Letters to the Editor

Coeliac disease in 108 patients with juvenile idiopathic arthritis: a 13-year follow-up study

Sirs,

Coeliac disease (CD) is an enteropathy caused by sensitivity to gluten that affects about 1% of the European population (1). CD is characterized by autoimmune response, with the presence of specific auto-antibodies in serum. Many studies both in adults (3-5) and, more recently, in children (6, 7), have consistently reported an association between CD and various autoimmune diseases, including juvenile idiopathic arthritis (JIA). However, the risk of developing CD after JIA diagnosis has not been prospectively investigated yet. In this study the incidence of CD was evaluated in a cohort of 108 consecutive children (37 males, 71 females) diagnosed with JIA at our institution between 1992 and 2004. JIA was defined according to the Classification Taskforce of the Pediatric Standing Committee of the International League of Associations for Rheumatology (8). Patients or their guardians were informed about this study and consent was obtained from all children and/or their parents. The procedures we followed were in accordance with our institutional ethical standards and with the Helsinki Declaration.

At JIA diagnosis and thereafter every year, children were immunologically evaluated for anti-endomysium antibodies (EMA) and anti-tissue transglutaminase antibodies (tTGA) (from April 1998). In the case of EMA and/or tTGA positivity, another test was repeated after 4 months, and in the case of 2 consecutive positive tests a jejunal biopsy was performed to confirm CD diagnosis.

One girl with CD diagnosis made before JIA onset was considered as a “prevalent case” and was not tested for EMA and tTGA. The median age at JIA diagnosis of the remaining 107 patients was 5.7 years (range 0-15 years). JIA was defined as oligoarticular in 70 patients (65.4%), polyarticular in 24 (22.4%), and systemic in the remaining 13 (12.2%). At JIA diagnosis, all tested patients resulted negative for EMA, while only 1 patient was found positive for tTGA (29 AU/ml); none was found IgA deficient. Patients were followed-up for a median of 5.5 years (range 50 days - 12.3 years) (Fig. 1). The patient who was tTGA positive at diagnosis turned negative (tTGA < 5 AU/ml) at the following tests. However, two cases turned positive at the immunological tests and jejunal biopsy, 3.5 and 4.6 years after JIA diagnosis, respectively. Both were asymptomatic for intestinal, hematological and other clinical symptoms of CD, and were prescribed a gluten free diet, followed by an improvement of JIA symptoms.

In our study, the prevalence of CD at JIA diagnosis was 0.9% and 2.8% at the end of the 13 years respectively. Moreover, Stagi et al. (7), in a cross sectional Italian study on 151 JIA children, also reported a higher prevalence of CD in patients as compared to controls.

The results of our study show that systematic screening might help in early CD diagnosis in JIA patients. However, extensive screening programs may have elevated costs. Another approach might be to screen only a subset of the population defined on the basis of particular risk genotypes as HLA-DQ2/DQ8 (9, 10). Further studies are needed to identify the best and least expensive method to follow-up this population at risk.

Acknowledgements

We would like to thank Anna Capurro for her English language revision.

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Competing interests: none declared.

References