Atypical Nevi in a Patient After Toxic Epidermal Necrolysis

Dear Editor,

There are few literature data about nevi in patients with a history of toxic epidermal necrolysis (TEN) and little recommendations for follow-up and risks of melanoma (MM). Eruptive melanocytic nevi (EMN) is a rare phenomenon that has been associated with bullous disorders, immunosuppression, and immunodeficiency, but in some cases can occur without precipitating factors (1). The etiology is largely unknown, but there is evidence that immunosuppression might play a crucial role in nevogenesis, probably due to the inability of the immune system to inhibit melanocytic (MC) proliferation (2,3). We report the follow-up of a patient with a history of TEN who later developed atypical nevi.

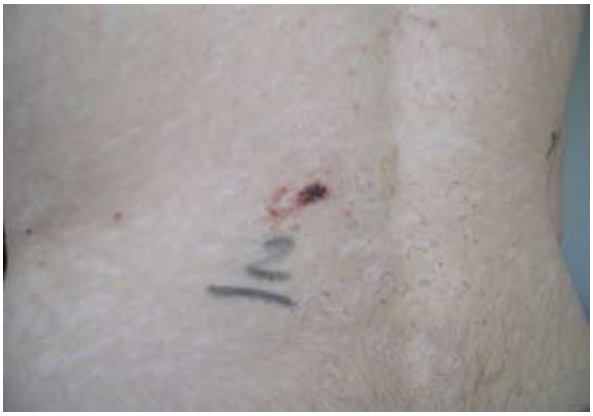

Figure 1. Detail from the skin of the patient on the back, with scarring and postinflammatory residual scattered hyper- and hypopigmentation, and atypical nevus.

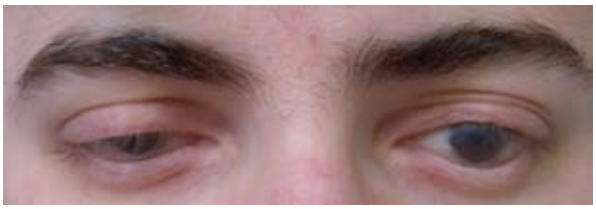

Figure 2. Patient presenting with symblepharon.

A 17-year-old man with a history of severe TEN two years earlier, most probably due to valproic acid and diclofenac, was referred to our Department due to atypical nevi. The patient presented with scars, scattered pigmentation (Fig. 1), and symblepharon as a consequence of TEN (Fig. 2). Most of his nevi developed in following two years after TEN. During the first visit in 2009, clinical and dermoscopic photodocumentation was performed. The patient presented with a moderate number of nevi (Fig. 3), dermoscopically subclassified as globular. One atypical MN was found on the back, with dermoscopic findings of reticular pattern and presence of suspected areas of regression (Fig. 4. a, b), and it was excised to rule out melanoma (Fig. 4, Fig. 5). The patient did not come to regular follow-up from 2009 to 2014, and presented in 2014 which was when comparative photo documentation was made. As this visit another, speckled type of newly-occurred nevus was excised. Both excised nevi were histopathologically characterized as dysplastic.

Only a few references are available on nevi development after TEN. Anticonvulsives and NSAIDs, as in our case, are often involved in the etiopathogenesis of TEN (4,5). Survivors may experience a variety of long-term complications; authors reported

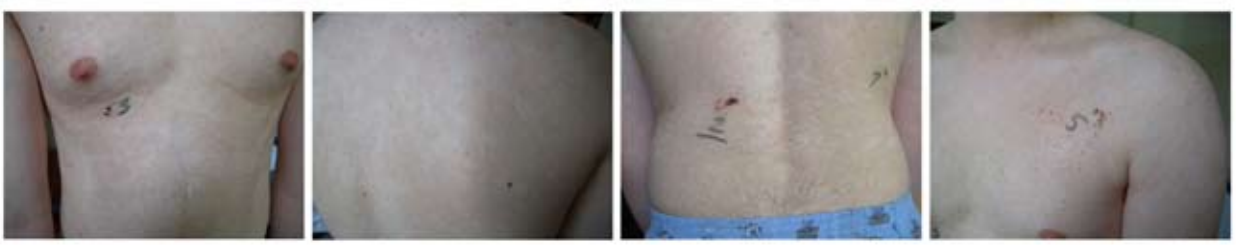

Figure 3. Patient presented with few to a moderate number of nevi.

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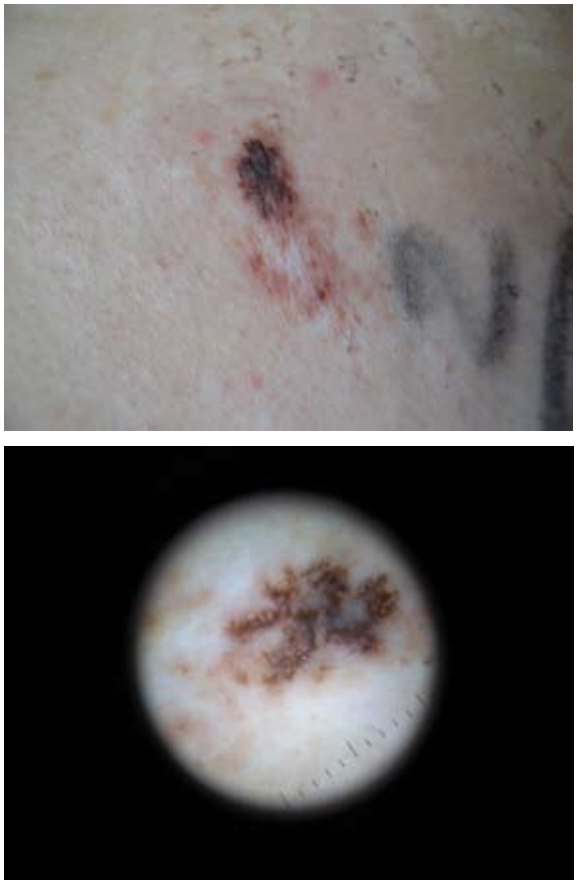

Figure 4. (a, b) Atypical nevus on the back of the patient, clinical and dermoscopic view. Dermoscopy showed combination of reticular structure, nonspecific areas of erythema, peppering and milky red color. Excision was made due to need to rule out melanoma (Delta Heine, ×10 magnification, Nikon Coolpix). Biopsy showed nests of nevus cells in the basal layers of the epidermis, merging of a few nests, and lymphocytic response around the blood vessels in the papillary dermis. Consequently, diagnosis of dysplastic nevus was established.

that 19% of their patients developed new nevi after TEN (6,7). EMN develop several years after TEN as a suddenly arising large number of nevi that may resemble speckled lentiginous nevi (8). Histologically. EMN demonstrate a proliferation of MC at the dermoepidermal junction and, if compound, in the papillary dermis, arranged mostly in nests. Junctional MC may appear slightly pleomorphic, but no significant cytological atypia or prominent pagetoid spread of MC was reported (9). EMN have been associated with a specific dermoscopic finding of a symmetrical peripheral rim of globules which represent pigmented junctional nests of MC in the periphery and are a specific feature of rapidly enlarging MC nevi (10,11). The pathogenesis of EMN is not known. The microenvironment of epidermal regeneration may have some effects on MC because MC hyperplasia develops after cutaneous trauma (observed in recurrent nevi). The cytokines and growth factors produced and secreted during epidermal regeneration might contribute to the proliferation of residual epidermal MC and subsequent nevus formation (12). Because most of the bullous disorders associated with EMN are transient, the authors believe that changes in local growth factors may also be temporary and MN remain stable without a propensity to malignant degeneration without further stimuli (13). This is corroborated by the fact that no reports of malignant change of EMN in patients with bullous disorders have been described. It is likely that the etiology and natural course of EMN differs between two main populations of patients, with EMN arising after bullous disorders being more likely to remain benign compared with those with ongoing immunosuppression, but this hypothesis has yet to be proven. The actual risk of MM in patients with EMN remains unknown. Since our patient did not have many nevi, he does not fit into the EMN category. Due to the atypical appearance of his nevi, long-term follow-up on 6-month basis is recommended.

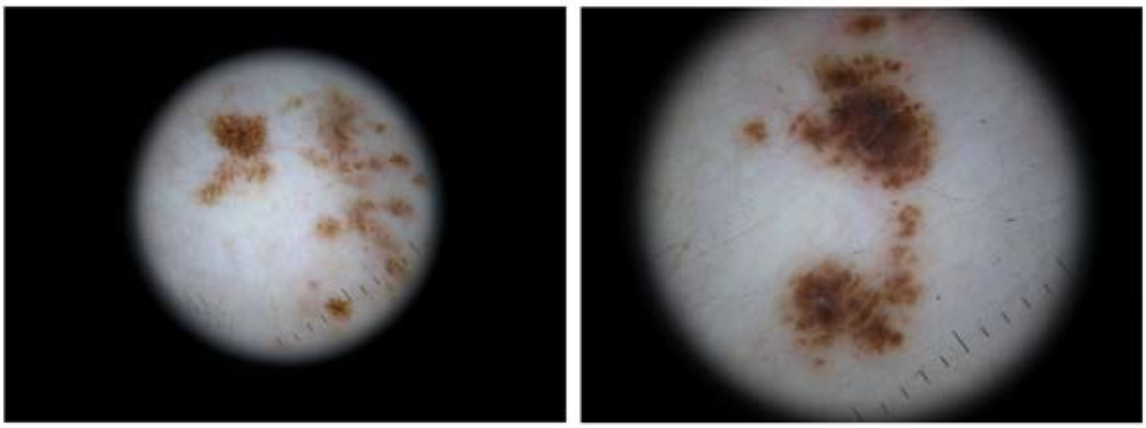

Figure 5. Speckled appearance of nevus, dermoscopically a combination of globules and nonspecific structure arranged in a speckled way, histopathologic diagnosis of dysplastic nevus was established after excision.

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