Submitted: 27 September, 2023 Accepted: 28 November, 2023 Published: 30 January, 2024 DOI:10.22514/jomh.2024.014

ORIGINAL RESEARCH

JOMH Journal of Men's Health

Analysis of risk factors for benign prostate enlargement with prostate calcification

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Abstract

The occurrence of benign prostatic hyperplasia (BPH) accompanied by calcification can reduce the quality of life and treatment effectiveness of patients. This study focuses on middle-aged and elderly patients in physical examination centers, and divides them into a control group and a study group based on their condition. By comparing the general information and serum indicators of the subjects, descriptive analysis methods are used to analyze the risk factors for prostate hyperplasia accompanied by calcification. Among 700 male physical examinations, there were 305 patients with benign prostate enlargement, with a prevalence rate of 43.57%. There were 290 patients with prostate calcification, accounting for 41.43%. Among them, there were 203 patients with prostate calcification in BPH, accounting for 66.56%. Multivariate logistic regression analysis showed that age, education and culture, diabetes, hypertension, uric acid (UA) and blood urea nitrogen (BUN) are independent risk factors for prostate calcification, while height is the protective factor. The prevalence rate of BPH and BPH with prostate calcification is high in middle-aged and elderly men. Although the cause of calcification is still unclear, age, education, occupation, diabetes, hypertension, UA, BUN and prostate-specific antigen (PSA) indicators are closely related to the disease. Therefore, early intervention should be carried out according to the above risk factors to prevent the occurrence of the disease as soon as possible.

Keywords

Benign prostatic enlargement; Prostate calcification; Risk factor analysis

1. Introduction

Benign prostatic hyperplasia (BPH) is a relatively common condition among middle-aged and elderly males. Recent data indicates that its prevalence in this demographic can be as high as 50% in China, with the risk of development increasing annually due to the aging Chinese population [1, 2]. Prostate calcification, also known as prostate calcification lesions or calcified plaques, involves the deposition of calcium salts in the prostate acini without apparent symptoms, primarily affects men aged 40–60 years, and its etiology remains complex and unclear. The presence of prostate calcification can lead to prostate stones, prostatitis and seminal vesiculitis, often co-occurring with prostatitis and BPH [3, 4]. Prostate calcification is typically not visible, and its diagnostic rate varies significantly depending on the technique used, with imaging calcification prevalence ranging from 36% to 71%, while histological identification is only around 30% [5, 6]. Prostatic calcification is generally considered an early sign of true prostatic calculi, and its coexistence with BPH often results in symptoms like urinary frequency, urgency, painful urination and hematuria, which not only impact patients' quality of life but also influence the severity of prostate-related conditions and treatment outcomes [7, 8]. Herein, we designed this study to investigate the incidence of prostate hyperplasia with concurrent prostate calcification and identify associated risk factors in middle-aged and elderly men.

2. Information and methods

2.1 General information

This study assessed the data of middle-aged and elderly men who underwent physical examinations at the Physical Examination Center of the Fifth Affiliated Hospital of Sun Yat-sen University between January 2022 and January 2023. Cases were included if they matched the following inclusion criteria: (1) age above 40 years; (2) underwent blood and urine testing, as well as prostate ultrasound monitoring, as part of their physical examination; (3) had complete physical examination data for study analysis; and (4) were informed about the study and voluntarily agreed to participate by signing the informed consent form. The exclusion criteria were: (1) a previous history of prostate, urethra or bladder surgery; (2) the presence of neurological disorders, urinary tract infections, renal insufficiency or malignancies that could impact urinary function; (3) the existence of prostate nodules or lesions in gonadal

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axis organs; (4) recent or ongoing use of medications that may affect research-related indicators, such as long-term use of drugs influencing blood lipids, which might interfere with the assessment of metabolic syndrome and sexual hormones, as well as 5-α Reductase inhibitors and other medications; (5) had blood total prostate-specific antigen (TPSA) levels exceeding 10 μg/L or blood TPSA levels between 4–10 μg/L with a total free/total PSA ratio less than 0.16; and (6) had communication disorders, incomplete physical examination results or unwillingness to sign the informed consent form.

2.2 Research methods

A total of 700 males were identified, and among them, 305 had prostatic hyperplasia and met the screening criteria. Among these cases, 110 individuals had prostate calcification with benign prostatic hyperplasia (designated as the study group), while 130 cases presented with benign prostatic hyperplasia alone (designated as the control group).

The assessed data included patients' age, height, weight, body mass index (BMI), blood pressure, occupation, metabolic syndrome indicators, blood lipid profiles (comprising high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triacylglycerol (TG) and total cholesterol (TC)), liver function parameters (such as Alanine aminotransferase (ALT) and aspartate transaminase (AST)), diabetes-related fasting blood glucose (FBG), albumin (ALB), total bilirubin (TBA) and total cholesterol (TC). Furthermore, total bilirubin (TBIL), direct bilirubin (DBIL), creatinine (Cr), blood urea nitrogen (BUN) and uric acid (UA) levels were determined using an automated biochemical analyzer. Serum prostate-specific antigen (PSA) levels were quantified using the Roche E601 analysis system and corresponding reagents, with the normal range being 0–4 ng/mL. The data results were provided by the Laboratory Department of our hospital.

2.3 Related diagnostic criteria

The diagnostic criteria for metabolic syndrome comprised (1) Hypertension: Blood pressure $\geq$130/85 mmHg; (2) Hyperglycemia: FBG $\geq$6.1 mmol/L or $\geq$7.8 mmol/L two hours after a meal; (3) Elevated TG $\geq$1.70 mmol/L, TC $\geq$5.20 mmol/L and LDL-C $\geq$3.40 mmol/L; (4) HDL-C $<$1.04 mmol/L; (5) elevated BMI $\geq$28 kg/m^2 or abdominal obesity with a waist circumference $\geq$90 cm in men. BMI $\geq$25 kg/m^2 was considered overweight. A diagnosis of metabolic syndrome was made when an individual was positive for three or more of these criteria [9].

Benign prostatic enlargement (BPE) is defined as the absence of histological evidence of BPH. The normal prostate typically has dimensions of approximately 40 mm (left to right), 30 mm (upper to lower), and 20 mm (anterior to posterior). The internal and external prostate gland ratio is normally around 1:1 but becomes imbalanced during hyperplasia and the presence of hyperplastic nodules. Either of these conditions can lead to a diagnosis of BPE. Prostate calcification was diagnosed through ultrasound imaging, where dense, echogenic spots, masses or light areas were observed. Regardless of the presence of silent shadows, these are displayed at the junction of the internal and external glands of the prostate, serving as diagnostic criteria [10]. The transabdominal ultrasound examination method requires filling the bladder before examination. The ultrasound probe is placed in the lower abdomen, and sound waves pass through the bladder to detect the prostate and measure its size.

2.4 Statistical methods

All data were compiled using Microsoft Excel (2019, Microsoft, Beijing, China) and analyzed using SPSS 23.0. (SPSS Inc., Chicago, IL, USA) Categorical data are presented as counts (percentages) and analyzed using the chi-square test. Continuous data of normal distribution were assessed using independent sample *t*-tests. Multivariate logistic analysis was performed to identify factors influencing benign prostatic enlargement with prostatic calcification. Results with *p*-values less than 0.05 were considered statistically significant.

3. Results

3.1 Detection rate of prostatic hyperplasia and prostatic calcification

In the initial cohort of 700 male individuals who underwent physical examinations, 305 cases of BPH were identified, representing 43.57% of the participants. Furthermore, 290 cases of calcified prostate were observed, accounting for 41.43% of the total cases. Notably, among these patients, 203 had calcified prostate with BPH, constituting 66.56% of the overall study population.

3.2 Comparison of the general information of the study subjects

Table 1 presents the general characteristics of the subjects in both groups. Comparative analysis reveals statistically significant differences in age, education, culture, occupation, diabetes, hypertension and smoking history between the two groups ($p < 0.05$). However, there were no statistically significant differences in occupation, education level, marital status, metabolic syndrome, history of alcohol consumption and the presence of prostate cysts, except for education and culture ($p > 0.05$).

3.3 Comparison of index levels between the two groups of subjects

Table 2 displays the index levels of the subjects in the two groups. Statistically significant differences were observed in height, systolic blood pressure, TBIL, UA, BUN and PSA levels between the two groups ($p < 0.05$). Conversely, BMI, diastolic blood pressure and the levels of TG, TC, HDL-C, LDL-C, AST, ALT, ALB, DBIL, FBG and Cr were comparable between the groups ($p > 0.05$).

3.4 Multivariate logistic regression analysis

Age, education and culture, diabetes, hypertension, smoking history, height, systolic blood pressure, TBIL, UA, BUN and PSA, which were significant factors in univariate analysis,

TABLE 1. Baseline characteristics of the two study groups.

Baseline characteristics	Control group (n = 130)	Study group (n = 110)	*t* value	*p*-value
Age				
40–49	23 (17.69)	37 (33.64)	16.485	0.001
50–59	42 (32.31)	17 (15.45)		
60–69	48 (36.92)	49 (44.55)		
≥70	17 (13.08)	7 (6.36)		
Career				
Water and electricity industry	16 (12.31)	21 (19.09)	2.102	0.147
Manufacturing	22 (16.92)	12 (10.91)	1.772	0.183
Building Construction	21 (16.15)	13 (11.82)	0.921	0.337
Education and Culture	19 (14.62)	28 (25.45)	4.445	0.035
Medical and Health	20 (15.38)	14 (12.73)	0.346	0.556
Agriculture	18 (13.85)	12 (10.91)	0.470	0.493
Freelance	14 (10.77)	10 (9.09)	0.186	0.666
Education level				
Junior high school and below	8 (6.15)	9 (8.18)	0.443	0.802
High school and junior college	45 (34.62)	39 (35.45)		
College and above	77 (59.23)	62 (56.36)		
Marital status				
Married	92 (70.77)	81 (73.64)	0.243	0.622
Single	38 (29.23)	29 (26.36)		
Diabetes				
Yes	14 (10.77)	22 (20.00)	3.982	0.046
No	116 (89.23)	88 (80.00)		
High blood pressure				
Yes	13 (10.00)	29 (26.36)	11.051	0.001
No	117 (90.00)	81 (73.64)		
Metabolic syndrome				
Yes	11 (8.46)	15 (13.64)	1.652	0.199
No	119 (91.54)	95 (86.36)		
Smoking history				
Yes	28 (21.54)	39 (35.45)	5.734	0.017
No	102 (78.46)	71 (64.55)		
History of alcohol consumption				
Yes	47 (36.15)	42 (38.18)	0.105	0.746
No	83 (63.85)	68 (61.82)		
Prostate cysts				
Yes	7 (5.38)	5 (4.55)	0.088	0.766
No	123 (94.62)	105 (95.45)		

TABLE 2. Comparison of index levels between the two groups of subjects.

Indicators	Control Group (n = 130)	Study Group (n = 110)	χ^2 value	*p*-value
Height	171.62 ± 5.90	166.77 ± 5.21	6.701	<0.001
BMI	24.81 ± 2.64	25.10 ± 2.67	0.832	0.406
Systolic blood pressure	124.25 ± 12.76	128.65 ± 14.28	2.519	0.012
Diastolic blood pressure	79.75 ± 8.89	81.62 ± 7.48	1.748	0.082
TG (mmol/L)	1.51 ± 0.28	1.54 ± 0.34	0.781	0.436
TC (mmol/L)	4.84 ± 0.63	4.89 ± 0.62	0.597	0.551
HDL-C (mmol/L)	1.24 ± 0.26	1.25 ± 0.30	0.211	0.833
LDL-C (mmol/L)	2.80 ± 0.44	2.88 ± 0.53	1.391	0.166
AST (U/L)	24.48 ± 2.84	24.05 ± 2.84	1.181	0.239
ALT (U/L)	29.78 ± 2.97	29.77 ± 3.92	0.030	0.976
ALB (g/L)	46.88 ± 4.87	46.13 ± 4.65	1.217	0.225
TBIL (μmol/L)	13.30 ± 2.60	14.76 ± 2.51	4.385	<0.001
DBIL (μmol/L)	3.48 ± 0.64	3.34 ± 0.63	1.639	0.102
FBG (mmol/L)	5.18 ± 0.73	5.32 ± 0.89	1.369	0.172
Cr (mmol/L)	76.38 ± 9.31	77.73 ± 9.54	1.114	0.266
UA (μmol/L)	371.77 ± 58.75	389.46 ± 66.85	2.181	0.030
BUN (mmol/L)	5.12 ± 0.80	5.45 ± 0.93	2.935	0.004
PSA (ng/mL)	1.67 ± 0.27	1.85 ± 0.23	5.731	<0.001

BMI: body mass index; TG: triacylglycerol; TC: total cholesterol; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; AST: aspartate transaminase; ALT: Alanine aminotransferase; ALB: albumin; TBIL: total bilirubin; DBIL: direct bilirubin; FBG: fasting blood glucose; Cr: creatinine; UA: uric acid; BUN: blood urea nitrogen; PSA: prostate-specific antigen.

were included for multifactorial logistic regression analysis, and the results showed that BUN was an independent risk factor for prostate calcification, while height was a protective factor (Table 3). Elevated systolic blood pressure and TBIL levels alone were not associated with prostate calcification ($p > 0.05$).

4. Discussion

Prostate cancer is one of the most frequently diagnosed malignancies among men worldwide, and prostate microcalcification has garnered attention as a potential prognostic marker for bone metastasis in this disease [11]. Previous research has indicated that while it often arises as a consequence of scar tissue formation following prostatitis resolution, several factors, including chronic prostatitis, prostatic urine retention, prostatic duct stenosis, abnormalities in calcium and phosphorus metabolism, and psychosocial factors, may contribute to its development [12–14]. Epidemiological studies have also linked poor dietary habits (such as irregular breakfasts and consumption of fatty snacks), alcohol consumption, and certain work-related habits (like prolonged sitting, late-night work, and the habit of holding in urine) to an increased risk of calcification [15, 16]. It was discovered that small calcification foci typically do not impact sperm quality; however, the formation of larger and coarser stones can have adverse effects. Additionally, the occurrence of calcification may be closely associated with the development of other urological conditions, underscoring the importance of routine monitoring of calcification foci [17, 18]. The progression of Benign Prostatic Hyperplasia (BPH) can elevate pressure within the prostatic ducts, lead to duct dilation, hinder glandular secretion flow, and prompt the deposition of stone components on the surrounding compressed cortex or envelope, ultimately resulting in calcification [19]. In a related study, it was observed that as many as 76.7% of patients diagnosed with prostate stones also had concurrent prostatic hyperplasia [20]. Given the unclear etiology of prostatic calcification and the predominantly asymptomatic nature of affected individuals, complete eradication remains challenging. Consequently, it is important to identify the associated risk factors and implement proactive interventions to mitigate its occurrence.

Our analysis revealed a BPH incidence of 43.57% in our local area, with 41.43% of the cohort having prostate calcification. Notably, 203 patients had prostate calcification with BPH, making up 66.56% of the studied group. These findings highlight the substantial prevalence of BPH and BPH with prostatic calcification in the middle-aged and elderly male

TABLE 3. Multivariate logistic regression analysis.

Influencing Factors	B	S. E	Wald	*p*-value	OR (95% confidence interval)
Age	0.077	0.014	29.163	<0.001	1.080 (1.050–1.110)
Education culture	0.691	0.331	4.353	0.037	1.995 (1.043–3.816)
Diabetes	0.728	0.370	3.875	0.049	2.071 (1.003–4.277)
High blood pressure	1.170	0.364	10.348	0.001	3.222 (1.580–6.573)
Smoking history	0.694	0.292	5.644	0.018	2.001 (1.129–3.546)
Height	−0.154	0.027	31.847	<0.001	0.857 (0.813–0.904)
Systolic blood pressure	0.552	0.343	2.586	0.108	1.736 (0.886–3.402)
TBIL	0.485	0.277	3.069	0.080	1.625 (0.944–2.797)
UA	0.536	0.263	4.133	0.042	1.708 (1.019–2.863)
BUN	0.444	0.156	8.081	0.004	1.560 (1.148–2.119)
PSA	3.068	0.614	24.948	<0.001	21.495 (6.450–71.639)

Note: Assignment of categorical variables: dependent variable: 1 = control group, 2 = study group; Independent variables: 1 = junior high school and below, 2 = high school and technical secondary school, 3 = college and above; 0 = no diabetes, 1 = diabetes; 0 = no hypertension, 1 = with hypertension; The test levels for introducing and removing variables are 0.05 and 0.10, respectively.
TBIL: total bilirubin; UA: uric acid; BUN: blood urea nitrogen; PSA: prostate-specific antigen; OR: Odds ratio.

population in our region.

These findings emphasize the importance of men in the region paying attention to their daily lifestyles and dietary habits, as well as undergoing regular medical checkups. Moreover, the results indicate a strong link between prostate gland calcification and the development of BPH. However, it's worth noting that some patients exhibit calcification without BPH, necessitating further investigation into the factors influencing this subgroup. Additionally, the prevalence of calcification in BPH varies across studies, ranging from 36.5% to 70% [21–23], indicating potential geographical differences in the risk of BPH with calcification, which may be associated with variations in local economic conditions, dietary patterns and lifestyle choices.

In recent years, there has been a noticeable year-on-year increase in the incidence of BPH and prostatic calcification [24]. However, there has been limited research on BPH with concurrent prostatic calcification within current clinical practice. Univariate and multifactorial logistic regression analyses revealed that the risk of BPH with calcification rises with advancing age and varies across different occupations, with educators exhibiting a higher likelihood of developing calcification. Moreover, BPH patients with diabetes and hypertension were found to have an increased risk of calcification. When assessing lifestyle factors such as smoking history and alcohol consumption, a significant difference was observed in the smoking history between the two groups, whereas a history of alcohol consumption was only numerically higher in the observation group without statistically significant difference. In terms of index levels, the study group was significantly older and had higher systolic blood pressure, TBIL, UA, BUN and PSA levels than the control group, while height was significantly lower in the control group. Multivariate logistic regression analysis revealed that age, education, diabetes, hypertension, UA, BUN and PSA were independent risk factors for prostate calcification, while height was a protective factor. These results align partially with previous reports, confirming that age (as an independent risk factor) could indeed contribute to increased disease risk, potentially due to age-related decline in organ function [25, 26]. The presence of chronic conditions like diabetes and hypertension may elevate the risk of developing diseases such as prostatitis, which, in turn, can lead to calcification [27, 28]. Generally, elevated uric acid levels are not associated with prostate calcification. Conversely, high levels of urea nitrogen may indicate the presence of conditions like acute and chronic nephritis, renal tuberculosis or renal dysfunction induced by tumors in patients. UA and BUN have been linked to kidney diseases and conditions like hyperuricemia. Additionally, dietary habits, particularly excessive consumption of protein-based foods, could contribute to a simple increase in urea nitrogen levels [29, 30]. PSA is an enzyme present in human prostatic acinar and ductal epithelial cells and is synthesized in normal, cancerous and metastatic cancer cells. It is the primary marker for distinguishing and diagnosing prostate cancer. Notably, we observed a significant overlap in PSA levels between individuals with prostate hyperplasia and those with prostate cancer within the range of 4–10 ng/mL. Studies have also indicated that PSA levels in patients with BPH and calcification may exhibit abnormal elevations. Therefore, for this subset of patients, further evaluation through ultrasound and additional serological indicators is necessary to differentiate prostate cancer [31]. Further, the association between height and prostate calcification, BPH or both remains uncertain, and further research is required to comprehensively understand its potential impact.

5. Conclusions

Age, education, occupation, diabetes, hypertension and elevated UA, BUN and PSA levels were identified as risk factors for BPH with prostate calcification. Early interventions are advisable for individuals with these risk factors, including effective blood glucose and blood pressure control, a balanced diet, regular routines, and physical activity to enhance circulation. Due to limited sample size, individuals with only one year of follow-up were included, and partial data on lower urinary tract symptoms was lacking. Therefore, a larger sample size and more comprehensive patient data are needed for further research to confirm our findings and investigate their potential mechanisms in order to better develop preventive strategies.

AVAILABILITY OF DATA AND MATERIALS

The authors declare that all data supporting the findings of this study are available within the paper and any raw data can be obtained from the corresponding author upon request.

AUTHOR CONTRIBUTIONS

XLC and YZH—designed the study and carried them out; supervised the data collection, analyzed the data, and interpreted the data; prepared the manuscript for publication and reviewed the draft of the manuscript. All authors have read and approved the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Ethical approval was obtained from the Ethics Committee of the Fifth Affiliated Hospital Sun Yat-sen University (Approval no. 2019028). Written informed consent was obtained from a legally authorized representative for anonymized patient information to be published in this article.

ACKNOWLEDGMENT

Not applicable.

FUNDING

This research received no external funding.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES

[1] Miernik A, Gratzke C. Current treatment for benign prostatic hyperplasia. Deutsches Arzteblatt International. 2020; 117: 843–854.

[2] Franco JVA, Jung JH, Imamura M, Borofsky M, Omar MI, Escobar Liquitay CM, *et al*. Minimally invasive treatments for benign prostatic hyperplasia: a Cochrane network meta-analysis. BJU International. 2022; 130: 142–156.

[3] Raynor WY, Borja AJ, Zhang V, Kothekar E, Lau HC, Ng SJ, *et al*. Assessing coronary artery and aortic calcification in patients with prostate cancer using 18F-sodium fluoride PET/computed tomography. PET Clinics. 2022; 17: 653–659.

[4] Kapogiannis F, Fasoulakis K, Fragkoulis C, Aggelopoulos A, Fasoulakis C. Total osseous calcification of the prostate gland. Cureus. 2020; 12: e9239.

[5] Lavi A, Nair SM, Halstuch D, Chin JL. Prostatic dystrophic calcification following salvage cryotherapy for prostate cancer—an under-reported entity? Scandinavian Journal of Urology. 2021; 55: 33–35.

[6] Kusuma Duarsa GW, Sari YA, Gde Oka AA, Santosa KB, Yudiana IW, Wisnu Tirtayasa PM, *et al*. Serum testosterone and prostate-specific antigen levels are major risk factors for prostatic volume increase among benign prostatic hyperplasia patients. Asian Journal of Urology. 2021; 8: 289–297.

[7] Yu SH, Lim DG, Kim S. Clinical significance of distally located periurethral calcification in patients with lower urinary tract symptoms of benign prostate hyperplasia. Asian Journal of Andrology. 2023; 25: 361–365.

[8] Madersbacher S, Sampson N, Culig Z. Pathophysiology of benign prostatic hyperplasia and benign prostatic enlargement: a mini-review. Gerontology. 2019; 65: 458–464.

[9] Lerner LB, McVary KT, Barry MJ, Bixler BR, Dahm P, Das AK, *et al*. Management of lower urinary tract symptoms attributed to benign prostatic hyperplasia: AUA GUIDELINE PART I—initial work-up and medical management. Journal of Urology. 2021; 206: 806–817.

[10] Dornbier R, Pahouja G, Branch J, McVary KT. The new American urological association benign prostatic hyperplasia clinical guidelines: 2019 update. Current Urology Reports. 2020; 21: 32.

[11] Mehra R, Shah T, Liu C, Plouffe KR, Wang X, Mannan R, *et al*. Highly recurrent IDH1 mutations in prostate cancer with psammomatous calcification. Modern Pathology. 2023; 36: 100146.

[12] Zhang X, Xiao L, Zhang L, Zhou J, Hao Z, Yang C, *et al.* Associated factors for prostate enlargement in Chinese adult men aged <40 receiving checkups. International Journal of Clinical Practice. 2022; 2022: 4792451.

[13] Erfani S, Raeisi N, Shakeri S, Askari E. Prostatic bed calcification with MDP uptake: easy to miss on planar images. To be published in Nuclear Medicine Review. Central & Eastern Europe. 2022. [Preprint].

[14] Kao J, Karwowski P, Pettit J, Barney AK, Atalla C. Multiparametric prostate MRI-based intensity-modulated radiation therapy guided by prostatic calcifications. The British Journal of Radiology. 2020; 93: 20200571.

[15] Singh S, Martin E, Tregidgo HFJ, Treeby B, Bandula S. Prostatic calcifications: quantifying occurrence, radiodensity, and spatial distribution in prostate cancer patients. Urologic Oncology: Seminars and Original Investigations. 2021; 39: 728.e1–728.e6.

[16] Ather MM, Aman S, Qamar H, Latif R, Baloch MB, Zehra U. Lower urinary tract symptoms in patients with small prostates: smooth muscle proliferation and calcification might be causative factors. Lower Urinary Tract Symptoms. 2022; 14: 267–272.

[17] Bazzi LA, Sigurdardottir LG, Sigurdsson S, Valdimarsdottir U, Torfadottir J, Aspelund T, *et al*. Exploratory assessment of pineal gland volume, composition, and urinary 6-sulfatoxymelatonin levels on prostate cancer risk. The Prostate. 2021; 81: 487–496.

[18] Thomas J, Khelif A, Arulanantham S, Lemaitre J, Lalive d'Epinay J. Discovery of primary hyperparathyroidism following an endoscopic resection of the prostate: case report. Journal of Surgical Case Reports. 2022; 2022: rjac247.

[19] Leonard AJ, Garg N, Chen TT, Monga M. Case of the Month from UCSD, San Diego, USA: treatment of giant dystrophic prostatic calcification using MOSES holmium laser. BJU International. 2022; 129: 306–308.

[20] Knight GM, Talwar A, Salem R, Mouli S. Systematic review and meta-analysis comparing prostatic artery embolization to gold-standard transurethral resection of the prostate for benign prostatic hyperplasia. CardioVascular and Interventional Radiology. 2021; 44: 183–193.

[21] Lerner LB, McVary KT, Barry MJ, Bixler BR, Dahm P, Das AK, *et al*. Management of lower urinary tract symptoms attributed to benign prostatic hyperplasia: AUA GUIDELINE PART II—surgical evaluation and treatment. Journal of Urology. 2021; 206: 818–826.

[22] Han C, Zhu L, Liu X, Ma S, Liu Y, Wang X. Differential diagnosis of uncommon prostate diseases: combining mpMRI and clinical information. Insights into Imaging. 2021; 12: 79.
[23] Lintin L, Barge T, Boardman P, Tong G, Tapping C. Predictors of technical outcome for prostatic artery embolisation using pre-procedural CT angiography. European Radiology. 2021; 31: 1308–1315.
[24] Agarwal DK, Krambeck AE. Challenging case of holmium laser enucleation of the prostate after prior prostatic focal laser ablations and Rezum. Urology. 2021; 153: 358.
[25] Calogero AE, Burgio G, Condorelli RA, Cannarella R, La Vignera S. Epidemiology and risk factors of lower urinary tract symptoms/benign prostatic hyperplasia and erectile dysfunction. The Aging Male. 2019; 22: 12–19.
[26] Wang X, Su Y, Yang C, Hu Y, Dong J. Benign prostatic hyperplasia and cardiovascular risk: a prospective study among Chinese men. World Journal of Urology. 2022; 40: 177–183.
[27] Lokeshwar SD, Harper BT, Webb E, Jordan A, Dykes TA, Neal Jr DE, *et al*. Epidemiology and treatment modalities for the management of benign prostatic hyperplasia. Translational Andrology and Urology. 2019; 8: 529–539.
[28] Lloyd GL, Ricke WA, McVary KT. Inflammation, voiding and benign prostatic hyperplasia progression. Journal of Urology. 2019; 201: 868–870.
[29] Jin S, Liu Z, Liu Y, Wu J, Yu Q. Analysis of risk factors and nursing strategies for postoperative hemorrhage of benign prostatic hyperplasia. Evidence-Based Complementary and Alternative Medicine. 2022; 2022: 4205015.
[30] Xu B, Liu M, Liu Y, Zuo J. Risk factors of urinary pathogenic bacteria infection after benign prostatic hyperplasia surgery and curative effect analysis of Shuangdong capsule intervention. Emergency Medicine International. 2022; 2022: 4069787.
[31] Becerra MF, Atluri VS, Bhattu AS, Punnen S. Serum and urine biomarkers for detecting clinically significant prostate cancer. Urologic Oncology: Seminars and Original Investigations. 2021; 39: 686–690.

How to cite this article: Xiaoling Chen, Yanzhi Han. Analysis of risk factors for benign prostate enlargement with prostate calcification. Journal of Men's Health. 2024; 20(1): 107-113. doi: 10.22514/jomh.2024.014.