Benign skin lesions – Part 1
Some common skin lesions in general practice

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This month sees the introduction of a three-part article on benign skin 'lumps and bumps' that may be encountered in general practice.

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This three-part article will discuss a variety of benign skin lesions, some very common and others seen only occasionally, covering aetiology, important clinical issues and management. It is intended to be a broad, but not comprehensive, guide to the various 'lumps and bumps' that may be encountered in general practice. Part 1 of this article covers a variety of benign skin lesions. Subsequent parts, to be published in future issues of *Medicine Today*, will cover melanocytic lesions (Part 2) and vascular, dermal and adnexal tumours (Part 3).

SEBORRHOEIC KERATOSES

Seborrhoeic keratoses (SbKs) are benign skin tumours of keratinocyte origin. An Australian epidemiological study found them in 30% of people under 30 years of age and in 100% of people over 50 years.[1] In the UK, about 75% of people over 70 years have them.[2] The keratinocytes above the basal layer proliferate and keratinise abnormally, often forming small, intraepidermal, keratin-filled cysts (pseudohorn cysts). An SbK with a lot of these cysts plus a deeply clefted surface will likely become quite hyperkeratotic. Some SbKs have more melanocytes than normal skin.

The cause of SbKs is unclear. They may be related to sun exposure, occurring more often in more exposed sites, but they are not uncommon in sites that are unlikely to ever see the sun. Various somatic mutations in oncogenes and mitochondrial DNA have been identified – but these genetic changes are different from those seen in cancers.[3,4] Wart virus may play a role – the presence of its DNA in SbKs has been described in two reports[5,6] but was absent in another.[7] SbKs sometimes evolve from a lentigo (see below).

Appearance

SbKs are usually easy to identify but their appearance is highly variable (Figures 1 to 6). They are classically described as slightly greasy warty growths with a 'stuck-on' appearance – like mud thrown on a wall. The plaques vary from being slightly velvety, almost macular, to quite thick and hyperkeratotic. Others are verrucous but not so hyperkeratotic and resemble warts. They may be smoother, dome-shaped lesions resembling compound melanocytic naevi. Colour ranges from red to very dark brown and is often variable. SbKs are not related to sebaceous glands but the surface keratin retains sebum, giving some lesions a greasy appearance. The thick surface scale may peel off.

SbKs can occur on any site except the palms, soles, glans penis and mucosae. They range in size from small papules to large plaques. When located in skinfolds they grow parallel to the fold line. Their number varies enormously, from a few to hundreds. Dermoscopy can be very helpful in identifying SbKs, particularly if

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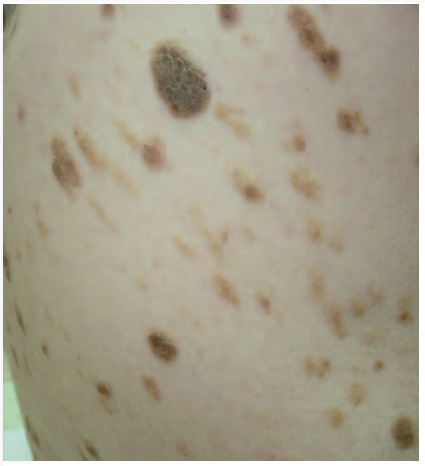


Figure 1. Typical SbKs on the flank of an elderly woman. Note the somewhat linear arrangement, a pattern that is seen in some patients.

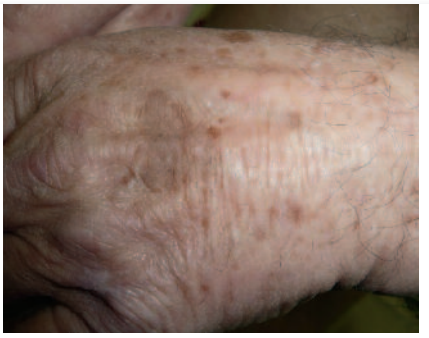


Figure 2. Flat SbK on the hand of an elderly woman, which is located among solar lentigos (and may have arisen from one). Note the velvety surface.

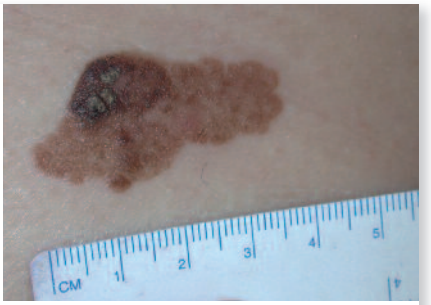


Figure 3. Three-tone, bigger SbK on the lower abdomen. Clinical features suggested an SbK, which was confirmed on dermoscopy, despite this lesion fitting the ABCDE rule for melanoma.

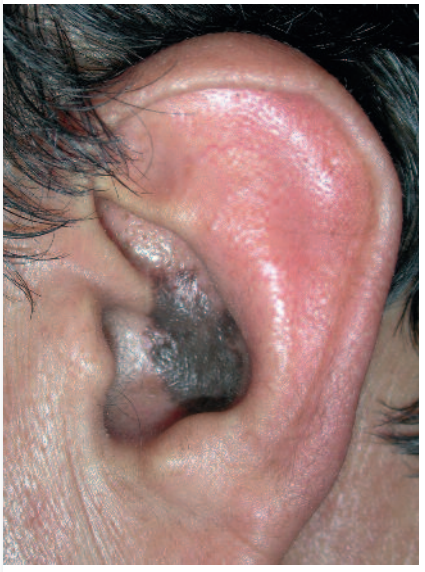
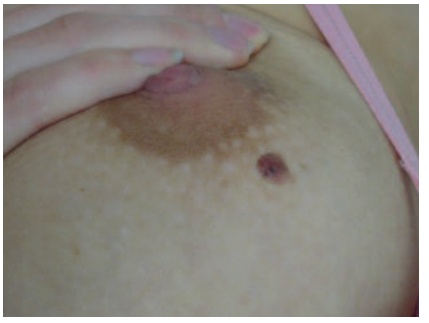
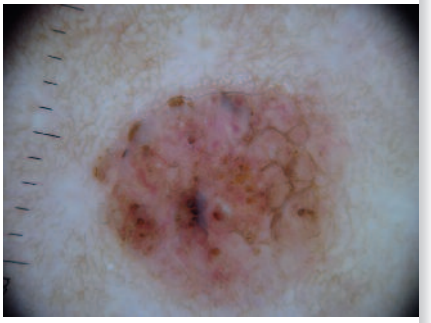


Figure 4. SbK on the ear, which was confirmed by biopsy. Dermoscopy was not possible on this site.





Figures 5a and b. Red SbK with brown flecks located on the breast.
a (above left). The lesion was located in a sun protected site and resembled a BCC.
b (above right). Dermoscopy showed a complex pattern with cerebriform areas, small white horn cysts but also some BCC-like areas. The diagnosis of SbK was confirmed by biopsy.

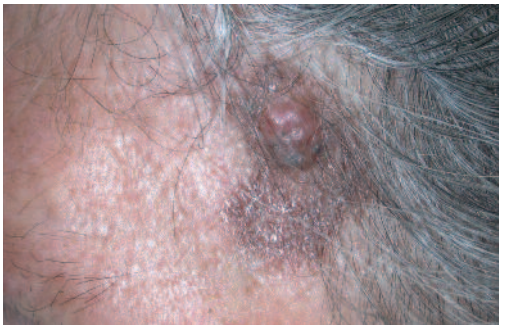


Figure 6. SbK of the temple with BCC-like central nodule. Dermoscopy suggested SbK, which was confirmed by biopsy.

a cerebriform structure or small white-yellow pseudohorn cysts are seen.

SbKs can become irritated spontaneously or from trauma (friction) and some develop into lichenoid keratosis (see below). Occasionally the lesions can become infected. They may regress spontaneously after these events.

Points of interest

The main difficulty is distinguishing SbKs from skin cancers, especially melanomas. This is particularly hard for patients who have many SbKs – a 'where's Wally?' situation. The ABCDE rule is useful for identifying melanoma (with 'E' for evolution). However, SbKs often fit the ABCDE rule. Patients have been shown to have a lot of difficulty using this type of approach.[8] I show my patients and their partners photographs of SbKs versus melanomas to help to demonstrate differences. I also ask them to report anything that they feel is unusual.

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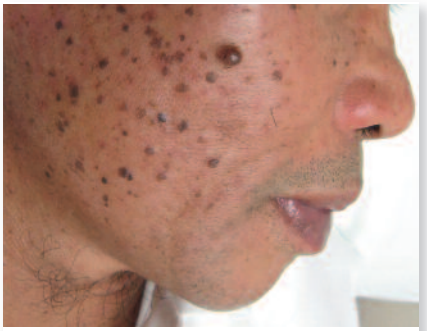

Figure 7. Dermatosis papulosa nigra.

Multiple eruptive SbKs are an uncommon presentation. They are usually small, red, wart-like lesions and often appear in large numbers over the trunk more than the limbs. These may develop after resolution of an inflammatory skin condition such as widespread dermatitis and they are also reported with HIV infection and acromegaly.

The presence of multiple eruptive SbKs raises the possibility of the sign of Leser-Trelat – that is, the sudden eruption of many SbKs, with or without pruritus, as an indicator of internal malignancy, particularly gastrointestinal adenocarcinoma or breast cancer. The true nature of this sign is disputed but it is worth considering, particularly when multiple eruptive SbKs occur in a younger patient and in the setting of a sudden onset of acanthosis nigricans.

There are isolated reports of various types of skin cancer developing in a SbK. However, it is likely that these are chance events and that the lesions are no more prone to cancer than the rest of the skin.

Treatment

SbKs are usually left alone. However, if treatment is desired then the following options can be considered.

- **Keratolytics.** These include over the counter heel balms and 6% salicylic acid in aqueous cream. The treatment is applied each night to soften the scale and make the lesion less rough. For patients with large numbers of SbKs, 70% glycolic acid chemical peels can be effective.
- **Cryotherapy.** This is usually the most practical method for treating SbKs. The degree of freezing needed will depend on the thickness of the lesion. There is a risk of unsightly hypopigmentation or donut-like pigmentation (dark rim, light interior), particularly in darker skinned people. Repeat freezing may be needed to clear large SbKs.
- **Shave excision or curettage and/or electrosurgery.** This treatment will leave a small scar, which may become hypertrophic.
- **Destructive laser ablation.**
- **Trichloroacetic acid.** Applications of trichloroacetic acid applications can clear SbKs, particularly thinner ones.
- **Tazarotene** (off-label). In a small study, topical tazarotene cream 0.1% was found to improve seven of 15 lesions.[9] However, this treatment is expensive and not usually used.

STUCCO KERATOSES

Stucco keratoses are closely related to SbKs but usually lack horn cysts. They are common, particularly in people with sun damaged skin.

Appearance

Stucco keratoses often occur in large numbers as small, rough, whitish, keratotic papules or plaques with the same 'stuck-on' appearance of SbKs (see above). The lesions usually occur on the legs and feet of older adults. They are easily picked off the skin with minimal bleeding. Patients may consider them to be unsightly, and women may find them annoying because they can cause tears in stockings.

Treatment

The treatment options for stucco keratoses are the same as those for SbKs (see above). However, their large numbers often makes treatment more difficult.

DERMATOSIS PAPULOSA NIGRA

The lesions of dermatosis papulosa nigra are very common in people with darker skin, especially those of African, Indian or Asian origin. The lesions may start in adolescence, but they are most commonly seen in older adults. Dermatosis papulosa nigra may be derived from hair follicles. The histopathology is similar to that of SbK.

Appearance

The lesions of dermatosis papulosa nigra appear as multiple, small, SbK-like papules or small plaques, which are often darker than the surrounding skin (Figure 7). They mainly occur on the face, mostly on the cheeks and forehead, but also on the rest of the head and neck, and much less on the upper trunk. New ones continue to appear slowly.

Treatment

Dermatosis papulosa nigra is usually left alone. If treatment is desired, the lesions are easily destroyed with light diathermy under local anaesthetic or EMLA. It is wise to treat a test area to see if unsightly post-inflammatory hypo- or hyperpigmentation occurs. Destructive laser ablation is an alternative treatment. Cryotherapy is less desirable because it has a higher risk of hypopigmentation.

LENTIGINES

Lentigines are a very common benign pigmented macule often confused with freckles (ephilides) or moles (melanocytic naevi). Histopathology shows an increased number of single melanocytes in the basal layer of the epidermis, often associated with elongation of the rete ridges (unlike moles, in which the melanocytes form nests). Freckles have a normal number of melanocytes, each producing more melanin than normal. Moles and lentigines have persistent pigmentation, whereas freckles fade in winter and darken with sun exposure.

The two main types of lentigo are:

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- lentigo simplex (which are solitary or few in number) – some transform into typical melanocytic naevi (with nests of melanocytes seen on histopathology) or hybrid pigmented lesions, which are still benign
- solar lentigo (which are multiple in number) – these occur particularly on previously sunburnt skin, months or many years later.

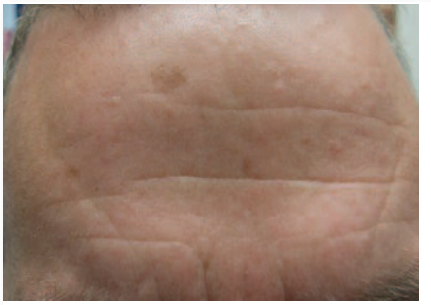

Figure 8. Lentigo, probably solar, on the forehead.

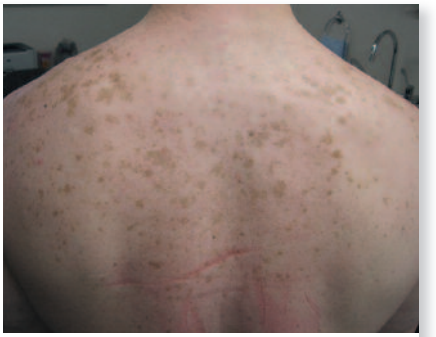

Figure 9. Multiple solar lentigines.

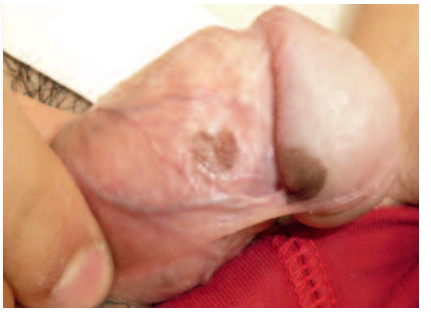

Figure 10. Penile melanotic macules.

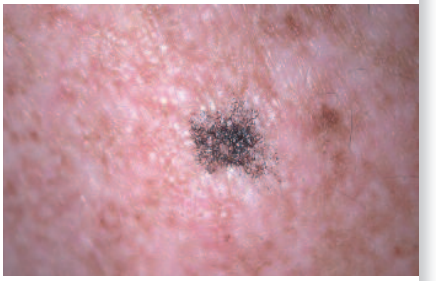

Figure 11. Ink spot lentigo on the back. This patient also had many solar lentigines.

Appearance

Lentigines appear as light-tan to mid-brown macules with a sharp outline (Figures 8 and 9). They can be up to a few centimetres in diameter. Most are evenly coloured but some are slightly two-toned. They are usually stable in size and colour but the bigger ones, in particular, often grow gradually – these can be difficult to distinguish from lentigo maligna (*in situ* melanoma, Hutchinson's melanotic freckle). Dermoscopy often struggles to help distinguish these; biopsy is required in this situation. Lentigo simplex differs from, but can be confused with, *café au lait* macules, which are larger and often oval in shape.

Lentigo simplex can occur anywhere, including the vermillion of the lips or the genitalia (melanotic macules, see Figure 10). Hyperpigmentation that is located on the lips, with or without intraoral melanotic macules, and pigmented streaks on the nails are features of Laugier–Hunziker syndrome. These lesions are not premalignant.

Ink spot lentigos, which are a subset of either solar lentigo or ephelides, are small, dark brown and angulated in shape (Figure 11). They are solitary or few in number, and usually located in a sea of more typical solar lentigines or ephelides, often on the upper back. They are most often seen in young people. Dermoscopy shows black reticular pigmentation.

Points of interest

Lentigo simplex can occur at any age, and usually appears as small (1 to 3 mm), light brown to dark brown lentigines, often on the proximal limbs, and may represent the earlier stage of a naevus (lentiginous junctional naevus). On histopathology, melanocytic naevi often show a lentiginous component (as defined above) and it is common for lentigo simplex to show isolated nests of melanocytes on serial sectioning.[10]

Solar lentigos are a marker of significant sun exposure and, thus, of an individual's overall increased skin cancer risk. Solar lentigos are usually benign, but there is debate about whether they can be precursor lesions to lentigo maligna. If so, the risk of transformation is so low that routine removal to prevent melanoma is not warranted. The main issue is whether or not to remove or biopsy a particular lesion based on clinical features – slow growth and substantial colour irregularity are good reasons to perform a biopsy. Occasionally, a solar lentigo transforms into a flat SbK; there is clinical and histological overlap between solar lentigos and SbKs.

There are several rare conditions in which the presence of multiple lentigos points to systemic issues. These include Peutz-Jeghers syndrome, Cronkhite-Canada syndrome (particularly seen in Japan) and Carney complex.

Treatment

If biopsy is not required then lentigines are generally left alone. However, if treatment is desired then the following options may be considered.

- **Light cryotherapy.** The risks of cryotherapy are less than for SbKs because the freezing required is less aggressive.
- **Laser treatment.** Pigment lesion laser (Q switched Nd-YAG, alexandrite or ruby, also KTP or fractionated lasers) or intense pulsed light (IPL) can be quite effective but treatment is not fully reliable. In addition, there is a

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risk of patchy hypopigmentation, particularly in darker skinned people.
- **Light to medium chemical peels.**
- **Cosmetics.** Patients may find camouflaging cosmetics helpful. Fading creams are usually ineffective.

LICHENOID KERATOSIS

Lichenoid keratosis (also known as benign lichenoid keratosis) represents a lichen planus-like immune attack against one of three entities: a lentigo, SbK or (precancerous) solar keratosis. Histopathology is required to distinguish these, but in practice biopsy is not always necessary because the lesions – particularly a lentigo or SbK – can be left untreated. The difficulty is it is often hard to distinguish lichenoid keratosis from a basal cell carcinoma (BCC), squamous cell carcinoma (SCC, particularly *in situ* type) or melanoma.

Appearance

The appearance of lichenoid keratosis depends on the activity of the process and time of presentation. The lesions are usually solitary and generally located on the upper trunk, distal upper limbs, face, bald scalp or neck (Figure 12). Early lichenoid keratosis is a red to dusky red-brown papule to plaque up to a centimetre or so in diameter. The lesion may be itchy or sting.

The inflammation is often mild, but occasionally it is quite active and even bullous forms occur. This lasts months to a year then settles, often with clearance of the original lesion. If, as is common, the original lesion was pigmented then residual post-inflammatory pigmentation remains, which is very slow to clear. Under the dermoscope this pigment often has a stippled appearance with a slight blue-grey tinge.

Treatment

Lichenoid keratosis that has developed from an existing lentigo or SbK is of cosmetic significance only and, as the condition settles spontaneously, such lesions can just be watched. However, a solar keratosis has the potential to transform into a SCC (although the likelihood is very low). Therefore, lichenoid keratosis that has developed from a solar keratosis is ideally treated with cryotherapy or shave excision (which has the advantage of providing specimens for histopathology but the disadvantage of leaving a scar).

The sudden onset and evolving nature of lichenoid keratoses mean it may be difficult to distinguish these lesions from skin cancers, so biopsy with excision is often required anyway. An alternative approach is observation with baseline photography and a set review date, which is appropriate if suspicion of melanoma is minimal. The post-inflammatory pigmentation is best left alone or concealed using cosmetics. Pigment lesion laser is an unreliable treatment, and fading creams do not work for this entity.

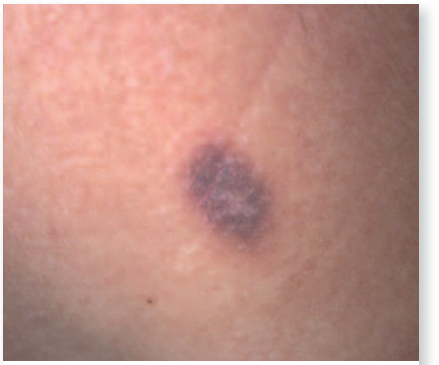


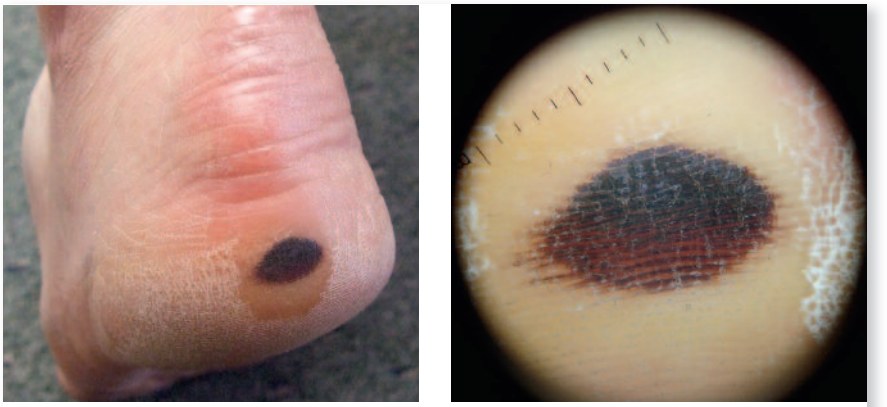
Figure 12. Lichenoid keratosis of the neck. Note the slight blue-grey hue and some remaining erythema. Dermoscopy showed stippled brown-grey pigment.

TALON NOIR

Talon noir (black heel) is simply a trauma-induced subepidermal bleed, usually from friction, in which the trauma is not enough to cause a blood blister (Figures 13a and b). They are easily mistaken for a melanocytic lesion like a melanoma because they appear quickly and can be large and irregular in shape. They usually occur in sports people and manual workers. Tache noir is the palmar version.

Appearance

Talon noir appears as a crimson-blue to brownish-black macule; it can be between a few millimetres and several centimetres in diameter. It occurs on sites of repeated friction, typically on the heel but sometimes on the toes. The macule may be continuous or broken with satellite macules. Dermoscopy highlights the



Figures 13a and b. Talon noir in a patient who is a runner. a (left). Clinical appearance. b (right). Dermoscopy showed predominantly brown pigment with a crimson tinge. The crimson colour is often more obvious.

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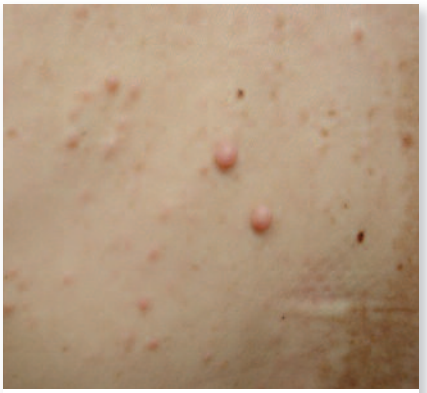

Figure 14. A localised collection of neurofibromas, probably nevoid neurofibromatosis, on the back.

crimson colour, which is usually on the top of the ridges and sometimes broken – this dermoscopic feature is known as 'pebbles on the ridges'. By distinction, continuous brown-black pigmentation on the ridges is a dermoscopic feature of acral lentiginous melanoma. The lesion gradually clears over months.

A similar, common lesion is the subungual haematoma, which is usually seen in toes (mostly the first, second or fifth) most prone to shearing trauma from footwear. It can also be caused by stubbing a toe or dropping an object on a toe.

Treatment

Patient reassurance is all that is required for those with talon noir. If the lesion is occurring repeatedly, a change in the design or fit of footwear may help.

SKIN TAGS

Skin tags (fibroepithelial polyps, soft fibromas, acrochordons) are very common. Histopathology shows the bulk of the tag is loose fibrous tissue, similar to the papillary dermis.

Appearance

Skin tags usually appear as multiple, polypoid lesions of small to medium size. They are usually found on the neck, upper chest and skinfolds, particularly the axillae and groin. The tags can be flesh-coloured, red or brown. They can become irritated by constant rubbing or catching on clothing. New ones continue to appear gradually.

Points of interest

Skin tags are more common in overweight people and may coexist with acanthosis nigricans. These are both pointers to type 2 diabetes and thus insulin resistance, being consequences of elevated levels of insulin or other hormones (both, for instance, may feature in acromegaly from excess growth hormone).[11] There may also be features of hyperandrogenism or lipodystrophy. It is not known whether

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substantial weight loss will finally reduce the number of skin tags or the number forming, but it is a good opportunity to press for a meaningful weight loss program. Fasting glucose should be monitored periodically, even if the patient is not currently diabetic.

Treatment

Skin tags are both a nuisance and a cosmetic concern, so patients often want them removed. The easiest approach is physical destruction with shave–snip excision and diathermy or a styptic like aluminium acetate hexahydrate to stop bleeding.

Destructive laser or, less reliably, cryotherapy are alternative treatments. Treated lesions heal slowly and unsightly postinflammatory hypo- or hyperpigmentation may remain, particularly in darker skinned people, so initial removal of only a few tags (as a test treatment) is recommended.

LARGE SOFT FIBROMAS

Large soft fibromas have a similar histopathology to skin tags but often have prominent fat content ('lipofibroma'). Polypoid intradermal melanocytic naevi and neurofibromas can look the same.

Appearance

A large soft fibroma appears similar to a large, flesh-coloured skin tag, usually on the trunk or proximal limb. The lesions, which are solitary or few in number, are more common in obese people but not necessarily associated with multiple skin tags.

Treatment

Large soft fibromas are removed by shave excision.

NEUROFIBROMAS

Neurofibromas are a variety of skin tumours of neural origin (Figure 14). Solitary neurofibromas are the most common. They arise from the endoneurium and contain a mixture of Schwann cells, fibroblasts and perineural fibroblasts with residual nerve axons. They are easily mistaken for intradermal melanocytic naevi or large soft fibromas.

Appearance

A neurofibroma appears as a slow growing, small, flesh-coloured papulonodule or polyp, between a few millimetres and 2 cm in diameter. They demonstrate the 'buttonhole sign', where protruding skin can easily be pushed into an apparent hole in the dermis. The lesions can occur at any site and are most often seen in adults.

Rare clinical variants include:

- diffuse neurofibromas, usually a solitary plaque affecting the trunk, head or neck of a teenager or young adult; these are not necessarily a part of neurofibromatosis type 1 (NF-1) and are different from the deeper often quite large plexiform neurofibromas (which are pathognomonic of NF-1)
- pigmented neurofibromas (nodules to large plaques); at least half of the patients with pigmented neurofibromas have NF-1.

Points of interest

Malignant transformation in neurofibromas is very rare. Solitary lesions are not related to NF-1. About 10% of patients who have common neurofibromas have multiple lesions; other features of NF-1 should be sought in these patients (e.g. multiple *café au lait* macules, axillary freckling).

Treatment

Treatment of neurofibromas, if desired, involves excision. MT

Parts 2 and 3 of this article about benign skin lesions, to be published in future issues of Medicine Today, will discuss melanocytic lesions and vascular, dermal and adnexal tumours.

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COMPETING INTERESTS: None.

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