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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

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GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board since 1994. GMN carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

GMN is indexed in MEDLINE, SCOPUS, PubMed and VINITI Russian Academy of Sciences. The full text content is available through EBSCO databases.

GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения. Журнал индексируется в MEDLINE, отражён в базе данных SCOPUS, PubMed и ВИНИТИ РАН. Полнотекстовые статьи журнала доступны через БД EBSCO.

GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

ჟურნალი ინდექსირებულია MEDLINE-ის საერთაშორისო სისტემაში, ასახულია SCOPUS-ის, PubMed-ის და ВИНИТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებიდან.

WEBSITE

www.geomednews.com

К СВЕДЕНИЮ АВТОРОВ!

При направлении статьи в редакцию необходимо соблюдать следующие правила:

1. Статья должна быть представлена в двух экземплярах, на русском или английском языках, напечатанная через **полтора интервала на одной стороне стандартного листа с шириной левого поля в три сантиметра**. Используемый компьютерный шрифт для текста на русском и английском языках - **Times New Roman** (**Кириллица**), для текста на грузинском языке следует использовать **AcadNusx.** Размер шрифта - **12**. К рукописи, напечатанной на компьютере, должен быть приложен CD со статьей.

2. Размер статьи должен быть не менее десяти и не более двадцати страниц машинописи, включая указатель литературы и резюме на английском, русском и грузинском языках.

3. В статье должны быть освещены актуальность данного материала, методы и результаты исследования и их обсуждение.

При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

5. Таблицы необходимо представлять в печатной форме. Фотокопии не принимаются. **Все цифровые, итоговые и процентные данные в таблицах должны соответствовать таковым в тексте статьи**. Таблицы и графики должны быть озаглавлены.

6. Фотографии должны быть контрастными, фотокопии с рентгенограмм - в позитивном изображении. Рисунки, чертежи и диаграммы следует озаглавить, пронумеровать и вставить в соответствующее место текста **в tiff формате**.

В подписях к микрофотографиям следует указывать степень увеличения через окуляр или объектив и метод окраски или импрегнации срезов.

7. Фамилии отечественных авторов приводятся в оригинальной транскрипции.

8. При оформлении и направлении статей в журнал МНГ просим авторов соблюдать правила, изложенные в «Единых требованиях к рукописям, представляемым в биомедицинские журналы», принятых Международным комитетом редакторов медицинских журналов - http://www.spinesurgery.ru/files/publish.pdf и http://www.nlm.nih.gov/bsd/uniform_requirements.html
В конце каждой оригинальной статьи приводится библиографический список. В список литературы включаются все материалы, на которые имеются ссылки в тексте. Список составляется в алфавитном порядке и нумеруется. Литературный источник приводится на языке оригинала. В списке литературы сначала приводятся работы, написанные знаками грузинского алфавита, затем кириллицей и латиницей. Ссылки на цитируемые работы в тексте статьи даются в квадратных скобках в виде номера, соответствующего номеру данной работы в списке литературы. Большинство цитированных источников должны быть за последние 5-7 лет.

9. Для получения права на публикацию статья должна иметь от руководителя работы или учреждения визу и сопроводительное отношение, написанные или напечатанные на бланке и заверенные подписью и печатью.

10. В конце статьи должны быть подписи всех авторов, полностью приведены их фамилии, имена и отчества, указаны служебный и домашний номера телефонов и адреса или иные координаты. Количество авторов (соавторов) не должно превышать пяти человек.

11. Редакция оставляет за собой право сокращать и исправлять статьи. Корректура авторам не высылается, вся работа и сверка проводится по авторскому оригиналу.

12. Недопустимо направление в редакцию работ, представленных к печати в иных издательствах или опубликованных в других изданиях.

При нарушении указанных правил статьи не рассматриваются.

REQUIREMENTS

Please note, materials submitted to the Editorial Office Staff are supposed to meet the following requirements:

1. Articles must be provided with a double copy, in English or Russian languages and typed or compu-ter-printed on a single side of standard typing paper, with the left margin of **3** centimeters width, and **1.5** spacing between the lines, typeface - **Times New Roman** (**Cyrillic**), print size - **12** (referring to Georgian and Russian materials). With computer-printed texts please enclose a CD carrying the same file titled with Latin symbols.

2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

8. Please follow guidance offered to authors by The International Committee of Medical Journal Editors guidance in its Uniform Requirements for Manuscripts Submitted to Biomedical Journals publication available online at: http://www.nlm.nih.gov/bsd/uniform_requirements.html
http://www.icmje.org/urm_full.pdf
In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

9. To obtain the rights of publication articles must be accompanied by a visa from the project instructor or the establishment, where the work has been performed, and a reference letter, both written or typed on a special signed form, certified by a stamp or a seal.

10. Articles must be signed by all of the authors at the end, and they must be provided with a list of full names, office and home phone numbers and addresses or other non-office locations where the authors could be reached. The number of the authors (co-authors) must not exceed the limit of 5 people.

11. Editorial Staff reserves the rights to cut down in size and correct the articles. Proof-sheets are not sent out to the authors. The entire editorial and collation work is performed according to the author's original text.

12. Sending in the works that have already been assigned to the press by other Editorial Staffs or have been printed by other publishers is not permissible.

Articles that Fail to Meet the Aforementioned Requirements are not Assigned to be Reviewed.

ავტორთა საყურადღებოდ!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე,დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx.** შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

3. სტატიაში საჭიროა გაშუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითონ საექსპერიმენტო ცხოველების სახეობა და რაოდენობა; გაუტკივარებისა და დაძინების მეთოდები (მწვავე ცდების პირობებში).

4. სტატიას თან უნდა ახლდეს რეზიუმე ინგლისურ, რუსულ და ქართულ ენებზე არანაკლებ ნახევარი გვერდის მოცულობისა (სათაურის, ავტორების, დაწესებულების მითითებით და უნდა შეიცავდეს შემდეგ განყოფილებებს: მიზანი, მასალა და მეთოდები, შედეგები და დასკვნები; ტექსტუალური ნაწილი არ უნდა იყოს 15 სტრიქონზე ნაკლები) და საკვანძო სიტყვების ჩამონათვალი (key words).

5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემაჯამებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფჩხილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

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ASSOCIATION BETWEEN GALECTIN-3 AND MICROVASCULAR COMPLICATIONS IN TYPE 2 DIABETES MELLITUS: A COMPARATIVE STUDY

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Abstract.

Background: Galectin-3 is a β-galactoside-binding lectin involved in inflammation and fibrosis and has been implicated in the pathogenesis of type 2 diabetes mellitus (T2DM) and its microvascular complications. This study aimed to evaluate the association between serum galectin-3 levels and microvascular complications in patients with T2DM and to determine whether this association persists after adjustment for relevant clinical factors.

Materials and Methods: This comparative cross-sectional study included 64 participants recruited from Thumbay University Hospital, Ajman, UAE. Participants were divided into two groups: 32 patients with T2DM and established microvascular complications and 32 healthy controls. Serum galectin-3 levels were measured using enzyme-linked immunosorbent assay (ELISA). Clinical and biochemical parameters, including body mass index (BMI), HbA1c, fasting blood glucose, lipid profile, and microalbuminuria, were assessed. Multivariable linear regression analysis was performed with microalbuminuria as the dependent variable, adjusting for BMI, HbA1c, and age.

Results: Serum galectin-3 levels were significantly higher in patients with T2DM and microvascular complications compared with healthy controls ($p < 0.001$). Galectin-3 showed a strong positive correlation with microalbuminuria ($\rho = 0.720$, $p < 0.001$) and a moderate positive correlation with HbA1c ($\rho = 0.599$, $p < 0.001$). However, in multivariable regression analysis, galectin-3 was not independently associated with microalbuminuria after adjustment for BMI, HbA1c, and age ($p = 0.197$), whereas HbA1c remained a significant independent predictor ($p < 0.001$).

Conclusion: Although serum galectin-3 levels are elevated in patients with T2DM and microvascular complications and demonstrate strong univariate associations with microalbuminuria, galectin-3 does not remain independently associated after adjustment for BMI and glycemic control. These findings suggest that galectin-3 may reflect underlying metabolic and inflammatory burden rather than acting as an independent predictor of microvascular complications in T2DM.

Key words. Type 2 diabetes mellitus, Galectin-3, microvascular complications, nephropathy, biomarker.

Introduction.

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia due to impaired insulin secretion, insulin resistance, or both. The disease results from a malfunction in the body's ability to effectively produce or utilize insulin. Insulin, a hormone secreted by pancreatic beta cells, plays a crucial role in regulating blood glucose levels by facilitating glucose uptake into cells for energy production [1,2]. Type 2 diabetes mellitus (T2DM) is the most prevalent form of diabetes and accounts for approximately 90% of all cases. It is a growing global health concern, with a rising incidence due to sedentary lifestyles, unhealthy dietary habits, and increasing obesity rates. According to the International Diabetes Federation (IDF), the global prevalence of diabetes is estimated at 10.5% in 2021 and is projected to increase significantly in the coming decades. T2DM is associated with severe complications affecting multiple organ systems, leading to significant morbidity and mortality. Chronic hyperglycemia in T2DM contributes to microvascular complications, including diabetic retinopathy, nephropathy, and neuropathy, primarily due to sustained damage to small blood vessels [3,4].

Recent studies have identified galectin-3, a β-galactoside-binding lectin, as a key mediator of inflammation and fibrosis. Galectin-3 has been implicated in the pathogenesis of various diseases, including diabetes mellitus and its complications [5]. Studies have demonstrated a significant association between elevated serum galectin-3 levels and the severity of microvascular complications in T2DM patients [4]. Cellular-level investigations suggest that increased galectin-3 expression may contribute to vascular dysfunction, chronic inflammation, and fibrosis, thereby exacerbating complications in diabetic patients [5,6].

For instance, studies have reported a twofold increase in galectin-3 levels in T2DM patients with nephropathy compared to those without, with a strong correlation between galectin-3 and albuminuria, suggesting its involvement in the progression of diabetic nephropathy [4,6,7]. Similarly, higher galectin-3 levels have been observed in patients with severe diabetic retinopathy, indicating its potential role in retinal vascular damage. Moreover, increased galectin-3 concentration has been linked to diabetic neuropathy, where elevated levels correlate with more pronounced muscle damage and peripheral nerve dysfunction [4].

Emerging evidence suggests that galectin-3 contributes to insulin resistance and pancreatic β- cell dysfunction by promoting inflammatory responses in insulin target organs [7]. Animal studies have demonstrated that galectin-3 induces inflammation in pancreatic islets, leading to β-cell failure, which may accelerate diabetes progression. These findings highlight the potential role of galectin-3 not only in the development of T2DM but also in its associated complications.

Given the growing evidence linking galectin-3 to microvascular complications in T2DM, further research is warranted to explore its potential as a biomarker for early detection and risk stratification. Understanding the molecular mechanisms underlying galectin-3's role in diabetes-related vascular damage may offer novel therapeutic insights for managing T2DM and preventing its complications.

Materials and Methods.

This correlation study was conducted at Thumbay University Hospital, Ajman, UAE. The study population consisted of patients with Type 2 diabetes mellitus (T2DM) who attended Thumbay University Hospital in Ajman, UAE. A total of 64 participants were recruited and divided into two groups: the study group (32 patients with type 2 diabetes mellitus (T2DM) and microvascular complications) and the control group (32 healthy individuals without diabetes or microvascular complications). Exclusion criteria included patients with chronic inflammatory conditions, pregnant females, and those with Type 1 diabetes mellitus (T1DM). Inclusion criteria: Patients with T2DM who met the World Health Organization (WHO) criteria were eligible [8]. The study was conducted over a period of 24 weeks, which included time for preparation, practical analysis, and data interpretation. All instruments used were validated for accuracy, precision, and linearity according to the laboratory's quality control protocols. All participants were in a state of fasting for 12 h before drawing blood.

Galectin-3.

Galectin-3 in vitro SimpleStep ELISA® (Enzyme-Linked Immunosorbent Assay) kit is designed for the quantitative measurement of Galectin-3 protein in human serum, plasma, cell culture supernatant, cell and tissue extract.

The kit had a detection range with a minimum detectable concentration of 1.5 ng/ml and a maximum detectable concentration of 100 ng/ml.

HbA1c.

HbA1c percentage level was measured using a method based on A turbidimetric immunoinhibition using for the measurement of glycated hemoglobin (HbA1c) % in human whole blood, is traceable to the International Federation of Clinical Chemistry reference method for the measurement of HbA1c, and its measuring range 3–18% HbA1c [9].

An HbA1c test result must be less than 5.7% to be considered normal or in the non-diabetic category. A person can be diagnosed with diabetes if their HbA1c is 6.5% or higher, but prediabetes is defined as anyone with a value between 5.7% and 6.4% [10].

Fasting blood glucose and the lipid profile were measured using Beckman Coulter DxC 700 AU Chemistry Analyzer.

Microalbuminuria.

The Urine Albumin-to-Creatinine Ratio (ACR) is the preferred method for detecting microalbuminuria in clinical and research settings due to its accuracy and convenience. It involves measuring albumin concentration relative to creatinine in a spot urine sample, typically an early morning specimen, to account for variations in urine concentration. ACR is commonly analyzed using immunoturbidimetry or immunonephelometry, both of which rely on antibody-based reactions to detect albumin levels. The results are expressed in milligrams of albumin per gram of creatinine (mg/g) or milligrams per millimole (mg/mmol). According to established guidelines, an ACR value of less than 30 mg/g (<3 mg/mmol) is considered normal, 30–300 mg/g (3–30 mg/mmol) indicates microalbuminuria, and greater than 300 mg/g (>30 mg/mmol) signifies macroalbuminuria [11].

Statistical analysis:

Descriptive statistics were performed by calculating the mean and standard deviation for the continuous variables. Categorical variables are presented as absolute numbers and percentages. Nominal categorical data between the groups were compared using the Chi-square goodness-to-fit test. The Student-t test was used to compare the mean of quantitative variables. Other tests used were analysis of variance (ANOVA) and multivariate analysis. The P value was taken as significant when less than 0.05 ($P < 0.05$).

The study included a total of 64 patients, divided into two groups: 32 patients in the Study Group, consisting of patients with T2DM and microvascular complications, and 32 patients in the Control Group, composed of healthy individuals. All participants were between the ages of 40 and 60. The two groups were age-matched ($p = 0.166$) and gender-matched ($p = 0.281$), ensuring comparability.

As presented in Table 1, the Study Group exhibited significantly higher levels of BMI, microalbuminuria, total cholesterol, triglycerides, LDL, HbA1c, and fasting blood glucose, while HDL levels were notably lower compared to the Control Group. Additionally, there was a statistically significant difference in Galectin-3 levels between the two groups ($p < 0.001$), with the Study Group displaying significantly higher Galectin-3 levels than the Control Group.

The study group exhibited significantly higher BMI compared with healthy controls, which may contribute to elevated galectin-3 levels due to obesity-related inflammatory activity.

The biochemical parameters of the two groups were compared. As presented in Table 2, the Pearson Chi-Square test for age ($p = 0.498$) shows no statistical significance, indicating that age is not significantly associated with microvascular complications. However, a strong correlation was observed between microvascular complications and BMI categories. The study group predominantly consists of overweight or obese individuals, whereas all individuals in the control group have a normal BMI. Additionally, obesity and overweight are more prevalent among patients with macroalbuminuria.

The distribution analysis highlights a significant relationship between BMI and microvascular complications, with higher BMI being associated with an increased risk of both macroalbuminuria and microalbuminuria. Furthermore, the Chi-Square test for HbA1C and fasting blood glucose (both $p < 0.001$) demonstrates a strong correlation between these parameters and microvascular complications. The data indicate that most patients with microalbuminuria have diabetes, whereas all patients with normoalbuminuria do not. Overall, there is a statistically significant difference between the study and control groups.

Table 3 shows the values of Lipid Profiles across the groups. Compared to the control group, the study group exhibits significantly higher amounts of LDL, triglycerides, and total cholesterol as well as significantly lower levels of HDL. These differences indicate that the study group has a less ideal lipid profile, which might be linked to increased complications.

Microalbuminuria demonstrated significant positive correlations with BMI, fasting blood glucose (FBS), total

cholesterol, triglycerides, low-density lipoprotein (LDL), and HbA1c, as shown in Table 4. These findings indicate an association between increased microalbuminuria levels and specific biochemical markers.

In contrast, microalbuminuria exhibited a moderate but non-significant negative correlation with high-density lipoprotein (HDL), suggesting no meaningful relationship within this study population. Except for HDL, all correlations were statistically significant at the 0.05 level, indicating that these associations are unlikely to have occurred by chance. These results suggest that elevated microalbuminuria, a key indicator of kidney injury, is associated with poor glycemic control (as reflected by HbA1c and FBS), high BMI, and dyslipidemia (elevated total cholesterol, triglycerides, and LDL).

The association between microalbuminuria and HbA1c levels (Figure 1) is moderately positive. Microalbuminuria, a sign of possible kidney injury, is correlated with higher HbA1c readings, which show poor blood sugar regulation. This association is significant because it implies that preserving lower HbA1c levels may help people, particularly those with diabetes, avoid or lessen kidney damage.

The data indicates a significant Positive correlation between Galectin-3 and both HbA1c and Microalbuminuria (Table 5). Specifically, Galectin-3 shows a moderate positive correlation with HbA1c ($\rho = 0.599$, $p < 0.001$) (Figure 3) and a strong positive correlation with microalbuminuria ($\rho = 0.720$, $p < 0.001$) (Figure 2). These correlations suggest that Galectin-3 could potentially be a marker for monitoring glycemic control (as indicated by HbA1c) and renal function (as indicated by microalbuminuria) in the studied population.

A multiple linear regression analysis was performed to evaluate whether galectin-3 was independently associated with microalbuminuria after adjustment for potential confounders, including body mass index (BMI), HbA1c, and age. In the adjusted model, HbA1c remained a significant independent predictor of microalbuminuria ($B = 26.06$, $p < 0.001$). However, galectin-3 did not remain significantly associated with microalbuminuria after adjustment ($B = 1.35$, $p = 0.197$). BMI and age were also not significant predictors in the multivariable model (Table 6).

The results further revealed that Gal-3 levels were significantly higher in T2DM patients compared to non-diabetic controls, aligning with previous studies that have reported an association between elevated Gal-3 and diabetes and its complications [12].

This suggests that Gal-3 may play a role in the development and progression of microvascular complications in T2DM. Elevated Gal-3 levels have been associated with more severe diabetes-related complications [4], supporting the hypothesis that Gal-3 not only serves as a biomarker for diabetic complications but may also contribute to their pathophysiology. The study group also exhibited a significantly higher BMI compared with healthy controls, which may contribute to elevated galectin-3 levels due to obesity-related inflammatory activity.

As a β-galactoside-binding lectin, Gal-3 is involved in several cellular processes, including apoptosis, fibrosis, and inflammation [3]. Its elevated levels have been implicated in conditions characterized by chronic inflammation and fibrosis, such as diabetes. Previous studies have demonstrated that Gal-3 promotes inflammatory responses and fibrosis in target organs, including the kidneys, eyes, and nerves, contributing to the pathogenesis of diabetic complications [5]. For instance, increased Gal-3 levels have been linked to diabetic kidney disease, a major microvascular complication of diabetes, through its role in inflammation and renal fibrosis. Additionally, Gal-3 has been associated with diabetic retinopathy and neuropathy, with higher levels observed in patients with severe manifestations of these conditions.

A strong positive correlation was observed between galectin-3 and microalbuminuria, a recognized marker of early diabetic nephropathy and endothelial dysfunction. This finding

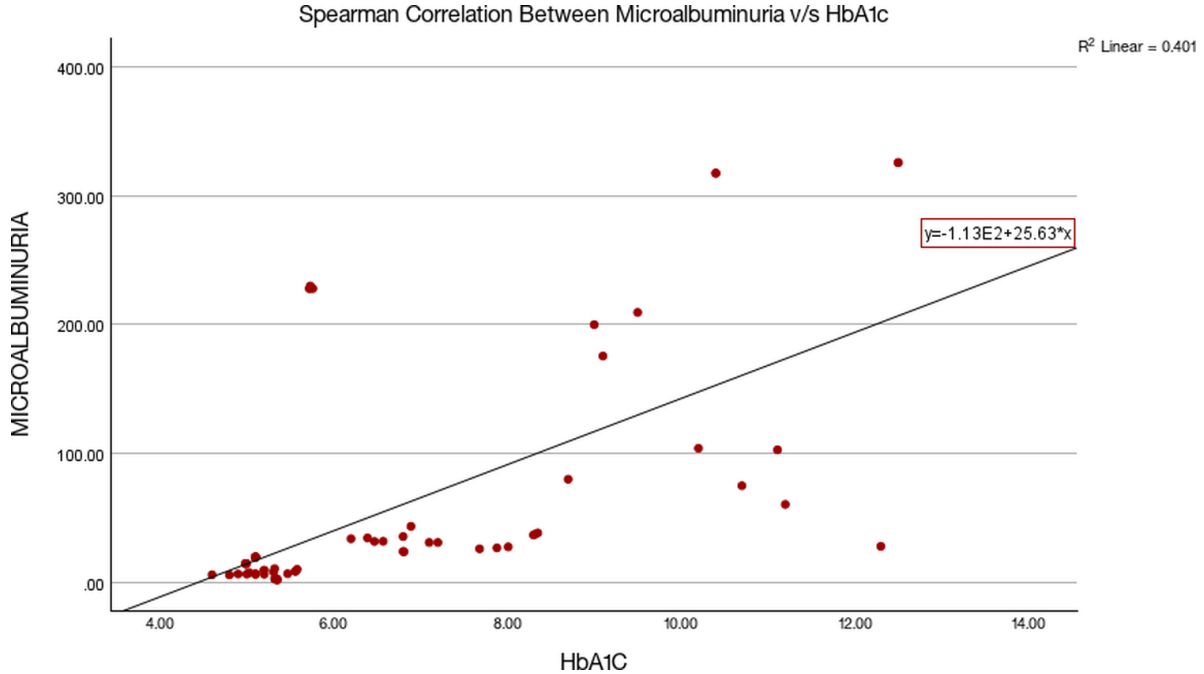

Figure 1. *Scatter Plot shows correlation between Microalbuminuria and HbA1c (n= 64) ($R^2 = 0.401$).*

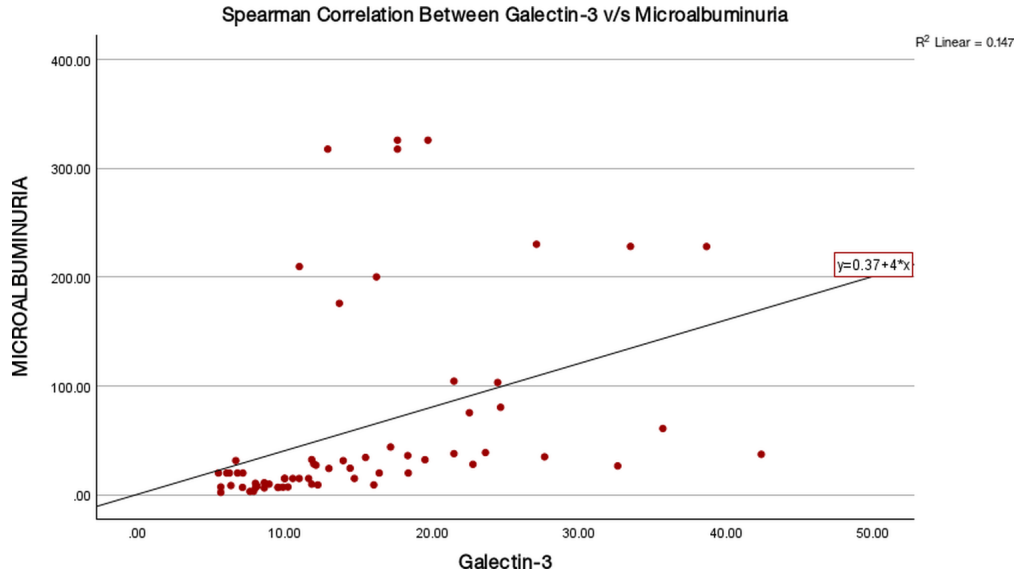

Figure 2. *Scatter Plot shows correlation between Galectin-3 and Microalbuminuria (n= 64) (R2 = 0.147).*

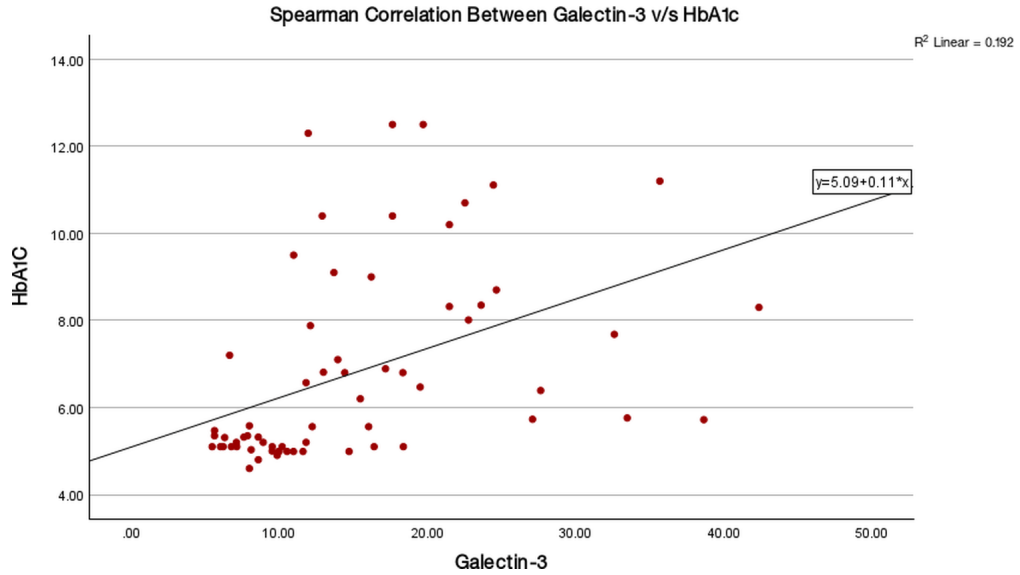

Figure 3. *Scatter Plot shows correlation between Galectin-3 and HbA1c (n= 64) (R2 = 0.192).*

Table 1. *Main characteristics of the study population.*

Parameter	**Study Group**	**Control Group**	***p*-Value**
Age (Years)	51.66 ± 5.33	49.34 ± 6.24	**0.166**
Male	24 (75%)	24 (75%)	**0.291**
Female	8 (25%)	8 (25 %)	**0.291**
BMI	28.18 ± 2.32	21.80 ± 1.82	<0.001
Microalbumin	103.93 ± 96.94	11.33 ± 5.73	<0.001
Total Cholesterol	207.38 ± 50.41	173.13 ± 25.63	<0.001
Triglyceride	189.94 ± 94.22	136.34 ± 83.22	0.019
HDL	47.09 ± 11.90	56.19 ± 12.78	0.005
LDL	123.94 ± 37.46	92.06 ± 29.23	<0.001
HbA1C	8.45 ± 2.07	5.14 ± 0.21	<0.001
FBS	190.47 ± 61.17	86.50 ± 12.96	<0.001
Galectin - 3	20.67 ± 8.65	9.48 ± 3.26	<0.001

BMI: Body Mass Index, HDL: High-Density Lipid, LDL: Low-Density Lipid, HbA1c: Glycated Hemoglobin, FBS: Fasting Blood Glucose. All values are the mean ± SD, p- Values <0.05 indicate statistical significance.

Table 2. *Relationship between Age, BMI, FBS, and HbA1c to Microalbuminuria Categories.*

Test	Parameter	Microalbuminuria Categories			*p*-value
Chi- square Test	**AGE**	Normoalbuminuria	Microalbuminuria	Macroalbuminuria	
	<45	11	4	1	
	46-54	12	13	4	
	>55	9	7	3	**0.498**
	BMI				
	Normal Weight	32	1	0	
	Overweight	0	16	6	
	Obese	0	7	2	<0.001
	FBS				
	Non-Diabetic	32	0	0	
	Diabetic	0	24	8	<0.001
	HbA1c				
	Non-Diabetic	16	3	13	
	Pre-Diabetic	12	2	10	
	Diabetic	4	1	3	<0.001

BMI: Body Mass Index, HbA1c: Glycated Hemoglobin, FBS: Fasting Blood Glucose. p- Values <0.05 indicates statistical significance.

Table 3. *Lipid Profile between the study group and control group.*

Test	Parameter	Study Group	Control Group	*p*-value
Independent Samples t- Test	Total Cholesterol	207.38 ± 50.41	173.13 ± 25.63	0.001
	Triglyceride	189.94 ± 94.22	136.34 ± 83.22	0.019
	HDL	47.09 ± 11.90	56.19 ± 12.78	0.005
	LDL	123.94 ± 37.469	92.06 ± 29.23	<0.001

HDL: High-Density Lipid, LDL: Low-Density Lipid. All values are the mean ± SD, and p- Values <0.05 indicate statistical significance.

Table 4. *Correlation between Biochemical Parameters and Microalbuminuria.*

Test	Parameter	ρ	*p*-value
Spearman Correlation	HbA1c	0.776	<0.001
	FBS	0.806	<0.001
	BMI	0.674	<0.001
	Total Cholesterol	0.425	<0.001
	Triglyceride	0.420	<0.001
	HDL	-0.128	0.313
	LDL	0.360	0.003
	Galectin-3	0.720	<0.001

BMI: Body Mass Index, HDL: High-Density Lipid, LDL: Low-Density Lipid, HbA1c: Glycated Hemoglobin, FBS: Fasting Blood Glucose. ρ Spearman Correlation Coefficient, p- Values <0.05 indicates statistical significance.

Table 5. *Correlation of Galectin-3 with HbA1c and Microalbuminuria.*

Test	Parameter	ρ	*p*-value
Spearman Correlation	HbA1c	0.599	<0.001
	Microalbuminuria	0.720	<0.001

HbA1c Glycated Hemoglobin.ρ Spearman Correlation Coefficient, p- Values <0.05 indicates statistical significance.

Table 6. *Multiple Linear Regression Analysis with Microalbuminuria as the Dependent Variable.*

Predictor	B (Unstandardized Coefficient)	Standard Error	t-value	p-value
Constant	−208.61	97.12	−2.15	0.036
Galectin-3	1.35	1.03	1.31	0.197
BMI	0.15	3.28	0.05	0.964
HbA1c	26.06	5.43	4.80	<0.001
Age	1.20	1.55	0.77	0.443

The multivariable regression model explained 42.4% of the variance in microalbuminuria ($R^2 = 0.424$; adjusted $R^2 = 0.385$) and was statistically significant ($F = 10.84$, $p < 0.001$); unstandardized regression coefficients (B) are reported, with $p < 0.05$ considered statistically significant.

suggests that galectin-3 may be closely associated with renal microvascular involvement in T2DM. Similar associations have been reported in previous studies linking elevated galectin-3 levels with diabetic nephropathy and progression of renal injury.

A significant moderate positive correlation was observed between Gal-3 and HbA1c levels ($\rho = 0.599$, $p < 0.001$). This observation may be attributed to the participants' effective diabetes management, which could have stabilized HbA1c levels independently of Gal-3 levels. Additionally, unaccounted lifestyle factors, such as alcohol and tobacco consumption, might have influenced Gal-3 levels beyond glycemic control.

Lipid profile analysis indicated that LDL, triglycerides, and total cholesterol levels were significantly elevated in the diabetic group compared to the control group, while HDL levels were markedly lower (47.09 ± 11.90). These lipid abnormalities were more pronounced in patients with elevated Gal-3 levels and are well-established risk factors for cardiovascular disease. This finding suggests a potential link between lipid dysregulation, elevated Gal-3, and an increased risk of vascular complications in T2DM. Despite the significance of these findings, several limitations warrant further investigation. Although the study establishes a correlation between Gal-3 levels and microvascular complications in T2DM, the precise mechanisms underlying this association remain unclear. Future research should focus on elucidating the molecular pathways through which Gal-3 contributes to diabetes-related inflammation and fibrosis.

Moreover, as this study was correlational, causal relationships could not be determined. Longitudinal studies are necessary to assess whether elevated Gal-3 levels predict the onset and progression of microvascular complications in T2DM. Additionally, given the association between Gal-3 and diabetic complications, future studies should explore its potential as a therapeutic target. Investigating modulators or inhibitors of Gal-3 could provide novel therapeutic strategies for managing diabetes-related complications. The study population was limited to a specific ethnic and geographic cohort, which may affect the generalizability of the findings. Future research should include diverse populations to validate these results. Furthermore, exploring interactions between Gal-3 and other inflammatory and fibrotic biomarkers could offer a more comprehensive understanding of its role in diabetic complications. Identifying potential biomarker combinations may enhance risk assessment and improve therapeutic strategies for T2DM patients.

Conclusion.

This study examined the association between serum galectin-3 levels and microvascular complications in patients with type 2 diabetes mellitus by comparing affected individuals with healthy controls. The findings demonstrate that patients with T2DM and microvascular complications exhibit significantly elevated galectin-3 levels compared with healthy individuals.

A strong positive association was observed between galectin-3 and microalbuminuria, suggesting that galectin-3 may reflect the presence of microvascular involvement in T2DM. Although galectin-3 levels were significantly elevated in patients with T2DM and microvascular complications and showed strong univariate associations with microalbuminuria, galectin-3 was not independently associated with microalbuminuria after adjustment for BMI, HbA1c, and age. These findings suggest that galectin-3 may reflect metabolic and inflammatory burden rather than acting as an independent determinant of microvascular complications in T2DM.analysis. Accordingly, the findings should be interpreted as associative rather than causal, and galectin-3 should not be considered an independent predictor of microvascular complications based on the present analysis.

These findings underscore the potential utility of galectin-3 as a biomarker reflecting inflammatory and fibrotic activity associated with microvascular involvement in T2DM. The consistent association between elevated Galectin-3 levels and the prevalence of microvascular complications suggests a possible mechanistic role of this protein in the pathogenesis of these conditions. Further validation in longitudinal and multivariable studies is required before galectin-3 can be considered for routine clinical application.

Despite these limitations, the consistent elevation of galectin-3 in patients with diabetic microvascular complications highlights its potential utility as a biomarker reflecting underlying inflammatory and fibrotic activity in T2DM.

Future studies incorporating T2DM patients without complications, larger sample sizes, longitudinal follow-up, and multivariable models using microvascular outcomes as dependent variables are required to clarify the independent and predictive role of galectin-3 in diabetic microvascular disease.

In conclusion, elevated Galectin-3 levels have been identified as a potential biomarker for early detection of microvascular complications in T2DM patients. The significant association between Galectin-3 and the prevalence of these complications provides a promising avenue for future research and clinical application. Implementing routine assessments of Galectin-3 could enhance current management strategies, alleviate the burden of diabetes-related complications, and ultimately improve the health and satisfaction of individuals living with T2DM.

Conflicts of Interest.

The authors declare no conflicts of interest.

Ethical Guidelines.

The study involving human participants included a consent form from each participant, detailing their rights and the study's purpose. The research was approved by the Institutional Review Board (IRB) at Gulf Medical University, with reference number IRB-COHS-STD-65- DEC-2023.

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