BENIGN SKIN TUMORS:

1. Seborrheic Keratosis:
   - It is the most common benign cutaneous neoplasm, usually appearing after the fifth decade with no sex predilection. They increase in number and size with age and are usually asymptomatic. The individual lesion is few mm to few cm in size, is usually brown in color, may have a digitated surface and classically described to have a “stuck on” appearance.
   - Differential diagnosis may include actinic keratosis (pigmented), lentigo maligna, pigmented basal cell carcinoma, wart.
   - Lesser Trélat sign: numerous seborrheic keratoses developing within a short period. Potential marker of internal malignancy.
   - Need not be treated. Ablative measures (e.g. cryotherapy, curettement)

2. Pyogenic Granuloma
   - It is neither pyogenic in origin nor granulomatous histologically. It represents lobular proliferation of capillaries and venules/exuberant granulation tissue - usually in response to traumatic injury.
   - Clinically: rapidly evolving pink/red nodules; they often bleed and at times profusely. The rapid growth and easy bleeding are peculiar features.
   - Treatment: Excision, cryotherapy

3. Hemangiomas
   - Can be classified into: ectatic and proliferative
   - Ectatic: Dilatation of small vessels - e.g. nevus flammeus, port wine stains - can be part of Sturge Weber syndrome. These tend to persist.
   - Proliferative: can be (1) capillary hemangioma - shiny red nodules/plaques - usually resolve with time (by the age of 7 years). (2) Cavernous - proliferation of larger vessels - appears bluish - red in color and feels spongy - tend to persist.
   - Management depends on type and location. Ectatic hemangiomas respond very well to the vascular flash lamp pumped dye laser.
   - Systemic associations

4. Senile Angioma
   - Few mm-sized capillary hemangiomas - bright red in color - usually affect the trunk, increase in number with age.
5. Skin Tags

- Skin colored 1-2 mm soft polypoid papules. Usually appear on neck and axillary areas. Because of their location - may get irritated by friction (clothes...)

6. Dermatofibroma

- A common fibrohistiocytic tumor - with variable degrees of vascular and collagenous components. Looked at as reactive in nature.
- Appears as few mm to 1-2 cm brown to red firm - sometimes tender nodule, with the dermal component attached to the overlying skin ("dimple" sign: lateral compression causes the central portion of the dermatofibroma to dimple) - Usually located on the lower extremities.
- Differential diagnosis - may include keloid, melanocytic nevus, Kaposi’s sarcoma, melanoma.
- Can be excised

7. Melanocytic Nevi (moles)

- Very common lesions present in virtually every person. Composed of nevus cells - that are derived from melanocytes. The vast majority is acquired, mostly during first two decades; size is less than 6 mm. Only 1-2% of the general population is born with moles.
- Usually moles start from the epidermis, later, can grow into the dermis. Can be classified into
  - **Junctional**: nevus cells confined to epidermis
  - **Compound**: nevus cells involve both epidermis and dermis
  - **Dermal**: nevus cells limited to the dermis.

- The features that should always be checked in a melanocytic nevus are: Architecture, border, color, diameter (A,B,C,D) - Asymmetry, irregular, ill-defined border, uneven color may represent malignant change, as well as rapid growth and inflammation.

Congenital moles (usually greater than 6 mm) are classified into small (less than 1.5 cm), intermediate, giant (more than 10/20 cm) - The giant congenital moles are associated with malignant change of 8-10%. 5% of such change appears before the age of 5 years. Excision is advised. The risk of malignant change in small congenital moles is not clear.
The possibility of systemic involvement (mainly CNS) - especially with congenital large moles.

8. Hypertrophic Scars and Keloids

Injury in a predisposed individual can result in an abnormally large scar. Blacks are more predisposed. Also sites where there is increased tension across the wound (e.g. upper chest, shoulders, upper back)

Hypertrophic scars: large, elevated scar - that remains confined to the wound site.

Keloid (Greek: Claw-like) - the hypertrophic scar extends beyond the site of wound injury.

Any injury can lead to these scars - e.g. acne - not only wounds.

9. Keratinous Cysts

Common skin lesions - usually derived from a pre-existing hair follicle, or an implanted tiny portion of the epidermis.

Cyst wall: stratified squamous epithelium - cystic content: keratinous

Usually appear as asymptomatic firm dermal nodules 1-3 cm in size - covered by normal appearing skin. Sometimes the keratinous content imparts a yellowish hue to the lesion. Commonly seen on scalp, trunk.

Histologically - epidermal, trichilemmal, hybrid

Associations - e.g. Gardner's syndrome.

10. Keratoacanthoma

A common keratinizing cutaneous squamous neoplasm, usually benign. The tumor is located on sun exposed areas in people past their 5th decade. It usually arises from follicular infundibula, and is characterized by 3 stages: rapid growth, maturation and resolution. Each phase lasts 2-8 weeks. Usually lesions resolve spontaneously leaving scar tissue.

Some keratoacanthomas follow an aggressive course. On the face they may cause cosmetic and functional difficulties.

The classical keratoacanthoma is a 1-2.5 cm firm skin-colored/erythematous nodule with a crateriform keratotic center. Uncommon variants: Giant keratoacanthoma (large, very destructive), keratoacanthoma centrifugum marginatum, and multiple type(s).

Laboratory studies: to confirm clinical impression one needs to perform an incisional/excisional biopsy that is deep enough and includes both edges, otherwise it may be difficult to differentiate it from squamous cell carcinoma.

Treatment modalities include excision, radiotherapy and intralesional chemotherapy.
11. **Pseudolymphomas of the skin**

A multitude of conditions may have clinical and/or histologic features that may be suggestive of lymphoma, however, they have a benign biologic course. These include lymphocytoma cutis, Jessner’s lymphocytic infiltrate, some arthropod bites, and some drug reactions (e.g. phenytoin derivatives). As always, a clinico-pathologic correlation is needed.
PRE-MALIGNANT SKIN LESIONS

1. *Actinic Keratosis*

- The most common cutaneous premalignant condition. It is the result of chronic sun exposure (ultraviolet-related). Affects usually middle-aged or older individuals. People with fair complexion are more prone to develop the ill-effects of sun exposure.
- Appears on sun exposed areas - mostly face. Usually manifests as 0.5-2 cm asymptomatic, single or multiple, mildly erythematous rough/scaly lesions - can be pigmented. The life-long chance of evolution into squamous cell carcinoma ranges between 2-12%.
- Squamous cell ca arising from an actinic keratosis has a more favorable prognosis (locally invasive, low metastatic potential).
- Treatment: ablative measures e.g. cryotherapy, electrosurgery excision, chemical peeling, topical chemotherapy (5 - Fluro-uracil)

2. *Actinic Cheilitis*

- Actinic keratosis of mucous membranes. Usually lower lip
- Manifests as persistent ill-defined scaling
- Associated with greater malignant degeneration and metastatic potential than actinic keratosis of the skin.
- Treatment as actinic keratosis, also laser vaporization.

3. *Arsenical Keratosis*

- Secondary to arsenic intake / exposure (contaminated water, old medical practices - arsenic containing drugs), agriculture workers.
- Develops at sites of friction / trauma, mainly palms and soles. Appear as multiple punctate (2-10 mm) hard scaly, corn-like papules. Development of erythema may reflect malignant progression.
- Malignant potential is greater than that of actinic keratosis. It is regarded as a potential marker of internal malignancy.
- Treatment is similar to other pre-malignant conditions

4. *Thermal Keratosis*

- Chronic infrared exposure - usually direct contact or close exposure to a heat source - can cause scaly lesions.
- Such scaly lesions are usually present against a background of net-like brown pigmentation (erythema ab igne).

5. *Chronic Radiation Keratosis*
Exposure to ionizing radiation (e.g. radiotherapy) can lead to premalignant keratosis. It appears as scaly erythematous papule/plaque - usually against a background of chronic radiation dermatitis.

6. Bowen’s Disease

Asymptomatic sharply demarcated erythematous scaly plaque - can appear on sun-exposed (usually secondary to UV exposure) or sun-covered (may be caused by exposure to carcinogens e.g. arsenic)

Bowen’s disease on sun covered areas may be regarded as potential marker of internal malignancy

It is squamous cell carcinoma in-situ

Treatment ablative measures - topical chemotherapy, photodynamic therapy.

7. Bowenoid Papulosis

Can appear as papules/plaques - usually genitalia - but can appear elsewhere.

Caused by oncogenic strains of Human papilloma viruses e.g. HPV 16, 18, 31, 33, 35 - (wart viruses).

Treatment local destruction

8. Leukoplakia / Erythroleukoplakia

Appear on mucosal surfaces - usually mouth - persistent slowly developing whitish/erythematous plaques.

Friction, smoking, certain chewing habits - among others, may be predisposing factors.

9. Dysplastic Melanocytic Nevi (DMN)

Can be very few in number or in hundreds. The latter is usually the case of familial melanoma. Can be sporadic or familial. They are acquired, usually appearing at puberty - on the trunk. Showing one or more atypical features (A,B,C,D).

In a patient with DMN(s), melanoma can arise from a DMN or can appear de novo (from normal appearing skin).

DMN (s) are looked at as marker of a group of persons with an increased risk of developing melanoma.

Close follow-up with possible biopsy and pathological evaluation.

LENTIGO MALIGNA
Precancerous lesion of melanoma - appearing on sun-exposed areas, usually the face - incidence of progression into invasive melanoma is around 5%.

Clinically, it appears as a brown macule with uneven pigmentation - usually few cm(s) in size.
MALIGNANT SKIN TUMORS

1. Basal Cell Carcinoma (BCC)

- It is the commonest malignancy affecting humans. Its incidence continues to increase. (Around 400,000 new cases every year - US statistics). The increased incidence is due to increased sun exposure without protection, immunosuppression and the aging population. It is the UVB that is mainly responsible, to a lesser extent is UVA.
- Clinically, the typical patient with a basal cell cancer is an elderly, white individual with type I, II, III skin with the tumor located on sun-exposed areas - mostly on the head and neck. In patients with multiple tumors, consider previous x-ray radiation or carcinogen exposure. The presence of a tumor in a child should raise the suspicion of underlying disorder e.g. xeroderma pigmentosum and nevoid basal cell carcinoma.
- Basal cell carcinoma is of different clinical types: nodular, noduloulcerative, pigmented, superficial, morpheic, and fibroepithelioma of Pinkus.
- The classical lesion is a skin-colored papule/nodule with an ulcer and “pearly” lobulated border.
- Basal cell cancers arise from cells in the basal cell layer of the epidermis and hair follicles. The slow growth rate of these tumors is a reflection of some kind of balance between cell division and necrosis. The tumor takes months or years to double its size. As it grows it invades surrounding tissues. Metastasis is very rare (tumor-stroma dependence). Once metastasis occurs: 50% of patients die in 8 months.
- Treatment includes curettage and electrodessication, excision, cryotherapy, Mohs micrographic surgery, radiation therapy and local chemotherapy (interferon α), synthetic retinoids.

2. Squamous Cell Carcinoma (SCC)

- It is the second most prevalent tumor of the skin (100,000 new cases every year - US statistics).
- Epidemiologic features are essentially the same as those of BCC. The etiology of SCC shares a lot with that of BCC. Namely chronic, cumulative sun exposure and blistering sunburn injuries. SCC may evolve from chronic inflammatory conditions (with or without scarring) such as discoid lupus erythematosus, acne conglobata, hidradenitis suppurativa, fistulous tracts, thermal burns, and nonhealing ulcers, and radiodermatitis. Immune suppression (including iatrogenic) as well as genetic disease (xeroderma pigmentosum and albinism) is predisposing factors. Certain HPV types are oncogenic and may lead to SCC.
- SCC arising from previous scars or ulcers are called Marjolin’s ulcers.
- Clinical presentation: No distinct clinical features. Usually it presents as a slowly growing painless skin-colored/erythematous papule/nodule/plaque with variable degrees of scale and crust formation (± ulceration).
Prognosis depends on several factors that include: histology, depth tumor size, etiology and anatomic site. Those SCC evolving from actinic keratoses have the best prognosis (rarely metastasize). In general those arising on mucous membranes are worse than their skin counterparts.

Treatment modalities: essentially the same as BCC. As always: individualize.

3. **Malignant Melanoma**

A tumor arising from melanocytes. It is one of the most malignant tumors of the body with high potential of widespread metastasis. Its incidence is rapidly increasing - in part - due to increased recreational sun exposure - sunburns during first one or two decades of life. Can evolve from a pre-existing mole or can start de novo.

Clinically, four types are recognized: (1) superficial spreading M (usually trunk, legs, haphazard combination of many colors); (2) Nodular M (usually dark brown/black nodule/plaque); (3) Acral lentiginous M (palms, soles, digits, more frequent in blacks); (4) Lentigo Maligna (older individuals, sites of chronic sun exposure, usually face).

Prognostic variables are multiple, they include sex, anatomic site, Clarck’s level, regression and others. The most important prognostic variable is the thickness of the melanoma (Breslow’s thickness). Thin melanomas (less than 0.85-mm thickness) have the best prognosis.

Need to keep in mind A,B,C,D(s) and work on increasing the public awareness regarding the ill-effects of sun exposure, self examination.

4. **Kaposi’s Sarcoma**


Clinical variants: Classic, endemic, iatrogenic and epidemic.

Treatment: excision, radiotherapy, cryotherapy, and chemotherapy.

5. **Mycosis Fungoides**

A cutaneous T-cell lymphoma - usually running an indolent course. Lesions appearing as poikiloderma - like - patches, indurated plaques/nodules, erythroderma.

Treatment includes photochemotherapy and chemotherapy.