Review paper

Pitfalls in the diagnosis of melanocytic tumours: is immunohistochemistry helpful?

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The diagnosis and differential diagnosis of benign, intermediate and malignant melanocytic neoplasms remain a recurring and clinically important challenge in dermatopathology. The difficulty is not limited to rare melanoma variants, it also includes non-melanocytic lesions that simulate melanocytic tumours, benign melanocytic proliferations with worrisome architectural or cytological features, and malignant melanomas with deceptively bland morphology. In such settings, immunohistochemistry is not a substitute for careful histopathological assessment, but it may provide decisive ancillary information when applied as a targeted panel and interpreted in the appropriate morphological and clinical context. This review summarises selected diagnostic pitfalls in melanocytic pathology and highlights the contribution, limitations and practical value of established and newer immunohistochemical markers.

Key words: naevus, melanocytoma, melanoma, immunohistochemistry, dermatopathology, diagnostic pitfalls.

Introduction

Malignant melanoma, its variants and its numerous mimics represent one of the most demanding areas of surgical pathology. The problem has become increasingly relevant because melanoma is being diagnosed with greater frequency in many countries, while the spectrum of recognised melanocytic lesions has expanded to include benign, atypical and intermediate categories with overlapping morphology. For the practising pathologist, the consequences of diagnostic error are substantial: a benign lesion recognised as melanoma may expose a patient to unnecessary treatment and anxiety, whereas an inconspicuous melanoma interpreted as a naevus may delay appropriate management.

This diagnostic field is further complicated by lesions whose morphology appears unfamiliar, incomplete or discordant with the clinical impression. Some benign tumours display alarming features, including increased cellularity, pigmentation, mitotic activity or intraepidermal spread. Conversely, certain melanomas show a deceptively orderly nested or nevoid pattern and may lack overt cytological atypia in routine sections. A reproducible diagnosis therefore depends on the integration of architectural features, cytomorphology, growth pattern, clinical information, and ancillary studies.

Immunohistochemistry has a central role in this integrated approach. Conventional melanocytic markers such as S100, SOX10, Melan-A and HMB45 help confirm lineage and highlight growth patterns, whereas markers including PRAME, p16, BAP1, PRKAR1A, 5-hmC, Ki-67 and pancytokeratin may refine the differential diagnosis in selected scenarios. None of these antibodies should be interpreted in isolation. Their value lies in the pattern of staining, the distribution of positive cells, the relationship to the dermal and epidermal components, and the concordance with the morphology. Table I summarises selected immunohistochemical patterns that may be useful in the differential diagnosis of melanocytic tumours and selected mimics.

Xanthogranuloma

Histiocytic proliferations may closely mimic melanocytic neoplasms, particularly Spitz tumours, and in some instances malignant melanoma. The distinction may be especially difficult in mononuclear xanthogranuloma, where the lesional cells lack the typical foamy cytoplasm and multinucleated giant cells that usually suggest a histiocytic process. When such lesions show increased cellularity or proliferative activity, they may be misinterpreted as an atypical Spitz tumour or even melanoma. A further pitfall is the occasional focal expression of S100 protein, which may falsely support melanocytic differentiation if the antibody is used in isolation. A broader immunohistochemical panel is therefore essential. Xanthogranuloma typically shows CD68 expression and lacks Melan-A and HMB45 expression, thereby supporting histiocytic rather than melanocytic lineage [1].

Melanocytic matricoma

Melanocytic matricoma is a rare tumour seen predominantly in elderly patients, with a reported male predominance. Clinically, it usually presents as a small dark or black nodule on sun-exposed skin and may raise suspicion for pigmented basal cell carcinoma, primary melanoma, or metastatic melanoma. Histologically, the lesion is well circumscribed and biphasic, consisting of an epithelial matrical component with matrical, supramatrical and shadow cells, admixed with pigmented dendritic melanocytes. Mild to moderate pleomorphism and mitotic activity may be present and may contribute to the diagnostic concerns. Immunohistochemistry is helpful because the melanocytic component expresses S100 protein, Melan-A and HMB45, whereas the epithelial component is highlighted by pancytokeratin, β-catenin and LEF1. Recognition of this dual differentiation prevents overdiagnosis as melanoma [2].

Pigmented epithelioid melanocytoma

Pigmented epithelioid melanocytoma (PEM) belongs to the expanding group of melanocytomas, a category of melanocytic tumours characterised by intermediate biological potential and different genetic alterations. Pigmented epithelioid melanocytoma occurs on the extremities, trunk, and head and neck region, most often in younger patients. Regional lymph node involvement may be encountered, but systemic metastasis and progressive disease are generally not observed. Morphologically, PEM is a cellular dermal neoplasm composed of heavily pigmented epithelioid melanocytes with enlarged vesicular nuclei, often showing a perinuclear halo that produces a so-called "fried-egg" appearance. Cytological atypia, mitotic figures and even necrosis may be present, and these features may create a strong impression of dermal or metastatic melanoma. Many cases show *PRKAR1A* inactivation with loss of nuclear PRKAR1A expression, an immunohistochemical finding that can support the diagnosis when interpreted in the appropriate setting [3, 4] (Figure 1).

BAP1-inactivated melanocytoma

BAP1-inactivated melanocytoma is a distinct melanocytic neoplasm defined by bi-allelic *BAP1* inactivation and corresponding loss of nuclear BAP1 expression. These lesions tend to occur in young patients and may be solitary or multiple. Clinically, they often appear as skin-coloured dermal papules or nodules and may be mistaken for conventional naevi or spitzoid lesions. Histologically, they typically show a peripheral dermal naevus component and a more cellular central component composed of enlarged spitzoid melanocytic cells. The enlarged cells may demonstrate increased atypia and proliferative activity, raising concern for melanoma. Immunohistochemical loss of nuclear BAP1 expression in the enlarged component, with retention in internal controls, is a useful diagnostic clue and helps place the lesion within the spectrum of *BAP1*-inactivated melanocytic tumours [5] (Figure 2).

Desmoplastic atypical spitzoid melanocytic tumour

Desmoplastic Spitz naevus is a variant of Spitz naevus seen mainly in adults. It is largely or entirely intradermal and is characterised by conspicuous collagen deposition within the lesion. The term desmoplastic atypical spitzoid melanocytic tumour is applied to lesions that are typically larger and show more worrisome architectural features, including an intraepithelial component composed of enlarged melanocytic cells. These cells may be distributed diffusely within the epidermis rather than arranged in well-formed nests, thereby simulating malignant melanoma. In this setting, the overall symmetry of the lesion, the circumscription of the dermal component and the low proliferative activity of the dermal tumour cells are important clues favouring a spitzoid lesion rather than melanoma. Immunohistochemistry is most useful when it demonstrates a low dermal Ki-67 index and a pattern that remains concordant with the morphology.

Nevoid melanoma

Nevoid melanoma, especially the small-cell variant, is among the most treacherous melanoma mimics because it may closely resemble a benign naevus

Table I. Selected immunohistochemical patterns useful in the differential diagnosis of melanocytic tumours and selected mimics

Lesion/ differential pitfall	S100/ SOX10	Melan-A/HMB45	PRAME	p16/Ki-67/5-hmC	BRAF V600E, PRKAR1A, BAP1	CD68/pan-CK/ other key tests	Practical diagnostic comment	References
Benign naevi	+/+	Melan-A +; HMB45 usually stratified with maturation gradient	Usually negative or only focal/weak; diffuse nuclear staining should prompt caution	p16 retained; Ki-67 low and usually superficial; 5-hmC retained	BRAF V600E variable in acquired naevi; not a malignancy marker by itself	CD68 only in melanophages; pan-CK negative	A retained maturation pattern is more informative than positivity for lineage markers alone	11–13
Low-CSD melanoma/ superficial spreading melanoma	+/+	Melan-A +; HMB45 often unstratified or dermal	Frequently diffuse nuclear positive	p16 loss variable; Ki-67 increased including a dermal component; 5-hmC reduced/lost	BRAF V600E relatively common but variable	pan-CK negative	Use PRAME with morphology; PRAME-negative melanomas occur	11–13
High-CSD melanoma/ lentigo maligna melanoma	+/+	Melan-A +; HMB45 variable, often highlights a junctional/invasive component	Frequently diffuse nuclear positive, but variable on a case-by-case basis	p16 loss variable; Ki-67 increased; 5-hmC reduced/lost	BRAF V600E less typical than in low-CSD melanoma; other genomic alterations may be relevant	pan-CK negative	Melan-A/SOX10 can overestimate melanocyte density in chronically sun-damaged skin; correlate with H&E and distribution	11, 13
Acral lentiginous melanoma	+/+	Melan-A +; HMB45 +/variable	Frequently diffuse nuclear positive in many cases	p16 loss variable; Ki-67 increased; 5-hmC reduced/lost	BRAF V600E usually less frequent; KIT/ CCND1/CDK4 changes may be more relevant but are not captured by this basic IHC table	pan-CK negative	Look for linear basal proliferation, field cells and anisodendrocytosis; IHC helps map extent but does not replace architecture	11, 13
Nevoid melanoma	+/+	Melan-A +; HMB45 may be dermal/ unstratified but can be deceptively reduced in pseudo-maturation	Often positive, but not uniformly	p16 loss supportive; Ki-67 often increased; 5-hmC reduced/lost	BRAF V600E variable	pan-CK negative	The most helpful pattern is discordance between bland nevoid cytology and abnormal maturation/proliferation profile	6, 11–13
Desmoplastic melanoma	+/+	Melan-A often negative; HMB45 usually negative or very limited	Variable; diffuse PRAME is much less frequent than in non-desmoplastic melanoma	p16 and Ki-67 variable; 5-hmC may be reduced/lost	BRAF V600E uncommon; NF1 alterations are more typical but not assessed in this table	pan-CK negative; p75/NGFR and neurotropic pattern may be supportive in context	SOX10 and S100 are key lineage markers; a negative Melan-A/ HMB45 panel does not exclude desmoplastic melanoma	11, 14

Table I. Selected immunohistochemical patterns useful in the differential diagnosis of melanocytic tumours and selected mimics (cont.)

Lesion/ differential pitfall	S100/ SOX10	Melan-A/HMB45	PRAME	p16/Ki-67/5-hmC	BRAF V600E, PRKAR1A, BAP1	CD68/pan-CK/ other key tests	Practical diagnostic comment	References
Spitz melanoma/ malignant Spitz tumour	+/+	Melan-A +; HMB45 may show deep/diffuse staining without maturation	Variable; PRAME is not a stand-alone discriminator in spitzoid lesions	p16 loss and high Ki-67 support malignancy but are not specific; 5-hmC may be reduced	BRAF V600E usually not typical for classic Spitz pathway; kinase fusions/HRAS/ MAP3K8 may require molecular testing	pan-CK negative	Use IHC as part of a tiered assessment; a molecular workup is often required for difficult spitzoid lesions	11–13
Nested melanoma	+/+	Melan-A +; HMB45 often unstratified/diffuse	Often positive, but data are still limited	p16 loss and increased Ki-67 support melanoma; 5-hmC may be reduced	BRAF V600E variable; genomic aberrations support malignant nature	A thin pan-CK-positive epithelial rim may surround nests, but the melanocytic tumour cells are pan-CK negative	Large confluent nests can mimic naevi; asymmetry, cytologic atypia and genomic aberrations are key	7, 8, 11
Pigmented epithelioid melanocytoma	+/+	Melan-A +; HMB45 +	Usually negative/ variable; PRAME is not defining	Ki-67 usually low-to-intermediate; p16 variable; 5-hmC data not definitive	PRKAR1A loss in a subset/classic PEM; BAP1 retained	CD68 may highlight melanophages; pan-CK negative	May metastasise to lymph nodes but usually lacks aggressive systemic behaviour; PRKAR1A loss supports, but does not absolutely define, the diagnosis	3, 4, 15
BAP1-inactivated melanocytoma/ BAP1-inactivated Spitz tumour	+/+	Melan-A +; HMB45 variable	Usually negative/ variable; not defining	Ki-67 usually low-to-intermediate; p16 variable	Loss of nuclear BAP1 in epithelioid/spitzoid component; BRAF V600E often present in this subset	pan-CK negative	Internal positive controls for BAP1 are mandatory; loss should be interpreted in the appropriate combined naevus /spitzoid context	5
Desmoplastic atypical spitzoid melanocytic tumour	+/+	Melan-A +; HMB45 variable, may be reduced in desmoplastic areas	Variable/usually not decisive	Ki-67 generally low-to-intermediate; p16 usually retained or variable; 5-hmC not decisive	BAP1/PRKAR1A loss not expected as defining features	pan-CK negative	Symmetry and low dermal proliferation favour an atypical Spitz-type lesion rather than desmoplastic melanoma	12, 13

Table I. Selected immunohistochemical patterns useful in the differential diagnosis of melanocytic tumours and selected mimics (cont.)

Lesion/ differential pitfall	S100/ SOX10	Melan-A/HMB45	PRAME	p16/Ki-67/5-hmC	BRAF V600E, PRKAR1A, BAP1	CD68/pan-CK/ other key tests	Practical diagnostic comment	References
Dermal clear cell sarcoma	+/+	Melan-A and HMB45 positive or variable	Variable; not sufficient for distinction from melanoma	p16/Ki-67/5-hmC not reliable discriminators	BAP1/PRKAR1A not defining	Molecular confirmation of EWSR1::ATF1 or EWSR1::CREB1 is decisive; pan-CK negative	IHC overlaps extensively with melanoma; a primary dermal/deep lesion lacking an epidermal melanoma component requires molecular exclusion of clear cell sarcoma	9
Cutaneous melanocytic tumour with CRTC1::TRIM11 fusion	+/+	Melan-A and HMB45 positive or variable	Variable/ limited data	Not established as a discriminator	BAP1/PRKAR1A not defining	CRTC1::TRIM11 fusion confirms the entity; pan-CK negative	Can resemble clear cell sarcoma or dermal melanoma; molecular testing is recommended in compatible dermal tumours	10
Melanocytic matricoma	Melanocytes S100/ SOX10 +; epithelial component SOX10 negative	Melanocytic component Melan-A/HMB45 +	Not established; not useful as a primary discriminator	Proliferation may be present in a matrical component; p16/5-hmC not defining	BRAF/BAP1/ PRKAR1A not defining	pan-CK, β-catenin and LEF1 highlight the matrical epithelial component	This is a biphasic adnexal tumour; marker interpretation must be component-specific	2
Xanthogranuloma/ mononuclear xanthogranuloma	S100 negative or focal +; SOX10 negative	Melan-A negative; HMB45 negative	Negative	p16/Ki-67/5-hmC not useful for melanocytic distinction	BRAF/BAP1/ PRKAR1A not applicable	CD68/CD163 positive; Factor XIIIa often positive; pan-CK negative	Focal S100 can be misleading; use a histiocytic panel and melanocytic markers together	*1*

+ – positive, 5-hmC – 5-hydroxymethylcytosine, CSD – cumulative sun damage, diffuse PRAME – broad nuclear staining used in the main PRAME literature, PEM – pigmented epithelioid melanocytoma, retained/loss – interpreted only when internal positive controls are adequate, variable – reported/expected variability depending on the tumour subtype, sampling and antibody clone

This table is not intended as a stand-alone diagnostic algorithm. Immunohistochemistry should be interpreted in the context of architecture, cytomorphology, clinical setting, anatomic site, age, and – where appropriate – molecular findings. Staining intensity, distribution, internal controls and laboratory-specific validation remain essential.

Laboratories should apply their own validated scoring thresholds. p21 was not retained in the revised table because its diagnostic use is insufficiently standardised for a compact, practical matrix.

Practical use: For routine diagnostic work, the most useful first-line approach is often: (1) melanocytic lineage confirmation with SOX10/S100 and/or Melan-A; (2) assessment of maturation/proliferation with HMB45, Ki-67 and p16; (3) PRAME as an adjunctive malignancy marker; (4) subtype-directed tests such as BAP1, PRKAR1A, β-catenin/LEF1, CD68/CD163 or molecular testing depending on the differential diagnosis.

at low and intermediate magnification. Despite this bland appearance, it is associated with an adverse clinical course. The lesions are composed of small, relatively monomorphic melanocytic cells with limited cytological atypia and may be entirely dermal or show verrucous or lentiginous architecture. Clues to malignancy include a size greater than expected for an ordinary naevus, lack of maturation, subtle nuclear enlargement, nucleolar prominence and an increased number of dermal mitoses. Immunohistochemical findings that may support the diagnosis include persistent dermal HMB45 expression, loss of p16 and 5-hydroxymethylcytosine expression, increased p21 expression and PRAME positivity. These findings should be interpreted in combination, as no single marker is sufficient to establish the diagnosis [6] (Figure 3).

Nested malignant melanoma

Nested melanoma is characterised by a predominantly nested architecture, a feature that usually favours a benign naevus. In nested melanoma, however, the nests are often large, confluent and distributed over an extensive horizontal component. The lesional cells show cytological atypia, and the overall architecture is inconsistent with a conventional naevus. A helpful microscopic clue is the presence of a narrow rim of epithelial cells at the base of the melanocytic nests, which can be highlighted by pancytokeratin immunohistochemistry. Immunostains such as HMB45 and PRAME may further support malignancy when they show an unstratified or diffuse pattern. The presence of multiple genomic aberrations in analysed cases provides additional evidence that these lesions represent a malignant melanoma variant rather than an unusual naevus [7, 8] (Figure 4).

Acral lentiginous melanoma

Acral naevi may show lentiginous growth and conspicuous intraepidermal ascent of melanocytes, features that may mimic acral lentiginous melanoma. The distinction is clinically important because acral melanoma may be subtle in early stages and may be under-recognised. In acral lentiginous melanoma, atypical melanocytes tend to be arranged singly and linearly along the basal layer of the epidermis,

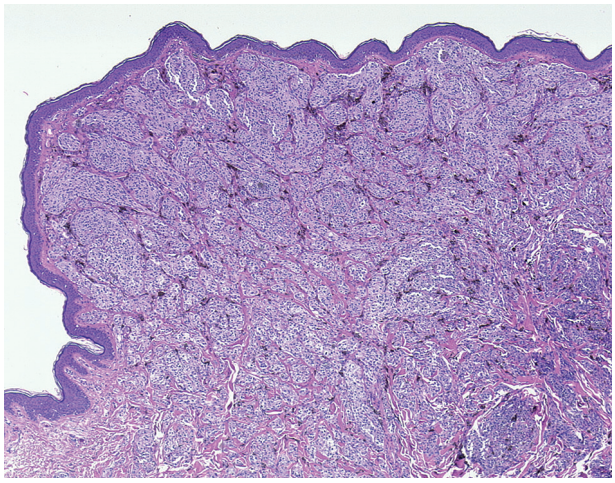

Figure 1. Pigmented epithelioid melanocytoma composed of heavily pigmented melanocytic cells

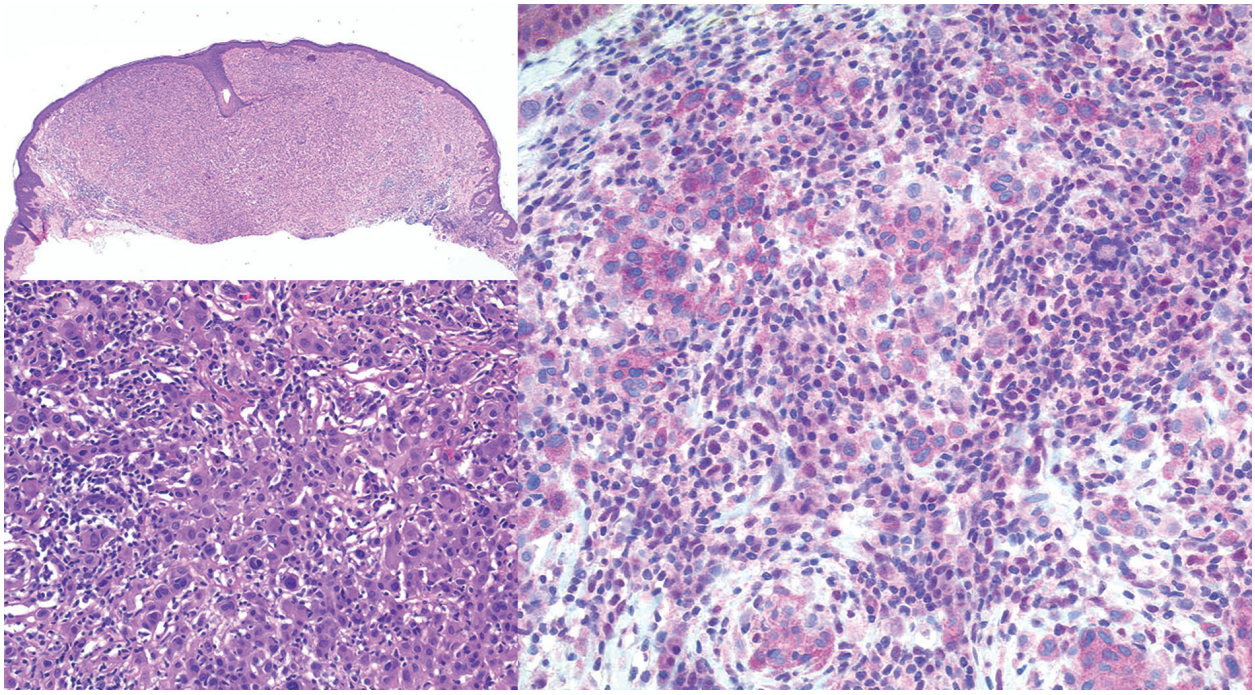

Figure 2. BAP1-inactivated melanocytoma with peripheral naevus cells and a centrally located component composed of enlarged atypical spitzoid cells (left). The enlarged cells show loss of nuclear BAP1 expression (right)

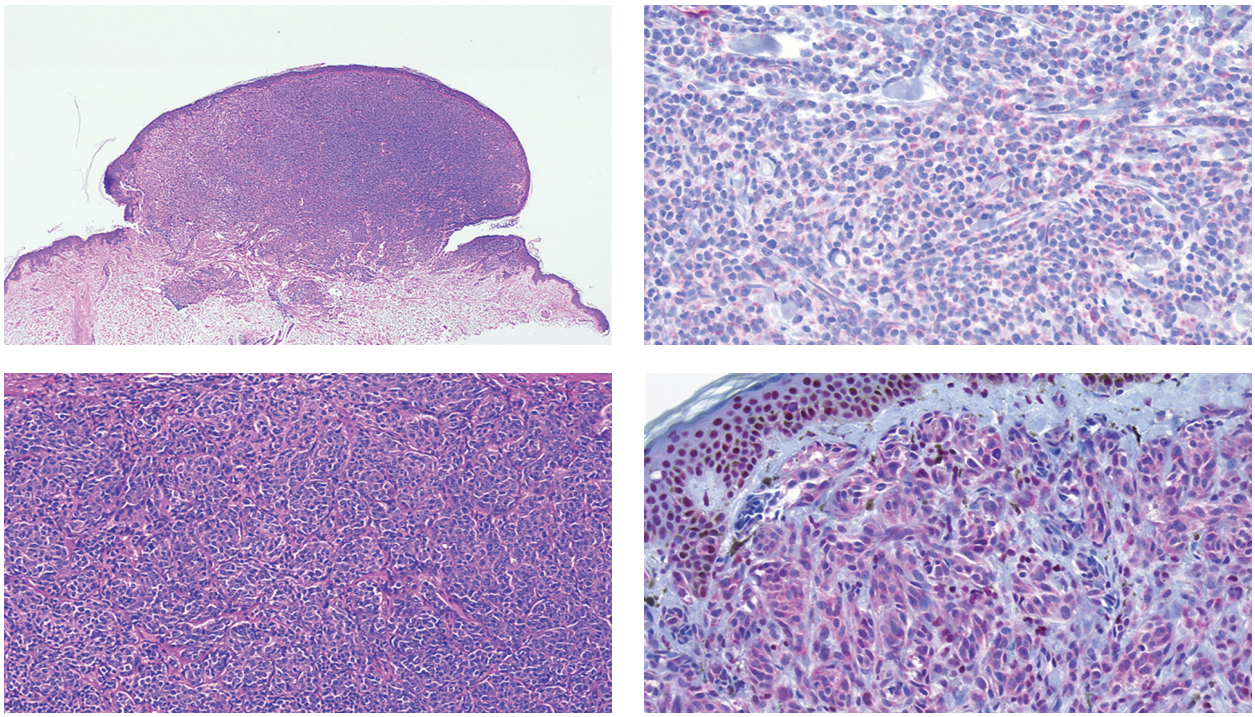

Figure 3. Nevoid melanoma composed of relatively uniform melanocytic cells with enlarged nuclei (left). Tumour cells show loss of p16 expression (upper right) and loss of 5-hydroxymethylcytosine expression (lower right)

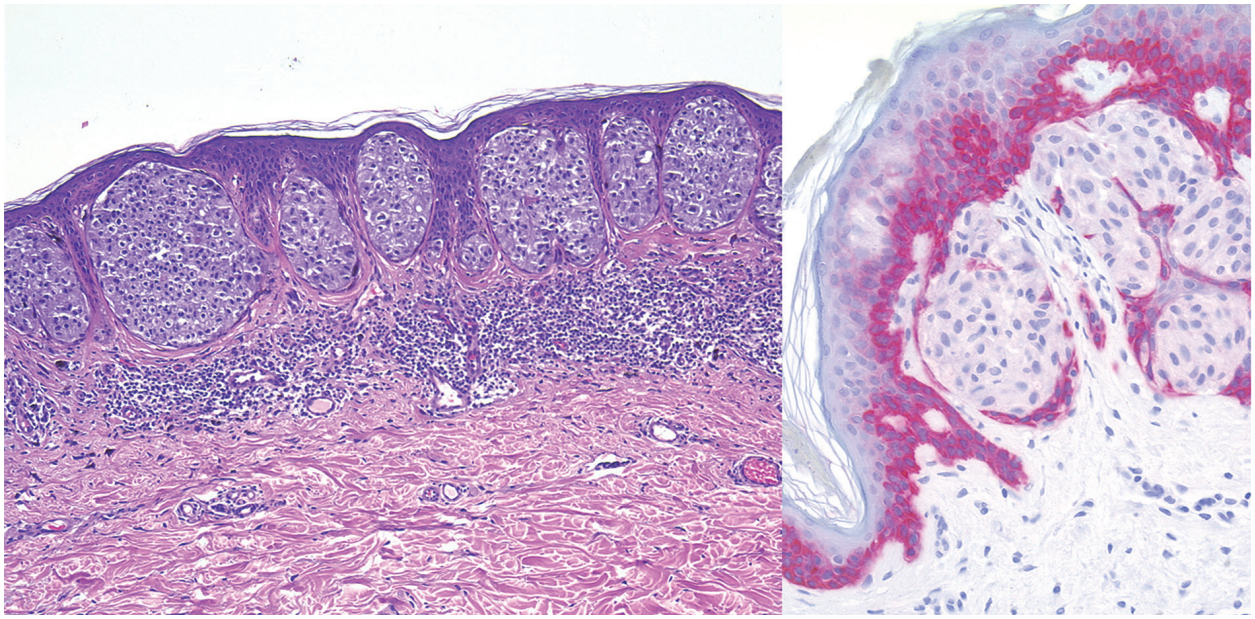

Figure 4. Nested melanoma composed of enlarged atypical melanocytic cells arranged in large confluent nests (left). A narrow rim of pancytokeratin-positive epithelial cells surrounds nests of melanocytic cells (right)

producing a pattern that may resemble mycosis fungoides. The tumour cells are usually medium-sized, dendritic or epithelioid, and show variable nuclear enlargement, pleomorphism and hyperchromasia. Prominent anisodendrocytosis is often present. So-called field cells, represented by isolated atypical melanocytes at the periphery of the lesion and sometimes far from the main tumour, are another important diagnostic clue. Melan-A or SOX10 immunohistochemistry may be useful to delineate the extent and distribution of the atypical melanocytic proliferation (Figure 5).

Dermal clear cell sarcoma

Clear cell sarcoma may rarely present in a superficial dermal location, and cases with an intraepithelial component have been described. This unusual presentation can place the lesion in the differential diagnosis for dermal melanoma or metastatic melanoma. Histo-

logically, clear cell sarcoma is composed of nests and fascicles of atypical melanocytic cells and may contain multinucleated wreath-like giant cells. A characteristic band-like hyalinization of the stroma can provide an additional clue. Because the immunophenotype overlaps substantially with melanoma, molecular confirmation is particularly important; demonstration of characteristic gene fusions supports a diagnosis of clear cell sarcoma and helps distinguish it from melanoma [9] (Figure 6). Conversely, cutaneous melanocytic tumour with *CRTC1::TRIM11* fusion may resemble clear cell sarcoma morphologically, but appears to have a more favourable prognosis, underscoring the value of integrating morphology, immunohistochemistry and molecular findings in difficult cases [10].

Conclusions

The lesions discussed in this review illustrate a recurring principle in melanocytic pathology: the most difficult cases are rarely solved by morphology or immunohistochemistry alone. Immunohistochemistry is most informative when it is used to test a specific diagnostic hypothesis and when the result is assessed as a staining pattern within the lesion rather than as a simple positive or negative reaction. In practice, the greatest diagnostic value is achieved by combining conventional melanocytic markers with markers of proliferation, tumour suppressor loss, lineage exclusion and, where appropriate, molecularly linked surrogate markers. This approach reduces the risk of both overdiagnosis and underdiagnosis and supports a more reproducible classification of benign, intermediate and malignant melanocytic tumours.

Disclosures

1. Institutional review board statement: Not applicable.
2. Assistance with the article: None.
3. Financial support and sponsorship: None.
4. Conflicts of interest: None.

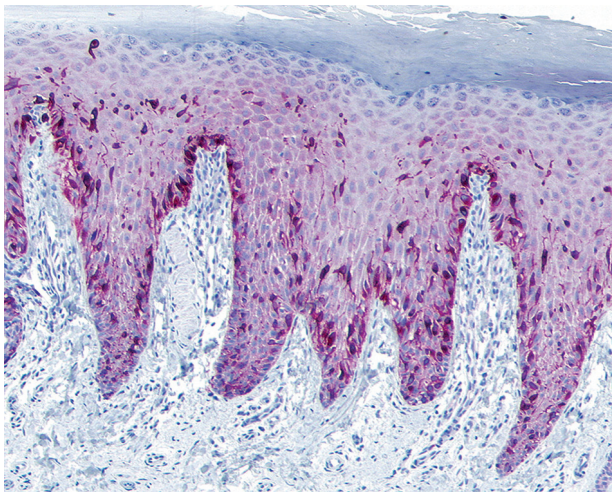

Figure 5. Melan-A staining in acral lentiginous melanoma showing irregular distribution of enlarged tumour cells and prominent anisodendrocytosis

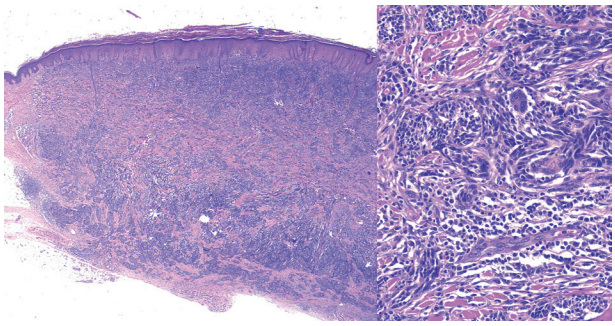

Figure 6. Superficial clear cell sarcoma arising in a 19-year-old female patient (left). Multinucleated wreath-like giant cells are present (right)

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