Acne & Rosacea

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Disclosures

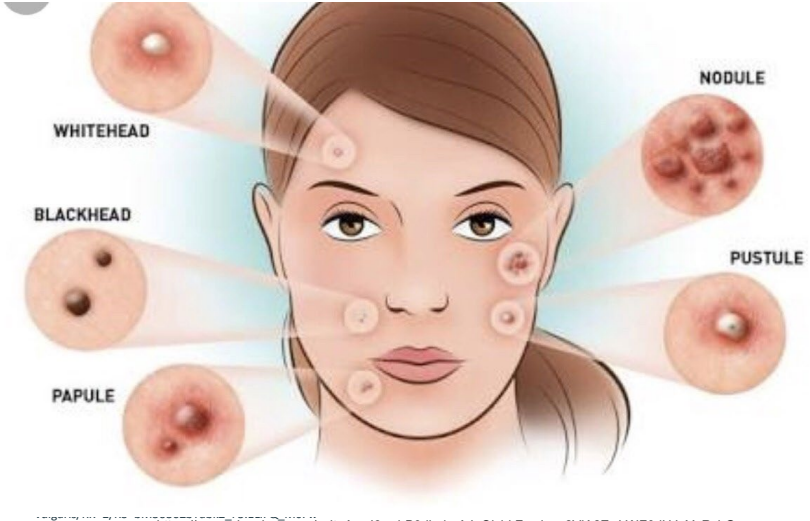
I do receive financial compensation from Journey Medical Corporation for speaking at dinner programs regarding the dermatology drugs they offer.

Learning Objectives

- Identify the pathophysiology and epidemiology of acne vulgaris and rosacea
- Understand the different subtypes of acne vulgaris and rosacea
- Understand and list exacerbating factors for these diseases
- Build a list of differential diagnoses for acne vulgaris and rosacea

What is Acne Vulgaris?

- Chronic inflammatory disease of the pilosebaceous unit
- Lesions and sequelae
  - Comedones (open and closed)
  - Papules & pustules
  - Nodules
  - Scarring
  - Post-inflammatory hyperpigmentation/erythema
- Commonly found on cheeks, nose, forehead, chin, ears, neck, upper trunk, upper arms, chest



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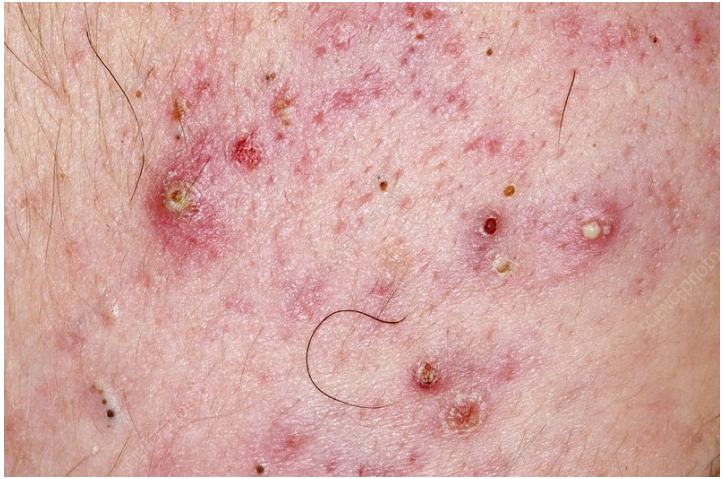
Epidemiology

- Neonatal and Infantile subtypes
- Considered physiologic in patients 12-24 yoa
  - Affects 85% of this population
  - Generally develops at adrenarche (8-12 yoa) with onset of comedones
- 33% women and 3% men will continue to have acne until their mid 40s



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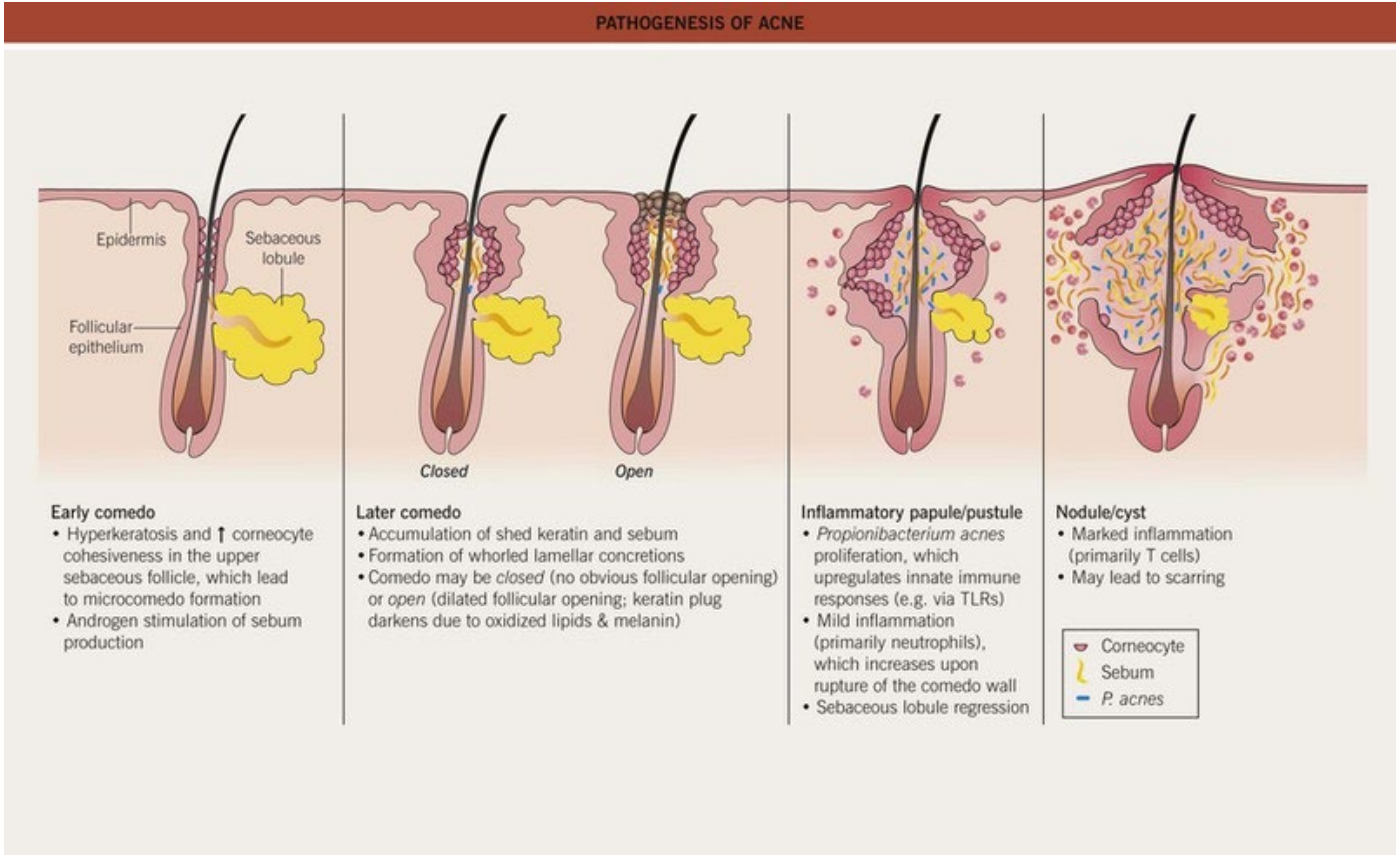
Pathogenesis

- Increased circulating levels of DHEAS leads to increased levels of DHT + testosterone → increased sebum production → comedo formation
- Impaction and distension of follicles with keratin plug develops due to hyperproliferation and abnormal differentiation of keratinocytes
- Sebum becomes entrapped by keratin plug → dilation and rupture of follicular epithelium
- Increased proliferation of *Propionibacterium* acnes and inflammation



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Differential Diagnoses of Acne Vulgaris

Comedonal Acne Vulgaris

- Closed
  - Milia
  - Osteoma Cutis
  - Sebaceous Hyperplasia
  - Trichoepithelioma (small/early)
  - Eruptive vellus hair cysts (trunk)
  - Steatocystoma multiplex (trunk)
  - Contact acne
  - Acne due to systemic steroids
- Open
  - Contact acne
  - Acne due to systemic steroids
  - Trichostasis spinulosa
  - Dilated pore of Winer
  - Favre-Racouchot
  - Nevus comedonicus

Inflammatory Acne Vulgaris

- Rosacea
- Perioral dermatitis
- Lupus miliaris disseminatus faciei
- Acne due to topical/systemic steroids
- Staph folliculitis
- Gram negative folliculitis
- Eosinophilic folliculitis
- Pseudofolliculitis barbae
- Acne keloidalis nuchae
- Furuncle/carbuncle
- Keratosis pilaris
- Neurotic excoriations/factitial

Neonatal

- Sebaceous hyperplasia
- Miliaria rubra
- Milia
- Candidal infections

Neonatal Acne

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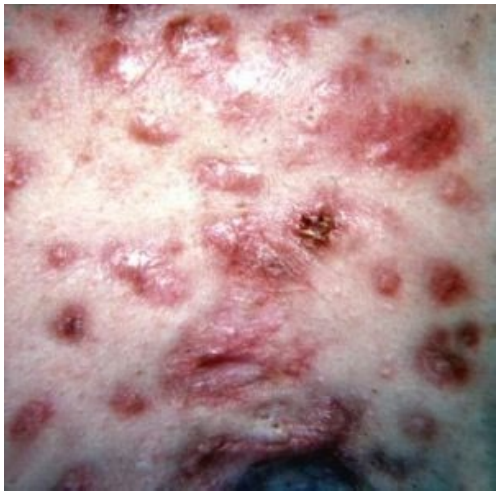
- Seen in 20% of healthy babies
- More common in males
- Spontaneously resolves within few weeks of birth
- Papules and pustules on cheeks, forehead, chin which sometimes extends to scalp
- No treatment necessary
- Does not scar

Infantile Acne

- Comedo formation present in addition to papules and pustules.
- Can lead to scarring
- Pathogenesis related to hormonal imbalances that occurs normally in this age
  - Boys → elevated LH and testosterone
  - Immature adrenal gland → elevated DHEA levels
- Generally resolves by 12 months of age, but can persist into puberty
- Important to treat in order to prevent scarring
- If other signs/symptoms of endocrine abnormalities or in severe cases, referral to endocrinologist may be warranted for work-up.

Acne Conglobata

- Severe nodulocystic acne, no systemic manifestations
  - Numerous comedones + large abscesses with interconnecting sinuses, grouped inflammatory nodules + pronounced scarring
  - Locations: back, buttocks, chest, forehead, cheeks, anterior neck, shoulders
- Associated with
  - Dissecting cellulitis of scalp
  - Hidradenitis suppurativa
  - Pilonidal cysts
- Population: young men
- Can be induced by anabolic steroids



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Acne Fulminans

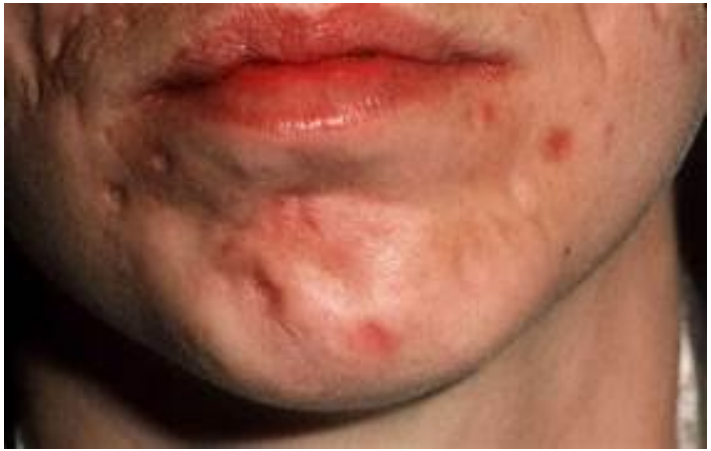
- Nodulocystic acne + systemic manifestations
- Abrupt onset of inflammatory nodules that coalesce into painful plaques with hemorrhagic crusting + various systemic symptoms
- Population: teenage boys
- Can be induced by anabolic steroids
- Systemic manifestations
  - Fever
  - Arthralgias/myalgias
  - Hepatosplenomegaly
  - Osteolytic bone lesions (clavicle, sternum, ankles, humerus, iliosacral joints)
- Lab Abnormalities
  - Elevated ESR
  - Proteinuria
  - Circulating immune complexes
  - Leukocytosis/anemia

Hormonal Acne

- Adult onset acne generally occurring in patients 20-50 yoa
- Estimated to affect up to 54% adult women
- Direct result of excess sebum in sebaceous glands due to androgens
- Population; primarily women
- Secondary to fluctuating hormones
  - Perimenopause or post menopause
  - Pregnancy
  - After start or discontinuation of OCP
  - Testosterone treatment in men
- Distribution on chin/jawline/upper neck

Other Subtypes of Acne

- Solid Facial Edema (Morbihan's Disease)
  - Distortion of midline face/cheeks due to soft tissue swelling
- Acne Mechanica
  - Secondary to repeated frictional obstruction of follicle
- Acne Excoriae
  - Young women who chronically pick lesions leaving crusted erosions that may scar
- Drug Induced
  - Abrupt onset monomorphous eruption of inflammatory papules
- Occupational Acne
  - Exposure to insoluble, follicle-occluding substances
  - Comedones predominate



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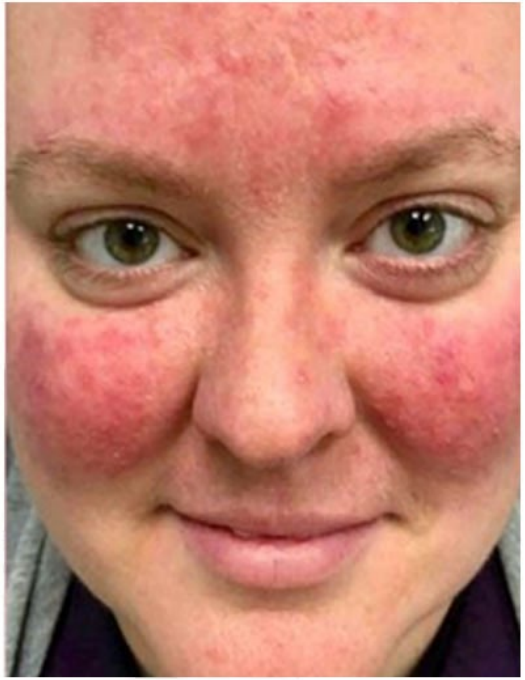
Endocrine Work-up in Acne

- Indicated in mid childhood acne, severe/ difficult to treat acne in females with other si/sx of hyperandrogenism
  - Hirsutism
  - Irregular menstrual periods
  - Precocious puberty
  - Coarsened voice
  - Muscular habitus
  - Androgenetic alopecia
  - Growth abnormality
  - Acanthosis nigricans
- Initial tests: serum total and free testosterone, DHEAS, LH/FSH ratio
- DHEAS
  - DHEAS > 8000 ng/mL → adrenal tumor
  - DHEAS 4000-8000 ng/mL or 17-hydroxyprogesterone > 3 ng/mL → CAH
  - Testosterone 150-200 ng/dL → PCOS
  - Testosterone > 200 ng/dL → ovarian tumor
- OCPs can mask endocrine disorder, so patients should discontinue 1 month prior to testing

Rosacea Epidemiology

- Most common in fair-skinned patients, though it can be seen with any skin type
- More common in women 30-50 yoa
- Men more likely to develop phymatous changes



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Rosacea Pathogenesis

- Multifactorial
- Associated with vascular hyper-reactivity to thermal and other stimuli
- Unclear why vasodilation promotes rosacea, but hypothesized that plasma extravasated by blush induces inflammation which worsens with repeated vasodilation
- Possible dysregulation of innate immune system and neurovascular control
- Demodex folliculorum mite may play role

- Triggers
  - Spicy Foods
  - Alcohol
  - Medications
  - Environment → heat/cold
  - Hot tea/hot coffee (not caffeine)
  - Topical steroids
  - Parkinson's disease (due to alteration of facial vasoreactivity)
  - Autoimmune CTD

Rosacea Subtypes

Erythematotelangiectatic (EET)

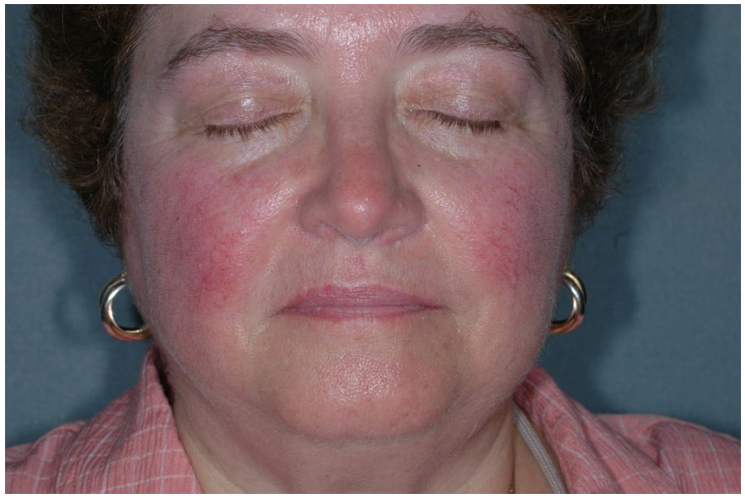
Papulopustular

Phymatous

Ocular

Erythematotelangiectatic Rosacea

- Recurrent and prolonged blush (>10 minutes) which can be associated with stinging/burning
- Telangiectasias
- Skin often finely textured/rough
- Skin sensitive



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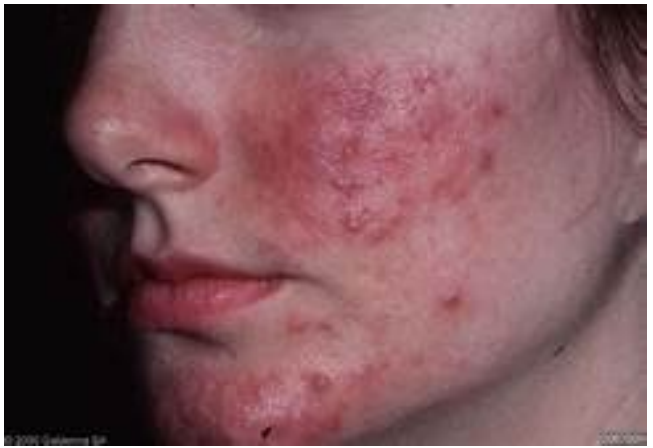
Papulopustular Rosacea

- Inflammatory papules and pustules with lack of comedones
- Face often centrally red
- Clinically, can appear similar to acne vulgaris
- Can overlap with acne vulgaris (mixed picture)



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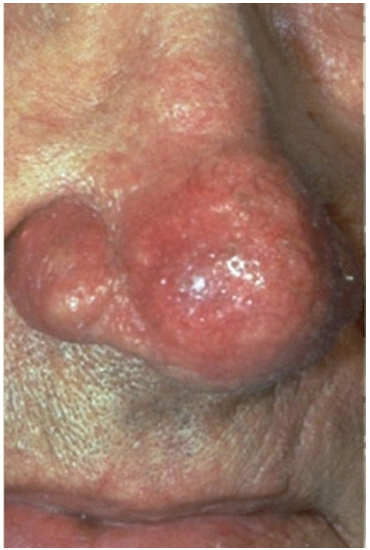
Phymatous Rosacea

- Overgrowth of sebaceous glands causing the skin to appear thick and bumpy
- Pores appear enlarged
- Skin oily
- Rhinophyma → sebaceous hyperplasia on nose
- Often history of teenage acne
- Seen more commonly in men



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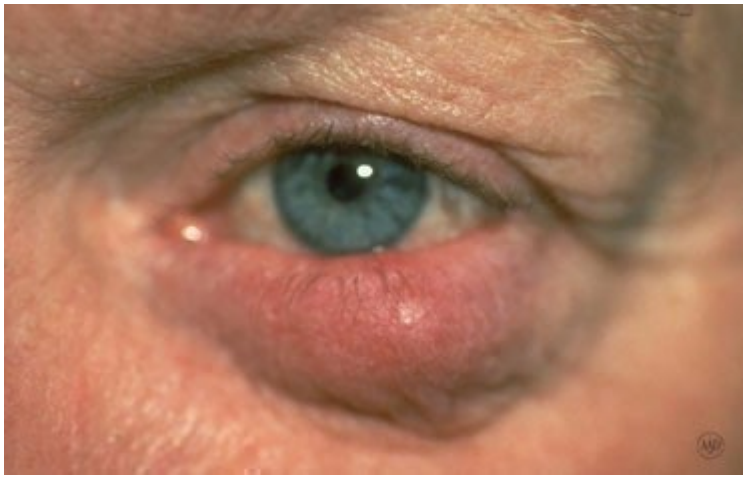
Ocular Rosacea

- Seen in up to 50% patients with rosacea
- Symptoms
  - Foreign body or gritty sensation or pain
  - Dryness
  - Tearing
  - Blurred vision and light sensitivity
  - Recurrent styes, chalazion
- May present prior to skin findings



https://www.aad.org/public/diseases/rosacea/what-is/symptoms

Rosacea Differential Diagnoses

- Persistent erythema of central face
  - Polycythemia vera
  - Carcinoid
  - Mastocytosis
  - Systemic Lupus erythematosus
- Haber syndrome (genodermatosis)
  - Facial lesions develop within first 2 decades of life
  - In addition to rosacea-like face lesions, will see verrucous lesions on skin protected from
- HIV/AIDS
  - Papulonodular eruption on face
  - Numerous Demodex mites seen under microscope
- Seborrheic dermatitis
- Acne vulgaris
  - Younger patient
  - Comedones present
- Basal cell carcinoma

Learning Pearls

- Examine patient's skin for evidence of more severe breakouts in the past which may indicate need for more aggressive therapy (ie scarring, PIE/PIH).
- Account for patient's age, race, sex, lesion distribution and severity when determining whether or not investigative work-up for other comorbidities is warranted.
- Remember that there can be overlap between acne vulgaris and rosacea presentation.
- Always discuss triggers and exacerbating factors with patient which can be a way to both help diagnose and treat acne and rosacea.

References

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