



Case Report

Abdominal compartment syndrome secondary to myxedema ileus

Vishal Thakur, Jagdish Kumar Gupta, Archit Gupta*

Department of General Surgery, IGMCM, Shimla, Himachal Pradesh, India

Submission : 30-Jan-2019
Revision : 20-Feb-2019
Acceptance : 18-Mar-2019
Web Publication : 23-Apr-2019

ABSTRACT

Acute pseudo-obstruction of the colon is known as Ogilvie syndrome (OS). There are varied causes of OS. Myxedema ileus (MI) as a cause of acute pseudo-obstruction is rare. Surgery in cases of MI is reserved only in cases of cecal distension of >12 cm, bowel ischemia, and perforated bowel. We present a rare case of a 71-year-old female who was operated for abdominal compartment syndrome secondary to MI.

KEYWORDS: *Myxedema ileus, Ogilvie syndrome, Pseudo-obstruction*

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 dilatation. 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 obstruction 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 71-year-old female who presented to us in the emergency department with acute compartment syndrome due to MI.

CASE REPORT

A 71-year-old female presented to emergency with a history of pain abdomen and nonpassage of flatus and stools for the past 3 days and altered sensorium for the past 1 day. There was a history of thyroid surgery 20 years ago. At present, the patient was not on any medications (even not taking thyroid replacement therapy). There was a history of constipation off and on for which she used to take laxatives. Along with this, there was cold intolerance and generalized xerosis. The patient also had a history of easy fatigability. On examination, the Glasgow coma scale of the patient was E1V1M3 (5/15), pulse rate was 120/min, temperature was 36.2°C, and blood pressure was 74/40 mmHg. The patient was cyanosed with a respiratory rate of 36/min. The oxygen saturation of the patient as measured by pulse oximeter was 35%. Urine output of the patient was 100 mL in the past 8 h. The abdomen was distended, and the intra-abdominal pressure (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. The patient was immediately intubated and was resuscitated. A flatus tube was inserted per rectally which was not successful in relieving obstruction. 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/μL, and platelet count was 275 thousand/μL. 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, pO₂ of 40 mmHg, pCO₂ of 45 mmHg, and HCO₃ 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 μg/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. In the postoperative period, the patient was started on intravenous thyroxine. The patient improved and was extubated on the postoperative day 3. Postoperative period was uneventful, and the patient was discharged on the postoperative day 8 on treatment for hypothyroidism with oral thyroxine. On 1-month follow-up, there were no complaints, there was no

*Address for correspondence:

Dr. Archit Gupta,
Department of General Surgery, IGMCM, Shimla - 171 001,
Himachal Pradesh, India.
E-mail: archit9th@gmail.com

Access this article online

Quick Response Code:



Website: www.tcmjmed.com

DOI: 10.4103/tcmj.tcmj_31_19

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Thakur V, Gupta JK, Gupta A. Abdominal compartment syndrome secondary to myxedema ileus. Tzu Chi Med J 2020;32(2):219-21.



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

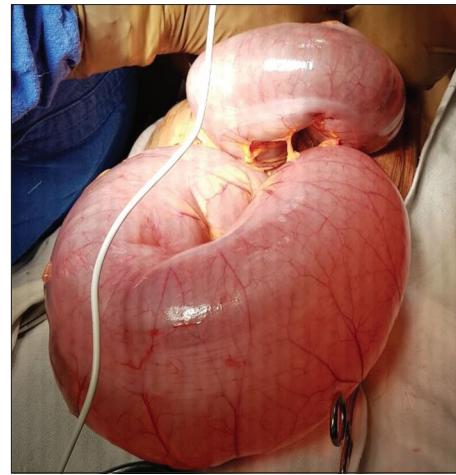


Figure 2: Intraoperative image showing massively dilated large bowel loops

episode of constipation, ability to tolerate cold had improved, and in general, there was a feeling of well-being.

DISCUSSION

Pseudo-obstruction is defined as distension of colon without any physical cause [3]. It may be primary (congenital) and secondary (acquired). It can also be classified into acute or chronic. OS is an acute colonic pseudo-obstruction. 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 sympathetic and parasympathetic innervation of colonic musculature may manifest as pseudo-obstruction [5]. Sympathetic innervation 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 hypothyroidism and so is the activity of the gastrointestinal system. Thyroid hormone deficiency decreases transepithelial flux transport by inhibiting $\text{Cl}^-/\text{HCO}_3^-$ anion exchange with a subsequent 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 hypokalemia 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 hypertension. A sustained increase of IAP ≥ 20 impairs organ perfusion; causing new organ dysfunction with improvement on decompression 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 measuring 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 management 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 exploration 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 compartment syndrome active decompression and damage control laparotomy is required. Any associated electrolyte abnormalities should be corrected cautiously as they may aggravate pseudo-obstruction. Decompression alone will recur until myxedema 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.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Ogilvie H. Large-intestine colic due to sympathetic deprivation; a new clinical syndrome. *Br Med J* 1948;2:671-3.
2. Durai R. Colonic pseudo-obstruction. *Singapore Med J* 2009;50:237-44.
3. Saunders MD. Acute colonic pseudo-obstruction. *Best Pract Res Clin Gastroenterol* 2007;21:671-87.
4. Vanek VW, Al-Salti M. Acute pseudo-obstruction of the colon (Ogilvie's syndrome). An analysis of 400 cases. *Dis Colon Rectum* 1986;29:203-10.
5. Maloney N, Vargas HD. Acute intestinal pseudo-obstruction (Ogilvie's syndrome). *Clin Colon Rectal Surg* 2005;18:96-101.
6. Douglass RC, Jacobson SD. Pathologic changes in adult myxedema: Survey of 10 necropsies. *J Clin Endocrinol Metab* 1957;17:1354-64.
7. Tenore A, Fasano A, Gasparini N, Sandomenico ML, Ferrara A, Di Carlo A, et al. Thyroxine effect on intestinal $\text{Cl}^-/\text{HCO}_3^-$ exchange in hypo- and hyperthyroid rats. *J Endocrinol* 1996;151:431-7.
8. Tomita R, Fujisaki S, Ikeda T, Fukuzawa M. Role of nitric oxide in the colon of patients with slow-transit constipation. *Dis Colon Rectum* 2002;45:593-600.
9. Jain A, Vargas HD. Advances and challenges in the management of acute colonic pseudo-obstruction (Ogilvie syndrome). *Clin Colon Rectal Surg* 2012;25:37-45.
10. Schulberg SP, Meytes V, Morin N, Ferzli G, Adler E, Kopatsis A, et al. Myxedema pseudovolulus: Case series and review of the literature. *Ann Laparosc Endosc Surg* 2017;2:56-61.