SKIN
ACNE AND HAIR

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DISORDERS PRESENTING IN THE SKIN AND MUCOUS MEMBRANES

Acne

- Inflammation of pilosebaceous unit
- 10-17 years of age in females
- 14-19 years of age in males
- May appear first >25 years of age
- More severe in males
- Milder in Asians
- Inflammatory acne is marked by erythematous papules, nodules, and pustules.
- Depending on the stage of the disease, open or closed comedones, papules, pustules, or deep inflammatory nodules may develop.

Acne

- Presents as
- Comedonal acne
- Open, have large patulous orifices
- Closed, lack orifice
- Papulopustular acne
- Nodulocystic acne
- Acne conglobata
- Sinus tract formation and dermal scarring
- Severe acne (conglobata) associated with XYY syndrome

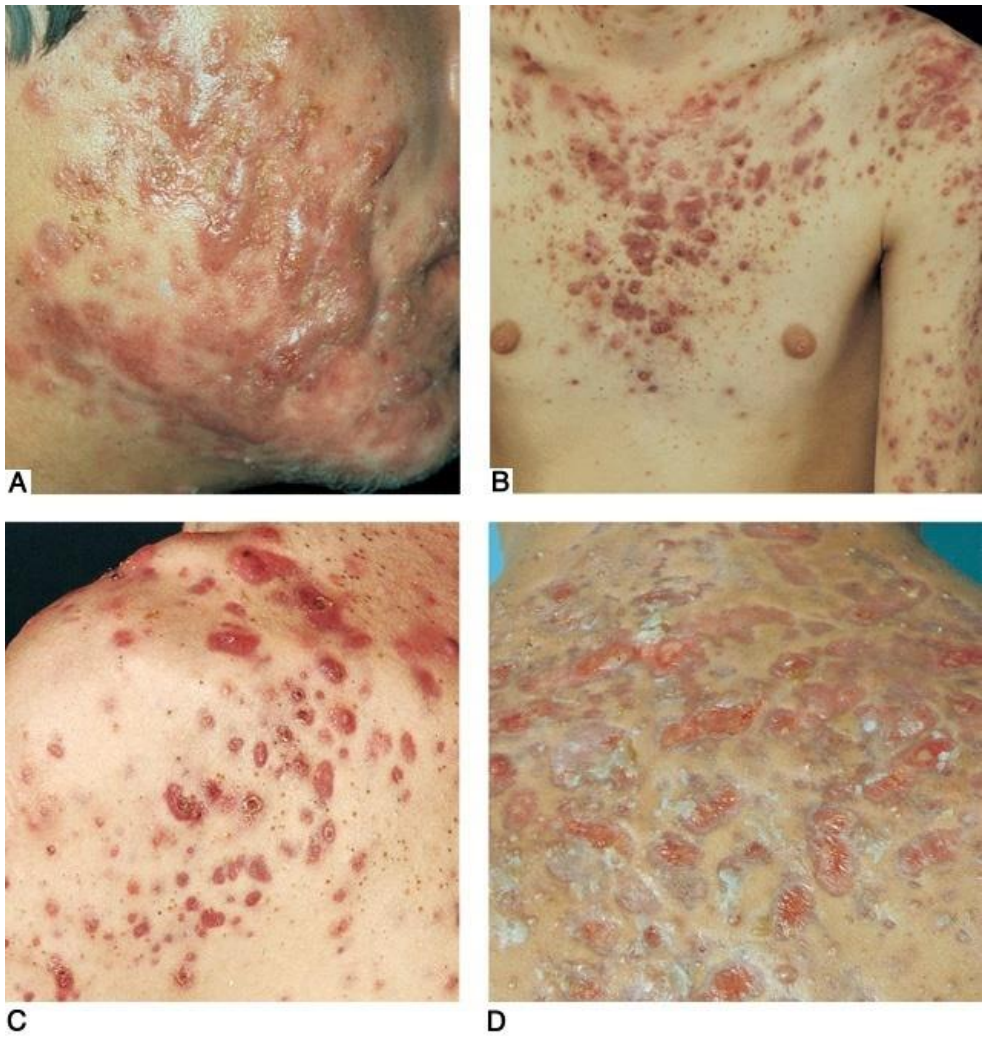
Acne

- Acne fulminans presents in 13-17 year-old males with severe cystic acne and ulceration
- Malaise, fatigue, fever, generalized arthralgias
- Pitted, depressed, or hypertrophic scars may follow
- Particularly nodulocystic acne
- Exacerbations caused by
- Emotional stress
- Occlusion and skin pressure
- Androgens
- 11-OH hydroxylase block

Acne

- Pathophysiology
- Androgens stimulate sebum production
- Propionibacterium acnes contains lipase that converts lipid into fatty acids
- Both cause sterile inflammatory response in the pilosebaceous unit
- Leads to hyperkeratinization, follicle plugging
- Black color is tyrosine oxidation
- Distended follicle walls may break, spill contents into dermis (particularly if closed comedone)
- Foreign body response

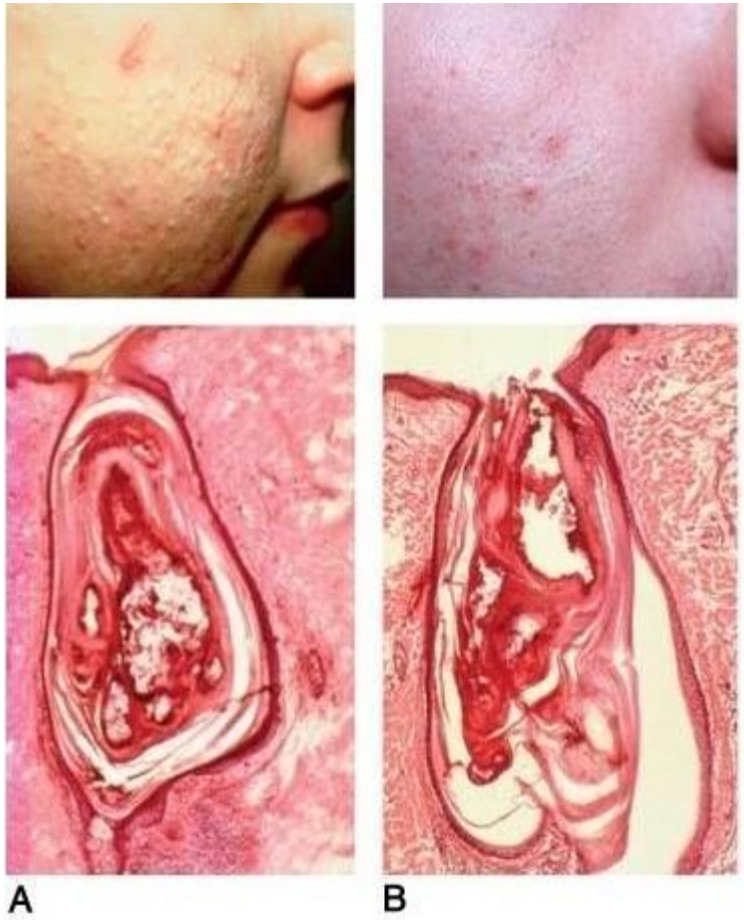
Acne



A. Large, confluent nodules formed by confluence of smaller lesions with interconnecting channels, associated with atrophic and hyperplastic scars.
B. Extensive nodules on the chest and arms with severe scarring.
C. Close-up of nodules, crusted ulcers, and scars on the shoulder.
D. Severe nodular acne of the back with little residual uninvolved skin.

Fig. 78-6
Accessed 07/20/2010

Acne



A. Closed comedone. The follicular infundibulum is distended, filled with keratin and sebum, and the follicular epithelium is attenuated. The follicular ostium is narrow.
B. Open comedone. Resembles the closed comedone with the exception of a patulous follicular ostium.

Wolf, K, Goldsmith, LA, Katz, SI, Gilchrest, BA, Paller, AS, Leffel, DJ, Fitzpatrick's Derrmatology in General Medicine, 7th edition. Fig. 78-3

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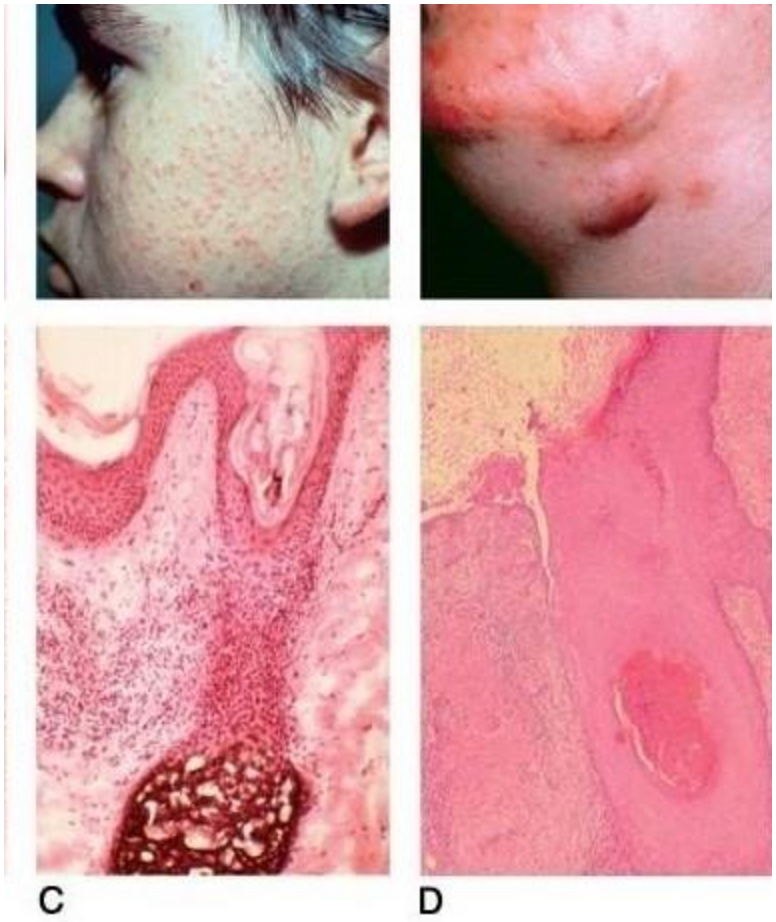
Acne



C. Inflammatory papule. Acute and chronic inflammatory cells surround and infiltrate the follicle, which shows infundibular hyperkeratosis.

D. Nodule. The follicle is filled with acute inflammatory cells. With the rupture of the distended follicle, there is a foreign body granulomatous response.

Wolf, K, Goldsmith, LA, Katz, SI, Gilchrest, BA, Paller, AS, Leffel, DJ, Fitzpatrick's Derrmatology in General Medicine, 7th edition. Fig. 78-3

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Therapy

- Comedonal acne treatment regimen
- Topical clindamycin and erythromycin
- Topical benzoyl peroxide gels
- Topical isoretinoin
- Inhibits sebaceous gland production and hyperkeratinization
- Teratogenic
- Cause hypertriglyceridemia
- Cause pseudotumor cerebri

Therapy

- Minocycline added as inflammatory response progresses.
- Oral contraceptives containing ethinyl estradiol are useful in women to reduce sebum production.
- Spironolactone may be useful in adult women with 11-OH hydroxylase deficiency
- Oral isoretinoin severe, recalcitrant, or nodular acne

Rosacea

- Unrelated to acne but may co-exist on face
- 30-50 years of age
- Females predominate
- Rhinophyma principally occurs in males (late stage)
- Rare in darkly pigmented skin
- History of episodic facial flushing in response to hot liquids, spicy foods, or alcohol
- Early lesions are small red papules or pustules
- Late lesions are telangiectatic
- Nodular (sebaceous hyperplasia and lymphedema)

Rosacea

- Symmetrical location on face is characteristic
- Rhinophyma
- Glandular, fibrous, fibroangiomatous types
- “Red eyes”
- Corneal keratitis as serious complication
- There are high cutaneous levels of cathelicidin
- The cathelicidin peptides present are qualitatively distinct from those seen in individuals without rosacea
- Alternative processing by kallikrein 5.
- Kallikrein 5 production up-regulated by toll like receptors

Rosacea

- Rosacea treated with topical metronidazole
- Minocycline added if progresses
- Oral isoretinoin in severe disease

DISORDERS OF HAIR FOLLICLES

Non-scarring alopecia

- Alopecia areata
- Localized area of hair loss in round or oval areas without visible evidence of inflammation
- Principally on scalp
- Distinguish from male pattern baldness
- Alopecia totalis
- Loss of all scalp and eyebrow hair
- Alopecia universalis
- Loss of all hair
- Occurs in those <25 years-old
- May see male predominance

Non-scarring alopecia

- If occurring after puberty, 80% regrow hair
- 33%/year after first episode
- Recurrences frequent
- Dystrophic nail changes may also be seen
- Dorsal nail plate has hundreds of small depressions ("hammered brass" appearance)
- Poor prognostic sign
- Etiology is not known
- Associated with auto-immune disorders
- Hair loss resumes after corticosteroid or cyclosporine therapy
- 30% respond to oral PUVA (photochemotherapy)

Non-scarring alopecia

- Telogen effluvium
- Transient increase shedding of club hairs from resting scalp hairs
- Shift in hair cycle from anagen (normally 80-90% of hairs) to catagen to telogen
- Hair thinning
- Second most common cause of hair loss after male pattern (androgenetic) alopecia
- Follows parturition, cessation of oral contraceptives, “crash” dieting
- Lithium
- Regrows

Non-scarring alopecia

- Anagen effluvium
- Growth arrest
- Diffuse growth scalp hair thinning
- Drug induced
- Colchicine
- Chemotherapy agents
- Thallium

Scarring alopecia

- Infectious folliculitis
- Tinea capitis
- Lichen sclerosis
- Burns
- Discoid lupus erythematosus
- Dermatomyositis

Hair growth

- Hirsutism
- Excessive hair growth secondary to increased androgenic activity
- Hypertrichosis Lanuginosa
- Vellus and terminal hairs do not replace white, blond fetal pelage
- Fetal pelage grows excessively
- Does not involve palms or soles
- Phenytoin common cause of acquired hypertrichosis
- Harbinger of malignancy