

Connections between atopic dermatitis and ageing

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Not so long ago, looking for connections between old age and atopic dermatitis (AD) would have been peculiar, as AD was considered a disease primarily of infancy and childhood that resolves in more than 50% of patients up to 10 years of age. Lately, our opinion about this disease has changed substantially owing to results of epidemiological, genetic, immunological and dermatological research and some data even support the concept that once developed, AD should be considered a lifelong disease.^{1,2} The high prevalence (it is the most common noncommunicable dermatosis), the previously mentioned lifelong occurrence, the lack of diagnostic and prognostic biomarkers and the difficulty of finding efficacious therapies that suit all patients indicate that it is not just one homogeneous disorder.³ AD rather can be considered as a spectrum of diseases with heterogeneous clinical characteristics, disease course and possibly distinct pathogenesis with varying therapeutic consequences.⁴ The characterization of subtypes of AD is going to be the main task of dermatologists and immunologists in the near future.

According to our present knowledge, on the basis of age-related clinical characteristics, AD can be stratified into four different kinds: infantile, childhood, adolescent/adult and elderly forms, the latter form seems to be rather underestimated. On the other hand, classification according to the natural history of the disease has identified six different types of onset. The very late-onset form (in those >60 years old) has been added more recently and seems to represent a subgroup of increasing significance.⁵

Currently our knowledge about the elderly type of AD is rather insufficient.^{6,7} According to studies, 2–3% of the elderly population is affected by AD, which seems to be strongly pruritic in older-age groups. At the same time, these numbers may be inaccurate because of the lack of clear diagnostic criteria and specific guidelines for dermatologists to distinguish AD from other pruritic dermatoses in elderly people. Another problem is that recommendations about specific treatments for AD in this age group, considering the frequent comorbidities such as hypertension, diabetes or risk of infections, are also lacking.

All these insufficiencies underline the importance of research articles and reviews on this topic. The article of Williamson and colleagues, published in this issue of the *BJD*, covers more than just a summary of our knowledge about AD in elderly people.⁸ They also review the most important data on the barrier alterations in ageing skin, and data on the

dysregulated immune system in older age, characterized by chronic inflammation and also referred to as 'inflammaging'. They indicate that defects in the skin barrier and the immune system are very similar to those that are observed in AD. Although they emphasize that similarities do not mean that these changes, if present, are mechanistically involved in the pathogenesis of elderly AD, their suggestion that ageing itself may be considered as a risk factor for this disease, or at least can worsen its course, seems an important and rational opinion. Specific studies are needed to further confirm causality between changes in skin and immune system characteristics in elderly people and between the pathogenesis and development of elderly type AD.

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Conflicts of interest

None to declare.

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