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Г.А. Игнатенко[1], Г.Г. Тарадин*[1,2], А.Э. Багрий[1], И.В. Ракитская[1], К.Э. Могилевская[1], В.И. Меркурьев[3], А.Л. Христуленко[1]

[1] – ГОО ВПО «Донецкий национальный медицинский университет им. М. Горького», Донецк, Украина
[2] — ГУ «Институт неотложной и восстановительной хирургии им. В.К. Гусака», Донецк, Украина
[3] — КУ «Центральная городская клиническая больница № 1», Донецк, Украина

СЛУЧАЙ БРАДИКАРДИИ, РАЗВИВШЕЙСЯ НА ФОНЕ ГИПЕРКАЛИЕМИИ У ПАЦИЕНТКИ ОТДЕЛЕНИЯ АМБУЛАТОРНОГО ГЕМОДИАЛИЗА

G.A. Ignatenko[1], G.G. Taradin*[1,2], A.E. Bagry[1], I.V. Rakitskaya[1], K.E. Mogilevskaya[1], V.I. Merkuriev[3], A.L. Khristulenko[1]

[1] — State Educational Organization of Higher Professional Education «M. Gorky Donetsk National Medical University», Donetsk, Ukraine
[2] — State Institution «V.K. Gusak Institute of Urgent and Recovery Surgery», Donetsk, Ukraine
[3] – Clinical Institution «Central City Clinical Hospital № 1», Donetsk, Ukraine

The Case of Bradycardia Occured in the Setting of Hyperkalemia in a Patient in Ambulatory Hemodialysis Department

Резюме

В статье представлено клиническое наблюдение развития брадикардии у 64-летней пациентки с хроническим заболеванием почек, находящейся на лечении в отделении амбулаторного гемодиализа. При регистрации электрокардиограммы была зафиксирована аритмия в виде ритма из атриовентрикулярного соединения. Характерные изменения на электрокардиограмме, наличие факторов риска, данные дополнительно собранного анамнеза позволили предположить развитие опасного состояния — гиперкалиемии. Диагноз подтвердился после определения уровня сывороточного калия. Приведенный случай иллюстрирует необходимость рассматривать вероятность гиперкалиемии у пациентов с хроническим заболеванием почек, включая находящихся на гемодиализе. При возникновении характерных клинических проявлений и изменений на электрокардиограмме необходимо как можно быстрее определить сывороточное содержание калия для своевременной и адекватной коррекции электролитного нарушения.

Ключевые слова: *брадикардия, узловой ритм, нарушения ритма сердца, гиперкалиемия, калий, факторы риска, хроническое заболевание почек, гемодиализ*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

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*Контакты: Геннадий Геннадьевич Тарадин, e-mail: taradin@inbox.ru
* Contacts: Gennady G. Taradin, e-mail: taradin@inbox.ru
ORCID ID: https://orcid.org/0000-0003-3984-8482

Abstract

The presented article contains the clinical observation of bradycardia development in 64-year-old patient with chronic kidney disease who was in ambulatory treatment at the hemodialysis department. During electrocardiogram recording an arrhythmia was detected as a junctional rhythm. The specific changes on electrocardiogram, presence of risk factors, and data of additional collection of history disease allowed purposing the development of dangerous condition — hyperkalemia. The diagnosis was confirmed after detection of the serum potassium level. This case illustrates the necessity to consider the possibility of hyperkalemia in patients with chronic kidney diseases including those who undergoing hemodialysis treatment. Relevant clinical manifestations and changes on the electrocardiogram require the urgent assessment of the serum potassium level for timely and adequate correction of the electrolyte disorder.

Key words: *bradycardia, junctional rhythm, cardiac arrhythmias, hyperkalemia, potassium, risk factors, chronic kidney disease, hemodialysis*

Conflict of interests

The authors declare that this study, its theme, subject and content do not affect competing interests

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AH — arterial hypertension, AV — atrioventricular, BP — blood pressure, CKD — chronic kidney disease, ECG — electrocardiogram, HR — heart rate, K^+ — potassium, MV — mitral valve

Introduction

Hyperkalemia is often among electrolyte disorders that develop in cases of chronic kidney disease (CKD); it is diagnosed at serum potassium concentration (K^+) above the upper normal limit (> 5.5 mmol/l) [1, 2]. Severe hyperkalemia is a critical condition that requires emergency interventions due to its ability to cause life-threatening rhythm and conduction disorders, even cardiac arrest and death. Electrocardiogram (ECG) is an affordable diagnostic tool for the detection of hyperkalemia. Based on the concepts of clinical significance, the type of ECG changes is a more important predictor of the outcome than the actual K^+ blood level [3].

Some chronic diseases increase the risk of hyperkalemia, especially progressive CKD, chronic heart failure, type 2 diabetes mellitus, and arterial hypertension [4]. Several agents also increase serum K^+ levels, particularly renin-angiotensin-aldosterone system inhibitors, angiotensin II receptor antagonists, potassium-sparing diuretics and non-steroidal anti-inflammatory drugs [5, 6]. Irrational intake of K^+ with foods with its high content, even in the cases of normal K^+ concentration in serum, can also be accompanied by adverse cardiovascular and renal events [7]. Risk factors for hyperkalemia are presented in the following table 1 [5].

Clinical manifestations of severe hyperkalemia, such as arterial hypotension, shock, severe weakness to complete inability to move the limbs, and cardiac arrest can easily be mistaken for a worsening of the underlying disease [3]. In such cases, ECG changes help establish the correct diagnosis before measuring serum K^+ level [3, 8].

Severe hyperkalemia slows down the rate of conduction in the His — Purkinje system, which can be manifested by different conduction disorders. ECG may reveal significant bradycardia or sinus arrest with the appearance of substitutive junctional or idioventricular rhythms, various atrioventricular (AV) conduction disorders, even complete transverse blockade [3, 9, 10]. Our observation describes one of such rhythm disorders in a patient with CKD.

Table 1. *Risk factors for the development of hyperkalemia**

Clinical Risk Factors	Medication Exposure
Male gender	Potassium supplements
Caucasian	Penicillin G
Diabetes mellitus	Digoxin
Cardiovasular diseases	Nonsteroidal anti-inflammatory drugs
Chronic heart failure	Angiotensin-converting enzyme inhibitors angiotensin receptor blocker
Acute kidney injury	Mineralocorticoids receptor antagonist
Chronic kidney disease	β-adrenergic blockers
Acidosis	Heparin
Urinary tract obstruction	Amiloride, triamterene, trimethoprim, pentamidine

* **Note:** Adapted from J.R. Montford et al. [5]

Case report

Patient K., female, 64, since 2013 has been undergoing outpatient treatment in the Hemodialysis Department for CKD stage V as the outcome of chronic mesangial proliferative glomerulonephritis, chronic tubulointerstitial nephritis, renal replacement therapy with hemodialysis since 10 JUL 2013; nephrogenic anemia; symptomatic AH grade 3.

The patient has glomerulonephritis since 1976, when proteinuria of up to 1 g/l was first detected; since 1985, occasional swelling on legs to the knee level — the patient asked for medical help, was examined, consulted with a nephrologist. In 1985, she was diagnosed with chronic glomerulonephritis with retained renal function, received treatment (heparin, dipyridamole, delagil, ascorutin).

In 1990, puncture biopsy of kidney was performed. Conclusion: chronic mesangial proliferative glomerulonephritis with moderate tubulointerstitial component, in combination with chronic tubulointerstitial nephritis.

Since 1990, AH has been registered at home, with blood pressure (BP) 180–220/100–120 mm Hg; antihypertensive agents were prescribed. At that time, serum urea and creatinine levels were within normal.

For a long period of time — until 2011 — the patient did not visit a nephrologist, was not examined, levels of creatinine, urea and electrolytes were checked extremely rarely; we found no available data. In January 2011, azotemia was revealed during planned hospitalization: serum urea — 15 mmol/l, creatinine — 0.28 mmol/l, glomerular filtration rate (GFR) — 16 ml/min/1.73 m^2 (CKD-EPI). After discharge from the hospital, the patient did not visit a nephrologist for one year.

However, according to the patient, her condition began to deteriorate: in January 2012, nausea, vomiting, severe weakness, shortness of breath at rest appeared, AH could not be corrected (the patient does not remember the agents used). Laboratory tests (05 MAR 2012) revealed urea level of 36.0 mmol/l, creatinine — 1.02 mmol/l, GFR — 3 ml/min/1.73 m^2 (CKD-EPI), K^+ — 4.6 mmol/l. In May 2012, live-saving renal replacement therapy with program hemodialysis was started, and the condition significantly improved: nausea, weakness, dyspnea decreased, blood pressure decreased to 140/80 mm Hg, urea (to 25 mmol/l) and creatinine (to 0.6 mmol/l). K^+ level during routine tests ranged from 3.1–5.4 mmol/l. According to the records in the Hemodialysis Department, no previous episodes of clinically significant hyperkalemia were observed in this patient.

Additional examinations during a visit to the Hemodialysis Department:

24 JUL 2020: Echocardiography revealed additional transverse trabeculae of left ventricle (in apex area), left ventricular myocardial hypertrophy (left ventricular myocardial mass 257.68 g, left ventricular myocardial mass index 154.3 g/m^2), moderate dilation of left atrium (LA diameter 4.8 cm), satisfactory myocardial contractility (contractility 36%, ejection fraction according to Teichholz 64.6%). Pulmonary pressure was 24 mm Hg. Mild mitral insufficiency was also found (MV leaflet sclerosis with fibrotic foci, calcification (grade 1–2) of the posterior MV leaflet, some limited excursion of MV leaflets). No signs of local contractility disorder were found.

28 JUL 2020: 24h ECG monitoring in the presence of sinus rhythm revealed the following disorders: single supraventricular extrasystole — 175 per day; group (3–4 complexes) supraventricular extrasystole — 4 per day; single monomorphic ventricular extrasystole — 3 per day. Average heart rate (HR) during daytime — 82 bpm, minimum — 57 bpm, and maximum — 132 bpm; average HR during night sleep — 60 bpm, minimum — 51 bpm, and maximum — 82 bpm. No diagnostically significant changes in repolarization processes were found in the course of examination.

Laboratory test results (Table 2) 18 AUG 2020

No ECG was registered during the patient's last visit to the Hemodialysis Department when K^+ level of 5.4 mmol/l was recorded.

The patient regularly takes moxonidine 0.2 mg twice a day, carvedilol 12.5 mg twice a day, and amlodipine 10 mg/day for her AH.

On 25 AUG 2020, the patient felt unwell before her next hemodialysis session: severe weakness, drowsiness, dizziness, increasing nausea, vomiting with gastric contents (once). However, the patient went for the hemodialysis procedure. On the way to Hemodialysis Department, BP increased to 160/100 mm Hg; the patient took a combined tablet of captopril 50 mg and hydrochlorothiazide 25 mg.

In the Hemodialysis Department, after connecting to the device at 08:10 a.m., bradycardia was registered on the monitor, with HR 30–32 bpm and BP 90/60 mm Hg. Medical help was provided (08:15 a.m.): atropine 2 mg i/v, mesatone 10 mg i/v — with no significant effect. ECG (08:15 a.m.): rhythm from AV connection with simultaneous excitation of atria and ventricles, HR 44 bpm, low voltage of the ECG in standard and amplified leads with maximum R wave amplitude in lead I — 4 mm. High pointed symmetrical positive T waves registered in leads V_2–V_4, QRS — 0.11 s, QT (abs.) — 0.58 s, QT (corr.) — 0.49 s (Fig. 1).

At 08:30 a.m., a joint examination was conducted by physicians of the Hemodialysis Department and an employee of the Prof. A.I. Dyadyk Department of Therapy of the Faculty of Internship and Postgraduate Education.

Objective findings: general state of moderate severity. The patient is awake, contact is limited, eyes are closed, the patient is obtunded. Skin with no rash, with high moisture content, pale. By percussion — symmetrical

vesicular resonance above the lungs, by auscultation — vesicular breathing in lungs, no wheezing or crepitus, respiratory rate 18 per min. Cardiac activity is rhythmic, muffled heart sounds, loud second heart sound above the aorta, systolic murmur at the apex. HR 32–40 bpm (cardiomonitor data throughout the examination period), BP 120/90 mm Hg. On palpation, abdomen is soft, painless, some regions of the intestine with normal properties. Swelling of lower legs.

Upon speaking with the patient, it turned out that she had eaten a watermelon, tomato salad with greens (parsley) the day before.

Table 2. *Results of laboratory tests*

Parameter	Results on 18.08.2020	Units	Normal range*
Red blood cells	3,15	10^{12}/L	3,70-4,70
Hemoglobin	95	g/L	115-145
Cell-color ratio	0,9	–	0,85-1,05
White blood cells	3,3	10^{9}/L	4,00-10,00
Platelets	166	10^{9}/L	170,00-400,00
ESR	24	mm/h	3,00-20,00
Band neutrophils	3	%	1,00-6,00
Segmented neutrophils	56	%	47,00-72,00
Eosinophils	1	%	1,00-5,00
Monocytes	5	%	3,00-12,00
Lymphocytes	35	%	19,00-37,00
Calcium	2,09	mmol/L	2,20-2,55
Inorganic phosphorus	2,06	mmol/L	0,81-1,45
Serum iron	19,2	μmol/L	10,70-32,20
Ionized calcium	1,06	μmol/L	1,10-1,30
K^+	5,4	mmol/L	3,50-5,50
Total blood protein	77	g/L	64,00-83,00
Blood urea	20,1	mmol/L	3,50-7,20
Creatinine	0,779	mmol/L	0,044-0,08
Blood glucose	5,3	mmol/L	4,10-6,10
Total bilirubin	12,1	μmol/L	5,00-21,00
Indirect bilirubin	12,1	μmol/L	calculated unit
Alanine aminotransferase	34	U/L	7-56
Aspartate aminotransferase	18	U/L	5-40

***Note:** normal values are given in accordance with the reference values of the laboratory in which the clinical and biochemical parameters of this patient were determined.
ESR — erythrocyte sedimentation rate; K^+ – potassium

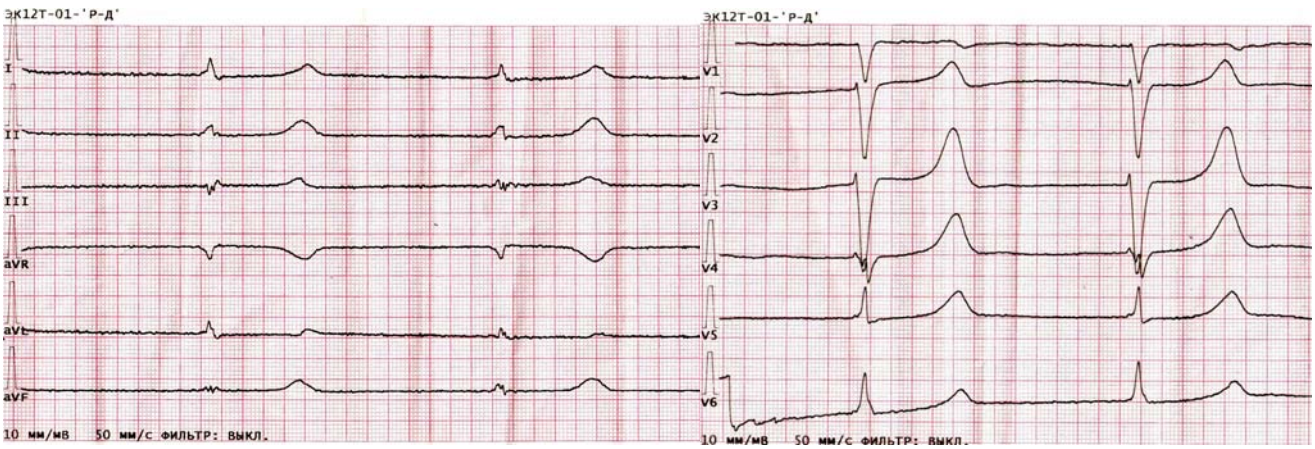

Figure 1. *Patient's electrocardiogram recorded during an episode of bradycardia, heart rate 44 beats/min.*

Based on complaints (severe weakness, drowsiness, nausea, vomiting), medical history (low-potassium diet violation), specific ECG data (new-onset junctional rhythm, high pointed symmetrical T waves in leads V_2–V_4, prolonged QT), hyperkalemia was suggested.

Blood test for K^+ (blood sample was taken urgently at 08:15 a.m., the result was obtained at 08:35 a.m.) — 7.2 mmol/l.

It was decided to continue the hemodialysis procedure. Treatment started: calcium gluconate 10% 30 ml i/v, metoclopramide 2 ml i/v, furosemide 20 mg i/v, glucose 20% 30 ml i/v. The patient was left under the care of physicians in the department; the required resources for cardiopulmonary resuscitation, if necessary, were provided.

Bicarbonate hemodialysis was performed for 3 hours. Composition of dialysis fluid: Na^+ — 136 mmol/l, HCO_3^- — 34 mmol/l; dialysate temperature was 37.5°C. Blood flow rate was 150 ml/min, dialysate flow rate was 300 ml/min.

At the 75th minute of the hemodialysis procedure, bradycardia resolved according to the monitor data. The hemodialysis session was 3 hours long. The patient's condition was considered satisfactory.

At 09:30 a.m., another ECG was registered, with positive changes: sinus rhythm was restored, regular, with HR 75 bpm, the voltage of QRS ventricular complex waves increased to 8 mm, QRS duration was 0.08 s, QT interval was normalized: QT (abs.) — 0.40 s, QT (corr.) — 0.44 s, PQ interval — 0.22 s (Fig. 2).

Repeated blood test for K^+ (result obtained at 10:00 a.m.) — 5.8 mmol/l. The patient felt satisfactory, no complaints. On 25 AUG 2020 at 02:00 p.m., the patient went home with recommendations to keep a low potassium diet and to take sodium polystyrene sulfonate 15 g three times a day.

27 AUG 2020 serum K^+ level — 5.2 mmol/l.

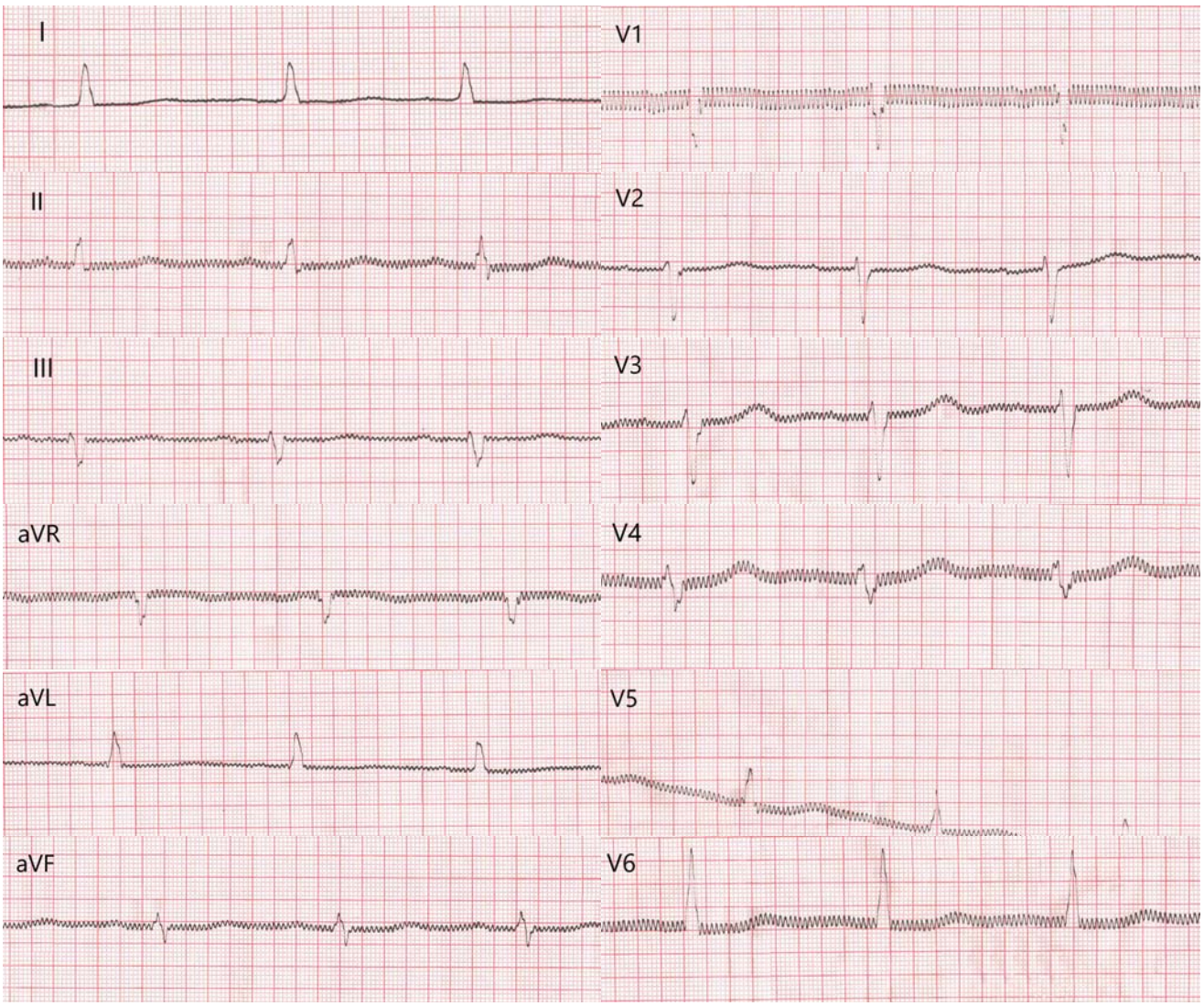

Figure 2. *The patient's electrocardiogram recorded after urgent medical care provided (02:00 p.m.), heart rate 73 beats/min.*

Discussion and conclusion

In our opinion, this case is interesting due to the fact that hyperkalemia manifested as junctional bradycardia, which was detected on the ECG during the hemodialysis session. In order to clarify the cause of the registered arrhythmia, a more detailed history was collected and serum K^+ level was determined, which allowed finding a life-threatening condition — hyperkalemia.

K^+ is a key element in maintaining the electrical potential of the cell membrane. On the ECG, early signs of hyperkalemia look like "sharp-pointed" T waves, more visible in precordial leads (V_2–V_4) [5]; they were also observed in our patient. A significant increase in the concentration of K^+ leads to delayed conduction in the AV node and His — Purkinje system due to the shortening of action potential and lengthening of phase IV of diastolic depolarization. On the ECG, this is visible as a prolonged PQ interval, decreased amplitude and enlargement of the P wave, as well as the prolonged QRS complex [5]. In our case, QRS width was 0.11 s.

In addition, the following is possible in cases of hyperkalemia: changes in the ST segment and axis position, new episode of conduction disorder (or aggravation of any of them), atrial fibrillation, ventricular tachycardia, ventricular fibrillation, asystoles. It should be noted that there are many reports of a normal or close to normal ECG in patients with severe hyperkalemia [11–13]. In the observed patient, we managed to register nodal rhythm with typical bradycardia (HR 30–44 bpm).

The prevalence of hyperkalemia among patients with end-stage renal failure is 5–10% [14]. Kidneys play a key role in maintaining potassium homeostasis. Normally, 80–90% of filtered K^+ is reabsorbed in proximal tubules and the loop of Henle, with total excretion of K^+ with urine determined primarily by its secretion into the lumen of the distal tubules of nephrons. Kidneys can increase K^+ excretion if it is in excess [15]. For this reason, patients with end-stage renal failure and significantly reduced glomerular filtration rate have an acute and/or chronic impaired K^+ excretion with the constant risk of hyperkalemia [16]. Such patients have a higher risk of adverse effects of hyperkalemia, general mortality rate, as well as mortality from cardiovascular causes [13, 17].

According to the results of a systematic literature review by E. Palaka et al. [4] that included 67 studies, the main risk factors for hyperkalemia were CKD or impaired renal function. Figure 3 shows the most common risk factors for hyperkalemia according to the systematic review [4].

Patients on dialysis for terminal renal failure deserve special attention because the risk of hyperkalemia can increase, in addition to the underlying renal pathology, due to various parameters of dialysis therapy, simultaneous intake of medical agents, or specific dietary features [12, 18].

Therefore, this case demonstrates the development of hyperkalemia in a patient with CKD on hemodialysis. Considering the high prevalence and severity of rhythm and conduction disorders due to increased serum K^+, physicians who treat patients with risk factors for hyperkalemia should pay special attention to dietary recommendations, careful selection and dosage of medical agents. If patients develop rhythm disorders, including bradycardia, it should be remembered that ECG changes in such patients may be the only manifestations of hyperkalemia, which should be considered during differential diagnosis of ECG-found changes and for providing adequate medical care.

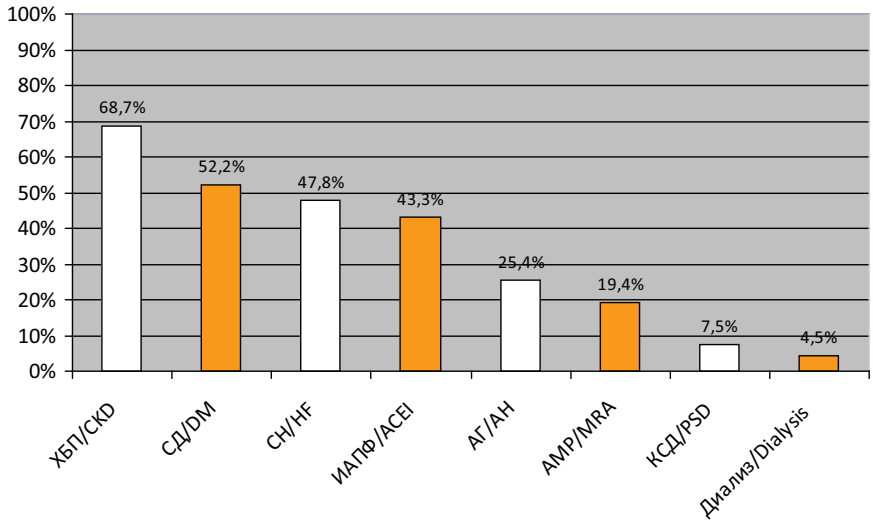

Figure 3. *The most common risk factors for hyperkalemia*

Notes: CKD — chronic kidney disease; DM — diabetes mellitus; HF — heart failure; ACEI — angiotensin-converting enzyme inhibitors; AH — arterial hypertension; MRA — mineralocorticoid receptor antagonist; PSD — potassium sparing diuretics. Adapted from E. Palaka et al. [4]

Вклад авторов:

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Игнатенко Г.А. (ORCID: https://orcid.org/0000-0003-3611-1186): создание идеи и концепции рукописи, утверждение окончательного варианта

Тарадин Г.Г. (ORCID: https://orcid.org/0000-0003-3984-8482): написание введения, обзорной части, окончательное редактирование рукописи

Багрий А.Э. (ORCID: https://orcid.org/0000-0002-0295-3724): создание дизайна рукописи, критический обзор материала, окончательное редактирование рукописи

Ракитская И.В. (ORCID: https://orcid.org/0000-0003-2694-6614): сбор и анализ литературных данных, написание обзорной части и заключения рукописи, редактирование рукописи

Могилевская К.Э.: сбор, анализ и подача клинико-лабораторных данных больной

Меркурьев В.И.: написание анамнеза, клинических данных и результатов исследований; описание пребывания больной в отделении гемодиализа

Христуленко А.Л. (ORCID: https://orcid.org/0000-0002-9954-4715): написание обсуждения и заключения

Contribution of Authors:

All the authors contributed significantly to the study and the article, read and approved the final version of the article before publication

Ignatenko G.A. (ORCID: https://orcid.org/0000-0003-3611-1186): generating the idea and the concept of the manuscript, approval of the final version

Taradin G.G. (ORCID: https://orcid.org/0000-0003-3984-8482): writing of the introduction, the review part, final editing of the manuscript

Bagry A.E. (ORCID: https://orcid.org/0000-0002-0295-3724): creating the article design, editing a manuscript

Rakitskaya I.V. (ORCID: https://orcid.org/0000-0003-2694-6614): collection and analysis of literature data, writing the review and conclusion of the manuscript, editing of the manuscript

Mogilevskaya K.E. — collection, analysis and presentation of clinical and laboratory data of the patient; investigation results

Merkuriev V.I. — writing of the history disease, clinical data and research results; description of staying of the patient in the hemodialysis department

Khristulenko A.L. (ORCID: https://orcid.org/0000-0002-9954-4715): writing of discussion and conclusion

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