LOWER URINARY TRACT SYMPTOMS, BENIGN PROSTATIC HYPERPLASIA, AND METABOLIC SYNDROME

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Abstract

***Introduction and Objective**: The aim of this study was to evaluate the relationship between lower urinary tract symptoms (LUTS), benign prostatic hyperplasia, and metabolic syndrome. **Methods**: 381 patients, over 45 years, with metabolic syndrome were divided in 2 groups: group A – 310 patients with BHP (MetS+BPH) and, group B – 71 patients without BPH (MetS-BPH). Fasting concentrations of plasma glucose, HbA1c, serum lipides (total cholesterol, triglycerides, high-density lipoprotein HDL-C), insulin, and prostate-specific antigen (PSA) were measured under standardised conditions at baseline, 6 months and one year. IR (insulin resistance) was determinated using homeostasis model assessment (HOMA-IR). The presence of LUTS was assessed using the International Prostate Symptoms Score. The prostate gland volume was measured using transrectal ultrasound (prostate volume over 30 cm3 was considered prostatic hyperplasia). **Results**: Prevalence of severe LUTS was 14.8% (n=46) in BPH patients and 4.2% (n=3) patients without prostatic diseases (0.032). Prevalence of moderate LUTS was 74.2% (n=230) in BPH patients and 78.9% (n=56) patients without prostatic diseases (0.032). IPSS score correlated positively with age, BMI, triglycerides, HDL-cholestrol, and HbA1c. **Conclusion**: LUTS are frequent in metabolic syndrome patients with or without benign prostatic hyperplasia. In patients with all five components of MetS there was an increased prevalence of LUTS. Resulting from this study it is our reccomendation that patients with MetS need follow up/seek advice from an urological specialist as well.*

***key words**: lower urinary tract symptoms (LUTS), benign prostatic hyperplasia, metabolic syndrome, diabetes.*

Introduction

Different studies have shown that the prevalence of LUTS increases with age and some authors have suggested that approximately 43% of all males over the age of 60 years suffer alterations in their quality of life as a result of the presence of urinary symptoms [1, 2, 3].

The prostatic hyperplasia is the most common disorder of the prostate in elderly males, its incidence increases with age, and its

prevalence is about 90% in men in their 80s [4, 5].

Age is the only unchangeable and established factor known so far, although androgens and their metabolites strongly influence the growth and development of the prostate gland [6, 7].

The metabolic syndrome is a cluster of cardiovascular risk factors: diabetes and raised fasting plasma glucose, abdominal obesity, high cholesterol levels and high blood pressure. The prevalence of the MetS in the general population is estimated to be around 20-25%, increases with age and is higher in men than women [8, 9]. Reports about MetS in Romania showed a higher prevalence of the metabolic syndrome and varies in men between 28.8%-43.1% [10, 11].

Material and methods

The study population was recruited from patients screened for prostate cancer in the research project "Optimization of the precocious diagnosis of prostate adenocarcinoma at patients with metabolic syndrome by correlating the genetic factors, anatomic-pathological and biochemical" acronim ADENODIAG. The study was held in three medical centers in Bucharest and from the 414 patients included in this study, 33 patients (7.97%) were diagnosed with cancer. As the prostate cancer patients did not completed all immunological and genetical tests, they were not included in this analysis. MetS was diagnosed according to IDF criteria (central obesity - waist circumference over 94 cm in men or BMI >30kg/m^2 plus any tow of the following factors: 1. triglycerides (TG) ≥1.695mmol/l (150mg/dl) or treatment; 2. lower high density lipoprotein-cholesterol (HDL-C) <40mg/dl or treatment; 3. blood pressure ≥130/85mmHg or medication; 4. fasting blood glucose ≥5.6mmol/l (100mg/dl) or medication for diabetes).

Biochemical tests including: fasting plasma glucose, HbA1c, total cholesterol, triglycerides, high-density lipoprotein (HDL-C), fasting plasma insulin, adiponectin, leptin, TNF alpha, IL-6 and prostate-specific antigen (PSA) were performed.

IR (insulin resistance) was determinated using homeostasis model assessment of insulin resistance (HOMA-IR) (fasting insulin level (mUI/l)x fasting glucose level (mg/dl)/405; a HOMA-IR index value of more than 2.0 was considered as the criterion of insulin resistance.

Body weight, waist circumference, hip circumference, blood pressure were determined.

Body mass index (BMI) was calculated (BMI= weight (kg)/ height (m^2)).

LUTS and symptom specific quality of life were assessed using the International Prostate Symptom Score (IPSS) and the IPSS quality of life scores. Test interpretation is the following: 0-7 mildly symptomatic; 8-19 moderately symptomatic; 20-35 severely symptomatic.

The volume of the prostate was measured using transrectal ultrasound (prostate volume over 30 cm^3 was considered prostatic hyperplasia).

Statistical analysis

Data are presented as mean ± SE or percentages, according to the variables. Correlations among continuous variables (age, anthropometry and blood tests) were determined using the Pearson correlation test. Statistical comparisons of continuous data were performed using paired *t*-test or one-way analysis of variance (ANOVA). The level of statistical significance was defined as $P < 0.05$

and all statistical tests were two-sided. Statistical analyses were performed using a commercially available analysis program, SPSS version 18.0 (SPSS, Chicago, IL, USA).

Caracteristics of patients

Of the 381 patients, 12.8% (n=49) had severe LUTS and 74.9% (n=286) moderate LUTS. Patients with severe LUTS were older, with higher BMI, with hypercholestrolemia, hyperinsulinemia, with greater HOMA-IR and bigger prostate volume ($p<0.05$) (Table 1). Educational level, employment, residential setting corelated with IPSS score (all $p<0.0001$)(Table 2). BMI, SBP, DBP, TG, HDL-c, HbA1c, fasting plasma glucose, PSA level, testosterone level do not differ between groups ($p>0.5$).

Table 1. Characteristics of patients

	IPSS mild		IPSS moderate		ISPP severe		p value
	Means (median)	SE (range)	Means (median)	SE (range)	Means (median)	SE (range)	
Age (years)	57.37	7.72	58.02	8.12	59.04	5.76	**0.04**
Weight (kg)	94.93	8.92	94.27	10.62	95.04	11.41	**0.02**
BMI (kg/m^2)	31.77	2.36	31.01	3.00	31.41	3.22	0.23
WC (cm)	106.74	7.83	108.21	8.20	109.20	8.46	0.34
HC (cm)	102.02	7.49	102.31	7.30	104.59	7.16	0.11
SBP (mmHg)	135.43	9.48	139.69	14.60	138.37	13.82	0.15
DBP (mmHg)	80.33	5.21	82.42	9.66	80.10	9.44	0.13
TG (mg/dl)	210.39	95.40	186.05	79.84	195.32	82.38	0.16
HDL-C (mg/dl)	40.36	10.05	40.08	9.04	38.94	8.66	0.69
Chol (mg/dl)	223.94	63.25	204.48	50.05	211.41	49.27	**0.05**
FPG (mg/dl)	122.83	52.31	127.02	50.80	134.16	61.58	0.05
FPI (uU/ml)	14.45	1.2-148.8	11.62	1.3-122.3	12.5	2.2-31.2	**0.009**
HOMA-IR	4.07	0.29-43.35	3.33	0.46-65.5	4.03	0.73-19.77	**0.01**
HbA1c (%)	7.22	1.75	7.38	1.78	7.39	1.42	0.80
PSA (ng/dl)	0.74	0.1-3.3	0.85	0.1-12.8	0.86	0.3-5	0.59
%FPSA	0.34	0.12	0.36	0.13	0.39	0.13	0.19
Testosterone (ng/ml)	3.85	1.26	3.63	1.31	3.58	1.29	0.52
PV (cm^3)	36.59	9.92	40.08	11.39	43.27	10.01	**0.01**

Table 2. Frecvency (%) of IPSS score in study population

		IPSS mild	IPSS moderate	ISPP severe	p
Educational level	Higher	0(0%)	126 (44.1%)	37 (75.5%)	p=0.0001
	Low	0(0%)	31 (10.8%)	1 (2%)	
	Medium	46 (100%)	129 (45.1%)	11 (22.4%)	
Smoker	No	37(80.4%)	172(60.1%)	49(100%)	p=0.0001
	Yes	9(19.6%)	114(39.9%)	0(0%)	
Home	Rural	5(10.9%)	125(43.7)	12(24.5)	p=0.0001
	Urban	41(89.1)	161(56.3)	37(75.5)	
Groups age	45-50	5(10.9%)	22(7.7%)	1(2%)	p=0.450
	50-59	27(58.7%)	155(54.2%)	33(67.3%)	
	60-69	11(23.9%)	74(25.9%)	12(24.5%)	
	70-79	2(4.3%)	32(11.2%)	3(6.1%)	
	80+	1(2.2%)	3(1%)	0(0%)	
Groups BMI	O1	34(73.9%)	140(49%)	21(42.9%)	p=0.019
	O2	2(4.3%)	18(6.3%)	7(14.3%)	
	O3	1(2.2%)	7(2.4%)	1(2%)	
	OW	9(19.6%)	121(42.3%)	20(40.8%)	
BMI 30 kg/m^2	No	9(19.6%)	121(42.3%)	20(40.8%)	p=0.013
	Yes	37(80.4%)	165(57.7%)	29(59.2%)	
TG-Met S	No	10(21.7%)	99(34.6%)	13(26.5%)	p=0.15
	Yes	36(78.3%)	187(65.4%)	36(73.5%)	
HDL-c Met S	No	18(39.1%)	126(44.1%)	18(36.7%)	p=0.559
	Yes	28(60.9%)	160(55.9%)	31(63.3%)	
BP-Met S	No	24(52.2%)	99(34.6%)	19(38.8%)	p=0.0071
	Yes	22(47.8%)	187(65.4%)	30(61.2%)	
DM-Met S	No	13(28.3%)	61(21.3%)	14(28.6%)	p=0.364
	Yes	33(71.7%)	225(78.7%)	35(71.4%)	
HOMA IR	No	10(21.7%)	76(26.6%)	11(22.4%)	p=0.685
	Yes	36(78.3%)	210(73.4%)	38(77.6%)	
Testosteron group (□3 ng/ml)	No	11(23.9%)	100(35%)	17(17%)	p=0.333
	Yes	35(76.1%)	186(65%)	32(65.3)	
Diagnostic	BPH	34(73.9%)	230(80.4%)	46(3.9%)	p=0.032
	Normal	12(26.1%)	56(19.6%)	3(6.1%)	

Abbreviations: OW, overweight; O1, obesity class 1; O2, obesity class 2, O3, obesity class 3.

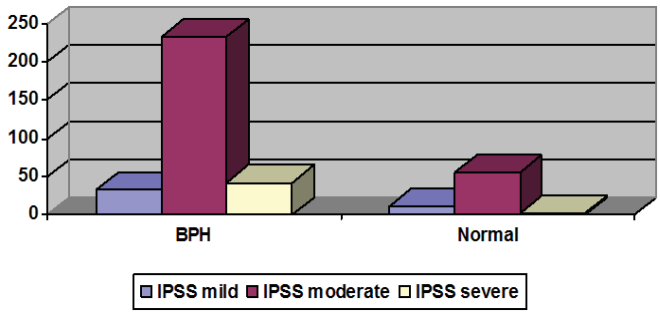

Figure 1. Prevalence of LUTS in metabolic syndrome patients (p=0.032)

Metabolic syndrome and and its components

Components of metabolic syndrome witch correlated with IPSS classification were body mass index (p=0.019), obesity (BMI over 30kg/m^2) (p=0.013) and blood pressure (p=0.0071) (Table 2).

If we analyzed IPSS score as over or under 8 we found a statistically significant correlation with obesity degree (p=0.0001), BMI over 30 kg/m^2 (p=0.0001) and blood pressure (p=0.034).

LUTS and prostatic diseases

Prevalence of severe LUTS was 14.8% (n=46) in BPH patients and 4.2% (n=3) in patients without prostatic diseases (0.032) (Figure 1) and with moderate LUTS was 74.2% (n=230) in BPH patients, respectively 78.9% (n=56) in patients without prostatic diseases (0.032).

In univariate analysis IPSS was correlated positive with age, BMI, weight, HbA1c, tryglicerides, fasting plasma glucose, PSA level, prostatic volume (all p<0.05) but not with testosterone, HOMA-IR, fasting plasma insulin (Table 3)(Figure 2, 3).

Table 3. Univariate analysis of correlation between IPSS and clinical and biological variables in BPH group

Variables	IPSS	
	r	p
Age (years)	0.213	0.0001
BMI (kg/m^2)	0.1	0.05
Weight (kg)	0.12	0.01
HbA1c %	0.265	0.003
Cholesterol (mg/dl)	-0.03	0.001
Triglycerides (mg/dl)	0.27	0.002
HDL-colestrol (mg/dl)	0.12	0.03
Fasting plasma glucose (mg/dl)	0.11	0.001
Fasting plasma insulin (mU/ml)	0.07	0.12
HOMA-IR	0.003	0.059
PSA (ng/ml)	0.34	0.026
Free PSA (ng/ml)	0.139	0.001
Prostatic volume (cm^3)	0.158	0.001

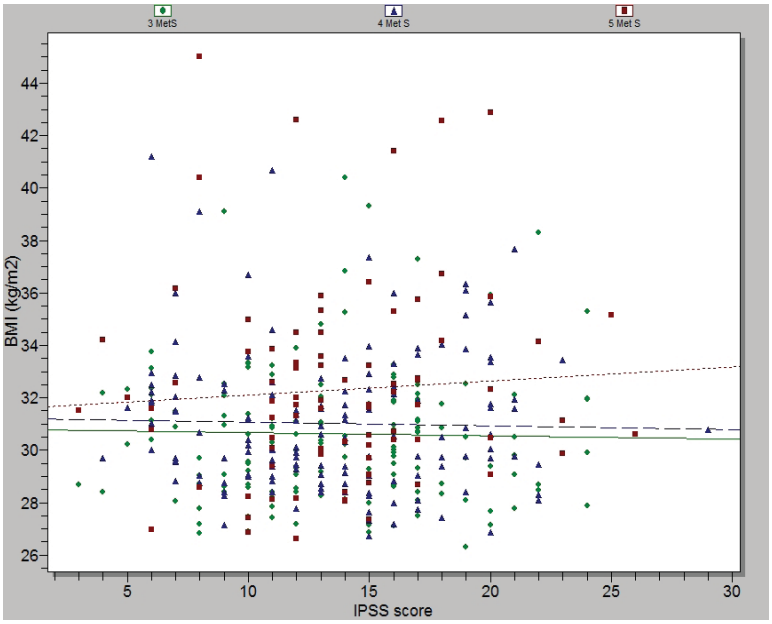

Figure 2. Correlation between IPSS score and BMI

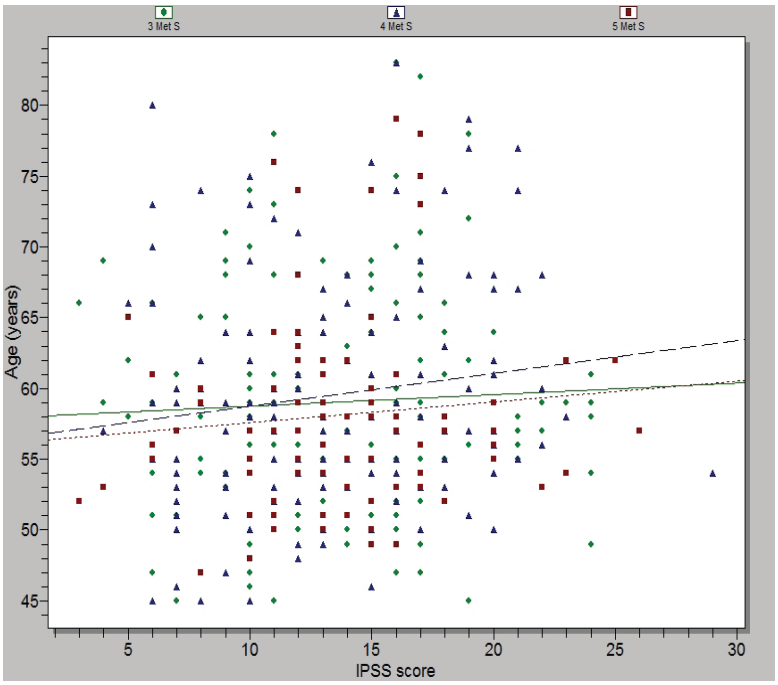

Figure 3. Correlation between IPSS score and age

We analysed whether clinical parameters, including age, anthropometry parameters and metabolic profiles, are discriminative for severe LUTS (IPSS=20) using the area under the receiver operating characteristic curve (AUROCC).

The area under the curve is a suitable parameter to summarize the overall discriminative or diagnostic value of a model and can range from 0.5 (flipping a coin, a useless model) to 1.0 (perfect discrimination). The larger the AUROCC approached 100% (i.e. the more the receiver operating

characteristic [ROC] curve approached the upper-left corner), the greater the predictive power.

Figure 4 presents the area under the receiver operating characteristic curve for the discriminative value of clinical parameters on severe LUTS. The AUROCC of prostatic volume was 61.5% (95% CI, 53.8%–69.2%; *P* = 0.009) for severe LUTS.

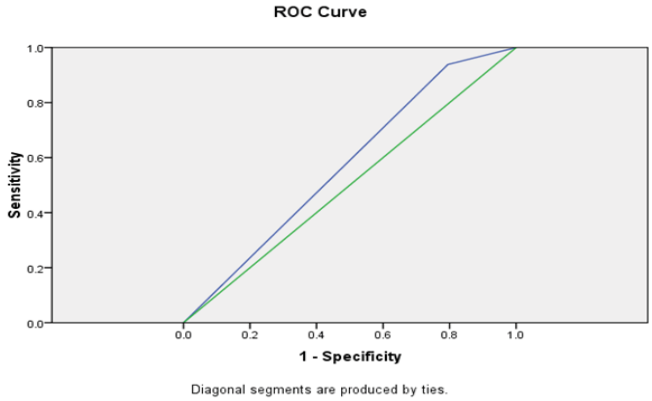

Figure 4. ROC curve for prostatic volume

Table 4. Area under the curve for various biochemical parameters

Test results	AUC	p value	95% Confidence Interval	
			Lower bound	Upper bound
PV (cm^3)	0.615	0.009	0.538	0.692
HC (cm)	0.575	0.091	0.492	0.657
FPG (mg/dl)	0.54	0.365	0.459	0.621
WC (cm)	0.539	0.383	0.455	0.622
Chol (mg/dl)	0.532	0.473	0.443	0.62
PSA (ng/dl)	0.532	0.473	0.451	0.613
TG (mg/dl)	0.521	0.634	0.431	0.611
BMI (kg/m^2)	0.52	0.644	0.431	0.61
FPI (uU/ml)	0.511	0.802	0.433	0.59
HbA1c (%)	0.511	0.804	0.422	0.6
SBP (mmHg)	0.479	0.635	0.392	0.566
Testosterone (ng/ml)	0.477	0.597	0.39	0.563
HDL-C (mg/dl)	0.471	0.516	0.388	0.554
DBP (mmHg)	0.447	0.23	0.361	0.533

Abbreviations: PV, prostatic volume; HC, hip circumference; FPI, fasting plasma glucose; WC, waist circumference; Chol, cholesterol; PSA, prostatic specific antigen; TG, triglycerides; BMI, body mass index; FPI, fasting insulin; HbA1c, glycated hemoglobin; SBP, systolic blood pressure; HDL-C, high-density lipoprotein cholesterol; DBP, diastolic blood pressure.

However, the area under the receiver operating characteristics curve for hip circumference, waist circumference, fasting plasma glucose, cholesterol, PSA, triglycerides, BMI, HbA1c, SBP, testosterone, HDL-cholesterol and DBP was not significant (data shown in table 4).

Hypertension (≥135/90 mmHg) and obesity (BMI over 30 kg/m^2) were strongly related to the presence of severe LUTS: OR was 2.006 for hypertension and 1.119 for obesity (Figure 5).

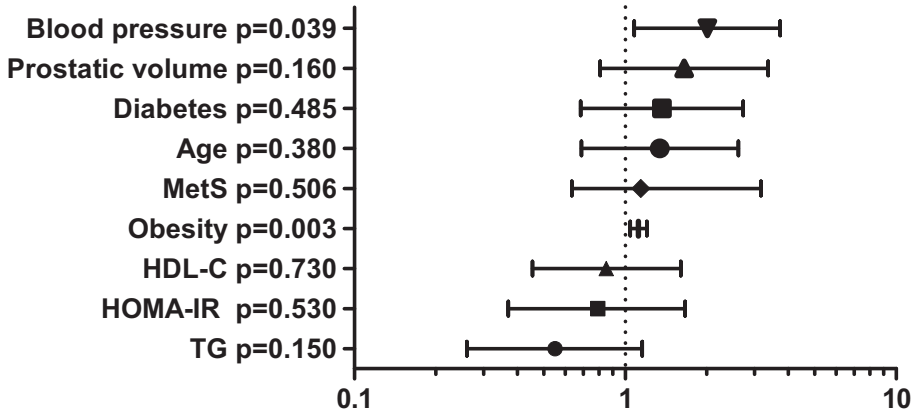

Figure 5. Independent risk factors for LUTS

Discussions

Of the 381 patients, 12.8% (n=49) had severe LUTS and 74.9% (n=286) moderate LUTS. Prevalence of severe LUTS is similar to that published by other studies in France [12] and Austria [13]; mild LUTS in our study was more frequent than in other studies conducted in Spain [14], Canada [15], Japan [16] and the United States [17] – with estimated prevalences of about 25-30%.

Higher prevalence of LUTS is explained by:

1. inclusion only of patients with metabolic syndrome;

2. patients included were recruited from hospital (urology clinics and diabetes clinics);

3. a higher percentage of patients with diabetes and metabolic imbalance (HbA1c) .

The observed increase in the prevalence of LUTS with the increase of age agrees with the findings published previously [18].

Contrary to other published studies some metabolic parameters, such as triglyceride and HDL-cholesterol levels, were not associated with LUTS. To our knowledge, this finding has not been described previously.

In our study we found that components of MetS such obesity and blood pressure are associated with LUTS and in this patients prevalence of LUTS increased with 1.19-2.006-fold.

Dahle et al. [19] have questioned the usefulness of BMI for assessing obesity and have recommended the measurement of abdominal obesity based on the waist-hip index, while Lee et al. [20] observed a bimodal association between abdominal obesity and LUTS, whereby moderate and severe LUTS were more frequent in obese and underweight individuals, compared with males with average body weight. In conclusion, obesity could be associated to prostate gland growth, though further evidence is needed in order to link this to LUTS.

We find a beneficial effect of glycemic control on prevalence of moderate to severe LUTS. Whereas there is general agreement that diabetes results in LUTS [21, 22] the

epidemiology of diabetes and LUTS is not clear. Much of the epidemiology in this area has been limited because prior studies that use markers of LUTS (e.g., trans-urethral resection of the prostate or TURP) [23] have included few men with diabetes, and/or included solely or primarily men with type 2 diabetes. Nonetheless, the Massachusetts Male Aging Study [24], and others have consistently reported diabetes or glucose levels to be associated with an increased risk of LUTS with or without benign prostatic hyperplasia. Progression of LUTS has been reported to be greater in men with diabetes than in those without diabetes, in the absence of a difference in prostate volume growth between the two groups [25], suggesting a progressive impact on the bladder. However, these studies have not examined how glycemic control affects LUTS.

The limitation of the study were: 1. we included only patients with metabolic syndrome and some conclusions can not be applied in another population; 2. we did not analysed other sociodemographic factors, such as marital status that might affect urinary symptoms.

Conclusions

LUTS are frequent in metabolic syndrome patients with or without benign prostatic hyperplasia. In patients with all five components of MetS prevalence of LUTS was much higher. Age, a low educational level, arterial hypertension, diabetes were independently associated to the presence of severe LUTS. Given the results of the study we recommend that patients with metabolic syndrome should have an urological follow-up.

Acknowledgements: No potential conflict of interest relevant to this article was reported.

This study was supported by the Romanian National Authority for Scientific research as a part of the PNCDI 2 program ADENODIAG 41-085/2007.

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