



CODEN [USA]: IAJPBB

ISSN: 2349-7750

## INDO AMERICAN JOURNAL OF PHARMACEUTICAL SCIENCES

<http://doi.org/10.5281/zenodo.2530220>Available online at: <http://www.iajps.co m>

Research Article

### ANALYSIS OF RELATIONSHIP OF PROSTATE VOLUME WITH INCIDENCE OF PROSTATE CANCER

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**Introduction:** Benign prostatic hyperplasia (BPH) and prostate cancer (PCa) are the most common diseases of the prostate; however, their interaction is not well studied. Historically, elucidating the epidemiology of BPH has been complicated by the lack of a uniform definition of clinical BPH, quantitative instruments for assessing the severity of lower urinary tract symptoms, and a noninvasive and accurate method for measuring both prostate volume and bladder outlet obstruction. **Objectives of the study:** The main objectives of the study is to find the relationship of prostate volume with incidence of prostate cancer. **Methodology of the study:** This study was done at Ghurki Trust and Teaching Hospital during 2018. For each patient prior to performing a warranted prostate biopsy, a complete history was collected and physical examination was performed. The prostate volume for each patient was estimated by DRE and confirmed by transrectal ultrasound (TRUS). Patients who underwent prior biopsies or prior surgeries were excluded from our study. **Results:** The study group comprised of 100 patients 72 (93.5%) were initial steroid resistant and 28 were late non-responders. Gender distribution showed 49 (63.6%) males and 28 (36.4%) females with a ratio of 1.75. We then determined the Gleason scores for prostates with a volume of <35 and >65 cc. Of the 110 patients testing positive on biopsy with a volume of <35 cc, 10 patients (9.09%) had a Gleason score of  $\geq 8$ . Of the 27 patients testing positive on biopsy with a volume of >65 cc, 1 patient (3.7%) had a Gleason score of  $\geq 8$ . Age range of patients was 1-15 years with a mean of  $8.11 \pm 3.58$  years. **Conclusion:** It is concluded that there is an inverse association of prostate volume with the incidence and biological aggressiveness of PCa. Data from this study and the outlined discussion should encourage other clinicians and investigators to further explore the relationship between prostate volume and the incidence and aggressiveness of PCa, to further investigate this phenomenon.

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Please cite this article in press Muhammad Tanveer Ashraf et al., Analysis of Relationship of Prostate Volume with Incidence of Prostate Cancer., Indo Am. J. P. Sci, 2019; 06(01).

**INTRODUCTION:**

Benign prostatic hyperplasia (BPH) and prostate cancer (PCa) are the most common diseases of the prostate; however, their interaction is not well studied. Historically, elucidating the epidemiology of BPH has been complicated by the lack of a uniform definition of clinical BPH, quantitative instruments for assessing the severity of lower urinary tract symptoms, and a noninvasive and accurate method for measuring both prostate volume and bladder outlet obstruction [1]. Several investigators in different countries found that the prevalence of clinical BPH is fairly uniform worldwide and also consistently age related. Symptomatic BPH affects ~20% of men in the age-group of 50–59 years, 30% of men in the age-group of 60–69 years, and 40% of men in the age-group of 70 years and older. Nephrotic syndrome is characterized by gross proteinuria, hypo albuminemia, hyperlipidemia, and peripheral edema [2]. The etiology of nephrotic syndrome in adults is complex and ranges from primary glomerulonephritis to secondary forms. Primary forms of nephrotic syndrome in adults are comprised of three histological disease entities: idiopathic membranous nephropathy (iMN), minimal change disease (MCD), and focal segmental glomerulo sclerosis (FSGS) [3]. Prostate-specific antigen (PSA) is a widely used, albeit controversial, diagnostic tool in the detection of PCa. Serum PSA levels of >4.0 ng/mL has a sensitivity of ~20% and a specificity in the range of 60–70%. The low sensitivity may be due to the fact that PSA serum levels can be elevated in the presence of benign pathology such as BPH and prostatitis [4,5].

**Objectives of the study**

The main objectives of the study is to find the relationship of prostate volume with incidence of prostate cancer

**METHODOLOGY OF THE STUDY:**

This study was done at Ghurki Trust and Teaching Hospital during 2018. For each patient prior to performing a warranted prostate biopsy, a complete history was collected and physical examination was performed. The prostate volume for each patient was estimated by DRE and confirmed by transrectal ultrasound (TRUS). Patients who underwent prior biopsies or prior surgeries were excluded from our study. A minimum of six biopsies were performed in each lobe in addition to biopsies obtained if suspicious lesions were encountered. PSA density data were obtained by dividing the PSA serum level by the TRUS-confirmed prostate volume. Demographic data of the patients, treatment received, and outcome of treatment and complications were recorded.

**Statistical analysis**

Student's t-test was performed to evaluate the differences in roughness between group P and S. Two-way ANOVA was performed to study the contributions (SPSS 19.0 for Windows, SPSS Inc., USA).

**RESULTS:**

The study group comprised of 100 patients 72 (93.5%) were initial steroid resistant and 28 were late non-responders. Gender distribution showed 49 (63.6%) males and 28 (36.4%) females with a ratio of 1.75. We then determined the Gleason scores for prostates with a volume of <35 and >65 cc. Of the 110 patients testing positive on biopsy with a volume of <35 cc, 10 patients (9.09%) had a Gleason score of  $\geq 8$ . Of the 27 patients testing positive on biopsy with a volume of >65 cc, 1 patient (3.7%) had a Gleason score of  $\geq 8$ . Age range of patients was 1-15 years with a mean of  $8.11 \pm 3.58$  years. Sixty nine (89.6%) patients underwent renal biopsy. Patients with focal segmental glomerulosclerosis (FSGS) were least likely to respond to treatment followed by mesangio proliferative glomerulonephritis and minimal change disease.

**Table 01:** Demography of 100 selected patients

Category	Number (%age)
Biopsied	69(89.6%)
Un biopsied	08(10.4%)
Initial SR	72(93.5%)
Late SR	05(6.5%)
Males	49(63.6%)
Females	28(36.4%)
Age(Years)	
<4	22(28.6%)
4-10	31(40.2%)
>10	24(31.2%)

**Table 2:** Gleason scores by prostate volume

Parameters	Volume	Volume	P-value
	<35 cc	>65 cc	
Number of patients	166	66	
Biopsy-positive patients, n (%)	110 (66.27)	27 (40.9)	P<0.001
Patients with Gleason score ≤7, n (%)	100 (90.9)	26 (96.29)	P<0.03
Patients with Gleason score ≥8, n (%)	10 (9.09)	1 (3.7)	

**DISCUSSION:**

Prostate volume of  $\leq 35$  cc had a 66% positive biopsy rate, whereas prostate volume of  $\geq 65$  cc had a 40% positive biopsy rate, ie, a reduction of 39.4%. A recent study described an inverse relationship between prostate symptom score and PCa [5]. Since a prostate symptom score would correlate with prostate volume, this pattern would be expected when compared with the studies mentioned earlier. However, this study could not show an increase in the accuracy of cancer detection in the multivariate analysis. Another study has shown an association between prostate volume and high-grade advanced PCa [7]. A recent publication examined the association of PCa volume and prostate size. Their data showed that small-volume cancers ( $<0.5$  g) were twice as common in larger glands ( $>50$  g) compared to smaller glands ( $<50$  g). This may imply that PCa may spread with less difficulty in smaller glands [8]. In another recent prospective study, a large cohort of 1,044 men underwent multiparametric magnetic resonance imaging (MRI) and then 12-core systemic mapping biopsy with additional MRI–fusion ultrasound (US) biopsy if the previous MRI had detected suspicious lesions of the prostate. This large study also revealed an inverse association of prostate volume with incidence and higher Gleason score ( $>7$ ) for PCa. Other previous clinical studies have also described the phenomenon of decreased cancer detection rates in larger prostates [9].

**CONCLUSION:**

It is concluded that there is an inverse association of prostate volume with the incidence and biological aggressiveness of PCa. Data from this study and the outlined discussion should encourage other clinicians and investigators to further explore the relationship between prostate volume and the incidence and aggressiveness of PCa, to further investigate this phenomenon.

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