A Case of Diarrhoea

A 68-year-old male presented with history of loose stools and lower abdominal pain and cramps of 10 days duration. He had multiple comorbidities which included systemic arterial hypertension, diabetes mellitus and coronary artery disease. He was recently hospitalised for ischaemic stroke and had suffered from hospital-acquired pneumonia (HAP) as well. On examination, he had low-grade fever and minimal lower abdominal tenderness. Haemogram revealed mild anaemia and polymorphonuclear predominant leukocytosis. Biochemical parameters were within normal limits. Sigmoidoscopy revealed multiple yellowish adherent plaques (Figs. 1A and 1B).

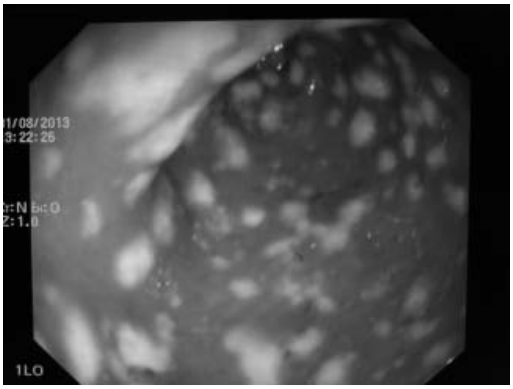

Fig. 1A. Multiple yellowish adherent plaques in the colon.

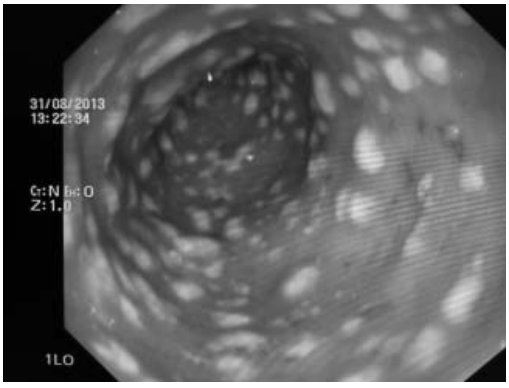

Fig. 1B. Multiple yellowish adherent plaques in the colon.

What is the diagnosis?

A. Ulcerative colitis
B. Lymphocytic colitis
C. Amoebic colitis
D. Pseudomembranous colitis
E. Collagenous colitis

Discussion

Pseudomembranous enterocolitis, first reported in 1893, used to be a rare disorder but ever since the widespread use of antibiotics, it is fast becoming a major complication of antibiotic therapy. Caused by anaerobic gram-positive spore-forming toxigenic bacilli called *Clostridium difficile (C. difficile)*, pseudomembranous enterocolitis is rapidly emerging as a major threat to antibiotic users. Oslon et al has reported that symptomatic *C. difficile* infection (CDI) follows antibiotic use; 96% within previous 14 days and 100% within previous 3 months of onset of diarrhoea.[1] The vast majority of CDI patients remain asymptomatic but symptomatic patients typically present with loose stools. Other less common manifestations include fever and abdominal pain and cramps.[1,2] Overt gastrointestinal (GI) bleeding is exceedingly rare in CDI. When severe, patients may present with ileus or toxic megacolon without diarrhoea.

At sigmoidoscopy, presence of pseudomembranes is virtually pathognomonic of *C. difficile* colitis. Pseudomembranes typically appear as yellowish plaques measuring between 2 mm to 5 mm in diameter. It may coalesce to cover larger areas of mucosa. Histopathology may reveal focal ulceration capped by pseudomembrane — the so-called "volcano" or "summit" lesion. Faecal assays for bacterial toxin remains the mainstay of diagnosis with tissue culture cytotoxicity assay remaining the gold standard for diagnosis.[3] Stool cultures for *C. difficile* or polymerase chain reaction for detection of toxins are also accurate methods for diagnosis.[3]

Treatment options include metronidazole (500 mg thrice daily) or vancomycin (125 mg 4 times daily) administered orally for a duration of between 10 to 14 days. Metronidazole is inexpensive compared to vancomycin and it is mostly

Answer: D

used in treatment of mild to moderate CDI. However, in severe cases, vancomycin remains the drug of choice. Other potential treatment options especially in the setting of recurrent CDI include fidaxomycin, rifaximin, nitazoxanide, cholestyramine, intravenous immunoglobulin and fecal microbiota transplantation.[4]

Our patient was an elderly male with multiple comorbidities. He was recently hospitalised and was on antibiotics for pneumonia. Considering this background and the current presentation, the diagnosis of pseudomembranous colitis was suspected and endoscopy suggested a similar diagnosis. The sigmoidoscopy revealed multiple yellowish plaques typical of pseudomembranous colitis; virtually ruling out all other options mentioned earlier. At endoscopy, the mucosa often appears normal in patients with lymphocytic as well as collagenous colitis and the diagnosis is based on histology.

Ulcerative colitis (UC) is a chronic, often relapsing and remitting, inflammatory colon disorder characterised by bloody diarrhoea. At endoscopy, there will be loss of normal mucosal vascular pattern, mucosal friability, ulcerations and pseudopolyps.

Amoebic colitis presents classically with dysentery, and colonoscopy reveals multiple punctate ulcerations most commonly involving the caecum and ascending colon. Again, stool examination in amoebic colitis may reveal the presence of ova and trophozoites.

Our patient subsequently underwent enzyme immunoassay for *C. difficile* toxins A and B which were reported reactive, confirming the diagnosis. He was treated with oral vancomycin 500 mg 4 times a day for 10 days with which he had symptomatic response.

Conclusion

Pseudomembranous colitis is rapidly emerging as a common complication of antibiotic use; hence a high index of suspicion and low threshold for evaluation will help to clinch the diagnosis and initiate early treatment. With this paper, we intend to introduce all readers to the endoscopic appearance of pseudomembranous colitis.

REFERENCES

1. Olson MM, Shanholtzer CJ, Lee JT Jr, Gerding DN. Ten years of prospective Clostridium difficile associated disease surveillance and treatment at the Minneapolis VA Medical Center, 1982-1991. Infect Control Hosp Epidemiol 1994;15:371-81.
2. Kelly CP, LaMont JT. Clostridium difficile--More difficult than ever. N Engl J Med 2008;359:1932-40.
3. Cohen SH, Gerding DN, Johnson S, Kelly CP, Loo VG, McDonald LC et al. Clinical practice guidelines for Clostridium difficile infection in adults: 2010 update by the society for healthcare epidemiology of America (SHEA) and the infectious diseases society of America (IDSA). Infect Control Hosp Epidemiol 2010;31:431-55.
4. Khanna S, Pardi DS. Clostridium difficile infection: new insights into management. Mayo Clin Proc 2012;87:1106-17.

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