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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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EVALUATION OF SERUM LEVELS OF INTERLEUKIN-6, FETUIN-A, LIPOCALIN-2, AND C-REACTIVE PROTEIN IN RHEUMATOID ARTHRITIS PATIENTS

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Abstract.

Background: Several biochemical factors have increasingly been reported as diagnostic tools associated with certain diseases. C-reactive protein (CRP) has been widely accepted as a systemic inflammatory marker, nonetheless, non-specificity remains innocent for diagnosis. Hence looking for alternative supportive more specific diagnostic markers was subjective for continuous research.

Aim: Identifying whether Interleukin-6, Fetuin-A, and Lipocalin-2 (LCN-2) could have a potential contribution to rheumatoid arthritis (RA) prognosis and diagnosis. **Methods:** RA patients were recruited, and equally matched control healthy individuals were assigned for comparison. Blood was withdrawn and serum was separated and stored at -80 for future analysis. Target parameters were measured including Interleukin-6, Fetuin-A, LCN-2, and CRP.

Results: The outcome has revealed potentially significant differences between measured parameters in RA patients compared to the control group, Fetuin, LCN-2, and CRP were significantly ($P<0.0$) elevated. **Conclusion:** The measured parameters could be considered as additional parameters for the diagnosis and prognosis of a patient with rheumatoid arthritis.

Key words. Interleukin-6, Fetuin-A, Lipocalin-2, C-reactive protein, Rheumatoid arthritis.

Introduction.

Rheumatoid arthritis (RA) is a progressive inflammatory ailment of illusive etiology with a systemic inflammatory response that affects 1% of the world's population [1,2]. Perhaps, RA characterized by chronic synovial inflammation targeting joints, usually several joints at the same time. The hand, wrist, and knee joints are usually affected by RA [3]. Joints that have RA have inflammation of the joint lining, which damage the joint tissue. This tissue damage can lead to instability (loss of balance), severe or persistent pain, and deformity (misalignment) [4]. It may also be associated with B-cell-induced antibody and T-cell immune responses. 2-Heremans Schmid glycoprotein is another name for fetuin-A (AHSG). The human AHSG gene, with a molecular weight of 60 kDa, is located on chromosome 3q27 and encodes a phosphorylated glycoprotein [5]. It is a plentiful plasma protein that is primarily produced by hepatocytes and discharged into serum, where it is highly concentrated. It has been demonstrated that fetuin-A serves as either a positive or negative acute-phase protein (APP) in pathogenic circumstances [6]. Fetuin-A is considered an inflammatory marker because it has been shown to promote the expression of inflammatory cytokines in adipocytes and macrophages as a positive acute-phase protein (APP) [7]. On the other hand, Fetuin-A glycoprotein has been found to have anti-inflammatory or protective effects in various disease states, including infection, sepsis, ischemic brain injury [8], endotoxemia, trauma, autoimmune diseases, and Alzheimer's disease [9].

Fetuin-A prevents the growth and precipitation of alkaline calcium phosphate and apatite precursor minerals (BCP). Without affecting bone mineralization, it prevents unwanted calcification in blood circulation [10]. The resistance mechanism of plasma proteins to apatite production is significantly reduced after the targeted depletion of fetuin-A in the serum. Numerous immune system cells, both innate and adaptive, create small peptides known as cytokines, and their main function is to regulate the development and behavior of immune effector cells [11].

A number of different cell types, including dendritic cells, macrophages, Kupffer cells, and endothelial cells, synthesize the pleiotropic cytokine interleukin-6 (IL-6) [12]. Due to its ability to stimulate macrophages, monocytes, and natural killer and, together with IL-12, induce Th1-type responses, activation of vascular smooth muscle, dendritic cells and Kupffer cells is directly implicated in innate and adaptive immune responses [13]. Thus, depending on the local cytokine environment, IL-6 can act as a cofactor in the formation of Th1 and Th2 cells [13].

Hydrophobic small molecules are transported by neutrophil gelatinase-associated lipocalin (LCN-2), a 25 kDa secreted glycoprotein initially found in active neutrophils [14]. During inflammation and injury, in addition to neutrophils, NGAL may be released by renal tubular cells, macrophages, epithelial cells, adipocytes, and hepatocytes [15].

This study intends to compare the serum concentrations of fetuin-A, interleukin-18, LCN-2, and C-reactive protein in patients who are suffering from rheumatoid arthritis and the healthy individuals who are included in the control group.

Materials and methods.

The target population for the trial was a total of 100 patients with confirmed rheumatoid arthritis who were identified at baseline using the ACR/EULAR 2010 RA classification criteria. All the participants of the study were between the ages of forty-one to sixty-four years old.

The total target population of the study was divided into two groups, based on the incidence of the disease. Group one was named the RA group, which included the participants with diagnosed RA. Furthermore, the second group included healthy individuals. The control group consisted of 100 subjects of similar geographic and socioeconomic origin, matched for sex and age, but without RA. blood donors from Kirkuk Blood Transfusion Center were used as healthy controls.

After sampling, the serum is centrifuged to separate it from the cells and stored frozen at -20°. Blood was drawn in dry test tubes. The latex agglutination technique was used to estimate rheumatoid factor (RF). The Westergren method was used to assess the erythrocyte sedimentation rate (ESR) [16]. Commercial ELISA kits from MBL Medical and Biological Laboratories, Japan were used to detect serum levels of IL-6, fetuin-A, and LCN-2according to the manufacturer's instructions.

Results.

The patient group consisted of 55 men and 45 women with a mean age (of 46.38± 11.81) years and a mean disease duration of around zero to ten years. The control group consisted of 100 healthy volunteers, sixty-four men and thirty-six women with a mean age of 47.84 ±10.80 years. 93 cases involved RF, while only 7 cases involved negative results (Table 1). RA criteria were used to diagnose RA in these individuals based on the American College of Rheumatology and European League Against Rheumatism collaborated [17].

Significant differences in diagnostic tests for rheumatoid arthritis, including RF, anti-CCP antibody, and ESR concentrations. The recorded values were as follows, RF (IU/mL) was 545.4 in the RA group, and 8 in the control group. Anti-CCP Antibody was 0.32 in the controlled group, and 545.4 in the RA group, and ESR (mm/h) was 11.1 in the controlled group and 53.7 in the RA group (Table 2).

The values of Fetuin-A (pg/ml) were 470 ± 320 in the RA group and 278 (SD ± 177) in the control group. The LCN-2 (ng/ml) was 67.13 (SD ± 58.71) in the RA group, and in the control group, the value was 45.82 (SD ± 31.01). The Interleukin 6 (pg/ml) was 5.27 (SD ±1.23) in the controlled group and 6.21±1.91 in the RA group. The HDL-cholesterol (mg/dl) was 48.77 (SD ±1.31) in the control group and 52.1 (SD±1.31). Lastly, the recorded Triglycerides (mg/dl) were 97(SD ±1.3) in the controlled group and 94±1.3 in the RA group (Figure 1).

Discussion.

The main finding of this study was that AR patients have significantly higher fetuin-A, LCN-2, CRP, and HDL levels than healthy controls. Triglycerides have significantly reduced in RA patients compared to control. IL-6 level has shown no changes between patients and the control group. A small selection of studies on fetuin-A in RA has produced conflicting findings. Fetuin-A levels were reported to be reduced in RA by

Table 1. *Clinical information about healthy controls and patients with RA.*

Group	RA (n=100)	Control (n=100)
Age (years)	46.38 ±11.81	47.84 ±10.80
Sex (F/M)	45/55	36/64

Table 2. *Biochemical parameters of study groups.*

Group	Control	RA
RF (IU/mL)	8	545.4
Anti-CCP Antibody (RU/mL)	0.32	75.02
ESR (mm/h)	11.1	53.7

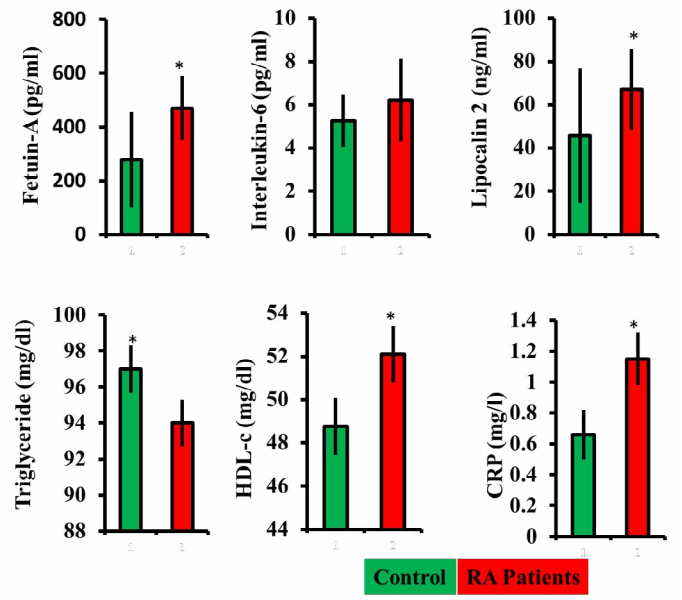

Figure.1. *Biomolecules profile in RA patients compared to control group. Data expressed as mean±SD, *p<0.05.*

HDL-c=High-density lipoprotein cholesterol, CRP=C-reactive protein.

Sato et al. [18]. Nevertheless, this research did not account for the potential impact of corticosteroid use on fetuin-A levels. According to Biswas et al. [19], RA patients had considerably greater fetuin-A levels in their synovial fluid than those in the control group. The current investigation found that RA patients' fetuin-A levels were not statistically significantly higher than those of the peripheral spondyloarthritis and control groups.

Regardless of the disease's severity, the elevated fetuin-A marker in RA raises the possibility that the condition is linked to vascular calcification or atherosclerosis. This could also confirm our results of the elevated plasma triglyceride levels with concomitant elevation of HDL in RA patients compared to the control group [20].

IL-6 has shown non-significant elevation in RA patients compared to the control group. Effects of IL 6 on disease activity, general complaints, and activation are evident in the aetiology of RA [21]. Additionally, research suggests that IL 6 might function as a biomarker for the foretelling of structural damage [21,22]. It is yet unknown what causes the elevated serum of RA patients to rise. IL6 values were not significantly greater than the control group in our investigation, nevertheless. A considerable rise in IL6 values in patients with high disease severity is indicated by elevated IL6 levels in the high disease activity and the significant link with ESR.

Despite the complicated aetiology of RA, the involvement of IL-1, IL-6, and LCN-2 is reported. A glycoprotein called LCN-2—also known as “neutrophil gelatinase-associated lipocalin”—was initially isolated from the renal cells of mice and people. Its participation in inflammation is indicated by the tissue distribution and expression of LCN-2 in neutrophils, bone marrow, and tissues exposed to pathogens, including the trachea, lung, stomach, salivary gland, and colon [23]. Lipopolysaccharide (LPS) and TNF- are the two potent inducers of LCN-2 synthesis in neutrophils, and LCN-2 secretion is

closely regulated by infection and the induction of inflammation [24].

In our study, RA patients had greater LCN-2 values than healthy patients. Parallel findings were made by Katano et al. [25], who discovered that RA patients had greater plasma LCN-2 values than osteoarthritis patients compared to healthy controls. However, negative outcomes were also mentioned [26]. Moreover, an alternative study conducted by Gulkesen et al. (2017), who have elucidated that LCN-2 is reciprocally matched with the severity of the RA indicated by fluctuation of plasma concentration of LCN-2 obtained in mild or moderate RA compared to severe cases. In addition to these factors, several other cytokines should be considered for inflammation regulation including IL-4, IL-8, IL-10, and IL-13[27,28]

Nevertheless, other parameters need to be considered, such as vitamin D deficiency [29] and osteoporosis [30]. Tissue hypoxia leads to cytokine profile modulation or cellular behaviour change [31-34].

Conclusion.

Future thorough investigations will reveal more about the key roles played by Feutin-A and IL-6 in the human inflammatory cascade. The results of our study could spotlight on the pathogenesis, diagnosis, and treatment of joint arthritis. But further investigation appears to be required for better understanding. The protein fetuin A has the possibility to be employed both as a joint treatment follow-up tool and as an indicator of the illness. So, in order to get the right strategy to the patient's sickness and therapy, as well as for other inflammatory conditions, we should be aware of the fetuin A as a marker of functioning. The assessment of LCN-2, IL-6, and Feutin A serum levels as a diagnostic tool in the etiological agent of joint diseases is also anticipated to be confirmed by further clinical research. Combining the determination of these parameters with a clinical evaluation of the patients increases the likelihood of both identifying the condition even in an initial point and minimizing the necessity for additional joint preservation techniques like complete arthroplasty.

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