Azərbaycan Kardiologiya Cəmiyyəti

HFA Heart Failure Association

HFrEF with comorbidities

(case reports)

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Dr. Aysel İSLAMLI

Azərbaycan Kardiologiya Cəmiyyəti

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- ✔ I have no conflict of interest regarding this presentation.

what is comorbidities ?

🎯 Side diseases that progress with the main disease and cause clinical worsening of the main disease are called comorbidities

🎯 Comorbidities may differently affect treatment response and cause-specific outcomes in heart failure (HF) reduced (HFrEF) ejection fraction

DM + HT+ CAD + HFrEF + CKD

- Multimorbidity (two or more comorbidities) is common in patients with heart failure.
- Multimorbidity complicates - guideline-directed pharmacological treatment and worsens prognosis.

DM + HF

> 60 age + T2DM + CVD

shortness the life expectancy by and average of 12 years

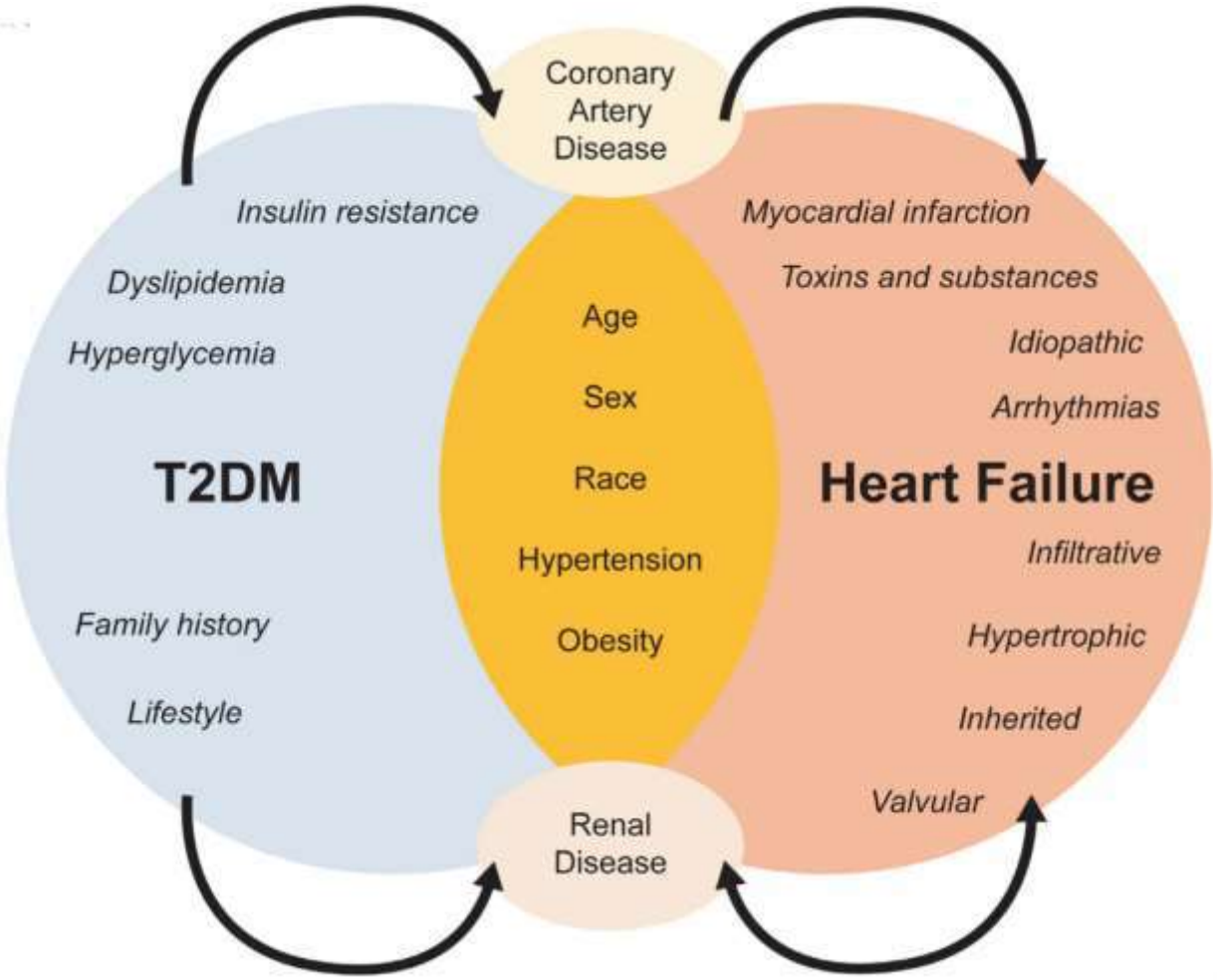

Figure 1. Risk factors for T2DM and congestive heart failure. T2DM = type 2 diabetes mellitus.

ed. www.ajconline.org https://doi.org/ 10.1016/j.amjcard.2019.10.024

assess key patient characteristics

- current lifestyle
- comorbities i.e. ASCVD, CKD, HF
- clinical characterictics i.e. age, HbA1, weight
- ıssues such as motivation and depression
- cultural and socio- economic context

case - 1

- 40 years old man, reffered for evalution of obesity, T2DM and dyspnea on exertion
- His graduated weight 75 kg, BMI 23.2 kg/m2
- Active during college, but then took sedantery job, began eating excessively, was less active and gained 18 kg over 15 years.
- Type2 DM diagnosed at age 36, when weight 104kg, BMI 33.3 kg/m2
- Hypertensive since in college, but untreated untill his PCI
- Multipl self-prescribed lifestyle attempts a weight loss have failed
- Family history + for T2DM, but only late onset ASCVD

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LabAnalyses

- ⬆ HbA1C 10.4%
- ⬆ serum creatinin 1.35 (eGFR 56.9 mL/min/1.73cm2)
- ➡ Hg/Ht 38.2/45
- ➡ Potassium 4.7
- ⬆ LDL

LDL-xolesterol	**208.9**	⬆	mg/dL	H(<100)
Triqliseridlər (TGL)	**167.6**	⬆	mg/dL	H(<150)

45 Yıl 11 Ay 28 Gün, Erkek

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Rapor Onay Tarihi : 05.08.2024 17:04

Hekim :
Kurum : İnci Laboratuvarları
Ruhsat Numarası : 377-MRK
Örnek Türü : Serum

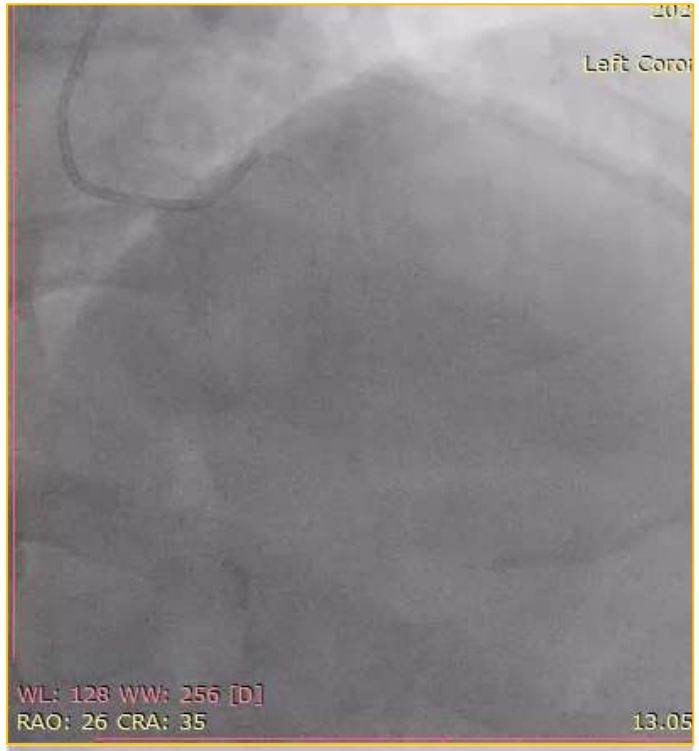
BİYOKİMYA

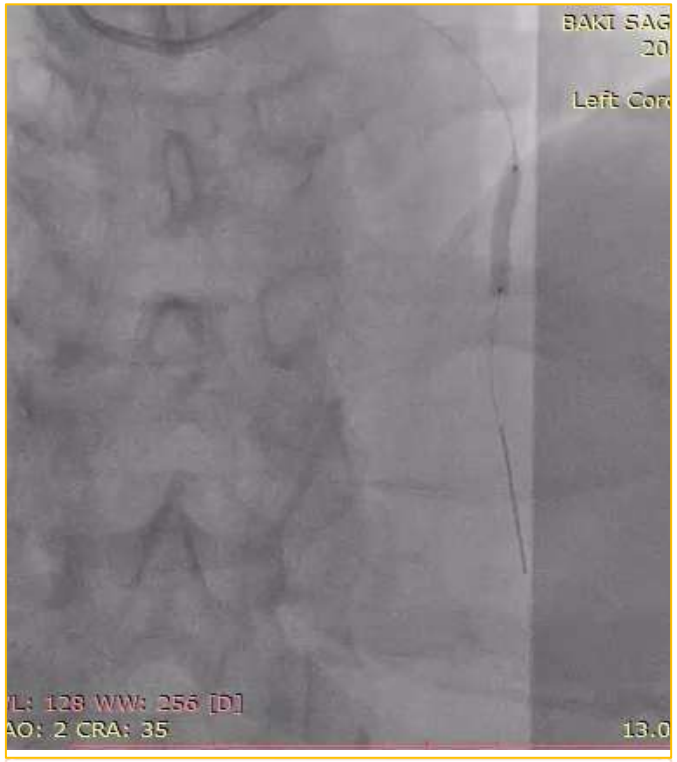
Test Adı	Sonuç	Birim	Referans Değer
Lipoprotein (a) * *	↑121.9	mg/dL	0 - 30

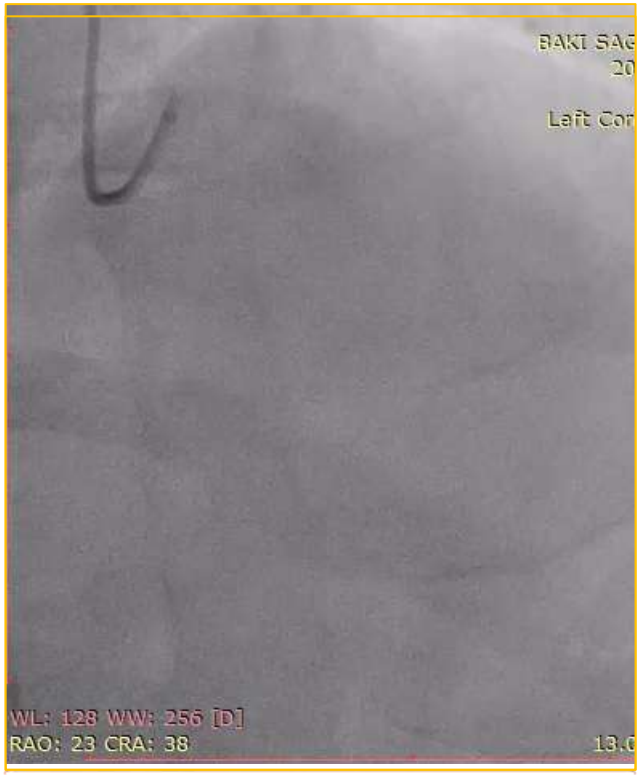
➡ ⬆ lipoprotein (a) 121.9

CAG ➡ PCI ➡ LAD DES

Developed left arm pain while bowling 2 years ago and had a PCI + drug eluting stent.







postPCI (EF %55, NT-pro BNP 230ng/dL)

- Clopidogrel 75 mg 1*1
- ASA 1*1
- Bisoprolol 2.5 mg 1*1
- Rosuvastatin 40 mg 1*1
- Perindopril + Amlodipin + İndapamid 5/1.25/5 mg 1*1
- Trimetazidin OD 80 mg 1*1

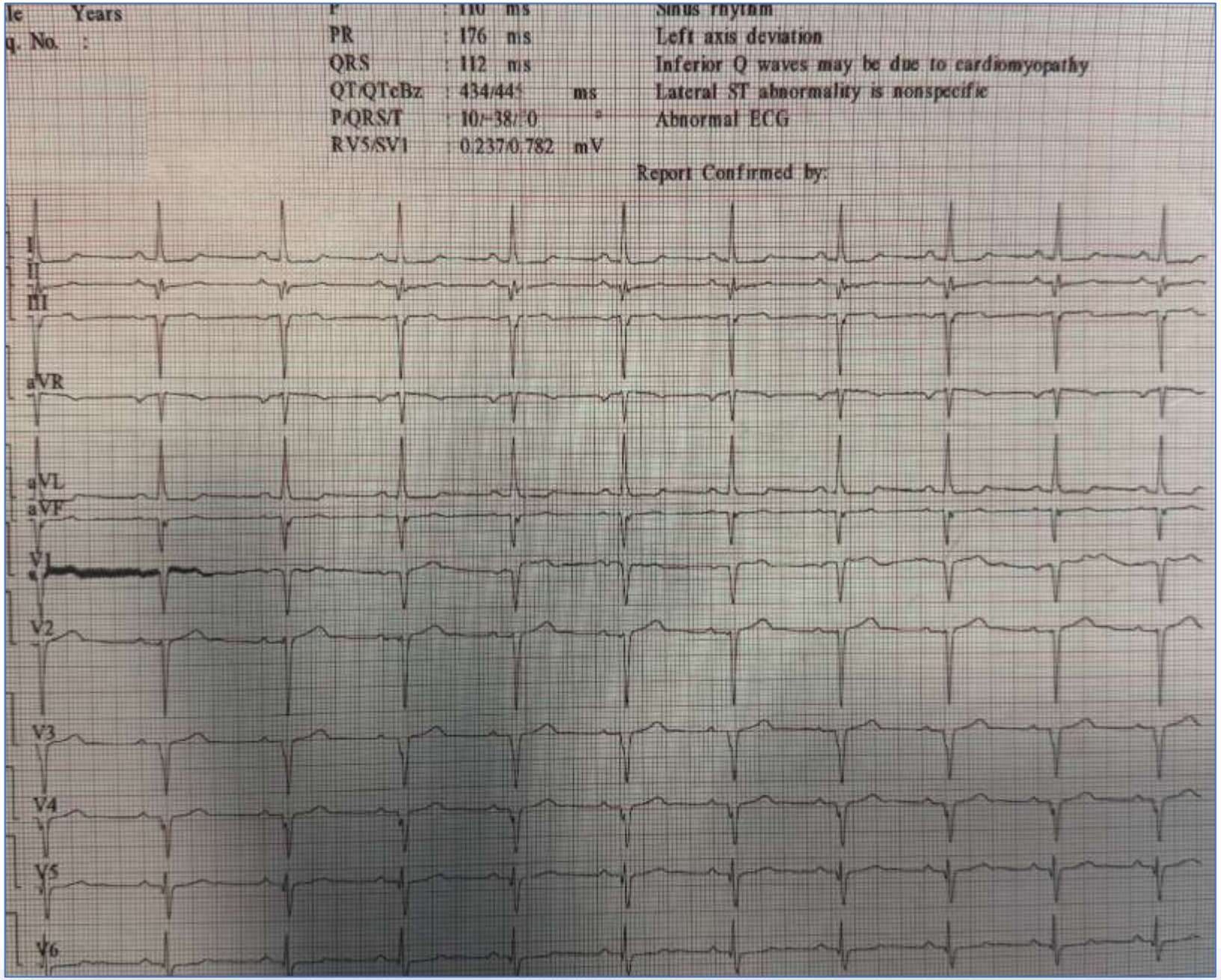
💧 Over the past 2 year he has noticed some dyspnea while walking the dog

- 🎯 During follow-up, we saw that creatinine levels increased and ejection fraction decreased.
- 🎯 He said that there was a decrease in urine output, decreased exercise capacity, increased shortness of breath, weakness
- 🎯 The patient was hospitalized, intravenous diuretics were started due to volume overload (pretibial edema, lung crackles), hourly urine output was monitored.
- 🎯 Echocardiographic examination showed that EF was suppressed. AP 190/110 mmHg, HR 98/min (wet, warm clinic)

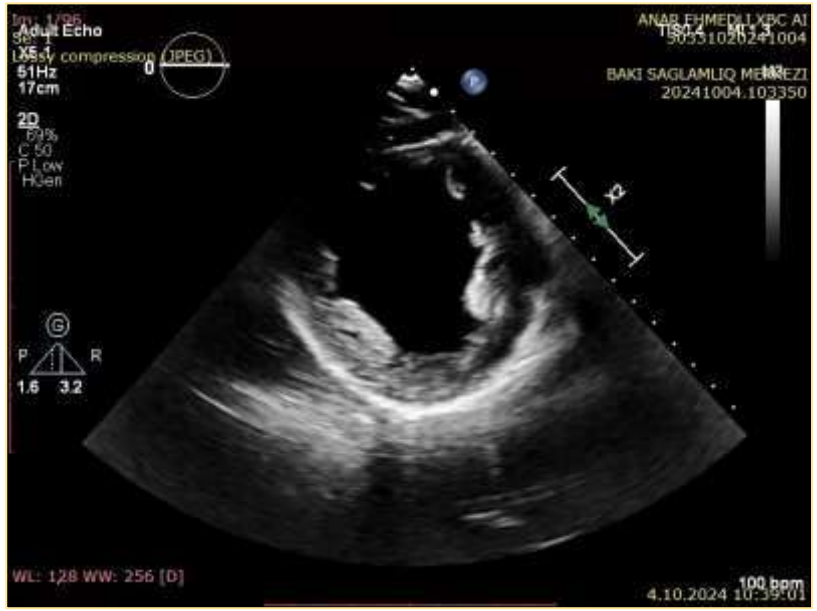
LabAnalyses

- ⬆️ HbA1C 7.2%
- ⬆️ serum creatinin 3.2 (eGFR 24 mL/min/1.73cm2)
- ➡️ Hg/Ht 31/39
- ➡️ Potassium 5.4
- ➡️ NT-pro BNP 8890 ng/dL
- ➡️ lactat 2.1
- ➡️ pH 7.33

ECG


le Years
q. No. :
P : 110 ms
PR : 176 ms
QRS : 112 ms
QT/QTcBz : 434/445 ms
P/QRS/T : 10/-38/ 0 °
RV5/SV1 : 0.237/0.782 mV
Sinus rhythm
Left axis deviation
Inferior Q waves may be due to cardiomyopathy
Lateral ST abnormality is nonspecific
Abnormal ECG
Report Confirmed by:
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

preECHO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Lossy compression (JPEG)
51Hz
17cm
2D
69%
C 50
P Low
HGen
1.6 3.2
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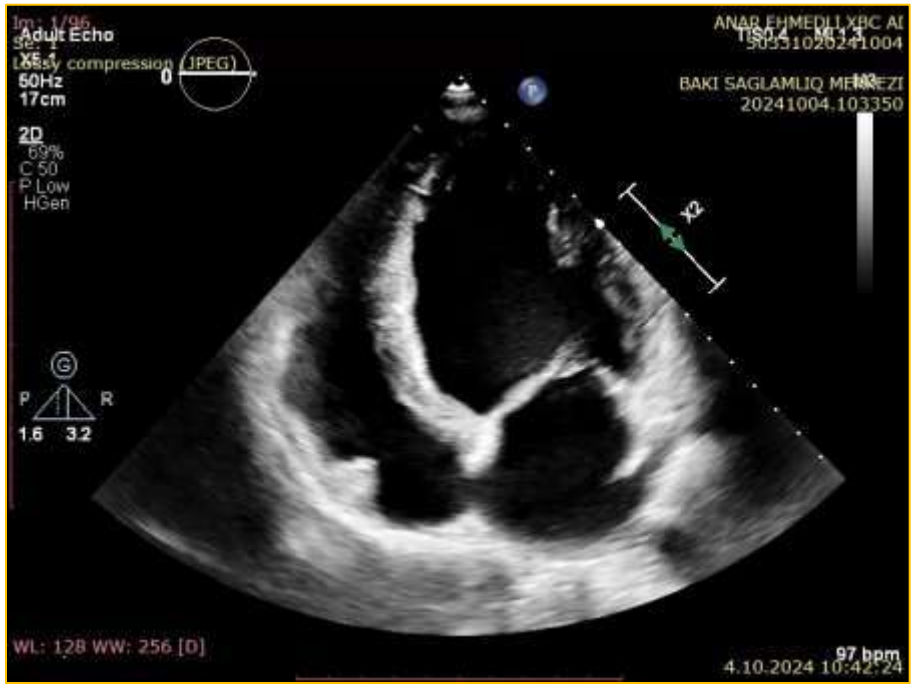
WL: 128 WW: 256 [D]
100 bpm
4.10.2024 10:39:01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1.6 3.2
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97 bpm
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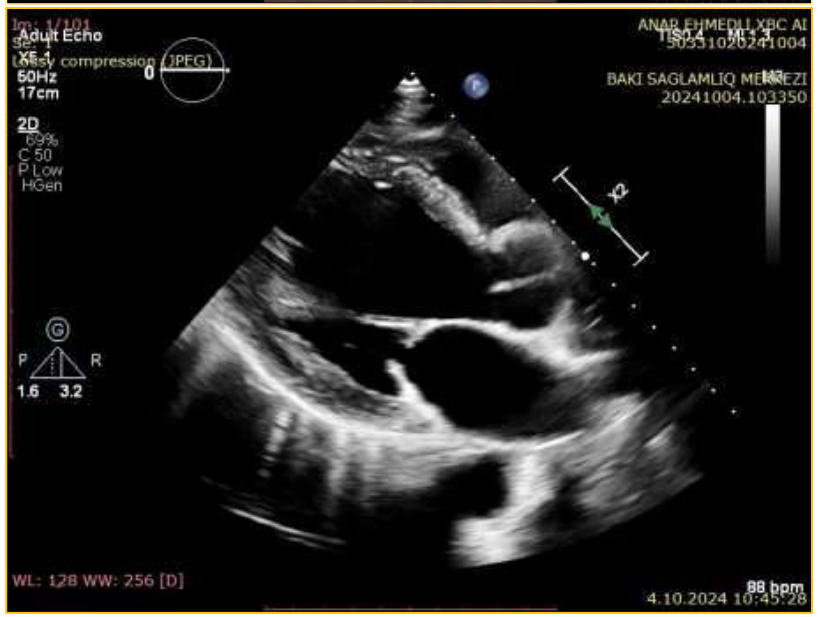
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Lossy compression (JPEG)
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69%
C 50
P Low
HGen
1.6 3.2
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88 bpm
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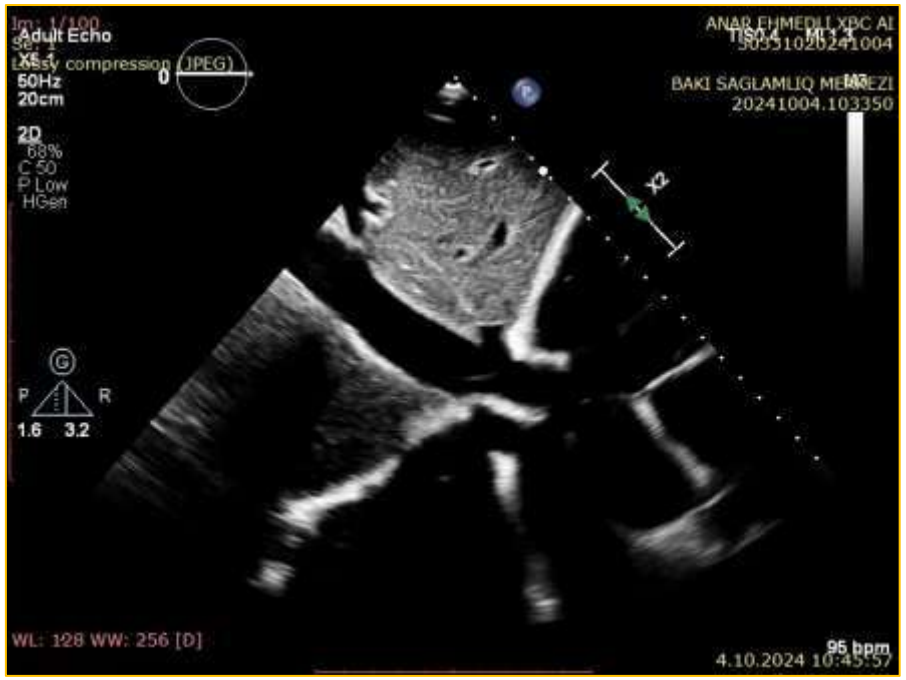
Adult Echo
Lossy compression (JPEG)
50Hz
20cm
2D
68%
C 50
P Low
HGen
1.6 3.2
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WL: 128 WW: 256 [D]
95 bpm
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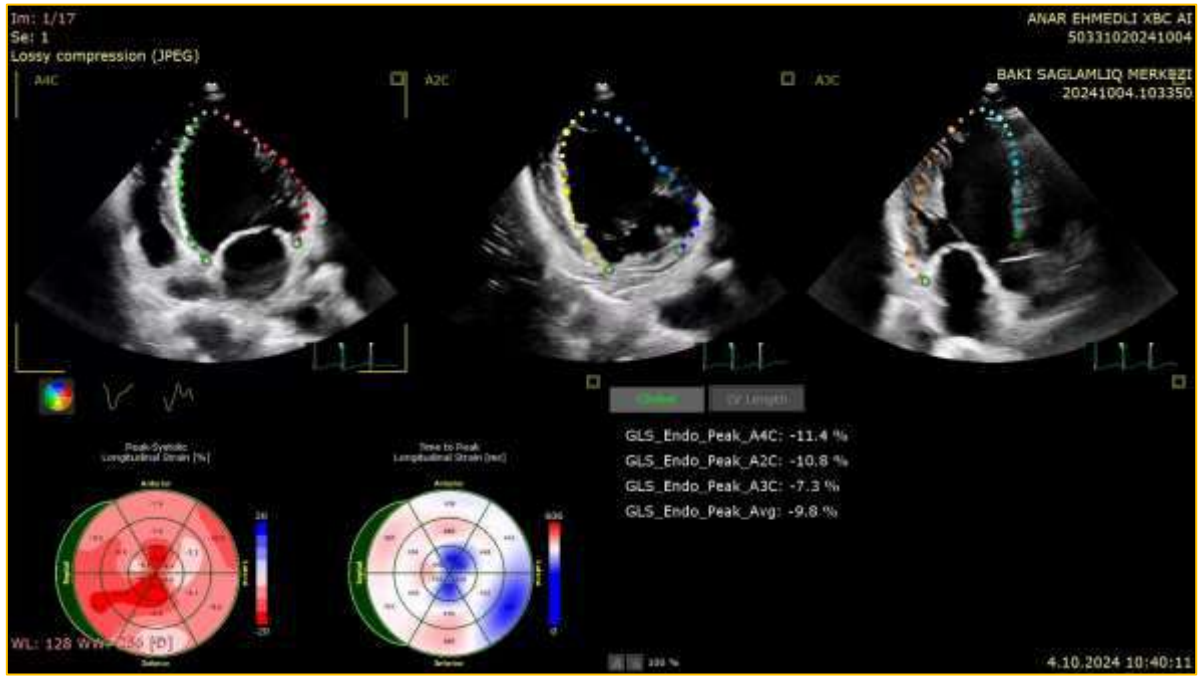
strain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Se: 1
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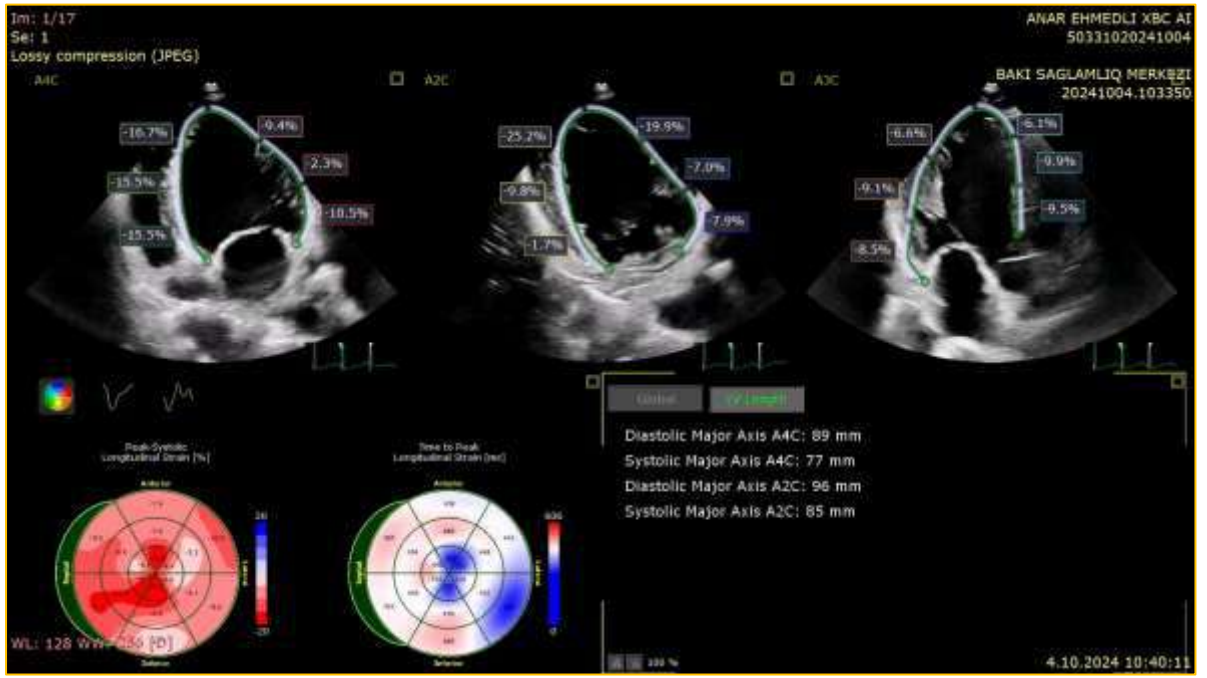
ANAR EHMEDLI XBC AI
50331020241004
BAKI SAGLAMLIQ MERKEZI
20241004.103350
A4C
A2C
A3C
GLS_Endo_Peak_A4C: -11.4 %
GLS_Endo_Peak_A2C: -10.8 %
GLS_Endo_Peak_A3C: -7.3 %
GLS_Endo_Peak_Avg: -9.8 %
4.10.2024 10:40:11


Im: 1/17
Se: 1
Lossy compression (JPEG)
ANAR EHMEDLI XBC AI
50331020241004
BAKI SAGLAMLIQ MERKEZI
20241004.103350
A4C
A2C
A3C
Diastolic Major Axis A4C: 89 mm
Systolic Major Axis A4C: 77 mm
Diastolic Major Axis A2C: 96 mm
Systolic Major Axis A2C: 85 mm
4.10.2024 10:40:11

discharge treatment

- After IV diuretic treatment, 9300 cc urine output was obtained, electrolytes were replaced as necessary, creatinine was 2.7 mg/dl (IV nitroglycerin was also ordered for the first 2 days and there was no need for inothropic)
- At discharge, oral diuretic and ARNI were added to the treatment he received
  - ASA 100 mg1*1
  - Bisoprolol 2.5 mg 1*1
  - Rosuvastatin 40 mg 1*1 → Atorvastatin 40 mg 1*1 (LDL 62 mg/dl)
  - Sacubitril/Valsartan 49/51 mg 2*1 B.I.D
  - Furosemid 40 mg 2*1 B.I.D

At the control visit, the patient was found to be euvolemic, VCI was 16 mm and collapsed.

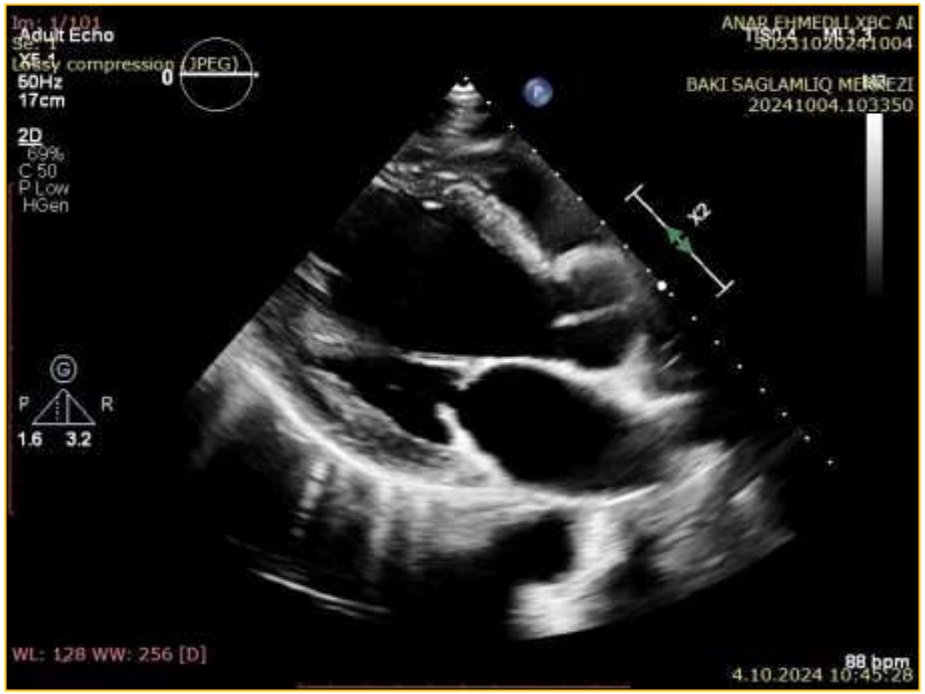
LVEF was evaluated as 38% – global hypokinetic. Creatinine was 2.5 and Potassium was 5.3. NT –pro bNP 2300. Nephrology was consulted, Antipotassium sachet was added to the treatment. No acidosis was observed in the arterial blood gas, lactate values were 1.2. Furosemide was suspended, ARNI was continued.

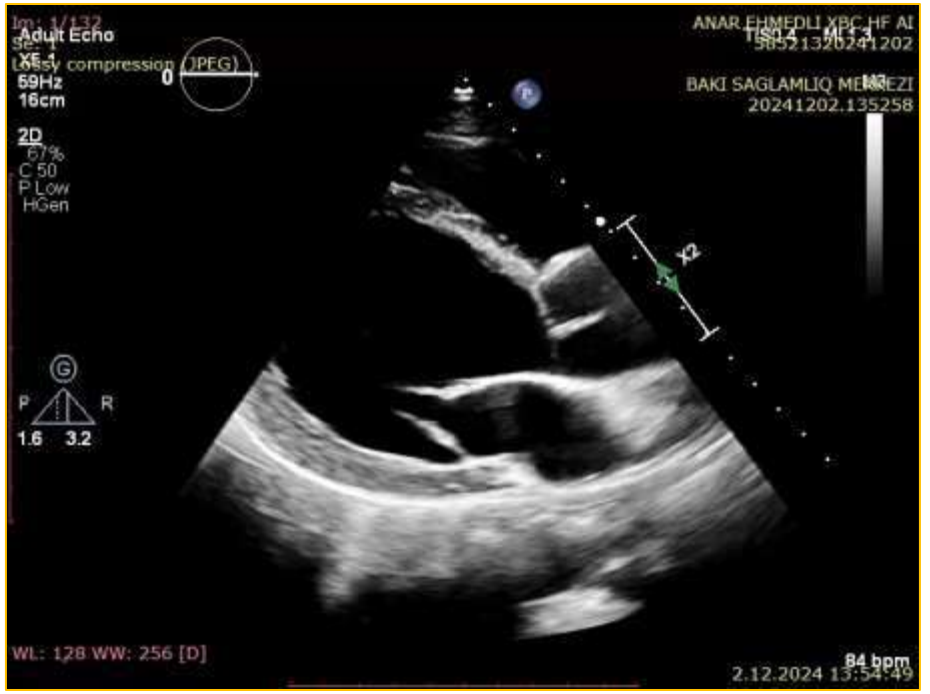
ARNI + SGLT2i added to treatment

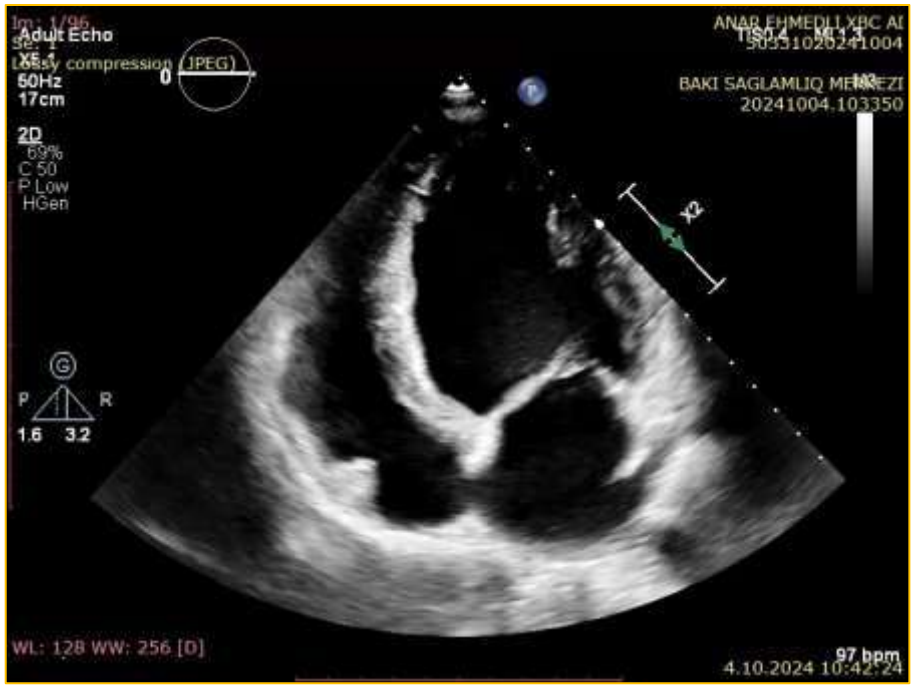
After 2 weeks, in the re-check visit, NT-pro BNP values were 3200 (moderate increase compared to the previous visit) and eGFR was 27.9 mL/min/1.73 m2, so Dapagliflozin 10 mg and Finerenon 10 mg were added to the treatment.

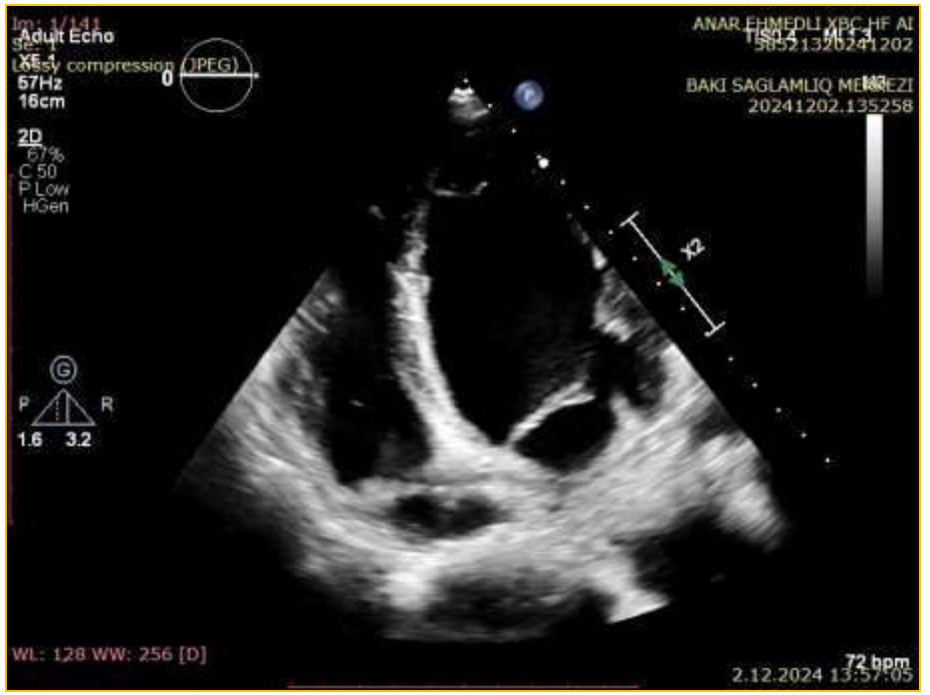
Frequent blood pressure monitoring was recommended.

ECHO after treatment









ARNI in CKD

- After oral administration, sacubitril/valsartan was divided into valsartan and prodrug sacubitril
- Valsartan is primarily excreted via the biliary route, and renal impairment does not affect its pharmacokinetics.
- Sacubitril is rapidly converted to the active neprilysin inhibitor sacubitrilat.
- Renal function does’nt play a significant role in the excretion of sacubitrilat. Less than 2% of the total dose is excreted in the urine and feces, while sacubitrilat is excreted primarily via the kidneys, suggesting that exposure increases when renal function decreases.

ARNI in CKD

1

✔ The optimal treatment of HF in patients with stage 4 or 5 CKD (eGFR, <30 mL/min/1.73 m2) is unclear as there is little evidence regarding this.

2

✔ **Unfortunately, most of the previous randomized clinical trials that guided the management of HFrEF with an ARNI defined CKD as baseline eGFR of <60 mL/min/1.73 m2 and excluded patients with severe CKD (eGFR, <30 mL/min/1.73 m2)**

3

✔ There is a risk of safety and toxicity of sacubitril/valsartan in patients with stage 4 or 5 CKD with eGFR below 30 mL/min/1.73 m2

ARNI in HFrEF + CKD

- In a real-world study, showed that patients with stage 4 or 5 CKD treated with sacubitril/valsartan had 28% fewer cardiovascular deaths or HF hospitalizations than those treated with standard HF treatment, including with eGFR of <30 mL/min/1.73 m2 among the whole study population of 932 patients with HFrEF.
- Quiroga et al. investigated 66 patients with stage 1 to 4 CKD and HFrEF (17% of stage 4 CKD) and found that sacubitril/valsartan was safe in patients with CKD, suggesting stability in CKD progression after 6 months.

DOI: 10.1159/000541939

https://doi.org/10.23876/j.krcp.21.900

ARNI in HFrEF + CKD

- There is not enough evidence of data - ARNI in patients with ESRD on dialysis.
- Our patient had worsened LV and renal functions, and elevated cardiac biomarkers - after ARNI treatment, clinical improvement and decrease in biomarkers were observed.
- Our patient was clinically stable for 1.5 years. Potassium values remained stable at 5.6-5.7.
- For this reason, Finerenon was suspended.

SGLT2 in HFrEF + CKD

- However, the benefits of SGLT2 inhibitors for HFrEF management in patients with severe CKD remain unclear. The glucosuric effect of SGLT-2 inhibition declines with the estimated glomerular filtration rate

- Currently, the use of dapagliflozin and empagliflozin is recommended in patients with eGFR of ≥30 mL/min/1.73 m2 and ≥20 mL/min/1.73 m2, respectively, since the glucosuric effects of SGLT2 inhibitors may be reduced in those with a lower eGFR.

DOI: https://doi.org/10.3949/ccjm.91a.23093

SGLT2 in HFrEF + CKD

- There are little data assessing the combination of an ARNI and an SGLT2 inhibitor, even though the benefit of SGLT2 inhibition was consistent in patients already treated with an ARNI in both Dapagliflozin (DAPA-HF) and Empagliflozin Outcome Trial in Patients With Chronic Heart Failure with Reduced Ejection Fraction (EMPEROR-Reduced)

Trials of sodium-glucose cotransporter 2 inhibitors in heart failure

Trial	Patients	Intervention	Primary composite end point	Primary composite results
Heart failure with reduced ejection fraction				
DAPA-HF (2019)[8]	4,744 adults EF ≤ 40% Established HF eGFR < 30 mL/minute/1.73 m²	Dapagliflozin 10 mg	Cardiovascular death or worsening heart failure	16.3% vs 21.2% (NNT = 21)
EMPEROR-Reduced (2020)[9]	3,730 adults EF ≤ 40% Established HF eGFR < 20 mL/minute/1.73 m²	Empagliflozin 10 mg	Cardiovascular death or worsening heart failure	19.4% vs 24.7% (NNT = 19)
Heart failure with preserved ejection fraction				
EMPEROR-Preserved (2021)[15]	5,988 adults EF > 40% New York Heart Association class II–IV HF eGFR < 20 mL/minute/1.73 m²	Empagliflozin 10 mg	Cardiovascular death or hospitalization for heart failure	13.8% vs 17.1% (NNT = 31)
DELIVER-HF (2022)[16]	6,263 adults EF > 40% Stabilized HF eGFR > 25 mL/minute/1.73 m² With or without diabetes mellitus	Dapagliflozin 10 mg	Cardiovascular death or worsening heart failure	16.4% vs 19.5% (NNT = 32)
Acute decompensated heart failure				
EMPULSE (2022)[19]	530 adults Any EF Acute decompensated HF eGFR < 20 mL/minute/1.73 m²	Empagliflozin 10 mg	All-cause death, heart failure events,[a] Kansas City Cardiomyopathy Questionnaire score	53.4% vs 39.7% Win ratio[b] 1.36 (95% confidence interval: 1.09–1.68)
SOLOIST-WHF (2021)[20]	1,222 adults Any EF Acute decompensated HF eGFR < 30 mL/minute/1.73 m² Type 2 diabetes	Sotagliflozin 200 or 400 mg	Events of cardiovascular deaths, hospitalizations and urgent visits for heart failure	51% vs 76.3% (NNT = 4)

DOI: https://doi.org/10.3949/ccjm.91a.23093

SGLT2 in HFrEF + CKD

All 3 trials showed benefit in reducing the risk of chronic kidney disease progression or cardiovascular death, with relative risk reductions ranging from 28% to 39%

Trials of sodium-glucose cotransporter 2 inhibitors in chronic kidney disease

Trial	Patients	Intervention	Primary composite end point	Primary composite results
CREDENCE (2019)[24]	4,401 adults eGFR 30–89 mL/minute/1.73 m^2 and UACR 301–5,000 mg/g Type 2 diabetes	Canagliflozin 100 mg	End-stage kidney disease,[a] double serum creatinine, or cardiovascular or renal death	43.2 vs 61.2 events/1,000 patient years (NNT = 22)
DAPA-CKD (2020)[22]	4,304 adults eGFR 25–75 mL/minute/1.73 m^2 and UACR 200–5,000 mg/g With or without diabetes mellitus	Dapagliflozin 10 mg	≥ 50% sustained decline in eGFR, end-stage kidney disease,[b] or cardiovascular or renal death	9.2% vs 14.5% (NNT = 19)
EMPA-KIDNEY (2023)[23]	6,609 adults eGFR 20–44 mL/minute/1.73 m^2 or eGFR 45–89 mL/minute/1.73 m^2 and UACR ≥ 200 mg/g With or without diabetes mellitus	Empagliflozin 10 mg	Kidney disease progression[c] or cardiovascular death	13.1% vs 16.9% (NNT = 26)

[a]CREDENCE: dialysis for at least 30 days, kidney transplantation, or eGFR < 15 mL/minute/1.73 m^2.
[b]DAPA-CKD: maintenance dialysis ≥ 28 days, kidney transplantation, or eGFR < 15 mL/minute/1.73 m^2.
[c]EMPA-KIDNEY: initiation of maintenance dialysis, receipt of kidney transplant, eGFR < 10 mL/minute/1.73 m^2, sustained decrease in eGFR ≥ 40%, or renal death.

CREDENCE = Canagliflozin and Renal Events in Diabetes with Established Nephropathy Clinical Evaluation; DAPA-CKD = Dapagliflozin and Prevention of Adverse Outcomes in Chronic Kidney Disease; eGFR = estimated glomerular filtration rate; EMPA-KIDNEY = Study of Heart and Kidney Protection with Empagliflozin; NNT = number needed to treat; UACR = urine albumin-to-creatinine ratio

The glucosuric effect of SGLT-2 inhibition declines with the estimated glomerular filtration rate. Therefore, at estimated glomerular filtration rates below 30 to 45 mL/minute/1.73 m2 , SGLT-2 inhibitors have minimal effect on blood glucose levels

- ✔ However, based on these studies, the American Diabetes Association recommends the use of an SGLT-2 inhibitor to reduce the risk of chronic kidney disease progression and cardiovascular events in patients with type 2 diabetes, diabetic kidney disease with a urine albumin-to-creatinine ratio of 200 mg/g or greater and an estimated glomerular filtration rate as low as 20 mL/min/1.73 m2.

DOI: https://doi.org/10.3949/ccjm.91a.23093

beware of hypotension !

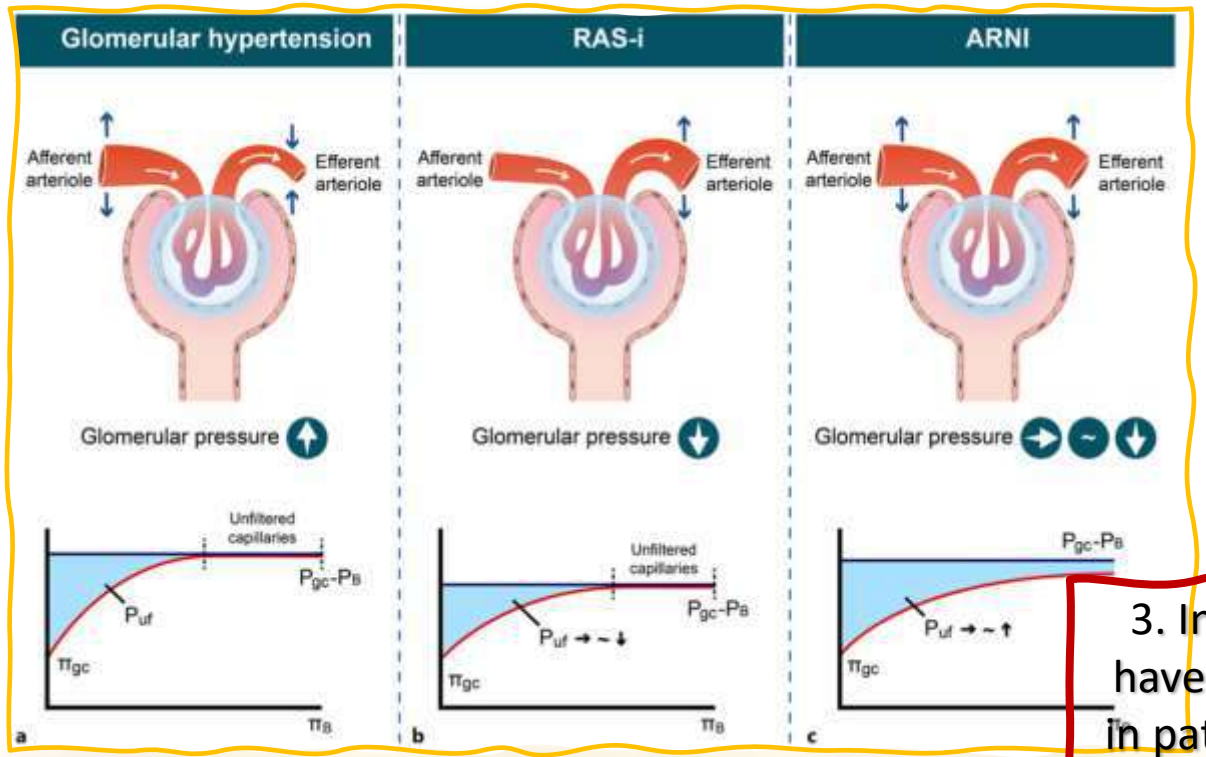
🎯 Excessive hypotension due to ARNI use may exacerbate renal dysfunction, especially in elderly patients with comorbid HF and low ejection fraction.

🎯 Therefore, careful blood pressure monitoring is essential to optimize the renal benefits of ARNI and minimize side effects.

🎯 The PARADIGM-HF study, together with subgroup analyses from PARAGON-HF, demonstrated that ARNI delayed the decline in renal function.

🎯 Regardless of CKD status, the sacubitril/valsartan group showed a significantly lower rate of eGFR decline compared to the enalapril group.

1. However, the United Kingdom Heart and Renal Protection-III (UK HARP-III trial), which focused on ARNI and the endpoint of renal events, didnt show a significant change in the rate of reduction of the estimated glomerular filtration rate (eGFR) with sacubitril/valsartan



2. Progression to end-stage renal disease (ESRD) unchanged with ARNIs, a newly renal composite outcome (eGFR decline of ≥50% or progression to ESRD) demonstrated a significant decrease in the sacubitril/valsartan group (hazard ratio [HR]: 0.63; 95% confidence interval [CI]: 0.42–0.95; p = 0.028)

3. In summary, although subgroup analyses from HF studies have suggested potential renoprotection of ARNI, particularly in patients with eGFR >30 mL/min and LVEF between 30% and 60%, the UK HARP-III study showed similar effects on renoprotection and albuminuria with sacubitril/valsartan and irbesartan in CKD patients without HF.

DOI: 10.1159/000541939

JAK2 MPD + Rituximab + HFrEF

🎯 Oncological diseases and hematological malignancies are another comorbid condition for heart failure.

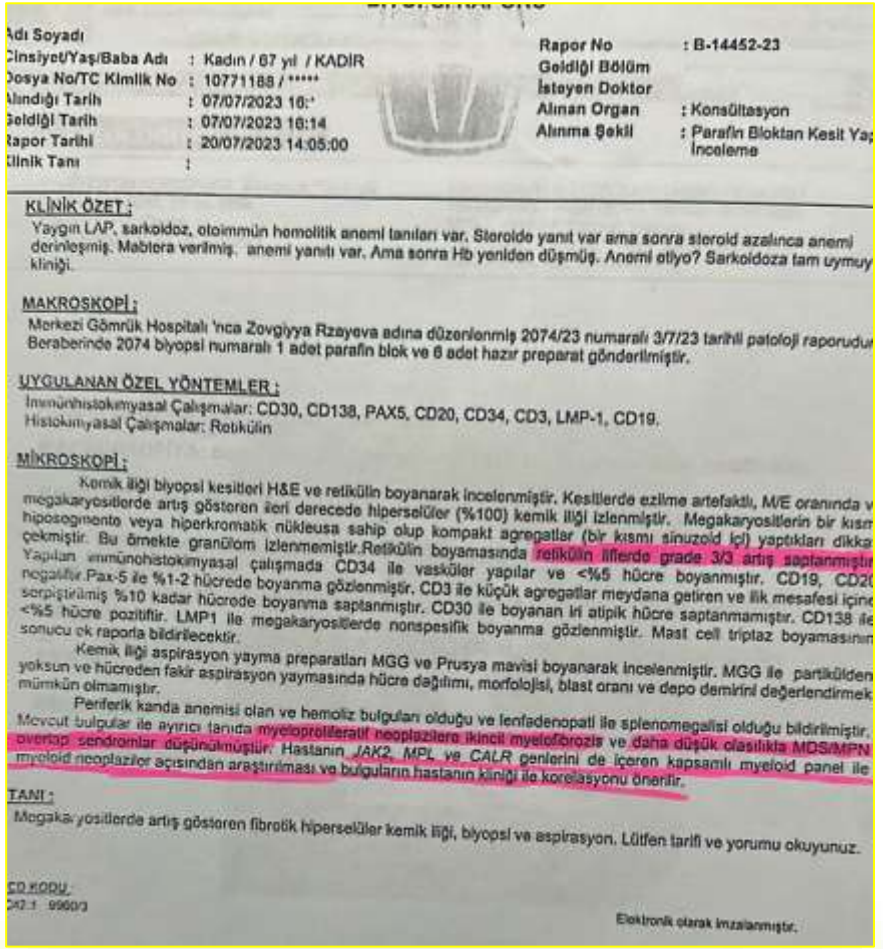
Alberto Aimo doi:10.1093/ehjcr/ytz227

JAK2 GENE MUTATION + HFrEF

A Janus kinase 2 gene mutation known as *JAK2mutV617F* in hematopoietic progenitor cells causes most Philadelphia chromosome–negative myeloproliferative disorders (MPDs) and may be associated with cardiac hypertrophy, atherosclerotic cardiovascular disease and heart failure

A 67-year-old woman with *JAK2mutV617F*-positive primary myelofibrosis (PMF) underwent splenectomy for symptomatic splenomegaly. And she has been receiving specific treatment for 4 years. She had a heart failure clinic for the last 1.5 years.

To describe this case of an MPD associated with primary cardiomyopathy in which *JAK2 mutation* was found in both myeloid cells



Adı Soyadı
Cinsiyet/Yaş/Baba Adı : Kadın / 67 yıl / KADİR
Dosya No/TC Kimlik No : 10771188 / *****
Alındığı Tarih : 07/07/2023 10:*
Geldiği Tarih : 07/07/2023 10:14
Rapor Tarihi : 20/07/2023 14:05:00
Klinik Tanı :

Rapor No : B-14452-23
Geldiği Bölüm
İsteyen Doktor
Alınan Organ : Konsültasyon
Alınma Şekli : Parafin Bloktan Kesit Yaş İnceleme

KLİNİK ÖZET :
Yaygın LAP, sarkoidoz, otoimmün hemolitik anemi tanıları var. Steroide yanıt var ama sonra steroid azalınca anemi derinleşmiş. Mabtera verilmiş. anemi yanıtı var. Ama sonra Hb yeniden düşmüş. Anemi atiyo? Sarkoidoza tam uymuy kliniği.

MAKROSKOPİ :
Merkezi Gömrük Hospitalı 'nca Zovgiyya Rzayeva adına düzenlenmiş 2074/23 numaralı 3/7/23 tarihli patoloji raporudur Beraberinde 2074 biyopsi numaralı 1 adet parafin blok ve 8 adet hazır preparat gönderilmiştir.

UYGULANAN ÖZEL YÖNTEMLER :
İmmünhistokemyasal Çalışmalar: CD30, CD138, PAX5, CD20, CD34, CD3, LMP-1, CD19.
Histokimyasal Çalışmalar: Retikülin

MİKROSKOPİ :
Kemik iliği biyopsi kesitleri H&E ve retikülin boyanarak incelenmiştir. Kesitlerde ezilme artefaktı, M/E oranında v megakaryositlerde artış gösteren ileri derecede hiperselüler (%100) kemik iliği izlenmiştir. Megakaryositlerin bir kısm hipositomento veya hiperkromatik nükleusa sahip olup kompakt agregatlar (bir kısmı sinuzoid içi) yaptıkları dikka çekmiştir. Bu örnekte granülom izlenmemiştir.Retikülin boyamasında retikülin liflerde grade 3/3 artış saptanmıştır Yapılan immünohistokimyasal çalışmada CD34 ile vasküler yapılar ve <%5 hücre boyanmıştır. CD19, CD2(negatiftir.Pax-5 ile %1-2 hücrede boyanma gözlenmiştir. CD3 ile küçük agregatlar meydana getiren ve ilik mesafesi içine serpiştirilmiş %10 kadar hücrede boyanma saptanmıştır. CD30 ile boyanan iri atipik hücre saptanmamıştır. CD138 ile <%5 hücre pozitiftir. LMP1 ile megakaryositlerde nonspesifik boyanma gözlenmiştir. Mast cell triptaz boyamasının sonucu ek raporla bildirilecektir.
Kemik iliği aspirasyon yayma preparatları MGG ve Prusya mavisi boyanarak incelenmiştir. MGG ile partiküler yoksun ve hücreden fakir aspirasyon yaymasında hücre dağılımı, morfolojisi, blast oranı ve depo demirini değerlendirmek mümkün olmamıştır.
Periferik kanda anemisi olan ve hemoliz bulguları olduğu ve lenfadenopati ile splenomegalisi olduğu bildirilmiştir. Mevcut bulgular ile ayırıcı tanıda myeloproliferatif neoplazilere ikinci myelofibrozis ve daha düşük olasılıkla MDS/MPN overlap sendromlar düşünülmüştür. Hastanın *JAK2, MPL ve CALR* genlerini de içeren kapsamlı myeloid panel ile myeloid neoplaziler açısından araştırılması ve bulguların hastanın kliniği ile korelasyonu önerilir.

TANI :
Megakaryositlerde artış gösteren fibrotik hiperselüler kemik iliği, biyopsi ve aspirasyon. Lütfen tarifi ve yorumu okuyunuz.

ICD KODU
D47.1 9960/3

Elektronik olarak imzalanmıştır.

HFrEF and oncological comorbidity

JAK2 MPD + Rituximab + HFrEF

- A Janus kinase 2 gene mutation known as JAK2mutV617F in hematopoietic progenitor cells causes Philadelphia chromosome–negative myeloproliferative disorders (MPDs) (1) and may be associated with cardiac hypertrophy
- The downstream signaling pathway of active JAK2 is known to be associated with myocardial hypertrophy. Our patient had a JAK2 gene mutation and we thought that his cardiomegaly was related to the mutated JAK2mutV617F.
- This patient received rituximab chemotherapy due to myeloproliferative disease, and the JAK2 mutation that causes MPD has been reported in the literature to be a trigger gene for heart failure

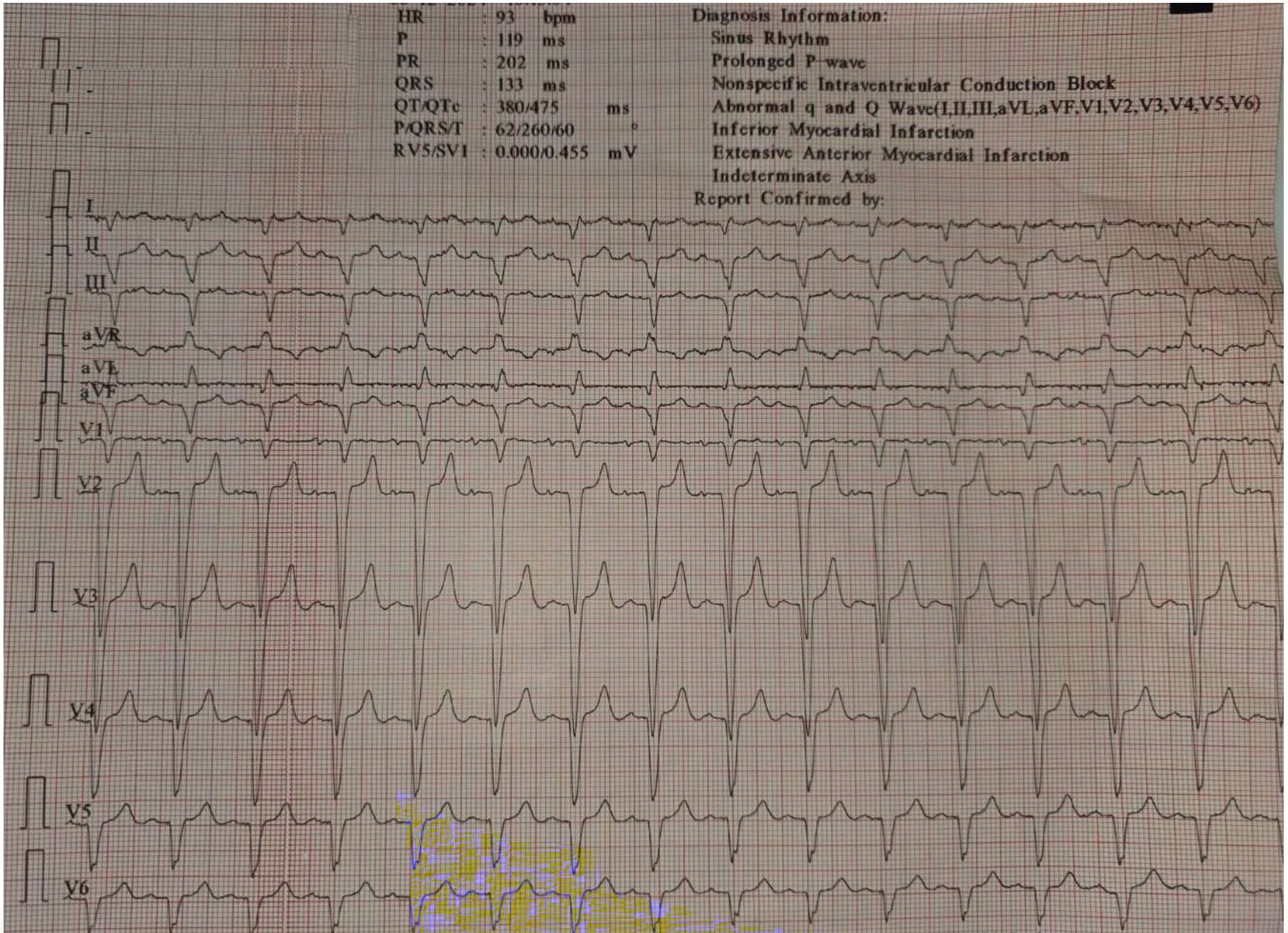
http://annals.org/pdfaccess.ashx?url=/data/journals/aim/20140

JAK2 MPD + Rituximab + HFrEF

🎯 Our patient has been receiving chemotherapy and immunomodulatory therapy for 4 years, and Mabthera and other specific treatments were last applied 1 .5 year ago. The patient had shortness of breath for the last 6 months, and the tests showed that the ejection fraction was suppressed

http://annals.org/pdfaccess.ashx?url=/data/journals/aim/20140

ECG


HR : 93 bpm
P : 119 ms
PR : 202 ms
QRS : 133 ms
QT/QTc : 380/475 ms
P/QRS/T : 62/260/60 °
RV5/SV1 : 0.000/0.455 mV
Diagnosis Information:
Sinus Rhythm
Prolonged P-wave
Nonspecific Intraventricular Conduction Block
Abnormal q and Q Wave(I,II,III,aVL,aVF,V1,V2,V3,V4,V5,V6)
Inferior Myocardial Infarction
Extensive Anterior Myocardial Infarction
Indeterminate Axis
Report Confirmed by:
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

rituximab

- Although it has been reported that the rituximab treatment our patient received decreased the heart failure symptoms with its anti-inflammatory effect, there are also articles reporting the opposite.
- Rituximab can be considered in patients with DCMi when the EMB shows a significant infiltrate of CD20+ cells (>7 cells/mm2) and no evidence of viral infection, particularly when the onset of HF symptoms

Alberto Aimo doi:10.1093/ehjcr/ytz227

🎯 Another important issue is the risk of cardiotoxicity related to rituximab therapy.

🎯 There is emerging data that Rituximab and other monoclonal antibody-based chemotherapy represent a newer class of medications that have cardiotoxic profiles.

🎯 Rituximab has been reported to cause hypotension, hypoxia, acute myocardial infarction, arrhythmias, and cardiogenic shock during the infusion process.

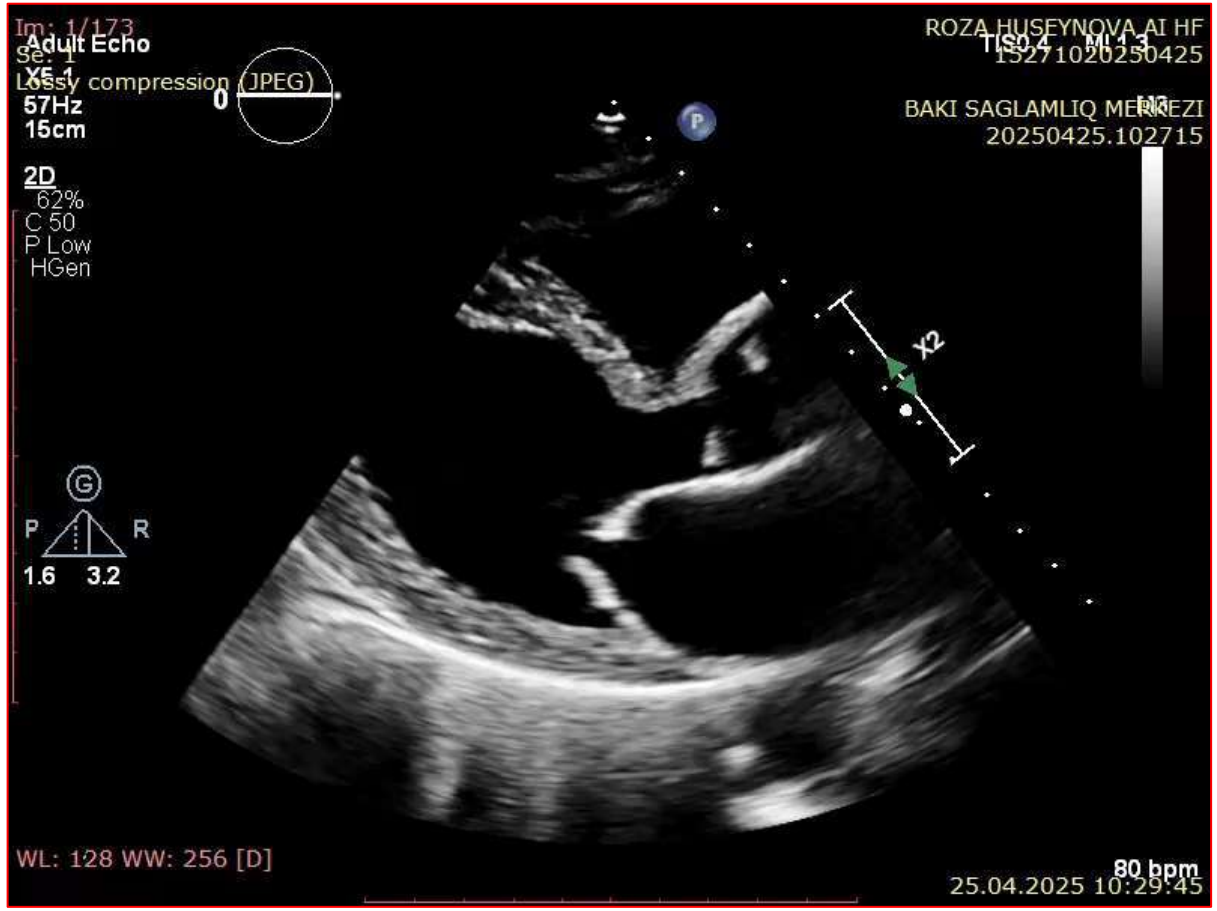
🎯 There are also reported incidences where rituximab has caused nonischaemic dilated cardiomyopathy

Alberto Aimo doi:10.1093/ehjcr/ytz227

- Our patient's heart failure findings emerged 1 year after chemotherapy, LVEF had normal values in the control echo before and after chemotherapy.
- For this reason, we thought that the event was related to the JAK2 gene mutation. Suppressed strain findings and poor left ventricular ejection fraction were seen in the echo and CMRI.

Alberto Aimo doi:10.1093/ehjcr/ytz227

ECHO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Adult Echo
Se: 1
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57Hz
15cm
2D
62%
C 50
P Low
HGen
ROZA HUSEYNOVA AI HF
152710202504 25
BAKI SAGLAMLIQ MERKEZI
20250425.102715
X2
P
R
1.6 3.2
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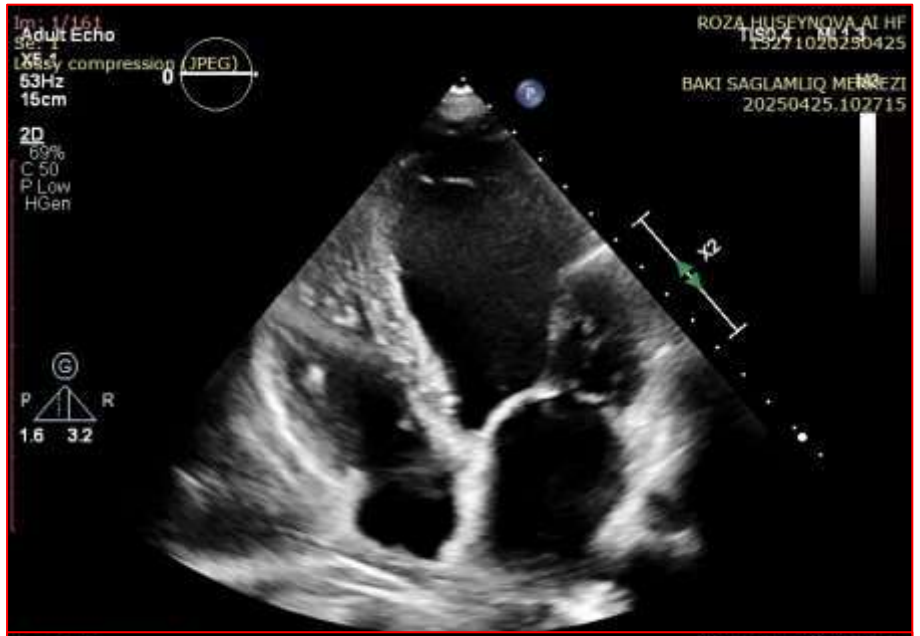
80 bpm
25.04.2025 10:29:45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15cm
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69%
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HGen
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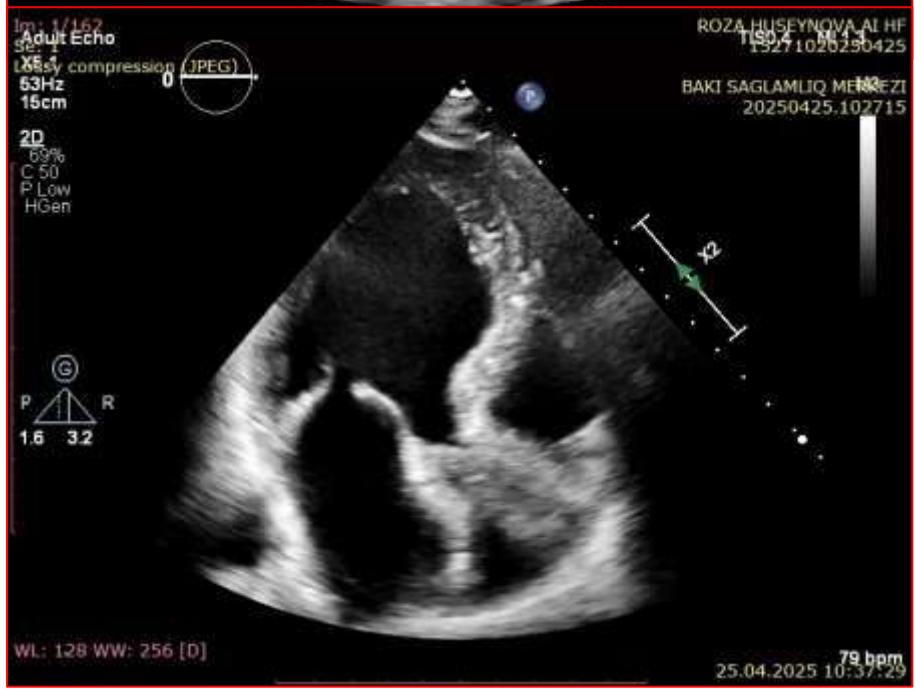
X2
P
R
1.6 3.2


Adult Echo
Lossy compression (JPEG)
53Hz
15cm
2D
69%
C 50
P Low
HGen
BAKI SAGLAMLIQ MERKEZI
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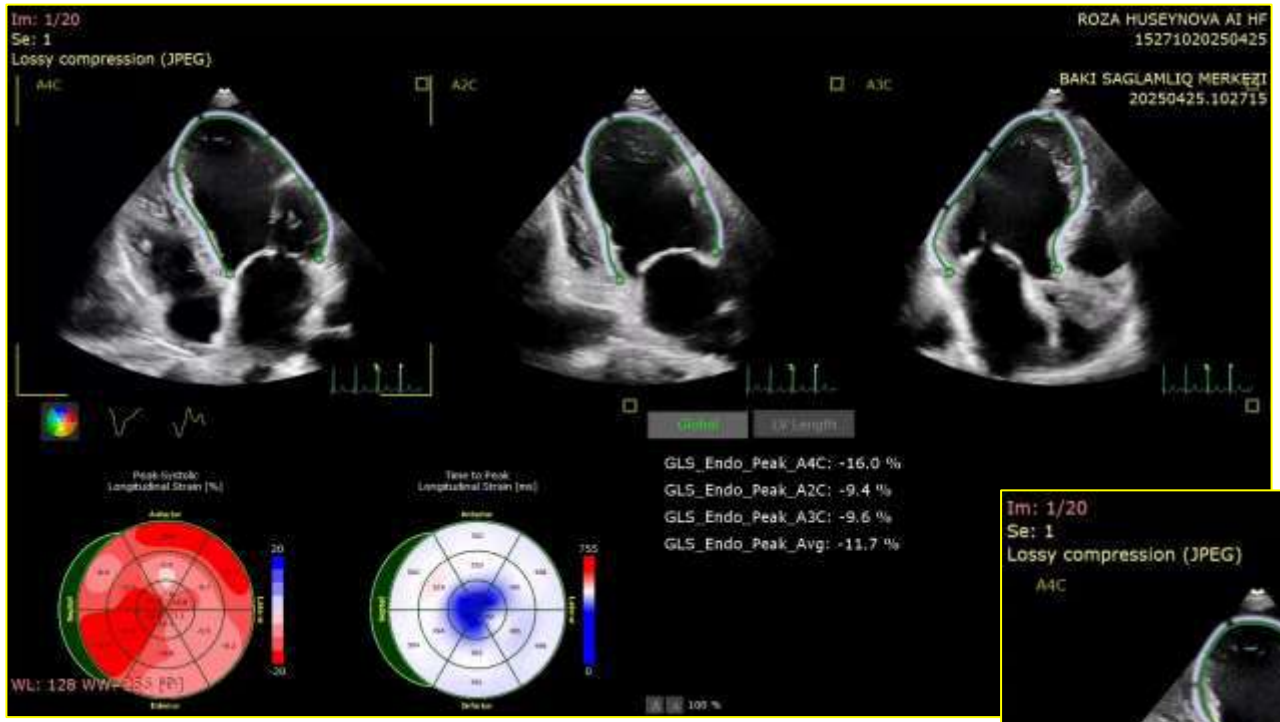
X2
P
R
1.6 3.2
WL: 128 WW: 256 [D]
79 bpm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Se: 1
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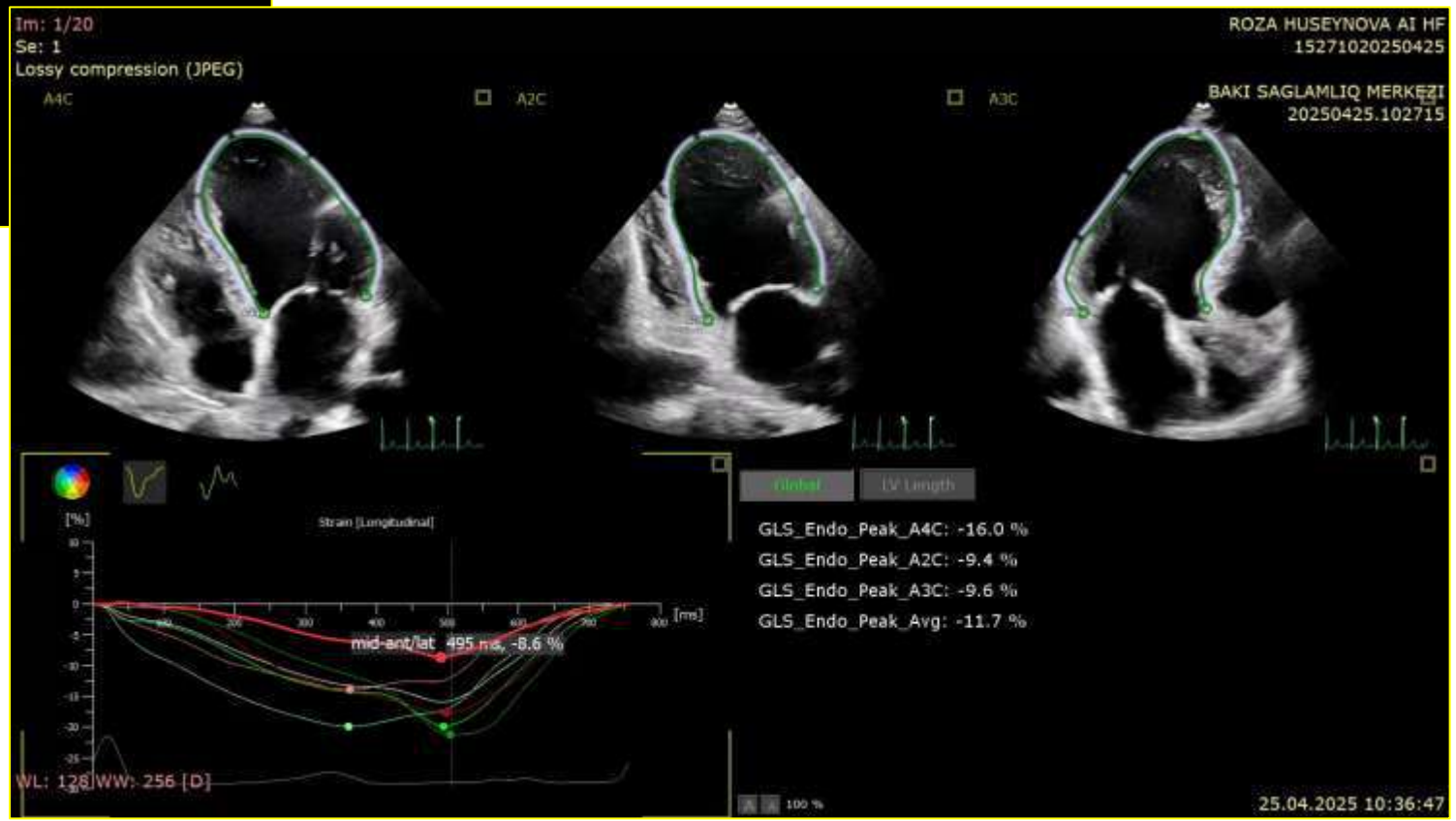
BAKI SAGLAMLIQ MERKEZI
20250425.102715
A4C
A2C
A3C
Global
LV Length
GLS_Endo_Peak_A4C: -16.0 %
GLS_Endo_Peak_A2C: -9.4 %
GLS_Endo_Peak_A3C: -9.6 %
GLS_Endo_Peak_Avg: -11.7 %
Peak-Systolic Longitudinal Strain [%]
Time to Peak Longitudinal Strain [ms]
WL: 128 WW: 255 [D]
100 %


Im: 1/20
Se: 1
Lossy compression (JPEG)
ROZA HUSEYNOVA AI HF
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BAKI SAGLAMLIQ MERKEZI
20250425.102715
A4C
A2C
A3C
Global
LV Length
GLS_Endo_Peak_A4C: -16.0 %
GLS_Endo_Peak_A2C: -9.4 %
GLS_Endo_Peak_A3C: -9.6 %
GLS_Endo_Peak_Avg: -11.7 %
Strain [Longitudinal]
[%]
[ms]
mid-ant/lat 495 ms, -8.6 %
WL: 128 WW: 256 [D]
100 %
25.04.2025 10:36:47

take home messages

➡️ In our daily outpatient clinic examinations, we can see many comorbid conditions along with heart failure. Here, we have to manage many comorbid conditions such as DM, HT, CKD, COPD, atrial fibrillation, oncological diseases, chemotherapeutic agents, rheumatological diseases, amyloidosis, sarcoidic myocarditis and ect

➡️ These patients need to be evaluated individually and followed up and treated on a patient basis.

ARNI in HFrEF + CKD

✔ Inhibiting neprilysin, which is the enzyme responsible for degrading vasoactive peptides such as natriuretic peptides, ARNI enhances the levels of these peptides, thus leading to vasodi- lation, diuresis, and inhibition of cardiac hypertrophy and fibrosis

✔ In addition, blockade of the RAAS by valsartan complements the effects of neprilysin inhibition, thus maximizing its therapeutic benefits and minimizing potential negative effects such as increased endothelin-1 and angiotensin II levels. This combination therapy has resulted in significantly improved outcomes in patients with HFrEF.

DOI: 10.1159/000541939