International Surgery Journal
Bishnoi D et al. Int Surg J. 2026 Sep;13(9):1689-1693
https://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: https://dx.doi.org/10.18203/2349-2902.isj20262996

Original Research Article

Effect of rigid cystoscopy and Foley urethral catheterisation on serum prostate-specific antigen levels: a prospective observational study

Deepika Bishnoi, Dharamanjai K. Sharma*, Akhil Yadav

Department of General Surgery, R. N. T. Medical College, Udaipur, Rajasthan, India

Received: 30 June 2026
Accepted: 06 August 2026

***Correspondence:**
Dr. Dharamanjai K. Sharma,
E-mail: drdksurg@gmail.com

ABSTRACT

Background: Serum prostate-specific antigen (PSA) is the cornerstone biomarker for prostate cancer detection, but its interpretation is complicated by transient elevations from non-malignant causes including mechanical urinary-tract manipulation. How much rigid cystoscopy and Foley catheterisation raise serum PSA individually and combined in one sitting remains poorly defined in the Indian setting. This study evaluated the magnitude and persistence of procedure-induced PSA elevation.
Methods: This prospective observational study was conducted at RNT Medical College and Maharana Bhupal Government Hospital, Udaipur, over one year (January–December 2025). Sixty male patients were grouped by procedure: Group 1 diagnostic rigid cystoscopy alone (n=39); Group 2 Foley catheterisation alone (n=14); Group 3 Foley catheterisation followed immediately by cystoscopy in the same sitting (n=7). Serum total PSA was measured by ELISA at baseline, 1 hour and 24 hours. Paired t-tests and one-way ANOVA with Bonferroni correction were applied.
Results: In Group 1, PSA rose from 1.96±1.51 to 2.72±1.81 ng/ml at 1 hour (ΔPSA 0.76 ng/ml; 38.7% rise; $p<0.001$) with no recovery at 24 hours ($p<0.001$). Group 2 showed only a small rise (ΔPSA 0.54 ng/ml; $p=0.035$). Group 3 showed the largest elevation (ΔPSA 3.97±5.17 ng/ml; 69.1% rise) and was significantly greater than Group 1 at 1 hour (Bonferroni $p=0.001$) and 24 hours ($p=0.024$). BPH patients had greater ΔPSA than non-BPH (1.95 vs 0.54 ng/mL; $p=0.011$); baseline PSA correlated strongly with ΔPSA ($r=0.753$; $p<0.001$).
Conclusions: Rigid cystoscopy causes a clinically significant, sustained PSA elevation that does not recover within 24 hours. Combining Foley catheterisation and cystoscopy in one sitting produces a markedly greater rise. PSA should be deferred at least 48–72 hours after these procedures and longer in BPH patients, to avoid misleading results and unnecessary biopsies.

Keywords: Benign prostatic hyperplasia, Cystoscopy, Foley catheterisation, Prostate-specific antigen, Urological procedures

INTRODUCTION

PSA is a glycoprotein enzyme produced by the secretory epithelium of the prostate gland and is the most widely used tool for prostate cancer screening and treatment monitoring. Its organ specificity is well recognised, but it is far from disease-specific elevated readings are routinely encountered in benign prostatic hyperplasia (BPH), prostatitis, acute urinary retention and after any mechanical disturbance to the prostate or lower urinary tract.[1,2]

Rigid cystoscopy and Foley urethral catheterisation are among the commonest procedures in any busy urology service. Cystoscopy allows direct visualisation of the urethra, bladder neck and bladder across a broad range of indications, while Foley catheterisation is performed both as preoperative preparation and for relief of acute urinary

retention. Both pass instruments through the urethra and contact periurethral prostatic tissue, with the potential to briefly disrupt the glandular epithelium and release PSA into the circulation.[3,4]

When a clinician orders a PSA test unaware of a recent cystoscopy or catheter insertion, a falsely elevated result can trigger a cascade of unnecessary investigations biopsies, imaging, oncology referrals and considerable patient anxiety. The problem is compounded when both procedures are performed in the same sitting, as is common in elderly BPH patients admitted with acute urinary retention. Indian data on exactly how much PSA rises after these procedures, how long the rise lasts and whether the combination is additive remain scarce.[5,6] This study was therefore designed to quantify the magnitude and persistence of PSA elevation after rigid cystoscopy and Foley catheterization performed alone and together and to identify clinical factors that modify the response.

METHODS

Study design and setting

This was a prospective, hospital-based observational study carried out in the Department of General Surgery at RNT Medical College and Maharana Bhupal Government Hospital, Udaipur, Rajasthan. Ethical clearance was obtained from the Institutional Ethics Committee before enrolment and written informed consent was taken from every participant.

Study duration and sample size

Enrolment ran for one full year (January–December 2025). Sample size was calculated using an expected PSA rise of approximately 38% following cystoscopy reported by Singh et al7, with 95% confidence, 80% power and a 10% dropout allowance, yielding a minimum of 50 patients; 60 were enrolled to provide additional power for subgroup comparisons.

Inclusion and exclusion criteria

Adult male patients aged 18 years and above scheduled for diagnostic rigid cystoscopy, Foley catheterisation or both, who gave written informed consent, were eligible. Patients were excluded if they had known prostate cancer, pre-existing abnormally elevated PSA, active urinary tract infection, traumatic catheterisation, a recent prostate biopsy or transurethral resection of the prostate within the preceding six weeks or were unable to consent.

Grouping and procedure

Patients were assigned to three groups by procedure: Group 1 (n=39) diagnostic rigid cystoscopy; Group 2 (n=14) Foley urethral catheterisation alone for preoperative bladder management; and Group 3 (n=7) Foley catheterisation followed immediately by rigid cystoscopy in the same sitting, all presenting with acute urinary retention due to newly diagnosed BPH.

Prostate-specific antigen measurement

Serum total PSA was measured on three occasions for each patient: immediately before the procedure (baseline), 1 hour after and 24 hours after, using a microplate immuno-enzymometric (ELISA) method. Values were interpreted using reference cut-offs: subnormal <1.0, normal 1.0–4.0, mildly elevated 4.0–10.0 and markedly elevated >10.0 ng/ml.

Statistical analysis

Analysis used SPSS version 26. Continuous data were presented as mean±SD. PSA values across the three time points within each group were compared by paired t-test and differences across groups by one-way ANOVA with Bonferroni post-hoc correction. Categorical variables were analysed by chi-square test and the relationship between baseline PSA and PSA change by Pearson correlation. A p value below 0.05 was taken as significant.

RESULTS

Sixty male patients completed the study, aged 19–87 years (mean 48.9±18.5). Group 3 was markedly older than the other groups (72.6±5.9 vs 47.4±17.5 in group 1 and 41.4±16.5 years in group 2; ANOVA F=8.874, p<0.001), reflecting that combined catheterisation and cystoscopy is most often performed in elderly men with BPH-related retention.

In group 1, ureteric and vesicoureteric-junction calculi (30.8%) and DJ-stent procedures (25.6%) were the commonest indications; every group 3 patient presented with acute urinary retention on a background of newly diagnosed BPH.

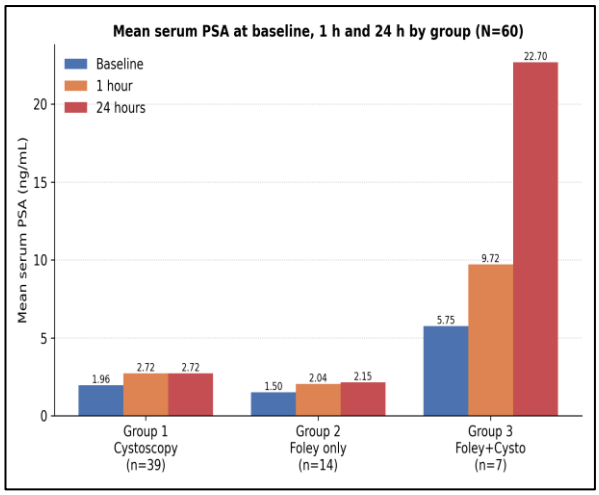

Figure 1: Mean serum PSA at baseline, 1 hour and 24 hours by group.

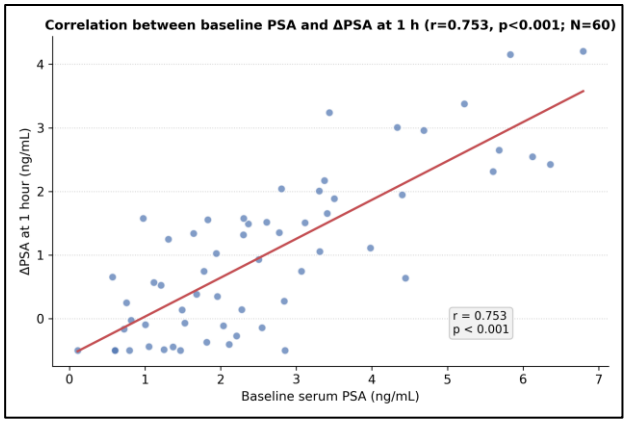

Figure 2: Correlation between baseline PSA and ΔPSA at 1 hour.

Baseline characteristics are summarised in Table 1. Diabetes mellitus was present in 15% (n=9) and hypertension in 30% (n=18). BPH on ultrasound was universal in group 3 (100%), less common in Group 1 (35.9%) and uncommon in group 2 (14.3%); prostate volumes mirrored this pattern, group 3 having the largest glands (50.7±14.4 ml). The key PSA findings are summarised in Table 2. In group 1, mean PSA rose from 1.96±1.51 to 2.72±1.81 ng/ml at 1 hour a rise of 0.76±0.82 ng/ml (38.7%; 95% CI 0.49–1.02; p<0.001) and did not fall at 24 hours (2.72±1.90 ng/ml; p<0.001). Group 2 showed a small but significant rise of 0.54 ng/ml (p=0.035). Group 3 recorded the largest increase (ΔPSA 3.97±5.17 ng/ml; 69.1%), which did not reach significance on paired testing (p=0.088), likely owing to the small group size (n=7).

On direct comparison (Table 3), one-way ANOVA showed a highly significant difference in ΔPSA at 1 hour (F=9.715; p<0.001). Bonferroni post-hoc testing localised this difference: Group 3 produced a far greater rise than group 1 (3.97 vs 0.76 ng/ml; p=0.001), while cystoscopy alone and Foley alone were statistically indistinguishable (p=1.000). This ordering held at 24 hours (F=5.251; p=0.008; group 3 vs group 1 p=0.024).

Patients with BPH on ultrasound showed a substantially greater PSA response than those without (ΔPSA 1.95±3.13 vs 0.54±0.73 ng/ml at 1 hour; p=0.011). Neither diabetes nor hypertension influenced the PSA rise, ruling them out as confounders. Baseline PSA was strongly positively correlated with post-procedural ΔPSA (Pearson r=0.753; 95% CI 0.61–0.86; p<0.001) the higher the baseline PSA, the larger the rise.

Table 1: Baseline characteristics of study participants by group (n=60).

Variables	Group 1 cystoscopy (n=39)	Group 2 foley only (n=14)	Group 3 Foley+Cysto (n=7)	P value
Mean age (in years)	47.4±17.5	41.4±16.5	72.6±5.9	<0.001
DM, n (%)	7 (17.9)	1 (7.1)	1 (14.3)	0.313
HTN, n (%)	10 (25.6)	2 (14.3)	4 (57.1)	0.098
BPH on USG, n (%)	14 (35.9)	2 (14.3)	7 (100)	<0.001
Prostate volume (ml)	37.2±21.7	29.3±14.1	50.7±14.4	0.114
Baseline PSA (ng/ml)	1.96±1.51	1.50±1.11	5.75±5.91	<0.001

Table 2: Serum PSA at baseline, 1 hour and 24 hours post-procedure with ΔPSA and statistical tests.

Statistics	Group 1 (n=39)	Group 2 (n=14)	Group 3 (n=7)	Overall (n=60)
PSA pre (ng/ml)	1.96±1.51	1.50±1.11	5.75±5.91	2.30±2.59
PSA 1 h (ng/ml)	2.72±1.81	2.04±1.80	9.72±10.60	3.37±4.32
PSA 24 h (ng/ml)	2.72±1.90	2.15±1.87	22.70±44.6	4.59±12.01
ΔPSA at 1 h (ng/m)	0.76±0.82	0.54±0.86	3.97±5.17	1.07±2.24
% rise at 1 h	38.7	36.0	69.1	—
95% CI for ΔPSA 1 h	0.49–1.02	0.04–1.04	−1.80–9.73	—
P value (paired t-test)	<0.001	0.035	0.088 (NS)	—

Table 3: Between-group comparison of ΔPSA one-way ANOVA with post-hoc bonferroni analysis.

Comparisons	Time point	F-statistic	ANOVA P value	Bonferroni P (G3 vs G1)
All three groups—ΔPSA	1 hour	9.715	<0.001	0.001
All three groups—ΔPSA	24 hours	5.251	0.008	0.024
G1 vs G2—ΔPSA	1 hour	—	—	1.000 (NS)

NS: not significant.

DISCUSSION

The findings confirm that diagnostic rigid cystoscopy raises serum PSA in a way that is both statistically significant and clinically meaningful. The mean ΔPSA of 0.76 ng/ml (38.7% rise) at one hour did not attenuate at 24 hours, making clear that a single day's wait is insufficient before measuring PSA. These results align closely with Singh et al, who reported significant rises at 1 hour ($p<0.001$) and 24 hours ($p<0.002$) after rigid cystoscopy in 50 patients of comparable age and with Oesterling et al who showed PSA after cystoscopy may be unreliable within 24–48 hours.[7,8]

The most clinically important finding concerns the combined procedure. In group 3 elderly BPH patients catheterized for retention and then cystoscoped in the same sitting the mean PSA jump was nearly five times that of the cystoscopy-alone group (3.97 vs 0.76 ng/ml; Bonferroni $p=0.001$). To our knowledge, no previous study has specifically examined this combined sequential scenario and the amplified response represents a novel observation.[9]

Foley catheterisation alone caused only a small rise (0.54 ng/mL at 1 hour; $p=0.035$) that is within the biological variability of PSA assays and unlikely to alter decisions. This agrees with Lynn et al who found prostatic manipulation had minimal effect on complexed PSA and Collins et al who reported that atraumatic urethral instrumentation did not meaningfully alter free or total PSA. Deliveliotis et al confirmed that cystoscopy and transrectal ultrasonography did not produce significant elevation in contrast to needle biopsy, while Bastani et al, found significant rises after cystoscopy, particularly with underlying urological pathology, corroborating our group 1 and group 3 findings.[10-13]

BPH patients showed a distinctly greater PSA response to instrumentation than non-BPH patients (1.95 vs 0.54 ng/ml; $p=0.011$). This is biologically logical: a larger gland contains more PSA-secreting epithelium, so a given mechanical insult releases proportionally more PSA. The strong correlation between baseline PSA and ΔPSA ($r=0.753$; $p<0.001$) reinforces this. Aliasgari et al and Bohnen et al similarly emphasized that BPH and urinary retention are among the commonest benign causes of elevated PSA, underscoring caution in interpreting post-procedural PSA in these patients.[14,15]

Reassuringly, neither diabetes nor hypertension affected the PSA response, indicating the between-group differences are driven by the type and extent of instrumentation rather than these metabolic conditions.[16] The persistence of elevation at 24 hours in all groups carries a direct practical message. DeCastro and Baker and Dew et al reported that even small elevations after flexible cystoscopy persisted through follow-up, suggesting 24 hours is insufficient for normalisation after rigid cystoscopy.[17,18]

Limitations

The combined-procedure group was small (n=7), limiting the power of within-group paired testing despite a large effect size and the wide confidence interval and high SD at 24 hours reflect this. The single-centre design and one-year window may limit generalisability and PSA was not tracked beyond 24 hours, so the exact time to normalisation could not be defined.

CONCLUSION

A single rigid cystoscopy produces a clinically significant, sustained PSA elevation that has not resolved by 24 hours, whereas Foley catheterisation alone causes only a trivial change. When both procedures are performed together as routinely happens in elderly men with BPH-related retention the resulting elevation is far greater than either alone, owing to combined mechanical disruption of an already enlarged, PSA-rich gland. A PSA measured within 24 hours of cystoscopy or on the same day as combined catheterisation and cystoscopy, should not be used to decide on prostate biopsy or oncological referral. We recommend a minimum gap of 48–72 hours after cystoscopy alone and at least one week after the combined procedure, before PSA is measured for clinical purposes.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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Cite this article as: Bishnoi D, Sharma DK, Yadav A. Effect of rigid cystoscopy and Foley urethral catheterisation on serum prostate-specific antigen levels: a prospective observational study. Int Surg J 2026;13:1689-93.