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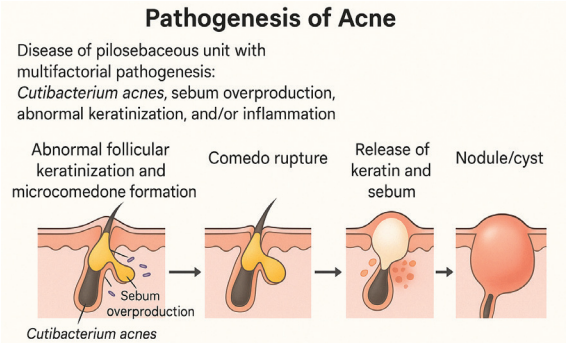
All about acne

By Benjamin Cooper, DO, Taha Rasul, MD, and Anthony Concilla, DO

Benjamin Cooper, DO, is a PGY-3 at HCA Florida Orange Park Hospital, in Orange Park, Florida.

Taha Rasul, MD, is a PGY-3 at HCA Florida Orange Park Hospital, in Orange Park, Florida.

Anthony Concilla, DO, is a PGY-4 at HCA Florida Orange Park Hospital, in Orange Park, Florida.

Acne vulgaris	
Epidemiology	Primarily in adolescence, affecting 85% of individuals between the ages of 11-30
Pathogenesis	Disease of pilosebaceous unit with multifactorial pathogenesis: 1. Follicular plugging 2. *Cutibacterium acnes* 3. Sebum overproduction 4. Inflammation 
Hormonal influence	Androgens (especially dihydrotestosterone [DHT] and testosterone) → ↑ growth of sebaceous glands/↑ sebum production. Androgen receptors found on the basal layer of sebaceous glands and outer root sheath of hair follicle
Cutibacterium acnes	*Cutibacterium acnes*: Gram-positive anaerobic rod which produce lipases that break down triglycerides in sebum into free fatty acids (FFAs), which are comedogenic and proinflammatory. *C. acnes* activates TLR-2 on macrophages and NLRP3 on neutrophils/monocytes, contributing to inflammation
Inflammation	Inflammation: neutrophil predominant → pustule; lymphocytes + foreign body-type giant cells + neutrophils → nodules/papules/cysts
Dietary factors	Factors that may contribute include skim milk, whey protein, vitamin B12, and high glycemic load
Clinical features	Most common sites (sebaceous areas): face, neck, behind ears, upper trunk, and upper arms. Frequently start as open and closed comedones. May develop more inflammatory lesions including papules, pustules, nodules, and cysts; nodulocystic lesions can coalesce into plaques and sinus tracts. As lesions resolve may leave post-inflammatory hyperpigmentation/erythema or scars (icepick, rolling, boxcar, anetoderma-like, hypertrophic, and keloidal)
Histopathology	Follicle filled with laminated keratin and debris with or without suppurative inflammation

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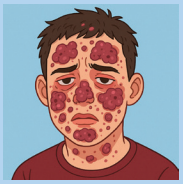
Management of acne vulgaris

Mild	Moderate to severe
Topical treatments: Multimodal therapy combining multiple mechanisms of action is recommended	**Systemic antibiotics:** Limit systemic antibiotic use to reduce resistance and side effects. Use with concomitant BP and other topical treatments
• Benzoyl peroxide (BP) or topical retinoid or topical antibiotic (monotherapy not recommended) • Combination of BP + topical retinoid • Combination of BP + topical antibiotic (concomitant use of BP can prevent antibiotic resistance) • Combination of topical retinoid and topical antibiotic	• Doxycycline (1st line) • Minocycline • Sarecycline **Hormonal agents** • Combined oral contraceptive pills (often ethinyl estradiol combined with either norethindrone, norgestimate, or drospirenone) • Spironolactone (potassium monitoring not needed in patients without risk factors (e.g., age >45, medical comorbidities, medications)) • Intralesional corticosteroids (adjuvant treatment for large papules or nodules at risk of scarring or for rapid improvement in pain) **Isotretinoin** • Patients with nodulocystic or scarring acne are considered candidates for isotretinoin. Though controversial, evidence suggests isotretinoin is safe for patients with psychosocial comorbidities

Acne variants

Condition	Epidemiology	Pathogenesis	Clinical features	Treatment/ clinical course	Additional information
Acne fulminans 	Males > females aged 13-22 years; individuals on systemic retinoid therapy	Most commonly idiopathic; isotretinoin therapy or dose increases are frequent triggers	Acute suppurative nodules, plaques, friable lesions, often with hemorrhagic crust and ulcers. Severe cystic acne with systemic manifestations (fever, leukocytosis, sterile osteolytic bone lesions)	Oral corticosteroids followed by isotretinoin after inflammation subsides	Polymorphisms in TLR-4 may be protective

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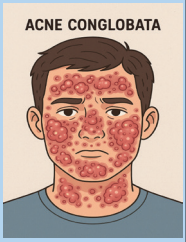
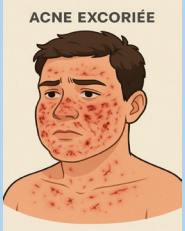
Condition	**Epidemiology**	**Pathogenesis**	**Clinical features**	**Treatment/ clinical course**	**Additional information**
Acne conglobata 	Males > females, young adults	Severe, eruptive nodulocystic acne, without systemic involvement	Large cysts, nodules, abscesses with sinus formation and thick, yellow fluid	Isotretinoin, possible prophylactic prednisone (0.5-1 mg/kg/day for 4-6 weeks) in patients at higher risk of acne fulminans	Component of follicular occlusion tetrad (acne conglobata, hidradenitis suppurativa, dissecting cellulitis of the scalp, pilonidal cysts)
Solid facial edema in acne 	Edema in individuals with history of acne	Swelling in midline face and cheeks with woody non-pitting induration, acne predates edema by 2-5 years	Edema in the face and cheeks, peau d'orange appearance	Isotretinoin, with or without ketotifen or prednisone	Clinical overlap with rosacea lymphedema, also known as Morbihan disease
Acne mechanica 	Adolescents and young adults; athletes, military recruits, and workers	Pressure and friction lead to blockage and inflammation of pilosebaceous units	Lesions in areas with repeated pressure or friction	Treatment of underlying cause of friction or pressure	
Acne excoriée (des jeunes filles) 	Young women, often associated with mental health disorders	Self-mutilation of acneiform lesions, often in young women with mental health conditions	Excoriated, crusted erosions due to self-mutilation of acne lesions	Psychotherapy, antidepressants, behavioral modification	

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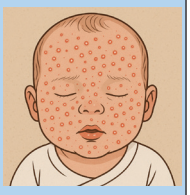
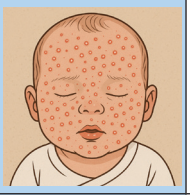
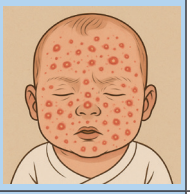
Condition	**Epidemiology**	**Pathogenesis**	**Clinical features**	**Treatment/ clinical course**	**Additional information**
Neonatal acne 	Occurs within first few weeks, resolves by 3 months, affects 20% of newborns	Associated with sebaceous gland hyperplasia driven by transiently elevated neonatal/maternal androgens	Characterized by the presence of comedones, papules, and pustules, most commonly on the cheeks	Usually resolves on its own; if mild, can use gentle soap and water. For more severe cases can use imidazole, topical retinoid, topical antibiotic, or benzoyl peroxide	The presence of comedones distinguishes this condition from neonatal cephalic pustulosis
Neonatal cephalic pustulosis 	Occurs within first few weeks, resolves by 3 months, affects 20% of newborns	Represents an inflammatory reaction to *Malassezia*. KOH may demonstrate *Malassezia* yeast	Papules and pustules, lacks comedones, cheeks and nasal bridge common sites	Usually resolves on its own, if moderate-severe can use topical ketonazole	
Infantile acne 	Appears around 3-6 months, usually resolves by 2-3 years	Hormonal imbalance, particularly hyperandrogenism	Comedones, cysts, and inflammatory lesions, potential for scarring	Topical retinoids, antibiotics, azithromycin, or isotretinoin for more severe cases	May need endocrinology evaluation for pubertal signs
Acne in the setting of endocrinologic abnormality 	Hyperandrogenism from PCOS, HAIR-AN syndrome, CAH, and others	Hyperandrogenism from underlying condition drives sebum overproduction and inflammation	Hirsutism, irregular menstrual cycles, and deep voice in females	Treat underlying endocrinologic abnormality, oral contraceptives, spironolactone	Consider lab workup for hyperandrogenism markers
Acne cosmetica 	Individuals using cosmetics containing comedogenic ingredients	Use of certain cosmetics causes blockage of follicles	Small, closed comedones, papules, pustules	Avoid offending cosmetics; use non-comedogenic products	Discontinue use of irritating products

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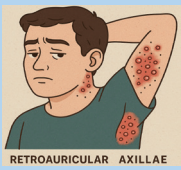
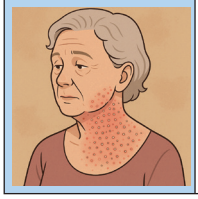
Pomade acne 	Individuals using oily substances on the scalp	Heavy use of oily products on scalp leads to comedones and pustules	Closed comedones on the forehead and temples	Discontinue use of greasy/oily products; treatment with topical treatments	Cosmetic changes or discontinuation of offending products can improve skin
Chloracne 	Workers in chemical, pesticide, or electronics industries; environmental disasters; poison victims; exposure to chlorinated aromatic hydrocarbons (e.g., Agent Orange)	Exposure to toxic chlorinated hydrocarbons causes inflammation in the sebaceous units	Recurrent outbreaks of acne-like lesions on areas exposed to chemicals	High-dose isotretinoin followed by low-dose maintenance, topical retinoids	Exposure to environmental toxins can cause persistent outbreaks
Radiation acne 	Caused by ionizing radiation exposure	Ionizing rays cause epithelial metaplasia and follicular blockage	Comedone-like papules in areas of previous radiation exposure	Topical retinoids, oral isotretinoin, or management of radiation dermatitis	Risk of delayed healing from ionizing radiation

Acneiform eruptions and acne-associated syndromes				
Condition	**Epidemiology/ pathogenesis**	**Clinical features**	**Treatment/ clinical course**	**Additional information**
Acneiform eruptions (drug-induced)	Associated with multiple medications: - Anabolic steroids, androgens (testosterone), corticosteroids, halogens (iodides, bromides), INH, OCPs, lithium, phenytoin, upadacitinib, EGFR inhibitors, chemotherapy (e.g., mTOR inhibitors, multikinase inhibitors) **Mnemonic: STOP HIVE** (**S**unitinib/**S**orafenib/ **S**irolimus, **T**estosterone, **O**CPs, **P**henytoin, **H**alogens, **I**soniazid, **V**itamins B2, B6, B12, **E**GFR inhibitors	Abrupt onset of monomorphous inflammatory papules/pustules (common in corticosteroid-induced acne), usually lacks comedones - Commonly on trunk and face - EGFR inhibitors cause eruption on acne-prone and sun-exposed areas, with severity correlating with clinical response to the drug	Discontinue the offending medication if possible - For EGFR inhibitors: doxycycline or minocycline prophylaxis - Avoid irritating agents like benzoyl peroxide - Topical retinoids are an effective treatment	EGFR inhibitors can cause severe acne-like eruptions, and severity positively correlates with the medication's clinical effectiveness

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Condition	**Epidemiology/ pathogenesis**	**Clinical features**	**Treatment/ clinical course**	**Additional information**
SAPHO syndrome	Affects children and young adults, especially in the third decade. More common in Japan - Inflammatory disorder of unclear etiology - Characterized by RF (–) osteoarthropathy and various skin manifestations	Characterized by **S**ynovitis, **A**cne, **P**ustulosis, **H**yperostosis, **O**steitis - Acne varies from mild to acne conglobata/ fulminans - May also present with hidradenitis suppurativa and dissecting cellulitis - Associated with IBD, and often involves the sternoclavicular area and chest wall	Treatment includes bisphosphonates, TNF- α inhibitors, methotrexate (MTX), NSAIDs, corticosteroids, colchicine, and anakinra	Characterized by a distinctive 'bull's head' sign on bone scintigraphy and more severe acne manifestations
PAPA syndrome	Autosomal dominant mutation in CD2-binding protein 1 (encoded by gene *CD2BP1* aka *PSTPIP1*)	Sterile **P**yogenic **A**rthritis, **P**yoderma Gangrenosum, and **A**cne Conglobata - A part of an autoinflammatory disease group with unopposed inflammation due to a mutation in CD2BP1	Treatment includes systemic or local corticosteroids, dapsone, infliximab, anakinra	Considered an autoinflammatory disease; CD2BP1 interacts with pyrin
HAIR-AN syndrome	A unique subtype of PCOS	**H**yperandrogenism, **I**nsulin **R**esistance, **A**canthosis **N**igricans - High risk for diabetes and cardiovascular disease. - Seen with signs of hormonal imbalance	Anti-androgens (e.g., spironolactone), OCPs, and insulin-sensitizing medications (e.g., metformin)	Higher propensity for metabolic complications like diabetes and cardiovascular disease
Apert syndrome (acrocephalosyndactyly)	Activating autosomal dominant mutation in fibroblast growth factor receptor 2 (FGFR2) → follicular hyperkeratosis and sebaceous gland hypertrophy	Acne distributed moderately to severely on the extensor arms, buttocks, and thighs - Associated with synostoses of bones of hands/feet, vertebral bodies, and cranium	Isotretinoin is the treatment of choice	Can be associated with some cases of nevus comedonicus

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2. Bolognia, Jean, et al. *Dermatology*. 5th Ed. Elsevier Health Sciences, 2024.
3. Guidelines of care for the management of acne vulgaris. Reynolds, Rachel V. et al. *J Am Acad Dermatol*. 90(5): 1006.e1-e30.