(*Refer to page 178*)

Answer: Pseudomembranous colitis (PMC)

Pseudomembranous colitis (PMC) was first described in 1893 but it was not until 1935 when the causative organism, then known as *Bacillus difficilis*, was isolated from the stool of a healthy infant. It was renamed *Clostridium difficile* (*C. difficile)* 40 years later, reflecting the difficulty of its culture and isolation. Discovered to be the inciting agent in antibiotic associated diarrhoea, PMC is now the leading cause of hospital acquired illness in developed countries. Its increasing incidence has been associated with a new hypervirulent strain. [1, 2]

C. difficile is an anaerobic, gram-positive, spore-forming bacillus. The spores are difficult to eradicate, being resistant to antibiotics and common sterilization techniques, including heat and acid. In the colon, they convert into their fully functional, vegetative, toxin producing form, becoming susceptible to antibiotics. Two potent exotoxins are released, toxin A and toxin B, which act as an enterotoxin and a cytotoxin respectively, resulting in inflammation and diarrhoea. Spores allow for persistent survival in the intestine, potentially contributing to recurrent disease. [1, 2]

Transmission occurs faecal-orally, with colonisation facilitated by disruption of the normal intestinal flora due to antimicrobial therapy. Other risk factors include advanced age, exposure to healthcare settings or long term care facilities and immunosuppressive agent or acid suppressant use. However in some cases, no risk factors are present as in this case. [1, 3]

C. difficile infection (CDI) can range from asymptomatic carriers to mild presentations of fever and trivial watery diarrhoea, to ileus without diarrhoea and toxic megacolon, to severe fulminant disease presenting as sepsis, multi-organ failure and death. [1,3] Diagnostic tests include identification of the toxins in the stool. Imaging and in particular endoscopy, are good options however, are much less sensitive albeit specific. Findings on radiological imaging include colonic-wall thickening, pericolonic stranding, the "accordion sign", the "double-halo" sign, and ascites. Colonoscopy may reveal "pseudomembranes" which appear as raised yellow or off-white plaques up to 2 cm in diameter, scattered over an inflamed colorectal mucosa. [3]

Treatment initially involves cessation of the inciting antibiotic. Antibiotics of choice for treatment include metronidazole and topical vancomycin. In severe cases, patients may need to be evaluated for a colectomy. Future developments in treatment strategies continue to evolve with options such as probiotics, concurrent administration of non-toxigenic *C. difficile*, faecal bacteriotherapy with administration of strained faeces by gastric intubation or enema and immunotherapy with antitoxin antibodies. [1, 2]

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