The management of malignant skin cancers

Shaheel Chummun
Neil R McLean

Abstract

Malignant skin cancers are common and are ever increasing annually. They can be divided into two main groups, non-melanoma skin cancers (NMSC), which include basal cell carcinoma and squamous cell carcinoma, and malignant melanoma. This paper reviews the various surgical and non-surgical treatment modalities available for the management of skin cancers.

Keywords Basal cell carcinoma; malignant melanoma; skin cancer; squamous cell carcinoma

Introduction

Skin cancer is one of the most common forms of cancer diagnosed in the United Kingdom, with large increases in the number of patients diagnosed in recent decades. Increased exposure to ultraviolet (UV) light remains the most important modifiable risk factor.

Skin cancer can be divided into two main groups, namely malignant melanoma (MM) and non-melanoma skin cancer (NMSC), of which basal cell carcinoma (BCC) and squamous cell carcinoma (SCC) are two of the most common types. Both BCC and SCC are caused by chronic and repeated exposure to sunlight, while malignant melanoma has a stronger association with sporadic intense sun exposure and a history of sunburn, instead of regular and prolonged exposure.

The use of sunbeds before the age of 35 has been shown to increase the risk of malignant melanoma and their use at any age increases the risk of squamous cell carcinoma. The Sunbeds (Regulation) Act 2010 makes it illegal for the under-18s to use sunbeds.

Epidemiology

Over the last 30 years, the incidence of malignant melanoma has quadrupled, and this increase has been more rapid than in any of the 10 commonest cancers in the UK. There has also been a steady increase in incidence with age, with highest rates noted in patients > 65 years. The increase in incidence has been more significant in MM with Breslow thickness <1 mm. Although this increase is partly due to increased detection and better public awareness, there is evidence that there may be a true rise in the incidence of MM. Mortality rates have also risen in the last 30 years and are worse in men, who tend to present late with thicker tumours.

NMSC is the most common skin cancer diagnosed in the white population worldwide, with BCC being four times more frequent than SCC. It is estimated that approximately 100,000 patients are diagnosed each year in the UK, with an average annual increase in incidence of 3–8%. Despite its high incidence, mortality from NMSCs is mainly from metastatic SCCs and accounts for <400 cases/year.

Premalignant lesions

The three most common premalignant lesions are actinic keratosis (AK) and Bowen's disease, which have the propensity to progress to SCC, and lentigo maligna (LM), which can evolve to lentigo malignant melanoma (LMM). These are caused by excessive UV light exposure, and are more common in patients with fair hair and light coloured eyes.

The skin consists of a superficial epidermal layer and a deeper dermal layer, separated by a basement membrane, with epidermal keratinocytes migrating upwards from the basement membrane. Any UV light damage can lead to aberrations in the keratinocyte differentiation process, producing what is clinically seen as AK and Bowen's disease. Lesions are referred to as being in-situ if the abnormal cells are restricted to the epidermis, and are malignant or invasive if the cells breach the basement membrane, where they can metastasize via the lymphatics and blood vessels. Invasive SCCs and melanomas frequently arise de novo.

Actinic keratosis (AK), also known as solar keratosis, occurs on sun exposed skin and 20% of fair-skinned individuals in the UK >60 years are estimated to have one or more AKs. AKs typically manifest as small, raised, scaly erythematous lesions over sun-exposed skin. Surrounding areas may show evidence of solar elastosis, such as telangiectasia, blotchy hyperpigmentation, and yellow discolouration of the skin. The actual rate of progression to SCC is unknown, and is thought to range between 0.1 and 16%; 25% of AKs are thought to regress spontaneously. Because of the variable natural history of AKs and the lack of clinical predictors to conversion to SCCs, they are best treated, either surgically or non-surgically.

Bowen's disease, known as SCC in-situ, is caused by prolonged exposure to sunlight, and unlike AK, has a greater female preponderance (3:1) and is more common on the legs. The rate of progression to invasive SCC ranges between 3% and 5%. Patients often present with an asymptomatic, slowly enlarging, erythematous, well-demarcated scaly patch or plaque. Bowen's disease also may occur on mucous membranes. Histological examination often shows features of thickened epidermis with an intact dermo-epidermal junction. A classic history is presentation of a non–steroid-responsive dermatosis. The diagnosis is often based on clinical features, and treatment is warranted because of its potential for progression to invasive SCC.

Lentigo maligna is a type of in-situ melanoma in which the abnormal melanocytes are restricted to the epidermis. Also known as Hutchinson's freckle, lentigo maligna usually occurs on sun-exposed skin and can be hard to distinguish from a solar lentigo. Between 5% and 15% of lentigo maligna will progress to lentigo maligna melanoma and surgical excision remains the

Shaheel Chummun *FRCS(Plast) MASurg(Cranio) is a BAAPS/BAPRAS Aesthetic Fellow at The Cadogan Clinic, London, UK. Conflict of interest: None.*

Neil R McLean *MD FRCS(Plast) is a Consultant Plastic and Reconstructive Surgeon at Wansbeck General Hospital, Ashington, UK. Conflict of interest: None.*

treatment of choice, although in some cases, radiotherapy may be advocated.

Dysplastic naevus

The dysplastic naevus (DN) remains a surgical conundrum; it shares the benign histological features of the benign common naevus, but also exhibits at the same time features of melanoma, including cytological atypia and dermal inflammatory response.[1] Clinically, it is very challenging to differentiate DN from a common naevus and a melanoma. Various therapeutic modalities have been tried in the management of DN, such as 5-fluouracil, imiquimod, isotretinoin and laser therapy. None has proven to be efficacious in ablating the DN.[1]

Surgical excision of the DN is commonly performed with the intention of removing the lesion. In cases of incomplete excision, the NIH Consensus Conference (1992) suggested a 2–5 mm re-excision for DN, although no indication for re-excision is defined. One of the reasons for re-excision is the concern that the lesion may represent a melanoma, based on either surgeon or patient's concerns or severe dysplasia on histology. Another reason for re-excision is to prevent recurrence, especially if it is thought that the dysplastic naevus may progress to a melanoma.

The National Institute of Health (2013)[1] has set out some recommendations for the management of dysplastic naevi. DN should be considered as a histological variant of the common naevus. Following biopsy, most dysplastic naevi do not require re-excision. However, clinically suspicious naevi should be excised, and those with severe histological dysplasia should be re-excised.

Basal cell carcinoma (BCC)

Basal cell carcinoma is the most common skin cancer in White populations, representing 75% of all skin cancers. They typically arise in the head and neck area (~83%), followed by the trunk and extremities; ~26% of all BCCs affect the nose alone. Histologically, BCCs arise from the basal layer of the epidermis. UV light plays a significant role in the development of BCCs, and patients with fair skin and light eye colour, who burn rather than tan, seem to be more predisposed to developing BCCs. However, the exact role of UV light is unclear, as patients who tend to develop BCCs usually show less features of photodamage, as is usually the case in patients with SCCs. Intermittent exposure to UV light in childhood and early adulthood may be more significant than chronic exposure, as is the case with SCCs. Other factors include advancing age, a family history of BCCs, immunosuppression in the form of AIDS or following immunosuppressive drugs, premalignant lesions (Naevus of Jadassohn, with a 10–15% risk of transformation to BCC) and the presence of predisposing conditions such as Gorlin's syndrome and xeroderma pigmentosum. Exposure to ionizing radiation, arsenic and hydrocarbons also contributes to the development of BCCs. The most common subtype of BCC is the nodular BCC (50–60%), which presents as a flesh coloured, pearly nodule with telangiectasia. Other subtypes include superficial spreading (15%), micronodular (15%), infiltrative (7%), pigmented (2%) and the morphoeic or sclerosing (2–3%) BCC, typically presenting as an enlarging scar, and is associated with a high rate of positive margins following excision (Figure 1). Features associated with an increased risk of recurrence include location (periorbital, nose, peri-oral, naso-labial folds, pre-and post-auricular), histological subtype (morphoeic or infiltrative), ill-defined borders, size of the tumour (>2 cm), and immunosuppression.

Squamous cell carcinoma

Squamous cell carcinoma is the second most frequent skin cancer, occurring mainly in sun-exposed areas. It can arise de novo or because of progression from pre-cancerous lesions such as AK or Bowen's disease. It has a predilection for the head and neck area, mainly the ear, scalp and lip (Figure 2). Other areas include the dorsum of the hand, forearm and leg. SCCs arise from the basal keratinocytes and undergo uncontrolled growth as they migrate from the proliferative basal layer. They can present as nodular lesions or plaques, with differing degrees of keratinization, which can be in the form of a keratin horn or plug. SCCs also present as ulcers, or arise from chronic wounds, such as burn scars, benign ulcers and sinus tracts (Marjolin's ulcer). SCCs tend to be more common in men, and demonstrate a higher incidence with geographical variation, increasing significantly at lower altitudes. Chronic exposure to UV light plays a significant role in the development of SCCs, especially in lightly pigmented skin. Exposure to carcinogens, such as pesticides, arsenic, organic hydrocarbons and betel leaves can lead to SCCs. Immunosuppression represents a significant risk, especially in renal transplant patients and those with human papilloma virus infection. As with other tumours, SCCs can be graded as well,

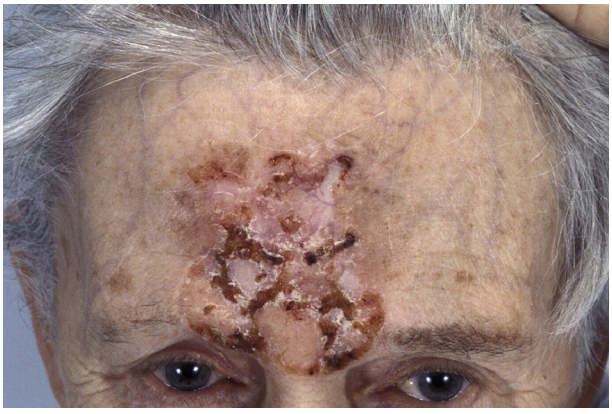

Figure 1 Morphoeic basal cell carcinoma of the forehead.

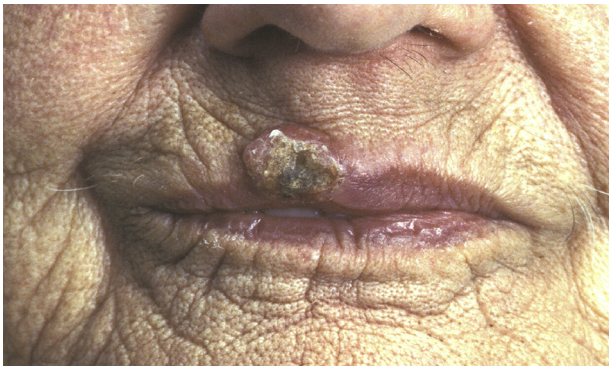

Figure 2 Squamous cell carcinoma of the upper lip.

moderately or poorly differentiated tumours, with the latter associated with a worse prognosis. Unlike BCCs, SCCs tend to be more aggressive and can metastasize not only to the regional nodal basin, but also to the lungs, liver, brain, skin and bone.

It is not clear whether keratoacanthomas represent another variant of SCCs that do not metastasize, or they are an entity on their own. A keratoacanthoma (KA) usually presents as a dome shaped lesion that has grown progressively and rapidly within a 4–6 week period, followed by a period of latency, with the lesion resolving within 6–10 weeks, leaving behind an atrophic scar. It can be difficult to differentiate between KAs and SCCs clinically and histologically, and it is advised that such lesion is best managed as an SCC with surgical excision and histological analysis.

The management of non-melanoma skin cancers

The management options for NMSCs can be categorized into surgical and non-surgical options. The aim is to remove the tumour, achieve a high cure rate, preserve as much local tissue as possible and produce an optimum cosmetic result. The treatment choice depends on the type and location of the tumour, the age and health status of the patient, available local services and experience, and the patient's choice.

The correct clinical diagnosis for NMSCs can be challenging, and a biopsy is usually required, with surgeons electing to perform an excision biopsy of the whole lesion to ascertain the diagnosis, rather than doing an incision biopsy. If the lesion is extensive or is in a cosmetically challenging area, an incisional or a punch biopsy may be more appropriate to confirm the diagnosis before formulating the appropriate management plan.

The most conventional way of managing NMSCs is surgical excision of the lesion. Other surgical options include Mohs micrographic surgery (MMS), curettage and electrodessication (E&C) and curettage. Non-surgical options may be more suitable in cases that have a potential for disfigurement and functional impairment, or because of the risks of surgery. The use of topical chemotherapeutic agents, biological-immune response modifiers, retinoids and photodynamic therapy are options that are currently available. Radiotherapy has also been used in specific cases, with varying degrees of success.

Mohs micrographic surgery (MMS)

MMS involves the serial excision of the tumour in horizontal sections, which are mapped, processed by frozen section and analysed microscopically. Both the deep and peripheral margins are analysed and re-excision is performed until the tumour is completely excised. This method allows for the entire tumour to be excised, which can be more accurate than standard tumour excision, where less than 1% of the margin is analysed, with maximal preservation of normal tissue. Contiguous tumour spread, as occurs in BCC and SCC cancers, is necessary for this technique to be effective. MMS is time consuming and is only performed in a few centres. Specific indications for MMS include primary tumours in locations associated with rates of recurrence, such as the midface and ears, tumours with ill-defined margins, tumours with aggressive growth patterns such as morphoeic or infiltrative BCCs, recurrent tumours and for the excision of tumours in cosmetically unique places. MMS allows immediate reconstruction once complete excision of the tumour has been achieved. In complicated cases, a multidisciplinary approach involving a plastic surgeon, a dermatologist, a histopathologist and an oncologist, is adopted. MMS is a very effective method of managing NMSCs, with a 5-year recurrence rate of 1% for BCC and 3% for SCC, compared to a 5.3% and 8% recurrence rates for standard excision.[2]

Excisional surgery

Excisional surgery is the most common modality of treatment for most NMSCs. It is useful for low-risk tumours and provides a cure rate that is acceptable and cost effective. For well-defined, small (<20 mm) BCCs, a surgical margin of 4 mm would achieve a clearance of 95%, while for large (>20 mm) and morphoeic BCCs, a wider margin of excision is advisable (5 mm margin: 82% clearance; 13–15 mm margin: >95% clearance).[3] In cases of incomplete excision, especially in critical midfacial areas or where the histology shows features of an aggressive tumour, it is advisable to re-treat these cases, either by re-excision or MMS.[3] For recurrent BCCs, peripheral excision with 5–10 mm have been suggested.[3] Surgical excision for cutaneous SCC remains the treatment of choice, allowing full histological analysis of the tumour. For clinically small (<20 mm) tumours, a 4-mm margin would allow complete excision in 95% of cases. In bigger tumours (>20 mm), poorly or moderately differentiated tumours, or tumours in complex areas such as the ear, lip, scalp, eyelids and nose, a wider margin of excision is advocated (6 mm or more) to achieve clearance.[4]

Radiation therapy

Radiotherapy is effective in the management of primary and recurrent BCCs, and can be used either as the primary treatment modality or as adjuvant therapy.[3] It is an excellent treatment choice for patients with high-risk disease, or in patients who are unwilling or unable to tolerate surgery. Radiotherapy can be delivered using superficial X-rays for superficial lesions, electron-beam therapy for deeper lesions and brachytherapy for lesions on convex or concave surfaces. Delivering the radiotherapy in fractionated doses results in a better cosmetic result as it allows for normal tissue to recover; tumour cells take longer to recover, thus fractionating the doses reduces the damage done to the surrounding normal tissue. Although radiotherapy is painless and suitable for debilitated patients, side effects include radionecrosis, alopecia, pruritus, depigmentation, skin atrophy and telangiectasia. The latter can also occur over time and radiotherapy is best reserved for patients over 50 years of age with uncomplicated tumours. Patients with ill-defined lesions, tumours on the hands, genitalia and lower legs, recurrent lesions on previously irradiated areas, and patients with syndromes such as basal cell naevus syndrome and xeroderma pigmentosum are unsuitable for radiotherapy. Studies have reported 5-year cure rates of 90–93% for small (<20 mm) primary BCCs and SCCs, similar to other treatment modalities.[5] Radiotherapy can also be used as adjuvant therapy in cases of aggressive SCC with perineural invasion or nodal metastasis.[5]

Cryotherapy

Liquid nitrogen cryotherapy (−196.5 °C) uses the destructive cellular effects of freezing to destroy the tumour cells of NMSCs.

Freezing at temperatures between minus 50–60 °C produces ice crystal formation intra-cellularly and extra-cellularly, leading to tissue damage. A double freeze/thaw cycle is generally advised for the treatment of facial BCCs, while superficial truncal BCCs may only require one cycle.[3] Small SCCs have been successfully treated with cryotherapy. However, histological diagnosis is normally required prior to treatment, and because of the variability in the use of liquid nitrogen, it is best that such treatment is used for selected cases only.[4]

Curettage and electrodessication

Curettage and electrodessication (C&E) is often used in the management of superficial NMSCs. The tumour is curetted down to normal looking tissue and the base is then electrodessicated to produce necrosis of the cells, and the process repeated as required. C&E is commonly used for the management of low-risk BCCs, and its use is contra-indicated in high-risk facial BCCs associated with a significant risk of tumour recurrence.[3] Various reports have reported excellent cure rates for the management of small, well-defined SCCs. However, the use of C&E in the management of SCCs should only be undertaken by experienced clinicians following careful selection of cases.[4]

Topical chemotherapy

The most common topical agent used is 5-fluorouracil (5-FU), which interferes with DNA synthesis, leading to tumour cell death. It is usually applied daily over a 4–6 weeks period, causing inflammation and blistering. The areas normally heal without scarring once the inflammation has settled down. It is frequently used on premalignant lesions such as actinic keratosis, and superficial BCCs and SCCs. Its inability to penetrate the dermis however limits its use to the superficial NMSCs.

Imiquimod: Imiquimod is an immune-response modulator that binds to cell surface receptors of Toll-like receptor TLR-7 and TLR-8, to promote both innate and cell-mediated immune responses, via the release of cytokines (e.g. IFNα, IFNγ, TNFα, IL-1, IL-6, IL-8, IL-10, IL-12) and activation of TH-1 cell mediated immunity. The latter is responsible for the antiviral and anti-tumour properties of imiquimod. Topical imiquimod appears to be effective in the treatment of small, primary superficial BCCs, with reported clearance rates of 82% noted following a 6-week treatment period.[6]

Interferon: Intralesional interferon (IFN) can be used in the management of patients with BCC, who are either not fit for surgery or in whom surgery would lead to disfigurement. IFN leads to apoptosis of BCC cells through the release of IL-2 and IL-10. Although cure rates of 50–80% have been reported, IFN may not be suitable for high-risk tumours.

Smoothened inhibitor (SI): Aberrations in the Hedgehog signalling pathway have been associated with an increased predisposition to BCCs.[7] They inhibit the normal function of patched homologue 1 (PTCH1), resulting in the lack of Smoothened inhibition. This leads to increased levels in GLI1 levels and subsequent tumorigenesis. Gain-of-function mutations of the Smoothened gene can also lead to increased incidence of BCCs. Vismodegib is the first and only U.S Food and Drug Administration (FDA) approved Smoothened inhibitor used in the management advanced and metastatic BCCs, which became commercially available in 2012. A phase II study and a subsequent study investigating the effects of Vismodegib have found response rates of 30% in patients with metastatic BCCs and 43% in patients with locally advanced BCCs.[8]

Photodynamic therapy (PDT)

PDT involves the application of a photosensitizing agent to the skin, which is preferentially absorbed by the tumour cells. Such agents include porphyrin 5-aminolevulinic acid (ALA) and the methyl ester of ALA (mALA), both of which are converted to protoporphyrin IX once absorbed. The latter is subsequently activated by a light source in the 450–750 nm wavelength range, producing oxygen radicals, which lead to apoptosis and damage to the tumour cells. PDT is a good treatment for the management of primary superficial BCCs and may provide a reasonable treatment option for low-risk nodular BCCs[9] and superficial SCCs.

Malignant melanoma

Primary malignant melanomas can arise either as new lesions (~60%) or from pre-existing melanocytic naevi. Their development appears to be multi-factorial, and is associated with various risk factors, including fair complexion, increased childhood sunburn, an increased number of dysplastic naevi, a positive family history and the presence of a changing mole. The exact sequence of events from normal melanocytes to melanoma remains unclear, although it is believed to involve several genetic mutations, which alter cell proliferation, differentiation and death, and thus increase its susceptibility to damage by UV light.

The four main subtypes of primary cutaneous melanomas are superficial spreading, nodular, lentigo maligna and acral lentiginous melanomas (Figure 3). Other rarer cutaneous melanomas include desmoplastic and amelanocytic melanomas. Primary melanomas can also be found in mucous membranes (e.g. gut, oral cavity, eye and nasal paranasal sinuses).

The ABCDE criteria have been described to increase the public's awareness of melanomas: Asymmetry, Border irregularity, Colour variegation, Diameter > 6 mm and Evolution. Nonetheless, a new mole that appeared after the onset of puberty with changing features, or a mole which has lost its symmetry, is itchy and bleeding, is growing, especially under the nail, should be dealt with a high degree of suspicion and the patient should be

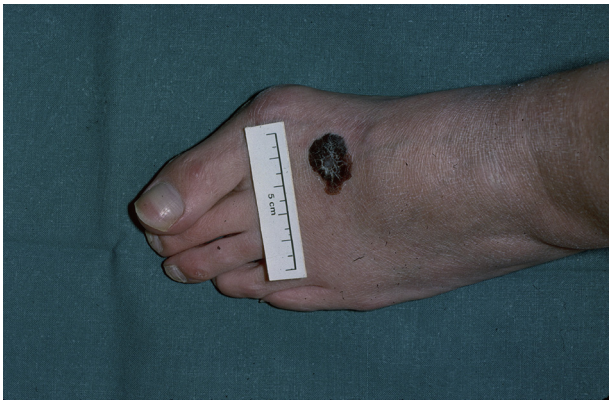

Figure 3 Nodular malignant melanoma on the dorsum of the foot.

referred to a specialist centre where the lesion can be assessed for melanoma.

A lesion suspected of being a melanoma should undergo an excision biopsy with a 2-mm margin, with the axis of excision oriented such that direct closure following a wider local excision may be subsequently possible. Once histological diagnosis of the excision biopsy is confirmed, subsequent definitive treatment, based on the Breslow thickness of the specimen, can be carried out. Lesions suspected of melanoma should not undergo shave biopsies because of inaccurate diagnosis due to sampling errors and difficulty in assessing the Breslow thickness. Also, incisional or punch biopsies of pigmented lesions is only acceptable in special cases, such as in obtaining a diagnosis for a lentigo maligna on the face or an acral melanoma. The Breslow thickness measures, in mm, the thickness of the tumour from the granular layer of the epidermis to the base of the tumour. The Clark level is also used to describe the thickness of the tumour, based on the degree of invasion of the tumour, relative to definitive anatomical layers of the skin. Because of the varying thickness of the skin, Breslow thickness is more accurate at describing the thickness of the tumour, with the Breslow thickness reflecting the patient's prognosis.

The lesion is subsequently graded and staged according to the TNM classification. Patients with stage I, II and IIIA disease do not require any investigations. Those with stage IIIB and IIIC disease should have a CT of the head, chest, abdomen, pelvis, while patients with stage IV disease should be imaged according to clinical requirements.

The only curative treatment for melanoma is surgery. Once the excision biopsy has been analysed for Breslow thickness and following discussion in a skin cancer multidisciplinary team meeting, a wider local excision of the excision biopsy scar is carried out, based on the Breslow thickness of the tumour (Table 1), to reduce the risk of local disease recurrence. The wound defect is subsequently closed directly, or reconstructed with a skin graft or local flap.

Elective lymph node dissection is of no value in the management of clinically negative node patients. The use of sentinel lymph node dissection can be considered by a specialist skin cancer multidisciplinary team (SSMDT) for patients with IB and upward disease. The final report of the MSLT 1 (Multicenter Selective Lymphadenectomy Trial) has shown a significant improvement in disease-free survival and melanoma-specific survival in patients with intermediate thickness melanomas.[9]

Margins for wider local excision of melanomas based on Breslow thickness

Breslow thickness of tumour	Margin for wider local excision
Lentigo maligna, in-situ superficial spreading MM	0.5 cm
Melanoma up to 1.0 mm	1 cm
Melanoma between 1.0 and 2.0 mm	1–2 cm
Melanoma between 2.0 and 4.0 mm	2–3 cm
Melanoma >4.0 mm	3 cm

Table 1

In cases of suspected lymph node disease, a fine needle aspiration (FNA) should be performed to confirm the diagnosis. If the first FNA is negative, it should be repeated or an image-guided core biopsy should be performed. Patients with positive nodal disease should undergo a radical lymph node dissection. Surgery remains the treatment of choice in cases of recurrent disease. However, in patients with multiple small cutaneous metastatic deposits, a CO_2 laser can be used to ablate these lesions. Patients with progressive recurrent metastatic disease, not responding to surgery or laser, or with deeper metastatic disease can be considered for regional chemotherapy with isolated limb infusion (ILI) and isolated limb perfusion (ILP).

Electrochemotherapy (ECT) is an established treatment modality for the management of cutaneous and subcutaneous metastases. Originally described in the treatment of metastatic head and neck cancer, it is now an established tool in the management of melanoma cutaneous and subcutaneous deposits. ECT relies on the physical properties of electroporation, where a short electrical pulse is applied to the desired area.[10] This in turn de-stabilizes the cell membrane and increases the permeability of the tumour cell membrane to the desired cytotoxic drugs, thus potentiating the cytotoxic effects of the drugs. ECT is a highly effective treatment in the local treatment of melanoma, with a favourable risk-to-benefit profile as a result of its simplicity, cost-effectiveness and limited toxicity.

Tumour genotyping and the advent of new drugs are changing the management algorithm of stage IV disease. Nowadays, most melanomas are genotyped for BRAF and c-KIT before selection of the appropriate therapy.[11] For most BRAF mutated melanomas, vemurafenib is the treatment of choice; dabrafenib and trametinib are novel therapies that have been approved in the management of patients with BRAF positive melanomas.[11] KIT inhibitors, such as imatinib, can be useful in KIT-mutated tumours, especially with those with mutations at exons 11 and 13. Ipilimumab, a CTLA-4 inhibitor, is a good alternative for tumours that progress despite vemurafenib or c-KIT inhibitors, or in patients with non-targetable or undetected mutations.[11]

If there is any histological or clinical doubt of the surgical margin following excision of a recurrence, adjuvant radiotherapy may be considered by a SSMDT. Patients with melanoma are followed up according to the stage of their disease and should be managed within a multidisciplinary team approach.

Imaging modalities

As the incidence of skin cancers continues to increase, plastic surgeons will encounter more patients with suspicious lesions and aggressive tumours, who require the appropriate imaging prior to any intervention.

Dermatoscopy is a simple tool used to assess the dermal and epidermal structures of lesions that are not commonly seen by the naked eye. It consists of a magnifying device with an integrated light, used with a liquid interface or polarized light.[12] An improved accuracy has been demonstrated in detecting melanomas with a dermatoscope, when compared to the naked eye examinations.[13] In a study undertaken by Townley et al., plastic surgeons were better at diagnosing skin cancers at the end of a dermotoscopy course.[12] While there is a learning curve with the

use of the dermatoscope, the reduced morbidity from reduced lesion excisions and the economic benefits cannot be understated.

Computed tomography (CT) remains the mainstay for the imaging of advanced cutaneous malignancies. CT scans are used for assessing bony cortical involvement, the extent of soft tissue tumours and nodal metastases. CT scan evaluation of the nodal basins of the chest, abdomen and pelvis are offered to patients with stage IIC melanoma who did not have a sentinel lymph node biopsy, and to patients with stages III and IV disease.[14] CT scanning is often the initial imaging modality for assessing extracutaneous involvement as it is fast, inexpensive and provides a good spatial resolution.[15] It is also thought to be better than magnetic resonance imaging (MRI) at assessing nodal status as focal deposits within nodes can be seen with greater clarity.

Positron emitted tomography- CT (PET-CT) is typically used in patients with head and neck malignancies and in the search of distant malignancies. The PET component relies on the increased glucose metabolism pathway in malignant cells with increased metabolic activity. Radiolabelled 18-flurodeoxyglucose (18-FDG) is injected intravenously and taken up by the cells. Malignant cells with an increased metabolic activity have an enhanced uptake, resulting in an increased emission of positrons, the intensity of which is detected and displayed. The combination of PET with CT has produced a greater sensitivity in detecting distant and nodal tumours, and it is commonly used in assessing visceral metastases, occult head and neck lymphadenopathy and tumour response to therapy.[15] The use of PET-CT is limited in the assessment of slow grow growing tumours, such as BCCs, but is commonly used in the staging and surveillance of SCCs and Merkel cell carcinomas, which are more metabolically active.[15]

Magnetic resonance imaging (MRI) is another imaging modality that is commonly used in the management of skin cancer. MRI relies on the application of a magnetic field to align of spins of hydrogen protons in soft tissue. As the spins of the protons realign in the magnetic field, a specific signal is emitted which is displayed as a diagnostic image.[15] MRI scans have a superior soft tissue contrast to CT scans and are more sensitive at detecting central nervous system and perineural invasion that are typically seen with aggressive neurotropic cutaneous malignancies.[15]

Ultrasonography (US) is another imaging modality that is commonly used in the management of skin cancers. It is typically used in the assessment of suspicious lymph nodes. Depending on the morphology of the node, it may be possible to distinguish between a benign and a malignant node. Benign lymph nodes tend to be ovoid or flat in the long axis, with an echogenic hilum, while malignant nodes are often round, with central necrosis and loss of an echogenic hilum.[15] In suspicious cases, a fine needle aspiration (FNA) can be performed to confirm the diagnosis. US-guided FNA has been shown to be more sensitive and specific than conventional FNA.[15]

Conclusion

In conclusion, malignant skin cancers are common with an ever-increasing annual incidence. They should be managed in a multidisciplinary setting, consisting of a plastic and reconstructive surgeon, a dermatologist, an oncologist and a histopathologist with an expertise in dermato-histopathology. ◆

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