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USPEŠNO LEČENJE NAIZGLED TERMINALNOG STADIJUMA HRONIČNE SRČANE INSUFICIJENCIJE SA SNIŽENOM EJEKCIONOM FRAKCIJOM I ANASARKOM UZ POSTIZANJE SRČANE KOMPENZACIJE - PRIKAZ SLUČAJA

Pavle Nešović (1), Silvana Babić (2), Mila Bastać (3), Zoran Joksimović (1), Dušan Bastać (1)

(1) INTERNISTIČKA ORDINACIJA "DR BASTAĆ" ZAJEČAR; (2) KLINIKA ZA ORL, UKC BEOGRAD; (3) MEDSCAN TADIĆ DIJAGNOSTIKA, ZAJEČAR

Sažetak: Uvod: Hronična Srčana insuficijencija (HSI) ili slabost predstavlja završnu fazu različitih kardiovaskularnih oboljenja i jedan je od vodećih uzroka hospitalizacija i mortaliteta kod starije populacije. Dekompenzovana hronična srčana insuficijencija (DHSI) sa ili bez akutizacije, često dovodi do zastoja u cirkulaciji i nakupljanja tečnosti, pri čemu anasarka (generalizovan edem) predstavlja jednu od najtežih manifestacija, uz kardijalnu kaheksiju. Uz četvoro "stuba" lečenja hronične srčane insufcijencije u raznim stepenima težine po klasifikaciji Njujorškog kardiološkog društva (NYHA), diuretska terapija je osnovni modalitet lečenja u terminalnom stadijum sa velikom kongestijom celog organizma, kao što je anasarka. Često postoje dijagnostički i terapijski izazovi usled komorbiditeta kao što su hronična bubrežna insuficijencija i anemija. **Prikaz slučaja**: Pacijent starosti 88 godina, muškarac, sa istorijom hronične srčane insuficijencije sa sniženom ejekcionom frakcijom leve komore (HFrEF), permanentnom atrijalnom fibrilacijom i značajnim komorbiditetima: hronična bubrežna bolest u stadijumu 3b i teška sideropenijska anemija. Pacijent na prijemu sa dispnejom u miru, tahipnejom, sa obostranim hidrotoraksom, ekstremnih otoka potkolenica i podlaktica-anasarka, izražene opšte slabosti. Laboratorijski nalazi pokazali su povišene vrednosti NT-proBNP, jako snižen hemoglobin 68g/L, potenciran hemodilucijom i smanjenu bubrežnu funkciju (brzina glomerularne filtracije-GFR=44ml/min/1,73m^2). Ehokardiografijom je utvrđena dilatacija leve komore (teledijastolna dimenzija leve komore-EDD=59mm) i leve pretkomore (volumen indeks leve prekomore, LAVI=45ml/m^2), cicatriks inferiornog zida sa aneurizmom bazalnog inferiornog segmenta i antero-septo-apikalno diskinezija. Značajno je snižena ejekciona frakcija leve komore EF=37%. Izmeren je sistolni pritisak desne komore (SPDK) 64 mmHg, globalni longitudinalni strejn (GLS)=11,6% i znaci dijastolne disfunkcije drugog stepena: odnos transmitralne brzine/brzina tkivnog doplera mitralnog anulusa E/e'=15,0. Terapijski pristup obuhvatio je hitnu parenteralnu primenu velikih doza furosemida u toku 3 dana u uslovima dnevne bolnice.(odbio hospitalizaciju i transfuziju krvi). Urađena je korekcija hronične terapije uključujući uvođenje sakubitril-valsartana (ARNI) i inhibitora natrijum glukoza ko-transportera 2 (SGLT2-I) inhibitora, restrikciju unosa soli i tečnosti i intenzivnija korekcija sideropenijske anemije. Tokom ambulantnog praćenja postignuto je značajno poboljšanje: velika diureza, gubitak telesne mase od 14 kg, odlična regresija edema, normalizacija nalaza na plućima sa značajnom redukcijom hidrotoraksa i poboljšanje EF na 47% a diastolne funkcija E/e' na 13,6, dok je SPDK je redukovan na 25 mmHg. Funkcija bubrega je čak normalizovana (GFR= 64 ml/min/1,73 m^2), a hemoglobin je porastao na 128 g/L. **Zaključak:** Ovaj prikaz slučaja ističe značaj individualizovane intravenske diuretske terapije u kombinaciji sa savremenim farmakološkim strategijama kod pacijenata sa najtežim oblikom dekompenzovane hronične srčane insuficijencije klase NYHA IV sa anasarkom. Pravovremeno uvođenje parenteralnih diuretika, optimizacija osnovne terapije i korekcija pridruženih poremećaja doveli su do značajnog kliničkog, ehokardiografskog i laboratorijskog poboljšanja. Ovaj prikaz naglašava važnost kontinuiranog praćenja teškog bolesnika EKG monitoringom i praćenjem drugih vitalnih parametara, kao i prilagođavanja terapije prema diurezi, telesnoj masi, krvnom pritisku i frekvenci pulsa i ehokardiografskim parametrima, radi postizanja optimalnog ishoda uz istovremeno lečenje komorbiditeta uz konsultaciju drugih specijalnosti.

Ključne reči: terminalna hronična srčana insuficijencija sa redukovanom ejekcionom frakcijom leve komore (HFrEF), dekompenzovana kongestivna srčana slabost, anasarka-generalizovani edemi, hronična bubrežna bolest, hronična sideropenijska anemija, farmakoterapija, furosemid, sakubitril-valsartan, dapagliflozin, Bisoprolol, Rivaroxaban, Spironolakton, Digoksin.

Adresa autora: Pavle Nešović, INTERNISTIČKA ORDINACIJA "DR BASTAĆ" ZAJEČAR
E-mail: pavlemnesovic@gmail.com
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SUCCESSFUL TREATMENT OF AN APPARENTLY TERMINAL STAGE OF HEART FAILURE WITH ANASARCA RESULTING IN ACHIEVED CARDIAC COMPENSATION – A CASE REPORT

Pavle Nešović (1), Silvana Babić (2), Mila Bastać (3), Zoran Joksimović (1), Dušan Bastać (1)

(1) INTERNAL MEDICINE PRACTICE "DR BASTAĆ", ZAJEČAR; (2) CLINIC FOR OTORHINOLARYNGOLOGY, UNIVERSITY CLINICAL CENTER OF SERBIA, BELGRADE; (3) MEDSCAN TADIĆ DIAGNOSTICS, ZAJEČAR

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..

UVOD

Hronična srčana slabost je progresivni kompleksni sindrom koji predstavlja završnu fazu različitih kardiovaskularnih oboljenja i jedan je od vodećih uzroka morbiditeta i mortaliteta širom sveta, dok je akutna srčana insuficijencija najteži klinički oblik sa kardiogenim edemom pluća i kardiogenim šokom, sa najvećom smrtnošću, zahteva hitno bolničko lečenje, ali je srećom mnogo ređa od hronične srčane insuficijencije. Prevalencija u opštoj populaciji procenjuje se na 1–2%, dok kod starijih od 75 godina dostiže i do 10% [1,2]. Globalno, više od 64 miliona ljudi živi sa ovim oboljenjem, a očekuje se dalji porast usled starenja populacije [3].

Dekompenzovana hronična srčana insuficijencija karakteriše se zastojem u malom i/ili velikom krvotoku i nakupljanjem tečnosti u organizmu. Najteži oblik ovog procesa je anasarka, difuzni generalizovani edem koji može uključivati ascites, pleuralne izlive i perikardni izliv [4]. Anasarka se, pored srčane insuficijencije, javlja i u drugim stanjima (nefrotski sindrom, ciroza jetre, teške hipoalbuminemije), ali u kontekstu srčane slabosti označava terminalni stadijum, iscrpljenost kompenzatornih mehanizama i lošu prognozu [5]. Dijagnostika i terapija pacijenata sa anasarkom su izazovni, jer je često prisutna kombinacija kardijalne, renalne i hepatičke disfunkcije. Terapija se zasniva na agresivnoj i individualizovanoj intravesnkoj diuretskoj terapiji, korekciji elektrolita, optimizaciji hemodinamike i uključivanju konsultanata [6]. Ovaj prikaz slučaja je jako značajan jer ukazuje da težak oblik srčane insuficijencije, naizgled refrakternog terminalnog stadijuma DHSI može da se uspešno leči intenzivnom terapijom sa dominacijom intravenske diuretske terapije visokim dozama Furosemida uz ostale stubove lečenja HSI u zbrinjavanju pacijenata sa anasarkom. Savremeni pristup lečenju HSI zasniva se na tzv. „četiri stuba terapije“ (ARNI/ACEI, beta-blokatori, MRA i SGLT2 inhibitori), koji značajno smanjuju mortalitet i hospitalizacije [6,7,8,9] a isto tako važan i u anasarki presudan je peti stub- diuretska terapija intravenskim diuretikom henleove petlje.

PRIKAZ SLUČAJA

Osnovni podaci o pacijentu: gospodin K.A., 88 godina, antropometrijski parametri: telesna masa: 78 kg, telesna visina 167 cm, indeks telesne mase (BMI) 29,4 kg/m^2, telesne površine (BSA) 1,9m^2, obim struka 92cm i saturacija krvi kiseonikom (SpO2) 96%.

Pacijent je 06.06.2025**.** godine primljen u stanju teške dekompenzacije hronične srčane insuficijencije sa izraženom kongestijom i anasarkom, što predstavlja klinički indikator uznapredovale bolesti [5].

Anamneza: Pacijent se javio zbog progresivne dispneje, otoka potkolenica i podlaktica i sa izraženom slabošću, sa zamorom i gušenjem na najmanji napor i u miru posle napora. Tegobe su prisutne poslednjih 14 dana, sa brzom progresijom bolesti. Pojačano zamaranje ima unazad dve nedelje (nije mogao da se popne na prvi sprat), praćeno ubrzanim i nepravilnim srčanim radom, velikim otokom potkolenica a zatim i podlaktica. Pregledan od strane interniste u bolnici pre nedelju dana i tada je uvedena mala doza Furosemid tbl 40 mg 1x1 peroralno, Spironolakton tbl 25mg i Rivaroxaban tbl a 15mg 1x1. Negira bolove u grudima. Krvni pritisak (TA) kod kuće je uglavnom niži. Pacijent navodi da je lečio hipertenziju unazad 4 godine a da pre toga nije uzimao lekove za srce.

Predhodna terapija koju je pacijent uzimao duže vreme: Bisoprolol 5 mg tableta (tbl) 1+0+1/2, Rivaroxaban 15 mg tbl 1x1, Ramipril + Hidrohlortiazid 5/25 mg tbl 1x1 ujutru, Ramipril 5 mg tbl 1x1 uveče, Alopurinol 100 mg tbl 1x1, Furosemid 40 mg tbl 1x1, Spironolacton 25 mg tbl 1x1 i suplementacija gvožđa tbl 30mg 1x1.

Fizikalni status na prijemu:

Opšti status: bled i dispnoičan, acijanotičan, afebrilan. Vitalni parametri: TA 110/60, frekvenca srca oko 82/min, nepravilan ritam. Saturacija kiseonikom (SpO_2) je 95%. Koža i sluznice blede.

Pluća auskultatorno: disajni šum oslabljen, obostrano bazalno nečujno disanje; perkutorni zvuk bazalno tmuo obostrano.

Srce: palpatorno proširen iktus, Frekvenca (Fr) 82/min, tonovi oslabljeni, ritam nepravilan po tipu atrijalne fibrilacije, holosistolni regurgitacioni sistolni šum nad iktusom 2-3/6, bez propagacije.

Abdomen: jetra se palpira 4 cm ispod desnog rebarnog luka (DRL) na medioklavikularnoj liniji, meke konzistencije; slezina se ne palpira. Bez znakova za ascit.

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.

Ekstremitetii: Testasti i hladni veliki edemi potkolenica (desno subpatelarno mensuracija daje obim 41cm, na sredini tibije 36cm, supramaleolarno 25cm; leva strana respektivno 35cm, 34cm, 27cm); blag otok šaka i podlaktica.

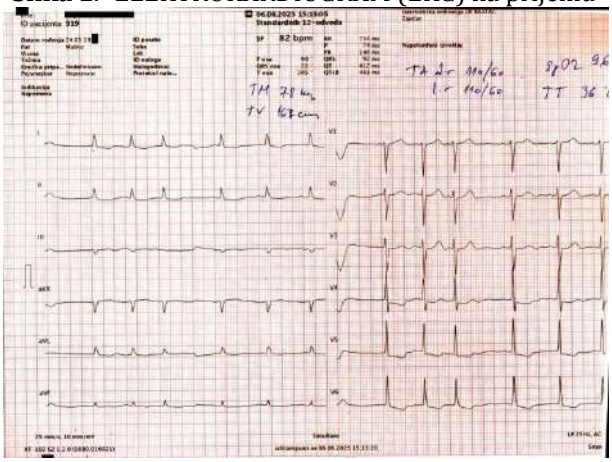
Laboratorijske analize:

NT-proBNP: 1314 pg/ml (<526 za dob pacijenta),
D-dimer: 0,46 (<0.4) µg/ml,
Urea: 17 mmol/L,
Kreatinin: 133µmol/L,
GFR 44 ml/min/1,73 m^2,
Jetreni enzimi: AST 96 U/L, ALT 165 U/L,
Hemoglobin: 68 g/L, Er $3.65x10^{12}$, MCV 67,7 fL

Laboratorijski nalazi su ukazivali na HSI sa kardiorenalnim sindromom i teškom anemijom, koji su česti komorbiditeti i dodatno pogoršavaju prognozu [10,11,12].

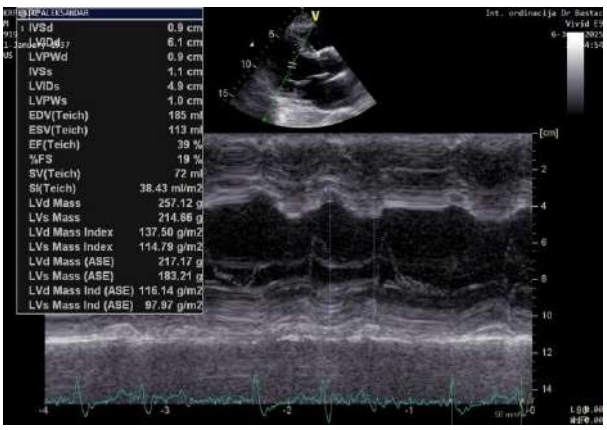
ELEKTROKARDIOGRAM (EKG): Fibrilacija pretkomora sa apsolutnom aritmijom komora, srčana frekvenca Fr=82/min, zbirni vektor srčane osovine intermedijaran, normalno trajanje QRS kompleksa, povremene VES, depresija ST do 2mm i negativan T u V4-V6, depresija ST do 0.5mm i negativan T u D1, D2, aVL (Slika 1).

Slika 1. -ELEKTROKARDIOGRAM (EKG) na prijemu



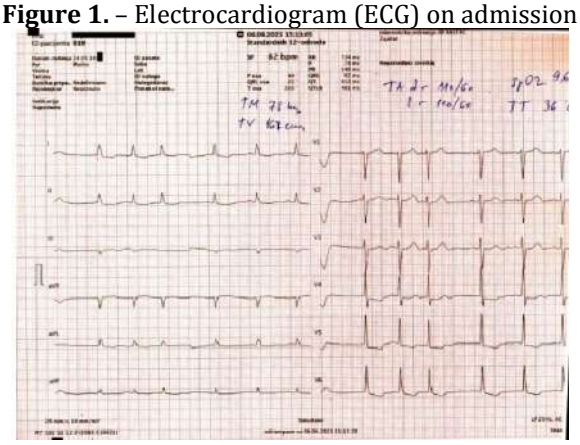
EHOKARDIOGRAFIJA: U nalazu dominira dilatacija leve komore, normalne debljine zidova leve komore , bez hipertrofije miokarda i sa sniženom globalnom sistolnom funkcijom: ejekciona frakcija (EF) leve komore je 39% u M modu (Teicholtz) (Slika 2), a po Simpsonu 35% i 39%, a biplejn 37% (Slika 3,4).

Slika 2. M MOD EHOKARDIOGRAM ejekciona frakcija EF=39% po Teicholz-u



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).).

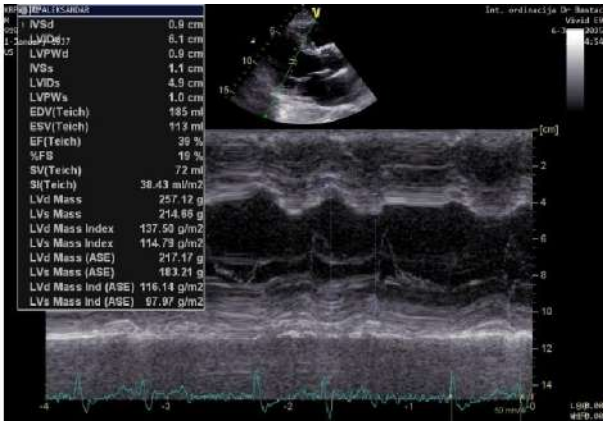
Figure 1. – Electrocardiogram (ECG) on admission



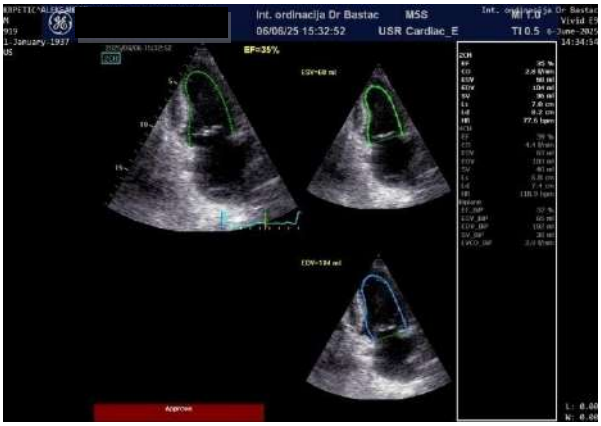
ECHOCARDIOGRAPHY:

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



Slika 3. B-MOD EHOKARDIOGRAM automatska DETEKCIJA ENDOKARDA-EJEKCIONA FRAKCIJA =35% po Simpsonu 2C



Slika 4. B-MOD EHOKARDIOGRAM automatska DETEKCIJA ENDOKARDA-EJEKCIONA FRAKCIJA =39% po Simpsonu 4C



Na inferiornom zidu manja aneurizma bazalnog segmenta sa suspektnim organizovanim trombom. Anteroapikalno i anteroseptoapikalno diskinezija. Najreprezentativniji parametar dijastolne funkcije E/e' odnos je jako povišen 15,0 (normalno <8,0, E/e'- odnos transmitralne brzine ranog punjenja leve komore-E/srednje brizine mitralnog anulusa na tkivnom dopleru-e'). Leva pretkomora je dilatirana, indeks zapremine leve pretkomore (LAVI) je 45 ml/m^2 (normalno<34 ml/m^2) (Slika 5). Izmerena je maksimalna brzina (Vmax) trikuspidne regurgitacije 3,3 m/s (trikuspidni gradijent je 44mmHg) a sistolni pritisak desne komore je 64mmHg.

Ultrazvuk pluća: Hidrotoraks, anteroposteriorno (AP) bazalno pleuralni, hipoehogeni "gust" izliv
levo 9,9 x 9,4cm (Slika 6, 7), APdijametar 6,5x4,6cm na nivou angulusa skapule,
desno 3cm ispod angulusa skapule AP, 7,0 x 7,0cm, lateralno 7,0 x 10,0cm

Slika 5. indeks zapremine leve pretkomore(LAVi)

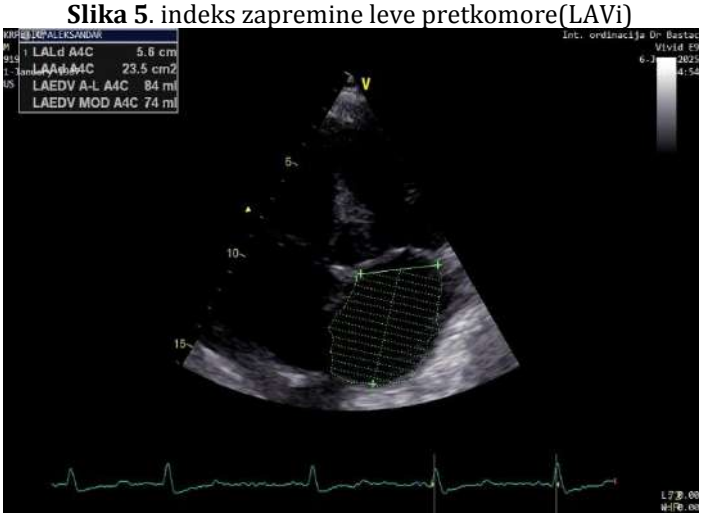


Figure 3. – B-mode echocardiogram: automatic endocardial border detection – ejection fraction (EF) = 35% by Simpson's method (2-chamber view)

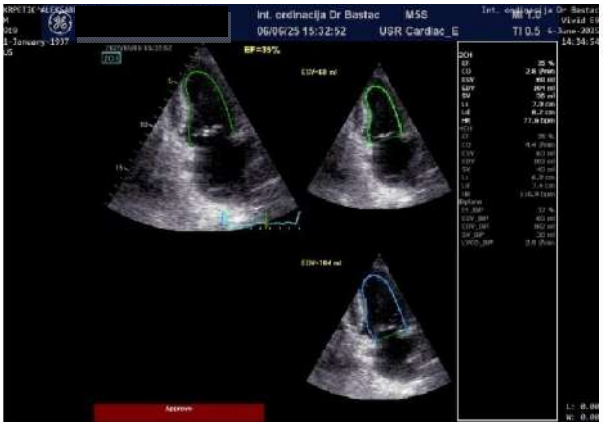


Figure 4. – B-mode echocardiogram: automatic endocardial border detection – ejection fraction (EF) = 39% by Simpson's method (4-chamber view)

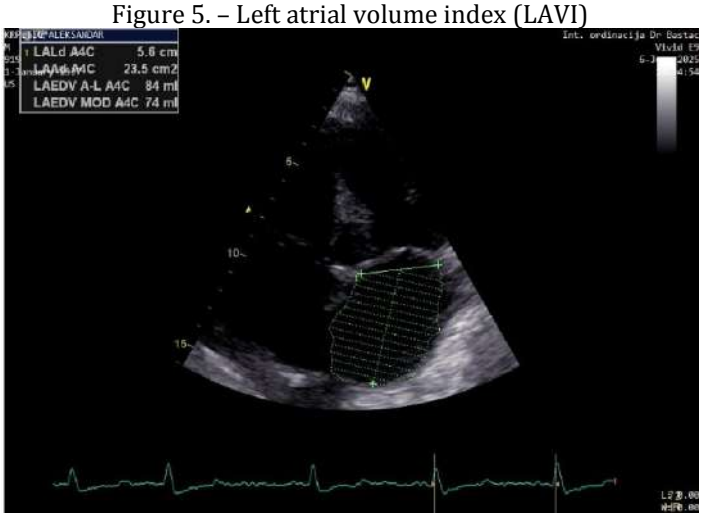


A small aneurysm of the basal segment was observed on the inferior wall, with a suspected organized thrombus. Anteroapical and anteroseptoapical dyskinesia were present. The most representative parameter of diastolic function, the E/e′ ratio, was markedly elevated at 15.0 (normal <8.0; E/e′ represents 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/m² (normal <34 mL/m²) (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.

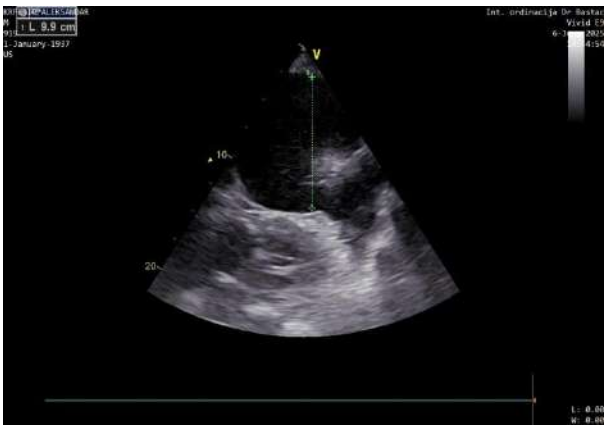
Figure 5. – Left atrial volume index (LAVI)



Slika 6. EHOKARDIOGRAM B MOD HIPOEHOGEN - "GUST" Pleuralni izliv AP DIJAMETAR 9,4 cm SA FIBRINSKIM BRIDAMA



Slika 7. EHOKARDIOGRAM B MOD "Pleuralni izliv CC DIJAMETAR 9,9cm



Radne dijagnoze: Insuff cordis congestiva I50 (srčana slabost sa ehokardiografski redukovanom ejekcionom frakcijom leve komore HFrEF=-35%) sa pratećim kardiološkim dijagnozama: Fibrillatio atriorum permanens, Cicatrix myocardii post infarctum parietis inferioris, Aneurysma functionalis ventriculi sinistri cordis parietis inferioris, Hydrothorax billateralis, Insuff valvulae mitralis et valvulae semilunaris aortae i Hypertensio arterialis pulmonalis. Prisutni su komorbiditeti: Morbus renalis gradus 3b; Anemia microcytica syderopenica chronica gradus gravis.

DALJI TOK BOLESTI DECURSUS MORBI

Pacijent odbija predloženu hospitalizaciju na Internom odeljenju bolnice ZC Zaječar, upozoren na životno ugrožavajuće stanje koje se leči u intenzivnoj nezi bolnice. Praćen je u dnevnoj bolnici naše ordinacije putem EKG monitoringa, merenjem krvnog pritiska i diureze, saturacije krvi i ostalih vitalnih parametara. Zbog anemije indikovana transfuzija deplazmatisanih eritrocita (za koju se nije javljao Službi za transfuziju).

Pacijentu je odmah uvedena intenzivirana parenteralna diuretska terapija: na dan pregleda Furosemid amp a 20mg No VIII (160mg) u 2 intravenska bolusa, što je u skladu sa preporukama za lečenje akutne dekompenzacije [6,13,14].

Rana i agresivna diuretska terapija dovela je do značajne redukcije volumenskog opterećenja, što je ključni terapijski cilj [13,14,15]. Dobija se odlična, promptna diureza. Sutradan dobija Furosemid intravenski amp a 20mg No IV (80mg). Korigovana je predhodna kućna terapija: Bisoprolol tbl 5mg 2x1/2; Rivaroxaban 15mg 1x1; Spironolakton 25mg 1x1 i suplement gvožđa a prekinuta terapija tbl Ramipril + Hidrohlortiazid 5/25mg 1x1ujutru, tbl Ramipril 5mg 1x1uveče, tbl Alopurinol 100mg 1x1. Uvedeni su ARNI: Valsartan+Sakubitril 26/24mg 2x1/2; tbl Dapagliflozin 10mg 1 x 1 što je u skladu sa savremenim preporukama [7,8,16-18]. Zbog bolje korekcije anemije uvedena je maksimalna doza preparata gvoždja umesto ranijeg suplementa gvoždja (300mg/dan).

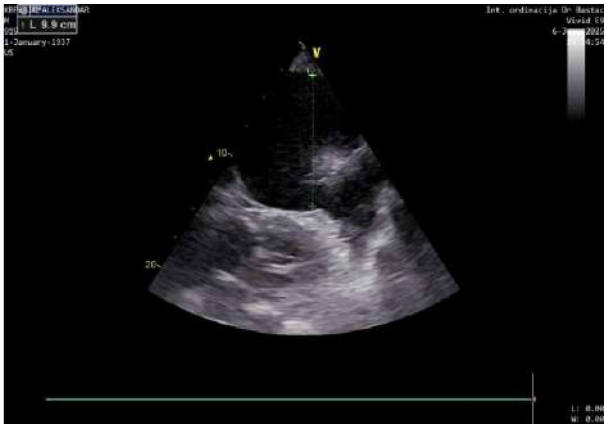
Korekcija anemije sprovedena je zbog njenog negativnog uticaja na funkcionalni status i ishod bolesti, pojačavajući tkivnu hipoksiju [12,19]. Pacijentu je od nefarmakoloških mera lečenja savetovana restrikcija unosa tečnosti i soli i zabrana fizičkih aktivnosti. Uz ovu terapiju postepeno je došlo do sve veće diureze i značajnog smanjenja edema.

Na prvoj kontroli već trećeg dana lečenja (07.06.2025. godine). utvrđuje se odličan uspeh u eliminaciji retencije tečnosti: pacijent je imao 7 kg manje, uz prestanak dispneje na najmanji napor i uz značajnu redukciju edema potkolenica (mensuracija: desna 40cm, 35cm, 24cm, leva 36cm, 34cm, 24cm) i porast hemoglobina na Hgb 72 g/L. Tada se od 4-tog dana u peroralnu terapiju uvodi Furosemid forte 1/2 tbl a 500mg , Digoksin tbl a 0.25mg 1/2 na drugi dan zbog atrijalne fibrilacije i hipotenzije. Upućen na višeslojnu kompjuterizovanu

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..

tomografiju MDCT skener grudnog koša, koji u daljem toku nije uradio.

Na drugoj kontroli petog dana lečenja (11.06.2025. godine) pacijent ima 12kg manje i ima minimalne edeme (mensuracija obima potkolenica: desna 33 cm, 33cm, 24cm a leva: 34cm, 33cm, 24cm). Nalaz na plućima: disajni zvuk normalan, bazalno lako oslabljeno disanje, ekspirijum nije produžen, perkutorni zvuk bazalno tmuo od 10-og rebra. Na ultrazvučnom pregledu srca došlo je do poboljšanja ejekcione frakcije i poboljšanja dijastolne funkcije odnosno komplijanse leve komore (odnos E/e' 7,5.). Dobijeni su laboratorijski rezultati: Serumsko gvožđe <1μmol/l (normalno 11-31), Feritin 19,2ng/mL (normalno 20-250 ng/mL).

Kontrola nakon dve nedelje (19.06.2025. godina): odlično poboljšanje kliničkog stusa se održava, sada još 2 kg manje (ukupno 14 kg manje od početka lečenja) što je odličan efekat primenjene terapije. Žali se na vrtoglavicu i nestabilnost koju povezuje sa hipotenzijom, TA=90/55 mmHG i 80/50 mmHg, pa se prema tome redukuju doze lekova koji deluju hipotenzivno: Furosemid tbl a 500mg 1/2 na drugi dan, i tbl sacubitril-valsartan26/24mg 2 x 1/4. Zbog bolova u podkolenicama dodaje se lek protiv venskih otoka Diosmin + Hesperidin tbl a 1000mg 1x1.

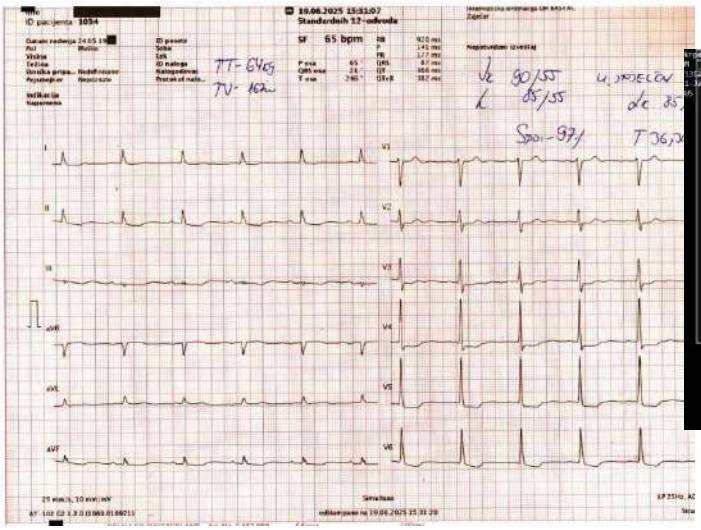
Novi relevatni rezultati laboratorijskih analiza: Hgb 88g/l, sedimentacija (Se) 55mm/h, urea 15,4 mmol/L, kreatinin 131μmol/L, GFR 44,8mL/min/1,73m^2, kalijum 4,4mmol/L.

EKG Fibrilacija pretkomora sa apsolutom komora, fr 65/min, depresija ST do 2mm i negativan T V4-V6, depresija ST do 0,5mm i negativan T u D1, D2, aVL (Slika 8).

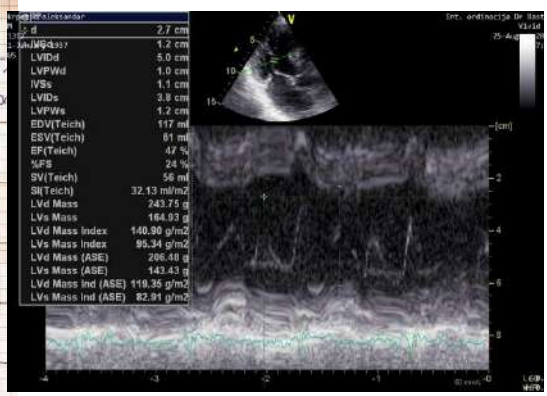
Rutinska kontrola posle 2 meseca 25.08.2025. godine. Pacijent bez tegoba, ima još 2kg manje, noge ne otiču, nije više hipotenzivan. Impresivan porast Hemoglobina na 128 g/L zbog terapije gvožđem ali i zbog korigovanja hemodilucije. Normalizacija bubrežne funcije GFR=64mL/min/1,73m^2. Ultrazvučnim pregledom srca (Slike 9,10 i 11) se registruje odličan porast ejekcione frakcije M mod EF 47%, a po Simpsonu BIPLEJN EF je 46% uz redukciju dilatacije leve komore (LK EDD=50mm, a ESD=40mm) i iščezavanje plućne hipertenzije (RVSP =25 mmHg).

Urađena je dalja korekcija terapije održavanja i redukcija doze Furosemid tbl a 500 mg 1/4 na drugi-treći dan.

Slika 8. - EKG na kontroli nakon 2. nedelje



Slika 9. M MOD Ehokardiogram Kontrolna ejekciona frakcija EF=47% po Teicholz-u



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

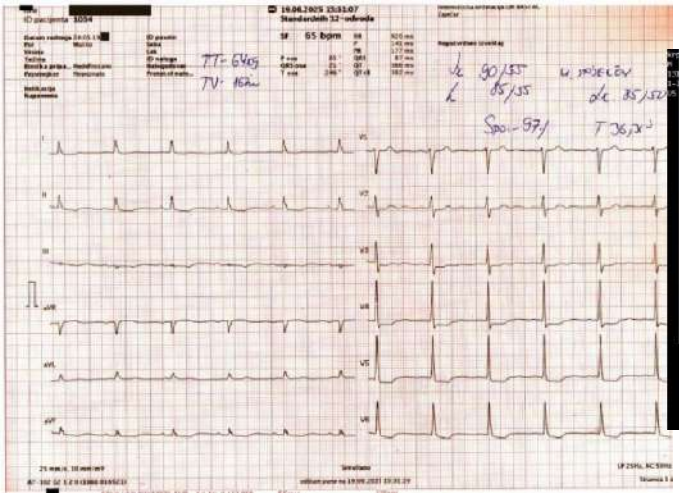
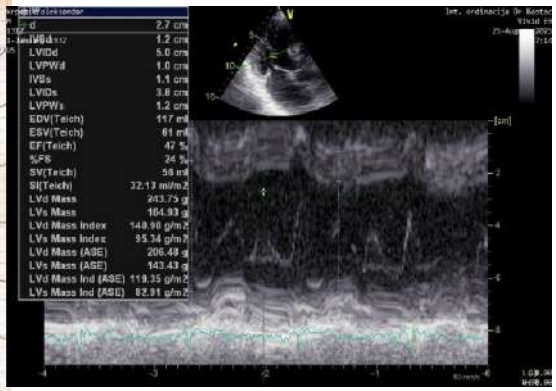
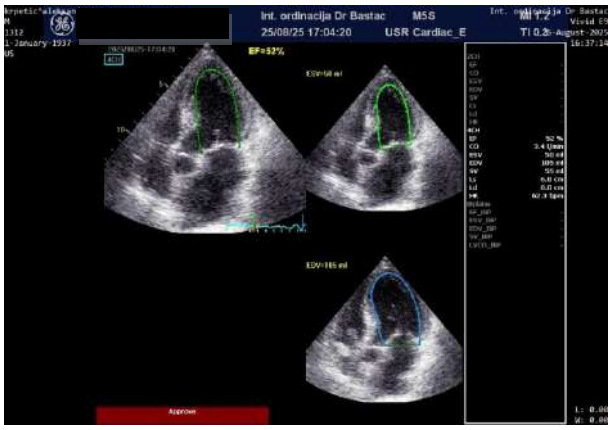


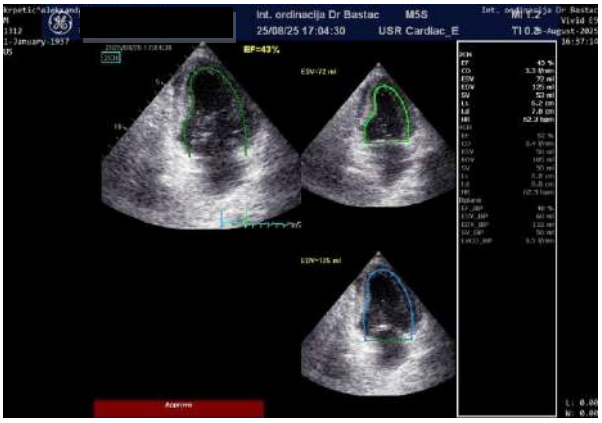
Figure 9. – M-mode echocardiogram: follow-up ejection fraction (EF) = 47% according to Teichholz method



Slika 10. B-MOD EHOKARDIOGRAM Ejekciona frakcija leve komore EF = 52%monoplane po Simpson-u 4C



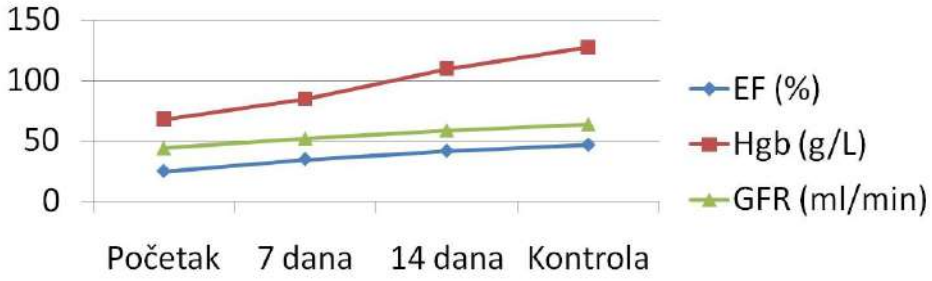
Slika 11. B mod ehokardiogram Ejekciona frakcija leve komore EF =43%monoplane po Simpson-u 4C



KLINIČKI ISHOD:

Nakon intenzivne intravenske diuretske terapije i posle 3 dana dnevne bolnice sa EKG monitoringom i praćenjem vitalnih parametara, došlo je do značajnog kliničkog poboljšanja. Tada se obavlja prelazak na peroralnu terapiju. Pacijent je izgubio ukupno 14 kg u periodu od dve nedelje, sa iščezavanjem perifernih edema, normalizacijom disajnog zvuka na plućima. Nastaje minimalizacija pleuralnih efuzija, održava se dobra ejekciona frakcija oko 46 do 47 % i poboljšanje dijastolne funkcije na ehokardiografiji. Laboratorijski parametri su pokazali postepeno poboljšanje renalne funkcije (GFR sa 44 na 64 ml/min/1,73 m^2) i značajan porast hemoglobina nakon primene preparata gvožđa (Grafikon 1 i 2).

Grafikon 1. Trend promena kliničkih parametara tokom terapije



Grafikon 2. Uporedni prikaz pre i posle efekta terapije

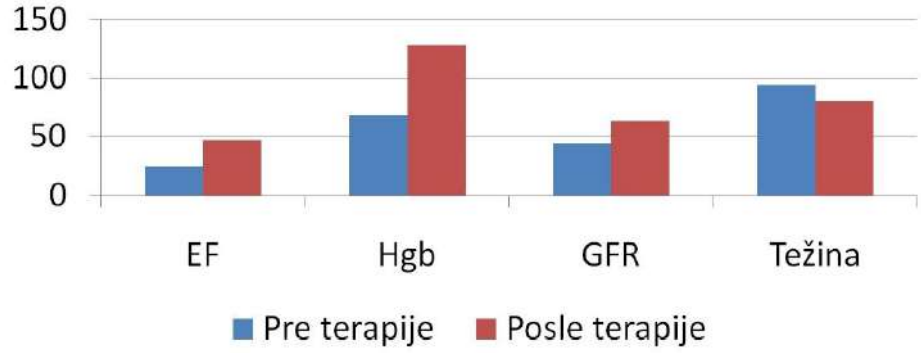


Figure 10. – B-mode echocardiogram: left ventricular ejection fraction (EF) = 52% (monoplane, Simpson's method, 4-chamber view)

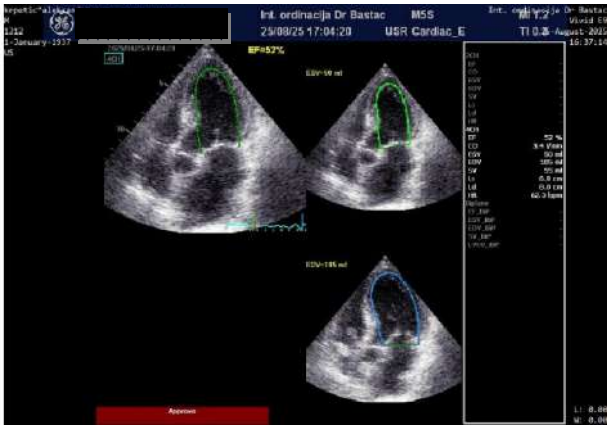
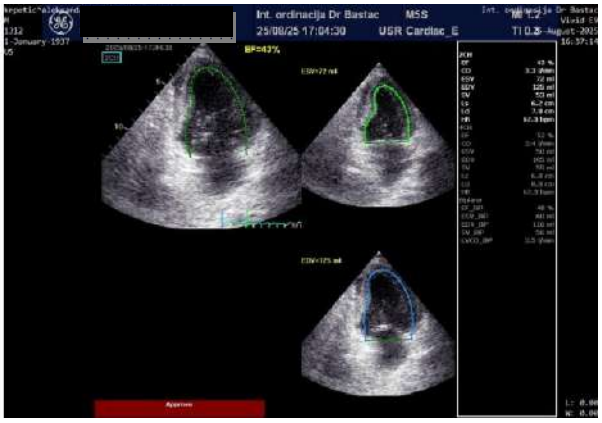


Figure 11. – B-mode echocardiogram: left ventricular ejection fraction (EF) = 43% (monoplane, Simpson's method, 4-chamber view)



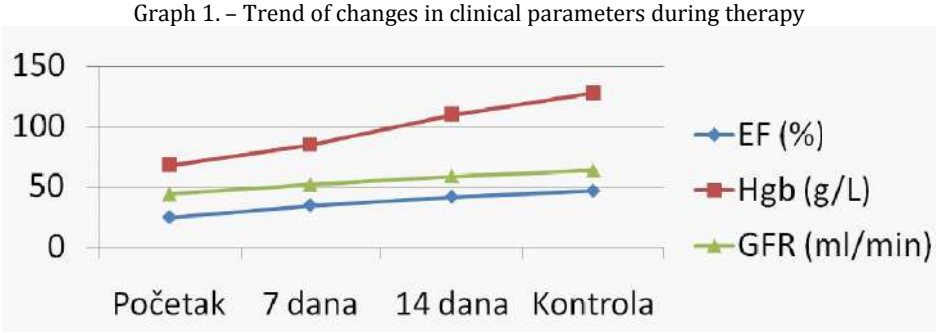
CLINICAL OUTCOME:

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.

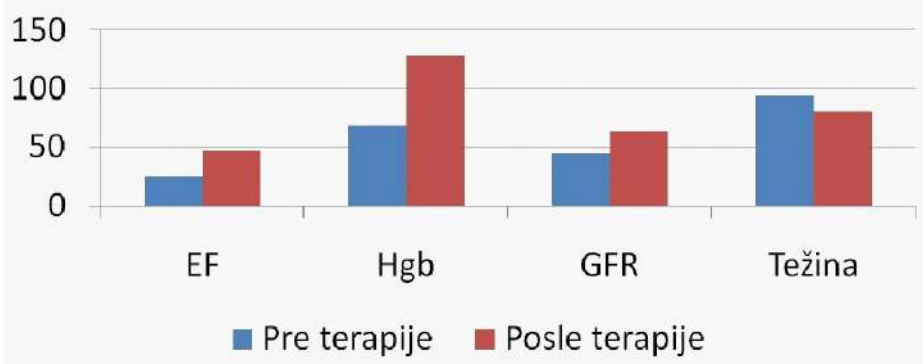
A total weight loss of 14 kg was recorded over a two-week period, accompanied by complete resolution of peripheral edema and normalization of lung auscultation findings. Pleural effusions regressed to minimal levels. Left ventricular systolic function remained preserved at approximately 46–47% ejection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



DISKUSIJA

Anasarka predstavlja ekstremni oblik retencije tečnosti i marker uznapredovale srčane insuficijencije sa lošom prognozom [5].

Diuretici ostaju osnov terapije kongestije, pri čemu intravenska primena omogućava bržu i efikasniju dekongestiju [6,13,14]. Međutim, njihova primena zahteva pažljivo praćenje zbog mogućeg pogoršanja bubrežne funkcije [10,20], hipokaliemije i retko hipovolemije i dehidratacije.

U ovom slučaju, poboljšanje renalne funkcije nakon terapije ukazuje na reverzibilnost kardiorenalnog sindroma nakon uklanjanja kongestije, što je već opisano u literaturi [10,11]. Savremene studije pokazuju da rana primena SGLT2 inhibitora dovodi do brzog kliničkog benefita i smanjenja hospitalizacija [17,21,22,23]. ARNI terapija dodatno doprinosi poboljšanju remodelovanja miokarda i smanjenju mortaliteta [16]. Savremene preporuke naglašavaju simultanu ili ranu sekvencijalnu primenu pet osnovnih terapijskih stubova, što je povezano sa najboljim ishodima [7,8,9,24].

Upravljanje kongestijom ostaje ključni terapijski cilj, a individualizovana intravenska primena diuretika uz praćenje telesne mase, diureze i renalne funkcije predstavlja osnov uspešnog lečenja [14,15]. Anemija je čest komorbiditet kod srčane insuficijencije i njena korekcija, posebno intravenskim preparatima gvožđa, poboljšava simptome i kvalitet života [12,19]. Ovaj slučaj ukazuje na značaj pravovremenog uvođenja intenzivne diuretske terapije, stalnog praćenja diureze i laboratorijskih parametara, kao i individualizovanog pristupa u zavisnosti od komorbiditeta. Ovaj prikaz slučaja takođe pokazuje da je i ambulantni pristup, uz adekvatan nadzor, moguć kod selektovanih bolesnika sa teškom dekompenzovane hronične srčane insuficijencije, naizgle terminalne i refrakterne na lečenje ali se ovakvi bolesnici najčešće leče u hospitalnim uslovima [25].

ZAKLJUČAK

Dekompenzovana srčana slabost sa anasarkom predstavlja ozbiljno i životno ugrožavajuće stanje koje zahteva agresivan, ali pažljivo titriran individualni terapijski pristup. Diuretska terapija ostaje osnovna mera u kontroli volumenskog opterećenja. Ovaj prikaz slučaja ističe značaj individualizovane diuretske terapije u kombinaciji sa savremenim farmakološkim strategijama kod pacijenata sa najtežim oblikom dekompenzovane srčane insuficijencije i anasarkom. Pravovremeno uvođenje velikih doza parenteralnih diuretika, optimizacija osnovne terapije i korekcija pridruženih poremećaja doveli su do značajnog kliničkog i laboratorijskog poboljšanja ovog bolesnika.

Ovaj prikaz slučaj naglašava važnost davanja intravenske terapije sa kontinuiranog EKG monitoringa u toku opservacija i praćenjem a i prilagođavanja terapije prema diurezi, krvnom pritisku i frekvenci pulsa, nalazu kalijuma i azotnih materija u serumu, krvnog pritiska i saturacije krvi kiseonikom, radi postizanja optimalnog ishoda. Posebno se naglašava značaj individualizacije terapije i pravovremenog prepoznavanja refraktornosti na standardne mere lečenja u sklopu hronične peroralne terapije. Kombinacija intenzivne diuretske terapije i savremenih farmakoloških strategija omogućava značajno kliničko poboljšanje čak i kod bolesnika sa uznapredovalom bolešću, kao što je naš prikaz slučaja i koji je uspešno kardijalno komopenzovan u ambulantnim uslovima dnevne bolnice.

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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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