

The summer skin check



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BACKGROUND

Skin cancer is a major public health problem in Australia and represents a substantial health cost. General practitioners provide the majority of care to patients with skin cancer, so becoming familiar with the clinical features and management of these tumours is important.



To provide a pictorial essay on the common types of benign and malignant skin lesions encountered in general practice, and briefly describe key clinical features, differential diagnosis and management options.

DISCUSSION

Examination for skin cancer should be considered in the general practice setting for all patients over the age of 40 years, particularly the elderly. A proper skin check requires a systematic approach and ideally an entire consultation should be set aside for this purpose. However, clinical examination conducted for other purposes provides an opportunity for screening and early detection of skin cancer.

History taking

The following points in history taking can help identify high risk patients:

- blistering sunburns
- occupational and recreational exposure to solar radiation
- family or personal history of melanoma or multiple atypical (dysplastic) naevi
- family or personal history of nonmelanoma skin cancer (NMSC)
- immunosuppression higher rates and earlier onset of skin cancer is present in transplant patients,¹ therefore a high index

of suspicion is necessary in this population

- exposure to arsenic is a known risk factor for the development of basal cell carcinoma (BCC)² and Bowen disease³
- a history of change or symptomatology in any lesion – skin cancers are changing lesions and the time course for this change is generally evident over a period of months.

Examination

The following factors are important in examination:

- good lighting, preferably natural light
- magnification a dermatoscope can be helpful in diagnosis of both melanocytic and nonmelanocytic lesions
- positioning patients should be undressed to their underwear, so a warm room or blanket for comfort is desirable. Having the patient lie on the examination couch can be useful so the soles of the feet are not missed
- look closely gently stretching the skin can often make BCCs more obvious.
- palpate dermatofibromas for example have a characteristic feel on palpation
- concentrate on high risk areas the highest rates for NMSC are found on the face, especially the nasolabial fold, eyelid and the nonmucosal skin of the lip followed by the ears, nose and cheek. In men, the neck, back and shoulders, and

- in women, the neck, shoulders and outer arms are also sites of predilection⁴
- scars should be checked for evidence of recurrence of previously excised lesions -BCCs may develop within longstanding scars and ulcers^{5,6}
- lymph nodes should be assessed for the early detection of metastatic disease if there is a history of melanoma or squamous cell carcinomas (SCCs).

Management

Biopsy of a lesion (punch or shave) is advisable when a firm clinical diagnosis cannot be made or when the treatment choice may be dictated by the tumour type or pattern of growth. Multiple 2 mm punch biopsies may be necessary to define poorly demarcated tumours. Tailor each management decision to the particular lesion in the individual patient. In general, simple surgical excision with primary closure is the treatment of choice for most skin cancers, although some anatomical areas require particular care (Table 1) and specialist referral may be desirable for technically difficult excisions. Prevention should be emphasised (Table 2) and changes to look for in suspicious lesions discussed. Follow up is recommended for three reasons: detection of further primary tumours, detecting local persistence of previously treated tumours, and early detection of metastatic disease.

Table 1. Problem areas requiring care in management

Face – for cosmetic reasons and the possibility of nerve damage

Eyelids – scarring can cause entropion **Lips and helix of the ear** – because of malignant potential

Neck – where the accessory nerve is superficial

Fingers – function is a major consideration

Lower limb - healing can be very slow

Table 2. Recommendations for prevention of skin cancer

Where possible use clothing as the primary means of photoprotection – preferably closely woven fabrics that aren't transparent when held up to the light

Wear hats and sunglasses Seek shade. Avoid the sun in the middle of the day

Use sunscreen – broad spectrum SPF15 or greater should be applied to all exposed areas of the skin as a last line of defence. Reapply every 2 hours Advise against the use of sunbeds, tanning booths and tanning lamps



Figure 1. Seborrhoeic keratosis

Seborrhoeic keratoses Clinical features

Seborrhoeic keratoses (*Figure 1, 2*) are very common benign tumours usually found on the face and trunk. They develop gradually as slightly elevated, ovoid yellow-brown, well defined papules with a greasy appearance,



Figure 2. Seborrhoeic keratosis

later thickening to become brownish-black. The surface is irregular and shows minute pits.

Management

When flat and pigmented, seborrhoeic keratoses may require biopsy to differentiate them from lentigo maligna melanoma. These can be removed with a number of destructive techniques such as cryotherapy, curettage, electrosurgery or shave excision.

Multiple eruptive seborrhoeic keratoses are rare but may be a sign of internal malignancy (Leser-Trelat sign)

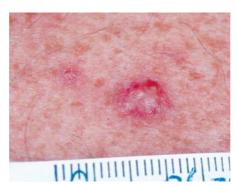


Figure 3. Dermatofibroma

Dermatofibroma Clinical features

Dermatofibroma (*Figure 3*) are benign tumours of fibroblasts that are firm, solitary dermal nodules usually found on the limbs of younger people. They have an 'iceberg' effect in that they feel larger than they look. The overlying epidermis is often lightly pigmented and dimples when the nodule is squeezed.

Management

No treatment is usually necessary, although biopsy may be performed if the diagnosis is uncertain.

Recurrence after excision is not uncommon as clinical margins are often underestimated



Figure 4. Porokeratosis

Porokeratoses Clinical features

Porokeratoses (*Figure 4*) are uncommon, distinctive, benign lesions due to clones of epidermal cells that show varying degrees of dysplasia. They develop as annular dry plaques surrounded by a raised fine keratotic wall. Porokeratoses may be isolated or multiple and most often appear on the limbs.

Management

None usually needed. If lesions are hyperkeratotic or multiple, cryotherapy may be performed.

The rare giant forms may be premalignant



Figure 5. Solar keratosis

Solar keratoses

Solar keratoses (*Figure 5*) are common lesions associated with dysplasia, particularly within basal keratinocytes. They represent a potential precursor of SCC, although only a small percentage evolve into invasive SCCs.

Clinical features

Solar keratoses are found on the chronically sun exposed sites of the head and neck, dorsa of the hands, and the forearms, and present as erythematous macule with superimposed hyperkeratosis. Solar keratoses are generally multiple and may be confluent.

Differential diagnosis

Bowen disease, SCC, solar lentigo or lentigo maligna if pigmented.

Management options

- No treatment
- Cryotherapy
- 5-flurouracil
- Imiguimod
- Curettage
- · Laser resurfacing.

Thickening and tenderness on lateral palpation are indicators of possible transformation into invasive SCC



Figure 6. Bowen disease

Bowen disease

Bowen disease (intraepidermal SCC) (Figure 6) is associated with full thickness epidermal dysplasia with follicular involvement and is classified as an intraepidermal SCC. Despite this, they tend to have a prolonged in-situ phase that may last many years.

Clinical features

Longstanding, slowly enlarging lesions that are generally asymptomatic with a predilection for the lower legs (especially in females) but may occur at any site. Lesions are a sharply defined, round to oval, erythematous hyperkeratotic plaque. The degree of hyperkeratosis may vary. The rate of transformation

to invasive SCC has not been established, but appears to be low.⁷

Differential diagnosis

- Superficial BCC
- Psoriasis
- SCC
- If pigmented, may mimic superficial spreading melanoma.

Management options

- Surgical excision
- Cryotherapy
- Imiquimod
- 5-flurouracil
- Curettage and diathermy
- Photodynamic therapy
- Superficial radiotherapy
- Laser ablation.

Due to extension of keratinocyte atypia, down hair follicles recurrence after treatment with superficial modalities (eg. cryotherapy) can occur, particularly on the face



Figure 7. Keratoacanthoma

Keratoacanthoma

Keratoacanthoma (*Figure 7*) are lesions that can be thought of as a well differentiated forms of SCC characterised by spontaneous resolution.

Clinical features

Keratoacanthoma are most common on the chronically exposed sites of the head, neck, hands, and forearms. They rapidly enlarge over a period of 4–8 weeks to form a tender, erythematous nodule with a central keratotic plug. Resolution takes 6–12 weeks, sometimes leaving a scar. A minority will persist as SCC.

Differential diagnosis

- SCC
- Atypical fibroxanthoma
- Merkel cell carcinoma
- Amelanotic melanoma.

Management options

- Partial biopsy will almost always be reported as a well differentiated SCC as pathology requires the entire architecture to suggest the possibility of keratoacanthoma. If in doubt, treat as a SCC
- Curettage +/- cryotherapy
- Shave excision
- Surgical excision
- Intralesional methotrexate
- Superficial radiotherapy.

Extra caution is required in immunosuppressed patients as keratoa-canthomas may progressively grow rather than involute¹⁰



Figure 8. Squamous cell carcinoma



Figure 9. Squamous cell carcinoma

Squamous cell carcinoma

Squamous cell carcinomas (*Figure 8, 9*) are characterised by lobular proliferation of keratinocytes with intradermal invasion and prominent keratin production. The majority of SCCs are thought to arise from solar keratoses.⁸

Clinical features

Squamous cell carcinomas are characterised by tender erythematous papules or nodules, with a variable amount of hyperkeratosis enlarging over a period of months, usually with associated tenderness. Bleeding and ulceration may develop and the risk of metastasis increases with tumour thickness, clinical immunosuppression and poorly differentiated tumours.⁹

Differential diagnosis

- Hyperkeratotic solar keratosis or Bowen disease – clinical diagnosis can be difficult in the early stages
- Nodular basal cell carcinoma
- Amelanotic melanoma
- Atypical fibroxanthoma particularly if on the scalp.

Management options

- Surgical excision
- Superficial radiotherapy.

Beware tumours located on lips, ears or scalp as they have a higher risk of early metastasis usually to local lymph nodes⁹

Basal cell carcinomas

Basal cell carcinomas (BCCs) are tumours derived from basal keratinocytes. Their onset is insidious. In months or years they extend peripherally as well as in depth and are capable of destroying cartilage and bone. Metastases rarely develop from BCCs,¹¹ the main risk being recurrence after incomplete removal. There are three common growth patterns of BCC (superficial multifocal, nodular and morphoeic) that have a distinctive clinical presentation. Any of these may also show ulceration or pigmentation.



Figure 10. Superficial BCC

Superficial multifocal BCC

Clinical features

Superficial BCCs generally occur on the trunk and limbs, are bright pink and shiny, and usually well defined erythematous macular lesion (*Figure 10*). Stretching the skin will highlight a thread-like pearly rim or islands of pearliness throughout the lesion. There is progressive enlargement over months to years but ulceration and bleeding is uncommon. With time, areas of nodular or even sclerosing growth pattern may supervene. Recurrence is common.

Differential diagnosis

- Bowen disease
- Solar keratosis
- Psoriasis
- Eczema.

Management options

- Cryotherapy
- Imiquimod
- 5-flurouracil
- Photodynamic therapy
- Surgical excision
- Curettage and diathermy
- Superficial radiotherapy.



Figure 11. Nodular BCC

Nodular BCC

Clinical features

Nodular BCCs (Figure 11) are more often found on the head and neck. They are a shiny, translucent (pearly), telangiectatic papule or nodule. Recurrent ulceration is frequent and pigmentation may suggest melanoma.

Differential diagnosis

- Nonpigmented intradermal naevi
- Neuromas
- SCC

Amelanotic nodular melanoma.

Management options

- Surgical excision
- Superficial radiotherapy
- Intralesional interferon alfa
- Imiguimod
- Photodynamic therapy.



Figure 12. Morphoeic BCC

Morphoeic BCC

Histology

Morphoeic lesions have a sclerosing growth pattern with fibrosis surrounding areas of BCC (*Figure 12*).

Clinical features

Morphoeic lesions present mostly on the head and neck and can be clinically difficult to detect. They are often indurated to palpation, are frequently asymptomatic and tend to look like a pale scar. Morphoeic lesions are usually longstanding and tend to be deeply invasive.

Differential diagnosis

Major differential diagnosis is a scar and biopsy is necessary to establish the diagnosis.

Management options

- Consider referral for specialist care
- Moh's micrographic surgery
- Superficial radiotherapy.

Morphoeic lesions need to be treated with respect as they can have extensive subclinical extension and perineural invasion particularly when located around the eyes, ears and nose. Recurrence is frequent due to incomplete excision



Figure 13. Solar lentigo

Solar lentigo Clinical features

Solar lentigo (*Figure 13*) represent a macular area of pigmentation appearing after either acute or chronic sun exposure. Histologically there is a linear increase of melanocytes at the dermoepidermal junction without cytologic atypia. Backs of the hands, face and shoulders are common sites. Over time they may evolve into seborrhoeic keratoses.

Management options

- Cryotherapy can lighten solar lentigo, but care should be taken in olive skinned patients as they may hyperpigment
- Laser

Shave biopsy may be necessary to distinguish solar lentigo from lentigo maligna or superficial spreading melanoma



Figure 14. Ink spot lentigo

Ink spot lentigo

Ink spot lentigo (*Figure 14*) are small (less than 5 mm) densely black macules, usually on sun exposed skin. They can be cause for concern because of their dark colour and irregular lateral margin. Melanocyte numbers and melanin production are increased in these lesions but there is no cellular atypia.

Although often conspicuous, ink spot lentigo are benign lesions



Figure 15. Atypical naevi

Atypical naevi Clinical features

Atypical (dysplastic) naevi (*Figure 15*) are usually larger than ordinary naevi, predominate on the trunk and often showing a mixture of tan, dark brown and pink areas. The surface texture is often 'pebbly' and the border 'smudgy'. The term atypical naevus syndrome refers to the occurrence of multiple (up to 80 or more) dysplastic naevi in an individual. Melanoma risk is increased in these patients¹² with the risk increased further in melanoma prone families with atypical mole syndrome.¹³

Management

Surveillance (baseline photography and regular 6–12 month follow up) enables early diagnosis of melanoma. This is much more cost effective in preventing life threatening melanoma than prophylactic excision of atypical naevi.¹⁴

Due to the increased risk of melanoma in these patients regular follow up is necessary for life

Melanoma

Patients are often most concerned about pigmented lesions because of the possibility of melanoma and its capacity to be lethal. *Table*

Table 3. Margins of re-excision for melanoma after initial excisional biopsy

Tumour thickness	Margin of re-excision
In situ (level 1)	0.5 cm
≤2.0 mm	1.0 cm
>2.0 mm	2.0 cm



Figure 16. Lentigo maligna melanoma

3 lists recommended excision margins. The four most common subtypes are:

Lentigo maligna melanoma

Lentigo maligna melanoma (*Figure 16*) account for 5–15% of all melanomas and occur mainly on the exposed skin of the elderly. An irregularly pigmented and shaped macule (lentigo maligna/Hutchinson melanotic freckle) may have been enlarging slowly for many years as an in-situ melanoma before an invasive nodule (lentigo maligna melanoma) appears.

Differential diagnosis

- Solar lentigo
- Flat pigmented seborrhoeic keratosis.

Management options

- Shave biopsy of the lesion's centre is recommended if there is diagnostic uncertainty
- Surgical excision
- Superficial radiotherapy.

Superficial spreading melanoma

Superficial spreading melanoma account for 50–75% of all melanomas and may develop on any part of the body (*Figure 17*) at any age. They have variegated colour and an irregular expanding margin.

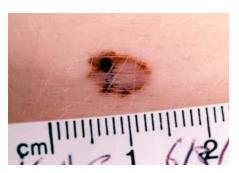


Figure 17. Superficial spreading melanoma

Differential diagnosis

- · Atypical (dysplastic) naevus
- Pigmented BCC
- Pigmented seborrhoeic keratosis
- Haemangioma.

Management

Surgical excision.

Nodular melanoma

Nodular melanoma represent 15–35% of all melanomas and appear as a pigmented (or occasionally flesh coloured) amelanotic nodule with no antecedent in-situ phase (*Figure 18*). It is the most rapidly growing and aggressive type of melanoma.

Differential diagnosis

- Pigmented BCC
- SCC or pyogenic granuloma if amelanotic.

Management

• Surgical excision.

Acral lentiginous melanoma

Acral lentiginous melanoma represent 5–10% of all melanomas and occur on the palmar, plantar and subungal skin (*Figure 19*). Although rare in caucasians, they are particularly common in both the Chinese and Japanese population.

Differential diagnosis

- Melanocytic naevus
- Subungal heamatoma
- Talon noir a pigmented petechial area on the heel following minor trauma.

Management

Surgical excision.

Partial biopsy of the suspected melanoma should be avoided as this will interfere with accurate histological staging

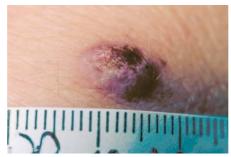


Figure 18. Nodular melanoma



Figure 19. Acral lentiginous melanoma

Acknowledgment

Thanks to Professor Robin Marks from St Vincent's Hospital Melbourne and Associate Professor John Kelly, Victorian Melanoma Service, Alfred Hospital, Melbourne, for kindly allowing reproduction of some of the clinical images in this article.

Suggested reading

National Health and Research Medical Council. Clinical Practice Guidelines. Nonmelanoma skin cancer: guidelines for treatment and management in Australia. Canberra: Commonwealth of Australia, 2003. Available through Ausinfo Government Info bookshops Australia wide, phone toll free 132 447 or at: www.noie.gov.au/projects/egovernment/Better_Information/infoaccessnetwork/index.html

Conflict of interest: none declared.

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