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Perioral Dermatitis: Modern Aspects of Etiopathogenesis, Clinical Presentation, Diagnosis and Therapy

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Abstract **Background:** Perioral dermatitis (PD) is a chronic inflammatory facial dermatosis characterized by erythematous papules, papulovesicles, and papulopustules localized mainly in the perioral, perinasal, and periocular regions. It predominantly affects women aged 25–40 years. The etiopathogenesis of PD remains incompletely understood and is considered multifactorial, with epidermal barrier dysfunction as a key mechanism. **Methods:** A review of current literature was conducted to summarize epidemiological data, etiological factors, pathogenetic mechanisms, clinical features, diagnostic approaches, and evidence-based treatment options for PD in adults and children. **Results:** PD is associated with multiple contributing factors, including topical corticosteroid use, occlusive cosmetic products, alterations in the cutaneous microbiome, and hormonal influences. Clinical presentation is characterized by typical inflammatory lesions in the affected facial areas. Diagnosis is primarily clinical. Management involves elimination of triggering factors and the use of topical and/or systemic therapies depending on disease severity. **Conclusion:** PD is a multifactorial inflammatory dermatosis in which epidermal barrier dysfunction plays a central role. Early identification of triggers and appropriate therapeutic strategies are essential for effective management and prevention of recurrence.

Keywords Perioral dermatitis, Periorificial dermatitis, Epidermal barrier, Topical corticosteroids, Tetracyclines, metronidazole, Calcineurin inhibitor

1. Introduction

Perioral dermatitis (PD) is a chronic inflammatory dermatosis of the face, clinically manifested by monomorphic erythematous papules, papulovesicles, and papulopustules, typically distributed in the perioral, perinasal, and periocular regions. The disease predominantly affects women of reproductive age, although cases are also reported in men and pediatric populations. Despite its relatively well-defined clinical presentation, the etiopathogenesis of PD remains insufficiently elucidated.

Contemporary data support a multifactorial pathogenesis with a key role of epidermal barrier dysfunction. A range of exogenous and endogenous factors has been implicated in disease development, including the use of topical corticosteroids, occlusive cosmetic products, alterations in the cutaneous microbiome, and hormonal influences. These factors are believed to act as triggers that initiate and perpetuate inflammatory processes in genetically or environmentally predisposed individuals. From a clinical perspective, PD is characterized by a chronic and relapsing course, which may lead to significant cosmetic concerns and reduced quality of life. Diagnosis is primarily clinical and relies on the recognition of characteristic lesion morphology and distribution.

Given the increasing prevalence and persistence of the condition, a comprehensive understanding of its epidemiology, pathophysiological mechanisms, and therapeutic approaches is essential for optimizing patient management.

This review **aims** to provide a critical synthesis of current evidence on perioral dermatitis, with particular focus on its epidemiology, etiopathogenesis, clinical presentation, diagnostic criteria, and evidence-based therapeutic strategies in adult and pediatric populations.

2. Methods

This study was conducted as a narrative review of the current scientific literature on perioral dermatitis. Relevant publications were identified through a structured search of major medical databases using predefined keywords and their combinations, including “perioral dermatitis,” “epidemiology,” “etiopathogenesis,” “clinical features,” “diagnosis,” and “treatment.” Both adult and pediatric populations were included.

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The selection criteria encompassed peer-reviewed original research articles, clinical guidelines, and review papers. No strict temporal restrictions were applied; however, priority was given to recent publications and sources with a high level of evidence to ensure the relevance and reliability of the findings. Studies were selected based on their scientific quality, relevance to the topic, and contribution to the understanding of epidemiology, pathophysiological mechanisms, clinical presentation, diagnostic approaches, and therapeutic management of perioral dermatitis.

The extracted data were critically evaluated and synthesized using a descriptive analytical approach to provide a comprehensive overview of the current state of knowledge on perioral dermatitis.

3. Results

PD is a chronic inflammatory disease of the facial skin, first recognized as a separate nosological entity in the early 1960s. The disease is characterized by the appearance of small (1–2 mm) hemispherical erythematous papules and papulopustules on the background of diffuse or localized erythema with the formation of a pathognomonic sign — a narrow strip of intact skin (2–5 mm) along the vermilion border of the lips. In modern dermatological literature, several synonymous terms are used: “perioral dermatitis”, “periorificial dermatitis”, “steroid-induced facial dermatitis”, “rosacea-like dermatitis”, “dermatitis perioralis”. The term “periorificial dermatitis” is considered the most correct from a clinical and anatomical point of view, as it reflects the possibility of lesion localization not only in the perioral region but also in the perinasal and periorbital areas. In the International Classification of Diseases, 10th revision, PD is coded under L71.0 and belongs to the rosacea group. The relevance of the problem is due to the steadily increasing incidence, chronic relapsing course, and pronounced negative impact on the psycho-emotional state and quality of life of patients.

Epidemiology. PD predominantly affects women of young and middle age (25–40 years); the ratio of women to men is approximately 20:1. According to Vashkevich et al. (2023), about 6% of women and 0.3% of men seeking dermatological care receive a diagnosis of PD [1]. The disease has been described in all age groups — from infants (starting from 6 months of age) to elderly individuals. In the pediatric population, PD accounts for a significant proportion of visits. According to a large retrospective cohort study by Neff et al. (2025, n=451), the age of patients ranged from 4 months to 72 years (mean age 32 years), 79% were women [2]. Goel et al. (2015, n=222) demonstrated that pediatric PD often begins at an early age, while the granulomatous form occurs predominantly in prepubertal boys [3].

Etiology. PD is multifactorial in nature, and to date no single etiological agent has been identified.

Topical corticosteroids the most convincingly documented etiological factor is prolonged use of topical glucocorticosteroids on the facial skin. The strong association with the use of topical corticosteroids led to the appearance of the synonym “steroid facial dermatitis”. The mechanism of PD formation during long-term application of topical corticosteroids includes suppression of lipid synthesis in the stratum corneum, epidermal atrophy, inhibition of antimicrobial peptide production, and disturbance of the local immune response with subsequent activation of opportunistic microflora. Of particular clinical importance is the phenomenon of “steroid dependence”: after withdrawal of topical corticosteroids, a predictable exacerbation develops — the so-called rebound effect, which forces the patient to resume applications, forming a vicious circle. Wells and Brodell (1993) emphasized the need to inform patients about the transient nature of exacerbation after discontinuation of topical corticosteroids [4]. At the same time, the concept of total dominance of the steroid factor is being revised. A large retrospective cohort study by Neff et al. (2025, n=451) showed that only 37% of patients with PD reported the use of corticosteroids within the 6 months preceding diagnosis, while topical corticosteroids were used by only 23%, which is significantly lower than historical data (72–96%) [2].

Occlusive cosmetic products based on petrolatum and paraffin, heavy moisturizing creams, fluoride-containing toothpastes, sunscreens, and some dermocosmetic products are considered potential provoking agents. According to Neff et al. (2025), 25% of patients reported predisposing factors other than corticosteroids, including sunscreens, whitening toothpastes, and heavy moisturizers [2].

Microbiological factor. The role of the microbial component has been studied for several decades. In 1977, Kalkoff and Buck formulated the concept of “fusobacteriosis”, according to which fusobacteria (in particular Fusobacterium nucleatum) play a key role in disease initiation, while corticosteroids create conditions for transformation of opportunistic flora into pathogenic flora [5]. Adaskevich et al. (2019) confirmed a high detection rate of fusiform bacteria: Fusobacterium nucleatum was found in 50% of cases [6]. The role of mites of the genus Demodex also deserves attention. Elistratova (2013) showed that in PD combined with demodicosis (51 of 98 examined patients), the skin microflora is characterized by greater species diversity; the detection rate of S. aureus and S. haemolyticus increases in severe forms [7], whereas in mild forms S. epidermidis and S. hominis predominate. Modern molecular-genetic studies of the microbiome (Ferček et al., 2024, n=74) revealed statistically significant changes in microbiota alpha diversity in patients with periocular dermatitis compared with the control group ($p<0.05$ according to Faith’s PD index) [8]. Mochizuki et al. (2025, n=50) found that the microbiota composition in PD differs significantly from that in rosacea and in healthy individuals, and an uncultivated bacterium of the order Neisseriales predominated in steroid-induced rosacea and PD and disappeared after antibiotic therapy [9].

Hormonal factors. Among additional predisposing factors, the use of oral contraceptives, gastrointestinal disorders, neurogenic inflammation, and psycho-emotional stress are

considered. The pronounced gender predominance indirectly indicates the importance of the hormonal factor.

Pathogenesis. The modern pathogenetic concept of perioral dermatitis is based on the understanding that impairment of the epidermal barrier is the key link in the pathological process. Balić et al. (2019) substantiated the concept that patients with PD are "hyper-reactors" — individuals with an initially impaired skin barrier function, especially in the perioral region, which anatomically is characterized by increased transepidermal water loss (TEWL) and reduced ceramide content [10]. Dysfunction of the skin barrier creates conditions for increased penetration of exogenous irritants and microorganisms, which triggers a cascade of inflammatory reactions. Mokos et al. (2015) emphasized that all described trigger factors — corticosteroids, occlusive cosmetic products, physical and hormonal influences — are united by a common pathogenetic mechanism, namely disruption of the epidermal barrier [11]. Topical corticosteroids aggravate barrier dysfunction through several interrelated mechanisms: suppression of lipid synthesis in the stratum corneum (ceramides, free fatty acids, cholesterol); epidermal atrophy due to inhibition of keratinocyte proliferation; suppression of antimicrobial peptide production (cathelicidins, defensins); disturbance of the local immune response with imbalance of Th1/Th2 cells; activation of opportunistic microflora and Demodex mites. Thus, PD is considered the result of convergence of multiple factors acting through a single pathophysiological pathway — disruption of the epidermal barrier followed by the development of chronic inflammation. The classical form of PD manifests as small hemispherical papules and papulopustules on the background of erythema and mild scaling. The lesions are usually symmetrically distributed, although unilateral presentation is possible. A pathognomonic feature is the presence of a narrow strip of unaffected skin along the vermilion border of the lips — the so-called "clear zone", which has important differential diagnostic value. Subjective symptoms include burning sensation, tightness, and dryness of the skin; pruritus is usually mild or absent. According to the topographic distribution of lesions, several variants are distinguished: perioral — the most common (up to 70% of cases in adults); perinasal — isolated or combined with perioral involvement; periocular — characterized by a torpid course; mixed — simultaneous involvement of several anatomical areas. According to Nguyen and Eichenfield (2006, n=79), in children and adolescents' perioral involvement occurred in 70% of cases, perinasal in 43%, and periocular in 25%, while 72% of patients had a history of corticosteroid exposure, and the mean duration of lesions before medical consultation was 8 months [12]. Larralde et al. (2011, n=48) found that inhaled corticosteroids were associated mainly with perioral and perinasal localization [13]. The course of PD is chronic and relapsing. Without adequate therapy, the disease tends to persist, especially with continued use of cosmetic products. Chronicity of the process, together with localization on exposed areas of the skin, leads to a significant decrease in patients' quality of life and the development of psycho-emotional disorders.

Diagnosis. Diagnosis of PD is based mainly on clinical data and history. The characteristic morphology of lesions, typical localization, and the presence of a clear zone around the vermilion border in most cases allow diagnosis without additional studies. Histopathological findings are nonspecific and include perivascular and perifollicular lymphohistiocytic infiltrate, sometimes with neutrophils, spongiosis, and dilation of follicular ostia. In the granulomatous variant, epithelioid cell granulomas without caseous necrosis are found. To objectify the severity of the process, the PODSI index (Perioral Dermatitis Severity Index) is used, including assessment of erythema, number of papules/pustules, and degree of scaling. Differential diagnosis is performed with rosacea (presence of telangiectasia, absence of clear zone), seborrheic dermatitis (greasy scales, scalp involvement), contact dermatitis (more pronounced itching, relation to allergen), acne (presence of comedones), demodicosis (verified by skin scraping microscopy). Careful history taking regarding the use of topical corticosteroids, inhaled corticosteroids, and cosmetic products is essential.

Treatment. Therapy of PD is based on the principles of staging and individualization taking into account severity and age. The first and mandatory step is discontinuation of all topical corticosteroids and potentially provoking agents. Most cases are self-limiting if exacerbating factors are eliminated. Patients should be warned about inevitable transient worsening (rebound phenomenon) during the first 1–2 weeks after withdrawal of corticosteroids. In steroid-induced PD with long history, gradual withdrawal may be required — from halogenated to non-halogenated forms followed by complete cessation.

Local therapy. Metronidazole (0.75–2% gel or cream) is one of the most widely used topical agents. In a randomized multicenter double-blind study (Veien et al., 1991, n=108) [12], topical 1% metronidazole reduced the median number of papules to 8% of baseline after 8 weeks, while oral tetracycline reduced it to 0%; both were effective, but tetracycline was superior. Boeck et al. (1997) showed complete resolution after 3–6 months of topical metronidazole in children with remission maintained for 2 years [15].

Topical erythromycin (2%) shows efficacy comparable to oral tetracycline. In a placebo-controlled study (Weber et al., 1993, n=99), topical erythromycin and oral tetracycline were both significantly more effective than placebo ($p<0.001$) and did not differ from each other [16]. Calcineurin inhibitors (pimecrolimus, tacrolimus) are steroid-sparing alternatives. Pimecrolimus does not accelerate complete resolution but reduces severity rapidly, especially after steroid use. Lee and Kim (2020, n=24) confirmed high efficacy of calcineurin inhibitors combined with metronidazole in children [17]. Activated zinc pyrithione is a promising agent; Trapeznikova et al. (2024) demonstrated its effectiveness and safety both as monotherapy and in combination therapy [18].

Azelaic acid (15–20%) is used as an adjuvant due to anti-inflammatory and antimicrobial effects. Systemic therapy: indicated in moderate to severe cases.

Tetracyclines (doxycycline, tetracycline, minocycline) are first-line systemic drugs. Tetracycline 250 mg 4 times daily for 3–4 months is recommended.Del Rosso (2011) justified the use of modified-release doxycycline 40 mg/day for anti-inflammatory effect without resistance [19].

Macrolides (erythromycin, azithromycin, clarithromycin) are alternatives, especially in children and pregnancy.

Skin barrier. Restoration of the barrier is essential. Studies show that dermocosmetics improve PODSI dynamics. Gentle cleansing, ceramide-containing emollients, and avoidance of occlusion are basic care.

Pediatric PD. According to Larralde et al. (2011), steroids preceded PD in 66.6%; inhaled steroids linked to perioral/ perinasal lesions [13]. Tetracyclines contraindicated under 8–12 years; topical therapy and macrolides preferred. Prognosis in children is good; Boeck et al. (1997) showed full remission in 3–6 months with long-term stability. Prognosis: generally favorable but relapsing. Prevention includes avoiding steroids, heavy cosmetics, fluoride toothpaste if related, barrier repair, and patient education [15].

4. Discussion

PD is a multifactorial inflammatory dermatosis with a complex and not fully elucidated pathogenesis. The findings of this review confirm that epidermal barrier dysfunction plays a central role in disease development, acting as a key mechanism through which various exogenous and endogenous factors exert their effects. Topical corticosteroid use remains one of the most consistently reported triggering factors, often associated with both the onset and exacerbation of PD, as well as with disease persistence due to rebound phenomena following withdrawal.

Additional contributing factors include occlusive cosmetic products, alterations in the cutaneous microbiome, and hormonal influences, which together may disrupt skin homeostasis and promote chronic low-grade inflammation. The interplay between these factors suggests that PD should be considered a condition arising from a combination of environmental exposures and individual susceptibility rather than a single causative agent.

From a clinical perspective, PD is characterized by a relatively uniform morphology and distribution of lesions, which facilitates diagnosis in most cases. However, differential diagnosis may be challenging due to overlap with other dermatological conditions such as acne vulgaris, rosacea, and seborrheic dermatitis. Therefore, accurate clinical assessment remains essential to avoid misdiagnosis and inappropriate treatment.

Management of PD is primarily based on the identification and elimination of triggering factors, particularly topical corticosteroids and occlusive cosmetics, followed by the use of topical and systemic therapies depending on disease severity. Evidence suggests that a stepwise and individualized therapeutic approach contributes to better clinical outcomes and reduces the risk of recurrence. Overall, the results of this review highlight the importance of a comprehensive understanding of PD pathophysiology and the need for early recognition and targeted management strategies to improve patient outcomes.

5. Conclusions

PD is a polyetiological inflammatory disease of the facial skin, the pathogenesis of which is based on impairment of the epidermal barrier. Topical glucocorticosteroids remain the most significant, but not the only trigger factor; modern data indicate the increasing role of cosmetic products, the skin microbiome, and disorders of local immunity. The therapeutic approach should be comprehensive, stepwise, and individualized — from “zero therapy” with elimination of provoking factors, through topical treatment, to systemic antibacterial drugs in case of insufficient effectiveness of local therapy. Restoration of the skin barrier is an essential component of the treatment strategy at all stages. Promising directions for future research include in-depth study of the perioral microbiome using next-generation sequencing methods; investigation of genetic markers of barrier dysfunction; development of targeted topical agents aimed at restoring specific components of the epidermal barrier; and conducting large randomized controlled trials to compare the effectiveness of existing therapeutic approaches.

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