CME Article

Common benign and malignant neoplasms of the skin

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Skin cancer is the seventh most common cancer among Singaporean males and the eighth commonest cancer among Singaporean females. Skin cancer is becoming more common among Singaporeans. From the period of 1968 to 2002, the average percentage increase in the age standardised rates of skin cancer was 1.27 in males and 1.55 in females.[1]

Skin tumours frequently present as a "lump" or "mole" on the unsuspecting patient. Differentiating and diagnosing these lesions could also prove to be a daunting challenge to the clinician. This article provides a brief overview of the common, or clinically important, skin tumours encountered in clinical practice.

BENIGN SKIN LESIONS

Seborrhoeic keratosis and inverted follicular keratosis

Aetiology

The aetiology of seborrhoeic keratosis (Fig. 1) is unknown. Patients with great numbers of lesions may have a positive family history which may reflect a genetic propensity. Its occurrence may also be related to sun exposure.[2,3]

Clinical presentation

They commonly occur after the age of 30 years, and can occur on any part of the body except mucous membranes. They begin as flat and sharply-demarcated brown macules. With progression, they develop a polypoidal and an uneven surface with a characteristic stuck-on appearance. Follicular prominence is also a characteristic feature presenting as pale follicular plugs within a darker lesion or dark (black or brown) plugs within a pale lesion. Multiple eruptive seborrhoeic keratoses (the sign of Leser-Trelat) is associated with multiple malignancies, including carcinomas of the colon, stomach and breast as well as lymphoma, leukaemias and melanoma.[4]

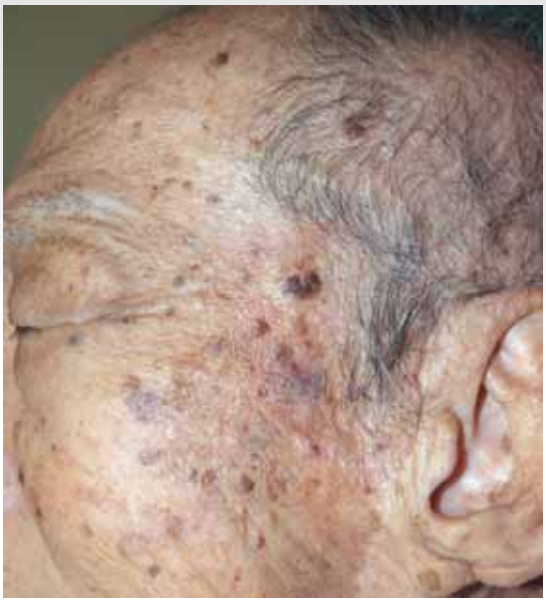

Fig. 1 Photograph shows seborrhoeic keratosis and inverted follicular keratosis.

Histology

This lesion consists of uniform basaloid cells with interspersed keratin-filled horn cysts. Melanocytes are also present and melanin production contributes to darker lesions. Other clinicopathological variants of seborrhoeic keratosis include reticulated seborrhoeic keratosis, clonal seborrhoeic keratosis, irritated seborrhoeic keratosis, stucco keratosis melanoacanthoma and dermatosis papulosa nigra. Inverted follicular keratosis is believed to be an inflammatory variant of seborrhoeic keratosis. It is characterised by an endophytic process within the epithelium of a pilosebaceous follicle.[5]

Treatment

Treatment is instituted when the lesions are functionally and cosmetically disturbing. Options include cryotherapy, electrodesiccation and laser therapy with Q-switched ruby laser. Surgical excision and histological evaluation are recommended for lesions that are atypical, and when malignancy is suspected.

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Sebaceous hyperplasia

Aetiology

In newborns, sebaceous hyperplasia results from maternal androgens causing overgrowth of the sebaceous glands.[5] In adults, its aetiology is related to decreased androgen levels resulting in decreased cell turnover of the gland, prolonged ultraviolet radiation exposure and direct causative effect of cyclosporine.[6]

Clinical presentation

Sebaceous hyperplasia occurs in individuals beyond the middle age. It presents as elevated soft and yellowish nodules with a central umbilication at the site of the ductal opening. In Fordyce's disease, similar lesions occur on the vermillion border of lips and oral mucosa. Sebaceous hyperplasia also commonly occurs in newborns.[7,8] These are characterised by tiny macules or papules at the opening of each pilosebaceous follicle over the nose and cheeks. Sebaceous hyperplasia is also seen in solid organ transplant patients, these are thought to be induced by cyclosporine.[6]

Histology

Lobules of mature sebaceous glands surround a dilated sebaceous duct which opens into the epidermis or mucosal surface.[9]

Treatment

Laser therapy, electrodesiccation and topical bichloracetic acid can be used to treat this condition. Oral isotretinoin is also effective for diffuse multiple lesions. Neonatal sebaceous hyperplasia needs no treatment and will resolve by 4–6 months.

Syringoma

Clinical presentation

Syringomas are benign tumours of the eccrine ducts. It occurs more commonly in females than in males, with onset usually at puberty or the third and fourth decade of life. It commonly involves the eyelids, axillae, umbilicus and pubic area. It is characterised by skin coloured or yellowish firm papules 1–3 mm in size. Eruptive syringoma (eruptive hidradenoma of Darier and Jaquet) is a rare variant. Large numbers of lesions occur on the neck, chest and abdomen, which may remain or disappear.[10]

Histology

The lesions consist of small cystic ducts and are lined by solid epithelial strands embedded within a fibrous stroma. Some of the epithelial cells possess a comma-like tail assuming a tadpole appearance. The ducts may connect with dilated cysts of intraepidermal ducts but do not connect with the secretory segment of glands.[5,11]

Treatment

These lesions can be treated with excision, electrodesiccation and curettage, dermabrasion and CO_2 laser resurfacing.

Xanthoma

Aetiology

It is caused by lipid (LDL) deposition under the skin or other sites, such as tendons or gastrointestinal tract. Possible mechanisms include capillary leakage of LDL, increased acetylated LDL or oxidised LDL uptake into macrophages and increased local tissue lipogenesis.[12]

Clinical presentation

They present as soft, flat yellowish plaques with sharply defined margins (Fig. 2). They can occur on any part of

Fig. 2 Photograph shows a xanthelasma, a xanthoma of the eyelid.

the body, such as elbows, knees, hands, feet, buttocks or eyelids (xanthelasma). They may be a sign of an underlying disorder, such as hyperlipidaemia, primary biliary cirrhosis, diabetes mellitus or familial hypercholesterolaemia.

Histology

They consist of xanthoma cells, foamy histiocytes laden with intracellular lipids (mainly cholesterol) in the upper dermis.[12]

Treatment

Excision, laser ablation, chemical cauterisation and electrodesiccation can be used, in addition to treating the underlying lipid disorder. The patient must be warned of recurrence.[13]

Naevus

Aetiology

They are caused by proliferation of melanocytes in the epidermis or dermo-epidermal junction (junctional), dermis (intradermal) or both (compound) (Fig. 3).

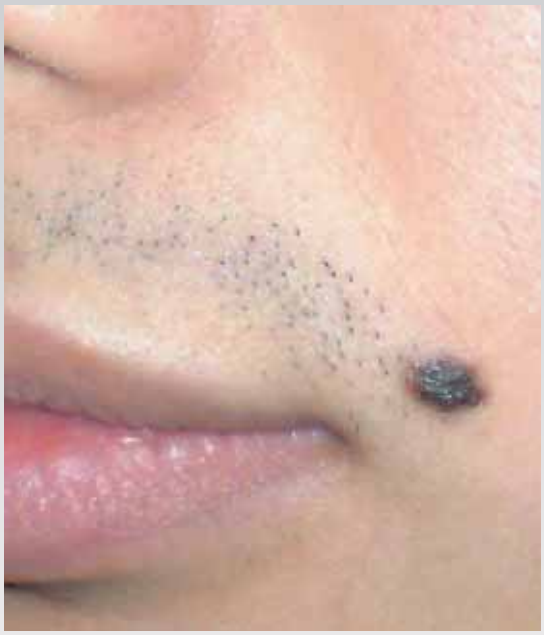


Fig. 3 Photograph shows a melanocytic naevus.

Clinical presentation

Nevomelanocytic naevi first appear after 6–12 months of life, remain relatively constant in numbers during adulthood, and decrease in numbers from the sixth decade.[14] Junctional naevi present as flat macules. Dermal naevi form raised pigmented lesions, while compound naevis have features of both junctional and dermal naevi. Other varieties of naevus include:

a) *Blue naevus*: collection of melanocytes deep in the dermis, accounting for its bluish appearance.
b) *Halo naevus*: Melanocytic naevus surrounded by a depigmented area (halo) caused by both humoral and cellular immune activity against the melanocytes. The entire naevus becomes depigmented with time.
c) *Epitheloid cell / spindle cell / Spitz naevus*: presents as a pink or tan papule composed of nests of epitheloid and spindle cells. Frequently associated with surrounding dermal inflammation.
d) *Becker's naevus*: Pigmented area with increased hair growth usually occurring in the upper trunk and shoulders.
e) *Naevus of Ota*: Blue-black or slate-gray macules intermingled with brown spots occurring in the skin supplied by the first and second trigeminal branches, mucosa, conjunctiva and tympanic membrane. Similar lesions occurring in the distribution of lateral supraclavicular and lateral brachial nerves are known as *Naevi of Ito*.

Histology

These consist of nests of nevomelanocytes with nuclei similar to, or larger than other melanocytes. Epidermal nevomelanocytes are larger and resemble epithelial cells, and gradually become smaller and rounder in appearance towards the dermis.[5]

Treatment

Excision biopsy is performed for cosmesis, irritation and features of malignant change, such as asymmetry, irregular border, bleeding, colour variation, itch and growth in size. Laser selective photothermolysis can also be used.

Cutaneous horn

Clinical presentation

It usually occurs in the elderly, and presents as a conical, dense, hyperkeratotic nodule due to unusual cohesiveness of keratinised material. It may be white or yellowish and straight, curved or twisted (Fig. 4). It may result from solar keratoses, seborrhoiec keratoses, filiform warts, trichilemmal keratoses, keratoacanthomas and basal cell epitheliomas. There is a need to exclude premalignant and malignant lesions in cutaneous horns.[15] Lesions with a low height-to-base ratio is more likely to be malignant.[16]

Histology

Areas of hyperkeratosis and parakeratosis are seen. A granular layer may be present under the hyperkeratotic areas with variable degree of acanthosis.

Treatment

Shave biopsy, including the base, is both diagnostic and curative.

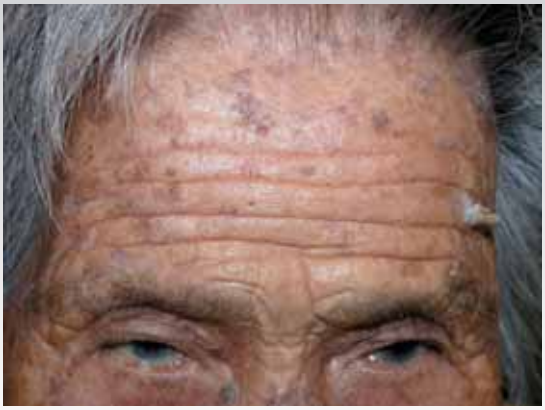


Fig. 4 Photograph shows a cutaneous horn on the forehead.

Epidermoid cyst

Aetiology

This lesion occurs as a result of proliferation of surface epidermal cells within the dermis. Epidermoid cysts (Figs. 5 a–b) result from occlusion of the pilosebaceous follicle, implantation of epidermal cells into the dermis following penetrating injury, or trapping of epidermal cells along embryonal fusion planes.

Clinical presentation

Presents as a dome-shaped lesion, tethered to the overlying skin, and is freely mobile over the underlying structures. The central punctum represents the obstructed orifice of the pilosebaceous follicle. The lesion is filled with keratinaceous material.

Histology

These cysts are lined with stratified squamous epithelium and are filled with keratinous material arranged in multiple layers, and sometimes contain melanin and calcified material.

Treatment

Excision biopsy. Infected cysts should be treated with a course of antibiotics, failing which drainage of the abscess is needed. Excision is then carried out after the infection and inflammation have subsided.

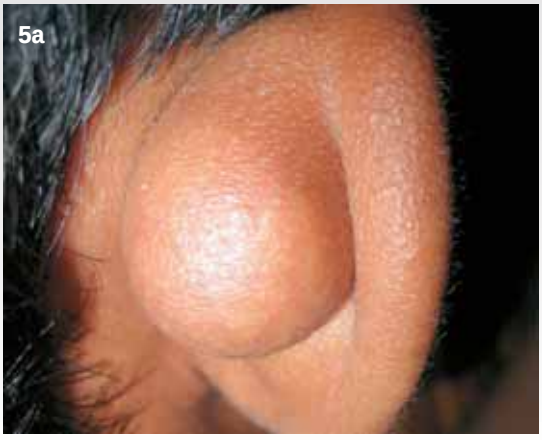


Fig. 5a Photograph shows a cyst at the postauricular surface.

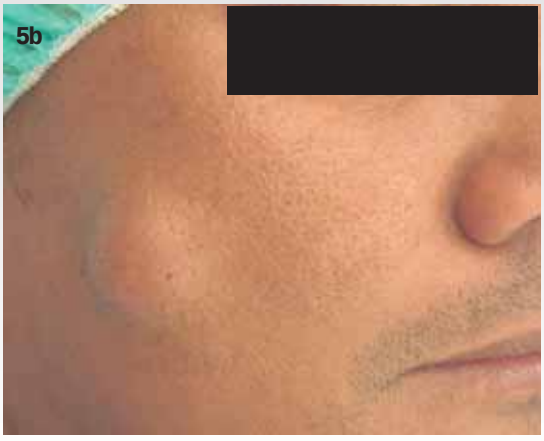


Fig. 5b Photograph shows a cyst at the right check.

Pyogenic granuloma

Aetiology unknown

Clinical presentation

Benign vascular lesion of the skin or mucosa (Fig. 6). It presents as a red nodule or papule which develops rapidly over a period of weeks, and is prone to bleeding and ulceration. May be associated with drug ingestion[17] (systemic retinoids, Indinavir) or pregnancy[18] (in second and third trimesters).

Histology

Numerous capillaries and venules arranged in radial pattern within an oedematous stroma containing variable amounts of inflammatory infiltrates. A regressing lesion shows extensive fibrosis.

Treatment

Excision biopsy, shave biopsy and electrocautery, cryotherapy, laser therapy.[19]

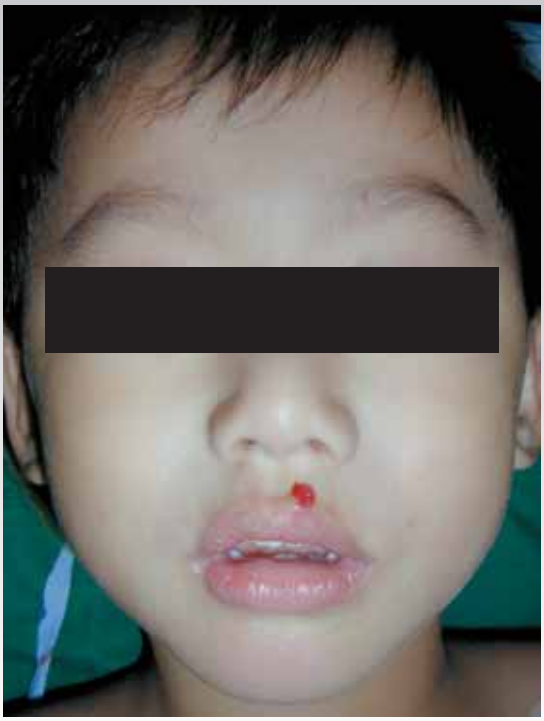


Fig. 6 Photograph shows a pyogenic granuloma at the upper lip.

Haemangioma

Aetiology

This lesion may represent a hamartomatous proliferation of endothelial cells (Fig. 7).

Clinical presentation

Its onset is usually after birth. Development is divided into proliferative (rapid growth for 3– 9) months, up to 18 months) and involutive phase (over 2–6 years). It presents as an erythematous macule or ecchymotic patch with irregular borders occasionally. Large lesions may be associated with ptosis, obstruction of vision, high output cardiac failure, thrombocytopenia or haemolytic anaemia. 50% of the cases will regress by five years and 70% will regress by seven years of age.[20]

Histology

Proliferation of endothelial cells and pericytes with formation of vascular spaces. The involution phase is characterised by progressive fibrosis and disappearance of blood vessels.

Treatment

Intralesional steroid injections can be given for small haemangiomas. Large life-threatening haemangiomas are treated with systemic glucocorticoids and interferon alfa-2a and 2b. Surgical excision is performed after regression has ceased, or when complications such as bleeding and ulceration arise.[20]

Fig. 7 Photograph shows haemangioma at the right upper eyelid.

PREMALIGNANT SKIN LESIONS

Congenital naevus

Aetiology

They develop probably between 40 days of gestation and six months in utero. Genetic mechanisms may account for familial aggregation.

Clinical presentation

These lesions are present at birth. They are characterised by pigmented lesions with regular margins, smooth or lobular surfaces (Fig. 8) and occasionally have long coarse hair. The risk of melanoma development is proportional to the size, especially if it involves over 5% of body surface, or > 20 cm in adolescents (large/giant congenital naevus). The risk of malignant change ranges from 5% to 40%.[21]

Histology

Similar to acquired naevus, but frequently involves the lower dermis and more likely to involve the dermal appendages and neurovascular structures.

Treatment

Regular follow-up and prophylactic excision, preferably before onset of puberty.[21,22]

Fig. 8 Photograph shows a giant hairy naevus.

Actinic keratosis

Aetiology

Actinic keratosis (Fig. 9) occurs in areas exposed to sunlight. UV-B radiation induces thymidine dimer formation in DNA and RNA, as well as p53 gene mutations and telomerase alterations.[23]

Clinical presentation

These lesions are more common in fair-skinned and blue-eyed individuals with chronic sun exposure and in immunosuppressed individuals. They occur in sun-exposed areas, presenting as rough, scaly papules and plaques. The risk of evolving to squamous cell carcinoma is 13%–20% over ten years.[23,24]

Histology

Atypical, pleiomorphic keratinocytes occur in the basal cell layer which may extend to the granular and cornified layers with hyperkeratosis and parakeratosis.

Treatment

Cryosurgery, topical fluorouracil, curettage, topical tretinoin, imiquimod 5% (interferon inducer) and photodynamic therapy with 5-aminolevulinic acid. Reduced sun exposure and regular use of sunblock prevent its development.[23]

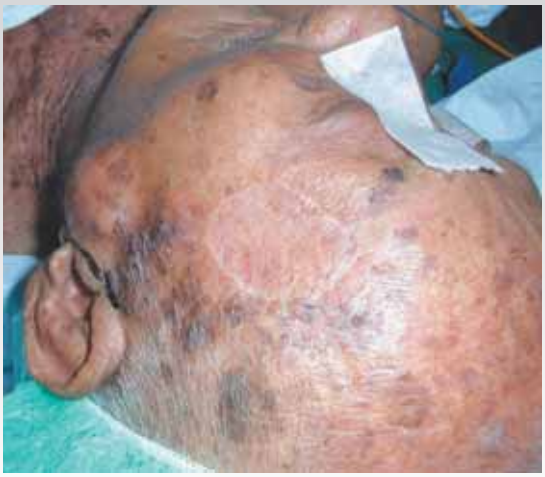


Fig. 9 Photograph shows actinic keratosis over the sealp.

Keratoacanthoma

Aetiology

The tumour arises from hair follicles, and is caused by exposure to sun and carcinogens, such as pitch and tar. Other aetiological factors, such as trauma, human papilloma virus, genetic and immunosuppression, have been implicated.[25]

Clinical presentation

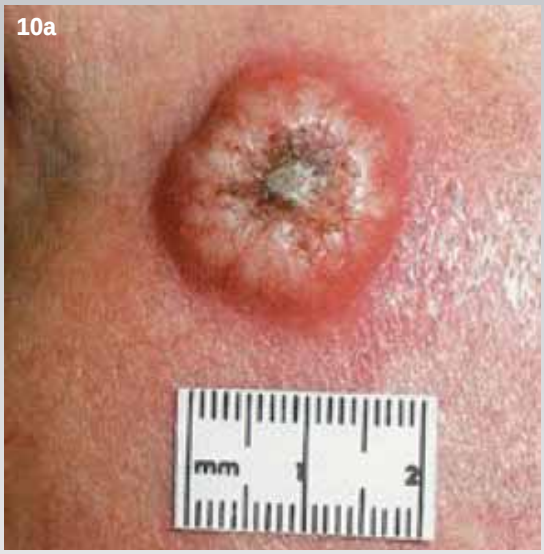
This lesion presents as a bud-shaped or dome-shaped lesions with a central keratinous crater (Figs. 10a–b). Rapid growth occurs within a period of six weeks, followed by a period of involution over 4–6 months, leaving a pitted scar.[25,26] Progression to squamous cell carcinoma is rare.[26]

Histology

It is composed of singularly well-differentiated squamous epithelium with little pleiomorphism and anaplasia with masses of keratin. Pseudocarcinomatous infiltration does not extend beyond the level of hair follicles and cutaneous glands.

Treatment

Surgical excision, intralesional 5-fluorouracil, bleomycin, steroids and methotrexate or systemic isotretinoin. Low-dose irradiation can also be employed.[26]



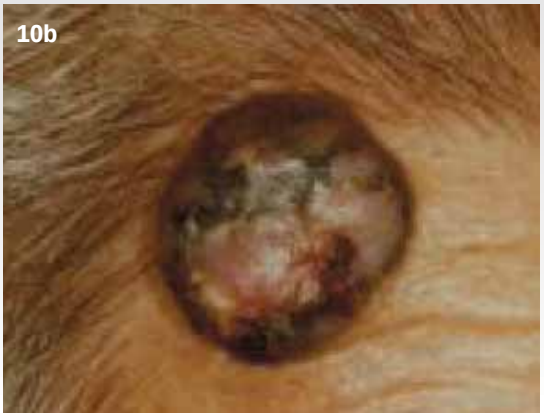


Fig. 10 Photographs show (a) umbilicated keratocanthoma at the cheek; and (b) keratocanthoma at the forehead.

Sebaceous naevus of Jadassohn

Aetiology

This may arise from pluripotential primary epithelial germ cells. Mutations in these cells may give rise to hamartomas with multiple cell lines.

Clinical presentation

Presents as a yellow-brown lesion with a linear configuration (Figs. 11a–c). Its development may be related to hormonal fluctuation—raised appearance at birth then flattened during childhood and raised again during puberty. 5%–7% may develop into BCC. May also develop into many other benign and malignant tumours.

Histology

Papillomatous hyperplasia is present in the epidermis with numerous sebaceous glands in the dermis. Apocrine glands, small hair follicles and buds of basaloid cells are also present.

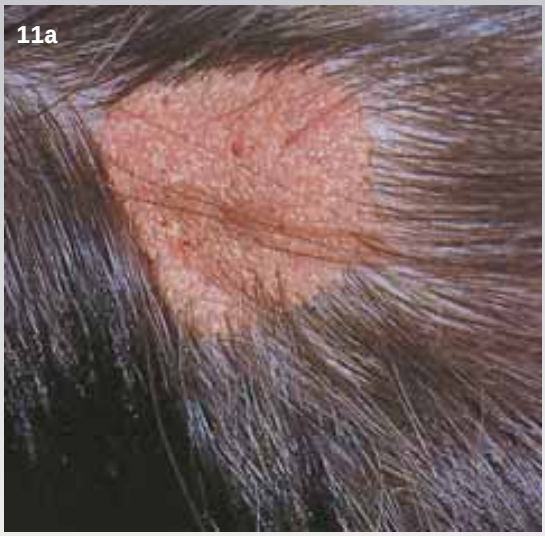


Fig. 11 Photograph shows (a) sebaceous naevus at scalp for case 1.

Treatment

Surgical excision before puberty, after which the risk of malignancy increases.

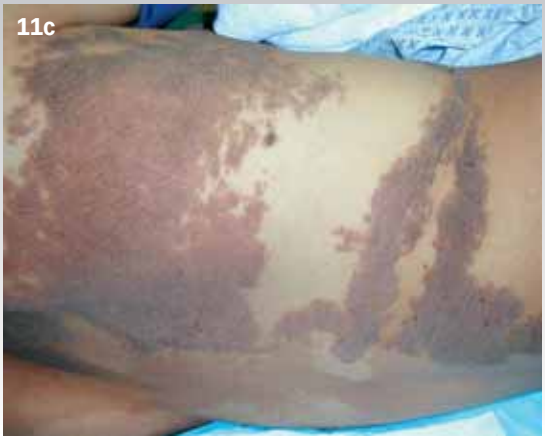


Fig. 11 Photographs show sebaceous naevus (b) at scalp for case 2; and (c) at trunk for case 3.

MALIGNANT SKIN LESIONS

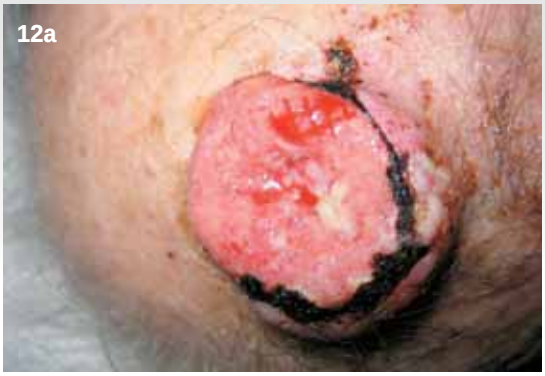
Squamous cell carcinoma

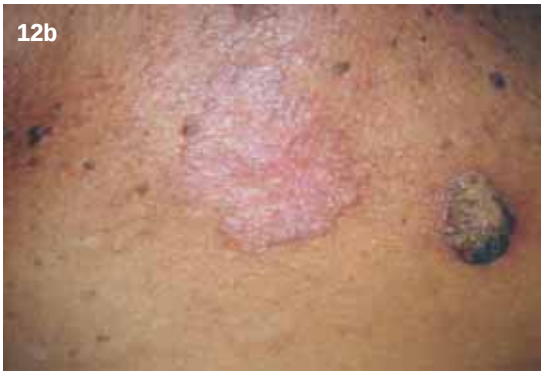
Aetiology

Ultraviolet irradiation from sun exposure, infrared irradiation or X-ray; chemical carcinogens such as arsenic, hydrocarbons, tar and pesticides; viral agents such as human papilloma virus; chronic wounds such as Marjolin's ulcer and chronic scars; and impaired immunity either from immunosuppression or immunodeficiency.[27,28]

Clinical presentation

It is the second commonest skin cancer in Singapore.[1] Invasive squamous cell carcinoma (SCC) can arise *de novo* or from precursor lesions, such as actinic keratosis, Bowen's disease, erythroplasia of Queyrat, leukoplakia and intraepidermal epithelioma. Depending on the site of occurrence, it can present as a raised, pink-coloured keratotic papule or plaque; an area of induration with erosion and ulceration (oral SCC); moist red plaques with subsequent induration and ulceration (anogenital SCC); white plaque (intraoral leukoplakia); or exophytic fungating lesions (verruca variant of SCC). As these lesions progress, they become locally invasive and destructive, fixed to underlying and adjacent structures and metastasise to distant sites (Figs. 12a–c).





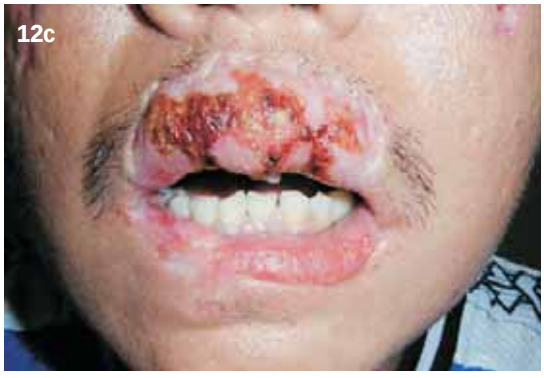


Fig. 12 Photographs show (a) SCC at scalp; (b) Bowen's disease with SCC; and (c) SCC at the upper lip.

Histology

There is proliferation of atypical keratinocytes in architectural disarray. Hyperkeratosis, parakeratosis and acanthosis is present. The atypical keratinocytes display pleiomorphism, hyperchromatic nuclei and mitoses. Keratinisation also occurs, forming horn pearls composed of concentric layers of squamous cells and keratin. Histological variants of SCC include adenoid, spindle cell, clear cell, papillary and signet ring SCC.[5]

Treatment

Surgical excision with a 4–6 mm margin, cryotherapy, electrodesiccation and curettage and Mohs micrographic surgery can be performed with excellent cure rates.[27] Other treatment modalities include laser ablation, photodynamic therapy, local immunotherapy (Imiquimod) and local chemotherapy (5-FU). Prevention by decreased exposure to UV radiation should be advised. Chemoprevention with oral retinoic acid agents are being evaluated.[29]

Basal cell carcinoma

Aetiology

Basal cell carcinoma (BCC) arises from the pluripotential cells of the basal cell layer of the epidermis and follicular structures. UV-induced mutation of the p53 gene and tumour suppressor genes in chromosome 9 (Gorlin syndrome, naevoid basal cell carcinoma) and mutations of the patched (PTCH) gene in the patched hedgehog pathway have been implicated. Arsenic exposure and immunosuppression also increases the risk of BCC.[5,30]

Clinical presentation

BCC (Figs. 13 a–c) is the commonest skin cancer in Singapore with the commonest subtype being *nodular BCC*. It is characterised by a dome-shaped papule with telangiectasia and pearly-white border associated with a central crusted or ulcerated area. Other clinical variants of BCC include:

a) *Superficial BCC*: Erythematous scaly plaque with a raised border and occasional central ulceration.
b) *Pigmented BCC*: Presence of brown and black pigment within the BCC.
c) *Cystic BCC*: presents as blue-gray cystic nodules.
d) *Micronodular BCC*: Clinically similar to nodular BCC but associated with subclinical extension through the dermis which may be widespread.
e) *Morpheaform/sclerosing BCC*: Presents as a whitish plaque with ill-defined margins and associated with aggressive growth.

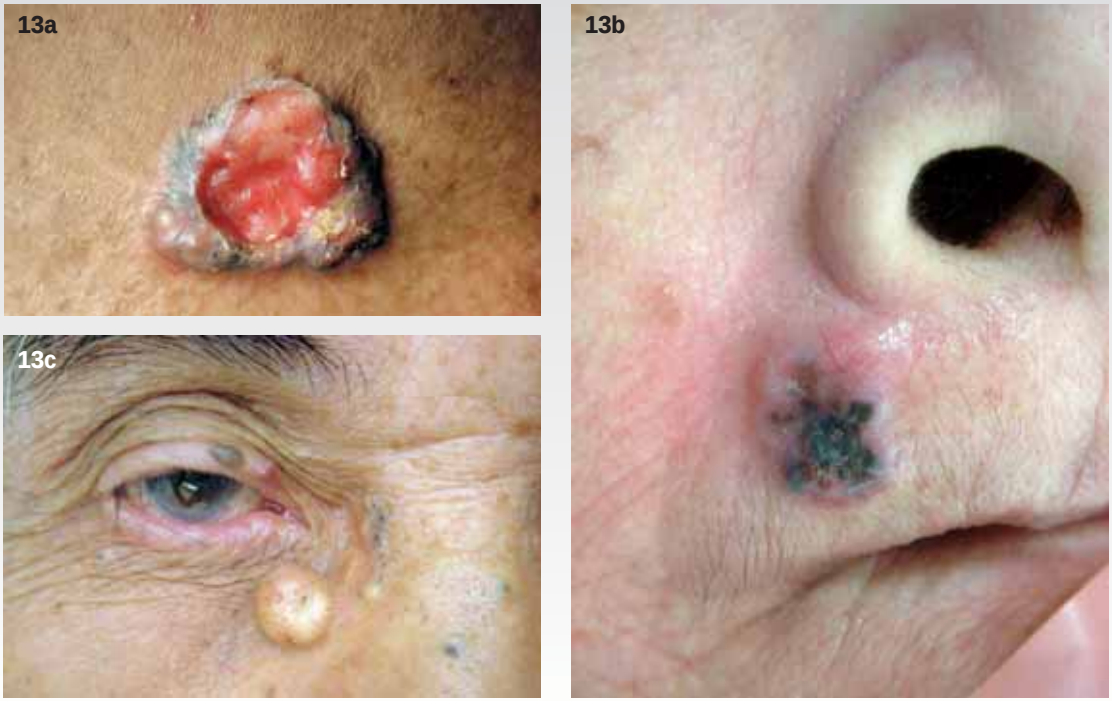


Fig. 13 Photographs show (a) BCC with rodent ulcer; (b) pigmented BCC; and (c) nodular BCC.

Histology

The cells have large, oval hyperchromatic nuclei with scanty cytoplasm. The peripheral cells are arranged in a palisading pattern. Tumour masses are surrounded by mucinous stroma upon which tumour growth is dependent. Chronic inflammatory infiltrates are also commonly seen around the tumour. High risk of recurrence is seen in the infiltrative tumour edge, tumour cells forming strands (morpheaform pattern), poor or absent peripheral palisading and marked nuclear pleiomorphism.

Treatment

Surgical excision with a 4-mm margin,[31] Mohs micrographic surgery, cryotherapy and electrodesiccation with curettage. Topical–FU, Imiquimod, radiotherapy and intralesional interferon alfa–2b,[32,33] have been used to treat superficial BCC.

Malignant melanoma

Aetiology

Risk factors include family history, multiple benign or atypical naevi, previous melanoma, immunosuppression, sun sensitivity and UV irradiation. The progression of a benign naevus to malignant melanoma occurs in a stepwise fashion. N-Ras (retrovirus-associated DNA sequences), BRAF (regulation of α-foetoprotein) gene mutations and abnormal activation of MAPK (mitogen-activated protein kinase) result in melanocyte hyperplasia. Mutations of CDKN2A (cyclin-dependent kinase inhibitor 2A), resulting in inactivation of tumour suppressor genes p16 and p19 and PTEN (phosphatase and tensin homologue), would then result in cytologic atypia and formation of dysplastic naevus. Increased PKB (protein kinase B) activity and increased cyclin D1 expression results in uncontrolled hyperplasia, clonal proliferation and decreased differentiation, precipitating in a radial growth phase. E-cadherin loss, with increased N cadherin and αVβ3 integrin expression results in a vertical growth phase. Finally, the absence of TRPM1 (melastatin 1) results in metastatic spread.[34]

Clinical presentation

Melanoma (Figs. 14 a–c) is rare in Singapore, and can be classified into several types as follows:

a) *Superficial spreading melanoma*: Most common type. Presents as a deeply pigmented macule or slightly raised plaque with colour variegation.
b) *Lentigo maligna melanoma*: Least common. Occurs in the older age group. Presents as tan, brown or black flat lesions with convoluted borders and prominent notching.
c) *Acral lentiginous melanoma*: More common in darker pigmented individuals. Occur in the sole, palm or beneath the nail plate. Presents as tan, brown or black flat lesions or nodules/papules. Hutchinson's sign (pigmentation of the posterior nail fold) in subungal melanoma is an ominous sign.
d) *Nodular melanoma*: Commonly arises *de novo* or from pre-existing naevi. Presents as a dark blue-black or bluish-red nodule or papule or even as a polypoid lesion with a stalk.
e) *Amelanotic melanoma*: Melanomas which lack pigment altogether.

Histology

a) *Superficial spreading melanoma*: Malignant melanocytes expand in multiple layers within the epidermis, and superficial papillary body of the dermis, in the radial growth phase. In the vertical growth phase, they extend into the reticular dermis and beyond.
b) *Lentigo maligna melanoma*: Consists of atypical melanocytes initially lined in a single layer along the basal layer above the basement membrane, and subsequently invades the dermis and deeper tissues.
c) *Acral lentiginous melanoma*: Macular areas consist of large melanocytes with large atypical nuclei and elongated dendrites which extend to the granular layer. In the papular/nodular areas, the melanocytes assume a spindle shape and extend into the dermis.
d) *Nodular melanoma*: It arises in the dermo-epidermal junction and invasion of the dermis may also occur with invasion of the epidermis. Consists of large epitheloid cells, spindle cells, small cells or combinations of these.

Treatment

Patients should be staged. Stages I and II denote local cutaneous disease, stage III disease involves regional nodes, while stage IV disease is associated with distant metasases. The depth of invasion can be classified according to the Breslow or Clark levels. The treatment of melanoma is surgical excision with a margin of 0.5–2 cm, depending on the thickness of the tumour. Sentinel lymph node biopsy aid in prognostication, but both elective lymph node or sentinel lymph node dissection do not confer survival advantage. Lymph node dissection can be performed in recurrent disease to improve local palliative control. Interferon α-2β is the only FDA approved adjuvant therapy in patients with high risk of disseminated disease. It increases disease-free survival but not overall survival.[35]

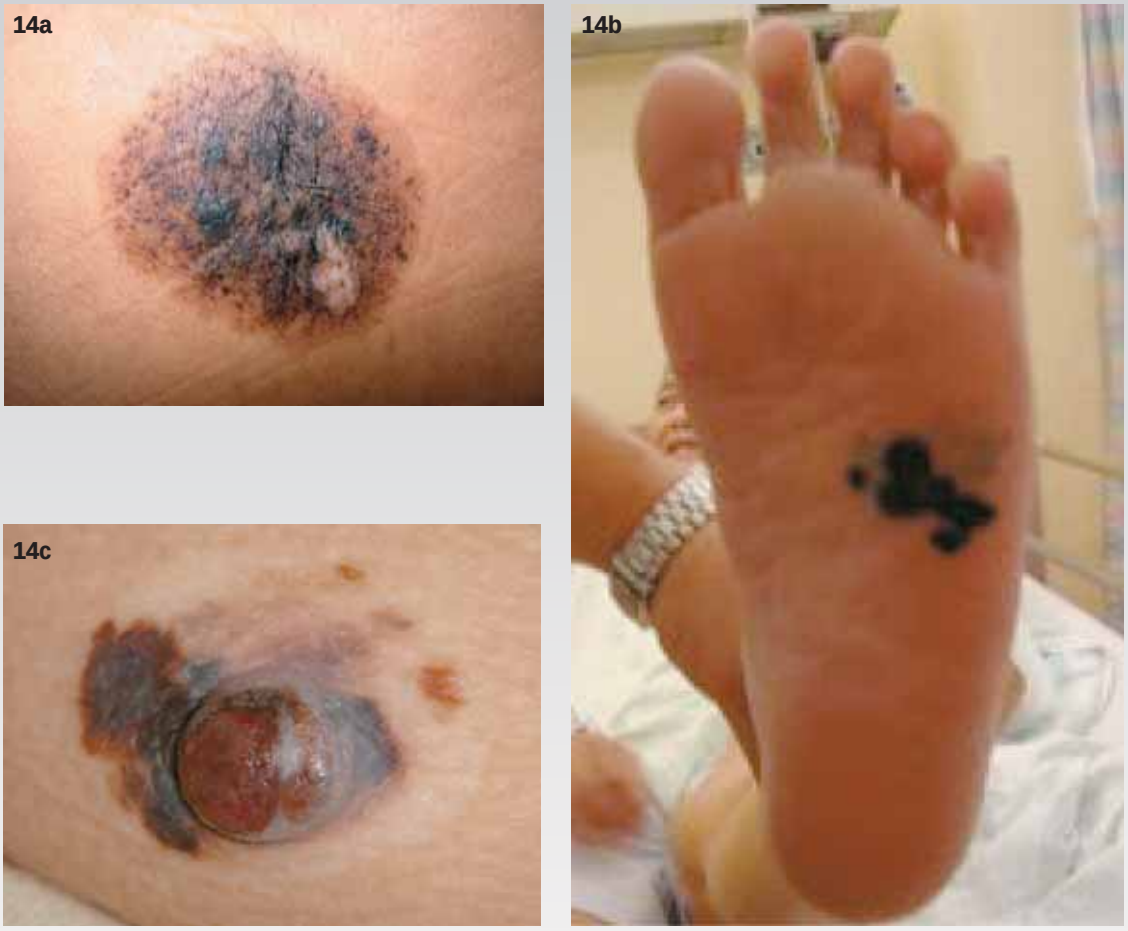


Fig. 14 Photographs show (a) superficial spreading melanoma; (b) acral lentiginous melanoma at the left sole; and (c) nodular melanoma.

CONCLUSION

It is sometimes difficult to differentiate benign from malignant skin tumours. Clinical features suggestive of malignancy include rapid growth in size, change in colour, presence of satellite nodules, asymmetry, contact bleeding and itch. Malignant skin cancers are also more common among fair-skinned individuals and hence, among the Chinese race in Singapore.[36] A high index of suspicion must be maintained, especially in fair-skinned patients. Suspicious lesions should be biopsied to obtain definitive histological diagnosis. As malignant lesions frequently resemble one another rather closely, histological analysis may be the only means of reaching a definitive diagnosis.

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SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME

Multiple Choice Questions (Code SMJ 200801A)

	True	False
Question 1. The following are features suggestive of skin malignancy:		
(a) Rapid growth in size.	☐	☐
(b) Well-demarcated and regular margins.	☐	☐
(c) Presence of satellite nodules.	☐	☐
(d) Atypical site of occurrence on the body.	☐	☐
Question 2. Indicate whether the following statements are true or false:		
(a) Skin cancers are more common among the Malay and Indian races.	☐	☐
(b) Malignant melanoma is common in Singapore.	☐	☐
(c) The most common skin cancer among Singaporeans is squamous cell carcinoma.	☐	☐
(d) The incidence rate of skin cancers among Singaporeans is increasing gradually.	☐	☐
Question 3. The following are subtypes of basal cell carcinoma:		
(a) Superficial spreading.	☐	☐
(b) Nodular and micronodular.	☐	☐
(c) Epitheloid.	☐	☐
(d) Morpheaform.	☐	☐
Question 4. The following skin tumours are caused by sunlight-induced damage:		
(a) Bowen's disease.	☐	☐
(b) Seborrhoiec keratoses.	☐	☐
(c) Marjolin's ulcer.	☐	☐
(d) Basal cell carcinoma.	☐	☐
Question 5. Indicate whether the following statements are true or false:		
(a) Xanthomas are caused by LDL deposition in tissues.	☐	☐
(b) Haemangiomas grow rapidly from birth up to 18 months, and subsequently involute over a period of 2–6 years.	☐	☐
(c) Cutaneous horns are never associated with any malignant potential.	☐	☐
(d) The risk of malignant change of a naevus is higher when it involves more than 5% of the body surface or is larger than 20 cm.	☐	☐

Doctor's particulars:

Name in full: _____

MCR number: _____ Specialty: _____

Email address: _____

SUBMISSION INSTRUCTIONS:
(1) Log on at the SMJ website: http://www.sma.org.sg/cme/smj and select the appropriate set of questions. (2) Select your answers and provide your name, email address and MCR number. Click on "Submit answers" to submit.

RESULTS:
(1) Answers will be published in the SMJ March 2008 issue. (2) The MCR numbers of successful candidates will be posted online at www.sma.org.sg/cme/smj by 15 March 2008. (3) All online submissions will receive an automatic email acknowlegment. (4) Passing mark is 60%. No mark will be deducted for incorrect answers. (5) The SMJ editorial office will submit the list of successful candidates to the Singapore Medical Council.

Deadline for submission: (January 2008 SMJ 3B CME programme): 12 noon, 25 February 2008.