Cardiorenal Syndrome: Key points towards better management

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URDU COUPLET

- DARD E DIL DARD E GURDE

- ASHIK BANYA AAP NE

Introducing Nephrocardiology

Parta Hatamizadeh

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Introduction

Although the term nephrocardiology, or in some instances cardionephrology, has been sporadically used in the medical literature (1,2), its implication has been mainly limited to cardiorenal syndrome and cardiac complications of CKD. Moreover, it has not consistently implied the same denotation in its different appearances and, until recently, it had never been clearly described as a medical discipline with a defined scope (3,4).

Despite the importance of cardiorenal syndrome and cardiac complications of CKD, the interaction between nephrology and cardiovascular medicine is much broader and includes important subjects that are not well addressed in either of the two specialties

Therefore, the next step was taken with the introduction of a practical classification system (6), which instead of indicating the initiating organ, signified the "dominant" clinical presentation at any point in time. By classifying cardiorenal syndrome into the following seven categories, it specifies the clinical finding that needs to be "addressed first": hemodynamic; uremic; vascular; neurohormonal (including electrolytes and acid-base disorders); anemia and iron metabolism; mineral metabolism; and malnutrition-inflammation-cachexia. This classification system can help clinicians streamline a patient's care plans over the course of the illness and communicate their findings and management plans among themselves (7). By demonstrating the broadness and complexity of the pathophysiologic

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Cardiorenal Medicine

Letter

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Advances in Cardiorenal Medicine: The Year 2023 in Review

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Keywords
Heart failure · Kidney disease · Cardiorenal medicine · Cardionephrology · Year 2023

Conceptual Considerations in Cardiorenal Nexus

In late 2023, the American Heart Association (AHA) published a presidential advisory to describe a new multisystem entity called cardiovascular-kidney-metabolic (CKM) syndrome [1]. The authors defined CKM syndrome as a systemic disorder characterized by

Nephrol Dial Transplant (2022) 37: 2386–2397
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Long-term impact of cardiorenal syndromes on major outcomes based on their chronology: a comprehensive French nationwide cohort study

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GRAPHICAL ABSTRACT

JCF
Journal of Cardiac Failure

HFSTATS

EPIDEMIOLOGY AND OUTCOMES

HF STATS 2024: Heart Failure Epidemiology and Outcomes Statistics
An Updated 2024 Report from the Heart Failure Society of America

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KEY POINTS IN EPEDEMIOLOGY

- One Out 4 Individuals will Develop heart failure in their life time USA.
- Prevalence of kidney disease in heart failure patients 26% to 45%.
- CVD in patients with CKD. 63% in Stage 1-2 and 75% in CKD 4-5.

Rev Esp Cardiol. 2024;77(1):50–59

Original article

Prevalence and clinical profile of kidney disease in patients with chronic heart failure. Insights from the Spanish cardiorenal registry

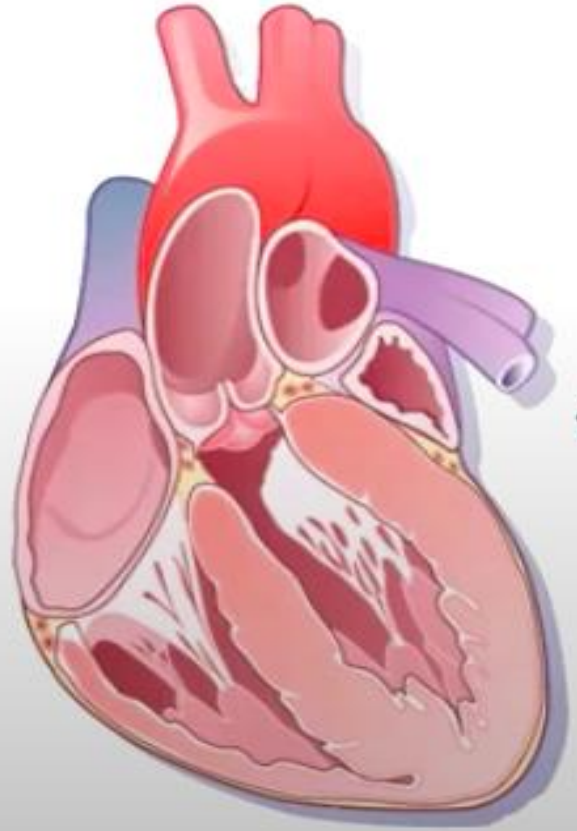
Review > Nat Rev Nephrol. 2022 Nov;18(11):696–707. doi: 10.1038/s41581-022-00616-6. Epub 2022 Sep 14.

Epidemiology and risk of cardiovascular disease in populations with chronic kidney disease

Kunihiro Matsushita [1 2 3], Shoshana H Ballew [4 5], Angela Yee-Moon Wang [6], Robert Kalyesubula [7 8], Elke Schaeffner [9], Rajiv Agarwal [10]

Cardiac dysfunction

Renal dysfunction



Bad stuff!

Defination

- **Definition of Cardiorenal Syndrome (CRS)**:A condition where heart and kidney dysfunction are interconnected and mutually exacerbate one another.
- **Types of CRS**:
  - **Type 1**: Acute heart failure leading to acute kidney injury (AKI).
  - **Type 2**: Chronic heart failure leading to chronic kidney disease (CKD).
  - **Type 3**: Acute kidney injury leading to acute heart failure.
  - **Type 4**: Chronic kidney disease leading to heart failure.
  - **Type 5**: Both heart and kidney failure due to systemic conditions (e.g., diabetes, sepsis).

NEW EMERGING CONCEPT IS CADIOVASCULAR KIDNEY METABOLIC HEALTH

Index patient

- 54 year Male
- DM HYPERTENSION DYSLIPIDEMIC IHD LOW EF 25%
- ER with Progressive Breathlessness and Swelling Of the Whole Body for a weeks duration.
- On Examination Restless
- Gained 8kg of weight HR 98/ min , JVP raised RR 30/ min at rest BP 110/70 Mmof Hg
- Pale , Edematous all over with Decreased breath sounds at the bases , and LVS3 gallop, Has a large palpable Liver with evidence of Ascites.
- Investigations Reveal HB 10.8 G/dl Low Iron saturation ,Bun 23 mmol/l and Creatnine -176umol/l (baseline 100 umol/l),Na 128 Mmol/l , Serum K 5.0 mmol/l Hco3 18 mmol/l. HBA1c 7.1
- Urine R/M Protein 1 + No active sediments
- He Is On Lasix 40 Bid , Telmisartan 40 Mg OD, Plavix 75mg OD, hydralazine, Long acting Insulin OD, Glucophage 100 XR and Novonorm 1mg TID, Rusvastatin 10 Mg OD

Index patient Continued

- Despite The guidelines for the treatment Of HF the application to patients remain dismal.
- What Went Wrong With this patient ? What is pathophysiology?
- What Extra could be done ?
- What are the recent Updates ?

2023 Focused Update of the 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

ESC Clinical Practice Guidelines

25 Aug 2023

Cardio-Renal Syndrome Pathophysiology

CKD-Associated Myocardial Changes
Myocyte hypertrophy
Myocyte dysfunction
↑↑Interstitial Fibrosis
↓Capillary density
↑↑LV Mass
Elevated serum troponin levels

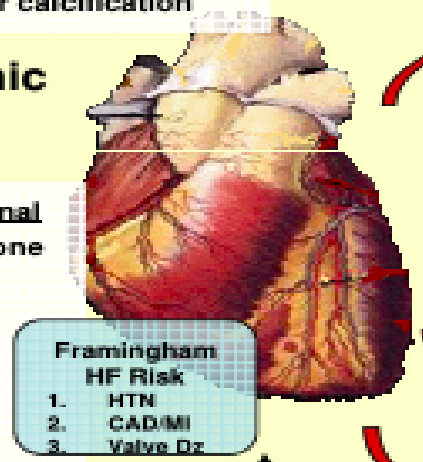
CKD-Associated Vascular Changes
Accelerated atherosclerosis
↑Vascular stiffness
↓Smooth muscle density
Osteoblastic VSMC transformation
Intra-and extracellular calcification

Acute on Chonic Cardiac Disease

Chronic Neurohormonal
↑SNS, RAS, Aldosterone
↓Vitamin D
↑PTH
↑PO4
Hypotestesteronism
↓EPO
↓Fe utilization
Marinobufagen

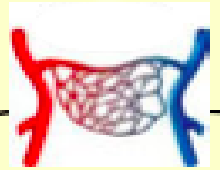
Inciting Events
↓Medical compliance
↑Sodium intake
Ischemia
Arrhythmias (AF)
OSAS

Added Insults
NSAIDS, TZDs



Systolic or Diastolic Dysfunction or Both

Altered Intra-renal Hemodynamics



Decreased Perfusion

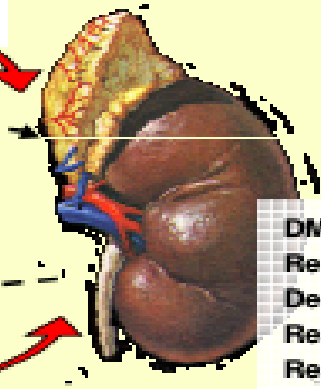
Increased venous pressure

Renal Congestion

Precipitators
Diuretics
Vasodilators
Procedures

Toxicity
Vascocostriction

Acute Neurohoromonal Activation
SNS+RAS+Aldosterone+ Endothelin+ADH+, renal vasoconstriction (adenosine)+prostaglandin dysregulation



ANP/ BNP

Natruretic Peptides

Blocked Natriuresis

Humoral signalling

Anemia/Relative ↓Epo/Fe transport blocked

Cytokine secretion

IL-1, TNF-Alpha

Monocyte Activation

Endothelial Dysfunction

↓Tubulo-Glomerular Function

↑Adhesion Molecules, ↑Enzymatic Activation, ↑Oxidative Stress

Acute on Chronic Kidney Injury

DM+HTN + other CKD
Renal hypoperfusion
Decreased GFR
Resistence to diuretics
Resistance to ANP/BNP
Na + H2O retention
Necrosis / apoptosis
Fibrosis

Biomarkers
↑BNP/NT-proBNP
↑ N-GAL
↑ KIM-1
↑IL-18
Catalytic iron
↑Cystatin-C
↑Creatinine
Urine albumin

Pathophysiology

- **Interaction between Heart and Kidney**: **Impaired cardiac function** reduces renal perfusion, contributing to kidney dysfunction. Low Cardiac output and Increased venous pressure

- **Reduced renal function** leads to fluid and electrolyte imbalances, worsening heart failure. NEPHROSARCA or renal Congestion

- **Vicious cycle**: Decreased renal blood flow → Activation of RAAS (Renin-Angiotensin-Aldosterone System) and SNS (Sympathetic Nervous System).

- INFLAMMATION AND FIBROSIS

- **RAAS & SNS** activation causes vasoconstriction, fluid retention, and increased cardiac workload. Declining Renal and Cardiac functions.

Treatment Continued

- **Improvement in cardiac function** (if heart failure is the predominant cause)

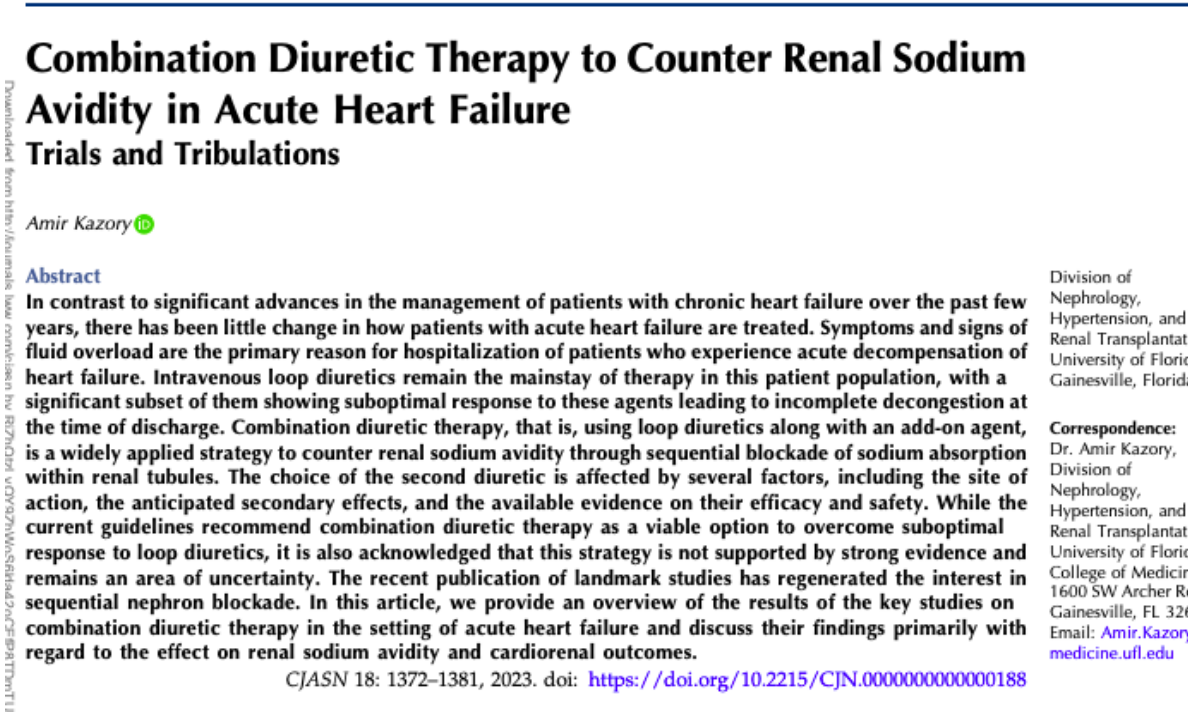
- .**Restoration of kidney function** (addressing renal injury or dysfunction).

- **Managing fluid balance**: Reducing volume overload while maintaining perfusion.

- **Minimizing complications** such as electrolyte imbalances, acidosis, and arrhythmias.

DIURETICS

- Loop Diuretics
- Thiazide Diuretics
- Mineralocorticoids Receptor antagonist
- Acetazolamide
- Vaptans



Combination Diuretic Therapy to Counter Renal Sodium Avidity in Acute Heart Failure

Trials and Tribulations

Amir Kazory

Abstract

In contrast to significant advances in the management of patients with chronic heart failure over the past few years, there has been little change in how patients with acute heart failure are treated. Symptoms and signs of fluid overload are the primary reason for hospitalization of patients who experience acute decompensation of heart failure. Intravenous loop diuretics remain the mainstay of therapy in this patient population, with a significant subset of them showing suboptimal response to these agents leading to incomplete decongestion at the time of discharge. Combination diuretic therapy, that is, using loop diuretics along with an add-on agent, is a widely applied strategy to counter renal sodium avidity through sequential blockade of sodium absorption within renal tubules. The choice of the second diuretic is affected by several factors, including the site of action, the anticipated secondary effects, and the available evidence on their efficacy and safety. While the current guidelines recommend combination diuretic therapy as a viable option to overcome suboptimal response to loop diuretics, it is also acknowledged that this strategy is not supported by strong evidence and remains an area of uncertainty. The recent publication of landmark studies has regenerated the interest in sequential nephron blockade. In this article, we provide an overview of the results of the key studies on combination diuretic therapy in the setting of acute heart failure and discuss their findings primarily with regard to the effect on renal sodium avidity and cardiorenal outcomes.

CJASN 18: 1372–1381, 2023. doi: https://doi.org/10.2215/CJN.0000000000000188

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Diuretics

- **Loop Diuretics** Main stay Na- K-2Cl Channel Ascending Loop Of Henle
- DOSE Trial High Dose.
- IV dose twice than home dose.
- ESC doubling the dose of if urinary Na <50meq/l or UOP <100ml/hr post 6 Hours of administration. BOLUS Vs Continuous.

- **Thiazides** Block the Na-Cl Cotransporter in DCT
- Loop USE hypertrophy of DCT with increase in Na-Cl transporter
- CLOROTIC Trial Greater efficacy in Combination of loop with thiazides
- Hydrochlorthiazide, Metalozone , Clorthalidone

Clinical Practice guidelines :Circuation 2022:145(18):e895-1032 DOSE

Thiazide Uses In advanced CKD J Am S Hypertens 2012:6(5)299-308 CLOROTIC

Drug	Site of Action	Duration of Action	Common Starting Dosage	Maximum Dosage	Common Side Effects
Loop diuretics	Inhibition of Na-K-Cl co-transporter in the thick ascending loop of Henle				Hypokalaemia, hypomagnesaemia, hyperuricaemia, hypocalcaemia, hyponatraemia, otoxicity
Furosemide		7 h	20 to 40 mg once or twice	600 mg	
Bumetanide		4 to 6 h	0.5 to 1.0 mg once or twice	10 mg	
Torasemide		12 to 16 h	10 to 20 mg once	200 mg	
Ethacrynic acid		6 h	25–50 mg once or twice	200 mg	

Thiazide-like diuretics	Inhibition of Na-Cl transporter at distal nephron				Hypokalaemia, hypomagnesaemia, hypercalcaemia, hyponatraemia, hyperuricaemia
Chlorothiazide		6 to 12 h	250 to 500 mg	Once or twice	1,000 mg
Chlorthalidone		24 to 72 h	12.5 to 25 mg once	100 mg	
Indapamide		36 h	2.5 mg once	20 mg	
Potassium-sparing diuretics	Inhibition of mineralcarticoid receptor or its effectors at distal nephron				Hyperkalaemia
Amiloride		24 h	5 mg once	20 mg	
Triamterene		7 to 9 h	50 to 75 mg twice	200 mg	
Spironolactone		1 to 3 h	12.5 to 25.0 mg once	50 mg	Gynecomastia

Diuretics

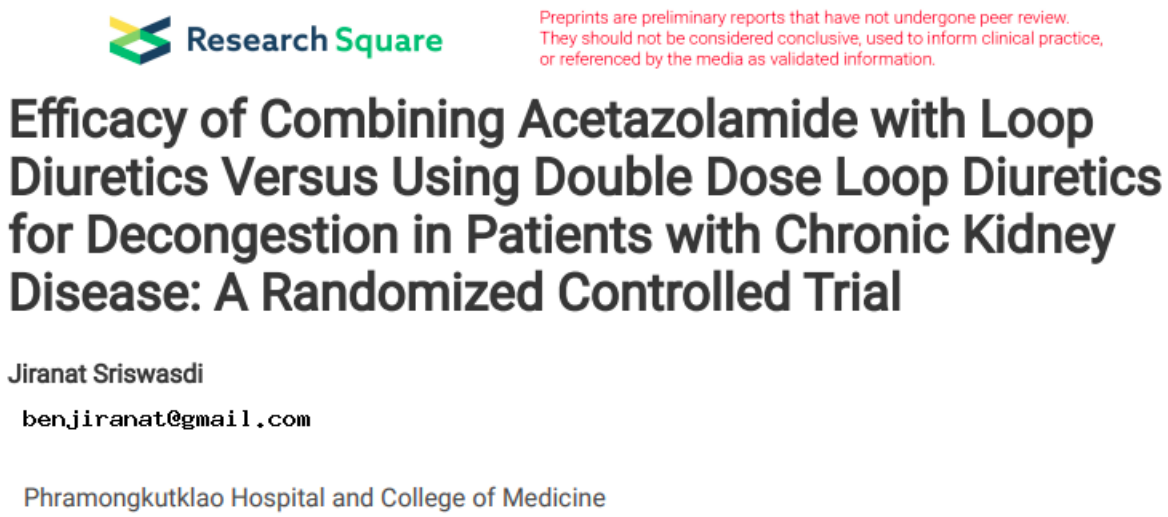
- **Acetazolamide:**
- **CA Inhibitor acts at Proximal Tubule Of Nephron**

- **Incorporated as a diuretic in case of significant metabolic alkalosis.**

- **Reduces sodium reabsorption Via Na/H Exchanger ,leading to Greater natriuresis.**

- **Effect is Lost after approx. 3 days**

- **IV Acetazolamide 500 Mg Iv Bolus**



- **Acetazolamide in Acute Decompensated Heart Failure with Volume Overload (ADVOR)**

N Engl J Med 2022;387:1185-1195

Diuretics Continued

- Vasopressin Antagonists
- Tolvaptan a selective V2 antagonist ,blocks the insertion of aquaporins into the distal tubule of the kidney and leads to Aquaresis.
- Everest Trial
- Randomized trial of 4133 patients with ADHF showed greater weight loss in Tolvaptan arm
- Addition of Tolvaptan could be considered among patients needing additional decongestion and hyponatremia.

- EVEREST out come Trial . JAMA 2007 ; 297(12)1319-1331

Continued

- ACE / ARB & Neprilysin Inhibitors
- Activation OF RAAS
- Blockade HELP in both HFrEF and HFpEF and CKD.
- SOLVD Trial
- PARADIGM trial & PARAGON trial
- Myocardial Stretch NP
- Inhibition of Neprilysin ,a metalloprotein Degrades Natriuretic Peptide Benefits both HPrEF and HPpEF and CKD when combined with ARB (Sacubitril/Valsartan)

Contd.

- Renin angiotensin aldosterone System and SNS activation
- MRA
- DIURETICS DCT
- ANTI INFLAMMATORY
- ANTI FIBROTIC
- SIDE EFEECTS
- Non Steroidal MINERALOCORTICOIDS ANATAGONIST

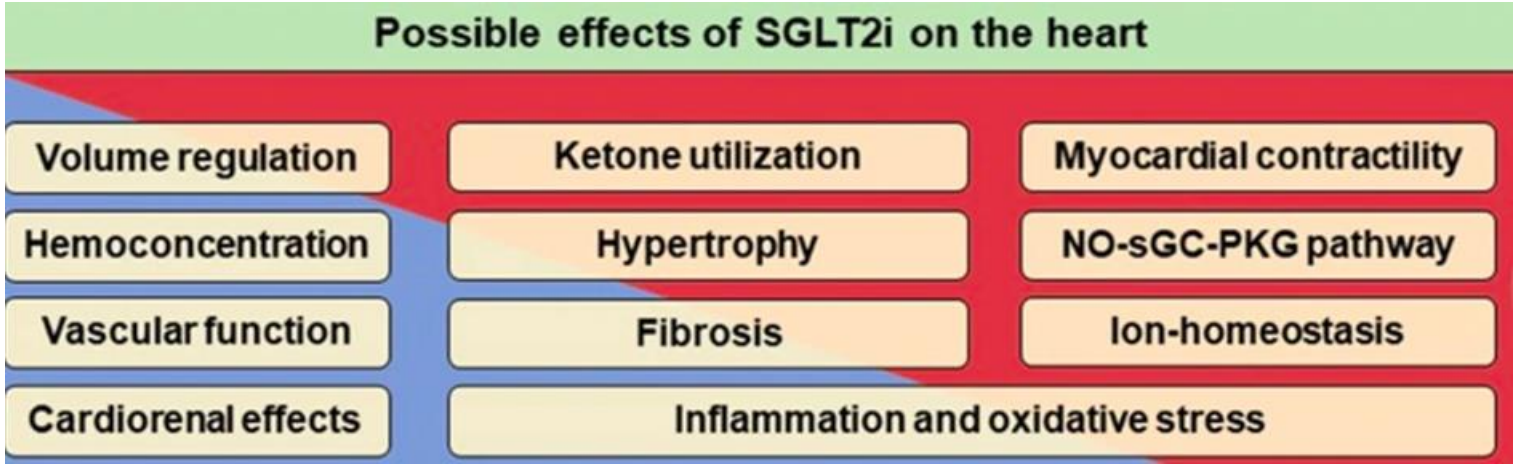
Volume 151, Issue 1, 7 January 2025; Pages 45-58
https://doi.org/10.1161/CIRCULATIONAHA.124.072011

ORIGINAL RESEARCH ARTICLE

Efficacy and Safety of Finerenone Across the Ejection Fraction Spectrum in Heart Failure With Mildly Reduce Preserved Ejection Fraction: A Prespecified Analysis o the FINEARTS-HF Trial

Treatment Contd

- SGLT2i
- FIRST LINE THERAPY IN GUIDELINES BOTH FOR HEART FAILURE DIABETIC KIDNEY DISEASE AND OTHER SUBTYPES OF KIDNEY DISORDERS.
- SGLT2 I leads to a wide range of improved Clinical Outcomes, including overall survival, cardiovascular outcomes, HF hospitalizations and kidney failure.



Sci Rep. 2023 Sep 23;13:15922. doi: 10.1038/s41598-023-42989-z

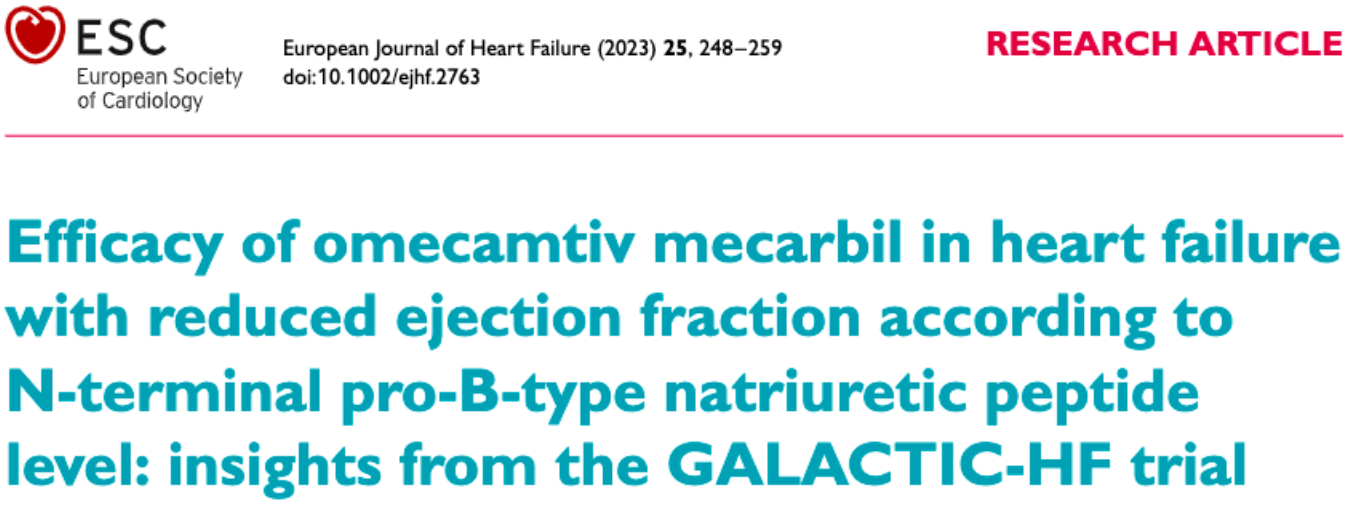
SGLT-2 inhibitors improve cardiovascular and renal outcomes in patients with CKD: a systematic review and meta-analysis

Thomas A Mavrakanas [1,✉], Michael A Tsoukas [2], James M Brophy [3], Abhinav Sharma [3], Karim Gariani [4]

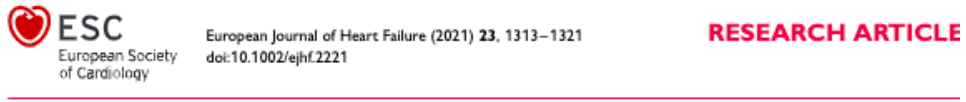
Author information · Article notes · Copyright and License information

Contd

- Vericiguat
- It helps by reducing oxidative stress, increasing cyclic GMP, and improving clinical HF
- By stimulating cardiac myosin, a protein responsible for converting chemical energy into the mechanical force that helps the heart contract, omecamtiv mecarbil may improve cardiac muscle performance.


ESC European Society of Cardiology
European Journal of Heart Failure (2023) 25, 248–259
doi:10.1002/ejhf.2763
RESEARCH ARTICLE

Efficacy of omecamtiv mecarbil in heart failure with reduced ejection fraction according to N-terminal pro-B-type natriuretic peptide level: insights from the GALACTIC-HF trial


ESC European Society of Cardiology
European Journal of Heart Failure (2021) 23, 1313–1321
doi:10.1002/ejhf.2221
RESEARCH ARTICLE

Renal function and the effects of vericiguat in patients with worsening heart failure with reduced ejection fraction: insights from the VICTORIA (Vericiguat Global Study in Subjects with HFrEF) trial

Treatment Continued

- β-Blocker therapy has consistently shown to be an effective therapy in reducing the risk of all-cause mortal-ity and combined end points of all-cause/cardio vascular death or HF hospitalization in patients with HFrEF and HFpEF

ESC European Society of Cardiology
European Heart Journal (2018) **39**, 26–35
doi:10.1093/eurheartj/ehx564
FASTTRACK CLINICAL RESEARCH
Heart failure/cardiomyopathy

Beta-blockers for heart failure with reduced, mid-range, and preserved ejection fraction: an individual patient-level analysis of double-blind randomized trials

Current Problems in Cardiology 49 (2024) 1023

Contents lists available at ScienceDirect

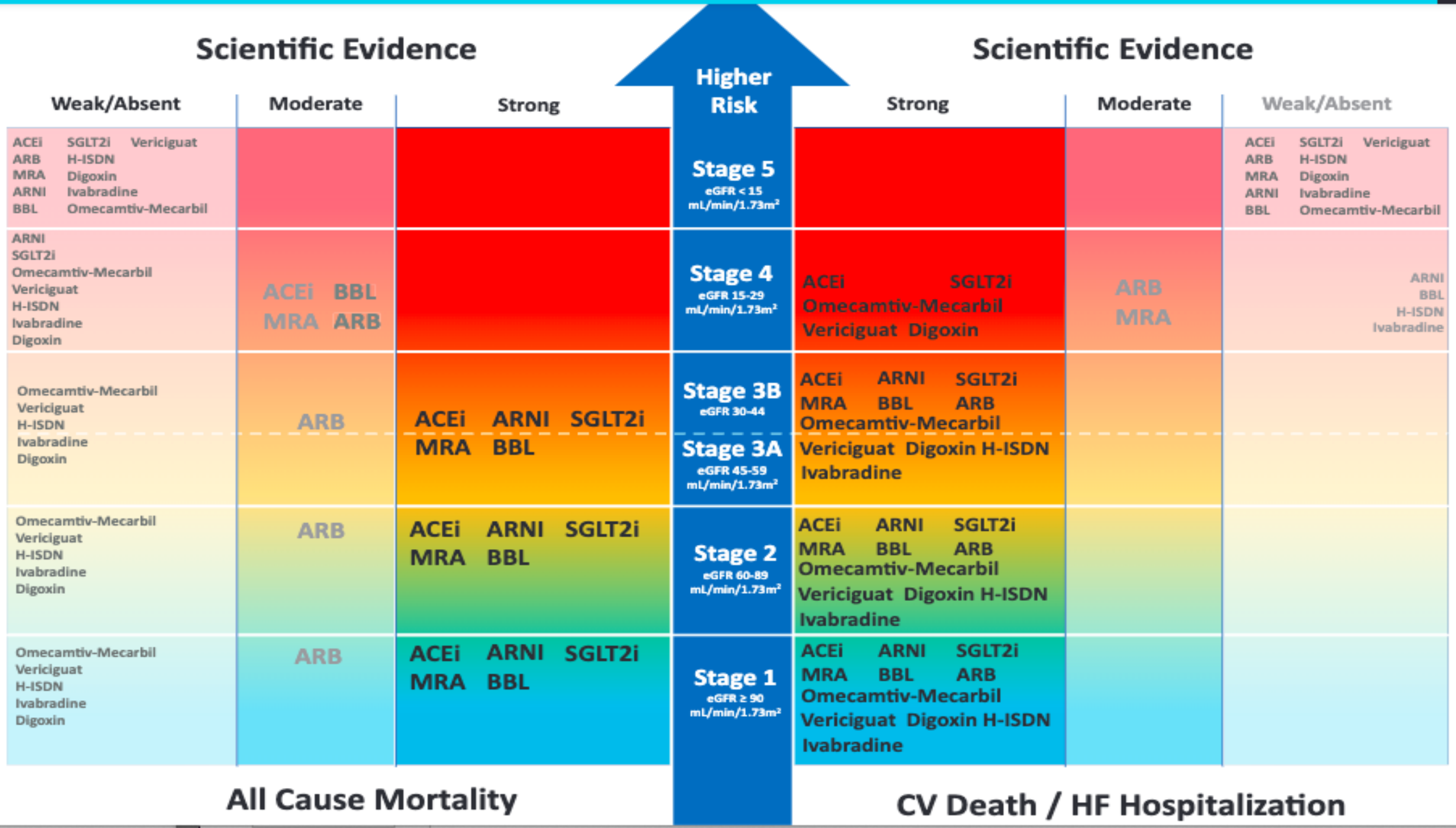
Current Problems in Cardiology

journal homepage: www.elsevier.com/locate/cpcardiol

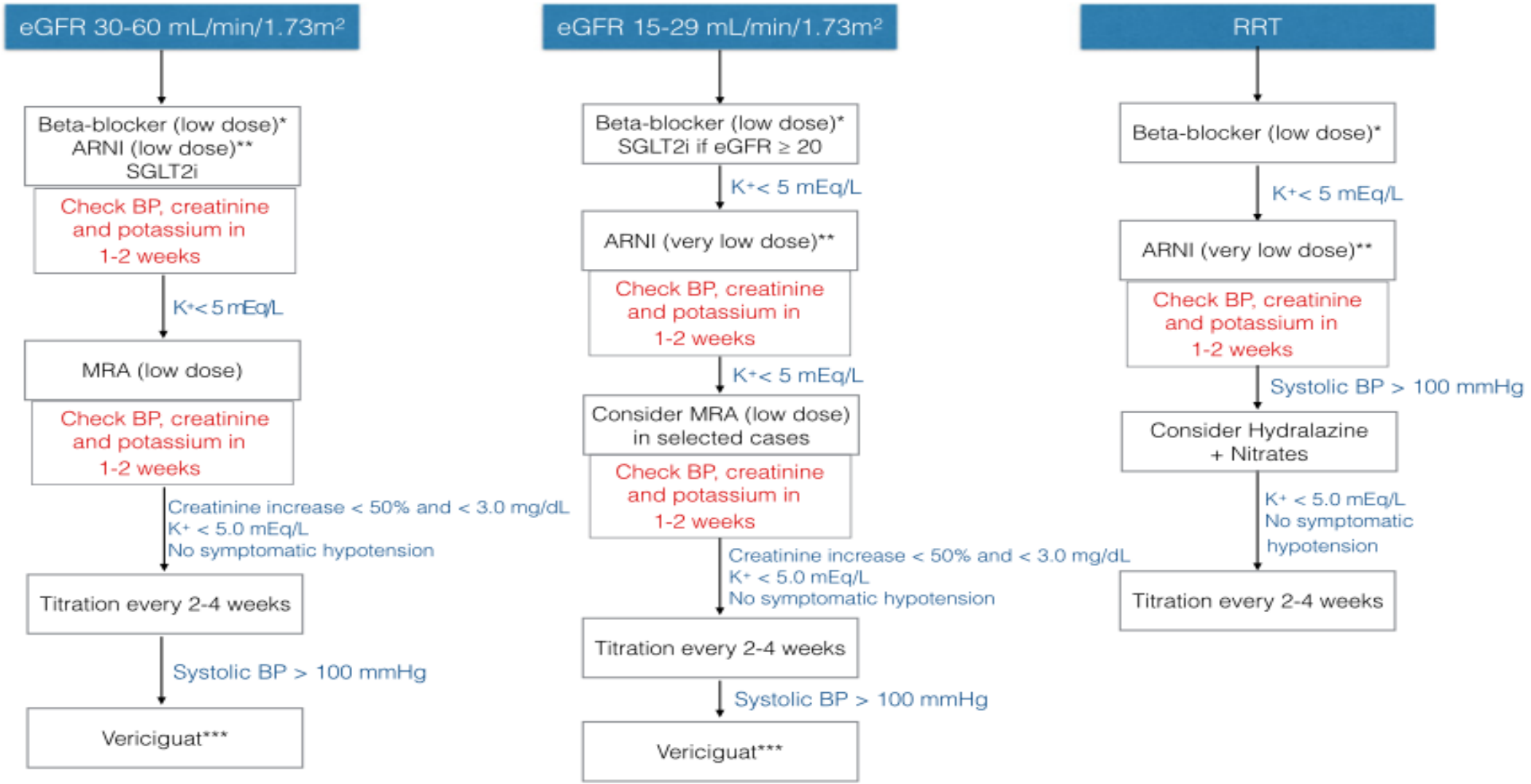
ELSEVIER

Invited Review Article

Beta-blocker therapy in heart failure with preserved ejection fraction (B-HFpEF): A systematic review and meta-analysis


Scientific Evidence
Scientific Evidence
Higher Risk
Weak/Absent
Moderate
Strong
Strong
Moderate
Weak/Absent
ACEi SGLT2i Vericiguat
ARB H-ISDN
MRA Digoxin
ARNI Ivabradine
BBL Omecamtiv-Mecarbil
Stage 5
eGFR < 15 mL/min/1.73m²
ARNI
SGLT2i
Omecamtiv-Mecarbil
Vericiguat
H-ISDN
Ivabradine
Digoxin
ACEi BBL
MRA ARB
Stage 4
eGFR 15-29 mL/min/1.73m²
ACEi SGLT2i
Omecamtiv-Mecarbil
Vericiguat Digoxin
ARB
MRA
ARNI
BBL
H-ISDN
Ivabradine
Omecamtiv-Mecarbil
Vericiguat
H-ISDN
Ivabradine
Digoxin
ARB
ACEi ARNI SGLT2i
MRA BBL
Stage 3B
eGFR 30-44
ACEi ARNI SGLT2i
MRA BBL ARB
Omecamtiv-Mecarbil
Stage 3A
eGFR 45-59 mL/min/1.73m²
Vericiguat Digoxin H-ISDN
Ivabradine
Omecamtiv-Mecarbil
Vericiguat
H-ISDN
Ivabradine
Digoxin
ARB
ACEi ARNI SGLT2i
MRA BBL
Stage 2
eGFR 60-89 mL/min/1.73m²
ACEi ARNI SGLT2i
MRA BBL ARB
Omecamtiv-Mecarbil
Vericiguat Digoxin H-ISDN
Ivabradine
Omecamtiv-Mecarbil
Vericiguat
H-ISDN
Ivabradine
Digoxin
ARB
ACEi ARNI SGLT2i
MRA BBL
Stage 1
eGFR ≥ 90 mL/min/1.73m²
ACEi ARNI SGLT2i
MRA BBL ARB
Omecamtiv-Mecarbil
Vericiguat Digoxin H-ISDN
Ivabradine
All Cause Mortality
CV Death / HF Hospitalization

HFrEF therapy depending on eGFR



* Evaluate blood pressure and heart rate before starting.

** Evaluate blood pressure, creatinine and potassium before starting. Initiate if systolic BP is > 100 mmHg and K < 5 mEq/L

*** In < 6 months from worsening HF. Titration every 4 weeks if systolic blood pressure is >100 mmHg.

Index Patient

- Diuretics Loop +Thiazides
- SGLT2 I
- Enteresto
- MRA
- B Blockers
- Had Hyperkalemia Was Given Sodium Zirconium Cryosilicate (Lokelma)
- General Measures
- Improved Symptoms
- Improved Renal Functions almost Reached Base line
- Discharged With an appointment CRT Device

CRT

- Cardiac resynchronization therapy (CRT) uses a special type of pacemaker called a biventricular pacemaker to treat heart failure.
- This pacemaker sends electrical pulses to make the ventricles pump at the same time.
- Cardiac resynchronization therapy with defibrillator (CRT-D) is preferred in patients who meet the established criteria. The need for cardiac pacing is increased three-fold in dialysis patients.

Review Article

Cardiac Resynchronization Therapy in the Cardiorenal Syndrome

Margot K. Davis and Sean A. Virani

Division of Cardiology, Gordon and Leslie Diamond Health Care Centre, The University of British Columbia, Vancouver, BC, Canada V5Z 1M9

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Journal of *Clinical Medicine*

Review

Cardiac Device Therapy in Patients with Chronic Kidney Disease: An Update

Bogdan Caba [1,2,†], Laura Vasiliu [1,2,†], Maria Alexandra Covic [1,*], Radu Sascau [1,2], Cristian Statescu [1,2] and Adrian Covic [1,3]

Asist Devices Continued

- MIRACLE TRIAL

Review Article

Cardiac Resynchronization Therapy in the Cardiorenal Syndrome

Emerging Individualized Approaches in the Management of Acute Cardiorenal Syndrome With Renal Assist Devices

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HIGHLIGHTS

- Renal assist devices are being developed targeting acute cardiorenal syndrome.
- These devices can target venous, arterial, and interstitial/lymphatic axis alterations.
- Ongoing trials are necessary to determine the overall risk benefit rate of these devices.
- Individualized phenotyping could help with identifying the optimal specific renal assist

Index Patient

- Patient was admitted again after 6 months.
- Same general Condition with superimposed Chest Infection.
- Had Hypotension Enteresto was stopped , Required Nor adrenaline.
- Had Progressive Worsening of Renal Functions with Oliguria despite maximum doses of Diuretics (Sequential blockade). Electrolytes were corrected.
- Diuretic Resistance HIGH INCIDENCE BAD PROGNOSIS.
- Had to be supported by CVVHF with ultrafiltration.Improved and is on Permanent HDF

Kidney Replacement in CRS

- DECONGESTION IS THE CORNER STONE
- Uremic Milleu. Refractory Hyperkalemia
- Severe and refractory Metabolic Alkalosis Hypokalemia
- Pre and Post Opeartive

Cardiorenal Medicine

Review Article

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Kidney Replacement Therapies and Ultrafiltration in Cardiorenal Syndrome

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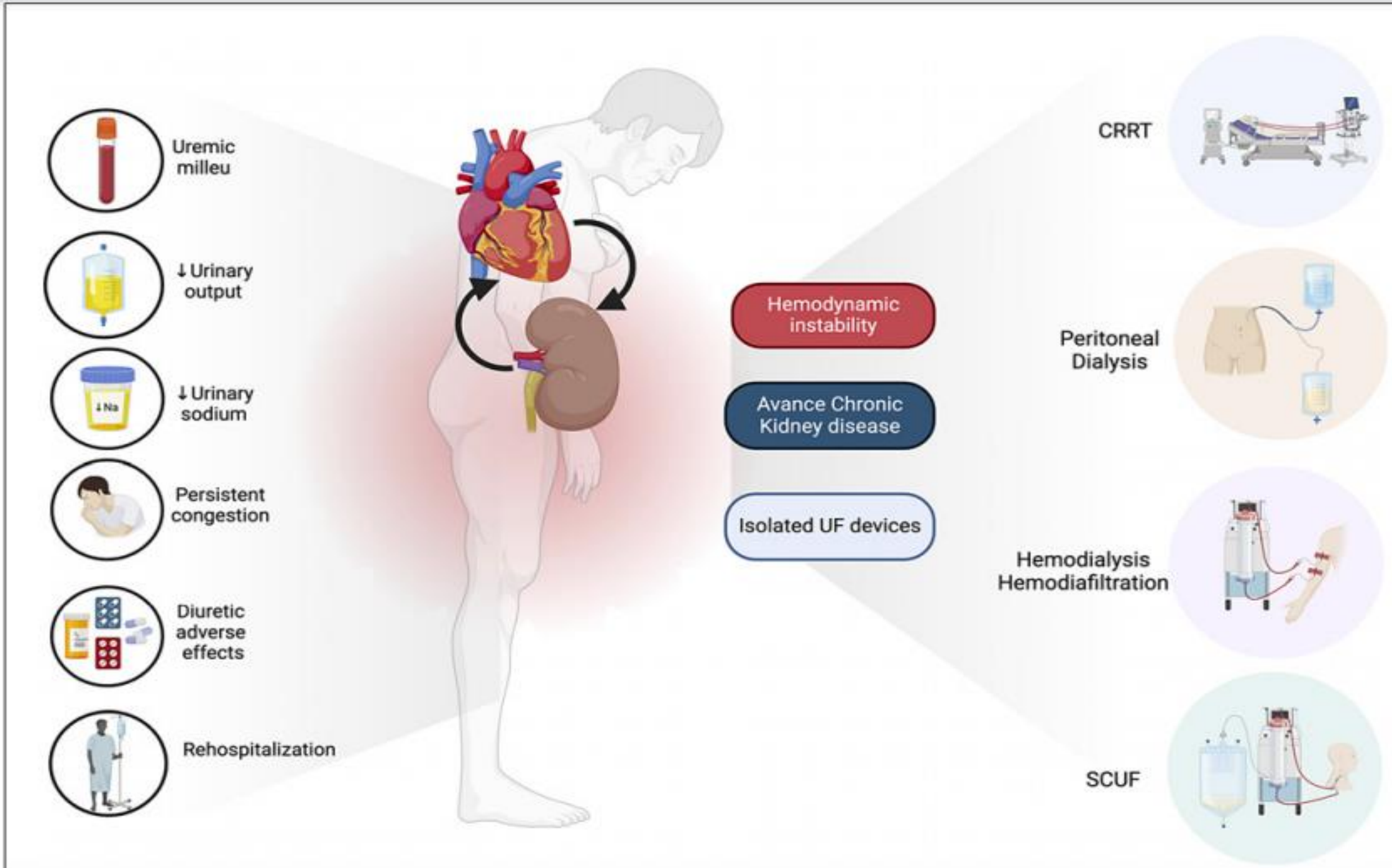
Jonathan S. Chávez-Iñiguez[a, e]

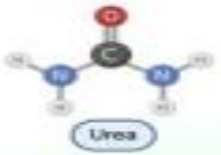

Uremic milleu
↓Urinary output
↓Urinary sodium
↓Na
Persistent congestion
Diuretic adverse effects
Rehospitalization
Hemodynamic instability
Avance Chronic Kidney disease
Isolated UF devices
CRRT
Peritoneal Dialysis
Hemodialysis Hemodiafiltration
SCUF

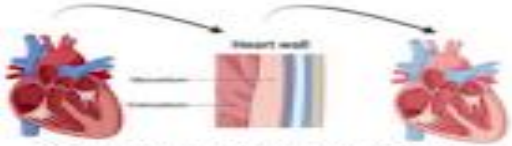
UF Or CRRT In CRS

Potential benefits of UF and KRT in CRS

↓ Hospital lenght of stay

↓ Uremic milleu

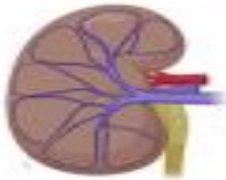




↑ Improve cardiac remodeling and ventricualr function

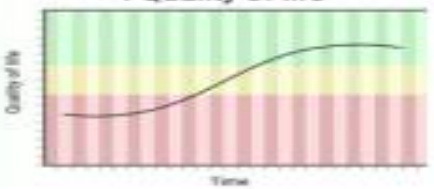
↑ Diuresis

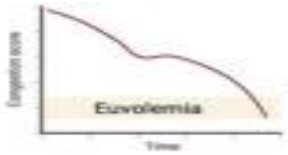
↓ Recurrent hospitalization



↓ Kidney venous congestion

↑ Quality of life

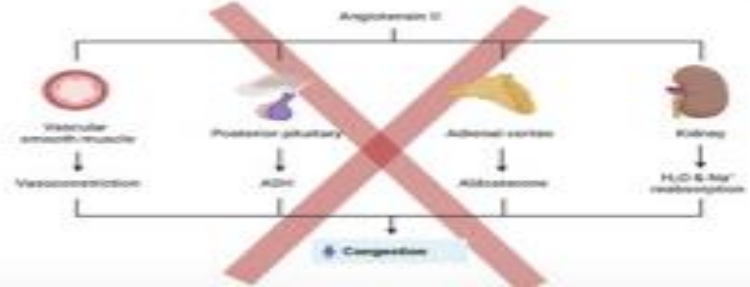




Rapid and adjustable decongestion

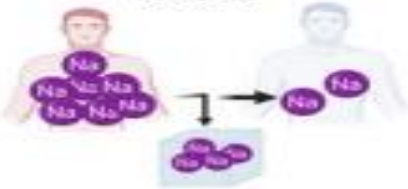
↓ Polypharmacy

Less neurohormonal activation



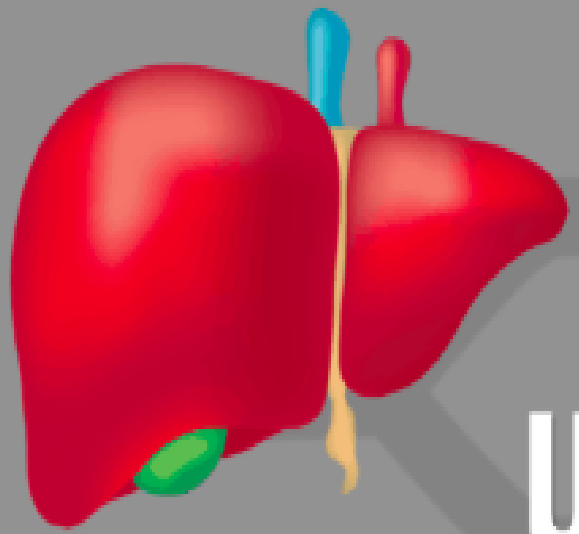
Easy correction of electrolyte and acid-base abnormalities

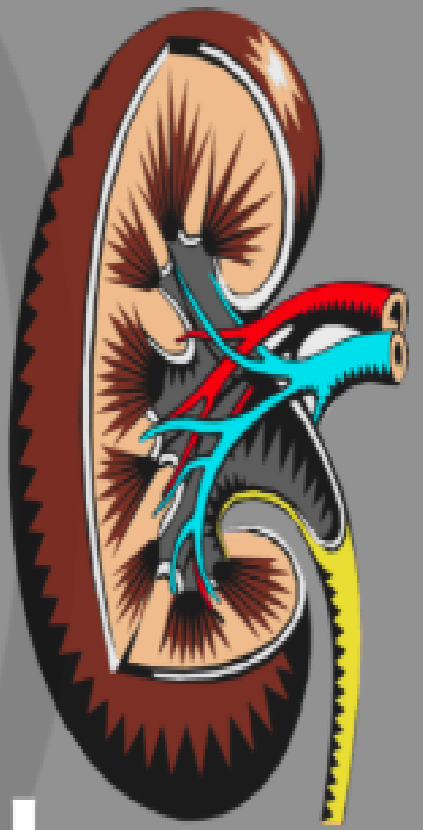
↑ Mass sodium removal




Thank
you
Doctors

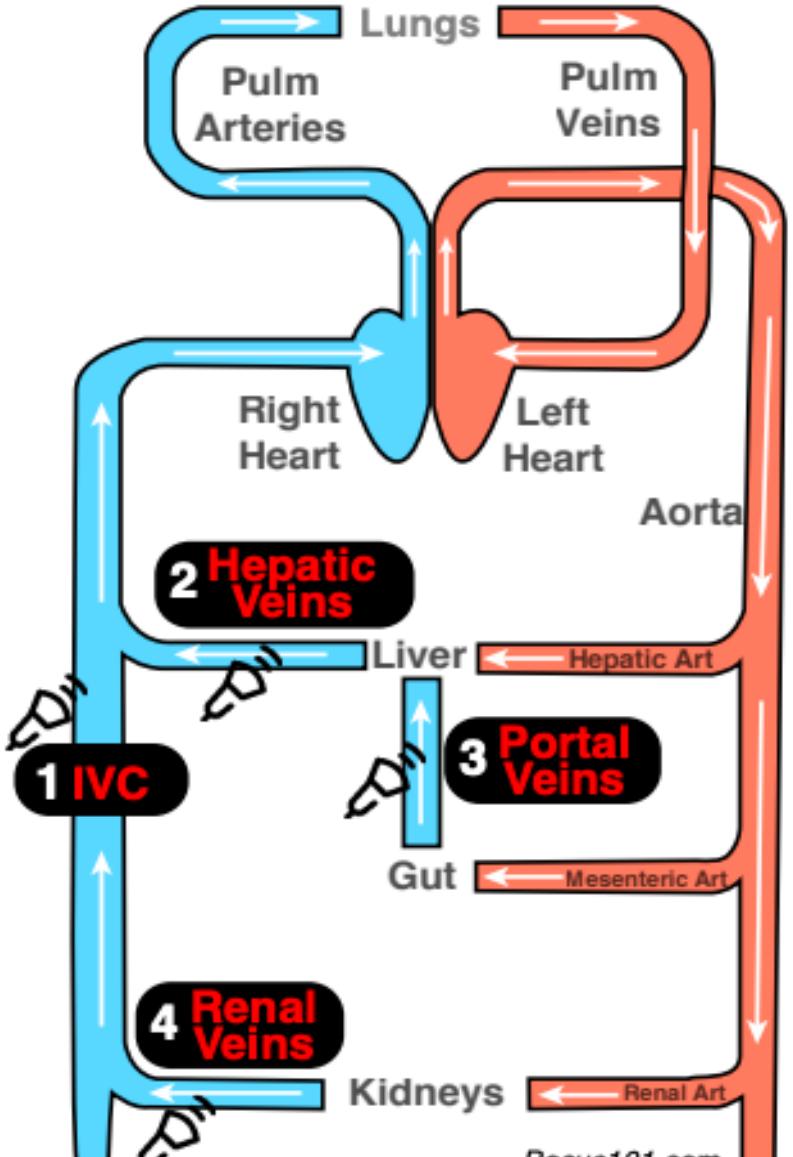
Venous Congestion Evaluation using Ultrasound





The VExUS Protocol

Venous Excess Ultrasound VExUS

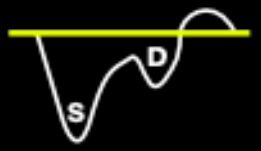


Step 1: IVC Diameter: If ≥2cm, proceed to step 2

Step 2: Hepatic Vein Doppler

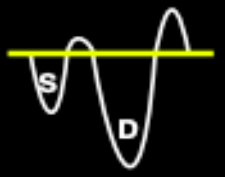
NORMAL

S>D



Mildly Abnormal

S<D



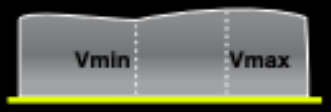
Severely Abnormal

S wave Reversal

Step 3: Portal Vein Doppler

NORMAL

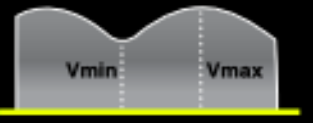
< 30% Pulsatility Index



*Pulsatility Index= (Vmax-Vmin)/Vmax

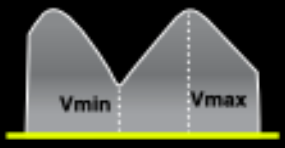
Mildly Abnormal

30-49% Pulsatility Index



Severely Abnormal

≥50% Pulsatility Index



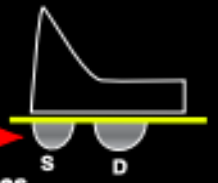
Step 4: Renal Vein Doppler

NORMAL

Continuous Monophasic Flow

Mildly Abnormal

Discontinuous Biphasic flow with Systolic/Diastolic Phases



Severely Abnormal

Discontinuous Monophasic flow with Only Diastolic Phase