Hemoperfusion in severe dimethoate poisoning

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Accepted: 4 September 1985

Key words: Dimethoate poisoning – Hemoperfusion

Dimethoate (Roxion) is an organothiophosphate widely used as a pesticide. It is a potent inhibitor of acetylcholinesterase in humans and mammals. The symptomatology of dimethoate poisoning includes all signs of cholinergic intoxication [1 - 5]:

1. Muscarinic effects: miosis, lacrimation, hypersalivation, diarrhoea, bradycardia, and bronchial hypersecretion.
2. Nicotinic effects: muscular fibrillations and fasciculations.
3. Effects on CNS: nausea, vomiting, ataxia, tonic-clonic convulsions, respiratory insufficiency, and coma.

In humans and rats 76–90% of a dose of radioactive dimethoate is excreted in the urine within 24 h [2]. Dimethoate is oxidized by the liver to omethoate and to three further metabolites, all inhibitors of acetylcholinesterase.

Severe poisoning with dimethoate is associated with an unfavourable prognosis [5]. Treatment includes administration of high doses of atropine and supportive measures. Reactivators of the cholinesterase have been recommended for the early stages [4, 5]. Extracorporeal detoxication with hemoperfusion (HP) and combined hemoperfusion/hemodialysis (HP/HD) have been reported and the efficacy of HD and HP have been studied in vitro by Okonek et al. [4]: Dimethoate has a dialysance of 59 ml/min (blood flow 100 ml/min, dialysate flow 600 ml/min; Ultraflow 200 membrane) and a HP clearance of 88 ml/min (flow 100 ml/min, activated charcoal). To our knowledge, only two cases using HP and HD/HP in severe dimethoate poisoning have been published:

1. Fifteen hours after suicidal ingestion of ~12 g dimethoate (plasma level before HP 5 μg/ml) HP with activated charcoal was performed. A dimethoate clearance of 87 ml/min (flow 162 ml/min) was calculated. A considerable rebound effect was observed on the day 6 after ingestion. The patient died on day 11 from adult respiratory distress syndrome after prolonged pneumonia [5].

2. Two hours after suicidal ingestion of ~10 g dimethoate (plasma level 2.34 μg/ml 30 min after ingestion), HP/HD was performed using activated charcoal for HP. A HP clearance of 95 ml/min and a dialysance of 85 ml/min (flow 200 ml/min, C-DAK hollow fiber dialyser Cordis Dow) was determined. 55.3 mg dimethoate was eliminated by HP, 25.3 mg by HD. No rebound effect was observed. During extracorporeal detoxication the dimethoate plasma half-life was 1.8 h [3].

We report a severe case of dimethoate poisoning and an initial plasma level which was much higher than the levels of the intoxications cited above. For extracorporeal detoxication 2 HPs were performed.

Case history

Two hours after ingestion of approximately 20 g dimethoate (50 ml Roxion), a 52-year-old male was admitted to the intensive care unit of the clinic.

On admission the patient was comatose with reduced reaction to painful stimulation. Further clinical findings included muscular fasciculation, extreme miosis, hypersalivation, and respiratory insufficiency. The blood pressure was 120/60 mm Hg and the pulse was 100/min. Pseudocholinesterase was unmeasurable (<200 U/l, normal range 3000–8000 U/l).

After intubation the patient was ventilated with 40% oxygen and a positive end expiratory pressure of 10 cm water (see Fig. 1). For detoxication, gastric la-
high plasma level, 34 μg/ml, two hemoperfusions with activated charcoal (Hämocol) and amberlite XAD 4 were performed 6 h after ingestion, each lasting for 4 h (average flow 200 ml/min). Atropine was given by infusion up to day 12.

With treatment the state of consciousness improved slowly but HP had no marked influence on the clinical condition. On day 3, persistent partial respiratory insufficiency due to pneumonia necessitated tracheotomy and prolonged assisted ventilation up to day 12. Additionally, alcoholic withdrawal symptoms developed and were treated with clomethiazole by infusion. Twenty-five days after admission, the patient was discharged fully recovered.

Methods and results

A toxicological screening showed that no substances other than dimethoate had been ingested. Dimethoate in plasma, urine, and gastric lavage fluid was determined by gas chromatography.

The plasma concentrations during therapy are depicted in Figures 1 and 2. A total of about 10 mg dimethoate was determined in the gastric lavage fluid (0.005% of the ingested dose). Ten mg of the unchanged pesticide was excreted in the urine during the first 4 days.

The amount of dimethoate eliminated by the HPs was calculated from the arteriovenous difference of dimethoate plasma concentrations and the flow. One hundred and forty-two mg (0.71% of the dose) and 14 mg (0.36%) dimethoate were removed by HP with activated charcoal and amberlite XAD 4, respectively.

![Fig. 1. Dimethoate plasma levels and clinical course of the intoxication](image1)

![Fig. 2. Arterial and venous dimethoate plasma levels during the two hemoperfusions](image2)