

was admitted to the intensive care unit, where he required hemodialysis for acute renal failure and treatment for sepsis. He made a complete recovery and was discharged on day 37.

DISCLOSURE

All authors disclosed no financial relationships relevant to this publication.

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Commentary

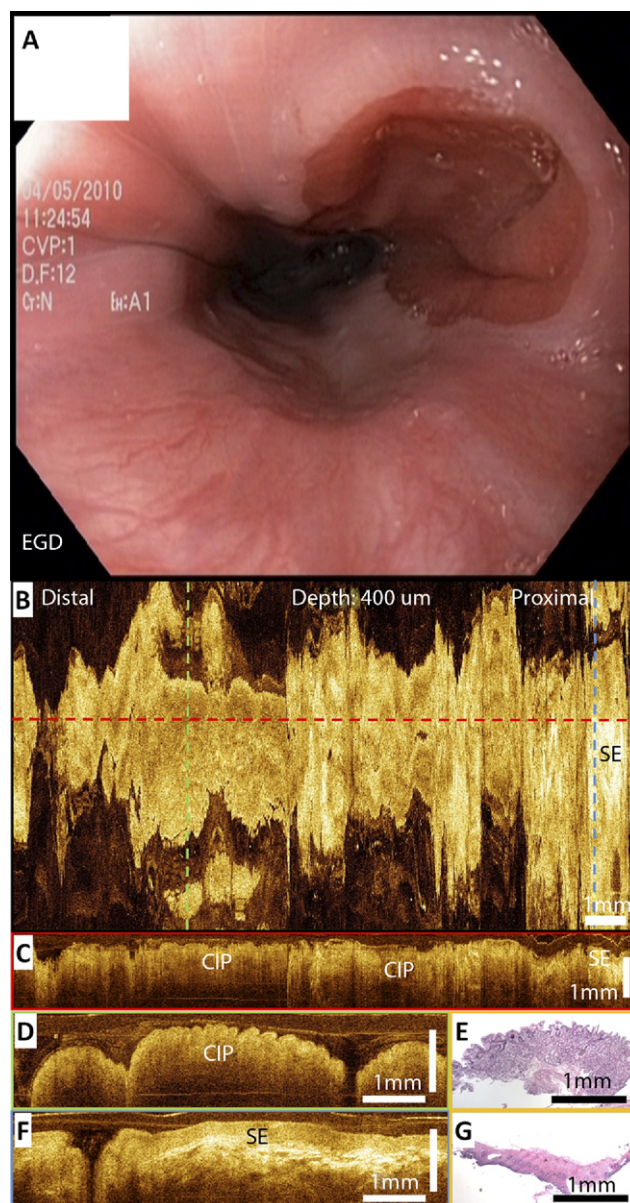
Hemorrhagic colitis in a septuagenarian connotes a differential diagnosis that includes ischemia, medication and allergy, and infection. As a gastroenterologic consultant, I would offer the physicians initially taking care of this patient the counsel that bloody diarrhea is never a manifestation of acute diverticulitis unless complicated by other processes. If the patient had been treated before the onset of bloody diarrhea with a penicillin, for example, amoxicillin-clavulanate, or less commonly a quinolone or cephalosporin antimicrobial, antibiotic-associated hemorrhagic colitis caused by the gram-negative bacterium *Klebsiella oxytoca* would have been a good diagnosis. Until recently, *K oxytoca* was thought to be just a normal commensal of the human intestinal tract. Because it produces β -lactamase, *K oxytoca* resists penicillins and survives to produce a cytotoxin that effects cell damage and death. In contrast to antibiotic-associated colitis of *Clostridium difficile* infection, which typically causes non-bloody diarrhea, exhibits pseudomembranes, is becoming more difficult to treat, and has a high recurrence rate, *K oxytoca* produces hemorrhagic colitis without pseudomembranes, involves the ascending colon in a segmental fashion, and usually resolves completely within a week of discontinuing the precipitating antibiotic. *Escherichia coli* O157:H7 is another cause of hemorrhagic colitis, the result of its production of Shiga-like toxins (Stxs). Stxs, named after Kiyoshi Shiga, who first described the bacterial (*Shigella dysenteriae*) origin of dysentery, cause disease by inhibition of protein synthesis within target cells. Stxs have two subunits, designated A and B. The B subunit binds to globotriaosylceramide on the host cell (Gb3), after which the A subunit enters the cell and is cleaved into two parts, the A1 component of which binds to the ribosome and disrupts protein synthesis. Gb3 is present in greater amounts in renal epithelium, central nervous system neurons, and endothelium; hence, renal toxicity (hemolytic uremic syndrome), neurotoxicity, and vascular occlusion with fibrin thrombi and resultant ischemic-like hemorrhagic colitis. The Centers for Disease Control and Prevention has estimated that close to 85% of *E coli* O157:H7 infections are foodborne in origin, and any food or beverage that becomes contaminated by animal (especially cattle) manure can result in the disease. Meat typically becomes contaminated with *E coli* during the slaughtering process, when the contents of an animal's intestines are allowed to come into contact with the carcass. Foods that have been identified as sources of contamination include ground beef, venison, sausages, dried (non-cooked) salami, unpasteurized milk and cheese, unpasteurized apple juice and cider, orange juice, fruits, nuts, berries, alfalfa and radish sprouts, lettuce, spinach, and water—even pizza and cookie dough. Because *E coli* organisms are mixed throughout the meat during the grinding process and are not just on the surface, ground beef must be cooked throughout to a temperature of 165°F. Symptoms of *E coli* O157:H7 infection usually last about a week and resolve without any long-term problems. Antibiotic treatment does not improve the illness, and some authorities believe that their use increases the risk of developing post-diarrheal hemolytic uremic syndrome. Thus, apart from good supportive care, there is no specific therapy for *E coli* O157:H7 infection. In 1935, Hans Zinsser wrote in *Rats, Lice and History*, "The plant does the work with its roots and its green leaves. The cow eats the plant. Man eats both of them; and bacteria eat the man." How correct he was. Fortunately, this patient escaped death's scythe.

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Three-dimensional endoscopic optical coherence tomography imaging of cervical inlet patch

A 30-year-old white man with established Barrett's esophagus (BE) and continued symptoms of chronic severe heartburn, persistent cough, throat irritation, and asthma was referred for surveillance EGD at the VA Boston Healthcare System. During retraction of the en-

doscope, a pink circular lesion (**A**) was observed under white light endoscopy in the upper esophagus (spanning 20-22 cm from the incisors). Three-dimensional endoscopic optical coherence tomography (OCT) images were obtained of the region under direct visual-



ization with white light by passing the probe through the standard accessory channel. An en face projection image (**B**) at 400- μ m depth underneath the tissue surface showed columnar epithelium consistent with a cervical inlet patch (CIP) and surrounding normal squamous epithelium (SE). Cross-sectional OCT images along the probe pull-back direction (**C**) and the probe rotation direction (**D** and **F**) clearly demonstrated columnar and squamous epithelium in the CIP region and the surrounding esophagus, respectively. Biopsy specimens taken from the imaged lesion confirmed the finding of CIP. The OCT features matched representative hematoxylin and eosin histology (**E** and **G**). Both esophageal and extraesophageal symptoms responded to increased antacid therapy.

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Commentary

Proton pump inhibitor–refractory GERD and BE prompted an EGD. The authors saw CIP, scanned it with OCT, compared it with SE, and submitted this neat FP. But let's look a few microns deeper into this case because what lies beneath (and what lies ahead) may surprise you. Heterotopic gastric mucosa, colloquially known as a cervical inlet patch, is more than a congenital endoscopic curiosity. Although most CIPs never come to the attention of the host (or the hurried endoscopist for that matter), they sure seem to have corrosive potential (odynophagia, dysphagia, and—true story—adenocarcinoma). BE and CIP may share an in utero etiology, and patients with coincident BE (like this one) may be at an increased risk of developing dysplastic Barrett's, both in the proximal and distal esophagus. But let's change gears because the star of the show here is the novel use of OCT. Just as US images are created by the differential acoustic reflectance of various tissue densities, OCT creates its remarkably impressionistic imagery by the differential reflection of light across ultrastructural cellular elements. Although the OCT probe is about the most highly specialized and temperamental device to ever see the inside of an accessory channel, one cannot argue that realizing the holy grail of the "optical biopsy" is close at hand. OCT is one of several experimental endoscopic imaging techniques (like confocal laser endomicroscopy and endocytoscopy) that take aim at our fairly poor ability to identify luminal dysplasia. Whether OCT will ever make it to an endoscopy center near you is less important than the continued pursuit of better ways to see. This case reminds me of the musings of the American author Og Mandino: "I will love the light for it shows me the way, yet I will endure the darkness because it shows me the stars."

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