References

1. López-Vicente J, Rodríguez-Alcalde D, Hernández-Villalba L, et al. Proctitis as the clinical presentation of lymphogranuloma venereum, a re-emerging disease in developed countries. Rev Esp Enferm Dig. 2014;106:59–62, http://dx.doi.org/10.4321/s1130-01082014000100011.
2. Craxford L, Fox A. Lymphogranuloma venereum: a rare and forgotten cause of rectal stricture formation. Int J STD AIDS. 2018;29:1133–5, http://dx.doi.org/10.1177/0956462418773224.
3. Bissessor M, Fairley CK, Read T, et al. The etiology of infectious proctitis in men who have sex with med differs according to HIV status. Sex Transm Dis. 2013;40:768–70.
4. Morgado-Carrasco D, Alsina-Gibert M, Bosch-Mestres J, et al. Infecciones de transmisiόm sexual con afectación anorectal: agentes causales, coinfecciones, infección por el VIH y conductas de riesgo. Med Clin (Barc). 2017, http://dx.doi.org/10.1016/j.medcli.2018.02.014.
5. Gallegos M, Bradly D, Jakate S, et al. Lymphogranuloma venereum proctosigmoiditis is a mimicker of inflammatory bowel disease. World J Gastroenterol. 2012;718:3317–21, http://dx.doi.org/10.3748/wjg.v18.i25.3317.
6. Høie S, Knudsen LS, Gerstoft J. Lymphogranuloma venereum proctitis: a differential diagnose to inflammatory bowel disease. Scand J Gastroenterol. 2011;46:503–10, http://dx.doi.org/10.3109/00365521.2010.537681.
7. Soni S, Srirajaskanthan R, Alexander SB, et al. Lymphogranuloma venereum proctitis masquerading as inflammatory bowel disease in 12 homosexual men. Aliment Pharmacol Ther. 2010;32:59–65, http://dx.doi.org/10.1111/j.1365-2036.2010.04313.x.
8. Svidler-López L, La Rosa L, Entrocassi AC, et al. Rectal lymphogranuloma venereum, Buenos Aires, Argentina. Emerg Infect Dis. 2019;25:598–9, http://dx.doi.org/10.3201/eid2503.180600.
9. Foschi C, Marangoni A, D'Antuono A, et al. Prevalence and predictors of lymphogranuloma venereum in a high risk population attending a STD outpatients clinic in Italy. BMC Res Notes. 2014;7:225, http://dx.doi.org/10.1186/1756-0500-7-225.

G. Mosquera-Klinger*, S. Berrio, J.J. Carvajal, F. Juliao-Baños, M. Ruiz

Unidad de Gastroenterología y Endoscopia Digestiva, Hospital Pablo Tobón Uribe, Medellín, Colombia

* Corresponding author. Calle 78B #69-240, Consultorio 154, Hospital Pablo Tobón Uribe, Medellín, Colombia.
E-mail address: gami8203@yahoo.com
(G. Mosquera-Klinger).

2255-534X/ © 2020 Asociación Mexicana de Gastroenterología. Published by Masson Doyma México S.A. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Spontaneous rupture of the stomach secondary to bicarbonate ingestion☆

Ruptura espontánea gástrica secundaria a ingesta de bicarbonato

Sodium bicarbonate ingestion in the general population is common, given that it is an antacid employed for dyspepsia and gastroesophageal reflux, but it has been associated with spontaneous gastric rupture. The first case was reported in 1842 by Thompson et al.[1] The phenomenon has not been widely studied, and the following are among the most common causes of spontaneous gastric perforation: abundant food ingestion by patients with psychiatric disorders (*anorexia nervosa* and bulimia), sodium bicarbonate ingestion, superior mesenteric artery syndrome, gastric infarction, strangulated hiatal hernia, gastric volvulus, and trauma. Because it is a pathology with a high mortality rate, it requires immediate intervention. Thus, emphasizing the existence of these cases is of the utmost importance.[2]

A 51-year-old man had illness onset 24 h prior to hospital admission. Upon ingesting sodium bicarbonate after having eaten an abundant amount of food, the patient had a sudden pain in the epigastrium, and went to the emergency department. He arrived conscious and aware of his surroundings, with a respiratory rate of 28 bpm, heart rate of 98 bpm, blood pressure of 130/98 mmHg, and temperature of 36.6 °C. His lungs were well ventilated, and his heart sounds had a regular rhythm. His abdomen was painful upon palpation and there were signs of peritoneal irritation in the four quadrants. Computed tomography showed intra-abdominal free air and identified gastric content leakage into the peritoneal cavity, as well as striation of the adjacent fat at the level of the lesser curvature (Fig. 1). The patient was taken to the operating room. A hollow organ perforation was revealed laparoscopically, showing free gastric fluid and generalized peritonitis. A perforation measuring approximately 5 × 5 cm was viewed in the lesser curvature of the stomach (Fig. 2). The procedure was converted and supraumbilical laparotomy was performed. The cavity was washed and partial gastrectomy, encompassing the lesser curvature, was carried out. The pylorus was preserved, utilizing a GIA linear stapler (Medtronic, USA), as was intestinal transit. The patient was released after seven days of hospitalization, tolerating oral diet, and with no complications. The pathology study reported no inflammatory process and ruled out neoplasia or peptic acid disease. At the postoperative follow-up at six months, the patient had no complications.

There are two theories that explain the pathogenesis of spontaneous rupture of the stomach: the mechanical theory and the atonic theory.[3,4] The atonic theory, proposed by Brinton in 1859, postulates that after prolonged periods of fasting, the stomach undergoes atony and muscular atrophy, associated with gastric wall weakness and delayed gastric emptying. That theory is related to patients with *anorexia nervosa* and bulimia, in whom sudden abundant food ingestion results in acute gastric dilation, causing an increase in intragastric pressure that exceeds the gastric venous pressure, with consequent wall ischemia.[1,2] In 1861, Karl Freiherr von Rokitansky postulated the mechanical theory, or superior mesenteric artery syndrome, in which the

☆ Please cite this article as: Patiño-Gallegos JA, González-Urquijo M, Padilla-Armendáriz D, Leyva-Alvizo A. Ruptura espontánea gástrica secundaria a ingesta de bicarbonato. Rev Gastroenterol Méx. 2021;86:315–317.

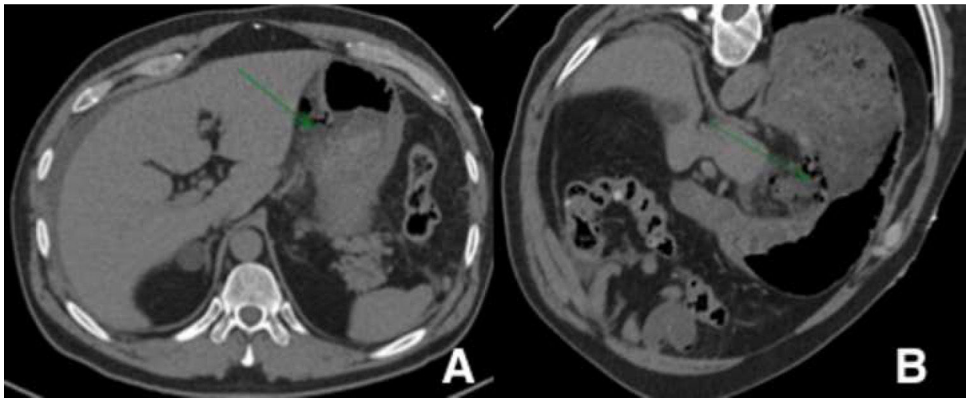

Figure 1 Computed tomography. The green arrow shows the rupture of the stomach with free air. A) Axial view, B) Oblique view.

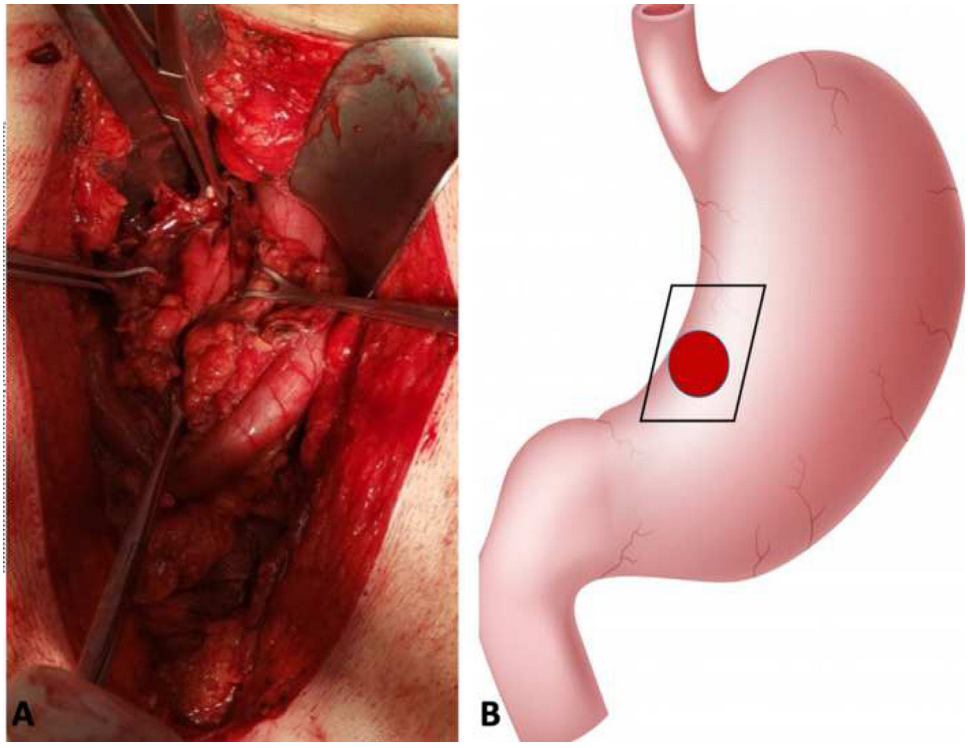

Figure 2 A) Rupture of the stomach in the lesser curvature, measuring 5 × 5 cm. B) Illustration. The red circle is the perforation site. The black rhombus indicates the resection margins.

vascular compression of the duodenum causes acute gastric dilation.[4,5] Both theories are related to acute gastric dilation, causing tissue ischemia due to reduced venous flow. Venous blood flow has been reported to require an intragastric pressure between 19—20 cm H_2O, or 3–4 L of fluid in the stomach.[3–6]

There are few reports in the literature on spontaneous rupture of the stomach due to sodium bicarbonate ingestion. One study confirmed that adding sodium bicarbonate to 2–3 L of diluted hydrochloric acid is sufficient for creating a rupture in the stomach, and also showed that the most important factors before adding the sodium bicarbonate were the volume of the solution, the quantity of reactants, the volume of air in the mixture, and the partial pressure of CO_2, plus the importance of the presence of food, alcohol, or carbonic anhydrase.[6] Those authors concluded that the ingestion of large quantities of sodium bicarbonate, with a distended stomach full of air, food, or liquid, could be an important factor for gastric rupture because it causes the release of hundreds of milliliters of gas, in less than three minutes.[7] Rupture can occur when intragastric pressure reaches 120 mmHg or 4 L of fluids.[2,4] Several cases have been reported, in which rupture of the stomach was due to abundant food intake, prior to taking sodium bicarbonate. In the majority of cases, rupture occurred in the lesser curvature, as in our patient, because that area of the stomach is shaped like a sphere, causing greater tension, and in turn, higher pressure.[8]

We conclude that acute gastric perforation after sodium bicarbonate ingestion is a rare entity with a high mortality rate, if not diagnosed opportunely, just like all perforations of the stomach. It is of the utmost importance to include the etiology described herein in the differential diagnosis of sudden epigastric pain, in patients with a history of eating

large quantities of food and using sodium bicarbonate as a medication, even if there is no history of an eating disorder.

Ethical considerations

The authors declare that no experiments were conducted on humans or animals for the present study, that they have followed the protocols of their work center on the publication of patient data, and that they have preserved patient anonymity at all times. Informed consent was obtained from the patient described in the article.

Financial disclosure

No specific grants were received from public sector agencies, the business sector, or non-profit organizations in relation to this study.

Conflict of interest

The authors declare that there is no conflict of interest.

References

1. Trindade EN, von Diemen V, Trindade MRM. Acute gastric dilatation and necrosis: a case report. Acta Chir Belg. 2008;108:602–3, http://dx.doi.org/10.1080/00015458.2008.11680297.
2. Evans DS. Acute dilatation and spontaneous rupture of the stomach. Br J Surg. 1968;55:940–2, http://dx.doi.org/10.1002/bjs.1800551215.
3. Mishima T, Kohara N, Tajima Y, et al. Gastric rupture with necrosis following acute gastric dilatation: report of a case. Surg Today. 2012;42:997–1000, http://dx.doi.org/10.1007/s00595-012-0162-4.
4. Sahoo MR, Kumar AT, Jaiswal S, et al. Acute dilatation, ischemia, and necrosis of stomach without perforation. Case Rep Surg. 2013:1–4, http://dx.doi.org/10.1155/2013/984594.
5. Powell JL, Payne J, Meyer CL, et al. Gastric necrosis associated with acute gastric dilatation and small bowel obstruction. Gynecol Oncol. 2003;90:200–3, http://dx.doi.org/10.1016/s0090-8258(03)00204-x.
6. Brismar B, Strandberg A, Wiklund B. Stomach rupture following ingestion of sodium bicarbonate. Acta Chir Scand Suppl. 1986;530:97–9.
7. Lazebnik N, Iellin A, Michowitz M. Spontaneous rupture of the normal stomach after sodium bicarbonate ingestion. J Clin Gastroenterol. 1986;8:454–6, http://dx.doi.org/10.1097/00004836-198608000-00015.
8. LeDoux MS, Sillers MJ, Atkins CP. Spontaneous rupture of the stomach in an adult. South Med J. 1991;84:399–400, http://dx.doi.org/10.1097/00007611-199103000-00024.

J.A. Patiño-Gallegos, M. González-Urquijo, D. Padilla-Armendáriz, A. Leyva-Alvizo*

Tecnológico de Monterrey, Escuela de Medicina y Ciencias de la Salud, Monterrey, Nuevo León, México

*Corresponding author at. Dr. Ignacio Morones Prieto 3000, Monterrey, 64710 Mexico. Tel.: +52 81 15027649.
E-mail address: adolfoleyva@gmail.com (A. Leyva-Alvizo).

2255-534X/ © 2020 Asociación Mexicana de Gastroenterología. Published by Masson Doyma M?xico S.A. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Seronegative celiac disease: to find it, you have to look for it. A pediatric case report☆

Enfermedad celiaca seronegativa: para encontrarla, hay que buscarla. Reporte de caso pediátrico

Celiac disease (CD) is an autoimmune enteropathy triggered by gluten ingestion in genetically susceptible individuals. Prevalence in Mexico is estimated at 0.9%, and its diagnosis is complicated by the variability of clinical manifestations.[1,2]

An 18-month-old female infant, the first child of nonconsanguineous healthy parents from Tabasco, with no family history of CD or autoimmune diseases, was delivered at full-term by cesarean section, weighing 3.5 kg and measuring 52 cm in length. She had her first bowel movement 24 h after birth. The infant was both breastfed and received anti-constipation formula from birth to 6 months of age, and then was breastfed until 12 months. Wheat was introduced to her diet after one year of age, a decision made by her mother. She presented with normal psychomotor development.

At the age of 15 months, the patient presented with constipation (no bowel movements for up to 2 weeks), vomiting, abdominal pain, and bloating. She received prokinetics, laxatives, and enemas, with no improvement. Physical examination revealed skin pallor, a painful and distended abdomen with a perimeter of 48 cm, liver 3-5-5 cm under the costal margin, and fecal impaction in the entire colon. Digital rectal exam found adequate sphincter tone and hard feces. The patient's weight was 10.6 kg, her height was 79 cm, and her brachial perimeter was 13 cm. A barium enema showed abundant feces in the descending colon, and no transition zone. Polyethylene glycol at 1.5 g/kg/day was started.

At the follow-up at 22 months of age, the patient continued to be constipated and dependent on up to 2.5 g/kg/day of polyethylene glycol. Her stools were classified as type 2 on the Bristol stool scale, and treatment adherence was poor, with intermittent suspensions. She presented with increased abdominal pain and bloating, gastro-biliary vomiting, and a generalized rash that remitted in 24-48 h and was not associated with food intake. Physical examination showed abdominal distension, liver 4-6-6 cm under the costal margin, dermatosis extending to the axillas, and the inguinal region characterized by erythema and eczema. The patient's weight was 12.9 kg, her height was 85 cm, and her brachial perimeter was 15 cm. She was well-nourished at the follow-up, but her weight was not reliable due to hepatomegaly.

☆ Please cite this article as: Espriu-Ramírez MX, Rivera-Suazo Y, Valencia-Mayoral PF. Enfermedad celiaca seronegativa: para encontrarla, hay que buscarla. Reporte de caso pediátrico. Revista de Gastroenterología de México 2021;86:317–319.