respiratory systems with high morbidity and mortality. Thorough clinical assessment and early therapeutic interventions are essential vital steps to save the life.

Supportive care in ICU includes decontamination, vigilant monitoring, anticholinergics and care of the end organs targeted by the poison.

CASE REPORT

A sixteen-year-old boy was brought to our hospital with a history of severe abdominal pain, vomiting, diarrhoea and muscle weakness. According to the parents, he ingested a pesticide bottle after a domestic quarrel. On admission he was drowsy responding to deep pain only, and retching with frothy salivation. Vital signs were; pulse *56* beats/min and of low volume, blood pressure *65/30* mmHg, respiratory rate 8/min. Extremities were cold and cyanosed. Pulse oxygen saturation was 80%.

Systemic examination revealed bilateral air entry in the lungs with signs of pulmonary congestion, the heart sounds were normal. Twitching of face and limb muscles was noted & both the pupils were constricted. Incontinence of urine and faeces was noted with exaggerated bowel sounds.

After clearing of the upper airway, oxygen face mask was applied, IV cannulation was done & blood samples were sent for CBC, sugar, electrolytes, urea, creatinine, LFT's & coagulation status. He was shifted to ICU, where airway was secured by endotracheal intubation and mechanical ventilatory support (IPPV) was started keeping in view the unstable haemodynamic status and impending respiratory failure. Nasogastric tube was passed and gastric residues were aspirated followed by gastric lavage with activated charcoal 1g/kg body weight. Atropine I.5 mg as I/V bolus and fluid resuscitation with dextrose-saline started. Atropine 1 mg was repeated every *15-20* min with next 3 doses. ECG revealed sinus bradycardia, ST elevation and prolonged QTc interval. Monitoring included 5-lead ECG, SpO_2, NIBP, CVP, temperature & urine output / hour. Since haemodynamic response to IV fluids was not adequate and only heart rate improved with atropine, dopamine infusion (4- 15 microgram/kg body wt/min) was started. Atropine was continued as 1 mg every 20-30 min till adequate clinical atropinization was achieved. Over the next 8 hours his haemodynamics and respiratory parameters started improving, tracheobronchial secretions were reduced and oxygenation improved. Chest X-ray was suggestive of pulmonary

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congestion and an ill-defined opacity in the right lower zone was seen.

Ventilatory management consisted of Vt 10ml/kg body weight; respiratory rate of 12/min, FiO_2 0.8, PEEP 4-6cm of H_2O and peak flow rate of 30 l/min. Close clinical monitoring was applied throughout (as the facilities for estimation of ABG's and $EtCO_2$ did not exist). On second day he developed generalized tonic-clonic convulsions, which were controlled with diazepam I/V and manitol. Nutritional support, nebulization, antibiotic therapy & nursing care with special attention to pressure points were taken care of. He was ventilated for four days. When the haemodynamic and respiratory parameters were within acceptable limits, and he was fully conscious and well oriented with good muscle tone and power, successful weaning from the ventilatory support was achieved through SIMV + pressure support mode. He was discharged home after 9 days.

DISCUSSION

Organophosphorus compounds (OP) were first synthesized in 1854 and have long been used as pesticides and as nerve gas in chemical warfare. Tetraethyl pyrophosphate (ThPP) is the most toxic of such agents.

A WHO report reveals that 3 million cases of serious pesticide poisoning occur all over the world annually, the majority by OP compounds with a higher number in the developing countries[2]. In Pakistan 16% of total ICU admissions are of such poisoning cases[3], whereas in India 12% of total ICU admissions are because of OP poisoning[4]. These are also common suicidal agents in Sirilanka[5]. 'Parathion' is most commonly used in such fatalities[6]. These are highly lipid soluble compounds and bind to acetyl cholinesterase (Ach esterase) irreversibly, thus acting as "neurotoxins" with muscurarinic, nicotinic or central neuronal stimulatory effects[7]. Clinical symptomatology varies widely but most of the cases have onset of symptoms within 24hrs resembling acute gastroenteritis so one has to be careful in establishing the real diagnosis and associated physical findings must be sought.In our patient physical signs and symptoms started appearing within 6 hrs of ingestion. Neurological picture also varies widely with diplopia, tremors, rigidity and muscular weakness leading to respiratory paralysis necessitating ventilatory support. This may be delayed for 24-96 hrs after the acute cholinergic crisis[8-9]. This is called intermediate syndrome (type II paralysis)[10]. One of the earliest finding is inability of the patient to lift the neck from the pillow (a useful clinical test to determine the likelihood of paralysis). Later on polyneuritis may occur with cranial nerve involvement.

An analysis of 46 patients in Jordan pointed towards various cardiovascular disturbances with prolonged PR and QT interval (same as in our patient), tachycardia or bradycardia, hypertension or hypotension and ST elevation resembling myocardial infarction. History and clinical features usually help in diagnosis. Altered neurological state, miosis, increased salivation and sweating with typical insecticide odor are important clues. One must not wait for all the clinical signs (muscarinic / nicotinic) to appear and therapeutic measures must be initiated immediately as early as the suspicion arises under such circumstances. Another important finding is the markedly decreasedlevels of both the true and pseudocholinesterase levels in OP poisoning. Though true cholinesterase levels correlate well with the severity of illness at presentation but pseudocholinesterase levels do not[11]. While looking for the reduced level of above-mentioned esterase it is important to rule out CCF, parenchymal liver disease and malnutrition. A 25% or more decrease in the level of true cholinesterase is taken as diagnostic of OP poisoning[12]. It is important that atropine toxicity must be avoided as it can lead to nervous irritability, hyperpyrexia and ventricular dysrrhythmias (VF) especially in a hypoxic heart. In children the bolus dose of atropine is 0.05mg/kg IV and maintenance dose is 0.02-0.05mg/kg. Adequate atropinization is depicted by heart rate of just over 100 / min, mid-dilated pupils and bowel sounds just present. Atropine infusion reduces the chances of toxicity and overall mortality[13]. Theophylline is contraindicated in OP poisoning[14]. Glycopyrrolate is equally effective in OP poisoning with less CNS side effects[15]. Progress of the disease is assessed by eye

rate, tidal volume or vital capacity. Weakness of neck muscles and serial estimations of ABG's, acid-base status and serum electrolytes are monitored (facilities for monitoring of ABG's, $EtCO_2$ and acid base status did not exist in our setup). Recently much stress has been laid upon the role of Oximes (pralidoxime). These compounds reactivate acetylcholine esterase by binding to organophosphorus molecule[16]. In one of the studies it has been concluded that high dose oxime therapy is associated with increased mortality whereas low dose is more effective[17]. Pralidoxime is not available in this country.

CONCLUSION

Organophosphorus poisoning is a great hazard to the health of society as fatal poisoning can occur quite easily with rapid onset, may it be suicidal, homicidal or accidental. Careful assessment, early therapeutic intervention, vigilant monitoring and overall good supportive nursing care can save many precious lives. Adequate atropinisation and ventilatory support are the hallmarks of the active intervention.

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