10.5152/turkjnephrol.2022.21076

Original Article

Turkish Journal of Nephrology

Could Peritoneal Dialysis be an Option for the Treatment of Congestive Heart Failure?

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ABSTRACT

Objective: Heart failure is a progressive and fatal disease even with appropriate treatment. Hypervolemia is a major cause of mortality and hospital admissions in these patients. Peritoneal dialysis has been successfully used for volume control in congestive heart failure patients with diuretic resistance in recent years. The present study aims to assess the effects of peritoneal dialysis on refractory heart failure.

Methods: The 2-year follow-up data of 12 heart failure patients with reduced ejection fraction who had undergone peritoneal dialysis at our center between 2014 and 2019 were retrospectively analyzed in 3-month periods. The effects of peritoneal dialysis on functional status, echocardiography, physical and biochemical parameters, hospital admission, and mortality rates were assessed.

Results: Functional capacity improved significantly ($P = .005$). When the sixth month was reached, no patient remained in the New York Heart Association class 4. A significant improvement was observed in mean arterial pressure (75.3 ± 17 vs. 91.3 ± 16.6; $P = .005$). There was an improvement in congestive symptoms (dyspnea, pleural effusion, and pretibial edema; $P = .037$; $P = .0002$; $P = .005$, respectively). Although statistical significance could not be reached, ejection fraction on echocardiography was found to increase ($28.7\% \pm 12\%$ vs. $37\% \pm 12\%$; $P = .113$). Despite statistical significance was not reached, there was a trend for an increase in hematocrit, serum albumin, and sodium levels and a decrease in uric acid level. The 1-year and 2-year mortality rates were 41.7% and 58.3%, respectively.

Conclusion: In heart failure patients with reduced ejection fraction with diuretic resistance, peritoneal dialysis improves congestive symptoms, improves functional capacity, and offers a treatment option in addition to pharmacological therapy.

Keywords: Heart failure, cardiorenal syndrome, peritoneal dialysis, ultrafiltration

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Received: April 1, 2021 **Accepted:** June 22, 2021

Cite this article as: Gibyeli Genek D, Alp A, Huddam B. Could peritoneal dialysis be an option for the treatment of congestive heart failure? *Turk J Nephrol.* 2022;31(2):155-164.

INTRODUCTION

The incidence and prevalence of heart failure have strikingly increased in the last 30 years. The worldwide prevalence of heart failure is 64.3 million patients, with the known prevalence of heart failure in developed countries ranging between 1% and 2% in adults.[1] With the continuing trend of an increase in life expectancy, heart failure has become a global pandemic. Despite significant advances in medicine and device therapies introduced in the last decade, the prognosis of heart failure remains substantially poor. Some studies have reported that the 5-year mortality rate is as high as 75% among heart failure patients older than 65 years of age.[2]

Congestive symptoms are one of the leading causes of frequent admissions due to heart failure. In heart failure, reduced cardiac output and a consequent reduction in renal blood flow lead to the activation of the renin–angiotensin–aldosterone axis and the sympathetic system while they also caused non-osmotic release of arginine vasopressin. As a result of these maladaptive neurohormonal mechanisms, salt and water excretion is

reduced, and venous congestion becomes persistent. Increased venous congestion and reduced renal perfusion in heart failure cause progressive worsening of glomerular filtration rate (GFR) and renal function (cardiorenal syndrome type 2).[3-5]

The current pharmacological therapy of chronic heart failure consists of diuretics, beta-blockers, renin–angiotensin–aldosterone system blockers, natriuretic peptides, neprilysin inhibitors, and salt and water restriction. When pharmacological therapy fails to improve congestive symptoms, ultrafiltration may become necessary. Peritoneal dialysis (PD) is a home-based treatment modality that is also used for patients with resistant heart failure. Slow ultrafiltration with PD may offer a viable option for correcting hypervolemia by preserving residual renal function without reducing cardiac output and thus hemodynamic status. It also allows the adequate use of pharmacological therapy which provides limited benefit in heart failure treatment due to worsening renal function.[4-6] Studies in recent years have shown that PD successfully improves congestive symptoms and reduces mortality and hospital admission rates in patients who are unresponsive to medical therapy and who develop diuretic resistance.[5-7] In addition, PD is known to reduce the levels of inflammatory cytokines, myocardial depression factors, and natriuretic peptides.[8,9]

METHODS

We retrospectively reviewed the medical records of patients with heart failure with reduced ejection fraction (HFrEF) but without end stage kidney failure, who had been hospitalized in the nephrology and cardiology clinics at our hospital and who had undergone PD due to diuretic resistance. Patients who had received the diagnosis of HFrEF according to the ACCF/AHA 2013 Heart Failure Guideline were enrolled.[10] Since there is no single generally accepted definition of diuretic resistance, we defined the latter as the failure to provide an apparent improvement of symptoms despite using an adequate furosemide dose (>80 mg/day).[11] All patients were implanted with a Tenckhoff PD catheter by a nephrologist using the percutaneous method. After catheter implantation, PD was started on the same day in patients with ascites and 24-48 hours later in other patients. In the first week, 500 mL was used as filling volumes, and it was increased to 1000-2000 mL in the following weeks. Icodextrin was used in PD in all patients except for a patient. Eight patients applied a single daily exchange while 4 others performed multiple exchanges. We investigated the effects of PD on congestive symptoms, cardiac functions, the course of renal function, and inflammation in these patients. We also analyzed the data on why and how frequently the patients were admitted to the hospital after starting PD. We collected the 3rd, 6th, 9th, 12th, and 24th months data of the patients who visited the PD outpatient clinic for monthly follow-ups after starting PD. Since only 5 patients continued to undergo dialysis after the ninth month, there were no sufficient data for statistical analysis for periods beyond that time point, and the baseline data could only be compared with the data pertaining to the third, sixth, and ninth months.

MAIN POINTS

- Refractory edema is an important factor that causes an increase in both mortality and morbidity in heart failure.
- Peritoneal dialysis can provide effective volume control in heart failure patients with reduced ejection fraction with its near-physiological ultrafiltration effect.
- One of the symptomatic treatment options for diuretic-resistant heart failure patients is peritoneal dialysis.

Evaluation of congestive symptoms secondary to heart failure was based on the presence of dyspnea, pretibial edema (assessed during physical examination and graded between +1/+4), pleural effusion (assessed with x-ray), and ascites. We also compared urine density measurements to evaluate the patient's volume status indirectly. Cardiac functions were evaluated by the New York Heart Association (NYHA) classification, echocardiography, and mean arterial pressure. In order to assess inflammation, malnutrition, and renal function, serum urea, serum creatinine, serum sodium, serum uric acid, serum albumin, hematocrit, neutrophil/lymphocyte ratio (NLR), and thrombocyte/lymphocyte ratio were determined. The study was started after it was approved by the Ethics Committee of Muğla Sıtkı Koçman University Training and Research Hospital.

Statistical Analysis

Statistical analyses were performed using SPSS software for Windows software package version 25.0 (IBM Corp., Armonk, NY, USA) and WEB-Based R software (R Foundation for Statistical Computing, version 3.5.2, package: nparLD, Vienna, Austria; r-project.org). The non-parametric Brunner–Langer method was used for repeated measurements. First, the Brunner–Langer F1-LD-F1 model where deceased-surviving groups were taken into consideration was used to analyze the differences between repeated measurements. As the effect of mortality was not significant in those analyses, it was removed from the model, with the final analyses having been done using the LD-F1 model. As a result of the analysis, the hypothesis controls were completed for differences between repeated measurements of the variables for which time was not found to be significant. Paired temporal comparisons of the variables for which time was found significant were carried out using the same model with Bonferroni correction. The patients' survival time was predicted using the Kaplan–Meier method, and the median survival time was determined with a 95% CI. All hypothesis controls were performed at a statistical significance of .05.

RESULTS

The data of 12 patients were analyzed. The demographic and baseline clinical and laboratory characteristics of the study

Table 1. Demographic Characteristics	
Number of patients (n)	12
Female/male	4/8
Age (mean, min-max) years	64 (46-81)
Diabetes (n)	3
Duration of dialysis (mean, min-max) months	10
Number of dialysis exchanges (n)	
Single	8
>1	4
Icodextrin use (n)	11
Length of survival (median, 95% CI) days	363 (95%, 0-823)
Length of hospital stay (mean, 95% CI) days	18.2 (95%, 6.1-30.1)

Table 2. Baseline Clinical and Laboratory Characteristics	
Number of patients (n)	12
Dyspnea	12/12
Presence of pleural effusion	12/12
Pretibial edema	
1+	1
2+	3
3+	5
4+	3
Presence of ascites	8/12
NYHA class	
Class 1	None
Class 2	None
Class 3	4
Class 4	8
Mean arterial pressure (mean, mm Hg)	75.3 ± 17
Echocardiography (mean, %ejection fraction)	28.7% ± 12
Presence of aortic calcification	6/12
Urea (mean, mg/dL)	92 ± 59
Uric acid (mean, mg/dL)	7.6 ± 1.7
Creatinine (mean, mg/dL)	2.08 ± 1.1
GFR (mL/min/1.73 m^2)	42.5 ± 32
Sodium (mean, mg/dL)	131 ± 7.1
Hematocrit (mean, %)	35.2 ± 4.1
Albumin (mean, gr/dL)	3.3 ± 0.4
Neutrophil/lymphocyte ratio (mean)	6.2 ± 3.4
Thrombocyte/lymphocyte ratio (mean)	208.1 ± 93
Urine density (mean)	1014 ± 7

GFR, glomerular filtration rate; NYHA, New York Heart Association.

population are shown in Tables 1 and 2. All patients had a diagnosis of heart failure and were receiving pharmacological therapy (angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blocker, beta-blocker, diuretic (furosemide and/or spironolactone). All patients were taking furosemide. At the same time, 5 patients were taking spironolactone, and 5 patients were taking spironolactone+thiazide. The patients' basal diuretic doses ranged from 160 mg to 600 mg for furosemide, 25-50 mg for spironolactone, and 25-50 mg for thiazide. Furosemide was continued at the initial dose in all patients except 1. Due to the development of ototoxicity in the patient who received only 600 mg of furosemide, dose was reduced to 160 mg. The treatment of patients who received spironolactone, which was initiated due to heart failure, continued throughout their follow-up. In 2 patients, thiazide was discontinued due to hyponatremia. It was not possible to say that there was a significant decrease in diuretic doses. Two patients had valvular heart disease, all patients had a history of hypertension, 1 patient had dilated cardiomyopathy, 9 patients had a history of coronary artery disease, and diabetes was present in 3 patients. Peritoneal dialysis was performed in 8 patients by applying icodextrin only once a day for 8-10 hours. In other patients, 4 changes per day were performed with hypertonic solutions (2.27%-3.66% or 2.5%-4.25% solutions and adding icodextrin). The mean PD time was 10 (1-32) months.

During the time of PD, 9 patients required hospital admission. Only 3 of the 9 patients were hospitalized due to PD complication (peritonitis). Apart from this, no complication of PD was observed. The other admission indications were infection (pneumonia, urinary tract infection, and diabetic foot), hyponatremia, and cardiac complications. At the end of the 2-year follow-up period, 7 (58.3%) patients died (Figure 1). The death occurred secondary to infection in a patient while it was of cardiac origin in the other 6 patients. No patient was lost to peritonitis (Table 3).

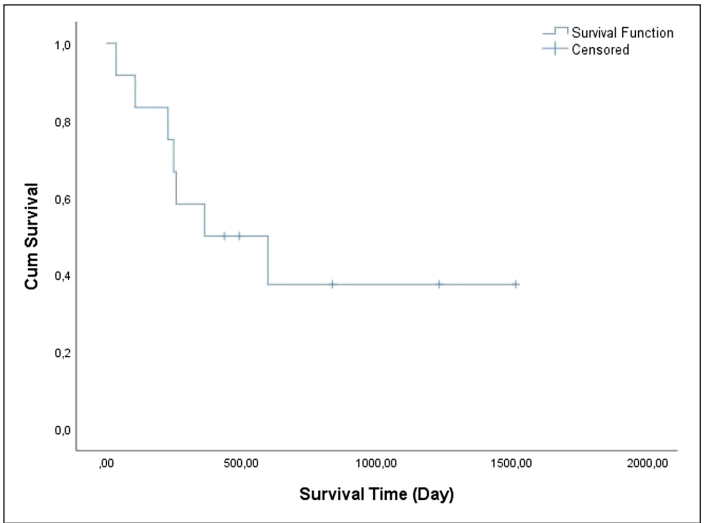

Figure 1. Overall survival among patients with heart failure that underwent peritoneal dialysis.

Table 3. Causes of Hospitalization and Death

Number of patients (n)	12
Mortality (%)	
1-year mortality	5/12 (41.7%)
2-year mortality	7/12 (58.3%)
Causes of mortality	
Infection (%)	1/7 (14.3%)
Cardiac (%)	6/7 (85.7%)
Length of survival (median, 95% CI) days	363 (95% CI: 0-823)
Length of hospital stay (mean, 95% CI) days	18.2 (95% CI: 6.1-30.1)
Length of hospital stay (mean) day/month	1.7
Causes of hospitalization	
Peritonitis (%)	3/9 (33.3%)
Other infections (%)	3/9 (33.3%)
Cardiac complications (%)	3/9 (33.3%)

There was a significant improvement, particularly in congestive symptoms, which most markedly occurred in the first month. It was noted that this observation did not vary by mortality status, with PD having similarly improved congestive symptoms in all patients irrespective of whether they died or survived. As for edema, a significant improvement occurred in dyspnea, pretibial edema, and pleural effusion and, as an indirect parameter, density showed a significant increase (Figures 2-4).

The urine and ultrafiltration volumes of the patients were based on the measurements given by them at the outpatient clinic (Table 4). That was the limitation of our retrospective study. However, we saw that the urine volumes of the patients at the end of the study decreased in 8 patients. The last urine volumes of these patients represented the last period of hospitalization due to worsening of their clinics. Afterward, it is seen that these patients died. A decrease in the amount of urine may be an indicator of acute kidney injury due to recent causes such as infection, cardiac failure, hypotension. Evaluating the weight changes, we realised a decrease in the last weight measurement in only 4 patients.

When the ninth month was reached, only 1 patient with pleural effusion remained. We also noticed that mean arterial pressure and the NYHA class, which we used for the assessment of cardiac functions, showed statistically significant change after the start of PD. Except 2, all patients showed improvement in their functional capacity. (Table 5). No patient had NYHA class 4 at the end of the study (Figures 5 and 6). Mean arterial pressure significantly increased in the first months after PD was started (75.3 ± 17 vs. 91.3 ± 16.6; $P = .005$).

No significant change was observed in the patients' renal functions. Urea/creatinine elevation, a functional response to renal hypoperfusion due to reduced cardiac output, gradually diminished over time. While urea levels decreased over time, there was an increase in the creatinine level. No significant change occurred in GFR measurements sufficient to change the number of dialysis exchanges. However, a non-significant increase in serum albumin, serum sodium, and hematocrit was apparent. There was also a trend for reduction in serum uric acid levels, although that difference did not reach statistical significance. Neutrophil/lymphocyte and thrombocyte/lymphocyte ratios, which have recently found a gradually increasing popularity as markers of subclinical inflammation among cardiac patients as well as patients undergoing PD, did not yield statistically significant results (Table 6).

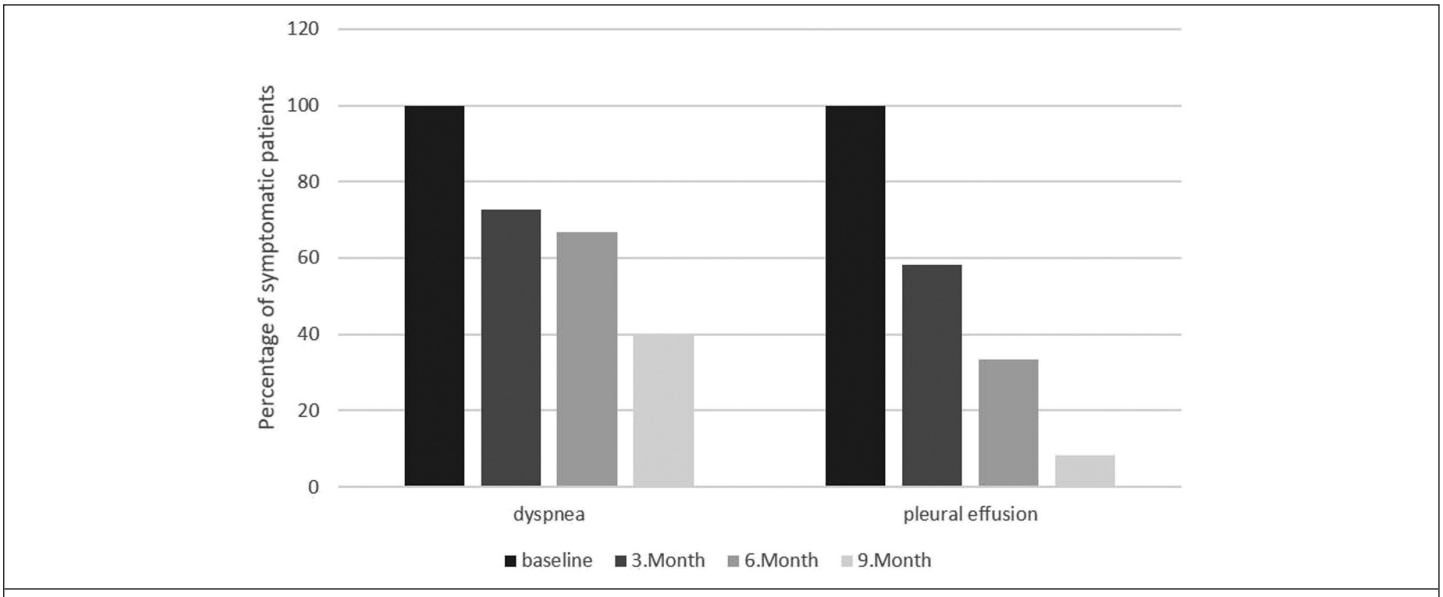

Figure 2. Temporal change of dyspnea and pleural effusion. Improvement/decrease in dyspnea over time was statistically significant ($P = .037$). Improvement/decrease in pleural effusion over time was statistically significant ($P = .0002$).

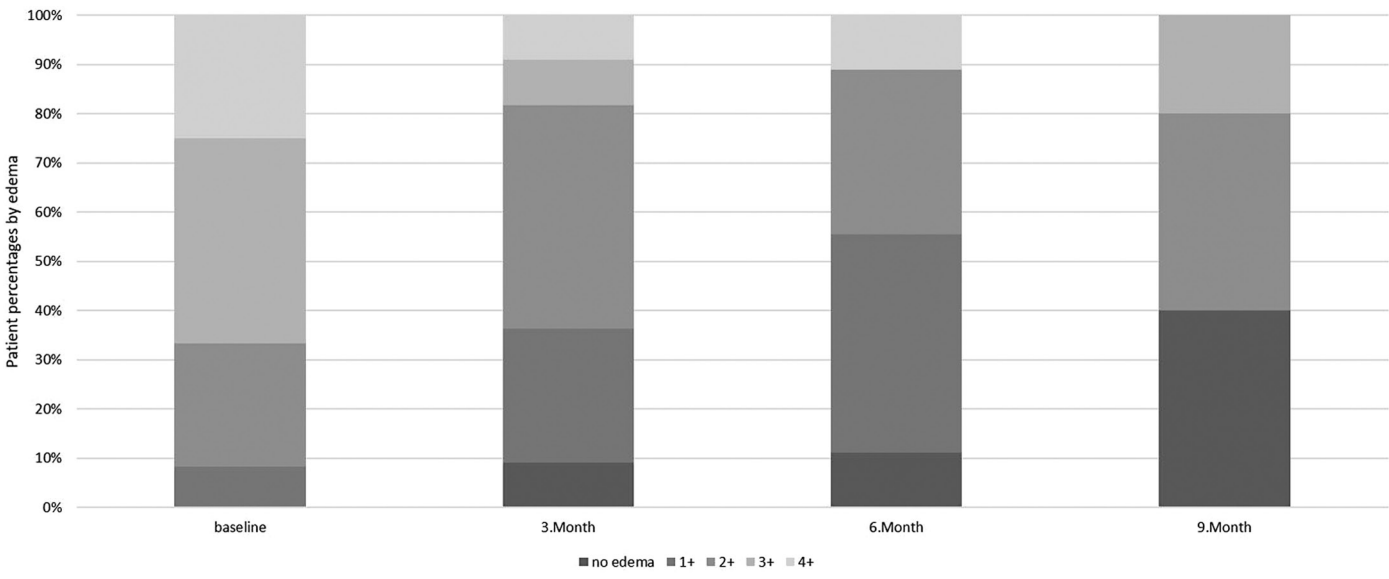

Figure 3. Temporal change of pretibial edema (P = .005).

The mortality rates by 1 year and 2 years were found to be 41.7% and 58.3%, respectively. The mean length of hospital stay was 18.2 days. No biochemical or physical data could be analyzed for their ability to predict mortality and hospitalization rates due to the low number of patients.

Of the patients who remained in the study after the ninth month, only 2 were still alive. One patient's PD treatment was terminated at the end of 6 months when euvolemia was achieved and adequate diuretic response was achieved with diuretics. The other patient continued continuous ambulatory peritoneal dialysis (CAPD) because he has reached the stage of end stage kidney disease.

DISCUSSION

In the present study, our findings include that the 12 patients who underwent PD tolerated it well; no patient wished to quit the treatment. Peritoneal dialysis was performed in these patients specifically because they had diuretic resistance and refractory congestion. We concluded that patients particularly benefited from the treatment with respect to their congestive symptoms. In parallel with this observation, we noted a marked improvement in their heart failure NYHA class. We believe that we can also indirectly tell an improvement in cardiac functions by looking at a significant increase in mean arterial pressure. As the study group had a high mortality rate, analysis of the study data was limited to the first 9 months. The ultrafiltration effect of PD on edema was more prominent in the first 3 months but it also persisted in the subsequent months. A single exchange with icodextrin was performed in 8 patients. A significant improvement in congestion could be achieved with a single exchange. The mortality rate was 41.7% by 1 year and 58.3% by 2 years. Death was due to cardiac causes in 85.7% of the patients and due to infections in the remainders. Our study detected a significant improvement in congestive symptoms of heart failure and NYHA class. These results appear to be the common conclusion of almost all prospective and retrospective studies performed so far to study the effects of PD in patients with heart failure.[12-16]

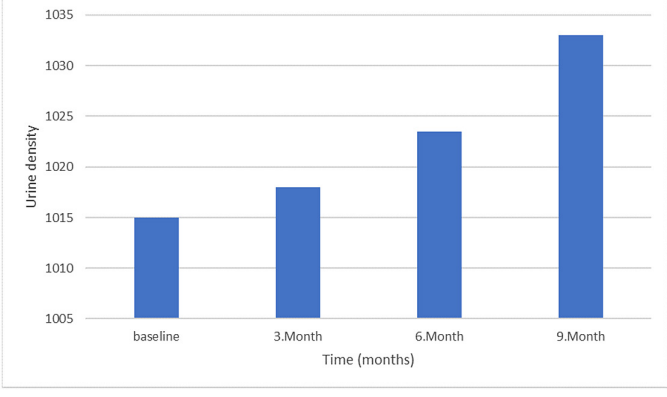

Figure 4. Temporal change of urine density (P = .009).

In heart failure patients, low blood pressure is common (10%-15%) and causes setbacks in starting pharmacological therapy in appropriate doses and titrating medications.[17] A low pressure (systolic blood pressure <90 mm Hg) indicates a poor prognosis particularly in acute heart failure.[18] However, such a relationship has not been reported for outpatients with chronic heart failure. A comprehensive review of pharmacological therapy in patients with chronic heart failure and low blood pressure was recently published.[19] In our study, our patients had a low baseline mean arterial pressure of 75.3 ± 17. Peritoneal dialysis could be easily performed in them. It was also found to exert a significant effect on mean arterial pressure (MAP) (baseline MAP

Table 4. Changes in Weight, Urine Volume, and UF Amounts of Patients

Patient No.	Time (Months)	Basal Weight (kg)	Last Weight (kg)	Basal Urinary Volume (mL)	Last Urinary Volume (mL)	Basal UF (mL)	Last UF (mL)
1	24	69	64	1250	1500	300	600
2	7	81	78	1500	2000	300	750
3	11	87	91	1500	600	1500	2000
4	7	65.9	69.5	1500	600	900	600
5	1	75	77	500	100	600	300
6	3	60.6	68.3	1500	750	680	1250
7	3	49	48	1500	800	300	500
8	8	63	78	500	100	2300	900
9	19	85	92	1000	500	1100	1700
10	8	42	42	1000	50	2000	1000
11	16	77.9	70.,6	1000	1500	500	1500
12	14	67	66.7	600	800	1500	1200

UF, ultrafiltration.

75.3 ± 17 vs. ninth month MAP 91.3 ± 16.6; $P = .005$). This feature of PD may mitigate the difficulties related to medication use in heart failure patients who suffer limitations to pharmacological therapy due to hypotension. In addition, it may spare patients from electrolyte disorders (hypokalemia, hyponatremia) and other side effects (ototoxicity) that potentially complicate progressive increase in medication doses. Whether it has favorable effects on hypotension should be investigated in prospective randomized controlled studies.

Table 5. Change in Functional Capacity According to New York Heart Association (NYHA) Classification

Patient No.	Basal	3 Months	6 Months	9 Months	12 Months	24 Months
1	3	2	2	2	2	2
2	4	3	2	a	a	a
3	4	3	3	3	3	x
4	4	3	3	x	x	x
5	4	x	x	x	x	x
6	3	2	x	x	x	x
7	4	2	a	a	a	a
8	4	3	3	x	x	x
9	3	2	2	2	2	x
10	4	4	4	x	x	x
11	3	2	2	2	b	b
12	4	3	2	2	b	b

x, exitus; a, patients discharged from PD; b, patients who had not completed their first year at the end of the study.

There is no universal definition of diuretic resistance. It is commonly defined as a failure to correct congestive symptoms despite adequate doses of diuretics. Normally, a urine output of 3-4 L is expected to occur in response to 40 mg furosemide. This response is impaired at varying levels in persons with diuretic resistance.[20] Extracorporeal ultrafiltration is recommended for patients with congestive symptoms unresponsive to medical therapy who develop diuretic resistance (class IIa, level of evidence B).[21,22] Ultrafiltration was shown to be beneficial in the UNLOAD-HF and AVOID-HF trials whereas its outcomes were worse and side effects were pronounced in the CARESS-HF trial.[23-25] In the CARESS trial, where 2 groups receiving venovenous ultrafiltration and diuretic (pharmacological) therapy were compared, the rates of bleeding from the catheter site, gastrointestinal bleeding, sepsis, pneumonia, thrombocytopenia, and heart and renal failure were higher in the venovenous ultrafiltration group.[25] Peritoneal ultrafiltration seems theoretically reasonable to avoid some side effects of extracorporeal ultrafiltration. The evidence regarding the feasibility of PD in heart failure has been accumulating since its first known use in 1949.[26] Considering also the results of other studies, it can be easily argued that it may be a good alternative to diuretic therapy especially for improving hypervolemia-related symptoms. Its advantages include providing slow ultrafiltration without impairing hemodynamics and renal function and allowing the use of spironolactone, ACE inhibitors, and angiotensin II receptor blockers, thanks to its favorable effects on electrolyte imbalance (particularly potassium). In addition, the absence of the need for anticoagulant therapy unlike venovenous ultrafiltration provides protection against bleeding and thrombocytopenia. Applicability at home and a lower exchange number particularly in patients with favorable renal function compared to patients with renal failure provide additional economic advantages.[27]

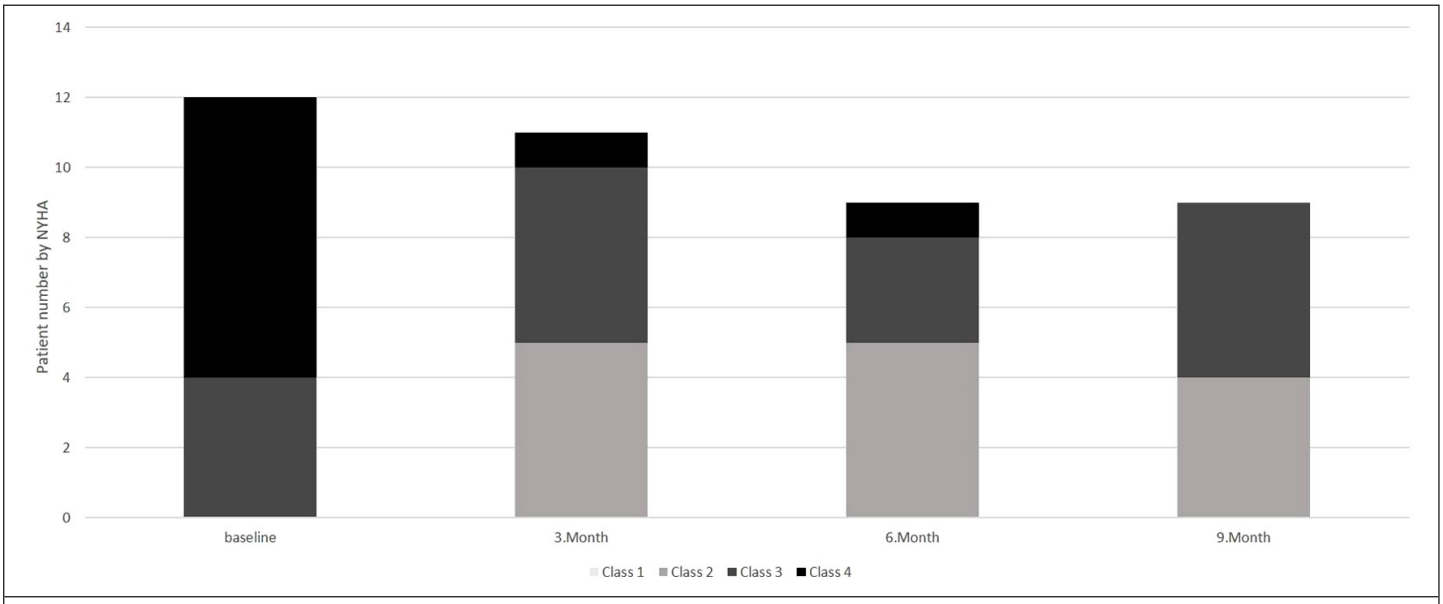

Figure 5. Temporal change of functional capacity by NYHA class ($P < .0001$). NYHA, New York Heart Association.

As with other studies, this study also showed that PD is well tolerated by patients[7,12-16] and is free of any metabolic or hemodynamic complication related to both catheter placement and dialysis procedure, except for peritonitis (3 patients). Furthermore, no peritonitis-induced death was observed.

The mortality rate by 1 year was 41.7% and by 2 years 58.3%, which was similar to rates reported in other studies.[12,16,27] In a study by Koch et al,[12] where 118 PD patients were followed, the third, sixth, and twelfth-month mortality rates were 23%, 29%, and 45%, respectively). A wide range of survival rates has been found by retrospective cohort studies in which patients with congestive heart failure undergoing PD were enrolled. The 1-year mortality rates have been reported to range between 15% and 42%.[7,13] In another retrospective analysis, the mean survival time in patients with cardiorenal syndrome type 2 treated with PD was found to be 12 ± 10 months.[28]

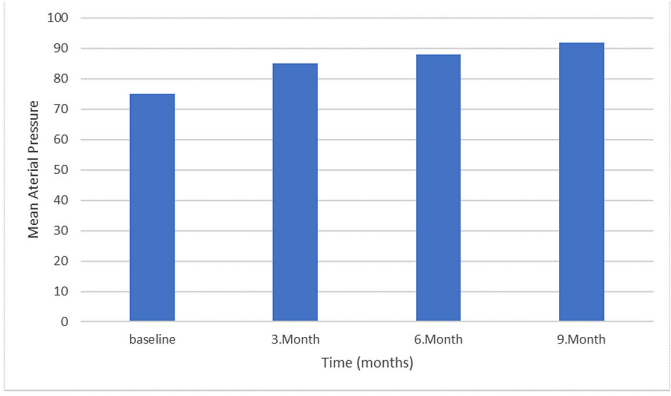

Figure 6. Temporal change of mean arterial pressure ($P = .005$).

In a prospective observational study, the median survival time was found at 14 months (1-41 months).[29] We found a median survival time of 363 days, which is approximately 12 months. In general, the mortality rate of heart failure patients has been reported to be 10.4% at 30 days, 22% at 1 year, and 42.3% at 5 years after hospitalization despite marked improvements in pharmacological and device therapies.[30] However, it is known that patients for whom a decision to administer PD is made are the ones with advanced heart failure who have higher mortality rate.[31] To date, the largest study involving heart failure patients undergoing PD is a cohort study involving 159 patients, conducted by Grossekettler et al.[16] In that study, the authors concluded that PD was successful by showing that PD achieved a 1-year mortality rate of 39.6% despite an expected mortality rate of 80% that was extrapolated using the Charlson Comorbidity Index.[16] We think similarly. Our study is not a prospective randomized controlled trial, which limits us. The results of our study, such as mitigation of congestive symptoms, improvement in MAP, and NYHA class, all make a positive contribution to the inclusion of PD among the treatment options of patients with advanced heart failure.

Another advantage stressed by the studies conducted with heart failure patients treated with PD is a marked reduction in the hospitalization rate.[7,27,29] Kunin et al[29] reported that the frequency of hospitalization dropped from 3.4 per month to 1.9 per month while Courivaud et al[7] reported a drop in the monthly number of hospitalizations from 3.3 to 0.3. During the study period, we observed that the length of hospitalization was 18.2 days (95% CI: 6.1-30.1), with a frequency of 1.7 per month. This finding is in accordance with the results of the aforementioned studies.

Several studies reported a favorable impact of PD on metabolic parameters such as hematocrit, albumin, uric acid, and sodium

Table 6. Temporal Change in Cardiac and Laboratory Findings

Laboratory Finding	Baseline	3 Months	6 Months	9 Months	*P*
Mean arterial pressure (mean, mm Hg)	75.3 ± 17	85.7 ± 15.1	88.8 ± 16.9	91.3 ± 16.6	.005
Echocardiography (mean, % ejection fraction)	28.7% ± 12	33.1 ± 12.3	33.8 ± 15.3	37 ± 12	.113
Urea (mean, mg/dL)	92 ± 59	64.8 ± 26.7	76.4 ± 38.8	73.8 ± 45	.512
Uric acid (mean, mg/dL)	7.6 ± 1.7	7.1 ± 1.7	7.3 ± 1.1	6.9 ± 0.4	.226
Creatinine (mean, mg/dL)	2.08 ± 1.1	2.1 ± 1.4	2.4 ± 1.5	3.1 ± 2.1	.162
GFR (mL/min/1.73 m^2)	42.5 ± 32	46.1 ± 29.8	36.8 ± 20.8	27 ± 19.7	.137
Sodium (mean, mg/dL)	131 ± 7.1	135 ± 6.3	133±7.7	135±4.6	.404
Hematocrit (mean, %)	35.2 ± 4.1	37.5 ± 4.6	36.5 ±7.1	38 ± 3.5	.226
Albumin (mean, gr/dL)	3.3 ± 0.4	3.4 ± 0.5	3.5 ± 0.5	3.6 ± 0.7	.399
Neutrophil/lymphocyte ratio (mean)	6.2 ± 3.4	5.3 ± 1.7	5.1 ± 1.8	7.4 ± 4.2	.560
Thrombocyte/lymphocyte ratio (mean)	208.1 ± 93	182.5 ± 59.3	158.4 ± 52.2	208 ± 92.5	.482

GFR, glomerular filtration rate.

levels.[15,16,29] This suggests that PD effective not only in relieving hypervolemia but also in removing uremic toxins and improving progressively impaired renal functions (cardiorenal syndrome). As expected, our study revealed a trend for an increase in serum albumin, hematocrit, and sodium levels and a trend for a decrease in uric acid level. We believe that PD effectively achieved the goal of ultrafiltration and that this effect occurred by correcting hypervolemic hyponatremia. Nevertheless, a significant difference could not be shown due to a limited number of patients. Considering that hyponatremia is associated with high mortality in heart failure, the efficacy of PD for correcting hyponatremia via ultrafiltration and diffusion should be further studied.

It is known that in end stage kidney disease, the serum levels of inflammatory mediators are high, including high sensitive C-reactive protein, tumor necrosis factor-a, and interleukin-6.[32] Recently, neutrophilia and relative lymphocytopenia have also been added to the list of independent predictors.[33] Beyond this notion, the NLR has gained recognition as a novel inflammatory marker for determining the severity and prognosis of cardiovascular disease.[34] Shown in a similar graphical representation, thrombocyte/lymphocyte ratio is another marker that has been recently used for the follow-up of inflammatory processes.[35] Neutrophil lymphocyte ratio and thrombocyte lymphocyte ratio has been found to be associated with cardiovascular disease in patients undergoing PD.[36-38] We aimed to determine if PD in heart failure patients without end stage kidney disease would effectively modify subclinical inflammation. Neither parameter showed a significant difference. This may have resulted from the low number of patients, non-homogenization of other comorbidities, and the study population being composed of patients with advanced heart failure.

CONCLUSION

A growing number of studies show that PD is a beneficial treatment strategy for controlling hypervolemia, limiting the number and length of hospitalizations, improving heart failure functional class, and increasing quality of life. Our study also supports the results of other studies in terms of hypervolemia control and improvement in heart failure functional capacity. It contributes to the literature in providing the option of PD in patients with prominent congestion symptoms. On the other hand, it is necessary to evaluate the effect of PD on mortality in patients with advanced heart failure, as well as its metabolic effects in prospective, randomized controlled studies with a large number of patients.

Ethics Committee Approval: Ethics committee approval was received for this study from the Ethics Committee of Muğla Sıtkı Koçman University Training and Research Hospital (Approval Date: March 29, 2021; Approval Number: 29/03/2021/75).

Informed Consent: Written informed consent was obtained from all participants.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - D.G.G.; Design - D.G.G.; Supervision - B.H.; Resources - D.G.G.; Materials - D.G.G.; Data Collection and/or Processing - D.G.G., B.H.; Analysis and/or Interpretation - B.H.; Literature Search - D.G.G.; Writing - D.G.G., A.A.; Critical Reviews - B.H., A.A.

Declaration of Interests: The authors have no conflict of interest to declare.

Funding: The authors declared that this study has received no financial support.

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