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Assessment of serum level of spexin in psoriasis vulgaris

Alaa H Oudah[1] , Hanan H.Sabry[1], Aliaa E.Mohammed[1] and Seham G.Ameen[2]

[1]Dermatology, Venereology and Andrology Dept., Faculty of Medicine, Benha University, Benha, Egypt

[2]Clinical and Chemical Pathology Dept., Faculty of Medicine, Benha University, Benha, Egypt

E-mail: nooralsaraf2000@gmail.com

Abstract

Background: Psoriasis vulgaris is a chronic inflammatory skin disease that manifests mainly as raised, erythematous, and scaly plaques. Psoriasis is associated with increased risk of several comorbidities, such as Crohn's disease, malignancy, obesity, and cardiovascular diseases. The relationship between psoriasis vulgaris and metabolic syndrome (MetS) has been a subject of increasing interest in recent years.

Objective: This article aims to review the link between metabolic syndrome and psoriasis vulgaris and its correlation with psoriasis severity.

Data Sources: The literatures on the causes, pathogenesis, clinical pictures of psoriasis vulgaris, discuss role of metabolic syndrome in patients with psoriasis vulgaris and its correlation with the disease's severity decline up to 2024 was sourced via a search of the Medline databases (Pub Med and Medscape).

Data Extraction: If the studies did not fulfill the inclusion criteria, they were excluded. Study quality assessment factors included whether ethical approval was gained, eligibility criteria specified, appropriate controls, and adequate information and well-defined evaluation measures. Data from each eligible study were independently abstracted using a data collection form to capture information related to our concerned study outcomes.

Conclusions: Both psoriasis vulgaris and MetS are associated with hormonal changes, insulin resistance, oxidative stress and chronic inflammation. Studies have shown that individuals with MetS are more likely to have severe psoriasis.

Keywords: Psoriasis; Obesity; Diet; Metabolic syndrome.

Introduction

Psoriasis is an inflammatory skin condition that develops over time with symptoms such as increased cytokine production, aberrant keratinocyte differentiation, T-lymphocyte infiltration, and epidermal hyperproliferation. Worldwide, psoriasis affects around 2-3% of the population [1].

The development of psoriasis is influenced by a combination of hereditary, immune, and environmental factors. Nevertheless, the exact cause has not been completely determined. Intrinsic and adaptive immune cells set off an inflammatory cascade in the dermis, which is the pathogenic mechanism in psoriasis [2].

Subcutaneous white adipose tissue may release cytokines like interleukin-36 (IL-36) and adipokines like leptin, which may exacerbate the cutaneous inflammation. Psoriatic lesions may form when their aberrant local and systemic expression affects immune cell activation, proliferation, and differentiation in addition to keratinocytes [3].

Materials and methods

Data Sources: Using the Medline databases (Pub Med and Medscape), the literature on psoriasis vulgaris was obtained, including articles on its etiology, pathophysiology, clinical images, the role of metabolic syndrome in psoriasis vulgaris patients, and its link with the disease's severity decrease up to 2024.

Study Selection: The inclusion of all research was determined by separate evaluations. Inclusion was contingent upon them meeting the following requirements: 1. The text was written and published in English. 2. Featured in journals that undergo a rigorous peer review process. Discuss the significance of metabolic syndrome in individuals with psoriasis vulgaris and its link with the severity of the illness. Review the etiology, pathophysiology, and clinical pictures of psoriasis vulgaris.

Data Extraction: Research was not considered for inclusion if it did not meet certain requirements. Ethical permission, clear eligibility criteria, suitable controls, sufficient information, and well-defined evaluation measures were all variables in determining the study's quality. We used a data collecting form to independently extract information relevant to our research results from all qualifying studies.

Review of literature:

Psoriasis

The red, scaley plaques that characterize psoriasis vulgaris, an inflammatory skin disorder that lasts for a long time, are surrounded by white hairs. Among the many chronic inflammatory skin disorders, psoriasis ranks high in prevalence. The skin and joints are affected by the autoimmune pathogenic features of psoriasis. Epidemiological studies show that psoriasis is more common in some countries than others, and that environmental factors, genetic

predisposition, and ethnicity all have a role in its onset and progression [5].

Adults and children alike are affected by psoriasis, which affects over 60 million people worldwide. With a median onset age of 33 years, psoriasis strikes equally males and females. According to genetic and immunological criteria, it may be classified into two subtypes: early onset, which manifests before the age of 40, and late onset, which manifests beyond that age [6]. It's possible for women to experience its symptoms sooner.

Psoriasis pathogenesis

It is thought that keratinocytes interact with several other skin cell types, leading to inflammation in the epidermal layers and the eventual creation of psoriatic plaques [7].

1. Contributions related to genes

The major risk factor for acquiring psoriasis is heredity. When comparing monozygotic twins to dizygotic ones, the risk is two to three times greater in the former. As the primary hereditary risk factor for psoriasis, HLA-C*06:02 is especially important for those who get the disease at a young age. There is no correlation between HLA-C*06:02 and late-onset illness, psoriatic arthritis, or pustular psoriasis [8].

Psoriasis cannot develop in the absence of an environmental trigger since the disease's onset is dependent on gene-environment interactions. Some examples of such factors include exposure to pharmaceuticals like lithium, antimalarials, and non-steroidal anti-inflammatory drugs; stress; infections (especially streptococcal); alcohol use; smoking; and, in rare cases, sunshine. Living with psoriasis may also cause weight gain and obesity, which are risk factors and triggers for the condition [9].

2. The immune system, both genetic and environmental, as well as feed-forward amplification

In addition to keratinocytes and other non-classic immune cells, certain traditional immune cells including dendritic cells and neutrophils play a role in disease development. Cytokines such as tumor necrosis factor (TNF-α), interferon (IFN-γ), IL-17, and IL-22 are the main means of cellular communication. Activation of keratinocytes also plays a role, promoting epidermal hyperproliferation and the production of antimicrobial proteins, growth factors, and chemokines. These factors contribute to the unique changes in psoriasis, which include the formation of new blood vessels, the invasion of neutrophils, and an increased number of type 1 (Th1) and type 17 (Th17) helper cells, which in turn create an inflammatory feedback loop [10].

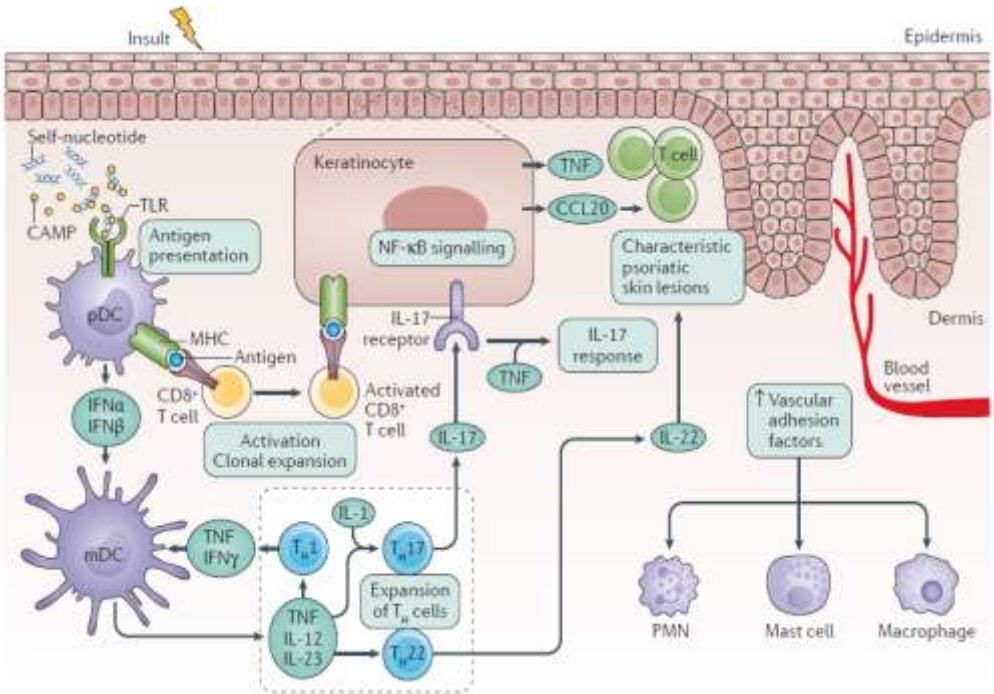

Fig (1): The mechanics of psoriasis. The release of self-nucleotides may be triggered by external insults such trauma, illness, or medicine. Keratinocyte-secreted antimicrobial peptides (AMPs) such cathelicidin antimicrobial peptide (CAMP) may bind to plasmacytoid dendritic cells' surface Toll-like receptor 7 (TLR7) when complexed with self-nucleotides. In turn, this causes pDCs to present antigens, which activate and clonally expand CD8+ T

lymphocytes specific to those antigens. Both local lymph nodes (where naïve T cells are activated) and the dermis (where memory resident T cells are activated) are capable of carrying out this procedure. [7].

How psoriasis manifests clinically

• Chronic plaque dermatitis

Clear, salmon-pink plaques decorated with silvery scales appear on white skin and grey plaques on black skin constitute the characteristic morphology. Minor bleeding spots, known as the Auspitz sign, may occur after the removal of adherent scales. There is a wide range in the size and thickness of plaques, which might be an indicator of disease activity (small plaques) or a therapeutic response (phototherapy is more effective on thinner plaques) [11].

• Psoriasis of the gut

A centripetal pattern of several tiny scaly papules is one possible symptom of psoriasis. Roughly 50% of patients have high streptozyme, anti-DNase B, or antistreptolysin O titres, and 65% have a history of pharyngitis or tonsillitis. Around 40% of guttate psoriasis instances develop into chronic plaque disease[12], despite the fact that the majority of cases spontaneously clear up within a few weeks or months.

• Epidermoderma

Symptoms such as scaling, exfoliation, or confluent redness covering more than 75% of the body's surface area are common indicators of this condition. Hypothermia, high-output heart failure, an electrolyte imbalance, pruritus, and skin discomfort are all possible side effects with this level of skin involvement [13].

• Pustules where skin is inflamed all over

Periodic flares, sterile pustules, and pyrexia are symptoms of this potentially fatal autoinflammatory illness. It affects more women than males and differs epidemiologically from chronic plaque psoriasis. Hypocalcaemia, illness, pregnancy, and the quick reduction of systemic and powerful topical corticosteroids are the triggers for generalized pustular psoriasis [14].

Pustulosis of the palms and soles

Typical symptoms include the development of red or brown macules after a few weeks of healing from sterile pustules that are yellow in color on the palms and soles. About 20% of individuals with palmoplantar pustulosis also have chronic plaque psoriasis, and the condition mostly affects middle-aged women who smoke [15].

When you have psoriasis on top of metabolic syndrome, you raise your risk of cardiovascular disease, stroke, and type 2 diabetes. Some examples of these health issues are hypertension, diabetes, abnormal cholesterol or triglyceride levels, and abdominal obesity [16]. Researchers have shown that psoriasis patients had a higher risk of developing metabolic syndrome than the general population. Patients with psoriasis are more likely to develop metabolic syndrome, according to a meta-analysis of observational data [17]. The pooled odds ratio for this condition is 2.14.Both psoriasis and metabolic syndrome are characterized by persistent inflammation, and this shared feature has prompted many theories on their shared causes. One feature of psoriasis is the secretion of cytokines that promote inflammation; these cytokines may exacerbate insulin resistance and other metabolic syndrome symptoms [18].Psoriasis and metabolic syndrome are both exacerbated by reactive oxygen species and inflammatory cytokines, which may be produced as a result of endoplasmic reticulum stress [19]. In both circumstances, there are changes in the levels of adipocytokines, including leptin and adiponectin. Metabolic syndrome and psoriasis may have a similar route that involves alterations in the makeup of the gut microbiota [20].When metabolic syndrome and psoriasis occur together, it might be difficult to treat either illness effectively. As an example, psoriasis therapies may not work as well in those who are overweight or have metabolic syndrome. Hence, in order to effectively treat psoriasis, it is essential to address the components of metabolic syndrome [21].

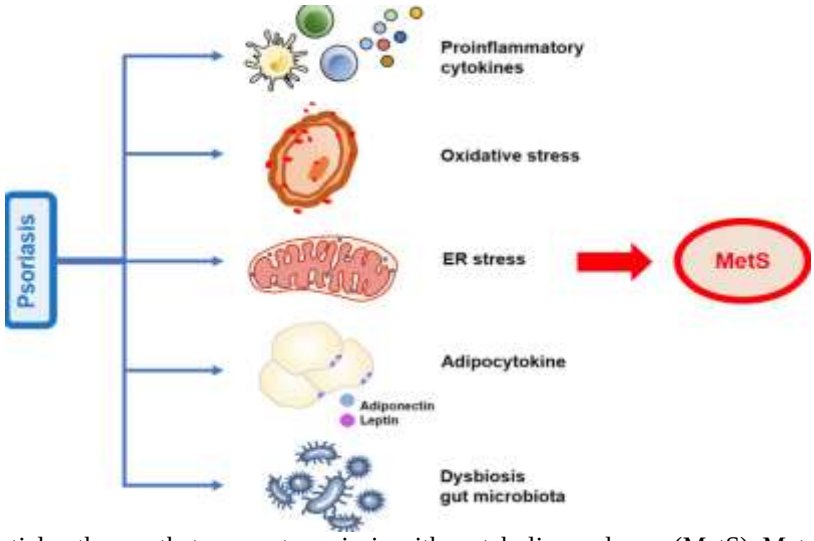

Fig (2): Identifying potential pathways that connect psoriasis with metabolic syndrome (MetS). Metabolic syndrome (MetS) develops in psoriasis due to an increase in pro-inflammatory cytokines, secretory adipocytokines from adipose tissue, activation of oxidative stress states, increased stress in the endoplasmic reticulum (ER), and dysbiosis of the gut microbiota [16].

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