

SEVERE DECOMPENSATED HEART FAILURE WITH ANASARCA ,PRESERVED EJECTION FRACTION and GLOBAL LONGITUDINAL STRAIN, CAUSED BY TRANSIENT CONSTRICTIVE PERICARDITIS AND AN ASSOCIATED CHRONIC MYELOPROLIFERATIVE PROCESS – report of a controversial case with complete cardiac compensation under therapy

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Summary: Introduction. Constrictive pericarditis is a complex clinical and hemodynamic syndrome characterized by restricted diastolic filling of the heart due to pathological changes of the pericardium - loss of tissue elasticity, and, less often, it may occur in a transient form. In advanced constriction, the clinical presentation is systemic venous congestion with ascites, hepatomegaly and anasarca, with preserved left ventricular systolic function, which is why, in the differential diagnosis, it may mimic heart failure with preserved ejection fraction (HFpEF), restrictive cardiomyopathy or other causes of predominantly right-sided heart failure, but also with pleural effusions and pulmonary congestion as a sign of additional left ventricular failure. Echocardiographic assessment plays a central role in recognizing constrictive pathophysiology, but a definitive diagnosis in complex cases requires a multimodality approach, sometimes including pericardial biopsy. **Case report.** We present a 73-year-old female patient with previous conditions of arterial hypertension, pericardial adhesions and a small encapsulated pericardial effusion from 2014, mild mitral regurgitation (since 2014), chronic renal failure, type 2 diabetes mellitus on insulin therapy and hypothyroidism, who developed severe global heart failure with dominant signs of systemic venous congestion. At her own request, she was discharged from the Internal Medicine Department of the Zaječar Health Center. The clinical picture consisted of orthopnea, bilateral pleural effusions, ascites, hepatosplenomegaly, peripheral edema and anasarca. Echocardiography revealed preserved left ventricular systolic function (EF 60–70%), paradoxical movements of the interventricular septum, pericardial thickening of 6–9 mm, parallel movements of the pericardial layers, right ventricular dilatation and dysfunction, significant tricuspid regurgitation and pulmonary hypertension with a maximum recorded right ventricular systolic pressure (RVSP) of 76 mmHg. After intensive decongestive therapy, including intravenous diuretics and inotropic support with dobutamine infusions, corticosteroid and anti-inflammatory therapy was given according to the algorithm for the treatment of constrictive pericarditis. Bilateral evacuating pleural punctures were also performed, and significant clinical and hemodynamic improvement was finally achieved. In parallel with corticotherapy, a nonsteroidal anti-inflammatory antirheumatic drug and colchicine were introduced. The overall therapeutic effect was the loss of dyspnea, complete clinical cardiac compensation with a reduction in body weight from 77 kg to 52 kg over 28 days, with complete resolution of pleural effusions, ascites and peripheral edema, as well as a reduction in RVSP from 76 to 44 mmHg. During follow-up, progressive leukocytosis from $50 \times 10^9/L$ to over $90 \times 10^9/L$, known since 2024, was recorded. A peripheral blood smear showed the presence of all developmental forms of the granulocytic lineage with a predominance of myelocytes and metamyelocytes and less than 2% blasts, which indicated a chronic myeloproliferative process. A hematologist was consulted, but the patient refused hospitalization at the hematology department of a higher-level institution. **Conclusion.** In patients with marked systemic venous congestion, hepatosplenomegaly, ascites and preserved left ventricular systolic function with pleural effusions and pulmonary congestion, constrictive pericarditis should be considered as a possible cause of heart failure. The simultaneous presence of marked leukocytosis, severe splenomegaly and a finding compatible with a chronic myeloproliferative process raises the possibility of secondary pericardial involvement by a hematological disease. This case highlights the importance of a multidisciplinary approach and the need for a complete hemodynamic and hematological evaluation, which is sometimes impossible in practice.

Keywords: constrictive pericarditis; HFpEF; right-sided heart failure; pulmonary hypertension; hepatosplenomegaly; pleural effusion; leukocytosis; myeloproliferative process.

INTRODUCTION

Constrictive pericarditis. Pericardial constriction is a chronic condition characterized by a thickened, heavily fibrotic and inelastic, and often calcified, pericardium, which leads to impaired diastolic filling of the heart. The term constrictive pericarditis (CP) should be used when pericardial constriction is associated with pericarditis [1]. Constrictive pericarditis is a consequence of chronic inflammation of the pericardium and is characterized by scarring, fibrosis and complete loss of pericardial elasticity. The main symptoms are dyspnea, edema, orthopnea and fatigue. The classic clinical picture is most often characterized by isolated right-sided heart failure, with normal or near-normal natriuretic peptide levels. Signs of venous congestion, hepatomegaly, pleural effusions and ascites may occur [2,3,4]. Constrictive pericarditis should initially be treated with empirical anti-inflammatory therapy for at least 3 to 6 months according to the 2025 recommendations of the European Society of Cardiology [1,5]. Patients in whom medical treatment has not led to improvement, as well as patients with pericardial constriction not associated with pericarditis, should be referred without delay for cardiac surgical treatment, i.e. pericardiectomy [1,5]. Severe hypotension may be a sign of chronic pericarditis with constriction and cardiac tamponade (CTP – constrictive tamponade physiology), which clinically manifests as Beck's triad: hypotension, jugular venous distension and muffled heart sounds.

Transient constrictive pericarditis. Transient constriction is a form of constrictive pericarditis (CP) with active inflammation present, which may resolve after anti-inflammatory therapy or regress spontaneously. It develops during pericarditis with or without pericardial effusion (effusive-constrictive pericarditis). In more than 50% of cases, constriction resolves after anti-inflammatory therapy [6,7,8]. In patients with newly diagnosed constrictive pericarditis, a trial of anti-inflammatory therapy lasting 3 to 6 months is recommended before referral for pericardiectomy [9,10]. To demonstrate active pericardial inflammation, the use of biomarkers (e.g. elevated C-reactive protein) and multimodality imaging is necessary, including cardiovascular magnetic resonance (CMR) [10,11].

Constrictive pathophysiology

It is important to distinguish constrictive pathophysiology from actual symptoms of heart failure [3,6,7,12]. Echocardiographic and hemodynamic signs of constriction may be present, such as: "septal bounce" (paradoxical or abrupt movement of the interventricular septum), respiratory phase-dependent shift of the interventricular septum and Doppler features of constriction, while the patient may be free of heart failure symptoms. The non-inflammatory phenotype of pericarditis is a form whose pathogenesis, with normal C-reactive protein levels, is unknown, and these cases are often therapeutically challenging and more difficult to treat [13].

Treatment of constriction

The algorithm for the treatment of constriction uses clinical data, biomarkers and imaging methods, including CMR (cardiovascular magnetic resonance), CT (computed tomography) and ECG (electrocardiogram) [1,5].

Empirical anti-inflammatory therapy is justified only in patients with evidence of pericarditis based on clinical criteria, biomarkers and/or imaging findings. Stratification of constrictive pericarditis by response to therapy

- *Low risk:* clinical remission after therapy, normalization of imaging findings.
- *Intermediate risk:* improvement of symptoms without complete remission, partial regression of signs of constriction.
- *High risk:* persistent symptoms, permanent signs of constriction on imaging.

Constrictive pericarditis is a complex clinical syndrome resulting from impaired diastolic filling of the cardiac ventricles due to thickening, fibrosis, inflammatory changes or other pathological changes of the pericardium. The clinical picture depends on the degree and duration of constriction, and signs of systemic venous congestion often dominate, including peripheral edema, ascites, hepatomegaly, pleural effusions and exercise intolerance. At the same time, left ventricular systolic function may remain preserved, which can make differentiation from HFpEF and restrictive cardiomyopathy difficult [1,14].

Echocardiography plays a central role in the initial recognition of the constrictive physiological pattern. Of particular importance are paradoxical movements of the interventricular septum, respiratory variations

in diastolic filling, the so-called annulus reversus: a higher tissue Doppler velocity of medial e' than of lateral e' , and certain hepatic vein flow patterns. A combination of several echocardiographic parameters increases diagnostic reliability, while individual signs are not sufficient for a definitive diagnosis [13].

In patients with preserved ejection fraction, heart failure may result from impaired diastolic function and elevated filling pressures, whereby secondary pulmonary hypertension and right ventricular failure may also play a significant role. Current criteria for HFpEF therefore require, in addition to symptoms and preserved ejection fraction, objective evidence of structural or functional abnormalities and/or elevated filling pressures [16].

A particular diagnostic problem in this case is the simultaneous presence of marked leukocytosis, hepatosplenomegaly and pathological changes of the pleura and pericardium. In such a context, a hematological process may play a significant role in the differential diagnosis, including infiltrative and extramedullary processes. However, the peripheral blood finding alone is not sufficient to establish a definitive diagnosis of myeloproliferative neoplasm [17-21].

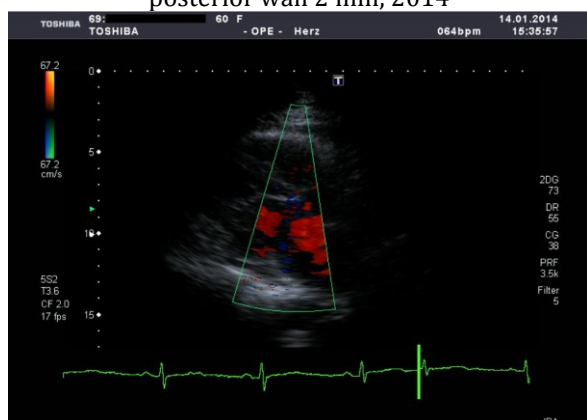
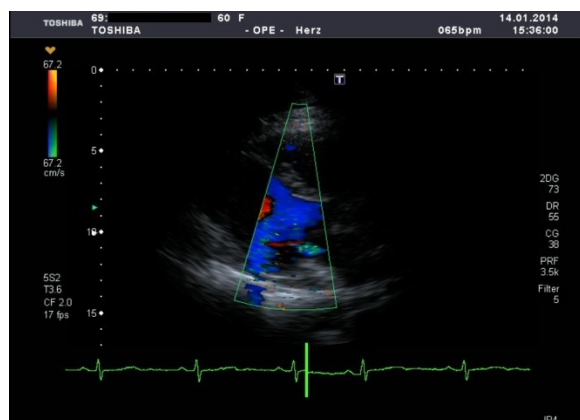
The aim of this report is to present the complex clinical course of a patient with severe systemic congestion, preserved left ventricular systolic function, echocardiographic signs of suspected constrictive pericardial physiology, pulmonary hypertension and a concurrent finding of marked leukocytosis compatible with a myeloproliferative process, as well as the diagnostic difficulties due to the patient's non-cooperation.

CASE REPORT:

MEDICAL HISTORY: We present a 73-year-old female patient with long-standing cardiovascular problems: hypertension, mitral regurgitation, pericardial adhesions and a small pericardial effusion from 2014. Based on the documentation she brought, newly developed G3 kidney disease is recorded; for 10 years she has had type 2 diabetes mellitus on insulin therapy, as well as hypothyroidism and marked leukocytosis. In May 2026, the patient was hospitalized at the Internal Medicine Department of the Zaječar Health Center due to a feeling of shortness of breath (dyspnea) and received therapy from which she did not feel any significant improvement. During the 2 months after this hospitalization in 2026, a pronounced clinical picture of systemic venous congestion developed, with orthopnea, bilateral pleural effusions, ascites, peripheral edema and hepatosplenomegaly, and typical ANASARCA. On 13.07.2026, i.e. the day before admission to the "Dr Bastać" Practice, she was discharged at her own request from the Internal Medicine Department of the Zaječar Health Center.

MAIN COMPLAINTS are marked fatigue, inability to walk, instability, episodes of fainting, a feeling of severe suffocation, difficulty breathing, shortness of breath, occasional stabbing chest pain and leg swelling.

The first contact was back in 2014 - at that time, echocardiography revealed mild mitral regurgitation, separation of the pericardial layers, with adhesions (Figures 1 and 2) on the lateral wall - signs of adhesive pericarditis, and a small encapsulated effusion behind the posterior wall of 2 mm. Tissue Doppler showed that: systolic and diastolic annular velocities were reduced, E/e' ratio 10,6 borderline. In the laboratory findings, only thrombocytopenia dominates, and since then she has been on bisoprolol 2,5 mg. Thrombocytopenia,

Figure 1. small encapsulated effusion behind the posterior wall 2 mm, 2014**Figure 2.** mild mitral regurgitation, 2014

Now, on admission, she gives no information and has no documentation that she was hospitalized at the hematology department of the Clinical Center Niš in 2024. It was later learned from the heteroanamnesis that a bone marrow biopsy had been performed, but the sample was not diagnostic. She refused the proposed further treatment and was discharged with a diagnosis of splenomegaly and suspected chronic myeloid leukemia. She also had laparoscopic gallbladder surgery with complications and conversion to classic laparotomy due to a calculus in the common bile duct.

CURRENT previous therapy: spironolactone 100mg 2x1, Bisoprolol 2,5mg 1x1, Empagliflozin 10mg 1x1, Ramipril- 5mg 1x1/2, Thyroxine 25mcg 2x1, Furosemide forte 500mg 1x1.

STATUS PRAESENS ON ADMISSION: 14.07.2026 – inspection reveals extreme dyspnea, Tachypnea, distended neck veins, orthopnoea- can lie down, examination difficult overall. Blood pressure was 130/70 mmHg, body weight 77 kg, and SpO₂ 95%. She refuses hospitalization and referral to a tertiary institution (written statement).

COR: HR 75 min, heart sounds muffled, rhythm regular, systolic regurgitant murmur over Erb's point 2/6. **PULMO:** breath sounds diminished bilaterally at the bases, dullness to percussion up to both scapular angles, early and late

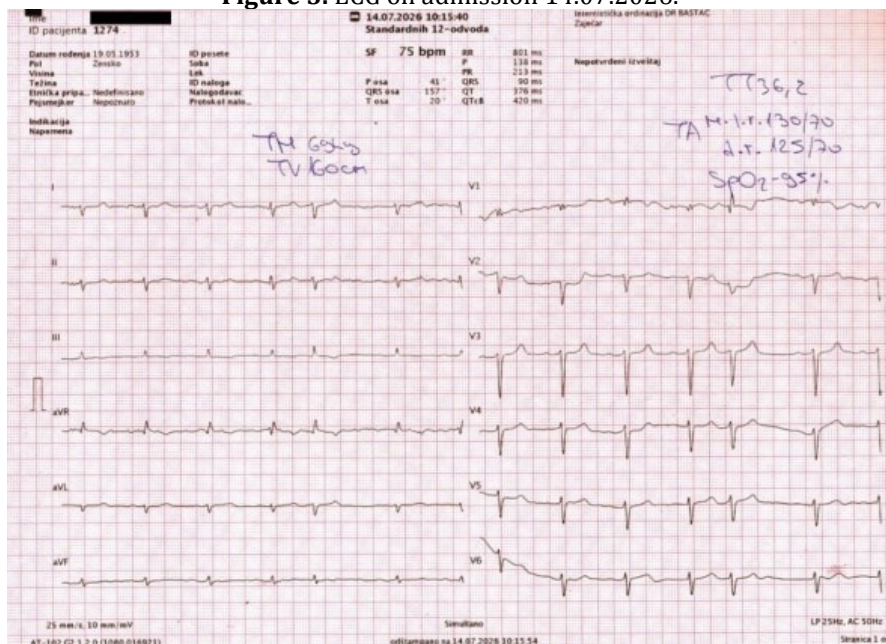
inspiratory crackles above the dullness, signs of large bilateral pleural effusions (hydrothorax). **ABDOMEN:** Soft and non-tender on palpation. The liver is palpable 4 cm below the RCM, the spleen is palpable up to 4 cm below the LCM. **EXTREMITIES:** large cold doughy edema of the lower legs 3+, circumference measurement: left leg 50/39/27cm, right leg 47/36/27cm, peripheral arterial pulses are symmetrically palpable.

LABORATORY ANALYSES ON ADMISSION 14.07.2026.

Urgent "point of care" laboratory analyses performed at the "Dr Bastać" Internal Medicine Practice **dominated by:** very high N-terminal pro-B-type natriuretic peptide (NT-proBNP 1175 pg/mL), signs of renal dysfunction with creatinine 122 µmol/L and GFR 37 mL/min/1,73 m², as well as hyponatremia of 130 mmol/L. hyperuricemia: 774 µmol/L, albumin normal 37 g/L, leukocytes 55x10⁹/cm³, neutrophilia 84%.

ECG ON ADMISSION: sinus rhythm with frequent supraventricular extrasystoles SVES, HR 75/min, low voltage of R waves, no R progression to v6 with deep S, extreme right axis with incomplete right bundle branch block, QRS duration 90ms and QTc interval 420ms normal, overall depolarization vector right-sided, rS in D1-2, aVL. rsr in V1, no R progression to V6, deep S in V2-V6 and no ST changes.

Figure 3. ECG on admission 14.07.2026.



Ultrasonography of the pleura and lungs and echocardiography on admission (technically difficult due to orthopnea)

left - pleural effusion thickness approx. 9,7cm x 12,4cm x 7,6cm, right - effusion approx. 5,3cm x 9,1cm x 4,4cm

Minimal ascites on the right between the liver and kidney 11 mm -12 mm, in the pouch of Douglas 9 mm (Figures 4 and 5).

Echocardiographic examination revealed a dilated left atrium, moderate mitral regurgitation (MR), MR vena contracta 4mm, with normal left ventricular dimensions. The interventricular septum showed paradoxical movements - septal bounce shift, and preserved

left ventricular systolic function was recorded, (Teicholtz EF=76%, and EF Biplane Simpson rule 65%, Figure 4). Diastolic dysfunction - impaired relaxation and compliance - was also measured ($E/e'=21,8$, $E/A=2,0$ and $DTE=113ms$) (Figure 5). The right ventricle was mildly dilated, with **TAPSE reduced to 12 mm** and significant tricuspid regurgitation TR 3+ and signs of moderate pulmonary hypertension: elevated right ventricular systolic pressure $RVSP=52$ mmHg. **The pericardial layers were separated and moved in parallel, with pericardial thickening: behind the posterior wall of the left ventricle 7-8 mm-/** (Figures 6 and 7) / [12,2,13].

Figure 4. Collapsed lung parenchyma in the effusion 14.07.2026.

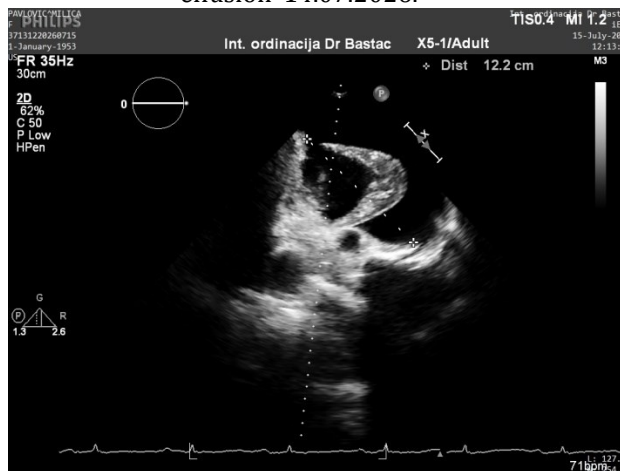


Figure 5. Large "dense" pleural effusion 14.07.2026.

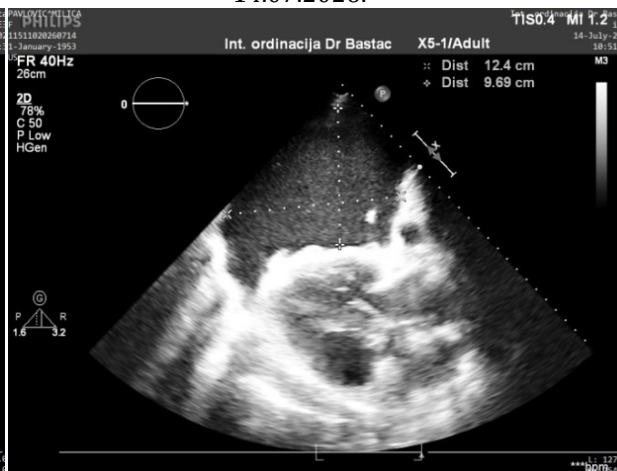
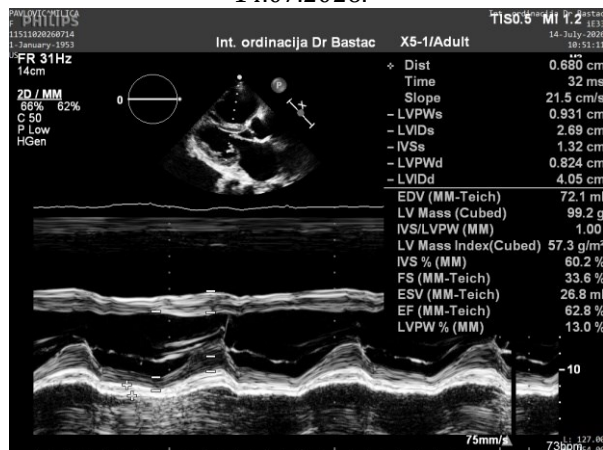
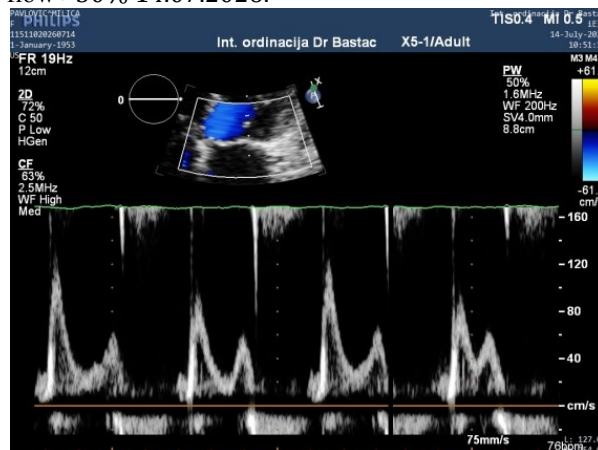


Figure 6. Paradoxical septal movements
14.07.2026.**Figure 7.** Increased respiratory variation of mitral flow >30% 14.07.2026.

The next day, 15.07.2026, the patient was still orthopneic, 3 kg lighter and with anasarca.

At that time the echocardiographic examination was supplemented (Table 1). Echocardiography revealed preserved left ventricular systolic function, EF 60 to 70%, paradoxical movements of the interventricular septum, (Figures 6 and 8) and pericardial thickening of 6–9 mm, parallel movement of the pericardial layers, right ventricular dilatation and dysfunction, (Figure 9) significant tricuspid regurgitation and

pulmonary hypertension with an increase in RVSP to 76 mmHg. The finding was interpreted as a syndrome of global decompensated heart failure with preserved left ventricular ejection fraction (HFpEF) with predominant right ventricular failure, secondary pulmonary hypertension and signs of constrictive pericardial pathophysiology - strain reversus (Figure 8) and respiratory variation of Doppler flow greater than 25% (Figures 7 and 9). Constrictive pericarditis was suspected [1,13,14].

Table 1. supplemented echocardiographic examination tISSUE DOPPLER AND STRAIN

	1	2	REF VAL 1	REF VAL 2
Septal e' (mitral annulus velocity Tissue dop PW) / S'systolic velocity [cm/s]	4,6/26	7,6	≥ 8	> 6
Lateral e' (mitral annulus velocity Tissue dop PW)/ S'systolic velocity [cm/s]	6,5/16,7	8	≥ 10	> 6
E/e' average (normal ≤ 8)	21,3		borderline 9 - 13	elevated ≥ 14
Tricuspid annulus S' / Tricuspid annulus E' [cm/s]	3,8	5,8	9,5 - 18	> 10
4C longitudinal strain is greater in the basal septal segment -33% than in the basal lateral segment 29% STRAIN REVERSUS SIGN				

Figure 8. Paradoxical septal movements SEPTAL BOUNCE 15.07.2026.

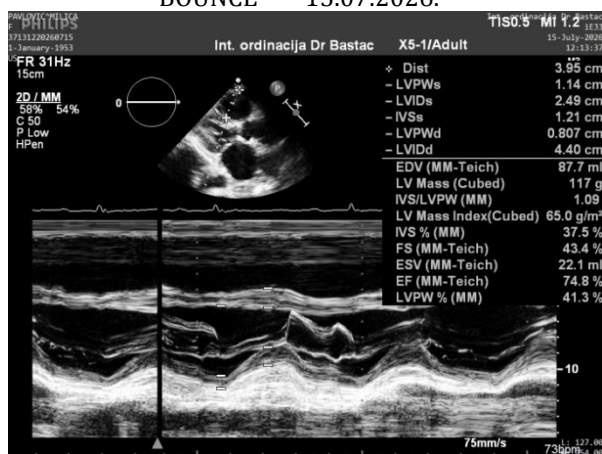


Figure 9. Right ventricular dilatation, larger than the left ventricle 15.07.2026.



Figure 10. sign strain reversus for constrictive pericarditis *4C longitudinal strain is greater in the basal septal segment -33% than in the basal lateral segment 29%*

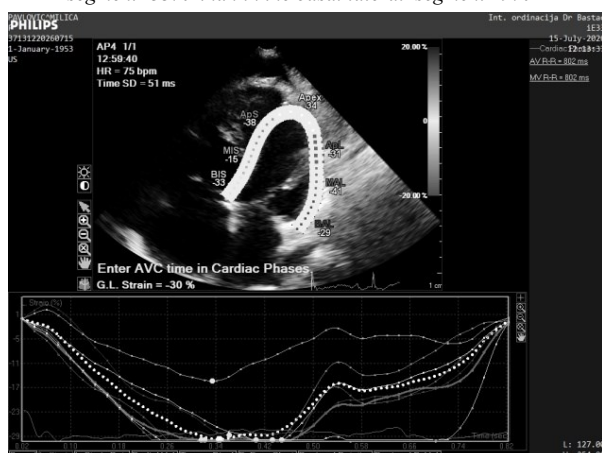
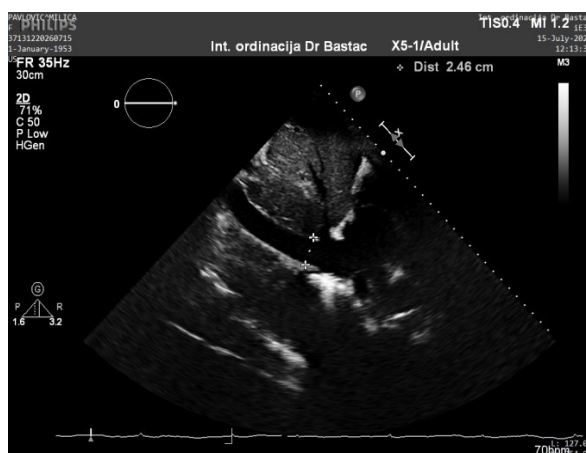


Figure 11.Dilatation of the inferior vena cava 25 mm without 50% inspiratory collapse . 15.07.2026.



After both evacuating pleural punctures, a biochemical **analysis of the pleural fluid was performed** 15.07.2026. clear transudate,

and at the same time abdominal ultrasound revealed splenomegaly, with a spleen diameter of about 17–18 cm [4,17,18].

Table. 2. biochemical analysis of the pleural fluid

Total protein IN FLUID	18	g/L	60 - 85
Albumin IN FLUID	13	g/l	33-55
Globulins	5	g/l	12-40

COURSE AND THERAPY

Initial urgent parenteral therapy in the form of a day hospital at the Int. practice of Dr Bastać

Due to the clinical picture of anasarca, ascites and bilateral hydrothorax, intensive decongestive therapy was introduced, including intravenous furosemide, spironolactone and an SGLT2 inhibitor, with further adjustment of

therapy according to the clinical condition [1,16].

Therapeutic course: Intensive decongestive therapy, supported by bilateral therapeutic evacuating pleural punctures (500 ml and 350 ml), led to a turnaround in treatment and complete loss of dyspnea on day 4 of treatment, with good regression of edema with high doses of furosemide, spironolactone and

empagliflozin, (with antihypertensives paused during treatment within the day hospital due to more severe episodes of hypotension), and was accompanied by significant clinical improvement.

Key dynamics of therapy and of the therapeutic response to intensive intravenous therapy (IV th) within the day hospital of the "DR BASTAĆ" Internal Medicine Practice (Table 3).

Table 3. Overview of the clinical course and the main intensive IV th within the day hospital of the "DR BASTAĆ" Practice

	13 and 14.07..	15 and 16.07.	17.07.	FURTHER ON
	Dose 4 amp (80 mg) IV Furosemide /100 ml 0,9%NaCl, II dose -6 amp (120 mg) Furosemide Methylprednisolone 40 mg + 40 mg dose 8amp (160mg) Furosemide +potassium +Mg	50 mg Dobutamine IN 200 ml 0,9% NaCl OVER 4 HOURS, Amp furosemide 3 x 80mg METHYLPREDNISOLONE 40mg + K+Mg PLEURAL PUNCTURE RIGHT 500 ML PUNCTURE LEFT 350 ML 500 ML	50 mg Dobutamine IN 200 ml Na Cl OVER 4 HOURS, Amp furosemide 3 x 80mg METHYLPREDNISOLONE 40 mg + K+ Mg THEN THE CONDITION IMPROVES and intensive diuresis is established	Switch to Per os th: Bisoprolol, Amiodarone, Dilacor, Forxiga, Entresto, Spironolactone, Furosemide forte 500 mg Euthyrox, Pronison, Allopurinol, Colchicine, Plavix, Nolpaza, K, Mg,,
DIURESIS according to body weight reduction	1 kg less	3 kg less	total 6 kg lower body weight	25 kg in 28 days
BP	130/70	100/65	100/65	105/60
PULSE	87/min	92/min	79/min	92/min
BREATHING	ORTHOPNEA	ORTHOPNEA	dyspnea	No dyspnea
EDEMA , LIVER, ASCITES , PULMO	Lower leg edema ++, ascites present, bilateral pleural effusion	Lower leg edema +, bilateral pleural effusion	Lower leg edema +, no ascites, bilateral pleural effusion	No edema, no pleural effusion, no ascites

Over the following days, clinical improvement was recorded with intensive diuresis and significant regression of symptoms (Table 3).

Table 4. 17.07.2026. Tissue Doppler and right ventricular function - Weakly expressed but still present sign - ANNULUS REVERSUS.

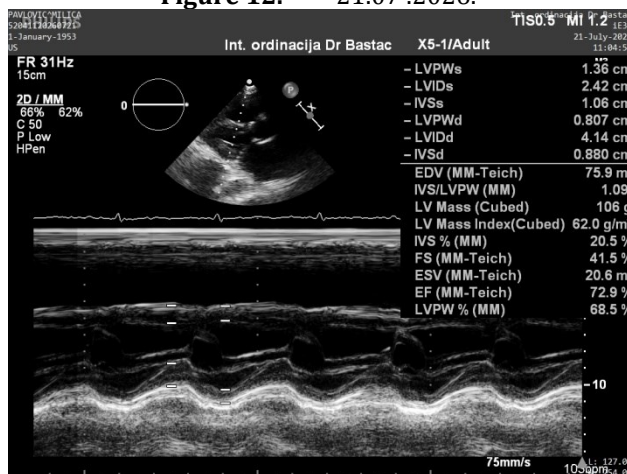
Septal e' (mitral annulus velocity Tissue dop PW) / S'systolic velocity [cm/s]	5,7		≥ 8
Lateral e' (mitral annulus velocity Tissue dop PW)/ S'systolic velocity [cm/s]	4,5		≥ 10
TAPSE	15	>17mm	
Right atrial (RA) area [cm2]	25	<18	

FOLLOW-UP **EXAMINATION**
21.07.2026.

Echocardiography still showed preserved left ventricular systolic function, EF 73% by Teicholz and approximately 60% by Simpson, with impaired relaxation and reduced compliance. The right ventricle was LESS dilated,

RVSP was 76 mmHg, with TR 3+, while the pericardium remained thickened, with a thickness of up to 9 mm. Diastolic dysfunction persists: E/e' was 21,8. On 21.07.2026, body weight was 68 kg. The patient reports less fatigue and reduced leg edema [1,13].

Figure 12. 21.07.2026.



At the examination on 21.07.2026 (Figure 12), preserved left ventricular systolic function was again described, with a finding interpreted as constrictive pericarditis, with marked tricuspid regurgitation, pulmonary hypertension and right ventricular failure.

The combination of clinical systemic congestion, pericardial thickening and paradoxical septal motion justifies the suspicion of constrictive physiology, but the morphological change of the pericardium alone is not sufficient for a definitive diagnosis [1,2,13].

Table 5. Serial echocardiographic follow-up (14.07–11.08.2026.)

Parameter	14.07.	17.07.	21.07.	25.07.	11.08.
LV EDD (mm)	40 b mode	49	41	41/4c40	44
LV ESD (mm)	27	23	24	22	29
EF (%)	69	76	73	78	63
LV mass (g)	99	118	106	90	140
LVMi (g/m ²)	57	67	62	54	92
E wave (m/s)	1,1	1,2	0,75	1,14	0,80
E/A ratio	1,3	2,0	1,4	1,8	1,7
e' medial septal			3,0	8,7	
e' lateral					
E/e'	21,3	21,8	21,8	21,9	15,0
GLS 4c	-30			-21	
TAPSE (mm)	-	12	9		16
RV basal diameter (mm)	39	45	46	45	45
RA area (cm ²)	-	25	24		21
TR Vmax (m/s)	3,8	2,8	3,7	4,0	3,3
TR Gmax (mmHg)	57	32	56	64	44
RVSP/SPDK (mmHg)	77	52	76	74	44
IVC exp./insp. (mm)	-	26/16	23/17	20/10	17/7
Liver CC (cm)	17,8	15	17	17	16
Mitral regurgitation	2+ mild	2+ mild	2+ mild	2+mild	2-3+ moderate
Tricuspid regurgitation	3+ moderate -	3+ moderate	3-4+	3+	at least moderate
Ascites	+	minimal	none	None	None

Chest CT scan finding (Dr Stanislav Tadić): hydrothoraces, pulmonary congestion, no pulmonary infiltrates and marked hepatomegaly and, splenomegaly, small ascites [1,13].

Serial echocardiographic evaluation initially showed an increase in pulmonary hypertension with improvement of left ventricular function and decongestion on

21.07.2026 (RVSP 76 mmHg, TAPSE 9 mm, TR Gmax 56 mmHg), and with excellent clinical improvement (right atrial dilatation 25 mm and 44 ventricles, significant reduction of hepatomegaly and of signs of systemic venous congestion..

BLOOD LABORATORY ANALYSES

Findings brought from 13.07.2026. - Marked leukocytosis was recorded. The leukocyte count

was $55,0 \times 10^9/L$, with 84% granulocytes, while on 21.07.2026. $58,8 \times 10^9/L$ leukocytes were

recorded, with 75,2% granulocytes.

Table 6. Dynamics of laboratory parameters (13–21.07.2026.)

Parameter	13.07.2026	14.07.2026	21.07.2026
Erythrocytes ($\times 10^{12}/L$)	-	5,0	5,4
Hemoglobin (g/L)	143	152	159
Leukocytes ($\times 10^9/L$)	55	Error	58,8
Neutrophils / Granulocytes (%)	84	error	75,2
Lymphocytes (%)	10	error	16,4
Platelets ($\times 10^9/L$)	293	163	166
CRP (mg/L)	-	17	41,9
Glucose (mmol/L)	5,1	4,76	11,4
Urea (mmol/L)	8,1	14,3	10,8
Creatinine ($\mu\text{mol}/L$)	97	122	69
GFR (mL/min/1,73 m ²)	48	37	72,3
Uric acid ($\mu\text{mol}/L$)	-	774	536
AST (U/L)	36	31	30
Albumin (g/L)	41	37	40
Total protein (g/L)	-	59	66
LDH (U/L)	962	-	-
TSH (mIU/L)	36,84	-	-
FT4 (pmol/L)	10,77	-	-
FT3 (pmol/L)	3,39	-	-
NT-proBNP (pg/mL)	-	1175	-

The immunological work-up performed on 23.07.2026 did not show laboratory indicators of a systemic autoimmune disease:

ANA 0,8 COI, anti-CCP <3,5 U/mL and dsDNA 12,1 U/mL.

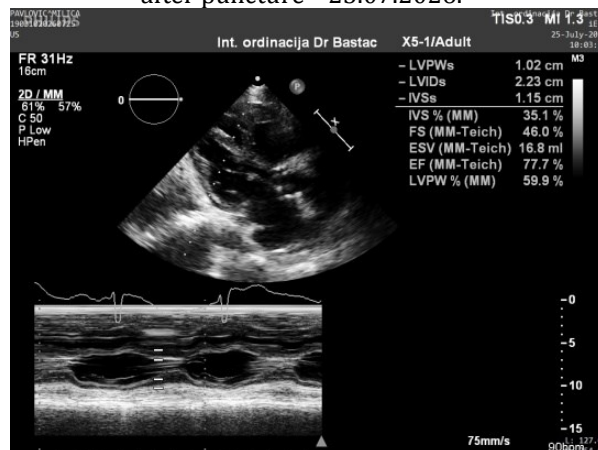
Table 7. Immunological work-up.

	<u>obtained val.</u>	<u>units</u>	<u>reference values</u>
s-Antibodies to cyclic citrullinated peptides (anti-CCP)	<3,5	U/ml	<5,0
ANA antinuclear ab	0,8	COI	Negative : <1,0 Borderline positive : 1,0-1,2 Positive : >1,2
Ds DNA ab	12,1	U/ml	<20,0

Figure 13. Major reduction of the pleural effusion after puncture 25.07.2026.



Figure 14. Major reduction of the pleural effusion after puncture 25.07.2026.



During follow-up until 11.08.2026, a significant reduction in right ventricular systolic pressure (RVSP or SPDK 44 mmHg) and an improvement in right ventricular function (TAPSE 16 mm) were recorded, with a reduction in the dimensions of the liver and the inferior vena cava, i.e. excellent regression of congestion. Left ventricular diastolic function is improving, E/e' (from 21 to 15), and there is a mild ANNULUS REVERSUS SIGN, moderate mitral regurgitation (2-3+) and residual pulmonary hypertension, indicating persistent diastolic dysfunction and chronic right heart overload [1,13].

At the follow-up on 11.08.2026, the clinical condition was significantly better, with almost complete resolution of peripheral edema and absence of bilateral pleural effusion on echocardiographic examination. At the same time, an improvement in right ventricular function parameters and a reduction in the estimated right ventricular pressure from 76 to 44 mmHg were recorded.

According to documented measurements, body weight decreased from 77 kg to 52 kg. This weight loss should be interpreted primarily in the context of marked

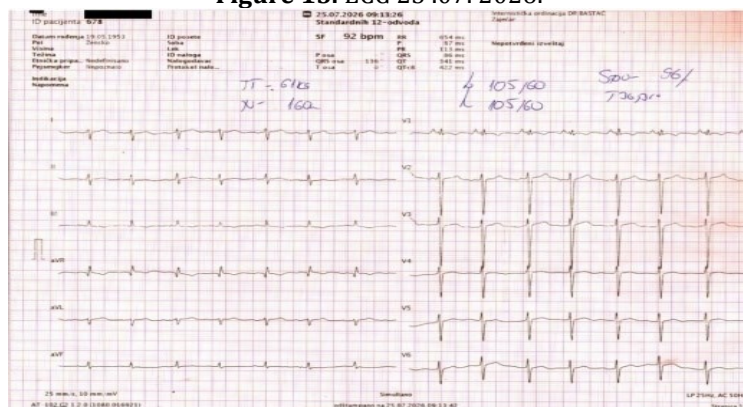
previous fluid retention and intensive decongestive therapy. Pleural effusions were no longer recorded [1,13].

HEMATOLOGIST - However, the hematological problem persisted. On 13.08.2026, a peripheral blood smear showed the presence of all elements of the granulocytic lineage, with a predominance of myelocytes and metamyelocytes and blasts up to 2%. Erythrocytes and platelets were morphologically described as normal, while all elements of the granulocytic lineage were represented, with a predominance of myelocytes and metamyelocytes. Blasts accounted for up to 2%. The finding was assessed as compatible with a myeloproliferative process, with a recommendation for referral to the Clinic of Hematology for further diagnostics.

Considering current classifications of myeloid neoplasms, this finding does not represent a definitive hematological diagnosis, but rather a basis for further morphological, molecular and clinical evaluation [17,18].

The finding was assessed as compatible with a myeloproliferative process, with a recommendation for further diagnostics.

Figure 15. ECG 25.07.2026.



Overview of the oral home therapy given at discharge from the day hospital of the Dr Bastać Practice (Table 8)

Table 8. oral home therapy .11.08.2026.

drug	morning	noon	evening	comment
Bisoprolol tab 2,5 mg	1+1/2			after meals
Amiodarone tab	0		0	
Digoxin tab	1/2			
Dapagliflozin 10 MG	1			after meals
spironolactone 100mg,	1/2			after meals
furosemide forte 500 mg	1			
Euthrox 25mcg,	1		1	before meals
Clopidogrel 75 MG	1			after meals
Allopurinol 100 mg	2		2	

Colchicine 0,5 mg	1		1	
Pronizon 20 mg	1			
Pantoprazole 40 mg	1		1	after meals
potassium powder 1 g		1		
Insulin 12 U				in the evening
magnesium 400 mg	1			

Follow-up 03.10.2026. - 80 days after the onset of the disease

At our invitation, the patient came in, stating that she feels very well, and denies complaints of significance for the cardiovascular system. In the period between the last two follow-ups, she was hospitalized for 2 weeks at the Zaječar General Hospital due to progression of leukocytosis (CBC during hospitalization Leu 147×10^9), but without a final diagnosis and with an indication for further evaluation by a hematologist, which the patient again refuses.

Clinical examination - slight deterioration compared to 11 August, when she was fully cardiac compensated.

Cor: HR 75/min, heart sounds clear, rhythm irregular, frequent SVES, systolic regurgitant murmur 3/6, p.max over Erb's point. Pulmo: breath sounds normal, diminished at the right base, early and late inspiratory crackles, basal crepitations, Expiration not prolonged. Abdomen: soft and non-tender on palpation. Liver of soft consistency, mobile with respiration, palpable 10 cm below the RCM, spleen of firm consistency, palpable 19 cm below the LCM. Extremities: doughy edema of the lower legs ++, peripheral arterial pulses are symmetrically palpable.

CRP 6.4, (normal up to 5 mg/l), mildly elevated

CBC: Hgb106, Er 3.57, Le 65.0, Creatinine 170, GFR 25.6, Uric acid 508, K ion 5.9, HbA1c 5.6, BNP 395.

Conclusion: Significant improvement is maintained,

reduced pulmonary hypertension, improved diastolic function, IAS bulging toward the left atrium. TR 3+ is recorded, the septum makes paradoxical movements. The pericardium is now of reduced thickness on three-dimensional echocardiography 3D/4D, 3,6 to 5,8 mm, unevenly thickened (also compared to 8 to 9 mm at the onset of the disease) and separated, without pericardial effusion.

Mild to moderate mitral regurgitation MR 2+ is recorded. Diastolic function parameters: impaired relaxation and reduced compliance. Left ventricle of normal endocavitary dimensions WITHOUT myocardial hypertrophy and with preserved global systolic function. EF=78%.

Pleural ultrasonography: minimal bilateral pleural effusions. On the left, pleural effusion in the mid-scapular line basally above the liver 2cm, in the mid-axillary 5mm, in the posterior axillary 11mm, in the anterior axillary line 4mm. On the right, effusion in the mid-scapular line 2,4 cm, posterior axillary line 14 mm, mid-axillary 15 mm

Lung ultrasonography: Basally about 8 B-lines. right and left, B-lines 7, from the scapular angle A-lines - no congestion.

Since the patient discontinued NSAIDs, corticotherapy and colchicine on her own initiative, a slight improvement occurred, so it was explained to the patient that it is very important to take this therapy for up to 6 months.

Figure 16. Minimal pleural effusion 03.10.2026.

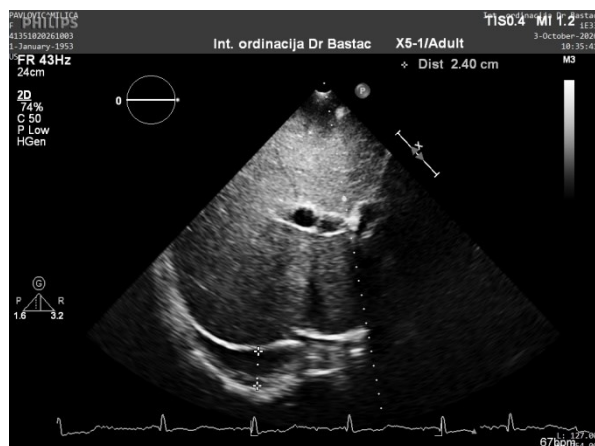


Figure 17. No respiratory variations over the mitral valve 03.10.2026

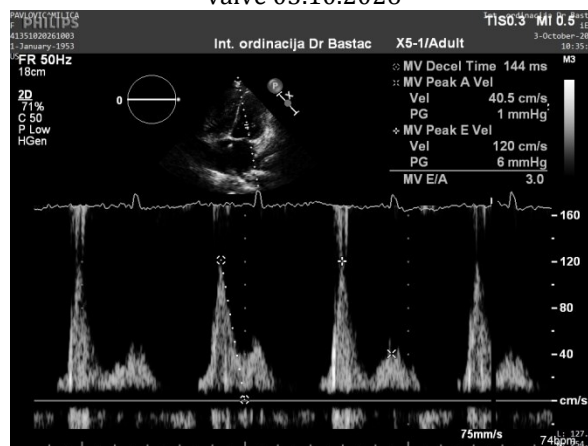


Figure 18. 03.10.2026
TD of the lateral annulus

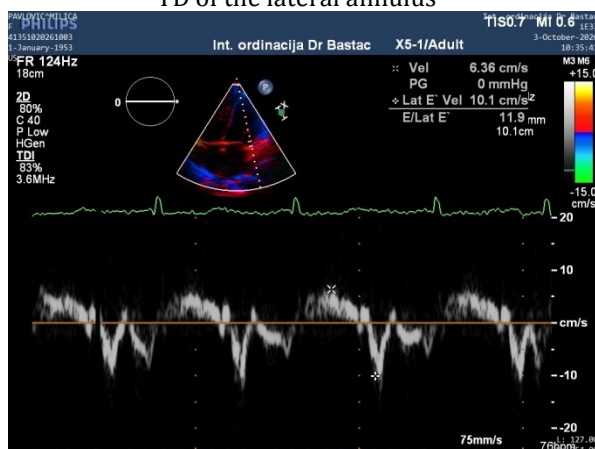


Figure 19. 03.10.2026
TD of the medial annulus Loss of annulus reversus

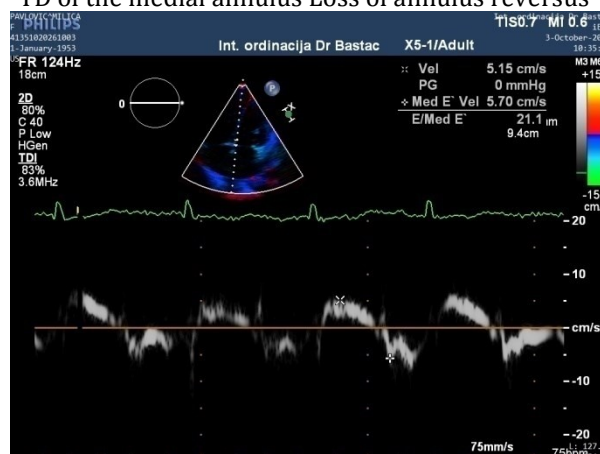
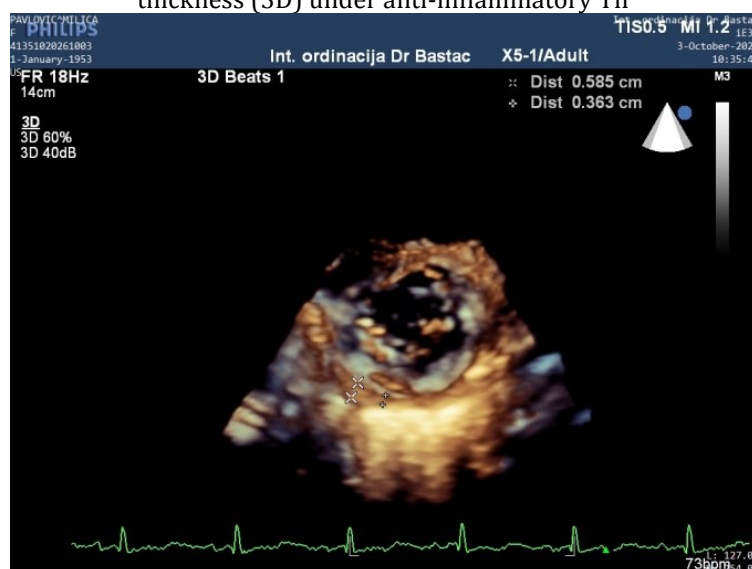


Figure 20. 03.10.2026. Reduction of pericardial thickness (3D) under anti-inflammatory Th



DISCUSSION

The presented case is characterized by a marked discrepancy between the preserved left ventricular systolic function and the severity of the clinical congestion syndrome. During the most severe phase of the disease, LVEF was preserved, while at the same time there were left ventricular diastolic dysfunction, significant tricuspid regurgitation, pulmonary hypertension, right ventricular dysfunction, right atrial dilatation and signs of elevated filling pressures.

The key diagnostic problem is distinguishing constrictive pericarditis from other conditions that can produce a similar hemodynamic phenotype, primarily HFpEF and

restrictive cardiomyopathy. Constrictive pericarditis can lead to predominant systemic venous congestion with relatively preserved left ventricular systolic function, which is why the clinical picture may be very similar to other forms of right-sided heart failure [1,14].

In this patient, several echocardiographic findings supported the possibility of constrictive pathophysiology. Particularly notable were paradoxical movements of the interventricular septum (septal shift and bounce), pericardial thickening, diastolic filling abnormalities, dilatation of the right heart chambers and significant tricuspid regurgitation. At the same time, left ventricular systolic function remained preserved, with EF

values of approximately 60–76% and normal global longitudinal strain [1,2,13].

The Mayo Clinic echocardiographic criteria include a combination of respiration-related septal shift, medial e' and changes in hepatic venous flow. Combining several criteria increases the specificity for constrictive physiology. [13]

In the presented case, however, it was not possible to carry out a complete hemodynamic protocol for definitive confirmation of constriction. The patient declined further hospitalization, and invasive pressure assessment was not performed [1,15]. Therefore, it is more appropriate to designate this finding as **echocardiographically suspected constrictive pericardial pathophysiology physiology**, rather than as definitively confirmed constrictive pericarditis [1,13].

An important element of the case is the marked leukocytosis. The leukocyte count was $55,0 \times 10^9/L$ as early as 13.07.2026, and $58,8 \times 10^9/L$ on 21.07.2026, with marked granulocytosis. In addition, splenomegaly was present. A later peripheral blood examination showed the presence of all elements of the granulocytic lineage, with a predominance of myelocytes and metamyelocytes and blasts up to 2%, and the finding was designated as compatible with a myeloproliferative process [17-21].

This finding is significant because it shows that the leukocytosis was not merely a transient laboratory change during the acute worsening of heart failure. However, based on the submitted documentation, it is not possible to definitively determine the type of hematological disease. Current classifications of myeloid neoplasms require the integration of clinical, morphological and molecular data [17,18].

It is precisely in this context that the question arises of a possible association of the hematological process with the changes in the pericardium and pleura. In the differential diagnosis, infiltrative processes, extramedullary hematopoiesis and other inflammatory or serositis mechanisms may be considered [22-24].

Extramedullary hematopoiesis in the pericardium is a very rare phenomenon, but it has been described in patients with myeloproliferative neoplasms. Haroun et al.

described extramedullary hematopoiesis involving the pericardium in a patient with polycythemia vera, with the development of cardiac tamponade [22]. Mahadevan et al. presented a case of pericardial extramedullary hematopoiesis with cardiac tamponade and reviewed previously published cases [23]. In 2025, Hayar et al. described a rare case of extramedullary hematopoiesis with pericardial involvement in a patient with a myeloproliferative disease [24].

These data provide a possible pathophysiological framework for considering a hematological-pericardial association in our case, but **they do not constitute evidence that the pericardial thickening in the presented patient is a consequence of extramedullary hematopoiesis**. Such a conclusion would require additional radiological, morphological and/or histological evidence [22-24].

Particularly significant is the therapeutic response to decongestive therapy, heart failure therapy and anti-inflammatory therapy. Intensive diuresis and evacuation of the pleural effusions led to a marked reduction in body weight and resolution of peripheral edema and pleural effusions. At the same time, a reduction in RVSP from 76 to 44 mmHg and an improvement in TAPSE from 9 to 16 mm were recorded [1,16]. Such a response confirms the importance of volume overload in the clinical picture, but by itself neither confirms nor excludes constrictive pericarditis [1,13]. Also, according to the recommendations for probable constrictive pericarditis from the European guideline for myopericardial diseases and the algorithm, triple anti-inflammatory therapy should be introduced: a corticosteroid, a nonsteroidal anti-inflammatory drug and colchicine for up to 6 months. If transient constrictive pericarditis is established, with clear clinical and imaging improvement and disappearance of the constrictive pathophysiology, then pericardiectomy is not required as a definitive treatment measure. Transient constriction is a form of constrictive pericarditis (CP) with active inflammation present, which may resolve after anti-inflammatory therapy or even regress spontaneously. It develops during pericarditis with or without pericardial effusion (effusive-constrictive pericarditis). In more than 50% of cases, constriction resolves after anti-inflammatory therapy [6,7,8]. In patients with

newly diagnosed constrictive pericarditis, a trial of anti-inflammatory therapy lasting 3 to 6 months is recommended before referral for pericardiectomy [9,10]. The presence of active pericardial inflammation was confirmed by inflammatory biomarkers (C-reactive protein, which was initially 42 mg/L and then fell to 7 mg/L), as well as by cardiovascular magnetic resonance (CMR), which showed pericardial edema and late gadolinium enhancement (LGE) [10,11]. In HFpEF with marked congestion and preserved ejection fraction, the assessment of filling pressures, right ventricular function and the pulmonary circulation is an important part of the diagnostic process [16].

For definitive confirmation of constrictive pericarditis in such a complex presentation, additional modalities would be of importance, including an ultrafast MDCT scanner or a photon-counting scanner and cardiovascular magnetic resonance, and, if necessary, right heart catheterization for direct hemodynamic assessment [1,2,15].

Precisely such further work-up was recommended to the patient, but it had not been carried out by the last follow-up, because she feels well and refuses hospitalization at a higher-level institution in cardiology or hematology.

CONCLUSION and EPILOGUE OF THE CASE:

In patients with marked systemic venous congestion, hepatosplenomegaly, ascites and anasarca and preserved left ventricular systolic function, but with signs of severe HFpEF with reduced left ventricular stroke volume and pulmonary congestion with pleural effusions, constrictive pericarditis should be considered in the differential diagnosis [1,13]. Constrictive

pericarditis was suggested by echocardiography: pericardial thickening, parallel movement of the pericardial layers and paradoxical septal movements and other Doppler signs of CP [1,2,13]. However, echocardiographic findings alone are not sufficient for definitive confirmation of the diagnosis without hemodynamic confirmation. The etiological diagnosis could be related to the existing chronic myeloproliferative process [17,18,22-24].

Despite the initial therapeutic failure in this patient with decompensated heart failure, with huge doses of diuretics, inotropic support, dobutamine AND corticosteroids, after bilateral pleural punctures, after 4 days of treatment a gradual recovery occurs. The peripheral edema and both pleural effusions disappear, as detected by lung ultrasound. She is later switched to per os th, and there is a reduction in body weight from 77 kg to 52 kg - a total of 25 kg over a period of 28 days. A reduction of RVSP to 44 mmHg also occurs. and an improvement in other echocardiographic dimensions and parameters [1,16]. At the same time, progressive leukocytosis is recorded, up to values above $90 \times 10^9/L$, while the examination of the peripheral blood smear by a hematologist showed a predominance of myelocytes and metamyelocytes with less than 2% blasts, which was compatible with a CHRONIC myeloproliferative process. The patient refuses further treatment at the hematology department of a higher-level institution and follow-up visits, and in a telephone conversation claims that she feels well. **After almost 2 months, she comes with moderate deterioration because she discontinued the anti-inflammatory therapy of her own accord.**

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