Patients with systemic lupus erythematosus (SLE) present with various gastrointestinal symptoms. The symptoms are caused by not only the disease itself, but also complicated inflammatory bowel diseases, adverse effects of therapeutic agents, and infections due to therapy related immunosuppression. Therefore, the incidence of gastrointestinal problems varies with the methods of studies (1). Around 40% of SLE patients have gastrointestinal problems, of which gastroduodenal mucosal lesions as adverse effects of nonsteroidal anti-inflammatory agents, corticosteroids or cytotoxic agents are the most common. On the other hand, disease itself causes abdominal pain in only 8% of the SLE patients (2). However, autopsy studies suggest that 60 to 70% of lupus patients have had an episode of peritonitis, though the incidence of clinically recognized ascites is about 10% (1). Most of the gastrointestinal lesions are not so serious, but mesenteric vasculitis and thrombosis, to which lupus anticoagulant is related, cause severe colon ischemia, leading to mesenteric vasculitis and thrombosis, to which lupus anticoagulant is related, cause severe colon ischemia, leading to intestinal infarction and perforation. Pancreatitis can also be serious. These conditions are life-threatening and require emergent surgical interventions. Of note, physical findings of bowel perforation and ischemia are often masked by the medication with corticosteroids and immunosuppressants.

Lupus enteritis, which may not be an established nomenclature, but frequently appears in the literature, is generally characterized by marked wall swelling of the diffuse small intestine (3). The patients present with abdominal pain, nausea, vomiting, and diarrhea. Abdominal X-ray reveals pseudo-obstruction of the gastric outlet, duodenal stasis, and disappearance of entire bowel gas which represents “gasless ileus”. The swelling of intestinal wall is shown as “accordion-like appearance” and “target-like appearance” by ultrasonography and CT scan, respectively. Endoscopic study does not necessarily demonstrate mucosal lesions. The conditions are generally reversible in response to treatment with corticosteroids, but the recurrence is usual. Peritonitis with ascites and lupus cystitis are often complicated with lupus enteritis. Protein losing enteropathy and malabsorption syndrome are also found in SLE patients, and can be the initial manifestation of lupus (1). Pneumatosis cystoides intestinalis is a rare complication of SLE, but may coexist with necrotizing vasculitis (1).

On the other hand, colon lesions are rarely found in SLE patients, as reported by Miyahara et al in the October issue (4). Multiple ulcers appear mainly in the rectum and sigmoid colon (5). The colon disease is more common in patients during the active stage than those in remission and can be the initial manifestation of SLE. Main symptoms include abdominal pain, diarrhea, and bloody stool. The symptomatic feature is indistinguishable from that in inflammatory bowel diseases, especially ulcerative colitis. Since ulcerative colitis is not an extremely rare complication in SLE patients (1), it is hard to determine whether the colon disease arises from SLE itself or concomitant ulcerative colitis. Furthermore, it is common that colitis precedes the onset of lupus symptoms (1). To this end, endoscopic examinations with histopathological evaluation are essential for the differential diagnosis. Sulfasalazine for the treatment of inflammatory bowel diseases is known as one of the causative agents of drug-induced lupus. Indeed, patients with inflammatory bowel diseases and inflammatory arthropathy who are treated with sulfasalazine often develop lupus signs and symptoms with emergence of disease-specific autoantibodies (6). In addition, cytomegalovirus enteritis is also another important differential diagnosis in patients receiving an intensive immunosuppressive therapy (7).

Because most patients develop lupus-related colon disease in active stages, high-dose corticosteroid is used as the first-line therapy. Immunosuppressive agents such as cyclophosphamide are optional choices. Sulfasalazine and mesalazine are also useful supportive agents, particularly for maintenance therapy. When bowel perforation and infarction are suspected, or a rapid response to steroid is not achieved, surgical intervention should be commenced immediately. Because delay of the decision can be lethal, it is important to start an immediate treatment following early diagnosis.
References


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