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Clinical Image

Clostridium Difficile Provoked Pseudomembranous Colitis: A Potential Risk Factor for Instigation of Toxic Megacolon and Septic Shock

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Clinical Image

Recently, there has been a massive upward trend in the usage of multiple antibiotics for infections. Commonly used antibiotics include penicillin, clindamycin, cephalosporins, trimethoprim-sulfamethoxazole, NSAIDS and fluoroquinolones [1]. The deleterious consequences of long-term usage of these commonly prescribed antibiotics stems from their inherent notorious nature to instigate destabilization of protective microflora colonizing the gut. Physiologically, these indigenous gut microflorae are principally responsible for upholding the gut mucosal health by supplying micronutrients, elevating gut protective immunoglobulins and maintaining a healthy mucosal layer [2,3]. In this regard, excess antibiotic usage used by the host can be detrimental as they potentiate excessive killing of gut commensals harboring in the gut, thence nullifying their protective effects as well as altering the physiological gut mucosal diversity [4,5]. This alteration in the gut microbial environment greases the wheels for materialization of clostridium difficile enterocolitis. Case control studies have classified the antibiotics based on their risk profile. Doxycycline, and Trimethorpim are classified as low risk, while Ampicillin, Cephalexin, Levofloxacilin and Linezolidare intrepreted as medium risk, whereas Amoxcillin-Clavulunate, Ciprofloxacilin, Cefuroxime and Clindamycin are deemed to be high risk for instigating clostridium Colitis.

Our patient had a past history of upper respiratory tract infection due to which she had used ciprofloxacin for 10-14 days, following which she presented with diarrhea, fever, leukocytosis, hypotension needing ICU admission, vasopressor support. Workup including stool culture showed Clostridium colitis. Urgent Gastroenterology consultation and colonoscopy showed classic pseudo-membranes (Figure 1) as shown in the figure. Since her septic shock was unabated, she was referred for surgical team for colectomy to remove the gangrenous colon.

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Clostridium difficile is an anaerobic gram-positive rod that exploits the disarrangement and anarchy of the colonic microenvironment effectuated by prior antibiotic use [6]. The pathophysiology of Clostridium difficile diarrhea entails the involvement of Toxins A and B whose permeation into the colonic epithelial cells is the triggering factor for inducing colonic epithelial cell death and disruption of tight junctions [6,7]. These cellular events set the stage for eventual migration of neutrophils, macrophages and monocytes into the site of injured colonic epithelium [7-10]. Active secretion of cytokines, chemokines and interleukins by these inflammatory cellular infiltrate greases the wheels for the formation of micro abscesses and pseudo-membranes, characteristic of clostridium colitis [11].

In a classic case of fulminant colitis, colonoscopy might reveal pseudomembranous colitis which is tell-tale sign of clostridium colitis [12]. Gross examination might reveal yellowish-green white plaques resting upon the hyperemic colonic mucosa interspersed with necrosis, a finding that is characteristic of clostridium difficile colitis, [13-15] (Figure 1). Nevertheless, these changes can sometimes can be seen in Bechet disease, inflammatory bowel disease, ischemic colitis, collagenous colitis and infectious colitis [12]. As the disease progresses, these plaques become more confluent and widespread by gradually marching to embrace longer segments of the colon. These above-mentioned changes transform the colon into necrotic and gangrenous, thus necessitating colectomy to remove the source of infection as well as improve survival rate.

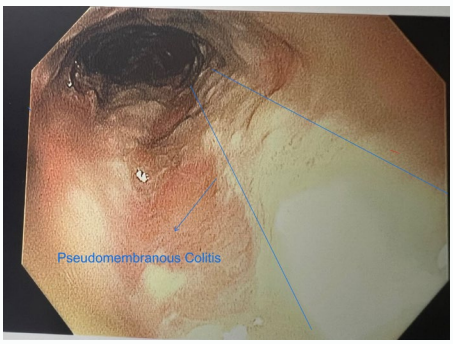

Figure 1: Pseudomembranous Colitis: Yellowish-green plaques that are adherent to the colonic mucosa in the patient presenting with cramping abdominal pain, diarrhea, fever, leukocytosis and hypotension. These changes are characteristic of pseudomembranous colitis.

Histopathologic examination reveals that these pseudomembranes are mainly composed of neutrophils, inflammatory cells and nuclear debris in association with dense purulent debris that springs out to the colonic surface from the infected crypts [6,15-17]. Underlying lamina propria might display dilated microcapillaries, microthrombi, vasodilation and platelet aggregation [15]. Some clinical researchers attempted to classify this pseudomembranous colitis into early stage I (focal epithelial inflammation/ necrosis, stage II (involvement of mucosa and submucosa with inflammation and necrosis) and late-stage III (transmural epithelial necrosis with significant inflammatory infiltrate) [17-19].

The clinical spectrum of Clostridium difficile enteritis is quite variable and can range from asymptomatic carrier, mild colitis, fulminant colitis and toxic megacolon [12,15]. Typical signs encountered as disease process progresses to fulminant colitis includes fever, leukocytosis, hypovolemia, hypotension, hypoalbuminemia, reactive arthritis and renal dysfunction [14,20,21]. Early diagnosis and treatment with vancomycin or fidaxomicin should be initiated as early as possible [22]. Careful monitoring is almost always necessary as these patients can deteriorate rapidly to develop severe complications such as severe ileus, pan colitis, septic shock, colonic dilation, toxic megacolon, colon perforation, peritonitis and septic shock [23,24]. Keeping in mind of these complications, it would be prudent to have a watchful eye and seek prompt gastroenterology consultation for colonoscopy and detection of pseudomembranous colitis. Fulminant colitis requires immediate surgical consultation for removing gangrenous bowel by performing life-saving colectomy. Early surgical intervention might improve survival rates as it might hasten the recovery from the septic shock and favor better mortality & morbidity rates. Recurrence of clostridium are not uncommon and mostly influenced by age, presence of risk factors, history of previous infections, and immunosuppression [15].

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