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RESEARCH ARTICLE

Association Between Postvoid Residual Urine Volume and Estimated Glomerular Filtration Rate in Men with Benign Prostatic Obstruction

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Abstract

Objective: The relationship between postvoid residual urine volume (PVR) and estimated glomerular filtration rate (eGFR) in men with benign prostatic obstruction (BPO) remains controversial. This study aimed to evaluate the association between PVR and eGFR.

Methods: This retrospective study included 172 men with lower urinary tract symptoms (LUTS) secondary to BPO. Correlations between eGFR and clinical variables were evaluated using Spearman correlation analysis. Patients were stratified into four PVR categories (<50, 50–99, 100–149, and ≥150 mL), and eGFR values were compared using the Kruskal-Wallis test. Multivariable linear regression identified variables independently associated with eGFR.

Results: Median age was 67 years, median PVR was 100 mL, and median eGFR was 84 mL/min/1.73 m^2. PVR showed a moderate negative correlation with eGFR ($\rho = -0.356$, $p < 0.001$). Median eGFR differed significantly across PVR categories (90.5, 85.0, 78.0, and 79.5 mL/min/1.73 m^2; $p < 0.001$). In multivariable analysis, only age ($\beta = -0.369$, $p < 0.001$) and PVR ($\beta = -0.288$, $p < 0.001$) remained independently associated with eGFR. International Prostate Symptom Score (IPSS), prostate volume (PV), and urinary flow parameters were not independently associated with eGFR.

Conclusion: Higher PVR was independently associated with lower eGFR in men with BPO after excluding major causes of CKD. PVR may represent a clinically useful marker of reduced eGFR and may provide additional clinical value beyond IPSS, urinary flow parameters, and PV. Prospective studies are warranted to confirm these findings.

Keywords: prostatic hyperplasia, urinary retention, glomerular filtration rate, lower urinary tract symptoms, renal insufficiency

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INTRODUCTION

Benign prostatic hyperplasia (BPH) refers to a benign increase in prostate size in elderly men. It occurs in nearly 10% of men in their 40 s and up to 90% of men older than 80 years of age (1). Aging, genetic factors, hormones, and metabolic conditions contribute to the development of BPH (2). The clinical picture of BPH includes the presence of lower urinary tract symptoms (LUTS). These symptoms include voiding problems, such as a weak or hesitant urine stream, and storage problems, such as frequent urination, urinary urgency, and nocturia (1,2). Chronic kidney disease (CKD) is another common age-related condition and one of the main public health problems around the world, affecting about 850 million people (3). CKD is characterized by evidence of kidney damage, which includes abnormal findings detected by imaging or proteinuria and/or a decline in kidney function as assessed by glomerular filtration rate (4).

Although BPH is the underlying pathological condition, LUTS and bladder outlet obstruction are more directly related to benign prostatic obstruction (BPO), which represents the functional manifestation of prostatic enlargement in affected patients. Many studies have investigated the relationship between parameters related to LUTS/BPO and kidney function. Results regarding symptom severity have been conflicting. Rule et al. (5) found that moderate-to-severe LUTS was independently associated with CKD. Nevertheless, following reports have found no evidence to support this relationship; Hong et al. (6) observed no independent association between the International Prostate Symptom Score (IPSS) and CKD, and Lee et al. (7) did not observe a significant correlation between the IPSS and estimated glomerular filtration rate (eGFR), and Hallan et al. (8), after adjusting for age and other risk factors, reported that LUTS was not independently associated with future kidney failure. Likewise, Bauer et al. (9) indicated that, after adjusting for important confounding factors, LUTS severity was not independently associated with multiple measures of kidney dysfunction.

There seems to be a more definite relation of impaired renal function with a reduced maximum urinary flow rate (Qmax), in contrast. According to the findings of Rule et al. (5) and Hong et al. (6), reduced peak flow rates are independently associated with CKD. In concordance with the previous studies, Lee et al. (7) found that Qmax was the single BPO-related parameter that was still significantly associated with eGFR after multivariable adjustment.

The current evidence related to the role of postvoid residual urine volume (PVR) in the progression of CKD appears to be both limited and inconsistent. Rule et al. (5) found a nonsignificant positive relation between chronic urinary retention and CKD, while Hong et al. (6) and Lee et al. (7) failed to show an independent association of PVR with the renal function. Contrary to this, however, Yamasaki et al. (10) found that as low as PVR ≥12 mL, are independently associated with CKD in BPH patients. These findings point out that the relationship between PVR and renal dysfunction remains unclear.

Therefore, given the conflicting evidence regarding the association between PVR and eGFR, we aimed to evaluate the association in men with LUTS due to BPO. The secondary analysis was conducted to explore the associations of IPSS and Qmax with eGFR.

MATERIALS AND METHODS

Study Design and Patient Selection

The retrospective study included male patients who were followed up at the urology outpatient clinic between November 20, 2022, and March 15, 2026, due to lower urinary tract symptoms (LUTS) and who received a clinical diagnosis of BPH/BPO. The diagnosis was based on clinical evaluation documented in medical records; urodynamic confirmation of bladder outlet obstruction was not available routinely. Patient medical files were analyzed to select eligible patients. The study was approved by the Clinical Research Ethics Committee of Ankara Etlik City Hospital (Approval No: AEŞH-BADEK2–2026–434, Date: June 30, 2026) and was conducted in accordance with the principles of the Declaration of Helsinki.

Patients who met the inclusion criteria were aged >40 years, had LUTS and had uroflowmetric studies, PVR, serum creatinine, and prostate volume (PV) available from the same examination period.

Patients with chronic kidney disease (eGFR <60 mL/min/1.73 m^2), diabetes mellitus, hypertension, neurogenic

bladder dysfunction, urethral stricture disease, urinary tract malignancy, active urinary tract infection, lower urinary tract reconstruction, hydronephrosis, obstruction of the upper urinary tract, urinary retention requiring catheterization, and incomplete clinical information were excluded.

Patients with very low Qmax (<5 mL/s) were excluded to reduce the potential inclusion of patients with unrecognized urethral stricture disease.

Data Collection

Demographic and clinical characteristics were extracted from electronic patient records. The recorded variables were as follows: age, body mass index (BMI), IPSS, Qmax, average urinary flow rate (Qavg), PVR, PV, prostate-specific antigen (PSA), serum creatinine level, and eGFR. PV and PVR were measured using transabdominal ultrasonography. Uroflowmetry was performed as part of the routine clinical assessment, and Qmax and Qavg values were recorded. Only uroflowmetry measurements with a voided volume ≥150 mL were included in the analysis, considering the influence of voided volume on uroflowmetry parameters (11). PVR was determined by bladder ultrasonography immediately after uroflowmetry. A single PVR measurement obtained during the index clinical assessment was used for the analysis. eGFR values, routinely calculated in our institution using the CKD-EPI equation and recorded in the electronic medical records, were used for analysis. To reduce the influence of biological and analytical variability, the mean of the three most recent eGFR measurements available before or during the clinical assessment period were used for analysis. The interval between these measurements was not standardized because of the retrospective study design.

Study Outcomes

The primary objective of the study was to evaluate the association between PVR and eGFR. Secondary objectives were to evaluate the associations of IPSS, Qmax, and PV with eGFR.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 22 (IBM Corp., Armonk, NY, USA). Data distribution was examined with the help of histograms and tests for skewness. Data were presented as mean ± standard deviation or median (interquartile range), as appropriate. The correlations of eGFR with the studied parameters were determined using the Spearman correlation test. To simplify clinical interpretation, PVR was divided into four groups (<50, 50–99, 100–149, and ≥150 mL) using PVR ranges previously described in men with LUTS, and values of ≥150 mL were grouped into one category (12) and eGFR values were compared with the Kruskal-Wallis test. Variables potentially associated with eGFR were subsequently entered into a multivariable linear regression model with eGFR as the dependent variable. A 2-sided p-value <0.05 was considered statistically significant.

RESULTS

A total of 172 patients were included in the final analysis. The median age was 67 years, median IPSS was 21, median Qmax was 9.0 mL/s, and median PVR was 100 mL. The median eGFR was 84 mL/min/1.73 m² (Table 1).

Spearman's correlation analysis showed that PVR and age had a moderate negative correlation with eGFR (both $p < 0.001$). In addition, no significant correlation was observed between eGFR and BMI, PSA, PV, IPSS, Qmax, or Qavg (all $p > 0.05$) (Table 2).

Table 1. Baseline characteristics of the study population

Variable	Value
Age (years), median (IQR)	67 (63–72)
BMI (kg/m²), mean ± SD	27.6 ± 4.0
Total PSA (ng/mL), median (IQR)	3.0 (1.6–9.0)
PV (mL), median (IQR)	65 (50–87.5)
IPSS, median (IQR)	21 (18–24)
Qmax (mL/s), median (IQR)	9.0 (7.2–11.4)
Qavg (mL/s), median (IQR)	4.0 (3.1–5.2)
PVR (mL), median (IQR)	100 (45–150)
eGFR (mL/min/1.73 m²), median (IQR)	84 (72–91)
Serum creatinine (mg/dL), median (IQR)	0.93 (0.83–1.04)

IQR, interquartile range; SD, standard deviation; BMI, body mass index; PSA, prostate-specific antigen; PV, prostate volume; IPSS, International Prostate Symptom Score; Qmax, maximum urinary flow rate; Qavg, average urinary flow rate; PVR, postvoid residual urine volume; eGFR, estimated glomerular filtration rate.

Median eGFR values were generally lower in the higher PVR categories (90.5, 85.0, 78.0, and 79.5 mL/min/1.73 m² for <50, 50–99, 100–149, and ≥150 mL, respectively). Overall differences between groups were statistically significant ($p < 0.001$) (Table 3, Figure 1).

In multivariable linear regression analysis including age, BMI, PV, IPSS, Qmax, Qavg, and PVR, only age and PVR (both $p < 0.001$) remained independently associated with eGFR. Neither BMI, IPSS, PV, nor urinary flow parameters showed a significant independent association with eGFR (all $p > 0.05$). The overall model explained 27.0% of the variability in eGFR ($R^2 = 0.270$, adjusted $R^2 = 0.238$, $p < 0.001$) (Table 4).

Table 2. Correlation between eGFR and clinical parameters

Variable	Spearman rho	p value
Age	-0.453	**<0.001**
BMI	-0.096	0.209
Total PSA	0.012	0.880
PV	-0.135	0.078
IPSS	-0.079	0.304
Qmax	-0.105	0.172
Qavg	-0.052	0.501
PVR	-0.356	**<0.001**

BMI, body mass index; PSA, prostate-specific antigen; PV, prostate volume; IPSS, International Prostate Symptom Score; Qmax, maximum urinary flow rate; Qavg, average urinary flow rate; PVR, postvoid residual urine volume; eGFR, estimated glomerular filtration rate.

Table 3. eGFR according to postvoid residual urine volume categories

PVR category	eGFR median (IQR)
<50 mL	90.5 (83.0–96.0)
50–99 mL	85.0 (76.5–92.0)
100–149 mL	78.0 (70.0–86.0)
≥150 mL	79.5 (68.0–89.0)

Kruskal–Wallis test, $p < 0.001$.

IQR, interquartile range; PVR, postvoid residual urine volume; eGFR, estimated glomerular filtration rate.

Table 4. Multivariable linear regression analysis for factors independently associated with eGFR

Variable	β	B (95% CI)	p value
Age	-0.369	-0.651 (-0.899 to -0.403)	**<0.001**
BMI	-0.132	-0.391 (-0.790 to 0.008)	0.055
PV	0.078	0.027 (-0.022 to 0.077)	0.279
IPSS	-0.026	-0.068 (-0.424 to 0.287)	0.704
Qmax	-0.157	-0.516 (-1.165 to 0.132)	0.118
Qavg	0.114	0.760 (-0.555 to 2.076)	0.255
PVR	-0.288	-0.050 (-0.074 to -0.026)	**<0.001**

Model statistics: $R^2 = 0.270$; adjusted $R^2 = 0.238$; $F = 8.649$; $p < 0.001$.

β, standardized regression coefficient; B, unstandardized regression coefficient; CI, confidence interval; BMI, body mass index; PV, prostate volume; IPSS, International Prostate Symptom Score; Qmax, maximum urinary flow rate; Qavg, average urinary flow rate; PVR, postvoid residual urine volume; eGFR, estimated glomerular filtration rate.

DISCUSSION

The clinical significance of elevated PVR remains an area of ongoing investigation. According to a recent study, the pathophysiology and long-term complications of chronic urinary retention are not fully understood, and further studies are needed to understand its effect on kidney function. The authors also proposed that patients with elevated PVR should be stratified according to their risk of renal deterioration and it can be noted here high-risk patients require close monitoring of kidney function (13).

Against this background, we evaluated the association between PVR and eGFR in men with LUTS secondary to BPO after excluding major causes of chronic kidney disease. We found that higher PVR was significantly associated with lower eGFR values. PVR showed a moderate negative correlation with eGFR and remained independently associated with eGFR after adjusting for age, BMI, IPSS, urinary flow parameters, and PV. Moreover, median eGFR varied significantly among PVR groups, as in general lower eGFR values have been observed in higher PVR groups. Although all patients had an eGFR ≥60 mL/min/1.73 m² by study design, the lower eGFR values observed in the higher PVR categories suggest that the association between PVR and eGFR may also be present among patients without established CKD.

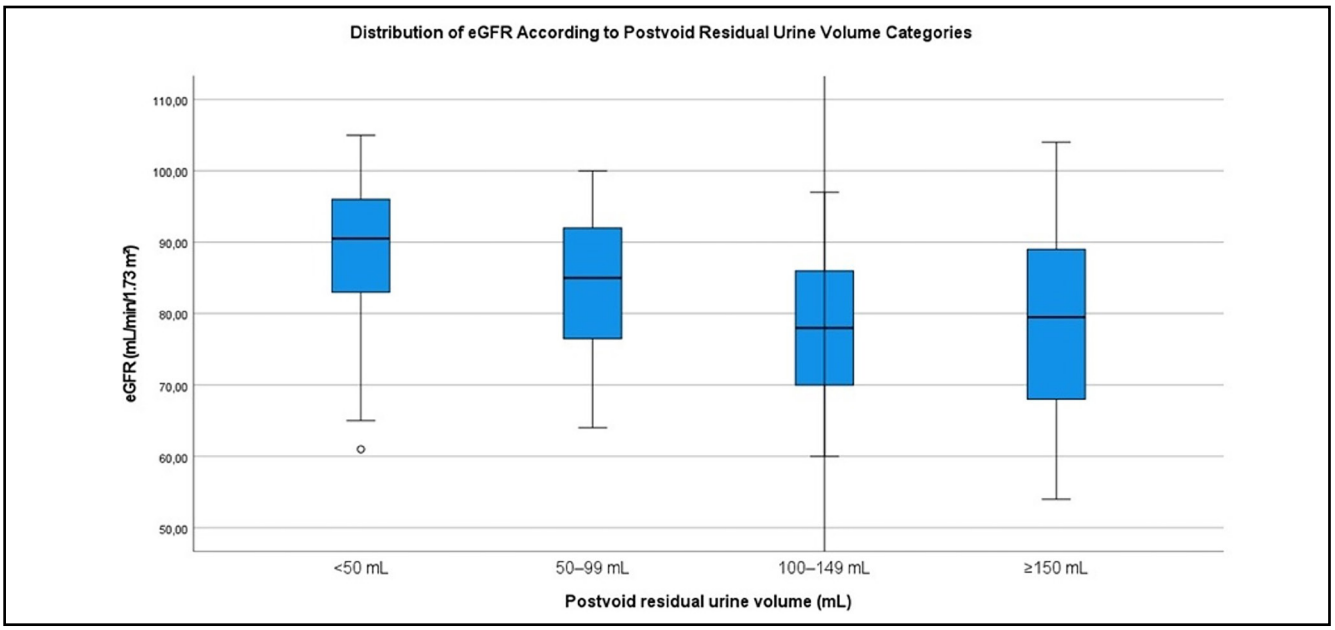


Figure 1. Distribution of eGFR According to Postvoid Residual Urine Volume Categories.

The relationship between PVR and eGFR is still unclear. A study highlighted that men with PVR >100 mL had higher odds of CKD, but this association was not statistically significant due to wide confidence intervals (5). Similar results were provided by Yamasaki et al. (10), who showed that relatively small amounts of PVR were independently associated with CKD in men suffering from BPH. In contrast, two studies demonstrated no independent association between PVR and kidney function (6,7). These findings suggest that the relationship between PVR and eGFR is potentially related to differences in study populations, definitions of CKD, and the existence of significant comorbidities. Our results contribute to the growing evidence suggesting an association between increased PVR and reduced eGFR and indicate that PVR may serve as a clinically useful marker of lower eGFR in selected patients with BPO.

There are several possible explanations for the association between high PVR and reduced eGFR. Chronic urinary retention may lead to persistent high intravesical pressure, resulting in functional obstruction at the ureterovesical junction and poor drainage of the upper urinary tract. Persistent increases in intravesical pressure may also lead to vesicoureteral reflux, hydronephrosis, and progressive kidney damage (14,15). Prior studies have indicated that detrusor hypertrophy, bladder wall remodeling, and reduced bladder compliance may play a significant role in upper urinary tract dysfunction and may be more critical factors in determining renal dysfunction than PVR itself (14,16). Furthermore, it has been suggested that recurrent urinary tract infections and bladder stone formation are possible contributing factors to chronic urinary retention and impaired renal function (15). These pathophysiological mechanisms serve as a reasonable biological explanation for the observed association in the present study.

In this study, PV, IPSS, and urinary flow rates were not individually correlated with eGFR. In previous studies, a negative correlation between Qmax and kidney function has been identified. Hong et al. (6) reported an independent correlation between lower Qmax and CKD among male patients with LUTS due to BPH, while Lai et al. (17) revealed significant relationships between Qmax and renal function parameters in cases of CKD. Nevertheless, data on symptom severity and PV remain controversial. Kwon et al. (18) found the relationship between LUTS and kidney function to be independent of PV, implying that mere anatomical enlargement does not indicate functional severity of the disease. Similarly, the independent correlation between symptom severity and renal dysfunction was not observed by Hamm and MacDermott et al. (19) and Ponholzer et al. (20). Taken into consideration, these findings indicate that objective measures of bladder emptying dysfunction may be more useful than IPSS or PV when assessing the potential

impact of BPO on eGFR. In our cohort, PVR was the only lower urinary tract parameter that remained independently associated with eGFR, supporting its potential value as a clinically relevant marker of reduced eGFR.

This study's key strengths were its well-defined and carefully selected study population. We excluded patients with diabetes mellitus, hypertension, and previously diagnosed chronic kidney disease, which are well-established risk factors for impaired renal function and CKD progression (21, 22). Because renal function is influenced by multiple systemic factors, residual confounding may partly explain the inconsistent findings reported in previous studies evaluating the association between LUTS/BPO-related parameters and kidney function (23). By excluding these major sources of confounding, we were able to more specifically evaluate the association between BPO-related parameters and eGFR in patients with BPO. This methodological approach may partly explain why PVR remained independently associated with eGFR after multivariable adjustment.

Study limitations: This study has several limitations. Firstly, due to its retrospective and single-center design, our study cannot establish the causality between PVR and reduced eGFR. Second, although the study eGFR was defined as the mean of the three most recent eGFR measurements, the interval between these measurements was not standardized because of the retrospective design. In addition, longitudinal follow-up data were not available; therefore, changes in eGFR over time could not be evaluated. Third, PVR was based on a single measurement, and potential intra-individual variability in residual urine volume could not be assessed. Fourth, other markers of kidney damage, such as albuminuria or proteinuria, were not routinely assessed. In addition, objective assessment of bladder outlet obstruction by urodynamic studies was not available. Despite these limitations, the rigorous exclusion of patients with major causes of renal dysfunction allowed a more specific evaluation of the independent association between PVR and eGFR in patients with BPO.

CONCLUSION

Increased PVR was independently associated with reduced eGFR in men with LUTS secondary to BPO after excluding major causes of chronic kidney disease. PVR may therefore provide additional clinical value beyond IPSS, urinary flow parameters, and PV when assessing reduced eGFR. Prospective studies are required for confirmation of the results and assessment of possible association between changes in PVR and eGFR in time.

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Conflict of Interest: The authors declare that they have no conflicts of interest related to this study.

Informed Consent: Written informed consent was obtained from all participants for the use of their clinical data for scientific purposes.

Ethical Approval: The study was approved by the Clinical Research Ethics Committee of Ankara Etlik City Hospital (Approval No: AEŞH-BADEK2–2026–434, Date: June 30, 2026) and was conducted in accordance with the principles of the Declaration of Helsinki.

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Supervision: AED
Data Collection and/or Analysis: MYA
Analysis and/or Interpretation: AED, MYA
Literature Search: AED, MYA
Writing: AED, MYA
Critical Review: AED

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