Benign Prostatic Hyperplasia: Epidemiology, Pathophysiology, and Clinical Manifestations

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Abstract

The prostate secretes 20% of the seminal fluid. One of its main pathologies is benign prostatic hyperplasia (BPH), the most common benign disease in older men. It has an 8–10% prevalence in men 40 years of age and older, increasing to more than 90% in men over 90 years, with lower urinary tract symptoms being one of its main complications. Although the etiology of BPH is not still fully known, testosterone and estradiol have shown a permissive role. Likewise, other factors have emerged, such as inflammation, growth factors, and prolactin, which influence the development of BPH. These factors act through binding to specific receptors, intervening in BPH and prostate cancer development. Existing treatments significantly reduce clinical symptoms, including lower urinary tract symptoms. However, it is a nonpreventable disease; some factors can reduce its incidence: diet, physical activity, and moderate consumption of alcohol and tobacco, some of which have been proposed to have a protective role. Therefore, this chapter aims to update the preclinical and clinical evidence on the etiology of this disease, briefly describing the epidemiology, clinical manifestations, and therapeutic and preventive modalities in managing BPH.

Keywords: BPH, age, LUTS, QOL, risk factors, diagnosis, treatments, prevention

1. Introduction

The prostate is an accessory gland whose primary function is to provide 20% of seminal fluid [1, 2]. Therefore, its location is decisive for developing benign prostatic hyperplasia (BPH), the most common disease in older men. Furthermore, its prevalence increases proportionally with the increasing age of the individual [3], the main complication being lower urinary tract symptoms, which directly impacts patients’ quality of life (QOL), producing sadness and depression [4].

Although it is an age-related etiology, multiple factors intervene in its development, such as metabolic syndrome, inflammation, hormonal changes, and growth factors, which influence and regulate the development of this pathology [3, 5–7]. BPH is not a condition with a high mortality rate; its high prevalence is related to complications of severe lower urinary symptoms, including sexual dysfunction, which notoriously affects the QOL of the elderly [2]. Although it is well characterized, its etiology is poorly understood. It is known that androgens play a permissive role [7]. The imbalance between the levels of androgens and estrogens plays a decisive role in developing BPH and cancer [6]. In addition,
other factors have emerged, with growth factors responsible for cell differentiation and proliferation, playing a decisive role in the development of BPH [6]. Furthermore, some studies have suggested the participation of other nonsteroidal hormones and proinflammatory cytokines in stimulating the growth of prostate epithelial tissue, contributing to the hyperproliferative process that accompanies BPH [8, 9].

Due to this, different combined treatments are used to reduce symptoms and improve the patient’s health. Although alternative therapies have shown improvements in “in vivo” and “in vitro” models, they do not offer the same efficacy as existing treatments. In severe cases, it is necessary to accompany the pharmacological treatment with surgical procedures, which significantly improve the symptoms, although they can cause side effects [8, 10].

This disease cannot be prevented. However, it has been reported that the modulation of certain habits can decrease BPH incidence. Among these are obesity, diet, physical activity, and the consumption of alcohol and tobacco, which can directly influence the development of this etiology [4, 11].

Due to the high incidence of this etiology in the elderly, it is essential to provide updated information on this pathology. Therefore, this chapter aims to provide recent information on BPH development in older men.

2. Prostate, the energetic reproductive gland in men

The prostate is an accessory sex gland that has an approximate volume of 20–30 g, which is reached between 18 and 20 years. One of its primary functions is to contribute approximately 20% of the secretions from the seminal fluid, together with those produced by the seminal vesicles and the bulbourethral glands [1, 2].

It has a very particular ubication in the lower pelvis, below the bladder. It is related in front with the pubis and behind with the rectum. It is shaped like an inverted cone, with a base of 4 cm in transverse diameter, 2 cm in the anteroposterior direction, and 3 cm in its vertical diameters. It is crossed in its vertical axis by the urethra and, in a more horizontal and posterior plane, by the ejaculatory ducts [2].

Histologically, three well-defined prostate areas have been reported: the transition zone (5%), located near the urethra surrounding the periurethral space; the central zone (25%), where the main ducts of the seminal vesicles and the prostate are found; and the peripheral zone (70%), the widest, which is palpable during the digitorrectal examination, being the site where 90% of prostate cancers (PC) are generated [3]. The periurethral area of the transition zone consists of two separate glands that lie immediately posterior to the periprostatic sphincter. Furthermore, a characteristic that is unique to the prostate gland in humans is that it is surrounded by the prostate capsule, which is formed by a thin layer of fibromuscular tissue that continues with the stroma, limiting the growth of the gland [5].

3. Benign prostatic hyperplasia: cause or consequence of complications

BPH is a hyperplastic process that results in the growth of epithelial and stromal cells located in the periurethral area of the submucosa and transitional zone of the prostate, the leading site where BPH develops. The elongation of this area is accompanied by changes in the tissue’s stromal/muscular characteristics [5]. It has a prevalence of 26.2% worldwide, which has remained constant in the last two decades [6]. It is considered the most common benign tumor in men over 40 years of age,
representing the second cause of surgical intervention and the first of consultation with a specialist (urologist) [2].

Although it is an age-related disease, there are other associated risk factors: race, diet, obesity, metabolic syndrome, type 2 diabetes mellitus, cardiovascular diseases, alcohol consumption, urinary tract infections (UTIs), and physical inactivity [3, 5–7].

Moreover, there are hereditary factors associated with BPH. In this sense, a relative risk increase of 3.3 has been demonstrated in monozygotic vs. dizygotic twins, in addition to a higher risk of incidence in siblings with an early onset of the disease. Similarly, some specific genetic risk factors have been evidenced. For example, the loss of the Y chromosome and the presence of variants of type II 5α-reductase gene are included [6, 7].

Interestingly, it has been reported that involution of the prostate is related to an alteration of the immune system; the presence of bacteriuria increases with age in men with a sixfold increase in the number of white blood cells in the prostate secretions of those with BPH than in those without, as well as a presence of bacteria in the 36.7% of the cases [12]. The first symptoms appear in the average 40 years, a period after which growth becomes significant; some reports indicate that the annual growth can be from 40 to 90% between 40 and 50 years. Of the total number of people who develop BPH, approximately between 10 and 41% will present lower urinary tract symptoms (LUTS), which increases in severity with age, being the prostatic capsule, the structure involved in development; limiting the growth of the prostate gland causes an increase in urethral resistance and the symptoms associated with LUTS [5, 6].

Among its main symptoms is the low frequency in urinary flow, the feeling of emptying and filling, in addition to post-void symptoms and those related to voiding volume [13]. Moreover, it has been reported that the severity of discomfort caused by LUTS is related to various sexual problems. It has been reported that the lengthening of the transitional zone is associated with a decrease in sexual desire and function, the ejaculatory process, as well as incontinence (on some occasions), which causes a significant impact on the patients’ QOL [5, 6].

4. Lower urinary tract symptoms and BPH

LUTS have been reported to have a combination of voiding, filling, and post-void symptoms. All these are present in different degrees of severity. However, voiding symptoms are the most prevalent, while filling symptoms are the most annoying and interfere with patients’ QOL [2, 13].

Filling symptoms include urgency, nocturia, frequency, and urge urinary incontinence; voiding consists of weak stream, urination in drip, intermittent stream, delay, effort, and voiding drip; post-void symptoms include the sensation of incomplete voiding and post-void dribbling [2].

The prevalence of LUTS increases with age, reporting 0–20% in men aged 40–60 years; in 70-year-old men, the frequency of moderate-to-severe symptoms is three times higher than in young men [13]; over 20% of men over 60 years of age will have complications from LUTS, a situation that will exceed 40% in those over 70 years of age, which will significantly affect the QOL [7], being one of the main complications, those related to erectile dysfunction (ED), which is generally associated with severe symptoms of LUTS.

4.1 LUTS and erectile dysfunction (ED)

ED is a chronic condition manifested by the inability to achieve/maintain an erection during sexual behavior to have satisfactory sexual function. This condition
is directly related to age. Therefore, it follows a very narrow pattern with the development of BPH and the appearance of LUTS [2].

Different causes associated with ED have been described, including psychological and organic. This last includes anatomical or hormonal defects, degeneration, impaired vasodilation of the penile vessels, and in some instances, a combination of both. Therefore, people with cardiovascular or neurodegenerative diseases are more likely to develop ED [2].

Furthermore, a multicenter study in several countries, including the United States, Italy, Netherlands, Germany, Spain, and England, showed that more than 50% of men suffered from sexual dysfunction due to LUTS. Similarly, the study showed an incidence of erection problems in 49% of men aged 50–80 years, suggesting LUTS as independent risk factors for the appearance of ED [2].

5. Risk factors associated with BPH

5.1 Androgens

The role of androgens in the development of the etiology of BPH has not yet been fully elucidated, so the evidence found shows contrasting results. However, in general, it is known that the normal growth and function of the prostate gland are dependent on the presence of testicular androgens. In this sense, it is known that, in the prostate, testosterone (T) is converted to dihydrotestosterone (DHT) through the action of the enzyme 5α-reductase type II, the primary androgen in this tissue, because it has a 10-fold higher affinity for androgen receptors (AR) than T [6, 8].

Surprisingly, it has been shown that DHT levels remain elevated in the older men (90% of its production being of prostate origin and 10% of adrenal origin), while peripheral circulating levels of T decrease with age [5, 6, 8]. Therefore, the effects associated with BPH are attributed to the autocrine, paracrine, and endocrine actions of DHT rather than T [7].

The action mechanism of androgens is carried out by binding to specific AR, which is expressed mainly within the lumen of epithelial cells and in a low proportion in prostate stromal cells. Interestingly, it has been shown that, like DHT, AR is upregulated in tissue with BPH compared with normal tissues. In addition, it has been shown that a deficiency in type II 5α-reductase enzyme does not produce the elongation associated with BPH, with an effect like that observed in eunuchs or patients with hypogonadism, in which the hyperplastic change characteristic of this pathology did not occur. However, until now, there is insufficient evidence about the importance of elevated DHT and AR in the etiology of BPH [6, 12].

5.2 Estrogens

Estrogens are steroid hormones synthesized mainly in the testes through the biotransformation of T to estradiol (E2) by the action of the aromatase enzyme. Once synthesized, it travels through the circulation to exert its activity in different tissues [1, 6].

They are mainly involved in female physiology; however, in men, one of the most studied estrogenic effects is the negative feedback on the secretion of T. This effect is associated with the inhibition of the secretion of luteinizing hormone (LH) at the pituitary gland, affecting the hypothalamic-pituitary-gonad axis (HPG) in charge of stimulating testicular Leydig cells to produce T. Because of this, there will be a decrease in T levels, as well as its active metabolite, DHT [1].
These effects are mediated by binding to specific estrogen receptors (ER; ERα and ERβ), with direct implications in the pathophysiology of the prostate. Evidence shows that ERα promotes proliferation, in contrast to ERβ, which has a pro-apoptotic effect, behaving as a protective factor in BPH and PC. This effect could be due to the ER location since ERα is mainly within the prostatic stromal tissue, while ERβ is primarily at the basal epithelial cells [1, 6].

However, when estrogen exposure is excessive, it is correlated with susceptibility to both benign and malignant hyperproliferative disorders. This seems to be the result of changes in the expression pattern of steroid hormone receptors in the epithelium, which goes from being a predominantly androgen-dependent tissue to one with greater sensitivity to estrogens and, therefore, more susceptible to the development of the BPH [1, 6].

5.3 Growth factors

Growth factors (GFs) are small peptide molecules that have a central role in regulating growth, differentiation, and programmed cell death. These are released mainly by stromal cells in the prostate, acting through autocrine/paracrine communication mechanisms to regulate prostate cell homeostasis [5, 6]. Among these factors, which DHT stimulates, are transforming growth factor-beta (TGF-β), fibroblast growth factor (FGF), and epidermal growth factor (EGF); On the one hand, TGF-β is an inhibitor of epithelial cell growth, in contrast to FGF and EGF, which stimulates cell growth and differentiation [5, 6].

Interestingly, there is a close relationship between GFs and steroid hormones in the development of BPH; the activation of the AR leads to the increase of the GFs responsible for cell proliferation. Specifically, it has been shown that in AR-expressing fibroblast cells, FGF is overexpressed. Similarly, it has been reported that TGF induces the differentiation of fibroblasts into myofibroblasts in the stroma, regulating the response of epithelial cells to insulin-like growth factor-1 (IGF-1), which increases the stromal cell proliferation observed in BPH [6].

Therefore, insulin also plays a vital role in establishing BPH; the evidence shows a higher incidence in diabetic patients. This effect is related to the previously discussed IGF-1 levels [3]. Similarly, it has been reported that patients with metabolic syndrome (hypertension, dyslipidemia, glucose intolerance, obesity, insulin resistance, etc.) have higher prostate volumes than those without it; a similar situation is observed in obese patients (with a high content of fatty tissue), hypertensive, and in those with low levels of high-density lipoprotein (HDL) [7].

5.4 Prolactin

Prolactin (PRL) is a protein hormone synthesized mainly in the adenohypophysis and whose regulation is carried out by topical inhibition of dopamine. It is synthesized in different tissues, including the mammary gland, the ovary, testes, seminal glands, and the prostate. PRL participates in a wide variety of physiological processes, including the regulation of metabolism, behavior, reproduction cell growth, differentiation, and proliferation [9, 14].

The mechanism by which PRL performs its effects is by binding to a specific membrane receptor, the prolactin receptors (PRLR), which belong to the class I cytokine receptor family, and share homology with the growth hormone receptor (GHR) and the thyrotropin-releasing hormone receptor (TRHR) [14]. Under normal conditions, PRL has been shown to increase androgen production in the prostate and the conversion of T to its metabolite DHT. Similarly, “in vitro” studies have demonstrated that PRL stimulates epithelial tissue growth, an effect independent of the presence of androgens, increasing growth, division, and DNA synthesis [9].
On the other hand, some reports indicate that PRL levels increase in patients with BPH, suggesting that it can directly stimulate the development of this pathology \[9, 12\]. Specifically, it has been shown that prostate androgen receptors and PRL are increased in patients with BPH without finding a direct association with T, highlighting the relationship between PRL and BPH development \[15\].

5.5 Inflammation and BPH

Recently, evidence has emerged on the participation of inflammatory processes in BPH development. In this sense, at the preclinical level, it has been shown that proinflammatory cytokines can stimulate the growth of prostate epithelial cells. Furthermore, this process is highly associated with leukocyte infiltration observed in patients with this etiology \[8\].

Although it is not known precisely how the inflammatory process originates, the presence of bacteria (Escherichia coli) or certain viruses (human papillomavirus; herpes virus), as well as an autoimmune response, infections, hormonal changes, obesity, and metabolic syndrome, is suggested \[7, 16\].

The initial stimulus causes the activation of T lymphocytes, as well as the release of cytokines and interleukins (IL) responsible for cell damage, as well as a cascade activation of different factors, among which are the increase in the expression of IL-15 in stromal cells; IL-17 on T cells; interferon-\(\gamma\) in basal and stromal cells and IL-8 in epithelial cells; events that promote a process of chronic inflammation whose consequence is the increase in the volume of the prostate gland. Interestingly, this process can cause the appearance of reactive oxygen species (ROS) and the release of the GFs mentioned previously. Furthermore, IL-8 can induce the production of local growth factors, such as vascular endothelial growth factor (VEGF) involved in tissue angiogenesis, an effect that causes neovascularization to provide the oxygen supply to proliferating cells, which has a determining role in the pathophysiology of BPH \[3, 7, 10\].

Furthermore, the chronic inflammation observed in BPH has been associated with greater positive regulation of cyclooxygenase 2 in the glandular epithelium, causing the release of proinflammatory prostaglandins responsible for prostate cell proliferation. Similarly, inflammatory cells have been reported in tissue from patients with BPH; T lymphocytes were positive in 81% of the specimens; B lymphocytes were positive in 52%; while macrophages were positive in 82% of these specimens \[7, 16\]. Therefore, inflammatory processes are decisive in developing BPH.

6. Diagnosis and treatments

6.1 Diagnosis

Diagnosis for BPH requires at least physical examination, laboratory testing, and other testings.

**Physical examination.** The digitorrectal examination (DRE) can analyze the prostate volume, tone, firmness, and asymmetry. In addition, the ultrasound of the suprapubic region can be examined for bladder distention signs. In some cases, the penile or neurological examination is also included in search of any disorder associated with LUTS \[7\].

**Laboratory testing.** Urinalysis can identify glucosuria, pyuria, and hematuria; glucosuria assesses diabetes as a risk factor; pyuria (or bacteriuria), some infectious process; and hematuria, a complication of the genitourinary tract. Serum creatinine increase is indicative of alterations associated with kidney diseases. Prostate-specific
antigen (PSA) allows discriminating between BPH and PC. It is not generally used as a biomarker in the initial stages of BPH; however, the increase in PSA values positively correlates with prostate enlargement (greater than 30 cc), being a good predictor in those patients who require surgical treatment. In addition, it has been shown that 88% of patients with PC have histological signs of BPH, so the elevation in PSA levels could be associated with the severity of this condition [8].

**Other testing.** The International Prosthetic Symptom Score (IPSS) is a validated questionnaire to assess lower urinary tract symptoms associated with BPH. It consists of eight questions, of which seven evaluate the intensity of the symptoms, on a scale of 1–5 depending on its severity; a scale of 1–7 is indicative of mild symptomatic; 8–19 of moderate symptoms; and above 20, severe symptoms [7, 17]. These studies can be accompanied by urinary cytology and transrectal ultrasound, among others, which are beneficial noninvasive methods to discriminate the type of treatment given to the urinary patient cytology recommended for patients who have symptoms secondary to treatment and have risk factors for bladder cancer. In contrast, although not recommended in the initial stages, transrectal ultrasound is necessary before surgical treatment [8].

6.2 Treatments

Due to the high presence of androgens in BPH, the primary therapies used in its treatment are the 5α-reductase enzyme inhibitors (5ARIs), which block T’s conversion into DHT. Finasteride, and Dutasteride are the most used at the clinical level. Finasteride blocks the activity of type II 5α-reductase enzyme at the prostate stroma, while Dutasteride blocks type I at the prostate epithelium. These drugs make it possible to improve urinary flow and decrease prostate volume with a reduction of between 20 and 25% in prostate volume and 40 and 60% in PSA levels in approximately 6 months to 1 year [6, 8, 17, 18].

In the specific case of lower urinary tract complications, the treatment will depend on the severity of the symptoms. In mild cases, only follow-up is recommended. In moderate cases, the treatment of choice is alpha-adrenergic antagonists or “alpha-blockers” since these can improve and relieve LUTS and urinary flow. In contrast, in severe cases, treatment may require surgical intervention and pharmacological treatment that includes the combination of alpha-blockers, 5ARIs, antimuscarinics, and phosphodiesterase 5 inhibitors (I-PDE5) [13, 17]. Specifically, muscarinic receptor antagonists (MRAs) have been considered adequate (in combination with alpha-blockers) in patients with filling symptoms. At the same time, I-PDE5 (Sildenafil, Tadalafil, and Vardenafil) is known for its efficacy in treating ED and increasing urinary flow. Similarly, several clinical studies have shown that the combination of alpha-blockers and I-PDE5 has been more effective for ED and improved urinary tract symptoms in patients with BPH [3, 8, 18]. Furthermore, blockers are used in the case of complications caused by the alteration of the IGF-1 axis, with metformin being the effective treatment to reduce cell proliferation in BPH [3].

Although the use of phytotherapy does not have sufficient evidence to be validated, the use of alternatives (supplements) has shown efficacy in reducing prostate volume, being the “saw palmetto,” which has demonstrated antiproliferative, anti-androgenic, and anti-inflammatory activity; however, there is not enough evidence to support its use in patients with BPH [8, 18]. Similarly, the use of polyphenols and vitamin D receptor agonists has been suggested to reduce BPH symptoms associated with inflammation [3].

Finally, 20–30% of men who reach the age of 80 require surgical intervention [10]. Also, it is considered for those patients in whom pharmacological treatment has
failed or who have severe complications of LUTS. For decades, transurethral prostatic resection (TURP) and transurethral prostatic incision (TUIP) were the most common endoscopic procedures when the prostate measures less than 80 g, and prostatectomy, when the gland exceeds 80 g, effectively improving LUTS symptoms [3, 7, 8, 10].

In addition, new technological tools have emerged to treat urinary flow obstruction (blockage) caused by BPH. Among these are laser therapies; holmium or thulium laser enucleation of the prostate (HoLEP and ThuLEP) is the endoscopic procedure of choice regardless of prostate size; however, HoLEP is the gold standard procedure. Photoselective vaporization of the prostate (PVP) is a minimally invasive procedure that uses lasers to clean excess prostate tissue associated with enlarged prostate, reestablishing urinary flow, and improving BPH symptoms. Other new technologies minimize the sexual side effects. For example, prostatic urethral lift (urolift) is used to insert an implant that compresses the prostate, dilating the urethra. While the steam therapy procedure (rezum), also called water vapor thermal therapy (WVTT), uses steam injections to remove obstructive tissue without damaging the urethra. All these technologies are included in the American Urological Association (AUA) guide for treating BPH [8, 19].

7. Modulable risk factors and BPH incidence

Benign prostatic hyperplasia represents a current challenge in public health, since not only is it a nonpreventable disease, so the treatments mentioned above only arrest the growth of the prostate and reduce its clinical symptoms. However, it has been shown that modifying certain habits can improve the patient's health [11].

**Obesity.** The elongation and volume of the prostate gland correlate with body mass index (BMI) and waist circumference. In this sense, it has been shown that obese individuals (BMI greater than 35 kg/m², according to the WHO classification) have a 3.5 times higher risk of developing BPH. Similarly, there is a 2.4 greater probability of developing this etiology in those with a waist circumference greater than 109 cm. However, the mechanism, by which anthropometric measurements influence the etiology of BPH, has been suggested that the rise in the ratio of fatty tissue could be causing a greater aromatization of circulating testosterone into estrogens, whose effect on the etiology of this pathology has already been discussed. Similarly, it has been shown that people with a higher glucose intake are three times more likely to suffer from BPH, while diabetic patients have a two times higher risk, both being associated with the development of LUTS, reporting that low levels of HDL cholesterol are associated with an increase in prostate volume [4, 11].

**Diet.** Although the relationship of macro- and micronutrients with the incidence of BPH is not well defined, evidence suggests that the consumption of cereals, eggs, red meat, eicosapentaenoic and docosahexaenoic acids, butter, margarine, and starch (bread, pasta, rice) increases the risk of developing BPH, while the consumption of green peas, beans, lentils, vegetables (including tomato, garlic, and onion as a source of antioxidants), fruits (with high levels of β-carotene, lutein, or vitamin C), polyunsaturated fatty acids (including omega-3 fatty acids included in salmon and sardines) decreases risk and helps to reduce the effect of prostaglandins and leukotrienes on inflammation associated with BPH [4, 10, 20]. Concerning the micronutrients, it has been shown that the presence of high levels of vitamins D and E, zinc, lycopene, and selenium has an inverse relationship with the development of BPH, so they are suggested as protective for the PC; interestingly, high zinc and sodium levels have been related to an increase in the risk of BPH and PC [10, 11].

**Physical activity.** The increase in physical activity directly correlates with decreased BPH risk compared with those who have a sedentary lifestyle. In this
sense, it has been shown that walking 2–3 hours a week produces a 25% lower risk of LUTS and BPH evaluated with the IPSS. Moreover, a lower risk (50%) has been reported in men who exercise 3–5 times a week than those who exercise less than two times a week [4, 11].

**Alcohol and smoking.** About the consumption of alcoholic beverages, it has been shown that moderate consumption causes a 30–41% decrease in the risk of BPH and associated symptoms (40% fewer LUTS, including nocturia). However, some studies indicate that the risk of BPH increases directly with the increase in alcohol consumption. In this sense, men who consume 6–10 units of alcohol per week (mainly beer, wine, and sake) had a 41% greater probability of developing moderate-to-severe symptoms of LUTS, which strongly indicates that the appearance of these symptoms has a dose-dependent relationship. Interestingly, a similar relationship has been observed with cigarette smoking. In this context, moderate tobacco use was associated with a 30% lower probability of presenting BPH and LUTS clinical symptoms. However, in the same way, as with alcohol, smoking more than 35 cigarettes a day directly impacted BPH development and LUTS severity. However, these results are controversial [4, 10, 21].

### 8. Conclusions

BPH is the most common disease in older men. Its prevalence increases proportionally with age. The main complication is the lower urinary tract symptoms, directly impacting patients’ health. Its etiology is poorly understood, but we know that the increase in the ratio of estrogens/androgens plays a decisive role in developing BPH and cancer. In addition, growth factors, nonsteroidal hormones, and proinflammatory cytokines stimulate the growth and neovascularization of prostate epithelial tissue, contributing to the hyperproliferative process that accompanies BPH. Although it is not a preventive or curative disease, different combined treatments reduce symptoms and improve patients’ QOL. Moreover, it has been reported that the modulation of diet, obesity, physical activity, and consumption of alcohol and tobacco can improve the clinical symptoms of BPH acting as protective factors. The knowledge of this topic is essential to reduce the clinical symptoms associated with BPH in men.

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### Conflict of interest

The author declares no conflict of interest.

### Acronyms and abbreviations

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<tr>
<td>BPH</td>
<td>benign prostatic hyperplasia</td>
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<td>QOL</td>
<td>quality of life</td>
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<td>PC</td>
<td>prostate cancer</td>
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<td>UTIs</td>
<td>urinary tract infections</td>
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<td>LUTS</td>
<td>lower urinary tract symptoms</td>
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ED  erectile dysfunction
T  testosterone
DHT  dihydrotestosterone
AR  androgen receptors
E2  estradiol
LH  luteinizing hormone
HPG  hypothalamic-pituitary-gonad axis
ERα/ERβ  estrogen receptors α and β
GFs  growth factors
TGF-β  transforming growth factor-beta
FGF  fibroblast growth factor
EGF  epidermal growth factor
IGF-1  insulin-like growth factor 1
HDL  high-density lipoprotein
PRL  prolactin
PRLR  prolactin receptors
GHR  growth hormone receptors
TRHR  thyrotropin-releasing hormone receptor
IL  interleukins
ROS  reactive oxygen species
VEGF  vascular endothelial growth factor
IPSS  International Prosthetic Symptoms Score
DRE  digitorrectal examination
PSA  prostate-specific antigen
5ARIs  5α-reductase enzyme inhibitors
I-PDE5  phosphodiesterase 5 inhibitors
MRAs  muscarinic receptor antagonist
TURP  transurethral prostatic resection
TUIP  transurethral prostatic incision
HoLEP and ThuLEP  holmium or thulium laser enucleation of the prostate
PVP  photoselective vaporization of the prostate
WVTT  water vapor thermal therapy
AUA  American Urological Association
BMI  body mass index

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References


