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Acute Renal Failure due to Potassium Bromate Poisoning

Seung Heon Oh, Ho Yung Lee, Suk Ho Chung, Chang Jin Kim * and In Joon Choi*

Department of Internal Medicine and Pathology, Yonsei University College of Medicine, Seoul, Korea*

Poisoning due to potassium bromate, cold wave neutralizer for permanent waving solution in hair styling, is rarely encountered and only few cases are as a cause of acute renal failure reported in medical literature. Recently, we saw a 18 year-old-female patient, hair stylist, who was admitted to Severance Hospital due to acute renal failure after accidental ingestion of potassium bromate. This is the first known case of acute renal failure due to potassium bromate poisoning in Korea.

An 18 year-old-female patient was admitted on 28 May 1980 because of epigastric pain and vomiting of 10 days duration and total anuria and hearing disturbance for 5 days.

On past history there was no definite renal problem. There was no history of hypotension during the episode. On admission, the body temperature was 37°C, the pulse rate 124/min and the blood pressure 105/60 mmHg. The patient appeared acutely ill.

She was mentally alert. The conjunctivae were pale. Moist rales were audible on the right lower lung field. The heart was not enlarged. The abdomen was soft and flat.

There was palpable tenderness in the low abdomen. There was + pitting edema in the lower extremities. The laboratory findings were as follows; Hct was 14.1%, Hgb 4.3 gm%, WBC 12,300/mm^3 with 90% neutrophils, 10% lymphocytes, platelet 223,000/mm^3, BUN 208 mg%, creatinine 19 mg%, FBS 145 mg%, Ca 8.5 mg%, phosphorus 8.1 mg%, uric acid 21.4 mg%, total protein 5.0 gm%, albumin 2.6 gm%, total bilirubin 0.8 mg%, SGOT 8 U, SGPT 19 U, LDH 520 U, alkaline phosphatase 89 IU, Na 134 mEq/L, K 4.2 mEq/L, Cl 91 mEq/L, CO_2 19 mM/L, serum amylase 215 U. The urine gave a +++ test for protein. The sediment contained many RBC and WBC. A test for CRP and RA factor were negative. A titer for ASO was 1:85. An X-ray film of the chest was normal, and the flat plate of the abdomen showed a reflex ileus pattern. An electrocardiogram was within normal limits.

She was thought to have complete hearing loss on clinical testing and an audiogram revealed a sensorineural type deafness. The cystoscopy and retrograde pyelography showed no evidence of ureteral obstruction. Maintenance hemodialysis was required two to three times a week for management of uremia. The first renal biopsy specimen 30 days after ingestion showed patchy degenerated, necrotic and desquamated proximal tubular epithelium and dilatation. The tubular basement membranes were irregularly thickened. The interstitial tissue was edematous and showed

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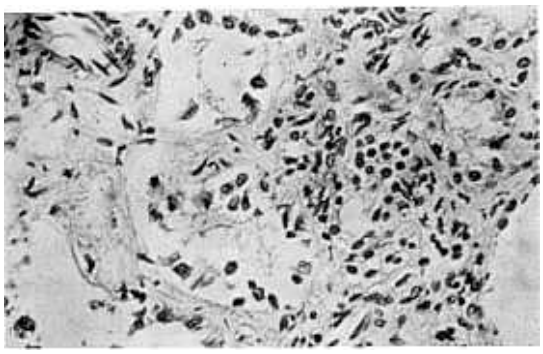

Fig. 1. The tubules are irregularly dilated, and the epithelium is degenerated and desquamated. The interstitium is edematous and minimally fibrotic. (1st biopsy, H-E, 400).

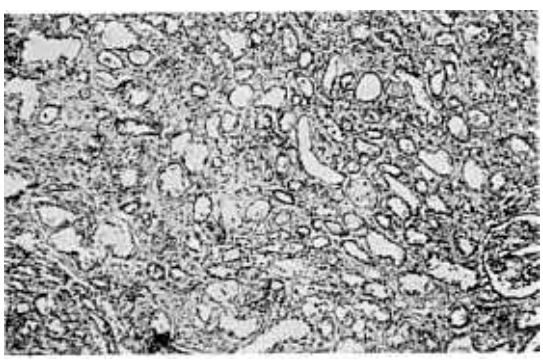

Fig. 2. The tubules are irregularly dilated and atrophic. The tubular epithelium is degenerated and the interstitium shows edema and prominent fibrosis. (2nd biopsy, H-E, 100).

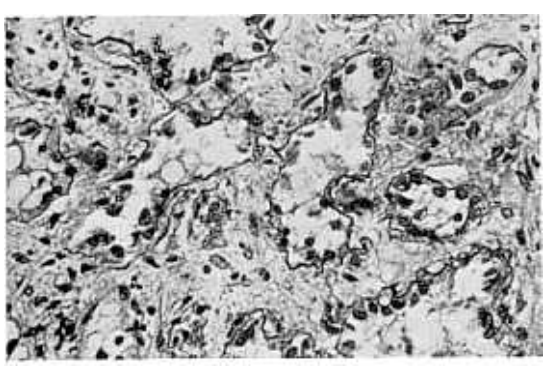

Fig. 3. The tubules are irregularly dilated, and the basement membrane is irregularly thickened focally without rrhexsis. The interstitium shows edematous widening and fibrosis. (2nd biopsy, PAS, 400).

minimal patchy focal fibrosis and cellular infiltration (Fig. 1). The glomeruli were normal appearing, but immunofluorescent study showed spotty deposits of C_3 and IgM in the mesangium.

Three weeks later, a second open renal biopsy revealed more aggravated pathological findings such as pronounced interstitial edema, fibrosis, partial tubular necrosis, low cuboidal epithelium, with atrophic and dilated tubules, in association with patchy focal cellular infiltration. (Fig. 2, 3).

On the 21st hospital day, tingling and a pain sense of the lower extremities developed.

An electromyography was normal. Analgesics were required to control the pain for about 4 weeks.

On the 24th hospital day, she complained of severe headache, tinnitus and visual disturbance and then generalized convulsions followed. Brain C-T scan showed no evidence of intracranial hemorrhage.

The accidental ingestion of a large volume of potassium bromate was discovered by her family and we were informed. During her two month's stay in this hospital maintenance hemodialysis was done 23 times. The BUN was controlled to around 30 mg% and the creatinine in the range of 6-11 mg%. From the 11th hospital day, she began to void, but the daily urine output was in the range of 100-500 ml.

The hearing impairment persisted without improvement. She was discharged with the recommendation of maintenance hemodialysis for the considered irreversible renal failure.

COMMENT

Patassium bromate is a colorless, odorless, tasteless substance that has been used as a neutralizer for permanent waving solutions in hair styling, a preservative in making bread, and a component of fusing material for explosives. The neutralizer supplied with home permanent

wave sets consisted of the bromate of either sodium or potassium, and more commonly the latter. This is supplied as a powder which is dissolved in water to make a solution. This solution is also colorless, odorless, tasteless.

Little is written regarding the effect of ingested potassium bromate. Santesson and Vickbury (1913) described vomiting, diarrhea, central nervous system symptoms (apathy and convulsions), hemolysis and renal damage in animal experiments on the toxicity of potassium bromate.

In our case, she developed vomiting, diarrhea, severe abdominal pain, anuria, convulsions and deafness after accidental ingestion of this solution. Potassium bromate is similiar to potassium chlorate but is estimated to be twenty to thirty times more toxic in experimental animals (Kitto and Dumars 1949). Neither the chlorate nor the bromate ion is destroyed to any extent by the body, but both are absorbed from the gastrointestinal tract and excreted slowly mostly by the kidneys. These salts produce gastrointestinal irritation such as vomiting, crampy abdominal pain and diarrhea. The renal damage is due in part to the products of hemolysis and in part to the oxidizing effects of bromate ion in the renal tubules. This patient showed acute renal failure and severe anemia with a Hgb of 4.5 gm%, hematocrit 14.1%, reticulocyte count of 6.2%. The cause of renal failure and anemia suggested in part the possibility of hemolysis in this case. Renal changes include hyperemia, parenchymal bleeding and epithelial degeneration which is most marked in the ascending portion of the loop of Henle. Quick and Chole (1975) reported that the kidney biopsy specimen showed interstitial edema, tubular atrophy and dilation 10 days after ingestion. Severe interstitial fibrosis, tubular atrophy, tubular basement membrane thickening and interstitial round cell infiltration three months later. But the degree of renal demage due to potassium bromate was variable.

In the early 1950's isolated cases of poisoning due to home permanent neutralizer were reported (Benson, 1951; Mackenna, 1950; Malcomson, 1951; Ollcrenshaw, 1951). These all resulted in hypotension, nausea, vomiting but no renal failure. Dunsky (1947) described a 17-month-old boy who died after ingestion of neutralizing solution from "home permanent kit." He became anuric two days after the ingestion and died on the third day. In 1941, Carratala and Urcaracy reported a case of potassium bromate poisoning with renal failure and subsequent recovery without sequele. Quick and Chole (1975) described a case of a 6-year-old boy whose renal function had gradually improved during the two years after ingestion. They explained that the slow improvement of the renal function probably represented hypertrophy of residual nephrons. But our case, unfortunately, has not improved and progressed to irreversible renal failure. She also suffered from deafness which was confirmed by audiogram as sensorineural type hearing loss. Thirteen cases of potassium bromate poisoning were reported in Japan (Hideo *et al,* 1966; Ichiro etal, 1969; Matsumoto, 1973; Nobuo *et al,* 1971). Eleven of them resulted in hearing loss. In 1975, Quick and Chole reported a case of deafness and acute renal failure due to potassium bromate poisoning. The hearing impairment has not improved. It is interesting that both the kidney and the inner ear are damaged. Several substances are known to have a toxic effect on both organs. The aminoglycoside antibiotics are also toxic to both organs. Ethacrinic acid and furosemide affect transport mechanism in the loop of Henle as well as the inner ear. In addition, antigenic similarities between the stria vascularis of the cochlea and the renal tubules are suggested. Quick *et al* (1973) using immunofluorescent staining techniques, found

that antisera to the stria vascularis showed reactivity with renal tubules. Physiologically the kidney and the inner ear are proved to be highly efficient systems for the transport of water and electrolytes across cellular barriers. We observed a generalized convulsion in this patient. Brain C-T scan revealed no intracranial hemorrhage or other abnormality. It was considered to be due to metabolic encephalopathy. Thomson HC and Wastfall (1949), Dunsky (1947) and Nobuo *et al* (1971) described a generalized convulsion due to potassium bromate poisoning. Nobuo *et al* reported as case of a 30-year-old female patient who developed schizophrenia after ingestion of cold wave neutralizer. But there was no personality change in our case. Treatment included the use of 4% sodium sulfate and hypertonic plasma as diuretics. The use of sodium thiosulfate as an antidote has been suggested.

SUMMARY

A case of acute renal failure due to poisoning by potassium bromate, cold wave neutralizer, was presented with a review of the literature. The case was notable because of severe vomiting, bloody diarrhea followed by irreversible renal failure and sensorineural type hearing loss. Maintenance homedialysis was required to control renal failure.

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