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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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SAFETY OF TAMSULOSIN ALONE OR IN COMBINATION WITH 5A-REDUCTASE INHIBITORS ON LIPID PROFILE, RENAL FUNCTION, AND LIVER PARAMETERS

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Abstract.

Background: Tamsulosin and 5α-reductase inhibitors (finasteride and dutasteride) are widely prescribed for benign prostatic hyperplasia (BPH). However, their systemic effects on metabolic, renal, and hepatic functions remain incompletely understood.

Objective: To evaluate the safety profile of tamsulosin monotherapy compared with combination therapies (tamsulosin plus finasteride or dutasteride) on lipid profile, renal function, liver enzymes, and bilirubin levels in men with BPH.

Methods: This cross-sectional study included 102 male participants aged >50 years, divided into five groups: healthy controls (n=26), newly diagnosed treatment-naïve BPH (n=25), BPH receiving tamsulosin monotherapy >3 months (n=26), BPH receiving tamsulosin+finasteride >3 months (n=11), and BPH receiving tamsulosin+dutasteride >3 months (n=14). Serum lipid profile (total cholesterol, triglycerides, HDL-C, LDL-C, VLDL, atherogenic index), renal function (urea, creatinine, eGFR, uACR), liver function (ALT, AST, ALP, GGT, albumin), and bilirubin fractions (total, direct, indirect) were measured using standardized automated methods.

Results: Lipid profile parameters showed no significant differences between treatment groups and controls, with all values remaining within desirable ranges. Renal function tests, including serum urea, creatinine, eGFR, and urinary albumin-to-creatinine ratio, demonstrated no clinically significant alterations across all BPH treatment groups compared to controls. Liver enzymes (ALT, AST, ALP, GGT) and serum albumin levels remained within normal limits across all groups. Total, direct, and indirect bilirubin concentrations showed no significant elevation, indicating preserved hepatic excretory function. Notably, no statistically significant differences were observed between tamsulosin monotherapy and combination therapy groups for any of the measured parameters.

Conclusion: Tamsulosin administered alone or in combination with finasteride or dutasteride for more than three months demonstrated non-significant differences in the measured biochemical parameters among the studied groups represented by no adverse effects on lipid metabolism, renal function, liver enzymes, or bilirubin homeostasis. These findings support the safe use of these therapeutic regimens in elderly BPH patients, including those with underlying metabolic or organ function concerns. Further prospective longitudinal studies with larger sample sizes are warranted to confirm these observations.

Key words. Benign prostatic hyperplasia, tamsulosin, 5-alpha reductase inhibitors, lipid profile, renal function, liver function.

Introduction.

Benign prostatic hyperplasia (BPH) is a prevalent urological condition affecting aging men, with its incidence increasing progressively after the fifth decade of life [1]. The medical management of BPH has evolved significantly, with alpha-blockers such as tamsulosin and 5α-reductase inhibitors (5-ARIs) including finasteride and dutasteride representing cornerstones of pharmacological therapy [2]. Tamsulosin, a selective alpha-1A adrenoceptor antagonist, provides rapid symptomatic relief, thereby addressing the underlying pathophysiology [3]. Although combination therapy has demonstrated superior efficacy in reducing symptom progression and preventing acute urinary retention, the long-term safety profile of these agents—particularly their extra-prostatic effects on metabolic, renal, and hepatic functions—remains incompletely characterized.

Emerging evidence suggests potential off-target effects of alpha-blockers and 5-ARIs on lipid metabolism, renal hemodynamics, and hepatic enzyme activities. Tamsulosin, through its effects on peripheral vascular tone, may influence lipid profiles and renal perfusion [4,5]. Similarly, 5-ARIs, by modulating androgen metabolism, could potentially alter hepatic synthetic function and lipid homeostasis. Given that patients receiving BPH therapy are often elderly with multiple comorbidities, including dyslipidemia, chronic kidney disease, and non-alcoholic fatty liver disease, understanding the systemic safety of these medications is of paramount clinical importance [3]. Despite the widespread use of tamsulosin alone or in combination with finasteride or dutasteride, comprehensive studies evaluating their concurrent effects on lipid profiles, renal function parameters (including urea, creatinine, estimated glomerular filtration rate, and urinary albumin-to-creatinine ratio), and liver function tests (including aminotransferases, alkaline phosphatase, gamma-glutamyl transferase, and bilirubin fractions) remain limited [2].

Therefore, this study was designed to evaluate the safety profile of tamsulosin monotherapy compared with combination regimens of tamsulosin plus finasteride or dutasteride on key metabolic, renal, and hepatic parameters in men with BPH. By comparing treatment-naïve BPH patients, treated patients, and healthy controls, this investigation aims to provide evidence-based insights into the systemic effects of these commonly prescribed medications, thereby guiding clinicians in optimizing therapeutic decisions for elderly patients with BPH and concomitant metabolic or organ dysfunction.

Materials and Methods.

Study design:

This cross-sectional study was conducted from November 2025 to March 2026 at Al-Salam Teaching Hospital, Al-Mosul General Hospital, Al-Jumhori Teaching Hospital, and the Research Hospital in Nineveh Governorate. A total of 102 male participants aged over 50 years were enrolled and divided into five groups: Group I (control, n=26) comprised healthy subjects with prostate size less than 30 cc; Group II (n=25) included

newly diagnosed benign prostatic hyperplasia (BPH) patients with prostate size exceeding 30 cc who had not yet received treatment; Group III (n=26) consisted of BPH patients receiving tamsulosin monotherapy for more than three months; Group IV (n=11) received combination therapy of tamsulosin and finasteride for more than three months; and Group V (n=14) received combination therapy of tamsulosin and dutasteride for more than three months. These medications were administered at daily dosing intervals of tamsulosin 0.4mg, finasteride 5mg, and dutasteride 0.5mg.

Ethical adherence: Ethical approval was obtained from the University of Mosul Collegiate Committee for Medical Research Ethics (No. 58, code PREC-25-3-17) and the Iraqi Ministry of Health/Nineveh Health Directorate (No. 52488). Written informed consent was obtained from all participants.

Demographic parameters including age, residence, marital status, occupation, and clinical data encompassing urinary symptoms (irritative/storage symptoms and voiding/obstructive symptoms), comorbidities, medication history, family history of prostate disease, smoking status, and alcohol intake were collected through face-to-face interviews. Anthropometric measurements included weight (kilograms) and height (meters), from which body mass index (BMI) was calculated as weight divided by height squared (kg/m²).

Biochemical Analyses:

Study population: Male over 50 years and older, the patients who have been diagnosed with benign prostatic hyperplasia (BPH), or those on treatment with tamsulosin alone or on combination therapy of tamsulosin plus finasteride or dutasteride and comparing them with normal prostate size participants (apparently healthy control group). Participants who have been presenting with specific symptom score (moderate to severe, IPSS ≥10), and those planning for medical or surgical treatment for BPH. About Consent, they must be able to provide informed consent and able to complete study questionnaires.

Exclusion Criteria: Participants below 50 years and any prior cancer diagnosis, especially prostate cancer were excluded. Other Urinary Conditions like: Prostatitis, urinary incontinence, urethral strictures, or other also were excluded. Previous surgery of the lower urinary tract or medical treatments; like current use of certain medications that affect BPH symptoms, such as diuretics, antihistamines, antimuscarinic or tricyclic antidepressant drugs. Medical conditions like immunocompromised status, uncontrolled diabetes (HbA1c >7%), cognitive or psychiatric, like neurological, physical, or psychiatric disturbances, chronic kidney disease stage 3 or higher (eGFR<60), chronic liver disease (cirrhosis, hepatitis), alcoholics were all excluded.

Venous blood samples (10 mL) were collected from each participant using sterile disposable syringes. The blood was placed in gel activator tubes for serum separation following centrifugation at 3000 rpm for 10 minutes.

Renal function assessment included serum urea and creatinine measured photometrically using Mindray BS-240 autoanalyzer with enzymatic kits (Shenzhen Mindray Bio-medical Electronics, China). Urea was determined by the urease-glutamate dehydrogenase UV method at 340 nm, while creatinine was measured by the sarcosine oxidase method at 546 nm. Estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI creatinine equation. Urinary albumin-to-creatinine ratio (uACR) was determined using AFIAS-6 fluorescence immunoassay (Boditech, Republic of Korea) for microalbumin, with urine creatinine measured by the enzymatic method on Mindray BS-240.

Liver function tests included alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), and serum albumin, all measured on Mindray BS-240 autoanalyzer using kinetic and colorimetric methods. ALT and AST were determined by IFCC-recommended methods without pyridoxal phosphate activation at 340 nm. ALP was measured using the ρ-nitrophenyl phosphate method at 405 nm. Gamma-glutamyl transferase (GGT) was quantified using FUJIFILM DRI-CHEM NX600 analyzer with reflective spectrophotometry at 400 nm. Total and direct bilirubin were measured by the vanadate oxidation method at 450 nm on Mindray BS-240, with indirect bilirubin calculated mathematically as total bilirubin minus direct bilirubin.

Lipid profile assessment included total cholesterol (TC), triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C) measured on Mindray BS-240 using enzymatic colorimetric methods (CHOD-POD for cholesterol, GPO-POD for triglycerides, and direct method for HDL-C). Very low-density lipoprotein (VLDL) was calculated as TG/5, while low-density lipoprotein cholesterol (LDL-C) was estimated using equation: LDL-C = [Total cholesterol − HDL-C] − TG/5. The atherogenic index (AI) was calculated as log (TG/HDL-C).

Statistical Analysis: Data were analyzed using SPSS version 16 for Windows (IBM Co., Chicago, IL, USA). Normality of continuous variables was assessed using the Shapiro–Wilk test, while non-continuous variables were evaluated using Fisher's exact test. One-way ANOVA followed by post-hoc testing at 95% confidence intervals was used for continuous variables, and Chi-square test for non-continuous variables. A P-value ≤ 0.05 was considered statistically significant.

Results.

Demographic data of participants: The age of the enrolled population in the study was 65.6±7.67 years, with majority were overweight (80.4%, n=82), while 19.6% (n=20) were normal BMI. Regarding social status, almost all participants were married (99.0%, n=101), majority of dwelled in urban areas (93.1%, n=95), only 42.2% (n=43) were employed and 57.8% (n=59) were retired. A positive family history for prostate hyperplasia reported in 23.5% (n=24) of patients, drug allergy reported in 11.8% (n=12) of patients, and smokers percentage was low 30.4%, (Table 1).

Renal function tests of prostate treated groups: Regarding renal function tests, urea concentration ranged from 5.50±0.95 mmol/L in the BPH+TD group to 6.85±3.09 mmol/L in the BPH group, (p=0.1514), creatinine concentrations demonstrated the lowest in BPH+TD group (81.54±20.73 μmol/L) and the highest in BPH group (103.58±46.77 μmol/L), (p = 0.2074). Regarding eGFR, the value was highest in BPH+TD group (87.98±14.22 mL/min/1.73m²), lowest in BPH (75.93±19.1 mL/min/1.73m²), (p=0.2929). For uACR, values were non-significantly different (p=0.0731) and ranged from 2.17±1.43 mg/mmol in the BPH+T group to 3.68±2.88 mg/mmol in the BPH group, (Table 2 and Figure 1).

Table 1. *Demographic and clinical status of all participants in the study.*

Demographic and clinical parameters		
Age, years mean and SD		**65.6±7.67**
BMI, n (%)	Normal	20 (19.6%)
	Overweight	82 (80.4%)
Marital status, n (%)	Single	1 (1%)
	Married	101 (99%)
Residency, n (%)	Rural	7 (6.9%)
	Urban	95 (93.1%)
Employee status, n (%)	Retire	59 (57.8%)
	Employee	43 (42.2%)
Drug allergy, n (%)	Yes	12 (11.8%)
	No	90 (88.2%)
Smoking, n (%)	Yes	31 (30.4%)
	No	71 (69.6%)

Table 2. *Kidney function tests in the prostate treated groups compared to control group.*

Kidney parameters	**C (n=26)**	**BPH (n=25)**	**BPH+T (n=26)**	**BPH+TF (n=11)**	**BPH+TD (n=14)**	**P value**
Urea, mmol/L	5.65±1.63	6.85±3.09	6.73±2.01	6.5±2.3	5.5±0.95	0.1514
Creatinine, µmol/L	92.96±25.87	103.58±46.77	87.09±16.09	91.92±27.89	81.54±20.73	0.2074
eGFR, ml/min./1.73m2	80.41±19.49	75.93±19.1	84.15±14.17	81.27±21.34	87.98±14.22	0.2929
uACR, mg/g	2.43±1.49	3.68±2.88	2.17±1.43	2.8±2.16	2.34±1.58	0.0731

The data presented as mean± standard deviation and demonstrated non-significant differences between groups at p value greater than 0.05 using one-way ANOVA followed by post-hoc test at confidence intervals 95%. C=Control group, BPH=Benign prostate hyperplasia group, BPH+T=BPH treated by tamsolusin, BPH+TF=BPH treated by tamsolusin and finasteride, BPH+TD=BPH treated by tamsolusin and dutasteride. eGFR=Estimated glomerular filtration rate, uACR= urine albumin to creatinine ratio.

Table 3. *Liver function tests in the prostate treated groups compared to control group.*

Liver parameters	**C (n=26)**	**BPH (n=25)**	**BPH+T (n=26)**	**BPH+TF (n=11)**	**BPH+TD (n=14)**	**P value**
ALT, U/L	20.47±15.41	18.27±7.41	18.91±13.13	20.4±9.29	19.74±18.25	0.9764
AST, U/L	20.08±7.02	18.42±6.93	19.3±6.88	18.88±5.14	19.4±7.11	0.9378
ALP, U/L	117.22±43.97	134.41±47.62	116.29±32.93	118.85±53.39	125.93±34.56	0.5417
GGT, U/L	27.58±18.72	29.64±18.17	25.39±13.33	19.64±6.93	18.86±7.98	0.1654

Data expressed as mean ±SD, the results demonstrated non-significant differences between groups at p values greater than 0.05 using one-way ANOVA followed by post-hoc test at confidence intervals 95%. C= control group, BPH= Benign prostate hyperplasia group, BPH+T= BPH treated by tamsolusin, BPH+TF= BPH treated by tamsolusin and finasteride, BPH+TD= BPH treated by tamsolusin and dutasteride. ALT=Alanine aminotransferase, AST=Aspartate aminotransferase, ALP=Alkaline Phosphatase, GGT=Gamma-glutamyl transferase.

Table 4. *Liver excretory and synthetic functions parameters in the prostate treated groups compared to control group.*

Bilirubin	**C (n=26)**	**BPH (n=25)**	**BPH+T (n=26)**	**BPH+TF (n=11)**	**BPH+TD (n=14)**	**P value**
Total bilirubin, mg/dL	0.8±0.38	0.9±0.66	0.98±0.44	0.67±0.19	0.64±0.27	0.1314
Direct bilirubin, mg/dL	0.28±0.16	0.26±0.18	0.29±0.15	0.22±0.11	0.26±0.19	0.7964
Indirect bilirubin, mg/dL	0.59±0.27	0.63±0.5	0.69±0.3	0.47±0.15	0.38±0.19	0.0541
Albumin, g/dL	4.22±0.33	4.08±0.43	4.18±0.31	4.2±0.19	4.09±0.46	0.6171

Data expressed as mean±SD, the results demonstrated non-significant differences between groups at p value greater than 0.05 using one-way ANOVA followed by post-hoc test at confidence intervals 95%. C= control group, BPH= Benign prostate hyperplasia group, BPH+T= BPH treated by tamsolusin, BPH+TF= BPH treated by tamsolusin and finasteride, BPH+TD= BPH treated by tamsolusin and dutasteride.

Table 5. *Lipid parameters in the prostate treated groups compared to control group.*

Lipid Parameters	**C (n=26)**	**BPH (n=25)**	**BPH+T (n=26)**	**BPH+TF (n=11)**	**BPH+TD (n=14)**	**P value**
TC, mg/dL	175.11±41.42	187.72±38.71	176.31±36.68	180.59±37.78	196.14±53.88	0.5016
TG, mg/dL	199.58±90.67	185.04±103.17	147.12±82.74	179.13±121.19	198.89±115.53	0.3551
HDL, mg/dL	39.18±9.74	38.33±10.12	38.93±8.35	39.41±12.96	42.76±12.05	0.7627
VLDL, mg/dL	39.61±17.52	37.14±20.74	29.42±16.43	35.86±24.15	39.81±23.17	0.3621
LDL, mg/dL	95.81±39.55	111.5±32.34	107.52±35.12	105.07±40.96	113.05±39.83	0.5454

Data expressed as mean±SD. The results demonstrated non-significant differences between groups at p value greater than 0.05 using one-way ANOVA followed by post-hoc test at confidence intervals 95%. C= control group, BPH= Benign prostate hyperplasia group, BPH+T= BPH treated by tamsolusin, BPH+TF= BPH treated by tamsolusin and finasteride, BPH+TD= BPH treated by tamsolusin and dutasteride. TC= Total cholesterol, TG=Triglyceride, HDL=High density lipoprotein, VLDL=Very low density lipoprotein, LDL=Low density lipoprotein.

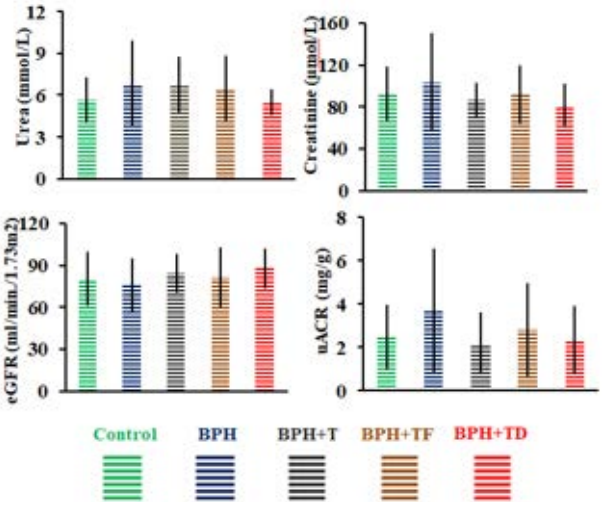

Figure 1. *Renal function parameters in the studied groups. The histogram bar and error bar represents mean±SD. The results demonstrated non-significant differences between groups at p value greater than 0.05 using one-way ANOVA followed by post-hoc test at confidence intervals 95%, the data shown normal distribution using the Shapiro–Wilk test. Control= control group, BPH= Benign prostate hyperplasia group, BPH+T= BPH treated by tamsulosin, BPH+TF= BPH treated by tamsulosin and finasteride, BPH+TD= BPH treated by tamsulosin and dutasteride. eGFR=Estimated glomerular filteration rate, uACR=urine Albumin-Creatinine ratio.*

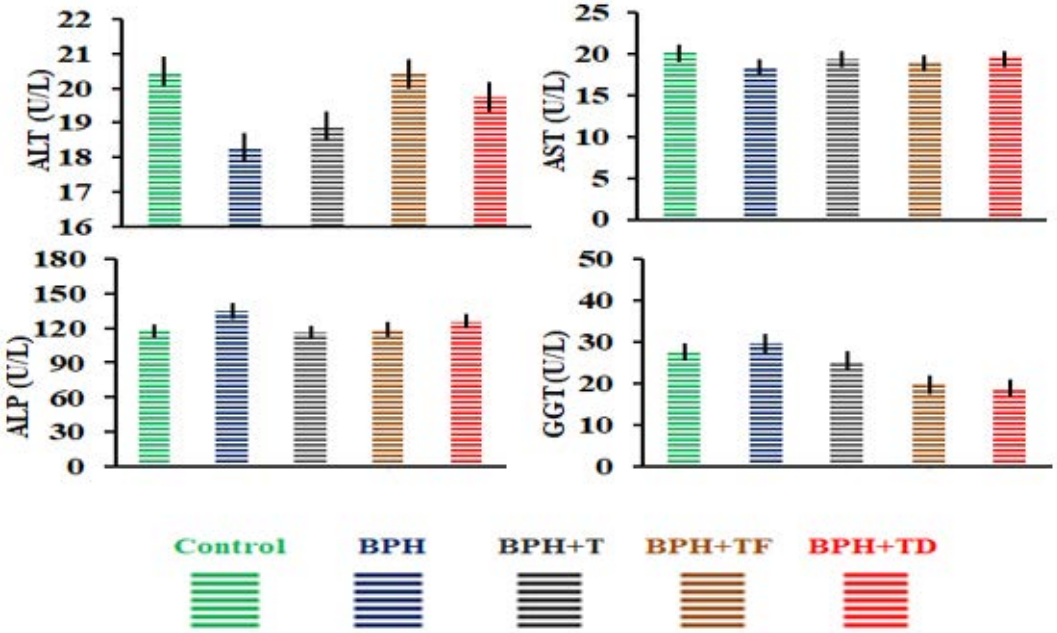

Figure 2. *Liver function parameters in the studied groups. The histogram bar and error bar represents mean±SD. The results demonstrated non-significant differences between groups at p value greater than 0.05 using one-way ANOVA followed by post-hoc test at confidence intervals 95%%, the data shown normal distribution using the Shapiro–Wilk test. Control= control group, BPH= Benign prostate hyperplasia group, BPH+T= BPH treated by tamsulosin, BPH+TF= BPH treated by tamsulosin and finasteride, BPH+TD= BPH treated by tamsulosin and dutasteride. ALT=Alanine aminotransferase, AST=Aspartate aminotransferase, ALP=Alkaline Phosphatase, GGT=Gamma-glutamyl transferase.*

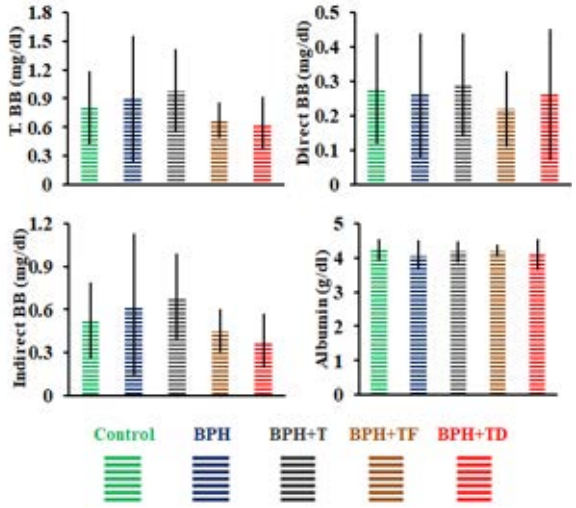

Figure 3. *Adjuvant liver parameters in the studied groups. The histogram bar and error bar represents mean±SD. The results demonstrated non-significant differences between groups at p value greater than 0.05 using one-way ANOVA followed by post-hoc test at confidence intervals 95%%, the data shown normal distribution using the Shapiro–Wilk test. Control= control group, BPH= Benign prostate hyperplasia group, BPH+T= BPH treated by tamsulosin, BPH+TF= BPH treated by tamsulosin and finasteride, BPH+TD= BPH treated by tamsulosin and dutasteride, T. BB=Total Bilirubin.*

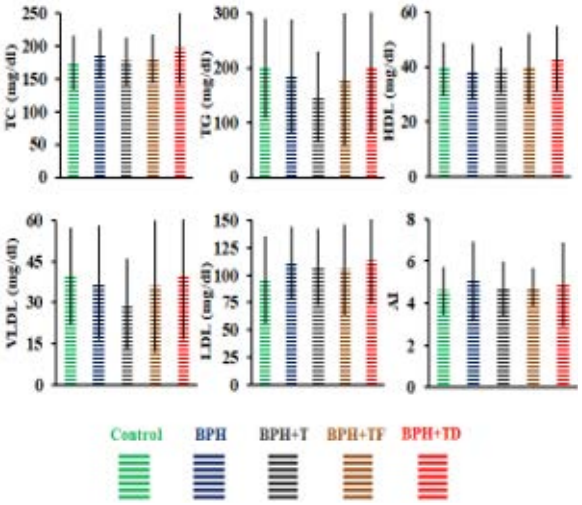

Figure 4. *Lipid profile in the studied groups. The histogram bar and error bar represents mean±SD. The results demonstrated non-significant differences between groups at p value greater than 0.05 using one-way ANOVA followed by post-hoc test at confidence intervals 95%%, the data shown normal distribution using the Shapiro–Wilk test. Control= control group, BPH= Benign prostate hyperplasia group, BPH+T= BPH treated by tamsulosin, BPH+TF= BPH treated by tamsulosin and finasteride, BPH+TD= BPH treated by tamsulosin and dutasteride. TC=Total cholesterol, TG=Triglyceride, HDL=High density lipoprotein, LDL= Low density lipoprotein, VLDL= Very low-density lipoprotein, AI=Atherogenic index.*

Liver function tests of prostate treated groups: The measured liver enzymes demonstrated non-significant ($p > 0.05$) changes among all studied groups. For ALT, the levels ranged from 18.27±7.41 U/L in the BPH group to 20.47±15.41 U/L in the control group, (p=0.9764). Similarly, AST levels remained consistent across all groups, lowest in the BPH group (18.42±6.93 U/L) and highest in control group (20.08±7.02 U/L), (p=0.9378). ALP levels were diverse across groups, with lowest in BPH+T group (116.29±32.93 U/L) and highest in BPH group (134.41±47.62 U/L), (p=0.5417). Regarding GGT, the levels declined in combination therapy [BPH+TF (19.64±6.93 U/L) and BPH+TD (18.86±7.98 U/L)] compared to BPH group (29.64±18.17 U/L), (p=0.1654), (Table 3 and Figure 2).

Liver excretory and synthetic functions tests of prostate treated groups: The measured bilirubin levels demonstrated non-significant ($p>0.05$) changes among all studied groups. For total bilirubin, the levels ranged from 0.64±0.27 mg/dl in the BPH+TD group to 0.98±0.44 mg/dl in the BPH+T group, (p=0.1314). Direct bilirubin levels were nearly stable among all groups, lowest in the BPH+TF group (0.22±0.11 mg/dl) and highest in the BPH+T group (0.29±0.15 mg/dl), (p=0.7964). Indirect bilirubin levels demonstrated a lower concentration in the combination therapy groups, particularly in BPH+TD (0.38±0.19 mg/dl) and BPH+TF (0.47±0.15 mg/dl), compared to the BPH group (0.63±0.5 mg/dl) and BPH+T group (0.69±0.3 mg/dl), (p=0.0541). Albumin remained consistent across all

groups, ranging from 4.08±0.43g/dl in the BPH group to 4.22±0.33 g/dl in the control group, (p=0.6171), (Table 4 and Figure 3).

Lipid profile tests of prostate treated groups: The measured lipid parameters demonstrated non-significant (p>0.05) changes among all studied groups. For TC, the concentrations ranged from 175.11±41.42 mg/dl in the control group to 196.14±53.88 mg/dl in the BPH+TD group, (p=0.5016). The highest level of TG levels were in the control group (199.58±90.67 mg/dl) and lowest in the BPH+T group (147.12±82.74 mg/dl), (p=0.3551). HDL levels remained close to each other's, ranging from 38.33±10.12 mg/dl (BPH) to 42.76±12.05 mg/dl (BPH+TD), (p=0.7627). Similarly, VLDL followed the same pattern, with the lowest concentration in the BPH+T group (29.42±16.43 mg/dl) and the highest in the BPH+TD group (39.81±23.17 mg/dl), (p=0.3621). LDL levels ranged from 95.81±39.55 mg/dl in control group to 113.05±39.83 mg/dl in the BPH+TD group, yielding non-significant differences (p=0.5454). Ultimately, the atherogenic index has shown non-significant changes between studied groups (Table 5 and Figure 4).

Discussion.

The present study characterized the wide range of safety profile represented by moderate lipid profile, liver function tests, and renal function tests. The age of studied groups at sixth and seventh decades, which is consistent with the typical age range for BPH presentation, as BPH incidence increases gradually after age 40 [4,5]. The higher rate of obese patients aligns with earlier epidemiological studies linking obesity to increased BPH risk and symptom severity [6,7]. Adipose tissue contributes to chronic low-grade inflammation and hormonal alterations, including increased estrogen-to-androgen ratios and insulin resistance, which may promote prostatic growth [8,9].

The majority of participants resided in urban areas and were retired, reflecting demographic patterns where higher healthcare access in urban settings may facilitate management [7]. The positive family history in 23.5% of participants supports a hereditary component in BPH pathogenesis, consistent with studies demonstrating familial aggregation of the condition [10-12].

The BPH group showed slightly elevated creatinine compared to controls, but this difference did not reach significance. Similarly, eGFR values were lowest in the BPH group and highest in the BPH+TD group, though not statistically significant.

The preservation of normal renal function parameters across all groups, including untreated BPH patients, suggests that BPH in this study had not progressed to the point of causing significant upper urinary tract dysfunction. Chronic bladder outlet obstruction from BPH can lead to elevated bladder pressures, which may be transmitted to the upper urinary tract, potentially causing hydronephrosis and eventual renal impairment [13,14]. However, this complication typically occurs in advanced or long-standing obstruction, and the relatively preserved renal function in our BPH group suggests early-to-moderate disease severity [2,14].

The absence of significant differences in treatment groups indicates that tamsulosin, finasteride, and dutasteride do not adversely affect renal function. Tamsulosin is primarily metabolized in the liver and does not require dosage adjustment in renal impairment, although it has not been studied in end-stage renal disease [15,16]. The safety profile of these medications with respect to renal function supports their use even in patients with underlying chronic kidney disease, though caution is warranted in severe impairment.

The lipid profile assessment revealed no differences between treatment groups for TC, TG, HDL, VLDL, or LDL. Earlier studies conducted on the role of ARI on lipid parameters have shown discrepant findings. In experimental animal study, mice lacking the 5α-reductase type 1 enzyme (Srd5a1$^{-/-}$mice) demonstrated increased susceptibility to diet-induced weight gain, impaired glucose tolerance, and fatty liver disease [17,18]. Dowman et al (2013) demonstrated that loss of 5α-reductase type 1 accelerates the development of hepatic steatosis in male mice, reflecting that inhibition of this enzyme system has metabolic consequences, particularly with respect to lipid accumulation in the liver and potentially in the vasculature [19]. In a study conducted by Hazlehurst et al (2016) demonstrated that ARI promotes hepatic lipid accumulation, establishing a direct link between these drugs and disturbed lipid metabolism in metabolic tissues [20]. Similarly, Upreti et al (2014) revealed that 5α-reductase type 1 modulates insulin sensitivity in men, an effect that is intrinsically connected to lipid handling [21]. Long-term dutasteride therapy has been shown to increase serum total cholesterol and low-density lipoprotein levels, as reported by Traish et al (2017) [15], providing direct evidence of the adverse effects on circulating cholesterol [16]. The molecular mechanisms of the role of 5α-reductase inhibition to dyslipidaemia, 5α-Reductases facilitate the A-ring reduction of 3-keto, 4 C19/C21 steroids, including cortisol, androgens, progestogens, and mineralocorticoids, all of which have well-recognised effects on energy and vascular homeostasis [22,23]. The reasons of no variation in lipid profile in the present study might be due to the fact that tamsulosin induced opposite effects balancing disturbances of that ARI or due to the fact that the present study sample were metabolically healthy.

The preservation of normal liver enzymes across all treatment groups is clinically important, as tamsulosin is metabolized extensively in the liver via cytochrome P450 enzymes (primarily CYP3A4 and CYP2D6) [24,25]. While severe hepatic impairment is a contraindication to tamsulosin use [26,27], moderate impairment does not substantially alter drug clearance. The GGT values showed a progressive decline from the BPH group to the combination therapy groups. While not statistically significant, this trend may reflect the metabolic effects of 5-ARIs or improved overall health status following symptom relief [28]. Finasteride and dutasteride are also hepatically metabolized [15,29], but have not been associated with significant hepatotoxicity in clinical trials. Similarly, bilirubin parameters—total bilirubin, direct bilirubin, and indirect bilirubin showed no significant differences. The nearly significant trend for indirect bilirubin with lower values in combination therapy groups compared to BPH did not reach statistical significance but may warrant investigation in larger studies.

Several limitations of the present study should be acknowledged. The relatively small sample size, particularly in the BPH+TF (n=11) and BPH+TD (n=14) groups, may

have limited statistical power to insufficiently detect smaller differences between treatment regimens. The cross-sectional design precludes assessment of longitudinal changes in parameters over time. Additionally, the lack of prostate volume measurement by transrectal ultrasound in all patients and the absence of urodynamic studies limit the objective characterization of bladder outlet obstruction severity.

Conclusion.

In summary, tamsulosin administered alone or in combination with finasteride or dutasteride for durations exceeding three months exhibits non-significant differences were in the measured biochemical parameters among the studied groups, including lipid homeostasis, renal function, liver enzyme activities, or bilirubin metabolism. These findings provide reassuring evidences for clinicians prescribing these medications to elderly BPH patients, including those with pre-existing dyslipidemia, chronic kidney disease, or hepatic disorders. However, the cross-sectional design of this study precludes causal inferences regarding long-term effects beyond the three-month treatment period. Future prospective longitudinal studies with larger sample sizes, extended follow-up durations, and inclusion of additional metabolic parameters (such as glycemic indices and inflammatory markers) are warranted to further validate and extend these observations. Nevertheless, the present findings contribute valuable evidence supporting the continued safe use of these widely prescribed therapeutic regimens in the management of benign prostatic hyperplasia.

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