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O. Montoya-Montoya[a,b,*], S. Palomino Ayala[a,b,c], M.O. Santana Montes[a,b]

[a] *Departamento de Medicina Interna, Hospital General De Occidente, Zapopan, Mexico*
[b] *Universidad de Guadalajara, Guadalajara, Mexico*
[c] *Departamento de Gastroenterología y Endoscopia, Hospital General De Occidente, Zapopan, Mexico*

* Corresponding author. Hospital General de Occidente, Av. Zoquipan No. 1050, Colonia Seatle, C.P. 45170, Zapopan, Jalisco, Mexico. Tel.: +52 (33) 14668298
E-mail address: oliviamontoyam@gmail.com (O. Montoya-Montoya).

Ulcerative proctitis associated with lymphogranuloma venereum☆

Proctitis ulcerada asociada a linfogranuloma venéreo

The differential diagnosis of proctitis in men who have sex with men (MSM) tends to be difficult, given that it includes numerous infectious, inflammatory, and even traumatic causes. Lymphogranuloma venereum (LGV) is a sexually transmitted disease caused by *Chlamydia trachomatis* (*C. trachomatis*). It usually manifests first as an ulcerated, painless papule in the genitals, then as inguinal lymphadenopathy, and finally as distal proctitis.[1] In relation to late diagnosis, disease progression can result in severe complications, such as rectal stricture, obstruction, and perforation.[1,2]

We present herein a 35-year-old patient, with a history of HIV diagnosed in 2012 in relation to Epstein Barr-associated meningitis, currently treated with highly effective antiretroviral therapy with raltegravir 400 mg and tenofovir/emtricitabine 300/200 mg. He had a CD4+ lymphocyte count of 248 cells and an undetectable viral load, and in addition, was identified as an asymptomatic carrier of hepatitis B infection.

He was admitted to the hospital due to clinical symptoms of intense pain in the rectoanal region of 3-month progression, painful defecation, straining, and tenesmus, associated with frequent episodes of rectal bleeding. In the systems review, the patient stated having occasional fever peaks, asthenia, adynamia, hyporexia, myalgias, and arthralgias.

Upon physical examination, the presence of pain in the hypogastrium, with no peritoneal irritation, stood out. The perianal evaluation revealed a deep posterior anal fissure, with marked edema of the anal canal. No adenopathies were palpated in the inguinal region, nor were there lesions on the skin. Due to the patient's medical history, coinfection with other sexually transmitted diseases or opportunistic infections was ruled out. A VDRL test and IgM for Epstein-Barr virus were ordered, along with rectosigmoidoscopy, to evaluate the mucosa and anal canal and take biopsies.

The rectosigmoidoscopy revealed severe inflammatory changes and deep inflammatory ulcers with irregular edges that compromised the middle and distal rectum, with anal canal involvement (Fig. 1A-C). Biopsies were taken to identify the causal agent. Included in the pathology study was abundant lymphoplasmacytic infiltrate of the mucosa, with no viral cytopathic changes, with atrophy, and no dysplasia. Direct testing with techniques for mycobacteria, cytomegalovirus, and fungi was negative, as were the Thayer-Martin agar for *Neisseria gonorrhoeae* infection and the PCR for fungi and mycobacteria, and so PCR in *C. trachomatis* tissue was ordered. The VDRL serologic test for syphilis was reactive at 16 dilutions. Thus, in addition to treatment with 100 mg, every 12 h, of oral doxycycline, 2.4 million units of benzathine penicillin was administered weekly for 3 weeks.

Two weeks later, the patient was readmitted to the emergency service for abdominal pain, with scant rectal bleeding. A computed axial tomography scan and rectosigmoidoscopy were ordered. The first image ruled out perforation and associated collections. The rectosigmoidoscopy revealed significant improvement of the inflammatory changes, as well as ulcers in the process of healing (Fig. 2A-C). After symptom control, the patient was released and completed the treatment with doxycycline in 21 days.

Infectious proctitis in MSM, especially those with a history of HIV, is varied. The most frequent pathogens are *Neisseria gonorrhoeae*, *C. trachomatis*, the herpes simplex virus, and *Treponema pallidum*.[2,3] In an Australian study, differences in the prevalence of the causal agents of infectious proctitis in MSM were found, according to their immune status.[3] The most frequent causal agent was the herpes simplex virus in men that had a history of HIV infection, whereas LGV was the most frequent in men that were HIV-negative. No statistically significant differences related to HIV status regarding symptoms were found in that study. LGV procti-

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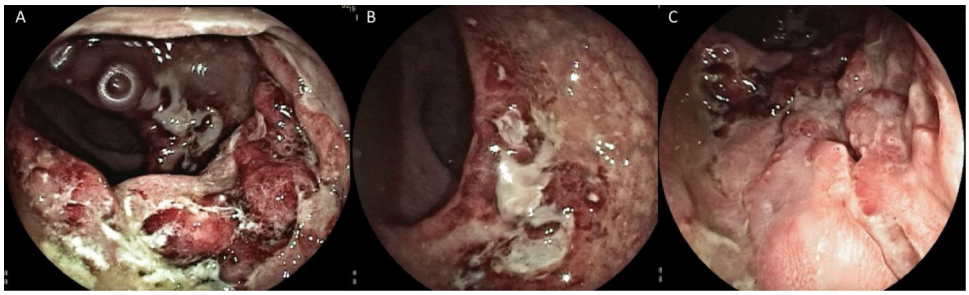

Figure 1 A) Rectosigmoidoscopy showing the severe inflammatory changes on the first and second Houston's valves: marked edema, erythema, and deep, fibrin-covered ulcer. B) Severe inflammatory involvement in the distal rectum, with obvious edema and mucosal thickening. C) Deformity and deep ulcer at the level of the distal rectum, with irregular edges and a sharply demarcated aspect, with anal canal involvement.

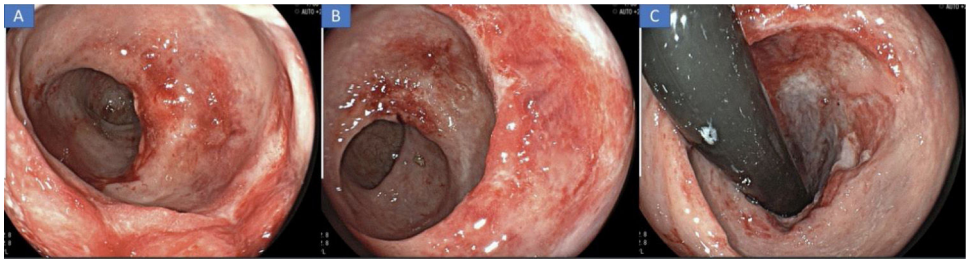

Figure 2 A) Rectosigmoidoscopy showing notable improvement of the inflammatory changes 2 weeks after treatment: first and second Houston's valves. B) Notable improvement of the inflammatory changes in the distal rectum. C) Retroflexion of the rectum, showing the mucosal cicatrization, as well as improvement in the findings close to the anal canal.

tis is characterized by a purulent anal discharge, straining, tenesmus, painful defecation, and altered bowel habit. On occasion, there can be fever, general malaise, weight loss, rectal bleeding, or hematochezia. In a Spanish study that analyzed anorectal manifestations in patients with sexually transmitted diseases, the most frequent symptoms were anal pain, painful defecation, purulent anorectal secretion, straining, tenesmus and/or rectal bleeding.[4] Those authors found that LGV was present in 74% of the patients that had anorectal symptoms lasting more than 1 month, and in all the patients that had documented proctitis associated with rectal ulcers.[4] Three stages of LGV are recognized: the first is characterized by the presence of painless or painful ulcers at the site of contagion that can last up to 4 weeks; in the second stage, lymphadenopathies and abscesses are formed; and in the third stage, if there has not been adequate treatment, the infection advances to include severe complications, such as fistulas, infertility, elephantiasis, or stricture.[1]

Endoscopic study findings range from mild inflammatory changes, deep ulcers with elevated edges and sharply demarcated morphology, and a frequently observed fibrinoid and/or mucopurulent exudate, to stricture and the appearance of tumors.[5–8] Those findings can be indistinguishable from inflammatory bowel disease, adenocarcinoma, or rectal lymphoma. The most frequent biopsy findings are granulation tissue with lymphoplasmacytic infiltrates and fibrosis, which are signs of nonspecific proctitis. Endoscopic findings are not specific, thus there must be a high degree of clinical suspicion in MSM that present with ulcerated proctitis, to be complemented with nucleic acid amplification through PCR testing from secretions or samples of affected tissue, even in the presence of other sexually transmitted diseases.[9]

Ethical considerations

The present work complies with the current bioethical research norms and was approved by the institutional ethics committee.

Data confidentiality. Written informed consent was not requested, given that the data were carefully protected. There are no clinical history or imaging data that allow the patient of the clinical case to be identified.

Right to privacy and informed consent. The authors declare that the present article contains no personal information that could identify the patient.

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Conflict of interest

The authors declare that there is no conflict of interest.

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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).

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

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