Histology reveals a greater or lesser degree of epidermal atrophy with flattening of the crests. The blisters are subepidermal, have a blood-stained content, and may be associated with edema of the papillary dermis. In the reticular dermis there are prominent thick bundles of hyalinized collagen that destroy the adnexal structures.

The main differential diagnosis of morphea is with lichen sclerosus et atrophicus, and the 2 diseases can coexist in some patients. In contrast to lichen sclerosus et atrophicus, the basal layer and the elastic fibers of the reticular dermis are unaffected in bullous morphea, which also does not show follicular plugs.

Numerous therapeutic strategies have been used, including phototherapy, topical and systemic corticosteroids, topical vitamin D analogs, antimalarials, various immunosuppressant agents, systemic retinoids, certain antibiotics, colchicine, and phenytoin. Also, isolated cases have been treated with extracorporeal photophoresis, N-acetylcysteine, imiquimod, sulfasalazine, or skin grafts, with varying results.

In conclusion, we have presented a case of bullous morphea in which lymphangiectasia was not detected on histology; this, together with the pretilial site of the lesions and their blood-stained content, leads us to speculate that unperceived trauma may have been the cause of the blisters in this case. Regarding treatment, it would appear reasonable to adopt an approach similar to that for nonbullous forms of morphea, starting with topical agents (corticosteroids, vitamin D analogs) or phototherapy, and advancing to systemic treatments such as methotrexate if no response is observed.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

References


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Amlodipine-Associated Photodistributed Telangiectasias

Telangiectasias fotodistribuidas asociadas a amlodipino

To the Editor:

Telangiectasias due to drugs or iatrogenic damage are very rare. Some of the responsible drugs are lithium, interferon-alpha, and isotretinoin. However, when telangiectasias appear in sun-exposed areas, they are usually related to the use of calcium channel blockers (CCB), although this has also been reported in association with other drugs such as venlafaxine and cefotaxime.

In these cases, lesions appear in sun-exposed areas a few months after starting the treatment. The prognosis is good, as a correct diagnosis followed by a change of treatment to a drug from another group, will lead to a rapid improvement and complete or almost complete disappearance of the lesions.

We present a patient with photodistributed telangiectasias associated with treatment with amlodipine. The lesions disappeared a few weeks after discontinuation of the treatment.

A 36-year-old woman consulted for the appearance of erythematous lesions that had arisen on the face and in the presternal region approximately 10 months earlier and for which she had received no treatment. A relevant finding in her past history was systemic hypertension on treatment with amlodipine for the previous year. She denied any history of rosacea or the use of topical corticosteroids. On examination, numerous erythematous-purpuric lesions and reticulated blood vessels were observed in the anterior thoracic region. All the lesions measured less than 1 cm in diameter and they blanched on pressure (Fig. 1). A few similar lesions were observed on the dorsum of the nose. Histology showed slight dilatation of the capillaries of the vascular plexus of the superficial dermis and a minimal lymphohistiocytic perivascular infiltrate; there were no signs of vasculitis and no increased presence of mast cells. Blood tests, including complete blood count, biochemistry, and basic autoimmune studies, were normal or negative.

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The differential diagnosis must include other causes of telangiectasias, such as liver disease and hyperestrogenic states, autoimmune diseases such as scleroderma, dermatomyositis, and lupus erythematosus, and telangiectasia macularis eruptiva perstans. A detailed medical history and physical examination, and improvement of the manifestations on discontinuation of the implicated drug enable us to reach the diagnosis.

Treatment is based on withdrawal of the responsible drug. The prognosis is good, with improvement or disappearance of the lesions within a few weeks after discontinuing treatment.

In conclusion, telangiectasia is a common cause of consultation in dermatology. Photodistributed lesions associated with CCBs are a rarely reported side effect, and targeted questions about these drugs must therefore be included in the history. The clinical course after starting treatment with the drug is the key to diagnosis, and management consists of drug withdrawal. Dermatologists must be aware of this entity to avoid unnecessary investigations.

Conflicts of Interest
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