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Communication

Tamsulosin Versus Terazosin for Benign Prostatic Hyperplasia: A Systematic Review

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The effectiveness and safety of tamsulosin and terazosin for patients with benign prostatic hyperplasia (BPH) was evaluated by literature review. PubMed, Embase, the Cochrane Library, Chinese biomedicine literature database (CBM), reference lists of reports, and reviews were searched for randomized controlled trials (RCTs), or quasi-RCTs of tamsulosin versus terazosin in BPH. Twelve studies involving 2,816 men were included. Outcomes included international prostate symptom score (IPSS), quality of life (QOL), maximum urinary flow rate (Q_{max}), average urinary flow rate (Q_{ave}), residual volume, prostate volume, and adverse effect (dizziness, severe hypotension, dry mouth). Relative risk was calculated for dichotomous data. Sensitivity analyses assessed the influence of baseline symptom severity. We found that tamsulosin is better than terazosin when assessed by IPSS (weighted mean difference (WMD) = −1.24 95% CI [−1.98, −0.51], there was no significant difference between the two groups in QOL (WMD = 0.04 95% CI [−0.16, 0.24]), Q_{max} (WMD = −0.38 95% CI [−1.18, 0.41]), Q_{ave} (WMD = −0.39 95% CI [−0.84, 0.06]), residual volume (WMD = −4.32 95% CI [−10.96, 2.33]), and prostate volume (WMD = −0.28 95% CI [−3.37, 2.81]). Fewer patients receiving tamsulosin experienced dizziness (relative risk (RR) = 0.38 95% CI [0.30, 0.48]), severe hypotension (RR = 0.16 95% CI [0.04, 0.68]), and dry mouth (RR = 0.14 95% CI [0.03, 0.77]), compared with patients receiving terazosin. Many of the high quality RCTs showed beneficial effects of tamsulosin in terms of improving IPSS. However, whether tamsulosin proves more efficacious than terazosin in long term therapy requires confirmation by additional large sample, high quality trials.

KEYWORDS benign prostatic hyperplasia, tamsulosin, terazosin

Abbreviations: BPH: benign prostatic hyperplasia; CBM: Chinese biomedicine literature database; RCTs: randomized controlled trials; IPSS: international prostate symptom score; QOL: quality of life; Q_{max}: maximum urinary flow rate; Q_{ave}: average urinary flow rate; WMD: weighted mean difference; CI: confidence interval; RR: relative risk; TUR: transurethral resection; LUTS: lower urinary tract symptoms.

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INTRODUCTION

Benign prostatic hyperplasia (BPH) describes the histological basis of a diagnosis of prostatic enlargement leading to a bladder outflow obstruction that presents as a lower urinary tract obstruction [Bosch et al. 1995]. Benign

growth of the prostate gland is accompanied by a significant increase in the rate of proliferation of epithelial cells in the hyperplastic acini [Barry et al. 1998]. Clinically, it is characterized by lower urinary tract symptoms (urinary frequency, urgency, a weak and intermittent stream, needing to strain, a sense of incomplete emptying, and nocturia), and can lead to complications, including acute urinary retention.

BPH is a chronic condition that increases in both incidence and prevalence with age. It is associated with progressive lower urinary tract symptoms and affects nearly three out of four men during the seventh decade of life. It was estimated that by 2006, approximately 115 million men in the 50 plus age bracket would suffer from BPH. As an age-related disease, the prevalence of BPH is likely to increase substantially during this century. Although not life-threatening, treatment costs are projected to rise to nearly 10 billion dollars and options have been presented [Ravish et al. 2007]. The symptomatic mechanisms and complications of BPH remain unclear, although obstruction of the bladder outlet is an important factor [Oishi et al. 1998]. The best documented risk factors are increasing age and functioning testes [Jacobsen et al. 1996]. Community and practice based studies suggest that men with lower urinary tract symptoms can expect a slow progression of the increased severity of symptoms [Barry et al. 1997]. However, symptoms can wax and wane without treatment. In men with symptoms of BPH, rates of acute urinary retention range from 1–2% a year [Jacobsen et al. 1997].

A greater awareness of BPH among patients and healthcare professionals is likely to increase the number of people presenting for treatment. As life-expectancy increases an effective treatment for BPH is required. The standard treatment for symptomatic BPH remains transurethral resection (TUR), but carries risks of morbidity and mortality, particularly in elderly men. Moreover, most patients with BPH reportedly choose less aggressive interventions than TUR, rather than just watchful waiting [McConnell et al. 1998]. Drug therapy represents an important alternative to TUR for patients with mild-to-moderate symptoms or for those who are unable or unwilling to undergo surgery because of the risk of both morbidity and mortality. Recently, several investigations have shown increased numbers of α_1-adrenergic receptors in hypertrophic prostatic tissue than in normal tissue [Kyprianou et al. 1996]. The use of α_1-adrenoreceptor antagonists to relax the smooth muscles of the bladder neck and prostate to decrease bladder outlet resistance and facilitate urinary flow without affecting detrusor smooth muscle contractility is a reasonably well established treatment modality for symptomatic BPH [Nordling and Hald 1997]. The high incidence of adverse reactions (particularly orthostatic hypotension) associated with the first-generation nonselective α_1/α_2-antagonists, e.g. phenoxybenzamine, often lead to cessation of treatment [Kirby et al. 1987]. Short acting α_1-selective adrenoceptor antagonists, e.g. prazosin and alfuzosin, were then introduced [Lepor et al. 1992]. The preference has been for longer acting and more uroselective α_1-adrenoceptor antagonists [Brawer et al. 1993]. Prazosin [Roehrborn et al. 1995], terazosin [Chapple et al. 1994], doxazosin [Jardin et al. 1994], and alfuzosin [Kunisawa et al. 1985], α_1-adrenoreceptor antagonists have been used to treat BPH and increase urinary outflow and lessen symptoms. However, their use is associated with adverse effects that included dizziness, severe hypotension and dry mouth. Dose titration is used to reduce these adverse effects, but approximately 10% of patients discontinue medication because of adverse reactions [Yamada et al. 1994].

Recent pharmacological studies of the α_1-adrenoreceptor have revealed three subtypes, α_{1A}, α_{1B} and α_{1D} [Lepor 1990]. The α_{1A}-adrenoreceptor subtype is present in the greatest concentration in human prostate and is responsible for prostate smooth muscle contraction [Hieble et al. 1985]. By contrast, the α_{1B}-adrenoreceptor subtype is involved in smooth muscle contraction of large human arteries [Ford et al. 1994]. Therefore, a drug with a relatively high affinity particularly for the α_{1A}-adrenoreceptor could be prostate specific. Tamsulosin is a new selective α_{1A}-adrenoreceptor subtype antagonist; it has been reported that the affinity of tamsulosin for α_{1A}-receptors is seven to 38 times greater than that for α_{1B}-receptors [Hieble et al. 1995]. Further, patients treated with tamsulosin rarely experience complications such as dizziness, severe hypotension and dry mouth. Therefore, a systematic literature review was carried out to determine the safety and efficiency of tamsulosin versus terazosin for patients with BPH.

RESULTS AND DISCUSSION

We identified 68 potentially eligible trials and subsequently excluded 56 trials. This identified 12 randomized controlled trials totaling 2,816 patients. The patient characteristic baselines were comparable as shown in Table 1. [Suzuki et al. 2001; Okada et al. 2000; Na et al. 1998; Lee and Lee 1997; Narayan et al. 2005; Tsujii 2000; Zhong et al. 2000; Hou et al. 1999; Jing 1999; Xu et al. 2000; Na et al. 1996; Lu 1998]. These 12 trials all reported adverse effects including dizziness, severe hypotension, dry mouth. Similarity of the comparator groups at baseline was ensured by stratified randomization based on age at entry and severity of disease.

Meta-Analysis

Efficacy

After 4 weeks of treatment, tamsulosin demonstrated a significant advantage when compared to terazosin in terms of IPSS (WMD = −1.24 95% CI [−1.98, −0.51]. In contrast there was no significant difference between the two groups in QOL (WMD = 0.04 95% CI [−0.16, 0.24]), Q_{max} (WMD = −0.38 95% CI [−1.18, 0.41]), Q_{ave} (WMD = −0.39 95% CI [−0.84, 0.06]), residual volume (WMD = −4.32 95% CI [−10.96, 2.33]), and prostate volume (WMD = −0.28 95% CI [−3.37, 2.81]). These results are shown in Figures 1–5.

Adverse Effects

As shown in Figure 6, fewer patients receiving tamsulosin experienced dizziness (RR = 0.38 95% CI [0.30, 0.48]), severe hypotension (RR = 0.16 95% CI [0.04, 0.68]), and dry mouth (RR = 0.14 95% CI [0.03, 0.77]), compared with patients receiving terazosin. A random effects model was used because the residual volume was heterogeneous among the trials ($P = 0.008 < 0.1$, $I^2 = 71.2\% > 50\%$). There was no significant difference between the two

TABLE 1 **Quality Assessment and Characteristics of Included Studies.**

Study	Randomization	Allocated concealment	Blinding	Drop-out (n)	Sample size	Age T/C	Grade
Narayan P 2005	Adequate	Not used	Yes	202	1993	61.7/61.7	**C**
Suzuki Y 2001	Adequate	Unclear	Not stated	0	38	65 ± 8.3	**B**
Okada H 2000	Adequate	Unclear	Yes	4	61	65.1/66.2	**B**
Na YJ 1998	Adequate	Not used	Yes	0	212	68.5/68.4	**B**
Lee E 1997	Adequate	Unclear	Yes	2	98	68.1/66.1	**B**
Tsujii T 2000	Adequate	Unclear	No	2	64	Not described	**C**
Zhong WD 2000	Adequate	Adequate	Yes	3	160	50–80	**B**
Hou SC 1999	Adequate	Adequate	Yes	3	60	50–80	**B**
Jing L 1999	Adequate	Unclear	Yes	0	70	64.5/64.5	**B**
Xu XR 2000	Adequate	Adequate	Yes	0	60	50–81	**B**
Na YQ 1996	Adequate	Adequate	Not stated	11	212	Not described	**B**
Lu GC 1998	Adequate	Not used	Yes	1	72	Not described	**C**

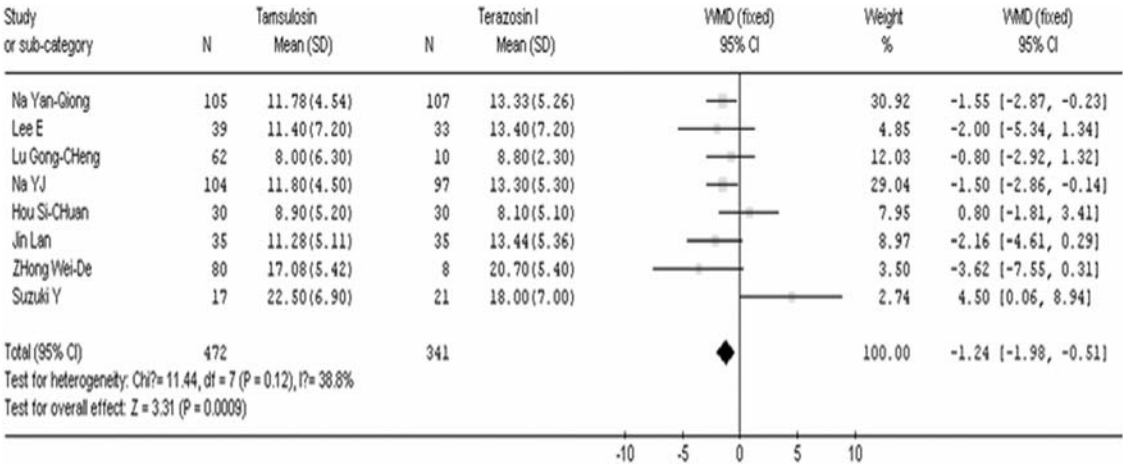

FIGURE 1 **IPSS for Tamsulosin versus Terazosin for benign prostatic hyperplasia.**

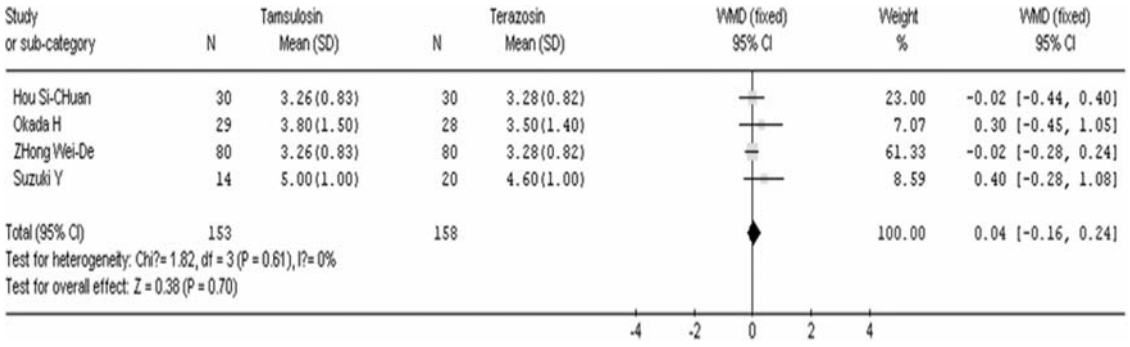

FIGURE 2 **QOL for Tamsulosin versus Terazosin for benign prostatic hyperplasia.**

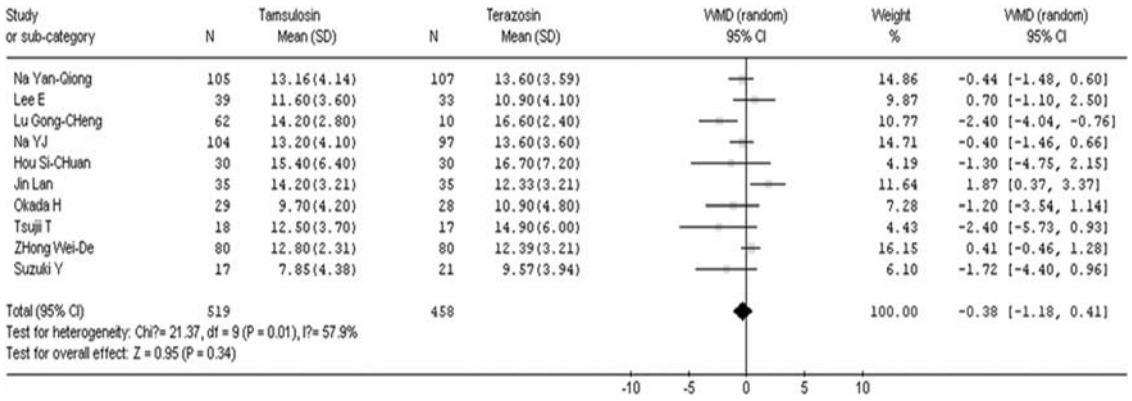

FIGURE 3 **Qmax for Tamsulosin versus Terazosin for benign prostatic hyperplasia.**

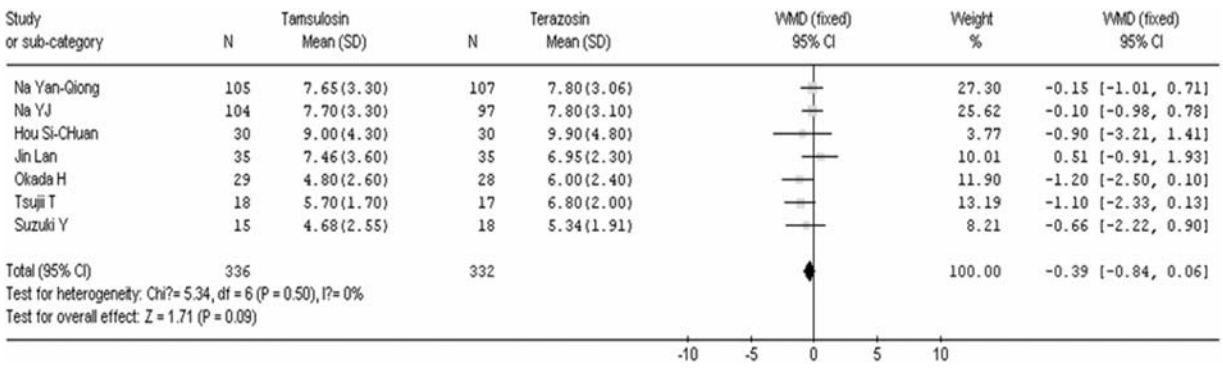

FIGURE 4 **Qave for Tamsulosin versus Terazosin for benign prostatic hyperplasia.**

groups (WMD = −4.32 95% CI [−10.96, 2.33]) as shown in Figure 7. Studies from both developing as well as developed countries. Sixty-eight prospective controlled trials related to the question of the safety and efficiency of tamsulosin versus terazosin for benign prostatic hyperplasia were identified. However, of these, only twelve randomized studies satisfied the inclusion criteria. The twelve studies varied in size of the study population from 35 to 1,983 patients, with a total of 2,816 subjects.

The studies identified in this systematic review were of varied quality. Two were multi-center randomized controlled trials. Nine of the twelve included trials were of relative higher quality ('B') and three trials were of low quality ('C').

All of the studies included adequate randomization yet only Lee and Lee [1997] referred to double blinding. Thus, high performance bias and measuring bias may be confounding factors. All of the studies indicated dropouts, yet an intention-to-treat analysis was omitted.

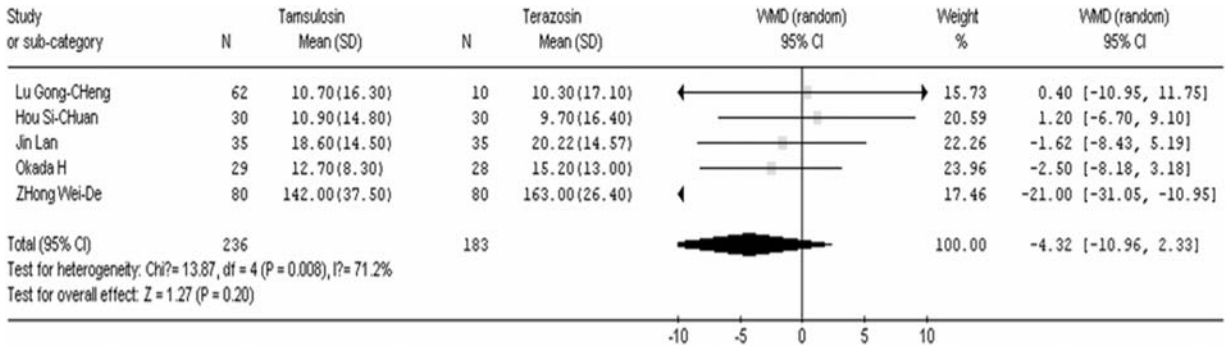

FIGURE 5 **Residual urine for Tamsulosin versus Terazosin for benign prostatic hyperplasia.**

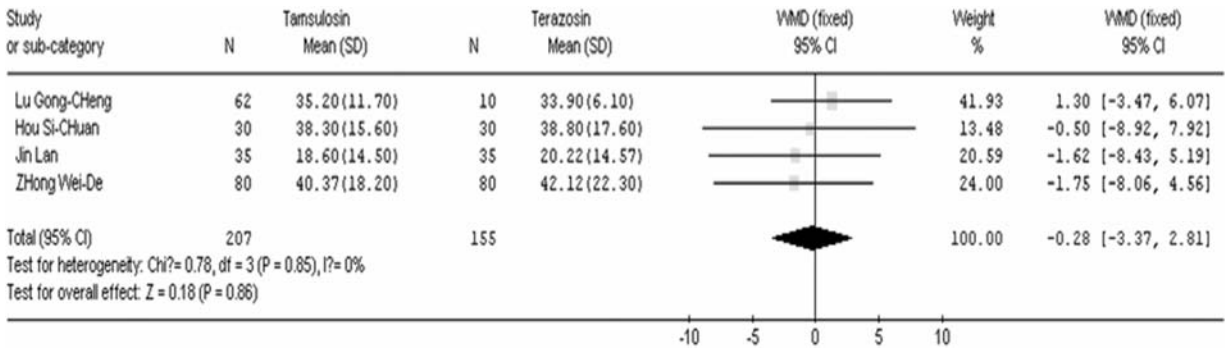

FIGURE 6 **Prostatic volume for Tamsulosin versus Terazosin for benign prostatic hyperplasia.**

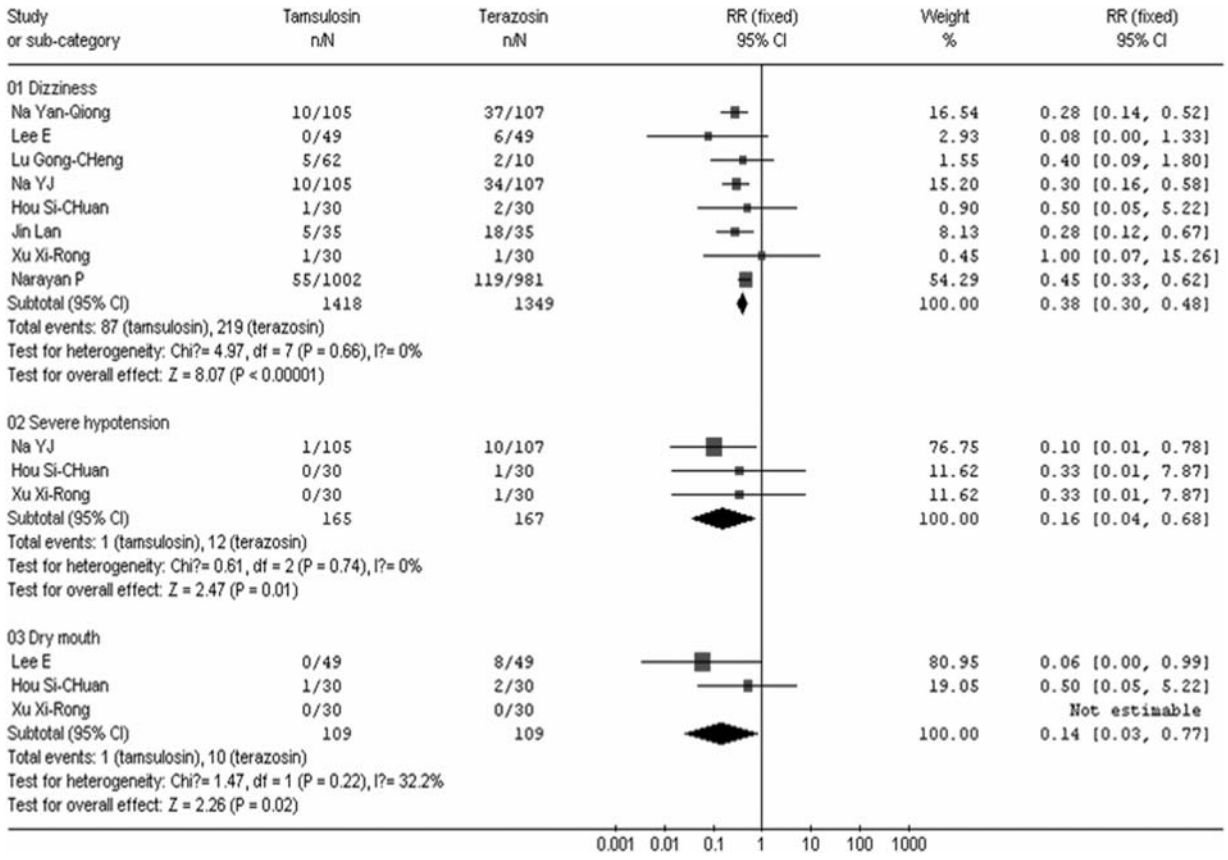

FIGURE 7 **Adverse effect for Tamsulosin versus Terazosin for benign prostatic hyperplasia.**

Of the 12 studies included, only four indicated allocated concealment. Selective bias could be introduced if the recruiter allocation assignment is modified. The dose-effect and time-effect relationship may not be clear. Together, along with the small number of participants in most studies as well as their quality, a robust conclusion may not be drawn. Further studies that consider these apparent caveats are warranted.

As α1-adrenergic receptor subtypes are also found in the human vas deferens and prostate gland, activation of these receptors also play a key role in the control of both the emission and expulsion phases of ejaculation. Theoretically, impairment results in ejaculatory dysfunction [Grasso et al. 2006]. Tamsulosin is associated with ejaculatory dysfunction, although this is possibly a central effect. In comparison the incidence of ejaculatory dysfunction or erectile dysfunction with the other α-blockers appears negligible [McVary 2005]. Future systematic review should adhere to recommendations designed to enhance the conduct, reporting and evaluation of retroactive ejaculation, and erectile function/potency of these therapeutics.

Targeting α1-adrenoceptors that maintain vascular tone, may affect the reduction of blood pressure, orthostasis, asthenia and light-headedness. The lack of need for initial dose titration of tamsulosin to limit these symptoms may be clinically advantageous compared with terazosin. Confirmation of the efficacy and dose of tamsulosin and that it is well tolerated in a more extensive large sample multi-center randomized controlled trial may show that selective α_{1A}-adrenoceptor antagonists provide suitable alternatives in α_1-adrenoceptor antagonists therapy. This alternative may be well-suited for the treatment of patients with mild to moderate symptomatic BPH, and in those awaiting surgery or unable to undergo surgery.

METHODS

Search Strategy

Pubmed (1966–2007), Embase (1974–2007), the Cochrane Library (2007 issue 4), Chinese biomedicine literature database (1978–2007), Chinese technological periodical full-text database (1989–2007.9), and Chinese periodical full-text database (1994–2007.9) were searched for randomized controlled trials comparing tamsulosin with terazosin. We manually searched key Chinese periodicals using search engines such as Google™ to search related references and references included in the studies. Hand searching of the reference lists of included studies and reviews was undertaken and contact was made with experts in the field; unpublished studies were not sought. No limits based on language were imposed.

The results of the above search strategy were independently screened by two reviewers, to confirm fulfillment of inclusion criteria and discard studies that were not applicable. Disagreements were resolved in consultation with a third expert party.

Inclusion Criteria

Inclusion criteria included randomized controlled trials with patients having mild to moderate benign prostatic hyperplasia with lower urinary tract symptoms (LUTS). Benign prostatic hyperplasia was diagnosed from the history, symptoms and physical examination where heart, liver and kidney function were normal. They could not have an indication for prostatectomy based on their clinical findings.

Exclusion Criteria

Patients with BPH who underwent prior prostatectomy, thermotherapy, anti-androgen therapy and catheterization because of urinary retention, those with other lower urinary tract disorders including prostatic cancer, neurogenic bladder, bladder stone and lower urinary tract infection, or those having cardiac, renal or hepatic insufficiency or dementia were excluded. Patients were not permitted to take other medications that could influence the outcome of the study, such as α/β-adrenoceptor agonists and antagonists, anticholinergics, antiandrogens, and steroid 5-alpha-reductase inhibitor.

Outcome Measures

Outcomes included international prostate symptom score (IPSS), quality of life (QOL), maximum urinary flow rate (Q_{max}), average urinary flow rate (Q_{ave}), residual volume, prostate volume, and adverse effect (dizziness, severe hypotension, dry mouth).

Statistical Analysis

The data was analyzed using Review Manager (version 4.2.10) and extracted and pooled data for summary estimates. The results were expressed for dichotomous outcomes (e.g., dizziness, severe hypotension, dry mouth) as relative risk (RR) with 95% confidence intervals (CIs) and continuous outcomes as weighted mean difference or standard mean difference (e.g., IPSS, QOL, Q_{max}, Q_{ave}, residual volume, prostate volume). The χ^2 statistic was used to assess heterogeneity between trials and the I^2 statistic to assess the extent of inconsistency. A fixed effect model was used to calculate summary estimates and their corresponding 95% CI. If there was significant heterogeneity, the results were confirmed using a random effects statistical model. Subgroup analyses were intended to explore important clinical differences that may be expected to alter the magnitude of treatment effect. Adverse effects were tabulated and assessed with descriptive techniques, because they were likely to be different for the various agents used.

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Declaration of Interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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