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Case Report

Adhesive small bowel obstruction in a virgin abdomen in an elderly: a case report

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ABSTRACT

Small bowel obstruction (SBO) is a major cause of morbidity and financial burden in hospitals around the world. Although adhesions account for more than 70% of SBO, they were thought to be less likely etiologies in a virgin abdomen. However, emerging evidence appears to challenge such a dogmatic approach. Here, we report a case of a 68 years old Maldivian man with a virgin abdomen who presented as small bowel obstruction secondary to de novo adhesions at terminal ileum. He was treated successfully with uneventful postoperative outcome in a government regional referral hospital.

Keywords: Small bowel obstruction, De novo adhesions, Virgin abdomen

INTRODUCTION

Small bowel obstruction (SBO) is a condition where there is interruption of forward flow of intestinal contents.[1] Bowel obstruction is one of the most common surgical emergencies encountered in general surgery units and it continues to be a major cause of morbidity and financial expenditure.[2]

SBO etiology in developed countries includes adhesions (74%), Crohn's disease (7%), neoplasia (5%), hernia (2%), radiation (1%), and miscellaneous (11%). In contrast, developing countries etiology includes adhesions (34%), hernia (16%), malignancy (13.5%) and tuberculous stricture (10%).[3] Potential mimickers of SBO described in the literature include ACE-inhibitor related bowel angioedema, eosinophilic enteritis, and different infectious etiologies.[4]

Intra-abdominal adhesions are abnormal connective tissue attachments between two or more peritoneal surfaces.[5] Adhesions are a major source of morbidity and are the most common cause of intestinal obstruction, secondary female infertility, ectopic gestation and chronic abdominal and pelvic pain.[6-9] It was first described by Hippocrates and first treatment was performed by Praxagoras in 350 BC.[1] First adhesions were identified at post-mortem of a patient in 1836.[1]

The term virgin abdomen was coined before widespread use of computed tomography, which now elucidates many SBO etiologies.[10] Although adhesions account for more than 70% of SBO, they are thought to be less likely etiologies in patients without previous abdominal surgery, where a hernia or malignancy is frequently believed to be the underlying cause.[11] However emerging evidence appears to challenge such a dogmatic approach as adhesions remain most common cause of obstruction in patients despite absence of prior abdominal surgery.[11]

CASE REPORT

The patient is a 68-year-old Maldivian male with no prior surgical history who had mild intermittent generalized abdominal pain for 4 months. He presented to the emergency with a 1-week history of colicky abdominal pain and 1 day history of abdominal distension, vomiting and obstipation. His vital signs were stable with no

tachycardia or fever. His abdomen was distended and tympanic with no tenderness and sluggish bowel sounds. Hernial sites were intact and DRE was unremarkable.

White blood cell count was 6.9×10^9/L, hemoglobin was 10.8 g/dl, sodium and potassium were 136 mmol/L and 3.8 mmol/L respectively and blood urea nitrogen and creatinine were 31.1 mg/dl and 0.9 mg/dl respectively. Abdominal x-rays (Figure 1) showed features of SBO and no free air under diaphragm. Tumor biomarkers carcinoembryonic antigen (CEA) and carbohydrate antigen (CA19-9) were checked and found to be within normal range.

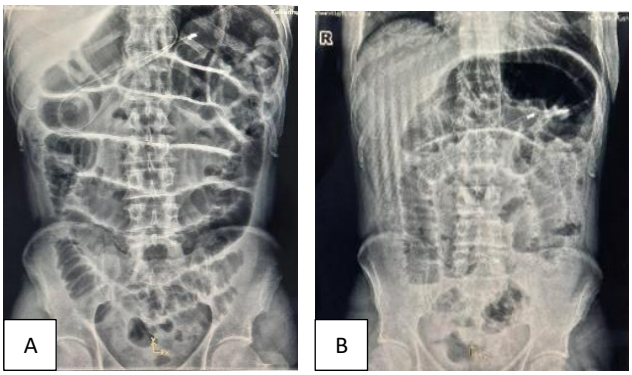

Figure 1 (A and B): Abdominal x-ray of dilated jejunum and ileum in supine film and air fluid levels in erect film.

A trial of non-operative management was adopted in view of SBO in clinically stable elderly patient with presumed clinical and biochemical non-malignant etiology. Patient responded well to conservative management using nasogastric decompression and fluid resuscitation.

A contrast enhanced computed tomography scan (CECT) of the abdomen demonstrated significant abrupt smooth long segment narrowing at distal part of ileum measuring 5 cm in length causing its luminal narrowing resulting in proximal dilatation of bowel loops. Such findings with history of recurrent symptoms prompted surgical intervention and patient was consented for an exploratory laparotomy with possible bowel resection.

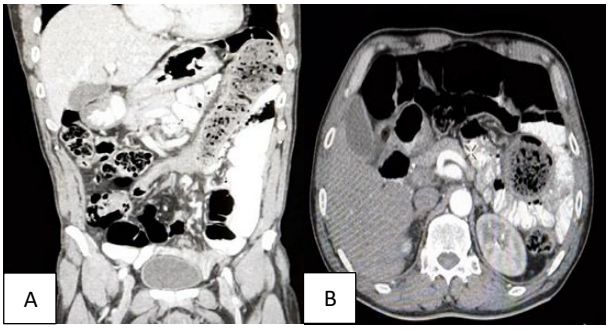

Figure 2 (A and B): CECT of abrupt narrowing of terminal ileum with dilated adjacent proximal loops.

Intra-operative findings noted were matted adhesions at terminal ileum 5 cm proximal to ileocecal junction which involved around 7 cm length of ileum causing almost complete luminal obstruction. The narrowed segment of ileum was healthy and adjacent proximal bowel was dilated. There was no mass noted, no mesenteric deposits or enlarged mesenteric lymph nodes, no peritoneal seedlings and no ascites. Patient underwent adhesiolysis and the narrowed segment was decompressed.

Post-operatively patient was initiated orally with sips on first post-operative day, however he developed prolonged paralytic ileus which increased morbidity; feeding was restarted on ninth postoperative day and patient was discharged on thirteenth post-operative day with normal oral intake and bowel and bladder habits. The patient is symptom free at 10 months post-operatively.

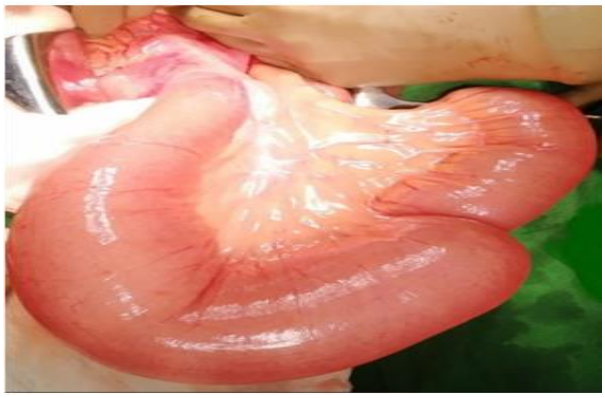

Figure 3: Intra-operative picture of matted band adhesions at terminal ileum.

DISCUSSION

SBO is a major cause of morbidity and financial burden in hospitals around the world.[3] Although adhesions account for more than 70% of SBO, they were thought to be less likely etiologies in patients without previous abdominal surgery.[11] Emerging evidence appears to challenge such a dogmatic approach as adhesions remain the most common cause of obstruction in patients despite the absence of prior abdominal surgery.[11] A systematic review and meta-analysis of 6 cohort studies involving 442 patients found that de novo adhesions (152/281, 54.1%) were the most common etiology of SBO in patients without abdominopelvic surgery history.[10]

Any source of peritoneal irritation can result in local fibrin production which may lead to adhesion formation.[12] The process of adhesion development begins when damage to peritoneal surfaces from any source (operative trauma, infection, foreign bodies, desiccation, irradiation, allergic reaction, or chemical injury) induces a series of biochemical/molecular biologic cascades.[13] The biology of peritoneal repair is now known to involve a concert of chemical mediators, cytokines, cell types, degradation products, and proteases to accomplish

healing.[14] Adhesion formation at the molecular level constitutes a complex interaction of cellular components (Peritoneal leukocytes, mesothelial cells, and macrophages) and humoral mediators (Transforming growth factor beta (TGF-b), Vascular endothelial growth factor (VEGF), interleukins, Tumor necrosis factor-alpha (TNF-a), Matrix metalloproteinase enzymes (MMPs) and tissue inhibitors of matrix metalloproteinases (TIMPs)) at or near the injured area.[13-15]

Adhesions may be congenital or acquired (post-inflammatory or post-operative).[1,7,8,15] Approximately 85% of adhesive SBOs are caused by postoperative adhesions, 10% are caused by peritonitis, and 5% have unknown or congenital etiologies.[5] Congenital adhesions are present from birth as embryological anomaly in the development of the peritoneal cavity (vitellointestinal bands, adhesions seen across the lesser sac).[15] Inflammatory adhesions arise after intra-abdominal inflammatory processes, such as appendicitis, acute cholecystitis, acute diverticulitis, pelvic inflammatory disease, and the previous use of an intrauterine contraceptive device.[15] Adhesions in patients with a virgin abdomen can be congenital or secondary to intra-abdominal infections such as tuberculosis.[1,11] However, in our case there was no history suggestive of previous intra-abdominal infections or any surgical intervention.

Post-operative adhesions form at surgical site, non-surgical site or after adhesiolysis, these three processes identified as adhesion formation, de novo adhesion formation or adhesion reformation, respectively.[17] Postoperative adhesions form after abdominal surgery due to injured peritoneal surfaces and pose a life-long risk of small bowel obstruction, chronic pain, infertility and complications during reoperations.[6] Factors contributing to postoperative adhesion formation include extent of surgical trauma, abrasion, desiccation and exposure to foreign materials such as gauze and talc or starch glove powder. Depending on the extent of surgical trauma, it has been suggested that laparoscopic surgery reduces the tendency to form adhesions compared to open surgery.[16]

Adhesions causing SBO are classed as either matted adhesions or solitary band adhesions.[16] In patients with matted adhesions, SBO results from angulation and kinking or from torsion of the intestines while with adhesive bands, SBO results from compression of the intestine caused by the band itself.[5] This is of clinical importance since SBO from solitary bands is more likely to be associated with bowel ischemia and necrosis caused by strangulation compared to SBO caused by matted adhesions.[16] Bands formed can be so tight as to case ischemia and necrosis of the affected small bowel segment leading to complete transaction.[1] The nature of adhesion-related bowel obstruction seems to affect the recurrence prognosis. Readmission after surgery for SBO caused by matted adhesions has been estimated to be 49% compared to 25% for patients with a solitary band.[16] Our patient had matted adhesions in a segment of terminal ileum causing SBO with healthy bowel. There is no history of readmission for SBO during 10 months follow up period.

The term virgin abdomen predates widespread computed tomography (CT) imaging, which now elucidates most SBO etiologies.[10] The utility of the CT scan of the abdomen in guiding operative management is increasingly relevant as the technology becomes readily available and efficient.[11] CT has been shown to be useful in determining the site, level and cause of SBO.[17] However adhesions are not clearly visualized in most cases and their identification remains a diagnosis of exclusion.[18] This diagnosis is based on the finding of an abrupt change in bowel caliber without evidence of another cause of obstruction.[17] In our case CECT was suggestive of SBO without evidence of any etiology. Recent advances in spatial resolution using multi-detector CT (MDCT) have enabled detailed assessment of the configuration of the SBO site.[5] The MDCT findings at the transition zone reflect the mechanisms of SBOs and provide useful information for differentiating between matted adhesions and adhesive bands.[5]

Despite recent efforts to re-evaluate clinical management standards, the evidence of underlying etiologies of SBO in the virgin abdomen and the safety of foregoing surgery remains unclear.[10] Surgical exploration is still considered mandatory in the setting of SBO in a virgin abdomen by some large centers.[4] Successful non-operative SBO management in patients without abdominopelvic surgery history may risk missing a potentially malignant etiology.[10] The main argument against mandatory exploration is a high rate of negative exploration.[4] However, no study had robust follow-up data to confirm that foregoing operative management of SBO in patients without abdominopelvic surgery history is innocuous.[10] An evidence-based algorithm for managing patients with SBO without abdominopelvic surgery history requires further study.[10]

CONCLUSION

Adhesive small bowel obstruction remains prevalent despite the absence of previous abdominal surgery. The treatment of choice for adhesive small bowel obstruction remains controversial, and relies on the surgeon's judgment. Adhesions are likely when CT confirms the presence of SBO and fails to reveal a cause. A high index of suspicion is therefore necessary for de novo adhesions in SBO in virgin abdomen without CT evidence of any etiology, hence early surgical intervention should be considered. An evidence-based algorithm for managing patients with SBO in virgin abdomen requires further study.

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