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Abdominal compartment syndrome secondary to myxedema ileus

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INTRODUCTION

Acute pseudo-obstruction of the colon is known as Ogilvie syndrome (OS). It was first described by Sir Heneage Ogilvie in 1948 [1]. It presents clinically as acute dilatation of cecum and colon. There are varied causes of this acute dila- tation. The term myxedema ileus (MI) is used when Ogilvie is due to hypothyroidism. Perforation is the most common complication associated with acute megacolon [2]. Abdominal compartment syndrome (ACS) complicating colonic obstruc- tion has been described. To the best of our knowledge, acute pseudo-obstruction presenting with compartment syndrome due to MI has never been reported. We are reporting a 70-year female who presented to us in the emergency department withacute compartment syndrome due to MI.

CASE REPORt

A 70-year-old female presented to emergency with a history of pain abdomen and nonpassage of flatus and stools for the past 4 days and altered sensorium for the past 1 day. At present, the patient was not on any medications.There was a history of constipation off and on for which she used to take laxatives. The patient also had a history of easy fatigability. On examination pulse rate was 120/min, temperature was 36.2°C, and blood pressure was 100/68 mmHg. Urine output of the patient was 100 mL in the past 8 h. The abdomen was distended, and the intra-abdominal pres- sure (IAP) of the patient was 40 cm of water (29.42 mmHg) measured using bladder pressure monitoring through Foley's catheter. On digital rectal examination, there was mucus with

no other significant finding.. A flatus tube was inserted per rectally which was not successful in relieving obstruc- tion. A clinical diagnosis of intestinal obstruction with acute compartment syndrome was made. Routine hematological investigations were done which revealed hemoglobin of 8.6 g/dL, total leukocyte count was 12,300/□L, and platelet count was 275 thousand/□L. Serum glucose was 100 mg/dL, blood urea nitrogen was 96 mg/dL, serum creatinine was 2.2 mg/ dL, and serum electrolytes were normal. Arterial blood gas examination showed pH of 7.21, pO_2 of 40 mmHg, pCO_2 of 45 mmHg, and HCO_3 of 21.4 mmol/L. Thyroid function tests of the patient revealed thyroid function test (TSH) of 94.2 mIU/L, T3 was 3.12 ng/dL, and T4 was 2.87 □g/dL. X-rays of the patient showed dilated large bowel with raised diaphragm [Figure 1]. With the diagnosis of ACS with acute intestinal obstruction, the patient was taken up for urgent exploratory laparotomy. On exploration, the entire large bowel from cecum to rectosigmoid junction was massively dilated [Figure 2]. The

small bowel was collapsed. There was no transition point. There was no growth, volvulus, or adhesions. Enterotomy and colonic decompression were done. Hence a final diagnosis of acute compartment syndrome secondary to acute pseudo-obstruction due to myxedema ileus was made. The patient improved on the postoperative day 3. Postoperative period was uneventful, and the patient was discharged on the postoperative day 8. On 1-month follow-up, there were no complaints, there was no episode of constipation, ability to tolerate cold had improved, and in general, there was a feeling of well-being.

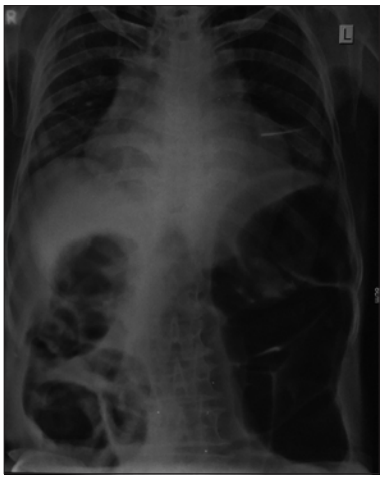

Figure 1: Abdominal X-ray of the patient showing distended large bowel with elevated diaphragm

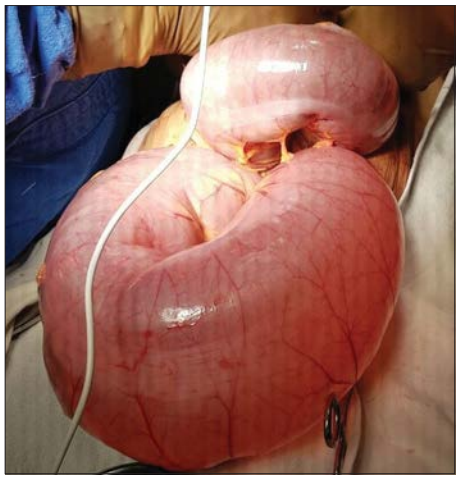

Figure 2: Intraoperative image showing massively dilated large bowel loops.

DISCUSSION

Pseudo-obstruction is defined as distension of colon without any physical cause [3]. It may be primary (congeni- tal) and secondary (acquired). It can also be classified into acute or chronic. OS is an acute colonic pseudo-obstruc- tion. OS due to hypothyroidism is known as MI. Decreased motility of intestine in MI may present as obstipation and decreased bowel movement frequency, paralytic ileus, or pseudo-obstruction with massive dilatation of the cecum (diameter >10 cm) and right colon (acute megacolon) and pseudo-volvulus [4]. Usually, in OS, cecal dilatation is maximum which is in accordance with Laplace's law, which states that the intraluminal pressure needed to stretch the wall of a hollow tube is inversely proportional to its diameter. The transition between dilated and collapsed bowel is usually near the splenic flexure but can occasionally occur in the distal or sigmoid colon.

Colonic motility is controlled by neurogenic and myogenic factors, so any myopathy of colon or imbalance between sympa- thetic and parasympathetic innervation of colonic musculature may manifest as pseudo-obstruction [5]. Sympathetic inner- vation leads to contraction and parasympathetic to dilatation. Several theories have been proposed to explain hypoactive gut in hypothyroidism, including autonomic neuropathy, decreased impulse transmission at the myoneural junction, intestinal edema and ischemia, reduction in adrenergic receptors, and intestinal myopathy. Deposition of mucopolysaccharides in interstitium leads to dissociation of Auerbach's and myenteric plexus from muscles leading to atony and later

degeneration of muscle fibers [6]. Basal metabolic rate is reduced in hypo- thyroidism and so is the activity of the gastrointestinal system. Thyroid hormone deficiency decreases transepithelial flux transport by inhibiting Cl^{-}/HCO^{3-} anion exchange with a sub- sequent effect on intestinal motility [7]. There is a deficiency of nitric oxide in hypothyroidism which is further responsible for gut motility disorder [8]. The increased intraluminal pressure leads to ischemia with longitudinal splitting of the serosa, herniation of the mucosa, and perforation. Perforation of cecum is the most common. Dehydration, electrolyte imbalance, hyponatremia, and hypoka- lemia may be seen [2]. ACS can occur after mechanical colonic obstruction, but MI presenting as ACS as seen in our case has never been reported.

The abdomen is a closed cavity with a normal pressure of 5 mmHg. IAP of >10 mmHg is labeled as abdominal hyperten- sion. A sustained increase of IAP ≥20 impairs organ perfusion; causing new organ dysfunction with improvement on decom- pression is labeled as ACS. Decreased abdominal wall compliance, increased intra-abdominal contents, and increased capillary leak increase IAP. Massive dilatation of colon in our case led to ACS. There are various methods used for measur- ing IAP. IAP can be measured by direct and indirect methods; transvesical is the most common bedside indirect technique used.

MI is usually managed conservatively. Conservative man- agement involves bowel rest, decompression by nasogastric tube, and rectal tube at least for 2 days. Prokinetic drugs such as neostigmine, domperidone, metoclopramide, and erythromycin are used in conservative management [9]. Surgical explora- tion may precipitate myxedema coma. Indications for surgery are cecal distension of >12 cm, bowel ischemia, and perforated bowel. Furthermore, in patients like ours, presenting with com- partment syndrome active decompression and damage control laparotomy is required. Any associated electrolyte abnormali- ties should be corrected cautiously as they may aggravate pseudo-obstruction. Decompression alone will recur until myx- edema is treated. Intravenous levothyroxine, triiodothyronine, or oral thyroxine is used to control hypothyroidism [10].

CONCLUSION

Myxedema as a cause of pseudo-obstruction is extremely rare. Treatment of MI is usually conservative with surgery reserved only for special circumstances. MI presenting as ACS has been rarely reported and requires urgent decompression.

Declaration of patient consent

We certify that we have obtained appropriate patient consent form. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initial will not be published and due efforts have been made to conceal her identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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