Linear Psoriasis and ILVEN: Is Lumping or Splitting Appropriate?

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In this issue, Thomas Hofer [1] presents an unusual case of linear psoriasis superimposed on psoriasis vulgaris. The author is discussing the nosological problem of whether ILVEN and linear psoriasis are different entities. Hofer favors the concept that all cases reported under either name should be taken as a mosaic manifestation of psoriasis vulgaris. In other words, ILVEN would always represent a mosaic form of psoriasis. Although a similar view of lumping was held in the past by some other authors [2], most experts today prefer to be ‘splitters’.

I likewise advocate splitting of the two disorders. ILVEN is usually far more itchy and tends to be unresponsive to the classical, time-honored methods of antipsoriatic treatment. Admittedly, it may sometimes be difficult or even impossible to distinguish the disorders on purely clinical grounds, but in most cases a definite diagnosis of either disease can be made.

For example, the case presented by Hofer [1] fulfils all of the clinical criteria of linear psoriasis [3]. A grandfather of the patient had had psoriasis vulgaris since his youth. The linear lesions appeared rather early, and a relapse, apparently triggered by a sore throat, was associated with disseminated psoriatic plaques. The linear inflammatory disorder responded to antipsoriatic treatment with tacalcitol, whereas the disseminated lesions cleared spontaneously. Hence, I think that a diagnosis of ILVEN is not appropriate.

In contrast to Hofer’s view, I propose to distinguish the following 3 categories: (1) cases that undoubtedly represent linear psoriasis [4–8]; (2) cases that undoubtedly

represent ILVEN [9–12]; (3) cases that can so far not be categorized with certainty. In such doubtful cases, immunohistochemical techniques may perhaps be helpful. For example, Vissers et al. [13] found an increased keratin 10 expression in ILVEN but not in psoriasis.

Furthermore, a word of caution seems appropriate regarding the use of the terms ‘segmental type 1 mosaic’ and ‘segmental type 2 mosaic’. It should be borne in mind that these terms only apply to monogenic skin disorders showing a Mendelian mode of inheritance in the form of autosomal dominant transmission [14]. In a polygenic trait such as psoriasis it is not possible to discriminate between a ‘genetically healthy’ and a ‘heterozygous’ embryo, which is why we cannot distinguish between a ‘segmental type 1’ and a ‘segmental type 2’ manifestation of psoriasis [15].

In order to avoid these terms in that context, I propose to simply distinguish between *linear psoriasis of the isolated type*, in which disseminated lesions of psoriasis vulgaris are constantly absent, and *linear psoriasis of the superimposed type*, in which a pronounced linear involvement is, either intermittently or constantly, associated with psoriasis vulgaris. Both clinical types may originate either from early loss of heterozygosity [15] or from any other mutation occurring at one of the many gene loci involved in psoriasis.

In conclusion, although I raise my voice of polite dissent with regard to the nosological classification, I highly recommend to read Hofer’s interesting report because it gives us food for thought, and thoughts for further discussion.

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