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Evaluation of Serum Human B-Defensins in Psoriasis Vulgaris Patients

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Abstract

Background: The overproduction of keratinocytes is a hallmark of the inflammatory skin condition known as psoriasis. The etiology of psoriasis is complex and includes both hereditary and environmental variables, including cigarette smoking, drug use, emotional and mental stress, trauma, and infections. Mucosal membranes are protected against microbes by a class of antimicrobial peptides called human beta-defensins (hBDs, −1, 2, 3). In addition to their antibacterial properties, they have other functions, such as cell activation, proliferation, migration, differentiation, angiogenesis, wound healing, and modulation of cytokine/chemokine production. The purpose of this article is to examine the relationship between the severity of psoriatic illness and the levels of human β-defensins in the serum. In conclusion, psoriasis may be influenced by serum levels of beta-defensin-2 and beta-defensin-3. Its potential as a biomarker of disease severity is shown by the association between human beta-defensin-2 and beta-defensin-3 levels and the severity of psoriasis.

Keywords: Topics covered include psoriasis and beta-defensins from humans.

Introduction

Inflammatory skin illness known as psoriasis is characterized by an overabundance of skin cells called keratinocytes (1). Drugs, smoking, emotional stress, trauma, infections, and genetics are all contributors to its complex etiology (2).

Because of the complex interplay between hereditary and environmental variables, the exact pathophysiology of psoriasis remains a mystery (3).

People with the condition starting young, before the age of 40, are more likely to have a genetic predisposition (4). Antimicrobial peptides (AMPs), dendritic cells, and T lymphocytes are the hallmarks of psoriasis's pathogenesis (5).

Through their production of pro-inflammatory cytokines (6), the β-defensins play a role in both innate and adaptive immunity. Psoriasis etiology is thought to include IL-1, IL-18, and IL-20, all of which may be induced by human β-defensin (7).

Materials and methods

Data Sources: Literature on psoriasis's etiology, pathophysiology, clinical images, and the function of human β-defensin serum levels in psoriasis patients, as well as its relationship to the severity of the disease's decline up to 2024, was retrieved from Medline databases (Pub Med and Medscape).

For the purpose of study selection, each research was evaluated separately. Inclusion was contingent upon them meeting the following requirements: 1. Expressed and made public in English. Published in journals that undergo a rigorous peer review process. 3. Examine the origins, development, and symptoms of psoriasis. Go over the function of human β-defensins serum level in psoriasis patients and how it relates to the severity of the condition.

When extracting data, studies were discarded if they did not meet the inclusion criteria. Ethical permission, clear eligibility criteria, suitable controls, sufficient information, and well-defined evaluation measures were all variables in determining the study's quality. For our concerned research outcomes, data were independently extracted from all qualifying studies utilizing a data collecting form.

Review of literature:

Psoriasis

Plaque, flexural, guttate, pustular, and erythrodermic psoriasis are some of the many subtypes of this chronic immune-mediated inflammatory skin disease (8). Although it may affect any part of the skin, the most typical areas to experience it are the lower back, scalp, elbows, and knees (9).

Compared to children, adults have about four times the prevalence of this illness. There is a bimodal incidence in the early and late adult years, peaking about 60 years of age. Peak onset occurs between the ages of 15 and 25 and 50 and 60, but it may begin at any age, including in infancy, in about a third of instances (10).

The underlying mechanisms of psoriasis

One school of thought holds that psoriasis is an inflammatory illness mediated by Th1 and Th17, rather than only a Th1-mediated sickness. Reduced suppressive activity of T regulatory cells (Tregs) in psoriatic lesions could cause other effector cells to behave uncontrollably. This leads researchers to believe that psoriasis is not driven by a specific fraction of T cells but rather by complex interactions between many different subsets of T cells (11).

The following is a basic timeline of the immunologic events described in psoriasis:

Antigenic stimuli encourage the activation of innate immune cells in the skin, including plasmacytoid dendritic cells (pDCs).

Myeloid dendritic cells (mDCs) in the skin are activated and migrate more often when innate immune cells produce interferon-α and other proinflammatory cytokines.

Cytokines, especially IL 23, generated by mDCs recruit, differentiate, and activate T cells.

The most important cytokine produced by recruited T lymphocytes is interleukin 17A (IL-17A), which, together with other cytokines, stimulates the proliferation of keratinocytes and the generation of AMPs and cytokines that promote inflammation.

The inflammatory process is maintained by positive feedback loops that include cytokines produced by immune cells and keratinocytes (12).

Psoriasis risk factors and triggers

Perceived risk factors may really be triggers that cause disease manifestation in genetically prone individuals; there is substantial overlap between psoriasis triggers and risk factors. In order to establish strategies for the prevention and treatment of psoriasis, it is crucial to identify the risk factors (13).

Genetic reasons account for the vast majority of psoriasis cases, however environmental and behavioral variables also play a role. There are two categories of these elements: those that are inherent to the host and those that are extrinsic, such as environmental and behavioral factors (14).

The immune system

There is still much mystery surrounding the pathophysiology of psoriasis. Dysregulation of immune cell function and keratinocyte proliferation/differentiation are potential

explanations. The pathophysiology of psoriasis is believed to revolve on the overactivation of some components of the adaptive immune system (15).

A maintenance phase defined by a chronic clinical progression (16) follows the initiation phase in the present pathogenic model, which may be initiated by trauma (Koebner phenomenon), infection, or medications.

The symptoms and signs of psoriasis

Pimples of the skin

Red, symmetrical plaques covered in silvery scale are characteristic of classic plaque psoriasis (16). A dilated capillary network under the epidermis and a thinner supra papillary plate are seen by the Auspitz sign, which is pinpoint bleeding that results upon removing the scale (17).

Peeling skin condition

Multiple tiny (2-10 mm) teardrop-shaped psoriatic lesions, mainly on the trunk, are the hallmark of acute guttate psoriasis, which is often accompanied by a sore throat that is linked with group B streptococcal infection (18).

Ethredetodermal psoriasis

Infection, medication, systemic illness, or the discontinuation of corticosteroid treatment might cause plaques to gradually consolidate, a feature that characterizes this type. Hypothermia, hypoalbuminemia, electrolyte imbalances, and high-output heart failure are possible complications in individuals with erythrodermic psoriasis (19).

Psoriasis with pustules

Sheets of many painful, sterile pustules that coalesce are the hallmark of this condition. Pustular psoriasis may affect specific areas or the whole body. Psoriasis pustulosa palmoplantaris and acrodermatitis continuous are two separate localized forms (20).

Pelvic plaque dermatitis

The hands and feet are the typical sites of symmetrical localized plaque psoriasis, which is characterized by confluent redness and scales. There could be no plaques at all, little plaques that look like keratoderma, or poorly defined scaly or fractured spots (20).

Opposite psoriasis

Within the intertriginous zones, it is visible. The inframammary, perineal, and axillary regions are affected by red, glossy lesions that do not include scale. This condition is also known as flexural psoriasis. Obese people are more likely to experience it (21).

Warts on the nails

Inflammation of the nail bed manifests as oil-drop staining, splinter hemorrhages, and onycholysis, whereas pitting, leukonychia, and onychodystrophy are symptoms of nail matrix involvement (22).

Acne on the scalp

It manifests as red, elevated plaques that are surrounded by silvery white scales on the scalp, which may be restricted inside the hairline or spread over the forehead, ears, and back of the neck. Dandruff, irritation, and hair loss may be symptoms of a severe case (23).

Defensins Beta

The hBDs, which are AMPs generated from epithelial cells, protect mucosal membranes from microbes. Aside from their antibacterial properties, they also have additional roles, including as regulating the production of cytokines and chemokines, activating cells, facilitating migration and differentiation, promoting angiogenesis, and aiding in wound healing (24).

Keratinocytes are the primary cells in the skin responsible for producing the AMPs hBD-2 and hBD-3 (25). Research has shown a strong link between hBD-2, compromised skin barrier function, and several skin disorders (26).

Red Skin Disease

Plaques on the skin, characteristic of psoriasis, often appear on the trunk, elbows, and knees. Chronic skin inflammation, epidermal thickening and hyperplasia, increased vascularity, and immune cell infiltration characterize psoriatic lesions. Multiple inflammatory peptides and cytokines are overexpressed in lesions. The first two β-defensins, hBD-2 and hBD-3, were extracted from psoriatic scales because the overexpression of local cytokines such TNFα, IFN-γ, and IL-1 causes a rise in β-defensin production inside the lesions (27).

Condition Known as Atopic Dermatitis

Atopic dermatitis lesions, in contrast to psoriatic plaques, show a reduction in the production of β-defensins and a reduced ability to induce peptide levels related to inflammation. The local Th2-skewed cytokine milieu and the consequent concentrated suppression of β-defensin expression (27), rather than copy number variation, have been identified as the causes of this phenomenon.

Inhibiting the destructive effects of bacterial proteases, including those produced by the prevalent atopic dermatitis lesional pathogen Staphylococcus aureus, is one of the functions of certain β-defensins like hBD2. This is important since the loss of barrier integrity is a key component of this illness (28).

Getting Rid of Wounds

According to reports, hBD2 stimulates keratinocyte migration and proliferation, which aids in the wound repair of intestinal cells both in vitro and in vivo (29). Furthermore, a higher bacterial burden in the skin is associated with an uptick in classically activated macrophages and a downward trend in alternatively activated macrophages in these wound sites (27).

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