DOI: https://doi.org/10.17816/RFD642563 EDN: TTSNSF

Asymptomatic Hyperphosphatemia in Healthy Young Men and Women: Potential Association With Electronic Nicotine Delivery Systems and Heated Tobacco Products

Alena M. Ovcharenko, Olga B. Ershova, Kseniia Yu. Belova

Yaroslavl State Medical University, Yaroslavl, Russia

ABSTRACT

BACKGROUND: Hyperphosphatemia is defined as fasting phosphorus levels higher than 1.45 mmol/L. Although it is often asymptomatic, it can cause adverse effects, including hypocalcemia, decreased calcitriol levels, secondary hyperparathyroidism, ectopic calcification, hemodynamic disorders, and increased morbidity and mortality. Therefore, it is crucial to promptly identify and treat the underlying causes of hyperphosphatemia. Hyperphosphatemia is typically associated with renal failure. Less common causes include hypoparathyroidism; burn injury; metabolic and respiratory acidosis; rhabdomyolysis; tumor lysis syndrome; acromegaly; and excessive intake of phosphorus from food or medication.

AIM: The study aimed to identify the possible causes of an asymptomatic increase in blood phosphorus levels in young healthy adults.

METHODS: Laboratory tests were performed in young healthy men and women aged 18 to 35 years.

RESULTS: Despite preserved renal function, asymptomatic hyperphosphatemia was diagnosed in 12 (9.9%) participants. A significant association was found between hyperphosphatemia and the use of electronic nicotine delivery systems and heated tobacco products (p = 0.0002).

CONCLUSION: More research is needed on the effects of electronic nicotine delivery systems and heated tobacco products on human health, especially calcium and phosphorus metabolism.

Keywords: phosphorus; hyperphosphatemia; electronic nicotine delivery systems; heated tobacco products.

To cite this article

Ovcharenko AM, Ershova OB, Belova KYu. Asymptomatic Hyperphosphatemia in Healthy Young Men and Women: Potential Association With Electronic Nicotine Delivery Systems and Heated Tobacco Products. *Russian Family Doctor.* 2025;29(2):63–70. DOI: 10.17816/RFD642563 EDN: TTSNSF

Submitted: 04.12.2024 **Accepted:** 11.02.2025 **Published online:** 17.06.2025

DOI: https://doi.org/10.17816/RFD642563 EDN: TTSNSF

Бессимптомная гиперфосфатемия у здоровых молодых мужчин и женщин: связана ли она с использованием электронных систем доставки никотина и систем нагревания табака?

А.М. Овчаренко, О.Б. Ершова, К.Ю. Белова

Ярославский государственный медицинский университет, Ярославль, Россия

АННОТАЦИЯ

Обоснование. Гиперфосфатемия (значения фосфора у взрослых натощак более 1,45 ммоль/л), часто протекая бессимптомно, способна вызвать большое количество неблагоприятных последствий, таких как гипокальциемия, снижение уровня кальцитриола, вторичный гиперпаратиреоз, внескелетная кальцификация, гемодинамические расстройства и даже увеличение заболеваемости и смертности. Это вызывает необходимость своевременного выявления и устранения причин гиперфосфатемии. Чаще всего ее этиология связана с почечной недостаточностью. Реже встречаются такие причины, как гипопаратиреоз, ожоговая травма, метаболический и дыхательный ацидоз, рабдомиолиз, синдром лизиса опухоли, акромегалия, избыточное поступление фосфора с пищей и лекарственными препаратами.

Цель — определить возможные причины бессимптомного повышения уровня фосфора в крови у молодых здоровых людей.

Материалы и методы. Выполнено лабораторное обследование молодых здоровых мужчин и женщин в возрасте от 18 до 35 лет.

Результаты. У 12 (9,9%) обследованных выявлена бессимптомная гиперфосфатемия на фоне сохраненной функции почек. Обнаружена достоверная связь гиперфосфатемии и использования электронных систем доставки никотина и систем нагревания табака (p=0,0002).

Заключение. Необходимо дальнейшее изучение влияния электронных систем доставки никотина и систем нагревания табака на здоровье человека, в частности на фосфорно-кальциевый обмен, а также последствий их использования.

Ключевые слова: фосфор; гиперфосфатемия; электронные системы доставки никотина; системы нагревания табака.

Как цитировать

Овчаренко А.М., Ершова О.Б., Белова К.Ю. Бессимптомная гиперфосфатемия у здоровых молодых мужчин и женщин: связана ли она с использованием электронных систем доставки никотина и систем нагревания табака? // Российский семейный врач. 2025. Т. 29. № 2. С. 63–70. DOI: 10.17816/RFD642563 EDN: TTSNSF

Рукопись получена: 04.12.2024 **Рукопись одобрена:** 11.02.2025 **Опубликована online:** 17.06.2025

BACKGROUND

Phosphorus is a macronutrient found in the body, mainly in bone tissue, as hydroxyapatite crystals [1]. Most phosphorus bound to calcium in the body (approximately 85%) is found in bone and dental tissue and the remaining phosphorus (approximately 14%) is intracellular. Serum and extracellular phosphorus accounts for only 1% of systemic phosphorus, including inorganic phosphate (30%), which is a variable and measurable component in clinical practice. Phosphorus is also an essential anion in the extracellular fluid [2].

The main natural source of dietary (organic) phosphorus is plant and animal proteins. Organic phosphorus is converted into inorganic phosphorus by intestinal enzymes, resulting in different bioavailability and differentiated absorption of phosphorus from different foods [3].

Inorganic phosphorus is found in multiple food additives, including orthophosphoric acid (E338), sodium phosphate (E339), potassium phosphate (E340), calcium phosphate (E341), dicalcium phosphate (E450), trisodium phosphate (E451), and polyphosphates (E452) used for food processing. 80%–100% of these supplements is absorbed in the intestine [3]. Cheese, egg yolk, sea fish, dry-cured sausages, muesli, brown rice, legumes, nuts, chocolate, and carbonated drinks have the highest levels of phosphorus.

Normal serum phosphorus levels are maintained within the range of 0.8 to 1.45 mmol/L in adults and vary throughout the day depending on dietary phosphorus intake. Approximately 60%–65% dietary phosphorus is absorbed in the small intestine. Phosphorus is absorbed in the intestine via the transcellular active pathway and the paracellular passive pathway. Phosphorus is primarily absorbed in the proximal small intestine via Type IIb sodium-phosphate cotransporter (NPT2b). NPT2b is encoded by the *SLC34A2* gene and expressed in the luminal membrane of enterocytes, where phosphorus is mainly absorbed in humans [4]. It is activated by low dietary phosphorus, the active form of vitamin D, and estrogen and inhibited by glucocorticoids and epidermal growth factor. At least 80% phosphorus is reabsorbed in the kidney proximal tubules; less than 10% is reabsorbed in distal nephrons [4]. The main factors enhancing renal transcellular phosphorus reabsorption are low dietary phosphorus levels, 1,25-dihydroxyvitamin D [$1,25(OH)_2D$], and thyroid hormones; whereas parathyroid hormone and fibroblast growth factor 23 reduce the availability of phosphorus cotransporters (NPT2a and NPT2c) in the brush border membrane of the tubules, stimulating their closure [4]. Plasma phosphorus levels are maintained by a complex interaction of intestinal absorption, renal tubular reabsorption, and transcellular phosphorus flux from intracellular fluid to bone storage pools and vice versa [5].

Hyperphosphatemia is a syndrome detected by tests when the blood phosphorus level increases to more than 1.45 mmol/L [2]. It is often associated with renal disorders. Less common causes include secondary hyperparathyroidism, hypoparathyroidism, burn injury, metabolic and respiratory acidosis, rhabdomyolysis, tumor lysis syndrome, acromegaly, and excessive intake of phosphorus with food and medications [4]. Abnormal serum phosphorus may be associated with various diseases, especially in critically ill patients [2]. Chronic hyperphosphatemia is often asymptomatic [4].

Phosphorus and calcium metabolisms are closely connected. Hyperphosphatemia promotes parathyroid hormone (PTH) synthesis by the parathyroid glands and fibroblast growth factor 23 synthesis by osteocytes and osteoblasts. This helps to reduce the phosphorus transport activity required for its reabsorption, promotes the excretion of phosphorus in the urine, and reduces the renal synthesis of the active vitamin D, reducing the active phosphorus transport from the intestine and its overall absorption. This system does not regulate its passive transport. In contrast, higher levels of active vitamin D also promote the synthesis of fibroblast growth factor 23 while releasing PTH in response to low vitamin D levels. PTH has the opposite effect on the renal synthesis of active vitamin D compared to fibroblast growth factor 23. PTH promotes hydroxylation, leading to increased intestinal absorption of both phosphorus and calcium. Thus, fibroblast growth factor 23, vitamin D, and PTH are key drivers of phosphorus metabolism. In addition, calcitonin contributes to hypophosphatemia by decreasing reabsorption and increasing excretion. Active vitamin D enhances the intestinal absorption of phosphorus, increases its blood level, and promotes the fixation of phosphorus and calcium salts in bone tissue [6].

Increased level of circulating blood phosphorus promotes PTH secretion and reduces calcium levels, leading to further promotion of PTH secretion. In response, PTH promotes calcium resorption from bone tissue, calcium reabsorption in renal tubules, renal hydroxylation of vitamin D to its active form, and renal excretion of phosphorus [7]. Chronic hyperphosphatemia can cause soft tissue calcification, vascular calcification, and left ventricular hypertrophy [4]. It is well known that in hyperphosphatemia, high PTH and fibroblast growth factor 23 in combination with low vitamin D levels cause calcium release from bones, vascular calcification, and reduce bone strength. Moreover, high serum phosphorus is associated with adverse cardiovascular events [8].

The study aimed to identify possible causes of asymptomatic increase in blood phosphorus levels in young healthy volunteers aged 18 to 35.

METHODS

Eligibility criteria: Men and women aged 18–35, who signed informed consent to participate in the study. Withdrawal

criteria: Acute and/or chronic diseases in the acute phase (decompensation) and/or requiring constant medication, history or suspicion of cancer of any location (except for benign neoplasms), pregnancy and breastfeeding, inadequate assessment of one's condition, and mental disorders.

We examined the medical history of all participants; all participants had a medical examination. We performed a blood chemistry panel to determine the total protein, creatinine, glucose, calcidiol [25(OH)D], C-reactive protein, total calcium, phosphorus, alkaline phosphatase, thyroid-stimulating hormone, testosterone, lipid profile, C-terminal telopeptide of type 1 collagen in serum, as well as dual-energy X-ray absorptiometry (DXA), echocardiography, and electrocardiography. The examination was conducted from March 09, 2023, to May 08, 2024.

Statistical analysis was performed using Statistica 12.5 software.

RESULTS

The study involved 121 young healthy volunteers aged 18 to 35, including 62 (51.24%) men and 59 (48.76%) women.

121 subjects included 12 subjects (9.92%) with an isolated increase in phosphorus levels to > 1.45 mmol/L and preserved renal function (EGFR (CKD-EPI)[1] > 90 mL/min/m^2). Eight participants with hyperphosphatemia had additional examination to identify the causes of asymptomatic increase in blood phosphorus (see Table 1).

The tests show that all 8 subjects with hyperphosphatemia had preserved renal function with EGFR (CKD-EPI) > 90 mL/min/m^2. It is noteworthy that the calcium level detected in 7 of 8 cases was normal, despite the fact that phosphorus and calcium changes are usually converse.

It should be noted that 7 of 8 cases had vitamin D deficiency or insufficiency.

PTH level abnormalities were identified in 3 of 8 cases; hyperparathyroidism was diagnosed in 2 cases and 1 case of hypoparathyroidism was identified. In both hyperparathyroidism cases, the males had vitamin D deficiency and normal calcium levels. Increased PTH levels associated with vitamin D deficiency supports the diagnosis of secondary hyperparathyroidism.

In addition, we identified a case of hypoparathyroidism with hyperphosphatemia and preserved renal function, normal calcium levels, and reduced vitamin D levels. In general, vitamin D deficiency causes secondary hyperparathyroidism. However, in this case, observed PTH level was as low as 13 pg/mL. Additional examination and the densitometry were within the expected range for age.

Alkaline phosphatase level was within the reference range in all but one female subject (29 U/L). A young female, 26 years of age, had no complaints and denied any chronic diseases. Her medical history showed vertebral fractures (at the age of 5 and 10), wrist fractures (at the age of 4, 5, 10, and 11), and fractures associated with falls from a horizontal bar and other sports activities. DXA tests were within the expected range for age. However, she had had molecular genetic testing at the Medical Genetics Center of the National Medical Research Center for Children's Health and no mutations were found.

DXA tests of 7 of 8 subjects showed bone mineral density consistent with age. Echocardiography and electrocardiography showed no significant abnormalities in the subjects. It is worth noting that all subjects denied excessive phosphorus intake with food or taking medications.

Table 1. Laboratory serum tests in participants with hyperphosphatemia

Таблица 1. Лабораторные показатели сыворотки крови пациентов с гиперфосфатемией

Subject, age, sex	Inorganic phosphorus (reference value: 0.81–1.45), mmol/L	Creatinine (reference value: 44–80), mmol/L	Creatinine clearance (reference value: 71–151), mL/min	Total calcium (reference value: 2.2–2.55), mmol/L	Parathyroid hormone (reference value: 15–65), pg/mL	Calcidiol [25(OH)D] (reference value: 30–100), ng/mL	Alkaline phosphatase (reference value: 40–129), U/L
A, 26 years old, female	1.63↑	74	121	2.39	35.37	29.9	54
B, 26 years old, female	2.22↑	61	121	2.23	61.4	17.97↓	29↓
C, 22 years old, female	1.63↑	56	120	2.14↓	40.7	20.05↓	51
D, 27 years old, male	1.52↑	92	127	2.39	85.1↑	13.98↓	54
E, 24 years old, male	1.50↑	95	96	2.38	27.56	21.75↓	87
F, 28 years old, male	1.48↑	73	120	2.23	89.02 ↑	6.72↓	83
G, 23 years old, male	1.82↑	92	101	2.42	36.96	11.67↓	56
H, 23 years old, male	1.57↑	89	105	2.31	13.77↓	22.42↓	55

Note. The arrows indicate an increase or decrease in the value.

[1] Glomerular filtration rate calculated using the Chronic Kidney Disease Epidemiology Collaboration formula.

DOI: https://doi.org/10.17816/RFD642563

Upon closer examination, it was found that all 8 volunteers with hyperphosphatemia regularly use electronic nicotine delivery systems and tobacco heating systems also known as e-cigarettes.

49 (40.50%) of 121 subjects use electronic nicotine delivery systems and tobacco heating systems. Moreover, in 38 (77.55%) of 49 users of such systems, the phosphorus level was within the reference range, but nearly every fourth subject (11 subjects or 22.45%) had hyperphosphatemia. It is worth noting that hyperphosphatemia was recorded in only 1 (1.39%) of 72 (59.50%) participants who do not use electronic nicotine delivery systems and tobacco heating systems (see Fig. 1). However, his medical history indicates long-term passive use of e-cigarettes.

The hypoparathyroidism case was a 23-year-old male subject G with no complaints and normal physical examination who denied any chronic conditions. He denied excessive intake of phosphorus from food and the use of medications. His medical history shows that the subject has been regularly (for about 3 years) using nicotine e-cigarettes. When using them, he takes up to 500 puffs per day. However, the examination, namely the calcium level within the reference range, allows to assume that it was hyperphosphatemia that caused the abnormal PTH level. Moreover, after vitamin D level was restored in 3 months [25(OH)D level of 34 ng/mL], the PTH level reached normal values (25.05 pg/mL), while the phosphorus level increased to 1.61 mmol/L. The phosphorus level may have increased due to continued use of e-cigarettes in the same amount.

Statistical processing and the χ^2 test showed the significant relationship between hyperphosphatemia and the use of electronic nicotine delivery systems and tobacco heating systems (p = 0.0002).

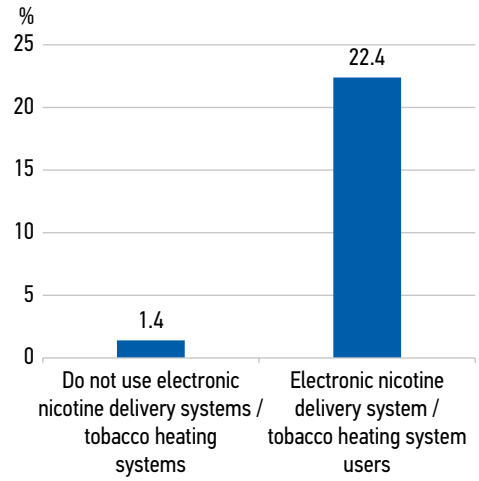

Fig. 1. The association between asymptomatic hyperphosphatemia and the use of electronic nicotine delivery systems and heated tobacco products.
Рис. 1. Бессимптомная гиперфосфатемия при использовании электронных систем доставки никотина и систем нагревания табака (ЭСДН/ЭСДПН).

DISCUSSION

Although e-cigarettes appear to be relatively safer than regular cigarettes, they expose users to known harmful components. However, their long-term health effects have been understudied [9]. It should be noted that data on various side effects associated with the use of e-cigarettes has already been published [10]. Nevertheless, the authors did not find any references to the relationship between hyperphosphatemia and the use of electronic nicotine delivery systems and tobacco heating systems in the available literature.

The most studied adverse health effects of e-cigarettes are the respiratory effects, including damage to the pulmonary epithelium and chronic inflammation of the bronchial mucosa. Sputum of e-cigarette users has higher observable levels of myeloperoxidase, neutrophil elastase, and proteinase 3 indicating activation of neutrophils [11].

We know about pulmonary dysfunction and E-Cigarette/Vaping-Associated Lung Injury (EVALI syndrome) [10].

Cardiovascular adverse effects have also been described; daily use of e-cigarettes has been shown to be an independent myocardial infarction risk factor. In addition, adverse neurological and gastrointestinal effects are known [11].

These studies indicate that e-cigarette aerosol contains measurable amounts of ethanol, volatile organic compounds, polycyclic aromatic hydrocarbons, silicon, lead, nickel, formaldehyde, acetaldehyde, isoprene, acetic acid, 2-butanedione, acetone, and propanol [11].

Flavoring agents in e-cigarette aerosols are deemed safe and often used in the food industry, but this does not mean they are safe to inhale [11].

Electronic nicotine delivery systems and tobacco heating systems mainly release water vapor, carbon dioxide, propylene glycol, glycerol, and acrolein [12]. It is likely that in constant use of innovative nicotine delivery systems and tobacco heating systems, an excess amount of carbon dioxide enters the human body, causing chronic respiratory acidosis as a possible cause of increased phosphorus levels in human blood [13] (see Fig. 2).

Moderate compensated acidosis occurs without pronounced clinical signs [14].

It should be noted that regardless of the etiology, hyperphosphatemia may have long-term effects. Higher serum phosphorus level reduces calcium levels; thereby indirectly promoting PTH secretion. Phosphorus has no direct effects on the PTH-secreting cells; however, if the renal excretion of phosphorus is impaired, secondary stimulation of the parathyroid glands occurs, leading to their hypertrophy. Higher phosphorus levels promote the binding of ionized calcium to phosphorus, leading to lower plasma calcium levels. Hypocalcemia further promotes PTH secretion [15]. PTH promotes a negative balance of bone tissue, i.e. osteolysis, observed, for example, in hyperparathyroidism [15]. In response

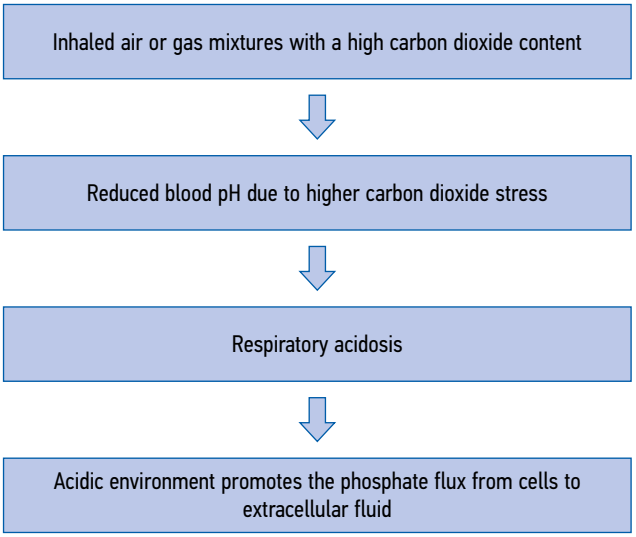

Fig. 2. Progression of gas acidosis and hyperphosphatemia with the use of electronic nicotine delivery systems and tobacco heating products.
Рис. 2. Схема развития газового ацидоза и гиперфосфатемии при использовании электронных систем доставки никотина и систем нагревания табака.

to hypocalcemia, hyperphosphatemia, and low calcitriol levels, secondary hyperparathyroidism develops [16], enhancing bone resorption. This is followed by musculoskeletal changes (osteoporosis, bone deformities, fractures), kidney conditions, and cardiovascular disorders.

Clinical studies have shown that higher serum phosphorus levels are a risk factor of vascular calcification and cardiovascular disease in the population with chronic kidney disease, especially in patients with dialysis dependent end-stage renal failure [17]. Phosphorus levels at the upper limit of the normal range have been found to be correlated with a higher risk of cardiovascular death in the general population, indicating potential phosphorus toxicity [17].

There is growing evidence that e-cigarettes may no longer be considered harmless devices [11].

According to a RIA Novosti report on May 31, 2024,[1] a fractional bill on a total ban of vapes in Russia was introduced to the State Duma. The World Health Organization recommends against using e-cigarettes as their use has not been sufficiently studied as a safe and effective tobacco use disorder treatment. To date, there has not been enough research to determine with certainty whether the use of clean and properly regulated electronic nicotine delivery systems and heated tobacco systems causes cardiovascular diseases, lung diseases, or cancer. The World Health Organization has developed and, on December 04, 2023, published a call to action[2] to take urgent and decisive measures to prevent the use of electronic cigarettes, which are harmful to health, to protect children and non-smokers, and to minimize harm to public health.

CONCLUSION

Regardless of the etiology, hyperphosphatemia has serious long-term effects. The influence of long-term use of electronic nicotine delivery systems and tobacco heating systems on human health (namely, phosphorus and calcium metabolism) and the individual effects of such innovative systems appear to be unsafe.

ADDITIONAL INFORMATION

Author contributions: A.M. Ovcharenko: investigation, formal analysis, writing – original draft, writing – review & editing; O.B. Ershova: conceptualization, supervision, writing – original draft, writing – review & editing; K.Yu. Belova: writing – original draft, writing – review & editing. All authors approved the version of the manuscript to be published, and agreed to be accountable for all aspects of the work, ensuring that questions related to the accuracy or integrity of any part of it are appropriately reviewed and resolved.
Ethics approval: The study was approved by the local Ethics Committee at the Yaroslavl State Medical University (Protocol No. 64 dated October 13, 2023). All participants provided written informed consent to participate in the study. The study and its protocol were not registered.
Funding sources: No funding.
Disclosure of interests: The authors have no relationships, activities, or interests over the past three years related to for-profit or not-for-profit third parties whose interests may be affected by the content of the article.
Statement of originality: The authors did not use any previously published information (text, illustrations, or data) in this work.
Data availability statement: All data generated during this study are included in this article.
Generative AI: No generative AI was used in preparing this article.
Provenance and peer-review: This work was submitted unsolicited and reviewed following the standard procedure. The peer review process involved two members of the editorial board.

ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Вклад авторов. А.М. Овчаренко — проведение исследования, анализ данных, написание черновика, пересмотр и редактирование рукописи; О.Б. Ершова — определение концепции, руководство исследованием, написание черновика, пересмотр и редактирование рукописи; К.Ю. Белова — написание черновика, пересмотр и редактирование рукописи. Все авторы одобрили рукопись (версию для публикации), а также согласились нести ответственность за все аспекты работы, гарантируя надлежащее рассмотрение и решение вопросов, связанных с точностью и добросовестностью любой ее части.
Этический комитет. Проведение исследования одобрено локальным этическим комитетом Ярославского государственного медицинского университета (№ 64 от 13.10.2023). Все участники исследования добровольно подписали форму информированного согласия на участие в исследовании. Исследование и его протокол не регистрировали.
Источники финансирования. Отсутствуют.

[1] Vape: What Is It? Are E-Cigarettes Dangerous? // Вейп: что это такое, какой вред несет курение электронных сигарет. https://ria.ru/20240531/veyp-1949605434.html. Accessed on November 14, 2024.
[2] Electronic cigarettes: call to action. WHO. https://www.who.int/publications/m/item/electronic-cigarettes---call-to-action. Accessed on November 14, 2024.

Раскрытие интересов. Авторы заявляют об отсутствии отношений, деятельности и интересов за последние три года, связанных с третьими лицами (коммерческими и некоммерческими), интересы которых могут быть затронуты содержанием статьи.
Оригинальность. При создании настоящей работы авторы не использовали ранее опубликованные сведения (текст, иллюстрации, данные).
Доступ к данным. Все данные, полученные в настоящем исследовании, доступны в статье.
Генеративный искусственный интеллект. При создании настоящей статьи технологии генеративного искусственного интеллекта не использовали.
Рассмотрение и рецензирование. Настоящая работа подана в журнал в инициативном порядке и рассмотрена по обычной процедуре. В рецензировании участвовали два внутренних рецензента из состава редакционной коллегии.

REFERENCES

1. Tsai SH, Kan WC, Jhen RN, et al. Secondary hyperparathyroidism in chronic kidney disease: a narrative review focus on therapeutic strategy. *Clin Med (Lond).* 2024;24(5):100238. doi: 10.1016/j.clinme.2024.100238
2. Xu X, Zhang L, Liu W, et al. Analysis of the relationship between early serum phosphate levels and short-term mortality in septic patients: a retrospective study based on mimic-IV. *Shock.* 2023;59(6):838–845. doi: 10.1097/SHK.0000000000002119
3. Clinical recommendations. Chronic kidney disease. 2021. [Internet]. Available from: https://rusnephrology.org/wp-content/uploads/2020/12/CKD_final.pdf Accessed: 19 Sept 2024). (In Russ.)
4. Koumakis E, Cormier C, Roux C, Briot K. The causes of hypo- and hyperphosphatemia in humans. *Calcif Tissue Int.* 2021;108(1):41–73. doi: 10.1007/s00223-020-00664-9
5. Leung J, Crook M. Disorders of phosphate metabolism. *J Clin Pathol.* 2019;72(11):741–747. doi: 10.1136/jclinpath-2018-205130
6. Mohort TV. Disorders of phosphorus metabolism in clinical practice. *Meditsinskie novosti*. 2023;(4):28–34. (In Russ.)
7. Volkov MM, Kayukov IG, Smirnov AV. Phosphorus-calcium metabolism and its regulation. *Nephrology (Saint Petersburg).* 2010;14(1):91–103. (In Russ.) doi: 10.24884/1561-6274-2010-14-1-91-103
8. Anand A, Aoyagi H. Understudied hyperphosphatemia (chronic kidney disease). Treatment targets and new biological approaches. *Medicina (Kaunas).* 2023;59(5):959. doi: 10.3390/medicina59050959
9. Boakye E, Obisesan OH, Osei AD, et al. The promise and peril of vaping. *Curr Cardiol Rep*. 2020;22(12):155. doi: 10.1007/s11886-020-01414-x
10. Golan R, Muthigi A, Ghomeshi A, et al. Misconceptions of vaping among young adults. *Cureus.* 2023;15(4):e38202. doi: 10.7759/cureus.38202
11. Virgili F, Nenna R, Ben David S, et al. E-cigarettes and youth: an unresolved Public Health concern. *Ital J Pediatr.* 2022;48(1):97. doi: 10.1186/s13052-022-01286-7
12. Enikeyev DA, Idrisova LT, Enikeyev OA, et al. Comparison of hazard and toxicity of tobacco cigarettes, electronic nicotine delivery systems, and tobacco heating systems (iqos) (review). *Patogenez.* 2019;17(3):25–33. doi: 10.25557/2310-0435.2019.03.25-33
13. Tsygan VN, editor. *Clinical pathophysiology:* a textbook for cadets and students of military medical universities. In 2 vol. Saint Petersburg: SpetsLit; 2018. Vol. 1: Pathophysiology. 430 p. (In Russ.)
14. Litvitsky PF. *Pathophysiology*. Textbook. In 2 vol. 2nd edition. Moscow: GEOTAR-MED; 2003. Vol. 1. p. 421.
15. Chagina EA, Turmova EP, Bashtovaya AE, Posadskaya MV. The pathogenetic role of phosphorus in the vital activity of the body. *International Journal of Humanities and Natural Sciences*. 2022;12(75):45–50. doi: 10.24412/2500-1000-2022-12-5-45-50
16. Pankiv VI. Hyperparathyroidism: diagnostics, clinical signs and symptoms, modern approaches to treatment. International Endocrinology Journal. 2013;1(49):87–96. (In Russ.)
17. Shutov EV, Sorokoletov SM. Hyperphosphatemia – meaning and methods of correction. *Clinical nephrology*. 2019;(2):69–75. (In Russ.) doi: 10.18565/nephrology.2019.2.69-75

AUTHORS INFO

*** Alena M. Ovcharenko**, MD;
address: 5 Revolutsionnaya St., Yaroslavl, 150000, Russia;
ORCID: 0009-0008-2402-8258;
eLibrary SPIN: 1253-2419;
e-mail: alenaovcharenko111@gmail.com

ОБ АВТОРАХ

*** Овчаренко Алена Максимовна**;
адрес: Россия, 150000, Ярославль, ул. Революционная, д. 5;
ORCID: 0009-0008-2402-8258;
eLibrary SPIN: 1253-2419;
e-mail: alenaovcharenko111@gmail.com

* Corresponding author / Автор, ответственный за переписку

Olga B. Ershova, MD, Dr. Sci. (Medicine);
ORCID: 0000-0001-7167-2187;
eLibrary SPIN: 8238-8201;
e-mail: yarosteoporosis@list.ru

Kseniia Yu. Belova, MD, Dr. Sci. (Medicine);
ORCID: 0000-0002-7856-1567;
eLibrary SPIN: 4372-8670;
e-mail: ksbelova@mail.ru

Ершова Ольга Борисовна, д-р мед. наук;
ORCID: 0000-0001-7167-2187;
eLibrary SPIN: 8238-8201;
e-mail: yarosteoporosis@list.ru

Белова Ксения Юрьевна, д-р мед. наук;
ORCID: 0000-0002-7856-1567;
eLibrary SPIN: 4372-8670;
e-mail: ksbelova@mail.ru