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Evaluation of Serum Endocan Level and Erectile Functions in Patients with Psoriasis Shaimaa M. Shalaby¹, Fatma M. El Esawy¹, Ghada M. Abd El Khalik¹ and Amira O. Abd El Ghafar²¹Dermatology, Venerology and Andrology Department, Faculty of Medicine, Benha University, Benha, Egypt ²Clinical and Chemical Pathology Department, Faculty of Medicine, Benha University, Benha, Egypt E-Mail: shaymaashalaby64@gmail.com

#### Abstract

Background: In psoriasis patients, circulating endocan is associated with disease severity and cardiovascular risk. Once again, endocan levels were correlated with inflammatory markers; individuals with psoriasis had greater amounts than controls. Elevated endocan levels may indicate inflammatory and endothelial factors contributing to erectile dysfunction pathophysiology (ED). As a new diagnostic indicator for the degree of erectile dysfunction, endocan could be helpful. The purpose of this review is to examine erectile dysfunction, serum endocan levels, psoriasis epidemiology, and pathophysiology in great detail. The endothelial and inflammatory components of the aetiology of both psoriasis and erectile dysfunction may be represented by increased endocan levels, according to the conclusions. One possible use for endocan is as a diagnostic marker for erectile dysfunction and psoriasis.

Keywords: Erectile Functions, Endocan, and Psoriasis are the main terms here.

### Introduction

Psoriasis is a common skin condition that may have both hereditary and non-hereditary causes, including things like substance abuse, cigarette smoking, emotional and mental stress, physical damage, and infections. The primary risk factors for psoriasis development are believed to be the combination between environmental and genetic variables [1].

Epidermal hyperproliferation, improved antigen presentation, T helper (Th) 1 cytokine secretion, T-cell expansion, and angiogenesis are hallmarks of psoriasis' pathophysiology [2].

Heart disease (CVD), diabetes (DM), and high blood pressure are all more common in those with psoriasis. Many consider endothelial dysfunction to be the first step in the onset of atherosclerosis [3].

Among males in their middle years and beyond, erectile dysfunction (ED) ranks high. Problems getting or keeping an erection strong enough for sexual intimacy are known as erectile dysfunction [4].

Midlife and older men may have erectile dysfunction due to a combination of physical and mental health issues, including high blood pressure, diabetes, cardiovascular disease, and mental health disorders including anxiety and depression. Reduced blood flow to the corporal bodies, which is necessary for attaining an erection, is a result of atherosclerosis and hypertension, which constrict and stiffen the arteries. Because diabetes may impact the penis's blood vessels and nerves, it is a prevalent cause of sexual dysfunction [5].

One multi-dimensional tool for gauging men's erection health is the International Evaluation of Erectile Function (IIEF) [6]. To measure the prevalence and severity of ED, a five-item IIEF (IIEF-5) was developed. One criterion for selecting these five items was their ability to detect ED [7].

Elevated levels of endocan, an immunoinflammatory marker, have been seen in

some illnesses associated with endothelial dysfunction [8].

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Cardiovascular risk and disease severity are both correlated with circulating endocan in psoriasis patients. Again, there was a link with inflammatory indices (e.g., C-reactive protein (CRP) and Psoriasis Area and Severity Index) [9]; endocan levels were greater in psoriasis patients compared to controls.

The elevated endocan levels might be a reflection of the inflammatory and endothelial factors involved in the development of erectile dysfunction. Endocan has the potential to become a valuable diagnostic tool for assessing the severity of erectile dysfunction [8].

This study aims to provide a thorough synopsis of what is currently known about psoriasis patients' serum endocan levels and erectile functioning.

**Psoriasis** 

More than 125 million individuals throughout the globe suffer with psoriasis, a persistent skin condition. In addition to affecting the nails, it leaves skin with scaly patches. The patient's QOL may be significantly affected [10].

The process is facilitated by communication between cells in the dermis, cells that produce keratin, and immunocytes such T-cells and antigen presentation cells (APCs) [11].

Symptoms include a propensity for symmetrical body distribution, red plaques with adhering silvery white scales, and damage to the skin, nails, and joints [12].

# **Epidemiology of psoriasis**

Although psoriasis may strike either sexe at any time, it tends to manifest sooner in females and in individuals who have a genetic predisposition. Men experience its highest incidence between the ages of 30 and 39 and 60 and 69, but women begin to feel its effects 10 years earlier [13].

There is a bimodal distribution of ages at which psoriasis symptoms could first appear. Two peaks occur at around 20 and 60 years, respectively. A greater genetic tendency is seen in those whose

sickness begins around the age of 20. The group with late onset illness had a weaker connection to hereditary factors [14].

Climate, genetics, and antigen exposure are all potential factors in the observed regional differences in psoriasis prevalence; however, it seems that the disease is more prevalent in countries farther from the equator, and that psoriasis is less common in countries closer to the equator overall [15].

Psoriasis is more prevalent among white people, and its incidence is unequally distributed between geographical areas [16]. It is documented to exist in 19% of nations globally. The overall incidence is greater in nations with a high per capita income, and it varies from 0.1% in eastern Asia to 1.5% in western Europe. Compared to adults, children have a lower prevalence and incidence rate [17]. The percentage of the Egyptian population affected by psoriasis varies between 0.19 and 3% [18].

How Psoriasis Develops

Psoriasis may be thought of as having two stages: the initiation phase, which may be brought on by trauma (the Koebner phenomenon), infection, or medications, and the maintenance phase, which is marked by a persistent clinical manifestation. progression (Figure 1) [19].

## **Erectile Dysfunctions**

"Recurrent and persistent inability, partial or total, to produce or maintain an erection firm enough for pleasant sexual intercourse in the presence of suitable erotic stimuli" is the definition of erectile dysfunction (ED), which takes the place of the earlier definition of "impotence" [20].

It's important to recognise ED as a societal issue since it may touch men of all ages and races, and it greatly affects the quality of life for both the patient and his partner [21].

The Study of ED Epidemiology

Prevalence estimates for ED ranged from 3.7% to 76.5% all across the world. The combination of the IIEF and a questionnaire generated from the Massachusetts Male Aging Study (MMAS) revealed a significant incidence of ED in young men, and there was a favourable association between ED and CVD as well [22].

Prevalence estimates for ED range from 14% to 48% based on epidemiological data. Disagreements in study methodology on age and socioeconomic level likely account for the large variation in these variables [23].

Keeping up a healthy sexual activity routine is heavily influenced by age. There is a wide fluctuation in the prevalence of moderate or total erectile dysfunction (ED) among males, from 9% in the 40-44 age group to 56% in the 65+ age group [21].

The underlying causes of impotence

Even when the smooth muscle contracts, the penis maintains its flaccid shape. An erection occurs when parasympathetic cholinergic nerve fibres release acetylcholine and nonadrenergic noncholinergic nerve fibres release nitric oxide (NO). This leads to increased cyclic GMP (cGMP) concentrations, decreased intracellular Ca2+ levels, and smooth muscle cell relaxation, all of which are regulated by adrenergic (noradrenaline) control, intrinsic myogenic control, and endothelium-derived contracting factors (prostaglandins and endothelins). Blood flows into the corpora cavernosa's lacunar spaces when smooth muscle relaxes, which presses on the subtunical venules and blocks their outflow (veno-occlusion). Phosphodiesterase type hydrolyzes cGMP, inverting the process (PDE5). Any disruption to any of these processes may lead to ED [24].

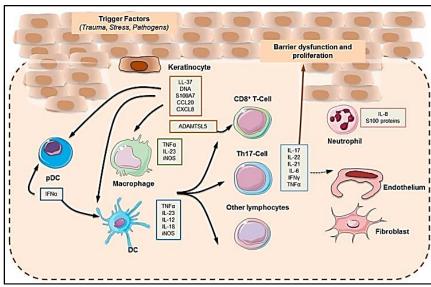


Fig (1) Psoriasis's pathogenesis [19].

### **Endocan**

Activated endothelial cells primarily release endocan, a soluble dermatan sulphate proteoglycan expressed by endothelial cells in the kidneys and lungs. Cytokines that promote inflammation regulate its release. Angiogenic factors include vascular endothelial growth factor (VEGF) and IFN- $\gamma$ , as well as TNF- $\alpha$  and IL-1 $\beta$ , lipopolysaccharide, and endocan, up-regulate its production and secretion [25].

It may have a role in the inflammatory state, as it may be up-regulated by several pro-angiogenic and pro-inflammatory cytokines [26]. The vascular endothelial cells that make up the endothelium barrier sit between the tissues in close proximity to the blood as it flows. The endothelial cells in your body are involved in both the immunological and circulatory systems, and they also operate as a mechanical protective barrier [27].

Because of its unique location, it controls inflammation, host defence, tissue-fluid balance, vascular tone, vascular remodelling, and angiogenesis. Both atherosclerosis and ischemic heart are significantly impacted by compromised endothelial cell function and integrity. Endothelial damage increases reactive oxygen species (ROS) generation, which in turn decreases NO production by raising cytoplasmic calcium ion concentrations [28].

It is possible to initiate and sustain an inflammatory condition of the blood vessel wall via an increase in ROS generation and a decrease in NO availability. This mechanism facilitates the development of atherosclerotic plaques by attracting white blood cells and allowing blood lipids and lipoproteins to flood the subendothelial region [29].

These characteristics highlight its possible use as an indicator of inflammation and endothelial dysfunction. Leukocyte adherence to endothelial cells is facilitated by endocan, a novel indicator of endothelial cell activity. Multiple clinical investigations have shown that endocan levels are substantially elevated in cardiovascular disease (CVD) patients and are associated with soluble intercellular adhesion molecule-1 (sICAM-1) and soluble vascular cell adhesion molecule-1 (sVCAM-1) levels in a manner that is distinct from one another [30].

Psoriasis and the Function of Endocan

One possible mechanism by which psoriasis develops is endocan's function in modifying cell adhesions. Microvascular permeability may be increased, leukocyte movement can be influenced, and endothelial cell production of proinflammatory cytokines can be stimulated [31].

Leukocytic adhesion to endothelial cells, migration to the dermis, cell proliferation, and inflammation are all processes that may include endocan. Several studies have detailed the mitogenic activity of endocan [32].

The endocan protein core binds to monocytes and lymphocytes by forming a high-affinity interaction with the integrin CD11a/CD18, which is also called LFA-1. One important step in the pathophysiology of psoriasis, known as angiogenesis, is vascular endothelial growth factor-A, and endocan has been shown to enhance its mitogenic and migratory capabilities [33].

It seems that endocan plays a role in the etiopathogenesis of psoriasis, since the levels of endocan in serum and lesional skin biopsies were significantly higher in the psoriatic group compared to the control group. It is plausible that endocan plays a role in the etiopathogenesis of psoriasis and contributes additional to the severity of the disease since blood and tissue endocan levels were much higher in patients with moderate-to-severe psoriasis compared to those with mild disease [34].

Patients with psoriatic arthritis showed a statistically significant connection with serum endocan. Inflammation plays a major role in the development of endothelial dysfunction, hence... [35].

Patients with psoriatic arthritis had a much higher serum endocan level compared to healthy controls. Serum endocan also showed a tendency to correlate with PASI and a strong correlation with the duration of psoriasis [36].

Importance of Endocan for Impotence

If endothelial dysfunction is to be evaluated in these patients, it may be pertinent to note that the control and ED groups had significantly different serum endocan levels. There are shared risk factors or predictors between atherosclerosis and ED [37].

Serum endocan levels differed significantly between individuals with and without severe erectile dysfunction, according to research by Onuk et al. (2018). The Sexual Health Inventory for Men Score was also shown to have a high negative correlation with endocan levels. This investigation confirmed that there is a correlation between plasma endocan levels and erectile dysfunction [38].

Researchers Elkamshoushi et al. (2018) investigated endocan levels in connection to erectile dysfunction. An detrimental correlation between endocan levels and erectile dysfunction was found (as measured by the IIEF-5 score). These authors claim that endocan levels are associated with ED severity [30].

The IIEF-5 score was used by Karabakan et al. (2017) to compare controls and ED patients. Endocan levels were higher in the ED group compared to the control group. The degree of erectile dysfunction was similarly associated with endocan levels [39].

Endocan showed promise as a novel diagnostic marker for the severity of erectile dysfunction (ED) [8]. Elevated endocan levels may reveal endothelial and inflammatory components of ED pathogenesis.

### Recommendations and future prospectives:

Activated endothelial cells primarily release endocan, a soluble dermatan sulphate proteoglycan expressed by endothelial cells in the kidneys and lungs. Cytokines that promote inflammation regulate its release. Angiogenic factors include vascular endothelial growth factor (VEGF) and IFN- $\gamma$ , as well as TNF- $\alpha$  and IL-1 $\beta$ , lipopolysaccharide, and endocan, up-regulate its production and secretion [25].

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#### **Conclusions**

Psoriasis and erectile dysfunction may have a common inflammatory and endothelial component, which may be shown by elevated endocan levels. As a possible diagnostic marker for psoriasis and erectile dysfunction, endocan might possibly be useful.

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