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ORIGINAL ARTICLE

Diagnosis and Management of Pediatric Psoriasis: An Overview for Pediatricians

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ABSTRACT

Pediatric psoriasis (PsO) and its associated comorbidities carry physical and psychosocial burdens in children and adolescents, which can negatively impact quality of life. However, features distinguishing pediatric PsO from eczema and other common inflammatory skin diseases may not be obvious to primary care providers, which may contribute to underrecognition and misdiagnosis. Accurate diagnosis of pediatric PsO is critical for managing the physical and psychological burdens associated with this disease. This review aims to support pediatricians with enough information to confidently diagnose pediatric PsO, assess associated physical and mental health comorbidities, and recommend first-line treatment options for children with mild to moderate PsO. To accomplish this, we provide information that distinguishes the appearance and symptoms of pediatric PsO from other common pediatric skin conditions. In addition, comorbidities and some of the mental health challenges associated with pediatric PsO are reviewed to help pediatricians provide appropriate care for patients in their clinical practice.

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INTRODUCTION

Psoriasis (PsO) is a chronic, inflammatory skin disease characterized by cutaneous features, extracutaneous comorbidities, and an unpredictable course.[1,2] PsO is the second most common chronic pediatric skin disorder after atopic dermatitis (AD) and is reported to affect 0.05% to 2.15% of children,[3] compared with a 15% to 20% prevalence of eczema.[4] PsO is often mistaken for eczema because both are chronic diseases that feature red, scaly skin, suggesting that the true prevalence of pediatric PsO may be higher.[5] The mean age of onset of PsO is between 8 and 11 years, and the prevalence increases with age, estimated at 0.13% in those under the age of 2 years and 0.67% in teenagers.[6,7] Approximately 30% of adults with PsO experienced symptoms before the age of 20 years.[8]

Clinical features of PsO in infants and children are somewhat different from those of adults, which may also make distinguishing pediatric PsO from eczema more difficult. In an anonymous survey, 53.7% of pediatricians (n=95) reported being uncertain or very uncertain about their ability to diagnose pediatric PsO, despite regularly seeing pediatric patients with PsO.[1] Pediatricians who are less confident in their diagnostic ability are also less likely to perform total skin examinations, screen for relevant comorbidities, and prescribe disease-specific treatment. None of the pediatricians surveyed prescribed standard-of-care systemic immunomodulating agents (eg, methotrexate and/or cyclosporine) or US Food and Drug Administration (FDA)–approved therapies labeled for this condition (including targeted biologics or retinoids) for their patients with PsO. A French national survey of clinicians who treat children with PsO found a much lower use of severity scores and systemic treatments among general practitioners and pediatricians compared with dermatologists, thereby limiting treatment options for pediatric patients.[9] Dermatologists more frequently prescribed topical corticosteroids and vitamin D analogs for pediatric patients with PsO than general practitioners, suggesting a reluctance to prescribe or lack of awareness of preferred treatments for pediatric PsO.[10]

Early intervention in pediatric PsO can reduce the impact and burden of the disease and possibly its comorbidities, emphasizing the need for accurate and early diagnosis of pediatric PsO. This review describes the features and triggers that distinguish PsO

FIGURE 1. Common features of childhood-onset PsO include (A) scalp involvement, (B) scaling and (C) redness associated with plaques on the knees and lower legs, (D) nail pitting and onycholysis, (E) genital involvement, and hypopigmentation from plaques, as shown here in examples on the (F) legs, (G) underarm, and (H) back.

from eczema and other chronic inflammatory skin disorders in children; defines mild, moderate, and severe disease; highlights the challenges pediatricians face in the diagnosis and management of pediatric PsO; and discusses standard first-line treatment for mild to moderate pediatric PsO and emerging treatment options for moderate to severe disease.

Clinical Characteristics of Pediatric PsO

Evolving understanding of the complex characteristics of both pediatric PsO and eczema has allowed recognition of multiple subsets of both diseases, supporting the concept of these conditions as phenotypes rather than single diseases. The clinical hallmarks of pediatric PsO are sharply circumscribed, scaly plaques occurring in characteristic sites of predilection that define subtypes (Table 1 and Figure 1).[3,7,11-18] Large plaque PsO is the most common and well-recognized subset of PsO, reported in 69% to 75% of pediatric cases. These lesions typically involve the scalp, elbows, and knees.[7,11,12] Posterior auricular scale and nail pits are subtle findings that support the diagnosis.[19] Guttate (small plaque) PsO is the second most common subset, reported in 14% to 29% of pediatric cases.[20] An initial guttate presentation has been associated with greater PsO severity.[20] Streptococcal infection is a well-recognized trigger of guttate PsO,[21] which may clear after treating the infection with antibiotics. Tonsillectomy has been demonstrated to induce remission in a minority of children with guttate PsO.[22] Other sites of predilection include palms and soles (palmoplantar PsO), skinfolds (inverse PsO), and ear canals (psoriatic otitis externa), which can be isolated or seen in children with large or small plaque disease.

In pediatric patients with PsO, nail involvement occurs in 17% to 39% of cases, and scalp involvement occurs in 18% to 79% of cases.[20,23-26] Nail involvement occurs more frequently in boys, while scalp involvement is reported significantly more often in girls.[20] Nail involvement may be a sign of a more prolonged course; however, unlike adult PsO, nail involvement has not been directly linked to psoriatic arthritis (PsA).[27]

Less common PsO subtypes may be more difficult to recognize[16] and include PsO-eczema overlap, pustular, isolated palmoplantar, inverse, annular, petaloid, erythrodermic, and tinea amiantacea. Inverse PsO presents with well-demarcated, pink-to-red, often macerated plaques in the axillary, inguinal, and gluteal creases and the umbilicus[14,15] and can be confused with infectious or eczematous intertrigo.[14] Itching, irritation from sweating, and tenderness are common.

Infants with PsO often present with involvement of the face and diaper area; 26% of children with PsO have a history of diaper rash.[28,29] Plaques in this area are characteristically well demarcated and often feature marked erythema with minimal scale. Koebnerization, a diagnostic and therapeutic feature of PsO, is the tendency to develop skin lesions at sites of friction or minor skin trauma.[30] Thumb involvement, representing Koebnerization from thumb sucking, is also a common feature of PsO in infants.[31]

PsO Triggers

Factors such as infections, high body mass index, and

TABLE 1.

Clinical Spectrum of PsO[3,7,11-18]		
Subtype	**Signs/Appearance**	**Location**
Plaque		
Large plaque	• Most common subtype (69%-75% of pediatric cases) • Sharply circumscribed, erythematosquamous plaques	• Scalp, face, extensor surfaces of the elbow and knee, umbilicus, and buttocks - Scalp is frequently the first site of involvement
Small plaque (guttate)	• Second most common subtype (14%-29% of pediatric cases) • Small, round, raised plaques that are scaly with hyperkeratosis • Commonly triggered by streptococcal or viral infection - May clear after treating infection or develop into chronic PsO	• Trunk, abdomen, and back
Inverse	• Well-demarcated, pink-to-red, often macerated plaques • Itching, irritation from sweating, and tenderness are common	• Skinfolds - Axillary, inguinal, and gluteal creases and the umbilicus
Psoriatic otitis externa	• Similar to large plaque PsO	• Ear canals
Pustular		
Localized or generalized	• Less common than plaque PsO (1.0%-5.4% of pediatric cases) • Superficial sterile pustules • Often accompanied by fever	• Diffuse or localized to the fingers, palms, soles, toes, and nail beds
Annular	• Ring-shaped pustular lesions	• Can be diffuse or localized
Other		
Palmoplantar	• Plaque or pustular lesions • Scaly, red plaques or pustules with deep painful fissures	• Palms and soles
Linear	• Erythematous papules or plaques • Often accompanied by Koebnerization and Auspitz sign	• Distributed along the lines of Blaschko
PsO-eczema overlap	• Plaque or pustular lesions • PsO or eczema lesions can develop from their respective triggers	• Facial, scalp, and nail involvement
Nail	• Pitting, leukonychia, and subungual hyperkeratosis	• Nails
Paradoxical	• Develops in response to anti-TNF treatment for other skin conditions • Plaque or pustular lesions • Usually resolves after discontinuation of treatment	• Diffuse, but palmoplantar regions most often affected
Erythrodermic	• Erythema and scaling on >90% BSA • Can be accompanied by severe hypothermia and hypoalbuminemia • Extremely rare	• Diffuse

Abbreviations: BSA, body surface area; PsO, psoriasis; TNF, tumor necrosis factor.

cutaneous trauma can trigger pediatric PsO. Upper respiratory tract infection, particularly group A β-hemolytic streptococcal pharyngitis, and some drugs (eg, propranolol, antimalarials, terbinafine, and lithium as well as following withdrawal of systemic corticosteroids) are other well-recognized triggers.[11,32] Environmental exposure to tobacco smoke and stressful life events have also been associated with pediatric PsO.[33-35] Paradoxical PsO refers to an emerging subtype of PsO first recognized in adults but increasingly reported in children.[36-40] This subtype develops in patients treated with a biologic agent that blocks tumor necrosis factor (TNF). Agents that target this pathway are effective, FDA-approved medications for PsO but when used for other indications (inflammatory bowel disease [IBD] or arthritis) can trigger PsO.

Pathophysiology

Well-defined, but not mutually exclusive, inflammatory pathways distinguish plaque PsO from AD, as supported by the evolving pipeline of targeted biologic therapy. In vitro studies initially identified the helper T (TH) 1 pathway as the most important signaling pathway in the pathophysiology of PsO.[41,42] Early clinical trials that followed this discovery demonstrated that blocking TNF alpha led to significant improvement in PsO, but subsequent studies yielded even better improvements with agents that block interleukin (IL)-17 and IL-23.[43] In contrast, TH2 inflammation is the major immunologic pathway that impacts AD, as supported by successful treatment with biologic agents that block IL-4 and IL-13.[44]

Assessment of Pediatric PsO

A common assessment tool for determining PsO disease severity is total body surface area (BSA) involvement using the "rule of 9's" measurement, with adjustment of relative proportions of regions based on age.[32,45] The rule of 9's general guidelines are that the head and each arm comprise 9% of the total BSA, each leg and the front and back of the torso, respectively, each make

TABLE 2.

Features That Distinguish Pediatric PsO From Eczema[13,98-100]		
	PsO	Eczema
Mean age of onset	8-11 years old	<2 years old
Clinical morphology		
Border	Sharp	Diffuse
Scale	Coarse	Fine
Pigment change	Hypopigmentation	Hyperpigmentation
Itch	+	+++
Sites of predilection	Face, scalp, axillary, inguinal and gluteal folds, umbilicus, palms/soles, diaper area, nail pits, orbital rim	Antecubital and popliteal fossae (spares diaper area)
Associated comorbidities	High BMI, hypertension, obesity, insulin resistance, metabolic syndrome, arthritis, IBD, PsA	Chronic rhinitis, asthma, food allergy, eosinophilic gastrointestinal disease
Triggers	Friction, minor skin trauma	Viral infection
Response to corticosteroids	Less effective, rebound after discontinuation, potential worsening	Very effective
Readily available biomarkers	-	High IgE, eosinophilia
Inflammatory pathways	TH1 and TH17	TH2
Cytokine targets	IFN-γ, IL-12, IL-17, IL-23, TNF-α	IL-4, IL-13, IL-25, IL-33

Abbreviations: BMI, body mass index; IBD, inflammatory bowel disease; IFN-γ, interferon gamma; IgE, immunoglobulin E; IL, interleukin; PsA, psoriatic arthritis; PsO, psoriasis; TH, helper T; TNF-α, tumor necrosis factor alpha.

up 18%, and the genitalia make up 1%. BSA involvement of <3% is considered mild, 3% to 10% is moderate, and >10% is severe disease. BSA is a component of the Psoriasis Area Severity Index (PASI), which also includes 4-point rating scales for erythema, induration, and flaking. Payers often require PASI scores before authorizing payment for newer, more expensive medications. However, PASI scores should not be the sole assessment of disease severity. Other important factors are involvement of sites that are difficult to treat topically (face, scalp, folds, groin, nails), arthritis, and psychometric symptoms such as social withdrawal.[32,45] The Children's Dermatology Life Quality Index (CDLQI) is a validated, easily usable tool for clinical experience and psychometric properties of PsO in pediatric patients age four years to 15 years and 11 months.[46] CDLQI may be used to evaluate pediatric patients' health-related quality of life (HRQOL) and considered along with PASI scores to determine the overall burden of disease in this age group. In fact, the Joint American Academy of Dermatology–National Psoriasis Foundation (AAD–NPF) guidelines recommend that both BSA and CDLQI be used as a measure of PsO severity.[45]

Differences Between Pediatric and Adult PsO

Children may be at higher risk for missed and/or delayed diagnosis compared with adults. The key clinical differences that distinguish childhood-onset PsO from that in adults include lesion morphology, sites of predilection, and disease burden. Plaques in children can be less indurated and the scale finer without the classic silvery quality.[7] Isolated involvement of the ear canals in children may be confused with otitis externa.[11] Eyelid margins are another site of predilection that can be isolated and mistaken for other forms of blepharitis.[19] Pruritus may often be present.

Differential Diagnosis of Pediatric PsO

Diagnosing pediatric PsO can be challenging for pediatricians, as the signs may appear similar to eczema, tinea, or other inflammatory skin conditions (Table 2). Pediatric PsO is not commonly associated with asthma or allergic rhinitis, whereas these are frequently found in patients with AD or members of their family. Both pediatric PsO and AD feature erythema, induration, and scale, and both respond to treatment with topical corticosteroids, but PsO is more likely to rebound with treatment discontinuation.[47] Eczema is often most prominent in the antecubital and popliteal fossae, flexor wrists, and dorsal aspects of the hands, while pediatric PsO lesions commonly localize to the scalp, palms, soles, and extensor surfaces of the elbows and knees.[47] Furthermore, eczema typically spares the diaper area and skinfolds, while PsO commonly involves this area. Nail involvement is another feature of pediatric PsO that can support differentiation from eczema,[47] although nail pits and dystrophy can occur in eczema, especially in the setting of paronychia. Misdiagnosis of pediatric PsO as eczema is also likely related to the higher frequency of eczema compared with PsO. Lesional skin biopsy can help distinguish pediatric PsO from other skin conditions.[11] Diagnostic histologic features include epidermal thickening with elongated rete ridges, hypergranulosis, and parakeratosis, but clinically atypical pediatric PsO is less likely to exhibit psoriatic histology. The histologic features of pediatric PsO have been reported in 57.6% of infants with this suspected diagnosis.[48] The inflammatory impact on pigmentation is

another feature that distinguishes pediatric PsO from eczema, with PsO most often causing hypopigmentation and eczema most often causing hyperpigmentation.[49] This feature is most apparent and upsetting for patients with darker skin tones. PsO-eczema overlap features skin signs of both eczema and PsO but may be less responsive to topical corticosteroids. Recognizing overlap is especially important when considering options for systemic treatment.[48]

Comorbidities

Extracutaneous comorbidities associated with pediatric PsO can contribute to the physical and psychosocial burden of disease and can negatively impact quality of life. Patients with pediatric PsO are at increased risk for arthritis, IBD, Crohn's disease, hypertension, bronchial asthma, hyperlipidemia, nail disorders, and arterial hypertension than those without pediatric PsO.[6,50,51] Obesity, diabetes, and metabolic syndrome have also been more frequently observed in pediatric patients with PsO than patients without PsO, suggesting that PsO is an independent risk factor for developing metabolic comorbidities.[51,52] PsO can also coexist with vitiligo, alopecia areata, and lichen planus, further complicating optimal treatment.[53] Hypermetabolic syndrome, in which elevated resting energy expenditure leads to insulin resistance and excessive breakdown of proteins and triglycerides, has also been associated with PsO.[54]

In light of these findings, the NPF and the Pediatric Dermatology Research Alliance (PeDRA) established the NPF-PeDRA–Pediatric PsO Comorbidity Screening Initiative, which recommends regular screenings for obesity, type 2 diabetes, dyslipidemia, hypertension, IBD, arthritis, mood disorders, and substance use disorder for pediatric patients with PsO.[55] These evidence-based guidelines are targeted toward all healthcare providers treating pediatric patients with PsO to help minimize the long-term health effects of PsO.

PsO-associated symptoms negatively impact psychosocial quality of life in children, resulting in a greater risk of mood disorders than are associated with healthy patients or those with other pediatric chronic diseases such as arthritis, asthma, and diabetes.[56-58] Pediatric patients with PsO reported a higher incidence of anxiety, depression, and suicidal ideation than pediatric patients without PsO.[50,59] Children aged 5 to 16 years with PsO or AD reported the greatest impairments in HRQOL compared with other common skin conditions such as localized eczema, acne, and urticaria.[60] These patients also reported greater impairments in HRQOL than children with epilepsy, enuresis, or diabetes.[60] Pediatric patients with PsO often experience teasing or bullying due to their appearance, which can negatively impact self-esteem and lead to feelings of social exclusion.[61] Of pediatric patients with PsO, 65% reported feeling stigmatization[62] due to bullying or teasing,[63] which negatively impacted family and social relationships.

Juvenile PsA is a chronic inflammatory disease affecting the joints that occurs in some patients with pediatric PsO and can complicate disease treatment and management strategies.[64] In an analysis using pooled US claims data, the estimated prevalence of PsA in pediatric patients with PsO was approximately 2%,[65] which is lower than the approximately 30% reported prevalence in adults.[66,67] However, since patients may present with signs of arthritis before or after development of pediatric PsO, the overall prevalence of arthritis in pediatric patients remains uncertain. In 80% of pediatric patients with juvenile PsA, joint inflammation develops before onset of skin disease, and the most common age ranges for joint involvement are 2 to 3 years and 10 and 12 years.[55] Juvenile PsA has been estimated to account for 6% to 8% of all cases of pediatric inflammatory arthritis.[68] Pediatric patients with PsA should be evaluated for uveitis.

Treatment Options for Pediatric PsO

Although an increasing number of treatments have been approved by the FDA for pediatric PsO, most treatments are prescribed off label. The currently available treatment options recommended by AAD–NPF guidelines are topical medications, phototherapy, oral retinoids, immunosuppressants, and biologic agents (Table 3).[69,70] A topical corticosteroid is most often used first line for children with mild to moderate PsO. A limited number of low-potency topical corticosteroids are the only choices labeled to treat pediatric PsO in children under the age of 12 years. Although narrowband UV-B phototherapy has been shown to be an effective treatment, second-line use in children is limited by cost and need for in-office visits 2 to 3 days per week. Coal tar can be used in combination with other therapies such as phototherapy. For patients with an inadequate response to topical treatments or with additional comorbidities, oral immunomodulating agents, such as methotrexate or cyclosporin, or systemic retinoids, such as isotretinoin or acitretin, may be used. Children with involvement that is widespread or affecting sites that are difficult to treat topically (such as the scalp, face, groin, palms, soles, and nails), juvenile PsA, or contraindication to oral agents are candidates for treatment with a biologic. Biologics that are labeled for pediatric use include inhibitors of TNF (etanercept in the United States and European Union and adalimumab in the European Union), IL-12/23 (ustekinumab), and IL-17A (ixekizumab and secukinumab). Dosing information and clinical trial results for biologics for the treatment of pediatric PsO were previously reviewed.[71] The topical phosphodiesterase-4 (PDE4) inhibitor roflumilast was also recently approved in the United States for the treatment of plaque PsO in patients ≥12 years. Other systemic medications currently under investigation for pediatric PsO include biologics such as the TNF inhibitor certolizumab pegol; the IL-17 receptor A inhibitor brodalumab; the IL-23 inhibitors guselkumab, tildrakizumab, and risankizumab; oral PDE4 inhibitors such as apremilast; the tyrosine kinase 2 inhibitor deucravacitinib; and new nonsteroidal topicals such as tapinarof (an aryl receptor inhibitor).

TABLE 3.

Treatment Options for Pediatric PsO			
	Medication	**Mechanism of action**	**Adverse effects**
FDA-approved treatments			
Topical	Calcipotriene (available as a foam)[101]; approved for children aged ≥4 years	Synthetic vitamin D_3 analog	• Application site erythema • Application site pain
	Calcipotriene and betamethasone (available as ointment, suspension, and foam)[102]; approved for children aged >12 years	Combination synthetic vitamin D_3 analog and corticosteroid	In addition to the potential adverse effects from calcipotriene: • Erythema • Folliculitis • Pruritus • Vesiculation
	Roflumilast[103]; approved for children aged ≥12 years (including for intertriginous psoriasis)	PDE4 inhibitor	• Application site pain • Diarrhea • Headache • Insomnia • Upper respiratory tract infection • Urinary tract infection
Biologic	Etanercept[94]; approved for children aged ≥4 years	TNF inhibitor	• Infections • Injection site reactions
	Ustekinumab[104]; approved for children aged ≥6 years	IL-12/IL-23 inhibitor	• Nasopharyngitis • Upper respiratory tract infection • Headache • Fatigue
	Ixekizumab[97]; approved for children aged ≥6 years	IL-17A inhibitors	• Injection site reaction • Upper respiratory tract infection • Tinea infection
	Secukinumab[105]; approved for children aged ≥6 years		• Upper respiratory tract infection • Nasopharyngitis • Diarrhea
Off-label treatments			
Topical	Triamcinolone acetonide, budesonide clobetasol propionate, desonide, fluocinolone acetonide, fluocinonide, hydrocortisone, and triamcinolone[106]	Corticosteroids	• Skin atrophy • Telangiectasia • Striae distensae • Acne • Folliculitis • Purpura • May exacerbate dermatoses • Contact dermatitis • Cushing syndrome • Cataracts • Glaucoma • Symptomatic hypothalamic-pituitary-adrenal axis suppression
	Tacrolimus[107]	Calcineurin inhibitors	• Malignancy • Infections • Lymphomas • Skin malignancies • Skin burning or pruritus
	Pimecrolimus[108]		• Application site burning • Headache • Nasopharyngitis • Cough • Influenza • Pyrexia • Viral infection

TABLE 3. TABLE 3. (CONTINUED)

Treatment Options for Pediatric PsO			
	Medication	**Mechanism of action**	**Adverse effects**
Off-label treatments			
Topical	Tazarotene[109]	Retinoid	• Pruritus • Burning/stinging • Erythema • Worsening of PsO • Irritation • Skin pain • Photosensitivity
	Crisaborole	Nonsteroidal PDE4 inhibitor	• None observed
	Anthralin[106]	Blocks DNA synthesis and increases reactive oxygen species release	• Skin irritation • Staining of skin and nails
	Coal tar[106]	Not well understood; potentially through suppression of DNA synthesis	• Irritant contact dermatitis • Folliculitis • Photosensitivity to UV-A • Pediatric patients should use with caution
Nonbiologic systemic	Methotrexate[110]	Dihydrofolate reductase inhibitor	• Nausea • Anorexia • Stomatitis • Fatigue • Myelosuppression • Hepatotoxicity • Pulmonary fibrosis • Gastrointestinal irritation • Psychosis (rare)
	Cyclosporin[110]	Calcineurin inhibitor	• Nephrotoxicity • Hypertension • Impaired renal function • Cutaneous squamous cell carcinomas • Hypertrichosis
	Acitretin[110]	Systemic retinoid	• Teratogenicity • Birth defects • Hepatotoxicity • Cheilitis • Dryness of the eyes, nasal, and oral mucosa • Epistaxis • Xerosis • Brittle nails • Hair loss • Burning or sticky skin • Retinoid dermatitis • Photosensitivity
Phototherapy	Narrowband UV-B phototherapy[111]	Downregulation of immune cell activity	• Burning • Lesional blistering • Potentially carcinogenic • Erythema • Reactivation of herpes simplex • Varicella
Therapeutics that can potentially trigger or worsen PsO			
Biologics	Etanercept, infliximab, adalimumab, certolizumab pegol[18,36-40]	TNF inhibitors	• Can lead to aggravation of preexisting immune-mediated inflammatory diseases and trigger new inflammatory diseases, including psoriasis and Crohn's disease

Abbreviations: FDA, US Food and Drug Administration; IL, interleukin; PDE4, phosphodiesterase-4; PsO, psoriasis; TNF, tumor necrosis factor.

TABLE 4.

Indications for Referring a Child With Suspected PsO to a Dermatologist	
Clinical parameter	**Indicators**
Clinical signs	• Suspicion of PsO based on clinical signs and symptoms, especially based on location, severity, and duration of lesions - Presence of lesions in sites that are difficult to treat with topical medication such as genitals, scalp, nails, or palmoplantar areas - BSA >10% - Severity affecting quality of life
Response to treatment	• Lack of response to weak topical corticosteroid
Comorbidities	• Presence of comorbidities highly associated with PsO such as joint pain, diabetes, thyroid disease, and IBD
Other	• If diagnosis is not definitive

Abbreviations: BSA, body surface area; IBD, inflammatory bowel disease; PsO, psoriasis.

TABLE 5.

Pediatric PsO Resources for Patients and Their Families	
Resource	**Link**
National Psoriasis Foundation	https://www.psoriasis.org/
Over-the-Counter Topicals	https://www.psoriasis.org/over-the-counter/
Integrative Approaches to Care	https://www.psoriasis.org/integrative-approaches-to-care/
Media for Patients	https://www.psoriasis.org/watch-and-listen/
Patient Navigation Center	https://www.psoriasis.org/navigationcenter/
Our Spot for Youth and Parents	https://www.psoriasis.org/our-spot/
American Academy of Dermatology	https://www.aad.org/public
Psoriasis Resource Center	https://www.aad.org/public/diseases/psoriasis
Good Skin Knowledge Youth Education	https://www.aad.org/public/parents-kids/lesson-plans
Camp Discovery for Kids	https://www.aad.org/public/public-health/camp-discovery
Children's Skin Disease Foundation	https://www.csdf.org/
Camp Wonder	https://www.csdf.org/camp-wonder

Abbreviation: PsO, psoriasis.

Management of Pediatric PsO

Pediatricians can initiate first-line treatment for children with PsO beginning with a topical corticosteroid applied no more than once a day. In many cases, topical corticosteroid therapy will yield improvement but not clearing, and rebound worsening once treatment is stopped is common. A corticosteroid-sparing topical medication can be added to address either of these suboptimal responses. These medications include synthetic vitamin D analogs (calcipotriol and calcitriol) alone or as 2-ingredient combination vitamin D/corticosteroid products, as well as calcineurin inhibitors (tacrolimus and pimecrolimus), retinoids (tazarotene), coal tar, salicylic acid, and anthralin. A dermatologist is typically more familiar with second-line topical choices and indications for systemic treatment and can also provide access to phototherapy (Table 4).

Successful treatment requires shared medical decision-making so that patients and their families are comfortable with the treatment plan, including the relative risks and benefits of available options and long-term safety.[13,72,73] Dosing schedules or treatment reminders can support medication adherence.[74,75] In addition to treating skin signs and symptoms, successful management of pediatric PsO requires consideration of other aspects of the disease, including triggers and associated mental health issues.[7,13,45] Ideal long-term management depends on choosing a medication that will not worsen or optimally will improve coexisting medical conditions.[7] Children and adolescents with psychiatric comorbidities can benefit from counseling to help manage the negative mental components of the disease.[7,13]

For pediatric patients with PsO and their families, several informational, emotional, and social support resources are available (Table 5). The NPF provides useful information for how pediatric patients can manage their PsO, including diet and lifestyle changes, such as increased physical activity, that

can help reduce the risk of comorbidities.[76] Use of a moisturizer that contains scale softeners, salicylic acid, lactic acid, glycolic acid, urea, or the anti-itch ingredients pramoxine, menthol, or calamine can augment skin care.[77] Other alternative management approaches include acupuncture, apple cider vinegar for scalp itch, capsaicin added to topical medications, dilute bleach, Dead Sea or Epsom salt baths, or tea tree oil; however, these approaches lack clinical research on their long-term effectiveness and safety,[78] and some can sting or cause skin irritation. The NPF website provides articles, webinars, podcasts, and videos about PsO and PsA, including treatment options and management, news, and stories from patients with PsO.[79] Other support resources provided by the NPF include a free patient navigation center to help with questions about PsO and a peer support network that matches patients and caregivers with people who have experienced a similar situation and can provide guidance and reassurance.[80] "Our Spot for Youth" is a patient resource center that provides welcome kits for pediatric patients with PsO and their families, tips on communicating with teachers and friends, and downloadable school resources.[81] The AAD also provides a PsO resource center with information about the disease, diagnosis, and treatment options as well as skin, hair, and nail care guides for patients with PsO.[82] These resources include a youth education campaign, "Good Skin Knowledge," which provides lesson plans and handouts to teach kids about common skin, hair, and nail conditions, such as PsO.[83]

Children with skin conditions, including PsO, are eligible to attend specialty summer camps. This experience can help improve self-esteem, social skills, body image, and skin care routines.[84] The AAD Camp Discovery is a no-cost summer camp designed for pediatric patients with chronic skin conditions.[85] The Children's Skin Disease Foundation's Camp Wonder is a week-long summer camp opportunity for children with chronic and life-threatening skin diseases provided free of cost for campers.[86]

Current Challenges for Pediatricians in the Treatment of Pediatric PsO

Misdiagnosis can prompt treatment of PsO with an oral or parenteral corticosteroid. This approach is well known to trigger rebound worsening or even pustular flares. Other pediatric-specific challenges can complicate treatment, including tactile aversion to topical medications, needle phobia, and anticipatory nausea or emesis.[87] Among the many systemic options FDA approved to treat PsO in adults, only 5 drugs are currently approved by the FDA for moderate to severe pediatric PsO. Insurance coverage is often denied for off-label treatments.[88,89] When access is available, out-of-pocket treatment for PsO has been documented to cost an average of $2528 per year, an important factor that limits optimal treatment.[90] Due to the difficulty in diagnosing pediatric PsO, patients are often misdiagnosed and prescribed treatments that can worsen their disease (Table 3). As skin lesions often resemble a rash, patients with PsO who are treated at emergency clinics are often prescribed oral, topical, or systemic corticosteroids that can worsen their PsO. Patients with PsO who are misdiagnosed and treated with TNF inhibitors may experience induction or exacerbation of PsO. Pediatricians should be aware that prescribing corticosteroids before an accurate diagnosis is made is not best practice and should consult a dermatologist if there is uncertainty about a diagnosis.[88,89]

Pediatricians should also be aware of potential adverse effects when prescribing topical corticosteroids for children. Although these medications are a time-honored and cost-effective approach, long-term safety data are limited. Safety is supported by using the lowest potency product that is effective for the patient.[45] Higher potency topical corticosteroids used more than once a day and applied under occlusion (eg, diaper area) and on the face and fold carry the highest risk of skin barrier compromise, percutaneous absorption, and hypothalamic-pituitary-adrenal axis suppression.[45] Phototherapy can be time-consuming and require high out-of-pocket costs, and improvement is typically not appreciated for several weeks. Potential long-term adverse effects of phototherapy include photoaging, actinic keratoses, and skin cancer,[91] although this risk is lower for narrowband UV-B than combination UV-A plus topical psoralens.[92] The need for protective eyewear also poses special risks for children undergoing phototherapy, and isolated, underreported retinal burns have occurred in children unwilling to leave eyewear in place.[56]

PsO that requires long-term use of systemic medication carries risks of drug-specific, treatment-emergent adverse effects (Table 3). Injection site reactions are the most common adverse effect of biologic agents.[93] Long-term safety concerns with TNF inhibitors include increased risk of serious infections (eg, tuberculosis), development of autoimmune phenomena (ie, IBD, diabetes, and paradoxical PsO),[93] and lymphomas and other malignancies,[94] although there were no reported malignancies in a long-term safety study of etanercept treatment in pediatric patients with PsO.[95] Pediatric patients receiving secukinumab or ixekizumab should be monitored for new or worsening IBD, which has occurred in adult patients with PsO receiving these biologics.[96,97] However, no confirmed cases of treatment-emergent IBD in pediatric patients receiving secukinumab have been observed in clinical trials to date. Hypersensitivity reactions and serious infections have been reported for every biologic approved for use in children. There are no data on the impact of biologic agents on vaccine response; therefore, up-to-date immunization status is recommended prior to starting any of these medications. Avoiding live virus vaccines is recommended in all children receiving immunosuppressant or biologic medication.

CONCLUSION

Pediatrician familiarity with the clinical presentation, diagnosis, and treatment of pediatric PsO will allow earlier and more effective management, alleviation of the physical and psychosocial burdens, and referral for long-term treatment when indicated.

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