Research Article

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Association of Obesity with Prostate Cancer at Time of Biopsy: A Multi-Centre 10-Year Retrospective Study

Cyril Kamadjou[1,2*], Divine Enoru Eyongeta[3], Quincy Jones Shumbang[3], Annie Kameni Wadeu[1], Boris Amougou[4] and Fru Angwafo[5]

[1]*Saint Cyr Endoscopy Urology Centre, Douala, Cameroon.*

[2]*Medico Surgical Centre of Urology and Mini Invasive Surgery, Douala, Cameroon.*

[3]*Department of Surgery, Faculty of Health Sciences, University of Buea, Cameroon.*

[4]*Faculty of Medicine and Pharmaceutical Sciences, University of Dschang, Cameroon.*

[5]*Faculty of Medicine and Biomedical Sciences, University of Yaoundé I, Yaoundé, Cameroon.*

***Correspondence:**
Cyril Kamadjou, Saint Cyr Endoscopy Urology Centre, Douala, Cameroon.

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ABSTRACT

Background and Aim: *The incidence of Prostate cancer (PCa) is on the rise in Cameroon, making it the most commonly diagnosed cancer amongst men. Factors, such as obesity, smoking, and the consumption of high fat diet have been reported to play a role in the aetiology and progression of cancers in general. However, the exact relationship between obesity and prostate cancer in Africans is still poorly described. The present study aimed to evaluate the association between obesity as measured by the body mass index (BMI) and high-grade prostate cancer in Cameroonians.*

Methods: *This was a retrospective analyses of the clinical records of 300 consecutive men who underwent transrectal ultrasound-guided core needle prostate biopsy between January 2010 and January 2020. Patients were grouped as obese (BMI ≥30kg/m²) and non-obese (BMI <30kg/m²) and binary logistic regression modelling was used to compare the association between obesity and patient characteristics, such as age, digital rectal examination (DRE) findings, prostate specific antigen (PSA) level, prostate volume, PSA density, and Gleason score (GS).*

Results: *The mean age at diagnosis of PCa was 65.46 ± 8.54 years (Range: 34-89 years), the mean BMI was 28.93 ± 4.57 kg/m² (Range: 18.35-42.37 kg/m²), and the median total PSA level was 44.2 ng/ml. In total, 142 men (47.3%) were obese and biopsies detected high-grade prostate cancer in 160 men (63.3%). Obese men were significantly younger and had a higher risk (OR= 2.74, p <0.001) of having high-grade PCa compared to non-obese men. Furthermore, 94 obese men (58.75%) had high-grade PCa. Obese men had higher age-adjusted PSA densities (P<0.001), while there was no statistical difference in DRE findings (OR= 1.06, p= 0.225).*

Conclusion: *This study demonstrated that obesity is associated with a higher risk of high-grade prostate cancer. This risk is more pronounced among younger patients and DRE findings are not influenced by BMI.*

Keywords
Obesity, Body mass index, Prostate cancer, High-grade prostate cancer.

Introduction
Prostate cancer (PCa) is the most commonly diagnosed malignancy worldwide and the sixth leading cause of cancer death in men [1]. PCa is a global public health issue such that the incidence and mortality of PCa is gradually increasing. As of 2018, there were 1,276,106 new cases of PCa in men, causing 358,989 deaths [2]. The overall pooled incidence of PCa in Africa in a systematic review and meta-analysis study was established to be 21.95 (95% Confidence Interval: 19.93–23.97)/100,000 population, with a median incidence of 19.47/100,000 population [3]. In Cameroon, PCa is the most commonly diagnosed cancer among men [4,5], with a jump in the incidence from 93.8 per 100,000 person-years in 1998, to 1047 per 100,000 person-years in 2015 [6,7]. This incidence, once underestimated, has been increasing since the popularization of screening methods, including digital rectal examination (DRE) and the prostate specific antigen (PSA) assay [8]. So far, only race, family history and age are the established non-modifiable epidemiological risk factors associated with the development of PCa [9]. However, the large geographical variation in PCa risk suggests that lifestyle factors including obesity, consumption of diet high in fat, and smoking which are all modifiable, play a significant role in the pathogenesis of cancer, particularly PCa [10,11].

The prevalence and mortality of obesity is increasing globally, with around 2.8 million people dying annually from clinical overweight or obesity, making it a global epidemic [12]. In 2013, 35% of the world's adult populations were overweight and 11% were obese [13-15]. Studies have shown that, the elderly are prone to obesity and PCa, probably due to increased sedentary lifestyle and life expectancy respectively [16,17]. While obesity is linked to physical inactivity, it can lead to the development of insulin resistance, which in turn, leads to chronically elevated blood levels of insulin that promotes the growth and proliferation of prostate cells, leading to cancer [18]. Furthermore, obese men tend to have a greater plasma volume which causes hemo-dilution of PSA , thus, masking PCa predictability [19-21]. Nonetheless, obese men are known to have a larger prostate size with or without an underlying prostate disease compared to non-obese men [20,21]. This may pose difficulties in identifying a pathologic prostate during DRE in obese men, and may lead to the misdiagnosis and late detection of PCa. Several studies have been carried out to assess the relationship between obesity and PCa, but their results are inconsistent [22-25]. In Cameroon, a study reported that, high BMI ($\geq 25 kg/m^2$) was associated with high-grade (Gleason score $\geq$ 7) PCa [25]. In general, a good number of studies have established a link between obesity and PCa. However, little has been done to the best of our knowledge in Cameroon on this topic. Therefore, this study aimed to evaluate the association between obesity and PCa in Cameroon, using our own epidemiological definition of obesity as BMI $\geq 30 kg/m^2$.

Methods
Study design, setting, and participants
This was a hospital-based, retrospective records analysis carried out in the Limbe Regional Hospital Limbe (LRH) and the Medico-surgical Centre of Urology (CMCU) in Douala, Cameroon. We reviewed the records from January 1st 2010 to January 1st 2020. The LRH, located in the South West region, is a secondary hospital that also serves as a university teaching hospital. CMCU Douala, located in the Littoral region, is a specialized center of adult and pediatric urologic surgery.

The records of individuals diagnosed with PCa through a transrectal Ultrasound (TRUS) guided core needle biopsy in LRH and CMCU Douala were included in the study. We excluded the records of patients with incomplete clinical data.

The sampling method for this study was a convenient sampling, and we projected a minimum sample size of 123 patient-files, using the formula for descriptive studies [26]. We ended up sampling 300 patient-files.

Study procedure
Administrative clearance for this study was obtained from the Regional Delegation of Public Health for the Southwest region and the Littoral region of Cameroon. Ethical approval was granted by the Institutional Review Board of the Faculty of Health Sciences, University of Buea, Cameroon. Participants' confidentiality was respected by not collecting identifying information.

Data collection, management, and analysis
Data were collected using a structured data collection sheet, including: i.) Socio-demographic data; age, height and weight in years, meters and kilogram respectively, profession and marital status, ii.) past history; family history of PCa, history of hypertension, history of type 2 diabetes mellitus and history of tobacco smoking, history of previous prostate biopsy and history of surgical treatment of prostate diseases (e.g. Transurethral resection of the prostate), iii.) Physical examination; DRE findings, iv.) Investigations; Free PSA, Total PSA, Prostate volume, Prostate biopsy; number of core biopsy and Histopathology results; Gleason score.

Collected data were entered into Epi Info version 7.2.2.4 [28] and analyzed using the Minitab software version 17.1.0 [29]. Continuous variables, including age, total PSA, free PSA, PSA ratio, prostate volume, PSA density, number of cores biopsy and BMI were presented as means (standard deviation) when the distribution was symmetrical or median when skewed. Categorical variables, including obesity, DRE findings, family history of PCa and Gleason score were presented as frequencies and percentages. Correlations and associations were computed using the chi-squared test and Odds ratios (OR) were used to quantify the degree of association. The threshold for statistical significance was set at a p-value< 0.05. Binary logistic regression analysis was used to assess association between obesity and high-grade PCa (GS $\geq$ 7), abnormal DRE findings and PSA density.

Results

General characteristics of the patients

The mean age at diagnosis of PCa was 65.46 ± 8.54 years (Range: 34-89 years). The mean BMI at diagnosis was 28.93 ± 4.57 kg/m² (Range: 18.35-42.37 kg/m²). Of the 300 men, 278 (92.67%) had an abnormal DRE finding while 22 (7.33%) had normal DRE. The median PSA density was 0.686 ng/ml/cc (IQ range: 0.303-1.506 ng/ml/cc). There were 140 (46.67%) men with low-grade tumours, while 160 (63.33%) men had high-grade tumours (Table 1).

Table 1: General characteristics of the patients in LRH and CMCU Douala, Cameroon.

Variables	Value
Age (years), Mean ± SD (range)	65.46 ± 8.54 (34–89)
BMI (kg/m²), Mean ± SD (range)	28.93 ± 4.57 (18.35 – 42.37)
No family history of prostate cancer, n (%)	20 (6.67%)
Positive family history of prostate cancer, n (%)	1 (0.33%)
Not sure of a family history of prostate cancer, n (%)	279 (93%)
Abnormal DRE findings, n (%)	278 (92.67%)
Normal DRE findings, n (%)	22 (7.33%)
Total PSA (ng/ml), median (IQ range)	44.2 (17.16 - 104)
Free PSA (ng/ml), median (IQ range)	1.151 (0.88 – 2.09)
PSA ratio, median (IQ range)	8.98 (7.47 – 15.79)
Prostate volume (cc), Mean ± SD (range)	73.87 ± 44.42 (16 – 408)
PSA density (ng/ml/cc), median (IQ range)	0.686 (0.303 – 1.506)
Number of core biopsy, n (%)	
<12	16 (5.34%)
12	265 (88.33%)
≥13	19 (6.33%)
Low-grade prostate cancer, n (%)	140 (46.67%)
High-grade prostate cancer, n (%)	160 (53.33%)

BMI= Body Mass Index, DRE= Digital Rectal Examination, IQ= Interquartile, PSA= Prostate Specific Antigen, SD = Standard Deviation.

When the men were stratified according to BMI, 142 (47.33%) were found to be obese and 2 (0.67%) were underweight. Obese men were significantly younger than non-obese men (Table 2).

Clinical and Para-Clinical Characteristics of the Patients

Of the 278 men with abnormal DRE findings, 132 (92.96%) were obese and 146 (92.41%) were non-obese. The median total PSA was 42.0 ng/ml among men with BMI ≥ 30 kg/m², compared to 44.4 ng/ml among non-obese men. High-grade PCa (Gleason score ≥ 7) was significantly more frequent among obese men compared to non-obese men (p-value< 0.001). However, there was no significant difference in the free PSA level (p= 0.260), PSA ratio (p= 0.341), prostate volume (p= 0.425), and PSA density (p= 0.587) between obese and non-obese men. When the PSA density adjusted for age, it became significant higher in obese men (p <0.001) (Table 3).

Association between obesity and high-grade prostate cancer

On crude analysis, obesity was found to significantly increase the risk of having high-grade PCa (OR= 2.73; p<0.001). When adjusted for age, the odds of obese men having high-grade PCa increased by 10% (OR= 3.00 and p<0.001). On multivariate-adjusted analysis, the odds of obese men having high-grade PCa were even higher (OR= 3.08 and p<0.001) as seen in Table 4.

Association between obesity and high-grade prostate cancer stratified by DRE finding

On crude analysis, when stratified by DRE finding, obesity was associated with a higher risk of high-grade prostate cancer, though this association was not statistically significant (OR= 1.06 and p= 0.225). Results remained the same after adjusting for age only (Table 5).

Table 2: Age distribution of the men with prostate cancer in LRH and CMCU Douala stratified by obesity status.

BMI (kg/m²) Category	Age (years)			Frequency (%)	p-value
	Mean	SD	Range (min–max)		
<18.5	76	± 2.82	74 - 78	2 (0.67)	
18.5 – 24.9	68.14	± 7.90	52 - 88	64 (21.33)	
25 – 29.9	66.46	± 7.71	48 - 89	92 (30.67)	
≥30	63.52	± 8.90	34 - 87	142 (47.33)	**0.001**
≤30	67.26	± 7.82	48 - 89	158 (52.67)	

SD = standard deviation, BMI= body mass index

Table 3: Clinical and para-clinical characteristics of the men with prostate cancer in LRH and CMCU Douala stratified by obesity status.

Variables	Obese patients	Non-obese patients	p-value
Total PSA (ng/ml), Median (IQ range)	42.0 (18.3 – 112.0)	44.4 (14.4 – 92.6)	0.754
Free PSA (ng/ml), Mean ± SD	2.60 ± 2.78	1.54 ± 1.79	0.260
PSA ratio (ng/ml), Mean ± SD	11.42 ± 7.67	16.63 ± 17.77	0.341
Prostate volume (cc), Median (IQ range)	62.0 (40.0 – 96.0)	67.5 (46.5 – 93.0)	0.425
PSA density (ng/ml/cc), Median (IQ range)	0.74 (0.30 – 1.74)	0.64 (0.30 – 1.45)	0.587
PSA density (ng/ml/cc), Mean ± SD	5.68 ± 45.80	3.60 ± 10.38	0.587
Age-adjusted PSA density			<0.001
Gleason score ≥7, n (%)	94 (58.75)	66 (41.25)	<0.001
Gleason score 7, n (%)	48 (34.29)	92 (65.71)	
Abnormal DRE finding, n (%)	132 (92.96%)	146 (92.41%)	

IQ= Interquartile, PSA= Prostate Specific Antigen.

Table 4: Association between obesity and high-grade prostate cancer in LRH and CMCU Douala.

	Odds of having high-grade prostate cancer (Gleason score ≥ 7)		p-value
	Obese men, OR (95% CI)	Non-obese men, OR (95% CI)	
Crude	2.731 (1.706 – 4.371)	0.366 (0.228 – 0.585)	**<0.001**
Age-adjusted	3.003 (1.840 – 4.899)	0.333 (0.204 – 0.543)	**<0.001**
Age and prostate volume-adjusted	2.989 (1.809 – 4.939)	0.334 (0.202 – 0.552)	**<0.001**
Age and total PSA-adjusted	3.141 (1.914 – 5.155)	0.318 (0.194 – 0.522)	**<0.001**
Multivariable-**adjusted†**	3.080 (1.848 – 5.132)	0.324 (0.194 – 0.541)	**<0.001**

†Adjusted for age, prostate volume, total PSA, and PSA-density.

Table 5: Association between obesity and high-grade prostate cancer in LRH and CMCU Douala stratified by DRE finding.

	Odds of having high-grade prostate cancer (Gleason score ≥ 7)		
	Obese men with abnormal DRE findings, OR (95% CI)	Non-obese men with abnormal DRE finding, OR (95% CI)	p-value
Crude	1.065 (0.443 – 2.561)	0.938 (0.390 – 2.254)	0.225
Age-adjusted	1.179 (0.479 – 2.905)	0.847 (0.344 – 2.087)	0.245
Age and prostate volume-adjusted	0.986 (0.384 – 2.533)	1.013 (0.394 – 2.601)	0.338

CI= Confidence Interval; OR= Odds Ratio.

Discussion

We retrospectively studied 300 men from LRH and CMCU Douala who had PCa. Nine in every ten participants had a 12-core TRUS guided prostate biopsy. Obese men were averagely younger and had a significantly higher age-adjusted PSA density. Obesity was found to be associated with a higher risk of high-grade PCa. The mean age at diagnosis of PCa was 65.46 ± 8.54 years and the ages of the men ranged from 34–89 years. The mean age in the current study was similar to that reported by Engbang et al. [7], Tchinda et al. [27] and Angwafo et al. [29] all in Cameroon who had an average age of 66.88 yrs, 67.82 yrs and 67 yrs, respectively. When the men were stratified according to BMI, those who were obese were significantly younger (63.52 yrs) than those with BMI< 30 kg/m² (67.26 yrs). This could be as a result of the increase sedentary lifestyle among young people [14].

Studies have shown that obese men with PCa have lower PSA values compared to non-obese men, due mainly to hemo-dilution of PSA as a result of plasma volume expansion [19-21]. In our study there was no significant difference (p-value = 0.754) in total serum PSA between obese and non-obese men. This finding was similar to that of Park et al. [23], in Korea, who reported no significant difference (p-value = 0.932) in total PSA between obese (BMI ≥ 25 kg/m²) and non-obese (BMI < 25 kg/m²) men, despite their difference in definition of obesity using BMI. On the contrary, Freedland et al. [24], in USA, had a significantly lower total PSA in obese men (P-value = 0.02). This could be because the rate of screening for PCa using PSA in Cameroon is fundamentally very low compared to the United Sates, where approximately 75% of men aged 50 years or older have their PSA levels measured at least once [7,30]. Hence, there is early detection of PCa in USA as compared to Cameroon where late detection is accompanied by cachexia and therefore these men will appear as non-obese at the time of biopsy (diagnosis). Furthermore, our study evaluated total PSA of participants diagnosed with PCa among obese and non-obese men. Hence, this will best reflect the relationship between obesity and total PSA and their impact on PCa grade at time of diagnosis (biopsy).

Review of literature shows that, obese men have larger prostate volumes in the presence or absence of an underlying prostate disease [20,21]. In our study, of the 300 men, 290 had their prostate volume recorded. Contrary to literature, we noticed no significant difference in prostate volume (p= 0.425) between obese men (n= 138) and non-obese men (n= 152). This is probably due to a difference in methodology in our study compared to other studies, as all the men included in our study already had confirmed diagnosed of PCa. In addition, we defined obesity as BMI ≥ 30 kg/m² as opposed to BMI ≥ 25 kg/m²) considered in other studies.

An important finding from the current study was that obesity was associated with high-grade PCa. This finding is similar to the findings reported by Li et al. [31], in China and Masuda et al. [22], in Japan, despite the difference in our definition of high-grade PCa, as we included Gleason scores of 3+4 and 4+3 in ours. Park et al. [23], in Korea also reported similar findings on their multivariate-adjusted analysis, as they found a significant association (OR = 1.49; p= 0.039) between obesity (BMI ≥ 25 kg/m²) and high-grade (Gleason score ≥ 4+3) PCa. Freedland et al. [24], in USA, reported findings different from ours, they found a non-significant association (OR = 1.82; p= 0.18) between obesity (BMI ≥30 kg/m²) and high-grade (Gleason score ≥ 4+3) PCa, when multivariate-adjusted for race, age, center, PSA, prostate volume and DRE findings. Freedland and colleagues did not consider Gleason 3+4 as high-grade, and they used a relatively smaller number (149) of men with PCa. In the current study obesity did not significantly increase the odds of having abnormal DRE findings (OR= 1.06; p= 0.225). Price et al. [32], in USA, carried out a similar study with 535 participants without a confirmed diagnosis of PCa. They reported that, it was less likely for obese men (BMI ≥ 30 kg/m²) to have an abnormal DRE finding (OR= 0.50 for mild obesity; OR= 0.28 for moderate and severe obesity; p= 0.03). It is certain, based on existing literature, that obese men have larger prostate sizes [20,21], usually detected on DRE. Nonetheless, it is more difficult to have abnormal DRE findings in obese patients due to the presence of perirectal fat which might compromise the DRE findings [33,34]. Moreover, obesity as determined by BMI does

not always correlate with central obesity (which is a stronger assessment tool for obesity).

PSA density is known to improve the sensitivity of PSA as a marker of PCa and by extension, factors that affect PSA would affect PSA density [35]. Obese men are therefore, expected to have lower PSA-densities at time of prostate biopsy (diagnosis). In our study, on crude analysis, obese men had a higher mean PSA density, though the difference was not statistically significant. However, because there is late diagnosis of PCa in our setting, people with PCa are usually cachectic at diagnosis, which might have influenced our results, as obese men with long standing undiagnosed PCa might have been diagnosed at a time when they had lost body weight. Because of our study design, we could not assess the relationship between central obesity and prostate cancer grade. We, therefore, recommend further studies to evaluate the influence of central obesity and age-adjusted PSA density on PCa grade at diagnosis.

Conclusion

In summary, this study demonstrated that obesity as determined by BMI is associated with a higher risk of high-grade prostate cancer at diagnosis. This risk is more pronounced among younger patients and DRE findings are not influenced by BMI. Further studies are recommended in Cameroon and sub-Saharan Africa to assess the association between central obesity, digital examination findings, age-adjusted PSA density and other biomarkers and PCa progression.

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