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RESEARCH ARTICLE

Assessment of Decorin Level and Some Biochemical Parameters in Iraqi Patients with Diabetic Nephropathy

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ABSTRACT

Diabetic nephropathy (DN) is a serious kidney complication that can occur in individuals with diabetes; around 20% to 30% of individuals with Type 2 diabetes (T2D) develop diabetic nephropathy (DN), which is a persistent microvascular complication. The DN is often considered the leading contributor to end-stage renal failure. This study aimed to assess decorin levels and their correlation with various biochemical variables among three different groups: 40 patients with T2DM nephropathy (G1), 40 patients with T2DM without nephropathy (G2), and a healthy control group (G3). The study included a total of 120 participants, divided into three groups T2DM with DN (G1), T2DM without DN (G2), and healthy control group (G3) which is consisted of 40 males and 40 females, with ages ranging from 35 to 63 years. The participants were recruited from patients attending the AL-Kindi teaching hospital in Baghdad, Iraq. Data collected from participants revealed a substantial and statistically significant increase ($P \leq 0.01$) in the levels of fasting blood glucose (FBG), insulin, homeostatic model assessment for insulin resistance (HOMA-IR), blood urea (B.urea), blood urea nitrogen percentage (BUN%), serum creatinine (S.creatinin), and uric acid. The results indicated that there was a highly significant increase ($P \leq 0.01$) in decorin, insulin and HOMA-IR levels in G1 group compared to G2 and G3 groups.. In conclusion, positive correlation coefficients between decorin levels and insulin, HOMA-IR in both the TD2M with and without DN. This suggests that decorin levels play an essential role in the pathophysiology of diabetic patients.

Keywords: Decorin, Diabetic nephropathy, Insulin resistance, Kidney function test, T2DM

Introduction

Hyperglycemia, a prevalent symptom of T2DM, is characteristic of a multifaceted and varied cluster of chronic metabolic disorders. The key distinguishing features of T2DM include insulin resistance and, over time, impairment of pancreatic cell function.[1] Research indicates that diabetic nephropathy (DN), a persistent microvascular complication of T2DM, is prevalent in approximately 20% to 30% of patients. Many experts consider it to be the leading cause of end-stage renal failure, necessitating renal replacement therapy. Numerous studies have revealed that patients with T2DM already frequently experience cardiovascular issues. The connection between hyperglycemia and the emergence of cardiovascular issues has long been understood.[2,3] In many connective tissues, a small leucine-rich proteoglycan called decorin can be found. Decorin plays an important role in facilitating the formation of collagen fibrils and maintaining the proper spacing between these fibrils. These functions are essential for ensuring the transparency of the cornea.[4] The building blocks of decorin are a core protein with a tandem leucine-rich repeat domain and a single glycosaminoglycan chain joined at the N terminus. The majority of decorin's

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ligand- binding domains are found at its core protein, although glycosaminoglycan chain plays a role in some interactions with ligands.[5] Decorin does not require dimerization for stabilization, as it can readily switch between homodimers and monomers. Following a spinal cord injury, decorin has demonstrated the ability to promote nerve axon growth in live organisms. Additionally, *in vitro* study has shown that decorin can effectively regulate and control inflammatory responses.[6] Previous research has demonstrated that decorin can enhance the regeneration of corneal sensory nerves and decrease the recruitment of macrophages in the week following a sterile epithelial injury.[7,8] Furthermore, it has been associated with an increased density of corneal intraepithelial dendritic cells (DCs) within six hours after topical application. Decorin (DCN) is one of the components found in the extracellular matrix. Many studies have focused on examining the interaction between kidney function, liver function, and hormonal regulation, and examining their association with various diseases and potential complications.[9,10]

Therefore, the main aim of this study was to evaluate decorin levels and their correlation with various biochemical parameters in distinct groups, including T2DM patients with and without nephropathy, as well as a control group. Also, to investigate the potential role of decorin in the increase of diabetes-related complications, particularly nephropathy.

Materials and methods

This study involved a total of 120 participants who were divided into three groups: Group 1 (G1) consisted of 40 T2DM patients with DN stage II, Group 2 (G2) included 40 T2DM patients without DN, and Group 3 (G3) comprised 40 healthy controls. There were an equal number of males and females, with a total of 60 participants of each sex. The age of the participants ranged from 35 to 63 years, mean ± SD (54.975 ± 10.21). The study was conducted at AL-Kindi Teaching Hospital in Baghdad, Iraq, spanning from November 2022 to June 2023. Data collection included information on age, sex, disease duration, measured blood pressure, and Body mass index (BMI). The BMI was calculated by dividing each patient's weight in kilograms by the square of their height in meters (kg/m^2). Venous blood samples were collected from fasting participants, and commercial kits (Biolabo SA-France) were used to quantify various biomarkers, including (HbA1c), FBG, total cholesterol, triglycerides (TG), and high-density lipoprotein cholesterol (HDL-c). Additionally, all patients underwent the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) calculation (HOMA-IR = Fasting Insulin × Fasting Blood Glucose / 204). Enzyme-linked immunosorbent assay (ELISA) kit tests to determine their insulin and decorin levels. To assess the significance of differences in the mean values among the various groups, an analysis of variance (ANOVA) was performed, using SPSS program version 23, with significance level set at $P \leq 0.05$, indicating statistical significance. Furthermore, the relationships between the different factors under examination were evaluated using the Pearson correlation coefficient (r) test.

Results and discussion

The results found a statistically significant increase in BMI, and systolic blood pressure (SBP) in G1 group as compared to G2 and G3 (including T2D with nephropathy, T2D without nephropathy, and the control group), as shown in Table 1. Conversely, there were no significant differences observed in age, and diastolic blood pressure (DBP) between these distinct groups, Table 1.

In Table 2. the results revealed, a significant increase ($p \leq 0.01$) in the levels of FBG, Insulin, HOMA-IR, B.urea, BUN%, S.creatinin, and uric acid of G1 group compared as to G2 and G3.

Furthermore, the results, as shown in Table 3, demonstrated a noteworthy and statistically significant increase ($p \leq 0.01$) in decorin levels across the groups, including in G1 group as compared to G2 and G3.

The correlation study revealed highly significant positive correlation coefficients between Decorin

Table 1. Anthropometric measurements among the study groups.

	Mean ± SD			
Variables	(G1) n = 40	(G2) n = 40	(G3) n = 40	p-value
Age (year)	54.975 ± 10.21	54.12 ± 10.35	53.6 ± 10.42	0.835 NS
BMI (kg/m^2)	29.45 ± 3.963	29.022 ± 4.434	26.36 ± 4.011	0.02 S
SBP (mm Hg)	19.78 ± 2.99	16.87 ± 2.46	12.70 ± 1.35	0.00 HS
DBP (mm Hg)	10.63 ± 0.99	8.95 ± 0.80	8.57 ± 0.94	0.175 NS

Data are expressed as mean ± SD * Significance: P-Value ≤ 0.05 ** High significance: P-Value ≤ 0.01,

Table 2. Investigation of some biochemical parameters among the study groups.

	Mean ± SD			
Variables	(G1) n = 40	(G2) n = 40	(G3) n = 40	p-value
Urea (mg/dL)	52.03 ± 16.516	27.66 ± 5.976	24.53 ± 6.6106	0.00 HS
BUN	24.4823 ± 7.58	12.895 ± 2.793	11.625 ± 3.297	0.00 HS
Creatinine (mg/dL)	1.7813 ± 0.59	0.827 ± 0.161	0.786 ± 0.1798	0.00 HS
Uric acid (mg/dL)	5.117 ± 0.7256	4.316 ± 0.882	3.913 ± 0.727	0.001 HS
FBS (mg/dL)	258.99 ± 88.92	191.46 ± 76.02	111.54 ± 27.35	0.00 HS
HbA1c	12.79 ± 1.06	7.87 ± 1.12	5.49 ± 0.32	0.001 HS
Insulin (μIU/ml)	32.73 ± 13.379	27.57 ± 9.34	15.21 ± 3.73	0.004 HS
HOMA-IR	19.96 ± 8.96	14.41 ± 6.79	4.18 ± 1.36	0.00 HS

Data are expressed as mean ± SD * Significance: P-Value ≤ 0.05 ** High significance: P-Value ≤ 0.01.

Table 3. Decorin level among the study groups.

	Mean ± SD			
Variable	(G1) n = 40	(G1) n = 40	(G3) n = 40	p-value
Decorin (ng/ml)	98.625 ± 62.84	57.442 ± 29.756	44.57 ± 10.57	0.00 HS

Data are expressed as mean ± SD * Significance: P-Value ≤ 0.05** High significance: P-Value ≤ 0.01.

Table 4. Correlation coefficients of serum Decorin with all variables in G1 and G2.

	G1		G2	
Variables	R	P	R	P
Ages (years)	0.464**	0.003	0.328*	0.039
BMI (kg/m^2)	0.374*	0.017	0.493**	0.001
Duration (years)	0.131	0.420	0.128	0.431
SBP (mm Hg)	0.381*	0.012	0.08	0.032 Sig.
DBP (mm Hg)	0.154	0.420	–0.34	0.041 Sig.
FBG (mg/dL)	0.558**	<0.001	0.516**	0.001
HbA1c	0.630**	<0.001	0.371*	0.019
Insulin (μIU/ml)	0.476**	0.002	0.208	0.054
HOMA-IR%	0.327*	0.016	0.301*	0.01
Urea (mg/dL)	0.708**	<0.001	0.179	0.270
BUN%	0.434**	0.005	0.201	0.213
Creatinin (mg/dL)	0.0389*	0.013	0.070	0.668
Uric acid (mg/dL)	0.628**	<0.001	0.046	0.776

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

levels with various factors in G1, including age, FBG, insulin, Urea, BUN% and uric acid as well as a significant positive correlation with BMI, SBP, HOMA-IR and creatinine. However, no correlation coefficients were observed between Decorin levels with duration and DBP, Table 3.

In G2 group, there was a significant positive correlation coefficient between Decorin levels with age, HbA1c and HOMA-IR while there was a highly positive correlation with BMI and FBS. Conversely, no correlation coefficients were found between Decorin levels with duration, SBP, DBP, insulin levels, B. Urea, BUN%, serum creatinine, and uric acid in this group, Table 4.

The kidneys have several key mechanisms to maintain glucose homeostasis within the body, including gluconeogenesis, renal glucose consumption, and the reabsorption of glucose in the proximal tubules.[11] Notably, diabetes is a significant contributor to kidney failure, responsible for approximately 44% of cases worldwide.[12] Prolonged elevated levels of glucose in the bloodstream can exacerbate beta-cell dysfunction and insulin resistance, thereby disrupting glucose homeostasis. The SGLT2 inhibitors have emerged as the preferred treatment for individuals with T2DM due to their capacity to lower glucose levels, promote weight loss, and provide protection against cardiovascular issues, including heart failure.[13] It is important to note that insulin resistance can lead to metabolic dysfunction and kidney damage, as it directly hampers mitochondrial function and protection. Conversely, reduced mitochondrial performance

can diminish insulin sensitivity.[14] Our research findings suggest a direct correlation between FBG levels, insulin resistance, and the declining renal function in diabetic patients. Numerous research studies have demonstrated the involvement of HOMA-IR in the development of DN. These studies have indicated that higher HOMA-IR levels may be associated with an increased risk of microalbuminuria. In individuals with T2DM who have early-stage renal conditions, elevated serum glucose levels have been observed.[15,16] While inflammation is known to play a role in the progression and outcomes of Chronic Kidney Disease (CKD), the exact relationship between inflammation and the onset of the disease remains a topic of debate. Similar to other chronic disorders, CKD is characterized by a persistent, low-grade inflammation, which the kidneys are particularly susceptible to, and affecting them in various ways. In comparison to the control group in this study, diabetics with nephropathy exhibited higher levels of decorin. This phenomenon may be attributed to the versatile nature of decorin as a protein, which interacts with a variety of receptors, enzymes, and cytokines. It has been found that decorin's influence extends to various biological processes, including autophagy, the cell cycle, inflammation, and angiogenesis. So, decorin is a good indicator for diabetic nephropathy. Several crucial components support decorin's function in autoimmune and inflammatory disorders. Firstly, decorin, categorized as one of the Damage-Associated Molecular Patterns (DAMPs), can interact with Toll-Like Receptors (TLRs) or Advanced Glycation End-Product (AGE) receptors, contributing to the activation of innate immune cells. Secondly, the immune system may generate autoantibodies against decorin, potentially hindering the proper functioning of soluble decorin. Thirdly, endothelial or epithelial cells can employ decorin to regulate autophagy processes. Lastly, decorin possesses the capability to counteract the effects of (TGF-β), particularly in the context of fibrosis.[17] It is important to note that in the normal kidney, decorin is primarily expressed by renal fibroblasts and is consequently predominantly located in the peritubular area as a secreted molecule. In contrast, the typical glomerulus contains only minimal levels of decorin produced by mesangial cells.[18]

In the absence of collagen type I, a binding partner, decorin does not remain localized at its site of production. Instead, it can be eliminated through diffusion into the circulation or, alternatively, via endocytosis. In various experimental and human nephropathies, there is an accumulation of decorin in regions characterized by tubulointerstitial fibrosis. This accumulation of decorin may serve as a valuable indicator of the progression of CKD.[19] In fact, decorin is primarily sequestered within the Extracellular Matrix (ECM) and may not always be readily available to neutralize TGF-β or function as a ligand for various receptors. Consequently, recent research suggests that the quantity of decorin observed in tissue sections may not always accurately reflect its physiological impact.[20] Research has reported that adipocyte hypertrophy is associated with insulin resistance.[21] Interestingly, despite experiencing impaired glucose regulation due to increased weight gain, there was no observable evidence of enlarged adipocyte size. This suggests that decorin depletion negatively affected glucose homeostasis independently of adipocyte hypertrophy.[22] A study that found a positive correlation between decorin levels and blood pressure in individuals with T2DM, both with and without nephropathy, aligns with other research indicating that decorin acts as a protective agent against DN by inhibiting apoptosis and fibrosis.[23] Moreover, therapy involving the overexpression of decorin using a recombinant adeno-associated viral (rAAV) vector has been shown to mitigate the effects of hypertension-induced cardiac fibrosis and improve cardiac function in individuals with spontaneous hypertension. Previous studies have demonstrated that Decorin promotes angiogenesis in diabetic hearts.[24,25] Despite the limited availability of data, we have observed a significant association between decorin levels and insulin resistance. In one study, it was found that individuals with T2DM had higher levels of decorin compared to those with normal glucose tolerance. However, animal research has indicated that decorin might actually facilitate glucose tolerance. Consequently, elevated decorin levels before the onset of T2DM may represent a compensatory mechanism.[26] The development of glomerular hyper filtration due to elevated glomerular capillary pressures is associated with the onset of DN in patients with T1DM. Consequently, proteinuria becomes evident, and hypertension often develops. This well-established connection is a key factor in the progression of renal disease in individuals with diabetes.[27,28] In contrast, the etiology of DN in individuals with T2DM is not fully comprehended, as multiple contributing factors may operate concurrently. However, angiotensin-converting enzyme medications have proven to be beneficial in delaying renal impairment in hypertensive individuals with T1DM and proteinuria. These medications achieve this by reducing systemic blood pressure and mitigating glomerular capillary pressure.[29,30]

Conclusion

Decorin levels can be associated with certain pathological processes, including complications of diabetes. In both the T2DM patients with nephropathy and T2DM group without nephropathy, the study observed strong positive correlation coefficients between Decorin levels and both insulin and HOMA-IR. These conclusions suggest that Decorin levels play a significant role in the pathophysiology of diabetes. Additionally, elevated Decorin levels may contribute to the development of various complications.

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Authors' declaration

- Conflicts of Interest: None.
- We hereby confirm that all the Tables in the manuscript are ours.
- No animal studies are present in the manuscript.
- Authors sign on ethical consideration's approval.
- Ethical Clearance: The project was approved by the local ethical committee at Mustansiriyah University.

Authors' contribution statement

F. S. A. contributed to the study design. Z.M. I. Carried out the experimental work, data collection and data analysis. Z.M. I. and F. S. A. contributed to the results discussion, writing, drafting and editing of the paper. F. S. A. checked and approved the overall manuscript, and all authors N. T. T., Z.M. I., A.H.I. and F. S. A. approved the final version of the manuscript.

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تقييم مستوى الديكورين وبعض المتغيرات الكيموحيوية لدى المرضى العراقيين المصابين باعتلال الكلية السكري

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[2]مركز السكر الوطني، الجامعة المستنصرية، بغداد، العراق.

الخلاصة

يصاب حوالي 20% إلى 30% من ألاشخاص المصابين بداء السكري من النوع الثاني باعتلال الكلى السكري ، وهو أحد المضاعفات المستمرة للاوعية الدموية الدقيقة. وكثيرا ما يعتبر العامل الاكثر شيوعا الذي يؤدي إلى الفشل الكلوي في المرحلة النهائية والذي يتطلب استبدال الكلى. هدفت هذه الدراسة إلى تقييم مستويات الديكورين والمتغيرات البيوكيميائية الاخرى بين المجموعات المختلفة داء السكري من النوع الثاني مع اعتلال الكلية، داء السكري من النوع الثاني بدون اعتلال الكلية ، والاصحاء. شملت هذه الدراسة مائة وعشرين 120 فئة مشاركة إلى ثلاث مجموعات : المجموعة الاولى 40 مصابين بداء السكري من النوع 2 مع اعتلال الكلية، المجموعه الثانية: 40 مصابين بداء السكري من النوع 2 بدون اعتلال الكلية ومقارنتها مع 40 من الاصحاء, 20 ذكراً و 20 أنثى (تتراوح أعمارهم بين 65-35 سنة)يراجعون مستشفى الكندي التعليمي، بغداد العراق. يشمل جمع البيانات لجميع المرضى العمر والجنس ومدة المرض وقياس ضغط الدم ومؤشر كتلة الجسم. كانت هناك زيادة معنوية للغاية في مستويات السكر في الدم، الانسولين، مقاومة الانسولين، اليوريا، الكرياتنين، حمض اليوليك بين المجموعات الثلاثة داء السكري من النوع الثاني مع اعتلال الكلية، داء السكري من النوع الثاني بدون اعتلال الكلية ، والاصحاء كما توجد زيادة معنويه كبيره في مستويات الديكورين بين المجموعات المختلفة. الاستنتاج: هناك معاملات ارتباط إيجابية للغاية بين مستويات الديكورين مقابل الانسولين، مقاومة الانسولين في كلا المجموعتين داء السكري من النوع الثاني مع اعتلال الكلية، داء السكري من النوع الثاني بدون اعتلال الكلية وهذا بسبب مستويات الديكورين تلعب دوراً هاماً في الفسيولوجيا المرضية لدى مرضى السكري تظهر، أولها أمراض القلب والاوعية الدموية.

الكلمات المفتاحية: ديكورين، اعتلال الكلية السكري، مقاومة الانسولين، اختبار وظائف الكلى، داء السكري النوع الثاني.