



Management of Dyspnea in Advanced Chronic Kidney Disease: A Prospective Case Series on Fluid Monitoring and Targeted Chest Physiotherapy

Dr. N. Murugesan¹, Dr. Venkatram Murugesan², Dr. Baby Carolin K³, Dr. Sumathy Masanam Kasi^{4*}

¹Chief Nephrologist, East coast hospitals, Puducherry.

²Chief Nephrologist, East coast hospitals, Puducherry.

³Assistant Professor, College of Allied Health Sciences, East Coast Institute of Medical Sciences, Puducherry.

⁴Research Coordinator, East coast hospitals, Puducherry.

*Corresponding Author

Dr. Sumathy Masanam Kasi

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Abstract: - Background: Dyspnea is a prevalent, debilitating symptom in advanced Chronic Kidney Disease (CKD), often driven by complex hydro-mechanical and uremic pathways. While pharmacological volume management is standard, fluid removal alone frequently fails to rapidly resolve respiratory distress, highlighting an unmet need for multimodal clinical interventions.

Objective: This prospective observational pilot case series evaluated the clinical utility and mechanical efficacy of pairing rigorous fluid kinetics tracking (via serial daily weights) with structured chest physiotherapy (CPT) to relieve dyspnea and optimize oxygenation in hospitalized advanced CKD patients.

Methods: Ten consecutive patients with advanced CKD (Stage 3, $n = 4$; Stage 4, $n = 6$) presenting with severe dyspnea (Modified Medical Research Council [mMRC] Grade ≥ 3) were observed over a 5-day standardized protocol. Patients underwent a comprehensive diagnostic battery (radiology, echocardiography, and sputum analysis) and were managed using a dual-track protocol: Track A involved calibrated daily morning weight checks to monitor fluid shifts, and Track B comprised twice-daily CPT sessions (high-Fowler's positioning, diaphragmatic/pursed-lip re-education, and incentive spirometry).

Results: The protocol successfully tracked a mean weight loss of $3.84 \pm 0.89 \text{ kg}$ by Day 5, reflecting systemic interstitial fluid clearance. Concurrently, the cohort demonstrated significant respiratory stabilization: the mean modified Medical Research Council (mMRC) dyspnea score decreased from 3.4 ± 0.52 to 1.0 ± 0.67 , mean respiratory rate dropped from 26.8 ± 3.1 to 17.4 ± 1.5 breaths per minute, and mean room-air SpO_2 improved from $89.2\% \pm 2.4\%$ to $96.5\% \pm 1.6\%$. Notably, 80% ($n = 8$) of patients reported immediate subjective relief within 30 minutes of their initial positioning and breathing session, preceding maximum physical fluid loss.

Conclusion: Monitoring serial weight fluctuations provides a reliable, real-time assessment of fluid velocity in advanced CKD. When paired with targeted chest physiotherapy, this non-invasive, interdisciplinary approach offers safe, immediate, and sustained relief from dyspnea by unloading diaphragmatic restriction and improving alveolar recruitment. These pilot findings generate a strong clinical rationale for larger randomized controlled trials.

Keywords: Chronic Kidney Disease; Dyspnea; Chest Physiotherapy; Diaphragmatic Realignment, mMRC modified Medical Research Council.



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Introduction

Chronic Kidney Disease (CKD) has emerged as a critical global health concern, carrying a profound burden of cardiovascular and pulmonary morbidity [1]. Within this clinical landscape, the relationship between the kidneys and the lungs is vital for maintaining systemic acid-base balance, fluid homeostasis, and arterial blood pressure control. Consequently, when renal function declines, a variety of pathogenic pathways are triggered, allowing kidney disease to exert a direct, negative impact on the pulmonary parenchyma and thoracic mechanics. This complex bidirectional interference significantly worsens the overall prognosis for individuals with chronic renal disease [2].

Dyspnea is one of the most common and distressing symptoms associated with advanced CKD, exerting a profound negative influence on the patient's functional quality of life [3]. Clinically, the appearance or worsening of breathlessness is a warning sign often associated with an adverse prognosis and increased mortality [3,4]. Despite its clinical importance, the precise multi-systemic pathophysiology of dyspnea in this population remains poorly understood [4,5]. While conventional therapeutic strategies like haemodialysis are utilized to aggressively address volume overload, clinical observations show that fluid removal alone often fails to significantly improve or fully resolve breathlessness [5-7]. This clinical gap strongly suggests that multiple, co-existing alternative aetiologies ranging from uremic toxins and profound metabolic shifts to respiratory muscle fatigue are simultaneously at play [7,8].

Patients with CKD exhibit a significantly higher prevalence of lung dysfunction regardless of the disease's stage. This includes a distinct predisposition to sleep apnea syndrome, pulmonary hypertension, and chronic obstructive pulmonary disease (COPD), with the risk of developing severe pulmonary consequences rising exponentially as kidney disease progresses [5-7]. To optimize care, clinicians must move past looking at fluid retention in isolation and instead explore how various underlying respiratory disorders present within the CKD population. The aim of this article is to discuss the main pathophysiologic mechanisms underlying dyspnea in CKD patients, tracking how structured, non-invasive mechanical interventions can support traditional metabolic management.

Objectives

Primary Objective: To determine the prevalence, clinical presentation, and underlying aetiologies of various respiratory diseases in patients presenting with advanced CKD and symptomatic dyspnea.

Secondary Objective: To evaluate the clinical utility of pairing rigorous daily fluid kinetics tracking (via serial weight measurements) with targeted chest physiotherapy to

relieve dyspnea, improve oxygenation, and restore diaphragmatic function.

Methodology

Study Design and Setting

This was a prospective observational cohort study conducted over a designated 6-month period within the inpatient wards and intensive care units of a tertiary care teaching hospital. A cohort of 10 consecutive patients with advanced CKD (Stages 3 and 4) who presented with a primary complaint of breathing difficulty (Modified Medical Research Council [mMRC] Grade ≥ 3) were observed longitudinally for a standardized duration of 5 admission days from the time of initial clinical presentation through to stabilization.

Clinical Evaluation and Data Collection

Upon admission, a detailed clinical history was obtained from each patient, with specific reference to the onset and progression of CKD symptoms, the duration since the initial renal diagnosis, and the history or duration of maintenance haemodialysis. Special emphasis was placed on identifying respiratory symptoms, co-morbid conditions (such as diabetes, hypertension, or pre-existing airway disease), and functional limitations. A comprehensive physical examination was performed on each subject, focusing on the systemic signs of chronic uremia, fluid retention status, and specific clinical markers of respiratory disease (such as respiratory rate, accessory muscle use, and chest auscultatory findings).

Diagnostic Investigations Protocols

To comprehensively map the underlying aetiologies of dyspnea, each subject underwent a standardized battery of diagnostic investigations tailored to evaluate both renal function and cardiopulmonary structural integrity:

Biochemical & Functional Profiling: Routine renal function tests (serum creatinine, blood urea nitrogen, serum electrolytes) and arterial blood gas (ABG) analyses to assess acid-base status.

Radiological Imaging: Standard digital chest X-rays were performed on all patients upon admission and stabilization. High-resolution Computed Tomography (CT chest) and ultrasonography of the thorax were conducted as clinically indicated to evaluate pleural effusions, interstitial thickening, or pulmonary congestion.

Specialized Pulmonary Testing: Sputum examinations for pathogens, pleural fluid analysis (where applicable), and pulmonary function tests (PFTs) were conducted to identify obstructive or restrictive airway defects.

Cardiovascular Evaluation: Transthoracic echocardiography was performed on all 10 subjects to evaluate left ventricular systolic and diastolic function,

estimate pulmonary artery systolic pressure, and screen for uremic cardiomyopathy.

Sleep Quality: Overnight polysomnography (sleep studies) was conducted in selected patients demonstrating clinical signs of sleep-disordered breathing or nocturnal hypoxia.

Intervention Protocol

During the observation period, patients were managed using a protocol designed to target both the volumetric and mechanical components of their breathlessness:

1. **Fluid Monitoring:** To ensure maximum accuracy over traditional fluid charts, serial daily weight was used as the primary objective marker for fluid shifts. Weights were measured every morning at the exact same time using a calibrated digital scale, following voiding and prior to the consumption of breakfast. The daily change in weight was tracked to quantify the velocity of tissue fluid gain or loss.

2. **Targeted Chest Physiotherapy:** Patients received supervised chest physiotherapy sessions twice daily, lasting 20 minutes per session. The intervention followed a strict sequence:

- **Positioning:** Placing the patient in high-Fowler's positioning (60° - 90°) or an orthopneic posture to lower the diaphragm and optimize baseline lung volumes.
- **Breathing Re-education:** Implementing coached pursed-lip and diaphragmatic breathing to minimize the use of accessory respiratory muscles and lower the work of breathing.
- **Alveolar Recruitment:** Utilizing active-assisted thoracic expansion exercises combined with incentive spirometry to reverse dependent atelectasis caused by fluid pooling in the lung bases.

Statistical Analysis

The collected data were compiled and analyzed using IBM SPSS version 22. Continuous variables (such as age, laboratory values, weight measurements, SpO_2 , and respiratory rates) were expressed in terms of mean $\pm$

standard deviation (Mean $\pm$ S.D.). Categorical measures and ordinal scales (such as gender, CKD stage, and mMRC dyspnea grades) were expressed as absolute frequencies and percentages ($n, \%$). Changes in physiological parameters between admission (Day 1) and stabilization (Day 5) were analyzed sequentially to establish clinical trajectories.

The study protocol was reviewed and approved by the Institutional Ethics Committee (IEC) (Ref:EC/NEW/INST/2024/4589/EIMS/2026/ 37). Following the written informed consent, 10 consecutive hospitalized patients with a confirmed diagnosis of chronic kidney disease (CKD) were prospectively enrolled to receive the physiotherapy intervention. To safeguard participant privacy, absolute data anonymity was rigorously maintained by de-identifying all personal identifiers prior to analysis

Results

Baseline Clinical Characteristics

A total of 10 hospitalized patients with advanced Chronic Kidney Disease (CKD Stage 3, $n = 4$, 40%; Stage 4, $n = 6$, 60%) presenting with severe dyspnea were observed over a 5-day protocol period. The mean age of the cohort was 58.4 ± 7.2 years. At admission, all 10 patients (100%) exhibited significant fluid retention, marked respiratory distress (mean respiratory rate: 26.8 ± 3.1 breaths per minute), and compromised baseline oxygenation with a mean room-air spo_2 of $89.2\% \pm 2.4\%$

Fluid Kinetics and Weight Fluctuations

The protocol successfully achieved a targeted negative fluid balance across the study participants through adjusted medical management, which was precisely monitored via daily morning weights. The mean weight loss from Day 1 to Day 5 was 3.85 ± 0.89 Kg serving as an objective surrogate marker for the mobilization and clearance of interstitial fluid excess.

Longitudinal Impact on Dyspnea and Oxygenation

Concomitant with fluid loss, the twice-daily structured chest physiotherapy (CPT) protocol led to rapid and significant improvements in respiratory mechanics.

Table 1: Pre- and Post-Intervention Changes in Fluid Volumetric Status, Dyspnea Severity, and Oxygenation Parameters Among Hospitalized CKD Patients (N = 10).

Patient ID	CKD Stage	Day 1 Weight (kg)	Day 5 Weight (kg)	Net Fluid Loss (kg)	Baseline mMRC Grade	Post-Intervention mMRC (Day 5)	Baseline SpO ₂ (%)	Day 5 SpO ₂ (%)
CKD-01	Stage 4	74.2	70.1	-4.1	Grade 3	Grade 1	89%	96%
CKD-02	Stage 3	68.5	65.2	-3.3	Grade 4	Grade 1	87%	95%
CKD-03	Stage 4	82.1	77.4	-4.7	Grade 3	Grade 0	91%	98%
CKD-04	Stage 4	61.0	58.2	-2.8	Grade 4	Grade 2	86%	94%
CKD-05	Stage 3	79.4	74.5	-4.9	Grade 3	Grade 1	90%	97%
CKD-06	Stage 4	85.6	80.2	-5.4	Grade 4	Grade 2	88%	95%
CKD-07	Stage 3	71.3	68.0	-3.3	Grade 3	Grade 0	92%	99%
CKD-08	Stage 4	66.8	63.9	-2.9	Grade 3	Grade 1	89%	96%
CKD-09	Stage 3	73.9	70.4	-3.5	Grade 4	Grade 1	88%	97%
CKD-10	Stage 4	91.2	87.7	-3.5	Grade 3	Grade 1	92%	98%

By Day 5, the mean mMRC dyspnea score decreased significantly from 3.4 ± 0.52 to 1.0 ± 0.67 . Mean room-air oxygen saturation increased to $96.5\% \pm 1.6\%$, and the mean respiratory rate stabilized at 17.4 ± 1.5 breaths per minute. Notably, subjective relief from breathing distress was reported by 80% (n=8) of patients within 30 minutes following the Phase 1 positioning and diaphragmatic breathing sessions, well before maximum physical weight loss was achieved.

Discussion

This prospective observational study highlights a critical physiological principle: managing dyspnea in advanced Chronic Kidney Disease (CKD) requires addressing both fluid accumulation and respiratory muscle mechanics simultaneously. The primary finding demonstrates that combining rigorous fluid tracking with targeted chest physiotherapy brings rapid, non-invasive relief to patients facing severe uremic or volume-induced breathing difficulties. Instead of managing these pathways in isolation, our approach treats volume excess and mechanical restriction as a single, interconnected clinical crisis.

Fluid Kinetics and the Accuracy of Daily Weight

While intake-output chart fluid values are often miscalculated in ward settings due to unmeasured insensible losses, unrecorded oral intake, or rushed charting, serial daily weight tracking acts as an unvarnished, highly reliable marker of true tissue fluid retention [3-5]. Our data reveals a direct, temporal tie between physical weight reduction and the resolution of the patient's air hunger. As tissue fluid weight dropped by a mean of 3.84 ± 0.89 Kg hydrostatic

pressure inside the pulmonary capillaries fell. This shifted trapped fluid out of the alveoli and back into vascular circulation, clearing the way for better alveolar-capillary gas exchange and systematically lowering the baseline work of breathing.

The significant change in this case observation was, dyspnea scores improved significantly before patients reached their final target weight loss. This early clinical change highlights effectiveness of the targeted chest physiotherapy works on a mechanical level, acting as an immediate rescue strategy while pharmacological diuresis is still underway.

Diaphragmatic Realignment: In advanced CKD, abdominal fluid retention (ascites) and systemic fluid overload push the diaphragm upward into a flattened, mechanically weak position. Placing patients in a steep high-Fowler's positioning 60 -90 degree unloads this cephalad (upward) abdominal pressure, allowing the diaphragm to return to its optimal resting length and contract efficiently [2-,7,8].

Work of Breathing Reduction: Pursed-lip and diaphragmatic breathing exercises help control respiratory panic, lower respiratory rates, and reduce the patient's reliance on accessory neck and intercostal muscles, which burn extra oxygen and accelerate physical exhaustion [5-8].

Alveolar Recruitment: Dependent atelectasis—the collapse of small air sacs due to fluid pooling at the base of the lungs—was reversed using thoracic expansion exercises and incentive spirometry. This optimization of lung volumes explains the steady, documented increase in room-air SpO_2 observed by Day 5 [7-10].

Educational and Research Implications

For medical, nursing and physiotherapist educators, these outcomes offer an important teaching paradigm. Traditional clinical training often treats fluid overload as a problem solved solely by pharmacology via loop diuretics. This study changes that educational narrative, proving that a multidisciplinary approach—combining metabolic fluid velocity monitoring with active physical rehabilitation—speeds up clinical recovery and returns functional capacity to the patient much faster, highlighting the power of collaborative, interdisciplinary ICU care.

Limitations

The primary limitations of this study include its small pilot sample size ($n = 10$) and the lack of a formal control group. Additionally, bioimpedance analysis was not used alongside daily weights to measure fluid volumes within specific body compartments. Future larger trials should look at how this combined protocol affects long-term hospital readmission rates.

Conclusion

Routinely measuring daily weight fluctuations provides an accurate view of fluid shifts in advanced CKD patients. When paired with structured chest physiotherapy, this strategy safely and effectively relieves dyspnea, improves oxygenation, and reduces respiratory fatigue, offering an essential tool for holistic patient care.

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