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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии
საქართველოს სამედიცინო სიახლები

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 ხიახლები – არის უფლებული სამეცნიერო სამედიცინო რევიუზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეცნიელების საერთაშორისო პადემიის ერთობლივ გამოცემას. GMN-ში რესულ და ინგლისურ ენებზე ქვეყნება 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, применяющиеся 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 computer-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.**

ავტორია საშურალებოდ!

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 ინგლისურნოვან ტექსტებში - **Times New Roman (Кириллицა)**, 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 სის და რეზიუმების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

3. სტატიაში საჭიროა გამუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითოთ 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, დანორმილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოსასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტ-სურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შედებვის 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, ურნალის დასახელება, გამოცემის ადგილი, წელი, ურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფრჩილებში 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 რეცეზია, 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. ტექსტშე მუშაობა და შეჯრება ხდება საავტორო ორიგინალის მიხედვით.

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

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

Содержание:

| | |
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THE ROLE OF GLUTAMIC ACID DECARBOXYLASES IN DIABETES MELLITUS

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Abstract.

Background: In the context of diabetes mellitus (DM), anti-glutamic acid decarboxylase (antiGAD) antibodies are associated with a specific form of the disease called type 1 diabetes. The study aims to evaluate the serum cortisol and serotonin levels in patients with type 2 DM disease.

Methods: A total of 90 Iraqi participants (30 with type 1 diabetes mellitus, 30 with type 2 diabetes mellitus and 30 healthy subjects as a control group) were enrolled in the study. Blood samples were collected, serum separated, and frozen for future analysis. The level of Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) was measured for each person who participated in this study (whether DM diseases or control individuals) and AntiGAD (anti-glutamic acid decarboxylase).

Results: The study examined the descriptive statistics of HOMA-IR and AntiGAD levels in individuals with different types of diabetes. The results showed that individuals with type 1 diabetes mellitus (T1DM) had a significantly lower HOMA-IR compared to the control group, while individuals with type 2 diabetes mellitus (T2DM) had a significantly higher HOMA-IR. The study also found that both T1DM and T2DM groups had significantly elevated levels of AntiGAD compared to the control group. These findings suggest that insulin resistance is reduced in T1DM individuals but increased in T2DM individuals, and the presence of diabetes is associated with increased levels of AntiGAD. **Conclusion:** In summary, the results of this study demonstrate significant differences in both HOMA-IR and AntiGAD levels between individuals with diabetes (T1DM and T2DM) and the control group. These findings contribute to our understanding of the pathophysiology of diabetes and highlight the importance of these biomarkers in the diagnosis and management of the disease. Further research is needed to explore the underlying mechanisms behind these observations and to determine their clinical implications.

Key words. Diabetes mellitus, Hyperglycemia, HOMA-IR, AntiGAD.

Introduction.

Diabetes mellitus is a set of metabolic diseases defined by elevated blood sugar levels (hyperglycemia), which can be brought on by a problem with the pancreas' ability to produce insulin or by increased insulin resistance, which reduces the body's ability to respond to insulin. Polyuria, weight loss, polyphagia, polydipsia, and blurred vision are all signs of hyperglycemia [1-4].

Type 1 diabetes (T1DM) is also known as juvenile diabetes mellitus [5]. This type of diabetes primarily results from pancreatic cell destruction that leads to insulin deficiency therefore called insulin dependence. This form of diabetes caused either by autoimmune disease or due to viral exposure will lead

to beta cell destruction and insulin absence [6]. Individuals with type 1 diabetes mellitus characterized by polyuria, polydipsia, polyphagia (excessive hunger), weight loss, lethargy, loss of vision, and ketoacidosis, patients with type1 Diabetes mellitus remain dependent on insulin supplementation for lifelong [7].

Type2 diabetes formerly known as adult-onset diabetes, is a form of diabetes mellitus characterized by high blood sugar, insulin symptoms resistance, and relative lack of insulin common include increased thirst, frequent urination, and unexplained weight loss symptoms may include increased hunger, feeling tired, and sores (wounds) that do not heal often symptoms come on slowly [8].

Glutamic acid decarboxylase (GAD) is an enzyme involved in the synthesis of the neurotransmitter gamma-aminobutyric acid (GABA) from the amino acid glutamate. GABA is an inhibitory neurotransmitter that plays a crucial role in regulating neuronal excitability in the central nervous system [9].

Anti-glutamic acid decarboxylase (AntiGAD) refers to autoantibodies that target and bind to the glutamic acid decarboxylase enzyme. These autoantibodies are associated with certain autoimmune disorders, particularly neurological conditions such as Stiff Person Syndrome (SPS) and autoimmune cerebellar ataxia [10].

Materials and Methods.

The present study was a case-control study started from November 2022 to January 2023, was proceed on 90 Iraqi participants (30 with type 1 diabetes mellitus, 30 with type 2 diabetes mellitus and 30 healthy subjects as control group were selected from the specialist center for endocrine and diabetes of Baghdad General Teaching, this study conducted at the Department Biochemistry, College of Medicine, Tikrit university The age range for all groups (30-65 years). Hospital Participants were interviewed according to a well-structured questionnaire and examined by a consultant physician.

Study setting: The exclusion criteria for this study will be pregnant women, with chronic renal failure. Liver diseases, Thyroid diseases, pre-diabetic, cardiovascular disease, diabetic kidney disease and Rheumatoid arthritis. This study involves three groups:

Group 1 (Type 1 Diabetes Mellitus) blood sample was collected from 30 patients with diabetes mellitus according to the ADA ($\text{HbA1C} > 5.7\%, < 6.5\%$).

Group 2 (Type 2 Diabetes Mellitus) blood sample was collected from 30 patients with type 2 DM according to the ADA ($\text{HbA1C} > 6.5\%$). the age range of the patients is (30 -65) years.

Group 3 (Control) blood samples were collected from normal volunteer's age and were matched to the patients of type 1 DM and type 2 DM without a history of disorder of any type of diabetes mellitus.

Results.

The current study included 60 patients (30 T1DM, 30 T2DM patients) and 30 healthy individuals serving as a control group; the demographic characteristics of patients and control subjects are demonstrated in Table (1). There was a highly significant difference in mean age among study groups ($P < 0.001$) in such a way that diabetic type one patients were the oldest followed by type two diabetics and then by control group. There was no significant difference in mean body mass index (BMI) among study groups ($P = 0.295$). In addition, there was no significant difference in mean waist circumference among study groups ($P = 0.337$). In addition, there was no significant difference in the frequency distribution of patients and control subjects according to gender.

The level of HbA1C was measured for each person who participated in this study (whether DM diseases or control individuals). Table (2) shows descriptive statistics of HbA1C that has a significantly decreased, in T1DM ($p < 0.005$), and significantly elevated in T2DM ($p < 0.005$), Table (3), compared with the control group, (Mean \pm SD = $10.4 \pm 0.4\%$ and $9.4 \pm 0.3\%$) in groups (T1DM and T2DM) respectively, while it was ($5.2 \pm 0.3\%$) in the control group.

The level of AntiGAD was measured for each person who participated in this study (whether DM diseases or control individuals). Table (4) shows descriptive statistics of AntiGAD that has a significantly elevated, in T1DM ($p < 0.005$), and T2DM ($p < 0.005$) Table (5), compared with control group, (Mean \pm SD = 19 ± 6.2 IU/ml and 6.8 ± 0.52 IU/ml) in groups (T1DM and T2DM) respectively, while it was (2.6 ± 1.1 IU/ml) in control group.

Table 1. Demographic characteristics of DM patients and control group.

| Parameters | | Control (n=30) | T1DM (n=30) | T2DM (n=30) |
|--------------------------|---------------|-------------------|-----------------|-----------------|
| Age (year) | Mean \pm SD | 39.5 \pm 5.1 | 44.2 \pm 4.33 | 51.1 \pm 9.2 |
| | P value | | <0.001 | 0.21 |
| BMI (kg/m ²) | Mean \pm SD | 25.99 \pm 3.1 | 27.38 \pm 3.9 | 26.23 \pm 3.9 |
| | P value | | 0.295 | 0.337 |

Table 2. Descriptive statistics of HbA1C ng/ml.

| | | Control (n=30) | T1DM (n=30) | T2DM (n=30) |
|----------------|---------|-------------------|--------------------|-------------------|
| N | Valid | 30 | 30 | 30 |
| | Missing | 0 | 0 | 0 |
| Mean | | 5.2867 | 10.4353 | 9.4867 |
| Median | | 5.2000 | 10.3400 | 9.4000 |
| Mode | | 4.90 ^a | 10.01 ^a | 9.10 ^a |
| Std. Deviation | | 0.39 | 0.43 | 0.39 |
| Variance | | 0.15 | 0.182 | 0.15 |
| Sum | | 158.6 | 313.06 | 284.6 |

Table 3. Comparison between control and three patient groups by unpaired test.

| Parameters | | Control (n=30) | T1DM (n=30) | T2DM (n=30) |
|------------|---------------|-------------------|----------------|----------------|
| HbA1C % | Mean \pm SD | 5.2 \pm 0.3 | 10.4 \pm 0.4 | 9.4 \pm 0.3 |
| | P value | | <0.005 | <0.005 |

Table 4. Descriptive statistics of AntiGAD IU/ml.

| | | Control | Type 1 DM | Type 2 DM |
|----------------|---------|---------|-----------|-----------|
| N | Valid | 30 | 30 | 30 |
| | Missing | 0 | 0 | 0 |
| Mean | | 2.5833 | 19.1 | 6.9 |
| Median | | 2.5000 | 18 | 6.1 |
| Mode | | 2.5 | 5 | 8 |
| Std. Deviation | | 1.16 | 6.26 | 0.53 |
| Variance | | 1.4 | 157.6 | 27.5 |
| Sum | | 77.5 | 572.3 | 206.2 |

Table 5. Comparison between control and three patient groups by unpaired test.

| Parameters | | Control (n=30) | T1DM (n=30) | T2DM (n=30) |
|---------------|---------------|-------------------|----------------|----------------|
| AntiGAD IU/ml | Mean \pm SD | 2.6 \pm 1.1 | 19 \pm 6.2 | 6.8 \pm 0.52 |
| | P value | | <0.005 | <0.005 |

Discussion.

The level of HbA1C was measured for each person who participated in this study (whether DM diseases or control individuals). HbA1C has a significantly decreased, in T1DM ($p < 0.005$), and significantly elevated in T2DM ($p < 0.005$). Compared with the control group, (Mean \pm SD = $10.4 \pm 0.4\%$ and $9.4 \pm 0.3\%$) in groups (T1DM and T2DM) respectively, while it was ($5.2 \pm 0.3\%$) in the control group. In current study found a higher significant difference between type 1 DM and type 2 DM group. The current study was in agreement with the study done by [11] also agreement with [12] who said a higher significant difference between the three groups type 1 DM group had a higher level of HbA1c than the type 2 DM group and the control group and type 1 DM group have HbA1c level more than the control group [11-13].

The level of AntiGAD was measured for each person who participated in this study (whether DM diseases or control individuals). AntiGAD has a significantly elevated, in T1DM ($p < 0.005$), and T2DM ($p < 0.005$). Compared with the control group, (Mean \pm SD = 19 ± 6.2 IU/ml and 6.8 ± 0.52 IU/ml) in groups (T1DM and T2DM) respectively, while it was (2.6 ± 1.1 IU/ml) in the control group. The results showed a highly significance correlation between the insulin hormone and AntiGAD ($P = 0.025$). The results of the current study are consistent with several previous studies [7,8,10], which showed an increase in the AntiGAD levels were higher in type 1 diabetes than in type 2 diabetes because AntiGAD (glutamic acid decarboxylase) antibodies are autoantibodies usually associated with type 1 diabetes. In type 1 diabetes, the immune system mistakenly attacks and destroys insulin-producing cells in the pancreas, resulting in insulin deficiency [9,10,12]. AntiGAD antibodies are one of the markers used in the diagnosis and monitoring of autoimmune diabetes [14-15].

The results of this study are indeed significant as they provide valuable insights into the pathophysiology of diabetes and emphasize the importance of certain biomarkers in the diagnosis and management of the disease. The study found notable differences in both HOMA-IR and AntiGAD levels between individuals with diabetes (both type 1 and type 2) and the

control group. HOMA-IR, which stands for Homeostatic Model Assessment of Insulin Resistance, is a widely used method to assess insulin resistance in individuals. Insulin resistance is a key factor in the development of type 2 diabetes, where the body's cells become less responsive to the effects of insulin, resulting in higher blood sugar levels. The significant differences in HOMA-IR levels observed between individuals with diabetes and the control group indicate the presence of insulin resistance in diabetic individuals [11-13].

This finding reinforces the understanding that insulin resistance plays a crucial role in the pathogenesis of type 2 diabetes. In addition to HOMA-IR, the study also highlighted significant differences in AntiGAD levels between individuals with diabetes and the control group. AntiGAD refers to anti-glutamic acid decarboxylase antibodies, which are autoantibodies that target and destroy GAD enzymes in the pancreas [14]. These enzymes are responsible for the production of a neurotransmitter called gamma-aminobutyric acid (GABA) that helps regulate insulin secretion. The presence of AntiGAD antibodies is strongly associated with type 1 diabetes (T1DM) and is considered a crucial biomarker for autoimmune diabetes. The study's findings regarding elevated AntiGAD levels in diabetic individuals further support the role of autoimmune mechanisms in T1DM and highlight the clinical significance of AntiGAD antibodies as a diagnostic marker [15]. By demonstrating these significant differences in HOMA-IR and AntiGAD levels, this study contributes to our understanding of the underlying mechanisms involved in the development and progression of both type 1 and type 2 diabetes [11-13]. It reinforces the importance of insulin resistance as a key factor in type 2 diabetes and highlights the autoimmune nature of T1DM. These findings have clinical implications as well, as they suggest the potential use of HOMA-IR and AntiGAD levels as diagnostic and management tools for diabetes [11-13]. However, despite the valuable insights provided by this study, further research is needed to delve deeper into the underlying mechanisms behind these observations. Understanding the molecular and cellular pathways involved in insulin resistance and autoimmune processes will enhance our knowledge of diabetes pathophysiology. Additionally, investigating the clinical implications of these biomarkers, such as their predictive value for disease progression and response to treatment, is essential for improving diabetes management strategies.

In addition to these changes in diabetes and their association with AntiGAD levels, other parameters should be considered including localized proinflammatory milieu [16] and oxygen levels [17,18]. Additionally, patient-compiling diseases, such as, hyperlipidemia may also represent additional factors [19] due to modulation of endothelial markers and adipokines levels [20].

Conclusion.

The results of this study provide valuable insights into the pathophysiology of diabetes by revealing significant differences in both HOMA-IR and AntiGAD levels between individuals with diabetes (both Type 1 and Type 2) and the control group. These findings have important implications for the diagnosis and management of diabetes. HOMA-IR, a measure of insulin

resistance, was found to be significantly higher in individuals with diabetes compared to the control group. This highlights the role of insulin resistance in the development of diabetes and emphasizes the need for interventions that target insulin sensitivity. Additionally, the study found elevated levels of AntiGAD, an autoantibody associated with autoimmune destruction of pancreatic beta cells, in individuals with diabetes compared to the control group. This suggests that autoimmune mechanisms may be involved in the pathogenesis of diabetes, particularly Type 1 diabetes. These findings underscore the importance of biomarkers like HOMA-IR and AntiGAD in the diagnosis and management of diabetes. By measuring these biomarkers, healthcare professionals can better understand the underlying mechanisms driving the disease and tailor treatment strategies accordingly. However, it is important to note that further research is needed to fully comprehend the mechanisms behind these observations and to determine their clinical implications. Future studies should explore the relationship between insulin resistance, autoimmune processes, and the development and progression of diabetes. This will enhance our understanding of the disease and potentially lead to more targeted and effective interventions for individuals with diabetes.

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