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Letter

Peritoneal Decompression Can Improve Acute Kidney Injury in Refractory Congestive Heart Failure

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1. Introduction

Heart failure (HF) is a public health problem. In patients with impaired ejection fraction, reduced renal function and impaired diuretic response are independent predictors of mortality [1,2], while hypervolaemia causes more than 90% of hospitalizations due to acute decompensated HF [3]. The majority of these patients have resistance to diuretic therapy [4]. Consequently, mechanical removal of fluid by ultrafiltration (UF) can be beneficial. UF can be provided by haemodialysis (HD, isolated UF) or peritoneal dialysis (PD) [5], but peritoneal UF (PUF) provides better preservation of residual renal function and haemodynamic stability.

This report presents a case of congestive HF with acute kidney injury (AKI), in which PUF not only achieved good volume control, but also significantly improved renal function after the patient was weaned off haemodialysis, highlighting the therapeutic effect of peritoneal decompression.

2. Case Description

A 60-year-old woman with a 5-year history of coronary artery disease and HF had been admitted 3–4 times annually for 3 years to cardiology wards or intensive care units with complaints of shortness of breath and abdominal distention. She had received an implanted cardioverter defibrillator 2 years previously and had undergone right coronary artery percutaneous coronary intervention 6 months prior due to an acute coronary syndrome. She reported no chronic diseases besides her cardiac history.

An admission transthoracic echocardiogram revealed a left ventricular ejection fraction of 25%, severely depressed left ventricular systolic function, advanced tricuspid regurgitation, right ventricular systolic dysfunction, and a 1.8 cm thrombus on the pacemaker lead. On 6 January, 2019, during a routine follow-up at the cardiology outpatient clinic, her consistent medication regimen was recorded as fosinopril (20 mg/day), carvedilol (12.5 mg/day), and furosemide (40 mg/day). Spironolactone/hydrochlorothiazide (25/25 mg daily) and warfarin (5 mg/day) were added to her regimen.

Three days later, on 9 January 2019, she presented to the emergency department due to a progressive decrease in urine output over the preceding 48 hours. On initial clinical examination, her blood pressure was 100/70 mmHg, heart rate was 88 beats per minute, and oxygen saturation was 94% on room air. Lung auscultation was normal without signs of overt pulmonary oedema or acute respiratory failure. Abdominal examination demonstrated tense, massive ascites with prominent abdominal distention, while pretibial oedema was absent. Given her clinical presentation of decreased effective circulating volume and compromised renal perfusion, a medication-induced or exacerbated component of AKI was considered. Her fosinopril, spironolactone/hydrochlorothiazide, and furosemide were immediately discontinued. Fosinopril was specifically suspected of precipitating an acute drop in transcapillary glomerular pressure.

Following the discontinuation of these medications, the patient was closely monitored in the nephrology clinic for worsening AKI between 18 and 28 January 2019. During this period, further escalation of diuretic doses was deliberately avoided; since the patient did not present with pulmonary oedema, it was reasoned that aggressive diuretic up-titration would severely compromise renal perfusion and further exacerbate the rise in blood urea and uric acid levels. Urgent renal replacement therapy was initially deferred between 18 and 24 January because the patient did not exhibit conventional emergency indicators: she remained free of orthopnea or pulmonary congestion, showed no signs of uremic encephalopathy or pericarditis, and her serum potassium levels remained strictly below 6.0 mmol/L.

However, on 28 January 2019, her serum potassium reached 6.7 mmol/L alongside refractory metabolic acidosis (bicarbonate 14.5 mmol/L), necessitating hospitalization in the nephrology ward and the initiation of emergency HD via a temporary central venous catheter. She subsequently received maintenance intermittent HD every other day (Table 1). While HD successfully managed her hyperkalaemia and acidosis, the patient's oliguria did not improve. Her daily urine output was less than 400 mL. She remained completely dialysis-dependent.

Faced with persistent dialysis dependence and a lack of intrinsic renal recovery under conventional HD, we decided to transition the patient to PD to achieve gradual ul-

Table 1. Serial laboratory parameters and clinical events throughout the treatment course.

Date	Creatinine (mg/dL)	Potassium (mmol/L)	Phosphorus (mg/dL)	Uric acid (mg/dL)	Bicarbonate (mmol/L)	Clinical status/Interventions
17.07.2018	0.8	4.1	—	—	—	Baseline historical value (Outpatient)
09.01.2019	1.3	3.5	—	—	—	Emergency admission; Oliguria onset
18.01.2019	6.9	3.8	—	—	24.6	Nephrology outpatient follow-up
22.01.2019	6.6	4.9	8.4	16.8	15.3	Metabolic acidosis, oral bicarbonate therapy was initiated
24.01.2019	6.15	5.6	10.0	18.1	15.3	Borderline hyperkalaemia; HD deferred
28.01.2019	5.14	6.7	—	—	14.5	Ward admission; Emergency HD started
18.02.2019	—	—	—	—	—	A PD catheter was inserted
08.03.2019	—	—	—	—	—	The last HD session was performed
10.03.2019	—	—	—	—	—	PD treatment was started
19.03.2019	1.59	4.0	4.4	—	—	Post-PD initiation follow-up
04.04.2019	1.19	4.9	4.0	—	—	Stable PD therapy
26.04.2019	1.07	3.9	3.9	—	—	Stable outpatient follow-up
21.06.2019	0.97	4.7	3.7	7.2	27.7	Stable outpatient follow-up
16.12.2019	0.81	3.5	3.1	—	—	9-month follow-up; Complete resolution

HD, haemodialysis; PD, peritoneal dialysis.

trafiltration, preserve residual renal function, manage her severe ascites, and reduce intra-abdominal pressure with the hope of enhancing diuresis. On 18 February 2019, a PD catheter was successfully inserted under local anaesthesia. Due to her massive intra-abdominal ascites, a minor pericatheter fluid leak was initially observed at the insertion edge. To reduce intra-abdominal pressure and mitigate this leakage, her ascites was drained through the peritoneal catheter daily during the early post-operative phase. Following this controlled decompression, her abdominal distention significantly decreased, her appetite improved, and her functional status, quality of life, and mobility substantially recovered, moving from an advanced symptomatic state to a markedly improved functional capacity.

On 10 March 2019, her HD catheter was removed, and she formally commenced maintenance chronic PD therapy. At the start of PD, the dialysate was exchanged three times daily in a regimen consisting of one bag of Icodextrin (Extraneal®, Baxter Healthcare Corporation) dialysate and two bags of glucose-based peritoneal solution. Oral furosemide was also restarted at a dose of 40 mg/day. By 4 April 2019, the minor pericatheter fluid leak had completely resolved, her daily urine output began to steadily rise, and her serum creatinine level demonstrated a remarkable decrease, normalizing to 1.19 mg/dL.

Her PD prescription was subsequently adjusted to two exchanges daily (one bag of icodextrin and one bag of glucose-based solution). However, this reduction resulted in immediate fluid retention, manifested by a noticeable weight gain trajectory and an increase in peripheral oedema. An escalation of oral furosemide to 120 mg/day was attempted to counteract this volume overload, but it failed to resolve the oedema or decrease her weight, suggesting persistent diuretic resistance under the reduced ultrafiltration regimen. Volume homeostasis was only successfully re-established after reverting her prescription to three daily exchanges by adding a third glucose-based solution exchange. Following this adjustment, the patient achieved stable functional capacity. At her 9-month follow-up, she remained symptom-free, maintaining a stable weight trajectory, a daily urine output of approximately 1 litre/day, and a peritoneal ultrafiltration volume of 800 mL/day. Notably, throughout this follow-up period, her serum creatinine remained stable within the normal range (0.81 mg/dL), and she required zero readmissions to cardiology wards or intensive care units for decompensated HF.

3. Discussion

Treatment options for end-stage heart failure (HF) complicated by refractory cardiorenal syndrome (CRS) are severely limited. Patients with advanced left ventricular dysfunction are highly vulnerable to acute drops in renal perfusion pressure, which can accelerate the transition from acute kidney injury (AKI) to irreversible end-stage kidney disease. In the presence of volume overload, mechanical fluid removal via ultrafiltration is an essential therapeutic approach to achieve tissue decongestion and relieve intractable symptoms. While haemodialysis (HD) offers rapid fluid and solute clearance, peritoneal dialysis (PD) provides key physiological benefits for long-term management of chronic cardiovascular frailty [6]. However, the role of peritoneal decompression in reversing HD-dependent AKI in such cases has been rarely reported.

The remarkable recovery of renal function observed in this case should be interpreted as a multifactorial process rather than attributed to a single intervention. The initial stabilization of life-threatening hyperkalaemia and metabolic acidosis was successfully achieved through tem-

porary intermittent HD. However, intermittent HD frequently causes rapid intravascular volume shifts, which carry an inherent risk of intradialytic hypotension. In patients with severe cardiomyopathy, these hypotensive episodes can induce recurrent ischaemic insults to the renal parenchyma, thereby compromising renal perfusion and delaying or preventing nephron recovery [7]. Conversely, PD facilitates a slow, continuous ultrafiltration process that avoids abrupt intravascular depletion, maintains hemodynamic stability, and preserves residual renal function [8,9].

A critical therapeutic mechanism in this patient was the effective management of her massive ascites. Tense ascites induces intra-abdominal hypertension, which directly elevates renal venous pressure, compresses the renal parenchyma, and reduces the trans-renal perfusion gradient. This mechanical congestion is a major underrecognized driver of worsening glomerular filtration in CRS. Avoiding excessive diuretic up-titration in the absence of pulmonary congestion prevented further volume depletion and avoided accelerating prerenal azotemia. Subsequently, the continuous drainage of ascitic fluid via the peritoneal catheter successfully decompressed the intra-abdominal compartment. This lowered renal afterload, optimized renal perfusion, and facilitated rapid restoration of an adequate glomerular filtration rate. Papasotiriou et al. [10] demonstrated that peritoneal ultrafiltration significantly reduced hospitalizations in New York Heart Association (NYHA) Class IV HF patients who had an estimated glomerular filtration rate (eGFR) >25 mL/min.

Nevertheless, when celebrating such favourable outcomes, clinicians must carefully consider the broader context of advanced HF management, including the “ceiling of care” and long-term prognosis. In cases of refractory CRS where renal recovery fails to occur, permanent dialytic therapy represents a major life-altering commitment. For patients with a left ventricular ejection fraction of 25% and recurrent decompensations, transplant candidacy for isolated renal transplantation is typically precluded by the prohibitive cardiac risk. Conversely, eligibility for advanced HF options—such as a ventricular assist device or orthotopic heart transplantation—is heavily dependent on the reversibility of secondary organ failure. Had this patient failed to achieve renal recovery, chronic dialysis would have served primarily as a palliative volume-management tool rather than a bridge to transplantation. Early multidisciplinary dialogue regarding care goals and advanced directives remains necessary.

The therapeutic approach in this case possesses distinct advantages and limitations that merit consideration. The primary advantage of PUF is its capacity to successfully break the cycle of recurrent volume overload in an outpatient setting and thus significantly improve the patient’s quality of life and functional capacity (advancing from NYHA Class IV to a stable Class II status). Furthermore, it entirely eliminated the need for repeated hospitalizations in cardiology or intensive care units. However, important limitations include the technical challenges of managing early pericatheter fluid leaks in the presence of high intra-abdominal pressures, the strict requirement for patient or caregiver compliance with daily exchanges, and the long-term risks of peritonitis or encapsulating peritoneal sclerosis. Additionally, while once-daily icodextrin is often sufficient for volume control in stable chronic settings, our patient required a more intensive regimen of three exchanges daily to maintain volume homeostasis. PUF prescriptions must be highly individualized based on real-time weight trajectories and fluid balance.

4. Conclusion

In conclusion, close cooperation between cardiology and nephrology specialists can optimize clinical outcomes in this fragile patient population. Peritoneal ultrafiltration represents a powerful complementary treatment modality for refractory HF. It can offer successful volume control, excellent ascites management, and a hemodynamically gentle profile that creates an ideal physiological environment for renal recovery.

Key Points

• Peritoneal ultrafiltration (PUF) can be an effective salvage therapy for patients with refractory congestive heart failure and associated acute kidney injury who do not show clinical improvement with conventional haemodialysis.

• In cases of cardiorenal syndrome complicated by massive ascites, the reduction of intra-abdominal pressure through PUF may significantly enhance renal perfusion and facilitate the recovery of glomerular filtration rates.

• Unlike haemodialysis, which carries a higher risk of intradialytic hypotension, PUF offers a gradual and continuous fluid removal process that better preserves residual renal function in fragile heart failure patients.

• The successful management of chronic volume overload and refractory oedema in advanced heart failure necessitates close multidisciplinary cooperation between cardiology and nephrology specialists.

Availability of Data and Materials

All data included in this study are available from the corresponding author upon reasonable request.

Author Contributions

EG and EY designed the case report. EG and EY were responsible for the clinical management of the patient's nephrological status, including the peritoneal dialysis process. EG and EY performed the acquisition and interpretation of clinical and laboratory data. EG drafted the manuscript. Both authors contributed to critical revision of the manuscript for important intellectual content. Both authors read and approved the final manuscript. Both authors

have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This case report was conducted in accordance with the ethical principles of the Declaration of Helsinki. Ethics committee approval was waived as this is a single case report with no experimental intervention. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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Conflicts of Interest

The authors declare no conflicts of interest.

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