Pediatric Dermatitis Seborrhoica - A Clinical and Therapeutic Review

Abstract

Pediatric dermatitis seborrhoica (DS) is a common inflammatory disorder of infancy and adolescence distinct from atopic dermatitis. We performed a narrative review on clinical and therapeutic aspects of the disease. The prevalence varies geographically and can reach up to 10%. There is a slight male predominance. Although etiopathology is not well known, both endogenous and exogenous factors contribute. Skin microbiome and its interaction with sebaceous gland function is crucial. The inflammatory pathways include innate immune function and skin barrier disturbances. *Malassezia* spp. and certain bacteria are increased in lesional skin. DS develops in different clinical subtypes, from localized cephalic to disseminated disease with a risk of erythroderma and eczema herpeticatum. Treatment consists of skin care and topical and rarely systemic medical therapy. Cornerstones of treatment are antifungals and mild corticosteroids. Targeted treatment is on the horizon. Pediatric DS is a common disorder important in the differential diagnosis of skin problems in infants and and children. Due to better understanding of its pathogenesis, new treatment options are developed.

Keywords: *Adolescents, dermatitis seborrhoica, infants, pathogenesis, treatment*

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Introduction

Dermatitis seborrhoica (DS) (syn. seborrheic dermatitis, seborrheic eczema) is an inflammatory dermatosis of the erythemato-squamous type, which occurs in infants and adults. The term was coined by Paul Gerson Unna in 1897, who differentiated DS (also known as Morbus Unna) from other eczematous skin diseases. He also provided a detailed description of the histopathology.[1] Even though DS belongs to the top 10 skin disorders of infancy and adolescence, research is sparse compared to atopic dermatitis or psoriasis.

Regions with the highest prevalence of SD are Sub-Saharan Africa and North America, while Central Asia and Eastern Europe show the lowest prevalence. There is a slight male predominance.[2] In contrast to atopic dermatitis, pruritus in DS is not a major symptom. This review will focus on pediatric DS only.

Pediatric seborrheic dermatitis – clinical symptoms, epidemiology, and complications

Children present with lesions located on the scalp, face, and folds ("seborrheic" areas). In the pediatric population, two clinical forms of DS have been described: infantile DS and adolescent DS (overlapping the adult form). Infantile DS has been frequently reported in the 1960s and 1970s, with an important decrease in cases after the year 2000, possibly due to hygienic measures that reduce the colonization with *Malassezia* (*Malassezia globosa* and *Malassezia restricta*), predominating the forms of manifestation at the level of the scalp ("milk crusts") and the eyebrow region.[3] The prevalence of DS in children is reported to vary between 4.6% and 10%.[4,5] In contrast, immunocompromised patients in general have an incidence of DS of up to 33%, while the incidence increases to 40%–80% in HIV-positive individuals.[6,7]

The combination of recalcitrant diarrhea, malabsorption, wasting, and recurrent infections with severe DS is called erythroderma desquamativum, or Leiner-Moussous disease. This is a pediatric emergency, and patients need to be hospitalized.[8] A rare complication of DS is eczema herpeticum.[9]

Pathogenesis of seborrheic dermatitis

The pathogenesis of DS is unknown, but changes in the skin microbiome seem to

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be crucial. In contrast to atopic dermatitis, lesional DS skin samples show a significantly increased bacterial load and diversity. *Enhydrobacter*, *Micromonospora*, and *Leptotrichia* load is increased in DS.[10] In a study on facial DS, *Staphylococcus* spp. was significantly more abundant (35.9%) than in controls (8.4%, *P* < 0.001). Furthermore, there is an increased relative abundance of *Cutibacterium* spp. and a decreased relative abundance of *Streptococcus* spp.[11] *Corynebacterium* spp. may also be involved.[12]

Skin microbiome can be influenced by clothing habits and protective masks (e.g. during the COVID-19 pandemic).[13,14]

An important intrinsic factor for DS is the maturation of seborrheic glands. This explains why DS typically develops after puberty (adolescent form). In infantile DS, seborrheic glands are stimulated by maternal sex hormones. Sebum amount and composition are critical factors.[1,3]

Malassezia spp. – commensal lipophilic yeasts – are the most abundant fungi on human skin, especially in sebum-rich areas.[15] Seventeen different species have been isolated from human and animal skin so far. Hydrolysis of sebum by *Malassezia* results in skin irritants, which are activating the inflammasome and innate skin immune system.[16] These yeasts are capable of inducing inflammasome activation and subsequent interleukin (IL)-1β secretion in human keratinocytes. In particular, IL-4, IL-8, and IL-17 are upregulated in response.[17] Through toll-like receptor (TLR)-2 stimulation, IL-8 production is increased. Inflammasome activation causes an influx of neutrophils and lymphocytes. That stimulates the release of Th2-cytokines such as IL-4, IL-5, IL-6, IL-9, IL-13, and IL-25.[18]

Transcriptomic analysis of dandruff in adults suffering from DS demonstrated an upregulation of genes coding interleukin-1 receptor antagonist gene (IL-1Ra), IL-8, and S100A 8, 9, and 11.[19]

DS leads to an impaired skin barrier function as measured by increased transepidermal water loss (TEWL) and skin roughness.[20] Seasonal climate variations can aggravate DS. Dupilumab used in atopic dermatitis is another exogenous aggravating factor for DS.[21]

A recent study evaluated micro-ribonucleic acids (miRNAs) in the DS of elderly males. The expression of hsa-miR-6831-5p and hsa-miR-7107-5p was downregulated. Upregulation was noted for hsa-miR-20a-5p, hsa-miR-191-5p, hsa-miR-127-3p, hsa-miR-106b-5p, hsa-miR-342-3p, and hsa-miR-6824-5p. The targets of these miRNAs are pathways involved in cell proliferation, cell cycle, apoptosis, and immune regulation.[22] Data for pediatric DS is not available yet.

There is no clear genetic predisposition for the disease, and in particular no specific findings. However, certain human leucocyte antigen (HLA) alleles, such as A * 32, DQB1 * 05, and DRB1 * 01 may be associated with an increased risk for DS.[18]

Histopathology and dermoscopy of seborrheic dermatitis

In clinical practice, a skin biopsy is rarely necessary for diagnosis. Histopathologic findings are nonspecific and include epidermal spongiosis (in the acute phase), hyperplasia, and hyperkeratosis. There is a mild superficial perivascular and perifollicular inflammatory infiltrate, consisting of lymphocytes and histiocytes.[23] These findings are probably a result of increased proteolytic activities in the epidermis due to activated cathepsin S and serine protease and diminished kallikrein-related peptidase (KLK) 5.[24]

Dermoscopy of the lesional skin shows yellowish scaling, branching and atypical thin arborizing vessels, and distinct unstructured white areas interrupted by honeycombed pigmentary networks.[25,26]

Clinical subtypes of pediatric seborrheic dermatitis

DS develops in infants, usually about 2 to 3 weeks after birth and lasts up to 6 months.

Clinically, four subtypes are described, differentiated by the appearance and location of the lesions. Most often the lesions are observed on the scalp and genital region (so-called DS with bipolar appearance).

Infantile DS on the scalp (cephalic DS, cradle head, pityriasis capitis, "milk crusts") is diagnosed in the age group 3 weeks–4 months, with a peak incidence at 3 months of life.[3] It is characterized by the presence of thick, yellow scales with a "greasy" appearance, adhering to the scalp in the fronto-parietal region, often associated with similar lesions in the eyebrow region, on the ciliary margin, nasolabial grooves, cheeks, retro-auricular, or even distant lesions (navel, diaper area, or folds). In the cephalic area, the erythema is discrete, in the other areas the erythema is more intense [Figures 1 and 2].[27]

Very rarely, this type of DS can progress to erythroderma and requires further investigation for possible immune deficiency. Pruritus is absent, being a clinical element differentiating it from atopic dermatitis.[28] It may be associated with pityriasis (tinea) amiantacea, a variety of inflammatory scalp reaction pattern also seen in atopic dermatitis or scalp psoriasis.[29]

Pediatric facial DS is defined by the presence of lesions only on the face; it is rare in monopolar form, and most often associated with scalp lesions. The differential diagnosis of this form must be made with atopic dermatitis, difficult at the age of 2–6 months when the two conditions can follow each other; DS regresses and atopic dermatitis

evolves [Figure 3].[28] Recent studies have noted, statistically, that the presence of DS, especially of the cephalic type, is more frequent in children who come from families with a history of atopic dermatitis or asthma.[30]

Infantile DS of the intertriginous type is characterized by lesions with an erythematous appearance, with fine scales, very well demarcated, with a homogeneous appearance, located in the armpits, navel, neck region, popliteal space, and the crook of the arm [Figure 4].

Genital infantile DS ("diaper type") has a particular appearance, discretely scaly, predominantly erythematous, and skin maceration-type damage.

The disseminated type is less common than the other subtypes [Figure 5].

The evolution of infantile DS is favorable, with complete resolution in 5–6 months, even in the absence of treatment;

chronic evolution beyond the age of 2–3 years has rarely been described.[31]

Comorbidities of DS in adolescence are acne, obesity, metabolic syndrome, insulin resistance, and diabetes mellitus.[31,32] In infants, there is no association to comorbidities known, although DS and atopic dermatitis may develop in the same individual.

Differential diagnoses of pediatric seborrheic dermatitis

Differential diagnoses in infants are napkin dermatitis, atopic dermatitis, psoriasis, Darier's disease, keratosis lichenoides chronica, Langerhans cell histiocytosis, etc., [Table 1].[33-35] In adolescents, in addition to face and neck dermatitis, lupus erythematosus, rosacea, contact dermatitis, and hidradenitis suppurativa (inverse acne) need consideration.[36,37]

Treatment of pediatric seborrheic dermatitis

There is no consensus on treatment recommendations for infantile DS.[30] The evolution of DS is favorable in most cases, sometimes residual hypopigmented macules may persist for several weeks. In colored ethnic skin, hypo- and hyperpigmentation can develop as a consequence of tissue inflammation. Infantile DS does not necessarily predispose to adolescent or adult DS. The relationship between psoriasis and atopic dermatitis is controversial.[38]

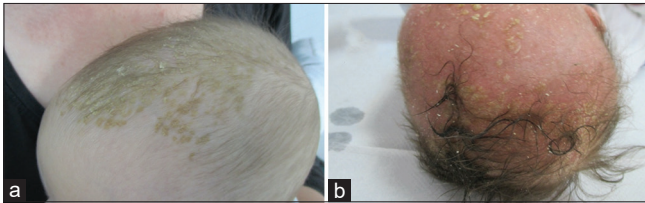There is recent progress in topical and systemic treatment options in DS, but unfortunately almost all

Figure 1: Infantile cephalic DS (a) Milder type with greasy, yellowish scales (b) more severe type with thicker scales and erythema

Figure 3: Facial DS in an adolescent girl with symmetrical facial involvement
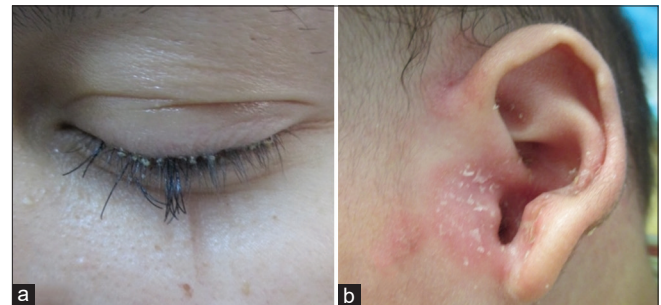

Figure 2: Infantile DS (a) Lid margin involved (b) External ear and preauricular area involved

Figure 4: Intertriginous infantile DS (a) On the neck folds (b) In the napkin area

Table 1: Differential diagnosis of pediatric DS

Diagnosis	Hallmarks
Langerhans cell histiocytosis	May present with extracutaneous symptoms such as involvement of liver, spleen, and bone marrow. SD has no extracutaneous manifestations.
	Diagnosis needs confirmation by histology. The inflammatory infiltrate contains various proportions of Langerhans cells with "coffee-bean" cleaved nuclei and eosinophilic cytoplasm. Positive immunostaining for CD1a and CD207 (langerin) are essential for a definitive diagnosis.
Darier's disease	Caused by germline mutation with variable inheritance (X-linked, autosomal dominant, autosomal recessive).
	The characteristic mucocutaneous triad consists of dysplastic nails, oral leukoplakia, and filigree, reticular skin pigmentation. The triad and bone marrow failure are the major symptoms of Darier's disease in children.
	In children, multiple extracutaneous symptoms may be present before mucocutaneous manifestations. These include pulmonary arteriovenous malformations, pulmonary fibrosis, liver disease, stenosis of the urethra, esophagus, or lacrimal ducts, avascular necrosis of the hips and/or shoulders, malignancies among others.
	Total lymphocyte flow fluorescent *in situ* hybridization (FISH) telomere lengths less than the first percentile for age have a sensitivity and specificity of 97% and 91% for differentiating patients from unaffected relatives. Other molecular biological techniques are Southern blot and real-time polymerase chain reaction (PCR).
Keratosis lichenoides chronica	The typical clinical symptoms of this rare dermatosis are DS-like facial eruptions, along with violaceous, papular, and nodular lesions on trunk and extremities and trunk that are arranged in a linear and reticulate pattern.
Atopic dermatitis (AD)	AD is a common inflammatory disorder. The leading symptom is pruritus. Clinical features are age-dependent. Infants present with acute, poorly defined itchy lesions consisting of papules and vesicles, often associated with serous exudate and crusts. Involved are face, trunk, the extensor surfaces of the limb, and sometimes the napkin region. In childhood, dry skin and lichenified papules and plaques affecting flexor surfaces is more common. Facial involvement becomes less frequent and is concentrated in the perioral and periorbital area. The diagnosis is made clinically and by medical history including family history of atopic disorders.
Psoriasis	Psoriasis is another important inflammatory skin disease. It is less frequent among infants and children compared to AD. Facial involvement and affection of the napkin area are a feature of infants and younger children. Napkin psoriasis can become macerated. Itch is not as intense as with AD. The diagnosis is made primarily clinically. Histology is rarely necessary.
Napkin dermatitis	The disease is seen frequently among infants. Irritant contact to urine and feces, and colonization by candida yeasts are the major pathogenetic factors. Secondary colonization by bacteria or mixed microbiology develops when the disease is diagnosed with delay and appropriate hygienic and medical approaches have been neglected. Diagnosis is made clinically.
Biotinidase deficiency (BD)	BD is a rare autosomal recessive disease caused by mutations in the *BTD* gen. Infants develop DS but more important are variable neurological manifestations (seizure, sensorineural hearing loss, ataxia, spastic paresis, visual impairment), apnea or stridor, infections, acidosis, and alopecia.
	Diagnosis is based upon clinical presentation and enzyme activity in serum or plasma. Diagnosis can be confirmed by genetic testing.
Riboflavin deficiency (RD)	RD, also known as ariboflavinosis, is seen in case of insufficient dietary intake of riboflavin. Vegan diet of the mother can cause RD in infants. Drugs and alcohol misuse are factors contributing to RD in adolescents and adults. Gastrointestinal and liver disorders and anorexia nervosa are other possible underlying diseases.
	The typical clinical manifestations are DS in association with cheilosis, angular stomatitis, glossitis, and severe anemia with erythroid hypoplasia. The diagnosis can be confirmed by laboratory investigations. Erythrocyte glutathione reductase which is dependent on the riboflavin cofactor FAD is considered the gold standard for assessment of riboflavin long-term status.

randomized controlled trials have been conducted in adults only [Table 2].[39-52]

Topical treatment is the gold standard in the pediatric population. Shampoos containing zinc pyrithione or selenium disulfide are used for scalp DS and dandruff.[53,54] Topical ketoconazole 1% in various pharmaceutical preparations can be recommended for 10–15 days. No significant systemic absorption has been demonstrated in neonates, but higher concentrations should be avoided.[55] Alternative treatment options include topical 1% ciclopirox or topical 2% miconazole. Sertoconazole 2% is more irritating and not recommended for infants.

Class 1 or 2 topical steroids such as hydrocortisone 1% monotherapy or in combination with ketoconazole, especially for DS within skin folds may be a good choice to defeat erythema.[53,54]

Emollients are important for the reconstitution of the disturbed epidermal barrier function. Daily application of baby shampoo combined with an emollient applied thereafter is ideal for long-term treatment.

Urea-based keratolytics of up to 10% are used for scalp lesions in adolescents. Salicylic acid 10% can be a temporary option in adolescents, but is contraindicated in infants due

Table 2: Randomized controlled trials (RCT) for seborrheic dermatitis (2010-2023)

Ref	Trial type	n	Drugs	Outcome	Age
Topical treatment					
[39]	IIa, DB, multicenter	226	Topical roflumilast foam 0.3% vs. vehicle foam 1x/d	IGA clear or almost clear or 2 grade improvement from baseline at week 8: 73.8% (verum) vs. 40.9% (vehicle); mean reduction of WI-NRS at week 8: 59.9% (verum vs. 36.6% (vehicle)	≥18 yrs
[40]	RCT, DB, maintenance therapy, single- center	48	1% Selenium disulphide (SeS2)-shampoo after 2 weeks topical corticosteroids + salicylic acid1x/d vs. vehicle	Relapse occurred in 16.7% in the SeS2 and in 54.2% in the vehicle group at week 8; absence of pruritus at week 10: 76.2% with SeS2 and 57.1% with vehicl	≥18 yrs
[41]	RCT, DB	60	Topical pimecrolimus 1% cream or sertaconazole 2% cream twice daily for 4 weeks	Pimecrolimus caused better improvement on day 14 and 28; relapse rate was significantly lower with pimecrolimus 4 weeks after cessation of treatment	≥18 yrs, facial SD
[42]	Multicenter RCT, DB, maintenance therapy	114	Topical tacrolimus 0.1% or ciclopiroxolamine 1% cream 2 times a week	Disease-free duration significantly longer with tacrolimus; relapse rate within 24 weeks: 22.2% with tacrolimus after a median delay of 91.5 days vs. 40.4% with ciclopiroxolamine after a median delay of 27 days	≥18 yrs, facial SD
[43]	RCT, single- center	30	Topical pimecrolimus 1% cream either twice daily for 2 weeks followed by 2 weeks of pimecrolimus for 4 weeks moisturizer OR pimecrolimus 2 weeks twice daily followed by pimecrolimus 1x/d for 2 weeks OR pimecrolimus twice daily for 4 weeks	After 4 weeks significantly better improvement with pimecrolimus twice daily	≥18 yrs, facial SD
[44]	RCT, single- center	48	Topical nicotinamide (NCT) cream 4% 1x/d, vs. placebo for 3 months	Erythema, scaling, and infiltration were reduced by 75%with NCT vs. 35% with placebo after 3 months	≥18 yrs
[45]	RCT, DB, multi-center	274	2% miconazole nitrate shampoo vs. 2% ketoconazole shampoo twice a day for 4 weeks	Equipotent improvement of CGIs, SSSD, and PGIs by both shampoos	≥18 yrs
[46]	RCT, DB, single- center	60	Topical sertaconazole 2% cream vs. pimecrolimus 1% cream twice daily for 4 weeks	Patient satisfaction after 4 weeks: 90% sertaconazole vs. 80% pimecrolimus	≥18 yrs
[47]	RCT, DB, single- center	138	Topical sertaconazole 2% cream vs. hydrocortisone 1% cream twice daily for 4 weeks	Patient satisfaction after 4 weeks: 85.1% sertaconazole vs. 76.9% hydrocortisone	≥18 yrs
[48]	RCT, DB, single- center	156	Topical sertaconazole 2% cream vs. metronidazole 1% gel twice daily for 4 weeks	Patient satisfaction after 4 weeks: 87.1% sertaconazole vs. 56.4% metronidazole	≥18 yrs
[49]	RCT, DB, single-center	128	Topical sertaconazole 2% cream vs. clotrimazole 1% cream twice daily for 4 weeks	Patient satisfaction after 4 weeks: 87.6% sertaconazole vs. 50% clotrimazole	≥18 yrs
[50]	RCT, DB, multi- center, proactive	75	Topical 0.1% tacrolimus once a week, twice a week, or vehicle twice a week, for 10 weeks	Topical pimecrolimus twice a week had the lowest relapse rate	≥18 yrs
[51]	RCT, SB, single- center	30	Topical tacrolimus 0.1% vs. hydrocortisone 1% ointment twice daily for 4 weeks	Topical pimecrolimus was significantly less often applied due to lack of symptoms compared to hydrocorticone	≥18 yrs facial SD
[52]	RCT, DB, single- center, split-side	75	Topical moisturizer containing 0.025% licochalcone or 1% hydrocortisone twice daily for 2 weeks	The cure rate of the moisturizer containing 0.025% licochalcone and 1% hydrocortisone group was 90% and 92% after 2 weeks	2 weeks to 1 year
[54]	RCT, single-center	326	Treatment phase (4 weeks): either clobetasol propionate shampoo 0·05% (CP) combined with Ketoconazole (KC) twice weekly OR CP twice weekly alternating with KC twice weekly	Combined treatment with CP + KC reduced erythema (85·4% and 86·5%) significantly better than KC (66·3%), reduced scaling (86.6% and 80.5%) than KC (63.8%), and reduced pruritus significantly more (86.6% and 89%) than KC (76.3%) after 4 weeks.	≥18 yrs

Contd...

Ref	Trial type	n	Drugs	Outcome	Age

Table 2: Contd...

Ref	Trial type	n	Drugs	Outcome	Age
			OR CP 4 times weekly alternating with KC twice weekly. Maintenance phase (4 weeks) KC once weekly. Follow-up phase: only mild shampoo	KC maintenance therapy was more effective after KC or CP (twice weekly) in contrast to CP 4 times a week+KC twice a week. There was no significant rebound at week 12 irrespective of previous treatment	
Oral treatment					
[55]	RCT, single- center	45	Isotretinoin 10 mg q.o.d. vs. anti- seborrhoical topical treatment for 6 months	Sebum reduction after 3 and 6 months significantly greater with isotretinoin; skin and lip dryness were a common adverse event with isotretinoin	≥18 yrs
[56]	RCT, single- center	68	Itraconazole 200 mg/daily or placebo for 1 week and then for the first 2 days of every month for the following 3 months	SDASI improvement after 4 months: 93.1% (itraconazole) vs. 53.6% in the placebo group; relapse rate significantly lower in the itraconazole group	≥18 yrs

Legend: CGIs, Clinical Global Impressions; DB, double-blind; IGA, investigator global assessment; q.o.d., every other day; PGIs, Patient Global Impressions; SB, single-blinded; SD, seborrheic dermatitis; SDASI, Seborrheic Dermatitis Area Severity. Index; SSSD, Symptom Scale of Seborrhoeic Dermatitis; WI-NRS - Worst Itch Numeric Rating Scale; KC: Ketoconazole?

Figure 5: Disseminated infantile DS, a differential diagnosis to pityriasis rosea

to possible systemic toxicity. In infants, a combination of dicaprylyl carbonate, polydimethylsiloxane, and silicones in fluid (Loyon; G. Pohl-Boskamp GmbH and Co. KG, Hohenlockstedt, Germany) is a safe option for cradle cap.[56]

In a systematic review of oral medications for DS, Gupta *et al*. (2014) have identified randomized controlled trials in adult patients only for ketoconazole, fluconazole, and terbinafine.[57] Oral medication with antifungals is rarely necessary in pediatric DS. The dosage in adolescents >40 kg is 250 mg of terbinafine/d, 5 mg/kg/d of itraconazole, and 5–6 mg/kg/d of fluconazole. The average duration of treatment is 4–-6 weeks.[58]

The following approaches are no longer recommended based on the principles of evidence-based medicine:
• oral biotin (lack of efficacy)
• topical olive oil (favors *Malassezia spp.*)
• topical flumethasone (risk of skin atrophy and systemic absorption)

• topical ketoconazole 2% for infants (systemic absorption).[57]

There are some new drugs on the horizon with a potential for use in DS. A possible new topical treatment option for severe facial DS in adolescents consists of tapinarof 1% ointment. Tapinarof is a first-in-class, topical small molecule aryl hydrocarbon receptor (AhR) agonist.[59] Another drug in the therapeutic pipeline for facial DS in adolescents is 1.5% ruxolitinib cream. Ruxolitinib is a Janus kinase (JAK) inhibitor selectively targeting JAK1 and JAK2.[60] Apremilast, an oral selective phosphodiesterase-4 (PDE-4) inhibitor, was used successfully in a case of adult recalcitrant DS.[61] The topical PDE-4 inhibitor roflumilast has been investigated for adult seborrheic dermatitis in a randomized controlled trial with success (see [Table 2])[39]

A summary of established treatment options for pediatric cases are given in Table 3.[45-49,56]

Conclusions

DS is a common inflammatory dermatosis of infants and adolescents. Clinical presentation, differential diagnosis, comorbidities, and treatment differ from adult DS. In infants, a bipolar appearance is commonly seen. Pruritus is absent. Recent investigations have demonstrated peculiar changes in the skin microbiome. This could open new directions for treatment and prevention in the future.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Table 3: Treatment options in pediatric DS

Age group	Indications	Severity	Treatment(s)	References
Infants	*Scalp*	Mild	Selenium sulfide 2.5% shampoo; zinc pyrithione 1%-2% shampoo	[61,62]
			Dicaprylyl carbonate/	[56]
			polydimethylsiloxane/silicones fluid	[61,62]
			ciclopirox 1% shampoo, 0.77% gel; ketoconazole 1%	
		Moderate/severe	Hydrocortisone 1% liniment or 0.1% lotion	[61,62]
	Non-scalp	Mild	Ciclopirox 1% cream; ketoconazole 1% cream	[62]
		Moderate/severe	Hydrocortisone 1% liniment or cream	[61,62]
Adolescents	*Scalp*	Mild	Salicylic acid 3% shampoo; tar 1-2% shampoo; selenium sulfide 2.5% shampoo; zinc pyrithione 1%-2% shampoo; ketoconazole 1-2% shampoo, 2% foaming gel, or 20 mg/g hydrogel	[61,62]
		Moderate/severe	Alcomethasone 0.05% ointment; desonide 0.05% cream	[61,61]
	Non-Scalp	Mild	Ketoconazole 2% foaming gel, or 20 mg/g hydrogel	[61,62]
			miconazole 2% cream, clotrimazole 1% cream	[61,63]
			sertoconazole 2% cream, lithium succinate/gluconate 8% ointment	[41,61]
		Moderate	Alcomethasone 0.05% ointment; desonide 0.05% cream;	[61,62]
		Severe	Itraconazole 100 mg caps; terbinafine 250 mg caps; fluconazole 50 mg caps isotretinoin 10 mg caps/d	[61,62] [52,64]

Conflicts of interest

There are no conflicts of interest.

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