The effects of three months aerobic exercise on novel atherosclerosis risk factors in untrained middle aged men

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Abstract: Recent researches have shown that inflammatory mechanisms are responsible for formation of atherosclerotic lesions. The aim of this study was to examine the effects of three months aerobic exercise on body composition and blood levels of homocysteine, hsCRP, lipids profile, glucose, fibrinogen, D-dimer, uric acid and white cells count in Iranian healthy and sedentary middle aged men. The subjects of this study were 21 healthy, overweight and inactive middle aged men living in Mashhad city. They were randomly assigned into control (n=10) and experimental (n=11) groups. Exercise training included three months (three days per week) running at 75% to 85% intensity of maximum heart rate. Blood samples were taken from the subjects before and after training. Statistical analysis showed significant reduction of homocysteine, hsCRP, TG, TG/HDL, uric acid, body mass index and obesity degree, also significant elevation of white cells count and Vo2max after training in the experimental group (P<0.05). Three months running exercise through the reduction of homocysteine, hsCRP, uric acid and TG can be effective in reduction of atherosclerosis risk in healthy middle aged men. More research is needed about mechanisms of involved in adaptation of atherosclerosis risk factors to regular exercise in this population.

Keywords: training, homocysteine, hsCRP, fibrinogen, novel risk factors.

Introduction

Nowadays, overweight and obesity have become a worldwide problem. Excess body fat is associated with several diseases such as cardiovascular disease (CVD), insulin resistance, hypertension, diabetes mellitus and dyslipidemia [1]. In recent decades, CVDs are the foremost cause of morbidity and mortality throughout the world [2]. Epidemiologic studies have shown that a number of risk factors including alcohol intake, dyslipidemia (high levels of LDL, TC and TG and low levels of HDL), smoking, physical inactivity and psychological stress are associated with CVD. Prevention strategies aiming to reduce these risk factors were accomplished but despite implementation of these strategies, it was observed that the prevalence of CVD especially atherosclerosis is escalating to a larger degree. This problem led to discovery of various novel cardiovascular risk factors. Homocysteine, high sensitive C-reactive protein (hsCRP), HDL, LDL, fibrinogen, D-dimer, uric acid, white blood cells (WBC) and red blood cell distribution width (RDW) are some novel and traditional
cardiovascular risk factors that predict the risk of atherosclerosis independently [3-9]. Homocysteine is a sulfur containing amino acid made during dietary amino acid methionine. Defects in remethylation and transsulfuration pathways such as enzymatic defects and vitamin B deficiencies are common causes of hyperhomocysteinemia and homocysteinuria [10]. Homocysteine through several mechanisms, incorporate into plaque formation in coronary artery diseases: endothelial damage and dysfunction, vascular smooth muscle cells proliferation, Platelet Aggregation and Thrombosis, foam cell formation, formation of proliferative fibrous plaques, cell structure damages, reduction in NO bioavailability, elevation of lipid peroxidation, upregulation of adhesion molecules, and fibrinolysis reduction [10-12]. CRP is an acute phase protein and its levels tend to be raised during inflammation, infection, and tissue injury. Primarily, this inflammatory marker is secreted by hepatocytes under stimulation of IL-6 but endothelial cells can also generate the CRP [13]. The CRP through several ways increases the risk of atherosclerosis. It binds to membranes of damaged cells and activates the opsonisation of these cells; also this protein stimulates the endothelial cells to express adhesion molecules and represses the synthesis of NO by these cells [14]. Fibrinogen is synthesized by the liver under induction of interleukin-6 (IL-6). It stimulates platelet and erythrocyte aggregation, and induces adhesion of platelets and leukocytes to endothelium through up regulating gene expression of Inter-Cellular Adhesion Molecule 1 (ICAM1) [15]. D-dimer that shows rate of fibrin turnover in blood stream, is a novel cardiovascular risk factor, because elevated levels of this marker is associated with formation of thrombin and coagulation of blood [16]. URIC ACID, a final product of purine metabolism in humans, by the several mechanisms raises the risk of atherosclerosis, including vascular smooth muscle cells growth and proliferation, suppression of nitric oxide synthesis, vascular inflammation and elevation of reactive oxygen species [17]. Monocytes have an effective role in endothelium injury and formation of thrombosis by release of oxidant products. Also when white blood cells are activated, induce inflammatory processes that lead to vascular injury and formation of fatty streaks [7]. RDW shows numerical variability in erythrocytes size and is a predictor of morbidity and mortality in males with acute coronary syndrome, because increased levels of RDW induce an inflammatory state. Deficiency of folate, B12 vitamin and iron are associated with raised levels of RDW [8]. Regular physical activity is an effective way to improve cardiovascular risk factors and reduce the prevalence of CVD. Recently two Meta analyses have shown that there is a little evidence about the effects of regular exercise on homocysteine and hsCRP levels [9, 10]. Heffernan et al (2009) stated that aerobic and resistance exercises are beneficial exercise trainings for primary CVD prevention and health promotion [18]. Kelley and Kelley (2008) in their Meta-analysis noted that there is scant evidence about the beneficial effects of exercise on homocysteine levels in adults with normal weights, but exercise may significantly reduce the homocysteine levels in overweight and obese adults. However, before any decisive remark, more studies should be conducted in this field [19]. Thomas et al (2007) reported that in children between 12 to 13 years old fitness and fatness were significantly correlated to
fibrinogen levels [20]. Balagopal et al (2008) showed that after three months regular physical activity, blood levels of fibrinogen and fibrin D-dimer reduced significantly in obese children aged between 14 to 18 years [21]. Tekin (2010) for determining the effect of aerobic and anaerobic exercise program on levels of URIC ACID trained twenty male subjects in duration of six weeks (five days a week). He reported significant raise in URIC ACID levels after exercise intervention [22]. Keen et al (1995) surveyed changes of leucocyte levels during a multi stage race (the Milk Race). They showed elevation of number of leucocytes (including basophils, eosinophils, monocytes, neutrophils and lymphocytes) after this race [23].

Several mechanisms are involved in development of atherosclerotic plaques and complexity of these processes and their interaction with physical stress is an interesting research subject. All adaptations of novel atherosclerosis risk factors to regular exercise in different populations are not clearly understood. Also no study was found about the effect of three months aerobic exercise on the blood levels of homocysteine, hsCRP, lipids, glucose, fibrinogen, D-dimer, uric acid, WBC and RDW in healthy and untrained middle aged men. Thus, this study was conducted to examine the effects of three months regular aerobic exercise training on body composition and aforementioned cardiovascular risk factors in healthy and inactive urban middle aged men.

### Material and Methods

This research plan was confirmed by Research Assembly of Physical Education and Sport Sciences faculty of Ferdowsi University of Mashhad, Iran. The subjects of current study included twenty one healthy and inactive middle aged men with overweight (25<BM1<30) living in Mashhad, Iran. They were recruited by several colleges of Ferdowsi University of Mashhad and randomly assigned into control (n=10) and exercise (n=11) groups (table 1). Their physical activity levels were determined by a physical activity questionnaire. Additionally the health status of the subjects was assessed by a physician (at a medical clinic) and a health questionnaire; their abilities to start the exercise training were assessed through the Physical Activity Readiness Questionnaire (PARQ). The qualifications of study subjects included: without history of metabolic and cardiac diseases, without special drug usage, without regular physical activity and having enough ability to regularly participate in training program. Before beginning the study, every subject signed an informed consent document [24,25].

Just before starting the exercise trainings, blood samples were obtained following 10 to 14 hours fasting from left brachial vein in sedentary position and sent to the laboratory for analysis. Additionally aerobic power (\(\text{Vo2max}\)) and the body composition status of the subjects were determined in the sport sciences laboratory in faculty of physical education and sports sciences of Ferdowsi University of Mashhad. The subjects were asked to avoid severe physical activity two days before the pretest measurements. Bruce protocol on a treadmill (TechnoGym, S.P.A, Class: Runrace 1400HC, Italy) was conducted for determination of subjects aerobic power. The body composition status of the subjects was measured through a body composition analyzer (InBody 720, English). All of these methods were repeated after three months regular exercise. Blood samples were taken from the subjects
after finishing three months exercise trainings.

The experimental subjects participated in running exercise for three months and three days per week. In each session, the subjects warmed up through stretching, running and limbering up stirs in 20 minutes. The main part of training protocol included 15 minutes continuous running with intensity of 75 to 85 percent of MHR (in each two sessions, one minute was added to the starting time reach to 30 minutes, This time was stable until the last session). Intensity of training controlled via Polar watches. The subjects performed 10 minutes walking and stretching activities for cool down [26].

During the study, the subjects were asked to avoid nutritional supplements (specifically B6, B9 and B12 supplements) [25]. The subject's weight, body mass index (BMI), waist to hip ratio (WHR), percent body fat (PBF), body fat mass (BFM), visceral fat area (VFA) and obesity degree were determined by body composition analyzer. All biochemical procedures were done via an auto analyzer system. Immunoturbidimetric method with commercial kits of Pars Azmoon Company (Iran, Tehran) was used to measure the serum hsCRP levels. For determination of homocysteine levels, Elisa method with an especial kit was used. Blood lipids and lipoproteins (TG, TC, LDL, and HDL) of the subjects were determined via enzymatic methods and glucose was measured through glucose oxidase method with especial kits. Lipid ratios including LDL/HDL, TG/HDL and TC/HDL were also calculated. CBC factors (including WBC, RBC, Hb, Hct, MCV, MCHC, Plt, Neutrophil count, Lymphocyte count, Monocyte count, Eosinophil count, RDW) were determined using CBC-MAC cell counter (KX-21), plasma fibrinogen and serum uric acid levels were measured using Pars Azmoon kit (Iran) and auto analyzer system, ELISA assay and Stago kits were used to determine plasma levels of fibrin D-dimer and levels of troponin-I molecules were specified using ELISA method.

The following statistical analysis was used in the present study: descriptive statistics (for mean and standard deviation of dependent variables), colmogorov smirnov test (for evaluation of data normality) and paired sample T test (for comparing of pre test and post test mean levels of dependent variables in each group). All of these tests were performed with SPSS version 15.0 and a value of 0.05 was accepted as reflecting significance.

**Results**

Before commencement of the training, the subjects of two groups were the same in age, height, weight, BMI, WHR, BFM, PBF, VFA and \( \text{Vo}_2\text{max} \). Kolmogorov Smirnov test showed the normality of data in both groups (P<0.05).

Statistical analysis showed significant reduction of homocysteine, hsCRP, TG, TG/HDLC, uric acid, RDW, body mass index and obesity degree, also significant elevation of WBC, RBC, Hb, Hct, MCHC, Plt, lymphocyte count, monocyte count and eosinophil count and \( \text{Vo}_2\text{max} \) after training in the experimental group (P<0.05). However, the above mentioned factors (except lymphocyte count) did not change significantly after 3 months (without training) in the control group (P>0.05) (tables 2 & 3). In exercise group levels of LDL, HDLC, TC, glucose, TC/HDL, LDL/HDL, fibrinogen and D-dimer did not change significantly (P>0.05).
Discussion
The results of present study showed a meaningful reduction of homocysteine levels in the exercise group. This finding is in agreement with the findings of Zuehlsdorff (2003) and Alipour et al (2007) [27, 28]. Zuehlsdorff (2003) in his research reported that twelve weeks daily exercise (muscular strength and endurance and cardio respiratory endurance) can significantly reduce the homocysteine levels in sedentary men and women who are between 32 and 50 years [27]. Alipour et al (2007) showed significant reduction of homocysteine levels in male rabbits after twelve weeks treadmill exercise [28]. However the finding of Okura et al (2006) was in disagreement with our results. Okura et al (2006) investigated the effect of 20 weeks aerobic exercise on homocysteine levels in 816 white and black men and women and concluded that in individuals with hyperhomocysteinemia, aerobic training had beneficial effects but after this training program the homocysteine levels increased slightly in those who had the normal levels of homocysteine [29]. In the study of Okura et al (2006), despite the significant increase of homocysteine levels in the whites after finishing of training period, the homocysteine levels in the blacks did not change significantly. Their subjects had the maximum TC levels of 350 mg/dl and the maximum TG levels of 500 mg/dl. However the subjects of the current this study had the maximum TC and TG levels of 252 and 235 mg/dl, respectively. In addition, in the study of Okura et al (2006), the homocysteine levels decreased in the subjects with hyperhomocysteinemia but this factor increased slightly in the subjects with normal levels of homocysteine [29]. Intensity of the training and age range explain the difference between our results and the findings of the Okura et al (2006). The main part of training protocol in the study of Okura et al (2006) included physical activity in cycle ergometer with intensity of 55% of V_o2max in first session (30 minutes) until 75% of V_o2max in the last six weeks (50 minutes) [29], however the main part of the present study protocol, included continuous running with intensity of 75 to 85 percents of MHR in first session (15 minutes) until 30 minutes with the same intensity in last sessions. Widespread age range of the study of Okura et al study (2006) (17-65 years) was the other effective factor that induced different results between their study and our study. In the current study, age range of the subjects was between 36 and 52 years (in this range, the homocysteine levels does not affect by age variations).

The mechanisms that regular exercise can induce the reduction of homocysteine levels are multiple. Long time exercise leads to increasing needs of energy supply through the catabolism of amino acids including methionine and the reduction of methionine through this mechanism can lead to the reduction of homocysteine [11]. Regular aerobic exercise through the elevation in absorption of vitamins B and the reduction of BMI and PBF induces the decrease of homocysteine levels [19,30,31]. Methionine metabolism is regulated by insulin action. Insulin does this action through the influence on several enzymes that involved in homocysteine metabolism (cystathionine beta synthase, methylenetetrahydrofolate reductase, cystathionine Gama lyase and betain homocysteine methyl transferase) [32-34]. Improved insulin sensitivity through regular exercise can decrease the levels of homocysteine in active persons. Regular exercise leads to improved insulin sensitivity through several mechanisms:
Several mechanisms are responsible for reduction of CRP levels after a long term exercise. Long term exercise is associated with high aerobic power, low psychological stress and low BMI and PBF (in this study only aerobic power, BMI and PBF were measured) and these factors are connected with CRP levels [38]. Regular physical activity leads to improved insulin sensitivity and reduced CRP secretion of endothelium [14]. Several adipokines including TNF-α, leptin and IL-6 stimulate hepatocytes for synthesis and secretion of CRP. Regular physical activity through reduction in secretion of these adipokines and reducing of PBF (that induce the reduction of TNFα, leptin and IL-6) can lessen CRP secretion by hepatocytes [38-40]. Overall, regular physical activity through reduction in production of cytokines from adipose tissue and endothelium, improvement in endothelial function, reduction of leptin and elevation of adiponectin can reduce CRP levels [40].

Results of this study showed nonsignificant reduction of plasma fibrinogen levels and nonsignificant reduction of fibrin D-dimer levels after three months exercise. Kushnick (2003) reported that 12 weeks strength training program does not affect fibrinogen levels in college aged women and men [41]. Furukawa et al (2008) studied the effect of 12 weeks walking program on fibrinogen in women aged between 32 and 57 years. They noted that after this program fibrinogen levels of women did not change significantly [42]. Borer (2001) examined the effect of 15 weeks endurance exercise (5 days per week) on levels of fibrinogen in postmenopausal women. They showed significant elevation of fibrinogen levels after regular endurance exercise [43]. In a cross sectional study, Myint et al (2008) reported inverse association between regular physical activity...
and fibrinogen levels [44]. These studies show that age and gender of subjects, type, intensity and duration of training are effective factors on response of fibrinogen to regular exercise, as low intensity endurance exercise has no beneficial effect on this atherosclerosis risk factor.

Regular exercise can improve this inflammation state and entail fibrinogen reduction. IL-6 (that is synthesized in the muscle and adipocytes) is a strong inductor of fibrinogen synthesis in the liver [15]. This marker was not measure in this study, but several authors noted that regular exercise decrease IL-6 synthesis by endothelial cells, muscles and adipocytes [14], and reducing IL-6 leads to reduction of fibrinogen levels. Also long term exercise increase production of adiponectin, which is an anti-inflammatory adipocytokine. Elevation of this adipokine is associated with decrease insulin resistance and inflammatory state [14]. These changes afford reduction of fibrinogen levels. Another mechanism includes reduction of BFM [42] that was observed in this study. Excess body fat is associated to an inflammation state that stimulates synthesis of fibrinogen.

Results of this study showed significant reduction of uric acid after aerobic trainings. No study was found about the effect of long term exercise on serum uric acid levels in healthy and sedentary middle aged men. Also there is limited data about the association between physical activity and uric acid levels and results of such studies are inconsistence. Some authors have reported no associations and some have reported inverse relationships [45]. Tekin (2010) studied the effect of 6 weeks (5 days per week) aerobic and anaerobic exercise program on xanthine oxidase and uric acid in male athletes. Exercise program in each session included strength, velocity, aerobic endurance, flexibility and coordination trainings. Results of his study showed significant elevation of uric acid after 6 weeks exercise trainings [22]. He noted that strenuous exercise can damage body cells but moderate intensity exercise may produce antioxidant effect in the body [12]. He emphasized on the necessity of research about the effect of long term exercise on uric acid levels.

Mechanism of reduction of uric acid levels after exercise program in this study includes significant elevation of aerobic power that was seen in subjects of exercise group. Regular endurance exercise program increases mitochondrial and myoglobin density, induces angiogenesis in the muscles vessels, increases blood volume (this change was seen in this study) and then increases cardiac output. Regular aerobic exercise also increases gene expression of oxidative enzymes in the muscles and reduces reliance on the compounds of high phosphoryl transfer potential when the muscles are active due to physical activity [46]. These adaptations reduce AMP deaminase activity for production of ATP during exercise. Therefore less inosine, hypoxanthine, xanthine and uric acid are synthesized [47]. Thus levels of uric acid decreases after long term exercise and this change is in favor of heart health, because uric acid is a cardiovascular risk factor.

After trainings levels of WBC count, platelet count, lymphocyte count, monocyte count and eosinophil count increased significantly. Because the levels of these inflammatory markers were in normal ranges, this adaptation may be beneficial for immune function and anti-allergy processes and organism defense against parasites and infection [48]. In a longitudinal research, to examine the effect of continuous exercise on WBC count in males with essential
hypertension, Lamina and Okoye (2009) assigned 217 male patients into experimental and control groups and trained subjects in experimental group on a bicycle ergometer with intensity of 33 to 59 percent of heart rate maximum (8 weeks). They concluded that continuous bicycle training can lower levels of WBC count in male subjects with essential hypertension [26]. In a cross-sectional study, Geffken et al (2001) observed inverse relationship between physical activity and markers of inflammation (CRP, WBC count, albumin, factor VIII activity and fibrinogen) [49]. In another cross-sectional study, Wannamethee et al (2002) observed negative association between physical activity and inflammatory variables including factors VIII and IX, fibrin D-dimer, von Willebrand factor, tissue plasminogen activator, platelet count, WBC count, CRP, fibrinogen and blood viscosity [50]. Church et al (2002) showed significant relationship between URIC ACID, WBC count and fibrinogen levels with fatness (positive) and fitness (negative) [45]. Abramson and Vaccarino (2002) suggested that regular physical activity has significant inverse relationship with concentrations of WBC count, CRP and fibrinogen [51]. 

It seems that body fat percentage and socio-economic status are effective factors on changes in levels of WBC in response to long term aerobic exercise [26,45,49,50,52]. Regular exercise through change in secretion of TNF-α and IL-6 from adipose tissue affects number of leucocytes in the bloodstream [45]. There is conflicting data about the effects of long term exercise on TNF-α levels, thus it is possible that changes of TNF-α were effective in changes of immune cells in this study. 

In this study, in experimental group, levels of RDW raised significantly. It was not found any study examining the effect of long term aerobic exercise on this variable. More study is needed in this issue.

### Conclusion

In conclusion, in healthy and inactive middle aged men from 39 to 52 years, regular three months aerobic exercise through increment in cardio respiratory fitness and reduction of PBF, BMI and serum levels of homocysteine, hsCRP, uric acid and TG may lead to improvement in lifestyle and total health. More research is needed about mechanisms of involved in adaptation of novel and traditional cardiovascular risk factors to regular exercise in this population.

### References

5. Rudnicka AR, Rumley A, Lowe GDO, Strachan DP. Diurnal, seasonal, and blood processing patterns in levels of circulating fibrinogen, fibrin D-dimer, C-reactive protein, tissue plasminogen activator, and von willebrand factor in a


### Tables

**Table 1:** Descriptive characteristics of subjects and differences between groups before the trainings.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Exercise Group</th>
<th>Control Group</th>
<th>P-Value (independent T test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>44.73 ± 4.43</td>
<td>41.16 ± 8.03</td>
<td>0.25</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.73 ± 0.04</td>
<td>1.73 ± 0.07</td>
<td>0.84</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>81.84 ± 9.36</td>
<td>78.18 ± 14.81</td>
<td>0.24</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>27.43 ± 2.78</td>
<td>27.58 ± 3.22</td>
<td>0.78</td>
</tr>
<tr>
<td>PBF (%)</td>
<td>25.92 ± 4.91</td>
<td>28.38 ± 7.55</td>
<td>0.96</td>
</tr>
<tr>
<td>VFA (cm²)</td>
<td>124.33 ± 30.86</td>
<td>148.34 ± 47.87</td>
<td>0.98</td>
</tr>
</tbody>
</table>

**Table 2:** Levels of body composition variables before and after three months aerobic exercise.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Exercise Group</th>
<th>Control Group</th>
<th>P</th>
<th>Pre test</th>
<th>Post test</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (kg)</td>
<td>81.84 ± 9.36</td>
<td>78.18 ± 14.81</td>
<td>0.74</td>
<td>81.71 ± 9.57</td>
<td>78.5 ± 14.95</td>
<td>0.11</td>
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<tr>
<td>BMI (m/kg²)</td>
<td>27.43 ± 2.78</td>
<td>27.58 ± 3.22</td>
<td>0.007</td>
<td>26.79 ± 2.73</td>
<td>27.08 ± 3.86</td>
<td>0.46</td>
</tr>
<tr>
<td>PBF (%)</td>
<td>25.92 ± 4.91</td>
<td>28.38 ± 7.55</td>
<td>0.38</td>
<td>25.16 ± 3.66</td>
<td>27.68 ± 5.96</td>
<td>0.41</td>
</tr>
<tr>
<td>VFA (cm²)</td>
<td>124.33 ± 30.86</td>
<td>148.34 ± 47.87</td>
<td>0.95</td>
<td>124.59 ± 27.04</td>
<td>148.46 ± 48.51</td>
<td>0.94</td>
</tr>
<tr>
<td>WHR</td>
<td>0.93 ± 0.03</td>
<td>0.86 ± 0.11</td>
<td>0.7</td>
<td>0.93 ± 0.03</td>
<td>0.85 ± 0.11</td>
<td>0.51</td>
</tr>
<tr>
<td>Vo₂max *</td>
<td>24.91 ± 6.74</td>
<td>19.8 ± 1.8</td>
<td>0.002</td>
<td>31.73 ± 6.03</td>
<td>20 ± 1.87</td>
<td>0.62</td>
</tr>
</tbody>
</table>

**Table 3:** Significant changes of dependent variables.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Exercise Group</th>
<th>Control Group</th>
<th>P</th>
<th>Pre test</th>
<th>Post test</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Homocysteine (µmol/l)</td>
<td>7.85±0.67</td>
<td>6.51±2.36</td>
<td>0.00 3</td>
<td>6.1±2.03</td>
<td>0.08</td>
<td></td>
</tr>
<tr>
<td>hsCRP (mg/l)</td>
<td>1.89±0.65</td>
<td>1.83±1.06</td>
<td>0.00 9</td>
<td>1.6±0.7</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>TG (mg/dl)</td>
<td>169.73±45.78</td>
<td>165.2±47.77</td>
<td>0.04</td>
<td>159.7±48.83</td>
<td>0.38</td>
<td></td>
</tr>
<tr>
<td></td>
<td>158 ± 19.79</td>
<td>272.9 ± 29.3</td>
<td>0.03</td>
<td>254.9 ± 29.02</td>
<td>232.1 ± 30.71</td>
<td>0.01</td>
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<tr>
<td>TG/HDL</td>
<td>3.75 ± 1.06</td>
<td>3.11 ± 1.15</td>
<td>0.23</td>
<td>3.57 ± 1.1</td>
<td>3.43 ± 1.1</td>
<td>0.45</td>
</tr>
<tr>
<td>Fibrinogen (mg/dl)</td>
<td>285.45 ± 19.79</td>
<td>272.9 ± 29.3</td>
<td>0.23</td>
<td>254.9 ± 29.02</td>
<td>232.1 ± 30.71</td>
<td>0.01</td>
</tr>
<tr>
<td>Uric acid (mg/dl)</td>
<td>6.13 ± 0.59</td>
<td>5.5 ± 0.51</td>
<td>0.00</td>
<td>5.88 ± 1.03</td>
<td>5.64 ± 0.71</td>
<td>0.19</td>
</tr>
<tr>
<td>WBC count (Cu/mm)</td>
<td>5790.91 ± 1182.7</td>
<td>6388.13 ± 1255.7</td>
<td>0.00</td>
<td>6610 ± 1934.7</td>
<td>6690 ± 1813.0</td>
<td>0.33</td>
</tr>
<tr>
<td>RDW (%)</td>
<td>13.2 ± 0.56</td>
<td>13.79 ± 0.69</td>
<td>0.00</td>
<td>13.19 ± 0.99</td>
<td>13.04 ± 0.83</td>
<td>0.64</td>
</tr>
<tr>
<td>Plt count (Cu/mm)</td>
<td>240727.27 ± 344</td>
<td>254566.59 ± 324</td>
<td>0.01</td>
<td>245600 ± 5436</td>
<td>242300 ± 4357</td>
<td>0.82</td>
</tr>
</tbody>
</table>

58.