

Bowel Perforation Due to Immobilization after Resurfacing Thumb with Anterolateral Thigh Free Flap in an Elderly Diabetic Woman

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Inevitable immobilization after surgery on lower extremities can induce chronic constipation. Elderly diabetic women usually express ambiguous gastrointestinal symptoms and signs. We present here a case of panperitonitis developed from severe fecal impaction in an elderly diabetic woman after hand reconstruction using material harvested from the lower extremities. A 68-year-old diabetic female underwent anterolateral thigh free flap and wound revision twice on the left thumb. Three weeks after surgery, she complained about mild abdominal pain though she had daily defecation. Despite encouraging ambulation, her compliance was low. Resection of the sigmoid colon and colostomy were performed after diagnosis with bowel perforation. However, the patient went into septic shock and died with multiorgan failure after the guardians issued a DNR (do not resuscitate) order. For preventing bowel perforation, increased uptake of dietary fiber and early ambulation postoperatively should be encouraged, after even hand surgeries.

Key Words: Disabled persons, Constipation, Aged, Diabetes complications

Bowel perforation is an uncommon event, which is usually caused by malignancy, amoebic colitis, diverticular disease, spontaneous perforation, stercoral ulceration, corticosteroid therapy, trauma, or ulcerative colitis. Stercoral perforation of the bowel is rare. Moreover, conditions leading to bowel perforation are frequently subclinical in the early stages. Patients with diabetic mellitus (DM) may be particularly likely to develop life-threatening stercoral perforation, as these patients often have gastrointestinal (GI) sensory-motor abnormalities, potentially involving any part of the GI tract.¹ Elderly women with DM often express ambiguous GI symptoms and signs.^{1,2}

Immobilization required after reconstructive surgery involving the lower extremities can produce constipation, which may also increase the risk of bowel perforation. We herein present a case of panperitonitis arising from stercoral perforation associated with severe fecal impaction in an elderly diabetic woman after thumb resurfacing with an anterolateral thigh free flap.

CASE REPORT

A 68-year-old female (146 cm, 36 kg) was diagnosed with DM 5 years previously, for which she was receiving metformin. She had a long history of severe, chronic constipation requiring medical treatment. The patient presented to our institution with cellulitis, which occurred after needle drainage of spontaneous localized swelling of her thumb. After failure of treatment with antibiotics and incision and drainage at local clinics, she visited our institution for further management when her thumb developed dry gangrene wrapped with eschar (Fig. 1). Computed tomography (CT) angiography showed narrowing of both proper digital arteries supplying the thumb, leading to the diagnosis of mummified thumb due to peripheral artery obstruction. Soft tissue coverage was performed with a thinned anterolateral thigh free flap, with the descending branch of the lateral femoral circumflex artery and venae comitantes anastomosed end-to-

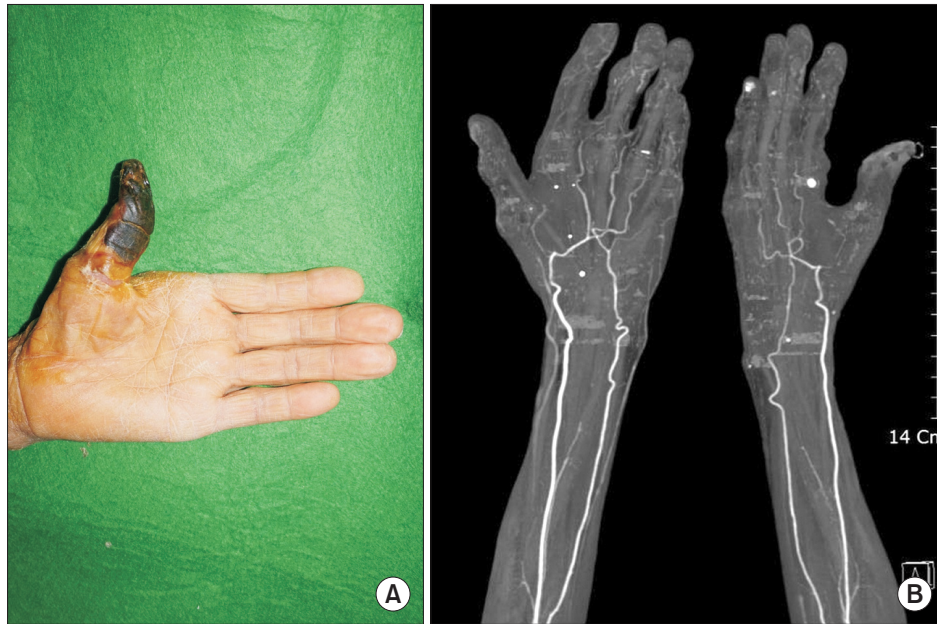


Fig. 1. Mummified left thumb (A) and subtle narrowing of proper digital arteries on computed tomography angiography (B).

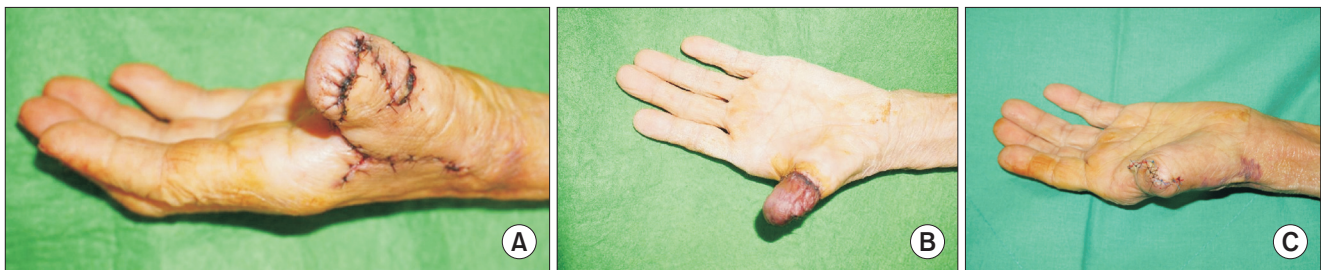


Fig. 2. Photograph of the thumb on postoperative days 4 (A) and 8 (B) after the initial flap surgery and the day after revision surgery (C).

side to the palmar arch. A week of absolute bed rest (with daily magnesium oxide) was recommended to facilitate donor site wound healing. The hand was elevated and immobilized in a splint. Flap color was pinkish and exhibited good blanching and clear Doppler sounds until the seventh postoperative day. On that day, the splint was removed because of shoulder and hand discomfort. On postoperative day 8, the flap turned dark and Doppler sounds and blanching were unclear (Fig. 2). By postoperative day 12, demarcation had taken place, and delayed flap failure was diagnosed. The patient underwent revision surgery 23 days after the initial flap surgery, which stabilized the condition of the thumb.

At 22 days after her initial flap operation, the patient abruptly reported mild abdominal pain, although she had daily bowel movements. Despite being encouraged to ambulate, her mobil-

ity had been limited. Chest x-ray and physical examination were normal, with no rebound tenderness or fever (Fig. 3). Medical staff recommended further radiologic studies, but the patient refused. At 24 days after flap surgery (i.e., day after the revision surgery), the patient had a bowel movement that was small, but otherwise normal. Physical examination showed normal bowel sounds and a soft abdomen with mild diffuse tenderness, but no signs of peritonitis. Her vital signs were normal (including a temperature of 36.4°C), but the white cell count, neutrophil fraction, and C-reactive protein were elevated (19,970 count/ μ L, 94.9%, and 146.5 mg/L, respectively). Erect abdominal x-ray and abdominal-pelvic CT showed pneumoperitoneum, diffuse fecal impaction in the colon, obvious luminal distention of the bowel, and other findings suggesting stercoral colitis and panperitonitis with fluid collection (Fig. 4, 5).



Fig. 3. Normal findings on chest x-ray.

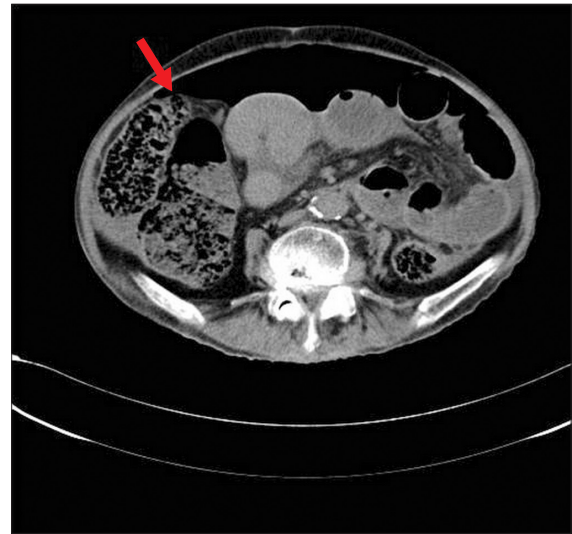


Fig. 5. Pneumoperitoneum and severe fecal impaction at the sigmoid colon noted on abdominal-pelvic computed tomography imaging (arrow).



Fig. 4. Free air under the diaphragm on an upright plain abdominal x-ray, suggesting the presence of bowel perforation (arrow).

After panperitonitis was diagnosed, we referred the patient to general surgeons, who immediately performed an exploratory laparotomy. A pinpoint perforation was found in the mid-sigmoid colon, and the proximal and distal sigmoid showed fecal impaction and diffuse adhesions. Permanent biopsy revealed extremely thin mucosa, focal perforation, and acute purulent peritonitis. On gross examination, a single large ulcer with perforation was observed. Hematoxylin and eosin histologic examination revealed absent mucosal lining, chronic inflammation, and ulceration. These findings confirmed that the perforation

developed from a stercoral ulcer. The general surgeons resected the sigmoid colon and created a colostomy. After surgery, the patient was transported to the surgical intensive care unit, where she was treated with intravenous antibiotics and intensive care management, including continuous renal replacement therapy. Although her condition temporarily improved, the patient subsequently developed septic shock and died of multiorgan failure (her do-not-resuscitate status limited aggressive management).

DISCUSSION

Common GI symptoms associated with DM include dysphagia, early satiety, reflux, constipation, abdominal pain, nausea, vomiting, and diarrhea. These symptoms may be severe and substantially decrease the quality of life. Their pathogenesis is complex and not well understood but appears to be multifactorial, involving such factors as motor dysfunction, autonomic neuropathy, glycemic control, and psychological factors.¹

Colonic transit time is often increased in patients with DM, leading to common complaints of constipation.³ Severe constipation with megacolon or colonic intestinal pseudo-obstruction may rarely occur as well. Complications of severe constipation, such as stercoral ulcer, perforation, and overflow diarrhea, are encountered infrequently.

Contractility of intestinal smooth muscle cells is altered in animal models of DM. Myocytes isolated from rat GI tracts

have increased expression of Na, K-ATPase, leading to increased intracellular calcium levels.⁴ Additionally, intracellular calcium binding proteins, such as calmodulin and protein kinase C, are reduced in intestinal smooth muscle cells. Similar changes are found in spontaneous diabetic rats, suggesting that DM alters smooth muscle contractility through changing intracellular signaling pathways of intestinal myocytes.⁵ Other animal studies suggest that impaired function of guano-sine triphosphate-binding proteins might alter the contractility of gastric smooth muscles,⁶ and reduced small intestinal motility may correlate with changes in myenteric innervation.⁷

Clinically, DM is often accompanied by abnormal GI sensory-motor function, as noted in our patient. Bowel dilatation leads to abnormal myenteric plexus innervation and density, which further impairs sensory-motor function. Intestinal muscle neuropathy and fecal impaction can lead to bowel perforation.

Chronic constipation is a risk factor for stercoral ulcer. Stercoral perforation has been defined as a large bowel perforation due to pressure necrosis from a fecal mass. Constipation, the presence of fecalomas, sigmoid colon location, and the absence of any other possible etiologies (e.g., diverticulosis) have been considered integral components of the definition.⁸ However, this definition primarily reflects causation and includes no histological criteria. Thus, stercoral perforation is not clearly discriminated from other types of colon perforation: idiopathic, spontaneous, or secondary (caused by another disease).

Severe chronic constipation is considered the main causal factor for stercoral perforation.⁹ Long-standing constipation can lead to stone-hard stool, producing persistent pressure on the bowel wall and leading to mucosal necrosis. Prolonged bedrest combined with poor bowel habits commonly contribute to constipation and the development of hard, inspissated feces. Obstruction, strictures, drugs, and decreased mucus production are contributing factors. Drugs, including opioids, tricyclic antidepressants, and certain antacids (aluminum hydroxide and bismuth), can worsen constipation. Antacid therapy may be used when the patient is misdiagnosed with a perforated peptic ulcer.⁸

Histopathologically, stercoral ulcers are basically nonspecific, but inflammatory changes can be found along their margins. In more than 90% of cases, stercoral perforation involves the antimesenteric margin of the sigmoid or rectosigmoid colon. The

edges are characteristically sharply demarcated, with the area of perforation confined to the ulcer's center. Hard fecal material juxtaposed to the ulceration helps confirm the diagnosis.⁸

Stercoral ulceration is more common in patients prone to constipation, especially elderly patients, opioid addicts, mentally ill individuals, and postoperative patients after various reconstructive operations. After hand reconstruction involving a flap from the lower extremity, postoperative immobilization is required, thereby increasing the risk of bowel perforation. However, most patients require only a short period of immobilization; sometimes, no immobilization is recommended.

Measures to identify and minimize or prevent potential risk factors for stercoral perforation are necessary. First, medical personnel must be aware of this potentially fatal complication and obtain a thorough history (and possibly a screening abdominal x-ray) to detect chronic constipation. Second, unnecessary long-term nonsteroidal anti-inflammatory drug use should be avoided, especially in older patients with DM and those who undergo prolonged immobilization. Their analgesic effects can mask abdominal pain, exacerbating these patients' impaired ability to notice abdominal pain and reduced stool quantity: this can delay the diagnosis of chronic constipation and stercoral ulceration or perforation. Third, direct therapy for the underlying cause of chronic constipation must be applied. Fourth, increased dietary fiber and use of polycarbophil calcium, rather than magnesium oxide, are recommended; polycarbophil calcium is an osmotic laxative that acts as a stool stabilizer, controlling GI fluid volume.¹⁰ Fifth, early postoperative ambulation should be encouraged to improve bowel motility. When patients are poorly compliant with this recommendation, their caregivers should monitor bowel movements and ambulation and provide support as needed.

In conclusion, chronic constipation can lead to bowel perforation because of fecal impaction in older diabetic female patients. Hand reconstruction involving a flap harvested from the lower extremity may increase the risk of constipation and perforation. To prevent bowel perforation, increased dietary fiber and early ambulation postoperatively should be considered.

REFERENCES

1. Zhao J, Frøkjær JB, Drewes AM, Ejlskjær N. Upper gastrointestinal sensory-motor dysfunction in diabetes mellitus. *World J*

- Gastroenterol 2006;12:2846-57.
2. Gonvers JJ. Gastroenterological problems in the elderly. *Schweiz Rundsch Med Prax* 1990;79:1008-10.
 3. Yarandi SS, Srinivasan S. Diabetic gastrointestinal motility disorders and the role of enteric nervous system: current status and future directions. *Neurogastroenterol Motil* 2014;26:611-24.
 4. Touw K, Chakraborty S, Zhang W, Obukhov AG, Tune JD, Gunst SJ, et al. Altered calcium signaling in colonic smooth muscle of type 1 diabetic mice. *Am J Physiol Gastrointest Liver Physiol* 2012;302:G66-76.
 5. Takahashi T, Kojima Y, Tsunoda Y, Beyer LA, Kamijo M, Sima AA, et al. Impaired intracellular signal transduction in gastric smooth muscle of diabetic BB/W rats. *Am J Physiol* 1996;270:G411-7.
 6. Lin S, Kajimura M, Takeuchi K, Kodaira M, Hanai H, Nishimura M, et al. Alterations of GTP-binding proteins (G α and Gq/11 α) in gastric smooth muscle cells from streptozotocin-induced and WBN/Kob diabetic rats. *Dig Dis Sci* 2000;45:1517-24.
 7. Demedts I, Masaoka T, Kindt S, De Hertogh G, Geboes K, Farré R, et al. Gastrointestinal motility changes and myenteric plexus alterations in spontaneously diabetic biobreeding rats. *J Neurogastroenterol Motil* 2013;19:161-70.
 8. Maull KI, Kinning WK, Kay S. Stercoral ulceration. *Am Surg* 1982;48:20-4.
 9. Bauer JJ, Weiss M, Dreiling DA. Stercoraceous perforation of the colon. *Surg Clin North Am* 1972;52:1047-53.
 10. Johanson JF. Review of the treatment options for chronic constipation. *MedGenMed* 2007;9:25.