P- ISSN 1991-8941 E-ISSN 2706-6703
2025,(19), (01):166 – 173

Journal of University of Anbar for Pure Science (JUAPS)

Open Access

Study the Association of Endostatin with Some Anthropometric Measurements and Blood Pressure in Iraqi Women with Rheumatoid Arthritis

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ARTICLE INFO

Received: 09 / 06 /2024
Accepted: 29/ 08 /2024
Available online: 19/ 06 /2025

DOI: 10.37652/juaps.2024.150362.1264

Keywords:
Anthropometric measurement SBP, DBP, WBCs and RBCs.

ABSTRACT

Rheumatoid arthritis (RA) is an autoimmune disorder of unknown etiology, which affects various body systems. It is characterized by inflammation of synovial joints, mainly small joints, and synoviocyte hyperplasia, leading to joint erosion, bone destruction, and articular damage. The present study aimed to evaluate serum endostatin levels in women with RA and assess the potential association of serum endostatin level with anthropometric measurement and blood pressure parameters. The study included 56 women with RA and 28 age-matched healthy controls (HCs), Endostatin serum level was evaluated by ELISA. The anthropometric parameters recorded included waist width, hip width, thoracic width, neck width, and body mass index (BMI). The patients with RA demonstrated considerably lower serum endostatin levels (ng/mL) than the HCs. Additionally, the patients with RA showed higher levels of white blood cells (WBCs) and lower levels of hemoglobin (Hb; g/dL), but no difference in the level of red blood cells (RBCs) was found between the patients and HCs. Decreased serum endostatin concentration was negatively correlated with the waist-to-thoracic and waist-to-neck ratios, WBC and RBC counts, and systolic blood pressure (mmHg) but positively correlated with waist-to-hip ratio, diastolic blood pressure (mmHg), Hb, and BMI (kg/m^2). The serum levels of endostatin showed extremely weak and nonsignificant correlations with all variables studied.

Introduction

Rheumatoid arthritis (RA) is one of the most common autoimmune diseases, with a complex and unknown etiology. It is primarily diagnosed by the presence of inflammation in the synovial joints, particularly small joints, and synoviocyte hyperplasia, leading to joint erosion, bone destruction, and articular damage.[1] The hallmark clinical feature of RA is symmetrical polyarthritis, which predominantly affects the synovial joints. Moreover, RA may affect other parts of the skeletal system, contributing to the development of tendinopathy, muscle atrophy, and osteoporosis).

It can lead to extra-articular manifestations, which may involve cardiac, cutaneous, pulmonary, hematologic, renal, and neurological systems.[2]

Several risk factors have been correlated with the progression of RA, which can be categorized into genetic, reproductive, and neuroendocrine factors. Additionally, environmental risk factors play a considerable role, including smoking; diet, macrobiota imbalance, viral infections, and bacterial factors.[3] Endostatin is naturally occurring protein formed from collagen XVIII through the action of protease enzymes. It plays a crucial role in many physiological processes, such as the inhibition, proliferation and migration of endothelial cells, tumor growth, and angiogenesis, and is widely recognized as an endogenous inhibitor.[4]

Endostatin has been characterized as an angiogenesis inhibitor, and its mechanism of action may

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be endosttin dependent or heparin independent. At elevated levels, endostatin is secreted and acts to suppres tumor growth by inhibiting angiogenesis and cell migration and promoting apoptosis and cell-cycle stoping. Conversely, reduced levels of endostatin increase angiogenesis, thereby promoting tumor growth.[5] The ability of endostatin to suppress angiogenesis in tumors highlights its potential as a therapeutic option for other pathological conditions characterized by abnormal blood vessel formation, including RA.[6]

Endostatin can contribute to the alleviation of a a metabolic disorder characterized by obesity through its antiangiogenic properties, playing a role as an obesity inhibitor. Through its regulatory effects on angiogenesis and adipose tissue development, endostatin has been shown to inhibit diet-induced obesity. Thus, endostatin plays an important role as an anti-obesity therapeutic.[7] A study conducted in 2003 examined the serum endostatin levels of patients with RA before and after treatment with the tumor necrosis factor-α (TNF-α)-inhibitor infliximab. Initially, no considerable differences in endostatin levels were found between patients with RA and healthy controls (HCs). However, following infliximab treatment, an increase in endostatin level was observed. This result suggests that infliximab help to restore the balance between the levels of growth and inhibitory factors in patients with RA.[8] Another study conducted on patients with RA in 2007 measured the levels of endostatin and vascular endothelial growth factor (VEGF). The results showed that the patients had higher VEGF levels than the HCs, but no statistically significant difference in endostatin level was found between the patients and HCs. The study suggested that an imbalance between inhibitory and growth factors contributes to increased angiogenesis and extra-articular manifestations (EMs) in RA.[9] Anthropometric measurements (AMs) play an essential significant role in estimating growth and development. AMs can be applied across different age groups, from childhood to old age, often relying on various factors, such as gender, race, and social, cultural, and economic conditions.[10] AMs involve the calculation of the body mass index (BMI), which is determined by dividing weight in kilograms by the square of height in meters (kg/m^2). Obesity is associated with the high prevalence of chronic autoimmune and inflammatory conditions. Previous data have indicated that over 60% of patients with RA exhibit a BMI equal to or exceeding 25 kg/m^2, categorizing them as overweight or obese. [11] A study on Iraqi patients with RA showed that their BMIs were considerably higher than those of HCs. The patients had considerably higher, waist-to-hip (W/H), weight-to-thoracic (W/T) and waist-to-neck (W/N) than the HCs.[12] Inflammation has been associated with hypertension in the general population.[13] Hence, investigating the correlation between inflammation and BP is important in RA treatment. Cardiovascular risk is 1.5–2.0 times that in HCs.[14, 15] Moreover, systolic blood pressure (SBP), diastolic blood pressure (DBP), and white blood cell count were considerably higher but the levels of hemoglobin (Hb) were slightly lower in the patients than in the HCs.[12]. Various Iraqi studies have explored the association between inflammation variables and many diseases.[16, 17]. However, the present study is the first to investigate the correlation of endostatin level with some AMs, blood pressure status, and hematological parameters in RA in Iraqi women .

The objective of this study was to precisely measure endostatin levels and evaluate their correlation with anthropometric parameters and blood pressure in healthy subjects and patients.

Materials and Method

A total of 46 women diagnosed with RA were enrolled in this study at the rheumatology department of Al-Yarmouk Teaching Hospital between January 2024 and April 2024. The diagnosis was made using the American Rheumatism Association's 1987 revised principles for the classification of RA [18]. Then, 28 age- and sex-matched HCs with no history of any disorder and autoimmune disease were enrolled. The study received ethical approval by the ethics committee of the University of Anbar, Iraq. Written informed consent was obtained from all the participants.

Venous blood samples were collected from 56 women with RA (35–65 years old) at the Al-Yarmouk Teaching Hospital, Baghdad, Iraq. Blood samples were also collected from the 28 HCs (35–65-years old). The women with RA and HCs provided demographic

information and AMs. Weight was recorded without shoes, socks, baggy clothes, or additional accessories, and was recorded to the nearest 0.1 kg. Height was measured using a stadiometer without shoes and socks and recorded to the nearest 0.01 m. BMI was determined by dividing weight (kg) by the square of height (m^2). AMs were also measured, including neck, hip, waist, and thoracic width (in centimeters). The mean arterial blood pressure (MAP) was determined using the equation **MAP = [(2 x DBP) + SBP]/3**.

To estimate the levels of endostatin, we used ELISA kits (BT-lab Company, China). The other parameters in the study were estimated using a CBC device (Gemmy, India).

Exclusion criteria

Study excluded individuals with other immune issues, chronic illnesses, thyroid diseases, infections, diabetes, inflammation, cancer, or renal diseases.

Statistical Analysis

Analysis performed using GraphPad Prism version 7. Results were expressed as mean ± standard deviation. ANOVA analysis was used to determine significant differences among group means for quantitative data, particularly when analyzing more than two groups. A p value of ≤0.05 was considered statistically significant. Pearson's correlation coefficient was used to assess the correlation between two quantitative variables. Receiver operator curve (ROC) analysis was conducted to evaluate the diagnostic performance specific parameters in distinguishing between patients and HCs. The area under the ROC (AUC) curve provided an estimate of discriminative ability of each parameter. Sensitivity (sen%), specificity (spec%), cut-off value, and likelihood ratio (LHR) were also calculated for each parameter.

Results

In current study, the mean ages of women with RA and HCs were 52 .16 and 48.86 years (p = 0.0926), respectively. The levels of endostatin were significantly lower in women with RA than in HCs (p = 0.0005; **Fig. 1**). The AMs and other parameters of all individuals are summarized in **Table 1**. The AMs, including W/T and W/N ratios, were higher in patients than in the HCs (p = 0.2151, 0.4127). However, no significant difference in W/T ratio was observed (p = 0.7688). The patients had significantly higher BMIs (p = 0.0133). The SBP levels of the patients were significantly higher (p = 0.0024), differences in MAP and RBC and WBC counts were nonsignificant (0.20; p = 0.0625, 0.984), and significantly lower Hb levels were observed in the patents (p = 0.3833, 0.0024). By contrast, No difference in DBP was observed (p = 0.38).

Table 1: Comparisons of Parameters Between Two Studied Groups

	Healthy Controls			RA Patients			p value
	Mean	SD	SEM	Mean	SD	SEM	
Age years	48.86	9.232	1.745	52.16	8.085	1.080	0.0962
BMI kg/m2	29.08	3.842	0.7261	32.07	5.617	0.7574	0.0133
W/H	0.8801	0.0374	0.0071	0.8595	0.0829	0.0111	0.2151
W/T	0.9424	0.0235	0.0045	0.9450	0.0419	0.0056	0.7688
W/N	2.515	0.2153	0.0401	2.570	0.3231	0.0432	0.4127
SBP mmHg	126.4	8.112	1.533	135.8	14.83	1.981	0.0024
DBP mmHg	80.18	4.406	0.8326	78.84	7.446	0.9950	0.3833
MAP mmHg	95.58	4.854	0.9173	97.83	8.657	1.157	0.2067
WBCs* 10^6/mL	6.780	1.400	0.2645	8.011	3.294	0.4402	0.0625
RBCs* 10^3/mL	4.545	0.4304	0.0814	4.548	0.5012	0.0670	0.9846
Hb g/dL	12.98	0.8929	0.1687	12.24	1.066	0.1425	0.0024

Endostatin ng/mL	10.34	1.925	0.3637	8.487	2.330	0.3113	0.0005

Table 2: Effect of Menstrual Cycle on Endostatin Levels

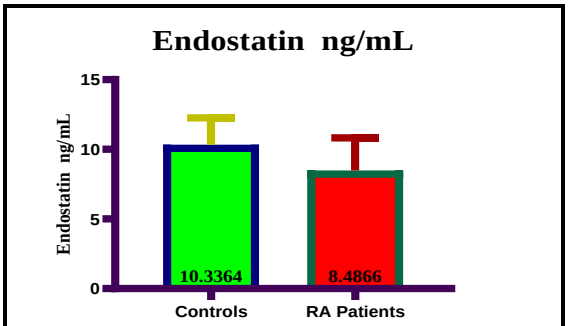

Fig. 1: Mean + SD for Endostatin in Control and RA Patients

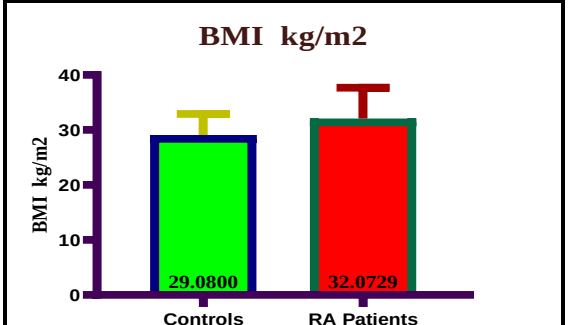

Fig. 2: Mean + SD for BMI in Control and RA Patients

Effect of Menstrual Cycle on Endostatin in Women with RA

Table2 summarizes the impact of menstrual cycle on endostatin level. The participants were divided into two groups (postmenopausal and menopausal). Postmenopausal women with RA had lower endostatin levels (8.5857) than the postmenopausal women in the HC group (10.729). Menopausal women with RA had lower endostatin levels (8.38575) than the HCs (9.9436).

Endostatin ng/mL	N	Mean	Std. Deviation	Std. Error	Min.	Max.
RA Postmenopausal W. (1)	28	8.5857	2.49566	.47164	5.16	12.55
RA menstrual cycle W. (2)	28	8.3875	2.19277	.41439	4.36	11.77
HCs Postmenopausal W (3)	14	10.729	2.12637	.56830	8.24	16.88
HCs menstrual cycle W. (4)	14	9.9436	1.68484	.45029	7.58	13.96
Total	84	9.1032	2.36035	.2575	4.36	16.8

Table 3 summarizes the effects of menstrual cycle on the studied groups. Postmenopausal women with RA showed higher in endostatin Levels than menopausal women with RA but significantly lower in endostatin levels than the postmenopausal women in the HC group. Menopausal women with RA showed significantly lower endostatin levels than postmenopausal women in the HC group and lower endostatin levels than menopausal women in the HC group. Postmenopausal women in the HC group showed higher endostatin levels than menopausal women in the HC group.

Table 3: Probability Values of Studied Groups

Dependent Variable	(I) groups	(J) groups	Mean Difference (I-J)	Std. Error	Sig.
Endostatin ng/mL	RA Postmenopausal W. (1)	2	.19821	.59287	.987
		3	-2.14357*	.72611	.012
		4	-1.35786	.72611	.249
	Menstrual cycle W. (2)	3	-2.34179*	.72611	.010
		4	-1.55607	.72611	.149
	HCs Postmenopausal W. (3)	4	.78571	.83844	.785

As shown in **Table 4** we don't notice any important correlation between endostatin and other studied parameters.

Table 4: Association of Endostatin with Studied Parameters.

Parameter	r (Endostatin ng/mL)	p-value
Endostatin ng/mL	1	0
Hb g/dL	0.095	0.388
WBCs*10^6/mL	-0.142	0.199
RBCs*10^3/mL	-0.159	0.148
DBP mm Hg	0.119	0.282
SBP mm Hg	-0.056	0.610
Age Years	0.064	0.561
BMI kg/m^2	0.038	0.736
W/H	0.014	0.896
W/T	-0.065	0.560
W/N	-0.026	0.815
MAP mm Hg	0.035	0.754

ROC analysis indicated that several biomarkers were useful in distinguishing between patients with RA and HCs. **Table 5** shows the ROC outcomes, ranked in descending order diagnostic accuracy: SBP (AUC = 0.7063; cut-off value > 132.0; Sen%: 50; Spec%: 78.57; and LHR: 2.333), Hb (AUC = 0.6993; cut-off value < 12.45; Sen%: 62.50; Spec%: 64.29; and LHR: 1.750),

endostatin (AUC = 0.6846; cut-off value: < 9.695; Sen%: 60.71; Spec%: 57.14; LHR: 1.417; **Fig. 3**), BMI (AUC = 06682; cut-off value > 29.09; Sen%: 67.27; Spec%: 64.29; LHR: 1.884), DBP (AUC = 0.6008; cut-off value < 77.50; Sen%: 42.86; Spec%: 85.71; LHR: 3), WBC count (AUC = 0.5966; cut-off value > 7.010; Sen%: 55.36; Spec%:57.14; LHR: 1.292), W/N (AUC = 0.5839; cut-off value > 2.595; Sen%: 55.36; Spec%: 53.57; LHR: 1.192), MAP (AUC = 0.5529; cut-off value > 95.83; Sen%: 60.71; Spec%: 50; LHR: 1.214), RBC count (AUC = 0.5156; cut-off value < 4.550; Sen%: 58.93; Spec%: 50; LHR: 1.179), W/T (AUC = 0.5102; cut-off value: > 0.9456; Sen%: 50; Spec%: 46.43; LHR: 0.9333), and W/H (AUC = 0.5099; cut-off value < 0,8835; Sen%: 46.43; Spec%: 46.43; LHR: 0.8667).

Table 5: Area Under ROC Curve for all Analyzed Parameters in Studied Subjects

Parameter	AUC	Positive if COV	Sensitivity%	Specificity%	Likelihood Ratio
Age years	0.5906	> 51.50	50.00	50.00	1.000
BMI kg/m2	0.6682	> 29.09	67.27	64.29	1.884
W/H	0.5099	< 0.8835	46.43	46.43	0.8667
W/T	0.5102	> 0.9464	50.00	46.43	0.9333
W/N	0.5839	> 2.595	55.36	53.57	1.192
SBP mmHg	0.7063	> 132.0	50.00	78.57	2.333
DBP mmHg	0.6008	< 77.50	42.86	85.71	3.000
MAP mmHg	0.5529	> 95.83	60.71	50.00	1.214
WBCs*10^6/mL	0.5966	> 7.010	55.36	57.14	1.292
RBCs*10^3/mL	0.5156	< 4.550	58.93	50.00	1.179
Hb g/dL	0.6993	< 12.45	62.50	64.29	1.750
Endostatin ng/mL	0.6846	< 9.695	60.71	57.14	1.417

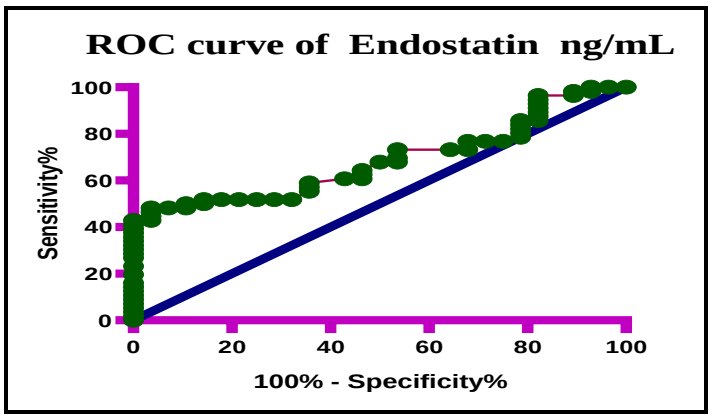

Fig.3: Area under ROC curve for Endostatin in studied subjects.

Discussion

RA is well-established chronic inflammatory disorder characterized by joint destruction, synovitis, synovial hyperplasia, infiltration of inflammatory cells in the stroma, angiogenesis, pannus formation, and progressive degradation of cartilage and bone tissues.[19] The infiltration of inflammatory factors into the inner lining of the synovial membrane and the imbalance between pro- and anti-angiogenic factors lead to persistent inflammation and generation of microvessels, expanding the synovial layer. Newly formed synovial tissues not only increase distance to the nearest blood vessel but also increase the demand for oxygen and nitration.[20]

Endostatin is produced from collagen cleavage is known as C-terminal proteolytic fragment of collagen XVIII and is found in vascular basement membrane zones in multiple organs.[21] A study conducted on mice indicates that recombinant human endostatin inhibits RA by reducing the expression of VEGF, suppressing TNF-α and interleukin-1 (IL-1) production.[22] Current study's results revealed significant decreases in the endostatin levels of women with RA compared with the HCs. These results are consistent with a previous study on patients RA, which reported reduced levels of endostatin and VEGF (angiogenic factor) in the patients compared with the HCs. These findings confirmed that an imbalance between inhibitory and growth factors leads to angiogenesis and contributes to EMs in RA.[9] The menstrual cycle is characterized by fluctuations in the absolute and relative levels of the hormones regulated by

the hypothalamic–pituitary–ovarian axis. These hormonal changes influence cellular function, cytokine production and heat shock protein expression. Consequently, the menstrual cycle affects immune cell activity and has been linked to the modulation of systemic diseases, such as autoimmune diseases, asthma, diabetes, and RA.[23] Postmenopausal women with RA had lower endostatin levels than the menopausal women with RA, significantly lower endostatin levels than postmenopausal women without RA, and lower endostatin levels in healthy menopausal women. Menopausal women with RA showed significantly lower endostatin levels than healthy postmenopausal women and lower endostatin levels than healthy menopausal women. These results suggest that the menstrual cycle affects endostatin levels.

W/H and W/N ratios were higher in patients with RA than in the HCs, and no significant difference in W/T ratio was found. These results are consistent with a previous study regarding W/H and W/N but not in W/T [12] BMIs were significantly higher in the patients with RA. This result is consistent with previous studies showing significantly higher BMIs in patients with RA compared with HCs.[11, 12] Individuals with obesity are more likely to develop RA. Hence, the accumulation of white adipose tissues contributes to this outcome because they secrete adipokines, such as leptin, adiponectin, resistin, and visfatin, which are involved in immunity and inflammation. These results indicate increased risk of RA in individuals with high BMIs.[24] Our study showed significantly elevated SBP, reduced DBP, and elevated MAP, contradicting a previous study showing higher levels of DBP and SBP.[12]

Our study showed a significant decrease in Hb levels and nonsignificant increase in WBC counts in patients with RA compared with HCs. This result disagrees with a previous study showing a slight decrease in Hb levels and a significant increase in WBC count.[12]

This study has many limitations. First, the sample size was not large enough. Second, analyses were conducted in private laboratories**.**

Conclusion

Serum endostatin levels showed extremely weak and nonsignificant correlations with all the variables studied, and the value of the AUC showed no statistical significance. Hence, this variable cannot be used to predict or diagnose RA in Iraqi women in Anbar Governorate.

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P- ISSN 1991-8941 E-ISSN 2706-6703 *Journal of University of Anbar for Pure Science (JUAPS)* Open Access
2025,(19), (01):166 – 173

دراسة ارتباط الاندوستاتين مع بعض القياسات الجسمية وضغط الدم في النساء العراقيات المصابات بالتهاب المفاصل الرثوي

نسيبة العلواني ، شاكر فارس طليب

قسم الكيمياء ، كلية التربية للعلوم الصرفة ، جامعة الانبار

الخلاة:

مرض التهاب المفاصل الرثوي هو من أكثر الأمراض المناعية شيوعا، يؤثر في أجهزة الجسم ويشخص من خلال الالتهاب في المفاصل الزلالية خاصة المفاصل الصغيرة. تهدف هذه الدراسة إلى تقييم المستويات المصلية للاندوستاتين في النساء المصابات بالتهاب المفاصل الرثوي وقياس أي ترابط محتمل بين مستويات الاندوستاتين والقياسات البشرية ومقاييس ضغط الدم. تضمنت هذه الدراسة 56 امرأة مصابة بالتهاب المفاصل الرثوي و 28 امرأة غير مصابة مطابقة للمصابات من حيث الجنس والعمر، تم قياس مستوى الاندوستاتين في المصل باستخدام تقنية الامتزاز المناعي المرتبط بالأنزيم ELISA)، وتم حساب محيط الخصر(W.W)، محيط الورك(W.H)، محيط الصدر (W.T)ومحيط الرقبة (W.N)وأيضا حساب مؤشر كتلة الجسم (BMI)، وقياس ضغط الدم الانقباضي والانبساطي وحساب متوسط ضغط الدم الشرياني لجميع الأفراد . المستويات المصلية للاندوستاتين كانت أقل بالمرضى مقارنة بالأصحاء، مستويات الاندوستاتين المنخفضة أظهرت ارتباطا ايجابيا مع نسبة الخصر إلى الورك(W/H)، ضغط الدم الانبساطي (DBP) ومع مؤشر كتلة الجسم(BMI) ، في حين أظهرت المستويات المنخفضة للاندوستاتين ارتباطا سلبيا مع نسبة الخصر إلى الصدر(W/T) ، نسبة الخصر إلى الرقبة وضغط الدم الانقباضي(SBP) ، توصلت هذه الدراسة أن المستويات المصلية للاندوستاتين مرتبطة ارتباطا ضعيفا جدا مع جميع المتغيرات المدروسة.

الكلمات المفتاحية: القياسات البشرية، ضغط الدم الانقباضي، كريات الدم البيضاء، كريات الدم الحمراء، العامل المضاد لتولد الأوعية الدموية.