Case Report

Acute ammonium dichromate poisoning in a 2 year-old child

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Abstract

Hexavalent chromium compounds are most commonly used in printing, dyeing, plastics and rayon manufacturing. Poisoning in children by ammonium dichromate, an odorless and bright orange-red crystal, are rarely reported. Acute poisoning will result in death due to multi-organ failure. The target organs that are affected by this poison are the respiratory system, kidneys, liver, eyes and skin. On ingestion, initially there is a relative lack of severe symptoms and signs. Hence, the delay in seeking medical attention could lead to the increased rate of mortality. In this case study, we report the ingestion of ammonium dichromate by a child. Despite appropriate management, such as hepatic supportive measures and plasma transfusion, the toxicity progressed to multi-organ failure and death.

Keywords: Ammonium dichromate, ascorbic acid, hepato-renal failure dimercaprol, N-acetylcysteine

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Introduction

Among the different chromium (Cr) compounds, relatively water soluble hexavalent (VI) compounds, include dichromates of sodium, potassium, and ammonium, are widely used in industrial processes and less toxic than the water insoluble compounds such as zinc chromate and calcium chromate. Cr occurs primarily in the trivalent state (III), which is the most stable form. Cr (VI) compounds are considered to be more toxic and strong oxidizing agents than the trivalent compounds.[1,2] Ammonium dichromate crystals are bright orange colored being used mainly in dye works, printing and painting works [Figure 1]. Acute poisoning is likely to occur through oral route, whereas chronic poisoning in industrial exposure is mainly via inhalation of dust and fumes.[2] Reports of dichromate poisoning in children are rare in Indian literature.

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There are many food products like squash making powder and sweetening agents that have a bright orange color, which may mimic the color of dichromate. Hence, there are great possibilities for ingestion and poisoning by this chemical in children. The problem concerned with ammonium dichromate poisoning is that in initial stages, there are no major manifestations, and hence, the patient may delay seeking medical attention. Furthermore, the children who ingested these crystals may not inform anybody unless the signs and symptoms of poisoning appear. If such cases are not treated early, all major organs are likely to be involved leading to multi-organ system failure and death.[2,3] Hence, it will be worthwhile to discuss about the manifestations and the early managements of dichromate poisoning in order to prevent its adverse effects. In this case study, we present a 2 year-old child who presented late with accidental ammonium dichromate ingestion.

Case Report

A 2 year-old boy brought to the hospital with symptoms of lethargy, abdominal pain, mucoid white semi-loose stools. History revealed that ingestion of an orange colored crystal-like substance that was kept in a packet at home. The father of this child was

working for a company printing letters on bags and purses. Initially, the child had vomiting, abdominal pain and was given symptomatic treatment locally. He then developed copious mucoid secretions from the rectum and presented to the hospital after about 14 h of ingestion. Clinical examination showed that the child was lethargic. Since, he had not passed urine, a fluid challenge was given with 0.9% normal saline in order to induce diuresis and correct his shock. He had a weak rapid pulse, shallow respiration, cold and clammy skin and developed seizures after an hour. The blood pressure was 88/60 mm Hg, respiration rate 22/min, body temperature 98.4°F and pulse rate 110 beats/min. Inotropic support with dose adjustments of dopamine (10 μg/kg/min) and dobutamine (10 μg/kg/min) were started as his shock was not responding to fluid resuscitation. He had tender hepatomegaly with tenderness in both flanks and mild icterus. Laboratory investigations at the time of admission and about 8 h after are depicted in Table 1. The child was in critical disseminated intravascular coagulation state with shock and prolonged activated partial thromboplastin time, along with the inotropic supports fresh frozen plasma (15 ml/kg) was given with compatible blood group. He passed only about 100 ml of urine in the next 10 h (urinary bladder was catherized). The seizures could be controlled with fosphenytoin infusion. However, the hypotension persisted and there was a continual mucous discharge from the rectum. Investigations revealed severe coagulopathy and hepato-renal failure [Table 1]. Per rectal bleeding was seen subsequently with deterioration of consciousness. The child was given large quantities of milk through Ryle's tube. Blood gas analysis, about 14 h after admission showed pH 7.32; pCO_2 25 mmHg; pO_2 67 mmHg; hematocrit 24%; and SO_2 70.7%. The patient continued to deteriorate and expired about 30 h after ingestion of the poison. Autopsy revealed that the brain was edematous with intracerebral and intraventricular hemorrhages. There were ulceration and hemorrhage over the mucosa of esophagus, stomach, duodenum and jejunum with edema and hemorrhages in the renal capsule and parenchyma. Scattered hemorrhages were seen in the liver with congestion, loss of architecture and destruction of the lobules.

Figure 1: Bright orange crystals of ammonium dichromate

Discussion

Ammonium dichromate is a strong oxidant that can generate free radicals. In undiluted form, it acts as a systemic poison and the potential adverse effects depend on the route of exposure.[4] Through inhalation, it is extremely destructive to tissues of the mucous membranes of upper respiratory tract and may cause ulceration and perforation of the nasal septum. Symptoms may include sore throat, coughing, shortness of breath, and labored breathing. It can produce pulmonary sensitization or allergic asthma. Higher exposures may cause pulmonary edema. Contact with skin produces redness, pain, and severe burn, whereas contact with broken skin may cause ulcers (chrome sores). Dusts and strong solutions may cause severe irritation. Contact with the eyes can cause blurred vision, redness, pain and severe tissue burns. It may cause corneal injury or blindness. Kolacinski *et al*.[1] reported a violent gastroenteritis with rice-water stools,

Table 1: Laboratory investigations at the onset and about 8 h after the admission

Investigations	At admission	At 8 h
Blood routine examination	Hb 12 g/dl, PCV 33.7%, total count 22,000/μL, neutrophils 57%, lymphocytes 38%, eosinophils 2.3%, monocytes 2.5%, basophils 0.2%, ESR 41 mm/h, and platelets count 160,000/μl	Platelets count 43,000/μl
Urine routine examination	Albumin+, sugar nil, pus cells 1-2/hpf, RBCs 4-6/hpf, bile salt negative, bile pigment negative	-
Electrolytes and renal function test	Serum total calcium 9.2 mg/dL, sodium 127 mmol/L, potassium 5.3 mmol/L, bicarbonate 11 mmol/L, and chloride 97 mmol/L Serum urea 77 mg/dL and creatinine 2.1 mg/dL	Serum sodium 122 mmol/L, potassium 5.4 mmol/L, bicarbonate 13 mmol/L, and chloride 97 mmol/L Serum urea 79 mg/dL and creatinine 2.9 mg/dL
Blood gas analysis	pH 7.29, pCO_2 28 mmHg, pO_2 63 mmHg, Hct 26%, SO_2 89.6%	pH 7.29, pCO_2 37 mmHg, pO_2 44 mmHg, Hct 29%, SO_2 91.6%
Liver function test	Serum total bilirubin 10.6 mg/dL, indirect bilirubin 11.2 mg/dL, albumin 3.2 g/dL, SGOT 1120 IU/L, SGPT 1340 IU/L, prothrombin time 26 s and activated partial thromboplastin time 57.2 s	Serum albumin 3.1 g/dL, SGOT 3340 IU/L, SGPT 2880 IU/L, prothrombin time 29 sec, activated partial thromboplastin time 58.2 s

Hb: Hemoglobin; PCV: Packed cell volume; ESR: Erythrocyte sedimentation rate; RBCs: Red blood cells; SGOT: Serum glutamic oxaloacetic transaminase; SGPT: Serum glutamic-pyruvic transaminase

yellow-green or coffee-ground emesis, corrosive burns of the mouth, esophagus, and gastrointestinal tract, and hemorrhage has occurred shortly after oral ingestion of chromates.

There are evidences of initial gastrointestinal irritation in the form of vomiting and diarrhea, but subsequently, the manifestations were mainly hepato-renal in nature.[5,6] Acute toxicity after ingestion is a result of gastrointestinal bleed more than of systemic poisoning. In this case, autopsy revealed gastrointestinal mucosal bleed and intestinal wall edema. Large doses of chromates induce albuminuria with desquamated cells, fatty degeneration, and necrosis in the kidney.[7] The fatty degeneration and necrosis can be ascribed to the free radical induced lipid peroxidation and cell membrane disruption.[4] The circulatory collapse and shock are frequently reported following overdose[8] and it was evident in this case too. Lethal dose of chromium salt varies between 6 and 8 g in adults,[4] whereas ingestion of ammonium dichromate 1 g may cause death in children.[3]

Diagnosis mainly depends on the history, direct identification of the compound, if available and clinical examination. Since, ammonium dichromate is a highly dissociable compound; it is not easily demonstrable by chemical analysis of viscera or body fluids. Hence, diagnosis and treatment of ammonium dichromate poisoning are the main challenges to the medical profession, especially when the compound ingested unknown. The treatment of dichromate poisoning and intoxication is challenging in the hands of an inexperienced care giver. Large quantities of water or if available, several glasses of milk may be given orally or through a Ryles tube. As dichromates are corrosive chemicals one should be vigilant of perforation of the gastrointestinal tract, bleeding, and hypovolemic shock. Pediatric age groups most affected seriously by hypovolemic shock are infants and children under 5 years. So correction of hypovolemic shock is crucial in the management. Various modalities of treatment using chelating agents such as dimercaprol, hemodialysis, peritoneal dialysis and exchange transfusion are the mainstay of management of dichromate poisoning.[9-12] Despite treatment with folic acid, dimercaprol, hemodialysis, and exchange transfusion, mortality had been reported in a 22-month-old infant ingested with sodium dichromate.[13] Similarly, hemodialysis and charcoal hemoperfusion appear to have little role in the management of chromium intoxication.[11] There are favorable reports of therapy with N-acetylcysteine (NAC)[14] and liver transplantation.[2] NAC can increase the excretion of chromium and also able to reverse the oliguria associated with the toxin.[11] The antioxidant properties of NAC may have theoretical value when there is multi-organ failure, especially liver, from oxidative injury. Stift *et al*.[2] reported the liver transplantation on day 6 after the ingestion of dichromate could maintain the normal liver and kidney functions. It was postulated that liver transplantation should be performed as late as possible, to let the patient's own liver to extract as much chromium from the circulation and to minimize the possibility of damage to the transplanted liver by the remaining chromium.[2]

Experimental studies postulated that substantial amounts of reducing the agent, ascorbic acid would need to be administered parenterally soon after the exposure,[10] but is not readily available. Though the exact dose and duration of ascorbic acid administration is not known, large doses of ascorbic acid (1 g/day) have been successfully used to prevent renal failure. According to Meert *et al*.,[3] an effective reduction of Cr (VI) to Cr (III) results in decreased toxicity to the cells and tissues by giving large doses of ascorbic acid. Biotransformation of Cr (VI) to Cr (III) generally reduces toxicity because the Cr (III) form does not cross cellular membrane and hence the gastrointestinal tract absorbs these compounds poorly. Hemodialysis and ascorbic acid (500 mg/day, intravenous [IV]) was also beneficial for a better outcome.[4] In this case, we could only administer large amounts of milk through Ryle's tube since IV ascorbic acid was not available. The preventive measures to avoid poisoning in young children include: (1) Not bringing poisonous substances home; (2) keep the chemicals out of the sight and reach of children; and (3) purchase chemical items in child-resistant containers. Furthermore, health care professionals should aware about the chromate poisoning and the benefits of early management, especially with IV doses of ascorbic acid.

Conclusion

The result of this case study concluded that the awareness of this rare poison if increased, could reduce the mortality rate as potential antidotes could be administered early.

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