Milia after Allergic Contact Dermatitis from Poison Ivy: Two Cases

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Milia have rarely been reported as a complication of severe allergic contact dermatitis. To our knowledge, milia have not previously been associated with poison ivy dermatitis. We present two cases of milia after allergic contact dermatitis to poison ivy.

MILIA are common, small, yellow white or white infundibular cysts 1 mm or smaller, resembling larger infundibular-type cysts that are filled with ''flaky'' orthokeratotic corneocytes. Milia are divided into primary and secondary forms.[1] Secondary milia are localized and are sometimes associated with (1) particular diseases (especially blistering disorders), (2) trauma, and (3) rarely, medications (including benoxaprofen, topical steroids, 5-fluorouracil, cyclosporine, penicillamine, and sorafenib). Secondary milia occur classically in subepidermal blistering disorders such as porphyria cutanea tarda, epidermolysis bullosa acquisita, dystrophic epidermolysis bullosa, and (uncommonly) bullous pemphigoid. To our knowledge, there are very few reports of milia occurring after allergic contact dermatitis (ACD)[2–6] and none specifically after ACD from poison ivy.

Case Reports

Case 1

A 38-year-old Caucasian woman presented with a 3-month history of asymptomatic white papules on both forearms. These lesions immediately followed an episode of confirmed severe ACD from poison ivy. A nurse practitioner at a pharmacy diagnosed and treated the patient's ACD with an oral prednisone taper and triamcinolone 0.1% cream, which was applied three to four times daily. The patient was otherwise in her usual state of good health; methimazole for hyperthyroidism was her only medication. Physical examination revealed linear streaks of white milia on both arms, which was consistent with the location of her previous rhus dermatitis (Fig 1). A therapeutic trial of topical retinoids was unsuccessful, but the patient improved with manual extraction of the milia.

Case 2

A 44-year-old Caucasian woman with a history of hypertension, rosacea, and granuloma annulare developed severe contact dermatitis on her arms from either poison ivy or Virginia creeper; she was treated with calamine lotion, oral antihistamines, and a 1-week prednisone taper. Three months later, she presented with numerous little white papules in the areas of the previous dermatitis (Fig 2). A biopsy specimen showed multiple cysts in the upper dermis, with thin stratified squamous epithelium

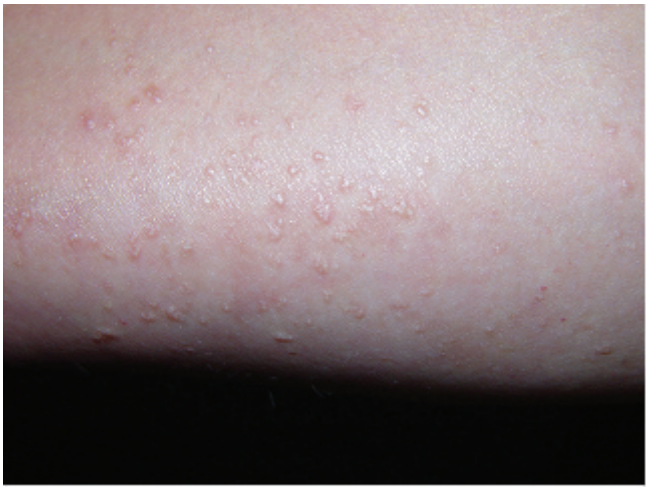

Figure 1. *Case 1,* Numerous small white papules on the arm in areas of prior contact dermatitis. Many lesions are grouped, linear, or both.

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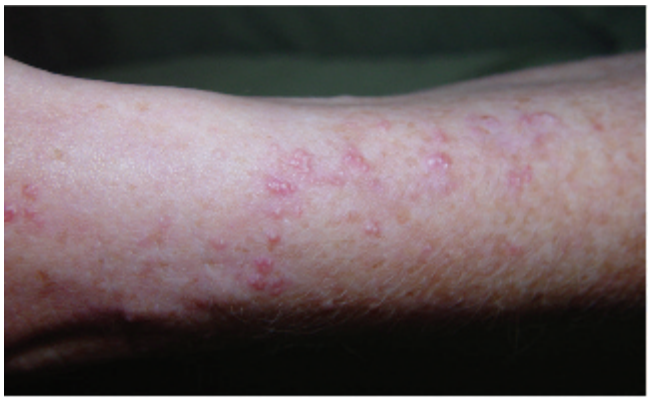

Figure 2. *Case 2,* Numerous small white papules on the arm in the areas of prior contact dermatitis.

and laminated corneocytes that were surrounded by a mononuclear inflammatory infiltrate (Fig 3, Fig 4, and Fig 5). Her 24-hour urine porphyrins were normal. Twenty lesions were extracted manually. The patient declined to return for further manual extraction because she noticed the spontaneous resolution of most lesions over the next few months.

Discussion

Milia have been reported rarely as a complication of ACD. Ibbotson and colleagues reported two such cases: (1) a 49-year-old man with severe ACD from para-aminobenzoic acid (requiring intravenous corticosteroids) in his sunscreen and (2) a 37-year-old woman with ACD on her hand secondary to contact with sticking plaster used to secure an intravenous canula.[2] Bryden and colleagues reported on a 59-year-old woman who developed milia after a polymorphous light eruption and who had a severe photoallergic contact dermatitis from sunscreens containing oxybenzone, avobenzone, and octyl methoxycinnamate.[3] Thormann and Andersen reported on a 54-year-old man with a known sensitivity to oak moss who developed milia on the forearm after severe ACD (requiring systemic corticosteroids) from a single application of chloroatranol

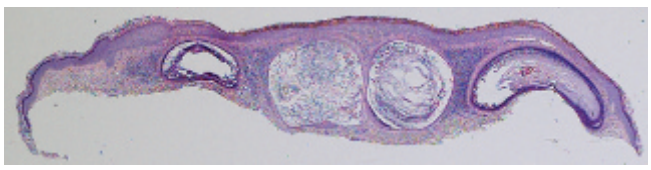

Figure 3. Biopsy specimen showing apparently four milia at scanning magnification. It is not known unequivocally whether they are interconnected, but it is possible (hematoxylin and eosin staining, ×20 original magnification).

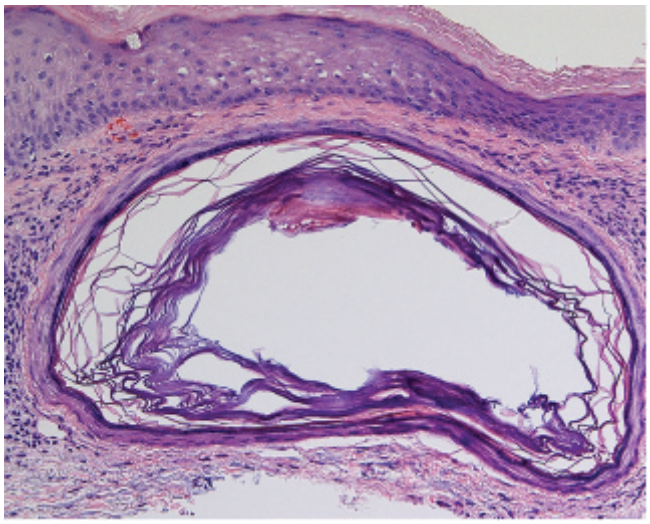

Figure 4. Classically, milia are identical to the larger infundibular cysts encountered commonly. In essence, they mimic the epidermal surface; their epithelium matures through a granular layer and produces orthokeratotic corneocytes in a basket-weave pattern. Lymphocytic inflammation is present around the milium (hematoxylin and eosin staining, ×200 original magnification).

(the allergen in oak moss) during an abortive attempt at repeated open application testing.[4] He had not developed milia from prior patch testing. Inman reported on a 45-year-old woman who developed milia on the forearm after developing ACD from ichthammol, which had been used to treat phlebitis.[5] Finally, Tolman reported on a 29-year-old nurse with milia on her dorsal hands after she

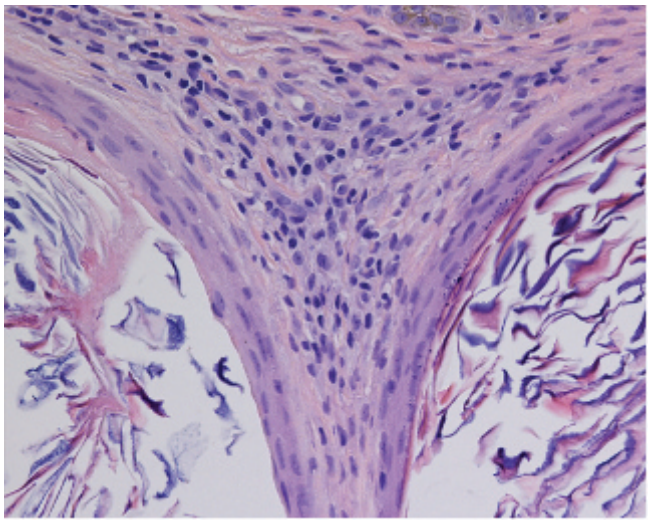

Figure 5. Some variation in maturation can occur in milia. For instance, the milium on the left lacks a granular layer while the one on the right maintains a somewhat diminished granular layer. However, both milia contain orthokeratotic "flaky" corneocytes. Lymphocytes and macrophages are present in the pericystic dermis (hematoxylin and eosin staining, ×400 original magnification).

developed contact dermatitis from holding a patient's flowers.[6]

Few studies have investigated the pathogenesis of secondary milia.[7–10] Based on studies that used serial sectioning, immunohistochemical staining, or both, primary milia seem to derive from the lower infundibula connected to vellus hair follicles. In contrast, secondary milia seem to usually derive from eccrine ducts (probably in syringometaplasia) rather than from the overlying epidermis, hair follicles, or sebaceous ducts, although this may depend on whether the milia are secondary to blisters or trauma and on the particular type of blisters or trauma. Autoimplantation has also long been thought to play a role in the pathogenesis of at least a subset of milia, those milia presumably deriving from the epidermal surface rather than from adnexa.[7]

In previous descriptions of milia that occur after ACD, none of the authors attempted to explain the pathogenesis of the milia, although several authors suggested that the occurrence of milia was an indicator of the ''severity and depth'' of the ACD.[3,4] We propose two hypotheses. The first hypothesis is that milia may follow severe ACD because of disruption of the dermoepidermal junction in association with advanced spongiotic vesiculation or a ''dermal ACD'' with marked subepidermal edema (or both). A second explanation is that the milia in these cases result not from the injury of the ACD but rather from autotransplantation of epithelium because of the patient's subsequent excoriation in response to intense pruritus. In support of the second hypothesis, the biopsy specimen from our case 2 shares similarities with the many biopsy specimens reported in the phenomenon known as ''prayer nodules,'' a condition likely due to repeated pressure and trauma described in elderly rural Shiite Muslim men whose foreheads have contact with prayer stones (mohrs) many times daily for decades.[11,12] Similar to the histopathologic findings of prayer nodules, the biopsy specimen from the case 2 patient contained numerous small dermal cysts lined by stratified squamous epithelium with laminated corneocytes, surrounded by a conspicuous mononuclear inflammatory infiltrate. Unfortunately, it is difficult to compare this to the findings from other reported cases of milia that occurred after ACD because in only one of the previously described cases in the literature was a biopsy performed.[5] Although no histologic images were published in that case report of milia occurring after ACD, the authors noted that a biopsy specimen revealed numerous milial cysts and a ''fairly heavy predominantly lymphocytic infiltrate...in the upper dermis,'' consistent with our biopsy findings.

Our cases show that milia are a rare complication of severe ACD. These cases demonstrate the unusual association of two very common disorders in causative relation.

Acknowledgments

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