Ethical disclosures

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this study.

Confidentiality of data. The authors declare that they have treated all patient data with confidentiality and anonymity, following the protocols of their work center.

Right to privacy and informed consent. The authors have followed the protocols of their work center in relation to the publication of patient data, preserving patient confidentiality and anonymity.

Financial disclosure

No financial support was received in relation to this study/article.

Conflict of interest

The authors declare that there is no conflict of interest.

References

1. Azar AR, Aabraham C, Coulier B, et al. Ileocecal herniation through the foramen of Winslow: MDCT diagnosis. Abdom Imaging. 2010;35:574–7.
2. Martin LC, Merkle EM, Thompson WM. Review of internal hernias: radiographic and clinical findings. AJR Am J Roentgenol. 2006;186:703–17.
3. Osvaldt AB, Mossman DF, Bersch VP, et al. Intestinal obstruction caused by foramen of Winslow hernia. Am J Surg. 2008;196:242–4.
4. Evrard V, Vielle G, Buyck A, et al. Herniation through the foramen of Winslow. Report of two cases. Dis Colon Rectum. 1996;39:1055–7.
5. Armstrong O, Hamel A, Grignon B, et al. Internal hernias: anatomical basis and clinical relevance. Surg Radiol Anat. 2007;29:333–7.
6. Antao B, Hamill J, Samuel M, et al. Herniation through the foramen of Winslow presenting as obstructive jaundice. Pediatr Surg Int. 2005;21:560–2.
7. González-Conde R, Pardo-Rojas P, Valeira-Domínguez E, et al. Correct preoperative diagnosis of herniation through the Foramen of Winslow: two case reports. Hernia. 2013;17:409–14.
8. Takeyama N, Gokan T, Ohgiya Y, et al. CT of internal hernias. Radiographics. 2005;25:998–1015.
9. Sikiminywa-Kambale P, Anaye A, Roulet D, et al. Internal hernia through the foramen of Winslow: a diagnosis to consider in moderate epigastric pain. J Surg Case Rep. 2014;6:1–3.
10. Van Daele E, Poortmans M, Vierendeels T, et al. Herniation through the foramen of Winslow: a laparoscopic approach. Hernia. 2011;15:447–9.

F.E. Bautista-Álvarez[a], R.H. Pérez-Soto[b,*], U. Clemente-Gutiérrez[b], A.C. Hernández-Villegas[c], M. Sierra-Salazar[b]

[a] *Departamento de Cirugía, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Mexico City, Mexico*
[b] *Servicio de Cirugía Endocrina y Laparoscopia Avanzada, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Mexico City, Mexico*
[c] *Departamento de Radiología e Imagen «Dr. Adan Pitol Croda», Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Mexico City, Mexico*

[*] Corresponding author at: Departamento de Cirugía, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Vasco de Quiroga 15, Colonia Sección XVI, Tlalpan, Código postal 14000, Mexico City, Mexico.
E-mail address: rafael.perez.dr@gmail.com (R.H. Pérez-Soto).

2255-534X/ © 2019 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/).

Cutaneous metastases as a manifestation of esophageal adenocarcinoma recurrence: A case report[☆]

Informe de caso: metástasis cutáneas como manifestación de recaída de un adenocarcinoma esofágico

A 49-year-old man with a past medical history of alcoholism and the diagnosis of adenocarcinoma of the distal esophagus was seen at the outpatient dermatology service. He had undergone an initial CAT scan of the chest and abdomen for staging, which showed no evidence of metastases. He then underwent transhiatal esophagectomy and the pathology study reported moderately differentiated adenocarcinoma at stage pT3N1 (1 out of 9 lymph nodes) M0. Following the surgery, adjuvant chemoradiotherapy was begun, utilizing the modified McDonald regimen (5 FU/LV). After the third chemotherapy cycle (6 months after surgery) the patient presented with a permanent lesion on his left cheek that became red, formed a scab, and bled upon removal of the scab, which was why the patient presented at our service. Upon physical examination of the left cheek, a shiny, raised nodule was palpated and its center was covered by a sanguineous crust measuring approximately 1 cm (Fig. 1). The lesion was biopsied and metastatic adenocarcinoma was reported (Fig. 2). After said diagnosis, the patient presented with dorsal pain. A thoracic spine magnetic resonance imaging scan was performed that identified bone metastasis. A new cycle of radiotherapy and second-line chemotherapy with cisplatin and 5FU were begun. We presented his case

[☆] Please cite this article as: Hania AC, Nuñeza E, Cuellarb I, Mejíac M, Ávila F. Informe de caso: metástasis cutáneas como manifestación de recaída de un adenocarcinoma esofágico. Revista de Gastroenterología de México. 2019. https://doi.org/10.1016/j.rgmx.2019.06.002

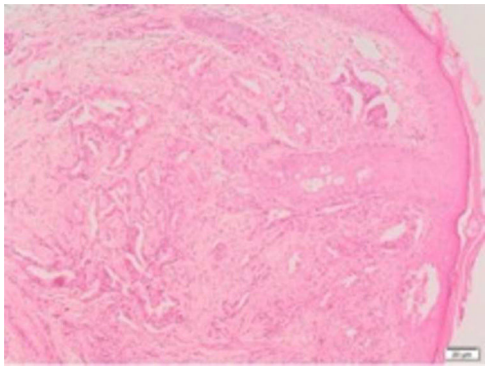

Fig. 1 Shiny raised nodule with center covered by a sanguineous micro-crust measuring approximately 1 cm.

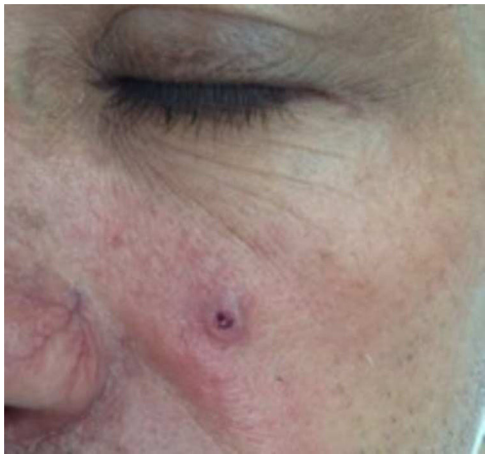

Fig. 2 Histologic section of the skin, showing tumor cell infiltration of the dermis.

due to the rareness of esophageal adenocarcinoma relapse manifesting in the skin.

Esophageal cancer is an uncommon neoplasia, with a high mortality rate. The 2012 GLOBOCAN project estimated 455,784 cases of cancer of the esophagus and 400,169 deaths caused by the disease.[1] Statistics are similar in Colombia, with the latest registers of 1-year survival reported at 37.2 %, decreasing to 20 % for 5-year survival.[1] Currently, 2 types of primary esophageal tumors are identified: squamous cell carcinoma and adenocarcinoma. The majority of esophageal adenocarcinomas develop in or close to the gastroesophageal junction and arise from Barrett's esophagus, which is a complication of chronic gastroesophageal reflux disease. Regarding metastases from the 2 subtypes, neighboring structures, such as the tracheobronchial tree, aorta, pericardium, and the recurrent laryngeal nerve are compromised. The liver and brain can be sites of distant metastasis, but metastasis to the skin is rare.[2] The majority of reports of metastasis to the skin in the literature are case presentations. Quint et al. estimated that the incidence of all cutaneous metastases originated from esophageal carcinomas, and 1 % were from adenocarcinomas and squamous cell carcinomas of the esophagus.[3] It is important to keep in mind that the presence of cutaneous metastases in any type of cancer is a poor outcome factor, given that survival rates have been reported at 4.7 months.[4] The clinical picture of those patients is often asymptomatic, and therefore requires a high degree of suspicion. The most frequent forms of presentation are papules, neoplastic alopecia, indurated erythematous plaques, or cutaneous nodules.[5] In the majority of cases, cutaneous metastases are secondary to primary tumors located in the lower third of the esophagus, and have been identified as both the first finding of disease and disease progression in patients with no previous metastatic involvement.[6] The case presented herein is important because the first manifestation of disease progression was in the skin. All patients with lesions that are suspected to be metastatic should undergo a dermatologic evaluation and adequate histopathologic study, given that the differential diagnoses range from benign lesions to primary neoplastic lesions of the skin.

Ethical disclosures

The publication of the present article was authorized by the ethics committee of the *Hospital Universitario San Ignacio* and a written statement of informed consent was signed by the patient authorizing the use of photographs in the article.

Financial disclosure

No financial support was received in relation to the present article.

Conflict of interest

The authors declare that there is no conflict of interest.

References

[1] Ferlay J, Soerjomataram I, Dikshit R, et al. Cancer incidence and mortality worldwide: sources, methods and major patterns in GLOBOCAN 2012. Int J Cancer. 2015;136:E359–86.
[2] Riley S, Wah T, et al. Cutaneous metastasis of esophageal adenocarcinoma with an unusual presentation. J Clin Ultrasound. 2007;35:289–92.
[3] Quint LE, Hepburn LM, Francis IR, et al. Incidence and distribution of distant metastases from newly diagnosed esophageal carcinoma. Cancer. 1995;76:1120–5.
[4] Lookingbill DP, Spangler N, Helm KF. Cutaneous metastases in patients with metastatic carcinoma: a retrospective study of 4020 patients. J Am Acad Dermatol. 1993;29:228–36.
[5] Doumit G, Abouhassan W, Piliang MP, et al. Scalp metastasis from esophageal adenocarcinoma. Ann Plast Surg. 2013;71:60–2.
[6] Ashutosh C, Sharma AK, Sunita BS. · Cutaneous metastasis as primary presentation in unsuspected carcinoma esophagus: Report of two cases. J Cancer Res Ther. 2015;11:667.

A.C. Hani[a], E. Nuñez[a], I. Cuellar[b], M. Mejía[c], F. Ávila[a,*]

[a] *Departamento de Gastroenterología y Endoscopia, Hospital Universitario San Ignacio, Bogotá, Colombia*
[b] *Departamento de Dermatología, Hospital Universitario San Ignacio, Bogotá, Colombia*
[c] *Departamento de Patología, Hospital Universitario San Ignacio, Bogotá, Colombia*

* Corresponding author.
E-mail address: fredyavila87@gmail.com (F. Ávila).

16 February 2019 24 June 2019

2255-534X/ © 2019 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/).

Cerebral venous sinus thrombosis in a pediatric patient with inflammatory bowel disease: A case report☆

Trombosis del seno venoso sagital superior en paciente pediátrico con enfermedad inflamatoria intestinal: reporte de caso

Thromboembolic events (TEEs) have been documented in patients with inflammatory bowel disease (IBD). Incidence in hospitalized pediatric patients with IBD is 117.9/10,000, with a relative risk of 2.36 (95% confidence interval: 2.15-2.58). An estimated 1.3 to 6.4% of adults and 3.3% of children with IBD develop cerebrovascular complications during the course of the disease, and they are more frequent during exacerbations.[1–3]

A male child seen at 2 years and 7 months of age, with no history of IBD or autoimmune disease, presented with lower gaastrointestinal bleeding, diarrhea, and nocturnal bowel movements starting at 14 months of age. Infections, allergies, and primary and secondary immunodeficiency were ruled out as causes. ANCA and ASCA antibodies were negative. Colonoscopy revealed a hyperemic, friable, and nodular cecal mucosa, as well as micro-ulcers located predominantly in the sigmoid colon and rectum. The histology study was consistent with IBD and immunohistochemistry was negative for Epstein-Barr virus and cytomegalovirus. The child was treated with 2 mg/kg/day of prednisone, 1 mg/kg/day of azathioprine, and due to failed remission, infliximab at a dose of 5 mg/kg.

At one month of hospitalization, the patient presented with two events of tonic-clonic focal onset motor seizures, located in the left hemibody and lasting for 1 min, without altering consciousness. Neurologic examination revealed normal cranial pairs, preserved muscle tone, 4/5 overall muscle strength, right ++/++++ left +++/++++ superior muscle stretch reflex, inferior muscles with bilateral exhaustible clonus, normal superficial sensitivity, negative Babinski reflex, and negative cerebellar, meningeal, and neurocutaneous signs. Table 1 shows the laboratory results at the time of the event. Computed axial tomography and nuclear magnetic resonance imaging of the head identified venous sinus thrombosis in the entire tract of the superior sagittal sinus and in two confluent veins of the frontal region, bilaterally. The cerebral parenchyma had zones of venous infarct in the frontal and left parietal-occipital regions, as well (Fig. 1). Normal homozygous MTHFR A1298C, heterozygous MTHFR C677 T, homozygous prothrombin G20210A, and homozygous Leiden factor V G1691A results ruled out primary thrombophilia, as did normal homocysteine, C protein, S protein, and antithrombin III values. An echocardiogram showed no images suggestive of thrombi or vegetations in the large vessels, valves, or cardiac cavities.

Treatment began with 2 mg/kg/day of subcutaneous enoxaparin and 20 mg/kg/day of levetiracetam but was suspended after 10 days due to increased disease activity and active bleeding.

The patient was released four months later, completely recovered and with no neurologic sequelae. A specific speech disorder was detected at outpatient follow-up.

Ever since the reported association between cerebral venous thrombosis and ulcerative colitis, pediatric case series have been documented that report the superior sagittal sinus as the most common site of venous thrombosis in the brain.[4,5]

Thrombosis pathophysiology in IBD is multifactorial: thrombocytosis/platelet activation, hyperhomocysteinemia, high fibrinogen levels, impaired fibrinolysis, autoantibodies, increased procoagulation factors, decreased anticoagulation factors, and procoagulation mutations.[6] Coagulation activity in IBD is related to the activity and colonic extension of the disease.[7] Anemia has been documented in 49% of cases with thrombosis, thrombocytosis in 26%, and no identifiable cause in up to 14%, as could be the case in our patient.[7]

In children with the first TEE, nonactive IBD, and an unrelated reversible triggering factor (immobilization, recent surgery, trauma, oral anticontraceptive use, or catheter), the Canadian Association of Gastroenterology recommends the use of anticoagulant therapy for 3 months, until the risk factor has been resolved for one month. In the context of active disease, the suggestion is to continue treatment until there have been 3 months of remission.[8]

Routine thromboprophylaxis is not routinely recommended for hospitalized children with IBD relapse that have no previous history of thrombosis. However, in a review of pediatric IBD cases, there was a 1% incidence of venous TEEs and the risk factors of older age, central venous catheter, parenteral nutrition, and hypercoagulability were identified, suggesting a possible benefit in relation to primary pharmacologic thromboprophylaxis. However, the possible side effects of intracranial bleeding and increased gastrointestinal bleeding must be kept in mind.[3,6]

Thromboprophylaxis is recommended in patients considered high-risk, such as hospitalized patients, those

☆ Please cite this article as: Rivera-Suazo Y, Argüello Calderon I, Vázquez-Frías R. Trombosis del seno venoso sagital superior en paciente pediátrico con enfermedad inflamatoria intestinal: reporte de caso. Revista de Gastroenterología de México. 2020. https://doi.org/10.1016/j.rgmx.2019.07.007