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Serum Asprosin and Meteorin-Like Protein Levels in Psoriasis: Associations with Disease Severity and Treatment Response

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Abstract

Psoriasis is a chronic immune-mediated inflammatory skin disease associated with systemic inflammation and metabolic disturbances. Adipokines have been suggested as key mediators linking metabolic dysregulation and inflammatory pathways in psoriasis. This study aimed to evaluate serum asprosin and meteorin-like protein (METRNL) levels in patients with psoriasis, investigate their relationship with disease severity, and assess their changes following treatment. A total of 38 newly diagnosed patients with psoriasis and 25 healthy controls were included. Blood samples were collected before and after treatment in patients and once in controls. Serum asprosin and METRNL levels were measured using enzyme-linked immunosorbent assay. Clinical and metabolic parameters, including Psoriasis Area and Severity Index (PASI), were also recorded. Serum asprosin levels were significantly higher in patients with psoriasis compared to controls and showed a significant decrease after treatment. In contrast, METRNL levels did not differ significantly between groups and remained unchanged following treatment. No significant correlations were observed between adipokine levels and PASI score or metabolic parameters. Receiver operating characteristic (ROC) analysis indicated that asprosin had moderate diagnostic performance. These findings suggest that asprosin may reflect systemic metabolic-inflammatory alterations in psoriasis and may be useful as a treatment-responsive biomarker, whereas METRNL appears to have limited value as a circulating marker.

Introduction

Psoriasis is a long-standing immune-mediated inflammatory disorder of the skin that typically presents with sharply demarcated erythematous plaques covered by silvery scale and develops on a background of abnormal keratinocyte turnover and persistent immune activation [1]. It affects nearly 2–3% of the population worldwide and remains one of the most prevalent chronic dermatologic diseases [2]. Although the cutaneous manifestations dominate the clinical picture, psoriasis is now widely regarded as a systemic inflammatory disorder because of its links with obesity, insulin resistance, metabolic syndrome, and cardiovascular disease [3].

The biological basis of psoriasis involves an interplay of hereditary predisposition, environmental influences, and dysregulated innate and adaptive immune responses [4]. Among the pathways implicated, the IL-23/Th17 axis is central to both initiation and perpetuation of lesional inflammation [5]. Pro-inflammatory mediators such as tumor necrosis factor-alpha, interleukin-17, and interleukin-23 enhance keratinocyte proliferation and maintain the self-amplifying inflammatory network characteristic of the disease [6].

Current evidence also points to a close relationship between psoriasis and dysfunctional adipose tissue biology. Beyond serving as an energy reservoir, adipose tissue functions as an endocrine organ that releases adipokines capable of modulating metabolism, inflammation, and immune signaling [7]. Disturbances in adipokine secretion have been described in several chronic inflammatory conditions, and similar alterations appear to contribute to psoriasis pathophysiology [8]. Earlier studies reported

abnormal circulating concentrations of leptin, resistin, and adiponectin in psoriasis, supporting the view that adipokine imbalance may participate in disease development and progression [9].

Asprosin is a recently described fasting-induced adipokine released predominantly by white adipose tissue. It promotes hepatic glucose output and has also been linked to appetite control and inflammatory signaling [10]. Increased circulating asprosin concentrations have been reported in obesity, insulin resistance, and type 2 diabetes mellitus [11]. Because psoriasis frequently coexists with metabolic disturbances, asprosin may represent one of the molecules connecting metabolic stress with systemic inflammation in affected patients.

Meteorin-like protein (METRNL) is another secreted factor expressed in adipose tissue, skeletal muscle, and epithelial barrier tissues including the skin and mucosa [12]. Experimental work suggests that METRNL supports anti-inflammatory macrophage polarization and may improve insulin sensitivity [13]. Its expression can also be modulated by inflammatory mediators such as TNF-alpha and IL-17, both of which are central to psoriasis biology [14]. These observations raise the possibility that METRNL may take part in immune regulation in chronic inflammatory skin disease.

Oxidative and nitrative stress pathways may further strengthen the link between metabolism and inflammation in psoriasis. Reactive oxygen and nitrogen species can intensify inflammatory cascades, impair endothelial function, and contribute to tissue injury in chronic inflammatory states [15]. Recent work evaluating low-density lipoprotein receptor-related protein-1 and nitrative stress in psoriasis vulgaris also underlines the contribution of oxidative-metabolic mechanisms to disease expression [16].

Despite growing interest in adipokines, evidence regarding circulating asprosin and METRNL in psoriasis is still sparse, and their relation to clinical severity and treatment response has not been defined clearly. Accordingly, the present study aimed to determine serum asprosin and METRNL levels in patients with psoriasis, explore their relationship with disease severity, and assess whether these markers change after routine treatment.

Materials and Methods

Study Design and Participants

This case–control study was conducted at the Department of Medical Biochemistry in collaboration with the Department of Dermatology at Kahramanmaraş Sütçü İmam University Faculty of Medicine. The study population consisted of patients who were newly diagnosed with psoriasis and healthy controls.

A total of 38 patients with clinically and/or histopathologically confirmed psoriasis and 25 healthy individuals were included in the study. Patients were recruited from the Dermatology outpatient clinic of Kahramanmaraş Sütçü İmam University Faculty of Medicine Hospital between January 2025 and November 2025. The control group consisted of age- and sex-matched healthy volunteers.

Individuals with chronic systemic diseases, autoimmune disorders, malignancies, or alcohol and tobacco use were excluded to minimize potential confounding effects.

Ethical Approval

Ethical approval for the study was obtained from the Ethics Committee of Kahramanmaraş Sütçü İmam University (Decision No: 03, Date: 21.10.2024). The study was conducted in accordance with the Declaration of Helsinki, and written informed consent was obtained from all participants prior to enrollment.

Clinical Evaluation

Demographic and anthropometric data, including age, sex, height, body weight, and body mass index (BMI), were recorded for all participants. Disease severity in patients with psoriasis was evaluated using the Psoriasis Area and Severity Index (PASI).

Blood Sample Collection

Venous blood samples were collected from all participants after overnight fasting. In patients with psoriasis, samples were obtained both before treatment and after routine therapy, whereas control subjects provided a single fasting sample. Blood samples were centrifuged at 3000 rpm for 10 minutes, and serum samples were stored at −80 °C until analysis.

Biochemical Measurements

Serum asprosin and meteorin-like protein (METRNL) levels were measured using commercially available enzyme-linked immunosorbent assay (ELISA) kits according to the manufacturers' instructions. Fasting glucose, insulin, glycated hemoglobin (HbA1c), total cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) levels were analyzed using standard automated methods.

Insulin resistance was calculated using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) according to the following formula:

HOMA-IR = [Fasting insulin (μIU/mL) × Fasting glucose (mg/dL)] / 405

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics software (version 25.0). Continuous variables were expressed as mean ± standard deviation. The normality of data distribution was assessed using the Kolmogorov–Smirnov test.

Between-group comparisons were performed using the independent samples t-test or Mann–Whitney U test, depending on data distribution. Paired samples t-test was used to compare pre-treatment and post-

treatment values in patients with psoriasis. Correlation analyses were conducted using Pearson or Spearman correlation coefficients as appropriate.

Receiver operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic performance of serum asprosin, and the area under the curve (AUC) was calculated. A p-value < 0.05 was considered statistically significant.

Results

A total of 63 individuals were included in the study, comprising 38 patients with psoriasis and 25 healthy controls. The two groups were comparable in terms of age, sex distribution, height, and body weight. However, BMI was significantly higher in the psoriasis group. The demographic and anthropometric characteristics of the study population are presented in Table 1.

Biochemical parameters are summarized in Table 2. There were no significant differences between patients and controls in fasting glucose, insulin, glycated hemoglobin (HbA1c), triglycerides, total cholesterol, low-density lipoprotein cholesterol (LDL-C), or HOMA-IR values. In contrast, high-density lipoprotein cholesterol (HDL-C) levels were significantly lower in patients with psoriasis.

Serum adipokine levels are shown in Table 3. Serum asprosin concentrations were significantly higher in the psoriasis group compared to controls. However, no statistically significant difference was observed in METRNL levels between the two groups.

Within the psoriasis group, treatment-related changes were also evaluated. Serum asprosin levels showed a significant decrease after treatment, whereas METRNL levels did not change significantly. These findings are summarized in Table 4.

Correlation analysis revealed no significant associations between serum asprosin or METRNL levels and fasting glucose, insulin, lipid parameters, HOMA-IR, or PASI score.

Receiver operating characteristic (ROC) curve analysis demonstrated that serum asprosin had moderate discriminatory ability, with an area under the curve (AUC) of approximately 0.67. The identified cut-off value showed high sensitivity but moderate specificity.

In logistic regression analysis, elevated serum asprosin levels were independently associated with psoriasis (odds ratio (OR) = 2.85, 95% confidence interval (CI): 1.32–6.12, p = 0.01). The detailed regression results are presented in Table 5.

Discussion

The present study examined two adipokines with potential metabolic and immunologic relevance in psoriasis and showed three main findings: serum asprosin was elevated in patients with psoriasis, asprosin decreased after treatment, and circulating METRNL did not differ significantly between patients

and controls. Taken together, these results suggest that asprosin may reflect the metabolic-inflammatory milieu accompanying psoriasis, whereas METRNL may have limited value as a systemic biomarker in this setting.

Psoriasis is increasingly understood as a systemic inflammatory disease rather than a disorder confined to the skin **[1,4].** The strong association of psoriasis with obesity, dyslipidemia, insulin resistance, and cardiovascular disease supports the idea that inflammatory skin disease and metabolic dysfunction share overlapping biological pathways**[3,9].** In this context, adipokines are particularly relevant because they influence both energy homeostasis and inflammatory signaling **[7,8].** Previous work has shown altered levels of leptin, adiponectin, and resistin in psoriasis and has suggested that adipose tissue dysfunction may contribute to disease-related inflammation **[17,19,20].**

Our finding of elevated asprosin in psoriasis is biologically plausible. Asprosin is known to increase hepatic glucose release and has been linked to obesity, insulin resistance, and type 2 diabetes mellitus **[10,11,21].** Because psoriasis is often accompanied by metabolic stress, higher circulating asprosin may represent one component of the broader metabolic-inflammatory disturbance observed in these patients. This interpretation is also consistent with the higher BMI and lower HDL cholesterol values seen in our psoriasis group.

The decline in serum asprosin after treatment is another noteworthy observation. Effective therapy in psoriasis can reduce systemic inflammatory burden and may also improve metabolic risk profiles **[22,23].** The treatment-associated fall in asprosin in our cohort may therefore indicate that this adipokine is responsive to changes in inflammatory activity or metabolic imbalance over time. Although the present study cannot establish mechanism, the dynamic behavior of asprosin supports its potential use in monitoring rather than diagnosis alone.

ROC analysis showed that asprosin had only moderate discriminatory performance. This suggests that asprosin should not be interpreted as a single definitive diagnostic marker. Instead, its greatest value may lie in combination with other inflammatory or metabolic indicators, as has been proposed for several biomarker panels in psoriasis**[24,26].**

In contrast, circulating METRNL did not differ significantly between groups and remained unchanged after treatment. METRNL has been reported to exert anti-inflammatory and insulin-sensitizing effects and is expressed in tissues relevant to barrier immunity, including skin **[12,13,25].** Nevertheless, our findings imply that serum METRNL may not adequately capture the systemic inflammatory state of psoriasis. It remains possible that tissue-level expression or local cutaneous regulation is more informative than circulating concentration.

No significant associations were observed between adipokine levels and PASI score. Similar observations have been reported in prior studies evaluating circulating adipokines in psoriasis, where systemic marker levels did not necessarily parallel clinical severity scores **[19,20].** One possible

explanation is that these markers reflect broader metabolic-inflammatory status rather than the extent of visible skin involvement.

The metabolic profile of our psoriasis cohort also deserves attention. Higher BMI and lower HDL cholesterol are both consistent with the recognized cardiometabolic burden of psoriasis. Epidemiologic studies have repeatedly shown that patients with psoriasis face increased cardiovascular risk and excess metabolic comorbidity compared with the general population **[3,28].** More recent mechanistic and population-based studies likewise support a tight relationship between chronic psoriatic inflammation and cardiometabolic disease **[29–33].**

This study has several strengths. It assessed metabolic variables and adipokines together, and it also included paired pre-treatment and post-treatment measurements. At the same time, some limitations should be considered. The sample size was modest, the design does not allow causal inference, and tissue expression of asprosin or METRNL in lesional skin was not analyzed. Additional inflammatory biomarkers were also not incorporated into the model.

To the best of our knowledge, this is the first study to evaluate serum asprosin and METRNL simultaneously in psoriasis and to examine how both markers behave after treatment. Our findings suggest that asprosin may be a candidate biomarker linked to psoriasis-related metabolic-inflammatory changes, whereas METRNL appears less informative as a circulating indicator. Larger prospective and mechanistic studies are needed to clarify how adipokine pathways contribute to psoriasis biology and whether they can be integrated into clinically useful biomarker strategies.

Conclusion

In patients with psoriasis, serum asprosin levels were higher than in healthy controls and decreased significantly after treatment. These findings support a possible relationship between asprosin and the metabolic-inflammatory background of psoriasis. By contrast, serum METRNL did not show meaningful between-group differences or treatment-related change.

Asprosin demonstrated moderate diagnostic performance and may be better viewed as a supportive or follow-up biomarker rather than an independent diagnostic test. Further large-scale prospective studies are required to define the diagnostic, prognostic, and therapeutic relevance of adipokine-related pathways in psoriasis.

Limitations

The study has several limitations. First, the number of participants was relatively small, which may reduce generalizability. Second, the study design does not permit causal interpretation. Third, only circulating levels of asprosin and METRNL were measured, whereas tissue-level expression in psoriatic lesions was not examined. Finally, a broader inflammatory biomarker panel was not included.

Despite these limitations, the study provides preliminary evidence that asprosin may be associated with metabolic-inflammatory alterations in psoriasis and may deserve further investigation in larger cohorts.

Declarations

Ethics Approval:

This study was approved by the Ethics Committee of Kahramanmaraş Sütçü İmam University (Decision No: 03, Date: 21.10.2024).

Informed Consent:

Written informed consent was obtained from all participants.

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Competing Interests:

The authors declare that they have no competing interests.

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Tables

Table 1

Demographic and anthropometric characteristics of patients with psoriasis and healthy controls

Variable	Psoriasis (n = 38)	Control (n = 25)	p-value
Age (years)	41.3 ± 12.1	39.7 ± 11.5	0.58
Sex (M/F)	18 / 20	12 / 13	0.94
Height (cm)	168.4 ± 8.9	169.1 ± 9.1	0.71
Body weight (kg)	78.6 ± 12.4	72.9 ± 11.3	0.09
Body mass index (kg/m²)	27.6 ± 4.3	24.9 ± 3.8	0.031

Values are expressed as mean ± standard deviation. BMI: body mass index. *Statistically significant ($p < 0.05$).

Table 2

Comparison of metabolic and biochemical parameters between psoriasis patients and controls

Parameter	Psoriasis (n = 38)	Control (n = 25)	p-value
Fasting glucose (mg/dL)	94.5 ± 11.8	92.3 ± 10.4	0.42
Insulin (μIU/mL)	10.6 ± 3.9	9.8 ± 3.4	0.39
HOMA-IR	2.46 ± 1.01	2.23 ± 0.89	0.33
HbA1c (%)	5.48 ± 0.42	5.39 ± 0.36	0.29
Triglycerides (mg/dL)	145.2 ± 58.4	132.7 ± 47.5	0.36
Total cholesterol (mg/dL)	192.5 ± 36.2	186.9 ± 31.4	0.47
LDL cholesterol (mg/dL)	118.4 ± 29.3	112.8 ± 25.7	0.41
HDL cholesterol (mg/dL)	43.1 ± 8.7	48.9 ± 9.1	0.020

Values are expressed as mean ± standard deviation. HOMA-IR: homeostasis model assessment of insulin resistance; LDL: low-density lipoprotein; HDL: high-density lipoprotein. *Statistically significant ($p < 0.05$).

Table 3

Comparison of serum adipokine levels between psoriasis patients and controls

Parameter	Psoriasis (n = 38)	Control (n = 25)	p-value
Asprosin (ng/mL)	1.09 ± 0.28	0.90 ± 0.32	0.023
METRNL (ng/mL)	0.78 ± 0.21	0.76 ± 0.24	0.616
Values are expressed as mean ± standard deviation. METRNL: meteorin-like protein. *Statistically significant (p < 0.05).			

Table 4

Changes in serum adipokine levels before and after treatment in patients with psoriasis

Parameter	Pre-treatment	Post-treatment	p-value
Asprosin (ng/mL)	1.09 ± 0.28	0.94 ± 0.26	0.006
METRNL (ng/mL)	0.78 ± 0.21	0.75 ± 0.23	0.416
Values are expressed as mean ± standard deviation. *Statistically significant (p < 0.05).			

Table 5

Logistic regression analysis of serum asprosin levels associated with psoriasis

Variable	Odds Ratio	95% CI	p-value
Asprosin	2.85	1.32–6.12	0.01
CI: confidence interval. *Statistically significant (p < 0.05).			

Figures

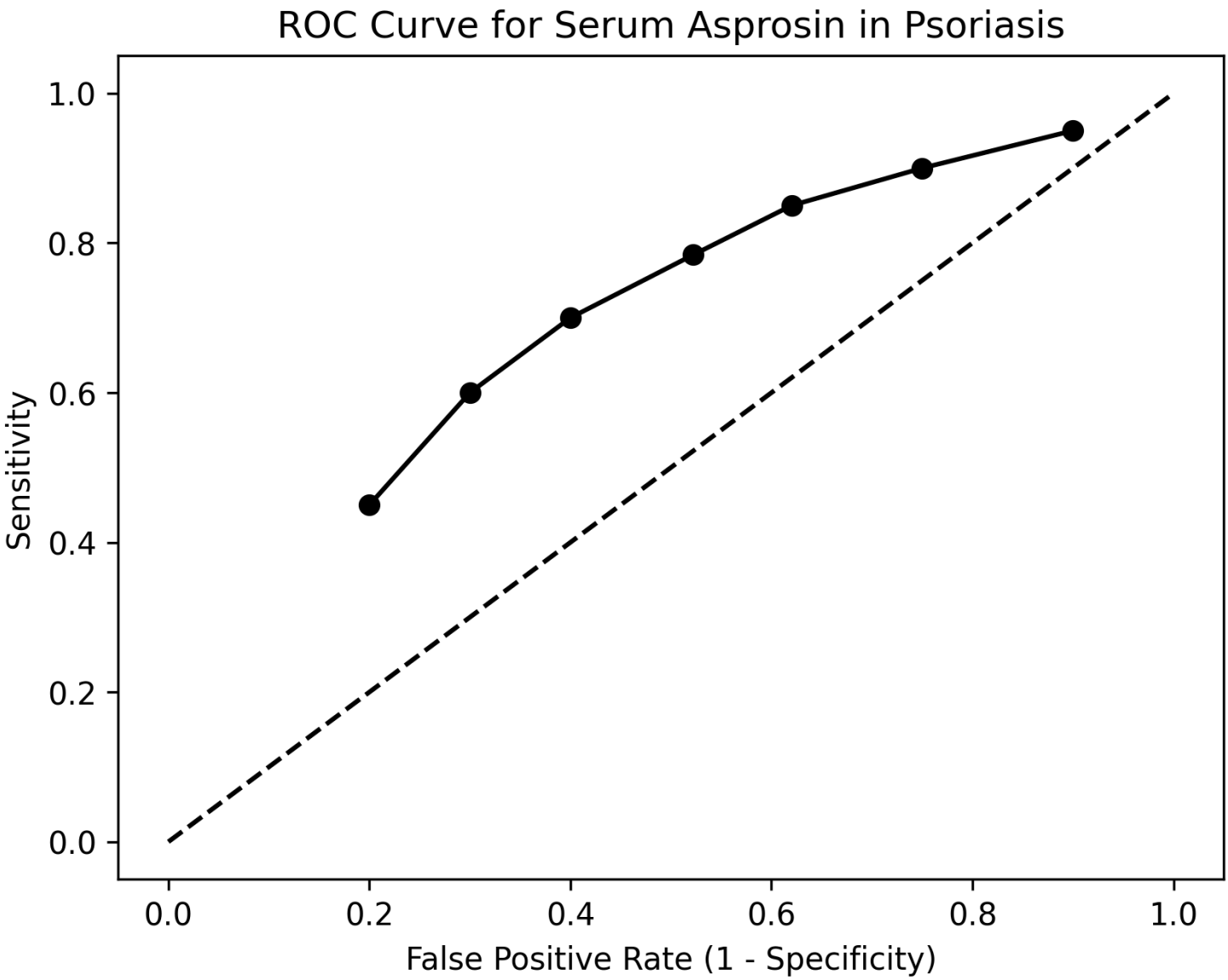


Figure 1

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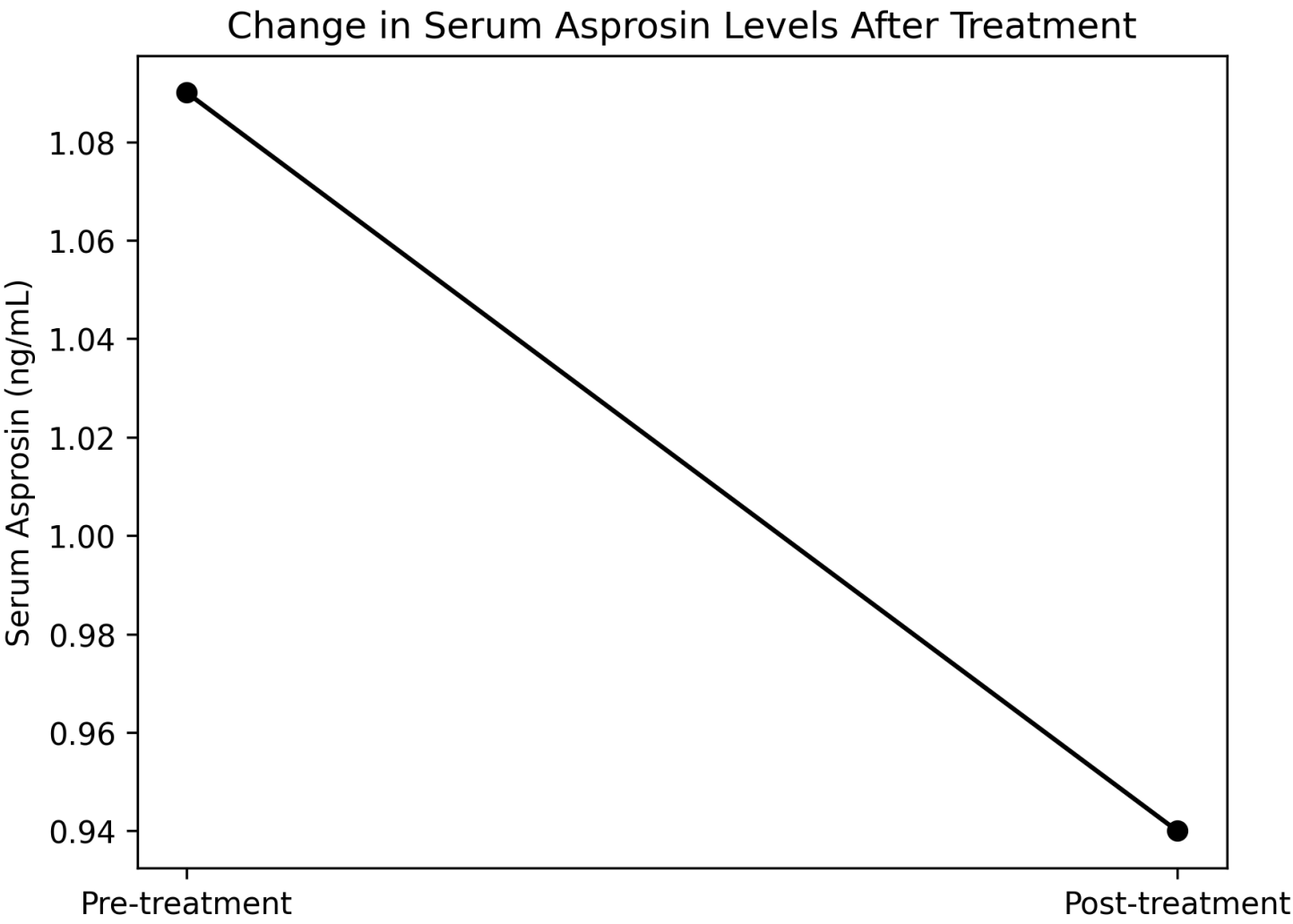


Figure 2

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