Conclusion: Our findings show that markers associated with inflammation in HF, especially increase in patients with ischemic HF. Therefore, we believe that the inflammatory process should be evaluated in diagnosis and treatment of patients with HF. However, this result needs to be validated in large-sized studies.

Key words: Heart Failure, Inflammation, C reactive protein.

PP-145

Cilostazol Decreases Total Atrial Conduction Time in Patients with Peripheral Artery Disease

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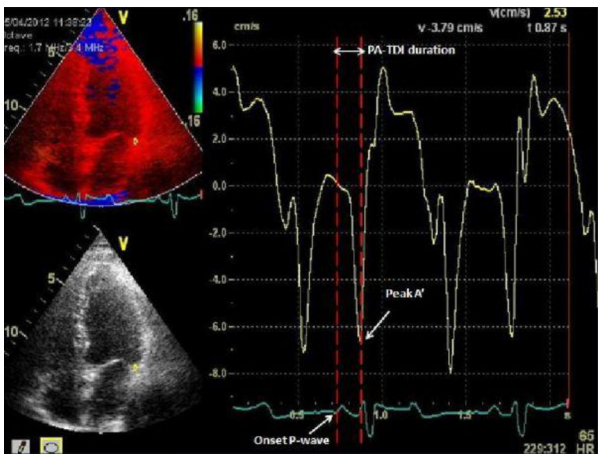
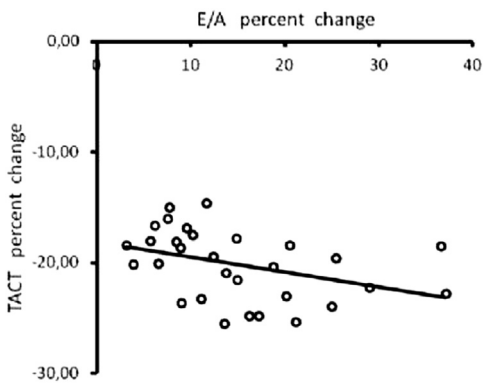
Background: Total atrial conduction time (TACT) is the most important parameter predicts the development of new-onset atrial fibrillation. We investigate the effect of cilastazol therapy on TACT in patients with peripheral artery disease.
Methods: Thirty patients with peripheral artery disease were treated with cilastazol (200mg/day) for 6 months. Baseline echocardiographic total atrial conduction time parameter was compared with the 6-month follow-up.
Results: The TACT duration (Figure 1) was decreased in all patients compared with baseline after therapy (121.8±19.3 vs. 109.1±15.9 milliseconds, p<0.001). However, LA diameter was not different at the sixth month of therapy compared with the baseline. The reduction of TACT duration was correlated with the increase in mitral E wave velocity/mitral A wave velocity ratio (r=-0.48, P < 0.003) (Figure.2).
Conclusion: Our result showed that 200-mg cilastazol treatment decreased TACT duration in patients with peripheral artery disease, which suggest that there might be a link with cilastazol treatment and atrial fibrillation development and/or recurrence.





PP-146

Increased Mean Platelet Volume May Reflect a Disturbance in the Autonomic Nervous System in Patients with Vasovagal Syncope

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Objective: Vasovagal syncope (VVS) is supposed to be modulated by increased sympathetic tone following an orthostatic maneuver. This pathological process mainly related to a generalised involvement of the autonomic nervous system (ANS). Increased mean platelet volume (MPV) may reflect increased platelet activation or increased numbers of large, hyperaggregable platelets an, is accepted as an independent cardiovscular risk factor. Increased sympathetic activity may have an important role in MPV, either by peripheral activation and splenic release or by effects on thrombocytopoiesis. Since sympathovagal balance is affected in patients with VVS, in the present study, we aimed to show the effects of increased sympathetic activity on platelet size during asymptomatic time periods in patients with VVS.
Materıal-Methods: Thirty seven patients with VVS were compared with age- and sex-matched 33 patients without VVS. All patients were underwent into 24-hour holter monitoring for assessing heart rate (HR) variability analysis. Time-domain HRV analysis were done. Blood samples were taken for MPV measurements were taken before the holter monitoring. Statistical analyses (Independent-Samples T test and Chi-Square tests) were used to evaluate the differences between two groups.
Results: Group 1 was consisted of 37 patients with VVS (mean age 35,27±20,09 years, 22 male (66,7%9)) and group 2 was consisted of 33 patients without VVS (mean age 29,27±11,68 years, 25 male (67,6%)). In terms of basal demographic characteristics, there was no statistically significant difference between two groups. When we evaluated the time-domain HRV analysis parameters, we observed that SDNN, SDNN index, SDSD, RMSDD, PNN50 count were significantly lower in patients with VVS (p<0.05 for all). Also, MPV was found significantly higher in patients with VVS. Pearson's correlation analysis showed that MPV was moderately negatively correlated with SDNN (r=-0,421), SDSD (r=-0,396), NN50 count (r=-0,395), RMSDD (r=-0,393). There was not a statistically significant correlation between MPV and time-domain HRV analysis parameters in patients without VVS.
Conclusion: We found that MPV was significantly higher in the patients with VVS, and MPV is also closely associated with increased sympathetic activity in patients with VVS. Our analysis supports the hypothesis that alterations of autonomic status may play a role in the development of platelet size.

	GROUP 1 (n=37)	GROUP 2 (n=33)	P value
Age, (years)	35,27 ± 20,09	29,27 ± 11,68	0,138
Male, n (%)	22 (66,7%)	25 (67,6%)	0,936
HT, n (%)	6 (18,2%)	8 (21,6%)	0,719
HL, n (%)	4 (12,1%)	5(13,5%)	0,862
DM, n (%)	5 (15,2%)	4(10,8%)	0,588
SMOKING, n (%)	8 (24,2%)	8 (21,6%)	0,592
ALCOHOL, n (%)	7 (21,2%)	6 (16,2%)	0,794
SDNN, (msec)	149,56 ± 51,55	181,98 ± 59,31	**0,017**
SDSD, (msec)	44,94 ± 23,40	63,29 ± 39,57	**0,020**
NN50 count, (%)	12495,81 ± 11010,67	19434,75 ± 13573,27	**0,021**
RMSDD, (msec)	44,98 ± 23,44	63,35 ± 39,59	**0,020**
SDANN, (msec)	59,99 ± 54,46	62,00 ± 57,84	0,884
SDNN index	56,97 ± 29,27	75,42 ± 39,94	**0,030**
PNN50	12,49 ± 12,02	22,71 ± 16,95	**0,005**
WBC	6786,48 ± 1787,01	6266,66 ± 1552,35	0,201
HB	13,84 ± 1,51	14,49 ± 1,35	0,064
HCT	41,74 ± 4,06	43,28 ± 3,65	0,102
PLT	254,18 ± 66,28	260,54 ± 52,94	0,662
MPV	9,21 ± 0,70	8,46 ± 0,73	**< 0,001**
PDW	16,39 ± 2,09	15,21 ± 1,87	**0,016**
MCV	85,56 ± 8,10	87,47 ± 4,36	0,232
RDW	14,64 ± 1,65	14,04 ± 1,55	0,084
Neutrophil	3984,05 ± 1569,48	3439,09 ± 1054,88	0,097
Lmphocyte	2063,51 ± 513,21	2137,36 ± 680,63	0,608
N/L ratio	2,03 ± 0,87	1,73 ± 0,78	0,150

PP-147

Association Between Serum Total Antioxidant Status and Flow Mediated Dilatation in Patients with Systemic Lupus Erythematosus

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Objective: To evaluate the relationship between the endothelial dysfunction assessed by flow mediated dilation (FMD) in the brachial artery and serum total antioxidant status (TAS) in SLE patients.
Methods: Thirty-four patients with SLE and thirty-nine healthy volunteers without any cardiovascular disease and atherosclerotic risk factors were included in this study. Doppler ultrasound system was used to measure FMD from the brachial artery in the antecubital fossa to assess endothelial function. Serum TAS was measured with TAS kit. High-sensitivity C-reactive protein (hs-CRP), a marker of inflammation, was also determined.
Results: The mean TAS value was significantly lower in patients with SLE than in controls (1.60±0.11 versus 1.73±0.15; p<0.0001). hs-CRP levels were significantly higher in patients with SLE than in controls (8.2±6.0 vs 2.9±4.0; p<0.0001). There was no difference between groups regarding baseline and hyperemic diameters. However, FMD percent was found to be significantly lower in SLE patients than in controls. FMD significantly positively correlated with TAS (r=0.448, p=0.001) and significantly inversely correlated with serum hs-CRP levels (r=-0.314, p=0.001). In regression analysis, only TAS was independently correlated with FMD (b=0.575, p=0.002).
Conclusion: SLE patients without cardiovascular risk factors have endothelial dysfunction and this can be releated with underlying inflammation and impairment of TAS.