accidental exposure. Kanerva et al[9] described sensitization in 2 workers who suffered burns after exposure to MCI/MI; within 24 hours these were complicated by allergic reactions. Patch testing was positive for MCI/MI. Other derivatives of isothiazolinone have also been shown to cause sensitization after accidental burns.[13,14]

Eczematous lesions appeared very rapidly (less than 120 hours) after the chemical burn. In this case, sensitization could have been caused at the time of the burn due to the quantity and purity of the chemical to which she was exposed, or because she had already become sensitized to the product by daily use (although the patient denied having previous skin lesions). Thus, it is impossible to establish when sensitization occurred as no patch tests were performed prior to the time of the burns.

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Primary Melanoma With Multiple Skin Metastases

Melanoma primario con metástasis cutáneas múltiples

To the Editor:

Disseminated skin metastases from melanoma are rare. Several hypotheses in the literature attempt to explain this unusual explosive spread but none is conclusive.[1] It has been suggested that some of the factors involved are adrenocorticotropic hormone, drug abuse, and metabolic or hormonal disorders.[2] It is sometimes difficult to distinguish these metastases from multiple primary melanomas and histology findings of the 2 diseases may overlap, making differential diagnosis impossible.[3]

We describe the case of a 41-year-old man who came to our clinic because of the sudden appearance of more than 300 pigmented lesions over the previous 2 months (Figure 1). Physical examination revealed the presence of an irregular, heterochromic pigmented lesion of 3 cm in diameter on the left scapula. It had appeared 2 years earlier but the patient had not considered it important. Dermoscopy revealed a lesion with multiple components, with irregular globules at the periphery, and a central blue-white veil (Figure 2). There were no palpable locoregional lymph nodes.

The clinical suspicion of melanoma with multiple skin metastases led us to perform exhaustive laboratory tests, which revealed parameters within normal limits.

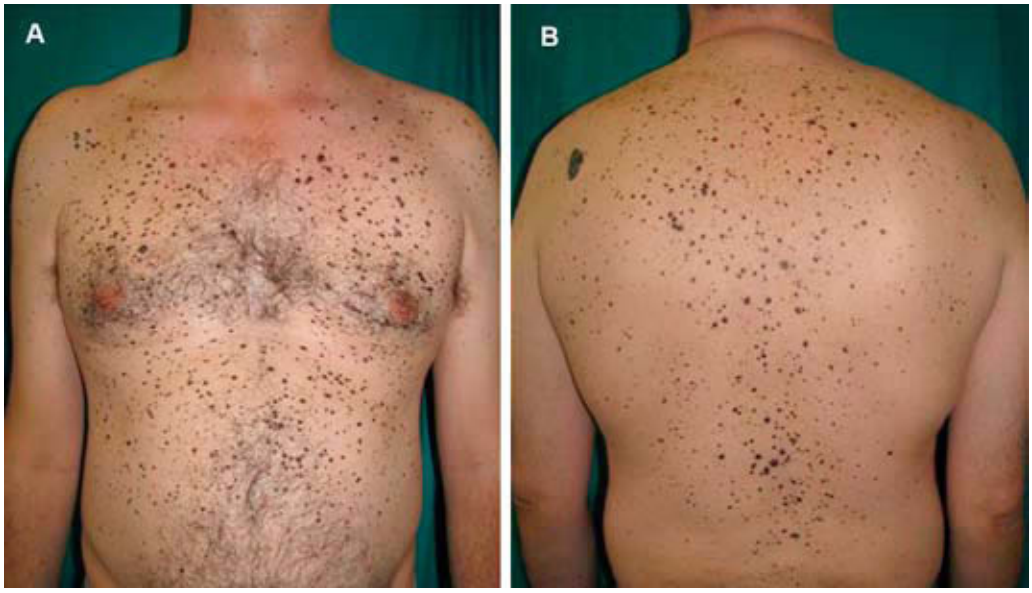

Figure 1 A, Anterior view. Numerous small pigmented lesions distributed widely on the abdomen and chest. B, Posterior view. Numerous small pigmented lesions and, on the left scapula, a larger pigmented lesion that is blackish and irregular.

Supraclavicular, mediastinal, and hilar lymphadenopathies were observed on computed tomography. The patient had a personal history of paranoid schizophrenia treated with risperidone and quetiapine, but there are currently no data in the literature to suggest that these drugs may be involved in metastatic spread. Because the patient's sister had had a melanoma 25 years earlier, we performed a genetic study of the patient that revealed no mutations in the genes most commonly altered in familial melanoma cases (*CDKN2A* and *CDK4*).[4]

We performed an excisional biopsy of the largest lesion and histopathology revealed a Clark level IV superficial spreading melanoma with a Breslow thickness of 2.5 mm. We observed a large number of mitoses

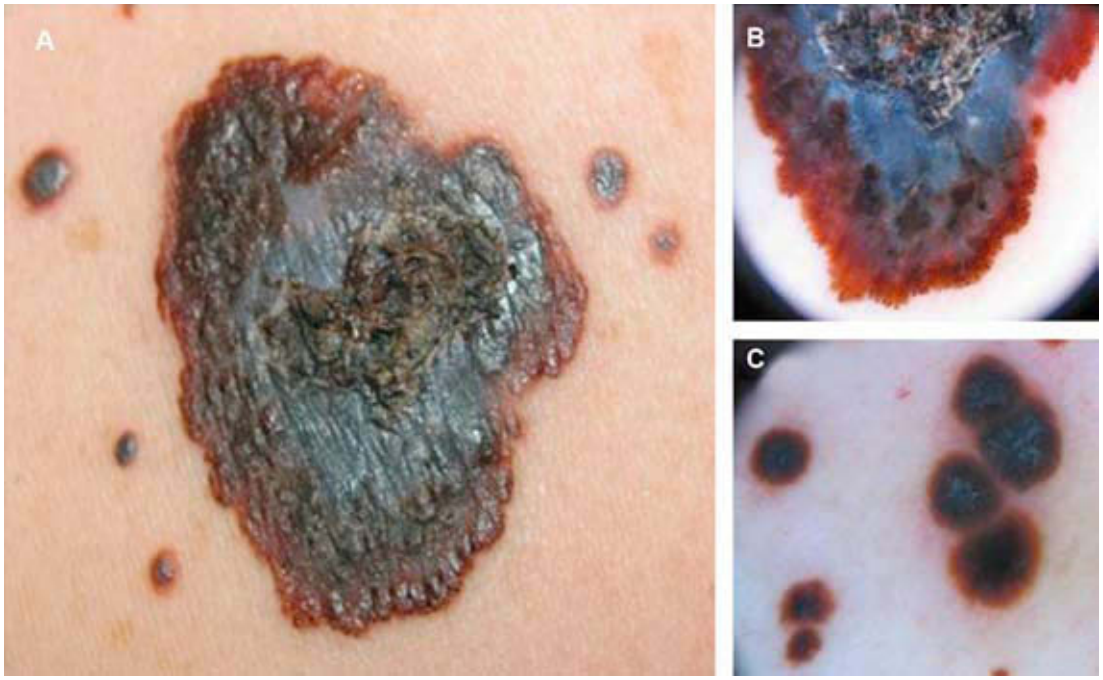

Figure 2 A, Under higher magnification the larger lesion is asymmetric and multicolored, with brownish, blackish, and bluish zones. B, Dermoscopy shows a sharp border, a pattern with multiple components, heterochromia, irregular globules at the periphery, and a central blue-white veil. C, Dermoscopy of the skin metastases shows well-defined borders with homogeneous blackish pigmentation and, in some lesions, a central steel-blue appearance.

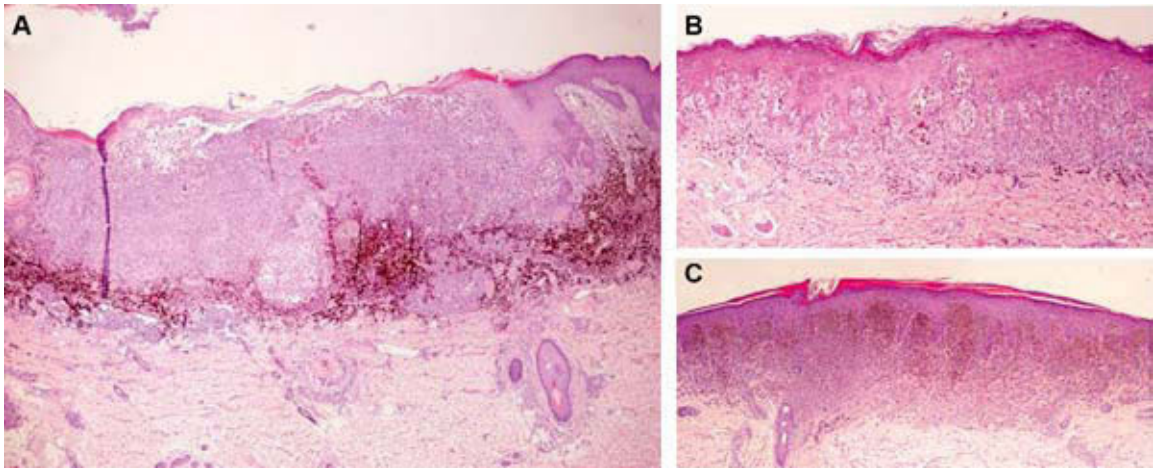

Figure 3 A, Histopathology of the lesion revealed a Clark level IV superficial spreading melanoma with a Breslow thickness of 2.5 mm (hematoxylin-eosin, original magnification ×100). B, Under higher magnification we see an intense pagetoid component, a finding typical of primary melanomas (hematoxylin-eosin, original magnification ×200). C, Histopathology of one of the small lesions shows a lesion with well-defined borders in the superficial dermis with no activity at the dermoepidermal junction and no inflammatory or pagetoid component (hematoxylin-eosin, original magnification ×40).

and an extensive pagetoid component, both of which are histopathological findings characteristic of primary melanomas (Figure 3). We also removed 2 small lesions whose histopathology revealed them to be well-defined lesions in the superficial dermis, with no activity at the dermoepidermal junction, no epidermotropism, and no pagetoid component (Figure 3C). Immunohistochemical studies with S-100, HMB -45, and Melan-A were performed on both samples, revealing intense, irregular, and asymmetric uptake of the markers by the melanocytic cells. This confirmed the malignancy of all samples, but no significant differences in the pattern of uptake of the reactants were observed between the primary melanoma and the skin metastases. With a firm diagnosis of primary skin melanoma with multiple skin metastases, the patient was referred to the oncology unit to start chemotherapy. Two months later he came to the emergency department because of severe dyspnea and was admitted to hospital. Radiographs revealed a miliary metastatic spread to the lungs and the patient died a few days later of cardiorespiratory arrest secondary to acute respiratory failure.

Although people who have had a melanoma have a higher risk of multiple primary melanomas (usually 2 and rarely 3) than the rest of the population, the rapid appearance of a large number of metastatic lesions in our case, causing the death of the patient, made it relatively easy to distinguish between primary melanomas and multiple skin metastases.[5] Histopathology did not reveal epidermotropism in our case; however, there have been reports that metastases can extend beyond the dermis to affect the dermoepidermal junction and the epidermis,[6] which can make differentiation of the 2 diseases difficult or even impossible. A series of clinical, histopathological, and immunohistochemical criteria must be applied in order to reach a correct diagnosis.[7] In clinical terms, primary lesions are usually asymmetrical, irregular, and multicolored, whereas metastases tend to be well-delimited, uniform lesions with homogeneous coloring. Histopathology of primary lesions usually reveals an extensive pagetoid component with activity at the dermoepidermal junction and atypical melanocytes in the epidermis. Metastases show greater dermal involvement and there is usually no activity at the dermoepidermal junction. These findings should be interpreted with caution because several cases of skin metastases of melanoma reported in the literature had conspicuous epidermotropism and were almost indistinguishable from primary melanomas on histopathology.[8]

In conclusion, clinical and pathological correlation and a comprehensive patient history are crucial to reach a correct diagnosis and prescribe suitable treatment, although future advances in genetics and immunohistochemistry will allow us to obtain more accurate diagnoses.

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Neoplasia de células dendríticas plasmocitoides

Plasmacytoid Dendritic Cell Tumor

To the Editor:

We describe 3 cases of the rare disorder known as plasmacytoid dendritic cell tumor.[1]

Case 1 corresponds to a 68-year-old man who presented with a 3-month history of asymptomatic, solid, erythematous-violaceous nodules and plaques on the trunk and extremities (Figure 1A). Skin biopsy revealed a dense infiltrate in the dermis that extended to the hypodermis, and the presence of a Grenz zone, with no invasion of the blood vessels or skin appendages. The pleomorphic infiltrate was composed of cells with a myelodysplastic appearance and prominent nucleoli, together with other elongated, hyperchromatic cells (Figure 2). Immunohistochemistry was positive for the CD4, CD7, CD43, CD45, CD56, and CD123 (Figure 2C) and for terminal deoxynucleotidyl transferase (TdT) (Table). A radiological extension study revealed subcentimeter lymph nodes, but no neoplastic cells were observed in a bone marrow biopsy. Treatment with 5 cycles of cyclophosphamide, doxorubicin, vincristine and prednisone (CHOP) chemotherapy led to remission of the skin lesions, but the patient died 4 months later as a consequence of disease progression.

The second patient was a 70-year-old man who presented with generalized violaceous macules, papules and flat tumors that spared the face and acral regions (Figure 1B). Histology and immunophenotype (Figure 3, A and B) were identical to the previous case, except for a positive result for type 1 T-cell lymphoma (TCL-1). The extension study revealed enlarged inguinal lymph nodes and polymorphic blast cell infiltration of the bone marrow. Administration of the acute leukemia protocol developed by PETHEMA (Spanish Program for the Study and Treatment of Hematological Malignancies) for medically fragile patients resulted in a 6-month remission, after which the disease progressed and the patient died.

Case 3 corresponds to a 52-year-old man who presented with brownish and erythematous macules and plaques on the trunk and extremities (Figure 1C). Biopsy revealed a dense infiltrate of monomorphic medium-sized blast cells with abundant mitoses throughout the dermis (especially in the deep dermis), and the presence of a Grenz zone. There was also evidence of bone marrow infiltration. Immunohistochemistry results are summarized in the Table. The patient achieved complete remission in response to treatment with daunorubicin and cytarabine. He subsequently received an allogeneic bone marrow transplant from a human leukocyte antigen-identical sibling, and, at the time of writing, has been in clinical remission for 12 months.

Plasmacytoid dendritic cell tumor, which occurs most frequently in older men, is estimated to account for 0.7% of all cutaneous lymphomas. It probably originates in nonantigen-presenting, interferon-producing type 2 dendritic cells, which differentiate into plasmocytoid precursor cells.[2] Plasmacytoid dendritic cell tumor is thought to be a form of aleukemic leukemia cutis, similar to myelogenous leukemia.[3]

Early symptoms appear in the skin in 50% of patients. Other organs are involved in the other 50% of patients (lymph nodes, 50%; spleen, 20%; mucosa, 10%; and bone marrow, 5%-25%). The early development of B symptoms and of leukemia is rare; cytopenia, however, may develop.[2] The most frequently described skin symptom is the appearance of disseminated, violet-colored plaques and tumors—as was the case with our patients—although presentation as a solitary tumor is also possible.[4] Cota et al[5] distinguish between 3 types of skin signs: tumors and generalized plaques, a solitary tumor, and disseminated macules.

Despite a good initial response to treatment in 80% of patients, the disease usually progresses rapidly to fulminant leukemia, which is myelomonocytic in 10% to 20% of cases.

Histology typically shows a dense infiltrate throughout the dermis that extends into the hypodermis. In up to 20% of cases, there is only a discreet perivascular or interstitial infiltrate. The presence of a Grenz zone is characteristic. The cells may be atypical medium-sized mononuclear cells with dispersed chromatin, or there may