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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

**GEORGIAN MEDICAL NEWS**

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

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

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

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

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

**WEBSITE**

www.geomednews.com

## К СВЕДЕНИЮ АВТОРОВ!

При направлении статьи в редакцию необходимо соблюдать следующие правила:

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

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

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

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

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

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

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

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

7. Фамилии отечественных авторов приводятся в оригинальной транскрипции.

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

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

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

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

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

**При нарушении указанных правил статьи не рассматриваются**.

## REQUIREMENTS

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

## ავტორთა საყურადღებოდ!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

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

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

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

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

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

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

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

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

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

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

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

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

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

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*Содержание:*

# LINKS BETWEEN DYSLIPIDEMIA AND RISK FACTORS IN ACUTE CORONARY SYNDROME

**Marwa AA Osman[1], Azza O Alawad[2], Tarig H Merghani[3]*, Minha M E Mohammed[4], Khalid AD Gasmalla[5].**

[1]*Internal Medicine Resident, Sudan Medical Specialization Board, Khartoum, Sudan.*

[2]*Department of Physiology, Faculty of Medicine, University of Al-Neelain, Khartoum, Sudan.*

[3]*Department of Physiology, RAK College of Medical Sciences, RAK Medical and Health Sciences University, RAK, UAE.*

[4]*Consultant Cardiology, Department of cardiology, Madani Heart Center, Sudan.*

[5]*Internal Medicine Resident, Sudan Medical Specialization Board, Khartoum, Sudan.*

## Abstract.

**Background:** Dyslipidemia, characterized by elevated low-density lipoprotein (LDL) cholesterol and triglycerides (TG) and low high-density lipoprotein (HDL) cholesterol, is one of the causes of acute coronary syndrome (ACS) and its complications. The interactions of lipid-abnormalities with established traditional cardiovascular risk factors guide prevention and management efforts. This article aims to estimate the presence of lipid profile abnormalities on patients with ACS and their relationship with demographic, clinical, and metabolic risk variables.

**Methods:** A hospital-based cross-sectional study was conducted at Madani Heart Center, Sudan. The study included 231 patients diagnosed with ACS based on clinical and laboratory findings. Data on demographic characteristics, cardiovascular risk factors (smoking, hypertension, diabetes mellitus), and biochemical markers (LDL, HDL, total cholesterol, TG, glycated hemoglobin, and random blood glucose) were collected. Statistical analysis included descriptive statistics, ANOVA, Chi-square tests, and bivariate correlations, with statistical significance set at $p \leq 0.05$.

**Results:** The mean age of participants was 60.6 ± 12.3 years. LDL cholesterol showed no significant association with smoking, hypertension, or diabetes. Triglycerides (TG) demonstrated positive correlations with glycated hemoglobin ($r = 0.180$, $p = 0.006$) and random blood glucose ($r = 0.163$, $p = 0.013$), indicating a strong link between dyslipidemia and glycemic control. HDL cholesterol correlated positively with body weight ($r = 0.149$, $p = 0.024$). Both TG and LDL levels showed insignificant association with ACS types, smoking and hypertension (p-values > 0.05).

**Conclusion:** Triglycerides showed significant associations with markers of impaired glycemic control in patients with ACS indicating the importance of triglyceride management, especially among diabetic individuals.

**Key words.** Dyslipidemia, acute coronary syndrome, triglycerides, LDL, cholesterol, HDL, glycated hemoglobin.

## Introduction.

### Overview of Acute coronary syndrome:

Acute coronary syndrome (ACS) is a spectrum of clinical presentations that include ST elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina. It is caused by a sudden reduction in coronary blood flow to the cardiac muscle, typically due to rupture of an atherosclerotic plaque within a coronary artery, leading to partial or complete thrombosis of the affected vessel. The cardinal symptom of ACS is chest pain or discomfort that is characteristically described as tightness, pressure, or a burning sensation. The pain is typically central but may radiate to the arm, shoulder, neck, back, upper abdomen, or jaw. Patients may also experience associated symptoms such as sweating, nausea, and shortness of breath.

ACS is a leading cause of death in many countries, particularly among individuals with multiple risk factors [1]. The high mortality rate underlines the significance of early diagnosis and adequate treatment. Mortality can be reduced by controlling modifiable risk factors such as hypertension (HT), diabetes mellitus (DM), cigarette smoking, and dyslipidemia. Among these, dyslipidemia, characterized by elevated low-density lipoprotein cholesterol (LDL-C) and reduced high-density lipoprotein cholesterol (HDL-C), is strongly linked to the progression of atherosclerosis and coronary artery disease [2].

Several studies have demonstrated the impact of traditional cardiovascular risk factors on the incidence and outcomes of ACS. Smoking, a well-established risk factor, accelerates atherosclerosis and promotes thrombogenesis, increasing the likelihood of plaque rupture and subsequent myocardial infarction. Diabetes mellitus, another major contributor, is associated with endothelial dysfunction, chronic inflammation, and dyslipidemia, all of which exacerbate coronary artery disease progression and worsen ACS outcomes. Additionally, hypertension plays a pivotal role by inducing vascular damage and increasing myocardial oxygen demand, further compounding the risk of adverse cardiac events [3].

In Sudan, the prevalence of ACS among diabetic patients is estimated to be 5.44%, with hypertension, advanced age, and longer duration of diabetes mellitus identified as significant risk factors [4]. Sudanese patients with ACS experience more complications compared to their counterparts in Western countries, with STEMI patients being particularly prone to in-hospital complications and more likely to receive reperfusion therapy [5]. Despite a positive trend in prescribing most individual medications for secondary prevention of ACS in Sudan, the use of guideline-recommended medications remains suboptimal [6]. Furthermore, the interplay between dyslipidemia and other cardiovascular risk factors in Sudanese ACS patients has not been extensively explored, warranting further investigation into their combined effects on disease progression and outcomes.

Although the prognosis of ACS patients has improved over the past two decades, there remains room for further advancement, particularly due to the high risk of recurrent cardiovascular events. As cholesterol plays a major role in CAD by contributing to atheroma formation, lipid-lowering therapy is a cornerstone of disease management. Evidence supports that early initiation of cholesterol-lowering drugs is associated with significant reductions in the risk of hospitalization and death due to CAD [7]. However, despite advancements in ACS management, high mortality rates and recurrent cardiovascular events remain a concern. A deeper understanding of how lipid profile abnormalities interact with other risk factors such as smoking, diabetes, and hypertension could help refine risk stratification and improve targeted interventions. This study investigates the association between LDL-C levels and the prognosis of ACS in patients undergoing coronary angiography, aiming to highlight the importance of effective lipid control in recovery and long-term outcomes.

## Methods.

### Study Design and Setting:

We conducted a hospital-based descriptive cross-sectional study at Madani Heart Center in Gezira State, Sudan, one of the main cardiology referral centers in the region, serving patients from across the country. The study included 231 patients diagnosed with acute coronary syndrome (ACS) who underwent coronary angiography and lipid profiling during their hospitalization. All patients were admitted to the cardiac care unit (CCU) or general cardiology ward and were managed according to standard ACS treatment guidelines.

### Ethical Approval:

Ethical approval for the study was obtained from the Sudan Medical Specialization Board, Research Department (7-2023). Permissions were also secured from the Federal Ministry of Health and the hospital director. Participant confidentiality was maintained by anonymizing the data that was used exclusively for research purposes. The study adhered to the principles and guidelines of medical research as outlined in the Declaration of Helsinki. Informed consent was obtained from all participants before inclusion in the study. Patients were provided with detailed information about the study objectives, procedures, and their right to withdraw at any stage without affecting their medical care.

### Inclusion and Exclusion Criteria:

All patients included in the study were admitted to either the cardiac care unit (CCU) or the general cardiology ward. Eligible participants were adult patients (≥18 years old) diagnosed with acute coronary syndrome (ACS) who had coronary angiography and a complete lipid profile assessment. Additionally, only those with comprehensive medical records, including documented cardiovascular risk factors and relevant laboratory results, were considered for inclusion.

Patients were excluded if they declined to participate, had missing or incomplete lipid profile data, or had chronic kidney disease (CKD) or chronic liver disease, as these conditions significantly affect lipid metabolism and could confound the study findings. To minimize potential bias in interpreting lipid profiles, we included only patients who were either untreated or had initiated therapy less than six months before presentation.

### Data Collection:

A structured data collection form was used to systematically gather comprehensive information from each participant. This included demographic details such as gender, age, and residence, along with clinical history covering key cardiovascular risk factors, including hypertension, diabetes mellitus, smoking status, and a family history of coronary artery disease. Electrocardiography (ECG) findings were documented, categorizing patients based on ST-elevation, non-ST elevation, or other abnormalities. Echocardiography results were also recorded, focusing on left ventricular ejection fraction (LVEF) and any structural abnormalities.

Coronary angiography outcomes were assessed by documenting the number and severity of coronary artery lesions. Laboratory investigations included a detailed lipid profile (total cholesterol, LDL-C, HDL-C, and triglycerides), blood glucose levels, and inflammatory markers. Data collection was carried out by trained medical personnel under the supervision of the research team. To ensure accuracy and reliability, all data entries were cross-checked by an independent reviewer before analysis.

### Statistical Analysis:

The data were analyzed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics, such as frequency tables, percentages, means, and standard deviations, were used to summarize the study variables. Comparisons between ACS subtypes (STEMI, NSTEMI, and unstable angina) were performed using ANOVA for continuous variables and the Chi-square test for categorical variables. Associations between lipid profile abnormalities and cardiovascular risk factors were assessed through bivariate analysis and Chi-square tests. A p-value of $\leq 0.05$ was considered statistically significant.

## Results.

The baseline characteristics of the study participants are summarized in Table 1. The mean age of the participants was 60.63 years (SD = 12.28), with a minimum age of 20 and a maximum of 87 years. The mean HbA1C level was 7.76% (SD = 2.31), and the mean random blood glucose (RBG) level was 201.58 mg/dl (SD = 97.21). The average low-density lipoprotein (LDL) level was 92.55 mg/dl (SD = 40.16), and the high-density lipoprotein (HDL) mean was 46.65 mg/dl (SD = 16.53). Other characteristics, including weight, total cholesterol, triglycerides, and ejection fraction, are detailed in Table 1.

The distribution of lipid profile categories, including LDL, HDL, total cholesterol, and triglycerides, across demographic characteristics, cardiovascular risk factors, glycemic status, and acute coronary syndrome (ACS) types is summarized in Table 2. Insignificant statistical differences were observed between LDL levels and gender, smoking status, hypertension, diabetes mellitus (DM), HbA1c categories, or ACS types. Similarly, HDL levels demonstrated similar insignificant association. Total cholesterol levels showed a statistically significant difference with hypertension ($p = 0.045$), with a higher proportion of participants without hypertension showing normal cholesterol levels. The associations with gender, smoking status, DM,

***Table 1.*** *General Characteristics of the Participants.*

| | N | Minimum | Maximum | Mean | Std. Deviation |
|---|---|---|---|---|---|
| Age (years) | 231 | 20 | 87 | 60.63 | 12.278 |
| Wight (kg) | 231 | 45 | 134 | 72.98 | 14.341 |
| HbA1C (%) | 231 | 4.7 | 15.0 | 7.762 | 2.3092 |
| RBG (mg/dl) | 231 | 58 | 536 | 201.58 | 97.208 |
| LDL (mg/dl) | 231 | 27.0 | 234.0 | 92.551 | 40.1591 |
| HDL (mg/dl) | 231 | 12.0 | 104.0 | 46.652 | 16.5303 |
| Total cholesterol (mg/dl) | 231 | 66 | 456 | 153.11 | 48.863 |
| Triglyceride (mg/dl) | 231 | 24 | 349 | 131.14 | 52.770 |
| Ejection Fraction% | 231 | 30 | 74 | 50.90 | 10.585 |

***Table 2.*** *Distribution of Lipid Profile Categories Across Demographic, Clinical, and Acute Coronary Syndrome Characteristics.*

| | Gender | | Smoking | | HT | | DM | | HbA1C | | ACS type | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Female | Male | Yes | No | Yes | No | Yes | No | < 6.5% | ≥ 6.5% | NSTE-MI | STEMI | UA |
| **LDL** | | | | | | | | | | | | | |
| Normal | 54 (23.4) | 99 (42.9) | 44 (19) | 109 (47.2) | 72 (31.2) | 81 (35.1) | 77 (33.3) | 76 (32.9) | 75 (32.5) | 78 (33.8) | 21 (9.1) | 90 (39) | 42 (18.2) |
| Borderline | 6 (2.6) | 7 (3) | 4 (1.7) | 9 (3,9) | 4 (1.7) | 9 (3,9) | 4 (1.7) | 9 (3,9) | 8 (3.5) | 5 (2.2) | 0 (0) | 11 (4.8) | 2 (0.9) |
| High | 27 (11.7) | 38 (16.5) | 17 (7.4) | 48 (20.8) | 38 (16.5) | 27 (11.7) | 34 (14.7) | 31 (13.4) | 26 (11.3) | 39 (16.9) | 9 (3.9) | 42 (18.2) | 14 (6.1) |
| P-value | 0.554 | | 0.905 | | 0.118 | | 0.356 | | 0.269 | | 0.362 | | |
| **HDL** | | | | | | | | | | | | | |
| Normal | 16 (6.9) | 22 (9.5) | 7 (3) | 31 (13.4) | 23 (10) | 15 (6.5) | 19 (8.2) | 19 (8.2) | 20 (8.7) | 18 (7.8) | 5 (2.2) | 19 (8.2) | 14 (6.1) |
| Borderline | 53 (22.9) | 89 (38.5) | 40 (17.3) | 102 (44.2) | 66 (28.6) | 76 (32.9) | 69 (29.9) | 73 (31.6) | 66 (28.6) | 76 (32.9) | 19 (8.2) | 95 (41.1) | 28 (12.1) |
| High | 18 (7.8) | 33 (14.3) | 18 (7.8) | 33 (14.3) | 25 (10.8) | 26 (11.3) | 27 (11.7) | 24 (10.4) | 23 (10) | 28 (12.1) | 6 (2.6) | 29 (12.6) | 16 (6.9) |
| P-value | 0.799 | | 0.216 | | 0.306 | | 0.867 | | 0.752 | | 0.181 | | |
| **Cholesterol** | | | | | | | | | | | | | |
| Normal | 77 (33.3) | 134 (58) | 59 (25.5) | 152 (65.8) | 99 (42.9) | 112 (48.5) | 104 (45) | 107 (46.3) | 100 (43.3) | 111 (48.1) | 24 (10.4) | 133 (57.6) | 54 (23.4) |
| Borderline | 6 (2.6) | 5 (2.2) | 4 (1.7) | 7 (3) | 9 (3.9) | 2 (0.9) | 6 (2.6) | 5 (2.2) | 5 (2.2) | 6 (2.6) | 4 (1.7) | 6 (2.6) | 1 (0.4) |
| High | 4 (1.7) | 5 (2.2) | 2 (0.9) | 7 (3) | 6 (2.6) | 3 (1.3) | 5 (2.2) | 4 (1.7) | 4 (1.7) | 5 (2.2) | 2 (0.9) | 4 (1.7) | 3 (1.3) |
| P-value | 0.442 | | 0.768 | | 0.045* | | 0.887 | | 0.978 | | 0.107 | | |
| **TG** | | | | | | | | | | | | | |
| Normal | 60 (26) | 83 (35.9) | 41 (17.7) | 102 (44.2) | 66 (28.6) | 77 (33.3) | 65 (28.1) | 78 (33.8) | 75 (32.5) | 68 (29.4) | 16 (6.9) | 92 (39.8) | 35 (15.2) |
| Borderline | 20 (8.7) | 56 (24.2) | 21 (9.1) | 55 (23.8) | 39 (16.9) | 37 (16) | 42 (55.3) | 34 (44.7) | 30 (13) | 46 (19.9) | 14 (6.1) | 43 (18.6) | 19 (8.2) |
| High | 7 (3) | 5 (2.2) | 3 (1.3) | 9 (3.9) | 9 (3.9) | 3 (1.3) | 8 (3.5) | 4 (1.7) | 4 (1.7) | 8 (3.5) | 0 (0) | 8 (3.5) | 4 (1.7) |
| P-value | 0.024* | | 0.957 | | 0.145 | | 0.187 | | 0.115 | | 0.340 | | |

**P< 0.05.*

***Table 3.*** *Correlation of Lipid Profiles with Clinical Parameters.*

| | | Age (years) | Wight (kg) | Hb (g/dl) | HbA1C (%) | RBG (mg/dl) | Ejection Fraction% |
|---|---|---|---|---|---|---|---|
| LDL (mg/dl) | Pearson Correlation | -0.063 | .125 | 0.064 | 0.116 | 0.113 | -0.078 |
| | Sig. (2-tailed) | 0.342 | 0.057 | 0.334 | 0.079 | 0.087 | 0.237 |
| | N | 231 | 231 | 231 | 231 | 231 | 231 |
| HDL (mg/dl) | Pearson Correlation | 0.061 | 0.149* | 0.007 | -0.024 | -0.069 | 0.015 |
| | Sig. (2-tailed) | 0.359 | 0.024 | 0.922 | 0.722 | 0.293 | 0.819 |
| | N | 231 | 231 | 231 | 231 | 231 | 231 |
| Total cholesterol (mg/dl) | Pearson Correlation | -0.099 | 0.089 | 0.037 | 0.009 | -0.015 | -0.086 |
| | Sig. (2-tailed) | 0.135 | 0.177 | 0.572 | 0.893 | 0.819 | 0.195 |
| | N | 231 | 231 | 231 | 231 | 231 | 231 |
| Triglyceride (mg/dl) | Pearson Correlation | -0.036 | 0.155* | 0.082 | 0.180** | 0.163* | -0.075 |
| | Sig. (2-tailed) | 0.586 | 0.018 | 0.215 | 0.006 | 0.013 | 0.256 |
| | N | 231 | 231 | 231 | 231 | 231 | 231 |

HbA1c levels, or ACS type are statistically insignificant. Triglyceride (TG) levels were significantly associated with gender ($p = 0.024$), with males more frequently demonstrating normal TG levels compared to females. Aside from this, TG levels showed insignificant associations with the examined variables.

Table 3 shows the correlations between lipid profiles (LDL, HDL, total cholesterol, and triglycerides) and various clinical parameters (age, weight, hemoglobin [Hb], HbA1C, random blood glucose (RBG), and ejection fraction). LDL levels showed insignificant correlations with any of the clinical parameters (p-values > 0.05). HDL levels were significantly correlated with weight ($r = 0.149$, $p = 0.024$), indicating a positive association. Triglycerides demonstrated multiple significant correlations. A positive correlation was observed with weight ($r = 0.155$, $p = 0.018$), HbA1C ($r = 0.180$, $p = 0.006$), and RBG ($r = 0.163$, $p = 0.013$), suggesting that higher triglyceride levels are associated with higher weight, HbA1C, and RBG values. No significant correlations were observed for total cholesterol across any clinical parameters.

## Discussion.

### Lipid profile abnormalities in patients with ACS:

This study evaluated lipid profile abnormalities in patients with ACS and their associations with demographic and clinical characteristics. Our findings showed no significant association between LDL cholesterol levels and smoking, hypertension, or diabetes. This aligns with reviews reporting that observational studies often fail to demonstrate strong correlations between LDL and individual risk factors, largely due to confounding variables and population heterogeneity [8,9]. However, intervention studies have consistently shown that LDL reduction is highly effective in lowering cardiovascular risk [9]. Interestingly, contrasting studies have reported significantly higher LDL levels in smokers and hypertensive patients that could be attributed to sample differences, dietary habits, or other lifestyle factors [10]. These discrepancies highlight the importance of considering population-specific variations in lipid profiles.

Triglycerides showed significant correlations with HbA1C and random blood glucose levels, emphasizing the close link between hypertriglyceridemia and metabolic disturbances. These findings are consistent with studies demonstrating strong associations between high TG levels, insulin resistance, and poor glycemic control, further underscoring TG's role in the pathophysiology of metabolic syndrome [9,10]. Elevated TG levels have also been implicated in promoting endothelial dysfunction and systemic inflammation, which contribute to cardiovascular risk. However, other studies have suggested that TG levels may have limited predictive value for cardiovascular outcomes when adjusted for other lipid markers, indicating that their role may be more nuanced in specific populations [9,11].

The weak positive correlation between HDL cholesterol and body weight observed in this study appears to contradict the conventional inverse association that is typically reported in epidemiological data. However, this finding may reflect population-specific metabolic characteristics, as ethnic variation in HDL regulation and lipid metabolism has been well documented [12]. Additionally, subsets of individuals with higher body weight may retain relatively preserved HDL-C levels, a pattern described within the framework of metabolically heterogeneous or "metabolically healthy" obesity [13]. Furthermore, higher body weight in some individuals may partly reflect greater fat-free mass rather than excess adiposity, and lean mass has been associated with more favorable HDL concentrations in several cohorts [14]. Consequently, our finding may not necessarily contradict established paradigms but rather reflect population-specific metabolic heterogeneity within the context of cardiovascular disease.

While traditionally viewed as a marker of cardiovascular health, recent studies emphasize that HDL functionality, including its role in reverse cholesterol transport and anti-inflammatory properties, may be a more reliable predictor of cardiovascular outcomes [8,15,16]. Some studies, however, argue that higher HDL levels remain inversely related to adverse outcomes, highlighting the complexity of HDL's role in cardiovascular health [17].

The analysis of lipid profiles in ACS subtypes revealed insignificant association with TG levels. Although some studies have reported higher triglyceride levels in ACS patients that are linked to pro-inflammatory mechanisms and endothelial dysfunction, our findings did not demonstrate such a relationship in this cohort [11]. No significant differences in LDL levels were observed across ACS types, supporting the notion that non-LDL mechanisms, such as inflammation and thrombosis, may play a more prominent role in acute cardiovascular events [9,10]. However, the absence of association between LDL and risk factors in this study may be influenced by residual confounding, including prior lipid-lowering therapy in some participants, which may have attenuated true LDL elevations.

Total cholesterol levels showed limited correlations with clinical markers and risk factors in this study. This is consistent with meta-analyses indicating that total cholesterol, as a standalone measure, has less predictive power compared to more specific lipid fractions like LDL or non-HDL cholesterol [8]. However, some studies have highlighted its utility in particular clinical contexts [8,9].

Our findings indicate that most lipid parameters did not vary significantly across demographic or clinical variables, suggesting a relatively homogeneous lipid distribution within this ACS cohort. The significant association between total cholesterol and hypertension is consistent with evidence linking elevated cholesterol to vascular stiffness and increased cardiovascular risk among hypertensive individuals while the gender-related difference observed in triglyceride levels, with males exhibiting more favorable TG profiles, reflects known sex-based metabolic patterns influenced by hormonal and lifestyle factors. It is worth noting that while LDL reduction remains a cornerstone of therapy, the significant correlation between triglycerides and glycemic parameters suggests that targeted interventions for hypertriglyceridemia, particularly in diabetic and insulin-resistant populations, should be emphasized. These insights should guide personalized lipid-lowering approaches in ACS patients, particularly those at high metabolic risk.

## Limitations.

This study has several limitations. First, it is a cross-sectional analysis, which limits the ability to establish causal relationships between lipid profiles and cardiovascular risk factors. Second, the study population was derived from a single center, potentially restricting the generalizability of findings to broader populations. Third, the analysis focused on traditional lipid parameters and did not incorporate advanced measures such as HDL functionality or non-HDL cholesterol, which may provide deeper insights into lipid-mediated cardiovascular risk. Fourth, although patients who had been on lipid-lowering therapy for more than six months were excluded, prior or recent medication use may still have influenced their lipid profile results, and some degree of residual confounding cannot be fully ruled out. Additionally, socioeconomic factors and dietary habits, which play a critical role in lipid metabolism, were not evaluated. Future studies should address these limitations to build a more comprehensive understanding of lipid abnormalities in ACS.

## Conclusion.

In general, our study emphasizes the importance of a comprehensive approach to lipid management in ACS. Triglycerides emerge as significant markers, particularly in patients with metabolic syndrome or diabetes, highlighting their role in residual cardiovascular risk. Future research should explore the long-term impact of lipid abnormalities on ACS prognosis and investigate personalized lipid-lowering approaches tailored to metabolic profiles. Given the evolving understanding of HDL cholesterol, future lipid management strategies should focus on assessing HDL functionality rather than solely relying on HDL levels. Public health initiatives aimed at improving lipid control through lifestyle interventions, medication adherence, and early screening may significantly reduce the burden of ACS, particularly in resource-limited settings like Sudan.

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## Conflict of interest.

The authors report no financial or any other conflicts of interest in this work.

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