A 26-year-old Caucasian male patient, known to have Crohn’s disease, presented to our clinic for evaluation. Examination of the skin revealed multiple annular, hyperpigmented, centrally atrophic lesions, localized over the anterior scalp, forehead, and right temporal area, forearms, and dorsum of the hands. Upon questioning, the patient reported no fever, myalgia, arthralgia, abdominal pain, nor throat pain prior to the onset of the cutaneous lesions. Adalimumab was suspended.

Three months after the onset of skin lesions, the patient presented to our clinic for evaluation. Examination of the skin revealed multiple annular, hyperpigmented, centrally atrophic lesions, localized over the anterior scalp, forehead, and right temple with no associated alopecia. Flat topped, polygonal, papular and annular, erythematous-to-violaceous lesions localized on the dorsal aspect of both forearms and hands were noted (Fig. 1). Koebner phenomenon was present secondary to itching over the right forearm. Patient had no nail or mucosal lesions. Two skin biopsies were taken from the forearm and dorsal hand lesions for histopathologic evaluation. Histopathologic findings included irregular epidermal acanthosis, compact orthokeratosis, hypergranulosis, and scattered necrotic keratinocytes. A superficial dermal perivascular lichenoid lymphocytic infiltrate with few eosinophils, encroaching upon the dermo-epidermal junction with vascular alteration was noted (Fig. 2). There was no histologic features suggestive of cutaneous lupus. These findings were compatible with lichenoid drug eruption.

Knowing that the patient was only taking adalimumab prior to the appearance of these skin lesions, a causal correlation between adalimumab administration and the appearance of the lichenoid lesions was assumed.

Patient was put on oral prednisone at a dose of 1 mg/kg per day tapered over 12 weeks, desloratadine per os, and clobetasol propionate cream.
TNF-α inhibitors have been previously described in the literature as a treatment modality for many cutaneous inflammatory diseases, including lichen planus [6-7]. Despite being effective in treating immuno-mediated conditions, TNF-α inhibitors have been associated with the induction of autoimmune cutaneous phenomena due to an uncontrolled production of interferon-α by plasmacytoid dendritic cells [8-9]. Adalimumab has been implicated in many cases of drug-induced lichen planus. One report described the occurrence of a mucosal lichenoid drug eruption one month after the initiation of adalimumab for treatment of psoriasis [10]. Asarch et al. also reported a case of mucocutaneous lichen planus-like lesions 16 months after initiating adalimumab for the treatment of psoriasis [11]. Another report by Flendrie et al. described the occurrence of a lichen-planus like eruption sparing the mucous membranes, three weeks after the initiation of adalimumab for treatment of rheumatoid arthritis [5].

Differentiating between lichenoid drug eruption and idiopathic lichen planus can be evident clinically and on histopathology. Lesions in lichenoid drug eruption are more generalized, photo distributed, and spare the classic sites of idiopathic lichen planus, mainly the mucous membranes [12]. Wickham’s striae that are frequently
present in idiopathic lichen planus are absent in lichenoid drug eruption. Lesions in idiopathic lichen planus appear to be shiny, flat-toped, polygonal, and violaceous. The main difference on histopathology is the presence of parakeratosis in lichenoid drug-induced eruptions which is absent in cutaneous idiopathic lichen planus. Varying degrees of eosinophilic and/or plasma cell infiltrates may also be present in lichenoid drug eruption (Table I) [12]. The main diagnostic clinical feature is the disappearance of the lesions after the cessation of the culprit drug [10]. Our patient had mild improvement after three months of cessation of adalimumab. In a review article by Asarch et al. seven out of the nine patients who stopped the TNF-α inhibitor after the appearance of the lichenoid drug eruption had complete recovery. The remaining two patients had mild-to-moderate improvement [11].

In conclusion, lichenoid drug eruption and idiopathic lichen planus have different clinical and histopathological features. In the majority of the cases, lesions clear after stopping the culprit drug. However, the diagnosis of TNF-α inhibitors induced lichenoid drug eruption should not be ruled out in the presence of other clinical and histopathological features, even if the lesions do not disappear few months after stopping the anti-TNF therapy.

REFERENCES