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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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MYELOPEROXIDASE AND COENZYME Q10 MODULATED IN THE CHRONIC KIDNEY DISEASE PATIENTS

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Abstract.

Background: Kidney failure, also known as end-stage kidney disease, is a medical condition in which the kidneys are functioning at less than 15% of normal. Kidney failure is classified as either acute kidney failure, which develops rapidly and may resolve; and chronic kidney failure, which develops slowly. Diagnosis of chronic failure is based on a glomerular filtration rate (GFR) of less than 15 or the need for renal replacement therapy. It is also equivalent to stage 5 chronic kidney disease.

Aim: The study aimed to evaluate the role of MPO and Co Q10 in different stages of CKD, and correlates this parameter with urea, Cr, Na, K, and eGFR.

Patients and Methods: A Case-control study is carried out in Baghdad in the Imamian Kadhimian Medical City and Al-Karamah Teaching Hospital between August 2022 and March 2023. The number of CKD males under study were 60 male whose ages were between 25 to 50 years old. In addition, the control group consisted of 30 healthy volunteer males aged between 25 to 50 years and they did not have any diseases. Blood samples were collected from each male for measurement of Myeloperoxidase (MPO), and Coenzyme Q10 by Enzyme-linked immunosorbent assay (ELISA). The study showed that the reduced mean level of myeloperoxidase (p value <0.001) in the patient's group compared with the control group, (19.9±6.82 ng/ml) and (42.4±4.98 ng/ml) respectively. Our study revealed that with increasing CKD stage, the myeloperoxidase levels decrease. Also, the reduced mean level of Coenzyme Q10 was 2.97±0.511 ng/ml in the patient group was highly significant than the control group's 7.07±2.41 ng/ml p value <0.001. Our study revealed that with increasing CKD stage, the coenzyme Q10 levels decrease. The study found a positive correlation of serum myeloperoxidase with coenzyme q10 in CKD patients.

Key words. Chronic kidney disease, Myeloperoxidase, Coenzyme Q10.

Introduction.

The kidneys have a bean-like form with medial and lateral concavities. They are located just below the rib cage, one on each side of your spine. They weigh between 120 and 135 g and 150 and 200 g for males and females, respectively. The typical measurements are 10–12 cm long, 5–7 cm broad, and 3–5 cm thick. Each kidney is about the size of a closed hand. They are situated between the transverse processes of T12 and L3 on the posterior abdominal wall, retroperitoneally. Both of the upper poles are normally somewhat medially and posteriorly oriented concerning the lower poles. If the upper renal poles are oriented laterally, it may be a sign of a horseshoe kidney or a superior pole renal tumour. The right kidney often sits a little lower than the left kidney, which is likely due to the liver [1]. Healthy kidneys filter about a half cup of blood every minute, removing wastes and extra water to make urine. The urine flows from the kidneys to the bladder through two thin tubes of muscle called ureters, one on each side of your bladder. Your bladder stores urine. Your kidneys, ureters, and bladder are part of your urinary tract.

The kidneys are made up of nephrons, there are about 2 million nephrons, which are tiny, autonomous functional units with one tubule that reabsorbs the majority of filtered molecules and secretes metabolic waste products, limiting urine production to 1-2 litres per day. Nephron counts are predetermined at birth and begin to diminish around the age of 25 [2]. Without adaptation, healthy persons at age 70 can function with only half of their initial number of nephrons due to the drop-in metabolic activity that occurs with ageing [3]. The incidence of CKD and kidney failure needing KRT rise in the aged population as a result of inadequate nephron endowment at birth or any nephron loss beyond that of normal ageing [4]. A network of capillary loops known as the glomerulus, which is enclosed by a double-layered epithelium known as Bowman's capsule to form a renal corpuscle, is supplied by an afferent arteriole. The glomerulus is drained by an efferent arteriole, which later develops into the vasa recta that feed the renal tubules.

Sequentially, from Bowman's capsule distal, there are the following structures: proximal convoluted tubule, proximal straight tubule, or thick descending limb of the Henle loop, thin descending limb of the Henle loop, thin ascending limb of the Henle loop, distal straight tubule, or think ascending limb of the Henle loop, distal convoluted tubule, collecting tubule, cortical The tubules originate in the cortex, go into the medulla, make a sharp turn in the short loop of Henle, and then return to the cortex close to where they first emerged from the renal corpuscle [5].

Acute Kidney Injury (AKI) can result in the irreversible loss of nephrons at any stage of life, which reduces the lifespan of the kidneys, making AKI and CKD-related conditions [6]. As a result, AKI is a significant risk factor for CKD, particularly in populations who are ageing. AKI disturbs homeostasis, hence severe AKI can be fatal unless kidney replacement therapy keeps the body in a stable state until kidney function returns. Despite kidney replacement therapy (KRT), AKI in conditions of multiorgan failure commonly results in death [7].

The final stage of renal failure is known as chronic kidney disease (CKD) [8]. Without proactive screening, CKD may not be discovered until just before symptoms of renal failure appear since it is typically quiet until its late stages. Few possibilities remain at this stage of the disease process to stop negative outcomes, such as further kidney function loss necessitating dialysis, cardiovascular issues, shorter lifespan, and poor quality of life.

The National Kidney Foundation (NKF) supports the Kidney Early Evaluation Program (KEEP), a national CKD screening program that identifies patients at risk of developing CKD.

KEEP is organized by the NKF from its central offices, but a team of volunteer medical experts manages the program in the majority of states through NKF affiliates. Patients can hear about KEEP in a variety of ways, such as through referrals from medical professionals or by reading about the program in lay publications and through NKF poster campaigns. The kidneys eliminate metabolic waste products, regulate bodily fluids, electrolytes, osmolality, and pH, and release hormones and bioactive substances.

Chronic kidney disease is characterized by a decline in kidney function, an estimated glomerular filtration rate (eGFR) of less than 60 ml/min/1.73 m2, or kidney damage indicators such albuminuria, hematuria, or abnormalities shown on imaging that have been present for at least three months [9].

Myeloperoxidase (MPO) is a member of the human peroxidase family of proteins expressed mainly in immune cells such as neutrophils, monocytes and macrophages [10], and to a lesser extent in other cell types such as certain subsets of human peripheral B, CD4(+) and CD8(+) T lymphocytes [11]. The enzyme is particularly abundant in neutrophils, where it accumulates in azurophil granules [12]. This family of proteins catalyzes the breakdown of peroxides and is involved in killing foreign bodies [13]. Upon activation, neutrophils release superoxide anions, which may then release other reactive oxygen species (ROS), like hydrogen peroxide (H2O2) [13,14]. MPO catalyzes the formation of hypochlorous acid (HOCl) from H2O2 and chloride ions [15], which functions as a potent microbicidal compound because of its ability to alter amino acids, lipids, and DNA [16,17], making MPO an important component of the innate immune system [18]. MPO is synthesized as a preproprotein that is proteolytically processed to remove a 48 amino acid (aa) signal peptide, a 116 aa propeptide, the C-terminal serine, and a 6 aa internal peptide which generates separate 60 kDa heavy and 12 kDa light chains [19]. MPO expression occurs in myeloid precursors in the bone marrow, mainly at the promyelocyte stage, but not in mature phagocytes [19,20]. The protein is formed as a pre-proMPO that undergoes several modifications, including insertion of a heme moiety, glycosylation, phosphorylation of mannose residues, proteolytic cleavage, and dimerization [20,21]. The MPO protein is a tetramer, with a total estimated molecular weight of 150 000 kDa. The tetramer is composed of two halves (hemi-MPO), composed of two heavy (~60 kDa) and two light (~15 kDa) components [22,23]. Previously, a range of molecular weights was reported at 120 000–160 000 kDa [24]. Human MPO contains (573) amino acids, and they are divided into light and heavy chains, the heavy ones containing (467) amino acids, while the light ones contain (106) amino acids. In this respect, this enzyme has been found to participate in several physiological and pathological conditions, including regulation of the inflammatory responses.

Coenzyme Q10, a fat-soluble organic molecule similar to a vitamin, is endogenously synthesized by human cells [25]. It comprises a benzoquinone group and a poly-isoprenoid side chain of ten isoprenoid units in humans. Its molecular formula is C59H90O4 (molecular weight 863.3 g/mol), also known as ubidecarenone, coenzyme Q10, and ubiquinone-10. Coenzyme Q10 exists in three oxidation states: the fully reduced ubiquinol form (CoQ10H2), the radical semiquinone intermediate (CoQ10H.), and the fully oxidized ubiquinone form Although similar in structure to some vitamins (eg, vitamin K), CoQ10 is not a vitamin since it is synthesized in the body, whereas vitamins must be obtained from the diet [26]. CoQ10 has a key role in the process of cellular energy supply via oxidative phosphorylation within mitochondria, shuttling electrons from complexes I and II to complex III of the mitochondrial respiratory chain. In addition to its role in mitochondrial function, CoQ10 is present in other subcellular organelles, including lysosomes, peroxisomes, Golgi apparatus and endoplasmic reticulum. As well as providing antioxidant protection for these organelle membranes from oxidative stress, CoQ10 has a role in maintaining the intralysosomal pH [27]. CoQ10 can be absorbed from the small intestine into the lymphatic system and then enter the blood circulation; bile is the main elimination route [28]. Higher amounts of CoQ10 are observed in tissues with high energy requirements or metabolic activity, e.g., heart, kidney, liver, and muscle [29].

According to reports, CoQ10 has a half-life of 21.7 hours. By being phosphorylated in the cells and then being transported to the kidneys, CoQ10 may be processed in all organs. Cell membranes commonly contain CoQ10, especially in mitochondria [30].

CoQ10 exerts its biological effect largely based on its lipophilic antioxidant capacity, scavenging free radicals by suppressing the initiation and development of lipid peroxidation in cell membranes [31]. The levels of CoQ10 are high in human organs with high metabolic activity (e.g., liver, kidney, and heart) [32]. CoQ10 is involved in the production of adenosine triphosphate (ATP), regulating mitochondrial respiratory chain complexes. In humans, CoQ10 levels are typically measured in blood samples, although there is some discussion as to how accurately blood CoQ10 levels reflect those within other tissues; CoQ10 levels may, therefore, be measured in muscle biopsy specimens although less frequently [33]. CoQ10 deficiencies have been found in patients with various diseases, including chronic kidney disease, cancers, cardiovascular diseases (e.g., statin myopathy, congestive heart failure, and hypertension), diabetes mellitus, dementia, hepatitis, Parkinson's disease, skin ageing, and renal diseases [34].

Patients and Methods.

A Case-control study is carried out in Baghdad city from August 2022 and March 2023. The number of CKD males under study were 60 male whose ages were between 25 to 50 years old. These patients were admitted to the hemodialysis unit and a consultant nephrologist at Imam Al-Kadhimi Teaching Hospital and Al-Karama Teaching Hospital in Baghdad. Chronic kidney disease was diagnosed based on the presence of the following criteria: high levels of urea and creatinine, presence of protein in urine especially albumin called (proteinuria) and decreased estimated glomerular rate that determines the stages of chronic kidney disease.

In addition, the control group consisted of 30 healthy volunteer males who did not have any diseases aged between 25 to 50 years and were asked to complete a general questionnaire. Patients with metabolic or some disorder diabetes mellitus,

viral hepatitis, and cancers, whether benign or malignant, who are taking certain medications, and excluded from the study by specific laboratory tests.

The approval permission was submitted to the Director of Health of Baghdad Al-Karkh/Al-Imamin Al-Kadhimin City Teaching Hospital and Al-Karama Teaching Hospital.

Materials and Methods.

Five ml of blood sample was taken by vein puncture from each subject enrolled in this study. Blood samples were added to gell tubes after blood clotting and centrifuged at 3000 rpm for 15 minutes then the clot was removed and remained re-centrifuged at 3000 for 10 minute and the obtained serum were aspirated using a mechanical micropipette and transferred into clean test tubes which labelled and measurement of Myeloperoxidase (MyBioSource/USA), and Co-enzyme Q10 (MyBioSource/USA) by Enzyme-linked immunosorbent assay (ELISA).

Results.

The results in Table 1 showed the descriptive statistics of age in both study groups. The minimum age was 25 and 28 years in the patients and control groups, respectively. While the maximum age was 52 and 48 years old in patients and control groups, respectively. There was no statistical difference between the mean age of patients 40.3±7.53 years and the control group 38±6.23 p value =0.117.

The results in Table 2 showed the reduced mean level of myeloperoxidase (p value <0.001) in the patient's group compared with the control group, (19.9±6.82 ng/ml) and (42.4±4.98 ng/ml), respectively.

The results in Table 3 showed the reduced mean level of CoQ-10 was 2.97±0.511 ng/ml in the patient group was highly significant than the control group's 7.07±2.41 ng/ml p value <0.001.

The results revealed that with increasing CKD stage, the myeloperoxidase levels decrease and the coenzyme Q10 levels decrease (Figure 1).

Table 1. *Age characteristics in study groups.*

Age	**Control (n=30)**	**Patient (n=60)**
Mean±SD	38.0±6.23	40.3±7.53
Minimum	28.0	25.0
Maximum	48.0	52.0
P value	0.117^{NS}	
NS: no statistical significance (p>0.05).		

Table 2. *Descriptive analysis of serum myeloperoxidase in study groups.*

Myeloperoxidase (ng/ml)	**Control**	**Patient**
Mean	42.4	19.9
Std. Deviation	4.98	6.82
p value	<0.001**	
**high statistically significant p≤0.001.		

Table 3. *Descriptive analysis of serum Coenzyme Q10 in study groups.*

Coenzyme Q10 (ng/ml)	**Control**	**Patient**
Mean	7.07	2.97
Std. Deviation	2.41	0.511
p value	<0.001**	
**high statistically significant p≤0.001.		

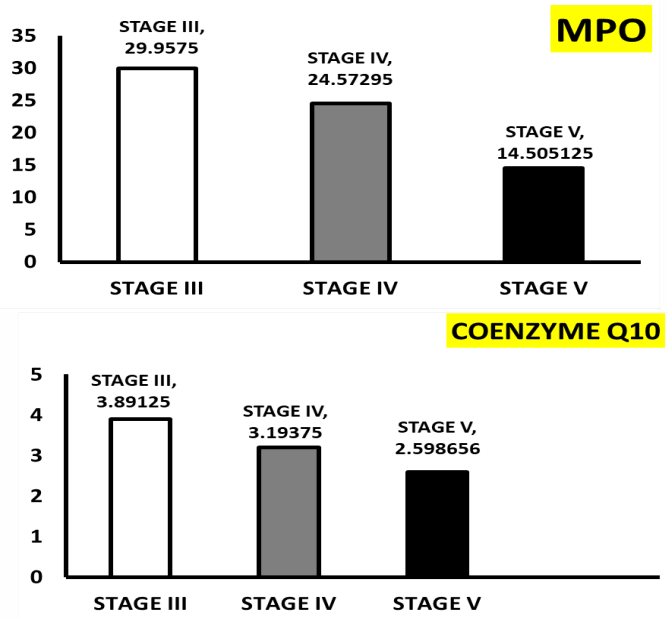

Figure 1. *The serum myeloperoxidase and Coenzyme Q10 levels in different stages in CKD patients.*

Discussion.

Our study included 60 Iraqi patients with chronic kidney disease (males only) with an age range of 38±6.23 years old 30 people were taken as a control group (males only) and their age was 40.3 ± 7.53 and they did not have any diseases. Chronic kidney disease, also called chronic kidney failure, involves a gradual loss of kidney function. Advanced chronic kidney disease can cause dangerous levels of fluid, electrolytes & wastes to build up in your body. As there is no cure for chronic kidney disease, treatment aims to help relieve the symptoms & stop it from getting worse. The best way to stop the progression of chronic kidney disease into more severe stages is early detection of patients with risk factors to progress. Are one of the tests that are better to be done in patients with different stages of chronic kidney disease to assess the severity & the predilection for progression into the more severe stage of chronic kidney disease.

Our study showed that Myeloperoxidase is significantly reduced in a patient with chronic kidney disease and the more severe the stage, the more Myeloperoxidase is decreased, the same result is concluded by Ahmed et al. (2013), [35] and Afshinniaet al. (2017), [36]. Myeloperoxidase levels decrease as the chronic kidney disease stage increases & the least level of Myeloperoxidase was found in stage V. Patients with a level of Myeloperoxidase equal to 10 ± 1 were found to be in more need of hemodialysis & as we informed by the doctors, they need medical consultation by cardiologist & two of them develop cerebrovascular accident (CVA). So, our study conducted that patients with low levels of Myeloperoxidase need frequent dialysis & they are prone to complications more than other patients with higher levels of Myeloperoxidase.

Correa et al. (2020), [37] found that myeloperoxidase levels were higher in patients with chronic kidney disease. The reason for this difference in results could be attributed to, a greater sample size, a higher concentration of more likely women, and different ethnicity. Our study shows that chronic kidney disease patients have low levels of Coenzyme Q10. These results come in agreement with Xu et al. (2019), [38] and Mehmetoglu et al. (2012) [39]. However, the limitation of the response could be jointly related to the involvement of trophic factors released from localized milieu [40,41], or because the patients may undergo hemodialysis leading to alteration of serum concentration of different biomolecules including Coq10 or MPO [42,43].

Conclusion.

Chronic kidney disease is a global disease that could affect any age & is associated with high morbidity & mortality. Myeloperoxidase & Coenzyme Q10 are decreased in patients with CKD, the higher the stage, the more decrease in MPO & CoQ10 levels.

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