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Editorial Open Acess

Pseudomembranous colitis

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Editorial

In 1978, *C. difficile* was identified as the causative agent of pseudomembranous colitis [1]. There has been a worldwide increase in the incidence and severity of *C. difficile*-associated diarrhea (CDAD) due to increased use of broad-spectrum antimicrobial, crowded hospital wards [2,3] and/or poor infection control [4,5]. Pseudomembranous colitis associated with antibiotic use may alter the balance of normal flora of the intestine and allow overgrowth of certain organisms such as *C. difficile* [6] which is a gram-positive, spore-forming, anaerobic bacillus its main virulence determinants are toxin A (enterotoxin) and toxin B (cytotoxin) [7]. The increased amounts of these toxins possibly related to a truncating mutation in the *tcdC* gene, which codes for a putative repressor of toxin A/B production [8,9]. Toxin A interferes with colonic mucosal cell adherence to colonic basement membrane and damages villous tips while toxin B enters the cell by endocytosis and induces apoptosis [10]. Not only clindamycin, lincomycin, ampicillin and cephalosporin have been implicated in most cases of pseudomembranous colitis but also antifungal, antiviral, and metronidazole could incite the disease. The illness is characterized by offensive-smelling diarrhea, fever, leukocytosis and abdominal pain. Life-threatening complications can develop in severe cases such as toxic megacolon [11]. Diabetics, elderly, intensive care unit patients and recent major surgery is the main risk factors [12] also there are some evidence that proton pump inhibitors are a risk factor for pseudomembranous colitis [13]. Non antimicrobial *C. difficile* pseudomembranous colitis was reported in many conditions as bowel ischemia, recent bowel surgery, uremia, chemotherapy and Hirsch sprung disease.

The toxins produced by *C. difficile* could be detected by various methods including enzyme immunoassay (EIA), cell cytotoxicity assay or real-time polymerase chain reaction (PCR) for toxin B gene (*tcdB*) determination [14]. There is some controversy regarding the sensitivity and specificity of the various toxin assays, so repeat toxin testing at least once after a negative result is necessary [15] or use a two-step method of detection, incorporating bacterial antigen or culture [16,17]. Pseudomembranous colitis could be treated by discontinuation of antibiotics and oral vancomycin, intravenous or oral metronidazole is recommended as first-line treatments for mild to moderate disease without complications. Complicated cases as toxic megacolon, perforation and acute abdomen may require surgical intervention by subtotal colectomy with ileostomy [18]. A randomized controlled trial using a probiotic drink containing *Lactobacillus casei, L. bulgaricus, and Streptococcus thermophilus* was reported to have some efficacy. However, this study was, sponsored by the company that produces the drink [13].

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