Oral Psoriasis: Report on a Case without Epidermal Involvement

Sir,

This report concerns a boy who became our patient when he was 6 years old. He was under our supervision until he reached the age of 18. He presented with intensely erythematous areas, covering most of the gingiva in both the lower and upper jaw. The lesions involved most of the gingiva, including both free and attached parts, except for the tips of the gingival papillae (Fig. 1). The clinical picture changed very little throughout the years. Neither the tongue nor any other part of the oral mucosa was affected. Repeated bacterial and fungal cultures were negative. Hygienic rinses with chlorhexidine and topical treatments with steroids had no beneficial effect. An initial biopsy from the affected area showed a classical psoriasis picture. A second biopsy made at the age of 18 displayed the same pattern (Fig. 2). The last tissue specimen was also examined by direct immunofluorescence on frozen sections, with negative results. In that way vesiculo-bullous diseases were excluded. No *Candida* pseudohyphae were visualized. HLA typing as well as clinical examination of the skin of the index case and his relatives were performed. Subjects with cutaneous psoriasis were marked on the pedigree (Fig. 3). No oral or nail involvements were seen in any of the other family members. As a teenager, the patient developed hyperkeratotic nails, with no laboratory signs of dermatophytes.

Fig. 1. The mucosal lesion present in the 6-year-old boy.

This case demonstrates the difficulties involved in making a diagnosis of psoriasis based on mere oral mucosal lesions without any cutaneous or nail stigmata (1–8). The micromorphological features of the mucosa, showing signs of classical psoriasis as it would have appeared if present on the skin, substantiated our diagnosis (Fig. 2). However, the boy was

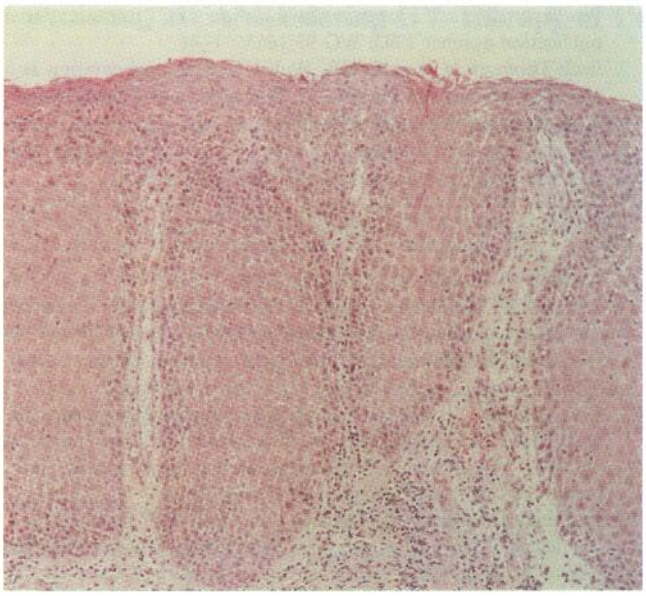

Fig. 2. The gingival biopsy, from the age of 18, showed the same picture as the one from his 6th year. The epithelium was elongated, with thickened rete ridges, intercellular oedema and infiltration of both lymhocytic and neutrophilic granulocytes, the latter accumulating in the lower part. Focally, the stratum granulosum was thin, with a corresponding overlying thin horny layer. Apart from this, stratum granulosum was missing, with parakeratosis of the horny layer. The connective tissue was heavily infiltrated by mononuclear cells. Magnification × 190.

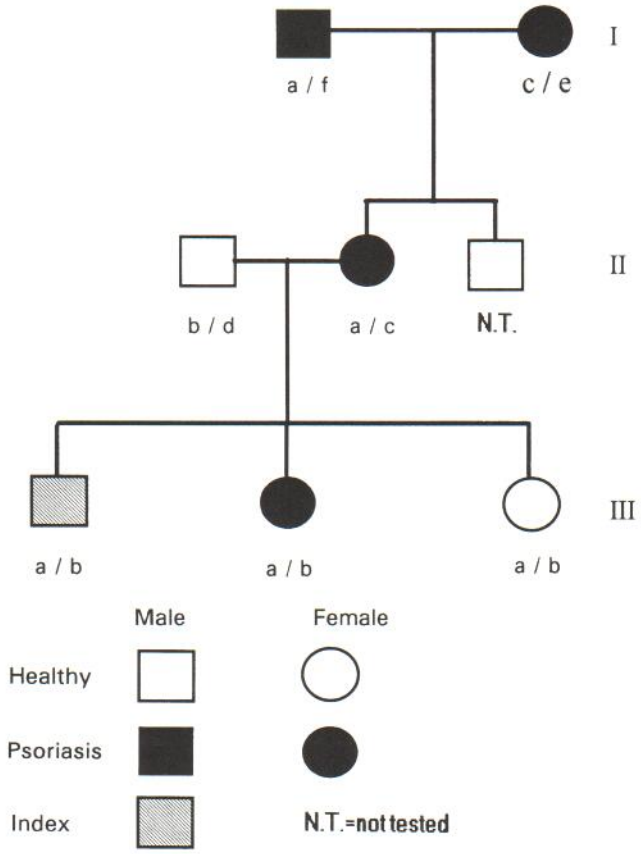

Fig. 3. Pedigree showing the HLA haplotypes of the family members. The following haplotypes are shown: a=A19, B13, CW6; b=A2, B12(44), Cw5; c=A9(24), B27, CW2; e=A2, B7, CW7; f=A11, B27, Cw2. One A-locus allele of III:2 could not be detected. *NT*=not tested (=HLA typed).

free from cutaneous manifestations during the 12-year observation period. In addition, the disease is frequent in the family.

Over the past two decades, numerous studies have been conducted concerning the association of psoriasis with HLA antigens (9). A close association with HLA B13, Bw57 (which is a subtype of B17) and a CW6 association have been shown. Moreover, a subdivision of psoriasis into types 1 and 2, based on the age of onset, has been possible. Thus, HLA CW6 and/or B13 (as well as Bw57) antigens occur at a higher frequency in patients with early onset of disease. The aforementioned genetic markers for psoriasis are found in this particular family (Fig. 3) and in the index case, further validating the diagnosis of gingival psoriasis.

In 1996, a case was reported by Robinson et al. with an oral lesion fulfilling the histological criteria for psoriasis but also with classical lesions of the skin (10). In their case it was easier to make the diagnosis due the presence of skin manifestations. In our case the oral lesions together with the family history, long observation period, presence of specific HLA antigens and resistance to topical therapies make the diagnosis of psoriasis most possible, despite the fact that there were no skin lesions typical of the disease.

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Accepted February 3, 1997.

B. Rozell[1], A-C. Grevér[2] and J. A. Marcusson[3]
[1]Department of Oral Pathology, School of Dentistry, Karolinska Institute, [2]Department of Paediatric Dentistry, Eastman Dental Clinic, Stockholm, and [3]Department of Dermatology, Huddinge Hospital, SE-141 86 Huddinge, Sweden.