Toxic Megacolon: A Conundrum

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1. Abstract
Toxic megacolon is defined as an acute non-obstructive inflammatory condition with dilatation of the bowel causing septicemia and peritonitis. The cut off point for the bowel dilatation is taken as 6cm and above with loss of haustration on imaging. The exact cause of toxic megacolon is still debatable but several postulations have been laid down. The etiology of toxic megacolon can be divided into several factors such as inflammatory, autoimmune, tumors and others. Toxic megacolon carries a high morbidity and mortality with delayed interventions. The incidence increases with age and male preponderance.

2. Introduction
Toxic megacolon(TM) is a rare and fatal surgical condition in which the colon is dilated at least 6cm and more causing impending obstruction and perforation. The etiology for TM is not exactly known however, several theories have been postulated.TM shows tendency towards male patients compared to females. Untreated TM may cause bowel perforation, sepsis and imminent death. Occasionally TM can be managed conservatively but if patient did not improve beyond three days, the patient should be operated. Proper history, clinical examination and imaging is needed to diagnose and treat TM. Despite its low prevalence, the mortality of toxic megacolon is about 7.9 %.[1, 2]. We would like to share our experience of TM due to clostridium difficile infection with impending perforation which we managed to operate promptly and save the patient.

3. Case Report
An elderly patient presented to our hospital with the complaints of severe abdominal pain, poor oral intake, nausea and vomiting for the past 2 weeks. Otherwise he was a healthy moderately built male. On general physical examination, noted patient was severely dehydrated, pale and lethargic looking. About 4 litres of normal saline was infused into him for resuscitation. Per abdomen noted tensed and distended with measuring abdominal girth of 160cm. Bowel sound was sluggish while digital rectal examination showed empty and roomy rectum. Routine blood investigations taken showed normal range except for low sodium and potassium. We also arranged for chest and abdominal x-rays which showed severely dilated large ascending bowel with the loss of haustra. The dilated large bowel extended from ascending till transverse bowel and it occupied the left diaphragm mimicking Chilaiditi syndrome. We started patient on high end antibiotics and pushed the patient to the operating room since patient was deteriorating despite adequate resuscitation. Intraoperatively we noted a hugely dilated ascending and transverse colon with color changes and thinning out of some part of the colon. Enterotomy was done and the coon was milked and decompressed. We performed a partial colectomy with a diverting loop ileostomy for this patient. He was nursed in the ICU for several days and was put on total parenteral nutrition. We also started patient on steroid therapy to reduce the inflammatory process. The segment of dilated bowel sent to the histopathology showed inflammatory process with positive to clostridrium difficile. The patient was treated with IV Vancomycin for one week and all the septic parameters improved over few weeks. He was discharge home after two weeks with a clinic appointment and arrangement for reversal of the ileostomy (Figures 1-4).
Figure 1: chest x-ray showing dilated bowels occupying the left hemi-diaphragm

Figure 2: Abdominal x-ray showing dilated small and large bowels with the loss of haustration

Figure 3 and 4: Intraoperative picture of the ascending colon and the transverse colon which were severely dilated and thinned out.

4. Discussion

Toxic megacolon is a fatal condition if not dealt with in the early stage. It can cause septic ileitis, intestinal obstruction as well as perforation and peritonitis. There are several factors which may cause toxic megacolon. Commonly seen are inflammatory causes such as clostridium difficile, cytomegalovirus, salmonella and shigella. Although C. difficile shows no signs and symptoms, it can cause severe inflammatory changes and sepsis [3, 4]. Diagnosis of toxic megacolon can be done by a thorough history, clinical examination as well as imaging. Increase in transverse colon size of more than 6cm is pathognomonic for toxic megacolon. Jalan et al described the diagnostic criteria of TM which should fulfill any 3 of the following: fever >38 degree, leukocytosis >10.5x10^3/microliter and anemia also any of the following; hypotension, hypovolemia, altered mental status or electrolytes imbalance. A typical imaging feature to diagnose TM is maximum diameter of the transverse colon more than 6cm, loss of haustration, segmental wall thinning with air fluid levels. Colonoscopy is contraindicated in TM as the bowel wall is very thin and flimsy and may cause perforation. A careful sigmoidoscopy may be a safer option [5]. The pathogenesis of TM is not entirely known, however there are few hypothesis that has been proposed such as mucosal inflammation. The inflammation releases mediators, bacterial products as well as the production of inducible nitric oxide synthase which causes bowel to dilate. There have been studies that showed patient with TM has high levels of inducible nitric oxide synthase in their muscularis propria compared to the normal populations [6]. There are many proposed treatment modalities for TM, however conservative management including bowel rest and nil by mouth should be considered before embarking into surgery. While in case of C.Def infection, the offending antibiotics should be stopped and steroids should be commenced to reduce the inflammation process. If within 2-3 days patient does not show improvement, surgical options should be considered. Criteria for surgery such as colonic perforation, bowel necrosis, full thickness ischemia, intra-abdominal hypertension,
abdominal compartment syndrome as well as peritonitis [7]. In our case of TM, we opted for surgery in view of patient does not show improvements despite aggressive resuscitation.

5. Conclusion
Toxic megacolon is a preventable and treatable condition and should be tackled judiciously at the first presentation. Despite the etiology of TM, conservative treatment should be considered before surgical. Combination of both medical and surgical treatment is crucial in treating TM as detrimental effect may occur such as perforation and sepsis. TM can be fatal if treatment is delayed, thus clinicians must be vigilant in dealing with TM.

References