# Table 2. Echocardiographic findings

	Normal group n=15	Mild-moderate MS n=20	Severe MS n=15
Mitral valve area(mm <sup>2</sup> )	> 40	14 +/- 1.7	10 +/- 0.8
Mean mitral gradient (mmhg)		6.8 +/- 3	10.8 +/- 2.1
IVS (mm)	8.4 +/- 0.7**	9.6 +/- 0.9	9.4 +/- 1.1
LVPW (mm)	8.6 +/- 0.6	9.6 +/- 0.94	9.0 +/- 1.2
LAD(mm)	33 +/- 2*	44 +/- 3	46 +/- 5
LVDD(mm)	28 +/- 3	29 +/- 4	28 +/- 3
LVSD(mm)	44 +/-2	46 +/- 3	43 +/- 10
FS(%)	35 +/- 3	35 +/- 4	36 +/- 4
EF(%)	65 +/- 3	65 +/- 6	65 +/- 5
SPAP(mmhg)	23 +/- 2 🖨 🖨	36 +/-12♦	52 +/- 13

\*p<0.001compared to mild-moderate and severe MS;\*\*p< 0.05 compared to mild-moderate and severe  $\Phi p < 0.01$  compared to mild-moderate MS;  $\Phi p < 0.001$  compared to severe MS;  $\Phi p < 0.001$  compared to severe MS; MS: mitral stenosis; IVS:interventricular septum; LVPW: left ventricular posterior wall; LAD: left atrial diameter, LVDD: left ventricular diastolic diameter; LVSD: left ventricular systolic diameter; FS: fractional shortening; EF: ejection fraction; SPAP: systolic pulmonary artery pressure.

### Table 3. Pulsed wave tissue Doppler recordings

	Normal group n=15	Mild-moderate MS n=20	Severe MS n=15			
Long axis						
SW1 (cm/s)	10.7 +/- 2.3*	7.9 +/- 1.3	6.2 +/- 1.4			
SW2 (cm/s)	5.4 +/- 0.7	6.0 +/- 1.2**	4.7 +/- 1.2			
Q-SW1 (ms)	145 +/- 32♦	199 +/- 43	180 +/- 46			
Q-SW2 (ms)	259 +/- 37	258 +/- 43	258 +/- 44			
Short axis						
SW1 (cm/s)	7.5 +/- 1.2	7.1 +/- 1.2	7.0 +/- 1.4			
SW2 (cm/s)	4.8 +/- 1.0	5.5 +/- 1.1	5.5 +/- 1.3			
Q-SW1 (ms)	182 +/- 28	178 +/- 38	168 +/- 28			
Q-SW2 (ms)	298 +/- 41*	245 +/- 37	234 +/- 26			
*p < 0.001 compared to mild-moderate and severe MS; **p < 0.01 compared to severe MS;						

 $\phi = 0.001$  compared to initid-indefine and severe ins,  $m_P < 0.01$  compared to severe ins,  $\phi = 0.001$  compared to mild-moderate MS MS: mitral stenosis; SW1: first systolic velocity peak, SW2: second systolic velocity peak; Q-SW1: the duration from Q wave to SW1; Q-SW2: the duration from Q wave to SW2

Table 4. The correlation between mitral valve area and pulsed wave tissue Doppler parameters

	Mitral valve area				
SW1 long axis	-,431**				
Pearson correlation	,002				
Sig. (2-tailed)					
SW2 long axis	,226				
Pearson correlation	,115				
Sig. (2-tailed)					
Q-SW1 long axis	0,483**				
Pearson correlation	,000				
Sig. (2-tailed)1					
Q-SW2 long axis	-,008				
Pearson correlation	,955				
Sig. (2-tailed)					
SW 1 short axis	-,116				
Pearson correlation	,423				
Sig. (2-tailed)2					
SW2 short axis	,228				
Pearson correlation	,111				
Sig. (2-tailed)					
Q-SW1 short axis	-,036				
Pearson correlation	,803				
Sig. (2-tailed)					
Q-SW2 short axis	-,471**				
Pearson correlation	,001				
Sig. (2-tailed)					
SW1: first systolic velocity peak, SW2: second sys	tolic velocity peak; Q-SW1: the duration from				
Q wave to SW1; Q-SW2: the duration from Q wave to SW2					

## PP-213

## Assessment of Relationship between Subclinical Atherosclerosis and Benign Prostat Hyperplasia using Epicardial Fat Thickness and Carotid Intima-Media Thickness

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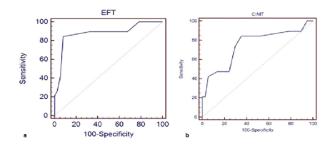
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**Objectives:** Benign prostatic hyperplasia (BPH) and lower urinary tract symptoms (LUTS) are most common disorders in elderly men. Although focused on hormones and genetic predisposition, the pathophysiology of BPH remains unclear, but it seems to be multifactorial. Currently, we have a lot of data about the relationship between the atherosclerosis and BPH, but association between subclinical atherosclerosis and this disease have not been evaluated in detail yet. In the present study we aimed to assess the relationship between subclinical atherosclerosis and BPH, using epicardial fat thickness (EFT) and carotid intima-media thickness (CIMT).

**Methods:** One hundred six men (including 56 patients with BPH and LUTS and control group which consisted of 50 healthy men with no clinical signs of BPH) were evaluated prospectively. Bladder outlet obstruction as verified by urodynamic testing, the prostate volume was calculated using abdominal ultrasonography and LUTS were evaluated using the international prostate symptom score (IPSS) and according to IPSS, patients were divided into two groups as mild - moderate (<20 point) and severe ( $\geq$ 20 point). EFT measured by transhoracic echocardiography (ECHO) and CIMT evaluated by ultrasonography (USG). The relationship between EFT, CIMT and another parameters was analysed.

**Results:** EFT, CIMT and fasting blood glucose were significantly higher in BPH group (p <0.001; p <0.001 and p=0.042, respectively) (Table 1). At multivariate logistic regression analysis EFT and CIMT were found to be independent predictors of BPH (Table 2). EFT and CIMT demonstrated an increase from IPSS <20 point to IPSS  $\geq$ 20 point (p<0.001 and p=0.003, respectively). In order to prediction of severe LUTS (IPSS  $\geq$ 20 point); receiver operating characteristic curve (ROC) analysis revealed a cut off value of 3.5mm for EFT (AUC = 0.88, 95% CI %81.8-%99.3, p<0.001) with a specificity of %91.9 and a sensitivity of %84.2 and 0.9mm for CIMT (AUC = 0.75, 95% CI %47.5-%79.8, p<0.001) with a specificity of % 64.9 and a sensitivity of %84.2 (Figure 1). At the bivariate correlation analysis; EFT and CIMT were negative correlation between Q max (r=-0.468, p<0.001, r=-0.525, p<0.001 respectively) (Table 3).

**Conclusion:** The present results support the hypothesis that subclinical atherosclerosis might have a role in the development of BPH and it can be predicted non-invasively with EFT and CIMT.



#### Table 2

	Multivariate OR(95% CI)	Multivariate p value			
EFT	2.65 (1.37-5.12)	$\mathbf{P}=0.004$			
CIMT	15.62 (1.28-200.00)	$\mathbf{P}=0.031$			
FBG 0.96 (0.92-1.00) P = 0.061					
The predictors of BPH in logistic regression analysis					

# Table 3

	Prostate volume, mL	PVR, mL	Qmax, mL/s		
EFT	r = 0.162,  p = 0.232	r = 0.257,  p = 0.056	r = -0.468, p < 0.001		
CIMT	r = 0.172,  p = 0.206	r = 0.238,  p = 0.078	r = -0.525, p < 0.001		
Results of correlation analysis					

# Table 1

	BPH Group (n = 56)	Control Group (n=50)	p value
Age, years	$\textbf{59.0} \pm \textbf{5.9}$	56.9 ± 7.2	0.103
BMI, kg/m2	$\textbf{28.4} \pm \textbf{4.4}$	$\textbf{30.9} \pm \textbf{21.5}$	0.416
WM, cm	$\textbf{96.6} \pm \textbf{9.7}$	$\textbf{95.6} \pm \textbf{11.1}$	0.603
HM, cm	$\textbf{100.4} \pm \textbf{7.3}$	98.6 ± 8.9	0.262
HT duration, years	2.9 ±4.9	$\textbf{2.1}\pm\textbf{3.2}$	0.300
Smoking duration, years	8.5 ±15.2	11.0 ±15.1	0.402
EFT, mm	$3.5\pm1.3$	$\textbf{2.5} \pm \textbf{0,7}$	<0.001
CIMT, mm	$\textbf{1.0} \pm \textbf{0.3}$	0.8± 0.2	<0.001
IVS, mm	$\textbf{11.5}\pm\textbf{3.0}$	$\textbf{12.0} \pm \textbf{1.9}$	0.321
LVEDD, mm	$\textbf{46.7} \pm \textbf{7.3}$	$\textbf{49.4} \pm \textbf{8.8}$	0.092
LVEDS, mm	$\textbf{32.5} \pm \textbf{7.8}$	$\textbf{31.9} \pm \textbf{7.1}$	0.642
PW, mm	$\textbf{11.9} \pm \textbf{6.7}$	$\textbf{12.0} \pm \textbf{1.6}$	0.937
EF, %	$\textbf{64.0} \pm \textbf{7.7}$	$\textbf{64.9} \pm \textbf{8.7}$	0.565
E, cm/sn	$\textbf{59.6} \pm \textbf{16.7}$	$\textbf{66.5} \pm \textbf{20.3}$	0.059
A, cm/sn	$\textbf{70.6} \pm \textbf{18.9}$	$\textbf{73.8} \pm \textbf{16.2}$	0.354
Em, cm/sn	10.3 ±5.3	$\textbf{9.8}\pm\textbf{3.5}$	0.559
Am, cm/sn	11.9 $\pm$ 4.2	$\textbf{11.7} \pm \textbf{3.5}$	0.800
S, cm/sn	$\textbf{9.6} \pm \textbf{2.2}$	$\textbf{9.3} \pm \textbf{2.0}$	0.448
FBG, mg/dl	$\textbf{95.7} \pm \textbf{15.9}$	101.3 $\pm$ 12.2	0.042
Creatinine, mg/dl	$\textbf{0.9}\pm\textbf{0.1}$	$\textbf{1.1} \pm \textbf{1.1}$	0.184
GFR, ml/min/1.73 m2	$\textbf{81.6} \pm \textbf{22.9}$	$\textbf{102.0} \pm \textbf{90.2}$	0.125
Total chelosterol, mg/dl	$\textbf{198.5} \pm \textbf{44.6}$	$\textbf{197.0} \pm \textbf{41.1}$	0.852
Triglycerides, mg/dl	$\textbf{137.2} \pm \textbf{73.7}$	$\textbf{155.1} \pm \textbf{70.6}$	0.203
HDL chelosterol, mg/dl	48.9 ±13.3	$\textbf{44.3} \pm \textbf{10.8}$	0.053
LDL chelosterol, mg/dl	$\textbf{121.2} \pm \textbf{40.9}$	$\textbf{130.9} \pm \textbf{37.0}$	0.203

Characteristics of patients BMI: body mass index; WMI: waist measure; HMI: Hip measure; HT: hypertension; IVS: interventricular septum; LVEDD: left ventricle end diastolic diameter; LVESD: left ventricle end sistolic diameter; PW: posterior wall; EF: ejection fraction; E: trans mitral doppler E wave; A: trans mitral doppler A wave; Em: lateral mitral annular tissue doppler E wave; Am: lateral mitral annular tissue doppler A wave; S: lateral mitral annular tissue doppler S wave FBG: fasting blood glucose; GFR: glomerular filtration rate; HDL: high density lipoprotein; LDL:low density lipoprotein.

## PP-214

The Effect of Percutaneous Mitral Valvuloplasty on Left Atrial Mechanical Functions in Mitral Stenosis: The Short and Mid Term Following

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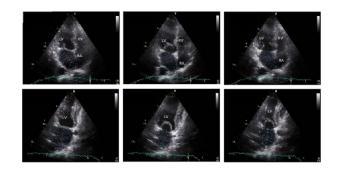
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**Objective:** Rheumatic mitral stenosis (MS) is still a common disease in developing countries with high morbidity and mortality rates. The aim of the study was to evaluate left atrial mechanical functions in MS before and after percutaneous mitral balloon valvuloplasty (PMBV) and following in short and mid term.

**Methods:** the study included 49 patients with critical mitral stenosis who have normal sinus rhythm (male/female:14 to 35; mean age: 42±11). Left atrial mechanical functions were evaluated before, after, 3 months after and 1 year after PMBV, including Left atrial passive emptying volume (LAAEV), LA active emptying volume (LAAEV), LA total emptying fraction (LAAEF), LA active emptying fraction (LAAEF), LA total emptying fraction (LAAEF) and conduit volume (figure1).

**Results:** The transthoracic echocardiography parameters of the mitral stenosis patients before, after, 3 months after and 1 year after percutaneous mitral balloon valvuloplasty were as follows: a) Mitral valve area: 1.1 cm<sup>2</sup> (0.9-1.6); 2.2 cm<sup>2</sup> (1.8-2.8) (p<0.001); 2.2 cm<sup>2</sup> (1.7-2.9) (NS); 2.1 cm<sup>2</sup> (1.8-2.7) (p<0.001); b) Left atrial maximal volume:59 ml/m<sup>2</sup> (42-76); 48 ml/m<sup>2</sup> (34-62) (p<0.001); 44 ml/m<sup>2</sup> (29-59) (p<0.001); 13 ml/m<sup>2</sup> (28-57) (p<0.001); c) Left atrial passive emptying volume (LAPEV): 13 ml/m<sup>2</sup> (9-27); 11 ml/m<sup>2</sup> (8-19) (p<0.001); 10 ml/m<sup>2</sup> (7-19) (p<0.001); 10 ml/m<sup>2</sup> (6-18) (p<0.001); 9 ml/m<sup>2</sup> (51-7) (p<0.001); 9 ml/m<sup>2</sup> (4-16) (NS) e) LA total emptying volume (LATEV): 26 ml/m<sup>2</sup> (19-50); 21 ml/m<sup>2</sup> (16-40) (p<0.001); 20 ml/m<sup>2</sup> (15-36) (p<0.001); 19 ml/m<sup>2</sup> (15-34) p<0.001); 10 cml/m<sup>2</sup> (31-42) p<0.001), respectively. However LA passive emptying fraction (LAPEF), LA active emptying fraction (LAAEF) and LA total emptying fraction (LATEF) were not altered after PMBV (tabel).

**Conclusion:** our finding showed that improvement of left atrial mechanical functions after successful treatment of MS with PMBV and this improvement continues for one year.



#### Table1

	Before PMBV	After PMBV	3st month	1 st year	p*	P1**	P2**	P3**	P4**
LAV max (ml/m <sup>2</sup> )	59 (42-76)	48 (34-62)	44 (29-59)	43 (28-57)	<0,01	<0,01	<0,01	<0,01	<0,01
LAV min (ml/m <sup>2</sup> )	33 (20-45)	27 (14-38)	24 (11-36)	23 (10-35)	<0,01	<0,01	<0,01	<0,01	<0,01
LATEF (%)	0,43 (0,32- 0.69)	0.45 (0.35- 0.66)	0.45 (0.33- 0.65)	0.45 (0.31- 0.66)	0,05	0,04	Ns	Ns	0,05
LATEV (ml/m <sup>2</sup> )	26 (19-50)	21 (16-40)	20 (15-36)	19 (15-34)	<0,01	<0,01	<0,01	<0,01	<0,01
LAV p	46 (29-61)	37 (21-49	34 (20-47)	32 (18-45)	<0,01	<0,01	<0,01	<0,01	<0,01
LAPEV (ml/m <sup>2</sup> )	13 (9-27)	11 (8-19)	10 (7-19)	10 (6-18)	<0,01	<0,01	Ns	0,03	<0,01
LAPEF (%)	0.21 (0.15- 0.37)	0.22 (0.15- 0.35)	0.23 (0.14- 0.38)	0.24 (0.14- 0.41)	Ns	Ns	Ns	Ns	Ns
cv	30 (22-44)	33 (26-46)	34 (30-42)	36 (31-42)	<0,01	<0,01	<0,01	<0,01	<0,01
LAAEV (ml/m <sup>2</sup> )	12 (8-23)	10 (6-21)	9 (5-17)	9 (4-16)	<0,01	<0,01	<0,01	Ns	<0,01
LAAEF (%)	0.27 (0.20- 0.51)	0.28 (0.20- 0.51)	0.28 (0.20- 0.51)	0.29 (0.16- 0.47)	Ns	Ns	Ns	Ns	Ns
Comparis	Comparison of before and after PMBV LA mechanic functional parameters measured by								

Transthoracic Echocardiography

#### **PP-215**

# Relation between Glycosylated Hemoglobin (Hemoglobin A1c) and Aortic Stiffness in Type 2 Diabetic Patients

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**Background:** Type 2 diabetes mellitus (DM) is a major risk factor for cardiovascular diseases and responsible for increased cardiovascular mortality. Chronic hyperglycemia is related with accelerated atherosclerosis. In this study, we tried to demonstrate the relation between glycosylated hemoglobin (HbA1c) level which is a marker of long standing hyperglycemia and aortic stiffness which is a marker of cardiovascular disease.

**Method and Results:** 100 patients with newly diagnosis type 2 diabetes mellitus were included this study. The patients divided into 3 groups according to HbA1c level (Group 1 HbA1c  $\leq 6$ , group 2 HbA1c 6-7 and group 3 HbA1c >7). Statistically significant difference was found between groups according to blood glucose level, DM duration and oral antidiabetic and insulin therapy (Table 1). Aortic systolic-diastolic diameter, aortic strain and aortic distensibility were similar between group 1 and group 2 whereas in group 3, aortic systolic-diastolic diameter, aortic strain and aortic distensibility were significantly lower than group 1 and group 2 (p<0.001) (Table 2). Significant correlation was found between aortic distensibility and HbA1c level (r=0.283; p:0.004) (Figure 1). Moreover, aortic distensibility was