

An Obstructing Fecal Bezoar in a Patient with Scleroderma with Successful Colonoscopic Disimpaction

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ABSTRACT

Scleroderma (SSc) is a disease caused by collagen deposition resulting in fibrosis within multiple organs, including the gastrointestinal tract, skin, joints, kidneys, lungs, and heart. We report a rare case of a patient with diffuse SSc who presented with a large bowel obstruction from a fecal bezoar impaction. The bezoar was successfully removed using colonoscopy after lavage, cold forceps, balloon dilator, and cap-assisted disimpaction. We demonstrate that patients with SSc are at risk for bezoar formation and true mechanical obstruction in the lower gastrointestinal tract, which may require more aggressive endoscopic treatment if conservative measures fail.

INTRODUCTION

Scleroderma (SSc) is a systemic disease characterized by collagen deposition that frequently involves the gastrointestinal (GI) tract. GI involvement of SSc manifests in various ways including gastroesophageal reflux (GERD), dysphagia, small intestinal bacterial overgrowth (SIBO), constipation, diarrhea, and intestinal pseudo-obstruction. Rarely, dysmotility from colonic involvement of SSc can lead to fecal bezoar formation, which can lead to intestinal obstructions.¹ We report a case of a patient with SSc who suffered a true mechanical bowel obstruction secondary to fecal impaction in the transverse colon. Her obstruction was successfully disimpacted using colonoscopy.

CASE REPORT

A 44-year-old woman with diffuse SSc presented with abdominal pain, nausea, and vomiting for 2 days. Her pain was crampy, diffuse, intermittent, and progressive. It was accompanied by diminished appetite, several episodes of vomiting, and small-volume diarrhea. She tried taking loperamide for the diarrhea with no relief. She denied any fevers, chills, hematemesis, melena, hematochezia, or constipation. Her medical history was also positive for hypertension and GERD, but otherwise, she reported no personal or family history of any GI conditions. Medications included oral prednisone 7 mg daily and a proton-pump inhibitor.

On physical examination, she was cachectic with SSc-related skin changes. Her abdomen was soft but distended and tender in all 4 quadrants. A firm mass was palpated in the right upper quadrant. Abdominal computed tomography with contrast revealed a 6-cm fecal bezoar in the transverse colon with proximal large and small bowel dilatation (Figure 1). Her serum potassium was 3 mEq/L and magnesium was 0.67 mmol/L; otherwise, her laboratory values were within normal limits.

The patient was diagnosed with a large bowel obstruction secondary to a fecal bezoar. She was given 4 liters of polyethylene glycol (PEG) by mouth, but she was unable to tolerate the full dose. Her symptoms persisted despite passing flatus and watery stool. Repeat abdominal imaging by x-ray showed minimal movement of the stool ball, therefore colonoscopy was recommended for disimpaction (Figure 2). During colonoscopy, the fecal bezoar was found at the hepatic flexure (Figure 3). Mechanical disimpaction was attempted with a rat

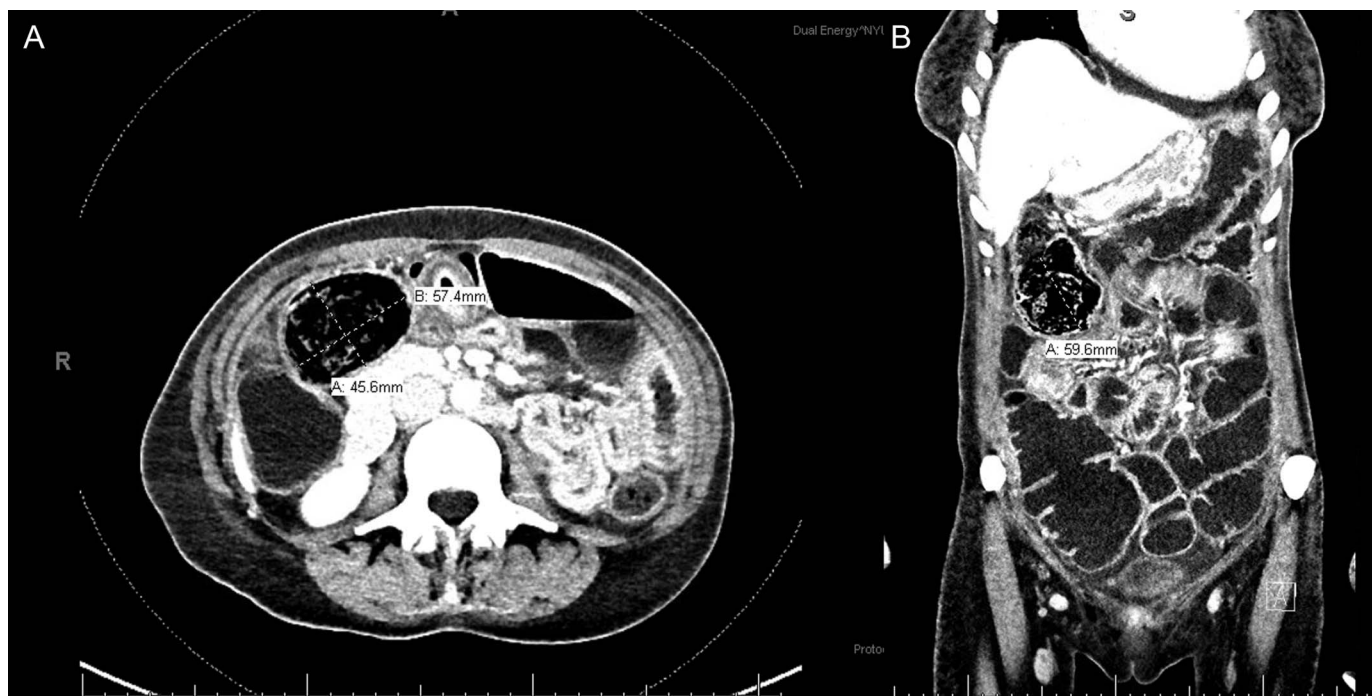


Figure 1. Contrast-enhanced abdominal computed tomography. (A) Axial view showing 4.6 × 5.7 cm mass with mottled appearance consistent with fecal bezoar in transverse colon along with inflammatory bowel wall thickening. (B) Coronal view showing moderate diffuse fluid-filled distention of loops of small and large bowel proximal to fecal bezoar in the proximal transverse colon, consistent with a developing upstream large and small bowel obstruction.

tooth, cold snare, and aggressive water irrigation with partial success. The patient then continued oral PEG treatment to further disrupt and pass the bezoar after the colonoscopy; however, abdominal x-ray the following morning revealed no significant improvement of her obstruction. Colonoscopy was repeated and revealed the fecal bezoar to be in the same location at the hepatic flexure. Several balloon dilation sweeps with a controlled radial

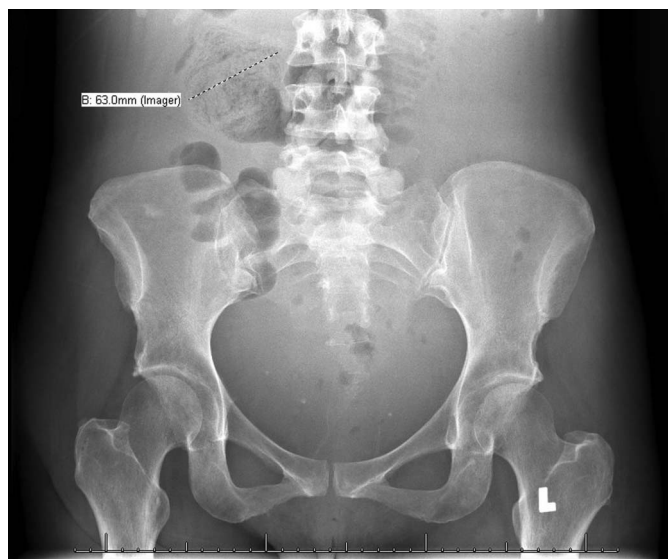


Figure 2. Plain abdominal radiograph showing the 6-cm fecal bezoar, unchanged in location in the transverse colon.

expansion wire-guided balloon (Boston Scientific, Marlborough, MA) were used to advance the bezoar out of the hepatic flexure, further distal in the transverse colon for better access. There was poor visualization proximal to the bezoar, and the distal tip of the balloon was guided blindly. Repeated washings were used to soften the bezoar so that tools such as cold biopsy forceps, rat tooth forceps, and a snare and lasso technique could more effectively break the bezoar into smaller pieces that were easier to pass spontaneously. Cap-assisted colonoscopy was helpful in protecting the camera from debris obstructing the view. After 3 hours of irrigation and mechanical disimpaction, the obstruction was relieved.

The patient's GI symptoms resolved postprocedure. After she could tolerate a normal diet, she was discharged with an aggressive oral bowel regimen and motility specialist follow-up.

DISCUSSION

SSc can affect any part of the GI tract, resulting in fibrosis and dysmotility of the gut wall. SSc's GI manifestations account for 6%–12% of the mortality rate of these patients.² The GI pathophysiology of SSc is explained by Sjogren's theory.³ In response to an unknown initial injury, collagen deposits in blood vessels within the gut wall.⁴ This causes recurrent vascular derangement, compression of nerves, and autoimmune-mediated injury.³ Immune cells infiltrate the smooth muscle, causing dysfunction, atrophy, and fibrosis.

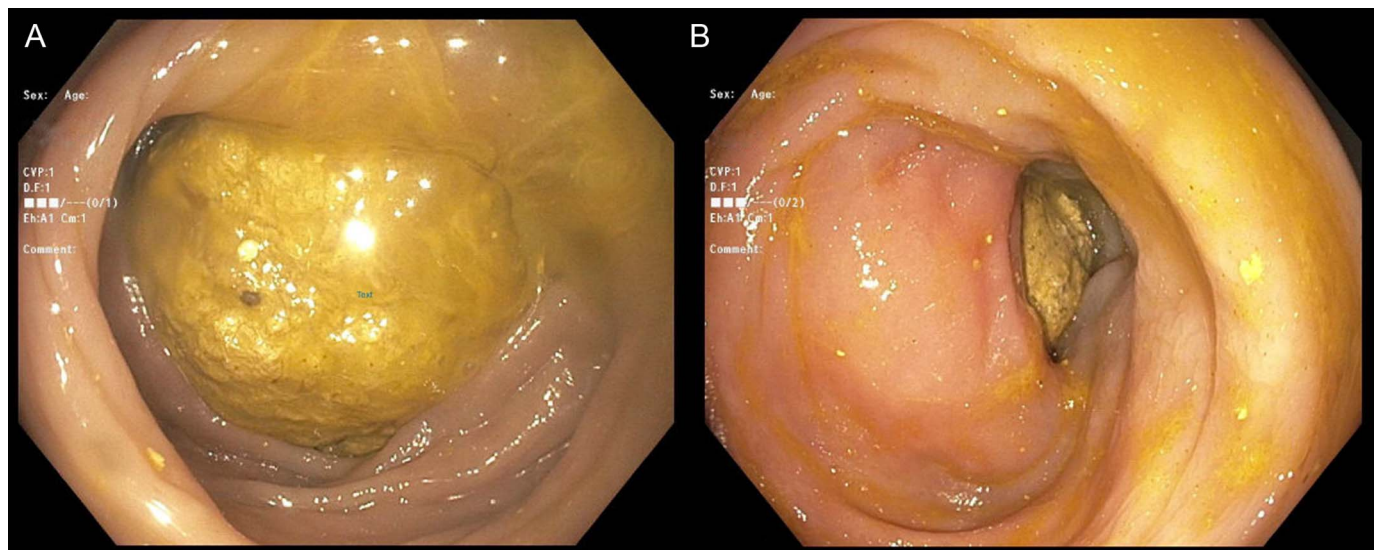


Figure 3. Colonoscopy showing (A) the full view of fecal bezoar in transverse colon prior to disimpaction, and (B) the fecal bezoar lodged in the hepatic flexure of the transverse colon prior to disimpaction.

The most common GI manifestation of SSc is esophageal dysfunction due to fibrosis of the lower two-thirds of the esophagus. It can lead to an incompetent lower esophageal sphincter, inadequate peristalsis, GERD, and/or stricture formation.⁵ Another common site of SSc involvement is the small intestine, resulting in hypomotility and stasis. Consequently, 10%–30% of these patients have SIBO, leading to malabsorption and diarrhea.⁶

Less commonly, SSc results in colonic hypomotility. Symptoms most often include bloating, abdominal pain, and constipation, which can ultimately result in overflow diarrhea that passes around the hardened stool. The progressive hypomotility of the colon can result in pseudo-obstruction, which refers to obstruction without a mechanical cause. However, in rare instances, a true mechanical obstruction can occur because of stool completely blocking the large intestine, but few case reports exist in the literature.¹ This rarity may be because SIBO and malabsorption lead to small intestinal diarrhea, which often mitigates stool impaction.² Nonetheless, for patients with severe colonic SSc, stool transit can be significantly impaired, allowing a fecal bezoar to accumulate over time despite overflow diarrhea.

Regarding management of bezoar-induced obstruction in patients with SSc, physicians should consider oral osmotic laxatives such as PEG, prokinetic agents, and, if the obstruction is distal to the transverse colon, mineral oil enemas.⁷ If conservative measures fail, colonoscopic intervention may be successful.⁸ In cases of mechanical obstruction, in all patients, surgical management is reserved for patients who do not respond to endoscopic treatment or who have bowel perforation. Limited resection of the colon outside of these circumstances is unlikely to be helpful, especially in patients with SSc due to the pan-colonic fibrosis inherent to the disease.⁹ Surgery is therefore almost never performed on these patients. For prevention of bowel

obstruction, the same conservative measures (PEG, prokinetic agents and mineral oil enemas) can be used in lower doses and frequencies, both in patients with SSc and other at-risk patients.¹⁰

Because of its rarity, recurrence rates—and epidemiologic data in general—for true bowel obstruction in patients with SSc are not known, although some data exist pertaining to pseudo-obstruction. One study showed that the mortality rate from acute intestinal pseudo-obstruction in patients with SSc was 16%.¹¹ Female patients who presented with pseudo-obstruction were more likely to have recurrence than male patients, although male patients had a higher mortality rate after pseudo-obstruction [ibid].

We present a rare case of a true mechanical bowel obstruction secondary to fecal bezoar formation in a patient with systemic SSc. The patient's obstruction was successfully relieved with medical and endoscopic therapy. Although most data on SSc-related bowel obstructions focus on pseudo-obstructions, our report highlights the potential for true mechanical obstruction in the GI tract. We demonstrate that endoscopic intervention can successfully disimpact obstructing fecal bezoars in patients with SSc.

DISCLOSURES

Author contributions: R. Sarnoff and B. Girmay wrote the manuscript. R. Mocharla edited the manuscript. All other authors contributed equally to the manuscript. R. Sarnoff is the article guarantor.

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Informed patient consent was obtained for this case report.

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