Sarcouncil Journal of Medical Series

ISSN(Online): 2945-3550

Volume- 01| Issue- 01| 2022

Review Article **Received:** 05-03-2022 | **Accepted:** 15-03-2022 | **Published:** 21-03-2022

Epidemiology and Etiology of Benign Prostatic Hyperplasia and Bladder Outlet Obstruction

DR.POOJA SHARMA (BHMS)

Punjab University Medical Coder Genushealthcare Solution

Abstract: Benign prostatic hyperplasia (BPH) is a histological diagnosis associated with disordered growth of connective tissue, smooth muscle, and glandular epithelium. BPH can compress the urethra and cause bladder outlet obstruction (BOO). BOO can manifest itself in the form of lower urinary tract symptoms (LUTS), infections, retention, and other adverse events. BPH and BOO have a significant impact on the health and health costs of older men. As the world's population ages, the incidence and prevalence of BPH and LUTS are increasing rapidly. Immutable risk factors such as age, genetics, and geography play important roles in the pathogenesis of BPH and BOO, but recent data are new for treatment and prevention, such as sex steroid hormones and metabolic syndrome. Revealed variable risk factors that provide a path Syndrome, and cardiovascular disease-disease, obesity, diabetes, diet, physical activity and inflammation. Epidemiological studies identify the natural history, definitions, and key risk factors for BPH and BOO.

Keywords: Etiology, benign prostatic hyperplasia, bladder outflow obstruction, epidemiology, genetics, public health.

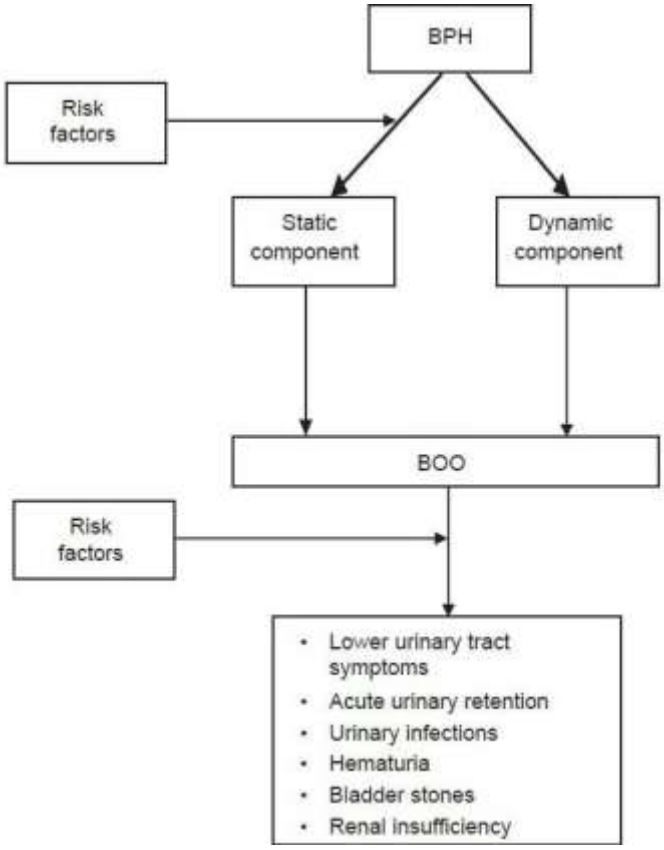
INTRODUCTION

Benign prostatic hyperplasia (BPH) is a histological diagnosis associated with the disordered growth of connective tissue, smooth muscle, and glandular epithelium within the prostate junction. And the interstitial element mainly composed of collagen and smooth muscle. In BPH, cell augmentation escalates the volume of the prostate gland and rapid growth of the stromal smooth muscle tone. There are two stages of BPH progression a described by McNeil. It also describes the escalation of BPH nodes in the periurethral zone in initial stage and there is a significant growth in the size of the glandular nodes in the next stage.

BPH causes physical compression of the urethra and can cause anatomical bladder outlet obstruction (BOO) through two different mechanisms. Second, an increase in interstitial smooth muscle tone called the dynamic component. Bladder Outlet Obstruction may then clinically refers to lower urinary tract symptoms (LUTS), urinary tract infections, acute urinary retention (AUR), renal failure hematuria, and bladder stones. In particular, two factors complicate the natural history and clinical manifestations of BPH, BOO, and LUTS. Overall, BPH, BOO, and LUTS are associated with increased risk of death, depression, falls, and poor health related quality of life, as well as billions of dollars in annual health care costs.

Over the last decade, the epidemiological models of BPH and BOO have evolved significantly. While age and genetics play important roles in the pathogenesis of BPH and BOO, recent data reveal new, modifiable risk factors that provide new paths for treatment and prevention. These risk factors appear to be able to affect the natural course of BPH and BOO at various stages of clinical progression. This is an overview of current concepts in the epidemiology and etiology of BPH and BOO.

***Corresponding Author:** DR.POOJA SHARMA (BHMS)



Definitions of BPH and BOO in Epidemiological Studies

The heterogeneity of case definitions in the literature presents a persistent challenge in interpreting data from population-based studies of BPH and OB. Researchers have used a variety of definitions for BPH, including histological analysis of prostate tissue, benign prostatic hyperplasia (BPE), decreased urine output, and appropriate urodynamic studies. With BOO, surgery is needed for BPH, AUR, and BPH and LUTS diagnosed by a doctor. LUTS describes a distinct phenotype of a group of disorders affecting the prostate and bladder with a common clinical presentation. In recent years, LUTS has become the preferred term for the study of urinary symptoms in men, as it allows for a broad epidemiological description of urinary symptoms without identifying an underlying cause. specific disease or condition. The most commonly used LUTS measures in epidemiological studies are the American Urological Association's (AUASI) Symptom Index (AUASI) and its validated international counterpart, the Prostate Symptoms Score International (IPSS). AUASI and IPSS are powerful and reliable metrics for measuring male LUTS. The AAU, European Association of Urology, and the World Health Organization's International Consultative Organization for Urological Diseases recommend the routine use of IPSS in the clinical evaluation of patients with suspected BPH and OB. The terms BPH, BOO, and LUTS remain interconnected in the modern treatment and research of urinary disorders in older men. However, previous epidemiological studies have not used the term "BOO" consistently. Instead, two terms commonly used in the literature to describe the clinical manifestations of BPH - i.e. the adverse clinical effects of BOO - are "BPH" and "LUTS". Therefore, the remainder of this review will primarily focus on the epidemiological risk factors associated with the etiology of BPH and LUTS in males.

Risk Factors for BPH and LUTS

At the population level, there are 2 major types of risk factors associated with BPH and LUTS: irreversible (age, geography, and genetics) and modifiable (sex steroid hormones, conversion syndrome) chemistry, obesity, diabetes, physical activity, diet, and inflammation).

Publisher: SARC Publisher

Non-modifiable	Modifiable
Age	Hormones
Genetics	Testosterone
Geography	Dihydrotestosterone
	Estrogen
	Metabolic syndrome
	Obesity
	Diabetes
	Diet
	Physical activity
	Inflammation

LUTS=Lower urinary tract symptoms, BPH=Benign prostatic hyperplasia, DHT=Dihydrotestosterone

The rate of HBP infection is clear with age. Studies of autopsy have observed the rate of 8%, 50% and 80% tissue for decades of the 4th, 6th and 9th, respectively. Some observational studies of Europe, the United States and Asia have proven older age as a risk factor for BHB's appearance and clinical progress with a number of different figures. [The rate of struggle also increased in older men. In the bone fracture joints in men, studying a unit study, a future study of 6,000 men of the community over 65 years old in the United States, 29% of people are not useful when starting to develop concerned struggles Clinical weight within 2 years. Tracking up; Of these ≥ 80 years, this rate increased to 34% to 34%. In the American county olmsted, 14% of non-litle men at the base then reported medium or heavy symptoms within 18 months after monitoring and 22% reporting medium symptoms or Serious within 42 months. Similarly, 21% of Japanese people, 26% of black Americans and 20% of men with null or light aggregates have submitted symptoms after 3, 4 and 5 years Tracking, respectively a recent study of Platz et al., According to 9628 men for the growth of 18 years on the basis of IPSs and observed the incidence and progress of struggles has increased significantly as older men, the progress rate higher than the incidence.

Genetics

Evidence suggests that there are genetic components to both BPH and LUTS. A case-control analysis, in which men under 64 years of age underwent BPH surgery, reported a 4- and 6-fold increased risk of age-specific BPH surgery. 'BPH in all male relatives and siblings, respectively, of cases. These researchers further estimated that 50% of men who had BPH surgery under the age of 60 had a genetic form of the disease. These and other results suggested an autosomal dominant mode of inheritance. Men with inherited forms of BPH tend to have larger prostheses and an earlier age of onset of clinical symptoms than men with sporadic BPH diseases. Homozygous twin concordance rates were 63% and 26% for LUTS and BPH, respectively, with one study estimating that genetic factors may contribute up to 72% to the risk of moderate or severe LUTS. severe in aged men.

Genetic polymorphisms have also been implicated in the development of brown planthoppers. In a study of 160 North Indian men with LUTS, deletion of the glutathione stransferase genes of the enzyme thought to confer cellular resistance to oxidative stress was significantly associated with an increased risk with symptomatic BPH. in men with a CAG repeat in the androgen receptor gene and a 16-fold increased risk in the presence of prostate-specific antigen (PSA) G158A single nucleotide polymorphism 5alpha reductase inhibitors (Finasteride and dutasteride) reduce serum DHT levels] and suppress clinical progression of BPH and LUTS.] First study suggests it may prevent the onset of BOO in men gender without symptoms. Unlike DHT, there is

Publisher: SARC Publisher

no clear pattern of estrogen, BPH and LUTS yet. Previous studies have reported positive, negative and null associations of endogenous estrogens with BPH and LUTS. However, one study observed increased efficacy in reducing stromal cell proliferation in human BPH through the use of selective estrogen receptor modulators in combination with 5 -alpha reductase.

The Metabolic Syndrome and Cardiovascular Disease

A notable relatively recent development in BPH and OB epidemiology is the recognition that modifiable lifestyle factors affect the natural history of these conditions. Increasing evidence suggests that many of the same metabolic disorders associated with cardiovascular disease and lifestyle factors that correct these disorders affect the risk of BPH and LUTS. These observations are important because they suggest new goals for prevention and treatment.

Metabolic syndrome is a collection of obesity, impaired glucose tolerance, lipid disorders, and hypertensive metabolic disorders that increase the risk of cardiovascular disease and are primarily due to diet. Other lifestyles common in Western society.

Despite heterogeneity in definition and diagnosis, accumulating evidence suggests an association between metabolic syndrome and an increased risk of BPH and LUTS In a cohort, men were diagnosed with at least three components: of metabolic syndrome had an increased incidence of SBAU to 80% compared with those without the component. Other studies have shown that men with heart disease have a significantly increased risk of BPH and LUTS

Obesity

Previous studies have consistently observed that increased obesity is positively correlated with prostate volume. The greater the amount of obesity, the larger the volume of the prostate. Body weight, body mass index (BMI), and waist circumference were all positively correlated with prostate volume in several different study populations. For example, in the BLSA cohort, for every 1 kg / m2 increase in BMI, the prostate volume increases by 0.41 cm3. In addition, participants with obesity (BMI ≥3
5 kg/m2) participant

Diabetes and Disruptions in Glucose Homeostasis

Impaired glucose homeostasis at several different levels, from changes in serum insulin growth factor (IGF) levels to the diagnosis of clinical diabetes, is associated with a high probability of BPH, BPE, and LUTS. Elevated serum levels of IGF1 and insulin-like growth factor-binding protein 3 are associated with an increased risk of clinical BPH and BPH surgery] Doctor-diagnosed diabetes, elevated serum insulin, and elevated fasting plasma glucose , Associated with benign prostatic hyperplasia. Increased risk of benign prostatic hyperplasia in associated clinical BPH, BPH surgery, and LUTS in several different cohorts involving cumulatively tens of thousands of men. Recent results from Olmsted County show that men with diabetes who are receiving medical treatment are less likely to develop moderate / severe LUTS than men who are not on medication.

Physical Activity

Increased physical activity and exercise was robust and consistently associated with BPH surgery, clinical BPH, histologic BPH and LUT low. 11 Published research (N = 43,083 people) meta-analysis is 25% from medium to strong physical activity, with higher level of protection effect to 25% for sesfer lifestyle It was shown to reduce BPH or clay risk to reduce. activity.

Diet

Although the pattern is inconsistent, there are several evidence that both macronutrients or trace nutrients can affect BPH and LUT risks. In the case of macro nutrients, increase in total energy intake, energy intensive all protein ingot, red meat, fat, milk, dairy, serial, bread, poultry and strength increase the risk of symptomatic BPH and BPH surgery There is a possibility. Vegetables (especially carotenoids), fruits, large numbers of unsaturated fatty acids, linoleic acid, vitamin A and vitamin D can reduce the risk of interactive BPH and LUT. [Trace nutrients, higher circulating concentrations of vitamin E, Lycopen, Selenium and carotene are opposite to BPH and LUT, LUT is zinc and vitamin C, increase and reduction of risk Finally, from the viewpoint of alcohol consumption, it was observed that alcohol intake was associated with the increase in risk and LUT.

Inflammation

Most of the observation research suggests that inflammation is associated with BPH and LUT onset. The underlying mechanism of this relationship is unknown. A possible explanation is

Publisher: SARC Publisher

that metabolic syndrome, which promotes systemic inflammation and oxidative stress, mediates the compound. Inflammation is associated as a major stimulus for the development of prostate cancer, and BPH may represent a non-malignant growth pathway promoted by oxidative stress and inflammatory mediators. There is a strong association between BPH and histological inflammation in surgical specimens, with the degree and severity of inflammation corresponding to the degree and BPH region of benign prostatic hyperplasia [high IPSS score] LUTS men have serum-active proteins. Is more likely. A marker of systemic inflammation, previous gonaditis infection or prostatitis increases the likelihood of BPH surgery, and high serum IgG antibody titers against LUTS PSA, cytomegalovirus, herpesvirus, human papillomavirus, and hepatitis are LUTS. Is related to. Conversely, blocking the inflammatory pathway may reduce the risk of BPH. In a community cohort, men who reported daily use of nonsteroidal anti-inflammatory drugs (NSAIDs) showed a significantly reduced risk of LUTS, decreased urinary flow, increased prostate volume, and increased PSA.

Other Risk Factors

Other modifiable risk factors for which no clear risk pattern has yet been identified include hypertension, serum lipids and lipoproteins, and smoking.

CONCLUSION

In conclusion, BPH and BOO are significant public health concerns affecting tens of millions of older men worldwide. Current disease trends in the United States, Europe, and elsewhere are that the incidence and prevalence of these diseases will increase in the near future due to the aging of the world's population and the increase in metabolic syndrome and its components, and thus further. It suggests that it will be a heavy burden. With finite resources. Age and genetic factors contribute to the development of BPH and BOO, but many variable variables are also variable factors to delay the onset, prevent progression, or alleviate symptoms. Contribute. Potential strategies include inhibition of DHT synthesis by 5-α-reductase inhibitors, regulation of metabolic risk factors by extensive lifestyle interventions including diet and physical activity, and suppression of inflammatory pathways by NSAIDs.

REFERENCES

1. Kaplan, S.A., Reis, R.B., Staimen, V.B. and Te, A.E. "Is the ratio of transition zone to total prostate volume higher in African-American men than in their Caucasian or Hispanic counterparts?." *British journal of urology* 82.6 (1998): 804-807.
2. Fowke, J.H., Munro, H., Signorello, L.B., Blot, W.J. and Penson, D.F. "Association between socioeconomic status (SES) and lower urinary tract symptom (LUTS) severity among black and white men." *Journal of general internal medicine* 26.11 (2011): 1305-1310.
3. Kang, D., Andriole, G.L., Van De Vooren, R.C., Crawford, D., Chia, D., Urban, D.A., Reding, D., Huang, W.Y. and Hayes, R.B. "Risk behaviours and benign prostatic hyperplasia." *BJU international* 93.9 (2004): 1241-1245.
4. Platz, E.A., Kawachi, I., Rimm, E.B., Willett, W.C. and Giovannucci, E. "Race, ethnicity and benign prostatic hyperplasia in the health professionals follow-up study." *The Journal of urology* 163.2 (2000): 490-495.
5. Sanda, M.G., Doehring, C.B., Binkowitz, B., Beaty, T.H., Partin, A.W., Hale, E., Stoner, E. and Walsh, P.C. "Clinical and biological characteristics of familial benign prostatic hyperplasia." *The Journal of urology* 157.3 (1997): 876-879.
6. Meikle, A.W., Bansal, A., Murray, D.K., Stephenson, R.A. and Middleton, R.G. "Heritability of the symptoms of benign prostatic hyperplasia and the roles of age and zonal prostate volumes in twins." *Urology* 53.4 (1999): 701-706.

Source of support: Nil; **Conflict of interest:** Nil.

Cite this article as:

Sharma, P. "Epidemiology and Etiology of Benign Prostatic Hyperplasia and Bladder Outlet Obstruction." *Sarcouncil Journal of Medical Series 1*.1 (2022): pp 1-5

Publisher: SARC Publisher