Table 1. Baseline characteristics and laboratory findings of the study groups

<table>
<thead>
<tr>
<th>Variables</th>
<th>Control group (n=46)</th>
<th>Risky group (n=36)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>13.91±1.31</td>
<td>14.56±1.73</td>
<td>0.060</td>
</tr>
<tr>
<td>Gender (Male)</td>
<td>26 (57)</td>
<td>20 (56)</td>
<td>0.930</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>19.78±3.25</td>
<td>20.29±3.59</td>
<td>0.500</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>111.85±12.75</td>
<td>113.31±14.73</td>
<td>0.633</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>69.46±8.11</td>
<td>72.44±9.10</td>
<td>0.121</td>
</tr>
<tr>
<td>Total cholesterol (mg/dl)</td>
<td>156.76±27.62</td>
<td>165.12±35.60</td>
<td>0.403</td>
</tr>
<tr>
<td>Triglyceride (mg/dl)</td>
<td>112.35±55.16</td>
<td>104.91±48.44</td>
<td>0.626</td>
</tr>
<tr>
<td>LDL-C (mg/dl)</td>
<td>84.23±22.26</td>
<td>93.76±32.30</td>
<td>0.282</td>
</tr>
<tr>
<td>HDL-C (mg/dl)</td>
<td>63.65±13.41</td>
<td>56.55±14.95</td>
<td>0.106</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>0.49±0.11</td>
<td>0.51±0.13</td>
<td>0.644</td>
</tr>
<tr>
<td>Fasting glucose (mg/dl)</td>
<td>87.63±9.91</td>
<td>90.27±9.19</td>
<td>0.275</td>
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<tr>
<td>Hemoglobin (g/dl)</td>
<td>12.35±0.85</td>
<td>12.31±1.25</td>
<td>0.899</td>
</tr>
<tr>
<td>hs-CRP (mg/L)</td>
<td>0.46 (0.24-1.62)</td>
<td>0.36 (0.23-0.75)</td>
<td>0.582</td>
</tr>
</tbody>
</table>

Table 2. Distribution of genotype and allele frequencies and odds ratios and 95% confidential intervals of the atherosclerosis risk among CRP, eNOS, and IL-6 polymorphisms

<table>
<thead>
<tr>
<th>SNP</th>
<th>Genotypes</th>
<th>Control group (n=46)</th>
<th>Risky group (n=36)</th>
<th>P value</th>
<th>Odds ratios (CI) 95%</th>
</tr>
</thead>
<tbody>
<tr>
<td>CRP-13444 C/T</td>
<td>G/G</td>
<td>24 (52.2)</td>
<td>11 (30.6)</td>
<td>0.585</td>
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</tr>
<tr>
<td></td>
<td>G/T</td>
<td>19 (41.3)</td>
<td>19 (52.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>T/T</td>
<td>2 (4.5)</td>
<td>2 (5.6)</td>
<td></td>
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<tr>
<td>eNOS-786 C/T</td>
<td>G/G</td>
<td>67 (73.8)</td>
<td>51 (70.8)</td>
<td>0.390</td>
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</tr>
<tr>
<td></td>
<td>G/C</td>
<td>25 (27.2)</td>
<td>21 (29.2)</td>
<td>0.275</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C/C</td>
<td>0.012</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IL-6 -572 C/T</td>
<td>G/G</td>
<td>43 (91.3)</td>
<td>25 (69.4)</td>
<td>0.012</td>
<td></td>
</tr>
<tr>
<td></td>
<td>G/C</td>
<td>2 (4.3)</td>
<td>10 (27.8)</td>
<td>3.48 (1.17-10.32)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C/C</td>
<td>1 (2.2)</td>
<td>1 (2.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CRP</td>
<td></td>
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</table>

PP-112

The Role of the Nonspecific Inflammatory Markers in Determining the Anatomic Extent of Venous Thromboembolism

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Objective: The aim of this study was to investigate the relationship between the anatomic extent of venous thromboembolism (VTE) and nonspecific inflammatory markers such as Neutrophil to lymphocyte ratio (NLR) and high-sensitivity C-reactive protein (hs-CRP).

Methods: We retrospectively enrolled 77 patients with VTE (Distal deep vein thrombosis (DVT) (n=19), proximal DVT (n=32) and pulmonary thromboembolism (PTE) (n=26)) and 34 healthy controls. Distal and proximal DVT was diagnosed by using peripheral vascular duplex ultrasonography. Proximal DVT was defined as thrombosis at the level of the popliteal veins or above. Distal DVT was defined as thrombosis occurring within the calf veins. The diagnosis of PTE was confirmed in all cases by computed tomography. Total and differential leukocyte count and hs-CRP were determined by standard laboratory.

Results: It was detected in patients with VTE that WBC (8.5±2.06 vs 6.5±0.95, p<0.001), CRP (3.41±1.41 vs 1.80±0.70, p<0.001) and hs-CRP (2.00±2.68 mg/L vs 0.68±0.35 mg/L, p=0.005) levels were significantly higher in comparison with the healthy control group. According to the analysis made by ANOVA, the levels of WBC, NLR and hs-CRP were clearly different among the groups (control, distal and proximal DVT and PTE) (p<0.001). According to ANOVA test with LSD as a post-hoc test performed to find the source of the difference between the groups, only hs-CRP was found high in PTE group in comparison with the other groups; and no difference was detected between the other groups. A significant increase from the control group to DVT and PTE was observed in the analysis made for NLR. In the analysis for WBC, while there was no difference between the control group and distal DVT, it was found significantly higher in the proximal DVT and PTE (Table 1). ROC curve analysis was performed in order to find the best cut-off values of these variables in predicting VTE. AUC for hs-CRP, high-sensitivity C-reactive protein, data are shown as n (%), mean±SD, median [IQR].

Conclusions: These findings suggest that the inflammatory process might have an important role in the prothrombotic state in patients with VTE. Also, NLR may be effective in determining the anatomic extent of VTE. NLR is insensitive and readily available markers that may be useful for risk stratification in patients with VTE.

PP-113

Serum Uric Acid Level is correlated with Decreased Blood Flow Velocity of Left Atrial Appendage in Patients with Atrial Fibrillation

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Objective: The coexisting clinical, echocardiographic and biochemical risk factors have been identified in relation to an increased risk of thromboembolism in patients treated with oral anticoagulants and have been suggested as possible markers of high thrombotic potential. The present study was aimed at evaluating the possible association between serum uric acid levels and the blood flow velocity of the left atrial appendage (LAA) in patients with permanent atrial fibrillation (AF).

Methods: The study included 160 patients (77 men, 83 women) with persistent AF, aged 65±10 years, who were treated with oral anticoagulants. The indications for anticoagulant therapy were atrial fibrillation with cardioembolic source, atrial fibrillation with heart failure, or atrial fibrillation with left atrial thrombus. The blood flow velocity of the LAA was evaluated by transesophageal echocardiography. The patients were divided into two groups according to their serum uric acid levels: low uric acid group (<7 mg/dl) and high uric acid group (≥7 mg/dl). The demographic characteristics, clinical and echocardiographic data of the patients were compared between the two groups with the Student’s t-test.

Results: The mean serum uric acid level of the patients was 6.5±2.5 mg/dl. The mean blood flow velocity of the LAA was 21.2±7.8 cm/s. The blood flow velocity of the LAA was significantly higher in the low uric acid group than in the high uric acid group (22.5±7.8 vs 19.9±6.3 cm/s, p=0.03). The mean serum uric acid level in the low blood flow velocity group was 5.9±2.1 mg/dl, while the mean serum uric acid level in the high blood flow velocity group was 7.3±2.7 mg/dl (p=0.007). The serum uric acid level was positively correlated with the blood flow velocity of the LAA (r=0.31, p=0.007).

Conclusions: The present study suggests that serum uric acid level is associated with the blood flow velocity of the LAA in patients with permanent AF. This association may be useful in the management of patients with AF.