GEORGIAN MEDICAL NEWS

ISSN 1512-0112

NO 7-8 (352-353) Июль-Август 2024

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
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Содержание:

GEORGIAN MEDICAL NEWS
No 7-8 (352-353) 2024

CHARACTERIZATION OF SERUM SERINE PROTEASE BIOCHEMICAL PROFILE IN PATIENTS WITH RENAL FAILURE

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Abstract.

The present study meticulously delineates the biochemical alterations in serine protease activity and various life variables in patients with kidney failure compared to a control group. By evaluating 160 samples, comprising 80 from individuals with renal failure and 80 from healthy controls, the researchers observed a significant elevation in serine protease activity among kidney failure patients (274.38 ± 1.55 U/L) relative to the control group (173.78 ±1.49 U/L). Beyond serine protease, other enzymes such as lactate dehydrogenase, basal phosphatase, myeloperoxidase, peroxidase, and aspartate aminotransferase also showed heightened activities in renal failure patients; alanine aminotransferase similarly exhibited a notable increase. Conversely, catalase and arylesterase activities were markedly reduced in these patients compared to controls. The mineral profile revealed substantial decrements in calcium, iron, copper concentrations alongside potassium levels in kidney failure sufferers while showing pronounced increments in phosphate, zinc, and sodium concentrations. Furthermore, protein profiles indicated a stark decrease in total protein, albumin levels along with triglycerides and various cholesterol forms except for high-density lipoprotein cholesterol which increased significantly alongside urea, creatinine and glucose levels; globulin and uric acid also saw considerable elevations when contrasted with the control group's data. These comprehensive findings underscore the profound metabolic disruptions inherent to kidney failure while providing pivotal insights into enzyme activities and mineral imbalances associated with this condition.

Key words. Renal failure, serine protease, arylesterase.

Introduction.

The kidneys, crucial for maintaining the body's internal environment through filtration of blood and removal of waste products, are susceptible to a myriad of diseases that can significantly impact their function [1,2]. Among these ailments are pyelonephritis, kidney cancer, kidney stones, autosomal dominant polycystic kidney disease (ADPKD), autosomal dominant tubulointerstitial kidney disease, azotemia, diabetic kidney disease, glomerular disease, renal papillary necrosis, proteinuria-related disorders, uremia, and various forms of renal failure [3]. Chronic kidney disease (CKD) alone poses a formidable global health challenge affecting nearly 850 million people worldwide [4]. Characterized by a long-term decline in renal function leading to ineffective waste filtration and fluid balance maintenance—as well as complications such as anemia, bone disorders, and cardiovascular issues—CKD necessitates interventions like dialysis or transplantation in advanced stages [5]. Conversely, acute renal failure (ARF), also known as acute kidney injury (AKI), represents a rapid deterioration in renal function over days with high mortality rates affecting 9.1% of the global population [6]. ARF can be triggered by factors including malignant tumors, bacteremia; certain medications and poisons; interrupted blood supply during surgeries; and severe multi-organ dysfunctions such as cardiovascular ailments or liver dysfunctions [7]. Therefore, early detection and management of both acute and chronic renal conditions are imperative to preserving kidney function and improving patient outcomes.

Chronic kidney disease (CKD), also known as chronic renal failure (CRF), is a significant global health issue characterized by the presence of kidney damage or a reduced GFR below 60 ml/min/1.73 m² for three months or more, regardless of the underlying cause [8]. The primary drivers of CKD include diabetes and hypertension, with additional factors such as HIV infection and aging contributing to its prevalence [9,10]. The insidious nature of CKD's progression often results in patients being unaware of their condition until it has advanced considerably, highlighting the critical need for early diagnosis and effective screening [11]. CKD is an independent risk factor for cardiovascular disease; individuals in advanced stages are at particularly high risk for cardiovascular mortality [12]. Despite indirect measurement of GFR being the standard reference method for assessing kidney function, this approach is impractical for routine use due to its complexity and duration. Consequently, markers such as serum creatinine and cysteine C are employed to estimate GFR (eGFR), though these methods can be influenced by non-renal factors like muscle mass, age, and gender, leading to inaccuracies especially among black Africans [9]. Furthermore, while albuminuria serves as an established marker for detecting early stages I and II of renal failure when GFR levels exceed 60 ml/min per 1.73 m², its predictive power remains limited [13]. Kidney biopsies provide diagnostic confirmation but involve high risks and potential complications. Staging CKD involves stratifying patients based on GFR levels: from stage G1 with a GFR ≥90 ml/min per 1.73 m² to stage G5 with a GFR <15 ml/min per 1.73 m² [14]. Similarly, albuminuria levels are classified using the albumin-to-creatinine ratio (ACR) into three categories: A1 (<30 mg/gm), A2 (30-299 mg/gm), and A3 (>300 mg/gm) [15]. Common. causes leading to end-stage kidney disease include Type II diabetes, hypertension, glomerulonephritis, interstitial tubular nephritis, hereditary diseases among others [10,16,17]. Factors like obesity exacerbate CKD progression through insulin resistance and dyslipidemia mechanisms. Symptoms become apparent predominantly in the latter stages of CKD—4 and 5—manifesting through nausea, vomiting, fatigue among other signs that severely impact quality of life if left unchecked.

Materials and Methods.

Samples: The study was conducted on human blood serum samples and samples were collected for the period from January 2022 to February 2022 in cooperation with Ibn Sina Hospital and Al-Salam Hospital in the Dialysis Department.

Control group: (80) blood samples were collected from apparently healthy persons, including (46) males and (36) females, aged between (32-61) years.

Patient group: (80) blood samples were collected for people with kidney failure diseases and included (40) males and (40) females, and their condition was diagnosed by doctors specializing in nephrology and urology in cooperation with Al-Salam Hospital and the Mosul Center for Nephrology and Surgery, their ages ranged between (34-69) years.

Blood samples collection: blood samples were collected after sterilizing the area with Heptin, where 8 ml of venous blood was withdrawn for the control group and the group of kidney failure patients and placed in plastic tubes and then left to coagulate at a temperature of (37°C), then a centrifugal of blood was conducted for a period of (20 min at a speed of (4000xg) to obtain blood serum.

Materials: In this study, several ready-made tests (standard Kit) from the French company (Biolabo) were used to measure the concentration of glucose, urea, creatinine, calcium, total cholesterol, triglycerides, high-density lipoprotein cholesterol, total protein, albumin and lactate dehydrogenase. The procedure was conducted as per manufacturer instructions.

Results.

The results showed significant ($p \leq 0.001$) serum increase in the activity of LDH, ALP, myeloperoxidase, peroxidase, AST, and ALT in patients with kidney failure compared to the control group, while arylesterase and catalase were significantly reduced in patients with renal failure compared to control group (Table 1).

Table 1. Enzymatic activity in the studied groups.

Biochemical Parameters	**Control group**	**Renal failure group**	**p value**
Arylesters (U/L)	105.36±1.93	69.24±1.46	P ≤ 0.001
Catalase (U/L)	50.85±1.57	31.23±1.82	P ≤ 0.001
LDH (U/L)	177.3±2.5	249.83±2.9	P ≤ 0.001
ALP (U/L)	141.97±1.99	325.06±35.08	P ≤ 0.001
Myeloperoxidase (U/L)	27.9±1.11	122.14±5.82	P ≤ 0.001
Peroxidase (U/L)	45.23±1.73	149.43±19.62	P ≤ 0.001
AST (U/L)	11.66±0.22	18.53±1.25	P ≤ 0.001
ALT (U/L)	11.38±0.134	16.88±1.43	P ≤ 0.01
Data expressed as mean±SE, p value considered significant less than 0.05 using Two sample t-test			

Table 2. Electrolyte changes in the studied groups.

Biochemical Parameters	**Control group**	**Renal failure group**	**p value**
Ca^{+2} (mg/dL)	8.983±0.0345	7.692±0.0592	**P ≤ 0.001**
Iron (mg/dL)	77.40±1.24	59.86±2.61	**P ≤ 0.001**
Phosphate (mg/dL)	30.967±0.0417	39.60±0.169	**P ≤ 0.001**
Mg^{+2} (mg/dL)	38.56±0.120	30.85±1.942	**P > 0.05**
Cu^{+2}(ug/dL)	106.375±1.264	81.250±2.524	**P ≤ 0.001**
Zn^{+2} (mg/dL)	75.974±1.429	56.737±1.195	**P ≤ 0.001**
Na^{+} (mmoL/dL)	166.59±4.37	142.51±1.39	**P ≤ 0.001**
K^{+}(mmoL/dL)	4.67±0.252	4.06±0.110	**P ≤ 0.05**
Data expressed as mean±SE, p value considered significant less than 0.05 using Two sample t-test			

Renal failure patients were associated with significant $P \leq 0.001$reduction in serum electrolyte levels including calcium, iron, magnesium, copper, zinc, sodium, and potassium. Conversely renal failure associated with significant $P \leq 0.001$ elevation of phosphate levels (Table 2).

Renal failure patients were associated with significant $P \leq 0.001$reduction in serum total protein, albumin, and lipid profile. Conversely renal failure associated with significant $P \leq 0.001$ elevation of urea, creatinine, globulin, uric acid, and glucose levels (Table 3).

Table 3. Renal and metabolic parameters in the studied groups.

Urea (mg/dl)	20.85±0.37	133.96±1.69	**P ≤ 0.001**
Creatnine (mg/dl)	0.752±0.011	8.958±0.050	**P ≤ 0.001**
Total Protein (g/L)	6.99±0.032	5.9313±0.048	**P ≤ 0.001**
Albumin (g/L)	4.5350±0.0577	2.3210±0.0369	**P ≤ 0.001**
Globulin (g/L)	2.4550±0.0359	3.6103±0.0730	**P ≤ 0.01**
LDL (mg/dl)	85.18±1.94	37.23±2.45	**P ≤ 0.001**
T.G (mg/dl)	120.60±1.68	82.90±3.84	**P ≤ 0.001**
Cholesterol (mg/dl)	152.12±2.26	100.95±2.46	**P ≤ 0.001**
VLDL (mg/dl)	23.84±0.29	19.56±2.02	**P ≤ 0.001**
Uric acid (mg/dl)	4.83±0.06	5.59±0.11	**P ≤ 0.01**
Glucose (mg/dl)	109.52±0.52	134.03±6.13	**P ≤ 0.001**
HDL (mg/dl)	43.17±0.64	52.9±0.81	**P ≤ 0.001**
Data expressed as mean±SE, p value considered significant less than 0.05 using Two sample t-test			

Discussion.

The results showed significant serum increase in the activity of LDH, ALP, myeloperoxidase, peroxidase, AST, and ALT in patients with kidney failure compared to the control group, while arylesterase and catalase were significantly reduced in patients with renal failure compared to control group. The multifaceted complications arising from renal failure extend beyond the kidneys themselves, implicating liver damage, bone issues, and cardiovascular risks. Hwang et al. (2020) highlight that liver damage and bone problems associated with renal failure can elevate the risk of cardiovascular complications [18]. Despite LDH being a useful marker for heart attacks, its accuracy is hindered by its wide distribution in various tissues [19]. Patients undergoing chronic renal failure frequently suffer from liver diseases such as viral hepatitis—often contracted during dialysis—and cholestasis caused by renal tubular dysfunction [20]. Additionally, research by Hani & Zainal (2024) has demonstrated elevated peroxidase activity in these patients, which helps mitigate oxidative stress through the neutralization of free radicals and ROS [21]. Similarly, increased serine myeloperoxidase levels are noted in conditions like pyelonephritis and glomerulonephritis, further exemplifying the systemic inflammatory responses characteristic of renal impairment [22]. Table 4-1 reveals a significant reduction in arylesterase activity in patients with kidney failure compared to healthy controls, suggesting that diminished enzyme function contributes to heightened oxidative stress—a key driver of kidney deterioration [23,24]. Furthermore, Hong and Park's study (2021) indicates a pronounced decrease in catalase effectiveness within this patient group [25]. This reduction

exacerbates mitochondrial dysfunction due to an overabundance of ROS and interstitial tubular fibrosis linked to lipid peroxide accumulation. Collectively, these findings underscore how renal failure instigates a cascade of biochemical abnormalities that contribute to systemic organ impairment and necessitate comprehensive clinical management strategies.

The results presented study revealed profound biochemical alterations in the blood serum of patients with kidney failure, characterized by significant reductions and increases in various minerals and electrolytes when compared to a control group. A highly significant decrease in the concentrations of iron, copper, and zinc was observed among the kidney failure patients [19,26-28]. This reduction can be linked to impaired erythropoietin production due to compromised kidney function, which inhibits iron absorption as evidenced by elevated hepserin levels [29]. Additionally, hypokalemia and diminished calcium concentrations were noted, which are perilous conditions given their association with cardiovascular complications and poor bone metabolism due to impaired vitamin D activation in renal impairment [29]. Despite these declines, no notable change was detected in magnesium levels—highlighting the mineral's unique regulation through diet, bone exchange, intestinal absorption, and renal secretion processes [30]. In stark contrast, there was a marked increase in phosphate and sodium concentrations among these patients. Elevated phosphate is particularly problematic as it precipitates hyperphosphatemia—a common occurrence especially in late-stage renal disease—leading to cardiovascular calcification and secondary hyperparathyroidism that further exacerbate metabolic bone disorders [31]. High sodium levels also correspond with adverse health outcomes reported in previous studies on renal dysfunctions, underscoring the intricate interplay between electrolyte balance disturbances and chronic kidney diseases [32].

The results presented highlighted significant biochemical alterations in patients with renal failure compared to a control group, underscoring the multifaceted metabolic dysregulations associated with kidney disease. Notably, there is a marked increase in serum glucose levels, suggesting impaired glucose metabolism potentially linked to insulin resistance or decreased renal gluconeogenesis. Additionally, urea concentration is significantly elevated, corroborating findings by Hameed et al. (2023) that chronic renal failure patients exhibit substantially higher urea levels due to reduced renal clearance [19]. The lipid profile reveals complex changes: a substantial decrease in total cholesterol, triglycerides, LDL, and VLDL cholesterol alongside an increase in HDL cholesterol. This dyslipidemia is often implicated in the heightened risk of cardiovascular disease among renal failure patients, particularly those on dialysis. Moreover, creatinine levels show a pronounced rise, aligning with studies indicating severe impairment in glomerular filtration rates among this population [19]. A notable decrease in total protein concentration further emphasizes the catabolic state observed in these patients—driven by hormonal imbalances and exacerbated by conditions such as acidosis and inflammation—which contributes to persistent negative nitrogen balance and muscle wasting [33]. Interestingly, uric acid levels also significantly increase, consistent with hyperuricemia resulting from factors like purine-rich diets and diminished excretion due to compromised kidney function [34]. Elevated globulin concentrations paired with low albumin indicate severe malnutrition and heightened mortality risks in dialysis patients—a concern echoed by Pai et al.'s study linking high globulin and low protein states with increased death rates from both cardiovascular causes and all-cause mortality. Lastly, the inverse relationship between worsening renal function and decreasing albumin concentration highlights another critical aspect of the clinical management of chronic kidney disease progression.

Conclusion.

Renal failure, a condition characterized by the kidneys' inability to adequately filter waste products from the blood, is often associated with significant disturbances in various biochemical and enzymatic parameters. Electrolyte imbalances such as hyperkalemia, hyponatremia, and hypocalcemia frequently accompany renal dysfunction due to impaired renal excretion mechanisms and disrupted homeostasis. Furthermore, liver enzymes like alanine aminotransferase (ALT) and aspartate aminotransferase (AST) may be elevated in renal failure patients, reflecting potential hepatorenal syndrome or concurrent hepatic injury. Renal function tests typically reveal elevated serum creatinine and blood urea nitrogen (BUN), signalling a decline in glomerular filtration rate (GFR) and overall kidney performance. Oxidative stress plays a critical role in the pathophysiology of renal failure; it is marked by increased levels of reactive oxygen species (ROS) and decreased activity of antioxidant enzymes such as superoxide dismutase (SOD), catalase, and glutathione peroxidase. These oxidative imbalances exacerbate renal tissue damage through lipid peroxidation, protein oxidation, and DNA damage pathways. This complex interplay between electrolyte disturbances, altered enzyme activities, impaired renal function tests, and oxidative stress highlights the multifaceted nature of renal failure's pathogenesis and underscores the necessity for comprehensive management strategies that address these diverse biochemical derangements.

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