

Clinical features

Acne is the easiest skin condition to diagnose because of its distinctive anatomic distribution, age of occurrence, and polymorphic appearance in which the comedones (blackheads especially) are the most unique and pathognomonic feature [Figures 9–11]. Typically it starts at puberty, in girls between age 12–14 years, and in boys between 14–16 years. Occasionally, it may start at age 7 or 8 years (adrenarche) and when it does, it portends severe acne. Acne waxes and wanes through adolescent years and early adult life, it peaks between 17–21 years. In about 90% of sufferers, acne will spontaneously remit before 30 years of age. In the rest, its course is unpredictable, and it may persist well into the fifth and the sixth decade (persistent acne). Less commonly, acne may make its first appearance in mature adults – after 30 years (adult acne). Adult acne is more common in women and is often a part of cutaneous hyperandrogenism [Figures 12-14].^[1,2]

Face is the theater of action. It is involved in 99% of acne sufferers.^[2] Back is involved in 60% and chest in 15%.^[2] Preadolescent acne and early adolescent acne is largely confined to the forehead [Figure 9]. With time there is cephalocaudal migration. Typical adolescent acne involves the entire face but shows predilection for cheeks [Figures 10, 11, 15-17]. In men who have grown beard, it relatively spares the lower face and neck. In severe cases, even external ears are involved. Adult acne is largely confined to the jaw area [Figure 18]. Acne vulgaris (common acne) shows variable involvement of seborrheic areas of upper torso, namely, upper back, sternal area, shoulders, and upper arms [Figures 19 and 20] [Table 2]. In some cases, acne is largely confined to the torso with little or no involvement of the face (acne corporis) [Figures 21 and 22]. Acne corporis denotes severe acne and often involves the entire back and in some cases

even buttocks and thighs. Acne does not occur on the scalp although folliculitis occasionally co-occurs and is misdiagnosed as acne. *Concomitant folliculitis*, especially pityrosporum folliculitis, is more common than is recognized [Figure 23].^[3]



Figure 9: Preadolescent acne



Figure 10: Adolescent comedonal acne. Note comedones on cheeks and chin also.

Table 2: Comparison of acne distribution for different age groups

Age group	Location of lesions	Type of lesions	Sex
Neonates	Cheeks, chin, eyelids, forehead	Papules and pustules, no comedones	Both
Infants	Full face	Comedones, papules, nodules, scars	Male
Preadolescent	Forehead, upper cheeks, nose	Predominantly comedonal, occasional papule	Both
Adolescent	Full face, seborrheic areas of torso	All types of lesions	Both
Adults	Chin, upper lip, jaws	Papules, excoriated papules	Female

Acne shows a variable and fluctuating mix of comedones, folliculocentric inflammatory lesions, scars, and pigmentary disturbances. The full spectrum



Figure 11: (a and b) Postadolescent comedonal acne. Acne venenata (cosmetica) presents in this manner



Figure 12: Adult acne. Typically sparse and occurring on chin and jaw areas, frequently associated with hyperandrogenism (PCOD and mild SAHA features in this case)



Figure 13: Acne on chin and jaw areas in mature adults is androgenic in character and appears deceptively mild



Figure 14: (a, b and c) Mild but persistent acne in a 65-year old, treated with low-dose isotretinoin and metformin



Figure 15: Adolescent acne vulgaris – grade I



Figure 16: (a and b) Adolescent acne – grade II. Note open and closed comedones, papules, pustules, macules, scars, and steatosis



Figure 17: (a and b) Acne – grade III. Typical bilateral asymmetry, polymorphic picture, displaying a range of comedones, inflammatory lesions, and scars, arising on a background of steatosis

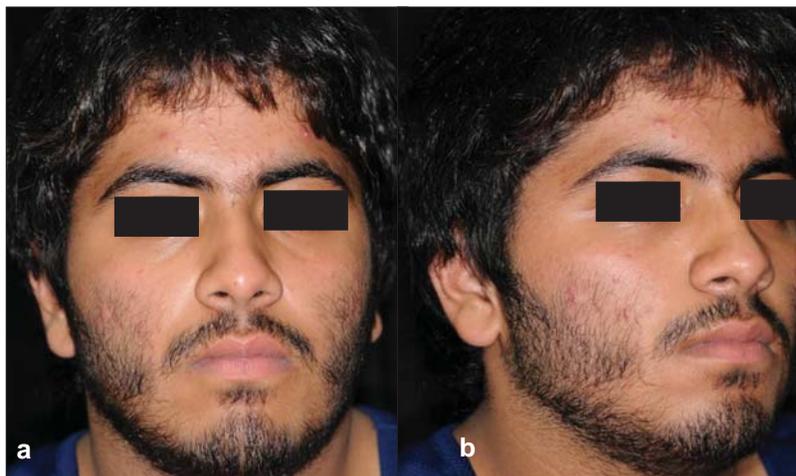


Figure 18: (a and b) Acne tends to spare beard and other hairy areas



Figure 19: (a, b and c) Acne vulgaris. Face is involved in 99%, back in 60% and chest in 15%



Figure 20: (a and b) Acne vulgaris. predominantly on the torso, face was less involved



Figure 21: Steroid-treated acne corporis. Note pus filled bullae, striae, and hypertrichosis



Figure 22: (a and b) Hidradenitis suppurativa is occasionally associated with nodulocystic acne



Figure 23: Pityrosporum folliculitis. Superficial pustules and crusted papules on glabella and paranasal areas are the clues



Figure 24: Showing open and closed comedones, macrocomedone, and a submarine comedone



Figure 25: Missed comedones come into view upon stretching the skin (b)

includes many types of comedones [Table 3] – blackheads (open comedones), whiteheads (closed comedones), missed comedones, macrocomedones, sandpaper comedones, submarine comedones [Figures 24-26];^[4] inflammatory lesions – papules, pustules, nodules, cysts, macules [Figures 27-31]; atrophic scars – rolling, boxcar, ice-pick; hypertrophic

scars and keloids; and acne hyperpigmented macules (AHM) [Figures 32-38].^[5] The precursor lesion is a microcomedo which evolves into open or closed comedone and further into papule or nodule. Microcomedo may also directly evolve into papule or nodule [Figure 2]. The diagnosis of acne is untenable in the absence of comedones. Yet 7% of acne patients do not show comedones at first evaluation.^[4] On the other hand, the presence of a single comedone is enough to diagnose acne! The preadolescent acne and the beginning of adolescent acne is largely comedonal.^[6] Acne papules, pustules, and nodules appear later. Acne severity is gauged by the number and types of inflammatory lesions [Table 4] [Figure 39].

Table 3: Types of comedones ^[7]
Open comedones (blackheads)
Closed comedones (whiteheads)
Grouped comedones
Missed comedones
Macrocomedones
Sandpaper comedones
Submarine comedones
Nevoid comedones
Drug-induced comedones

Acne papules are conical, measure 3 mm or less, and often show a pore at the summit. Pustules are similar in size and represent more intense inflammation; they are sterile on routine cultures. Nodules measure 4 mm



Figure 26: Grouped comedones are pathognomonic of acne conglobata



Figure 27: (a, b and c) Acne vulgaris. Note predilection for cheeks and bilateral asymmetry



Figure 28: (a, b and c) Grade II scarring acne showing pan-facial distribution, a mix of comedones, active and resolving inflammatory lesions, scars, and hyperandrogenism



Figure 29: Acne macules represent an intermediate stage between papules and scars



Figure 30: Excoriated acne papules, acne macules, and AHM's



Figure 31: Persistent acne showing open and closed comedones, resolving inflammatory lesions, a variety of scars, and hirsutism



Figure 32: Ice-pick acne scars



Figure 33: Rolling scars



Figure 34: Boxcar scars



Figure 35: Hypertrophic acne scars. Acne scars as a rule are atrophic on the face and hypertrophic on the sternal area and back



Figure 36: Perifollicular elastolysis appearing as minute pits is a sequela of acne vulgaris. Also seen are shallow boxcar scars, comedones, and a solitary papule

Table 4: IAA grading of acne severity

Mild acne (Grade I)	Comedones < 30
Predominance of comedones	Papules < 10
	No scarring
Moderate acne (Grade II)	Comedones any number
Predominance of papules	Papules > 10
	Nodules < 3
	Scarring ±
Severe acne (Grade III)	Comedones any number
Many nodules	Papules any number
	Nodules/ cysts > 3
	Scarring +

Grading of acne is a complex issue. Many grading systems have been devised and deployed over the years to meet different clinical and research requirements. The above grading system is proposed, to pair with algorithms developed by the IAA, to facilitate uniformity and consistency in application of the latter

or larger and show tenderness that is proportional to size. Nodules sometimes soften and become fluctuant when they are erroneously described as cysts; at best they are pseudocysts [Figures 40-41]. Such lesions may further evolve into hemorrhagic crusts, abscesses, or sinus tracts [Figure 42]. Nodules carry high potential for scarring, especially in the genetically prone. Acne macules represent resolved papules/nodules and often precede atrophic scars [Figure 29]. Acne scars [Table 5] are invariably atrophic on the cheeks, and hypertrophic/ keloidal over the jaw angles [Figure 35], shoulders, upper back, and sternal areas. Papular scars are sometimes encountered on the chin [Figure 37] and nose, perifollicular fibrosis and perifollicular elastolysis on the back, chest, and neck. Macular



Figure 37: Papular acne scars on chin



Figure 38: Acne hyperpigmented macules. A case of late-onset adult acne with lower face predilection. AHM's are more common in acne excoriee

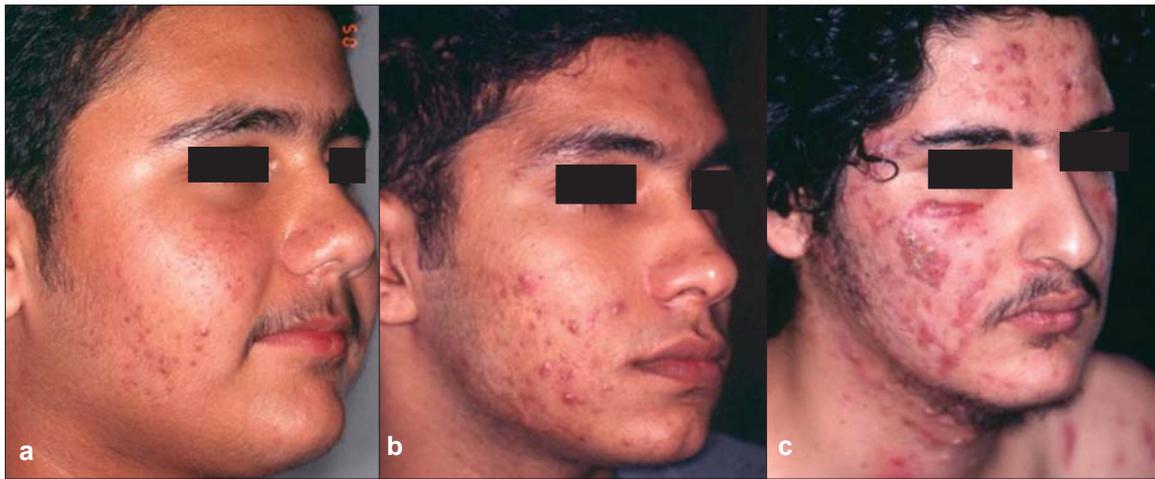


Figure 39: (a, b and c) Grade I, II, III acne

Table 5: Classification of acne scars^[9]

Atrophic scars	Ice-pick scars Boxcar scars Rolling scars Perifollicular elastolysis Macular atrophic scars
Hypertrophic scars	Keloids Papular scars Perifollicular fibrosis
Mixed scars	
Unclassifiable scars	

atrophic scars are large, 5–20 mm, somewhat circular, soft, and erythematous, and occur on the back. Mixed scars and unclassifiable scars are also encountered [Figure 43]. Scarring is seen in 22% of acne sufferers. Rarely, severe acne scars are complicated with osteoma cutis.^[8]

Acne variants [Figures 44-55]: Acne offers a wide spectrum of clinical subtypes. Some subtypes are aggressive or severe and are associated with constitutional symptoms and systemic involvement. This group includes acne conglobata [Figure 44], acne fulminans, pyoderma faciale (rosacea fulminans) [Figure 45], and SAPHO syndrome (synovitis, acne, pustulosis, hyperostosis, osteitis) [Figure 46]. SAHA syndrome (seborrhea, acne, hirsutism, alopecia) [Figures 47-49] denotes acne in the context of endocrine abnormality, particularly polycystic ovarian syndrome. Infantile acne is sometimes severe and prolonged and warrants evaluation for adrenal or gonadal tumor. Acne excoriée [Figures 29 and 56] is often a manifestation of dysmorphophobia. Frictional acne, acne mechanica, acne cosmetica, and tropical acne (acne Mallorca) are mild variants or acne look



Figure 40: Acute acne cyst in an otherwise quiescent disease



Figure 41: (a and b) Nodulocystic acne. Initial flare up after starting isotretinoin therapy. Note impetigo on preauricular area, also related to isotretinoin



Figure 42: Acne sinus tract (arrow). These are elongated fluctuant swellings and result from coalescing acne cysts



Figure 43: (a and b) Mixed atrophic acne scars: boxcar (blue arrows), ice-pick (black arrows), perifollicular elastolysis (yellow arrow)

alikes. Drug-induced acne includes drug aggravation of pre-existing acne (androgenic hormones, anabolic steroids, INH, lithium) or a de novo acneiform eruption (oral corticosteroids, halogens) [Table 1].

Clinical differential diagnosis: Many skin conditions mimic acne [Table 6]. Some are more acneiform than

Table 6: Differential diagnosis of acne

Rosacea
Perioral dermatitis
Lupus miliaris disseminatus faciei (previously called acne agminata)
Papular sarcoidosis
Folliculitides (pityrosporum, candidal, demodex, staphylococcal, Gram negative, herpetic)
Adenoma sebaceum
Verruca plana
Molluscum contagiosum



Figure 44: (a, b, c and d) Acne conglobata. Note giant abscesses on the back and upper sternal area, and grouped comedones on the shoulder



Figure 45: (a and b) Pyoderma faciale successfully treated with steroids, antibiotics, isotretinoin, and metformin



Figure 46: Acne fulminans in a case of SAPHO syndrome (Synovitis, Acne, Pustulosis, Hyperostosis, Osteitis)



Figure 47: SAHA syndrome. Seborrhea, Acne, Hirsutism, Alopecia

others. This is a large group and includes: rosacea, perioral dermatitis, folliculitides – namely, Gram-negative folliculitis, other bacterial folliculitides, pityrosporum folliculitis, candida folliculitis, demodex folliculitis, herpetic folliculitis, and HIV-associated eosinophilic pustular folliculitis, lupus miliaris disseminatus faciei, adenoma sebaceum, milia,



Figure 48: SAHA syndrome with melasma. Acne and melasma often co-occur in Indian patients



Figure 49: SAHA syndrome

verruca plana, and molluscum contagiosum. All of the above conditions lack comedones and are thus easily distinguished from acne. However, some of these conditions may co-occur with acne and that may pose diagnostic difficulty.

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Figure 50: (a and b) Acanthosis nigricans is increasingly being documented in acne patients most of whom also have hirsutism, FAGA, and recalcitrant seborrhea capitis



Figure 51: Acne as a part of HAIR-AN syndrome



Figure 52: HAIR-AN syndrome. HyperAndrogenism, Insulin Resistance, and Acanthosis Nigricans. Note coarse, steatotic skin with comedones, acanthosis on lateral forehead, periorbital, perioral areas and neck, and bleached facial hair



Figure 53: (a and b) Acne and acanthosis nigricans. Additional treatment with metformin is beneficial in such cases



Figure 54: (a and b) Late-onset congenital adrenal hyperplasia; before and after treatment with Diane-35, low-dose isotretinoin and dexamethasone suppression



Figure 55: Folliculitis nuchae, previously termed acne keloidalis nuchae



Figure 56: Acne excoriee. Dysmorphophobia is a frequent underlying cause

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