A 75-year old woman with a complex past medical history was found unresponsive in her nursing home room. She was transported to the Emergency Department where attempts at life support were unsuccessful. She had been admitted to the hospital two weeks earlier because of abdominal pain. According to the history, she had been experiencing diarrhea for the week prior to that admission. Her physician had initiated metronidazole for empiric treatment of *Clostridium difficile*, and her medication list also included a proton pump inhibitor and a probiotic. During that hospital stay, metronidazole was discontinued following one week of treatment after a negative *C. difficile* toxin assay was obtained. She was started on ciprofloxacin for a urinary tract infection for an unknown time period and was discharged to her nursing home.

Autopsy revealed bilateral upper lobe thrombo-emboli, mild coronary atherosclerosis and extensive pseudomembranous colitis involving the entire colon and rectum. Grossly, the colonic mucosa was covered with yellow-white plaques measuring up to 0.4 cm in thickness with areas of confluence. (Figure 1A) Microscopically, volcano-like eruptions of pseudomembranes were present over the mucosa (Figure 1B: MM, muscularis mucosae; SM, submucosa; MP, muscularis propria). The pseudomembranes contained mucus, fibrin, epithelial debris and neutrophils. (Figure 1C)

*CLOSTRIDIUM DIFFICILE COLITIS*

*C. difficile* is an anaerobic, gram-positive, spore-forming and highly toxigenic bacterium that has unfortunately become a daily part of medical practice. It is the leading cause of hospi-
tal-associated diarrhea in patients taking antibiotics. Risk factors for this infection include age over 65, the presence of significant co-morbidities, immunocompromised states, inflammatory bowel disease, recent gastrointestinal procedures and the use of gastric-acid suppressants. Management of recurrent C. difficile infections is controversial. Even with appropriate treatment, 20-25% of patients will have a relapsing infection. The infection is diagnosed by detecting components of the organism with the most common testing modality being enzyme immunoassays (EIA). Real-time polymerase chain reaction (PCR) for the C. difficile toxin B gene is becoming the method of choice because of its rapidity and high levels of sensitivity and specificity. Toxin B is necessary and sufficient for virulence. The increased severity of C. difficile infections is related to the ribotype 027 strain which has an attributable mortality of 16.7% and now causes 30-40% of C. difficile infections in North America. This strain has a mutation of the tcdC gene that can lead to 10-20 times more toxin production in in vitro studies.

REFERENCES

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