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From Comedones to Scars: Understanding Acne Pathogenesis, Clinical Types, and Contemporary Management

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Abstract **Review Article**

Acne vulgaris is a multifactorial, chronic inflammatory disorder of the pilosebaceous unit that predominantly affects adolescents but can persist or emerge in adulthood. Its pathogenesis involves an intricate interplay among sebaceous hyperactivity, follicular hyperkeratinization, colonization by *Cutibacterium acnes*, and inflammatory responses. Clinically, acne presents a broad spectrum, from non-inflammatory comedones to severe nodulocystic lesions. Management strategies are guided by severity, type, and patient-specific factors, utilizing topical, systemic, and hormonal therapies. Complications such as post-inflammatory hyperpigmentation and scarring are common and may require specialized interventions. This review discusses acne's pathophysiology, clinical variants, current therapeutic strategies, and complications, integrating contemporary evidence-based recommendations.

Keywords: Acne vulgaris, Pathogenesis, Pilosebaceous unit, Cutibacterium acnes, Inflammation, Therapeutic strategies.

INTRODUCTION

Acne vulgaris is among the most widespread dermatological conditions, with an estimated global prevalence of approximately 9.4%, making it the eighth most prevalent disease worldwide (Tan & Bhate, 2015). While traditionally considered a condition of adolescence, adult-onset acne—particularly in women—is increasingly recognized. The disease significantly impacts patients' quality of life, often contributing to psychosocial distress, anxiety, and depression (Cunliffe, 2002).

Understanding acne's multifactorial pathogenesis has led to more nuanced therapeutic strategies. Contemporary treatment aims not only at lesion clearance but also at minimizing complications such as scarring and pigmentation, and addressing the psychological burden. This review explores acne's pathogenic mechanisms, clinical subtypes, therapeutic approaches tailored to disease severity, and the management of sequelae.

Pathophysiology and Background

Acne develops within the pilosebaceous unit, and four core mechanisms contribute to its pathogenesis (Dreno *et al.,* 2018):

1. **Sebum overproduction** driven by androgenic stimulation.
2. **Follicular hyperkeratinization**, which leads to comedone formation and obstruction of the follicular canal.
3. **Proliferation of *Cutibacterium acnes***, an anaerobic bacterium that contributes to inflammation through biofilm formation and induction of cytokines.
4. **Inflammatory response**, both innate and adaptive, leading to clinical lesion progression.

Emerging evidence points to additional contributing factors such as genetic predisposition, dietary habits (notably high glycemic index and dairy consumption), and hormonal imbalances. The skin microbiome's disruption and immune dysregulation also play significant roles in the perpetuation of acne lesions (Kirk *et al.,* 2021).

Clinical Variants of Acne

Acne manifests across a wide clinical spectrum, categorized by lesion type and severity:

- **Comedonal acne**: Characterized by open (blackheads) and closed (whiteheads) comedones; typically non-inflammatory and early in onset.
- **Papulopustular acne**: Inflammatory lesions including red papules and pustules; common in moderate disease.
- **Nodulocystic acne**: Severe variant with deep, painful nodules and cysts; often leads to permanent scarring.
- **Acne conglobata**: Aggressive and chronic form with interconnected abscesses and sinus tracts.
- **Acne fulminans**: Rare, systemic variant with ulcerative nodules, fever, and arthralgias; requires systemic immunosuppression.
- **Adult female acne**: Often presents with inflammatory lesions predominantly on the lower face and jawline, commonly associated with hormonal fluctuations.

Current Treatment Approaches

Treatment selection should consider acne severity, lesion morphology, patient age, gender, and psychosocial impact. The therapeutic approach is tiered:

1. Comedonal Acne

- **Topical retinoids** (adapalene, tretinoin): Normalize follicular desquamation and comedone formation.
- **Benzoyl peroxide (BPO)**: Antimicrobial and mildly comedolytic.

2. Papulopustular Acne

- **Mild to moderate**: Combination of topical retinoids, BPO, and topical antibiotics (e.g., clindamycin).
- **Moderate to severe**: Oral antibiotics (doxycycline, minocycline) used for 8–12 weeks. Avoid monotherapy to prevent resistance.
- **Adjunctive**: Hormonal therapies (oral contraceptives or spironolactone) for women with androgenic features.

3. Nodulocystic Acne / Severe Acne

- **Oral isotretinoin**: Targets all major pathogenic mechanisms. It is the most effective option for severe or refractory acne.
- Requires baseline and monthly monitoring for lipids, liver function, and pregnancy (teratogenic).

4. Adult Female Acne

- **Hormonal modulation**: Combined oral contraceptives, anti-androgens like spironolactone.
- Consider screening for underlying endocrine disorders (e.g., PCOS).

5. Maintenance therapy

- Long-term use of topical retinoids is recommended to maintain remission and prevent relapse.

Complications and Their Management

1. Post-inflammatory Hyperpigmentation (PIH)

More prevalent in Fitzpatrick skin types III–VI.

Management:

- Topical depigmenting agents (hydroquinone, azelaic acid, retinoids)
- Chemical peels (e.g., glycolic, salicylic acid)
- Laser therapy (e.g., QS Nd:YAG) with caution in darker skin tones

2. Scarring

Affects approximately 20% of patients with inflammatory acne (Fabbrocini *et al.,* 2010). Types include:

- **Atrophic**: icepick, rolling, and boxcar scars.
- **Hypertrophic/keloidal**: More common in darker skin tones and upper trunk.

Therapeutic options:

- Microneedling
- Subcision
- Chemical reconstruction using TCA (CROSS technique)
- Fractional lasers (ablative/non-ablative)
- Fillers (e.g., hyaluronic acid)
- Surgical revision for severe cases

3. Psychological Impact

The burden of acne on mental health is well documented. Patients may experience depression, anxiety, and social withdrawal. Early recognition and referral to mental health services are essential in comprehensive care (Layton, 2021).

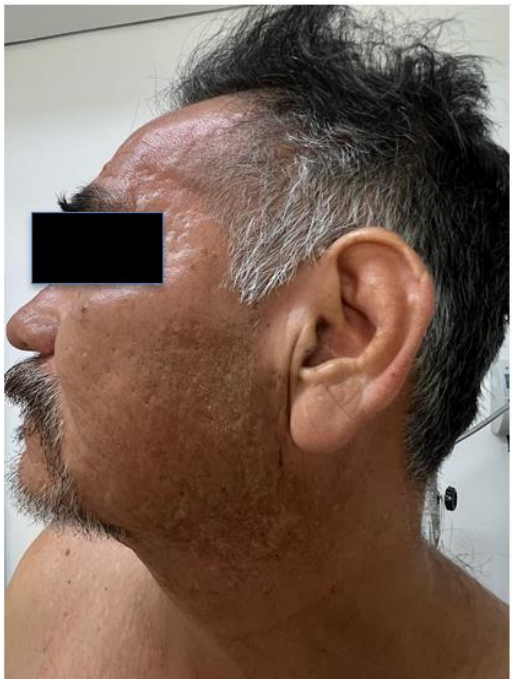

Figure 1

The patient presents with post-inflammatory hyperpigmentation and textural skin changes predominantly affecting the lower face, including the cheeks, jawline, and periauricular area. The skin surface demonstrates evidence of atrophic scarring and enlarged pores, likely secondary to previous inflammatory acne lesions. There is also perifollicular hyperpigmentation and some residual erythematous macules, consistent with resolving acne. No active nodules or pustules are observed at the time of the image.

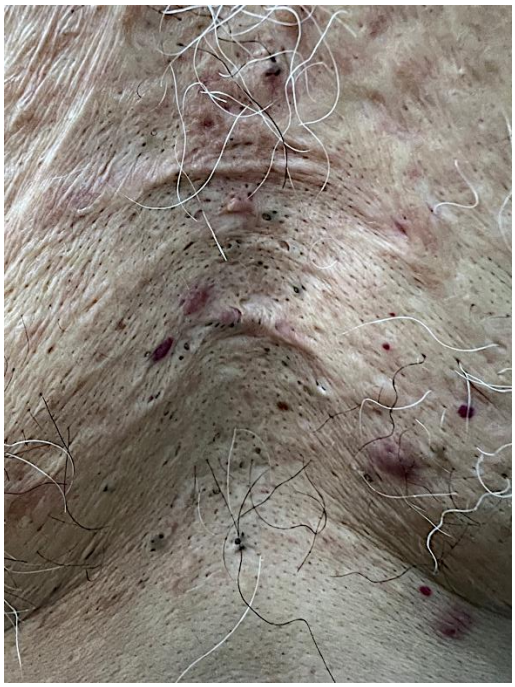

Figure 2

The image reveals several comedones (both open and closed) are visible, along with scattered erythematous papules and nodules. Some nodules exhibit a violaceous hue suggestive of inflammatory activity. There are areas of hyperpigmentation and post-inflammatory changes, as well as a few pustular elements. The presence of terminal and vellus hairs is noted, and the overall texture of the skin appears thickened and irregular, consistent with chronic inflammation.

CONCLUSION

Acne vulgaris is a complex inflammatory disorder with significant medical, cosmetic, and psychological implications. Advances in understanding its pathogenesis have improved treatment strategies, allowing for more personalized and effective care. Clinicians must tailor therapies according to acne type and severity, emphasize early intervention to prevent complications, and integrate psychosocial support when necessary. Continued research and multidisciplinary collaboration will further optimize patient outcomes.

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