**Listeria monocytogenes** sepsis in ulcerative colitis

A 62-year-old man with a history of coronary artery disease and ulcerative colitis of 10-years duration presented with worsening diarrhea, malaise, fever, and chills for 1 week. He had been started on azathioprine 6 weeks previously. Initially his symptoms had improved, but a week prior to presentation he had developed diarrhea. He reported that his wife had experienced diarrhea and fever as well, but that her symptoms had resolved spontaneously. He denied any international travel. He had recently been hospitalized because of new-onset atrial fibrillation, for which he had been commenced on coumarin and was scheduled to undergo elective cardioversion. On physical examination he appeared sick, toxemic, and was drenched in sweat. His temperature was 40°C and his pulse was irregular at a rate of 102 beats per minute. His abdomen was soft but there was tenderness on palpation of the left lower quadrant. His laboratory data showed a leukocytosis of 17×10⁹/L (normal range 4–10×10⁹/L) with left shift (80% neutrophils and 8% band forms, with normal eosinophils and monocytes), an elevated C-reactive protein (CRP) of 12.35 (normal <0.5 mg/dL), an elevated international normalized ratio (INR) of 3.2 (normal < 1.0), and negativity for cytomegalovirus DNA. The remaining laboratory data were unremarkable. A plain abdominal X-ray showed no evidence of megacolon. Blood and stool cultures were obtained. He was started on intravenous fluids, intravenous steroids, and antibiotics (metronidazole andampicillin) and his fever slowly settled. He had been commenced on coumarin and was scheduled to undergo elective cardioversion. On physical examination he appeared sick, toxemic, and was drenched in sweat. His temperature was 40°C and his pulse was irregular at a rate of 102 beats per minute. His abdomen was soft but there was tenderness on palpation of the left lower quadrant. His laboratory data showed a leukocytosis of 17×10⁹/L (normal range 4–10×10⁹/L) with left shift (80% neutrophils and 8% band forms, with normal eosinophils and monocytes), an elevated C-reactive protein (CRP) of 12.35 (normal <0.5 mg/dL), an elevated international normalized ratio (INR) of 3.2 (normal < 1.0), and negativity for cytomegalovirus DNA. The remaining laboratory data were unremarkable. A plain abdominal X-ray showed no evidence of megacolon. Blood and stool cultures were obtained. He was started on intravenous fluids, intravenous steroids, and antibiotics (metronidazole and ciprofloxacin). A sigmoidoscopy revealed circumferential proctosigmoiditis with multiple rounded, irregular, raised erosions with a yellow exudate in the center. Over the next 48 hours, he continued to have fever spikes up to 41°C, but then the blood cultures performed shortly after admission were reported as growing *Listeria monocytogenes*. His antibiotic therapy was immediately changed to ampicillin and his fever slowly settled. He was discharged in a stable condition 2 weeks later.

Common microorganisms that infect patients with ulcerative colitis are salmonella, shigella, and campylobacter [1,2]. Cytomegalovirus has also been associated with flare-ups of inflammatory bowel disease (IBD) not responding to immunosuppressive therapy [3]. Although the bacterium *L. monocytogenes* characteristically infects children, the elderly, alcoholics, and immunosuppressed patients, it has also been very rarely reported in patients with ulcerative colitis [4–6]. In the few reports of *L. monocytogenes* bacteremia, most episodes have occurred after colonoscopy [4,5]. Based on this, it was proposed that *L. monocytogenes* colonizes the colon of patients with IBD and that the bacterium is introduced into the circulation through translocation as a result of the manipulation [4,5]. However, in a large study searching for *L. monocytogenes* in patients with IBD and controls, Chen et al. found no significant colonization with *L. monocytogenes* in IBD patients [7].

Our case report is interesting for several reasons. First, our case adds to the scarce literature on listeriosis in ulcerative colitis. We speculate that the additional initiation of azathioprine further decreased the immune response of our patient. Second, we provide the first endoscopic description of *L. monocytogenes* infection in ulcerative colitis. The roundish, occasionally confluent, slightly raised erosions with a yellow exudate were somewhat atypical for ulcerative colitis. If such lesions are seen during endoscopy, additional histology, stool cultures, and blood cultures should be obtained. Finally, our case demonstrates that the presence of acute diarrhea and an ongoing systemic inflammatory response in a patient with ulcerative colitis should prompt the search for uncommon pathogens.

**References**

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ulcerative colitis patient receiving ACTH. Am J Gastroenterol 1990; 85: 216

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