A 60-year-old man with Crohn colitis controlled with mesalamine was hospitalized for fever, bloody diarrhea, and lower abdominal pain. Two weeks of empiric treatment with metronidazole, ofloxacin, and oral steroids did not result in significant improvement. Sigmoidoscopy revealed inflamed mucosa covered by white membranes, compatible with pseudomembranous colitis (Fig. 1); a stool sample was positive for *Clostridium difficile* toxin.

Steroid treatment was stopped and the patient was treated with intravenous metronidazole and oral vancomycin for 10 days, but there was no clinical improvement. On repeat sigmoidoscopy, the endoscopic picture was compatible with Crohn exacerbation, and no pseudomembranes were seen (Fig. 2).

A repeat stool test was negative for *C. difficile* toxin. Intravenous corticosteroids were initiated and there was rapid clinical resolution of symptoms.

In an inflammatory bowel disease (IBD) patient with gastrointestinal symptoms and a stool test positive for *C. difficile*, three etiologic processes may be considered: IBD flare with mere colonization by *C. difficile*, or superimposed *C. difficile*-associated disease (CDAD) without IBD flare, or perhaps both processes occurring simultaneously [1, 2]. Endoscopy could presumably show different abnormalities in these three conditions, thereby possibly pointing out the dominant inflammatory process.

In our patient, once CDAD was definitely diagnosed, effective antibiotic treatment did not result in clinical improvement, although the pseudomembranes had disappeared at the repeat sigmoidoscopy and the stool test was negative for *C. difficile* toxin. The sigmoidoscopic findings of the subsequent endoscopy suggested an IBD flare, and the patient’s condition stabilized only with the addition of steroid therapy. Thus, we believe this patient’s course illustrates that pseudomembranes can totally mask a simultaneous underlying mucosal inflammation due to ongoing Crohn disease activity. It follows that the finding of *C. difficile* pseudomembranes in patients with a flare-up of IBD cannot be interpreted as evidence that CDAD is the sole inflammatory process responsible for their symptoms, and cannot exclude the coexistence of mucosal inflammation due to underlying IBD activity.

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References


Bibliography


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