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Research Article

Effect of moderate intensity exercise on cardiovascular autonomic function in obese female adults of Western Nepal

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ABSTRACT

Background & Objectives: Obesity is a known risk factor for metabolic disorders such as high blood pressure, body fat abnormality, and glucose intolerance. In addition, obesity has been associated with dysregulation of autonomic function in humans, which may influence the morbidity and mortality of cardiovascular (CV) diseases. Being physically active has been proven to be a protective measure against CV diseases. Thus, this study aimed to determine the effect of moderate-intensity aerobic exercise on CV modulation among obese females in the mid-western region of Nepal.

Materials and Methods: We screened 28 healthy subjects to study the correlation between exercise and CV reactivity (Autonomic Nervous System (ANS) modulated) in obese subjects. Anthropometric and autonomic function parameters were measured before and after the intervention of four months of aerobic exercise and analysed.

Results: The result showed a strong correlation of exercise with Resting Heart Rate (RHR) (p= 0.02), baseline systolic blood pressure (SBP) (p= 0.01), and diastolic blood pressure (DBP) (p =0.03). It also showed a significant correlation between exercise and CV reactivity change in SBP (p = 0.01) during the cold pressor test (CPT). However, some variables like change in DBP during CPT did not have a significant correlation (p= 0.94) with exercise.

Conclusion: As obesity-related morbidity and mortality are burgeoning in our society, it is imperative to focus on exercise training to prevent and reverse CV neuropathy. Relevant aerobic exercise intervention can serve as an excellent countermeasure for the prevention of CV disease (CVD) by CV autonomic modulation.

Keywords: anthropometric variables, blood pressure, cold pressor test, exercise, obesity,

INTRODUCTION

Obesity is a global problem, the prevalence of which is rising continuously in almost every country mostly because of decreased physical activity [1]. It is a result of a chronic imbalance between energy intake and energy expenditure [2]. Worldwide more than half a billion people are estimated to have obesity. The rise in the prevalence of obesity is not only limited to developed countries but to developing nations like Nepal as well, making it a significant public health concern [3,4]. Economic transition and the urbanization process precipitate increased lifestyle-related risk factors such as low physical activity and unhealthy dietary habits which ultimately lead to obesity [4].

One of the most reliable and valid indicators of obesity is body mass index (BMI), a non-invasive measure based on an individual's weight and height. Based on unequivocal data on morbidity; a BMI of 30 or more is taken as the threshold for obesity [2]. Studies in adults provide overwhelming evidence that obesity is the major risk factor for non-communicable diseases (NCDs) such as diabetes, cardiovascular disease (CVD), and cancer [2]. A strong link has been identified between obesity and autonomic nervous system (ANS) dysfunction. The ANS plays a vital role in regulating heart rate (HR) and vascular tone [5]. So, it is recommended to reduce the body weight to an optimal level to prevent a wide range of diseases [4,6].

One of the best ways to reduce weight is regular exercise. Regular physical exercise has several beneficial effects on overall health. Regardless of age, it reduces the risk of cardiovascular disease through the adaptation of the heart and vascular system by reducing the resting heart rate (RHR), blood pressure (BP), and atherogenic markers, and by causing physiological cardiac hypertrophy [6-8].

Both sympathetic nervous system (SNS) and parasympathetic nervous system (PNS) activities can be assessed by different non-invasive cardiovascular autonomic function tests. RHR, an indirect measure of cardiac vagal activity, is found to be directly related to cardiovascular disease and mortality and inversely related to life expectancy [8,9]. It is well known that higher exercise capacity is strongly associated with lower RHR, suggesting that the parasympathetic branch of the ANS may play a key role in optimizing exercise performance [9].

Similarly, the cold pressor test (CPT), an easy non–invasive method, assesses the cardiovascular activity (left ventricular response) in response to any stress [10]. The impaired CPT in obese person could possibly be seen because of hypofunctional SNS as compared to their nonobese counterparts [11]. The assertion that weight loss via exercise training improves cardiovascular vagal activity in a variety of populations including obese individuals is supported by many studies [12]. However, no such studies have been done in the western part of Nepal. With this background, the main aim of our study was to determine the effect of four months of moderate-intensity aerobic exercise done thrice a week on cardiac autonomic function as measured by RHR, CPT, in obese female adults of western Nepal.

MATERIALS AND METHODS

This cross-sectional prospective study was conducted in the Department of Physiology, Nepalgunj Medical College Teaching Hospital (NGMCTH) from October 2018 to July 2019.

The ethical approval for this study was obtained from institutional review committee of Nepalgunj Medical College. A total of 32 sedentary obese female subjects of the age ranging 30 to 55 years who gave their consent were recruited in this study. Out of the 32 subjects, 28 completed the study. Other participants withdrew from the study due to their inability to comply with the scheduled visits.

The subjects with a history of hypertension (HTN) in the past 5 years, with long-term chronic disease, under medication (β-blockers, antidepressants, vasodilators), and current smokers were excluded from our study. On the day before testing, all the participants were medication-free and stable in terms of cardiopulmonary function. All tests were carried out at the same time of the day between 09:00 am and 12:00 pm to minimize the possible diurnal variation. All the parameters (Anthropometric and Autonomic function) were recorded before and after four months of supervised aerobic training given thrice a week.

Anthropometric Measurement

The anthropometric measurement was taken by standard procedures. The height was measured by a stadiometer with subjects having their shoes removed [13]. The body weights of the subjects were measured in light clothing, without shoes [13]. BMI was determined by dividing weight in kilogram (kg) by height in square meters (m^2) [14]. According to guideline adopted by WHO (World Health Organization) in 1997, a BMI of 18.5 kg/m^2 or more and less than 25 kg/m^2 is classified as normal, BMI of 25 kg/m^2 or more and less than 30 kg/m^2 is overweight and BMI of 30 kg/m^2 or more is considered obese [15]. Our study included 28 female subjects who had a BMI of more than 30 kg/m^2.

Autonomic Function Test

Autonomic function tests were performed to test the integrity of both sympathetic and parasympathetic cardiac innervations.

A. Resting heart rate (RHR)

It was recorded for the evaluation of the parasympathetic branch of ANS. It was recorded with Lead II of ECG (Sono Scape IE3 model, China) [16].

B. Cold pressure test (CPT)

The change in blood pressure (BP) after the CPT helps in evaluating the cardiovascular autonomic activity to stress. (Sympathetic branch of ANS) [16,17].

Resting BP was recorded from the right arm in a sitting position with a mercury sphygmomanometer (Niscomed, India). Then the subjects were asked to immerse the left hand up to the wrist in a big plastic mug containing ice cold water (temperature was maintained at 4- 10 degrees Celsius) for 1 minute (min). The BP was measured from the opposite arm at 1 min of immersion. Failure of the blood pressure to rise by 16-20 mm Hg systolic and 12-15 mm Hg diastolic was taken as an indication of autonomic neuropathy [11].

Weight loss protocol

The participants were enrolled in a 16-week program of aerobic exercise. Under the supervision of trained personnel, they performed thrice-a-week sessions of physical activity. In order to produce the desired metabolic effects, each exercise session lasted

50 min; 10 min of warm-up, 30 min of activity (brisk walking, light running), and 10 min of cool-down [18].

Data Analysis

Microsoft Excel and the Statistical program available in SPSS 20 (Statistical Package for Social Science) for Windows computer operating systems are utilized in this analysis. Anthropometric and cardiovascular variables were compared before and after exercise intervention for 16 weeks using paired samples t-test and data are presented as mean & standard deviation. The P value <0.05 was considered significant.

RESULTS

Table 1 provides the mean (M) and standard deviation (SD) of the BMI of female subjects before and after 4 months (16 weeks) of the exercise intervention. The fourth column in the table gives p – a value, where a value of less than 0.05 indicates significantly reduced BMI after the exercise intervention. The age range of the population was from 30 to 55 years with the mean age 39.89 ± 7.31 years (data not shown).

The mean(M) of RHR, resting SBP, and resting DBP before and after the exercise intervention has been presented in Table 2. The result indicates that all the parameters were decreased after the exercise training. Paired sample T-test was used to determine the effect of exercise on autonomic modulation. It was noted that post- exercise means of RHR and SBP (M± S.D =78.07±5.44, 127.57± 4.12 respectively) were significantly lower than the pre-exercise (M± S.D = 78.07 ± 5.44, 129.42±4.31 respectively) with the p-value of 0.02 and 0.01 respectively. The result also reveals that there was no significant decrease (p=0.94) in DBP after the exercise (M± S.D = 80.27±4.95) when compared with before exercise (M± S.D =81.42±6.08).

The autonomic function test included in this study provides the evaluation of the cold pressor test (CPT) done to evaluate the sympathetic neural outflow, so table 3 provides the mean increase in SBP and DBP before and after the exercise intervention. The data shows that the increase in SBP (M± S. D= 9.07 ±3.05) and DBP (M± S. D= 7.46 ± 3.19) during the CPT before the commencement of exercise were less indicating the neuropathy. The results also show that, when the same subjects were examined after the exercise training there was an increment in the ΔSBP (M± S. D=12.14± 4.1) during CPT. But ΔDBP (M± S. D= 7.07 ± 3.35) did not show any such modulation.

Table 1: Mean Body Mass Index (BMI) before and after 4 months of exercise training

Variable	Mean BMI (Before Exercise) Mean± S.D	Mean BMI (After 4 months of Exercise) Mean± S.D	Difference (Δ) Mean± S.D	*P value (paired T-Test*
BMI	31.36 ± 1.14	30.49 ± 1.44	0.87 ±1.02	0.00

(Correlation is significant at the 0.05 level)

Table 2: Mean Resting Heart rate (HR), Systolic blood pressure (SBP), and diastolic blood pressure (DBP) before and after 4 months of exercise training

Variable	Mean value (Before Exercise) Mean± S.D	Mean value (After 4 months of Exercise) Mean± S.D	Difference (Δ) Mean± S.D	*P value (paired T-Test*
RHR	79.92 ± 5.59	78.07± 5.44	1.85 ±2.92	0.02
Resting SBP	129.42±4.31	127.57± 4.12	1.85 ± 2.71	0.01
Resting DBP	81.42±6.08	80.27±4.95	0.85±2.06	0.03

(Correlation is significant at the 0.05 level)

Table 3: Mean increase in SBP &DBP during CPT (pre- and post-exercise)

Variable	Pre-exercise		Pot- exercise	
	Before-immersion (Mean±S.D)	After –immersion 1 minute (Mean±S.D)	Before-immersion (Mean ± S.D)	After –immersion 1 minute (Mean ± S.D)
SBP - CPT	129.42±4.31	138.5 ± 4.67	127.57±4.04	139.79±4.93
DBP – CPT	81.42±6.08	88.89 ± 6.13	80.57±4.86	87.64 ± 5.50
Difference (Δ) in SBP (Δ SBP – CPT) (Mean ± S.D)	9.07 ±3.05		12. 14 ± 4.1	
Difference (Δ) in DBP (Δ DBP – CPT) (Mean(M) ± S.D)	7.46 ± 3.19		7.07 ± 3.35	

(Correlation is significant at the 0.05 level)

Table 4: Comparison of difference in blood pressure (BP) before and at 1 minute after the Cold Pressor Test (CPT) -before & after exercise training

Variable	Δ in BP (CPT) Before Exercise (Mean± S.D)	Δ in BP (CPT) After Exercise (Mean± S.D)	*P value (PairedT-Test)*
Δ SBP - CPT	9.28 ±2.98	12.14±3.70	0.02
Δ DBP - CPT	7.46 ± 3.19	7.42±3.49	0.94

(Correlation is significant at the 0.05 level)

The paired T-test correlation between the mean difference (Δ) in SBP and DBP during CPT before exercise with after exercise has been presented in Table 4.

Here the result indicates that the change in Δ SBP - CPT before (M± S. D= 9.28 ±2.98) and after the exercise (M± S. D= 12.14±3.70) is statistically significant (p= 0.02). Whereas no such correlation (p=0.94) was observed in the case of Δ DBP- CPT. The mean Δ DBP- CPT – post-exercise was marginally less (M± S. D= 7.42±3.49) than Δ DBP- CPT pre-exercise (M± S. D= 7.46 ± 3.19).

DISCUSSION

The participants were able to lose 2 kg on average (data not shown) over a period of 4 months by moderate-intensity aerobic exercise and so BMI was decreased after exercise (30.49 ± 1.44) compared to pre-exercise level (31.36 ± 1.14) as shown in the table no 1. Many studies have reported that diet, exercise, or a combination of both induces weight loss, decreases visceral adiposity, and lowers plasma triglycerides (TG), plasma glucose, and BP [19-22]. Importantly, several of these beneficial effects of exercise are evident independent of weight loss, all of which are associated with increases in cardiac vagal tone [7]. Variant to this, some studies endorse the statement that the improvement in vagal tone is attributable to the decrease in weight loss [18]. There are inconsistencies in their finding, which can be ascribed to the diversity in the sample population. Additionally, there might be some methodological differences behind inconsistency in results. However, one should not undermine that the effect of exercise is not dependent on the weight loss of the person.

In the present study, the results presented in Table 2 indicate that the RHR before the exercise intervention was higher (79.92 ± 5.59) than the post-aerobic exercise levels (78.07±5.44). Obesity and cardiac ANS are intrinsically related. A 10% increase in body weight is associated with a decline in parasympathetic tone, accompanied by a rise in mean RHR and BP. Conversely, RHR declines after regular exercise [18, 23-25].

It was discerned that our training program significantly reduced RHR (p= 0.02). The observed effect of exercise interventions using moderate intensities is similar to the change reported previously in overweight middle-aged individuals [26]. Cardiac autonomic function can be improved by better physical exercise training. The exact mechanism for this is still unknown. One hypothesis is that regular exercise modulates cardiac autonomic control by enhancing vagal tone and decreasing sympathetic influence. It could possibly be related to subsequent improvements in body fat distribution, atherogenic lipoprotein profiles, and BP, as well as beneficial effects on muscular capillary density and ANS balance [27]. Moreover, it is also proven in some of the findings that an acute bout of exercise elicits a number of transient physiological responses, whereas accumulated bouts of acute exercise will lead to more permanent chronic adaptations [28].

Consistent with previous research [7], our data demonstrate that resting SBP and DBP were affected by exercise training (table no: 2). Chronic exercise training elicited a significant (p= 0.01 & 0.03 for ΔSBP and ΔDBP respectively) reduction in resting BP (ΔSBP:1.85±2.92 mmHg and ΔDBP: 0.85±2.06mm Hg). SBP indicates the force of contraction of the heart, whereas DBP is an indication of peripheral resistance [7]. There is ample evidence proving that moderate-intensity aerobic exercise decreases BP and prevents hypertension [28]. It may be due to the fact that exercise induces the activation of sympathetic cholinergic nerves supplying skeletal blood vessels and a surplus of released metabolites and increased body temperature will apparently cause the vasodilatation of these vessels. Due to this, TPR is decreased and finally, there is a fall in BP as well [29]. These findings of the present study are in accordance with the findings of some other studies [30-32]. Several studies have reported an inverse relationship between BP and physical activity [33,34].

The present results also highlight the ability of moderate-intensity exercise to elicit significant improvement in response to CPT.

From the combined results from Tables III and IV, we can conclude that the change in SBP during the CPT was lower (9.28 ±2.98) before the exercise training and was significantly increased (p= 0.02) after the training (12.14±3.70). The results of the study correlate with observations made by other authors [34]. Contrary to our result, some authors found no association between physical activity on SBP and DBP during the CPT [35]. The disparity between these investigations could be attributable to differences in methodology, diversity in sample population, and sample size.

However, we did not find any significant change in DBP during CPT even after the intervention of exercise as shown in Table no: IV. Even though both SBP and DBP were increased during CPT and the increase of SBP was statistically significant but increase of DBP was not statistically significant (p= 0.94). The effect of CPT on SBP was greater than the DBP. This may be due to the fact that CPT stress increases in sympathetic innervation which influences SBP by enhancing cardiac contractility. DBP is an indication of peripheral resistance. Nevertheless, the association with DBP has been reported in some studies [30]. Our result is in concordance with intervention studies [31,35,36] but at variance with others [33-34].

Therefore, our study has found that intervention of thrice-a-week moderate-intensity aerobic exercise for sixteen weeks can reverse the neuropathy related to obesity and it also improves cardiac rhythm regulation as measured by RHR and CPT in obese female adults.

CONCLUSION

Obesity is associated with cardiac autonomic neuropathy due to a decline in parasympathetic tone. Our study suggests that regular moderate-intensity aerobic exercise could serve as a potential adjunct therapy in the management of obesity to prevent cardiovascular complications and should be incorporated into public messages and awareness programs.

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