Letter to Editor

Papular elastorrhexis localized to the wrist, the intravenous infusion drip site

Sir,

Papular elastorrhexis (PE) is a rare acquired cutaneous disorder that usually appears in the second decade of life with a predilection for women. It is characterized by multiple asymptomatic, nonfollicular, nonconfluent, flesh-colored or hypopigmented, monomorphous papules between 1 and 5 mm in size, with symmetrical distribution on the trunk and proximal upper limbs. In addition, this condition usually occurs without any previous history of trauma, acne, inflammation, or infection.[1] Herein, we report an unusual and interesting case of PE confined to the wrist and associated with antecedent trauma, a needle injection for an intravenous infusion drip, in a middle-aged woman.

A 54-year-old woman presented with a 1-month history of several flesh-colored papules on the wrist [Figure 1a]. She denied a history of any other inflammatory skin conditions of the involved area. However, she mentioned that the area had been injected with intravenously for an infusion drip. There was no family history of similar lesions or other skin diseases. A representative biopsy specimen from a lesion revealed a normal epidermis and a focal area of homogenized collagen in the dermis [Figure 1b and c]. Verhoeff–Van Gieson staining demonstrated decreased elastic fibers, and the residual elastic fibers appeared thin and partially fragmented [Figure 1d]. No treatment was introduced because of the asymptomatic character of the lesions and the absence of cosmetic concern. The lesions persisted during the follow-up of 6 months.

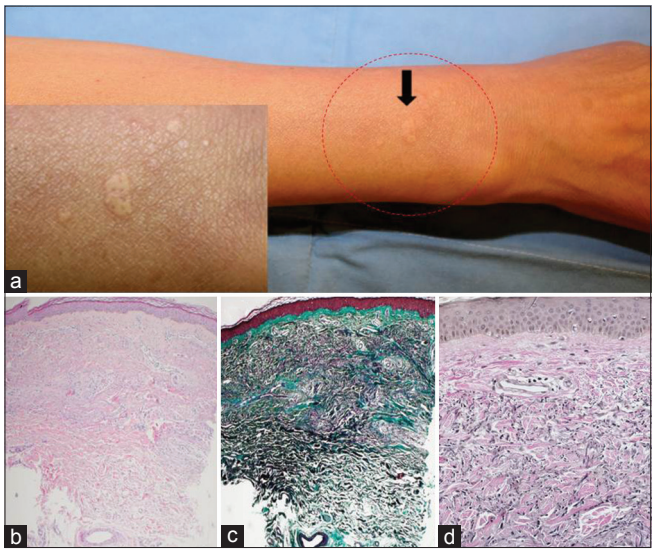

Figure 1: (a) Several asymptomatic, nonfollicular, flesh-colored papules on the right wrist. The arrow indicates the injection site for the intravenous infusion drip. (b and c) Histologic examination showing a normal epidermis and homogenized collagen in the dermis (b; H and E, ×40, c; Masson trichrome, ×40). (d) Verhoeff–Van Gieson staining showing decreased elastic fibers in the dermis, which appeared thin and fragmented (×100)

PE, first described by Bordas *et al*.[2] in 1987, is an uncommon disorder of the elastic tissue, characterized by substantial fragmentation of the elastic bundles rather than simply decreased elastic fibers in the reticular dermis.[1] The latter is the most prominent feature of nevus anelasticus, one of the differential diagnoses of PE, which also include acne scarring, perifollicular elastolysis, collagenoma, mid-dermal elastolysis, and anetoderma.[1] On the basis of the lacking LEMD3 gene defect and decreased rather than increased number of elastic fibers, PE has been classified as a distinctive entity, different from Buschke–Ollendorff syndrome.[3,4]

In our patient, the negative family history, clinical features of the lesion, acquired outbreak, absence of other extracutaneous abnormalities, and histological evidence of elastic fiber fragmentation were consistent with PE. However, the features of our case that differed from typical PE were as follows: (1) The presence of antecedent trauma, (2) the location and asymmetric distribution of the lesions, and (3) the late onset. Some previous reports have described skin lesions appearing on an atypical site or in the fifth decade of life.[5] However, there are no cases of PE accompanied by a previous trauma history.

The pathogenesis of PE is still largely unknown.[1] In addition, no reports in the literature directly address the debate as to whether a history of trauma is an absolute exclusive criterion. Further reports or larger studies for analysis of the etiology and pathogenesis are needed to answer this question.

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Conflicts of interest
There are no conflicts of interest.

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