KI REPORTS
KIReports.org

Chronic Peritoneal Drainage in Refractory Right Heart Failure and Ascites

Rajashri Yarragudi[1], Noemi Pavo[2], Andja Bojic[3], Martin Hülsmann[2] and Andreas Vychytil[1]

[1]Division of Nephrology and Dialysis, Department of Medicine III, Medical University of Vienna, Vienna, Austria; [2]Division of Cardiology, Department of Medicine II, Medical University of Vienna, Vienna, Austria; and [3]Department of Medicine I, Medical University of Vienna, Vienna, Austria

Correspondence: Andreas Vychytil, Division of Nephrology and Dialysis, Department of Medicine III, Medical University of Vienna, Spitalgasse 23, 1090 Vienna, Austria. E-mail: andreas.vychytil@meduniwien.ac.at

Received 17 December 2021; revised 3 April 2022; accepted 18 April 2022; published online 28 April 2022

Kidney Int Rep (2022) **7,** 1703–1706; https://doi.org/10.1016/j.ekir.2022.04.084
KEYWORDS: ascites drainage; glomerular filtration rate; heart failure; peritoneal dialysis; peritoneal drainage; right heart failure

INTRODUCTION

Venous congestion (backward failure) plays an important role in the pathogenesis of cardiorenal syndrome. These patients develop ascites, which is associated with patient's discomfort and increased intra-abdominal pressure,[1] contributing to diuretic resistance. Paracentesis of cardiac ascites is associated with clinical and renal improvement.[2,3] Owing to direct access to the peritoneal cavity and better hemodynamic stability, ascites can be better controlled with peritoneal dialysis (PD) compared with extracorporeal ultrafiltration. Clinical studies reveal that in refractory heart failure (HF), PD leads to rapid clinical improvement and decrease of hospitalization.[4,5,6–8,S1,S2] Few authors looked especially on patients with right HF (RHF).[8] Furthermore, there is lack of data on the effect of chronic peritoneal drainage (without instillation of PD fluid) in refractory HF and ascites. This manuscript summarizes our experience with peritoneal drainage in patients with refractory RHF and ascites.

RESULTS

There were 52 of 77 RHF patients (68%) who had treatment-refractory ascites at time of PD catheter implantation (Supplementary Figure S1). Of 52 patients, 21 were excluded from the analysis because PD (instead of peritoneal drainage) was started. A total of 31 patients were included in the analysis. Table 1 displays baseline characteristics. In echocardiography, all patients had impaired right ventricular function and 20 patients (65%) additionally had a left ventricular ejection fraction <35%. Coronary artery disease was present in 18 patients (58%). Baseline estimated glomerular filtration rate (eGFR) (Modification of Diet in Renal Disease equation) was <25 ml/min per 1.73 m^2. Primary kidney disease was present in 15 patients (48%). Evidence of liver cirrhosis was 39%. There were 20 patients who needed assistance in performing peritoneal drainage, by either a family member ($n = 12$) or a nurse in the outpatient department ($n = 8$). The protocol of peritoneal drainage is described in the Supplementary Patient and Methods.

In 22 of 31 patients (71%), ascites drainage was performed <3 months. PD had to be started in these patients within 2 weeks ($n = 6$), 4 weeks ($n = 8$), 2 months ($n = 5$), or <3 months ($n = 1$) because of refractory overhydration and/or deterioration of renal function. There were 2 patients who received peritoneal drainage as a bridge to left ventricular assist device at 15 and 33 days, respectively, and were excluded.

Patient survival was 74.2% after 1 year, 45.2% at 2 years, and 38.7% at 3 years (Supplementary Figure S1).

Of the 29 patients, only 9 (31%) were treated by peritoneal drainage ≥3 months (Supplementary Figure S1). In 6 patients, PD was required subsequently between 3 to 6 months ($n = 5$) and 6 to 12 months ($n = 1$), respectively, owing to refractory overhydration and/or deterioration of renal function. There were 2 patients who remained stable with peritoneal drainage but died at 4.75 months and 13.5 months, respectively. After 11 months of peritoneal drainage, 1 patient received a left ventricular assist device. Heart transplantation was performed 2 months after.

Hospitalization days due to unplanned or cardiac reasons decreased significantly for the entire study population (in days: 43 [interquartile range (IQR): 8–87] vs. 8

Table 1. Baseline characteristics of the patients with therapy-refractory cardiac ascites and peritoneal drainage

	Baseline ($n = 31$)	Peritoneal drainage <3 mo ($n = 20$)	Peritoneal drainage ≥3 mo ($n = 9$)	*P* value
Age, yr (IQR)	65 (61–73)	66 (63–72)	65 (57–73)	0.835
Male, *n (%)*	24 (77%)	16 (80%)	6 (67%)	0.534
Weight kg, (IQR)	78 (68–91)	79 (69–90)	69 (67–84)	0.183
Heart rate, bpm (IQR)	65 (60–77)	67 (61–76)	61 (60–91)	0.765
Systolic blood pressure, mm Hg (IQR)	99 (92–115)	98 (91–113)	99 (93–115)	0.594
Quality of life, MLHFQ (IQR)	69 (58–81)	68 (57–83)	70 (54–80)	0.922
Comorbidities				
Ischemic CMP, *n* (%)	18 (58%)	13 (65%)	4 (44%)	0.567
Stroke/TIA, *n* (%)	7 (23%)	4 (20%)	3 (33%)	0.443
PAD, *n* (%)	10 (32%)	8 (40%)	2 (22%)	0.384
Diabetes mellitus, *n* (%)	13 (42%)	9 (45%)	3 (33%)	0.817
Arterial hypertension, *n* (%)	23 (74%)	15 (75%)	7 (78%)	0.712
COPD, *n* (%)	6 (19%)	4 (20%)	1 (11%)	0.508
Liver cirrhosis, *n* (%)	12 (39%)	7 (35%)	4 (44%)	0.694
Intracardiac devices				
PM, *n* (%)	6 (19%)	6 (30%)	0 (0%)	0.129
ICD, *n* (%)	13 (42%)	9 (45%)	3 (33%)	0.817
CRT, *n* (%)	13 (42%)	9 (45%)	3 (33%)	0.817
Chronic kidney disease specific				
Urinary volume, ml/24 h (IQR)	1300 (800–1900)	1275 (815–1925)	1450 (850–1800)	0.764
Proteinuria, g/24 h (IQR)	0.16 (0.00–0.41)	0.17 (0.08–0.40)	0.14 (0.00–0.42)	0.660
eGFR, ml/min per 1.73 m^2 BSA (IQR)	22.5 (17.1–34.0)	20.4 (11.7–25.7)	29.5 (21.5–43.6)	0.055
GFR, ml/min per 1.73 m^2 BSA (IQR)	12.7 (8.6–23.3)	10.4 (7.5–18.1)	23.3 (10.9–36.4)	0.026
KFRE, 8-variable, 2 yrs using eGFR (probability in %)	8.1 (1.1–29.5)	11.2 (5.3–50.5)	1.4 (0.7–9.5)	0.034
KFRE, 8-variable, 2 yrs using GFR (probability in %)	23.6 (4.4–39.3)	29.4 (10.4–64.4)	2.5 (1.5–29.1)	0.044
CREA, mg/dl (IQR)	2.68 (1.78–3.49)	3.17 (2.26–4.52)	1.87 (1.45–2.68)	0.013
Blood urea nitrogen, mg/dl (IQR)	62.6 (36.0–95.6)	91 (62–96)	31 (30–37)	0.005
Uric acid, mg/dl (IQR)	9.1 (7.2–11.6)	9.35 (7.35–11.55)	8.40 (6.40–12.0)	0.908
pH, — (IQR)	7.36 (7.31–7.40)	7.35 (7.31–7.39)	7.39 (7.36–7.41)	0.140
Bicarbonate, mmol/l (IQR)	23.6 (21.6–24.6)	22.6 (21.3–24.3)	24.3 (23.7–27.0)	0.049
Base excess, mmol/l (IQR)	−0.08 (−2.40–1.60)	−0.90 (−3.00 – 1.50)	1.50 (0.50–4.30)	0.035
Ascites, ml/d (IQR)	2000 (1200–3000)	2000 (1160–2955)	2000 (1500–3000)	0.835
Electrolytes				
Serum Na, mmol/l (IQR)	137 (135–139)	137 (135–139)	137 (136–139)	0.749
Serum K, mmol/l (IQR)	4.21 (4.02–4.40)	4.17 (4.06–4.55)	4.17 (3.80–4.41)	0.500
Serum Ca, mmol/l (IQR)	2.31 (2.18–2.41)	2.33 (2.12–2.42)	2.24 (2.22–2.34)	0.945
Serum phosphate, mmol/l (IQR)	1.29 (1.03–1.75)	1.37 (1.19–1.90)	1.14 (1.03–1.32)	0.127
Others				
CRP, mg/dl (IQR)	0.87 (0.40–2.22)	0.77 (0.31–2.38)	0.81 (0.51–1.16)	0.982
NT-proBNP, pg/ml (IQR)	17,124 (7925–27,972)	18,497 (13,160–30,490)	7925 (1675–19,510)	0.026
Hb, g/dl (IQR)	10.3 (9.1–11.5)	10.5 (9.3–11.4)	10.0 (8.6–11.4)	0.945
Leukocyte count, g/l (IQR)	5.80 (5.40–6.79)	6.12 (5.42–6.82)	5.46 (5.32–5.70)	0.127
Albumin, g/l (IQR)	37.1 (33.5–40.4)	35.5 (33.0–40.1)	38.7 (36.3–40.6)	0.444
AP, U/l (IQR)	111 (81–153)	111 (68–139)	126 (90–165)	0.172
AST, U/l (IQR)	22 (17–26)	23 (19–26)	20 (17–26)	0.532
ALT, U/l (IQR)	12 (9–16)	13 (10–17)	11 (10–15)	0.694
GGT, U/l (IQR)	99 (53–156)	98 (60–166)	122 (81–156)	0.532
BChE, kU/l (IQR)	3.58 (2.73–4.29)	3.28 (2.70–4.36)	3.85 (2.77–4.25)	1.000

ALT, alanine transaminase; AP, alkaline phosphate; AST, aspartate transaminase; BChE, butylcholinesterase; bpm, beats per minute; BSA, body surface area; Ca, serum calcium; CMP, cardiomyopathy; COPD, chronic obstructive pulmonary disease; CREA, creatinine; CRP, C-reactive protein; CRT, cardiac resynchronization therapy; eGFR, estimated glomerular filtration rate; GGT, gamma glutamyl transferase; GFR, glomerular filtration rate (calculated as average of renal creatinine and urea clearance using 24-h urine samples); Hb, hemoglobin; ICD, intracardiac defibrillator; IQR, interquartile range; K, potassium; KFRE, Kidney Failure Risk Equation (probability of kidney failure requiring dialysis); MLHFQ, Minnesota Living with Heart Failure Questionnaire; Na, sodium; NT-proBNP, N-terminal prohormone of brain natriuretic peptide; PAD, peripheral artery disease; PM, pacemaker; TIA, transitory ischemic attack. Two patients with an observation time <3 months were excluded from subgroup analysis (see the Results section). All baseline laboratory parameters, urine volume, and GFR were calculated before catheter implantation. Parameters are displayed for the total population and according to the duration of peritoneal drainage without dialysis. Parameters were compared by the Mann–Whitney *U* test and the Fisher exact test; *P* values are indicated.

[IQR: 0–48], $P = 0.001$, and 36 [IQR: 13–87] vs. 8 [IQR: 0–32], $P = 0.002$) and for the subgroup with peritoneal drainage <3 months, but not in those treated >3 months (Supplementary Figure S2). Mortality or days hospitalized (total, due to cardiac reasons or unplanned) did not differ between patients with peritoneal drainage <3 months

versus ≥3 months (P = nonsignificant for all comparisons) (Supplementary Figures S2 and S3).

Compared with patients who could be treated with peritoneal drainage ≥3 months, those who received this treatment <3 months had significantly higher baseline concentrations of N-terminal pro B-type natriuretic peptide, serum blood urea nitrogen, and serum creatinine and lower baseline GFR and serum bicarbonate (Table 1). The number of patients with consecutive RHF after severe impairment of left ventricular function (ejection fraction <35%) tended to be higher in patients with peritoneal drainage <3 months compared with those treated ≥3 months (15 of 20 vs. 3 of 9, P = 0.053). Patients with peritoneal drainage for <3 months had significantly more often primary kidney disease (13 of 20 vs. 2 of 9, P = 0.038).

In patients treated with peritoneal drainage ≥3 months (n = 9), quality of life improved significantly at 3 months compared with baseline (Minnesota Living with HF score: 70 [IQR: 54–80] at baseline vs. 55 [IQR: 41–67] at 3 months, P = 0.028). Systolic blood pressure increased significantly within 3 months of peritoneal drainage (in mm Hg: 99 [IQR: 93–115] at baseline vs. 113 [IQR: 101–126] at 3 months, P = 0.025). Although all 9 patients reached a well-tolerated edema-free state, body weight declined only temporarily after start of treatment (68.6 [IQR: 67.0–84.0] kg at baseline, 64.3 [IQR: 60.6–76.0] kg at 4 weeks, 69.9 [IQR: 61.7–82.8] kg at 3 months, P = 0.0327). There was no significant difference between baseline and 3 months in urine volume, GFR (24 hours), diastolic blood pressure, and diuretic dose.

Supplementary Results and Supplementary Table S1 provide details of peritonitis rates.

DISCUSSION

Venous congestion and ascites are associated with increase of i.p. pressure in decompensated HF.[1] There is a considerable lack of evidence to what extent peritoneal drainage (without instillation of PD fluid) contributes to clinical improvement over longer time periods[S3–S5] (Supplementary Table S2). In the study by Kunin *et al.*,[3] 18 of 69 patients with refractory chronic HF were treated by paracentesis via the PD catheter without requiring PD, revealing a median survival of 13.5 months. The authors reported improvement of New York Heart Association functional class and a decline of serum creatinine and N-terminal pro B-type natriuretic peptide. Importantly, our data reveal that only a small percentage of patients with refractory RHF could be adequately treated with peritoneal drainage beyond 3 months. However, these patients reported a significant improvement in quality of life and blood pressure.

Patients with biventricular failure (consecutive RHF after left ventricular failure in contrast to isolated RHF), with higher pro B-type natriuretic peptide levels and those with more progressive kidney disease (as demonstrated by significantly higher creatinine, serum urea concentrations, lower GFR values, and more frequent primary kidney disease) did not respond adequately to peritoneal drainage.

In agreement with our data, other authors[9,S6] stated that venous congestion (backward failure) impairs GFR preferentially when forward failure is concomitantly present. Increased N-terminal pro B-type natriuretic peptide levels in our study cohort probably reflect both severe impairment of cardiac function and decrease of renal function.

Although in the study by Kunin *et al.*[3] serum creatinine declined significantly with peritoneal drainage, GFR did not change within 3 months of treatment in our study, though only 2 of these 9 patients had primary kidney disease. Probably, our patients had larger impairment of cardiac and kidney function (reflected in markedly higher N-terminal pro B-type natriuretic peptide and lower GFR at baseline).

The marked difference between GFR and eGFR at baseline (Table 1) was also reported in previous studies,[S7–S10] suggesting creatinine-based equations are not accurate in patients with refractory HF. Reduced muscle mass in advanced HF may be one explanation for this finding.[S11]

Because this was an uncontrolled study, a conclusion that peritoneal drainage is superior to other treatments (e.g., intermittent large-volume paracentesis or earlier start of PD) in patients with RHF and ascites cannot be drawn.

In summary, peritoneal drainage is a long-term treatment option in a minority of patients with severe refractory HF and impaired renal function. Earlier start with PD (instead of peritoneal drainage) should be considered in patients with biventricular failure (consecutive RHF after left ventricular failure in contrast to isolated RHF), marked renal insufficiency, and primary kidney disease. Nevertheless, those treated with peritoneal drainage ≥3 months had improvement in blood pressure and quality of life.

DISCLOSURE

AV has received lecturer fees and travel grants from Baxter and Fresenius (manufacturers of peritoneal dialysis solutions) and consulting fees from Baxter unrelated to this trial. RY and MH have received lecturer fees from Baxter unrelated to this trial. All the other authors declared no competing interests.

SUPPLEMENTARY MATERIAL

Supplementary File (Word)

Supplementary Patient and Methods.

Supplementary Results.

Supplementary References.

Figure S1. Flow chart of the study.

Figure S2. Survival analysis for patients with therapy refractory cardiac ascites and initiation of peritoneal drainage.

Figure S3. Hospitalization of patients with therapy refractory cardiac ascites and peritoneal drainage.

Table S1. Peritonitis in patients with peritoneal drainage and peritoneal dialysis (PD).

Table S2. Overview of clinical studies focusing on peritoneal drainage in refractory heart failure patients.

REFERENCES

1. Mullens W, Abrahams Z, Francis GS, et al. Prompt reduction in intra-abdominal pressure following large-volume mechanical fluid removal improves renal insufficiency in refractory decompensated heart failure. *J Card Fail*. 2008;14:508–514. https://doi.org/10.1016/j.cardfail.2008.02.010
2. Mullens W, Abrahams Z, Shouri HN, et al. Elevated intra-abdominal pressure in acute decompensated heart failure: a potential contributor to worsening renal function? *J Am Coll Cardiol*. 2008;51:300–306. https://doi.org/10.1016/j.jacc.2007.09.043
3. Kunin M, Mini S, Abu-Amer N, et al. Regular at-home abdominal paracentesis via Tenckhoff catheter in patients with refractory congestive heart failure. *Int J Clin Pract*. 2021;75:e14924. https://doi.org/10.1111/ijcp.14924
4. Auguste BL, Agrawal A, Ibrahim AZ, et al. A single-center retrospective study on the initiation of peritoneal dialysis in patients with cardiorenal syndrome and subsequent hospitalizations. *Can J Kidney Health Dis*. 2020;7:2054358120979239. https://doi.org/10.1177/2054358120979239
5. Chionh CY, Clementi A, Poh CB, et al. The use of peritoneal dialysis in heart failure: a systematic review. *Perit Dial Int*. 2020;40:527–539. https://doi.org/10.1177/0896860819895198
6. Grossekettler L, Schmack B, Meyer K, et al. Peritoneal dialysis as therapeutic option in heart failure patients. *ESC Heart Fail*. 2019;6:271–279. https://doi.org/10.1002/ehf2.12411
7. Lu R, Mucino-Bermejo MJ, Ribeiro LC, et al. Peritoneal dialysis in patients with refractory congestive heart failure: a systematic review. *Cardiorenal Med*. 2015;5:145–156. https://doi.org/10.1159/000380915
8. Pavo N, Yarragudi R, Puttinger H, et al. Parameters associated with therapeutic response using peritoneal dialysis for therapy refractory heart failure and congestive right ventricular dysfunction. *PLoS One*. 2018;13:e0206830. https://doi.org/10.1371/journal.pone.0206830
9. Damman K, Navis G, Smilde TD, et al. Decreased cardiac output, venous congestion and the association with renal impairment in patients with cardiac dysfunction. *Eur J Heart Fail*. 2007;9:872–878. https://doi.org/10.1016/j.ejheart.2007.05.010