IgA Deficiency and Psoriasis: Relevance of IgA in the Pathogenesis of Psoriasis

Abstract

A psoriatic patient with absolute deficiency of IgA is reported. The manifestations of psoriasis appeared at the age of 6 months and proved to be resistant to various treatments. The present case report and the data available in the literature on IgA and psoriasis all converge on the hypothesis that IgA is a systemic factor which belongs to the ‘off switches’ of the psoriatic process.

Inflammation, epidermal proliferation and abnormal differentiation of the epidermis are the well-known abnormalities in the pathogenesis of psoriasis. The immunology of psoriasis has been reviewed recently [1]. One part of the immunology in psoriasis regards the ‘noncellular components’. An important observation, highlighting the relevance of non-cellular systemic factors in psoriasis is the fact that serum from patients with stable psoriasis repressed the Köbner reaction, whereas serum derived from patients with unstable relapsing psoriasis failed to suppress this phenomenon [2]. This observation may indicate that serum factors in chronic stable psoriasis have an antipsoriatic potential, protecting the patient against progression.

Functionally, serum factors may modulate chemotaxis of monocytes and neutrophils and may interfere with angiogenesis and proliferation of fibroblasts [3–6]. Non-cellular systemic factors which might be involved in the immunopathogenesis of psoriasis are numerous. It has been suggested that IgA and IgA immune complexes are involved in the pathogenesis of psoriasis [7–9].

Recently, we had the opportunity to treat a patient with a complete deficiency of IgA who had psoriasis of the chronic plaque type with an unusual course: initiation of psoriasis was at the age of 6 months, and the course was resistant to various treatments.
biopsy taken from the left elbow revealed the classical picture of an
active lesion with pronounced acanthosis, parakeratosis, a mixed
inflammatory infiltrate and prominent micropustules of Kogoj and
microabscesses of Munro.

The patient was admitted to the inpatient department for treatment
with dithranol paste. However, due to irritation, already at low
concentrations, the treatment had to be changed to a short-contact
schedule with dithranol creams, tar preparations, corticosteroids,
hydrocolloid dressing and UVB phototherapy. Due to insufficient
improvement, additional treatment with acitretin (35–50 mg/day)
was given. Following a 10-week admission, the intensified combined
treatments had resulted in a considerable improvement, although total
clearing was not reached.

**Discussion**

The accumulation of T lymphocytes and polymorphonuclear
leukocytes and the abnormal functioning of these cells are thought to be relevant and crucial aspects of the patho-
genesis of psoriasis [1, 10]. Both cell types have been
reported to possess receptors for the Fc portion of the IgA
molecule [11–14]. With respect to function of the poly-
morphonuclear leukocyte, it has been shown that IgA para-
proteins and polymeric forms of IgA inhibit the chemotaxis
and the bactericidal activity of these cells [15–17]. Schrö-
der et al. [18] reported on the neutrophil chemotaxis inhib-
iting activity of sera from patients with psoriasis and other
neutrophilic dermatoses. The inhibition of chemotaxis
proved to correlate with increases in serum IgA. Patients
with chronic stationary psoriasis showed high IgA levels
and reduced neutrophil chemotactic activity whereas those
patients with relapsing psoriasis had normal IgA levels and
increased neutrophil chemotactic activity [18]. This obser-
vation lends support for the supposition that an increase in
serum IgA might be an antipsoriatic mechanism, stabilizing
the psoriatic process. However, although neutrophil chem-
otaxis inhibiting activity of sera from patients with psoriasis
has been shown to depend upon IgA polymeric forms in
vitro, it is not clear whether this is important in vivo [19].

Different groups have reported that serum IgA levels are
increased in psoriasis [8, 20–24]. Circulating IgA immune
complexes have also been reported to be increased in psoriasis
[9, 25]. Guilhou et al. [8] reported that the increase in
IgA is specific for psoriasis as no patient with eczema had
such an increase. By these authors, the increase in IgA-con-
taining immune complexes was observed in 67% of a group
of psoriatics whereas a similar increase was found in only 1
out of 25 patients with hyperkeratotic disorders. Fraser et
al. [20] reported increased levels of IgA in patients with
dermatitis herpetiformis, acne vulgaris, rosacea and discoid
lupus erythematosus. A consistent observation by various
groups is the correlation between increased serum levels of
IgA and the extent of the skin manifestations and severity of
arthritis [18, 25]. However, it has been reported that psor-
iasi with arthropathy may coexist with a selective IgA
deficiency [26]. Two groups have reported on decreased
IgA levels in psoriatic patients [27, 28]. The correlation
between disease activity and IgA levels [18, 25] suggests
that IgA is of significance in the pathogenesis of psoriasis.
It is attractive to hypothesize that IgA is an ‘antipsoriatic
factor’. Indeed, relatively high levels of IgA have been
measured in chronic long-standing plaque psoriasis and in
patients with extensive lesions, in contrast to the low levels
of IgA in relapsing and guttate-type psoriasis [18]. Patients
with decreased or undetectable IgA levels and psoriasis
[26–28] may have an unfavorable prognosis according to
this hypothesis.

The present case report is in line with a protective role of
IgA with respect to the relapse of psoriasis. The unusual
course – onset at the age of 6 months and the unstable fea-
tures, characterized by extreme dithranol sensitivity and
therapy resistance – in a patient with IgA deficiency may
suggest that IgA indeed belongs to the ‘off switches’ of the
psoriatic process. However, it is remotely possible that the
episodes of recurrent infections might have been respon-
sible for the onset of psoriasis in early childhood.

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