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**Serum Uric acid and Renal function determinants in Prostate Cancer Patients attending the University of Calabar Teaching Hospital, Calabar, Nigeria.**Bassey, I.E. <sup>1\*</sup>, Akpan, U.O. <sup>1</sup>, Isong, I.K.P <sup>1</sup>, Esiere, K.S.U. <sup>1</sup>, Nehemiah, E.D. <sup>1</sup>, Gladstone, E.U. <sup>1</sup>, Omini, I.U. <sup>1</sup>, Udoh, A.E. <sup>1</sup>*Department of Medical Laboratory Science, Faculty of Allied Medical Sciences, College of Medical Sciences, University of Calabar, Calabar, Cross River State, Nigeria*<sup>1</sup>.*Author for Correspondence \*:* iyantui@yahoo.com/+234-803-547-2034**Abstract**

Renal function decline in prostate cancer patients may result in a double burden of disease. It is therefore necessary to look for economical and accurate markers to monitor the progress of renal damage and to avoid complications arising from renal damage. This study investigated the changes in urinary protein, serum uric acid, creatinine and uric acid clearance in prostate cancer patients. Seventy participants were consecutively recruited and comprised 20 treatment-naïve and 20 androgen deprivation therapy (ADT) treated-prostate cancer patients and 30 controls. Serum and urine creatinine, uric acid and urine protein were determined using colorimetric methods, prostate specific antigen by ELISA and creatinine, uric acid clearance and total urinary protein output were calculated. Data were analyzed by analysis of variance at  $p < 0.05$ . Total urine protein output was significantly higher in both treatment naïve ( $p = 0.001$ ) and ADT-treated prostate cancer patients ( $p = 0.039$ ) compared to controls. The treatment naïve prostate cancer patients had significantly higher serum uric acid compared to both ADT-treated prostate cancer patients ( $p = 0.037$ ) and controls ( $p = 0.012$ ) while the ADT-treated prostate cancer patients had significantly higher ( $p = 0.012$ ) systolic blood pressure compared to controls. There were no significant variations ( $p > 0.05$ ) in creatinine and uric acid clearance among the groups. Since only proteinuria predicted renal functional impairment in the prostate cancer patients, it is suggested that proteinuria may be a better predictor of renal functional decline, for these groups of patients. We therefore advocate that it be included in routine evaluation during management of prostate cancer patients.

**Keywords:** Renal function, prostate cancer, ADT, creatinine clearance, Black men**Introduction**

Prostate cancer is the foremost cancer in men with increasing incidence and morbidity in males of African ancestry (Akinremi *et al.*, 2011). Prostate cancer is a disease in which cancer develops in the prostate gland a part of the male reproductive system. It occurs when the cells of the prostate gland mutate and begin to multiply out of control. Patients often present at a late stage with complications (Akinremi *et al.*, 2014). However, compared with other forms of cancer, the prognosis of prostate cancer is favorable, with 5 years relative survival after diagnosis estimated at 88%, if diagnosed early and managed properly.

Prostate cancer may cause pain and difficulty in urinating, problems during sexual intercourse, erectile dysfunction and other symptoms. However, these symptoms are present only in an advanced stage of the disease (Miller *et al.*, 2003) which is very common in the African setting (Bassey *et al.*, 2019).

Patients with prostate cancer may experience impairment renal function owing to age, disease-related factors as well as treatments that are nephrotoxic thereby resulting in a double burden of disease for the prostate cancer patient. Renal insufficiency has been reported to be associated with reduced overall survival and increased cancer-related mortality (Launay-Vacheret *et al.*, 2016). It is therefore necessary to look for economical and accurate markers to monitor the progress of renal damage and avoid

complications arising from renal damage. The number of published urinary biomarker studies is increasing rapidly. Most of these studies have looked at the soluble urine protein fraction, focusing on the identification of potential biomarkers in diseases of the urogenital tract (Thomas *et al.*, 2016; Arixa *et al.*, 2015).

Proteinuria has been shown to be an independent risk factor for the progression of kidney disease rather than simply being a marker for glomerular function. The assessment of proteinuria is clinically and diagnostically an important index in renal function generally and particularly that of the nephrons (Sandilands *et al.*, 2013).

In clinical practice, creatinine clearance is used to measure glomerular filtration rate (GFR). Creatinine is produced naturally by the body (Gounden and Jialal, 2019). It is filtered freely by the glomerulus. However, it is also secreted actively by the renal tubules in very small amounts such that creatinine clearance overestimates actual GFR by 10-20%. This margin of error is acceptable considering the ease with which creatinine clearance is measured (Stevens *et al.*, 2006).

Hyperuricemia has been associated with the development of acute oligonuric renal failure caused by renal tubular obstruction by urates and uric acid crystal. This is observed mostly in settings of malignancy (Hahn *et al.*, 2017; Vargas-Santos and Neogi, 2017). This study investigated the changes in urinary protein, serum uric acid and creatinine and uric acid clearance in prostate cancer patients

## Materials and Methods

### Subject Selection

A total of 70 male participants of Nigerian descent resident in Calabar were consecutively recruited into this case-control study. Forty prostate cancer patients enrolled in this study were attending the Urology Clinic in the University of Calabar Teaching Hospital, Calabar. Thirty age-matched apparently healthy men without prostate cancer or benign prostatic hyperplasia were recruited as controls. Patients with prostate cancer were divided into two subcategories: those who had received no

treatment (treatment-naive patients with prostate cancer) and those who had received Androgen Deprivation Therapy (ADT-treated patients with prostate cancer). Treatment modalities included use of medical castration (biclutamide, GnRH, and flutamide) and/or surgical orchiectomy. Ethical clearance was obtained from the University of Calabar teaching hospital research ethical committee. The nature and purpose of the research was explained to all participants and written informed consent obtained.

### Inclusion and Exclusion criteria

All the subjects were male and above forty years. Subjects with benign prostatic hyperplasia were excluded.

### Measurement of anthropometric indices and blood pressure

Their height and weight were measured with a stadiometer and weighing balance respectively. Body mass index was calculated as a ratio of weight to height squared. The blood pressure was measured on the right arm with a mercury sphygmomanometer (cuff size 12.5 x 40 cm) with the patient in a seated position and after a 5 minutes rest. The systolic and diastolic blood pressures were recorded.

### Sample collection

Five milliliters of venous blood were collected from both subjects and controls under aseptic conditions and dispensed into a dry plain container. The blood samples were allowed to clot and retract and were spun at 3000rpm for 5 minutes. The separated serum was dispensed into appropriately labeled containers and stored frozen. 24-hour urine samples were also collected alongside the blood specimen. The urine samples were collected into a 4-liter container containing 10ml of concentrated Hydrochloric acid which served as a preservative. The urine samples-(24 hours) were measured and the total volume recorded. Aliquots were collected in universal containers, appropriately labeled and stored frozen.

### Determination of Analytes

Determination of Prostate-specific antigen (PSA) was done with an enzyme-linked immunosorbent assay kit obtained from Syntro

Bioresearch (Carlsbad, CA U.S.A). Serum and urine creatinine were determined using photometric colorimetric test for kinetic measurements. The reagent kit obtained from Germany is a modified method of Bartels and Böhmer (1973). Serum and urine uric acid was determined using kits obtained from Giese Diagnostics (Italy). Urine protein was determined using the 3% Trichloroacetic acid turbidometric method. After obtaining serum and urine creatinine values, creatinine clearance was calculated using the formula:

$$\text{Creatinine clearance (ml/min)} = (U \times V) / P$$

Where U = Urine creatinine concentration ( $\mu\text{mol/l}$ )

V = Urine Volume (ml)

P = Serum creatinine concentration ( $\mu\text{mol/l}$ ).

The uric acid clearance was calculated from the total urine volume, serum uric acid concentration and the urine uric acid concentration using the equation:

$$\text{Uric acid clearance (ml/min)} = (U \times V) / P$$

Where P = plasma concentration of uric acid in mg/100ml.

U = Urine concentration of uric acid in mg/100ml.

V = the volume of the 24-hour urine in ml.

### Statistical Analysis

Statistical analysis was performed using PAW statistic 18, a statistical package from SPSS (Chicago, IL). Results are expressed as mean  $\pm$  standard deviation. Student's t-test and analysis of variance were used to analyze the data. Post hoc analysis was performed using least significant difference. The level of significance was set at 95% CI, where a probability value of  $p < 0.05$  was regarded as statistically significant.

### Results

A comparison of age, blood pressures, body mass index, prostate specific antigen, total urine protein output, serum uric acid, uric acid clearance and total uric acid output in prostate cancer patients and controls is shown in Table 1. The mean levels of systolic blood pressure ( $p=0.020$ ), prostate specific antigen ( $p=0.0001$ ) and total urine protein output ( $p=0.0001$ ) of the prostate cancer patients were significantly higher than those of the controls. There were no significant differences ( $p > 0.05$ ) in other parameters between the groups.

Table 2 shows a comparison of blood pressures, body mass index, prostate specific antigen, total urine protein output, serum uric acid, uric acid clearance and total uric acid output in treatment naïve, ADT-treated prostate cancer patients and controls. There was a significant variation in the mean levels of systolic blood pressure ( $p=0.039$ ), prostate specific antigen ( $p=0.0001$ ) and total urine protein output ( $p=0.003$ ) and serum uric acid ( $p=0.039$ ) among the groups. There were however no significant variations ( $p > 0.05$ ) in the other parameters among the groups.

A Post hoc analysis using least significant difference (LSD) showed that the mean values of PSA and total urine protein output were significantly higher in both treatment- naïve ( $p=0.001$ ) and ADT-treated prostate cancer patients ( $p=0.039$ ) compared to controls. The treatment- naïve prostate cancer patients had significantly higher serum uric acid compared to both ADT-treated prostate cancer patients ( $p=0.037$ ) and controls ( $p=0.012$ ) while the ADT-treated prostate cancer patients had significantly higher ( $p=0.012$ ) systolic blood pressure compared to controls (Table 3). There were no significant correlations between PSA and any of the parameters in any of the groups.

**Table 1: Age, blood pressures, body mass index, prostate specific antigen, total urine protein output, serum uric acid, uric acid clearance and total uric acid output in prostate cancer patients and controls**

Parameters	Prostate cancer n=40	Controls n=30	Calc. t	p-value
Age (years)	67.5±9.79	64.3±10.47	1.310	0.195
Systolic BP (mmHg)	145.3±18.36	134.0±21.15	2.384	0.020*
Diastolic BP (mmHg)	88.1±12.28	85.8±12.60	0.764	0.448
Body mass index (kg/m <sup>2</sup> )	24.7±4.06	25.5±3.26	0.837	0.406
Prostate specific antigen (ng/ml)	28.0±24.83	1.7±1.33	6.691	0.0001*
Total urine protein output (g/24hrs)	0.40±0.41	0.14±0.11	3.744	0.0001*
Serum uric acid (mg/dl)	7.1±2.78	6.1±1.88	1.669	0.100
Total uric acid output (g/24hr)	0.19±0.14	0.22±0.20	0.805	0.424
Uric acid clearance (ml/min)	2.41±2.32	2.56±1.92	0.296	0.768
Creatinine clearance (ml/min)	119.4±65.12	117.5±39.43	0.148	0.883

*Results expressed as Mean ± SD \* Significant at p<0.05*

**Table 2: Blood pressures, body mass index, prostate specific antigen, total urine protein output, serum uric acid, uric acid clearance and total uric acid output in treatment naïve, ADT-treated prostate cancer patients and controls**

Parameters	Treatment	ADT-treated CaP n=20	Controls n=30	Calc. F	p-value
Systolic BP (mmHg)	142.0±18.53	148.5±17.85	134.0±21.15	3.399	0.039*
Diastolic BP (mmHg)	84.5±13.17	91.8±10.42	85.8±12.60	2.070	0.134
Body mass index (kg/m <sup>2</sup> )	24.5±4.11	24.9±4.10	25.5±3.26	0.397	0.674
Prostate specific antigen (ng/ml)	46.9±19.12	9.05±12.10	1.7±1.33	89.742	0.0001*
Total urine protein output (g/24hrs)	0.46±0.56	0.34±0.17	0.14±0.11	6.232	0.003*
Serum uric acid (mg/dl)	7.9±2.45	6.3±2.92	6.1±1.88	3.725	0.029*
Total uric acid output (g/24hr)	0.19±0.14	0.19±0.14	0.22±0.20	0.319	0.728
Uric acid clearance (ml/min)	2.33±2.59	2.49±2.07	2.56±1.92	0.070	0.932
Creatinine clearance (ml/min)	113.1±66.04	124.7±65.56	117.5±39.43	0.210	0.811

*Results expressed as Mean ± SD \* Significant at p<0.05*

**Table 3: Post hoc analysis of blood pressures, prostate specific antigen, total urine protein output and serum uric acid, in treatment naïve, ADT-treated prostate cancer patients and controls**

Parameter	Groups		Mean Difference	Std Error	p-value
	Treatment naïve CaP n=20	ADT-treated CaP n=20			
Systolic BP (mmHg)	142.0±18.53	148.5±17.85	-6.500	6.175	0.296
Prostate specific antigen (ng/ml)	46.9±19.12	9.05±12.10	37.835	3.820	0.0001*
Total urine protein output (g/24hrs)	0.46±0.56	0.34±0.17	0.123	0.102	0.230
Serum uric acid (mg/dl)	7.9±2.45	6.3±2.92	1.597	0.751	0.037*
Parameter	Treatment naïve CaP n=20	Controls n=30	Mean Difference	Std Error	p-value
	Systolic BP (mmHg)	142.0±18.53			
Prostate specific antigen (ng/ml)	46.9±19.12	1.7±1.33	45.232	3.487	0.0001*
Total urine protein output (g/24hrs)	0.46±0.56	0.14±0.11	0.317	0.093	0.001*
Serum uric acid (mg/dl)	7.9±2.45	6.1±1.88	1.781	0.686	0.012*
Parameter	ADT-treated CaP n=20	Controls n=30	Mean Difference	Std Error	p-value
	Systolic BP (mmHg)	148.5±17.85			
Prostate specific antigen (ng/ml)	9.05±12.10	1.7±1.33	7.397	3.487	0.038*
Total urine protein output (g/24hrs)	0.34±0.17	0.14±0.11	0.20	0.093	0.039*
Serum uric acid (mg/dl)	6.3±2.92	6.1±1.88	0.184	0.686	0.790

**Results expressed as Mean ± SD** \* **Significant at p<0.05**

**Discussion**

Urinary profile often changes in malignant diseases sometimes due to renal function decline leading to a double burden of disease in those groups of patients. This investigated the changes in urinary protein, serum uric acid and creatinine and uric acid clearance in prostate cancer patients.

The significantly higher levels of total urine protein output seen in the treatment naïve cases and ADT- treated cases shows that prostate carcinoma is associated with or may cause derangement in renal function. This derangement is may be due to acute urinary retention which results from increased resistance to flow of urine caused by the prostatic enlargement (Muhammed and Abubakar, 2012). When the prostate gets enlarged, the layer of

tissue surrounding it stops it from expanding causing a press against the urethra (Miyazato *et al.*, 2013). Consequently, the bladder wall becomes thicker and irritable and begins to contract even when it contains small amounts of urine triggering frequent urination and urgency (Miyazato *et al.*, 2013). In due course, the bladder weakens and loses its ability to empty itself resulting in retention of urine (Speakman and Cheng, 2014). Complications arising from this include kidney stones formation, increase in bacterial growth and thus inflammation (Khan *et al.*, 2016; Muruganandham *et al.*, 2007). If this urine backs up into the kidneys, with time, it can damage the kidneys leading to derangement in renal function, thus the appearance of abnormally high protein in urine. Interestingly, the renal function impairment observed in our study may be attributed to derangement to the

glomerular filtration rather than the secretory or absorptive processes of the kidney as shown by higher total urinary output in both the treatment naïve and ADT-treated prostate cancer patients and no change in either creatinine or uric acid clearance, despite the significantly higher levels of uric acid in the treatment naïve prostate cancer patients. This also suggests that proteinuria may be a better predictor of renal functional decline for these groups of patients.

Previous reports (Gandaglia *et al.*, 2014 and Lapi *et al.*, 2013) indicate that treatment with androgen deprivation therapy may worsen renal function. However, that was not the case with our study as the ADT-treated group had lower total protein out albeit not significantly ( $p > 0.05$ ) than the treatment naïve group and comparable creatinine and uric acid clearance with both the treatment naïve and control groups. This was in spite of the fact that the ADT-treated group had higher systolic blood pressure than the controls. Increased blood pressures have been associated with proteinuria in other studies (Bassey *et al.*, 2019). Our findings agree with those of Koji *et al.* (2016) whose study among Japanese men showed that ADT did not impair renal function in patients with prostate cancer.

The higher levels of uric acid observed in the treatment naïve prostate cancer patients when compared with the ADT-treated and control groups may be due to untreated cancer. Malignancy can cause hyperuricemia through cancer related cell death (Wang *et al.*, 2015). Elevated serum uric acid, the dominant monosodium form of uric acid at physiological pH, was found to exhibit strong statistical association with increased premature cancer death in both men and women (Fini *et al.*, 2012) suggesting a more complex role for UA in cancer biology than that of a general antioxidant. This finding differs from the findings in previous reports (Benli *et al.*, 2018 and Park *et al.*, 2018) where the uric acid levels measured in serum of patients with a prostate cancer diagnosis were reduced compared to the control group. This they attributed to uric acid been consumed during neutralization of increasing oxidant compounds in the inflammatory environment (Benli *et al.*, 2018; Park *et al.*, 2018).

In view of the fact that only proteinuria predicted renal functional impairment in the prostate cancer patients, we conclude that proteinuria may be a better predictor of renal functional decline, for these groups of patients. We therefore advocate that it be included in routine evaluation during management of prostate cancer patients.

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