blood cell count of 8200 cells/mL, 70% neutrophils, and left shift (7% band forms). Globular sedimentation rate was 25 mm/h and the chest x-ray revealed no significant abnormalities. Histology of a biopsy sample taken from a lesion on the forearm showed the presence of a subepidermal blister with intense neutrophilic inflammatory infiltrate, but no signs of leukocytoclastic vasculitis. Serology was negative for syphilis, herpes simplex, and hepatitis B and C, but positive for HIV-1 in an enzyme-linked immunosorbent assay. This finding was confirmed in a second test. The skin lesions improved after initiating treatment with a tapering course of oral prednisone (beginning at 50 mg/d) for 6 weeks, with no subsequent relapse. The patient was referred to the infectious diseases department of the hospital, with initial analysis showing a low CD4 T-cell count (285 cells/mL) and a viral load of 100 000 copies/mL.

Other suggested pathogenic factors include photosensitivity, reactions to antiretroviral therapies, or those related to phenomena of sudden immune restoration in patients in whom antiretroviral therapy has been recently initiated. Cofactors may have played a role in some of the cases reported, for example the treatment of drug-induced aplasia in HIV-positive patients with granulocyte colony-stimulating factor (G-CSF).

The presence of blister-like lesions in Sweet Syndrome is described relatively frequently as the clinical outcome of intense edema and inflammatory infiltrate in the dermis, which leads to subepidermal detachment.

In this case, factors associated with Sweet syndrome other than infection by HIV were not observed, given that the patient reported none of the traditional indicators (catarrh, new medication). Even if the exact mechanism of the association is uncertain, we must stress the significance of considering infection with HIV in patients with Sweet syndrome, especially in young patients exhibiting associated risk behaviors.

References


Benign Lymphangiomatous Papules and Plaques After Radiotherapy

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To the Editor:

Lymphangiomas are tumors that normally appear at birth. They are formed from dilated lymph vessels that may extend to the subcutaneous cellular tissue.

A number of causes of acquired lymphangiomas such as radiotherapy and surgery have been reported. The area irradiated during radiotherapy may develop benign vascular proliferations such as acquired progressive lymphangioma or malignant processes such as high-grade angiosarcoma, even when low doses of radiation are used.1,2 Within what are considered acquired

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References

lymphangiomatous lesions, benign lymphangiomatous papules after radiotherapy have specific characteristics.

Our patient was a 54-year-old woman with a history of stage T2 N1b M0 infiltrating ductal carcinoma in the right breast, diagnosed in 1998, and treated by tumorectomy and right axillary lymphadenectomy, chemotherapy, hormone therapy, and external radiotherapy of the entire chest wall and right breast at a dose of 50 Gy. She attended our clinic for the evaluation of progressive asymptomatic lesions on the irradiated skin of the right breast. The lesions had appeared approximately 1 year earlier (6 years after receiving radiotherapy).

Physical examination of the right breast showed yellowish skin coloring. This skin was covered with multiple erythematous papules that coalesced in places to form small vesicular plaques and lesions filled with clear or occasionally bloody fluid (Figure 1). There was no associated lymphedema.

Biopsy of one of the vesicles revealed marked vascular dilation in the papillary dermis that extended into the epidermis (Figure 2). As the vessels penetrated deeper into the dermis, they got narrower and more irregular and tortuous, and took on a lymphatic appearance (Figure 3).

The vascular spaces were covered by a single discontinuous strand of endothelial cells with oval hyperchromatic nuclei that protruded towards the lumen and that coalesced to form small plaques. Sometimes, as was the case in our patient, vesicles may be present. The latency period between radiotherapy and the onset of the first lesions is long, between 3 and 20 years. Characteristically, the patients do not report associated symptoms or present lymphedema.

Histology reveals marked vascular dilation in the papillary dermis that may extend into the epidermis, thereby giving the lesion a tuberous appearance. The vascular lesion is relatively well circumscribed, although not encapsulated, and may reach the deep dermis and occasionally extend to the subcutaneous cellular tissue, although this happens more often with malignant tumors such as angiosarcomas.

The vascular spaces are covered by a single discontinuous thread of endothelial cells that may have flat or large, oval nuclei that are hyperchromatic and that protrude towards the lumen. Occasionally, small nucleoli may be present. Mitotic and atypical cells are not present.

In summary, we believe that, in agreement with Díaz-Cascajo et al, lymphangiomatous papules and plaques after radiotherapy are a variant of acquired lymphangioma. Likewise, the term benign lymphangiomatous papules...
or plaques after radiotherapy is the most appropriate because it makes reference to the clinical presentation of the lesions, their nature, and their relationship with radiotherapy.

References