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The Relationship Between Metabolic Syndrome and Benign Prostate Enlargement: A Case–Control Study in a Peruvian Military Hospital

ABSTRACT

Objective: This study aimed to determine how metabolic syndrome is related to benign prostatic enlargement in males under 60 years old in a national military hospital in Peru.

Materials and methods: We conducted a retrospective, quantitative, case–control study. By simple random sampling, 87 cases with benign prostatic enlargement and 174 controls were included, with a statistical power of 80%. The benign prostatic enlargement was evaluated by clinical picture and ultrasound and the metabolic syndrome was evaluated according to the Adult Treatment Panel III criteria. The statistical analysis was performed using the STATAv14 program, the chi-square statistical test was used and odds ratio was obtained, at a significance level of 5%.

Results: The mean age of the cases and controls was 55 (51-58) and 52 (46-57), respectively. By multivariate analysis, the factors related to benign prostatic enlargement were the presence of benign prostatic enlargement (adjusted odds ratio: 2.71, 95% CI: 1.27-5.80; $P = .010$), waist circumference ≥102 cm (adjusted odds ratio: 6.51, 95% CI: 3.09-13.71; $P < .001$), elevated fasting glucose (adjusted odds ratio: 1.38, 95% CI: 0.65-2.91; $P = .399$), high triglycerides (adjusted odds ratio: 5.29, 95%: CI 2.40-11.64; $P < .001$), and arterial hypertension (adjusted odds ratio: 4.67, 95% CI 2.19-9.95; $P < .001$). Elevated high-density lipoprotein cholesterol was a protective factor (adjusted odds ratio: 0.09, 95% CI: 0.04-0.20; $P < .001$).

Conclusion: The present study showed that metabolic syndrome and its components (waist circumference, hypertension, triglycerides, and high-density lipoprotein cholesterol) are factors related to benign prostatic enlargement in patients under 60 years old in a military hospital in Peruvian population. Waist circumference as an indicator of overweight/obesity is a practical anthropometric marker of interest in public health.

Keywords: Risk factors, benign prostatic hyperplasia, hypertension, waist circumference, hypertriglyceridemia

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Introduction

Benign prostatic enlargement (BPE) is a common benign disease in middle-aged and older men. On many occasions, it may cause symptoms that affect the quality of life and require medical attention or even surgical interventions.[1]

The American Association of Urology (AAU) defines the variable "benign prostatic enlargement" as a clinical condition with prostatic symptoms in the patient and an increase in prostate volume by ultrasound of >30 mL that may require treatment according to medical judgment. Within BPE, the most frequent cause is benign prostatic hyperplasia (BPH), which is usually confirmed with a biopsy as an invasive procedure.[1]

In Europe, there is a calculated prevalence of BPE of 11.8% among the population of men aged 40 years and over. In addition, it occupies the first place as cause of urological

consultations and is considered the second cause of surgical intervention in patients over 50 years.[2] In the USA, it affects approximately 70% of American men aged 60-69 years and 80% of those older than 70 years.[3] In Colombia, it is estimated that for every 100 men seen in urology service clinics, 1% is hospitalized for BPE[4]; Ecuador presented 4001 annual cases in its registries and cataloged prostatic hyperplasia as the second cause of morbidity in people over 65 years of age.[5]

In hospitals pertaining to the Peruvian Health System, 50% of patients in urology services are over 40 years of age and have symptoms due to benign prostatic growth; this represents the first cause of care in urology clinics, being an important cause of hospitalized patients.[6]

Gerald Reaven was the first to describe metabolic syndrome (MetS) in 1988.[7] It describes a complex set of disorders related to metabolic aberration that includes central obesity, arterial hypertension, dyslipidemia, insulin resistance with hyperinsulinemia, and glucose intolerance that increases the individual risk of cardiovascular diseases, diabetes, tumors, among others.[8]

On the other hand, MetS is the grouping of cardiovascular risk factors present in a person, such as insulin resistance, high blood pressure, dyslipidemia, type 2 diabetes, and other metabolic abnormalities. Currently, MetS has an elevated prevalence worldwide, with around 47 million inhabitants in the USA alone.[9,10] According to global estimates, there is a prevalence of MetS between 20% and 25% of the adult population worldwide.[11]

Historically, aging and androgens were considered the 2 determining risk factors for BPE. However, the etiology and natural history of BPE are not fully elucidated. In recent years, biological foundations and evidence have been available for the association between MetS and BPE.[12,13]

Due to the growing importance of metabolic changes with prostate enlargement and the fact that limited information is available both in the population under 60[12] and in the Latin American population, it is relevant to develop studies on the subject.[14] The objective of this study was to determine the relationship between MetS and BPE in Peruvian males under the age of 60 years.

Materials and Methods

Ethical Considerations

For the development of this study, international ethical principles were followed in accordance with the Declaration of Helsinki, and the privacy and confidentiality of the patients were respected. The participants provided written informed consent. The study was approved by the Ethics and Research Committee of the Faculty of Medicine of Universidad Ricardo Palma and by the Institutional Permits and Ethics Committee of Hospital Militar Central (Approval no: 4115-2019-FMH-D).

Study Design and Population

This study was carried out as a quantitative, observational, retrospective, analytical, case and control study and was completed in 2021 in the context of VII Medicine Thesis Degree course.[15]

Study Selection

From the Hospital Militar Central Department of Urology, 261 patients were selected between 2016 and 2018. The inclusion criteria for the group of cases were as follows: patients from the Military Hospital (HM) urology department with an ultrasound diagnosis of BPE (prostatic volume > 30 mL), who have a legible and complete clinical history, and who are less than or equal to 60 years of age. Patients ≤ 60 years of age from the HM urology department with an ultrasound not showing benign prostatic growth were included in the control group. Patients with a diagnosis of prostate cancer by biopsy and those with a previous history of surgery related to the urinary bladder or prostate were excluded. Simple random sampling was used. For the calculation of the sample size, the formula proposed by Díaz et al[16] was used. The frequency of exposure among the controls was 26% and the expected odds ratio (OR) was 2.2 as estimated according to the study by Yangua.[17] A 95% CI was used, and the statistical power was 80%. The case–control ratio was 1:2. The sample was made up of 87 patients for the case group and 174 patients for the control group.

An unmatched hospital-based case–control study was conducted from 2016 to 2018. Cases and controls were included from the same source population from the same time period. All cases that met the selection criteria were considered and controls were randomly selected. Other characteristics were described in Table 1.

Study Quality Assessment

The data were processed in the statistical package vSTATA 14, (StataCorp LLC, Tex, USA, RRID:SCR_012763, programs for Windows). Once the data were entered, the quality control of the database was carried out through debugging and consistency, allowing correcting typing errors, as well as avoiding erroneous or missing data, thus reducing information bias.

MAIN POINTS

- *This study was carried out in a Peruvian military population that represents a Latin American context.*
- *Within the metabolic syndrome (MetS), we emphasize that abnormal waist circumference was related to excess weight, and the elevated high-density cholesterol is a protective factor possibly related to the physical activity of the study population.*
- *A significant relationship between MetS and benign prostatic enlargement in Peruvian men under 60 years of age was evidenced by both bivariate and multivariate analysis.*

Table 1. Components That Make Up the Metabolic Syndrome, According to the National Cholesterol Education Program-Adult Treatment (ATP III)

	ATP III (2005 Revision)
Triglycerides greater than or equal to 150 mg/dL or pharmacological treatment	X
HDL-C lower than 40 mg/dL in men and 50 mg/dL in women or pharmacological treatment	X
Blood pressure greater than 130/85 mmHg or pharmacological treatment	X
Fasting glucose greater than 100 mg/dL or pharmacological treatment	X
Abdominal obesity (≥ 102 cm in male)	X
Criteria	≥3

ATP III, Adult Treatment Panel III; HDL-C, high-density lipoprotein cholesterol.

Statistical Analysis

Descriptive Analysis

For quantitative variables, measures of central tendency (mean) and measures of dispersion (standard deviation or interquartile range) were used, and qualitative variables were analyzed using absolute and relative frequencies.

Inferential Analysis

To determine risk factors for BPE, bivariate and multivariate analyses were performed. The binary logistic regression model of the statistical software STATA version 14 was used. The chi-square test was used, and to demonstrate the association between BPE and MetS, an adjusted and crude (un-adjusted) OR was calculated, with their respective CIs and *P* value. A statistical significance level of 5% was considered.

Definitions and Procedures

A data collection sheet was prepared according to the 5 variables contained in MetS, according to Adult Treatment Panel III (ATP III) (high-density cholesterol (HDL-C), triglycerides, fasting glucose, waist circumference, and blood pressure). Waist circumference was determined at the midpoint between the rib cage and the top of the iliac crest. The cut-off point used was 102 cm. A mercury sphygmomanometer was used to measure blood pressure. Chemical samples were obtained from venous blood after 12 hours of fasting. The cut-off points used to define normal values for glucose, triglycerides, and corresponding HDL-C were obtained from the ATP III of the National Cholesterol Education Program. The reference parameters used in the present study are shown in Table 1, considering MetS when they fulfilled more than 3 criteria.[8,18]

We defined subjects with BPE according to the American Urology Guideline[1] and according to the criteria used by Zhao et al[13] for case group. Criteria for defining BPE included men 45 years of age or older; sustained clinical manifestations of the lower urinary tract (International Prostate Symptoms Score >7), self-administered questionnaire obtained from the clinical history of the patients; and enlargement of the prostate according to ultrasound (total prostate volume >30 mL).

Results

Of the 261 patients included in the study (87 cases and 174 controls), the mean age of the cases was 55 years (51-58) and that of controls was 52 years (46-58). The other characteristics are mentioned in Table 2.

In the bivariate analysis (Table 3), a significant relation was found between the presence of MetS and BPE, as well as for each of the components of the MetS (waist circumference, triglycerides, glucose value, high blood pressure, and HDL-C levels).

In Table 4, the multivariate analysis was performed and it was found that the categorical independent variables were related to BPE. In general, MetS presents a significant relationship (ORa: 2.71, 95% CI: 1.27-5.80; $P = .010$).

In the multivariate analysis of the numerical variables (Table 4), it was found that the increased waist circumference presented an ORa of 6.51, 95% CI: 3.09-13.71; $P < .001$ for the BPE; likewise, triglyceride values greater than or equal to 150 mg/dL showed an ORa of 5.29, 95% CI: 2.40-11.64; $P < .001$, high blood pressure ≥ 130/85 mmHg presented an ORa of 4.67, 95% CI: 2.19-9.95; $P < .001$, showing significant association in relation to BPE. High-density lipoprotein cholesterol level greater than or equal to 40 mg/dL presented an ORa of 0.09, 95% CI: 0.04-0.20; $P < .001$ corresponding to a protective factor. High fasting glucose showed an ORa of 1.38, 95% CI: 0.65-2.91; $P = .399$, not significant.

Table 2. Description of the Characteristics of the Hospital Military Population

Independent Variables	Mean	Range
Age–Cases	55 ± 3 years	(52-58)
Age–Controls	52 ± 6 years	(46-58)
	Frequency	**Percentage**
Metabolic syndrome		
Yes	133	51.0
No	128	49.0
Rank		
Officer	87	33.3
Petty officer	169	64.8
Volunteer service	5	1.9
Marital status		
Single	73	28.0
Living common law	41	15.7
Married	147	56.3
No. of children		
None	57	21.8
One	58	22.2
Two	70	26.8
Three or more	76	29.1
Waist circumference		
≥ 102 cm	112	42.9
< 102 cm	149	57.1
HDL cholesterol		
≥ 40 mg/dL	122	46.7
<40 mg/dL	139	53.3
Fasting glucose		
≥ 100 mg/dL	137	52.5
< 100 mg/dL	124	47.5
Hypertension		
Yes	83	31.8
No	178	68.2
Triglycerides		
≥ 150 mg/dL	77	29.5
< 150 mg/dL	184	70.5

HDL, high-density lipoprotein.

Discussion

Both bivariate and multivariate analysis showed a significant relationship between MetS and BPE in Peruvian men under 60 years of age treated in a national military hospital.

The results of the study show that males with a diagnosis of MetS presented 2.7 times more risk of presenting BPE than those without a diagnosis of MetS. These results coincide with the meta-analysis

Table 3. Bivariate Analysis Between Benign Prostatic Enlargement and the Independent Variables Studied

	Benign Prostatic Enlargement			Crude OR	
Independent Variables	**Yes (n = 87)**	**No (n = 174)**	**Total (n = 261)**	**OR (95% CI)**	***P***
Metabolic syndrome					
Yes	60 (69.0%)	73 (42.0%)	133 (51.0%)	3.01 (1.78-5.30)	<.001
No	27 (31.0%)	101 (58.0%)	128 (49.0%)	Ref.	
Waist circumference					
≥ 102 cm	61 (70.1%)	51 (29.3%)	112 (42.9%)	5.66 (3.22-9.94)	<.001
<102 cm	26 (29.9%)	123 (70.7%)	149 (57.1%)	Ref.	
HDL cholesterol					
≥ 40 mg/dL	12 (13.8%)	110 (63.2%)	122 (46.7%)	0.09 (0.05-0.18)	<.001
<40 mg/dL	75 (86.2%)	64 (36.8%)	139 (53.3%)	Ref.	
Fasting glucose					
≥100 mg/dL	56 (64.4%)	81 (46.6%)	137 (52.5%)	2.07 (1.22-3.53)	.007
<100 mg/dL	31 (35.6%)	93 (53.4%)	124 (47.5%)	Ref.	
Hypertension					
Yes	46 (52.9%)	37 (21.3%)	83 (31.8%)	4.15 (2.38-7.24)	<.001
No	41 (47.1%)	137 (78.7%)	178 (68.2%)	Ref.	
Triglycerides					
≥150 mg/dL	42 (48.3%)	35 (20.1%)	77 (29.5%)	3.71 (2.12-6.49)	<.001
<150 mg/dL	45 (51.7%)	139 (79.9%)	184 (70.5%)	Ref.	

HDL, high-density lipoprotein; OR, odds ratio.

Table 4. Multivariate Analysis Between Benign Prostatic Enlargement and the Variables That Were Significant in the Bivariate Analysis

	Benign Prostatic Enlargement			Adjusted OR	
Independent Variables	**Yes (n = 87)**	**No (n = 174)**	**Total (n = 261)**	**OR (95% CI)**	***P***
Metabolic syndrome					
Yes	60 (69.0%)	73 (42.0%)	133 (51.0%)	2.71 (1.27-5.80)	.010
No	27 (31.0%)	101 (58.0%)	128 (49.0%)	Ref.	
Waist circumference					
≥102 cm	61 (70.1%)	51 (29.3%)	112 (42.9%)	6.51 (3.09-13.71)	<.001
<102 cm	26 (29.9%)	123 (70.7%)	149 (57.1%)	Ref.	
HDL cholesterol					
≥40 mg/dL	12 (13.8%)	110 (63.2%)	122 (46.7%)	0.09 (0.04-0.20)	<.001
<40 mg/dL	75 (86.2%)	64 (36.8%)	139 (53.3%)	Ref.	
Fasting glucose					
≥100 mg/dL	56 (64.4%)	81 (46.6%)	137 (52.5%)	1.38 (0.65-2.91)	.399
<100 mg/dL	31 (35.6%)	93 (53.4%)	124 (47.5%)	Ref.	
Hypertension					
Yes	46 (52.9%)	37 (21.3%)	83 (31.8%)	4.67 (2.19-9.95)	<.001
No	41 (47.1%)	137 (78.7%)	178 (68.2%)	Ref.	
Triglycerides					
≥150 mg/dL	42 (48.3%)	35 (20.1%)	77 (29.5%)	5.29 (2.40-11.64)	<.001
<150 mg/dL	45 (51.7%)	139 (79.9%)	184 (70.5%)	Ref.	

HDL, high-density lipoprotein; OR, odds ratio.

carried out by Zou et al[19] where patients with MetS have an increased risk for BPE. The cohort study conducted by Zhao et al[13] showed a significant relationship between MetS and a higher risk of BPE (HR: 1.29; 95% CI, 1.08–1.50; $P < .001$). Additionally, the study carried out by Nandy,[20] in a hospital of the Indian Armed Forces, with a similar population to the one in this research and the study carried out by Huang et al[21] in patients around 50 years old have similar conclusions. Some systematic reviews have shown that metabolic disorders induced by MetS play a fundamental role in the development of BPE.[22,23]

High-density lipoprotein cholesterol level greater than 40 mg/dL is a protective factor, obtaining an ORa of 0.09. Zhang et al[9] showed that low HDL-C is considered a related factor for prostate enlargement in elderly Chinese men. In a prospective cohort, a relationship between low HDL-C levels and BPE was evidenced by multivariate analysis with a HR of 1.56 (95% CI, 1.08–2.04; $P = .012$).[13] It has been reported that elevated levels of testosterone can suppress HDL-C either by increasing hepatic triglyceride lipase activity or by enhancing catabolism of HDL-C particles. Insulin resistance can also lead

to dyslipidemia characterized by high triglycerides and low HDL cholesterol.[24]

In our study, the association between elevated levels of triglycerides and BPE was 5 times its value, similar to another study,[25] emphasizing the importance of the association between hypertriglyceridemia and the risk of prostate cancer.[25]

We also found that arterial hypertension was associated with BPE with an ORa more than 4 times its value in several epidemiological studies where treated patients with high blood pressure had a larger prostate volume (mean: 51.0 vs. 44.0 mL; $P = .003$) and a higher annual BPE rate (mean: 1.06 vs. 0.90 mL/year; $P = .002$) with significantly higher odds of urinary tract symptoms lower than their counterparts without such a history (OR: 1.8; 95% CI: 1.2-2.6).[26]

On the other hand, a finding to emphasize was 6.51 times more waist circumference with an aOR with the other components, in other words, it had a statistically significant risk associated with BPE, similar to the studies reviewed.[9,17] In a prospective cohort study, a significant association was found in the multivariate analysis between patients with central obesity and BPE with an HR of 1.93,[13] similar to other studies.[21,27] The practical implications and possible applications of this anthropometric indicator must be evaluated in the public health context.

Worldwide, diabetes, obesity, and their related disorders are among the main reasons for medical consultation. Recent evidence from epidemiological and basic research, as well as from translational and clinical intervention studies, supports the emerging hypothesis that the MetS may be an important etiological factor for the initiation of tumors. Insulin resistance is considered the underlying pathophysiological mechanism and is related to other components of the syndrome. Insulin resistance is a prediabetic state and is strongly linked to obesity.[9]

At the biochemical and biomolecular level, several systemic disorders are associated with MetS, including a systemic pro-inflammatory state, altered sex steroids, elevated IGF-1, and autonomic hyperactivity, which have been implicated in the course of BPE. However, this association may be influenced by various factors such as racial and ethnic differences, comorbidities, exposure time, genetic profiles, etc.[28]

Evidence indicates that chronic inflammation is a major mechanism involved in the connection between MetS and BPE. Recently, the importance of prostatic inflammation and metabolic parameters has been recognized.[29] Along with infectious agents, prostate inflammation can be triggered by metabolic stimuli, such as dyslipidemia, an important component of the metabolic syndrome.[30]

Although we have strong evidence of the biological plausibility between MetS and BPE, the relationship found in epidemiological studies has not always been linear, homogeneous, or unifactorial. However, it is important to note that neoplastic and preneoplastic diseases are multifactorial, multivalent, and multidirectional.[31]

Adipose tissue has a plethora of functions. Adipogenesis, angiogenesis, and cell proliferation have been linked. Various endocrine, paracrine, and metabolic mechanisms have been described for adipose tissue. Several bioactive substances such as leptin, adiponectin, tumor necrosis factor, interleukin-6, C-reactive protein, fibrinogen, and plasminogen inhibitor have been implicated.[31]

Although, in recent years, some studies have reported no association between MetS and BPE,[9,32] however, both clinical and preclinical investigations consider the connection between MetS and BPE. Its implications are of interest to readers in various fields and include clinicians and research scientists in the search for potential intervention and prevention targets.

Several studies support the notion that the MetS may be an important target for the prevention and intervention of BPE. Emerging evidence indicates that various factors related to lifestyles such as obesity, diet, and lack of physical activity would have an important role in the etiology of BPE. In addition, it continues to be recognized that BPE is at least partially preventable.[20]

A question to ponder is whether these 2 phenomena MetS and BPE are simply a direct consequence[33] of aging or actually reflect the accumulated and dynamic interaction between the genome of individuals and the ambiome. This interactive dialogue is exercised by people's lifestyle habits and, in this case, by men, from birth through the first 5 or 6 decades of life.[34] The impact of BPE on the quality of life and health of the male population has led to the study and analysis of potential behavioral factors for BPE. Given the current epidemic numbers of MetS, the use of this connection with the BPE could have profound implications in terms of reducing the magnitude of the disease in terms of public health.[31]

The present study showed that MetS and its components (waist circumference, hypertension, triglycerides, and HDL-C) are factors related to BPE in Peruvian men patients under 60 years of age in a military hospital. Waist circumference as an indicator of overweight/obesity is a practical anthropometric marker of interest in public health.

Limitations

Within the limitations of this research, we share those of a retrospective, single-center study. Further studies are needed to confirm the said data. As it corresponds to a military hospital, it presents a possible selection bias. However, the results could be applicable to similar populations. In this study, the variable antidiabetic pharmacological treatment that the patients could have received in relation to fasting glucose levels was not evaluated. The protective effect of HDL-C could be related to the beneficial effect that the studied population shares, due to the physical activity that this type of military population usually develops. Including the evaluation of serum and molecular inflammatory markers in this population could increase our understanding of this topic.

Data Sharing Statement

Restrictions apply to the availability of these data which were used under license for the current study and so are not publicly available. However, data are available from the authors upon reasonable request.

Ethics Committee Approval: *Ethical committee approval was received from the Ethics Committee of Universidad Ricardo Palma (Approval no: 4115-2019-FMH-D).*

Informed Consent: *Written informed consent was obtained from all participants who participated in this study.*

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References

1. Dornbier R, Pahouja G, Branch J, McVary KT. The new American Urological Association benign prostatic hyperplasia clinical guidelines: 2019 update. *Curr Urol Rep*. 2020;21(9):32. [CrossRef]
2. Cozar JM, Solsona E, Brenes F, et al. Manejo asistencial del paciente con hiperplasia benigna de próstata en España. *Actas Urol Esp*. 2011;35(10):580-588. [CrossRef]
3. Medical treatment of benign prostatic hyperplasia. Available at: https://www.uptodate.com/contents/medical-treatment-of-benign-prostatic-hyperplasia?search=BENIGNGPROSTATIC%20GROWTH%20&source=search_result&selectedTitle=1~150&usage_type=default&display_rank=1. Accessed March 15, 2022.
4. Alpi SV, Fernández H, Sierra F, Monsalve M, Cano EJ, Tobón ST. Creencias asociadas a la hiperplasia prostática benigna. *Pensam Psicol*. 2005;1(5):7-16.
5. Álvarez P, Pazmiño L, Villalobos A, Villacís J. *Normas y protocolos de atención integral de salud de las y los adultos mayores*. Quito, Ecuador: Ministerio de Salud pública; 2010. Available at: https://www.academia.edu/24822111/MSP_Normas_y_protocolos_de_atenci%C3%B3n_integral_de_salud_de_los_adultos_mayores. Accessed March 20, 2022.
6. Ortiz-Colorado ME, Bueno-Ordóñez S, Urquiaga-Melquiades DT. Características clínicas y epidemiológicas de pacientes con hiperplasia prostática benigna sintomática en el Hospital Regional Docente de Cajamarca, 2018". *Rev Med Trujillo*. 2021;16(2). Available at: https://revistas.unitru.edu.pe/index.php/RMT/article/view/3631.
7. Reaven GM. The metabolic syndrome: is this diagnosis necessary? *Am J Clin Nutr*. 2006;83(6):1237-1247. [CrossRef]
8. Robles L, Carlos J. Síndrome metabólico: concepto y aplicación práctica. *An Fac Med*. 2013;74(4):315-320.
9. Zhang X, Zeng X, Liu Y, Dong L, Zhao X, Qu X. Impact of metabolic syndrome on benign prostatic hyperplasia in elderly Chinese men. *Urol Int*. 2014;93(2):214-219. [CrossRef]
10. Saklayen MG. The global epidemic of the metabolic syndrome. *Curr Hypertens Rep*. 2018;20(2):12. [CrossRef]
11. Hirode G, Wong RJ. Trends in the prevalence of metabolic syndrome in the United States, 2011-2016. *JAMA*. 2020;323(24):2526-2528. [CrossRef]
12. Wu C, Chen X, Cai Y, et al. Risk factors associated with acute respiratory distress syndrome and death in patients with coronavirus disease 2019 pneumonia in Wuhan, China. *JAMA Intern Med*. 2020;180(7):934-943. [CrossRef]
13. Zhao SC, Xia M, Tang JC, Yan Y. Associations between metabolic syndrome and clinical benign prostatic hyperplasia in a northern urban Han Chinese population: A prospective cohort study. *Sci Rep*. 2016;6(1):33933. [CrossRef]
14. Xu XF, Liu GX, Guo YS, et al. Global, regional, and national incidence and year lived with disability for benign prostatic hyperplasia from 1990 to 2019. *Am J Mens Health*. 2021;15(4):15579883211036786. [CrossRef]
15. De La Cruz-Vargas JA, Correa-Lopez LE, Alatrista-Gutierrez de Bambaren M del S, Sanchez Carlessi, HH Luna Muñoz C, Loo ValverdeM, et al. Promoviendo la investigación en estudiantes de Medicina y elevando la producción científica en las universidades: experiencia del Curso Taller de Titulación por Tesis. *Educ Médica*. 2019;20(4):199–205. [CrossRef]
16. Díaz P. Cálculo del poder estadístico de un estudio. Available at: https://www.aefa.es/wp-content/uploads/2014/04/C%C3%A1lculo-del-poder-estad%C3%ADstico-de-un-estudio.pdf.
17. Yangua Oliva CR. Síndrome metabólico como factor asociado a hiperplasia benigna de próstata. Universidad Privada Antenor Orrego - UPAO; 2018. Available at: https://repositorio.upao.edu.pe/handle/20.500.12759/3906. Accessed March 20, 2022.
18. Domínguez-Reyes T, Quiroz-Vargas I, Salgado-Bernabé AB, Salgado-Goytia L, Muñoz-Valle JF, Parra-Rojas I. Las medidas antropométricas como indicadores predictivos de riesgo metabólico en una población mexicana. *Nutr Hosp*. 2017;34(1):96-101. [CrossRef]
19. Zou C, Gong D, Fang N, Fan Y. Meta-analysis of metabolic syndrome and benign prostatic hyperplasia in Chinese patients. *World J Urol*. 2016;34(2):281-289. [CrossRef]
20. Nandy PR, Saha S. Association between components of metabolic syndrome and prostatic enlargement: an Indian perspective. *Med J Armed Forces India*. 2016;72(4):350-355. [CrossRef]
21. Huang MJ, Yang YY, Chen C, et al. Comparison of the predictive value of anthropometric indicators for the risk of benign prostatic hyperplasia in southern China. *Asian J Androl*. 2022. [CrossRef]
22. Omran A, Leca BM, Oštarijaš E, et al. Metabolic syndrome is associated with prostate enlargement: a systematic review, meta-analysis, and meta-regression on patients with lower urinary tract symptom factors. *Ther Adv Endocrinol Metab*. 2021;12. [CrossRef]
23. Li J, Peng L, Cao D, Gou H, Li Y, Wei Q. The association between metabolic syndrome and benign prostatic hyperplasia: a systematic review and meta-analysis. *Aging Male*. 2020;23(5):1388-1399. [CrossRef]
24. Baykam MM, Aktas BK, Bulut S, et al. Association between prostatic resistive index and cardiovascular risk factors in patients with benign prostatic hyperplasia. *Kaohsiung J Med Sci*. 2015;31(4):194-198. [CrossRef]
25. Chhetri RK, Baral S, Thapa N. Metabolic syndrome and benign prostatic hyperplasia. *J Lumbini Med Coll*. 2018;6(2):58-61. [CrossRef]
26. Abdollah F, Briganti A, Suardi N, et al. Metabolic syndrome and benign prostatic hyperplasia: evidence of a potential relationship, hypothesized etiology, and prevention. *Korean J Urol*. 2011;52(8):507-516. [CrossRef]
27. Mampa E, Haffejee M, Fru P. The correlation between obesity and prostate volume in patients with benign prostatic hyperplasia at Charlotte Maxeke Johannesburg Academic Hospital. *Afr J Urol*. 2021;27(1):60. [CrossRef]
28. Breyer BN, Sarma AV. Hyperglycemia and insulin resistance and the risk of BPH/LUTS: an update of recent literature. *Curr Urol Rep*. 2014;15(12):462. [CrossRef]
29. Madersbacher S, Sampson N, Culig Z. Pathophysiology of benign prostatic hyperplasia and benign prostatic enlargement: A mini-review. *Gerontology*. 2019;65(5):458-464. [CrossRef]
30. Rastrelli G, Vignozzi L, Corona G, Maggi M. Testosterone and benign prostatic hyperplasia. *Sex Med Rev*. 2019;7(2):259-271. [CrossRef]
31. Lifshitz K, Ber Y, Margel D. Role of metabolic syndrome in prostate cancer development. *Eur Urol Focus*. 2021;7(3):508-512. [CrossRef]
32. Gacci M, Sebastianelli A, Salvi M, et al. Central obesity is predictive of persistent storage lower urinary tract symptoms (LUTS) after surgery for benign prostatic enlargement: results of a multicentre prospective study. *BJU Int*. 2015;116(2):271-277. [CrossRef]
33. Doğan Y, Uruç F, Aras B, et al. The relationships between metabolic syndrome, erectile dysfunction and lower urinary tract symptoms associated with benign prostatic hyperplasia. *Turk J Urol*. 2015;41(1):7-12. [CrossRef]
34. Ausfeld MJR, Rabito AA. Relación entre el estado nutricional y el síndrome metabólico en adultos. *Mem Inst Investig Cienc Salud*. 2015;13(2). Available at: http://archivo.bc.una.py/index.php/RIIC/article/view/600.