Hair and nails disorders

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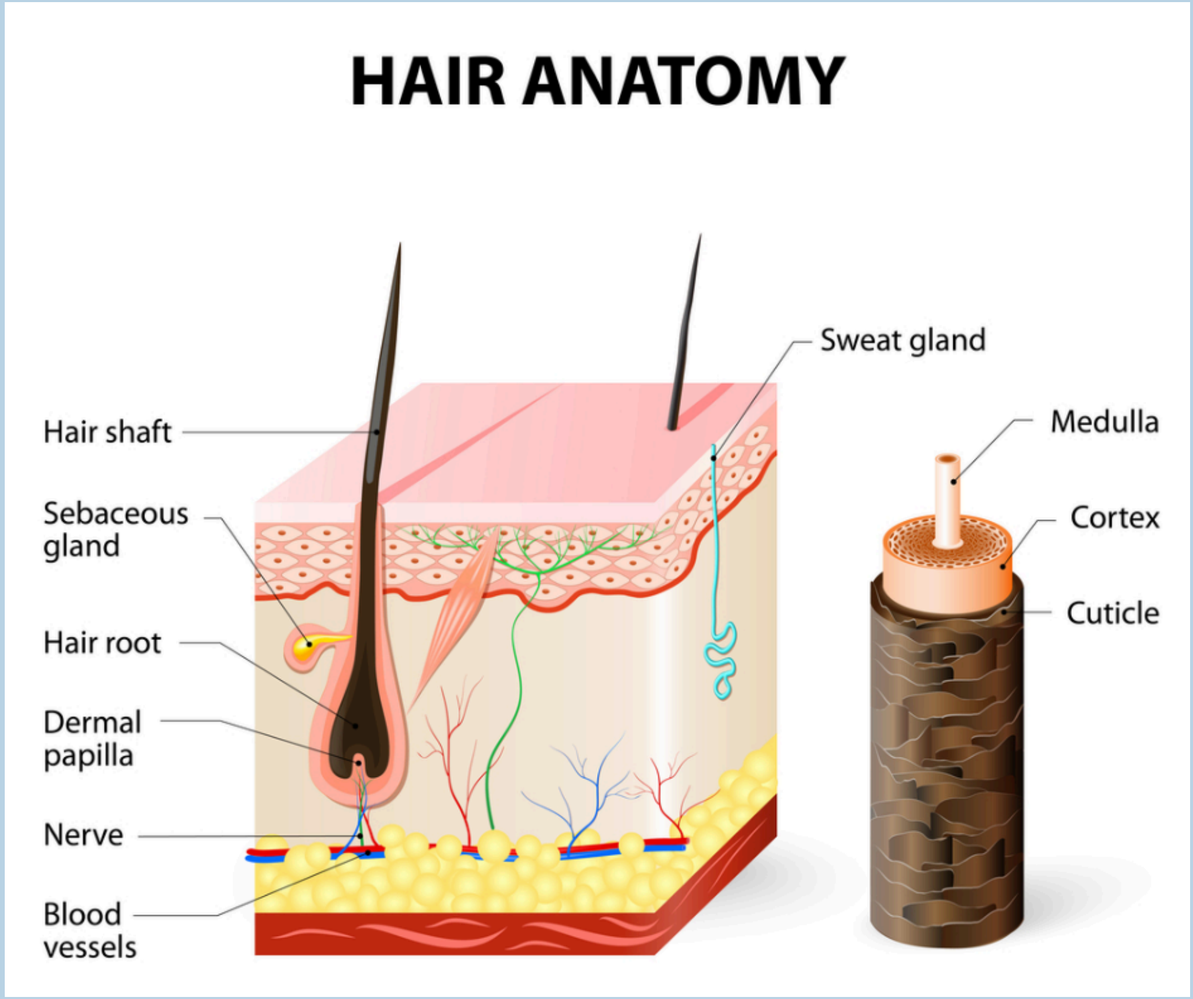
Introduction

- Human hair plays a significant role in the self-image of individuals and the image they present to the world.
- Healthy hair conveys a sense of well-being, vitality and youthfulness.

- Excess hair growth in females, particularly in prominent sites such as the face, is not only an embarrassment but may indicate underlying systemic disease

Basic Hair structure

Hair is a filamentous structure made of hard keratin, produced by the hair matrix of the hair follicle located in the dermis, and is a modified epidermal structure.



❖**Hair is composed of:**

1. Shaft (Exposed part above the skin):
Composed of Medulla, Cortex and Cuticle

2. Hair follicle (Under the skin):
Composed of Hair bulb (Dermal papilla and the Matrix) and the root Sheath

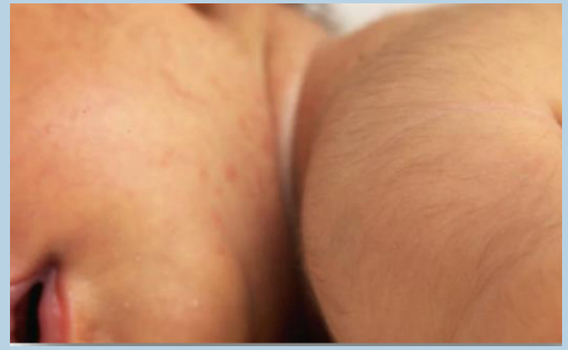
❖**Accessory structures of Hair:**

1. Arrector pilli muscle.
2. Sebaceous glands.
3. Hair root plexus

Types of hair

❖**Lanugo hair:**

o Very thin, soft, usually unpigmented and long hair, produced by fetal hair cells and is usually shed before birth.



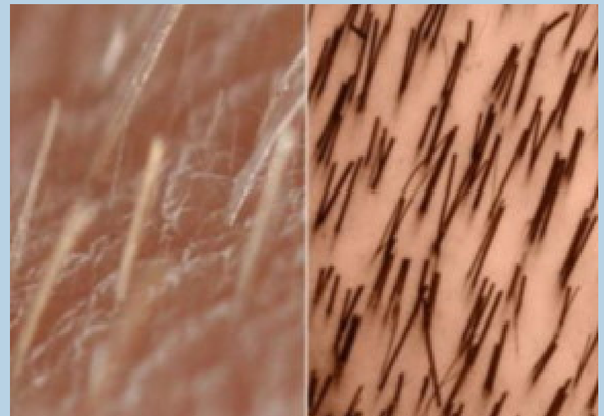
❖**Vellus hair:**

o Short, thin, light colored and barely noticeable hair that develops on most of a person's body childhood sparing the palms and soles.

❖**Terminal hair:**

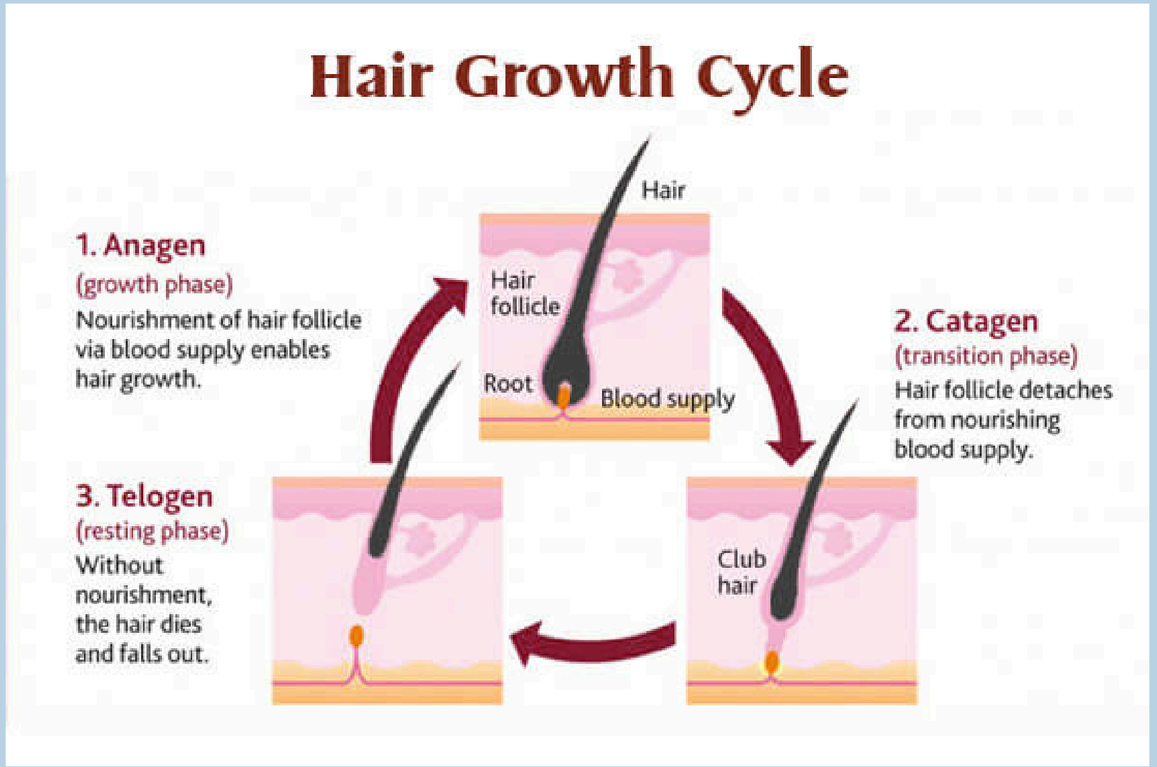
o Thick, long and dark, it is limited to the eyebrows, eyelashes and scalp until puberty.

o During puberty, the increase in androgenic hormone levels causes vellus hair to be replaced with terminal hair in certain parts of the human body, also secondary terminal hair develops in the axillae, pubic region and central chest in men in response to androgens.



Hair cycle

- Hair follicles undergo a repetitive sequence of growth and rest known as the hair cycle
- the timing of the phases varies due to individual's age and stage of development, nutritional habits, or environmental alterations



o **Anagen (growing phase)**:

The active growth phase, which typically lasts **1000 days** depending on predetermined genetic factors, it determines the length of our hair

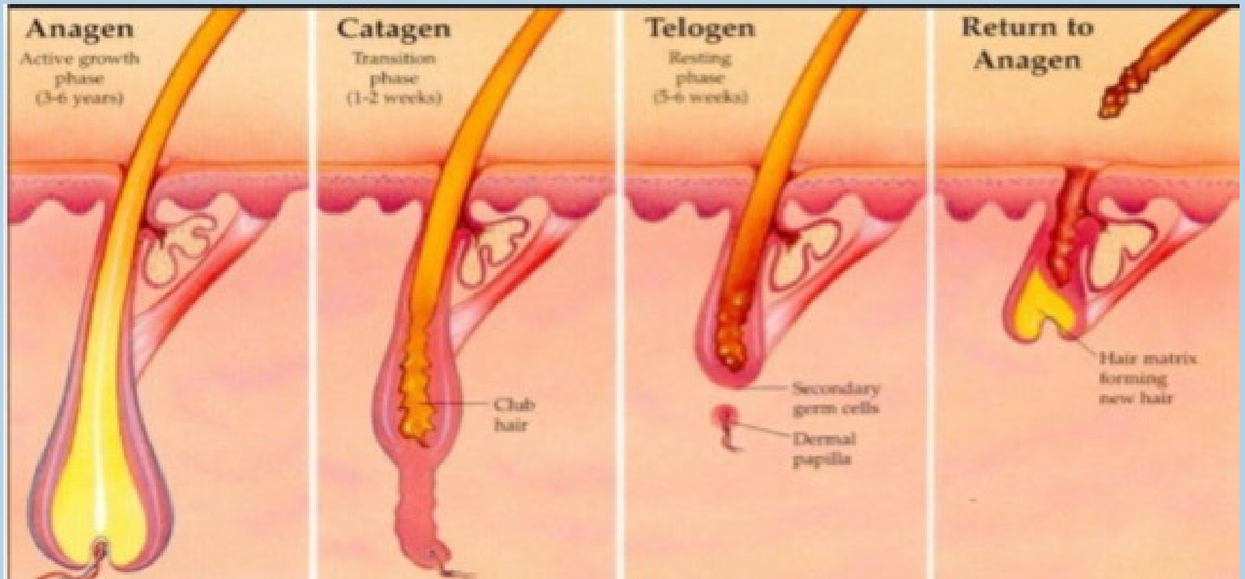
o **Catagen (transition phase):**

The short growth arrest phase, of approximately **10 days**; due to cessation of protein and pigment production and regression of the follicle due to detachment from the dermal papilla

o **Telogen (resting phase):**

The resting phase, lasting approximately **100 days** irrespective of location,, Whilst the old hair is resting, a new hair begins the growth phase.

o **Exogen (new hair phase):** This is part of the resting phase where the old hair sheds and a new hair continues to grow



❖ The ratio of anagen to telogen hairs is 9:1 reflecting the fact that only a few hairs at a time are in catagen phase. On average, 100 hairs are shed per day although seasonal variation does occur.

❖**Active** phase of hair growth is **anagen**
❖The **transition** state of hair is **catagen**
❖The **resting** state of hair is **telogen**
❖The **new hair** phase of hair is **exogen**

Hair growth rate 1 cm / month

Hair loss

Hair loss or **alopecia** can be divided into **scarring** and **non-scarring**

types depending on the underlying pathological process, and these can then be further categorized according to **distribution, either diffuse or localized.**

Classification of alopecia

Localized (Patchy)

- Non-scarring
  - Tinea capitis
  - Alopecia areata
  - Androgenetic alopecia
  - Traumatic (trichotillomania, traction, cosmetic)
  - Syphilis
- Scarring
  - Idiopathic
  - Developmental defects
  - Discoid lupus erythematosus
  - Herpes zoster
  - Pseudopelade
  - Kerion

Diffuse

1. Androgenetic alopecia
2. Telogen effluvium
3. Metabolic
4. Hypothyroidism
5. Hyperthyroidism
6. Hypopituitarism
7. Diabetes mellitus
8. HIV disease
9. Nutritional deficiency
10. Liver disease
11. Post-partum
12. Alopecia areata
13. Syphilis
14. Discoid lupus erythematosus
15. Radiotherapy
16. Folliculitis decalvans
17. Lichen planus pilaris

1) Androgenetic alopecia

Androgenetic alopecia (AGA) is synonymous with male pattern baldness and is the most prevalent form of hair loss, affecting about 50% of Caucasian males to some extent by the age of 50.

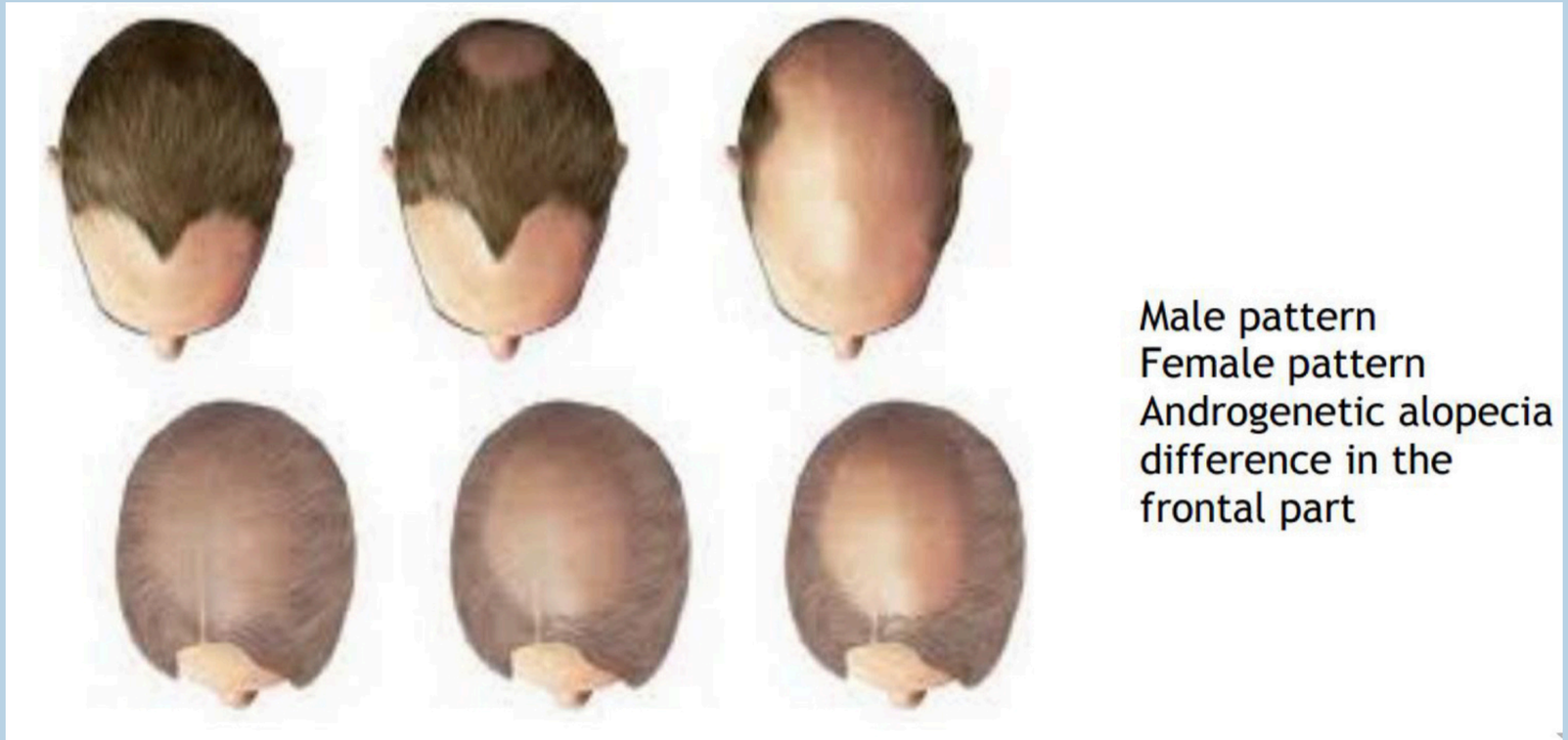
- It also affects a significant number of women and is synonymous with female pattern hair loss (FPHL)

❖Androgen dependent, non-scaring

❖The most common cause of hair loss in both males and females

❖It affects both men and women in a different specific pattern of hair loss of each sex

- It causes hair loss over the temples (fronto-temporal recession) or vertex in men and may progress to leave a horse-shoe distribution of hair over the ears and occipital scalp.
  - Women experience thinning over the central scalp. In contrast to men, there is usually preservation of the frontal margin.
  - Over time, the follicles become smaller, producing shorter and finer hairs.



- some premenopausal women, there may be other signs

of androgenisation suggesting gonadal (e.g. polycystic ovarian syndrome) or rarely adrenal disorders and elevated circulating androgens maybe present.

- The diagnosis particularly in men is straightforward

Treatment of androgenetic alopecia

***Currently there is no cure for AGA and few treatments have been shown to be effective.
Two drugs which are currently licensed to promote hair growth in men with AGA are :

- o Minoxidil lotion or cream (first line treatment)
- o Oral Finasteride

• Minoxidil provides improvement in around 60% of men after use for at least 3–6 months. Benefit may be sustained from continued use, but is lost
• Finasteride is a 5ά-reductase inhibitor and reduces circulating DHT levels.
Daily dosing with 1mg orally may slow hair loss and improve growth.

There is a small increased incidence of sexual dysfunction, for example, impotence in men.

Treatment of female pattern :

- **Minoxidil**
- **Anti-androgens such as spironolactone, flutamide and cyproterone acetate are most useful in women with hyperandrogenism,**
- **Finasteride may also be beneficial**

- **Hair transplantation is an option where medical therapy fails.**

2) Alopecia Areata

AA is an organ-specific autoimmune disease, which leads to nonscarring alopecia.

• It affects 0.15% of the population and can affect any hair-bearing part of the body.

The age of onset is usually in the first two decades. But can occur at any age .

The course of AA is difficult to predict.

Types of Alopecia Areata

01. Alopecia areata (patchy)

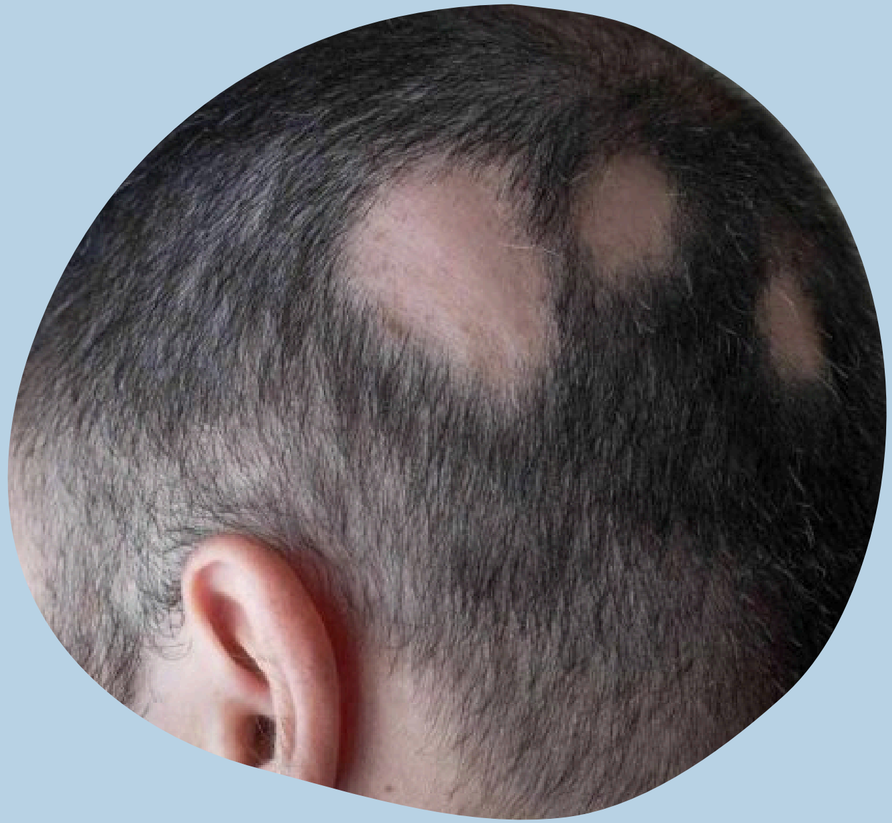
02. Alopecia Totalis

03. Alopecia Universalis

04. Alopecia ophiasis

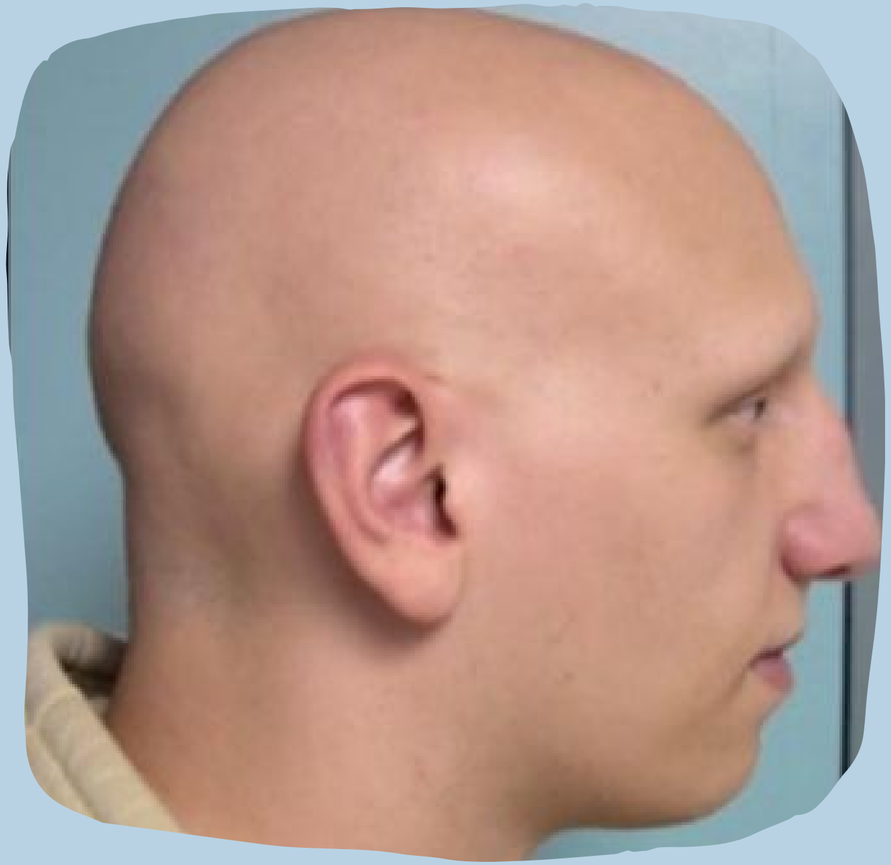
Alopecia Areata (patchy)

Most common type smooth round or oval patches of nonscarring hair loss on the scalp.



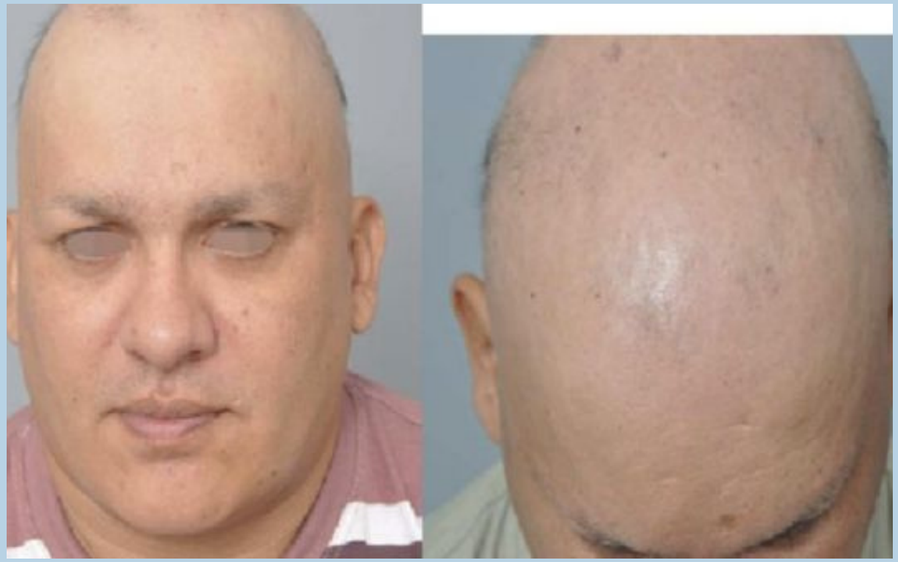
Alopecia totalis

complete or near-complete loss of all hair on the scalp.



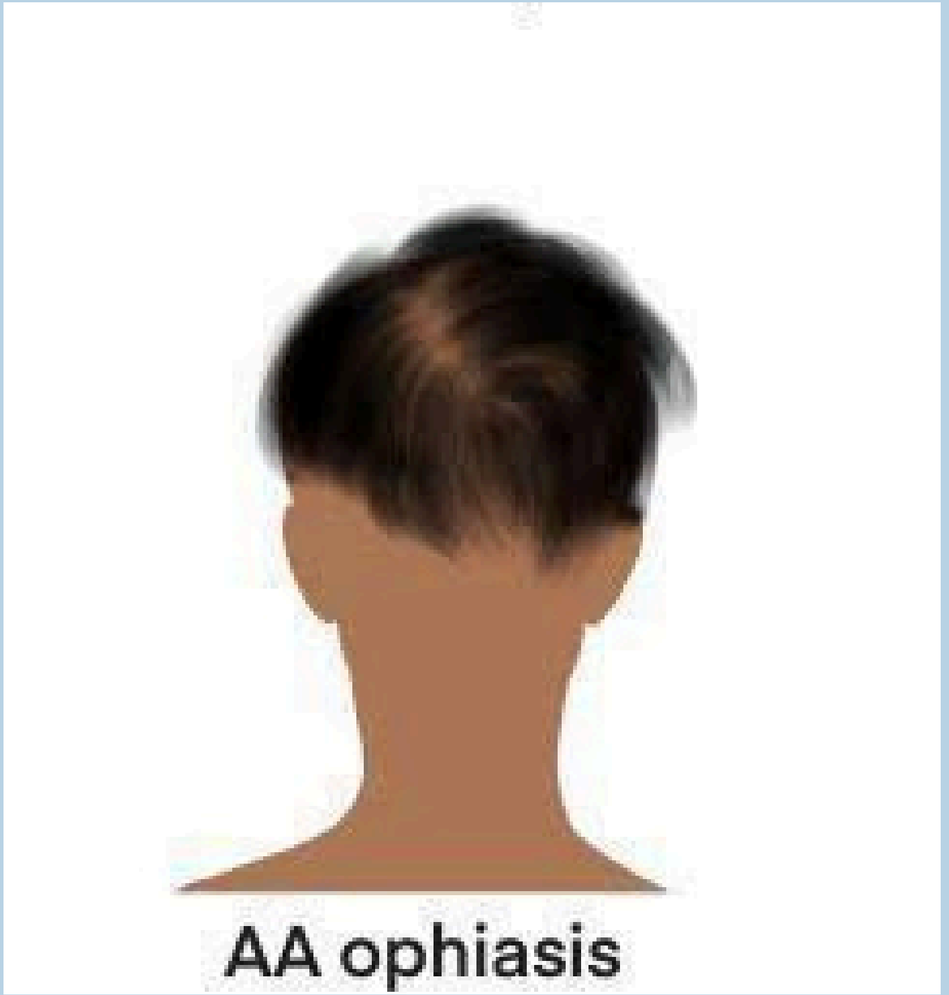
Alopecia Universalis

Add a subheathe most extensive form, resulting in the total or near-total loss of hair from the scalp, face, and all other hair-covered surfaces of the body



Alopecia ophiasis

Hair is lost in a band-like pattern along the sides and back of the scalp, around the circumference of the head



clinical signs

- Exclamation mark hairs, when present, are diagnostic of AA.

These characteristic hairs break at their distal point as they taper and lose pigment proximally, giving them the appearance of an exclamation mark and occur at the periphery of patches of alopecia.

- Nail abnormalities, predominantly pitting or roughening, may occur inassociation with this condition.

- Other organ-specific autoimmune disorders such as vitiligo and thyroiditis are occasionally associated with AA.
- Investigation of associated diseases is usually indicated if symptomatic.

- Poor prognostic markers include:

  - childhood onset of disease
  - atopy
  - ophiasis (band of alopecia in occipital region)
  - nail dystrophy
  - family history of other autoimmune disorders
  - presence of autoantibodies.

Differentialdiagnosis:

Trichotillomania
traction alopecia
telogen effluvium
Androgenic alopecia (AGA)
tinea capitis

should all be excluded by clinical examination, appropriate mycology and skin biopsy where there is diagnostic difficulty

- In AA, the hair follicle is not injured and maintains the potential to regrow hair should the disease go into remission.

- There is, however, no cure for AA and no universally proven treatment to stimulate hair regrowth and sustain remission.

- It is unclear if any of the treatment options available alter the course of the disease.

- Treatment is therefore guided by the extent of the disease and the age of the person being treated.

Current treatments include the following:

- Topical/intralesional corticosteroids.
- Potent topical corticosteroids can be used on the scalp for 2–3 months on localised patches of alopecia.
- Intradermal injection of triamcinolone diluted with local anaesthetic can be used. there is a risk of causing atrophy.
- Systemic immunosuppression. This includes short-term systemic corticosteroids and oral psoralens with exposure to ultraviolet light A (PUVA).
- Contact sensitisation using either irritants (dithranol or retinoids) or allergens (diphencyprone).
- Topical minoxidil (also used in combination with corticosteroids).

Telogen effluvium

However, following a number of stimuli the majority of hair follicles may enter the resting phase (telogen) at the same time (synch ronously) resulting in diffuse shedding approximately 2 months after the triggering event, often described as the hair ‘falling out by the roots’.

- This is usually an acute self-limiting phenomenon, usually resolving within 6months; however, chronic telogen effluvium may occur.

Symptoms & signs of Telogen Effluvium

Noticeable hair loss during washing or combing hair

Hair loss that is temporary

A generalised hair thinning

Losing hair that has white bulb & lack of shiny sheath

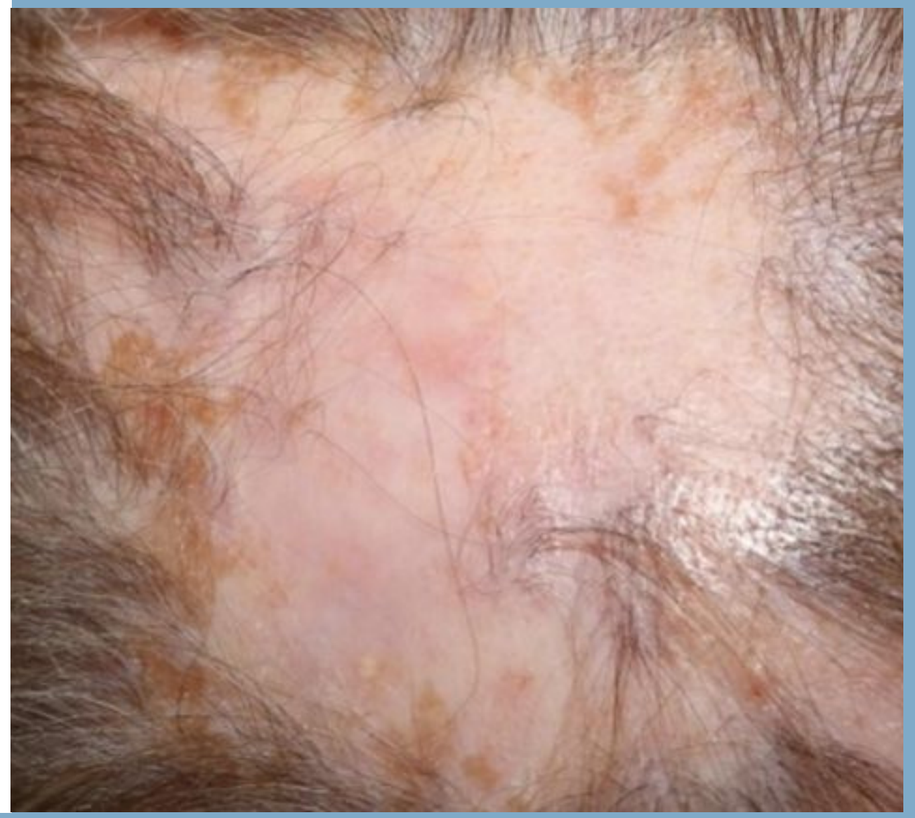
Dry, lusterless hair that are sparse & easily pluckable

Hair colour changes from dark to brown or red, brown to blond

TE : Causes

- **Endocrine**
  - Hypo/hyperthyroidism
  - Post-partum
  - Peri/post-menopausal
- **Nutritional**
  - Biotin deficiency
  - Iron deficiency
  - Kwashiokor/marasmus
  - Zinc deficiency
  - Essential FA deficiency
- **Stress**
  - Anaemia
  - Surgery
  - Systemic illness
  - Psychological stress
  - Pregnancy/ abortion
  - Severe weight loss
- **Drugs**

Triggers



Excessive hair

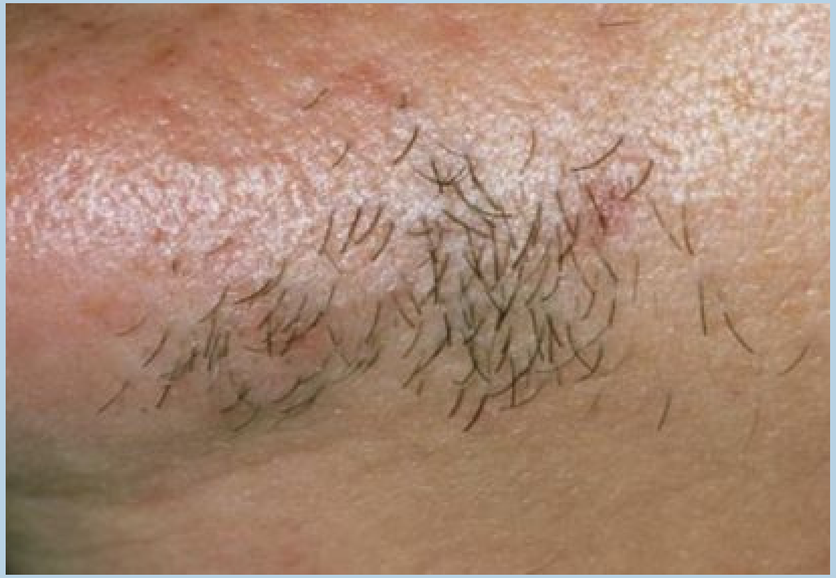
Two patterns of hair overgrowth are recognized: hirsutism & hypertrichosis

Hirsutism

- This is defined as increased growth of the terminal hairs in androgensensitive areas such as the beard and moustache regions in females.

• The assessment of the patient with hirsutism must include a general examination to identify an underlying endocrine abnormality, particularly if there is a relatively short history associated with amenorrhoea and signs of virilisation.

• Features suggestive of virilisation in addition to hirsutism include deepening of the voice, increased muscle bulk and cliteromegaly, an extremely sensitive sign.



Causes of hirsutism

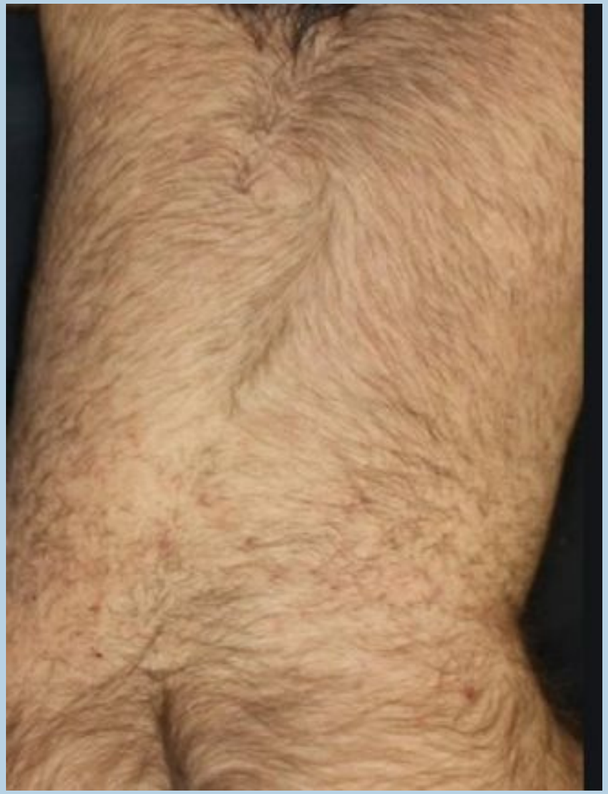
- Excessive production of androgens by the ovaries (polycystic ovary syndrome, tumor)
- Excessive sensitivity of hair follicles to androgens (genetic)
- Excessive production of androgens by the adrenal glands (non-classical adrenal hyperplasia [NCAH])
- Insulin resistance
- Hyperandrogenism, insulin resistance, acanthosis nigricans (HAIR-AN syndrome)
- Excessive production of cortisol by the adrenal glands (Cushing syndrome)
- Menopause
- Medications

- Treatments include suppression of androgens, peripheral androgen blockade and mechanical or cosmetic treatment.
- The use of eflornithine cream is a useful new adjuvant licensed for facial hirsutism that inhibits ornithine decarboxylase, an enzyme involved in controlling hair growth and proliferation. It is most effective when combined with local cosmetic or depilatory treatments, including laser hair removal.

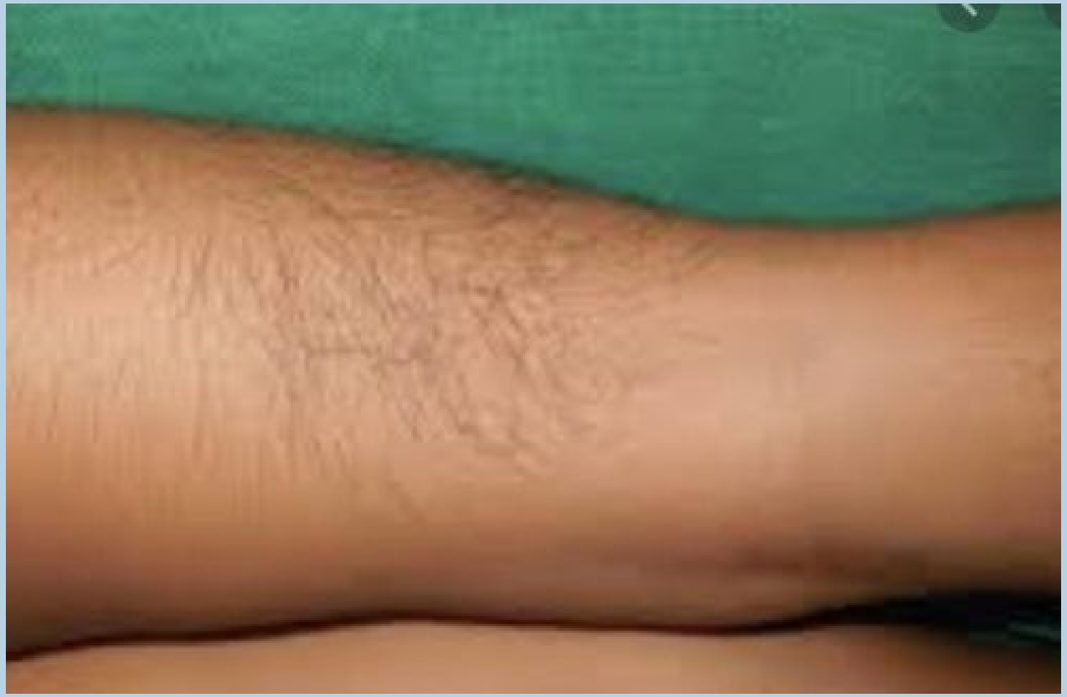
Hypertrichosis

- This describes the excessive growth of hair in any part of the body
  - and may be localised (e.g. Becker’s naevus) or generalised
- Causes may be congenital or acquired; important systemic diseases associated hypertrichosis include hyperthyroidism, porphyria and anorexia nervosa.
- Treatment is directed at the underlying cause and stopping any
  - implicated drug, where possible.
- Symptomatic approaches include depilation using creams, shaving
  - and waxing.

Generalized acquired hypertrichosis may be associated with: porphyria cutanea tarda, malnutrition, malignancy, drugs (ciclosporin, androgen steroids, minoxidil)



Localised acquired hypertrichosis may be associated with:Increased vascularity, Repetitive rubbing or scratching , Application of plaster cast (temporary), Repeated application of minoxidil, potent topical steroids, iodine, psoralen).

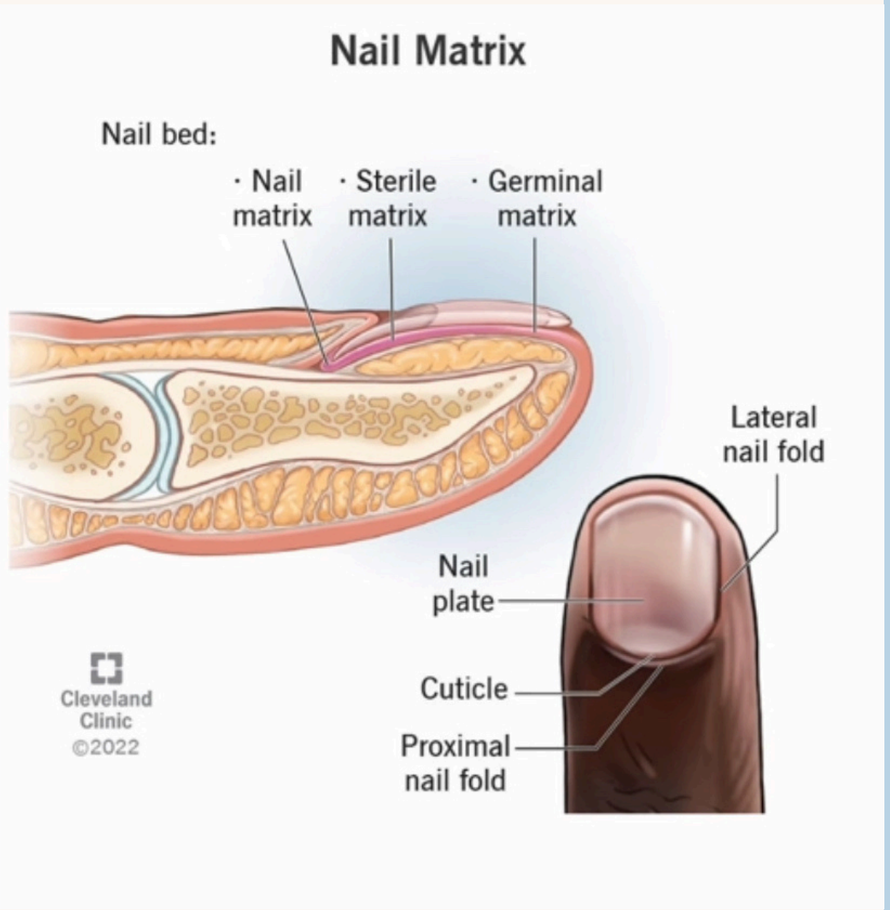


Nail disorders

Nail

The human nail is an ectodermal structure, the nail consists of the nail plate, produced by the nail matrix, and supported by the nail bed and grows in a proximal-to-distal direction, with an average growth rate of about 3 mm per month in fingernails and 1 mm per month in toenails.

- Two main functions: protects the digits from trauma and aids in sensation, increases dexterity (like picking up a coin)
- Nail unit made up of a hard nail plate (keratin), resting on a nail bed containing a matrix (from which the plate grows), skin surrounding form the nail folds



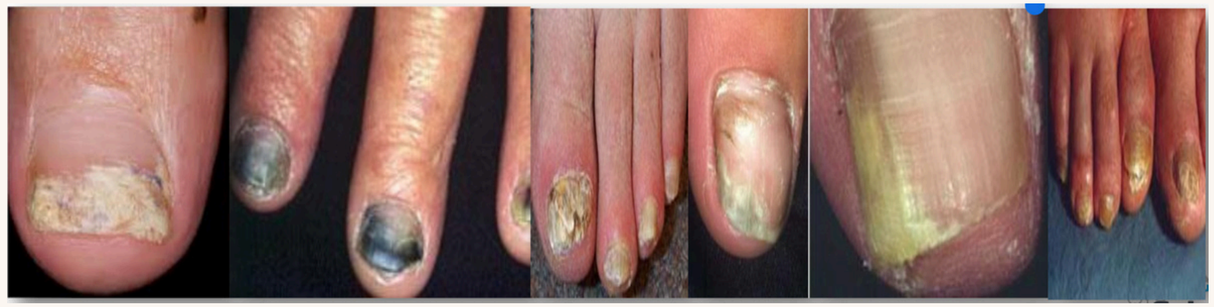
1)Leukonychia spots

- Lesion description: Small white spots are groups of whitish nail cells trapped inside the nail plate.
- Underlying cause: Minor trauma to the Matrix.

Pseudoleukonychia: surface layers develop a whitish flaky appearance due to a lack of moisture in the nail plate sometimes caused by picking off or removal of nail polish

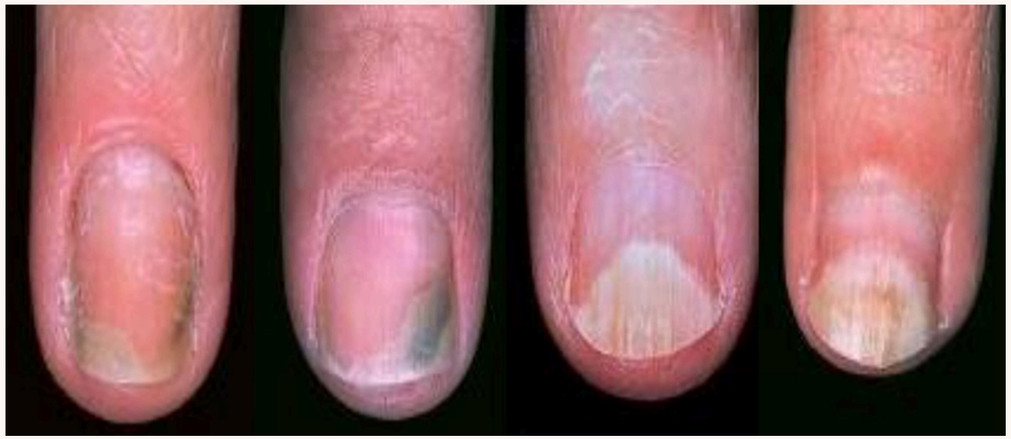
2)Onychomycosis

- Lesion description:
- white spots that can be scraped off the surface, or long yellowish streaks
- within the nail substance
- The disease attacks the free edge and moves its way to the matrix.
- The infected portion is thick and discolored.
- Underlying cause: Fungal infection most commonly Tinea unguium.



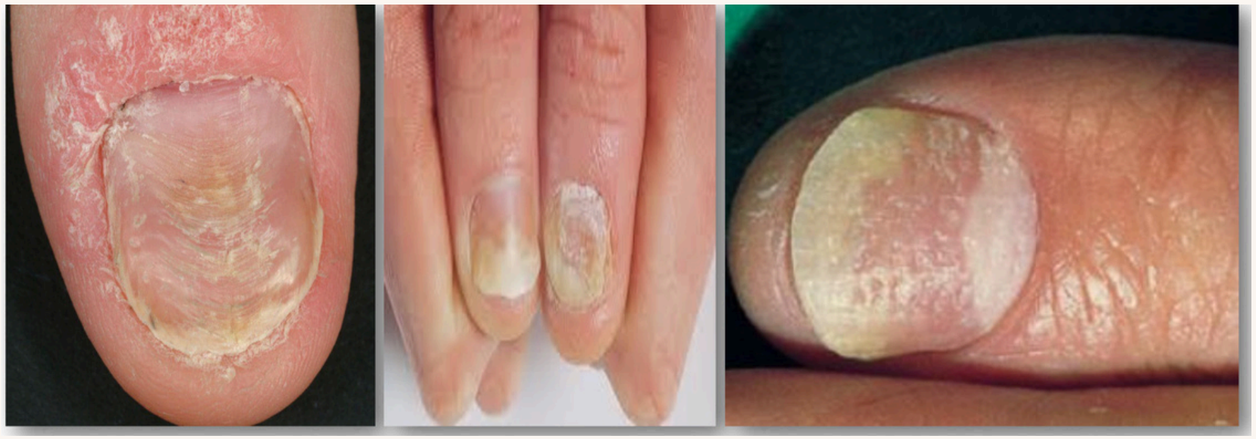
3)Onycholysis

- Lesion description: Nail plate separates from the nail bed
- Underlying cause:
- Most commonly associated with external trauma to the nail (e.g., fungal infection)
- Can also be associated with an internal disorder (e.g., psoriasis



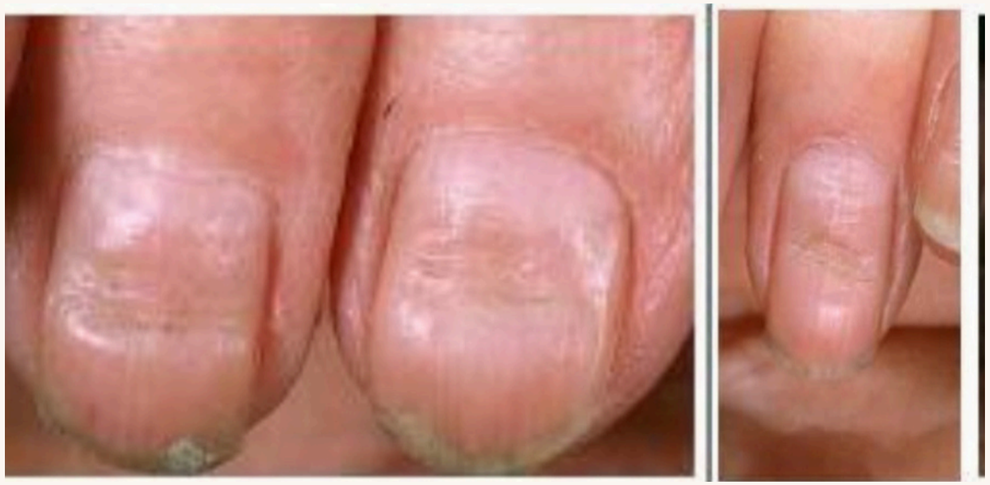
4)Nail pitting

- Lesion: Fine or coarse pits in nail.
- Underlying cause: Psoriasis, eczema, alopecia areata, lichen planus.



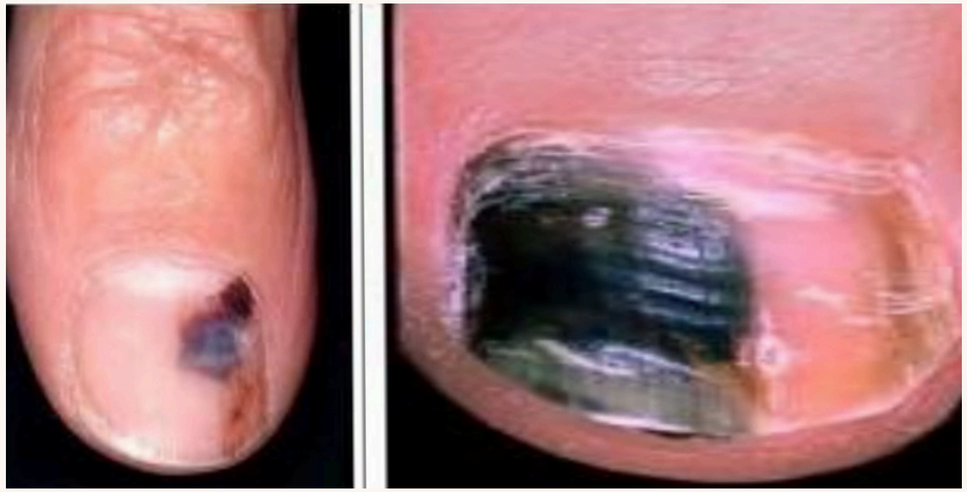
5)Beau’s lines

- Lesion description: Single horizontal ridge, all fingers involved.
- Underlying cause: Sequela of any severe systemic illness, such as a heart
- attack, measles that affects growth of the nail matrix
- Differential diagnosis: Nail biting (usually one nail is involved



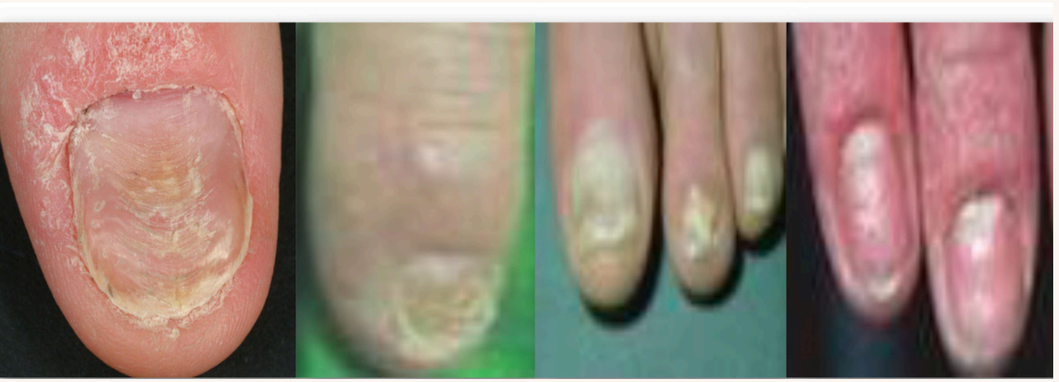
6)Bruised nail

- Lesion description: Dark, congealed spots of blood between nail plate
- and bed, extension of the pigment due to elongation of the nail
- Underlying cause: Crush injury, blunt trauma, repetitive microtrauma
- Differential diagnosis: Melanoma (the pigment is fixed



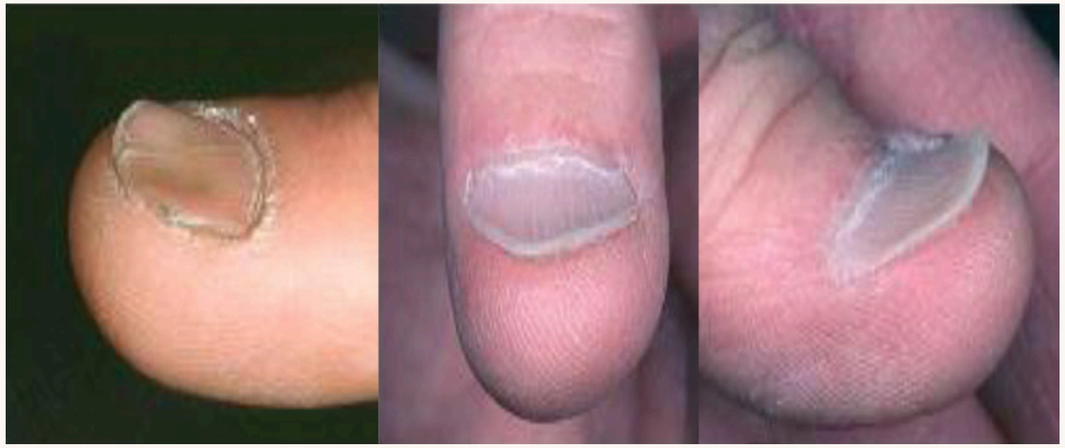
7)Eczema of the nail

- Lesion description: Can affect the eponychium, nail plate and bed causing pitting and onycholysis.



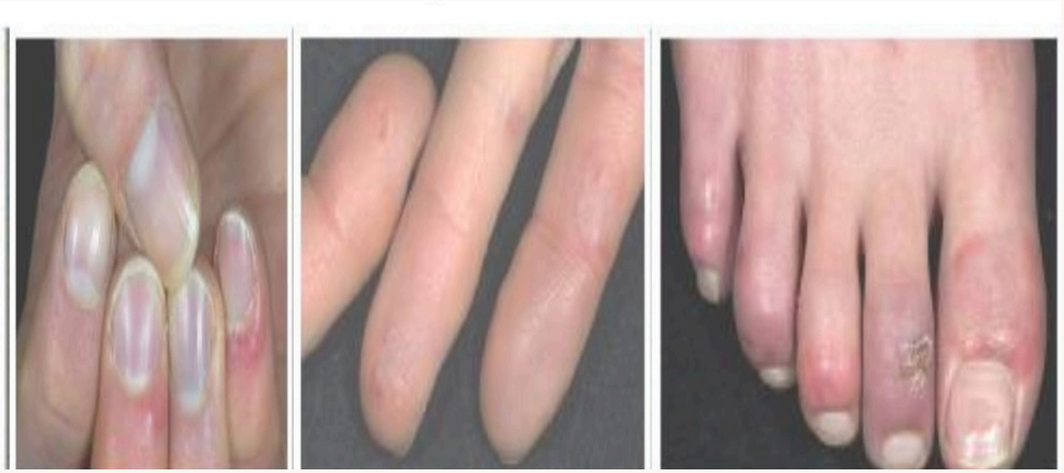
8)Koilonychia (spoon nail)

- Lesion description: Flat or spoon shaped nail often thin and soft.
- Underlying cause: iron deficiency (anaemia), excessive exposure to harsh chemicals etc., or is a congenital condition.



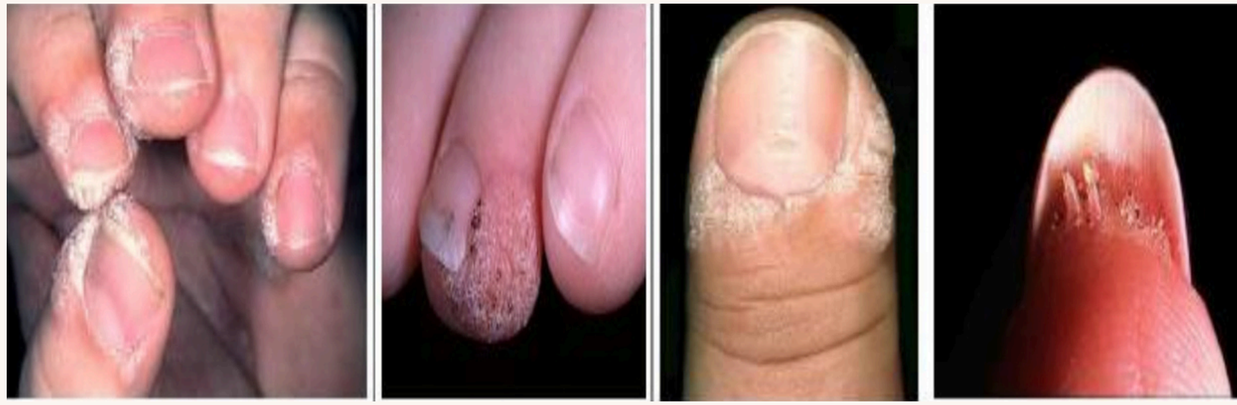
9)Chilblains

- Lesion description: An itchy, sore, tingly, red area resulting in broken skin.
- Underlying cause: Prolonged exposure to cold and poor circulation.
- Differential diagnosis: Raynaud's phenomenon.



10)Verruca vulgaris (common warts)

- Lesion description: Raised lumps of horny tissue in areas of pressure.
- Underlying cause: HPV 1-4
- Note: Cryotherapy may damage the nai matrix.

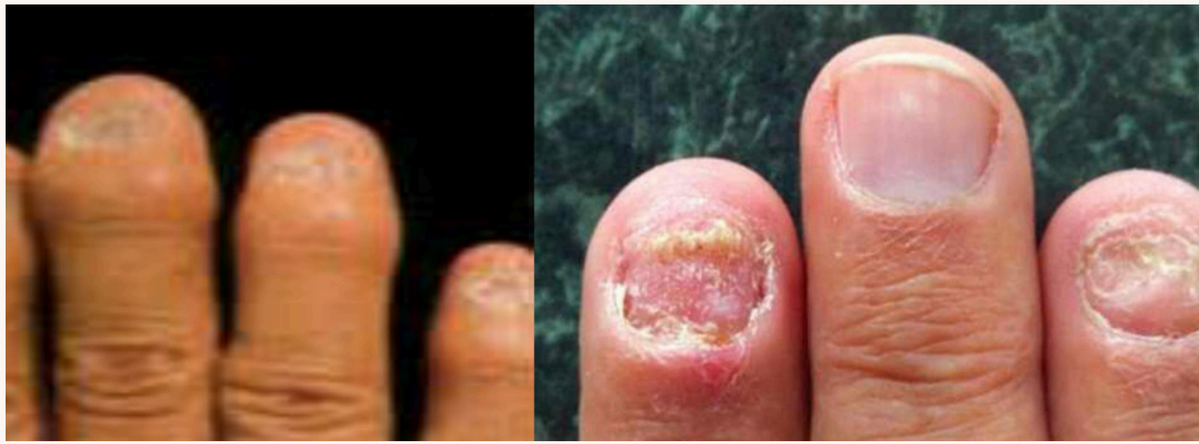


11)Onychophagy

- Lesion description: Bitten nails, often no free edge is visible. Nails look ragged and distorted; skin and nail bed can be exposed and raw.
- Management: Regular manicure or apply nail enhancements to discourage the client from biting their nail.

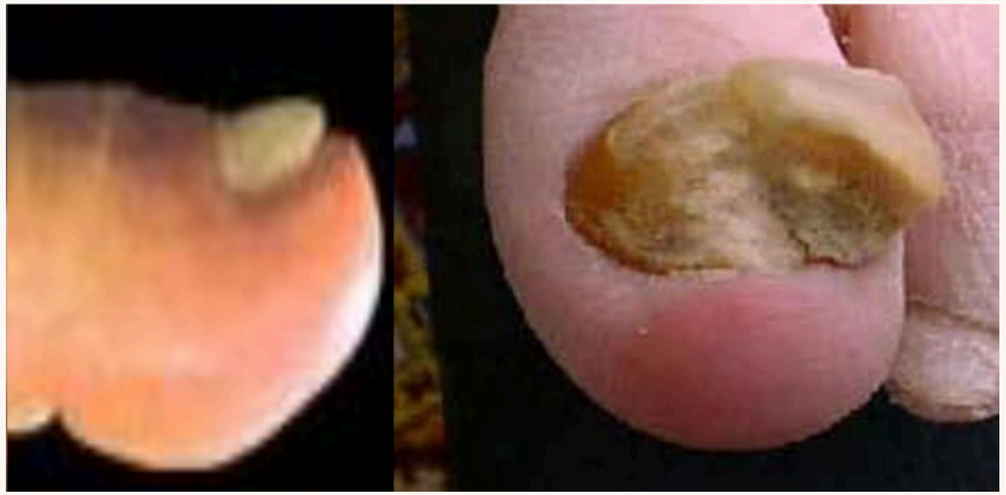
12)Onychatrophia

- Lesion description: The wasting away of the nail, causing it to lose its lustre and become smaller. The nail can also shed completely.
- Underlying cause: injury or disease.



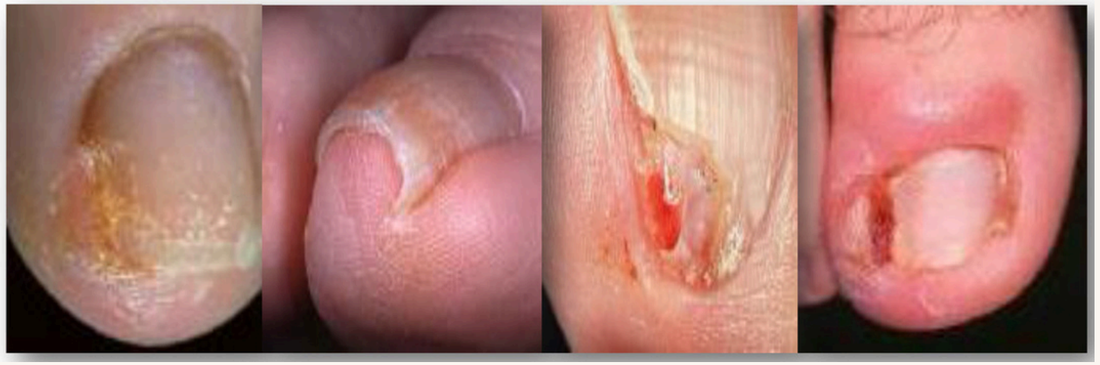
13)Onychauxis

- Lesion description: An overgrowth of the nail, in thickness rather than in length.
- Underlying cause: Internal disturbance, such as a local infection.



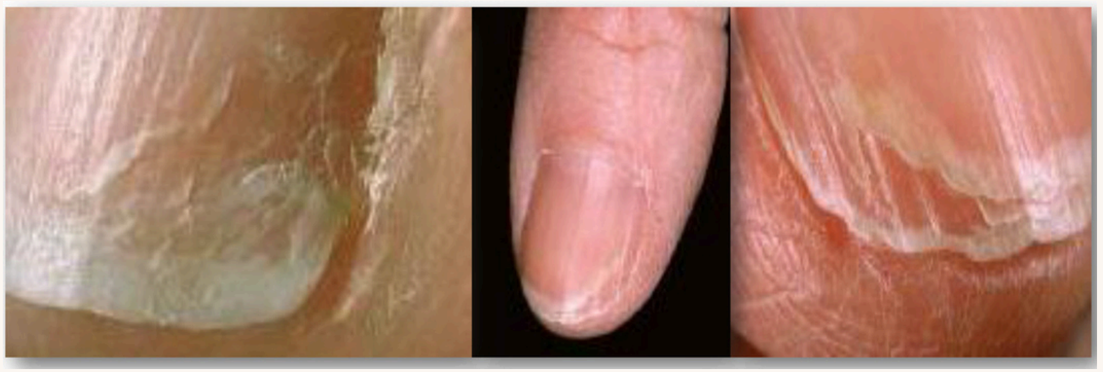
14)Ingrown nails

- Lesion description: The nail grows into the sides of the flesh and may cause infection.
- Underlying cause: Nail grows into the sides of the flesh.
- Differential diagnosis: -Filling the nails too much in the corners.- Failing to correct hang nail



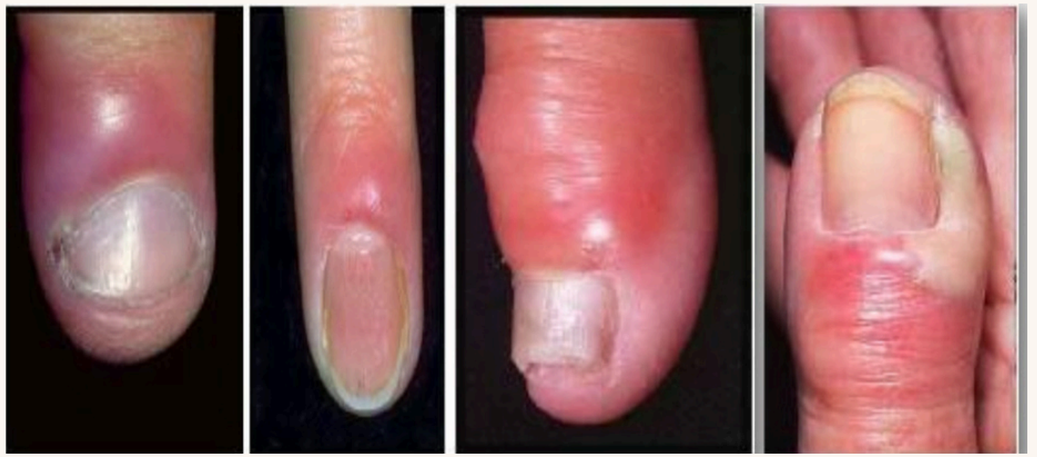
15)Onychorrhexis

- Lesion description: Split or brittle nails
- Underlying cause: injury to the finger or exposure to harsh chemical.



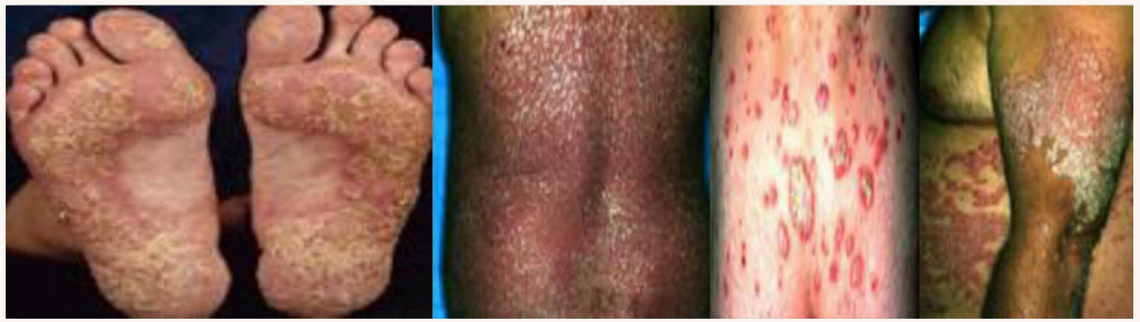
16)Paronychia

- An infectious and inflammatory condition of nail folds.
- Chronic paronychia may weaken defenses and increase the risk of developing a fungal infection of the nail or may permanently deform the nail plate.
- Acute paronychia: Staphylococcal; Chronic paronychia: Candidiasis.



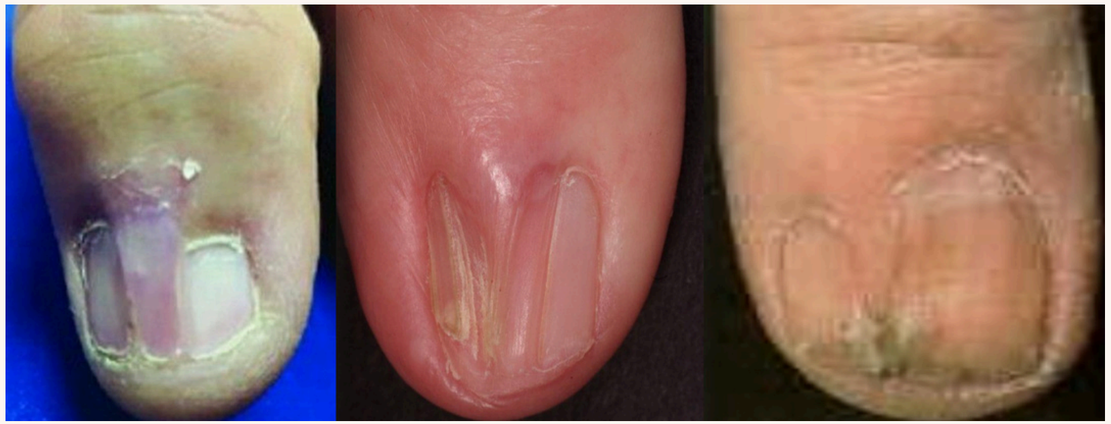
17)Psoriasis

- Lesion description: Nail pitting, oil drop–like patterns of yellow or salmon discolouration, nail thickening, Onycholysis and discolouration.
- Underlying cause: Psoriasis
- Plaques which form around the nail plate can cause pitting. Those which form beneath the nail plate can cause Onycholysis.



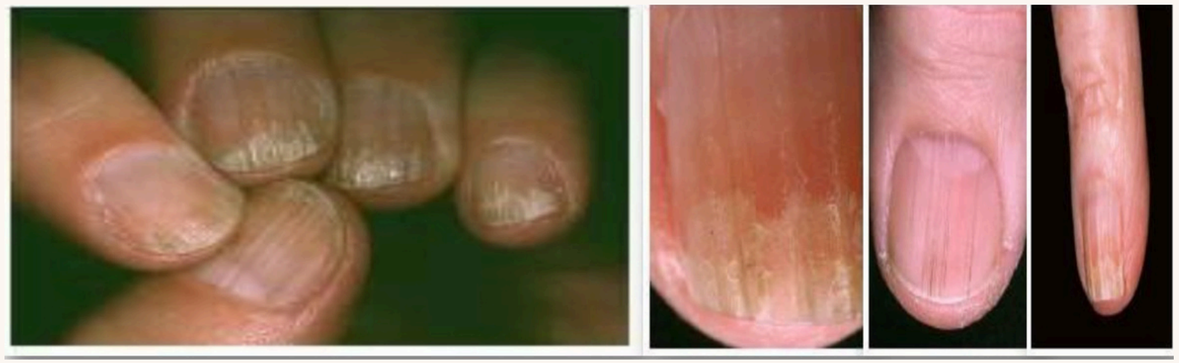
18)Pterigium

- Lesion description: An abnormal winged like growth of cuticle on the nail plate. The skin is slowly stretched and dragged along the bed.
- Underlying cause: Most commonly caused by severe trauma such as warts, burns & blood circulation disorders and lichen planus.



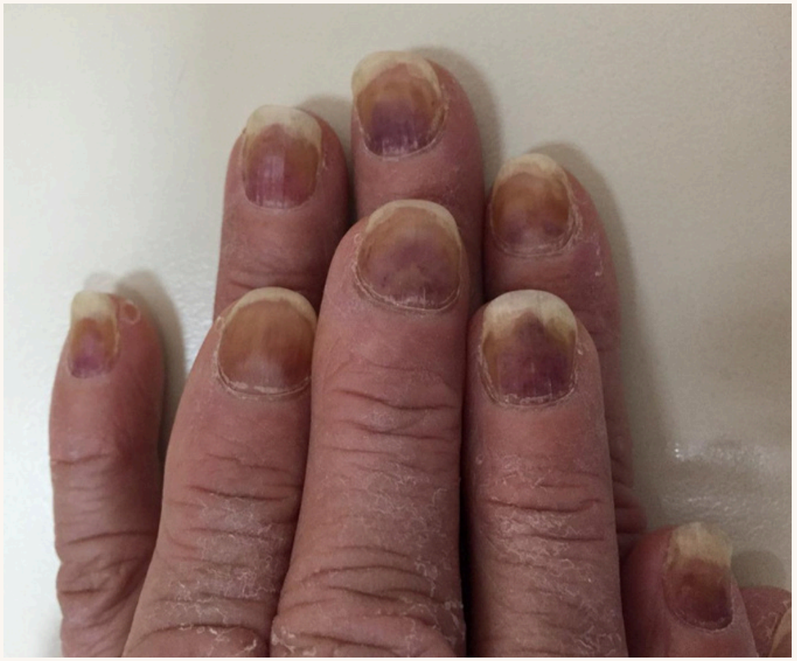
19)Ridges, furrows, corrugations

- Lesion description: Multiple shallow/deep ridges.
- Underlying cause: illness or injury, excessive dieting, incorrect removal of nail enhancements, pregnancy, etc.



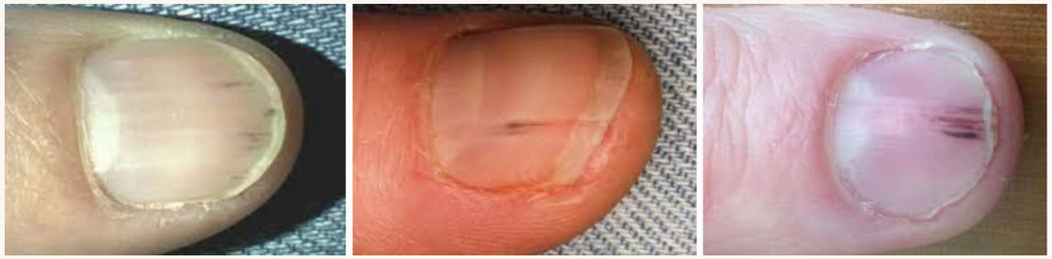
20)Discoloured nail

- Due to exposure to chemicals including dyes & nicotine, some medications, bacterial infections and systemic disorders.



21)Splinter hemorrhage

- Lesion description: Small red streaks that lie longitudinally in nail plate.
- Underlying cause: Trauma, infective endocarditis.



22)Muehrcke's lines

- Lesion description: Narrow, white transverse lines.
- Underlying cause: Decreased protein synthesis or protein loss.

Thank you