Suicidal intoxication with potassium chlorate successfully treated with renal replacement therapy and extracorporeal liver support

We present a case of a 22-year-old male who, in a suicide attempt, ingested approximately 200 g of potassium chlorate. Upon admission to the hospital, he presented in full respiratory failure with cyanosis. Methylene blue antidote was given but found to be ineffective. The patient was intubated and mechanical ventilation was initiated. Because of renal failure with anuria, intermittent haemodialysis (IHD) followed by continuous venovenous hemodiafiltration (CVVHDF) was performed. His hospital stay was also complicated by hemolysis, disseminated intravascular coagulation, and atrial fibrillation. Transfusions of packed red blood cells, platelets, and fresh frozen plasma were necessary to correct the deficits. He also developed liver failure and required two sessions of molecular adsorbent recirculating system (MARS) therapy. On day 14 of his hospitalization, he regained consciousness, as well as full respiratory and circulatory function. There are no controlled studies addressing management of potassium chlorate poisoning. We suggest that early renal replacement therapy should be strongly considered.

Case report

A 22-year-old man, addicted to alcohol and illegal psychoactive substances, was admitted to our Toxicological Unit (TU). This occurred approximately 24 hours after ingestion of 200 g of potassium chlorate in a suicide attempt.

At the time of admission to our Toxicological Unit, the patient was unconscious, with Glasgow Coma Score of 8. His vital signs were as follows: blood pressure of 90/70 mm Hg, heart rate of 120-140 beats/min., respiratory rate of 28-35 breaths/min., temperature of 37°C, and urine output of <0.2 ml/kg/h. Cyanosis and dyspnea were present.

The patient was immediately intubated with 8.5 mm endotracheal tube and mechanical ventilation was star-
The initial blood gas analysis showed pH 7.24; pCO₂ 41.9 mmHg; pO₂ 24.9 mmHg; HCO₃⁻ 17.3 mmol/l; BE –10; SatO₂ 45.6%; MethHb 16%; serum lactate 4.1 mmol/l (N ± 2.5 mmol/l). Other biochemical results were: Na 133 mmol/l; K 8.5 mmol/l; Glu 126 mg/dl; osmolality 275.5 mmol/kg; anion gap 8.3 mmol/l; creatinine 4.1 mg/dl (N 0.7-1.2); urea 78 mg/dl (N 10-50); WBC 66.5 G/l; RBC 4.23 Tr/l; Hb 14 g/l; Ht 37.4%; Plt 85 G/l, INR 2.07 (N 0.8-1.2); APTT 79 s (N 24-35); TT 161.9 s (N 14-21); fibrinogen 1.53 g/l (N 2-4 g/l); AST 211 U/l (5-37); ALT 72 U/l (5-41); LDH 6872 U/l (N 120-240).

Because of acute kidney injury, intermittent hemodialysis (iHD) was initiated. In the first 24 hours, the patient underwent two sessions of iHD, each lasting 5 hours. On day 2, first of two 72 hour sessions of continuous veno-venous haemodiafiltration (CVVHDF) was started.

Also, during the first four days, our patient received transfusions of 6 units of packed red blood cells, 7 units of platelets, and 5 units of fresh frozen plasma to correct hematologic anemia and disseminated coagulopathy.

On day 3, atrial fibrillation was observed. It was successfully treated with synchronized electrical cardioversion.

Because of encephalopathy grade III/IV, increased level of AST up to 7748 U/l, ALT up to 4184 U/l, and bilirubin up to 12.2 mg/dl, 20 hour cycles of Molecular Adsorbent Recirulating System (MARS) therapy were performed immediately after two sessions of CVVHDF.

On day 10, the patient was extubated. Four days later he was transferred back to the city hospital in a stable condition.

Discussion
Potassium chlorate (KCIO₃) (CAS nr 3811-04-9) is a crystalline, white powder widely used in the chemical industry. There is very limited data regarding management and specific therapy of potassium chloride poisoning [2,5,7,8,10]. Toxic dose of KCIO₃ is approximately 5 g, while a single dose of 15-35 g may be fatal for adults [3,5].

Clinical features of acute poisoning include nausea, vomiting, diaphoresis, and dyspnea. Methemoglobin formation, anoxia, and disseminated intravascular coagulation are usually responsible for death in the early stage of potassium chloride poisoning, while later, renal failure is responsible for the unfortunate outcome.

To the best of our knowledge, there are no controlled studies addressing treatment of potassium chlorate poisoning [5,9,10]. Most authors propose therapy which consists of gastric lavage, use of activated charcoal, transfusion of blood products, treatment with bicarbonate, heparin, and sodium thiosulfate. Hyperbaric oxygen treatment, and in case of renal failure, intermittent hemodialysis or continuous renal replacement therapy (CRRT) are also recommended [2-9].

Treatment of methemoglobinemia with methylene blue is ineffective because potassium chlorates not only oxidize hemoglobin but also denature enzymes of erythrocytes. Inactivation of glucose-6-phosphate dehydrogenase makes methylene blue unable to reduce hemoglobin. This antidote may also aggravate hemolysis [1,7-10]. It seems that rarity of chlorate poisoning and lack of clear guidelines for KCIO₃ induced methemoglobinemia treatment, resulted in use of methylene blue in our case.

Also chlorates increase rigidity of erythrocyte membrane [7,9,10]. This results in an impairment of microcirculation, destruction of erythrocytes, and disseminated intravascular coagulation. Massive hemolysis is life-threatening, and often leads to acute kidney injury. Early initiation of RRT may prevent cardiopulmonary arrest, correct electrolyte and acid-base abnormalities, and remove toxic chlorate anions. We suggest performing iHD followed by CVVHDF. We found this treatment effective in our case.

Acute liver failure (ALF) can be another complication of acute KCIO₃ poisoning. Our report is the first one documenting ALF in acute intoxication with chlorate salts. It probably was caused by hypoxia and disseminated intravascular coagulation. We treated our patient with MARS therapy with good outcomes. We believe this intervention should be implemented early in patients with multi-organ failure.

Transfusions of blood products, in our opinion, are also necessary to correct poisoning induced anemia, thrombocytopenia, and disseminated intravascular coagulopathy, however, Ranghino et al. suggested that such procedure could be considered as a ‘constant supply’ of chlorate main target [6].

Conclusions
1. Chlorate salts can induce methemoglobin formation, haemolysis, acute kidney injury, and acute liver failure.
2. Aggressive supportive treatment with timely introduction of iHD, CRRT and ELS therapy may decrease mortality associated with acute poisoning with KCIO₃. Further research is needed.
3. Methylene blue, used as an antidote in methemoglobinemia, is not helpful in the treatment of chlorate salts poisoning.

References