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The Association between Testosterone Levels and Premature Coronary Artery Disease in Men

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Background: The increased incidence of CAD in men compared with premenopausal women suggests a detrimental role of male hormones on the cardiovascular system. From a large study performed at the University Hospital Center of Tirana, who has study the correlation of testosterone levels with CAD. We have analyzed correlation in the group of young men.

Aim: This study was planned to determine the relationship between serum testosterone levels and premature development of coronary artery disease in men.

Methods: 225 men under age 50 who performs coronaryography 143 men with documented coronary artery disease (mean age 44.06±4.2) constituted the study group. Control group consisted of 82 men with similar age and coronary angiography normal (mean age 42.13±4.6). Within this group we have analyzed the correlation in the youngest group: 32 men until 40 years of age (mean age 37.3±3.01) with documented CAD. Control group consisted of 29 men with similar age (mean age 36.8±2.7) and normal coronary angiography. Levels of testosterone, low-density lipoprotein, and high-density lipoprotein cholesterol, and triglyceride levels were measured, and compared between the two groups.

Results: Mean age, body mass index, the frequency of hypertension, and family history of coronary artery disease was similar between the two groups; however, diabetes mellitus, smoking, were more frequent in the (CAD) coronary artery disease group p=0.001. Low levels of HDL-c, high level of triglyceride, were more frequent in the CAD group p<0.05. The testosterone levels of the patients with coronary artery disease were (5.2±1.8 nmol/ml) significantly lower than those of controls (7.6±2.4 nmol/ml) p<0.000. This difference is visible in the younger group: The testosterone levels of the patients with CAD were (5.14±1.7 nmol/ml) significantly lower than those of controls 7.2±2.33nmol/ml p<0.000. The multivariate logistic regression analysis of all risk factors for CAD in the patients of our study, revealed that besides diabetes mellitus (p=0.002), HBP (p=0.0048), smoking (p=0.31), BMI (p=0.029), low free testosterone level (p=0.000; odds ratio=0.57; 95% confidence interval = 0.52 – 0.63) was an independent risk factor for CAD.

Conclusion: A low level of testosterone related to the development of premature coronary artery disease in men. A low level of testosterone result as an independent risk factor for CAD.

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Decreased heart rate variability in irritable bowel syndrome has no effect on carotid intima-media thickness and pulse wave velocity

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Background: Irritable bowel syndrome (IBS), a subgroup of functional somatic disorders, may be associated with autonomic dysfunction (AD). Heart rate variability (HRV), a measure of autonomic dysfunction, may predict survival. The aim of this study was to investigate the effect of IBS on HRV parameters, carotid intima-media thickness (CIMT) and carotid-femoral pulse wave velocity (cf-PWV) as surrogates of AD, subclinical atherosclerosis and arterial stiffness, respectively.

Methods: Thirty consecutive patients with IBS and 30 control participants underwent 24-hour Holter monitoring, cf-PWV assessment and CIMT measurement. The diagnosis of IBS was based on Rome III criteria. There were 24 patients with IBS-Constipation (80%), 4 patients with IBS-Diarrhea (13.3%), and 2 patients with IBS-Mixed (6.7%) in IBS group.

Results: Biochemical parameters did not differ between groups except for slightly increased creatinine in patients with IBS. PWV and CIMT values were similar between groups. SDNN index and RMSSD were significantly impaired in patients with IBS compared to controls. Frequency analyses revealed lower LF, HF, and VLF in subjects with IBS.

Conclusion: We demonstrated decreased parasympathetic modulation in patients with constipation predominant IBS. However, we could not demonstrate any changes in vascular structure and functions measured by carotid intima-media thickness and pulse wave velocity. Our results do not support accelerated atherosclerosis in IBS population

Heart rate variability and vascular tests in two groups

| N (60) | IBS group (30) | Control group (30) | P value |
|--|----------------|--------------------|---------|
| HRV measurements | | | |
| Time domain HR variability | | | |
| SDNN, ms | 136±35 | 151±39 | NS |
| SDANN, ms | 120±27 | 137±39 | 0.060 |
| SDNN index, ms | 51±12 | 62±18 | 0.010 |
| RMSSD,ms | 28±9 | 38±15 | 0.002 |
| Total power | 2718±1201 | 4004±2112 | 0.007 |
| Frequency domain HR variability | | | |
| LF | 585±302 | 919±436 | 0.002 |
| LFnu | 0.70±0.12 | 0.70±0.09 | NS |
| HF | 228±177 | 405±275 | 0.006 |
| Hfnu | 0.26±0.07 | 0.28±0.08 | NS |
| VLF | 1871±803 | 2646±1461 | 0.016 |
| LF/HF ratio | 3.1±1.6 | 2.8±1.5 | NS |
| Vascular tests | | | |
| CIMT, mean (mm) | 0.64±0.16 | 0.70±0.23 | NS |
| CF-PWV (m/s) | 7.42±2.11 | 7.8±1.95 | NS |

HRV, heart rate variability; RMSSD, Square root of the mean squared differences of successive normal-to-normal intervals; SDNN, Standard deviation of all normal-to-normal intervals; SDANN, standard deviation of the average normal-to-normal intervals calculated over 5-minute periods of the entire recording; HF, high frequency power; LF, low frequency power; LFnu, normalized low frequency power; HFnu, normalized high frequency power;CF-PWV, carotid-femoral pulse wave velocity; CIMT, Carotid intima-media thickness

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Subclinical Left Ventricular Systolic Dysfunction in Patients with Coronary Artery Anomaly: A Speckle Tracking and Velocity Vector Imaging-Based Study

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Background: Based on current literature, coronary artery anomalies (CAA) are classified as benign and potentially serious coronary anomalies. Although most of CAA are classified as benign and asymptomatic, it can cause myocardial ischemia without atherosclerosis. In the present study, we aimed to evaluate subclinical left ventricular (LV) systolic dysfunction in patients with CAA, without any cardiovascular disease and with preserved ejection fraction (EF), by using two strain imaging methods, "speckle tracking echocardiography" (STE) and "velocity vector imaging" (VVI).

Methods: We studied 25 patients with CAA (age 58.44±10.57 years, 45% female) and 20 age and sex-matched control subjects, without any cardiac disease and with normal LV EF. Among 25 patients, 15 had anomalous origin of left circumflex (LCx) artery, 10 had anomalous origin of right coronary artery (RCA). Conventional echocardiography and 2- dimensional (2D) strain imaging were performed to analyze subclinical LV systolic function.

Results: Conventional echocardiographic measurements (LV end diastolic diameter, LV end systolic diameter and LV EF) were similar between the groups. Based on VVI analysis, LV longitudinal peak systolic strain (13.14±1.59% to 23.43±1.77%, p=0.0001) and strain rate (0.46±0.09 1/s to 4.56±0.76 1/s, p=0.0001) were significantly impaired in patients with CAA, compared to controls. Regarding STE, LV longitudinal peak systolic strain (13.05±1.25% to 17.37±2.39%, p=0.0001) and strain rate (0.38±0.09 1/s to 1.34±0.33 1/s, p=0.0001) were also markedly impaired in patients with CAA. When we correlated the two distinct 2D strain imaging modalities, we obtained a significant positive correlation. (r=0.785, p=0.0001 for strain; r=0.931, p=0.0001 for strain rate measurements).

Conclusions: It may be essential to assess the subtle changes in LV myocardial contractility in patients with CAA, because clinical consequences may be of particular importance. 2D strain imaging-based novel echocardiographic techniques may provide additional data for detecting preclinical systolic dysfunction in patients with CAA.