Lupus Enteritis Detected by Capsule Endoscopy

Hiroki Endo1, Yasuyuki Kondo1, Kei Kawagoe1, Tomohiko R Ohya1, Tatsuuro Yanagawa1, Masako Asayama1, Kantaro Hisatomi1, Takuma Teratani1, Masato Yoneda2, Masahiko Inamori2, Atsushi Nakajima2 and Nobuyuki Matsuhashi1

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Almost half of all systemic lupus erythematosus (SLE) patients present with gastrointestinal tract symptoms (1, 2), but only a few patients develop intestinal lesions, a condition referred to as lupus enteritis. Moreover, most cases of small-intestinal lupus enteritis develop as an acute condition (3). Here, we present a patient with SLE in whom small intestinal ulcers were detected before the establishment of the diagnosis.

Capsule endoscopy (CE) is a new tool for evaluating small bowel pathology that has a higher diagnostic yield than barium follow-through or push enteroscopy, and it is also effective for detecting small-intestinal lesions (4), as in the present case.

A 72-year-old woman presented with melena and was admitted to our hospital. Examinations that included esophagogastro-duodenoscopy, colonoscopy and computed tomography (CT) failed to reveal the cause of the bleeding. No cutaneous, mucosal, or neurological abnormalities were present. Hematological, biochemical, and serological tests were within normal limits. Stool cultures were negative for several pathogens. Cytomegalovirus antigenemia was negative. We performed CE to search for the cause of the abdominal pain and intestinal bleeding and for possible vasculitis, and CE revealed multiple small ulcers and scars in the small intestine (Fig. 1).

Four months later the patient was readmitted to our hospital, this time because of abdominal pain. On admission, her lymphocyte count was decreased (683/μl), and her anti-nuclear antibody (×320), and anti-double stranded DNA antibody titers (44.7 IU/ml) were elevated. The results of tests for anti-cardiolipin antibody, PR3-ANCA, and MPO-ANCA were all negative. Chest CT revealed a small amount of pericardial effusion, but no pleural effusion. The echocardiography findings were normal. Abdominal angiography failed to show any aneurysms, arterial occlusion, or stenosis. Thus, the patient manifested serositis, lymphopenia, and elevated titers of anti-double stranded DNA antibody and anti-nuclear antibody; consequently, she was diagnosed as having SLE.

The patient’s symptoms improved promptly after administration of 30 mg of prednisolone per day. The anti-double stranded DNA antibody titer also decreased (17.6 IU/ml). Her condition is currently being controlled on a maintenance dose of prednisolone, although she developed a malar rash and Raynaud’s phenomenon during the prednisolone dose reduction.

Discussion

Gastrointestinal tract symptoms are common in SLE patients, but most of them are thought to be related to nonsteroidal anti-inflammatory drugs (NSAIDs). Some SLE patients present with gastrointestinal symptoms caused by SLE itself, but few develop intestinal lesions caused by vasculitis, a condition referred to as lupus enteritis (5). Lupus enteritis is classified as an acute ischemic enteritis that occurs mainly in the small intestine and chronic multiple ulcers occurring mainly in the colon.

The initial manifestation in the present patient was melena. Since repeated upper and lower gastrointestinal tract endoscopy and CT examinations failed to detect any pathological findings, small intestinal lesions were suspected to be the cause of melena, and small ulcers were detected by CE. Mucosal edema induced by vasculitis of the submucosal or serous tissues has been reported to be the sole endoscopic finding in lupus enteritis of the small intestine, and mucosal lesions, such as erosions or ulcers, are absent. However, in our patient small intestinal mucosal lesions preceded the di-

1Department of Gastroenterology, Kanto Medical Center NTT EC, Tokyo and 2Gastroenterology Division, Yokohama City University, Yokohama

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Correspondence to Dr. Hiroki Endo, t066011b@yokohama-cu.ac.jp

Figure 1. Capsule endoscopy (CE) findings in the small intestine. The arrowheads indicate the small ulcers and scars in the small intestine.
agnosis of SLE. Moreover, she did not present with sudden-onset abdominal pain, which has been reported to be common among patients with ischemic-type lupus enteritis. Thus, our case suggests that a certain form of vasculitis can give rise to mucosal lesions that can cause bleeding.

We were able to observe the erosions and ulcers in the small intestine in the present case by CE. CE is an extremely non-invasive method of gastrointestinal tract examination that enables the entire small intestine to be easily examined. Despite these advantages, CE has some limitations. For example, therapeutic interventions, including biopsy, polypectomy, and foreign body removal, cannot be performed by CE. While vasculitis-induced ulcers and perforations have been reported in SLE patients, small intestinal CE findings have never been described. Recent major advances in small intestinal endoscopy, specifically CE and double-balloon endoscopy, are shedding light on many aspects of small intestine pathology. The establishment of interpretation schemes for the small intestinal endoscopy findings of intestinal lesions associated with systemic disorders, including SLE, is strongly desired.

References


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