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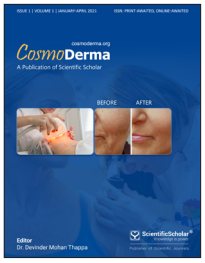

Review Article

Systemic corticosteroids in vitiligo: Evidence-based insights and practical considerations

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ABSTRACT

Vitiligo is an acquired pigmentary disorder marked by the progressive destruction of melanocytes, leading to depigmented patches. Autoimmune mechanisms are recognized as the primary pathogenic driver. Systemic corticosteroids have been employed as immunomodulatory agents, particularly in cases of rapidly progressive or active disease, to halt melanocyte loss and promote repigmentation. This review provides a comprehensive, evidence-based overview of their role in vitiligo management, covering therapeutic regimens, efficacy, safety, and clinical application. A structured literature search of PubMed, Scopus, Web of Science, and Cochrane Library databases was conducted in accordance with Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines, including clinical trials, meta-analyses, observational studies, and practice guidelines. Findings indicate that systemic corticosteroids – especially in pulse regimens such as dexamethasone oral mini-pulse (OMP) and betamethasone OMP – are effective in arresting disease progression and achieving partial repigmentation, with a more favorable safety profile than continuous daily therapy. Careful patient selection, appropriate dosing schedules, and regular monitoring are crucial to balancing efficacy and minimizing adverse effects. Overall, systemic corticosteroids remain a cornerstone in the management of active vitiligo, often serving as an adjunct to other therapeutic modalities for optimal outcomes.

Keywords: Dexamethasone, Oral mini-pulse, Pulse therapy, Systemic corticosteroids, Vitiligo

INTRODUCTION

Vitiligo is a chronic, acquired pigmentary disorder that affects approximately 0.5–2% of the global population, characterized by the selective destruction of melanocytes resulting in well-demarcated depigmented macules and patches on the skin and mucous membranes. Although it is a benign condition, vitiligo carries substantial psychosocial burdens, particularly in dark-skinned populations, due to the stark cosmetic disfigurement and social stigma associated with its appearance.[1]

The pathogenesis of vitiligo is multifactorial, with autoimmune, oxidative stress, neural, and genetic components contributing to melanocyte destruction. The autoimmune hypothesis remains the most widely accepted, supported by the presence of melanocyte-specific antibodies and T-cell infiltration in vitiliginous lesions.[2] This immunological basis has informed the rationale for using immunomodulatory agents, including corticosteroids, as part of therapeutic interventions in vitiligo.

While topical corticosteroids and calcineurin inhibitors are the mainstay for localized disease, systemic therapies become indispensable in cases of rapidly spreading or extensive vitiligo,

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where the aim is to arrest disease progression and induce repigmentation. Systemic corticosteroids, particularly when administered in carefully tailored pulse regimens, have been found to be effective in stabilizing disease activity, especially during the early inflammatory phase.[3]

Despite their efficacy, systemic corticosteroids pose a challenge in dermatologic practice due to their well-documented adverse effect profile, including metabolic, psychiatric, and bone-related complications. However, the advent of oral mini-pulse (OMP) therapy and intermittent dosing protocols has mitigated some of these risks, enabling their safer use in dermatologic contexts.[4]

Over the past two decades, a growing body of evidence has explored the use of systemic corticosteroids in vitiligo, evaluating various agents (e.g., dexamethasone, betamethasone, and prednisolone), regimens (daily vs. pulse), dosages, and combinations with phototherapy or other immunomodulators. Nevertheless, clinical consensus regarding optimal treatment protocols and long-term safety remains limited, particularly in special populations such as children, pregnant women, and the elderly.[5]

This review aims to provide a comprehensive, evidence-based synthesis of current knowledge regarding systemic corticosteroids in the treatment of vitiligo. We summarize the mechanistic rationale, clinical protocols, efficacy outcomes, adverse effects, and practical considerations, with the goal of guiding clinicians in optimizing therapeutic strategies while balancing safety and efficacy.

METHODOLOGY

This review was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines [Figure 1]. A comprehensive literature search was performed using four major databases: PubMed, Scopus, Web of Science, and the Cochrane Library, covering studies published from January 2000 to June 2025. The final search was conducted in July 2025.

The following keywords and MeSH terms were used in various Boolean combinations: "vitiligo," "systemic corticosteroids," "oral corticosteroids," "pulse therapy," "dexamethasone," "betamethasone," "OMP," "immunosuppression," and "repigmentation."

Inclusion criteria

- Human studies published in English.
- Randomized controlled trials (RCTs), cohort studies, retrospective studies, case series (≥10 patients), and meta-analyses.
- Studies assessing systemic corticosteroids in vitiligo (as monotherapy or in combination).

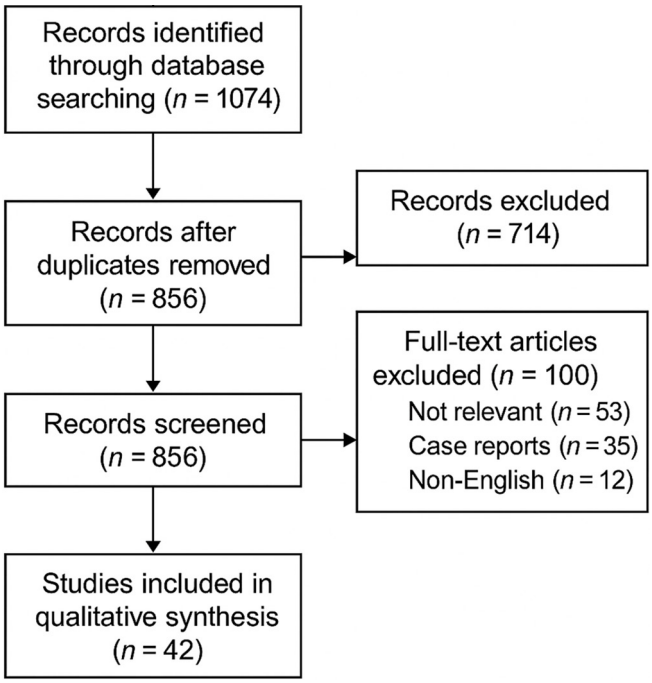

Figure 1: Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) flow diagram.

- Studies reporting clinical outcomes, including stabilization, repigmentation, or adverse effects.

Exclusion criteria

- Animal studies, conference abstracts, case reports, or expert opinions.
- Studies focusing exclusively on topical corticosteroids or non-corticosteroid systemic immunosuppressants.
- Non-English articles.

Study selection and data extraction

After removal of duplicates, two independent reviewers screened the titles and abstracts for relevance. Full-text articles were retrieved for potentially eligible studies. Disagreements were resolved by consensus or consultation with a third reviewer.

Key data extracted from each study included:

- Author(s), publication year, and country.
- Study design and sample size.
- Type and regimen of systemic corticosteroid.
- Duration of treatment and follow-up.
- Primary outcomes: Disease stabilization and repigmentation rates.
- Secondary outcomes: Adverse events, recurrence rates, and combination therapies.

Quality assessment

The Cochrane risk of bias tool was applied to RCTs. Observational studies were evaluated using the Newcastle-Ottawa scale. Only studies with moderate to high quality were included.

PATHOGENESIS OF VITILIGO AND RATIONALE FOR CORTICOSTEROID USE

Vitiligo is widely recognized as a complex autoimmune skin disorder characterized by the progressive destruction of epidermal melanocytes. Although the exact etiology remains incompletely understood, multiple interrelated mechanisms have been implicated in disease initiation and propagation, including autoimmunity, oxidative stress, genetic predisposition, neurogenic factors, and intrinsic melanocyte abnormalities.[6]

Autoimmune mechanisms

The strongest evidence supporting an autoimmune origin of vitiligo comes from the detection of melanocyte-specific autoantibodies and autoreactive cytotoxic $CD8^+$ T cells in affected individuals. These T cells target melanocyte differentiation antigens such as tyrosinase, gp100, and MART-1. Their presence correlates with disease activity and is supported by histological findings of perilesional lymphocytic infiltrates.[7] Moreover, vitiligo often coexists with other autoimmune diseases such as autoimmune thyroiditis, type 1 diabetes mellitus, and alopecia areata, further reinforcing its autoimmune nature.[8]

Oxidative stress and melanocyte vulnerability

Elevated levels of reactive oxygen species and markers of lipid peroxidation have been observed in both lesional and non-lesional skin, suggesting oxidative stress plays a pivotal role in melanocyte damage. The deficiency of catalase and other antioxidant enzymes in vitiligo skin may render melanocytes more susceptible to oxidative injury and immune recognition.[9]

Genetic and epigenetic contributions

Genome-wide association studies have identified several genes associated with immune regulation and melanocyte biology in vitiligo, such as NLRP1, TYR, PTPN22, and FOXP3. These findings underscore the role of genetic susceptibility in facilitating both autoimmunity and melanocyte dysfunction.[10]

Neurogenic and inflammatory hypotheses

Neuropeptides such as substance P, released from peripheral nerve endings, may contribute to local immune activation and melanocyte apoptosis. Furthermore, elevated levels of pro-inflammatory cytokines such as interferon-gamma (IFN-γ), tumor necrosis factor-alpha (TNF-α), interleukin (IL)-1β, and CXCL10 are consistently detected in vitiliginous skin and systemic circulation, serving as potential mediators of T-cell recruitment and melanocyte killing.[11]

Rationale for systemic corticosteroid use

Given the autoimmune and inflammatory underpinnings of vitiligo, systemic corticosteroids are logical therapeutic agents, especially in the early, rapidly progressive stages of disease. Corticosteroids exert pleiotropic immunosuppressive and anti-inflammatory effects by:

- Inhibiting T-cell activation and proliferation.
- Suppressing the release of cytokines such as IFN-γ, TNF-α, and IL-2.
- Reducing antigen presentation and leukocyte migration.
- Stabilizing lysosomal membranes and suppressing inflammatory gene transcription through nuclear factor kappa-light-chain-enhancer of activated B cells inhibition.[12]

In vitiligo, systemic corticosteroids are primarily employed to:

1. Arrest disease progression by suppressing autoreactive T cells and preventing the formation of new lesions.
2. Facilitate repigmentation by reducing perilesional inflammation, which may enable residual or follicular melanocytes to repopulate depigmented areas.
3. Prevent relapse, especially when used in early or active stages, systemic steroids may stabilize the autoimmune process long enough to allow adjunct therapies to work effectively.[13]

Systemic corticosteroid therapy is particularly beneficial in unstable or "inflammatory vitiligo," which is clinically characterized by trichrome lesions, confetti depigmentation, and koebnerization. Prompt initiation of therapy in such patients often halts further pigment loss and supports favorable outcomes.[14]

TYPES AND REGIMENS OF SYSTEMIC CORTICOSTEROID THERAPY IN VITILIGO

Systemic corticosteroids have emerged as a key therapeutic option for active, progressive vitiligo, with various regimens tailored to balance efficacy and safety. The principal strategies include daily oral administration and pulse therapy, particularly OMP regimens. Each protocol has unique pharmacologic considerations, dosing schedules, and risk profiles.

Commonly used corticosteroids

Several corticosteroids are utilized in systemic treatment of vitiligo, including:

- Dexamethasone: A long-acting corticosteroid with minimal mineralocorticoid activity; often used in OMP regimens.
- Betamethasone: Another potent, long-acting corticosteroid used similarly to dexamethasone.
- Prednisolone: Intermediate-acting corticosteroid; used in daily low-dose regimens or tapering courses.

The choice of agent depends on clinical preference, availability, and patient comorbidities.

OMP therapy

OMP therapy involves administering corticosteroids on 2 consecutive days/week (typically Saturday and Sunday), thereby minimizing cumulative dose and adverse effects.

- Dexamethasone OMP: Commonly used at doses of 2.5–5 mg/day on 2 consecutive days/week (total weekly dose 5–10 mg).
- Betamethasone OMP: Dosed at 5 mg on 2 consecutive days/week.

Advantages of OMP

- Minimizes hypothalamic-pituitary-adrenal (HPA) axis suppression.
- Reduces long-term adverse effects (e.g., weight gain and Cushingoid features).
- Improves compliance and quality of life.

Several studies have demonstrated that OMP therapy is effective in halting disease progression and achieving repigmentation in more than 60–80% of patients with active vitiligo.[15]

Daily low-dose regimens

Low-dose daily prednisolone (e.g., 0.3–0.5 mg/kg/day) is used in some patients with severe or rapidly spreading vitiligo. These regimens are typically administered for 2–3 months, followed by a tapering process.

- Efficacy: Good in rapid stabilization and partial repigmentation.
- Limitations: Higher incidence of systemic side effects, including weight gain, mood changes, acne, and hyperglycemia.

Due to the higher risk of adverse events, daily dosing is generally reserved for short-term use or in select patients who fail pulse therapy.[16]

High-dose pulse therapy

Some protocols advocate monthly intravenous methylprednisolone pulses (e.g., 1 g/day for 3 days/month), especially in extensive or refractory cases. However, this approach is not routinely practiced in dermatology due to resource constraints and limited long-term data.

Duration of treatment

- The optimal duration for systemic corticosteroid therapy remains debatable.
- Most studies advocate 3–6 months of therapy, after which tapering is initiated.
- Long-term therapy beyond 6 months is discouraged due to potential cumulative toxicity.

Disease activity should be reassessed monthly using clinical markers such as new lesion formation, Koebner phenomenon, or confetti-like depigmentation.[17]

Two main approaches are utilized in systemic corticosteroid therapy for vitiligo: Continuous daily dosing and OMP therapy [Table 1].

Comparative studies, such as that show that OMP therapy yields similar efficacy with fewer adverse effects compared to daily dosing, make it the preferred regimen for many dermatologists treating progressive vitiligo.[12,13]

Predictors of response

Response to systemic corticosteroids is more favorable in:

- Short-duration disease (<6 months).
- Younger patients.
- Presence of inflammatory signs (e.g., erythema and trichrome lesions).
- Absence of leukotrichia or bony prominences.

Clinical evidence and comparative studies

Multiple clinical studies, observational trials, and comparative analyses have assessed the efficacy and safety of systemic corticosteroids in the treatment of vitiligo. These studies provide important insights into disease stabilization rates, repigmentation outcomes, and the relative benefit-risk profiles of various corticosteroid regimens.

Efficacy of OMP therapy

One of the earliest studies supporting OMP therapy was conducted by Pasricha and Khaitan, who introduced OMP betamethasone (5 mg twice/week) in vitiligo patients.[11] They observed disease stabilization in over 89% of patients and significant repigmentation in nearly 40% over 3 months.[11]

Subsequent trials have validated these findings. In a prospective study of 100 patients with active vitiligo, Majid reported that 5 mg dexamethasone OMP halted disease activity in 91% of patients, and 60% achieved moderate to marked repigmentation within 12 weeks.[18]

Betamethasone OMP has also been studied. In a randomized trial comparing betamethasone OMP versus placebo, disease progression was arrested in 90% of treated patients, with 45% showing clinical improvement compared to 11% in controls.[19]

Daily low-dose oral corticosteroids

Several studies have investigated low-dose daily prednisolone regimens. In a cohort of 60 patients treated with 0.3 mg/kg/day prednisolone for 2 months followed by tapering, stabilization was achieved in 85%, and partial repigmentation was observed in 50% of patients.[18]

However, daily regimens are often associated with more frequent systemic adverse events, especially when extended beyond 2–3 months. This includes weight gain, moon facies, and transient glucose intolerance.

Long-term efficacy and relapse rates

Long-term follow-up studies indicate that once the disease is stabilized using corticosteroids, maintenance of repigmentation requires adjunctive therapy such as phototherapy or topical immunomodulators.

In one study, relapse occurred in up to 30% of patients within 12 months of steroid withdrawal, particularly in those with high baseline disease activity or poor adherence to follow-up care.[20]

Meta-analyses and systematic reviews

A meta-analysis of eight trials involving 542 patients concluded that oral corticosteroids significantly improve stabilization rates compared to placebo or no treatment (risk ratio: 2.87; 95% confidence interval: 2.01–4.02), with acceptable safety in short-term use.[21]

Another review emphasized that OMP therapy remains the most widely accepted and safe systemic approach for active vitiligo, especially in resource-limited settings.[22]

A recent systematic review of 42 studies (including 6 studies of OMP corticosteroids) indicated that systemic corticosteroid treatments are generally effective in halting vitiligo progression, especially with OMP regimens, and are associated with acceptable safety in many cases.[13]

Furthermore, combination therapies using corticosteroids along with narrowband ultraviolet B (NB-UVB) phototherapy or topical calcineurin inhibitors have demonstrated synergistic effects. A trial showed that OMP dexamethasone combined with NB-UVB led to faster repigmentation and higher patient satisfaction compared to monotherapy.[14]

Despite this evidence, it is important to note that systemic corticosteroids are not curative, and their primary role is to halt disease progression, especially in the early inflammatory phase. Their efficacy diminishes in long-standing stable vitiligo with significant melanocyte loss.

Overall, the clinical utility of systemic corticosteroids is well established in selected patients – particularly those with progressive, generalized, or segmental vitiligo not amenable to topical therapy alone.

SAFETY PROFILE AND ADVERSE EFFECTS

The systemic use of corticosteroids in vitiligo is generally considered safe when employed in judiciously selected patients and under careful medical supervision. This is particularly true for low-dose or OMP regimens, which have shown a favorable balance between efficacy and tolerability. Nonetheless, corticosteroids are not without risk, and clinicians must remain vigilant for adverse effects that may arise even at lower or intermittent dosing schedules.[23]

Commonly reported side effects in patients receiving corticosteroids for vitiligo include weight gain, moon facies, acneiform eruptions, and mild gastrointestinal disturbances such as gastritis or dyspepsia. In a prospective study by Majid, 12% of patients developed noticeable weight gain, while 9% reported gastritis and 5% experienced acne.[18] Fortunately, these effects were generally mild and reversible on dose reduction or discontinuation of therapy. Facial puffiness and menstrual irregularities, particularly in women of reproductive age, are also observed with systemic corticosteroid use, alongside mood disturbances such as irritability, anxiety, or insomnia, which tend to resolve after treatment cessation.[18]

Corticosteroids also impact endocrine and metabolic function, including glucose metabolism and bone homeostasis. In some patients, especially those receiving daily regimens or higher cumulative doses, hyperglycemia or dyslipidemia may be observed, warranting periodic fasting glucose and lipid profile monitoring. Prolonged corticosteroid exposure can lead to reduced bone mineral density, increasing the risk for osteopenia or osteoporosis. Calcium and Vitamin D supplementation, along with weight-bearing exercise, may be recommended for patients on extended therapy. Although HPA axis suppression is uncommon with OMP therapy, it remains a theoretical risk, particularly in regimens extending beyond 3–6 months, and may necessitate morning cortisol monitoring if clinical suspicion arises.[21]

In pediatric patients, systemic corticosteroids raise additional concerns. The potential for growth suppression, delayed puberty, and behavioral changes such as hyperactivity or mood lability requires careful dosing, close clinical follow-up, and clear communication with families. Nevertheless, OMP regimens using dexamethasone at 0.1 mg/kg twice weekly have demonstrated a reasonable safety profile and

can be effective in arresting rapidly progressive vitiligo in children. Growth chart monitoring and psychosocial support are essential components of pediatric care.[9]

Psychiatric and neurologic adverse effects, while less common, may include steroid-induced mood swings, anxiety, or, in rare cases, psychosis. These effects are more likely with higher or prolonged dosing but may occur even at low doses in predisposed individuals. Patients with a personal or family history of psychiatric illness should be screened before initiation, and early warning signs should prompt dose adjustment or discontinuation.[11]

Absolute contraindications to systemic corticosteroid use include active systemic infections such as tuberculosis, uncontrolled diabetes mellitus, peptic ulcer disease, and hypersensitivity to corticosteroids. Relative contraindications include hypertension, glaucoma, osteoporosis, and a known history of psychiatric illness. As a precaution, baseline investigations including blood pressure, fasting blood glucose, and liver function tests should be obtained before starting therapy.[4]

Finally, appropriate tapering of systemic corticosteroids is essential to avoid both disease flare-up and the risk of adrenal insufficiency. Abrupt cessation, especially after prolonged therapy, may provoke rebound depigmentation or systemic symptoms. Tapering schedules should be individualized based on the initial dose and duration of treatment; in OMP protocols, gradual reduction over 4–6 weeks is generally well tolerated.

Pulse therapy and safety

The OMP strategy has emerged as a safer alternative to traditional daily dosing. By administering corticosteroids (typically dexamethasone 2.5–5 mg) only 2 days/week, OMP regimens significantly reduce cumulative steroid exposure, thereby minimizing side effects while maintaining immunosuppressive efficacy.[9,16]

Several studies report minimal adverse events with OMP therapy, the most common being transient weight gain, mild acne, and gastritis. Importantly, HPA axis suppression is rare with short-term or pulse use, though it may still occur with long-term therapy exceeding 3–6 months [Table 1].[17]

Table 1: Comparative table: Daily versus pulse systemic corticosteroid therapy in vitiligo.

Parameter	Daily dosing	Pulse dosing (mini-pulse)
Common drugs used	Prednisolone	Dexamethasone, betamethasone, methylprednisolone
Typical dosage	0.3–0.5 mg/kg daily	2.5–5 mg twice weekly (usually on two consecutive days)
Treatment duration	2–3 months (sometimes longer with tapering)	3–6 months or longer
Onset of disease control	Rapid control in active disease	Effective control, especially in early or progressive disease
Efficacy in repigmentation	Moderate to good, particularly in combination with phototherapy	Comparable or better when initiated early
Side effects	More frequent (weight gain, hypertension, acne, mood changes)	Fewer and milder (gastritis, menstrual irregularity)
Relapse rate after withdrawal	Moderate to high	Lower than daily in some studies
Patient tolerability	Lower due to daily intake and cumulative dose	Better due to lower cumulative steroid load
Compliance	May be reduced due to the daily burden	Generally better (twice weekly)
Common indications	Rapid disease control in severe or widespread vitiligo	Progressive or early vitiligo; maintenance after disease arrest
References	Imran *et al.*[13], Davoudi *et al.*[12], Bae *et al.*[20]	Pasricha and Khaitan,[11] Majid,[18] Sharma *et al.*[22]

SPECIAL POPULATIONS: PEDIATRIC, PREGNANT, AND ELDERLY PATIENTS

The therapeutic use of systemic corticosteroids in vitiligo requires nuanced consideration in special populations, particularly pediatric patients, pregnant or lactating women, and elderly individuals. Each group presents unique physiological and safety challenges that must be addressed to ensure effective and safe treatment.[23]

In pediatric patients, vitiligo can have a profound psychological and social impact, especially when it affects visible areas such as the face or hands. Systemic corticosteroids may be indicated in rapidly progressive or unstable vitiligo when topical therapies fail or are impractical

Table 2: Summary table of included studies on systemic corticosteroids in vitiligo.

Study (Author, year)	Study design	Population	Intervention	Duration	Outcome measures	Key findings
Silverberg *et al.*, 2011[8]	Retrospective study	17 pediatric patients	Oral mini-pulse dexamethasone	3–6 months	Repigmentation rate, side effects	Significant repigmentation in the majority; mild, transient side effects.
Alghamdi *et al.*, 2012[9]	Literature review	Multiple studies	Prednisone, dexamethasone (daily/pulse)	Variable	Clinical efficacy, relapse rate, side effects	Pulse therapy is effective in early/progressive vitiligo; fewer side effects.
Davoudi *et al.*, 2009[12]	Prospective trial	30 patients with active vitiligo	Prednisolone (oral, 0.3 mg/kg daily)	2 months	Disease progression, repigmentation	Arrested disease in 76%; repigmentation in 60%.
Pasricha and Khaitan, 1993[11]	Observational	400 patients	Oral mini-pulse betamethasone (5 mg twice/wk)	6–12 months	Arrest of progression, pigmentation	89% achieved disease control; early intervention was most effective.
Imran *et al.*, 2013[13]	Comparative study	60 patients	Daily vs. pulse prednisolone	2 months	Disease arrest, repigmentation	Both regimens are effective; pulse is better tolerated with fewer adverse effects.
Majid, 2010[18]	Open trial	100 patients	Oral mini-pulse methylprednisolone	3–6 months	Arrest of progression, pigmentation	76% had stabilization; early vitiligo responded best.
Sharma *et al.*, 2015[22]	Retrospective analysis	400 patients	Oral mini-pulse betamethasone	Variable	Disease activity, pigmentation	85% stabilization; side effects in 12% mostly mild.
Bae *et al.*, 2016[20]	Systematic review/ meta-analysis	15 studies	Various systemic steroids	Variable	Efficacy, adverse events	Overall effectiveness confirmed; pulse regimens had superior tolerability.
Rathod *et al.*, 2011[21]	Observational	100 patients	Low-dose oral mini-pulse therapy	6 months	Repigmentation, adverse events	Comparable efficacy with fewer side effects than the conventional dose.
Taieb *et al.*, 2013[24]	Expert consensus (VETF)	N/A	Systemic corticosteroids	N/A	Therapeutic recommendation	Suggested early corticosteroids for progressive vitiligo; pulse preferred.

due to extensive involvement. However, children are more susceptible to corticosteroid-related adverse effects, including growth suppression, delayed pubertal development, and behavioral disturbances. OMP therapy, particularly using dexamethasone at 0.1 mg/kg on 2 non-consecutive days/week, has shown favorable outcomes in arresting disease progression with a relatively low incidence of adverse effects. Clinical monitoring should include growth velocity assessments, pubertal staging, and periodic evaluation for metabolic disturbances. Parental counseling is critical to ensure adherence and to address concerns about long-term safety.[25]

Pregnancy presents a more complex scenario. The use of systemic corticosteroids during pregnancy is generally reserved for refractory or severe cases of vitiligo and should only be considered when the potential benefits outweigh the risks to the fetus. Although corticosteroids such as prednisone are considered relatively safe in pregnancy due to their inactivation by placental enzymes, there remains a small but documented risk of orofacial clefts, low birth weight, and maternal complications such as gestational diabetes and hypertension. Low-dose, short-duration regimens are preferred, and topical alternatives should be exhausted before initiating systemic therapy. During lactation, systemic corticosteroids can be used with caution. Studies have shown that low-dose prednisone or dexamethasone yields minimal transfer into breast milk and is unlikely to cause harm to the infant. However, feeding should ideally be delayed by 3–4 h after dose ingestion to minimize exposure.[24,26]

In the elderly, the approach to systemic corticosteroid therapy must account for age-related physiological decline and the higher baseline prevalence of comorbidities such as hypertension, diabetes, osteoporosis, and cardiovascular disease. These factors increase the risk of corticosteroid-induced complications, including fluid retention, glucose intolerance, and bone demineralization. Therefore, the threshold for initiating systemic therapy in elderly patients is higher, and the treatment duration should be as short as possible with close

monitoring. Dose adjustments may be necessary based on renal and hepatic function. Bone density assessment, regular blood pressure checks, and fall risk evaluations are prudent measures to mitigate complications.[27,28]

Overall, systemic corticosteroids can be used in special populations with careful risk-benefit assessment, appropriate dose modifications, and vigilant follow-up. Multidisciplinary collaboration – particularly with pediatricians, obstetricians, and geriatricians – can enhance treatment safety and optimize outcomes in these vulnerable groups. Summary table of some included studies of systemic corticosteroids in vitillgo [Table 2].

Contraindications and monitoring

Systemic corticosteroids are contraindicated in patients with uncontrolled metabolic disorders, active infections, psychosis, or advanced osteoporosis. Baseline assessment should include blood glucose, blood pressure, liver function tests, and bone density scans, especially in high-risk patients.[29] Guidelines stress routine monitoring every 4–6 weeks during therapy.

Duration of therapy

There is a uniform consensus that systemic corticosteroids should be limited to short-term usage, ideally not exceeding 12 weeks, with careful tapering based on disease activity.[25,27,30] Pulse therapy regimens are preferred over daily dosing due to their lower risk of HPA axis suppression.

COMBINATION THERAPIES AND ADJUNCTIVE OPTIONS

The management of vitiligo, particularly in its progressive or extensive forms, often necessitates a multimodal therapeutic strategy. While systemic corticosteroids are effective in halting disease progression, their combination with other treatment modalities can enhance repigmentation outcomes, reduce corticosteroid dosage, and minimize adverse effects. Increasingly, clinicians are adopting combination approaches to achieve better disease control and cosmetic improvement.[2]

One of the most commonly employed adjuncts to systemic corticosteroid therapy is NB-UVB phototherapy. The immunomodulatory and melanocyte-stimulating properties of NB-UVB make it an ideal complement to corticosteroids. When used concurrently, systemic corticosteroids suppress the inflammatory process responsible for melanocyte destruction, while NB-UVB promotes melanocyte proliferation and migration from hair follicles. This synergistic effect has been supported by several clinical trials. For example, a randomized controlled trial demonstrated that the combination of OMP dexamethasone and NB-UVB phototherapy led to superior repigmentation rates and earlier disease stabilization compared to either treatment alone.[31]

Topical therapies are also frequently used alongside systemic corticosteroids to enhance local efficacy and reduce the systemic dose burden. Topical corticosteroids, calcineurin inhibitors (such as tacrolimus or pimecrolimus), and Vitamin D analogs may be applied to localized lesions while systemic corticosteroids address widespread activity. This approach is particularly valuable in facial or genital vitiligo, where systemic treatment may be excessive, yet local immune modulation is still required. A split-body study using systemic steroids and topical tacrolimus on one side versus steroids alone on the other showed better repigmentation in the combined treatment group, highlighting the potential of localized adjuncts.[32]

In select cases, antioxidants such as Vitamins C and E, alpha-lipoic acid, and ginkgo biloba have been used as supportive therapy to mitigate oxidative stress, a known contributor to melanocyte apoptosis in vitiligo. Although evidence remains preliminary, these agents may support overall treatment goals when used in conjunction with systemic corticosteroids. Similarly, zinc and folic acid supplementation has been explored, particularly in patients with confirmed deficiencies or as part of holistic care models.

Recent advancements have also introduced the possibility of using Janus kinase (JAK) inhibitors, both topical and systemic, in refractory vitiligo. Although not yet standard care, their immunosuppressive mechanisms offer potential synergy with corticosteroids, especially in difficult-to-treat cases. However, data on their combined use are limited, and safety concerns necessitate caution.[33]

In children or patients reluctant to use phototherapy, systemic corticosteroids may be combined with herbal or traditional medicine under supervision, though robust evidence for these combinations remains sparse. It is critical to evaluate each adjunctive therapy for potential interactions, cumulative toxicity, and patient-specific contraindications.[34]

Ultimately, combination therapy in vitiligo should be tailored based on disease extent, activity level, age, comorbidities, and patient preferences. The strategic use of corticosteroids with adjunctive treatments holds promise for achieving optimal outcomes with fewer side effects, making individualized multimodal therapy the cornerstone of modern vitiligo management.

GUIDELINES AND CONSENSUS STATEMENTS ON SYSTEMIC CORTICOSTEROID USE IN VITILIGO

Systemic corticosteroids are primarily reserved for patients with rapidly progressive vitiligo, where they are used to arrest disease activity rather than to induce repigmentation.

The International Vitiligo Task Force, in its 2023 global position statement, recommends the OMP regimen with dexamethasone 2.5–5 mg or betamethasone 5 mg on 2 consecutive days/week for a minimum of 3 months, extendable to 6 months if needed, while discouraging long-term continuous dosing. The task force also strongly advises combining systemic corticosteroids with narrowband UVB phototherapy for optimal disease control.[35]

The British Association of Dermatologists (BAD) 2021 guideline likewise restricts systemic corticosteroids to patients with rapidly spreading vitiligo. It recommends oral betamethasone at approximately 0.1 mg/kg (commonly 5 mg) twice weekly on 2 consecutive days, administered for 3 months, followed by a taper over subsequent months. The BAD explicitly cautions against prolonged daily corticosteroid regimens due to the risk of systemic adverse effects and advocates concurrent use of NB-UVB phototherapy.[36]

The European Dermatology Forum and Vitiligo European Task Force consensus also supports OMP regimens in active vitiligo, highlighting their role in halting progression. Short-term daily prednisolone at 0.3 mg/kg/day may be considered in selected cases, although pulsed regimens are favored for improved safety. The consensus emphasizes that systemic corticosteroids should rarely be used in isolation, but rather as part of combination therapy strategies with phototherapy and topical agents.[37]

In the United States, the American Academy of Dermatology (AAD) acknowledges systemic corticosteroids as an option for patients with new or rapidly progressive vitiligo but provides less detailed dosing protocols compared with European and Indian recommendations. The AAD stresses that systemic therapy should be used only as short courses, usually not exceeding 3 months, and should not be continued long-term due to metabolic, skeletal, and psychiatric risks.[38]

The Indian Association of Dermatologists, Venereologists, and Leprologists (IADVL) consensus statement is one of the most detailed in terms of systemic corticosteroid protocols. It recommends either low-dose daily prednisolone (around 0.3 mg/kg/day for 1–3 months with tapering) or OMP regimens using dexamethasone 2.5–5 mg or methylprednisolone 32 mg on 2 consecutive days/week for 3–6 months. These regimens have been supported by multiple Indian case series and clinical studies showing effective disease stabilization with relatively few adverse effects. The IADVL strongly advises regular monitoring for systemic complications and generally recommends concomitant NB-UVB phototherapy as the standard of care.[39,40]

The Japanese Dermatological Association (2013 guidelines) also recognizes systemic corticosteroids for rapidly progressive disease, with OMP or pulse regimens used cautiously. In some refractory cases, intravenous methylprednisolone pulse therapy (e.g., 500 mg daily for 3 consecutive days monthly) has been reported, though this remains less standardized. Japanese guidelines emphasize individualizing therapy by age and comorbidity, with special caution in pediatric and elderly patients.[41]

The German S1 guideline (2022) similarly restricts systemic corticosteroids to short-term use in selected cases of active vitiligo. Both OMP and short daily regimens are considered acceptable, but the guideline prioritizes topical and phototherapy options where feasible. It underscores the risks of systemic adverse effects, particularly metabolic syndrome, and stresses structured monitoring and early transition to safer long-term modalities.[42]

Overall, despite regional variations in dosing, all major international, American, European, Indian, Japanese, and German guidelines converge on the principle that systemic corticosteroids are useful only in short-term regimens for rapidly progressive vitiligo, preferably as OMP schedules for 3–6 months, almost always in combination with phototherapy, and never as long-term maintenance monotherapy [Table 3].

LIMITATIONS OF CURRENT EVIDENCE AND FUTURE DIRECTIONS

Despite the widespread use of systemic corticosteroids in vitiligo management and their inclusion in numerous treatment guidelines, several limitations in the current body of evidence hinder the formulation of universally standardized protocols. These limitations relate to both the quality and quantity of data, as well as the lack of long-term studies assessing efficacy, safety, and relapse rates.

One of the most pressing challenges is the heterogeneity in study design across available clinical trials and observational studies. Many studies exploring the efficacy of systemic corticosteroids – particularly OMP regimens – suffer from small sample sizes, lack of blinding, or inadequate control groups. As a result, comparison across studies is difficult, and meta-analyses often face issues related to data pooling and bias. In addition, standardized outcome measures such as the vitiligo disease activity score, vitiligo area scoring index, or patient-reported quality of life indices are inconsistently applied, making objective comparison of results challenging.

Another limitation lies in the underrepresentation of certain patient populations in clinical trials, including children, pregnant women, and the elderly. These groups are often excluded due to ethical or safety concerns, leaving clinicians to rely on anecdotal evidence or extrapolated data. Furthermore, there is limited research on how comorbid autoimmune or metabolic conditions may influence corticosteroid efficacy or safety in vitiligo, despite these being common in affected individuals.

Table 3: Guidelines and consensus statements on systemic corticosteroid use in vitiligo.

Guideline/Society (Region)	Recommended systemic steroid regimen (example)	Typical duration	Notes/Practical points (evidence and safety)
International Vitiligo Task Force — Position statement (International, 2023)[35]	Oral mini-pulse (OMP): Dexamethasone 2.5–5 mg or betamethasone 5 mg on 2 consecutive days/week (weight-adjusted per protocol).	3 months (extendable to 6 months) with periodic reassessment.	Recommends OMP as first-line systemic option to halt rapid progression; use combined with NB-UVB when feasible; Avoid long continuous steroid courses; monitor metabolic/HPA parameters.
British Association of Dermatologists (BAD, 2021)[36]	Example: Oral betamethasone ~0.1 mg/kg (practical dosing often cited as 5 mg) twice weekly on 2 consecutive days.	Three months, then taper (BAD suggests structured taper; e.g., gradual reduction over the following months).	BAD restricts systemic steroids to rapidly progressive cases only, advises combining with NB-UVB; warns against prolonged daily dosing due to systemic adverse effects.
European Dermatology Forum/VETF (Europe)[37]	OMP (dexamethasone/ betamethasone) on 2 consecutive days/week; daily low-dose prednisolone (e.g., ~0.3 mg/kg/day) may be used short-term in selected cases.	Three months may extend to up to 6 months with strict monitoring.	Emphasizes early use in active disease to arrest spread, limit cumulative exposure, and combine systemic therapy with phototherapy/ topicals.
American Academy of Dermatology (AAD) — Clinical guidance/USA[38]	Short courses of oral corticosteroids (e.g., prednisolone/prednisone ≈0.3 mg/kg/day or intermittent OMP regimens) may be used for rapidly spreading disease.	Short courses (weeks to 3 months); long-term continuous use discouraged.	AAD endorses short-term systemic therapy to stop the spread but provides less regimented dosing than European statements; stresses monitoring and limiting duration due to metabolic/psychiatric risks.
Indian guidance/ expert consensus/ IJDVL literature (India)[39,40]	OMP commonly used (dexamethasone 2.5–5 mg on 2 consecutive days/week) or low-dose daily prednisolone ~0.3 mg/kg/day for short courses.	3–6 months in many Indian series/consensus documents; tailored per response.	Numerous large Indian case series and pilot RCTs support OMP effectiveness and tolerability; the Indian consensus often gives more practical dosing examples and emphasizes combination with NB-UVB and close monitoring in children.
Japanese Guidelines (Japan, 2013)[41]	Systemic corticosteroids (pulse or OMP) may be used in rapidly progressive cases; pulse IV methylprednisolone described in small series for refractory cases (e.g., 500 mg/day ×3 days).	Variable — short courses; some reports use pulse cycles (monthly) or OMP for months.	Japanese guideline endorses cautious, individualized systemic use with dose adjustment for age/comorbidity; highlights alternative interventions (phototherapy, topical agents) when possible.
German S1 Guideline (DDG, 2022)[42]	Short-term systemic steroids (OMP or short daily courses) as an option to arrest activity; prefer topical/ phototherapy when possible.	Short-term (commonly ~3 months); emphasize minimizing cumulative exposure.	S1 highlights risks of metabolic complications and recommends restriction to selected active cases; suggests combination therapy and structured monitoring.

OMP: Oral mini-pulse, NB-UVB: Narrowband ultraviolet B, BAD: British Association of Dermatologists, HPA: Hypothalamic-pituitary-adrenal, IV: Intravenous, RCTs: Randomized controlled trials, AAD: American Academy of Dermatology

Long-term safety data on systemic corticosteroids in vitiligo are sparse. Most studies focus on short-term endpoints such as disease stabilization and initial repigmentation, without adequate follow-up to assess relapse rates or adverse effects after discontinuation. Given the well-known complications of corticosteroids – including metabolic, endocrine, musculoskeletal, and psychological effects – future research must extend observation periods to better understand the risk-benefit profile of such therapies over time.

There is also an unmet need for comparative studies that assess corticosteroids against newer therapeutic agents such as JAK inhibitors, biologics, or emerging small molecules. As the treatment landscape for vitiligo continues to evolve, systemic corticosteroids will need to be evaluated not only in isolation but also in the context of multimodal and personalized treatment approaches. Trials exploring head-to-head comparisons or combination regimens could provide more practical insights into optimizing treatment sequences and combinations.

From a mechanistic standpoint, additional research is needed to elucidate precisely how systemic corticosteroids modulate the immune environment in vitiligo, particularly at the

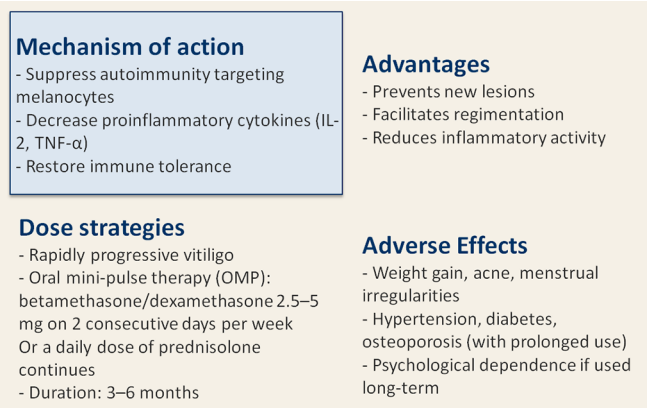

Figure 2: Summary of systemic corticosteroid in vitiligo.

molecular level. The emerging understanding of the disease as a complex neuro-immuno-cutaneous disorder involving T-cell dysregulation, oxidative stress, and neural peptides suggests multiple potential targets beyond glucocorticoid pathways. Advanced imaging, transcriptomic profiling, and biomarker studies could help refine patient stratification and predict treatment response.

Finally, despite being a global condition, most data come from specific geographic regions (e.g., South Asia) and may not generalize across diverse populations with different genetic, environmental, or cultural backgrounds. This geographic bias limits the external validity of findings and underscores the need for international, multicenter collaborations.

In summary, while systemic corticosteroids remain a cornerstone in the management of progressive vitiligo, significant evidence gaps persist. Future research should aim for rigorous, multicenter, long-term trials with standardized protocols and broader patient inclusion to optimize and personalize corticosteroid therapy in vitiligo.

CONCLUSION

Systemic corticosteroids have long held a pivotal role in the management of vitiligo, particularly in cases characterized by rapid progression, widespread involvement, or significant psychosocial impact. Their immunosuppressive and anti-inflammatory properties allow for disease stabilization and, in many cases, facilitate repigmentation when used appropriately. Among various regimens, OMP therapy with dexamethasone or betamethasone remains the most studied and preferred method due to its favorable balance of efficacy and safety, especially in halting disease activity with fewer systemic side effects [Figure 2].

Despite their clinical utility, the use of systemic corticosteroids must be carefully tailored to individual patient profiles, considering factors such as age, comorbidities, disease duration, and psychosocial burden. In special populations – namely, pediatric, pregnant, and elderly patients – risk-benefit assessments and close monitoring are paramount. Adjunctive strategies, including combination with phototherapy or topical agents, have shown synergistic effects and may enhance treatment outcomes while minimizing corticosteroid exposure.

Current guidelines and consensus statements reflect the value of systemic corticosteroids in vitiligo, yet also highlight the need for restraint, emphasizing short durations, dose minimization, and close clinical follow-up. However, limitations in existing evidence, such as small sample sizes, inconsistent methodologies, and a lack of long-term data, present significant challenges in standardizing care. Moreover, with the advent of novel therapies like JAK inhibitors and targeted biologics, corticosteroids must now be considered as part of an evolving therapeutic paradigm rather than a standalone solution.

Future research should aim to fill existing knowledge gaps through well-designed, multicenter randomized trials, with particular attention to long-term safety, relapse rates, and optimal combination strategies. Advances in understanding the immunopathogenesis of vitiligo may eventually guide more targeted use of corticosteroids or entirely new classes of therapeutics. Until then, systemic corticosteroids remain a valuable option when used judiciously and within the framework of evidence-based, patient-centered care.

Clinical recommendations

- Early initiation: Systemic corticosteroids should be considered early – ideally within 6 months of disease onset – when active spreading is evident.
- Preferred regimen: OMP therapy (e.g., dexamethasone 2.5 mg twice weekly) is preferred over daily dosing due to superior safety and tolerability.
- Close monitoring: Regular follow-up is critical to detect and manage potential side effects, especially in children and those on long-term therapy.
- Combination therapy: For enhanced repigmentation outcomes, corticosteroids should be combined with narrowband UVB phototherapy where available.
- Gradual tapering: Avoid abrupt discontinuation; instead, taper corticosteroids gradually once disease stabilization is achieved.
- Patient counseling: Educate patients about treatment goals, expectations, and potential side effects to foster compliance and shared decision-making.
- Pediatric and special populations: Use with caution in pediatric patients, and adjust doses based on weight and growth monitoring parameters.

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