In this report we clinically characterise a considerable number of cases of an unusual episodic self-limited eczematous eruption in skin folds. We suggest the term “flexural pellagroid dermatitis”, reflecting its similarity to dermatitis observed in pellagra. Furthermore, we discuss the role of sodium lauryl sulphate (SLS) as its possible aetiology.

METHODS

The study was conducted in the outpatient dermatology clinic of Rabin Medical Center, a tertiary, university-affiliated facility, after being approved by the local Helsinki committee. The medical files of patients who presented with a flexural dermatitis in 1989–2011 were retrospectively analysed for common epidemiologic and clinical features. Relevant data from medical history and physical examination, as well as the results of patch tests, biopsies, and microbiological tests were collected.

RESULTS

The study group consisted of 47 patients, 39 females and 8 males. Twenty-six patients were aged 17–40 years, 15 were aged 41–60 years, and 6 were aged 61–80 years; most of the patients (n = 38, 80.8%) were less than 50 years old. Eight patients (17%) had a personal (n = 7) or family (n = 1) history of atopic disease. Most of the patients presented to our clinic in the early 1990s, with similar cases only rarely encountered thereafter.

Patients complained of acute, self-limited episodes of a skin-fold eruption that occurred mostly in the summer months and were sometimes related to excessive sweating. The episodes recurred over a span of up to 10 years (Table SI1), with each episode lasting 2–4 weeks. Mean duration of the clinical course was 2.92 years. To identify the cause of the eruption in our series, patients were questioned about their personal hygiene habits, and reported using the particular brands of the soapless cleansing bars of the local manufacturer (containing: SLS 40–44%, Sorbitol, Glycerin, Cetostearyl alcohol, Stearic acid, Starch). Physical examination revealed an eruption with a flexural distribution and multiple skin-fold involvement (Fig. 1). The most frequent sites affected were the antecubital fossae, axillae, intermammary and inframammary areas, and neck (Table SII). The eruption was resistant to topical corticosteroid therapy.

Two successive phases of the eruption were observed. The first inflammatory phase was characterised by a well-demarcated erythema that caused a stinging and burning sensation. After several days, the erythema evolved into flat, scaly, rust-brown plaques, with cigarette-paper-like surface changes and less demarcated borders, resembling very much the dermatitis observed in pellagra. It was usually followed by desquamation leading to clearing of the eruption. The eruption did not recur after the use of the soapless cleansing bars was discontinued.

No evidence of infection was found on Wood’s lamp examination, bacterial culture, potassium hydroxide direct microscopy or fungal culture, performed in the early cases.

Four-millimeter punch biopsies of the affected skin during the pellagroid phase were performed in 6 patients. Microscopic examination revealed acanthosis and compact hyperkeratosis, with alternating vertical and horizontal ortho- and parakeratosis (“checkerboard pattern”) in all patients. Some of the hair follicles were filled with keratinous plugs. The granular layer was preserved and contained keratinocytes displaying

1http://www.medicaljournals.se/acta/content/?doi=10.2340/00015555-1862

Fig. 1. Flexural pellagroid dermatitis. Clinical presentation. Inflammatory erythemic phase (A). Pellagroid phase (B). Desquamation (C).
prominent granules. A mild perivascular infiltrate composed of lymphocytes was observed in the upper dermis (Fig. S1). Thirty-eight patients (80.8%) were patch-tested with the European standard series consisting of 23 allergens (Chemotechnique Diagnostics AB, Malmö, Sweden), using Finn Chambers on Scanpor tape (Hermal, Reinbeck, Germany). When clinically relevant, other patch test series (cosmetics, textile, dental) were applied as well. The patch-testing was performed according to the guidelines of the International Contact Dermatitis Research Group (1). Positive results were found in 14 of the 38 patients tested (36.8%). The most frequent positive readings were for potassium dichromate, fragrance mix, and 2-phenoxyethanol (Table SIII). The results were concluded as irrelevant to the skin eruption.

DISCUSSION

Intertriginous eruptions have a broad differential diagnosis. The most frequent causes are infections (such as dermatophytosis, candidiasis, erythrasma and periflexural exanthema), atopic dermatitis, either allergic or irritant (2). In our patients, infectious causes were ruled out on clinical grounds. Atopic dermatitis was excluded by the unique clinical manifestations (e.g. biphasic short lived flexural eruption with well demarcated borders, the same developmental stage of the lesions and spontaneous healing with prominent desquamation), the absence of itch, and the lack of increased prevalence of atopy in our cohort compared to general population (3).

The aforementioned clinical manifestations and the failure of topical steroid treatment pointed out against allergic contact dermatitis. Although we found a 36.8% rate of positive patch tests, the findings were interpreted clinically as irrelevant to the skin condition. Histologically, a unique type of dermatitis with similar histopathological features was observed in all biopsies. No necrotic keratinocytes were detected. The most prominent feature was compact hyperkeratosis, with alternating vertical and horizontal ortho- and parakeratosis. Similarly, parakeratosis was reported by Willis et al. (4) in patients with mild to moderate clinical reactions following 48 h patch test with 5% SLS, while necrotic keratinocytes were noticed only in the case of severe irritant response to SLS.

The single common denominator in our patients was their use of the particular brands of the soapless cleansing bars produced by an Israeli manufacturer at the time of appearance of the rash. When the patients discontinued use of these products, the eruption resolved with no recurrences. The majority of adverse skin reactions to personal care products are presumed to be caused by irritant substances contained in these products (5). Irritant ingredients such as benzalkonium chloride (6–8) could be potentially trapped in skin folds causing irritant dermatitis of flexural distribution.

Soapless cleansing bars contain numerous substances, including surfactants, particularly of an anionic type. Irritant potential of many anionic surfactants is well established, with SLS serving as a model irritant in experimentally induced contact dermatitis (9–11). The soapless cleansing bars used by our patients contained 40–44% SLS, suggesting high irritant potential of those products, but no benzalkonium chloride.

In recent years, SLS in most products has been largely replaced by less irritating surfactants, such as sodium laureth sulphate (SLES), and it is no longer considered a major irritant in daily life (12). Accordingly, the peak presentation of our patients in the early 1990s may be attributed to the introduction soon thereafter of new liquid shower cleansing products containing 11.9% SLES instead of SLS, produced by the same manufacturer. After the use of the soapless cleansing bars was discontinued, most patients were lost from the follow-up, because of complete resolution of their complaints.

The authors declare no conflict of interest.

REFERENCES