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TOXICOLOGY CASE FILE

Baking Soda Can Settle the Stomach but Upset the Heart: Case Files of the Medical Toxicology Fellowship at the University of California, San Francisco

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Case Presentation

A 50-year-old man with a past medical history of diabetes mellitus, hypertension, and Zollinger–Ellison syndrome came to the emergency department (ED) with a history of increasing vomiting and diarrhea for 1 week and poor oral intake for a few days. He described generalized weakness and lethargy the day before presentation. During the initial ED evaluation, he suddenly had an episode of ventricular tachycardia treated with a single synchronized cardioversion followed by an amiodarone drip. His initial laboratory investigation revealed marked hypokalemia (potassium 2.0 mEq/L), hyponatremia (sodium 122 mEq/L), hypochloremia (59 mEq/L), and elevated serum bicarbonate (46 mEq/L). The BUN was 8 mg/dL, creatinine was 0.9 mg/dL, and glucose was >600 mg/dL. Arterial blood gases revealed a pH of 7.6, pO_2 100 mm Hg, pCO_2 59 mm Hg, and calculated bicarbonate 60 mEq/L. Serum calcium was 8.1 mg/dL, magnesium 1.6 mg/dL, phosphate 1.3 mg/dL, and albumin 2.7 g/L.

On further questioning, it was discovered that the patient had been experiencing persistent heartburn and gastric upset for the past 7 years following gastrinoma removal. He had not been compliant with his usual medication regimens for hypertension, diabetes, and gastric hyperacidity and had been using baking soda as an antacid, adding a teaspoon of the white powder to his drinks on an as-needed basis daily for years.

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What Is the Sodium/Bicarbonate Content of Baking Soda?

According to a manufacturer, each teaspoon of baking soda contains 4.8 g, corresponding to 59 mEq of sodium and 59 mEq of bicarbonate [1]. By comparison, oral sodium bicarbonate tablets (650 mg) contain only 7.7 mEq of sodium and 7.7 mEq of bicarbonate [2].

What Are the Common Situations in Which Baking Soda Toxicity Is Seen?

The most common case reports of baking soda toxicity involve its excessive use as an antacid [3–5]. Baking soda has been suggested as a safe replacement for sodium bicarbonate in the management of chronic metabolic acidosis [6]; however, there are case reports of morbidity when baking soda was used for this purpose [7]. Surprisingly, there is one case report of systemic toxicity resulting from topical application of baking soda as a treatment for diaper rash [8].

What Are the Electrolyte and Acid–Base Abnormalities Seen with Baking Soda Overdose?

Alkalosis Metabolic alkalosis due to excessive intake of bicarbonate is not usually seen because the kidney responds to a high bicarbonate load by increasing bicarbonate excretion. However, when bicarbonate excretion is impaired, it may accumulate and result in alkalosis [9–11]. The kidney responds initially to an abnormal bicarbonate load by inducing

a bicarbonate diuresis in which there will be loss of sodium, chloride, potassium, and volume. The reduction in volume can decrease the glomerular filtration rate, which will further decrease the filtered load of bicarbonate [9]. Hypokalemia and hypochloremia also contribute to bicarbonate retention [3, 12].

Hypokalemia It is the most common serious complication of sodium bicarbonate intoxication and it may also worsen the alkalosis. Alkalosis causes an intracellular shift of potassium [13, 14]. Excessive urinary excretion of bicarbonate takes sodium along with it, followed by potassium as sodium is depleted [15]. With depletion of sodium and then potassium, hydrogen ions are increasingly excreted as the cation accompanying bicarbonate. This worsens systemic alkalosis while paradoxically acidifying the urine [16]. Once sustained hypokalemia occurs, it can directly induce metabolic alkalosis by stimulating proximal renal H^+ ion excretion and net bicarbonate reabsorption [17].

Hypochloremia It is the one of the most common electrolyte abnormalities seen with alkalosis, yet its central role is often underrecognized [4]. The loss of gastric fluid, which contains 60 to 140 mM HCl, is the most common reason for chloride loss. In the Zollinger–Ellison syndrome or pyloric stenosis, these losses may be massive. Once hypochloremia occurs, it can worsen the alkalosis by the following mechanism: bicarbonate-secreting (type B) intercalated cells in the distal collecting ducts express pendrin on their luminal membranes. This protein normally transports bicarbonate into the luminal fluid in exchange for chloride. If there is insufficient chloride in the luminal fluid (e.g., chloride depletion alkalosis), this exchanger will not be able to excrete the bicarbonate [18]. Studies in rats show that correction of the hypochloremia can correct the alkalosis even without correcting the volume and sodium depletion [18], although this is moot in clinical practice because saline (containing both sodium and chloride ions) is commonly used in resuscitation fluids.

Hypocalcemia It occurs because alkalosis causes a decrease in ionized calcium by changing protein-calcium binding, and can result in tetany and cardiac dysrhythmias [7, 19].

Hypernatremia Hypernatremia from the sodium load in sodium bicarbonate is a common finding and can cause CNS manifestations including irritability, restlessness, lethargy, and seizures [20–22]. Our patient had hyponatremia, a portion of which might be explained by "pseudohyponatremia" owing to his severe hyperglycemia (glucose >600 mg/dL), as well as chronic GI losses, ADH secretion in response to hypovolemia, and possibly diuretic use.

Can a Urinary Sodium Level Be Used in Patients with Alkalosis to Determine the Volume Status?

Measurement of the urine sodium concentration is used in many conditions to support the diagnosis of volume depletion, with urine levels <20 mmol/L suggesting hypovolemia [15, 23]. However, volume depletion in patients with metabolic alkalosis may not be associated with low urine sodium levels because bicarbonate gets excreted largely as the sodium salt, resulting in an inappropriately high urine sodium level. Urine chloride is a more accurate measure of volume status in patients with metabolic alkalosis because the urine chloride concentration will be low, reflecting both the true volume status and the associated hypochloremia [18].

What Are the Respiratory, Cardiac, and Neurological Effects of Excessive Use of Baking Soda?

Respiratory Effects Hypercapnia is a well-documented complication of severe metabolic alkalosis, with apnea being reported in children [20, 24]. Compensatory hypoventilation is a well-known physiological response to metabolic alkalosis but is commonly thought to be limited by the development of hypoxia [24–26]. However, alkalosis has been shown to reduce the sensitivity of peripheral chemoreceptors to hypoxia [25]. This may explain why severe hypercapnia can be seen in patients with significant metabolic alkalosis, despite the presence of marked hypoxia.

Cardiac Effects Metabolic alkalosis can cause a decrease in myocardial contractility due to a blunted response to epinephrine [27]. Also, electrolyte disturbances such as hypokalemia and hypocalcemia can cause QT interval prolongation and ventricular arrhythmias, with hypokalemia-induced ventricular tachycardia reported in patients with baking soda intoxication [3, 12].

Neurological Effects Neurological effects including paresthesias, muscle twitching, tetany, and myoclonus have been reported and are probably due to hypocalcemia [7, 12, 19].

Are There Any Other Special Populations That May Be Adversely Affected by Use of Excessive Amounts of Baking Soda?

There are case reports of significant morbidity and mortality from heavy baking soda use in alcoholic patients [3, 28, 29]. Many alcoholics have chronic dyspepsia and may overuse over-the-counter antacids including baking soda [3]. Also, alcoholic patients are at greater risk of volume depletion

either due to poor intake or excess loss from vomiting, leading to frequent ED visits and hospitalizations for intravenous hydration and correction of electrolyte disturbances [4]. Hypertension, rhabdomyolysis, and renal failure requiring dialysis were reported in an alcoholic patient with long-term abuse of baking soda [29].

Pregnant patients with pica leading to repeated ingestion of baking soda have presented with signs and symptoms mimicking preeclampsia [30, 31]. Rhabdomyolysis has been reported in pregnancy as well [31].

Patients taking diuretics should be advised against use of baking soda, not only because of the sodium load but also because of the risk of hypokalemia [3, 15].

Are There Any Warnings or Safety Instructions on Baking Soda Products?

The following are the exact instructions for oral use of one of the most popular baking soda products (Arm and Hammer®; Church and Dwight, Princeton NJ) as found on the side of the package:

"Add one half teaspoon to one half glass (4 fl. oz.) of water every 2 h, or as directed by physician. Dissolve completely in water. Accurately measure one half teaspoon. Do not take more than the following amounts in 24 h: seven one half teaspoons or three one half teaspoons if you are over 60 years" [1].

The package also warns against use of the maximum dose for more than 2 weeks and recommends the user to ask a doctor before using if the patient is on a sodium restricted diet or taking other medications. After December 1990, the printed instructions were also modified to advise against administering the product to children under age 5 years because of reported seizure and respiratory depression in children [20, 24]. The seizure occurred in a 6-week-old baby who had being receiving "a pinch" of baking soda in water from his mother to help the infant burp [20]. The package also warns the user not to take the product when the stomach is overly full from food or drink. This warning was added at the request of the US Food and Drug Administration because of multiple case reports of spontaneous gastric rupture due to production of large volumes of carbon dioxide on neutralization of stomach acid by the ingested bicarbonate; this potentially lethal effect was also demonstrated in cadaver studies [32, 33].

Case Conclusion

In the ED, the patient was given 2 L of intravenous normal saline and was started on intravenous potassium chloride replacement. He was also started on insulin using a sliding scale regimen. He was admitted to the intensive care unit. He had no more cardiac events. His electrolytes normalized over the course of 3 days. He was discharged home on day 6 with advice to stop using baking soda and to restart his proton pump inhibitors.

Conflict of Interest None of the authors have any conflicts of interest.

References

1. ARM & HAMMER® Baking soda package. http://www.armandhammer.com/solutions/solution-53/Antacid.aspx. Accessed 9 Oct 2012
2. Sodium bicarbonate tablet [Rugby Laboratories, Inc.] Drug fact sheet. http://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?id=33107. Accessed 9 Oct 2012
3. Fitzgibbons LJ, Snoey ER (1999) Severe metabolic alkalosis due to baking soda ingestion: case reports of two patients with unsuspected antacid overdose. J Emerg Med 17(1):57–61
4. Thomas SH, Stone CK (1994) Acute toxicity from baking soda ingestion. Am J Emerg Med 12(1):57–59
5. Ajbani K, Chansky ME, Baumann BM (2011) Homespun remedy, homespun toxicity: baking soda ingestion for dyspepsia. J Emerg Med 40(4):e71–e74
6. Booth BE, Gates J, Morris RC Jr (1984) Grocery store baking soda a source of sodium bicarbonate in the management of chronic metabolic acidosis. Clin Pediatr (Phila) 23(2):94–96
7. Razzavi B (2000) Letter to editor. Baking soda toxicity. Am J Med 108(9):756–757
8. Gonzalez J, Hogg RJ (1981) Metabolic alkalosis secondary to baking soda treatment of a diaper rash. Pediatrics 67(6):820–822
9. Abreo K, Adlakha A, Kilpatrick S, Flanagan R, Webb R, Shakamuri S (1993) The milk alkali syndrome. A reversible form of acute renal failure. Arch Intern Med 153(8):1005–1010
10. Khanna A, Kurtzman NA (2006) Metabolic alkalosis. J Nephrol 19(Suppl 9):S86–S96
11. Gawarammana IB, Coburn J, Greene S, Dargan PI, Jones AL (2007) Severe hypokalaemic metabolic alkalosis following ingestion of Gaviscon®. Clin Toxicol (Phila) 45(2):176–178
12. Mennen M, Slovis CM (1988) Severe metabolic alkalosis in the emergency department. Ann Emerg Med 17(4):354–357
13. Burnell JM, Scribner BH, Uyeno BT, Villamil MF (1956) The effect in humans of extracellular pH change on the relationship between serum potassium concentration and intracellular potassium. J Clin Invest 35(9):935–939
14. Aronson PS, Giebisch G (2011) Effects of pH on potassium: new explanations for old observations. J Am Soc Nephrol 22(11):1981–1989
15. Yi JH, Han SW, Song JS, Kim HJ (2012) Metabolic alkalosis from unsuspected ingestion: use of urine pH and anion gap. Am J Kidney Dis 59(4):577–581
16. Palmer BF, Alpern RJ (1997) Metabolic alkalosis. J Am Soc Nephrol 8(9):1462–1469
17. Gennari FJ (2011) Pathophysiology of metabolic alkalosis: a new classification based on the centrality of stimulated collecting duct ion transport. Am J Kidney Dis 58(4):626–636
18. Luke RG, Galla JH (2012) It is chloride depletion alkalosis, not contraction alkalosis. J Am Soc Nephrol 23(2):204–207

19. Kaye M, Somerville PJ, Lowe G, Katis M, Schneider W (1997) Hypocalcemic tetany and metabolic alkalosis in a dialysis patient: an unusual event. Am J Kidney Dis 30(3):440–444
20. Nicholas MH, Wason S, Gonzalez del Rey J, Benfield M (1995) Baking soda a potentially fatal home remedy. Pediatr Emerg Care 11(2):109–111
21. Puczynski MS, Cunningham DG, Mortimer JC (1983) Sodium intoxication caused by use of baking soda as a home remedy. Can Med Assoc J 128(7):821–822
22. Fuchs S, Listernick R (1978) Hypernatremia and metabolic alkalosis as a consequence of the therapeutic misuse of baking soda. Pediatr Emerg Care 3(4):242–243
23. Assadi F (2012) Hyponatremia: a problem-solving approach to clinical cases. J Nephrol 25(4):473–480
24. Pappano D (2011) Alkalosis-induced respiratory depression from infantile hypertrophic pyloric stenosis. Pediatr Emerg Care 27(2):124
25. Javoaheri S, Shore NS, Rose B, Kazemi H (1982) Compensatory hypoventilation in metabolic alkalosis. Chest 81(3):296–301
26. Perrone J, Hoffman RS (1996) Compensatory hypoventilation in severe metabolic alkalosis. Acad Emerg Med 3(10):981–982
27. Khanna A, Kurtzman NA (2001) Metabolic alkalosis. Respir Care 46(4):354–365
28. Niewiński G, Korta T, Debowska M, Kosiński C, Kubik T, Romanik W, Kański A (2008) Cardiac arrest in chronic metabolic alkalosis due to sodium bicarbonate abuse. Anestezjol Intens Ter 40(3):173–177 (Article in Polish, Abstract in English)
29. Forslund T, Koistinen A, Anttinen J, Wagner B, Miettinen M (2008) Forty years abuse of baking soda, rhabdomyolysis, glomerulonephritis, hypertension leading to renal failure: a case report. Clin Med: Case Rep 1:83–87
30. Johnson BE (1989) Resistant hypertension due to pica (baking soda). Lancet 1(8637):550–551
31. Grotegut CA, Dandolu V, Katari S, Whiteman VE, Geifman-Holtzman O, Teitelman M (2006) Baking soda pica, a case of hypokalemic metabolic alkalosis and rhabdomyolysis in pregnancy. Obstet Gynecol 107(2pt2):484–486
32. Lazebnik N, Lellin A, Michowitz M (1986) Spontaneous rupture of the normal stomach after sodium bicarbonate ingestion. J Clin Gastroenterol 8(4):454–456
33. Food and Drug Administration (1994) Antacid drug products for over-the-counter human use. Fed Regist 59(22)