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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 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.

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

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 საყურადღებო!

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, შემავსებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრაფიის ფოტოსურათები 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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ASSOCIATION BETWEEN INFLAMMATORY MARKERS, LIVER ENZYMES, LIPID PROFILE, AND VITAMIN D STATUS IN ADULTS WITH OBESITY: A CROSS-SECTIONAL STUDY

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Abstract.

Background: Vitamin D has pleiotropic effects on insulin secretion, lipid metabolism, immune modulation, and blood pressure regulation through ubiquitously expressed vitamin D receptors.

Objective: This cross-sectional study sought to investigate the associations between inflammatory markers, liver enzymes, lipid profile, glycemic parameters, hemodynamic measures, and vitamin D status in adults with obesity.

Methods: A total of 68 adults with obesity were enrolled and stratified by age younger than 40 years versus older than 40 years and BMI (normal weight BMI < 25 kg/m² versus overweight BMI ≥ 25 kg/m², or both. Demographic characteristics, smoking status, and anthropometric measurements were recorded. Lipid profile, glycemic control, liver enzymes, vitamin D, and inflammation markers were measured.

Results: Adults older than 40 years had significantly higher systolic blood pressure ($p=0.016$), fasting serum glucose ($p=0.017$), and HbA1c ($p=0.005$) compared to younger adults. Overweight individuals demonstrated significantly higher CRP levels ($p=0.040$) compared to normal-weight individuals. Triglycerides (AUC=0.638) and systolic blood pressure (AUC=0.634) demonstrated a weak association with vitamin D deficiency.

Conclusions: These results demonstrated the multifactorial nature of vitamin D regulation in obesity and reflect the unreliability of measuring serum vitamin D in clinical settings as a diagnostic tool in obese individuals, particularly when coexisting with ageing.

Key words. Obesity, Vitamin D, lipid profile, liver enzymes, glycemic control, inflammation.

Introduction.

Obesity is a public health challenge of the twenty-first century, carrying extensive loads on global healthcare systems, with approximately 1.9 billion adults considered overweight in 2016 [1]. This increasing rate is conjoined with substantial morbidity and mortality, resulting in doubled annual deaths alongside a rising risk of chronic diseases, including metabolic diseases, cardiac disorders, and malignancies, with an aetiology combining genetic tendency, lifestyle habits, physical sedentariness, and increased high-calorie diets [2-5].

Vitamin D is a lipophilic secosteroid hormone and is characteristically known for its necessary function in calcium and phosphate homeostasis, enhancing intestinal calcium absorption and promoting sufficient bone mineralization [6,7]. Vitamin D deficiency has arisen as a prevalent nutritional issue, influencing billions of people worldwide [8,9]. However, recent evidence has demonstrated its marked extra-skeletal

functions, directed through vitamin D receptors (VDR) that are endogenously expressed over different human tissues, including pancreatic β -cells, adipose tissue, skeletal muscle, and immune cells [10-12]. Through these receptors, vitamin D exerts pleiotropic effects on insulin secretion, immune modulation, cell differentiation, lipid metabolism, and inflammatory regulation [13,14].

The interaction between overweight and vitamin D insufficiency has acquired great scientific consideration in recent years [11,15,16]. Epidemiological evidence has demonstrated an inverse relationship between adiposity and circulating 25-hydroxyvitamin D [25(OH)D] levels; studies have confirmed that obesity causes vitamin D shortage rather than the reverse [17]. Several mechanisms have been considered to rationalize this association, including dilution by adipose tissue mass, trapping of vitamin D in the lipophilic milieu of adipose tissues, and obesity-induced mutilations in vitamin D metabolism and hydroxylation [11,12].

Vitamin D plays a role in metabolism via shaping insulin secretion, lipid metabolism, and blood pressure regulation [13,18]. Deficiency has been associated with insulin resistance, dyslipidemia, and overactivation of the renin-angiotensin-aldosterone system (RAAS), perhaps potentiating obesity-mediated metabolic dysregulation [19]. Moreover, inflammation is the cause of obesity, with adipose tissue functioning as an active endocrine organ secreting pro-inflammatory cytokines, which stimulate hepatic C-reactive protein (CRP) production [20]. Given the anti-inflammatory feature of vitamin D, its shortage may further jeopardize the inflammatory environment for obesity [21,22].

The association between vitamin D and metabolic markers remains inconclusive. Several earlier studies confirmed an inverse relationship between vitamin D and triglycerides, blood pressure, and glycemic parameters. Other studies reported discrepant results of vitamin D supplementation on metabolic outcomes and liver metabolism. Despite this evidence, the association of vitamin D and metabolic health remains inconclusive. Therefore, this cross-sectional study was designed to investigate the association between inflammatory markers, liver enzymes, lipid profile, and vitamin D status in adults with obesity.

Materials and Methods.

Study design: This cross-sectional study enrolled a total of 68 participants, subdivided according to age (<40 years versus ≥40 years) and body mass index (BMI < 25 kg/m² versus ≥25 kg/m²). A written consent form was collected from individual participants before enrollment. The study was approved by the Ethics Committee of the College of Medicine at Ninevah University (Approval session number 07 on 08 January 2026).

The study was conducted from February to July 2026 at City General Hospital (Mosul, Iraq).

Inclusion criteria:

Obese (BMI ≥ 25 kg/m²) adults (≥ 18 years) who agreed to participate in the study and were willing to provide fasting blood samples

Exclusion criteria:

- Obese adults with any sort of chronic disease were excluded, such as diabetes, chronic liver or kidney diseases, cardiovascular diseases, and thyroid disorders.
- Obese adults with current use of medications, including vitamin D supplementation,
- lipid-lowering agents or antihypertensive agents, corticosteroids, immunosuppressive agents, and anti-inflammatory agents
- Obese adults during pregnancy or lactation

Sample size: A total of 68 cases were enrolled based on the following equation.

$$n = \frac{z^2 p(1 - p)}{e^2}$$

Where,

n= sample size (68)

Z= confidence level (90%)

P= population proportion (10%)

e= margin of error (5%)

90% Confidence (z=1.645), e=0.06

$n = (1.645)^2 \times 0.10 \times 0.90 / (0.06)^2 = 67.7 \approx 68 \checkmark$

Based on the above equation, the calculated sample size was nearly 68 in total. With n=68, p=10%, and 90% confidence, a margin of error of 6% is acceptable, making the sample size of 68 suitable for identifying the overall effects. However, the results still need to be cautiously explained for marginal p-values, which do require larger sample size studies.

Data collection: Demographic parameters (age, sex, smoking status, and BMI) were collected for all participants. Blood pressure was obtained using standard procedures.

Serum collection: Venous blood was withdrawn from 12-hour fasting participants into collection tubes, part centrifuged and stored at -20°C for further analysis, and the other part immediately analysed for HbA1c assessment.

Biochemical analyses: All biochemical parameters were assessed using reagents and assays provided by Biolabo (France).

Glycemic profile: FSG was measured using enzymatic methods on an automated biochemical analyser. Glycated haemoglobin (HbA1c) was determined using turbidimetric immunoassay methods, following the manufacturer's recommended procedures.

Lipid profile: TC, TG, and HDL-C were quantified using enzymatic colourimetric methods on an automated analyzer.

LDL-C was calculated using **Friedewald's formula:** LDL-C (mg/dL)=TC - HDL-C - (TG/5.0).

Liver enzymes: Based on manufacturer instructions, ALT and AST were detected using enzymatic methods on an automated analyzer.

Inflammatory marker: Based on manufacturer instructions, CRP was quantified by a quantitative turbidimetric immunoassay method based on the photometric measurement of turbidity using an antigen-antibody reaction measured at optical density 340nm using the endpoint method.

Vitamin D: Based on manufacturer instructions, the level of 25-OH Vitamin D was quantified using ELISA methods measured at optical density 450nm using a microplate reader.

Statistical analysis: Results were analysed using GraphPad Prism (V10, USA). Data were expressed as mean $\pm$ standard deviation (SD) or frequencies and percentages for continuous and non-continuous variables, respectively. Subgroups were compared using independent t-tests and chi-square (χ^2) tests for continuous and non-continuous variables, respectively. A p-value<0.05 was considered significant.

ROC analysis was conducted to assess the diagnostic benefits of measured biochemical markers for discriminating vitamin D deficiency based on a vitamin D cutoff value of <20 ng/mL. For individual parameters, the area under the curve (AUC) was measured with 95% confidence intervals and interpreted as below.

AUC ≤ 0.50 : no diagnostic value.

AUC=1.00: complete diagnostic value.

AUC 0.50–0.69: poor diagnostic value.

AUC 0.70–0.79: acceptable diagnostic value.

AUC 0.80– ≥ 0.90 : excellent diagnostic value.

Results.

The analytical and biochemical data were compared based on age into two groups: <40 years (n=30) group and ≥ 40 years (n=38) group (Table 1). The percentage of males was not significantly higher (p=0.181) (65.80% versus 46.70%), and the percentage of smokers was not significantly lower (p=0.083) (23.70% versus 46.70%) than in the older group. No significant difference (p=0.169) was detected in BMI between the two groups (29.93 $\pm$ 4.28 kg/m² versus 28.22 $\pm$ 5.90 kg/m²). The older age group showed significantly higher SBP compared to the younger group (120.79 $\pm$ 10.43 mmHg versus 120.00 $\pm$ 10.12 mmHg, p=0.016). However, DBP did not differ significantly between the groups (p=0.154). FSG and HbA1c were significantly (p=0.017; p=0.005) elevated in the older group. However, the lipid results, liver enzymes, CRP, and vitamin D demonstrated non-significant (p>0.05) differences between the groups.

The analytical and biochemical data were compared based on BMI into two groups: Normal BMI <25 kg/m² group (n=11)

and Overweight ≥ 25 kg/m² group (n=57) (Table 2). For sex and smoking, non-significant ($p>0.05$) differences were demonstrated between the groups. The blood pressures were similar between the normal BMI and overweight groups (SBP, $p=0.581$; DBP, $p=0.860$). Glycemic control, assessed by FSG and HbA1c, did not differ significantly between the two BMI groups (FSG, $p=0.947$; HbA1c, $p=0.611$). The lipid profile demonstrated no significant differences between the groups ($p>0.05$), with a slight non-significant elevation of TG levels in the overweight group ($p=0.116$). Non-significant differences

in liver enzymes were observed between the normal BMI and overweight groups for either ALT ($p=0.851$) or AST ($p=0.603$). The overweight group demonstrated significantly ($p=0.040$) higher CRP compared to the normal group (2.80 ± 7.00 mg/L versus 0.55 ± 1.81). Conversely, vitamin D demonstrated a non-significant ($p=0.850$) difference between groups.

When comparing groups of older individuals with high BMI with younger individuals with normal BMI, blood pressure has shown non-significant differences between groups (SBP,

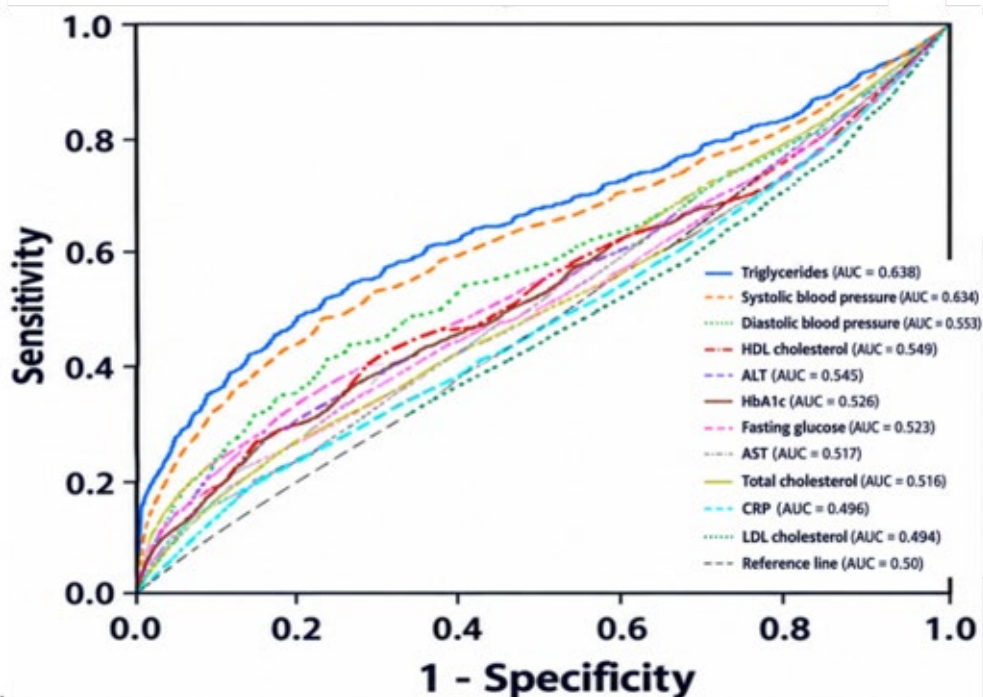


Figure 1. Receiver operating characteristic (ROC) curves for biochemical parameters in predicting vitamin D deficiency.

Table 1. Comparison of demographic, clinical, and biochemical characteristics according to age group.

Variables		<40 years (n=30)	≥ 40 years (n=38)	χ^2	P value
Demographic characteristics	Age, years	28.83 $\pm$ 6.23	51.58 $\pm$ 8.64	----	0.001
	Male, n (%)	14 (46.70)	25 (65.80)	1.79	0.181
	Female, n (%)	16 (53.30)	13 (34.20)		
	Smokers, n (%)	14 (46.70)	9 (23.70)	3.00	0.083
	BMI, kg/m ²	28.22 $\pm$ 5.90	29.93 $\pm$ 4.28	----	0.169
Blood pressure	SBP, mmHg	120.00 $\pm$ 10.12	120.79 $\pm$ 10.43	----	0.016
	DBP, mmHg	80.13 $\pm$ 10.90	80.45 $\pm$ 10.88	----	0.154
Glycemic profile	FSG, mg/dL	99.73 $\pm$ 17.71	115.21 $\pm$ 30.92	----	0.017
	HbA1c, %	5.27 $\pm$ 0.52	5.84 $\pm$ 0.97	----	0.005
Lipid profile	TC, mg/dL	170.80 $\pm$ 34.46	180.87 $\pm$ 41.35	----	0.288
	LDL-C, mg/dL	101.57 $\pm$ 33.84	107.37 $\pm$ 36.65	----	0.505
	HDL-C, mg/dL	41.77 $\pm$ 9.18	43.61 $\pm$ 17.52	----	0.604
	TG, mg/dL	130.50 $\pm$ 64.66	154.47 $\pm$ 75.62	----	0.172
Liver enzymes	ALT, U/L	33.62 $\pm$ 28.11	24.89 $\pm$ 17.40	----	0.121
	AST, U/L	24.98 $\pm$ 16.24	21.23 $\pm$ 11.31	----	0.267
CRP, mg/L		1.30 $\pm$ 3.66	3.34 $\pm$ 7.99	----	0.200
Vitamin D, ng/mL		19.37 $\pm$ 8.62	20.78 $\pm$ 10.31	----	0.551

Continuous variables expressed as mean $\pm$ SD; non-continuous variables expressed as frequency and percentages; p-values less than 0.05 considered significant using independent-samples t-tests. χ^2 =Chi-square, BMI=Body mass index, SBP=Systolic blood pressure, DBP=Diastolic blood pressure, FSG=Fasting serum glucose, HbA1c=Glycated hemoglobin, TC=Total cholesterol, LDL=Low density lipoprotein, HDL=High density lipoprotein, TG=Triglyceride, ALT=Alanine aminotransferase, AST=Aspartate aminotransferase

Table 2. Comparison of demographic, clinical, and biochemical characteristics according to BMI groups.

Variables		Normal BMI (<25 kg/m ²) (n=11)	Overweight/Obesity (≥25 kg/m ²) (n=57)	χ ²	P value
Demographic characteristics	Age, years	31.64±13.25	43.49±13.04		0.016
	Male, n (%)	5 (45.50)	34 (59.60)	0.29	0.590
	Female, n (%)	6 (54.50)	23 (40.40)		
	Smokers, n (%)	5 (45.50)	18 (31.60)	0.29	0.587
Blood pressure	SBP, mmHg	120.27±10.01	120.47±10.42		0.581
	DBP, mmHg	80.27±10.68	80.32±10.94		0.860
Glycemic profile	FSG, mg/dL	107.73±36.34	108.51±25.09		0.947
	HbA1c, %	5.47±0.81	5.61±0.86		0.611
Lipid profile	TC, mg/dL	183.00±40.95	175.16±38.28		0.567
	LDL-C, mg/dL	109.36±37.05	103.93±35.22		0.661
	HDL-C, mg/dL	47.27±11.27	41.93±14.84		0.191
	TG, mg/dL	116.36±57.41	149.21±73.14		0.116
Liver enzymes	ALT, U/L	29.95±22.92	28.51±23.19		0.851
	AST, U/L	25.71±20.17	22.34±12.28		0.603
CRP, mg/L		0.55±1.81	2.80±7.00		0.040
Vitamin D, ng/mL		20.72±10.80	20.05±9.40		0.850

Continuous variables expressed as mean±SD; non-continuous variables expressed as frequency and percentages; p-values less than 0.05 considered significant using independent-samples t-tests. χ²=Chi-square, BMI=Body mass index, SBP=Systolic blood pressure, DBP=Diastolic blood pressure, FSG=Fasting serum glucose, HbA1c=Glycated hemoglobin, TC=Total cholesterol, LDL=Low density lipoprotein, HDL=High density lipoprotein, TG=Triglyceride, ALT=Alanine aminotransferase, AST=Aspartate aminotransferase

Table 3. Comparison of demographic, clinical, and biochemical characteristics according to combined age and BMI groups.

Variables		Age <40 & BMI <25 (n=9)	Age ≥40 & BMI ≥25 (n=37)	P value
Blood pressure	SBP, mmHg	120.00±10.90	120.80±11.40	0.072
	DBP, mmHg	80.10±10.60	80.40±10.90	0.359
Glycemic profile	FSG, mg/dL	95.60±11.80	116.30±31.40	0.004
	HbA1c, %	5.10±0.33	5.88±0.98	0.001
Lipid profile	TC, mg/dL	181.20±43.50	180.40±41.10	0.958
	LDL-C, mg/dL	108.60±40.70	108.30±36.50	0.983
	HDL-C, mg/dL	48.20±11.50	42.70±10.80	0.183
	TG, mg/dL	109.70±49.80	154.80±76.50	0.031
Liver enzymes	ALT, U/L	27.60±20.80	29.70±24.60	0.813
	AST, U/L	24.90±18.30	22.00±10.90	0.643
CRP, mg/L		0.42±1.12	3.11±7.94	0.049
Vitamin D, ng/mL		21.90±10.70	19.80±9.80	0.580

Continuous variables expressed as mean±SD; non-continuous variables expressed as frequency and percentages; p-values less than 0.05 considered significant using independent-samples t-tests. BMI=Body mass index, SBP=Systolic blood pressure, DBP=Diastolic blood pressure, FSG=Fasting serum glucose, HbA1c=Glycated hemoglobin, TC=Total cholesterol, LDL=Low density lipoprotein, HDL=High density lipoprotein, TG=Triglyceride, ALT=Alanine aminotransferase, AST=Aspartate aminotransferase

p=0.072; DBP, p=0.359). However, older high BMI demonstrated significantly higher glycemic indices compared to younger normal BMI (FSG, p=0.004; HbA1c, p=0.001). Regarding lipid profile, non-significant differences existed between older high BMI and younger normal BMI groups except for TG, which have shown significantly higher levels in TG have shown in older high BMI than younger normal BMI (154.80±76.50 mg/dL versus 109.70±49.80 mg/dL, p=0.031). Regarding liver enzymes, non-significant differences existed between older high BMI and younger normal BMI (ALT, p=0.813; AST, p=0.643). Older high BMI was associated with significantly higher CRP compared to younger normal BMI (3.11±7.94 mg/L versus 0.42±1.12 mg/L, p=0.049), whereas vitamin D concentration demonstrated non-significant differences between the groups (p=0.580).

The ROC analysis, based on a vitamin D cutoff value of <20 ng/mL, demonstrated that among measured parameters, only TG and SBP revealed the highest AUC values, 0.638 and 0.634, respectively. Despite being lower than the threshold of 0.70, this reflects the poor diagnostic potential of vitamin D deficit compared with those with normal vitamin D levels. DBP, glycemic parameters, lipid parameters, CRP, and liver enzymes showed AUC values from 0.494 to 0.553, reflecting no diagnostic usefulness.

Discussion.

This cross-sectional study demonstrated that older adults presented with higher systolic blood pressure and glycemic control parameters compared to the younger group, with no

changes in lipid profile, liver enzymes, CRP, and vitamin D concentration between age groups. Overweight was associated with only elevated CRP levels compared to normal-weight groups, with the remaining markers stable. ROC analysis systemically revealed that TG have slight diagnostic potential for vitamin D deficiency, with comparable diagnostic potential for SBP.

The obese older adults group presented with slightly higher blood pressure, metabolic, and glycemic parameters compared to the younger group, reflecting the progressive nature of the ageing process in this population. This outcome is congruent with the concept that ageing contributes to the worsening of glycemic status and vascular dysfunction, despite normal adiposity [23,24]. Catena et al. demonstrated that serum vitamin D concentration was inversely proportional to age, blood pressure, and glycemic status in hypertensive patients [25], reflecting that age-associated vitamin D deficiency may drive these metabolic upset [26]. The lack of age-associated differences in lipid and liver parameters in the present study might be explained in the context of obesity-associated lipid and hepatic deficits appearing early and not advancing extensively with ageing [27,28]. Otherwise, it could reflect that the metabolic influence of overweight on lipid dysregulation and liver parameters plateaued after ageing, with subsequent worsening chiefly jeopardising glycemic and hemodynamic challenges [29,30].

The rise in CRP in the overweight group offers an additional clue for the pro-inflammatory condition reflected by surplus of adiposity. This outcome is congruent with earlier reports defining the role of adipose tissue as an endocrine organ generating pro-inflammatory markers, such as interleukin-6 and tumour necrosis factor-alpha, which consequently promote hepatic CRP formation [31,32]. A study conducted on obese subjects by Esteghamati et al. revealed that overweight, unhealthy subjects are associated with lower vitamin D levels and higher proinflammatory parameters compared to healthy normal overweight individuals [33]. This distinction is relevant to our findings, as the elevated CRP in our overweight/obese group suggests a higher proportion of metabolically unhealthy obesity, which may have implications for cardiovascular risk stratification [34].

Metabolic parameters and liver enzymes were not changed. This lack of association between systemic metabolic markers and CRP elevation could reflect that CRP is a more precise marker for obesity compared to metabolic parameters. Lipid parameters were unchanged with vitamin D levels; these outcomes align with the findings of meta-analyses, which have reported that supplementation with vitamin D has no impact on lipid parameters [15]. The unchanged liver enzymes contradict earlier studies [35,36], which might be explained in light of moderate obesity in participants of the present study.

ROC analysis confirmed that the measured biochemical markers have no clear and reliable diagnostic values, revealing that vitamin D regulation is multifactorial in nature. However,

TG and blood pressure have shown higher AUC and could be considered as cofactors for diagnosis of vitamin D deficiency; this is congruent with a systematic review that revealed that supplementation with vitamin D reduced TG and moderated blood pressure [15]. CRP demonstrated a weak association with vitamin D deficiency despite being elevated in obesity, reflecting the complex nature of CRP as a marker for systemic inflammation associated with obesity. Moreover, the lipid profile demonstrated no diagnostic value for obesity or ageing or both. The observed low, non-conclusive AUC values have been reported in earlier large-scale stud [16], which reported that the AUC for TG association with vitamin D deficiency is as low as AUC=0.626.

The discrepancy in the relationship between TG and vitamin D is confounded by factors such as obesity, sedentary lifestyle, sun exposure, and poor metabolic health. The obesity trap: vitamin D in the tissues, reducing skin synthesis of vitamin D, dilution effects by adipose tissue [15,17]. The results of this research revealed that clinicians should rely on direct measurement of 25-hydroxyvitamin D rather than measuring routine biochemical factors, especially in obese subjects [11,16]. The distinctive elevation in CRP among other biochemical factors perhaps necessitates incorporation of CRP in routine obese adult investigations [37].

The worthy limitations of the present study that need to be recognised include the cross-sectional nature of the study, limiting the variations between individuals during the study period, alongside the small sample size, which precludes generalizability of the study and hinders the statistics between groups. The use of CRP alone limits the characterization of the whole picture of inflammation associated with obesity. Hence, we do recommend conducting a larger-sample-size longitudinal study to capture the effects of confounding parameters; moreover, a mechanistic study profiling biomarkers joining vitamin D and inflammatory pathways. A gender-based study is required to facilitate understanding the impact of differences in sex hormones on vitamin D.

Conclusion.

The present study revealed that in obese adults with obesity, the metabolic derangement affects glycemic status and hemodynamic parameters, alongside obesity-associated changes in inflammation represented by CRP levels, reflecting the complex nature of vitamin D involvement in various metabolic and hormonal signalling, with a weak proportional association of vitamin D levels with all measured metabolic parameters.

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