tologic, immunologic, or clinical findings to suggest the diagnosis. The therapeutic response confirmed the diagnosis.

Until 2017, only 5 cases of postgestational EGE had been reported in the international literature, none of which involved three layers; one of the cases recurred in a second pregnancy.[4] There has been an exponential increase of reports in the past five years, suggesting the possibility that pregnancy could act as a trigger. The predominant maternal immune response during pregnancy is humoral, which is why cell-mediated diseases, such as rheumatoid arthritis, improve during pregnancy, whereas others, such as systemic lupus erythematosus, worsen. This is consistent with a downregulated Th1-mediated immune response and an enhanced Th2-mediated response. Thus, it is possible that these changes during pregnancy caused the symptoms of postgestational EGE in the patient described herein.[6]

Ethical considerations

The authors declare that this article contains no personal information that can identify the patient, preserving her anonymity according to institutional protocol. Informed consent was not requested for the publication of this case because no personal data or images are presented that could identify the patient. This article meets the current bioethical research regulations, and no experiments were conducted on animals or humans. The institutional ethics committee of the *Hospital Universitario del Caribe* in Cartagena, Colombia, authorized the present publication.

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Conflict of interest

The authors declare that there is no conflict of interest.

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Intestinal histoplasmosis in an immunocompetent patient: A case report

Histoplasmosis intestinal en un paciente inmunocompetente: reporte de un caso

Histoplasmosis is an endemic mycosis caused by the *Histoplasma capsulatum* fungus. This fungus is acquired through the inhalation of microconidia and more than 90% of cases are asymptomatic.[1] Symptomatic intestinal involvement is extremely rare and clinical presentation depends on patient age and immunosuppression status, as well as on the size of the inoculum.[2]

A 45-year-old man from Tarapoto, Peru, came to the hospital presenting with diffuse, colicky abdominal pain, bloody diarrhea, diaphoresis, and fever of 38 °C for a period of three weeks. He went to the emergency service for having presented with hematochezia. Laboratory analyses reported the following: leukocytes: 10,000/mm^3, bands: 0%, segmented cells: 88.9%, hemoglobin: 7.8 g/dl, platelets: 346,000/mm^3, ELISA HIV: nonreactive, HTLV I and II: negative. Colonoscopy: multiple ulcers in the ascending colon, transverse colon, descending colon, sigmoid colon, and rectum, with congestive edges and whitish fibrin in the wound bed (Fig. 1a). The pathologic anatomy study of the colonic biopsies showed chronic inflammation and multiple macrophages, with microorganisms in their interior, consistent with histoplasma (Fig. 1b). Gomori staining was positive for mycosis (Fig. 1c). Intravenous liposomal amphotericin B, 3 mg/kg/day, was administered for 2 weeks. The patient had clinical improvement and was discharged. As an outpatient, he continued treatment with itraconazole, 200 mg

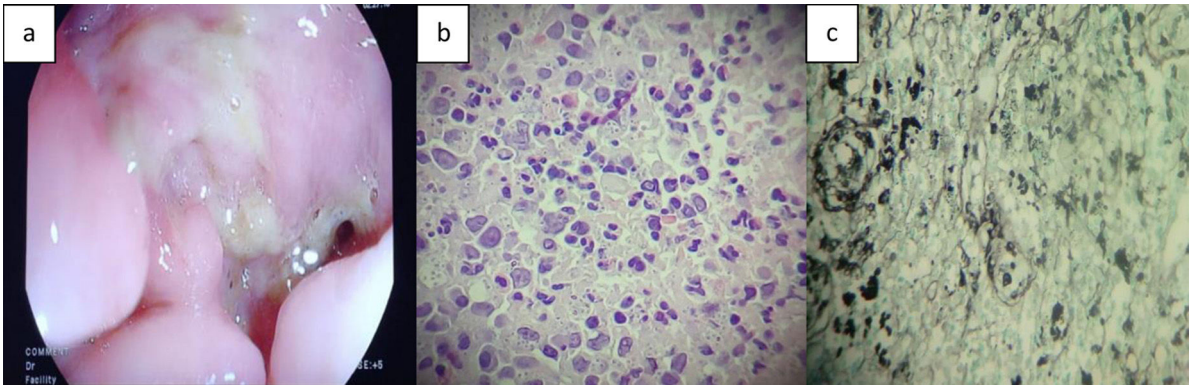

Figure 1 a) the presence of ulcers in the sigmoid colon, b) the pathologic anatomy study with hematoxylin-eosin staining revealed multiple purple, sphere-shaped microorganisms, surrounded by a whitish halo, consistent with histoplasmosis, and c) Gomori staining showed the presence of multiple microorganisms consistent with mycosis.

every 12 hours, for one year. At the one-year follow-up, the patient is asymptomatic.

In Latin America, histoplasmosis is highly endemic, with a prevalence of 32%. This mycosis can manifest from the mouth to the anus; the most frequently affected sites are the ileum and colon.[3] Intestinal manifestations occur in 2-3% of cases and the most frequently reported clinical symptoms are abdominal pain, diarrhea, and fever.[4] Endoscopically, histoplasmosis involving the colon can present as an ulcer or pseudotumor, and there can be complications, such as gastrointestinal bleeding, intestinal obstruction, or intestinal perforation.[5] Diagnosis consists of the presence of microorganisms in the histology study, which in hematoxylin-eosin staining are characteristically round, purple, and surrounded by a whitish halo. Other infections, such as tuberculosis, inflammatory bowel disease, or neoplastic processes, such as lymphoma, should be considered in the differential diagnosis.[6] In disseminated forms, the treatment of choice is 3 mg/kg/day of liposomal amphotericin B for 1-2 weeks, followed by itraconazole 200 mg every 8 hours for 3 days, and then itraconazole 200 mg every 12 hours for 12 months.[7,8]

In conclusion, intestinal histoplasmosis can appear in immunocompetent patients from endemic zones, with the clinical symptoms of abdominal pain, diarrhea, and fever.

Ethical considerations

The authors declare that no experiments were conducted on humans for this research. We utilized the protocols of our work center for obtaining patient databases, preserving patient anonymity (thus informed consent was not requested). This study meets the current bioethical research regulations.

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Jejunal perforation associated with immune reconstitution inflammatory syndrome due to cytomegalovirus: A case report

Perforación en yeyuno asociada a síndrome inflamatorio de reconstitución inmunologica por citomegalovirus: reporte de un caso

Gastrointestinal infection due to cytomegalovirus (CMV) in patients with HIV has not been frequently reported. Its main presentation is perforation at the level of the colon, ileum, and appendix.[1] Therefore, the aim of this work was to present a case of intestinal perforation at the level of the jejunum due to CMV during antiretroviral treatment (ART), as part of immune reconstitution inflammatory syndrome (IRIS).

A 48-year-old man had a past medical history of HIV infection. At the time of diagnosis, he presented with a viral load of 100,000 copies with a count of 47 CD4+ cells, signifying stage C3 disease.

The patient's illness began in November 2021. He presented with asthenia, adynamia, and a productive cough that caused vomiting and dyspnea, but no cyanosis. Symptomatology had persisted for 21 days. He also had dyspnea after small and medium exertion and polypnea. Fourteen days after symptom onset, he presented with blood in sputum and a rise in body temperature accompanied by chills, piloerection, and diaphoresis, for which he sought medical attention at the emergency service of our hospital. The patient was evaluated and admitted to the internal medicine service on November 11, 2021, diagnosed with pneumonia in an immunocompromised patient. ART was started with tenofovir/emtricitabine (1 tablet of lopinavir/ritonavir every 24 hours and 2 tablets every 12 hours, together with trimethoprim-sulfamethoxazole and ceftriaxone). About two weeks after starting treatment, the patient began to have colicky abdominal pain located in the left iliac fossa, with intermittent intensity of 7/10, accompanied by nausea but no vomiting. Physical examination revealed abdominal pain and a soft abdomen upon palpation, rebound tenderness, and abdominal wall rigidity. A chest x-ray showed subdiaphragmatic free air. Pneumoperitoneum was identified in the supramesocolic recesses, predominantly in the right subdiaphragmatic region, with data of intestinal obstruction due to dilation of the stomach. He also presented with segments of the small intestine measuring up to 49 mm and intestinal pneumatosis, and a transition zone at the level of the distal ileum was observed.

The laboratory work-up reported leukocytes $7.1 \times 10^3/\mu L$, hemoglobin 12.4 g/dL, hematocrit 36.7%, platelets $445.0 \times 10^3/\mu L$, neutrophils 95%, absolute neutrophils $6.7 \times 10^3/\mu L$, lymphocytes 4.3%, absolute lymphocytes 0.31, CD4 0.68%, and absolute CD4 6.10 cells/μL.

The patient was taken to the operating room, where 100 mL of intestinal fluid was drained, and perforation at the level of the jejunum, 60 cm from the angle of Treitz in 30% of its lumen, was identified. Friable circular lesions also occupied 20% of the intestinal lumen at 100 cm from the angle of Treitz (Fig. 1). Primary closure was carried out with PDS 4.0 on two planes and samples of the edges of the intestinal perforation were sent to the pathology service of the hospital.

The pathologic analysis identified an extensive cytopathic effect in endothelial cells and macrophages, characteristic of CMV infection that was confirmed through immunohistochemistry and a PCR test (Fig. 2). Treatment was started with ganciclovir for 14 days. The patient had adequate clinical progression and was released in 30 days.

Intestinal perforations due to IRIS associated with CMV infection are not often reported. It affects immunodepressed patients, particularly those with CD4 levels below 50 cells/mm^3, which can cause ulceration, enterocolitis, ischemia, and perforation.[2–7] To the best of our knowledge, the case presented herein is the first to be reported in Mexico. At the time of this writing, only three cases of perforation at the level of the jejunum, including this one, have been reported.[4,5]

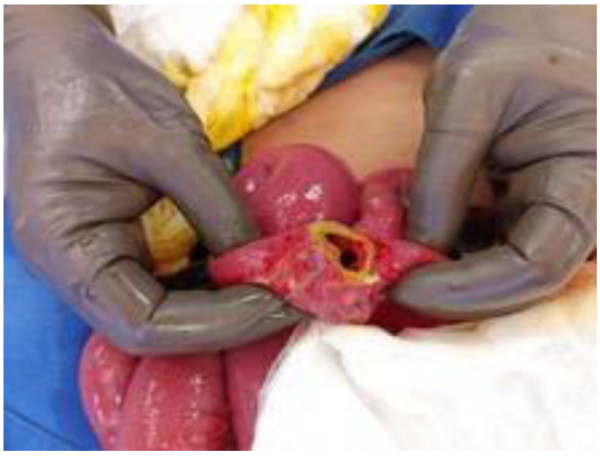

Figure 1 Jejunal perforation 60 cm from the angle of Treitz.