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Case Report
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A Case Report of Crohn's Disease Combined with Psoriasis

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Abstract

Both Crohn's Disease (CD) and psoriasis are autoimmune diseases whose pathogenesis is unclear. CD mainly presents as chronic inflammatory lesions of the gastrointestinal tract and often involves extraintestinal organs, including the skin, whereas psoriasis mainly presents as chronic inflammatory lesions of the skin. Here, we present a case of a 28-year-old man with a 1-year history of recurrent abdominal pain. He underwent intestinal CTE, colonoscopy, and histopathological evaluation, and in combination with the patient's past medical history, a diagnosis of CD with psoriasis was considered. In conclusion, CD with psoriasis is a relatively rare clinical case, and their relationship needs further studies to understand.

Keywords: Crohn's disease; Psoriasis; Biologics

Introduction

CD is a chronic, relapsing, and unexplained inflammatory disease that can affect the entire gastrointestinal tract. Its pathogenesis is unclear and may be related to genetic, immune, environmental and gut microbiota. Psoriasis is a systemic inflammatory disease, and CD is one of the major comorbidities of psoriasis [1]. To data, CD has become a global disease with increasing incidence in newly industrialized countries where societies have become more westernized [2]. Epidemiological studies have shown that patients with psoriasis are at higher risk of developing CD than the general population, but the relationship between psoriasis and CD remains unclear and psoriasis and CD share many common genetic loci and overlap in inflammatory pathways [3]. In this paper, we report a special case of CD with psoriasis, which emphasizes the clinical thinking of integrative medicine in individualized diagnosis and treatment strategies, and provides some reference for clinical work.

Case Presentation

A 28-year-old male patient was seen at the Department of Gastroenterology. He had a 1-year history of intermittent periumbilical distension and pain, and the disease has relapsed and worsened in the past two months. He underwent a colonoscopy before admission, which showed "multiple erosive ulcers and proliferative changes in the large intestine". He did feel that he had been in general mental, physical and sleep conditions, poor appetite, stool as described above, urine as usual, and weight loss of about 5 kg since the onset of the disease. He had a 10-year history of psoriasis, denied a family history of autoimmune disease, and his general physical examination, including abdominal examination, and was unremarkable.

Physical examination

Vital signs: T: 36.4°C, P: 103 bpm, R: 20 bpm, BP: 98/79 mmHg, clear consciousness, mental health, no enlargement of superficial lymph nodes, no yellow sclera, clear breath sounds in both lungs, no dry and wet rales, neat heart rhythm, flat and soft abdomen, no obvious pressure pain and rebound pain, no subcostal liver and spleen, no obvious masses palpable, mobile turbid sounds (-), slightly active bowel sounds, no edema in both lower limbs. His dermatological examination (Figure 1) showed that: Infiltrative erythema, papules and maculopapular rash of soybean to palm size were seen on the trunk and extremities of the patient, with partial fusion of the lesions, accumulating 10% of the body surface area, with a large amount of white scaly flakes, positive for scraping wax phenomenon, film phenomenon and punctate hemorrhage phenomenon, no pustules, joint deformity and generalized flushing were seen.

Laboratory and ancillary tests

Complete blood count five categories: Platelet count 652 × 10^9/L↑ (125-350), lymphocyte

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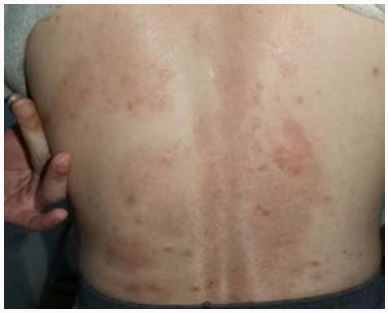

Figure 1: Specialist skin examination: Skin changes on the back.

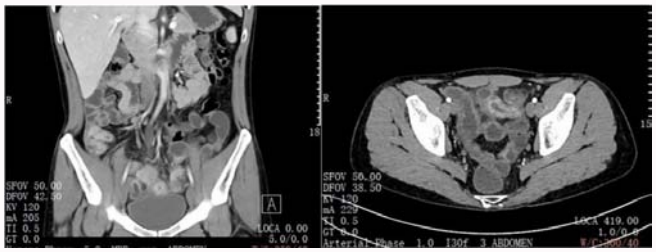

Figure 2: The intestinal CTE; Pelvic cavity ileum, back to the blind and the right colon bowel wall thickening with obvious.

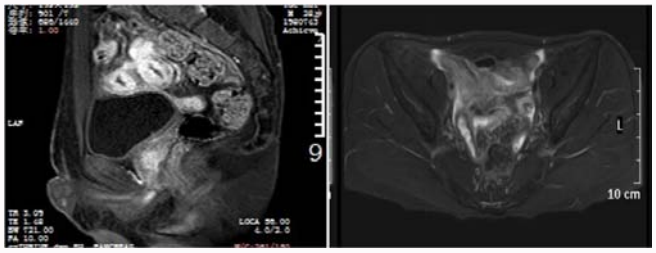

Figure 3: MR enhancement scan of the pelvis: Thickening and swelling of the ileocecal wall in the pelvis with peri-intestinal exudation, more inflammatory lesions are considered.

count 0.75 × 10^9/L↓ (1.1-3.2), neutrophil percentage 75.40↑ (40-75), lymphocyte percentage 11.50%↓ (20-50); C-reactive protein: 3.76 mg/dL↑ (0~(0.6); Erythrocyte Sedimentation Rate: 27 mm/h↑ (0~15); liver function eleven: Alanine aminotransferase 8.1 U/L↓ (9~50), aspartate aminotransferase 10.7 U/L↓ (15~40), albumin 36.1 g/L↓ (40~55), white sphere ratio 1.19↓ (1.2~2.4); coagulation routine: Fibrinogen 5.080 g/L↑ (2-4); EBV-related antibody test: EBV capsid antigen IgG antibody, EBV nuclear antigen IgG antibody, EBV early antigen IgG antibody positive, the rest negative; stool examination, bacterial culture, urine examination, pancreatitis screening, electrolyte five, kidney function five, A function three, anti-nuclear antibody profile (IgG) test, rheumatoid full set, vasculitis screening, immunoglobulin quantification, pre-transfusion examination, serum troponin, cardiac enzyme profile, AFP, CA199, CEA, EBV-DNA, CMV-DNA, PPD test, T-SPOT were not found to be significantly abnormal. Ultrasound of superficial lymph nodes throughout the body and CT scan of the chest were abnormal. He also had an intestinal CTE, MR enhancement scan of the pelvis, gastrointestinal endoscopy and histopathological examination, and transoral small bowel examination. The results are as follows: The intestinal CTE (Figure 2): Pelvic cavity ileum, back to the blind and the right colon bowel wall thickening with obvious reinforcement, combined with the patient's medical history, inflammatory bowel disease, Crohn's

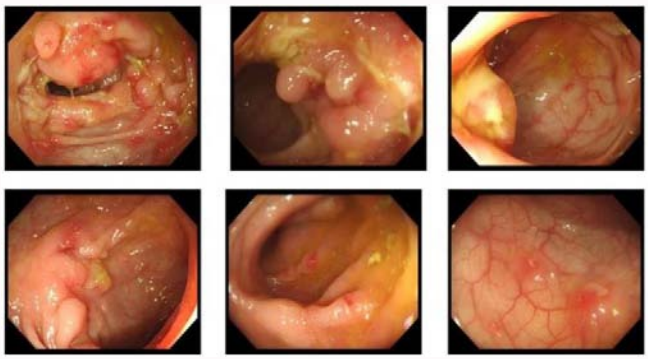

Figure 4: Colonoscopy (The biopsy was performed twice); the ileocecal valve was obviously swollen, making endoscopic passage difficult; segmental multiple indurated ulcerative changes were seen from the ascending colon to the transverse colon, and the hepatic flexure was biopsied twice; scattered erosions were seen in the descending colon and sigmoid colon, and white moss was seen apically, and one biopsy was performed in the descending colon.

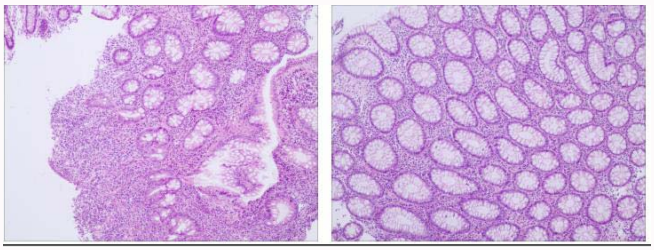

Figure 5: Colonoscopic pathology.

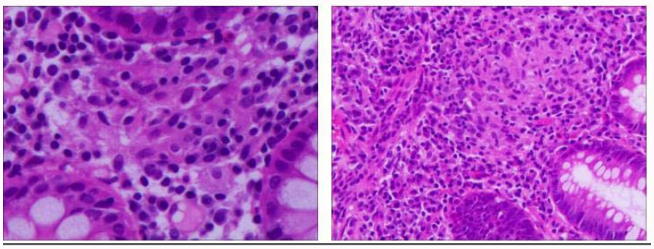

Figure 6: Colonoscopy pathology immunohistochemistry.

disease, likely to identify intestinal tuberculosis, white disease, suggest combined with clinical, soft tissue shadow inside pelvic cavity intestinal gap with abnormal strengthening, stingy, does not exclude the lesions involving the mesangial, and adjacent small intestinal wall perforation and package, the right liver lobe calcifications.

MR enhancement scans of the pelvis (Figure 3): 1. Thickening and swelling of the ileocecal wall in the pelvis with peri-intestinal exudation, more inflammatory lesions are considered, it is recommended to review, 2. A small amount of fluid in the pelvis. 3. Cystic foci in the sacral canal and a few subcutaneous exudative changes in the buttocks next to the bilateral sciatic nodes, 4. Possible sacroiliac arthritis in the left side.

Colonoscopy (Figure 4): Multiple erosive ulcers and proliferative changes in the colon (biopsy, nature to be determined), internal hemorrhoids.

Colonoscopic pathology (Figure 5): 1. (Cecum) chronic active inflammation of the mucosa with erosion, cryptitis and crypt abscess, individual glands twisted and slightly branched, 2. (Hepatic flexure)

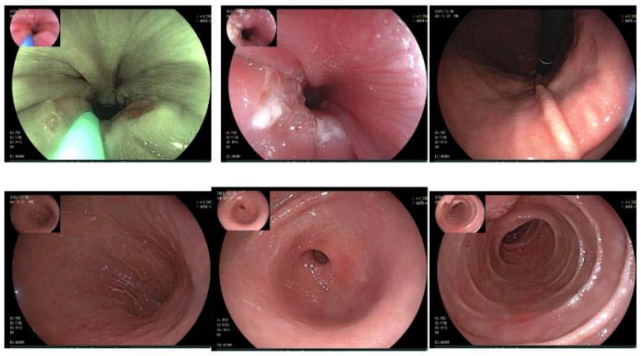

Figure 7: Gastroscopy.

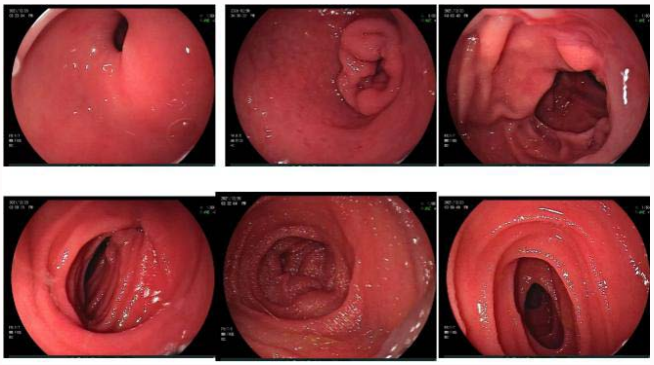

Figure 8: Transoral small bowel examination.

chronic active inflammation of the mucosa, 3. (Descending colon) chronic active inflammation of the mucosa with cryptitis and crypt abscess.

Colonoscopy pathology immunohistochemistry (Figure 6): At the request of clinical diagnosis and treatment, this patient N2111653-3 immunohistochemistry test, renumbered as N2111763, integration of the previous section, the results are as follows the degree of inflammation of the tissue sent for examination varies, focal interstitial suspicious micro granulomatous inflammation, inclined to Crohn's disease, please take into account the clinical history and endoscopic views, excluding drug-related injuries and other diseases, immunohistochemistry CMV (-), EBER in situ hybridization negative, *Mycobacterium tuberculosis* PCR test negative (G2103342).

Gastroscopy (Figure 7): Chronic non-atrophic gastritis with erosion (gastric body and sinus biopsy). Gastroscopy pathology: 1. Mild chronic inflammation of the mucosa (anterior wall of the gastric sinus) with focal activity, 2. Mild chronic inflammation of the mucosa (mid-body greater curvature of the stomach).

Transoral small bowel examination (Figure 8): Transoral small bowel examination was evaluated without significant abnormalities.

Combined with the patient's clinical manifestations, past medical history and related auxiliary examinations, the current diagnosis is considered as follows: (1) Crohn's disease ileocolic, penetrating, active, and mild; (2) Psoriasis; (3) Erosive gastritis; (4) abdominal infection.

Treatment history: After improving systemic status with levofloxacin injection for anti-infection, oral mesalazine extended-release tablets for intestinal repair, snake venom injection for hemostasis, pantoprazole injection for gastric protection, topical Xitolio treatment for skin, energy support, Partial Enteral Nutrition (PEN), etc., according to the "Diagnosis and treatment of inflammatory bowel disease of Consensus opinion (2018-Beijing)", 300 mg of infliximab was administered on the 11th day of hospitalization. After receiving a total of 6 times of infliximab treatment, blood test results showed that C-reactive protein, erythrocyte sedimentation rate and albumin gradually returned to the normal range. The repeat colonoscopy suggested that (Figure 9): Multiple scar changes near the terminal ileum, ileocecal valve, cecum and hepatic flexure; transverse colon, descending colon, sigmoid colon and rectal lumen had normal morphology, smooth mucosal surface, clear vascular network, and no erosion, ulcer or swelling was seen. In addition, the clinical symptoms of the patient were relieved, the area of skin lesions was significantly reduced, and the body weight increased by 3 kg.

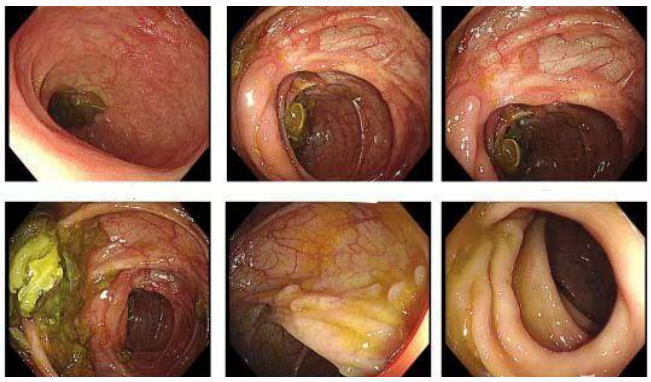

Figure 9: The repeat colonoscopy suggested that: multiple scar changes near the terminal ileum, ileocecal valve, cecum and hepatic flexure.

Discussion

CD, together with Ulcerative Colitis (UC), is called Inflammatory Bowel Disease (IBD), which is an abnormal immune response in individuals with genetic susceptibility to the stimulation of environmental factors and the joint participation of intestinal microorganisms, ultimately manifesting as an inflammatory response of the intestine. It is a systemic inflammatory disease with a predominantly intestinal inflammatory response. In addition to intestinal involvement, up to 50% of patients with IBD may present with at least one Extraintestinal Manifestations (EIM), which may involve almost any organ or system, and EIM occurs more frequently in patients with CD than in those with UC [4]. The skin is one of the most frequently involved organs in EIM and is classified as atopic and reactive depending on the pathogenesis, and its atopic and reactive lesions are usually similar to the pathogenesis of CD, and the appearance of the rash is often associated with Gastrointestinal (GI) disease activity, predicting the need for more aggressive treatment, including early use of biological agents [5]. If involved extraintestinal can significantly affect the Quality of Life (QoL) of patients with CD, sometimes more than intestinal disease.

Psoriasis is a chronic, relapsing, inflammatory skin disease. The etiology of psoriasis is currently unclear and is thought to be a disease caused by a combination of immune factors, environmental, and psychological factors in a genetic background. More and more studies have shown that a strong link between diseases such as cardiovascular disease, obesity, inflammatory bowel disease, metabolic syndrome, tumors and infections and psoriasis [6-7]. The clinical manifestations

include: Common, visceral, pustular, pustular, and erythrodermic [8]. This patient has obvious symptoms and signs.

Currently, Kim et al. found that patients with CD have an increased risk of psoriasis and that psoriasis increases the risk of CD [8], and another study found similar results, with patients with CD being 7 times more likely to develop psoriasis than the general population, and patients with both CD and psoriasis having an earlier age of onset than those with either disease alone [9]. In an article on the epidemiological association between IBD and psoriasis, 12,502 patients with psoriasis and 24,287 controls were studied, and the prevalence of CD was significantly higher in patients with psoriasis than in controls [10]. Even when patients treated with anti-TNF drugs were excluded, this association remained statistically significant, so there was a strong correlation between psoriasis and CD.

The exacerbation of psoriasis in patient was followed by an increase in CD symptoms, suggesting a close correlation between the two. The reason for this association lies in the sharing of susceptibility genes and a common immune mechanism. Genome-wide association studies, some genetic correlations between psoriasis and IBD have been reported by genome-wide association analysis studies (Genome Wide Association Study), which have identified 13 psoriasis susceptibility loci (called PSORS1-13) and 28 IBD susceptibility loci (called IBD1-28). However, the pathogenic relevance of these findings must be tested in an experimental setting. The main susceptibility loci for the association of psoriasis with IBD are located mainly on chromosomes 6p22, 16q, lp31 and 5q33. One study identified the chromosome 6p22 single nucleotide polymorphism rs6908425 as a CD susceptibility locus [11]. Many studies have been reported on the molecular mechanisms of psoriasis and IBD, but few have attempted to compare these mechanisms, especially when these conditions affect the same patient at the same time [12]. According to current knowledge, both psoriasis and CD recognize two pathogenic moments, the first involving innate immunity triggered by unknown stimuli and the second involving adaptive immunity, which is due to the release of cytokines dendritic cells from cells of the innate immune system that affect the activity of T-cell subtypes such as T-helper17 (T-Helper17, Th17) and T-regs.

Since psoriasis and Crohn's disease are very closely related, it remains to be further investigated whether they are manifestations of the same disease in different systems or 2 separate diseases. The commonalities in the pathogenesis of these 2 diseases may provide some ideas for treatment. Biological agents are now widely used in the treatment of CD and have become the main treatment modality for the disease, such as the anti-tumor necrosis factor alpha monoclonal Anti-TNF, the anti-integrin monoclonal antibody vedelizumab, the anti-IL-12/23 monoclonal antibody Ustekinumab, and the Janus kinase inhibitor tofacitinib [13]. Some drugs have been studied that can be used to treat these 2 diseases simultaneously, such as needle infliximab and adalimumab, Ustekinumab against anti IL-23, and secukinumab against IL-17, of which anti-TNF and anti-IL-23 have been successfully used in the treatment of the disease [14]. More and more biologic agents are being used in the clinic, providing more options for the treatment of CD patients. The selection of biologic agents requires comprehensive consideration of guidelines and consensus recommendations, clinical characteristics of CD patients, efficacy of biologic agents, safety of drug use, Pharmacoeconomics, patient's wishes, and other aspects to develop an individualized treatment plan with an adequate balance of risks and benefits. Future large-scale clinical trials are still needed to investigate the balance between the minimum drug concentration of biologics and the maximum benefit to patients.

Patients with suspected Crohn's disease need to be referred to a specialist for further definitive diagnosis with attention to regular follow-up and standardized treatment [15]. Patients with CD combined with psoriasis have a high prevalence of anxiety and depressive symptoms, with up to one-third affected by anxiety symptoms and one-quarter by depressive symptoms, as well as an increased prevalence in patients with active disease [16]. Encouraging gastroenterologists to screen for and treat these disorders may improve the prognosis of patients [17]. In recent years, it has been increasingly recognized that due to the complexity of IBD disease and the number of disciplines involved in treatment, Multidisciplinary Team (MDT) collaboration is required to properly diagnose the disease as well as provide efficient and standardized treatment for patients and achieve optimal individualized treatment of IBD patients [18].

There are several outstanding questions: Is the early use of biologics recommended in the context of IBD-related EIM? Is combination therapy preferable to EIM treatment?

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