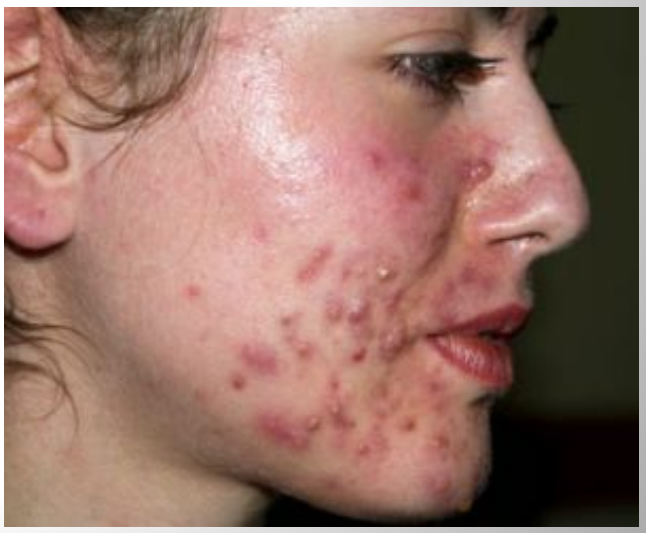
Acne and Rosacea



Definition :

Acne vulgaris is a common. Self- limiting chronic inflammatory disease of the piloseb. unit (sebaceous follicles).

Most cases of acne are polymorphic, presenting with a variety of lesions consisting of comedones, papules, pustules, nodules and as a sequel to active lesions, pitted or hypertrophic scars.

Prevalence:

It's a disease of adolescent and frequently resolves by mid-twenties.

* Both gender are equally affected.

Age of onset between 12- 14 years. Tending to be earlier in females

* Peak age of severity : In females is at 16-17 years

In male is at I7-I9 years.

Etiology and pathogenesis:

1) Increased sebum production or excretion:

Acne patients, males and females excrete more sebum than normal subjects and the level of secretion correlates with the severity of the acne.

2) Hormones:

Androgens (from the testis, ovaries and adrenals) are the main stimulants of sebum excretion. In acne the seb. glands respond excessively to what are usually normal levels of these hormones (increased target sensitivity), this may be caused by 5 alfa reductase activity being higher in the target seb. glands than other parts of the body.

50% percent of females with acne have raised free testosterone levels usually because of a low level of sex hormone binding globulin (SHBG) rather than a high total testosterone.

3) Bacterial:

Propioni bacterium acne, an anaerobic diphtheroid, a normal skin commensal and the principle components of the microbial flora of piloseb. Follicle

P. acne activity :

- Breaks down triglycerides releasing free fatty acids.

- Produces substances chemotactic for inflammatory cells and induces the ductal epithelium to secrete pro-inflammatory cytokines

4) ductal hypercornification:

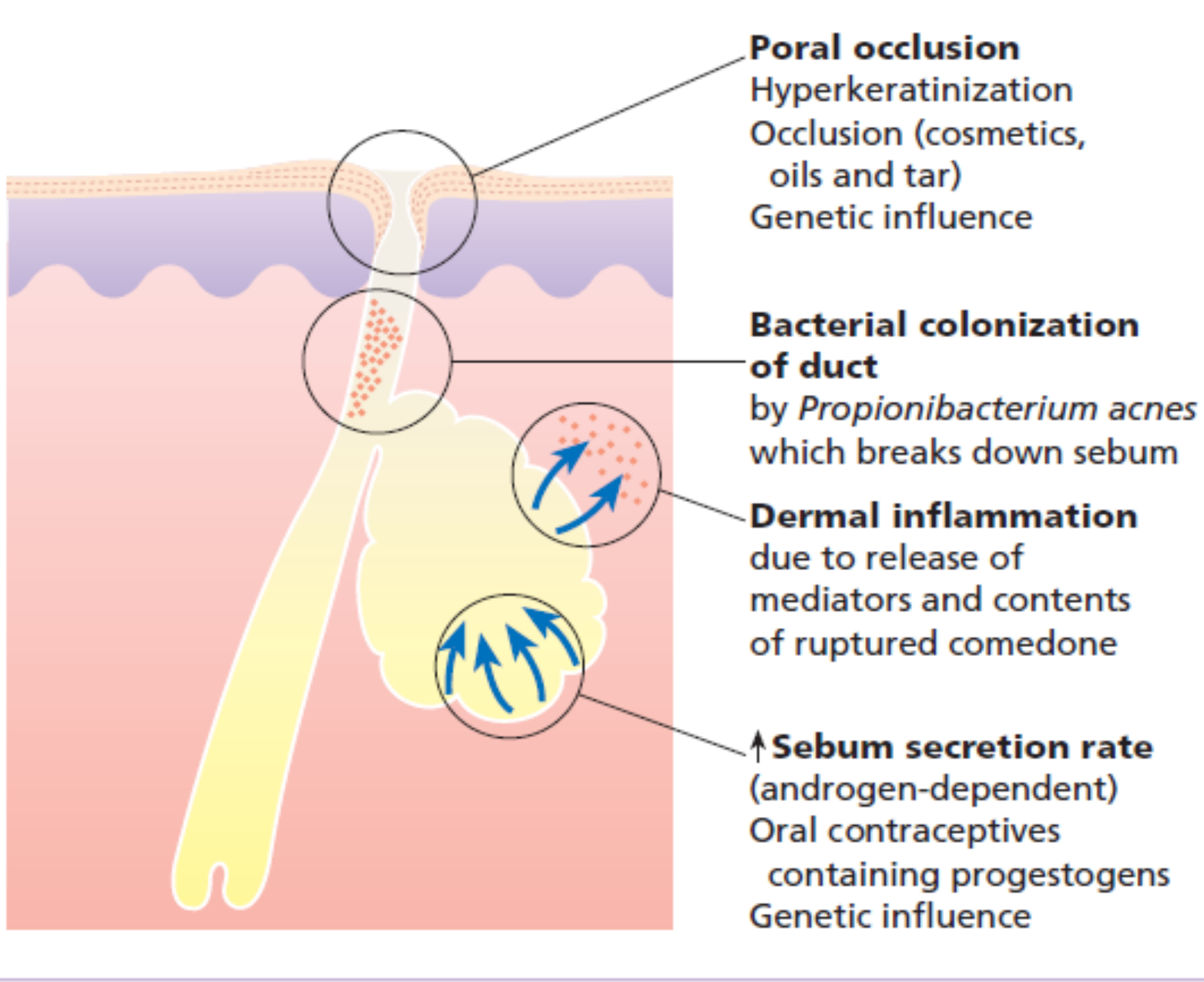
This may result from: - An irritant effect of seb. lipids as they move through the duct.

5) genetic:

The condition is familial in about 50% of those with acne.

6) host response:

May also be important Skin testing with killed suspension of p. acnes show that subjects with severe acne produce a greater inflammatory response at 48 hours than other subjects.


Poral occlusion
Hyperkeratinization
Occlusion (cosmetics, oils and tar)
Genetic influence
Bacterial colonization of duct
by *Propionibacterium acnes* which breaks down sebum
Dermal inflammation
due to release of mediators and contents of ruptured comedone
↑**Sebum secretion rate**
(androgen-dependent)
Oral contraceptives containing progestogens
Genetic influence

7) Other factors:

Factors which help or aggravate acne

* **Drugs**: corticosteroids, androgenic and anabolic steroids, gonadotrophins, oral contraceptive, lithium, Iodide, anti TB, anticonvulsant therapy.

* **Diet**: there is little evidence that any dietary constituent causes acne.

***Sweating and humidity**.

***Stress.**

***Premenstrual flare**.

***Occupation**: induction of chloracne by accidental release of halogenated hydrocarbons.

***Friction and trauma:** (hair ablation).

Clinical features:

The primary site of acne is the face and to a lesser degree the back, shoulders and chest. On the trunk they tend to be numerous near the midline.

- Acne is polymorphic.

- Lesions are either

1- inflammatory

2- non-inflammatory

Non-inflammatory:

Comedones 1) Closed. 2) Opened.

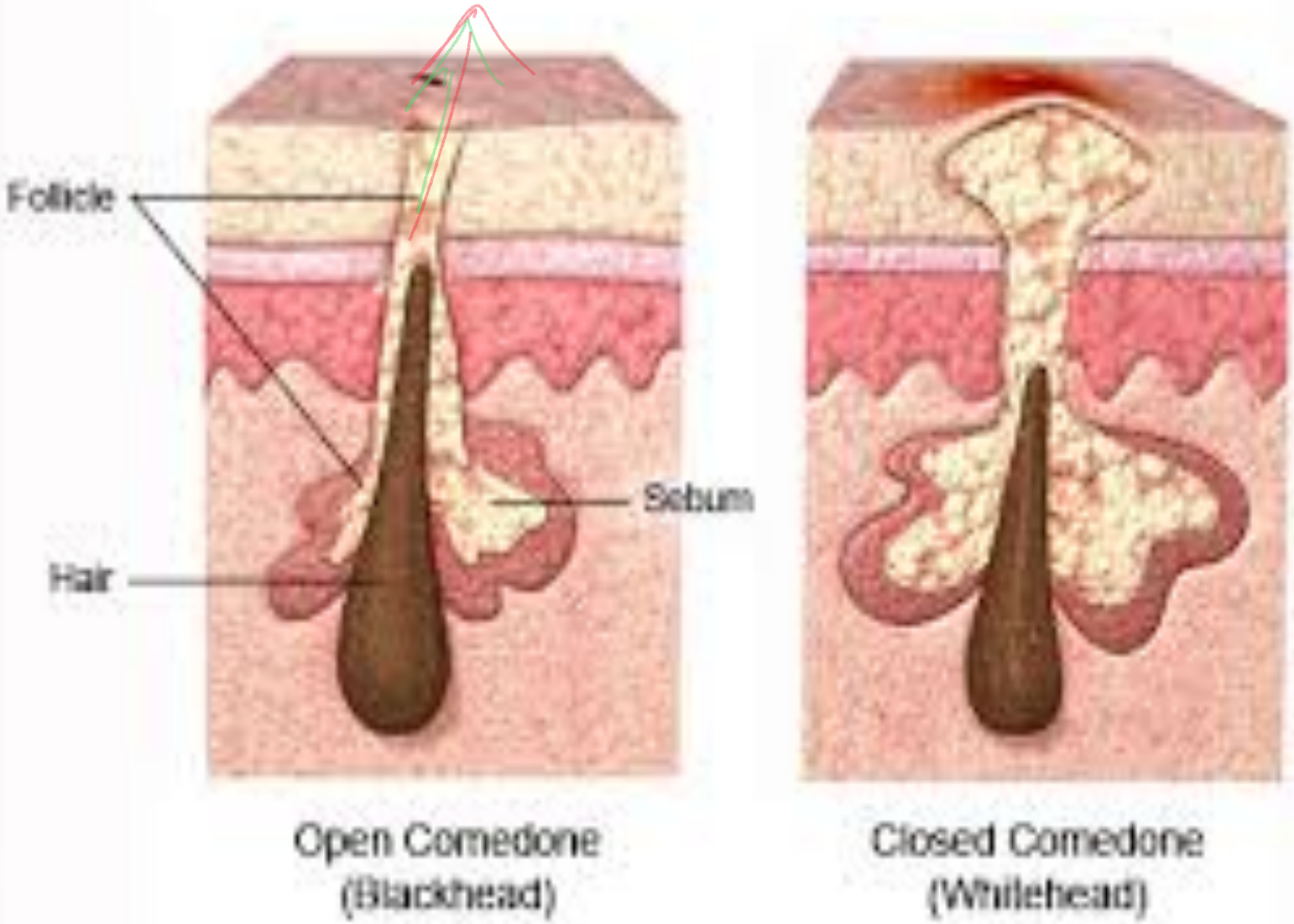
Opened comedones (black head) appear as flat or slightly raised lesion with a central black plug.

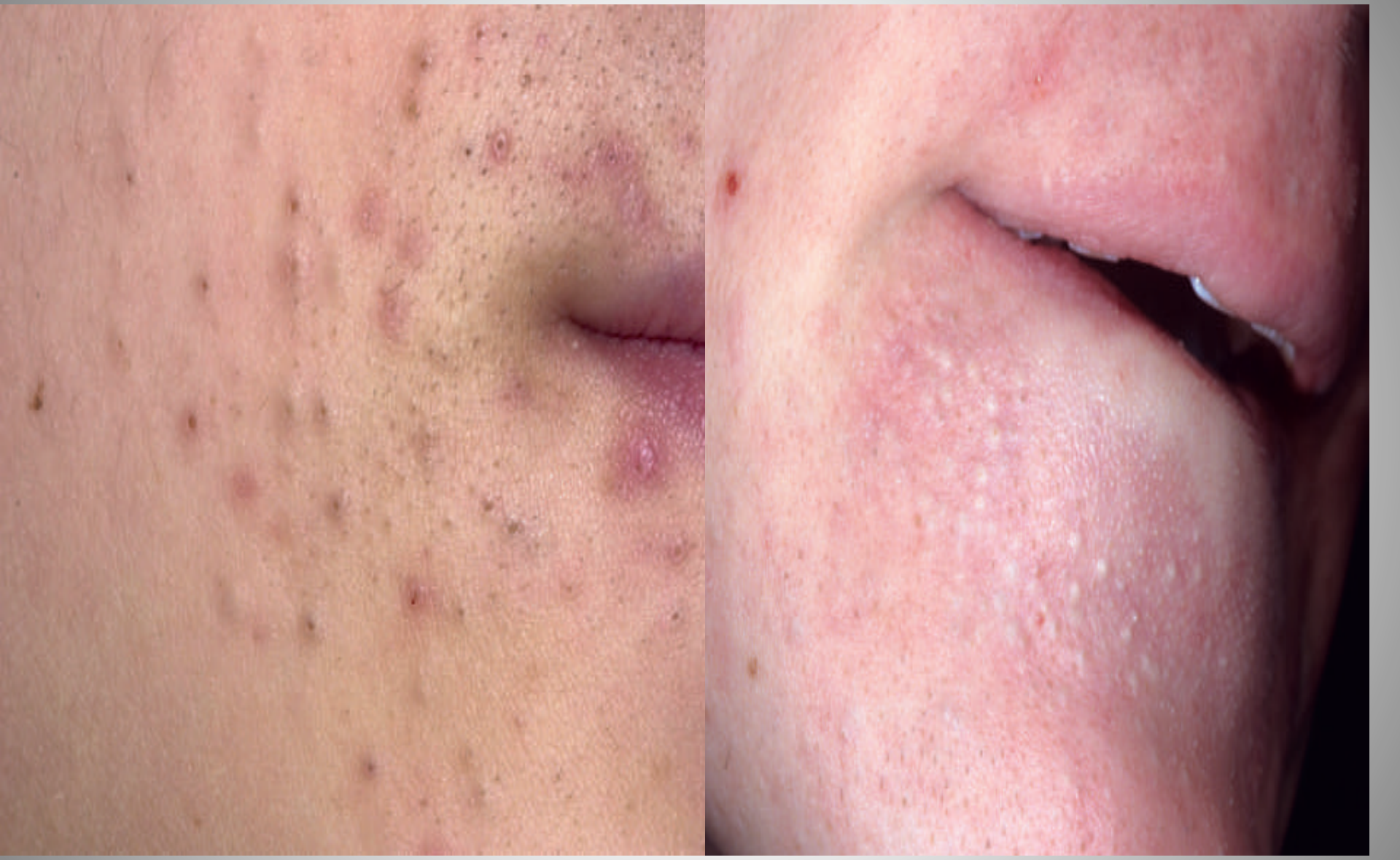
Closed comedones (white heads): appear as slightly elevated pale papules which do not have a Clinically Visible orifice - Closed comedones are potential precursors for large inflammatory lesion

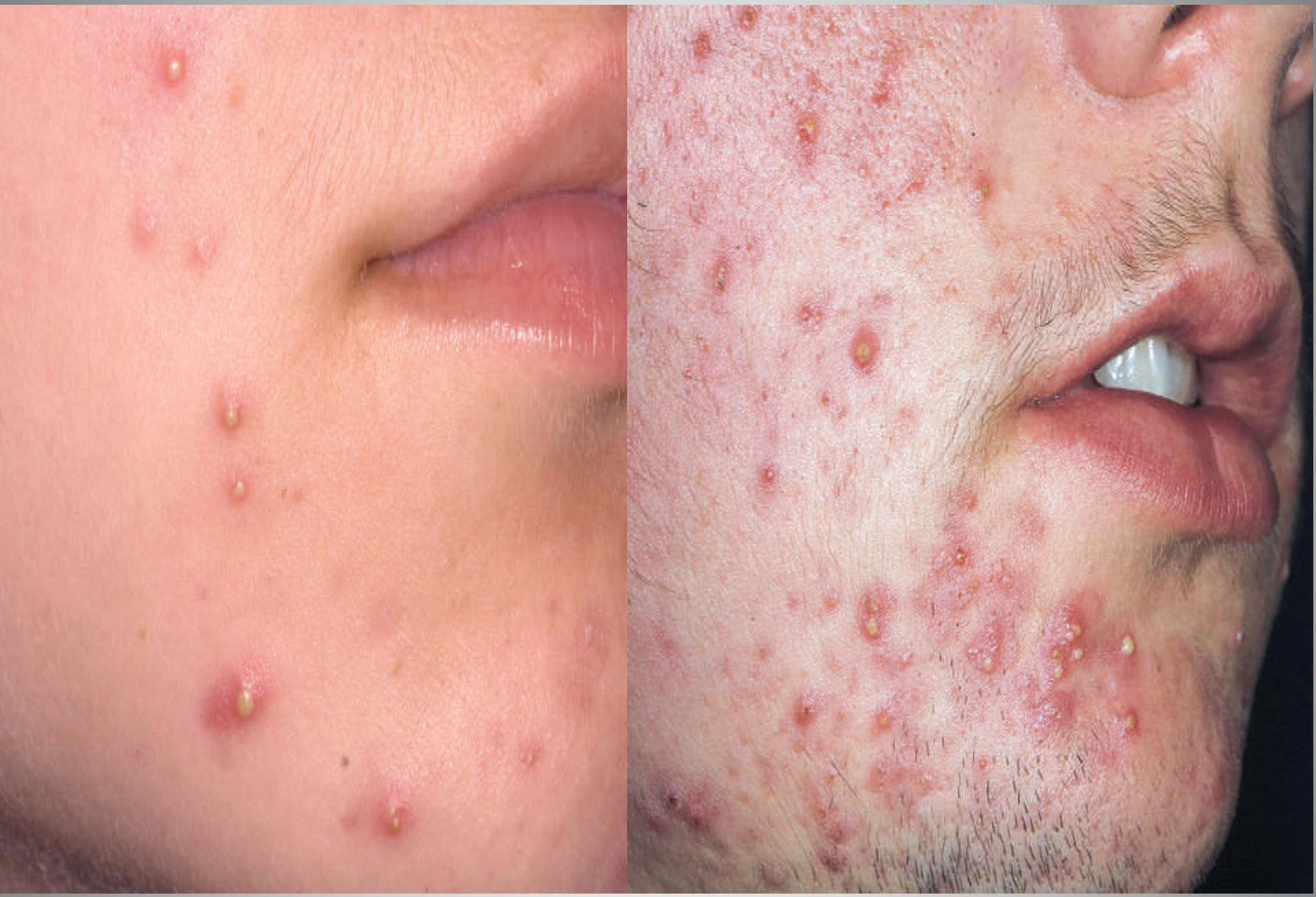
Comedones are the primary lesion of acne.

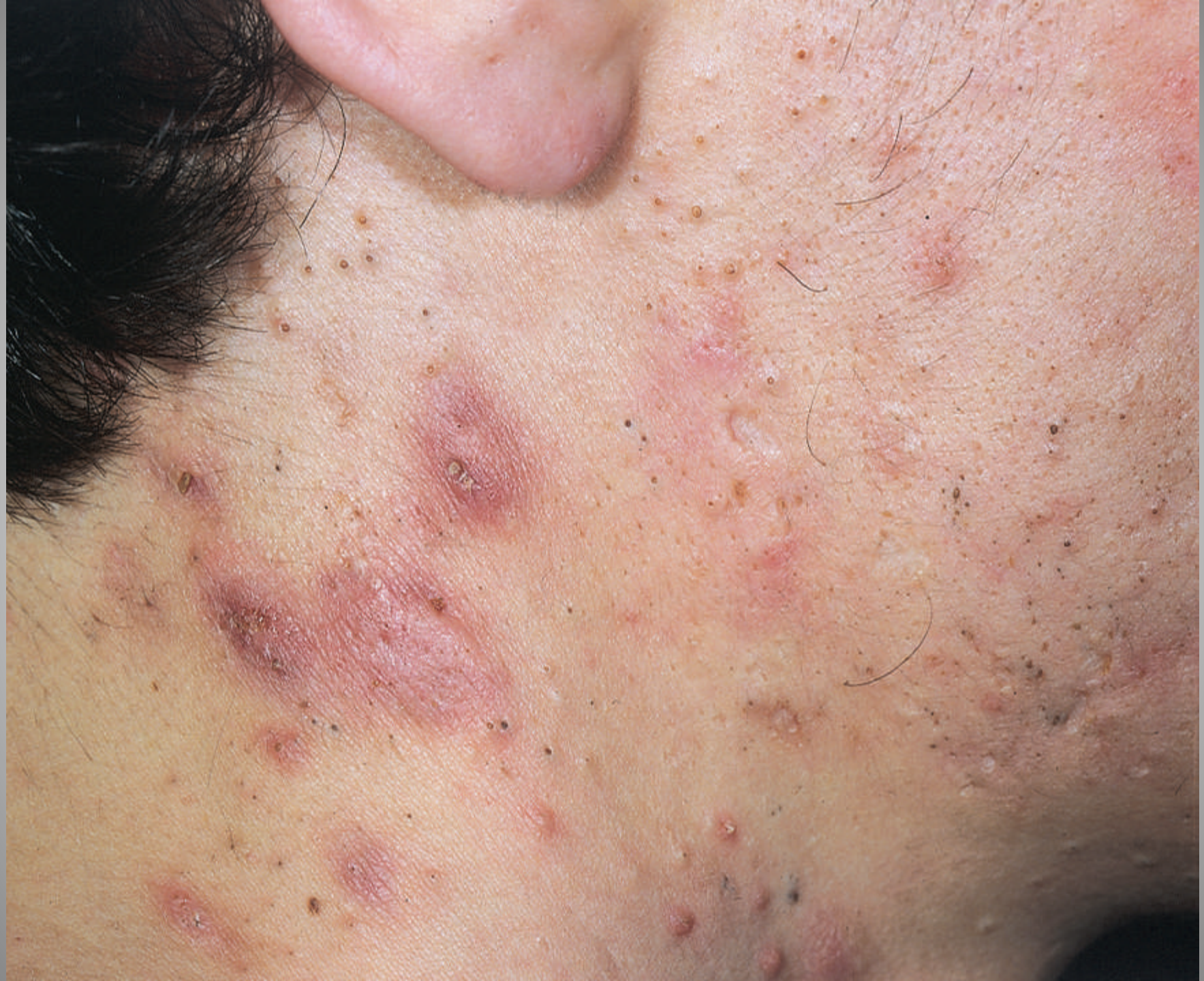
***Inflammatory lesions:**

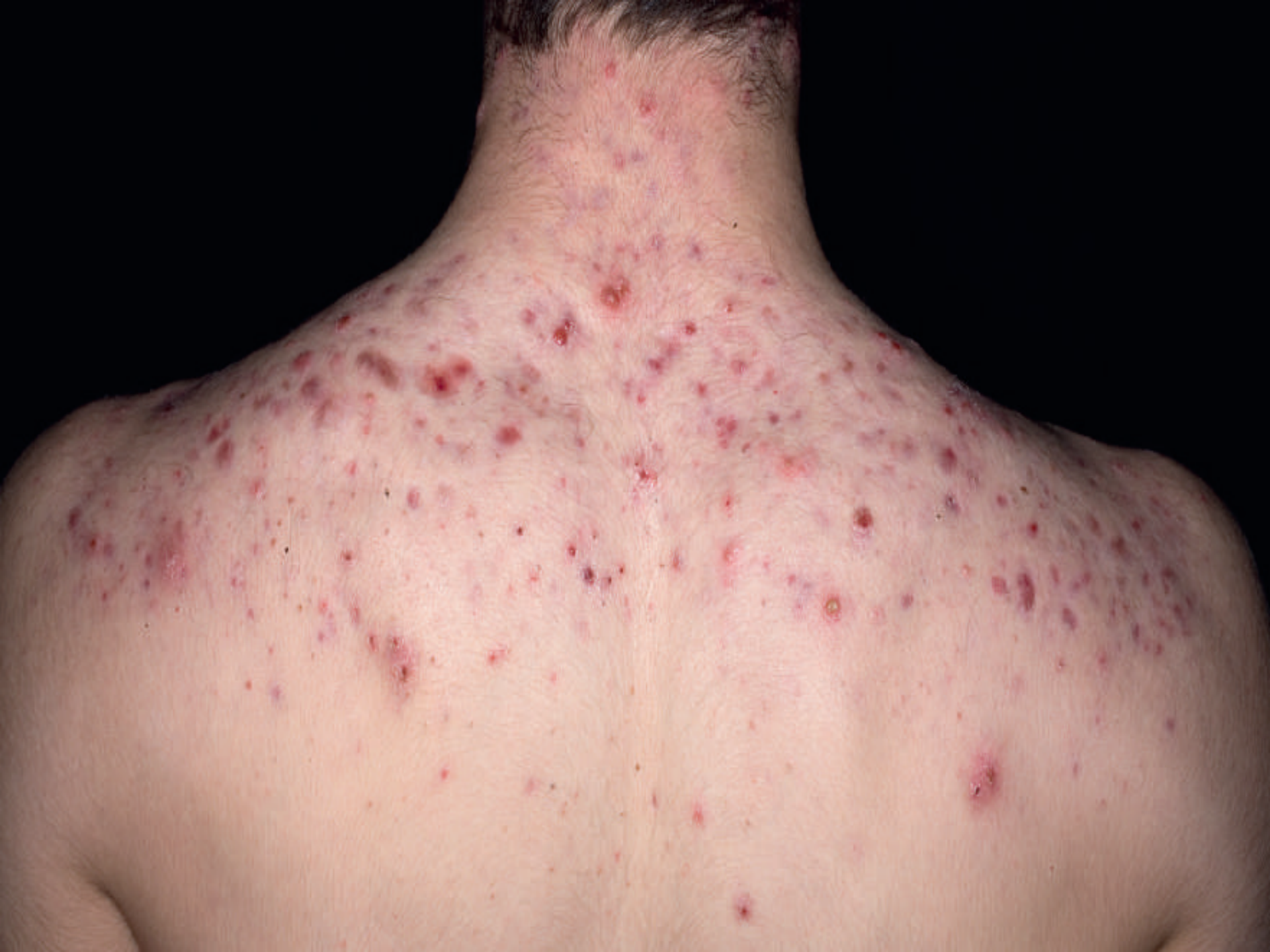
Vary from papules, pustules and
large tender confluent nodules


Follicle
Sebum
Hair
Open Comedone
(Blackhead)
Closed Comedone
(Whitehead)









Complications:

1) gram - ve folliculitis:

A rare but one can easily missed with prolong AB treatment, G -ve organism will proliferate in the ant. nares and spread to the surrounding skin.

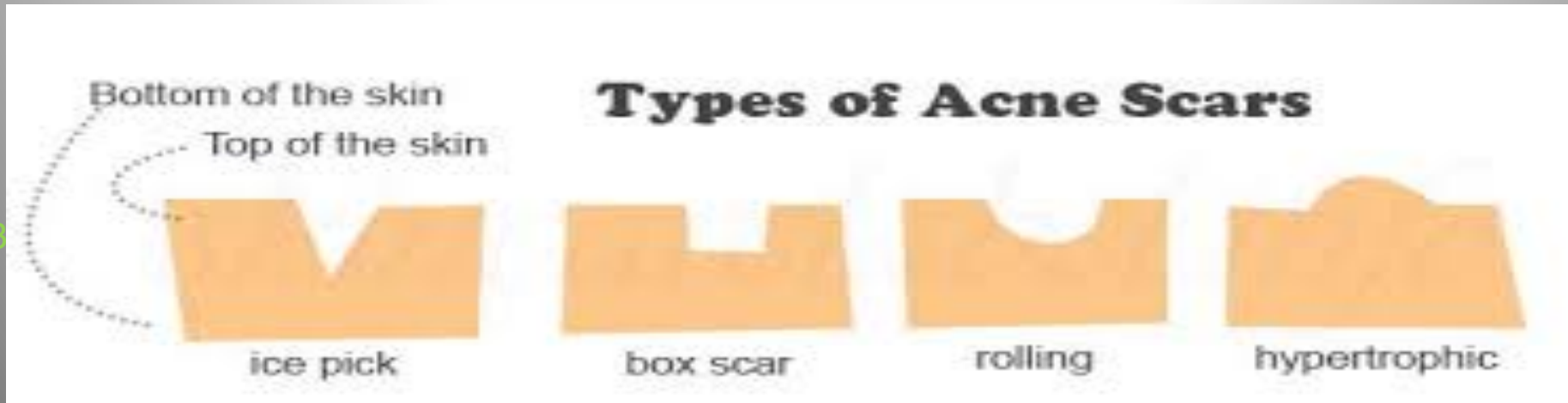
Clinically there is sudden flare with pustules and nodules in patient who otherwise improving Caused mainly by Enterobact. Or klebsiella or proteus.

2) Scars: Three main forms: **A.** Ice-pick scars: sharply punched out pits.

B. Saucer shaped scars: wide and large depressed scars.

C. Hypertrophic and keloidal scars.

3) Mental scars: anxiety, depression and social isolation



Diagnosis and differential Diagnosis:

Diagnosis usually made by finding the polymorphic rash on face, back and trunk (clinical diagnosis usually)

Differential diagnoses:

a. Rosacea.

b. Folliculitis.

c. Pseudofolliculitis barbae.

Treatment :

Five main principles:

1) correct the altered pattern of follicular keratinization.

2) decrease seb. gland activity.

3) decrease follicular bacterial population particularly p. acne and inhibit the production of extracellular inflammatory products (either directly or indirectly by inhibiting the bacterial organism).

4) produce an anti-inflammatory effect.

5) Antiandrogen.

Local and systemic treatment:

A) Local treatment:

1) Cleaning: with soap and water to remove surface sebum.

2) Topical agents:

a) Topical vit A derivatives: Tretinoin

* normalize follicular keratinization and have comedolytic action, prescribed as a lotion, cream and gel, in different concentrations 0.025%, 0.05% and 0.1% usually once daily at night

* Side effect: irritant, teratogenic

* New synthetic retinoids : Trazrotene 0.1% gel, once daily more effective than tretinoin.

Adapalene 0.1% gel, work quicker and tolerated better than tretinoin

b) Benzoyl peroxide: powerful antibacterial used at night, for inflammatory lesions

S.E : dryness and irritation.

c) Topical antibiotics: topical clindamycin and topical erythromycin

d) Azelaic acid cream: 20% cream, It is a bacteriocidal for p. Acne and anti-inflammatory, it inhibit the formation of comedones by reducing hyperkeratinization used twice daily not more than 6 months.

B) Systemic therapy

1. antibiotics:

A) Tetracyclines:

is usually the 1st antibiotic prescribed cause decrease in FFA by increase the esterified fatty acid

dose: 250 mg qid or 500 mg bid on an empty stomach

S.E. : candida vulvovaginitis

C/I : children less than 12 years, pregnant and lactation

Other tetracyclines:

Doxycycline : useful and is well absorbed even with meal, lipid soluble and penetrate the sebaceous follicle effectively

Dose:100 mg once or twice daily

S.E: photosensitivity

minocyclines: most effective AB in treatment of acne but expensive

dose: 50mg/day initial dose then slowly increased to 100 mg/d

S.E: bluish hyperpigmentation of healing lesions (temporary)

B) erythromycin:

next AB of choice, prescribed for females who might become pregnant in a dose of 1g/d in divided doses

C) co-trimoxazole:

used only in patients who don’t respond to other AB

2. Hormonal: a combined antiandrogen-estrogen treatment used and this help resistant acne in female. 2 mg cyproterone acetate + 0.035 mg ethinyestradiol, other antiandrogens is Spironolactone
Glucocorticoids, in low dose used in certain limited conditions only suppress the adrenal androgen excess

3) Retinoids:

Isotretinoin (oral synthetic retinoids) which inhibits :

- sebum excretion
- The growth of p.acne
- Acute inflammatory process

It used in severe recalcitrant cases and acne conglobata

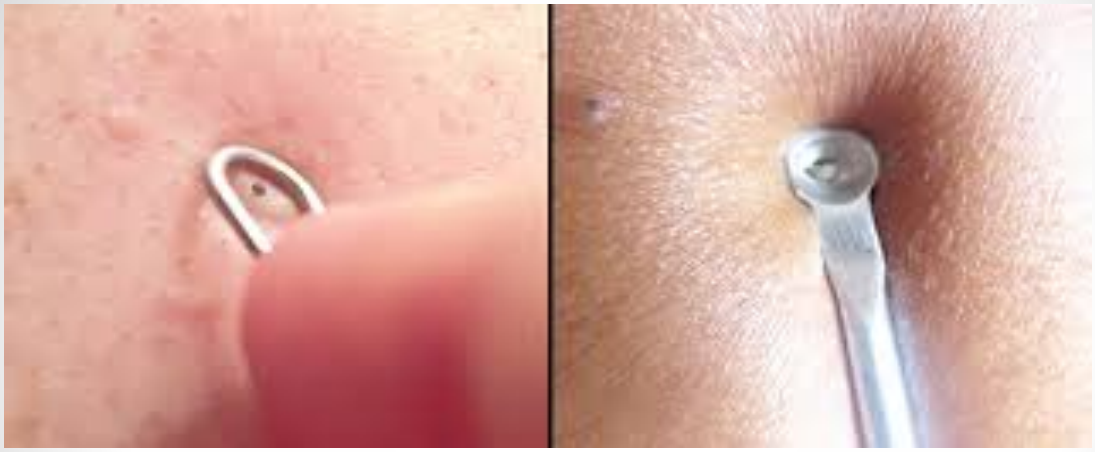
Dose: 0.5 – 1 mg/kg body wt/day for 4-6 months

S.E : dryness, highly teratogenic

Physical therapy

a. acne surgery: removal of the comedones by certain instruments especially with closed comedones if they persist leading to inflammation

Opened comedones: removal only for cosmetic purposes



b. intralesional steroids: in cases of cystic acne

Diet: no evidence to support the elimination of diet in acne

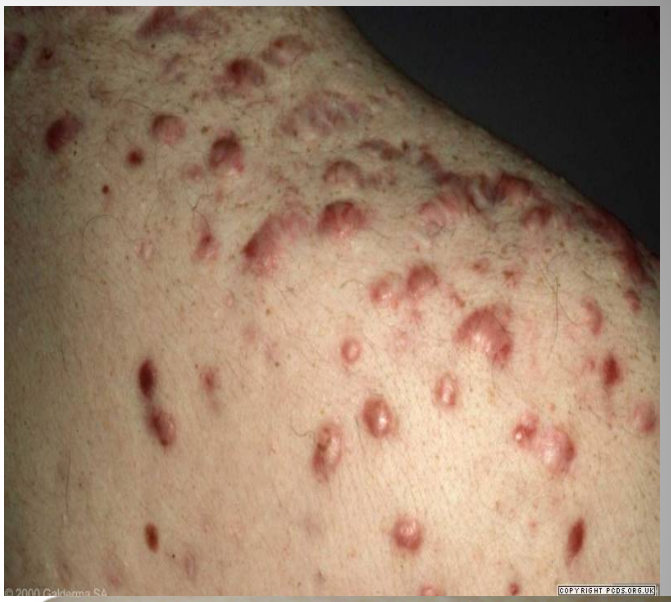
Treatment of complications:

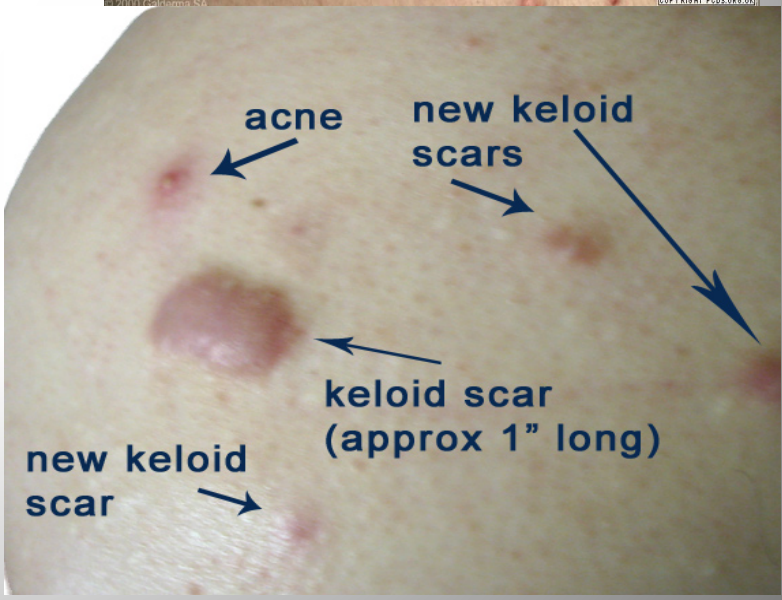
A) Scarring can be decreases by:

1. Skin resurfacing using CO2 laser, Erbium yag laser.
2. RFmicroneedling
3. Dermabrasion
4. Chemical peeling
5. Hyaluronic acid injection

B) Hypertrophic scars and keloids: Intralesional injection of steroids, don't excise and remove

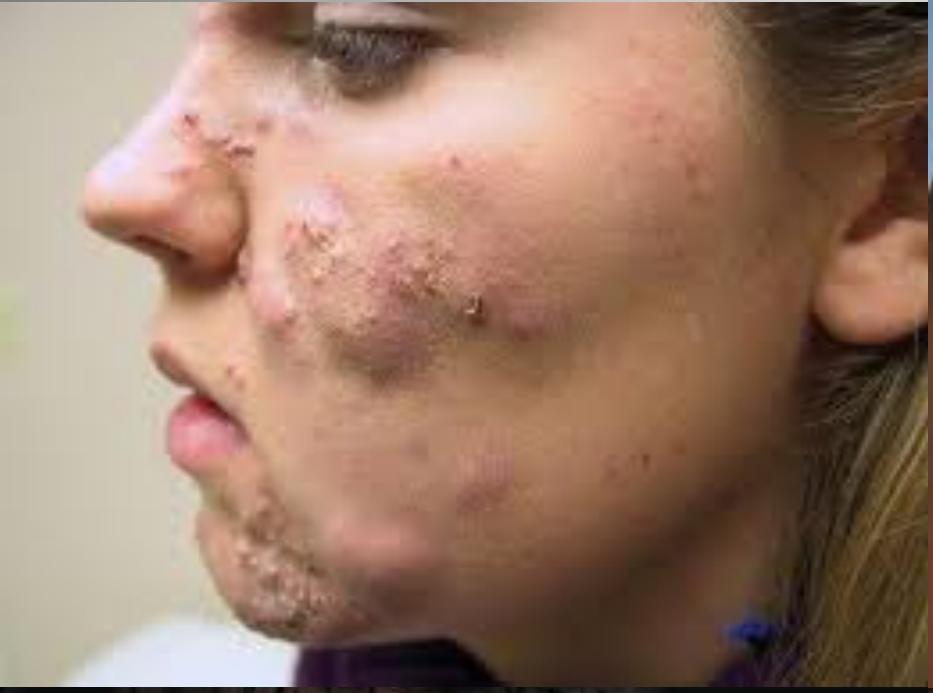
C) Mental scarring: Treatment of depressive mood produced by acne

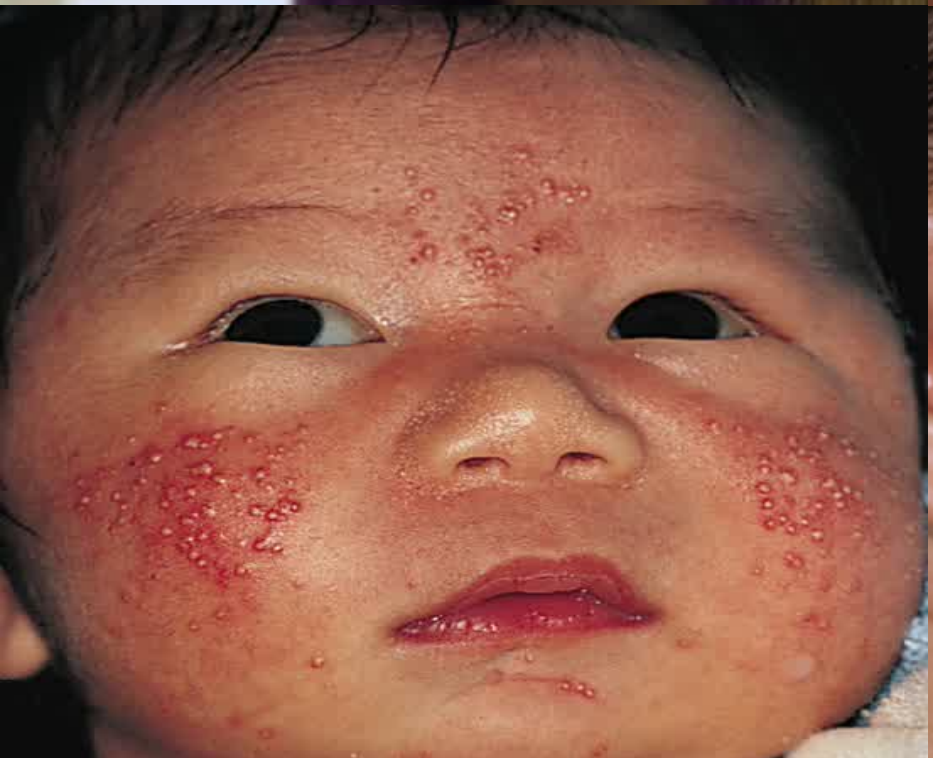
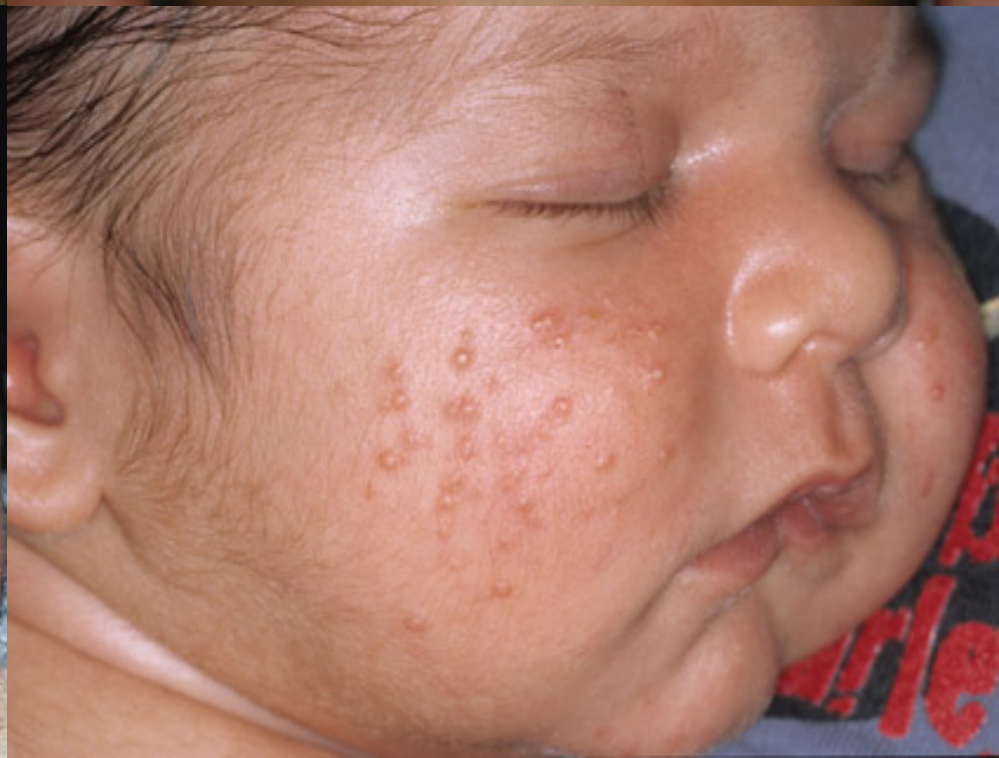




Miscellaneous types of Acne:

1. Neonatal acne: an acne form eruption may develop on nose and cheeks due to trans- placental activation of sebaceous glands by maternal androgens
2. Drug acne or acne medicamentosa
3. excoriated acne: common in young girls. Obsessional picking with rubbing leaves discrete denuded areas
4. Occupational acne
5. Tropical acne
6. Acne Aestivalis: uniform multiple red papule occur after exposure to sun light
7. Acne cosmetica: various cosmetic compounds may induce comedones so avoid greasy oil occlusive cosmetics
8. acne conglobate: severe form of acne with abscess or cysts with intercommunicating sinuses that contain thick fluid or pus and on resolution it leaves deeply or hypertrophic scars.



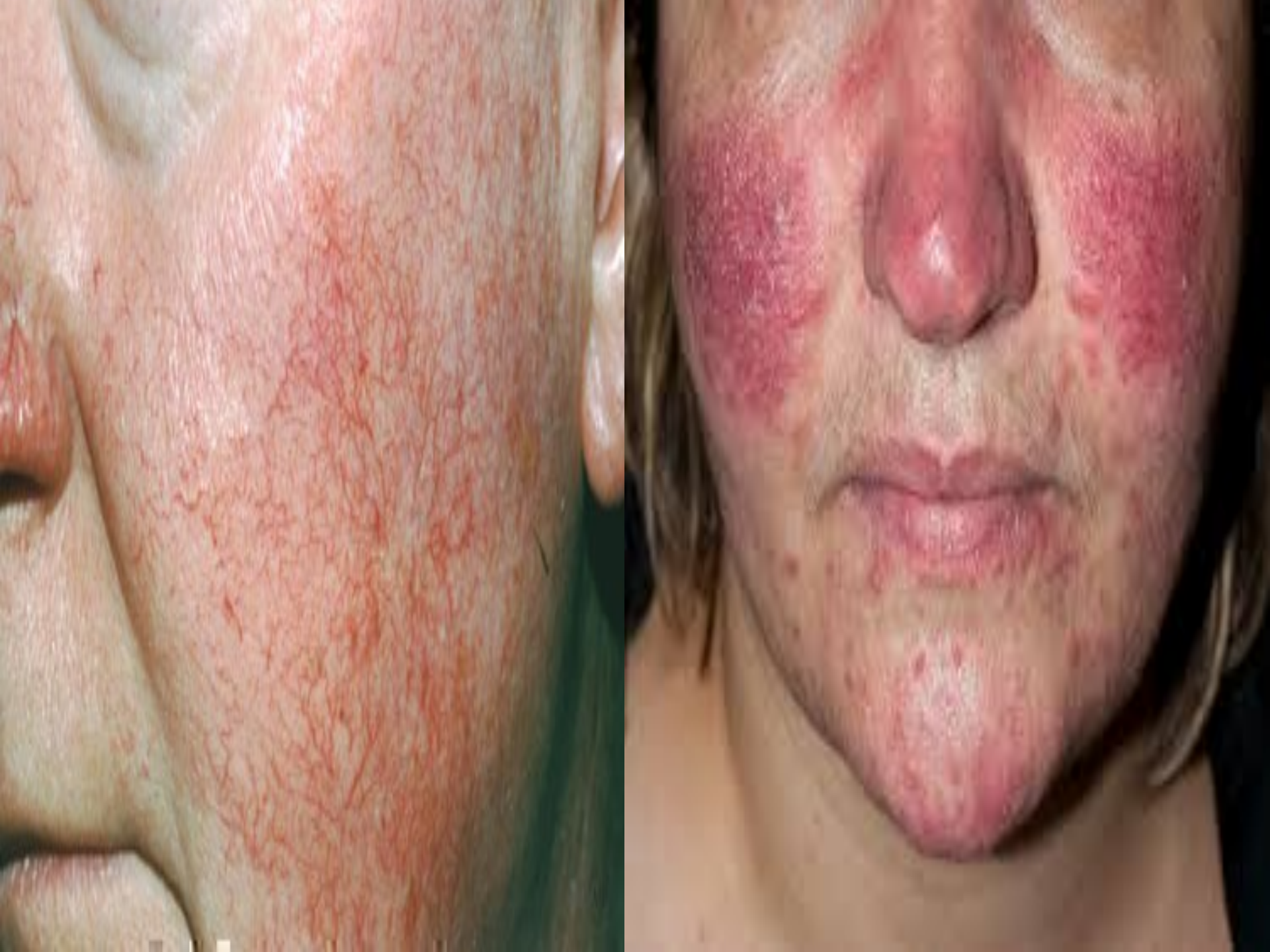
Rosacea

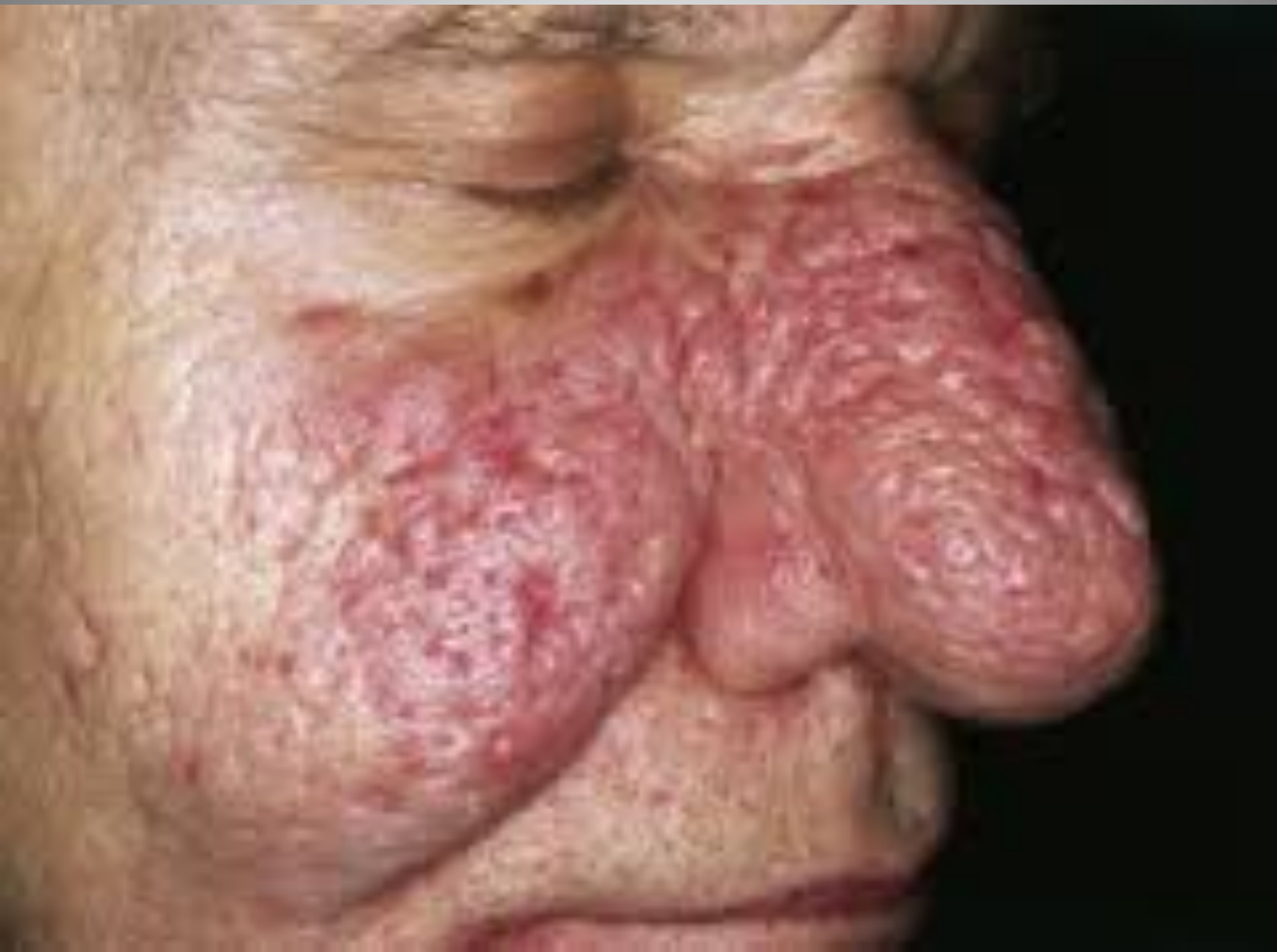
chronic inflammatory disease of the flush areas, patient presents with a prior history of idiopathic facial burning or flushing over prolonged periods leading to polymorphic picture of

1) persistent erythema
2) telangiectasia
3) inflammatory papules and pustules
4) later rhinophyma.

Epidemiology

* Rosacea is much more common in fair skinned individuals, rarely seen in black people.
* Women are more commonly affected than men, although rhinophyma is more common in males.
* It is a disease of adults, usually the patient is a middle aged female between 30-50 years of age.





Aetiology

Unknown, however certain factors have been proposed but unconfirmed. The aggrevating factors include:

1. GIT: alcohol ingestion, hot foods and drinks, infection with helicobactor pylori
2. Sun exposure
3. Psychological stress
4. Drugs: topical steroids
5. Infection with Demodex follicularum and HIV

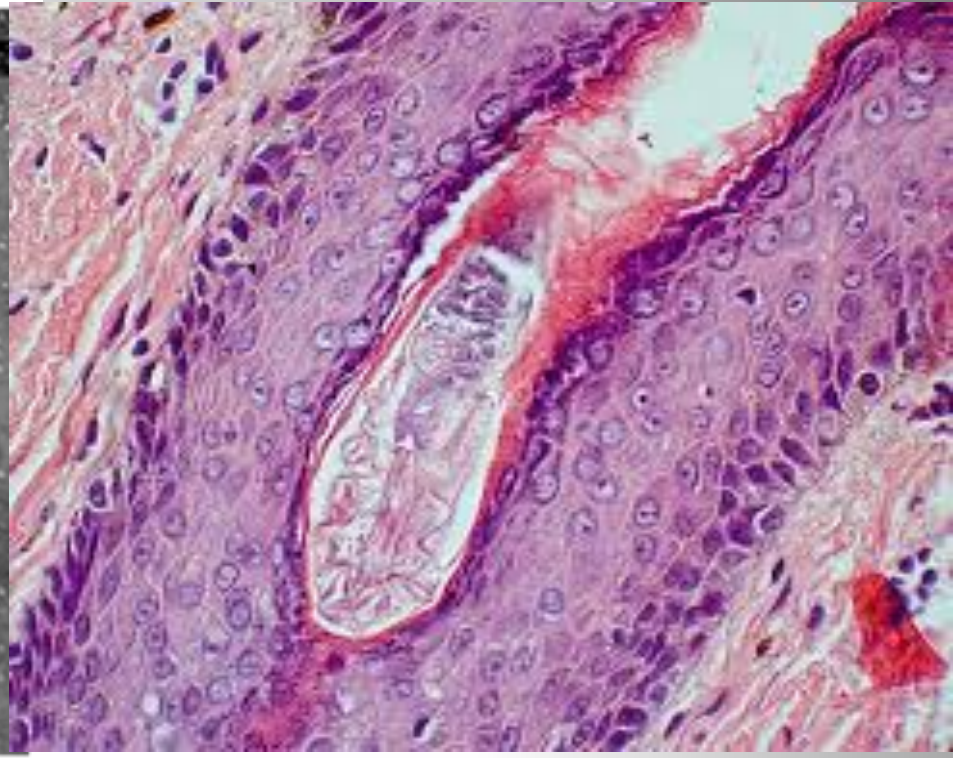
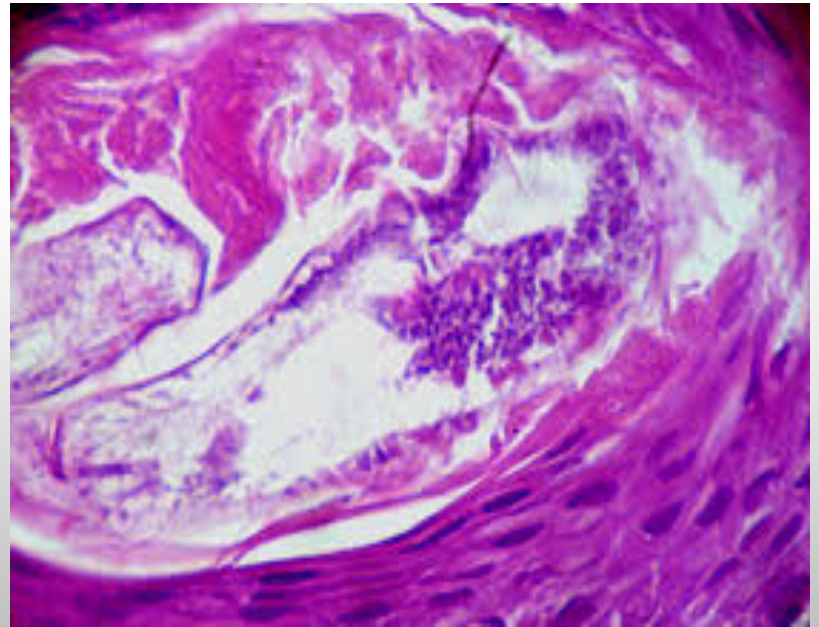


Figure 2. *Demodex folliculorum* under light microscope (X100 magnification).



:Clinical features

1) Flush area are the cheeks, nose, center of the forehead and chin sparing and periorbital and perioral areas.

2) Episodes of intermittent flush followed by fixed erythema and telangiectasia

3) Discrete inflamed papules and pustules

4) Rosacea unlike acne has no comedones or seborrhoae with prolonged course of exacerbations and Remissions

Complications:

1) Ocular: conjunctivitis, blepharitis and sometimes keratitis leading to blindness

2) Rhinophyma: excessive proliferation of the sebaceous glands of the nose

3) Lymphedema: especially periocular

:Differential diagnoses

1-Acne vulgaris

2-Seborrheic dermatitis

3-SLE

4-Dermatomyositis

Treatment:

Similar to acne vulgaris

1. Broad spectrum antibiotics as tetracyclines, erythromycin, co-trimoxazole and metronidazole
2. Corticosteroids should not be given except for the weak form to decrease erythema
3. Systemic retinoid
4. Sun screens
(5. Certain specific make up to cover the erythema (cover mark
6. Electrocautery for the telangiectasia
7. Surgery for rhinophyma