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Epsom-salts Poisoning and a Review of Magnesium-ion Physiology

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Report of a Case

A healthy 28-year-old woman had two martinis with dinner, followed by a postprandial bottle of beer. Several hours later she experienced gastric distress and took several antacid tablets. When this did not produce relief, she obtained a "box" of epsom salts and took "several tablespoonfuls" orally. This did not result in catharsis, and the possibility exists that she repeated the dose. By midnight, the patient was feeling weak and sleepy. Why this was considered abnormal at midnight was not made clear, but the patient was brought to the hospital, where she complained of dyspnea.

Examination revealed a sleepy, healthy woman, who appeared to have enlarged breasts and a lower abdominal swelling, believed at first to be due to pregnancy, but later found to be a greatly distended bladder. After catheterization the swelling disappeared, but the patient lost consciousness.

On the basis of the history and events, an intern drew a sample of blood for toxicologic examination and administered calcium gluconate, 1 g, intravenously. This was followed by the return of consciousness, and the patient appeared to be improved. The duration of the recovery was brief; she soon began to complain of weakness in her legs. Neurologic examination did not reveal a sensory deficit, but there was obvious lower-limb weakness. Calcium gluconate was repeated, with a slight subjective improvement, but without materially changing the muscle weakness.

On rectal examination, a soft normal-colored well-formed stool was detected. There was no evidence of diarrhea. Between 1:00 AM and 2:45 AM, there was progressive ascending muscle weakness, which appeared to delineate at the fourth thoracic dermatome. The patient continued to complain of difficulty in breathing, although when asked to breathe deeply, there were normal respiratory excursions. Calcium chloride was now injected, but without subjective effect. At 3:00 AM the patient became agitated and complained bitterly of her inability to breathe, despite satisfactory diaphragmatic excursion, good vocal power and full activity of the neck and jaw muscles. Additional information was sought relative to other drugs that might have been taken, but none could be confirmed. Differential diagnosis included a thrombosis of the anterior spinal artery, Guillain-Barré syndrome, myasthenia gravis and magnesium toxicity.

Fifteen milligrams of edrophonium (Tensilon) were administered without effect. Ventilation was assisted with a breathing bag, but the patient pleaded that the mask be removed. Gastric distention was noted. An attempt was made to pass a nasogastric tube, but the patient vocally objected although she was unable to move her arms and hands in protest. Despite this, she could

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shake her head and prevent the use of a laryngoscope by clenching her teeth. Efforts to insert the nasal tube were temporarily abandoned. An additional gram of calcium chloride failed to cause improvement and, in fact, possibly led to more sedation. A gastric tube was finally passed, followed by profuse vomiting around and through the tube of approximately 2,500 ml of translucent yellow fluid. The patient did not appear to have aspirated any of the fluid, but she lost consciousness. A cuffed tube was placed easily into the trachea and the cuff inflated, but a profuse amount of pink frothy fluid bubbled out of the tube.

Blood pressure was unobtainable at this time. Respiration was controlled with oxygen; closed-chest massage was instituted immediately, and electrocardiographic leads were attached. There was no evidence of cardiac electrical activity. An additional ampule of calcium chloride was given, followed by an intracardiac injection of epinephrine, 4 ml of a 1:10,000 dilution. The electrocardiogram remained isoelectric. An external electrical stimulus was applied twice, without effect. After a third intracardiac injection of epinephrine, 1 mg, a normal spontaneous sinus rhythm appeared at a rate of 60/min with a detectable radial blood pressure of 90 mm Hg systolic. Total flaccidity and unconsciousness persisted. The pupils were dilated.

At 4:00 AM, cardiac arrest occurred again. Identical therapy was applied, requiring three separate intracardiac injections of 1 mg epinephrine before spontaneous rhythm returned. Electrical stimulation was not required. The voltages of all ECG leads were minimal. By this time, the abdomen was extremely distended in spite of a functioning nasogastric tube. The pupils remained widely dilated. The evidence of pulmonary edema seen previously had disappeared. Five hundred ml of urine drained from the Foley catheter in an hour. Additional calcium, for a total dose of 7 g, was administered without effect, and the blood pressure declined to 60 mm Hg, systolic. At 4:40 AM, a mixture of 0.4 mg atropine sulfate and 1 mg prostigmin was injected intravenously at the suggestion of the Poison Control Center, but after only half of this dose had been given, asystole developed, lasting one minute. Additional calcium was without effect, and at 5:00 AM cardiac arrest occurred for the fourth time and was successfully treated again with intracardiac epinephrine. At 5:20 AM, the fifth cardiac arrest appeared, and repeated efforts failed to restore heart activity. The patient was pronounced dead at 5:45 AM.

Autopsy revealed all organ systems normal, except for minimal congestion of the lungs. The bowel was not injected (carefully examined because of the possibility that botulinus toxins might have been present in the beer). There was little fluid in the bowel lumen. Microscopic examinations failed to reveal organ abnormalities. The following day the results of blood tests were reported: calcium, 17.5 mg/100 ml or 8.7 mEq/l (normal 9–11 mg/100 ml or 5 mEq/l); magnesium, 43 mg/100 ml or 35.8 mEq/l (normal 1.5–2 mEq/l).

Additional Information

The patient's intake of magnesium sulfate must have exceeded the dose she admitted taking. The average human diet contains 20 to 40 mEq of magnesium in foods ingested per day, of which 10 mEq usually is absorbed from the upper portion of the gastrointestinal tract.[1] Magnesium (unbound) is excreted through the kidney.[1] There did not seem to be any restriction to renal excretion in this patient considering the high urinary output. One gram of magnesium sulfate as the hydrate is equivalent to 8.12 mEq of magnesium. Generally, only 10 per cent of an ingested dose is rapidly absorbed.[1] The patient, therefore, must have taken as much as half a pound of epsom salts; the empty can was found subsequently. How this amount could have been tolerated is unclear. Magnesium ions in the antacid tablets may have been additive; the patient had ingested an unknown number of these. The influence of alcohol on the absorption of epsom salts in this patient is uncertain. However, the patient's mother reported, postmortem, that her daughter had experienced an episode of weakness following an epsom-salts "purge" three years earlier.

Hypermagnesemia

This syndrome may follow acutely the ingestion of toxic doses, but is more prone to occur with chronic renal insufficiency.[1] Toxic responses are generally noted if the blood magnesium exceeds 4 mEq/l; above 10 mEq/l complete heart block will occur.[1,5] Magnesium sulfate sometimes is employed to treat toxemia of pregnancy, where it probably lowers blood pressure by peripheral vasodilation. Hypermagnesemia, whether it appears in chronic renal disease, during the treatment of eclampsia, or in poisoning, is supposedly effectively treated by antagonizing the magnesium effect with the calcium ion.[1,5] With magnesium toxicity, patients become sedated or unconscious, but they awaken with calcium. The rapidity with which patients awaken has not been well explained.[1,2]

One specific effect of magnesium is to block neuromuscular transmission by: a) decreasing

the endplate potential by blocking the release of acetylcholine; b) decreasing the endplate response to the available acetylcholine; or c) decreasing the muscle-fiber excitability in response to direct stimulation.[1, 4, 6, 8] With specific reference to the heart, magnesium appears to decrease the upstroke velocity of the action potential.[6, 7]

Recent studies of animals suggest that magnesium does not have general anesthetic properties, but that the sleeplike state probably results from cerebral hypoxia associated with decreased cardiac function.[3] Data obtained from cats confirms the clinical observation that patients treated with magnesium sulfate require smaller doses of muscle relaxants during operative procedures.[2]

Discussion

Review of this case history suggests several areas in which therapy might have been improved. First, a nasogastric tube should have been inserted earlier. Second, the blood levels of calicum and magnesium should have been determined soon after admission. Third, renal dialysis, which is normally effective in removing excesses of magnesium ions, should have been considered. All of these considerations appear logical retrospectively.

The use of prostigmin and a cardiac pacemaker make for interesting speculation. On the assumption that electrical cardiac activity did not appear possible despite calcium therapy, electrical pacing was not even considered. Studies suggest that under experimental conditions changes in the concentration of magnesium have little effect on the transmembrane potentials of cardiac muscle unless the level of the calcium is low.[7] Under this condition, a simultaneous decrease in magnesium gives rise to a marked prolongation of the action potential of both the atrium and the ventricle. In the case reported, calcium and magnesium concentrations both were high. Prostigmin should permit more acetylcholine to be available at the endplate by inactivating cholinesterase, *if* it is capable of being released. If, however, acetylcholine cannot be released, because the release is blocked by excessive magnesium, then in theory the action of prostigmin would be directly on the endplate itself.[1] This would further block the endplate and, in fact, may explain why such a small dose of prostigmin in this patient caused the third cardiac arrest.

Finally, does alcohol change either the uptake rate of epsom salts or the site of uptake? In our case it would appear that the site of absorption must have been in the stomach rather than in the bowel, since the bowel was neither congested nor full of fluid at autopsy, and a normally-formed stool was detected prior to death. The distention of the stomach and the bladder and the failure of intestinal motility might be related to a neural black, which developed early.

While the literature indicates that excesses of magnesium may be balanced by therapeutic increase in calcium levels, this appears not to be correct when huge doses of magnesium are involved. Whether electrical cardiac pacing is worthwhile in such a situation was not explored, but should be attempted in similar situations in the future.

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