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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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UNCONTROLLED TYPE 2 DIABETES MELLITUS MODULATED PLASMA LEVELS OF LIPID CATABOLIC PROTEINS

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Abstract.

Background and Objectives: This research is considered the first of its kind in Kirkuk City to study the activity of lecithin cholesterol acyl transferase (LCAT), the levels of apolipoprotein A-1 (Apo A-1), and Fatty acid binding protein-4 (FABP4) and some biochemical variables in T2DM patients who uncontrolled the disease (glycated hemoglobin (HbA1C) > 8 %) and compared with patients who controlled the disease (HbA1C < 7 %) and healthy controls (HbA1C ≤ 5.5 %), and find the correlation among them.

Methods: This research included 184 persons [128 patients (70 uncontrolled DM and 58 controlled DM + 56 healthy patients] aged (40–80) years from both sexes. The current study is divided into three groups: uncontrolled DM patients (G1) and controlled DM patients (G2) are compared to healthy controls (G3). Activity of LCAT, concentration of Apo A1, FABP4, insulin, fasting serum glucose (FSG), , lipid profile and HbAc1 % were evaluated and -correlated.

Results: The study found a significant rise in in HbAc1(%), FSG, TG, Cholesterol, LDL, VLDL, and FABP4 levels in uncontrolled diabetic patients (G1) compared to controlled diabetes (G2) and healthy controls (G3). At the same time, there are no significant (P value =0.2) differences between controlled DM patients (G2) and controlled healthy groups (G3). The study found no significant differences in insulin level among DM patients (G1), controlled DM patients (G2), and healthy controls (G3) (P = 0.2) while the LCAT activity, the concentration of HDL and ApoA1showed significant decreased (P=0.01) among G1, G2, compared to G3.

Key words. Apolipoprotein A1, fatty acid binding protein-4, lecithin cholesterol acyl transferase, diabetes mellitus, lipid profile.

Introduction.

Diabetes mellitus is a metabolic illness that affects the body's capacity to regulate glucose levels in the blood [1]. It is caused by a failure in insulin synthesis, insulin action, or both [2]. Genetic, environmental, and behavioral risk factors all contribute to diabetes [3]. Unlike other types of diabetes, T2DM patients do not require insulin therapy for their survival [4]. Undoubtedly, several factors contribute to the occurrence of this kind of DM. Autoimmune destruction of ß-cells is not present in this kind of DM. However, it is worth noting that most patients with this form of diabetes are obese, and obesity itself contributes to insulin resistance to some extent [5].

Cholesterol acyltransferase (LCAT) is an enzyme that increases the absorption of free cholesterol from lipid surfaces, particularly those found on high-density lipoproteins. LCAT is mostly produced in the liver, with tiny quantities produced in the testes and in stellate cells in the brain [6]. The plasma concentration of the LCAT enzyme is around 5-6 mg/L and is regulated by a variety of variables including age, diet, and smoking [7]. LCAT showed a preventative benefit against cardiovascular disease under normal physiological conditions [8]. However, various pathological and physio-pathological changes cause modifications in serum LCAT levels, resulting in a subsequent decrease in its protective effects [7]. The utilization of advanced measuring methods and the implementation of metabolomics and proteomics will unveil the connection between LCAT and additional disorders [8].

Apolipoprotein A (ApoA), an integral protein component of high-density lipoprotein (HDL), has garnered significant attention in the study of diabetes due to its crucial role in lipid metabolism and potential influence on glucose homeostasis [9]. As a major constituent of HDL, ApoA plays a critical function in reverse cholesterol transport, which is essential for removing excess cholesterol from peripheral tissues to the liver for excretion [10]. This process not only contributes to cardiovascular health but also appears to intersect with metabolic pathways implicated in diabetes. Studies suggest that lower levels of ApoA and HDL are associated with an increased risk of type 2 diabetes, potentially due to impaired lipid handling and systemic inflammation that exacerbate insulin resistance [11]. Furthermore, ApoA has been shown to exhibit anti-inflammatory and antioxidant properties that may protect pancreatic beta-cells from glucotoxicity and lipotoxicity—two key contributors to the pathogenesis of diabetes [12]. In this regard, therapeutic strategies aimed at increasing ApoA concentrations or enhancing HDL functionality might provide novel avenues for managing diabetic conditions by mitigating dyslipidemia and improving insulin sensitivity [9,10].

Fatty Acid Binding Protein 4 (FABP4), also known as adipocyte FABP or aP2, plays a significant role in the development and progression of diabetes, primarily through its involvement in lipid metabolism and inflammatory pathways [13]. As an intracellular lipid chaperone, FABP4 is abundantly expressed in adipocytes and macrophages, where it facilitates the transport and storage of fatty acids. Elevated levels of FABP4 have been associated with increased insulin resistance, a hallmark of type 2 diabetes, due to its capacity to disrupt normal insulin signaling pathways [14]. Furthermore, circulating FABP4 acts as an adipokine that exacerbates systemic inflammation by promoting the release of pro-inflammatory cytokines such as TNF-α and IL-6 [15]. This inflammatory response contributes to the deterioration of pancreatic β-cell function over time, reducing insulin secretion and further complicating glucose homeostasis. Intriguingly, studies have shown that genetic deletion or pharmacological inhibition of FABP4 can ameliorate metabolic abnormalities and enhance insulin sensitivity in murine models [14]. These findings underscore the potential for targeting

FABP4 as a therapeutic strategy for managing diabetes; however, more research is needed to translate these results into effective human treatments. The present study aimed to evaluate the concentration of ApoA, LCAT, and FABP4 in patients with uncontrolled diabetes.

Materials and Methods.

Subjects: This study included 184 individuals (128 patients and 56 controls) aged between 40-80 years, (85 males 46%, 99 females 54%) and they were divided into two groups of T2DM patients, according to their HbA1c, which indicates the control of diabetes by patients compared with apparently healthy controls. Group 1 (G1) (70 cases, 31 males, 39 females) included patients whose HbA1c value was more than 8% (disease uncontrolled) and who were taking either metformin. The second group (G2) included (58 patients, 30 males and 28 females) with HbA1c levels less than 7% (well controlled with metformin. The third group included healthy controls (G3) (56 people, 25 males, 31 females) with HbA1C ≤ 5%. All of them were patients reviewed in a Kirkuk Teaching Hospital, Azadi Teaching Hospital, and private clinics in the City of Kirkuk / Iraq during the period from November 2023 until May 2024, and they were diagnosed under the supervision of specialists. Patients suffering from liver, kidney, and pancreas diseases and those taking lipid-lowering medications were excluded.

Samples Collecting and Storing: After an overnight fasting, without having any treatments for lowering of sugar or lipids a venous blood samples were collected aseptically from the subjects via venipuncture. (6 ml) was collected and divided into two parts, (5ml) was kept in a plain tube without any anticoagulant at room temperature for 30 minutes. The tube was centrifuged (3000×g) for 10 minutes, the clear fresh serum was used in the estimation of lipid profile and FBG, and the remained quantity was pipetted into clear dry Eppendorf tubes and stored at (-20) C◦ until being used for different investigations (LCAT, ApoA-1, FABP4, and Insulin) while 1 ml of the whole blood kept in a tube with anticoagulant (EDTA) and used for determination of HbA1c.

Estimation the Activity of LCAT, the Concentration of Apo-A1, FABP4, And Insulin: The blood levels of LCAT, Apo-A1, FABP4, and insulin were measured by enzyme-linked immunoassay (ELISA) technique using a microplate that was pre-coated with an antibody specific to each of LCAT, Apo-A1, FABP4, and insulin. Standards or samples for each test were introduced into the corresponding wells of the microplate together with a biotin-conjugated antibody that specifically targets LCAT, Apo-A1, FABP4, and insulin. The Avidin-Horseradish Peroxidase (HRP) conjugate was then put into each well of the microplate and incubated. After adding the TMB substrate solution, only the wells containing LCAT, Apo-A1, FABP4, insulin biotin-conjugated antibody, and enzyme-conjugated Avidin showed a detectable change in color. To complete the enzyme-substrate reaction, a sulphuric acid solution was added, and the color change was measured using a spectrophotometer at 450 nm ± 10 nm. The concentrations of LCAT, Apo-A1, FABP4, and insulin in the samples are then determined by comparing their optical density (O.D.) to the standard curve.

Determination of FSG, Cho, TG, and HDL-Cholesterol: The fasting serum glucose (FSG), cholesterol (Cho), triglyceride (TG), and HDL-cholesterol, levels were determined by spectrophotometer instrument using a reagent kit manufactured by BIOLABO, a company based in France.

Evaluation of LDL: The serum concentration of low-density lipoprotein cholesterol (LDL) was determined using an indirect method. The study evaluated the levels of cholesterol, TG, and HDL cholesterol. LDL cholesterol was calculated using the Friedewald equation based on the starting data. The recommended amount is less than 130 milligrams per deciliter. to calculate LDL cholesterol and VLDL, use the following formulas:

LDL (mg/dl) = Total cholesterol-(HDL+TG/5)

VLDL (mg/dl) = TG/5

Determination of HbA1c (AFIAS HbA1c Neo): The test employed a sandwich immunodetection technique. The antibodies in the buffer detected the presence of antigens in the sample and bound to it creating antigen-antibody complexes. These complexes then moved onto a nitrocellulose matrix and were collected by immobilized streptavidin on a test strip. A higher concentration of antigens in the sample resulted in the formation of a greater number of antigen-antibody complexes. This, in turn, led to a more intense fluorescence signal produced by the detector antibodies. The apparatus used for AFIAS testing processes the signal to determine the percentage of glycated hemoglobin about the total hemoglobin present in the sample.

Statistical Analysis: All data were statistically analyzed using SPSS version 26 for Windows programs. The differences between groups were statistically examined using one-way analysis of variance and the ANOVA test. Descriptive statistics were given as mean ± SD values. Differences were judged significant at $p < 0.05$.

Results.

The current study's findings, as detailed in Tables 1 and 2, reveal a significant decrease in LCAT activity among patients with both uncontrolled (DM G1) and controlled diabetes mellitus (DM G2), compared to those without type 2 diabetes (G3). This reduction can be primarily attributed to hyperglycemia-induced glycation of apoA-1, a key apolipoprotein presents abundantly in high-density lipoproteins (HDL) which is essential for the activation of LCAT.

Table 1. *Levels of LCAT, insulin, FSG and HbA1c in the studied groups.*

Variables	T2DM (G1+G2,n=128)	controls (G3, n=56)	P value
LCAT (ng/ml)	74±15	116.47±6.85	0. 01
Insulin (pg/ml)	46.66±3.22	47.64±4.32	0.2
FSG (mg/dl)	183.25±4.96	99.12±13.71	0.001
HbA1c %	8.35±2.46	4.6±0.7	0.02
Data expressed as mean±SD, P<0.05 is considered as significant differences using Two sample t-test			

In the context of diabetes mellitus (DM), the study at hand reveals crucial insights into the dysregulation of lipid metabolism, highlighting significant elevations in FABP4, cholesterol, triglycerides (TG), low-density lipoprotein (LDL), and very-

low-density lipoprotein (VLDL) levels among DM patient groups [(G1) + (G2)] compared to healthy controls (G3). The statistically significant increase, indicated by a P value ≤ 0.05, underscores the altered lipid profile characteristic of diabetic pathology. In parallel with these findings is the observed decrease in high-density lipoprotein (HDL) and apolipoprotein A1 (Apo-A1), further substantiating the lipid imbalance pervasive in DM populations relative to group G3. Such alterations are consistent with known risk factors for cardiovascular diseases associated with diabetes. These results depart from previous research by Albahrani and Ali, who reported no significant impact of apoA1 polymorphism on lipoprotein phenotypes. This discrepancy might be attributed to differences in study design or genetic diversity among participants; however, it underscores the complexity of genetic interactions affecting lipid profiles in diabetic conditions. Table 3 and Table 4 encapsulate these findings vividly, providing empirical evidence that elaborates on how metabolic derangements influence lipid biomarker deviations in DM. Thus, this study advances our understanding of dyslipidemia within diabetic cohorts and emphasizes the need for personalized approaches to manage lipid abnormalities amid diverse genetic backgrounds.

Table 2. *Activity of LCAT and levels of insulin, FSG and HbA1c % in G1,G2 in the studied groups.*

Variables	G1 (n=70)	G2 (n=58)	G3 (n=56)	P value
LCAT (ng/dl)	62±9c	88.94±5.35b	116.47±6.85a	0.001
Insulin (pg/ml)	46.26±3.22a	47.14±3.17a	47.64±4.32a	0.21
FSG (mg/dl)	231.78±6.70a	124.67±2.31b	99.12±13.71c	0.001
HbA1c (%)	10.34±1.64a	6.21±0.33b	4.6±0.70c	0.01
Data expressed as mean±SD, Different letter indicate significant differences at P<0.05, while similar letter indicate non-significant differences at p>0.05 using One Way Anova with Tukeys post hok tests.				

Table 3. *Activity of LCAT and levels of insulin, FSG and HbA1c % in the studied groups.*

Variables	DM (G1+G2, N= 128)	Control (G3, N= 56)	P value
FABP4 (µg/ml)	26.4±10.66	14.85±2.3	0. 03
TC(mg/dl)	192.54±4.25	114.82±9.98	0.001
TG (mg/dl)	212.26±2.84	115.19±9.62	0.001
LDL (mg/dl)	110.53±36	41.82±10.85	0.001
VLDL (mg/dl)	42.45±12.56	23.03±1.9.24	0.003
HDL (mg/dl)	39.56±6.89	49.96±1.8	0.002
Apo A1(ng/ml)	93.83±7.32	150.94±14.92	0.001
Data expressed as mean±SD, P<0.05 is considered as significant differences using Two sample t-test			

From a comprehensive list of variables studied, there was no significant correlation between LCAT activity and Insulin concentration (rho = 0.069, P = 0.798), ApoA1 (rho = 0.561, p = 0.010) and HDL (rho = 0. 692, p = 0.001) recorded significant positive correlation with LCAT activity, while there was a strong significant negative correlation between each of [FABP4 (rho = - 0.540, p = 0.014), FSG (rho = - 0.785, p = 0.000), HbA1c (rho = - 0.773, p= 0.001), TG (rho = - 0.809, p = 0.000), Cholesterol (rho = - 0.710, p= 0.000), LDL (rho = - 0.701, p= 0.001) and VLDL (rho = - 0.803, p= 0.000)] with LCAT activity (Table 5).

Table 4. *Levels of FABP4 (µg/L), ApoA1 (ng/ml) and lipid profile for G1,G2 and controls G3.*

Variables	G1 (n=70)	G2 (n=58)	G3 (n=56)	P value
FABP4 (µg/L)	35.42±4.87a	15.51±1.95b	14.85±2.36c	0.01
Cho (mg/dl)	227.81±18.08a	150.10±4.67b	114.82±9.98c	0.001
TG (mg/dl)	267.95±16.2a	144.01±4.89 b	115.19±9.62c	0.001
LDL (mg/dl)	140.0±18.66a	74.87±4.73 b	41.82±10.85c	0.003
HDL (mg/dl)	33.87±2.9c	46.43±2.7b	49.96±1.80a	0.01
V LDL (mg/dl)	53.59±3.24a	29.00±0.97 b	23.03±1.92c	0.005
ApoA1 (ng/ml)	61.37±6.22c	133.01±14.14b	150.94±14.92a	0.01
Data expressed as mean±SD, Different letter indicate significant differences at P<0.05, while similar letter indicate non-significant differences at p>0.05 using One Way Anova with Tukeys post hok tests.				

Table 5. *Correlation coefficient (rho) between LCAT and the studied variables in patients with uncontrolled DM (G1).*

Variables	Correlation coefficient (rho)	p
LCAT and Insulin	0.069	0.798
LCAT and ApoA1	0.561	0.010
LCAT and FABP4	- 0.540	0.014
LCAT and FSG	- 0.785	0.0001
LCAT and HbA1c	- 0.773	0.001
LCAT and TG	- 0.809	0.0001
LCAT and TC	- 0.710	0.0001
LCAT and HDL	0.692	0.001
LCAT and LDL	- 0.701	0.001
LCAT and VLDL	- 0.803	0.0001

Discussion.

According to the findings in the current study which were illustrated in Tables 1,2, patients in the DM G1 (uncontrolled DM) and DM G2 (controlled DM) groups had significantly lower levels of LCAT activity than those without type 2 diabetes G3 because hyperglycemia in patients with uncontrolled illness results in the glycation of apoA-1which is existing in large amounts in HDL and has an important function in the activation of LCAT. Deficiency of LCAT can also be attributed to the deficiency or absence of its catalytic activity, which catalyses the formation of cholesterol esters in lipoproteins and is encoded by the LCAT gene [15]. These findings are similar to the previous studies, which found that LCAT activity is lower in diabetics with insulin-dependent or non-insulin-dependent diabetes mellitus [16,17]. Previous studies have shown that people with type 2 diabetes had lower LCAT activity than those without the illness. This decline in LCAT activity is associated with higher HbA1c levels [18]. This study observed that the level of FSG (mg/dl) and HbA1c% which are used as important diagnostic parameters and for level was significantly increased (P value < 0.05) among DM groups (G1+ G2) when compared with apparently healthy controls (G3), when the body has too little insulin amount or if it can't use insulin properly (insulin

resistance) but in this study insulin concentration (pg/ml) recorded no significant differences (P > 0.05) in DM (G1+G2) patients when compared (G3), and there was no significant difference between the concentration of insulin in G2 and G3 these results are compatible with [19].

Dyslipidemia is one of the main complications of T2DM which causes of atherosclerotic plaque. This study investigated the relationship between LCAT, ApoA1, FABP4, and lipid profile in T2DM patients. The previous studies explored the relationship between the ApoA1 and Coronary artery disease (CAD) in T2D patients conducted a cross-sectional study to investigate the association between the ApoA1 and diabetes. It was shown that increasing the ApoA1 was related with an increased risk of diabetes in both genders [20]. The levels of the FABP4, cholesterol, TG, LDL, and VLDL in the current study showed a significant increase (P value ≤ 0.05) in (mean ± SD) in DM patient's groups [(G1) + (G2)] in comparison to their apparently healthy controls (G3). The current study observed that the level of HDL (mg/dl) and Apo-A1 was decreased significantly (P value ≤ 0.05) compared with group (G3) as showed in Table 3, Table 4. The current study differed from Albahrani and Ali, who found no significant link or impact of the apoA1 polymorphism on lipoprotein phenotypes. T2DM is accompanied with dyslipidemia, which is defined by high triglycerides and low HDL cholesterol, and it is tempting to believe that these complicating circumstances may overshadow the role of apoA1 [21]. Rahnemaei et al. found that diabetics had higher levels of total cholesterol (TC) and triglycerides (TG) than healthy controls. The DM (G1,G2) groups demonstrated the most significant difference in lipid profiles compared to the control group (G3), particularly in terms of elevated blood TG levels. It may be concluded that increased serum TG had the largest influence on DM. The DM group showed greater levels of TC, LDL, VLDL, and FABP4, but lower levels of ApoA1c and HDL. As a consequence, these indications might be utilized to accurately identify diabetes [22]. Suryawanshi et al. found that uncontrolled diabetic patients in group G1 had higher levels of serum total cholesterol, triglycerides, and LDL cholesterol (p < 0.05). Furthermore, these persons exhibited lower HDL cholesterol levels than non-diabetics [23]. Merkhan et al. discovered no statistically significant differences in the levels of total cholesterol (TC), high density lipoprotein (HDL-c), and low-density lipoprotein (LDL-c) between the uncontrolled and controlled groups [24].

Visceral adiposity causes insulin resistance and obesity, increasing the risk of hypertension, type 2 diabetes, dyslipidemia, and heart disease [25]. Ghanei et al. study is also consistent with our prior findings, which demonstrated that LCAT activity was lower in patients with type 2 diabetes than those without type 2 diabetes and inversely related to HbA1c% [26]. This study produced different results from several previous studies that found elevated levels of Apolipoproteins in diabetics. It has been observed that those with type 2 diabetes had atherogenic lipid profiles, including increased ApoB and TG [27,28]. Abnormal lipid metabolism, including raised TC serum levels, TGs, LDL-c, and (apo) B Low HDL-c and ApoAI levels have been identified as major risk factors for the development of atherosclerosis (CAD) [29]. To the best of our knowledge, this was the first research to investigate the role of APOA1 in T2DM with and without control for early diagnosis of cardiovascular hazards. In the current study, we found that fatty acid binding protein-4 (FABP-4) was substantially greater in T2DM patients than in nondiabetic controls (P-Value ≤ 0.05). In this investigation, we revealed that diabetic individuals with CVD have significantly higher FABP4 levels, even after correcting for potential confounding variables (relative to uncontrolled diabetes patients). A sensitivity study was carried out to determine the robustness of this conclusion, which remained valid. Furthermore, correlation studies have shown that as diabetic patients' CVD worsens hemodynamically, circulating FABP4 levels rise. These findings imply that FABP4 levels rise as the severity of CVD in diabetic individuals increases. As a result, our findings point to an independent link between FABP4 and CVD in diabetics [30]. Only a few research have previously looked into the link between FABP4 and CVD. For example, Hoebaus et al. reported a substantial link between elevated FABP4 levels and future cardiovascular events [31]. The more uncontrolled diabetes [32], the more deleterious impact and presence of diabetes increase with infection further aggravate the disease [33].

Conclusion.

The results of the current study may support a significant positive relationship between low levels of LCAT and uncontrolled Type-2 DM. ApoA1 and HDL recorded significant positive correlation with LCAT activity, while There was a strong significant negative correlation between each of FABP4, FSG, HbA1c, TG, Cholesterol, LDL, and VLDL with LCAT activity.

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