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Association of Celiac Disease and Crohn's Disease: A Case Report of 3 Patients

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Abstract | **Case Report**

The association between celiac disease (CeD) and Crohn's disease (CD) is rare, but some clinical cases suggest a pathophysiological interaction between these two chronic inflammatory bowel diseases. We present three observations of patients with this dual association. The clinical manifestations, biological results, endoscopic investigations and treatments varied from case to case, but all patients showed a favourable response to combined treatment with a gluten-free diet and immunosuppressive or biological therapy. These observations highlight the importance of considering the co-occurrence of coeliac disease and Crohn's disease in the differential diagnosis and treatment of patients with complex gastrointestinal symptoms.

Keywords: Crohn's disease, celiac disease, association, etiopathogeny.

INTRODUCTION

Celiac disease (CeD) is a chronic autoimmune enteropathy induced by exposure to gluten in genetically predisposed individuals. It affects around 1% of the world's population, although this prevalence may be underestimated due to the diversity of clinical presentations and the high number of paucisymptomatic or silent forms.

Crohn's disease (CD) is a chronic inflammatory disease of the digestive tract with multifactorial aetiology, involving genetic, immunological and environmental factors. It manifests as transmural inflammation that can affect the entire digestive tract, with a predilection for the terminal ileum.

Although the two diseases are distinct, several studies have reported a possible association between them, suggesting common immunopathogenic mechanisms. However, this association remains rare and poorly documented in the literature, posing a diagnostic and therapeutic challenge for clinicians.

We report here three clinical observations illustrating this unusual association between coeliac disease and Crohn's disease, with the aim of discussing the diagnostic difficulties, therapeutic implications and underlying pathophysiological hypotheses.

OBSERVATIONS

OBSERVATION 1

The patient was Miss N.I., aged 22, followed in paediatrics since the age of 13 for celiac disease, diagnosed on the basis of chronic liquid diarrhoea (5 to 6 stools per day), delayed height and weight and delayed puberty, as well as microcytic hypochromic anaemia (ferritin: 7 ng/mL) and hypo albuminemia (14 g/L). Celiac serology was strongly positive : IgA anti-transglutaminase antibodies at 2178 IU/mL and IgG at 164 IU/mL.The patient was put on a strict gluten-free diet from June 2019. The initial course was marked by persistent diarrhoea, which later became bloody and glairy. In addition, she developed complex ano-perineal lesions, including anal fistulae (some perianal, others suggesting tracts to the labia majora), and oedematous marisci with regenerative buds. A proctological operation was carried out to insert a surgical drain.

Biological tests revealed severe anaemia (haemoglobin: 7.2 g/dL), thrombocytosis (978,000/mm³) and hypoalbuminemia at 32 g/L.

The clinical and biological picture, together with the ano-perineal manifestations, led to a revision of the initial diagnosis in favour of Crohn's disease. The patient was put on azathioprine-based immunosuppressive therapy, while maintaining a gluten-free diet. The clinical and biological outcome was favourable.

In February 2025, she was admitted to hospital with severe acute colitis, manifested by bloody diarrhoea (around 10 stools/day) and chronic proctalgia. The clinical examination revealed a delay in growth and development, paleness of the skin and mucous membranes, diffuse abdominal tenderness (maximum in the periumbilical area), and inflammatory proctological lesions: circumferential marisci, external fistulous orifice at 9 o'clock (closed, no discharge). Biologically, the patient presented with anaemia with hg at 6.5g/dl and lymphopenia at 540, so the decision was to stop the immunosuppressive treatment and switch to biotherapy.

OBSERVATION 2

The patient was Mrs G.R., aged 44, with a medical history of treated arterial hypertension. She had been followed for several years for coeliac disease, diagnosed at the age of 40 during an aetiological work-up for chronic watery diarrhoea. An oesophago-duodenal fibroscopy (OGDF) revealed Marsh stage 3 villous atrophy, confirming the diagnosis of coeliac disease. However, at the age of 44, the patient was admitted to hospital because of the onset of bloody diarrhoea associated with flare-ups and remissions, as well as Koenig's syndrome (abdominal pain with perianal lesions).

Endoscopic exploration revealed rectal lesions, with a recto-vaginal fistula, and colonoscopy showed an erythematous and fragile mucosa, particularly haemorrhagic. Polypoid-like lesions were seen at 15 cm from the anal margin, accompanied by deepening ulcerations at 40 cm from the anal margin, requiring progress to be halted due to severe endoscopic signs. Pathology showed chronic colitis in acute relapse, with signs of cryptitis and cryptic abscesses.

Biologically, the patient presented with microcytic hypochromic anaemia (haemoglobin at 10 g/dL) and hypoferritinaemia, and was diagnosed with severe colitis.Despite initial treatment with corticosteroid therapy, the patient became resistant to these treatments and developed intolerance to mercaptopurine (severe anaemia at 7 g/dL after 3 years of treatment). A therapeutic escalation was envisaged with anti-TNFα, and she received infliximab until the 14th week, but the patient was lost to follow-up after treatment was stopped. On her return, a new relapse of the disease was treated with oral corticosteroids, followed by methotrexate 25 mg/week, but the treatment was stopped due to haematological toxicity (severe anaemia). Biotherapy was not initiated, but the patient was put on 5-ASA 3 g/d with a gluten-free diet, with good clinical and endoscopic progression.

OBSERVATION 3

The patient was Mrs H.Z., aged 65, being treated for undocumented heart disease and put-on anti-arrhythmic medication. She has been followed since 1996 for ileo-caecal Crohn's disease of the stenosing phenotype, clinically revealed by liquidy diarrhoea sometimes accompanied by mucus (7 stools per day). The course was marked by relapses interspersed with remissions, associated with Koenig's syndrome, without rectal syndrome. Ano-perineal lesions in the form of lateralized anal fissures were noted.

The evolution was marked by the appearance of a sub-occlusive syndrome, which led to the indication for a surgical procedure consisting of ileo-caecal resection. Biological findings included elevated calprotectin, an inflammatory syndrome and microcytic hypochromic anaemia (haemoglobin 10 g/dl).

Endoscopically, the disease progressed as far as the ileocolic anastomosis, where the mucosa was moderately prepared, with some blind areas. Erythematous mucosa was seen at the anastomosis. The resection specimen showed an area of segmental stricture interspersed with macroscopically healthy areas, as well as foci of whitish coating and a blind fissure. Enteric MRI showed bi-segmental thickening of the last anastomotic ileal loop with target contrast, consistent with evidence of active Crohn's disease.

Therapeutically, the patient is intolerant of immunosuppressive drugs, in particular azathioprine (digestive intolerance) and methotrexate (hepatic cytolysis), which led to the indication of a therapeutic step up to biotherapy with anti-TNF alpha.

At the same time, the patient presented with atypical chronic epigastralgias, persistent microcytic hypochromic anaemia despite iron treatment, and peripheral arthralgias with an inflammatory appearance. OGDF revealed an erythematous and atrophic antifundial mucosa, with a polypoid ‘earlobe’ lesion in the fundus and slightly effaced duodenal folds. Pathology showed chronic non-atrophic fundic gastritis with minimal activity and focal abrasions with no evidence of malignancy.

Histological examination of the duodenal mucosa reveals moderate subacute to chronic inflammatory changes, with partial villous atrophy (modified Marsh stage 3a, Corazza grade B1).

The immunological work-up revealed negative anti-transglutaminase IgA and anti-endomysium antibodies, as well as negative HLA DQ8 and positive HLA DQ2. The diagnosis of coeliac disease was accepted.

The patient was put on a gluten-free diet and biotherapy, with a good clinical and biological outcome at follow-up.

DISCUSSION

Celiac disease and Crohn's disease are two chronic dysimmune enteropathies with different etiopathogenies rarely found in the same person, but according to the literature, the prevalence of IBD, particularly Crohn's disease, in patients with celiac disease is 5 to 10 times higher than in the general population. However, some studies suggest a higher prevalence of celiac disease in patients with Crohn's disease, while others find no significant increase in risk compared with the general population [1] [2], Sonnenberg and Genta conducted a large-scale case-control study in 2023 and found that celiac disease is less common in patients with Crohn's disease or ulcerative colitis [3]. In contrast, the study by Mårild et al is a Swedish national cohort of over 100,000 patients showed that patients with coeliac disease have an increased risk of developing chronic inflammatory bowel disease, particularly Crohn's disease, and vice versa [4].

This coexistence is most often found in young male subjects, unlike in our series, where the 3 patients were women; this epidemiology is not explained [5] [6].

This coexistence should be borne in mind when the diagnosis is unclear or the patient's condition does not improve with appropriate treatment, as in our patients in the various series in the literature [5] [7].

The predominant clinical symptomatology in both diseases is diarrhoea, and the characteristics of this diarrhoea point to one or other of the two diseases. In coeliac disease, the diarrhoea is fatty and abundant, whereas bloody diarrhoea indicates IBD. The presence of anoperineal lesions points towards a diagnosis of Crohn's disease, as in the case of our patients, who developed ano-perineal lesions and so the diagnosis was rectified [2] [7] [8]. Extradigestive lesions may also be observed, as in the case of our third patient who developed inflammatory joint pain that may be explained by the malabsorption syndrome found in both diseases.

The main complications of celiac disease, ulcerative jejunitis and intestinal lymphoma, cause more severe clinical manifestations that may resemble Crohn's disease, such as acute and persistent abdominal pain, weight loss, signs of intestinal obstruction or gastrointestinal bleeding, fever or signs of marked malnutrition [9]. It has been suggested that complicated celiac disease should be considered in CD patients who do not respond to immunosuppressive or biological therapies [2].

The etiopathogenesis of the association between Crohn's disease (CD) and coeliac disease (CoeD) in the same individual is based on a complex interaction between genetic, immunological and environmental factors. Genetic mutations, particularly those associated with HLA antigens, play a central role in this shared pathogenesis. The presence of HLA-DQ2 or HLA-DQ8 haplotypes is essential for the development of CoeD. These genes encode major histocompatibility complex (MHC) class II molecules, which are responsible for the presentation of gliadin peptides to T cells, triggering a pathological immune response [10]. A study published in 2023 examined the involvement of HLA-DQ molecules in the development of coeliac disease (CoeD) and suggested that HLA-DQ8, in particular haplotype DQA103: 01-DQB103:02, may also play a role in susceptibility to Crohn's disease (CD). Although this study focuses mainly on CeD, it highlights the importance of HLA-DQ molecules in chronic inflammatory bowel diseases, including CD, and could provide clues to understanding the immunological mechanisms shared by these two diseases [11].

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At the same time, recent research has highlighted the importance of intestinal dysbiosis in the development of both diseases. The intestinal microbiota plays a fundamental role in maintaining digestive homeostasis, preserving intestinal permeability and regulating mucosal immunity. In both coeliac disease and Crohn's disease, qualitative and quantitative alterations in the microbiota have been observed.

In patients with CeD, a reduction in beneficial bacteria such as Lactobacillus and Bifidobacterium has been reported, in favour of pro-inflammatory species such as Proteobacteria [12]. Similarly, CD is associated with a reduction in Faecalibacterium prausnitzii, a butyrate-producing bacterium with anti-inflammatory properties [13] [14]. Increased intestinal permeability is

a key mechanism in the pathogenesis of Crohn's disease (CD) and celiac disease (CeD). In these two diseases, alteration of the intestinal barrier allows food fragments such as gliadin to enter the body, where they are perceived as foreign agents by the immune system. This translocation of antigens triggers T-cell activation and an inflammatory response, often mediated by cytokines such as TNF-α, IL-6 and IFN-γ. These cytokines contribute to chronic inflammation in intestinal tissues, exacerbating the symptoms of Crohn's disease. Continued exposure to these gliadin fragments in a dysfunctional gut environment could both aggravate Crohn's disease and promote the development of celiac disease, and vice versa, creating a breeding ground for the simultaneous onset of both conditions in the same patient. This dynamic underlines the interconnection between the two diseases, fuelled by altered intestinal permeability and immune activation.

Finally, the impact of environmental factors, such as exposure to infections or the use of antibiotics, has also been identified as a possible trigger for this association [16].

Therapeutically, the treatment of celiac disease is based exclusively on a strict gluten-free diet, whereas CD often requires immunosuppressive or biological treatment. However, some studies have observed symptomatic improvement in CD patients following the introduction of a gluten-free diet, as in our series [17].

Moreover, knowledge of this association calls for particular vigilance. For example, in patients with Crohn's disease resistant to conventional treatments, screening for CeD may be appropriate, particularly in cases of persistent malabsorption. Conversely, persistent digestive symptoms in a well-adherent coeliac patient should prompt a search for associated IBD.

The emergence of targeted treatments (anti-TNF, anti-integrin, JAK inhibitors) offers promising avenues for patients suffering from both diseases. Finally, modulation of the intestinal microbiota by probiotics or fecal microbiota transplantation (FMT) is a rapidly expanding field of research [18] [19].

Conclusion

The association between Crohn's disease and celiac disease is now well documented. It is based on solid epidemiological data, shared immunological and genetic mechanisms, and common environmental factors. A better understanding of this co-occurrence could help refine diagnostic and therapeutic strategies. Clinicians must remain alert to the possibility of a dual pathology, particularly in the case of atypical or treatment-resistant forms. Future studies are needed to assess the value of integrated therapeutic strategies targeting these two entities.

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