Coeliac Disease with Rheumatoid Arthritis: An Unusual Association

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Abstract Rheumatoid arthritis (RA) and celiac disease (CD) belong to the autoimmune disease family. Even though these diseases are separate entities, they share multiple aspects. This association has rarely been reported. We had the opportunity to study the case of a patient with CD and RA.

Keywords: coeliac disease, autoimmunity, rheumatoid arthritis


1. Introduction

The CD is a complex etiopathogenic enteropathy, susceptible to gluten, occurring in genetically predisposed individuals. RA is a chronic inflammatory disease characterized by an inflammation of the synovial tissue of multiple joints leading to pain and deformities. These two entities are autoimmune diseases. Their association is rare but possible. They differ in their HLA predispositions and specific predictive and diagnostic biomarkers.

2. Case Presentation

F.K. is a 46-year-old woman, thyroidectomized under levothyroxine, followed for CD, evolving for the last 8 years, retained in front of a non-glary bloody diarrhea with duodenal biopsy: histological aspect in favor of a type 3c CD according to the Marsh classification. The patient returned to remission after a gluten-free diet. Then she was admitted to the rheumatology department for management of inflammatory symmetric and deforming polyarthritis of the hands (Figure 1) and the feet, evolving for 22 years. Laboratory workup revealed GB = 10120/mm³, Hb = 10.3 g/dl; Platelets = 356000/mm³; C-reactive protein = 31 mg/L; ESR = 36 mm/H; Rheumatoid factor was positive. Radiographs of hands and wrists showed destruction of the carp, erosion and pinching of metacarpophalangeal joints and proximal interphalangeal (Figure 2). Radiographs of the foot showed erosion of the fifth head metatarsophalangeal joint in bilateral (Figure 3). According to the patient’s clinical profile and radio-biological results, she was diagnosed to be suffering from RA. A treatment based on prednisone 10 mg/day with methotrexate 15 mg/week was started, with a clear improvement, regression of inflammatory syndrome and improvement of quality of life.

Figure 1. Deforming chronic polyarthritis with a touch of piano, a boutonniere of the fifth finger bilaterally with thumb adductus.
Figure 2. Radiographs of hands and wrists showed destruction of the carp, erosion and pinching of metacarpophalangeal joints and proximal interphalangeal

Figure 3. Radiographs of the foot showed erosion of the fifth head metatarsophalangeal joint in bilateral

3. Discussion

The prevalence of celiac disease (CD) in RA is not significantly different from the reported prevalence of CD in the general population [1]. The association has been described in individual cases [2]. Gut-derived antigens may contribute in the pathogenesis of other autoimmune disorders including RA. Many of the susceptibility genetic loci for CD are shared with those for RA suggesting shared immunological and autoimmune mechanisms [3,4]. Despite having different target organs, HLA pre-dispositions, specific predictive and diagnostic biomarkers, both diseases share multiple aspects: gastrointestinal abnormalities in RA vs rheumatological ones on CD, epidemiology trends,
associated environmental inducers, autoantibodies and genes, dysbiosis and breached intestinal permeability [5]. Kadioglu and Sheldon suggested in their study that aberrant lymphocytes from the gut mucosal-associated lymphoid tissue of RA patients may migrate pathologically to the synovial fluids [4]. RA and CD have a strong association with class II HLAs: individuals carrying HLA-DQ2.5 and/or HLA-DQ8 alleles have an increased risk of developing CD, whereas those carrying HLA-DR shared epitope alleles exhibit a higher risk of developing RA [6]. There are also studies reporting improvement of symptoms of RA with dietary modifications, suggesting the role of gut-derived antigens as a driver of the inflammatory process in the joints [7,8]. There seems to be an association between arthritis and CD with a prevalence of 26% in Italian coeliac patients [9] although none of the patients with arthritis had rheumatoid factor. Exploration of the intestinal mucosal events initiating and maintaining articular and extra-articular etiological pathways in RA may lead to intestinal-targeted mechanism-specific strategies for prevention, early diagnosis or new therapies in RA.

4. Conclusion

In this unique case, we have been able to shed light on the association between RA and CD.

References


