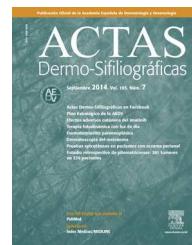


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Diagnostic and Therapeutic Techniques in Lentigo Maligna: An Update[☆]



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FR-Lentigo maligno, actualización en técnicas diagnósticas y terapéuticas

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Imiquimod y radioterapia

Lentigo maligna (LM), an *in situ* lesion, and lentigo maligna melanoma (LMM), which invades the dermis, constitute a diagnostic and therapeutic challenge.

Differential diagnosis includes seborrheic keratosis, solar lentigo, lichenoid keratosis, actinic keratosis, and pigmented actinic keratosis. Dermoscopic criteria may assist in the diagnosis of LM and LMM, but lack full sensitivity and specificity. While histologic diagnosis is the gold standard, atypical melanocytes are rare and sparsely distributed in these two conditions. LM is thus difficult to distinguish from melanocytic hyperplasia in photodamaged skin, and lesion margins are hard to define. Some authors have recently highlighted the importance of questioning any histopathological diagnosis of junctional nevus, dysplastic nevus, or atypical lentiginous nevus for lesions occurring in photodamaged skin on the face and neck. Nevi in this area are typically papule-type lesions of the same color as adjoining skin, but are clinically different from LM. Any of the above nevus diagnoses may underestimate an LM lesion as a benign junctional melanocytic proliferation.¹ Immunohistochemistry can be of use in these equivocal cases. Typical markers include S-100, human melanoma black-45 antigen (HMB-45), melan-A (MART-1), and microphthalmia-associated transcription factor (MITF), all of which are highly sensitive to melanocytes. However, these markers fail to differentiate melanocytic nevi from melanoma lesions; P16 protein expression and proliferation markers Ki-67 and pHH-3 help with this. Identification of specific mutations in the *BRAF*, *NRAS* and *KIT* genes may be of use, especially given the recent availability of targeted drugs.²

Reflectance confocal microscopy (RCM) was introduced to improve diagnostic accuracy, define the extent of a lesion,

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and detect recurrences. RCM allows the performance of what might be termed an *in vivo* optical biopsy with no need for surgery. There is a published diagnostic algorithm with 93% sensitivity and 82% specificity,³ and RCM is particularly useful for distinguishing facial LM from benign pigmented macules without the need for a biopsy.⁴ Moreover, presurgical mapping may help reduce positive-margin rates and recurrence rates.

The treatment of choice for LM and LMM is surgical excision. Several authors have indicated that 5-mm margins may be inadequate for LM, particularly owing to amelanotic spread on the periphery of the lesion; 9-mm resection margins are recommended instead.⁵ Mohs micrographic surgery is more precise than excision and has a lower recurrence rate (0%-6.25%). Certain cases may be selected for second-line treatments such as imiquimod, radiotherapy, and cryosurgery. A review of radiotherapy for LM found a recurrence rate of 5% and a rate of only 1.4% for progression to LMM.⁶ The authors recommend mapping the lesion first with RCM and irradiating the lesion area plus a 1-cm margin of healthy skin at a depth of 5 mm to ensure irradiation of hair follicle melanocytes. They highlight the presence of blue-gray pigmentation after treatment and the fact that RCM can distinguish this coloring from persistent or recurrent LM.

Developing a familiarity with new imaging techniques and therapeutic options for LM and LMM is important in order to

offer our patients greater diagnostic accuracy and appropriate treatments, and to meet current growing demand for noninvasive, cosmetically acceptable techniques.

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