

CASE REPORT
SPONTANEOUS PERFORATION OF THE SIGMOID IN
CHRONIC RENAL FAILURE

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Abstract

Spontaneous perforation of the sigmoid was found in association with chronic renal failure. A review of the literature disclosed that the spontaneous perforation is a rare occurrence. Its association with renal failure seems to be furthermore rare, but recently it is gaining a clinical attention. The constipation is a common complication in patients with chronic renal failure on hemodialysis. Those previously reported cases and ours suggest that the longstanding constipation is somehow responsible for the perforation of the colon.

INTRODUCTION

The perforation of the colon has been occasionally reported in association with various colonic conditions such as carcinoma, obstruction, inflammatory diseases, diverticulosis, and trauma¹⁾. Spontaneous occurrence of the perforation without obvious predisposing disorders seems rare²⁾. Recently, we have encountered a patient on hemodialysis for chronic renal failure who terminally suffered from the perforation of the sigmoid. Such an association is furthermore rare to our knowledge. Four reports of such cases have appeared in the English literature^{3, 4, 5, 6)}, and one in Japanese⁷⁾. We, therefore, consider it warranted to describe this case in order to alert clinicians to its occurrence. A possible mechanism of perforation in such cases is also discussed here.

CASE REPORT

A 69-year-old woman with a known history of hypertension and chronic renal failure was transferred to the hospital of Kawasaki Medical School on July 5, 1978.

Her present illness dated back to four years prior to admission, when she

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had suffered from pyelonephritis and manifested chronic renal failure. She had been on Furosemide, Methyldopa, and Aluminum-base antacid with a poor response. Physical examination on admission was unremarkable. Pertinent laboratory data included BUN 68 mg/dl, Creatinine 11.8 mg/dl, GFR 16.9 ml/min, RPF 148 ml/min, and PSP 5% (15') and 22% (120'). She was put on hemodialysis on regular bases. Although she was severely constipated and took laxatives continuously, her hospital course had been relatively uneventful. Six months after admission she suddenly complained of left lower quadrant abdominal pain after defecation. Stool was reported to be blood-stained. Blood pressure was noted down to 86/60mmHg. Pulse was 84/min and regular. Temperature was 37.2°C. Physical examination showed a soft and flat abdomen with mild tenderness in the left lower quadrant, where masses were palpated. Tumorous masses were noted by digital examination to be in the Douglas' pouch. The liver, spleen, and kidneys were unremarkable as were heart and chest. The impression of the intra-abdominal dissemination of ovarian tumor was given by gynecologist. Her general condition gradually deteriorated and despite of intensive care, she died in shock condition 6 days after the appearance of abdominal pain.

Post-mortem examination revealed severe panperitonitis with hard fecal masses exposed in the Douglas' pouch. The sigmoid colon was perforated 5.0

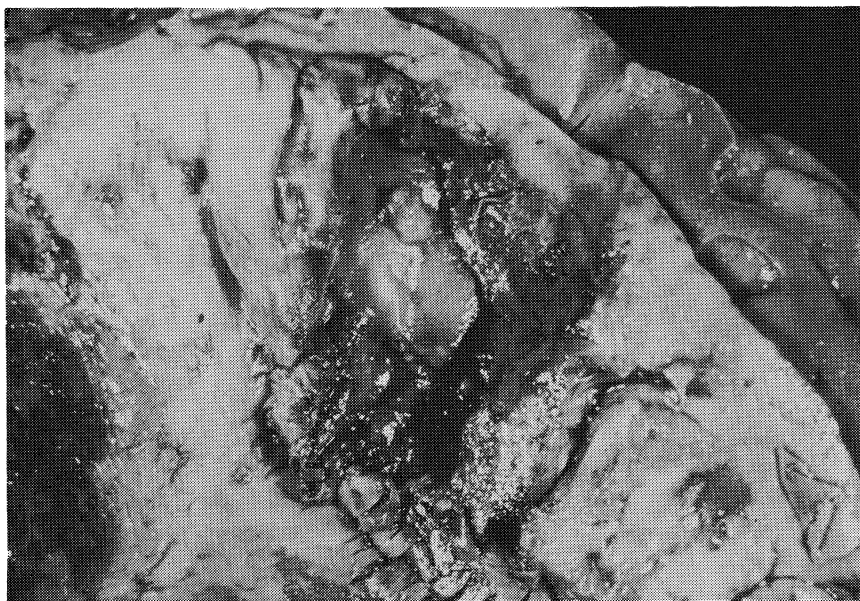


Fig. 1. Serosal surface of the sigmoid at perforation. Its margin is dark red and minimally inflamed.

×4.0 cm in greatest dimension at 40 cm from the anal verge. Grossly, its edge was free of inflammation or bleeding (Fig. 1). Rest of the sigmoid colon was unremarkable except for a focal small erosion near the perforation. There was no evidence of carcinoma, diverticular disease, obstruction, inflammatory bowel disease, or foreign body throughout the intestinal tract. The careful examination of feeding arteries and veins failed to reveal any abnormalities.

Microscopic examination of the sigmoid mucosa adjacent to the perforation revealed the presence of necrotic tissue and exudates with a large number of neutrophils (Fig. 2). The eroded mucosa near the perforation was essentially

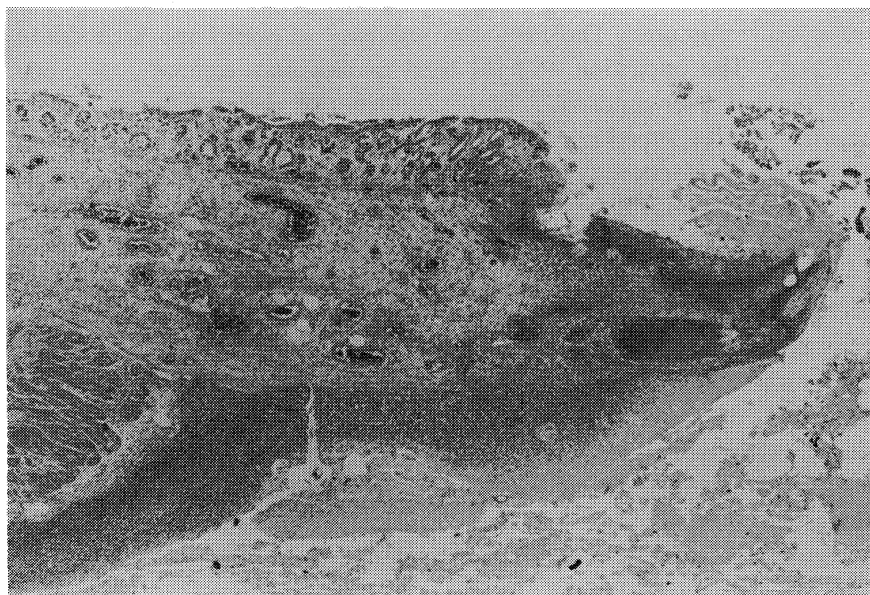


Fig. 2. Photomicrograph of perforated colon. Note that the inflammatory reaction is less severe at perforated site compared to the serosa. Adjacent mucosa appears free of inflammation. (H & E, ×100)

non-specific with minimal inflammation and necrosis. Furthermore, the serosa of the intestinal tract was entirely covered by pyogenic material. Multiple sections from the rest of the sigmoid and other portions of the colon and small intestine were unremarkable, although generalized atrophy and focal disruption of muscular layer by the insertion of vascular and nervous system were noted. The kidneys were contracted with chronic pyelonephritis.

DISCUSSION

Perforation of the colon was reported sporadically and mostly associated

with various conditions, such as carcinoma, diverticular disease, obstruction, inflammatory bowel disease, foreign body, and trauma¹⁾. A few case reports, on the other hand, have appeared without such an association, thereby termed "spontaneous" or "idiopathic" perforation of the colon. Our case lacked all the possible etiological factors as listed above, and fulfilled criteria of spontaneous perforation of the colon made by Castleton¹⁾.

On the etiology and/or pathogenesis of the so-called spontaneous perforation of the colon, several speculations have been entertained^{8,9)}, but it still remains to be elucidated. For almost all reported cases were complicated by the prolonged constipation, such intractable constipation was incriminated as a cause of the colonic rupture. The following sequence of events appears a reasonable speculation. The impaction of hard fecal masses may compress to erode the colonic mucosa (stercoraceous ulcer). Sudden rise of intracolonic pressure at defecation may be superimposed to such condition to finally ensue perforation (stercoraceous perforation)^{10,11,12,13,14)}. Senility with the atrophy of colonic wall may be another contributor. All these factors may have been operative in our case, since clinically defecation preceded abdominal pain.

Recent reports suggest a close association between spontaneous perforation and chronic renal failure. So far, five examples have been reported^{3,4,5,6,7)}. Scarcity of such reports in the past may depend upon a recent advent of hemodialysis with prolongation of the patient's life. Further improvement in the management of chronic renal failure, therefore, may result in a frequent encounter with spontaneous perforation of the colon in the future. Patients with chronic renal failure on hemodialysis are commonly troubled by a long-standing constipation. The ingestion of aluminum-base antacid¹⁵⁾, dehydration, electrolyte imbalance and patient's inactivity are considered to cause constipation⁵⁾. This may evoke the previously mentioned sequence of events to result in the stercoraceous perforation. The present case seems to seriously warn us, and clinicians must be alert to such an occurrence.

Addendum

After the submission of the manuscript, we experienced a similar case. A 63-year-old man with a known history of nephrotic syndrome and chronic renal failure on regular hemodialysis was transferred to our hospital because of possible peritonitis. History disclosed longstanding intractable constipation. At operation, 4×3 cm perforation was found at sigmoid colon. Fecal masses were exposed in the peritoneal cavity. The patient died one month later. No predisposing factors have been revealed surgically as well as at autopsy.

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