

## PROSTATIC ADENOMA (BENIGN PROSTATIC HYPERPLASIA)

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**Abstract:** Benign prostatic hyperplasia (BPH), also known as prostatic adenoma, is one of the most prevalent urological conditions affecting aging men. It is characterized by nonmalignant enlargement of the prostate gland due to hyperplasia of stromal and epithelial components. This enlargement contributes to lower urinary tract symptoms (LUTS), significantly impairing quality of life. This review provides an in-depth analysis of epidemiology, pathophysiology, molecular mechanisms, clinical manifestations, diagnostic approaches, and modern management strategies, including pharmacological and surgical interventions.

### 1. Introduction

Benign prostatic hyperplasia represents a major global health burden among elderly men. Although histological changes begin as early as the fourth decade of life, clinical symptoms typically manifest later. The disease progression varies widely among individuals, making management highly individualized.

BPH is not a malignant condition; however, it may coexist with prostate cancer, which complicates diagnosis and clinical decision-making. Understanding its multifactorial nature is essential for effective treatment.

### 2. Epidemiology

BPH prevalence increases with age:

~40% in men aged 50–60 years

~70% in men aged 60–70 years

Up to 90% in men older than 80 years

Despite high histological prevalence, only a subset develops clinically significant symptoms.

Risk factors include:

Aging

Hormonal imbalance

Genetic predisposition

Metabolic syndrome

### 3. Anatomy and Histology of the Prostate

The prostate gland surrounds the proximal urethra and is divided into zones:

Peripheral zone



Central zone

Transition zone (primary site of BPH)

Histologically, the prostate consists of:

Glandular epithelium

Fibromuscular stroma

BPH arises predominantly in the transition zone, leading to urethral compression.

#### **4. Etiology and Pathogenesis**

##### **4.1 Hormonal Regulation**

Dihydrotestosterone (DHT) plays a central role:

Testosterone is converted into DHT by 5 $\alpha$ -reductase

DHT binds to androgen receptors

Stimulates cell proliferation and inhibits apoptosis

Aging alters hormonal balance:

Increased estrogen levels

Enhanced androgen receptor sensitivity

##### **4.2 Cellular Mechanisms**

BPH results from:

Increased cell proliferation

Decreased programmed cell death

Growth factors involved:

Fibroblast growth factor (FGF)

Transforming growth factor-beta (TGF- $\beta$ )

##### **4.3 Role of Inflammation**

Chronic inflammation contributes to disease progression:

Cytokine release

Tissue remodeling

Stromal expansion

#### **5. Pathophysiology**



### **5.1 Static Component**

Physical enlargement compresses urethra

### **5.2 Dynamic Component**

Increased smooth muscle tone mediated by  $\alpha$ 1-adrenergic receptors

### **5.3 Bladder Response**

Early: detrusor hypertrophy

Late: detrusor decompensation

Consequences:

Reduced urinary flow

Increased residual urine

Bladder dysfunction

## **6. Clinical Manifestations**

### **6.1 Storage Symptoms**

Urinary frequency

Urgency

Nocturia

### **6.2 Voiding Symptoms**

Weak stream

Hesitancy

Intermittency

Straining

### **6.3 Post-micturition Symptoms**

Dribbling

Sensation of incomplete emptying

### **6.4 Complications**

Acute urinary retention

Recurrent urinary tract infections

Bladder stones



Renal insufficiency

## **7. Diagnostic Evaluation**

### **7.1 Clinical Assessment**

International Prostate Symptom Score (IPSS)

Digital rectal examination (DRE)

### **7.2 Laboratory Tests**

Prostate-specific antigen (PSA)

Urinalysis

### **7.3 Imaging**

Ultrasound (transabdominal or transrectal)

Measurement of prostate volume

Post-void residual urine

### **7.4 Urodynamic Studies**

Uroflowmetry (Qmax)

Pressure-flow studies

### **7.5 Differential Diagnosis**

Prostate cancer

Urethral stricture

Neurogenic bladder

## **8. Medical Management**

### **8.1 Alpha-1 Adrenergic Blockers**

Examples:

Tamsulosin

Alfuzosin

Mechanism:

Relax smooth muscle

Advantages:

Rapid symptom relief



## **8.2 5 $\alpha$ -Reductase Inhibitors**

Examples:

Finasteride

Dutasteride

Mechanism:

Reduce DHT levels

Indications:

Enlarged prostate (>40 mL)

## **8.3 Combination Therapy**

Superior efficacy in moderate-to-severe BPH

## **8.4 Other Pharmacological Options**

Antimuscarinics

Beta-3 agonists

PDE-5 inhibitors

## **9. Surgical Management**

### **9.1 Indications**

Failure of medical therapy

Recurrent urinary retention

Renal impairment

### **9.2 Standard Procedures**

TURP

Gold standard

Effective symptom relief

HoLEP

Suitable for large prostates

Lower bleeding risk

Open Prostatectomy

Indicated for very large glands



### **9.3 Minimally Invasive Techniques**

UroLift

Rezūm therapy

Laser vaporization

### **10. Complications**

#### **10.1 Medical Therapy**

Orthostatic hypotension

Sexual dysfunction

#### **10.2 Surgical Complications**

Retrograde ejaculation

Urethral stricture

Incontinence

### **11. Prognosis and Disease Progression**

BPH progression varies:

Risk factors:

Large prostate volume

Elevated PSA

Severe LUTS

Some patients remain stable, while others experience gradual worsening.

### **12. Recent Advances**

Laser technologies (HoLEP improvements)

Robotic-assisted prostatectomy

Molecular-targeted therapies

AI-based diagnostic tools

### **13. Discussion**

BPH management requires balancing symptom relief with side effects. Modern treatment emphasizes minimally invasive approaches and patient-specific strategies.

Future research focuses on:

Genetic markers



Novel pharmacologic targets

Personalized medicine

#### **14. Conclusion**

Benign prostatic hyperplasia is a multifactorial disease with significant clinical impact. Advances in understanding its pathophysiology have improved treatment outcomes. A structured, stepwise approach ensures optimal patient care.

