Benign skin lesions – Part 2
Melanocytic lesions

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This month we continue a three-part article on benign skin 'lumps and bumps' that may be encountered in general practice. Part 2 focuses on melanocytic lesions, including some variants of melanocytic naevi.

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This is the second in a series of three articles of benign skin lesions. Part 2 focuses on a range of melanocytic lesions, including some variants of melanocytic naevi. Common forms of melanocytic naevi (junctional, compound and intradermal melanocytic naevi) are not covered in this series, as these are described to some extent in my earlier article, entitled 'Checking pigmented skin lesions', which was published in 2007 in *Medicine Today*.[1]

HALO NAEVUS

Halo naevus is common and seen particularly in children and young adults (Figure 1). In the eastern Australian childhood nevus study, 5.3% of schoolchildren (aged 6 to 15 years) were found to have at least one halo naevus; 0.3% of children had three or more.[2]

In halo naevi, there is a vitiligo-like immune attack against the melanocytes of the parent pigmented lesion, but the level of inflammation in the mole component is considerably more than that seen in vitiligo. The halo process can occur against a variety of pigmented lesions, which is usually typical melanocytic naevi (moles). Immunologically-induced regression of a melanoma is a similar concept, where there is usually partial white change, sometimes with scarring, within a melanoma; however, halo change occurs around the pigmented lesion and often leads to clearing of the pigmented lesion. True halo change can also occur around a melanoma as a part of regression.

It is not known why only a small proportion of an affected individual's moles undergo halo change – presumably the pigmented lesion becomes immunogenic or loses immune privilege. Why this occurs for multiple pigmented lesions in different sites is mysterious. Occasionally, halo change is an indicator of vitiligo – occurring either concurrently or later.

Appearance

Halo naevi have vitiligo-like loss of skin colour around a pigmented lesion, which is usually a flat or nodular melanocytic naevus. Usually halo naevi are asymptomatic, but occasionally they are itchy or inflamed. The mole itself usually involutes within a year or so. Typically, the depigmented halo gradually repigments within a year or two, unless vitiligo supervenes.

Treatment

Unless there are features of melanoma (rare, particularly in young children), halo naevi are usually left alone because they regress spontaneously.

MEYERSON'S NAEVUS

Meyerson's naevus is a melanocytic naevus with dermatitis on and around a mole. Unlike in halo naevus, the mole

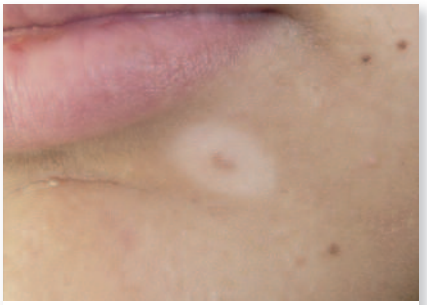


Figure 1. Halo naevus. Note that the mole is starting to disappear.

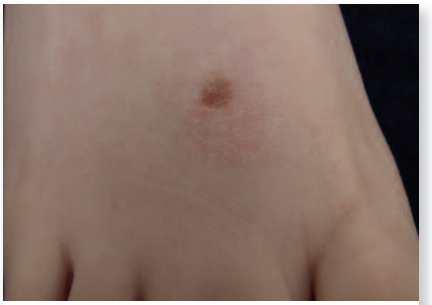


Figure 2. Meyerson's naevus of the foot with surrounding dermatitis.

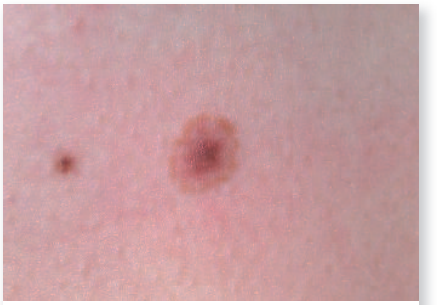


Figure 3. Cockarde naevus on the abdomen.

only occasionally eventually disappears. The frequency of Meyerson's naevus is not reported, but it is uncommon.

Appearance

Meyerson's naevus is a normal melanocytic naevus that later develops red, dry dermatitis on and around the mole (Figure 2). It is frequently itchy. Meyerson's naevus is often solitary, but an individual may have more than one affected mole. There may or may not be atopic dermatitis elsewhere. A case of similar change has been reported around an early melanoma.[3]

Treatment

Potent topical corticosteroids will settle the dermatitis of Meyerson's naevus, but it may recur.

COCKARDE NAEVUS

Cockarde naevus is uncommonly reported, but it is not infrequently seen in clinical practice. It is a benign variant of a melanocytic naevus, with no known excess malignant potential.

Appearance

Cockarde naevus has darker tan to brown pigment around the entire rim of the pigmented lesion (Figure 3). Centrally, there may be a darker junctional or papular component, so potentially there may be three tones. The pigmentation is symmetrical and the dermoscopic features benign. The lesions can be multiple and may occur on any site but seem to be more common on the scalp.

Treatment

The treatment for cockarde naevus is the same as for any other mole.

BLUE NAEVUS

Blue naevus is a benign melanocytic naevus seen in about 1 to 3% of Australians. It is more common in Asian populations. In the eastern Australian childhood nevus study, 1.2% of schoolchildren (aged 6 to 15 years) were found to have at least one blue naevus; 0.1% of children had three or more.[2] The blue colour is caused by the deep location of melanocytes in the dermis and the Tyndall effect, in which red light is diffused by the dermis and blue light is allowed to be reflected.

Appearance

There are two main types of blue naevi: 'common' and 'cellular'.

'Common' blue naevi are small, macular, and sometimes mildly raised (Figure 4). They often look like a small tattoo. Colour is an even or two-tone dark blue-grey. Occasionally they have a paler grey-brown centre and a blue rim. They are usually stable in size and can occur on any site (including, occasionally, the mucosal surfaces).

'Cellular' blue naevi are less common, bigger papules to nodules (up to 3 cm). They are usually seen on the dorsal feet and hands (Figure 5), buttocks or face.

Rarer variants of blue naevus include combined naevus, which has elements of a blue naevus and a common acquired melanocytic naevus. Another rare variant is the deep penetrating naevus, which occurs more often on the head and neck,

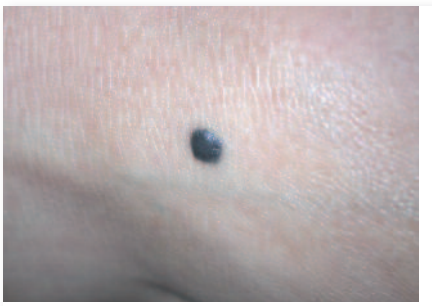


Figure 4. Papular blue naevus on the side of the foot.

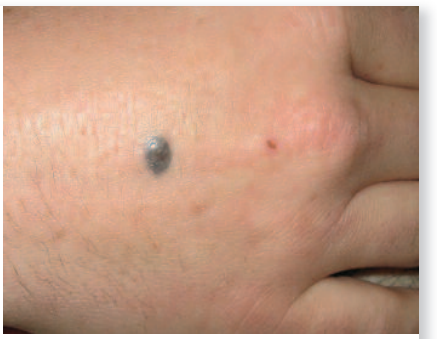


Figure 5. Cellular blue naevus.

with a deep blue or black colour and a diffuse, irregular colour laterally. A deep penetrating naevus can have the histological features of a Spitz naevus; both look unusual so are often excised.

Multiple blue naevi are a feature of the rare Carney complex, occurring together with lentigines on the central face and genitals, atrial myxomas (prone to causing embolic strokes) and harmless mucocutaneous myxomas. Malignant (melanoma) change in a blue naevus is reported very rarely.

Points of interest

Blue naevi differ from the various types of dermal melanocytosis that histologically show melanocytes dispersed in a ribbon-like pattern between the dermal collagen and around neurovascular bundles. The most common type of dermal melanocytosis is 'mongolian spots', where there is macular confluent or patchy larger areas of blue-grey pigmentation in the lumbosacral area or beyond, present at birth. They usually disappear during the first decade of life. They are commonly seen in infants from Asia but are rare in white-skinned European infants.

Other forms of dermal melanocytosis, also most often seen in people from Asia, are naevus of Ota (unilateral dermal melanocytosis on the forehead, temples and cheeks plus sclera and sometimes oral mucosa) and naevus of Ito (unilateral, over a shoulder and upper trunk). These naevi are generally not present at birth, but appear during childhood and persist. There are very rare reports of melanoma, including meningeal melanoma, developing in naevus of Ota.

Treatment

Blue naevi do not usually require treatment. Occasionally, however, *de novo* melanoma or skin metastases of melanoma resemble blue naevi. Progressive enlargement in a blue naevus is uncommon, and if it is occurring to a significant degree then the lesion should be excised. However, most commonly excision is performed for cosmetic reasons.

ATYPICAL (DYSPLASTIC) MELANOCYTIC NAEVUS

'Atypical' melanocytic naevus (AMN) is a clinical description of an entity diagnosed histologically as 'dysplastic' melanocytic naevus (DMN). There are multiple pathological criteria for diagnosis of DMN, including cellular and architectural atypia (dysplasia) – not enough to call melanoma. Dermatologists who decide to leave these lesions alone often describe them as clinically atypical. An AMN is not always described by the pathologist as a DMN – and vice versa – the correlation is not high.

These lesions are common. The eastern Australian childhood nevus study found 21% of 15-year-old schoolchildren had at least one AMN.[2] Across the study population (aged 6 to 15 years), 12% of children had one and 2.7% had three or more (biopsies were not performed).[2]

Appearance

There is a clinical continuum between the various appearances of benign melanocytic naevi and melanoma, with AMN straddling the two. Although the diagnosis of moles is usually straightforward, the wide range of clinical variants of these and other pigmented lesions makes the distinction of benign from malignant sometimes difficult. A useful description of AMN is:

- presence of a macular component for some or all of the lesion; if a raised component is present it is often in the centre
- size ≥5 mm
- irregular, 'fuzzy' border
- irregular, not necessarily symmetrical, pigmentation within the lesion.

AMN are sometimes described as a 'fried egg' in appearance (Figure 6), but this description is too limiting. Unlike melanoma, AMN tend to be stable in size over a prolonged period of time; if enlarging significantly then biopsy or excision is warranted. They are most commonly located on the trunk or proximal limbs (Figures 7 and 8).

Points of interest

AMN are probably the result of a more proliferative phenotype of the nested melanocytes, responsible for the histological features and slightly increased melanoma risk. There are genetic and histochemical markers suggesting AMN are intermediate between benign melanocytic naevus and melanoma.[4]

Melanoma risk

An Australian study found 23% of 1101 melanomas had histological evidence of a pre-existing mole (56% benign melanocytic naevus, 38% dysplastic and 6% congenital).[5] For superficial spreading melanomas, 26% showed an associated melanocytic naevus versus 3% for nodular melanoma.[5] Other studies have found higher rates (e.g. 58% and 51%).[6,7] For a 20-year-old individual, the estimated lifetime risk of a single benign melanocytic naevus turning into a melanoma is roughly 1:3000 for men and 1:10,000 for women. The transformation risk per mole is higher particularly in older men. For AMN, the risk may be about five times higher.[8]

People with multiple AMN have a higher risk of developing a melanoma anywhere on the skin – more often from normal skin than from a pre-existing pigmented lesion. In one study, the presence of one AMN was associated with a two-fold increase in melanoma risk; 10 or more AMN were associated with a 12-fold increase.[9] In a meta-analysis researchers found that an individual with 100 to 115 benign melanocytic naevi has a melanoma risk that is seven to 12 times higher than that for an individual with 10 to 15 benign melanocytic naevi. They found about a six-fold higher melanoma risk in an individual with five AMN,

compared with no AMN.[10] (Note the average number of benign melanocytic naevi in 15-year-old Australians is 68,[11] so the comparison with people with 10 to 15 of these lesions is not to the population norm.)

Atypical (dysplastic) mole syndrome

The atypical (dysplastic) mole syndrome (AMS) is characterised by a significantly increased number of benign melanocytic naevus and AMN (roughly more than 100 and five, respectively). This condition is often familial. It is common for people with a high mole count to develop the occasional new benign melanocytic naevus or AMN – even by middle age. Normally, an individual's mole count gradually diminishes with age from its peak at about 40 years of age.

High counts of benign melanocytic naevi, particularly with higher numbers of AMN (as in the atypical mole syndrome), are strong clinical markers for melanoma risk. It has recently been recognised that a subset of people with AMS and a family history of AMS also have an increased familial risk of pancreatic cancer (by carrying mutations of the *CDKN2A* oncogene). Affected family members should be offered appropriate surveillance.[12]

Atypical lentiginous melanocytic naevus

A separate entity is the atypical lentiginous melanocytic naevus, which is seen in older patients with chronic sun damaged skin, often in a sea of other features of sun damage.[13] These lesions are usually located on the trunk or limbs and are generally solitary or few in number. Often they appear as macules, less than 1 cm in diameter, with some variation in colour and border. Dermoscopy shows an indistinct border and a more irregular and broken pigment network.

Atypical lentiginous melanocytic naevi are prone to change and clinically they are often indistinguishable from lentigo maligna (which are more commonly seen on the head and neck) so biopsy is usually required. It can be a challenge for the pathologist to distinguish this type of naevus from melanoma, and probably a significant proportion are evolving to lentigo maligna and in some cases to small cell (naevoid) melanomas.[13]

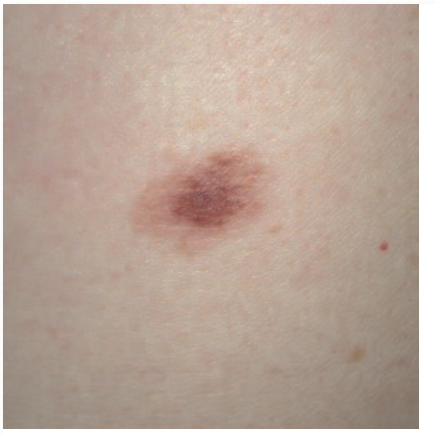


Figure 6. An AMN that has the 'fried egg' appearance.

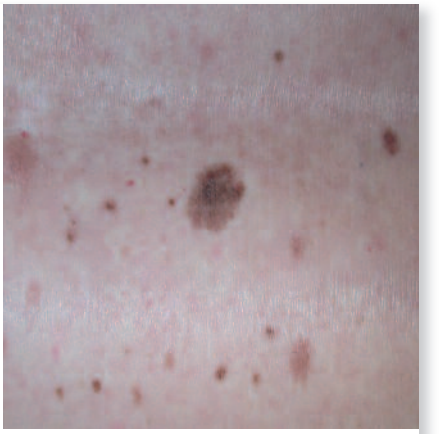


Figure 7. AMN located on the back.

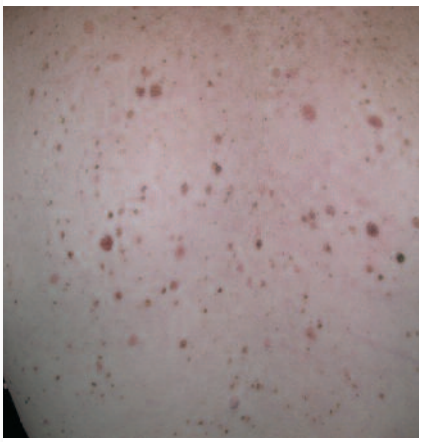


Figure 8. Many moles, with a considerable number of clinically atypical melanocytic naevi, on a man's back.

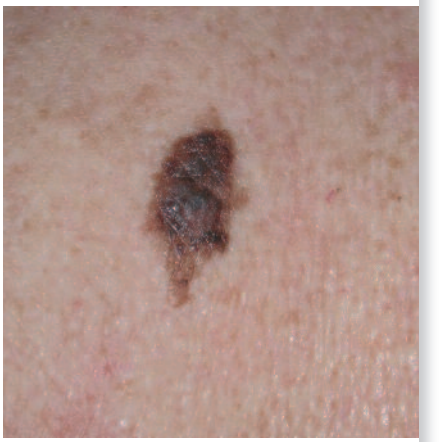
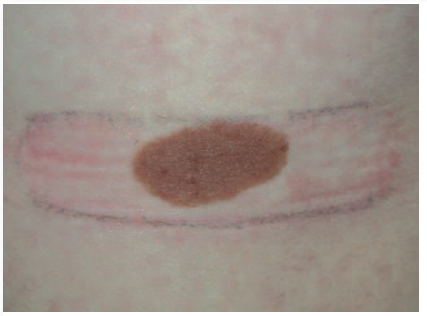


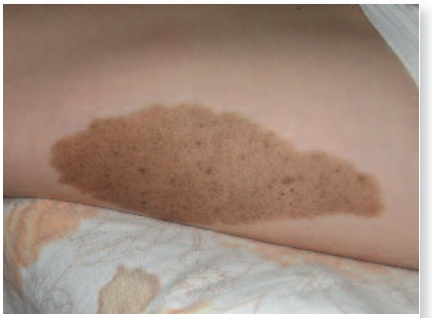
Figure 9. Atypical (dysplastic) naevus, proven by biopsy. The lesion has clinical features suggesting possible melanoma.

Treatment

The important task is to distinguish AMN from melanoma (Figure 9). Individuals with thin melanomas have a high five-year survival rate (compared with thicker melanomas), so early detection is vital. Clinically AMN often fit the ABCDE description of melanoma. Dermoscopy is of assistance, but its diagnostic accuracy is insufficient and dependent on experience. Very helpful is the usual size stability over a prolonged period. Overall, the distinction has a degree of gestalt. Photographic surveillance and mole mapping services are very helpful, especially for an individual with a high mole count or atypical naevus syndrome.

It is important to point out to patients





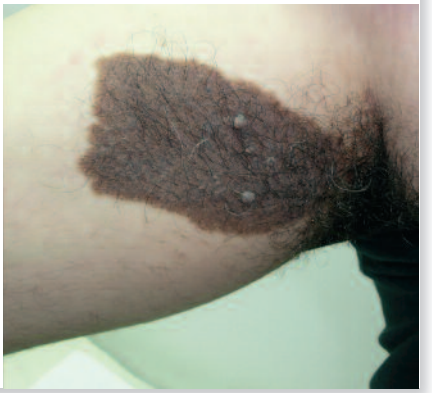


Figure 10 (above). Small CMN on the abdomen, which had been unnecessarily covered for sun protection.

Figure 11 (top right). Intermediate CMN on the thigh of a teenage boy.

Figure 12 (right). CMN with darker hairs located near the axilla. Note the small nodules within the naevus, which were stable in size for a few years so were left alone.

that if a melanoma appears, it is more likely to arise from normal skin than from a pre-existing mole. I show patients and their partners (or parents) photographs of benign melanocytic naevi compared with AMN and melanoma. Some patients want to have an AMN removed for purely cosmetic reasons. The general view is the risk of AMN transforming into melanoma is too low to recommend their routine removal.[14] The judgement to remove or observe is a balance of clinical features, desire and anxiety of the patient and doctor versus the cost and morbidity of removal and the risk of unsightly scarring. I have a low threshold for removing AMN because pathology is the final arbiter.

Deep shave excision or formal excision and suture are the best methods for removal (so, if the lesion is a melanoma, its depth can be determined). Cryotherapy or laser ablation is not recommended because there is no histopathology. The main alternative is close clinical surveillance, with photography (including dermoscopic photography) and lesion removal if there is subsequent change.

People who have AMN removed are sometimes told 'it was close to turning to a melanoma'. I do not phrase it this way because these are usually stable lesions with a low risk of transformation. Experienced skin histopathologists often grade DMN as mildly, moderately or severely dysplastic. The severely dysplastic grade is more difficult to distinguish from melanoma, so if excision is incomplete then re-excision is usually recommended. Here, clinicians vary as to the safety margin they take (up to 5 mm, depending on the level of suspicion of melanoma). Incomplete excision of a mildly dysplastic naevus is less concerning and the decision to re-excise is as much to do with the level of anxiety of the patient or clinician.

An instructive study of 278 adults with five or more AMN followed with photographic surveillance for a mean of 42 months in a private dermatology practice in Melbourne found 20 new melanomas in 16 patients (46 times the age-adjusted incidence in the Victorian population in 1990).[14] Of these, 11 were detected via changes using baseline photographs and the rest by patients or their partners. Thirteen of the melanomas arose as new lesions and only three were from an AMN. Melanoma risk rose with increasing number of AMN. The study involved 1554 consultations (78 for each melanoma) and 210 biopsies on 104 patients (10 biopsies for each melanoma).[14] Not all changes in pigmented lesions led to a biopsy – some were just observed.[14] A later study at the same practice showed the value of removing new or changed pigmented lesions in high-risk patients, with a higher rate of pre-existing AMN in the melanomas being detected in this study.[15]

CONGENITAL MELANOCYTIC NAEVUS

Congenital melanocytic naevus (CMN), by definition, is present at birth. Congenital-like melanocytic naevus appears after birth (usually before the age of 2 years) but has the same histological pattern of CMN. Arbitrarily, these lesions have been classified by size. Most are small (<1.5 cm, Figure 10), some are intermediate (1.5 to 20 cm, Figure 11), and the rare lesion (incidence <0.005%) is giant (>20 cm).

There are differences in the histopathology of CMN compared with acquired melanocytic naevi, which are much more common. Melanocytic nests in CMN are located more deeply – to underlying fat or beyond in some. In the eastern Australian childhood nevus study, 4.4% of schoolchildren (aged 6 to 15 years) were found to have at least one congenital-like melanocytic naevus, 0.2% of children had two, and none had more.[2]

Appearance

CMN are usually dark brown. Initially they may be macules or plaques, usually oval in shape. As the child gets older, CMN increase in size in proportion to the child's growth, but may darken, lighten or become thicker or more warty (verrucous).

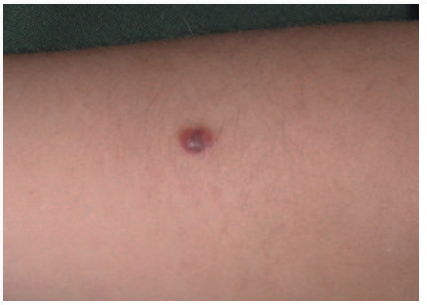


Figure 13. Spitz naevus on a girl's forearm.

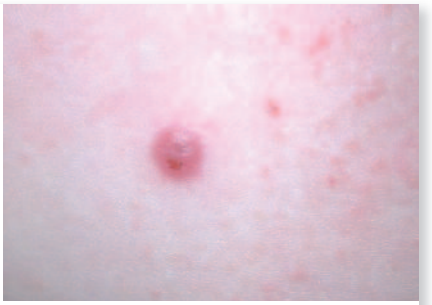


Figure 14. Spitz naevus on the thigh of a baby girl.

CMN sometimes develop an additional nodule (or nodules) within them, which vary from flesh coloured to dark brown to black and probably reflect a clonal change in an area of the naevus (Figure 12). In this case, it is usually wise to biopsy or excise the nodules or the whole mole because the change can be due to malignancy – usually melanoma.

CMN often have thicker or darker hairs within the mole. In giant CMN, these hairs are often in a whorled pattern. For CMN located on the scalp, these hairs tend to be quite dark at birth and may be more like pubic hairs. Over time (years) it is common for these hairs and the CMN to partially spontaneously normalise, markedly improving the cosmetic appearance.

A giant CMN may be one big plaque, often with new satellite lesions gradually appearing as the child gets older. Some giant CMN are multiple smaller CMN grouped in an area (agminated giant CMN).

Points of interest

The main issues for CMN are appearance and melanoma risk. The latter is in proportion to the size of the CMN. Small and smaller medium-sized CMN have a small risk – insufficient to recommend routine excision. Giant CMN have an elevated risk (estimated from 4.5% to 14%, with a significant part of that risk occurring by 15 to 20 years of age), so they are different from other melanomas. They appear as a nodule or other change to the CMN. However, in one large systematic review of CMN, metastatic melanoma with an unknown primary occurred in seven cases (14%).[16]

Neurocutaneous melanosis is an infrequent consequence of giant CMN, usually of those concentrated on the lower back of the head or neck. Here, there is leptomeningeal melanosis, sometimes causing life-threatening raised intracranial pressure and, occasionally, primary neural melanomas. Neurocutaneous melanosis is diagnosed by MRI, which will also detect asymptomatic cases. There is debate whether MRI should be performed routinely in patients with giant CMN, as treatment options for neurocutaneous melanosis are few. Patients and their parents should keep a sensible eye on a CMN. Digital photographs of CMN (showing a tape measure), taken by doctor or parents, are helpful in detecting significant change.

Treatment

Most CMN can be safely left alone and observed by the patient and parents. Giant and large CMN should be periodically checked by a doctor. If treatment is desired or necessary because there are changes in the CMN or they are causing problems (e.g. rubbing on clothing) then excision and suture will be the best option, with the possibility of scarring and other surgical morbidities being the main issue.

Small CMN are easy to remove but have the least need to be removed. Giant CMN are difficult to remove – if this is attempted the cosmetic result is likely to be poor and it is unknown if debulking alters the melanoma risk. As the pigment is deep, they may regrow after more superficial methods like curettage. Pigment lesion laser is useful for hair removal but does not penetrate deeply enough to remove enough of the CMN. Shave excision or curettage can be used for debulking, which may flatten CMN enough to get value from camouflaging cosmetics or Microskin camouflage.

SPITZ NAEVUS

American pathologist Dr Sophie Spitz described these as 'juvenile melanomas' in 1948 as a result of their unusual pathological appearance. It is now recognised that most are not melanomas – thus the current name of Spitz naevus (also known as spindle and epithelioid cell naevus). There are three types, which are sometimes difficult to distinguish from each other:

- Spitz naevus (benign)
- atypical Spitzoid tumour of unknown malignant potential, and
- possibly, Spitzoid melanoma (which some pathologists do not differentiate from typical melanoma).

Some authors include the spindle cell naevus of Reed (see below) as a fourth type of Spitz naevus.

Australian data show the prevalence of Spitz naevus to be 1.4 per 100,000 population.[2] Around 50% occur before 14 years of age, 25% between 14 and 30 years and 25% over the age of 30 years.

Appearance

Spitz naevus is usually a firm, dome-shaped or ovoid small nodule (Figures 13 and 14). Colour is a fairly even red or reddish-brown (usually light brown but can be dark). The colour is due to an increased vascular supply, and often a few telangiectatic vessels are visible. They are prone to mild bleeding. Blanching with a dermoscope shows their true colour.

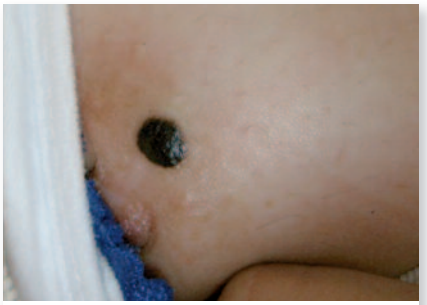


Figure 15. Pigmented spindle cell naevus of Reed located near the nipple. Dermoscopy suggested the diagnosis, which was confirmed on deep shave excision.

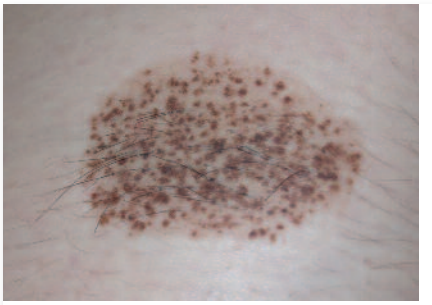


Figure 16 (above). Speckled and lentiginous naevus on the thigh.

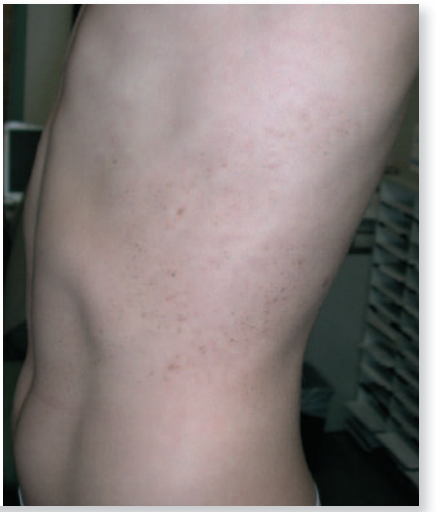


Figure 17 (right). Large (segmental) naevus spilus on the flank of a teenager.

Spitz naevi usually grow moderately rapidly over three to six months and can reach a diameter of up to 2 cm but are usually less than 1 cm. About 40% occur on the head and neck.

Rare variants include agminate Spitz naevus (a group of otherwise typical Spitz naevi in an area with a faint tan background), which is mainly seen in children. Multiple Spitz naevi (up to hundreds of otherwise typical Spitz naevi all over the body except the palms, soles or mucosae) are very rare and mainly seen in adults. Desmoplastic Spitz naevi (firm-to-feel, small red nodules like small keloids) is a rare variant that is mainly seen in adults.

Treatment

Histopathology is the key to diagnosing Spitz naevi. The distinction from melanoma can be difficult. The first step is to recognise the entity clinically, as biopsy is recommended if a Spitz naevus is suspected and excision with a clear margin if it is diagnosed on biopsy or strongly suspected clinically. It is very rare for Spitz naevi to metastasis and it is felt that, if metastasis does occur, prognosis is better than for melanoma – this entity is hotly debated in terms of whether it is typical melanoma.

If a decision is made not to biopsy or excise a typical Spitz naevus in a young child then the lesion should be measured and observed three monthly for significant change. In this situation, it is best to refer the child to a paediatric dermatology or surgery unit where the lesion can be excised under sedation or general anaesthesia.

SPINDLE CELL NAEVUS OF REED

Spindle cell naevus of Reed is considered a variant of the Spitz naevus by some, but most dermatologists consider it to be a separate entity. It is uncommon. Most affected patients are teenagers or young adults, and female.

Appearance

Spindle cell naevus of Reed is a small (3 to 6 mm), often new, single macule or a slightly raised papule to small nodule (Figure 15). The lesions are round or somewhat irregular or angular in shape, and brown in colour to densely pigmented. They occur most commonly on the limbs, particularly the thighs; once they have appeared they are usually stable in size. Under the dermoscope there is even dark colour, sometimes with peripheral radial streaming (starburst pattern) or globules.

Points of interest

Spindle cell naevus of Reed is a benign lesion. It probably has the same malignant potential as a normal mole, possibly more if it is a variant of Spitz naevus but there are no data suggesting this is an issue.

Treatment

A spindle cell naevus of Reed is often removed as a possible melanoma. If a lesion is incompletely excised, it should be re-excised with a narrow margin. If pathology shows atypical features then a somewhat wider margin (say, 5 mm) is recommended and the patient should be reviewed as if the lesion were a melanoma.

SPECKLED AND LENTIGINOUS NAEVUS

Speckled and lentiginous naevus (SLN, also known as naevus spilus) is present at or near birth, so it is a type of congenital melanocytic lesion. It is reasonably common – the eastern Australian childhood nevus study found 1.8% of schoolchildren (6 to 15 years of age) had at least one naevus spilus; 0.1% had two and none had more.[2]

Appearance

In SLN, there is a faint to mildly tan *café au lait* macule-like background with small darker moles peppered within it (Figure 16). The lesions are usually 1 to 4 cm in size. They can be large – appearing in a unilateral, segmental or zosteriform

distribution or follow Blaschko's (embryonal development) lines and can involve a substantial portion of skin – say, an entire extremity or half the trunk (Figure 17).

Points of interest

SLN probably results from somatic mutations occurring during embryogenesis so only part of the skin is affected. Pathology of the *café au lait* macule-like background shows a mild lentiginous proliferation of melanocytes. The darker pigmented lesions within are usually lentigos or benign melanocytic naevi but uncommonly are all or in part AMN, blue naevi or Spitz naevi. There are rare reports of melanoma developing within an SLN.

Treatment

Generally, SLN are simply observed because the resultant scar from excising them is usually uglier than the SLN (particularly for larger lesions). Pigment lesion laser or intense pulsed light (IPL) therapy may be effective. The lesions are largely macular so cosmetic camouflaging techniques are an option. Given that there are reports of melanoma developing within SLN, the lesions should be photographed, measured and observed. MT

Part 3 of this article about benign skin lesions, to be published in a future issue of Medicine Today, will discuss vascular, dermal and adnexal tumours.

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