Ethical disclosures

The authors declare they have complied with all the ethical responsibilities regarding data protection, the right to privacy, and informed consent.

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References


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Stricturing ulcerative colitis: a case of rapid disease progression∗

Colitis ulcerosa con estenosis: un caso de progresión rápida de la enfermedad

Ulcerative colitis (UC) is considered an idiopathic disease of the large bowel that consists of chronic mucosal inflammation due to a complex interaction between genetic predisposition and environmental factors. Worldwide incidence is reported to have plateaued in North America (19.2 per 100,000 person-years) and Europe (24.3 per 100,000 person-years), whereas low incidence regions, specifically in developing countries, appear to have experienced an increase in inflammatory bowel disease (IBD) that is most likely due to industrialization.1 Epidemiologic data on UC are scarce in those countries, as they are in Mexico. In a recently published nationwide cohort study, encompassing more than 15 years (2000-2017), incidence was reported at 0.16 per 100,000 person-years and prevalence at 1.45 per 100,000 person-years for UC in Mexico, revealing a 5.3-fold increase.2 The extent and clinical course of the disease can vary, ranging from rectal involvement to pancolitis in a continuous manner, and the disease is characterized by a relapsing and remitting course. The phenotype at diagnosis in patients with UC is generally split equally between proctitis, left-sided disease, and pancolitis. Both IBD subtypes, namely UC and Crohn’s disease (CD), are chronic diseases consisting of chronic inflammation with subsequent constant tissue repair. CD presents with transmural disease with activation of mesenchymal cells and a subsequent strictureing disease course, whereas fibrosis and scar formation in long-standing UC is usually limited to the mucosa, including pseudopolyposis and bridging fibrosis.3 An increased risk of colorectal cancer has been recognized for UC, in particular, with a cumulative incidence of 2% in 10 years, 8% in 20 years, and 18% in 30 years.5 Those data highlight why neoplasia should be the primary suspicion when a colonic stricture is diagnosed. Nevertheless, in their original articles dating back to 1964, Edwards and Truelove reported benign stric-

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Figure 1  Colonic stricture in colonoscopy, with ulcerated mucosa, spontaneous bleeding, and loss of vascular and haustral patterns. A stricture inhibiting the passage of the colonoscope is identified (arrow).

Figure 2  Dissected macroscopic colon. Both strictures are indicated by the metal forceps, in the transverse colon and descending colon, respectively.

mechanism in UC is most likely caused by thickening of the muscularis mucosae induced by b-FGF-positive inflammatory neutrophils. Currently, the focus of treatment in patients with IBD has incorporated a ‘‘treat to target’’ approach, thereby shifting the emphasis onto endoscopic and histologic targets over clinical or symptomatic ones. Biologic agents have played a pivotal role in preventing long-term complications. Previously, the mean duration from disease onset to stricture formation was reported to be 15.6 ± 8.6 years, and longer disease duration was associated with malignancy. Our patient had severe disease progression, with benign stricture formation at only two years from disease onset. Due to the current lack of disease markers for strictureing UC, the present case underlines the importance of early recognition and aggressive treatment, including patient information and education that, in turn, promotes treatment adherence.

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Verbal informed consent from the patient was obtained for the case report. Written informed consent was not obtained since the present manuscript does not include personal information that could identify the patient.

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References

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