Objective
Chlorates are strong oxidizers and used as ingredients in matches, explosives and dyestuffs as well as herbicides. Although commercial use of chlorate-containing weed killers is banned within the EU, they can still be purchased by private consumers as “stone cleaners” in large quantities, i.e. in containers with up to 10 kilograms. We report on three cases of chlorate poisoning that occurred in spring 2011.

Case series

1. PIC Erfurt:
Accidental ingestion of a sip from a glass containing a table(?) spoonful of sodium chlorate dissolved in water was asymptomatic in a male adult after gastric lavage, activated charcoal and laxative within 2 hours after ingestion.

2. PIC Erfurt:
Inappropriate storage of a chlorate dissolution in a beverage bottle led to accidental ingestion of a sip by a 59-year-old man and resulted in gastrointestinal symptoms and haemolysis with acute renal failure. The patient recovered after haemodialysis.

3. PIC Erfurt - Case report

Patient:
37-year-old man

Route of exposure and dose:
suicidal ingestion of a larger quantity of potassium chlorate

Time of admission:
approx. 1 h after ingestion

Clinical features: (see also fig. 1)
vomiting, diarrhoea, tachycardia, methaemoglobinaemia, cyanosis, respiratory failure, leucocytosis, hyperkalaemia, coma, hypotension, renal failure, anuria

Treatment: (see also fig. 1)
administration of thiosulfate, methylene blue, toluidine blue exchange transfusion, haemodialysis

Outcome: (see also fig. 1)
The patient died in the course of multiple organ failure two days after ingestion.

Toxicity of chlorates

General:
→ adult LD₅₀: 7.5 - 35 g (~ 100 - 500 mg/kg)
→ doses of 150 grams or greater have been survived

Sodium chlorate:
→ adult LD₅₀: 214 mg/kg (~ 15 g)
→ child LD₅₀: 185 mg/kg (~ 2 - 5 g)

Potassium chlorate:
→ adult LD₅₀: 429 mg/kg (~ 30 g)

Clinical features:
- Gastrointestinal:
  → nausea, vomiting, diarrhoea, abdominal pain
- Hematologic:
  → haemolysis with methaemoglobin formation
  → secondary disseminated intravascular coagulation (DIC), cyanosis, dyspnoe, metabolic acidosis; CNS-depression
- Decreased renal function / Acute renal failure:
  → direct toxicity of chlorates to the proximal renal tubule
  → secondary to haemolysis and haemoglobinuria
- Hyperkalaemia:
  → may occur primary to large amounts of potassium chloride and secondary to haemolysis

Management of chlorate poisonings

Prevention of absorption:
- activated charcoal, gastric lavage (5% sodium bicarbonate)
- sodium thiosulfate p.o./l.v. (inactivates chlorate → chloride)

Enhanced elimination:
- (early) exchange transfusion combined with haemodialysis

Treatment of methaemoglobinaemia:
- methylene blue, toluidine chloride, ascorbic acid

Conclusion
A dose of 7.5 grams of potassium chlorate and 15 to 35 grams of sodium chlorate (1), respectively, has been lethal in adults. As strong oxidizers, chlorates can directly oxidize haemoglobin and even small doses may cause significant methaemoglobinaemia and haemolysis which consequently lead to acute renal failure. In addition, chlorates may also be directly toxic to the proximal renal tubule. Treatment of methaemoglobinaemia with methylene blue and toluidine chloride (toluidine blue), respectively, may have a limited efficacy due to chlorate-induced inactivation of glucose-6-phosphate dehydrogenase, an enzyme required for reduction of methaemoglobin. Thiosulfate has been suggested to inactivate the chlorate ion by reduction to the chloride ion, however, the efficacy of thiosulfate administration has not been confirmed. Exchange transfusion and haemodialysis may be efficient in patients with severe poisoning.

References:
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