



ETIOLOGY, CLINICAL STAGES, AND TREATMENT MODALITIES OF ACNE VULGARIS

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Abstract. Although it is often interpreted as a temporary adolescent skin problem, modern dermatology considers acne a multifactorial disease involving follicular hyperkeratinization, increased sebum production, Cutibacterium acnes-associated microbial activity, immune inflammation, hormonal influence, genetic predisposition, environmental exposure, and behavioral skin-care factors. The study uses a descriptive analytical design based on clinical guidelines, dermatological textbooks, national protocols, and scientific literature. The results show that successful acne management depends on correct severity classification, early recognition of scarring risk, rational use of topical and systemic therapies, avoidance of antibiotic monotherapy, patient education, and long-term maintenance care. The article is intended for educational and scientific purposes and does not replace individual medical consultation.

Keywords: acne vulgaris; etiology; pathogenesis; comedone; inflammation; dermatology; treatment; prevention; post-acne scarring.

Introduction

Acne vulgaris is a chronic inflammatory dermatosis centered on the pilosebaceous unit, which consists of a hair follicle and its associated sebaceous gland. The disease most frequently affects the face, forehead, chin, chest, shoulders, and upper back, where sebaceous glands are most active. Clinically, acne may appear as open and closed comedones, inflammatory papules, pustules, nodules, cyst-like lesions, post-inflammatory hyperpigmentation, and scarring. The visible nature of the disease gives it a social and psychological dimension that is often underestimated in routine communication with patients.

The old belief that acne is only an ordinary sign of puberty is weak and clinically risky. Many cases do improve after adolescence, but a significant number persist into adulthood or leave long-term skin changes when inflammation is not controlled. Acne can affect self-esteem, social activity, academic performance, professional communication, and emotional well-being. For this reason, acne should not be reduced to a cosmetic inconvenience; it is a medical condition requiring structured evaluation and rational treatment.

The relevance of this topic is determined by three practical factors. First, acne is highly prevalent among adolescents and young adults, the group most sensitive to visible skin changes. Second, inappropriate self-treatment is common: harsh scrubs, alcohol-based solutions, repeated squeezing of lesions, random antibiotic use, and unverified online remedies can worsen irritation and increase the risk of scarring. Third, modern therapeutic options are effective only when they are selected according to the clinical form and severity of disease. A patient with mainly comedonal acne, a patient with inflammatory papules, and a patient with painful nodular lesions should not be managed by the same simplified approach.

The aim of this article is to describe the etiology, clinical stages, and treatment modalities of acne vulgaris in a systematic international-article format. The main tasks are to classify etiological factors, explain the principal pathogenetic mechanisms, present the clinical grading logic, summarize treatment options by stage, and outline preventive care strategies that reduce relapse and post-acne complications.

Materials and Methods

This article was prepared using a descriptive and analytical method. The material base included dermatology textbooks, international clinical recommendations, national clinical protocols, review articles, and Uzbek medical educational sources translated into English for reference-list consistency. The article does not report a clinical trial, does not include personal patient data, and does not provide individual prescription schemes.

The methodological work was carried out in three stages. At the first stage, etiological and pathogenetic factors were grouped into biological, hormonal, microbial, inflammatory, environmental, and behavioral categories. At the second stage, clinical forms of acne were organized according to lesion type and severity: comedonal, mild inflammatory, moderate inflammatory, severe nodular, and complicated acne. At the third stage, treatment modalities were arranged into an algorithmic table that distinguishes topical therapy, systemic therapy, hormonal options, isotretinoin-based specialist treatment, skin care, and post-acne management.

The analysis followed a practical clinical logic: treatment should be chosen not by the patient's general complaint alone, but by the dominant lesion type, severity, duration, recurrence pattern, scarring risk, age, sex, pregnancy possibility, previous treatment response, and tolerance of local irritation. This method makes the article useful for academic discussion while preserving the boundary between educational information and individualized medical advice.

Table 1. Main etiological and pathogenetic factors in acne vulgaris

Factor	Pathogenetic role	Practical significance
Follicular hyperkeratinization	Excessive adhesion and accumulation of keratinocytes narrow or block the follicular canal.	Forms microcomedones and creates the structural basis for open and closed comedones.
Increased sebum production	Androgen-sensitive sebaceous glands produce more lipid-rich secretion.	Supports follicular obstruction and provides a favorable environment for inflammation.
Cutibacterium acnes	Participates in local microbial metabolism and stimulates inflammatory mediators.	Important in inflammatory papules and pustules; does not mean acne is a simple contagious infection.
Immune-inflammatory response	Cytokines, innate immunity, and perifollicular inflammation intensify tissue reaction.	Explains redness, tenderness, pustules, nodules, and the risk of post-inflammatory changes.
Hormonal influence	Androgens and hormonal imbalance affect sebaceous activity and lesion persistence.	Relevant in adolescent acne, adult female acne, menstrual flares, and hyperandrogenic signs.
Genetic predisposition	Family tendency can influence severity, persistence, and scarring risk.	Helps identify patients who need earlier and more careful control.



External and behavioral factors	Occlusive cosmetics, friction, squeezing, inappropriate cleansing, and some medications may aggravate lesions.	Modifiable risk factors should be corrected together with medical treatment.
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Results

The analysis confirms that acne develops through several interacting mechanisms rather than a single cause. The earliest event is usually microcomedone formation. When follicular keratinocytes do not desquamate normally, they accumulate inside the follicular canal. At the same time, increased sebaceous secretion makes the environment more lipid-rich and more prone to obstruction. Cutibacterium acnes can contribute to inflammation, but acne should not be described as a simple infectious disease. This distinction is important because it prevents the mistaken idea that antibiotics alone can solve the entire problem.

Clinically, the disease should first be divided by lesion type. Comedonal acne is dominated by closed and open comedones, usually without strong redness or pain. Mild inflammatory acne includes a limited number of papules and pustules. Moderate acne shows more numerous inflammatory elements, broader distribution, and a higher risk of residual spots. Severe nodular acne involves deep painful lesions and a high probability of scarring. Complicated acne includes post-inflammatory hyperpigmentation, atrophic scars, hypertrophic scars, or considerable psychological distress.

Table 2. Clinical classification of acne vulgaris by severity

Clinical stage	Typical manifestations	Management priority
Comedonal acne	Open and closed comedones; minimal inflammation.	Normalize follicular keratinization and prevent progression.
Mild inflammatory acne	Limited papules and pustules with mild redness.	Control inflammation and reduce new lesion formation.
Moderate inflammatory acne	More numerous papules and pustules; wider facial or truncal involvement.	Use combination therapy and monitor response.
Severe nodular acne	Deep painful nodules, cyst-like lesions, rapid scarring tendency.	Refer to a dermatologist and consider specialist systemic treatment.
Complicated acne	Hyperpigmentation, scars, recurrent flares, or psychological discomfort.	Stop active inflammation, prevent further scars, and plan post-acne correction only when appropriate.

Treatment results are most predictable when therapy is matched to severity. In comedonal acne, topical agents that target follicular plugging are central. In inflammatory acne, benzoyl peroxide, topical retinoids, azelaic acid, and limited topical antibiotic combinations may be used depending on clinical need and tolerance. In moderate cases, systemic antibiotics may be considered by a physician, but they should not be used casually or indefinitely. In severe nodular or scarring acne, dermatological supervision is necessary, and isotretinoin-based treatment may be considered when clinically justified.

A separate result of the analysis is that maintenance care is not optional. Many patients stop therapy immediately after visible improvement, which increases relapse risk. Maintenance strategies may include gentle cleansing, non-comedogenic moisturizers, sun protection, continued use of



selected topical agents as advised, and avoidance of lesion squeezing. Patient education improves adherence because acne treatment rarely produces a complete response in a few days. Most topical regimens require weeks of consistent use before stable improvement becomes visible.

Table 3. General treatment algorithm according to clinical stage

Clinical condition	Main treatment direction	Key cautions
Comedonal acne	Topical retinoid or azelaic acid; non-comedogenic skin care.	Some retinoids are unsuitable in pregnancy; irritation and dryness should be monitored.
Mild inflammatory acne	Benzoyl peroxide, topical retinoid, azelaic acid, or selected topical combinations.	Long antibiotic monotherapy increases resistance risk.
Moderate acne	Topical combination therapy with physician-supervised systemic options when necessary.	Systemic antibiotics should be time-limited and combined rationally.
Hormonal pattern in females	Assessment for endocrine or gynecological factors; selected hormonal approaches when appropriate.	Self-medication is unsafe; contraindications must be checked.
Severe nodular acne	Specialist management; isotretinoin may be considered in resistant or scarring disease.	Requires strict medical supervision, pregnancy prevention where relevant, and laboratory monitoring.
Post-acne scars or marks	After active inflammation is controlled: peels, laser, microneedling, or other specialist procedures.	Aggressive procedures during active acne may worsen irritation and pigmentation.

Discussion

The weakest approach to acne management is to treat every case as the same surface-level rash. That approach ignores lesion type, severity, scarring risk, sex-specific hormonal features, and previous treatment response. A more defensible approach begins with classification. Comedones, papules, pustules, nodules, scars, and pigmentation do not represent the same therapeutic problem. Each reflects a different balance between follicular obstruction, inflammation, depth of tissue involvement, and healing response.

Topical retinoids are valuable because they target the comedonal process and help prevent new lesions. Benzoyl peroxide is important in inflammatory acne because it reduces bacterial load and inflammation while also helping limit antibiotic resistance when used in combinations. Azelaic acid is useful in some patients because it can address both inflammatory lesions and post-inflammatory pigmentation with relatively broad tolerability. However, all topical treatments must be introduced with attention to irritation, dryness, skin sensitivity, and patient adherence.

Antibiotic use deserves special criticism. Using topical or oral antibiotics alone for a long period is a poor strategy because it encourages antimicrobial resistance and does not adequately treat comedone formation. In rational acne therapy, antibiotics are not a universal answer; they are one component reserved for selected inflammatory cases and usually combined with non-antibiotic



topical agents. This principle is especially important in settings where antibiotics are easily purchased without adequate medical supervision.

Isotretinoin is one of the most powerful options for severe or treatment-resistant acne, but it is also one of the most misunderstood. It should not be viewed as a cosmetic shortcut. Its use requires assessment of indications, side effects, pregnancy-related risk where applicable, laboratory monitoring, and follow-up by a qualified specialist. When used correctly in appropriate patients, it can reduce severe inflammation and scarring risk; when used casually, it creates avoidable safety problems.

Diet and lifestyle should be discussed without exaggeration. High-glycemic foods and some dairy-related factors may aggravate acne in certain individuals, but strict and extreme diets are not scientifically justified for every patient. A balanced approach is stronger: reduce excessive sugar intake, observe individual triggers, avoid unnecessary restrictions, and maintain general health. For adolescents, aggressive dietary limitation may create more harm than benefit.

Skin care is often where patients damage their own outcomes. Harsh scrubbing, frequent washing, alcohol-based products, heavy oily cosmetics, and squeezing of lesions can intensify irritation and increase the likelihood of post-inflammatory spots or scars. The practical standard should be simple: gentle cleansing, non-comedogenic products, appropriate moisturization, sun protection, and consistent continuation of medically advised therapy. This is not secondary advice; it is part of the treatment system.

Table 4. Skin care and relapse-prevention directions

Direction	Recommended practice	Expected benefit
Cleansing	Use a mild cleanser suitable for facial skin once or twice daily.	Reduces irritation and removes excess oil without damaging the barrier.
Cosmetics	Choose non-comedogenic and oil-free products; remove makeup before sleep.	Decreases follicular obstruction and cosmetic acne flares.
No squeezing	Avoid pressing papules, pustules, and nodules by hand.	Reduces scarring, pigmentation, and secondary infection risk.
Sun protection	Use a light non-comedogenic sunscreen when exposed to sunlight.	Helps limit post-inflammatory hyperpigmentation.
Maintenance care	Continue dermatologist-recommended maintenance after visible improvement.	Reduces relapse and supports long-term control.
Specialist referral	Seek dermatological care for painful nodules, scars, fast worsening, pregnancy, or hormonal signs.	Allows early prevention of serious complications.

Conclusion

Acne vulgaris is a chronic, multifactorial inflammatory disease rather than a simple cosmetic defect. Its development involves follicular hyperkeratinization, increased sebum production, Cutibacterium acnes-related inflammatory stimulation, hormonal effects, genetic predisposition, immune response, and modifiable external factors. Correct evaluation of clinical stage is the foundation of effective management. Comedonal acne requires a different strategy from inflammatory acne, and severe nodular acne requires early specialist involvement because the risk of scarring is high.



The most important principles of acne management are early classification, rational combination therapy, avoidance of prolonged antibiotic monotherapy, patient education, protection of the skin barrier, prevention of lesion manipulation, and continuation of maintenance care after improvement. Dermatological literacy is essential because incorrect self-treatment may transform a manageable disease into a long-lasting problem with scars and psychological consequences. A stage-based and evidence-informed approach can improve skin outcomes and reduce the burden of acne on quality of life.

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