Clinics in Dermatology (2025) **43**, 315–322

Clinics in Dermatology

Melanoma *in situ* and low-risk pT1a melanoma: Need for new diagnostic terminology

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Abstract The incidence of melanoma has risen rapidly, at least until recently, while the mortality rate has changed only a little, a phenomenon suggestive of overdiagnosis, which can be defined as the diagnosis as "melanoma" of a lesion that would not have had the competence to cause death or symptoms even if it had not been excised. Overdiagnosis has been attributed to efforts at early diagnosis ("overdetection") and to changes in criteria resulting in diagnosis as melanoma of lesions previously termed nevi ("overdefinition"). In terms of overdefinition, there is evidence that criteria for the histopathologic diagnosis of melanoma have changed over a period of approximately two decades. Specialization may play a role in overdefinition; research has shown that when pathologists interpret the same lesion, dermatopathologists are more likely to diagnose low-stage (American Joint Committee on Cancer T1a) melanomas and general and/or surgical pathologists are more likely to diagnose atypical nevi. An important subset that contributes to overdiagnosis is melanomas that lack the property of tumorigenic vertical growth phase, thus lacking metastatic competence and perhaps not warranting diagnosis as overt melanomas. Studies have defined subsets of patients with very low-stage lesions diagnosed as melanomas in which observed survival has been 100%. In the past, many of these lesions would have been diagnosed as nevi, constituting overdefinition. Other key characteristics for very low-risk (or no-risk) lesions that are currently termed invasive "melanomas" include low Breslow thickness, Clark's level II invasion, absence of mitoses, and clinically, lack of observed or experienced dynamic changes. We propose a provisional terminology for diagnosing extremely low-risk subgroups as "melanocytic neoplasms of low malignant potential," aimed at reducing the negative personal and social effects of a cancer diagnosis for patients whose health and wellbeing are in reality not affected by an overdiagnosed "melanoma." With additional confirmation and appropriate consensus, it is likely that some of these subgroups can be reclassified as atypical or dysplastic nevi.

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https://doi.org/10.1016/j.clindermatol.2024.09.006

The incidence of melanoma has increased dramatically over the last 50 years, while the mortality rate has remained constant. This scenario appears to be explained by the phenomenon of "overdiagnosis," in which lesions diagnosed as melanoma appear to lack any capacity to cause death or signs.[1] The surgical cure rate for melanoma is more than 90%, consistent with this concept. Efforts directed at diagnosis of earlier, more subtle melanocytic proliferations as melanoma have led to the laudable goal of reducing mortality by identifying melanoma in its early, curable stages. Although there is no doubt that some lethal melanomas do arise from these (or similar) early stages, data suggest that such events are extremely rare when viewed from the perspective of any given individual potential precursor lesion. In other words, these lesions may be potential precursors; however, the rate of realization of this potential is very low.

Studies have identified a subset of patients with diagnosed melanomas but with no increase in the rate of melanoma-specific mortality. Similar observations in other tumor systems have resulted in changes in nomenclature such that these lesions are given terminology that does not label them as frank malignancies. We propose that such changes in terminology are warranted for these minimal or zero-risk melanomas, and the term "melanocytic neoplasm of low malignant potential (MNLMP)" has been suggested. Research continues to identify those subsets in which this low potential is effectively zero.

A brief history of histopathologic diagnosis of melanoma

The diagnosis of melanoma 200 years ago was typically made clinically based on a large ulcerated and bleeding black tumor that often had widespread metastases at diagnosis.[2,3] Histopathologic diagnosis was readily confirmed because of cytologic features such as anaplastic nuclear atypia and frequent mitoses with abnormal mitoses. A seminal contribution that expanded the histopathologic diagnostic criteria for melanoma enumerated the histopathologic properties of melanomas that had metastasized.[4] Most of these are properties of the intraepidermal component of these lesions, properties that are not correlated with metastatic risk. As a result, the diagnosis became uncoupled from the prognosis. Many of the diagnostic criteria that have been proposed, including most genomic criteria, have been developed against the gold standard of expert histopathologic diagnosis rather than outcome. Criteria that are related to outcome include Clark's levels of invasion, Breslow thickness, mitogenicity, and tumorigenicity, among a few others that we discuss.

Stepwise tumor progression in the melanocytic system

It was recognized early[5-8] that melanomas often had an adjacent component that was confined to the epidermis and/or to the papillary dermis without tumor formation. This was later termed the "radial growth phase (RGP),"[6] a term that was based on the clinical morphology of the lesions spreading as it were along the radius of an imperfect circle on the skin. The properties that define the next stage, the vertical growth phase (VGP), are tumorigenicity, referring to the dermis cells' ability to form an expansile mass, and mitogenicity, their ability to undergo cell division in that location.[7,8] Lesions in the RGP, by definition, lack these properties despite being invasive (Figures 1 and 2). Clinical and histologic images of melanomas can clearly demonstrate these 2 phases of tumor progression in many, though not all, melanoma lesions. There is a subset of lesions confined to RGP with a very good prognosis and another subset, called "nodular melanoma," in which tumorigenic VGP is present without an observable adjacent RGP.

Low-risk and high-risk melanomas

The process of tumor staging and microstaging has resulted in an elaborate and largely successful system to categorize melanomas according to risk.[9] The lowest risk category is that of T1 melanoma, defined largely according to Breslow thickness criteria. In his seminal 1,976 study of only 105 cases, Breslow[13] observed that tumors thinner than 0.76 mm did not metastasize. This "Breslow number," rounded to 0.8 mm, is still in use today for identifying "thin" melanomas lacking competence for metastasis, along with the absence of ulceration, which is uncommon in these thin lesions.[10] T1 melanomas are classified as having a Breslow thickness of $\leq$1.0 mm and have an overall mortality of approximately 4% at 10 years. A subset of T1a melanomas with a thickness of $<$0.8 mm was identified as a category for which sentinel node staging was inappropriate because of the good prognosis. Clark's levels of invasion have been largely but not entirely supplanted by Breslow thickness; however, there are subsets in which Clark's level is more predictive than Breslow thickness.[11] This includes the subset of T1a melanomas, especially those lacking VGP below.

Overdiagnosis of melanoma

Overdiagnosis of cancer has been defined as the diagnosis of a lesion as a cancer or malignancy that would not have had the capacity to cause death, signs, or symptoms in the lifetime of the host. This term has been applied to melanoma largely

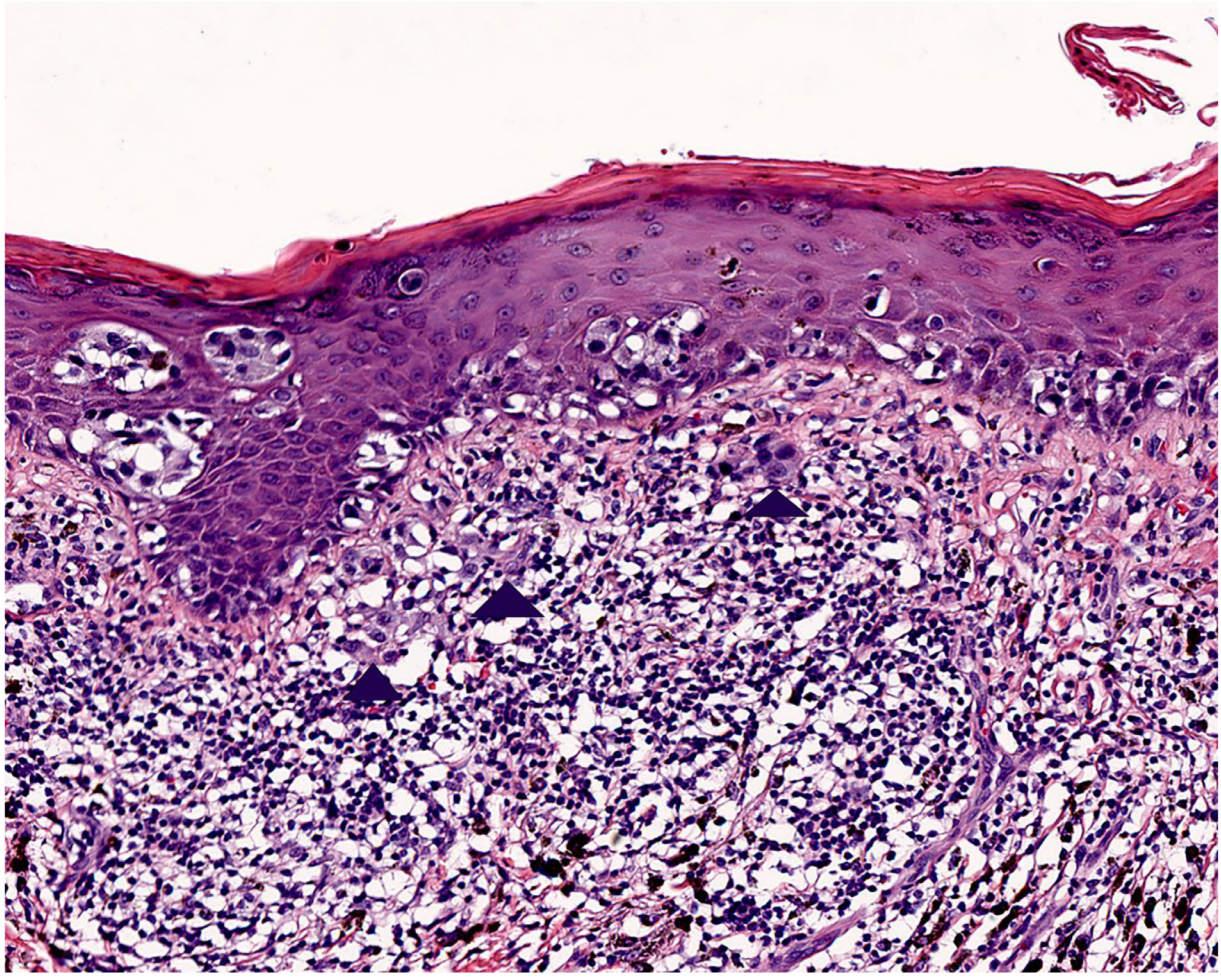

Fig. 1 Malignant melanoma, superficial spreading type, invasive radial growth phase. There are clusters of cells in the dermis that are not larger than the largest clusters in the epidermis (arrows), representing invasive melanoma confined to the radial growth phase in this single field of view.

in studies in which two prerequisites for cancer overdiagnosis are thought to occur: the existence of a silent disease reservoir and activities leading to its detection (particularly cancer screening).[12] The epidemiologic signature of overdiagnosis of melanoma, ie, rising incidence with steady and unchanging mortality, is shown in Figure 3.[13,14] A common thought on first observing these curves is to consider that the lesions in the incidence curve were all excised and therefore "cured," thus explaining the steady and relatively unchanging mortality rate in the presence of rising incidence. When the incidence of cancer is truly rising, mortality rates usually rise along with incidence, although generally at a slightly lesser rate, because not all of the "new lesions" that could have caused mortality can be identified and cured.[16] The unidentified lesions in the community, therefore, will progress and drag up the mortality curve. These curves for melanoma are convincing evidence of the presence of overdiagnosis at a high rate in melanoma.

Overdiagnosis has been linked, although not exclusively, to efforts at increasing early diagnosis by screening skin examinations, resulting in increased excision of asymptomatic lesions and their submission for histopathologic diagnosis.[15,16] In understandable efforts to avoid missing an early melanoma that could progress to a dangerous lesion, pathologists have also applied criteria at increasingly high levels of sensitivity, with less attention to specificity. There is direct evidence for this in a study that reviewed lesions diagnosed as nevi 20 years earlier in which 13% of the lesions were classified as melanomas despite having been diagnosed as benign nevi (albeit dysplastic) at the earlier time.[21] There was no evidence presented that such lesions had metastasized or caused signs or symptoms after their excision with a benign diagnosis 20 years earlier.

Indirect evidence for overdiagnosis in melanoma is provided by the finding that dermatopathologists in the United States are more likely to diagnose thin lesions as melanomas, compared with surgical pathologists who are likely to classify them as atypical or dysplastic nevi,[22] as illustrated in Figure 4. It can be postulated that dermatopathologists have been more exposed, compared with surgical pathologists, to

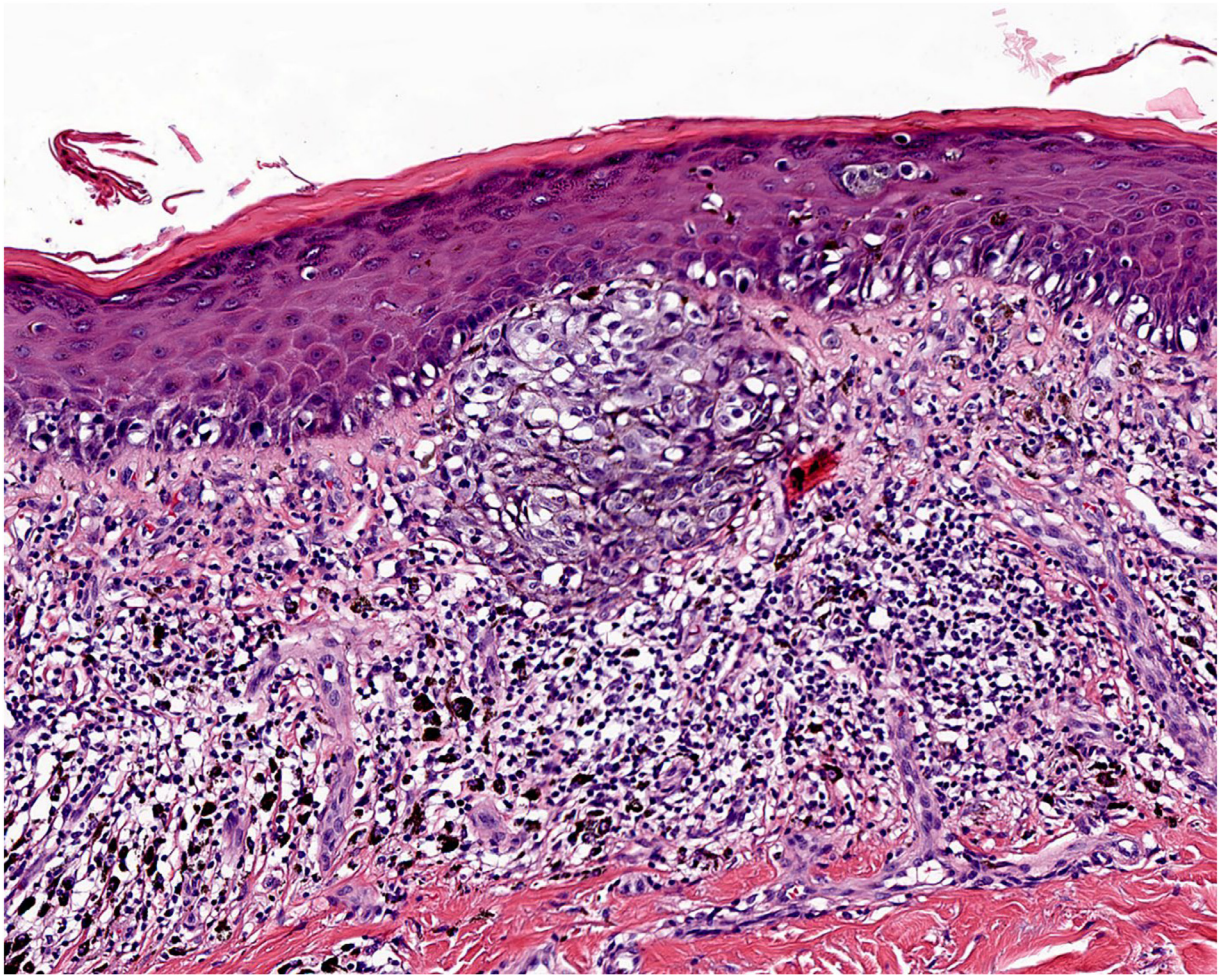

Fig. 2 In this field, from the same lesion as in Figure 3, there is a cluster of cells that is larger than any of the clusters that were present in the lesion within the epidermis. This cluster is separated from the epidermis by a very narrow zone of collagen, suggesting that it may have recently separated. There are rare mitoses, not evident in the image, indicating that this lesion represents tumorigenic and mitogenic vertical growth phase despite being Clark's level II (papillary dermis is expanded but not filled by the tumor) and Breslow thickness 0.3 mm. Such lesions, although low-risk, have at least a finite risk for metastatic progression.

education promoting the use of more sensitive criteria for the diagnosis of melanomas, producing a tendency for dermatopathologists to give more severe diagnoses than surgical pathologists interpreting the same lesions.

The significance of melanoma *in situ*

Given that melanoma *in situ* (MIS) is often observed adjacent to invasive and tumorigenic (and sometimes lethal) melanomas, it was easy to conclude that these were precursors or early stages of the melanomas with clear genomic evidence that this has occurred at least in those lesions[17]; however, the understandable inference that MIS is a dangerous precursor of melanoma has not been borne out after 50 years of observations. If MIS is included in the "overdiagnosis curve," the rise in incidence is even steeper, without any effect on mortality (Figure 3). If MIS is a dangerous precursor, and its incidence is truly increasing, then lesions "left behind" in the community should progress to more advanced and potentially lethal melanomas; this phenomenon has not been observed.

The significance of MIS has been called into question. Studies have demonstrated that any presumption that MIS left untreated is almost certain to progress and cause the death of a patient is unfounded.[18,19] Because severely dysplastic and/or atypical nevi cannot be reliably distinguished from MIS, there is justification for collapsing these into a single category, as in the proposed new M-PATH classification system.[20] Anecdotal experience, small case series, and the original observation that MIS is often seen in contiguity with more advanced stages of melanoma should, in our opinion, lead to a cautious approach to its management, with minimal complete excision being reasonable to recommend.[21] Even with

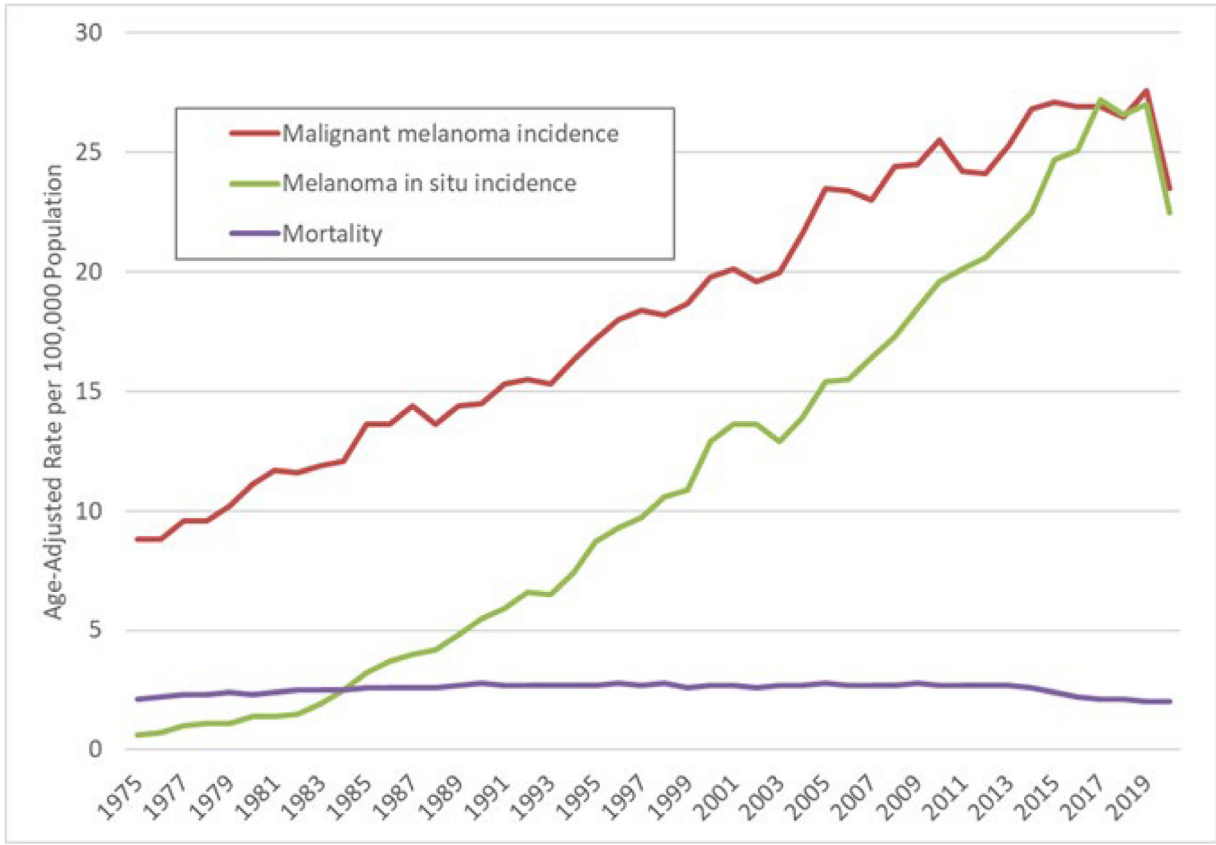

Fig. 3 The age-adjusted incidence rate of invasive and *in situ* melanoma and mortality rate, 1975 to 2020. There has been a dramatic increase in incidence, which is more pronounced for *in situ* melanoma, without a commensurate increase in mortality. The rate of increase has leveled off as per data from the last 5 years. The fall in incidence in 2020 is likely related to disruption of medical services during the Covid pandemic. The drop in the rate of mortality over the last 5 years began before this and is likely attributable to improved therapy for advanced disease.

this consideration, patients with these lesions should not be encouraged to believe that they suffer from any form of dangerous cancer.

Subsets of invasive melanoma with minimal or zero risk

Several studies have identified subsets of melanoma that have essentially no, or actually no, observed risk of death for patients in whom these lesions are diagnosed. In a study from the Penn Pigmented Lesion Group, with a follow-up study a few years later, there were no deaths observed in a cohort of 161 prospectively diagnosed patients with melanomas that lacked VGP.[22,11] This prognostic significance of VGP has been confirmed in other studies.[23–34] Despite this, VGP is not included in national databases, and one criticism has been that its diagnosis is poorly reproducible. In one study, the diagnosis of VGP in thin tumors was found to be about as reproducible as that for Breslow thickness.[24] Studies of tumor progression markers, including proliferation markers, progression-associated antigens, and genomic studies, have found clear differences between RGP and VGP melanoma cells.[23,25,35] Taken together with the observed excellent survival, these studies lead to the conclusion that this subset of thin T1a melanomas are not true biological malignancies.

In a recent study using the Surveillance, Epidemiology, and End Results database, a group of almost 1,000 patients under the age of 44 years, with T1 melanomas that were Clark's level II (lacking level III or greater) had an observed seven-year survival of 100%.[14] The significance of Clark's level II may be related to its being a good but not perfect surrogate for the absence of VGP. Clark's level II is classified as a tumor that enters the papillary dermis but does not fill and expand it (Figure 1). VGP is classified as a cluster of cells in the dermis that is larger than the largest cluster of cells in the epidermis (Figure 2), consistent with the notion that such clusters in the dermis have acquired the capacity to proliferate there. Evidence suggests that in thin RGP melanomas, the cells in the dermis do not have this capacity as judged by proliferation markers and other studies.

Although VGP has not been found to be an independent predictor in most studies of prognosis in melanoma,[36] most of these have not focused on the thin melanoma subgroup, and in addition, there may be confounding between the two

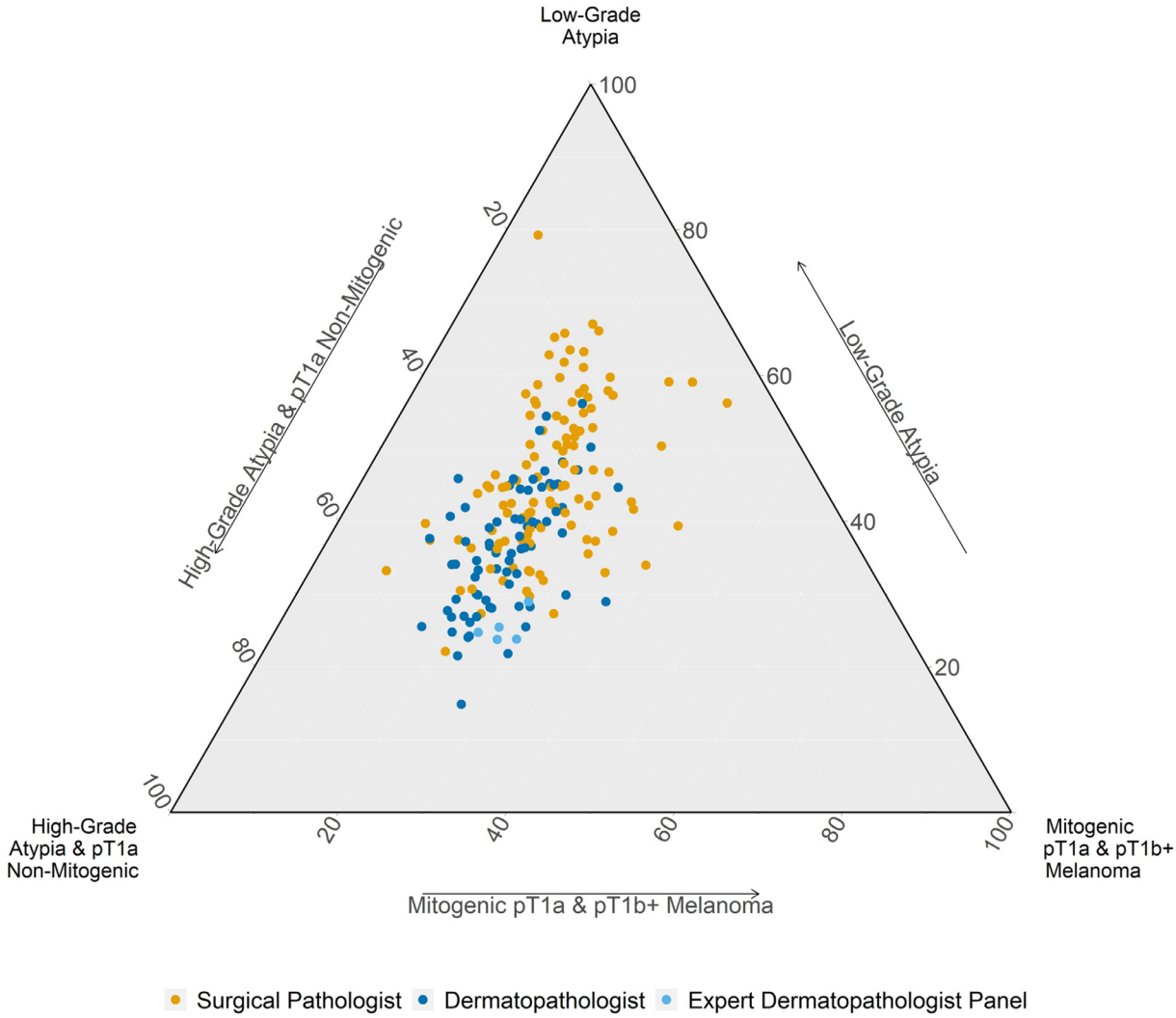

Fig. 4 Ternary diagram indicating the distribution of diagnoses made by 113 surgical pathologists, 74 dermatopathologists, and an expert dermatopathologist panel. Each independently interpreted a set of 48 melanocytic lesions. Dots closer to one corner of the triangle (e.g., closer to Low-Grade Atypia) indicate more diagnoses in that category among the set of 48 cases. Dermatopathologists, as well as the expert panel, tended to diagnose more lesions as high-grade atypia or representing non-mitogenic T1a melanomas.

defining variables of VGP, mitogenicity, and tumorigenicity. In a study of prospectively registered thin melanoma cases where these 2 factors were distinguished, VGP did not directly enter the final multivariate model as an independent prognostic factor; however, it did enter a prognostic tree as a factor for explaining heterogeneity in metastasis rates among patients with thin nonmitogenic melanomas, with 10-year metastasis rates of 2.6% compared with 1.2% for tumorigenic and nontumorigenic melanomas, respectively, in this very low-risk category.[14]

Taking these findings together, it seems clear that certain subsets, at least, of thin T1a melanomas lack competence for metastasis and should not be considered as dangerous malignancies. As already mentioned, similar situations in other cancers have led to changes in terminology such that patients are no longer labeled with terms that may indicate to them that they have a potentially lethal malignant tumor. These considerations suggest that VGP should be returned to the American Joint Committee on Cancer staging system (and thus also to the Surveillance, Epidemiology, and End Results database), where its relevance is greatest for melanomas in the T1 category, because VGP is almost universally present in thicker melanomas.

Proposal for changes in terminology of MIS and T1a low-risk melanomas

In view of the considerations discussed above, we have proposed the introduction of the term "MNLMP," which may

seem cumbersome but is reasonably descriptive. The term "melanoma" (including MIS) is replaced by "neoplasm," which is a technical term that does not include the obligatory consideration of malignancy. The term "low malignant potential" has been used in other contexts for tumors that, in most cases, will behave as benign lesions while allowing for the fact that there may be rare exceptions.[37] With respect to severely dysplastic nevi and MIS,[38] these types of lesions, to which we would add the MNLMP subset of T1a invasive melanoma, may be better conceptualized as risk factors for melanoma, rather than obligate precursors to invasive melanoma, and, to invasive VGP melanoma.

Management

Management of these lesions should be discussed in consensus groups of stakeholders, including patient advocates, family practitioners, dermatologists, surgeons, and pathologists, and an international conference may be needed to formulate a consensus statement. In our opinion, it is premature to identify any such group of lesions for which patients can be completely reassured without at least limited further management, sufficient to interrupt tumor progression if it should be fated to occur in that lesion.

Options for management would include careful observation of the lesional site, perhaps by the patient or perhaps by a physician or other health care provider, and this might be preceded by a procedure to ensure that the lesion has been completely removed, with at a minimum, a clear margin of normal tissue around the scar of the biopsy procedure and any residual lesion.

Conclusions

There is a subset of patients diagnosed with melanomas but with no increase in melanoma-specific mortality. In other tumor systems, this has resulted in changes in nomenclature so that these lesions are not labeled as frank malignancies. It seems that this subset of lesions presently diagnosed as T1a melanomas should be relegated from the category of melanomas into the category of severely dysplastic or atypical nevi, for which, again at present, complete excision is recommended management, along with follow-up based on the patient's individualized risk for future development of melanoma.[26] In conclusion, we propose changes in terminology for these minimal or zero-risk melanomas, and the term "melanocytic neoplasm of low malignant potential (MNLMP)" has been suggested. Research continues to identify those subsets where this low potential is effectively zero.

Declaration of competing interest

The authors declare no conflicts of interest.

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