Infantile psoriasis

A short clinical study

E. Kassay, A. Sáringer, É. Török and Z. Szalai

SUMMARY

Psoriasis is a lifelong disease. The signs are exacerbating and waning without any apparent reason. Infants with psoriasis have high incidence of the disease at 5-13 years of age, but it is also possible that they will be free of symptoms during their entire life. In the treatment a conservative approach is advocated, there are few cases where retinoids are needed. It is important for the parents to be familiar with the nature and management of the disease. We hope that gene therapy will play a positive role in the treatment in near future.

KEY WORDS

psoriasis, infantile, napkin psoriasis, erythrodermic, pustular

Introduction

Psoriasis is a chronic disease with accelerated epidermal turnover and epidermal hyperplasia. It has a prevalence of 1-3 % in the general population, but represents 4.1% of all dermatoses encountered in children younger than 16 years of age (1,2). In about 2-6 % of all psoriasis cases, the first signs appear before the age of two years (3.4).

The pathogenesis of the disease is multifactorial: multiple genetic and environmental factors play a role in infantile psoriasis. The data on the HLA antigens predisposing to psoriasis are somewhat conflicting: HLA B-13, B-17, B-27, Bw 57, Cw 2, Cw6 and DR-7 (5,6,7). The triggering factor in infantile psoriasis is usually an infection. Associated diarrhoea, along with the urine in the napkin dermatitis may also contribute to the development of the disease. Infections also play a major role in exacerbating the disease (7). The importance of family history in psoriasis has been stressed years ago (1,7). Interestingly, 60 % of our patients as observed last year had a previous infection.

Children suffering from psoriasis have higher serum total IgE than the normal population (5). Atopic dermatitis and psoriasis often occur together, but the reports on their concordance are contradictory (8, 9).

Clinical manifestations

Napkin psoriasis is the most frequent form of psoriasis in infants. Last year 80% of our patients suffering

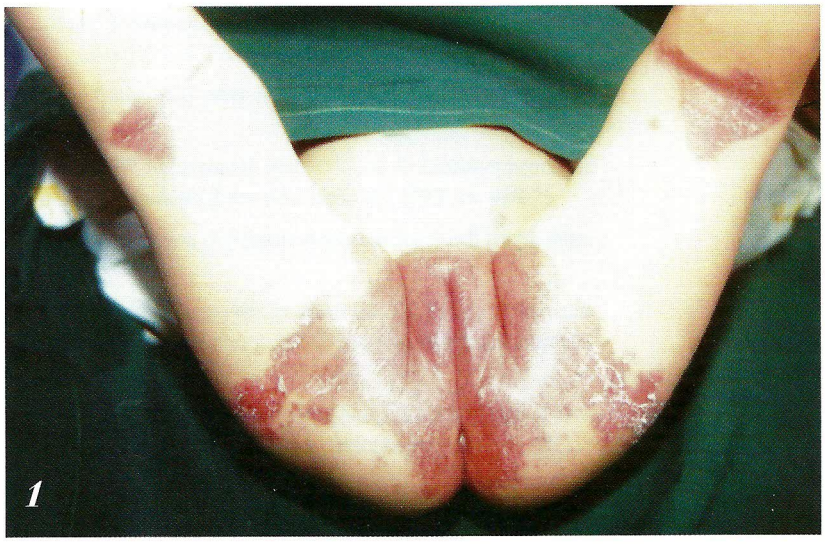


Figure 1. Napkin psoriasis, beginning lesions in the anal and gluteal area in an infant.

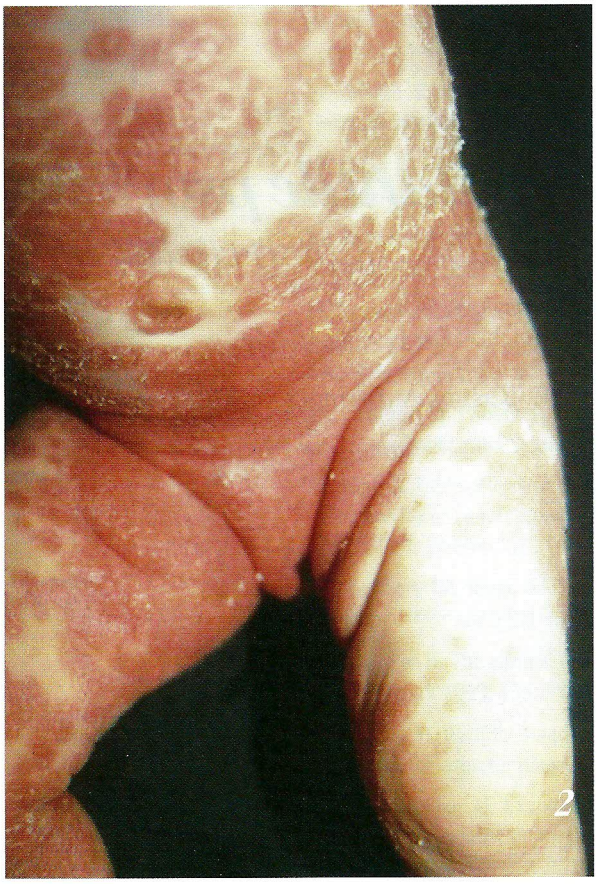


Figure 2. Napkin psoriasis, developed lesions in an infant.

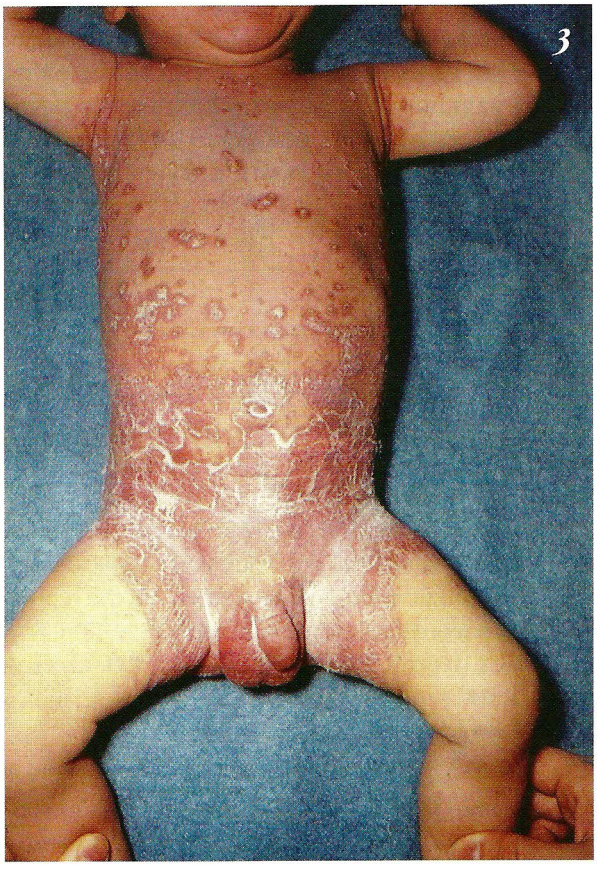
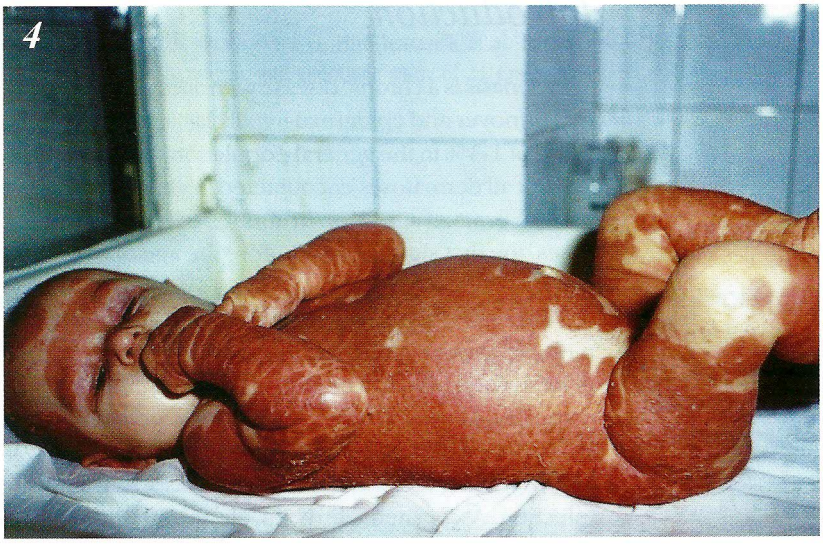


Figure 3. Napkin psoriasis spreading to the body.

Figure 4. Erythrodermic psoriasis in an infant



from infantile psoriasis presented the first signs in the gluteal region (Figure 1,2). The first lesions appeared in the perianal and perigenital area as sharply demarcated red, shiny papules. In warm and humid environment (e.g. areas covered by napkins) the papules were thin and not scaly. Later they spread to the whole region, and a confluent, erythematous, not scaling area of the skin was involved (Figure 3). Some weeks later secondary lesions, scaly white plaques and papules appeared all over the body. Lesions tended to be symmetrical. Other commonly affected areas included the scalp as cradle cap, the face, the flexural regions (neck folds, antecubital and popliteal creases).

Histopathological examination usually reveals the diagnosis of psoriasis. Only a long-term follow up can however confirm whether the patient has true psoriasis or a psoriasiform dermatitis. Patients with infantile psoriasis have a high incidence of psoriasis at 5-13 years of age (10).

Severe erythrodermic/generalized pustular type of psoriasis is rare in infants (Figure 4). Pustular psoriasis may follow seborrhoeic dermatitis. Psoriatic erythroderma develops from pustular psoriasis or from generalized napkin psoriasis or it can be the initial sign of the disease. Complications are hypalbuminemia, sideropenia, and ectropium. Erythroderma may persist for years (2). One of our patients is 22 years old and she has had erythroderma since she was 1.5 year old. Her familiar history is negative. She had dry skin from birth and she had seborrhoeic eczema several times. The first pustular psoriasis attack occurred at one and a half-year of age, following an upper respiratory tract infection. Later on the pustular psoriasis evolved into psoriatic erythroderma. This condition has persisted for years: occasionally her condition improved, but the girl has never been free of symptoms. During this time she has received different treatments. Systemic prednisolon, methotrexat, etretinate, PUVA and plasmapheresis were applied. As these therapies failed isotretinon was introduced at a dose of 20 mg/day. She has been taking it for two years and she is in a relative good and stable condition. As we tried to discontinue the isotretinoin, the ectropium worsened. Sunlight was beneficial, while infections worsened the course of the disease.

In a few instances the first lesions appeared on other regions, as recurrent intertrigo, or as a single plaque resembling nummular eczema, but this was very rare. The diagnosis was difficult, because the single lesions may persist for a long time.

Only a few cases of congenital psoriasis have been published.

Differential diagnosis.

Napkin psoriasis, cradle cap and widespread lesions must be differentiated from candida infection, seborrhoeic or nummular eczema. Psoriatic erythroderma might resemble Leiner's disease or non-bullosus ichtyosiform erythroderma (2).

Therapy

Topical therapy. Moisturizers and emollients are helpful in preventing attacks. Napkin psoriasis and secondary widespread lesions usually responded well to the steroid creams or /and antifungals. The lesions disappeared in 2-3 months. Mild keratolytics are the first choice for the cradle cap. Coal tar was well tolerated.

Systemic therapy. The actual infection must be treated. In severe erythrodermic/pustular psoriasis retinoids (0.01-0.1 mg/kg) are recommended during a few weeks but sometimes they need to be administrated for years. Side effects are rare.

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AUTHORS' ADDRESSES

Erzsébet Kassay, MD, Heim Pál Children's Hospital, Department of Dermatology, Ûllõi ût 86, 1089 Budapest, Hungary
Attila Saringer, MD, Heim Pál Children's Hospital, Department of Internal Medicine, same address
Éva Török, MD, PhD, professor of dermatology, same address
Zsuzsanna Szalai, MD,PhD, same address