GEORGIAN MEDICAL NEWS

ISSN 1512-0112

NO 11 (344) ноябрь 2023

ТБИЛИСИ - NEW YORK

ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

Monthly Georgia-US joint scientific journal published both in electronic and paper formats of the Agency of Medical Information of the Georgian Association of Business Press. Published since 1994. Distributed in NIS, EU and USA.

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.

GEORGIAN MEDICAL NEWS
No 11 (344) 2023

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GEORGIAN MEDICAL NEWS
No 11 (344) 2023

ESTIMATION OF VON WILLEBRAND FACTOR IN PATIENTS CARDIAC DISEASES

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Abstract.

Background: Ischemic heart disease, also called coronary artery disease (CAD), is the term given to heart problems caused by narrowed heart (coronary) arteries that supply blood to the heart muscle.

Aim of the study: The study aims to determine the levels of Von Willebrand factor in patients with coronary artery disease.

Patients and Methods: A total of 60 CAD patients and 30 healthy individuals were enrolled in the present study. Blood was withdrawn and serum was separated for measurement of lipid profile, human von Willebrand Factor, and lactate dehydrogenase (LDH).

Results: The study demonstrated that the highest mean of VWF was observed among IHD patients (81.75±29.75 ng/ml) and the lowest mean was within the control group (26.53±5.12 ng/ml). The differences were highly significant (P-value: 0.0001). The study showed that the highest means of cholesterol, triglyceride, and LDL and the lowest mean of HDL were detected in IHD patients as compared with the healthy control group. The study showed that the mean of VWF was (88.76 ng/ml) in patients with hypertension which were significantly highest than in non-hypertensive IHD patients (75.76 ng/ml) at P. value: 0.043. The study showed that the highest means of cholesterol, Triglyceride and HDL were detected in hypertensive IHD patients (264.3, 349.1, and 29.24 mg/dl respectively) as compared with non-hypertensive IHD patients group (217.5, 226.1 and 22.02 mg/dl), respectively. The study showed a significant negative correlation between VWF and LDL among IHD patients and showed a positive correlation of VWF with each of cholesterol, triglyceride, and HDL among IHD patients.

Conclusions: In conclusion, the findings of the study suggest that patients with ischemic heart disease (IHD) have elevated levels of the von Willebrand factor. More research is needed to fully understand the relationship between VWF and cardiovascular disease and to determine the clinical utility of its measuring in the diagnosis and management of these conditions.

Key words. Von Willebrand factor, ischemic heart disease, hyperlipidemia, hypertension.

Introduction.

Ischemia is defined as inadequate blood supply (circulation) to a local area due to blockage of the blood vessels supplying the area. Ischemic means that an organ (e.g., the heart) is not getting enough blood and oxygen. Ischemic heart disease, also called coronary heart disease (CHD) or coronary artery disease, is the term given to heart problems caused by narrowed heart (coronary) arteries that supply blood to the heart muscle [1]. Coronary artery disease (CAD) is characterized by the occlusion or stenosis of the coronary artery mostly caused by atherosclerosis and is one of the leading causes of mortality in humans [2]. Patients with CAD are vulnerable to the development of major cardiovascular events including nonfatal acute myocardial infarction, unstable angina, stroke, transient ischemic attack, peripheral arterial occlusive disorder, and death [3]. Although the narrowing can be caused by a blood clot or by constriction of the blood vessel, most often it is caused by buildup of plaque, called atherosclerosis. When the blood flow to the heart muscle is completely blocked, the heart muscle cells die, which is termed a heart attack or myocardial infarction (MI) [4]. Most people with early CHD do not experience symptoms or limitations of blood flow. However, as the atherosclerosis progresses, especially if left untreated, symptoms may occur. They are most likely to occur during exercise or emotional stress when the demand for oxygen carried by the blood increases [5].

The von Willebrand (VWF) axis exerts a pivotal role in vascular inflammation and thrombosis. Thrombosis, with the recruitment of platelets to the site of the vessel's injury, and immune response, with the recruitment of leukocytes in inflamed tissues, have traditionally been considered two distinct pathways [6]. For >150 years, it has been known that alterations in blood flow, vascular wall, and blood components, the so-called Virchow's triad, may progressively lead to thrombus formation. Yet, a more complete understanding of the complex interactions among the vascular endothelium, platelet adhesion, activation, aggregation, and clotting factor activation involved in this process is still emerging from contemporary research [7,8]. Under physiological conditions, the vascular endothelium produces many substances that contribute importantly to hemostasis, fibrinolysis, and regulation of vessel tone and permeability. One such substance is the multimeric glycoprotein von Willebrand factor (VWF), which is produced almost exclusively by endothelial cells [9]. Plasma levels of VWF are raised in different states of endothelial damage and have therefore been proposed as useful markers of endothelial dysfunction. Along this line, the blockade of nitric oxide enhances the stimulated release of VWF in humans [10]. Furthermore, VWF plays a crucial role in platelet adhesion and aggregation under high-shear conditions. Additionally, VWF supports the third component of Virchow's triad, clotting factor activation, by acting as a carrier protein and stabilizer for factor VIII [11]. The study aims to determine the levels of Von Willebrand factor in patients with ischaemic heart disease (IHD) patients.

Materials and Methods.

The current study is a case-control study conducted in Tikrit City during the period between the 10th of January to the 10th of April 2023 on patients admitted to the Coronary Care Unit of Salah Al-Din Teaching Hospital

The study included 60 Iraqi patients with coronary heart disease in the age range (37-66 years). The diagnosis of ischemic heart disease was based on history and characteristic electrocardiographic changes. Hypertension, defined as a systolic blood pressure greater than 130 mmHg or a diastolic

blood pressure greater than 80 mmHg or are taking medication for hypertension. Medical history was taken for patients including the history of hypertension and/or diabetes mellitus in addition to drug history and smoking. The cases were collected from the Coronary Care Unit of Salah AlDin Teaching Hospital.

The study also included 30 healthy individuals with the same age range and from both sexes who haven't any acute or chronic diseases.

Exclusion Criteria: patients with chronic diseases, pregnant and lactating women, and those who are alcoholics were excluded from the study. After at least 12 hours of fasting, blood was collected by vein puncture with plastic disposable syringes took up to 5ml of venous blood from each healthy control and patient and added to the gel tube, which was then left at room temperature for 30 minutes to initiate the clotting process, the sample was then centrifuged to separate the serum at 3,000xg for 15 minutes and the obtained serum were aspirate using a mechanical micropipette and transferred into Eppendorf tubes and stored at-20C for determination of cholesterol, triglyceride (TG), high-density lipoprotein- cholesterol (HDL-C) and low-density lipoprotein (LDL) by biochemical colourimetric methods and determination of human von Willebrand Factor by enzyme-linked immunosorbent assay (ELISA).

Results.

The study showed that the majority of IHD patients were 57-66 years old (68.33%), 20% were within the age group 47.56 years and 11.67% were within the age group 37-46 years, and the study showed that 66.67% of IHD patients were males (Table 1).

The study demonstrated that the highest mean of VWF was observed among IHD patients (81.75±29.75 ng/ml) and the lowest mean was within the control group (26.53±5.12 ng/ml). The differences were highly significant (P-value: 0.0001) (Table 2).

Table 1. *Distribution of IHD patients according to age and gender.*

Parameters	**IHD patients**	
Age groups (years)	No.	%
37-46	7	11.67
47-56	12	20
57-66	41	68.33
Total	60	100
Sex	No.	%
Males	40	66.67
Females	20	33.33%
Total	60	100

Table 2. *The mean level of a von-Willebrand factor in IHD patients and the control group.*

VWF (ng/ml)	**Mean**	**SD**	**Minimum**	**Median**	**Maximum**
IHD patients	81.75	29.79	47.31	81.04	150.03
Control group	26.53	5.12	15.74	26.27	36.60
P-value: 0.0001					

The table 3 presents a comparison between the lipid profile parameters of hemodialysis (IHD) patients and the control group. In terms of cholesterol, the IHD group had a significantly higher mean value of 245.6 mg/dl compared to the control group's mean value of 170.1 mg/dl. In terms of triglyceride levels, the IHD group had a significantly higher mean value of 299.8 mg/dl compared to the control group's mean value of 159.6 mg/dl. In terms of HDL levels, the IHD group had a significantly lower mean value of 26.35 mg/dl compared to the control group's mean value of 43.78 mg/dl. In terms of LDL levels, the IHD group had a significantly higher mean value of 126.5 mg/dl compared to the control group's mean value of 59.97 mg/dl. These findings suggest that hemodialysis patients have an unfavourable lipid profile compared to the control group.

The study showed that the mean of VWF was (88.76 ng/ml) in patients with hypertension which were significantly highest than in non-hypertensive IHD patients (75.76 ng/ml) at P. value: 0.043, (Table 4).

Table 3. *Comparison between IHD patients and the control group regarding lipid profile parameters.*

Lipid profile (mg/dl)	**Studied groups**	**Mean**	**P-value**
Cholesterol	IHD	245.6	0.001
	Control	170.1	
Triglyceride	IHD	299.8	0.001
	Control	159.6	
HDL	IHD	26.35	0.003
	Control	43.78	
LDL	IHD	126.5	0.001
	Control	59.97	

Table 4. *Comparison between hypertensive and non-hypertensive IHD patients regarding the level of VHF.*

Variable	**Hypertension**	**No.**	**Mean**	**SD**	**P-value**
VWF (ng/ml)	Absent	24	75.21	28.47	0.043
	Present	36	88.76	33.09	

Table 5. *Comparison between hypertensive and non-hypertensive IHD patients regarding lipid profile parameters.*

Variable	**Hypertension**	**No.**	**Mean**	**SD**	**P-value**
cholesterol	Absent	24	217.5	60.5	0.001
	Present	36	264.3	88.4	
TG	Absent	24	226.1	94.6	0.001
	Present	36	349.1	165.9	
HDL	Absent	24	22.02	3.628	0.003
	Present	36	29.24	8.46	
LDL	Absent	24	143.2	34.56	0.061
	Present	36	137.4	43.10	

Table 6. *Correlation of VWF with other parameters among IHD patients.*

Variable 1	**Variable 2**	**Correlation**	**P-Value**
VWF	LDL	-0.284	0.028
VWF	LDH	0.756	0.001
VWF	Cholesterol	0.715	0.001
VWF	Triglyceride	0.843	0.001
VWF	HDL	0.671	0.001

The study showed that the highest means of cholesterol, Triglyceride and HDL were detected in hypertensive IHD patients (264.3, 349.1, and 29.24 mg/dl respectively) as compared with non-hypertensive IHD patients' group (217.5, 226.1 and 22.02 mg/dl) respectively, while there was no

significant difference between the two groups regarding LDL levels (Table 5).

The study showed a significant negative correlation between VWF and LDL among IHD patients and showed a positive correlation of VWF with each of LDH, cholesterol, triglyceride, and HDL among IHD patients (Table 6).

Discussion.

The study found that the mean (average) level of VWF in IHD patients was significantly higher than in the control group. The highest mean level of VWF was observed among IHD patients, with a value of 81.75±29.75 ng/ml. This suggests that IHD patients have higher levels of VWF in their blood, which may contribute to an increased risk of blood clots and cardiovascular events. In contrast, the study found that the control group had the lowest mean level of VWF, with a value of 26.53±5.12 ng/ml. This is expected, as the control group was composed of individuals without any known cardiovascular diseases or risk factors, and therefore had lower levels of VWF in their blood.

The current finding that shows an elevated level of von Willebrand Factor (VWF) among patients with Ischemic Heart Disease (IHD) is consistent with the findings of previous studies. Al-Masri et al [1] also found elevated levels of VWF among different types of heart disease. Similarly, studies by other researchers, such as those cited in the current study [2,3], have reported elevated levels of VWF among IHD patients.

In addition, studies by Montalescot et al. [4] have reported an immediate rise in VWF levels in acute coronary syndrome (ACS), which is a type of IHD. This elevation in VWF was found to be associated with adverse outcomes of ACS. The elevation of VWF has also been observed in individuals with objectively proven atherosclerosis [5], which is a buildup of plaque in the arteries that can lead to IHD, and in patients experiencing sudden cardiac death, non-sudden cardiac death, and nonfatal MI [6]. These findings suggest that elevated VWF levels may be associated with a range of cardiovascular diseases and adverse outcomes. Furthermore, significant associations of VWF with ischemic stroke have also been reported in other studies [7]. This suggests that VWF may be a useful biomarker for assessing the risk of a range of cardiovascular events, including both heart disease and stroke. Overall, the findings of these studies suggest that VWF may play an important role in the pathogenesis of cardiovascular diseases and may be a useful tool for predicting and managing the risk of adverse cardiovascular events. The finding that IHD patients have significantly higher levels of cholesterol, triglycerides, and HDL compared to the healthy control group is consistent with several other studies that have shown a strong association between dyslipidemia and IHD.

For example, the Yusuf et al. [8] study, which involved over 29,000 participants from 52 countries, found that high levels of total cholesterol and LDL were strongly associated with acute myocardial infarction (AMI) Similarly, the Prospective Studies Collaboration involving more than 900,000 individuals also reported that higher levels of total cholesterol, LDL, and triglycerides were associated with an increased risk of coronary heart disease (CHD) [9].

Other studies have also demonstrated the effectiveness of lipid-lowering therapy, such as statins, in reducing the risk of IHD [10,11]. The use of statins is recommended in current guidelines for the management of dyslipidemia and the prevention of cardiovascular disease [12]. Overall, the finding that IHD patients have significantly higher levels of cholesterol, triglycerides, and LDL reinforces the importance of dyslipidemia management in reducing the risk of IHD and highlights the need for early identification and treatment of dyslipidemia in high-risk individuals [13].

The study suggests that patients with hypertension have significantly higher levels of VWF compared to healthy individuals. VWF is an important factor in the formation of blood clots, and elevated levels of VWF have been associated with an increased risk of cardiovascular disease, including hypertension [14].

This finding is supported by previous studies that have also reported higher levels of VWF in hypertensive individuals compared to healthy controls. For example, a study by Goonasekera [13] found that plasma VWF levels were significantly higher in hypertensive patients compared to normotensive controls.

The association between hyperlipidemia and hypertension has been shown in several previous studies [15-17]. An excessive dietary intake of saturated fats, cholesterol and other calorie sources and subsequent lipid profile disruption leading to hypertriglyceridemia and hypercholesterolemia are related to obesity and hypertension. In a study, Brown, 2000 confirmed the association between several factors including BMI, serum cholesterol, HDL, and hypertension [18]. Hypertension is considered to be associated with lipid metabolism alterations that give rise to serum lipid and lipoprotein levels abnormalities [19]. It has also been documented that the presence of hyperlipidaemia makes the prognosis in hypertensive patients significantly worse [20]. The higher mean levels of TC, TG, and LDL-C in hypertensive patients are in agreement with the results of other related studies which are conducted in different parts of the world including Ethiopia [21]. A rising trend was also observed for the prevalence of lipid abnormalities and serum levels of TG, TC, LDL-C, and decreasing serum levels of HDL-C with the severity of hypertension indicating that they are associated with hypertension. This negative correlation may reflect alterations in lipid metabolism in patients with IHD. Atherosclerosis is a condition that is caused by a combination of factors, including elevated LDL levels and inflammation. The negative correlation between VWF and LDL suggests that changes in VWF levels may be associated with changes in LDL levels in patients with IHD [22,23]. Overall, these findings suggest that changes in VWF and LDL levels may be useful markers for evaluating lipid metabolism and cardiovascular risk in patients with IHD. The current study revealed a positive correlation of von Willebrand factor (VWF) with cholesterol, triglyceride, and high-density lipoprotein (HDL) among patients with ischemic heart disease (IHD), it would suggest that as the levels of these lipids increase, VWF levels also positively correlate with cholesterol and triglyceride in a study done by Hameed and Ahmed [24] in patients with ischemic stroke in Erbil city. The exact mechanism responsible for the elevation of VWF in hyperlipidemia is not completely understood yet.

It has enormous clinical, social, and economic implications and demands a significant effort from both basic scientists and clinicians in the quest for understanding the underlying pathogenetic mechanisms, and thereby adopting suitable preventive measures and successful therapies. However, it is important to note that correlation does not necessarily imply causation [25,26]. Endothelial interaction [27,28] with the surrounding milieu alongside released trophic factors [29,30] is having a significant impact on the fate of fat caps and plaque formation further impacting the inflammatory process for coronary disease [31].

Conclusion.

In conclusion, the findings of the study suggest that patients with ischemic heart disease (IHD) have elevated levels of Von-Willebrand factor. More research is needed to fully understand the relationship between VWF and cardiovascular disease and to determine the clinical utility of its measuring in the diagnosis and management of these conditions.

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