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ОСТРОЕ ПОВРЕЖДЕНИЕ ПОЧЕК У ПАЦИЕНТКИ С ТРОМБОЭМБОЛИЕЙ ЛЕГОЧНОЙ АРТЕРИИ (КЛИНИЧЕСКИЙ СЛУЧАЙ)

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Acute Kidney Injury in A Female Patient with Pulmonary Embolism (Clinical Case)

Резюме

Представлен клинический случай, посвященный тромбоэмболии легочной артерии (ТЭЛА) высокого риска у 74-летней пациентки, осложнённой развитием острого повреждения почек (ОПП). В статье рассматриваются манифестация, клинические, диагностические и терапевтические аспекты ведения пациентов с ТЭЛА, включая применение тромболизиса и антикоагулянтной терапии. Описаны клинические проявления заболевания, включая выраженную одышку, слабость, отёки нижних конечностей. Течение ТЭЛА осложнилось развитием ОПП, установленного по олигурии и высокому уровню сывороточного креатинина. Представлены данные лабораторных и инструментальных исследований, демонстрирующие динамику восстановления функции почек после проведенного лечения. Оговаривается возможная роль врождённой аномалии развития (агенезии) левой почки у пациентки, как условия предрасположенности к ОПП. Приводятся основные сведения о патофизиологических механизмах ОПП при ТЭЛА. Обсуждается влияние острой правожелудочковой недостаточности, которая приводит к повышению центрального венозного давления, пассивной почечной гиперемии, увеличению интерстициального давления и отёку интерстиция почек. В развитии ОПП выделяют также снижение сердечного выброса с последующей гипоперфузией почечной паренхимы. Полагают, что сопутствующие заболевания, такие как сахарный диабет, артериальная гипертензия, хроническая болезнь почек, являются как факторами риска развития ОПП, предрасполагающими к повреждению почек в условиях тяжелого гемодинамического стресса, так и факторами, усугубляющими почечную дисфункцию при гипоперфузии и застойной нефропатии. Особое внимание уделено влиянию ОПП на прогноз ТЭЛА. Авторы делают вывод о необходимости комплексного подхода к оценке состояния больных с ТЭЛА, мониторингу почечной функции и разработке индивидуальных терапевтических стратегий для минимизации рисков повреждения почек. Статья подчеркивает значимость своевременного вмешательства и дифференцированной тактики лечения пациентов с ТЭЛА и сопутствующим ОПП.

Ключевые слова: *венозные тромбоэмболические осложнения, тромбоэмболия легочной артерии, диагностика, лечение, клиническая картина, острое повреждение почек, осложнения*

Конфликт интересов

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

Источники финансирования

Авторы заявляют об отсутствии финансирования при проведении исследования

Соответствие принципам этики

Пациент дал согласие на опубликование данных лабораторных и инструментальных исследований в статье «ОСТРОЕ ПОВРЕЖДЕНИЕ ПОЧЕК У ПАЦИЕНТКИ С ТРОМБОЭМБОЛИЕЙ ЛЕГОЧНОЙ АРТЕРИИ (клинический случай)» для журнала «Архивъ внутренней медицины», подписав информированное согласие

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Abstract:
The article presents a clinical case of high-risk pulmonary embolism (PE) in a 74-year-old patient complicated by the development of acute kidney injury (AKI). The article discusses the manifestations, clinical, diagnostic, and therapeutic aspects of managing patients with PE, including the use of thrombolysis and anticoagulant therapy. Clinical manifestations of the disease are described, including severe shortness of breath, weakness, and swelling of the lower extremities. The course of PE was complicated by the development of AKI, established by oliguria and high serum creatinine levels. The data from laboratory and instrumental studies are presented, demonstrating the dynamics of recovery of kidney function after treatment. The possible role of congenital malformation (agenesis) of left kidney in the patient as a condition of predisposition to AKI is mentioned. The basic information about the pathophysiological mechanisms of AKI in PE is presented. The effect of acute right ventricular failure, which leads to increased central venous pressure, passive renal hyperemia, increased interstitial pressure, and renal interstitial edema, is discussed. In the development of AKI, a decrease in cardiac output is also distinguished, followed by hypoperfusion of the renal parenchyma. It is believed that concomitant diseases such as diabetes mellitus, arterial hypertension, and chronic kidney disease are both risk factors for the development of AKI, predispose to kidney damage under severe hemodynamic stress, and factors that exacerbate renal dysfunction with hypoperfusion and congestive nephropathy. Special attention is paid to the effect of AKI on the prognosis of PE. The authors conclude that an integrated approach is needed to assess the condition of patients with PE, monitor renal function, and develop individual therapeutic strategies to minimize the risks of kidney damage. The article highlights the importance of timely intervention and differentiated treatment tactics for patients with PE and concomitant AKI.

Key words: *venous thromboembolism, pulmonary embolism, diagnostics, treatment, clinical picture, acute kidney injury, complications*

Conflict of interests
The authors declare no conflict of interests

Sources of funding
The authors declare no funding for this study

Conformity with the principles of ethics
The patient consented to the publication of laboratory and instrumental research data in the article «Acute Kidney Injury in A Female Patient with Pulmonary Embolism (Clinical Case)» for the journal «The Russian Archives of Internal Medicine» by signing an informed consent

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BP — blood pressure, VTE — venous thromboembolism, EH — essential hypertension, CAD — coronary artery disease, LV — left ventricle, LA — left atrium, CBC — complete blood count, AKI — acute kidney injury, PT — prothrombin time, RV — right ventricle, HF — heart failure, Amb — ambulance, PE — pulmonary embolism, AFib — atrial fibrillation, CKD — chronic kidney disease, CVP — central venous pressure, RR — respiratory rate, HR — heart rate, ECG — electrocardiogram, EchoCG — echocardiography

Introduction

Venous thromboembolism (VTE), including deep vein thrombosis and pulmonary embolism (PE), are the third globally prevalent acute cardiovascular syndrome (after myocardial infarctions and strokes) [1–3]. It is very hard to define an accurate number of VTE cases, accounting for the fact that the major proportion of events remains undiagnosed [4]. 375,000–425,000 VTE cases are reported annually in the USA, not including undiagnosed and untreated cases [1, 3, 5, 6]. According to the US National Registry, PE incidence increased from almost 60,000 (23 per 100,000 people) in 1993 to 202,000 (65 to 100,000 people) in 2012 [7]. Empiric estimates of annual PE incidence in European countries range from 15 to 16 per 100,000 population [8]. According to the Ministry of Health of the Russian Federation, approximately 80,000 new VTE cases are reported annually in Russia, while the PE incidence is 35–40 per 100,000 people, i.e. 51,000–58,000 cases per year [1, 9].

Despite the fact that hospital mortality in acute PE reaches 9–15 %, 30-day all-cause mortality among patients with a high-risk PE ranges from 40 to 65 % [1, 3, 10]. The pulmonary artery obstruction significantly impairs the pulmonary blood flow and gas exchange. Depending on PE severity, right ventricular (RV) postload increases, right heart chambers dilate with the development of tricuspid regurgitation, increased tension of right heart chamber walls, and increased central venous pressure (CVP) [2, 11].

Due to high CVP and macro- and/or microcirculatory renal hypoperfusion, PE is often complicated by the acute kidney injury (AKI), which is accompanied

by the unfavorable prognosis even with mild and reversible renal dysfunction [12, 13]. Despite a relatively high PE incidence, the issue of acute renal dysfunction is not sufficiently analyzed, and we have considered it interesting to demonstrate a case of reversible AKI in the setting of PE.

Clinical Case Report

Patient Information

The female patient V., 74 years old, was hospitalized into the cardiological department of the Central City Clinical Hospital No. 1 (Donetsk) on January 14, 2025. Upon admission the patient complained of severe dyspnea at rest worsening with minimum physical exertion, significant general weakness, edema of thighs, legs, and feet. The patient also noted the decreased urine output within the preceding several days ("about a glass daily").

Medical history. Within 15 years she periodically suffered from pressure-like pain in the precordial region during physical exertion and blood pressure (BP) elevation to 200/100 mm Hg. She was followed up in the outpatient setting concerning the coronary artery disease (CAD), essential hypertension (EH); she did not take the recommended therapy regularly, although she used enalapril during BP elevation episodes. The patient reported that her usual BP values were 160–170/90–95 mm Hg.

Her latest exacerbation emerged on January 9, 2025, when significant dyspnea, general weakness, hypotension (100–80/70–50 mm Hg) emerged; the patient called the ambulance several times, but refused to hospitalize. On January 14, 2025 her dyspnea worsened, she again called the ambulance and was hospitalized into the cardiological department at 12:55 p.m.

History: in 1986, during the elective ultrasound examination of the abdominal cavity, the patient was diagnosed with a single right kidney, and the diagnosis of congenital unilateral agenesia of the left kidney was established.

Physical examination: severe condition. The patient was alert and oriented in herself, time and place. Normal constitution, overweight. Peripheral lymph nodes and the thyroid gland were not enlarged. Pupils: D=S. Skin and visible mucous membranes were clean. Moderate cyanosis of lips. SpO_2 95 % on room air. Respiratory rate (RR) 22–24/min. Chest percussion demonstrated the pulmonary sound over the whole lung surface; during lung auscultation, vesicular breathing with no rales was detected. Relative cardiac dullness borders: left — shifted 1.5 cm outwards from the left midclavicular line; right — 0.5 cm outwards from the right sternal border; upper — 3rd rib. Regular heart rhythm was auscultated with muffled heart sounds. Heart rate (HR) 65/min, BP 120/70 mm Hg. During palpation, the abdomen was soft and non-tender, but enlarged, with the dull percussion sound in the flanks. The bowel palpation was difficult due to a significant amount of subcutaneous fat. The liver protruded 2 cm from under the costal arch. Kidneys and the spleen were not palpable. Symmetric soft edema of thighs, legs, and feet.

Electrocardiogram (ECG) (January 14, 2025): regular sinus rhythm, heart rate 60 min^{-1}. Right axis deviation (α angle: +106°). $S_IQ_{III}T_{III}$ sign, negative T waves in II, III, aVF, V_1–V_6 (Fig. 1).

Common blood count (CBC): Hb 73 g/L, RBC 4.08×10^{12}/L, Ht 25.2 % (36–45 %), mean corpuscular volume 61.8 fL (80.0–96.1 fL), mean corpuscular hemoglobin 17.9 pg (27.5–33.2 pg), mean corpuscular hemoglobin concentration 29 % (33.4–35.5 %), RBC distribution width 21 % (11.5–14.5 %), platelet crit 0.213 % (1.0–10.3 %), CI 0.56 (0.85–1.0). Other CBC parameters were within reference ranges.

Biochemistry panel: total protein 58 g/L (65–85 g/L), urea 20.5 mmol/L (2.5–8.3 mmol/L), glucose 7.1 mmol/L (3.38–5.55 mmol/L).

Coagulation panel: prothrombin time (PT) 17 seconds (13–17"), prothrombin index 80 % (80–100 %), international normalized ratio 1.31. Semi-quantitative D-dimer test: positive.

Cardiac markers: troponin I (cTnI) (January 14, 2025; 1:10 p.m.) 12 ng/mL (<0.2 ng/mL).

Amino-terminal fragment of brain natriuretic peptide (NT-proBNP) 1056 pg/mL (<600 pg/mL).

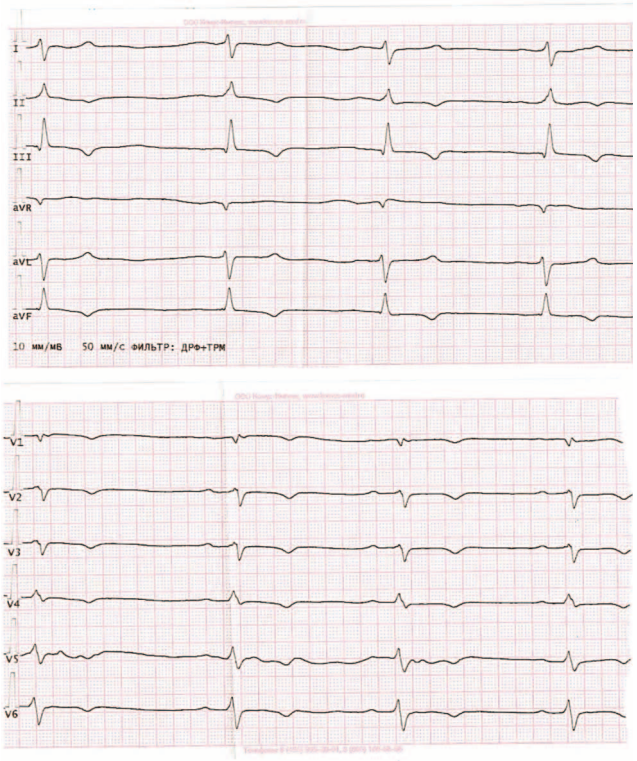

Figure 1. *Electrocardiogram on the day of admission (14.01.2025). Description in text*

Based on the aforementioned complaints, history, physical examination, laboratory tests, and ECG changes, the following preliminary diagnosis was established: 1) CAD: Acute non-ST elevation acute coronary syndrome (January 9, 2025; based on the history). Heart failure (HF), Stage I, with the undetermined left ventricular (LV) ejection fraction, functional class IV. Stage II, Grade 3 essential hypertension, risk of cardiovascular complications 4 (very high). 2) PE (January 9, 2025).

Concomitant diseases: Congenital unilateral agenesia of the left kidney.

The following treatment was administered upon admission: enoxaparin 60 mg subcutaneously twice daily; furosemide 40 mg intravenously once (since January 15, 2025 — furosemide 20 mg in the morning and afternoon); dexamethasone 8 mg intramuscularly once; spironolactone 25 mg in the afternoon; atorvastatin 40 mg in the evening; acetylsalicylic acid 100 mg in the evening; clopidogrel 75 mg in the morning; omeprazole 40 mg in the morning, 30 minutes before meals.

Next day (January 15, 2025), by 6:00 a.m. no diuresis was reported since the previous evening (for 10 h), and the patient's condition worsened (dyspnea worsened, the saturation and BP decreased). Physical examination: very severe general condition. The patient was alert and oriented in herself, time and place. Significant diffuse cyanosis. RR 34/min; the patient lies supine with no orthopnea; body temperature 36.5 °C, SpO_2 85 % (on room air), 95–96 % (with O_2 inhalations). Regular heart rhythm with muffled heart sounds. HR 60/min, BP 80/50 mm Hg. The abdomen was soft and non-tender on palpation; due to the severe patient's condition, the detailed palpation of abdominal organs was impossible. Symmetric edema of legs and feet; leg palpation was non-tender, with preserved pulses bilaterally. ECG (January 15, 2025; 6:15 a.m.): sinus regular rhythm, rate 64/min. Right axis deviation (α angle: +112°). $S_IQ_{III}T_{III}$ sign, negative T waves in leads III, V_1–V_6. Signs of right heart strain. Compared to ECG on January 14, 2025, the right axis deviation worsened (α angle increased from +106° to +112°), the Q_{III} wave became deeper (from 1.5 mm to 2.5 mm), and the depth of the negative $T_{V1–V6}$ increased as well, which were considered signs of RV strain worsening (Fig. 2).

Transthoracic echocardiography (EchoCG) (January 15, 2025; 8:30 a.m.): pulmonary artery pressure 50–54 mm Hg. Inferior vena cava dilation 3.29 cm, collapsing <50 % on inspiration. Right atrial volume index 85.83 mL/m² (<27 mL/m²), RV wall thickness 0.38 cm, proximal RV diameter 4.5 cm (<3.5 cm), EDV index 70.63 mL/m² (29–61 mL/m²). Dilated right heart chambers and LV. Paradoxical interventricular septum motion (sign of RV strain). Left atrial (LA) diameter 4.36 cm, LA volume index 27.92 mL/m², LV ejection fraction 55 %.

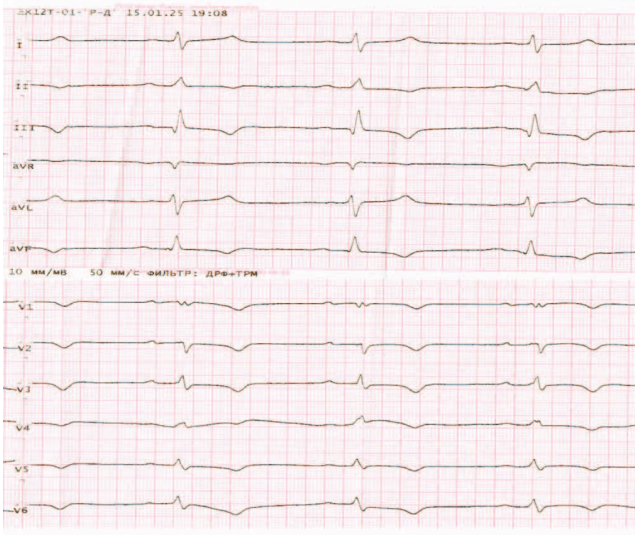

Figure 2. *Electrocardiogram at the time of impairment of the condition (01/15/2025, 06:15). Description in the text*

Ultrasound duplex scanning of veins in lower extremities was not arranged due to technical limitations.

Common blood count and biochemistry panel (January 15, 2025; 9:00 a.m.): WBC 15.14×10^9/L, 12 % lymphocytes, RBC 4.08×10^{12}/L, Hb 73 g/L, total protein 58 g/L, urea 23.1 mmol/L, alanine aminotransferase 304.8 mmol/L, aspartate aminotransferase 244.2 mmol/L, PT 24.4 sec. Urinalysis could not be arranged due to the absence of urine.

Based on the patient complaints of worsening dyspnea, significant weakness, physical findings (edema of lower extremities, hypotension, tachypnea), decreased SpO_2, ECG results (worsening right axis deviation, increased depth of pathological Q wave in lead III and T waves in chest leads), and EchoCG (pulmonary hypertension, inferior vena cava dilation, absence of its adequate inspiratory collapse, dilated right heart chambers), high-risk PE (PESI mortality risk 10–24.5 %) with the circulatory collapse (January 9, 2025) was diagnosed in the patient. Pulmonary hypertension (mean pulmonary artery pressure 54 mm Hg).

No urination for 10 hours with the significant drop in diuresis within the prior days, prolonged hypotension emerging in the setting of PE helped to establish the diagnosis of prerenal AKI, RIFLE class/AKIN Stage 3, in the setting of CKD: congenital unilateral agenesia of the left kidney.

Due to the very high-risk PE, the patient was administered thrombolysis with recombinant prourokinase in the total dose of 8 million U (2 million U as a bolus, 6 million U as an IV drip). The enoxaparin dose was increased to 80 mg subcutaneously twice daily (before the creatinine results turned in), while aspirin and clopidogrel were discontinued. After thrombolysis, the

patient's condition remained stably severe, with BP of 115/70 mm Hg, while dyspnea did not get worse.

The result of the creatinine test was received on January 15, 2025 on 12:30 p.m. — 540 μmol/L (N 30–110 μmol/L). The treatment administered was corrected: enoxaparin, spironolactone, and atorvastatin were discontinued, and a combined phospholipid + glycyrrhizinic acid drug was started (2.5 g twice daily, IV drip).

Coagulation panel (January 15, 2025): PT 24.4 sec, PTI 61%, fibrinogen 1.9 g/L, D-dimer 2,420 ng/mL (0–500 ng/mL).

On January 17, 2025 (Day 3 of inpatient hospitalization), the patient started to excrete urine (350 mL/day). Urinalysis: proteinuria (1.31 g/L), leukocyturia, casturia (see the Table for urinalysis changes).

During the period of January 17 to 21, 2025, the patient continued complaining of significant dyspnea at rest worsening with minimum activity, severe general weakness. Her condition was stably severe. Symmetric, soft edema of lower extremities spanned up to the middle of thighs; it was more prominent by the evening. The patient was on constant oxygen therapy, with SpO_2 95–97% (during O_2 inhalations), HR 70–84 bpm,

Table. *Dynamics of the patient's general urine analysis during her hospital stay*

Parameter	Date (day in hospital)				
	17.01.2025 (4)	20.01.2025 (7)	28.01.2025 (15)	31.01.2025 (18)	04.02.2025 (22)
Urine volume, ml/day	350	1200	2700	2400	1700
Relative density	l/u	l/u	1026	1018	1013
Protein, g/L	1,31	0,246	0,749	1,3	0,104
White blood cells, single in f/v	7-12	–	10-15	8-0	30-40
Red blood cells, single in f/v	–	–	10-12	1-2	–
Cylinders, single in f/v	6-9	–	–	–	–

Notes: l/u — little urine, f/v — field of view.

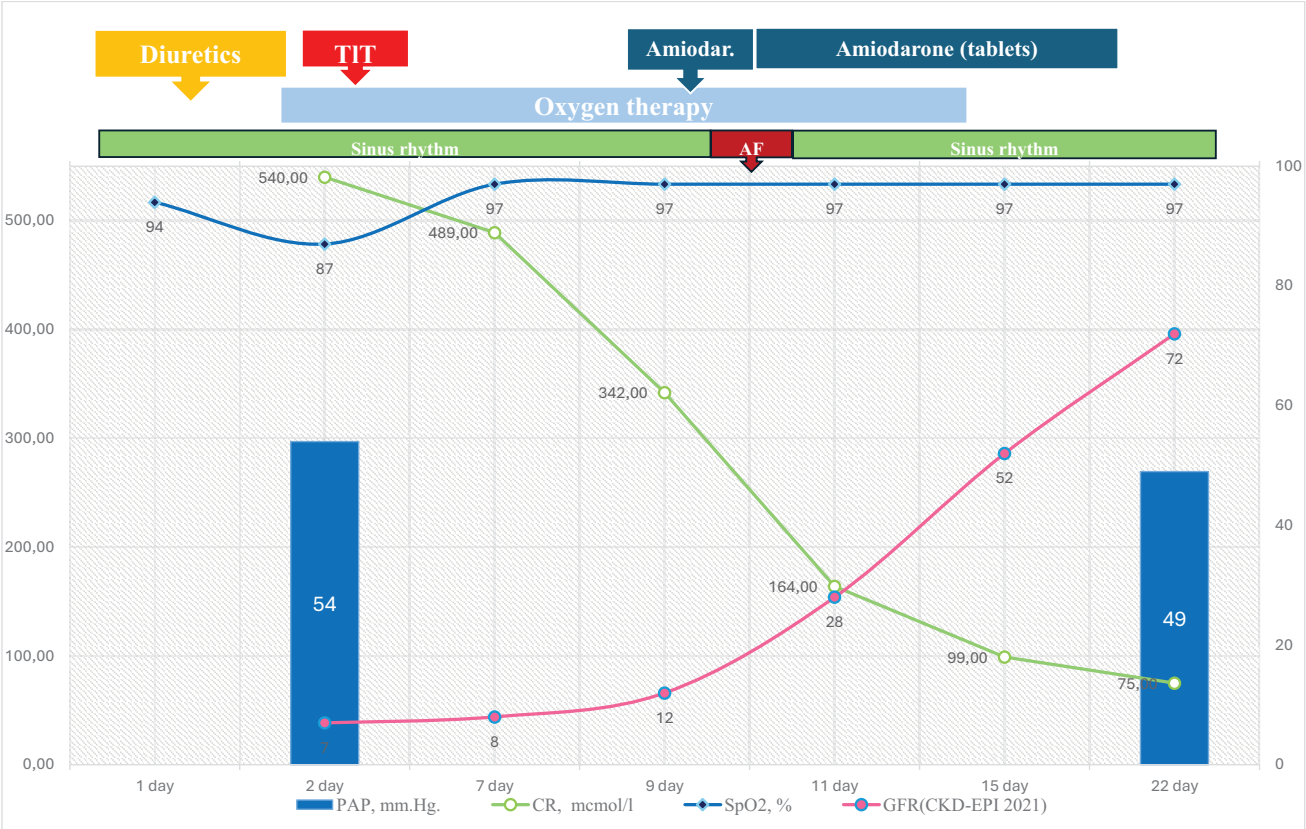

Figure 3. *Dynamics of laboratory and instrumental parameters of the patient during her hospital stay*

Notes: TIT — thrombolytic therapy, AF — atrial fibrillation, PAP — pulmonary artery pressure, SpO_2 — blood oxygen saturation level, %, GFR — glomerular filtration rate, CKD-EPI 2021 — formula for calculating GFR according to Chronic Kidney Disease Epidemiology Collaboration 2021, CR — creatinine, mcmol/

RR 20–22/min, BP 130–120/80–70 mm Hg. The diuresis restored since January 20 (800–1200 mL daily). See Figure 3 for changes in hemodynamic parameters, heart rhythm, blood saturation, common blood count and biochemistry panel results during the whole treatment period in the inpatient setting.

On Day 9 of inpatient treatment (January 22, 2025), the patient noted the improved well-being, while the dyspnea intensity decreased. Her condition was still stably severe. Physical examination revealed moderate cyanosis of lips. Vesicular breathing was auscultated in lungs. SpO_2 97 % (during O_2 inhalations), RR 20/min. Regular heart rhythm, with muffled heart sound. HR 85 bpm, $BP_D = BP_S$ = 120/70 mm Hg. Due to creatinine level decrease to 305 μmol/L and GFR of 17 mL/min, enoxaparin was again added to treatment (60 mg/day, one-time).

ECG (January 22, 2025): regular sinus rhythm, rate 85/min. Right axis deviation (α angle +109°). S_IQ_{III} sign, isoelectric T wave in leads II, III, aVF, weakly positive T wave in leads V_2–V_6. Compared to prior ECGs, negative T waves became flattened and weakly positive.

On January 23, 2025, at 8:05 a.m. (Day 10 of hospital stay), the patient noted decreased dyspnea intensity and improved well-being in the setting of general weakness, however she started complaining of palpitations. During the examination, irregular heart rhythm with the rate of 100/min was detected; RR 23/min. ECG (January 23, 2025; 8:25 a.m.): tachysystolic atrial fibrillation (AFib) (Fig. 4).

Amiodarone 300 mg (diluted in 0.9 % saline, 400 mL), IV drip infusion was started on 8:40 a.m. The paroxysm was stopped at 12:10 p.m., the patient noted that her condition improved, while the dyspnea intensity decreased; RR 19/min. ECG (January 23, 2025; 12:00 p.m.): irregular sinus rhythm due to atrial extrasystoles (bigeminy) with the rate of 120/min. Right axis deviation (α angle +102°). S_IQ_{III} sign, positive T waves in leads V_1–V_2 (Fig. 5).

During the treatment (amiodarone 300 mg IV drip one, followed by 200 mg orally three times daily; oxygen therapy; combined phospholipid + glycyrrhizinic acid drug 2.5 g/day; furosemide 20 mg twice daily; pantoprazole 40 mg; iron [III] hydroxide dextrane solution 2.0 mL twice daily), the patient's condition improved. The patient noted the disappearance of palpitations, while her dyspnea and general weakness improved;

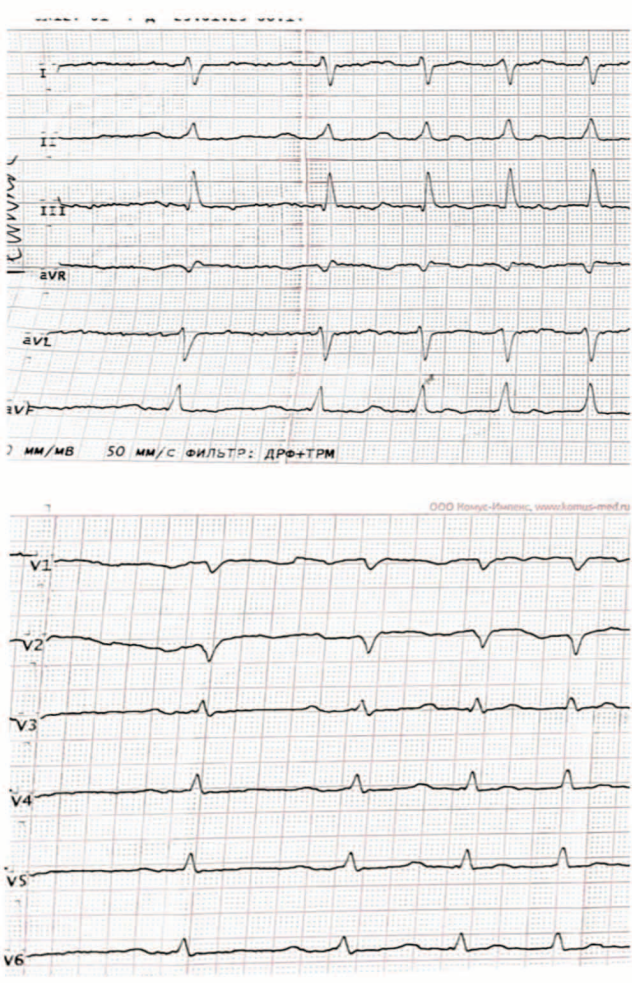

Figure 4. *ECG of the patient during arrhythmia (01.23.2025, at 08:25)*

Description: atrial fibrillation with ventricular rate (VR) of 140 min^{-1}. Right axis deviation (α angle +109°). S_IQ_{III} sign, weakly negative T wave in leads V_1–V_2.

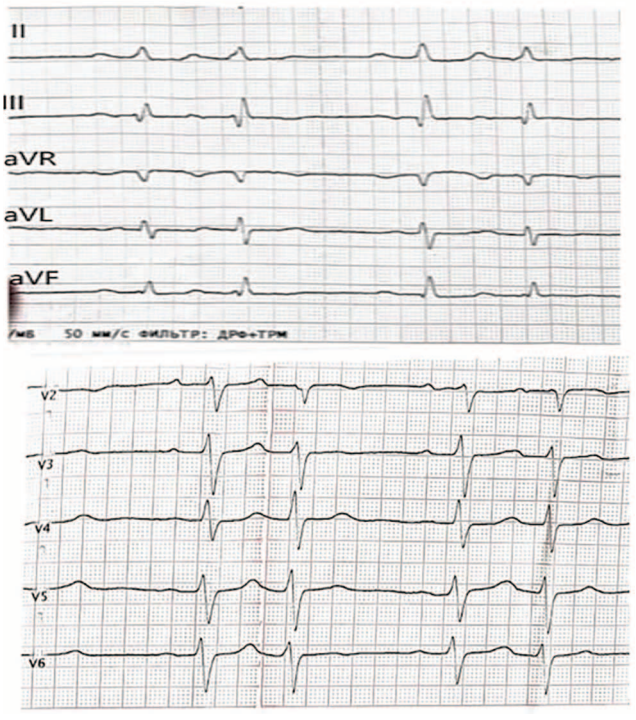

Figure 5. *ECG after abortion of an atrial fibrillation attack (01/23/2025, 12:00).*

Description: irregular sinus rhythm due to atrial bigeminy with heart rate of 120 min. The electric axe is deviated to the right (angle α +102°). S_IQ_{III}, positive T in V_1-V_2

the motor activity enhanced. Edema of thighs, legs, and feet improved. The patient preserved a sinus rhythm with HR 60–80/min, BP in the range of 120–130/76–80 mm Hg. SpO_2 98 % on room air, RR 18/min. Cardiac tones were regular, muffled. Vesicular breathing with no rales or crepitation was auscultated in lungs.

Laboratory tests before discharge: CBC (February 4, 2025): RBC 3.95×10^{12}/L, Hb 82 g/L, Ht 28.1 %, mean corpuscular volume 71 fL, mean corpuscular hemoglobin 20.7 pg, mean corpuscular hemoglobin concentration 29.2 %, RBC distribution width 15.4 %, platelets 148 g/L (150–450 g/L), platelet crit 0.141 %; all other parameters were within reference ranges.

Biochemistry panel: total protein 50 g/L, creatinine 75 μmol/L, serum iron 8 μmol/L (9–30 μmol/L), ferritin 8 μg/L (15–150 μg/L), transferrin 3.5 g/L (2.0–4.0 g/L), vitamin B_{12} 250 pg/mL (190–900 pg/mL); all other parameters were within reference ranges, including aminotransferase levels.

EchoCG (February 4, 2025): pulmonary artery pressure decreased from 54 to 49 mm Hg, LA volume index increased from 27.92 mL/m^2 to 52.75 mL/m^2, right atrial volume index decreased from 85.83 mL/m^2 to 64.75 mL/m^2.

On February 5, 2025 the patient was discharged under the monitoring of physicians at place of residence with the following diagnosis:

Main disease: High-risk PE (in-hospital mortality risk >15 %) with the circulatory collapse on January 9, 2025 (based on history), recurrent course. Condition after thrombolysis (prourokinase) on January 15, 2025. Pulmonary hypertension (mean pulmonary artery pressure 49 mm Hg based on EchoCG dated February 4, 2025) (ICD code I26).

Complications of the main disease: prerenal AKI (RIFLE Stage 3) dated January 14, 2025, with the renal function restored on February 4, 2025, in the setting of CKD Stage 2: agenesia of the left kidney combined with the hypertensive nephropathy of a single right kidney. Hypoxic hepatitis.

CAD: first-detected tachysystolic AFib paroxysm (January 23, 2025), drug-induced cardioversion (amiodarone) on January 23, 2025, CHA_2DS_2-VASc 4 points, HAS-BLED 5 points, HF 1 with preserved LV ejection fraction (61.99 % based on EchoCG dated February 4, 2025), functional class II. Essential hypertension, Stage II, Grade 3, Risk 4 (very high).

Moderately severe iron deficiency anemia.

She was recommended to continue regular follow-up at the general practitioner and the cardiologist. Outpatient diagnosis to determine the cause of anemia. EchoCG, Holter ECG monitoring to be arranged annually. Monitoring serum creatinine, iron, potassium levels; common blood count and urinalysis in 1 month. Salt-restricted (<5 g daily) and fluid-restricted (1–1.5 L daily) diet. Medications recommended: bisoprolol 2.5 mg in the morning, long-term treatment with HR and BP monitoring; lisinopril 2.5 mg daily; amiodarone 100 mg in the afternoon after 1 month with HR and ECG monitoring; rivaroxaban 20 mg daily during meals for 3 months with the following cardiologist counseling; iron [II] sulphate in combination with ascorbic acid 2 tablets daily with hemoglobin and serum iron level monitoring.

Discussion

PE is a variant of VTE, which comes third among the most common causes of cardiovascular mortality, giving way just to myocardial infarction and stroke [14]. Timely detection of acute PE and immediate initiation of anticoagulants, thrombolytics in combination with mechanical thrombectomy provides a significant decrease in mortality risks [15, 16]. PE manifestations may simulate a wide range of other conditions; thus, the most common cause of death in PE is the inability to confirm a correct diagnosis [14]. PE is often called a "great pretender", as this condition may be very complex for detection and final diagnosis. Patients with PE often demonstrate symptoms simulating acute myocardial infarction, HF, syncope caused by arrhythmias, pneumonia, influenza, asthma, panic attack, depression, or other diseases. The diagnosis of PE primarily requires that the physician includes it into the differential diagnosis; however, unfortunately, it is not uncommon that this life-threatening condition is not even considered a potential cause of symptoms in a patient [14, 17, 18].

Typical clinical PE manifestations usually included the sudden-onset complaints of variable dyspnea, chest pain, dizziness, presyncope and syncope, pain and edema of extremities, hemoptysis, cough, malaise, diaphoresis, and other symptoms [9, 19–23]. Based on T. Holder et al. [21] who analyzed the features of acute PE among 829 patients, the rate of presenting symptoms was as follows: dyspnea, 55.2 %; chest pain, 28 %; cough, 12.2 %; pain and edema of lower extremities, 8.8 % and 11.3 %, respectively; weakness, 8.9 %; presyncope and syncope, 8.3 %; hemoptysis, 2.3 %. One should not that dyspnea (38 %) and chest pain (20 %) were relatively rare in the high-risk patient subgroup (n=50), while hemoptysis and cough were absent completely. Based on the analysis of several reports by Doralisa and Vincenzo Morrone [20], clinical manifestations of acute PE were characterized by dyspnea (in 32–88 % cases), tachypnea (60–66 %), tachycardia (30–40 %), chest pain (40–70 %), syncope (6–39 %), cough (9–37 %), hemoptysis (2–13 %), and fever (7–10 %).

In our case the patient complained of dyspnea at rest, significant general weakness, edema of lower extremities. At the very onset of the disease (January 9, 2025), hypotension (80/60 mm Hg) was noted along with severe

dyspnea and general weakness, confirming the hemodynamic collapse. Despite multiple requests from the ambulance physicians, the patient refused the hospitalization. Unfortunately, the medical charts have not been presented, but it is very likely that the physicians considered the patient's condition worsened due to the exacerbation of the main disease, accounting for the history of CAD and EH. Even upon admission to the cardiology department on January 14, 2025, the diagnosis of PE was put second after the proposed ACS. The ambiguity concerning PE was associated with the complex exclusion of ACS in the setting of CAD and the absence of EchoCG results during the first day of hospital stay, although ECG already demonstrated right axis deviation, the $S_IQ_{III}T_{III}$ sign, negative T waves in chest leads. Only on January 15, 2025, when the patient's condition significantly worsened in the morning (due to increasing dyspnea, weakness), PE was diagnosed after obtaining the EchoCG results.

It should be noted that clinical PE manifestations are not always characterized by the combination of all complaints, i.e. dyspnea, chest pain, hemoptysis, palpitations. As presented above, such typical PE sign as dyspnea is not detected in all patients. Accounting for the PE severity and its potentially lethal prognosis, cases with the non-"classic" PE presentation (with the "classic" meaning the simultaneous combination of all hazardous signs) should be assessed very attentively [17, 23].

Despite the fact that PE is one of the leading causes of cardiovascular morbidity and mortality, insufficient attention is usually paid to the renal function assessment in this complication [24, 25]. Renal dysfunction is observed rather frequently in patients with PE, with the rate ranging from 5 % to 60 % [25, 26]. Based on the ICOPER registry covering 2,454 patients with acute PE, renal dysfunction (creatinine levels >176.8 μmol/L) was observed in 5.1 % cases, being an independent mortality predictor (HR 2.0; 95 % CI 1.4–3.0) [27]. When analyzing the association of various biomarkers and prognosis in 100 patients with acute PE, M. Kostrubiec et al. [28] detected renal failure (serum creatinine levels >135 μmol/L) in 13 patients (13 %); the hazard ratio (HR) for the renal failure-associated mortality was 6.4 (95 % CI 2.22–18.61). Based on the results of the meta-analysis (13 studies enrolling 35,662 patients), signs of renal failure were observed in each third patient (32.8 %) [29].

According to the large Russian SIRENA registry enrolling 604 patients with PE, renal dysfunction (assessed as glomerular filtration rate <60 mL/min/1.73 m^2) was detected in 320 (53 %) patients, with severe dysfunction in 63 (10 %) examined subjects [30]. AKI was diagnosed in 59.6 % PE patients in the study of P.F. Klimkin et al. [31]. Besides, AKI in patients with PE was associated with the severity of respiratory failure, systolic pulmonary hypertension, and RV dysfunction parameters.

In our cases PE course was complicated with AKI, defined as a sudden-onset renal function loss evaluated based on serum creatinine level increase and decreased diuresis (oliguria) lasting up to 7 days [32–34].

Several mechanisms are considered in the pathogenesis of AKI in the setting of PE. Acute right heart strain leads to tricuspid regurgitation and increased CVP, which leads to passive renal hyperemia, increased interstitial pressure, and renal interstitium edema [12, 25, 35]. To describe the renal disorder due to decreased renal venous blood flow and increased renal interstitial pressure, the term "congestive nephropathy" is used — it can be reversible if the venous circulation parameters are restored [25, 36]. The renal venous congestion activates the hormonal activation with enhanced sodium resorption, which leads to worsening volume overload, increased intraabdominal pressure, and (consequently) increased RV wall tension. Thus, decreased renal perfusion along with the worsening vascular congestion and, thus, increased CVP leads to renal function worsening in acute HF [35]. E.M. Boorsma et al. [37] proposed the "renal tamponade" hypothesis to explain the renal function worsening with increased CVP in HF. Increased CVP leads to the increased interstitial pressure in kidneys with the compression of renal structures (tubules, intrarenal vessels, glomeruli) in the encapsulated kidney, which is rather rigid for expansion.

Besides, acute PE with the drop in the stroke volume leads to renal hypoperfusion and hypoxia, neurohumoral activation enabling vasoconstriction and additional sodium resorption [12, 35]. Such concomitant diseases as diabetes mellitus, hypertension, CKD, congenital anomalies, are both risk factors for AKI, predisposing to renal injury in the setting of severe hemodynamic stress and factors worsening renal injury in the setting of hypoperfusion and congestive nephropathy [12, 25, 26, 38]. Thus, when analyzing AKI features in 36 patients with PE, V.V. Filimonova et al. [24] detected CKD in 24 (67 %), *de novo* AKI — in 12 (33 %) examined patients. Our patient was earlier diagnosed with a single kidney and a congenital agenesia of the left kidney, which, being a variant of CKD, predisposes to AKI [39].

AKI course in the case presented was characterized by the period of oliguria, azotemia (maximum creatinine level 540 μmol/L) in the setting of PE. The management of patients with PE and AKI presumes quick blood flow restoration in the pulmonary circulation, RV strain and CVP improvement, diuresis restoration. In our case, all nephrotoxic medications and drugs increasing the risk of adverse effects were discontinued after establishing the final diagnosis.

Besides severe renal dysfunction, acute HF and respiratory failure in the setting of PE in our case led to hypoxic hepatitis, manifesting with more than 10-fold elevated aminotransferase levels. According to the up-to-date information, hypoxic hepatitis is associated with complex disorders of hemodynamics and neurohumoral regulation; hepatocyte hypoxia is the initial cause of their injury [40]. The analysis of multiple studies helped to define the following mechanisms of hepatocyte hypoxia: ischemia and venous congestion (17–78 % cases of acute HF); hypoxemia (12 % cases of respiratory failure, anemia) [40]. Our patient had several mechanisms combined, including ischemia, venous congestion, and hypoxemia. Aminotransferase levels in hypoxic hepatitis typically normalize in 10–15 days, which was the case for our patient.

AKI in PE is associated with an unfavorable prognosis. In the study cited above, the analysis of PE features depending on the presence (n=36) or the absence (n=75) of AKI detected that 16 (44 %) patients with kidney injury and 26 (23 %) patients without AKI eventually died [24]. The in-hospital mortality risk was increased among patients with AKI: OR 5.2 (95 % CI 2.02–13.39; p <0.001). Besides, authors demonstrated that the risk of 30-day mortality based on the PESI scale in patients with AKI was higher than in patients with AKI (120.0 (87.5–158) and 90 (87.5–158.0), respectively; p=0.004) [24]. Based on the detected clear association between the decreased glomerular filtration rate and increased in-hospital mortality, the Russian SIRENA registry experts [30] propose to add the renal dysfunction (glomerular filtration rate <60 mL/min/1.73 m^2) to the simplified PE Severity Index (sPESI) in order to improve the risk stratification and identify patients with the high risk of in-hospital mortality.

Conclusion

The case presented illustrates the emergence of AKI in high-risk PE in a patient with a pre-existing congenital renal anomaly. The AKI course was characterized by oliguria and increased serum creatinine levels. AKI in PE is associated with several mechanisms, in particular with congestive nephropathy and severe hypoperfusion caused by the decreased stroke volume and hypotension. AKI in PE is associated with unfavorable prognosis evaluated based on the in-hospital mortality level. Knowing the renal dysfunction features provides the complex approach to the evaluation of the clinical status of PE patients, helps to monitor diuresis and serum creatinine levels, as well as to develop the differentiated algorithms of patient management.

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