

THE ECHO-DRIVEN DECISION IN HEART FAILURE MANAGEMENT

CASE-BASED DISCUSSION

SILFI PAULINE SIRAIT, MD

☎ 0811-1900-8855

✉ scientific_ihefcard@inahfcarmet.org

📷 @ina.hf

www.ihefcard.com

11-13 JUNE 2026

Sheraton Grand Jakarta, Gandaria City

iHEF CARD

CASE 1

Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease

☎ 0811-1900-8855

✉ scientific_ihefcard@inahfcardmet.org

📷 @ina.hf

www.ihefcard.com

11-13 JUNE 2026

Sheraton Grand Jakarta, Gandaria City

CASE ILLUSTRATION

Patient

Mr. X · Male, 58 years old

Chief Complaint

Progressive dyspnea over 2 weeks, now at rest

Associated

3-pillow orthopnea · PND · Bilateral leg swelling × 5 days

Denies

Chest pain · Fever · Recent illness

Past History

Hypertension · Type 2 DM



Physical Examination

Vital Signs

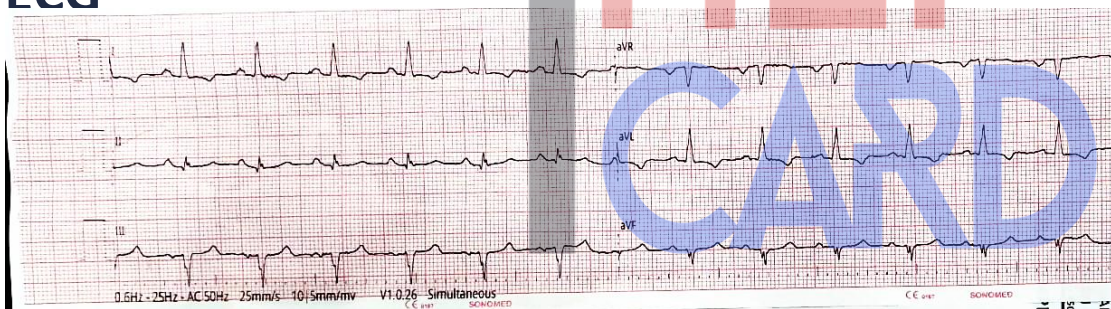
BP	162 / 98 mmHg
HR	114 bpm · Sinus Tachycardia
RR	24 / min
SpO ₂	89% on Room Air
Temp	37.2 °C

Clinical Findings

- Raised JVP ~14 cmH₂O
- S3 gallop on auscultation
- Bibasal crackles
- 3+ pitting oedema — to knees
- Diaphoretic, mildly distressed

Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease

ECG

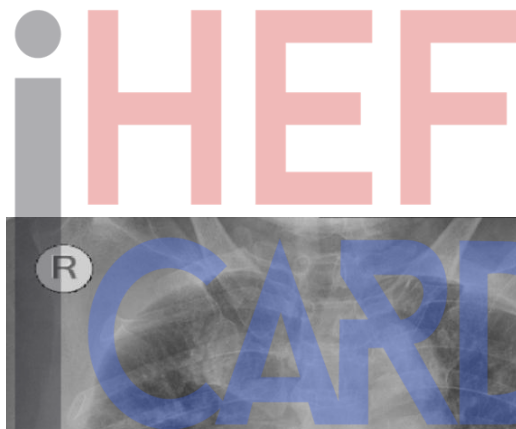


Indonesian Symposium on Heart Failure and Cardiometabolic Disease

Sinus tachycardia - Old myocardial infarction at anterior, inferior, and lateral walls



Chest X-Ray



Indonesian Symposium on Heart Failure and Cardiometabolic Disease

Cardiomegaly with signs of congestion

Lab Results

NT pro BNP 2,140 pg/mL

Na⁺ 133 mEq/L

Creatinine 1.4 mg/dL

eGFR 54 mL/min

Troponin I 0.08 ng/mL

HbA1c 8.2%

Q1

Clinical profile: "warm and wet" with elevated BP (162/98), SpO₂ 89%, crackles, raised JVP, oedema.
What is the most appropriate first-line treatment in the ER?

A

IV furosemide alone

B

IV furosemide PLUS IV nitrates

C

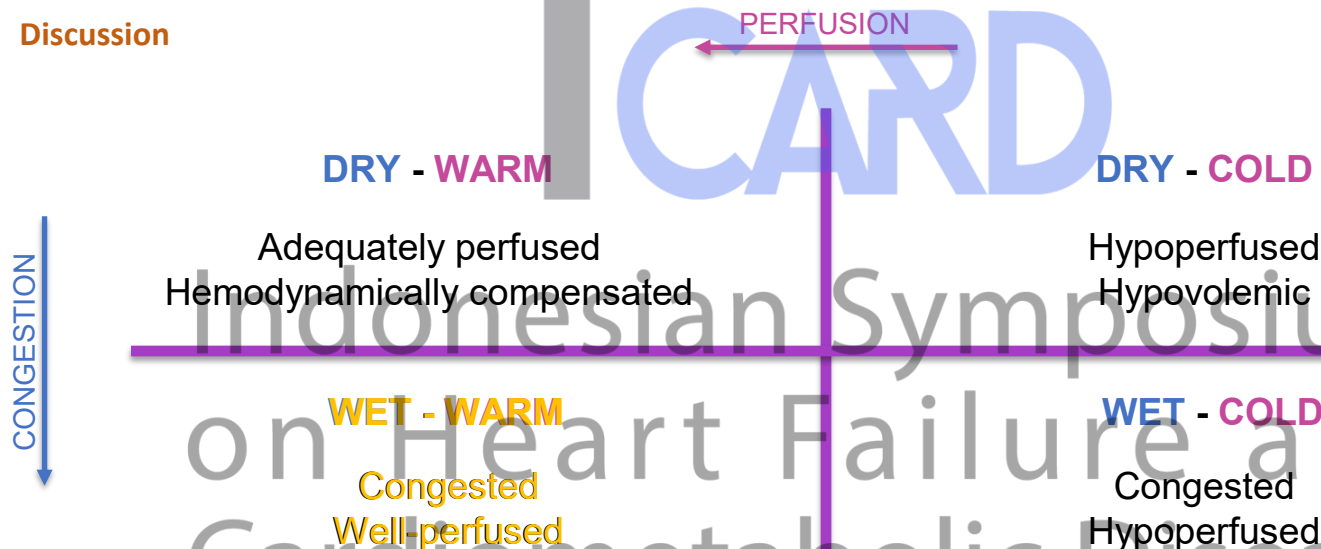
IV dobutamine

D

Oral beta-blocker

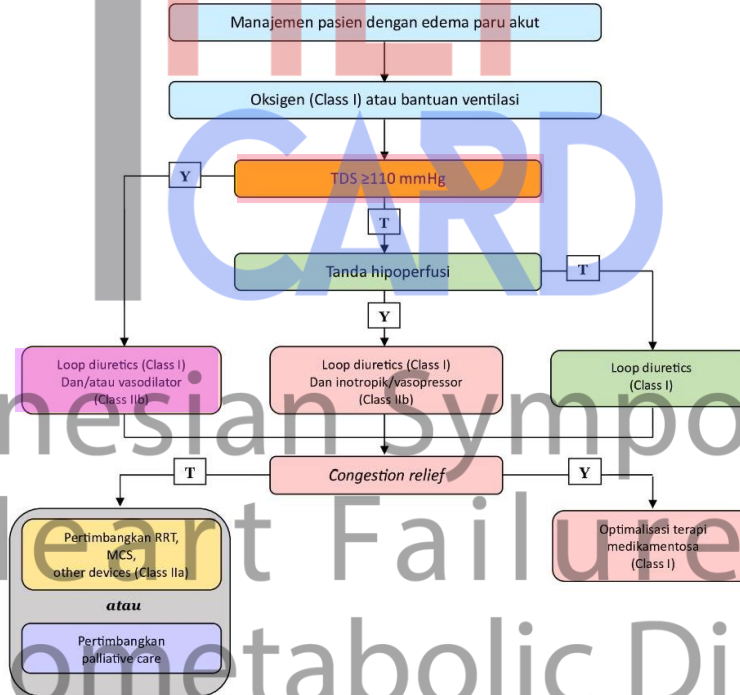
Q1 ✓ Answer B: IV furosemide PLUS IV nitrates

Discussion



Q1 ✓ Answer B: IV furose

Discussion



Q1 ✓ Answer B: IV furosemide PLUS IV nitrates

Discussion

How IV nitrates work in acute heart failure:

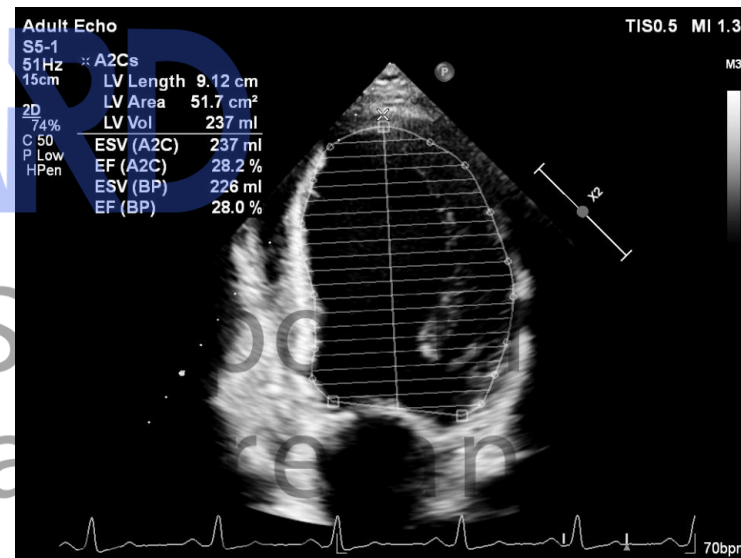
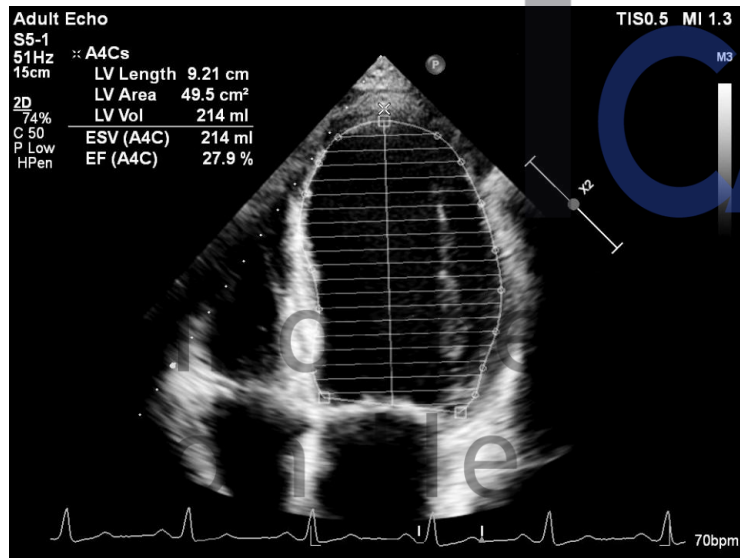
- At lower doses → venodilation → ↓ preload → relieves pulmonary congestion
- At higher doses → arterial dilation → ↓ afterload → reduces work of the failing heart

Indonesian Symposium on Heart Failure and Cardiometabolic Disease

Baseline Echo

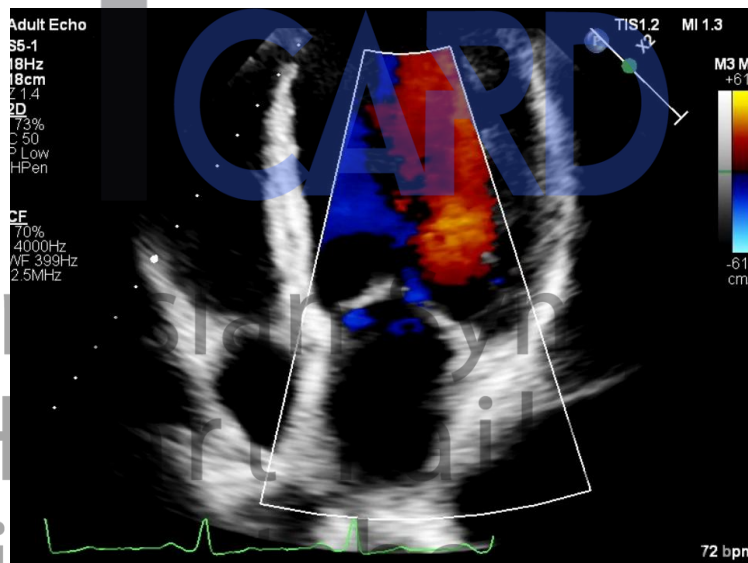
- Dilated left atrium and left ventricle
- Reduced LV systolic function, EF 28% (Simpson's Biplane)
- RWMA (+)
- Eccentric LVH with grade II diastolic dysfunction
- Normal RV contractility, TAPSE 1.7 cm

Baseline Echo



Cardiometabolic Disease

Baseline Echo



Indonesian Symposium on Heart Failure and Cardiometabolic Disease

Q2

Classify the patient based on EF.

After IV nitrates + diuretics, patient improving but still mildly wet.

Which GDMT should you initiate EARLY during admission?

A

HFmrEF — start only SGLT2 inhibitor for now

B

HFrEF — wait until fully dry before starting any GDMT

C

HFrEF — start SGLT2i + ARNI (or ACEi/ARB) + MRA during admission

D

HFpEF — start a thiazide diuretic

Q2 ✓ Answer C: HFrEF — start SGLT2i + ARNI + MRA during admission

Discussion

HF with reduced EF (HFrEF):

- HF with LVEF $\leq 40\%$

HF with mildly reduced EF (HFmrEF):

- HF with LVEF 41–49%

HF with preserved EF (HFpEF):

- HF with LVEF $\geq 50\%$

HF with improved EF (HFimpEF):

- HF with a baseline LVEF $\leq 40\%$, a ≥ 10 point increase from baseline LVEF, and a second measurement of LVEF $> 40\%$

Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease

Bozkurt B, et al. Eur J Heart Fail. 2021 Mar;23(3):352-380.

Q2 ✓ Answer C: HFrEF — start SGLT2i + ARNI + MRA during admission

Discussion

Randomized Controlled Trial. Lancet. 2023; Dec 3;400(10367):1938-1952

doi: 10.1016/S0140-6736(23)01233-1

Recommendation

Class^a

Level^b

Safely An intensive strategy of initiation and rapid
direct up-titration of evidence-based treatment before
multidisciplinary discharge and during frequent and careful follow-up
visits in the first 6 weeks following a HF
hospitalization is recommended to reduce the risk of
HF rehospitalization or death.^{c,d,e 16}

**Rapid
reduction**

I

B

© ESC 2023

DMT

Hadiza Saidu¹³, Etienne Gayat¹⁴, Peter S Pang¹⁵, Jelena Celutkiene¹⁶, Gad Cotter²

Q2 ✓ Answer C: HFrEF — start SGLT2i + ARNI + MRA during admission

Discussion

What to monitor while initiating each drug

SGLT2i

- Safest pillar. No BP/HR effect
- Check eGFR (use if ≥ 20); small initial dip 5–10% expected

ARNI

- PIONEER-HF — safe in hospital
- Check SBP ≥ 100 , eGFR, K^+
- 36-hour wash-out after ACEi

MRA

- Diuretic + disease-modifying
- Check K^+ + Cr at baseline and 1 week

Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease

iHEF iCARD

DAY 3 OF ADMISSION

Patient is dry and warm — euvolemic, stable

BP 138/86 · HR 84 (sinus) · SpO₂ 97% RA · No crackles · No oedema

Already on: dapagliflozin · sacubitril/valsartan · spironolactone

Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease

Q3

Patient stable: BP 138/86, HR 84, SpO₂ 97%. Already on dapagliflozin + sacubitril/valsartan + spironolactone. What medication should you start next?

A

Start **carvedilol** 3.125 mg twice daily

B

Add **ivabradine** 5 mg twice daily for heart rate control

C

Add **digoxin** for additional rate control

D

Hold any new medication — wait 1 month before adding anything

Q3 ✓ Answer A: Start carvedilol 3.125 mg twice daily

Discussion

Why a beta-blocker NOW?

COPERNICUS trial — beta-blockade gives mortality benefit even with EF <25%, PROVIDED the patient is stable at initiation.

STRONG-HF supports initiation before discharge.

This patient meets all criteria: euvolemic, no IV diuretics, no resting tachycardia, no symptomatic hypotension.

Start LOW, go SLOW.

Q3 — Why carvedilol may be preferred for THIS patient

Carvedilol is a third-generation BB with non-selective β_1/β_2 + α_1 -adrenergic blockade + antioxidant properties.

Three patient-specific reasons:

1

Persisting elevated BP (138/86) despite optimised ARNI + diuretics

The **α_1 -adrenergic blockade adds vasodilation** — lowers BP more effectively than pure β_1 -selective agents. Clinically advantageous in hypertensive HFrEF where additional afterload reduction is desirable.

2

Type 2 diabetes mellitus

GEMINI trial (Bakris et al., JAMA 2004): carvedilol improved insulin sensitivity compared with metoprolol in hypertensive diabetic patients, with less worsening of glycaemic control.

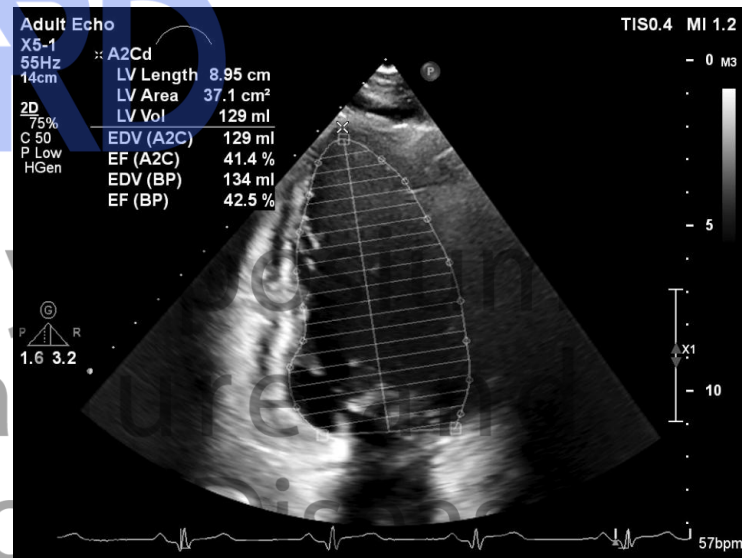
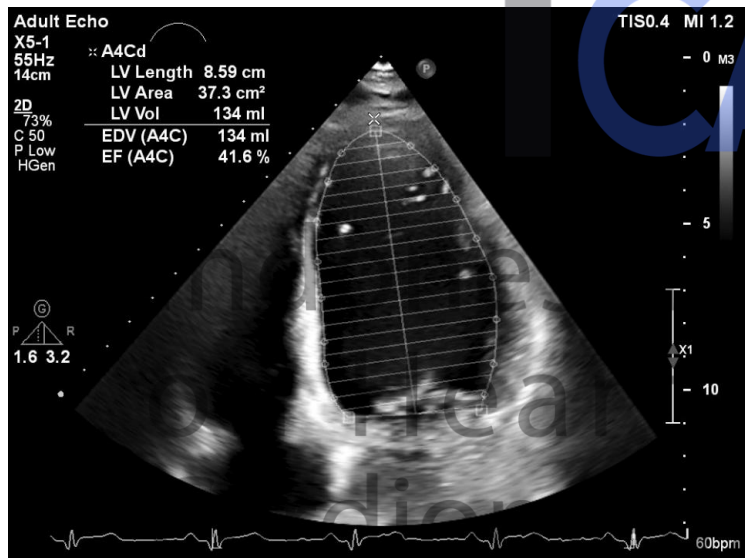
3

Ischaemic cardiomyopathy (anterior OMI)

The **CAPRICORN trial:** carvedilol reduced mortality in post-MI LV dysfunction (HR 0.77). Carvedilol's antioxidant properties and α_1 -blockade may reduce oxidative stress and afterload — both relevant in post-infarct LV remodelling.

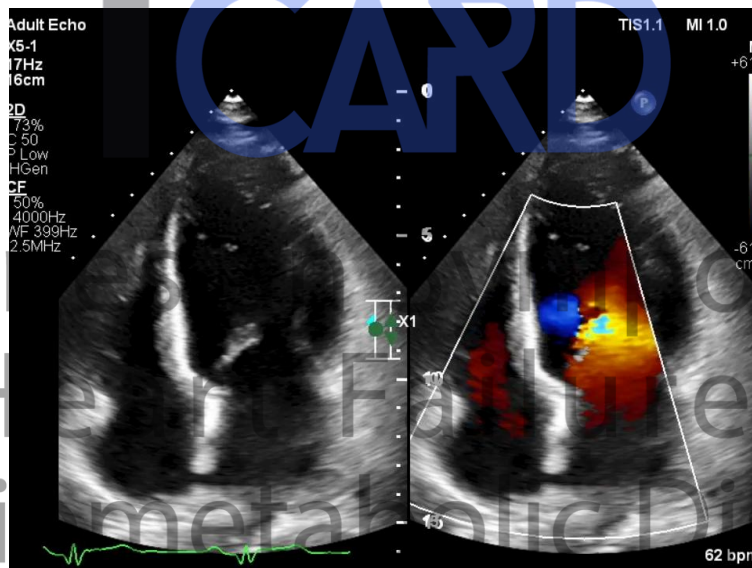
Follow-Up Echo — 6 Months on Optimised GDMT

Carvedilol uptitrated to target · ARNI · MRA · SGLT2i · NYHA Class I



Follow-Up Echo — 6 Months on Optimised GDMT

Carvedilol uptitrated to target · ARNI · MRA · SGLT2i · NYHA Class I



Q4

6 months later: NYHA Class I, feels well. Repeat echo: EF 43% (was 28%). Based on the universal definition of HF, what category does this patient now belong to?

A

HFpEF — heart failure with preserved EF

B

HFmrEF — heart failure with mildly reduced EF

C

HFimpEF — heart failure with improved EF

D

Heart failure recovered — no longer classified

Q4 ✓ Answer C: HFimpEF — heart failure with improved EF

Discussion

Definition of HFimpEF (all criterias required):

1. Baseline LVEF $\leq 40\%$ (i.e. previously HFrEF)
2. ≥ 10 -point absolute increase in LVEF from baseline
3. Second LVEF measurement $> 40\%$

This patient meets all three:

- Baseline EF 22% ($\leq 40\%$ ✓)
- Improvement of 24 points (≥ 10 ✓)
- Current EF 46% ($> 40\%$ ✓)

Q5

Now that the patient meets criteria for HFimpEF and feels well, what is the most appropriate decision regarding his medications?

A

Slowly stop GDMT one drug at a time, starting with the beta-blocker

B

Stop everything

C

Switch to HFpEF treatment protocol

D

Continue all four GDMT pillars indefinitely

Q5 ✓ Answer D: Continue all four GDMT pillars indefinitely

Discussion

HFimpEF is remission, NOT cure.

TRED-HF trial*:

Patients with dilated CMP whose EF had recovered to >50% on GDMT were randomised to continue or progressively withdraw their medications.

Results:

- 40% of those who stopped GDMT relapsed within 6 months
- Patients who continued GDMT did not relapse
- Long-term follow-up: >65% met relapse criteria over 6+ years, even those re-started on low-dose GDMT after the trial

**Halliday et al., Lancet 2019.*

Key Teaching Pearls — Case 1

1. Echo as Diagnostic Anchor

Clinical findings raise the suspicion; echo findings confirm HFrEF and trigger the GDMT pathway.

2. Echo Tracks Therapy Response

LV size, EF, and functional MR included, all change with effective GDMT. Serial echo measures whether your therapy is working — and how to continue optimisation.

3. Echo Defines HFimpEF

The 6-month echo follow-up meets the Universal Definition criteria for HFimpEF → your evidence to continue all four pillars indefinitely (TRED-HF).

iHEF CARD

CASE 2

Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease

☎ 0811-1900-8855

✉ scientific_ihefcard@inahfcardmet.org

📷 @ina.hf

www.ihefcard.com

11-13 JUNE 2026

Sheraton Grand Jakarta, Gandaria City

CASE ILLUSTRATION

- Patient** Mrs. Y · Female, 67 years old · BMI 33 (obese)
- Chief Complaint** Progressive exertional dyspnea over 3 months — NYHA III
- Associated** Fatigue · Lower-limb swelling · Orthopnea (mild, 2 pillows)
- Denies** Chest pain · Syncope · Recent illness
- Past History** Hypertension (on amlodipin and candesartan) · Type 2 DM



Physical Examination

Vital Signs

BP 158 / 92 mmHg

HR 92 bpm · regular

RR 20 / min

SpO₂ 94% on Room Air

Clinical Findings

- Mildly raised JVP
- Bibasal fine crackles
- Trace pitting oedema (ankles)

Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease

ECG



Indonesian Symposium on Heart Failure and Cardiometabolic Disease

SR · LVH by voltage criteria · No acute ischaemic changes



Chest X-Ray



Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease

Mild cardiomegaly with aortic elongation



Lab Results

NT pro BNP

100 pg/mL

Na⁺

139 mEq/L

Creatinine

1.1 mg/dL

eGFR

58 mL/min

Troponin I

<0.03 ng/mL

HbA1c

7.8%

Q1

Seeing her demographic profile (older woman, obese, hypertensive, diabetic), before sending her for echo, you apply the H2FPEF score (clinical-only pre-test probability for HFpEF). What is her score and what does it mean?

A

Score 0–1 — low probability, HFpEF excluded

B

Score 2–5 — intermediate, echo + further testing needed

C

Score 6 — high probability, HFpEF very likely

D

Score cannot be calculated without invasive haemodynamics



Discussion

	Clinical Variable	Values	Points
H₂	H heavy	Body mass index > 30 kg/m ²	2
	H ypertensive	2 or more antihypertensive medicines	1
F	Atrial F ibrillation	Paroxysmal or Persistent	3
		Doppler Echocardiographic estimated	

Score range 0–9:

• Low: 0–1

Q1 ✓ Answer B: H2FPEF score 2–5 — intermediate, echo + further testing needed

F	F illing Pressure	Doppler Echocardiographic E/e' > 9	1
H₂FPEF score			Sum (0-9)
Total Points 0 1 2 3 4 5 6 7 8 9 Probability of HFpEF 0.2 0.3 0.4 0.5 0.6 0.7 0.8 0.9 0.95			

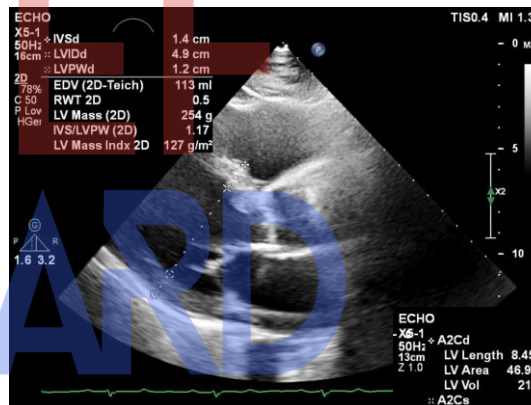
needed)

• High: 6–9 (HFpEF very likely)



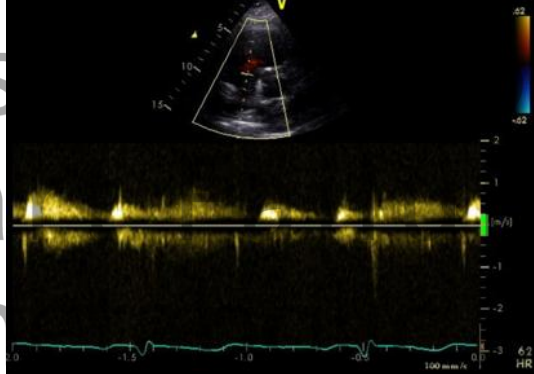
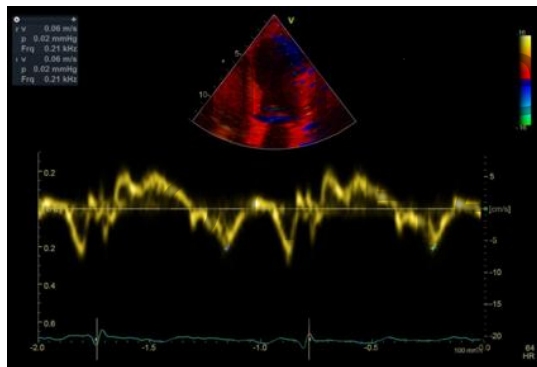
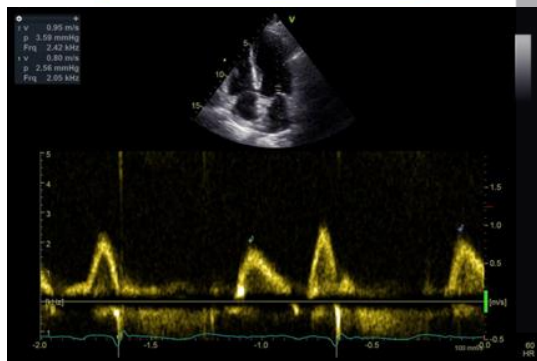
Resting Echo

- Dilated LA (LAVI 42 ml/m²)
- Concentric LVH (LVMI 127 gr/m², RWT 0.5)
- Good LV systolic function, EF 61%
(Simpson's Biplane)
- Global normokinetic



How about the diastolic function?

- Normal RV contractility, TAPSE 1.7 cm



Resting Diastolic Findings

E wave: 91 cm/s

e' med: 7 cm/s

e' lat: 6 cm/s

E/e' avg: 13.5

TR Vmax: undetected

H2FPEF SCORE

	Clinical Variable	Values	Points
H ₂	Heavy	Body mass index > 30 kg/m ²	2
	Hypertensive	2 or more antihypertensive medicines	1
F	Atrial Fibrillation	Paroxysmal or Persistent	3
P	Pulmonary Hypertension	Doppler Echocardiographic estimated Pulmonary Artery Systolic Pressure > 35 mmHg	1
E	Elder	Age > 60 years	1
F	Filling Pressure	Doppler Echocardiographic E/e' > 9	1
H ₂ FPEF score			Sum (0-9)
Total Points			
Probability of HFpEF			

Score range 0–9:

- Low: 0–1
- Intermediate: 2–5 (further testing needed)
- High: 6–9 (HFpEF very likely)

Q2

**Apply the HFA-PEFF score (the echo + biomarker-based diagnostic algorithm).
Based on the echo findings and her NT pro BNP of 100 pg/mL, what is her HFA-PEFF
score, and what is the conclusion?**

A

Score 0–1 — HFpEF unlikely

B

Score 2–4 — intermediate,
consider further testing

C

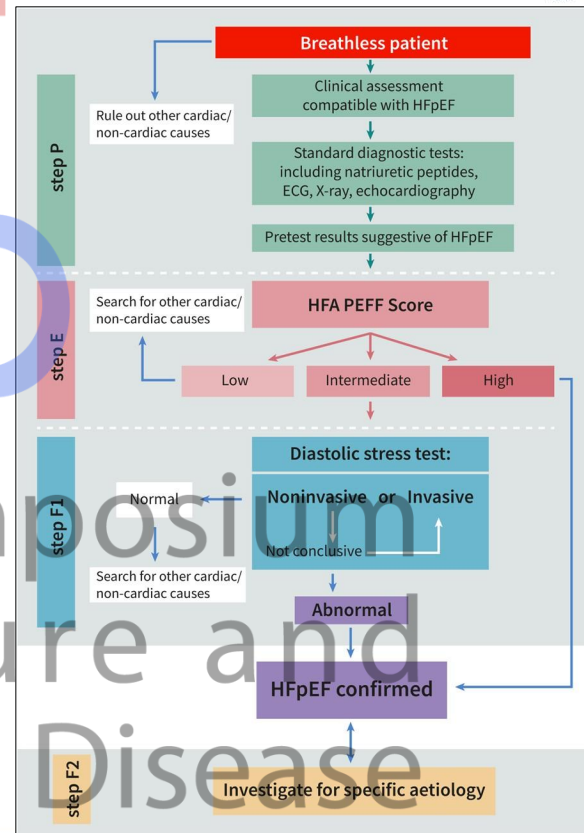
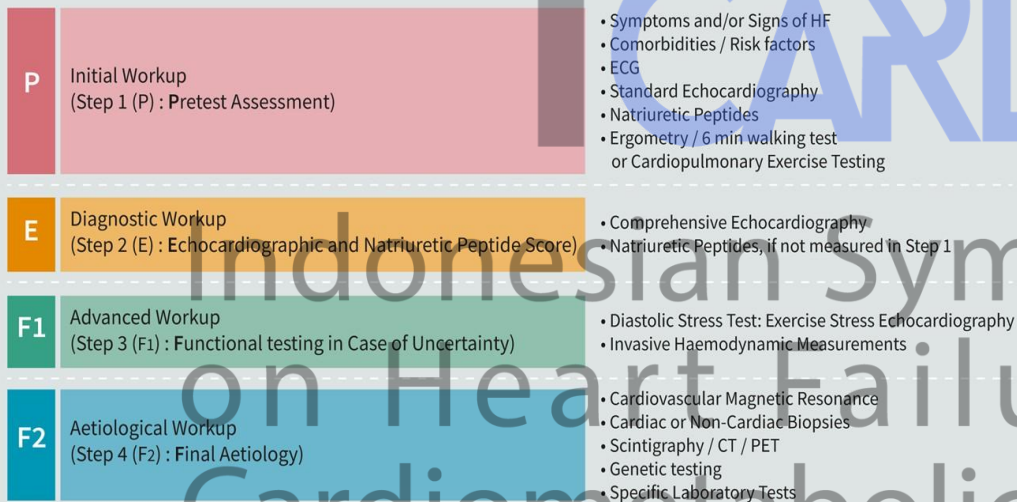
Score 5–6 — HFpEF confirmed

D

HFA-PEFF cannot be used in this
scenario

HFA-PEFF Diagnostic Algorithm

The HFA-PEFF Algorithm for the Diagnosis of HFpEF



HFA-PEFF Diagnostic Algorithm: Step 2 (E)

	Functional	Morphological	Biomarker (SR)	Biomarker (AF)
Major	septal $e' < 7$ cm/s or lateral $e' < 10$ cm/s or Average $E/e' \geq 15$ or TR velocity > 2.8 m/s (PASP > 35 mmHg)	LAVI > 34 ml/m ² or LVMI $\geq 149/122$ g/m ² (m/w) and RWT > 0.42 #	NT-proBNP > 220 pg/ml or BNP > 80 pg/ml	NT-proBNP > 660 pg/ml or BNP > 240 pg/ml
Minor	Average $E/e' 9-14$ or GLS $< 16\%$	LAVI $29-34$ ml/m ² or LVMI $> 115/95$ g/m ² (m/w) or RWT > 0.42 or LV wall thickness ≥ 12 mm	NT-proBNP $125-220$ pg/ml or BNP $35-80$ pg/ml	NT-proBNP $365-660$ pg/ml or BNP $105-240$ pg/ml
Major Criteria: 2 points		≥ 5 points: HFpEF		
Minor Criteria: 1 point		2-4 points: Diastolic Stress Test or Invasive Haemodynamic Measurements		

Echo parameters:

- Septal and lateral e'
- Average E/e'
- TR Velocity (PASP)
- LAVi
- LVMI and RWT
- GLS
- LV wall thickness

How the HFA-PEFF score works (max 6 points — score ≥ 5 confirms HFpEF)

F — Functional	M — Morphological	B — Biomarker
Major (+2) e' septal < 7 OR lateral < 10 E/e' avg ≥ 15 $TRV > 2.8$ m/s Minor (+1) E/e' avg 9–14 $GLS < 16\%$ (less negative)	Major (+2) $LAVi > 34$ (SR) or > 40 (AF) $LVMI \geq 149/122$ (M/F) or $RWT > 0.42$ Minor (+1) $LAVi$ 29–34 $LVMI$ 115–149/95–122	Major (+2) $NT-proBNP > 220$ (SR) / > 660 (AF) or $BNP > 80$ (SR) / > 240 (AF) Minor (+1) $NT-proBNP$ 125–220 (SR) / 365–660 (AF) or BNP 35–80 / 105–240
Major (+2): e' lateral 6 → +2 points	Major (+2): $LAVi$ 42 (SR) → +2 points	None → 0 points

Total HFA-PEFF score: $2 + 2 + 0 = 4 \rightarrow$ HFpEF not yet CONFIRMED

Q2 ✓ Answer B: Score 2–4 — Intermediate

Q3

What is the NEXT BEST step to confirm or exclude HFpEF?

A

Empirical HF treatment without further testing

B

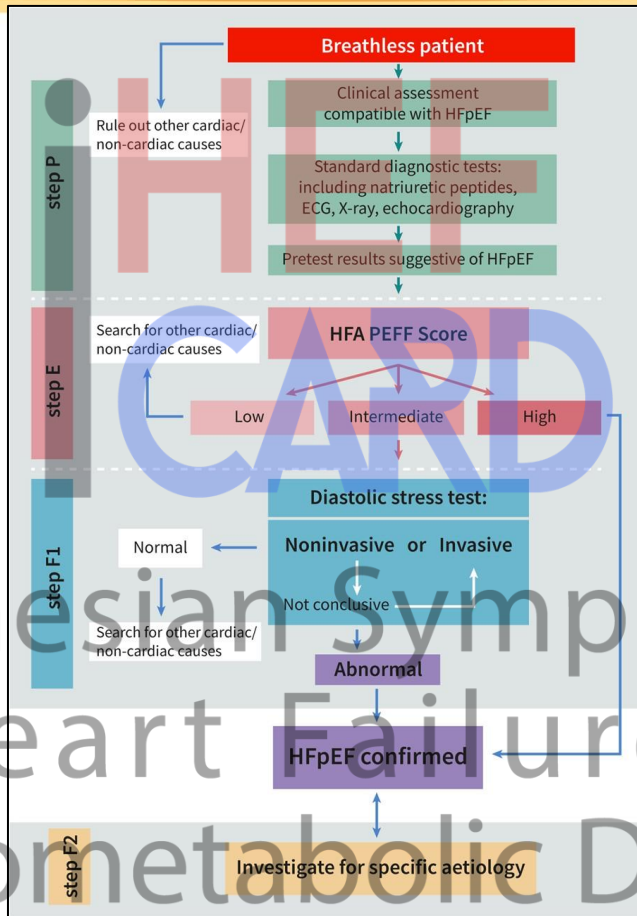
Repeat resting echo in 6 months

C

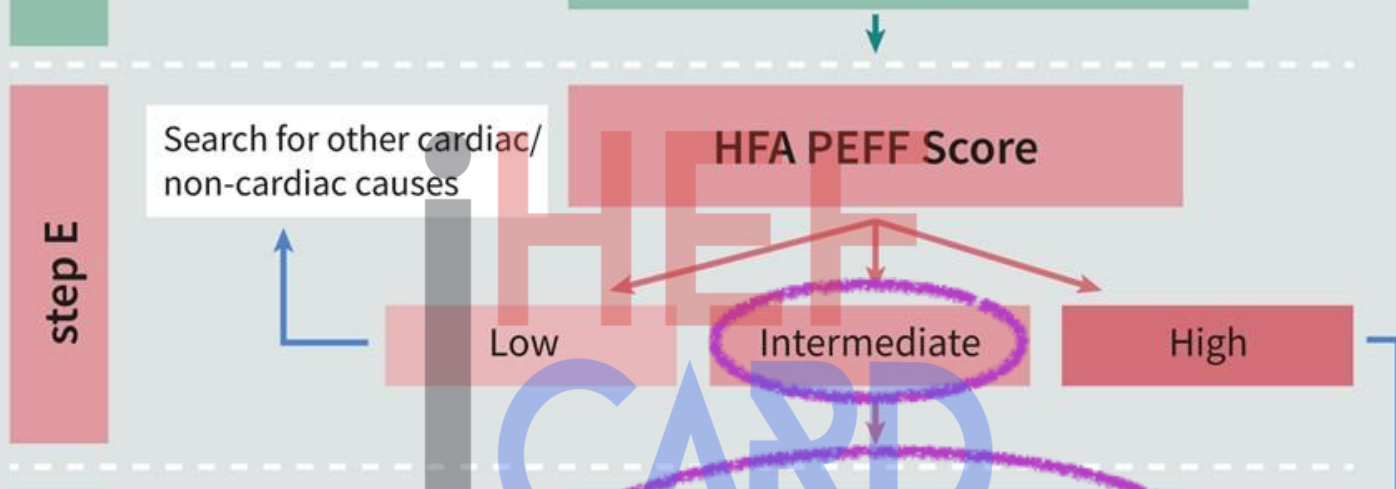
Cardiac MRI for tissue characterisation

D

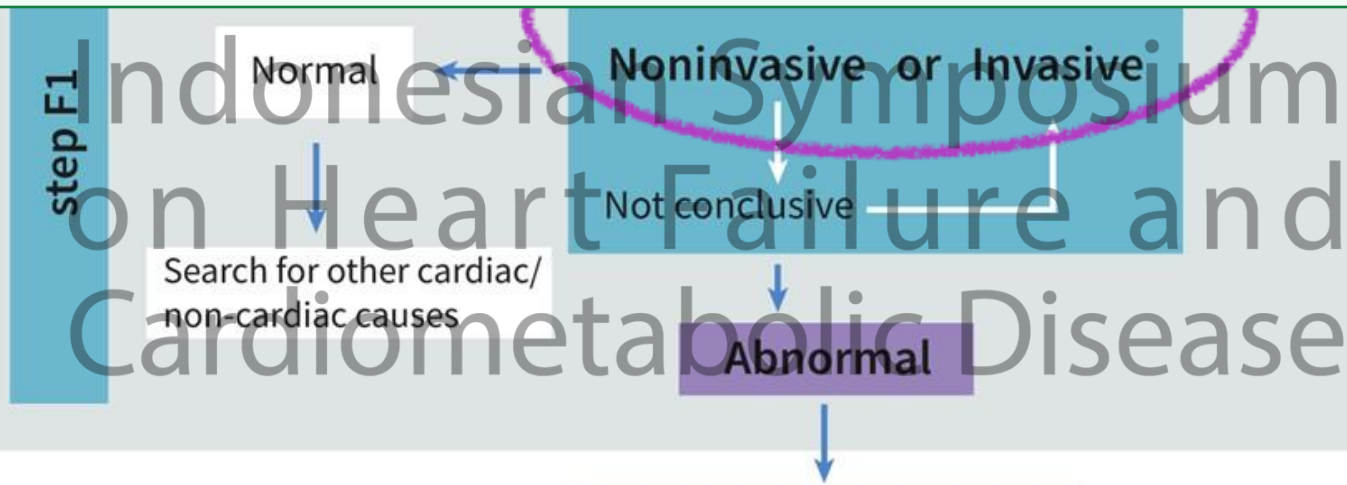
Diastolic stress echo (exercise echo with E/e' + TRV assessment)



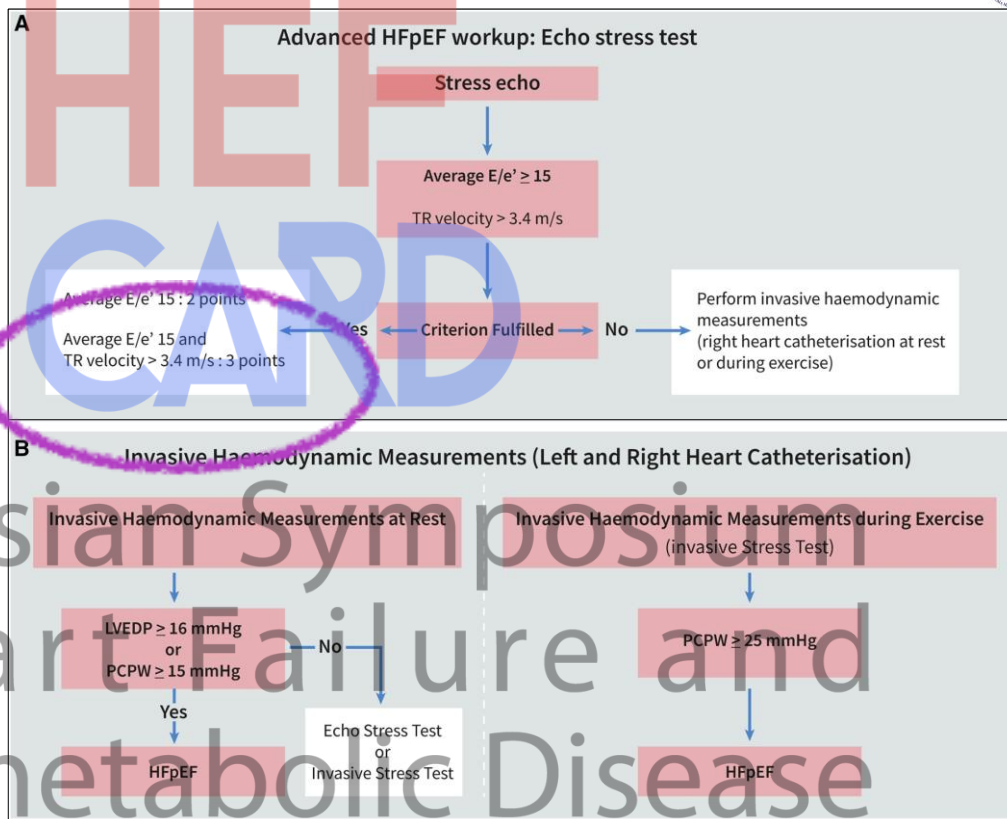
Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease



Q4 ✓ Answer D: Diastolic stress echo — exercise echocardiography



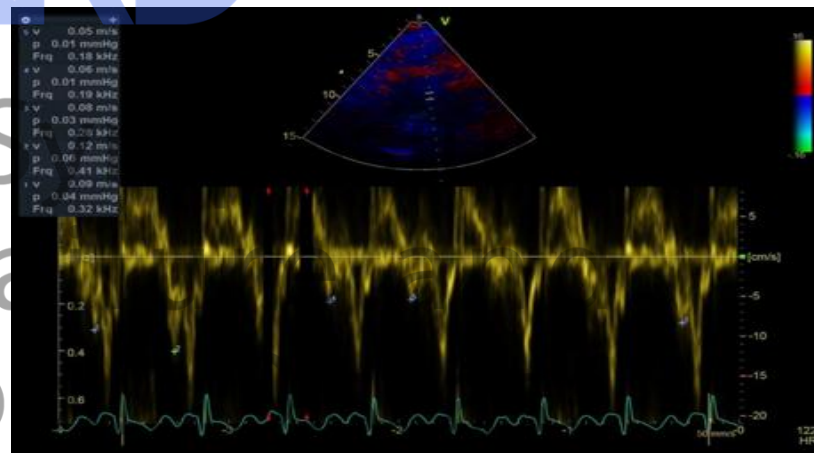
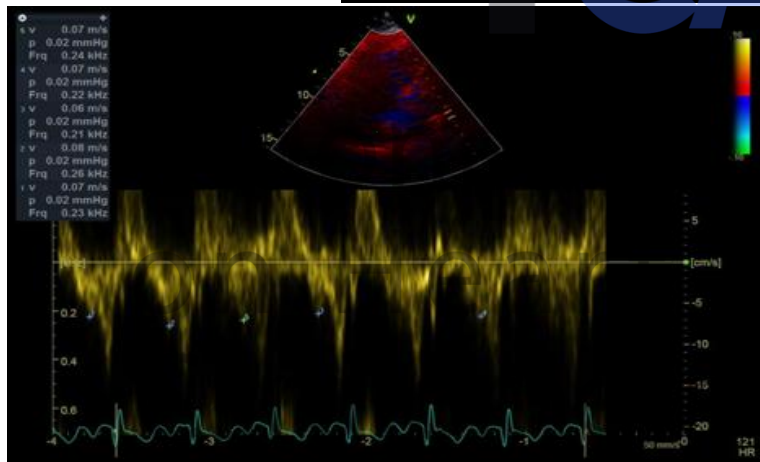
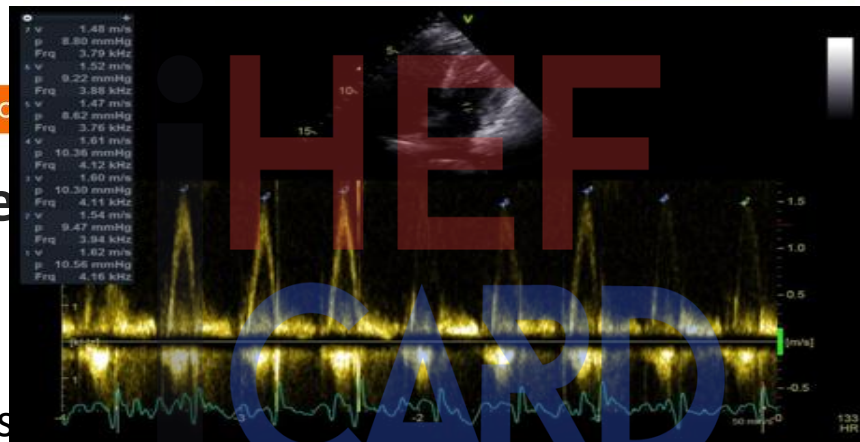
HFA-PEFF Diagnostic Algorithm: Diastolic Stress Test



Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease

Diastolic strain rate and strain

- Peak exercise: 10.56 mmHg
- Ended due to s



How the HFA-PEFF score works (max 6 points — score ≥ 5 confirms HFpEF)

F — Functional	M — Morphological	B — Biomarker
Major (+2) e' septal <7 OR lateral <10 E/e' avg ≥15 TRV >2.8 m/s Minor (+1) E/e' avg 9–14 GLS <16% (less negative) Major (+2): e' lateral 6 → +2 points	Major (+2) LAVi >34 (SR) or >40 (AF) LVMI ≥149/122 (M/F) or RWT >0.42 Minor (+1) LAVi 29–34 LVMI 115–149/95–122 Major (+2): LAVi 42 (SR) → +2 points	Major (+2) NT-proBNP >220 (SR) / >660 (AF) or BNP >80 (SR) / >240 (AF) Minor (+1) NT-proBNP 125–220 (SR) / 365–660 (AF) or BNP 35–80 / 105–240 None → 0 points
Diastolic stress test	Average E/e' ≥ 15 → +2 points	
Total HFA-PEFF score: 2 + 2 + 0 + 2 = 6 → HFpEF CONFIRMED		

Q4

HFpEF is now confirmed. Aside from optimising her comorbidities (BP, weight, diabetes), which medication has Class I evidence for reducing HF hospitalisation in HFpEF and should be started?

A

ARNI (sacubitril/valsartan) —
Class I in HFpEF

B

Beta-blocker — Class I in HFpEF
for mortality

C

SGLT2 inhibitor (dapagliflozin or
empagliflozin)

D

Loop diuretic only — no
disease-modifying therapy exists
for HFpEF

Recommendation

Class^a

Level^b

An SGLT2 inhibitor (dapagliflozin or empagliflozin) is recommended in patients with HFpEF to reduce the risk of HF hospitalization or CV death.^{c 6,8}

I

A

© ESC 2023

Q4 ✓ Answer: SGLT2 inhibitor

HFpEF Treatment

SGLT2i

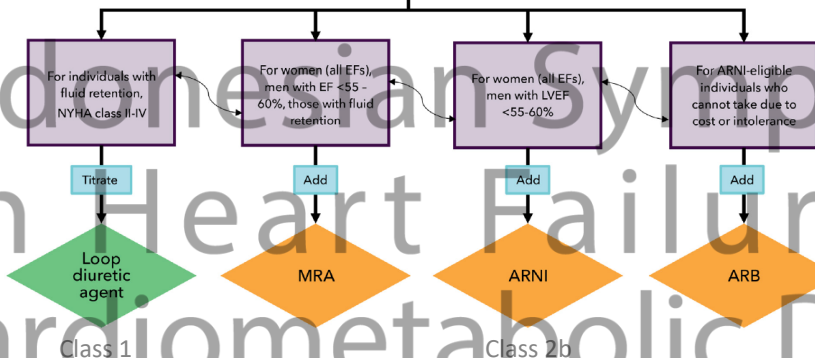
Class 2a

hospitalisation by 21% (HR

18% (HR 0.82) in

Discussion

- **EMPEROR-Prese** (HR 0.79) in HFpEF.
- **DELIVER** — dapagliflozin in HFmrEF/HFpEF, in



ACC/AHA 2022

Anker et al., NEJM 2021
Solomon et al., NEJM 2022

Key Teaching Pearls — Case 2

1. Echo Defines HFpEF — But Clinical Suspicion Comes First

H2FPEF is the clinical pre-test probability. HFA-PEFF is the echo + biomarker confirmation.
Use BOTH — high concordance = highest diagnostic certainty.

2. Echo Diastolic Parameters Are the Diagnostic Currency of HFpEF

$E/e' \text{ avg} \geq 15$, $LAVi > 34$ (or > 40 in AF), $TRV > 2.8 \text{ m/s}$, $\downarrow e'$.
These cannot be assessed at the bedside — echo is irreplaceable for HFpEF diagnosis.

3. Resting Echo Equivocal? Stress Echo Unmasks HFpEF

Intermediate HFA-PEFF (2–4)? Exercise echo with $E/e' \geq 15$ OR $TRV > 3.4$ at peak confirms HFpEF.
Invasive exercise haemodynamics is the gold standard.

iHEF CARD

CASE 3

Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease

☎ 0811-1900-8855

✉ scientific_ihefcard@inahfcardmet.org

📷 @ina.hf

www.ihefcard.com

11-13 JUNE 2026

Sheraton Grand Jakarta, Gandaria City

Clinical Vignette

Patient

Mrs. Z · Female, 64 years old

Diagnosis

HFrEF (EF 30%) with normal
RV function, diagnosed 8
months ago · started on full
GDMT

Past Follow-up

Last clinic visit 5 months ago — lost to follow-up since

Current Complaint

Worsening fatigue, exertional dyspnea (NYHA III), abdominal distension
× 2 months

Patient Reports

Still taking medications regularly



Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease

Physical Examination

Vital Signs

BP	124 / 78 mmHg
HR	118 bpm · Irregularly Irregular
RR	20 / min
SpO ₂	96% on Room Air
Weight	Up 6 kg from last visit

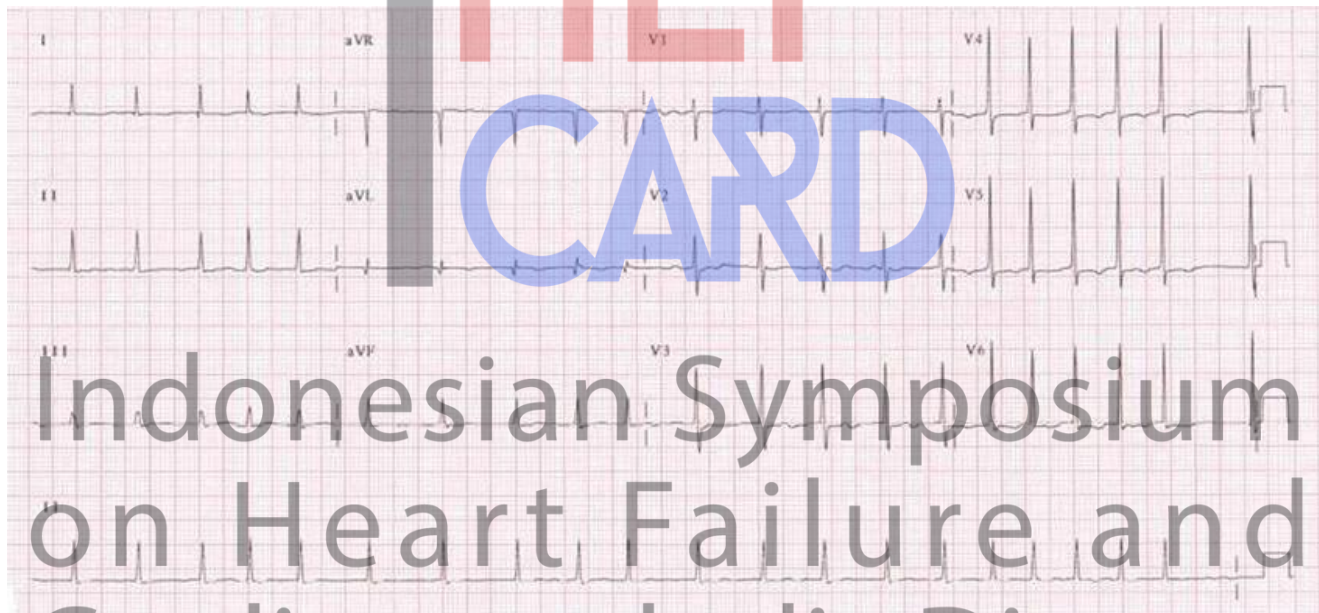
Clinical Findings (Right-Sided Predominant)

- Raised JVP ~12 cmH₂O
- Hepatomegaly + positive hepatojugular reflux
- Mild ascites + early satiety
- 3+ pitting oedema to thighs
- Lungs relatively clear

Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease



ECG



Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease

Atrial fibrillation · LVH · No acute ischaemic changes

Q1

Patient is now in newly-detected AF with right-sided congestion despite reportedly adherent GDMT. What is the MOST APPROPRIATE next diagnostic step before adjusting therapy?

A

Increase loop diuretic dose empirically
— she is congested

B

Repeat echo with a COMPLETE RV
assessment (not just TAPSE)

C

Refer urgently for LV assist device
evaluation

D

Order CT pulmonary angiogram to rule
out PE

Q1 ✓ Answer B: Repeat echo with a COMPLETE RV assessment — not just TAPSE

Discussion

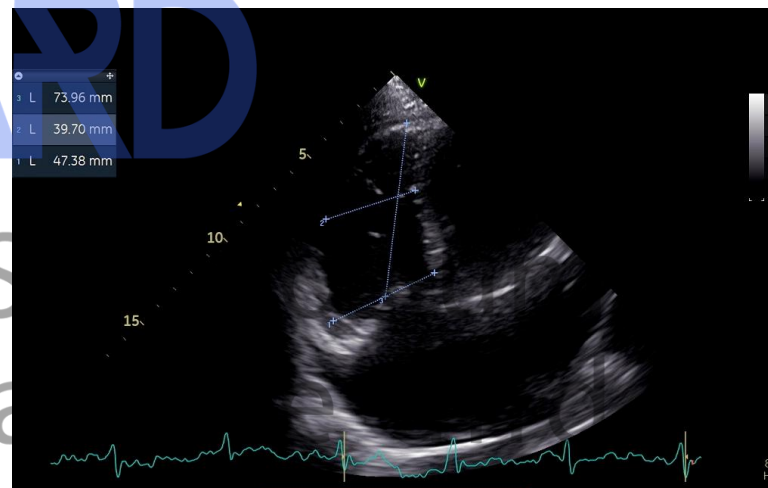
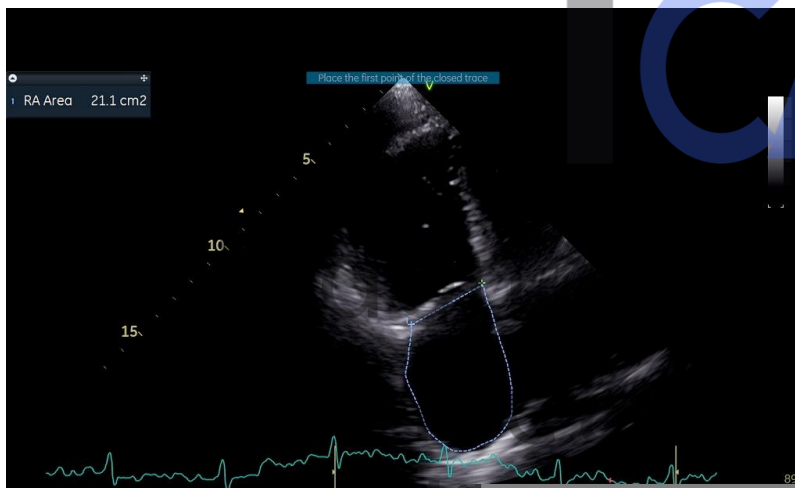
Right-sided congestion, AF, abdominal distension, but clear lungs → clinical picture of RIGHT heart failure, even though the LV was the original problem.

Clinical Reasoning

Echo is the diagnostic anchor that distinguishes:

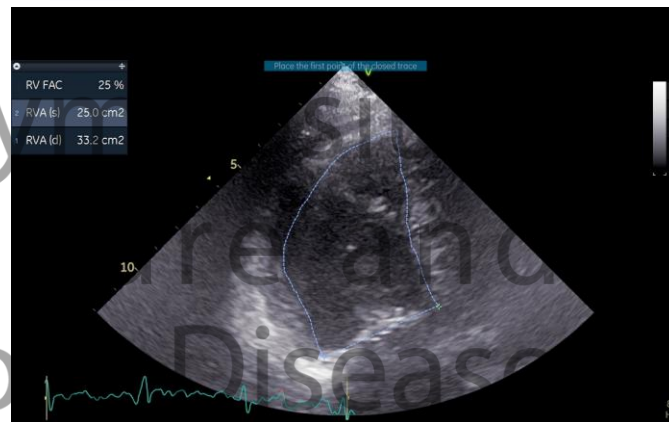
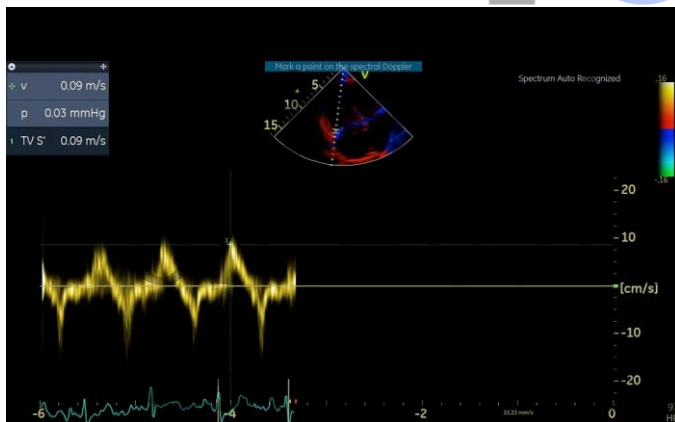
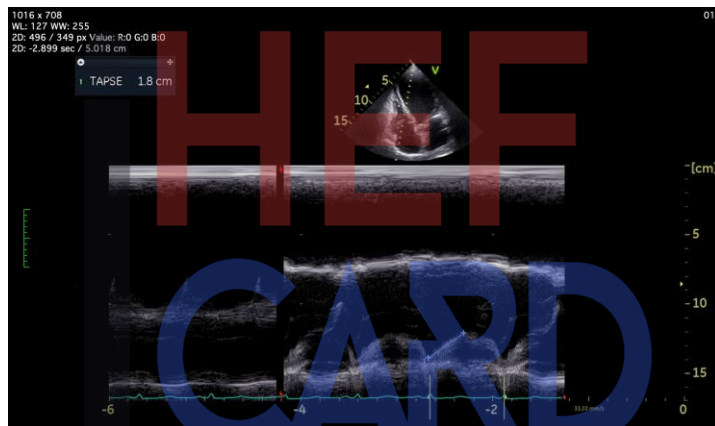
- Left-sided recurrence (suggest non-adherence or LV deterioration)
- Right-sided dominant problem (suspected here)
- Other specific culprits (AF, PH, RV ischaemia, TR progression)

Echo Findings



Cardiometabolic Disease

Echo Findings



Echo Findings Summary

LVEF	38% (improved from 30%)	TAPSE	18 mm (>17 = "normal")
RV FAC	25% (<35% abnormal)	RV FWLS	Not performed
RV basal diameter	47 mm (>41 dilated)	RA area	21 mL/m ² (>18 abnormal)
TR severity	Severe (VC 7.5 mm, EROA 0.45)	TRV / PASP	3.6 m/s · PASP 60 mmHg
TAPSE / PASP	18 / 60 = 0.30	IVC	24 mm, <50% collapse

Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease

Q2

Patient has raised JVP, hepatic congestion, ascites, peripheral oedema, IVC 24 mm. BP 124/78. What is the SAFEST fluid management approach?

A

IV furosemide 80 mg bolus immediately — she is congested

B

Start dobutamine to support cardiac output before any diuresis

C

Withhold diuresis — she may need fluid resuscitation given the RV dysfunction

D

Cautious IV diuresis with close monitoring of BP, urine output, and RV preload markers

Q2 ✓ Answer D: Cautious IV diuresis with close monitoring of BP, urine output, and RV preload markers

Discussion

Why NOT aggressive diuresis:

- The dysfunctional RV is highly **PRELOAD-DEPENDENT** — it relies on **higher filling pressure** to generate adequate forward output
- Over-diuresis → RV **preload collapses** → RV **stroke volume drops** → systemic hypotension

Why NOT under-diuresis:

- **Persistent venous congestion** drives hepatic dysfunction, gut oedema, diuretic resistance, and worsens TR via RV stretch
- Congestion itself is a **poor prognostic marker**

Q3

TAPSE/PASP ratio is 0.30 (severely uncoupled — <0.36). She is in uncontrolled AF at HR 118. What should be the THERAPEUTIC PRIORITY for improving RV-PA coupling?

A

Add sildenafil to reduce pulmonary vascular resistance

B

Restore AF rate control — and consider rhythm control if persistent

C

Increase ARNI dose to further reduce LV filling pressures

D

Add ivabradine for heart rate reduction

Q3 ✓ Answer B: Restore AF rate control — and consider rhythm control if persistent

Discussion

RV-PA uncoupling (TAPSE/PASP <0.36) → the RV cannot generate enough force to overcome its afterload.

AF and RV-PA uncoupling:

- Tachycardia (HR 118) → shortens diastolic filling → raises LV diastolic + LA pressure → raises PASP
- Loss of atrial kick → reduced RV filling → reduced forward output
- Atrial functional TR (from RA dilation) → RV volume overload → progressive dilation
- Tachycardia-mediated cardiomyopathy can directly impair RV contractility

Q3 ✓ Answer B: Restore AF rate control — and consider rhythm control if persistent

Discussion

Rate control:

- Restart beta-blocker carefully, OR digoxin
- Avoid non-DHP CCBs (verapamil, diltiazem) — **contraindicated** in HFrEF
- Target HR ≤ 110 at rest initially

Rhythm control:

- **CASTLE-AF**: ablation reduced death/HF admission by 38% in AF + HFrEF
- **CASTLE-HTx**: ablation reduced death/LVAD/transplant by 76% in advanced HF + AF
- Strong consideration for ablation referral once stabilised

Q4

You decide on rate control. Patient is on dapagliflozin, sacubitril/valsartan, spironolactone, and carvedilol (per chart — but she has been off for ~5 months). BP 124/78, HR 118 (AF). Which is the SAFEST initial step?

A

Restart carvedilol at 3.125 mg BID (lower than her previous dose)

B

Add diltiazem 120 mg daily for rapid rate control

C

Load IV amiodarone 150 mg then 1 mg/min infusion

D

Add digoxin 0.125 mg daily and reassess

Q4 ✓ Answer A: Restart carvedilol at 3.125 mg BID (lower than previous dose)

Discussion

Restarting beta-blockers requires the same caution as initiating from scratch →
“Start low, go slow”

Why beta-blocker is the right choice:

- Beta-blocker = Class I for HFrEF (mortality benefit) **AND** first-line for AF rate control in HFrEF
- Carvedilol is appropriate (her original drug) — antioxidant + α_1 effects useful given likely persistent BP issues + diabetes

Q5

**Patient successfully undergoes AF ablation, is in sinus rhythm, and clinically improved.
What is the most appropriate echo follow-up strategy?**

A

Single echo at 12 months —
TAPSE only

B

Serial echoes with FULL RV
protocol — track FAC, FWLS,
TAPSE/PASP, TR, RA size

C

No further echo needed since
she is now in sinus rhythm

D

Echo only if symptoms recur

Q5 ✓ Answer B: Serial echoes with FULL RV protocol — track FAC, FWLS, TAPSE/PASP, TR, RA size

Discussion

Why full RV protocol and not just TAPSE:

- TAPSE **recovers earlier** than other parameters → can be misleadingly reassuring
- **True RV functional recovery** requires improvement in FAC, FWLS, RV size, RA size, and TR severity
- TAPSE/PASP (RV-PA coupling) is the **most prognostically important** parameter to track

Key Teaching Pearls — Case 3

1. Don't Forget the Right Side — and Don't Trust TAPSE Alone

Single-parameter assessment is the #1 RV blind spot.

Always combine TAPSE + S' + FAC + FWLS + RV size + TAPSE/PASP.

2. AF + Tachycardia is a Top Cause of RV Decompensation in Optimised HFrEF

AF reduces RV filling (lost atrial kick + tachycardia + atrial functional TR), worsens PH, and drives RV-PA uncoupling.

Rate/rhythm control before reaching for pulmonary vasodilators.

3. TAPSE/PASP <0.36 is the Actionable Echo Parameter for Management

Defines RV-PA uncoupling — both prognostic and therapeutically targetable.

Reduce afterload (treat cause of high PASP) rather than push RV harder.

Thank you

Indonesian Symposium
on Heart Failure and
Cardiometabolic Disease