A.J. Chaar[a,*], J.S. Arbelaez[b], M.J. Vargas[b], R.A. Cañadas[a]

[a] *Departamento de Gastroenterología y Endoscopia, Hospital Universitario San Ignacio, Pontificia Universidad Javeriana, Bogotá, Colombia*
[b] *Departamento de Patología Clínica, Hospital Universitario San Ignacio, Pontificia Universidad Javeriana, Bogotá, Colombia*

*Corresponding author at: Unidad de Gastroenterología y Endoscopia, Área de Fisiología Digestiva. Hospital Universitario San Ignacio. Pontificia Universidad Javeriana. Código Postal 11001. Bogotá, Colombia. Tel.: (57) 3043809979.
E-mail address: chaarha@javeriana.edu.co (A.J. Chaar).

Esophageal Dieulafoy's lesion treated with band ligation: Individualized treatment for an atypical presentation☆

Enfermedad de Dieulafoy esofágica tratada con ligadura por banda: tratamiento individualizado para una presentación atípica

Dieulafoy's lesion is a cause of gastrointestinal bleeding secondary to the rupture of a feeding artery brought on by multiple extrinsic or intrinsic factors, which can occur 5 cm, distally, from the gastroesophageal junction, the biliary tree, and even at the level of the bronchial wall.[1] Classic Dieulafoy's lesion corresponds to an aberrant submucosal blood vessel that erodes the overlying epithelium, in the absence of a primary ulcer. This occurs because the vessel does not undergo the normal branching within the wall of the stomach, thus its caliber is up to 10-times higher than the normal caliber of the mucosal capillaries (ranging from 1 to 3 mm) and protrudes into the lumen, predisposing to spontaneous ruptures.[2,3] At present, 75 to 90% of the lesions reported in the current literature are located in the proximal stomach and up to 10% of the remaining lesions can present at the esophagus, small bowel, colon, rectum, anus, and the bronchial tree.[4] Esophageal lesions can be a diagnostic challenge, not only because they are rare, their incidence is unknown, and many authors report on them anecdotally, but also because of the anatomic characteristics of the esophagus that makes their visualization difficult.[5] A 48-year-old woman diagnosed with rheumatoid arthritis of 21-year progression and recently diagnosed with type 2 diabetes and primary high blood pressure, was taking methotrexate, leflunomide, and etoricoxib, previously with indomethacin/betamethasone/ methocarbamol, hydroxychloroquine, and deflazacort. The patient came to our medical unit, after presenting with hematemesis and melena of 24 h progression. At the emergency room, her vital signs were: heart rate 90 bpm, respiratory rate 16 breaths per minute, blood pressure 110/70 mmHg, pulse oximetry 96%, and temperature 36.4 °C. Initial laboratory test results were: hemoglobin 13.37 g/dl, platelets 259,000 cells/mcl, leukocytes 7,500 cells/mcl, INR 1.0, activated thromboplastin time 27 s, glucose 423 mg/dl, creatinine 0.77 mg/dl, blood urea nitrogen 19 mg/dl, AST 20 U/l, ALT 34 U/l, LDH 195 U/l, GGT 42 U/l, lipase 45 U/l; venous gasometry with pH 7.37, CO_2 40 mmHg, O_2 35 mmHg, and HCO_3 24 mEq/l. Later, during her hospital stay, the patient had 3 episodes of melena associated with a decrease in hemoglobin of 2.9 g/dl and signs of low blood pressure. Upper GI endoscopy revealed a visible vessel in the esophagus 32 cm from the upper dental arcade (UDA) that merited band ligation (Fig. 1a and b) and showed an 8 mm ulcer at the gastroesophageal junction (34 cm from the UDA), with a fibrin base and no visible vessel. Esophageal Dieulafoy's lesion and a Forrest III ulcer at the gastroesophageal junction were diagnosed.

At the end of the procedure, the patient's hemodynamic status continued to be monitored, with no eventualities, and 20 mg of oral levo-pantoprazole every 12 h was started. The patient was discharged 48 h later, continuing with levo-pantoprazole and 60 mg of domperidone every 24 h. Three months after her discharge, there was no new evidence of bleeding at the gastroenterology control follow-up appointment, and so the patient was definitively discharged.

As stated above, esophageal Dieulafoy's lesion is exceptional, and as a result, experience with its approach is minimal. Soetikno et al. reported the first case of Dieulafoy's lesion treated with band ligation.[6] Guerrero-Garcia et al. described the case of a woman in the seventh decade of life who presented with Dieulafoy's lesion. She was initially treated with an injection of 6 cm^3 of adrenaline at a 1:10.000 dilution, followed by the application of 2 Hemoclips®. Those authors emphasized the difficulty in initially visualizing the lesion, as well as the potential risk for perforation with the adrenaline/clip technique.[4] Inayat et al. published a case series on 3 patients with esophageal Dieu-

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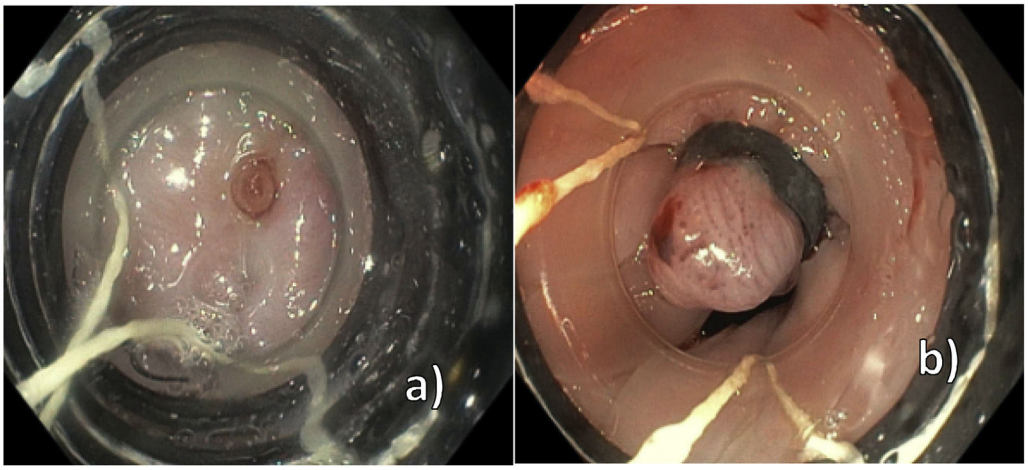

Figure 1 Upper GI endoscopy. A): Visualization of the vessel 32 cm from the superior dentate arcade. B): Successful ligation of the visible esophageal vessel.

lafoy's lesion, all of whom were treated with Hemoclips®, with no new bleeding evidence at follow-up.[7] After Soetikno's article was published, another report of a case of esophageal Dieulafoy's lesion appeared, in which the patient was treated with band ligation, after presenting with two episodes of rebleeding following the use of adrenaline and clips.[8] Band ligation could be a safe alternative for treating this uncommon disease, based on the existing evidence from its use in other anatomic presentations of Dieulafoy's lesion.[9]

Ethical considerations

Informed consent was requested from the patient to receive the treatment described herein. Authorization by a bioethics committee was not required because this is a case report utilizing a previously documented treatment. The article contains no information that could identify the patient, preserving his/her anonymity.

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Conflict of interest

The authors declare that there is no conflict of interest.

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I. Bartnicki-Navarrete[a], J.A. Garay-Ortega[b,*], L.F. Robles-Fernandes[c]

[a] *Departamento de Endoscopia, Centro Médico Nacional Siglo XXI, Mexico City, Mexico*
[b] *Departamento de Gastroenterología, Centro Médico Nacional Siglo XXI, Mexico City, Mexico*
[c] *Departamento de Medicina Interna, Centro Médico Nacional Siglo XXI, Mexico City, Mexico*

*Corresponding author at: Av. Cuauhtémoc 330, Doctores, Cuauhtémoc, 06720 Mexico City, CDMX.
E-mail address: jorge.garay11@hotmail.com
(J.A. Garay-Ortega).

Chronic bowel obstruction secondary to MALT lymphoma☆

Oclusión intestinal crónica secundaria a linfoma tipo MALT

In healthy adults, mucosa-associated lymphoid tissue (MALT) accounts for 80% of all immune cells of the body. MALT has 3 functions: to protect mucous membranes against pathogens; to prevent the uptake of antigens in food, commensal microorganisms, and airborne matter; and to prevent a pathologic immune response to external antigens if they cross the mucosal barrier.[1,2]

MALT lymphoma is a marginal zone-type indolent B cell non-Hodgkin lymphoma (NHL) There are 3 main types of marginal zone lymphomas (MZLs): splenic MZL, extranodal MZL of MALT, and nodal MZL. MALT lymphoma belongs to the extranodal MZL group. Biopsies of MALT lymphoma show perivascular and parafollicular infiltration by atypical monocytoid lymphocytes with folded nuclear edges that are positive for CD19, CD20, and CD79a, but negative for CD5, mainly, as well as CD10.[2]

This disease is associated with White advanced-age (>60 years) populations and patients present with lymphocytosis, with or without cytopenia and splenomegaly. They usually present with lymphadenopathy and can have fever, night sweats, and weight loss greater than 10% of their previous total body weight (known as B symptoms).[3]

A 66-year-old woman sought medical attention, due to colicky pain that increased during food intake. She presented with intake intolerance of 6-month progression and immediate postprandial pain, with nausea and occasional vomiting. After vomiting she had pain that was accompanied by the sensation of a hard abdominal mass. She showed no signs of bowel obstruction. A relevant fact of her medical history was that she had a second-degree relative with Hodgkin's lymphoma. Physical examination revealed a soft depressible abdomen, increased peristalsis upon superficial palpation, and increased volume in the left hypochondrium. The rest of the physical examination was normal.

The patient had previously undergone colonoscopy for gastrointestinal symptoms and was diagnosed with lymphocytic colitis. Laboratory test results reported carcinoembryonic antigen 1.45 ng/mL and CA 19−9 2.5 U/mL.

Preoperative complete blood count and blood chemistry analyses, as well as coagulation tests, were within normal limits, with adequate controls.

The following laboratory work-up results stood out: glucose 92 mg/dl, blood urea nitrogen 7.9 mg/dl, creatinine 0.5 mg/dl, blood urea nitrogen/creatinine ratio 15.8, total cholesterol 136 mg/dl, and triglycerides 67 mg/dl.

Contrast and non-contrast computed tomography (CT) of the abdomen and pelvis, with axial views and multiplanar reformation, were carried out (Fig. 1).

The CT scans identified small bowel segments distended with air and neutral fluid. There was homogeneously enhanced concentric wall thickening (with no stratification pattern) up to 21 mm thick at the level of the distal ileum, which conditioned a narrowing of approximately 70% of the lumen, causing retrograde dilation of the ileum. Air and residual material were observed in the colon.

The thickening of the distal ileum walls ended with a retrograde obstructive defect and suspicious data of hepatic and para-aortic retroperitoneal lymph node metastatic activity, suggesting a carcinoid tumor. Exploratory laparotomy revealed a small bowel tumor at 210 cm from the angle of Treitz and 160 cm from the ileocecal valve that obstructed 80% of the intestinal lumen, causing wall thickening up to 10 cm. Intestinal resection with a 10 cm proximal and distal extension of healthy tissue was performed, along with an end-to-end intestinal anastomosis. Liver examination identified smooth edges and no superficial or deep tumors were palpated. Given the absence of bulges or macroscopic alterations, retroperitoneal exploration was not performed.

The diagnosis was extranodal marginal zone lymphoma, clinical stage II Be, low-risk MALT-IPI. The results of the complementary immunohistochemical tests were:

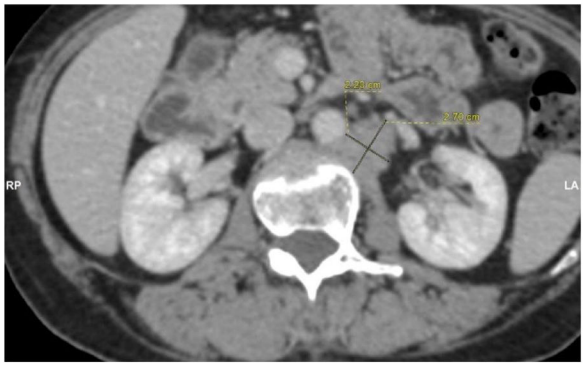

Figure 1 Computed axial tomography scan showing a left-sided 2.40 × 2.70 cm mass in the ileum.

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