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GUIDELINES

Topical non-pharmacological treatment of eczema: an Italian consensus

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ABSTRACT

Eczematous diseases (contact dermatitis, atopic dermatitis, hand eczema) are among the most frequent findings in dermatological clinical practice. A large body of evidence exists on structural and functional skin barrier damage in eczematous diseases, and on the importance of interventions aimed to repair such damage. While there is substantial agreement on pharmacological treatment, more sparse data are available on role, indications and usefulness of topical non-pharmacological treatments, despite significant research and progress in the composition and technology of emollients, cleansers and barrier creams significantly changed and expanded the functional activities of these products. This often leads to inadequate prescription and/or use, which increase individual and social costs of the disease and make the products useless or, in some cases, even counterproductive. This consensus document, discussed and compiled in a series of meetings by a group of Italian dermatologists experienced in the field of eczematous diseases, summarizes epidemiology and clinical features of the nosological entities of the "eczema family", illustrates the chemical/biochemical structure of emollients, cleansers and barrier creams, and aims to help physicians to exploit the full potential of available products, by providing a detailed but practical guide on characteristics, indications and correct use of non-pharmacological treatments currently available for eczematous diseases.

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KEY WORDS: Eczema; Dermatitis, irritant; Dermatitis, allergic contact; Dermatitis, atopic; Hand; Emollients.

The "eczema family" includes some very common, non-infectious inflammatory diseases, namely contact dermatitis (CD), atopic dermatitis (AD) and hand eczema (HE). The acute phase of these diseases is mainly characterized by erythema, edema, vesicles, exudation and crusts, while chronic lesions show reactive epidermal changes such as lichenification and desquamation.[1-5] Alterations of the skin barrier function, acquired and/or constitutional, are always present.[2]

Multiple mechanisms may concur in the pathogenesis of eczema, and therefore mixed clinical pictures, with characteristics of different eczematous diseases, are frequent in real life conditions.[5] Particularly when chronic and/or localized to sensitive areas, these diseases have high individual and social costs, direct and indirect, may significantly impair the quality of life and, when hands are involved, negatively affect the ability to work and/or perform daily activities.[4, 6, 7]

Avoidance of irritants and/or contact allergens is obviously the best option to obtain complete and durable resolution of eczema in the case of CD and is helpful also in the case of AD, but it is often neither easy nor immediate

to achieve, and medical treatment is required to reduce inflammation, alleviate the suffering of patients and restore barrier function as much as possible.[8] While there is substantial agreement on pharmacological treatment, more sparse data are available on role, indications and usefulness of topical non-pharmacological treatments,[9] despite significant research and progress in the composition and technology of emollients, cleansers and barrier creams. The lack of consensus in this field too often prevents patients and physicians from fully exploiting the potential of available products, and in some cases leads to inadequate advice, resulting in a decrease of efficacy of pharmacological treatment, or even further physical/functional damage to the skin barrier, not to mention the unfruitful costs and the loss of confidence in topical non-pharmacological treatments.

This consensus document, which was discussed and compiled in a series of meetings by a group of Italian dermatologists experienced in the field of eczematous diseases, represents an attempt to fill this void in the current scientific literature, providing physicians with a practical guide on characteristics, indications and correct use of topical non-pharmacological treatments currently available for eczema.

Epidemiology

Contact dermatitis

Despite CD is a common condition, several studies focused their attention only on its frequency in special workers (*e.g.* healthcare workers[10] or hairdressers[11]).

Indeed, assessment of the frequency of CD in the general population is particularly tricky, not only because of the possible underreporting of less severe cases, but also because this condition may co-exist with corticosteroid-responsive dermatoses, and, for this reason, remain neglected. Thus, any epidemiological study may underestimate the real prevalence of CD. Another prominent obstacle in defining the prevalence of CD is the agreement between different patch test sets, especially for irritant substances (*e.g.* formaldehyde); furthermore, the sometimes imperfect reproducibility of results may also affect the overall diagnoses.[12, 13]

Irritant CD (ICD) is by far the most common type of CD, as it is estimated to represent about 80% of total cases; it is mainly caused by degreasing agents, cosmetics, dust, foods, solvents, excessive use of soap and water.[12] For which concerns allergic CD (ACD), Alinaghi *et al.*, in a recent metanalysis of 28 studies, found that 20.1% of the general population was affected; the prevalence was higher in adults than in children (21.4% *vs.* 16.5%), in women than in men (27.9% *vs.* 13.2%), and in girls than in boys (19.0% *vs.* 12.4%).[14]

The trend of increasing prevalence from childhood to adulthood is consistent with the increase of cumulative allergenic stimulation and reactivity of the immune system.[5] Similarly to allergies and autoimmune diseases,[15-17] ACD is more frequent among females. No geographic differences were found between Europe and America, while no studies were conducted in Africa.[14] For which concerns Asia, epidemiological studies were carried out only in China, and showed a prevalence similar to the European one.[14, 18, 19] Interestingly, data are not consistent with the Global Burden of Diseases data on atopic dermatitis,[20-22] a pathological condition in which the skin barrier is impaired and the immune system may over-react to environmental allergens.

A new element in the epidemiology of CD has arisen during the COVID-19 pandemics. In this period, the prolonged and large-scale use of masks and gloves has caused a significant increase of ACD and ICD, because of skin barrier alterations and allergic sensitization caused by the components of such personal protective equipment.[23-28]

Moreover, use of hand sanitizers (alcohol- or not alcohol-based) and frequent handwashing, recommended for the prevention of COVID-19, have dramatically increased hand ICD. No extensive estimates of the modification of the frequency of ICD in the general population are currently available, but dermatological societies produced alerts and position papers suggesting to limit hand exposure.[29]

Atopic dermatitis

Epidemiological data are widely variable between studies, depending not only on the population considered, but also on discrepancies between methods used, disease definitions and diagnostic criteria, particularly for which concerns adult AD.[30]

AD is prevalently a childhood disease, although adults can also be affected. In about 80% of cases, it occurs in the first years of life, and in 60% of them undergoes spontaneous remission in adolescence.[30] The majority of adult AD patients are those who do not undergo such remission, but adult-onset AD represents 26.1% of all cases.[31]

Overall, females are affected more frequently than males.[30] Studies performed in the last 20 years report a point prevalence in children up to 18.2%; one-year prevalence is between 0.96% and 22.6%, one-year incidence between 10.2 and 95.6 per 1000 person years.[30] In adults,

one-year prevalence is between 1.2% and 17.1%, lifetime symptom prevalence is between 3% and 17.7%, one-year incidence is between 6.27 and 8.74 per 1,000 person years.[30]

Hand eczema

As in the case of CD, several studies suggest that epidemiological data concerning HE are likely underestimated: in detail, only two thirds of patients affected by HE refer to a physician for proper advice and treatment, and less than a half (44% or less) to a dermatologist.[3, 32]

According to literature, the estimated point prevalence of HE in the general population is around 4%, while one-year prevalence is approximately 10%.[3, 32] Lifetime prevalence is slightly higher, *i.e.* 11.3%.[32] Incidence is 5.5-8.8 cases per 1000 person-years, with a higher rate in females and substantially no difference between adolescents and adults.[3, 32]

Clinical aspects

Irritant contact dermatitis

Acute ICD is caused by exposure to an irritant or a caustic. Lesions develop soon after contact and are strictly confined to the exposed areas. Clinical manifestations (Figure 1) range from mild inflammation (erythema, oedema) to vesiculation, bullae and/or necrosis. Manifestations of chronic ICD may vary, depending on nature and concentration of the irritant, frequency of exposure and individual factors.[33-36] Typically, lesions are not well-demarcated,[35, 36] bilateral (sometimes asymmetrical and prevalent in the dominant hand), and initially affect the back of the fingers and the interdigital folds, progressively extending to the back of the hands and then to the palms. The extensor surface of the forearms may be involved, but the volar surface of the wrists is usually spared.[34]

Lesional skin appears moderately erythematous, xerotic and fissured; lichenification and fine or lamellar desquamation may be present (Figure 2). These aspects are typical of "wet workers". In some patients, hyperkeratotic forms with painful ragadiform fissures may develop, and severely affect quality of life. Acropulpitis is frequent in shoemakers and workers who handle small metal components; in these cases, thickening of the stratum corneum is favored by friction or traumatism, and may be responsible for the disappearance of fingerprints.[37] Exudation, blistering and crusted elements are less frequent.[35] Occasionally, chronic ICD of the hands may manifest with patches of nummular eczema located mainly on the dorsal surface (*e.g.* in the case of occupational exposure to cutting oils).[35] Another peculiar picture is apron dermatitis, characterized by erythema, xerosis and fissures on the palmar surface of 2-3 adjacent fingers, with semicircular extension on the palm. In chronic forms, ICD may also cause onycholysis, longitudinal striae or lamellar onychoschizia.[38]

For which concerns symptoms, ICD is characterized by a burning or stinging sensation, sometimes associated with pain. Itching may occasionally be intense and disabling.[33]

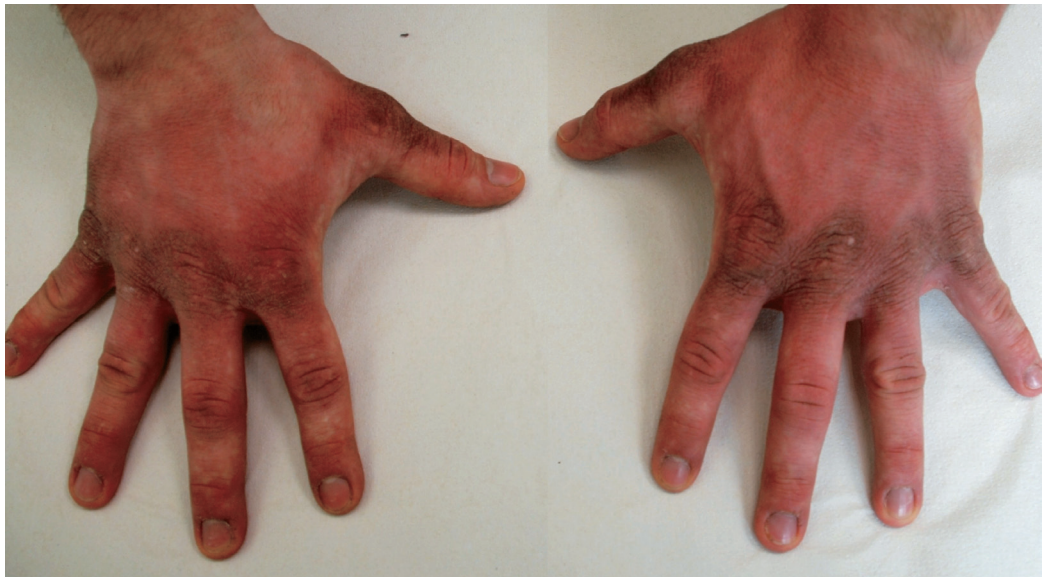

Figure 1.—Acute irritant contact dermatitis of the hands.

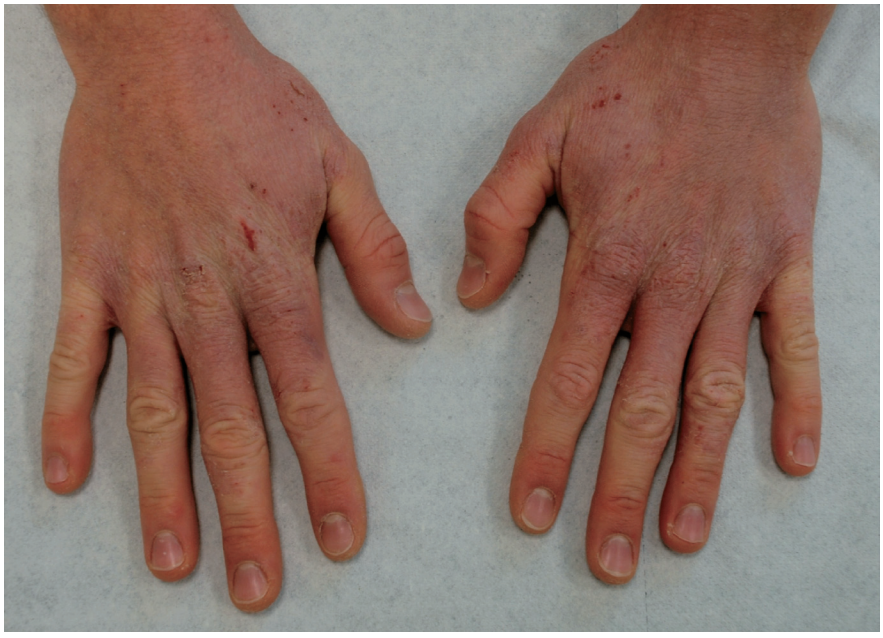

Figure 2.—Chronic irritant contact dermatitis of the hands.

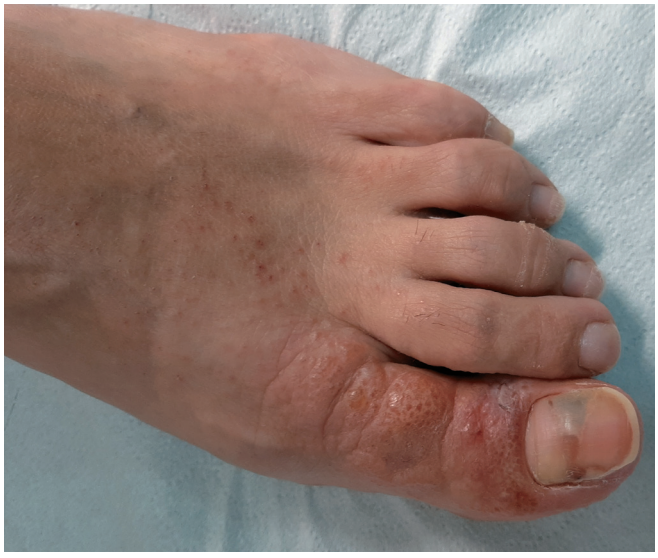

Figure 3.—Allergic contact dermatitis to an antifungal topical drug.

Allergic contact dermatitis

ACD is characterized by skin lesions with not well-demarcated borders, spreading beyond the area of contact with the causative allergen(s). The clinical picture (Figure 3) is usually eczematous, but it may vary depending on location and duration of the contact with allergen(s). In most cases, acute eruptions are characterized by erythema, edema and minute, barely raised, translucid, short-lasting vesicles, evolving into exudative and oozing lesions associated with yellowish crusts. Itching is always present, sometimes very severe. When hyperacute or related to peculiar haptens (non-steroidal anti-inflammatory drugs), ACD may present with bullae. Clinical signs and symptoms self-heal in a few days when no further contact with allergen(s) occurs, otherwise evolution is toward scaling and fissuration.[39]

ACD of the scalp is usually mainly characterized by severe scaling, while on eyelids and scrotum it often appears severely edematous.[39]

Other clinical pictures of ACD are: purpuric (mainly observed on the lower legs), lichenoid, pigmented (especially reported in oriental populations) and lymphomatoid.[39]

The term ectopic dermatitis refers to ACD caused by autotransfer or heterotransfer (by proxy) of allergens. Airborne ACD is instead due to contact with allergens suspended in the air, and typically involves areas not covered by clothes.[39]

Regional (via lymphatic vessels) or generalized (via blood vessels) dissemination of lesions is defined as "idic eruption," and may be eczematous or show atypical features (*e.g.* erythema multiforme-like appearance).[39]

Systemic reactivation of ACD (SRCD)[40] may occur when a patient with contact sensitization to an allergen is systemically re-exposed to the same (or a chemically closely related) allergen, and is characterized by a rash

with a symmetric pattern. Common causal agents are drugs, plants, preservatives, metals and fragrances. SRCD must be considered in all cases of widespread or recurrent dermatitis occurring in patients sensitized to contact allergens.

Contact allergy in atopic dermatitis

The relationship between AD and ACD is frequently debated in literature, especially for which concerns children.[41-43] Contact sensitivity is more frequent in AD patients compared with the general population,[42] and 40-65% of AD patients show contact sensitivity when patch tested.[41, 43] Indeed, in contrast with old hypotheses,[44] the two conditions share some immune pathways (Th1, Th2, Th9, Th17).[45, 46]

Epidermal barrier dysfunction enhances percutaneous absorption of allergens and irritants, including those contained in topical drugs and personal care products.[47] On the other hand, irritants further increase epidermal barrier dysfunction,[48] and the combined action of allergens and irritants may influence the elicitation threshold in ACD.[49] Moreover, the bacterial and fungal colonization observed in AD favors the onset of contact sensitivity, by stimulating an inflammatory environment.[48, 50]

Differential diagnosis between AD and ACD may be difficult: AD is more frequent in children, ACD in adults. ACD may involve flexures, mimicking AD. Patch test in AD patients is useful when performed by expert operators on the basis of clinical, therapeutic, and occupational criteria.[41, 51]

- Clinical criteria: patch test is recommended in children and adult AD patients with worsening or generalized dermatitis, and in case of involvement of atypical AD sites (Figure 4-6).
- Therapeutic criteria: patch test is recommended when AD does not improve, or worsens, with topical therapy, or relapses quickly after the end of treatment. In these cases, active ingredients and additives/preservatives contained in topical products may be responsible for ACD.
- Occupational criteria: patch test is recommended in adult AD patients affected by HE and exposed to occupational contact allergens (*e.g.* cleaners, hairdressers, housewives, metalworkers, mechanics, healthcare personnel).

In all cases of positive patch test, relevance of sensitization(s) must be investigated.[13] It is noteworthy that a lower irritation threshold, especially for fragrances, metals, formaldehyde, and lanolin, is frequent in AD patients,[52] and also that false negative patch test results may occur during active AD and when AD is severe.[47]

The most frequently reported contact allergens in AD patients are metals (nickel sulphate, cobalt chloride, potassium dichromate), fragrance markers (fragrance mix I, fragrance mix II, *Myroxylon pereirae*, and hydroxyisohexyl-3-cyclohexenecarboxaldehyde), isothiazolinones, corticosteroids, acrylates, lanolin, neomycin, formaldehyde, sesquiterpene lactone mix, and *Compositae* mix.[43, 53-55]

Hand eczema

HE is a common, often chronic, heterogeneous disease localized to the hands and wrists, and frequently associated with foot eczema. An eczema is generally considered chronic in case of persistence for more than 3 months or occurrence of more than two relapses/year.[56] Clinical manifestations of HE can be multiple, and pathogenetic classification is sometimes difficult. Molin *et al.* identified eight major subtypes: ICD, ACD, combined allergic and irritant contact dermatitis (each with or without atopy), atopic HE and idiopathic HE. For each form, three morphological patterns may exist: hyperkeratotic-rhagadiform, dyshidrotic or mixed.[57]

Chronic and severe forms (5-7% of cases) may have a serious impact on daily activities, social life and working ability of patients,[58, 59] and represent a relevant economic burden for the society.[60-62]

Several endogenous and exogenous factors, not yet clearly identified, are involved in relapses and transition to chronicity.

Endogenous factors are related to the skin barrier function. A less robust and more permeable barrier, *e.g.* in the case of atopy or atopic dermatitis, enhances percutaneous penetration of allergens and irritants, leading to local immune activation.[32, 63]

Exogenous factors include all exposures, occupational and/or domestic, that may damage the skin barrier (*e.g.* water/wet work, irritants). Cold and dry weather, as well as decreased indoor humidity, may also be important. Contact allergy represents another risk factor for the development of HE.[32, 64, 65] Protein CD is due to immediate hypersensitivity to proteins but may also cause chronic HE in the long term, and is diagnosed through prick test or specific serum IgE. The most frequent triggers are food allergens and latex.[66]

The tendency to develop chronic HE is strongly influenced by work, hobbies or household chores. Chronic HE more frequently occurs in young women, probably because of a higher burden of wet work.[32, 67]

Acute clinical manifestations of HE are papules, macules and vescicles, which appear exudative in later stages,

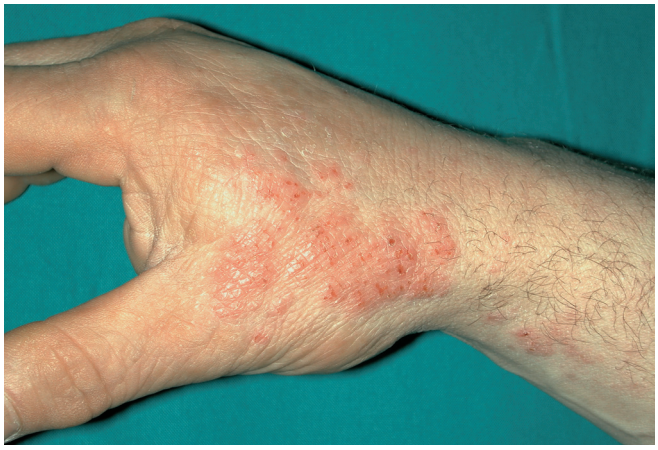

Figure 4.—Occupational allergic contact dermatitis in atopic patient, caused by potassium dichromate contained in leather gloves.

with oozing and crusting scales; in the chronic form, HE is characterized by lichenification, hyperkeratosis and fissuration. All clinical stages are accompanied by intense itching.[32, 67]

Histology of acute HE shows intercellular oedema, spongiosis, acanthosis and hyperparakeratosis, and, in the upper dermis, perivascular infiltration of lymphocytes that migrate into the epidermis. In chronic lesions, spongiosis is barely discernible, while acanthosis is remarkably accentuated, often with a thick, hyperkeratotic horn associated with minimal parakeratosis.[32, 67, 68]

One of the most used parameters for the evaluation of HE is the physician's global assessment (PGA) of overall severity, which includes five categories, classified with numbers from 0 to 4: "clear" (0), "almost clear" (1), "mild" (2), "moderate" (3) and "severe" (4).[6]

The modified Total Lesion Symptom Score (mTLSS) is the sum of seven items (erythema, oedema, vesicles, desquamation, hyperkeratosis, fissures, and pruritus/pain), each scored on a 4-point scale (0= absent, 1= mild, 2= moderate, 3= severe). A high mTLSS represents severe HE. The mTLSS relates to the PGA, and a photographic guide has been developed to train observers.[6]

A more disease-specific scoring system is the Hand Eczema Severity Index (HECSI),[69] that evaluates, in five anatomical locations (fingertips, rest of fingers, palm of hands, back of hands, wrists), the expression of six signs (erythema, infiltration/papulation, vesicles, fissures, scaling, edema) with a score from 0 (absent) to 3 (most severe), and the extension of lesions with a score between 0 and 4 (0=not involved, 1=up to 25% involved, 2=26-50% involved, 3=51-75% involved, 4=76-100% involved). The multiplication of the total sign score by the extent score yields a number between 0 (no HE) and 360 (worst possible HE).

Differential diagnoses of HE include tinea manuum, palmar psoriasis, scabies, mycosis fungoides, porphyria cutanea tarda, hand-foot-and-mouth disease.[32, 67]

According to literature data, traditionally reported risk

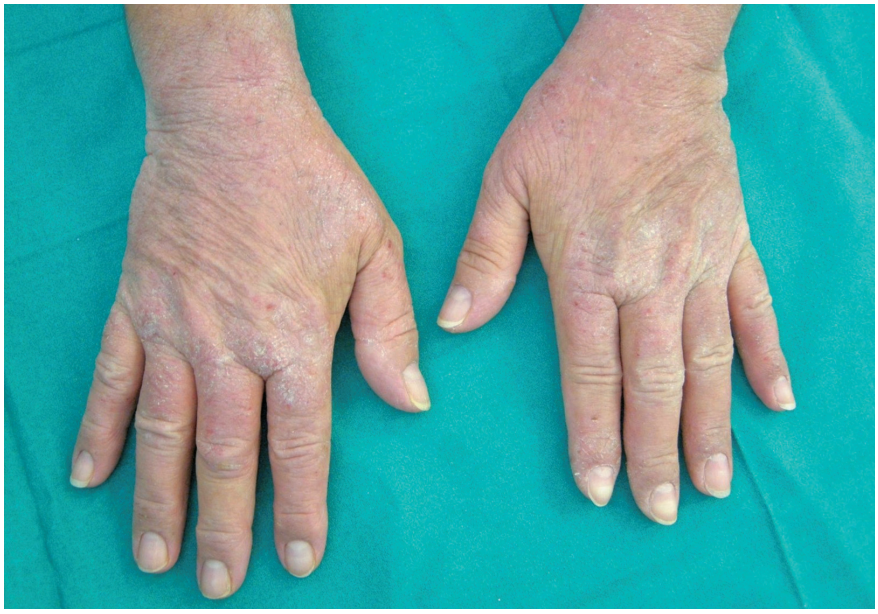

Figure 5.—Non-occupational allergic contact dermatitis in atopic patient, caused by thiurams contained in gloves for housework.

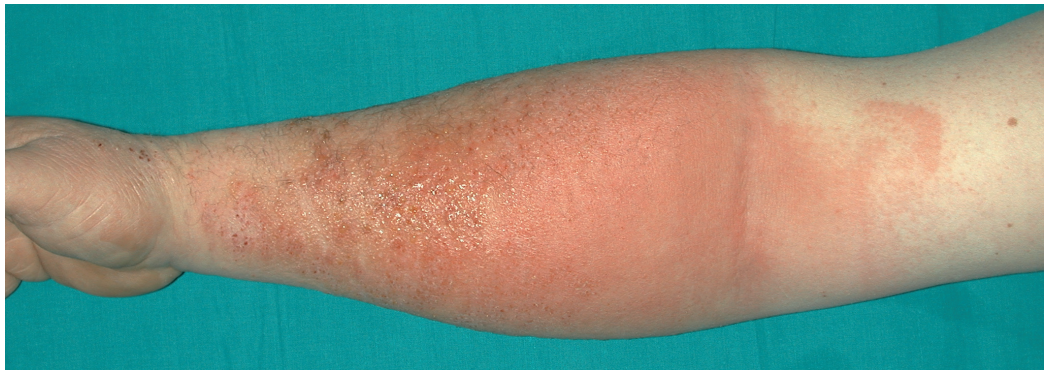

Figure 6.—Exuding allergic contact dermatitis in atopic patient, caused by propolis.

factors, such as AD and contact sensitization, do not influence prognosis of HE, while lifestyle factors, tobacco smoking, obesity, stress and lack of physical exercise are negative prognostic factors.[9, 65, 67]

Topical non-pharmacological treatments of eczema

Moisturizers

Management strategies for eczema include active treatments, that address inflammatory lesions, and adjunctive therapies, to optimize skin barrier function and prevent flare-ups.[70, 71]

Moisturizers or emollients represent a treatment that prevents loss of skin water, hydrates and makes skin less sensitive and irritated. Ultimately, the functions of emollients are to reduce the dryness of skin, decrease trans-epidermal water loss (TEWL), improve comfort and reduce itch.[72, 73]

Composition and technology of emollients greatly evolved over time, hand in hand with the increase in knowledge of the structure of skin barrier and the progress of chemical and biochemical industry. This evolution led from the use of occlusive and hygroscopic molecules, good to improve hydration but with many shortcomings (objective and also in terms of patient compliance), to second generation products, containing substitutes of the natural moisturizing factor, and later to formulations aimed to structurally and functionally restore the skin barrier. Recently, the term prescription emollient devices (PEDs) was introduced to identify a new class of topical agents, designed to target specific defects of skin barrier function and containing several components, including anti-inflammatory molecules (Table I). These agents play an important role in reducing inflammation and itching in eczema.[74]

PEDs are approved as medical devices, under the claim that they play a structural role in the skin barrier function and do not exert their effects through chemical actions. PEDs contain mixtures, variable by nature and proportions of ingredients, of lipids, ceramides, fatty acids, and natural anti-inflammatory agents such as glycyrrhetinic acid, as well as other ingredients to alleviate itch and inflammation.[75]

Regular use of emollients reduces relapses of eczema. Since xerosis is one of the most important features of this condition, skin emollients constitute an integral part of the standard treatment for eczema of any severity.[70, 76] Indeed, various authors demonstrated that regular and correct use of emollients significantly reduces the use of topical corticosteroids and topical calcineurin inhibitors, as well as the number of flares, in AD patients;[77] less data, but equally promising, are available on their role in the treatment of other eczematous diseases.[78]

Indications on the use of emollients in eczemas of different nature and severity are shown in Table II. Moisturizing the skin may be sufficient in many cases to control mild eczema, plays an important role in association with topical and systemic drugs– in the management of more severe forms, and may also have a role in the prevention of eczema flares.

Barrier creams

Barrier creams, also known as skin protective creams or invisible gloves, act as a physical barrier, in order to prevent or reduce the penetration of irritants and allergens into the epidermis.[79, 80] They are commonly used to protect hands, face and neck, especially in occupational contact dermatitis; effectiveness could also be associated with their emollient activity. Application of barrier creams is recommended before and during work and, for this reason, they are also called pre-work creams.[81]

In 2003, the Federal Register of the Food and Drug Administration provided a series of active ingredients, and their quantitative range of use, approved for barrier cream formulations.[82] The most commonly used ingredients are classified as emollients, humectants and hydrating agents. Emollients, which mainly consist of lipids and oils, reduce TEWL by creating a hydrophobic film over the skin. Humectants show high affinity for water (hygroscopicity), enhancing water absorption from dermis and external environment. The most extensively used humectant agents are glycerol, propylene glycol, and 70% sorbitol solution. Hydrating agents maintain the water of the stratum corneum. Urea, allantoin and hyaluronic acid are frequently used as hydrating agents.[74]

Barrier creams can be classified as water-repellent and water-soluble (oil-repellent) products.[79]

Water-repellent creams can be classified as occlusives, because they create a hydrophobic film which prevents contact between the skin and water-soluble substances. Petrolatum, silicones and paraffin waxes are the most common ingredients with water-repellent properties. They are water-resistant, but can be removed by oils and fats. They should be used to protect against acids, alkalis, soaps, detergents and water-soluble irritants.[79]

Water-soluble creams act as a repellent for oils and solvents, and should be used to protect against oils, varnishes, organic solvents and lipo-soluble irritants. They consist of hydrophilic substances such as glycerin, sorbitol, talc, zinc oxide and kaolin. Because of solubility in water, they lose efficacy in case of profuse sweating.[79]

Active barrier creams, very popular in the past, consist of creams containing active ingredients which could trap or transform allergens. Some authors reported that barrier creams containing tartaric acid and glycine show beneficial results in some chromate-sensitive workers, because they are able to chelate chromate.[83] Other studies demonstrated that some chelating substances, such as EDTA (ethylenediaminetetraacetic acid), are able to bind nickel or other metals or reduce their penetration through the skin, thus reducing symptoms in sensitive workers.[84]

The ideal barrier cream should be easy to apply and to remove, have a long duration and good tolerability for the patient. From a cosmetological point of view, it should be hypoallergenic, not sensitizing, fragrance-free, alcohol-free, and not comedogenic.[79] To carry out its protective function, it should be applied frequently and in generous quantities, without forgetting some skin areas such as back of the hands, fingertips and interdigital spaces. The barrier cream should be reapplied regularly and, at least, after every hand washing.

Although barrier creams are highly recommended to prevent eczema, particularly occupational contact dermatitis, their protective efficacy is controversial.[85, 86] Their use should be limited to contact with low-grade irritant substances such as water, detergents, organic solvents and cutting fluids. In fact, barrier creams cannot guarantee a protection comparable with gloves or other personal protection equipment. However, they may be used to increase protection against irritants, and often remain the only possible preventive measure in case of occupations which require high tactile sensitivity or high mobility of fingers, or when protective clothing cannot be safely used.

In conclusion, barrier creams could play a preventive role and reduce the risk of eczema, especially in working environment. Regular, frequent, and correct application should be encouraged in order to increase their efficacy. Nevertheless, their protective efficacy is controversial and further studies are required.[79-86]

Cleansing in patients with eczema

Patients with eczema generally have a sensitive skin. But what is a sensitive skin? It is described as "an unpleasant sensory response to different stimuli that should not provoke such sensations."[87] While this is a good explanation, it is sometimes difficult to objectively identify people with sensitive skin. The concept is usually based on anamnes-

TABLE I.—*Characteristics of different generations of emollients.*

1st generation	2nd generation	3rd generation	4th generation
Occlusive, hygroscopic molecules	Substitutes of the NMF (natural moisturizing factor)	Epidermal lipids	Emollients "plus" or PEDs (prescription emollient devices)
e.g. vaseline, paraffin, fatty alcohols, polysaccharides, chitosan, glycerol	*e.g.* urea, lactic acid, pyrrolidone carboxylic acid, aminoacids	*e.g.* ceramides, cholesterol, polyunsaturated fatty acids	*e.g.* plant extracts, bacterial lysates, engineered derivatives of hyaluronic acid, antioxidants
Target: hydration improvement	Target: NMF enrichment	Target: skin barrier restoration	Target: inflammation, oxidative stress, microbiome

TABLE II.—*Indication for use of emollients in eczemas of different type and severity.*

	ICD Irritant contact dermatitis	ACD Allergic contact dermatitis	CHE Chronic hand eczema	AD+CD Atopic dermatitis + contact dermatitis
Single treatment	Mild 2-3 times a day for at least 2 weeks	Mild Twice daily for at least 3 weeks	Mild Twice daily for at least 3 weeks	Mild Twice daily for at least 3 weeks
Association with pharmacological therapy	Moderate/severe Once daily for the duration of the pharmacological therapy, then twice daily for at least 2 weeks	Moderate/severe Once daily for the duration of the pharmacological therapy, then twice daily for at least 4 weeks	Moderate/severe Once daily for the duration of the pharmacological therapy, then twice daily for at least 4 weeks	Moderate/severe Once daily for the duration of the pharmacological therapy, then twice daily for at least 4 weeks
Flares prevention	(3rd generation emollients)	Week-end therapy twice daily for 4-8 weeks + 3rd generation emollients	Week-end therapy twice daily for 4-8 weeks + 3rd generation emollients	Week-end therapy twice daily for 4-8 weeks +3rd generation emollients

tic history of patients. Although some methods have been developed to identify sensitive skin, they have limitations, not all patients with sensitive skin develop specific sensory reactions to every irritant used in the test (like lactic acid or sodium lauryl sulfate), reactivity is different between anatomical sites of the body and in relation to gender, age and ethnicity, and, in addition, even intrapersonal variability exists in individual response to different irritants.[88, 89] As an example, normal skin of the face is considered more sensitive than other parts of the body by more than half of the general population. All the more so, skin affected by eczema of any kind is sensitive. A typical case is the skin of AD patients, who have a defective skin barrier and increased vascular reactivity.

The non-pharmacological management of skin affected by eczema begins with appropriate cleansing.[90] A cleanser must be gentle, but water alone is not recommended. In a study, 130 patients with AD used water alone to wash their skin, but they experienced persistent lesions despite treatment with topical corticosteroids.[91] In addition, water alone does not remove all the impurities on skin surface.

Soaps are products resulting from the action of alkaline substances on fatty acids, a process known as saponification. When a soap has a high pH, its molecules bind to stratum corneum proteins, inducing swelling and hyperhydration of the skin. After washing, the excess of water evaporates, and the soap molecules bound to proteins reduce the ability to hold water, ultimately causing skin dryness.[92] On the other hand, altering the ratio between fatty acids and alkali results in lower cleansing ability.

Nowadays, synthetic detergents (syndets) are more used. They can be easily formulated at a pH closer to that of skin, to decrease their ability to remove cutaneous lipids. Syndets can be packaged as bars (the most used form) or liquids. However, bars and liquids have a similar composition, and can be combined into a formulation known as a combar. Syndets are usually added to lipophilic moisturizing ingredients as petrolatum, vegetable oils or shea butter. If water concentration is high, after rinse these moisturizing ingredients are deposited on the skin in a thin film, without damage to skin barrier.[93]

Many other formulations are also used, such as cold cream cleansers, cleansing milks, cleansing oils, oil cleansing balms. Cold creams, composed of water, beeswax and mineral oils, are usually used as facial cleansers and cosmetic removers for patients with dry skin. Cleansing milks are a combination of water and lightweight oils, like olive oil, jojoba oil, sunflower oil, and emollients such as glycerin; they are mainly used to remove eye cosmetics. Cleansing oils are water in oil emulsions, usually containing mineral oil, castor oil, jojoba oil and olive oil. Oil cleansing balms are similar to cold creams, but have a petrolatum jelly consistency; they liquefy when they come in contact with warm skin. These balms contain different oils (mineral oil, sunflower oil, mango seed butter), combined with either beeswax or shea butter.[92]

Cold creams, cleansing oils and cleansing baths are usually prescribed for dry-sensitive skin. For patients with this kind of skin, non foaming cleansers are recommended, which contain glygerin, stearyl alcohol, cetyl alcohol and sodium lauryl sulfate as a surfactant. Glycerin, cetyl and stearyl alcohol leave on the skin surface a thin moisturizing film, which protects the epidermal barrier.

Cleansers must be free of perfumes, which increase the risk of sensitization.[91] Other elements to be considered for appropriate cleansing of sensitive skin are water temperature and duration of bath. The temperature of bath water should not exceed 37 °C; a temperature between 34°C and 36°C is ideal. A bath should not last more than 5 minutes, because overhydrated superficial layers of the epidermis

become more fragile. In patients with AD, it is suggested to gently apply detergent directly on the skin, using hands, and then rinse it off with water. The quantity of emollients deposited on the skin via a bath additive is lower than that from direct application.[94]

When discussing about AD skin, which is the prototype of sensitive skin, possible colonization by microorganisms, in particular *Staphylococcus aureus,* must be considered. For this situation, some authors recommend frequent baths to remove scales and crusts, while other authors suggest to avoid frequent baths, that could irritate dry skin.[94] Antiseptic baths should be reserved for patients with impetiginization. Potassium permanganate at very low concentration (1/10,000) or chlorhexidine (5/1000-5/10,000) are considered safe and well tolerated by some authors, but in some cases, episodes of allergy have been described.[95]

As reported by Cheong in a review, patients with sensitive skin should be advised that correct cleansing is an important part of their treatment and can enhance the effect of medications.[90] In fact, some authors have shown that non aggressive cleansing, in combination with topical steroid treatment, resulted in a faster improvement in AD patients, and other authors suggested that use of a syndet bar reduced the severity of eczema and maintained hydration of the skin over 28 days in patients with mild AD treated with topical corticosteroids.[91]

Conclusions

In the light of available data, proper use of emollients, cleansers and barrier creams should be considered a useful complement of pharmacological therapy and an integral part of the overall treatment, management and even prevention of all forms of eczema. Modern products are able not only to increase skin hydration and contribute to the repair/maintenance of barrier function, but also to reduce inflammation and oxidative stress and help restoring the equilibrium of skin microbiota. They can be sufficient to prevent flares and even treat cases of mild eczema, while in more severe forms they can work in synergy with pharmacological treatments and act as steroid-sparing or, more generally, drug-sparing agents. In subjects at risk because of their activities and/or sensitive/reactive skin, regular use of these products, in association with adequate prevention measures, may decrease the incidence of eczema. While costs may appear a limit in the short term, also because they are usually not covered by national health systems, this approach proves to be cost-effective in the long term and stably improves the clinical picture and quality of life of patients.

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