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

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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-dimerin 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 themore 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 dehydration. 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_{max} for first 2 weeks (first 6 sessions) and 75% HR_{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 weredetermined 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 themeasurement of tissue plasminogen activator activity a separate tube was prepared with 0.5 ml acetate buffer (pH= 3.9) and filled with l ml citrated blood. The tube was centrifuged immediately (30 min, 1800#) and the plasma separated. Thecollected plasma was acidified with 20% acetic acid (final pHof the plasma sample 4.0-4.1), then stored at -70 °C in plastictubes; 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 inhibitorthe lest kit of KabiVitrumDiagnostiea COA-Set PAI was used.

The tissue-plasminogen activator activity test was also fromKabiVitrumDiagnostica as well as the antigenic tissue plasminogen activator test (KabiVitrumttiagnostica, CoalizatPA test). The piatelet aggregation tests were performed onplatelet-rich plasma which was prepared by immediate centrifugation of citrated blood (prepared as described above) at 200 gfor 10 minutes at room temperature. After gentle aspiration ofthe platelet-rich-plasma, using a plastic pipette, the feriiainingblood 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 obtainplatelet-free plasma. Platelet-rich plasma and platelet-free plasma were used for standardization of the end concentration of the piatelet 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 numberin 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 numberin aerobic, control and resistance groups.

Variables	Groups		Statistics Stages	Mean differences	SE	P
Fibrinogen	Resistance	Control	Pre test- post test	-65	34.3	0.032●
		Aerobic		18.4	32.5	0.417
prothrombin time		Control		-1.32	0.52	0.02●
		Aerobic		-0.43	0.51	0.349
Parthialthromboplastin time		Control		-0.65	4	0.014●
		Aerobic		-1.92	4	0.126
Platelets number		Control		-2320	1.05	0.013●
		Aerobic		-2439	1.07	0.697

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

Variables	**Groups**		**Mean differences**	**SE**	**P**
d-dimer	Resistance	Control	790.7	290.5	0.001●
		Aerobic	189.9	290.7	0.629

DISCUSSION

Recently Panza et al. (20) concluded in their study onabnormal endothelium-dependent vascular relaxationin patients with essential hypertension, that endotheliummediated vasodilation is impaired in patientswith essential hypertension. This defect might play animportant 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 bloodis maintained by the endothelial cells. For this purposethe endothelial cells synthesize a number of active substances like fibronectin, heparansulphate, inter-leukin-1, tissue plasminogen activator, plasminogenactivator inhibitor, prostacyclin, nitric oxide, platelet-activating factor and endothelin-1 (21, 22).Moreover the von Witt brand factor is known to besynthesized by endothelial cells, which is importantfor the platelet-vessel wall interaction (18). RecentlyStruykerBoudier et al. (23) summarized the threemechanisms thought to be responsible for the overallvascular resistance increase in hypertension: the rarefaction of arterioles and capillaries, the decreasedinternal diameter of the arterioles and the increase ofthe arterial and arteriolar wall mass. We were therefore interested in possible interactions between themore resistant vessel wall and platelets and coagulation/fibrinolysis factors circulating in the blood.

In a considerable number of the patients (35.7%) wefound an enhanced in vitro reactivity towards ADP(2 μηοI/O-íικΚκκα aggregation, which is in agreement with earlier fmdings of Yamanishi et al. (24) in patientswith different stages of essential hypertension. One could speculate that this phenomenon is a sequal ofthe diminished prostacyclin synthesis and/or releaseby the endothelial cells. Isles et al. (25) and Gavras etal. (26) showed that malignant hypertension was associated with increased mean levels of fibrinogen,factor VIII:c, decreased urokinase, increased fibrin(ogen) degradation products and decreased platelet count. In our patients with relatively mild hypertension we can confirm the significant elevation ofthe fibrinogen level, but not that of factor VIILc. Wedemonstrated a significant decrease of tissue plasminogen activator activity, but a significant increase oftissue plasminogen activator antigen and the level ofplasminogen activator inhibitor level.

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