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Dermatopathology

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Preface

In any histopathology department, cutaneous biopsies form the major part of the workload. In many instances these may be readily dealt with by experienced pathologists and their documentation represents an essential step in organising therapy. However, in a number of areas dramatic changes have occurred in dermatopathology. The rapidly changing incidence of pigmented lesions of the skin and the availability of diagnostic cell markers in the complex cutaneous lymphomas have meant that for many pathologists a review in these fields will be valuable. The distribution and pattern of skin pigmentation may allow us to make important assertions and draw important conclusions about the genetics of skin disease, but also about human variability. Cutaneous vasculitis is a difficult problem where clinicopathological consultation is vital in diagnosis and the review presented has been written with this very much in mind. In the same way, many diagnostic difficulties exist in assessing buttous lesions in the skin and for this reason a review was invited.

Finally, our understanding of certain skin diseases has been radically altered by knowledge of the dynamics of the changes seen, a field which has also been studied with advantage in other systems in pathology.

This volume is intended as an aide to those many pathologists and dermatologists who, often together, are responsible for the provision of clinical care in this demanding field.

London C.L. BERRY

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Melanoma and Other Melanocytic Skin Lesions

D. WEEDON

Introduction

The literature on malignant melanoma and other melanocytic skin lesions has expanded greatly in recent years making it difficult for the average clinician and pathologist to keep abreast of the recent trends. The introduction of new statistical techniques such as multiple regression analyses and the Cox proportional hazards model has provided new insights into the important prognostic variables in melanoma. Furthermore, several new concepts such as the dysplastic naevus syndrome and minimal deviation melanoma have emerged in recent years which have profound significance on the interpretation of some melanocytic lesions. It therefore seems timely to review this subject, with an emphasis on those aspects which have been published in recent years, or which are neglected in standard textbooks of dermatopathology.

1. Lesions with Basal Hyperpigmentation

Increased melanin can be found in the basal layer in many different circumstances. These include racial pigmentation, chloasma of pregnancy, in a freckle, associated with heavy metal pigmentation, certain endocrine disturbances, Albright's syndrome, Peutz Jegher's syndrome, urticaria pigmentosa, acromelanosis and the various punctate and reticulate hyperpigmentation syndromes. These have recently been reviewed (FULK 1984), and it is not proposed to discuss this group further.

2. Lesions with Basal Melanocyte Proliferation

2.1 Lentigo Simplex

Simple lentigos are small, usually uniformly pigmented macules without predilection for sun-exposed areas. Multiple lentigines may be unilateral in distribution (THOMPSON & DIEHL 1980) or generalized, the latter form sometimes being a marker of underlying developmental defects. These syndromes which include the Leopard syndrome have recently been reviewed, and will not be considered further (FULK 1984). Atrial myxoma has also been reported in association with lentigines (PETERSON & SERRILL 1984).

On histological examination, a lentigo shows basal hyperpigmentation with an increased number of single melanocytes within the basal layer. There is usually epidermal hyperplasia with regular elongation of the rete ridges. There is often a sparse lymphohistiocytic infiltrate in the papillary dermis, including scattered melanophages. Some lesions form junctional nests and evolve into lentiginous naevi (ACKERMAN & RAGAZ 1984). These lesions are discussed in Section 2.5.

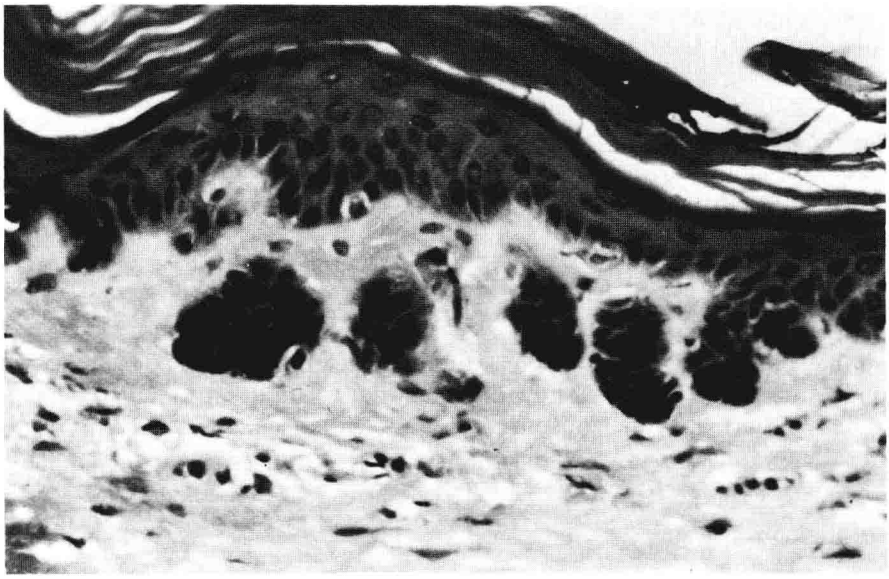

Fig. 1. Solar lentigo. There is bulbous expansion of the rete ridges

2.2 Solar Lentigo

Solar (senile) lentigos are light to dark brown macules, usually 5–15 mm in diameter which develop on the sun-damaged skin of elderly patients. They are often multiple. They may increase in size slowly over many years. ACKERMAN & RAGAZ (1984) believe that patches of solar lentigo evolve into reticulated seborrhoeic keratoses.

On histological examination, there is elongation of the rete ridges, some of which are short and bulb-like, while others have finger-like rete ridges connecting with adjacent rete to form a reticular framework (Fig. 1). These latter lesions resemble so-called seborrhoeic keratoses of reticulate type. There is basal hyperpigmentation, usually some melanophages in the papillary dermis, and often solar elastosis. There is an increase in melanocytes, particularly at the bases of the clubbed and budding rete ridges (MONTAGNA et al. 1980), although sometimes the increase is not noticed on casual examination of a slide. On electron microscopy, the melanosome complexes inside the keratinocytes are much larger than those found in non-involved skin (MONTAGNA et al. 1980).

2.3 Becker's Melanosis

Becker's melanosis, also known as Becker's naevus is usually found in the region of the shoulder girdle of young men as unilateral, hyperpigmented, irregular areas. Hypertrichosis develops after the pigmentation. Recently, lesional tissue

has been found to show an increased level of androgen receptors, suggesting that exquisite local androgen sensitivity may explain the clinical manifestations (PERSON & LONGCOPE 1984). There may be associated structural abnormalities (GLINICK et al. 1983).

On histological examination there may be slight acanthosis, with basal hyperpigmentation and some melanin incontinence. Melanocyte proliferation is mild and not always obvious. Hair follicles may be enlarged and there may be smooth muscle hyperplasia or hamartomas in the dermis.

2.4 Café au Lait Spots

In routine H and E sections, no increase in basal melanocytes can be recognized, although an increase has been shown by quantitative studies. In DOPA-stained epidermal sheets, giant melanosomes may be seen in café-au-lait spots from many patients with neurofibromatosis.

2.5 Lentiginous Naevus

This is a neglected entity which is usually grouped with lentigo simplex. It has also been called naevoid lentigo and naevus incipiens (STEWART et al. 1978). It appears to represent the progression of a lentigo into a junctional and then a compound naevus (ACKERMAN & RAGAZ 1984). They usually present clinically as deeply pigmented lesions on the trunk of adults between the ages of 20 and 40 years. Because of their deep pigmentation they often cause undue worry when found on medical practitioners and their relatives, and they are therefore removed. Their small size and regular margin contrast with that of malignant melanoma.

At the advancing edge there is a lentiginous proliferation of melanocytes, usually associated with some elongation of the rete. In the central area of the lesion there is junctional nest formation, and in some lesions intradermal nests are also present. There are usually melanin-containing macrophages in the dermis. They should not be confused with dysplastic naevi on histological examination. Similar lesions were reported recently by ENG (1983) as small junctional naevi.

2.6 Speckled Lentiginous Naevus

Speckled lentiginous naevus (STEWART et al. 1978) is the preferred term for a lesion composed of small dark hyperpigmented speckles, superimposed on a tan-brown macular background. They often have a zosteriform distribution (MATSUDO et al. 1973; SIMÕES 1981). They may be present at birth or appear in childhood.

On histological examination, the background pigmented area resembles lentigo simplex, while the speckled areas show the features of a lentiginous naevus with lentigo-like areas progressing to junctional and compound naevi (STEWART et al. 1978).

Similar cases have been reported under the term naevus spilus (ITO & HAMADA 1952; COHEN et al. 1970), but this name should be used in its original sense for a large light to dark brown plaque showing mild epidermal thickening and basal hyperpigmentation, without an increase in basal melanocytes or naevus-cell nest formation (STEWART et al. 1978).

The term zosteriform lentiginous naevus has also been used in a confusing way. It has been used synonymously with speckled lentiginous naevus (MATSUDO et al. 1973) and for lesions without a background macular pigmentation (PORT et al. 1978). These latter lesions are really clusters of lentiginous naevi, in a zosteriform distribution (RUTH et al. 1980). The term spotted grouped pigmented naevus has been used by the Japanese for lesions which are similar to the speckled lentiginous naevus and which have been present since birth. They have shown greater maturation on histological examination, with well-formed intradermal naevi often present (MORISHIMA et al. 1976; SATO et al. 1979). They included in this group lesions in which the naevus cell proliferation was localized around eccrine ducts and which were first described by MISHIMA (1973) as eccrine-centred naevi.

Finally, this entity of speckled lentiginous naevus should not be confused with cases of regional or segmental lentigines, which have recently been referred to as lentiginous mosaicism (FULK 1984).

2.7 PUVA Lentigo

Freckles (BLEEHEN 1978), and lentigines (FARBER et al. 1983; RHODES et al. 1983c) have been reported in patients treated with psoralens and ultraviolet A (PUVA), particularly in sun-protected sites. Some lesions are characterized by a lentiginous proliferation of relatively large, sometimes cytologically atypical melanocytes (RHODES et al. 1984). PUVA lentigines were found on the buttocks of 53% of psoriatic patients treated with PUVA in one series (RHODES et al. 1983c). Also of interest, is the development of melanoma in situ in two patients treated with PUVA (MARX et al. 1983).

3. Melanocytic Naevi

Descriptions of the histological features of junctional, compound and intradermal naevi can be found in standard textbooks of pathology, and will not be discussed in this review. Brief mention is made for completeness only. Interesting secondary changes are sometimes seen in naevi, and these will be considered in section 3.3.2. The following brief descriptions apply to acquired naevi. Congenital naevi will be considered in section 3.7.

3.1 Junctional Naevi

Junctional naevi are characterized by the presence of nests of naevus cells at the dermo-epidermal junction. These sometimes bulge into the underlying der-

mis. There is often some epidermal acanthosis and even bulbous epidermal rete ridges in some lesions. The small active junctional naevi reported recently by ENG (1983) in adolescents have some resemblance to the lesions described elsewhere in this review as lentiginous naevi.

3.2 Compound Naevi

Compound naevi have both junctional nests and an intradermal component of naevus cells. Whereas cells in the upper dermis are usually cuboidal with cytoplasmic melanin granules, deeper cells are usually smaller and contain less melanin. The cells usually lie in nests. The overlying epidermis may be flat, show some acanthosis, or have a seborrhoeic-keratosis like configuration (BENTLEY-PHILLIPS & MARKS 1976).

3.3 Intradermal Naevi

Intradermal naevi are dome-shaped, nodular or polypoid lesions which are lightly pigmented or flesh-coloured. On microscopic examination, naevus cells are confined to the dermis where they are arranged in nests and cords. Multinucleated naevus cells are often seen. The overlying epidermis may show any of the changes listed for compound naevi. In the deeper parts of the tumour the naevus cells may assume a neuroid appearance, with spindle-shaped cells and structures resembling Meissner's tactile bodies. These age-related changes are discussed further in the following section.

3.3.1 Age-related Changes

With advancing age, there is a progressive decrease in the number of naevi in a person. The average number of naevi in young adults is from 15 to 40, but this decreases markedly over the age of 50 years (STEGMAIER & BECKER 1960; NICHOLLS 1973).

MAIZE and FOSTER (1979) have reviewed the changes that occur in naevocellular naevi with increasing age of the patient. They found decreased junctional proliferation and total cellularity with age and replacement of naevus cells within the dermis by collagen, fat, elastin and ground substance. There was no tendency for naevi to assume a polypoid configuration, which would argue against the hypothesis that naevi transform into tags, which eventually fall off (STEGMAIER 1959). They concluded that the formation of cylindrical neuroid structures represented the end stage of differentiation of naevus cells (MAIZE & FOSTER 1979).

3.3.2 Secondary Changes in Naevi

Several interesting secondary changes can be found in naevocellular naevi. These were considered in some detail in my review article published in 1982, and they will be considered here only briefly, for completeness (WEEDON 1982a).

Amyloid. Small deposits of amyloid are a rare incidental finding in the upper dermis (MACDONALD & BLACK 1980).

Bone. Naevi containing spicules of bone are called osteonaevi of Nanta (BURGDORF & NASEMANN 1977). These naevi are usually found on the face (RUPEC & HUCK 1976). The metaplastic bone formation may follow infection or trauma. Bone has also been reported in blue naevi (BARAN & CIVATTE 1960) and Spitz naevi (CIVATTE et al. 1972; PACOT et al. 1972).

Eczematous change. This refers to the presence of a subacute spongiotic change involving the epidermis overlying and adjacent to a naevus (WEEDON & FARNSWORTH 1984). There is an underlying superficial perivascular inflammatory infiltrate, but there is no evidence of regression of the lesion. Clinically, an eczematous halo develops around one or more pigmented naevi. This clears over a period of three to ten months. The aetiology of this secondary change is unknown (MEYERSON 1971; KRIVANEK et al. 1977).

Elastosis. Coarse fibres, with the features of solar elastosis can sometimes be seen in naevi removed from sun-damaged skin (WEEDON 1982a). Elastic fibre hyperplasia may also be found in naevi (MEHREGAN & STARICCO 1962).

Folliculitis and cysts. Cystic dilatation of a hair follicle (HABER 1952), folliculitis (CANIZARES 1968), abscess formation in the dermis (SAUNDERS 1957) or epidermal cyst formation (FREEMAN & KNOX 1962) may be found as a secondary change towards the deep edge of a naevus. These changes may produce sudden enlargement, irritation or focal colour change in a naevus. Rupture of an inflamed follicle or associated epidermal cyst usually produces a focal foreign body giant cell reaction.

Artifacts. The injection of local anaesthetic into a naevus will result in the separation of naevus cells into parallel rows (SAGEBIEL 1972). The formation of clefts and spaces resembling vascular or lymphatic spaces, associated with shrinkage and separation of naevus cells has been regarded as a processing artifact (SAGEBIEL 1972). However the author has seen a case with abundant mucopolysaccharide within these pseudoangiomatous spaces, suggesting that the change, at least in some cases, may not be an artifact.

Cockarde Naevus. This term has been applied to naevi which develop a peripheral pigmented halo with an intervening narrow zone that is non-pigmented (MEHREGAN & KING 1972; HAPPLE 1974; WARIN 1976; JAMES & WELLS 1980). The central naevus is of compound or junctional type, while the peripheral halo is usually composed of junctional nests. The intervening non-pigmented zone is usually devoid of naevus cells.

Associated other tumours. Naevocellular naevi have been found in association with trichoepitheliomas (RAHBARI & MEHREGAN 1975), syringomas (SCHELLANDER et al. 1974), basal cell carcinomas (SIGAL & SANDERS 1967), abortive hair follicle formation (MEHREGAN & COSKEY 1972) and sweat duct proliferation (unpublished observations). Naevi have also been described in which the naevus cell proliferation was closely related to eccrine sweat ducts (MISHIMA 1973; MORISHIMA et al. 1976).

Pseudomelanoma change and activation of naevi. The term 'pseudomelanoma' was coined by KORNBERG and ACKERMAN (1975) for the worrisome changes seen in recurrent naevi, following their partial surgical removal by ablation.

These recurrent naevi have sharp circumscription of epidermal melanocytes with no lateral extension. There is some upward epidermal spread. The atypical melanocytes are confined to the epidermis, while there are mature naevus cells often seen in the dermal component. There is usually fibrosis in the papillary dermis, resulting from the previous procedure.

During pregnancy, naevocellular naevi may become 'reactivated' with the formation of renewed junctional activity (WEEDON 1982a). Similar changes have been described in naevi removed from patients with a malignant melanoma (TUCKER et al. 1980) but these changes were described before the recognition of sporadic cases of the dysplastic naevus syndrome. The changes described include marked junctional activity with lack of lateral margination, inflammation in the upper dermis, increased pigment formation and some mitotic figures (TUCKER et al. 1980).

Other changes. Other very rare changes reported include the presence of psammoma bodies (WEITZNER 1968), the development of alopecia around naevi in the scalp (YESUDIAN & THAMBIAH 1976) and the presence of focal acantholytic dyskeratosis in the adjacent epidermis (WEEDON 1982a). The author has seen massive epidermal necrosis, and another case with numerous solitary 'dyskeratotic' cells overlying an intradermal naevus. There was no apparent aetiology for these changes. Rarely, a lesion of molluscum contagiosum may overlie a naevus (MARKS & WHITE 1980).

3.4 Balloon Cell Naevus

The balloon cell naevus is a rare lesion, clinically indistinguishable from ordinary melanocytic naevi, which tends to occur in the first three decades of life (WILSON JONES & SANDERSON 1963). To qualify as a balloon cell naevus, the lesion should contain a preponderance (over 50%) of foam cells (SCHRADER & HELWIG 1967). Focal aggregations of balloon cells can be found in up to 2% of naevi, but lesions composed entirely of these cells are extremely rare (GOETTE & DOTY 1978). In addition to clear cells with a single basophilic nucleus, multinucleated balloon cells are commonly present. Sometimes transitions between naevus cells and balloon cells can be seen.

Ultrastructural studies have shown that balloon cells are formed by the progressive vacuolization of melanocytes and naevus cells brought about by the enlargement and eventual destruction of melanosomes (HASHIMOTO & BALE 1972; OKUN et al. 1974b).

Balloon cell melanomas have also been reported (GARDNER & VASQUEZ 1970; FRIEDMAN et al. 1982; HORTON & MACDONALD 1983).

3.5 Halo Naevus

The development of a depigmented halo around a naevus is most often an idiopathic phenomenon which precedes the lymphocytic destruction of the naevus cells and the clinical regression of the lesion. This change may involve

one or several naevi. Halo formation has been described in patients with vitiligo and also following severe sunburn (WHIMSTER 1974). After regression of the central naevus, the depigmented halo may repigment or may remain permanently leukodermatous. Occasionally there is erythema in the depigmented halo in the early stages (BERMAN & HERSZENSON 1981). Circulating antibodies which react against melanoma cells have been found in a high proportion of cases (COPEMAN et al. 1973).

On histological examination, there is usually a dense lymphocytic infiltrate within the tumour associated with the destruction of naevus cells. There are some macrophages in the infiltrate. Skin from the depigmented halo appears normal except for the absence of melanin in the epidermis and the replacement of epidermal melanocytes by Langerhans' cells. There may be dilated vessels in the early stages with an erythematous halo. There have been cases of halo naevus reported in which the tumour itself was devoid of inflammatory cells (BERGER & VOORHEES 1971; BROWNSTEIN et al. 1977; GAUTHIER et al. 1978). No regression would occur in such cases. It should also be noted that the lymphocytic destruction of a naevus can take place without the development of a clinical halo. Ultrastructural studies have shown non-specific injury changes in some naevus cells, with destruction of other cells (HASHIMOTO 1974; GAUTHIER et al. 1975). It is possible that cell death takes place by apoptosis as well as necrosis, but this remains to be confirmed.

3.6 Spitz Naevus

The eponymous designation 'Spitz nevus' is the preferred term for the entity which is also known as 'spindle cell naevus', 'epithelioid cell naevus', 'naevus of large spindle and/or epithelioid cells' and 'benign juvenile melanoma'. Such a term gives recognition to Sophie Spitz who published for the first time criteria for the diagnosis of a specific lesion of childhood, which despite some histological resemblance to malignant melanoma was known to behave in a benign fashion (SPITZ 1948). There have been many series of cases published since that time, the most comprehensive literature review being contained in the report of 40 cases by KOPF and ANDRADE (1966). Other series include those by MCWHORTER and WOOLNER (1954) – 11 cases, DUPERRAT and DUFOURMENTEL (1959) – 40 cases, KERNEN and ACKERMAN (1960) – 27 cases, GARTMANN (1962) – 67 cases, ALLEN (1963) – 308 cases, JAKUBOWICZ (1965) – 50 cases, COSKEY & MEHREGAN (1973) – 202 cases, WEEDON and LITTLE (1977) – 211 cases, and PANIAGO-PEREIRA et al. (1978) – 200 cases. A further review of this subject has been completed recently by the author (WEEDON 1984).

3.6.1 Clinical Features

Spitz naevi account for approximately 0.5 (WEEDON 1984) to 1 per cent (KOPF & ANDRADE 1966) of surgically excised naevi of children. The annual incidence, in our material was 1.4 cases per 100,000 of the population (WEEDON & LITTLE 1977).

Table 1. Spitz naevi – Diagnostic criteria

Major Criteria	*Minor criteria*
(a) Cell type	(a) Junctional cleavage
(b) Symmetry	(b) Oedema and telangiectasia
(c) Maturation in depth	(c) Giant naevus cells
(d) Lack of single cell upward epidermal spread	(d) Absence of nuclear pleomorphism
(e) Coalescent eosinophilic globules	(e) Perivascular inflammation
	(f) Deep outlying cells
	(g) Lack of deep mitoses

The typical Spitz naevus is a pink or flesh-coloured papule or nodule, arising on the face, trunk or extremities, particularly the lower, of children or young adults. Pigmented variants and lesions in other sites or in older individuals are not unknown. Most lesions are less than 1.2 mm in diameter. Rapid clinical growth is sometimes documented. The natural history of untreated lesions remains controversial. Multiple Spitz naevi have been described either in the form of clustered (agminate) or disseminated lesions (BROWNSTEIN 1972; WALLACE 1974; CAPETANAKIS 1975; WEIMAR & ZUEHLKE 1978; LANCER et al. 1983). Agminate lesions have a predilection for the cheeks (GOULD & BLEEHEN 1980), and may follow sunburn (KRAKOWSKI et al. 1981).

3.6.2 Histological Features

The majority of Spitz naevi are compound in type although approximately 5 to 10% are junctional and 20% are intradermal lesions. No single histological feature is diagnostic, but more weight can be placed on some changes than on others (PANIAGO-PEREIRA et al. 1978). These diagnostic features may be grouped into major and minor critera (Table 1).

Cell type. Two types of Spitz naevi are found. In one form, spindle cells, sometimes quite plump, are present, while in the other type there are epithelioid cells. The diagnosis of Spitz naevus should not be considered unless the cell type is characteristic.

Symmetry. On scanning magnification, Spitz naevi are usually symmetrical, in contrast to melanoma where epidermal melanocytic activity may extend unevenly on either side of the dermal component.

Maturation in depth. This refers to the tendency of cells in the deeper parts of the tumour to become smaller, like the cells in an ordinary naevus. This change is found in less than 25% of cases, but it may be a helpful feature in borderline lesions (WEEDON & LITTLE 1977).

Upward epidermal spread. Single cell upward epidermal spread is uncommon in the Spitz naevus, although sometimes small clumps of three or more cells may be seen within the epidermis.

Eosinophilic bodies. Single or coalescent eosinophilic globules may be found at the dermo-epidermal junction in Spitz naevi, particularly if multiple step sections are examined (KAMINO et al. 1979; ARBUCKLE & WEEDON 1982). Single