Coexistence of Crohn's disease and systemic lupus erythematosus: a case report and literature review
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Lupus enteritis and Crohn's disease are two common immune diseases involving the gastrointestinal tract. There are many similar clinical manifestations, therefore it is very difficult to distinguish between them. The differentiation between Crohn's disease and lupus enteritis is difficult because both the two diseases can show similar clinical signs and symptoms such as abdominal pain, bloating, intestinal obstruction and so on. In addition, it is known that coexistence of SLE and Crohn's disease is very rare. Crohn's disease may occur before or after the diagnosis of SLE, but Crohn's disease prior to lupus is commonly seen in drug-induced cases during Crohn's disease treatment. Crohn's disease's presence after SLE is extremely rare. Below we describe a rare case of concomitant Crohn's disease and SLE.

Case presentation
A 50-year-old Chinese woman was admitted to our hospital because of fever and relapsing episodes of abdominal pain over the last 9 months. The patient was diagnosed with SLE (9 months ago) based on fever, relapsing episodes of abdominal pain, bloating, leucopenia [white blood cells (WBC) 2.24 x 10\(^9\) /L] positive antinuclear antibodies (ANA), anti-double-stranded (dsDNA) antibody, anti-Smith antibody, ribonucleoprotein (RNP)/Smith antibody and polyclonal antibodies (pleural effusion, ascites and pericardial effusion). The symptoms were relieved after she was treated with prednisolone and mycophenolate mofetil. However, symptoms of fever (38.5\(^\circ\)C) and abdominal pain relapsed again, accompanied by hair loss, dry mouth, reduced defecation in the period of gradual prednisolone decrease.

When she was presented to our hospital, she had no other symptoms such as dry eyes, rampant teeth, photosensitivity, oral ulcer, joint pain or Raynaud's phenomenon, with only fever, abdominal pain, as well as reduced defecation. The physical examination was unremarkable, except where there was pain at the lower right abdominal quadrant. Her medical history included osteoporosis and tuberculosis infection. The tuberculosis infection was found 4 months ago, and had been treated by rifampicin and isoniazid for 3 months; however, the abdominal pain did not relieve. Her family history was unremarkable.

Introduction
Systemic lupus erythematosus (SLE) is a chronic and serious autoimmune disease, characterized by erythema discoid, myalgia and arthralgias. It is a multisystem disease and can induce renal, digestive, neurologic and hematologic disorders. SLE tends to occur primarily in women aged 15–44. Gastrointestinal involvement in SLE appears almost regular. The differentiation between Crohn's disease and lupus enteritis is difficult because both the two diseases can show similar clinical signs and symptoms such as abdominal pain, bloating, intestinal obstruction and so on. In addition, it is known that coexistence of SLE and Crohn's disease is very rare; Crohn's disease may occur before or after the diagnosis of SLE, but Crohn's disease prior to lupus is commonly seen in drug-induced cases during Crohn's disease treatment. Crohn's disease's presence after SLE is extremely rare. Below we describe a rare case of concomitant Crohn's disease and SLE.

Keywords: Crohn's disease, lupus enteritis, literature review, systemic lupus erythematosus

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Radiologic evaluation with computed tomography (CT) showed pneumatosis lumen, accompanied by air-fluid level and mesangial exudation. Laboratory tests were within normal limits except for leucopenia (WBC 2.56 × 10⁹/L) and anemia (hemoglobin of 102 g/L). Erythrocyte sedimentation rate, C-reactive protein and procalcitonin were all normal, and blood culture for fungi and bacteria was negative. Immunological tests showed positive rheumatoid factor 153 IU/mL, decreased complement components C3 0.71 g/L (normal 0.85–1.93), positive ANA 1/320, as well as positive anti-Smith antibody, anti-dsDNA and anti-RNP antibodies. Tuberculosis (TB)-spot test was positive. The PET-CT found no abnormality. After the infection, intestinal tuberculosis and cancer were excluded, and SLE, mesenteric vasculitis and intestinal obstruction were considered. After treatment with methylprednisolone (120 mg/day *11d), cyclophosphamide (0.2 g/q2qod), gastrointestinal decompression and enteral and parenteral nutrition support, the patient’s fever subsided, and WBC returned to normal. However, the incomplete intestinal obstruction, with reduced defecation, still existed. Abdominal X-ray examination still showed the air-fluid level (Fig. 1). Enteroscopy revealed a small intestinal ulcer with intestinal cavity stenosis, and the biopsy showed that intestinal mucosa had moderate chronic inflammation, with mild acute inflammation and erosion, except for granulation tissue formation (Fig. 2). Small intestine simulation CT revealed that part of the intestinal cavity was slightly expanded, with wall enhancement and multiple air-fluid planes (Fig. 3). Further laparoscopy, partial appendectomy and small intestine resection were performed after a comprehensive disease evaluation. Pathological examination showed longitudinal ulceration, stenosis and dilatation were observed in the intestinal canal and a lymph node was detected in the pericentric adipose tissue by gross observation. Microscopically, it could be seen that transmural inflammation, local erosion and ulcer formation. Inflammatory granulation tissue formation occurred at the ulcer site. Adipose tissue hyperplasia of the whole intestinal wall and outside the serous membrane, multiple lymphoid hyperplasia and reduce mucosal crypt could be seen. Besides, the submucosa of the surrounding intestinal wall was widened and swollen, and the blood vessels were dilated and congested. One lymph node was seen around the intestine (Fig. 4). It was diagnosed as multiple autoimmune diseases, SLE concomitant to Crohn’s disease. The treatment regimen was adjusted to add cyclophosphamide (800 mg/month), thalidomide (75 mg/day) and metronidazole 0.4 g two times a day. Symptoms improved significantly and after 5 months, simulated small intestine-enhanced CT showed no stenosis in the lumen. The enteroscopy showed the ileum anastomosis was smooth and there was no ulcer stenosis.

**Discussion**

Although the patient’s history is short, the diagnosis is extremely complicated, covering the differential diagnosis of three acute abdomens, which is one of the most valuable points of this case report. Second, the treatment of this case is extremely clever in the selection of immunosuppressants, which quickly stabilized the condition without the selection of biological agents.

Patients with SLE presenting with gastrointestinal symptoms account for about 50% of patients with SLE.
[2], mainly including lupus enteritis (including lupus mesenteric vasculitis, protein-losing enteropathy and IPO), acute pancreatitis and peritonitis [3]. The prevalence of lupus enteritis in SLE is up to 53% [1]; however, only 2% of patients with SLE usually have clinical symptoms of bowel vasculitis [4]. Patients with lupus enteritis are also prone to occur ischemic enteritis, bleeding or perforated peritonitis. It can present as a variety of gastrointestinal symptoms such as oral ulcer, dysphagia, anorexia, nausea, vomiting, bleeding, abdominal distension, abdominal pain, etc. Early radiographic manifestations of lupus enteritis were thumb-printing, targeted sign, comb sign, pseudo-obstruction and segmental bowel dilatation, and ‘collar button types’ when involving the colon by barium enema [5,6]. Ultimately, lupus enteritis is divided into two types: type 1 with perforated blood vessels and type 2 with nonspecific ulcers and granulomatous colitis [7,8]. The type 2 lupus enteritis is the one easily confused with Crohn’s disease.

Crohn’s disease is a chronic granulomatous inflammatory bowel disease (IBD) of unknown etiology. It is a type of IBD with diverse clinical manifestations and no clear diagnostic criteria. Crohn’s disease can also present with abdominal segmenting colic, abdominal distension, and even ulceration of the oral mucosa, skin nodular erythema, arthritis, eye disease as well as other extraintestinal manifestations [9,10]. Crohn’s disease is difficult to distinguish from lupus enteritis when it presents as extraintestinal manifestations involving the skin, eyes and joints. Therefore, pathological examination is the key to identifying and further distinguishing between the two diseases, by revealing the presence of inflammatory epithelial-giant cell granuloma. Lupus enteritis is more prone to perforation, which may occur even if the disease is well controlled [4]. Therefore, clinical treatment needs to be more active, and large doses of prednisolone or cyclophosphamide shock therapy is preferred. The patient had repeated intestinal obstruction under the definitive diagnosis of lupus, and lupus enteritis was initially considered. Though, after treatment involving large doses of prednisolone, the obstruction symptoms were not alleviated. According to the 2016 European Crohn’s and Colitis Organization (ECCO) consensus and the 2018 consensus of IBD in China, the intraoperative pathological results were consistent with the pathological diagnostic criteria of Crohn’s disease (Table 1). This did not support autoimmune enteropathy, and thus lupus enteritis was excluded.

Intestinal tuberculosis is a chronic and specific infection caused by the invasion of Mycobacterium tuberculosis into the intestinal tract, which can involve the upper and lower digestive tract, causing abdominal pain and intestinal obstruction. In addition, it can be accompanied by systemic symptoms such as low fever, night sweats and weight loss. Crohn’s disease is an autoimmune disease; however, the clinical manifestations of abdominal pain, diarrhea, weight loss, fever and nutritional disorders, are clearly similar to the clinical symptoms of intestinal tuberculosis. Hence, it is easy to confuse the diagnosis of Crohn’s disease with intestinal tuberculosis. The tuberculin test was strongly positive, and the gastroscopy showed...
Table 1 Differential diagnosis

| Clinical feature | Lupus enteritis | Crohn’s disease | Intestinal tuberculosis |
|------------------|----------------|-----------------|------------------------|
| Pathology        | Chronic, nonspecific mucosal inflammation or vascular ischemic changes | Inflammatory epithelial-giant cell granuloma | Caseous necrosis |
| Microbiome       | N/A | N/A | Acid-fast bacilli smear/culture positive |
| Image examination | Fingerprint sign, false obstruction, dilatation of intestinal segment | Bowel involvement/comb sign/jumping lesion | Cecum involvement, transverse ulcer/dilated ileum valve |
| Endoscope        | Colon involvement, segmental, local irregular ulcer, spacious, clean and no moss ulcers, which discontinuous involved all gastrointestinal tract | Colon, sigmoid colon involvement, longitudinal ulcers, mouth sores, cobblestone lesions, lumen stenosis and jumping lesions/aphtous oral ulcer | |

1. Transmural inflammation
2. Distribution of aggregated inflammation, permeable lymphocyte hyperplasia
3. Submucosal thickening (caused by fibrosis – fibromuscular tissue destruction and inflammatory edema)
4. Fissures (fissuring ulcers)
5. Noncaseous granuloma (include lymph node)
6. Abnormalities in the gut nervous system (submucosal nerve fiber hyperplasia and ganglionitis, intermuscular nerve fibers proliferate)
7. Relatively normal epithelial-mucus secretion is preserved (Goblet cells are usually normal)

It has been suggested that a diagnosis of Crohn’s disease should be made when three features are present in the absence of granulomas, or when an epithelioid granuloma is present with one other feature provided that specific infections are excluded.

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Table 2 Pathological diagnosis of Crohn’s disease

- Transmural inflammation
- Distribution of aggregated inflammation, permeable lymphocyte hyperplasia
- Submucosal thickening (caused by fibrosis – fibromuscular tissue destruction and inflammatory edema)
- Fissures (fissuring ulcers)
- Noncaseous granuloma (include lymph node)
- Abnormalities in the gut nervous system (submucosal nerve fiber hyperplasia and ganglionitis, intermuscular nerve fibers proliferate)
- Relatively normal epithelial-mucus secretion is preserved (Goblet cells are usually normal)

Transverse ulceration around the cecum, ileocecal flap, as well as openings. Also, pathological features showed typical caseous necrotizing granuloma, which clearly identified intestinal tuberculosis [11]. The identification points between the three diseases are shown in Table 2. During the course of the disease, tuberculosis infection was found in this patient, but the symptoms did not relieve after 3 months of anti-tuberculosis treatment. According to the report by Kedia et al. [11], when the diagnosis of intestinal tuberculosis and Crohn’s disease is uncertain, it is recommended to try to fight tuberculosis for 3 months. Crohn’s disease is considered if endoscopic or radiological reexamination shows the disease is still active after 2–3 months of anti-tuberculosis treatment. In conclusion, intestinal tuberculosis was excluded and Crohn’s disease was considered.

Studies have shown that SLE concomitant with Crohn’s disease is extremely rare. In this study, the concomitant cases of Crohn’s disease and SLE since 1985 were summarized, and a total of 15 cases were found [7, 12–25]. Table 3 shows comparisons of reported patients with SLE complicating Crohn’s disease. A total of nine cases of SLE prior to Crohn’s disease have been reported. SLE often occurs after the diagnosis of Crohn’s disease, which is mostly caused by drugs involved in treatment. The use of sulfasalazine [26, 27], 5-aminosalicylic acid (5-asa) agents [28, 29] and anti-tumor necrosis factors [20] used in the treatment of IBD may, therefore, lead to drug-induced lupus. There are four relevant case reports, including three cases of SLE induced by treatment with tumor necrosis factor and 1 case of Crohn’s disease merged with lupus encephalopathy. The incidence of SLE coexistence with Crohn’s disease is even more rare, and only two cases have ever been reported. This patient was diagnosed with Crohn’s disease 9 months after the diagnosis of lupus. During the course of the disease, the patient was mainly manifested by repeated intestinal obstruction. Except for the specific antibodies found for lupus, there were no extraintestinal manifestations, so we considered the SLE as co-occurring with Crohn’s disease. We found that among the 15 patients with SLE concomitant with Crohn’s disease, 7 patients were less than 30 years old and 4 patients were more than 50 years old. Consequently, we speculate that SLE concomitant with Crohn’s disease may occur more frequently in people less than 30 years old or more than 50 years old. It was noted that this patient was just in the high-risk group. All of these patients were treated with steroids or immunosuppressants and ended up in remission. Only one male was untreated for economic reasons and eventually died. Perhaps we can speculate that SLE with Crohn’s disease has a better prognosis.

According to the response of Crohn’s disease to steroid treatment, it can be divided into three categories: steroid dependence Crohn’s disease, steroid-refractory Crohn’s disease and steroid intolerance Crohn’s disease. In this case, the symptoms were relieved after treatment with high-dose steroids and enzymatic phenolic esters at the onset of the disease, and the recurrence of symptoms during the course of steroid reduction was considered to be related to steroid reduction. Since lupus was assessed as mildly active according to the systemic lupus erythematosus disease activity index score in 2000, the diagnosis of SLE combined with steroid dependence Crohn’s disease was considered. For steroid dependence Crohn’s disease, steroids are the first line of treatment, and although steroids contribute to Crohn’s disease remission, they are not effective for maintenance treatment. The ECCO Guideline proposed that steroids contribute to the maintenance therapy of immunosuppressants such as methotrexate, azathioprine and cyclophosphamide in patients with recurrent Crohn’s disease [30, 31]. After treated with methylprednisolone and cyclophosphamide, the intestinal obstruction was not completely relieved. Thus, it may be transformed into steroid-refractory Crohn’s disease, and at this condition, tumor necrosis factor-α (TNF-α) was recommended by the guidelines. However, the patient had a history of tuberculosis infection, and TNF-α may induce TB activity, so the final choice of surgical treatment was reasonable.
| Case | Age/gender | SLE/Crohn's disease duration (years) | Symptoms | ESR (mm/h) | Immunological findings | Endoscopic findings | Histopathological examination biopsy | Treatment | Publication date |
|------|------------|--------------------------------------|----------|------------|------------------------|---------------------|-------------------------------------|-----------|----------------|
| 1    | 28/M       | SLE 7 years                          | Diarrhea, pyoderma, gangrenosum | 89         | ANA 1:1280 dsDNA 1:160 | Deep linear and ulceration, pseudopolyps, skip lesion | Acute and chronic inflammation | Prednisolone | 1985 [12]   |
| 2    | 15/F       | SLE 3 years                          | Abdominal pain, diarrhea, blood-stained stool | 68         | ANA 1:1280 dsDNA 1:50 | Multiple ulcers with linear ulcer, skip lesion, pseudopolyps | Infiltration of chronic inflammatory cells in the lamina propria mucosa with marked depletion of goblet cells without vasculitis | Salicylazosulfapyridine | 1989 [13]   |
| 3    | 55/F       | SLE 12 years                         | Intermittent hematochezia, tenesmus and loose bowel movements | 35         | ANA 1:80 dsDNA 1:80 | Multiple ulcers with linear ulcer, diffuse aphthous ulcers | Active colitis with noncaseating granulomas | Prednisolone | 1995 [14]   |
| 4    | 25/F       | Crohn's disease 4 years              | Watery diarrhea, lower abdominal pain, perianal abscesses | N/A        | ANA 1:160 dsDNA 800 IU/mL, pANCA (+) | Longitudinal ulcers and mucosal erosion | Focal cryptitis with noncaseating Salicylazosulfapyridine granuloma | Prednisolone | 1998 [15]   |
| 5    | 37/M       | SLE 9 years                          | Diarrhea, Hematochezia | 65         | ANA 1:320 dsDNA 1:320 | Longitudinal ulcers, linear ulcer, cobblestone appearance, pseudopolyps | Nonspecific colitis without vasculitis | Salicylazosulfapyridine, azathioprine | 1999 [16]   |
| 6    | 49/F       | SLE 5 years                          | Diarrhea, abdominal pain, massive bloody stool | N/A        | ANA (+) dsDNA 234 IU/mL | Emergent operation with a right hemicolectomy was performed. | Transmural fibrosis and inflammation with lymphocyte aggregation, but no evidence of vasculitis. | Prednisolone | 2008 [17]   |
| 7    | 19/F       | Crohn's disease 5 years (SLE induced by infliximab and adalimumab) | Swelling in her fingers, wrists, elbows, knees, and ankles | N/A        | ANA 1:5120 dsDNA 1:40 | N/A | N/A | Mesalazine | 2009 [18]   |
| 8    | 18/F       | Crohn's disease 4 years              | Abdominal pain, diarrhea, keratitis | N/A        | ANA (+) dsDNA (+) | Segmental colonic ulcer | Epithelial granuloma | Prednisolone, azathioprine, chloroquine | Prednisolone | 2001 [19]   |
| 9    | 53/F       | Crohn's disease 6 years (lupus encephalopathy induced by Adalimumab) | Nuchal headache, dizziness, a visual, defect in her right eye, chest pain, pleuritic pain | N/A        | ANA 1:320 dsDNA 1:320 | N/A | N/A | Prednisolone | 2011 [20]   |
| 10   | 27/F       | Crohn's disease 5 years (SLE induced by infliximab) | Multiple joint swelling and pain, abdominal pain | 66         | ANA (+) dsDNA (+) | N/A | N/A | Prednisolone, azathioprine | 2012 [21]   |
| 11   | 55/F       | SLE 36 years                         | GI bleeding, diarrhea | 48         | ANA (+) | N/A | Noncaseating granuloma and no evidence of vasculitis | Infliximab | 2012 [7]    |
| 12   | 40/F       | SLE 2 years                          | Abdominal pain, diarrhea, upper and lower extremities arthralgias | 32         | ANA1:640 dsDNA1:40 | Colonic inflammation with ulcers and pseudopolyps | Supportive of Crohn's disease | Prednisolone, azathioprine, hydroxychloroquine, mesalazine corticosteroids | 2013 [22]   |
| 13   | 34/F       | SLE 21 years (lupus nephritis and Crohn's disease) | Abdominal pain, diarrhea, weight loss | N/A        | N/A | N/A | Supportive of Crohn's disease | Corticosteroids | 2015 [23]   |
| 14   | 22/F       | Concurrence of Crohn's disease FEVER, arthralgia, diarrhea, vomiting | | 73         | ANA 1:160 dsDNA 7.5 IU/mL | A discontinuous cobblestone appearance and longitudinal ulcers | A noncaseating granulomatous lesion in the colonic mucosa | Prednisolone, cyclosporine, infliximab | 2017 [24]   |
| 15   | 71/F       | Concurrence of Crohn's disease Abdominal pain, diarrhea, and SLE | | 130        | ANA1:320 | Multiple ulcers in the terminal ileum | Tended to Crohn's disease (there Prednisolone were ganglion cell and crack shape ulcer) | 2019 [25]   |

ANA, antinuclear antibodies; ESR, erythrocyte sedimentation rate; GI, gastrointestinal; N/A: Not available; SLE, systemic lupus erythematosus.
The patient’s symptoms were relieved after the operation. Maintenance therapy included the application of cyclophosphamide to control SLE and thalidomide to prevent Crohn’s disease recurrence. Thalidomide has a similar effect to TNF-α and has a lower risk of inducing TB activity. Finally, the patient’s symptoms were well controlled, and Crohn’s disease and SLE did not recur.

We hypothesized that the concurrent occurrence of SLE with Crohn’s disease was not accidental. SLE and Crohn’s disease are similar to other immune-mediated inflammatory diseases, but their etiology and pathogenesis are unknown. Currently, it is believed that they are produced by microorganisms and genetic/immune/environmental factors [32]. Autoimmune diseases are known to cluster in families, and studies have found that SLE, Crohn’s disease and certain alleles on major histocompatibility complex-II are related. For example, the alleles of DR2 and DR3 in the human leukocyte antigen region are susceptible to SLE, and these loci genes are also closely related to Crohn’s disease [33,34]. In addition, data from 1305 published and unpublished SLE patients with the Computer Aided Reliability Date (CARD) risk allele showed that only patients with IBD with CARD (908R) gene were at high risk of SLE. Dana et al. used linear regression to analyze 5018 patients with SLE in the CHS database and 25 090 patients excluding SLE and confirmed the correlation between SLE and Crohn’s disease [35].

The coexistence of SLE (subsequently lupus enteritis) and Crohn’s disease in a patient represents a diagnostic challenge, as they have many similar clinical manifestations and signs. In some cases, Crohn’s disease may satisfy the classification criteria of SLE. Therefore, repeated gastrointestinal symptoms in patients with SLE should be considered as Crohn’s disease after infectious and visceral vasculitis are excluded. It is difficult to distinguish lupus enteritis from Crohn’s disease by laboratory tests and imaging, which is where endoscopy and biopsy become key in diagnosis. Especially for patients resistant to methylprednisolone, it is recommended a biopsy is conducted as soon as possible to make a clear diagnosis. Although corticosteroids and immunosuppressive drugs are equally effective for both, they have a different emphasis on the selection of immunosuppression, as well as different treatment intensities. As an example, lupus enteritis is more effective on steroids, cyclophosphamide, enzymatic phenolate esters and calcineurin preparations. Whereas Crohn’s disease patients see more positive results when using hormones, thalidomide and TNF-α. So as a result, early diagnosis is very important for the prognosis of patients with these conditions.

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The patient signed a written informed consent form for the purpose of publication of the results of this case study.

Conflict of interest
There are no conflicts of interest.

References
1 Katsanos KH, Tsianos VE, Tsianos EV. Intolerance of folic acid in a patient receiving methotrexate for Crohn’s disease. J Crohns Colitis 2012; 6:960.
2 Bailey M, Chapin W, Licht H, Reynolds JC. The effects of vasculitis on the gastrointestinal tract and liver. Gastroenterol Clin North Am 1998; 27:747–82, v.
3 Sultan SM, Ioannou Y, Isenberg DA. A review of gastrointestinal manifestations of systemic lupus erythematosus. Rheumatology (Oxford) 1999; 38:917–932.
4 Gladman DD, Ross T, Richardson B, Kulkarni S. Bowel involvement in systemic lupus erythematosus: Crohn’s disease or lupus vasculitis? Arthritis Rheum 1985; 28:466–470.
5 Kröner PT, Tolaymat OA, Bowman AW, Abri A, Lacey BE. Gastrointestinal manifestations of rheumatological diseases. Am J Gastroenterol 2019; 114:1441–1454.
6 Tsachiya M, Okazaki I, Asakura H, Okubo T. Radiographic and endoscopic features of colonic ulcers in systemic lupus erythematosus. Am J Gastroenterol 1975; 64:277–285.
7 Yamashita H, Ueda Y, Kagawuchi H, Suzuki A, Takashashi Y, Kaneko H, et al. Systemic lupus erythematosus complicated by Crohn’s disease: a case report and literature review. BMC Gastroenterol 2012; 12:174.
8 Kurlander DJ, Kirschner JB. The association of chronic “nonspecific” inflammatory bowel disease with lupus erythematosus. Ann Intern Med 1964; 69:799–815.
9 Bernstein CN, Blanchard JF, Rawsthorne P, Yu N. The prevalence of extraintestinal diseases in inflammatory bowel disease: a population-based study. Am J Gastroenterol 2001; 96:1116–1122.
10 Ricart E, Panaccione R, Loftus EV Jr, Tremaine WJ, Harmsen WS, Zinsmeister AR, Sandborn WJ. Autoimmune disorders and extraintestinal manifestations in first-degree familial and sporadic inflammatory bowel disease: a case-control study. Inflamm Bowel Dis 2004; 10:207–214.
11 Kedia S, Das P, Madhusudhan KS, Dattagupta S, Sharma R, Sahni P, et al. Differentiating Crohn’s disease from intestinal tuberculosis. World J Gastroenterol 2019; 25:418–432.
12 Johnson DA, Diehl AM, Finkelstein FD, Cattau EL Jr. Crohn’s disease and systemic lupus erythematosus. Am J Gastroenterol 1985; 80:869–870.
13 Nagata M, Ogawa Y, Hisano S, Ueda K. Crohn’s disease in systemic lupus erythematosus: a case report. Eur J Pediatr 1989; 148:525–526.
14 Buchman AL, Wilcox CM. Crohn’s disease masquerading as systemic lupus erythematosus. South Med J 1996; 88:1081–1083.
15 Nishida Y, Murase K, Ashida R, Sasaki O, Ozono Y, Mizuta Y, et al. Familial Crohn’s disease with systemic lupus erythematosus. Am J Gastroenterol 1998; 93:2599–2601.
16 Shimizu T, Nishinariya S, Son K, Tomita Y, Yoshirhoi, Matsukawa, et al. Crohn’s disease with the onset resembling systemic lupus erythematosus. Nihon Rinsho Menneki Gakkai Kaishi 1999; 22:164–169.
17 Su KY, Tsai ST, Tsay SH, Lee HT, Chen WS, Huang DP. A patient with systemic lupus erythematosus and Crohn’s disease complicated with massive lower gastrointestinal bleeding, mimicking intestinal vasculitis. Lupus 2008; 17:1049–1050.
18 Zella GC, Weinblatt ME, Winter HS. Drug-induced lupus associated with infliximab and adalimumab in an adolescent with Crohn disease. J Pediatr Gastroenterol Nutr 2009; 49:355–358.
19 Toumonde P, Aliche L, Vallée F, Selves J, Duffaut M. Association of disseminated lupus erythematosus and Crohn’s disease. Rev Med Internes 2001; 22:385–388.
20 Vannucchi V, Grazzini M, Pianelli F, Giannotti M, Biasioli C. Adalimumab-induced lupus erythematosus with central nervous system involvement in a patient with Crohn’s disease. J Gastrointestin Liver Dis 2011; 20:201–203.
21 Farkas K, Nagy F, Kovács L, Wittmann T, Molnár T. Anti-tumor necrosis factor-α-induced systemic lupus erythematosus in a patient with metastatic Crohn’s disease – what is the role of anti-TNF antibody? J Crohns Colitis 2013; 7:e143–e145.
22 Katsanos KH, Voulgarli PV, Goussia A, Christodoulou D, Voulgari PV, Goussia A, Oikonomou P, Christodoulou D. Coexistence of Crohn’s disease in a patient with systemic lupus erythematosus: what is the role of anti-TNF antibody? World J Gastroenterol 2013; 20:2431–2436.
23 João-Magalhães M, Lago P, Pedrotto I. Crohn’s disease and systemic lupus erythematosus: a rare and challenging association. Rev Esp Enferm Dig 2015; 107:394–395.
24 Kagaia Y, Sakamoto H, Yano T, Sunada K, Lefer AK, Niki T, et al. Mucosal healing of Crohn’s disease in a patient with concurrent systemic lupus erythematosus using infliximab. Clin J Gastroenterol 2017; 10:244–249.
25 Zhu XL, Xu XM, Chen S, Wang QM, Zhang KG. Lupus enteritis masquerading as Crohn’s disease. BMC Gastroenterol 2019; 19:154.
26 Carr-Locke DL. Sulfasalazine-induced lupus syndrome in a patient with Crohn’s disease. Am J Gastroenterol 1982; 77:614–616.
27 Clementz GL, Dolin BJ. Sulfasalazine-induced lupus erythematosus. Am J Med 1988; 84:535–538.
28 Pent MT, Ganapathy S, Holdsworth CD, Channer KC. Mesalazine induced lupus-like syndrome. BMJ 1992; 305:159.
29 Kirkpatrick AW, Bookman AA, Habal F. Lupus-like syndrome caused by 5-aminosalicylic acid in patients with inflammatory bowel disease. Can J Gastroenterol 1999; 13:159–162.
30 Gomollón F, Dignass A, Annese V, Tilg H, Van Assche G, Lindsay JO, et al.; ECCO. 3rd European Evidence-based Consensus on the Diagnosis and Management of Crohn’s Disease 2016: part 1: diagnosis and medical management. J Crohns Colitis 2017; 11:3–25.
31 Gionchetti P, Dignass A, Danese S, Magro Dias FJ, Rogler G, Lakatos PL, et al.; ECCO. 3rd European Evidence-based Consensus on the Diagnosis and Management of Crohn’s Disease 2016; part 2: surgical management and special situations. J Crohns Colitis 2017; 11:135–149.
32 Nikitakis NG, Papaioannou W, Sakkas Li, Kousvelari E. The autoimmunity-oral microbiome connection. Oral Dis 2017; 23:828–839.
33 Fernando MM, Stevens CR, Walsh EC, De Jager PL, Goyette P, Plenge RM, et al. Defining the role of the MHC in autoimmunity: a review and pooled analysis. Plos Genet 2008; 4:e1000024.
34 David T, Ling SF, Barton A. Genetics of immune-mediated inflammatory diseases. Clin Exp Immunol 2018; 193:3–12.
35 Shor DB, Dahan S, Comaneshter D, Cohen AD, Amital H. Does inflammatory bowel disease coexist with systemic lupus erythematosus? Autoimmun Rev 2016; 15:1034–1037.