ORIGINAL RESEARCH



Comparison of prostate size and anthropometric parameters between diabetic and non-diabetic Congolese patients who underwent transurethral prostate resection in the Democratic Republic of Congo

L. E. Mubenga^{1*}, D. Chimanuka¹, P. De Groote², E. Bwenge³, M. P. Hermans⁴ and B. Tombal⁵

Abstract

Background: Benign prostate hyperplasia (BPH) and type 2 diabetes mellitus are prevalent in older men, and both represent a challenge to public health. Prior studies reported a correlation between BPH and (hyper)glycaemia, a component of the metabolic syndrome, which is on the increase in sub-Saharan Africa (SSA) due to rapid modernization. This study was designed to evaluate the association of prostate volume and anthropometric parameters among diabetic and non-diabetic patients who had transurethral resection of the prostate (TURP) for BPH.

Results: We analyzed data of 159 selected patients who had TURP over a three-year period (February 2014–January 2017) for histologically confirmed BPH. Mean age in the entire cohort was 68 ± 8.5 years. Out of the 159 patients, 94 (59.1%) were non-diabetics and 65 (40.9%) were diabetics. International Prostate Symptom Score (IPSS) and fasting blood glucose were significantly higher in diabetic than in non-diabetic group ($28.6 \pm 4.3 \text{ vs } 25.6 \pm 6.4$, and p 0.005; $121.7 \pm 45.7 \text{ vs } 85.4 \pm 11.7 \text{ mg/dl}$, and p < 0.001, respectively). BMI and waist circumference were statistically greater in non-diabetics than in diabetics ($25.1 \pm 3.3 \text{ kg/m}^2 \text{ vs } 23.6 \pm 3.5 \text{ kg/m}^2$, and p 0.008; 94.6 cm \pm 10.3 vs 90.6 \pm 10.4 cm, and p 0.018). Diabetic patients had larger prostate volume than non-diabetic in the working age subgroup only (< 65 years of age); beyond 65 years, this difference was not consistent ($62.6 \pm 23.1 \text{ cc vs } 50.1 \pm 20.7 \text{ cc}$, and p 0.027; $56.2 \pm 23.7 \text{ cc vs } 49 \pm 20.2 \text{ cc}$, and p 0.15, respectively). Prostate size was significantly associated with fasting blood glucose (p = 0.002) and PSA (p = 0.027). However, prostate size was not related to age, presence of diabetes, BMI, waist circumference, IPSS, quality of life score, and duration of symptoms.

Conclusion: Prostate volume is not correlated with anthropometric parameters in diabetic and non-diabetic Congolese patients who had TURP in South Kivu. Diabetics were not obese and yet had larger prostate volume than non-diabetics < 65 years of age. It is hoped that these results would form groundwork for further studies on this topic in SSA region.

Keywords: Diabetes, Anthropometrics, Prostate volume

*Correspondence: leonmubenga@yahoo.fr;

emmanuelmubengalm@gmail.com

¹ Department of Urology, Université Catholique de Bukavu (UCB), Bukavu,

Democratic Republic of Congo

Full list of author information is available at the end of the article



© The Author(s) 2019. This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made.

1 Background

Modernization and greater access to healthcare in sub-Saharan Africa (SSA) increase life expectancy and consequently an increasing prevalence of age-related conditions such as benign prostate hyperplasia (BPH) [1–3]. This modernization also leads to lifestyle changes including dietary fat intake, typical of the Western world, lack of physical activity, and use of motorized machines to perform daily life activities. This western lifestyle exposes SSA populations to the emergence of newer chronic diseases including cardiometabolic conditions such as the metabolic syndrome (MetS) [4–7].

MetS involves different but interconnected entities, namely whole-body insulin resistance (IR) and secondary compensatory hyperinsulinemia, overweight or obesity, dyslipidemia, type 2 diabetes mellitus (T2DM), and hypertension [4–7]. Tissue insulin resistance and secondary hyperinsulinemia promote the synthesis and release of tissue growth factors which also act on the prostatic tissue level. Moreover, chronic hyperglycemia has a trophic role on various tissues, including prostatic cells [8–10]. Hyperinsulinemia and hyperglycemia are the two major components of the common form of T2DM (i.e., with MetS), a phenotype equally frequent in elderly people [11, 12].

Therefore, it would be logical to expect patients with T2DM, who are often obese or at least overweight, to have a prostate larger than that of age-matched nondiabetic men. Thus, several authors reported positive correlations between prostate volume on the one hand and raised blood glucose or insulin on the other [13–18]. However, the correlation between prostate volume and anthropometric parameters is not unanimously accepted among authors [7, 11]. Furthermore, it is not determined whether hyperglycemia in the absence of obesity would induce such a relationship in populations with low prevalence of obesity, such as South Kivu in the Democratic Republic of Congo (DRC), or in elderly patients with both T2DM and BPH, who are often neither obese nor overweight.

The objective of this study was to evaluate the relationship between prostate volume and anthropometric parameters including body mass index (BMI) and waist circumference in diabetic and non-diabetic patients from South Kivu.

2 Methods

We reviewed the medical records of patients with BPH who had transurethral resection of the prostate (TURP) over a three-year period between February 1, 2014, and January 31, 2017, in three medical institutions of Bukavu, the main city of South Kivu.

Patients were divided into two groups: diabetic and non-diabetic.

Age, International Prostate Symptom Score (IPSS), quality of life score, duration of symptoms, known diabetes, BMI, waist circumference, fasting glycaemia, prostate-specific antigen, and prostate volume were recorded for each patient.

We excluded patients with known prostate cancer that had a palliative channel cut, and those in whom incidental prostate cancer or urethral stenosis was found.

2.1 BPH assessment

All patients completed the IPSS questionnaire to evaluate the severity of lower urinary tract symptoms (LUTS) and had digital rectal examination (DRE). BPH was postoperatively confirmed by pathologist's examination.

Prostate volume was calculated by trans-rectal ultrasonography (using a Bruel and Kjaer Medical scanner 7, 5 MHZ), according to the ellipsoid formula, multiplying height (H) obtained by transaxial scanning, width (*W*), and length (*L*) by 0, 524 ($H \times W \times L \times \pi/6$) [19].

PSA level was determined using an ELISA method, and all patients with elevated PSA level had prostate biopsy.

2.2 Diabetes mellitus

Information on diabetes mellitus (DM) diagnosis was based on self-reporting of the disease, current use of glucose-lowering medication(s), and/or laboratory-measured fasting blood glucose \geq 126 mg/dl [13–16].

2.3 Overweight-obesity

Overweight–obesity were defined from BMI cutoffs, with BMI expressed as weight in kilogram divided by square height in meter [12, 20–23]. According to WHO classification, patients were divided in four categories: underweight if BMI <18.5 kg/m²; normal weight if BMI 18.5–24.9 kg/m²; overweight if BMI \geq 25 kg/m²; and obese if BMI \geq 30 kg/m².

Central obesity was defined using the International Diabetes Federation recommendations for pathological waist circumference [12, 20–23]. Waist circumference was measured from midway between the lowest rib and the iliac crest and considered normal \leq 94 cm.

2.4 Statistical analysis

Statistical analyses were carried on using Stata 13 software. Continuous data are presented as means or medians with standard deviation (SD) and interquartile range (IQR), respectively. Categorical data are presented as frequencies or proportions. The distribution of continuous data was assessed, and skewed data were log-transformed prior to testing. Comparison between background characteristics of the study population was made using student's *t* test for normally distributed data. Whenever the shape of the distribution remained asymmetrical after log-transformation, Wilcoxson's sum rank test was used.

To examine the relationship between prostate volume and correlates, we carried out both bivariable and multivariable linear regression analyses, with heteroscedasticity–robust standard errors. Variables were brought into the multivariable model if they met the inclusion criteria set to $p \le 0.1$ at bivariable analysis, or based on biological plausibility. We reported unadjusted and adjusted slopes with 95% confidence intervals (CIs) and p-values. Statistical significance for all analyses was set to $\alpha = 5\%$.

3 Results

3.1 Patients' characteristics

Data of 159 patients aged 45–96 years that had TURP were analyzed. Out of the 159 patients, 94 (59.1%) were non-diabetic and 65 (40. 9%) were diabetic.

Mean age was 68 ± 8.5 years in the entire cohort: 69.3 ± 8.1 years in the diabetic group and 67.13 ± 8.8 years in the non-diabetic group.

IPSS, fasting blood glucose, and prostate volume were significantly higher in the diabetic than in the non-diabetic group: 28.6 ± 4.3 versus 25.6 ± 6.4 (*p* 0.005); 121.7 ± 45.7 versus 85.4 ± 11.7 mg/dl (*p* < 0.001); and 58.5 ± 23.5 versus 49.4 ± 20.3 g (*p* 0.027), respectively.

BMI and waist circumference were statistically greater in the non-diabetic than in the diabetic group: 25.1 ± 3.3 versus 23.6 ± 3.5 kg/m² (*p* 0.008) and 94.6 ± 10.3 versus 90.6 ± 10.4 cm (*p* 0.018).

There were no statistically significant differences between the two groups regarding age, quality of life score, duration of symptoms, and prostate-specific antigen level.

Descriptive data of the study population are listed in Table 1.

3.2 Comparison of variables between diabetic group and non-diabetic group according to age

Patients were divided into two subgroups based on age: <65 years and \geq 65 years, the latter corresponding to retirement age in the DRC in order to compare anthropometric parameters and prostate size. Prostate volume was greater among the diabetic group in the <65-year subgroup (62.6 ± 23.1 vs 50.11 ± 20.7, and *p* = 0, 027). BMI and waist circumference were greater in the non-diabetic group than in the diabetic group among the >65-year subgroup, with a statistically significant difference (25.3 ± 3.4 vs 23.6 ± 3.2 kg.m⁻² (*p* 0.012) and 95.3 ± 10.8 vs 90.4 ± 10.7 cm (*p* 0.029) (Table 2).

Table 1 Patients' characteristics

Variable	All patients	Diabetic group	Non-diabetic group	р	
	n=159	n=65	n=94		
Age (years)	68±8.5	69.3±8.1	67.13±8.8	0.12	
	67 (62, 74)	69 (62, 76)	66 (62, 72)		
IPSS	n = 159	n=65	n = 94		
	26.8 ± 5.8	28.6 ± 4.3	25.6 ± 6.4	0.005 ^w	
	27 (24, 32)	29 (27, 32)	27 (21, 31)		
QOL	n = 159	n=65	n=94		
	6.2 ± 0.8	6.3 ± 0.7	6.2 ± 0.8	0.24 ^w	
	6 (6, 7)	6 (6, 7)	6 (6, 7)		
DS	n = 159	n=65	n=94		
	4.11 ± 3.1	4.2 ± 2.7	4.1 ± 3.3	0.38 ^L	
	3 (2, 5)	3 (2, 6)	3 (2, 5)		
PSA	n = 159	n = 65	n = 94		
	5.8 ± 4.9	5.8 ± 4	5.8 ± 5.5	0.4^{L}	
	4 (3, 7)	4.2 (3.2, 4.2)	4 (2.7, 7)		
FBG	n = 154	n = 65	n=89		
	100.7 ± 35.7	121.7 ± 45.7	85.4 ± 11.7	< 0.001 ^w	
	91.5 (81, 107)	110 (96, 131)	87 (76, 95)		
BMI	n = 159	n=65	n = 94		
	24.5 ± 3.4	23.6 ± 3.5	25.1 ± 3.3	0.008	
	23.5 (22, 26.7)	22.7 (21.7, 23.9)	24.7 (22.8, 27.5)		
WC	n = 159	n=65	n=94		
	92.9 ± 10.5	90.6 ± 10.4	94.6 ± 10.3	0.018	
	91 (86, 96)	88 (85, 93)	94 (88, 98)		
PV	n=159	n = 65	n = 94		
	53.2 ± 22	58.5 ± 23.5	49.4 ± 20.3	0.027 ^w	
	50 (35, 66)	60 (42, 71)	45 (33, 63)		

IPSS International Prostate Symptom Score (0–35), *QOL* quality of life score (1–7), *DS* duration of symptoms (years), *BMI* body mass index (kg/m²), *WC* waist circumference (cm), *FBG* fasting blood glucose (mg/dl), *PSA* prostate-specific antigen (ng/ml), *PV* prostate volume (cc)

WWilcoxon's sum ranked test

L statistical tests were run on log-transformed variables

3.3 Risk factors for BPH

In the univariate logistic regression analysis, FBG, PSA, diabetes, and QOL score were significantly related to prostatic hyperplasia. In the multivariate analysis, only FBG (p=0.002) and PSA (p=0.027) were predictors of prostatic hyperplasia (Table 3).

4 Discussion

BPH is the commonest benign tumor in men beyond 40 years of age. Two etiologies have long been considered to contribute to the development of this condition: aging and androgens [5]. In recent decades, several other environmental factors were proposed, including chronic inflammation [24], cigarette smoking [25], and MetS [3–7, 14–17]. However, the metabolic syndrome with its

Table 2 Comparisonofanthropometricparametersand prostate volume between diabetic and non-diabeticpatients according to age distribution

Variable	Diabetic group	Non-diabetic group	p	
Age < 65 years ($n = 60$)	n=24	n=36		
BMI	23.7 ± 4	24.7 ± 3.2	0.29	
	22.9 (21.4, 23.9)	24.7 (22.3, 27.3)		
WC	90.9 ± 10.1	93.4±9.2	0.34	
	88 (85.5, 93)	94 (88, 98)		
PV	62.6 ± 23.1	50.1 ± 20.7	0.027 ^w	
	63.5 (44, 80)	44.5 (35.5, 62.5)		
Age \geq 65 years ($n = 99$)	n=41	n=58		
BMI	23.6 ± 3.2	25.3 ± 3.4	0.0112	
	22.6 (21.8, 23.8)	24.6 (22.8, 27.9)		
WC	90.4 ± 10.7	95.3 ± 10.8	0.029	
	88 (85, 93)	94 (88, 100)		
PV	56.2 ± 23.7	49 ± 20.2	0.15 ^w	
	60 (35, 66)	50 (32, 63)		

 $\it BMI$ body mass index (kg/m²), $\it WC$ waist circumference (cm), $\it PV$ prostate volume (cc)

W Wilcoxon's sum ranked test

corollaries, namely diabetes and obesity/overweight, is among major risk factors for BPH [3–7, 14–17].

In developed countries, the link between BPH and metabolic syndrome has been established. In several studies, the presence of a MetS was associated with prostatic hyperplasia [3–7, 14–17]. T2DM is an important component of the metabolic syndrome phenotype; it is associated with several other urological pathologies such as aggressive prostate cancers, erectile dysfunction, overactive bladder, urinary tract infections, and urolithiasis [3].

MetS prevalence is increasing rapidly in SSA, because of increased urbanization and modernization of lifestyles [4]. Insulin, often considered as an abundance hormone, favors fat storage in adipocytes, with hyperinsulinemia promoting central adiposity and general obesity. Insulin resistance and hyperinsulinemia, two major underlying components of the MetS, promote the synthesis and release of insulin-like growth factors (IGFs), directly involved in prostate enlargement and prostatic cancer [8-10].

The observations gathered from our survey do not demonstrate an association between prostate size and increased BMI. Furthermore, neither whole-body obesity (inferred from BMI) nor central adiposity (assessed by waist circumference) correlated with diabetes mellitus. This is opposite to what is currently described in the literature regarding positive correlations between BMI, T2DM, and prostate volume. It is worth noting that non-diabetic patients from South Kivu had higher anthropometric indices than their diabetic counterparts, a situation opposite to that of Caucasian populations, in which T2DM patients are usually obese (Table 1). This paradox is often referred to as the "metabolically obese but normal weight" (MONW) phenotype, more represented in SSA and Asian populations [26].

In addition, absence of obesity markers among diabetic patients in this study might be due to a restrictive diet and urinary caloric loss as a consequence of poor glycemic control (Table 1).

Parsons et al. [3] performed a prospective cohort study in 422 subjects and concluded that obesity, fasting blood glucose, and diabetes markedly increase the risk of BPH. Daniel [27] demonstrated that prostate volume in men undergoing TURP was correlated with obesity. Central obesity increases aromatization of androgens to estrogens, an imbalance which is hypothesized to contribute to increase prostate volume [12–14]. Nevertheless, Kim et al. [18] found no correlation between BMI and prostate size. Further, Badmus et al. [12] reported that prostate

Table 3 Logistic regression analysis for predictors of prostate hypertrophy

	Unadjusted slope			Adjusted slope		
	β	95% CI	p value	β	95% Cl	<i>p</i> value
FBG	0.174	0.109-0.240	< 0.001	0.149	0.057-0.241	0.002
PSA	1.161	0.482-1.840	0.001	1.093	0.129-2.057	0.027
Diabetes	9.112	2.210-16.015	0.010	2.850	- 5.588 to 11.290	0.505
Age	0.119	- 0.271 to 0.510	0.547			
IPSS	0.445	0.134-1.024	0.131			
QOL	5.612	1.340-9.883	0.010			
BMI	- 0.627	1.627-0.373	0.217			
WC	- 0.145	0.476-0.186	0.389			
DS	0.142	0.987-1.271	0.804			

CI confidence interval

volume correlates with age but not with anthropometrics in Southwestern Nigerians.

In our study, the absence of obesity or overweight might be due to restrictive diets, high levels of occupational physical activity, poor glycemic control (with substantial urinary caloric loss) as result of insufficient resources and healthcare, limited access to high-energy foods or glucose-lowering drugs and glucose monitoring, and absolute insulinopenia in later-stage T2DM that characterize many diabetic patients in SSA. Therefore, obesity is less commonly associated with diabetes in rural areas, such as South Kivu, as also described in other SSA countries [26, 28–30].

TURP was recently made available in Bukavu, the main city of South Kivu. This new technique was performed for the first time in Bukavu in February 2014. It attracted a lot of patients from rural areas who otherwise did not seek help, considering the long delays between onset of symptoms and consultation. Obesity markers were scant among these patients since westernized lifestyles are uncommon in rural areas.

Kachunga et al. [29] found no obese among diabetics in rural areas of South Kivu during a study conducted in 2012. They attributed this observation to atypical characteristics of African T2DM including lower prevalence and severity of insulin resistance and preservation of tissue insulin sensitivity [26, 28, 30]. The correlation between this particular subtype of diabetes and BPH is not yet established. Impaired insulin sensitivity does not seem to be a major contributor to this phenotype.

In our study, duration of obstructive symptoms was not different among diabetics and non-diabetics. However, IPSS was significantly higher in diabetics. The static component due to prostate enlargement is known to associate with a dynamic component induced by increased sympathetic activity in diabetics [7]. Hence, this association has a detrimental effect on the bladder musculature.

Conversely, lower bladder compliance, detrusor instability, and neuropathy induced by a poor glycemic control are susceptible to cause severe LUTS in diabetic patients, mimicking a prostate enlargement [31, 32].

In addition, prostate volume was significantly greater in the diabetic group than in non-diabetic, but only in the subgroup of working age (<65 years); beyond 65 years, this difference was not consistent. At retirement, physical inactivity due to sedentarity and occupational activity cessation is a likely contributor to hyperglycemia in susceptible individuals, with the potential to exert trophic effects on prostatic cells.

Ozcan et al. [33] reported that testosterone levels were lower in diabetic patients. Ryl et al. [34] supported that carbohydrate disorders may contribute to the reduction in testosterone concentration. However, hypogonadism is also known to be a physiological state associated with aging [34, 35]. This might explain why the prostate volume was not statistically different among diabetic and non-diabetic patients beyond 65 years.

While FBG was expectedly higher in diabetic patients, high FBG is also a risk factor for BPH in non-diabetics [18]. According to Parsons et al. [3], elevated fasting plasma glucose and diabetes are risk factors for BPH, whereas improved glycemic control slows BPH progression.

An obvious study limitation of the present survey is the lack of information about other components of the MetS, such as elevated triglycerides and low HDL cholesterol. Another limitation is the selection bias due to the fact that most patients undergoing TURP suffered from severe dysuria. Other large-scale studies are recommended to confirm and extend our findings. However, this study highlights the fact that prostate volume in diabetics is larger than that of non-diabetics even in the absence of obesity or overweight. We hope that these results will serve as a basis for furthermore comprehensive and mechanistic studies on this important topic.

5 Conclusion

Anthropometric parameters such as overweight and waist circumference were not related to higher risk of benign prostatic hypertrophy among Congolese patients who had TURP in South Kivu.

Diabetics were not obese and yet had larger prostate volume than non-diabetics < 65 years of age. Beyond 65 years, prostate volume was not different between diabetic and non-diabetic patients.

Abbreviations

BMI: body mass index; BPH: benign prostate hyperplasia; CI: confidence interval; DM: diabetes mellitus; DRC: Democratic Republic of Congo; DRE: digital rectal examination; FBG: fasting blood glucose; IGFs: insulin-like growth factors; IPSS: International Prostate Symptom Score; IQR: interquartile range; IR: insulin resistance; LUTS: lower urinary tract symptoms; MetS: metabolic syndrome; MONW: metabolically obese but normal weight; PSA: prostate-specific antigen; PV: prostate volume; QOL score: quality of life score; SD: standard deviation; SSA: sub-Saharan Africa; T2DM: type 2 diabetes mellitus; TURP: transurethral resection of the prostate; WC: waist circumference.

Authors' contributions

LEM realized general and urological assessment of the patients and was a major contributor in writing the manuscript. DC actively participated in documentary research and patients' follow-up. PDG provided documentation and trained Bukavu surgical team in TURP procedures. EB performed statistical analysis. MPH assessed and corrected this manuscript. BT assessed and authorized the submission of this manuscript for publication. All authors read and approved the final manuscript.

Acknowledgements

The authors thank the medical students of the Catholic University of Bukavu for their active participation in data collection.

Competing interests

The authors declare that they have no competing interests.

Availability of data and materials

Supplementary data to this article are available and can be obtained if needed.

Consent for publication

Not applicable.

Ethics approval and consent to participate

The study was approved by the Catholic University of Bukavu Ethics and Research Committee (Reference: UCB/CIE/NC/002/2016). Each patient approved to participate in this study. At each step of the present study, confidentiality and anonymity rules were observed.

Funding

We received technical and logistical facilities from the direction of the Provincial General Referral Hospital of Bukavu during patients care and documentation of this study.

Author details

¹ Department of Urology, Université Catholique de Bukavu (UCB), Bukavu, Democratic Republic of Congo. ² Department of Urology, Saint Michel-Cliniques de l'Europe, Brussels, Belgium. ³ Institute of Health and Society - Institut de Recherche Santé et Société (IRSS) School of Public Health, Université Catholique de Louvain, Brussels, Belgium. ⁴ Division of Endocrinology and Nutrition, Cliniques Universitaires St-Luc and Institut de Recherche Expérimentale et Clinique (IREC), Université Catholique de Louvain, Brussels, Belgium. ⁵ Department of Urology, Université Catholique de Louvain (UCL), Brussels, Belgium.

Received: 16 October 2019 Accepted: 21 October 2019 Published online: 16 December 2019

References

- Hall V, Thomsen RW, Henriksen O, Lohse N (2011) Diabetes in sub Saharan Africa 1999–2011: epidemiology and public health implications. A systematic review. BMC Public Health 11:564
- Jaffiol C (2011) The burden of diabetes in Africa: a major public health problem. Bull Acad Natl Med 195(6):1239–1253
- Parsons JK, Carter HB, Partin AW, Windham BG, Metter EJ, Ferrucci L, Landis P, Platz EA (2006) Metabolic factors associated with benign prostatic hyperplasia. J Clin Endocrinol Metab 91(7):2562–2568. https://doi. org/10.1210/jc.2005-2799
- Ejike CECC, Ezeanyika LUS (2008) Metabolic syndrome in sub-Saharan Africa: "smaller twin" of a region's prostatic diseases? Int Urol Nephrol 40:909–920
- Yim SJ, Cho YS, Joo KJ (2011) Relationship between metabolic syndrome and prostate volume in Korean men under 50 years of age. Korean J Urol 52:390–395
- Jeong JH, Kim ET, Kim DK (2011) Association of metabolic syndrome and benign prostate enlargement in young Korean males. Korean J Urol 52:757–762
- Damber JE, Hammarsten J (2015) Insulin resistance, Obesity and LUTD. Conclusive findings on the link between metabolic syndrome and LUTD need more research. EUT Congress news 2015:20
- Wang Z, Olumi AF (2011) Diabetes, growth hormone-insulin-like growth factor pathways and association to benign prostatic hyperplasia. Differentiation 82:261–271
- Sarma AV, Jaffe CA, Schottenfeld D, Dunn R, Montie JE, Cooney KA, Wei JT (2002) Insulin-like growth factor-1, Insulin-like growth factor binding factor-3, and body mass index: clinical correlates of prostate volume among black men. Urology 59(3):362–367
- Winter DL, Hanlon AL, Raysor SL, Watkins-Bruner D, Pinover WH, Hanks GE, Tricoli JV (2001) Plasma levels of IGF-1, IGF-2, and IGFBP-3 in white and African-American men at increased risk of prostate cancer. Urology 58(4):614–618

- Wang CC, Chancellor MB, Lin JM, Hsieh JH, Yu HJ (2009) Type 2 diabetes but not metabolic syndrome is associated with an increased risk of lower urinary tract symptoms and erectile dysfunction in men aged < 45 years. BJU Int 105:1136–1140
- Badmus TA, Asaleye CM, Badmus SA, Takure AO, Ibrahim MH, Arowolo OA (2013) Benign prostate hyperplasia: average volume in southwestern Nigerians and correlation with anthropometrics. Niger Postgrad Med J 20(1):52–56
- 13. Otieno CF, Mwendwa FW, Vaghela V, Ogola EN, Amayo EO (2005) Lipid profile of ambulatory patients with type 2 diabetes mellitus at Kenyatta National Hospital, Nairobi. East Afr Med J 82(12 Suppl):S173–S179
- Sarma AV, St. Sauver JL, Hollingsworth JM, Jacobson DJ, McGree ME, Dunn RL, Lieber MM, Jacobsen SJ, the Urologic Diseases in America Project (2012) Diabetes treatment and progression of benign prostatic hyperplasia in community-dwelling Black and White Men. Urology 79(1):102–108
- 15. Wallner LP, Hollingsworth JM, Dunn RL, Kim C, Herman WH, Sarma AV, the Urologic Diseases of America Project. Hyperglycemia (2013) Hyperglycemia, hyperinsulinemia, insulin resistance, and the risk of BPH/LUTS severity and progression over time in community dwelling black men: the Flint Men's health study. Urology 82:881–886
- Kwon H, Kang HC, Lee JH (2013) Relationship between predictors of the risk of clinical progression of benign prostatic hyperplasia and metabolic syndrome in men with moderate to severe lower urinary tract symptoms. Urology 81:1325–1329
- 17. Hammarsten J, Högstedt B (2001) Hyperinsulinemia as a risk factor for developing benign prostatic hyperplasia. Eur Urol 39(2):151–158
- Kim WT, Yun SJ, Choi YD, Kim GY, Moon SK, Choi YH, Kim IY, Kim WJ (2011) Prostate size correlates with fasting blood glucose in non-diabetic benign prostatic hyperplasia patients with normal testosterone levels. J Kor Med Sci 26:1214–1218
- Kim SB, Cho IC, Min SK (2014) Prostate volume measurement by transrectal ultrasonography: comparison of height obtained by use of transaxial and midsagittal scanning. Korean J Urol 55:470–474
- Yang HJ, Doo SW, Yang WJ, Song YS (2012) Which obesity index best correlates with prostate volume, prostate-specific antigen, and lower urinary tract symptoms? Urology 80:187–190
- 21. Prando R, Cheli V, Melga P, Giusti R, Ciuchi E, Odetti P (1998) Is type 2 diabetes a different disease in obese and non-obese patients? Diabetes Care 21(10):1680–1685
- Napoli N, Mottini G, Arigliani M, Creta A, Giua R, Incammisa A, Carotti S, Sihom F, Yimagou I, Alombah R, Mbanya JC, Pozzilli P (2010) Unexpectedly high rates of obesity and dysglycemia among villagers in Cameroon. Diabetes Metab Res Rev 26:10–12
- Kalk WJ, Joffe BI, Sumner AE (2011) The waist circumference of risk in Black South African Men is lower than in men of european ancestry. Metab Syndr Relat Disord 9(6):491–495
- Kramer G, Mitteregger D, Marberger M (2007) Is benign prostatic hyperplasia (BPH) an immune inflammatory disease? Eur Urol 51:1202–1216
- Matzkin H, Soloway MS (1993) Cigarette smoking: a review of possible associations with benign prostatic hyperplasia and prostate cancer. Prostate 22(4):277–290
- Hermans MP, Amoussou-Guenou KD, Bouenizabila E, Sadikot SS, Ahn SA, Rousseau MF (2016) The normal-weight type 2 diabetes phenotype revisited. Diab Met Syndr Clin Res Rev 10(2 Suppl 1):82–88
- Daniel HW (1993) Larger prostatic adenomas in obese men with no associated increase in obstructive uropathy. J Urol 149(2):315–317
- Dehout F, Haumont S, Gaham N, Amoussou-Guenou KD, Hermans MP (2008) Metabolic syndrome in Bantu subjects with type 2 diabetes from sub-Saharan extraction Prevalence, gender differences and HOMA hyperbolic product. Diab Met Syndr Clin Res Rev 2:5–11
- Kachunga P, Masumbuko B, Belma M, Kashongwe Z, Hermans M, M'buyamba JR (2012) Age and living in an urban environment are major determinants of diabetes among South Kivu Congolese adults. Diabetes Metab 38:324–331
- Choukem SP, Sobngwi E, Gautier JF (2007) Les particularités du diabète chez le sujet originaire d'Afrique noire. Sang Thrombose Vaisseaux 19(10):513–518
- Ding J, Qi L, Zu X, Shen P (2010) Urodynamic studies on benign prostatic hyperplasia combined with diabetes mellitus. J Central South Univ 35(7):705–710

- Stamatiou K, Lardas M, Kostakos E, Koutsonasios V, Michail E (2009) The impact of diabetes type 2 in the pathogenesis of benign prostatic hyperplasia: a review. Adv Urol 2009;818965
- Ozcan L, Besiroglu H, Dursun M, Polat EC, Otunctemur A, Ozbek E (2017) Comparison of the clinical parameters of benign prostate hyperplasia in diabetic and non-diabetic patients. Arch Ital Urol Androl 89(1):26–30
- Ryl A, Rotter I, Slojewski M, Dolegowska B, Grabowska M, Baranowska-Bosiacka I, Laszczynska M (2015) Hormone concentration, metabolic disorders and immunoexpression of androgen and estrogen-alpha

receptors in men with benign prostatic hyperplasia and testosterone deficiency syndrome. Folia Histochem Cytobiol 53(3):227–235

 Sciarra F, Toscano V (2000) Role of estrogens in human benign prostatic hyperplasia. Arch Androl 44(3):213–220

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Submit your manuscript to a SpringerOpen[™] journal and benefit from:

- Convenient online submission
- ► Rigorous peer review
- ► Open access: articles freely available online
- ► High visibility within the field
- ▶ Retaining the copyright to your article

Submit your next manuscript at ► springeropen.com