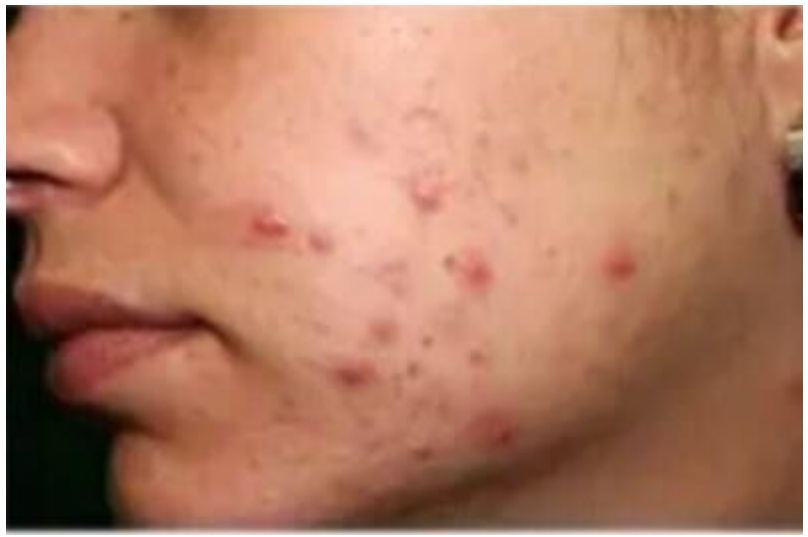
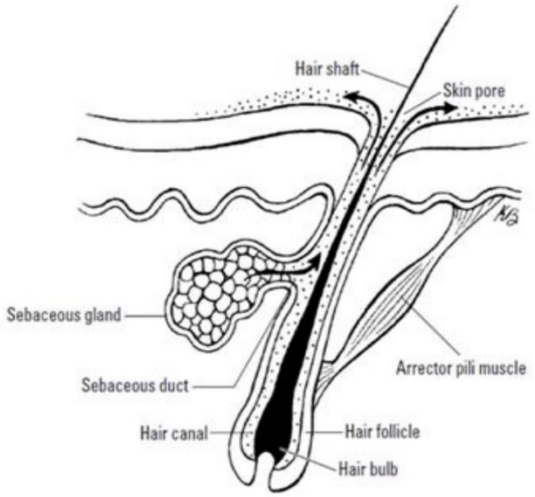
Acne Vulgaris



BY Dr.Sanaa Abd AL-Hussain 2021

Definition :

•Chronic inflammatory disorder of pilosebaceous apparatus characterized by formation of comedones, papules, pustules, cysts and scars .



Epidemiology :

- Prevalence: Nearly all teenagers have some acne (acne vulgaris) .
- Sex: Both sex are equally affected.
- Age:
    - * Start usually at age of 12 - 14 y, tending to be earlier in females .
    - * Peak age for severity in females is 16–17 & in males 17–19 years

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Etiopathogenesis:

Acne is a multifactorial disease involving 4 main pathologic events:

1. **Increased Sebum production:**

   Mainly due to androgenic stimulation at puberty due to end-organ hypersensitivity of sebaceous gland to normal level of plasma androgen.

2. Follicular hyperkeratinization ((Comedone)):

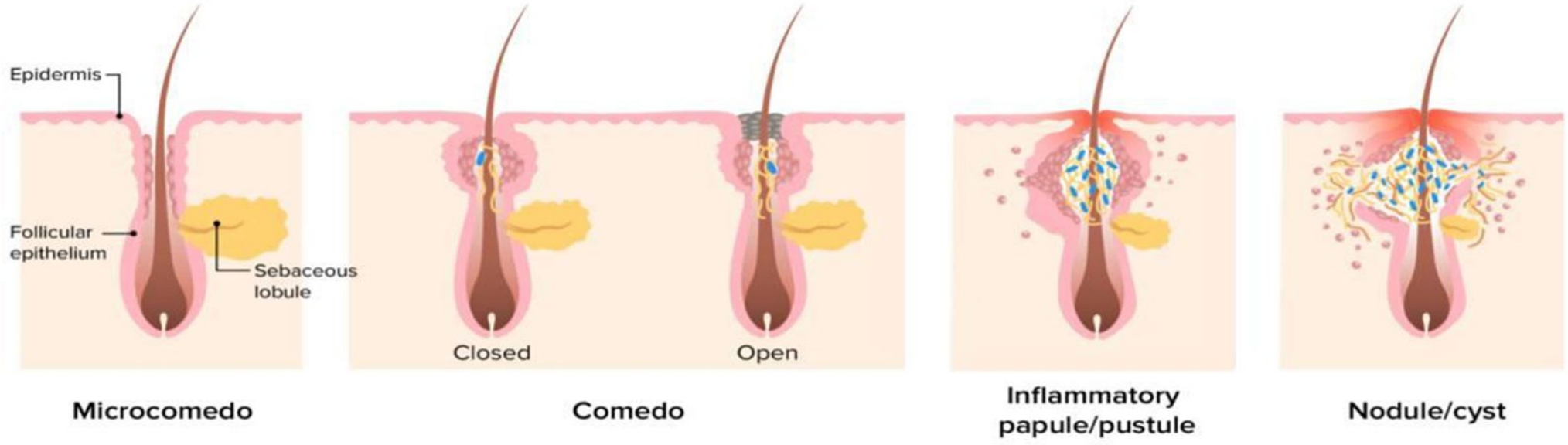
* Due to excessive accumulation of ductal corneocytes_ hyperkeratotic plugging in the upper follicle , causing retention of sebum and leading to a microcomedone

3. Proliferation of propionibacterium acnes ((P.acnes)):

* P.acnes is a normal skin commensal, plays a pathogenic part .
* It colonizes the pilosebaceous ducts:-
    1. Secrete lipases → break down TGs releasing free fatty acids → chemotactic for inflammatory cells and induces the ductal epithelium to secrete pro-inflammatory cytokines.
    2. Activate toll-like receptor 2.

4. Inflammation:

* Due to lipase enzyme from P.acnes that produce FFA → erosions & disruption of follicular wall → escape of follicular contents to dermis → Non- specific inflammatory reaction and increased production of cyotokines IL-8 and IL-1α.



Other factors in aetio-pathogenesis of acne:

1. Genetic: Family history is often positive.
2. Diet:
    * High glycemic diet may exacerbate acne.
    * Milk intake may affect comedogenesis.
3. Premenstrual flare.
4. UVR: may improve acne.
5. Stress: may exacerbate acne.
6. Sweating: may exacerbate acne

Clinical picture:

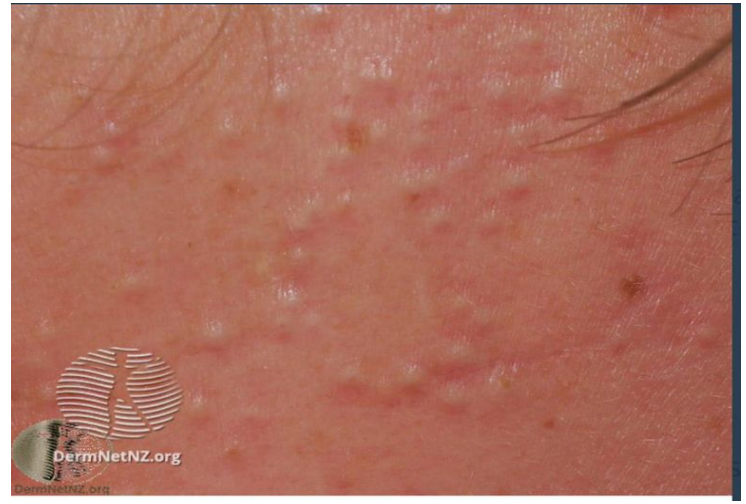
1. Comedones:

* These are the starting point of all acne.
* They begin as very small blockages in the pores called Microcomedone.
* Microcomedones either:
  - Progress to become larger comedones, or
  - Rupture internally → inflammatory lesions

Types of comedones:-

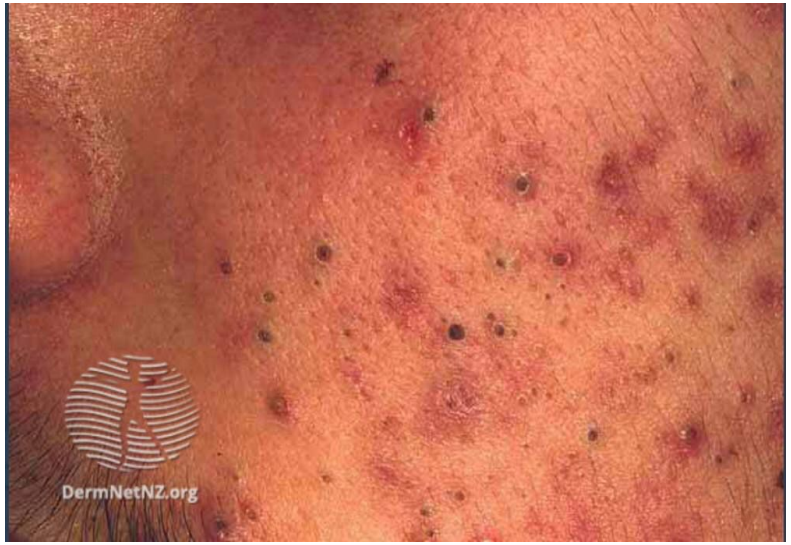
1. Closed comedone - Called white heads.

   - Tiny, white, dome-shaped papules with asmall follicular orifice .



2. Open comedone - Called Black heads.

   - Black color may be due to melanin or oxidized sebum.

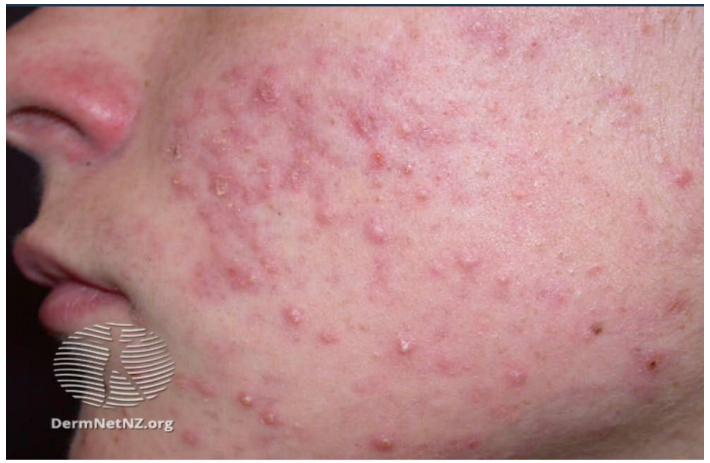


2. Inflammatory lesions:

* It is unusual for black heads to develop into inflamed lesions.

* 75% of white heads develop into inflamed lesions.

* Inflamed lesions include:



1. Papules and pustules:

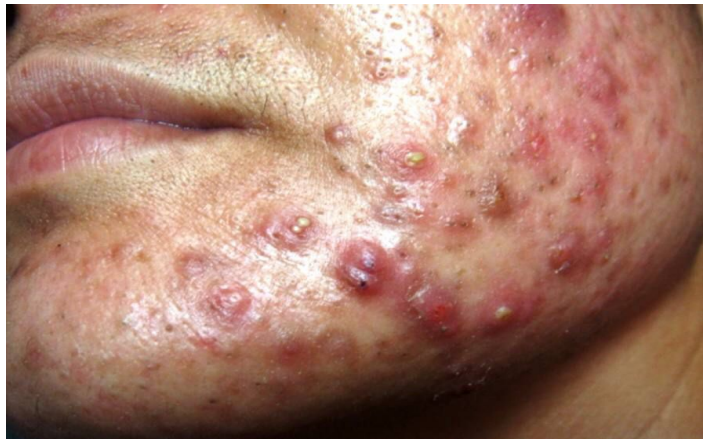
✓ Papules are less than 5mm with no pus visible .

✓ Cause pain associated with swelling and inflammation.

2. Nodules and cysts:

✓ Cysts are bags of liquid that is a mixture of pus and bacteria .

✓ Often associated with scarring



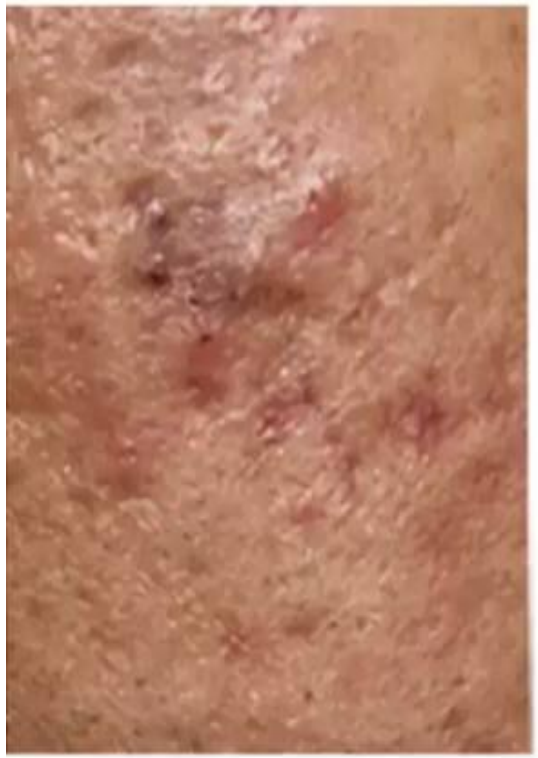
3. Post-acne Scars:

1. Increased tissue formation:

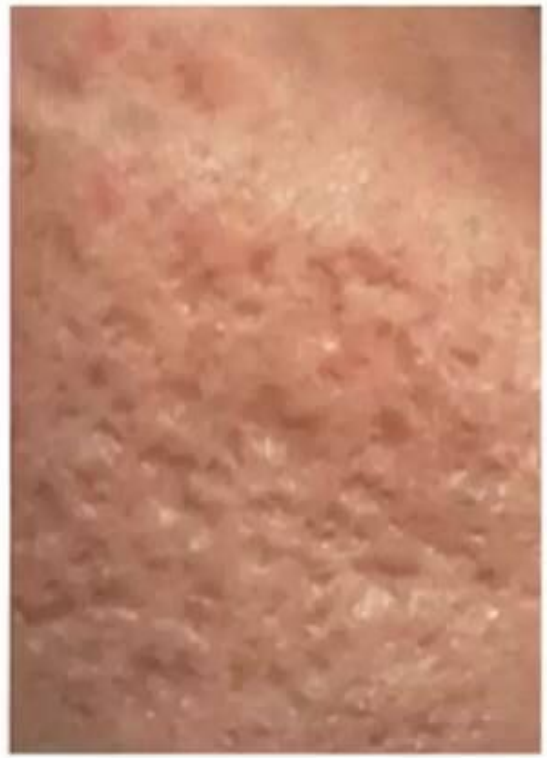
- ✓ Hypertrophic scar.
- ✓ Keloid.

2. Tissue damage (Atrophic):- The most common type .

- ✓ Ice pick scar.
- ✓ Soft rolling scar.
- ✓ Boxcar or depressed scar.

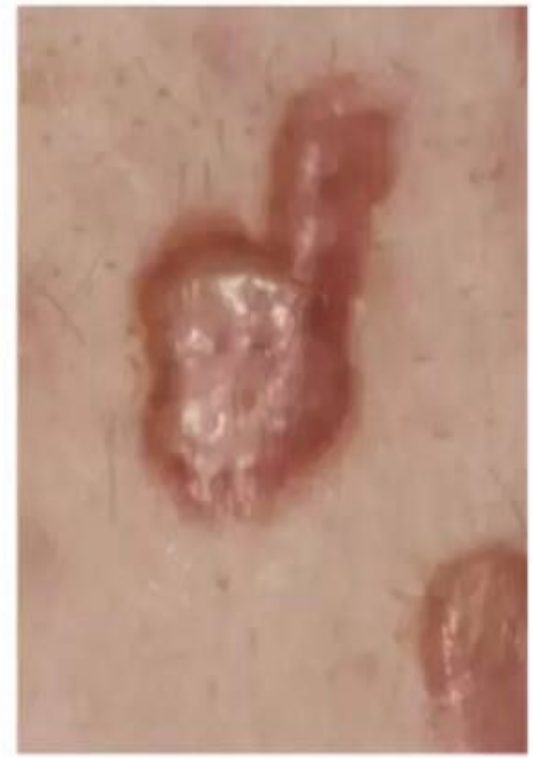


Rolling Scar



Boxcar Scar

Ice Pick Scar



Keloid (Hyperthropic)

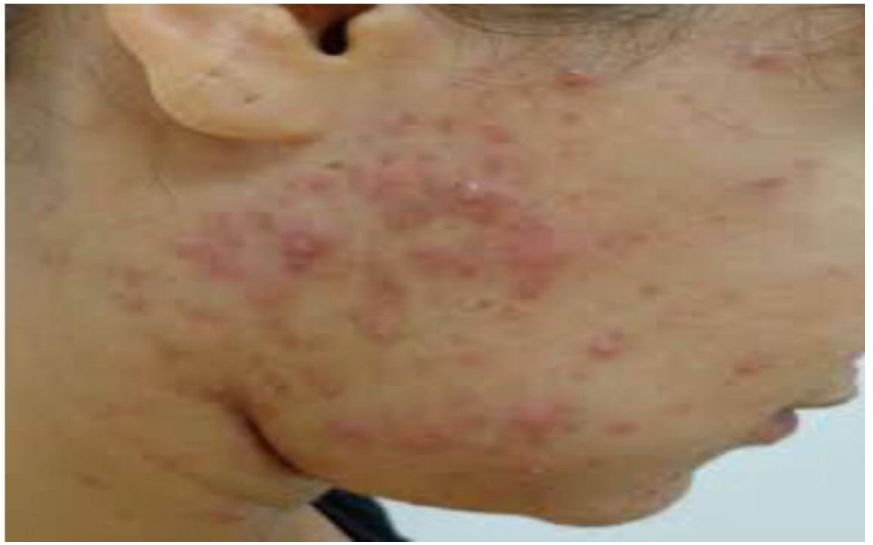
Treatment of acne:

1. Topical	2. Oral	3. Miscellaneous
A. Predominantly anti-comedonal: * Topical retinoids. * Adapalene. * Azelaic acid. **B. Predominantly anti-microbial:** * Topical antibiotic ✓ Erythromycin. ✓ Clindamycin. ✓ Tetracyclin. ✓ Erythromycin+zinc. * Azelaic acid. * Benzoyl peroxide (BP). * Benzamycin(BP+erythromycin) **C. Predominantly anti-inflammatory** *Adapalene. * Topical antibiotics.	1. Antibiotics. 2. Hormonal. 3. Antiandrogen. 4. Retinoids. 5. Others	1. UVR 2. Liquid nitrogen. 3. IL steroids. 4. LASERS.

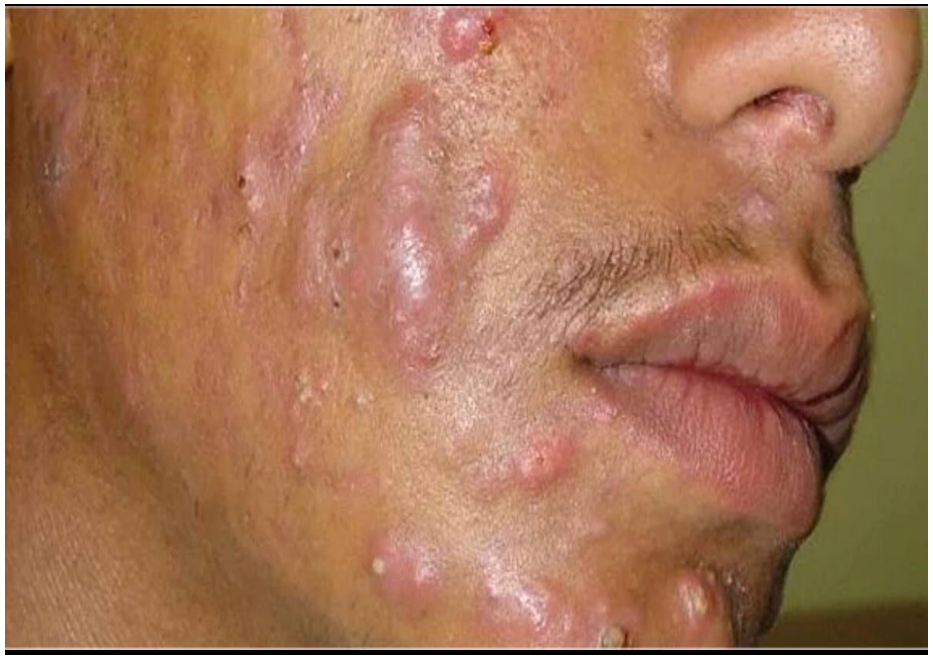
Variants of acne:

1. Post-adolescent acne:

- ✓ Also called “Late onset acne”.
- ✓ This occurs mainly in women.
- ✓ Limited to chin and margin of mandible (lower third of face).
- ✓ Nodular and cystic lesions predominate.

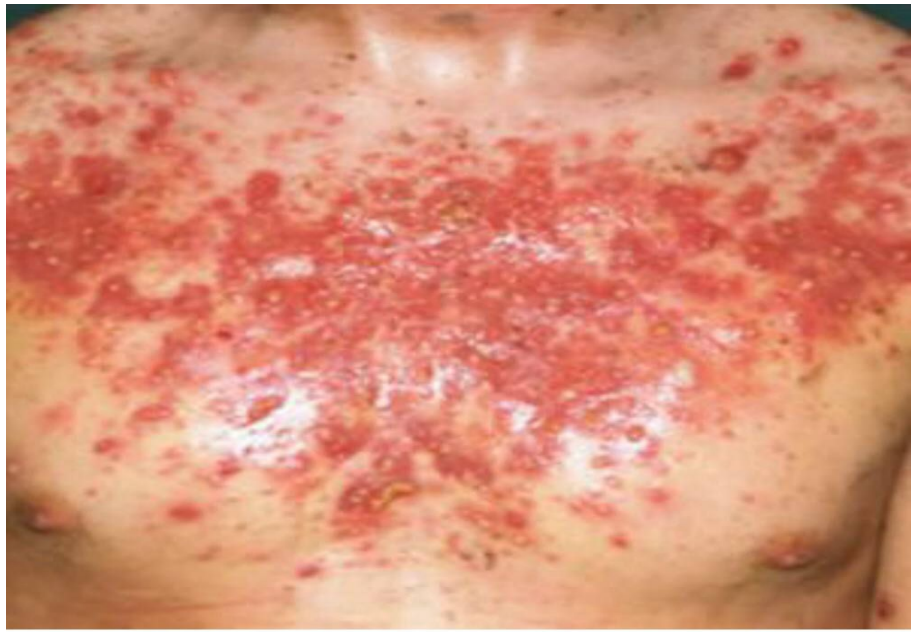


	2. Acne fulminans:	3. Acne conglobate:
Age	13-16 years	20-25 yrears
Sex	adolescent white boys	common in young males
Onset	Sudden	Slow
Site	Face – Neck- Chest – Back	The same
Lesion	*Hemorrhagic nodules & plaques → suppurative degeneration → leaving ragged ulceration filled with necrotic debris.	*Nodules & tender large cysts → may fuse to form multiple draining sinuses. *Grouped multiple fused blackheads.
Systemic manifestations	Very common Fever, Malaise, Polyarthralgia, ↑ ESR, Proteinuria.	Uncommon
Treatment	*Oral Isotretinoin (0.5 mg/kg) is continued for 5 months. *Oral corticosteroids (e.g., prednisolone 0.5 to 1.0 mg/kg) are the primary therapy for at least 2 months. *Antibiotic therapy is not effective.	*Oral Isotretinoin:- Start with low dosages of (0.5 mg/kg or lower) to avoid exacerbation. *Do not require oral steroids. *Cyst with thin roofs are incised and drained. *Deeper cysts are injected with triamcinolone acetonide (kenalog 2.5 to 10 mg\ml.



Acne conglobate

Acne Fulminans

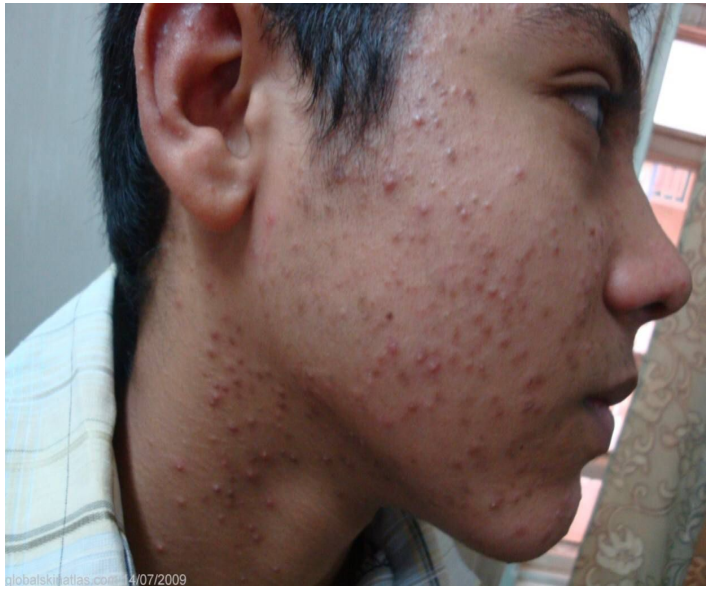


4. Acne medicamentosa:

- ✓ Monomorphous folliculitis→ small erythematous papules and pustules without comedones.
- ✓ Onset:- Appears suddenly and coincides with prescription of a drug known to cause acneiform lesions.

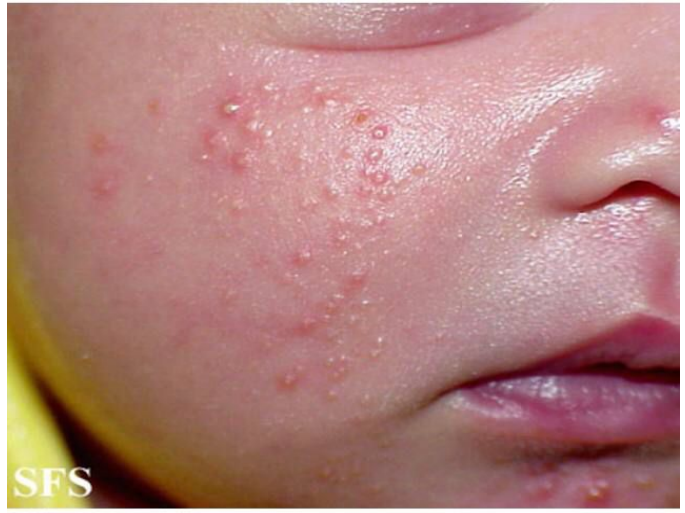
Drugs reported to cause acne-like eruption:-

1. Androgenic & anabolic steroids.
2. Gonadotrophins.
3. Oral contraceptives.
4. Antituberculosis.
5. Anticonvulsant therapy



5. Neonatal acne:

- ✓ Synonym:- Neonatal cephalic pustulosis.
- ✓ Age:- Affect new born between the 1st and 6th week of age.
- ✓ Sex:- more common in males.
- ✓ Cause:- due to passing of transplacental androgen.



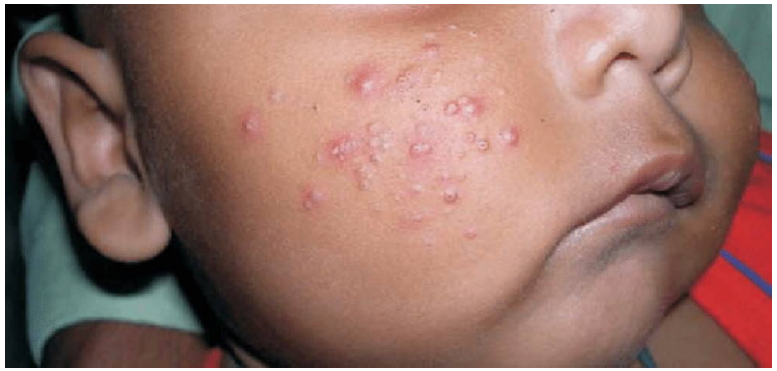
- ✓ Morphology:-
  - * Mainly inflammatory comedones on nose and cheeks.
  - *Not associated with increased incidence of acne in later life.

6.Infantile acne:

✓ Cause:- 1. Hormonal imbalance.

2.Trans-placental stimulation of sebaceous glands by maternal androgens (minor role).

✓ Age:-begin between 3rd – 6th month → persist to age of 3-5 years.



✓ Sex:-more common in males.

✓ Morphology:-

* As common acne on the cheecks.
* Associated with increased risk of severe acne in adolescence.

Rosacea (Acne-Rosacea)

Definition:

Chronic, vascular inflammatory disorder, usually limited to the centre of face and characterized by:

- ✓ Persistent erythema.
- ✓ Telangectasia.
- ✓ Papules & Pustules

Epidemiology:

* Age:- more common 30-50 years.
* Sex:- more common in females

Clinical features:

* Site: ✓ The convex areas of the nose, cheeks, chin, and forehead are the characteristic distribution .
✓ Rarely, it may affect arm, legs, chest, scalp, scrotum.

* Primary features of rosacea:
✓ Flushing.
✓ Non-transient erythema.
✓ Papules & pustules.
✓ Telangiectasia.

* Secondary features of rosacea:-
✓ Facial burning.
✓ Plaques.
✓ Dry appearance.
✓ Phymatous changes.
✓ Peripheral flushing.
✓ Ocular manifestations

Etiology:

* Exact etiology is unknown.

* Exacerbating factors:

1. Vascular reactivity:

Rosacea is induced by chronic repeated triggers of flushing exposure, eg.:-

1. Hot or cold temperature.
2. Sunlight.
3. Hot drinks.
4. Spicy foods.
5. Alcohol.
6. Emotional disteress.
7. Certain cosmetics.
8. Topical irritants & Medications

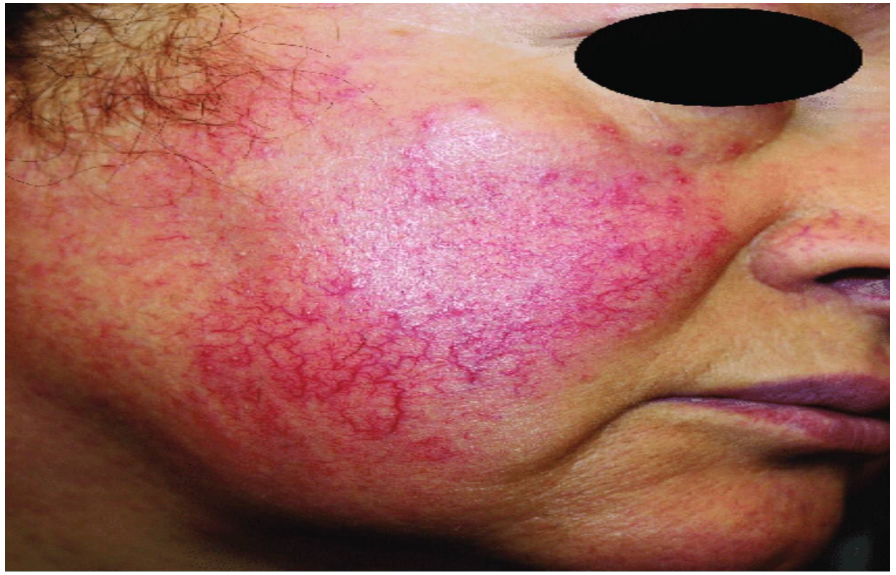
2. The role of microbe – induced follicle based inflammation:

1) Commensal organisms which reside in hair follicles and sebaceous glands may trigger folliculocentric inflammatory papules.

2) Demodex folliculorum (mite) or associated bacteria (bacillus oleronius).

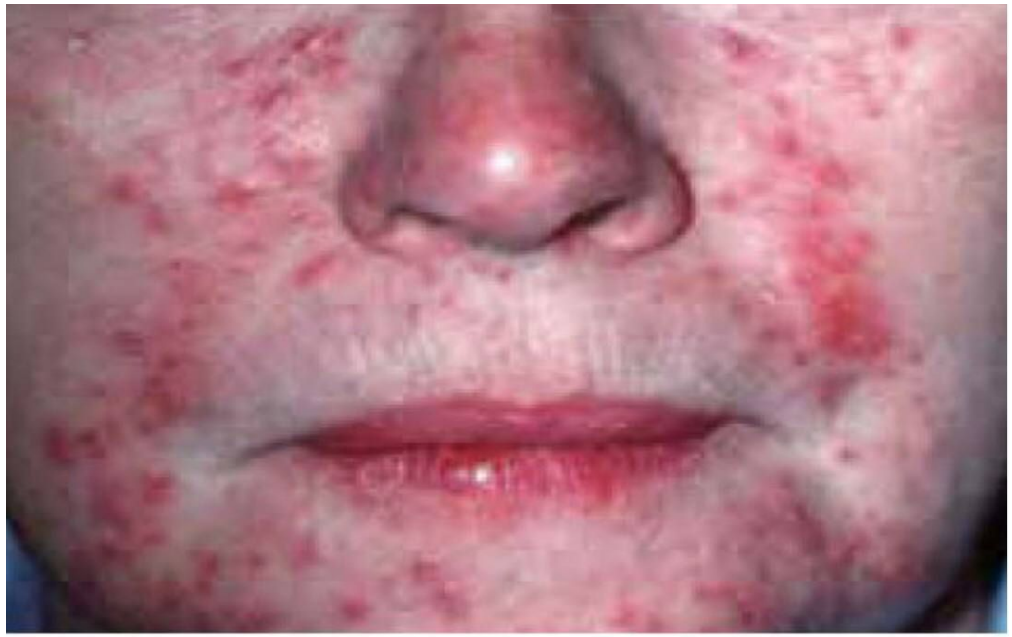
Rosacea subtypes:-

1. **Erythemato-Telangiectatic Rosacea (ETR) or Vascular:**
   * Flushing:- recurrent then permanent.
   * Telangectasia:- begin on ala nasi then nose & cheeks.
   * Edema:- feeling of fullness of cheeks.

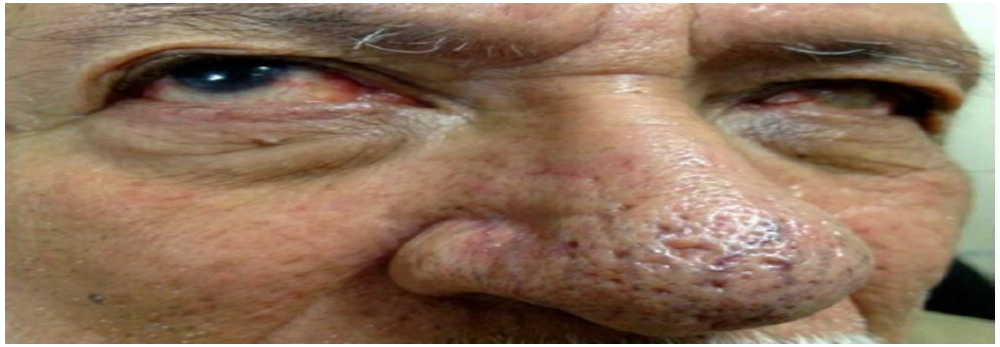


2- Papulo-Pustular Rosacea (PPR)or inflammatory:-

* Varies from small papules & pustules to deep persistant nodules.
* Papules are asymptomatic.
* No Comedones.
* No scarring

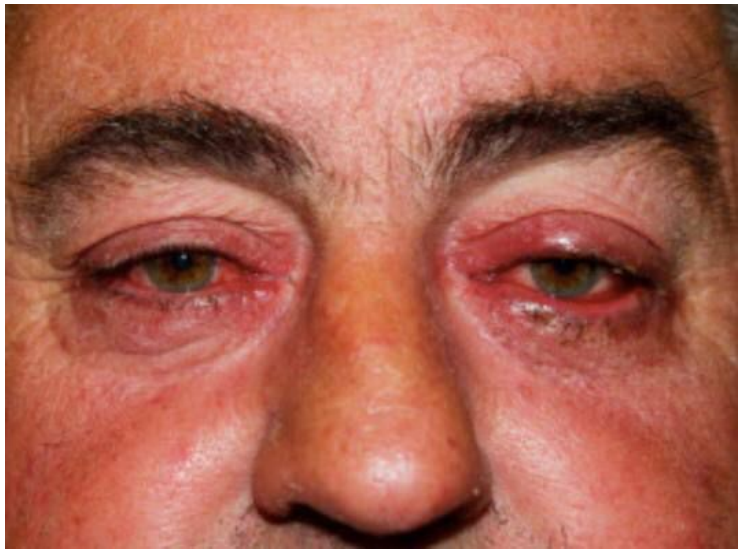


3. Phymatous rosacea:



* Occur in long-lasting cases especially men.
* Appear as :- Deep red irregular lobulated thickening of skin of nose with follicular dilatation.
* Represent hyperplasia of sebaceous glands & connective tissue

4. Ocular rosacea:



Can occur without cutaneous manifestation.

* Mild cases:- scaling of eye lid margin.
* More active disease:- Blepharitis, conjunctival injection.
* Severe cases:- Keratitis, Corneal neovascularization, Scleritis.

Treatment:

A. General measures: I. Sun screen

II. Avoid the exacerbating factors.

B. Systemic treatment:

1. Oral antibiotics:

* Tetracyclines:- 1. Tetracycline:- 250 mg 3 times daily, 3-4 weeks.

2. Doxycycline:- 100 - 200 mg/day.

3. Minocycline:- 100 - 200 mg/day.

* Macrolides:- 1. Erythromycin:- 1 g /day.

2. Azithromycin:-

- ➢ 500 mg 3 times /week in the first month.
- ➢ 250 mg 3 times /week in the second month

Treatment:

2. Oral Metronidazole: 200 mg twice daily .

3. Isotretinoin:-0.5 mg/kg/day for 20 weeks in severe, refractory rosacea .

C. Topical treatment:

* FDA approved topical medications for rosacea:-

1. Metronidazole(MetroGel) :

➢ 0.75% applied twice daily or 1% metronidazole applied once daily

Treatment:

2. Brimonidine 0.33% topical gel (Mirvaso):-

➢ Indicated for persistent facial erythema of rosacea.

3. Azelaic acid 20% cream or 15% gel:

➢ Effective and well tolerated in ttt of papulopustular rosacea


Thank you!!!!!!