The first published data concerning a possible relationship between psoriasis and pathologies of the gastrointestinal tract appeared as long as 30 years ago (1, 2), and subsequently there have been numerous reports on psoriasis and coeliac disease (3–5). These reports have included investigations into the presence of antibodies specific to coeliac disease in patients with psoriasis compared with controls. The findings demonstrated, in some cases, no correlation (6–8), but in others the presence of high titre in the IgA antibodies anti-gliadin, anti-reticulin, anti-endomysium and tissue anti-transglutaminase (9, 10). Furthermore, the presence of these antibodies and their increase was linked to a greater severity in the skin pathology (4). There have also been reports of patients with extensive psoriasis lesions and positivity for anti-gliadin, whose skin lesions regressed significantly after following a gluten-free diet for 3–6 months only to reappear on stopping the diet (11). In particular, it was also found that using a gluten-free diet as sole therapy in patients with psoriasis, who were positive for anti-gliadin and with high transglutaminase titres, improvement in skin lesions occurred along with a reduction in transglutaminase titre (12).

As regards the pathogenetic mechanism behind this possible association, little is known, although there have been diverse hypotheses (3, 13): (i) the anomalous intestinal permeability in people with coeliac disease could represent a “trigger factor” for psoriasis; (ii) in coeliac disease gliadin induces a sensibilization of T cells, and their activation could be associated with the pathogenesis of the cutaneous lesions; (iii) psoriasis lesions in people with coeliac disease could be related to vitamin D deficiency, a characteristic of both diseases.

Because in Sardinia there is a high prevalence of subclinical coeliac disease (14), we performed a case-control study to evaluate a possible association between this disease and psoriasis.

MATERIALS AND METHODS

We used ImmunoCAP 100 Celikey® IgA Fluoroenzyme immunoassay to value anti-transglutaminase antibody on 100 patients with psoriasis attending the PSOCARE Outpatients Surgery of the Dermatology Clinic, University of Sassari between October and December 2008, and on 100 controls unaffected by either dermatological or gastrointestinal pathologies from general surgery outpatients in the same period. On the hypothesis of observing at least one individual with coeliac disease among the controls, the power of our study to detect a significant association between coeliac disease and psoriasis was 48%, 67%, and 80% if the number of coeliac individuals among the cases was ≥ 6, 8, or 10, respectively.

The psoriasis group comprised 44 females and 56 males between the ages of 5 and 87 years. The control group comprised 56 females and 44 males in the same age range. All the subjects were born and resident in Sardinia. All gave their informed consent and answered to questions about familiality of coeliac disease.

Ninety-one of the patients with psoriasis presented solely skin manifestations of the disease, while the remaining 9 had psoriatic arthropathy.

RESULTS

Anti-transglutaminase antibody testing in both groups revealed 2 psoriasis patients with over 7.1 U/ml, i.e. above threshold level. Further testing on these patients, including an intestinal biopsy, supported the diagnosis of coeliac disease. However, the association between coeliac disease and psoriasis was not statistically significant (p-value = 0.49).

The remaining subjects in the psoriasis group and in the controls had values lower than the threshold value, although the group with psoriasis presented slightly higher values than the controls (Fig. 1), with 3 patients in particular in the interval 1.1–2 and 4 between 2.1–3 U/ml.

Very interestingly, marginal significance was observed when assessing the association between first-degree familiality for coeliac disease and psoriasis (p-value = 0.059).
DISCUSSION

As previous studies have shown, coeliac disease is a high-incidence pathology in the Sardinian population. The prevalence of 1.06% observed in Sardinian children (14) is in fact much higher than that of the rest of Italy (15, 16). Given these data, we would have expected to observe, on average, one coeliac individual in 100 controls (95% confidence interval 0–2). This expectation is compatible with what we have observed, given the number of individuals in our study. Therefore, we had expected a greater number of our patients with psoriasis also to have coeliac disease. In fact, among the psoriasis sufferers, only two patients presented anti-transglutaminase antibody values above the threshold level and had a confirmed diagnosis of coeliac disease. Another four patients referred first-degree familiality for coeliac disease, whereas the control group had no coeliac sufferers and presented no familiality for the disease.

In conclusion, due to lack of power, our study can neither support, nor reject, the hypothesis of a significant association between psoriasis and coeliac disease. However, the suggestive evidence of an association between coeliac disease familiality and psoriasis needs to be tested in larger studies.

REFERENCES