

Crusted Scabies in Acquired Selective IgA Deficiency

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Crusted scabies, an unusual clinical variant of human scabies mite infestation, is usually reported in cases of gross debility, mental deficiency, or immunosuppression. We report here the occurrence of crusted scabies in a 40-year-old man with acquired selective IgA deficiency suspected to be caused by long-term medication with phenytoin for epilepsy. Key words: Mite infestation; Immunosuppression; Epilepsy.

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Crusted scabies is caused by overwhelming infestation of *Sarcoptes scabiei* (1–3). Several factors are believed to be of importance in its development – mental deficiency, gross debility and lack of skin sensation are traditionally implicated (1). Other authors speculate on the role of vitamin A (4). In addition, the involvement of immune suppression in some diseases (5, 6) and iatrogenic mechanisms such as immunosuppressant (1, 7) or steroid therapy (8) have been frequently discussed.

Hancock & Ward (9) reported a correlation between scabies infestation and low serum levels of IgA, the predominant antibody component of external secretions. They postulated that reduced IgA

secretion due to low serum IgA levels may predispose to scabies infestation.

We report a case of crusted scabies occurring in a patient with acquired selective IgA deficiency suspected to be caused by epilepsy treatment with phenytoin.

CASE REPORT

A 40-year-old man with epilepsy was referred to our clinic, suffering from widespread, slightly pruritic hyperkeratotic or crusted eruptions of 2 months' duration. Ten months previously he had been admitted to another hospital, suffering from cholestatic liver dysfunction. Though the course of liver dysfunction was good, the patient developed pruritic eruptions interdigitally and which extended to the trunk. Thickened scaly lesions then developed.

Clinical features showed numerous brownish crusted papules on the trunk and extremities. The palms and soles were grossly hyperkeratotic with large scales. A marked feature was the hyperkeratotic or crusted plaques, particularly on the finger webs, back and buttocks (Fig. 1). Scrapings from hyperkeratotic lesions disclosed numerous mites. A punch biopsy specimen obtained from the buttock showed portions of mite body in the stratum corneum and a mild, non-specific inflammatory cell infiltration. The patient had had epilepsy, for which he received periodic psychiatric treatment. For 10 months, he had been medicated with phenytoin (600 mg/day). His intelligence was deemed slightly subnormal.

Laboratory data included the following (normal range in parentheses). Serum immunoglobulins: IgG, 1020 mg/dl (1000–1700 mg/dl); IgA, 1.0 mg/dl (90–330 mg/dl); IgM, 79

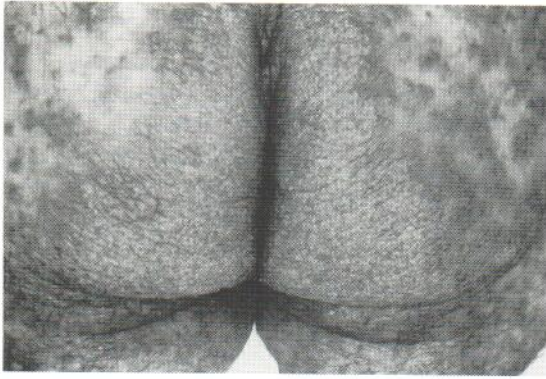


Fig. 1. Hyperkeratotic lesion on buttocks.

mg/dl (50–230 mg/dl); IgE, < 100 IU/ml (< 400 IU/ml). Salivary IgA was below the detectable level. Lymphocyte subpopulation: T cells, 81% (76–86%); B cells, 9% (8–16%); OKT3, 61.2% (57.9–76.3%); OKT4, 36.5% (32.8–49.4%); OKT8, 39.2% (22.6–33.0%); OKT4/OKT8, 0.93 (0.9–1.8). Sensitization to topical application of dinitrochlorobenzene was demonstrated. The PPD skin test proved positive. Blood vitamin A concentration was 65 µg/dl (49.5–64.5 µg/dl). Anti-nuclear and anti-DNA antibodies were negative. Total bilirubin was 3.0 mg/dl (0.5–1.4 mg/dl). Other laboratory data including liver function were within normal limits.

The patient had not received immunosuppressant or steroid therapy for the previous 6 months. His IgA level, when tested 10 months earlier (before starting on phenytoin treatment), had been normal (102 mg/dl).

Treatment with 5% precipitated sulfur in petrolatum for 8 weeks cleared the scaly lesions and no mites were found in the skin, but the low IgA concentration was not changed (1.5 mg/dl). After treatment of the scabies, medication with phenytoin was stopped. Three months after the cessation of phenytoin medication, IgA levels had improved (55 mg/dl).

DISCUSSION

Our patient showed low levels of both serum and salivary IgA, but no evidence of autoimmunity. However, other immunoglobulins and cell-mediated immune response were within normal limits. As IgA levels had been normal on admission 10 months previously and there was no history of immunoglobulin abnormality, his case was considered to be due to acquired selective IgA deficiency (10). The mechanism of acquired IgA deficiency remains obscure. Several authors note the relation of IgA deficiency to autoimmune disease (10–12).

Falk reported that low concentrations of serum IgA in scabies increased to normal levels after treatment (13). Those results suggest that IgA deficiency could even be a secondary phenomenon deriving from scabies.

On the other hand, correlation of IgA deficiency and use of antiepileptic agents such as phenytoin has been described (14–16). In our patient, the low IgA level persisted throughout the treatment of scabies, and cessation of the medication improved IgA levels. Though the possibility that the low IgA concentration was a secondary phenomenon from the scabies cannot be completely ruled out, it is strongly suspected that the drug was a factor. Attention should be given not only to the patient's condition and mental state, but also to drugs with immunomodulating potential.

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