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Recent findings in the epidemiologic evidence, classification, and subtypes of acne vulgaris

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Acne affects between 40 to 50 million individuals in the United States. Recent findings regarding the multifactorial pathogenesis of acne have facilitated a reexamination of the classification of acne and acne-related disorders. Disorders without a microcomedo as the initial pathologic condition are no longer classified as "acne." Research has also identified that the clinical characteristics of acne vary with age, pubertal status, gender, and race. These findings may have implications for the clinical management of acne and acne-related disorders.
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Acne is a disease with an initial pathologic condition involving a microcomedo. True acne is characterized by a follicular eruption of the comedo, which initiates an inflammatory reaction. The formation of papules, pustules, and/or cysts can result from the inflammation. Subtypes of acne vulgaris are indicative of the particular cause of the disease, such as acne fulminans or cosmetica. Acne can also be further defined by the age at onset, as with neonatal or infantile acne.

The identification of the pathogenesis of acne as originating with a microcomedo resulted in a

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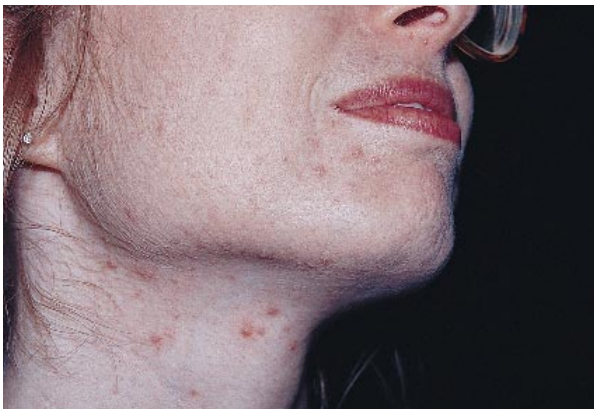

Fig. 2. Adult acne: young woman with the typical inflammatory acne of the lower face and neck. The condition relapsed after 2 courses of isotretinoin.

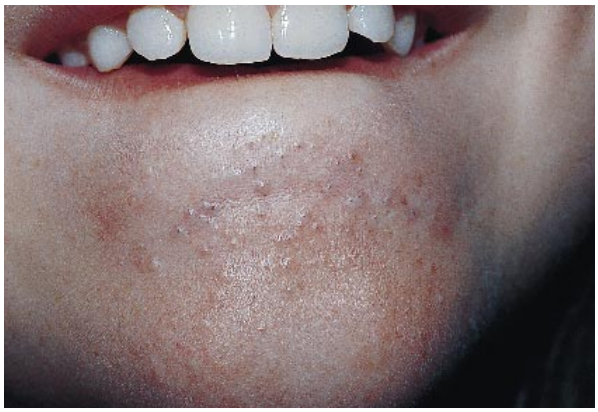

Fig. 1. Comedonal acne: multiple open and closed comedones are seen in this preteenaged patient.

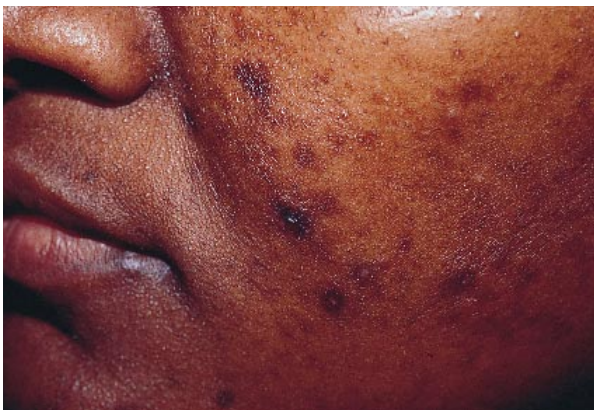

Fig. 3. Hyperpigmented macules: hidden inflammation in the darker-skinned patient with acne produces prominent postinflammatory hyperpigmented macules. Much of the melanin is epidermal.

Table I. Number of individuals in the United States with acne

Age range (yrs)	Prevalence (%)	US population	Calculated no. of individuals with acne
12-24	85	47,311,000	40,214,350
25-34	8	40,393,000	3,231,440
35-44	3	43,257,000	1,297,710
Total			44,743,500

Table II. Comparison of trends in acne for various ages

Age group	Location	Morphologic condition	Sex
Neonates	Nose, cheeks, forehead	Comedonal	Both
Infants	Face	Inflammatory	Males
Preteens	Centrofacial	Comedonal	Both
Teens	Face, trunk	Mixed	Both
Adults	Perioral, jawline, chin	Inflammatory	Women

reevaluation of acne-like disorders historically classified as acne. Disorders such as rosacea and steroid acne, although clinically similar to acne, are now classified as acneiform eruptions, because their initial pathologic conditions involve inflammatory lesions, bypassing the comedo stage. The pathogenesis of these disorders differs significantly from that of acne and may result in the need for different therapeutic approaches.

Difficulties abound in estimating the prevalence of this disease. Prior estimates most likely included acneiform eruptions in their counts; in addition, it is difficult to identify the criteria used for inclusion. Although clinicians have objective criteria to diagnose the disease, there is controversy regarding the minimal presentation of symptoms necessary for this diagnosis (e.g., a patient experiencing 1 pimple every other month versus a teenage boy with numerous comedones). One approach to this determination is to use the current census data in combination with acne prevalence data. Table I combines 1996 census data[1] with prevalence rates of acne by age,[2] and shows that approximately 45 million individuals in the United States or between 40 to 50 million individuals in the United States have acne.

ACNE THROUGHOUT THE LIFE CYCLE

People of all ages are affected by acne, ranging from neonatal and infantile acne throughout adult acne vulgaris. Generally, the clinical presentation and morphologic evidence of acne at the different ages varies (Table II). However, there is significant overlap among the different age categories.

Neonatal acne

Neonatal acne is characterized by multiple small, erythematous closed comedones on the nose, forehead, and cheeks of a neonate, with an onset frequently between 0 and 6 weeks of age. Nearly 1 in 5 neonates exhibit mild neonatal acne. Less frequently, neonates have open comedones, inflammatory papules, or pustules. While the cause is not clearly defined, it is believed to be influenced by the stimulation of the neonatal sebaceous glands from maternal and infant androgens. Neonatal acne lesions predominantly resolve spontaneously within 1 to 3 months, with no scarring. There does not appear to be any relationship between neonatal acne and the later development of acne vulgaris.

Infantile acne

Acne that is seen later in infancy, with an onset beginning in months 3 to 6, is classified as infantile acne. It is less common than neonatal acne and is characterized by more numerous inflammatory papules and pustules, with occasional presentations of nodular acne. Scarring from infantile acne is a risk. Infantile acne affects more male infants than female infants and is postulated to be associated with the precocious secretion of gonadal androgens. Patients with infantile acne may have an increased risk of the development of severe acne vulgaris during their teenage years. In some severe cases, medical testing for an underlying cause may be indicated; an endocrinologic examination may focus on detecting elevations in dehydroepiandrosterone sulfate (DHEAS) or free testosterone.

Acne vulgaris

Teenage years. Hormonal surges before and during puberty are often related to the onset of typical acne vulgaris. Teenaged boys are more frequently affected than teenaged girls, with some

estimates suggesting that 100% of teenaged boys are affected by acne. Adrenal maturation and gonadal development cause androgen production and subsequent sebaceous gland enlargement. Both sebum production and the levels of DHEAS increase during puberty. In fact, an oily face and scattered comedones throughout the face and chin may be one of the first signs of puberty (Fig. 1).

In 1 study by Lucky et al,[3] 439 African American and 432 white girls aged 10 to 15 years were evaluated by their pubertal stage, hormone levels, and acne severity. This study found that acne severity correlated better with pubertal stage than chronologic age; however, the best predictor of the severity of acne at age 15 years was the severity of acne at age 10 years. Among the serum hormone levels assessed, DHEAS correlated best with acne; testosterone and free testosterone each manifested a weak correlation. Although this study suggested that DHEAS levels can be used to predict acne severity in the general population, DHEAS levels are not predictive for specific individuals: only 29% of girls with severe acne had DHEAS levels of more than the 90th percentile, whereas 77% of girls with DHEAS levels more than the 90th percentile did not have severe acne.

Adult acne. After peaking during the teenage years, the prevalence of acne progressively decreases, affecting approximately 8% of adults aged 25 to 34 years and only 3% of adults aged 35 to 44 years. Most teenaged boys with acne can anticipate a clearing by the age of 20 to 25 years. In contrast, women may continue to experience acne well into their adult life, up to and even beyond age 40 years (Fig. 2). Young adult acne can be a continuation of teenage acne or start de novo. Clinically, it may differ from teenage acne (Table II) in that it tends to be more inflammatory with fewer comedones. In addition, lesions of adult acne are located most commonly around the mouth, chin, and jawline.

One study[4] reviewed 200 patients with acne aged 25 to 55 years (mean age, 35.5 years), with the majority being women (76%); among this cohort, inflammatory lesions with mild to moderate severity and scarring predominated. Persistent acne was present in most of the patients, although 18.4% of women and 8.3% of men had onset of acne after age 25 years. Most patients (82%) had not responded to multiple courses of oral antibiotics, and approximately one third of the patients had experienced a relapse after treatment with 1 or more courses of isotretinoin. Eighty-five percent of the women experienced a premenstrual flare of their acne. Cosmetics, drugs, and occupation were not found to be etiologic factors.

Numerous causalities have been proposed to explain adult onset acne, including stress[5], cosmetics, and a difference in *P. acnes* among adults[6-7]. Although none of these propositions have been substantiated, one study did find a difference in women between late-onset acne and acne that persisted from adolescence; there were significantly higher sebum excretion rates (as versus controls) among women whose acne originated during the teenage years[8].

Acne in African Americans. Until recently, it was believed that inflammation was far more common among Caucasian patients than African American patients.[9] A recent investigation,[10] however, found marked histologic inflammation in facial acne lesions that appeared clinically to be noninflammatory. In addition, it was found that much of the melanin of postinflammatory hyperpigmented macules is epidermal and not dermal[10-11] (Fig. 3). Clinically, this finding suggests that the superficial localization may facilitate a topical approach to the treatment of postinflammatory hyperpigmented macules.

SUBTYPES OF ACNE

Acne can be delineated into a variety of subclassifications. Acne conglobata and acne fulminans are both forms of cystic acne characterized by the formation of deep inflammatory lesions that often cause scarring.

Acne cosmetica is most commonly found in women between the ages of 20 to 40 years and is associated with the use of cosmetics containing comedogenic substances. It is characterized by persistent, low-grade, and closed comedones and slowly resolves with the cessation of use of the causative agent. Pomade acne is a variation of acne cosmetica seen almost exclusively in African Americans and is associated with the use of greases and oils applied to the scalp and face. Lesions are closely packed closed comedones found only at the site of pomade application.

ACNEIFORM ERUPTIONS

Historically, acne-like diseases, such as rosacea, steroid acne, and Gram-negative folliculitis, were

considered to be subcategories of acne. However, these diseases are now classified as acneiform eruptions because of the absence of a comedo stage in their pathogeneses. This reclassification may have ramifications on the clinical management of these disorders.

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