Dermatology. **Lecture: 3**
Dr. Intisar Ahmed **Stage: 5**

Acne & Rosacea

Acne:

*Is a disorder of pilosebaceous apparatus characterized by comedones, papules, pustules, cysts and scars.

Prevalence:

* Nearly all teenagers have some acne.
* equall sexes.
* starting between 12 and 14 yrs, tending to be earlier in females.
* The peak age for severity in females is 16-17 and in males 17-19 years.

Cause:

*Many factors combine to cause acne:

1. Sebum over-production.

2. Hormonal:

*Androgens (from testes, ovaries, adrenals and sebaceous glands themselves) are the main stimulants.

*Increased target organ sensitivity ➞ Sebaceous glands respond excessively to normal levels of hormones.

*low level of sex hormone binding globulin ➞ slightly raised free testosterone.

3. Poral occlusion:

*Genetic and environmental factors (e.g. some cosmetics) cause the epithelium to overgrow the follicular surface.

*Follicles then retain sebum that has an increased concentration of bacteria and free fatty acids.

*Rupture of these follicles is associated with intense inflammation and tissue damage.

4. Increased bacterial colonization:

*Propionibacterium acnes, a normal skin commensal, plays a pathogenic part.

*It colonizes the pilosebaceous ducts, breaks down triglycerides releasing free fatty acids, that induces the ductal epithelium to secrete pro-inflammatory cytokines.

*Activated neutrophils release lysosomal enzymes which lead to follicular rupture.

*The release of follicular content results in a foreign body reaction which increase the inflammatory reaction.

5. Genetic:

6. Diet: dairy products (particularly milk) and high glycaemic load may be associated with increased risk and severity of acne.

Presentation:

Common type:

*Lesions are confined to the face, shoulders, upper chest and back.
*Seborrhoea is often present.
*Open comedones (**blackheads**), because of the plugging by keratin and sebum of the pilosebaceous orifice.
*closed comedones (**whiteheads**), caused by overgrowth of the follicle openings by surrounding epithelium.
*Inflammatory papules, nodules and cysts occur with one or two types of lesion predominating.
*Depressed or hypertrophic scarring and post-inflammatory hyperpigmentation can follow.
*hyperpigmentation usually transient, but can persist, particularly in dark skin.
*Psychological depression is common in persistent acne.

Conglobate type: is name given to a severe form of acne with all of the above features as well as abscesses or cysts with intercommunicating sinuses that contain thick serosanguinous fluid or pus. On resolution, it leaves deeply pitted or hypertrophic scars, sometimes joined by keloidal bridges.

Neonatal acne:

*Occurs in more than 20% of healthy newborn babies between **2 weeks to 3 months** of age.
*It may follow transplacental stimulation of a child's sebaceous glands by maternal androgens.

Infantile acne:

*This rare type of acne is present at **3-6 months** of age and typically resolves in 12 months.
*It is more common in males and may last up to 3 years.
*morphology is like that of common acne.
*Maternal hormones have a minor role at this age.
*The immature infantile adrenal gland produces elevated dehydroepiandrosterone (DHEA) and delayed maturation of the gonadal feedback system results in increased

levels of luteinizing hormone (LH), follicle-stimulating hormone (FSH) and testosterone.

Late onset:

*mainly in women and is often limited to the chin and jawline .
*Nodular and cystic lesions predominate.
*It is stubborn and persistent.

Acne fulminans:

*Rare variant in which conglobate acne is accompanied by fever, joint pains and a high (ESR).

Tropical type:

*Heat and humidity are responsible.
*Affects Caucasoids with a tendency to acne.
*Mainly on the trunk and may be conglobate.
*Sweat causes follicular occlusion by causing the perifollicular epidermis to swell.

Excoriated type:

*Most common in young girls.
*Obsessional picking or rubbing leaves discrete denuded areas.

Exogenous type:

*Tars, chlorinated hydrocarbons, oils and oily cosmetics may induce comedone formation or precipitate inflammation around vellous hair follicles.
*Distribution is odd and comedones predominate.

Drug-induced:

*Corticosteroids, androgenic and anabolic steroids, gonadotrophins, oral contraceptives, lithium, iodides, bromides, antituberculosis and anticonvulsant therapy can all cause an acneiform rash.
*Suspicion should be raised when acne, dominated by monomorphous papulopustules rather than comedones, appears suddenly in a non-teenager and coincides with the prescription of a drug known to cause acneiform lesions.

Follicular occlusion tetrad:

*Severe nodulocystic acne, dissecting cellulitis of the scalp, suppurative hidradenitis and pilonidal cysts.

Polycystic ovarian syndrome:

*In obese females with oligomenorrhoea or secondary amenorrhoea or infertility, Glucose intolerance, dyslipidaemia, hirsutism and hypertension may be other features.

Congenital adrenal hyperplasia:

*Hyperpigmentation, ambiguous genitalia, history of salt-wasting in childhood and aJewish background are all clues to this rare diagnosis caused by 21-hydroxylase deficiency.

Androgen-secreting tumours:

*These cause the rapid onset of virilization (clitoromegaly, deepening of voice, breast atrophy, male pattern balding and hirsutism) as well as acne.

Course:

*Clears by the age of **23–25 years in 90%** of patients, but some **5%** of women and **1%** of men still need treatment in their thirties or even forties.

Investigations:

*None are usually necessary.
*Cultures: occasionally to exclude a pyogenic infection, an anaerobic infection or Gram-negative folliculitis.
*Hormonal assay, plasma levels of total and free testosterone, sex hormone binding globulin, LH, FSH, DHEA sulfate, androstenedione, 17hydroxyprogesterone, urinary free cortisol and.
*ultrasound examination or computed tomography scan of the ovaries and adrenals.

Differential diagnosis:

1.Rosacea: comedones are absent, the papules and pustules occur only on the face.
2.Pyogenic folliculitis can be excluded by culture.
3.Hidradenitis suppurativa.
4.Pseudofolliculitis barbae.
5.keloidalis nuchae.

Treatment:

Topical treatment:

1. Antibacterial agent: benzoyl peroxide.

*Is not associated with development of bacterial resistance.
*For inflammatory lesions.
*Applied at night initially, then twice daily.
*Start with a 2.5 or 5% preparation, moving up to 10%.
*S.E: dryness, irritation and bleaches coloured materials.

2.Vitamin A (retinol) analogues: tretinoin, isotretinoin, adapalene, tazarotene.

*For comedonal acne.
*Normalize follicular keratinization, reduce sebum production.
*Applied every 2–3 nights, increasing frequency after several weeks as tolerated.
*S.E: irritation and photosensitivity.
*Patients with oily skin may prefer gels which are drying, sensitive skin prefer emollient creams.
*Eczema is a contraindication to their use.
*Topical retinoids should **not** be prescribed for pregnant woman with acne.

3.Azelaic acid.

*Is bacteriocidal for P. acnes.
*Anti -inflammatory, inhibit formation of comedones by reducing the proliferation of keratinocytes.
*lighten post-inflammatory hyperpigmentation.
*Applied twice daily.

4.Topical antibiotics: clindamycin, erythromycin and sulfacetamide.

*Antibacterial resistance of P. acnes is a growing problem.
*Combining antibiotics with benzoyl peroxide reduces P. acnes numbers and the likelihood of resistant strains emerging.

5.Topical dapsone 5% gel.

*Is a newer option for treating acne.
*It is safe in patients with a deficiency in glucose-6phosphate dehydrogenase.

6. Cosmetic camouflage.

*Help some patients, especially females, whose scarring is unsightly.
*Cover-ups also obscure post-inflammatory pigmentation.

Systemic treatment:

1. Antibiotics.

*The prevalence of antibiotic-resistant P.acnes is rising.
*Advantage: decrease P. acnes numbers, direct anti-inflammatory effect.
*Should be used in combination with topical benzoyl peroxide or retinoids to limit colonization by antibiotic resistant bacteria.

A. Tetracyclines.

*Starting dosage 500 mg twice daily, but up to 1.5 g/day may be needed in resistant cases for 3 months.
*Taken on an empty stomach, 1 hour before meals or 4 hours after food.
*Tapering with maintenance dosage 250–500 mg/day with clinical improvement.
*S.E: candidal vulvovaginitis.
*C.I: pregnancy, children under 9 years as they are deposited in growing bone and developing teeth, causing stained teeth and dental hypoplasia

B. Doxycycline.

*100 mg once or twice daily.
*S.E: Phototoxic skin reactions.

C. Erythromycin.

*Used in women who might become pregnant.
*S.E: nausea and the widespread development of resistant Propionibacteria, which leads to therapeutic failure.

D. Trimethoprim.

*Is used with or without sulfamethoxazole third-line when a tetracycline and erythromycin have not helped.
*White blood cell counts should be monitored.

E. Ampicillin is another alternative.

2. Hormonal treatment.

*Very effective for female, even with normal level of serum androgen.
*Indicated for: Adult women with persistent inflammatory papules involving the chin and jawline who report flares around their menstrual cycle.
*Combined antiandrogen–oestrogen treatment (Dianette), is available.
*S.E: venous thrombo-embolism.
*Course not more than 3 months after the acne has cleared.

*Not for males.
*They reduce ovarian androgen synthesis and, by increasing sex hormone binding globulin, reduce free testosterone levels and sebum production.

3. Spironolactone.
*Blocks the androgen receptor and inhibits 5α-reductase, thus reducing sebum production.
*May be added to the OCP after 3 months if there has been an inadequate response.
*Dose is 25-100 mg/day with food.
*SE: hyperkalaemia.
*Pregnancy should be avoided as there is a risk of causing abnormalities of the fetal male genitalia.

4. Isotretinoin.
*Is an oral retinoid, inhibits sebum excretion, the growth of P. acnes and acute inflammatory processes.
*Indicated for severe nodulocystic acne, unresponsive to the measures outlined above.
*Given for 4–6 months, in a dosage of 0.5–2 mg/kg body weight/day.
* Flare on initiation of isotretinoin may occur, so a lower starting dose and concomitant administration of prednisone may prevent it.
*The goal is to achieve a total cumulative dose of 120–150 mg/kg to reduce the risk of relapse.
*A full blood count, liver function tests and fasting lipid levels should be checked before the start of the course and then monthly.
*Highly teratogenic so tests for pregnancy before starting and then monhtly and two effective method of contraception must be used **1 month before starting isotretinoin, throughout treatment and for <u>1 month</u> after stopping it**.
*S.E: depression, dry skin, dry and inflamed lips and eyes, nosebleeds, facial erythema, muscle aches, hyperlipidaemia, hair loss, changes in night-time vision, pseudotumor cerebri, pancreatitis, hepatotoxicity, blood dyscrasias, hyperostosis and hearing loss.
*Avoid: pregnancy, breastfeeding, Giving blood, taking vitamin A, cosmetic procedures, excessive natural or artificial UVR.

<u>Physical treatment:</u>
1. dermabrasion.
2. Chemical peels.
3. extraction with an 18 gauge needle.

4. Cysts can be incised and drained with or without a local anasthesia.
5. Intralesional injections of 0.1 mL triamcinolone acetonide (2.5–10 mg/mL) hasten the resolution of stubborn cysts, but can leave atrophy.

Treatment with light and laser therapies:

*Light and laser treatments reduce acne by photoactivation of porphyrins naturally produced by P. acnes, leading to reduction in the growth of P.acnes.

Rosacea:

*Affects face of adults, usually women.
*Peak incidence is in the thirties and forties.
*More common in fair-skinned individuals.
*It may coexist with acne but is distinct from it.

Cause:

*Unknown.
*Both genetic and environmental factors seem to have a role.
*Rosacea is often seen in those who flush easily inresponse to warmth, sunlight, spicy food, alcohol and embarrassment.

Presentation

*The cheeks, nose, centre of forehead and chin are most commonly affected.
*Peri-orbital and peri-oral areas are spared.
*Intermittent flushing is followed by a fixed erythema and telangiectases.
*Discrete domed inflamed papules, papulo-pustules and, rarely, plaques or nodules develop.
*Rosacea, **unlike** acne, has no comedones or seborrhoea.
*Its course is prolonged, with exacerbations and remissions.
*Rosacea is classified into four major subtypes.

1. **Erythematotelangiectatic rosacea:**

Is characterized predominantly by its vascular features of flushing and fixed erythema, with or without telangiectasia.

2. **Papulopustular rosacea**:

Consists of fixed erythema with inflammatory papules and/or pustules.

3. **Phymatous rosacea**:

More common in males, is a result of overgrowth of sebaceous glands and connective tissue. It predominately affects the nose (rhinophyma) but can develop in extra nasal sites such as chin, forehead, cheeks or ears.

4. **Ocular rosacea:**

Is frequently undiagnosed but quite common, with up to 50% of rosacea patients affected. Complications include blepharitis, conjunctivitis and, occasionally, keratitis.

Differential diagnosis:

1.Acne: rosacea differs from it by
-Its background of erythema and telangiectases.
-Absence of comedones.
-The distribution of the lesions it affects the central face but not trunk.
-Appears after adolescence.
2.Sun-damaged skin.
3.Seborrhoeic eczema.
4.Peri-oral dermatitis.
5.Systemic lupus erythematosus.

Treatment:

Treatment is best directed toward the subtype

1.Erythromatotelangiectatic type:
-Topical metronidazole.
-Topical azeleic acid.
-Decrease flushing.
-Cover-up makeup.
-Colour-correcting gels (green).
-Pulsed dye laser and intense pulsed light.

2.Papulospustular type:
-Combination of topical agent with oral antibiotic.
-Topical metronidazole.
-Topical azeleic acid.
-Topical sulfacetamide/sulfur.
-Oral tetracyclines.

3.Phymatous type:
-Ablative lasers.
-Electrosurgery.

4.Ocular type:
-Oral doxycycline.
-Artifical tears.

-Lid cleansing.

*Antiobiotics are used for their anti-inflammatory properties rather than antimicrobial effects.
*Courses should last for at least 10 weeks.
*Rarely, systemic metronidazole or isotretinoin is needed for stubborn rosacea.
*Rosacea and topical steroids go badly together.
*Sunscreens help rosacea if sun exposure is an aggravating factor, but changes in diet or drinking habits are seldom of value.