SUCCESSFUL TREATMENT OF AN APPARENTLY TERMINAL STAGE OF HEART FAILURE WITH ANASARCA RESULTING IN ACHIEVED CARDIAC COMPENSATION – A CASE REPORT

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Abstract: Introduction: Chronic heart failure (CHF) represents the end stage of various cardiovascular diseases and is one of the leading causes of hospitalization and mortality in the elderly population. Decompensated chronic heart failure (DCHF), with or without acute exacerbation, often leads to circulatory congestion and fluid accumulation, with anasarca (generalized edema) being one of the most severe manifestations, along with cardiac cachexia. In addition to the four "pillars" of CHF treatment across different severity stages according to the New York Heart Association (NYHA) classification, diuretic therapy remains the cornerstone of treatment in the terminal stage with severe systemic congestion, such as anasarca. Diagnostic and therapeutic challenges are frequently present due to comorbidities such as chronic kidney disease and anemia. Case report: An 88-year-old male patient with a history of chronic heart failure with reduced left ventricular ejection fraction (HFrEF), permanent atrial fibrillation, and significant comorbidities: stage 3b chronic kidney disease and severe iron-deficiency anemia. On admission, the patient presented with dyspnea at rest, tachypnea, bilateral hydrothorax, severe edema of the lower legs and forearms (anasarca), and marked general weakness. Laboratory findings showed elevated NT-proBNP levels, markedly reduced hemoglobin (68 g/L), aggravated by hemodilution, and impaired renal function (glomerular filtration rate, GFR = 44 mL/min/1.73 m^2). Echocardiography revealed left ventricular dilation (end-diastolic diameter, EDD = 59 mm) and left atrial enlargement (left atrial volume index, LAVI = 45 mL/m^2), an inferior wall scar with an aneurysm of the basal inferior segment, and anteroseptal-apical dyskinesia. The left ventricular ejection fraction was significantly reduced (EF = 37%). The right ventricular systolic pressure (RVSP) was 64 mmHg, global longitudinal strain (GLS) was 11.6%, and there were signs of grade II diastolic dysfunction (E/e' = 15.0).
The therapeutic approach included urgent parenteral administration of high-dose furosemide over three days in a day-hospital setting (the patient refused hospitalization and blood transfusion). Optimization of chronic therapy was performed, including the introduction of sacubitril/valsartan (ARNI) and sodium-glucose co-transporter 2 (SGLT2) inhibitors, along with restriction of salt and fluid intake and more intensive correction of iron-deficiency anemia. During outpatient follow-up, significant improvement was achieved: marked diuresis, body weight reduction of 14 kg, excellent regression of edema, normalization of lung findings with a significant reduction in hydrothorax, improvement in EF to 47%, improvement in diastolic function (E/e' = 13.6), and reduction of RVSP to 25 mmHg. Renal function normalized (GFR = 64 mL/min/1.73 m^2), and hemoglobin increased to 128 g/L. Conclusion: This case report highlights the importance of individualized intravenous diuretic therapy in combination with contemporary pharmacological strategies in patients with the most severe form of decompensated chronic heart failure (NYHA class IV) with anasarca. Timely initiation of parenteral diuretics, optimization of baseline therapy, and correction of associated disorders led to significant clinical, echocardiographic, and laboratory improvement. This report emphasizes the importance of continuous monitoring of critically ill patients using ECG monitoring and other vital parameters, as well as therapy adjustment according to diuresis, body weight, blood pressure, heart rate, and echocardiographic parameters, to achieve optimal outcomes alongside the management of comorbidities in consultation with other specialties.
Keywords: terminal chronic heart failure with reduced ejection fraction (HFrEF), decompensated congestive heart failure, anasarca (generalized edema), chronic kidney disease, chronic iron-deficiency anemia, pharmacotherapy, furosemide, sacubitril/valsartan, dapagliflozin, bisoprolol, rivaroxaban, spironolactone, digoxin..

INTRODUCTION

Chronic heart failure is a progressive and complex syndrome representing the end stage of various cardiovascular diseases and is one of the leading causes of morbidity and mortality worldwide. Acute heart failure is the most severe clinical form, characterized by cardiogenic pulmonary edema and cardiogenic shock, with the highest mortality, requiring urgent hospital treatment, although it is fortunately much less common than chronic heart failure. The prevalence in the general population is estimated at 1–2%, while in individuals older than 75 years it reaches up to 10% [1,2]. Globally, more than 64 million people live with this condition, and a further increase is expected due to population aging [3].

Decompensated chronic heart failure is characterized by congestion in the pulmonary and/or systemic circulation and fluid accumulation in the body. The most severe form of this process is anasarca, a diffuse generalized edema that may include ascites, pleural effusions, and pericardial effusion [4]. In addition to heart failure, anasarca also occurs in other conditions (nephrotic syndrome, liver cirrhosis, severe hypoalbuminemia), but in the context of heart failure it indicates a terminal stage, exhaustion of compensatory mechanisms, and poor prognosis [5].

The diagnosis and management of patients with anasarca are challenging, as a combination of cardiac, renal, and hepatic dysfunction is often present. Treatment is based on aggressive and individualized intravenous diuretic therapy, correction of electrolyte imbalances, optimization of hemodynamics, and a multidisciplinary approach involving consultants [6].

This case report is particularly significant as it demonstrates that a severe form of heart failure, seemingly refractory terminal-stage decompensated chronic heart failure, can be successfully treated with intensive therapy dominated by high-dose intravenous furosemide, in addition to the standard pillars of heart failure management in patients with anasarca.

The contemporary approach to chronic heart failure treatment is based on the so-called “four pillars of therapy” (ARNI/ACE inhibitors, beta-blockers, mineralocorticoid receptor antagonists, and SGLT2 inhibitors), which significantly reduce mortality and hospitalizations [6–9]. Equally important in the setting of anasarca is the “fifth pillar” — diuretic therapy with intravenous loop diuretics.

CASE REPORT

Basic patient data: Male patient, K.A., 88 years old; anthropometric parameters: body weight 78 kg, height 167 cm, body mass index (BMI) 29.4 kg/m^2, body surface area (BSA) 1.9 m^2, waist circumference 92 cm, and oxygen saturation (SpO_2) 96%.

The patient was admitted on June 6, 2025, in a state of severe decompensated chronic heart failure with marked congestion and anasarca, representing a clinical indicator of advanced disease [5].

Medical history: The patient presented with progressive dyspnea, swelling of the lower legs and forearms, marked weakness, fatigue, and shortness of breath on minimal exertion and at rest following exertion. Symptoms had been present for the past 14 days, with rapid progression. Increased fatigue had been noted over the previous two weeks (he was unable to climb to the first floor), accompanied by rapid and irregular heart rhythm and significant swelling of the lower legs, followed by the forearms.

He was examined by an internist in the hospital one week earlier, when low-dose therapy was initiated: furosemide 40 mg once daily orally, spironolactone 25 mg once daily, and rivaroxaban 15 mg once daily. The patient denied chest pain. Blood pressure at home was generally low. He reported a history of treated hypertension over the past four years, without prior use of cardiac medications.

Previous long-term therapy: bisoprolol 5 mg (1 + 0 + ½), rivaroxaban 15 mg once daily, ramipril/hydrochlorothiazide 5/25 mg once daily in the morning, ramipril 5 mg once daily in the evening, allopurinol 100 mg once daily, furosemide 40 mg once daily, spironolactone 25 mg once daily, and iron supplementation 30 mg once daily.

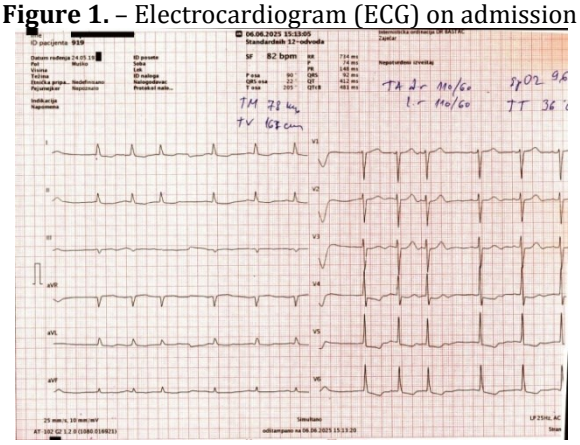
Physical examination on admission:

General condition: Pale and dyspneic, acyanotic, afebrile. Vital parameters: blood pressure 110/60 mmHg, heart rate approximately 82/min, irregular rhythm. Oxygen saturation (SpO_2) 95%. Skin and mucous membranes pale.

Lungs (auscultation): Breath sounds diminished, bilaterally absent at the bases; percussion note dull at the lung bases on both sides.

Heart: Displaced apical impulse on palpation. Heart rate 82/min, heart sounds attenuated, rhythm irregular consistent with atrial fibrillation. A holosystolic regurgitant murmur grade 2–3/6 was heard over the apex, without radiation.
Abdomen: The liver was palpable 4 cm below the right costal margin in the midclavicular line, with soft consistency; the spleen was not palpable. No signs of ascites.
Extremities: Massive, pitting, cold edema of the lower legs (right side: subpatellar circumference 41 cm, mid-tibial 36 cm, supramalleolar 25 cm; left side respectively 35 cm, 34 cm, 27 cm); mild edema of the hands and forearms.
Laboratory findings:
NT-proBNP: 1314 pg/mL (<526 for patient's age)
D-dimer: 0.46 μg/mL (<0.4)
Urea: 17 mmol/L
Creatinine: 133 μmol/L
GFR: 44 mL/min/1.73 m^2
Liver enzymes: AST 96 U/L, ALT 165 U/L
Hemoglobin: 68 g/L, RBC 3.65 × 10^{12}/L, MCV 67.7 fL
Laboratory findings indicated chronic heart failure with cardiorenal syndrome and severe anemia, which are common comorbidities and further worsen prognosis [10–12].
Electrocardiogram (ECG):
Atrial fibrillation with absolute ventricular arrhythmia, heart rate 82/min, intermediate electrical axis, normal QRS duration, occasional ventricular extrasystoles (PVCs), ST depression up to 2 mm with negative T waves in leads V4–V6, and ST depression up to 0.5 mm with negative T waves in leads I, II, and aVL (Figure 1).).

Figure 1. – Electrocardiogram (ECG) on admission



ECHOCARDIOGRAPHY:

The findings are dominated by left ventricular dilation, with normal left ventricular wall thickness, no myocardial hypertrophy, and reduced global systolic function: the left ventricular ejection fraction (EF) was 39% by M-mode (Teichholz method) (Figure 2), and 35% and 39% by Simpson's method, with a biplane EF of 37% (Figures 3, 4)..

Figure 2. – M-mode echocardiogram: ejection fraction (EF) = 39% according to Teichholz

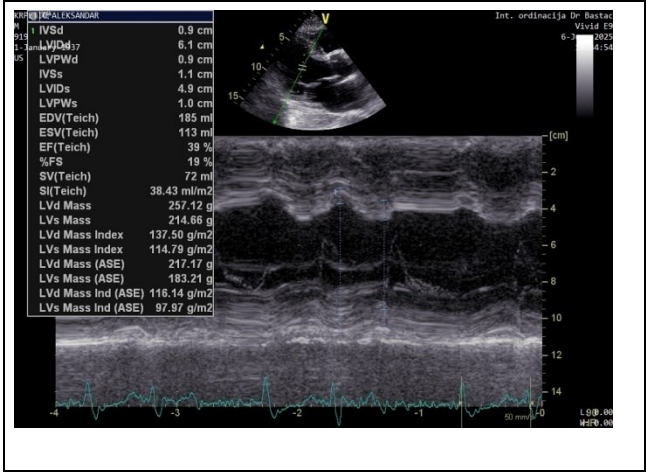


Figure 3. – B-mode echocardiogram: automatic endocardial border detection – ejection fraction (EF) = 35% by Simpson's method (2-chamber view)

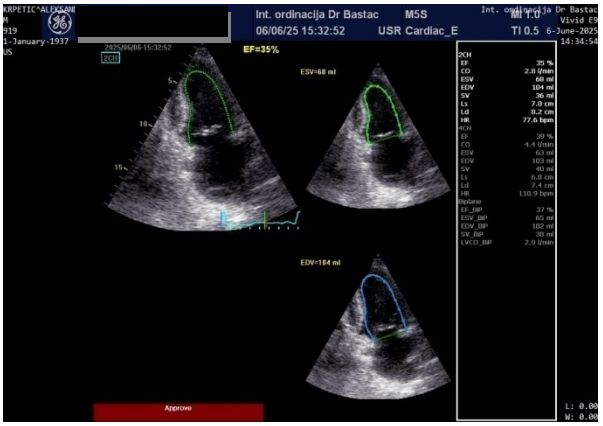
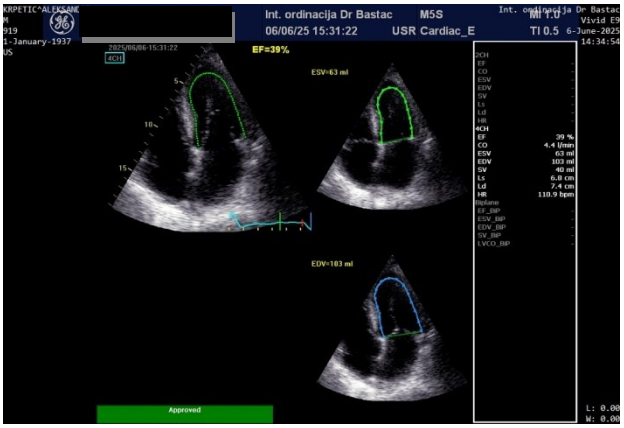


Figure 4. – B-mode echocardiogram: automatic endocardial border detection – ejection fraction (EF) = 39% by Simpson's method (4-chamber view)



A small aneurysm of the basal segment was observed on the inferior wall, with a suspected organized thrombus. Anteroapical and anteroseptoapical dyskinesia were present. The most representative parameter of diastolic function, the E/e′ ratio, was markedly elevated at 15.0 (normal <8.0; E/e′ represents the ratio of early transmitral inflow velocity (E) to the average mitral annular velocity on tissue Doppler imaging (e′)).

The left atrium was dilated, with a left atrial volume index (LAVI) of 45 mL/m^2 (normal <34 mL/m^2) (Figure 5). The maximum velocity (Vmax) of tricuspid regurgitation was measured at 3.3 m/s (tricuspid gradient 44 mmHg), and the right ventricular systolic pressure was 64 mmHg.

Lung ultrasound: Hydrothorax was present, with an anteroposterior (AP), basal pleural, hypoechoic "dense" effusion: on the left side measuring 9.9 × 9.4 cm (Figures 6, 7), with an AP diameter of 6.5 × 4.6 cm at the level of the scapular angle; on the right side, 3 cm below the scapular angle (AP), measuring 7.0 × 7.0 cm, and laterally 7.0 × 10.0 cm.

Figure 5. – Left atrial volume index (LAVI)

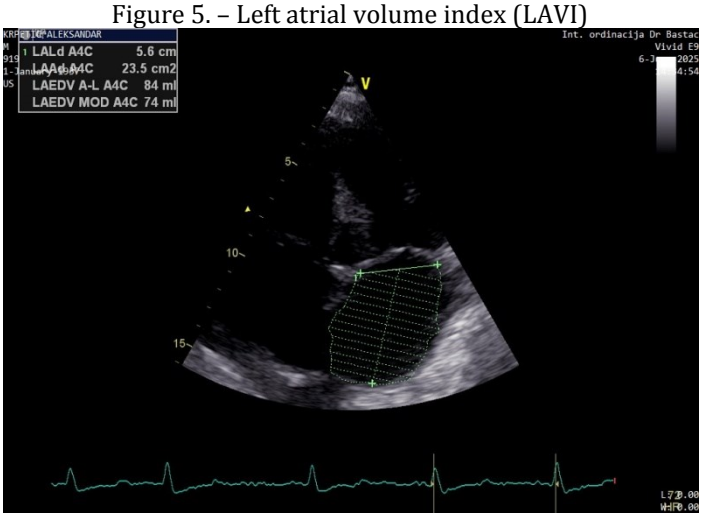


Figure 6. – B-mode echocardiogram: hypoechoic "dense" pleural effusion, AP diameter 9.4 cm with fibrin strands

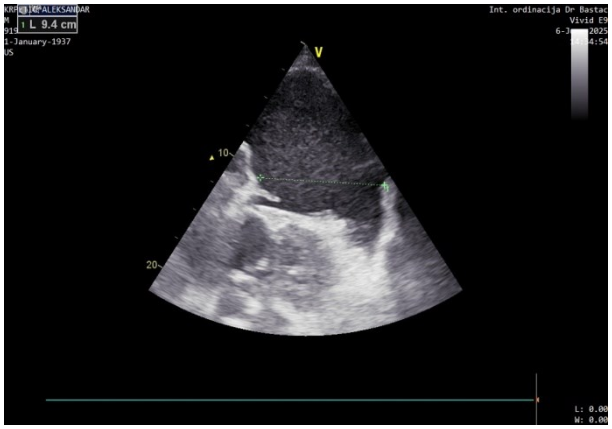
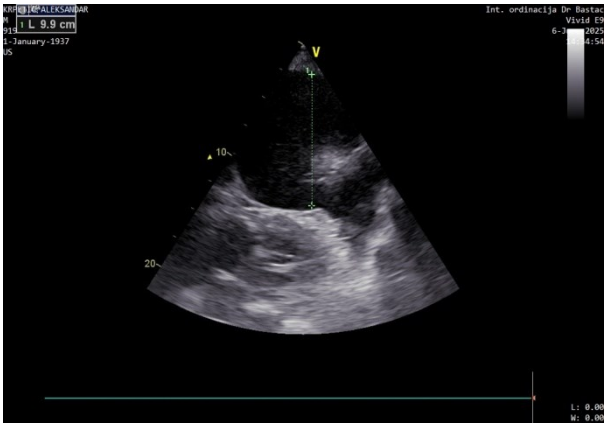


Figure 7. – B-mode echocardiogram: pleural effusion, cranio-caudal (CC) diameter 9.9 cm



Working diagnoses:

Congestive heart failure (ICD-10: I50), with echocardiographically reduced left ventricular ejection fraction (HFrEF ≈ 35%), accompanied by the following cardiac conditions: permanent atrial fibrillation (fibrillatio atriorum permanens), post-infarction myocardial scar of the inferior wall (cicatrix myocardii post infarctum parietis inferioris), functional left ventricular aneurysm of the inferior wall (aneurysma functionalis ventriculi sinistri cordis parietis inferioris), bilateral hydrothorax, mitral valve insufficiency and aortic semilunar valve insufficiency, and pulmonary arterial hypertension.

Comorbidities: Chronic kidney disease stage 3b (morbus renalis gradus 3b) and severe chronic microcytic iron-deficiency anemia (anemia microcytica sideropenica chronica, gradus gravis)..

COURSE OF DISEASE (DECURSUS MORBI)

The patient refused the proposed hospitalization at the Department of Internal Medicine, ZC Zaječar, despite being informed about the life-threatening condition requiring intensive care management. He was followed in a day-hospital setting at our outpatient facility with continuous ECG monitoring, blood pressure and urine output measurements, oxygen saturation monitoring, and other vital parameters. Due to severe anemia, blood transfusion of packed red blood cells was indicated; however, the patient did not present to the Blood Transfusion Service. The patient was immediately started on intensified parenteral diuretic therapy: on the day of examination, furosemide ampoules 20 mg, total No VIII (160 mg), administered in two intravenous boluses, in accordance with recommendations for the treatment of acute decompensation [6,13,14]. Early and aggressive diuretic therapy resulted in significant reduction of volume overload, which represents a key therapeutic goal [13–15]. A prompt and excellent diuresis was achieved.

On the following day, intravenous furosemide was continued at 20 mg ampoules No IV (80 mg). Previous outpatient therapy was adjusted: bisoprolol 5 mg tablets ½ tablet twice daily; rivaroxaban 15 mg once daily; spironolactone 25 mg once daily; and iron supplementation. Ramipril/hydrochlorothiazide 5/25 mg once daily in the morning, ramipril 5 mg once daily in the evening, and allopurinol 100 mg once daily were discontinued.

Guideline-directed medical therapy was introduced with ARNI: sacubitril/valsartan 26/24 mg, ½ tablet twice daily, and dapagliflozin 10 mg once daily, in accordance with current recommendations [7,8,16–18]. For improved correction of anemia, the iron therapy was intensified to a maximum dose of iron preparation (300 mg/day) instead of the previous supplementation regimen.

Correction of anemia was initiated due to its negative impact on functional status and clinical outcome, as it further aggravates tissue hypoxia [12,19]. Non-pharmacological measures included strict fluid and salt restriction and prohibition of physical activity. Under this therapeutic approach, a progressive increase in diuresis and significant reduction of edema were observed..

At the first control visit on the third day of treatment (June 7, 2025), an excellent response in fluid removal was observed: the patient had a weight reduction of 7 kg, with complete resolution of dyspnea on minimal exertion and a significant reduction of lower limb edema (circumference measurements: right leg 40 cm, 35 cm, 24 cm; left leg 36 cm, 34 cm, 24 cm). Hemoglobin increased to 72 g/L. At this point, from day 4, oral therapy was introduced with furosemide forte ½ tablet of 500 mg, and digoxin 0.25 mg ½ tablet every second day due to atrial fibrillation and hypotension. The patient was referred for multidetector computed tomography (MDCT) of the chest, which was not performed later in the course.

At the second control visit on the fifth day of treatment (June 11, 2025), the patient had a total weight loss of 12 kg and minimal residual edema (leg circumference: right 33 cm, 33 cm, 24 cm; left 34 cm, 33 cm, 24 cm). Lung examination showed normal breath sounds with mildly reduced basal ventilation, no prolonged expiration, and dullness to percussion at the bases below the 10th rib.

Echocardiographic evaluation demonstrated improvement in left ventricular ejection fraction and improved diastolic function (left ventricular compliance), with an E/e′ ratio of 7.5. Laboratory results showed serum iron <1 µmol/L (normal 11–31) and ferritin 19.2 ng/mL (normal 20–250 ng/mL).

At the follow-up after two weeks (June 19, 2025), the patient maintained excellent clinical improvement, with an additional 2 kg weight loss (total 14 kg reduction from baseline), representing a very good therapeutic response. He reported dizziness and instability, attributed to hypotension (BP 90/55 mmHg and 80/50 mmHg), leading to dose reduction of hypotensive medications: furosemide 500 mg ½ tablet every second day and sacubitril/valsartan 26/24 mg ¼ tablet twice daily. Due to lower leg pain, diosmin + hesperidin 1000 mg once daily was added for venous symptoms.

New laboratory findings included: hemoglobin 88 g/L, erythrocyte sedimentation rate (ESR) 55 mm/h, urea 15.4 mmol/L, creatinine 131 µmol/L, GFR 44.8 mL/min/1.73 m^2, and potassium 4.4 mmol/L.

ECG: Atrial fibrillation with absolute arrhythmia, heart rate 65/min, ST depression up to 2 mm with negative T waves in V4–V6, and ST depression up to 0.5 mm with negative T waves in leads I, II, and aVL (Figure 8).

At routine follow-up after two months (August 25, 2025), the patient was asymptomatic, with a further 2 kg weight reduction, no leg edema, and no longer hypotensive. A marked increase in hemoglobin to 128 g/L was observed, attributed to iron therapy and correction of hemodilution. Renal function normalized (GFR = 64 mL/min/1.73 m^2).

Echocardiography (Figures 9, 10, and 11) showed a significant improvement in left ventricular systolic function, with M-mode EF of 47% and biplane Simpson EF of 46%, along with a reduction in left ventricular dilation (LVEDD = 50 mm, LVESD = 40 mm) and resolution of pulmonary hypertension (RVSP = 25 mmHg).

Further optimization of maintenance therapy was performed, including reduction of furosemide dose to 500 mg ¼ tablet every second or third day..

Figure 8. – ECG at 2-week follow-up

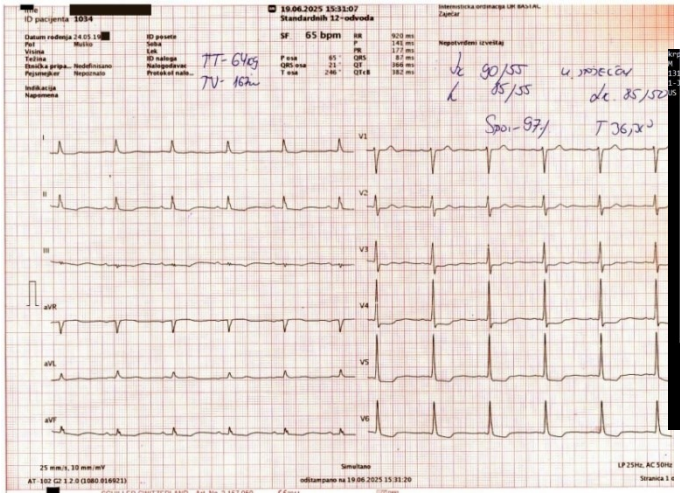


Figure 9. – M-mode echocardiogram: follow-up ejection fraction (EF) = 47% according to Teichholz method

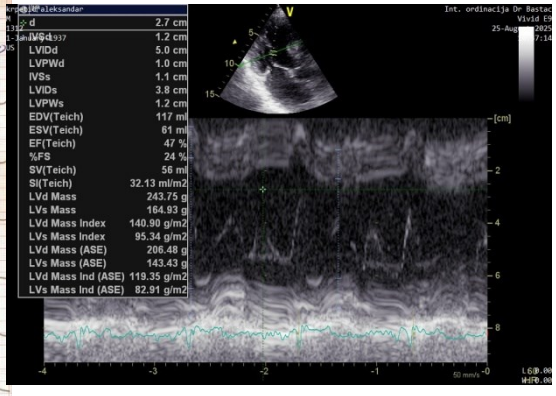


Figure 10. – B-mode echocardiogram: left ventricular ejection fraction (EF) = 52% (monoplane, Simpson's method, 4-chamber view)

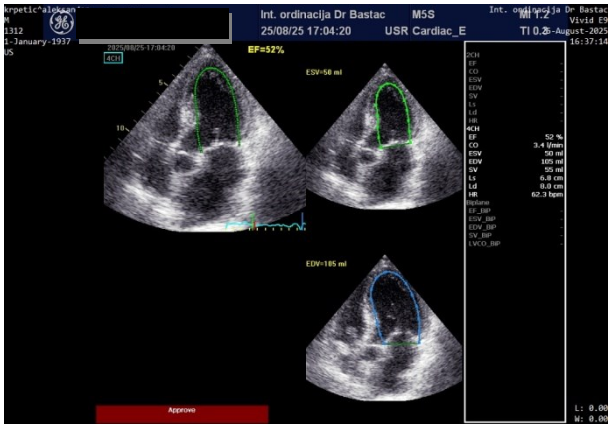
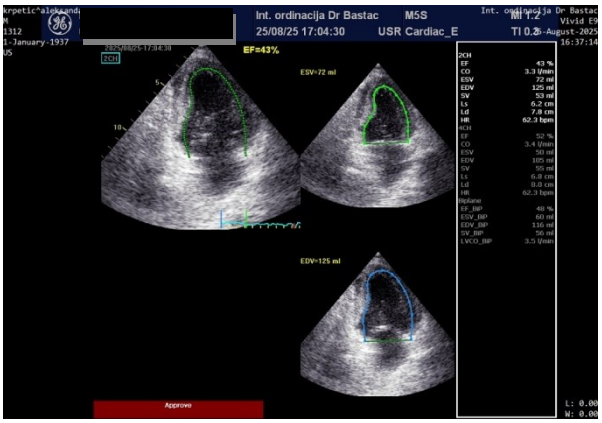


Figure 11. – B-mode echocardiogram: left ventricular ejection fraction (EF) = 43% (monoplane, Simpson's method, 4-chamber view)



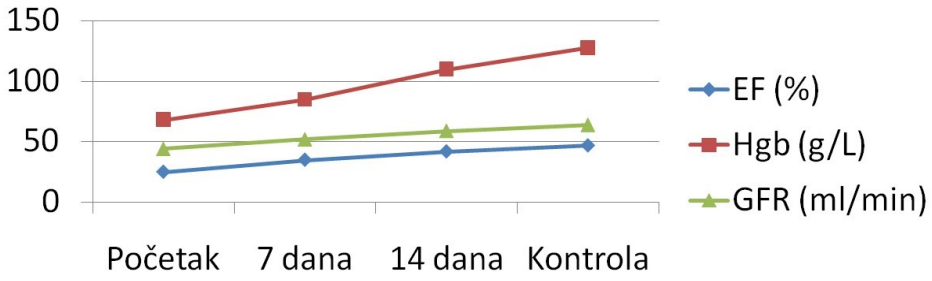
CLINICAL OUTCOME:

Following intensive intravenous diuretic therapy and three days of day-hospital management with ECG monitoring and continuous assessment of vital parameters, a significant clinical improvement was achieved. The patient was subsequently transitioned to oral therapy.

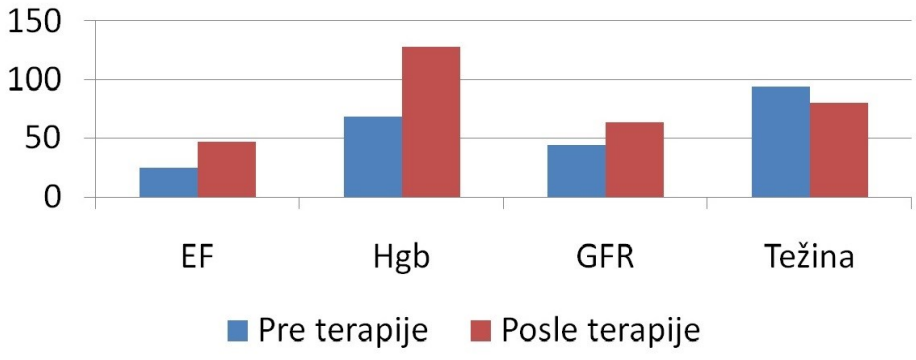
A total weight loss of 14 kg was recorded over a two-week period, accompanied by complete resolution of peripheral edema and normalization of lung auscultation findings. Pleural effusions regressed to minimal levels. Left ventricular systolic function remained preserved at approximately 46–47% ejection fraction, with improvement in diastolic function on echocardiography.

Laboratory parameters demonstrated progressive recovery of renal function (GFR improved from 44 to 64 mL/min/1.73 m^2) and a significant increase in hemoglobin levels following iron supplementation (Graph 1 and 2)

Graph 1. – Trend of changes in clinical parameters during therapy



Graph 2. – Comparative presentation of pre- and post-therapy effects



DISCUSSION

Anasarca represents an extreme form of fluid retention and a marker of advanced heart failure with a poor prognosis [5].

Diuretics remain the cornerstone of congestion therapy, with intravenous administration enabling faster and more effective decongestion [6,13,14]. However, their use requires careful monitoring due to the potential deterioration of renal function [10,20], hypokalemia, and, less frequently, hypovolemia and dehydration.

In this case, the improvement in renal function following therapy suggests reversibility of cardiorenal syndrome after congestion relief, as previously described in the literature [10,11]. Contemporary studies indicate that early initiation of SGLT2 inhibitors provides rapid clinical benefit and reduces hospitalization rates [17,21–23]. ARNI therapy further contributes to improved myocardial remodeling and reduced mortality [16]. Current guidelines emphasize the simultaneous or early sequential implementation of the five foundational therapeutic pillars, which is associated with the best clinical outcomes [7–9,24].

Congestion management remains the key therapeutic target, and individualized intravenous diuretic therapy with careful monitoring of body weight, urine output, and renal function is essential for successful treatment [14,15]. Anemia is a common comorbidity in heart failure, and its correction—particularly with intravenous iron preparations—improves symptoms and quality of life [12,19].

This case highlights the importance of timely initiation of intensive diuretic therapy, continuous monitoring of diuresis and laboratory parameters, and an individualized approach depending on comorbidities. It also demonstrates that an outpatient approach, under adequate supervision, may be feasible in selected patients with severe decompensated chronic heart failure that appears terminal and refractory to treatment, although such patients are most commonly managed in hospital settings [25].

CONCLUSION

Decompensated heart failure with anasarca represents a severe and life-threatening condition requiring an aggressive yet carefully titrated individualized therapeutic approach. Diuretic therapy remains the cornerstone in controlling volume overload. This case report highlights the importance of individualized diuretic therapy combined with contemporary pharmacological strategies in patients with the most severe forms of decompensated heart failure and anasarca. Timely initiation of high-dose parenteral diuretics, optimization of baseline therapy, and correction of associated disorders led to significant clinical and laboratory improvement in this patient.

This case also emphasizes the importance of intravenous therapy administration under continuous ECG monitoring, along with close observation and adjustment of treatment according to urine output, blood pressure, heart rate, serum potassium and nitrogenous waste levels, blood pressure, and oxygen saturation, in order to achieve optimal outcomes. Particular importance is given to individualized therapy and early recognition of refractoriness to standard oral treatment strategies in chronic heart failure management.

The combination of intensive diuretic therapy and modern pharmacological strategies can lead to substantial clinical improvement even in patients with advanced disease, as demonstrated in this case, which was successfully stabilized in an outpatient day-hospital setting.

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