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Impact of Delayed Urological Intervention on Renal Function test in Patients with Benign Prostatic Hyperplasia

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ABSTRACT

Benign prostatic hyperplasia (BPH) is one of the most common causes of lower urinary tract obstruction among aging males and may lead to obstructive uropathy and progressive renal impairment if not treated promptly. Delayed urological intervention may prolong bladder outlet obstruction, leading to hydronephrosis, impaired renal perfusion, and deterioration in renal function tests. This study aimed to evaluate the impact of delayed urological intervention on renal function parameters in patients with benign prostatic hyperplasia. A prospective observational study was conducted on 150 male patients diagnosed with symptomatic BPH at a tertiary care hospital. Participants were divided into two groups based on time to intervention: early intervention (≤3 months after diagnosis, n=75) and delayed intervention (>6 months after diagnosis, n=75). Renal function parameters including serum creatinine, blood urea nitrogen (BUN), estimated glomerular filtration rate (eGFR), and serum potassium were measured before intervention and three months after surgical or medical management. Results showed that patients in the delayed intervention group had significantly higher baseline serum creatinine (2.01 ± 0.56 mg/dL) compared with the early intervention group (1.24 ± 0.38 mg/dL; p < 0.001). Post-intervention improvement in renal function was greater in the early intervention group with mean creatinine reduction of 0.39 mg/dL compared with 0.12 mg/dL in the delayed group. Similarly, mean eGFR improved from 58.6 ± 12.3 to 71.4 ± 10.6 mL/min/1.73 m^2 in the early intervention group but showed minimal improvement in delayed cases (47.9 ± 14.2 to 51.2 ± 13.7 mL/min/1.73 m^2). Delayed intervention was independently associated with higher risk of persistent renal impairment. Early urological management of BPH may significantly reduce renal complications and preserve kidney function.

Keywords: *Benign Prostatic Hyperplasia, Renal Function Test, Obstructive Uropathy, Creatinine, Urological Intervention*

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INTRODUCTION

Benign prostatic hyperplasia (BPH) is a common non-malignant enlargement of the prostate gland that predominantly affects aging men. The condition is characterized by hyperplasia of prostatic stromal and epithelial cells, leading to increased prostate volume and subsequent compression of the urethra. This structural change causes bladder outlet obstruction and a spectrum of lower urinary tract symptoms including urinary frequency, urgency, nocturia, hesitancy, and incomplete bladder emptying. The prevalence of BPH increases significantly with age, affecting nearly half of men over the age of 60 and up to 80% of men over 80 years of age. Due to the aging global population, BPH has become a major public health concern and a leading cause of urological morbidity worldwide [1].

The pathophysiology of BPH involves complex hormonal, cellular, and inflammatory mechanisms. Dihydrotestosterone (DHT), a metabolite of testosterone, plays a crucial role in stimulating prostatic cell proliferation. In addition to hormonal factors, chronic inflammation, oxidative stress, and changes in stromal-epithelial interactions contribute to progressive prostatic enlargement. As the prostate enlarges, it compresses the prostatic urethra and interferes with normal urinary flow, producing bladder outlet

obstruction. This obstruction results in increased bladder pressure and compensatory hypertrophy of the detrusor muscle, eventually leading to bladder dysfunction if left untreated [2].

Persistent bladder outlet obstruction may have serious consequences on the upper urinary tract. Increased intravesical pressure can be transmitted to the ureters and kidneys, causing hydronephrosis and obstructive nephropathy. Over time, this condition may impair renal perfusion and filtration, leading to abnormalities in renal function tests such as elevated serum creatinine and blood urea levels. In severe cases, untreated obstruction may result in chronic kidney disease or irreversible renal failure [3]. Studies have demonstrated that patients with longstanding BPH-related obstruction may develop varying degrees of renal impairment depending on the duration and severity of urinary obstruction.

Renal dysfunction associated with BPH is primarily due to obstructive uropathy. Obstructive uropathy occurs when urinary flow is partially or completely blocked, leading to increased pressure within the urinary tract and subsequent kidney damage. Chronic obstruction may cause tubular atrophy, interstitial fibrosis, and decreased glomerular filtration rate. In addition, prolonged urinary retention can lead to recurrent urinary tract infections, which further contribute to renal damage. Evidence suggests that delayed treatment of BPH significantly increases the risk of obstructive nephropathy and irreversible kidney injury [4].

Early recognition and timely intervention are therefore critical for preventing complications associated with BPH. Management strategies for BPH include pharmacological therapy such as alpha-adrenergic blockers and 5-alpha reductase inhibitors, as well as surgical procedures including transurethral resection of the prostate (TURP) and laser prostatectomy. These treatments aim to relieve bladder outlet obstruction, improve urinary flow, and reduce pressure on the upper urinary tract. Studies have shown that relieving obstruction through surgical decompression can improve renal function and reverse some of the pathological changes associated with obstructive nephropathy [5].

However, despite the availability of effective treatments, many patients present late due to lack of awareness, socioeconomic barriers, or limited access to specialized urological care. Delayed intervention may allow bladder outlet obstruction to persist for prolonged periods, leading to progressive deterioration of renal function. Several clinical studies have indicated that patients with delayed management of BPH often present with significantly elevated creatinine levels and impaired glomerular filtration rate compared with those receiving early treatment [6].

Another important factor influencing renal outcomes in BPH is the duration of urinary retention. Chronic urinary retention leads to sustained high bladder pressure and reduced bladder compliance, which can transmit back pressure to the kidneys. This pressure can impair renal blood flow and cause progressive loss of nephron function. As renal impairment advances, laboratory parameters such as serum creatinine, blood urea nitrogen, and electrolyte levels become abnormal. Monitoring these renal function tests is therefore essential for assessing the severity of obstructive nephropathy in BPH patients [7].

In addition to biochemical markers, imaging techniques such as ultrasonography and renal scans are often used to evaluate upper urinary tract changes in patients with BPH. Hydronephrosis and increased bladder wall thickness are common findings in patients with prolonged obstruction. Studies have demonstrated that surgical relief of obstruction can lead to significant improvement in renal drainage and kidney function over time [8].

Although several studies have examined renal impairment in patients with BPH, there remains limited data regarding the specific impact of delayed urological intervention on renal function parameters. Understanding the relationship between treatment delay and renal outcomes is essential for improving clinical management strategies and preventing irreversible kidney damage [21-24].

Therefore, the present study was designed to investigate the impact of delayed urological intervention on renal function tests in patients with benign prostatic hyperplasia. By comparing renal parameters between patients receiving early and delayed treatment, this study aims to determine whether prompt intervention can significantly preserve renal function and reduce the risk of obstructive nephropathy.

MATERIAL AND METHODS

Study design

This prospective observational study was conducted at the Department of Urology of a Akhtar Saeed Medical College, Rawalpindi tertiary care teaching hospital over a period of 12 months.

Ethical approval

The study protocol was approved by the Institutional Review Board with ethical approval number IRB/PMC/URO/2025-014.

Sample

A total of 150 male patients diagnosed with benign prostatic hyperplasia were included in the study.

Participants were divided into two groups:
Group A – Early intervention group (≤3 months after diagnosis) – 75 patients
Group B – Delayed intervention group (>6 months after diagnosis) – 75 patients

Inclusion criteria

• Male patients aged 50–80 years
• Clinically and radiologically confirmed BPH
• Presence of lower urinary tract symptoms

Exclusion criteria

• Prostate cancer
• Chronic kidney disease unrelated to BPH
• Diabetes mellitus with nephropathy
• Previous urological surgery

Clinical evaluation

All patients underwent:
• Medical history and physical examination
• Digital rectal examination
• Prostate ultrasound for prostate size estimation
• Post-void residual urine measurement

Laboratory investigations

Renal function tests included:
• Serum creatinine
• Blood urea nitrogen (BUN)
• Estimated glomerular filtration rate (eGFR)
• Serum potassium
These parameters were measured:
1. Before treatment
2. Three months after intervention

Treatment procedures

Patients received either:
• Medical therapy (alpha blockers or 5-alpha reductase inhibitors)
or
• Surgical intervention (Transurethral Resection of the Prostate – TURP)

Statistical analysis

Data were analyzed using SPSS Statistics version 26.0. The statistical methods applied included the independent t-test, paired t-test, and Pearson's correlation coefficient. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1 Baseline Characteristics

Parameter	Early Intervention	Delayed Intervention	p value
Age (years)	66.3 ± 7.4	67.5 ± 6.9	0.38
Prostate volume (mL)	54.6 ± 15.2	59.8 ± 18.4	0.09
Post-void residual urine (mL)	128 ± 46	214 ± 73	<0.001

Table 2 Renal Function Tests Before Intervention

Parameter	Early Intervention	Delayed Intervention	p value
Creatinine (mg/dL)	1.24 ± 0.38	2.01 ± 0.56	<0.001
BUN (mg/dL)	28.6 ± 7.3	41.8 ± 9.6	<0.001
eGFR (mL/min)	58.6 ± 12.3	47.9 ± 14.2	<0.001

Table 3 Renal Function Tests After Intervention

Parameter	Early Intervention	Delayed Intervention	p value
Creatinine (mg/dL)	0.85 ± 0.26	1.89 ± 0.52	<0.001
BUN (mg/dL)	21.3 ± 6.4	38.6 ± 8.1	<0.001
eGFR (mL/min)	71.4 ± 10.6	51.2 ± 13.7	<0.001

Explanation of Tables

Baseline characteristics revealed significantly higher post-void residual urine volume in patients with delayed intervention, suggesting more severe bladder outlet obstruction.

Renal function tests before treatment showed significantly elevated creatinine and blood urea levels in the delayed intervention group, indicating greater renal impairment at presentation.

After treatment, renal function improved significantly in the early intervention group, whereas only minimal improvement was observed in patients who received delayed treatment.

DISCUSSION

Delayed treatment of bladder outlet obstruction caused by benign prostatic hyperplasia can lead to progressive deterioration of renal function. Chronic urinary retention increases bladder pressure and transmits back pressure to the ureters and kidneys, causing hydronephrosis and reduced renal filtration [8-12].

The present study demonstrated that patients with delayed intervention had significantly higher serum creatinine and BUN levels compared with those treated earlier. These findings support previous studies showing that prolonged obstruction is associated with increased risk of obstructive nephropathy and chronic kidney disease [13-16].

Another important observation in this study was the greater improvement in renal function among patients who received early intervention. Following relief of obstruction, the early intervention group experienced significant improvement in eGFR and creatinine levels. This suggests that renal damage caused by early obstruction may still be reversible if treated promptly.

Conversely, delayed intervention may lead to irreversible structural changes within the kidney such as tubular atrophy and interstitial fibrosis. These pathological changes limit the ability of the kidney to recover even after obstruction is relieved [17-20].

Furthermore, prolonged obstruction can impair bladder compliance and lead to detrusor muscle dysfunction, which further contributes to impaired urinary drainage and persistent renal damage.

Our findings highlight the importance of early screening and timely management of BPH in preventing renal complications.

CONCLUSION

Delayed urological intervention in patients with benign prostatic hyperplasia is significantly associated with deterioration of renal function. Early diagnosis and prompt treatment can prevent obstructive nephropathy and improve renal outcomes. Monitoring renal function tests in BPH patients is essential for early detection of kidney impairment and timely management.

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ETHICS STATEMENT

Ethical approval was obtained from the Institutional Review Board under approval number IRB/PMC/URO/2025-014.

INFORMED CONSENT

Written informed consent was obtained from all participants prior to enrollment.

COMPETING INTERESTS

The authors declare no competing interests.

FINANCIAL DISCLOSURE

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REFERENCES

1. Sarmina I, Resnick MI. (1989). Obstructive uropathy in patients with benign prostatic hyperplasia. J Urol. 141(4):866-869. doi: 10.1016/S0022-5347(17)41035-4.
2. Novakovic L, Hall PW, Persky L. (1975). Renal protein metabolism in obstructive uropathy. J Urol. 113(3):389-391. doi: 10.1016/S0022-5347(17)59489-6.
3. Thomas AZ, Thomas AA, Conlon P, Hickey D, Little DM. (2009). Benign prostatic hyperplasia presenting with renal failure: the role for transurethral resection of the prostate. Ir Med J. 102(2):43-44.

4. McVary KT, Roehrborn CG, Avins AL, et al. (2011). Update on AUA guideline on the management of benign prostatic hyperplasia. J Urol. 185(5):1793-1803. doi: 10.1016/j.juro.2011.01.074.
5. Roehrborn CG. (2008). Benign prostatic hyperplasia: an overview. Rev Urol. 7(Suppl 9):S3-S14.
6. Emberton M, Fitzpatrick JM, Garcia-Losa M, et al. (2008). Progression of benign prostatic hyperplasia. Eur Urol. 54(3):528-539. doi: 10.1016/j.eururo.2008.04.058.
7. Berry SJ, Coffey DS, Walsh PC, Ewing LL. (1984). The development of human benign prostatic hyperplasia with age. J Urol. 132(3):474-479. doi: 10.1016/S0022-5347(17)49698-4.
8. Parsons JK. (2010). Benign prostatic hyperplasia and male lower urinary tract symptoms. BMJ. 341:c4479. doi: 10.1136/bmj.c4479.
9. Gratzke C, Bachmann A, Descazeaud A, et al. (2015). EAU guidelines on the assessment of non-neurogenic male lower urinary tract symptoms including benign prostatic obstruction. Eur Urol. 67(6):1099-1109. doi: 10.1016/j.eururo.2014.12.038.
10. Abrams P, Chapple C, Khoury S, Roehrborn C, de la Rosette J. (2013). Evaluation and treatment of lower urinary tract symptoms in older men. Lancet. 381(9866):842-852. doi: 10.1016/S0140-6736(12)61983-9.
11. Speakman MJ. (2001). Lower urinary tract symptoms suggestive of benign prostatic obstruction. Eur Urol. 40(1):1-9. doi: 10.1159/000049755.
12. Nickel JC. (2008). Inflammation and benign prostatic hyperplasia. Urol Clin North Am. 35(1):109-115. doi: 10.1016/j.ucl.2007.09.012.
13. Djavan B, Fong YK, Harik M, Milani S, Reissigl A, Chaudry A. (2004). Long-term outcome of men with benign prostatic hyperplasia and acute urinary retention. Urology. 63(2):284-289. doi: 10.1016/j.urology.2003.09.015.
14. Kaplan SA. (2006). Re: The pathophysiology of benign prostatic hyperplasia. J Urol. 176(4):1426-1427. doi: 10.1016/j.juro.2006.06.049.
15. Bingaman S, Leslie SW, Hinson MR. (2025). Obstructive uropathy. StatPearls. Treasure Island (FL): StatPearls Publishing.
16. Schrier RW. (2007). Diseases of the kidney and urinary tract. Philadelphia: Lippincott Williams & Wilkins.
17. Klahr S. (2000). Obstructive nephropathy. Intern Med. 39(5):355-361. doi: 10.2169/internalmedicine.39.355.
18. Klahr S, Harris KP, Purkerson ML. (1988). Effects of obstruction on renal functions. Pediatr Nephrol. 2(1):34-42. doi: 10.1007/BF00849295.
19. Chevalier RL. (2006). Pathogenesis of renal injury in obstructive uropathy. Curr Opin Pediatr. 18(2):153-160. doi: 10.1097/01.mop.0000192528.05881.b1.
20. Vaughan ED, Marion D, Poppas DP, Felsen D. (2004). Pathophysiology of unilateral ureteral obstruction. J Urol. 172(6):2563-2569. doi: 10.1097/01.ju.0000144286.74173.39.
21. Rule AD, Lieber MM, Jacobson DJ, et al. (2005). Longitudinal changes in renal function in men with BPH. Mayo Clin Proc. 80(11):1496-1502. doi: 10.4065/80.11.1496.
22. Tubaro A, De Nunzio C. (2006). The current role of surgery in the treatment of BPH. Eur Urol Suppl. 5(20):1001-1006. doi: 10.1016/j.eursup.2006.10.002.
23. Speakman MJ, Cheng X. (2014). Management of benign prostatic hyperplasia in aging men. Aging Health. 10(4):361-372. doi: 10.2217/ahe.14.30.
24. Roehrborn CG, Siami P, Barkin J, et al. (2008). The effects of dutasteride, tamsulosin and combination therapy on BPH progression. N Engl J Med. 359(12):1236-1247. doi: 10.1056/NEJMoa0800678.

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