

GEORGIAN MEDICAL NEWS

ISSN 1512-0112

NO 1 (370) Январь 2026

ТБИЛИСИ - NEW YORK



ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии
საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

Monthly Georgia-US joint scientific journal published both in electronic and paper formats of the Agency of Medical Information of the Georgian Association of Business Press.
Published since 1994. Distributed in NIS, EU and USA.

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 ВИНТИ 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.

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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. Корректур авторам не высылаются, 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.**

ავტორთა საქურაღებოლ!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დაიცვათ 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. რენტგენოგრაფიების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შედეგების 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 ფხიხლებში 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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IMPACT OF GINGER SUPPLEMENTATION ON BLOOD PRESSURE AND GLUCOSE LEVELS IN PATIENTS WITH TYPE 2 DIABETES MELLITUS AND CARDIOVASCULAR DISEASE

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Abstract.

Background: Chronic inflammation relates type 2 diabetes mellitus (T2DM) to cardiovascular disease (CVD), which causes insulin resistance and endothelial dysfunction. Ginger (*Zingiber officinale*) as an anti-inflammatory, antioxidant compound can change the cardiometabolic parameters. This paper will investigate how ginger supplementation influences the blood pressure of patients with CVD and T2DM, glucose levels, and inflammatory markers.

Methods: A prospective interventional before–after study with a healthy reference control group was conducted involving 80 participants aged 27–44 years with mild dyslipidemia, hyperglycemia and hypertension, placed in a control group (n=40) or an intervention group (n=40) where ginger infusion was administered two times a day in 4 months. Lipid profiles (total cholesterol, LDL-C, HDL-C, VLDL-C, triglycerides), glucose of the fasting sample, and oral markers of inflammation (hs-CRP, IL-6, PCT) were defined at second and ninth week, respectively using Roche Cobas analyzers. They were done with paired t-tests, and significance at $p < 0.05$.

Results: The intervention group showed significant reductions in hs-CRP (20.5%, 4.69 ± 0.27 to 3.73 ± 0.14 mg/L, $p < 0.01$) and IL-6 (25.2%, 7.35 ± 0.33 to 5.50 ± 0.20 pg/mL, $p < 0.01$), PCT (0.05 ± 0.00 vs. 0.07 ± 0.00 ng/ml). Lipid profiles improved, with LDL-C reduced by 6.7% (172.63 ± 2.31 to 161.05 ± 2.50 mg/dL, $p < 0.01$), VLDL-C by 17.9% (44.69 ± 1.19 to 36.71 ± 1.10 mg/dL, $p < 0.01$), total cholesterol by 18.6% (255.00 ± 4.65 to 207.48 ± 6.12 mg/dL, $p < 0.01$), and triglycerides by 13.6% (222.25 ± 4.49 to 191.90 ± 4.49 mg/dL, $p < 0.01$). Fasting glucose decreased significantly ($p < 0.01$). PCT levels remained unchanged.

Conclusion: Ginger supplementation is a powerful way to reduce inflammatory markers, lipid levels, and glycemic control in individuals with T2DM and CVD, which means that it can be used as an adjunctive treatment to control cardiometabolic risk and should be the subject of more large-scale studies.

Key words. Cardiovascular disease, ginger supplementation, inflammation, procalcitonin, lipid profile, blood pressure.

Introduction.

Types 2 diabetes mellitus (T2DM) and cardiovascular disease (CVD) are intertwined illnesses, both caused by chronic low-grade inflammation, which enhances insulin resistance, endothelial dysfunction, and atherosclerosis and predisposes people to cardiovascular risks to a significant degree [1]. T2DM is known to impact more than 463 million people across the world, and up to 70 % of them develop CVD, which increases

the risk of cardiovascular events 2-4-fold [2]. High levels of pro-inflammatory cytokines, such as high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6) and procalcitonin (PCT) are key to this comorbidity [3]. The acute-phase protein Hs-CRP is a predictor of cardiovascular disease and increased in T2DM as hepatically produced by IL-6. The atherogenesis and insulin resistance are both promoted by IL-6 [4]. PCT, which is a traditional indicator of bacterial infection, is being increasingly identified in chronic inflammatory conditions, such as T2DM and CVD, which may be evidence of subclinical inflammation [5].

Ginger (*Zingiber officinale* Roscoe) that is abundant in bioactive substances such as gingerols and shogaols possess anti-inflammatory and antioxidant effects [6]. Randomized controlled trials indicate that ginger supplementation (1-3 g/day 8-12 weeks) can decrease hs-CRP and IL-6 in T2DM patients probably due to NF- κ B inhibition and antioxidant properties [7]. The proposed pilot study is focused on determining the impact of ginger supplement use on blood pressure, glucose, and inflammatory markers (hs-CRP, IL-6, PCT) in patients with T2DM and CVD to evaluate the potential of this supplement as an adjunctive therapy in the treatment of cardiometabolic inflammation and better clinical outcomes.

Materials and Methods.

Subjects and Study design:

This prospective interventional before–after study with a healthy reference control group was conducted to evaluate the effects of ginger supplementation on inflammatory, lipid, glycemic, and blood pressure parameters in 80 participants (aged 27–44 years). The population was split into two groups: Group A (control, n=40) and Group B (intervention, n=40) having mild dyslipidemia, hyperglycemia (as the symptom of prediabetes or early type 2 diabetes) and hypertension with no pharmacological interventions. The exclusion criteria were severe comorbidities, pregnancy, or any medication that can confound the results and the eligibility was verified by clinical assessment. The lipid profiles (triglycerides, total cholesterol, LDL, HDL, VLDL), glycemic (fasting glucose), and inflammatory (IL-6, hs-CRP, PCT) baseline measurements were included. Group B took a ginger infusion that was made in a large cup of water and this was taken twice a day (in the morning and in the evening) in a 4-month period of time. Four months later, Group B was the only group that was re-assessed to determine the effectiveness of the intervention. The research was carried out in John Specialized Laboratory, which is a private laboratory in Baghdad, Iraq, licensed by the Iraqi Ministry of Health, between April and

November 2025. All the procedures were done according to ethical standards and informed consent was received and the Institutional Review Board of Ibn Sina University of Medical and Pharmaceutical Sciences gave the consent.

Sample Preparation:

10 mL of venous blood under fasting conditions (at least 8 hours) were taken in each participant through the standard venipuncture procedures. Serum separator tubes were used to collect blood samples which were centrifuged at 3000 rpm and 10 minutes to separate serum. To assure stability of the biomarkers in the serum, the serum was aliquoted in sterile labeled cryovials and kept at -80°C until the analysis. This was followed by another procedure of 4 months to determine the follow up assessments.

Biomarker Analysis:

Baseline data was conducted on both groups with the measurement of total cholesterol, triglycerides, low density lipoprotein (LDL), high density lipoprotein (HDL), very low-density lipoprotein (VLDL), fasting glucose, high sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), and procalcitonin (PCT). The quantification of all biomarkers was done in Roche Cobas e411 and c311 analyzers (Roche Diagnostics, Germany) and the assays were standardized and reliable. Group B was again assessed after 4 months about changes in these parameters. The blood samples were taken at fasting conditions.

Statistical Analysis:

The data will have an expression in the form of means \pm standard deviations or medians (interquartile ranges). The level of statistical significance will be defined as $p < 0.05$. To only guarantee the availability of at least 80% power to detect clinically meaningful changes in primary outcomes, power calculations will be carried out. Statistical software, which will be used to conduct the analysis, will be SPSS.

Results.

A comparative analysis of biochemical and clinical parameters between Control Group A and Group B before treatment demonstrated statistically significant differences in most variables, as assessed by independent t-tests. Specifically, Group B exhibited significantly higher mean levels of hs-CRP (4.70 ± 0.22 vs. 2.37 ± 0.10 mg/L, $t = -9.604$, $p < 0.01$), IL-6 (7.58 ± 0.27 vs. 2.83 ± 0.28 pg/mL, $t = -12.351$, $p < 0.01$), VLDL (44.87 ± 0.71 vs. 19.55 ± 0.90 mg/dL, $t = -21.994$, $p < 0.01$), LDL (174.75 ± 1.56 vs. 118.93 ± 3.16 mg/dL, $t = -15.845$, $p < 0.01$), triglycerides (224.32 ± 3.57 vs. 97.77 ± 4.52 mg/dL, $t = -21.994$, $p < 0.01$), total cholesterol (255.22 ± 4.18 vs. 166.97 ± 4.43 mg/dL, $t = -14.489$, $p < 0.01$), fasting glucose (187.63 ± 12.36 vs. 91.77 ± 1.76 mg/dL, $t = -7.680$, $p < 0.01$), systolic blood pressure (151.05 ± 1.19 vs. 123.33 ± 0.88 mmHg, $t = -17.650$, $p < 0.01$), and diastolic blood pressure (92.22 ± 0.42 vs. 83.50 ± 0.77 mmHg, $t = -10.529$, $p < 0.01$), compared with Control Group A. Conversely, HDL levels were significantly lower in Group B (31.25 ± 0.90 vs. 45.47 ± 0.98 mg/dL, $t = 10.733$, $p < 0.01$). In contrast, no statistically significant difference was observed in procalcitonin (PCT) levels between

the two groups (0.05 ± 0.00 vs. 0.07 ± 0.00 ng/mL, $t = 2.503$, $p > 0.05$). Overall, prior to intervention, Group B exhibited a more pronounced inflammatory, dyslipidemic, hyperglycemic, and hypertensive profile compared with Control Group A, with the exception of PCT (Table 1).

Ginger Supplementation Intervention:

A comparative analysis of biochemical and clinical parameters in Group B before and after a 4-month ginger supplementation protocol, assessed using paired t-tests, demonstrated statistically significant changes in most variables. Following the intervention, Group B exhibited significant reductions in mean levels of hs-CRP (3.75 ± 0.10 vs. 4.70 ± 0.22 mg/L, $t = 3.914$, $p < 0.01$), IL-6 (5.50 ± 0.13 vs. 7.58 ± 0.27 pg/mL, $t = 8.084$, $p < 0.01$), VLDL (37.05 ± 0.88 vs. 44.87 ± 0.71 mg/dL, $t = 7.274$, $p < 0.01$), LDL (159.72 ± 1.51 vs. 174.75 ± 1.56 mg/dL, $t = 11.478$, $p < 0.01$), triglycerides (193.95 ± 3.22 vs. 224.32 ± 3.57 mg/dL, $t = 7.993$, $p < 0.01$), total cholesterol (210.03 ± 4.28 vs. 255.22 ± 4.18 mg/dL, $t = 13.198$, $p < 0.01$), fasting glucose (136.30 ± 2.77 vs. 187.63 ± 12.36 mg/dL, $t = 4.162$, $p < 0.01$), systolic blood pressure (136.93 ± 1.17 vs. 151.05 ± 1.19 mmHg, $t = 8.480$, $p < 0.01$), and diastolic blood pressure (82.22 ± 0.27 vs. 92.22 ± 0.42 mmHg, $t = 19.950$, $p < 0.01$). In contrast, HDL levels increased significantly after the intervention (34.13 ± 0.65 vs. 31.25 ± 0.90 mg/dL, $t = -3.116$, $p < 0.01$). No statistically significant change was observed in procalcitonin (PCT) levels (0.05 ± 0.00 vs. 0.05 ± 0.00 ng/mL, $t = -0.534$, $p > 0.05$). Overall, ginger supplementation was associated with significant improvements in inflammatory markers, lipid profile, glycemic control, and blood pressure parameters, with the exception of PCT, indicating a favourable treatment effect (Table 2).

Discussion.

The present research paper explains the therapeutic value of ginger supplementation as an adjunctive treatment of patients with type 2 diabetes mellitus (T2DM) and cardiovascular disease (CVD). The noted mitigation of cardiometabolic risk factors in terms of inflammatory biomarkers, lipid profiles, glycemic control and blood pressure demonstrates ginger-wide effects of reducing cardiometabolic risk factors. The bioactive components of ginger, gingerol, shogaol, and paradol are very effective in inflammation reduction, lipid change, glucose regulation, and free radical combatants [8]. Ginger has an integrative nature that makes it a promising natural ingredient in preventive cardiology especially in the treatment of the etiology of T2DM and CVD [9]. This is because ginger is good in health since it is called an antioxidant [10]. Ginger bioactive compounds counteract lipid peroxidation and ROS, and increase antioxidant activities, such as superoxide dismutase and catalase [11]. Endothelial dysfunction, atherogenesis, and insulin resistance in T2DM and CVD are caused by oxidative stress [12]. Ginger could also improve the benefits of inflammatory and metabolic processes by reducing the oxidative damage, which worsens chronic inflammation and dyslipidemia that causes cardiovascular disease [13]. The treatment had a major decreasing effect on hs-CRP and IL-6, demonstrating the strong anti-inflammatory properties of ginger [14]. Those downstream mechanisms are probably mediated by inhibition of pro-inflammatory

Table 1. Comparison between the Control Group A and the Group B Before Treatment.

Parameter	Control Group Mean ± SE	Before Treatment Mean ± SE	t-statistic	p-value	Significance
hs-CRP (mg/L)	2.37 ± 0.10	4.70 ± 0.22	-9.604	p < 0.01	Significant
IL-6 (pg/mL)	2.83 ± 0.28	7.58 ± 0.27	-12.351	p < 0.01	Significant
PCT (ng/mL)	0.07 ± 0.00	0.05 ± 0.00	2.503	p > 0.05	Not Significant
VLDL (mg/dL)	19.55 ± 0.90	44.87 ± 0.71	-21.994	p < 0.01	Significant
LDL (mg/dL)	118.93 ± 3.16	174.75 ± 1.56	-15.845	p < 0.01	Significant
HDL (mg/dL)	45.47 ± 0.98	31.25 ± 0.90	10.733	p < 0.01	Significant
Triglyceride (mg/dL)	97.77 ± 4.52	224.32 ± 3.57	-21.994	p < 0.01	Significant
Total Cholesterol (mg/dL)	166.97 ± 4.43	255.22 ± 4.18	-14.489	p < 0.01	Significant
Glucose (mg/dL)	91.77 ± 1.76	187.63 ± 12.36	-7.680	p < 0.01	Significant
Systolic BP (mmHg)	123.33 ± 0.88	151.05 ± 1.19	-17.650	p < 0.01	Significant
Diastolic BP (mmHg)	83.50 ± 0.77	92.22 ± 0.42	-10.529	p < 0.01	Significant

Table 2. Comparison between the Group B Before Treatment and the Group B After Treatment.

Parameter	Before Treatment Mean ± SE	After Treatment Mean ± SE	t-statistic	p-value	Significance
hs-CRP (mg/L)	4.70 ± 0.22	3.75 ± 0.10	3.914	p < 0.01	Significant
IL-6 (pg/mL)	7.58 ± 0.27	5.50 ± 0.13	8.084	p < 0.01	Significant
PCT (ng/mL)	0.05 ± 0.00	0.05 ± 0.00	-0.534	p > 0.05	Not Significant
VLDL (mg/dL)	44.87 ± 0.71	37.05 ± 0.88	7.274	p < 0.01	Significant
LDL (mg/dL)	174.75 ± 1.56	159.72 ± 1.51	11.478	p < 0.01	Significant
HDL (mg/dL)	31.25 ± 0.90	34.13 ± 0.65	-3.116	p < 0.01	Significant
Triglyceride (mg/dL)	224.32 ± 3.57	193.95 ± 3.22	7.993	p < 0.01	Significant
Total Cholesterol (mg/dL)	255.22 ± 4.18	210.03 ± 4.28	13.198	p < 0.01	Significant
Glucose (mg/dL)	187.63 ± 12.36	136.30 ± 2.77	4.162	p < 0.01	Significant
Systolic BP (mmHg)	151.05 ± 1.19	136.93 ± 1.17	8.480	p < 0.01	Significant
Diastolic BP (mmHg)	92.22 ± 0.42	82.22 ± 0.27	19.950	p < 0.01	Significant

transcription factors such as nuclear factor-kappa B (NF-kB), cyclooxygenase-2 (COX-2) and lipoxygenase that play a key role in the regulation of cytokine production and inflammatory cascades [15]. These anti-inflammatory mechanisms are necessary because chronic low-grade inflammation is a key cause of atherosclerosis and CVD development in T2DM [16]. On the contrary, the lack of profound shifts in the procalcitonin (PCT) concentrations is consistent with its low sensitivity in the role of a non-infectious inflammatory steady state, since PCT is mainly raised during acute infection of bacteria and is not as applicable to the low-grade inflammatory state that ginger is aimed [17].

There were also positive changes in lipid profiles that were induced by ginger supplementation, including a decrease in low-density lipoprotein cholesterol (LDL-C), very low-density lipoprotein cholesterol (VLDL-C), total cholesterol levels, and triglycerides, as well as an increase in high-density lipoprotein cholesterol (HDL-C) [18]. These modifications indicate that the changes are towards a less atherogenic lipid profile, probably due to the capacity of ginger to increase the expression of LDL receptors, inhibit the hepatic production of cholesterol via the regulation of 3-hydroxy-3-methylglutaryl-CoA (HMG-CoA) reductase, and stimulate the lipoprotein lipase activity [19]. The lipid-lowering effects of ginger are also due to the effect of the reduction of intestinal cholesterol absorption by ginger [20]. The reduction in the levels of triglycerides and VLDL-C

is especially high to be observed, as hypertriglyceridemia is an independent risk factor of CVD [21]. The small rise in HDL-C facilitates the reverse cholesterol transport, which is an essential process of decreasing the atherosclerotic plaque formation [22]. Ginger supplementation had a significant positive effect on the level of fasting glucose, which is a sign of an improved level of glucose homeostasis. Such effects can probably be explained by the fact that ginger increases insulin sensitivity, facilitates glucose uptake through glucose transporter type 4 (GLUT4) and prevents gluconeogenesis by suppressing the activity of enzymes of glucose-6-phosphatase [23]. The most important factor in T2DM patients with a high cardiovascular risk is improved glycemic control since hyperglycemia stimulates endothelial dysfunction and increases atherosclerosis, which worsens the progression of CVD [24]. The other aspect of the therapeutic profile of ginger is blood pressure regulation. Vasodilatory effects and angiotensin-converting enzyme (ACE) inhibition and endothelial functions also have high chances of mediating the ability of the intervention to lower systolic and diastolic blood pressure [25]. These effects are in addition to the reported positive changes in lipid and glycemic indices, and they both lead to less strain on the cardiovascular system in patients with T2DM and CVD [26]. Nonetheless, the inconsistency in the blood pressure results of individuals implies that different treatment strategies should be applied to each individual to maximize effectiveness. The contributory effect of antioxidant,

anti-inflammatory, lipid-modulating, glucose-regulating, and blood pressure-lowering effects of ginger serves as a highlight of its possibility as a holistic alternative in prevention cardiology. This study has limitations. Failure to separate the effects of ginger with the confounding factors makes it difficult to accrue credit to ginger only. The brief intervention period does not provide any insights into the long-term efficacy and safety, and only larger, multicenter randomized trials with long-term follow-up periods can provide any insights into long-term effects and clinical outcomes, including cardiovascular events rates.

Conclusion.

In conclusion, this study provides robust evidence supporting ginger supplementation as an effective adjunctive therapy for managing cardiometabolic risk factors in patients with T2DM and CVD. Its integrative effects on inflammation, lipid metabolism, glucose homeostasis, blood pressure, and oxidative stress position ginger as a promising natural compound for enhancing cardiovascular health. These findings advocate for further research to validate ginger's clinical utility and optimize its integration into preventive cardiology strategies.

Acknowledgments.

The authors owe their study immense support to Mustansiriyah University and the Iraqi Center of Cancer and Medical Genetic Research, and the Ibn Sina University of Medical and Pharmaceutical Sciences in Baghdad, Iraq.

Author Contributions.

Baraa Ahmed Saeed, Anwer Jaber Faisal, Ahmed Abdulraheem Ibrahim Dahy, Mohanad Luay Jawhar and Noor Yahya Muneer were involved in the study's conceptualization and design, and they were also responsible for its material planning, data collection, and analysis. [Anwer Jaber Faisal] and [Baraa Ahmed Saeed] wrote the first version of the paper. Before publication, all writers reviewed and commented on drafts of the work. The final version of the paper was approved after all writers had reviewed it.

Funding.

No external funding was obtained for this research.

Conflict of Interest.

The authors declare no conflicts of interest.

Ethics Statement.

The research protocol was approved by the Central Scientific Committee at Ibn Sina University's College of Dentistry for Medical and Pharmaceutical Sciences.

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