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*(Research Article.)*

# Assessment of Endocan Serum Levels in Psoriatic Patients and Their Relation To Endothelial Dysfunction

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**Abstract:** Psoriasis is considered an independent risk factor for cardiovascular diseases (CVD). It is crucial to find new biomarkers that help with the early detection of endothelial dysfunction. This study aimed at investigating the clinical relevance of serum endocan concentrations in psoriatic patients and their value in the detection of endothelial dysfunction (ED). This is an observational cross-sectional study that was conducted on 60 patients who consecutively presented to the Outpatient Clinics at Mansoura Dermatology & Leprosy Hospital, and were diagnosed with plaque psoriasis during the study period, and an equal number of healthy individuals as a control group. For the identification of patients with ED, flow-mediated vasodilatation (FMD) was assessed. The levels of endocan in psoriatic patients (versus levels in normal individuals) and the relation between endocan levels and the indicator of endothelial dysfunction (FMD%) were assessed. The patients' ages ranged between 18 and 65 years. Females constituted 55% and 51.8% of the two groups, respectively. The patient and control groups were comparable in age and sex. The disease duration was significantly longer in psoriatic patients with ED than in those without ED at $p < 0.05$. This study showed a significantly higher prevalence of ED in psoriatic patients, comprising 71.1% of the patients versus 35% in control subjects. Serum concentrations of endocan were significantly higher among psoriatic patients in comparison with healthy individuals. Higher serum endocan concentrations were detected in patients with ED. Correlation analysis showed that serum endocan concentrations were positively correlated with Psoriasis Area and Severity Index (PASI) score, and C-reactive protein (CRP), but there was significant negative correlation with FMD%. In conclusion, patients with psoriasis are particularly predisposed to endothelial dysfunction as an earlier form of CVD. Endocan showed significant associations with psoriasis-associated ED. We suggest its use in the routine screening of patients with psoriasis.

**Keywords:** Psoriasis, cardiovascular disease, endothelial dysfunction, endocan



## 1. INTRODUCTION

Psoriasis is an inflammatory disease that is immune-mediated and accompanied with many comorbid conditions that impact human life.[1] Psoriasis is found worldwide and the prevalence ranged from 2 to 4% of the world's population. It varies among different ethnic groups. In Egypt, it varies from 0.19 to 3%.[2] By far, cardiovascular disease (CVD) has been one of the most recognized and debatable psoriasis-associated comorbidities.[3]

Currently, besides being associated with various risk factors predisposing to CVD, such as obesity and metabolic syndrome, psoriasis is considered an independent risk factor for CVD due to its related chronic inflammatory state.[4] There is conspicuous similarity between the immunological and inflammatory pathogenic backgrounds implicated in psoriasis and atherosclerosis.[3] This inflammatory state results in endothelial dysfunction (ED),[3] which is a key atherogenic step and a mainstay for the development of CVD.[5,6]

The precise pathogenic mechanisms linking psoriasis to CVD and atherosclerosis are still a matter of debate and a subject of extensive research over the past few decades. Multiple studies showed that

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individuals with psoriasis have a high risk of developing CVD compared to those without the skin condition.[2-4,5,6] Several factors contribute to this association, and these include chronic inflammation. Psoriasis is a chronic inflammatory immune-mediated disease which is associated with increased circulatory concentrations of inflammatory cytokines. Chronic inflammation is a key contributor to the development and progression of atherosclerosis, a condition where fatty deposits build up inside arteries, narrowing the blood vessels and reducing blood flow. The ongoing inflammatory state in psoriasis may accelerate the atherosclerotic process, increasing the risk of cardiovascular complications like heart attacks and strokes. In addition, psoriatic patients often have a higher prevalence of traditional cardiovascular risk factors which include obesity, hypertension, dyslipidemia, and diabetes. These risk factors are independent contributors to cardiovascular disease, and their coexistence in psoriasis patients further increases the overall risk. [2-4,5,6] Moreover, there is evidence suggesting that psoriasis may be linked to insulin resistance and resulting hyperglycemia. Insulin resistance is associated with a high risk of developing type 2 diabetes and CVD. Furthermore, psoriasis is not merely a skin disorder; it has systemic effects on various body systems and organs. The chronic inflammatory state in psoriasis can have far-reaching consequences beyond the skin, affecting the cardiovascular system and contributing to the development of CVD. Additionally, psoriasis can significantly impact a person's quality of life and mental well-being due to its visible nature and potential discomfort. Chronic stress and psychological factors associated with psoriasis may also contribute to a high risk of cardiovascular disease through various mechanisms, including alterations in hormone levels and unhealthy coping behaviors. Also, some medications used to treat moderate to severe psoriasis, such as certain immunosuppressants and systemic treatments, might have potential cardiovascular side effects. Finally, psoriasis is associated with endothelial dysfunction, which promotes the development of atherosclerotic plaques and compromises overall cardiovascular health. [2-4,5,6]

The most popular method used to detect endothelial dysfunction is the assessment by ultrasound of the FMD of brachial artery.[7] Applying a combined tool approach to evaluate ED would yield a more solid stratification of patients according to their risk of CVD. Therefore, it is crucial to find new biomarkers that help

A recently identified endothelial-specific molecule, endocan, has been studied. It is a vascular endothelium-synthesized proteoglycan that is involved in the leukocytes adhesion to endothelial cells. It is regulated by pro-inflammatory mediators and is implicated in inflammatory and vascular diseases.[8,9] Therefore, it is assumed to be an important marker for ED and CVD.[10]

While research on endocan is ongoing, there is limited direct evidence linking endocan to ED or CVD. Elevated levels of endocan are observed in conditions associated with increased inflammation, such as atherosclerosis.[11] This suggests a potential link between endocan and the inflammatory processes contributing to endothelial dysfunction. Endocan may also influence oxidative stress,[12] which can damage endothelial cells, leading to endothelial dysfunction. Moreover, it may affect vascular tone by impacting the release of endothelial nitric oxide (NO),[13] which is crucial for relaxing blood vessels.

High blood pressure, a major risk factor for CVD, has also been linked to endocan,[14] indicating its potential contribution to hypertension. Some studies have even shown that elevated endocan levels are correlated with heart failure, often arising as a consequence of underlying cardiovascular diseases. [15,16] Endocan levels have been explored as a prognostic marker for CVD, with higher levels associated with an increased risk of adverse cardiovascular events. [17]

Despite the extensive research into the association between psoriasis and CVD, there remains a need for a deeper understanding of the mechanisms underlying this connection and the development of more effective strategies for early detection and management. This study seeks to address this gap by exploring the clinical significance of endocan, a novel endothelial-specific molecule, in psoriatic patients and its potential value in detecting endothelial dysfunction, a precursor to CVD. By doing so, we aim to contribute to the ongoing efforts to unravel the complex interplay between psoriasis, endothelial dysfunction, and cardiovascular complications, ultimately improving patient care and outcomes.

This study aimed at investigating the clinical relevance of endocan in psoriatic patients and their value in the detection of endothelial dysfunction.

## 2. METHODS

This is an observational study that was performed over two year (from January 2019 to January 2021). The approval for the study was obtained from the ethical committees of Faculty of pharmacy (Girls) - Al-Azhar University (Approval number: 109/2017) and Mansoura Dermatology & Leprosy hospital. The choice of the time frame was to ensure that we collected data from a large and diverse sample of

**Table 1: The demographic and clinical data in the studied groups**

| | **Patients group (n =60)** | | | | **Control group (n =60)** | | | | **p** |
|---|---|---|---|---|---|---|---|---|---|
| | **With ED (n= 43)** | | **Without ED (n= 17)** | | **With ED (n= 21)** | | **Without ED (n= 39)** | | |
| | **No.** | **%** | **No.** | **%** | **No.** | **%** | **No.** | **%** | |
| **Sex (n%)** | | | | | | | | | |
| Male | 19 | 44.2 | 8 | 47.1 | 11 | 52.4 | 18 | 46.2 | 0.687 |
| Female | 24 | 55.8 | 9 | 52.9 | 10 | 47.6 | 21 | 53.8 | |
| **Age (years)** | | | | | | | | | |
| Mean ± SD | 42.83 ± 14.53 | | 38.83 ± 14.49 | | 41.57 ± 13.28 | | 33.63 ± 6.48 | | 0.029 |
| | $p_1$=0.612, $p_2$=0.980, $p_3$=0.387 | | | | | | | | |
| **BMI (kg/m$^2$)** | | | | | | | | | |
| Mean ± SD | 28.88 ± 5.56 | | 25.38 ± 4.15 | | 25.15 ± 5.89 | | 26.08 ± 4.24 | | 0.017 |
| | $p_1$=0.039, $p_2$=0.024, $p_3$=0.950 | | | | | | | | |
| **Smoking (n%)** | | | | | | | | | |
| No | 31 | 72.1 | 13 | 76.5 | 16 | 76.2 | 32 | 82.1 | 0.796 |
| Yes | 12 | 27.9 | 4 | 23.5 | 5 | 23.8 | 7 | 17.9 | |
| **PASI (n%)** | | | | | | | | | |
| Moderate | 19 | 44.2 | 10 | 58.8 | – | – | – | – | 0.438 |
| Severe | 24 | 55.8 | 7 | 41.2 | – | – | – | – | |
| **PASI- score** | | | | | | | | | |
| Mean ± SD | 12.80 ± 6.26 | | 11.36 ± 5.79 | | – | | – | | 0.297 |
| **Disease duration (months)** | | | | | | | | | |
| Mean ± SD | 88.97 ± 88.85* | | 49.50 ± 62.05 | | – | | – | | 0.013 |
| **FMD%** | | | | | | | | | |
| Mean ± SD | 4.57 ± 1.26 | | 9.99 ± 2.62 | | 5.96 ± 0.79 | | 12.69 ± 4.73 | | 0.001 |
| $p_1$<0.001, $p_2$=0.033, $p_3$=0.283 | | | | | | | | | |

$p_1$: indicating the comparison among the **patient with ED and patient without ED**

$p_2$: indicating the comparison between the **patient and control (with ED)**

$p_3$: indicating the comparison between the **patient and control (without ED)**

*: Statistically significant at $p \leq 0.05$

individuals with psoriasis. Psoriasis is known to have various triggers, and the severity of the condition can fluctuate over time. By selecting a two-year window, we aimed to include a wide range of participants who may have experienced different levels of psoriasis severity during this period. The study adhered to the Declaration of Helsinki.

Patients who consecutively presented to the Outpatient Clinics at Mansoura Dermatology & Leprosy Hospital, Dakahlia, Egypt, and were diagnosed with plaque psoriasis during the study period were recruited to the study. An equal group including age- and sex-matching healthy subjects who attended the hospital for blood donation was the control group. To choose the control group, individuals with known acute or chronic illness, or cardiovascular risk factors, including hypertension, diabetes, and smoking, were excluded from the control group. Also, individuals with dermatological conditions, or those on regular medications were excluded.

The study participants were assessed clinically with taking history and general physical examination. The diagnosis of psoriasis vulgaris in the patients' group was based on the typical clinical criteria.[18] The PASI score is calculated as follows: [19]

1. Erythema (Redness): Each of four anatomic regions (head, upper extremities, trunk, and lower extremities) was assessed, and the degree of erythema was scored on a scale of 0 to 4, with 0 indicating no redness and 4 indicating maximum redness.
2. Induration (Thickness): Similar to erythema, the degree of induration in each region was scored on a scale of 0 to 4, where 0 represents no thickness and 4 represents maximum thickness.
3. Desquamation (Scaling): The scaling was assessed in the same four anatomic regions, with scores ranging from 0 (no scaling) to 4 (maximum scaling).
4. Extent of Involvement: The extent of body surface area involved was estimated as a percentage of the patient's total body surface area.

Once these individual scores were determined for each parameter, the PASI score was calculated using the following formula:

PASI = (Erythema Score x 0.3) + (Induration Score x 0.3) + (Desquamation Score x 0.2) + (Body Surface Area Score x 0.1)

This calculation results in a PASI score, which can vary from 0 (no psoriasis) to a maximum of 72, with higher scores indicating more severe psoriasis. Psoriasis severity was classified according to the PASI. We used the following criteria to classify psoriasis severity:

1. Mild Psoriasis: PASI score less than or equal to 10.
2. Moderate Psoriasis: PASI score between 10 and 20.
3. Severe Psoriasis: PASI score greater than 20.

Adult patients with moderate or severe psoriasis were enrolled in our study. The exclusion criteria included patients who administered systemic or narrow band treatment for psoriasis or recent anti-inflammatory drugs, patients with overt CVD, infection, inflammatory joint disease, pulmonary disease, liver or kidney disease, or malignancy, and pregnant females.

Informed written consent was obtained from each included patient. The informed consent process involved explaining the purpose, procedures, potential risks, and benefits of the study to each participant. We also emphasized the voluntary nature of participation and the right to withdraw at any time without consequences.

Furthermore, we ensured that all participants' personal information and data were handled with strict confidentiality and in compliance with data protection regulations.

After obtaining written consents from all included subjects, the included participants underwent assessment of their height and weight, based on which the BMI was calculated, and laboratory analysis.

For the identification of patients with endothelial dysfunction, ultrasound assessment of the brachial artery FMD was done. Each participant was instructed to fast for 6 hours, and an ultrasound machine was used to assess the brachial artery diameter with a 7.5-MHz probe on B mode. The baseline diameter of the right artery was assessed 3–5 cm proximal to the cubital fossa. After a sphygmomanometer cuff was placed and secured around the proximal arm, it was inflated to a level higher than the patients' systolic pressure. After about five minutes, cuff deflation was done. The brachial artery was then in a dilatory response (reactive hyperemia), and its diameter was measured

**Table 2. The biochemical parameters in the studied groups**

| | Patient (n =60) | | Control (n =60) | | p |
|---|---|---|---|---|---|
| | **With ED (n= 43)** | **Without ED (n= 17)** | **With ED (n= 21)** | **Without ED (n= 39)** | |
| **Glucose (mg/dL)** | | | | | |
| Mean ± SD. | 101.71 ± 13.69 | 101.42 ± 13.35 | 95.87 ± 12.77 | 92.33 ± 10.79 | 0.012 |
| | $p_1$=1.000, $p_2$=0.288, $p_3$=0.033 | | | | |
| **CRP (mg/L)** | | | | | |
| Mean ± SD. | 6.98 ± 4.19 | 5.08 ± 4.46 | 5.07 ± 1.95 | 3.13 ± 1.55 | <0.001 |
| | $p_1$<0.001, $p_2$=0.167, $p_3$=0.060 | | | | |
| **Endocan (ng/L)** | | | | | |
| Mean ± SD. | 306.34 ± 48.85 | 192.60 ± 64.49 | 182.47 ± 36.11 | 171.47 ± 55.39 | <0.001 |
| | $p_1$<0.001, $p_2$<0.001, $p_3$=0.073 | | | | |

*p: p value for the comparison among the studied groups*

*$p_1$: indicating the comparison between the **patient with ED and patient without ED***

*$p_2$: indicating the comparison between the **patient and control (with ED)***

*$p_3$: indicating the comparison between the **patient and control (without ED)***

**: Statistically significant at $p \leq 0.05$*

**Table 3. Correlation analysis between serum endocan levels and different parameters**

| | **Endocan** | |
|---|---|---|
| | **Correlation coefficient (r)** | **p** |
| Disease duration (months) | 0.193* | 0.140 |
| PASI- score | 0.359* | 0.005 |
| CRP | 0.749* | <0.001 |
| FMD% | -0.655* | <0.001 |

$r_s$: Spearman coefficient, * statistically significant at $p \leq 0.05$

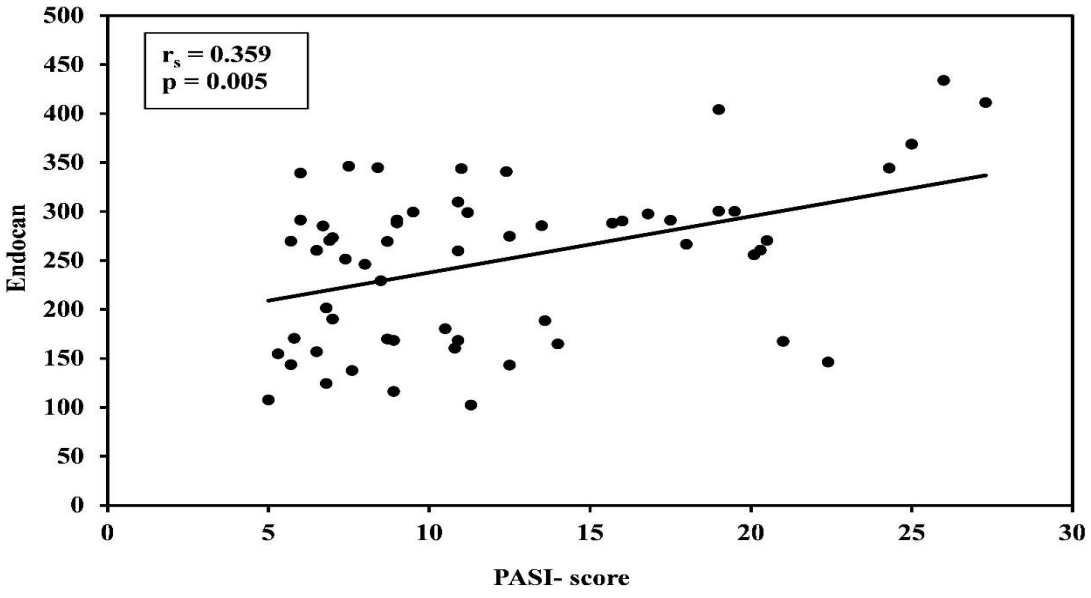


**Figure 1**. Correlation between endocan and PASI- score in Patient group (n=60)

at this stage. The FMD% underwent calculation as the difference between the reactive hyperemia diameter and the baseline diameter divided by the baseline diameter, then multiplied by 100.[20] Endothelial dysfunction was considered when the median FMD was less than 7%.[21]

The participants underwent laboratory investigations, including the assessment of fasting serum glucose levels by using enzymatic colorimetric method, and serum C-reactive protein (CRP) by quantitative turbidimetric assay.

Assessment of the serum endocan was determined by using ELISA kits following the instructions provided by the manufacturer (Cat. No. E3160HU, Shanghai Korian Biotech Company, China).

**Statistical analysis**

The patients' collected data were statistically analysed by SPSS (IBM Corp., Armonk, NY, USA), version 28. Nominal variables were expressed as frequencies and percents and compared as appropriate. Quantitative values were tested for normality, and the suitable tests was performed accordingly. Spearman correlation analysis was performed. A p-value < 0.05 was considered statistically significant.

## 3. RESULTS

This observational study included 60 psoriatic patients and an equal number of healthy individuals. The patients' ages ranged between 18 and 65 years. The patient and control groups were comparable in age and sex. Table 1 present the demographic and clinical data of the study groups. The disease duration was significantly longer in psoriatic patients with ED than in those without ED at $p < 0.05$.

According to the measured FMD%, endothelial dysfunction was found in 43 patients (71.7%) and 21 control subjects (35%), with a statistically significant difference ($p<0.001$). Both the patients and control groups were subdivided into 2 groups according to the presence of ED. They differed significantly in age ($p = 0.029$), with the control subgroup without endothelial dysfunction showing the youngest age. BMI also differed significantly among the subgroups ($p = 0.017$), since patients with ED had a higher mean BMI compared with patients without ED and with control group. The disease duration also showed statistically significant increase in patients with ED than in those without ED ($p = 0.013$). In contrast, a non-statistically significant difference was found among the subgroups in the prevalence of smoking ($p = 0.796$), PASI ($p = 0.297$), and PASI score ($p = 0.297$). As the measure for endothelial dysfunction, a significant difference was revealed among the four subgroups in the FMD% values ($p<0.001$). Patients with ED had lower mean values compared with patients without ED ($p<0.001$), as did the control subjects ($p = 0.033$) (Table 1).

***Assessment of Biochemical parameters:***

The biochemical parameters and serum endocan concentrations in the study groups are demonstrated in Table 2.

Pair-wise comparisons revealed that fasting blood sugar level was significantly higher ($p = 0.033$) in patients without endothelial dysfunction compared to their counterparts in the control group. CRP levels were significantly higher among patients with ED compared with those without ED ($p< 0.001$).

Patients with ED showed higher mean endocan concentrations compared with patients without ED ($p<0.001$), and with control subjects ($p<0.001$).

The correlation between endocan concentrations in the serum and different clinical and biochemical parameters in patients' group is presented in Table 3 and Figures 1, 2, 3 and 4. Serum endocan levels showed statistically significant positive correlation with BMI ($p=0.015$), PASI score ($p=0.005$), and CRP ($p<0.001$), but there was significant negative correlation with FMD% ($p<0.001$).

## 4. DISCUSSION

The early depiction of atherosclerosis and cardiovascular affection, while still in the preclinical stage in patients with psoriasis, could aid in improving patients' outcomes through early treatment and strict, regular follow-up monitoring.[22] Also, this can provide targets for new therapeutic options. Therefore, this work aimed to assess the usefulness of endocan in the detection of endothelia dysfunction, an early stage of atherosclerosis.

The present work showed a significantly higher prevalence of endothelial dysfunction among psoriatic patients, comprising 71.1% of the patients. Our findings emphasize the high risk of CVD in these patients. This observation was also made by Erfan et al.[21] who reported that 80% of patients with psoriasis had endothelial dysfunction. On the other hand, Ibrahim et al.[9] reported an obviously lower percentage (17.9%). This variation is likely attributed to the differences in the FMD% cutoff value that was used to diagnose endothelial

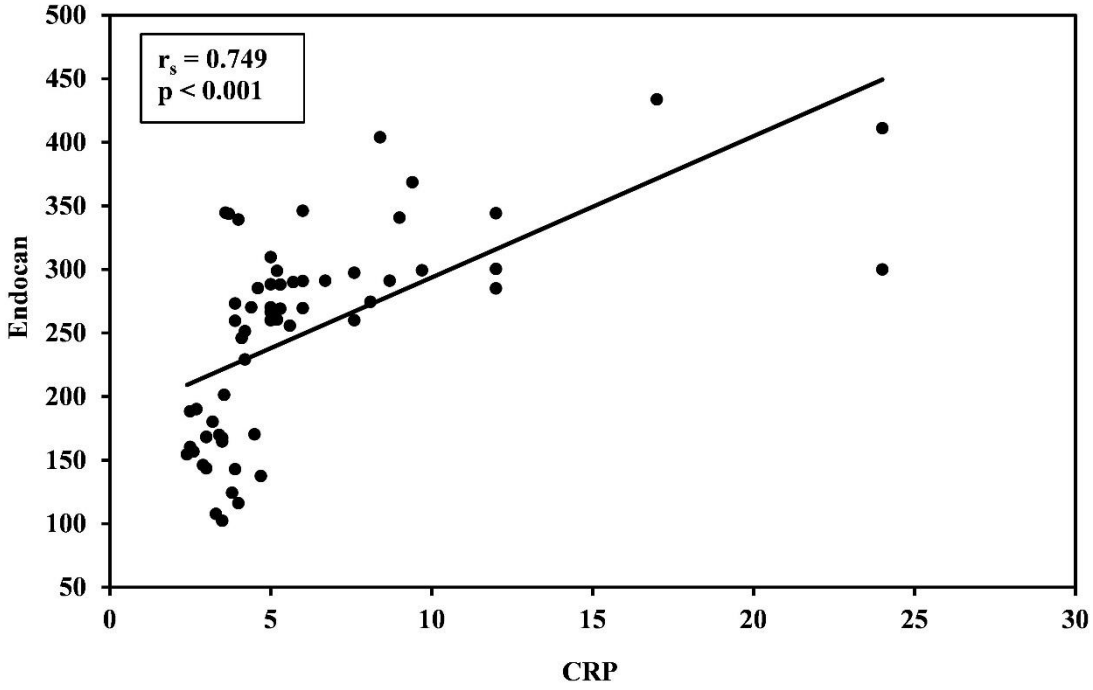


**Figure 2.** Correlation between endocan and CRP in patient group (n=60)

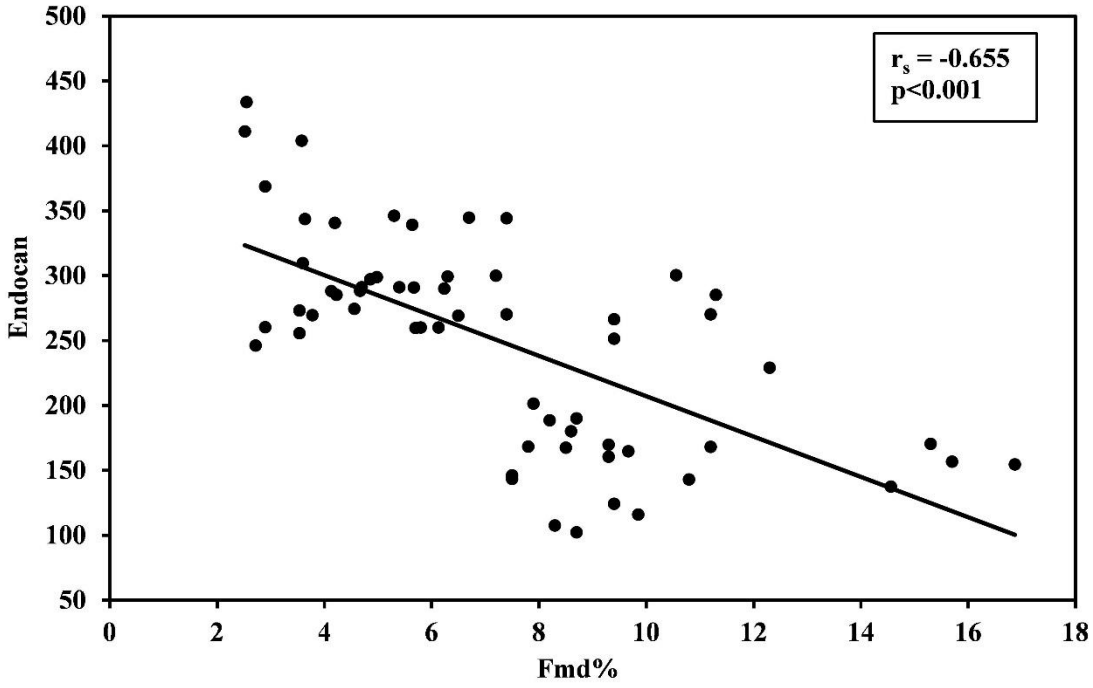


**Figure 3.** Correlation between endocan and Fmd% in patient group (n=60)

dysfunction. In our study, as well as Erfan et al.'s[21] an FMD% of less than 7% was considered to have endothelial dysfunction, compared to a cutoff value of 4.5% adopted by Ibrahim et al.[9]

With regard to the assessment of endocan, an important biomarker for inflammation and CVD, the current study showed that endocan concentrations were significantly higher among psoriatic patients in comparison with control subjects. Also, it showed significant association with disease severity as evident in the positive correlation between the PASI and the endocan concentrations. This was consistent with Ibrahim et al.[9], Balta et al. [23], Farag et al.[24], Sabry et al.[25], Elkamshoushi et al.[26], and Elgarib et al.[27], who reported higher endocan concentrations in psoriatic patients compared to control subjects. Within the same context, Gobrial et al.[28] and Abdou et al.[29] reported upregulation of endocan in the affected skin of psoriatic patients, which was more evident in those having severe psoriasis. These data support the implication of endocan in psoriasis pathogenesis. The higher concentrations of endocan in psoriatic patients and its positive correlation with disease severity that was found in this study consistently with other studies could be attributed to the inflammatory and endothelial dysfunction processes inherent to psoriasis. These findings suggest that endocan may play a role in the pathogenesis of psoriasis and may also have implications for the cardiovascular risk associated with the condition.

Furthermore, the present work demonstrated significant elevation in the serum endocan concentrations in patients with ED. This was confirmed by the significant negative correlation between the endocan concentrations and FMD%. Scarce evidence is available regarding the endocan relation to ED in patients with psoriasis. In agreement with this study, Ibrahim et al.[9] confirmed this relationship. Sabry et al.[25] and Elkamshoushi et al.[26] reported the association between atherosclerosis and cIMT and endocan levels. A plausible explanation is the reported data regarding the role of endocan in the promotion of inflammatory particle transport and leukocyte adhesion to endothelial cells.[8,9] This theory support the positive correlation between the serum endocan and CRP concentrations.

While the exact mechanisms are not completely understood, based on this study, there are several potential justifications for higher endocan concentrations in psoriatic patients. First, ED: since psoriasis is associated with endothelial dysfunction,[4] which can lead to increased release of endocan from the damaged endothelial cells. Second, angiogenesis: psoriasis is known to involve an increased formation of new blood vessels (angiogenesis) in the skin lesions.[30] Endocan is associated with angiogenesis, and its levels may increase in response to the vascular changes seen in psoriatic skin. Third, inflammation: Psoriasis is fundamentally an inflammatory disease. Inflammatory cytokines and immune cells can stimulate the production of endocan.[3,4] The higher levels of inflammation in psoriasis patients could be a contributing factor to increased endocan levels.

Our findings offer a potential avenue for improving patient care. By incorporating endocan into routine screening tests for patients with psoriasis, we have the opportunity to identify endothelial dysfunction at an earlier stage, allowing for more timely interventions. Further investigations are warranted to establish standardized cutoff values for endocan in psoriasis patients and to explore the cost-effectiveness and feasibility of implementing endocan testing on a broader scale. Moreover, in-depth mechanistic studies could help elucidate the precise role of endocan in the pathogenesis of psoriasis and its implications for cardiovascular risk. Long-term prospective studies may provide insights into the predictive value of endocan for cardiovascular outcomes in psoriasis patients.

It's essential to note that medical research is an ongoing process, and new findings may shed further light on the relationship between endocan levels and psoriasis.

This study indicates that endocan could be clinically meaningful marker for the early detection of endothelial dysfunction in psoriatic patients. However, the study is limited by the relatively small sample size and being non-longitudinal study. Further larger studies on its potential use as a target for new therapeutic solutions are recommended5.

## CONCLUSIONS

Patients with moderate or severe plaque psoriasis are particularly predisposed to endothelial dysfunction, an earlier form of cardiovascular disease (CVD). Endocan has been shown to have significant associations with psoriasis-associated endothelial dysfunction. These findings offer a potential avenue for improving patient care. By incorporating endocan into routine screening tests for patients with psoriasis, we have the opportunity to identify endothelial dysfunction at an earlier stage, allowing for more timely interventions.

However, it's important to acknowledge that there may be limitations and potential challenges associated with implementing this recommendation. These could include issues related to the availability and cost-effectiveness of endocan testing on a large scale, as well as the need for further research to establish specific cutoff values and guidelines for interpretation. Balancing the benefits and limitations of endocan as a screening tool will be crucial in realizing its full potential in psoriasis patient care.

While this study has shed light on the potential utility of endocan in psoriasis care, it is essential to look toward future research directions. Further investigations are warranted to establish standardized cutoff values for endocan in psoriasis patients and to explore the cost-effectiveness and feasibility of implementing endocan testing on a broader scale. Moreover, in-depth mechanistic studies could help elucidate the precise role of endocan in the pathogenesis of psoriasis and its implications for cardiovascular risk. Long-term prospective studies may provide insights into the predictive value of endocan for cardiovascular outcomes in psoriasis patients.

By emphasizing these avenues for future research, we aim to encourage the advancement of knowledge in this field and provide guidance for researchers working on the intersection of psoriasis, endothelial dysfunction, and cardiovascular health.

**Supplementary Materials:**

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**Ethical Statement:** The study obtained its approval from the ethical committees of Faculty of pharmacy (Girls) -Al-Azhar University (Approval number: 109/2017).
**Author Contribution:** This research was equally contributed among authors.

**List of Abbreviations:** CRP: C-reactive protein, CVD: cardiovascular disease, ED: endothelial dysfunction, FMD: flow-mediated vasodilatation, PASI: Psoriasis Area and Severity Index.

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