CASE REPORT

Organophosphate Insecticide Poisoning

Maj Azhar Niwaz, MCPS

Maj Manzoor Ahmed Faridi, MCPS

INTRODUCTION

CMH Pano Aqil is situated about 35 KM north of twin cities of Rohri and Sukkur. It is a cotton growing area and insecticides are frequently used to save crops. So in the season of cotton we off & on receive cases of organophosphorus insecticides poisoning. It may occur following absorption via the gastrointestinal system, respiratory tract or skin. Deliberate or accidental ingestion is a common mode of poisoning. Insecticide poisoning with suicidal intent is common in this part of Pakistan because of its easy availability in every house.

Organophosphorus (op) compounds are possibly the most widely used insecticides in the world, some of which are as follows:-

- Diazinon
- Dichlorvos - Dimethoate - Fenitrothion
- Leptophos - Malathion - Parathion
- Trichlorfon

These agents are utilized to combat a large variety of pests. The major effect of these agents is inhibition of acetylcholinesterase. The organophosphate, forms very stable irreversible bonds to the enzyme. The signs and symptoms that characterize acute intoxication are due to inhibition of this enzyme, resulting in the accumulation of acetylcholine. Some of these agents also possess direct cholinergic activity.

In addition to and independent of inhibition of acetylcholine esterase, some of these agents are capable of phosphorylating another enzyme present in neural tissue, the so called "neurotoxic esterase" or neuropathy target esterase (NTE) this results in development of a delayed neurotoxicity characterized by Polyneuropathy, associated with paralysis and axonal degeneration.

CASE REPORT

A 39 years old married lady with 4 small children was residing with the joint family in a nearby village of Pano Aqil. On 25 August 98, she had a quarrel with another family member over a small dispute, she went into room and drank insecticide "KARATE" in an attempt to suicide, which was noticed when her brother entered in that room. He found her lying semi conscious on bed and an empty bottle of insecticide was lying on floor. There was also vomitus near the bed. Vehicle was arranged and she was evacuated to the MI Room of CMH Pano Aqil, where she was attended by classified medical specialist.

On arrival she was comatose grade II Breathing was gasping, there were excessive salivary secretions.

Pulse was 40/min regular, thin volume
B.P : 80/40 mmHg
Temp 98°F
Pallor +ve Pupils - pin point

RESPIRATORY SYSTEM

- Wheezing +ve bilaterally
- Labored breathing
- Bilateral pulmonary rales were audible suggestive of increased bronchial secretions.

C V S :- bradycardia HR 40/min
Hypotension B.P 80/40 mmHg
C N S :- Unconscious
No spontaneous movement of any part of the body.
Pupils-pin point.

On the basis of history and clinical examination the diagnosis of organophosphorus (op) poisoning was made. She ultimately developed ventilatory failure due to the involvement of respiratory muscles.

Patient was admitted in ITC, intubated without any resistance and placed on ventilatory support with the following settings:
Mode CMV
FiO₂ - 0.50
RR - 12/min

As arterial blood gases facility is not available at CMH Pano Aqil, therefore patient was monitored with pulse oximeter, which depicted 100% saturation (SPO₂). Wide bore NG tube was passed and stomach wash performed. 50 ml of gastric aspirate collected for toxicological analysis. Inj atropine 2mg I/V state and than 1 mg intravenously after every hour was administered. I/V anti-biotic started and infusion dopamine 8-10 micro gram/kg was started, urinary catheter passed and urine output recorded.

Injection Pralidoxime was prescribed but couldn't be procured even from Sukkur.

After 6-7 hours of treatment, patients level of consciousness improved. She started opening eyes to stimuli but respiratory effort was still very poor.

The next day patient showed improvement. She was haemodynamically stable and infusion dopamine was tapered off. Ventilatory mode changed to SIMV and RR kept at 8/min and FiO₂ reduced to 0.30.

Interval of Inj atropine 1.0 mg was increased to 4 hourly.

On the 4th day of admission she was successfully weaned off the ventilator. Haemodynamically stable, fully conscious and was oriented in time and space.

On 29-8-98, she was shifted to family ward, where after staying for another three days, she was discharged from the hospital with full recovery and without any residual damage to any organ.

**DISCUSSION**

During the past four decades, some 15000 individual compounds and more than 35000 different formulations have come into use as pesticides. Organophosphorus compounds are possibly the insecticides most widely used in the world for control of insects affecting agriculture.

These are potent inhibitors of the enzyme acetylcholinesterase. Organophosphates are absorbed through the skin, lungs and gastrointestinal tract, are distributed widely in the tissues, and are slowly eliminated by hepatic metabolism. These compounds produce muscarinic, nicotinic, and CNS effects. Manifestations occur within 30 min to 2 hr following exposure. Muscarinic effects, include nausea, vomiting, abdominal cramps, salivation, lacrimation, miosis, diaphoresis, bronchospasm, and bradycardia. Nicotinic signs include twitching, fasciculations, weakness and skeletal muscles paralysis (apnea). CNS effects include anxiety, restlessness, tremors, convulsions and coma. Organophosphate over dosage may be followed by delayed peripheral neuropathy involving distal muscles of the extremities. Clinical diagnosis is relatively easy and is based on the characteristic symptoms and signs and history of exposure to organophosphorus agent. The response to atropine may also be useful aid to diagnosis. All patients should be managed as emergencies in the hospital, contaminated clothing should be removed and skin should be washed with soap and water. Gastric lavage is most effective within 30 minutes of ingestion.

Activated charcoal may be administered to reduce further absorption of the organophosphorous agent from the stomach. Atropine, muscarinic receptor antagonist should be administered for muscarinic effects. Recommended dose is 2 to 4 mg I/V repeated at interval of 5 to 10 minutes initially and continued until signs of atropinization appear.

Pralidoxime, an oxime that reactivates cholinesterases, is indicated for nicotinic symptoms in organophosphate poisoning. The dose is 1 to 2 gm intravenously over 5 to 10 minutes. The dose may be repeated every 4 to 6 hours, until nicotinic signs resolve.

Diazepam 5 to 10 mg I/V is used for controlling the seizures.

Weakness of the muscles of breathing may require mechanical support of ventilation.

The usual mode of death is related to cardiac complications and ventilatory failure. Early supportive therapy and mechanical ventilation by whatsoever means available, may save many precious lives. in this regard it may be stressed that every medical officer must be adequately trained in the skills of intubation and Ambu ventilation.

Preventive measures should be considered during the use of insecticides in agriculture.

At present, the goal of safe and effective use of insecticides is achieved best by an agro medical approach to pesticide management. Integrated, inter disciplinary applications of skills and knowledge of
agriculture, applied chemistry and medicine. May suggest means of prevention.

REFERENCES

Quick Action Plans
Pulmonary Aspiration in the Anaesthetized Patient

IDENTIFICATION
1. Reduction in SpO₂
2. Coughing
3. Tachypnoea
4. Bronchospasm
5. Possible hypotension and /or fever (usually late sings)
6. Tracheal aspiration is acid on pH setting.

ACTION
1. Place patient in head-down position and on side.
2. Laryngoscopy and suction (consider bronchoscopy if solid matter inhaled).
3. Is the patient already paralyzed?

A. PARALYZED PATIENT
1. Proceed with intubation (cuffed endotracheal tube).
2. Suction via endotracheal in head down position. (Test aspirate with pH paper. If acidic, this confirms gastric aspiration.
3. IPPV with at least 50% O₂, plus PEEP if required.
4. If bronchospasm develops-treat with salbutamol 250 mcg IV or aminophylline 250mg IV.
5. As necessary:
   - blood gas analysis.
   - colloid for hypotension.
6. Proceed with surgery if essential and patients condition permits.

7. Pass nasogastric tube and aspirate stomach.

B. PATIENT NOT PARALYZED
1. Allow to wake up.
2. High inspired oxygen concentration by mask.
3. If bronchospasm develops, treat with nebulised salbutamol (2.5 mg). If this is inadequate use salbutamol 250 mcg IV or aminophylline 250mg IV.
4. As necessary;
   - blood gas analysis.
   - colloid for hypotension.
5. If surgery is essential, consider regional technique.
6. If general anaesthesia indicated;
   a) Pass nasogastric tube and aspirate stomach. Give 30 ml sodium citrate
   b) Pre-oxygenate. Re-induce and intubate using cricoïd pressure.

FURTHER MANAGEMENT
If clinical evidence of aspiration the patient will require observation in I.C.U. (or probably H.D.U) and may require possibly elective IPPV. All require post-op O₂. It is generally thought that antibiotic treatment should be directed at proven infection, rather than given prophylactically. Steroids are no longer recommended.