with atrial fibrillation (AF). Serum uric acid (SUA) is the end product of purine metabolism catalyzed by xanthine oxidoreductase, which has been found in the left atrium of patients with AF, and increased SUA level could be a sign of oxidative damage and inflammation, which play a role in the pathogenesis of AF. Although it has been demonstrated that SUA level is tend to be higher in patients with AF, the relationship between the contractile function of left atrial appendage (LAA) and SUA level has not been fully elucidated. However, elevated SUA level may be an additional cumulative risk factor for increased thromboembolic events in patients with AF. We aimed to examine the relationship between SUA level and contractile function of LAA in a short cohort of patients with AF.

Material-Methods: One hundred thirty patients with AF in whom transesophageal echocardiography was performed to guide the decision about when to perform cardioversion were enrolled to this study. Patients were categorized according to their blood flow velocity of the LAA. Group 1 was consisted of 68 patients with low LAA flow velocity (<35 cm/s) and group 2 was consisted of 62 patients with normal LAA flow velocity (\ge 35 cm/s). Statistical analyses (Independent-Samples T test and Chi-Square tests) were used to evaluate the differences between two groups.

Results: The blood flow velocity of LAA was 24.82±5.75 cm/sn in group 1 and 48.41±12.68 cm/sn in group 2 (p value < 0.001). Although there was no difference for the presence of thrombi in the LAA between two groups, spontaneous echo contrast was more prominent in patients with group 2 (p value <0.001). Also, we found that AF patients with low blood flow velocity were composed of older patients (p value 0.013). We found a statistically significant difference for SUA between two groups (7.11±1.89 mg/dl for group 1 and 6.07±1.49 mg/dl for group 2, p value 0.001). SUA levels were positively correlated with left atrial diameter (r=0.336, p value 0.008) and there was a negative correlation between SUA and blood flow velocity of LAA (r=-0.256, p value 0.003). (Table 1).

Conclusion: The measurement of SUA is a simple, easily determined marker of inflammation and oxidative stress at a low cost. Elevated SUA levels may be associated with the decreased contractile function of LAA and may provide additional prognostic information with future thromboembolic events in patients with AF. However, it is controversial whether an elevated SUA is a cause or a consequence of decreased contractile function of LA and/or LAA. Further randomized, prospective, large-scale studies are needed to elucidate the exact pathophysiologic and prognostic role of SUA in this setting and assess its potential clinical relevance.

Results of the Study

	GROUP 1 (n=68)	GROUP 2 (n=62)	P value
Age, years	68.54 ± 9.39	63.09 ± 14.87	0.013
Male, n (%)	36 (52.9%)	46 (74.2%)	0.018
CAD, n (%)	25 (36.8%)	20 (32.3%)	0.712
HT, n (%)	33 (48.5%)	21 (33.9%)	0.110
HL, n (%)	17 (25%)	10 (16.1%)	0.280
DM, n (%)	9 (13.2%)	9 (14.5%)	1.000
Heart rate (bmp)	90.45 \pm 21.93	92.12 \pm 18.06	0.638
DBP (mmHg)	71.14 ± 13.28	73.95 \pm 12.65	0.465
SBP (mmHg)	123.82 \pm 21.75	124.45 ± 24.45	0.926
Blood flow velocity of LAA (cm/sn)	24.82 ± 5.75	48.41 ± 12.68	< 0.001
SEC, n (%)	44 (67.6%)	14 (22.6%)	< 0.001
LAA Thrombus, n (%)	14 (20.6%)	6 (9.7%)	0.095
LVIDd, (mm)	$\textbf{51.20} \pm \textbf{6.04}$	$\textbf{49.42} \pm \textbf{8.39}$	0.363
IVSd, (mm)	11.03 \pm 2.36	$\textbf{10.33} \pm \textbf{2.16}$	0.316
LA, (mm)	47.64 ± 9.26	41.67 ± 7.62	0.008
LVEF, (%)	49.02 \pm 11.89	$\textbf{53.05} \pm \textbf{14.53}$	0.224
Glucose, (mg/dl)	106.42 ± 24.54	120.25 \pm 51.74	0.059
Urea, (mg/dl)	47.31 \pm 32.46	$\textbf{39.04} \pm \textbf{13.37}$	0.064
Creatinine, (mg/dl)	$\textbf{1.17} \pm \textbf{0.34}$	$\textbf{1.08} \pm \textbf{0.28}$	0.108
Uric Acid, (mg/dl)	7.11 \pm 1.89	$\textbf{6.07} \pm \textbf{1.49}$	0.001
HDL-C, (mg/dl)	43.41 ± 13.56	45.46 ± 14.72	0.409
LDL-C, (mg/dl)	107.42 ± 33.50	113.15 \pm 33.84	0.334
TG, (mg/dl)	135.20 \pm 91.41	139.59 \pm 75.30	0.767
Total-C, (mg/dl)	176.05 ± 44.47	187.29 ± 47.23	0.165

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Association between Ascending Aorta Dilatation and Serum $\gamma\text{-}Glutamyltransferase\ Levels}$

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Introduction: Recent studies identified possible roles of oxidative stress and increased inflammatory status in the pathogenesis of ascending aortic dilatation. High serum gamma-glutamyltransferase (GGT) levels indicate a marker of oxidative stress.GGT level is positively associated with the development of hypertension. In this study we aimed to eveluate the relation between serum GGT levels and assendan aort dilatation.

Method: There were 79 patients enrolled whose assendan aorta was measured with two dimentional ecocardiography and classified to two groups as those with dilated assendan aorta n:36 and those with a normal aortic diameter n:43. We have measured the GGT,CRP,glucose,uric ascit levels and lipid prophyl in th blood samples of the patients mentioned.

Results: Serum GGT and CRP levels were significantly higher in those patients with dilated assendan aorta (p:0,001 and p:0,01 respectively). Also hypertention and diabetes mellitus frequency was significantly higher in the same group (p:0,001 and p: 0,002 respectively). Statisticely positive correlation was showen with serum CRP (r=0.3, p=0.006),GGT (r=0.4, p=0.01) levels and assenden aorta diameter. Logistic regression analysis showed that HT,GGT and DM were independent predictors for dilatation of assendan aorta.

Conclusion: Serum GGT levels were higher in those patients with dilated assendan aorta. Likewise the other studies our study showed that serum GGT levels are considerable for the patogenesis of HT also can lead to aortic dilatation via causing oxidative stress and inflammatuary stress responce.

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Time in Therapeutic Range (TTR) Value of Patients who use Warfarin and Factors which Influence TTR

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Background and Aim: Atrial fibrillation (AF) and presence of prosthetic valve are common comorbidities that necessitate warfarin treatment. The risk of complication increases when INR (International normalized ratio) values are out of therapeutic range. Mean time in therapeutic range (TTR) below 60% indicates that warfarin is inefficient. In our study, we aimed to determine TTR values in patients who were on warfarin treatment and had valvular AF, nonvalvular AF and prosthetic valve. We also aimed to identify the factors affecting TTR in these patients.

Method: Among patients admitted to out-patient clinics between June 2012 and December 2012, we enrolled 248 patients who were on warfarin treatment for valvular-nonvalvular AF or prostethic valve. TTR values of patients were calculated by using linear interpolation method. A cut off value of 60% was used to assess efficiency of TTR. Patients were classified into two groups according to their TTR values (≥60% vs. <60%) and the characteristic features of these groups were compared. Patients were classified further into three groups according to their indications for warfarin (valvular AF, nonvalvular AF and prostethic valve). HASBLED score was used to identify tendency for bleeding complication. Weekly warfarin dosage and duration of warfarin use were noted for each patient.

Results: The mean age of the patients was 57.21 ± 12.45 years and 66.9% of the patients were female. Mean TTR value was $55.92 \pm 27.84\%$ and 48% of the patients had TTR values above 60% threshold. There was no statistical difference among nonvalvular AF, valvular AF and prosthetic valve groups in terms of TTR values and TTR efficiency (p:0.668 and p:0.901, respectively). We did not find any clinical and demographic differences between two TTR groups (TTR $\geq 60\%$ vs. < 60%). The patients with nonvalvular AF were older (p<0.001), had higher prevalence of coronary artery disease and hypertension (p:0.004 and p:0.001, respectively). The leeding scores were higher while total warfarin doses were lower in nonvalvular AF group (p:0.03 and p:0.004, respectively). The percentage of patients with a history of previous thromboembolic or bleeding complications and the duration of warfarin use were higher in the prosthetic valve group (p:0.027 and p:<0.001, respectively). The mean TTR value did not differ according to socioeconomic status and comorbidities. There was no relationship between the presence of a previous complication and TTR (p:0.995).

Conclusions: In our study, we found that our study subjects had low TTR values and that TTR values were not associated with the indication for warfarin or with the characteristics of the patients.

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