МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
«БУКОВИНСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ»
"BUKOVINIAN STATE MEDICAL UNIVERSITY"
Індексований у міжнародних наукометричних базах:

Academy (Google Scholar)
Ukranian Research&Academy Network (URAN)
Academic Resource Index Research Bib
Index Copernicus International
Scientific Indexing Services
Включений до Ulrichsweb™ Global Serials Directory

КЛІНІЧНА ТА ЕКСПЕРИМЕНТАЛЬНА ПАТОЛОГІЯ
KLINICHNA TA EKSPERIMENTAL'NA PATOLOGIYA
CLINICAL & EXPERIMENTAL PATHOLOGY

На всі статті, опубліковані в журналі «Клінічна та експериментальна патологія», встановлюються цифрові ідентифікатори DOI

Т. XXIII, № 2 (88), 2024

Щоквартальний український науково-медичний журнал. Заснований у квітні 2002 року

Свідоцтво про державну реєстрацію Серія КВ №6032 від 05.04.2002 р. Ідентифікатор медіа R30-03395 (Витяг з Реєстру суб'єктів у сфері медіа-реєстрантів Національної ради України з питань телебачення і радіомовлення від 28.03.2024 № 1037)

Засновник і видавець: Буковинський державний медичний університет, м. Чернівці

Адреса редакції: 58002, Чернівці, пл. Театральна, 2, видавничий відділ БДМУ
Тел./факс: (0372) 553754. E-mail: tkachuk.svitlana14@bsmu.edu.ua; lapagalina46@gmail.com
Офіційний web-сайт журналу: http://cep.bsmu.edu.ua
Електронні копії опублікованих статей передаються до **Національної бібліотеки ім. В.І. Вернадського** для вільного доступу в режимі on-line
Реферати статей публікуються в **"Українському реферативному журналі"**, серія "Медицина"

Наказом Міністерства освіти і науки України від 11.07.2019 р., № 975 журнал «Клінічна та експериментальна патологія» включено до переліку наукових фахових видань України, категорія Б

Рекомендовано до друку та поширення через Інтернет рішенням Вченої ради Буковинського державного медичного університету (протокол № 15 від 25.06.2024 р.)

Матеріали друкуються українською та англійською мовами

Рукописи рецензуються. Редколегія залишає за собою право редагування

Передрук можливий за письмової згоди редколегії

Комп'ютерний набір і верстка – О.Ю. Воронцова

Наукове редагування – редакції

Редагування англійського тексту – Г.М. Лапи

Коректор – І.В. Зінченко

Група технічно-інформаційного забезпечення: І.Б. Горбатюк, Л.І. Сидорчук, В.Д. Сорохан

ISSN 1727-4338
DOI 10.24061/1727-4338.XXIII.2.88.2024

Founded in 2002
Publishing four issues a year

UDC: 616.12-008.331.1-06:616.61-036.12]-008.9:546.41

METABOLIC PATTERN, CHOLECALCIFEROL, PARATHYROID HORMONE AND IONIZED CALCIUM VALUES IN PATIENTS WITH ARTERIAL HYPERTENSION AND CHRONIC KIDNEY DISEASE

B. A. Lytvyn, L. P. Sydorchuk

Bukovinian State Medical University, Chernivtsi, Ukraine

Key words:
arterial hypertension, chronic kidney disease, albuminuria, vitamin D, parathyroid hormone, Cystatin-C, lipids, HDL-C

Clinical and experimental pathology 2024. Vol.23, №2 (88). C. 94-99.

DOI 10.24061/1727-4338. XXIII.2.88.2024.15

E-mail:
dr.bogdan@ukr.net

Arterial Hypertension (AH), Diabetes Mellitus along with other metabolic disorders are among the leading causes of kidney damage worldwide.

***Objective** – to investigate the metabolic pattern, cholecalciferol and ionized calcium blood values in patients with essential AH (EAH) and chronic kidney disease (CKD) depending on gender.*

***Material and methods.** 100 EAH patients and 60 practically healthy individuals participated in the study. All participant underwent a complex of clinical and laboratory examinations. Metabolism was analyzed by the lipid profile (total cholesterol (TC), high-, low density lipoproteins (HDL-C, LDL-C), atherogenity index (AI), triacylglycerols (TG)), blood glucose, creatinine, cystatin-C, bilirubin, cholecalciferol, ionized calcium, parathyroid hormone (PTH) and albuminuria values. Body mass index (BMI), waist circumference (WC), hip circumference (HC) and waist to hip ratio (WHR) were also determined.*

***Results.** The CKD appearance in EAH patients is accompanied by a higher blood pressure (BP) values (systolic BP – 5.98 % (p=0.012), diastolic BP – 5.57 % (p=0.007)), mainly in women – 6.34 % (p=0.008) and 5.81 % (p=0.013), respectively; a higher bilirubin blood concentration – 27.79 % (p=0.007), creatinine and cystatin-C regardless of gender (stronger in men) – by 21.76-26.67 % (p<0.001), glucose (significantly in men) – 33.49 % (p=0.035), with a lower level of ionized Ca2+ (p=0.05), HDL-C – 16.81 % (p=0.02), which cause an increase of the AI (reliably in men) – 35.36 % (p=0.027); higher BMI – 24.67 % (p=0.003) and WC – 14.97 % (p=0.023) in men. Also, in EAH patients with CKD, more massive albuminuria is observed in general – 32.45 % (p=0.02), reliably only in women – 44.20 % (p=0.004). Changes in the vitamin D metabolites concentration and PTH were not associated with CKD in EAH patients in our study.*

***Conclusion.** A proatherogenic pattern, hyperglycemia, excess body weight/or obesity (mainly in men), albuminuria and hemodynamic disorders (mainly in women) generally form metabolic changes in EAH patients with CKD.*

Ключові слова:
артеріальна гіпертензія, хронічна хвороба нирок, альбумінурія, вітамін Д, паратгормон, цистатин-С, ліпіди, ХС ЛПВЩ.

Клінічна та експериментальна патологія 2024. Т.23, №2 (88). С. 94-99.

МЕТАБОЛІЧНИЙ ПАТЕРН, РІВНІ ХОЛЕКАЛЬЦИФЕРОЛУ, ПАРАТГОРМОНУ ТА ІОНІЗОВАНОГО КАЛЬЦІЮ У ХВОРИХ НА АРТЕРІАЛЬНУ ГІПЕРТЕНЗІЮ І ХРОНІЧНУ ХВОРОБУ НИРОК

Б. А. Литвин, Л. П. Сидорчук

Буковинський державний медичний університет, м. Чернівці, Україна

Артеріальна гіпертензія (АГ), цукровий діабет поряд з іншими метаболічними розладами є одними з провідних причин ураження нирок у світі.

***Мета роботи –** дослідити метаболічний патерн, рівні холекальциферолу, паратгормону та іонізованого кальцію у хворих на есенційну артеріальну гіпертензію (ЕАГ) і хронічну хворобу нирок (ХХН) з урахуванням статі.*

***Матеріали і методи.** У дослідженні взяли участь 100 хворих на ЕАГ і 60 практично здорових осіб групи контролю, які пройшли комплекс клінічно-лабораторних обстежень. Метаболізм аналізували за ліпідним профілем (загальним холестеролом (ЗХС), ліпопротеїнами високої і низької щільностей (ХС ЛПВЩ, ХС ЛПНЩ), коефіцієнтом атерогенності (КА), триацилгліцеролами (ТГ)), вмістом у крові глюкози, креатиніну, цистатину-С, білірубіну, холекальциферолу, іонізованого кальцію, паратгормону (ПТГ) та альбумінурією. Також визначали індекс маси тіла (ІМТ), обвід талії (ОТ), стегон (ОС), їх співвідношення (ОТ/ОС).*

***Результати.** Поява ХХН у хворих на ЕАГ супроводжується вищим рівнем артеріального тиску (АТ) (систолічного АТ – на 5,98 % (p=0,012), діастолічного АТ – на 5,57 % (p=0,007)), переважно у жінок – на 6,34 % (p=0,008) і 5,81 % (p=0,013) відповідно; вищою концентрацією у крові білірубіну – на 27,79 % (p=0,007), креатиніну та цистатину-С незалежно від статі (суттєвіше у чоловіків) – на 21,76-26,67 % (p<0,001), глюкози (вірогідно у чоловіків) – на 33,49 % (p=0,035), за нижчого рівня іонізованого Ca2+ (p=0,05), ХС ЛПВЩ – на 16,81 % (p=0,02), що зумовило збільшення*

коефіцієнту атерогенності (вірогідно у чоловіків) – 35,36 % (p=0,027); більшим ІМТ – на 24,67 % (p=0,003) і ОТ – на 14,97 % (p=0,023) у чоловіків. Також при ХХН у хворих на ЕАГ спостерігається масивніша альбумінурія загалом – на 32,45 % (p=0,02), достовірно тільки у жінок – на 44,20 % (p=0,004). Зміни концентрації сумарних метаболітів вітаміну D і паратгормону не асоціюють із ХХН у пацієнтів із ЕАГ у нашому дослідженні.

***Висновок**. Проатерогенний патерн, гіперглікемія, надмірна маса тіла /чи ожиріння (переважно у чоловіків), альбумінурія і гемодинамічні розлади (переважно у жінок) формують загалом зміни метаболізму при ХХН у хворих на ЕАГ.*

Introduction

Arterial Hypertension (AH) is a primary risk factor for cardiovascular disease and global mortality. There is a close interrelation between AH and chronic kidney disease (CKD), as hypertension can lead to declining renal function, while progressive CKD can exacerbate hypertension [3, 4, 14]. The pathophysiology of AH in CKD is intricate and involves multiple factors, such as a decreased number of functioning nephrons, sodium retention, volume expansion, activation of the sympathetic nervous system, hormonal influences like the renin-angiotensin-aldosterone system upregulation, and endothelial dysfunction [7, 12].

Blood pressure (BP) variability in the chronic kidney disease (CKD) population is significant and requires close monitoring for appropriate management. With accumulating evidence, the diagnosis and management of AH in CKD and vice versa have been evolving over the last decade. The Kidney Disease: Improving Global Outcomes (KDIGO) Organization in 2024 upgraded Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease [8].

Diabetes Mellitus (DM), other metabolic disorders in addition to AH are among the leading causes of kidney damage worldwide. Essential AH (EAH) and diabetes account for 63 % of all CKD cases [8]. In the American population, CKD is registered in 23 % of hypertensive patients [22], in the Italian population – in 42 % likewise [9]. Thus, the problem of early diagnosis of genetic, cardiometabolic, and immunological causes of CKD development in EAH patients in the cardiovascular diseases' continuum is on cutting edge and requires further investigation.

Objective

To investigate the metabolic pattern, cholecalciferol, parathyroid hormone and ionized calcium blood values in patients with EAH and CKD depending on gender.

Research material and methods

The EAH patients were selected in accordance with the guidelines and recommendations of the National Ukrainian and European Societies of Cardiology and Hypertension (ESC, ESH 2018, 2023) [1, 12].

The research protocol was approved by the Bioethics commission at the Bukovinian State Medical University. 100 patients with Hypertension-mediated organ damage (IInd stage), 1st-3rd degrees of arterial blood pressure (BP) elevation, moderate, high, or very high cardiovascular risk were screened and selected for the study. The patients age ranged from 45 to 70 years (59.87±7.98 years on average), 21.0 % of them were men, 79.0 % were women. The control group consisted of 60 practically healthy people (22 men (36.67 %), 38 women (63.33 %)), aged 44.39±5.92 years ($p<0.001$). The groups did not differ by sex. All participants signed an informed consent to participate in the study.

Comprehensive examination included: anthropometric (body mass index, (BMI), waist and hip circumference (WC, HC), waist-to-hip ratio (WHR)), general clinical, laboratory (general blood and urine tests, urine protein, fasting plasma glucose, serum creatinine, cystatin-C, bilirubin, lipid spectrum), instrumental (12-lead ECG, Echocardiogram, office BP, ultrasound of the kidneys), as well as consultations of an ophthalmologist and a neurologist, if necessary. Besides, the level of ionized calcium was determined in all patients by the potentiometric method using the blood electrolyte analyzer SINO 005 (Sinnowa, China). The lipid panel was studied using colorimetry after serum content of total cholesterol (TC), triacylglycerols (TG) and high-density lipoprotein cholesterol (HDL-C). Serum low-density lipoprotein cholesterol (LDL-C) was estimated by the Friedewald equation, atherogenicity index (AI) – according to Klimov's equation [5, 10, 18, 19, 21].

The parathyroid hormone (PTH) and cholecalciferol (the total metabolites of 25(OH) vitamin D) blood concentration were determined by the immunochemiluminescence method according to the manufacturer's instructions using the device «MAGLUMI 1000 Plus» («SNIBE», China) [20].

CKD was diagnosed in 43 EAH patients according to the US National Kidney Association recommendations (KDIGO 2024) [8]. Glomerular filtration rate (GFR) was calculated using the CKD-EPI equation based on cystatin-C and creatinine serum values (depending on gender). There were 35 women (44.30 %) and 8 men (38.10 %) among EAH patients with CKD. A decrease in GFR was defined as ≤60 ml/min/1.73m2, for ≥3 months with or without other signs of kidney damage, according to the KDIGO recommendations (2024) [8].

The statistical analysis was carried out by the variational statistics methods using the Statistica v.7.0 software (StatSoft Inc., USA). The differences between groups for independent samples were verified using the unpaired Student's t-test (if the data distribution were close to normal according to the Kolmogorov-Smirnov tests and the Shapiro-Wilk W-test), or the Wilcoxon-Mann-Whitney U-test (for an uneven data distribution). Differences were considered significant at $p<0.05$.

Results and Discussion

In EAH patients with CKD (GFR ≤60 ml/ min/1.73m^2) higher systolic and diastolic BP (SBP, DBP) were

established – 5.98 % (p_1=0.012) and 5.57 % (p_1=0.007), mostly in women: SBP – 6.34 % (p_1=0.008), DBP – 5.81 % (p_1=0.013), respectively (Tab.1). Some anthropometric parameters also prevailed in EAH patients with CKD, over those without CKD, but only in men: for BMI – 24.67 % (p_1=0.003), for WC – 14.97 % (p_1=0.023). Most anthropometric data in men with CKD exceed those in women with CKD. SBP and DBP in practically healthy subjects of control group, in contrast to patients, are higher in men than in women – 3.76 % (p_W=0.036) and 6.58 % (p_W=0.035), as well as WC and WHR – 21.96 % and 20.51 % (p_W<0.001).

Table 1

Some clinical and anthropometric parameters depending on glomerular filtration rate according to Cystatin-C and gender

Parameters		Control group, n=60	Patients (study group), n=100	
			GFR ≤60 ml/min/1.73m², n=43	GFR >60 ml/min/1.73m², n=57
BMI, kg/m²	W	25.18±1.15	30.96±1.45 p<0.001	31.45±1.38 p<0.001
	M	27.02±0.83	35.58±1.43 p<0.001; p_W=0.028	28.54±0.66 p_1=0.003; p_W=0.01
SBP, mm Hg		116,33±2,36	158.97±4.48 p<0.001	150.0±3.57 p<0.001; p_1=0.012
DBP, mm Hg		76,0±2,58	98.12±2.80 p<0.001	92.94±1.89 p<0.001; p_1=0.007
WC, см	W	78.79±2.93	98.17±2.40 p<0.001	100.67±2.97 p<0.001
	M	96.09±2.24 P_W<0.001	115.50±4.41 p=0.007; p_W=0.002	100.46±2.60 p_1=0.023
WHR, yo	W	0.78±0.02	0.88±0.01 p<0.001	0.90±0.02 p<0.001
	M	0.94±0.02 p_W<0.001	1.0±0.02 P_W<0.001	0.97±0.02 p_W=0.004

Note. P – probability of data differences with the control group; P_1 – probability of differences with patients whose GFR ≤60 ml/min/1.73m2; Pw – probability of differences between women and men within each subgroup according to a particular indicator; BMI – body mass index; SBP, DBP – systolic, diastolic blood pressure; WC – waist circumference; WHR – Waist-to Hip Ratio; W – women; M – men

EAH patients with CKD have a higher blood creatinine and Cystatin-C values, regardless of gender, more in men – 21.76-26.67 % (p_1<0.001), as well as more massive albuminuria in general – 32.45 % (p=0.02), but reliably only in women – 44.20 % (p=0.004).

The glucose blood concentration is higher in EAH patients with CKD (GFR ≤60 ml/min/1.73m2) than without CKD, but only in men – 33.49 % (p_1=0.035), and the content of HDL-C on the contrary is lower – 16.81 % (p_1=0.02), which led to an increase in AI in general 13.29 % (p_1=0.055), significantly in men – 35.36 % (p_1=0.027). Most parameters of lipid-carbohydrate metabolism in patients deviated from reference values and those in controls (glucose, TG in women, AI and HDL-C) (Tab. 2).

Table 2

Parameters of lipids metabolism and glucose level depending on glomerular filtration rate according to Cystatin-C and gender

Parameters		Control group, n=60	Patients after GFR value, n=100	
			≤60 ml/min/1.73m², n=43	>60 ml/min/1.73m², n=57
Blood glucose, mmol/l	W	5.0±0.17	7.45±0.85 p=0.005	7.47±0.82 p=0.002
	M	5.28±0.13	8.65±0.84 p<0.001	6.48±0.45 p=0.017; p_1=0.035
Total cholesterol, mmol/l		5.55±0.22	5.63±0.32	5.64±0.29
Triacylglycerols, mmol/l		1.64±0.17	2.19±0.25 p=0.019	1.87±0.23
LDL-C, mmol/l		3.95±0.22	4.18±0.29	4.17±0.26
HDL-C, mmol/l	W	1.53±0.07	1.28±0.07 p=0.014	1.31±0.06 p=0.005
	M	1.24±0.09 p_w=0.043	0.99±0.05 p=0.024; p_w=0.009	1.19±0.06 p_1=0.02
AI, U		3.18±0.36	3.92±0.38 p=0.04	3.46±0.23 p_1=0.055

Note. P – probability of data differences with the control group; P_1 – probability of differences with patients whose GFR ≤60 ml/min/1.73m²; Pw – probability of differences between women and men within each subgroup according to a particular indicator; W – women; M – men; AI – atherogenicity index

The cholecalciferol concentration in EAH patients is lower than in the control group, regardless of GFR values, but reliably only in women – 11.76 % (p=0.022) and 10.0 % (P=0.048) with higher PTH content mainly in women – 24.61-30.64 % (p≤0.046-0.022). The level of ionized calcium (Ca^{2+}) in EAH patients with CKD (GFR ≤60 ml/min/1.73m2) is generally marginally lower than in those without CKD (p=0.05) (Tab. 3).

The kidney performs numerous functions, including excretory, endocrine, and metabolic roles. GFR is a key indicator of excretory function and is widely accepted as the best overall measure of kidney function. GFR typically decreases following extensive structural damage to the kidneys, and most other kidney functions decline in parallel with GFR in CKD. Metabolic disorders are pathological conditions where multiple risk factors for CKD, such as obesity, hypertension, hyperglycemia and dyslipidemia (characterized by high triglycerides and low HDL-C), coexist in individuals [2, 15, 25]. The incidence of metabolic disorders is on the rise as well as the prevalence of CKD has shown a steady annual increase in recent years. This trend poses a serious threat to life and health, imposes economic burdens on society and families, and diminishes the quality of life for those affected [8, 16].

Table 3

Parameters of metabolic-hormonal activity and ionized calcium depending on glomerular filtration rate according to Cystatin-C and gender

Parameters	Control group, n=60	Patients (study group), n=100	
		GFR ≤60 ml/min/1.73m^2, n=43	GFR >60 ml/min/1.73m^2, n=57
Total metabolites of vitamin D, ng/ml	23.86±1.26	21.69±1.43 p=0.048	21.35±1.25 p=0.022
Parathyroid hormone, pg/ml	57.76±3.48	72.58±7.64 p=0.025	75.46±8.77 p=0.022
Ionized calcium, mmol/l	1.16±0.01	1.16±0.009	1.18±0.007 p=0.052 p_1=0.05

Note. P – probability of data differences with the control group; P_1 – probability of differences with patients whose GFR ≤60 ml/min/1.73m^2

Xiao H. et al. proved that metabolic syndrome as its single or combined components are independently associated with CKD in populations of Southern China aged 40 years and older, in both non-diabetic and non-obese subjects [23]. In our study the EAH and CKD comorbidity also associate with metabolic and electrolyte changes: higher blood level of bilirubin, creatinine and Cystatin-C (more in men) – 21.76-26.67 %, atherogenicity index – 35.36 %, with lower ionized Ca^{2+} and HDL-C – 16.81 %.

Non-communicable diseases and conditions such as hypertension [11], diabetes [4] or insulin resistance [17], obesity [6] and dyslipidemia [13] are also risk factors for CKD [24]. In our study EAH patients with CKD had higher SBP and DBP (significantly in women) – 6.34 % and 5.81 % and higher BMI 24.67 %, WC – 14.97 % and glucose – 33.49 % as well, than those without CKD.

There are numerous kidney disorders that may manifest with tubular dysfunction rather than only a decrease in GFR. These conditions primarily lead to symptoms such as polyuria, albuminuria, and/or electrolyte imbalances, and they may or may not progress to reduced GFR or chronic kidney failure. Therefore, relying solely on GFR for diagnosing CKD would not be sufficient. It is crucial to recognize and utilize various markers that reflect different aspects of kidney function to obtain a comprehensive diagnosis. Therefore, in our research we rely upon not exclusively on GFR in diagnosing CKD, but use albuminuria level and electrolyte imbalance as well.

Thus, understanding the linkage between metabolic disorders and CKD, as well as implementing effective intervention measures to control them, will help alleviate the burden that chronic diseases affect medical resources worldwide.

Conclusions

1. In patients with essential arterial hypertension (EAH), the onset of chronic kidney disease (CKD) is accompanied by a higher level of blood pressure, mainly in women – 6.34 % and 5.81 %; higher BMI – 24.67 % and WC – 14.97 % in men; higher blood concentration of bilirubin – 27.79 %, creatinine and Cystatin-C regardless of gender (stronger in men) – 21.76-26.67 %, glucose (more in men) – 33.49 %, with lower level of ionized Ca^{2+}, HDL cholesterol – 16.81 %, higher atherogenicity index (reliably in men) – 35.36 %.

2. The presence of CKD in EAH patients is characterized by more massive albuminuria in general – 32.45 %, but reliably only in women – 44.20 %.

Prospects for further research

We see the prospects for further research in the study of metabolic changes in EAH patients taking into account salt sensitivity / salt resistance and genetic factors.

Список літератури

1. Williams B, Mancia G, Spiering W, Agabiti Rosei E, Azizi M, Burnier M, et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. The Task Force for the management of arterial hypertension of the European Society of Cardiology (ESC) and the European Society of Hypertension (ESH). Eur Heart J. 2018;39(33):3021-104. doi: 10.1093/eurheartj/ehy339
2. Comini LO, de Oliveira LC, Borges LD, Dias HH, Batistelli CRS, da Silva LS, et al. Individual and combined components of metabolic syndrome with chronic kidney disease in individuals with hypertension and/or diabetes mellitus accompanied by primary health care. Diabetes Metabol Syndr Obes. 2020;13:71-80. doi: 10.2147/dmso.s223929
3. De Bhailis ÁM, Kalra PA. Hypertension and the kidneys. Br J Hosp Med (Lond). 2022;83(5):1-11. doi: 10.12968/hmed.2021.0440
4. Duan J, Wang C, Liu D, Qiao Y, Pan S, Jiang D, et al. Prevalence and risk factors of chronic kidney disease and diabetic kidney disease in Chinese rural residents: a cross-sectional survey. Sci Rep [Internet]. 2019[cited 2024 Jun 10];9(1):10408. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6639314/pdf/41598_2019_Article_46857.pdf doi: 10.1038/s41598-019-46857-7
5. Dzhuryak V, Sydorchuk L, Sydorchuk A, Kamyshnyi O, Kshanovska A, Levytska S, et al. The cytochrome 11B2 aldosterone synthase gene CYP11B2 (RS1799998) polymorphism associates with chronic kidney disease in hypertensive patients. Biointerface Research in Applied Chemistry. 2020;10(3):5406-11. doi: 10.33263/BRIAC103.406411
6. Garofalo C, Borrelli S, Minutolo R, Chiodini P, De Nicola L, Conte G. A systematic review and meta-analysis suggests obesity predicts onset of chronic kidney disease in the general population. Kidney Int. 2017;91(5):1224-35. doi: 10.1016/j.kint.2016.12.013
7. Gupta A, Nagaraju SP, Bhojaraja MV, Swaminathan SM, Mohan PB. Hypertension in Chronic Kidney Disease: An Update on Diagnosis and Management. South Med J. 2023;116(2):237-44. doi: 10.14423/smj.0000000000001516
8. CKD Work Group. KDIGO 2024 clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney Int. 2024;105(Suppl 4): S117–S314. doi: 10.1016/j.kint.2023.10.018
9. Leoncini G, Viazzi F, Agabiti Rosei E, Ambrosioni E, Costa FV, Leonetti G, et al. Metabolic syndrome and chronic kidney disease in high-risk Italian hypertensive patients: the I-DEMAND study. J Nephrol. 2012;25(1):63-74. doi: 10.5301/jn.2011.7752
10. Литвин БА, Сидорчук ЛП, Яринич ЮМ, Каражбей ІС, Гутніцька АФ. Частота загострень артеріальної гіпертензії, депресія і тривога в умовах війни в Україні: окрема статистика та результати опитування. Буковинський медичний вісник. 2023;27(4):58-62. doi: 10.24061/2413-0737.27.4.108.2023.11

11. Maeda T, Yoshimura C, Takahashi K, Ito K, Yasuno T, Abe Y, et al. Usefulness of the blood pressure classification in the new 2017 ACC/AHA hypertension guidelines for the prediction of new-onset chronic kidney disease. J Hum Hypertens. 2019;33(12):873-8. doi: 10.1038/s41371-019-0198-7

12. Mancia G, Kreutz R, Brunström M, Burnier M, Burnier M, Grassi G, Januszewicz A, et al. 2023 ESH Guidelines for the management of arterial hypertension The Task Force for the management of arterial hypertension of the European Society of Hypertension: Endorsed by the International Society of Hypertension (ISH) and the European Renal Association (ERA). J Hypertens. 2023;41(12):1874-2071. doi: 10.1097/hjh.0000000000003480

13. Miljkovic M, Stefanovic A, Simic-Ogrizovic S, Vekic J, Bogavac-Stanojevic N, Cerne D, et al. Association of dyslipidemia, oxidative stress, and inflammation with redox status in VLDL, LDL, and HDL lipoproteins in patients with renal disease. Angiology. 2018;69(10):861-70. doi: 10.1177/0003319718780041

14. Mustedanagic M, Paponja K, Jelakovic B, Stambuk SK. Prevalence and control of arterial hypertension among patients with glomerular diseases. J Hypertens. 2024;42(Suppl 1):211-2. doi: 10.1097/01.hjh.0001021604.23865.04

15. Pammer LM, Lamina C, Schultheiss UT, Kotsis F, Kollerits B, Stockmann H, et al. Association of the metabolic syndrome with mortality and major adverse cardiac events: a large chronic kidney disease cohort. J Intern Med. 2021;290(6):1219-32. doi: 10.1111/joim.13355

16. Saran R, Robinson B, Abbott KC, Bragg-Gresham J, Chen X, Gipson D, et al. US renal data system 2019 annual data report: epidemiology of kidney disease in the United States. Am J Kidney Dis. 2020;75(Suppl 1): A6-7. doi: 10.1053/j.ajkd.2019.09.003

17. Spoto B, Pisano A, Zoccali C. Insulin resistance in chronic kidney disease: a systematic review. Am J Physiol Renal Physiol. 2016;311(6): F1087-108. doi: 10.1152/ajprenal.00340.2016

18. Sydorchuk A, Sydorchuk L, Gutnitska A, Margaryan A, Dzhuryak V, Sydorchuk R, et al. Linkage of Metabolic Disorders, Endothelial Dysfunction and NOS3 (rs2070744) and GNB3 (rs5443) Genes Polymorphisms in Hypertensive Patients. Biointerface Research in Applied Chemistry Open-Access Journal. 2023;13(2):1-12. doi: 10.33263/BRIAC132.123

19. Sydorchuk LP, Dzhuryak VS, Sydorchuk AR, Levytska SA, Knut RP, Sokolenko MO, et al. Association of lipids' metabolism disorders with aldosterone synthase CYP11B2 (–344C/T) gene polymorphism in hypertensive patients depending on glomerular filtration rate. PharmacologyOnLine. 2020;2:230-42.

20. Sydorchuk A, Sydorchuk L, Tymkul D. The cohort study of adipocytes' secretory activity, vitamin d, parathyroid hormone, leptin resistance and obesity in hypertensive patients. J Hypertens. 2024;42(Suppl 1):225. doi: 10.1097/01.hjh.0001021760.19349.5e

21. Sydorchuk L, Dzhuryak V, Sydorchuk A, Levytska S, Petrynych V, Knut R, et al. The cytochrome 11B2 aldosterone synthase gene rs1799998 single nucleotide polymorphism determines elevated aldosterone, higher blood pressure, and reduced glomerular filtration, especially in diabetic female patients. Endocr Regul. 2020;54(3):217-26. doi: 10.2478/enr-2020-0024

22. Virani SS, Alonso A, Benjamin EJ, Bittencourt MS, Callaway CW, Carson AP, et al. Heart Disease and Stroke Statistics-2020 Update: A Report From the American Heart Association. Circulation [Internet]. 2020[cited 2024 Jun 10];141(9): e139-e596. Available from: https://www.ahajournals.org/doi/epub/10.1161/CIR.0000000000000757 doi: 10.1161/cir.0000000000000757

23. Xiao H, Shao X, Gao P, Zou H, Zhang X. Metabolic Syndrome Components and Chronic Kidney Disease in a Community Population Aged 40 Years and Older in Southern China: A Cross-Sectional Study. Diabetes Metab Syndr Obes. 2022;15:839-48. doi: 10.2147/DMSO.S353305

24. Xie K, Bao L, Jiang X, Ye Z, Bing J, Dong Y, et al. The association of metabolic syndrome components and chronic kidney disease in patients with hypertension. Lipids Health Dis [Internet]. 2019[cited 2024 Jun 10];18(1):229. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6935087/pdf/12944_2019_Article_1121.pdf doi: 10.1186/s12944-019-1121-5

25. Yun HR, Kim H, Park JT, Chang TI, Yoo TH, Kang SW, et al. Obesity, metabolic abnormality, and progression of CKD. Am J Kidney Dis. 2018;72(3):400-10. doi: 10.1053/j.ajkd.2018.02.362

References

1. Williams B, Mancia G, Spiering W, Agabiti Rosei E, Azizi M, Burnier M, et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. The Task Force for the management of arterial hypertension of the European Society of Cardiology (ESC) and the European Society of Hypertension (ESH). Eur Heart J. 2018;39(33):3021-104. doi: 10.1093/eurheartj/ehy339

2. Comini LO, de Oliveira LC, Borges LD, Dias HH, Batistelli CRS, da Silva LS, et al. Individual and combined components of metabolic syndrome with chronic kidney disease in individuals with hypertension and/or diabetes mellitus accompanied by primary health care. Diabetes Metabol Syndr Obes. 2020;13:71-80. doi: 10.2147/dmso.s223929

3. De Bhailis ÁM, Kalra PA. Hypertension and the kidneys. Br J Hosp Med (Lond). 2022;83(5):1-11. doi: 10.12968/hmed.2021.0440

4. Duan J, Wang C, Liu D, Qiao Y, Pan S, Jiang D, et al. Prevalence and risk factors of chronic kidney disease and diabetic kidney disease in Chinese rural residents: a cross-sectional survey. Sci Rep [Internet]. 2019[cited 2024 Jun 10];9(1):10408. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6639314/pdf/41598_2019_Article_46857.pdf doi: 10.1038/s41598-019-46857-7

5. Dzhuryak V, Sydorchuk L, Sydorchuk A, Kamyshnyi O, Kshanovska A, Levytska S, et al. The cytochrome 11B2 aldosterone synthase gene CYP11B2 (RS1799998) polymorphism associates with chronic kidney disease in hypertensive patients. Biointerface Research in Applied Chemistry. 2020;10(3):5406-11. doi: 10.33263/BRIAC103.406411

6. Garofalo C, Borrelli S, Minutolo R, Chiodini P, De Nicola L, Conte G. A systematic review and meta-analysis suggests obesity predicts onset of chronic kidney disease in the general population. Kidney Int. 2017;91(5):1224-35. doi: 10.1016/j.kint.2016.12.013

7. Gupta A, Nagaraju SP, Bhojaraja MV, Swaminathan SM, Mohan PB. Hypertension in Chronic Kidney Disease: An Update on Diagnosis and Management. South Med J. 2023;116(2):237-44. doi: 10.14423/smj.0000000000001516

8. CKD Work Group. KDIGO 2024 clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney Int. 2024;105(Suppl 4): S117–S314. doi: 10.1016/j.kint.2023.10.018

9. Leoncini G, Viazzi F, Agabiti Rosei E, Ambrosioni E, Costa FV, Leonetti G, et al. Metabolic syndrome and chronic kidney disease in high-risk Italian hypertensive patients: the I-DEMAND study. J Nephrol. 2012;25(1):63-74. doi: 10.5301/jn.2011.7752

10. Lytvyn BA, Sydorchuk LP, Yarynych YuM, Karazbey IS, Gutnitska AF. Chastota zahostren' arterial'noi hipertenzii, depresiia i tryvoha v umovakh viiny v Ukraini: okrema statystyka ta rezul'taty opytuvannia [Arterial hypertension exacerbation frequencies, depression and anxiety in the condition of war in Ukraine: some statistics and survey results]. Bukovinian Medical Herald. 2023;27(4):58-62. doi: 10.24061/2413-0737.27.4.108.2023.11 (in Ukranian)

11. Maeda T, Yoshimura C, Takahashi K, Ito K, Yasuno T, Abe Y, et al. Usefulness of the blood pressure classification in the new 2017 ACC/AHA hypertension guidelines for the prediction of new-onset chronic kidney disease. J Hum Hypertens. 2019;33(12):873-8. doi: 10.1038/s41371-019-0198-7

12. Mancia G, Kreutz R, Brunström M, Burnier M, Burnier M, Grassi G, Januszewicz A, et al. 2023 ESH Guidelines for the management of arterial hypertension The Task Force for the management of

arterial hypertension of the European Society of Hypertension: Endorsed by the International Society of Hypertension (ISH) and the European Renal Association (ERA). J Hypertens. 2023;41(12):1874-2071. doi: 10.1097/hjh.0000000000003480

13. Miljkovic M, Stefanovic A, Simic-Ogrizovic S, Vekic J, Bogavac-Stanojevic N, Cerne D, et al. Association of dyslipidemia, oxidative stress, and inflammation with redox status in VLDL, LDL, and HDL lipoproteins in patients with renal disease. Angiology. 2018;69(10):861-70. doi: 10.1177/0003319718780041

14. Mustedanagic M, Paponja K, Jelakovic B, Stambuk SK. Prevalence and control of arterial hypertension among patients with glomerular diseases. J Hypertens. 2024;42(Suppl 1):211-2. doi: 10.1097/01.hjh.0001021604.23865.04

15. Pammer LM, Lamina C, Schultheiss UT, Kotsis F, Kollerits B, Stockmann H, et al. Association of the metabolic syndrome with mortality and major adverse cardiac events: a large chronic kidney disease cohort. J Intern Med. 2021;290(6):1219-32. doi: 10.1111/joim.13355

16. Saran R, Robinson B, Abbott KC, Bragg-Gresham J, Chen X, Gipson D, et al. US renal data system 2019 annual data report: epidemiology of kidney disease in the United States. Am J Kidney Dis. 2020;75(Suppl 1): A6-7. doi: 10.1053/j.ajkd.2019.09.003

17. Spoto B, Pisano A, Zoccali C. Insulin resistance in chronic kidney disease: a systematic review. Am J Physiol Renal Physiol. 2016;311(6): F1087-108. doi: 10.1152/ajprenal.00340.2016

18. Sydorchuk A, Sydorchuk L, Gutnitska A, Margaryan A, Dzhuryak V, Sydorchuk R, et al. Linkage of Metabolic Disorders, Endothelial Dysfunction and NOS3 (rs2070744) and GNB3 (rs5443) Genes Polymorphisms in Hypertensive Patients. Biointerface Research in Applied Chemistry Open-Access Journal. 2023;13(2):1-12. doi: 10.33263/BRIAC132.123

19. Sydorchuk LP, Dzhuryak VS, Sydorchuk AR, Levytska SA, Knut RP, Sokolenko MO, et al. Association of lipids' metabolism disorders with aldosterone synthase CYP11B2 (–344C/T) gene polymorphism in hypertensive patients depending on glomerular filtration rate. PharmacologyOnLine. 2020;2:230-42.

20. Sydorchuk A, Sydorchuk L, Tymkul D. The cohort study of adipocytes' secretory activity, vitamin d, parathyroid hormone, leptin resistance and obesity in hypertensive patients. J Hypertens. 2024;42(Suppl 1):225. doi: 10.1097/01.hjh.0001021760.19349.5e

21. Sydorchuk L, Dzhuryak V, Sydorchuk A, Levytska S, Petrynych V, Knut R, et al. The cytochrome 11B2 aldosterone synthase gene rs1799998 single nucleotide polymorphism determines elevated aldosterone, higher blood pressure, and reduced glomerular filtration, especially in diabetic female patients. Endocr Regul. 2020;54(3):217-26. doi: 10.2478/enr-2020-0024

22. Virani SS, Alonso A, Benjamin EJ, Bittencourt MS, Callaway CW, Carson AP, et al. Heart Disease and Stroke Statistics-2020 Update: A Report From the American Heart Association. Circulation [Internet]. 2020[cited 2024 Jun 10];141(9): e139-e596. Available from: https://www.ahajournals.org/doi/epub/10.1161/CIR.0000000000000757 doi: 10.1161/cir.0000000000000757

23. Xiao H, Shao X, Gao P, Zou H, Zhang X. Metabolic Syndrome Components and Chronic Kidney Disease in a Community Population Aged 40 Years and Older in Southern China: A Cross-Sectional Study. Diabetes Metab Syndr Obes. 2022;15:839-48. doi: 10.2147/DMSO.S353305

24. Xie K, Bao L, Jiang X, Ye Z, Bing J, Dong Y, et al. The association of metabolic syndrome components and chronic kidney disease in patients with hypertension. Lipids Health Dis [Internet]. 2019[cited 2024 Jun 10];18(1):229. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6935087/pdf/12944_2019_Article_1121.pdf doi: 10.1186/s12944-019-1121-5

25. Yun HR, Kim H, Park JT, Chang TI, Yoo TH, Kang SW, et al. Obesity, metabolic abnormality, and progression of CKD. Am J Kidney Dis. 2018;72(3):400-10. doi: 10.1053/j.ajkd.2018.02.362

Відомості про авторів:
Литвин Б. А. – аспірант кафедри сімейної медицини, Буковинський державний медичний університет, м. Чернівці, Україна.
E-male: dr.bogdan@ukr.net
Сидорчук Л. П. – доктор медичних наук, професор, завідувач кафедри сімейної медицини, Буковинський державний медичний університет, м. Чернівці, Україна.
E-mail: lsydorchuk@ukr.net
ORCID ID: https://orcid.org/0000-0001-9279-9531

Information about the authors:
Lytvyn B. A. – Post-graduate, Department of Family Medicine, Bukovinian State Medical University, Chernivtsi, Ukraine.
E-male: dr.bogdan@ukr.net
Sydorchuk L. P. – Doctor of Science, Professor, Head of the Department of Family Medicine, Bukovinian State Medical University, Chernivtsi, Ukraine.
E-mail: lsydorchuk@ukr.net
ORCID ID: https://orcid.org/0000-0001-9279-9531

Стаття надійшла до редакції 04.06.2024