Rosacea and Acneiform eruptions

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• I have no financial conflicts of interest
• I have no financial affiliation with the companies that manufacture medications or products presented in this presentation.
Objectives

- Review the spectrum of acneiform eruptions
- Identify the major forms of rosacea
- Become familiar with common treatments for rosacea
Acneiform eruptions

- Skin eruptions that resemble acne vulgaris.
- Most commonly:

  Acne vulgaris / Rosacea / Seborrheic Dermatitis

  Drug reactions

  Infections

  Genetic or other metabolic disorders
Acne vulgaris

• Acne vulgaris

• Characterized by:
  – Comedones (black heads)
  – Inflammatory papules (often in various stages of healing)
  – Pustules
PATHOGENESIS OF ACNE

A. Early comedone
   - Infundibulum
     - hyperkeratosis
     - corneocyte cohesiveness
     - Androgen stimulation of sebum secretion

B. Later comedone
   - Accumulation of shed keratin and sebum
   - Formation of whorled lamellar concretions

C. Inflammatory papule/pustule
   - Propionibacterium acnes proliferation
   - Sebaceous lobule regression
   - Mild inflammation

D. Nodule/cyst
   - Marked inflammation
   - Scarring

Rosacea

- Rosacea

- Characterized by:
  - History of facial flushing
  - Inflammatory papules (often monomorphic)
  - Fibrous changes of the nose
  - Perioral or periorificial involvement
Seborrheic dermatitis

• Seborrheic dermatitis

• Characterized by:
  – Patchy erythema, often with thicker greasy scale
  – Occasional erythematous papules
  – Distributed in hair bearing skin of head, central face, and upper torso
Acneiform drug reactions

- Drug reactions

- Characterized by:
  - Sudden onset
  - Monomorphic lesions
  - Improvement/resolution after discontinuation of inciting drug
  - Corroborating history
Acneiform drug reactions

- Acute generalized exanthematous pustulosis
- Steroids
- Kinase inhibitors
- Halogenated hydrocarbons
Acute Generalized Exanthematous Pustulosis

- Often pustular eruption soon after initiation of drug
- Typically in the folds of the torso
- Accompanied by fever and neutrophilia
- Sometimes there is accompanying hepatitis
Steroid acne

• Initially steroid acne is noncomedonal.

• High dose steroids over a few weeks

• Can be complicated by exacerbation of acne vulgaris
Kinase inhibitors

- Imatinib

- Epidermal Growth Factor Receptor Inhibitors
  - Cetuximab
  - Erlotinib
  - Gefitinib

- Small molecular weight kinase inhibitors
  - Sorafenib and vemurafinib
Halogenated hydrocarbons

• Acne-like eruption after exposure to halogenated drugs or compounds

• Iodinated contrast dyes, $I^{131}$ treatment, potassium iodide treatment

• Fluoride dental gels

• Hydrocarbons alone also cause acneiform eruptions
  – Cutting oils
  – Crude petroleum
Infectious acneiform eruptions

- Pityrosporum folliculitis
- Demodex mites
- Gram-negative folliculitis
- HIV/AIDS (eosinophilic folliculitis)
- Secondary syphilis
- Deep fungal infection (Majocchi’s granuloma)
• Rosacea
• Rosacea

• Chronic inflammatory condition of the face of as yet unknown origin

• Characteristics:
  • Transient, later permanent, facial flushing
  • Thick and irregular skin of the face and nose may occur
  • Sometimes plaque-like, edematous changes may occur
Variants of Rosacea

• Erythematotelangiectatic
  – Facial flushing with/without elangiectatic mats

• Papulopustular
  – Erythematous papules, generally central face
  – Can become chronic plaques of edema

• Phymatous
  – Thickened and irregular nodularity of the skin

• Ocular
  – Dry eyes, foreign body sensation, lid/periocular edema
Pathophysiology

- Possibly genetic? Celtic and Northern European predominance

- Dysregulation of the innate immune system in skin
  - Increased cathelicidin expression
  - Increased Toll-like receptor 2 expression
  - Matrix metalloproteinase expression increased
  - Increased kallikrein 5 expression

- Local colonization may trigger the inflammatory pathways.
  - *Demodex* mites
  - *Staphylococcus epidermidis*
  - *Helicobacter pylori* ??
• Pathophysiology (cont’d)
  – UV radiation
    • Sun exposure triggers similar metalloproteinase activity and fibrosis
  – Neurogenic
    • Transient receptor potential (TRP) channels are more highly expressed.
  – Altered Skin Barrier Function?
    • Increased transepidermal water loss in rosacea
• Treatments

• Patient education
  – Identify potential modifiable triggers of rosacea

• Skin care
  – Moisturization and barrier protection of skin
  – Sunscreen use to block UV effects
• Treatments (cont’d)

• Topicals
  – Topical metronidazole 0.75 – 1% (lotion, cream, gel)
  – Topical sodium sulfacetamide
  – Azelaic acid
  – Permethrin
  – Ivermectin
  – Benzoyl peroxide
  – $\alpha$-adrenergic agonists
  – Calcineurin inhibitors
• Systemic treatments

• Tetracycline-class antibiotics
  – Anti-inflammatory effects

• Beta-blockers
  – Constriction of dermal blood vessels

• Isotretinoin
  – Decreased TLR2 expression?
Summary

• Acneiform eruptions
  – Include more than acne vulgaris, rosacea, and seborrheic dermatitis
  – Infectious etiologies
  – Exposure to hydrocarbons, especially halogenated compounds
  – Certain chemotherapeutics, especially kinase inhibitors
  – Genetic disorders
Rosacea

- Likely caused by immune dysregulation
  - Inflammation
  - Vascular vasodilation
  - Presence of commensal organisms may exacerbate

- Treatments
  - Identify triggers
  - Topical antibiotics
  - Tetracycline-class systemic antibiotics
  - Topical alpha-agonists
  - Isotretinoin
• Questions?

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