Atopic dermatitis and seborrheic dermatitis

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Abstract

Atopic dermatitis is a common and frequently familial inflammatory dermatosis, which usually appears during infancy or early childhood and is often associated with other atopic diseases such as asthma, allergic rhinoconjunctivitis, food allergies or eosinophilic esophagitis. It is a complex genetic disease with environmental influences, characterized by intense pruritus and a chronic course in flares. In infants and young children, cheeks, scalp and extensor surfaces of the extremities are affected and in older children and adults the lesions are usually located in the flexion folds, or in specific locations such as the perioral area, eyelids, hands or feet. In most patients the disease disappears when puberty arrives, but it can also appear in adulthood.

Seborrheic dermatitis is a common inflammatory dermatosis, with an infantile and an adult clinical form. Lesions are located on the scalp, ears, face, central part of the chest and intertriginous areas. There is an etiological relationship with active sebaceous glands, alterations in sebum composition and the genus Malassezia (Pityrosporum).

Key words: Atopic dermatitis; Eczema; Atopy; Seborrheic dermatitis; Malassezia (Pityrosporum).

Resumen

La dermatitis atópica es una dermatosis inflamatoria común y frecuentemente familiar que, habitualmente, aparece durante la lactancia o la primera infancia, y se asocia con frecuencia a otras enfermedades atópicas, como: asma, rinoconjuntivitis alérgica, alergias alimentarias o esofagitis eosinofílica. Es una enfermedad genética compleja con influencias medioambientales, caracterizada por un prurito intenso y una evolución crónica en brotes. En lactantes y niños pequeños, se afectan: mejillas, cuero cabelludo y superficies de extensión de las extremidades; y en niños mayores y adultos, las lesiones suelen localizarse en los pliegues de flexión o en localizaciones específicas, como: zona perioral, párpados, manos o pies. En la mayoría de los pacientes, la enfermedad desaparece cuando llega la pubertad, pero también puede aparecer en la edad adulta.

La dermatitis seborreica es una dermatosis inflamatoria común, con una forma clínica del lactante y otra del adulto. Las lesiones se localizan en: cuero cabelludo, orejas, cara, parte central del tórax y áreas intertriginosas. Hay una relación etiológica con las glándulas sebáceas activas, las alteraciones en la composición del sebo y el género Malassezia (Pityrosporum).

Key words: Dermatitis atópica; Eczema; Atopia; Dermatitis seborreica; Malassezia (Pityrosporum).

Atopic dermatitis

Introduction / definition

Atopic dermatitis is characterized by flares of inflammatory, itchy lesions, with a characteristic distribution and personal and / or family history of atopy (allergic rhinoconjunctivitis, asthma, food allergies, etc.).

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Atopic dermatitis (AD) and food allergy have a predilection for infants and young children; whereas, asthma prevails in older children and rhinoconjunctivitis predominates in adolescents. This characteristic age-dependent sequence is called “atopic march”, however, it does not always manifest, as these diseases may or may not appear, and do so simultaneously or at different ages. It is more prevalent in children (10-20%) than in adults (1-3%) and, in 90% of cases, it appears in childhood (45% during the first 6 months of life and 60% before the first year of life). At least 50% of atopic children will continue to express certain manifestation of the disease during adolescence and 20% also in adult life(1,2).

Most of the cases can be considered mild, but 10% of the patients suffer a
severe form which is more prevalent in the adult population. The prevalence of severe atopic dermatitis in adults in Spain is estimated to be 0.08% (3).

In the most severe forms, the sleep cycle is disturbed leading to irritability, which affects school and sport performance, self-esteem, social relationships, as well as routine and leisure activities.

On the other hand, AD, and especially severe AD, implies a significant economic expense, both direct (medical visits, pharmacological cost) and indirect costs (loss of school hours and productivity), and both at a personal level as well as for health systems. This emphasizes the need to evaluate its impact on the family environment and on the patients’ caregivers. Hence, it should be recognized as a “family” disease, rather than as an individual one and, as such, it must be evaluated(4).

Etiopathogenesis

A deficient skin barrier action, an abnormal immune response, alteration of the cutaneous microbiome and an important psychosomatic influence are the main etiopathogenic factors.

It is a complex genetic disease, with interactions between different genes and of these with the environment.

In patients with AD, the lesional skin and, to a lesser extent, the non-lesional skin, present a defective cutaneous barrier, with: increased transepidermal water loss, alteration of skin lipids, increased epidermal proliferation, reduction of Filaggrin expression, inflammation, and increased number of IgE receptors on Langerhans cells. The uninjured skin also shows different immunological profiles to normal skin (17% more T cells than normal skin and increased expression of Th1, Th2 and Th22 lymphocytes). Antigens that cross the skin reach the ganglia (via dendritic cells) and stimulate Th2 response with the consequent increase in the production of IgE and several other mediators from various inflammatory and epidermal cells. Immunity mediated by the Th1 pathway (which is attenuated) and the innate immune system contribute to skin inflammation with the release of cytokines and a deficiency of antimicrobial peptides.

Patients with AD show important changes in skin microbiome, mainly involving a decrease in *Staphylococcus Epidermidis* and a dominant colonisation of *Staphylococcus Aureus* (in up to 90% of the lesions), which correlates with the severity of the disease. Conversely, recovery of microbiome diversity precedes resolution of flare-ups.

All these abnormalities interact with each other; so that: barrier dysfunction alters the skin’s microbiome and immune response; the skin microbiome alters the immune response and the skin barrier; and, lastly, immune dysregulation also alters the cutaneous microbiome and the skin’s barrier function(5).

Manifestations and diagnosis

The diagnosis of AD is mainly clinical and remains based on the classical criteria described by Hanifin and Rajka(6): 3 or more major criteria, and 3 or more minor criteria (Table I).
The diagnosis of AD is primarily clinical (Figs. 1-5). Characteristically, the typical lesions of atopic eczema are erythematous areas of skin, often poorly defined, with intense itching, although their distribution and characteristics vary with age. There are also some characteristic clinical manifestations that must be known (Table II).

It is also important to distinguish between acute, subacute and chronic forms, as the topical treatment differs (Table II) and to identify other clinical manifestations of AD, which are very common (Table III).

### Differential diagnosis

The symptoms of AD are highly characteristic, thus most of the possible differential diagnoses can be excluded by the medical history (scabies, seborrheic dermatitis, irritant contact dermatitis and ichthyosis). In some cases, it may be necessary to perform biopsies (cutaneous T-cell lymphomas and psoriasis) or to perform contact tests (allergic contact or airborne dermatitis), or special studies (photosensitive diseases, diseases due to deficiencies of the immune system.

### Table II. Clinical forms of atopic dermatitis

<table>
<thead>
<tr>
<th>According to age</th>
<th>Lesions are located on: cheeks, trunk, and extensor surfaces of extremities</th>
</tr>
</thead>
<tbody>
<tr>
<td>From 3 - 6 months to 2 years old</td>
<td>Lesions are typically located in: popliteal fossa and anterior surface of elbows, eyelids, retroauricular folds and around the mouth, and other lesions appear, such as: prurigo, Pityriasis amiantacea on the scalp, digitopulpitis, juvenile plantar dermatitis or dyshidrosis. It is mainly during this period, when the minor criteria must be taken into account: keratosis pilaris, white dermographism, pityriasis alba, third crease in the lower eyelid (Dennie-Morgan sign), itch with exercise, intense xerosis (rough skin), etc. (Table I)</td>
</tr>
<tr>
<td>After 12 years old</td>
<td>Lesions do not have a preferential location and can appear in any of the previously described locations</td>
</tr>
</tbody>
</table>

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<tr>
<th>According to course</th>
<th>Lesions are typically located in: popliteal fossa and anterior surface of elbows, eyelids, retroauricular folds and around the mouth, and other lesions appear, such as: prurigo, Pityriasis amiantacea on the scalp, digitopulpitis, juvenile plantar dermatitis or dyshidrosis. It is mainly during this period, when the minor criteria must be taken into account: keratosis pilaris, white dermographism, pityriasis alba, third crease in the lower eyelid (Dennie-Morgan sign), itch with exercise, intense xerosis (rough skin), etc. (Table I)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute eczema</td>
<td>They present with: intense erythema, edema and exudation, sometimes microvesicles appear and, occasionally even blisters. Pruritus is intense</td>
</tr>
<tr>
<td>Impetiginized eczema</td>
<td>Atopic dermatitis lesions can become impetiginized (especially acute ones) and, in this case, there are yellowish (meliceric) crusts over eczema lesions</td>
</tr>
<tr>
<td>Subacute eczema</td>
<td>There is a less intense erythema and “less itching”. Exudation has practically disappeared and a slight peeling can be observed</td>
</tr>
<tr>
<td>Chronic eczema (neurodermatitis or lichen simplex chronicus)</td>
<td>They are areas of skin with infiltrated papules or plaques, with intense itching and significant dryness of the skin</td>
</tr>
</tbody>
</table>
Atopic Dermatitis And Seborrheic Dermatitis

Erythroderma due to other causes, etc.). It is worth mentioning that the usefulness of atopy patch tests used in the last 20 years for the diagnosis of food and aeroallergens allergies is very limited, because although they are specific, they are not sensitive. The latter holds true especially in children with gastrointestinal symptoms related to food allergy and, in the case of aeroallergens that could trigger AD, there is insufficient data (7,8).

Table III. Other clinical manifestations of atopic dermatitis

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Popular urticaria</td>
<td>They are flares of papulovesicular lesions (sometimes even bullous) irregularly distributed, especially on the limbs, with intense itching. There is usually a triggering factor 24 - 48 h before, for instance: insect bites</td>
</tr>
<tr>
<td>Pityriasis amiantacea</td>
<td>They are clusters of scales and crusts adhering to each other and to the hairs, as a result of exacerbations of eczema on the scalp that, often times, have remained unnoticed</td>
</tr>
<tr>
<td>Dyshidrosis or dyshidrotic eczema</td>
<td>They are vesicular lesions that appear on the lateral surfaces of the fingers and/or toes, and they can spread to the palms and soles. They are acute, intensely itchy lesions, which usually resolve spontaneously within 1 – 2 weeks. On many occasions the triggering factor are sudden changes in temperature, but interdigital ringworm must always be ruled out, as it can also be the trigger for flares</td>
</tr>
<tr>
<td>Digitopulpitis and juvenile plantar dermatitis</td>
<td>They are fissures that appear on the pads of the fingers (digitopulpitis) or toes (juvenile plantar dermatitis), especially on the 1st finger/toe, accompanied by extreme dryness, which evolve chronically appearing at the beginning of adolescence, and disappearing spontaneously beyond 12 - 13 years of age</td>
</tr>
</tbody>
</table>

There are multiple assessment scales for the disease and quality of life, which were used primarily in research and which have now been transferred to routine clinical practice, although as we have already mentioned, only for patients with severe AD clinical pictures (9) (Table IV).

Table IV. Most widely used atopic dermatitis severity rating scales

<table>
<thead>
<tr>
<th>Scale</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eczema Area Severity Index (EASI)</td>
<td>An objective assessment scale (performed by the physician) that ranges from 0 to 72 points and that counts lesions based on 4 body regions (head and neck, trunk, lower and upper extremities), as well as their extent and severity</td>
</tr>
<tr>
<td>Scoring atopic dermatitis (SCORAD)</td>
<td>It is an objective assessment scale (area / intensity), but it also includes a subjective assessment of the patient (itching / sleep) that ranges from 0 to 103 points</td>
</tr>
<tr>
<td>Investigator / Physician Global Assessment (IGA / PGA)</td>
<td>Objective scale, which indicates the global involvement, in a range that goes from 0 to 4 points</td>
</tr>
<tr>
<td>Body Surface Area (BSA)</td>
<td>Indicates the affected body surface in terms of percentage (from 0 to 100%)</td>
</tr>
<tr>
<td>Visual Analogue Scale (VAS)</td>
<td>A subjective scale in which the patient scores the intensity of itching on a linear scale ranging from 0 to 10 points</td>
</tr>
<tr>
<td>Dermatology Life Quality Index (DLQI, Children DLQI)</td>
<td>It is a 10-question test inquiring about how the patient feels, with a score ranging from 0 to 30 points</td>
</tr>
</tbody>
</table>

Assessment of AD severity

In recent years, the objective assessment of the severity of AD has become especially important in the daily clinic, with the appearance of new treatments for severe AD and their high cost, as the fundamental criterion for deciding: first, its administration (limited to severe forms and with lack of response to classical treatments); and, subsequently, the continuity of its administration.

Approach / Treatment

In the management of AD, the treatment of the disease is as important or more as the prevention of the factors that trigger the flare-ups. Recommendations include: avoiding triggers, making a regular use of emollient creams and treat flare-ups, so as to contain subclinical inflammation and symptomatic exacerbations (9,10).

Prevention

In preventing flare-ups, the education of children and parents, although time consuming, is particularly important. A study of an educational program for children, showed that 97% of those who received education on atopic dermatitis, obtained a significant decrease in SCORAD (Scoring Atopic Dermatitis) (Table IV) after 6 months (11).

In addition to educating on the characteristics and likely progress of the disease along with the basic measures to prevent flares, it must be taken into account that, in our society where “everyone” has an opinion, there are many things that are unproven and therefore must be avoided. For example:

- **Only in patients with a proven food allergy, have exclusion diets proven certain degree of usefulness, although unable to fully control AD flare-ups.** Only double-blind, placebo-controlled provocation tests are reliable. Allergy tests, RAST and prick tests, are positive in more than 40% of patients, without this involving clinical relevance. Epicutaneous tests to foods, which can be useful in patients with negative RAST and prick tests, although specific, they are not very sensitive. It has been found that the exclusion of the most common allergens (cow’s milk, eggs, nuts, soy and fish)
in high-risk children or in their mothers during pregnancy and lactation, decreases the prevalence of atopy during the first 2 years of life, although these differences are not sustained in the long term. These restrictive diets are very difficult to maintain overtime in children and can even turn out to be harmful (for instance, there is an increase in the frequency of allergy to peanut by delaying its introduction). Some epidemiological studies have shown a significant association between food diversity given during the 1st year of life and protection against allergy associated with AD. Some studies looking into the potential benefit of prebiotics and probiotics, remain currently inconclusive.

- Exclusive breastfeeding is recommended up to 4 months of age, as a method to prevent against food allergy associated with AD, but with a C level of evidence; that is, only one notch above experts’ opinion (D). Also, in children at high risk (first-degree relative with allergic symptoms), if breastfeeding is not possible, the use of hypoallergenic milk formulas is recommended; in this case, with a somewhat higher level of evidence (B)(9).

- Studies looking into the potential benefit of prebiotics and probiotics, remain currently inconclusive(14-16).

- Treatments not recommended by the doctor should be avoided (as they have not been proven to be effective or safe), such as: montelukast (leukotriene antagonist), topical capsaicin, essential fatty acids (topical or oral), phytotherapy (Chinese herbs), acupuncture, autologous blood, bioresonance, homeopathy, massage therapy or aromatherapy, salt baths and balneotherapy, vitamins and minerals, vitamin B12 in avocado oil, etc.(10).

- There is insufficient evidence to support that the use of non-sedating antihistamines reduces itching in patients with AD; however, they can be useful prior to exercise, because they reduce the itchiness caused by sweating. Similarly, sedative antihistamines, such as hydroxyzine, do not seem to control AD symptoms, however, by inducing sleep, they can improve rest and reduce scratching.

Healthy skin care is essential in these patients given the alterations in their skin barrier and increased transepidermal water loss, therefore: showers or baths should be brief (5 minutes) and with lukewarm water; bathing only 1-2 times a week is not a good idea, because it increases the skin proliferation of Staphylococcus Aureus; mildly irritating soaps should be used (oatmeal, oil soaps, etc.) and limited in extension (to surfaces that could be dirtier); the use of daily moisturizers or oil baths is essential; and the causes of itching and/or flare-ups must be avoided, such as the use of wool clothing directly on the skin as well as clothing or footwear that promote sweating, as well as exposure to clinically relevant allergens (diagnosed in allergy tests).

When relapses are frequent, it is useful to apply calcineurin inhibitors or topical corticosteroids (two/three times a week) between flares, in the areas usually affected by atopic dermatitis. The usefulness of twice weekly bleach baths is more doubtful (various studies have shown no differences with water-only baths) and there are no references to the usefulness of nasal mupirocin in AD(10).

Table V. Topical treatment of atopic dermatitis

<table>
<thead>
<tr>
<th>Acute eczema</th>
<th>Cold or lukewarm baths with astringent and anti-inflammatory solutions (oatmeal, chamomile, tar)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Medium-low potency corticosteroid cream or solution, 1-2 applications / day</td>
</tr>
<tr>
<td></td>
<td>High tolerance fluid moisturizers</td>
</tr>
<tr>
<td>Impetiginized eczema</td>
<td>Cold compresses with an antiseptic solution (1:1,000 copper / zinc sulfate or 1:10,000 potassium permanganate) 2-3 times a day</td>
</tr>
<tr>
<td></td>
<td>Medium or low potency corticosteroid cream + antibiotic / antiseptic</td>
</tr>
<tr>
<td>Subacute eczema</td>
<td>Medium or low potency corticosteroid cream, 1-2 times a day, until symptoms improve</td>
</tr>
<tr>
<td></td>
<td>Tacrolimus or pimecrolimus, 2 times / day, initially or when corticosteroids are discontinued, so as to prevent the “rebound” that is usually produced by the application of topical corticosteroids, up to 8-10 days after the last application of the latter</td>
</tr>
<tr>
<td></td>
<td>High tolerance fluid moisturizers</td>
</tr>
<tr>
<td>Chronic eczema</td>
<td>It is important to insist that the patient should AVOID SCRATCHING. Replace scratching with cold compresses with water or the application of cold moisturizers</td>
</tr>
<tr>
<td></td>
<td>Corticosteroids are the treatment of choice. They should be in the form of ointment or salve, of medium-high potency, and applied 1-2 times / day, even in occlusive dressing if necessary, for 8-15 days</td>
</tr>
<tr>
<td></td>
<td>Moisturizing ointments or salves several times a day</td>
</tr>
<tr>
<td></td>
<td>In cases with large extension, before proceeding to systemic treatment, the following may be useful:</td>
</tr>
<tr>
<td></td>
<td>The use of wet wrap pajamas with emollients or even low-medium strength corticosteroids</td>
</tr>
<tr>
<td></td>
<td>Oral antibiotics (cloxacillin, amoxicillin clavulanate or cefuroxime) on a short course (7-8 days) in the event of flares with lesions “colonized” by Staph. Aureus</td>
</tr>
<tr>
<td></td>
<td>Topical antifungals (ketoconazole or ciclopirox olamine) or systemic (itraconazole or fluconazole) in patients with head and neck involvement, and sensitization (specific IgE) to Malassezia spp.</td>
</tr>
</tbody>
</table>

Treatment

Anti-inflammatory treatment is necessary, even in subclinical inflammation, in order to restore the balance in the inflamed skin as soon as possible.
Although eczema flare-ups tend to heal spontaneously within 1-2 weeks, all flare-ups need to be treated; because, if disregarded, they tend to become subintranent and spread out. Treatment must be adapted to the characteristics of the patient and the lesions they present and, hence, we will distinguish various situations with their corresponding treatment (19).

In mild forms (limited to 4 - 6 sites), anti-inflammatory treatment should be topical (Tables V and VI) and corticosteroids are the first choice, although calcineurin inhibitors are an option in case of subacute lesions.

In severe forms (with a significant extension or resistant to topical treatment, or with intense involvement of particularly important areas, such as the hands or face), it is necessary to maintain topical treatment and to add systemic treatment: oral corticosteroids, cyclosporine, methotrexate, azathioprine, myophenolate mofetil or phototherapy. Since January 31st, 2020, a biological drug, dupilumab, is also approved, initially only for children over 12 years of age, but from December 2nd, 2020 also for those over 6 years of age, which has shown results in children similar to those found in adults (20).

Microbial colonization of lesions and superinfection (Staphylococcus Aureus throughout the body and Malassezia furfur in head and neck lesions) seem to play a role in exacerbating flares and thus, justify the association of antimicrobials when these situations take place.

Treatments used

Topical corticosteroids are the first-line anti-inflammatory treatment in all phases of AD, but especially in the acute exacerbations. Except in chronic forms, where high-potency corticosteroids, such as clobetasol propionate are necessary, in the rest, low and medium-potency corticosteroids, such as clobetasone (low potency) or methylprednisolone aceponate (medium-high potency), usually suffice.

Calcineurin inhibitors (tacrolimus and pimecrolimus) are the alternative to topical corticosteroids and, more commonly nowadays, they are used in association to them so as to reduce their adverse effects. Tacrolimus 0.1% has an anti-inflammatory potency similar to that of a medium-potency corticosteroid and is clearly superior to pimecrolimus 1%.

Crisaborole 2% is an inhibitor of phosphodiesterase 4, mainly 4B, which acts by reducing the release of pro-inflammatory cytokines, such as: TNF, IL12 and IL23. It has been approved for children over 2 years of age by the EMA (European Medicines Agency, March 27th, 2020), although it does not yet have the mandatory "therapeutic positioning report". Its use is restricted to a maximum of 40% of the body surface and, until now, its superiority against topical corticosteroids or calcineurin inhibitors has not been demonstrated.

Other topical treatments that have demonstrated their usefulness, as adjuvants in the treatment of pruritus, are: doxepin (antihistamine), topical agonists of cannabinoid receptors, topical antagonists of l-opioid receptors or topical anesthetics, but they are NOT to be routinely recommended given their adverse effects, but also because its use is not approved for AD.

Systemic corticosteroids (at initial doses no greater than 0.5 mg/kg/day) produce rapid, albeit temporary, improvements with prompt relapses, which is why they should be used in acute exacerbations or when a quick response is required. “Corticophobia” is relatively frequent, both among doctors and patients, which requires pausing to explain the advantages and disadvantages of these drugs, as well as careful managing, clearly specifying the dose and duration.

Phototherapy (narrow band ultraviolet B and ultraviolet A1) is only authorized from 12 years old onwards, but it is very useful, although the need to travel and time schedule difficulties are an obstacle, sometimes insurmountable.

Cyclosporine is the only drug with authorized indication in Spain and the fastest acting one. Its efficacy is 53-95%, but this efficacy is limited by its medium and long-term side effects (fatigue, gingival hyperplasia, increased blood pressure, hypertrichosis, kidney failure, etc.).

Azathioprine or methotrexate, both of them used off-label in severe AD, are drugs with theoretically similar efficacy (26-39% and 42%, respectively), which are indicated in severe cases of AD. They are slow acting (it takes more than a month for improve-
Seborrheic dermatitis

Introduction

Seborrheic dermatitis is a common eczema, self-limited in childhood and with a chronic and relapsing course in adults.

Seborrheic dermatitis (SD) is a common eczema, with two clinical forms, the one found in the infant and that of the adult. The first is self-limited to the first 3 months of life; while, the second is chronic and, although it can present in puberty, its frequency is highest during the fourth to sixth decades of life. The latter affects men more than women.

Etiopathogenesis

There is a relationship between seborrheic dermatitis and: overproduction of sebum (seborrhea), alterations in its composition and commensal yeasts of the genus Malassezia (Pityrosporum).

The etiopathogenesis is not fully known, but there is a relationship with: overproduction of sebum (seborrhea), alterations in its composition and commensal yeasts of the genus Malassezia (Pityrosporum). In babies, sebum is produced for a few weeks after birth, and the adult form of SD does not develop before puberty, supporting a role of androgens in the activation of the sebaceous glands. However, patients with SD may have normal sebum production, and those with excessive sebum production usually do not have SD. Malassezia Furfur and other species can be isolated from SD lesions, including infant SD, but there is no relationship between the number of yeasts and the severity of SD (unaffected skin may have a microorganism load similar to that of lesions). Anyhow, with antifungal treatment, skin lesions improve and, these increase again when SD relapses. It has also been discovered that a major component of the resident skin microflora, Propionibacterium acnes, is greatly diminished in SD; thus, it could be associated with an imbalance of the microbial flora.

Seborrheic dermatitis of the infant

It usually begins a week after birth and can persist for several months. Initially, they are greasy scales, attached to the vertex and anterior fontanel, which can spread to the entire scalp (cradle cap) and face (Fig. 6). Lesions in armpits, inguinal folds, neck, and retroauricular folds are usually more inflamed, although well defined. Lesions may become superinfected by Candida and, more rarely, by group A Streptococcus. A widespread rash of psoriasiform-appearing erythematous-scaly lesions may develop after an intensely inflammatory or superinfected SD, especially in the diaper area.

Seborrheic dermatitis in adults

In the scalp, the lesions are predominantly located in the parietal and vertex regions, but also in the occipital region, with a quite diffuse pattern. On the face, the lesions are located symmetrically in: nasogenial folds, eyebrows and retroauricular folds, but also in: the forehead, on the implantation edge of the scalp, in the glabella region and, occasionally, on the back of the neck, and also on the hairline. The
lesions are not usually infiltrated and the scaling is fine and loose. Lesions on the trunk are located in the pres- 
ternal area (in men) and in the center 
of the back where they tend to adopt a petaloid morphology. They are rarer in the armpits. Flare-ups lasting 1-2 
weeks are more or less recurrent and they are frequently related to stress. Pruritus is usually moderate, but may 
be severe, and folliculitis (Pityrosporum) and inflammation of the mei-
bomian glands in the palpebral tarsus (meibomitis) may be seen.

Differential diagnosis

The main differential diagnosis of sebor-
heic dermatitis is with atopic dermatitis.

Infantile SD differs from atopic dermAtitis by: its earlier onset, the different distribution pattern and the 
absence of itching, irritability and 
insomnia. Irritant diaper rash is limited 
to the diaper area and usually does not affect the folds. Yeast (Candida) 
diaper rash preferentially affects the 
folds, with occasional fissuring, and 
satellite lesions are usually present 
around the main lesions. In inverted psoriasis of the diaper area, the lesions are more infiltrated and demarcated, 
and the presence of typical lesions in 
other locations aids in the diagnosis.

Pityriasis amiantacea (thick asbes-
tos-like flakes which adhere to strands 
of hair on the scalp, in an irregular 
shape) which could be confused with 
cradle cap, occurs in atopic dermatitis and psoriasis.

Other rare disorders, which must 
also be considered in the differential 
diagnosis, especially in treatment-
resistant forms, are: Langerhans cell histiocytosis, acerdermatitis enteropa-
this, and Leiner’s disease. The latter, 
previously considered the maximal 
expression variant of infantile SD, is 
now considered erythroderma in the 
context of underlying immunosup-
pression.

Another differential diagnosis, to 
be considered especially in prepubertal children, and primarily in black ethni-
city, with scaling of the scalp, is tinea capitis (due to Trichophyton tonsurans).

When SD is extensive and severe in 
adults, HIV infection should be ruled 
out. The main differential diagnosis is 
with psoriasis of the scalp. In this case, 
the lesions are erythematous plaques 
covered by whitish and adherent scales, 
especially in the occipital and temporal 
regions.

Pityriasis simplex (dandruff) is defined as a diffuse, more or less 
intense, flaking of the scalp and beard, 
but without significant erythema or 
irritation. This common disorder can 
be considered the mildest form of seborrheic dermatitis of the scalp.

Treatment

Washing the lesions with a 2% ketocona-
ZOLE shampoo and the application of an 
emollient cream suffice in most cases.

Both, infantile and adult forms of 
SD satisfactorily respond to washing 
with mild shampoos and application of 
emollients. Ketoconazole (2%) or 
ciclopirox olamine cream or 
shampoo is indicated in more extensive or 
persistent cases. In acute phases, 
short courses of low-potency topical 
corticosteroids or calcineurin inhibi-
tors (although not approved for this 
indication) can be used to suppress 
inflammation.

The role of the Primary Care 
pediatrician

The role of the Primary Care pedia-
trician is to recognize the typical and 
less typical forms of both diseases, in 
order to be able to treat mild or mode-
rate forms, and refer the most serious 
forms or those that do not respond to 
treatment to the specialist. In the case of 
AD, he plays a fundamental role in 
the prevention of flare-ups, in the 
education of patients and their families 
(should not be forgotten that it is 
often a familial disease) as well as in 
screening for other diseases associa-
ted with AD (asthma, environmental 
allergy, etc.).

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the opinion of the authors.

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Recommended bibliography


It summarizes the current knowledge on the etiopathogenesis of atopic dermatitis.


They both reflect the current European consensus in the treatment of atopic dermatitis.


It highlights how things are currently being done and what has to be improved.

Clinical case

11-year-old patient who has noticed for months, the appearance of cracks in the 1st and 2nd toes of both feet. The examination is shown in figure 7.

Figure 7.

Accreditation quiz

The Accreditation Questionnaires for FC topics can be done at “On line” through the web: www.sepeap.org and www.pediatriaintegral.es.

To obtain the single continuous training accreditation from the accreditation system for health professionals for the entire national health system, 85% of the questions must be answered correctly. The accreditation questionnaires on the different issues in the journal may be carried out during the period stated in the online questionnaire.
Accreditation quiz

Subsequently, the following accreditation quiz of Pediatría Integral collects questions on this topic, which must be answered online through the website: www.sepeap.org. In order to obtain certification by the Spanish “formación continuada” national health system for health professionals, 85% of the questions must be answered correctly. The accreditation quizzes of the different numbers of the journal may be submitted during the period indicated in the “on-line” quiz.

Atopic dermatitis and seborrheic dermatitis

1. Which of the following is NOT a minor criterion for atopic dermatitis (AD)?
   a. Dennie-Morgan’s fold sign.
   b. Keratosis pilaris.
   c. Very pale skin
   d. Wool intolerance.
   e. White dermographism.

2. What should be DISCARDED in the event of recurrent (subintrantr) dyshidrotic episodes of the feet?
   a. The application of topical corticosteroids.
   b. Food allergy.
   c. Candida superinfection.
   d. Staphylococcus Aureus superinfection.
   e. An interdigital ringworm.

3. What is the INITIAL treatment in an acute flare of atopic dermatitis on the face?
   a. Cold compresses with chamomile.
   b. Clobetasol propionate applied 2 times a day.
   c. Oral antihistamines.
   d. All of the above.
   e. None of the above.

4. What DATA suggests seborrheic dermatitis in a 3-month-old infant with lesions on the face?
   a. The presence of Pityriasis amiantacea.
   b. Intense itch (scratching lesions).
   c. Elevation of serum IgE concentrations.
   d. Involvement of the nasogenian folds.
   e. Exudative lesions.

5. What is the ROLE of the Primary Care pediatrician in atopic dermatitis?
   a. Recognizing the disease.
   b. Educating patients and parents.
   c. Preventing the appearance of flares.
   d. All of the above.
   e. None of the above.

6. What DISEASE does this patient have
   a. Acute atopic dermatitis.
   b. Dyshidrosis
   c. Juvenile plantar dermatitis.
   d. Allergic contact eczema.
   e. Interdigital *tinea pedis*.

7. What could be the recommended TREATMENT?
   a. Cold compresses with 1:10,000 potassium permanganate, applied 2 times a day.
   b. Low potency corticosteroids, applied 2 times a day.
   c. Ointments containing 10% urea, applied 1-2 times a day.
   d. High tolerance moisturizers.
   e. Ointments that combine corticosteroids and antibiotics.

8. What is the PROGNOSIS?
   a. Persistence throughout his life.
   b. Spontaneous healing after the age of 13-14 years.
   c. Flare-up episodes throughout his life.
   d. Healing after an adequate treatment.
   e. To progress towards chronic eczema (neurodermatitis).

Clinical case

6. What DISEASE does this patient have