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Case Report

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The Influence of Acute Morning and Evening Exercise on Homocysteine, Fibrinogen and Platelet

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Abstract

Background: There are accumulated evidences that plasma concentration of homocysteine (Hcy), platelet (plt) and fibrinogen (Fib) is a prognostic factor for cardiovascular morbidity and mortality.

The aim of the present study was to investigate the influence of a bout of incremental exercise during morning and evening on hcy, fib, and plt.

Methods: 15 healthy trained women (age: 18-25 years, BMI: 21 ± 2.35) participated in the study voluntarily and performed maximal incremental Bruce exercise test in morning (8 am) and evening (10 pm) during two separate days. Blood samples were taken before and after every exercise test session. Statistical method of repeated measures was used for analysis of data.

Results: Finding of study indicated a significant increase in Hcy after the exercise in the evening compared to morning (P=0.015). There were no significant difference in fib and plt during morning and evening before and after exercise and hcy before exercise during morning and evening (P>0.05).

Conclusion: According to hcy increase after evening compared to morning exercise we can recommend evening as more proper time for exercise than morning.

Keywords: Morning exercise; Evening exercise; Homocysteine; Fibrinogen; Platelet

Background

Although physical exercise is recommended for prevention of cardiac disease ,there are discrepancies regarding the safety or priority of morning or evening exercise in this regard [1]. Some studies indicate that acute ischemic events show a circadian rhythm which correlate with increased activity of the sympathetic nervous system and increased platelet aggregation during early morning [2,3]. Elevation of plasma catecholamine following sympathetic nervous system activation can result in increased platelet aggregation (possibly by activating platelet α_2-adrenergic receptors) [4]. Some studies revealed that acute vigorous physical activity or exercise may trigger coronary syndrome and induce thrombogenesis [5]. So interaction of circadian rhythm and exercise in cardiac events is very important. Abnormalities in endogenous coagulation and fibrinolysis may contribute in increasing the risk of an acute cardiovascular event [6].

Hcy is a non-essential sulfur-containing amino acid methionine [7]. Hcy impairs nitric oxide production and is implicated in the generation of oxidized species leading to endothelial dysfunction and platelet activation [8,9]. It has been implicated as a significant contributor in mortality [10,11]. Since elevated Hcy levels can frequently be found in elderly persons, its measurement is generally recommended to this age group [12]. While, recent studies demonstrated that high Hcy can also be observed in young people [13,14]. More recently, plasma Hcy levels were also shown to have circadian rhythm in humans with an evening peak and a morning fall [3]. Little information is available on the effect of exercise on Hcy. Although a few studies on endurance trained men and untrained young women have shown that a single bout of intensive exercise acutely increased Hcy [15].

Fib represents a major step in platelets aggregations [16]. Fib is also the main substrate for coagulation cascade and forms a polymerized fibrin clot. Platelet-dependent thromboembolism is an important mechanism of arterial thrombosis and the basic role of platelets in thrombosis is now highly accepted [17]. Its role in the arterial thrombosis is focused on adhesive properties and ability to respond to stimuli with quick activation and finally aggregation [18]. Some researchers have indicated increasing of fib and decreasing of plt following acute single boat of high intensity exercise [19-21]. While acrophases of fib and plt in circadian rhythm has been reported in afternoon and evening no study was found related to influence of exercise during morning and evening on fib, plt and hcy [22]. So the purpose of present study was evaluating influence of acute morning and evening incremental exercise on fib, plt and homocystein.

Objectives

Because the number of studies related to circadian rhythm of homocystein were, however, too small to provide conclusive evidence for effect of acute exercise on Hcy. So the aims of this study were to investigate the influence of acute incremental exercise during morning and evening on Hcy, Fib, and plt. We also investigated if the time (i.e., morning or evening) had a different effect on the resultant plasma Hcy, fib and plt concentration during rest or as a result of exercise.

Patients and Methods

Subjects of study included 15 healthy trained women (age: 18-25 years, BMI: 21 ± 2.35) who participated in this study voluntarily. They were qualified for general and cardiovascular health status by a physician and signed informed consent.

Subjects performed maximal incremental Bruce test on treadmill during morning (8- 8:30 a. m) and evening (9-9:30 p.m) during 2 separate days (48 hours as rest). Venous blood samples were taken before and immediately after every exercise sessions. Blood specimens were kept on ice for 20 min. After clotting, serum was separated immediately from the blood cells by centrifugation, transported on ice to the laboratory and then stored at -20°C until analysis. Hcy was

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assessed by fluorescence detection (immunodiagnostic, Blenheim, Germany).

Fibrinogen was assessed by the time titration method using the ST-4 coagulation instrument (Zymutest Fibrinogen, ELISA, Hyphen Biomed, Neuville sur Oise, France). Platelets were counted by Act-Diff-Coulter Electronics, Krefeld, Germany.

Spss (version 16) was used for data analysis. Statistical method of repeated measure was used to compare variables during morning and evening, before and after exercise sessions.

Results

Findings of study revealed a significant increase in serum Hcy after the exercise in the evening compared to morning (P=0.015). Hcy concentration increased after every exercise sessions in the morning and evening (Table 1), although this increase was statistically significant in the evening (P= 0.05) but not in the morning (P>0.05). .

Platelet increased after every exercise sessions and in the evening compared to morning (Table 1), but these differences were not significant statistically (P>0.05). Fibrinogen fluctuations during morning and evening and after every exercise sessions were not statically significant (P>0.05).

Discussion

Findings of study indicated that hct increased significantly after exercise in the evening compared to exercise in the morning. Although, hct increases after morning or evening exercise compared to before exercise were not statistically significant.

Available literature indicates that exercise intensity can be important effective factor on hct fluctuations. König et al. (2003) indicated that intense acute exercise increased plasma Hcy level in young healthy men [23]. Wright et al. (1998) indicated that moderate average intensity exercises will not probably increase plasma Hcy level [24]. Hermann et al. (2003) found that endurance exercise can cause a significant increase in plasma Hcy which fluctuate as a result of different duration and intensity of exercise [15]. In the present study, subjects performed Bruce exercise testing until exhaustion, but, probably, its duration was not enough to induce significant hcy increase after exercise. However, hcy increase after evening exercise was significantly higher than morning exercise, although there was no significant difference between pre exercise morning and evening hcy. Lavie and Lavie (2004) reported that there was a daily rhythm in plasma concentration of Hcy which was independent of sleep-wake and food consumption [25]. Hcy peak was reported around 22.00 to 24.00. but because the most usual training time was at evening about 19.00 to 21.00 , blood samples were taken at about 21.00 (± half an hour) and this may resulted in no significant difference in hcy between morning and evening.

Table 1: Homocystein , Fibrinogen and Platelet before and after exercise during morning and evening.

Variable			Mean ± SD
Homocystein (µm/lit)	Morning	Pre exercise	10.09 ± 4.30
		Post exercise	11.60 ± 3.19
	Evening	Pre exercise	11.45 ±7.33
		Post exercise	15.93 ± 4.79* ©
Platelet (number/1000µl)	Morning	Pre exercise	176.50 ±54.35
		Post exercise	179.90 ± 50.08
	Evening	Pre exercise	177.00 ±53.09
		Post exercise	182.70 ± 51.76
Fibrinogen (mg/dL)	Morning	Pre exercise	228.00 ±49.60
		Post exercise	260.90± 62.20
	Evening	Pre exercise	228.80 ±42.92
		Post exercise	234.20± 94.54

*significant difference between morning and evening fluctuations
©significant difference between pre and post exercise

The metabolism of Hcy is performed by two major pathways, remethylation into methionine and transsulfuration to cystationine [26]. In remethylation process, Hcy gains a methyl group from N-5-methyltetrahydrofolate or from betaine and make methionine. In the transsulforation pathway, Hcy condenses with serine and make cystationine in an irreversible reaction catalyzed by the pyridoxal-5'-phos phate (PLP)-containing enzyme, cystationine beta syntheses. The underlying mechanism responsible for the daily rhythm in plasma Hcy is not clear yet and most probably, it is related to the balance between its rates of production and clearance. A high Hcy concentration could be due to a high rate of production, a decreased rate of trans sulforation, remethylation to methionine, or combination of these processes Lavie and Lavie [25]. However, according to findings of present study we can conclude that interaction of exercise and time of day effects probably have elevated production of hcy compared to its elimination.

The catecholamines including epinephrine and norepinephrine have indicated circadian rhythm and are potent vasoactive hormones that contribute to vasoconstriction, endothelial function and platelet activation [27] .

According to findings of present study platelet and fibrinogen did not change significantly after incremental exercise to exhaustion during morning and evening. Some studies confirm our findings and indicate no significant influence of exercise on fibrinogen [28] and platelet count [29,30], but some studies indicate increasing of fibrinogen [31,32] and increasing of platelet count [33-37] after acute exercise. Differences in exercise intensity, mode, duration and day time of exercise, fitness level, sex and health condition of subjects may have caused controversies in research findings. Lippi et al (2008) found that baseline cortisol levels in the morning might predict higher fibrinogen in the general population [38]. Cortisol increasing as a result of exercise may conflict with its circadian rhythm and have affected our findings; Small number of subjects is a limitation of this study and may affected our findings. So, further studies are required for more clarification.

In general, according to findings of his study, evening post exercise increased Hcy concentrations which may be associated with increased cardiovascular risks of sport participation during evening. However more research is required for better clarification. These findings may have implications for the health of sport men/ women.

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References

1. Atkinson G, Drust B, George K, Reilly T, Waterhouse J (2006) Chronobiological considerations for exercise and heart disease. Sports Med 36: 487-500.
2. Tofler GH, Brezinski DA, Schafer AI, Czeisler CA, Rutherford JD, et al. (1987) Concurrent morning increase in platelet aggregability and the risk of myocardial infarction and sudden death. N Engl J Med 316: 1514-1518.

3. Atkinson G, Jones H, Ainslie PN (2010) Circadian variation in the circulatory responses to exercise: relevance to the morning peaks in strokes and cardiac events. Eur J Appl Physiol 108: 15-29.

4. Hjemdahl P, Larsson PT, Wallén NH (1991) Effects of stress and β-blockade on platelet function. Circulation 84: VI44-161.

5. Thrall G, lane D, Caroll D, Lip GY (2007) A systematic review of the effects of acute psychological stress and physical activity on haemorheology, coagulation, fibrinolysis and platelet reactivity: Implications for the pathogenesis of acute coronary syndromes. Thromb Res 120: 819–847.

6. Kamath S, Blann AD, Lip GY (2001) Platelet activation: assessment and quantification. Eur Heart J 22: 1561–1571.

7. Ganji V, Kafai MR (2003) Demograph, health, lifestyle, and blood vitamin determinants of serum total homocysteine concentrations in the third National Health and Nutrition Examination Survey. Am J Clin Nutr 77: 826-833.

8. Welch GN, Loscalzo J (1998) Homocysteine and atherothrommbosis. N Engl J Med 338: 1042-1050.

9. Van Guldener C, Stehhouwer CD (2000) Hyperhomocysteinemia, vascular pathology, and endothelial dysfunction. Semin Thromb Hemost 26: 281-289.

10. Nygard O, Nordrehaug JE, Refsum H, Ueland PM, Farstad M, et al. (1997) Plasma homocysteine levels and mortality in patient with coronary artery disease. N Engl J Med 337: 230-236.

11. Anderson JL, Muhlestein JB, Horne BD, Carlquist JF, Bair TL, et al. (2000) Plasma homocysteine predicts mortality independently of traditional risk factors and C-reactive protein in patients with angiographically defined coronary artery disease. Circulation 102: 1227-1232.

12. Herrmann W, Quast S, Ullrich M, Schultze H, Bodis M, et al. (1999) Hyperhomocysteinemia in high aged subjects: relation of B-vitamins, folic acid, renal function and the ethylenetetrahydrofolate reductase mutation. Atherosclerosis 144: 91-101.

13. Nelen WL (2001) Hyperhomocysteinaemia and human reproduction. Clin Chem Lab Med 39: 758-763.

14. Herrmann W, Schorr H, Purschwitz K, Rassoul F, Richter V (2001) Total homocysteine, vitamin B(12), and total antioxidant status in vegeterains. Clin Chem 47: 1094-1101.

15. Herrmann M, Wilkinson J, Schorr H, Obeid R, Georg T (2003) Comparison of the influence of volume-oriented training and high-intensity interval training on serum homocysteine and its cofactors in young, healthy swimmers. Clin Chem Lab Med 41: 1525-1531.

16. Bombeli T, Schwartz BR, Harlan JM (1998) Adhesion of activated platelets to endothelial cells: evidence for a GPIIbIIIa-dependent bridging mechanism and novel roles for endothelial intercellular adhesion molecule I(ICAM-1), alphavbet3 integrin, and GPIbalpha. J Exp Med 187: 329-339.

17. Viles-Gonzalez JF, Fuster V, Badimon JJ (2004) Atherothrombosis: a widespread disease with unpredictable and life-threatening consequences. Eur Heart J 25: 1197-1207.

18. Ruggeri ZM (2002) Platelets in atherothrombosis. Nat Med 8: 1227-1234.

19. Gibbs CR, Blann AD, Edmunds E, Watson RD, Lip GY (2001) Effects of acute exercise on hemorheological, endothelial, and platelet markers in patients with chronic heart failure in sinus rhythm. Clin Cardiol 24: 724–729.

20. Li-Saw-Hee FL, Blann AD, Edmunds E, Gibbs CR, Lip GY (2001) Effect of acute exercise on the raised plasma fibrinogen, soluble P-selectin and von Willebrand factor levels in chronic atrial fibrillation. Clin Cardiol 24: 409–414.

21. van den Burg PJ, Hospers JE, van Vliet M, Mosterd WL, Bouma BN, et al. (1995) Changes in haemostatic factors and activation products after exercise in healthy subjects with different ages. Thromb Haemost 74: 1457–1464.

22. Berger J (2004) Chronohaematology. J appl Biomed 2: 179-185.

23. König D, Bisse E, Deibert P, Muller HM, Wieland H, et al. (2003) Influence of training volume and acute physical exercise on the homocysteine levels in endurance-trained men: interactions with plasma folate and vitamin B12. Ann Nutr Metab 47: 114-118.

24. Wright M, Francis K, Cornwell P (1998) Effect of acute exercise on plasma homocysteine. J Sports Med Phys Fitness 38: 262-265.

25. Lavie L, Lavie P (2004) Daily rhythms in plasma levels of homocysteine. J Circadian Rhythms 2: 5

26. Selhub J (1999) Homocysteine metabolism. Annu Rev Nutr 19: 217-246.

27. Yang S, Zhang L (2004) Glucocorticoids and vascular reactivity. Curr Vasc Pharmacol 2: 1–12.

28. Speiser W, Langer W, Pschaick A, Selmayr E, Ibe B, et al. (1988) Increased blood fibrinolytic activity after physical exercise: comparative study in individuals with different sporting activities and in patients after myocardial infarction taking part in a rehabilitation sports program. Thromb Res 51: 543–555.

29. Martos E, Pucsok J, Malomsoki J, Ekes E, Hoffmann A (1988) Platelet aggregation, lipids and excretion of catecholamines after acute physical exercise in patients with myocardial infarction. Acta Physiol Hung 71: 175–182.

30. McGill D, McGuiness J, Lloyd J, Ardlie N (1989) Platelet function and exercise-induced myocardial ischaemia in coronary heart disease patients. Thromb Res 56: 147–158.

31. Li-Saw-Hee FL, Blann AD, Edmunds E, Gibbs CR, Lip GY (2001) Effect of acute exercise on the raised plasma fibrinogen,soluble P-selectin and von Willebrand factor levels in chronic atrial fibrillation. Clin Cardiol 24: 409–414.

32. Gibbs CR, Blann AD, Edmunds E, Watson RD, Lip GY (2001) Effects of acute exercise on hemorheological, endothelial, and platelet markers in patients with chronic heart failure in sinus rhythm. Clin Cardiol 24: 724–729.

33. Mustonen P, Lepantalo M, Lassila R (1998) Physical exertion induces thrombin formation and fibrin degradation in patients with peripheral atherosclerosis. Arterioscler Thromb Vasc Biol 18: 244–249.

34. van den Burg PJ, Hospers JE, van Vliet M, Mosterd WL, Bouma BN, et al. (1995) Changes in haemostatic factors and activation products after exercise in healthy subjects with different ages. Thromb Haemost 74: 1457–1464.

35. Naesh O, Hindberg I, Trap-Jensen J, Lund JO (1990) Post-exercise platelet activation-aggregation and release in relation to dynamic exercise. Clin Physiol 10: 221–230.

36. Ersoz G, Zergeroglu AM, Yakaryilmaz A (2002) The effect of submaximal exercise on platelet aggregation during late follicular and midluteal phases in women. Thromb Res 108: 147–150.

37. Gunga HC, Kirsch K, Beneke R, Boning D, Hopfenmuller W, et al. (2002) Markers of coagulation, fibrinolysis and angiogenesis after strenuous short-term exercise (Wingate-test) in male subjects of varying fitness levels. Int J Sports Med 23: 495–499.

38. Lippi G, Franchini M, Salvagno GL, Montagnana M, Guidi GC (2008) Higher morning serum cortisol level predicts increased fibrinogen but not shortened APTT. J Thromb Thrombolysis 26: 103–105.

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