Dermatology. **Lecture: 7**
Dr. Intisar Ahmed **Stage: 5**

Skin tumors

Derived from	Benign	Premalignant/carcinoma *in situ*	Malignant
Epidermis and appendages	Viral wart	Actinic keratosis	Basal cell carcinoma
	Cutaneous horn		Squamous cell carcinoma
	Seborrhoeic keratosis		Malignant melanoma
	Skin tag		Paget's disease of the nipple (although, strictly, a breast tumour)
	Linear epidermal naevus		
	Melanocytic naevus		
	Sebaceous naevus		
	Epidermal/pilar cyst		
	Milium		
	Chondrodermatitis Nodularis helicis		
Dermis	Haemangioma		Kaposi's sarcoma
	Lymphangioma		Lymphoma
	Glomus tumour		Dermatofibrosarcoma protuberans
	Pyogenic granuloma		Merkel cell carcinoma
	Dermatofibroma		Metastases
	Neurofibroma		
	Neuroma		
	Keloid		
	Lipoma		
	Lymphocytoma cutis		
	Mastocytosis		

Tumors of the *epidermis* and its appendages:

Benign tumors:

Cutaneous horn:

Common horn-shaped excrescence arising from keratinocytes, resemble viral wart clinically.

*Treatment:

-Excision.

-Curettage with cautery to the base.

Seborrhoeic keratosis:

Common benign epidermal tumor, unrelated to sebaceous glands.
*Cause:
1.Inherited.
2.Follow an inflammatory dermatosis.
3.Sudden eruption of hundreds lesions is associated with an internal neoplasm (adenocarcinoma of the gastrointestinal tract), **Leser Tr´elat sign**.
*Presentation:
*Arise after age of 50 years.
*Multiple or single.
*Most common on the face and trunk.
*Equally sex
*A distinctive '**stuck-on**' appearance.
*Surface may be smooth or verrucous.
*Colour varies from yellow–white to dark brown–black.
*Treatment:
-Can safely be left alone.
-Curette under local anaesthesia.
-Cryotherapy.

Skin tags (acrochordon)

*Benign out growths of skin affect mainly middle-aged and elderly.
*Cause:
1. Unknown.
2. Familial.
3. obesity.
4. Associated with tuberous sclerosis, acanthosis nigricans, acromegaly and diabetes.
*Presentation and clinical course:
*Soft skin-coloured or pigmented pedunculated papules commonly found around the neck and within the major flexures.
*Treatment:
-Excision by scissors.
-Frozen with liquid nitrogen

Naevi

*Skin lesion has a localized excess of one or more types of cell in a normal cell site.
*Composed of keratinocytes (epidermal naevi), melanocytes (congenital melanocytic naevi), connective tissue elements (connective tissue naevi) and a mixture of epithelial and connective tissue elements (sebaceous naevi).

Melanocytic naevi (moles)

*Classification:
-Congenital melanocytic naevi.
-Acquired melanocytic naevi.
-Junctional naevus.
-Compound naevus.
-Intradermal naevus.
-Spitz naevus.
-Blue naevus.
-Atypical melanocytic naevus.

*Cause and evolution:
1. Unknown.
2. Genetic.
3. Excessive sun exposure during childhood.
*Appear in early childhood, increase in numbers:
during adolescence, after severe sunburn, during pregnancy, oestrogen therapy, flare-ups of lupus erythematosus, chemotherapy and immunosuppression.
*New melanocytic naevi appear less often after the age of 20 years.
***Congenital melanocytic naevi:**
*Present at birth or in neonatal period.
*Colour varies from brown to black or blue–black.
*Some protuberant and hairy, with a cerebriform surface.
*Giant naevi with a diameter greater than 20 cm have risk of developing **melanoma** (lifetime risk up to **5–7%**).
*Posterior midline location associated with multiple satellite naevi should prompt a consultation with a **neurologist and MRI scan to rule out neurocutaneous melanosis.**

*Complications:
1. Inflammation.
2. Depigmented halo.
3. Malignant change: considered in case of: **Enlargement; Increased or decreased pigmentation; Altered shape; Altered contour; Inflammation; Ulceration; Itch; or Bleeding.**
***So examined carefully, remembering the '**ABCDE'** features of malignant melanoma:
-**A**symmetry
-**B**order irregularity
-**C**olor variability
-**D**iameter greater than 0.5 cm
-**E**volution (change)
*Treatment:
-Excision is needed when:
1. Anaevus is unsightly.
2. Malignancy is suspected or is a known risk.
3. Anaevus is repeatedly inflamed or traumatized.

Premalignant tumors

Actinic keratoses

*Discrete rough-surfaced lesions crop up on sun damaged skin.
*Cause:
1. Cumulative sun exposure.
2. Fair complexions.
*Presentation:
*Affect middle-aged and elderly.
*Pink or grey rough scaling macules or papules seldom exceed 1 cm in diameter.
*Rough surface, better felt than seen.
*Complications:
-Transition to an invasive squamous cell carcinoma.
*Treatment:
-Freezing with liquid nitrogen.
-Shave removal or curettage.
-5 fluorouracil cream.
-Imiquimod.
-Photodynamic therapy.

Malignant epidermal tumors

Basal cell carcinoma (rodent ulcer)

*__Most__ common form of skin cancer.
*Most common on the faces of the middle-aged or elderly.
*Never metastasize.
*Cause:
1. Prolonged sun exposure.
2. Fair skinned individuals.
3. Scars caused by radiation exposure, vaccination or trauma.
3. Photosensitizing pitch, tar and oils can act as cocarcinogens with ultraviolet radiation.
4. Previous treatment with arsenic.
5.Genetic: Gorlin'ssyndrome, xeroderma pigmentosum, albinism.
*Presentation:
1. Nodular: **most** common type, small translucent skin-coloured papule slowly enlarges with central necrosis leaves an ulcer with an adherent crust and a rolled edge, telangiectatic vessels run across surface .
2. Cystic: firstly like nodular type, later cystic changes predominate.
3. Morphoeic: yellow or white waxy plaques with an ill-defined edge.
4. Superficial (multicentric): on the trunk, red pink or brown scaly thin plaque with a fine 'whipcord' edge, can grow to more than 10 cm in diameter.
5. Pigmented: pigment may be present in all types of BCC.
*Clinical course:
*The slow growth destroys tissue locally.
*Untreated, can invade underlying cartilage or bone or damage important structures such as the tear ducts.
*Differential diagnosis:
-nodular: intradermal melanocytic naevus, squamous cell carcinoma, giant molluscum contagiosum, keratoacanthoma.
-Pigmented: seborrhoeic warts, malignant melanomas.
-Cicatricial: morphoea, scar.
-Superficial: intraepidermal carcinoma, psoriasis, nummular eczema.
*Treatment:
1. Excision with 4 mm of surrounding normal skin.
2. Electrodessication and curettage.
3. Mohs' micrographic surgery: indicated for: 1.morphoeaform, 2.infiltrative, 3.lesions near vital structures such as the nose, ear, eyelids, eyebrow, 4.large tumours (>2cm), 5.perineural or perivascular invasion, 6.recurrent lesions 7.immunosuppressed pt.

4. Radiation therapy.
5. Cryotherapy.
6. Imiquimod and photodynamic therapy.
7. palliative treatment with curettage and cautery.
8. vismodegib: for advanced and metastatic disease.

Squamous cell carcinoma

*Common tumour.
*large and invasive are associated with a significant risk of **metastasis**, so require careful evaluation and aggressive management.
*Cause:
-Same as BCC.
*Clinical presentation and course:
*May arise as thickenings in an actinic keratosis or, de novo, as small scaling nodules, or as ulcers with a granulating base and an indurated edge.
*Common on the lower lip and in the mouth.
*Metastasis common if tumors arising in **scar, chronic draining sinuses, chronic ulcers, areas of previous X-radiation, thermal injury, chronic inflammation, more than 2 cm in diameter, greater than 2 mm in depth, poorly differentiated, perineural involvement, immunosuppressed pt**.
***Treatment:
-Same as BCC.

Keratoacanthomatous

*Rapidly growing squamous cell tumors do not invade and occasionally resolve spontaneously.
*Mainly on exposed skin of fair individuals.
*Commonly on the face and arms.
*Starts as a pink papule rapidly enlarges may reach a diameter of 1 cm in a month or two, After 5–6 weeks the centre of the nodule forms either a keratinous plug or a crater.
*If left, occasionally resolve spontaneously over **6–12 months** leaving an ugly depressed scar.
*Treatment:
-Excision.
-Curettage and cautery.

Malignant melanoma

*lethal.
*Cause:
1. Genetic.
-Susceptibility genes: 10% familial, mutations in CDKN2A, CDK4 and MC1R.
-Susceptible phenotypes: white people with blond or red hair.
2. Sunlight: episodic exposure to intense sunlight is main cause, so number of sunburns more relevant than cumulative ultraviolet radiation dose.
3. Pre-existing melanocytic naevi: especially atypical naevi, congenital melanocytic naevi
*Prevention and early diagnosis is critical.
*Clinical features:
*Four main types of malignant melanoma:
1. Lentigo maligna melanoma: on the exposed skin of the elderly, irregularly pigmented, irregularly shaped macule enlarge slowly.
2. Superficial spreading melanoma
is the most common type in fair skinned individuals between 30 and 50 years of age.
3. Acral lentiginous melanoma: on palms and soles in the elderly most common in blacks.
4. Nodular melanoma: pigmented nodule, most rapidly growing and aggressive type, found on the legs and trunk.
5. A melanotic melanomas: rare, on soles of the feet.
6. Desmoplastic melanomas: rare, on head, neck, palms and soles, aggressive with perineural extension.
7. Subungual melanomas: painless, areas of pigmentation expanding under the nail and on the nail fold (Hutchinson's sign).
*Metastatic melanoma spread to surrounding skin, regional lymph nodes or to other organs.
*Poor prognostic indicators:
-Depth of primary tumour >4.0 mm, 5-year survival 45%.
-Males sex.
-After 50 years of age.
-On trunk, upper arms, neck and scalp.
-Ulceration.
-Mitosis.
-Sentinel node.

*Differential diagnosis:
melanocytic naevus, seborrhoeic keratosis, pigmented actinic keratosis, pigmented basal cell carcinoma.

*Treatment:
-Surgery.
-Adjunctive therapies: immunotherapy.
-Chemotherapy.
-Follow-up care.

Tumours of the dermis

Benign

Haemangiomas

*Benign endothelial cell neoplasms, occur in about **10%** of infants at age **1** year.
*Appear within a few weeks of birth, and grow for a few months, forming a raised compressible swelling with a bright red surface.
*Spontaneous regression then follows by the age of **5 years in 50%** of children and in **90% by the age of 9** years, leaving area of slight atrophy.
*Bleeding and ulceration is common complication.
***Common** in:
1.Girls,
2.Preterm infants with low birthweight,
3.Multiple gestations pregnancy,
4.Advanced maternal age.
*Large cervicofacial haemangiomas may be associated with other congenital anomalies.
*Patients with multiple lesions may have visceral involvement.
* Treatment:
-Observation.
-Intralesional corticosteroid.
-Prenisolone at 2–4 mg/kg/day.
-Beta-blockers: propranolol 2–3 mg/kg/day in 2–3 divided doses. With careful monitoring of blood sugar and blood pressure
-Pulsed dye lasers.
-Plastic surgery.

Malignant

Kaposi's sarcoma

*Malignant tumor of proliferating capillaries and lymphatics.
*Four types, Human herpesvirus type 8 (**HHV8**) has been linked to all types.
1. Classic (sporadic): most in elderly Mediterranean and Eastern European men. usually on the feet and ankles, as dark blue to purple macules progressing to tumors and plaques which ulcerate and fungate. Oedema of the legs may be severe.
2. Endemic African Kaposi's sarcoma: primarily in men who are HIV seronegative in Africa.
3. Epidemic Kaposi's sarcoma: associated with **AIDS** caused by the human immunodeficiency virus (HIV-1). Lesions appear any where but are most common on the upper trunk and head and neck, bruise-like lesions then become raised, increasingly pigmented and evolve into nodules and plaques. May arise on the oral mucous membranes. Prognosis is poor.
4. Kaposi's sarcoma and immunosuppression: similar to the epidemic form, occurs following organ transplantation or immunosuppressive therapy.
*Treatment:
-Surgery.
-Liquid nitrogen cryotherapy.
-Intralesional chemotherapy.
-Radiotherapy.
-Chemotherapy, with chlorambucil or vinblastine.
-Topical retinoids.