Georgian Scientists
ქართველი მეცნიერები
Vol. 8 Issue 4, 2026
https://doi.org/10.52340/gs.2026.08.04.14



Infective Endocarditis Masked as Pulmonary Artery Thromboembolism – A Case Report

Nino Lekveishvili[2,3], Marina Kuparadze[1,2], Giorgi Imedashvili[1], Irina Lezhava[2], Anzor Melia[2], Teimuraz Mosiava[3]

[1]Tbilisi State Medical University, Tbilisi, Georgia; [2]Cardiology Clinic "Guli", Tbilisi, Georgia; [3]David Aghmashenebeli University of Georgia

Abstract

Introduction. In people who inject drugs, infective endocarditis primarily affects the right side of the heart and often presents as septic pulmonary embolism. This clinical presentation can be easily misdiagnosed as pulmonary embolism, pneumonia, or primary cardiomyopathy, leading to delays in specific treatment.

Case Description. A 50-year-old man who had been enrolled in a methadone maintenance program for four years was hospitalized with dyspnea at rest, orthopnea, fever, and lower-extremity edema; his oxygen saturation on room air was 85%. Echocardiography revealed dilation of all cardiac chambers, an ejection fraction of 18–20%, a pulmonary artery systolic pressure of 90 mmHg, and two mobile masses (17×8 mm and 17×7 mm) on the tricuspid valve leaflets. D-dimer levels exceeded 4000 ng/mL, and computed tomography angiography revealed bilateral segmental filling defects against a background of cavitating nodular lesions; the veins of the lower extremities were fully compressible. Antibiotic therapy remained empirical throughout, with the regimen changed three times. Despite 16 days of conservative treatment, fever, vegetations, and elevated inflammatory markers persisted, prompting the patient's transfer to a specialized clinic. There, preoperative echocardiography showed that the ejection fraction had already improved to 52%; coronary angiography ruled out hemodynamically significant coronary artery disease, and the tricuspid valve was replaced with a biological prosthesis. The patient was discharged in improved condition, and nine months later, heart failure corresponded to Functional Class II.

Conclusion. Right-sided infective endocarditis should be considered in the differential diagnosis of any patient with a history of injection drug use, fever, and multiple focal pulmonary lesions-even when computed tomography shows filling defects in the pulmonary artery. Severe systolic dysfunction in these patients may be partially reversible and should not, in itself, be a reason to decline surgical intervention.

Keywords: Infective endocarditis; tricuspid valve; septic pulmonary embolism; bioprosthetic valve; congestive heart failure.

Introduction

Infective endocarditis remains a disease with high mortality, and its management requires the coordinated efforts of a cardiologist, an infectious disease specialist, and a cardiac surgeon [1].

People who inject drugs constitute a distinct clinical group. According to a scientific statement from the American Heart Association, endocarditis in this population primarily affects the right-sided heart valves; it is characterized by a high rate of recurrence and requires not only antimicrobial and surgical treatment but also concurrent, systematic management of opioid dependence [2].

A retrospective comparative study conducted in Bucharest showed that among people who inject drugs, the tricuspid valve was involved in 72.5% of cases, the prevalence of hepatitis C reached 98.1%, and the 12-month mortality rate was significantly higher than that of the non-user group (27.5% versus 13.7%). A five-year observational study of a Finnish cohort confirmed that right-sided cardiac involvement predominates in these patients (62.7%), with the tricuspid valve affected in 70.7% of cases; furthermore, a new episode of endocarditis develops in 20% of patients within the five-year period [3,4].

The clinical presentation of right-sided endocarditis is often respiratory, with fever, dyspnea, and multiple focal pulmonary lesions. Radiologic signs of septic pulmonary embolism include bilateral opacities (100%), nodular lesions (73.9%), and cavitation (57.9%). This clinical picture creates a diagnostic trap: elevated D-dimer levels and filling defects on computed tomography (CT) suggest pulmonary embolism, whereas the actual source of the emboli is a vegetation on the right side of the heart. [5]

An additional challenge arises from the inability to verify microbiologically: up to 30% of endocarditis cases present with negative blood cultures, largely because antibiotic therapy is initiated before diagnostic testing. In such scenarios, treatment relies on empirical regimens, and the assessment of therapeutic efficacy is based on monitoring clinical status, echocardiographic findings, and inflammatory markers. [6]

The case presented here is notable because it encompasses all three of these issues simultaneously: a "respiratory mask" (presentation mimicking a respiratory condition) for endocarditis, the absence of microbiological verification, and a critically reduced ejection fraction-which was initially misidentified as an independent cardiomyopathy. The case is further distinguished by the fact that the patient received sequential treatment at two different institutions, using distinct approaches: conservative management and surgical intervention.

Case Presentation

A 50-year-old male, unemployed; living conditions are unsatisfactory. Physical examination reveals excessive subcutaneous adipose tissue; palpation of the liver and spleen was not possible due to obesity.

Reason for referral and primary complaints: shortness of breath (dyspnea) occurring with minimal exertion and at rest, palpitations, the need to remain seated (orthopnea), and swelling of the lower extremities, and a sensation of suffocation.

Approximately one month before the current hospitalization, the patient was treated at a district hospital for pneumonia. During that same period, he was diagnosed with dilated cardiomyopathy for the first time and was subsequently discharged in improved condition. After discharge, his condition deteriorated sharply: physical activity became severely limited-manifesting as intense dyspnea after even a few steps-along with difficulty breathing, rapid heartbeat (tachycardia), and swelling of the lower extremities.

History of prior hospitalization and refusal of surgical treatment. On May 28, 2024, the patient was admitted to a regional hospital, where the following diagnoses were established: acute endocarditis, unspecified anemia, unspecified thrombocytopenia, specified pleural conditions, acute respiratory failure, chronic viral hepatitis C, atherosclerosis of the limb arteries, unspecified pneumonia, tricuspid valve insufficiency, and heart failure. The patient received a transfusion of red blood cell mass. A cardiac surgical intervention on the tricuspid valve was recommended; however, the patient declined the procedure and was discharged from the hospital in improved condition.

A few days after discharge, the patient developed chills, tremors, fever, and difficulty breathing. By the morning, these symptoms were accompanied by a sensation of suffocation, prompting the patient's family to take them to the diagnostic center of the "First Clinic." The patient's condition was assessed as acute, and emergency hospitalization was initiated.

Medical history: arterial hypertension; mental and behavioral disorders due to opioid use with dependence syndrome, currently in remission while undergoing substitution therapy (enrolled in a methadone program since 2020); chronic viral hepatitis C; inpatient treatment in 2021 for acute respiratory failure, hemoptysis, and unspecified anemia; unspecified pneumonia; unspecified thrombocytopenia; atherosclerosis of the limb arteries; tricuspid valve insufficiency; heart failure, functional class IV (as of May 28, 2024).

In 2023, the patient completed a six-month course of treatment for hepatitis C as part of the elimination program, finishing in December 2023. The patient’s family history is significant for cardiovascular disease.

Clinical Findings

Upon hospitalization, the general condition was assessed as severe; the patient's position in bed was forced; consciousness was clear; reflexes were brisk; no meningeal signs were present; and sensation was intact.

The skin was cold, with markedly reduced turgor, and appeared pale and cyanotic; the visible mucous membranes were also pale and cyanotic; the sclerae were normal; no rash was noted. Pronounced edema of the extremities was present. A trophic ulcer was present over the medial malleolus of the right lower extremity. Subcutaneous adipose tissue was markedly developed. Peripheral lymph nodes and the thyroid gland were not palpable.

Cardiovascular system: Pulse 97 beats per minute; blood pressure 160/80 mmHg; heart sounds were muffled; a systolic murmur was heard.

Respiratory system: Respiratory rate 26–28 breaths per minute; oxygen saturation 85%; percussion reveals dullness bilaterally. Auscultation reveals diminished breath sounds in the lower lobes bilaterally, extending below the inferior angle of the scapula; fine, moist rales (suggestive of congestion) and crepitations are present in the middle and lower lung fields bilaterally.

Digestive system: The tongue is moist, swallowing is unimpaired, and the abdomen is soft; the liver and spleen could not be palpated due to excess body weight. Urogenital system: The kidneys were not palpable on bimanual examination; the costovertebral angle tenderness test (tap sign) was negative bilaterally.

Body temperature upon admission was 36.4 °C. During hospitalization, temperature was monitored every two hours; persistent subfebrile temperatures were recorded, with occasional episodes of chills, shivering, and hectic (spiking) fevers.

At the time of discharge from the initial clinical facility: blood pressure 120/70 mmHg; pulse 80, regular; respiratory rate 18 breaths per minute; oxygen saturation 98% on room air; temperature 36.5 °C; heart sounds muffled but regular. Breath sounds are diminished in the lower lung lobes; the abdomen is non-tender; the lower extremities are free of edema, with elastic stockings in place. Despite normalization of vital signs, the condition was assessed as "prognostically severe and unfavorable."

At an outpatient follow-up visit nine months later: pulse 62, regular; blood pressure 130/70 mmHg; heart sounds are muffled; vesicular breath sounds in the lungs are slightly diminished; the lower edge of the liver does not extend beyond the costal margin; the spleen is not palpable; the "tap" sign (costovertebral angle tenderness) is negative bilaterally; the lower extremities are free of edema; and dorsalis pedis pulsation is preserved bilaterally.

Diagnostic assessment

At admission, the primary clinical syndrome was congestive heart failure involving both the systemic and pulmonary circulations. A record from the previous month documenting dilated cardiomyopathy-along with echocardiographic evidence of dilation in all cardiac chambers and a critically reduced ejection fraction-naturally pointed to a diagnosis of primary cardiomyopathy. Consequently, infective endocarditis was not initially considered, even though the echocardiography report from that same day already described two mobile masses on the tricuspid valve leaflets.

The patient presented with oxygen desaturation, bilateral hydrothorax, and pulmonary hypertension, prompting an active search for pulmonary causes. An elevated D-dimer prompted a computed tomography (CT) angiogram, and a duplex ultrasound of the lower-extremity veins was performed to identify a potential source of embolism. The simultaneous presence of three findings shifted the diagnostic focus: large, mobile masses on the tricuspid valve; multiple nodular lesions in the pulmonary parenchyma (with basal cavitation)-interpreted by the radiologist as secondary lesions or "septic emboli"; and the absence of deep vein thrombosis in either lower extremity. This combination pointed to septic pulmonary embolism-rather than classic thromboembolism-as the most likely mechanism. A formal diagnosis of infective endocarditis was established on the second day of hospitalization, following an assessment by an infectious disease specialist.[5]

Laboratory studies

At the first clinic, laboratory monitoring was conducted daily. Table 1 presents the dynamics of key indicators on the dates when a complete blood panel was obtained.

Table 1. Dynamics of key laboratory parameters in the first clinic

Test (unit)	31.05	01.06	03.06	06.06	08.06	11.06	14.06	Reference range
Leukocytes ($\times 10^9$/L)	7.64	8.32	11.72	7.34	12.96	6.04	5.98	4.0–10.0
Hemoglobin (g/L)	75	75	82	68	81	81	77	110–165
Erythrocytes ($\times 10^{12}$/L)	2.56	2.48	2.66	2.27	2.69	2.80	2.71	3.8–5.8

Hematocrit (%)	22.84	21.82	23.85	20.69	24.07	25.22	23.64	35.0–50.0
Platelets (×10⁹/L)	165	166	267	121	161	139	121	100–300
Erythrocyte sedimentation rate (mm/h)	50	90	80	88	70	120	110	<40
C-reactive protein (mg/L)	205	–	131.8	215	250	203.8	200	<5.2
Procalcitonin (ng/mL)	–	–	–	0.47*	1.13	–	0.31	0.0–0.5
Creatinine (µmol/L)	73	–	83	151	124	91	83	<115
Sodium (mmol/L)	–	–	137.0	140.0	136.0	–	130.4†	135–155
Potassium (mmol/L)	–	–	2.38	3.03	3.66	–	4.14†	3.6–5.5
International normalized ratio	1.54	–	–	–	1.79	–	1.39	0.9–1.15 (without anticoagulant therapy)
Activated partial thromboplastin time (s)‡	42.5	68.1	75.0	–	47.1	–	39.4	19.5–36
Fibrinogen (mg/dL)	237	–	–	–	321	–	370	150–400
Albumin (g/L)	–	18.6	–	–	–	–	–	34–50
D-dimer (ng/mL)	>4000	–	–	–	–	–	–	<200
Troponin I (ng/mL)	0.62	–	–	–	–	–	–	0.0–0.3

Inflammatory markers. On the first day of hospitalization, C-reactive protein (CRP) was 205 mg/L (reference range <5.2 mg/L); it peaked at 250 mg/L and remained above 200 mg/L until discharge. This persistence-despite dual antibiotic therapy-served as the primary laboratory evidence of inadequate control of the infectious focus, prompting two changes to the antimicrobial regimen. Procalcitonin, a more specific marker for bacterial infection, measured 0.47 ng/mL; it rose to 1.13 ng/mL over three days, then fell to 0.31 ng/mL by day five. The divergent behavior of these two markers is clinically significant: the decline in procalcitonin suggested partial control of the bacteremia, whereas persistently elevated C-reactive protein and unchanged vegetations confirmed that the primary infectious focus remained active. The erythrocyte sedimentation rate (ESR) remained elevated throughout the period, reaching a maximum of 120 mm/h on June 11; however, in the context of severe anemia, this parameter loses its independent diagnostic value.

Hematological parameters: Hemoglobin levels were 75 g/L at the time of hospitalization, fell to a minimum of 68 g/L, and recovered to 81 g/L following a transfusion; however, they never approached the lower limit of the normal range (110 g/L). Severe anemia, occurring alongside a critically reduced ejection fraction, poses a compounded risk: low cardiac output and reduced blood oxygen-carrying capacity exacerbate one another, keeping tissue oxygen delivery at a critical threshold. Platelet counts fluctuated between 121 and 267×10^9/L, with no critical decline observed despite prolonged heparin infusion. The leukocyte count was within the normal range upon admission (7.64×10^9/L) and increased later to 12.96×10^9/L. This observation is noteworthy, as a normal white blood cell count (leukocytogram) does not rule out active infective endocarditis; had C-reactive protein levels not been measured at this stage, the true extent of the inflammatory process would have gone unnoticed.

Protein metabolism and nutritional status. On June 1st, the albumin level was 18.6 g/L (within the normal range of 34–50 g/L). This degree of hypoalbuminemia reflects both prolonged inflammatory catabolism and nutritional deficiency; it contributes to the persistence of edema and fluid accumulation in body cavities-even despite diuretic therapy-and serves as an independent factor that increases surgical risk.

Electrolytes and renal function. Intensive diuretic therapy led to two predictable, yet dangerous, consequences. First, persistent hypokalemia: potassium levels dropped to 2.38 mmol/L (on June 3rd) and repeatedly fell outside the reference range in the following days, necessitating daily correction via potassium chloride infusions. The arrhythmogenic potential of this hypokalemia was particularly high in this patient, as an electrocardiogram during the same period revealed a prolongation of the QT interval to 518 milliseconds. Second, a transient deterioration in renal function: creatinine levels rose from 73 to 151 μmol/L, then recovered to 83 μmol/L after dosage adjustments. During the second week of treatment, hyponatremia developed (reaching a minimum of 130.4 mmol/L)-a finding considered an unfavorable prognostic sign in the context of severe heart failure.

Markers of coagulation and embolism. The D-dimer level exceeded 4000 ng/mL (against a reference range of <200 ng/mL), which served as the indication for computed tomography (CT) angiography. It is important to note a key point here: this marker is non-specific and is equally consistent with both thrombotic and septic embolism; therefore, it confirms the occurrence of an embolism but not its etiology. During heparin infusion, the activated partial thromboplastin time (aPTT) fluctuated between 32.9 and 122.6 seconds (based on 18 separate measurements), indicating frequent titration of the infusion rate; no hemorrhagic complications were observed. Following the transition to warfarin, the International Normalized Ratio (INR) reached a maximum of 1.79 and never approached the target range.

Myocardial and hepatic markers. Upon admission, the Troponin I level was 0.62 ng/mL (reference range: 0.0–0.3 ng/mL). In this context, such a mild elevation is most consistent with acute right ventricular overload and a systemic inflammatory response, rather than acute coronary syndrome-a hypothesis subsequently confirmed by coronary angiography. Transaminase levels were within the normal range (alanine aminotransferase: 18.9 IU/L; aspartate aminotransferase: 30.2 IU/L), which argues against acute hepatic injury in the setting of chronic hepatitis C. Meanwhile, mild hyperbilirubinemia-predominantly involving the direct fraction (total: 25.6 µmol/L; direct: 9.06 µmol/L)-is highly consistent with hepatic venous congestion secondary to right ventricular failure.

Gas exchange. Blood gas analysis revealed a partial pressure of oxygen of 33.9 mmHg (reference range: 83–108 mmHg), with normal acid-base balance and a normal partial pressure of carbon dioxide. Even after two weeks of treatment, this profound hypoxemia confirmed a persistent impairment of gas exchange-attributable to a residual left-sided hydrothorax and a focal pulmonary lesion-and, despite the apparent stabilization of vital signs, it was one of the reasons why the patient's condition was still assessed as "prognostically severe" at the time of discharge.

Figure 1. Dynamics of inflammatory markers in the first clinic (31.05.2024–16.06.2024)

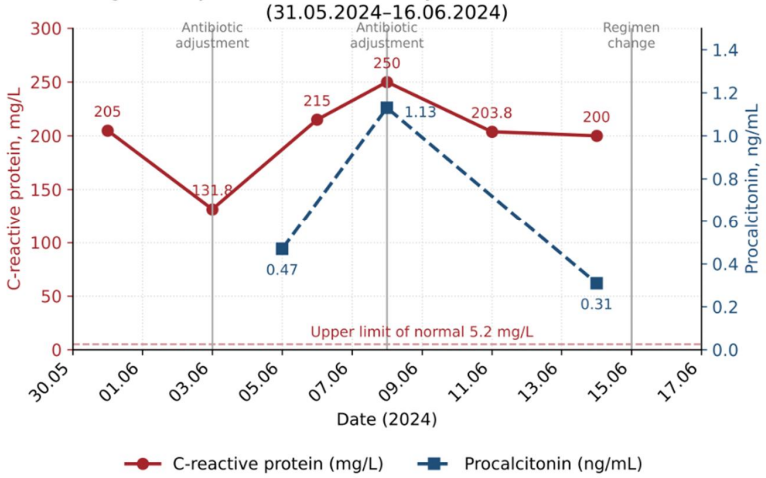


Figure 1. Dynamics of inflammatory markers in the first clinic. C-reactive protein fell from 205 to 131.8 mg/L within three days of starting dual antibiotic therapy, but then rose again, peaking at 250 mg/L on

08.06.2024, together with the procalcitonin peak. Vertical lines mark the dates of antimicrobial regimen changes. Despite three adjustments, the value never fell below 200 mg/L, which became the main laboratory argument for inadequate control of the infectious focus.

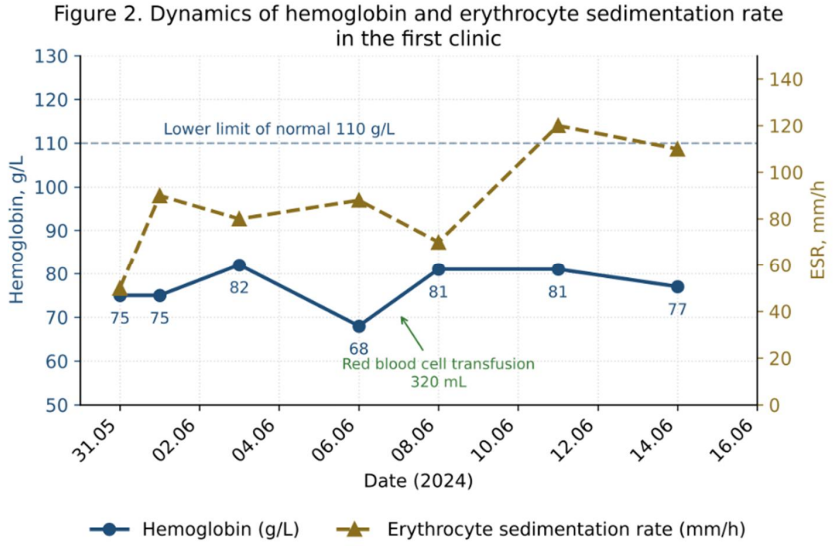


Figure 2. Dynamics of hemoglobin and erythrocyte sedimentation rate in the first clinic. The hemoglobin nadir (68 g/L) was recorded on 06.06.2024 and prompted transfusion of 320 mL of packed red blood cells; after transfusion the value recovered to 81 g/L but never approached the normal range. The erythrocyte sedimentation rate remained elevated throughout the period, peaking (120 mm/h) on 11.06.2024.

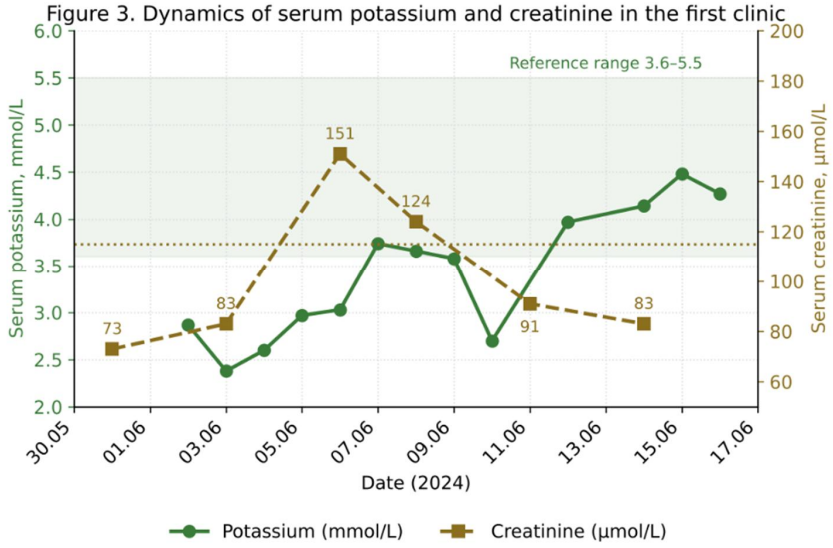


Figure 3. Dynamics of serum potassium and creatinine in the first clinic. During intensive diuretic therapy, the potassium level repeatedly fell below the reference range (minimum 2.38 mmol/L) and required daily correction; the creatinine peak (151 µmol/L) coincided with the hemoglobin nadir and reflects a transient deterioration in renal function, which fully recovered after dose adjustment.

Instrumental examinations

Electrocardiography. Sixteen examinations were performed during the hospitalization. Throughout this period, sinus rhythm was maintained (heart rate ranging from 68 to 97 bpm), with a leftward deviation of the heart's electrical axis and incomplete R-wave progression in the precordial leads. Clinically significant changes included the appearance of negative T-waves in leads V2–V6 (starting June 5th) and a prolongation of the QT interval to 518 milliseconds; this coincided with a period of severe hypokalemia, further exacerbating the risk of arrhythmia. By the time of discharge, the QT interval had shortened to 414 milliseconds. In an outpatient follow-up assessment nine months later, a block of the anterior fascicle of the left bundle branch (left anterior fascicular block) was noted for the first time.

Chest Radiography. The bronchovascular markings are accentuated, the hila appear dense, transparency is reduced in the lower lung fields, and the costophrenic angles are obscured. The aortic arch is dilated, the left cardiac border is enlarged, and the "waist" of the heart is filled in. 13.06.2024: Lung transparency is reduced in the middle and lower zones, more pronounced on the left, and the transverse dimensions of the heart are increased. The radiographic findings correlate with echocardiographic data indicating persistence of a left-sided hydrothorax.

Computed Tomography (CT) Angiography: This examination is the key imaging study for this case. Findings include: multiple, scattered nodular foci of varying sizes and shapes throughout the pulmonary parenchyma, with small areas of cavitation in the basal regions; bilateral, faint infiltration and a small pleural effusion; subsegmental thickening of the bronchial walls; mediastinal lymph nodes measuring 7–11 mm with preserved internal structure; and a small amount of effusion within the pericardial recesses. Vascular dimensions: pulmonary trunk – 36 mm, left pulmonary artery – 31 mm, right pulmonary artery – 30 mm, ascending aorta – 38×35 mm, descending aorta – 29×28 mm. After contrast administration, filling defects caused by thrombus masses were observed bilaterally within the segmental lumina of the pulmonary arteries. The radiologist noted that the parenchymal changes were "highly suggestive of secondary lesions and a pattern consistent with septic emboli." This presentation-a combination of cavitating nodular foci and segmental filling defects-aligns with the established radiological appearance of septic pulmonary embolism [5].

Duplex sonography of the lower extremity veins: In both limbs, the external iliac, common femoral, superficial femoral, deep femoral, and popliteal veins are patent and fully compressible. Spectral Doppler interrogation of the common femoral veins (bilaterally) showed a pulsatile flow pattern synchronous with the cardiac cycle, in place of the normal respiratory phasicity. There is insufficiency of the saphenofemoral junction valve. Synechiae are visualized within the left femoral veins. Multiple enlarged lymph nodes are present bilaterally in the inguinal region. No acute deep vein thrombosis was detected-a finding that directly contradicts the mechanism of classic venous thromboembolism and instead supports a cardiac, septic source. Coronary

angiography. An urgent examination performed at the second clinic revealed no hemodynamically significant lesions of the coronary arteries. This ruled out the working hypothesis formulated at the first clinic-"cardiomyopathy, unspecified, of possible ischemic origin"-and shifted the diagnostic focus toward non-ischemic causes of left ventricular dysfunction.

Other investigations. Additional studies included computed tomography (CT) of the head and brain, CT of the chest and abdomen, abdominal ultrasound, Doppler ultrasound of the arteries of the neck and limbs, as well as the veins of the lower extremities, and assessment of the thyroid. hormonal profile, rapid testing for syphilis, hepatitis B, and human immunodeficiency virus (HIV), an indirect Coombs test, and three separate measurements of vancomycin blood concentration.

Echocardiographic progression. Table 2 summarizes all eight echocardiographic examinations performed across the two medical facilities. The severity of valvular regurgitation is indicated by the notation used in the original records.

Table 2. Dynamics of echocardiographic findings in the two clinics

Date / Clinic	Ejection fraction	Tricuspid vegetation	Tricuspid regurgitation	Pulmonary artery systolic pressure	Fluid in cavities and other findings
31.05.2024 / First clinic	18–20% (fractional shortening 20%); left ventricle 76/69–68 mm; left atrium 60 mm; right ventricle 52 mm; right atrium 52 mm	17×8 mm and 17×7 mm, mobile	Grade IV	90 mmHg	Hydrothorax: right 650 mL, left 350 mL; pericardium 1–3 mm; aortic regurgitation grade I; mitral regurgitation up to grade III; calcification of the aortic annulus, non-coronary cusp and mitral annulus

Date / Clinic	Ejection fraction	Tricuspid vegetation	Tricuspid regurgitation	Pulmonary artery systolic pressure	Fluid in cavities and other findings
03.06.2024 / First clinic	Critically reduced; regional wall-motion abnormality in the territories of the left anterior descending and circumflex arteries	17×8 mm and 17×7 mm, unchanged	Grade IV	55 mmHg	Left hydrothorax up to 200 mL
05.06.2024 / First clinic	18–20% (fractional shortening 10%); end-diastolic volume 306 mL, end-systolic volume 248 mL	17×8 mm and 17×7 mm, hyperechoic, floating	Grade IV	45 mmHg	Inferior vena cava 20 mm, collapse >50%; left hydrothorax up to 200 mL
10.06.2024 / First clinic	No numerical value; right atrium 60/80 mm, right ventricle 60 mm	On the septal leaflet 18×8 mm, floating into the right atrium; on the anterior leaflet 13–14 mm	Grade IV	45 mmHg	Dilated pulmonary trunk 35 mm; left hydrothorax 500 mL with fibrin strands; small-volume ascites; mitral regurgitation grade II–2.5
12.06.2024 / First clinic	No numerical value	Not described	Not described	40 mmHg	Left hydrothorax 300 mL with fibrin strands; small-volume ascites

Date / Clinic	Ejection fraction	Tricuspid vegetation	Tricuspid regurgitation	Pulmonary artery systolic pressure	Fluid in cavities and other findings
14.06.2024 / First clinic	32% (33% in the discharge summary); right ventricular systolic function reduced, tricuspid annular plane systolic excursion 10 mm	On the septal leaflet, floating, 16×6 mm; on the anterior leaflet, fixed, 10 mm	Grade IV (severe)	50 mmHg	Pericardial separation 12 mm at the posterior wall and 8–9 mm laterally; inferior vena cava 34 mm, non-collapsing; left hydrothorax 350–400 mL; peak aortic gradient 20 mmHg; gallbladder stones
17.06.2024 / Second clinic	52%; grade I diastolic dysfunction; paradoxical interventricular septal motion	Floating echogenic structure 0.89×0.75 cm; an echogenic structure also at the basal segment of the posterior mitral leaflet	Moderate	Moderate pulmonary hypertension (no numerical value)	Pericardial separation 1.74 cm at the inferior wall and 2.3 cm laterally; left pleural separation 5.6 cm; mildly dilated aortic bulb
25.06.2024 / Second clinic	Described only as “positive dynamics”; no numerical data	–	–	–	Postoperative follow-up examination

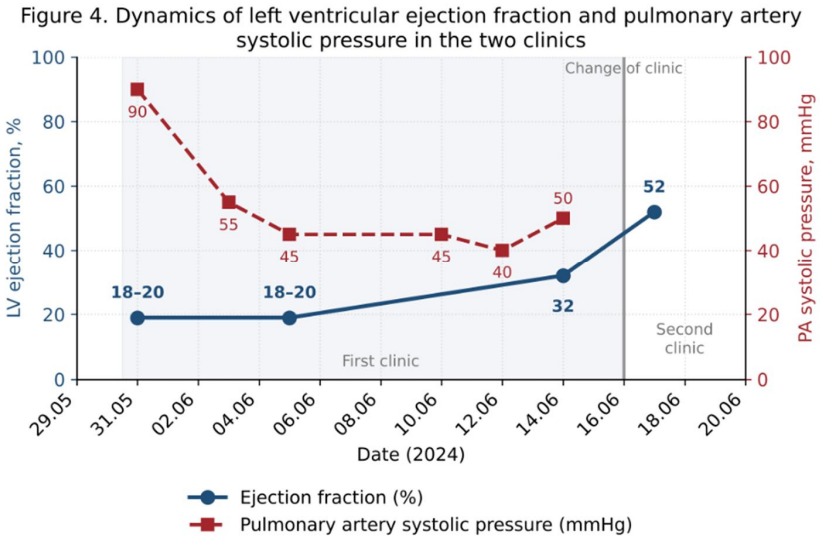


Figure 4. Dynamics of left ventricular ejection fraction and pulmonary artery systolic pressure in the two clinics. Pulmonary artery systolic pressure fell from 90 to 55 mmHg in the first days of dehydration therapy and subsequently remained in the 40–50 mmHg range. The ejection fraction increased from 18–20% to 32% in the first clinic and reached 52% in the second clinic on the day before surgery. The vertical line marks the transfer between institutions (16.06.2024).

Treatment at the First Clinic

Status at Admission and Initial Strategy. The patient was hospitalized in a severe condition, presenting with respiratory failure and marked congestion in both circulatory circuits. The strategy for the first 24 hours focused on stabilizing hemodynamics: dehydration therapy, correction of electrolyte imbalances, and oxygen therapy were initiated. In the admission records, the patient's condition was classified as "unstable."

Anticoagulation. On the same day as the CT angiography, an intravenous infusion of heparin was started (the record identifies the medication by its trade name, specifying heparin at 5000 IU/mL, 5 mL), with monitoring of the activated partial thromboplastin time (aPTT). This monitoring was performed several times a day-a total of 18 measurements were taken. The values fluctuated widely, from 32.9 to 122.6 seconds, indicating frequent titration of the infusion rate. Subsequently, warfarin (2.5 mg tablets) was added to the treatment regimen. The International Normalized Ratio (INR) failed to reach the target range throughout the entire period (maximum 1.79).

Antimicrobial therapy and its three subsequent adjustments: Following an assessment by an infectious disease specialist, dual empirical antibiotic therapy was initiated: Cefepime (2 g three times daily) and Moxifloxacin (400 mg, administered as a 400 mg/250 ml infusion). Moxifloxacin was subsequently replaced by Vancomycin (1 g twice daily). Due to elevated inflammatory

markers, Cefepime was replaced with Meropenem (1 g vial). Based on the infectious disease specialist's recommendation, the final regimen was established as Meropenem and Vancomycin.

Treatment for heart failure included: Furosemide (1%, 2 ml), Spironolactone (25 mg tablet), diuretics of the Torasemide class (10 mg tablet and 20 mg/4 ml solution), an Enalapril-containing medication (5 mg tablet), a combined potassium and magnesium asparaginate preparation, 4% Potassium Chloride (200 ml), and 0.9% Sodium Chloride solutions. Blood transfusion. Against a background of stable hemodynamics and following appropriate premedication, 320 ml of red blood cell concentrate-matched for blood group and Rhesus factor-was transfused without complications. Blood group: B (III), Rhesus factor: positive.

The patient's subjective condition improved, and signs of congestion partially subsided; systolic pulmonary artery pressure decreased from 90 mmHg to 40–50 mmHg, while the ejection fraction increased from 18–20% to 32%. At the same time, control over the infectious focus was not achieved: low-grade fever persisted, accompanied by periodic hectic fevers with chills; C-reactive protein levels remained above 200 mg/L, and the size of the vegetations did not significantly decrease. Three significant complications were observed: worsening anemia (hemoglobin dropping to 68 g/L), transient deterioration of renal function (creatinine rising to 151 μmol/L), and an increase in pericardial effusion.

Status at discharge and recommendations. The patient was discharged in a vitally stable condition, yet with a prognosis described as "grave and unfavorable." The sole recommendation recorded was a transfer to a specialized clinic-arranged in advance-for a surgical intervention on the tricuspid valve.

Following the exhaustion of all conservative treatment options, a surgical intervention was deemed necessary. The patient spent 16 days at the first clinic, during which the antimicrobial regimen was adjusted three times. Despite this, the infectious disease specialist summarized the patient's status with a single phrase: "suboptimal response to conservative treatment." This conclusion was based on four documented facts. First, the fever did not resolve: a low-grade fever persisted, and in recent days, intermittent episodes of high, spiking fever with chills occurred. Second, the size of the vegetations did not decrease significantly; the measurements of 17×8 mm and 17×7 mm recorded on May 31 were followed by 16×6 mm and a fixed 10 mm on June 14-indicating that two weeks of antibiotic therapy had failed to produce a structural response. Third, C-reactive protein levels never fell below 200 mg/L and reached as high as 250 mg/L. Fourth, new complications emerged: the pericardial effusion increased (with separation reaching 12 mm along the posterior wall of the left ventricle), anemia worsened, and renal function transiently deteriorated. In other words, by that point, the conservative strategy had become not only ineffective but also detrimental due to its own adverse effects.

The initial consultation with a cardiac surgeon resulted in a conditional conclusion: the presence of large vegetations, a history of pulmonary artery embolism, and a low ejection fraction rendered

surgical intervention a "very high-risk" procedure-though one to be considered only "in the event that antibiotic therapy proves ineffective." In a second cardiac surgical assessment issued that same day, the language became categorical: "Given the suboptimal response to conservative treatment, surgical intervention on the tricuspid valve is indicated, despite the high risk." Thus, the transfer to another medical facility was prompted not by new clinical findings but by a re-evaluation of existing data following the occurrence of a contingency anticipated 12 days earlier.

By prior arrangement, the patient was transferred to a facility where a tricuspid valve replacement was performed under cardiopulmonary bypass. The decision was based on the assessment of the cardiac surgeon who had initially evaluated the patient's data at the first clinic; in other words, the patient was transferred not to an unfamiliar team, but to the very same surgeon who was already familiar with the case. The second facility offered three essential resources-without which the patient's surgery would have been impossible: an extracorporeal circulation service, postoperative intensive care with vasopressor and respiratory support, and transfusion services (during the hospital stay, the patient received 10 units of packed red blood cells).

Treatment at the second clinic

The patient was admitted to the emergency department. Their condition was severe, characterized by profound weakness and physical inactivity (hypodynamia). An electrocardiogram showed a sinus rhythm with no signs of acute focal injury. Level I intensive care was initiated, and, in consultation with a cardiologist, the patient was transferred to the cardiac intensive care unit.

Upon admission, the patient's complaints were limited to shortness of breath, general weakness, and fatigue; orthopnea and sensations of suffocation were no longer present. This is consistent with the decongestion achieved at the first clinical facility. At the same time, the amount of fluid in the pericardial sac and the left pleural cavity was found to be significantly greater than documented two days earlier.

The primary diagnostic contributions from the second clinical facility consist of three investigations. The first was an urgent coronary angiography, which ruled out hemodynamically significant coronary artery disease, thereby excluding the possibility of ischemic cardiomyopathy. The second was a computed tomography (CT) scan of the brain, chest, and entire abdominal cavity, performed to search for evidence of systemic embolic events. Third, bacteriological analysis-including antibiotic susceptibility testing-was performed on blood, urine, and the tip of the central venous catheter (which had been inserted at the first clinical facility). Additionally, monitoring of vancomycin blood concentrations was initiated.

The primary diagnosis remained essentially unchanged: acute and subacute infective endocarditis, complicated by pulmonary artery embolism, secondary pulmonary hypertension, and pleural involvement. The treatment strategy, however, was altered: the conservative

approach was replaced by urgent surgery. This change was necessitated by an inadequate response to conservative treatment, in the context of large vegetations and the occurrence of pulmonary embolism.

Continuity of substitution therapy. Due to the inability to provide methadone, and in consultation with an addiction specialist, the patient was administered injections of morphine and diazepam. A few days later, the regular daily intake of methadone was resumed and continued without interruption both before and after the surgery.

The patient underwent the implantation of a biological tricuspid valve prosthesis. There were no intraoperative complications. The patient was admitted to the postoperative intensive care unit. Following separation from cardiopulmonary bypass, a noradrenaline infusion (0.06 mcg/kg/min) was initiated to stabilize hemodynamics; the patient was placed on mechanical ventilation using a synchronized intermittent mandatory ventilation (SIMV) mode with volume control.

Postoperative course: Tracheal extubation was performed without complications, followed by inhalation therapy, active non-invasive ventilation (CPAP mode), and monitoring of blood glucose levels and urine output. Pleuro-pericardial drains were removed without complications, and the patient was transferred to the cardiology department. Additionally, the temporary pacing lead was removed without complications; a follow-up echocardiogram showed positive progress. During the hospitalization, a total of 10 units of packed red blood cells (matched for blood type and Rhesus factor) were transfused.

Following the treatment, the patient's condition improved, allowing gradual increases in physical activity and expansion of the patient's regimen. The patient's subjective well-being and laboratory values improved, and both objective clinical findings and hemodynamic parameters stabilized. The patient was discharged in an improved condition.

Upon discharge, the following therapy was prescribed: completion of the antimicrobial treatment course at home. Cardiological medications were prescribed, and outpatient follow-up by a cardiologist, an infectious disease specialist, and a cardiac surgeon was recommended.

Results

Early postoperative period. Tracheal extubation was successfully performed 9 hours after the operation; on the second day, the pleuro-pericardial drains were removed and the patient was transferred to the cardiology department; on the sixth day, the temporary electrode was

extracted. All these stages proceeded without complications. Follow-up echocardiography showed positive dynamics (numerical data are not provided in the available documentation).

Changes in symptoms and objective findings. At the time of admission, the patient could not tolerate even a few steps and was forced to remain seated; at the time of discharge, the condition was assessed as improved, with stable hemodynamic parameters. Nine months later, the only complaint was fatigue during physical exertion exceeding a moderate level; the patient's lifestyle was described as active, and the heart failure was classified as Functional Class II. Edema, hepatomegaly, and signs of congestion were no longer observed.

Echocardiographic and laboratory findings during the long-term follow-up period. An electrocardiogram and Doppler echocardiography were performed, revealing two significant changes in the diagnosis: mitral insufficiency decreased from grade III to moderate, and heart failure improved from functional class IV to class II. The record also notes the new appearance of a block in the anterior fascicle of the left bundle branch, and the classification of arterial hypertension has been upgraded from grade II to grade III.

Complications and course of treatment. Two issues were documented. First, the patient had unilaterally discontinued their prescribed medications over the past month. Second, in February 2025, a single episode of hemoptysis (coughing up blood) occurred while the patient was taking warfarin (half of a 2.5 mg tablet). Both factors necessitated a review of the therapeutic regimen: warfarin was replaced with rivaroxaban (15 mg once daily), with instructions to monitor platelet levels.

Follow-up plan. Recommendations include a lipid-lowering diet, restriction of table salt and mineral-rich fluids, walking, home monitoring of pulse, blood pressure, and body temperature; a laboratory panel (complete blood count, urinalysis, glucose, coagulogram, creatinine, blood gases and electrolytes, lipid profile, transaminases, bilirubin, thyroid-stimulating hormone, vitamin D, and C-reactive protein); 24-hour electrocardiographic monitoring; and outpatient follow-up by both a cardiologist and an addiction specialist. The latter is particularly important, as the prevention of endocarditis recurrence in this population is directly linked to the sustained management of the addiction [2,4].

Final status: The patient is alive and under outpatient follow-up; heart failure is classified as Functional Class II; the biological prosthesis is functioning (with no evidence of dysfunction); and there have been no documented episodes of recurrent endocarditis or prosthetic valve infection.

Discussion

An additional clue came from the lower-extremity duplex examination itself. Although the study was requested to search for an embolic source, it also revealed pulsatile flow in the common femoral veins. Because the common femoral vein communicates with the right atrium through

the iliac veins and the inferior vena cava, which lack competent valves, a large systolic regurgitant volume across the tricuspid valve can be transmitted to the groin, converting the normally phasic venous signal into a pulsatile one. Pulsatile lower-limb venous flow has been linked to elevated right atrial pressure and tricuspid regurgitation, with high specificity but low sensitivity [14,15]. In our patient, this finding indicated hemodynamically significant tricuspid regurgitation, independent of echocardiography. It was concordant with grade IV regurgitation on echocardiography, the later-documented dilated, non-collapsing inferior vena cava (34 mm), and biochemical signs of hepatic venous congestion. The patient is included in an ongoing retrospective study at our center examining femoral venous pulsatility on routine lower-limb duplex as a marker of tricuspid regurgitation of grade II or higher. In practice, a pulsatile femoral venous signal on duplex ultrasound performed for suspected thrombosis should prompt targeted echocardiographic assessment of the tricuspid valve and right heart. Diagnostic challenges: The clinical presentation of endocarditis was entirely overshadowed by signs of heart failure; furthermore, elevated D-dimer levels and filling defects observed on computed tomography (CT) scans cannot, in themselves, distinguish between thrombotic and septic emboli. On the day of admission, the complete blood count showed no leukocytosis (leukocytes: 7.64×10^9/L), which made a systemic infection less likely; only the additional measurement of C-reactive protein (205 mg/L) revealed the true extent of the inflammatory process. Furthermore, the severe impairment of systolic heart function was initially misattributed to an ischemic origin-a possibility that was ruled out only after a coronary angiogram was performed at a second medical facility.

The patient spent a total of 31 days across two medical facilities for the same condition; however, this did not constitute an interruption and subsequent restart of treatment. Rather, the two hospitalizations represented two stages of a single, continuous clinical strategy, in which the first stage established the conditions that made the second possible.

Another equally critical factor influenced the timing of the surgical intervention: the patient had to be both transportable and fit for surgery. Upon admission, the patient met neither criterion: presenting with orthopnea, a respiratory rate of 26–28 breaths per minute, an oxygen saturation of 85%, bilateral hydrothorax (totaling up to one liter), and a systolic pulmonary artery pressure of 90 mmHg. Given this condition, it is highly likely that the patient would not have survived a surgery requiring extracorporeal circulation. On the day of discharge, the clinical picture is different: blood pressure is 120/70 mmHg, pulse is 80, respiratory rate is 18 breaths per minute, oxygen saturation is 98% on room air, there is no edema in the lower extremities, systolic pulmonary artery pressure is 40–50 mmHg, hemoglobin is 77 g/L following a transfusion, and creatinine levels have returned to the normal range. Thus, the primary outcome of the 16-day treatment at the First Clinic was not the resolution of the infection-which was not achieved-but rather the creation of a "window of operability." Current clinical guidelines dictate precisely this sequence: hemodynamic stabilization and antimicrobial therapy are not alternatives to surgical

intervention; rather, they constitute a preparatory phase for patients in whom infection control has not yet been achieved.

Surgical treatment had been proposed to the patient even earlier-on May 28, 2024, at a regional hospital-from which the patient was discharged after declining the surgery. Twenty-two days elapsed between this initial presentation and the operation; during this period, a pulmonary embolism developed, the anemia worsened, and a pericardial effusion emerged.

Conclusion

The primary feature of this case is that infective endocarditis of the tricuspid valve manifested sequentially as pneumonia, dilated cardiomyopathy, and pulmonary embolism-with each diagnosis fully substantiated by the available data when considered individually.

The main diagnostic challenge was distinguishing septic from thrombotic pulmonary embolism, given markedly elevated D-dimer levels and computed tomography findings showing vascular filling defects. Three additional factors proved decisive: large, mobile vegetations on the tricuspid valve; cavitating pulmonary nodules; and the absence of deep vein thrombosis. The same duplex examination also revealed a less obvious sign: pulsatile common femoral venous flow, which suggested significant tricuspid regurgitation. The primary therapeutic challenge was deciding when to proceed with surgical intervention, given a critically reduced ejection fraction, severe anemia, hypoalbuminemia, and severe pulmonary hypertension. The key clinical finding is that a severely reduced ejection fraction in the context of infective endocarditis may be significantly reversible; therefore, it should not, in itself, serve as the sole justification for declining surgical intervention. In this patient, surgical control of the infectious focus achieved an outcome that two weeks of empirical antimicrobial therapy alone could not attain.

In similar clinical scenarios, physicians should consider four practical points: blood cultures must be obtained before the first dose of antibiotics; computed tomography (CT) findings should be evaluated with attention to both vascular and parenchymal structures; the response to conservative treatment must be assessed against predefined, time-limited criteria; and the continuity of replacement therapy should be planned at the outset of hospitalization as an integral component of the overall treatment strategy.

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ინფექციური ენდოკარდიტი ფილტვის არტერიის თრომბოემბოლიის ნიღბით - შემთხვევის აღწერა

ნინო ლეკვეიშვილი[1,2], მარინა ყუფარაძე[1,2], გიორგი იმედაშვილი[1], ირინა ლეჟავა[2], ანზორ მელია[2], თეიმურაზ მოსიავა[3]

1. თბილისის სახელმწიფო სამედიცინო უნივერსიტეტი, თბილისი, საქართველო;
2. კარდიოლოგიური კლინიკა “გული”, თბილისი, საქართველო;
3. საქართველოს დავით აღმაშენებლის სახელობის უნივერსიტეტი, თბილისი, საქართველო.

აბსტრაქტი

შესავალი. ინექციური ნარკოტიკების მომხმარებლებში ინფექციური ენდოკარდიტი უპირატესად მარჯვენა გულს აზიანებს და ხშირად სეპტიური ფილტვისმიერი ემბოლიით ვლინდება. ასეთი სურათი ადვილად ინიღბება ფილტვის არტერიის თრომბოემბოლიად, პნევმონიად ან პირველად კარდიომიოპათიად, რაც სპეციფიკურ მკურნალობას აყოვნებს.

შემთხვევის აღწერა. 50 წლის მამაკაცი, რომელიც ოთხი წელია მეტადონით ჩანაცვლებით პროგრამაში იმყოფებოდა, ჰოსპიტალიზებულ იქნა მოსვენებულ მდგომარეობაში ქოშინის, ორთოპნოეს, ცხელებისა და ქვემო კიდურების შეშუპების გამო; ჟანგბადით გაჯერება ატმოსფერულ ჰაერზე 85%-ს შეადგენდა. ექოკარდიოგრაფიულად გამოვლინდა გულის ყველა ღრუს დილატაცია, განდევნის ფრაქცია 18–20%, ფილტვის არტერიის სისტოლური წნევა 90 მმ ვწყ. სვ. და ტრიკუსპიდური სარქვლის კარედებზე ორი მოძრავი წარმონაქმნი (17×8 მმ და 17×7 მმ). D-დიმერი 4000 ნგ/მლ-ს აღემატებოდა, ხოლო კომპიუტერულ-ტომოგრაფიულმა ანგიოგრაფიამ აჩვენა ორმხრივი სეგმენტური

ავსების დეფექტები რღვევადი ნოდულური კერების ფონზე; ქვემო კიდურების ვენები სრულად კომპრესირებადი იყო. ანტიბიოტიკოთერაპია მთელი პერიოდის განმავლობაში ემპირიული იყო, რეჟიმი კი სამჯერ შეიცვალა. 16-დღიანი კონსერვატიული მკურნალობის მიუხედავად, შენარჩუნდა ცხელება, ვეგეტაციები და ანთებითი მარკერების მაღალი დონე, რის გამოც პაციენტი პროფილურ კლინიკაში გადაიყვანეს. იქ ოპერაციამდე ჩატარებულმა ექოკარდიოგრაფიამ აჩვენა, რომ განდევნის ფრაქცია უკვე 52%-მდე იყო გაუმჯობესებული; კორონაროგრაფიამ გამორიცხა კორონარული არტერიების ჰემოდინამიკურად მნიშვნელოვანი დაზიანება და ჩატარდა ტრიკუსპიდური სარქვლის პროთეზირება ბიოლოგიური სარქვლით. პაციენტი გაუმჯობესებული მდგომარეობით გაეწერა, ხოლო ცხრა თვის შემდეგ გულის უკმარისობა II ფუნქციურ კლასს შეესაბამებოდა.

დასკვნა. მარჯვენა გულის ინფექციური ენდოკარდიტი უნდა განიხილებოდეს ყოველი იმ პაციენტის დიფერენციულ დიაგნოზში, რომელსაც ინექციური ნარკოტიკების მოხმარების ანამნეზი, ცხელება და ფილტვის მრავლობითი კეროვანი დაზიანება აქვს - მაშინაც კი, როდესაც კომპიუტერული ტომოგრაფია ფილტვის არტერიის ავსების დეფექტებს აჩვენებს. ღრმა სისტოლური დისფუნქცია ასეთ პაციენტებში შეიძლება ნაწილობრივ შექცევადი აღმოჩნდეს და თავისთავად არ უნდა იქცეს ქირურგიულ მკურნალობაზე უარის თქმის მიზეზად.

საკვანძო სიტყვები: ინფექციური ენდოკარდიტი; ტრიკუსპიდური სარქველი; სეპტიური ფილტვისმიერი ემბოლია; სარქვლის ბიოლოგიური პროთეზი; გულის შეგუბებითი უკმარისობა.