Celiac disease and skin: Psoriasis association

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Received: 2007-01-20 Accepted: 2007-03-08

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TO THE EDITOR

We read with great interest the recent review by Rodrigo et al[4] on the celiac disease (CD). The author analyzed all aspects of CD. Contrary to common belief, this disorder is a systemic disease, rather than a pure digestive alteration. In fact, CD is associated with several diseases: skin manifestations, endocrine disorders, iron-deficiency anemia, osteoporosis, hypertransaminasemia, neurologic disorders and cancer. In the description of skin manifestations, the author reported the association between CD and dermatitis herpetiformis (DH). This condition affects about 15%-25% of patients with CD[3]. Antibodies for CD have been detected in patients with DH[4]. As CD and DH may be vastly different, they both share a unique intestinal sensitivity to gluten. The rash of DH is thought to be an external marker of the underlying intestinal sensitivity that is likely to be the result of molecular mimicry between the auto-antigen tissue transglutaminase resident in the gut and the skin derived epidermal transglutaminase[5]. However, our study group[6] has reported all CD-associated skin manifestations described in the literature, and in particular psoriasis.

Psoriasis is a chronic, relapsing dermatosis characterised by scaling, erythema, and less commonly pustulation[6]. It has been demonstrated that psoriasis is an immunological disease with hyperproliferation of T-cell mediated keratocytes. Immune mechanisms play an important role in the pathogenesis of this disease. In particular, an over-expression of T helper cell type 1 (Th1) cytokines and a relative under-expression of Th2 cytokines have been shown in psoriatic patients[7]. Recent studies showed an association between CD and psoriasis and an improvement of skin lesions after 3-6 mo of gluten free diet (GFD), without other pharmacological approaches[8]. The authors evaluated the effect of GFD in 33 antigliadin antibody (AGA) positive patients and six AGA negative patients with psoriasis in an open study. Of the 33 AGA-positive patients, two had IgA anti-endomysial antibodies (EMA), and at the duodenal biopsy 15 showed an increased number of lymphocytes in the epithelium, but in some patients, this increase was only slight. GFD was started for 3 months. Thirty of 33 patients strictly complied with GFD, have showed a significant decrease of psoriatic lesions. This included a significant decrease in the 16 AGA positive patients with normal histology in duodenal biopsy. The AGA negative patients did not improve. There was also a significant decrease in serum of eosinophil cation protein in patients with elevated AGA. In conclusion, the positive effects of GFD were observed not only in patients with an increased number of lymphocytes in the duodenal epithelium, but also in patients with normal epithelium. We reported severe psoriasis in a CD patient, not responding to specific therapies for psoriasis and in whom the regression of skin lesions after GFD was very rapid[6]. The association between psoriasis and CD was subsequently confirmed by Ojetti et al[8]. The authors evaluated the prevalence of CD in patients affected by psoriasis, and found a high frequency of CD (4.34%) in psoriatic patients.

At present, the mechanisms implicated in this association, and the effect of GFD on psoriatic skin lesions are not known. There are some hypotheses[8]: (1) Abnormal small intestinal permeability could be a triggering factor between CD and psoriasis; (2) T cells play an important role in the pathogenesis of both psoriasis and CD. In CD patients, gliadin induces a sensitisation of T cells and this may play a role in the pathogenesis of psoriatic skin lesions; (3) Psoriatic lesions in CD patients could be related to vitamin D deficiency, which is present in both CD and psoriasis. In a recent study, the prevalence of malabsorption in 55 psoriatic patients was evaluated[9]. The authors found that malabsorption was more prevalent among psoriatic patients than among controls and suppose that celiac disease and other diseases associated with psoriasis, such as bacterial overgrowth, parasitic infestations and eosinophilic gastroenteritis, could be the causes of malabsorption in these patients.

In conclusion, CD is an enteropathy associated with various extra-intestinal manifestations, including several skin diseases. DH represents the cutaneous manifestation of celiac disease. However, other skin manifestations of CD have been reported in literature, particularly the psoriasis. At present, the data are not homogeneous and
most of the evidences on the association between CD and skin disorders are based on “case-reports”, making it different to draw a definitive conclusion on this topic. Controlled studies are consequently needed to verify the real involvement of the skin district in CD. Nevertheless, despite these limitations, the investigations in the possible presence of CD in some dermatological patients seems necessary.

REFERENCES


S- Editor Zhu LH  L- Editor Ma JY  E- Editor Liu Y