Ischemic Colitis Associated with Systemic Lupus Erythematosus; Report of a Case

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ABSTRACT. A case of ischemic colitis associated with systemic lupus erythematosus (SLE) is herein described. A female patient, who had been diagnosed as SLE, complained of left upper quadrant pain and anal bleeding. Barium enema examination demonstrated involvement of the splenic flexure with thumbprinting, and colonoscopy revealed reddish, edematous mucosa with multiple erosions and longitudinal ulcers in the left side of the colon. Although follow-up colonoscopy performed four weeks later confirmed improvement of the lesion, subsequent colonoscopic observations showed recurrence of a shallow ulceration in the previously affected segment of the colon. The ischemic colitis in this case seems to have been prolonged because of the patient's underlying disease, SLE.

Key words: systemic lupus erythematosus (SLE) — ischemic colitis

The gastrointestinal manifestations of systemic lupus erythematosus (SLE) were first reported by Osler in 1895.[1] Since this original report, it has become well known that SLE possibly involves any site within the gastrointestinal tract. According to large surveys of patients with the disease, gastrointestinal symptoms occur in approximately 50% of the patients, with intestinal bleeding from ulcers being the most frequent symptom (36.4%).[2,3]

Because we recently experienced a case of ischemic colitis during the course of SLE, the possible correlation between the two diseases is discussed.

CASE REPORT

A 59-year-old Japanese female with an established diagnosis of SLE was referred to our institution in July, 1992, because of left upper quadrant pain and hematochezia. She had no prior history of gastrointestinal disease. Because the patient had developed a left crural ulcer four months previously, she had been hospitalized at a neighboring clinic, at which prednisolone was prescribed (10 mg/day). On the day of admission, left upper quadrant pain suddenly occurred, accompanied by anal bleeding of fresh blood.

Upon physical examination, she had a body temperature of 37.0°C. Her left upper quadrant was slightly tender without any palpable mass. Laboratory data on admission revealed mild anemia (hemoglobin; 10.5 g/dl),

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hypoproteinemia (5.7 g/dl), and positive serum C-reactive protein (24 mg/dl). Neither leukocytopenia nor thrombocytopenia was evident. The antinuclear antibody was positive (titer; 1 : 1024), but she was negative for both lupus anticoagulant and hypocomplementemia.

Barium enema examination on the day of admission demonstrated thumbprinting in the splenic flexure (Fig 1). Under colonoscopy performed on

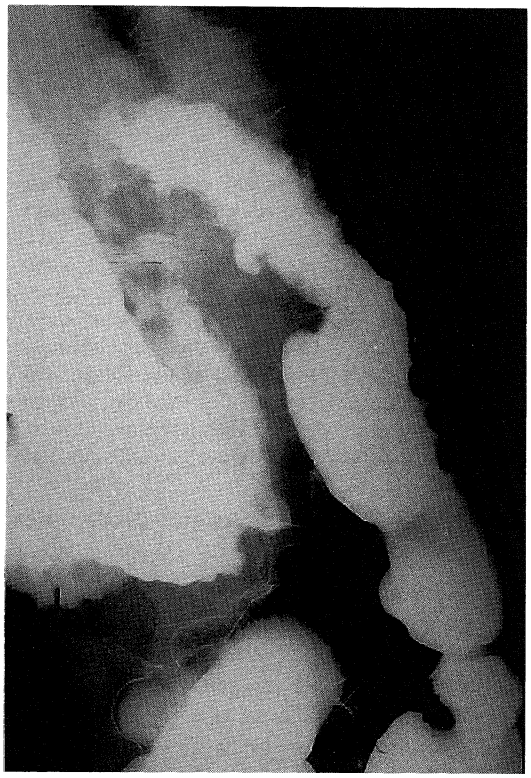

Fig 1. Barium enema examination on admission disclosed thumbprinting in the splenic flexure.

the next day, reddish, edematous mucosa and longitudinal ulcers were detected in the splenic flexure and descending colon (Fig 2). Histological examination of the biopsy specimens obtained from these areas revealed submucosal edema and fibrosis with minimal acute inflammatory infiltrates. A barium meal study showed no abnormalities in either the stomach or the small intestine.

Based upon the above findings, her condition was diagnosed as ischemic colitis. She was managed by total parenteral nutrition. In addition, because there remained a possibility that the patient had ischemic colitis secondary to lupus vasculitis, prednisolone (40 mg/day) was administered. After 10 days of treatment, her symptoms markedly improved. Follow-up colonoscopy and barium enema examination performed four weeks after the treatment revealed improvement of the ulcers (Fig 3).

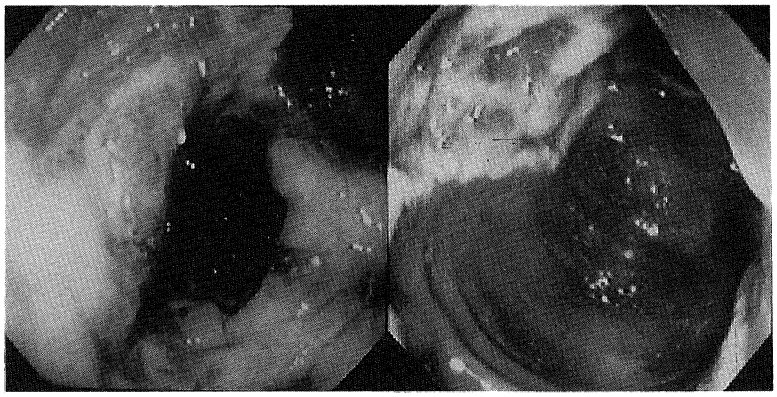

Fig 2. Colonoscopy revealed reddish, edematous mucosa with longitudinal ulcers in the descending colon.

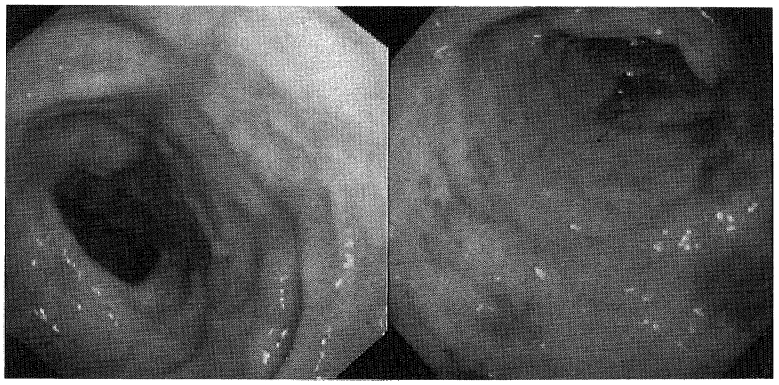

Fig 3. Follow-up colonoscopy performed four weeks after admission showed improvement of the ulcers.

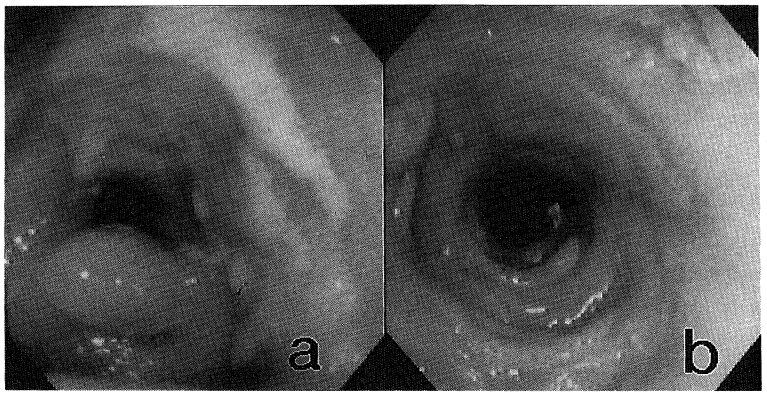

Fig 4. Follow-up colonoscopy showed a shallow ulceration at the same segment of the descending colon four months (a) and seven months (b) after the onset.

The patient was discharged with predonisolone (10 mg/day). Although she subsequently remained free of intestinal symptoms, follow-up colonoscopy performed four and seven months after the onset of ischemic colitis demonstrated a shallow ulcer in the descending colon (Fig 4).

Colonoscopy performed three years after the onset showed the ulcer to be completely scarred with luminal narrowing (Fig 5).

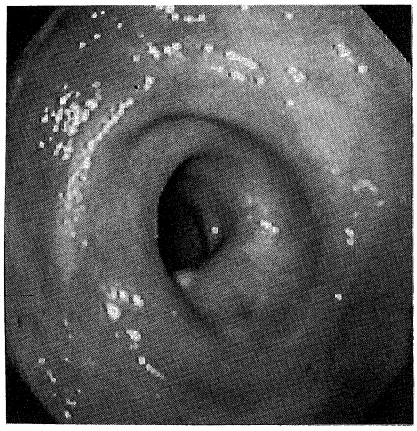

Fig 5. Follow-up colonoscopy performed three years after the onset showed complete improvement of the ulcer.

DISCUSSION

It is well known that patients with SLE frequently develop gastrointestinal manifestations, such as peritonitis, intestinal obstruction, ulceration, perforation, ischemia, hemorrhage, and protein losing enteropathy.[2,4-6] The most severe complications, including mesenteric infarction and bowel perforation, require emergency laparotomy.[7-9]

Kurlander and Kirsner[10] divided these intestinal manifestations into two categories; one being predominantly caused by vasculitis with resultant ulceration, perforation, or bleeding, and the other being chronic inflammatory bowel disease. Iida et al.[6] reported that the former lesions could be further divided into three groups; ischemic enteritis form, colonic multiple ulcer form, and protein-losing form.

The abovementioned colonic multiple ulcers are round-or oval-shaped discrete ulcers, so called "punched-out" ulcers,[11] and occur in multiplicity throughout the colon. In contrast, our patient had longitudinal ulcers with narrowing. These findings were compatible with lesions of ischemic enteritis form, rather than multiple colonic ulcers. In addition, our patient's colitis could be classified as the stricturing form of ischemic colitis, according to Marston's criteria of ischemic colitis.[12]

Colonic disease in SLE is generally regarded as a result of mesenteric vasculitis.[13] In previous articles, lupus vasculitis with an intestinal lesion was confirmed by histopathological studies.[7,14] In a case reported by Weiser *et al*,[15] severe intestinal venulitis and a thickened base membrane with deposits

of complement C3 were detected in the resected colon. Gladman *et al*[16] also demonstrated deposits of IgG in vessel walls of the colon. In our patient, histopathological examinations of biopsy specimens failed to identify any vasculitis. However, when our patient developed ischemic colitis, she had an active left crural ulcer which originated from SLE, and her condition continued to deteriorate. Furthermore, when compared with the natural course of usual ischemic colitis without collagen-like disease, our case had prolonged and relapsing colitis even after treatment. Therefore, a possibility remained that the ischemic colitis in our patient had close correlation with her underlying disease, SLE.

There have been reports suggesting that steroid therapy may predispose the colon to perforation or vasculitis in patients with both SLE and gastrointestinal involvement.[17,18] However, an increase in the dose of steroid at onset seemed to result in a rapid improvement in our patient. This fact seems to further support the possibility of a close correlation between ischemic colitis and SLE.

Clinicians who deal with SLE patients should keep in mind that during the follow-up of this disorder, it may possibly be complicated by severe ischemic colitis, especially during the active phase of SLE.

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