Cardiorenal Medicine

Review Article

Cardiorenal Med 2024;14:320–333
DOI: 10.1159/000539547

Received: March 17, 2024
Accepted: May 13, 2024
Published online: May 29, 2024

Kidney Replacement Therapies and Ultrafiltration in Cardiorenal Syndrome

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Abstract

Background: Some patients with cardiorenal syndrome 1 and congestion exhibit resistance to diuretics. This scenario complicates management and is associated with a worse prognosis. In some cases, rescue treatment may be considered by starting kidney replacement therapies or ultrafiltration. This decision is complex and necessitates a profound understanding of these techniques and the pathophysiology of this syndrome. These modalities are classified into continuous, intermittent, and ultrafiltration therapies, each with its own advantages and disadvantages that are pertinent in selecting the optimal treatment. ***Summary:*** In patients with diuretic-resistant cardiorenal syndrome, extracorporeal ultrafiltration and kidney replacement therapies have the potential to relieve congestion, restore the neurohormonal system, and improve quality of life. ***Key Messages:*** (i) In cardiorenal syndrome, the resistance to diuretics is common. (ii) Extracorporeal ultrafiltration and renal replacement therapies are rescue options that may improve the management of these patients. (iii) Better understanding of these modalities will help the development of new devices which are friendlier, safer, and more affordable for patients in these clinical settings.

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Cardiorenal Syndromes with Congestion

Cardiorenal syndrome (CRS) depicts adverse interactions between the heart and kidney [1]. In this review, we will focus on CRS type 1, where acute heart failure can lead to acute kidney injury (AKI). This type is associated with five times higher mortality rates, which may persist for years after the index event [2]. The pathogenesis of CRS type 1 is multifactorial, and many factors can contribute to the development of AKI in acute HF, including uncontrolled activation of the renin-angiotensin-aldosterone system, inflammation, oxidative stress, the low cardiac output, and the increased venous pressure, with subsequent water and sodium (Na) retention [3]. These events conclude with vascular congestion and systemic interstitial edema. In the kidney, e.g., this phenomenon is called nephrosarca or renal congestion, which is associated with declining kidney and cardiovascular function [1]. These physiopathogenic mechanisms contemplate a more inclusive approach that acknowledges the interconnectedness of the cardiovascular system and the kidneys, not only CRS type 1. This innovative approach proposes a unified CRS, departing from categorizing patients based solely on subjective differences in clinical presentation. This broader perspective allows for greater applicability and relevance

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across various clinical scenarios, potentially advancing our understanding and management of cardiorenal disorders [1–4].

Although the diagnosis is usually clinical, some diagnostic tools can confirm CRS, such as findings of point-of-care ultrasonography findings (e.g., presence of pulmonary B lines [4], morphological changes of blood flow within the central venous system assessed by the VExUS score [5], or urinary and plasma biomarkers of acute myocardial or kidney damage [6, 7]). The primary treatment includes volume removal with diuretics, aiming for rapid and effective decongestion [8]. Consequently, functional improvement is expected in organs where high venous and interstitial pressure have negative repercussions [9]. To guide decongestion, knowing hemodynamics and intravascular volume assessment, cardiac and kidney function evaluation, and the kinetics of different biomarkers is necessary [10].

Resistance to Diuretics in CRS

It is important to remember that only 15% of patients achieve decongestion within 3 days after admission to the hospital when only loop diuretics [11] are used. Indeed, diuretic resistance (DR) is associated with a significant worsening of the disease course and prognosis [12].

DR is a common complication during decongestion in CRS patients. Although there is no standardized definition, DR is often considered when there is an unsatisfactory rate of diuresis or absence of weight loss despite using optimized (ceiling dose) intravenous diuretic dosages [13–16]. These events occur more frequently in patients with more comorbidities. Therefore, it is not surprising that higher doses of furosemide have a greater association with mortality [17]. Consequently, there is an increasing need for more reliable decongestion strategies among DR patients. These strategies include sequential nephron blocking [18].

When approaching DR, one must ensure that the patient is hemodynamically stable without urinary obstruction. The next step is to determine the presence and severity of volume overload [19, 20]. Some of the classic causes of DR include decreased glomerular filtration rate, slow capillary intravascular refill rates, hypochloremia, and hypokalemia. These factors activate the neurohormonal system or decrease absorption of the oral diuretic at the intestinal level due to gut edema [21]. Approximately 70% of the variability in the diuretic response is likely determined by tubular factors [22]. Among those with long-term use of loop diuretics, distal tubular hypertrophy can also lead to further DR [22, 23].

Also, other factors, including reduced cardiac output, high Na intake, increased proximal Na absorption, or reduced delivery of diuretics to the distal nephron, hypoalbuminemia, albuminuria, or increased concentrations of organic ions, can lead to DR [24, 25] (Table 1). One of the most important triggers of extracellular volume overload is Na accumulation [26]. Therefore, inadequate natriuresis can lead to a positive Na balance and a higher risk of mortality [27]. Consequently, it has been suggested to try to achieve high natriuresis (usually >100 mmol/L per day) by optimizing the doses and combinations of diuretics. A calculator for predicting natriuresis (http://cardiorenalresearch.net./) can be used after urinary Na and creatinine measurement 2 h after the first intravenous loop diuretic administration. This equation has been shown to have high accuracy for detecting low natriuresis rates, with an area under the receiver operating characteristic curve of 0.90 [28].

The first step in managing DR is to double the dose of intravenous diuretics [29]. High doses of diuretics should start with a bolus to ensure that drug levels are above the desired therapeutic threshold. A sequential nephron blockade with different diuretics is recommended as the next step. The combination of diuretics with different mechanisms of action that seeks to optimize natriuresis and urinary volume and limit neurohormonal reactivation on different segments of the kidney tubule is reasonable. Other diuretics include carbonic anhydrase inhibitors (e.g., acetazolamide) [30], thiazide diuretics [31], SGLT2 inhibitors [32], collecting duct diuretics (e.g., spironolactone) [33], or antidiuretic hormone antagonists (vaptans) [34]. Using a combination of hypertonic saline with loop diuretics [34] or compressive lower extremity bandages [35] is another step in persistent DR management. Among patients with hypochloremia, chloride replacement with hypertonic saline or lysine chloride has been shown to be effective in increasing urine [34, 36] output. If the tubular causes of DR are addressed and patients remain resistant to diuretics, other causes of DR must be evaluated (Table 1) [24].

Kidney Replacement Therapy for CRS

Before the kidney replacement therapy (KRT) initiation in patients with CRS, it is essential to optimize the medical management. These patients present with

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Table 1. DR in CRS patients, its causes, physiopathogenic mechanism, and strategies to correct them

Cause	Mechanism/approach	Corrective strategies
Inappropriate diuretic dose	Lack of monitoring of response parameters, not considering the clinical context (in cases of ADHF)	Increase dose until goals are met (urinary output, natriuresis, decongestion, clinical improvement)
Lack of adherence	Measure therapeutic adherence	Identify the reasons for poor adherence
No drug intake	**Objective**: drug levels, tablet counting, telemonitoring, pharmacy databases	Family support network
	Subjective:	Assess attendance at medical appointments
	MARS	Assess therapeutic efficacy
	BMQ	Assess the adverse effects of diuretics
	DAI-10	
	MMAS-8	
High salt intake	A negative salt balance is not achieved due to "post-diuretic Na retention"	Natriuresis in 24 h should exceed salt intake. Low salt intake allows excretion to exceed intake, and a negative balance is achieved
	High salt intake compensates for initial natriuresis	
	Monitor natriuresis to establish average intake	
Pharmacokinetics		
Simultaneous intake with food	Delay in the absorption of furosemide, reducing its peak concentration	Take furosemide on an empty stomach and do not modify its administration schedule
Decreased absorption	Intestinal edema and low duodenal blood flow decrease peak plasma concentration levels	
Inadequate secretion into the tubule lumen	Competitive drugs in OAT	Intravenous high dose
Chronic kidney disease and heart failure	States with Na retention require a higher peak concentration to be effective	Drug interactions
	Circulating proteases can directly activate ENaC	Intravenous high dose
NSAIDs	They may compete for OAT transporters that affect the secretion of the loop diuretic at the tubular level	Review drug interactions
Probenecid	The initial decrease in the diuretic effect with a subsequent increase. Initially, there is a competition for OATs. Decreased clearance of furosemide	Substitute when possible
Nephrotic syndrome	Na retention, neurohormonal activation Hypoproteinemia	Targeted treatment of nephrotic syndrome
NSAIDs	Reduce synthesis of prostaglandins and renal vasodilation	Review drug interactions
Antihypertensive agents	Decreased renal blood flow and hypotension	Check blood pressure
Low renal blood flow	Na retention by limiting its filtration, increasing reabsorption, and decreasing diuretic delivery in the proximal tubule	Avoid hypotension Evaluation of renal blood flow by monitoring with Doppler ultrasound
Nephron remodeling	Hypertrophy and hyperplasia of the distal nephron lead to an increase in the Na reabsorption capacity Activation of the NaCl channel is partially mediated by aldosterone, which in turn activates ENaC Greater arrival of solutes to distal segments with increased transepithelial solute flow and a greater synthesis of new proteins Metabolic alkalosis and hypokalemia Objective evaluation through histological study markers of nephron remodeling	Multisegmental nephron blockage

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Table 1 (continued)

Cause	Mechanism/approach	Corrective strategies
Neurohormonal activation	It constitutes a mechanism of resistance but also adaptation to chronic use of diuretics to avoid excessive volume depletion (braking phenomenon)	Extracorporeal UF or PD techniques

BMQ, Beliefs About Medication Questionnaire; DAI-10, Medication Attitudes Inventory; MARS, Medication Adherence Reporting Scale; MMAS-8, Morisky Medication Adherence Scale; NSAIDs, non-steroidal anti-inflammatory drugs; ENaC, epithelial sodium channel; NaCl, sodium chloride; OAT, outer anionic channel; PD, peritoneal dialysis; UF, ultrafiltration.

unmet medical needs, including symptom burdens, poor quality of life, high mortality, and healthcare resource demands. Concurrent use of KRT on patients who do not respond to optimized medical management is strongly suggested [37].

Time of Initiation

Management of congestion has been the cornerstone of treatment for patients with cardiorenal syndrome 1 (CRS1). Consideration of KRT initiation in patients with difficult-to-manage congestion, particularly in the presence of persistent AKI, would likely lead to improved outcomes [38]. For example, cardiogenic shock accompanied by pulmonary edema in patients on mechanical ventilation with high oxygen demand despite diuretics may benefit from KRT initiation. The need for preprocedural decongestion in cardiac surgeries with the intention of improving the probability of wound closure and preventing postsurgical right heart failure is another example of when KRT initiation could be considered [39]. The worsening of kidney function evaluated by the increase in serum creatinine alone should not be the sole decision-making factor for KRT initiation [8]. Of note, an increase in the serum creatinine concentration by ≥0.5 mg/dL is considered to be permissible when aggressive diuresis in heart failure patients is considered as long as appropriate decongestion is achieved [8, 13]. Other possible indications for KRT initiation include the need to remove "dialyzable" solutes or the presence of diuretic contraindications [40, 41], e.g., severe and refractory metabolic alkalosis or hypokalemia which arises from loop diuretics and thiazides [42]. Choosing the optimum time of KRT initiation for CRS1 patients is similar to all other causes of AKI, i.e., to initiate KRT following medical management optimization and only for clinical indications. According to recent meta-analyses, the rate of mortality is similar between lead versus early initiation of KRT, while late initiation is associated with a lower risk of hypotension, hypophosphatemia, and long-term dialysis dependence [43–45]. Accelerated KRT initiation could be considered for postcardiac surgery patients with volume overload and/or earlier stages of AKI, in whom early initiation is shown to be associated with a lower risk of death [46, 47].

The three most common outcomes following KRT initiation in patients with CRS with DR include as follows: (1) following KRT initiation, patients are liberated from dialysis after recovery; (2) those who initiate and are maintained on KRT during follow-up; and (3) those who start KRT and died within the hospital. The urinary output in these three groups differs, based on a recent study, i.e., 800, 650, and 345 mL, respectively [48]. Therefore, a low urinary volume before KRT initiation is a strong marker of mortality risk. In CRS, the death rate is also associated with uremic toxin levels [49]. Therefore, the reduction of uremic toxins could lead to lower mortality risk [50]. A blood urea nitrogen >120 mg/dL could be considered a sufficiently acceptable threshold to start KRT [51].

One of the criteria for KRT initiation could be an ineffective natriuresis. It has been reported that natriuresis <87 mmol/L per day [52] or <65 mmol/L in spot urine after optimized doses of loop diuretics [53] is associated with a worse prognosis.

Indications and Benefits

Among the benefits found with the use of KRT in CRS is the removal of Na mass. The fluid that is removed by extracorporeal ultrafiltration (UF) is iso-osmolar with plasma and, therefore, contains more Na than a similar volume of urine following diuretic therapy [54]. Each liter of UF contains approximately iso-osmolar Na concentration. Diuretic-associated diuresis often contains half of the Na concentration in each liter of urine than the equivalent volume of UF [55].

Another potential advantage of UF is the control over the rate of volume removal. In the case of diuretics,

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individuals who receive loop diuretics alone, adding acetazolamide, hydrochlorothiazide, or empagliflozin on average, would add 250 mL, 375 mL, and 385 mL of additional urine volume, respectively [56]. This added urine volume may not be adequate for patients with heart and respiratory failure who may need more rapid volume removal rates. Therefore, on impending respiratory and complete heart failure, using extracorporeal UF in addition to diuretics may provide additional benefit.

Other potential benefits of extracorporeal UF in patients with CRS include improving renal hemodynamics by preventing the vicious cycle of DR and worsening heart failure, which leads to neurohormonal activation, persistent congestion, and electrolyte alterations. In comparison, UF could provide accelerated decongestion and shorter hospital admission [55, 57]. On the other hand, among patients with DR, decongesting kidneys with UF could enhance their response to diuretics. Therefore, UF could be used as an adjunct for diuretic therapy in patients with CRS.

Outcomes and Evidence

Several clinical trials evaluated the clinical impacts of extracorporeal UF compared with diuretic therapies, with conflicting results (Table 2). Notably, a recent meta-analysis demonstrated that compared with diuretics, UF led to greater fluid removal (−1,372 mL), more weight loss (−1.6 kg), a lower likelihood of chronic heart failure worsening (−47%), and a lower risk of rehospitalization (−66%) in patients with CRS [67].

Meanwhile, the decision to consider extracorporeal UF should be made carefully by calculating its related complications, including the need for dialysis catheter placement, which could lead to bleeding, pneumothorax, central vein stenosis, and infections. Using miniaturized devices that work with peripheral venous accesses could mitigate the risk of line placement. These devices are portable, with a blood volume capacity of only 33 mL and blood flow rates of 10–40 mL/min [68].

Modalities of KRT for CRS

Choosing the appropriate KRT modality for patients with CRS is essential. For example, for patients with hemodynamic instability, continuous therapies such as continuous renal replacement therapy (CRRT) or peritoneal dialysis (PD) may be better options. On the other hand, if there is a substantial need for solute clearance, e.g., severe hyperkalemia or severe metabolic acidosis, intermittent therapies such as hemodialysis or hemodiafiltration would be better choices. Prolonged intermittent renal replacement therapy is a hybrid hemodialysis modality that is longer than conventional hemodialysis (usually >6 h) and can be given intermittently for days, and offers diffuse solute clearance and a slow UF [58, 59].

When the goal of KRT is solely volume removal, UF, whether intermittently or with slow continuous ultrafiltration (SCUF), would be the option of choice. Knowing the strengths and weaknesses of each KRT modality is essential for enhancing the safety and effectiveness of the treatment. Unfortunately, regardless of the modality of KRT utilized in CRS patients, the mortality rates remain very high [58, 59], as shown in Figure 1.

PD UF in CRS

Using PD for CRS could provide some advantages. PD is a continuous KRT modality that does not require anticoagulation or central venous access. The peritoneal catheter can be placed by a nephrologist or surgeons.

In a clinical trial of patients with CRS, with a creatinine level of 3.1 mg/dL and an average left ventricular ejection fraction (LVEF) of 31%, tidal PD with 12–14 cycles per day, compared with SCUF with UF rates of 75–125 mL/h, demonstrated a better creatinine reduction, greater weight loss, and lower risk of hospitalization at 90 days [58]. In a cohort of 147 patients with CRS with a mean serum creatinine level of 4.0 mg/dL and a blood urea nitrogen concentration of 60 mg/dL, PD was associated with improved survival, especially when negative balances were reached in the first 5 days [59].

Several centers in Spain developed PD management algorithms for patients with chronic heart failure refractory to diuretics. In these centers, the nephrologist places the peritoneal catheter. When the kidney dysfunction is severe, their algorithms allow exchanges using a combination of dextrose and icodextrin. In cases with reasonable kidney function, the PD is done with only one exchange per day with icodextrin [69].

A meta-analysis of 21 cohort studies after the inclusion of 673 patients with CRS showed PD can lead to more weight loss (3.6 kg) in comparison with other modalities. In addition, PD resulted in less kidney function loss

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Table 2. Some studies of KRT in CRS

Study	Modality	Description	Objective	KRT	Results	Comments
PD						
Peritoneal and Urinary Sodium Removal in Refractory Congestive Heart Failure Patients [53]	DPCA	66 patients with CHF	Mortality and ADHF episodes through urinary and peritoneal Na removal	1–4 exchanges per day with 1.36 and 2.27% PD glucose solution or icodextrin based on kidney function	CAPD increased Na excretion and was associated with lower mortality and ADHF episodes	High Na removal identifies patients with lower cardiovascular risk. PD optimizes decongestion
Tidal PD versus UF in CRS1: A Prospective Randomized Study [58]	PDT versus UF	88 patients with SCR1, randomized to PDT or UF	Change in serum creatinine from baseline and LVEF at 72 and 120 h. Followed for 90 days after hospital discharge	DPT group: 20–25 L/day, filling volume of 1.5–2 L for 90–120 min dwell time 12–14 cycles/day UF group: blood flow rate 100–170 mL/min and UF rate 75–120 mL/h	UF was inferior to DPT with respect to changes in serum creatine and LVEF Net UF was greater in DPT Greater adverse events in the UF group DPT had fewer rehospitalizations for ADHF (14.2% vs. 32.5%)	The use of PDT was superior to UF for the preservation of renal function and improvement of cardiac function
Outcomes after Acute PD for Critical CRS1 [59]	PD	147 patients with CRS1, creatinine 4.0 mg/dL, and BUN 60 mg/dL	In-hospital and 30-day mortality UF and net water balance in the first 5 PD sessions	Filling volume of 1.5 L, 36 or 18 L volume/day, adjusted to cycles of 3–6 h depending on volume and metabolic profiles	30-day mortality of 73.4% The change in water balance in the first 5 days was different between survivors and non-survivors	PD was associated with better survival, especially if negative balances in the first days
PD in Patients with Refractory Congestive Heart Failure: A Systematic Review [60]	DP	Meta-analysis of 21 cohorts with 673 patients	Describe the risk-benefit ratio on PD use in CHF	PD techniques were not reported for all studies Glucose, icodextrin, and glucose + icodextrin were used	DP reduced weight (−3.6 kg) and reduced risk of loss of 5 mL/min in eGFR, and LVEF increased by 4%	The deterioration of functional class could be prevented, and 5 days of hospitalization per year could be saved
Extracorporeal techniques						
AVOID-HF [61]	UF adjustable versus diuretics	224 congestive patients	Time to first ADHF event 90 days after hospital discharge	Adjusted during fluid removal	UF rate 138 mL/h for 70 h	At 30 days, the UF group had fewer cardiovascular and ADHF events Changes in kidney function and mortality at 90 days were similar in both groups
CUORE [62]	UF versus standard therapy	56 patients with CHF	Rehospitalization for ADHF	UF until fluid removal greater than 2 L	UF reduces the risk of rehospitalization for ADHF by 86%	No differences in mortality
CARRES-HF [63]	UF versus stepped diuretic	188 patients with CRS1	Change in creatinine and body weight at 96 h	UF started 8 h after randomization, with an average duration of 40 h UF rate 200 mL/h with a negative balance of 3.4 L	Creatinine UF versus diuretic: increase of 0.23 + 0.70 mg/dL versus decrease of 0.04 + 0.53 mg/dL, respectively Change in body weight: 5.5 + 5.1 kg versus 5.7 + 3.9 kg	UF was associated with an increase in creatinine without improvement in fluid removal or clinical improvement compared with step diuretic therapy

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Table 2 (continued)

Study	Modality	Description	Objective	KRT	Results	Comments
UNLOAD [64]	UF versus intravenous diuretics	200 patients hospitalized for congestive ADHF	Weight loss and dyspnea in 48 h	UF rate up to 500 mL/h	Weight loss 5.0 + 3.1 kg versus 3.1 + 3.5 kg, and fluid loss 4.6 versus 3.3 L were higher in the UF group	In ADHF, UF produces greater weight and fluid loss compared to diuretics
RAPID-CHF [65]	Single UF session versus usual care	40 patients with ADHF	Weight loss after 24 h	8-h session with UF rate 500 mL/h	Fluid removal in 24 h of 4,650 mL in UF and 2,838 mL for usual care Weight loss of 2.5 kg and 1.86 kg in the UF and usual care, respectively	Early UF is well tolerated and allows weight and fluid loss
Apparent Paradox of Neurohumoral Axis Inhibition after Body Fluid Volume Depletion in Patients with Chronic Congestive Heart Failure and Water Retention [66]	UF	22 patients with CHF	Determine whether an intravascular volume deficit explains patterns that exceed the limits of a homeostatic response	UF 500 mL/h until right atrial pressure is reduced to 50% of the initial value	In UF, with a 20% reduction in plasma volume, there was a moderate decrease in cardiac output, norepinephrine levels, plasma renin activity, and aldosterone	UF improves cardiac indices and decreases neurohormonal activity

ADHF, acute decompensated heart failure; BUN, blood urea nitrogen; CAPD, continuous ambulatory peritoneal dialysis; CRS1, cardiorenal syndrome 1; CHF, chronic heart failure; GFR, glomerular filtration rate; KRT, kidney replacement therapy; LVEF, left ventricular ejection fraction; PD, peritoneal dialysis; PDT, peritoneal dialysis tidal; UF, ultrafiltration.

(5.0 mL/min in the eGFR), improvement in LVEF by 4%, with less deterioration of the functional class, and about 5 days shorter hospital days per year [60]. These results are consistent with those of a recent meta-analysis that included 20 studies in which these findings were reaffirmed. The recent systematic review confirmed PD prevents declining eGFR, a 4% increase in LVEF, an improvement of 1.3 points in the functional class, and a reduction in hospitalization days, with a mortality of 37% per year [70]. One of the most attractive characteristics of PD is its efficacy in removing Na [71]. PD has limitations that must be considered: some centers do not use the catheter until a few days have passed for wound healing, the risk of peritonitis, and that it is not precise in the UF that is desired to obtain.

Extracorporeal Continuous UF versus Intermittent Hemodialysis in CRS

Extracorporeal UF in patients with CRS has been studied in the last three decades. One of the first studies demonstrated intense UF, defined as a UF of 500 mL/h until a decline in right atrial pressure by 50% and improved cardiac indices with subsiding neurohormonal activation [66]. In another study, intense UF of up to 4,800 mL per day markedly improved pulmonary hemodynamics, peripheral vascular resistance, and cardiac output [62].

In the CARRESS-HF clinical trial [63], patients with CRS were randomized to receive UF up to 4,800 mL/day (200 mL/h) versus those receiving adjustable diuretics. Patients in both groups lost the same amount of weight. However, in the UF group, there were more complications related to vascular access and rising serum creatinine levels. However, rising serum creatinine does not necessarily indicate an adverse outcome during volume removal among HF patients [63].

The ULTRADISCO clinical trial randomized 30 patients with CRS and DR to receive UF up to 300 mL/h compared to diuretics to achieve a urine output of >2,000 mL. The UF group exhibited greater weight and volume loss, with improvements in myocardial, hemodynamic, and neurohormonal metrics [72].

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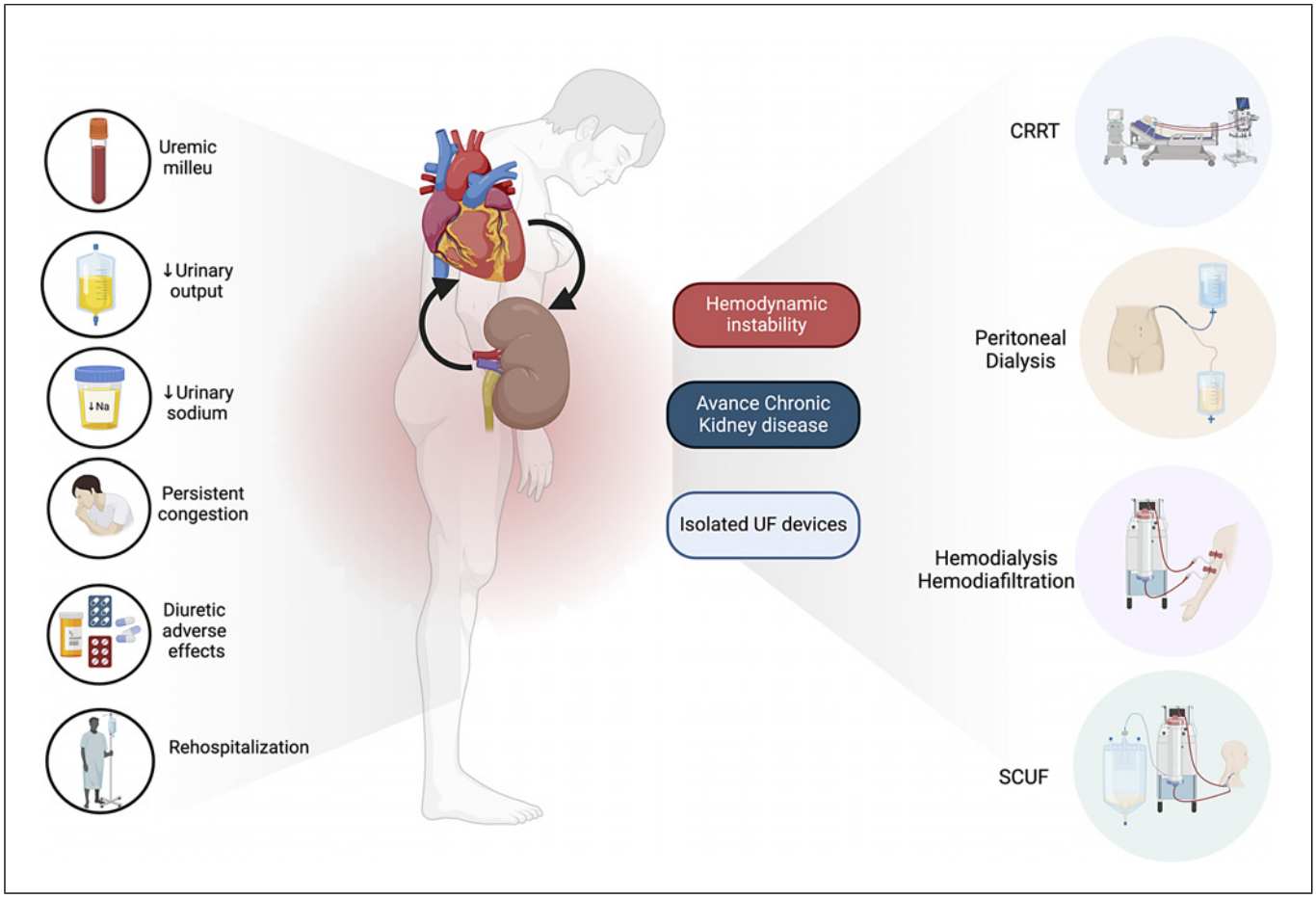


Fig. 1. Clinical scenarios for initiation of KRT in people with CRS. In patients with CRS, clinical scenarios may arise where the initiation of KRT should be considered, such as a uremic milieu, ineffective urinary volume for the predicted decongestion goals, or when adverse events occur from the use of diuretics and limit their continuity such as hypokalemia, metabolic alkalosis, or hypochloremia. In these cases, the choice of KRT will depend on the hemodynamic stability, predilection of the patient and their family, hospital logistics, and the realistic objectives set by the cardiorenal team. CRRT, continuous renal replacement therapy; KRT, kidney replacement therapy; SCUF, slow continuous ultrafiltration.

The AVOID-HF trial was discontinued due to budget deficits. However, among 110 CRS randomized patients who received adjustable UF versus diuretics, there were no differences between the groups in rehospitalization rates or the serum creatinine concentration. Still, the UF group had fewer days of hospitalization [61].

Devices have been developed that only perform UF through a peripheral catheter, which have been implemented in short-stay decongestion clinics in hospital units. The possibility of performing effective UF with portable devices has been successfully investigated for decades [73, 74]. The new miniaturized technologies allow UF for outpatients through an 11.5 French catheter with low doses of heparin and low blood flow (5–60 mL/h). This device enables the UF rate of 3–5 mL/min (1,800 mL in 6 h). It is portable and weighs 1 kg [75].

Continuous and Hybrid Renal Replacement Therapies for CRS

CRRT provides certain clinical benefits because of its slow and continuous nature, promoting improved hemodynamic stability, offering convective and diffusive techniques, and allowing a slow UF. Despite these benefits, the mortality rate of patients who receive CRRT for CRS remains high, at about 70%. In a Japanese cohort of 154 patients with CRS1 refractory to diuretics, CRRT was found to cause effective decongestion. Only 32% of patients survived, and just 12% recovered their kidney function. One of the reasons for these poor outcomes could be the inclusion of patients who had a higher risk of death, i.e., older than 80 years, receiving vasopressors,

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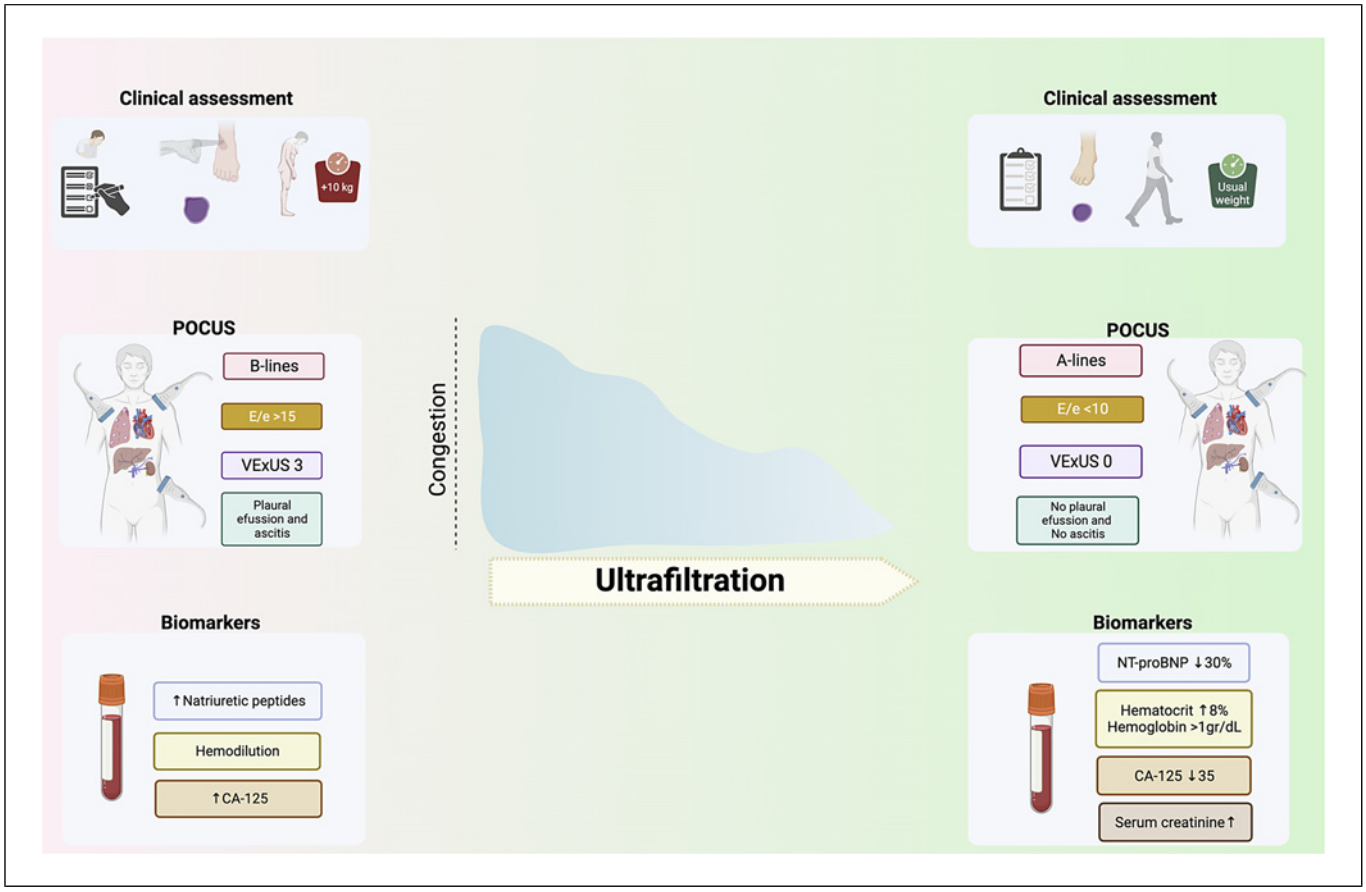


Fig. 2. Multimodality assessment of decongestion during UF in CRS. During decongestion with extracorporeal therapies, UF must be continuously evaluated and modulated through multimodality assessment, and it is the set of measurements that range from the clinical evaluation of symptoms, body weight, POCUS viewing central vein morphology, echocardiography, and the trajectory of laboratory variables that suggest decongestion, such as the reduction of natriuretic peptides, CA-125, increases in hematocrit, hemoglobin, and serum creatinine. POCUS, point-of-care ultrasound.

inotropic, and mechanical ventilation at the time of CRRT initiation [76].

Greater survival has been reported in patients who receive continuous venovenous hemofiltration in a cohort of 120 patients with CRS. When compared with SCUF, those with dilated cardiomyopathy were found to have improved modulation of systemic inflammation and cardiac function when they received continuous venovenous hemofiltration, a convective therapy [77].

Guide Extracorporeal UF in CRS Patients

The most common reason for starting KRT in CRS1 patients is persistent congestion [78]. Of note, inappropriate fluid removal rates could be associated with the risk of tissue hypoperfusion, organ damage, and mortality [79]. Hemodynamic instability and slow vascular refilling are among the risk factors of hypoperfusion during UF [80]. Several quality indexes are proposed to provide safe UF in different clinical contexts, e.g., AKI [81] or chronic HD [82]. In AKI, it is proposed that a net UF of 1.01–1.75 mL/kg/h is considered safe [81, 83]. In chronic HD, UF rates of 6.8 mL/kg/h and <10 mL/kg/h are associated with a lower risk of mortality [84].

In CRS1, some patients who have acute pulmonary edema require higher UF rates, which usually exceed the recommended safe rates for AKI and chronic HD. However, the urgency of the need for volume removal warrants promoting this momentary intense UF. Once the goals of relieving pulmonary edema or severe congestion have been achieved, to optimize UF, reducing

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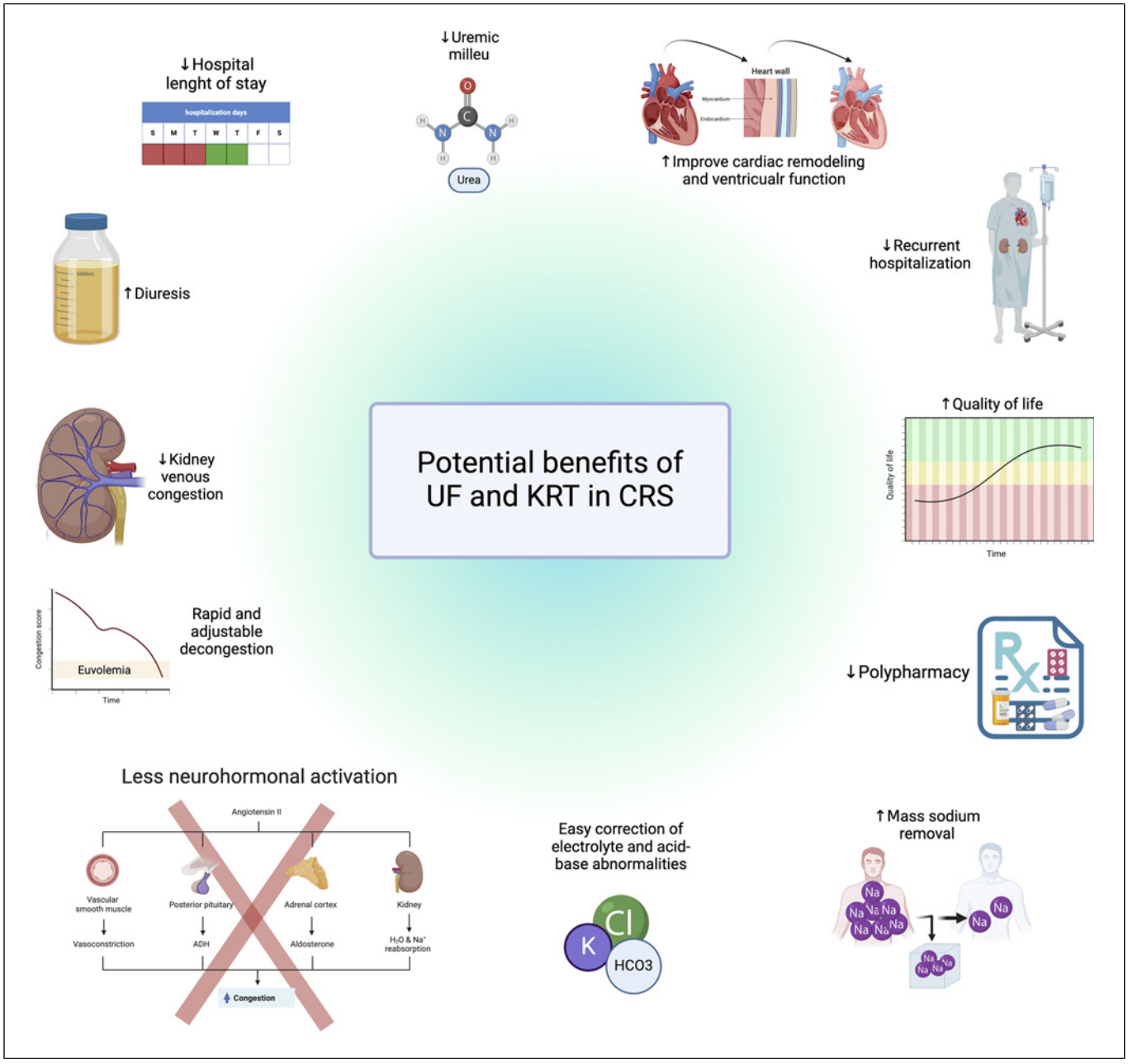


Fig. 3. Potential benefits of KRT or UF in patients with CRS. The use of KRT or UF in patients with CRS has demonstrated multiple benefits, which may be sufficient arguments to consider its use, especially in patients where diuretics have been insufficient for acceptably satisfactory management. ADH, antidiuretic hormone; K, potassium; Cl, chloride; CRS, cardiorenal syndrome; HCO3, bicarbonate; Na, sodium; KRT, kidney replacement therapy; UF, ultrafiltration.

temperature and increasing Na in the dialysate would help achieve UF, limiting hemodynamic instability. UF could be reduced to avoid hypoperfusion states.

Several tools could be used, such as serial point-of-care lung and cardiac ultrasound and determination of the VExUS score, CA-125 levels, natriuretic peptide, increase in hematocrit, serum creatinine, total protein or serum albumin levels, and bioimpedance in chronic settings [85–87], as shown in Figure 2. We do not know the clinical impact that the measurement of parameters

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related to systemic inflammation during KRT could have in this context, but it seems rational to continue exploring this association.

Liberation from KRT in CRS

KRT liberation in patients with CRS is challenging. Before KRT initiation, realistic objectives must be set, including appropriate rates and amounts of UF, goals of the removal of the uremic toxins, and controlling inflammatory states. Similar to the KRT initiation for other causes of AKI, liberation from KRT remains complex, with unknown optimal time. However, setting a-priori objectives could improve the care processes. Patients who are discharged from the hospital on KRT need close follow-up within the first 4 weeks for signs of potential kidney function recovery [88]. Ideally, a multidisciplinary team, including pharmacists, specialized nurses, rehabilitation specialists, psychologists, nutritionists, nephrologists, and cardiologists, should follow these patients [89]. Individualized HD or PD prescriptions to be adjusted based on the clinical progress, maintenance of decongestion and hemodynamic stability, and to promote the recovery of kidney function should be devised. Unfortunately, 82% of patients maintain the same dialysis prescription during follow-up [89].

We do not consider that there is sufficient evidence to support greater or lesser recovery of renal function with any modality of KRT or UF in people with CRS. In a cohort study, investigators demonstrated that a rehabilitation program consisting of individualized sessions, medication reconciliation, and patient education was associated with a 59% reduction in the risk of rehospitalization or death [90].

Finally, de-escalation of treatment should be considered when the objectives have not been satisfactorily met, and the care is considered futile. In this circumstance, palliative management should be implemented. In these scenarios, transitioning KRT to palliative care and symptomatic control could be reasonable. Patients with refractory cardiogenic shock, severe frailty, or cognitive impairment may benefit from this approach. It is important to consider patients' desire to stop or continue KRT should always take priority [37].

Conclusions

In patients with CRS, volume overload is common. A rapid and aggressive decongestion is often required. Factors such as comorbidities, DR, adverse drug events, or an increase in uremic toxins may prevent the achievement of appropriate decongestion. In these cases, initiating KRT modalities, e.g., PD, CRRT, HD, or SCUF, is reasonable. The choice of the KRT modality and time of initiation should be based on an individualized plan of care for each patient and available resources, as shown in Figure 3. Multidisciplinary cardiorenal clinics, the advent of drugs with benefits in primary cardiorenal objectives, and new miniaturized and portable UF devices can improve the quality of life of these patients.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Funding Sources

No fundings were received to develop this manuscript.

Author Contributions

All authors contributed similarly to the development of this manuscript.

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