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A Case Report on Severe Dimethoate Poisoning : Do We Know Everything?

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ABSTRACT *Dimethioate is widely used organophosphate insecticide used to kill insects on contacts. It causes severe and profound hypotension which is refractory to any supportive therapy. We selected this case report to highlight potency and lethal effects of this compound.*

A 40 year old male patient admitted with alleged history ingestion of DEVIGON 30 substances,approx 40-50 ml, 2-3 hours before hospitalization After ingestion, he had 3-4 bouts of foul smelling, vomiting, incontinence of stool and urine. He lost consciousness after 15 minutes.

On examination, he was deeply comatose with gasping breathing, heart rate-87per min, SpO_2-67%on room air, bilateral crepitations, pupils bilateral normal reacting to light. No response to deep painful stimuli noted. His muscle tone was increased and planters were flexor. Fasciculations all over body present.

Smell of OP compound from breath present

Patient was immediately intubated and put on ventilator with volume control mode with FiO_2 50%.

Ryles tube aspiration was done and Charcoal 40gms was given followed by duphalacsyryp 30ml and repeated 6 hourly for four doses. Duphalac enema was given 8 hourly for whole bowel irrigation. Skin decontamination done by changing cloths and sponge bath was given.

Inj PAM 2 gms stat followed by 8mg/kg/hr started.

inj atropine 2amp bolus given, target heart rate 100 achieved , and chest was clear ,then atropine infusion was started at 3 ml per hour.

Antibiotics and other supportive care given.

Serum and RBC choline esterase levels sent for analysis at our lab & NIOH.

Baseline investigations showing, Hb-12.8, total count-17900, DC-60/35/4/0, pletlets-2,84.000 ,Peripheral smear showing normocytic RBC'S

RBS-206MG%, S. CREATININE-1.01MG%, SGPT-21, ALT-48U/L, S.BILLI-0.8 MG%, S. POTASSIUM-4.O MEQ/L, S. SODIUM-135 MEQ/L

PLASMA CHOLINESTERASE LEVELS-2598U/L (2900-5800),
(RBC Choline esterase levels could not be done because of technical problems)

On second day, patient developed hypotension, systolic blood pressure was 80 mm Hg, was put on nor adrenaline infusion. He remained comatose. urine output maintained. He was on ventilatory support.

BLOOD UREA 18 MG%, S. CREAT- 2.17 MG%, POTASSIUM 4.13, SODIUM 142
We continued aggressive management in form of noradrenaline infusion which was gradually titrated to maximum dose along with fluid at rate of 150 ml per hour to ensure urine output of 60 to 70 ml per hour. PAM infusion, atropine infusion and other supportive therapy. Urine output was 3 litres.

On third day, patient again had hypotension; vasopressin infusion followed adrenaline infusion was added to the management.

ABG-PH-7.0048, PO_2-122.7, PCO_2-40.3, HCO_3-10.9, BE—19, O_2sat-95.5 %
$NaHCO_3$ infusion was given 50 ml bolus followed by 20 ml per hour infusion.Vasopressor infusions titrated to maximum doses but there was no effect on blood pressure.

Patient was deeply comatose and was not showing any motor response. His pupils were bilateral dilated and fixed but still spontaneous triggering of respiration was there. FINALLY PATIENT SUFFRED A CARDIAC ARREST AND DIED.

DISCUSSION:
Dimethoate is used against a broad range of insects such as thrips, aphids, mites, and whiteflies(5), and on a number of crops including citrus, cotton, fruit, olives, potatoes, tea, tobacco and vegetables(6). It is also permitted for the control of the flies in livestock accommodation(7), home gardens and food storage(8) .

Production and use
Dimethoate is used in a large number of products. In the US, it has 166 approved labels(10). In 1998, there were 16,250 metric tonnes of sales globally, with a value of $US 180 million (11).

Acute toxicity
Dimethoate is moderately toxic (World Health Organisation class II) by ingestion, inhalation and dermal absorption (12). Most oral LD50s (dose at which half the sample is dead) in rats range from 150-400 mg/kg body weight(13). For mice, rabbits and guinea pigs, the LD50s are 160,

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300 and 350 mg/kg respectively(14). As with all OPs, dimethoate is rapidly absorbed through the skin, and easily absorbed through the lungs(15).

The population as a whole is not generally subject to exposure to dimethoate from air, water or food(16); however occupational exposure may occur during manufacture, formulation and use. This mainly occurs through inhalation and dermal absorption, although occupational exposure can occur by accident or as a result of incorrect handling (17).

Where humans are exposed to dimethoate, there are many effects: when inhaled, the first effects are usually respiratory and may include a bloody or runny nose, coughing, chest discomfort, difficult or short breath, and wheezing due to constriction or excess fluid in the bronchial tubes. Skin contact may cause skin sensitisation. Eye contact will cause pain, bleeding, tears, pupil constriction and blurred vision. Following exposure by any route, other systemic effects may begin within a few minutes or be delayed for up to 12 hours. These may include pallor, nausea, vomiting, diarrhoea, abdominal cramps, headache, dizziness, eye pain, and blurred vision. Severe poisoning will affect the central nervous system producing lack of coordination, slurred speech, loss of reflexes, weakness, fatigue, involuntary muscle contractions, twitching, tremors of the tongue or eyelids, and eventually paralysis of the body extremities and the respiratory muscles(18).

Chronic effects

In humans, repeated or prolonged exposure to OPs may result in the same effects as acute exposure, including the delayed symptoms. Other effects reported in workers repeatedly exposed include impaired memory and concentration, disorientation, severe depressions, irritability, confusion, headache, speech difficulties, delayed reaction times, nightmares, sleepwalking and drowsiness or insomnia (19).

Multilpleanimal studies has concluded that Dimethoate is suspected human teratogen, possible human carcinogen, has mutagenic potential and also have effects on reproduction.

Effects on wildlife

The toxicity of dimethoate for aquatic organisms and birds is moderate to high.(20) One study found that it causes temporary rhythm alterations in some bird seed-eating species. Products containing dimethioate warn not to apply to crops in open flower nor when flowering weeds are present. (21)

Fate in the environment

Dimethoate is a mobile, yet relatively non-persistent OP insecticide. The primary route of dissipation appears to be microbially-mediated degradation in anaerobic (oxygen-rich) soil, particularly under moist conditions, with a half-life (time taken to degrade to half its initial strength) of 2.4 days(22). The 1999 ACP review discussed its previous concerns over environmental fate and behaviour. They concluded that dimethoate was unlikely to leach because it is so rapidly degraded in soil, is non-volatile, was slightly persistent in sediment/water systems with a DT50 of 13-17 days and did not significantly partition to sediment(23).

The cocktail effect

It appears dimethoate creates a metabolite called demethoxon that plays a dominant role in the toxicity of dimethoate for insects and mammals. Dimethoxon is also used as an insecticide known as omethoate. Omethoate, is about 10 times more toxic and is more of a potential inhibitor to cholinesterase activity than dimethoate(24). This is an important issue as, in the past, the intake of dimethoate and omethoate have been considered separately, and consumer exposure from individual crop uses has remained below the Acceptable Daily Intake (ADI). However, if the total diet is taken into account, the ADI could be exceeded for toddlers by both dimethoate and omethoate residues and for infants by dimethoate residues. The Pesticides Safety Directorate is in the UK is currently examining the issue of combined residues(25).

Conclusions

Unlike organochlorine pesticides, OPs such as dimethoate do not persist in the environment. Instead, their problem lies in their health effects on humans and other organisms. There is little concrete evidence regarding the chronic effects of dimethoate, for example on its carcinogenicity, however its acute effects on the nervous systems of humans and wildlife have been widely observed. Even though safer alternatives to OP insecticides are available, dimethoate still remains one of the most widely used insecticides in the world. Further safer alternatives should be developed, and an alternative approach based on the encouragement of natural pest enemies widely adopted. It is also very important to consider that dimethioate poisoning is more dangerous(i.e causing high mortality ,early mortality and higher rate of intubation)than other OP compounds such as chlorpyrifos(1).This compound cause severe hypotension in intial period which pauses a challenge to clinical management.(3)In literature apart from conventional therapy advanced measures like fresh frozen plasma and haemopetrfusion are described(2).Which can be considered in earlier stages when patient is haemodynamically stable. Looking to the grievous prognosis in dimethoate poisoning early aggressive management may be fruitful to the patient.

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