

**Background:** Several studies have demonstrated the presence of an association between male pattern baldness (androgenic alopecia) and atherosclerotic vascular disease. Hypertension is one of the strongest risk factors of atherosclerosis. Association between androgenic alopecia and hypertension has been established. However, there is no data on arterial stiffness measures of asymptomatic young adults with androgenic alopecia.

**Methods:** A hundred and seventy four asymptomatic male medical personnel aged between 18-45 years were consecutively enrolled to the study. Data collected included age, history of hypertension, diabetes mellitus, smoking, hypercholesterolemia, familial history of coronary artery disease, systolic and diastolic blood pressures, body-mass index. Subjects were considered to have androgenic alopecia if they have grade 3 vertex or more alopecia according to Hamilton-Norwood scale. Subjects were dichotomized according to presence of androgenic alopecia. Arterial stiffness was assessed by cardio ankle vascular index (CAVI) and defined as abnormal if CAVI is measured >8.

**Results:** Clinical and laboratory characteristics between subjects with and without androgenic alopecia were summarized in Table 1. Subjects with androgenic alopecia had higher mean CAVI than patients without androgenic alopecia ( $7.62 \pm 0.92$  vs.  $7.23 \pm 0.88$ ,  $p=0.001$ ). Carotid intima media thickness (CIMT) and ankle brachial index (ABI) were not significantly different in patients with and without androgenic alopecia. Binary logistic regression analysis was performed to find the independent factors associated with abnormal CAVI. The covariates included age, body mass index, smoking status, family history of coronary artery disease, presence of hypertension, hypercholesterolemia, and alopecia. In this model, presence of androgenic alopecia (OR, 6.7; 95% CI, 2.2-20.0,  $p=0.001$ ), hypertension (OR, 8.4; 95% CI, 1.9-37.4,  $p=0.006$ ), hypercholesterolemia (OR, 7.0; 95% CI, 1.4-35.0,  $p=0.02$ ) and age (OR, 1.1; 95% CI, 1.0-1.2,  $p=0.001$ ) were found to be independently associated with abnormal CAVI (Table 2).

**Conclusion:** Androgenic alopecia is independently associated with arterial stiffness in asymptomatic young adults.

**Table 1. Comparison of clinical and laboratory characteristics between subjects with and without androgenic alopecia.**

	Alopecia (+) n=100	Alopecia (-) n=74	p
CAVI	$7.62 \pm 0.92$	$7.23 \pm 0.88$	0.006
ABI	$1.10 \pm 0.08$	$1.12 \pm 0.09$	NS
CIMT*	0.4 (0.4-0.5)	0.4 (0.4-0.5)	NS
Age*, years	34 (29-39)	32 (29-38)	NS
Diabetes mellitus, n (%)	0 (0 %)	0 (0 %)	NS
Hypertension, n (%)	8 (8 %)	4 (5.4 %)	NS
Hypercholesterolemia, n (%)	2 (2 %)	8 (10.8 %)	NS
Smoking, n (%)	48 (48 %)	30 (40.5 %)	NS
BMI*, (kg/m <sup>2</sup> )	27 (25-29)	26 (25-31)	NS
Family history of CAD, n (%)	30 (30 %)	10 (13.5 %)	0.01
Systolic blood pressure, mmHg	$135 \pm 11$	$133 \pm 15$	NS
Diastolic blood pressure, mmHg	$84 \pm 10$	$81 \pm 8$	NS

ABI, ankle brachial index; BMI, body mass index; CAD, coronary artery disease; CAVI, cardio ankle vascular index; CIMT, carotid intima media thickness, NS, non-significant. Data are expressed as no. (%) or mean  $\pm$  standard deviation. \*Data are presented as median and interquartile ranges.

**Table 2. Binary logistic regression analysis showing independent factors associated with abnormal CAVI (CAVI  $\geq$  8)**

Variables	CAVI < 8 n=131	CAVI $\geq$ 8 n=43	OR (95 % CI)	P
Alopecia, n (%)	66 (50.4 %)	34 (79.1 %)	6.7 (2.2-20.0)	0.001
Hypertension, n (%)	3 (2.3 %)	9 (20.9 %)	8.4 (1.9-37.4)	0.006
Age, years	$32.7 \pm 5.8$	$37.7 \pm 4.8$	1.1 (1.0-1.2)	0.001
Hypercholesterolemia, n (%)	5 (3.8 %)	5 (11.6 %)	7.0 (1.4-35.0)	0.02

The covariates included age, body mass index, smoking status, family history of coronary artery disease, presence of hypertension, hypercholesterolemia, and alopecia.

## PP-022

### Corelation Between the 24-Hour Urine Aldosterone Levels and Atrial Electromechanical Conduction Time

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**Purpose:** Number of studies have been performed on the hypertensive patients, related to the level of circulating aldosterone which leads to the cardiac fibrosis, and which is associated to the atrial fibrillation. The aim of our research is to see if there is a relation between atrial electromechanical conduction time (As an indicator of the amount of the circulating aldosterone) and urinary amount of aldosterone observed in 24 hours in normotensive people.

**Method:** The study included both an office and ambulatory blood pressure (BP) which were monitored in normotensive people (office blood pressure (BP) <140/90mmHg, and the average 24-hour BP <130/80 mmHg) in individuals who did not use any drugs and who were less than 60 years of age. A total of 85 people (54 women, with an average age of  $46.2 \pm 9.1$  years) were included. Individuals with any chronic disease, people who are smoking or who have used the drugs continuously were excluded from the study. Electromechanical atrial conduction time and both intra-atrial and interatrial conduction delays were measured by using tissue Doppler imaging. A time from the beginning of the P wave, which was noticed by using tissue Doppler, until the time from the beginning of the late diastolic wave (A wave) which is obtained from the septal, the right ventricle and tricuspid annulus was measured. Electromechanical delay between the atrial represents the difference between lateral PA and the tricuspid PA. The differences between septal PA and tricuspid PA, and lateral PA and septal PA are defined as electromechanical delay of the right atrial and electromechanical delay of the left atrial, respectively. By continuing with the normal nutrition, urin was collected in the 24 hours as sample to measure the level of aldosterone, and, under the adequate conditions, at the end of the study, the level of aldosterone was measured from all the samples (with the diametra aldosteron kit).

**Results:** Demographic data are shown in Table 1. The researches performed on normotensive people showed that there is a correlation between the 24-hour amount of aldosterone and atrial conduction time.

**Conclusion:** The results of our research related to the amount of circulating aldosterone (which has been identified by measuring 24-hour urinary aldosterone), showed that it can cause the atrial arrhythmias such as atrial fibrillation.

**Table 1. demographic data**

	n=85
Age	$46.2 \pm 9.1$
Female, n (%)	54 (63.5)
BMI (kg/m <sup>2</sup> )	$29.2 \pm 4.5$
LVEF (%)	$66.1 \pm 4.4$
LA diametre (mm)	$32.8 \pm 3.1$
Office SBP(mmHg)	$125.9 \pm 8.2$
125.9 $\pm$ 8.2	$74.9 \pm 9.3$
24h SBP (mmHg)	$120.1 \pm 7.3$
24h DBP (mmHg)	$72.4 \pm 4.4$
Daytime SKB (mmHg)	$124.8 \pm 9.1$
Daytime DKB (mmHg)	$75.5 \pm 4.8$
Night time SKB (mmHg)	$112.5 \pm 9.2$
Night time DKB (mmHg)	$66.4 \pm 5.5$
Urinary aldosterone (mg/day)	9.1 (6.5, 13.7)

Body mass index (BMI) Left ventricular ejection fraction (LVEF) Left atrium (LA) systolic blood pressure (SBP) Dystolic blood pressure (DBP)

**Table 2. Analysis of the correlation between atrial conduction time and 24-hour urinary aldosterone**

	tricuspid PA (ms)	Septal PA (ms)	Lateral PA (ms)	Septal PA-tricuspid PA (right ventricular conduction delay)	Lateral PA-Septal PA (left ventricular conduction delay)	Lateral PA-tricuspid PA (conduction delay between the atria)
Urinary aldosterone (mgr/day)	0.2 (0.069)	0.21 (0.050)	0.31 (0.004)	0.18 (0.099)	0.25 (0.019)	0.32 (0.003)

**PP-023**

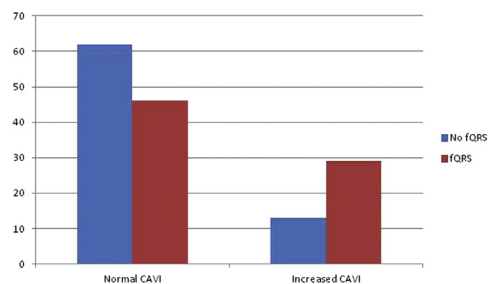
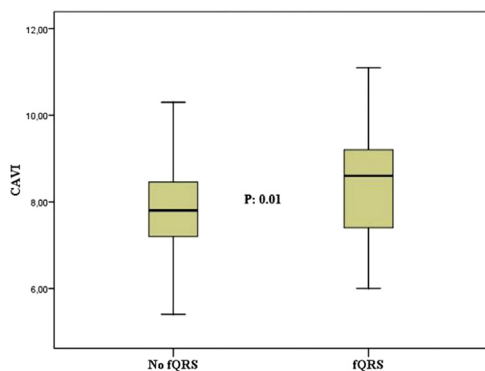
**Fragmented QRS and Cardio Ankle Vascular Index in Asymptomatic Hypertensive Patients**

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**Objective:** Patients with hypertension are predisposed to atherosclerosis of large vessels and are at increased risk of target organ damage and related clinical sequelae. Cardio-ankle vascular index (CAVI) is a novel parameter of arterial stiffness and surrogate marker of subclinical atherosclerosis. The aim of present study was to investigate the relation between fragmented QRS (fQRS) and CAVI in asymptomatic hypertensive subjects.

**Method and Results:** Seventy five asymptomatic hypertensive patients with fQRS and 75 control subjects without fQRS were enrolled. Patients with fQRS had higher CAVI values compared to those without fQRS ( $8.6 \pm 1.4$  versus  $7.9 \pm 1.3$ ,  $p:0.01$ ). In univariate analyse, there was significant association between increased CAVI and age ( $p<0.001$ ) and fQRS ( $p:0.003$ ). Multivariate binary logistic regression analyse demonstrated fQRS: [95% confidence interval (CI): 0.122 – 0.675,  $p:0.004$ ] and age [95% (CI): 1.022 – 1.105,  $p:0.002$ ] as the independent determinants of increased CAVI.

**Conclusion:** Presence of fQRS on ECG may provide important predictive information of arterial stiffness in asymptomatic hypertensive subjects.



**PP-024**

**Mean Platelet Volume and Abnormal Left Ventricle Geometric Patterns in Patients with Untreated Essential Hypertension**

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**Objective:** Abnormal left ventricle (LV) geometric patterns are associated with a greater risk of hypertensive vascular complications. The mean platelet volume (MPV) reflects the platelet activity, and is associated with overall vascular mortality. Although association between MPV and LV hypertrophy in hypertensive patients has been investigated, relation between abnormal LV geometric patterns and MPV was not studied so far. The aim of the study is to investigate the relationship between MPV and abnormal LV geometric patterns in hypertensive patients.

**Methods:** Measurements were obtained from 223 patients with untreated essential hypertension (Mean age =  $52.1 \pm 5.2$  years). Four different geometric patterns (NG; normal geometry, CR; concentric remodelling, EH; eccentric hypertrophy, CH; concentric hypertrophy) were determined according to LV mass index (LVMI) and relative wall thickness (RWth). MPV, high sensitive C-reactive protein (hs-CRP) and other biochemical markers were measured in all patients.

**Results:** The highest MPV values were determined in CH group compared with NG, CR and EH groups ( $p<0.05$ , for all). MPV values were similar among the NG, CR and EH groups ( $p>0.05$ , for all) (Table). MPV was associated with age, glucose, hs-CRP, RWth, LVMI and LV geometry in bivariate analysis ( $p<0.05$ , for all). Age ( $\beta=0.110$ ,  $p=0.033$ ), LVMI ( $\beta=0.471$ ,  $p<0.001$ ) and hsCRP ( $\beta=0.525$ ,  $p<0.001$ ) were independent predictors of high MPV in multiple linear regression analysis.

**Conclusion:** The highest MPV values were observed in CH group. This result may be associated with increased inflammation and LV hypertrophy in this geometric pattern.

**Table. Comparison of baseline, laboratory and echocardiographic characteristics**

Variables	NG group (n=50)	CR group (n=44)	EH group (n=42)	CH group (n=87)	P value
Age, years	52.5±4.2	51.0±4.6	51.4±4.6	52.7±6.1	0.236
MPV, fL	9.4±1.8	9.6±1.7	9.8±1.6	11.2±1.8	<0.001
Platelet count, x10 <sup>9</sup> /L	281.2±48.7	267.5±51.8	264.9±52.3	261.0±52.8	0.269
LVMI, g/m <sup>2</sup>	88.8±10.1	96.7±11.9	128.0±9.6	148.7±24.3	<0.001
HsCRP, mg/dl	0.80±0.31	0.83±0.33	0.88±0.36	0.95±0.34	0.075

MPV; mean platelet volume, HsCRP; high sensitive C-reactive protein, LVMI; left ventricular mass index

**PP-025**

**The Echocardiographic Evaluation of Right Ventricular Function in Patients with Non-Dipper Hypertension**

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**Objective:** Hypertension is a major risk factor for cardiovascular disease. The non-dipper form of hypertension is associated with progressive end organ damage. Diastolic dysfunction may increase the left ventricular end diastolic pressure leading to