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Right Lower Quadrant Pain in a Patient With Inflammatory Bowel Disease of Undetermined Type

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A 48-year-old man diagnosed with inflammatory bowel disease of undetermined type 6 years earlier presented with sharp nonradiating right lower quadrant pain. He denied fever, chills, nausea, vomiting, diarrhea, or anorexia. The patient had been in clinical remission on azathioprine and mesalamine. Physical examination was significant for right lower quadrant tenderness without rebound or guarding. His white blood cell count and hemoglobin were normal.

A computed tomography scan demonstrated cecal inflammation with associated 5-cm phlegmon (Figure A, *arrow*). The appendix was not visualized. The terminal ileum appeared narrowed at the ileocecal valve. The patient was treated with antibiotics and bowel rest. Colonoscopy revealed a 4-cm nonobstructing mass in the cecum (Figure B). The remainder of the colon was normal without mucosal breaks or friability. Biopsies of the mass revealed normal colonic mucosa with mild inflammation and mild architectural change.

The patient underwent diagnostic laparoscopy revealing an abnormal enlarged appendix. Laparoscopic appendectomy was performed. The surgical specimen measured 5.6 × 4.5 × 3.0 cm and contained no masses. Histologic examination (Figure C; hematoxylin and eosin, bar = 200 μm; Courtesy of W. Sandy Twadell, MD) demonstrated transmural acute and chronic inflammatory infiltrates. Crypt destruction with microabscess formation (*arrowhead*) and architectural distortion in the form of crypt branching (*small arrow*) were present, as were poorly formed granulomas with focal giant cells (*large arrows*). The findings favored a diagnosis of Crohn's disease (CD), changing our patient's diagnosis to Crohn's colitis with appendiceal involvement.

Data from our patient's initial diagnosis were reviewed. Colonoscopy revealed patchy colitis with moderate to severe ulceration. The terminal ileum was not intubated, although small bowel follow through revealed a normal fold pattern in the terminal ileum. Random biopsies demonstrated active colitis and areas of acutely inflamed granulation tissue without granulomas or chronic changes. Colonoscopy performed 1 year after diagnosis demonstrated

pseudopolyps from the ascending to sigmoid colon and few erosions from the transverse to sigmoid colon. Biopsies revealed chronic active colitis without granulomas.

Granulomatous involvement of the appendix is rare and is found in 0.1%–2% of all appendectomy specimens.¹ Involvement of the appendix in patients with CD is as high as 40%²; however, symptomatic appendicitis caused by appendiceal CD is uncommon. Our patient developed appendicitis in the setting of mucosal healing of the colon while on immunosuppressive therapy. The reasons for this are unclear and speculative, but may be caused by persistent inflammation in the submucosa, mucosa, and/or serosa of the appendix resulting in luminal obstruction and appendicitis. It is also possible that lymphoid cells within the appendix served as a focal point for recurrent CD. Finally, we cannot exclude the possibility that our patient coincidentally developed “classic” appendicitis superimposed on histologic CD.

Symptoms of granulomatous disease of the appendix are similar to acute appendicitis and include fever, right lower quadrant pain, nausea, and anorexia. Other causes of appendiceal granulomatous disease include infection (eg, *Yersinia*, *Mycobacterium tuberculosis*, *Campylobacter*), foreign body reaction, appendiceal or cecal diverticulitis, chronic beryllium poisoning, and sarcoidosis.

Our patient did well postoperatively. Azathioprine and mesalamine were continued. He will begin surveillance colonoscopies next year.

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Figure.