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REVIEW

NONUREMIC INDICATION FOR PERITONEAL DIALYSIS FOR REFRACTORY HEART FAILURE IN CARDIORENAL SYNDROME TYPE II: REVIEW AND PERSPECTIVE

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Cardiorenal syndrome (CRS) type II is a serious condition in which chronic cardiac abnormalities cause worsening kidney function, leading to permanent chronic kidney damage. Management of CRS type II coupled with diuretic-resistant congestive heart failure (CHF) has been an issue of dispute. However, since the early 1990s, reports indicating the clinical usefulness of peritoneal dialysis (PD) as maintenance therapy for intractable CHF in this population have been accumulating. The present manuscript reviews the mechanisms by which kidney dysfunction develops within CHF, and then examines recent experiences of PD as chronic supportive therapy for intractable CRS type II, reviews the contributing mechanisms, and discusses the rationale for using PD as a new therapeutic approach in the nonuremic setting of CHF.

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KEY WORDS: Congestive heart failure; cardiorenal syndrome type II; nonuremic indications.

With an increasing number of patients worldwide developing both congestive heart failure (CHF) and chronic kidney disease (1–3), the co-existence of these two conditions has become a matter of concern. Congestive heart failure often accompanies decreased kidney function, and chronic kidney disease worsens pre-existing CHF. Cardiorenal syndrome (CRS) is a concept that classifies patients with cardiac and kidney dysfunction into four clinical types according to the basic mechanisms of the respective disorders (4):

- Type I: abrupt worsening of cardiac function leads to acute kidney injury
- Type II: chronic cardiac abnormalities cause worsening kidney function, leading to permanent chronic kidney damage
- Type III: abrupt worsening of kidney function leads to acute cardiac injury
- Type IV: chronic kidney disease causes chronic cardiac load, leading to permanent chronic cardiac damage

Regardless of type, CRS is a vicious cycle that results in clinical worsening of both kidney and cardiac function.

Ultrafiltration (UF) is a powerful nonpharmacologic, extracorporeal intervention for diuretic-resistant CHF (5). This therapy is best used in patients with acute decompensated heart failure (ADHF) in CRS types I and III. In contrast, the role of UF as a chronic maintenance therapy is less well known in patients with CRS types II and IV, except for uremic patients with end-stage kidney disease who have CRS type IV. Management of CRS types II and IV—and particularly CRS type II with diuretic-resistant CHF—has been an issue of dispute (6). However, since the early 1990s, reports indicating the clinical usefulness of peritoneal dialysis (PD) as maintenance therapy for intractable CHF in this population have been accumulating (7,8).

The present manuscript first reviews the clinical types of CHF and the mechanism by which kidney dysfunction

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develops within that disorder. It then examines recent experiences of PD as chronic supportive therapy for intractable CRS type II, reviews the contributing mechanisms, and discusses the rationale for using PD as a new therapeutic approach in the nonuremic setting of CHF.

CHF AND KIDNEY DYSFUNCTION

Pathophysiology of the Development of Kidney Dysfunction and CRS type II: Heart failure (HF) is clinically classified into two types: systolic and diastolic (9). Systolic HF is characterized primarily by decreased ejection fraction. It is often complicated by lesions from coronary ischemia. A therapeutic approach using beta-blockers and angiotensin-converting enzyme inhibitors has been established. Diastolic HF is characterized by decreased diastolic capacity, with relatively preserved ejection fraction; it is a common clinical occurrence that is complicated by aging, diabetes, and uremia. Notably, no pharmacologic agents have been proved effective for diastolic HF (9,10), and an increased risk of death in diastolic compared with systolic HF has been reported for patients complicated with CRS type II (11).

In terms of factors leading to the development of kidney dysfunction in CRS type II, several mechanisms have been proposed. The first is altered intrarenal hemodynamics. One of the primary physiologic responses in patients with CHF is maintenance of circulating volume against low cardiac output. During the course of CHF, the following pathologic process is hypothesized (12,13): In the early stage of HF, elevation of renin secretion and angiotensin II increases cardiac afterload because of arterial vasoconstriction. The sympathetic nervous system is activated by elevated angiotensin II, and baroreceptors in the aorta and aortic arch are activated because of hypoperfusion. Those two changes result in vasoconstriction of the afferent arterioles in the kidney, leading to enhancement of sodium reabsorption in the proximal tubules. On the other hand, serum arginine vasopressin markedly increases because of nonosmotic baroreceptor-mediated release from the posterior pituitary, activating water reabsorption via V2 receptors in the collecting duct (14). In addition, increased arginine vasopressin stimulates the V1a receptors of the vascular smooth muscle cells, leading to vasoconstriction of the arterial and venous systems and an increase of preload and afterload (12). These nervous and hormonal changes all accelerate to shift intrarenal hemodynamics from the superficial to the juxtamedullary nephrons (15). The latter change increases oxygen consumption in the thick ascending limb of Henle, rendering this hypoxia-susceptible area at increased risk of hypoxic injury and thus development of tubular damage by prolonged hypoperfusion (16).

The second factor is renal congestion. The extent of congestive symptoms in CHF does not necessarily correlate with total fluid volume, and it is known that symptoms develop even in patients who are not overhydrated. The mechanism of congestion includes changes in preload and afterload to the heart caused by increased vascular resistance or decreased reservoir volume of the vasculature, or both. Patients with CHF often have increased arterial resistance and stiffness, and decreased reservoir capacity may also be involved (17). Recent studies have highlighted the clinical significance of kidney congestion as a crucial factor for the exacerbation of kidney function in CHF (18,19). It has been shown that central venous pressure, rather than cardiac output, is closely linked to serum creatinine levels in patients with CRS type II (18), indicating that kidney congestion leads to a decrease in renal plasma flow. Based on these insults triggered by CHF, ischemic lesions might develop in the kidneys. In fact, interstitial inflammatory cellular infiltration and increased fibrosis in the medulla, but only faint abnormalities in the glomerulus, are noted in chronic CHF (20), indicating the significant role of persistent hypoperfusion and hypoxia in the medulla as pathologic factors in the development of CRS type II (21).

Critique of Diuretics and UF Method for CRS type II: Based on the aforementioned pathologies, several clinical factors should be considered in the management of CHF.

The first is the adverse effects of diuretics. The loop diuretic furosemide has been a mainstay in relieving the vicious pathologic cycle of CHF, although evidence supporting its use is lacking. It is supposed that furosemide helps to ameliorate congestive symptoms by decreasing excess fluid accumulation and by directly increasing the venous reservoir (22), a unique ability that helps to improve congestive symptoms before diuresis starts. However, some metabolic adverse effects of diuretics such as hypokalemia and hyperuricemia (23) have to be addressed. Those adverse effects may potentially contribute to excess morbidity and mortality in CHF by increasing the risk for progressive decline in kidney function. Furthermore, in excess, diuretics could activate the renin–angiotensin and the sympathetic nervous systems, which adversely alleviate diuretic effects by decreasing delivery of furosemide to the tubules (24). The unfavorable effects of diuretics on these refractory patients require use of a treatment strategy other than diuretics.

The second clinical point to be addressed is the practical aspect of the extracorporeal UF method. As mentioned, the UF method plays a pivotal role in treating

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diuretic-resistant refractory HF, particularly in patients with ADHF. Silverstein *et al.* originally used extracorporeal UF in the mid-1970s to treat severe fluid overload (25). Since then, the application of venovenous hemofiltration in patients with ADHF has become common. Recently, a randomized controlled study—the UNLOAD trial—showed the clinical significance of UF compared with diuretic therapy with respect to a reduction in unplanned hospitalizations after treatment of ADHF (26). However, it is not realistic to apply the UF modality outside of hospital as a supportive therapy to prevent worsening of CHF without hampering the patient's quality of life at home.

PD APPLICATION IN CHF

Clinical Outcomes of PD for CRS Types II and IV with CHF: In contrast to UF, PD is not an established therapeutic option for CHF. However, the therapeutic history of PD for treating CHF is longer than that of venovenous hemofiltration. Schneierson (27) originally applied peritoneal irrigation to treat intractable edema of cardiac origin during the 1940s. Since then, accumulating case series have shown a benefit of PD in managing CHF (28–53), although most studies remain descriptive and do not reveal the precise mechanisms of correcting and preventing CHF.

Case series in which PD was applied are classified into two clinical groups by kidney function (Table 1): patients with end-stage renal disease (39,40,43,46,47,51,53) and nonuremic pre-dialysis patients (37–40,42–46,48–53). The former group includes patients with CRS type IV, and case reports for those patients indicate that PD induction can benefit end-stage renal disease patients complicated with severe CHF. With respect to the clinical impact of PD in chronic dialysis patients, a conflicting series was reported from France, showing poorer outcomes in PD patients with CHF than in hemodialysis

TABLE 1
Reports About the Application of Peritoneal Dialysis for Congestive Heart Failure in Cardiorenal Syndrome (CRS) Types II and IV

Reference	Pts (*n*)	CRS	ESRD	Kidney function at induction	Observation (months)	Clinical outcomes (NYHA grade) At entry	Final
Konig *et al.*, 1991 (37)	13	Yes	No	Cr: 2.7 mg/dL (range: 1.5 – 4.6 mg/dL)	6–67	IV (all)	II (all)
Tormey *et al.*, 1996 (38)	3	Yes	No	ND	18±10	IV (all)	II (all) Reduction in re-hospitalization rate
Stegmayer *et al.*, 1996 (39)	16	Yes	Yes	9 Uremic 7 Nonuremic	24 (average)	IV (*n*=10) III (*n*=6)	
Ryckelynck *et al.*, 1998 (40)	16	Yes	Yes	ND	15.6 (range: 4–33)	IV and III	Reduction in re-hospitalization rate
Elhalel–Dranitzki *et al.*, 1998 (42)	9	Yes	No	eGFR: 34±4 mL/min/1.73 m^2	17.3 (*n*=5 died) 16.2 (*n*=4 alive)	IV in all	Reduction in re-hospitalization rate
Sheppard *et al.*, 2004 (43)	5	Yes	No	ND	ND	4.0 (average)	3.1 (average)
Kagan *et al.*, 2005 (44)	11	Yes	Yes		5–45 (CRS pts) 13–45 (ESRD pts)		
Bertoli *et al.*, 2005 (45)	2	Yes	No	CCr: 25 and 30 mL/min	12 each	IV, III respectively	III, II respectively Icodextrin solution used once daily

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TABLE 1 (cont'd)

Reference	Pts (*n*)	CRS	ESRD	Kidney function at induction	Observation (months)	Clinical outcomes (NYHA grade) At entry	Final
Gotloib *et al.*, 2005 (46)	20	Yes	Yes	eGFR: 14.8±3.9 mL/min/1.73 m^2	12	IV (all)	II (all)
Takane *et al.*, 2006 (47)	16	No	Yes	Mean eGFR: 4.6 mL/min/1.73 m^2	12	III (all)	Improved in 87%
Phadke *et al.*, 2008 (48)	1	Yes	No	Cr: 2.3 mg/dL	ND	Improvement with PD in patient with right ventricular heart failure	
Prochnicka *et al.*, 2009 (49)	1	Yes	No	eGFR: 47.0 mL/min/1.73 m^2	7	IV	II
Basile *et al.*, 2009 (50)	4	Yes	No	Cr: 3.6±1.1 mg/dL	11–43	IV (all)	Improvement in all pts Icodextrin used once daily in 3 pts
Nakayama *et al.*, 2010 (51)	12	Yes	Yes	CKD stages 3–5	Median: 26.5	IV in 3, III in 9	II in 3, I in 9
Sanchez *et al.*, 2010 (52)	17	Yes	No	eGFR: 35±6 mL/min/1.73 m^2	15±9	IV in 10, III in 7	III in 1, II in 13, I in 3
Cnossen *et al.*, 2010 (53)	24	Yes	Yes	eGFR: 15±10 mL/min/1.73 m^2	Median survival: 12 (range: 0.3–41)	Reduction in re-hospitalization rate	

Pts = patients; ESRD = end-stage renal failure; NYHA = New York Heart Association; Cr = creatinine; ND = not described; eGFR = estimated glomerular filtration rate; CCr = creatinine clearance.

patients (54). However, that study was a retrospective analysis, and no adjustment was made for clinical CHF score. In addition, the inclusion criteria for severe CHF and the preferential use of PD for CHF patients because hemodialysis was inconvenient in some cases could have affected the findings. Thus, the clinical role of PD for end-stage renal disease patients (CRS type IV), particularly those with severe CHF, should be separately discussed. On the other hand, the nonuremic pre-dialysis patients included some with CRS type II. At an outpatient clinic, PD was applied in those nonuremic patients to manage refractory congestive symptoms. Surprisingly, most patients recovered from a severely ill state (New York Heart Association III/IV) to regain their ability to take part in standard activities of daily living (New York Heart Association I/II). The clinical benefits have been very promising from the viewpoints of reduced hospitalization (38,40,42,53), reduction in the need for diuretics (43), and notably, improved quality of life (39,41,42,52).

Mechanism of Therapeutic Action of PD: The possible mechanisms of clinical improvement in HF through PD seem to be multifactorial. First, PD continuously draws ultrafiltrate; its physiologic effect therefore has a lesser risk of abrupt hypotension that would exaggerate organ hypoxia and kidney damage. Second, UF in PD is driven by the osmotic power of the PD solution (glucose or glucose polymer) indwelling within the peritoneal cavity which is drained through the extended network of microvessels in the visceral and parietal peritoneum (55,56). This unique process of ultrafiltrate driving fluid from the compartment of microvessels to the peritoneal cavity might decrease the amount of interstitial edema. That mechanism notably highlights the potential of PD to mimic a physiologic state similar to that of expanded peripheral microvessel capacity and increased vascular compliance. This characteristic of PD closely matches the therapeutic principle of CHF treatment: that is, to lessen kidney and central venous congestion. Third, the

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metabolic effects of PD therapy—such as glucose load from the solution, and correction of acidosis—favor the correction of nutrition and anemia. In fact, the notion of cardiorenal–anemia syndrome was proposed based on clinical experiences in PD patients (57). Fourth, the removal of proinflammatory factors (for example, tumor necrosis factor α and cardiac depressant factor) into the PD fluid might improve cardiac function (58). Finally, PD preserves residual kidney function by slowing fluid removal, leading to less stimulation of the renin–angiotensin system or the sympathetic nervous system, or both (51,52).

In respect to Na removal, excretion of excess Na from the body is supposed to play a crucial role in reducing congestive symptoms (59). In most patients, furosemide renders urinary Na levels hyponatric within the 60 – 70 mmol/L range (60). In addition, PD renders UF fluid hyponatric because of the sodium-sieving effect of the peritoneum through aquaporin (61). However, the ultrafiltrate Na level is about 100 mmol/L (62), which is higher than that in urine produced by furosemide. That difference might contribute to the correction of excess Na retention in patients with CHF.

Rationale for PD in a Nonuremic Indication for CHF: Accumulating case series have indicated the clinical merit of PD in patients with CHF, and this unique and important contribution of PD may play a significant palliative role for these severely ill patients, as indicated by the report that hope among hospitalized patients with CHF is stronger than it is among healthy subjects (63). In addition, use of PD may benefit patients by preventing decline into end-stage renal failure, which results in excess hospitalization and related costs.

Regarding the PD prescription, icodextrin solution is a long-acting osmotic agent that allows the patient's UF volume to gradually increase for up to 12 hours (55,64) and might contribute to a PD-based therapeutic strategy for CHF (45,50). In nonuremic patients, the simple use of this solution once daily could benefit patients or caregivers by reducing their burden at home. With respect to a pragmatic way to achieve isonatric UF removal by PD, hyponatric PD solution permits isonatric fluid removal (65) and is expected to be studied in patients with CHF (66).

After a review of the reported case series, it appears that PD-related complications such as peritonitis, malnutrition because of protein loss, increased intra-abdominal pressure, and socio-economic influences are minimal. Thus, it seems that the benefits of PD therapy outweigh its potential risks.

In consideration of the growing number of patients with CRS type II, studies including prospective analyses to elucidate the clinical significance and possible risk of PD in patients with CHF are warranted. The results of such studies may expand the role of PD in the coming decades as a novel therapeutic modality for severe CHF.

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DISCLOSURES

The author has no financial conflicts of interest to declare.

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