Journal of Practical Business Law

J. Practical Buss. Law, 09: 47-51, 2022

Research Paper

The Effect of Endurance Exercise on High-Sensitivity C - ReactiveProtein in Serum of Inactive Allergic Asthma Participants

Daniel Tarmast (PhD)
Assistant Professor, Department of Physical Education and Sport Sciences, Faculty of Humanities, Parand Branch, Islamic Azad University, Parand city, Tehran, Iran

Abstract

The aim of this study was to evaluate the effects of endurance training on serum CRP (C-reactive protein) levels in adult asthma patients. For this purpose, pre and post aerobic training (3 times-weekly/ 3 months) blood samples were taken after overnight fast in 32 adult men with asthma patients that wasrandomly divided to exercise and control groups. Anthropometrical indexes were also monitored in two occasions. All data changes were compared by *t* - tests. At baseline, there were no differences in the serum CRP, body weight and other anthropometrical indexes between the two groups. Compared to pre-training, the CRP levels decreased significantly after aerobic training in the exercise group but not in the control subjects. We observed a significant decrease in all anthropometrical indexes in exercise group. All paper remained without any changes in the control group. Thus, we conclude that the endurance exercise activity improves systemic inflammation in asthma patients.

***Keywords*:** Asthma Patients, Endurance Exercise, High-Sensitivity C-Reactive Protein.

Background and purpose

To date, the current management of asthma focuses on the optimal control of airway inflammation as a central component of asthma control. Asthma is a disorder characterized by inflammation of the airways and inflammation cytokines plays an important role in this disease (12). Accumulating evidence indicates that asthma is a complex syndrome with many clinical phenotypes. Its major characteristics include a variable degree of airflow obstruction, bronchial hyper-responsiveness, and chronic airway inflammation. There is considerableevidence that circulating CRP has emerged as one of the most powerful independent predictors of cardiovascular disease risk and cardiovascular death (34). It has been long known that C-reactive protein (CRP) is a major inflammation sensitive plasma protein in humans, and its synthesis by the liver is regulated to a large extent by the pro-inflammatory cytokine interleukin (IL)-6. It was reported that after adjustment for age, smoking status, diabetes,blood pressure and the use of hormone replacement therapy, the relative risk in top CRP is more than anthers cardiovascular risk factors such as LDL (30). A large body of evidence suggests that Systemic inflammation is a possible element in the link between respiratory impairment and cardiovascular events. It was alsoreported that reduced lung function has been associated with various inflammation sensitive plasma proteins (24,25). In this area, some researchers suggests that systemic inflammation, as measured by using C-reactive protein (CRP) levels, might be important in the relationship between obesity and asthma (2,26). Several studies have found a high inverse relation between CRP and function such as forced expiratory volume in one second (FEV1) (31). Some studies have indicated a positive correlation between asthma and increased CRP levels (7,9,15,29). There are few longitudinal studies that physical activity or aerobic training decreases systemic inflammation in obesity or chronic diseases (4). But, there is limited literature on CRP responses to exercise training in asthma patients. Therefore, the present study aims to evaluate the significance of a long term aerobic exercise on serum CRP in adult men with asthma.

Materials and methods

The study was approved by the Ethics Committees of Islamic Azad University, Parand Branch. The objective of this semi-experimental study was to evaluate serum CRP response to aerobic exercise program in group asthma patients. For this purpose, thirty two adult asthmatic men (39±5 years, BMI = 29.06±3 Kg/m^2) participated in this study by

accessible sampling and divided to exercise (3 days for 12 weeks) and control (no exercise) groups by randomly. Spirometry tests (Minispire model, Made in Italy) was performed in order to asthma diagnosis as well as to determine the asthma severity. FEV1 and forced expiratory volume in 1 s / forced vital capacity (FEV1/FVC) were measured. All patients were asked to avoid having tea or coffee as well as other airways dilator food for at least 4 hours prior to spirometry test. Before signing a consent form, all participants received written and verbal explanations about the nature of the study.

All subjects were inactive, and none reported engaging in systemic (more than one time per week) sport activities before the study. Inclusion criteria for study group were determined as existing asthma for at least 2 years. Exclusion criteria included medicationsthat alter carbohydrate metabolism, diabetes, inability to exercise, and history of hypertension or heart disease (18-20). All participants of both groups completed the anthropometrical measurements, and blood samples before and after the exercise protocol. Measurements of height (m) and weight (kg) were performed with the barefoot and dressed in shorts and shirt. With these measures, the body mass index (BMI = weight/height^2) was calculated. Visceral fat, and body fat percentage was determined using body composition monitor (OMRON, Finland). The subjects were advised to avoid any physical activity 48 hours before the blood sampling. Blood samples (5 ml) were taken between 8:00 and 9:00 a.m. after 10 to 12 hours overnightfast to measure serum CRP from brachial vein in sitting position. Serums were immediately separated and stored at – 80 °C until the assays were performed. In fact, fasting blood samples were taken pre-training (pre-test) and 48 h after the training (post- test).

Serum CRP was determined by ELISA method (Diagnostics Biochem Canada Inc. HsCRP). Exercise training program was performed at 60 to 80 percent of maximum heart rate of the participants. Aerobic exercises included walking or running, and stationary cycling. The intensity of the activity of the participants was controlled by the heart rate monitor (Polar). All participants in the control group were barred from contributing in any other exercise. Finally, all measurements consist of fasting blood sampling; anthropometric measurements repeated 48 h after the last exercise trial.

The Kolmogorov-Smirnov test was used to determine the variables with normal distribution. Independent sample t test used to observe group mean difference at baseline. Pre- to post training changes were determined by two-tailed *t*-tests, and the level of significance was set at $p< 0.05$. All data were analyzed using the Statistical Package for Social Sciences (IBM SPSS Statistics version 22.0).

Results

Baseline and post training CRP levels, and anthropometrical indexes of two groups are presented in Table 1. Thespirometric test showed that all patients in mild to moderate asthma severity (FEV1 = 74 ± 6.5 (%), FEV1 / FVC = 67.3 ± 4.2). The finding by independent *t* - test showed no differences in the age, spirometry markers, body weight and other anthropometrical indexes and serum CRP between the two groups ($p\leq0.05$).

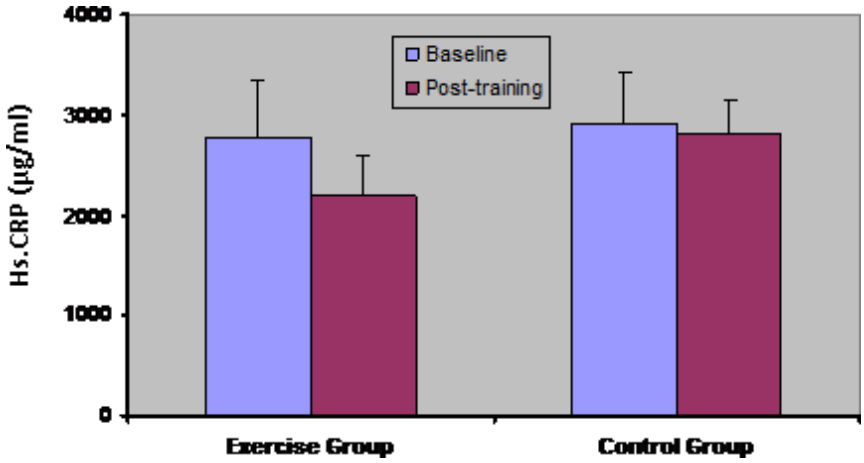

Fig. 1. The changes pattern of serum Hs.CRP in baseline and by interventions in two groups

Compared to pre-training, serum CRP significantlyreduced (p = 0.011) after exercise training, but not in the control groups (Fig. 1). This finding indicates the positive the role of endurance exercise training on CRP and inflammatory cytokine. Anthropometrics variables improved significantly after the therapy in exercise group. Body mass index levels were significantly decreased in response to exercise program when compared with baseline levels (p = 0.021). In addition, we found that endurance exercise reduced body fat percentage, body weight and visceral fat in exercise group (p> 0.05).

Discussion

Our study showed that aerobic exercise training for three months decreases serum CRP in adult men with asthma patients. In present study, we also observed that a reduction in anthropometric variables after aerobic training in exercise group. Adipose tissue secretes a variety of bioactive mediators including adipocytokines such as adiponectin, leptin, resistin or classical cytokines such as the pro-inflammatory mediators TNF-α and interleukin 6 (IL-6) (13,35). On the other hand, reported studies have found an inverse relationship between lung function and markers of systemic inflammation (1). So that, impaired respiratory function such as FEV1 is strongly related with cardiovascular risk factors, atherosclerosis, arterial stiffness, cardiovascular disease and mortality, although themechanisms underlying this response are a matter of some debate. These changes are associated with disturbance in immune response in the lung. High sensitivity C-reactive protein is an inflammatory mediator known to be related to inflammation, and cardiovascular diseases (8). On theother hand, a positive relationship has been reported between raised CRP levels and current asthma (10,16), respiratory impairment (4,11), and bronchial responsiveness (26,27) intervention.A recent study has been reported positive association between raised hs-CRP levels, current asthma, respiratory impairment and bronchial hyper-reactivity (24,16). The relation between asthma and CRP is by no means clear, however, and could at least in part reflect the role of obesity in CRP production (31). Some recent study showed that CRP level in stable asthma is significantly higher compared to those without asthma state. These researchers support the hypothesis that not only local but also systemic inflammation exists in bronchial asthma. To support this data, Jousilhti et al (15-17) demonstrated that asthma increased gradually with increasing CRP. The finding of another showed an inverse linear relationship between CRP concentrations and measures of pulmonary function in subjects without pulmonary disease and in never-smokers (5). It was reported that exercise training for long time is associated with low systemic inflammation in chronic diseases (13,33), although the molecular mechanisms for this are less understood.

In current study, we observed a significant reduction inserum CRP by three months aerobic exercise program in asthma patients. On the other hand, decreased serum CRP was accompanied a reduction in all anthropometrical markers such as body weight, BMI, body fat percentage and visceral fat. Since most previous studies have emphasized the fact that amplified adipose tissues especially abdominal obesity is associated with increased inflammatory cytokine, it appears reduced body weight along with decreased body in the subjects caused by exerciseis one of the main reasons of decline in serum levels of CRP. In fact, it appears that reduced levels of body fatthrough exercise may be attributed to reduced secretion of this inflammatory cytokine from adiposetissue into the bloodstream. In this context, some other studies on obese subjects or subjects other chronic diseases such as diabetesor cardiovascular diseases have also reported that once physical activity is associated with a significant reduction in body fat levels it leads to the decrease of inflammatory cytokines, such as IL-6 - TNF-α and/or the rise of anti- inflammatory cytokine (30). Despite these findings, failure to measure other cytokines is one of themain limitations of this study which calls for evaluation in future studies (21-23).

Conclusion

The CRP levels declined subsequent to the endurance activity in the exercise group but. Also the anthropometrical indexes in exercise group were significantly decreased. Hence, we conclude that the endurance exercise improves systemic inflammation in asthma patients.

References

1)Amina H, Abdul G, Abdul K, Jasim M .2010. Association Between C Reactive Protein and Asthma. TurkishToraxDergisi 11(3), 098 -104.
2)Butland BK, Strachan DP, Rudnicka AR. 2008.C-reactive protein, obesity, atopy and asthma symptoms in middle-aged British adults. EurRespir J 32, 77-84.
4)Danesh J, Wheeler JG, Hirschfield GM. 2004.C–reactive protein and other circulating markers of inflammationin the prediction of coronary heart disease. N Eng J Med 350, 387-97.
de Salles BF, Simão R, Fleck SJ, Dias I, Kraemer-Aguiar LG, Bouskela E. 2010. Effects of resistance training on cytokines.Int J Sports Med 31(7), 441-50.
5)Doron A, Inon R, Mordechay Y, Arthur K, Ophir A, Ron S, Peter B. 2006. Inverse Association between Pulmonary Function and C -reactive protein in Apparently Healthy Subjects. Am J RespirCrit Care Med 174, 626–632.
7)Ebrahim R , Hassan E, Hossein A, Vajihe C,Armin R. 2011. Armin Razi Evaluation of HighSensitivity C-Reactive Protein in Acute Asthma Tanaffos 11(1), 32-37.
8)Eizadi M, Kohandel M, kasbparast JR M, Sarshin A. 2011. Hs-CRP and TNF- alpha inresponse to a stepwise incremental bicycle test in adult obese men 1(3), 35-43.
9)Ford ES. 2003. Asthma, body mass index, and Creactive protein among US adults. Journal of Asthma 40(7), 733-
10) Ford ES. 2005. The epidemiology of obesity and asthma. J Allergy ClinImmunol 115, 897-909.
11)Gann WQ, Man SFP, Senthilselvan A. 2004.Association between chronic obstructive pulmonary disease and systemic inflammation: a systematic review and meta analysis. Thorax 59, 574-80.
12)Heidenfelder B, Johnson M, Hudgens E, Inmon J, Hamilton RG, Neas L, Gallagher JE. 2010.Increased plasma reactive oxidant levels and their relationship to blood cells, total IgE, and allergenspecificIgE levels in asthmatic children. J Asthma 47(1), 106-11.
13)Hotamisligil GS. 2006. Inflammation and metabolic disorders. Nature 444, 860-7.Huang H, Iida KT, Sone H, Ajisaka R. 2007.The regulation of adiponectin receptors expression by acute exercise in mice.ExpClinEndocrinol Diabetes 115(7),417-22.
15)Jousilahti P, Salomaa V, Hakala K, Rasi V, Vantera E, Palosuo T. 2002. The association of sensitive inflammation markers with bronchial asthma.Annals of Allergy, Asthma and Immunology89(4), 381-5.
16)Jousilahti P, Salomaa V, Hakala K, RasiV,Vantera E, Palosuo T. 2002. The association of sensitive inflammation markers with bronchial asthma.Ann Allergy Asthma Immunol 89, 381-5.
17)Jousilahti P, Salomaa V, Hakala K. 2002. The association of sensitive systemic inflammation markers with bronchial asthma.Ann Allergy Asthma Immunol 89, 381-5.
18)Jedariforoughi A. A qualitative survey about a patient's experiences and expectations of meeting healthcare professionals during multiple acute hospitalizations and follow up periods. Doctmedico Journal. 2022;2(1):180-93.
19)Jedariforoughi A, Karimpour F. The review of recent Developments in use of artificial intelligence in cancer detection. www doctmedico com. 2021.
20)Jedariforoughi A. Review article about developments in cancer prevention in public health in recent years. www doctmedico com. 2021.
21)Khosravaniardakani S. Vaping Products And Asthma In Youths: A Review Of The Prospective Study. International Journal of Medical Investigation. 2022;11(1):0-.
22)Khosravaniardakani S, Bokov DO, Mahmudiono T, Hashemi SS, Nikrad N, Rabieemotmaen S, et al. Obesity Accelerates Leukocyte Telomere Length Shortening in Apparently Healthy Adults: A Meta-Analysis. Frontiers in nutrition. 2022:139.
23)Khosravaniardakani S. The Effect of Cigarette and Its Nicotine Content on The Heart Health. International Journal of New Chemistry. 2022;9:26-41.
24)Kony S, Zureik M, Driss F, Neukirch C, Leynaert B, Neukirch F. 2004. Association of bronchial hyperresponsiveness and lung function with C-reactive protein (CRP): a population based study. Thorax 59, 892-6.
25)Kony S, Zureik M, Driss F. 2004. Association of BHR and lung function with CRP: a population based study.Thorax 59, 1-5.
26)Mendall MA, Strachan DP, Butland BK. 2000.C-reactive protein: relation to total mortality, cardiovascular mortality and cardiovascular risk factors in men. Eur Heart J 21, 1584-90.
27)Mendall MA, Strachan DP, Butland BK. 2000.C-reactive protein: relation to total mortality, cardiovascular mortality and cardiovascular risk factors in men. Eur Heart J 21, 1584-90.
28)O'Connor GT, Sparrow D, Weiss ST. 1995. Aprospective longitudinal study of methacholine airway responsivenessas a predictor of pulmonary-function decline: the Normative Aging Study. Am J RespirCrit Care Med 152, 87-92.
29)Olafsdottir IS, Gislason T, Thjodleifsson B, Olafsson I, Gislason D, Jögi R. 2005. C reactive protein levels are increased in nonallergic but not allergic asthma: a multicentre epidemiological study. Thorax 60(6), 451- 4.
30)Ridker PM. 2000. C-reactive protein and othermarkers of inflammation in the prediction of cardiovascular

disease inwomen. N Engl J Med 342,836–843.
31)Rijcken BJ, Schouten JP, Xu X. 1995. Airway hyperresponsiveness to histamine associated with accelerated declinein FEV1. Am J RespirCrit Care Med 51, 1377-82.
32)Sahoo RC, Acharya PR, Noushad TH, Anand R, Acharya VK, Sahu KR. 2009. A Study of HighSensitivityC - reactive protein in Bronchial Asthma. Indian journal of chest diseases & allied sciences 51(4), 213-6.
33)Tang Z, Yuan L, Gu C, Liu Y, Zhu L. 2005. Effect of exercise on the expression of adiponectin mRNA and GLUT4mRNA in type 2 diabetic rats.J HuazhongUnivSciTechnolog Med Sci 25(2), 191-3.
34)Tice JA. 2003. The relation of C-reactive protein levels to total and cardiovascular mortality in older U.S. women. Am J Med 114, 199–205.
35)Tilg H, Moschen AR. 2006. Adipocytokines: mediators linking adipose tissue, inflammation and immunity. Nat Rev Immunol 6, 772-83.

30)Varady KA, Tussing L, Bhutani S, Braunschweig CL. 2009. Degree of weight loss required to improve adipokine concentrations and decrease fat cell size in severely obese women.Metabolism 2009 58(8), 1096-101.
31)Yudkin JS, Stehouwer CD, Emeis JJ. 1999. C reactive protein in healthy subjects: associations with obesity, insulin resistance and endothelial dysfunction: a potential role for cytokines originating from adiposetissue? ArterioscIerThrombVasc Biol. 19, 972-8.