The 3rd Indonesia Symposium on Heart Failure and Cardiometabolic Disease 2023

# Finding The Best Way to Cope with HFrEF – Advanced Kidney Disease: Personalized Drug Management


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POKJA INAHF-CARMET


2023

# GOALS

1

Improve understanding of benefits of GDMT in HF w/ CKD

2

Improve understanding of acute & chronic hemodynamic e-GFR changes

3

Improve multidisciplinary and personalized care


Bagaimana penatalaksanaan pasien HF + CKD di RS sejawat?

a. SpJP dan SpPD/nefrolog rawat bersama, namun blm ada komunikasi langsung
b. SpJP dan SpPD/nefrolog rawat bersama, membuat keputusan bersama dan bahkan berkomunikasi langsung
c. Ditatalaksana SpJP tanpa konsultasi SpPD oleh karena suatu alasan
d. Ditatalaksana SpPD tanpa konsultasi SpJP oleh karena suatu alasan

# Which formula?

# CKD-EPI?

# ault?

## CURRENT CHRONIC KIDNEY DISEASE (CKD) NOMENCLATURE USED BY KDIGO

*CKD is defined as abnormalities of kidney structure or function, present for > 3 months, with implications for health. CKD is clas*
*categ*

**Prognosis o**

**Prognosis of CKD**
**albuminuria categori**

| GFR categories (ml/min/1.73 m²) Description and range | | | | | | |
|---|---|---|---|---|---|---|
| | G1 | Normal | | | | |
| | G2 | Mildly de | | | | |
| | G3a | Mild moderately | | | | |
| | G3b | Modera severely d | | | | |
| | G4 | Severely decreased | 15–29 | | | |
| | G5 | Kidney failure | < 15 | | | |

Green: low risk (if no other markers of kidney disease, no CKD); Yellow: moderately increased risk; Orange: high risk; Red: very high risk.

## Acute Kidney Injury: Rifle Criteria

Renal Dysfunction

| | Serum creatinine | Urine output |
|---|---|---|
| Stage 1 | ≥26 μmol/L (48 hours)<br>≥1.5-1.9 x (7 days) | <0.5ml/kg/hr<br>(>6 hours) |
| Stage 2 | ≥2.0-2.9 x (7 days) | <0.5ml/kg/hr<br>(>12 hours) |
| Stage 3 | ≥354 μmol/L<br>≥3 x reference<br>If on RRT | <0.3ml/kg/hr (>24 hours)<br>Anuric (>12 hours) |

D-EPI formula has the
ch, it is therefore com-
drug dose adjustments.

age) x LBW [kg]
[mg/dL] x 72
Multiply by 0.85

$)^{-1.154}$ x (Age) $^{-0.203}$
1.742 for women
for African ancestry

$\max\left(\frac{SCr}{\kappa}\right)^{-1.209} \times 0.993^{age}$
1.018 for women
for African ancestry
is −0.329 for females and −0.411 for males,
min indicates the minimum of SCr/κ or 1, & max indicates the maximum of SCr/κ or 1

# Definitions in 2016 ESC HF Guidelines

- " CKD is generally defined as an eGFR < 60 mL/min/1.73 m2 and/or the presence of albuminuria (high 30 –300 or very high >300 mg albumin/1 g of urine creatinine)."



- "A further deterioration in renal function, termed worsening renal function (WRF), is used to indicate an increase in serum creatinine, usually by >26.5 mmol/L (0.3 mg/dL) and/or a 25% increase or a 20% drop in GFR."

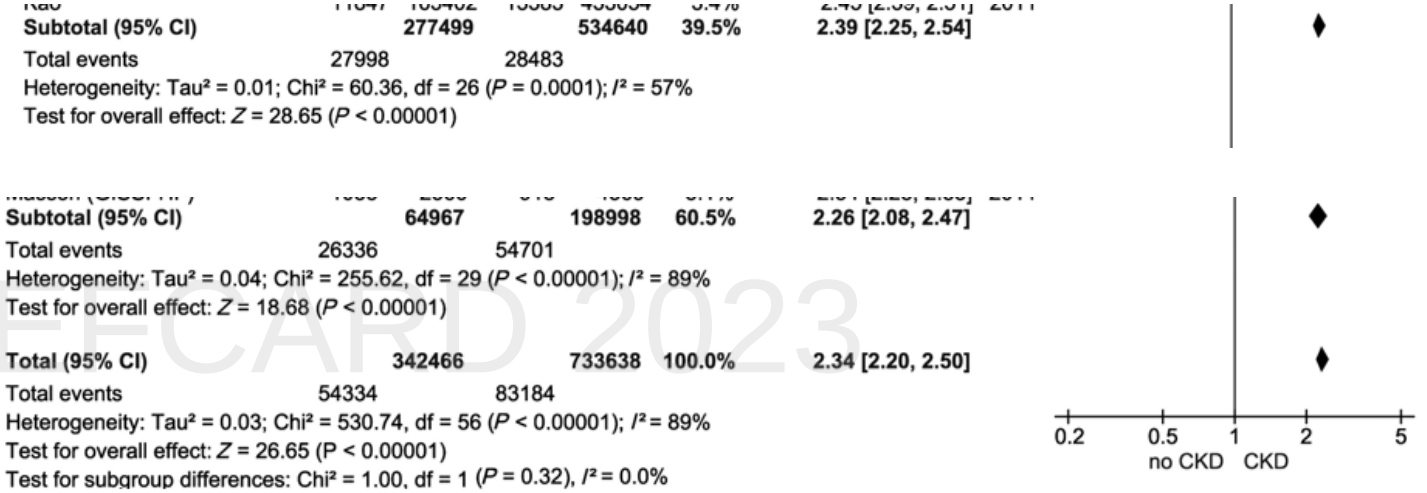
(ESC) Warming Up - Question 1

Which patient has the **worst** prognosis?



A. Geriatric, male, **reduced EF, e-GFR 60**, high NT-pro BNP, overload
B. Geriatric, male, **preserved EF, e-GFR 40**, high NT-pro BNP, overload

- CKD (eGFR < 60) affects 50 % of HF patients
- **CKD = doubling of risk** for all-cause mortality
- CKD = far more **stronger predictor** than LVEF




**High prevalence of CKD in HF (trials, and registries)**
**...and prognostic significance**

Damman K, et all. Renal impairment, worsening renal function, and outcome in patients with heart failure: an updated meta-analysis. *European heart journal*. 2014;35:455-469.

# Challenges in HF- CKD

| | |
|---|---|
| With drug therapy: | **Changes in GFR** |
| | Hyperkalemia |
| | Poor **evidence** in advanced CKD |
| | Impact of CKD in drug pharmacokinetics |
| | Other comorbid condition |



REAL NIGHTMARE?
for patients & doctor

# GDMT Implementation?

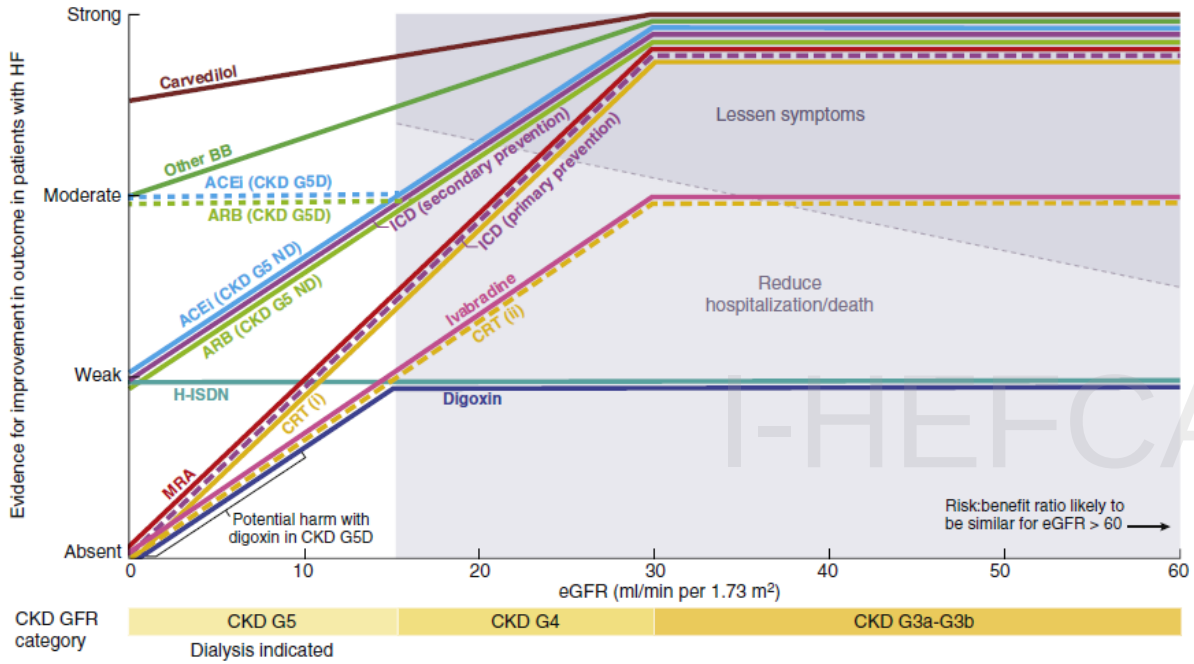
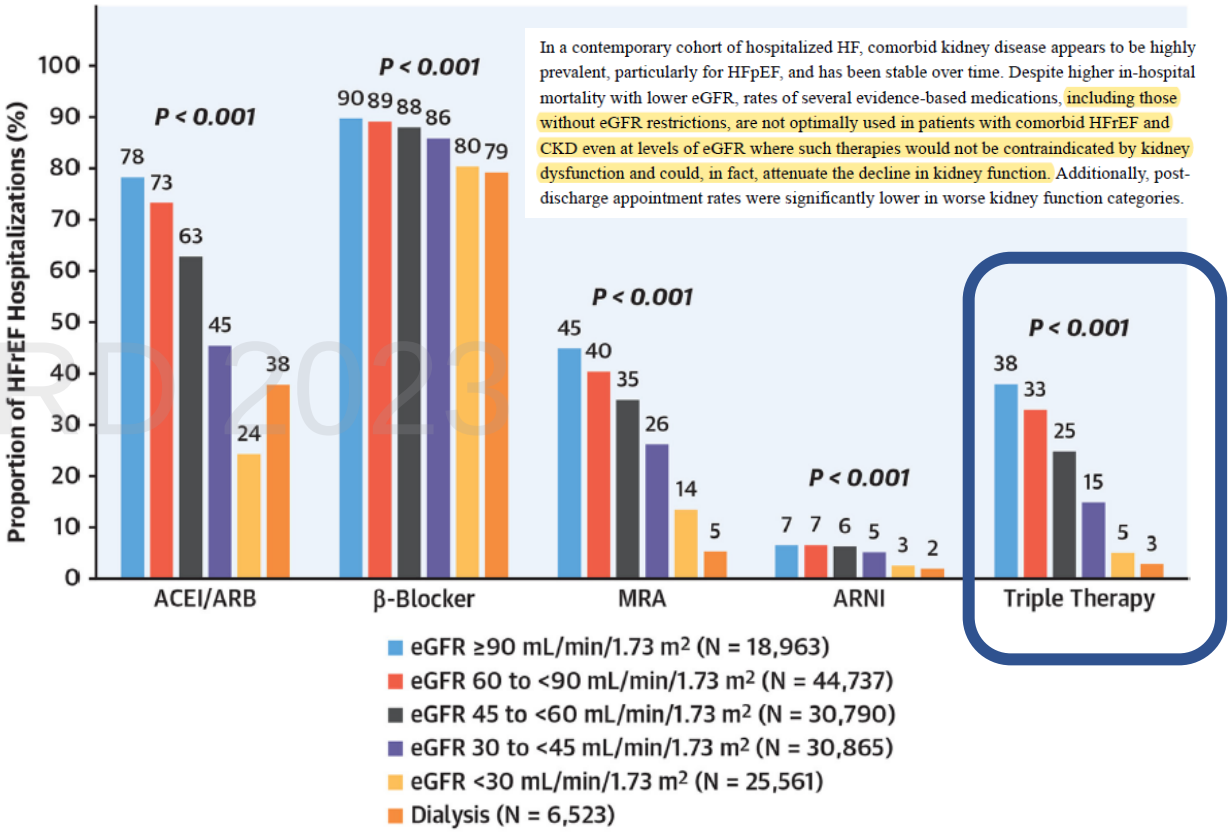



**Figure 5 | Positioning of heart failure (HF) therapies according to left ventricular ejection fraction and renal filtration function.** ACEi angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; BB, β-blocker; CKD G5D, chronic kidney disease glomerular filtratic rate category 5 patient on dialysis; CKD G5 ND, chronic kidney disease glomerular filtration rate category 5 patient not on dialysis; CRT, cardiac resynchronization therapy; EF, ejection fraction; eGFR, estimated glomerular filtration rate; GFR, glomerular filtration rate; H-ISDN, hydralazine-isosorbide dinitrate; ICD, implantable cardioverter-defibrillator; LBBB, left bundle branch block; MRA, mineralocorticoid receptor antagonist.

Patel et al. Page 21

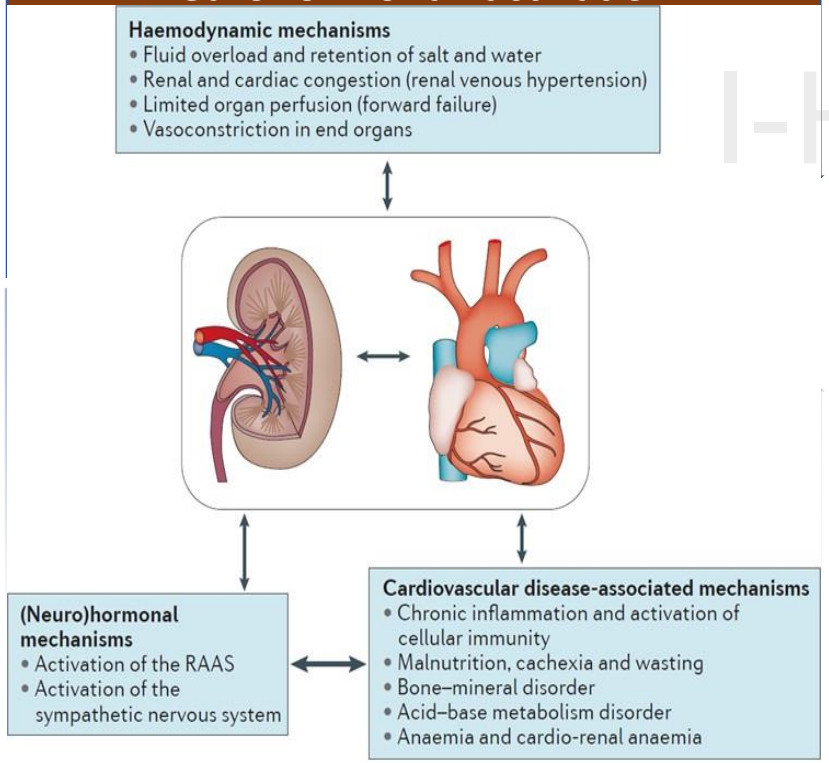
In a contemporary cohort of hospitalized HF, comorbid kidney disease appears to be highly prevalent, particularly for HFpEF, and has been stable over time. Despite higher in-hospital mortality with lower eGFR, rates of several evidence-based medications, including those without eGFR restrictions, are not optimally used in patients with comorbid HFrEF and CKD even at levels of eGFR where such therapies would not be contraindicated by kidney dysfunction and could, in fact, attenuate the decline in kidney function. Additionally, post-discharge appointment rates were significantly lower in worse kidney function categories.




Patel, R.B. et al. J Am Coll Cardiol. 2021;78(4):330-43.

# WHY RENAL FUNCTION IS IMPORTANT?

the organ that is in the end responsible for the maladaptive salt and water retention in response to neurohormonal activation




there where evidence-based treatments in HFREF exert their action **(influence and influenced)**

one of the **strongest** predictors of clinical outcome

# STUMBLE UPON RENAL DYSFUNCTION?

**Table 6.1** Important considerations when approaching a HFREF patient with renal dysfunction

| |
|---|
| *Current situation: Hemodynamics* |
| Is the patient stable? If not, this should be the first treatment goal. |
| Excessive congestion? Evidence of edema? |
| Hypo or hypertensive? |
| *Predisposing conditions that can cause (more than expected) renal impairment* |
| Diabetes mellitus |
| Atherosclerosis |
| Hypertension |
| *Background therapy* |
| Any medical therapy that can compromise renal function? |
| Any medical therapy that is renally cleared? |
| What about HF therapy: what is the current type and dose of evidence based HF therapy, especially RAAS inhibitors? |
| Use of (loop) diuretics? |
| *Dynamics in renal function* |
| What was the course of eGFR/serum creatinine in the past weeks/months? |
| What was the most likely reason for the change? |
| Any indication of organ damage? What about albuminuria (especially in hypertensives, diabetics) |
| *Any indication of adverse events linked to renal dysfunction?* |
| Hyperkaleamia |
| Gout like symptoms |
| Muscle cramps |

Abbreviations: eGFR: estimated Glomerular Filtration Rate, HF: Heart Failure, HFREF: Heart failure with reduced ejection fraction, RAAS: Renin Angiotensin Aldosterone System

# RENAL FUNCTION IN **ACUTE** HEART FAILURE

(Clinical scenario patient admitted with congestion &/ worsening renal function)

# Let me show you the case……

Female, 64 years old w/ HFrEF – Non ICM - DM



2021: Lab **Cr 1.3 (e-GFR 49)**

Tx: ARNI 2 x 25 mg po, Bisoprolol 5 mg, Spironolactone 50 mg, SGLT2-inh, Furosemide prn, insulin

EF 28% → 50% → type C hospital

2023 June: ER with dyspnea, CXR: congestion + pleural effusion + BRPN, minimal pretibial edema, gallop (+)

Tx: Candesartan 16 mg, ISDN 5 mg tid, Amlodipine 5 mg po, Bisoprolol 5 mg, Furosemide 40 mg od, insulin

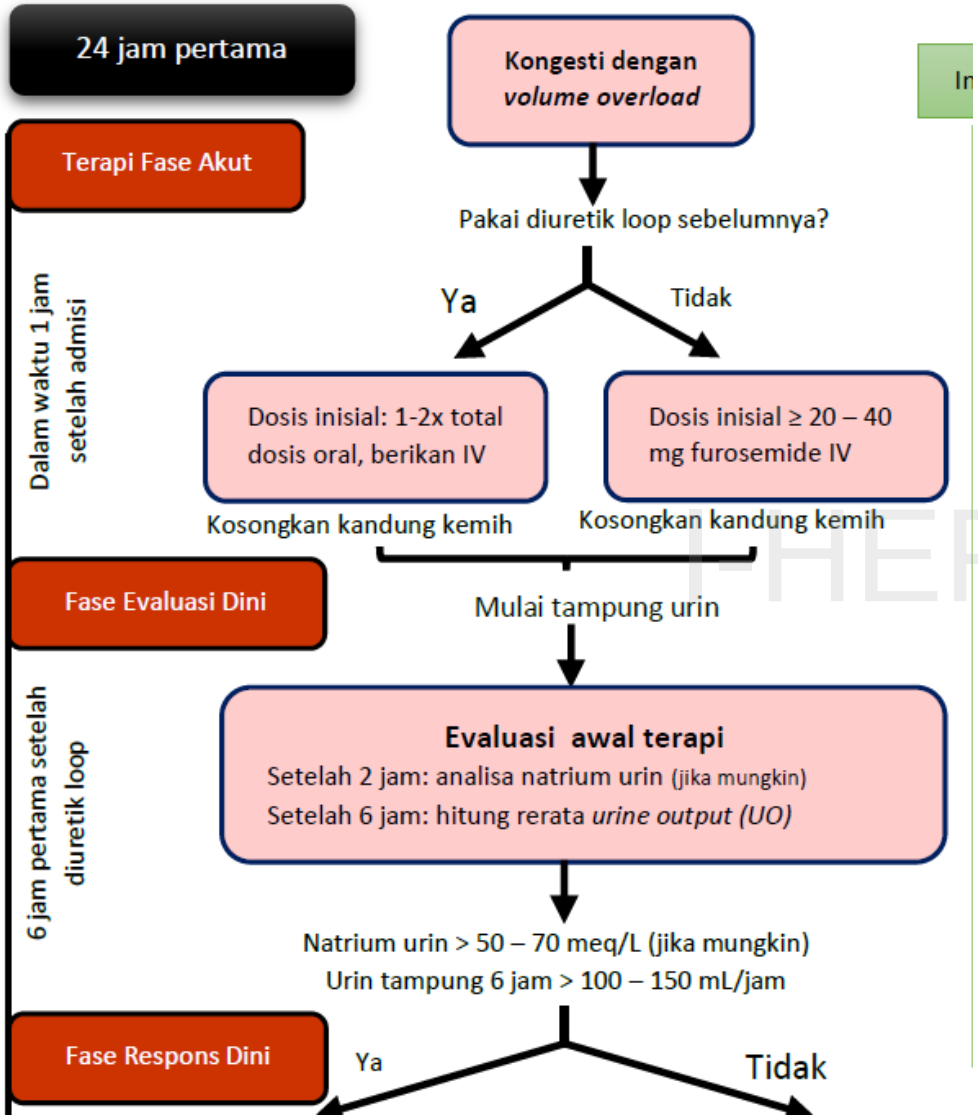
Lab: **Cr 2.3 (e-GFR 29), Na 128, K 4.9**

Question 2

What is your preferred diuretic strategy?



A. Discharge with increased doses of loop diuretics
B. Furosemide 80 mg iv bolus
C. Furosemide 80 mg iv bolus + MRA
D. Else




Good loop diuretic response (HFA position statement) if:

- Urinary sodium concentration > 50–70 meq/L 2 h after diuretic
- Urinary output > 100-150 ml/h during first 6-h after diuretic
- Total diuresis > 3-4 L first 24 h

# IMPORTANT RULES

1) Door to diuretic time
2) Evaluation within 2-6 hours
3) Appropriate dosing according natriuresis/diuresis
4) Only stop when the patient is 'dry'
5) Continue GDMT

**Intervensi Paralel**

(1) Lanjutkan GDMT (2) pertimbangkan untuk menggunakan MRA dini pada kondisi kalium yang rendah, (3) restriksi cairan dan garam, (4) kalium dan magnesium IV dapat diberikan jika diperlukan

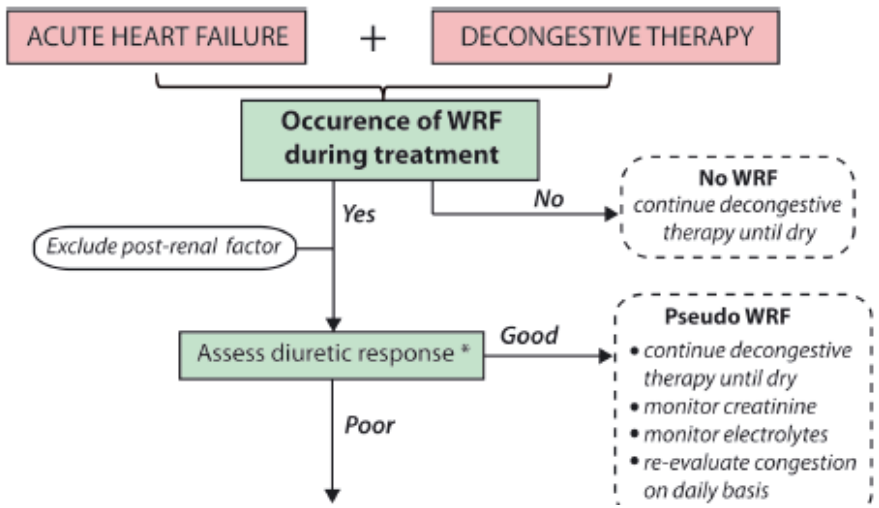
Question 3

Fluid balance in 10 hours: Diuresis 3500 cc

**BUT**, Creatinine goes up! **Cr 2.2 → 3.1** , K 5.4. Which one is true?



A. Evaluate non cardiac causes and evaluate congestion
B. Keep and maintain the dose of ACEi, MRA
C. Halve the dose of ACE-i and MRA
D. Keep the ACE-I and halve the MRA
E. Stop MRA and see nephrologist




**Cr 2.2 → 3.1** , K 5.4.

**MRA**

- Start with a low dose (see above).
- Consider dose up-titration after 4–8 weeks.
- Check blood chemistry at 1 and 4 weeks after starting/increasing dose and at 8 and 12 weeks; 6, 9, and 12 months; 4-monthly thereafter.
- If $K^+$ rises above 5.5 mmol/L or creatinine rises to 221 µmol/L (2.5 mg/dL)/eGFR <30 mL/min/1.73 $m^2$, halve a dose and monitor blood chemistry closely
- If $K^+$ rises to >6.0 mmol/L or creatinine to >310 µmol/L (3.5 mg/dL) eGFR <20 mL/min/1.73 $m^2$, stop MRA immediately and seek specialist advice.
- A specialist HF nurse may assist with education of the patient, follow-up (in person or by telephone), biochemical monitoring, and dose up-titration.

**ACE/ARB**

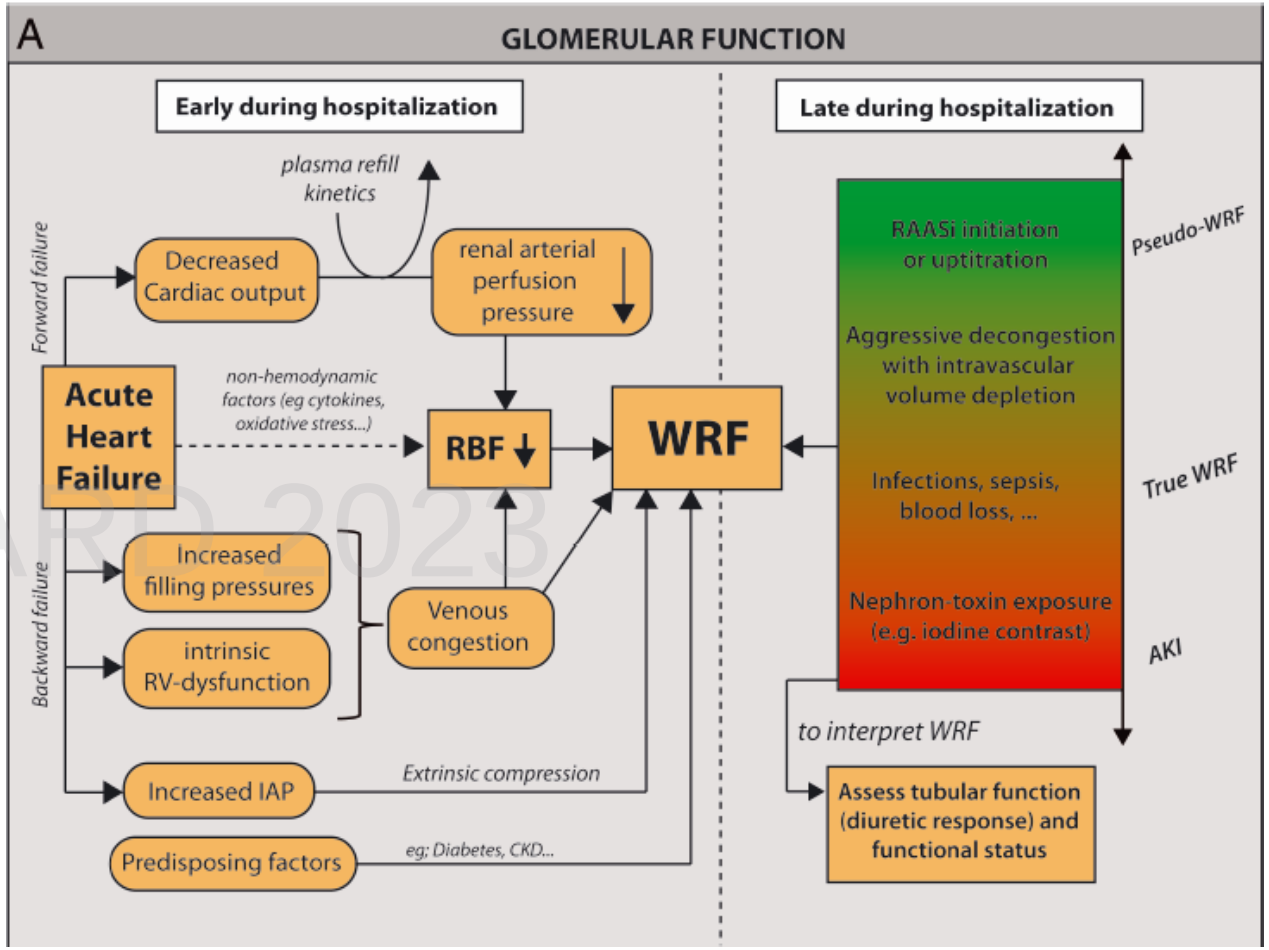
**WRF and hyperkalaemia:**

- Some rise in urea (BUN), creatinine, and $K^+$ is to be expected after an ACE-I; if an increase is small and asymptomatic, no action is necessary.
- An increase in creatinine of up to 50% above baseline, or 266 µmol/L (3 mg/dL)/eGFR <25 mL/min/1.73 $m^2$, whichever is the smaller, is acceptable.
- An increase in $K^+$ to ≤5.5 mmol/L is acceptable.
- If urea, creatinine, or $K^+$ does rise excessively, consider stopping concomitant nephrotoxic drugs (e.g. NSAIDs)[d] and other $K^+$ supplements or retaining agents (triamterene, amiloride) and, if no signs of congestion, reducing the dose of diuretic.
- If greater rises in creatinine or $K^+$ than those outlined above persist despite adjustment of concomitant medications, the dose of the ACE-I (or ARB) should be halved and blood chemistry re-checked within 1–2 weeks; if there is still an unsatisfactory response, specialist advice should be sought.
- If $K^+$ rises to >5.5 mmol/L or creatinine increases by >100% or to >310 µmol/L (3.5 mg/dL)/eGFR <20 mL/min/1.73 $m^2$, the ACE-I (or ARB) should be stopped and specialist advice sought.
- Blood chemistry should be monitored frequently and serially until $K^+$ and creatinine have plateaued.

# WRF in acute setting

1. First 3 days = shows hemodynamic derangements
2. Later changes
    1. Effective decongestion = NOT BAD
    2. Initiation/up titration of neurohormonal blockade = NOT BAD
    3. Concomitant conditions
    4. Administered nephrotoxic agents

(27). Mullens et al. demonstrated that it is the elevation of the admission baseline CVP, mean CVP, and discharge CVP rather than the cardiac index (CI) or other hemodynamic parameters that are strongly correlated with the increased risk of WRF for patients with acute decompensated heart failure (ADHF)




Reduced cardiac output = minor role
Central venous pressure = strongly affect

Circulation: Heart Failure

ORIGINAL ARTICLE

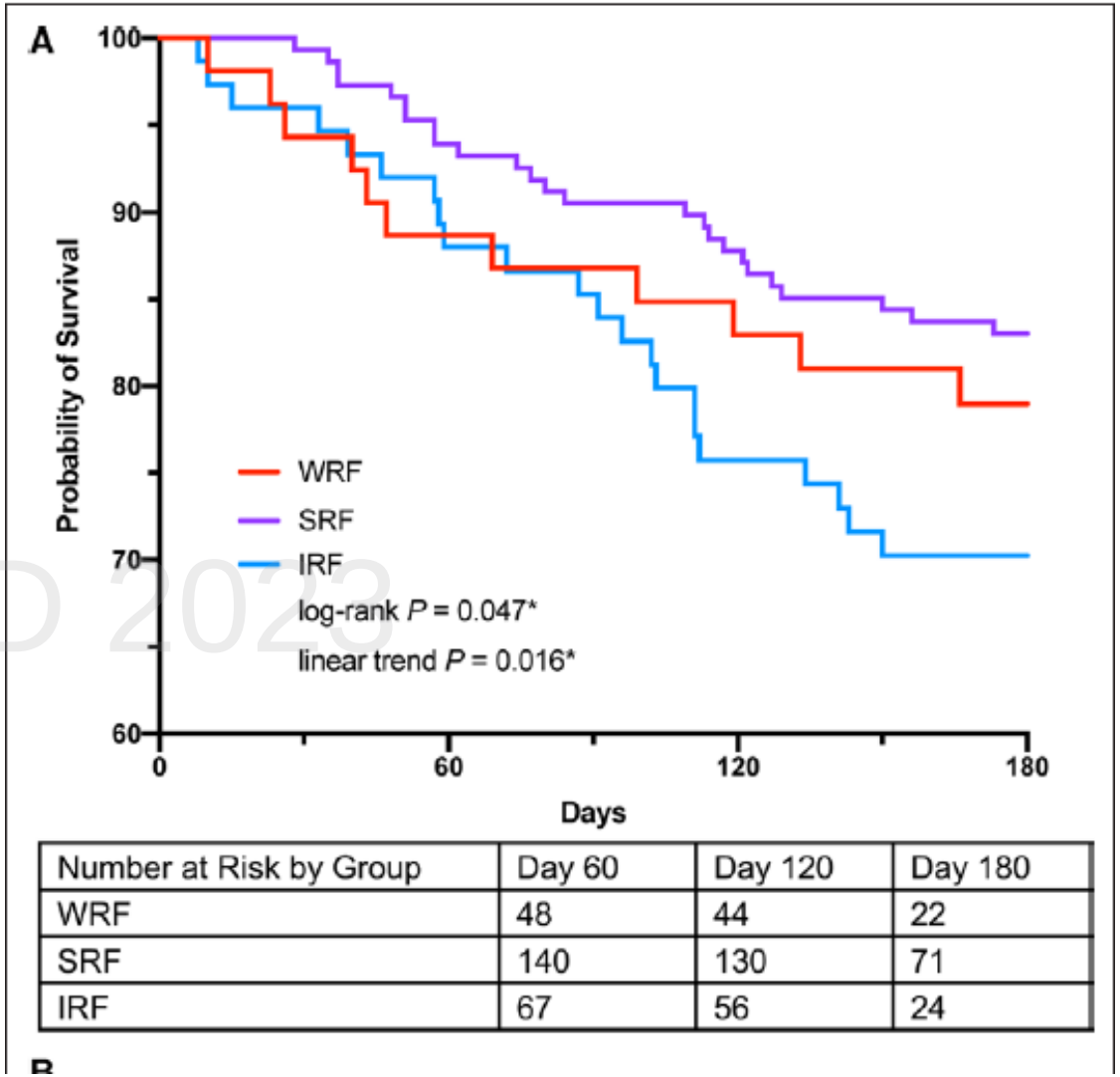
# Improvement in Renal Function During the Treatment of Acute Decompensated Heart Failure: Relationship With Markers of Renal Tubular Injury and Prognostic Importance

Improvement in renal function observed during ADHF management is a marker for greater disease severity, including more severe renal dysfunction and likely more congestion.

**Creatinine fluctuations = transient change = not a kidney damage**

**ADHF therapies should not be tailored toward a goal of improvement in renal function!**

The focus of care should be identifying and preventing the factors that result in cardiorenal destabilization.

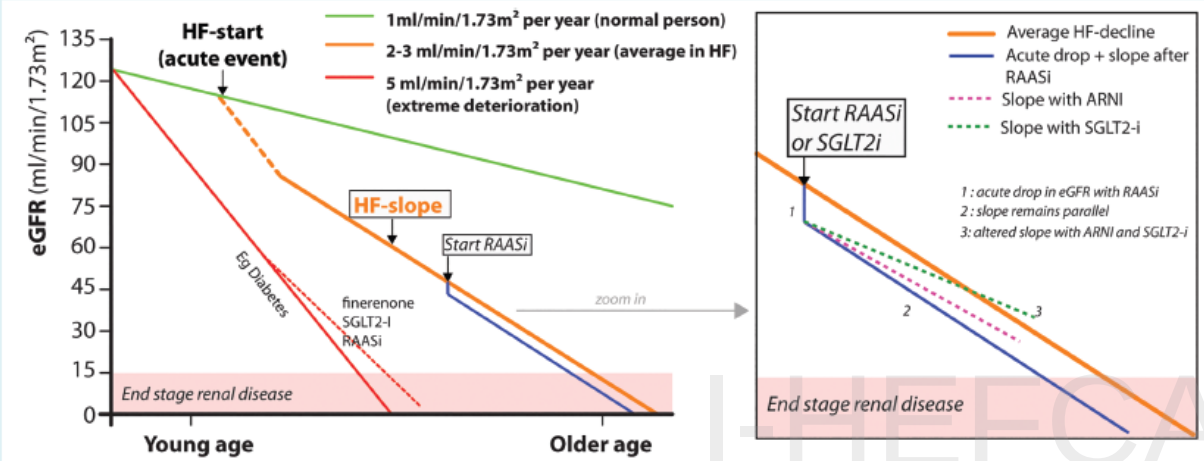



| Number at Risk by Group | Day 60 | Day 120 | Day 180 |
|---|---|---|---|
| WRF | 48 | 44 | 22 |
| SRF | 140 | 130 | 71 |
| IRF | 67 | 56 | 24 |

B

Natov et al,Circulation: Heart Failure. 2023;16

# Acute dip of GFR?

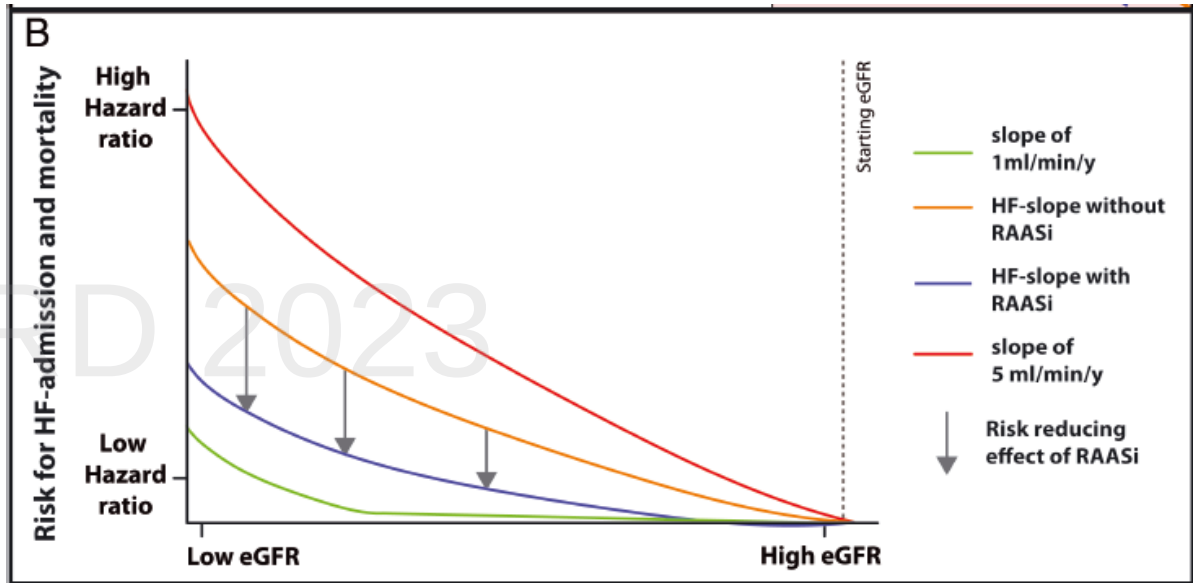



**Key messages**

1. Acute drop in GFR with RAASi, ARNI and SGLT2-i does not diminishes treatment effect
2. A reduction in slope deterioration in HFrEF with ARNI and SGLT2-i is associated with reduced hard renal endpoints

**Figure 2** Effect of drugs on renal slope. ARNI, angiotensin receptor–neprilysin inhibitor; eGFR, estimated glomerular filtration rate; HF, heart failure; HFrEF, heart failure with reduced ejection fraction; RAASi, renin–angiotensin–aldosterone system inhibitor; SGLT2-i, sodium–glucose cotransporter 2 inhibitor. Adapted from Mullens *et al.*[3]

**Don't be afraid, it's OK!**




Decongestion (+) , Initiation/Up-titration (+)

**Look the medication, not e-GFR only!**

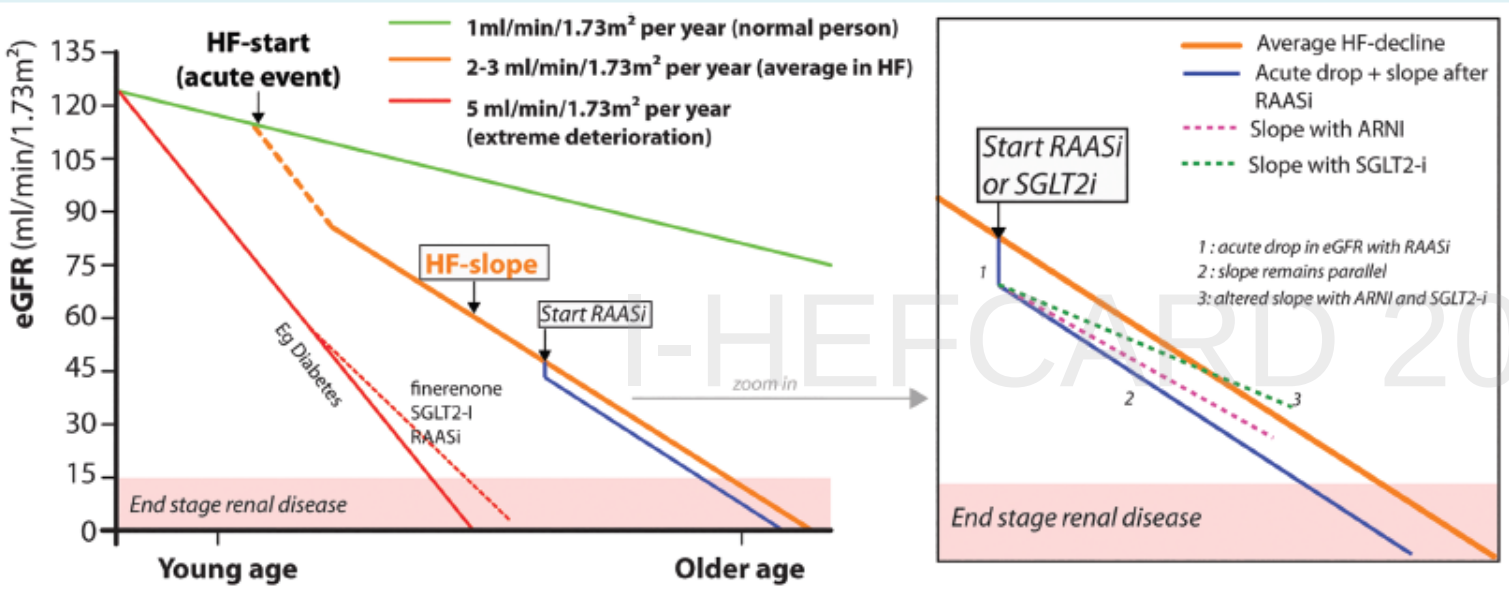
Mullens W, Eur J Heart Fail 2020; 22:584-603.

# RENAL FUNCTION IN CHRONIC HEART FAILURE



(Clinical scenario: the patient with renal dysfunction)

# Normal vs HF

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In chronic heart failure, changes in GFR over time relate to a progressive **loss of functioning nephrons.**

# FOUR PILLARS THERAPY IN HF- ADVANCED CKD

**Table 4** Initiation of heart failure drugs in relation to baseline chronic kidney disease status

| Drug | Evidence across GFR strata according to baseline eGFR enrolment criteria | | | | Acute drop GFR | Impact on GFR slope in HF trial | CKD treatment interaction | Treatment effect with CKD |
|---|---|---|---|---|---|---|---|---|
| | ESKD | 15–30 | 30–60 | >60 | | | | |
| ACE-I/ARB | Moderate evidence if dialysis, weak evidence if not on dialysis | | | | Yes | No (beneficial effect of around 1–2 ml/min/1.73 $m^2$ per year in CKD trials) | No | Relative benefit: ~<br>Absolute benefit: ↑ |
| Beta-blockers | | | | | No | No | Yes (potentially but some conflicting results) | Relative benefit: ~<br>Absolute benefit: ↑ |
| MRA | | | | | Yes | No | No | Relative benefit: ~<br>Absolute benefit: ↑ |
| ARNI | | | | | Yes | Yes (around 0.5 ml/min/1.73 $m^2$ per year) | No | Relative benefit: ~<br>Absolute benefit: ↑ |
| SGLT2-i | | >20 | | | Yes | Yes (around 1–2 ml/min/1.73 $m^2$ per year) | No | Relative benefit: ~<br>Absolute benefit: ↑ |
| Ivabradine | | | | | No | No | No | Relative benefit: ~<br>Absolute benefit: ↑ |
| Vericiguat | | | | | No | No | No | Relative benefit: ~<br>Absolute benefit: ↑ |
| Omecamtiv mecarbil | | | | | No | No | No | Relative benefit: ~<br>Absolute benefit: ↑ |

*A decrease in eGFR over time does not automatically mean RAASi/SGLT2-i need to be downtitrated or discontinued*

Dark green, strong evidence; light green, moderate evidence; red, not advised; light grey, no data. ACE-I, angiotensin-converting enzyme inhibitor; ABR, angiotensin receptor blocker; ARNI, angiotensin receptor–neprilysin inhibitor; CKD, chronic kidney disease (eGFR <60 ml/min/1.73 $m^2$); eGFR, estimated glomerular filtration rate; ESKD, end-stage kidney disease; HF, heart failure; MRA, mineralocorticoid receptor antagonist; RAASi, renin–angiotensin–aldosterone system inhibitor; SGLT2-i, sodium–glucose cotransporter 2 inhibitor.

# MRA

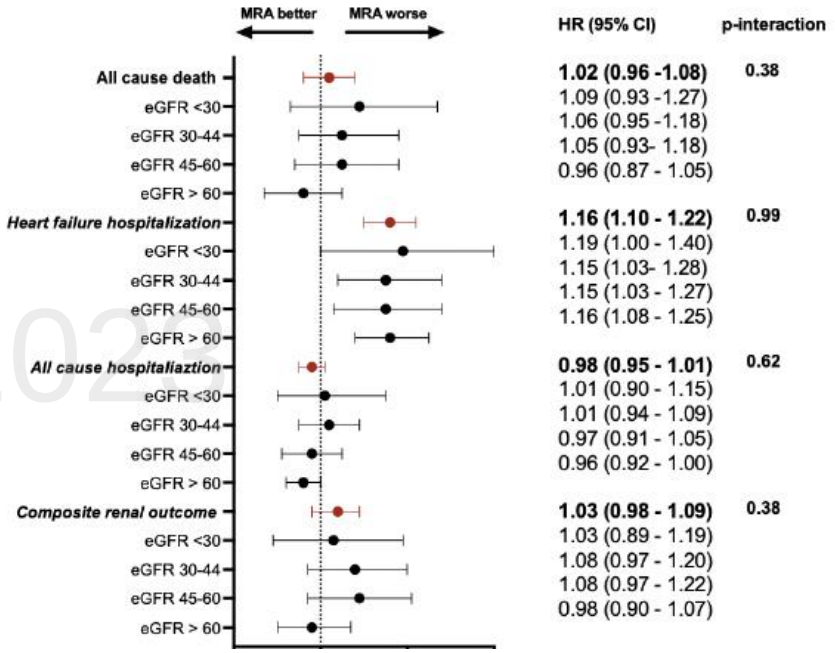
| Findings in CKD Subgroups | | |
|---|---|---|
| | All Cause Mortality | CV Death / HF Hospitalization |
| Overall | 15 – 30% RRR | 13-37% RRR |
| CKD Stages (eGFR in mL/min/1.73m²) | | |
| CKD stage 1 (> 90)<br>CKD stage 2 (60-89) | 34% RRR[50] | 8-24% RRR[48,50,51] |
| CKD stage 3A (45-59)<br>CKD stage 3B (30-44) | 32% RRR[50] | 34-38 % RRR[48,50,51] |
| CKD stage 4 (15-29) | Limited data<br>No evidence of harm | Limited data<br>No evidence of harm |
| CKD stage 5 (<15/Dialysis) | No information in HFREF | |
| **Effect on Renal Function** | | |
| The precise pathophysiology of the effect of MRA on renal function is unclear | | |
| Early decline in eGFR after initiation (2.3 to 6.7 mL/min/1.73m²)[48] | Long term slope in eGFR slightly steeper with eplerenone vs placebo (-0.3 vs -0.1 mL/min/1.73m²/year)[48,51] | WRF during uptitration of MRA-inhibition not associated with worse outcome[39,43] |
| **Management of substantial increase in serum creatinine/drop in eGFR during initiation/uptitration** | | |
| In the context of uptitration of MRAs some increase in serum creatinine / drop in eGFR is expected and acceptable. The survival benefit seen with this class of drugs far outweigh the risks associated with this perceived worsening of renal function (WRF) | | |

| Δ serum creatinine (%) | Max serum creatinine (mg/dL) | Min eGFR mL/min/1.73m² | Max serum potassium (mmol/L) | Action advised |
|---|---|---|---|---|
| < 50 | 2.5 mg/dL | 30 | 5.0 | None, uptitrate and evaluate renal function and electrolytes |
| 50-100 | 3.5 mg/dL | 20 | 5.5 | Evaluate clinical status and other causes of WRF. Consider halving MRA and re-evaluate |
| > 100 | > 3.5 mg/dL | < 20 | > 6.0 | Evaluate clinical status and other causes of WRF. Consider stopping MRA and re-evaluate |
| Rechallenge after 2-4 weeks (if possible at lower dose) when dosing reduced or stopped all together if renal function and/or potassium has improved | | | | |

## Results:

### Safety of Continuing MRAs in Patients with HFrEF and severe CKD:

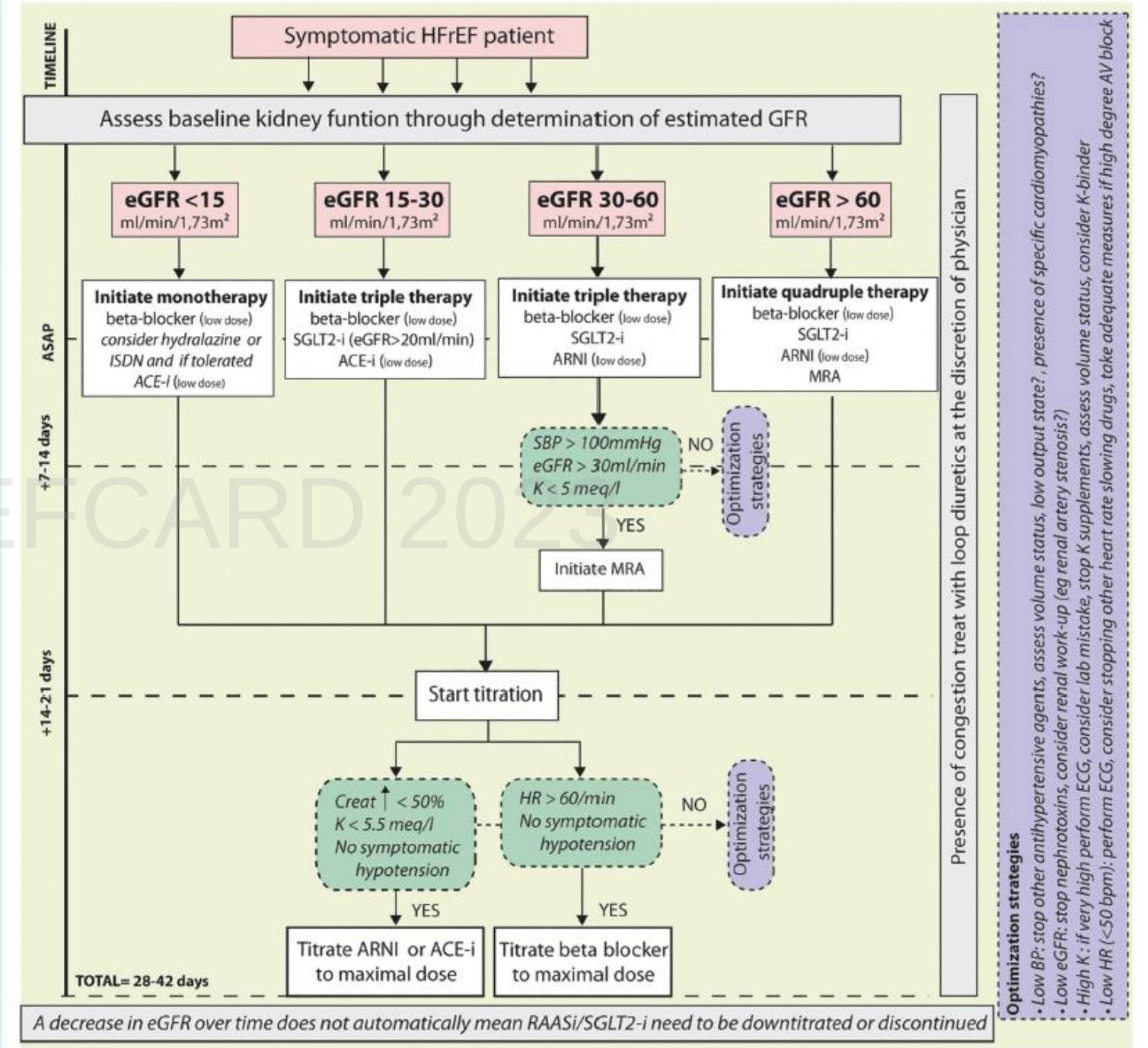
### Data from Swedish Heart Failure Registry




- An GFR > 30 ml/min/1.73 m² was the strongest predictor of MRA-use
- MRA-use was not associated with a higher risk of mortality, all-cause hospitalization and renal mortality and morbidity, however it was associated with a higher risk of HF hospitalization
- The safety profile was consistent across the different eGFR classes

Heart Failure & World Congress on Acute Heart Failure 2023

# Renal approach to initiate and titrate quadruple therapy



# Medical Management of Heart Failure With Reduced Ejection Fraction in Patients With Advanced Renal Disease

Aaron M. Hein, BS,[a] Julia J. Scialla, MD, MHS,[a,b] Daniel Edmonston, MD,[a,b] Lauren B. Cooper, MD, MHS,[a,c] Adam D. DeVore, MD, MHS,[a,b] Robert J. Mentz, MD[a,b]

**FIGURE 1** Recommended Dosage Adjustments and Monitoring Considerations in Patients With HFrEF and CKD Stages G4 to G5

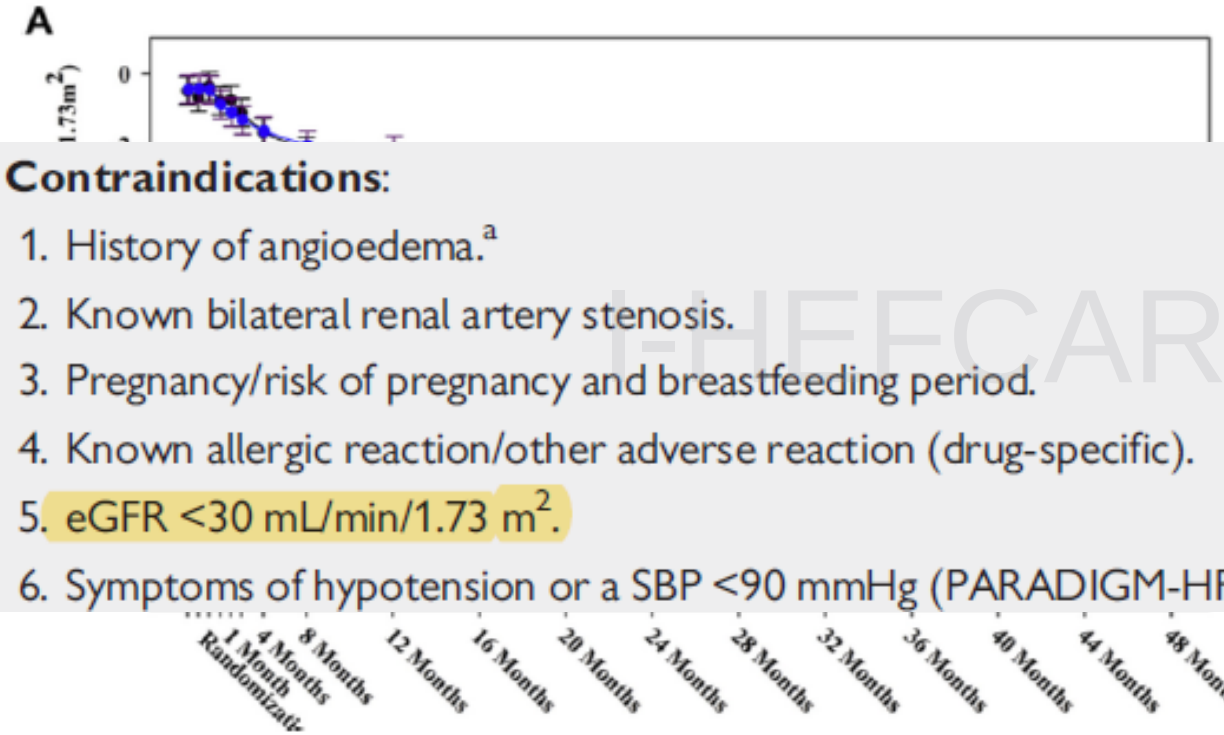
| | ACE-Is* | ARBs | ARNIs | MRAs | Beta-Blockers |
|---|---|---|---|---|---|
| Dosing Considerations | **No adjustment:**<br>- Fosinopril<br>**Half-dose initially:**<br>- Captopril† - Enalapril<br>- Lisinopril - Trandolapril<br>**Quarter-dose initially:**<br>- Ramipril<br>**Not recommended:**<br>- Perindopril | **No adjustment:**<br>- Candesartan‡ (untested in eGFR <15 mL/min/1.73m²)<br>- Losartan<br>- Valsartan‡ (untested in eGFR <10 mL/min/1.73m²) | Sacubitril/valsartan<br>**Initial dose§:**<br>24 mg/26 mg, double every 2-4 weeks as tolerated<br>**Target dose:** 97 mg/103 mg | **Contraindicated:**<br>- Spironolactone<br>- Eplerenone | **No adjustment:**<br>- Carvedilol<br>- Metoprolol succinate<br>**Half-dose initially:**<br>- Bisoprolol |
| Monitoring | Blood pressure, serum potassium, kidney function | | | Blood pressure, heart rate, blood glucose in patients with diabetes mellitus | |

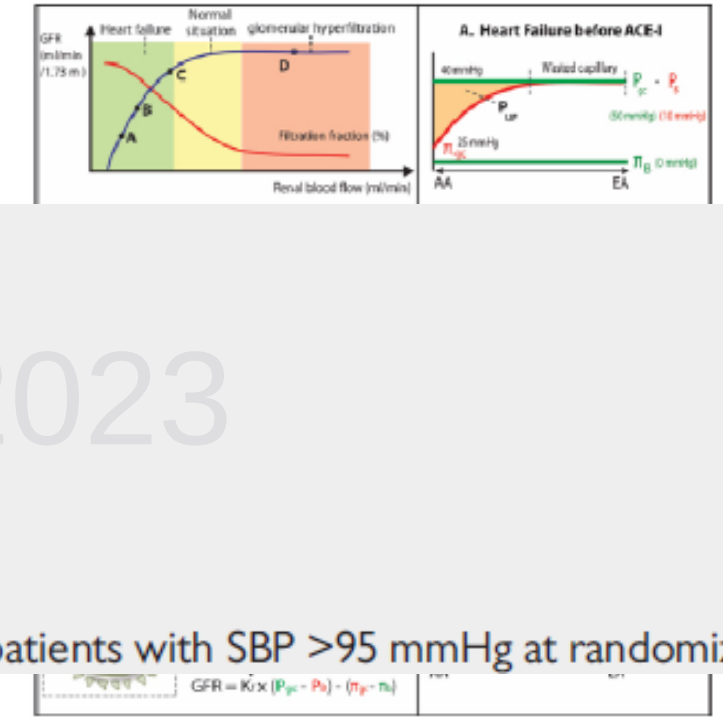
Dosage adjustments and monitoring adjustments are shown for patients with HFrEF and CKD stages G4 to G5. Adjustments refer to initial dosage with titration as tolerated based on safety. Dosing adjustments primarily derived from manufacturer labeling and package insert data when possible. †Reduced rate of titration recommended for captopril. ‡Use with caution. §Twice daily dosing. ACE-Is = angiotensin-converting enzyme inhibitor; ARNI = angiotensin receptor blocker/neprilysin inhibitor; eGFR = estimated glomerular filtration rate; HFrEF = heart failure with reduced ejection fraction; MRA = mineralocorticoid receptor antagonist.

# CAN WE DO SOMETHING FOR RENOPROTECTIVE ACTION?

# ARNI

## Sacubitril/Valsartan lowers decline of eGFR



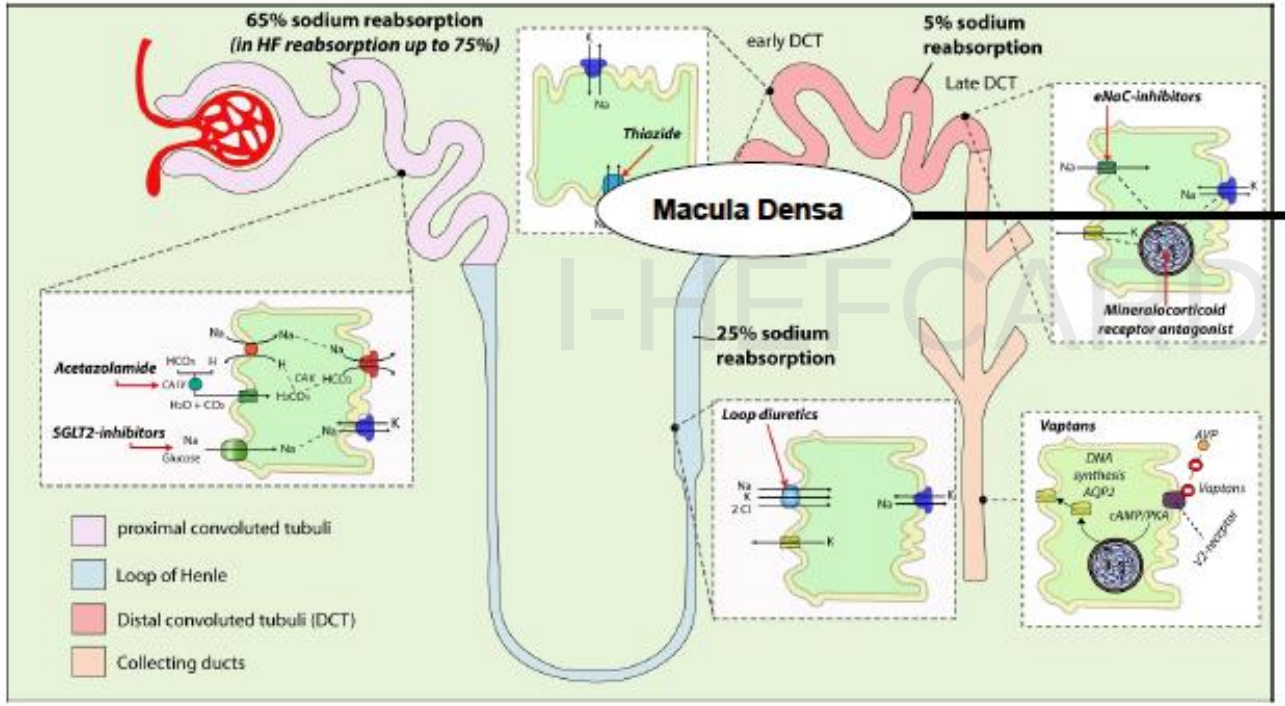

**Contraindications**:

1. History of angioedema.[a]
2. Known bilateral renal artery stenosis.
3. Pregnancy/risk of pregnancy and breastfeeding period.
4. Known allergic reaction/other adverse reaction (drug-specific).
5. eGFR <30 mL/min/1.73 $m^2$.
6. Symptoms of hypotension or a SBP <90 mmHg (PARADIGM-HF enrolled patients with SBP >95 mmHg at randomization)

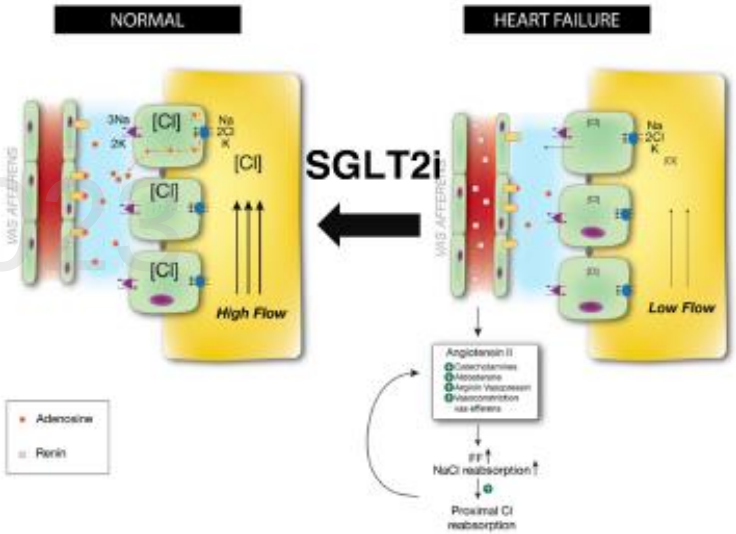


*Damman K et al JACC Heart Fail 2018*
*Mullens W, Martens P. JACC Heart Fail 2018.*

## HF induces an increased renal sodium reabsorption, especially in the proximal parts




## SGLT2i decrease renin release @ macula densa




Mullens W, Eur J Heart Fail 2019; 21:137-155.

# HOW TO USE SGLT2-I?

## Who are the suitable candidates for in-hospital sodium-glucose co-transporter 2 inhibitor initiation?

Patients hospitalized for AHF without symptomatic hypotension, inotropic support, with need for increasing IV diuretic dose, or using IV vasodilators within the previous 6 h may be eligible for therapy. On the other hand, unsta-

**Contraindications:**

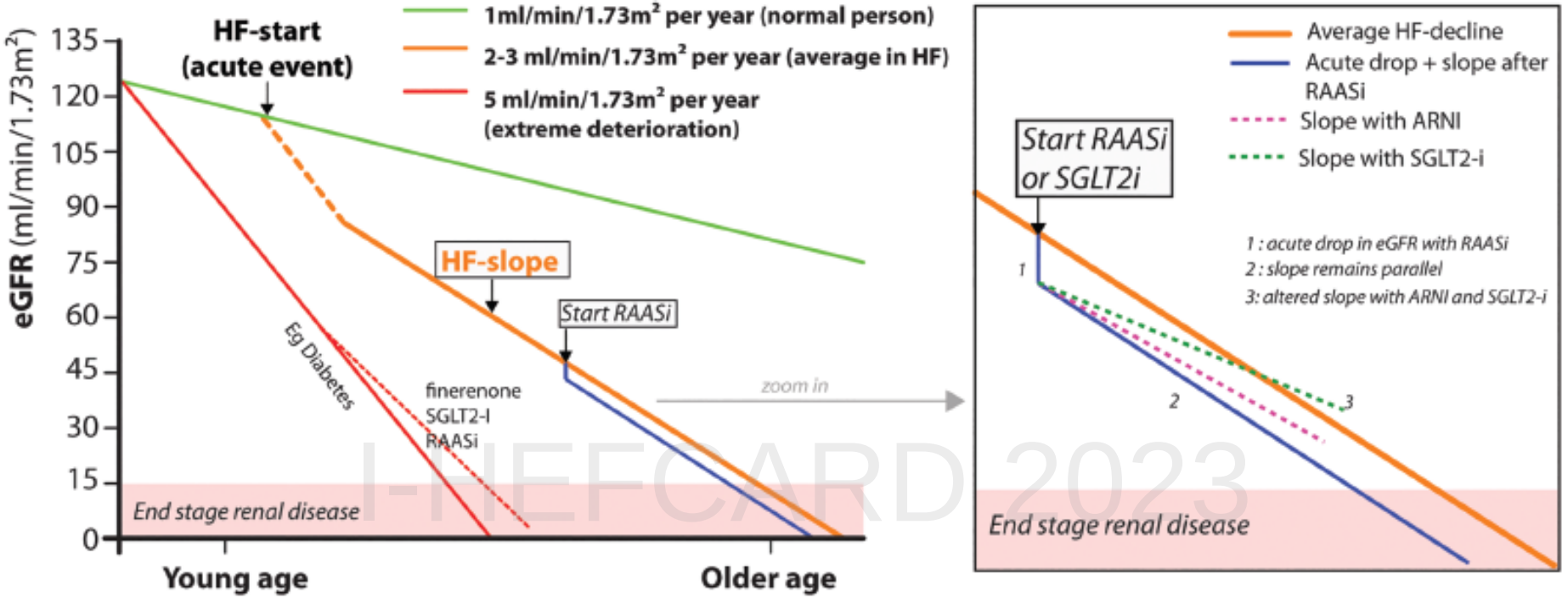
1. Known allergic reaction/other adverse reaction (drug-specific).
2. Pregnancy/risk of pregnancy and breastfeeding period.
3. eGFR <20 mL/min/1.73 $m^2$.*
4. Symptoms of hypotension or a SBP <95 mmHg.

**DAPA-CKD (dapagliflozin) enrolled patients with an eGFR >25 mL/min/1.73 $m^2$*

Sodium-glucose co-transporter 2 inhibitors improve clinical outcomes and are safe in patients with AHF and, unless contraindicated, should be rapidly initiated (within the first 5 days) in patients admitted to the hospital for decompensated HF.



**ANY MOMENT IN TIME!**



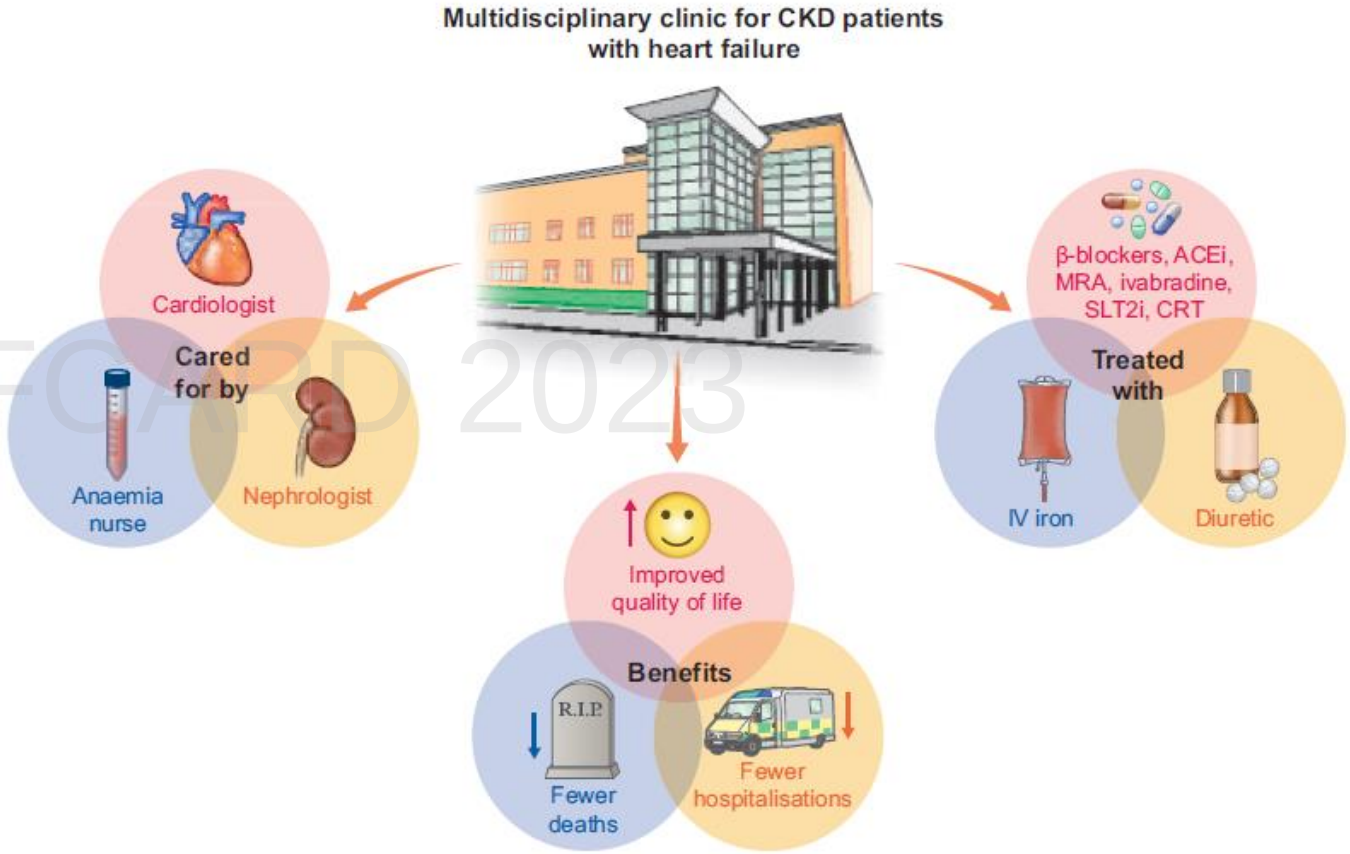



## Key messages

1. Acute drop in GFR with RAASi, ARNI and SGLT2-i does not diminishes treatment effect
2. A reduction in slope deterioration in HFrEF with ARNI and SGLT2-i is associated with reduced hard renal endpoints

**Figure 2** Effect of drugs on renal slope. ARNI, angiotensin receptor–neprilysin inhibitor; eGFR, estimated glomerular filtration rate; HF, heart failure; HFrEF, heart failure with reduced ejection fraction; RAASi, renin–angiotensin–aldosterone system inhibitor; SGLT2-i, sodium–glucose cotransporter 2 inhibitor. Adapted from Mullens *et al.*[3]

# Multi disclipinary approach



# TAKE HOME MESSSAGES……

- Both CKD and HF are intertwined, prevalent, and related to worse outcome
- Consider the **current situation**. Right analysis >> right decision.
- Don’t overreact with WRF. Worsening renal function **should not stop** decongestion and/or neurohormonal blockers. Acute dip e-GFR following initiation and up-titration RAAS blocker is OK.
- **SGLT2/ARNI do lower down the progression of CKD in HF**
- Personalized therapy and multidiscipline approach are needed

# Thank you

# Which beta blocker in dialysis?

# Goals?

1) Thorough decongestion
2) Maintain GDMT
3) Ensure adequate perfusion pressure