International Journal of Clinical Urology
2025, Vol. 9, No. 2, pp. 120-129
https://doi.org/10.11648/j.ijcu.20250902.13

Review Article

Erectile Dysfunction and Benign Prostatic Enlargement: A Tale of Two Sisters

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Abstract

Erectile dysfunction (ED) refers to the persistent inability to achieve and maintain an erection sufficient for satisfactory intercourse. Lower Urinary Tract Symptoms (LUTS) are caused by benign prostatic enlargement which is a noncancerous increase in size of prostate gland. Age and LUTS are important correlates of ED in many population based studies. Epidemiological evidence provides a clear association between ED and symptomatic BPE in aging men worldwide. In the Cologne Male Survey of approximately 5000 German men aged 30 to 80 years; the prevalence of LUTS was 72% in men with ED versus 38% in those without ED. Another clinic-based population study in Western countries showed that the prevalence of ED in patients with LUTS ranged from 41%-71% and this was statistically significant ($p<0.05$). The aim of this review is to establish the pathophysiological link between ED and BPE and further emphasize on the need to look out for both conditions in a holistic manner. Current evidence suggests that several common pathogenetic mechanism are involved in the development of both ED and symptomatic BPE. These mechanism includes alteration of the nitric oxide and cyclic guanosine monophosphate pathway, enhancement of RhoA-Rho-Kinase (ROCK) signaling, autonomic hyperactivity, pelvic atherosclerosis and chronic inflammation and sex steroid ratio imbalance. Many evidenced based studies has observed a clear link between ED and BPE with predictable aetiopathogenetic mechanisms and advised that patients presenting with one of these conditions should be routinely screened for the other condition in other to ensure a holistic evaluation with appreciable improvement with quality of life (QoL). Erectile dysfunction and benign prostatic enlargement have an obvious relationship due to the common pathophysiological mechanisms. BPE may be an indicator of ED and patients should be evaluated holistically due to the high prevalence of ED in men with symptomatic BPE and positive correlation between both pathologies observed in several studies.

Keywords

Erectile Dysfunction, Benign Prostatic Enlargement, Lower Urinary Tract Symptoms, Quality of Life

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Received: 28 July 2025; **Accepted:** 7 August 2025; **Published:** 21 August 2025

1. Introduction

Erectile dysfunction (ED) is defined as persistent inability to achieve and maintain an erection sufficient for satisfactory intercourse [1]. Lower Urinary Tract Symptoms (LUTS) are caused by benign prostatic enlargement which is a noncancerous increase in size of prostate gland. Many evidenced based studies has observed a clear link between ED and BPE with predictable aetiopathogenetic mechanisms and advised that patients presenting with one of these conditions should be routinely screened for the other condition in other to ensure appreciable improvement with QoL [1, 4, 25].

Preservation of sexual function remains a vital aspect of quality of life (QoL) and needs to be considered in the management of patients with symptomatic benign prostatic enlargement (BPE) [1]. A population-based multinational study investigated the link between symptomatic BPE and sexual dysfunction in 4800 men aged 40 to 70 years. The overall prevalence of ED was 21%, which was significantly associated with increasing age [6].

Epidemiological evidence provides a clear association between ED and symptomatic BPE in aging men worldwide [3]. In the Cologne Male Survey of approximately 5000 German men aged 30 to 80 years; the prevalence of LUTS was 72% in men with ED versus 38% in those without ED [7]. This was significant ($p<0.05$) and further emphasizes on the need to consider both conditions in patient management. Another clinic-based population study in Western countries showed that the prevalence of ED in patients with LUTS ranged from 41%-71% and this was statistically significant ($p<0.05$) [2].

Age and LUTS are important correlates of ED in many population based studies [8]. Kinsey et al reported that the prevalence of ED was age related, being less than 2% until the age of 40 years, increasing to 6.7% by 55 years and 24% by 70 years [9]. Based on their findings, there may be link between ED and BPE since the latter is common with advancing age. Ngai K. H et al showed a significant negative relation between ED and BPE with statistical significance [10].

Vallancien G et al investigated the prevalence of sexual dysfunction in men with prostatic enlargement. The study revealed that the prevalence rates of ED ranged from 41% to 71% [11]. A longitudinal based study of 428 Brazilian men without ED at baseline indicated that the adjusted relative risk of developing ED is 3.67 (95% confidence interval, 1.17 to 11.48) for patients with symptomatics BPE after a mean follow-up of 2 years [12]. Seftel A. D et al, found that co-existence of ED and symptomatic BPE increased with age, ranging from 59 to 86% among men aged 40 to 60 years in Primary Health Care and 79 to 100% in treatment-seeking men with BPE aged 50 to 70 years [4]. Darab M et al studied 357 men aged 50 to 80 years presenting at urology clinic using IPSS and IIEF questionnaires to assess LUTS caused by BPE and ED respectively. The frequency of sexual attempts was inversely related to LUTS severity and sexual dysfunction was present in 68.2% of patients [13]. Gacci M et al in a community-based and clinical data demonstrated a strong and consistent association between ED and symptomatic BPE, suggesting that elderly men with LUTS should be evaluated for ED and vice versa [5]. Nakamura M et al, found that daytime frequency and incomplete emptying showed significant correlation with IIEF-5 score whereas nocturia and urethral pain being independent factors for low IIEF-5 [14].

Systemic review of epidemiological evidence undertaken in 2013 in Nigeria showed a prevalence of 43% amongst men living in Nigeria [15]. This is highly significant and further supports the need for establishing coexistence of symptomatic BPE and ED.

The aim of this review is to establish the pathophysiological link between ED and BPE and further emphasize on the need to look out for both conditions in a holistic manner.

2. Mechanism of Erection

2.1. Functional Anatomy of the Penis

The penis is composed of three cylindrical structures: the paired corpora cavernosa and the corpus spongiosum, covered by a loose subcutaneous layer and skin [16]. The tunica of the corpora cavernosa is a bilayer structure. The inner circular bundle supports and contains cavernosa tissue while the outer longitudinal layer extends from the glans penis to the proximal crura and inserts into the inferior pubic ramus [17]. It comprises of two spongy paired cylinders within a thick envelop of tunica albuginea. The corpora cavernosa is supported by fibrous skeleton including the tunica albuginea, the septum, the intracavernous pillars, the intracarvenous fibrous framework and the periarterial and perineural fibrous sheath [15]. The structure of corpus spongiosum and glans is similar to that of corpora cavernosa, except that the sinusoids are larger and the tunica thinner in spongiosum with only circular layers ensuring a lower pressure structure during erection.

The common penile artery, derived from the internal pudendal artery, branches into the dorsal, bulbourethral and cavernous arteries.

The dorsal artery provides for engorgement of the glans, while the bulbourethral artery supplies the bulb and the corpus spongiosum. The carvenous artery effects tumescence of the corpus carvenosum and thus is principally responsible for erection. The carvenous artery gives off numerous helicine arteries, which supply the trabecular erectile tissue and the sinusoids [18]. Venous return of the copora originates in the tiny venules from the sinusoids immediately beneath the tunica albuginea. The venules travel in the trabeculae between the tunica and peripheral sinusoids to form the subtunical venous plexus before exiting as the emissary veins [18].

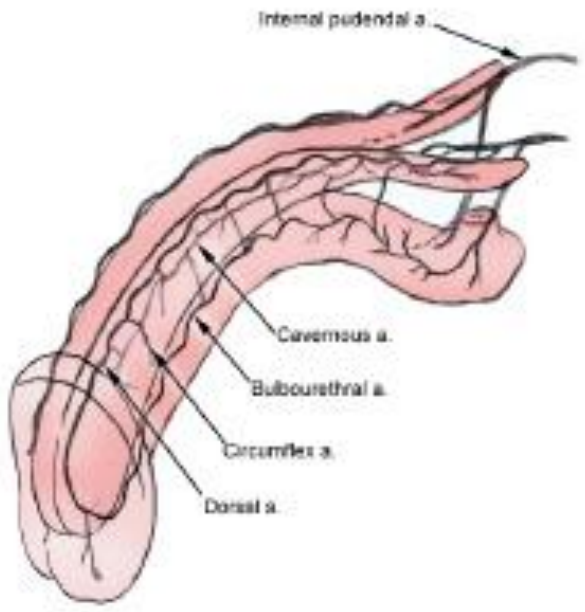

Figure 1. *Penile arterial supply.*

Adapted from Tom F Lue. Physiology of penile erection and pathophysiology of erectile dysfunction. In: Campbell-Walsh Urology, Louis R. K, Alan W. P, Andrew C. N and Craig A. P; 10th edition; 2012; 691.

2.2. Physiology of Erection

The autonomic spinal erection center is located in the intermediolateral nuclei of the spinal cord at level S2-S4 and T12-L2. Nerves fibers from thoracolumbar (sympathetic) and sacral (parasympathetic) spinal segments join to form the inferior hypogastric and pelvic plexuses. Carvenous nerve travel along posteriolateral aspect of seminal vesicle and prostate innervating both the corpora cavernosa and corpus spongiosum. Carvenous nerve also innervate the helicine arteries and trabecular smooth muscle responsible for the vascular events during tumescence and detumescence [17].

The center of somatic motor nerve located at the onuf's nucleus (S1-S4) sends motor fibers to join pudendal nerve to innervate the bulbocarvenosus and ischiocarvernosus muscle. These somatic sensory nerves originate from receptor in the penis to transmit pain, temperature, touch and vibratory sensation. The brain modulates the spinal pathway of erection via the medial pre-optic area, paraventricular nucleus of the hypothalamus, periaqueductal gray of the midbrain and nucleus paragigantocellularis of the medulla [17]. In flaccid state, the arteriolar, cavernous and arterial smooth muscles are tonically contracted due to tonic adrenergic discharge, allowing only small amount of arterial flow for nutritional purposes with a blood partial pressure of oxygen (PO_2) of about 35 mmHg. Following release of neurotransmitters (nitric oxide and nonadrenergic-noncholinergicneuroeffector system), the smooth muscles relax, arterial and arteriolar vasodilatation occurs and sinusoids expand to receive large increased flow and cause lengthening and widening of the penis. Expansion of sinusoidal walls and the tunica albuginea results in compression of subtunical venous plexus and emissary veins effectively reducing venous flow. Intracavernous pressure (ICP) rises to 100 mmHg while PO_2 rises to 90 mmHg, thus raising the penis from a dependent position to an erect state [17].

2.3. Molecular Mechanism of Erection

Penile smooth muscle contraction is regulated by Ca^{2+}. Following increase in cytosolic free Ca^{2+} to 500-700 nm, calmodulin-4 Ca^{2+} complex binds to myosin light chain kinase. Inhibition of the myosin-actin interaction ceases when the activated kinases phosphorylates the light chain initiating the contraction cycle. With the return of cytosolic Ca^{2+} to basal level, further increase in Calcium sensitivity occur due to activation of excitatory receptors coupled to G proteins. This pathway involves RhoA, a monomeric G protein. RhoA activates Rho-kinase which phosphorylates and inhibits myosin phosphatase, thus maintaining the contractile tone [17].

During sexual stimulation, nitric oxide (NO) diffuses into the trabecular cells and arterial smooth muscle to activate a second messenger guanylylcyclase catalyzing the formation of cyclic guanosinomonophospahte (cGMP). cGMP activates protein kinase G, phosphorylating potassium and calcium channels. This results in smooth muscle relaxation. Cyclic adenosine monophosphate (cAMP) is another second messenger involved in smooth muscle relaxation and is activated by signaling molecules including adenosine, calcitonin gene-related peptides and prostaglandins [17].

Detumescence occurs following degradation of cGMP and cAMP to GMP and AMP respectively by specific phosphodiesterases.

2.4. Types of Penile Erections

Three types of erection are noted in humans.

1) Genital stimulated erection (contact, tactile or reflexogenic): This is induced by tactile stimulation of the genital area and preserved in upper spinal cord lesions. Genital stimulated erection is short and poorly controlled by the individual.
2) Centrally stimulated erection: It is more complex and result from memory, fantasy, visual or auditory stimulation.
3) Centrally originated erection: This can occur spontaneously without stimulation or during sleep. It occurs mostly during rapid eye movement (REM) sleep. During REM sleep, cholinergic neurons in the lateral pontine tegmentum are activated while the adrenergic neurons in the locus ceruleus and the serotonergic neurons in the midbrain raphe are silent [16, 19].

2.5. Phases of Erection

Activation of the autonomic nerves produces full erection secondary to filling and trapping of blood in the carvenous bodies. Contraction of ischiocavernosus muscle compresses the

proximal corpora, raising the intracorporal pressure well above the systolic blood pressure, resulting in rigid erection [17].

Erection process can be divided into phases.

1) Flaccid phase: In flaccid phase, there is minimal arterial and venous flow. Blood gas values are the same with venous blood.
2) Latent (filling) phase: it is characterized by increased flow in the internal pudendal artery, decreased pressure in the internal pudendal artery and unchanged intracarvenous pressure. Some elongation occurs in this phase.
3) Tumescent phase: There is rising intracarvenous pressure until full erection is achieved. Penis shows more elongation and expansion. The arterial flow rate decreases as the pressure rises. With the intracarvenous pressure rising above diastolic pressure, flow occurs only in the systolic phases.
4) Full erection phase: The pressure in the internal pudendal artery increases but remains slightly below the systolic blood pressure. Arterial flow is much less than the initial flow. Although the venous channel are mostly compressed, the venous flow rate is slightly higher than during the flaccid phase and blood gas values approach those of the arterial phase.
5) Skeletal or rigid erection phase: The intracarvenous pressure rises above the systolic blood pressure, resulting in rigid erection. This is because of contraction of the ischiocavernous muscle. During this phase no blood flows through the cavernous artery, but the short duration prevents ischemia or tissue damage.
6) Detumescent phase: Following ejaculation or cessation of erotic stimuli, sympathetic discharge resumes and results in contraction of the smooth muscle of the sinusoids and arterioles. The arterial flow is diminished to flaccid level, large portion of blood expelled from the sinusoidal space and venous channels are re-opened. The penis thus returns to its flaccid state.

2.6. Classification and Pathogenesis of Erectile Dysfunction

The classification system of ED most commonly used comprises organic, psychogenic and mixed etiologies of ED and endorsed by the International Society of Impotence Research.

2.6.1. Organic Erectile Dysfunction

This consists of neurogenic, hormonal, arterial, cavernosal (venogenic) and drug related including antiandrogens used in the treatment of benign prostatic enlargement.

Neurogenic ED results from peripheral (cavernous and pudendal nerve) or central pathologies (spinal cord injury, dementia, stroke, brain tumors and Shy-Drager syndrome). Iatrogenic trauma from prostatectomy and abdominal perineal resection can result in ED from direct injury to cavernous or pudendal nerves [17].

Hormonal disorder including hypogonadism due to hypothalamic or pituitary tumor, estrogen or androgen therapy or orchidectomy can suppress sexual drive and nocturnal erection. Veronelli et al noted that hyperprolactinemia, Cushing's syndrome, hyperthyroidism, hypothyroidism and Addison's disease can suppress libido and result in ED due to hormonal fluctuations [20].

Arteriogenic ED may be congenital or due to trauma. Traumatic occlusive disease involving the hypogastric, cavernous or helicine arteries can decrease flow to sinusoidal spaces thus decreasing rigidity or prolonging time to maximal erection.

In cavernous veno-occlusive dysfunction, degenerative changes and traumatic injury to the tunica albuginea can impair the compression of sub-tunical and emissary veins. Deveci et al found that fibroelastic alteration of the endothelium can cause venous leakage [21].

Medication-induced erectile dysfunction includes drugs that interfere with central neuroendocrine and local neurovascular control of penile smooth muscle are viable causes of ED. Antipsychotic, centrally acting antihypertensives and antidepressants can affect the serotonergic, noradrenagic and dopaminergic pathways of erection [22, 23]. Beta-adrenergic blocking drugs may potentiate alpha-1 adrenergic activity in the penis. Alpha-adrenergic blocking drugs such as doxazosin and tamsulosin may cause retrograde ejaculation due to relaxation of bladder neck [24]. Antiandrogens modulates sexual desire via central nervous system androgen receptors.

2.6.2. Psychogenic Erectile Dysfunction

Performance anxiety, strained relationship, depression and schizophrenia can either cause or aggravate ED. Possible mechanism may be due to an imbalance of central neurotransmitters, over inhibition of spinal erection center in the brain.

2.6.3. Mixed Erectile Dysfunction

This is the most common type of ED and it is a combination of organic and psychogenic ED.

2.7. Assessment of Erectile Dysfunction

The most commonly used validated questionnaires are the 15-item International Index of Erectile Function (IIEF). This has been simplified to a 5-item version (IIEF-5), which is more suited for office use. Sexual function measured by IIEF-5 includes erectile function, orgasmic function, sexual desire, intercourse satisfaction and overall satisfaction. They determine baseline erectile function and assess the impact of a specific treatment modality.

ED severity is classified as severe (5-7), moderate (8-11), mild to moderate (12-16), mild (17-21), and no ED (22-25) [25].

2.8. Anatomy of the Prostate

The prostate is a male organ that resides in the pelvis posterior to the pubic symphysis. It is separated from the pubic

symphysis by retropubic space of Retzius. Denonvilliers' fascia separates the posterior surface from the rectum. The base is continuous with the bladder neck and the apex rests on the upper surface of the urogenital diaphragm [26].

The adult prostate weighs between 18 and 25 g and several formulae can be used to deduce the size of the prostate. McNeal introduced the concept of zonal anatomy of the prostate viz: the peripheral zone, central zone and transitional zone. BPE originates from the transitional zone.

The arterial supply is from inferior vesical and middle rectal arteries which are branches of internal iliac artery. Venous drainage is from dorsal venous complex which receives deep dorsal vein and vesical branches and subsequently drains into the internal iliac vein.

2.9. Pathophysiology of Benign Prostatic Enlargement

Lower urinary tract symptoms result from bladder outlet obstruction (BOO) most commonly due to prostatic enlargement, urethral injury/stricture and calculi in the lower urinary tract etc. These pathologies can cause LUTS either by obstructing urine flow or due to secondary response of the bladder to the outlet resistance [27]. The obstructive component of LUTS due to BPE can be subdivided into mechanical or dynamic obstruction. The mechanical obstruction from prostatic enlargement results from intrusion into the urethral lumen or bladder neck leading to a higher bladder outlet resistance.

The dynamic component explains the variable nature of the symptoms. The prostate is composed of smooth muscle, collagen and is also rich in adrenergic nerve supply. The level of autonomic stimulation sets a tone to the prostatic urethra and can be decreased by alpha-blocker resulting in a decrease in outlet resistance [17].

Secondary response of the bladder to the increased outlet resistance produces the storage symptoms. BOO leads to detrusor muscle hypertrophy and instability due to increased detrusor collagen seen as thickening of the bladder wall. The thickened detrusor muscle bundles and coursing of the muscle strands are seen as trabeculation with widening of gaps in between them to form saccules. Mucosa herniation between detrusor muscle bundles ensues causing diverticular formation [28].

The increased muscle mass and intravesical pressure are associated with changes in the intracellular and extracellular parts of smooth muscle cells. This leads to detrusor instability manifesting with storage symptoms.

The terminal ureters become kinked as they traverse the thickened bladder with failure of ureterovesical valves resulting in vesico-ureteric reflux, bilateral hydroureters and hydronephrosis. The increased intrapelvic pressure from hydronephrosis destroys the renal papillae, nephrons and parenchyma leading to impaired renal function resulting in decreased fluid, solutes and electrolyte handling resulting in dehydration, edema, acidosis and acute/chronic renal failure [28].

2.10. International Prostate Symptoms Score (IPSS)

This is a self-administered questionnaire developed by American Urological Association (AUA). It is a valid and reliable tool for assessing the severity of LUTS associated with BPE, determining treatment and monitoring response to therapy. It has 4 voiding and 3 storage scoring items. The voiding symptoms of IPSS are incomplete emptying, intermittency, weak stream and straining. The storage symptoms of IPSS are frequency, urgency and nocturia. This assessment focuses on the 7 items that asks patients to quantify the severity of their voiding or storage complaints on a scale of 0-5 [28]. The score ranges from 0-35. A total symptom score of 0-7 is mild, 8-19 is moderate and 20-35 is severe.

Quality of life due to urinary symptoms is assessed using a single question. The answers range from 'delighted' to 'pleased', 'mostly satisfied', 'mixed (both equally satisfied and dissatisfied', 'mostly dissatisfied', 'unhappy', 'terrible'. It is scored from 0 to 6. IPSS correlates well with the QOL, but does not capture the complete picture of the prostate size as perceived by individual patients [29].

3. Transrectal Ultrasound of Prostate

3.1. Advantages of TRUS

1) Allows better delineation of prostatic anatomy and prostate volume compared to transabdominal and transperineal ultrasound [29].
2) Detects and allows targeted biopsy of suspicious lesions compared to transurethral ultrasound and will not predispose patients to urethral injury.
3) Used in delivering treatment such as brachytherapy and in high intensity focused ultrasound. [29]
4) Detects other pathologies such as ejaculatory duct cyst and prostatic calculi.

3.2. Disadvantages of TRUS

1) Causes a little discomfort and pain to patients.
2) May underestimate the prostate volume.
3) It is non-specific for prostate cancer detection and is inaccurate in evaluation of capsular penetration [29].

3.3. Technique of TRUS

Transrectal ultrasound is the most common imaging modality for prostate. Modern endorectal probes are available in both side and end-fire models and the transmitted frequency varies from 6 to 10 MHz. Volume estimation is obtained by scanning the prostate in both sagittal and transverse planes. Image magnification is adjusted to allow volume estimation as well as detection of pathologies. Magnification is low during prostate volume determination to make the entire gland

visible. The optimal brightness setting results in a medium-gray image of normal prostate which also serves as a reference point for assessing pathological prostate [30].

TRUS is performed in both transverse and sagittal planes with patient in left lateral decubitus position. In transverse imaging, advancing the probe cephalad images the prostate base, seminal vesicle and bladder base while pulling out the probe will image the prostate apex and proximal urethra. Transverse image is achieved by angling the probe left or right using the anal sphincter as the fulcrum.

Two approaches to probe manipulation in sagittal imaging are by probe rotation and angling. Urologist prefer probes angling because of it is similar to manipulation of cystoscope and it is less uncomfortable [30].

3.4. Calculation of Prostate Volume

This involves measurement of transverse and anteroposterior (AP) dimension in the axial plane as well as measurement of the longitudinal dimension in the sagittal plane. Several formulae exist depending on the geometric shape of the prostate viz:

1) Ellipse shape: 0.523 x transverse diameter x AP diameter x longitudinal diameter.
2) Sphere: 0.523 x transverse diameter^3.
3) Prolate (egg shape) spheroid: 0.523 x transverse diameter^2 x AP diameter.

Another accurate way of determining prostate volume involves the use of planimetry especially when brachy-therapy is envisaged [30]. All formulae reliably estimate prostate volume with correlation coefficient greater than 0.90 and 1 cm^3 equals 1 g.

Udeh et al observed 120 men aged 45 to 85 years in Jos, North-central Nigeria, with a mean prostate volume of 72.79 +/- 44.38 cm^3. The Pearson's correlation between pre-treatment IPSS and prostate volume was -0.0035 ($p>0.05$) [31]. This shows that no significant relationship exists between IPSS and prostate volume in Africans.

4. Pathophysiological Pathway of Erectile Dysfunction and Benign Prostatic Enlargement

The tone of corpus cavernosal smooth muscle and penile vasculature regulating penile erection and detumescence is under control of various central and peripheral mechanisms and involves multiple neurotransmitters. Centrally acting neurotransmitters are acetylcholine, nitric oxide (NO), dopamine, oxytocin, noradrenaline and serotonin. Noradranaline and endothelins are promoters of penile detumescence, whereas NO promotes erection. NO binds to soluble guanosine monophosphate (cGMP), which signals phosphodiesterase, protein kinase and ion channel resulting in smooth muscle relaxation and penile erection [3].

Current evidence suggests that several common pathogenetic mechanism are involved in the development of both ED and symptomatic BPE [6].

Figure 2 demonstrates the pathophysiological pathways leading to ED and symptomatic BPE [3].

Mechanism currently considered includes:

1) Alteration of the nitric oxide and cyclic guanosine monophosphate pathway.
2) Enhancement of RhoA-Rho-Kinase (ROCK) signaling.
3) Autonomic hyperactivity.
4) Pelvic atherosclerosis.
5) Chronic inflammation and sex steroid ratio imbalance.

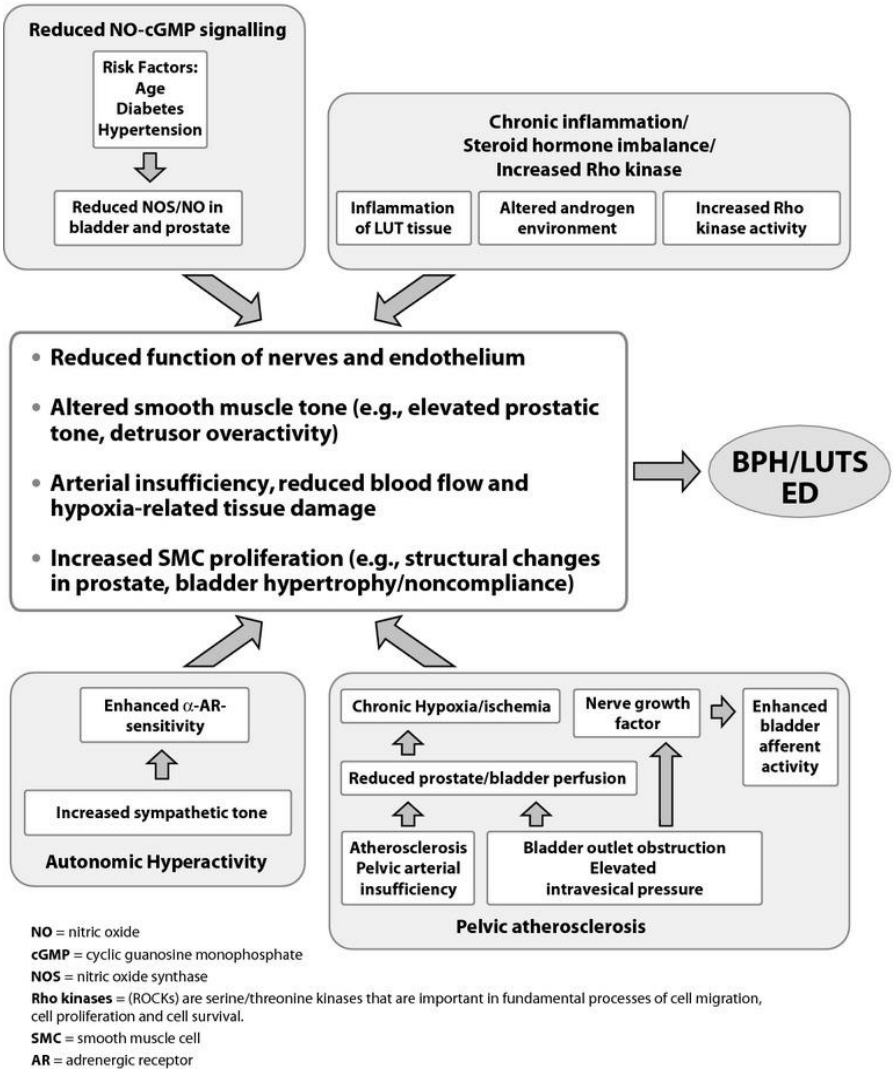

Figure 2. *Potential pathophysiological pathways of erectile dysfunction and BPE.*

Copied from Kirby M, Chappel C, Jackson G et al. Erectile dysfunction and lower urinary tract symptoms: a consensus on the importance of co-diagnosis. Int. J. Clin. Pract. 2013; 67(7): 606-618.

4.1. Diagnosis of Erectile Dysfunction with Benign Prostatic Enlargement

Diagnosis of ED with BPE may be rewarding due to the following reasons:

1) Given evidence from studies of possible correlation between ED and symptomatic BPE, patients presenting with ED or LUTS should be screened and treated for the other condition to ensure patient's satisfaction.
2) Management of ED with BPE may be improved by modifying the shared risk factors.
3) Early diagnosis and treatment of ED in patients with BPE could help reduce the risk of subsequent cardiovascular disease (CVD). Risk factors such as obesity, hypertension, arteriosclerosis, smoking and alcohol are associated with increased incidence of ED and CVD. Therefore prompt attention and modification of the risk factors can lower the incidence of CVD with reduced morbidity and mortality from symptomatic BPE.

Figure 3 summaries the importance diagnosis of ED with symptomatic BPE and provides a vital algorithm for the diagnosis of both conditions [3].

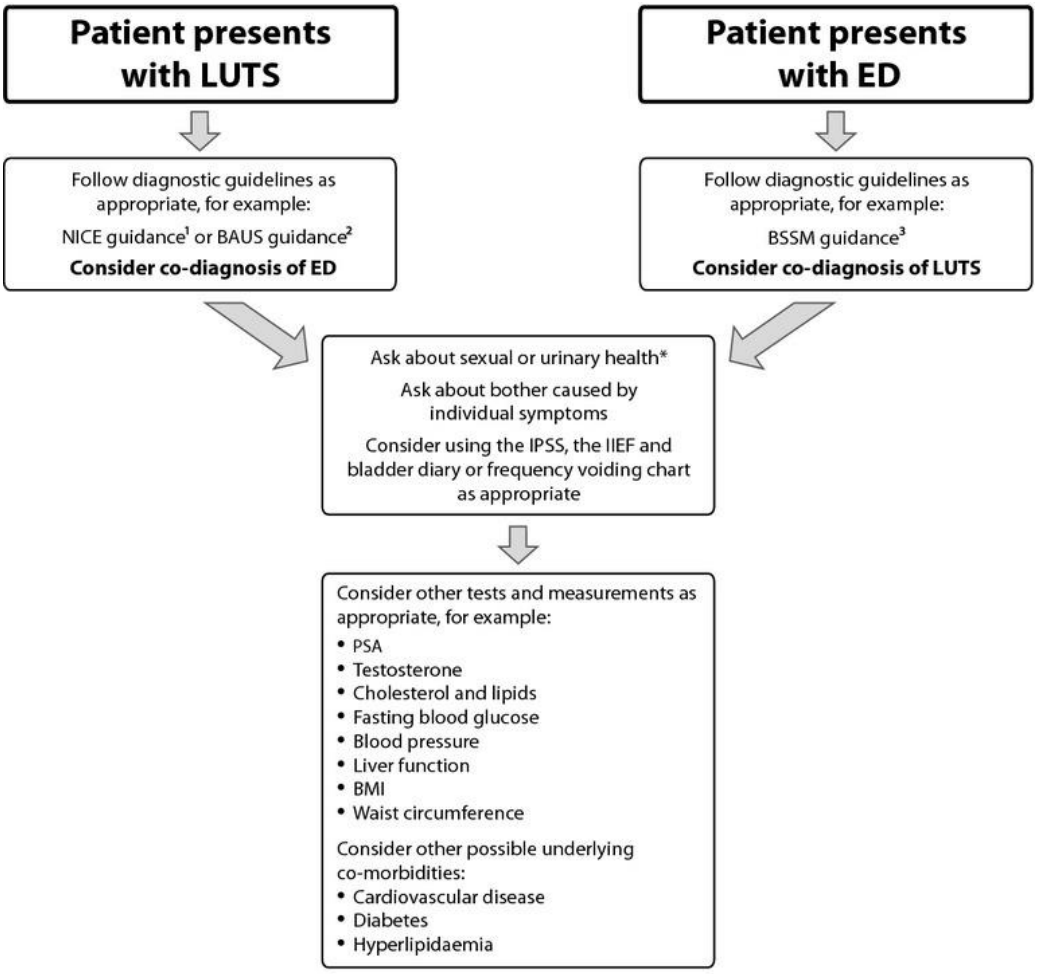

Figure 3. *Co-diagnosis algorithm for erectile dysfunction and lower urinary tract symptoms.*

Copied from Kirby M, Chappel C, Jackson G et al. Erectile dysfunction and lower urinary tract symptoms: a consensus on the importance of co-diagnosis. Int. J. Clin. Pract. 2013; 67(7): 606-618.

4.2. Correlation Between Erectile Dysfunction and Symptomatic Benign Prostatic Enlargement

Obiatuegwu et al found that the severity of ED was directly related to prostate size an exponential trend in the severity of ED and prostate size. The prevalence of ED in patients with BPE according to Obiatuegwu et al was 76.9% [1]. Braun et al noted similar trend and the prevalence of ED in men with BPE was 72% in approximately 5000 German men [7]. This confirms an obvious link between ED and BPE and further supports the need to consider the QoL and sexual performance in the management of symptomatic BPE as they will likely benefit from daily low dose tadsanafil.

Olugbenga-Bello et al observed that the overall prevalence of ED in Nigerian was 46% which was at variance with previous studies above [15]. The variation and higher prevalence noted by Obiatuegwu et al may be due to the study population as the latter dwelt more on patients with symptomatic BPE while Olugbenga-Bello et al focused on overall prevalence of ED. This shows that there may be a link between ED and symptomatic BPE which probably accounted for the higher prevalence of ED and supports the need for establishing co-existence ED in patients with symptomatic BPE.

5. Conclusion

Erectile dysfunction and benign prostatic enlargement have an obvious relationship due to the common pathophysiological mechanisms illustrated above. BPE may be an indicator of ED and patients should be evaluated holistically due to the high prevalence of ED in men with symptomatic BPE and positive correlation between both pathologies observed in several studies [1, 7, 15].

Abbreviations

ED	Erectile Dysfunction
BPE	Benign Prostatic Enlargement
LUTS	Lower Urinary Tract Symptoms

IPSS	International Prostate Symptoms Score
IIEF-5	International Index of Erectile Function
QoL	Quality of Life

Author Contributions

Obiatuegwu Kenenna: Conceptualization, Funding acquisition, Methodology, Project administration, Resources, Supervision, Validation, Writing – original draft, Writing – review & editing

Magnus Felix: Conceptualization, Funding acquisition, Project administration, Validation, Visualization

Aniede Ernest: Methodology, Project administration, Supervision, Visualization

Funding

This review article was funded by the authors.

Availability of Data and Materials

All data generated or analyzed in this study are included in this published article.

Conflicts of Interest

The authors declare no conflicts of interest.

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