**Understanding and Managing Skin Lesions in Primary Care Transcript.**

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0:01 **Chair:** Welcome to this webinar on all things dermatological. I've got great pleasure in introducing Dr Jonathan Bertalot, who's a GP with a special interest in dermatology. So I'm sure it's going to be an excellent session.

0:25 **Speaker:** Thank you very much everyone. So my name is Jonathan Bertalot. I am a GP Partner in Llangefni and a Speciality Doctor in Dermatology, working four sessions/two days a week exclusively in skin lesions and skin cancer. I'm going to talk about skin lesions, benign, malignant and pre-malignant and how to recognise them and a bit of a framework as to how to manage them.

0:48 **Speaker:** So this is an important primary care problem. We all know that it constitutes a big part of your workload and there's very little training in dermatology and even less in skin lesions. So we end up with many unnecessary referrals being made. And as you may be aware, here in Bangor Hospital, we have started a new system for trying to triage these referrals and to try and give advice and guidance where needed and avoid unnecessary trips to the hospital for the patients.

1:21 **Speaker:** So you may have seen posters like this, which you have in GPs reception rooms or in dermatology departments. And it's a sort of top down approach where you try and memorise what these lesions look like. So you see a big ugly melanoma, or a stereotypical BCC and you try and remember, and you think: ‘Right, that's what they look like. I'm going to look for that next time’.

1:42 **Speaker:** But it's worth trying to remind yourself of the anatomy of the skin and the pathology that causes these lesions. So more of a bottom up approach which should increase your diagnostic accuracy when it comes to trying to work out what these things are. And the importance of this approach is that it means that you shouldn't hopefully miss the red flag features.

2:06 **Speaker:** When it comes to deciding about skin lesions - are they nasty or nice? It's very simple - because it's a one-step algorithm. There are no complicated flowcharts to memorise. All you need to do is decide:

* Is this benign? So do I leave and reassure the patients or
* Is this suspicious? Do I treat the lesion and the patient? And by treatment, there's obviously a spectrum of things you can do.

So this has been called the best protocol, benign leave and reassure or suspicious and treat. So very straightforward. So you would leave the benign seb-k in this photo, and you would obviously treat the superficial spreading malignant melanoma.

2:50 **Speaker:** Assessing lesions. Do not forget the cornerstone of medicine of course, which is history, examination and special investigations. The history is all important and it's the one thing that tends to be forgotten with dermatology and lesions, because, of course, it's a very, very visual speciality. There's a lot of spot diagnosis that takes place. Examination is easier if you stretch the skin, and you have good lighting. And when it comes to special investigations, the one we mean, of course, is biopsy.

3:19 **Speaker:** Now, just a quick reminder of the anatomy of the skin, because this is all important when it comes to working out where the lesions arise from. So you have the epidermis at the top. There are many layers of the epidermis. The predominant cell type is the keratinocyte, and the keratinocytes are made along basal membrane along the dermo epidermal junction, and they migrate upwards through the surface until they form the stratum corneum on the outer surface where they swell from. The dermis is below the epidermis and projects upwards into the epidermis with a series of finger like projections which form the rete ridges. And then within the dermis you have the pile of sebaceous units, which includes the hair follicle, sweat gland and the arrector pili muscles. You have intradermal vessels and subdermal or hypodermal vessels.

4:14 **Speaker:** So when it comes to making this diagnostic decision (is it benign or is it suspicious?) it helps to be able to recognise the benign lesions. So we're just going to do a very quick reminder of what these benign lesions are, and the salient features that will allow you to make a spot diagnosis.

Let's dive straight in.

**Dermatofibroma.**

What causes dermatofibroma? Well, everyone knows insect bites cause dermatofibroma. How do you know that? Because somebody told us. Well, it's not quite as simple as this. And there's long been a debate about whether they are inflammatory - so post insect bite or post items, for example, or whether they are in fact, neoplastic. And there have been recent genetic studies that suggest they are neoplastic. It could, of course, be a combination of the two. So a malignant process or neoplastic process triggered by trauma. But they're hard, firm papules, light brown to dark brown. They are common in the third and fourth decades of life, on the arms and legs. And when you give them a squeeze from the sides, you've got this pinch test. So there's a dimpling of the lesion; the centre of a lesion appears depressed. It's a very, very useful sign. Of course, they can cause diagnostic difficulties. And when you get a hyper pigmented dermatofibroma that stands out as an ugly duckling, definitely refer. That’s a fair enough referral. They undergo a period of rapid growth and then stabilisation, and by the time you see them, they have generally stabilised, so they are no longer changing. If it is changing, then that's a red flag. And any new evolving, growing, changing lesion on an adult, needs excision. So that's one of the take messages. Dermatofibroma is normally simple, but it can be tricky if they are in the rapid growth phase. They arise, the clue is in the name, from the fibroblasts of the dermis. So it's a dermal tumour.

6:24 **Speaker:** OK, moving on to the next one, Haemangioma.

**Haemangioma.**

These are either dilated capillaries or very small arterial venous malformations. Extremely common. Many, many types. You see them all over the body and in all different age groups. This is a typical Campbell de Morgan spot. They are often multiple. Often on the limbs. And in terms of diagnostic difficulty - well, if they're traumatised, of course, they can resemble melanomas. So here’s a traumatised haemangioma that looks like a melanoma and that could be referred on a 2 week wait query melanoma. These arise generally in the subdermal plexus. So this cartoon isn't actually accurate because the lower plexus vessel is within the dermis here. Really, it should be within the fat, just below the dermis. And they are either capillary dilatations or venous malformations.

7:24 **Speaker:** Right. So that was the second of the two benign lesions. The third spot diagnosis you need to make is one of the commonest that you will not go a day in general practice without seeing, which is bbenign keratinocytic lesions.

**Benign keratinocytic lesions.**

These are solar lentigo and the Seborrhoeic keratosis and these lie on a spectrum. Solar lentigos are typically well demarcated macules, flat brown with a notched or moth-eaten border in sun-exposed areas. Hands, face, trunk. Very, very common. They can look old; they can have odd shapes and stand out. They can be larger than the other legions like this one. They can have very strange shapes like this. But they are completely benign as long as they remain flat and brown. A particular diagnostic difficulty becomes when they are on the face because flat pigmented lesions is a tricky area, and the differential is either really solar lentigo which is benign or lentigo maligna which is malignant melanoma in situ. And it's a tricky differential to make and it can be difficult. Another differential is pigmented actinic keratosis. But Naevus is not in the differential test because Naevi on the face are never flat. Naevi on the face are dermal Naevi which are raised and fleshy. So flat brown macules on the face – you think solar lentigo, lentigo maligna or maybe pigmented actinic keratosis. On the same spectrum – benign keratinocytic, Seborrhoeic keratosis. And of course we've all seen patients like this with hundreds of these typical stuck on lesions; they are greasy, you can pick them off, they've got well-defined borders. And the difficulty of course, comes in when you have a patient like this or show the previous slide, how do you pick out the melanoma amongst all these? And it's difficult. There's no easy way around this. You need to spend a lot of time examining the patient, a long time with a with a dermatoscope for help.

9:42 **Speaker:** Now, Seborrhoeic keratosis are keratinocytic. They arise from keratinocytes, but they can be pigmented. Now the pigment in the keratinocytes is melanin. So it comes from melanocytes, but they are keratinocytic but melanotic, because its melanin causing the pigment. And when they're deeply pigmented and when they're traumatised, they also can cause diagnostic difficulties and we get an awful lot of referrals on the two-week wait, which turn out to be Seb-Ks, but query melanoma. So the keratinocytes that form these solar lentigo and Seborrhoeic keratoses, they are the basal layer of keratinocytes. So in this diagram, they are set just above the dermal-epidermal junction, hence the synonym basal cell papilloma for Seborrhoeic keratosis.

10:36 **Speaker:** Moving on to another benign lesion, in the group of five which you should be able to spot diagnose, sebaceous hyperplasia. Sebaceous hyperplasia

**Sebaceous hyperplasia**

This is very common in men with greasy skin. They frequently get misdiagnosed as BCCs because they look very, very similar. They are raised, they have vessels in the centre, and they are frequent/multiple. So that makes diagnosis easier. Like on this chap on the slide for example. That makes diagnosis very straightforward. If they're solitary like they are here, then they can easily be confused with BCCs and dermoscopy, as we shall see later, helps you differentiate.

11:23 **Speaker:** OK, this is hypoplasia of the sebaceous gland. Why does it occur? Well, it’s hormonal factors, genetic factors, sun exposure may play a role we're not really sure.

11:30 **Speaker:** Finally, onto the next big group of benign lesions, the Naevus. And this is the one that does cause diagnostic confusion with melanomas. This is the biggest differential that gives you a headache. Is it a Naevus or is it melanoma?

**Naevus**

That's a huge topic in itself. So just to summarise it, there's a big range of types that are mainly classified as Junctional versus Compound versus Dermal, which refers to the depth of invasion through the skin. It can be classified as congenital versus acquired. And the hallmark of these histologically is a proliferation of benign nested melanocytes. So as opposed to malignant melanoma, which are an atypical proliferation of malignant melanocytes, these are benign melanocytes that clump together.

Now, this is a zoomed in on the dermal epidermal junction and the epidermis and the melanocytes exist along the dermal epidermal junction, usually at a ratio of about between 1:10 to 1:40 keratinocytes. So that's the usual state of affairs. But in Naevi, there are many more of them. So this cartoon here demonstrates what happens when the junctional melanocytic Naevi start to penetrate deeper. So they are junctional when the melanocytes remain on the dermal epidermal junction. As the melanocytes deepen and involve the dermis, they also form a nodule on the outer surface; they become more fleshy and raised. And then as they go deeper still, you will get predominantly dermal melanocytes, which caused quite a raised, fleshy nodule on the outer surface. So that’s the progression of Naevi as they move from junctional to compound to dermal. And again, you frequently see patients like this with many, many Naevi in the diagnostic difficulty they give you is that it's difficult to pick out the melanoma amongst these Naevi. As I mentioned before, on the face Naevi are raised. So a flat brow mark on the face is not a Naevus. A raised fleshy dome on the face may well be a Naevus. Terminal hairs are a useful clue to the benign nature of these lesions. They don't always mean it has to be benign, but it's a very useful clue and a very reassuring if you see them.

These are **Giant** **Congenital Naevi**. The only reason you need to know about these really is not a) they are rare and b) if they are over 20 centimetres in size, they massively increase the risk of melanoma for that patient in the future. It's one of the one of two things that really do increase the risk. One is giant congenital Naevi, over 20 centimetres in size, like this patient has and the other is a high Naevus count over 100 Naevi will increase your risk of melanoma. But what's interesting to note is that in both those cases, the risk is to the patient, not the Naevi. So most melanomas in both those situations will arise in normal skin, not in an existing Naevus, and the reasons for that are not fully clear.

14:49 **Speaker**: A different type of benign Naevi is **Blue Naevi**. Now, these are melanocytes, which are in an atypical anatomical location. These are actually melanocytes within the dermis rather than the epidermal junction. How do they get to the dermis? Well, it's not really known, but it's speculated that it might be to do with migration of the melanocytes during embryological development. So they come from the neural crest in the exoderm and migrate to the epidermis during early life. But some melanocytes get arrested in this transition in the dermis. And it's thought that this might give rise to dermal naevi. So these are very homogenous lesions, and they can mimic malignant melanoma and it's difficult to tell them apart. And the only real reliable way is in their history. You will get a history of stability with these. They've been there for a long time, unchanged. If you see what looks like a blue Naevus and it's been there for two weeks, you excise it because any new, growing, nodular lesion in an adult, needs excision. Again, that's that one take-home message, if you remember nothing else, remember that.

16:00 **Speaker**: So to summarise, so far, we've done the benign lesions: Dermatofibroma; Haemangioma; Benign keratinocytic – solar lentigo / seborrheic keratosis; Sebaceous gland hyperplasia; Naevi - junctional / compound / dermal / giant / blue.

Is it benign? Can I name it as one of those five? A history of stability is key and always palpate the lesions.

16:17 **Speaker**: We're going to move on to malignant and pre-malignant lesions now.

What was formerly called non melanoma skin cancer is now called Keratinocyte Cancer (KC), and the reason for this is that non melanoma skin cancer is a bit of a strange definition; it’s everything that is not melanoma, which obviously creates a lot of strange and wonderful things. Far better to categorise things from their cell of origin and both the commonest skin malignancies in the world – BCC and SCC arise from keratinocytes and hence are called Keratinocyte Cancers (KCs). The pathology of their development is different. So, you can see on this slide on the top line, there is a progression from Actinic Keratosis to Bowen’s disease to SCC. Basal cell cancer is different.

So first we're going to look at the progression of **Actinic keratosis, Bowen’s disease, SCC.**

17:16 **Speaker**: And the point here is that they are all due to dysplastic keratinocytes. Dysplastic keratinocytes have developed and arisen because of ultraviolet radiation causing genetic abnormalities and dysplasia, and it occurs in the superficial levels first and then penetrates deeper through the skin. So when it is very superficial, that is an AK; that is in Actinic Keratosis. The risk factors are increasing age, sun exposure, lighter skin type and immunosuppression. It can be a field change where you rub your hand over this chap's forehead and it’s all rough like sandpaper and dry. Or it can be lesions where it coalesces into little palpable lesions. And of course, both can coexist at the same time.

18:11 **Speaker**: Here's another example of field change and lesions. Lesions can become hyperkeratotic when you get the typical Keratin horn and these all represent dysplastic keratinocytes, which are really quite superficial on the skin.

18:23 **Speaker**: Are considered pre-malignant? Well, the risk of progression to an SCC is very low. You will see lots of figures quoted, you know, I've seen it quoted as 3% of patients with AK will develop an SCC. I've seen it quoted as if there are 10 or more lesions, the risk of developing an SCC within five years is 10 to 15%. But the point is it can develop into Bowen’s disease and then an SCC. However, what's interesting is that you would think from that observation, treating AKs should reduce the risk of SCCs, because that's logical, isn't it? We know they progress, so surely if you prevent, if you treat the early stage, you prevent the SCCs. However, as yet, there is zero published evidence that treating AKs result in less SCCs. That doesn't mean it doesn't work, just that there is zero evidence so far.

19:27 **Speaker**: How do you treat them? You advise them about sun damage, keep out the sun. You advise them to apply moisturiser, a sunblock, wear a hat and explain the features that would lead them to think that there might be a progression in terms of depth of those keratinocytes that would require treatment for Bowen’s or for SCC.

19:49 **Speaker**: There's a very good summary which I’ll come to in second on the PCDS (Primary Care Dermatology Society) website. So you can treat the field, or you can treat the lesion. Field treatment involves a choice of diclofenac or 5FU, which is Efudix or Aldara or Actikeral. Lesions can be treated with cryotherapy, with liquid nitrogen or curetage or Actikeral or sometimes Efudix. And all these depend on your skill set.

20:19 **Speaker**: There is a very good table here from the Primary Care Dermatology Society website. Now I have no affiliation with them whatsoever, but I find the website very, very clear, very, very helpful, and you can download excellent treatment guides like this, which allow you to discuss with the patient what's best for them and to choose the most appropriate treatments.

20:40 **Speaker**: Bowen’s disease occurs when those dysplastic keratinocytes thicken through the epidermis and reach the dermal epidermal membrane. So it's full thickness dysplasia. The classic site is the legs. It's also called Squamous Cell Carcinoma in-situ or Intraepidermal Carcinoma, which demonstrates that the only difference between this and invasive SSC is the depth of invasion. The keratinocytes look the same. Now, just one point I'll mention here is that if you if you do biopsy AKs, or Bowen’s or SSCs in your practice, you need the architecture of the lesion in order for the histopathologist to give you the correct diagnosis. In other words, if you take a few samples of the cell, be it through a curettage or a small biopsy, like a 3mm biopsy, then the pathologist is basically working with cytology, and they cannot appreciate the depth of the invasion and the architecture of that lesion. So, if you biopsy them, either take a good punch biopsy that goes through the full thickness of the skin, 4 millimetres minimum, better if 5 millimetres, or you can use a curette and take a good big scoop of it that goes down to include the papillary dermis so the pathologist can see if there was a malignant keratinocyte invading through the dermal epidermal junction.

22:12 **Speaker**: So these are classic Bowen’s disease. A patch on the legs is the commonest; scaly, rough, generally not itchy or painful, usually asymptomatic.

22:25 **Speaker**: These are Bowen’s disease that could have been SCCs. I biopsied them. They came back as Bowen’s disease.

22:34 **Speaker**: The treatment is sun protection. Cryotherapy can help but be careful on the pre-tibial area because it can cause ulceration. Efudix can help. Curettage can help. All these things can be done in primary care depending on your skill set.

22:48 **Speaker**: Moving on to the dangerous cancers.

**Squamous Cell Carcinoma.** So the cells now have penetrated through basement membrane and invaded into the dermis. And this is where they become dangerous. The risk factors, of course, are the same for AK and Bowen’s disease. UV light exposure, Fitzpatrick skin type 1 and 2, and immunosuppression. So renal transplant patients are particularly at risk of these. Chronic inflammation in the legs is also a risk.

23:23 **Speaker**: And the risk depends on the site, the size and the degree of differentiation. They can be well differentiated, moderately differentiated or poorly differentiated, which is the worst one. So here the malignant keratinocytes have gone deeper and they're now in the dermis are now at risk of metastasising and they now can be very, very dangerous. So that's full thickness. Full thickness, meaning through the skin invasion of a dysplastic keratinocyte.

23:45 **Speaker**: High risk areas are the areas of the head and neck mainly, but they are common in all the sun exposed areas: back of the hand, scalp, ear.

23:55 **Speaker**: So this ticks all the boxes, obviously, for being a high risk cancer. It’s on the head and neck, it's over 2cms, poorly differentiated, which is the worst. And even if this were excised with clear margins, that patients will be followed up for several years in dermatology to check the lymph nodes and do a full skin check afterwards.

24:17 **Speaker**: How do you tell whether something has progressed from Bowen’s disease to SCC? This is a common conundrum and it's something that I'm trying to perpetually hone my own skills on because, you know, it's frequent to biopsy things and it comes back as a Bowenoid Actinic Keratosis where you have suspected an SCC. But there are two there are two tips that do help differentiate. If you have a keratin horn, like on the upper photograph here, that generally indicates if it is going to be an SCC or differentiated keratinising SCC. It's more likely to be an SCC as opposed to Bowen’s disease if it has a nodular fleshy base, like this lesion does here. So that's one clue. Now, the other clue is pain. SCCs become painful and actually it's been demonstrated in published research that the depth of invasion of that SCC correlates to the pain as marked on a pain score by the patient preoperatively. So in other words, if the patient says the lesion is really painful, then not only is it likely to be an SCC, but it also is likely to have invaded more deeply than a less painful lesion.

Why are they painful? Well they invade nerves. And it's something that all pathologists will report on the histopathology report, is whether or not there is perineural invasion, which is deemed to be significant if the nerve that is being invaded is greater than 0.2 millimetres. So perineural invasion also correlates with pain. But they are the two ways of swaying your decision-making as to whether it's an SCC.

26:04 **Speaker**: How do you treat them? You refer them as USC, urgent suspected cancer, because of the potential to metastasise. So if you're not clear about what it could be, biopsy it but send the biopsy to USC and that will give you an answer. Often, we do biopsy things, before excising them. The treatments, if they're confirmed or highly suspect SCCs is to excise them, and complete excision will cure the patient in most cases.

26:37 **Speaker**: A variant of SCC is **Keratoacanthoma**. There's been a controversy for years and years about whether this is a separate entity or is it part type of an SCC. Again, more recently, genetic studies have shown that it is actually a subtype; there is very strong shared genetics with SCCs. So this is a subtype of SCC, but it behaves differently as we know. It grows very, very rapidly and then it regresses. They do tend to look quite classical, like this one here in the picture with a crater-like volcano area in the middle of a load of keratin on the outside. By the time you refer them, they may have doubled in size already or they may be on the way to regressing maybe to a point of invisibility sometimes. However, it's a subtype of SCC. It's very difficult to tell apart histopathological. And so the treatment is the same. You refer them as query SSCs; excised on a USC basis.

27:34 **Speaker**: The second type of keratinocyte cancer is a **Basal Cell Carcinoma**, which is the commonest skin cancer. Over 80% of all cases are BCCs. Loads of diagnoses per year in the UK, probably a third lifetime risk of all men getting one of these; slightly less for women. They rarely metastasise, but they do cause problems because they arise in sun-damaged areas on the head and neck where there are many, many structures they can invade into. So it's a very slow rate of growth; 2-3mm per year. And I think for this reason, people often do neglect them because they are such slow growing lesions. It is not uncommon for people to come to us with things that have been slowly eroding away for 5 or 10 years and purely by local effects that can cause significant morbidity.

28:35 **Speaker**: So as opposed to the AK/Bowen’s/SCC spectrum, these begin on the basement membrane. So it's malignant basal keratinocytes this time and immediately they become dysplastic, they invade the basement membrane, but they tend to cling to the underside of the basement membrane. So they’re immediately in the dermis, but they tend not to spread particularly deeply. And as we mentioned, they grow very, very slowly. Now, the pattern of spread determines the subtype, and it can generally be of two types.

29:05 **Speaker**: Most types are low risk, and most will either be superficial or nodular. In the superficial variety, those keratinocytes which have penetrated the basement membrane will spread laterally, and they will cling to the under surface in a thin sort of sheet and spreads laterally. Or they can become nodular where it forms a discrete nodule on the under surface of the basement membrane. What's interesting is that the diagnosis of a nodular BCC histologically might differ from what you call a nodular BCC clinically. So the histopathologist will see a nodule on his slide and will call it a nodular BCC. Now usually, that nodule will cause a doming of the skin on the surface, and you will be able to stretch the skin and see the rolled edge and call it a nodular BCC, but not always. So sometimes you get the histopathology report that comes back as a nodular BCC and you think to yourself, what's going on here? This was a flat lesion. So it can happen. Superficial and nodular BCCs are low risk and the significance of that is that low risk tumours in low risk areas are suitable for treatments in primary care if you have adequate skills. So it's worth learning about them and recognising them.

30: 33 **Speaker**: The high risk BCCs involve infiltrative subtypes, where you get strands of malignant keratinocytes that extend out in all directions into the dermis. Morphoeic or morpheaform, which are scar like white areas. Basal squamous that are on the way to becoming SCCs and Micronodular. Nodular BCCs are low risk; micronodular are high risk. And the pathologist will measure the size of the nodules and if a large proportion of those nodules are under a certain size it is classified as micronodular, which is high risk.

31: 03 **Speaker**: These are the stereotypical superficial BCC. It's a flat red patch, slowly expanding; there maybe scale. They may be multiple and if you confuse these with a patch of discord eczema or a patch of psoriasis, don't worry, we've all done it. It's a perfect situation in primary care where if you're not sure what it is, a punch biopsy is very, very useful. So I’d encourage everyone to skill themselves up and be comfortable doing punch biopsies because you will then know do you treat as a superficial BCC, or do you treat as psoriasis or whatever it may be. Very useful.

31: 42 **Speaker**: This is the classic nodular BCC. The rolled edge. You can stretch the skin to accentuate that rolled edge. Very commonly there is the classic history of scabbing, bleeding, crusting, drying and that occurs on repeat.

32: 02 **Speaker** Morphoeic BCCs or infiltrative, can be very, very difficult to spot. It is a very subtle, white scar-like area under the eye.

32:12 **Speaker** And this is a close up of the classic, nodular BCC when you get the rolled edge that is sometimes called a whipcord edge and you get a vessel, Telangiectasia. Now, incidentally, those vessels are very sharply focussed, which is why they are so easy to spot. Dermatoscopically, they appear as sharply focussed branched vessels. And that is because the stroma of a low risk BCC tends to be clear and jellylike. So there is good penetration of the light, and the vessels appear as sharply focussed vessels. The higher risk BCCs (infiltrative and Morphoeic BCCs) tend to have a very dense fibrous stroma, so you cannot see the vessels as clearly. So you tend to get the classic vessels in classic nodular low risk BCCs.

33:03 **Speaker** On the face, this is a perfectly acceptable mistake to make. Is this an intradermal naevus or is it a BCC? And it can be difficult. So don't worry if you make that mistake. Again, we've all done it.

33:19 **Speaker** A couple of other examples of superficial BCC and infiltrative BCC. So quite variable in their appearance would be a high index of suspicion.

33:27 **Speaker** The treatment. Nodular and superficial BCCs are suitable for treatment in primary care. So you can all do this if they are in low risk locations, these low risk BCCs can be treated by yourselves. Superficial BCCs you can curette them, just scrape them off; excise them, cut around them. You can use in Imiquimod, which is Aldara or in certain elderly patients with multiple BCCs who understands what he has and whether to treat or not treat, you can agree to do nothing - perfectly reasonable as well. Nodular BCCs - the gold standard is to excise them with 4 mm margins, aiming for complete lateral and deep clearance of that lesion which will be reported on in the pathology report. If you have an infiltrative BCC, for example, that you've proven on a punch biopsy you will refer that patient because they are tricky to manage. They need wide margins to make sure you've got the whole lesion out.

34:23 **Speaker** Moving on to the one that we do not want to miss. **Melanomas.** Melanomas arise from malignant melanocytes on the basal membrane. So as I mentioned, they are normally in a ratio of 1:10 to 1:40 melanocytes to keratinocytes. With melanomas you get clumping of those lesions, and this slide here gives you some stats about them. Stats on slides can sometimes be difficult to remember, and if anything like you don't always remember these things. But the point is that they're getting commoner, and you need a high index of suspicion for these because you will see one or more than one as a GP. I guess most GPs might agree they pick up a melanoma every three to four years, I would imagine. We do see a lot in dermatology.

35:20 **Speaker** You only need one melanoma to undergo abnormal division to form a clone of malignant melanocytes, and then that is a malignant melanoma. They can then spread, and they can spread in a pagetoid fashion, rising up through the epidermis, or they can spread in a lentiginous fashion, expanding laterally along the dermal epidermal membrane. Provided it's limited to the epidermis by the dermal epidermal membrane, it will be a malignant melanoma in situ, which on the face is called lentigo maligna. Then as soon as it penetrates the basement membrane, that is invasive melanoma, and that's when you measure the thickness.

36:09 **Speaker** So some people would call lentigo maligna, pre-malignant. I consider it malignant, but obviously with a very, very low risk of metastasis. So the risk factors, large Naevi counts and large congenital Naevi, Fitzpatrick skin type 1 or 2, previous melanoma, family history, increasing age and increasing UV sun exposure.

These are the standard screening questions that I ask when I have somebody who is sat in front of me and has been referred with a suspicious dark brown mole:

Have you or a family member had any type of skin cancer?

Have you ever lived abroad?

Can you remember burning as a child to the point of blistering or peeling?

Do you tan easily or go red first?

to try and determine their skin type.

37:05 **Speaker** These are the types of melanomas. You can guess at them clinically, but differentiation is really by histology.

37:12 **Speaker** And let's take a look at some melanomas. So here’s a superficial spreading melanoma. And this would fail the ABCE criteria that you all have heard about before: A

for asymmetry, B for irregular borders, C for different colours, D for different dimension, which is more or less obstinate now as we shall see. They are generally flat about palpation until they go into the rapid vertical growth phase.

37:37 **Speaker** Nodular melanomas undergo a rapid vertical growth phase at the earlier stage. And these are often a raised lesion.

37:45 **Speaker** This is the one on the face that we've seen before, which is Lentigo maligna. So these are confined to the epidermis by the dermal epidermal junction. It can be very difficult to tell them apart from solar lentigo. They can be very slow growing. A patient can have these for decades before he even thinks of seeing the GP. Usually a nondescript, flat, brown patch, and sometimes the only clue would be different shades of colouration.

38: 17 **Speaker** So as I touched on earlier, is lentigo maligna, pre-malignant? Well, I think it is because those melanocytes, they don't know whether they're in the epidermis or whether they're in the dermis. They've got the same genetics; they are from the same clone of malignant melanoma that is undergoing uncontrolled divisional growth. So in my opinion, this is malignant. It just happens to be confined to the epidermis and therefore has a very low rate of metastasis. I know you see on documents still that they're called pre-malignant, but that's not my opinion. Malignancy does not necessarily mean the potential to metastasise. It can be defined merely as locally destructive.

39:05 **Speaker:** Acral melanomas behave differently. They are not on some exposed areas. They are the commoner type of melanoma on darker skin types, and they're frequently confused Naevi and they can be frequently misdiagnosed because of the history of trauma.

39:25 **Speaker:** Subungual melanomas are very uncommon. So having biopsied a great many of these subungual melanomas exactly like this that you see in photograph, I have yet to diagnose one. So I still stick to this one rule, which is another take home take home rule that I would encourage you to remember - any new longitudinal subungual pigmentation in an adult, mandates a USC referral. Don't be led astray by trauma because trauma of the feet is so common that people often say they have a history of trauma, and they will associate that with the discolouration in their own mind. And it's so common. In fact, there have been postulated aetiological links with trauma and suggestions that trauma can trigger melanoma. That has never been proven one way or the other, but it is common. In fact, we all know that Bob Marley died of subungual melanomas. Well, for years he actually put down his melanoma down to trauma from playing barefoot football. So don't be led astray by a history of trauma.

40:30 **Speaker:** Tips to diagnose. So the differentiation is frequently between melanoma and junctional naevi, which are flat pigmented lesions, also melanocytic. The history is crucial. So you always ask about a history of rapid change: changing colour, changing shape and changing modularity. If things that were flat become raised – that is a red flag.

40:56 **Speaker:** The ugly duckling sign is useful. I'm sure you know about this. Is it the one your eyes are first drawn to when you see that patient as in this case here?

41:07 **Speaker:** These are the ABCDE criteria, and they are helpful, as an aide memoire to deciding what to refer. So when it comes to asymmetry, you're looking at the chaotic nature of malignant tissue that grows in an uncontrolled manner and the depth of the invasion in different parts of the lesion lead to different colorations. The border irregularity is very important. So chaos of border symmetry is also a very useful clue. Colours multiple and dimensions, which was originally described as over six millimetres when the ABCD criteria was first introduced, dimension is not relevant. Those malignant melanocytes, they are malignant melanocytes whether they are 4 cells, or they are 400,000 cells. They don't know if they're 2mm big or 6mm big. We know because we can measure it. But since the development of dermoscopy, it is common to diagnose melanomas of 2-3mm in diameter. So this is no longer relevant. Think of EFG as elevated, firm and growing, which is a red flag that any new lesion.

42:28 **Speaker:** If you suspect it, refer it USC. Do not biopsy as in primary care. The reason for this is that a partial biopsy has a limited chance of diagnosing the lesion. The lesions are very heterogenous in their histopathological distribution of malignant cells versus regressed cells and so on. Since a high chance of missing the diagnosis with a partial biopsy, refer them USC and secondary care takes over the management from there.

Usually it's a two stage procedure with one diagnostic excision or biopsy, and then if confirmed, they will have a wide local excision with further treatment if needed. All melanoma's except in situ will be followed up for full skin check and lymph node exam. So you need a high index of suspicion for these.

43:25 **Speaker:** These are Amelanotic melanomas. They are difficult to diagnose, but they come under the EFG rule: elevated, firm and growing. You will refer these to secondary care. **So the one take home message** – any new nodular, growing lesion on an adult is a USC referral.

43:30 **Speaker:** So this is a summary of malignant lesions.

Melanomas:

* Nodular and superficial spreading are most common.
* Be careful and younger people
* Assess with the risk factors, the history and do use the ABCDEFG criteria as a useful aid memoir.
* And if you suspect them, you refer them to USC.

Keratinocyte cancers:

* BCC is the most common.
* Try and classify them into high risk or low risk. If they're low risk, you can treat them in primary care. If you don't yet have the skills, the surgical skills, then upskill yourself. There are many courses to do that. And it's a very, very satisfying adjunct to your skills as a primary care physician.
* If you suspect as SCC, you referred USC. And remember how to try and pick out the SCCs from the other sun damaged cutaneous conditions of AK and Bowen’s. Pain and modularity are the two cardinal features of SCC.

44:45 **Speaker:** OK, now I'm going to mention just very briefly dermoscopy. I'm aware that I don't want to go on for too long, so I've tried to go quickly. It will be recorded, so you can go over it again.

I just want to mention dermoscopy now. Many of you, I'm sure you'll be familiar with Dermoscopy or Dermatoscopy, whichever way you want to call it. But for those of you who aren't, I just want to explain and explain its utility and usefulness, because this is a technique that's been around for 10/15 years, maybe even 20 years in common usage that is, and it has transformed the examination of skin conditions - skin lesions primarily, although that's increasingly being used for inflammatory skin conditions as well. And it's incredibly useful and it is incredibly good fun and very, very satisfying so if you haven't got into it, try and get into it. It really does increase your enjoyment of dermatology.

So when you look at the epidermis, you cannot see very deeply through the epidermis at all. And the epidermis is about 90 microns thick, which is 0.09mm. Now if you shine normal light at the skin, at the stratum corneum it will be reflected in a scattered fashion and it will shine off and cause glare. Now, there are two ways of getting around this. You can either use a fluid such as an ultrasound gel, which prevents the scatter of light, or you can use polarised light beams, which means the light beams run in parallel rather than running at various angles each other. This increases the depth of what you can see to 150 microns, so 0.15mm, which will take you just through the epidermis and into the capillary superficial dermis. And this opens up a whole world of structures and colours and shapes that you can then interpret by various methods.

46:50 **Speaker:** The evidence that this helps you is now overwhelming. There are just a few examples here of studies that demonstrate how teaching dermoscopy/using dermoscopy will not only increase your diagnostic accuracy, but it will reduce your unnecessary referral rate and it will reduce unnecessary excision rates. So obviously all these things are worth achieving and it can be done with very short, focussed teaching sessions by anybody. You don't have to be a dermatologist. You don't have to be a GP, a practise nurse, nurse, a health care assistant. Anyone can learn dermoscopy. It is not difficult.

47:30 **Speaker:** So here's a few examples.

I'm not going to teach you dermoscopy now, but I just want to show you a few occasions of where it is helpful.

Now, you may remember this lefthand photograph was the chap I showed at the beginning of the tour with a benign lesion, a dermatofibroma. But this was one that stood out like an ugly duckling because it's dark, it's pigmented, it's a solitary lesion. It could be a USC referral. Is this a melanoma? Well, put the dermatoscope on it. Look on the right, you see a central scar-like area. Around that you

see a rim of pigment that fades into the periphery in all directions with even distribution of that pigment. This is stereotypical for a dermatofibroma and this patient does not need an excision.

48:22 **Speaker:** Here's another example. Look at the guy on the left. He was referred as a USC Query malignant melanoma. He's got a new lesion. It's a solitary lesion. It's an obvious ugly duckling. You put the dermatoscope on and it takes you half a second and you see a characteristic bunch of grapes appearance, dark red or purple with white fibrous septa, splitting the modulated blood vessels. This is a Band 4 haemangioma. He doesn't need an excision and discharge him from the clinic.

49:02 **Speaker:** Here's another one. Do you remember this chap? He was also a USC melanoma referral. Completely fair enough. It is a very ugly looking lesion, isn’t it? Put the dermatoscope on and once again, you see that bunch of grapes appearance. The darker areas are simply traumatised, congealed blood on the surface. This is a haemangioma. So again, you can reassure the patient.

49:23 **Speaker:** This was the chap who had the strange looking lesion that was bigger than all the others. There's no obviously worrying features, but it is an ugly duckling. It stands out. Now have a look at the dermatoscopic image. It has what's called a motheaten, notched border. So it's irregular with lots of bites taking it to the centre. The colouration is light brown and medium brown, I would guess, which is reassuring. There's no dark or black there. The pattern, although it doesn't come out well, is very fine, curved lines. It's called a fingerprint pattern. And this is a solar lentigo.

50:12 **Speaker:** You can see the pattern better in this photograph here where you can see the very fine fingerprinting. So this is another typical solar lentigo. So you can reassure these patients. And these are patients that were referred to secondary care because they had odd looking lesions.

50:29 **Speaker:** This is probably the commonest time that it will help. Seborrhoeic Keratosis are so common, and they can be so darkly pigmented, frequently multiple, but frequently they're not. Now, look at this guy on the left of the photograph on left, a single, very dark, standout, ugly duckling lesion. Look at the picture on the right. There is a uniformly abrupt border. There are milia-like cysts, which mean those little white dots, which is basically keratin, which is under the surface, and it is not oxidised. You sometimes see yellow clods, which are keratin where it's broken through to the surface and it becomes oxidised and changes colour. You see these micro ulcerations which are sometimes called comedo-like openings. So this can only be a seborrheic keratosis. It is so satisfying when you've got a patient like this referred USC query melanoma, you stick on the dermatoscope and you say, no, it's absolutely fine, don't worry about it. And a very short amount of dermoscopy training could get you to that level in primary care. So if you aren’t familiar with this yet, then I'll show you some resources.

51:52 **Speaker:** I've had a few questions - can I do some dermoscopy teaching? I would love to do some dermoscopy teaching. Obviously, I wasn't able to include it into this, but if there is a call for it, I would love to do some in this a similar format.

52:16 **Speaker:** Here's another example. This chap with a solitary lesion on his left cheek, it was referred query BCC. You put the dermatoscope on and you see an aggregation of yellowish globules in the centre. You see these vessels are coming to the globules from the side, but do not cross the midline. So this is a sebaceous hyperplasia and that also is a very easy one to diagnose when you know what you're looking for with the dermatoscope.

52:41 **Speaker:** So these are two excellent books. There are very, very many good books on Dermatoscopy. These are two that I have. I’ve read them both cover to cover. And I would recommend them. The PCDS that I mentioned earlier also run study days, which are superb at Cardiff University. Also, there's a 12 week online course or at least they did do this. I did it about five/six years ago. So they are two very good resources and there's an International Dermoscopy Society Facebook page. Believe me, if you haven't got into it, then it's such a satisfying thing because the steepness of your curve of new knowledge is very steep indeed. And it's immediately satisfying. And you got immediate feedback, so that is another take home message - get into dermoscopy.

53:33 **Speaker:** So just to summarise the talk, because we're coming to the end now. I'm going to take a breath and slow down. I know I've been rattling along at a tremendous rate to try and fit it all.

So when it comes to assessing skin lesions, there's only one decision you need to make. Is it benign? Can I leave it alone? Or is it suspicious? Do I need to do something? Doing something might mean a referral or biopsy or excision, depending on your skill set.

The common benign lesions are spot diagnoses, so think about those five I mentioned and familiarise yourself with the cardinal features of each one of those.

Suspicious lesions are keratinocyte cancers and melanomas. Keratinocyte cancers are either on the keratinising spectrum - AK, Bowen’s, SCC or they are BCCs, and you need to try and think, is it high risk or low risk? And then the very most dangerous ones, the melanocytic lesions, malignant melanomas, assess them with ABCE criteria, but be aware of the of the limitations of that method.

But remember, the take home message above all else, is any new, elevated, changing or growing lesion refer it USC.

If you wanted to improve your skills, I would strongly advise learning some dermoscopy. And in order to manage those lesions that you can do as a primary care physician, low risk BCCs as well as biopsying of all lesions really, learn some surgical skills. So I hope that's been very helpful to you. I hope to be useful to you.

If there are any questions, then I will try and answer some.

55:36 **Speaker:** Any tips on skin photography?

Yes. Here in north-west Wales, we triage our referrals, and we ask for photographs with every single lesion. What I advise patients - we want photographs in good light and in focus. Use the main camera on the back of the phone, not the selfie camera, because the quality of the main camera is better. We want two pictures, one showing the lesion close up and one from a distance and the distance photograph just has to show it in context, so you don't have to see the whole person, just that body part/the face. It's useful to have a measuring tape and a patient's identifier as well on that distance photograph. So they are my tips. You don't need to be a great photographer, but you just need a few simple points you can pass on to the patients.

56:35 **Speaker:** SCC direct referrals next. Well, there's no there's no hard and fast rule about this. In dermatology, we excise a lot of these things. So it's absolutely fair enough, if you're not sure, to refer to dermatology first of all. The ones that we are wary of is high risk. So in high risk areas above the clavicle and high risk in terms of size above two centimetres. And often if I see that on a referral I won't see the patient, I will just direct it to either plastics or actually Maxfacs in our area. The maxillofacial team take on the SCC referrals. So if you think that this is a high risk lesion, it's above the clavicle, it's over 1.5cms in size then by all means refer directly to Maxfacs, but otherwise smaller SCCs from the neck down, that is our bread and butter in dermatology and we're more than happy to take those referrals. We're more than happy to give advice if these things are sent in.

57:41 **Speaker:** What age do you leave a congenital strawberry naevus to get better before a referral?

There's no there's no there's no hard and fast rule about this. Most strawberry naevi will resolve in mid to late childhood. It depends on whether it's bothering the parents and whether it's bothering the child. In most cases it’s not actually or rather if it is bothering one or two of them, then it's the parents rather than the child. Treatment would tend to be (because these are children) in a tertiary centre with paediatric dermatology, tertiary specialists who have access to laser treatments for those lesions if it ends up at that point. So usually beta blockers are tried to shrink down these lesions and then if not, if they're causing cosmetic problems, then laser treatments are very, very good option. So if the parents or childhood need require treatment, then we would tend to refer them into Alder Hey, which happens to be our closest tertiary referral centre for paediatric dermatology.

58:53 **Speaker:** So there was a question: can you use a dermatoscope through coloured skin? Yes, absolutely. It doesn't affect the salient features, the diagnostic features that you're looking for in each particular lesion. It can sometimes make it trickier, but those cardinal features must still be there if you look for them.

59:14 **Speaker:** BCC excision margin for a low risk BCC?

So a nodular BCC below the clavicle, you excise them with a 4mm macroscopic margin. So what that means is you will mark the lesion and you will measure four millimetres around the sides, and you will excise that amount of normal skin. When the histology report comes back, you will have a smaller margin on the histology report for a couple of reasons. Firstly, because skin will shrink. In other words, whenever you excise a specimen, it always contracts and appears smaller than it was in situ. And secondly, the formula that you put it in will further shrink the lesion, but also because macroscopically you cannot really see the margin and usually it's extended a little bit beyond where you can see. So you take a 4mm macroscopic clinical margin and that is very likely to give you an over 1mm histological margin, which is adequately treated. So if it's over 1mm histological on the pathology report, you consider that to be fully treated and it shouldn't need any follow up.

1:00:30 **Speaker:** Any recommendations for first affordable dermatoscope?

I can't really recommend any because there are so many on the market. The common makes that are very good – there’s Heine; a make called Oxiclar, which are very good; and there's a make called Dermlite which is very good. I've actually got a Dermlite DL4 is the model. But they all work. So it doesn't really matter. So if you are not sure and you do not want to spend too much money, pick up a cheaper one. There are plenty of very, very cheap dermatoscopes available. When I say cheap, I'm talking about £2-300. Believe me, you will use it more than your stethoscope, your ophthalmoscope, your otoscope combined. I'm sure of it. And in my humble opinion, I think the stethoscope is an outdated and rarely useful instrument, whereas the dermatoscope is an invaluable instrument that changes and informs your management every time you use it.

1:01:55 **Speaker:** Last question from David. What is the best regime for solar keratosis?

So if a patient has solar keratosis, I use I would tell them to use the same regime as for Bowen’s disease, which is one application per night for three to four weeks. You warn the patients that it will create inflammation and soreness. When that occurs, they can stop the regime at that point because it will have worked. And you can reassure them that if it gets pain from sore, that shows that the treatment is working. They can use a mild topical steroid to help settle down inflammation and then just leave it for three months. And if it's done the trick when you review them at three months it should be healed, should be a lot better. But often with solar keratosis, I start with diclofenac solareze because it's much less irritant.

1:01:55 **Speaker:** OK, thank you, David. Yes.

1:01:56 **Chair:** Well, thank you so, so much. Well, that was like fitting a duvet sized worth of information into a pillowcase. You've done extremely well. And thanks everybody for listening and for the questions. If there's enough call for it, I'm sure Jonathan will be very happy to do a session on dermoscopy, so we'll see what we get in the feedback. But thanks everybody for listening.