Immunohistochemical study of the expression of N-cadherin in cutaneous melanoma and in dysplastic melanocytic nevi

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It has been suggested that the invasive and metastatic potential of melanoma cells reflects their ability to undergo epithelial-mesenchymal transition (EMT)-like phenotypic changes (1). Important hallmarks of EMT include the loss of E-cadherin expression and increased expression of the cell adhesion molecule N-cadherin. This cadherin switch leads melanoma cells to lose contact with keratinocytes in the epidermis and interact instead with stromal fibroblasts and endothelial cells, thus promoting dermal and vascular melanoma invasion (2). In melanoma, up-regulation of N-cadherin can be induced by the overexpression of the transmembrane receptor Notch1, thus providing a mechanism that underlines increased melanoma cell adhesion, survival, growth, and tumor progression when Notch signaling is activated (3). In this study, the expression of N-cadherin and Notch1 was evaluated by immunohistochemical analysis in primary cutaneous melanomas and lymph node metastases. First, we evaluated the prognostic impact of high N-cadherin expression on survival in melanoma patients. Second, we correlated the expression of N-cadherin with the full clinicopathological data of patients. Third, we investigated the relationship between the expression of N-cadherin and Notch1. Moreover, N-cadherin expression was evaluated in dysplastic melanocytic nevi and in normal skin. The results will be discussed.

References

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