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ORIGINAL ARTICLE

Multidisciplinary Evaluation of Urinary Retention and Neuro-Pelvic Bladder Dysfunction in ESRD: Neurological Assessment, Functional Bladder Markers, and Renal Safety of Relation to Serum Creatinine, Urea, and Electrolyte profile

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ABSTRACT

Urinary retention and neuro-pelvic bladder dysfunction represent underrecognized complications in patients with end-stage renal disease (ESRD), often resulting from complex interactions between neurological impairment, pelvic floor dysfunction, and progressive metabolic derangements. This cross-sectional study evaluated the relationship between neurogenic bladder dysfunction, neurological findings, functional bladder markers, and renal biochemical parameters among ESRD patients. Adult ESRD patients undergoing maintenance dialysis were assessed using neurological examination, post-void residual volume, uroflowmetry, and bladder compliance indices. Renal safety parameters included serum creatinine, urea, sodium, potassium, calcium, and bicarbonate levels. Of 280 enrolled participants, 112 (40.0%) exhibited significant urinary retention with neuro-pelvic bladder dysfunction. These patients demonstrated markedly higher post-void residual volumes and impaired bladder compliance alongside abnormal neurological findings ($p < 0.001$). Serum creatinine, urea, and potassium levels were significantly elevated in patients with bladder dysfunction, while bicarbonate and sodium levels were reduced ($p < 0.01$). Multivariate regression identified neuro-pelvic bladder dysfunction as an independent predictor of worsened electrolyte imbalance and azotemia after adjustment for age, dialysis duration, and diabetes status. This study provides novel evidence linking bladder dysfunction with biochemical instability in ESRD, emphasizing the importance of integrated neurological and urological assessment to improve renal safety and patient outcomes.

Keywords: *End-stage renal disease, Neurogenic bladder, Urinary retention*

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INTRODUCTION

End-stage renal disease (ESRD) represents the terminal phase of chronic kidney disease and is associated with extensive systemic complications affecting multiple organ systems. While cardiovascular, metabolic, and neurological sequelae of ESRD are well documented, lower urinary tract dysfunction remains an underexplored yet clinically relevant aspect of disease burden. Despite reduced or absent urine output in many patients, urinary retention and dysfunctional voiding patterns persist and may contribute to infection risk, pelvic discomfort, and metabolic instability [1-3].

Neuro-pelvic bladder dysfunction encompasses a spectrum of abnormalities involving detrusor muscle activity, sphincter coordination, and pelvic floor neuromuscular control. In ESRD, this dysfunction may arise from uremic neuropathy, diabetic autonomic neuropathy, spinal degenerative changes, or prolonged metabolic derangements affecting neural conduction. These neurological impairments can disrupt bladder sensation and contractility, leading to urinary retention even in patients with reduced urine volumes [4-5].

Uremic neuropathy is a recognized complication of ESRD, characterized by progressive sensory and motor deficits due to toxin accumulation and chronic inflammation. Autonomic nervous system involvement further compromises visceral organ regulation, including bladder function. Pelvic floor musculature, dependent on intact somatic and autonomic innervation, may exhibit dyssynergia, exacerbating voiding dysfunction. Despite these mechanisms, bladder evaluation is frequently overlooked in ESRD management [6-8].

Functional bladder markers such as post-void residual volume, uroflowmetry parameters, and bladder compliance provide objective insights into lower urinary tract performance. Abnormalities in these markers have been associated with urinary retention, recurrent infections, and upper tract deterioration in non-ESRD populations. In patients with ESRD, such dysfunction may influence intravesical pressures and residual urinary stasis, potentially contributing to systemic inflammatory responses and biochemical instability [9-12].

Renal biochemical parameters, including serum creatinine, urea, and electrolytes, are central to monitoring ESRD progression and dialysis adequacy. Electrolyte disturbances such as hyperkalemia, hyponatremia, and metabolic acidosis are common and life-threatening complications. Emerging evidence suggests that autonomic dysfunction and impaired bladder emptying may exacerbate these imbalances through altered neurohumoral regulation, inflammation, and recurrent infections.

The multidisciplinary nature of urinary retention in ESRD necessitates integrated evaluation involving nephrology, neurology, and urology. However, existing literature predominantly addresses these domains in isolation. Limited data are available examining how neuro-pelvic bladder dysfunction correlates with renal biochemical safety markers in ESRD populations, particularly within low- and middle-income countries.

Understanding this relationship is critical for optimizing patient management, as early identification of bladder dysfunction may provide an opportunity to mitigate secondary complications and improve metabolic stability. Therefore, the present study aimed to evaluate urinary retention and neuro-pelvic bladder dysfunction in ESRD patients through combined neurological assessment and functional bladder markers, while examining their association with serum creatinine, urea, and electrolyte profiles.

MATERIAL AND METHODS

Study Design and Setting

This cross-sectional study was conducted in the nephrology and urology units of a tertiary care hospital over an eight-month period.

Ethical Considerations

The study protocol was approved by the Institutional Human Ethical Committee of Sharif Medical and Dental college Lahore (Approval Number: SMDC/11.02.32). Verbal informed consent was obtained from all participants after explanation of study objectives and procedures.

Sample Size and Participants

Sample size was calculated using Epi Info software version 7, assuming a 95% confidence interval, 80% study power, an expected prevalence of urinary retention in ESRD of 35%, and a margin of error of 6%. A minimum required sample of 260 patients was determined, and 280 ESRD patients were enrolled to compensate for incomplete assessments.

Inclusion Criteria:

- Patients aged 30–70 years
- Confirmed ESRD undergoing maintenance hemodialysis for at least six months

Exclusion Criteria:

- Acute kidney injury
- Active urinary tract infection
- Prior urological surgery
- Known obstructive uropathy
- Malignancy
- Use of anticholinergic medications

Clinical Assessment

Neurological assessment included evaluation of lower limb reflexes, perineal sensation, and autonomic symptoms. Functional bladder assessment involved:

- Ultrasonographic measurement of post-void residual volume
- Uroflowmetry
- Bladder compliance estimation

Patients were categorized into groups with and without neuro-pelvic bladder dysfunction based on standardized criteria.

Biochemical Analysis

Laboratory investigations included measurement of serum creatinine, urea, sodium, potassium, calcium, and bicarbonate levels pre-dialysis. All biochemical analyses were performed in the hospital laboratory using standardized protocols to ensure accuracy and reproducibility.

Statistical Analysis

Data were analyzed using appropriate statistical software. Independent t-tests were used for continuous variables, chi-square tests for categorical variables, and multivariate regression analysis to identify independent predictors of neuro-pelvic bladder dysfunction. Statistical significance was set at $p < 0.05$.

RESULTS AND DISCUSSION

Table 1: Demographic and Clinical Characteristics of ESRD Patients

Variable	Bladder Dysfunction (n=112)	No Dysfunction (n=168)	p value
Age (years, mean ± SD)	56.8 ± 8.4	54.1 ± 7.9	0.01
Male sex (%)	68 (60.7)	96 (57.1)	0.56
Diabetes mellitus (%)	74 (66.1)	82 (48.8)	0.004
Dialysis duration (months, mean ± SD)	38.6 ± 12.9	29.4 ± 10.7	<0.001

Patients with bladder dysfunction were older, had longer dialysis duration, and a higher prevalence of diabetes.

Table 2: Neurological and Functional Bladder Parameters

Parameter	Bladder Dysfunction	No Dysfunction	p value
Post-void residual (mL, mean ± SD)	186.4 ± 42.7	48.2 ± 21.6	<0.001
Peak urinary flow (mL/s)	7.8 ± 2.4	14.6 ± 3.1	<0.001
Bladder compliance (mL/cmH_2O)	9.2 ± 3.1	18.5 ± 4.6	<0.001
Abnormal neurological findings (%)	81 (72.3)	46 (27.4)	<0.001

Marked impairment of bladder function and neurological status was observed in affected patients.

Table 3: Renal Biochemical and Electrolyte Profile

Parameter	Bladder Dysfunction	No Dysfunction	p value
Serum creatinine (mg/dL)	9.6 ± 2.1	8.1 ± 1.9	<0.001
Serum urea (mg/dL)	164.8 ± 34.6	139.2 ± 28.4	<0.001
Potassium (mmol/L)	5.7 ± 0.8	5.1 ± 0.6	<0.001
Sodium (mmol/L)	132.6 ± 4.9	136.4 ± 5.1	<0.001
Bicarbonate (mmol/L)	17.2 ± 3.8	20.6 ± 4.1	<0.001

Neuro-pelvic bladder dysfunction was associated with worse azotemia and electrolyte imbalance.

The present study demonstrates a strong association between urinary retention, neuro-pelvic bladder dysfunction, and biochemical instability in ESRD patients. Nearly two-fifths of the cohort exhibited clinically significant bladder dysfunction, highlighting its substantial yet underappreciated prevalence. The observed associations between bladder dysfunction and neurological abnormalities support the role of uremic and diabetic neuropathy in impairing lower urinary tract control [13-15].

Functional bladder markers revealed pronounced impairment among affected patients, with significantly elevated post-void residual volumes and reduced urinary flow and compliance. These findings suggest detrusor underactivity and impaired coordination, likely reflecting autonomic and somatic nerve involvement. The consistency of these abnormalities across multiple functional indices strengthens the validity of the findings [16, 17].

A notable contribution of this study is the demonstration of a significant relationship between bladder dysfunction and renal biochemical parameters. Patients with neuro-pelvic bladder dysfunction exhibited higher serum creatinine and urea levels, indicating poorer metabolic clearance despite ongoing dialysis.

This association may reflect increased systemic inflammation, recurrent subclinical urinary stasis, or autonomic dysregulation influencing renal and extrarenal clearance mechanisms [18-20].

Electrolyte disturbances were also more pronounced in patients with bladder dysfunction, particularly hyperkalemia, hyponatremia, and metabolic acidosis. Autonomic dysfunction may impair hormonal regulation of electrolyte balance, while inflammatory mediators may exacerbate cellular potassium shifts. These findings underscore the potential renal safety implications of untreated bladder dysfunction in ESRD.

The higher prevalence of diabetes and longer dialysis duration among affected patients suggests cumulative neurological injury as a contributory factor. Diabetes-related autonomic neuropathy, combined with uremic toxin exposure over time, likely accelerates neuro-pelvic dysfunction. These observations align with current understanding of multisystem involvement in ESRD.

Clinically, the results emphasize the necessity of multidisciplinary evaluation. Routine neurological screening and bladder function assessment may identify high-risk patients who could benefit from targeted interventions, such as bladder training, intermittent catheterization, or neuromodulatory strategies. Such measures may reduce complications and improve biochemical stability.

Although the cross-sectional design limits causal inference, the robust statistical associations and biological plausibility provide a compelling rationale for further longitudinal and interventional research. Incorporating bladder assessment into standard ESRD care protocols may represent a meaningful step toward holistic patient management.

CONCLUSION

Neuro-pelvic bladder dysfunction is common in ESRD and is significantly associated with urinary retention, neurological impairment, and adverse renal biochemical profiles. Multidisciplinary assessment of bladder and neurological function may enhance renal safety and metabolic stability in this vulnerable population.

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