Overview of skin cancer / pre-cancerous lesions

Dr Farhana Ravat
Consultant Dermatologist
Hillingdon, Mount Vernon and Amersham Hospitals
Chair of skin cancer LMDT/SSMDT for Northern sector of WLCN

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GP Masterclass
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Objectives

• Ensure everyone is aware of the current guidance in relation to skin cancer services
• Be aware of what information pathologists require
• Recognise different types of skin tumours and be aware of the different therapeutic modalities
• Know how to treat pre-cancerous lesions
• Know where to get more information
• Will not cover rare skin cancers
Key Documents

- **NICE**: Improving outcomes for people with skin tumours including melanoma (IOG); published Feb 2006
- **NCAT**: Manual for Cancer Services 2008: Skin Measures
- **DOH**: Implementing care closer to home: The accreditation of GPwSIs (generic) and Guidance and competencies for the provision of services using GPwSIs (speciality specific)
IOG recommendations

• Cancer networks should establish 2 levels of MDTs (LMDT/SSMDT)
• All healthcare professionals who knowingly treat patients with skin cancer should be members of one of these teams
  – ie. individual GPs should not knowingly treat skin cancer unless part of MDT and accreditation of community service / facilities / individual (March 2009) has taken place
WLCN

• THH dermatology consultants have been members of the skin cancer TWG of the WLCN since inception
• Includes clinicians from NW LH, CM dx, WM dx, CWH, St Mary’s, Ealing/Hammersmith, CXH
• Includes multiple specialities: dermatology, plastics, pathology, maxillofacial surgery, oncology, primary care, cancer managers and patient representation etc.
• Skin cancer has been peer reviewed in July 2009
IOG recommendations

- People with pre-cancerous lesions should be either treated entirely by their GP or referred for diagnosis, treatment and follow-up to Drs working in the community who are members of the LMDT/SSMDT.
- If there is any doubt about the diagnosis, people with pre-cancerous lesions should be referred directly to their local skin cancer specialist, normally a dermatologist who is a member of the LMDT/SSMDT.
IOG recommendations

• Patients with low risk BCCs should be diagnosed, treated and followed up by Drs working in the community as part of the LMDT/SSMDT (usually a dermatology GPwSI) or a local hospital skin cancer specialist, normally a dermatologist, who is a member of the LMDT/SSMDT and to whom they have been directly referred

• Where there is doubt about the lesion being low or high grade, the patient should be referred directly to the LMDT/SSMDT
IOG recommendations

• All patients with a suspicious pigmented skin lesion that may be a high risk BCC, a SCC or a melanoma or where the diagnosis is uncertain should be referred to a Dr trained in the specialist diagnosis of skin malignancy, normally a dermatologist, who is a member of either an LMDT / SSMDT
IOG recommendations

• Cancer networks should ensure through the skin cancer network site–specific group that LMDT/SSMDTs work to network agreed protocols for
  – Referral
  – Review of patient care by the MDT
  – Mx and audit of services for precancerous lesions and skin cancer services
IOG recommendations

• Cancer networks should ensure provision of ongoing education for all healthcare professionals about this very common group of tumours
IOG recommendations

• The follow up of patients after treatment should be jointly agreed between patient and Dr

• After appropriate instruction, patients with low risk disease will normally practice self examination but follow up may be offered in a community setting where appropriate

• Patients with a high risk of recurrence of their skin cancer should normally be followed up in hospital but should still be instructed in self examination and provided with written and photographic information
IOG recommendations

- All patients / carers should have access to high quality information, in an appropriate style and format, about their condition and its Mx and about access to relevant support services.
- Skin cancer network site specific groups should follow protocols covering the Mx of high risk groups or those with special needs eg. transplant patients, genetic predisposition to skin cancer, rare tumours (including cutaneous lymphoma) and children and young people.
- Commissioners of cancer services should create an infrastructure for well conducted research to take place in order to contribute to the skin cancer evidence base in epidemiology, treatment and Mx.
**Specimen type:** 4mm punch biopsy / excision biopsy / shave biopsy / curettage from RIGHT temple (intention must be clear ie diagnostic or attempted removal)

**Clinical:** Never write ‘lesion’ as this isn’t a diagnosis and is unhelpful

9 month history of pearly nodule at same site as BCC previously treated by radiotherapy 20 yrs ago ?Recurrent nodular BCC. Excised with 5mm margin. Marker suture at 12 o’clock
Referral

• If MM / SCC suspected, ideally refer via WLCN proforma for a 2 week appt (or fax letter directly to THH)
• If it looks like BCC, but rapid growth (weeks), use WLCN proforma for 2 week appt
• If unsure, state colour(s), provide relevant history, use ABCDE criteria in letter and secondary care triage will prioritise
Referral

• If BCC suspected, refer via letter. Usually seen within 6 weeks
• If you are fairly confident that the ‘mole’ is benign, but want a second opinion, refer via letter for a routine appt (usually 6 wks)
• If you have accidently operated on a skin cancer, you need to alert the LMDT
Malignant melanoma

- Increasing incidence, rate doubling every 10-20 years in white populations
- CRUK: ~9000 cases/yr; 1800 deaths/yr
- 2% MM patients have an affected relative
- Celtic phenotype
- XS sun exposure (blistering in childhood); controllable risk factor
ABCDE

• Asymmetry
• Border irregularity
• Colour variation
• Diameter > 6mm
• Evolving / enlarging
ABCDE

• But will miss
  – Early melanoma
  – Atypical lesions
  – Pick up excess benign lesions
Glasgow 7 point scale

• Major
  – Changing size
  – Irregular shape
  – Irregular colour

• Minor
  – Diameter > 7mm
  – Inflammation
  – Ooze
  – Changed sensation
# Breslow / Survival

<table>
<thead>
<tr>
<th>Breslow depth</th>
<th>5 year survival (approx)</th>
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<tbody>
<tr>
<td>&lt;1 mm</td>
<td>95-100 %</td>
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<tr>
<td>1-2 mm</td>
<td>80-96 %</td>
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<tr>
<td>2.1 – 4 mm</td>
<td>60-75 %</td>
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<tr>
<td>&gt;4 mm</td>
<td>50 %</td>
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Management

- Surgical excision; margins dependent on BD
- Imiquimod for selected cases (experimental)
- Stage 3 dis: surgical / intransit dis: DXT; surgery; ILP; imiquimod
- Stage 4: dismal prognosis; symptomatic Tx
- FU visits: looking for new MM/NMSC; recurrence; mets; self-examination; mole watching; sun protection
Squamous cell carcinoma

- Second most common skin tumour
- UV related; esp fair skinned
- Inherited predisposition eg XP
- Chronic wounds / thermal burns
- Immunosuppressed / transplants (HPV)
- Arsenic / smoking / Bowen’s
Management

• Surgical excision with 4-5mm margin
• C+C
• Cryotherapy
• Radiotherapy

• 95% of recurrences/mets detected within 5 years
Prognosis

• Depends on site; size; rate of growth; aetiology; histological differentiation; host immunity

• Increasing metastatic potential: sun exposed / non ear/lip > lip > ear > non sun exposed site > arising from leg ulcer, burn, Bowen’s, chronic inflammation
Basal Cell Carcinoma

- Commonest type of cancer in humans.
- 80% on H+N, 15% shoulders/trunk, 5% other
- > 62,000 cases of NMSC diagnosed each year in the UK (4/5 are BCCs).
- Can occur after DXT for eg T capitis in childhood
- Most are sporadic with rare hereditary cases
  - Basal cell naevus syndrome (Gorlin-Goltz)
  - Xeroderma pigmentosum.
Basal Cell Carcinoma

- Cell of origin not defined
- Genetics
  - p53 mutations seen in 56% of sporadic cases with UV-signature observed in 65%.
  - PTCH
    - germline mutation responsible for Gorlin-Goltz syndrome.
    - 30-40% of sporadic BCC have PTCH alterations but only 41% have characteristic UV-signature mutations
Differential diagnosis

- Actinic keratosis
- Bowen’s disease
- SCC (dorsal aspect of hands)
- Viral wart
- Stasis / discoid eczema (lower legs)
- Seborrhoeic keratosis
Risk stratification

• Tumour size
  – >2cm
• Tumour site ie. H+N
  – nose / paranasal / periocular / ears / scalp / temple / lips
• Recurrent tumour
• Clinically morphoeic BCC (flat, thick, hard lesion)
• Histological subtype
  – morphoeic/infiltrative / micronodular / basisquamous
• Histological features
  – perineural invasion / invasion below dermis
• Immunosuppression / genetic disorders
Histological subtypes

• Circumscribed cohesive growth pattern – eg. nodular / cystic
Histological subtypes

- Diffuse growth pattern – morphoeic / micronodular / infiltrative / superficial
Treatment of BCC

Surgical
• Excision with predetermined margins
• Curettage and cautery
• Mohs’ micrographic surgery
• Cryotherapy

Medical
• Imiquimod
• Photodynamic therapy (PDT)
• Radiotherapy
• 5-Fluorouracil (patients may buy over the internet!!)
Curettage and Cautery (C+C)

- Relies on lack of adhesiveness of tumour cells compared to normal skin hence can scrape away the tumour cells and define a margin.
- Useful for selected low risk lesions.
- Not suitable for morphoeic, recurrent or high risk site tumours.
- 5 year cure rate of up to 92.5% in experienced hands.
Cryotherapy

• Useful for selected lesions with non aggressive histology – esp. large superficial BCC at a low risk site.
• May require local anaesthetic.
• Not suitable for recurrent tumours.
• Can get full thickness burn as 2 x 30s freeze/thaw cycle and permanent hypopigmentation.
• 5 year cure rate of 91% in experienced hands.
• [AK may have been treated with cryo but turns out to be BCC (usu recurs in centre)]
Excision with predetermined margins

- Primary BCC <2cm well defined tumour
  - 3 mm margin clears tumour in 85%
  - 4-5 mm margin clears tumour in 95%.
- Primary morphoeic BCC
  - 3 mm margin clears tumour in 66%
  - 5 mm margin clears tumour in 82%
  - 13-15 mm margin clears tumour in >95%.
General points relating to surgery

- Majority of BCC recurrences will occur within 2 years, but can be many years later.
- Recurrent BCCs are more likely to recur again so Mohs’ or wide excision is preferred Mx.
- Disadvantages of *closure by flap* includes increased risk of bleeding, infection, extensive BCC recurrence *under* flap.
- Recurrence after C+C appears early and is easily visible.
Incompletely excised (IE) BCC

- Re-excision will reveal residual BCC in ~50%
- IE at deep margin is esp difficult to cure with re-excision
- Recurrent BCC is more difficult to cure
- Re-excision if IE at high risk site; +ve deep margin; aggressive histological subtype
- Needs MDT as multiple options incl Mohs’ / RT / WLE / observation
Mohs’ micrographic surgery

- 5 year cure rate of 99%.
- Useful for:
  - Site: eyes / ears / lips / nose / nasolabial fold
  - Histological subtype: morphoeic / micronodular / infiltrative
  - Recurrent
  - Size > 2cm
  - Perineural spread
Radiotherapy

• Expensive, time-consuming.
• Useful for frail patients unable/unwilling to undergo surgery.
• Also used post-op for patients with perineural spread or recurrent tumours.
• 91.3% 5 year cure rate, 7.5% 4 year recurrence rate and risk of developing secondary BCC.
Topical 5’-Fluorouracil

- Useful for low-risk, extra-facial BCC.
- Does not eradicate infiltrative BCC and may mask deeper component by treating only the superficial part.
- > 20% recurrence at 10 years.
- More effective if combined with curettage / cryotherapy.
- Locally irritating.
Photodynamic therapy (PDT)

- Selective accumulation of topical methyl-ALA in malignant tumour cells.
- Exposure to non-ionising radiation 3 hrs later using Aktilite lamp results in cell death.
- Depth of penetration of the photosensitizer is a limiting factor, thus only suitable for superficial BCCs (87% cured vs 53% nodular BCC).
Imiquimod 5% cream

- Immune response modifier.
- Upregulates production of IFN$\alpha$ and TNF$\alpha$ in the dermis 1-2 hours after application.
- Used successfully to treat superficial BCC (5 x wk for 6 wks, 85% CRR), solar keratoses and Bowen’s disease.
- Side effects – itching, erythema, discharge, and tenderness (dose dependent), post inflammatory pigmentary disturbance.
- Biopsy before and after.
General points

- If you find a skin cancer, look for others
- Remove crust to see what is underneath
- Do not cryotherapy a lesion that you cannot diagnose or which recurs after cryotherapy
- If your patient served in the Forces, they are entitled to compensation as part of their war pension so tell them about it
Helpful sites for info

• **www.bad.org.uk** for PILS and provides guidelines for management of
  – Secondary care: BCC / SCC / Melanoma / CTCL / PDT
  – **Primary care**: Actinic keratoses / Bowen’s disease
  – GP’s can obtain skin cancer information packs

• **www.dermnetnz.org** is also a good source for PILS and has photos
Actinic keratoses

- Keratotic lesions on chronically sun exposed skin in middle aged / elderly
- Risk factors: UVB (p53 mutations), sunbeds, immunosuppression, arsenic
- Focal areas of abnormal keratinocyte proliferation and differentiation
- Epithelial dysplasia may be restricted to basal layer or could be full thickness
- Low risk of progression to SCC (<1/1000 per yr, but if >7 AK’s, have 10% risk of SCC over 10 yr period)
Extensive solar keratoses
Actinic keratoses

- Variants: hypertrophic, lichenoid, bowenoid, acantholytic, pigmented
- 15-25% of AK’s will regress spontaneously over 1yr
- Excess incidence of developing future BCC/SCC
- Diagnosis is clinical, but occasionally skin biopsy as differentials include SBCC, Bowens, invasive SCC and amelanotic MM
- Marker of sun damage so need to increase sun avoidance measures
Treatment

• No treatment if mild AKs
• Topical:
  – Emollient + sunblock bd
  – 2% salicylic oint to remove keratin pre Efudix
  – 5% Efudix cream bd for 6 wks (various regimes / s.e.)
  – Solaraze gel od for 16 wks (fewer s.e.)
  – Aldara cream 3 x wk for 16 wks (expensive / s.e.)
Treatment

- Cryotherapy (effective, esp thicker lesions, nb. scar)
- Photodynamic therapy (effective esp for superficial confluent AKs; good cosmetic results; useful for lower legs, expensive)
- Curettage (x2-3 cycles) of thicker lesions
- Excision if cannot rule out SCC
Bowen’s disease

- Intraepidermal (in situ) SCC
- Genital = erythroplasia of Queyrat in men and some VIN in women, Bowenoid papulosis both sexes, perianal nb HPV
- Gradually enlarging well demarcated erythematous plaque with irregular border, crusting and scaling
- 70-85% cases in women, 60-85% on lower legs, 10-20% multiple lesions
Bowen’s

- Risk factors: irradiation (solar/DXT/PUVA); carcinogens eg arsenic; immunosuppression; HPV; chronic injury/dermatosis
- 30-50% of patients with BD may have previous or subsequent NMSC
- 3-5% risk of progression to invasive SCC
- ?10% for genital BD
Bowen’s disease differentials

- Eczema
- Psoriasis
- LP
- AK
- Wart
- Seb wart
- Superficial BCC
- Amelanotic melanoma
- Paget’s disease
- Merkel cell tumour
Treatment

- Depends on location, size, whether single/multiple, good/poor healing site
- Observation esp thin, elderly, lower leg
- Efudix 5% cream od-bd for up to 2/12 (s.e.)
- Aldara cream for larger lesions, HPV (s.e.)
- PDT (more effective than cryo/Efudix esp for lower legs but expensive)
Bowen’s treatment

- Cryotherapy
- Curettage (effective and heal faster than cryo, benefit of histo to exclude SCC)
- Wide excision when genital/perianal
- DXT effective but poor healing
Learning points

• Be aware of the current guidance in relation to skin cancer – esp in relation to BCC
• Skin cancer can be difficult to recognise
• Referral pathways specified
• Recognise different types of skin cancers and be aware of the different therapeutic modalities and when dermatologists use them
• Primary care: treat pre-cancerous lesions, look for skin cancer and encourage sun protection