# Single Dose Tamsulosin Versus Double Dose Tamsulosin in The Initial Management Of Patients Suffering from Acute Urinary Retention Due to Benign Prostatic Hyperplasia

#### Thesis

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# List of Abbreviations

Abb.	Full term
AUR	Acute urinary retention
BOO	Bladder outlet obstruction
BPH	Benign prostatic hyperplasia
CUR	Chronic urinary retention
DHT	Dihydrotestosterone
DRE	Digital rectal examination
E2	Estradiol
FGF-7	Keratinocyte growth factor
HIFU	High intensity focused ultrasound
HoLEP	Holmium laser enucleation
HoLRP	Holmium laser resection of the prostate
IPSS	International prostate symptom score
LUTS	Lower urinary tract symptoms
PKVP	Plasma Kinetic vaporization of the prostate
PSA	Prostate specific antigen
PSAD	Prostate specific antigen density
PVR	Post-void residual urine
$TGF$ - $\beta$	Transforming growth factors
Tm:YAG	Thulium:yttrium-aluminium-garnet laser
TUIP	Transurethral incision of the prostate
TUMT	Transurethral microwave thermotherapy
TUNA	Transurethral needle ablation of the prostate
TURP	Transurethral resection of the prostate
TUVP	Transurethral vaporization of the prostate
TWOC	Trail without catheter
UR	Urinary retention
UTI	Urinary tract infection

#### Abstract

This study proved the superiority of the double dose Tamsulosin over the single dose Tamsulosin in a successful TWOC also improve the Qmax and post voiding residual urine.

Also the IPSS improved with double dose more than with single dose group as reported by the patients.

To summarize the study, it proved that it is better and more effective to use the double dose Tamsulosin as a medical treatment from the start for patients who had AUR caused by BPH without fearing the adverse effects of the drug.

Also this study proved the importance of a TWOC after medical treatment for patients who had their first attack of AUR caused by BPH as the overall success rate was 63.3%. So we have to offer this chance for every patient in order to delay the need for any operations or to avoid it.

**Keywords:** Acute urinary retention- Chronic urinary retention-- Urinary retention Estradiol- Urinary tract infection

# INTRODUCTION

enign prostatic hyperplasia (BPH) is one of the most Common urinary disorders in elderly males (Kumar and **Dewan**, 2000). The symptoms of BPH include impaired physiological and functional well-being, which interferes with daily living (Kara and Yazici, 2014).

Globally, benign prostatic hyperplasia affects about 210 million males at 2010 (6% of the population). The prevalence rate is 2.7% for men aged 45–49, it increases to 24% by the age of 80 years (Verhamme et al., 2002).

BPH may cause physical compression on the urethra and result in anatomic bladder outlet obstruction (BOO) through two distinct mechanisms: First, an increase in prostate volume, termed the static component; second, an increase in stromal smooth muscle tone, termed the dynamic component. BOO, in turn, may present clinically as lower urinary tract symptoms (LUTS), urinary tract infections, acute urinary retention (AUR), renal failure, hematuria, and bladder calculi (Stroup et al., 2012).

Notably, two factors complicate the natural history and clinical presentation of BPH, BOO and LUTS; first, prostate volume does not linearly correlate with the severity of BOO or LUTS; and second, progressive BPH and BOO can lead to primary bladder dysfunction, which in turn can exacerbate the severity of LUTS independently of BOO. Collectively, BPH,

BOO and LUTS are associated with increased risks of mortality, depression, falls and diminished health-related quality-of-life as well as with billions of US dollars in annual health expenditures (Patel and Parsons, 2014).

Early estimates of the incidence of AUR varied widely, but recent population-based studies suggested an incidence of 5-25 per 1000 person per years or 0.5-2.5% per year (Roehrborn et al., 2001).

The initial intervention for AUR is the insertion of a urinary catheter to relieve the symptoms. A trial without catheter (TWOC) carries a 23-28% success rate as reported. Nevertheless, most patients still require TURP, either as an emergency or elective surgery (Stamatiou, 2009).

Alpha-1 ( $\alpha$ 1)-blockers decrease smooth muscle tone in the prostate, thereby rapidly improving urinary symptoms and flow. Currently available  $\alpha$ 1-blockers include the selective  $\alpha$ 1-blockers terazosin, doxazosin and alfuzosin and the highly selective α1Ablocker Tamsulosin. These agents have comparable efficacy, and the major differences between these agents are their tolerability profiles (Montorsi and Moncada, 2006).

By decreasing the resistance, α1-blockers can help relieve AUR and improve the chances of successful TWOC. However, the optimum duration of therapy has not been fully assessed, and there is controversy regarding the length of time a

catheter should remain in situ during the initial therapeutic phase *(McNeill, 2001)*.

The advantage of Tamsulosin and slow-release alfuzosin over doxazosin and terazosin in the management of AUR is that a therapeutic dose can be administered at the onset of AUR, thereby reducing the time for attempting catheter removal (Altarac, 2006).

Third-generation alpha-blockers (alfuzosin, Tamsulosin) are infrequently associated with cardiovascular side effects, in contrast to their predecessors (doxazosin, terazosin, prazosin) (Kuritzky, 2005). Alpha-blocker therapy may also improve sexual functioning, with the exception of ejaculation disorders, predominantly associated with subtype selective alpha-blockers (Kuritzky, 2005).

The appropriate dose for Tamsulosin in BPH and AUR is 0.4 mg orally once daily, 30 minutes after same meal each day and maximum dose is 0.8 mg once daily. But no study done regarding the ideal dose of Tamsulosin in patients with AUR. And the most commonly reported side effects were abnormal ejaculation, influenza-like symptoms, headache, dizziness, and rhinitis (Roehrborn et al., 2000).

# AIM OF THE WORK

s to compare the efficacy and safety of single dose Tamsulosin and double dose Tamsulosin in patients suffering from acute urinary retention caused by benign prostatic hyperplasia (BPH).

### Chapter One

# PATHOPHYSIOLOGY OF BENIGN PROSTATIC HYPERPLASIA

#### Introduction

he pathophysiology of benign prostatic hyperplasia (BPH) is complex and not fully understood. The androgens like testosterones and dihydrotestosterone are of a great role as well as growth factors and other hormones like estrogens that may have a role. BPH is a process of hyperplasia of prostatic glandular-epithelial and stromal/muscle tissue, resulting in measurable growth with different configurations that can be manifested by several symptoms and complications. BPH is a histological condition which is considered one of the causes of urinary tract symptoms that is not necessarily be accompanied by prostatic enlargement or bladder outlet obstruction. Recognizing the different entities in patients may help in decision making (Roehrborn and McConnell, 2002).

Benign prostatic hyperplasia (BPH) is one of the pathological processes that may cause lower urinary tract infection in elderly. Inspite of research efforts in the past fifty years to illustrate the etiology of prostatic growth in elderly, For example, androgens are a necessary but not the only cause of BPH. Prostatism is a result of a mass-related increased resistance in urethra is too simplistic. Now obviously a significant portion of LUTS is caused by detrusor dysfunction as a part of the aging

process. Bladder outlet obstruction may result in several neural alterations in the bladder and leads to symptoms as well. Moreover, bothering LUTS includes polyuria, sleep disorders and several systemic symptoms that are not related to the prostate (Roehrborn and McConnell, 2002).

### **Etiology of BPH**

Histopathologically, BPH is characterized by periurethral increase in number of epithelial and stromal cells in the prostate. The observation of a new epithelial gland formation is normally seen only in fetal development and gives rise to the concept of embryonic reawakening of the stroma cell's inductive potential. The precise molecular etiology of this hyperplastic process is uncertain. The cellular number increase may be caused by proliferation in epithelial and stromal elements of the prostate or due to impairement in apoptotic function leading to cellular accumulation. The cause of hyperplasia is due to the actions of androgens, estrogens, stromal—epithelial interactions, growth factors and neurotransmitters (*Cunha*, 1994).

# 1. The role of androgens

Although androgens do not cause BPH, the development of BPH requires the presence of testicular androgens during prostate development, puberty and aging *(McConnell, 1995)*. Patients castrated before puberty or who are affected by a variety