# Skin Cancer:
## A Lesson in Lesions

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As with any malignancy, the diagnosis of skin cancer is based upon a suspicious clinical presentation (signs, symptoms) that forms the basis for a tissue biopsy that may be complemented by special procedures (*e.g.*, staging). The physician's challenge regarding skin malignancies is that a multitude of skin lesions and blemishes appear in ever-increasing numbers with advancing age. The physician can, thus, be forgiven for subjecting the patient to a skin biopsy reported as a benign lesion, as an early diagnosis is always beneficial, irrespective of the cancer type.

This discussion will review clinical aids and management approaches that will help lead to the early diagnosis and eradication of basal cell carcinoma (BCC), squamous cell carcinoma (SCC) and keratoacanthoma (KA).



## BASAL CELL CARCINOMA

Basal cell carcinoma (BCC) (also called basalioma, basal cell epithelioma and rodent ulcer) is the most common cancer among Caucasians and accounts for about 75% of all skin cancers. Metastasis is rare (between 0.0028% and 0.1%), but BCC can be very aggressive locally, causing disabling and cosmetically disfiguring tissue destruction.

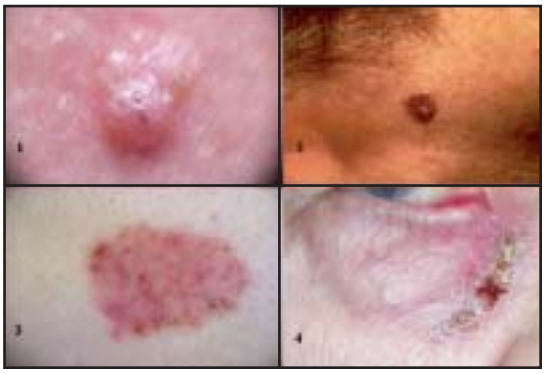

Figure 1.Clinical variants of BCC. Panel 1: Nodular BCC; Panel 2: Pigmented BCC; Panel 3: Superficial BCC; Panel 4: Rodent ulcer.

The precursor or stem cell for BCC is postulated to be hair follicle keratinocytes, hence the absence of BCC on hairless skin (*e.g.*, palms). Skin type (fair-skin that burns easily and does not tan), positive family history, age and ultraviolet light exposure are predisposing factors for BCC.

As a cautionary note, the recognized occurrence of BCC on sun-protected areas and in patients under 30 years of age points to the need for vigilance when examining all body sites in all age groups. Nonetheless, simple factors, such as skin type, family history, extent of sun damage and age (older than 50 years) should certainly arouse the physician's suspicion and provide a context for the skin examination.

Generally, BCC evolves over a period of at least a few months; we have witnessed new BCCs that were not clinically evident on a cutaneous examination conducted three to four months prior.

BCC presents as five main morphologic types:

- nodular,
- rodent ulcer,
- pigmented,
- superficial and
- morpheaform (Figure 1).

The salient clinical features of each are presented in Table 1 (see page 68). Attention to fine clinical detail helps in making an early diagnosis.

All suspicious lesions should be subjected to a skin biopsy (definitive or diagnostic). For the primary-care physician, a diagnostic punch biopsy (3 mm or 4 mm) provides an excellent tissue sample.

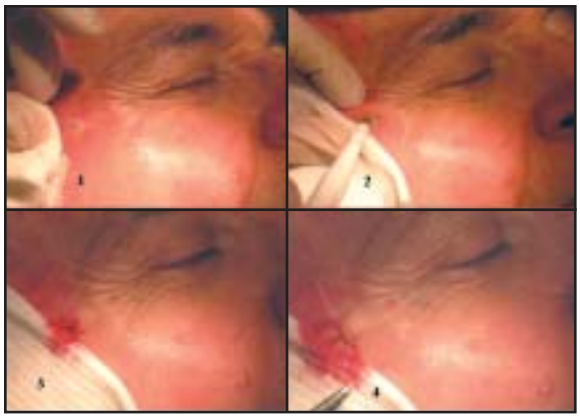

Figure 2. Performance of diagnostic punch biopsy of skin. Following cleansing of the biopsy site, to obtain a sterile field, local anesthesia is injected, generally 1% or 2% lidocaine with or without epinephrine (Panel 1). A 3-mm or 4-mm punch is used to obtain a core of skin down to subcutaneous fat (Panel 2); care must be taken in certain anatomic sites where nerves or vessels course superficially and are, hence, subject to damage. The defect (Panel 3) is then sutured with one or two nylon sutures (Panel 4). An appropriate dressing is then applied. Sutures are removed after approximately one week.

Figure 2 illustrates the steps in performing a punch biopsy of skin. Generally, a diagnostic biopsy is best taken from the edge of the suspicious lesion. The principal therapies for BCC include surgery, immunotherapy (*e.g.*, imiquimod, a topical product that enhances endogenous cytokine production), interferon-a (an inducer of apoptosis), topical 5-fluorouracil (also an inducer of apoptosis), radiotherapy and aggressive cryotherapy (superficial BCC only). Mohs microscopically controlled surgery is indicated for BCCs that are recurrent, of an aggressive subtype (*e.g.*, infiltrative) or occur in cosmetically or functionally-sensitive areas.

Counselling regarding sun exposure is relevant to prevent new BCCs, especially in younger patients.

## SQUAMOUS CELL CARCINOMA

Squamous cell carcinoma (SCC) is less common than BCC, but accounts for the majority of non-melanoma skin cancer deaths in North American Caucasians.

The main predisposing factors are:

- skin type (fair complexion, blue eyes, red hair, a poor tanning reaction),
- age (older than 40 years),
- immunosuppression,
- ultraviolet light and ionizing radiation exposure,
- scars and
- the human papilloma virus.

Incidence rates in men are double those in women. Precursor lesions include actinic keratoses (Figure 3) and Bowen's disease (SCC *in situ*; Figure 3). Morphologic variants of SCC include papular SCC, ulcerative SCC, nodular SCC, pigmented SCC, the cutaneous horn and verrucous SCC (Figure 3). Presentations deserving special mention are lip SCC, genital SCC and scar SCC.

Table 1 lists the salient features of the clinical presentations of SCC and precursor lesions. In general terms, metastatic risk is least for skin SCC (< 5%), but increases significantly for lip, genital and scar SCCs (> 15%). However, true metastatic risk must take into further consideration the tumour size and depth, the histologic grade and subtypes and invasion of blood vessels and nerves on pathologic examination.

Treatment modalities include surgical excision (margins of at least 4 mm) or Mohs microscopically controlled surgery for high-risk lesions (depth > 6 mm, diameter > 1 cm, transplant patients). Radiation therapy can also be applied either alone or as an adjuvant to surgery. If a diagnostic biopsy is performed, it should be of sufficient depth to identify invasive disease (*e.g.*, 4-mm punch biopsy to subcutaneous fat) in order to allow differentiation from *in situ* disease, and should be directed to the part of the tumour with the greatest depth clinically or to the heaped ulcer edge if ulcerative.

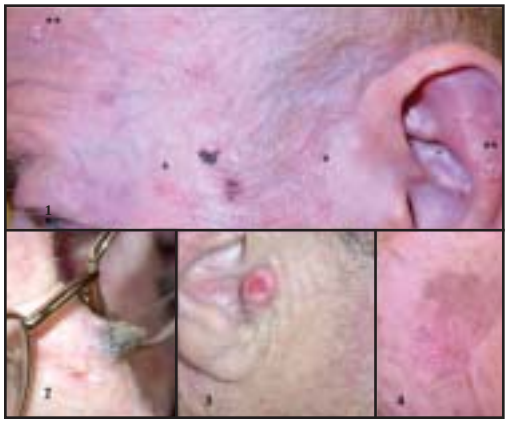

Figure 3. Clinical variants of SCC and precancerous skin lesions. Panel 1 illustrates actinic keratoses (single asterisk) and papular SCCs (double asterisk) on a background of severe actinic damage. Panel 2 shows a cutaneous horn on the bridge of the nose, Panel 3 a KA and Panel 3 a case of Bowen's disease (SCC *in situ*).

Following one SCC, the cumulative risk of another non-melanoma skin cancer is almost 50%, hence the necessity for close follow-up and counselling regarding sun protection.

## KERATOACANTHOMA

Many experts in this field view the KA as a subtype of SCC that grows rapidly at onset (can reach a few centimetres in weeks), occurs predominantly in elderly patients and favours sun-exposed areas, particularly the extremities.

Clinically, a KA resembles a miniature volcano—a dome-shaped nodule with a smooth surface and a central keratotic crater (Figure 4). Although viewed by many as a benign tumour, these are best treated as described above for the classic SCC. If the lesion is large and requires a diagnostic biopsy, it is suggested that a wedge-shaped section of the tumour be excised with the wide base at the edge of the KA and the central crater at the apex of the wedge.

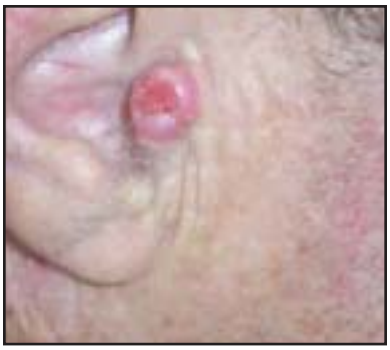

Figure 4. Keratoacanthoma.

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Turn the page for Table 1

Table 1

## Clinical features of variants of BCC and SCC *Bold comments are important determinants

### Basal cell carcinoma (BCC)

| | |
|---|---|
| Nodular | **Translucent** papule or nodule with superimposed **telangiectasia** and characteristic **rolled border**, most common on head and neck; the **rodent ulcer** (Figure 1, Panel 4) is a variant of nodular BCC with **central necrosis** giving rise to a slowly evolving, **non-healing ulcerated or eroded lesion**; usually **asymtomatic** (Figure 1, Panel 1) |
| Pigmented | Variant of nodular BCC with **hyperpigmentation**; must be differentiated from melanoma (Figure 1, Panel 2) |
| Superficial | Usually **solitary**, well-demarcated red plaque that **resembles eczema** (*e.g.*, nummular dermatitis), but usually not pruritic and without response to topical steroids; most common on the **trunk** (Figure 1, Panel 3) |
| Morpheaform | **Aggressive** variant of BCC that resembles a **scar** and is of an ivory-white colour; there is **absence of prior trauma or surgery** at the site; most common on the **face**; most difficult type of BCC to recognize clinically |

### Squamous cell carcinoma (SCC)

| | |
|---|---|
| Papular | **Firm**, flesh-coloured or red **keratotic** papule over **sun-exposed areas** (face, neck, dorsum of hands, arms); clinically appears and feels **superficial** (Figure 3, Panel 1) |
| Nodular | **Firm** lesion with significant **depth** and a smooth or keratotic surface; found over **sun-exposed areas**; commonly encountered with local **recurrences** of previously excised SCCs (usually smooth surface in such instances) |
| Ulcerative | **Ulcerated**, **non-healing** papule or nodule; **absence** of translucency and rolled edges help differentiate from BCC (rodent ulcer) |
| Pigmented | Any variant of SCC may be **pigmented**, making differentiation from **melanoma** difficult |
| Cutaneous horn | **Hard**, **keratotic projection** with significant elevation relative to the base of the lesion (Figure 3, Panel 2) |
| Verrucous | **Wart-like** lesion with keratotic, **filiform projections**; requires high index of suspicion leading to a diagnostic biopsy |

### Precancerous lesions

| | |
|---|---|
| Actinic keratosis | 2 mm to 6 mm, red, rough or **scaly papule**, usually on a background of significant sun damage (Figure 3, Panel 1); can be pigmented and coalesce into larger plaques that can appear as eczema; keratosis can be of other etiologies, including radiation, thermal, arsenical, scar and viral |
| Bowen's disease | Form of **SCC *in situ*** (*i.e.*, limited to epidermis) with potential to evolve to invasive SCC; presents as well-demarcated, discrete, red to pink plaque with irregular borders and overlying scale (Figure 3, Panel 4); may be pigmented; any body site can be involved, although sun-exposed areas are predisposed; risk to evolve to invasive SCC is estimated to be 5%; often misdiagnosed as plaque of psoriasis or eczema |