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Guttate Psoriasis in Skin of Color: Underrecognized Presentations and Diagnostic Challenges

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ABSTRACT

Background: Guttate psoriasis is an acute variant of psoriasis marked by drop-like papules, often following streptococcal infection. In individuals with skin of color (SOC), presentations are underrecognized due to atypical morphology and diagnostic challenges.

Objective: To review the literature on guttate psoriasis in SOC, with a focus on clinical features, diagnostic hurdles, and management.

Methods: A narrative review (2015–2025) of case reports, series, observational studies, and clinical trials across all age groups and immune statuses was performed. Findings were organized into epidemiology, clinical features, diagnostics, and treatment outcomes.

Results: Guttate psoriasis represents up to 25% of psoriasis cases, most often in children and young adults. In SOC, lesions may appear violaceous, brown, or gray rather than classic erythematous, with frequent post-inflammatory pigmentary changes, leading to delayed or missed diagnosis. Pediatric flares commonly follow streptococcal infection, while immunocompromised and atopic patients may develop guttate eruptions after infections (HIV, COVID-19) or medications. First-line therapy includes topical corticosteroids and vitamin D analogs; narrowband UVB is highly effective. Systemic agents (methotrexate, cyclosporine, acitretin, apremilast) are used for refractory cases, and biologics (anti–IL-17, IL-23) can achieve rapid clearance. Treating streptococcal infection is advised, though antibiotics have a limited effect on cutaneous disease.

Conclusions: Guttate psoriasis in SOC requires heightened clinical suspicion, with dermoscopy or biopsy aiding diagnosis. Management should follow a stepwise approach from topical therapy to biologics. Greater SOC representation in research and dermatology education is vital to reducing disparities.

Keywords: Psoriasis; Guttate Psoriasis; Skin of Color; Diagnostic Disparities; Phototherapy; Biologics; Pediatric Dermatology; Post-Inflammatory Hyperpigmentation

Abbreviations: PLC: Pityriasis Lichenoides Chronica; PIH: Post-Inflammatory Hyperpigmentation; SOC: Skin of Color; LP: Lichen Planus

Introduction

Psoriasis is a chronic inflammatory dermatosis with an estimated prevalence of 2–3% of the general population worldwide [1]. Guttate psoriasis is a variant that may comprise up to one fourth of all psoriasis cases [2]. It classically presents as an acute eruption of multiple small "drop-like" papules with fine scale. It is often precipitated by an antecedent group A streptococcal infection such as streptococcal pharyngitis [3]. Guttate lesions most commonly affect the trunk and proximal extremities, and the rash tends to erupt in children, adolescents, and young adults [4]. Guttate psoriasis is often self-limited and will resolve within 3–4 months in the majority of patients, but

a substantial proportion of patients will go on to develop recurrent or chronic disease; approximately 25–40% of patients will eventually develop chronic plaque psoriasis [2]. Patients of color (including African, Asian, Latinx, or other non-Caucasian ethnic backgrounds) have historically faced challenges with recognition and management of psoriasis. The greater melanin content of the skin of color (SOC) may mask the inflammatory erythema, and lesions may present with a different coloration (e.g., violaceous, hyperpigmented) than the classic well-demarcated red lesions on lighter skin [1]. Guttate psoriasis in SOC is frequently underrecognized or misdiagnosed, as the more subtle presentation may be difficult to clinically differentiate from other papulosquamous disorders that are more common in these populations [5,6].

Nicholas, Chan, and Hessami-Booshehri [1] reported that racial and ethnic disparities in the care of psoriasis have been well documented in the literature, with Black and Hispanic patients historically reporting lower prevalence of the condition (1.9% and 1.6%, respectively) compared with white patients (3.6%), a difference partially attributed to underdiagnosis and the lack of data on psoriasis in these populations [1]. The historical underrepresentation of SOC in dermatology research and training has led to persistent knowledge gaps and under recognition of SOC variants of psoriasis, including guttate psoriasis [7]. In this narrative review, we focus on the presentation of guttate psoriasis in SOC, in particular, how it may differ from that in patients with lighter skin phototypes, as well as the diagnostic challenges it can present, contributing to inequities in care. We also review evidence for this entity across all relevant patient populations – pediatric, adult, elderly, and immunocompromised – to provide the most comprehensive picture. We prioritized data from primary studies in our recent literature review (2015–2025) to best inform clinical practice for clinicians, including insights into differential diagnosis, as well as a thorough discussion of treatment options (topical agents, phototherapy, systemic therapies, and biologics) across the full armamentarium. By highlighting the underrecognized clinical features of guttate psoriasis in SOC and summarizing available evidence for managing this condition in the modern era, we hope to equip clinicians to make better and more timely diagnoses and deliver effective, equitable care to all of their patients.

Methods

The research team conducted a literature search targeting primary studies on guttate psoriasis with skin of color (SOC) or diverse patient populations. PubMed, Scopus, and Web of Science databases were searched from 2015 to 2025 using combinations of the following keywords: “guttate psoriasis,” “skin of color,” “dark skin,” “pediatric psoriasis,” “psoriasis case report,” and “psoriasis treatment.” Clinical studies of guttate psoriasis (case reports or series, cohort studies, clinical trials, randomized controlled trials) and other primary research (animal and laboratory studies, translational research, other case series) were prioritized. Review articles were analyzed for contextual information on guttate psoriasis in SOC. No language filters were applied; however, only articles published in English-language journals were included. Studies solely focused on chronic plaque psoriasis or other psoriasis subtypes were excluded unless they provided data relevant to guttate psoriasis or analyzed guttate cases as a subgroup. Data extraction focused on: patient demographics (including race/ethnicity when available), precipitating factors (e.g., infections), clinical morphology, diagnostic methods (dermoscopy, biopsy), and treatment approaches. As this was a narrative (non-systematic) review, no formal risk of bias assessment was performed; however, evidence synthesis emphasized larger studies or those with more rigorous designs. Findings were tabulated and grouped into the following thematic categories:

1. Epidemiology and patient demographics;
2. Clinical presentation in SOC vs lighter skin;
3. Diagnosis and differential diagnosis;
4. Special populations (children, older adults, immunocompromised) and
5. Management strategies (topical, phototherapy, systemic, and biologic therapies, including SOC-specific considerations). Representative cases were included to highlight key or novel observations.

This narrative review synthesizes available primary research on guttate psoriasis in SOC, highlighting patterns in epidemiology, presentation, diagnosis, and treatment. While data remain limited, grouping studies by key clinical domains allowed identification of recurring themes and knowledge gaps that may guide future research and clinical care.

Results

Epidemiology and Underrecognized Nature of Guttate Psoriasis in SOC

Guttate psoriasis may present more commonly in younger patients. For example, among pediatric patients, an extensive review noted that guttate flares account for a substantial proportion of psoriasis cases, with another large pediatric review estimating a prevalence of 0.5–2% in children overall [8]. Guttate is often the initial form of psoriasis in young patients [3]. As with adult patients, patients with guttate psoriasis frequently have a recent history of infection: in series, streptococcal infection is estimated to precede guttate eruptions in 56–97% of cases, depending on the study [9]. Viral triggers for guttate have been described increasingly over time: new-onset guttate psoriasis has been reported after COVID-19 infection [3] as well as coxsackievirus (hand-foot-mouth disease) [10], and likely any infection that stimulates the immune system may be a potential trigger. Post-viral guttate flares have been described in both immuno-

competent and immunocompromised hosts. Medications that modify immune function have also been increasingly linked to guttate psoriasis, especially in the setting of "paradoxical" psoriasiform reactions to medicines intended for other uses. A recent case described a 9-year-old Black child with atopic dermatitis who developed an eruption consistent with guttate psoriasis after starting the IL-4 receptor antagonist dupilumab, as an example of a paradoxical reaction to an atopic dermatitis biologic [11]. TNF inhibitors for other indications have also been linked to the development of psoriasis, including guttate-type lesions in rare cases [12].

Earlier research found that non-white patients exhibited lower psoriasis prevalence rates. For example, Black and Asian people have about half the prevalence of psoriasis as white people in the United States in population surveys [1]. However, more recent data suggest that this discrepancy may be at least partly a result of underdiagnosis in SOC patients. Rao et al. investigated a comprehensive health database with varied patient groups, demonstrating that underrepresented racial populations experience greater underdiagnosis of psoriasis than white patients, even when accounting for clinic access and visit frequency. Many SOC patients with symptoms and signs compatible with psoriasis had not received a diagnosis [13]. Krefting et al. performed a study of dermatologists' ability to recognize different skin disorders on images; they found that dermatologists were significantly less accurate when attempting to identify lesions, including psoriasis, on darker skin compared to lighter skin [6,14]. These studies and others suggest that, in fact, guttate psoriasis in SOC is underreported and is under-recognized or often misdiagnosed as other skin disorders.

Clinical Presentation Differences in Skin of Color

The classic clinical presentation of guttate psoriasis on light skin is well-described as numerous, scattered pink to salmon-colored papules or small plaques with fine silvery-white scale. In patients with SOC, morphology may vary in some key ways. Lesions on darker phototypes may have a violaceous, purplish, or grey-brown hue instead of the pink to salmon-red of erythematous psoriasis on lighter skin [1]. They may also be less overtly inflamed: the erythema may be obscured by overlying epidermal melanin and thus inflammation may be underappreciated on exam [1]. Instead of bright-red plaques, a patient may present with numerous dusky or hyperpigmented drop-like macules with overlying scale. The scale in guttate psoriasis is characteristically thin, so on lighter skin it is often white or micaceous. On SOC, scale may still be present but can be finer and at times appears grayish or off-white against the background skin [3]. Nicholas, et al. found that psoriasis lesions in patients of color had less obvious scaling on the surface and a more modest elevation than in white patients [1].

Another key differentiating feature is the sequelae of lesions: post-inflammatory pigmentary alteration (hyperpigmentation or hypopigmentation) is widespread in SOC guttate psoriasis [5]. It has been shown to disproportionately affect patients with SOC and their quality of life [15]. As the active lesions heal, they often leave behind areas of hyperpigmentation or hypopigmentation, sometimes for weeks to months [16]. Leung et al. found that children with guttate psoriasis in darker skin had extensive post-inflammatory hyperpigmented macules after the rash resolved, which at times was more distressing to the patients/parents than the acute rash itself [9]. This post-inflammatory pigment change may make it difficult to assess if psoriasis is active or to differentiate it from other dyschromic conditions. By contrast, in lighter skin types, resolving guttate lesions may leave a transient, faint pink or brown mark, but generally not to the same extent of dyschromia (Table 1).

Table 1: Key differences in guttate psoriasis between skin of color and light skin.

Feature	Skin of Color (darker phototypes)	Light Skin (Types I–III)
Lesion color	Violaceous, purple, or gray-brown papules; erythema is muted or not obvious [1]. May be misperceived as hyperpigmented spots.	Pink, red, or salmon-colored papules with obvious erythema [3]. Classic "red drop" appearance.
Scale	Often fine and gray-white in color, sometimes less prominent to the naked eye [3]. Scale may be scant in early lesions.	Typically, fine silvery-white scale on most lesions. Readily visible against red background.
Post-inflammatory changes	Hyperpigmentation or hypopigmentation is common after lesions heal, leading to a spotted appearance [1]. Can last for months and be a primary patient concern.	Usually minimal residual pigment change; healed lesions may leave slight pinkness or none.
Diagnostic delay	Higher chance of misdiagnosis (tinea, eczema, etc.) and need for biopsy to confirm [1,3]. Lesions may be widespread by the time of diagnosis.	Often recognized clinically on initial exam due to characteristic appearance; biopsy is seldom needed.
Dermoscopy	Dotted vessels and diffuse white-gray scale are visible, aiding diagnosis when clinical redness is absent [3].	Dotted vessels on red background (consistent with plaque psoriasis patterns) [3]. Dermoscopy corroborates clinical diagnosis.
Psychosocial impact	Noticeable pigmentary sequelae can cause distress or cosmetic concern; patients may feel stigmatized by dark spots. Some report being told "black people don't get psoriasis," reflecting misinformation [1].	Visible rash and scaling can be embarrassing, but less chronic pigmentary change. Better public awareness of psoriasis exists in some regions for lighter skin.

Importantly, aside from color, the distribution of guttate lesions is similar across skin types – predominantly on the torso, back, and proximal limbs, with relative sparing of palms, soles, and face [3]. However, because of the subtlety on dark skin, patients with SOC might present later or after the rash has spread more extensively. Some case series have reported more widespread or confluent guttate lesions in Black and Asian patients at presentation, potentially due to a delay in recognition of the initial smaller crops [1,17]. Figure 1 illustrates the contrast in appearance of guttate psoriasis on different skin tones. Figure 1A shows guttate lesions on brown skin manifesting as faint violaceous spots with overlying scale and significant post-inflammatory hyperpigmented macules [18]; Figure 1B shows the classic erythematous "drop-like" plaques on a light-skinned patient, with bright red coloration and minimal lasting pigmentation [16]. Clinical presentation of guttate psoriasis differs significantly between lighter and darker phototypes. In skin of color, erythema may be subtle or absent, while residual hyperpigmentation is more prominent, contributing to under recognition and diagnostic delays [1,16]. Comparative images highlight how visibility and morphology vary by skin tone.

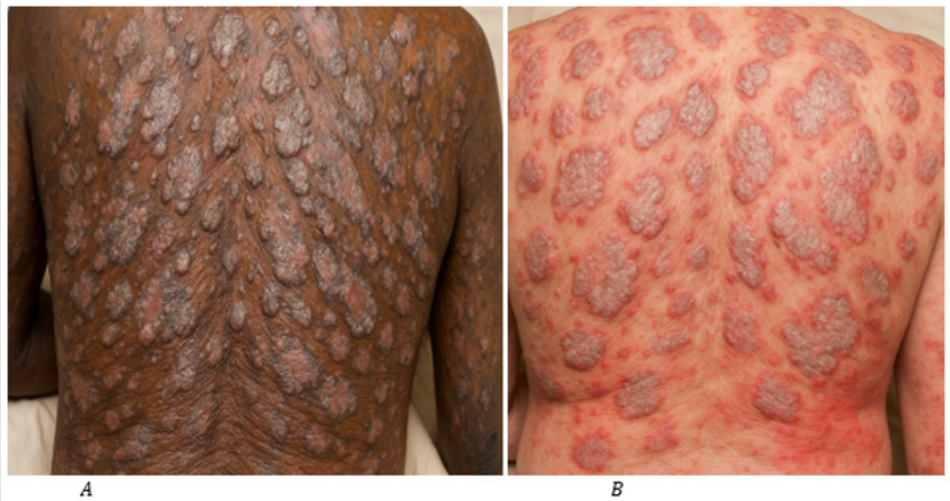


Figure 1: Clinical Presentation of Guttate Psoriasis in Different Skin Types.

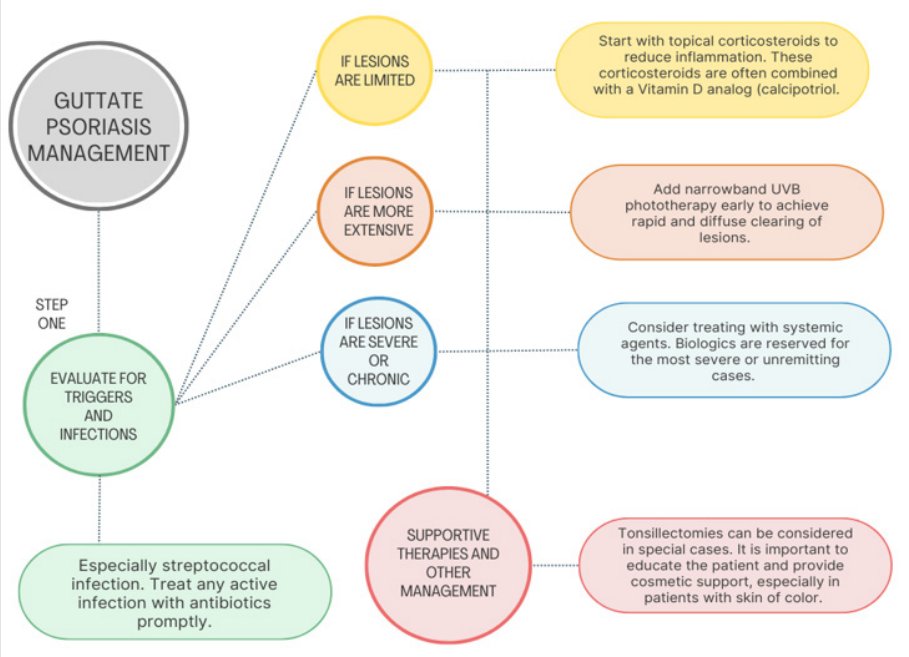
- A: Guttate psoriasis in skin of color: violaceous to hyperpigmented papules with fine scale and post-inflammatory hyperpigmentation; erythema is subtle.
- B: Guttate psoriasis in lighter skin: bright red, drop-like papules with thin white scale, readily visible due to high color contrast [1,16].

Diagnostic Challenges and Differential Diagnosis

The atypical coloration of guttate psoriasis in SOC contributes to diagnostic uncertainty. Clinicians unfamiliar with psoriasis manifestations in darker skin may misidentify lesions as other conditions. Earlier work has also noted that psoriasis in special populations, including patients with HIV, presents additional diagnostic and therapeutic challenges [19-21]. Groh, et al. [22] conducted a large-scale image-based diagnostic study. They found that dermatologists and general practitioners were notably less accurate when evaluating darker skin, with accuracy dropping by approximately four percentage points compared to light skin, underscoring the risk of misdiagnosis in darker phototypes [22]. Similarly, Dickerson, et al. [23] found that Black patients were more likely to experience delayed or incorrect diagnoses, often being told they had dermatitis or fungal infection before psoriasis was considered [23]. In practice, a SOC patient with acute scaly papules may initially be treated for tinea corporis or pityriasis rosea, delaying appropriate management for psoriasis. Physicians may also resort to biopsy more frequently in SOC patients to confirm psoriasis. A recent analysis showed that non-White patients with psoriasis underwent diagnostic biopsy at higher rates than White patients, reflecting clinicians' lower diagnostic confidence in these populations [24]. While biopsy can distinguish guttate psoriasis from mimickers, it is invasive, and even histopathology may be subtle, showing only mild psoriasiform hyperplasia with focal Munro microabscesses, sometimes interpreted as non-specific dermatitis if the clinical context is lacking [25].

Distinguishing guttate psoriasis from other papulosquamous disorders is particularly challenging in SOC, where overlapping morphologies obscure classic textbook features. Pityriasis rosea (PR) is a frequent confounder: in lighter skin, it often shows a herald patch, collarette scaling, and lesions aligned along skin tension lines, but in SOC, the herald patch may be absent, and lesions appear hyper-

pigmented [18,25]. Pityriasis lichenoides chronica (PLC) can mimic guttate psoriasis with small scaly papules, but lesions persist longer, appear in various stages, and may leave hypopigmented spots. Lichen planus, especially lichen planus pigmentosus, can also present with small flat-topped papules, but the violet-brown coloration, predilection for wrists/legs, pruritus, and Wickham striae aid differentiation. Secondary syphilis—known as the "great imitator"—produces widespread reddish-brown papulosquamous lesions, frequently involving palms, soles, and mucous membranes, often accompanied by systemic symptoms such as fever and lymphadenopathy [26]. Because missing syphilis has serious implications, serologic testing should be performed whenever clinical ambiguity exists. These key mimickers and distinguishing features are summarized in Table 2, which outlines the differential diagnosis of guttate psoriasis and emphasizes clinical clues particularly relevant in skin of color.



Note: Stepwise approach: screen and treat strep infection, use topicals for mild disease, NB-UVB for widespread eruptions, systemic agents for refractory cases, and biologics for severe disease; tonsillectomy may help in recurrent strep-associated flares.

Figure 2: Proposed Management Algorithm for Guttate Psoriasis.

Table 2: Differential diagnosis of guttate psoriasis and distinguishing clinical features.

Condition	Distinguishing Features	Key Diagnostic Aids
Pityriasis rosea	Herald patch precedes eruption; lesions along Langer's lines with collarette scale. In SOC, hyperpigmented lesions with minimal scale.	Serology to rule out syphilis; dermoscopy: yellow-white scale & patchy vessels vs white scale & diffuse vessels (guttate) [17,18].
Pityriasis lichenoides chronica (PLC)	Small scaly papules in various stages; some crusted from prior PLEVA. Chronic waxing/waning course.	Biopsy shows lymphocytic vasculitis/interface changes; responds differently to antibiotics or phototherapy; no strep trigger.
Nummular dermatitis	Coin-shaped scaly plaques, often pruritic, with crust/lichenification. Common on extremities.	Histology: spongiotic dermatitis. Look for atopic history/flexural sites. KOH prep if tinea suspected.
Tinea (dermatophyte)	Annular plaques with raised border and central clearing; guttate lacks this. PIH is common in SOC.	KOH/culture positive; lesions improve with antifungals (psoriasis does not).
Lichen planus	Violaceous, flat-topped papules on wrists/ankles; Wickham striae. Often itchy; leaves PIH in SOC.	Dermoscopy: Wickham striae. Biopsy: lichenoid dermatitis. Check meds for drug-induced LP.
Secondary syphilis	Rash often on palms/soles; coppery papulosquamous lesions ± collarette scale. Systemic signs (fever, mucous patches, condyloma lata).	Serology (RPR/VDRL, treponemal tests) positive. Rapid response to penicillin, unlike psoriasis.

Clinical diagnosis of guttate psoriasis in darker-skinned patients requires dermatologists to use supplementary tools according to anecdotal reports [14,22].

Dermoscopy is one such tool that can be helpful. Dermoscopic features of guttate psoriasis include a dull red or salmon-pink background color and the presence of uniformly distributed dotted blood vessels (dilated capillaries in dermal papillae) with superimposed fine white-gray scale [3]. Dermoscopic features have been noted in multiple studies of psoriasis in dark skin types (see References 1 and 2 for reviews). These findings are present in the skin of all colors, but are especially useful in SOC, where the naked eye might miss the redness. Dermoscopy may make visible these vascular dots even in lesions that appear "non-erythematous" by filtering out surface pigmentation. In a 2021 observational study of dermoscopy in dark skin, Jindal et al. reported that white-gray scale and regularly distributed red dots were seen in 80% of guttate psoriasis lesions in their SOC cohort, in contrast to pityriasis rosea lesions, where the scale was more often yellow-white and the vessels patchier in distribution [17]. A second study by Makhecha, et al. [18] demonstrated that guttate psoriasis could be reliably differentiated from pityriasis rosea by dermoscopy, even when the clinical presentation was confusing, by recognizing the peripheral collarette scale in PR versus the diffuse scale in guttate psoriasis [18,27]. Thus, dermoscopy is a noninvasive way to add confidence to a psoriasis diagnosis in SOC patients. If still in doubt, a skin biopsy can be diagnostic. Histologic findings in guttate psoriasis include psoriasiform epidermal hyperplasia, a thin confluent parakeratotic scale, neutrophils in the stratum corneum (Munro microabscesses), and mild superficial perivascular lymphocytic infiltrates [25].

These findings may be less fully developed than in chronic plaque psoriasis. Still, they should generally be recognizable from the focal mounds of parakeratosis seen in pityriasis rosea or the interface changes of lichen planus. Ahmed, et al. [24] reported in practice that biopsies were disproportionately performed in patients of color with psoriasis, suggesting that clinicians still frequently fall back on histopathology in SOC cases [24]. Of course, a biopsy can definitely diagnose guttate psoriasis. Still, it is invasive and itself leaves a scar or potential pigmentation, so ideally, more robust clinical recognition can reduce its need.

Special Populations and Clinical Considerations

The relative paucity of SOC images in medical textbooks and training materials compounds this. Onasanya, et al. [28] demonstrated that skin of color remains significantly underrepresented in dermatology curricula, limiting trainees' diagnostic confidence [28]. More recently, Groh, et al. [22] confirmed that diagnostic accuracy is lower when evaluating psoriasis in darker skin tones, underscoring the ongoing impact of underrepresentation in training resources [22]. Guttate psoriasis presents unique diagnostic and management challenges across different patient populations, including children, the elderly, and the immunocompromised. It is particularly common in pediatric patients, often representing the first manifestation of psoriasis [3]. Most cases are triggered by acute streptococcal pharyngitis or tonsillitis, with >80% showing positive cultures or elevated antistreptococcal titers [29]. Lesions are typically small and numerous; in fair-skinned children, they appear bright red and mimic viral exanthems, while in SOC children, they may be mistaken for eczema or pityriasis lichenoides [9]. Additional triggers include perianal streptococcal dermatitis in toddlers [30] and, less commonly, viral infections such as varicella or COVID-19 [3]. The psychosocial impact on children can be profound, with teasing and family anxiety common. Long-term monitoring is essential, as one-third of pediatric cases progress to chronic plaque psoriasis [4], and parents should be counseled about recurrence with infections and the likelihood of post-inflammatory hyperpigmentation in SOC [9].

In contrast, new-onset guttate psoriasis is uncommon in the elderly, where it warrants a thorough evaluation for triggers such as streptococcal infection [4]. Older patients may be more prone to progression toward chronic plaque disease, and medication-related triggers such as abrupt corticosteroid withdrawal are particularly relevant [31]. The differential includes lichenoid drug eruptions and cutaneous T-cell lymphoma [26,32,33], and biopsy is often necessary, especially in SOC, where atypical features and photodamage obscure diagnosis. Treatment must account for comorbidities and polypharmacy; methotrexate requires caution in renal or hepatic impairment, and phototherapy dosing may need adjustment for thinner skin or concurrent use of photosensitizing drugs. Although therapies remain effective, tolerability and contraindications are common concerns [34,35]. In immunocompromised patients—including those with HIV/AIDS, organ transplants, or chronic immunosuppressive therapy—psoriasis may present atypically [21]. Advanced HIV can paradoxically worsen guttate or erythrodermic psoriasis despite immune suppression [36], while coexisting dermatoses such as Kaposi's sarcoma or seborrheic dermatitis may complicate diagnosis. Psoriasis in this setting often improves with initiation of antiretroviral therapy [37,38]. Transplant recipients or patients on TNF inhibitors may develop paradoxical guttate eruptions [19], and similar flares have been reported with dupilumab therapy through Th2 suppression and Th1/Th17 upregulation [11].

Management is complex, as systemic agents may be contraindicated, but phototherapy is often a practical alternative when carefully monitored [20]. Eradicating infectious triggers remains critical; for example, recurrent streptococcal tonsillitis may justify tonsillectomy in transplant patients [39,40]. In summary, guttate psoriasis in children, the elderly, and immunocompromised patients requires tailored recognition and management to address unique triggers, risks, and treatment challenges.

Management and Treatment Modalities

Management of guttate psoriasis requires treating both the eruption and its underlying triggers. Principles are broadly consistent across skin types, but in patients with skin of color (SOC), additional considerations include avoiding pigmentary sequelae and, less commonly, keloid formation. Topical therapy is first-line for limited disease. Mid- to high-potency corticosteroids (e.g., triamcinolone 0.1%, fluocinonide 0.05%) reduce inflammation and pruritus, with flattening of papules over 2–4 weeks [2]. Lower-potency or intermittent regimens are advised in children and when treating large body areas to minimize atrophy, striae, and hypopigmentation. In SOC, steroid-induced hypopigmented patches can be conspicuous; tapering and early transition to steroid-sparing agents are recommended. Vitamin D analogs (calcipotriene/calcipotriol) are commonly combined with corticosteroids, improving efficacy while reducing steroid exposure, and remain well established in psoriasis management [2,40]. Calcineurin inhibitors have limited roles, typically for sensitive sites [3]. Coal tar and anthralin are rarely used due to irritation and cosmetic drawbacks, especially staining in SOC. Emollients remain helpful adjuncts for scaling and barrier repair. Phototherapy is the preferred treatment for more extensive guttate eruptions. Narrowband UVB (NB-UVB, 311 nm) two to three times weekly induces remission within 4–8 weeks [2,3]. Zhou, et al. [2] identified NB-UVB as the modality with the strongest evidence for guttate psoriasis [2].

Because erythema can be difficult to detect in SOC, treatment is initiated at ~70% of the minimal erythema dose with cautious titration [1]. Hyperpigmentation or tanning is common and generally acceptable, but burns must be avoided to prevent dyschromia. Studies confirm that sub-erythema dosing achieves clearance in darker skin equivalent to standard dosing in lighter skin [1]. Recent randomized clinical trial evidence further supports both office- and home-based NB-UVB as safe and effective modalities for psoriasis, expanding access to phototherapy [41]. PUVA is rarely used given its long-term carcinogenic and photoaging risks [2], and excimer laser is impractical for numerous guttate lesions. Systemic therapies are reserved for severe or refractory guttate psoriasis. Methotrexate (7.5–15 mg weekly) effectively suppresses T-cell activity and is especially useful with psoriatic arthritis, though it requires monitoring and is used cautiously in children and women of childbearing potential [2]. Cyclosporine acts rapidly, often within 2–4 weeks, but is limited to ≤4 months due to nephrotoxicity and hypertension [19]. Acitretin is moderately effective, particularly in combination with phototherapy, and is safe in immunocompromised patients, but contraindicated in women of childbearing potential [2]. Apremilast, a PDE4 inhibitor, is immunomodulatory rather than broadly immunosuppressive, with case reports supporting efficacy in guttate psoriasis [38]. Antibiotics should be given only for documented streptococcal infections and are not recommended as primary therapy unless infection is confirmed [2,3].

Biologic therapies, though not standard, are increasingly reported in severe or persistent guttate psoriasis. Ustekinumab has induced remission in resistant cases [39], while TNF inhibitors (etanercept, adalimumab) can clear guttate lesions but paradoxically may also induce psoriasis [12]. IL-17 inhibitors (e.g., ixekizumab) have demonstrated rapid clearance [42], and IL-23 inhibitors (e.g., guselkumab) achieved complete or near-complete remission in case reports after only one or two doses [43,44]. Early biologic intervention is being studied for potential prevention of chronic plaque progression [39]. Biologics are equally effective in SOC, though access disparities remain. Tonsillectomy may benefit select patients with recurrent guttate flares linked to streptococcal tonsillitis, particularly those positive for HLA-C*06:02 [45]. Ferzli, et al. [36] reported a young adult with refractory guttate psoriasis who improved markedly after tonsillectomy [36]. While not routine, tonsillectomy can be considered when there is a clear recurrence tied to tonsillar infection [45]. A practical stepwise overview of these therapies is illustrated in Figure 2. In addition to the algorithm, a detailed comparison of therapeutic modalities is necessary to highlight practical considerations for both general use and SOC-specific outcomes. These are presented in Table 3. In summary, guttate psoriasis management follows a tiered framework based on disease severity, with modifications to reduce pigmentary sequelae in SOC and tailored use of systemic or biologic therapies for severe or refractory disease.

Table 3: Therapeutic Modalities for Guttate Psoriasis and Considerations for SOC.

Treatment	Description & Use	Considerations in Skin of Color	Evidence/Comments
Topical corticosteroids [2]	First-line for localized lesions; anti-inflammatory. Potency based on site/severity; short-term use (2–4 weeks).	Effective, but prolonged use can cause hypopigmentation or atrophy more visible in SOC. Ointments penetrate hyperkeratotic areas best.	Strong evidence; often combined with calcipotriene. Safe in children with monitoring.
Vitamin D analogs (calcipotriene) [2]	Regulates keratinocyte proliferation/differentiation. Used alone or with steroids.	Safe; does not alter pigmentation. Irritant dermatitis may trigger PIH in SOC.	Good evidence in plaque psoriasis; helpful in guttate, especially in steroid combos.
Narrowband UVB phototherapy [3,20]	311 nm UVB, 2–3×/week for 15–30 sessions. Treats widespread disease via T-cell apoptosis.	Effective across all skin types. In SOC, erythema is harder to detect; use sub-erythemal doses. May cause temporary PIH or tanning.	Strongest non-systemic option; safe for children. Limited by access/logistics.

Methotrexate [34]	Weekly low-dose (oral/SC) immunosuppressant for severe or unresponsive guttate.	No SOC-specific effects; screen for hepatitis in higher-risk groups.	Long track record in psoriasis; effective though slower onset (4–6 weeks). Teratogenic.
Cyclosporine [19]	Oral calcineurin inhibitor for rapid short-term control (≤4 months).	Rapid clearance is useful in severe SOC flares. Monitor BP/renal function.	Highly effective; clears guttate in <1 month. Used as bridge therapy due to toxicity.
Acitretin [2]	Oral retinoid; normalizes turnover. Mainly for pustular/chronic disease; sometimes with phototherapy.	Causes dryness, which may accentuate ashiness in SOC. Teratogenic for 3 years post-use.	Moderate efficacy; better combined with phototherapy. Not first-line for guttate.
Apremilast [38]	Oral PDE4 inhibitor; reduces cytokine release. Off-label for guttate.	Well-tolerated, no lab monitoring; useful where access is limited.	Limited case evidence; oral alternative when phototherapy/biologics not feasible.
Biologics [35]	Targeted monoclonals (TNF, IL-17, IL-23, IL-12/23). For severe guttate or progression to plaque.	Equally effective in SOC; underrepresentation in trials remains an issue. No pigment effects.	Excellent efficacy; rapid clearance in reports. Access/cost are major barriers.
Antibiotics (adjunct) [3]	Penicillin/amoxicillin for documented strep; sometimes empiric in classic guttate.	Safe in SOC; monitor for drug eruptions that may mimic psoriasis.	Necessary for strep eradication, but has an inconsistent effect on skin. Tonsillectomy for recurrent cases.

Discussion

Guttate psoriasis in patients with skin of color (SOC) exemplifies how variation in clinical appearance can contribute to disparities in care. While the underlying pathophysiology is consistent across populations—a T-cell–mediated immune reaction often triggered by streptococcal antigens—the skin findings differ with pigmentation. In darker skin, erythema may be subtle, and post-inflammatory pigmentation is prominent, making guttate psoriasis less readily recognized. This under recognition leads to delays in diagnosis and treatment [22,23]. Misdiagnosed patients may undergo unnecessary antifungal therapy or repeated biopsies before receiving appropriate management [22,24]. Such delays have consequences: approximately 17–40% of guttate cases progress to chronic plaque psoriasis [5], and persistent inflammation can result in psychosocial distress and long-term pigmentary sequelae [10,14]. These disparities highlight the need for culturally competent dermatologic education. Clinicians must recognize that "erythematous" conditions do not appear the same in brown or Black skin. Representation of SOC in educational resources and atlases remains limited but is critical [32]. Dermoscopy may enhance recognition, and studies such as Krefting et al. demonstrate that accuracy improves when clinicians emphasize distribution, scale, and history rather than color alone [6]. Community education is equally important: Nicholas et al. found that some patients believed "Black people do not get psoriasis," leading to delayed care [1].

Empowering patients with knowledge that psoriasis occurs in all skin tones may promote earlier consultation. Management also requires attention to precipitating factors and comorbidities. Streptococcal infection remains the most common trigger, and treatment of active infection can accelerate resolution [3]. Although antibiotics are not a cure, untreated strep may prolong or recur, and tonsillectomy may be considered in recurrent cases [43]. Similarly, control of HIV with antiretroviral therapy improves outcomes, reinforcing the importance of optimizing immune status [19]. Preventive strategies such as prophylactic antibiotics or tonsillectomy may benefit select patients, though evidence is limited [43]. Access to treatment is another area of disparity. Yadav, et al. [44] reported that psoriasis patients of color in North America were less likely to receive advanced therapies despite similar severity. Barriers include access, cost, and referral bias. Equity requires ensuring SOC patients have the same opportunity for biologics and phototherapy as White patients [45,46]. Evidence does not suggest reduced efficacy of modern biologics in SOC; outcomes appear comparable [1]. Practical adjustments such as lower initial phototherapy doses to prevent burns [1] and accommodating patients with limited resources through flexible scheduling can improve adherence and outcomes. From a mechanistic standpoint, guttate psoriasis exemplifies infection-driven autoimmunity. Streptococcal superantigens activate broad T-cell repertoires, including skin-homing subsets that cross-react with keratin peptides and initiate lesion development.

Nearly all guttate patients carry HLA-C*06:02, which efficiently binds streptococcal peptides, supporting a genetic predisposition [47]. A 2022 Science study showed that Group A Streptococcus can induce skin-resident T cells in susceptible individuals, which later mediate psoriasis flares [35]. Flora and Frew further proposed that early biologic therapy—particularly IL-17 inhibitors targeting tissue-resident memory T cells—may reduce progression to chronic disease if administered immediately after a flare [33]. Figure 3 demonstrates this immunopathogenesis and highlights intervention points, including infection control and immune modulation. Streptococcal infection (often tonsillitis) provides the inciting antigens. Superantigens

activate tonsillar T cells, including skin-homing subsets that migrate to the skin. There, they cross-react with keratinocyte epitopes or are re-stimulated by streptococcal antigens, releasing pro-inflammatory cytokines (IFN-γ, IL-17, TNF-α) that generate guttate papules [28]. This mechanism explains the temporal link between infection and rash and highlights potential interventions such as eradicating infection (antibiotics or tonsillectomy) and modulating immune responses (e.g., immunosuppressants or biologics) [35]. Most studies published between 2015–2025 are small and lack SOC-specific analysis. Large-scale trials are needed to assess whether disease progression, treatment responses, and adherence differ across populations.

The psychosocial impact of pigmentary sequelae also warrants investigation; SOC patients may experience heightened quality-of-life impairment from visible hyperpigmentation even after disease resolution. Patient-reported outcome studies could inform more holistic management strategies, including cosmetic camouflage or procedures to address pigmentation. Multidisciplinary care and patient education are crucial for improving outcomes. Training primary care physicians and pediatricians to recognize guttate psoriasis in SOC may facilitate earlier dermatology referral. Educational materials and patient resources should depict psoriasis in diverse skin tones to resonate with SOC patients. Psoriasis advocacy groups are encouraged to engage communities of color to dispel stigma and promote awareness. Encouragingly, resources such as Fast Facts: Dermatoses in Skin of Color are expanding representation and may reduce diagnostic delays [48,49]. In summary, guttate psoriasis in skin of color underscores how differences in clinical presentation, healthcare access, and research inclusion shape patient outcomes. Recognition of these challenges, combined with culturally competent education, equitable access to treatment, and inclusion of diverse populations in research, is essential. These themes frame the broader implications discussed in the conclusion, where we highlight priorities for clinical practice and future investigation.

Conclusion

Guttate psoriasis is an acute subtype marked by drop-like papules, typically triggered by infections such as streptococcal pharyngitis. This condition poses distinct diagnostic and clinical challenges in patients with skin of color (SOC) due to its underrecognized presentation. Lesions in SOC often appear violaceous, gray-brown, or hyperpigmented rather than the classic erythematous appearance seen in lighter skin. This variance can lead to misdiagnoses, frequently confusing guttate psoriasis with tinea corporis, pityriasis rosea, or lichen planus.

This review underscores the critical need for dermatologists to maintain a high index of suspicion when evaluating papulosquamous eruptions in SOC. Dermoscopy can be a valuable diagnostic aid, revealing dotted vessels and a fine gray-white scale even when erythema is not visible clinically. Biopsy remains a helpful tool, although it is more frequently used in SOC patients due to diagnostic uncertainty, despite similar histologic patterns to plaque psoriasis, such as psoriasiform hyperplasia and Munro microabscesses. Management of guttate psoriasis in SOC mirrors general treatment guidelines. Still, it requires added sensitivity to cultural and cosmetic concerns—especially around post-inflammatory hyperpigmentation, which is common and often distressing in SOC patients. This emphasis on cultural sensitivity in treatment is crucial to making the audience feel empathetic and understanding. Topical corticosteroids remain first-line for limited disease, but caution is needed to avoid steroid-induced dyspigmentation.

Narrowband UVB phototherapy is highly effective and safe across skin types, though dosing must be adjusted in SOC due to the lack of visible erythema as a marker for burning. Systemic therapies—including methotrexate, cyclosporine, and newer oral agents like apremilast—are employed for more extensive disease. Biologics (e.g., IL-17 and IL-23 inhibitors) have demonstrated rapid clearance in recalcitrant or severe cases and are gaining favor in specific populations, though disparities in biologic access persist for SOC patients. Treating streptococcal infections remains an adjunctive step, with some instances warranting tonsillectomy in patients with recurrent triggers. Significantly, guttate psoriasis affects all age groups. Pediatric patients often present following infections and may be misdiagnosed due to the subtle clinical presentation. In the elderly, guttate psoriasis is rarer but warrants thorough trigger evaluation. Immunocompromised patients, such as those with HIV or on immunosuppressants, require individualized regimens balancing efficacy and immune safety. Across these demographics, the proactive approach of early diagnosis and trigger management can influence disease progression and prevent evolution to chronic plaque psoriasis. In conclusion, improving recognition of guttate psoriasis in SOC is essential for equitable dermatologic care. Enhanced training on SOC presentations, the stress on the need for diverse imagery in education, and awareness of treatment nuances are critical to closing existing care gaps.

This stresses the need for diverse imagery in education, which is crucial to making the audience feel included and represented. Future research should explore whether early biologic use can alter disease trajectory in SOC patients and investigate strategies to minimize pigmentary sequelae post-treatment. By combining clinical vigilance with culturally competent care, clinicians can ensure timely diagnosis, appropriate therapy, and improved quality of life for all patients with guttate psoriasis.

Conflicts of Interest

The authors declare no conflicts of interest.

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