Acne and Acne related disorders

Objectives :

- **Not given (below are some of the course objectives related to this lecture)**
- To enable medical students to recognize the most common skin diseases and to manage
  them.
- To be familiar with the diagnostic laboratory tests pertinent to dermatology.
- To help students to formulate decent differential diagnoses of skin diseases.

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Sources*: 434 slides (doctor didn't give us this year slides), doctor notes , FITZPATRICK color atlas + 433 team male and female +434 team*

[Color index : Important | Notes | Extra]

Acne

Importance:

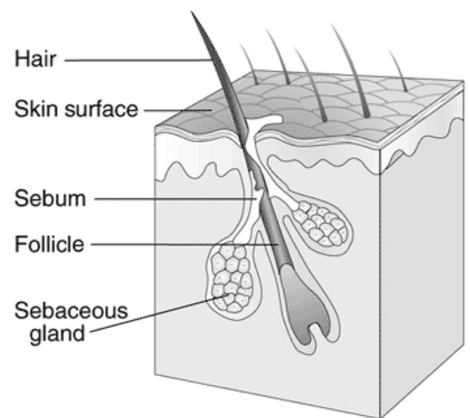
- 85% adolescents experience it *if they ask you what is the primary lesion in acne it's most likely to be **comedone***
- Prevalence of comedones(lesions) in adolescents approaches 100%
- Acne vulgaris is the most common cutaneous disorder in the U.S.
- 10 percent of all patient encounters with primary care physicians.
- Pts can experience significant psychological morbidity and, rarely, mortality due to suicide.
- Important that physicians are familiar with Acne Vulgaris and its treatment.

Definition:

Acne vulgaris is a common chronic skin disease involving blockage and/or inflammation of pilosebaceous units (hair follicles and their accompanying sebaceous gland) resulting in greasiness and polymorphic skin eruption.

Acne can present as non-inflammatory lesions, inflammatory lesions, or a mixture of both, affecting mostly the face but also the **back** and chest.

IF you are suspecting rosacea check the back if it's involved it's more likely to be acne



pilosebaceous units in the dermis:

❖ These units consist of hair follicle and the associated sebaceous glands.

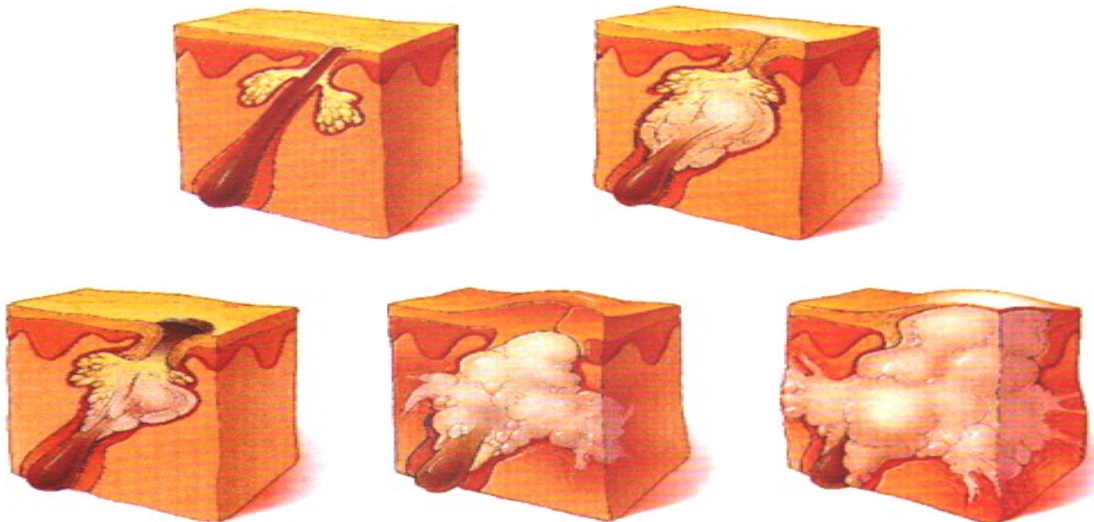
❖ They are connected to the skin by a duct(infundibulum) through which the hair shaft passes.

❖ The cause of acne is an increase in the activity of the sebaceous glands and the epithelial tissue lining the infundibulum.

Etiology:

Acne vulgaris is a disease of pilosebaceous follicles and one of the following could be the cause:

- ❖ Retention hyperkeratosis. the lining of sebaceous gland becomes hypertrophied which makes the sebum cannot go out (Potential hyperkeratosis which means the lining of sebaceous gland is hypertrophied which means when there's blood it will be blocked & the sebum will not go out then you have swelling and have pustules, papules and cyst)
- ❖ Increased sebum production.
- ❖ Propionibacterium acnes (P.acne) within the follicle.
- ❖ Inflammation
- ❖ It also has a genetic aspect (Acne runs in family).
- ❖ Occupation (Environmental, Mechanical) e.g. exposure to acnegenic mineral oil (Pomade acne), dioxin.
- ❖ Drugs Oral and topical Hydrocortison (Steroid acne), Lithium, Hydantoin, contraceptives.



- ❖ The most important cause is the hormonal imbalance.
- ❖ A shift from pre puberty to puberty causes an increase in hormones which would affect the pilosebaceous gland (Sebaceous glands secrete the oily, waxy substance called *sebum) causing it to increase the production of sebum*
- ❖ hypertrophied sebaceous gland produce large amounts of sebum
- ❖ Other cause would be the bacterial cause, which flourishes in case of increased sebum production

Retention Hyperkeratosis: a healthy pore will shed approximately one layer of dead skin per day inside the pore. An acne-prone pore will shed up to 5 layers per day! The skin cannot keep up by expelling all these dead skin cells so they start to build up in the pore As the skin cells start to build-up inside the pore, they start to become sticky. At this point, a microcomedone has started to form. A microcomedone cannot be seen just by looking at your skin

Postinflammatory hyperpigmentation mentioned by the doctor but not in the slides:
Acne can lead to something called post inflammatory hyperpigmentation that can persist for years not because of scarring and in some cases is considered the worst thing that can happen to the patient.

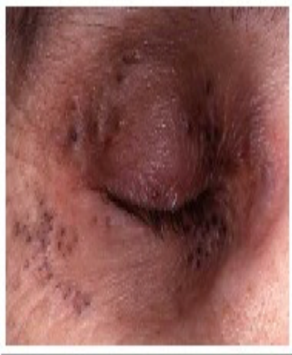
Types and Definitions:

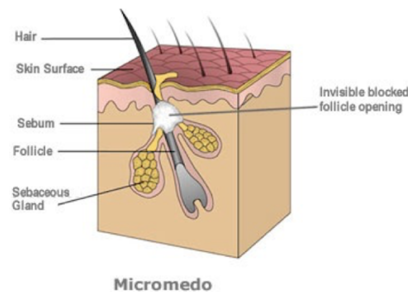
Microcomedone:

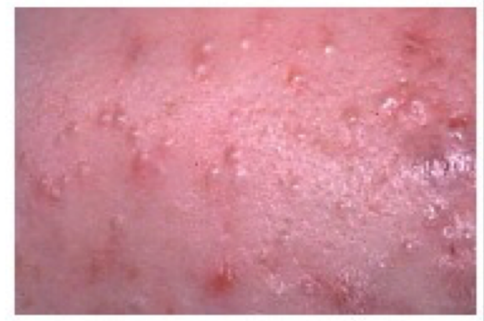
❖ Hyperkeratotic plug made of sebum and keratin in follicular canal

❖ Tow types:

- Closed comedones (whiteheads)
- Open comedones (blackhead)







Inflammatory Acne:

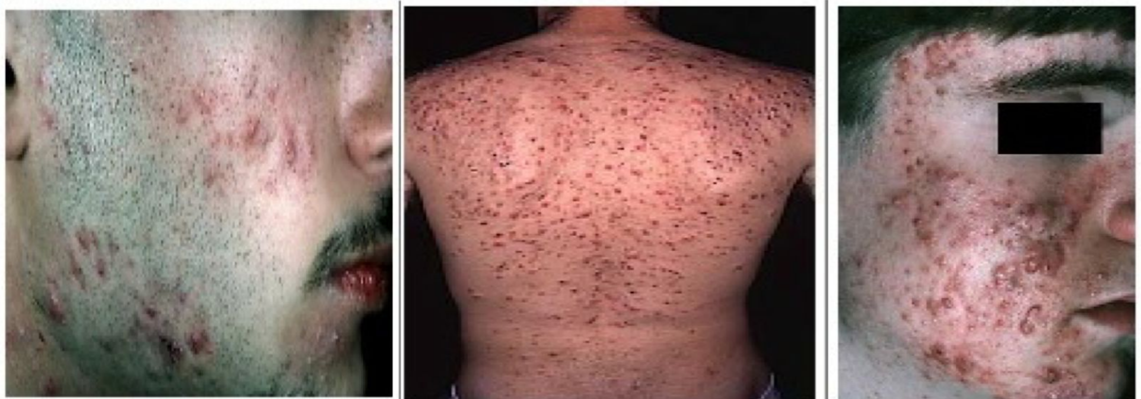
❖ Acne characterized by inflammation surrounding the comedones, papules, pustules, and nodulocystic lesions. it may cause permanent scarring.

❖ Normal sebum does not contain free fatty acids and is nonirritating, however, in the presence of **biolytic enzymes** produced by P.acne , triglycerides of the sebum are split and release fatty acids which are irritating to the tissue.

❖ The inflamed follicle or pustules either heal in about a week or develop in to cyst or sterile abscesses, which can lead to scaring.

Cysts:

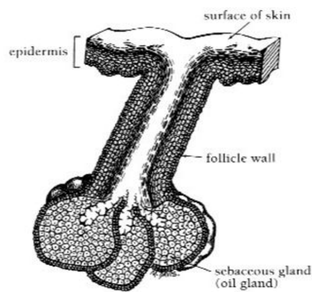
When follicles rupture into surrounding tissues, resulting in papule/pustule/nodule.

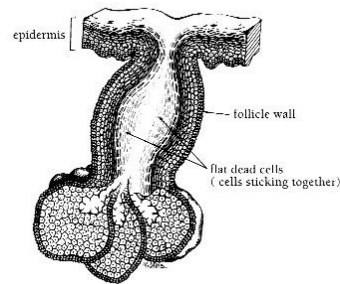


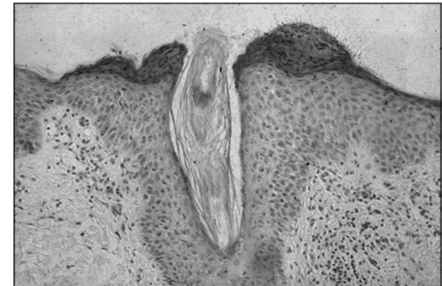
Pathogenesis: (three main steps recognized and hypothesized):

1. **Follicular Hyperkeratosis** (the cause not fully understood) theory suggest:

   - Deficiency in Linoleic acid.
   - The effect of 5-*a* reductase enzyme on converting Androgen (Testosterone) hormone to the active acnegenic and potent (Dihydrotestosterone) DHT.
   - The direct effect of Interleukin-1 on follicular hyperkeratosis.







Perifollicular Hyperkeratosis histology

2. **Seborrhoea** (a common feature between patients with acne).

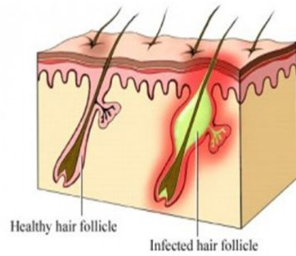
   - Abnormal production of abnormal sebum increasing the ratio of wax ester to cholesterol and cholesterol ester and is believed to be the response of sebaceous glands to DHEA.

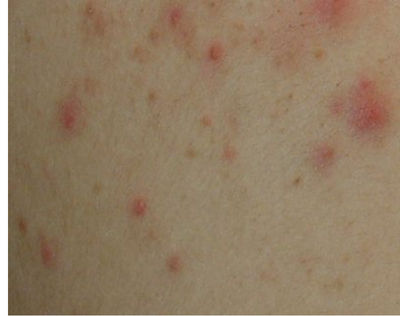
3. **Colonization** of the affected unit with bacteria:

   - Propionibacterium *acne* and yeast called Malassezia *furfur.*

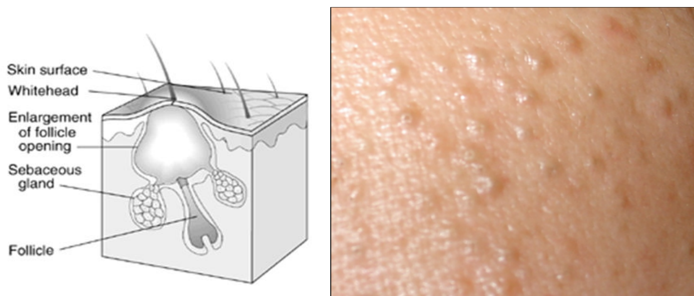
Clinical features:

- Papules: (Less than 0.5 cm).
  - Inflammatory papules.

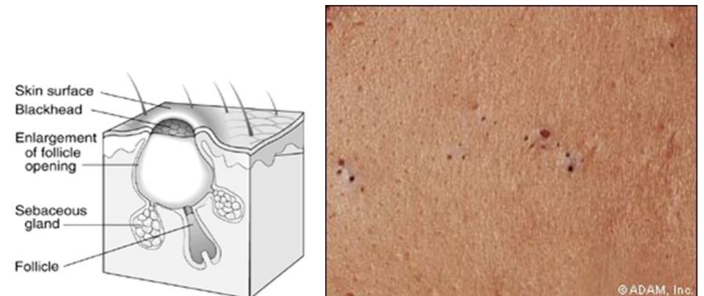




- Comedones (Open “Blackheads” or closed “Whiteheads”):
  - Open Comedones (Blackheads)
  - Closed Comedones (Whitehead)

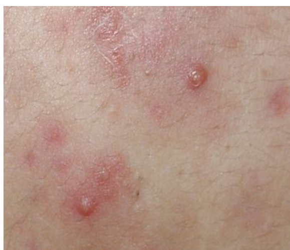


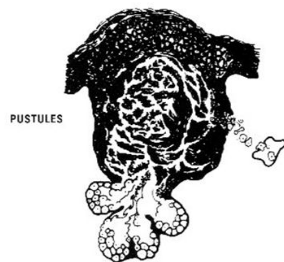
Closed comedone



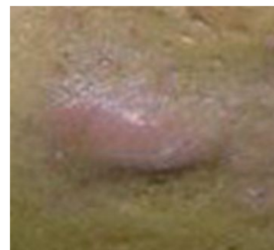
Open comedone

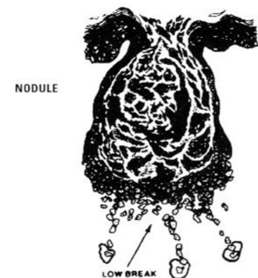
- Pustules.



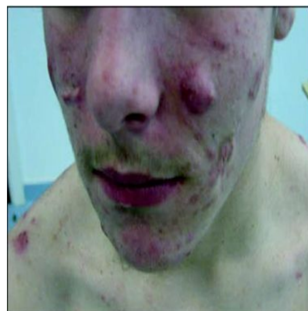


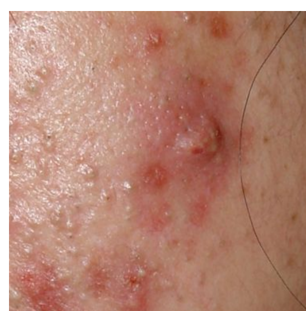
- Nodule (more than 0.5 cm).





- Cystic acne: the cysts are usually large 1-4 cm.





Severity of Acne:

- **Typical mild acne:** comedones are predominate
- **More severe cases:** pustules and papules predominate (heal with scar if deep)
- **Acne Conglobata:** suppurating cystic lesions predominate (severe scarring may results)

Aggravating Factors:

- Changes in the sebaceous activity and hormonal level (e.g. before or during premenstrual cycle)
- High humidity conditions
- Local irritation or friction
- Rough or occlusive clothing
- Cosmetics(having greasy base)
- Diet: chocolate, nuts, fats colas, or carbohydrates.

In case of chocolate has been studied a lot and they didn't find any relation

- Oils greases or dyes in hair product.

Medications That Can Cause Acne:

- ACTH
- Barbiturates
- Lithium
- Iodides
- Steroids
- Azathioprine
- Isoniazid
- phenytoin Disulfiram (antiepileptic)
- Vitamins B2,6,12
- Cyclosporine (chemotherapy)
- Halogens (are five chemically related elements: fluorine (F), chlorine (Cl), bromine (Br), iodine (I), and astatine (At).)

The dr. said "i don't want you to forget that corticosteroid can cause acne if used as cream even though that it can be used as a treatment in some cases as intralesional injections.

Treatment options of Acne Vulgaris: For me there is two important classification that change your approach for treatment 1- scarring 2- non scarring

- Depends on type of clinical lesions
- Microcomedone matures in 8 weeks
- Therapy must continue beyond this time frame
- Considering the heterogeneity in the acne literature, and no clear evidence-based guideline are available

Over The Counter (OTC) products:

- ❖ Sulfur 2--‑10 % other forms, such as zinc sulfide or sodium thiosulfate.
- ❖ Sulfur presents a paradox in that it helps resolve formed comedones but may promote the formation of new ones.

Due to this comedogenic effect The use of salicylic acid or resorcinol is preferred.

- ❖ Benzoyl peroxide (5 to 10%)a primary irritant.
- ❖ Salicylic acid is used in concentration of o.5 to 2%.
  Applied at night after washing the affected area with soap and water.
- ❖ Resorcinol (1 to 4%) may produce a dark brown scale on some black skinned people.

Tretinoin Trans Retinoic Acid: مثل differin

لما يجيني مريض اقول لا تستخدم غسول معه لانها تسبب dryness, redness and irritation

- ❖ The acid form of vitamin A, is a strong primary irritant.

- The products are applied at night. They cause a feeling of warmth or slight stinging .

- ❖ Optimum Results occur in3 to 4 months.
- ❖ Care should be taken to avoid touching with eyes, nose, and mouth with tretinoin.
- ❖ Exposure to strong sunlight should be avoided because of the increased sensitivity of the skin. (Photosensitivity)

Antibiotics:

- ❖ Tetracycline and some other antibiotics orally administered reduce bacterial population and the concentration of the fatty acids in the sebaceous follicle.
- ❖ Topical antibacterial agents generally are ineffective, because acne is not an infection.
- ❖ Erythromycin reduces level of fatty acid of the follicles. It is lipid soluble antibiotics which can penetrate the sebaceous follicle
- ❖ ERYTHROMYCIN: the antibiotic of choice in pregnancy we don't use it so commonly because of the high rate of resistant (clindamycin is better)

We mainly use doxycycline but check for pregnancy and age (you never give it to someone who is younger than 9) +causes gi upset

Management: based on the severity

Comedonal acne:

- ❖ Topical agents are useful when topical retinoids not tolerated
- ❖ Salicylic acid (promotes desquamation)
- ❖ Azelaic acid (antimicrobial, reduces hyperpigmentation)o Glycolic acid
- ❖ Sulfur in OTC (keratolytic)

Mild to moderate inflammatory Acne:

- ❖ Benzoyl peroxide(antimicrobial, anti comedonal, pregnancy risk)
- ❖ Topical antibiotic
- ❖ Combination of both

Moderate to severe Acne:

1- Oral isotretinoin: It is routinely given for 4–6 months only, in a dosage of 0.5–1 mg/kg body weight/day

❖ MOA: Reduces sebaceous gland size/sebum production and regulates cell proliferation and differentiation

❖ Effect last 1 year after cessation (1 pill 2 months in the body)

❖ Only med altering course of A. Vulgaris (complete remission in most cases)

2- Oral antibiotics:

- Tetracycline - TMP-SMX - Minocycline - Erythromycin - Clindamycin

-Doxycycline

❖ Given daily over 4-6 months with taper.

1. **Tetracycline :** Even with long courses, serious side-effects are rare, although candidal vulvovaginitis may force a change to a narrower spectrum antibiotic such as erythromycin , or Antifungal is given along with tetracycline. Both tetracycline and isotretinoin may cause Pseudotumor cerebri (benign intracranial swelling leading to increased intracranial pressure i.e Headache) should not be taken in pregnancy or by children under 12 years as they are deposited in growing bone and developing teeth, causing stained teeth and dental hypoplasia
2. **Minocycline:** liver abnormalities + lupus-like syndrome + pseudotumor cerebri + it may cause pigmentation (rare)
3. **Deoxycycline:** Photosensitivity

^432 teamwork

Oral isotretinoin:

❖ A full blood count,liver function tests and fasting lipid levels should be checked before the treatment and every month after starting the treatment(especially LFTs).

❖ Isotretinoin is highly teratogenic missing limb.

❖ FDA practice rules:

- 2 negative pregnancy tests before treatment Pregnancy test each month
- Pregnancy risk patients must use 2 contraceptive for at least 1 month prior to treatment

❖ Suicidal and depression risk. (it is controversial because acne could cause depression and roaccutane so we don't know which really cause depression in the pt bu that what is written in the book and drugs company said that to protect themselves)

❖ Effects of isotretinoin include a dry skin,dry and inflamed **lips** and eyes, nosebleeds, facial erythema, muscle aches, hyperlipidemia and hair loss bone marrow suppression, hepatotoxicity

Frequently Asked Questions by PTs:

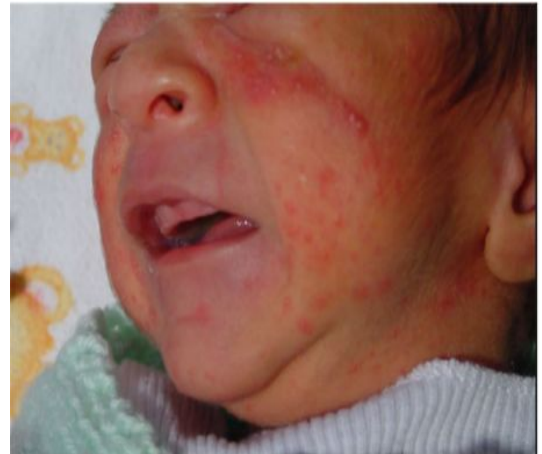
❖ Soaps, detergents remove sebum but do not alter production

❖ Water based cosmetic better than oil based

❖ Diet modification no role in Rx

Acne Related Disorders

The doctor didn't dig deep and mentioned that some types they never see it but it's nice to know -_-



Neonatal Acne:

- First four weeks of life
- Develops a few days after birth
- Facial papules or pustules
- Cases that persist beyond 4 weeks or have an onset after rule out acne cosmetic, acne venenata, drug-induced acne

Usually subsides by itself treatment to Avoid scarring
Treatment depends on severity mild acne treated with topical agents if the treatments fails or there is many lesions then it is moderate and we add systemic treatment if their is scars it is severe and we use isotretinoin .

SAPHO Syndrome:

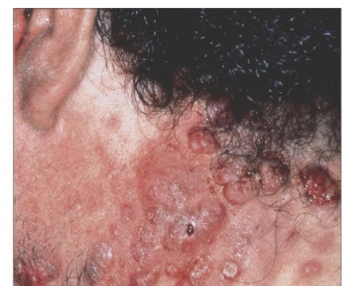
dr said very rare just read about it never seen it but they ask about in the exams

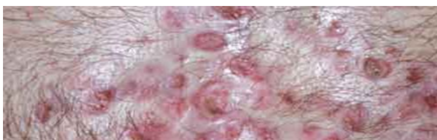
- **S**ynovitis, **A**cne, **P**ustulosis, **H**yperostosis, and **O**steomyelitis = SAPHO Syndrome.
- Acne fulminans, acne conglobata, pustular psoriasis, and palmoplantar pustulosis .
- Chest wall is most site of musculoskeletal complaints.

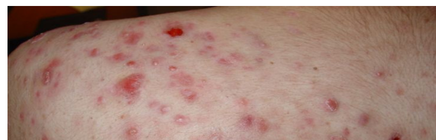
Acne Conglobata:

- Conglobate: shaped in a rounded mass or ball
- Severe form of acne characterized by numerous comedones, large abscesses with sinuses, grouped inflammatory nodules
- Suppuration (pus formation)
- Cysts on forehead, cheeks, and neck
- Occurs most frequently in young men
- Follicular Occlusion Triad: acne conglobata, hiradenitissuppurva, cellulitis of the scalp
- Heals with scarring
- **Treatment:** oral isotretinoin for 5 months (in the beginning, we give them antibiotics to make it better then we give them isotretinoin because it is an irritant and could make it worse in the beginning)

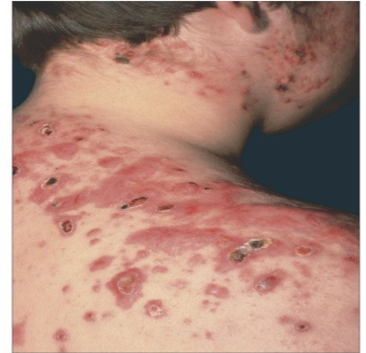
Sometimes in severe cases of acne like in acne conglobata there is a chance that isotretinoin will worsen the symptoms







Acne Fulminans: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- ❖ Rare form of extremely severe cystic acne
- ❖ Teenage boys, chest and back
- ❖ Rapid degeneration of nodules leaving ulceration
- ❖ Fever, myalgia, leukocytosis, arthralgias are common, labs shows high ESR, patients get admitted.
- ❖ **Treatment:** oral steroids, isotretinoin (althouh it can be induced by high inital dose of isotretinoin)

Tropical Acne: in tropical climates didn't talk about it but it is mentioned in the slides

- ❖ Nodular, cystic, and pustular lesions on back, buttocks, and thighs
- ❖ Face is spared
- ❖ Young adult military stationed in tropics

Acne Venenata:didn't talk about it but it is mentioned in the slides

- ❖ Contact with acnegenic chemicals can produce comedones (Chlorinated hydrocarbons, cutting oils, petroleum oil, coal tar)
- ❖ Radiation therapy

Acne Cosmetica: didn't talk about it but it is mentioned in the slides

- ❖ Closed comedones and papulopustules on the chin and cheeks
- ❖ May take months to clear after stopping cosmetic product
- ❖ Pomade Acne, blacks, males, due to greases or oils applied to hair

Acne Detergicans : didn't talk about it but it is mentioned in the slides

- ❖ Patients wash face with comedogenic soaps Closed comedones
- ❖ **Treatment:** wash only once or twice a day with non-comedogenic soap .

Acne Aestivalis :didn't talk about it but it is mentioned in the slides

- ❖ Aka: Mallorca acne
- ❖ Rare, females 25---40 yrs
- ❖ Starts in spring, resolves by fall
- ❖ Small papules on cheeks, neck, upper body
- ❖ Comedones and pustules are sparse or **absent**
- ❖ **Treatment:** Retinoic acid, antibiotics don't help

Acneiform Eruptions:

❖ Originate from skin exposure to various industrial chemicals. Papules and pustules not confined to usual sites of acne vulgaris. Chlorinated hydrocarbons, oils, coal tar.

❖ Oral medications: iodides, bromides, lithium, steroids (steroid acne).

Excoriated Acne:picker's acne didn't talk about it

❖ Girls, minute or trivial primary lesions are made worse by squeezing

❖ Crusts, scarring, and atrophy

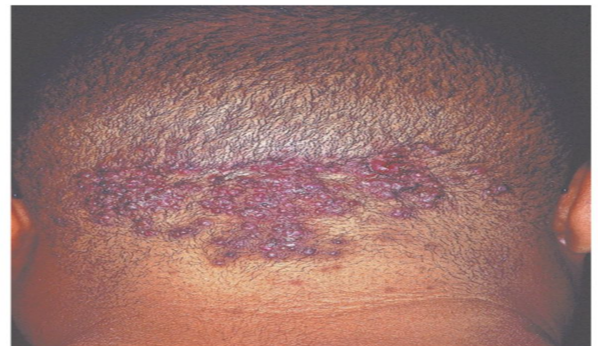
❖ **Treatment:** eliminate magnifying mirror, rule out depression

Gram Negative Folliculitis:didn't talk about it but it is mentioned in the slides

❖ Occurs in patients treated with ANTIBIOTICS for acne over a long-term Enterobactor, Klebsiella, Proteus Anterior nares colonized

❖ **Treatment:**isotretinoin, Augmentin

Acne Keloidalis: it's not actually an acne (**misnomer**)



❖ Folliculitis of the deep levels of the hair follicle that progresses into a perifolliculitis.

❖ Occurs at nuchal area in **blacks** or Asian men Not associated with acne vulgaris

❖ Hypertrophic connective tissue becomes sclerotic, free hairs trapped in the dermis contribute to inflammation .

❖ **Treatment:** intralesional Kenalog

Hidradenitis Suppurativa: it's called hidradenitis suppurativa if it appears in body folds.

It's a misleading name because it is considered by some to be a disorder of apocrine gland (Sweat gland) but In my opinion Acne inversa affects primarily the Pilo Seb. Unit and affect secondarily the sweat gland, hence the correct name Acne inversa rather than Hidradenitis suppurativa is preferred.

❖ Disease of the **apocrine** gland ? (Pilosebaceous unit according to New slides !)??

❖ **Axillae, groin, buttocks, also areola**

❖ Obesity ، smoking and **genetic** tendency to acne (obese and smoker usually they don't need treatment if they stop smoking and reduce weight)

❖ Tender red nodules become fluctuant and painful

❖ Rupture, suppuration, formation of **sinus** tracts

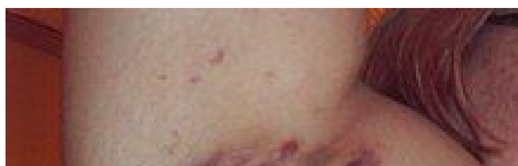
❖ Most frequently axillae of young women

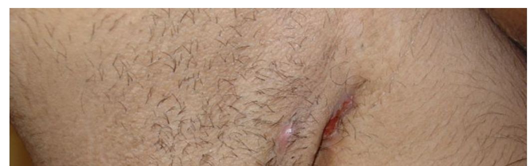
❖ Men usually groin and perianal area

❖ Follicular keratinization with plugging of the apocrine duct; dilation and inflammation

❖ Oral antibiotics, culture S. aureus, gram-negatives

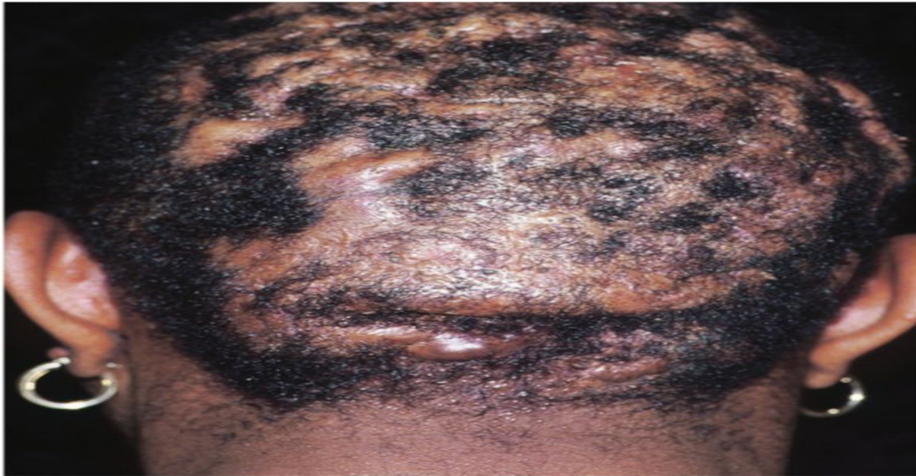
❖ Intralesional steroids, **surgery,** Isotretinoin helpful in some cases





Dissecting cellulitis of the scalp:

- ❖ Uncommon suppurative disease.
- ❖ Nodules suppurate and undermine to form sinuses.
- ❖ Scarring and **alopecia**. (حتى الزراعه صعبة لان فيه سكارنج فايبروسس)
- ❖ Adult black men most common, vertex and occiput.
- ❖ **Treatment** intralesional steroids, isotretinoin, oral abx, surgical incision and drainage.



Pyoderma Faciale:

❖ Post-adolescent girls, reddish cyanotic erythema with **abscesses** and cysts Distinguished from acne by absence of comedones, rapid onset, fulminant course and absence of acne on the back and chest .

❖ **Treatment** oral steroids followed by ISOTRETINOIN

Rosacea It is not in the slides

the doctor mentioned that the slides are an old version and rosacea is **supposed to be covered**

There is a major overlapping between acne and rosacea but the difference that the major lesion is erythema

ROSACEA ICD-10: L71

- A common chronic inflammatory acneiform disorder of the facial pilosebaceous units.
- Coupled with an increased reactivity of capillaries leading to flushing and telangiectasia.
- May result in rubbery thickening of nose, cheeks, forehead, or chin caused by sebaceous hyperplasia, edema, and fibrosis.

Epidemiology:

- 10% of fair-skinned people.
- Age Of Onset From 30t o 50 years
- Affects **Females** predominantly, but rhinophyma occurs mostly in males.
  Rhinophyma is a condition causing development of a large, bulbous nose associated with granulomatous infiltration, commonly due to untreated rosacea.

Factors That Can Cause Rosacea:

- Coffee and Tea
- Stress
- Sun exposure
- alcohol

Rosacea might affect the eyes and lead to a condition called ocular rosacea a condition characterized by hot red dry eyes and could lead to ulcers

Pathogenesis:

-Not known but thought to be related to a vascular related disease
-Also demodex Mites in the skin is though to play a role

Treatment: Same as acne but some with differences

-Mild to moderate > use **metronidazole (treatment of choice in rosacea and it is not used in acne)**
-If it doesn't work use Brimonidine (it cause vasoconstriction but when they stop it there will be a rebound)
-If it doesn't then Systemic antibiotic
-If it doesn't Isotretinoin in very small doses

DDX:

Acne vulgaris
Seborrheic dermatitis
Systemic lupus Erythematosus "photosensitivity" (because of the erythema in the face but there is no joint pain in rosacea)