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ORIGINAL ARTICLE

EFFECTS OF ANDROGEN DEPRIVATION THERAPY ON FASTING BLOOD GLUCOSE IN PROSTATE CANCER PATIENTS AT A TEACHING HOSPITAL IN SOUTHWESTERN NIGERIA - A PILOT STUDY

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ABSTRACT

Background: Prostate cancer is the most common cancer and the leading cause of cancer death in Nigerian men. There are many modalities of treatment of prostate cancer, among them is hormonal treatment with the use of androgen-deprivation therapy (ADT), and this has been reported to have effects on the blood glucose level, causing hyperglycaemia.

Objective: To determine the effects of three-month administration of ADT on fasting blood glucose in prostate cancer patients.

Methods: This prospective pilot study involved 15 histologically diagnosed prostate cancer patients who were not diabetic. Baseline measurements of fasting blood glucose were done before the commencement of ADT. Follow-up assessments occurred after 3 months of ADT administration. Data was analysed using logistic regression.

Result: Fifteen patients who met the study's selection criteria were recruited. The age of the patients ranged from 56 to 88 years, with a median age of 70 years. There was a 5.8% increase in the mean FBS from 95.1mg/dl (±11.2mg/dl) at baseline to 100.6mg/dl (±11.8mg/dl) at 3 months (p=0.171). Subgroup analysis revealed a statistically significant 6.1% increase in the median FBS from 90.0mg/dl (range: 80 – 114mg/dl) at baseline to 95.5mg/dl (range:88 – 119mg/dl) at 3 months (p=0.027) among patients with hypertension, however, the increase in the mean was not statistically significant (p=0.09).

Conclusion: This pilot study suggested that ADT therapy may not be associated with hyperglycaemia after 3 months. Further research will further provide insight into the risks associated with men on ADT developing hyperglycaemia and the timing of these metabolic changes in the course of their treatment. This will help in the early identification and management of this complication.

Keywords: ADT, Prostate cancer, hyperglycaemia, fasting blood glucose.

INTRODUCTION

Prostate cancer is the second most common cancer in men worldwide and the fifth leading cause of cancer death in men.[1] In Nigeria, it is the most common cancer and the leading cause of cancer death in men.[2] There are many modalities of treatment in early and metastatic stages of prostate cancer, among them is the hormonal treatment with the use of androgen-deprivation therapy (ADT) that renders the men hypogonadal. Male hypogonadism is associated with an unfavourable metabolic profile.[3]

Diabetes and abnormalities of glucose metabolism are common in the age group of people susceptible to the development of prostate cancer. ADT has been shown to affect fasting glucose levels, insulin levels, and insulin resistance.[4] It is therefore important to understand the relationships between ADT and glucose metabolism to forestall any untoward effects in patients undergoing treatment.

Previous studies on ADT and hyperglycaemia were predominantly conducted in the Caucasian population and have consistently demonstrated that ADT is linked with hyperglycaemia.[5] However, one cross-sectional study on black African men showed that Androgen deprivation therapy does not result in elevated blood glucose.[6,7] This finding requires further validation as the relationship between ADT use among indigenous black African men and hyperglycaemia has not been extensively explored.

In this pilot study, we sought to investigate the relationship between ADT use and the risk of developing hyperglycaemia after three months among Nigerian prostate cancer patients.

MATERIALS AND METHODS

Study Design and Study Population

This pilot study was a prospective investigation conducted over four months at the University College Hospital (UCH), Ibadan, Nigeria. Ethical approval with the number UI/EC/23/0673 was obtained from the institutional Ethical Committee before the commencement of this study.

It was conducted among 15 randomly selected, histologically diagnosed prostate cancer patients to be treated with ADT attending the Oncology clinics of the University of Ibadan, Nigeria. The ADT of interest included gonadotropin-releasing hormone (GnRH), bilateral orchiectomy and oral anti-androgens, which are commonly administered in the hospital.

The nature of the study was explained to the patients, and Informed consent was obtained from them before enrollment. Patients aged 50 and above were enrolled. Patients with a previous or current history of derangement in glucose metabolism were excluded.

Data collection procedure

Clinical data, including socio-demographic and disease data, were obtained using an interviewer-administered questionnaire.

Fasting blood glucose assay

A fasting blood glucose was carried out on the patients before ADT was commenced as a baseline and at the 3rd month of being on ADT.

Blood samples were aseptically collected after an overnight fast by pricking the patients '

fingertips with a lancet at baseline, then at 3 months of being on ADT. Fasting blood glucose levels of >126 mg/dL (7 mmol/L) were considered high. [6]

Data analysis

Data was analysed using SPSS V 23: Descriptive statistics were used to present socio-demographic and clinical data and plasma glucose values. The proportion of participants with high fasting blood glucose values (> 126mg/dL) was calculated. Paired t-test and Wilcoxon signed rank test were used to compare mean and median FBS at baseline and three months, respectively. Student t-test and Mann-Whitey U test were used to compare mean and median FBS across two groups, respectively, while ANOVA and Kruskal-Wallis test were used to compare mean and median FBS across three groups, respectively. The level of significance was set at ≤ 0.05.

RESULTS

Sociodemographic and clinicopathologic characteristics are shown in Tables 1 and 2, respectively. Fifteen patients were recruited with a median age of 70 years (range: 56 – 88 years). Forty per cent (40%) of the patients were hypertensive, while 53.3% and 13.3% were overweight and obese, respectively. The median PSA at diagnosis was 17.9ng/ml (range: 5 – 3289ng/ml).

Forty per cent (40%) of the patients had initial PSA >20ng/ml. The most common T stage, Gleason score and risk group were T2 (73.3%), 3+3 (40.0%) and high-risk group (60.0%), respectively, as seen in Figure 1. All patients had ADT, and Bicalutamide was the most frequently used ADT (73.3%).

There was a 5.8% increase in the mean FBS from 95.1mg/dl (±11.2mg/dl) at baseline to 100.6mg/dl (±11.8mg/dl) at three months (p=0.171) [Tables 3 and 4, Figure 1]. Subgroup analysis revealed a statistically significant 6.1% increase in the median FBS from 90.0mg/dl (range: 80 – 114mg/dl) at baseline to 95.5mg/dl (range:88 – 119mg/dl) at three months (p=0.027) among patients with hypertension, however, the increase in the mean was not statistically significant (p=0.09). Also, there was a statistically significant 11.3% increase in the mean FBS from 94.1mg/dl (±13.2mg/dl) at baseline to 104.7mg/dl (±12.3mg/dl) at 3 months (p=0.046) among those with high-risk prostate cancer [Table 4].

Table 1: Sociodemographic Characteristics

Variables	Frequency	Percentage
Age (Years)		
< 65	5	33.3%
≥ 65	10	66.7%
Employment Status		
Employed	6	40.0%
Unemployed	9	60.0%
Hypertension		
Yes	6	40.0%
No	9	60.0%
Body Mass Index		
Underweight	1	6.7%
Normal	2	13.3%
Overweight	8	53.3%
Obese	2	13.3%
Missing	2	13.3%

Table 2: Clinicopathologic Characteristics

Variables	Frequency	Percentage
T Stage		
T2	11	73.3%
T4	4	26.7%
Gleason Score		
3+3	6	40.0%
3+4	2	13.3%
4+3	4	26.7%
4+5	3	20.0%
PSA Range		
<10	4	26.7%
10-20	4	26.7%
>20	6	40.0%
Missing	1	6.7%
Risk Group		
Low	2	13.3%
Intermediate	3	20.0%
High	9	60.0%
Missing	1	6.7%
Treatment		
Orchidectomy	2	13.3%
Zoladex	2	13.3%
Bicalutamide	11	73.3%

Table 3: Comparison of Baseline and 3-Month Fasting Blood Sugar (FBS) Levels Across sociodemographic and clinical characteristics.

Variables	Mean	SD	P-value	Median	Range	P-value
BMI	26.7	5.5		26.3	17.0 - 40.5	
Initial PSA (ng/ml)	431.2	908.9		17.9	5.0 – 3289	
Baseline FBS (mg/dl)	95.1	11.2	0.171	91.0	80.0 – 114.0	0.172
3 months FBS (mg/dl)	100.6	11.8		98.0	83.0 – 119.0	
Age (years)						
< 65						
Baseline FBS (mg/dl)	95.0	12.0	0.439	91.0	80.0 – 110.0	0.345
3 months FBS (mg/dl)	102.2	13.8		109.0	83.0 – 117.0	
≥ 65						
Baseline FBS (mg/dl)	95.1	11.5	0.303	92.0	81.0 – 114.0	0.332
3 months FBS (mg/dl)	99.8	11.4		95.5	88.0 – 119.0	
Hypertension						
Yes						
Baseline FBS (mg/dl)	91.3	11.8	0.090	90.0	80.0 – 114.0	**0.027**
3 months FBS (mg/dl)	100.0	11.7		95.5	88.0 – 119.0	
No						
Baseline FBS (mg/dl)	97.6	10.6	0.573	95.0	81.0 – 110.0	0.678
3 months FBS (mg/dl)	101.0	12.6		100.0	83.0 – 117.0	
BMI						
Underweight						
Baseline FBS (mg/dl)	90.0	-	-	90.0	-	-
3 months FBS (mg/dl)	93.0	-		93.0	-	
Normal						
Baseline FBS (mg/dl)	98.3	10.4	0.597	95.0	90.0 – 110.0	0.593
3 months FBS (mg/dl)	100.3	14.5		93.0	91.0 – 117.0	
Overweight						
Baseline FBS (mg/dl)	98.0	12.4	0.648	99.0	81.0 – 114.0	0.726
3 months FBS (mg/dl)	101.0	13.1		99.0	83.0 – 119.0	
Obese						
Baseline FBS (mg/dl)	83.0	4.2	0.434	83.0	80.0 – 86.0	0.180
3 months FBS (mg/dl)	99.0	14.1		99.0	89.0 – 109.0	

Table 4: Comparison of baseline and 3-month fasting blood sugar (FBS) levels across risk-stratified groups and treatment modalities

Variables	Mean	SD	P-Value	Median	Range	P-Value
Risk Group						
Low Risk						
Baseline FBS (mg/dl)	90.5	0.7	0.395	90.5	90.0 – 91.0	0.180
3 months FBS (mg/dl)	101.0	11.3		101.0	93.0 – 109.0	
Intermediate Risk						
Baseline FBS (mg/dl)	96.3	7.1	0.570	95.0	90.0 – 104.0	0.593
3 months FBS (mg/dl)	90.7	7.5		91.0	83.0 – 98.0	
High Risk						
Baseline FBS (mg/dl)	94.1	13.2	**0.046**	90.0	80.0 – 114.0	**0.05**
3 months FBS (mg/dl)	104.7	12.3		109.0	88.0 – 119.0	
Treatment						
Orchidectomy						
Baseline FBS (mg/dl)	95.5	20.5	0.363	95.5	81.0 – 110.0	0.180
3 months FBS (mg/dl)	115.0	2.8		115.0	113.0 – 117.0	
Zoladex						
Baseline FBS (mg/dl)	90.5	0.7	0.395	90.5	90.0 – 91.0	0.180
3 months FBS (mg/dl)	101.0	11.2		101.0	93.0 – 1O9.0	
Bicalutamide						
Baseline FBS (mg/dl)	95.8	11.3	0.642	94.0	80.0 – 114.0	0.656
3 months FBS (mg/dl)	97.9	11.5		93.0	83.0 – 119.0	

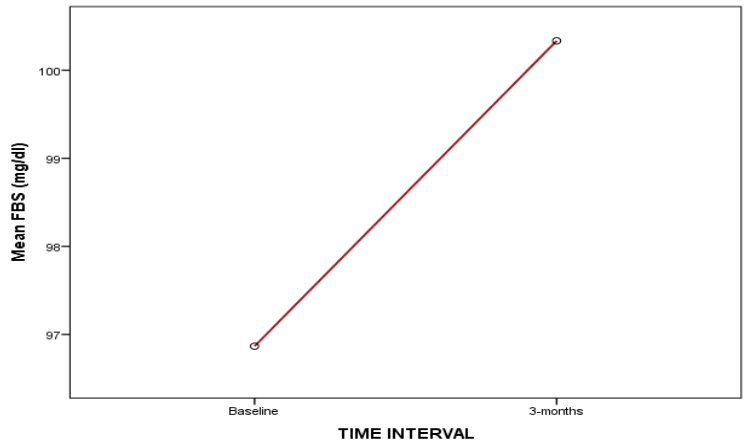

Figure 1: FBS Trend at Baseline and at Three Months

DISCUSSION

We sought to carry out a pilot study to investigate the effect of ADT on fasting blood glucose among Nigerian men before the commencement of ADT and after three months of being on ADT. The median age of patients was 70 years (range: 56 – 88 years). After three months of ADT, the mean FBS increased by 5.8% from 95.1mg/dl (±11.2mg/dl) at baseline to 100.6mg/dl (±11.8mg/dl) at 3 months (p=0.171). This finding was similar to that reported by Smith et al. They reported a 2.2% increase in mean fasting plasma glucose from 93.0mg/dl (±2mg/dl) at baseline to 95.0mg/dl (±2mg/dl) at three months (p=0.200) among prostate cancer patients on ADT.[8] Saglam et al. also reported a 1.0% increase in mean fasting blood glucose in men with prostate cancer on ADT in Turkey, from 101.0mg/dl (±10mg/dl) at baseline to 102.0mg/dl (±9mg/dl) at three months.[9] Another study done by Wong et al revealed a 4.8% increase in mean FBS at six months (p<0.001) and 6.6% at twelve months among Asian men who had ADT (p<0.001).[10] Also in tandem with our findings, a cross-sectional study by Basaria et al. showed that the mean FBS was higher among prostate cancer patients who had ADT (135.0±11.46mg/dl) compared to prostate cancer patients who did not have ADT (99.94±5.0mg/dl) and controls without prostate cancer (99.17±3.9mg/dl) (p=0.002).[11] In contrast, with our finding, Irawan and Warli reported a 1.6% decrease in mean fasting blood glucose from 104.0mg/dl (±14.6mg/dl) at baseline to 102.3mg/dl (±15.8mg/dl) at three months among prostate cancer patients who had ADT in Indonesia.[12] The inconsistencies observed across studies may be attributed to study duration, sample size, or the specific type of ADT used. Also, a cross-sectional study carried out among Nigerian men by Bassey et al. showed similar mean fasting plasma glucose in patients with prostate cancer who had ADT (4.5±1.5mmol/l) compared to treatment-naïve prostate cancer patients (5.08±2.4mmol/l) and controls without prostate cancer (4.60±2.34mmol/l) (p=0.336).[7] ADT, which is a treatment option for prostate cancer, has been associated with insulin resistance and hyperglycaemia.[11,13,] This can be explained by low testosterone levels or blockage of testosterone effects following ADT use, which has been linked to metabolic syndrome.[14,15] Suggested molecular pathways by which androgen signaling affects metabolic controls involve multiple factors and signaling among tissues targeted by insulin.[16]

Subgroup analysis from our study revealed that among participants with hypertension, there was a 6.1% increase in median FBS after three months, which was statistically significant (p=0.027). However, the 9.5% increase in the mean FBS was not statistically significant (p=0.09). Basaria et al demonstrated that the prevalence of hypertension was higher among prostate cancer patients who had ADT (45%) compared to those who did not (28%) and controls without prostate cancer (40%) (p=0.53).[4] This finding could be because hypertension is a component of metabolic syndrome, which has been demonstrated to have a higher prevalence among patients with prostate cancer who had ADT (55%) compared to prostate cancer patients who did not receive ADT (22%) and controls without prostate cancer (20%) (p=0.03).[4] Also, Lauren and Hal demonstrated an association between prostate cancer and components of metabolic syndrome, such as hypertension and also suggested that clinical prostate cancer is a component of metabolic syndrome.[17]

This result also aligns with findings by Smith et al and Christos V. Rizos et al, who noted that

ADT-induced metabolic alterations were more common in patients with pre-existing cardiovascular risk factors, including hypertension. A possible explanation is that hypertensive patients often have underlying insulin resistance, which ADT may exacerbate. In addition, some hypertensive medications, such as beta-blockers and diuretics, may contribute to glucose dysregulation.[18]

In contrast to these findings, a study by Chi-Fai Ng et al. found no significant relationship between hypertension and ADT-induced metabolic changes. It was proposed that the discrepancies could be related to the sample size, study designs, racial differences, short follow-up period and selection bias.[19,20]

Among patients with high-risk prostate cancer, there was a significant increase in mean FBS (p=0.046) with an 11.3% increase from 94.1mg/dl (±13.2mg/dl) at baseline to 104.7mg/dl (±12.3mg/dl) at three months. No significant increase was seen in other risk groups. This may suggest that the severity of prostate cancer may contribute to the metabolic effects of ADT.

A study by Hammarsten and Högstedt demonstrated a higher prevalence of non-insulin-dependent diabetes mellitus among prostate cancer patients with high-grade disease compared to those with low-grade disease (p=0.356).[17] Their findings, which suggest that the prevalence of metabolic syndrome is higher among prostate cancer patients with high-grade disease, could explain the statistically significant increase in mean FBS among patients with high-risk prostate cancer in our study.[17]

Conversely, not every study reports this association. Some have indicated that the metabolic effects of ADT are independent of prostate cancer risk stratification.[21] This could be due to variations in the duration of ADT, patient selection, and differences in baseline metabolic profiles.

We found no significant impact of age, BMI, or type of ADT on changes in FBS at three months. While age is a risk factor for some metabolic changes, studies do not consistently show that age independently affects the intensity of ADT-induced changes in FBS. Older patients may have more pronounced metabolic effects due to the co-morbidities they may have and decreased activity, but this is not specifically attributed to age alone.[22]

Obesity has been shown to exacerbate metabolic changes, including insulin resistance and increased FBS, but studies do not show that ADT independently causes the effects on FBS.[22,23]

With the different types of ADT (e.g., GnRH agonists vs. antagonists), studies have shown varying effects on metabolic parameters, but there is no clear proof that the type of ADT specifically influences changes in FBS levels. Nevertheless, the addition of antiandrogens to GnRH agonists may lessen some metabolic effects, but this does not precisely address FBS changes.[9,24]

Limitations

The pilot study has given useful data on the metabolic effects of Androgen deprivation therapy among indigenous black men with prostate cancer after three months. As a pilot study, the findings give initial data but may not be robust enough to draw definitive conclusions due to the small cohort. A study conducted in a larger, multi-centre setting would be important to confirm these findings.

There was no non-ADT control group in the study, which would have shown whether the observed changes in fasting blood glucose are solely due to ADT or affected by other factors in the patients, like disease progression or lifestyle habits.

Some potential confounders, like exercise, diet, supplements, and other metabolic parameters, were not controlled for in this study. These could affect glucose metabolism and might have contributed to the observed changes in the study.

The study assessed fasting blood glucose levels over only three months. Even though this provides initial insights into metabolic changes that commence early, the period may not be enough to show long-term metabolic changes like the development of diabetes.

CONCLUSION

Prostate cancer remains a significant health burden. Its progression is driven by androgens; hence, ADT remains an important approach in its management. Our pilot study gave the preliminary evidence that ADT among Indigenous prostate cancer patients increased the mean FBS over three months (p=0.171) and an increase in FBS among hypertensive patients (p=0.027) and those with high-risk prostate cancer (p=0.046).

The study shows the need for metabolic monitoring in prostate cancer patients undergoing ADT, especially those with preexisting hypertension or those with a high-risk disease.

To build upon these preliminary findings from this pilot study, it is recommended that future studies expand on these findings to come up with strategies for mitigating metabolic risks related to ADT, which improves the long-term health outcomes among prostate cancer patients.

DECLARATIONS

Author contributions

All contributing authors listed in the manuscript are aware of and agree to the submission of this manuscript.

Data Availability

The data generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

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