Fulminant pseudomembranous colitis of the left colon successfully treated by surgical resection

Sol kolonun fulminan psödomembranöz kolitinde cerrahi rezeksiyon ile başarılı tedavi

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INTRODUCTION

Accumulated evidence has established antibiotic use as the primary risk factor for pseudomembranous colitis and opportunistic Clostridium difficile infection as the cause of antibiotic–associated colitis, regardless of pseudomembrane formation (1, 2). The use of antibiotics can disrupt the normal bowel ecosystem and colonize the empty bowel. Although most antibiotics have been associated with predisposition to C. difficile infection, the most commonly implicated have been clindamycin, cephalosporins and ampicillin (3).

Five toxic factors of C. difficile have been described, but only two of these, toxins A and B, have been studied in any detail with good evidence of involvement in disease. The other three factors were described as a second, unstable, enterotoxic protein (4).

Disease may be associated with a spectrum of severity, ranging from mild diarrhea, through moderately severe disease, to life–threatening and sometimes fatal pseudomembranous colitis (PMC). This may be accompanied by toxic megacolon, electrolyte imbalance and occasional bowel perforation. PMC is a progressive, systemic inflammatory state that may develop in patients with C. difficile colitis unresponsive to medical therapy (5).

Characteristically, only the colon is involved and endoscopy reveals irregular ulcerations covered with pathognomonic fibrinous pseudomembranes and mucosal edema. There is formation of a pseudomembrane, comprising mucin, fibrin, leukocytes and cellular debris (6).

Sigmoidoscopy reveals the classic adherent yellow plaques that range in diameter from 2 to 10 mm. Intervening mucosa may look normal or plaques may become confluent. The left colon is most commonly affected, but there is rectosigmoid sparing in up to two-thirds of cases, and in 10% of cases, the disease is confined to the right colon. Although C. difficile infection has been thought of as a pure colitis, it is now clear that the small bowel is also vulnerable and may even form pseudomembranes (7).

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Endoscopy is an unreliable method of confirming or refuting the diagnosis of *C. difficile* infection (8). PMC arises when the normal colonic bacterial flora is disrupted such that *C. difficile* can colonize the bowel and produce enterotoxin. Therapy with broad-spectrum antibiotics is the key event allowing this to happen in most cases (9). Management of PMC with oral metronidazole and/or vancomycin is usually sufficient. However, progression to fulminant colitis with systemic toxin effects and acute abdominal symptoms is seen with increasing frequency, especially in the elderly and immunosuppressed populations (10, 11).

This case report presents a patient with severe pseudomembranous inflammation confined to the left colon, which was successfully treated by surgical resection.

**CASE REPORT**

Our patient was a 60-year-old female who was transferred to our surgical intensive care unit from a medical care unit. Her medical history revealed that she was given prophylactic treatment of 480 mg of trimethoprim-sulfamethoxazole bid per oral due to chronic renal insufficiency for two years. Chief complaints on admission to our clinic included diffuse abdominal pain, nausea, vomiting and a very small amount diarrhea. On examination her temperature was increased (39°C) and pulse rate was 110/minute. There was excessive tenderness and guarding in the left upper and lower quadrants. The abdomen was slightly distended but no masses. Digital and rectosigmoidoscopic examination of the rectum was normal. Laboratory tests included a white blood cell count of 17,000 cells/mm³ and a positive *C. difficile* toxin assay. Stool culture showed *C. difficile*. Plain abdominal radiographs showed hydroaerobic levels in bowels without pneumoperitoneum. An abdominal ultrasonography demonstrated moderate diffuse bowel wall thickening. Repeated white blood count was 18,000 cells/mm³. The patient had been treated in the first day of illness with intravenous vancomycin and electrolyte replacement in the medical care unit before being transferred to our surgical unit because of peritoneal irritation and acute abdomen.

At the same time, emergency laparotomy was performed. At operation, left colon demonstrated diffuse wall thickening and inflammation; the remainder of the colon and rectum was normal. A left hemicolecotomy and end to end anastomosis were performed. Vancomycin was given intravenously during four days in the postoperative stage.

She was afebrile and had no abdominal pain. Postoperative convalescence was uneventful. The stool culture was negative at the postoperative sixth day for *C. difficile*. The operative specimen showed multiple irregular ulcers with overlying pseudomembranes through the entire colon. Mucosa between the ulcers exhibited edema and congestion. The lesions stopped abruptly at the splenic flexure and distal sigmoid colon (Figure 1).

**Figure 1.** The lesion extending the splenic flexure and distal sigmoid colon

Histology showed generalized inflammation throughout all coats, with foci of superficial necrosis of the mucosa covered by fibrin and mucus in which no protozoan fungi could be demonstrated. A spore forming anaerobic Gram-positive bacillus (*C. difficile*) was demonstrated (Figures 2-3). There was no intramural hemorrhage and the lymph nodes were not enlarged.

**Figure 2.** This is a representative section demonstrating pseudomembrane covered with mucosal surface (HE x 50)
prophylactic antibiotic treatment due to long-standing chronic renal insufficiency.

PMC was first recognized as a clinical entity in the 1950s. The advent of broad spectrum antibiotics in the 1960s and 1970s led to a marked rise in the numbers of patients developing PMC, an increase that has continued to the present day.

The first presentation of *C. difficile* infection might be a surgical emergency with complications more commonly associated with fulminant ulcerative colitis, namely toxic dilatation and perforation (12-14). Volvulus has also been reported in association with PMC and *C. difficile* infection (15). Patients may undergo emergency laparotomy before the true diagnosis is established, particularly in the case of suspected bowel obstruction with marked systemic upset and a distended tender abdomen with or without peritonitis. Plain abdominal radiographs may show dilated small and/or large bowel (16).

In our case, the patient had diffuse peritoneal irritation, particularly in left upper and lower quadrants, and a very little diarrhea output. Patient’s plain abdominal radiographs showed dilated small bowel and partially proximal colon. These appearances cannot be distinguished reliably from those of obstruction or ischemia. Colonoscopy has been recommended as a diagnostic procedure and as a means of decompression but this may induce perforation. Water-soluble contrast enema examination may help to exclude mechanical obstruction, but it is not without risk (1, 17).

Computed tomography (CT) may show the “clover leaf” in less than 20% of PMC cases. Bowel wall thickening, pericolic streaking and ascites are noted in over half of the patients. The severity of disease does not correlate reliably with appearance on CT (18). In our patient, abdominal CT was not performed but ultrasonography showed bowel wall thickening and dilated small and large bowel without free fluid in the peritoneal cavity.

The first approach in the treatment of *C. difficile* infection must be to stop the precipitating antibiotic whenever possible. Studies have indicated that approximately 15–25% of patients respond to this approach alone. Specific treatment is indicated if the patient has systemic symptoms, particularly if there is evidence of severe colonic inflammation or pseudomembrane formation (3). Emergency surgery is required for patients with perfo-
racion and for those who fail to respond to medical treatment (19). The disease is often fatal and in many cases the diagnosis is made only at nec-

REFERENCES