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Review

Diagnosis and classification of psoriasis

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ABSTRACT

Psoriasis is a chronic inflammatory multi organ disease with well characterized pathology occurring in the skin and often the joints. Although the disease has many characteristic and even pathognomonic features, no established diagnostic criteria exist for cutaneous psoriasis and there is no unified classification for the clinical spectrum of the disease. Prior approaches that have been taken to classify psoriasis include age of onset, severity of the disease, and morphologic evaluation. The latter has yielded plaque, guttate, pustular, and erythrodermic as subtypes of psoriasis. Unlike other autoimmune diseases, histopathological examination and blood tests are generally not valuable tools in making the diagnosis of psoriasis. However, on occasion, dermatopathologic evaluation may be helpful in confirming the diagnosis of psoriasis. Thus, in most cases the diagnosis of psoriasis is dependent primarily on pattern recognition that is morphologic evaluation of skin lesions and joints.

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Contents

1. Introduction

Psoriasis, a chronic skin disease is known to be the most prevalent autoimmune disease in humans [1,2]. Approximately 2%–5% of the world's population suffers from psoriasis [1,2]. According to the National Institutes of Health (NIH), as many as 7.5 million Americans have psoriasis[2]. The precise etiology of psoriasis remains poorly understood,

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but psoriasis is thought to result from a complex interplay between genetics, environment, skin barrier disruption, and immune dysfunction [1,3,4].

There is no cure for the spectrum of psoriatic diseases, which comprise various different subtypes of psoriasis and psoriatic arthritis. Psoriasis is associated with a high degree of morbidity and the current medications can have severe side effects. Several clinical studies have reported a strong association between psoriasis and atherosclerosis, type 2 diabetes, and metabolic syndrome, together the leading causes of mortality in the Western world [5]. In addition, patients with psoriasis, like those with other major medical disorders, have reduced levels of employment and income as well as a decreased quality of life [6,7]. The combined costs of long-term therapy and social costs of the disease have a major impact on health care systems and on society in general. According to the National Psoriasis Foundation, USA, total annual direct and indirect costs of psoriasis are over US $11 billion, with missed workdays accounting for 40% of the cost burden [2].

The focus of this article is to discuss the clinical spectrum of psoriasis and how to diagnose the protean manifestations of psoriasis. An expert dermatologist should be able to diagnose plaque psoriasis and other clinical variants of psoriasis by evaluating their characteristic morphologic appearance. Neither histopathological examination nor any specific blood tests are recommended for the diagnosis of psoriatic disease. However histopathological and immunohistochemical features of psoriasis are unique and on rare occasions clinicopathological correlations may be helpful in diagnosis of psoriasis.

2. Pathogenesis and the pathology of psoriasis

In this section we will briefly review the histopathological features (Figs. 1 and 2) and immunopathogenesis of psoriasis. Both psoriasis and psoriatic arthritis (PsA) are complex genetic diseases. The genetic basis of psoriasis is supported from evidence from family and twin studies, linkage studies, and population-based association studies. These studies illustrate the importance of both keratinocytes and the immune system in the pathophysiology of psoriasis. It has been reported that NF-kB mediated responses in the skin are directed by CARD14 and that a subset of rare CARD14 variants leads to psoriasis and psoriatic arthritis [8,9]. Elder and his colleagues have reviewed the SNP analyses of several major studies in this field to provide collective information about an association between psoriasis and various loci of the immune system, such as the TH17 pathway (*IL12B*, *IL23A*, *IL23R*, *TRAF3IP2*, *TYK2*), innate immunity [NFκB and IFN] signaling pathways (*TNFAIP3*, *TNIP1*, *NFKBIA*, *REL*, *TYK2*, *IFIH1*, *IL23RA*) and βdefensin, the TH2 pathway (*IL4*, *IL13*), and adaptive immunity involving CD8 T cells (*ERAP1*, *ZAP70*) [10].

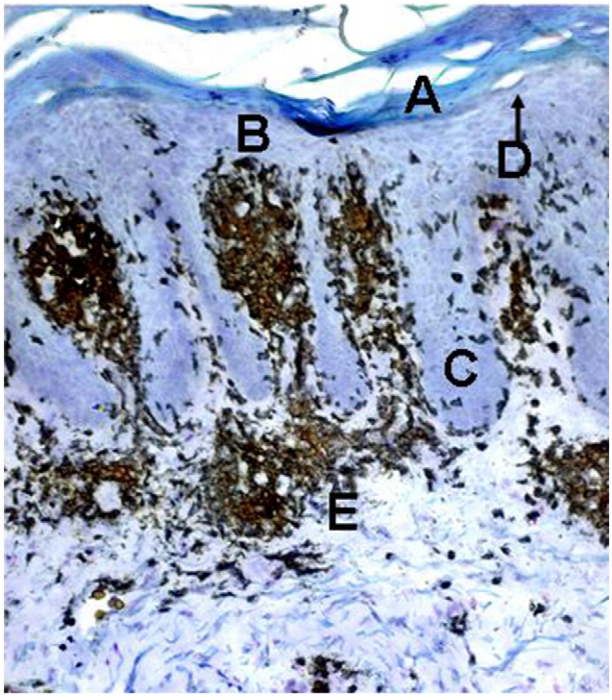

Fig. 1. A psoriasis plaque stained with an anti-CD3 antibody and counter stained with hematoxylin demonstrates unique histological features of psoriasis — A. Parakeratosis. B. Suprapapillary thinning. C. Elongated rete pegs. D. Loss of stratum granulosum (marked by the arrow). E, Diffuse infiltration of $CD3^+$ T cells in the deep and superficial dermis along with the epidermotropism of $CD3^+$ T cells.

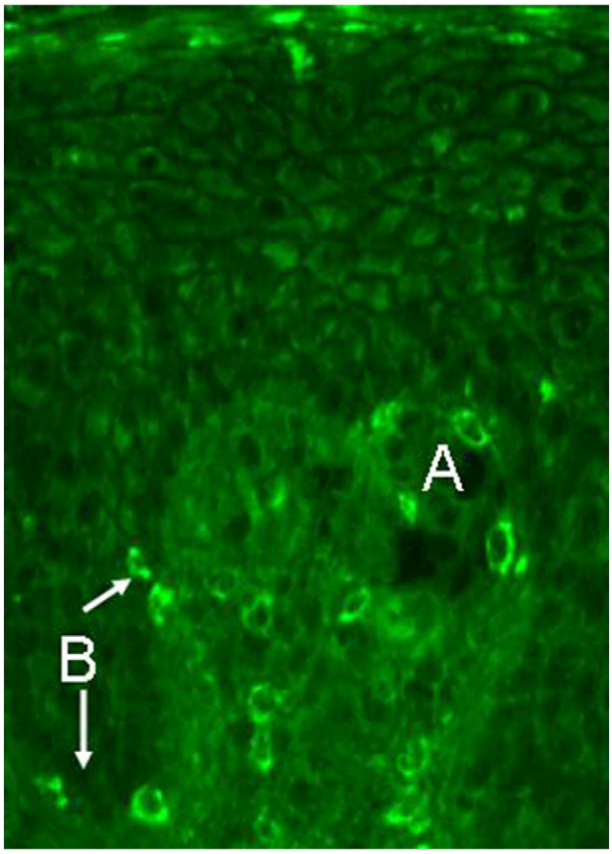

Fig. 2. A psoriasis plaque stained with an ant-CD8 antibody (immuno fluorescence microscopy) demonstrates epidermotropism of significant number of $CD8^+$ T cells (A, B).

Hyperproliferation and abnormal differentiation of keratinocytes are the two critical outcomes of the underlying patho-physiologic dysregulation in psoriasis. Histologically, there is marked thickening of the epidermis due to increased proliferation of keratinocytes in the interfollicular epidermis, and epidermal rete peg become very elongated [11–15]. Keratinocyte differentiation is also extensively altered in psoriasis, paralleling 'regenerative maturation,' an alternative cell differentiation program that is transiently expressed during wound repair. The granular layer of the epidermis, in which terminal differentiation begins, is greatly reduced or absent in psoriatic lesions [12–15]. Consequently, a stratum corneum forms from incompletely differentiated keratinocytes that aberrantly retain a cell nucleus (parakeratosis).

An active role of T cells in the pathogenesis is strongly substantiated by the following observations: (i) immunotherapy targeting the T cells or T cell cytokine such as IL-17 clears active plaques of psoriasis [16–20] and (ii) in SCID mice, transplanted nonlesional psoriatic skin converts to a psoriatic plaque following administration of intradermal administration of antigen-activated T cells [21]. In addition to traditional inflammatory mediators, experiments in the SCID mouse model, have demonstrated that Nerve Growth Factor (NGF) and substance P (SP) activated lymphocytes can also convert nonlesional psoriatic skin transplants to psoriasis [22]. Thus cytokines (Th1,Th17 and Th22), chemokines, adhesion

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