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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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A STUDY OF THE ASSESSMENT OF SERUM ADROPIN LEVEL AS A RISK FACTOR OF ISCHAEMIC HEART DISEASE IN TYPE 2 DIABETES MELLITUS CASES

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Abstract.

Background: Adropin is a peptide hormone that was first identified in 2008 and was first thought to have a significant role in the balance of fatty acids and glucose in peripheral tissues. We look at the relationship between adropin and diabetes individuals' ischemic heart disease.

Objective: The objective of the study is to evaluate the serum adropin level as a potential indicator of ischemic heart disease in people with type 2 diabetes mellitus.

The 90 participants in this case-control study were split into three groups: Group (I) consisted of 30 T2DM patients with ischemic heart disease Group (II) consisted of 30 T2DM patients without ischaemic heart disease Group (III) consisted of 30 healthy persons as the control group.

HbA1c, lipid profile (cholesterol, triglycerides, LDL-C, HDL), HOMA IR serum creatinine, AST, ALT, and serum adropin were also evaluated. Fasting plasma glucose, 2h postprandial plasma glucose, Carotid artery intimal thickness using ultrasound, and Carotid artery intimal thickness were also measured.

Results: Patients with diabetes who did not have ischemic heart disease had a statistically significant rise in serum Adropin hormone (p value 0.001), with values of (26.867 10.037) ng/L and (87.500 40.509) ng/L, respectively. Additionally, there was a bad correlation between serum adropin and CIMT and fasting insulin.

Conclusion: Assessment of serum adropin levels may serve as a risk indicator for the emergence of ischemic heart disease in people with type 2 diabetes mellitus.

Key words. Adropin, type 2 diabetes mellitus, and ischemic heart disease.

Introduction.

90–95% of all cases of diabetes are type 2 diabetes, often known as "noninsulin-dependent diabetes." People with peripheral insulin resistance and relative insulin insufficiency are included in this category. These patients may not require insulin therapy to survive, at least initially and typically for the rest of their lives [1].

Due to a rise in obesity, an increase in life expectancy, and better identification of the condition, diabetes mellitus, the most common endocrine disease in affluent nations, is predicted to almost double by 2030 [2].

It is commonly acknowledged that decreased endothelial nitric oxide synthase (eNOS) activity and increased generation of reactive oxygen species (ROS) occur in diabetes mellitus, which leads to decreased nitric oxide (NO) bioavailability and subsequent vascular changes [3].

There are several methods for DM to cause endothelial dysfunction. The so-called diabetic condition, in which changes to the artery wall result in the pathogenesis of arterial thrombus, is caused by increased oxidative stress, altered lipogenesis, decreased nitric oxide, and altered endothelial progenitor cells' (EPC) activity.

The thickening of the vascular wall is caused by a series of events that result from damage to the arterial wall (such as the development of atherosclerotic plaque), altering blood flow and enhancing platelet aggregation [4].

The most prevalent cardiovascular disease, CAD, poses a severe danger to people's health due to its high morbidity and death rates. The major significant causes of CAD are typically considered to be endothelial dysfunction, vascular inflammation, and lipid metabolism problem [5].

The heart, brain, liver, and coronary endothelial cells all express adropin, a recently discovered endogenous bioactive molecule. Adropin has anti-inflammatory properties, helps with insulin resistance, protects vascular endothelial cells, and regulates lipid metabolism. Adropin expression in the central nervous system raises the possibility that this adipokine functions as a neuropeptide [3]. Adropin may also play autocrine or paracrine activities in peripheral tissues [3]. Adropin may also enhance nitric oxide (NO) release, activate vascular endothelial growth factor receptor 2 (VEGFR2), and modify neovascularization in addition to having an endothelial protective effect [6].

Aim of the work.

Evaluation of the serum adropin level as a potential indicator of ischemic heart disease in people with type 2 diabetes mellitus.

Patients and Methods.

The 90 participants in this case control study, whose ages ranged from 35 to 65 and had a mean of 48.63 8.52 years, were chosen from the national heart institute, internal medicine, and endocrinology outpatient clinics of Ain Shams University Hospitals between August 2019 and June 2020 after each participant signed an informed consent form.

Three groups were formed from them: (I) Group: 30 T2DM patients with ischemic heart disease make up Group (I). 30 T2DM individuals in group (II) who do not have ischemic heart disease Group (III): 30 control patients in good health. Patients with type 2 diabetes, CAD, an old myocardial infarction (MI), and stable angina pectoris (SAP) were required to meet the inclusion criteria.

All participants underwent a full clinical examination, including anthropometric measurements of weight, height, BMI, waist circumference, and waist/hip ratio, measurement of

blood pressure, and carotid intima thickness. Exclusion criteria included subjects known to have thyroid gland problems, chronic diseases such as severe respiratory illnesses, liver or kidney organ dysfunction, malignancies, liver cell failure, and cases of recent myocardial infarction and unstable angina within 6 months of onset.

Laboratory and imaging studies: Fasting blood sugar, two-hour post-meal blood sugar, HbA1c, lipid profile (cholesterol, triglycerides, LDL-C, HDL), serum creatinine, AST, ALT, serum adropin by ELISA using kits (Biotechnology assay Human Adropin ELISA Kit, China), and carotid artery intimal media thickness using ultrasound are all examples of blood tests.

Results.

Table 1 show highly statistically significant difference between the studied groups as regard fasting glucose and insulin , post prandial glucose, glycated hemoglobin and insulin resistance with high levels in patient groups versus control group by ANOVA test.

There is non-significant difference between diabetics with and without IHD as regard FBS and glycated hemoglobin but there is highly statistically significant difference between diabetics with and without IHD as regarding post prandial glucose, fasting insulin and insulin resistance.

Table 2 statistically show significant difference between the studied groups as regard carotid intima media thickness with high levels in patient groups versus control group by ANOVA test.

There is non-significant difference between diabetics with and without IHD as regard carotid intima media thickness.

Table 3 statistically show highly significant difference between the studied groups as regard adropin level versus control group with high adropin levels in control group versus patient's groups by ANOVA test.

There is highly significant difference between diabetics with and without IHD as regard adropin hormone level with raised adropin levels within diabetics without IHD.

Table 4 show a significant negative correlation between adropin concentration and fasting insulin and carotid intima media thickness among diabetics without ischaemic heart disease.

Table 5 show the cutoff value of adropin (≤40) if less that means high risk, sensitivity, specificity and positive and negative predictive values of this peptide.

Discussion.

In line with Agnieszka et al., findings that the concentrations of adropin across all subgroups according to duration of diabetes were lower than that in the control group, we discovered in our study that adropin level was significantly decreased in T2DM patients compared with control subject [7].

Gao et al., further explained that by clever in vivo work that showed mice induced obesity by diet and exogenous adropin causes increase in insulin receptor substrate 1&2 (IRS1, IRS2), and AKT phosphorylation implying that adropin rises hepatic insulin sensitivity, they found that adropin suppresses glucose production in hepatocytes that is mediated by cAMP/PKA.

This signaling system is essential for planning hepatic glucose production [8].

This was in contrast to Ahmad et al.'s finding that T2DM patients' serum adropin levels were considerably greater than those of healthy controls [9].

Adropin level was highly statistically significant difference between group 1 (diabetic group with ischaemic heart disease)

Table 1. *Comparison between studied groups as regard fasting glucose and insulin and post prandial glucose.*

		Groups			ANOVA		TUKEY'S Test		
		Group 1 Diabetics with IHD	Group 2 Diabetics without IHD	Group 3 Control pool	F	P-value	Groups 1,2 P-value	Groups 1,3 P-value	Groups 2,3 P-value
FBG (mg/dl)	**Mean ±SD**	174.700 ± 78.830	199.233 ± 82.922	92.700 ± 7.892	21.298	**<0.001***	0.328	**<0.001***	**<0.001***
2 h P.P (mg/dl)	**Mean ±SD**	224.2 ± 75.84	281.13 ± 112.54	131.567 ± 14.524	27.534	**<0.001***	**0.017***	**<0.001***	**<0.001***
Fasting insulin	**Mean ±SD**	2.523 ± 0.785	3.625 ± 1.183	2.498 ± 0.855	13.556	**<0.001***	**<0.001***	0.994	**<0.001***
HbA1c	**Mean ±SD**	7.901 ± 1.522	8.259 ± 1.423	6.027 ± 0.249	29.3	**<0.001***	0.491	**<0.001***	**<0.001***
HOMA-IR	**Mean ±SD**	1.134 ± 0.741	1.701 ± 0.685	0.576 ± 0.209	26.852	**<0.001***	**0.001***	**0.001***	**<0.001***

P value statistically significant if <0.05

Table 2. *Comparison between studied groups as regard carotid intima media thickness.*

CIMT	Groups			ANOVA		TUKEY'S Test		
	Group 1 Diabetics with IHD	Group 2 Diabetics without IHD	Group 3 Control pool	F	P-value	Groups 1,2 P-value	Groups 1,3 P-value	Groups 2,3 P-value
Mean ±SD	1.033 ± 0.092	1.027 ± 0.105	0.900 ± 0.207	8.160	**0.001***	0.982	**0.002***	**0.003***

P value statistically significant if <0.05

Table 3. *Comparison between studied groups as regard adropin hormone level.*

Adropin	Groups			ANOVA		TUKEY'S Test		
	Group 1 Diabetics with IHD	Group 2 Diabetics without IHD	Group 3 Control pool	F	P-value	Groups 1,2 P-value	Groups 1,3 P-value	Groups 2,3 P-value
Mean ±SD	26.867 ± 10.037	87.500 ± 40.509	162.500 ± 97.040	37.233	**<0.001***	**0.001***	**<0.001***	**<0.001***

P value statistically significant if <0.05

Table 4. *Correlations between patients groups as regard adropin level.*

Correlations				
	Adropin			
	Diabetics with IHD		Diabetics without IHD	
	r	P-value	r	P-value
Age (Years)	-0.053	0.781	-0.326	0.079
BMI	-0.142	0.453	-0.006	0.976
Hb A1c	0.148	0.436	0.103	0.590
HOMA-IR	0.122	0.521	-0.216	0.251
CIMT	-0.088	0.643	-0.361	**0.050***
SBP (mmHg)	-0.069	0.717	0.186	0.324
DBP (mmHg)	0.297	0.111	0.189	0.318
Height (cm)	0.302	0.105	-0.132	0.488
Weight (kg)	0.015	0.938	-0.045	0.812
FBG (mg/dl)	0.033	0.862	-0.033	0.861
2 h P.P (mg/dl)	0.195	0.302	0.266	0.155
Fasting insulin	0.162	0.393	-0.374	**0.041***
Creatinine (mg/dl)	0.027	0.887	0.114	0.548
Urea	0.342	0.064	0.066	0.727
Chol	0.025	0.895	0.048	0.799
TG	-0.036	0.851	-0.017	0.929
LDL	0.108	0.570	0.082	0.665
HDL	-0.208	0.271	-0.130	0.493
AST	-0.260	0.165	-0.168	0.374
ALT	-0.265	0.157	0.070	0.715
CK-T	0.001	0.997	0.177	0.350
CK-MB	0.480	0.107	-0.004	0.983
HB	0.067	0.724	-0.233	0.215
Troponin	-0.333	0.072	-0.025	0.895
D.M Duration	-0.111	0.558	-0.338	0.068

P value statistically significant if <0.05

Table 5. *Validity of adropin among diabetics with IHD and control.*

ROC curve between Diabetics with IHD and Control						
	Cutoff	Sensitivity	Specificity	PPV	NPV	Accuracy
Adropin	≤40	96.67	96.67	96.7	96.7	99.6%

and group 2 (diabetics without ischaemic heart disease), with low adropin level among ischaemic heart patients, and group 1 (diabetic group with ischaemic heart disease) and group 3 (healthy control group) with higher adropin level.

In line with Mu et al., finding that the level of serum adropin in the CAD group was lower than that in healthy controls [10] and Yu et al., discovery that serum adropin levels in CAD patients were lower than those of healthy controls, the adropin level was highly statistically significant different between group 2 (diabetic group without ischaemic heart disease) and group 3 (healthy control group) [11]. According to Wu et al., persons with type 2 diabetes and atherosclerosis have lower blood levels of adropin than people without these conditions [12].

Additionally, in line with Kume et al., result that adropin levels were shown to be significantly negatively linked with insulin, HOMA-IR, and atherogenic lipid profiles, we identified a very significant negative association between fasting insulin and adropin of T2DM patients in our study [13].

In line with the findings of Gao et al., who observed that adropin considerably lowered fasting blood glucose, HbA1c (%), HOMA-IR, and insulin levels, we discovered that adropin was extremely significantly negatively linked with insulin

concentrations in our investigation. Adropin may directly impede the synthesis of the glucose metabolism.

Adropin therapy improved glucose tolerance, insulin action, and metabolic flexibility toward glucose uptake in his tests on diet-induced obese mice with insulin resistance (8).

In this investigation, we discovered that serum adropin and CIMT among T2DM without IHD had a strong negative connection. This finding is consistent with Pereira et al. (2017), who discovered that CIMT was considerably greater in T2DM patients than in control subjects (14). According to Yosaee et al., CIMT was also inversely correlated with serum adropin, suggesting that low serum adropin may be utilised as a standalone predictor for subclinical atherosclerosis (2016). (15).

Conclusion.

A decline in serum adropin levels may be a sign of developing ischemic heart disease in people with type 2 diabetes.

List of Abbreviations.

ADA	:	American diabetes association
IHD	:	Ischaemic heart disease.
BMI	:	Body mass index
CAD	:	Coronary artery disease
CIMT	:	Carotid intima-media thickness
eNOS	:	Endothelial nitric oxide synthase
ERK1/2	:	Extracellular signal-regulated kinases 1/2
EPC	:	endothelial progenitor cells
HDL	:	High density lipoprotein
HOMA-IR	:	The homeostatic model assessment of insulin resistance
IFG	:	Impaired fasting glucose
IGT	:	Impaired glucose tolerance
IRS1, IRS2	:	Insulin receptor substrate 1&2
NO	:	Nitric oxide.
PI3K	:	Phosphoinoside-3 kinase
PKC	:	Protein kinase-c
ROS	:	Reactive oxygen species
Hb A1c	:	Glycated haemoglobin
SBP (mmHg)	:	Systolic blood pressure
DBP (mmHg)	:	Diastolic blood pressure
FBG (mg/dl)	:	Fasting blood glucose
2 h P.P (mg/dl)	:	2 hours post prandial
Chol	:	Cholesterol
TG	:	Triglycerides
LDL	:	Low density lipoprotein
AST	:	Aspartate transaminase
ALT	:	Alanine transaminase
CK-T	:	Creatine kinase total
CK-MB	:	Creatine kinase MB
HB	:	Haemoglobin

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