

Chapter 2

Clinical Presentation of Acne

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2.1 Introduction

Acne is a disease of the pilosebaceous unit that typically begins on the face and has a spectrum of lesions and severity (Table 2.1). The classic lesion is a pustule, but inflammatory papules and nodules are common. The primary lesion, from which all others develop, is the microcomedo, an impaction and distention of the follicle with sebum and improperly desquamated keratinocytes from the follicular epithelium. When microcomedones become visible, they are described as open or closed comedones. An open comedo has a visible pore that appears as a dark spot (Fig. 2.1). The pigment is not dirt, but is oxidized lipid and melanin. Closed comedones have a pore too small to see and appear as white bumps (Fig. 2.2).

In patients who are hypersensitive to *Propionibacterium acnes* that colonizes follicles, [1] inflammatory lesions may develop from the microcomedones. Papules and pustules may be superficial or deep and scarring depending on the vigor of hypersensitivity (Fig. 2.3). Nodules are inflammatory lesions >0.5–1 cm in size. Nodules may develop into abscesses, which have incorrectly been termed “cysts” (Fig. 2.4). The term “nodulocystic acne” is incorrect, but probably too deeply ensconced to readily fall from use. *Conglobate* lesions are intensely inflamed neighboring nodules that merge into a loculated abscess. Secondary lesions such as scars, keloids, sinus tracts, and true cysts may follow in the most inflammatory disease.

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Table 2.1 The spectrum of acne lesions

<i>Noninflammatory</i>
Microcomedo
Open comedo
Closed comedo
<i>Inflammatory</i>
Papule
Pustule
Nodule/abscess
Conglobate lesions
<i>Secondary lesions</i>
Scars, keloids, hypertrophic scars
True cysts
Sinus tracts

Fig. 2.1 Mixed open comedones (blackheads) and closed comedones (whiteheads) on the forehead of a teenage girl (Photo credit: Joshua A. Zeichner, M.D.)



Fig. 2.2 Closed comedones (whiteheads) on the forehead of a teenage boy (Photo credit: Joshua A. Zeichner, M.D.)



2.2 The Onset of Acne

Acne usually begins with the onset of puberty [2, 3]. Androgens stimulate sebaceous secretion and microcomedones inflate with sebum and become visible. Typically initial lesions are noninflammatory and centrafacial (Fig. 2.5). As maturation progresses

Fig. 2.3 Mixed comedonal acne with inflammatory papules and scattered pustules on the forehead of a teenage girl (Photo credit: Joshua A. Zeichner, M.D.)



Fig. 2.4 Severe, inflammatory acne on the cheeks of an adult woman. Pustules, papules, nodules, and scars are clinically apparent (Photo credit: Joshua A. Zeichner, M.D.)



inflammatory lesions may appear, and the distribution spreads across the face and perhaps to the trunk (Fig. 2.6). Severity of acne is clearly correlated with the stage of puberty. Lucky et al. [4] has shown that early in puberty comedonal acne is common, but inflammatory acne is rare. In later stages of puberty, inflammatory acne may reach a 50 % incidence in boys [5]. Mourlatos and colleagues [6] demonstrated that both sebaceous secretion and follicular *P. acnes* colonization are elevated early in those children destined to develop acne.

In some patients, adrenarche is a sufficient stimulus and acne may appear in 7–11-year-olds. Lucky [4] has shown that early acne in girls reflects rising levels of adrenal dehydroepiandrosterone sulfate (DHEAS). She also found that such patients tended to have more severe acne as teens, though a sign of difficult acne to come, such as *preadolescent acne*, is not a cause for medical concern. However, acne that occurs between 1 and 7 years, termed *mid-childhood acne*, is much more unusual and may reflect an underlying medical condition such as endocrinopathy or tumor. Work-up by a pediatric endocrinologist is indicated [3].

Fig. 2.5 Open comedones (blackheads) on the nose of an adolescent boy. This acne was one of the first signs of puberty (Photo credit: Joshua A. Zeichner, M.D.)



Fig. 2.6 Truncal acne characterized by inflammatory papules on the back (Photo credit: Joshua A. Zeichner, M.D.)



2.3 Grading Acne Severity

Grading acne is a surprisingly difficult task. The severity of acne varies widely among patients and even during the course of disease in a single patient. The traditional grading method in clinical trials involves counting inflammatory and noninflammatory

lesions, but this is confounded by the variable severity of inflammatory lesions. Papules may be barely visible, or deep, scarring, or just short of a nodule, and still be counted as equivalent lesions.

Determination of acne severity in clinical trials is problematic. Tan and colleagues [7] have analyzed the gradable aspects of acne and list lesion type, number of lesions, extent of lesions, regional involvement, secondary lesions, and patient experiences as important considerations. Cunliffe and colleagues [8] and more recently Dreno and coworkers [9] have developed grading systems based on comparison of the patient with standardized photos of acne of varying severity that in some measure answers this problem. A limitation of these systems is that they are not as quantifiable as lesion counting. Clinical trials typically rely on a combination of lesion counting and severity grading in order to generate both quantifiable and comparable data. The situation is not yet ideal.

Judging severity in day-to-day practice is much less complex. At the initial visit, experienced clinicians look at the type and number of lesions, the severity of inflammation, the presence of pigmentary disturbance and scarring, and the distribution of lesions and quickly have a grasp of the severity and likely response to different treatments. The presence of deep inflammation, nodules, scarring, and trunk lesions all point toward more severe acne. A second but important factor in judging acne severity is the effect of the disease on the patient. If each pimple is a tragedy, then even mild disease is severe.

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