CASE REPORT

Acute Megacolon (Acute Colonic Pseudo-Obstruction)- A Case Report

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ABSTRACT

Acute megacolon is a condition that is poorly understood in its entirety. It is characterized by dilatation of the colon and in some cases, rectum. It is called colonic pseudo-obstruction when no obstructive cause is found. It presents classically with features of intestinal obstruction. There are several management options including conservative, medical and surgical. It is peculiar in the sense that none of these options give complete cure and it has a high tendency to recur. This is a case report of a 30-year old lady who had acute megacolon following a long period of hospitalization for necrotizing fasciitis.

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Key words: Megacolon; Intestinal Obstruction; Colectomy

INTRODUCTION

Megacolon describes a condition in which there is dilatation of the colon\(^1\). It could affect either the colon or the rectum alone, a situation respectively called megacolon and megarectum.

Megacolon is divided into 3: Acute megacolon in which there is no obvious colonic disease (colonic pseudo-obstruction or Ogilvie’s syndrome); Chronic megacolon which could be congenital, acquired or idiopathic; toxic megacolon, which occurs in association with inflammation of the colon.

The cause for this altered motility is unclear; however, there are several proposed mechanisms. Abnormalities of autonomic nervous system of the intestinal tract and myenteric plexus have been suggested\(^2\). Electrolyte abnormalities, metabolic causes like hypo/hyperthyroidism, drug use like anti-diarrhoeals, antipsychotics and anti-depressants, inflammatory causes like inflammatory bowel disease, infections like Clostridium difficile\(^3\) colitis and Chagas’ disease may be involved.

Differentials include toxic megacolon and chronic megacolon.

Investigations include urea, electrolytes and creatinine levels, complete blood counts stool m/c/s, thyroid function tests, plain abdominal X-rays and contrast studies. Plain X-rays show massive colonic distensions, which may involve all sections of the large bowel. CT scan, MRI, enema may be used to exclude obstruction. Colonoscopy, if available and if patient is stable, may be diagnostic and therapeutic.

CASE REPORT

The patient is a 30-year old female unemployed graduate who presented with a 4-week history of constipation, 2-week history of abdominal swelling and pain and 2-day history of non-projectile vomiting. Pain was colicky and there was passage of flatus. She took herbal remedies for constipation and abdominal pain.

Twenty-six days before presentation, she was discharged from a previous 16-week hospital admission on account of a right gluteal abscess and necrotizing fasciitis for which she had incision and drainage and debridement under general anaesthesia. She was having daily wound dressing until about 2 weeks later when a fistula was noticed discharging faecal matter. She was then pregnant, at 30 weeks gestation. Her PCV was 19%. She was transfused with 3 pints of blood and placed on parenteral augmentin based on sensitivity of wound m/c/s result obtained which yielded mixed flora of E.Coli, pseudomonas aerugenosa, Staphylococcus Aureus , and metronidazole.

She had intra-uterine faetal death (IUFD) while on admission and was promptly attended to by the Obstetrics and Gynaecology unit. Following this, her husband left her and she was left alone to fend for herself. She lost appetite and had insomnia, lost weight remark-
ably with crying episodes. Her episode of constipation started around this time.

The wound granulated well on daily dressing and serial debridement and almost healed except for the fistula which was still discharging.

After about 45 days on admission, she requested for discharge. Examination revealed a cachectic lady, sad affect, dyspneic, not pale, anicteric and mildly dehydrated.

Abdomen was distended, moves with respiration, tympanic to percussion, non-tender, absent bowel sounds.

Per rectal examination showed full rectum.

An assessment of Acute Intestinal Obstruction secondary to faecal impaction to rule out adhesion was made. She was to be admitted for conservative management with nil per os, intravenous fluids, antibiotics nasogastric tube for aspiration with possible surgical intervention. However, she refused admission. Two weeks after, she returned with worse signs and features than before. She was booked for surgery but also declined only to return the third time.

She did plain abdominal X-ray (erect and supine views). It shows distension of large bowel. Electrolytes and Urea were normal and PCV was 34%. She had exploratory laparotomy under general anaesthesia. Findings included massively dilated colon (Figures 1), rectum and small bowel. The diameter of the caecum was 14cm, transverse colon 10cm and sigmoid colon was 14 cm. No perforation, tumour, adhesion, volvulus or pelvic collection. Decompression was achieved by a sigmoidotomy and ileotomy. A transverse colostomy was created, peritoneal cavity irrigated and abdomen closed in layers.

Naso-gastric aspiration was minimal and was removed on the 3rd day. Colostomy functioned on the 1st day after surgery. She made adequate urine and urethral catheter was removed 3rd day. She developed abdominal distension on the 12th day post-surgery while the transverse colostomy was still functioning. Per rectal examination revealed dense scar tissue around the anal opening narrowing the anal orifice. Digital examination with a well lubricated finger, after initial resistance, showed tight anal orifice, watery faecal matter in the rectum. Anal dilatation was done. Removal of the examining finger provoked rectal discharge of watery content, passage of flatus and reduction of abdominal girth. She has since been emptying per rectum.

Her wound healed well and was eventually discharged on the 20th after surgery

**DISCUSSION**

Acute megacolon is as yet, a condition whose pathogenesis is as misunderstood as its management. It can present as gastroparesis, ileus or colonic pseudo-obstruction. Initially thought to be associated with post-operative states, it is now believed to be associated with in-patients with underlying serious medical or surgical conditions for which they have been treated or from which they are recovering. This could be psychiatric illness like depression and psychosis. Lim et al found an association between risperidone and megacolon. Our patient was just recovering from a long hospitalization for necrotizing fasciitis with resulting faecal fistula. In addition, the loss of her pregnancy and soon after, her abandonment by her husband and occasional weepy spells with sad affect were pointers to the fact that she may have suffered from depression. This may however be unrelated to the illness as she was not placed on any anti-depressant.

It could be due to abnormalities involving enteric nerve plexus and intestinal smooth muscles leading to poor motility and dilatation.

Histologically, while atrophy and fibrosis of the tendinous fibre net of muscularis propria have been found in megacolon to inhibit peristalsis thus allowing unlimited colonic distension, thickening of the enteric smooth muscle was found in patients with idiopathic megarectum. They are however in agreement that there were no abnormalities in the enteric nerve plexuses where abnormalities of neurotransmission involving neurotransmitters like VIP and Nitric Oxide probably have a significant role to play.

There is paucity of information about incidence rate and age groups involved. However, while some authors believe it occurs equally in both sexes, others say it is 7 times more common in females.

They present usually with abdominal distension with or without abdominal pain. There is history of varying degrees of constipation or obstipation with bowel motion from less than 3 per week to once in 3 months. Our patient had a 28-day history of constipation although she continued to pass flatus. This tallies with the findings of

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**Figure 1** showing distended sigmoid, caecum and loops of small bowel.

**Figure 2** This shows dilated loops of small bowel, caecum, transverse colon and sigmoid colon.
Maloney et al[6] who found that 40% to 50% of patients continue to pass flatus. There may be nausea and vomiting. Examination findings are those of massive abdominal distension causing breathlessness, tympanic percussion notes and bowel sounds which may be present or absent.

Diagnosis is usually reached by plain abdominal films erect and supine which will reveal dilatation of the large bowel. Because of the difficulty in differentiating colonic pseudo-obstruction from obstructive causes, it may be necessary to do a colonoscopy, enema studies or MRI. Full thickness rectal biopsy is necessary. A biopsy was not done here because she came in with acute obstruction and for the fact that the whole of the large bowel was uniformly involved in the megacolon, including the rectum.

Our patient presented as acute intestinal obstruction, therefore we could not do many investigations apart from plain abdominal X-rays which did not get to us until after surgery. Diagnosis was basically clinical. At surgery we found massive dilatation of the colon from caecum to the rectum (Figure 2). The distal small bowel too was involved. The caecal diameter was 14 cm while those of the ascending, transverse and sigmoid colon were 12, 10 and 14 cm respectively. Diagnosis of megacolon could be made with enema with caecal diameter more than 6.5 cm[12]. It has been shown that caecal perforation is rare at less than 12 cm caecal diameter but occurs more commonly at 14 cm and above[6].

There are many modalities of treatment of this condition ranging from medical to surgical. It requires a multidisciplinary approach including the colorectal or general surgeon, physician, psychiatrist, psychologist and a stoma care specialist.

Patients with less than 12 cm caecal diameter, without signs of ischemia and perforation, should have conservative management[6]. This includes nasogastric aspiration, nil orally, placement of rectal tube and stoppage[12] or dose reduction[7] of aggravating medications. Electrolyte and fluid derangement if any should be corrected and underlying infective processes treated. There should be close monitoring with plain abdominal X-rays, electrolytes and patient’s clinical state. Conservative management should cease once there are signs of perforation, evidence of increasing caecal diameter beyond 12 cm or lack of response within 72 hours. Response is usually seen within 6 days[6].

Medical management includes use of pharmacologic agents. Neostigmine, an anticholinesterase has been used with very good results[17,18]. It is given IV 2-2.5 mg over 3-5 minutes. It should be used with caution, because of the possibility of bradycardia. Others that have been tried include use of prokinetic agents and epidermal block. Since some of these patients have an underlying depression, hypothyroidism[19] and other conditions, these conditions need to be treated.

Colonoscopic decompression provides temporary relief as it reduces diameter of the caecum. It also provides the added advantage of viewing the colon to rule out obstructive cause. Placement of a rectal tube especially if passed up to the right side of the colon has limited success rates as it gets blocked easily.

Surgical management is complex and no procedure is found to be 100% successful in achieving cure. The numerous surgical modalities available for this condition underline the mystery and confusion surrounding it. Subtotal colectomy is done for patients with megacolon, with ileorectal anastomosis, while in the presence of megarectum as well, Proctocolectomy is done with ileoanal anastomosis. Both procedures are associated with significant morbidity[20], most developing bowel obstruction. Segmental resection has been tried with limited success. There is high recurrence of symptoms. Significant number of patients who had sigmoid colectomy alone had recurrence of constipation[21]. Other modalities include Duhamel operation, protectomy with coloanal anastomosis especially in patients with megarectum and pelvic floor procedures. However, according to Reddy et al[4], no proof of the effectiveness of these procedures. Our patient had peri-anal hypertrophic scar and tight anal orifice on the 12th post-operative day occasioned by healing by fibrosis following gluteal abscess and fasciitis. This was not there at surgery. This responded to simple digital anal dilatation.

Creation of stomas is seen as a very safe option to prevent ischemia and perforation. There is rapid relief of symptoms. The patient should however be informed of the possibility of it been permanent[22]. They may also be temporary for immediate relief while awaiting corrective surgery. These include cecostomy, ileostomy or colostomy. Cecostomy and ileostomy are both associated with high morbidity with difficulty in managing it. There may be faecal impaction proximal to the colostomy which may cause stoma prolapse.

CONCLUSION

Acute megacolon (Ogilvie syndrome or acute colonic pseudo-obstruction) is a condition that is poorly understood in its entirety and the multiplicity of surgical procedures associated with it underlines that fact. High index of suspicion is needed in identifying it. There is also the need to differentiate it from toxic megacolon which needs urgent intervention. The need to communicate with the patient every step of the way on treatment and on long term expectation cannot be overemphasized. While conservative management is important, it is necessary to know when to stop and proceed to surgical intervention when it is obvious non-surgical method has failed and especially when there is ischaemia and perforation, the two factors responsible for high mortality in this condition.

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