Case Report

Accidental Potassium Bromate Poisoning in Nine Adults

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Abstract

Accidental Potassium bromate poisoning is uncommon in adults, can have varied manifestations in different patients and can sometimes be deceiving. Potassium bromate white powder and oxidizing agent is used predominantly in bakeries as a maturing agent for flour and as a dough conditioner. It is also occasionally used as a neutralizer in hair kits.

This paper deals with nine cases of accidental potassium bromate poisoning working in a bakery. Almost all the patients present with pain abdomen, vomiting and diarrhea. Severe gastritis leading to hematemesis is one of the dreaded complications. Acute renal failure can ensue after 24-48 hours of intake and thus patient must be investigated in this line. All of them ingested potassium bromate powder considering it to be milk powder. Potassium bromate poisoning must be considered as a possibility in every case presenting as acute gastroenteritis like symptoms after intake of bakery products. Strict legislation is required to decrease the risk of such incidents.

Key Words: Potassium Bromate, Poisoning, Serum Creatinine, Blood Urea, Bakery

Introduction:

Potassium bromate is an oxidizing agent used predominantly in bakeries as a maturing agent for flour and as a dough conditioner. It is a bromate of potassium which takes the form of white powder. It is also occasionally used as a neutralizer in hair kits. [1] Accidental ingestion of potassium bromate tablets in children have been reported in the past but this form of accidental poisoning in adults is uncommon. Potassium bromate has been banned in many countries but still being used in some including India in local bakeries. Whether they maintain the upper permissible concentration guidelines or not is a highly questionable issue and thus many cases of potassium bromate intoxication may go unnoticed. It is thus essential to have a high index of suspicion in patients presenting with gastroenteritis like symptoms after intake of bakery products.

History:

Nine adult workers of bakery presented to medical emergency with complain of colicky abdominal pain, loose motions watery in nature and vomiting. They had a history of intake of some sweets last night in their dinner. Considering the possibility of infective gastroenteritis, patients were treated with fluids and antibiotics. After 45-50 minutes one patient developed one episode of hematemesis containing 40-50 ml of fresh blood with bits of altered blood. Now the index of suspicion was high and on taking the history in detail again it was found that all these workers had made this sweet themselves in the bakery using milk powder and sugar available in that bakery itself.

On enquiring about the ingredients repeatedly that powder was found to be actually potassium bromate rather than milk powder. Bakery officials later confirmed that the workers by mistake used potassium bromate powder rather than milk powder to prepare their sweet and then the diagnosis of potassium bromate poisoning was confirmed.

Physical Examination:

All the nine patients were conscious, alert and had stable vitals. All of them had significant dehydration. Two patients out of the lot had severe epigastric tenderness. There was no apparent abnormality detected in respiratory, cardiovascular and central nervous system. All the patients had normal urine output.

Laboratory Investigations:

Day 1

Complete blood counts, renal function tests, liver function tests, electrolytes, random blood sugar, routine urine examination, chest X-ray and ECG were performed in all the patients.
and all the investigations were found to be within normal limits.

**Treatment:**
1. Gastric lavage was not performed because by the time diagnosis of potassium bromate was ascertained, it was already more than 24 hours.
2. IV fluids were administered to combat dehydration.
3. IV proton pump inhibitors were given.
4. Input/output charting was done.
5. Dicylomine intramuscular was kept SOS for paain abdomen.

**Clinical Course of Patients over Next Few Days:**

The same patient who developed hematemesis on the day of admission developed two more episodes of hematemesis containing fresh blood (about 40-50 ml) in an interval of 2 hrs. Patient was kept on continuous pantoprazole infusion and oral antacids. He was given fresh blood transfusion. Patient responded to treatment and no further episodes of hematemesis occurred. Loose motions and vomiting stopped by day 2 in all the patients. A hematemesis occurred.

Two patients developed oliguria and nausea despite adequate fluid administration on day 3. On investigations it was found that their blood urea and serum creatinine were raised and they had developed acute renal failure. Electrolytes were however within normal range. The course of serum creatinine and blood urea in those two patients is shown in Table 1.

**Table 1: Course of Serum Creatinine and Blood Urea**

<table>
<thead>
<tr>
<th>Day of Admission</th>
<th>Patient 1</th>
<th>Patient 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Blood urea</td>
<td>Serum creatinine</td>
</tr>
<tr>
<td>Day 1</td>
<td>35</td>
<td>0.9</td>
</tr>
<tr>
<td>Day 2</td>
<td>42</td>
<td>1.1</td>
</tr>
<tr>
<td>Day 3</td>
<td>154</td>
<td>2.9</td>
</tr>
<tr>
<td>Day 4</td>
<td>166</td>
<td>3.5</td>
</tr>
<tr>
<td>Day 7</td>
<td>75</td>
<td>1.8</td>
</tr>
<tr>
<td>Day 12</td>
<td>40</td>
<td>0.6</td>
</tr>
</tbody>
</table>

**Graph 1: Serum Creatinine in Two Patients Who Developed Renal Failure**

![Graph 1: Serum Creatinine in Two Patients Who Developed Renal Failure](image1)

**Graph 2: Blood Urea Levels in Patients Who Developed Renal Failure**

![Graph 2: Blood Urea Levels in Patients Who Developed Renal Failure](image2)

Patients were treated conservatively with diuretics and fluid. None of them required dialysis. Seven patients remained asymptomatic and had normal investigation reports throughout and were discharged by day 7.

**Discussion:**

Potassium bromate poisoning can be deceiving at times. Potassium bromate is an odorless, tasteless white powder which can be confused with milk powder. It is an oxidizing agent widely consumed in bread in which it is used as an additive in the baking process. Various methods have been described for the determination of bromate residues with accuracy in variety of baked goods.

The nephrotoxicity results from interplay of increased formation of reactive oxygen species, lipid peroxidation induced DNA fragmentation, micronuclei formation and cellular proliferation. Massive haemolysis and thrombocytopenia may be seen in children with bromate poisoning.

The mechanism of bromated toxicity is not clearly understood, but proposed that renal failure could result from direct tubular toxicity due to active oxygen radicals, reduced renal perfusion from dehydration and possibly decreased vasomotor tone, hemolytic anaemia with haemoglobinuria and possibly play a role. A number of case reports of acute poisoning by potassium bromate solution have been reviewed. In children 1.5-3 years of age, ingestion of 2-4oz (53-133gm) of a 2% solution of potassium bromated cause nausea and vomiting, usually with epigastric pain and/or abdominal pain; diarreha and hematemesis occurred in some cases.

In both children and adults, oliguria and death from renal failure have been observed, partial hearing loss and complete deafness have also been reported. The toxic effects or lethal dose of potassium bromate in human has not been accurately established, but a
dose of 500mg caused serious symptoms in a 15 months old child. [19]

Usage in bakeries without quality control in high concentrations can lead to adverse outcomes. This type of intoxication can be easily misdiagnosed as infective gastroenteritis but can be far more dangerous and thus high index of suspicion is required for making the diagnosis.

Almost all the patients present with pain abdomen, vomiting and diarrhea. Severe gastritis leading to hematemesis is one of the dreaded complications. Acute renal failure can ensue after 24-48 hours of intake and thus patient must be investigated in this line. Renal failure usually ensues after 36-48 hrs of ingestion of potassium bromate. Continuous monitoring of renal function is thus warranted daily for at least one week after the suspected ingestion. (Graph 1 &2)

Possible mechanisms causing renal failure include direct tubulotoxicity due to induction of active oxygen radicals, lipid peroxidation, induced DNA fragmentation and reduced renal perfusion from dehydration and possible decreased vasomotor tone. Management is predominantly supportive.

Conclusion:

Potassium bromate poisoning must be considered as a possibility in every case presenting as acute gastroenteritis like symptoms after intake of bakery products. Strict legislation is required to decrease the risk of such incidents. Hematemesis and renal failure as the possible adverse outcomes of potassium bromate poisoning have been reported in this case report.

References: