



## Perioral Dermatitis: Clinical Considerations for Dental, Pharmacy, and Nursing Professionals

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### Abstract

**Background:** Perioral dermatitis is a common inflammatory facial dermatosis, clinically characterized by erythematous papules and pustules distributed around the mouth, nose, and eyes. It predominantly affects young adult females and is frequently linked to the use of topical corticosteroids, which can initiate a cycle of steroid dependence and rebound flares. The condition's multifactorial etiology also involves other triggers like fluorinated toothpaste, occlusive cosmetics, and skincare products.

**Aim:** This article aims to synthesize the clinical presentation, pathophysiology, and management of perioral dermatitis for an interprofessional audience, including dental, pharmacy, and nursing professionals. The goal is to enhance recognition, promote trigger identification, and guide effective, steroid-sparing treatment strategies to improve patient outcomes.

**Methods:** A comprehensive review of the condition is presented, covering its clinical features, histopathology, and epidemiology. Diagnostic approaches based on morphology and exposure history are detailed, alongside a systematic overview of management strategies, including topical and oral therapies, with a strong emphasis on corticosteroid cessation and trigger avoidance.

**Results:** The cornerstone of management is the immediate discontinuation of all facial corticosteroids, often requiring a structured taper to mitigate rebound flares. First-line therapies include topical metronidazole, calcineurin inhibitors, and azelaic acid. For moderate-to-severe cases, oral tetracyclines are highly effective. Adjunctive measures, such as simplifying skincare routines and substituting potential irritants, are critical for achieving and maintaining remission.

**Conclusion:** Perioral dermatitis is a frequently mismanaged condition that requires a coordinated, interprofessional approach. Successful outcomes depend on accurate clinical diagnosis, patient education trigger avoidance, and the consistent application of steroid-sparing therapeutic regimens to break the cycle of dependency and inflammation.

**Keywords:** Perioral dermatitis, topical corticosteroids, steroid-induced dermatitis, metronidazole, tetracyclines, calcineurin inhibitors, skincare, interprofessional care..

### Introduction

Perioral dermatitis is a benign yet often distressing inflammatory eruption characterized by clusters of small papules and pustules or ill-defined pink, scaly patches distributed predominantly around the mouth in young adult females. Although the perioral region is the prototypical site, clinically similar lesions frequently extend to the periocular and paranasal areas, which has prompted the broader term periorificial dermatitis to capture its typical anatomic spread (see Image. Perioral Dermatitis). The condition can present with burning or tightness rather than pruritus, and patients may report cosmetic impairment that disproportionately affects quality of life and adherence to care plans, underscoring the importance of sensitive counseling by dental, pharmacy, and nursing professionals [1].

Epidemiologically, the disorder is not confined to a single demographic; pediatric and male cases occur, often linked to topical or inhaled corticosteroids or cutaneous barrier disruption, making careful medication reconciliation and exposure history essential at first contact [2]. Pathophysiologically, perioral dermatitis appears to reflect a corticosteroid-modulated dysregulation of the innate cutaneous immune response and stratum corneum barrier, with downstream perturbations in microbial communities and neurovascular reactivity. Clinically meaningful triggers include potent or prolonged topical corticosteroids applied to the face; initial improvement may paradoxically entrench a cycle of reliance in which withdrawal precipitates rebound flares and chronicity [1]. Additional contributors—inhaled or intranasal steroids, fluorinated toothpaste, chewing gum,

occlusive or fragranced cosmetics, sunscreens, certain dental materials, prolonged facemask occlusion, and suboptimally fitted CPAP interfaces—warrant systematic review in interprofessional settings. Hormonal influences are suspected in cyclical flares, and barrier-disruptive behaviors such as over-cleansing or exfoliation can perpetuate disease activity [2].

Diagnosis is chiefly clinical, integrating lesion morphology, periorificial distribution, and a compatible exposure history. Biopsy is reserved for atypical or refractory presentations to exclude mimics such as acne vulgaris, rosacea, seborrheic dermatitis, allergic contact dermatitis, or granulomatous variants. A recurrent pitfall is mislabeling the eruption as eczematous dermatitis and prescribing facial steroids, a practice that offers short-lived relief but amplifies long-term disease via tachyphylaxis and rebound, thereby increasing iatrogenic harm [1]. For dentists and dental hygienists, recognition of perioral patterns at chairside—including perinasal extensions and angular involvement—can prompt early referral and counseling about product triggers, toothpaste formulations, and dental materials. Pharmacists play a central role in deprescribing facial steroids, identifying hidden steroid exposures (e.g., combination antifungal-steroid creams), and guiding evidence-based substitutions. Nurses are pivotal in patient education, monitoring adherence, and coaching on gentle skin care routines that restore barrier integrity [2]. Management begins with discontinuation of facial corticosteroids, preferably via a short taper to mitigate rebound when prior use has been frequent or high potency. First-line pharmacotherapy includes topical metronidazole or calcineurin inhibitors; oral tetracyclines are effective for moderate-to-severe or recalcitrant disease. Adjunctive steps—substituting non-fluorinated toothpastes when implicated, simplifying skin care to non-occlusive, fragrance-free products, optimizing mask or CPAP fit, and avoiding heavy cosmetics—reduce trigger load and enhance remission durability [1][2]. While outcomes are generally favorable, perioral dermatitis may follow a persistent or relapsing course; setting expectations about time-to-response and relapse-prevention strategies improves satisfaction and reduces unnecessary corticosteroid re-exposure. Across settings, coordinated, interprofessional stewardship—identifying triggers, halting steroid-driven cycles, and implementing tiered therapies—remains the cornerstone of safe, effective care for this common yet frequently mismanaged condition [1][2].



**Figure-1:** Perioral Dermatitis.

### Etiology

The etiology of perioral dermatitis remains multifactorial and incompletely elucidated, involving an

interplay between environmental, pharmacologic, hormonal, and microbiologic influences. Although the precise cause is unknown, clinical and epidemiological evidence consistently supports a link between external irritants and immunologic dysregulation within the pilosebaceous unit and epidermal barrier. The condition is increasingly recognized as a prototypical example of iatrogenic dermatosis, where therapeutic interventions—especially topical corticosteroids—initiate or perpetuate a chronic inflammatory response. Environmental exposures, cosmetic practices, and hormonal variations further compound the susceptibility, particularly among young women, who constitute the demographic majority of affected patients [1].

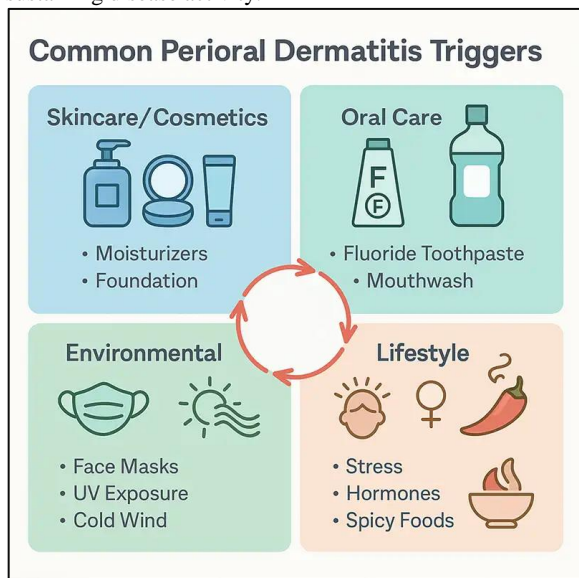
### Corticosteroids and Perioral Dermatitis

Among the most significant and well-established etiologic factors in perioral dermatitis is the use of corticosteroids, particularly when applied topically to facial skin. Numerous reports document that topical steroid use often precedes the onset of the eruption, establishing a temporal and causal relationship [1]. Initially, these agents suppress inflammation, leading to transient improvement. However, as treatment continues or ceases abruptly, the disease frequently recurs with greater severity, resulting in a phenomenon termed steroid dependence [2][3]. Chronic exposure to mid- or high-potency corticosteroids can progressively aggravate the inflammatory cycle, predisposing to a granulomatous subtype of perioral dermatitis characterized by deeper nodules and persistent erythema. The relationship between corticosteroids and perioral dermatitis extends beyond topical preparations. Nasal and inhaled corticosteroids, commonly prescribed for allergic rhinitis and asthma, have also been implicated as potential triggers [4][5]. This observation underscores the systemic nature of steroid exposure and its potential for periorificial cutaneous effects. The pathophysiologic mechanism remains speculative, but several hypotheses have been proposed. Corticosteroids are thought to induce epidermal barrier dysfunction, alter the innate immune response, and modify the cutaneous microbiome, particularly around hair follicles. This microbial dysbiosis may enable opportunistic colonization or inflammation involving *Candida albicans*, *Demodex* mites, or fusiform bacteria, all of which have been isolated in affected regions [6][7][8]. These findings support the theory that steroid exposure may set in motion a cascade of immunologic imbalance and microbial proliferation, ultimately manifesting as perioral dermatitis.

### Additional Potential Etiologies

Beyond corticosteroids, several environmental and lifestyle-related factors have been implicated in the pathogenesis of perioral dermatitis. One frequently reported association is the use of fluorinated toothpastes, which may provoke irritant or allergic reactions in perioral skin, either directly through contact or indirectly via inflammatory cross-talk between oral and perioral tissues [9]. Likewise, chewing gum and dental materials, such as amalgam or composite resins, have been cited as potential contributors, possibly due to mechanical irritation, salivary contamination, or hypersensitivity reactions to chemical components [10][11]. These associations are particularly relevant for dental professionals, who should remain vigilant when evaluating facial eruptions that may overlap with contact or irritant dermatitis of dental origin. Cosmetic and skincare products represent another well-documented group of triggers. The frequent combination of moisturizers, foundations, and occlusive sunscreens can create an

environment conducive to follicular occlusion and microinflammation [12][13]. This phenomenon is exacerbated by repetitive application of emollients or physical sunscreens that contain zinc oxide or titanium dioxide, which may disrupt skin pH balance and barrier function. Patients often report worsening symptoms after switching to new cosmetic regimens or during periods of heavy product layering, reinforcing the concept that product formulation and overuse are critical in precipitating or sustaining disease activity.



**Figure-2:** Etiologies of perioral dermatitis.

The marked female predominance of perioral dermatitis has prompted investigation into hormonal influences. While hormonal fluctuations may sensitize sebaceous and follicular units, some studies have intriguingly found that oral contraceptive pills can improve disease outcomes, possibly by stabilizing hormonal variations that otherwise aggravate the skin's inflammatory milieu [14]. Such findings indicate that endocrine balance, rather than simply female gender, may be a determining factor in disease expression. Emerging evidence also highlights the role of mechanical and environmental occlusion. During the COVID-19 pandemic, widespread and prolonged facemask use has been correlated with a notable increase in perioral dermatitis incidence [15]. Friction, humidity, and microbial trapping beneath masks likely contribute to this surge, illustrating how lifestyle adaptations can modulate dermatologic epidemiology. Similarly, the improper use of continuous positive airway pressure (CPAP) devices, particularly when associated with activated oxygen or ill-fitting interfaces, can provoke periorificial inflammation through both mechanical and chemical irritation [16]. In summary, the etiology of perioral dermatitis reflects a complex interaction between exogenous irritants, pharmacologic agents, microbial imbalances, and host susceptibility. Corticosteroids remain the most significant precipitant, but modern environmental exposures—from fluorinated oral hygiene products to occlusive facial devices—play increasingly recognized roles. Understanding these multifactorial influences is essential for accurate diagnosis, targeted prevention, and long-term disease control [1][2][3][4][5][6][7][8][9][10][11][12][13][14][15][16].

### Epidemiology

Perioral dermatitis demonstrates a characteristic demographic profile, with the highest burden observed among young adult females between approximately 20 and 45 years of age [17][18]. This pattern aligns with clinical impressions that intersect dermatologic, dental, and cosmetic exposures typical of this life stage, including frequent use of facial products, intermittent topical steroid application for cosmetic or eczematous flares, and lifestyle factors that may increase occlusion or barrier disruption. Although adult women represent the plurality of cases in specialty clinics, the condition is not confined to this group; pediatric presentations are well documented and should be considered in children with periorificial papulopustular eruptions that defy conventional acne or eczematous diagnoses [19]. In children, available data do not indicate a significant difference in frequency by sex or race, suggesting a more uniform susceptibility pattern in the pediatric population compared with adults, where female predominance is pronounced [20]. True incidence and prevalence are difficult to establish because perioral dermatitis is frequently underrecognized or misclassified as acne, rosacea, or eczematous dermatitis in primary care and dental settings. Health care utilization patterns can therefore skew apparent epidemiology, with referral centers reporting higher proportions of steroid-associated disease. Nonetheless, several recurring epidemiologic themes are evident across age strata. First, exposure to topical or inhaled corticosteroids remains a consistent antecedent in case series, implying that prescribing practices and over-the-counter access may influence observed rates in both community and tertiary contexts [17][18]. Second, product-related factors—including frequent use of occlusive cosmetics, emollients, and certain oral-hygiene formulations—appear to cluster among affected adults, potentially explaining the female predominance, whereas such exposures are more variable in children and may be mediated by caregiver habits or treatment for atopic conditions [19]. Third, the anatomic distribution around perioral, perinasal, and periocular sites means that dentists, pharmacists, and nurses often encounter early or subclinical stages; patterns of recognition and counseling in these frontline settings can shape time-to-diagnosis and thus the number of cases captured in formal datasets [20]. Overall, epidemiologic understanding supports a model in which age-linked behaviors and exposures, rather than immutable host factors, drive the adult female predominance, while pediatric cases exhibit more balanced demographic distribution across sex and race [17][18][19][20].

### Pathophysiology

The pathophysiology of perioral dermatitis remains incompletely defined, yet it is widely accepted to involve a complex interplay of inflammatory, microbial, and barrier-related mechanisms. The hallmark histopathologic feature is perifollicular and perivascular inflammation, leading to erythematous papules and papulopustules concentrated around the mouth, nose, and eyes [21]. These lesions reflect localized immune activation centered on the pilosebaceous units and small dermal vessels. The inflammatory infiltrate, typically composed of lymphocytes, histiocytes, and occasional neutrophils, produces visible erythema and papulation. Chronic cases may show granulomatous transformation, suggesting a sustained antigenic or microbial stimulus that perpetuates the immune response. The initiating event appears to involve disruption of the epidermal barrier and innate immune dysregulation. Exogenous irritants—especially topical corticosteroids—

alter keratinocyte function, reduce antimicrobial peptide expression, and impair barrier lipids. These changes render the skin more permeable to irritants and microorganisms, triggering an exaggerated inflammatory cascade. Corticosteroids, while initially suppressing inflammation, paradoxically cause rebound vasodilation and cytokine activation upon withdrawal, perpetuating the inflammatory cycle. Concurrently, microbial shifts within the follicular microenvironment, particularly involving *Demodex* mites, *Candida albicans*, and fusiform bacteria, may act as secondary amplifiers of inflammation, stimulating Toll-like receptor-mediated immune responses [21].

Hormonal and genetic factors also contribute to disease susceptibility. The predominance in women of reproductive age supports a hormonal influence, with fluctuations in estrogen and progesterone potentially modifying sebaceous gland activity and immune tone. Genetic predispositions that affect innate immunity, barrier integrity, or sebaceous function may further explain interindividual variability in disease onset and chronicity. Environmental factors—such as ultraviolet radiation, occlusive cosmetics, and humidity from facemasks—can intensify local irritation, microocclusion, and microbial overgrowth, collectively fueling inflammatory processes. Ultimately, perioral dermatitis emerges as a multifactorial disorder of barrier dysfunction and dysregulated inflammation, wherein environmental triggers and pharmacologic exposures interact with individual genetic and hormonal susceptibilities. The condition exemplifies how exogenous perturbations of cutaneous homeostasis can transform transient irritation into chronic dermatosis. Continued investigation into epidermal immune signaling and microbiome composition may clarify why certain patients develop recurrent disease and guide the development of targeted therapies [21].

### Histopathology

The histopathologic features of perioral dermatitis provide valuable insight into the underlying inflammatory processes that give rise to its clinical manifestations. Although the diagnosis is primarily clinical, biopsy findings can help confirm uncertain cases and differentiate perioral dermatitis from other papulopustular dermatoses such as acne vulgaris, rosacea, or seborrheic dermatitis. The characteristic histologic hallmark is a perifollicular and perivascular lymphohistiocytic infiltrate within the superficial and mid-dermis, occasionally accompanied by sparse plasma cells [22]. This inflammatory pattern correlates with the disease's folliculocentric presentation, where erythematous papules and pustules cluster around the pilosebaceous units in perioral, perinasal, and periocular regions. Notably, while follicular spongiosis—intercellular edema within the follicular epithelium—may be observed, the conventional eczematous features typical of dermatitis, such as pronounced epidermal spongiosis or parakeratosis, are often minimal or absent [22]. This histologic paradox underscores the misnomer embedded in the term “dermatitis,” as the inflammatory process in perioral dermatitis is primarily perifollicular rather than epidermal. The epidermis usually remains intact, showing only mild acanthosis or focal parakeratosis, with no significant exocytosis of inflammatory cells into the epidermal layers. The dermal infiltrate, composed predominantly of lymphocytes and histiocytes, reflects a cell-mediated immune response likely triggered by exogenous or microbial antigens.

A particularly important histologic subset is the granulomatous variant of perioral dermatitis, sometimes referred to as *granulomatous periorificial dermatitis*. In this form, dermal epithelioid granulomas and multinucleated giant cells coexist with the standard perivascular and perifollicular lymphohistiocytic infiltrates [23][24]. These granulomatous reactions suggest a more chronic or immune-mediated process, potentially driven by prolonged antigenic stimulation, foreign material deposition, or microbial persistence. The granulomas are typically non-caseating and lack the necrotic centers seen in infectious granulomatous diseases such as cutaneous tuberculosis or sarcoidosis, helping to distinguish this entity histologically. The presence of granulomatous inflammation may also indicate an exaggerated delayed-type hypersensitivity reaction, aligning with clinical observations of chronicity and resistance to conventional therapy in such cases. Occasionally, mild vascular dilatation and endothelial swelling are present, consistent with low-grade chronic inflammation. The absence of neutrophilic microabscesses or comedones further differentiates perioral dermatitis from acneiform eruptions. In summary, histopathologic examination of perioral dermatitis reveals a predominantly lymphohistiocytic inflammatory process centered on hair follicles and dermal vasculature, with variable granulomatous transformation in chronic or atypical presentations. The lack of prominent epidermal involvement challenges the traditional nomenclature of the disease, emphasizing instead its perifollicular nature. Recognition of these subtle but distinctive histologic features is essential for pathologists and clinicians alike, ensuring accurate differentiation from other facial papular dermatoses and guiding appropriate therapeutic strategies [22][23][24].

### History and Physical

Perioral dermatitis is primarily a clinical diagnosis, established through careful history-taking and focused cutaneous examination. Patients most often present with clusters of erythematous, grouped papules in a symmetric, sometimes unilateral, distribution around the mouth, nose, and eyes; the condition is therefore also referred to as periorificial dermatitis to emphasize involvement of multiple facial orifices. Lesions may be accompanied by fine scaling, occasional vesicles, and superficial pustules, creating a papulopustular texture on close inspection. A classic bedside clue is the sparing of the vermilion border of the lips—the erythematous papules encircle the mouth but halt at the sharp cutaneous–mucosal junction, producing a narrow, pale “halo” of uninvolved skin. Patients frequently volunteer a history of fluctuating intensity linked to topical products or prior corticosteroid exposure, and many have attempted over-the-counter creams that transiently soothe before worsening the eruption upon withdrawal. Eliciting a targeted exposure history is central to the physical assessment. Clinicians should ask about recent or ongoing use of topical corticosteroids on the face, nasal or inhaled steroids, fluorinated toothpaste, occlusive cosmetics, and mask wear patterns, as these may parallel disease onset or flares. On examination, the perioral zone often bears the greatest lesion density, but careful survey of the alar grooves, nasolabial folds, and infraorbital skin is essential, as satellite papules in these locations are common. Patients frequently describe burning, stinging, or hyperesthesia out of proportion to visible inflammation; pruritus can occur but is less prominent. Product intolerance—stinging with cleansers, moisturizers, or

sunscreens—is a frequent complaint and may perpetuate the cycle of irritation and impaired barrier recovery [27][28].

A granulomatous variant presents with flesh-colored to erythematous or yellow-brown papules in the same periorificial distribution and may, on rare occasions, extend beyond the face to the ears, neck, scalp, trunk, vulva, or extremities [25][26]. Because periocular skin is commonly involved, clinicians should screen for ocular irritation and crusting; when symptoms suggest lid margin disease or conjunctival involvement, ophthalmologic evaluation is prudent to assess for blepharitis or conjunctivitis and to prevent surface complications [26]. Systemic manifestations are not typical, and the review of systems is usually otherwise negative, helping to differentiate perioral dermatitis from systemic inflammatory or infectious dermatoses that present with fever or malaise. Bedside maneuvers aid clinical confidence. Diascopy of densely clustered, red-brown papules in lupoid perioral dermatitis may reveal a characteristic “lupoid” or apple-jelly-like infiltrate, supporting this more severe variant and prompting consideration of granulomatous histology if biopsy is pursued [29]. Differential diagnosis includes acne vulgaris (notably comedones absent), papulopustular rosacea (central facial erythema and flushing more prominent), seborrheic dermatitis (greasy scale in scalp, eyebrows), and allergic or irritant contact dermatitis (greater epidermal spongiosis, indistinct follicular pattern). Overall severity can be gauged by papule count, distribution breadth (perioral, perinasal, periocular), and symptom burden, particularly burning or tenderness, which often drives quality-of-life impairment. Integrating these historical and physical findings—while avoiding reflexive corticosteroid use on the face—anchors accurate recognition and guides steroid-sparing management.

#### Evaluation

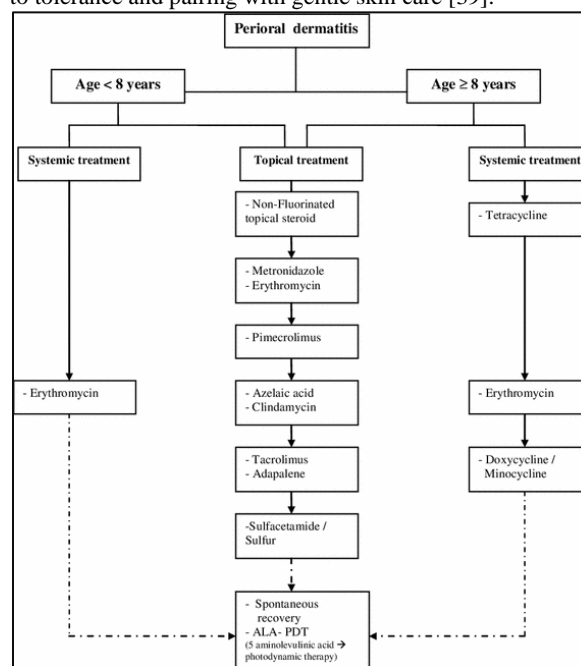
Most patients with suspected perioral dermatitis can be diagnosed on clinical grounds, integrating lesion morphology (perifollicular papules and papulopustules), periorificial distribution, and a focused exposure history that probes for topical, nasal, or inhaled corticosteroid use and other recognized triggers. A careful review of prior treatments is essential, as transient improvement with facial steroids followed by rebound flaring strongly supports the diagnosis. Physical examination should document the extent of perioral, perinasal, and periocular involvement and note sparing of the vermilion border, a helpful bedside clue. When the presentation is classic and the differential diagnosis narrow, laboratory testing is typically unnecessary. Ancillary studies are appropriate for atypical, refractory, or diagnostically ambiguous cases. A skin biopsy may be pursued when lesions are unusually indurated, nodular, or distributed beyond expected sites, or when they fail to improve with steroid withdrawal and first-line therapies; histopathology can help distinguish perioral dermatitis from acneiform eruptions, papulopustular rosacea, seborrheic dermatitis, and granulomatous diseases. If secondary bacterial infection is suspected—suggested by honey-colored crusts, increased purulence, or disproportionate tenderness—a bacterial culture from an intact pustule or erosive edge can guide antimicrobial selection and avoid unnecessary broad-spectrum treatment. When *Candida* is a consideration, especially in settings of recent antibiotic exposure, occlusion, or maceration, a bedside scraping with potassium hydroxide (KOH) preparation can identify yeast elements and prompt antifungal management. In more severe or granulomatous-

appearing disease, diascopy performed by an experienced clinician may reveal a lupoid infiltrate that raises suspicion for granulomatous variants and supports biopsy. Selective use of these evaluations—guided by pretest probability and response to initial therapy—optimizes diagnostic accuracy while minimizing invasive procedures.

#### Treatment / Management

##### Topical Therapies

Management of perioral dermatitis begins with a steroid-sparing, trigger-minimizing plan anchored by topical anti-inflammatory agents. First-line options include metronidazole (cream or gel), clindamycin (lotion or gel), erythromycin (gel), sulfur or sulfacetamide-sulfur preparations, and azelaic acid gel, each leveraging antimicrobial and—more importantly in this disease—anti-inflammatory effects rather than classic antibacterial killing [30][31][32][33]. Metronidazole is commonly used twice daily and is well tolerated on sensitive periorificial skin; patients typically note gradual reduction in erythema and papule count over several weeks, with continued gains through the third month as innate immune signaling normalizes [30][33]. Clindamycin and erythromycin, likewise, applied once or twice daily, can be valuable in patients who prefer light gel vehicles or who cannot tolerate other agents; their benefit appears to stem from neutrophil chemotaxis modulation and decreased inflammatory cytokine signaling [31][32][33]. Azelaic acid gel offers keratinization normalization and reactive oxygen species scavenging in addition to anti-inflammatory action, which can be helpful when subclinical dysseborrhea or background rosacea-like features coexist [32][33]. Topical sulfur or sulfacetamide-sulfur preparations provide adjunctive benefit by reducing surface microbial load and exerting mild keratolytic effects; their characteristic odor and potential for dryness necessitate careful counseling and use with bland, fragrance-free moisturizers to preserve barrier integrity [38][39]. Topical retinoids are not routine first-line therapy in perioral dermatitis because of irritancy risk; however, low-irritancy formulations such as adapalene may be introduced cautiously in selected patients with follicular plugging or relapse after initial control, titrating frequency to tolerance and pairing with gentle skin care [39].



**Figure-3:** Treatment of Perioral dermatitis.

Calcineurin inhibitors—tacrolimus ointment and pimecrolimus cream—are especially useful in steroid-dependent or steroid-exacerbated disease because they quell T-cell-mediated inflammation without provoking the rebound that follows corticosteroid withdrawal [34][35][36][37]. Application once or twice daily can reduce stinging and erythema within 1–2 weeks, while longer courses consolidate remission with far less risk of epidermal atrophy or telangiectasia than steroids on facial skin [34][36]. For highly refractory, cosmetically impactful cases, photodynamic therapy using 5-aminolevulinic acid as a photosensitizer has been reported to offer improvement, presumably through selective targeting of pilosebaceous units and local immunomodulation; this option should be reserved for experienced centers and carefully consented owing to post-procedure photosensitivity and variability in response [40]. Adjunctive non-pharmacologic measures materially influence outcomes. Patients should be counseled to simplify routines to a gentle, non-soap cleanser; apply non-occlusive, fragrance-free moisturizer; stop heavy or occlusive cosmetics; and, when relevant, modify triggers such as fluorinated toothpaste or mask occlusion. These steps reduce micro-occlusion and barrier disruption, thereby augmenting topical therapeutic efficacy and shortening time to remission [30][33][38].

**Oral Treatment Approaches**

When topical therapy is insufficient or disease is extensive, oral antibiotics with anti-inflammatory properties are appropriate bridge treatments. Tetracycline class agents reduce matrix metalloproteinase activity and inhibit neutrophil chemotaxis, effects that produce clinical improvement more rapidly than topical monotherapy. Typical regimens include tetracycline 250–500 mg twice daily, doxycycline 100 mg once or twice daily, or minocycline 100 mg once or twice daily, generally for an 8–12 week taper guided by clinical response [41][42]. The aim is to quell inflammation quickly while concurrent topical agents—metronidazole, azelaic acid, sulfur/sulfacetamide, or calcineurin inhibitors—build a durable maintenance effect. Because topical therapies may not reach peak efficacy until approximately 12 weeks, many clinicians plan to taper and discontinue oral antibiotics by the three-month mark, continuing topical maintenance thereafter [33][41]. Contraindications to tetracyclines—pregnancy, nursing, and children younger than eight years—necessitate alternatives such as erythromycin 250–500 mg daily, balancing efficacy with gastrointestinal tolerability and drug-interaction considerations [41][42]. A subset of patients experiences relapse upon rapid cessation of systemic therapy or cannot be maintained with topical regimens alone; in these scenarios, extended low-dose courses can be considered judiciously with periodic reassessment to minimize antimicrobial resistance and microbiome perturbation [41]. For recalcitrant, severe, or granulomatous variants unresponsive to the above measures, low-dose oral isotretinoin may be employed, typically initiating at approximately 0.2 mg/kg/day and tapering to 0.1–0.05 mg/kg/day as control is achieved [43][44]. This approach requires rigorous counseling and monitoring for teratogenicity, mucocutaneous side effects, lipid alterations, and mood changes, and is best coordinated in collaboration with dermatology [43][44]. Throughout systemic treatment, the therapeutic arc should be transparent: oral agents offer speed; topicals offer sustainability. Setting expectations about time-to-response, planned tapering, and maintenance

reduces anxiety and mitigates the temptation to resume facial corticosteroids for transient symptom relief [33][41].

**Corticosteroid Considerations**

Because perioral dermatitis is not a primary eczematous process, topical corticosteroids should be avoided on facial skin. Although they often produce short-term symptomatic improvement, withdrawal typically triggers rebound vasodilation, stinging, and a surge of papules that intensify disease chronicity and patient distress. Patients who have been applying medium- or high-potency steroids require a structured exit strategy: abrupt cessation can precipitate marked flaring, so a brief taper using a low-potency corticosteroid such as hydrocortisone may be employed before complete discontinuation, with immediate substitution of non-steroidal anti-inflammatories (e.g., calcineurin inhibitors or metronidazole) and strict trigger avoidance [45]. Clear anticipatory guidance that the eruption may transiently worsen over the first 1–2 weeks reassures patients and supports adherence to the steroid-sparing plan [33][45]. Steroid stewardship extends beyond topical prescriptions. Clinicians should screen for covert exposures—combination antifungal-steroid creams, over-the-counter hydrocortisone “spot” treatments, nasal or inhaled corticosteroids whose perioral deposition can perpetuate disease—and counsel on technique to minimize periorificial contact when such agents are medically necessary [34][35]. In granulomatous perioral dermatitis, where inflammatory momentum may be greater and conventional agents slower to act, emerging data suggest potential benefit from targeted immunomodulation: topical ruxolitinib 1.5% cream (a JAK inhibitor) has recently shown promising results, presumably by blocking downstream cytokine signaling central to granulomatous inflammation [46]. While longer-term safety and durability data are evolving, this therapy offers a steroid-free option for difficult cases and should be considered within specialist care pathways [46].

**Differential Diagnosis**

Distinguishing perioral dermatitis from its clinical mimics relies on a deliberate synthesis of lesion morphology, anatomic distribution, symptom profile, trigger history (especially facial corticosteroids and occlusive products), and targeted bedside or laboratory tests. The canonical pattern—clusters of perifollicular erythematous papules and occasional pustules arrayed around the mouth, alar grooves, and infraorbital regions with relative sparing of the vermilion border—provides a strong diagnostic anchor. Yet several disorders share overlapping features and may coexist, so a structured differential is essential to avoid corticosteroid use on the face, which can aggravate perioral dermatitis and obscure the picture. Rosacea sits closest on the spectrum of papulopustular facial dermatoses and is frequently central facial distribution, emphasizing the cheeks, nose, and glabella with persistent background erythema, telangiectasia, and often a history of heat- or alcohol-induced flushing. Ocular involvement in rosacea can include blepharitis and conjunctival hyperemia, whereas perioral dermatitis more typically shows periocular papules without the same degree of flushing. Importantly, both conditions commonly respond to metronidazole, azelaic acid, and tetracyclines, which has led many authors to conceptualize perioral dermatitis as a rosacea-adjacent entity; nonetheless, the absence of comedones distinguishes both conditions from acne vulgaris [33]. When the history reveals potent topical steroid exposure with rebound flares

on withdrawal and a periorificial ring sparing the vermilion, perioral dermatitis is favored over rosacea [33].

Acne vulgaris overlaps most through inflammatory papules and pustules but differs by the presence of open and closed comedones and frequent involvement of the chest and back. Adult female acne can localize to the chin and jawline, mimicking perioral distributions; however, the vermilion-sparing “halo,” lack of comedones, and sensitivity or burning with skincare products point back to perioral dermatitis. A history of friction from chin straps or helmet wear may produce acne mechanica, further muddying the waters; close inspection for comedones and follicular keratin plugs, along with response to comedolytic therapy, clarifies the diagnosis. Sarcoidosis can present with red-brown papules in periorificial zones, simulating both rosacea and perioral dermatitis. Clues include broader anatomic dispersion, coalescent plaques, or indurated papulonodules and systemic features such as fatigue, cough, dyspnea, or lymphadenopathy. Diascopy demonstrating “apple-jelly” translucency suggests a granulomatous process. When sarcoidosis is suspected, a punch biopsy for noncaseating granulomas and appropriate systemic evaluation (chest imaging, serum markers as clinically indicated) are warranted. Seborrheic dermatitis offers another common mimic but is typically characterized by ill-defined erythematous patches with greasy, yellow scale in sebum-rich sites—scalp, eyebrows, glabella, paranasal skin, nasolabial folds, beard area, and presternal chest. While perioral dermatitis can reach the nasolabial folds, the discrete perifollicular papules and vermilion sparing stand in contrast to the patchy, scaly plaques of seborrheic dermatitis. Response to antifungal shampoos, mild topical antifungals, and low-irritancy keratolytics (rather than tetracycline-class anti-inflammatory therapy) also helps discriminate the two [33].

Allergic contact dermatitis (ACD) is a key consideration, particularly when perioral dermatitis fails to improve or when there is a history of new skincare, sunscreens, or oral-hygiene products. ACD tends to manifest as ill-defined scaling macules, patches, or lichenified plaques with prominent pruritus and can extend beyond the periorificial zone to any site of allergen contact or transfer (e.g., fingertips to eyelids). Patch testing should be considered in refractory cases to identify allergens such as fragrance mixes, preservatives, cinnamic aldehyde in toothpaste, or acrylates from cosmetics and dental materials. Notably, topical corticosteroids often quell ACD but can worsen steroid-dependent perioral dermatitis, so a paradoxical rebound after steroid withdrawal supports the latter. Irritant cheilitis, including lip licker’s cheilitis, shows erythema and scaling that involve the cutaneous lip and, critically, affect the vermilion itself. This contrasts sharply with the vermilion border sparing of perioral dermatitis. History of habitual lip licking, exposure to wind or cold, or irritant toothpaste flavors points toward irritant cheilitis. Emollient repair and behavior modification typically effect rapid improvement, whereas perioral dermatitis requires anti-inflammatory therapy and trigger withdrawal. Demodex folliculitis presents with scattered, monomorphic erythematous papules and pustules that can closely mimic both rosacea and perioral dermatitis. Suspicion rises when lesions are uniform, pruritic or burning, and refractory to conventional therapies. Expressing or unroofing pustules and examining contents under light microscopy may reveal numerous Demodex mites, guiding therapy toward

acaricidal agents. The strong association between Demodex and rosacea complicates interpretation; however, in clear periorificial rings with vermilion sparing and steroid history, perioral dermatitis remains the leading diagnosis [33].

Tinea faciei can masquerade as annular or arciform erythematous plaques with peripheral scale and central clearing, sometimes with follicular papules at the advancing edge. KOH preparation of scale reveals hyphae and promptly redirects therapy to topical or systemic antifungals instead of antibiotics or calcineurin inhibitors. As with all facial dermatoses, empiric steroid application can exacerbate tinea (tinea incognita), potentially simulating steroid-altered perioral dermatitis; direct microscopy prevents this pitfall. Cutaneous adnexal neoplasms, such as syringomas or trichoepitheliomas, may mimic perioral dermatitis by presenting as multiple flesh-colored to faintly erythematous papules clustered on the periorbital or perioral skin. These lesions are typically asymptomatic, monomorphic, and stable over months to years, lacking the inflammatory tenderness, burning, or waxing-and-waning course characteristic of perioral dermatitis. Dermoscopy can suggest adnexal tumors, but a diagnostic biopsy is often necessary for confirmation and to exclude other papular neoplasms [33].

Two additional scenarios deserve emphasis. First, granulomatous perioral (periorificial) dermatitis can extend beyond the face to ears, neck, scalp, trunk, vulva, or extremities and may display yellow-brown papules; in these cases, diascopy and biopsy help distinguish from sarcoidosis, lupus miliaris disseminatus faciei, or cutaneous tuberculosis. Second, steroid-modified dermatoses—whether acneiform eruptions from fluorinated steroids or eczematous-appearing rashes temporarily suppressed by hydrocortisone—can cloud the clinical picture. A careful inventory of prescribed, over-the-counter, nasal, and inhaled corticosteroids, along with stepwise withdrawal and nonsteroidal anti-inflammatory substitutes, is both diagnostic and therapeutic. In practice, the differential narrows through pattern recognition and a few judicious tests. Comedones favor acne vulgaris; persistent central facial erythema with telangiectasia suggests rosacea; annular plaques with positive KOH indicate tinea; involvement of the vermilion border points to cheilitis; greasy scale in classic seborrheic sites supports seborrheic dermatitis; monomorphic papules with microscopic mites implicate Demodex; indurated red-brown papules with systemic signs raise sarcoidosis; and stable, asymptomatic papules suggest adnexal tumors. When uncertainty persists, especially with granulomatous papules or atypical spread, biopsy clarifies the diagnosis while preventing prolonged or harmful steroid exposure. This disciplined approach safeguards patients from iatrogenic worsening and ensures that perioral dermatitis is treated with the steroid-sparing strategies to which it most reliably responds [33].

#### Prognosis

Perioral dermatitis typically follows a waxing-and-waning course whose trajectory depends on early recognition, prompt removal of inciting factors, and adherence to steroid-sparing therapies. In a subset of patients, the eruption resolves completely once offending agents—most importantly facial topical corticosteroids, but also occlusive cosmetics, fragranced skincare, or irritating oral-hygiene products—are discontinued. This remission can be durable when coupled with a simplified, non-irritating skincare routine and targeted topical therapy. More commonly, however, perioral dermatitis behaves as a

chronic, relapsing condition. Flares are often precipitated by seasonal changes, lapses into corticosteroid use, overexfoliation, or renewed exposure to occlusive products or mask-related occlusion. Patients may experience prolonged low-grade activity characterized by intermittent papules and erythema that respond to treatment but recur when maintenance regimens are relaxed. The natural history is therefore best conveyed as controllable rather than definitively curable; realistic expectations about time to improvement—often measured in weeks to several months—help sustain adherence and reduce anxiety. With guideline-concordant care, most patients achieve meaningful reduction in lesion count, erythema, and symptoms such as burning or stinging, and many can transition to minimalist maintenance with nonsteroidal topicals. Prognosis is generally favorable even in moderate disease, although a minority require escalations to oral tetracyclines or, rarely, low-dose isotretinoin for recalcitrant courses. Granulomatous variants may follow a more persistent trajectory and can demand longer treatment horizons. Quality-of-life impact tends to improve rapidly once rebound flaring after steroid withdrawal is navigated. Long-term outcomes are optimized by interprofessional counseling about trigger avoidance, careful review of all steroid exposures (including nasal and inhaled formulations), and the use of a steady, gentle barrier-supportive routine that reduces the likelihood of relapse [33].

### Complications

Complications of perioral dermatitis arise from both the disease process and its treatment, with psychosocial ramifications often eclipsing cutaneous morbidity. The conspicuous facial distribution can provoke substantial emotional distress, including embarrassment, social withdrawal, and diminished self-confidence. These effects frequently intersect with professional and interpersonal domains, amplifying perceived disease burden and reducing adherence, especially when early steroid withdrawal triggers transient worsening. Persistent erythema, clustered papules, and episodes of burning or stinging contribute to poor quality of life; sleep disruption and avoidance of physical activity are not uncommon when mask wear or perspiration aggravates symptoms. Iatrogenic complications stem primarily from inappropriate or prolonged facial corticosteroid use, which can engender rebound flares, telangiectasia, epidermal atrophy, and steroid dependence. Secondary bacterial colonization may complicate excoriated lesions, necessitating culture-guided management. In the rarer granulomatous or lupoid subtype, chronic inflammation can increase the risk of textural change; scarring has been reported in association with the lupoid variant, underscoring the importance of timely recognition and escalation to steroid-sparing therapies or systemic agents when indicated [29]. Ocular surface irritation may occur when periorcular skin is involved; unaddressed, this can progress to blepharitis or conjunctival symptoms that further degrade comfort and daily functioning. Finally, therapeutic misadventures—such as repeated short bursts of topical or systemic corticosteroids for transient relief—entrench chronicity, delay effective care, and perpetuate the cycle of rebound and relapse. Preventing these complications requires anticipatory guidance, trigger modification, and coordinated care that privileges consistent, nonsteroidal anti-inflammatory regimens [29].

### Consultations

Consultation strategies should be individualized to clinical severity, anatomic extent, and treatment response.

Primary care clinicians and dermatologists typically lead diagnosis and initial management, but collaboration enhances safety and outcomes. Because periorcular involvement is common, clinicians must screen for ocular surface symptoms—burning, itching, tearing, foreign-body sensation, crusting at the lid margins—and examine for signs of blepharitis or conjunctival hyperemia. When these features are present or when symptoms persist despite cutaneous improvement, an ophthalmology consultation is warranted to evaluate for ocular comorbidity, guide lid hygiene, and prevent surface complications. Dental teams may provide valuable input when oral-hygiene products, dental materials, or intraoral appliances plausibly contribute to irritation; counseling on toothpaste formulations and procedural aftercare can mitigate periorificial exposure. Pharmacist collaboration supports steroid stewardship, identifies hidden corticosteroid sources (e.g., combination antifungal-steroid creams, nasal or inhaled steroids), and tailors regimens to minimize adverse effects. In patients using continuous positive airway pressure (CPAP) devices, respiratory or sleep-medicine specialists can help optimize mask fit and interface materials to reduce mechanical occlusion and periorificial friction. Patch testing through contact-dermatitis specialists should be considered when the eruption is refractory or the history strongly suggests allergic contact dermatitis to skincare, sunscreens, or oral-care agents, thereby refining avoidance strategies. For recalcitrant or granulomatous variants that fail conventional therapy, referral to dermatology for consideration of systemic agents, procedural interventions, or emerging topical immunomodulators is prudent. Ultimately, timely and targeted consultations shorten diagnostic delays, forestall iatrogenic harm, and support comprehensive, patient-centered care [29].

### Patient Education

Deterrence and patient education are foundational to durable control of perioral dermatitis. At the first visit, clinicians should deliver clear, consistent messaging that topical corticosteroids—whether prescription or over-the-counter—are contraindicated on the face for this condition. Patients benefit from a simple explanation: steroids can temporarily suppress redness but drive rebound flares when stopped, perpetuating a cycle of dependence and chronicity. This rationale reduces the temptation to reach for “quick fixes” during early treatment when improvement may be gradual. Education should also catalogue common triggers and exposures, including fluorinated toothpaste, fragranced or occlusive cosmetics, certain sunscreens, aggressive cleansing and exfoliation, and, when relevant, prolonged mask wear or poorly fitted CPAP interfaces. A “less is more” skincare plan—gentle non-soap cleanser, non-occlusive fragrance-free moisturizer, and prescribed nonsteroidal anti-inflammatories—should be outlined in writing, with explicit instructions on what to stop and what to start. Setting expectations is equally critical. Patients should be told to anticipate a potential rebound flare in the first one to two weeks after steroid discontinuation and to expect steady improvement over weeks to months rather than days. Reinforcing adherence strategies—fixed daily routines, reminders, and follow-up check-ins—helps sustain momentum through this period. Photoprotection, avoidance of harsh weather exposure, and mindful product re-introduction (one new product at a time after remission) further reduce relapse risk. Patients should understand the role of oral antibiotics as time-limited “bridges” for more rapid relief, with plans to taper once topicals achieve

maintenance control. Clear guidance on when to seek help—new or worsening ocular symptoms, spreading nodules suggestive of granulomatous change, or intolerance to medications—empowers early course correction. Ultimately, consistent education delivered by the whole care team improves adherence, shortens the active disease phase, and minimizes recurrences by aligning daily behaviors with the biology of the disorder [33][34][35].

#### Enhancing Healthcare Team Outcomes

Optimizing outcomes in perioral dermatitis depends on a coordinated, interprofessional framework that aligns diagnostic vigilance, trigger mitigation, and steroid-sparing therapy. Physicians and advanced practitioners should lead with pattern recognition and a disciplined exposure history, documenting topical, nasal, and inhaled corticosteroid use; skincare routines; cosmetic layering; and environmental occlusion. Standardized care pathways—steroid cessation with or without a short taper for medium- to high-potency users, initiation of nonsteroidal topicals, and criteria for adding oral tetracyclines—promote consistent management and reduce variation. Nurses and medical assistants operationalize these pathways by delivering structured education, coaching on gentle skincare, and reinforcing expectations about rebound and gradual improvement. Pharmacists extend safety by reviewing for drug–drug interactions, counseling on correct application and adverse effects, and identifying covert steroid exposures that sabotage progress; they also troubleshoot intolerance to tetracyclines, including photosensitivity and gastrointestinal upset, and recommend mitigation strategies. Communication loops are the backbone of high-functioning teams. Shared documentation that tracks triggers, therapies, and response over time prevents mixed messages, while scheduled follow-ups allow titration of treatment intensity and evaluation for contact allergens in nonresponders. Rapid escalation pathways—direct messaging from nurses or pharmacists to prescribers when patients report ocular symptoms, severe rebound, or spreading granulomatous papules—shorten time to intervention and reduce complications. Case reviews can spotlight patterns, such as frequent relapses tied to specific product categories, informing clinic-wide education materials. Collaboration with ophthalmology for periocular disease, dentistry for oral-hygiene contributors, and sleep medicine for CPAP-related occlusion integrates specialty expertise into a unified plan. Finally, cultivating a therapeutic alliance—acknowledging the cosmetic and psychosocial weight of facial eruptions, validating setbacks during steroid withdrawal, and celebrating incremental wins—improves adherence and satisfaction. In aggregate, a coordinated, patient-centered approach that leverages the complementary strengths of physicians, advanced practitioners, nurses, pharmacists, and consultants translates evidence-based recommendations into sustained, real-world remission.

#### Conclusion:

In summary, perioral dermatitis is a complex dermatosis whose successful management hinges on a fundamental paradigm shift: the cessation of topical corticosteroids and the adoption of steroid-sparing strategies. The condition, while benign, carries a significant psychosocial burden due to its conspicuous facial location. A thorough patient history is paramount to identify and eliminate inciting factors, ranging from prescription steroid creams to everyday products like fluorinated toothpaste and occlusive cosmetics. First-line treatment with topical agents like metronidazole or calcineurin inhibitors, supplemented

by oral tetracyclines in more severe cases, effectively controls inflammation. Critically, patients must be counselled to expect a potential rebound flare after steroid withdrawal, requiring reassurance and adherence to the new treatment plan. Long-term management focuses on maintaining remission through gentle skincare, continued trigger avoidance, and, if necessary, low-dose maintenance therapy. Ultimately, a collaborative, interprofessional approach involving physicians, nurses, pharmacists, and dental professionals is essential to provide consistent education, support, and stewardship, ensuring patients avoid the iatrogenic harm of corticosteroids and achieve durable control of their condition.

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