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PROGRESSIVE AND EXTENSIVE PITYRIASIS ALBA: SAME DISEASE, DIFFERENT NAMES?
Editor

We read with much attention the article by di Lernia and Ricci (2005) as for many years now we have been greatly interested in the pigmentary disorder called progressive and extensive hypomelanosis and which we indicate as progressive macular hypomelanosis, according to Guiliet et al. (1988). We have the following comments:

(1) In 1987 Borelli (1987) published an article about a new disorder with hypopigmentations on the trunk, which he named ‘cutis trunci variata’. Comparison of his clinical description with that of Guiliet et al. (1988) suggests that the same disorder is described in these two publications. Therefore, it appears that Borelli, and not Guiliet et al. was the first to publish this disorder.

(2) Clinical description and light microscopy suggest that extensive pityriasis alba described by Zaynoun et al. (1983) and progressive macular hypomelanosis described by Guiliet et al. (1988) might indeed be the same disorder. However, we want to point out that the electron microscopic findings are different. The electron microscopic investigations conducted by Zaynoun et al. (1983) on nine patients with extensive pityriasis alba showed reduced melanocytes and those present contained fewer and smaller melanosomes. Furthermore, a reduction in the density of functional melanocytes in the affected areas without any change in cytoplasmic activity was observed. Melanosomal transfer to keratinocytes was generally not disturbed. They concluded that the hypopigmentation might thus be primarily a result of the reduced numbers of active melanocytes and a decrease in number and size of melanosomes in the affected skin. Histological investigations by Guiliet et al. (1988) showed a slight reduction of melanin granules in the basal cell layer with a variable decrease of melanin transfer to keratinocytes. Ultrastructural investigations showed a switch from stage IV single melanosomes (these are the types of melanosomes normally seen in black skin) in the healthy looking skin to small type stage I–III aggregated melanosomes (these are the types of melanosomes normally seen in white skin) in the hypopigmented spots.

(3) If extensive pityriasis alba is indeed identical to progressive macular hypomelanosis, we advise that the first name is abandoned. The term pityriasis alba is generally accepted to indicate a disorder with minimal eczematous characteristics (Weedon 2002). These are not present in progressive macular hypomelanosis. Extensive pityriasis alba is therefore a misnomer.
(4) We have recognized hundreds of patients of both sexes and different ethnicities with progressive macular hypomelanosis in the Netherlands since the early eighties of the last century. Since 1989 we have presented our findings at several international and Dutch meetings and have also published these findings. (References are available on request).

(5) In 2004 we proposed the hypothesis that progressive macular hypomelanosis is caused by *Propionibacterium acnes* (*P. acnes*), based on clinical and microbiological investigations (Westerhof *et al.* 2004). Studies on PMH patients by Wood’s lamp in a dark room showed red follicular fluorescence in lesional skin, which was not present in normal skin. Red fluorescence is also seen in patients with acne and represents excitation of porphyrins produced by *P. acnes* bacteria. Furthermore, bacterial cultures of hair follicles in lesional skin in seven out of eight patients with PMH were positive for *P. acnes* while bacterial cultures of non-lesional skin in the same patients showed no bacterial growth. This proves that PMH is a distinct disease entity with a separate aetiopathogenesis. Based on these observations we soon hope to publish the results of a clinical trial concerning the treatment of the disease, which so far is intractable.