To the Editor:

The presence of numerous acquired comedones on the trunk and limbs of an adult patient, with no other family member affected and with no triggering factors to explain their presence, requires us to rule out those conditions in which comedones are the predominant clinical feature. The fact that these lesions appear in association with areas of lichen ruber planus presents the dilemma of whether the lesions develop simultaneously by chance or whether there could be some pathogenic relationship. In the case we present below, we observed that, from a clinical point of view, the 2 types of lesion (comedones and lichen) were frequently closely related, with the comedones presenting a lichenoid halo, but they were sometimes separate. However, in all samples analyzed, histological study showed lichenoid infiltration of the comedones.

A 72-year-old woman came to our outpatient clinic for mildly pruritic lesions that had been present on the trunk and limbs for approximately 2 years. The patient had a history of hypertension and dislipidemia, for which she had been on treatment with felodipine and pravastatin for at least 7 years. There was no family history of similar skin lesions. On physical examination, we observed small, shiny, erythematous-violaceous plaques and papules with a smooth surface, suggestive of lichen ruber planus (Figure 1). In addition, numerous comedones were identified on the trunk, arms, and thighs; some of these had an erythematous halo of lichenoid inflammatory appearance (Figure 2). A whitish, reticulated pattern was observed on the mucosa of both cheeks. There were no alterations of the hair or nails. The patient was unable to say whether the 2 types of lesion appeared simultaneously or in succession, and there was no history of the use of comedogenic products.

We performed biopsies of the comedones, with the interesting finding of a lichenoid pattern around the infundibular microcysts of the comedones (Figure 3); this infiltrate was observed irrespective of whether there was a visible erythematous halo around the comedone or not. The biopsy from the violaceous plaques showed a linear lymphoid infiltrate in the superficial dermis and the presence of necrotic keratinocytes, also confirming the clinical impression of lichenoid lesions. There were no relevant pathological findings in the routine laboratory tests and serological tests for viral hepatitis were negative. The patient was treated with oral isotretinoin (0.5 mg/kg) for 4 months, with resolution of the lichenoid lesions and a marked reduction in the number of comedones.

The presence of numerous comedones with a generalized distribution on the trunk and limbs, appearing in adult life, with no triggering factors and no family history of similar lesions is very rare. Recently, Zhang and Zhu1 reported a similar case in a boy who, like our patient, presented disseminated idiopathic comedones. The histological...
findings, the distribution of the lesions, the age at presentation, and the absence of triggering factors led us to rule out similar conditions known at the present time, such as familial dyskeratotic comedones, comedo nevus, follicular keratosis, physically or chemically induced acne, and follicular mycosis fungoides.

Interestingly, our patient presented a lichenoid dermatitis of the lichen planus type; the cause was unknown as she did not report any associated systemic symptoms and the complementary tests performed were normal or negative. We cannot totally exclude a relationship between these lesions and the treatment that was being taken; in fact, 2 cases of lichenoid reaction related to pravastatin have been reported in the literature.2,3 However, in our case, this seems very unlikely due to the absence of any temporal relationship and due to the presence of lesions on the mucosas, which are uncommon in drug-induced lichenoid reactions.

Up to now, the relationship between lichen planus and comedones has been limited to cases of lichen planus follicularis tumidus with cysts and comedones, clinically characterized by edematous plaques with cysts and comedone-type lesions, usually in a retroauricular position.4,6 The involvement of the pilosebaceous unit in lichen planus is observed in cases of lichen planus follicularis, which typically affects the scalp, although and there has been 1 case report in which the trunk was affected.7 However, the clinical presentation in those cases, so different from what is described here, makes us think that there could be no relationship. Lucke et al8 described the development of milia-like cysts during the course of lichen planus. Like those authors, we considered the possibility that the comedones formed due to inflammation or degeneration of the basal layer caused by the lichen; however this hypothesis appeared unlikely in our case, as many of the comedones were found in skin with no previous lichenoid lesion. Independently of the temporal relationship between the 2 types of lesion, we believe it is more likely that the lichenoid infiltration of the comedones was secondary, and it could even be a typical occurrence in lichen planus. In the literature, there have been sporadic reports of lichenoid infiltration of similar structures, such as of an epidermal cyst in a patient with lichen planus.9 Although the excision of benign lesions is not common practice in an inflammatory disorder, histological study of this type of lesion in patients with lichen planus could corroborate this hypothesis.

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Conflicts of Interest
The authors declare no conflicts of interest.

References

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Figure 3. A lymphoid infiltrate with a clearly lichenoid pattern was observed around the infundibular microcysts of the comedones. Hematoxylin–eosin, x10.