

Benign prostate hyperplasia prevalence: Enugu state of Nigeria: A case study

Ugwuene Francis O ^{1,*}, Soronnadi Clara N ² and Asogwa Benneth E ³

¹ Department of Medical Laboratory Science, Faculty of Allied Health Sciences, Enugu State University of Science and Technology (ESUT); Enugu, Nigeria.

² Department of Physiology, Faculty of Basic Medical Sciences, ESUT; Chemical Pathology Enugu, Nigeria.

³ Chemical Pathology Department, Esut Teaching Hospital, Enugu, Nigeria.

World Journal of Advanced Research and Reviews, 2025, 26(02), 2281-2285

Publication history: Received on 30 March 2025; revised on 09 May 2025; accepted on 11 May 2025

Article DOI: <https://doi.org/10.30574/wjarr.2025.26.2.1578>

Abstract

The prevalence of BPH varies from race to race, tribe to tribe and from country to country. It became necessary to know the BPH prevalence in Enugu State of Nigeria following the rising cases of BPH, Prostatitis and Prostate cancer in Nigeria. A total of 237 adult men at the age range of 40 to 80 years were tested for PSA, using the principle of immunological antibody antigen reaction. It was statistically noted that the PSA of men between the ages of 40 to 49 years fell within normal physiological reference range. PSA of men within 40 to 60 years showed significant increase ($P < 0.05$) while that of men within 61 to 80 years had significant increase of PSA ($p < 0.05$). This work concludes that there is a prevalence of both histological enlargement of prostate and clinical BPH in Enugu state of Nigeria.

Keywords: Prevalence; Prostate; BPH; PSA

1. Introduction

Benign Prostate Hyperplasia (BPH) also known as Benign Prostate Hypertrophy is a condition in men in which the prostate gland is enlarged and not cancerous. On the other hand, it is a multifocal, non-malignant hyperplastic, and progressive histopathological change in stromal and epithelial cells in the transitional zone of the prostate gland, resulting in discrete prostatic nodules, inflammation, fibrosis and changes in smooth muscle activity, which can cause partial or complete obstruction of the urethra (1,2).

The prostate gland is an exocrine gland that lies in the pelvic cavity between the urinary bladder and the base of the penis. It also lies in front of the rectum (3). It is about the size of a walnut with the weight of about 8g in male youth but progressively hypertrophies with age and could weigh 40g by the age of 50 years. The prostate is divided into two lobes, the right and the left lobes. The upper part of the prostate is called the base and it rests against the lower part of the bladder, while the lower part is called the apex. The prostate is made up of many branching ducts surrounding the stroma. The cells that line the prostate ducts make prostate fluid which is mixed with the sperm and other fluids to make alkaline semen (4). The alkalinity and enzyme content of the prostate fluid helps to thicken the semen, increase the motility and life span of the sperm cells in the vagina. The sodium, chloride, potassium, calcium, magnesium, zinc, proteins, citrate and sugars among some antigens and enzymes such as Prostate Specific Antigens (PSA) help to maintain the potency of the sperm cells. The PSA and ACP are often used along with radionic scans/ultrasound, and Computer Thermography (CT) scan as diagnostic tools for the investigation and management of prostrate disorders including BPH (4,5).

Sonographically, there are about eight types of BPH, namely: Type 0, shows an equal or less than 25cm³ prostate showing little or no zonal enlargement. Type one shows bilateral transition zone (TZ) enlargement. Type two shows

* Corresponding author: Ugwuene Francis O.

retro urethral enlargement, Type three shows bilateral TZ and retro urethral enlargement while Type four is pedunculated enlargement. Type five shows pedunculated enlargement with bilateral TZ and or retro urethral enlargement, while type six is seen with subtrigonal or ectopic enlargement. Type seven shows other combinations of enlargement (6).

The prostate is basically of four zones; anterior fibromuscular stroma, central zone, peripheral zone and transition zone. The transition zone is a balloon-shaped component of the prostate that is located in the periurethral region. Most prostatic adenocarcinomas develop in the peripheral zone, although some arise in the transition zone. The transition zone is the exclusive site of benign prostatic hyperplasia. Both adenocarcinoma and BPH affecting the transition zone may cause urinary obstruction. Carcinomas that develop in the transition zone are generally well differentiate (6,7,8,9).

When a man reaches the age of 40 years and especially on the mid 40s, the area of the prostate gland that encircles the urethra especially the area of the transitional zone, begins to grow beyond the normal size. The prostate growth at this stage is more dependent on the hormonal secretion of the testes known as the androgens, especially the testosterone. The testosterone is converted to dihydrotestosterone by the catalytic activity of 5 - reductase (10,11).

The hyperplasia of prostate gland that is mainly associated with age is a non-cancerous increase in size of the prostate (12).

Apart from age, genetics and hormones, other factors such as metabolic syndrome (type 2 diabetes, and low level of high-density lipoprotein in take), recurrent bacterial infection and recurrent activation of growth factors, are associated with development of BPH rises markedly with increased in age (13,14).

The age specific prevalence of benign prostate hyperplasia (BPH) has been estimated from autopsy studies to be 8% in fourth decades of life, 50% in the six decades of life, and 80% in the ninth decades of life (15). There is yet no clear pattern of prevalence of BPH due to race, as variable results have been produced in observational studies comparing black, Asian and white men (15). There is a genetic component of BPH prevalence. A case control analysis in which men below 64 years underwent surgery for BPH noted that male relatives, respectively and brothers had 4-fold and 6-fold same age range risks for BPH surgery (16). It was equally observed that about 50% men below 60 years undergoing surgery for BPH has a heritable form of the disease and other findings suggest an autosomal dominance pattern of inheritance (17). There were indications that modifiable lifestyle factors and macro and micronutrients may substantially influence the natural history of BPH. Macro nutrients, increased total energy in-take, energy-adjusted total protein in-take, red meat, fat, milk and- dairy products, cereals, bread, poultry and starch, all potentially increase the risks of clinical BPH and BPH surgery, while vegetables, fruits, polyunsaturated fatty acids, linoleic acid and vitamin D, potentially decrease the risk of BPH. Also, higher circulating concentration of Vitamin E, lycopenes, selenium and carotene have been inversely associated with BPH (15).

Physical activity is another factor that is associated to BPH prevalence. A meta-analysis of some published work indicate that moderate to vigorous physical activity reduced the risk of BPH by as much as 25% relative to sedentary lifestyle (18,19). Metabolic syndromes such as diabetes, disruption of glucose homeostasis and obesity have been linked to increased prostate size and volume and prevalence of BPH and BPH surgery (15) Inflammatory conditions brought about by inflammatory cytokines from adipose tissues is associated with increased risk of prevalence of BPH (15). There is also overwhelming evidence to support that erectile dysfunction and lower urethral tract diseases may be contributing to prevalence of BPH [20].

Worldwide prevalence of BPH varies from 20-62% in men over 50 years and this includes USA, UK, Japan. While reports from Ghana and South Africa have BPH prevalence of 20-62% in men over 50 years and 50% in adult males of 60 years respectively (15). In Ekiti State of Nigeria (Western Nigeria), the prevalence rate is between 104 per 1000 and for men at fifth and seventh decade of life and 429 per 1000 between eighth to ninth decades of life and above (21). This work centered more on determining the BPH prevalence of men between 4th to 5th decades of life, 6th to 7 decades of life and 8 and above decades of life in Enugu State using prostate specific antigen as diagnostic index.

2. Materials and methods

This research work was carried out in Enugu metropolis of Enugu state of Nigeria. A total of 237 adult men at the age range of forty (40) to eighty (80) years were used in the study. The subjects were grouped into three, and blood samples were collected respectively and their prostate specific antigen tests done using the principle of immunological antibody-antigen reaction.

Group 1 was made up of 36 men at the age range of forty (40) to forty-nine (49), group 2 was made up of 117 men at the age range of fifty (50) to sixty (60) years while group 3 was made up of 84 men at the age range of sixty one (61) to eighty (80) years. The mean and standard deviation of each group were obtained from their respective raw data and represented as shown below.

The PSA test was based on the principle of immunological antibody-antigen reaction. The reaction of the prostate specific antigen (PSA) in the conjugates in the system produces pinkish purple with the incorporated chromogen. The color concentration is directly proportional to the concentration of the prostate specific antigen in the serum semen respectively (20).

Consent was obtained from each of the 237 men used for this study. Data analysis was done by the use of students t-test statistical method.

3. Result

Table 1 PSA levels of three age groups of men in Enugu metropolis of Nigeria

PSA	GROUP 1 AGES 40-49 YRS	GROUP 2 AGES 50-60 YRS	GROUP 3 AGES 61-80
	N = 36	N = 117	N = 84
PSA mg/ml (Mean \pm SD)	3.3 \pm 1.8	6.5 \pm 3.3	9.0 \pm 1.36

Table 1 shows the progression of increase of PSA levels from the average ages of 40 – 49, 50 – 60 and 61 – 80.

Table 2 Statistical comparison of degree of significance of PSA levels of group 1 and group 2

PSA	GROUP 1 AGES 40-49 YRS	GROUP 2 AGES 50-60 YRS	t- Test
	N = 36	N = 117	
PSA mg/ml (Mean \pm SD)	3.3 \pm 1.8	6.5 \pm 3.3	P < 0.05

Table 2 shows that there was significant increase in PSA levels of men within the ages of 50 – 60 years when compared with those of 40 – 49 years.

Table 3 Statistical comparison of degree of significance of PSA levels of group 2 and group 3

PSA	GROUP 2 AGES 50-60 YRS	GROUP 3 AGES 61-80 YRS	t - Test
	N = 117		
PSA mg/ml (Mean \pm SD)	6.5 \pm 3.3	9.0 \pm 1.3	P < 0.05

Table 3 shows that there was significant increase in the PSA levels of men within the ages of 61 – 80 years when compared with those of 50 – 60 years.

4. Discussion

This study of benign prostatic hyperplasia prevalence in Enugu State of Nigeria centered more on evaluating the BPH prevalence of men between fourth to fifth decades of life, sixth to seventh decades of life and eighth and above decades of life. This was done through the biochemical estimation of the total PSA of the above written groups of men in Enugu

state of Nigeria. The Prostate Specific Antigen (PSA) status of the three groups were compared against the known research established normal physiological reference range of ≤ 4.0 ng/ml. In this Enugu Metropolis research work, the mean and standard deviation of group 1 (40 – 49) years men serve also as a normal reference control for group 2 and 3 men.

The results of the PSA estimation among the three age range groups of men showed that statistically the PSA levels of the first group of men (40-49 years) fell within the standard physiological normal reference range of PSA of men. However, the PSA of a few men in the raw data of that group showed a marginally raised PSA, indicating that the normal physiological reference range of PSA levels of men between 40 to 49 years is not absolute.

The PSA results of group 2 men (50 - 60) years showed a significant increase when compared with group 1 men ($P < 0.05$). This finding agrees with some past research findings which stated that both histological enlargement of prostate and clinical BPH have been observed in men within the ages of 50 to 60 years (21,22,23).

The PSA results of group 3 men (61-80 years) showed a significantly raised value when compared with the group 2 men ($P < 0.05$). This is also in consonance with Ojowola, et al 2017 (18) which stated that between 5th to 9th decades of life there was a prevalence of BPH in Ekiti State of Nigeria. Generally, the molecular etiology of BPH has been observed through complicated and poorly understood processes, yet several risk factors for its development have been identified to include age, genetics (family history), obesity, type 2 diabetes mellitus, hormones, growth factors, inflammation and life style factors such as poor exercise (22,23,). During this research work, it was observed that a good percentage of men within the age of 50 to 60 years with raised PSA levels had no clinical symptoms of BPH.

5. Conclusion

This work therefore concludes that there was a prevalence of both histological enlargement of prostate gland and clinical BPH in Enugu State of Nigeria within the ages of 50 to 80 years of men.

Compliance with ethical standards

Disclosure of conflict of interest.

There was no conflict of interest.

Statement of informed consent

Consent was obtained from all individual participants used in the study.

References

- [1] McVary KT, Roehrborn CG, Avins Al et al (2011). Update on the management of benign prostate hyperplasia. *Journal of Urology* 185:1793-17-1803.
- [2] Bushman W (2009). Etiology, epidemiology, and natural history of benign prostate hyperplasia. *Urological clinic of North America* 36:403 - 415.
- [3] Yonemabu H. Nakamura M, et al (2004). Anatomical features of periprostatic tissue and its surroundings: a histological analysis of 79 radical retropubic prostatectomy specimens. *Journal of clinical oncology. JPN.*
- [4] Waugh A. and Grant A. (2014). Reproductive System in Ross and Wilson Anatomy and Physiology in Health and illness. 12th Ed. Pg 462.
- [5] Kavanaugh J.P (1985). Sodium Potassium, calcium, zinc, citrate and chloride content of Human prostate and seminal fluid. *Journal of Reproduction and Fertility.* 75:35-4.
- [6] Serk and Guneli, Emily Ward, Stephen Thomas, Ambereen Nehal-Yusuf, Igor Trillisky, Yabus Peng, Tat Jana Antic and Aytekin Ofo (2016). The Magnetic Resonance Imaging of Benign Prostate Hyperplasia. *Journal of Diagnostic Interventional Radiology.* 22 (3): 215 – 219.
- [7] MacNeal JE(1992). Normal histology of the prostate. *American Journal of Surgical Pathology.* 1 12:619 – 623.
- [8] Walsh P.C, Lepor H, Eggleston J.L (1983) Radical Prostatectomy with preservation of the sexual function: anatomical and pathological consideration. *Prostate* 4: 473 - 485.

- [9] Walsh P.C, Epstein JI, Lowe F.C (1987) Potency following radical Prostatectomy with wide unilateral excision of neurovascular bundle. *Journal of Urology* 138:823 - 827.
- [10] Wen Chang H.C, Tian J, Shang Z, Niu Y, Chang C (2015). Stromal androgen receptor roles in the development of normal prostate, benign prostate hyperplasia and prostate cancer. *Annal Journal of Pathology*. 185L:293 – 301.
- [11] National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) (2014). Prostate Enlargement September.
- [12] Lee JC, Muller LH, Rothman I et al (2003) Prostate biopsy culture finding of men with chronic Pelvic pains, do not differ from those of Healthy controls. *Journal of Urology* 169 (2): 584-587.
- [13] Kok Bin Lim (2017). Epidemiology of Clinical Benign Prostate Hyperplasia. *Asian Journal of Urology* 4 (3): 148-151.
- [14] Sanda M.G, Betv. H, Stutzman RE, Childs B, Walsh P.C (1994), Genetic Susceptibility of Benign Prostate Hyperplasia. *Journal of Urology* 152: 115.119.
- [15] Pearson J.D, Lei H.H, Beauty T.H, Wiley K.E, Isaacs S.D, Isaacs WB (2003). Familial aggregation of brotherhood Benign Prostatic hyperplasia. *Urology* 71: 781-785.
- [16] Fowke J.H, Philip S, Koyama T, Byerly S, Concepcion R, Motley S.S, (2013), Association between physical activity, lower urinary tract symptoms and prostate volume. *Bristol Journal of Urology Int. Ill*: 122 - 128.
- [17] Persons J.K, Bergstrom J, Berrett-Conor E (2008). Lipid, lipoproteins and the risks of benign prostatic hyperplasia in community dwelling men. *British Journal of Urology International*. 101:313 - 318.
- [18] Ojewole R.W, Oridota E.S, Balogun O.S, Olabi T.O, Ajayi A.I, Olajide T.A, Tijani K.H, Jeje E.A, Ogunjimi M.A, Ogundare E.O (2017). Prevalance of Clinical Benign Prostatic Hyperplasia amongst community-dwelling men in South Western Nigerian rural setting: A cross sectional study. *African Journal of Urology* 23 (2) June 109-115.
- [19] Fitz -Patrick J.M (2006). The Natural history of Benign Prostate Hyperplasia. *British Journal of Urology International*.97(suppl 2): 3-6.
- [20] Oesterling J.E, Jacobsen S.J, Chute C.G, Guess H.A, Girman C.J, Panser L.A, Lieber M.M (1993). Prostate Specific Antigen in the Diagnosis and Treatment of Prostate Cancer. *Journal Urology*, 150(2), 332-337.
- [21] Altok M, Bagci O, Umiu M, Gunes M, Akuyz M, Unic F et al (2016). *British Journal of Urology International (Suppl 2)*: 3 6,
- [22] Chughtai B, Lee R, Te A, Kaplans (2011). Role of inflammation in BPH. Review. *Urology* 13:147-150.
- [23] Parson, J.K (2010). Benign Prostatic Hyperplasia and male lower urinary tract symptoms: Epidemiology and risk factors. *Current Bladder dysfunction Reports*. 5: 212 -218.