

Prevalence of Hypertension among Prostate Cancer Patients: A Hospital Based Study

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Abstract

Original Research Article

Background: Prostate cancer is a global health problem affecting men in their advancing years. Both prostate and hypertension incidence increases with age. Human and animal studies have attempted to explain an association between the two disease entities. The interplay of androgen activity has been strongly suggested. The aim of this study was to evaluate the prevalence of hypertension among prostate cancer patients. **Materials and methods:** One hundred and nineteen (119) patients with prostate cancer were studied. Information retrieved from their case notes included biodata, clinical, laboratory, imaging and prostate biopsy results. Hypertensive men were selected based on preceding history of hypertension or absence of such history for non-hypertensives. **Results:** mean age of patients was 68.68 ± 8.38 years ranging from 48 to 91 years. Non-hypertensives were slightly older than hypertensive men (Table 1). Mean Gleason score was 7.97 ± 1.15 and mean prostate specific antigen (PSA) was 56.20 ± 37.30 ng/ml. Most men were in their 8th decade of life (table 2i). The prevalence of hypertension was 43.7%. Most of them had prostate specific antigen in excess of 10.0ng/ml and Gleason score of 9. Hypertensive men were also associated with slightly higher Gleason score and prostate specific antigen values. **Conclusion:** Evaluation of hypertensive men should include a full urological assessment to diagnose those at increased risk of prostate cancer bearing in mind documented high prevalences associated with both disease conditions.

Keywords: Hypertension, Prostate cancer, Prevalence.

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INTRODUCTION

Prostate cancer (Pca) is a global health problem affecting men in their advancing years. Well documented risk factors include advancing age, African-American race and positive family history of Pca. Recently studied risk factors include hypertension, obesity and diabetes mellitus [1]. In a Norwegian study of 29,364 men, it was reported that hypertension was associated with increased incidence of Pca [2]. Even men in the highest quartile of systolic blood pressure (> 150 mmHg) were shown to have a greater risk of Pca than others [3]. Other studies also documented poorer prognosis in surgically treated Pca patients with background hypertension [4]. The exact mechanism is not known. However, human and animal studies have revealed some associations which will need further studies to unravel the exact links. Reckelhoff *et al.*, [5] documented that male rats have been known to have higher blood pressure (BP) than female rats and thought this could be related to androgen levels in the males. Generally known, is the location of androgen receptor

(AR) in the proximal tubules of the kidneys. Binding of androgen to these receptors increase sodium re-absorption via angiotensin II or androgen-mediated stimulation of aldosterone and water retention. In addition to this association, Schunkert *et al.*, [6] reported a positive correlation between serum levels of aldosterone, dihydroepiandrosterone (DEA) sulfate (metabolite of testosterone) and BP in hypertensive men. Comparative studies also documented higher aldosterone levels and BP in men than women [7]. Mao *et al.*, [8] in their work with hypertensive men receiving rennin-angiotensin (RA) inhibitors inferred a reduced risk of prostate cancer (RR 0.92% CI 0.87-0.98). Similarly, medical or surgical castration has been known to slow the progression of Pca. These findings suggest that androgens may play an important role in the causation of both hypertension and prostate cancer.

The aim of this study was to evaluate the prevalence of hypertension among prostate cancer patients.

MATERIALS AND METHODS

This was a retrospective study of one hundred and nineteen (119) prostate cancer patients who underwent evaluation and treatment in the University of Uyo Teaching Hospital, Uyo between January 2019 and December 2021. Their case notes were retrieved from the Health Information Department of the Hospital and relevant data retrieved. These included patient's biodata, history of lower urinary tract symptoms and complications due to the disease. Findings on general physical examination and digital rectal examination of the prostate were also documented. Prostate biopsy results with grades of tumor and Gleason score were noted. Other findings on abdominopelvic and transrectal ultrasound examination of the prostate, fasting blood sugar, renal function test, PSA and full blood count were recorded. Hypertensive men were selected based on preceding medical history of hypertension or absence of such history for non-hypertensives. Age of patients was categorized at intervals of 10 into groups 1 to 6 represented by 40-49, 50-59, 60-69, 70-79, 80-89, 90 years and above respectively. Categorization of PSA was done as follows; <4.0ng/ml in group 1, 4-10 ng/ml in group 2 and >10ng/ml in group 3. Grading of Pca utilizing the international society of urological pathologist (ISUP) grading system into aggressive and non-aggressive tumours were as follows; groups 3-5 and 1-2 respectively.

All relevant data were entered into proforma sheets designed for this study and inputed into the

statistical package for social sciences (SPSS) version 20.0 and used for analysis.

Exclusion Criteria

Exclusion criteria were diagnosis of any prostate lesion other than Pca, patients with anorectal and bladder cancers, men with incomplete clinical, laboratory, imaging and histopathological information.

Statistical Analysis

Frequency and percentages for categorical variables were calculated while continuous variables were expressed as means and standard deviations. Student's T-Test was used to assess the difference in mean age between hypertensives and non-hypertensives.

RESULTS

We evaluated 119 prostate cancer patients who met the inclusion criteria. The mean age was 68.68±8.38 years ranging from 48 to 91 years. Hypertensive men were slightly younger than non-hypertensives, although this was not statistically significant P>0.05 (Table 1). Mean Gleason Score was 7.9±71.15 ranging from 5 to 10 for all patients while mean PSA was 56.20±37.30ng/ml ranging from 6.90 to 185.70ng/ml. Most men were in their 8th decade of life (Table 2). The prevalence of hypertension was 43.7% (Table 3). Hypertensive men were also associated with slightly higher Gleason score and prostate specific antigen values. Among PSA categories, most patients (94.1%) had PSA in excess of 10ng/ml (Table 4), and Gleason Score of 9 predominated Table (5).

Table 1: Means and standard deviation for continuous variables

Variable	Means	Std	Min	Max
Age (Years)	68.68	8.38	48	91
Gleason Score	7.97	1.15	5	10
PSA (ng/ml)	56.20	37.30	6.90	185.70
Age (Hypertensives)	68.54	7.80		
Age (Non-hypertensives)	68.79	8.86	T-test =	P>0.05

Table 2: Frequency of categorical variables

Variable	Frequency (n)	Valid %	Cumulative %
Table (2i) Age:			
40 – 49	1	0.8	0.8
50 – 59	14	11.8	12.6
60 – 69	44	37.0	49.6
70 – 79	50	42.0	91.6
80 – 89	9	7.6	99.2
90 and above	1	0.8	100.0
	119	100.0	

Table 3: Hypertensives and Non-hypertensives

	Frequency (n)	Valid %	Cumulative %
Hypertensives	52	*43.7	43.7
Non-hypertensives	67	56.3	100.0
	119	100.0	

*** Prevalence of hypertension is 43.7%**

Table 4: PSA categories

Variable	Frequency (n)	Valid %	Cumulative %
<4ng/ml	0	0.0	0.0
4 –10ng/ml	7	5.9	5.9
>10ng/ml	112	94.1	100.0
	119	100	

Table 5: Gleason Score Categories

Variable	Frequency (n)	Valid %	Cumulative %
5	1	0.8	0.8
6	11	9.2	10.1
7	34	28.6	38.7
8	24	20.2	58.8
9	42	35.3	94.1
10	7	5.9	100.0
	119	100.0	

DISCUSSION

The exact cause of prostate cancer remains unknown, however, well documented risk factors include advancing age, race and a positive family history. Many authors have made attempts to expand this list to include components of the metabolic syndrome for which hypertension is an integral element [9-11]. Flint men's health study conducted among African – American men in 2007 concluded that both abdominal obesity and hypertension were independently related to incident Pca [9]. Hypertensive patients were more likely to experience biochemical recurrence in the post radical prostatectomy period in separate studies by Asmar *et al* [12] and Post *et al* [12]. Both human and animal studies have documented a possible link between androgen activity and the incidence and prevalence of Pca and hypertension [5-8]. Touyz *et al.*, attempted to explore a possible association between the two disease entities and postulated that hypertension is associated with chronic inflammation and excess reactive oxygen species which could promote prostate cancer cell growth [14]. Moreover, epidemiological studies demonstrated a positive correlation between prostate inflammation (prostatitis) and sexually transmitted infection with prostate cancer [15]. With these body of knowledge, it is apparent that hypertension could be one of the risk factors for Pca. However, further research and multicentre collaborations will be needed to establish the true link.

The mean age of men in this study was 68.68 + 8.38 years ranging from 48 to 91 years. A similar mean age had been previously documented in this centre in a study of Pca patient and in another similar Nigerian study [16, 17]. Majority of the patients were in their 8th decade of life consistent with above study in this centre [16]. Comparable study in Jamaica [17] also reported majority of their men in same decade of life supporting the fact that advancing age is a strong risk factor worldwide aside from race and a family history.

A greater number of patients had PSA in excess of 10.0ng/ml and on histology reports; Gleason score of 9 predominated. Both variables denote aggressive and poorly differentiated tumors. Similar data was also reported in this part of the country [18]. This may probably be due to genetic component of the black race and of course late presentation for evaluation.

Both Pca and hypertension incidence increases with age. Non-hypertensives were slightly older than hypertensives ($P > 0.05$) (Table 1). Hypertensive men are more likely to be diagnosed early with Pca especially in this era of PSA testing because they are usually exposed to various investigations among which is PSA than the non-hypertensives who may not have the need to visit the hospital for care. The prevalence of hypertension in this study is 43.7%, far higher than that reported in urban dwelling adult citizens in same South-South Nigeria (28.6%) [19]. Similar study in Western Nigeria reported 20.8% prevalence of hypertension [20]. These figures represent both males and females and the number of female patients in both studies may dilute the expected prevalence albeit small, whereas in our study, all patients were males. Higher values had been recorded among African American hypertensive men (73%) who were also being evaluated for Pca and 72% in the white population [21]. This was also higher than 29% prevalence of hypertension in their general population (males and females) according to reports of the 2016 US centres for disease control and prevention [22]. The high prevalence of hypertension among prostate cancer patients in this study and the American study support a possible association between the two disease conditions. The mechanisms involved may be related to androgen mediated events and chronic inflammatory state reported in both diseases. Higher prevalence in the American Study may be due to increased awareness and disease surveillance in their population than what obtains here. As a follow up, levels of tumor aggressiveness in such countries may also be low unlike what is documented in this study. Hypertensive men were associated with slightly higher

Gleason score and prostate specific antigen values. These are also markers for aggressive prostate cancer. It follows that, evaluation of hypertensive men should include full urological workup to diagnose early disease with suitable curative intent.

CONCLUSION

Documented evidences show that the prevalence of hypertension among prostate cancer patients is consistently high in both black and white populations. Hypertension may be a risk factor for prostate cancer.

Authors Contribution:

Elijah Asuquo Udoh: Substantial contributions to conception and design, Acquisition of data, Drafting the article, revising it critically for important intellectual content, data analysis and Final approval of the version to be published.

Ifiok Udo Essiet: Substantial contributions to conception and design, revising it critically for important intellectual content and final approval of the version to be published.

Anthony Joseph Usoro: Substantial contributions to conception and design, revising it critically for important intellectual content and final approval of the version to be published.

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