

# Journal of Nephropathology



## Peritoneal dialysis versus hemodialysis in end-stage kidney disease patients with congestive heart failure: A comparative review

Irvan Rahmat Amanu<sup>1</sup><sup>✉</sup>, Jonny Jonny<sup>2,3,4,5</sup><sup>✉</sup>, Adrianus Jonathan Sugiharta<sup>2</sup><sup>✉</sup>

<sup>1</sup>Cardiology and Vascular Medicine, University of Indonesia, Jakarta, Indonesia

<sup>2</sup>Department of Internal Medicine, Gatot Soebroto Central Army Hospital, Jakarta, Indonesia

<sup>3</sup>Faculty of Medicine, Prima University, Medan, Indonesia

<sup>4</sup>Faculty of Military Medicine, Indonesia Defense University, Bogor, Indonesia

<sup>5</sup>Faculty of Medicine, Jakarta Veterans National Development University, Jakarta, Indonesia

### ARTICLE INFO

*Article type:*  
Review

*Article history:*

Received: 5 May 2025

Revised: 27 May 2025

Accepted: 14 Dec. 2025

Published online: 26 Jan. 2026

*Keywords:*  
Peritoneal dialysis  
Hemodialysis  
Heart failure  
Chronic kidney disease

### ABSTRACT

Congestive heart failure (CHF) is a major cause of morbidity and mortality in patients with end-stage kidney disease (ESKD), where fluid overload often necessitates kidney replacement therapy. While both hemodialysis (HD) and peritoneal dialysis (PD) are viable options, PD has been suggested to offer hemodynamic advantages due to its gradual ultrafiltration process. This review examines the comparative effects of PD and HD in ESKD patients with CHF undergoing maintenance dialysis, focusing on hospitalization rates, cardiac function, survival outcomes, and volume management. Several studies suggest that PD is associated with reduced hospitalization rates, particularly in diuretic-resistant CHF patients, and improved left ventricular ejection fraction (LVEF), especially in those with heart failure with reduced ejection fraction (HFrEF). Additionally, PD's continuous ultrafiltration may lower the risk of intradialytic hypotension (IDH) compared to HD. However, survival outcomes remain inconsistent, with some studies reporting higher mortality in PD patients, likely due to selection bias, as PD is often used in hemodynamically unstable CHF patients. Despite these findings, there is no definitive consensus on whether PD offers a survival advantage over HD in CHF patients. Given the limitations of existing studies, further large-scale, prospective research is required to determine the optimal dialysis modality for CHF patients with ESKD and to clarify its impact on long-term clinical outcomes.

### Implication for health policy/practice/research/medical education:

This review highlights the potential benefits of peritoneal dialysis in patients with end-stage kidney disease (ESKD) and congestive heart failure (CHF), particularly in improving volume management and reducing hemodynamic instability. These findings support more individualized dialysis modality selection and emphasize the need for further research to guide clinical decision-making in this high-risk population.

*Please cite this paper as:* Amanu IR, Jonny J, Sugiharta AJ. Peritoneal dialysis versus hemodialysis in end-stage kidney disease patients with congestive heart failure: a comparative review. J Nephropathol. 2026;15(2):e27644. DOI: 10.34172/jnp.2026.27644.

### Introduction

Heart failure (HF) remains a significant global health burden, affecting approximately 26 million individuals worldwide, with projections indicating a prevalence exceeding eight million in the United States by 2030 (1-4). The economic impact is also profound, with HF management consuming up to 2% of national healthcare budgets in developed countries and contributing to an estimated \$108 billion in global healthcare expenditures

(5,6). Hospitalization and readmission rates also remain high, primarily due to congestion-related complications, and despite the widespread use of diuretics, a subset of patients develops diuretic resistance, necessitating alternative volume management strategies (1,4,7,8).

In patients with end-stage kidney disease (ESKD), congestive heart failure (CHF) is associated with poor survival in patients with a 25%-35% additional risk of death (9). Although diuretics remain the cornerstone

*\*Corresponding author:* Jonny Jonny,  
Email: jonnyunhan@idu.ac.id

of pharmacologic therapy in CHF, congestion may become refractory as the disease progresses, even with intensive diuretic treatment (7,10,11). While both hemodialysis (HD) and peritoneal dialysis (PD) serve as kidney replacement therapies, PD has emerged as a potential alternative due to its slower and more physiologic ultrafiltration (UF) profile, which may mitigate intradialytic hypotension (IDH) and improve hemodynamic stability. PD may also reduce hospitalization rates and improve functional status in CHF patients with refractory volume overload (7,12,13). However, evidence regarding its impact on survival remains inconclusive, with some reports indicating a potential survival disadvantage compared to HD, possibly due to patient selection bias and differences in cardiovascular risk profiles.

Therefore, this review is aimed to critically examines current literatures comparing PD and HD in CHF patients with ESKD, focusing on hospitalization rates, cardiac function, survival outcomes, and treatment efficacy. By synthesizing existing evidence, we aim to clarify the role of PD as a therapeutic strategy in this high-risk population.

## Discussion

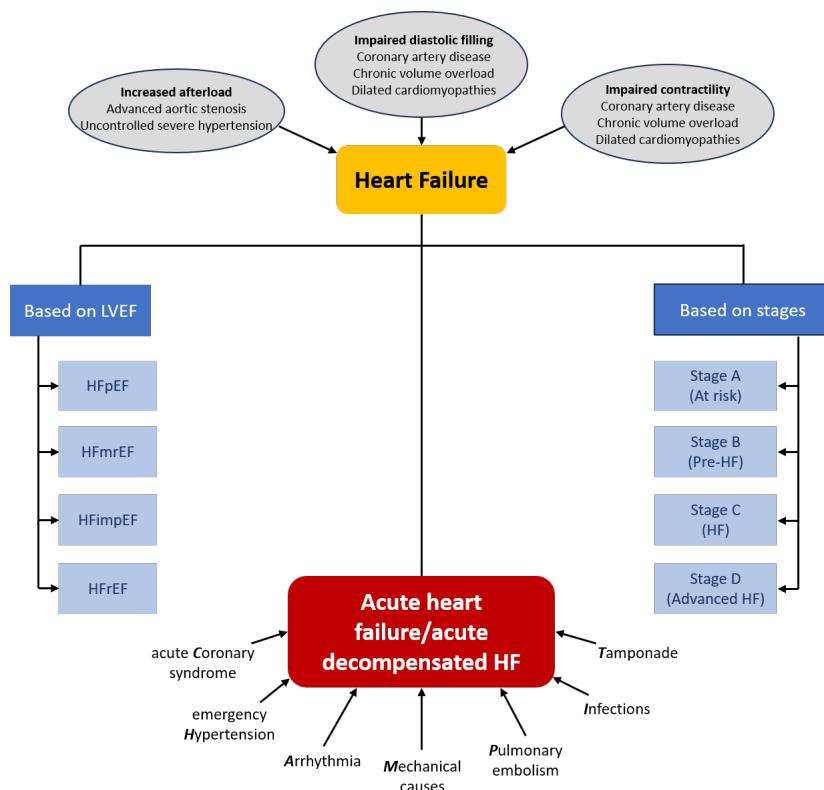
### Congestive heart failure

Congestive heart failure (CHF) is a clinical syndrome characterized by symptoms such as exertional dyspnea,

orthopnea, ankle swelling, and fatigue, often accompanied by signs of congestion, including elevated jugular venous pressure, pulmonary crackles, pulmonary edema, and ascites. CHF arises from impaired ventricular filling, contractile dysfunction, or hemodynamic derangements that lead to systemic congestion and reduced cardiac output (11,14).

Heart failure is classified based on left ventricular ejection fraction (LVEF) into four categories; 1) HF with reduced ejection fraction (HF<sub>re</sub>EF, LVEF <40%), characterized by impaired systolic function; 2) HF with mildly reduced ejection fraction (HF<sub>mr</sub>EF, LVEF 41%–49%); 3) HF with preserved ejection fraction (HF<sub>pe</sub>EF, LVEF ≥50%), primarily involving diastolic dysfunction; and 4) HF with improved ejection fraction (HF<sub>imp</sub>EF), defined as a prior LVEF <40% with subsequent recovery to >40% on follow-up (14,15).

The pathophysiology of HF is multifactorial and typically involves (a) impaired ventricular contraction and ejection, leading to systolic dysfunction; (b) increased afterload, which exacerbates myocardial workload; or (c) impaired ventricular relaxation and filling, resulting in diastolic dysfunction. Many patients exhibit overlapping features of both systolic and diastolic HF, contributing to diagnostic and therapeutic challenges (16). The etiology and classification of HF are summarized in Figure 1.



**Figure 1.** Etiology and classification of heart failure (14,15). LVEF; Left ventricular ejection fraction; HF<sub>pe</sub>EF: Heart failure with preserved ejection fraction; HF<sub>mr</sub>EF; Heart failure with mildly reduced ejection fraction; HF<sub>imp</sub>EF: Heart failure with improved ejection fraction; HF<sub>re</sub>EF: Heart failure with reduced ejection fraction.

In systolic HF (HFrEF), myocardial contractility is impaired due to myocyte dysfunction, fibrosis, or excessive afterload, leading to reduced stroke volume (SV) and increased end-systolic volume. As a compensatory response, preload increases via the Frank-Starling mechanism, initially helping maintain cardiac output. However, as the disease progresses, excessive ventricular dilatation leads to elevated left ventricular end-diastolic pressure (LVEDP), increased left atrial pressure, and pulmonary venous congestion, manifesting as dyspnea and peripheral edema. On the contrary, diastolic HF (HFpEF) is characterized by impaired ventricular relaxation and reduced compliance, leading to elevated filling pressures despite a preserved ejection fraction (EF). These patients are particularly sensitive to volume overload, as even small increases in intravascular volume can cause significant pulmonary and systemic congestion (16,17) (Figure 2).

Given these pathophysiological differences, dialysis modality selection for HF patients should be individualized based on cardiac function, volume status, and hemodynamic tolerance. Patients with severe systolic dysfunction or recurrent IDH may benefit from PD due to its gentler UF profile, whereas those with better hemodynamic stability may tolerate HD with careful UF management. However, survival differences between HD and PD remain debated, and patient selection biases often influence study outcomes (18-20). Further research is needed to determine the optimal dialysis strategy for these particular population.

### Dialysis

Dialysis serves as a kidney replacement therapy for ESKD patients, facilitating the removal of metabolic waste and excess fluid through diffusion and UF (21,22). While dialysis does not fully replicate native kidney function, it plays a crucial role in maintaining homeostasis in patients with severe kidney impairment. Generally, dialysis

initiation is considered when the estimated glomerular filtration rate (eGFR) falls below 15 mL/min/1.73m<sup>2</sup>, particularly in symptomatic individuals (21,23). However, the timing of dialysis varies across clinical guidelines and healthcare systems, reflecting differences in practice patterns and patient-specific factors (24-26).

### Hemodialysis

Hemodialysis is a therapy to remove additional fluid and waste products rapidly and balance electrolytes in patients with reduced kidney function. The basic summary of HD circuit is shown in Figure 3 (27,28).

Hemodialysis operates through three primary mechanisms; diffusion, UF, and convection. Diffusion allows small solutes, such as creatinine and urea, to pass through a semipermeable membrane, while larger molecules like albumin and red blood cells are retained (Figure 4a). The Gibbs-Donnan effect further influences ion movement across the membrane by attracting positively charged sodium ions to negatively charged proteins (Figure 4b). UF removes excess fluid by applying the law of hydrostatic pressure, allowing water to move across the membrane from areas of higher to lower pressure (Figure 5). Convection facilitates the clearance of middle and large molecular weight solutes, such as  $\beta$ 2-microglobulin, by dragging them along with fluid movement, particularly in high-flux dialyzers (Figure 6). These complex mechanisms help maintain electrolyte balance and fluid homeostasis in patients with ESKD (27,28).

During HD, rapid fluid removal (29) can lead to hemodynamic instability and various complications, particularly in patients with preexisting cardiovascular disease (21,30). As UF progresses, fluid shifts from the interstitial space into the vasculature to maintain blood volume until the patient's dry weight is reached. Under normal physiological conditions, compensatory mechanisms, such as increased vascular resistance,

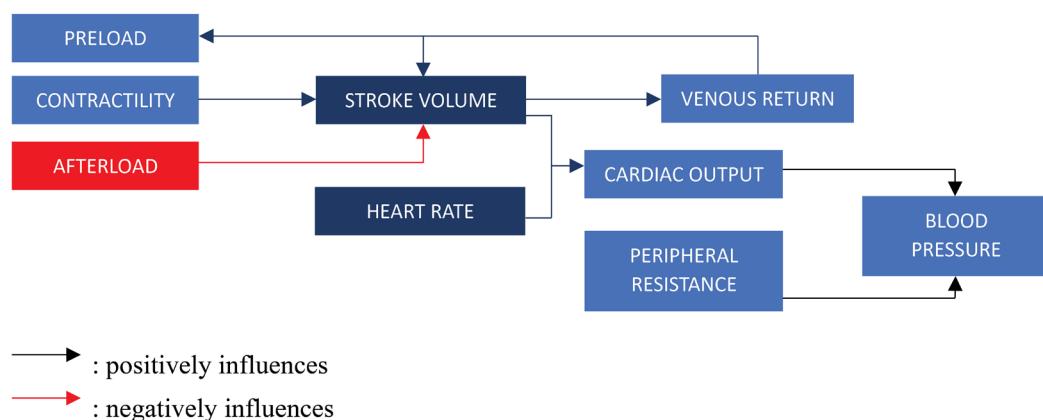


Figure 2. The intricate relationship of various factors responsible for cardiac function.

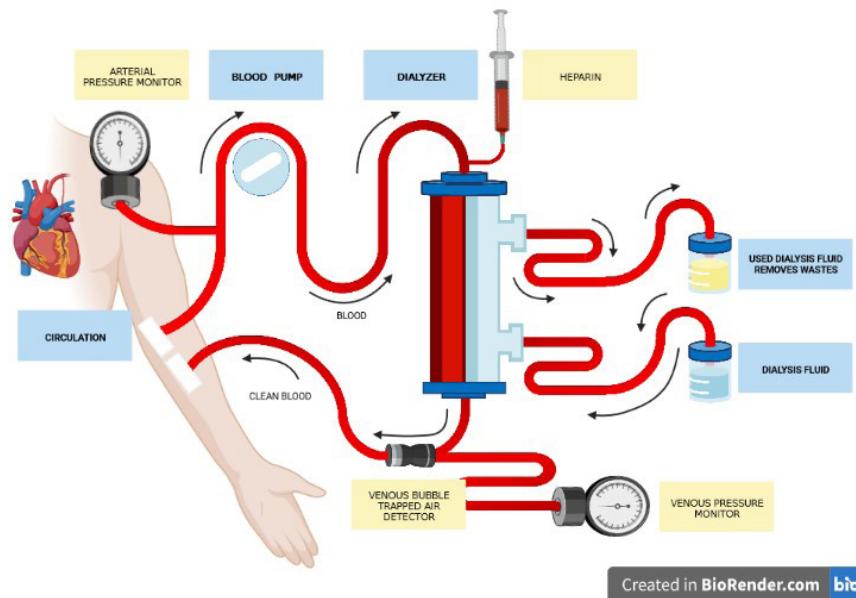


Figure 3. Basic summary of HD circuit (27,28). Created in BioRender. Jonny, J. (2025) <https://BioRender.com/s3qj2ya>.

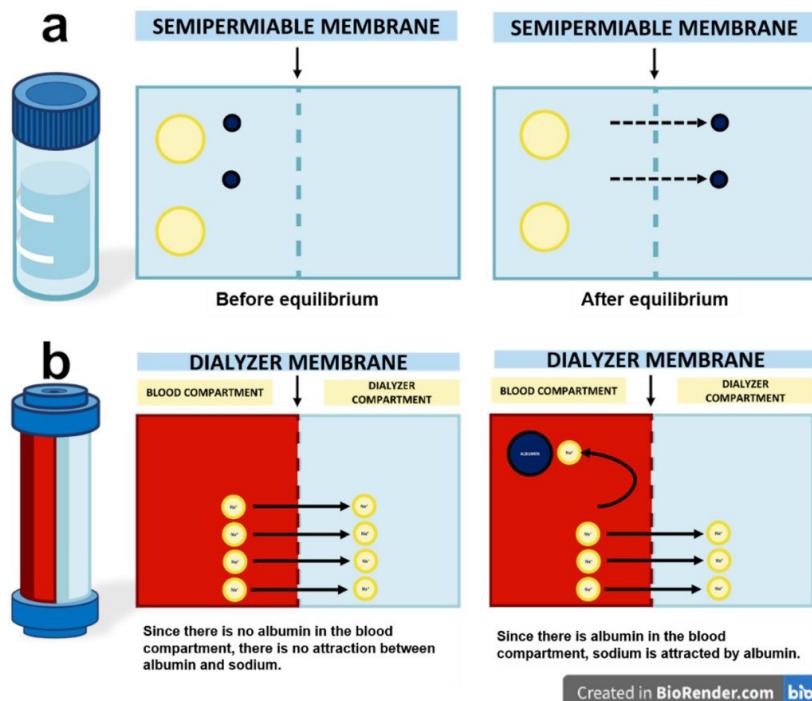
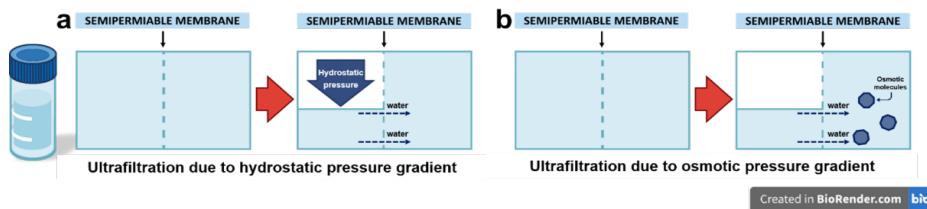


Figure 4. (a) Diffusion mechanism in HD; (b) The Gibbs-Donnan effect. Created in BioRender. Rahmat amanu, I. (2025) <https://BioRender.com/ulfoynl>.

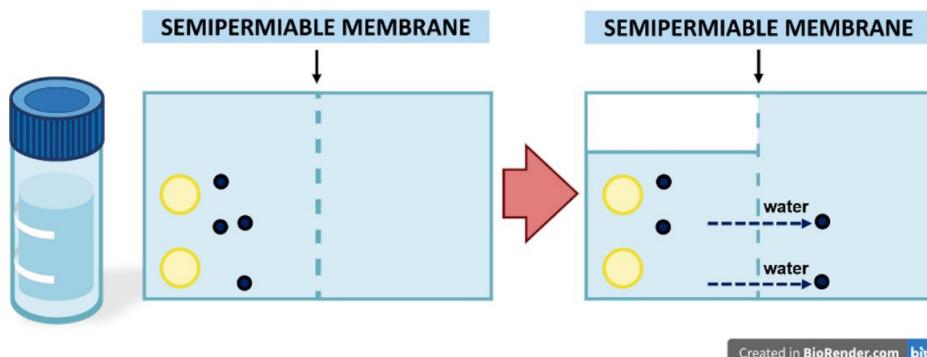
activation of the sympathetic nervous system, and redistribution of blood flow, help counteract these effects. However, when fluid removal is too rapid or excessive, or if these compensatory responses are impaired, hypotension may develop (31-33).

Intra-dialytic hypotension is the most common complication of HD, occurring in approximately 8-40% of dialysis cases and often contributing to treatment insufficiency and increased mortality (33,34). Several

mechanisms contribute to the development of IDH, including excessive UF, leading to reduced circulating blood volume, decreased venous return, and subsequent drop in cardiac output (35,36). Other contributing factors include imbalance between UF rate and plasma refill rate, inaccurate dry weight assessment, and the use of short-acting antihypertensive medications prior to dialysis (32,33). Additionally, interdialytic weight gain >1 kg/day and food intake during dialysis, particularly carbohydrate



**Figure 5.** Ultrafiltration caused by (a) hydrostatic (b) osmotic pressure gradient. The large octagon represents the osmotic molecule. Created in BioRender. Rahmat amanu, I. (2025) <https://BioRender.com/uh43ldl>.



**Figure 6.** Basic mechanism of convection. Small navy circles indicate small molecules such as urea, arrows indicate fluid displacement. Created in BioRender. Rahmat amanu, I. (2025) <https://BioRender.com/an1a8o5>.

and fat-rich meals, can exacerbate IDH by shifting blood flow to the splanchnic circulation, reducing venous return and leading to hemodynamic instability (37).

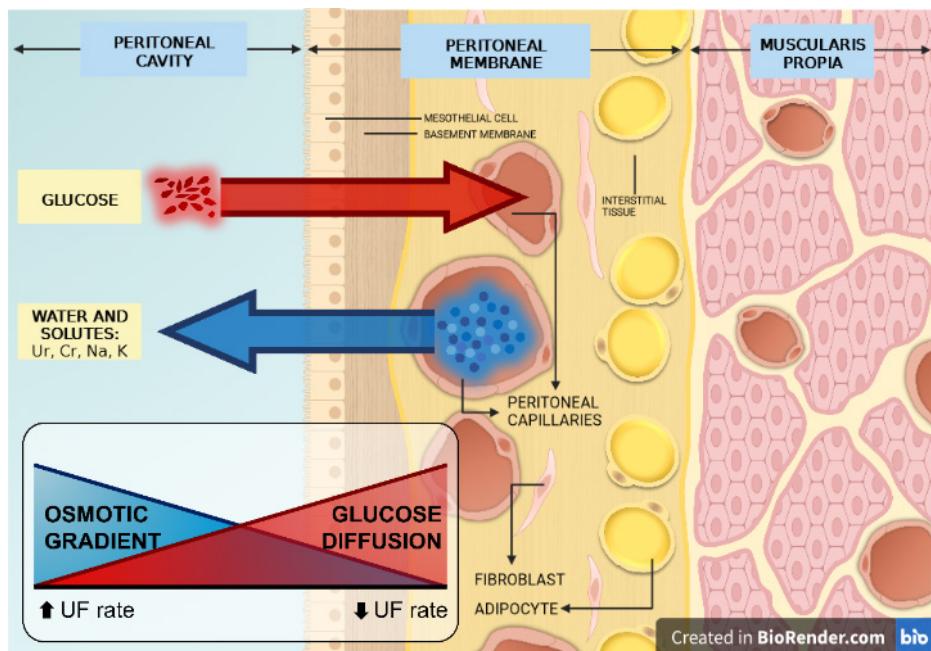
More than 50% of patients undergoing dialysis have HFpEF, characterized by impaired cardiac filling and reduced ventricular compliance, which limits the heart's ability to accommodate venous return (38). This leads to decreased preload and SV, contributing to hemodynamic instability, particularly during UF (16,31). As chamber pressures rise, even with the same venous return volume, the increased left atrial and pulmonary venous pressures promote pulmonary congestion and transudation of fluid into the interstitium, manifesting as dyspnea and edema. Similarly, around 20% of these patients have HFrEF, where impaired ventricular emptying leads to low SV and hypotension (18). In both HFpEF and HFrEF, persistently elevated left ventricular pressures are transmitted to the left atrium and pulmonary circulation, exacerbating pulmonary congestion and fluid overload-related complications during dialysis. The combination of reduced cardiac reserve and impaired compensatory mechanisms makes these patients particularly vulnerable to IDH and volume management challenges.

#### *Peritoneal dialysis*

Peritoneal dialysis utilizes the peritoneal membrane as a semipermeable dialysis interface, allowing gradual UF through osmotic-driven fluid removal. Unlike HD, which relies on hydrostatic pressure for rapid fluid extraction,

PD provides continuous UF, minimizing abrupt hemodynamic shifts. The hyperosmolar dialysate instilled into the peritoneal cavity creates an osmotic gradient, facilitating the removal of excess sodium and fluid while maintaining more stable intravascular volume Figure 7 (27,39-42). This particular process may be particularly advantageous for patients with HF, as it reduces the risk of IDH and excessive preload fluctuations, which are common in HD. Given these physiological differences, PD may offer better volume control and hemodynamic stability in HF patients compared to HD (2,4,43).

Currently, there are two types of dialysis solutions, namely dextrose-based solutions and solutions containing icodextrin (2,42). Conventional dextrose-based dialysates utilize glucose as an osmotic agent, where higher glucose concentrations generate greater osmotic pressure, leading to higher UF rates (44). However, solute transfer in these solutions is bidirectional, meaning creatinine, urea, and other waste products diffuse into the dialysate, while glucose diffuses into the bloodstream, where it is metabolized for energy (42,44). Increased peritoneal vascularization can accelerate glucose absorption, reducing the osmotic gradient and leading to poor or even negative UF. In such cases, icodextrin-based solutions, which do not diffuse across the peritoneal membrane, may be beneficial (42). Unlike dextrose-based dialysates, icodextrin is absorbed via convective fluid movement through the lymphatic system, maintaining sustained UF for 12-16 hours (42,45). Moreover, in patients with



**Figure 7.** Mechanism of peritoneal dialysis. Created in BioRender. Amanu, I. (2025) <https://BioRender.com/dfzcisj>.

diabetes or advanced age, icodextrin-based PD has been associated with better survival outcomes (46).

#### *PD versus HD in HF patients*

##### *Hospitalization rate*

Peritoneal dialysis has been associated with a significant reduction in HF-related hospitalizations, particularly in patients with chronic refractory heart failure (CRHF) (47,48). Among patients with symptomatic late-stage CHF, PD was linked to fewer hospital admissions and reduced hospitalization days during the first year of therapy (49). Additionally, in end-stage HF patients undergoing peritoneal ultrafiltration (pUF) therapy, hospitalization rates declined, with the greatest benefit observed in those with HFpEF (4). In refractory HF patients receiving pUF, hospitalization rates dropped significantly from 43 days per patient-year to 11 days per patient-year (47,50).

In a similar fashion, these patients also experienced a marked reduction in hospitalizations for cardiac-related complications compared to the pre-dialysis period (51). A significant decline in hospitalization rates was also observed in New York Heart Association (NYHA) class IV HF patients who were fluid-overloaded and resistant to maximum diuretic therapy (52). Despite this, data supporting long-term outcome remain inconclusive, as one study found no significant difference in hospitalization rates over nine years between CHF patients receiving PD or HD (13). These findings suggest that while PD may provide short-term benefits in volume management and hospitalization reduction, further studies are needed to determine its long-term impact compared to HD.

##### *Survival rate*

Among 126 patients with CRHF receiving PD, survival rates were 85% at one year and 56% at two years (47,48). Similarly, in 48 patients with refractory HF and a mean eGFR of  $20.8 \pm 10 \text{ mL/min}/1.73 \text{ m}^2$  who underwent pUF therapy, survival rates were also 85% at one year and 56% at two years (47,50). In patients undergoing continuous ambulatory peritoneal dialysis (CAPD), survival varied based on LVEF: at one, three, and five years, survival rates were 97%, 88%, and 75% for those with LVEF  $>60\%$ ; 96%, 80%, and 62% for LVEF 50–60%; and 97%, 79%, and 57% for LVEF  $<50\%$  (53). However, compared to HD, PD patients had a shorter survival time from the initiation of dialysis (13).

##### *Symptoms and quality of life*

In patients with symptomatic end-stage HF, PD was associated with a significant improvement in NYHA class within three months, which was sustained over time (49). Similarly, in end-stage HF patients undergoing pUF therapy, both HFrEF and HFpEF patients experienced notable improvements in NYHA grade (4). Among patients with refractory HF and a mean eGFR of  $20.8 \pm 10 \text{ mL/min}/1.73 \text{ m}^2$  who received pUF therapy, 85% (41 patients) showed an improvement of at least one NYHA class after one year (47,50). Additionally, in patients with HF NYHA class IV and eGFR  $>25 \text{ mL/min}$  who were fluid-overloaded and resistant to maximal diuretic therapy, PD therapy led to significant NYHA class improvement within three months (52).

### Weight loss

In HF patients, weight loss can be a poor prognostic factor, often indicating muscle wasting and malnutrition due to cardiac cachexia or protein loss through dialysate in PD patients (54-56). However, in the context of PD and pUF therapy, weight reduction is generally associated with better volume management rather than malnutrition (4,49). A study of 143 end-stage HF patients undergoing pUF found significant weight loss in both HFrEF and HFpEF groups, without evidence of wasting syndrome, as albumin levels remained stable, suggesting adequate nutritional compensation (4).

Furthermore, in patients with symptomatic end-stage HF, PD was associated with weight loss in patients receiving the therapy (49). PD-induced weight loss is typically transient, occurring within the first three months of therapy in CRHF, before stabilizing thereafter (47,48,50). In congestive right HF, weight loss after PD initiation was temporary, with patients returning to baseline within a year (51). Similarly, in NYHA class IV HF patients with eGFR >25 mL/min/1.73 m<sup>2</sup>, significant weight loss was observed at three months post-initiation, followed by weight regain between the third and sixth months, with an average increase of 3.8 kg (52). These findings suggest that while PD facilitates short-term fluid removal and volume control, long-term weight trends may vary based on individual patient characteristics and disease progression.

### Cardiac function improvement

It is essential to determine whether symptomatic improvement in HF patients receiving PD is primarily due to reduced tissue and pulmonary congestion or if it reflects actual improvement in cardiac function. In CRHF patients, PD therapy led to significant clinical improvement, with a notable increase in ejection fraction in those with LVEF <30%, as shown in several studies (47,48). However, in symptomatic end-stage HF patients, PD did not result in a significant improvement in LVEF, despite a reduction in NT-proBNP levels, suggesting a beneficial effect on volume status and cardiac workload (49).

Among end-stage HF patients undergoing pUF therapy, a significant increase in LVEF was observed only in those with reduced ejection fraction (HFrEF) (4). Similarly, in refractory HF patients, those with low baseline LVEF experienced a significant EF improvement following pUF therapy (50). In NYHA class IV HF patients with eGFR >25 mL/min/1.73 m<sup>2</sup> who were overhydrated and resistant to maximal diuretic therapy, a modest but significant increase in LVEF was noted six months after PD initiation (52). These findings suggest that

PD and pUF therapy may enhance cardiac function in select HF patients, particularly those with severe systolic dysfunction.

### Mortality rate

Among 126 patients with CRHF undergoing PD, 79% (100 patients) died within 15 years (48). In 159 patients with symptomatic late-stage CHF, the one-year mortality rate was approximately 40%, increasing to 60% at two years in those receiving PD (49). Similarly, in 48 refractory HF patients treated with pUF, 46% (22 patients) died within two years (50). Mortality rates were also high among 40 patients with right-sided CHF receiving PD, with 18 deaths within one year, 26 within two years, and 29 within three years (51). In NYHA class IV HF patients who were overhydrated and resistant to maximal diuretic therapy, 23 out of 32 died within an average of 16.65 ± 12.3 months, including 9 deaths within the first year (52). Moreover, one study evaluated 594 patients CAPD therapy, where 127 of them died during a median follow-up of 39.6 months, with 57.5% of deaths attributed to cardiovascular causes. Notably, this was most likely due to a decline in LVEF with the data showed the highest mortality occurs in patients with LVEF <50% and the lowest in those with LVEF ≥60% (53). Some studies also reported higher mortality rates in CHF patients undergoing PD compared to HD, potentially due to selection bias, where hemodynamically unstable patients are more likely to receive PD (9,13,57).

### Conclusion

Despite the theoretical hemodynamic advantages of PD over HD in patients with HF, existing studies provide conflicting evidence regarding clinical outcomes. While PD has been associated with reduced hospitalization rates, improved quality of life, and better volume management, findings on long-term survival remain inconsistent. Some studies suggest increased LVEF, particularly in patients with HFrEF, but direct comparisons between PD and HD have yielded mixed results. Two studies, which compared PD therapy with HD in patients with CHF, found no significant difference in hospitalization rates but reported lower survival time and higher cardiovascular mortality in PD patients, likely due to selection bias, as more hemodynamically unstable and high-risk CHF patients were preferentially treated with PD. Given these limitations, definitive conclusions regarding the optimal dialysis modality for HF patients remain elusive, highlighting the need for large-scale, prospective studies to determine the best approach for managing volume overload and improving long-term outcomes in this high-risk population.

## Authors' contribution

**Conceptualization:** Irvan Rahmat Amanu, Jonny Jonny  
**Data curation:** Irvan Rahmat Amanu, Adrianus Jonathan Sugiharta.

**Formal analysis:** Irvan Rahmat Amanu, Adrianus Jonathan Sugiharta.

**Investigation:** Irvan Rahmat Amanu, Adrianus Jonathan Sugiharta.

**Resources:** Irvan Rahmat Amanu, Adrianus Jonathan Sugiharta.

**Supervision:** Jonny Jonny.

**Validation:** Jonny Jonny.

**Writing—original draft:** Irvan Rahmat Amanu, Adrianus Jonathan Sugiharta.

**Writing—review & editing:** Irvan Rahmat Amanu, Jonny Jonny, Adrianus Jonathan Sugiharta.

## Conflicts of interest

The authors declare that they have no competing interests.

## Ethical issues

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

## Funding/Support

This article receives no funding interest. The author(s) received no financial support for the research, authorship, and/or publication of this article.

## References

1. Groenewegen A, Rutten FH, Mosterd A, Hoes AW. Epidemiology of heart failure. *Eur J Heart Fail.* 2020;22:1342–56. doi:10.1002/ejhf.1858.
2. Kazory A, Bargman JM. Defining the role of peritoneal dialysis in management of congestive heart failure. *Expert Rev Cardiovasc Ther.* 2019;17:533–43. doi:10.1080/14779072.2019.1637254.
3. Hunt SA, Baker DW, Chin MH, Cinquegrani MP, Feldman AM, Francis GS, et al. ACC/AHA guidelines for the evaluation and management of chronic heart failure in the adult: executive summary. *J Heart Lung Transplant.* 2002;21:189–203. doi:10.1016/S1053-2498(01)00776-8.
4. Grosskettler L, Schmack B, Brockmann C, Wanninger R, Kreusser MM, Frankenstein L, et al. Benefits of peritoneal ultrafiltration in HFrEF and HFpEF patients. *BMC Nephrol.* 2020;21:1–12. doi:10.1186/s12882-020-01777-x.
5. Lesyuk W, Kriza C, Kolominsky-Rabas P. Cost-of-illness studies in heart failure: a systematic review 2004–2016. *BMC Cardiovasc Disord.* 2018;18:1–11. doi:10.1186/s12872-018-0815-3.
6. Urbich M, Globe G, Pantiri K, Heisen M, Bennison C, Wirtz HS, et al. Medical costs associated with heart failure in the USA (2014–2020): a systematic review. *Pharmacoeconomics.* 2020;38:1219–36. doi:10.1007/s40273-020-00952-0.
7. Pernias V, González M, Miñana G, Górriz JL, Juan I, Chorro FJ, et al. Refractory congestive heart failure: when the solution is outside the heart. *ESC Heart Fail.* 2020;7:311–4. doi:10.1002/ehf2.12411.
8. François K, Ronco C, Bargman JM. Peritoneal dialysis for chronic congestive heart failure. *Blood Purif.* 2015;40:45–52. doi:10.1159/000430084.
9. Sens F, Schott-Pethelaz AM, Labeeuw M, Colin C, Villar E. Survival advantage of hemodialysis relative to peritoneal dialysis in ESRD patients with heart failure. *Kidney Int.* 2011;80:970–7. doi:10.1038/ki.2011.233.
10. Marín-García J, Goldenthal MJ, Moe GW. Cardiomyopathy and heart failure in aging. In: Marín-García J, editor. *Aging Heart: Post-Genomic View.* 2008. p. 209–38. doi:10.1007/978-0-387-74072-0\_7. ISBN:9780387740713.
11. Heidenreich PA, Bozkurt B, Aguilar D, Allen LA, Byun JJ, Colvin MM, et al. 2022 AHA/ACC/HFSA Guideline for the management of heart failure. *Circulation.* 2022;145:E895–E1032. doi:10.1161/CIR.0000000000001063.
12. Kunin M, Ganon L, Holtzman EJ, Dinour D. Hyponatremia in refractory heart failure treated with icodextrin-based PD: a case series. *Nefrologia.* 2018;38:87–91. doi:10.1016/j.nfro.2017.05.010.
13. Kunin M, Klempfner R, Beckerman P, Rott D, Dinour D. CHF treated with PD or HD: patient profile and outcomes. *Int J Clin Pract.* 2021;75:1–8. doi:10.1111/ijcp.13727.
14. McDonagh TA, Metra M, Adamo M, Gardner RS, Baumbach A, Böhm M, et al. 2021 ESC Guidelines for diagnosis and treatment of heart failure. *Eur Heart J.* 2021;42:3599–726. doi:10.1093/eurheartj/ehab368.
15. Bozkurt B, Coats AJS, Tsutsui H, Abdelhamid CM, Adamopoulos S, Albert N, et al. Universal definition and classification of heart failure. *Eur J Heart Fail.* 2021;23:352–80. doi:10.1002/ejhf.2115.
16. Miranda D, Lewis GD, Fifer MA. Heart Failure. In: Lilly L, editor. *Pathophysiology of Heart Disease.* 6th ed. Philadelphia: Wolters Kluwer; 2016. p.229. ISBN:9781451192759.
17. Klabunde RE. *Cardiovascular physiology concepts.* 3rd ed. Philadelphia: Wolters Kluwer; 2022. 265 p. ISBN:9781975150075.
18. Roehm B, Gulati G, Weiner DE. Heart failure management in dialysis patients. *Semin Dial.* 2020;33:198–208. doi:10.1111/sdi.12878.
19. Richardson T, Gardner M, Salani M. Cardiovascular disease and dialysis. *Kidney Dial.* 2025;5:5. doi:10.3390/kidneydial5010005.
20. He Z, Liang H, Huang J, Zhang D, Ma H, Lin J, et al. Dialysis modality and survival in ESRD patients with CHF. *Front Med.* 2022;9:898650. doi:10.3389/fmed.2022.898650.
21. Vadakedath S, Kandi V. Dialysis complications in chronic renal failure. *Cureus.* 2017;9:1603. doi:10.7759/cureus.1603.
22. Hara M, Tanaka S, Taniguchi M, Fujisaki K, Torisu K, Masutani K, et al. Prognostic value of pre-dialysis BP in hemodialysis. *Medicine (Baltimore).* 2018;97:e13485. doi:10.1097/MD.00000000000013485.
23. Chan CT, Blankestijn PJ, Dember LM, Gallieni M, Harris DCH, Lok CE, et al. Dialysis initiation, modality choice, access, and prescription. *Kidney Int.* 2019;96:37–47.

doi:10.1016/j.kint.2019.01.017.

24. Gilg J, Pruthi R, Fogarty D. UK renal registry 17th annual report: incidence of renal replacement therapy in 2013. *Nephron*. 2015;129:1–29. doi:10.1159/000370271.
25. Chapter 8: Transition of Care in Chronic Kidney Disease. USRDS Annual Data Report. 2017. Available from: [https://wwwUSRDS.org/media/1671/v1\\_c08\\_tcckd\\_17.pdf](https://wwwUSRDS.org/media/1671/v1_c08_tcckd_17.pdf).
26. ANZDATA Registry. End stage kidney disease in Aotearoa New Zealand. 42nd report. 2016. Available from: <http://www.anzdata.org.au>.
27. Kim YL, Kawanishi H, editors. The Essentials of Clinical Dialysis. Singapore: Springer; 2018. Available from: <http://link.springer.com/10.1007/978-981-10-1100-9>.
28. Levy J, Brown E, Lawrence A. Oxford Handbook of Dialysis. 4th ed. Oxford University Press; 2016. Available from: <https://academic.oup.com/book/30045>.
29. McGuire S, Horton EJ, Renshaw D, Jimenez A, Krishnan N, McGregor G. Hemodynamic instability during dialysis. *Biomed Res Int*. 2018;2018:8276912. doi:10.1155/2018/8276912.
30. Stefánsson BV, Brunelli SM, Cabrera C, Rosenbaum D, Anum E, Ramakrishnan K, et al. IDH and risk of CVD. *Clin J Am Soc Nephrol*. 2014;9:2124–32. doi:10.2215/CJN.02680314.
31. Song JH. Complications of hemodialysis. In: Kim YL, Kawanishi H, editors. The Essentials of Clinical Dialysis. 2018. p.107. ISBN:9789811093296.
32. Davenport A. Intradialytic hypotension: mechanisms and outcomes. *Kidney Int Rep*. 2023;8:405–18. doi:10.1016/j.ekir.2022.10.031.
33. Kanbay M, Ertuglu LA, Afsar B, Ozdogan E, Sirojol D, Covic A, et al. Review of intradialytic hypotension. *Clin Kidney J*. 2020;13:981–93. doi:10.1093/ckj/sfaa078.
34. Kuipers J, Verboom LM, Ipema KJR, Paans W, Krijnen WP, Gaillard CAJM, et al. Prevalence of intradialytic hypotension. *Am J Nephrol*. 2019;49:497–506. doi:10.1159/000500877.
35. Sars B, van der Sande FM, Kooman JP. Intradialytic hypotension: mechanisms and outcome. *Blood Purif*. 2020;49:158–67. doi:10.1159/000503776.
36. Reeves PB, McCausland FR. Mechanisms and treatment of intradialytic hypotension. *Clin J Am Soc Nephrol*. 2018;13:1297–303. doi:10.2215/CJN.12141017.
37. Borzou SR, Mahdipour F, Oshvandi K, Salavati M, Alimohammadi N. Effect of mealtime during hemodialysis on complications. *J Caring Sci*. 2016;5:277–86. doi:10.15171/jcs.2016.029.
38. Escoli R, Carvalho MJ, Cabrita A, Rodrigues A. Diastolic dysfunction in dialysis. *Ther Apher Dial*. 2019;23:108–17. doi:10.1111/1744-9987.12756.
39. Gologorsky RC, Roy S. Ultrafiltration for fluid overload in HF. *Artif Organs*. 2020;44:129–39. doi:10.1111/aor.13549.
40. Yang B, Wang M, Tong X, Ankawi G, Sun L, Yang H. Experimental models in peritoneal dialysis. *Exp Ther Med*. 2021;21:1–5. doi:10.3892/etm.2021.9671.
41. Mehrotra R, Devuyst O, Davies SJ, Johnson DW. The current state of peritoneal dialysis. *J Am Soc Nephrol*. 2016;27:3238–52. doi:10.1681/ASN.2016010112.
42. Teitelbaum I. Peritoneal dialysis. *N Engl J Med*. 2021;385:1786–95. doi:10.1056/NEJMra2100152.
43. Puttagunta H, Holt SG. Peritoneal dialysis for heart failure. *Perit Dial Int*. 2015;35:645–9. doi:10.3747/pdi.2014.00340.
44. Zeier M, Schwenger V, Deppisch R, Haug U, Weigel K, Bahner U, et al. Glucose degradation products in PD fluids. *Kidney Int*. 2003;63:298–305. doi:10.1046/j.1523-1755.2003.00705.x.
45. Chang TI, Ryu DR, Yoo TH, Kim HJ, Kang EW, Kim H, et al. Effect of icodextrin on residual renal function in PD. *Medicine (Baltimore)*. 2016;95:e2991. doi:10.1097/MD.0000000000002991.
46. Wang IK, Lu CY, Lin CL, Liang CC, Yen TH, Liu YL, et al. Risk of CVD in hemodialysis vs peritoneal dialysis. *Int J Cardiol*. 2016;218:219–24. doi:10.1016/j.ijcard.2016.05.036.
47. Goumenos DS, Papachristou E, Papasotiriou M. Renal replacement therapy in heart and kidney failure. *Pril (Makedon Akad Nauk Umet Odd Med Nauki)*. 2016;37:43–7. doi:10.1515/prilozi-2016-0015.
48. Courivaud C, Kazory A, Crépin T, Azar R, Bresson-Vautrin C, Chalopin JM, et al. PD reduces hospitalization days in refractory HF. *Perit Dial Int*. 2014;34:100–8. doi:10.3747/pdi.2012.00149.
49. Grossekettler L, Schmack B, Meyer K, Brockmann C, Wanninger R, Kreusser MM, et al. PD as a therapeutic option in HF patients. *ESC Heart Fail*. 2019;6:271–9. doi:10.1002/ehf2.12411.
50. Bertoli SV, Musetti C, Ciurlino D, Basile C, Galli E, Gambaro G, et al. Peritoneal ultrafiltration in refractory HF. *Perit Dial Int*. 2014;34:64–70. doi:10.3747/pdi.2012.00290.
51. Pavo N, Yarragudi R, Puttinger H, Arfsten H, Strunk G, Bojic A, et al. Parameters associated with therapeutic response to PD in refractory HF. *PLoS One*. 2018;13:1–15. doi:10.1371/journal.pone.0206830.
52. Papasotiriou M, Liakopoulos V, Kehagias I, Vareta G, Ntrinius T, Papachristou E, et al. Favorable effects of PD in refractory HF. *Perit Dial Int*. 2022;42:48–56. doi:10.1177/0896860820970097.
53. Wang Y, Xiong L, Xu Q, Li W, Peng X, Shen J, et al. LV systolic dysfunction and mortality in PD. *Nephrology*. 2018;23:927–32. doi:10.1111/nep.13154.
54. Kamisaka K, Kamiya K, Iwatsu K, Iritani N, Imoto S, Adachi T, et al. Weight loss in HFpEF: FLAGSHIP study. *ESC Heart Fail*. 2021;8:5293–303. doi:10.1002/ehf2.13619.
55. Krysztofiak H, Wleklik M, Migaj J, Dudek M, Uchmanowicz I, Lisiak M, et al. Cardiac cachexia in HF. *Clin Interv Aging*. 2020;15:2041–51. doi:10.2147/CIA.S273967.
56. Guedes AM. Peritoneal protein loss in PD. *Perit Dial Int*. 2019;39:201–9. doi:10.3747/pdi.2018.00138.
57. Wang IK, Lin CL, Yen TH, Lin SY, Sung FC. Survival in hemodialysis vs peritoneal dialysis in the era of icodextrin. *Eur J Intern Med*. 2018;50:69–74. doi:10.1016/j.ejim.2017.11.017.