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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 ინგ-ლისურენოვან 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. ექსპერიმენ-ტული 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, შემა-ჯამებელი 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. მიკროფოტო-სურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სუ-რათის 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 უცხო-ური 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 ხელმძღვანე-ლის 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 შე-ჯერება 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.

Содержание:

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ASSESSMENT OF THE LIPID SPECTRUM IN GEORGIAN CHILDREN WITH TYPE 1 DIABETES MELLITUS

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Abstract.

Background: Children and adolescents with type 1 diabetes mellitus (T1DM) are at increased risk of future cardiovascular disease, and dyslipidemia is one of the major modifiable contributors to this risk. In recent years, atherogenic lipid indices have been considered more sensitive markers of cardiovascular risk than isolated lipid parameters.

Objective: To assess the lipid spectrum in Georgian children with T1DM and to determine the association between glycemic control and dyslipidemia.

Methods: This retrospective cross-sectional study included 230 children and adolescents aged 9-18 years (mean age 13.22 ± 2.82 years) with T1DM from the Megalab clinical cohort. Dyslipidemia was defined as one or more abnormal lipid or lipoprotein values according to pediatric criteria. The following parameters and indices were evaluated: total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglycerides (TG), non-HDL-C, TC/HDL-C, LDL-C/HDL-C, non-HDL-C/HDL-C, TG/HDL-C, atherogenic index of plasma [AIP = log10(TG/HDL-C)], and LDL-C minus HDL-C. Statistical analysis was performed using SPSS version 23.

Results: Participants with inadequate glycemic control (HbA1c ≥ 7%; n = 72) had significantly higher TC, LDL-C, TG, non-HDL-C, and all assessed atherogenic indices except HDL-C, compared with those with HbA1c < 7% (n = 158). In logistic regression analysis, age (B=0.12; p=0.028). OR = 1.12(95% CI: 1.01–1.24) and HbA1c (B=0.20; p=0.002), OR = 1.22(95% CI: 1.08–1.39) were independent predictors of dyslipidemia, while sex was not.

Conclusions: In children with T1DM, inadequate glycemic control (HbA1c ≥ 7%) is associated with a more unfavorable atherogenic lipid profile. Atherogenic lipid indices are clinically important in children with T1DM because they reflect the overall effect of atherogenic lipoproteins better than any single lipid parameter alone. HbA1c is an independent predictor of dyslipidemia in children with T1DM.

Key words. Children, type 1 diabetes mellitus, lipid spectrum, dyslipidemia.

Introduction.

Globally, in 2024, approximately 9.15 million people were living with clinically diagnosed type 1 diabetes mellitus (T1DM) [1], of whom 1.81 million (19.8%) were younger than 20 years. In patients with T1DM, the risk of cardiovascular disease is substantially higher than in non-diabetic peers [2]. Children with T1DM often face age-specific challenges in disease management, which increase the relative risk of metabolic complications and adverse health outcomes [3].

In patients with diabetes, the pathophysiology of cardiovascular disease is multifactorial, and dyslipidemia plays a central role [4].

In children with T1DM, dyslipidemia is a frequent complication and is closely related to the quality of glycemic control. Studies have shown that children and adolescents with poor metabolic control more often exhibit hypertriglyceridemia, elevated LDL-C, and reduced HDL-C [5-7]. These abnormalities contribute to an increased risk of early atherosclerosis and subsequent cardiovascular disease from a young age [8-10].

In recent years, special attention has been directed toward atherogenic lipid indices, which are considered more sensitive markers of cardiovascular risk than isolated components of the lipid profile [4].

Indices such as TC/HDL-C, LDL-C/HDL-C, non-HDL-C/HDL-C, and TG/HDL-C reflect the balance between atherogenic and antiatherogenic lipoprotein fractions and may serve as important indicators of early vascular injury in children with T1DM [11].

Aim of the Study.

The aim of this study was to assess the lipid spectrum in Georgian children with T1DM and to determine the association between glycemic control and dyslipidemia.

Materials and Methods.

This retrospective cross-sectional study was based on biochemical data from 230 children and adolescents with T1DM aged 9-18 years (mean age 13.22 ± 2.82 years) from the Megalab clinical cohort.

A cross-sectional, analytical study was conducted. Inclusion criteria: children and adolescents with a confirmed diagnosis of type 1 diabetes mellitus, informed consent of the parent/guardian. Exclusion criteria: other metabolic or endocrine diseases, taking medications that affect the lipid profile, incomplete data.

Glycemic control was assessed by glycosylated hemoglobin (HbA1C) [12].

Biochemical assessment was performed after a 12-hour overnight fast. Serum total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), fasting glucose, and glycated hemoglobin (HbA1c) were analyzed on the Atellica® CH Analyzer (Siemens Healthcare Diagnostics Inc., USA) using manufacturer-recommended methods. LDL-C was measured by a direct homogeneous assay without off-line pretreatment or centrifugation. The assay uses a two-reagent format in which non-LDL lipoproteins are selectively solubilized first, whereas the remaining LDL particles are subsequently solubilized and quantified by an enzymatic colorimetric reaction measured at 545/694 nm. HDL-C was determined by a direct enzymatic assay based on selective masking of non-HDL lipoproteins followed by HDL-specific solubilization and color development. Triglycerides were measured by an enzymatic method involving lipoprotein lipase, glycerol kinase, and glycerol-3-phosphate oxidase, with endpoint detection at 505/694 nm. Total cholesterol was determined by an enzymatic colorimetric assay after hydrolysis by cholesterol esterase and oxidation by cholesterol oxidase, with endpoint measurement at 505/694 nm. Fasting glucose was measured using the hexokinase method with absorbance measured at 340/410 nm. HbA1c was measured by an enzymatic assay in anticoagulated venous whole blood, and results were reported in DCCT/NGSP units (%).

Dyslipidemia was defined as the presence of one or more abnormal lipid or lipoprotein values. According to the American Diabetes Association criteria used in the source manuscript, dyslipidemia was defined by LDL-C ≥ 100 mg/dL, HDL-C < 40 mg/dL in males and < 50 mg/dL in females, total cholesterol (TC) ≥ 200 mg/dL, and triglycerides (TG) ≥ 130 mg/dL [13], and was considered present if one or more of these parameters were abnormal [14,15].

The following lipid indices were evaluated: non-HDL-C, TG/HDL-C, atherogenic index of plasma (AIP) = log10(TG/HDL-C), HDL-C/LDL-C, Castelli index I (TC/HDL-C), Castelli index II (LDL-C/HDL-C), non-HDL-C/HDL-C, and LDL-C - HDL-C.

The study protocol was approved by the ethics committee of David Aghmashenebeli University of Georgia.

For quantitative variables, mean values and standard deviations were calculated; for categorical variables, frequencies and percentages were used. Differences between quantitative variables were assessed using Student's t-test for independent samples, with equality of variances evaluated using Levene's test. For categorical data, Fisher's exact test was used. Multivariable binary logistic regression analysis was performed to estimate the relative odds of dyslipidemia. A p value < 0.05 was considered statistically significant. Statistical analysis was conducted using SPSS version 23.

Results.

The age distribution of the participants is shown in Figure 1. The mean age was 13.22 ± 2.82 years, and the highest frequency was observed at 15 years of age (15.2%).

The baseline characteristics of children with T1DM are presented in Table 1.

The proportion of girls was 112 (48.7%), indicating an approximately balanced sex distribution. The mean HbA1c was 7.13±2.7%, with a minimum of 5.0% and a maximum of 13.6%. According to international recommendations for pediatric diabetes management, the target HbA1c is < 7% [16]. These findings suggest that average glycemic control in the study population was broadly acceptable, although the maximum value and the relatively large standard deviation indicate marked variability and poor control in some patients.

The mean total cholesterol was 175.92 ± 37.08 mg/dL, which is close to the borderline range, whereas the maximum value of 317.29 mg/dL indicates hypercholesterolemia in some patients. The mean LDL-C was 106.99 ± 29.54 mg/dL, near the upper limit of normal, suggesting a tendency toward increased atherogenic lipoproteins. The mean HDL-C was 45.07 ± 12.31 mg/dL, at the lower end of the desirable range, which may indicate a partial reduction in antiatherogenic protection. The mean triglyceride level was 119.28 ± 67.52 mg/dL, also within a borderline range, while the maximum value of 372.60 mg/dL suggests severe hypertriglyceridemia in a subset of patients. Overall, the atherogenic indices indicate a potential increase in cardiovascular risk.

We compared lipid characteristics according to glycemic control: good glycemic control (HbA1c < 7%; Group I, n = 158)

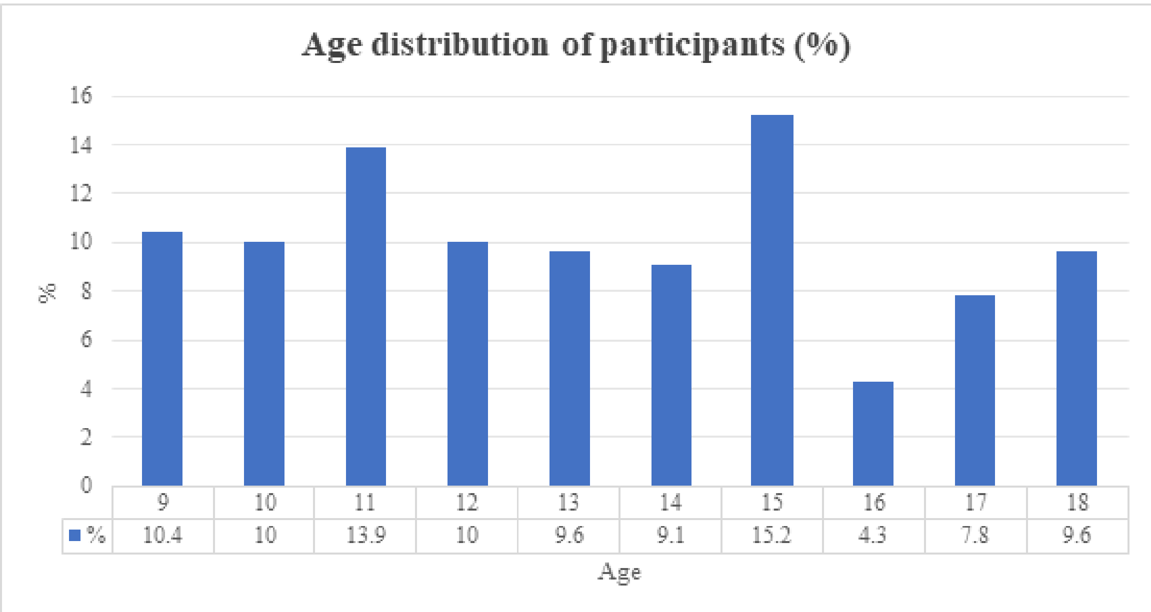

Figure 1. *Age distribution of participants (%).*

Table 1. Baseline characteristics of children with T1DM.

Variable	Minimum	Maximum	Mean	SD
Age, years	9.00	18.00	13.22	2.82
HbA1c, %	5.00	13.6	7.13	2.79
TC, mg/dL	100.12	317.29	175.92	37.08
LDL-C, mg/dL	47.51	201.17	106.99	29.54
HDL-C, mg/dL	23.54	85.07	45.07	12.31
TG, mg/dL	34.34	372.60	119.28	67.52
Non-HDL-C, mg/dL	58.75	270.49	130.84	36.23
TC/HDL-C	2.22	8.96	4.14	1.27
LDL-C/HDL-C	1.02	6.27	2.55	0.99
Non-HDL-C/HDL-C	1.22	7.96	3.14	1.27
TG/HDL-C	1.00	13.46	2.95	2.09
log10(TG/HDL-C)	0.00	1.13	0.38	0.27
LDL-C - HDL-C, mg/dL	1.15	156.81	61.92	32.19

Table 2. Comparison of lipid characteristics according to glycemic control.

Variable	Group I (HbA1c < 7%; n=158)		Group II (HbA1c ≥ 7%; n=72)		t	p
	Mean	SD	Mean	SD		
TC, mg/dL	169.06	31.97	190.68	43.16	-4.24	<0.001
LDL-C, mg/dL	103.08	27.97	115.56	31.23	-3.02	0.003
HDL-C, mg/dL	45.59	12.38	43.66	12.56	1.09	0.275
TG, mg/dL	101.95	44.59	157.30	90.41	-6.22	<0.001
Non-HDL-C, mg/dL	123.47	31.24	147.02	41.11	-4.33	<0.001
TC/HDL-C	3.93	1.16	4.65	1.46	-3.99	<0.001
LDL-C/HDL-C	2.44	0.97	2.84	1.10	-2.65	0.009
Non-HDL-C/HDL-C	2.93	1.16	3.65	1.46	-3.99	<0.001
TG/HDL-C	2.47	1.44	4.04	2.80	-5.62	<0.001
log10(TG/HDL-C)	0.33	0.23	0.50	0.31	-4.69	<0.001
LDL-C - HDL-C, mg/dL	57.49	31.46	71.90	31.94	-3.21	0.002

When comparing age between groups divided by HbA1c, a statistically significant difference was revealed: Group I (HbA1c <7%): 12.96 ± 2.77 years, Group II (HbA1c ≥7%): 13.81 ± 2.87 years, (t = -2.11; p=0.037).

Table 3. Distribution of age and HbA1c according to dyslipidemia status.

Factor	Without dyslipidemia (N=85)		With dyslipidemia (N=145)		t	p
	Mean	SD	Mean	SD		
HbA1c, %	6.23	2.05	7.66	3.03	-3.86	<0.001
Age	12.53	2.60	13.63	2.87	-2.97	0.003

Table 4. Logistic regression analysis of dyslipidemia risk according to age, sex, and HbA1c.

	B	S.E.	Wald	p.	Exp(B)	95% C.I for OR	
HbA1c	0.20	0.07	9.74	0.002	1.22	1.08	1.39
Age	0.12	0.05	4.80	0.028	1.12	1.01	1.24
Female	0.18	0.29	0.38	0.537	1.20	0.68	2.11
Constant	-2.43	0.77	9.86	0.002	0.09		

and inadequate glycemic control (HbA1c ≥ 7%; Group II, n = 72) (Table 2).

Analysis of the lipid profile showed that several atherogenic parameters were significantly higher in children with inadequate glycemic control. Specifically, total cholesterol was higher in Group II than in Group I (190.68 ± 43.16 mg/dL vs 169.06 ± 31.97 mg/dL; t = -4.24; p < 0.001). LDL-C was also higher (115.56 ± 31.23 mg/dL vs 103.08 ± 27.97 mg/dL; t = -3.02; p = 0.003), and non-HDL-C was markedly elevated in the poor-control group (147.02 ± 41.11 mg/dL vs 123.47 ± 31.24 mg/dL; t = -4.33; p < 0.001), indicating a more pronounced atherogenic lipid profile.

HDL-C did not differ significantly between the groups (45.59 ± 12.38 mg/dL vs 43.66 ± 12.56 mg/dL; t = 1.09; p = 0.275), suggesting that, in this sample, glycemic control did not substantially affect HDL-C concentration.

Triglycerides were significantly higher in Group II than in Group I (157.30 ± 90.41 mg/dL vs 101.95 ± 44.59 mg/dL; t = -6.22; p < 0.001).

Assessment of atherogenic indices showed that all evaluated indices were significantly higher in Group II. Specifically, TC/HDL-C was 4.65 ± 1.46 versus 3.93 ± 1.16 (p < 0.001), LDL-C/HDL-C was 2.84 ± 1.10 versus 2.44 ± 0.97 (p = 0.009), non-HDL-C/HDL-C was 3.65 ± 1.46 versus 2.93 ± 1.16 (p < 0.001), TG/HDL-C was 4.04 ± 2.80 versus 2.47 ± 1.44 ($p < 0.001$), log10(TG/HDL-C) was 0.50 ± 0.31 versus 0.33 ± 0.23 ($p < 0.001$), and LDL-C - HDL-C was 71.90 ± 31.94 versus 57.49 ± 31.46 ($p = 0.002$).

These findings indicate that inadequate glycemic control in children with T1DM is associated with a more unfavorable lipid profile and higher atherogenic indices, which may be linked to an increased risk of future cardiovascular complications.

In the next stage of the analysis, participants were divided according to the presence of dyslipidemia: Group I without dyslipidemia and Group II with dyslipidemia (Table 3).

In the dyslipidemia group, both age and HbA1c were significantly higher than in the group without dyslipidemia.

Using logistic regression, we assessed the risk of dyslipidemia according to age, sex, and HbA1c (Table 4).

Female sex was not a statistically significant factor ($p = 0.537$), indicating that sex was not independently associated with dyslipidemia in this cohort.

Age was a statistically significant factor (B=0.12; p=0.028), OR = 1.12(95% CI: 1.01–1.24). Each additional year of age was associated with an approximately 12% increase in the odds of dyslipidemia.

HbA1c was also statistically significant (B=0.20; p=0.002), OR = 1.22(95% CI: 1.08–1.39), indicating that higher HbA1c was associated with a 22% increase in the odds of dyslipidemia.

Discussion.

In the study by Selvaraj et al. [15], the prevalence of dyslipidemia among children and adolescents with type 1 diabetes mellitus was 67.3%. In our cohort, dyslipidemia was present in 63.04% of participants (145/230).

Lipid reference values in childhood and adolescence may vary with age and pubertal development. Nevertheless, major clinical guidelines (American Diabetes Association) recommend standardized lipid cutoffs for screening and risk stratification in high-risk pediatric populations such as those with diabetes. Because Tanner stage data were unavailable, we could not stratify analyses by pubertal status, which may have introduced some misclassification when applying uniform thresholds across ages.

HbA1C remains the most important modifiable determinant of the occurrence of dyslipidemia [16]. The analysis of the obtained results suggests that, although some parameters remained within normal limits, children with T1DM showed a tendency toward the development of an atherogenic lipid profile [17].

Older age was associated with higher HbA1c levels and poorer metabolic control, a trend particularly evident during adolescence. During puberty, insulin resistance increases; growth hormone and sex steroids reduce insulin effectiveness, making glycemic control more difficult and HbA1c levels higher [18].

The poor glycemic control group had significantly higher total cholesterol, LDL-C, triglycerides, non-HDL-C, and nearly all atherogenic indices. This suggests that, in our study population, lipid abnormalities were more strongly related to the quality of metabolic control than to age differences. Similar associations have been reported in contemporary pediatric studies, where worse glycemic control is associated with a less favourable metabolic and cardiovascular profile [19].

In T1DM, chronic hyperglycemia and relative insulin deficiency promote lipolysis, excessive hepatic very-low-density lipoprotein production, and accumulation of triglyceride-rich lipoproteins; LDL metabolism is also altered, ultimately producing a more atherogenic lipid profile [20]. In our data, the increases in total cholesterol, LDL-C, and triglycerides in the HbA1c ≥ 7% group are consistent with these mechanisms and with the concept that LDL-C remains the principal modifiable lipid risk factor in T1DM, while improvement of glycemic control and lifestyle measures remains the cornerstone of management in children and adolescents [21].

In our study, HDL-C did not differ significantly between groups ($p = 0.275$). According to the literature, the quantitative HDL-C level in children and adolescents with T1DM does not always reflect actual vascular risk, because HDL may remain normal or even relatively high while its antiatherogenic functionality is impaired. Therefore, an unchanged HDL-C level does not exclude cardiovascular risk, especially when triglycerides, LDL-C, non-HDL-C, and atherogenic indices are elevated [22].

The increase in lipid parameters in the HbA1c ≥ 7% group indicates that such patients require not only optimization of glycemia but also targeted lipid monitoring and, when necessary, specific intervention [23].

Particularly noteworthy is the significant increase in non-HDL-C, TC/HDL-C, LDL-C/HDL-C, non-HDL-C/HDL-C, and TG/HDL-C in the HbA1c ≥ 7% group. This indicates that poor glycemic control is associated not only with higher isolated lipid values but also with the formation of a more globally atherogenic lipid environment. In American Diabetes Association documents, non-HDL-C is considered an important marker of atherosclerosis risk and persistent dyslipidemia in children and adolescents. In this context, the increase in non-HDL-C and the related ratios found in our study may be interpreted as markers of early cardiovascular risk [24].

Our findings support the view that HbA1c ≥ 7% in childhood may be regarded not only as a marker of hyperglycemia but also as an important marker of atherogenic dyslipidemia. Clinically, these data are consistent with recommendations that regular lipid screening should be performed in children with T1DM. Contemporary guidance recommends lipid assessment after glycemic stabilization at diagnosis or within the first months thereafter, followed by regular reassessment. When LDL-C and triglycerides exceed target thresholds, management should include improved glycemic control as well as dietary and physical activity interventions; in selected cases, statin therapy may need to be considered [25].

According to the literature, TC/HDL-C < 3.5 is considered a low cardiovascular risk marker, whereas values ≥ 4-5 are associated with an increased risk of atherogenic processes [26]. In our study, TC/HDL-C was 3.93 ± 1.16 in the HbA1c < 7% group and 4.65 ± 1.46 in the HbA1c ≥ 7% group ($p < 0.001$), indicating a higher cardiovascular risk in the poor-control group. Similarly, non-HDL-C/HDL-C was 2.93 ± 1.16 in the HbA1c < 7% group and 3.65 ± 1.46 in the HbA1c ≥ 7% group ($p < 0.001$). Higher values of this index have been associated with excess atherogenic lipoproteins, and the findings in the poor-control group suggest increased cardiovascular risk [27].

Elevated TG/HDL-C has been associated with insulin resistance, metabolic syndrome, and early signs of atherosclerosis [28]. In our cohort, TG/HDL-C was 2.47 ± 1.44 in the HbA1c < 7% group and 4.04 ± 2.80 in the HbA1c ≥ 7% group ($p < 0.001$), suggesting a significant increase in metabolic and cardiovascular risk in the latter group.

AIP values > 0.21 have been associated with increased cardiovascular risk [29]. In our study, AIP was 0.33 ± 0.23 in the HbA1c < 7% group and 0.50 ± 0.31 in the HbA1c ≥ 7% group ($p < 0.001$). Both groups had elevated mean values, but the poor-control group had a significantly more pronounced atherogenic profile.

Logistic regression analysis showed that age and HbA1c were independently associated with dyslipidemia. Each additional year of age was associated with a 12% increase in the odds of dyslipidemia. Female sex was not independently associated with dyslipidemia in this cohort; this finding is consistent with evidence that the usual female protective effect on cardiovascular risk is attenuated in type 1 diabetes mellitus [30].

The literature suggests that lipid parameters worsen with age in children with type 1 diabetes mellitus [6] and may also vary by sex [31]. Our data are consistent with the conclusion that age is independently associated with dyslipidemia in this population.

Because lipid parameters depend on diabetes control [32], our finding that higher HbA1c is independently associated with dyslipidemia is biologically plausible and clinically relevant.

Conclusion.

In children with T1DM, inadequate glycemic control (HbA1c ≥ 7%) is associated with a more unfavorable atherogenic lipid profile.

Atherogenic lipid indices are clinically important in children with T1DM because they reflect the overall effect of atherogenic lipoproteins better than any single lipid parameter alone.

HbA1c is independently associated with dyslipidemia in children with T1DM.

Study Limitations.

This was a retrospective cross-sectional study; therefore, causal relationships cannot be established with certainty.

Some potential confounding factors were not available, including dietary habits, body mass index and obesity status, physical activity level, and duration of diabetes, all of which may affect lipid metabolism. Because body mass index and obesity status could not be included in the multivariable analysis, residual confounding is possible and the magnitude of the observed associations may be over- or underestimated.

Conflict of Interest.

The authors declare no conflict of interest.

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