

Original Research Article

The Use of Dutasteride in Patients Undergoing Trans-Urethral Resection of Prostate

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Abstract: **Background:** Transurethral resection of the prostate (TURP) remains the gold standard surgical treatment for benign prostatic hyperplasia (BPH). However, perioperative bleeding continues to be one of the most common complications associated with the procedure. Dutasteride, a dual 5 α -reductase inhibitor, has been suggested to reduce prostatic vascularity and consequently decrease surgical bleeding. **Objective:** To study the effect of dutasteride on surgical bleeding during Transurethral Resection of the Prostate (TURP). **Patients and Methods:** A prospective study was carried out at Al-Imamein Al-Kadhimein Medical City between January 2014 and October 2015. Twenty-four patients with benign prostatic hyperplasia were enrolled and divided into two groups. Group 1 consisted of ten patients who underwent TURP alone and served as the control group. Group 2 included fourteen patients who received dutasteride 500 μ g once daily for four weeks before undergoing TURP. The two groups were compared regarding operative duration, intraoperative blood loss, postoperative blood loss, and the amount of irrigation fluid used during both the intraoperative and postoperative periods. **Results:** Patients receiving dutasteride demonstrated significantly shorter operative duration (50.21 ± 9.11 vs. 73.30 ± 5.42 min; $P < 0.0001$), significantly lower intraoperative blood loss measured by hemoglobin reduction (1.34 ± 0.55 vs. 2.48 ± 0.47 g/dL; $P < 0.0001$), and significantly lower intraoperative irrigation fluid requirements (11.29 ± 1.68 vs. 16.70 ± 2.41 L; $P < 0.0001$) compared with the TURP-alone group. However, no significant differences were observed regarding postoperative blood loss (1.27 ± 0.30 vs. 1.31 ± 0.29 g/dL; $P = 0.7550$) or postoperative irrigation fluid requirements (8.29 ± 0.87 vs. 8.60 ± 0.81 L; $P = 0.3743$). Early postoperative complications were generally comparable between the groups. No patient required blood transfusion or experienced perioperative mortality. Postoperative urinary retention occurred in 10% and 14%, dysuria in 20% and 21%, and storage urinary symptoms in 20% and 21% of patients in Groups 1 and 2, respectively. TURP syndrome, renal impairment, and stroke each occurred in one patient in the TURP-alone group, whereas none of these complications were observed in the dutasteride group. **Conclusion:** Pretreatment with dutasteride (500 μ g daily for four weeks before surgery) appears to reduce intraoperative blood loss, operative time, and intraoperative irrigation fluid requirements during TURP. This beneficial effect is likely related to reduced prostatic vascularity. However, dutasteride did not significantly reduce postoperative bleeding or postoperative irrigation requirements.

Keywords: Dutasteride, Benign Prostatic Hyperplasia, TURP, Transurethral Resection of the Prostate, Surgical Blood Loss.

INTRODUCTION

Benign prostatic hyperplasia (BPH) is one of the most common benign diseases affecting aging men and represents the principal cause of lower urinary tract symptoms (LUTS) worldwide. Histologically, BPH is characterized by hyperplasia of both stromal and epithelial cells within the transition zone of the prostate, resulting in benign prostatic enlargement (BPE), bladder outlet obstruction (BOO), and progressive urinary symptoms that significantly impair quality of life [1,2]. The prevalence of histological BPH increases steadily with age, affecting approximately 50% of men in their

sixth decade and up to 80–90% of those older than 80 years [3]. Although not a malignant condition, BPH constitutes a substantial public health burden because of its high prevalence, chronic progression, and considerable healthcare costs [2,4]. The pathophysiology of BPH is multifactorial and involves complex interactions between hormonal regulation, stromal-epithelial signaling, chronic inflammation, growth factors, and programmed cellular proliferation. Dihydrotestosterone (DHT), synthesized from testosterone by the action of the 5 α -reductase enzyme, plays a central role in prostatic growth by activating androgen receptors and stimulating cellular proliferation

[5]. In addition to androgenic stimulation, angiogenic mediators such as vascular endothelial growth factor (VEGF), hypoxia-inducible factor-1 alpha (HIF-1 α), fibroblast growth factors, and inflammatory cytokines contribute to increased vascularity and progressive enlargement of the prostate gland [6]. These molecular mechanisms have formed the basis for developing pharmacological therapies that target androgen metabolism and angiogenesis [5,6]. Treatment of BPH depends on symptom severity, prostate volume, associated complications, and patient preference. Medical therapy with α -adrenergic blockers and 5 α -reductase inhibitors is considered first-line treatment for many patients with moderate to severe symptoms [2,3]. However, surgery remains indicated in men with refractory urinary retention, recurrent urinary tract infection, recurrent gross hematuria, bladder calculi, renal insufficiency, or persistent bothersome LUTS despite optimal medical therapy [2]. Among available surgical options, transurethral resection of the prostate (TURP) continues to be regarded as the gold-standard procedure for prostates of moderate size because it provides durable symptomatic relief and significant improvement in urinary flow parameters [7]. Despite considerable advances in endoscopic instrumentation, anesthesia, and electrosurgical technologies, bleeding remains the most common complication of TURP [8]. Intraoperative hemorrhage may obscure the surgical field, prolong operative time, increase irrigation fluid absorption, and elevate the risk of capsular perforation, TUR syndrome, and blood transfusion [8,9]. Furthermore, postoperative bleeding may necessitate prolonged bladder irrigation, catheterization, clot evacuation, and extended hospitalization, thereby increasing healthcare costs and patient morbidity [8]. Patients with larger prostates, urinary tract infection, chronic catheterization, or anticoagulant therapy are particularly susceptible to excessive perioperative bleeding [2]. Dutasteride is a dual 5 α -reductase inhibitor that suppresses both type I and type II isoenzymes, producing profound inhibition of intraprostatic DHT synthesis. Compared with finasteride, dutasteride achieves greater suppression of serum and prostatic DHT concentrations and has been shown to reduce prostate volume and disease progression [5,10]. Beyond its hormonal effects, experimental studies have demonstrated that dutasteride decreases prostatic vascularity through downregulation of VEGF, HIF-1 α , adrenomedullin, and microvessel density, thereby suppressing angiogenesis within hyperplastic tissue [6,10]. These biological effects provide a strong rationale for administering dutasteride before TURP to reduce operative bleeding and improve endoscopic visualization. Several clinical studies have reported that short-term preoperative administration of dutasteride significantly reduces intraoperative blood loss, decreases hemoglobin decline, shortens operative time, and lowers irrigation fluid requirements during TURP, although its effect on postoperative bleeding remains controversial [11,12]. Because perioperative hemorrhage continues to

be an important determinant of surgical morbidity and postoperative recovery, strategies aimed at reducing prostatic vascularity remain clinically valuable. Therefore, the present prospective study was designed to evaluate the effect of four weeks of preoperative dutasteride therapy on intraoperative and postoperative bleeding parameters in patients undergoing transurethral resection of the prostate for benign prostatic hyperplasia.

Patients and Methods

This prospective comparative study was conducted at Al-Imamein Al-Kadhimein Medical City, Baghdad, Iraq, between January 2014 and October 2015. The study enrolled 24 patients diagnosed with benign prostatic enlargement having a prostate volume of less than 75 mL, as measured by pelvic ultrasonography.

Study Population

Twenty-four eligible patients were included in the study after clinical evaluation.

Exclusion Criteria

Patients were excluded if they had any of the following:

- Vesical tumor
- Vesical stone
- Renal impairment
- Previous treatment with **5-alpha reductase inhibitors (5-ARIs)**

Study Groups

The study population was divided into two groups:

Group I (Control Group):

- 10 patients underwent **Transurethral Resection of the Prostate (TURP)** alone.

Group II (Dutasteride Group):

- 14 patients received **dutasteride 500 μ g orally once daily for four weeks** before undergoing TURP.

Preoperative Evaluation

All patients underwent complete urological assessment including:

- Detailed medical history
- Drug history
- Assessment of medical comorbidities including:
 - Hypertension
 - Diabetes mellitus
 - Coronary artery disease
- Medical consultation when indicated
- Complete physical examination including **digital rectal examination (DRE)**.

Laboratory and Radiological Investigations

The following investigations were performed preoperatively:

- Urinalysis
- Hemoglobin concentration (Hb)
- Hematocrit (PCV)
- Blood grouping and cross-matching
- Serum creatinine
- Blood urea
- Random blood glucose

- Prothrombin time (PT)
- Partial thromboplastin time (PTT)
- Prostate-specific antigen (PSA)
- Electrocardiography (ECG)
- Chest radiography (when indicated)
- Pelvic ultrasonography for measurement of prostate size and post-void residual urine.

Surgical Procedure

All patients underwent monopolar transurethral resection of the prostate (TURP) using standard endoscopic techniques. The operative procedure was performed under appropriate anesthesia using continuous irrigation and electrocautery resection until adequate removal of obstructing prostatic tissue was achieved. The same operative protocol was followed for both study groups.

Outcome Measures

The primary endpoint of the study was perioperative blood loss.

The following operative variables were recorded:

- Duration of resection
- Intraoperative blood loss
- Intraoperative irrigation fluid volume
- Postoperative blood loss
- Postoperative irrigation fluid volume

- Hemoglobin reduction before and after surgery
- Postoperative complications.

Assessment of Blood Loss

Blood loss was estimated indirectly by measuring changes in hemoglobin concentration before surgery and during the postoperative period.

Two parameters were calculated:

- **First Δ Hb:** Difference between preoperative and immediate postoperative hemoglobin values, representing intraoperative blood loss.
- **Second Δ Hb:** Difference between postoperative hemoglobin measurements, representing postoperative blood loss.

Statistical Analysis

The collected data were entered into a computerized database and analyzed statistically. Continuous variables were expressed as mean values with standard deviations, whereas categorical variables were presented as frequencies and percentages. Comparisons between the two study groups were performed to determine differences in operative duration, blood loss, irrigation fluid requirements, and postoperative outcomes. A **P value <0.05** was considered statistically significant.

RESULTS

The study showed that patients in the TURP-alone group had a mean age of 68.5 ± 6.92 years and a mean prostate volume of 67.5 ± 5.84 mL. The average operative (resection) time was 73.3 ± 5.42 minutes. The mean intraoperative hemoglobin reduction (First Δ Hb), representing intraoperative blood loss, was 2.48 ± 0.47 g/dL, while the mean postoperative hemoglobin reduction (Second Δ Hb) was 1.31 ± 0.29 g/dL. The mean intraoperative irrigation fluid requirement was 16.7 ± 2.41 L, whereas the mean postoperative irrigation fluid requirement was 8.6 ± 0.81 L. These findings describe the operative characteristics of patients who underwent conventional TURP without preoperative dutasteride treatment.

Table 1. Baseline Characteristics of Patients Undergoing TURP Alone

Age (years)	Prostate size (mL)	Resection time (min)	First Δ Hb (g/dL)	Intraoperative irrigation (L)	Second Δ Hb (g/dL)	Postoperative irrigation (L)
63	57	62	1.9	12	1.1	7.5
69	60	70	2.4	14	1.4	8.0
79	63	68	2.2	15	1.8	8.0
66	67	75	1.8	16	1.0	8.0
76	68	75	2.1	19	1.7	10.0
69	70	73	2.8	17	1.2	8.0
60	70	79	3.4	18	1.5	9.0
75	72	77	2.1	19	1.3	9.5
70	73	80	2.6	18	0.9	9.0
58	75	74	3.0	19	1.2	9.0
Mean $\pm$ SD	68.5 ± 6.92	67.5 ± 5.84	73.3 ± 5.42	2.48 ± 0.47	16.7 ± 2.41	1.31 ± 0.29

The study demonstrated that patients treated with dutasteride before TURP had a mean age of 71.07 ± 5.21 years and a mean prostate volume of 66.57 ± 6.71 mL. The mean operative time was 50.21 ± 9.11 minutes, which was considerably shorter than that observed in the TURP-alone group. The mean intraoperative hemoglobin reduction was 1.34 ± 0.55 g/dL, indicating lower intraoperative blood loss. The average volume of irrigation fluid used during surgery was 11.29 ± 1.68 L. Postoperatively, the mean hemoglobin reduction was 1.27 ± 0.30 g/dL, while the mean postoperative irrigation fluid requirement was 8.29 ± 0.87 L.

Table 2. Baseline Characteristics of Patients Receiving Dutasteride Before TURP

Age (years)	Prostate size (mL)	Resection time (min)	First ΔHb (g/dL)	Intraoperative irrigation (L)	Second ΔHb (g/dL)	Postoperative irrigation (L)
64	57	42	1.0	10	1.2	8.0
69	58	40	1.0	12	1.0	8.5
67	60	45	1.3	12	1.5	9.0
75	60	42	1.3	13	1.0	9.0
71	61	44	1.3	9	1.2	7.0
66	64	43	2.3	9	1.1	7.0
78	66	43	1.1	12	1.4	8.5
73	66	50	1.5	13	1.5	9.0
75	70	48	1.1	14	1.8	10.0
69	72	63	1.2	10	1.2	7.5
73	73	55	1.8	9	1.3	7.5
80	75	60	1.4	11	0.9	8.0
62	75	65	1.0	11	1.0	8.0
73	75	63	1.5	13	1.8	9.0
Mean ± SD	71.07 ± 5.21	66.57 ± 6.71	50.21 ± 9.11	1.34 ± 0.55	11.29 ± 1.68	1.27 ± 0.30

The study showed that the mean age of patients was **68.5 ± 6.92 years** in the TURP-alone group and **71.07 ± 5.21 years** in the dutasteride-treated group. The difference was **not statistically significant (P = 0.3361)**. Likewise, the mean prostate volume was **67.5 ± 5.84 mL** in Group 1 and **66.57 ± 6.71 mL** in Group 2, with **no statistically significant difference (P = 0.7218)**. These findings indicate that both study groups were well matched regarding baseline demographic and prostate characteristics before intervention.

Table 3. Comparison of Age and Prostate Size Between Patients Undergoing TURP Alone and Those Receiving Dutasteride Before TURP

Parameter	Group 1 (n = 10) Mean ± SD	Group 2 (n = 14) Mean ± SD	P value
Age (years)	68.5 ± 6.92	71.07 ± 5.21	0.3361
Prostate size (mL)	67.5 ± 5.84	66.57 ± 6.71	0.7218

Figure 1 illustrates the comparison of the mean age between patients who underwent TURP alone and those who received preoperative dutasteride before TURP. The mean age was **68.50 years** in the TURP-alone group compared with **71.07 years** in the dutasteride group. Statistical analysis demonstrated **no significant difference** between the two groups (**P = 0.3361**), indicating that both groups were comparable regarding age and minimizing the potential influence of age as a confounding factor on the study outcomes.

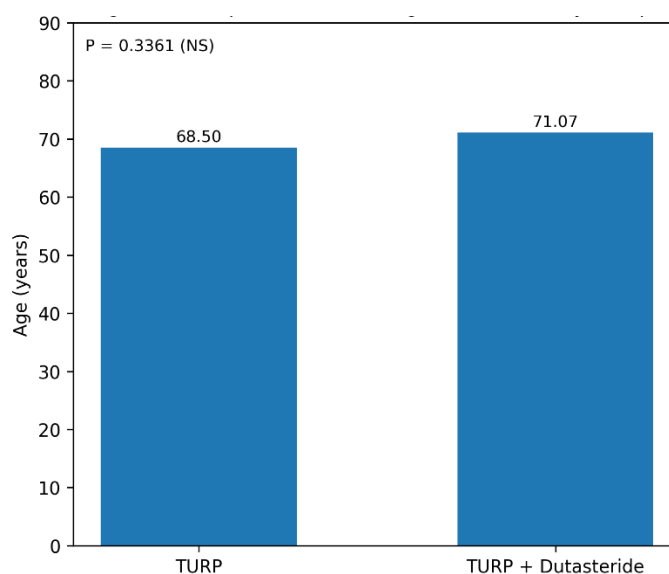


Figure 1. Comparison of Mean Age Between Patients Undergoing TURP Alone and Those Receiving Dutasteride Prior to TURP.

Figure 2 presents the comparison of the mean prostate volume between the two study groups. The mean prostate size was **67.50 mL** in patients treated with TURP alone and **66.57 mL** in patients who received dutasteride before surgery. The difference between the two groups was **not statistically significant ($P = 0.7218$)**. These findings indicate that the groups were well matched with respect to prostate size before intervention, thereby ensuring that subsequent differences in operative outcomes were unlikely to be attributable to variations in baseline prostate volume.

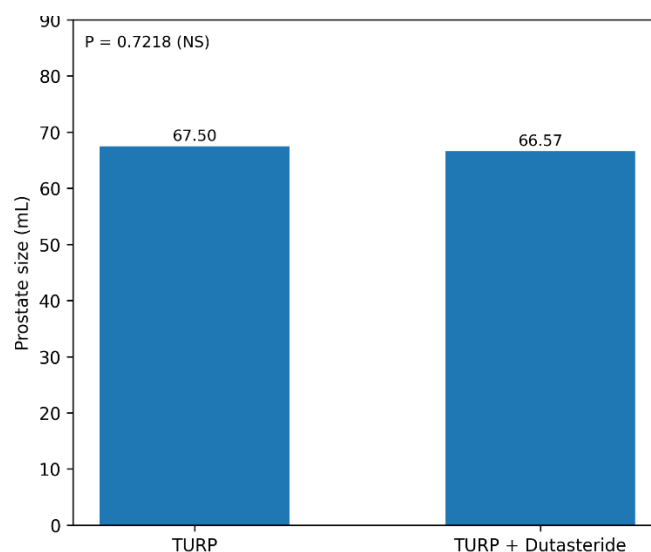


Figure 2. Comparison of Mean Prostate Size Between Patients Undergoing TURP Alone and Those Receiving Dutasteride Prior to TURP.

The study demonstrated that patients who received dutasteride for four weeks before TURP experienced significantly improved intraoperative outcomes compared with those who underwent TURP alone. The mean operative duration was significantly shorter in the dutasteride group (50.21 ± 9.11 minutes) than in the TURP-alone group (73.30 ± 5.42 minutes) ($P < 0.0001$). Likewise, the mean intraoperative hemoglobin reduction (First Δ Hb), reflecting intraoperative blood loss, was significantly lower in the dutasteride group (1.34 ± 0.55 g/dL) compared with the TURP-alone group (2.48 ± 0.47 g/dL) ($P < 0.0001$). In addition, the volume of intraoperative irrigation fluid required was significantly reduced among patients receiving dutasteride (11.29 ± 1.68 L vs. 16.70 ± 2.41 L, $P < 0.0001$).

In contrast, postoperative outcomes were comparable between the two groups. The mean postoperative hemoglobin reduction (Second Δ Hb) was 1.27 ± 0.30 g/dL in the dutasteride group and 1.31 ± 0.29 g/dL in the TURP-alone group, with no statistically significant difference ($P = 0.7550$). Similarly, the mean postoperative irrigation fluid requirement did not differ significantly between the groups (8.29 ± 0.87 L versus 8.60 ± 0.81 L, $P = 0.3743$). These findings suggest that preoperative dutasteride effectively reduced intraoperative bleeding and operative time, while it had no significant effect on postoperative bleeding or postoperative irrigation requirements.

Table 4. Comparison of Operative Parameters Between Patients Undergoing TURP Alone and Those Receiving Dutasteride Prior to TURP

Parameter	Group 1 (TURP Alone) (<i>n</i> = 10) Mean $\pm$ SD	Group 2 (Dutasteride + TURP) (<i>n</i> = 14) Mean $\pm$ SD	P value
Duration of resection (min)	73.30 $\pm$ 5.42	50.21 $\pm$ 9.11	<0.0001
Intraoperative blood loss (First Δ Hb, g/dL)	2.48 $\pm$ 0.47	1.34 $\pm$ 0.55	<0.0001
Intraoperative irrigation fluid (L)	16.70 $\pm$ 2.41	11.29 $\pm$ 1.68	<0.0001
Postoperative blood loss (Second Δ Hb, g/dL)	1.31 $\pm$ 0.29	1.27 $\pm$ 0.30	0.7550
Postoperative irrigation fluid (L)	8.60 $\pm$ 0.81	8.29 $\pm$ 0.87	0.3743

The statistical analysis demonstrated a significant positive correlation between prostate size and the duration of resection among patients in the TURP-alone group ($P = 0.002$). As prostate volume increased, a longer operative time was required to complete the transurethral resection, indicating that larger prostates were associated with more prolonged surgical procedures.

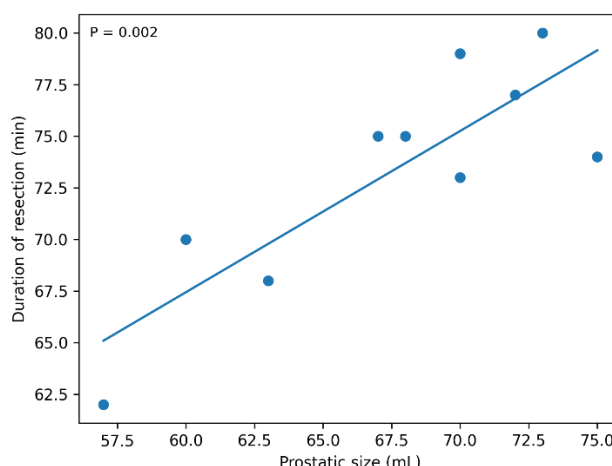


Figure 3. Correlation Between Prostatic Size and Duration of Resection in Group 1.

A **highly significant positive correlation** was also observed between prostate size and the duration of resection in patients who received preoperative dutasteride ($P < 0.001$). Despite the overall reduction in operative time associated with dutasteride treatment, increasing prostate volume remained significantly associated with longer resection time.

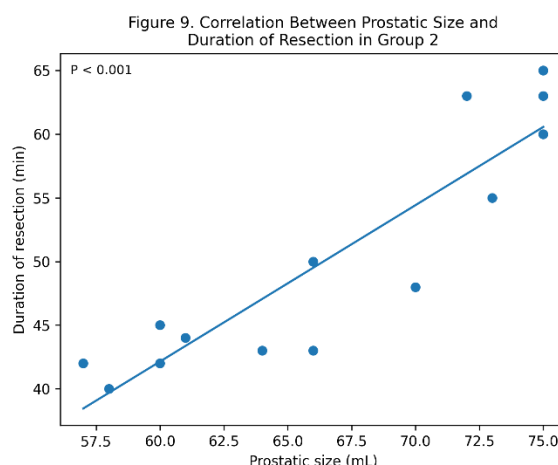


Figure 4 Correlation Between Prostatic Size and Duration of Resection in Group 2

The study demonstrated that the incidence of early postoperative complications was generally low in both groups. Following urethral catheter removal, postoperative urinary retention occurred in 1 patient (10%) in the TURP-alone group and 2 patients (14%) in the dutasteride group. Dysuria developed in 2 patients (20%) and 3 patients (21%), respectively, while storage lower urinary tract symptoms, including urinary frequency and urgency, were reported in 2 patients (20%) in Group 1 and 3 patients (21%) in Group 2. Importantly, no patient in either group required blood transfusion, indicating satisfactory perioperative hemostasis. Regarding procedure-related complications, TURP syndrome occurred in one patient (10%) in the TURP-alone group but was not observed in the dutasteride group. Similarly, renal impairment and stroke were each reported in one patient (10%) in Group 1, whereas no cases were recorded in Group 2. Furthermore, no perioperative deaths occurred in either study group. Overall, the findings suggest that preoperative dutasteride administration was not associated with an increased risk of early postoperative complications and may have contributed to the absence of TURP syndrome in the treated group.

Table 5. Early Postoperative Complications Among Patients Undergoing TURP Alone and Those Receiving Dutasteride Before TURP

Parameter	Group 1 (TURP Alone)(n = 10)	Group 2 (Dutasteride + TURP)(n = 14)
Postoperative urinary retention	1 (10%)	2 (14%)
Blood transfusion	0 (0%)	0 (0%)
Dysuria	2 (20%)	3 (21%)
Frequency and urgency	2 (20%)	3 (21%)
TURP syndrome	1 (10%)	0 (0%)
Renal impairment	1 (10%)	0 (0%)

Stroke	1 (10%)	0 (0%)
Death	0 (0%)	0 (0%)

Abbreviations: TURP, transurethral resection of the prostate

DISCUSSION

Transurethral resection of the prostate (TURP) remains the gold standard surgical treatment for symptomatic benign prostatic hyperplasia (BPH), particularly in patients with moderate-sized prostates who fail conservative or medical management. Despite continuous advances in endoscopic equipment, electrosurgical systems, irrigation techniques, and perioperative care, perioperative bleeding remains one of the most important complications associated with the procedure because it adversely affects operative visibility, prolongs surgical time, increases irrigation fluid absorption, and may ultimately increase postoperative morbidity [13–16]. Consequently, considerable interest has focused on pharmacological interventions capable of reducing prostatic vascularity before surgery, among which dutasteride has received increasing attention owing to its ability to inhibit both type I and type II 5 α -reductase enzymes and markedly suppress intraprostatic dihydrotestosterone (DHT) production [17–20]. The present study demonstrated that both study groups were well matched before intervention. The mean age was 68.5 ± 6.92 years in the TURP-alone group and 71.07 ± 5.21 years in the dutasteride-treated group, with no statistically significant difference ($P = 0.3361$). Likewise, the mean prostate volume was 67.5 ± 5.84 mL in Group 1 compared with 66.57 ± 6.71 mL in Group 2 ($P = 0.7218$). These findings indicate successful baseline matching between the two groups and minimize the possibility that subsequent differences in operative outcomes were attributable to demographic or anatomical differences rather than the therapeutic intervention. Similar baseline comparability has been reported by Kravchick *et al.*, Robert *et al.*, and Kim *et al.*, who also demonstrated no significant differences in age, prostate size, or baseline clinical characteristics between patients receiving preoperative dutasteride and untreated controls before TURP [21–25]. Another important observation in the present study was the positive correlation between prostate size and operative duration in both study groups. Statistical analysis demonstrated significant correlations in Group 1 ($P = 0.002$) and Group 2 ($P < 0.001$), indicating that increasing prostate volume was associated with longer resection time irrespective of treatment allocation. This finding is biologically expected because larger prostates contain greater amounts of hyperplastic tissue requiring resection, possess increased vascularity, and demand more extensive endoscopic manipulation before adequate decompression of the prostatic urethra can be achieved. Previous anatomical and surgical studies have similarly demonstrated that prostate volume represents an independent predictor of operative duration, blood loss, and irrigation fluid requirements during TURP [26–30]. One of the principal findings of the present study

was the significant reduction in operative time among patients pretreated with dutasteride. The mean duration of resection decreased from 73.3 ± 5.42 minutes in the TURP-alone group to 50.21 ± 9.11 minutes in the dutasteride group ($P < 0.0001$). The present findings agree with those reported by Kravchick *et al.*, who demonstrated that six weeks of dutasteride therapy before TURP significantly shortened operative duration through improved operative visibility and reduced vascularity of prostatic tissue [31]. Likewise, several investigators have suggested that suppression of prostatic angiogenesis by dutasteride facilitates more efficient tissue resection by reducing continuous venous oozing during surgery [32,33]. Perhaps the most clinically important finding of this study was the marked reduction in intraoperative blood loss. The mean intraoperative hemoglobin reduction (First Δ Hb), which was used as an indirect measure of operative blood loss, decreased from 2.48 ± 0.47 g/dL in the TURP-alone group to 1.34 ± 0.55 g/dL in the dutasteride group ($P < 0.0001$). This substantial reduction confirms that four weeks of dutasteride pretreatment significantly decreases surgical bleeding during TURP. Because intraoperative hemorrhage directly affects visualization of the operative field, reduced bleeding provides considerable technical advantages for the surgeon and may contribute to safer and faster completion of the procedure. These findings are consistent with the results reported by Moon *et al.*, who demonstrated that pretreatment with dutasteride before surgery significantly reduced operative blood loss and improved visualization during TURP [35]. Similar observations were reported by Kim *et al.*, who concluded that two weeks of dutasteride therapy before TURP significantly reduced perioperative bleeding and postoperative hemoglobin decline [36]. In contrast, Shanmugasundaram *et al.* found no statistically significant reduction in blood loss despite administering dutasteride for both preoperative and postoperative periods. Such discrepancies probably reflect differences in study design, treatment duration, sample size, surgical experience, and methods of blood loss estimation [37]. In the present study, intraoperative blood loss was assessed using the reduction in hemoglobin concentration between preoperative and immediate postoperative measurements, which remains one of the most widely accepted indirect methods for estimating blood loss during endoscopic prostate surgery. The reduction in operative bleeding observed in the present study is supported by well-established biological mechanisms. Dutasteride inhibits both isoforms of the 5 α -reductase enzyme, producing profound suppression of intraprostatic DHT concentrations. Reduced androgenic stimulation subsequently downregulates vascular endothelial growth factor (VEGF), hypoxia-inducible factor-1 alpha (HIF-1 α), fibroblast growth factors, and adrenomedullin, all of which play important

roles in angiogenesis and maintenance of prostatic microvascular density [38–41]. Consequently, suppression of these angiogenic pathways decreases the number and caliber of prostatic blood vessels, thereby reducing intraoperative hemorrhage. Experimental studies have demonstrated significant reductions in microvessel density following dutasteride administration, while Doppler ultrasonography has confirmed decreased prostatic blood flow after treatment, providing additional evidence supporting this mechanism [42–44]. The significant reduction in intraoperative blood loss observed in the present study was accompanied by a marked decrease in the amount of irrigation fluid required during surgery. The mean intraoperative irrigation fluid volume was 16.7 ± 2.41 L in the TURP-alone group compared with 11.29 ± 1.68 L in the dutasteride group, representing a highly significant reduction ($P < 0.0001$). This finding is clinically relevant because excessive irrigation is not only associated with increased operative cost but also increases the risk of fluid absorption, electrolyte disturbances, prolonged operative time, and the development of TURP syndrome. Reduced bleeding provides a clearer operative field, allowing the surgeon to identify the surgical capsule more accurately and complete tissue resection more efficiently with less interruption for hemostasis. Consequently, less irrigation fluid is required to maintain adequate visualization throughout the procedure. The present findings are consistent with those reported by Kravchick *et al.*, who demonstrated that preoperative dutasteride significantly reduced the amount of irrigation fluid required during TURP owing to improved operative visibility and decreased venous oozing from hypervascular prostatic tissue [45]. Similarly, several investigators have suggested that suppression of angiogenesis before surgery improves endoscopic visualization sufficiently to reduce both operative duration and irrigation requirements [46,47]. Conversely, Robert *et al.* found no statistically significant reduction in intraoperative irrigation fluid despite observing a tendency toward decreased bleeding in patients treated with dutasteride [48]. Differences among studies may be attributed to variations in irrigation protocols, surgeon experience, operative technique, treatment duration, and baseline prostate characteristics. Nevertheless, the highly significant reduction observed in the present study strongly suggests that the decrease in irrigation fluid was a direct consequence of improved hemostasis during resection. The relationship between intraoperative bleeding and irrigation fluid consumption is well recognized in endourological practice. As bleeding increases, visualization deteriorates, compelling the surgeon to increase irrigation flow to clear the operative field. Increased irrigation, however, prolongs surgery and increases the likelihood of systemic absorption through opened venous sinuses within the prostatic fossa. Therefore, reduction of bleeding by dutasteride creates a beneficial cascade in which improved visibility facilitates faster resection, shorter operative duration,

lower irrigation fluid consumption, and potentially fewer procedure-related complications.

Although preoperative dutasteride significantly improved intraoperative outcomes, it did not significantly influence postoperative bleeding in the present study. The mean postoperative hemoglobin reduction (Second Δ Hb) was 1.31 ± 0.29 g/dL in the TURP-alone group and 1.27 ± 0.30 g/dL in the dutasteride group ($P = 0.7550$). The absence of a significant reduction in postoperative bleeding differs from the findings of Kim *et al.*, who reported significantly lower postoperative blood loss during the first 24 hours after surgery in patients receiving dutasteride [49]. However, other investigators have similarly failed to demonstrate significant postoperative differences between treatment groups, suggesting that the postoperative benefits of dutasteride remain controversial [50]. Variations in sample size, duration of treatment, timing of postoperative hemoglobin measurements, and methods of estimating blood loss may explain these inconsistent findings. In the present study, postoperative bleeding in both groups was generally mild, with stable vital signs, unobstructed urinary catheters, and gradually clearing hematuria, reducing the likelihood of detecting clinically meaningful differences. An important observation in the present study was the occurrence of TURP syndrome in one patient (10%) who underwent TURP alone, whereas no cases were observed among patients receiving dutasteride. The affected patient subsequently developed transient renal impairment followed by cerebellar ischemia requiring intensive neurological management before gradual recovery. Although conclusions cannot be drawn from a single event, the absence of TURP syndrome in the dutasteride group may reflect the significantly shorter operative time and lower irrigation fluid requirement achieved following pretreatment. Both prolonged operative duration and excessive irrigation fluid absorption are well-established risk factors for TURP syndrome [51–54]. Consequently, interventions that reduce bleeding and shorten surgery may indirectly decrease the likelihood of this potentially life-threatening complication. Importantly, no perioperative deaths occurred in either group. This finding is consistent with contemporary TURP series demonstrating extremely low mortality rates owing to improvements in surgical techniques, anesthesia, perioperative monitoring, and patient selection [55–57].

CONCLUSION

Preoperative administration of dutasteride (500 μ g daily for four weeks) before TURP significantly improved intraoperative outcomes in patients with BPH. It reduced operative time, intraoperative blood loss, and irrigation fluid requirements, probably through decreasing prostatic vascularity and angiogenesis. However, dutasteride did not significantly affect postoperative blood loss, postoperative irrigation fluid, or early

postoperative complications. Overall, preoperative dutasteride appears to be a safe and effective adjunct that facilitates TURP without increasing postoperative morbidity.

Study Limitations

This study was limited by its small sample size, single-center design, and lack of randomization or blinding, which may affect the generalizability of the findings. Intraoperative blood loss was estimated indirectly using hemoglobin changes rather than direct measurement, and only early postoperative outcomes were evaluated. In addition, histopathological markers of angiogenesis were not assessed, and the results are limited to conventional monopolar TURP, which may not fully apply to bipolar or laser techniques.

REFERENCES

1. Mebust WK, Holtgrewe HL, Cockett AT, Peters PC. Transurethral prostatectomy: immediate and postoperative complications. Cooperative study of 13 participating institutions evaluating 3,885 patients. *J Urol.* 2002;167:999–1003.
2. Reich O, Gratzke C, Bachmann A, et al. Morbidity, mortality and early outcome of transurethral resection of the prostate: a prospective multicenter evaluation of 10,654 patients. *J Urol.* 2008;180:246–249.
3. Descaseaud A, et al. Impact of oral anticoagulation on morbidity of transurethral resection of the prostate. *World J Urol.* 2010;29:211–216.
4. Ahyai SA, Gilling P, Kaplan SA, et al. Meta-analysis of functional outcomes and complications following transurethral procedures for lower urinary tract symptoms resulting from benign prostatic enlargement. *Eur Urol.* 2010;58:384–397.
5. Kavanagh LE, Jack GS, Lawrentschuk N. Prevention and management of TURP-related hemorrhage. *Nat Rev Urol.* 2011.
6. Al-Barqawi A. Benign Prostatic Hyperplasia. University of Colorado Denver; 2012.
7. McAninch JW, Lue TF, editors. *Smith & Tanagho's General Urology.* 18th ed. New York: McGraw-Hill; 2013. p.350–354.
8. Arya M, Shergill IS, Kalsi JS, editors. *MasterPass Viva Practice for the FRCS (Urol) Examination.* Oxford: Radcliffe Publishing; 2010.
9. Wein AJ, Kavoussi LR, Novick AC, Partin AW, Peters CA, editors. *Campbell-Walsh Urology.* 10th ed. Philadelphia: Elsevier Saunders; 2012. p.2611.
10. McAninch JW, Lue TF, editors. *Smith & Tanagho's General Urology.* 18th ed. New York: McGraw-Hill; 2013. p.351–352.
11. Loughlin KR, Nimmo J. 100 Questions and Answers About Prostate Disease. Sudbury: Jones & Bartlett; 2007.
12. Wein AJ, Kavoussi LR, Novick AC, Partin AW, Peters CA, editors. *Campbell-Walsh Urology.* 10th ed. Philadelphia: Elsevier Saunders; 2012. p.2571–2572.
13. Kirby RS, McConnell JD, Fitzpatrick JM, editors. *Therapeutic Treatment for Benign Prostatic Hyperplasia.* 2nd ed. London: Taylor & Francis; 2005.
14. Feldman BJ, Feldman D. The development of androgen-independent prostate cancer. *Nat Rev Cancer.* 2001;1(1):34–45.
15. Roberts RO, Jacobson DJ, Rhodes T, Klee GG, Leiber MM, Jacobsen SJ. Serum sex hormones and measures of benign prostatic hyperplasia. *Prostate.* 2004;61(2):124–131.
16. Page ST, Lin DW, Mostaghel EA, Hess DL, True LD, Amory JK, et al. Persistent intraprostatic androgen concentrations after medical castration in healthy men. *J Clin Endocrinol Metab.* 2006;91(10):3850–3856.
17. Klotz L. 5-Reductase Inhibitors: Their Contemporary Uses and Risks. *AUA Update Series.* 2013;32:362–363.
18. Cantagrel V, Lefeber DJ, Ng BG, Guan Z, et al. SRD5A3 is required for converting polyprenol to dolichol and is mutated in a congenital glycosylation disorder. *Cell.* 2010;142(2):203–217.
19. Mori H, Maki M, Oishi K, Jaye M, Igarashi K, Yoshida O, Hatanaka M. Increased expression of genes for basic fibroblast growth factor and transforming growth factor type 2 in human benign prostatic hyperplasia. *Prostate.* 1990;16:71–79.
20. Coffey DS. The molecular biology, endocrinology, and physiology of the prostate and seminal vesicles. In: Walsh PC, Retik AB, Stamey TA, Vaughan ED Jr, editors. *Campbell's Urology.* 6th ed. Philadelphia: WB Saunders; 1992. p.221–251.
21. Wein AJ, Kavoussi LR, Partin AW, Peters CA, editors. *Campbell-Walsh Urology.* 11th ed. Philadelphia: Elsevier Saunders; 2016. p.2428–2430.
22. Partin AW, Page WF, Lee BR, et al. Concordance rates for benign prostatic disease among twins suggest hereditary influence. *Urology.* 1994;44:646–650.
23. Pearson JD, Lei HH, Beaty TH, et al. Familial aggregation of bothersome benign prostatic hyperplasia symptoms. *Urology.* 2003;61:781–785.
24. Lepor H. Evaluating men with benign prostatic hyperplasia. *Rev Urol.* 2004;6(Suppl 1):S8–S15.
25. Welch G, Weinger K, Barry MJ. Quality-of-life impact of lower urinary tract symptom severity:

- results from the Health Professionals Follow-up Study. *Urology*. 2002;59:245–250.
26. Barry MJ, Fowler FJ Jr, O'Leary MP, Bruskewitz RC, Holtgrewe HL, Mebust WK, et al. The American Urological Association symptom index for benign prostatic hyperplasia. *J Urol*. 1992;148:1549–1557.
 27. Frederick LR, Wei JT, McVary KT. Benign prostatic hyperplasia. *AUA Update Series*. 2013;32:Lesson 21.
 28. Meldrum DR, Gambone JC, Morris MA. The link between erectile dysfunction and cardiovascular health: Canary in the coal mine. *Am J Cardiol*. 2011;108(4):599–606.
 29. Wein AJ, Kavoussi LR, Novick AC, Partin AW, Peters CA, editors. *Campbell-Walsh Urology*. 10th ed. Philadelphia: Elsevier Saunders; 2012. p.2465.
 30. McConnell JD. *Benign Prostatic Hyperplasia: Diagnosis and Treatment*. Clinical Practice Guideline No. 8. Rockville (MD): Agency for Health Care Policy and Research; 1994.
 31. McConnell JD, Barry MJ, Bruskewitz RC. Benign prostatic hyperplasia: diagnosis and treatment. Clinical Practice Guideline. Rockville (MD): Agency for Health Care Policy and Research; 1994.
 32. McAninch JW, Lue TF, editors. *Smith & Tanagho's General Urology*. 18th ed. New York: McGraw-Hill; 2013. p.350–354.
 33. Reynard J, Brewster S, Biers S. *Oxford Handbook of Urology*. 3rd ed. Oxford: Oxford University Press; 2013.
 34. McNicholas TA, Speakman MJ, Kirby RS. Evaluation and nonsurgical management of benign prostatic hyperplasia. In: Wein AJ, Kavoussi LR, Partin AW, Peters CA, editors. *Campbell-Walsh Urology*. 11th ed. Philadelphia: Elsevier Saunders; 2016. p.2472–2495.
 35. Foley SJ, Bailey DM. Microvessel density in prostatic hyperplasia. *BJU Int*. 2000;85:70–73.
 36. Haggstrom S, Lissbrant IF, Damber JE. Testosterone induces vascular endothelial growth factor synthesis in the ventral prostate in castrated rats. *J Urol*. 1999;161:1620–1625.
 37. Nugroho EA, Muslim R, Riwanto I, et al. The efficacy of dutasteride and green tea combination towards angiogenesis and bleeding on benign prostatic hyperplasia after transurethral resection of the prostate: study of VEGF, microvessel density and hemoglobin. *Int J Sci Eng*. 2015.
 38. Ku JH, Shin JK, Cho MC, et al. Effect of dutasteride on the expression of hypoxia-inducible factor-1 α , vascular endothelial growth factor and microvessel density in rat and human prostate tissue. *Scand J Urol Nephrol*. 2009;43(6):457–465.
 39. Nikitenko LL, Leek R, Henderson S, Turley H, et al. Adrenomedullin and tumour angiogenesis. *Br J Cancer*. 2006;94:1–8.
 40. Mitterberger M, Pinggera G, Horninger W, Strasser H, Halpern EJ, Pallwein L, et al. Dutasteride prior to contrast-enhanced colour Doppler ultrasound prostate biopsy increases prostate cancer detection. *Eur Urol*. 2008;53:112–117.
 41. Kim TB, Oh JK, Kim KH, et al. Dutasteride: who is it more effective for? Second-to-fourth digit ratio and the relationship with prostate volume reduction by dutasteride treatment. *BJU Int*. 2012;109:E857–E863.
 42. Mehta D, editor. *British National Formulary*. 54th ed. London: BMJ Publishing Group and RPS Publishing; 2007. p.393.
 43. Welliver C, Ahmad AE, Stein BS. Prostatic gizmos and treatments for benign prostatic hyperplasia: past, present and future. *AUA Update Series*. 2015;34:Lesson 11.
 44. Han M, Partin AW. Simple prostatectomy: open and robot-assisted laparoscopic approaches. In: Wein AJ, Kavoussi LR, Partin AW, Peters CA, editors. *Campbell-Walsh Urology*. 11th ed. Philadelphia: Elsevier Saunders; 2016. p.2536–2554.
 45. Emedicine. Urology: Benign Prostatic Hyperplasia. Medscape Reference. 2003.
 46. Abrams P, Chapple C, Khoury S, Roehrborn C, de la Rosette J. Evaluation and treatment of lower urinary tract symptoms in older men. *J Urol*. 2009;181(4):1779–1787.
 47. Hohenfellner J, Stolzenburg JU, editors. *Manual of Endourology*. Heidelberg: Springer; 2005. p.87.
 48. Millan-Rodriguez F, Izquierdo-Latorre F, Montlleó-González M, Rousaud-Barón F, Rousaud-Barón A, Villavicencio-Mavrich H. Treatment of bladder stones without associated prostate surgery: results of a prospective study. *Urology*. 2005;66:505–509.
 49. Teber D, Kuntz R, Hofmann R. Complications of transurethral resection of the prostate (TURP): incidence, management, and prevention. *Eur Urol*. 2006;50(5):969–979.
 50. Loughlin KR, Nimmo J. *100 Questions and Answers About Prostate Disease*. Sudbury: Jones & Bartlett; 2007. p.47.
 51. Yao FSF, Fontes ML, Malhotra V. *Yao & Artusio's Anesthesiology: Problem-Oriented Patient Management*. 7th ed. Philadelphia: Lippincott Williams & Wilkins; 2008. p.818.
 52. Kulkarni AP, Divatia JV, Patil VP. *Objective Anesthesia Review: A Comprehensive Textbook for Examinees*. 3rd ed. New Delhi: Jaypee Brothers Medical Publishers; 2013. p.2231.
 53. Rassweiler J, Teber D, Kuntz R, Hofmann R. Complications of transurethral resection of the

- prostate (TURP): incidence, management, and prevention. *Eur Urol.* 2006;50(5):969–979.
54. Leslie SW. *Urology Board Review*. 3rd ed. New York: McGraw-Hill; 2009. Chapter 17: Prostatic Hyperplasia.
55. Lue TF, Hellstrom WJG, McAninch JW, et al. Priapism: a refined approach to diagnosis and treatment. *J Urol.* 1986;136:104–108.
56. Doll HA, Black NA, McPherson MC, et al. Mortality, morbidity and complications following transurethral resection of the prostate for benign prostatic hyperplasia. *J Urol.* 1992;147:1566–1573.
57. Oelke M, Bachmann A, Descazeaud A, et al. *EAU Guidelines on the Management of Male Lower Urinary Tract Symptoms (LUTS), including Benign Prostatic Obstruction (BPO)*. Arnhem: European Association of Urology; 2013.