Collagenous Colitis and Ankylosing Spondylitis☆

Colitis colágena y espondilitis anquilosante

To the Editor:

Collagenous colitis (CC) is a type of microscopic colitis, which along with lymphocytic colitis has a low prevalence and an unknown etiology, characterized by persistent watery diarrhea. Its association with autoimmune diseases has been described in different studies.[1–6] However, the coexistence with spondylitis is rare.

We report the case of a 55-year-old man diagnosed with ankylosing spondylitis at age 25 according to the New York criteria and with positive HLA-B27. He did not refer a family history of psoriasis, uveitis, urethritis or prior inflammatory bowel disease. He began treatment with nonsteroidal antiinflammatory drugs (NSAIDs), showing a moderate clinical response. Five years after the diagnosis he underwent a total hip arthroplasty. During the 20 years of progression of the disease, he remained with acceptable pain control and normal acute phase reactants on full-dose NSAIDs.

In June 2010 he presented diffuse colicky abdominal pain, accompanied by constant diarrhea, without fever. He lost 2 kg in a month. He improved his diet with astringents to avoid diarrhea but had clinical recurrence after abandoning the diet. During the first days, the intestinal manifestations were accompanied by limited knee arthritis which lasted for a week. Abdominal examination was normal. The laboratory results showed no significant changes except hemoglobin 11.7 g/dL and slightly elevated C-reactive protein of 8.8 mg/L. The search for parasites and bacteria, including *Clostridium difficile*, was negative.

Colonoscopy was normal and serial biopsies were collected, which showed a subepithelial collagen band and irregular distribution, with an increased inflammatory infiltrate in the lamina propria and focal intraepithelial lymphocytosis compatible with CC.

Treatment was started with budesonide tapering until suspension at 9 months, with resolution of abdominal symptoms. The patient has not presented any relapses and is asymptomatic, with NSAIDs at lower doses than the previous ones.

CC was first described in 1976 by Lindström. It has an annual incidence of 1.1–5.2/100 000 and usually occurs in middle-aged women. The pathogenesis is unknown, although familial cases have been described, but little data exist on its genetic predisposition. Various etiologies have been suggested, one of which is its possible association with NSAID use.[1,6]

Clinically, it is characterized by an increase of stool frequency (4–9 per day), liquid diarrhea without fever and a chronic intermittent course, accompanied by nausea, diffuse abdominal pain, fecal urgency and weight loss. Colonoscopy and barium enema are usually normal. The diagnosis is made by biopsy of the colonic mucosa, where there is a thick subepithelial collagen band ranging from 7 to 100 m, intraepithelial lymphocytosis and inflammatory infiltrates in the lamina propria. For its treatment, possible related drugs should first be removed antidiarrheal medication employed. If symptoms persist, budesonide is recommended and in case of a lack of response sulfasalazine, cholestyramine or prednisone 0.5–1 mg/kg may be indicated. Diarrhea may be resolved in weeks, with or without treatment, but relapses are frequent. CC has not been associated with an increased mortality.[6]

CC has been described in connection with autoimmune diseases, with a frequency ranging from 13% to 56%.[2,3] To date, the association of spondylitis and CC has been described only as reports in the literature, with 11 cases collected.[2,4,7]

Spondylitis are associated with various extra-articular manifestations. Besides ulcerative colitis and Crohn's disease, joint involvement also occurs in gastrointestinal parasitic infections, pseudomembranous colitis, Whipple's disease, Behcet's disease, celiac disease or after[8] bariatric surgery. For some authors, CC could be considered a cause of enteropathic arthritis.[2,5]

In our case, the clinical worsening of the joints when gastrointestinal symptoms began lead us to believe that there is a correlation between the two entities that could act as a trigger for the outbreak of arthritis.[5] Moreover, one cannot rule out the contribution of NSAIDs in the development of CC. It is postulated that inhibition of prostaglandin synthesis increases intestinal permeability, allowing access of the luminal content to the lamina propria. This would cause inflammation and pericriptal fibroblast activation, leading to thickening of the collagen layer.[1] Most of our patients are treated with NSAIDs; therefore, further studies are needed to find out what role they play in the development and worsening of the natural course of CC.

When faced with a patient with spondyloarthritis that during the course of illness presents diarrhea, we must consider CC as a possible differential diagnosis of enteropathic arthritis.

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