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# Assessment of Serum Level of N-Terminal Pro B-Type Natriuretic Peptide as A Predictor of Cardiovascular Disease in Patients with Psoriasis Vulgaris

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## ABSTRACT
**Background:** Psoriasis is a chronic immune-mediated inflammatory illness that impacts two to three percent of the global population. Cardiovascular (CV) comorbidities in cases with psoriasis may be asymptomatic and may even result in sudden mortality. Despite its widespread recognition, the N-terminal pro B-type natriuretic peptide (NT-proBNP) is not yet a standard tool for predicting (CV) outcomes in patients. **Objective:** The research's goal was to estimate NT-proBNP, as a predictor of CVD, in psoriasis cases and compare it with healthy non psoriatic controls and searching for any correlation among the level of serum NT-proBNP and severity of psoriasis, as defined by PASI ( Psoraisis Area and Severity Index).
**Patients and Methods:** 50 participants with psoriasis and 40 matched healthy controls underwent laboratory test for serum level of NT-proBNP: Each participant had 3 ml of venous blood drawn, centrifuged at 3000 g for five minutes. The serum was stored at −70 degrees Celsius for further biochemical examination. Serum paraoxonase-1 concentrations were measured utilizing ELISA kits Cat. No E3041Hu.
**Results:** In cases who had psoriasis, NT-proBNP levels were considerably greater than in the control group (median=148.5 against 53; $p<0.01$). Psoriasis severity was inversely related to NT-proBNP rise. Among psoriasis cases, those with hyperlipidemia had significantly higher NT-proBNP level when compared to those with non-hyperlipidemia.
**Conclusion:** Increased levels of NT-proBNP were seen in the studied psoriatic individuals, supporting the possibility of utilizing this biomarker to predict CV hazard in people with psoriasis.
**Keywords:** Psoriasis, NT-proBNP, Cardiovascular (CV).

## INTRODUCTION

The chronic immune-mediated inflammatory illness psoriasis affects two to three percent of the world's population [(1)]. Immune system, heredity, keratinocytes, and environmental factors interact in a complex manner in the pathogenesis of psoriasis [(2, 3)].

The fact that psoriasis is linked to other major hazards such hypertension, diabetes, dyslipidemia, obesity and smoking does not explain why those with severe psoriasis have a higher hazard of heart attack. Those with more severe psoriasis appear to be at greatest risk for developing CVD [(4)]. Psoriatic inflammation may raise CVD risk as well as other risk factors and treatments [(5)].

High levels of proinflammatory cytokines in the blood of psoriatic patients may also encourage the migration of inflammatory cells across the endothelium of blood vessels, which can result in endothelial dysfunction and plaque formation [(1, 6)]. It is important to identify reliable biomarker(s), which may be beneficial for early diagnosis, prognosis and prevention of certain cardiovascular problems.

Brain natriuretic peptide (BNP) is a 32-amino acid peptide produced by the ventricles, Prepro-BNP is synthesized first, then pro-BNP is cleaved off and finally BNP is produced [(7)]. BNP is predominantly produced and stored in the cardiac ventricles from membrane granules and BNP release is continuous because of ventricular volume expansion and pressure overload [(8)].

N-terminal pro-brain natriuretic peptide (B-type natriuretic peptides) has been recognized as a marker for incipient cardiac risk [(9)]. It has gained credibility as a helpful biomarker in HF diagnosis and prognosis [(10)].

To our knowledge, only two studies investigated serum level of (NT-proBNP) in psoriasis. The two studies reported higher NT-proBNP concentrations in psoriatic cases related to healthy controls [(11, 12)].

## AIM OF THE WORK

This study's objective was to determine the serum level of NT-proBNP, a predictor of CVD, in psoriasis cases and contrast it with healthy non-psoriatic controls. Also, to search for any correlation among the level of serum NT-proBNP and severity of psoriasis, as defined by PASI.

## PATIENTS AND METHODS

Fifty people with psoriasis and forty people without the skin condition were studied. The controls were of a similar age and gender to the participants. They were randomly selected from the outpatient clinic of the Dermatology Department at Mansoura University Hospital. Patients were diagnosed on the basis of typical clinical manifestations. Age, gender, SBP, DBP, and heart rate were all determined for both patients and controls. The PASI score was also utilized to determine the severity of the psoriasis. All patients underwent laboratory test for lipid profile, FBS, ESR and CRP.

Serum NT-proBNP levels were measured in all cases. Each participant had 3 ml of venous blood drawn, centrifuged at 3000 g for five minutes, the resulting serum was maintained at -70 degrees Celsius for biochemical testing.



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Serum paraoxonase-1 concentrations were measured utilizing ELISA kits from Germany(Cat. No. E3041Hu). This kit is an Enzyme-Linked Immunosorbent Assay (ELISA). The plate has been pre-coated with Human **NT-proBNP**. **NT-proBNP** present in the sample is added and binds to antibodies coated on the wells This kit is an Enzyme-Linked Immunosorbent Assay (ELISA). The plate has been pre-coated with Human **NT-proBNP**. **NT-proBNP** present in the sample is added and binds to antibodies coated on the wells. Kits.

**Ethical consent:**

**In accordance with Helsinki Declaration, this research (MS.20.05.1132) was approved by the Institutional Review Board (IRB) of the Mansoura University Faculty of Medicine. Participants were not included in the study until they gave permission. Data privacy was given the highest priority. The collected information was used for research only.**

***Statistical analysis***

The collected data were cleaned, processed, tabulated and imported onto a computer using the Statistical Package for the Social Sciences (IBM Corp., 2017). Armonk, New York: IBM Corp., 2015, IBM SPSS Statistics for Windows, Version 25.0. According to the nature of the data collected for each parameter, the appropriate presentation and investigation were performed.

- Parametric numerical data were analyzed using Mean±SD, whereas non-parametric data were analyzed utilizing median and range and the 2 groups were compared by independent t-test, if the data were parametric and by Mann Whitney Test (U test) if non-parametric.
- Non-numerical data was expressed using frequency and percentage and were compared by Chi-square test. When the predicted count was < 5 (in higher than twenty percent of cells), Fisher's exact test was performed instead of Chi-square test.
- The Kruskal-Wallis test was employed to determine whether or not a difference in non-parametric variables existed among three groups.
- If the *p*-value was < 0.05 (at the 95% confidence interval), then the result was significant.

**RESULTS**

Fifty psoriasis patients and forty controls of similar age and gender were involved in this study. From Mansoura University Hospital's Dermatology outpatient clinic, patients were selected at random. Age and gender were matched between the 2 groups. No tremendous contrasts were found regarding anthropometric measures between them (Table 1).

**Table (1): Comparison of demographic data and anthropometric measures amongst studied groups**

| | | Control | Psoriasis | *p* |
|---|---|---|---|---|
| | | n=40 | n=50 | |
| Age (days) mean±SD | | 41.9±8.8 | 45.5±10.2 | 0.081 |
| Gender | Male N (%) | 10 (25%) | 21 (42%) | 0.092 |
| | Female N (%) | 30 (75%) | 29 (58%) | |
| weight (kg) mean±SD | | 74.5±9.8 | 72.4±9.5 | 0.301 |
| Height (m) mean±SD | | 162.7±6.3 | 163.8±7.2 | 0.467 |
| BMI (kg/m$^2$) mean±SD | | 25.8±3.0 | 26.8±2.9 | 0.125 |

(**SD** standard deviation**, BMI** body mass index)

All control subjects were non hyperlipidemic, while 32% of cases with psoriasis had hyperlipidemia. The difference was significant between the 2 groups (Table 2 and figure 1).

**Table (2): Comparison of laboratory data amongst studied groups**

| | Control | Psoriasis | *p* |
|---|---|---|---|
| | n=40 | n=50 | |
| TC (mg/dl) mean±SD | 127.3±9.7 | 190.7±28.2 | **<0.001** |
| TG (mg/dl) mean±SD | 116.5±12.4 | 137.5±33.5 | **0.047** |
| HDL (mg/dl) mean±SD | 63.2±9.4 | 52.4±9.0 | **0.046** |
| FBG (mg//dl) mean±SD | 91.6±13.8 | 113.2±27.1 | **0.038** |
| HGB (g/dl) mean±SD | 11.4±0.8 | 10.1±0.6 | 0.287 |
| RBC (X10$^6$/L) mean±SD | 4.2±0.4 | 3.6±0.5 | 0.137 |
| RDW mean±SD | 13.7±1.1 | 20.4±4.8 | **<0.001** |
| ESR (mm/hour) mean±SD | 14.6±2.6 | 25.8±4.1 | **<0.001** |
| Positive CRP, N (%) | 1 (2.5%) | 14 (28%) | 0.001 |

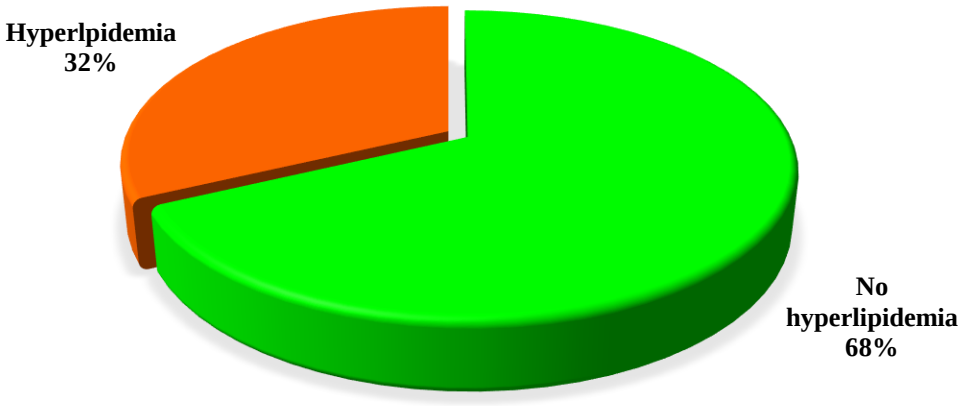


**Figure (1): Hyperlipidemia among studied cases.**

**Table (3): Psoriasis grades among studied cases**

| | Psoriasis |
|---|---|
| | n=50 |
| **PASI median (range)** | 12.6 (2.9-38.2) |
| **Mild N (%)** | 15 (30%) |
| **Moderate N (%)** | 27 (54%) |
| **Severe N (%)** | 8 (16%) |

**Table (4): Association of BP with psoriasis grades**

| | Mild | Moderate | Severe | *p* |
|---|---|---|---|---|
| | n=15 | n=27 | n=8 | |
| **SBP (mmHg) mean±SD** | 110±10.4 | 109.4±11.0 | 118.1±11.9 | 0.144 |
| **DBP (mmHg) mean±SD** | 69.7±6.4 | 68.9±7.6 | 75.0±8.0 | 0.126 |

Just TC showed critical relationship expanded grades. Psoriasis grades were not related altogether research facility boundaries (Table 5).

**Table (5): Association of laboratory data with psoriasis grades**

| | Mild | Moderate | Severe | *p* |
|---|---|---|---|---|
| | n=15 | n=27 | n=8 | |
| **TC (mg/dl) mean±SD** | 173.6±33.2 | 191.2±16.6 | 221.3±22.9 | **<0.001** |
| **TG (mg/dl) mean±SD** | 132.7±38.1 | 134.4±43.8 | 156.8±44.8 | 0.726 |
| **HDL (mg/dl) mean±SD** | 49.3±9.5 | 54.9±8.9 | 50.0±6.2 | 0.103 |
| **FBG (mg//dl) mean±SD** | 102.1±23.0 | 97.6±31.2 | 124.4±32.4 | 0.374 |
| **HGB (g/dl) mean±SD** | 10.3±0.4 | 10.0±0.7 | 10.2±0.9 | 0.543 |
| **RBC (X106/L) mean±SD** | 3.7±0.4 | 3.6±0.5 | 3.5±0.5 | 0.478 |
| **RDW mean±SD** | 22.0±6.5 | 19.3±3.6 | 20.8±6.2 | 0.357 |
| **ESR (mm/hour) mean±SD** | 26.2±4.8 | 25.1±4.8 | 27.0±6.6 | 0.621 |
| **Positive CRP N (%)** | 5(33.3%) | 5(18.5%) | 4(50%) | 0.198 |

NT-proBNP showed significant higher level in psoriasis group than control group (Table 6).

**Table (6): Comparison of NT-proBNP amongst studied groups**

| | Control | Psoriasis | *p* |
|---|---|---|---|
| | n=40 | n=50 | |
| | median (min-max) | median (min-max) | |
| **NT-proBNP** | 53 (37-140) | 148.5 (44-250) | **<0.001** |

Psoriasis severity was positively correlated with an increase in NT-proBNP levels (Table 7).

**Table (7): Comparison of NT-proBNP according to psoriasis grades**

| | | NT-proBNP | *p* |
|---|---|---|---|
| | | median (min-max) | |
| **Grades** | **mild** | 132(44-147) | **<0.001** |
| | **moderate** | 149(134-171) | |
| | **severe** | 171(162-250) | |

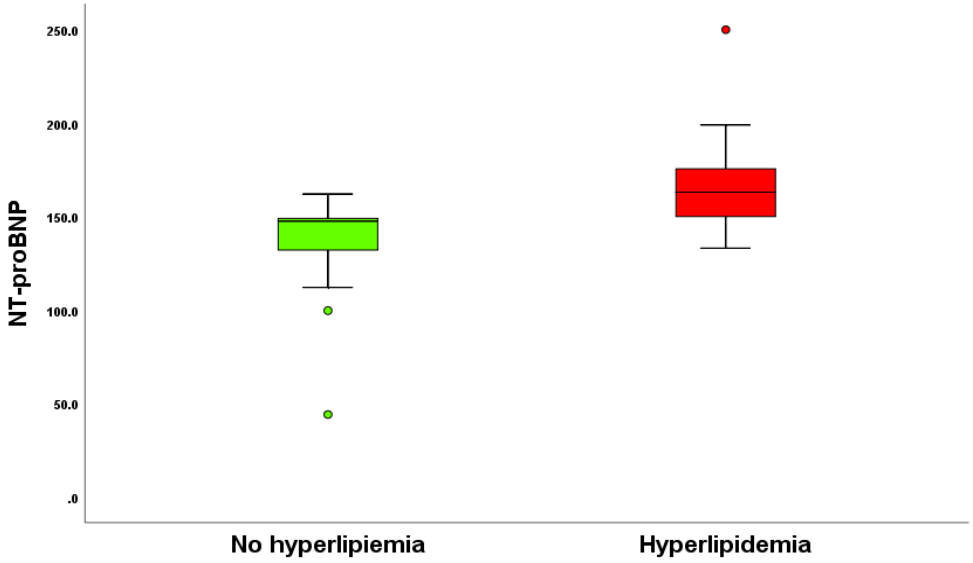


**Figure (2): NT-proBNP according to hyperlipidemia among Psoriasis cases**

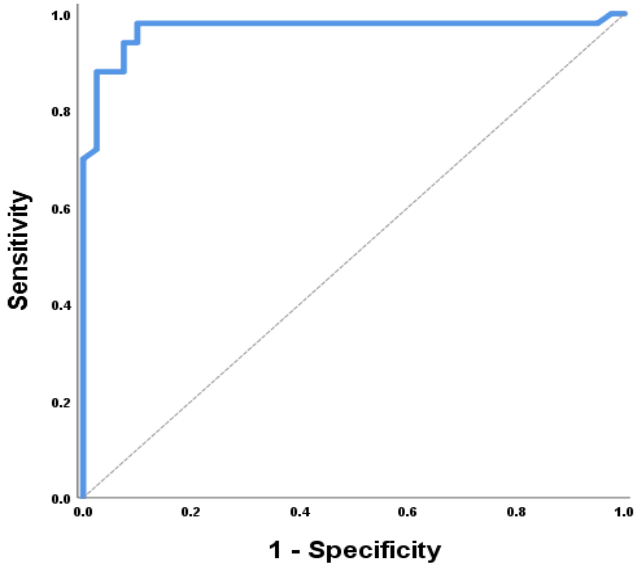


**Figure (3): ROC curve of NT-proBNP for discrimination among psoriasis participants and control groups**

NT-proBNP showed critical positive relationships with SBP, DBP, TC, TG, LDL, FBG, ESR, CRP, and PASI, and huge negative connections LDH (Table 8).

**Table (8): Correlations of NT-proBNP with other parameters among all studied subjects**

| | NT-proBNP | |
|---|---|---|
| | ***rs*** | ***p*** |
| **age** | 0.053 | 0.622 |
| **BMI** | -0.123 | 0.247 |
| **SBP** | 0.426 | **<0.001** |
| **DBP** | 0.436 | **<0.001** |
| **TC** | 0.882 | **<0.001** |
| **TG** | 0.227 | **0.031** |
| **HDL** | -0.200 | **0.049** |
| **FBG** | 0.268 | **0.011** |
| **HGB** | 0.162 | 0.461 |
| **RBC** | 0.279 | 0.266 |
| **RDW** | 0.163 | 0.460 |
| **ESR** | 0.639 | **<0.001** |
| **CRP** | 0.347 | **0.001** |
| **PASI** | 0.835 | **<0.001** |

## DISCUSSION

Autoantigens, psoriasis-associated susceptibility loci, the immune system and environmental variables all have a role in causing psoriasis [13,14]. As a result of its association with other comorbidities, this disorder is now classified as a systemic inflammatory illness [15].

Psoriasis is linked to conventional cardiovascular risk factors, since major CV events are the leading reason for mortality in psoriasis cases [16]. Psoriasis has been linked to obesity, dyslipidemia, DM2, and hypertension. Activation of the Th1 and Th17 pathways, proinflammatory cytokines, and elevated oxidative stress are likely to blame for this correlation's existence. In addition to favoring a prothrombotic condition, all of these variables also encourage leucocyte adhesion [17].

Finding biomarkers of CVD that may be used for diagnosis, prevention and prognosis might be very helpful. When wall tension rises, cardiomyocytes produce the hormone B-type natriuretic peptide (BNP). Prohormone BNP is processed by the endoprotease furin inside of cardiomyocytes to produce the hormone BNP and the inactive NT-proBNP.

The use of NT-proBNP measurement has been validated for the identification of LV systolic and/or diastolic dysfunction, in addition to for the screening of CVD and the diagnosis of heart failure. Biomarker NT-proBNP predicts cardiovascular disease hazard in healthy persons[12].

In the current research, the average age of psoriasis group was 45.5 years. They were 21 men (42%) and 29 women (58%). As well as forty healthy control subjects of matched age and sex were included. This average age was consistent with[18-20]. **Megna *et al.*** [19] revealed that the average age of psoriasis diagnosis was 45.8 ± 14.3 years.

Our research results showed that women were slightly predominant (58%). **Similarly, Egeberg *et al.*** [21] found that 54% were women.

In comparison to what has been proposed so far, **Praveenkumar *et al.*** [22], **and others** [23, 24] found that obesity (high BMI) resulted in a higher incidence of psoriasis.

In a meta-analysis showed by **Duan *et al.*** [25] that psoriasis-related inflammation leads to vascular inflammation and hypertension through inflammatory pathways, endothelin-1 overexpression, and enhanced oxidative stress [26]. Moreover, in the pathogenesis of psoriasis, chronic systemic inflammation and endothelial dysfunction may accelerate the development of atherosclerosis in the vessel walls [27] resulting in a greater likelihood of hypertension and CVD in younger individuals [28].

In the current study, only TC showed significant association with increased grades. Otherwise, psoriasis grades were not associated significantly with age, gender, BMI, SBP, DBP and laboratory parameters (TG, HDL, FBG, HGB, RBC, RDW, ESR and Positive CRP) ($p>0.5$ for each). In line with this, **Yazdanpanah *et al.*** [29] found a significant associations among the severity of psoriasis and cholesterol ($p<0.05$), indicating an increase in cholesterol with the increase in severity of psoriasis.

**In the present study,** psoriasis cases had substantially higher levels of NT-proBNP than control subjects (median=148.5 vs 53; $p<0.01$).

In general, there are a few investigations of NT-proBNP in psoriasis [18, 25, 31]. In agreement with our results **Shahidi-Dadras *et al.*** [12] who found that the median serum level of NT-proBNP has been shown to be greater among people with psoriasis compared to healthy controls (median= 26.67 versus 17.45; $p=0.0001$).

These results corresponded with **Pietrzak *et al.*** [11] who discovered that the NT-proBNP levels in normolipidemic and hyperlipidemic psoriasis cases was substantially higher than in normolipidemic and hyperlipidemic controls. Only 28 (38.36%) of psoriatic individuals had normal lipid profiles, whereas 45 (61.64%) had elevated TG and/or TC. Also, while the lipid profile was within normal ranges in 14 of the controls (31.11 percent), it was elevated in 31 (68.8 percent) of the controls. **However,** in both psoriatic groups, i.e., normo- and hyperlipidemic, the NT-proBNP levels were significantly higher in contrast to the normo- and hyperlipidemic controls, $P = 0.02$ and $P = 0.001$ correspondingly. There were no substantial associations among NT-proBNP mean concentrations and lipids in either the normolipidemic or hyperlipidemic study and control groups.

In the current study, serum NT-proBNP showed significant +ve correlations with SBP, DBP, TG, TC, LDL,

FBG, ESR, CRP, and PASI, and significant -ve correlations with HDL. Otherwise, no significant correlations were found regarding of NT-proBNP with other parameters among studied subjects. In the same line, **Shahidi-Dadras *et al.*** [(12)] found that total cholesterol and LDL levels were positively linked with serum NT-proBNP levels in psoriatic individuals.

Although the reasons behind the increased cardiac biomarker levels in psoriasis are not well understood, inflammatory cytokines can directly damage cardiomyocytes, whereas systemic inflammation might cause endothelial dysfunction or generate cardiac strain indirectly [(30)]**.** C-reactive protein (CRP) and pro-inflammatory cytokines are both observed in elevated levels in the blood of people with psoriasis, there have been several research aimed at reducing the inflammatory load in patients with inflammatory rheumatic illnesses, all of which lend credence to the inflammatory theory of CVD in psoriatic cases. Treatment with tumour necrosis factor-alpha (TNFα) inhibitors decreased the development of carotid plaques in males and improved vascular inflammation in females with psoriatic disease in a prospective cohort trial [(31)]**.**

## CONCLUSION

The findings demonstrate the existence of elevated NT-proBNP levels in the study patients, which may be a useful biomarker for predicting CV danger in psoriatic individuals. Serum NT-proBNP level was positively correlated with clinical severity of psoriasis measured with PASI ($P < 0.001$). Serum NT-proBNP level was significantly associated with SBP, DBP, TC, TG, LDL, FBG, ESR and CRP while it has a substantial negative connection with HDL ($p = 0.049$).

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**Author contribution:** Each author contributed equally to the research.

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