Hypothyroidism-Induced Stercoral Sigmoid Colonic Perforation

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A 49-year-old woman presented for evaluation of diffuse abdominal pain, nausea, and nonbilious, nonbloody vomiting.

According to the Centers for Disease Control and Prevention, abdominal pain is the leading reason for ED visits in the United States, with approximately 10 million visits per year.[1] Though a large number of presentations are due to nontraumatic causes of abdominal pain, one etiology is among the most time-sensitive and critical diagnoses: acute colonic perforation.

Colonic perforations can be caused by diverticulitis, trauma, malignancy, ulcerative colitis, and other etiologies.[2] A rare, yet life-threatening cause of colonic perforation, of which only a few cases have been documented in the literature, is stercoral colonic perforation.[2]

Regardless of the etiology, the critical actions for any colonic perforation are quick recognition, medical stabilization, and surgical evaluation. This case report highlights the diagnosis and treatment of acute stercoral colonic perforation with peritonitis secondary to hypothyroidism.

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Case

A 49-year-old woman with a medical history significant for hypothyroidism pre-

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sented to the ED for evaluation of diffuse abdominal pain, nausea, and nonbilious, nonbloody vomiting that started in the early evening of presentation. The patient denied any previous pain or associated symptoms, and said she had a small, hard bowel movement 1 day prior to arrival. She began experiencing mild abdominal pain on the morning of presentation. Her symptoms acutely worsened at approximately 5:00 PM. She denied having any diarrhea, melena, or hematochezia. Her surgical history was significant for a cesarean delivery. She denied any history of small bowel obstruction. The patient admitted that she had stopped taking levothyroxine (150 mcg daily) prescribed for hypothyroidism approximately 1 month earlier, but had been restarted on it approximately 2 to 3 days prior to her presentation.

Table. Causes of Hypothyroidism[7,8]

Primary
Agenesis
Gland destruction
Surgical removal
Irradiation
Autoimmune disease
Infiltrative process
Idiopathic atrophy
Inhibition of thyroid hormone synthesis and release
Iodine deficiency
Excess iodine
Drugs
Inherited enzyme defect
Transient hypothyroidism
Postsurgical
Postpartum
Thyroiditis
Secondary
Hypothalamic disease
Pituitary disease
Genetic
Sheehan's syndrome (also known as Simmond's syndrome)
Surgery
Trauma
Tumor
Resistance to thyroid hormone

On physical examination, her vital signs were: heart rate, 156 beats/min; blood pressure, 134/84 mm Hg; respiratory rate, 20 breaths/min, and temperature, 97.4°F. The patient appeared ill and diaphoretic, writhing on the stretcher. Abdominal examination was significant for diminished bowel sounds, diffuse abdominal distension, rigidity, and tenderness with light palpation.

Laboratory evaluation showed an elevated lactic acid level of 7.7 mmol/L, a white blood cell count of 7,200 cells/mm^3 (segment form, 69.5%), and the following abnormal blood chemistry results: creatinine, 2.08 mg/dL; aspartate aminotransferase, 176 U/L; alanine aminotransferase, 138 U/L; and thyroid-stimulating hormone (TSH), 225.3 mcIU/mL. Other laboratory results were within normal range. Her electrocardiogram showed sinus tachycardia with a rate of 154 beats/min, a QTc within normal limits, and no ST elevations or depressions.

An abdominopelvic computed tomography (CT) scan revealed free air, free fluid, and possibly stool within the abdomen and pelvis. The findings were consistent with a ruptured hollow viscus, possibly a sigmoid colonic perforation. The radiologist also noted hepatomegaly and significant hepatic steatosis. A surgeon was immediately notified and evaluated the patient in

the ED. The working diagnosis was stercoral colonic perforation secondary to severe hypothyroidism, and the patient was taken emergently to the operating room for repair.

Intraoperatively, the patient underwent exploratory laparotomy, which revealed gross fecal contamination of the abdomen. The surgeon noted that there was fecal staining along the serosal surface of the small bowel and throughout the pelvis. There were also large, hard stool balls outside of the colon. The perforation was along the mesenteric surface of the sigmoid just above the rectosigmoid junction.

The abdomen was copiously irrigated, the perforated segment was resected, and a Hartmann colostomy was created. The diagnosis was stercoral sigmoid perforation with peritonitis, and the patient was transferred to the intensive care unit for antibiotic treatment and further medical care, including intravenous (IV) levothyroxine.

She was extubated uneventfully on postoperative day 2, and the acute renal failure improved with supportive care only. Her bowel function slowly returned without complication. She was switched to oral levothyroxine on postoperative day 3. On day 13, she was given strict instructions for continuation of her thyroid medication and close monitoring for postsurgical complications, and was discharged home with appropriate follow-up.

Discussion

Multiple contributing factors can lead to bowel perforation. In this case, severe hypothyroidism with constipation caused a colonic perforation. Our patient had severe constipation that increased intraluminal pressure, causing the bowel wall to become ischemic and subsequently perforate.[3] Any disease that causes significant constipation or obstruction of transit could lead to the same catastrophic result.

According to Huang et al,[4] as of 2002, fewer than 90 cases of general stercoral bowel perforation had been reported, with no clear age range. However, patients in their mid-50s to mid-60s appear to be the most commonly affected age group.[4] Our patient was younger than this age group, making identification of the problem by age alone difficult.

Hypothyroidism

The incidence of hypothyroidism in iodine-replete communities varies between 1% to 2% of the general population.[5] The condition is more common in older women, affecting approximately 10% of those over age 65 years. In the United States, the prevalence of biochemical hypothyroidism is 4.6%; however, clinically evident hypothyroidism is present in only 0.3%.[6] Common causes for hypothyroidism are listed in the **Table**.[7,8] Typical symptoms include fatigue, weight gain, constipation, and cold and heat intolerance.[6] Hypothyroidism typically is diagnosed by a patient's TSH and total and free thyroxine (T_4) and L-triiodothyronine (T_3) laboratory values.[7] Treatment includes starting a synthetic thyroid hormone, such as levothyroxine.

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Myxedema Coma

Untreated, hypothyroidism can lead to potentially fatal conditions, such as myxedema coma, which is characterized by hypothermia, hypotension, bradycardia, respiratory depression, and altered mental status.[7] Severe myxedema coma can result in cardiovascular collapse, and eventual death. Electrocardiography findings of severe hypothyroidism include bradycardia, low-voltage QRS, and widespread T-wave inversions.[7] Our patient was tachycardic

and did not have any acute findings to suggest myxedema coma.

Treatment for myxedema coma includes supportive care with ventilatory support and pressor support if necessary. Patients should be given IV hydrocortisone, 100 mg, to treat possible adrenal insufficiency and T_4, 4 mcg/kg by slow IV infusion.[7] Caution should be taken if giving a patient T_3 due to the risk of dysrhythmias and myocardial infarction (MI).[7] As our patient was not displaying myxedema coma, the surgeon elected not to start IV thyroid replacement to avoid exacerbating the patient's tachycardia and possibly precipitating an MI intraoperatively.

Conclusion

Our case underscores the importance of promptly recognizing the signs and symptoms of stercoral colonic perforation in patients who present with nontraumatic abdominal pain accompanied by nausea and nonbilious, nonbloody vomiting. Although stercoral colonic perforation is a rare cause of nontraumatic abdominal pain, as with any type of colonic perforation, it constitutes a life-threatening medical emergency. As our case illustrates, prompt diagnosis through a thorough history taking, physical examination, and laboratory and imaging studies is critical to ensure medical stabilization and surgical management to reduce morbidity and mortality.

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