Comparing the fibrinogen, prothrombin time, Partial thromboplastin time and Platelets number and d-dimer in aerobic, control and resistance groups in Sari elderly sedentary men

1Mojtaba Mirsaiedi, 1zeynolabedin Fallah, 1Parvin Farzanegi and 2Mostafa Behzad Khameslu

1Department of Physical Education, Islamic Azad University, Sari, Iran
2Payam Noor University, Sari, Iran

ABSTRACT

Hypertensive patients are at high risk for the development of cardiovascular diseases. The aim of this study was to comparing the fibrinogen, prothrombin time, Partial thromboplastin time and Platelets number and d-dimer in aerobic, control and resistance groups. This study subjects included 36 apparently healthy elderly men. The protocol used in this study was reviewed and approved by Sari University’s Institutional Review Board prior to participant recruitment and all participants provided written informed consent prior to beginning the study. Blood samples were drawn after fasting overnight, after a resting period of 20 min. Fibrinogen was determined by the clotting assay of Clauss. The von Willebrand factor was quantities with an ELISA test kit of Boehringer Mannheim Corp. there are significant differences on fibrinogen, prothrombin time, Partial thromboplastin time and Platelets number and d-dimer between aerobic, control and resistance groups. We were therefore interested in possible interactions between the more resistant vessel wall and platelets and coagulation/fibrinolysis factors circulating in the blood.

Key words: fibrinogen, prothrombin time, Partial thromboplastin time and Platelets number and d-dimer.

INTRODUCTION

Hypertensive patients are at high risk for the development of cardiovascular diseases (1), whereas several studies have shown that treatment of hypertension diminishes the prevalence of cardiovascular diseases (2-5). The existence of hypertension, especially in combination with other risk factors, is disadvantageous for the prognosis of cardiovascular diseases (6). The integrity of the blood vessels is essential, because damage of the intima (which may occur in hypertension can finally cause atherosclerosis. Especially this kind of patient is likely to develop increased platelet aggregation with heart and blood vessel problems as possible sequelae (7). Moreover, blood vessel damage activates the coagulation System, which may also stimulate the progress of atherosclerosis.

Coagulation abnormalities in pregnant women have been reported to be more serious in women with hypertension (preeclampsia) than in those without hypertension (8). In patients with borderline hypertension, even before the appearance of clinical manifestations of vascular damages (9), coagulation activation seems to be already present. Besides platelet aggregability and coagulation activation, fibrinolysis, i.e. plasma tissue-type plasminogen activator activity, appears to be a major factor related to the risk of cardiovascular disease (10-15).
MATERIALS AND METHODS

This study subjects included 36 apparently healthy elderly men. The protocol used in this study was reviewed and approved by Sari University’s Institutional Review Board prior to participant recruitment and all participants provided written informed consent prior to beginning the study. As assessed by a medical history questionnaire, each participant was free of cardiovascular and neurological diseases, severe musculoskeletal injuries and low back pain. Firstly Subjects were tested between 8:00 and 10:00 h, according to the regular training. Participants attended having performed no vigorous exercise in the 24 h prior to testing and with diet standardized for 48 h proceeding in each test. Players were required to consume 500 mL of water 2 h prior to testing to ensure hydration. Thereafter the subjects consumed no fluid so as to control for the possible influence of hydration status on performance. Subjects participated in the test 30 min after having a standard breakfast. All subjects completed familiarization trials of the virtual task in the rested state on a minimum of previous laboratory visits.

For measuring fibrinogen, ptand ptt the Coagulation method and for d-dimer measurement the Elisa was used. The platelet number analyzed via Diatron abacus machine. The Stago machine used for measuring the fibrinogen, ptand ptt. The aerobic training group uses the ergometer for 30 min with 65% HR\textsubscript{max} for first 2 weeks (first 6 sessions) and 75% HR\textsubscript{max} for 35 min in second 2 week (last 6 sessions) that heart rate controlled with polar watch. The resistance training group performed the 6 movements (3 movements for upper extremity and 3 movements for lower extremity) including: chest press, lat pull, triceps with halter, squat, quadriceps and hamstrings in first 2 week (first 6 sessions) with 40% of 1-RM and second 2 week (last 6 sessions) with 60% of 1-RM that each of movements performed in 3 sets with 8 repetitions. Rest intervals between repetitions and sets were 1 to 1.5 minutes. Aerobic training group warmed up with 5 minutes biking on the ergometer and afterward performed his main program. For active recovery period, those biking 5 min on the ergometer and last 10 minutes includes the stretching movements.

Blood samples

Blood samples were drawn after fasting overnight, after a resting period of 20 min. Except for tissue plasminogen activator activity, all coagulation and fibrinolysis parameters were determined in citrated plasma. This was prepared by centrifugation of a mixture of nine volumes freshly drawn blood with one volume trisodium citrate (0.11 mol/1) for 30 min (1600g) at 25 °C. The plasma was stored at 70 °C in plastic tubes and thawed with tap water for 5 min before serial analysis. For the measurement of tissue plasminogen activator activity a separate tube was prepared with 0.5 ml acetate buffer (pH = 3.9) and filled with 1 ml citrated blood. The tube was centrifuged immediately (30 min, 1800#) and the plasma separated. The collected plasma was acidified with 20% acetic acid (final pH of the plasma sample 4.0-4.1), then stored at -70 °C in plastic tubes; it was thawed with tap water for 5 min before analysis.

Fibrinogen was determined by the clotting assay of Clauss. The von Willebrand factor was quantities with an ELISA test kit of Boehringer Mannheim Corp (Mannheim, FRG). For the factor VIII:c determination an activated partial thrombin time (aPTT) on stage clotting assay was performed, using a deficient plasma from Behring (Marburg, FRG). Fibrin monomer concentrations were assessed with the chromogenic COA-Set FM-test of KabiVitrumDiagnostica. Thrombinantithrombin III was determined with an ELISA kit of the Behring Corporation (Marburg, FRG). D-Dimer was assayed in plasma with an ELISA method of Boehringer MannheimCorp. (Mannheim, FRG). For plasminogen activator inhibitor the test kit of KabiVitrumDiagnostica COA-Set PAI was used.

The tissue-plasminogen activator activity test was also from KabiVitrumDiagnostica as well as the antigenic tissue plasminogen activator test (KabiVitrunmtagnostica, CoazlatPA test). The platelet aggregation tests were performed on platelet-rich plasma which was prepared by immediate centrifugation of citrated blood (prepared as described above) at 200 g for 10 minutes at room temperature. After gentle aspiration ofthe platelet-rich plasma, using a plastic pipette, the ferriainingblood was centrifuged at 2000 £ for 10 min at room temperature, the resulting platelet-poor plasma was aspirated and subsequently centrifuged at 10000g at 4°C for 10 min to obtain platelet-free plasma. Platelet-rich plasma and platelet-free plasma were used for standardization of the end concentration of the platelet count in the test.

Statistical analysis

The one way ANOVA and LSD POSTHOC an alpha level of (0.05) was used in determining statistical significance using the SPSS program for Windows, version 18.0.
RESULTS

The one way ANOVA test results for fibrinogen, prothrombin time, Partial thromboplastin time and Platelets number in aerobic, control and resistance groups were shown in table 1. Results of one way ANOVA test for differences in d-dimer variable in posttest between 3 groups (aerobic, control and resistance) are shown in table 2.

Table1. The one way ANOVA test results for fibrinogen, prothrombin time, Partial thromboplastin time and Platelets number in aerobic, control and resistance groups.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Groups</th>
<th>Statistics Stages</th>
<th>Mean differences</th>
<th>SE</th>
<th>P</th>
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<tbody>
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<td>Control</td>
<td>Pre test- post test</td>
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<td>18.4</td>
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<td>prothrombin time</td>
<td>Control</td>
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<td>-0.43</td>
<td>0.51</td>
<td>0.349</td>
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<tr>
<td>Partial thromboplastin time</td>
<td>Control</td>
<td></td>
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<td>Platelets number</td>
<td>Control</td>
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<td>1.05</td>
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<td>-24.39</td>
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<td>0.697</td>
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</table>

Table2. Results of one way ANOVA test for differences in d-dimer variable in posttest between 3 groups (aerobic, control and resistance)

<table>
<thead>
<tr>
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<td>0.629</td>
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DISCUSSION

Recently Panza et al. (20) concluded in their study on abnormal endothelium-dependent vascular relaxation in patients with essential hypertension, that endothelium-mediated vasodilation is impaired in patients with essential hypertension. This defect might play an important role in the functional abnormality of elevated vascular resistance, which is observed in hypertensive patients. Under regular circumstances the patency of the blood vessels and the fluidity of the blood is maintained by the endothelial cells. For this purpose the endothelial cells synthesize a number of active substances like fibronectin, heparansulfate, inter-leukin-1, tissue plasminogen activator, plasminogen activator inhibitor, prostacyclin, nitric oxide, platelet-activating factor and endothelin-1 (21, 22). Moreover, the von Willebrand factor is known to be synthesized by endothelial cells, which is important for the platelet-vessel wall interaction (18). Recently Struyker Boudier et al. (23) summarized the three mechanisms thought to be responsible for the overall vascular resistance increase in hypertension: the rarefaction of arterioles and capillaries, the decreased internal diameter of the arterioles and the increase of the arterial and arteriolar wall mass. We were therefore interested in possible interactions between more resistant vessel wall and platelets and coagulation/fibrinolysis factors circulating in the blood.

In a considerable number of the patients (35.7%) we found an enhanced in vitro reactivity towards ADP (2 μmol/O-muKXκα aggregation, which is in agreement with earlier findings of Yamanishi et al. (24) in patients with different stages of essential hypertension. One could speculate that this phenomenon is a sequel of the diminished prostacyclin synthesis and/or release by the endothelial cells. Isles et al. (25) and Gavras et al. (26) showed that malignant hypertension was associated with increased mean levels of fibrinogen, factor VIII-c, decreased urokinase, increased fibrinogen degradation products and decreased platelet count. In our patients with relatively mild hypertension we can confirm the significant elevation of the fibrinogen level, but not that of factor VIII-c. We demonstrated a significant decrease of tissue plasminogen activator activity, but a significant increase of tissue plasminogen activator antigen and the level of plasminogen activator inhibitor level.

REFERENCES