Original Article

Assessment of Cardiovascular Parameters on Submaximal Treadmill Exercise in Obese versus Nonobese Adults

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Abstract

Background: Obesity and overweight, irrespective of metabolic status, confer an increased risk of adverse consequences including cardiovascular diseases (CVDs). The mechanisms underlying altered cardiovascular responses in obese individuals during and after submaximal treadmill exercise are the subjects of great interest to public health. After graded exercise, delayed heart rate recovery (HRR), exaggerated blood pressure (BP) response, and prolongation of QT interval are the powerful predictors of cardiovascular morbidity and mortality that may facilitate timely identification of individuals at risk of CVD and help to evaluate the effectiveness of treatment interventions. **Aim:** To compare the cardiovascular parameters on submaximal treadmill exercise in obese and nonobese adults. **Materials and Methods:** This study was conducted on 80 subjects, 40 obese (cases) and 40 normal-weight individuals (controls), belonging to the age group of 18–60 years. Each participant was subjected to submaximal treadmill exercise according to the Bruce protocol. Heart rate (HR), systolic BP and diastolic BP (SBP and DBP), mean arterial pressure (MAP), QT and corrected QT (QTc) intervals, and rate pressure product (RPP) were measured preexercise, immediately after exercise, and during passive recovery at 1 min and 5 min after exercise. The Chi-square test and Mann–Whitney *U*-test, whichever is appropriate, were employed for the comparison of variables between the two study groups. $P < 0.05$ was considered statistically significant. **Results:** Mean HR immediately after exercise, at 1 min and 5 min postexercise was significantly higher in obese when compared to nonobese participants ($P = 0.006$, $P = 0.001$, $P = 0.001$) despite similar resting HR in both the groups ($P = 0.874$). Mean SBP, DBP, MAP, and RPP were significantly higher in obese in comparison to nonobese subjects in all stages, i.e. before exercise, immediately after exercise, at 1 min and 5 min after exercise. QT and QTc intervals were also found to be significantly greater in obese than nonobese subjects in all stages ($P = 0.001$ each). **Conclusion:** Obese subjects had higher resting BP (SBP, DBP, and MAP), QT/QTc interval, RPP, and increased response to submaximal treadmill exercise activity. Delayed HRR after exercise was also noted in obese subjects which indicates that obese populations are at risk of developing CVDs due to alteration in autonomic functions with sympathetic hyperactivity.

Keywords: *Cardiovascular parameters, nonobese, obese, submaximal treadmill exercise*

Introduction

Obesity is a universal health problem; its prevalence continues to increase at an alarming speed and has emerged as one of the leading modern-day pandemics. Obesity is defined as abnormal or excessive fat accumulation that presents a risk to one's health. A body mass index (BMI) over and equal to 30 is considered as obesity.[1] As per the World Health Organization data, more than 1.4 billion adults are overweight; of which, about 300 million women and over 200 million men worldwide are obese.[1] In India, obesity has escalated to the level of a severe public health concern, particularly among men.[2] This epidemic results from a combination of genetic susceptibility, increased availability of high-calorie foods, and decreased requirement for physical activity in modern society which threatens global well-being. Obesity and overweight confer an increased risk of adverse consequences including cardiovascular diseases (CVDs). In individuals free of CVDs, obesity increases the risk of developing heart failure.[3] Obesity causes structural and functional abnormalities in the left ventricle in the long run.

The mechanisms underlying altered cardiovascular responses in obese individuals during and after submaximal treadmill

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exercise are subjects of paramount importance to public health. After graded exercise, the delayed reduction in the heart rate (HR) measured during the 1st min and the 5th min is a powerful predictor of cardiovascular morbidity and mortality.[4] An exaggerated blood pressure (BP) response during treadmill exercise points towards a heightened cardiovascular risk.[5] The QT interval and corrected QT (QTc) interval in electrocardiograph (ECG) tracing can be used as a simple indicator of CVDs as they represent ventricular function.[6] There is thus an urgent need to evaluate cardiovascular health at rest and assess cardiovascular responses to exercise during and after in normal and obese adults, as the population is becoming progressively more overweight and obese. With the aid of this information, persons with a higher risk of CVD may be identified sooner and the efficacy of treatment approaches may be assessed. Hence, this study was carried out to compare the cardiovascular effects of submaximal treadmill exercise in obese and nonobese adults.

Materials and Methods

This hospital-based observational study was conducted in the Medicine department of a tertiary care teaching hospital. The study enrolled 80 subjects, 40 obese (cases) and 40 normal-weight individuals (controls), aged between 18 and 60 years, attending the inpatient or outpatient departments, using a nonprobability purposive sampling technique.

The inclusion criteria were adults of any gender, of the age 18–60 years; cases: Obese subjects with BMI >30 kg/m^2; controls: Nonobese subjects with BMI between 18.5 and 24.9 kg/m^2.

For both cases and controls, patients with established cardiopulmonary disease, diabetes mellitus, hypertension, mental and physical impairment leading to inability to exercise adequately; subjects in whom treadmill exercise was contraindicated, and those on medical therapy with beta-blockers, digitalis, and calcium channel blockers were excluded from this study.

The study was conducted following the Helsinki Declaration. Written informed consent was obtained from each respondent and the study was carried out after approval from the Institutional Ethics Committee vide letter no. GGS/IEC/07 dated April 19, 2021. All the participants were told about the purpose of the study and were ensured strict confidentiality.

Anthropometric measurements were noted for all subjects. Weight was measured without wearing shoes on a weighing scale that was evenly balanced. While standing barefoot against a wall with feet close together, height was measured in centimeters (cm) using a measuring tape. BMI was derived using Quetelet's index. Submaximal treadmill exercise was performed according to the Bruce protocol, i.e. a total of 6 min duration; the speed of the treadmill during the initial 3 min was 2.74 km/h or 1.7 mph with 10% grade and in the next 3 min, the treadmill speed was 4.02 km/h or 2.5 mph with 12% grade. The activity was carried out in a well-lit, ventilated space. All participants were instructed not to consume beverages and heavy meals and to refrain from engaging in any strenuous activity 24 hours before the test. The exercise protocol was properly explained to them. HR, BP measurements including systolic BP (SBP), diastolic BP (DBP) and mean arterial pressure (MAP), QT interval, corrected QT interval, and rate pressure product (RPP) were measured before exercise, immediately after exercise, and during recovery at 1 min and 5 min postexercise. RPP was calculated as the product of HR and SBP. QT interval was calculated using ECG, between the start of the Q wave and the end of the T-wave. The corrected QT interval (QTc) was determined using Bezett's equation: $QTc = QT/\sqrt{RR}$.

The data collected were entered as a data matrix in Microsoft® Excel® and analyzed using International Business Machines (IBM®, Armonk, New York, United States) Statistical Package for Social Sciences (SPSS®) software for Windows, version 21.0. The continuous variables were expressed as mean and standard deviation, whereas the categorical variables were presented in frequencies and percentages (%). The Chi square test and Mann–Whitney *U*-test, whichever is appropriate, were employed for the comparison of variables between the two study groups.[7] $P < 0.05$ was considered statistically significant.

Results

Table 1 summarizes the age distribution of study participants. The mean age of participants in the obese (case) and nonobese (control) groups was 40.63 ± 8.25 years and 40.90 ± 10.19 years, respectively. The two groups were comparable with respect to age distribution ($P = 3.04$). The number of males in the obese and nonobese groups was 23 (57.5%) and 20 (50%), respectively; while, the females in the obese and nonobese groups were 17 (42.5%) and 20 (50%), respectively. The mean BMI of obese and nonobese individuals was 33.05 ± 1.81 kg/m^2 and 23.09 ± 1.28 kg/m^2, respectively.

Table 2 shows the mean HR preexercise, immediately after exercise, and during the recovery phase (at 1 min and 5 min postexercise) in obese and nonobese groups. There was no statistically significant difference in the

Table 1: Age distribution of the study participants

Age group (years)	Number of subjects (%)		Total	*P*
	Obese group (case)	Nonobese group (control)		
<30	4 (10.0)	8 (20.0)	12	3.04
31–40	19 (47.5)	13 (32.5)	32	
41–50	10 (25.0)	9 (22.5)	19	
>50	7 (17.5)	10 (25.0)	17	
Total	40 (100.0)	40 (100.0)	80	

Chi-square test

mean preexercise HR between the two groups ($P = 0.874$). However, the mean HR immediately after exercise, 1 min and 5 min after exercise were significantly higher in the obese group than in the nonobese group ($P = 0.006$, $P = 0.001$, and $P = 0.001$, respectively), indicating delayed HR recovery (HRR) in obese subjects.

Table 3 depicts the mean SBP and DBP in the two study groups preexercise, immediately after exercise, at 1 min, and 5 min after exercise. The mean values of SBP were greater in the obese group than in the nonobese group before exercise, immediately after exercise as well as during recovery, i.e., at 1 min and 5 min after exercise, with statistically significant difference between the two groups ($P = 0.001$, $P = 0.011$, $P = 0.001$, and $P = 0.001$, respectively). As in the case of SBP, mean DBP was also higher in the obese group before exercise, immediately after exercise, at 1 min, and 5 min after exercise ($P = 0.001$, $P = 0.001$, $P = 0.004$, and $P = 0.001$, respectively).

Table 4 shows the MAP in the two groups. The mean values of MAP in the obese group were significantly higher than those in the nonobese group before exercise, immediately after exercise, at 1 min, and 5 min after exercise ($P = 0.001$ each).

The mean QT and QTc intervals in the two study groups before exercise, immediately after exercise, at 1 min, and 5 min after exercise are summarized in Table 5. It was observed that there was a statistically significant difference in these intervals between the two groups, before and after exercise ($P = 0.001$ each). The mean values of QT and QTc intervals were higher among obese patients as compared to nonobese patients.

Table 6 summarizes the mean values of RPP in the two study groups. It was observed that mean RPP was higher among the obese subjects preexercise ($P = 0.027$) and immediately after exercise ($P = 0.045$) in comparison to nonobese subjects. The difference was statistically highly significant, especially during the recovery period, i.e., at 1 min and 5 min after exercise ($P = 0.001$ each).

Discussion

The mean age of obese subjects in this study was 40.63 ± 8.25 years with maximum subjects in the age group of 31–40 years, followed by 41–50 years. The mean age of nonobese subjects was 40.90 ± 10.19 years with maximum subjects in the age group of 31–40 years followed by >50 years. Thus, the two study groups were age-matched. In the studies conducted by Majeed *et al*.[8] and Azeem *et al*.,[9] the mean age of obese subjects was 42.4 ± 3.1 years and 40.22 ± 1.5 years, respectively; while, the nonobese subjects were aged 38.15 ± 1.50 years and 38.15 ± 1.50 years, respectively. In this study, 54% were male and 46% were female with male-to-female ratio of 1.16:1. Similar findings were observed in the studies conducted by Gowdhami *et al*.[10] and Dimkpa and Oji[11] who observed a male-to-female ratio of 1:1 and 1.04:1, respectively.

Table 2: Heart rate in different stages in the study participants

HR (beats/min)	Mean (SD)		*Z*	*P*
	Obese group (case)	Nonobese group (control)		
Preexercise	85.38 (8.39)	85.80 (8.42)	−0.159	0.874
Immediate after exercise	150.75 (15.23)	140.30 (17.47)	−0.996	0.006
1 min after exercise	125.90 (11.83)	108.38 (8.55)	−5.994	0.001
5 min after exercise	101.30 (4.74)	90.35 (4.87)	−6.864	0.001

Mann–Whitney *U*-test. HR: Heart rate; SD: Standard deviation

Table 3: Systolic blood pressure and diastolic blood pressure in different stages in the study subjects

SBP and DBP (mm/Hg)	Mean (SD)		*Z*	*P*
	Obese group (case)	Nonobese group (control)		
Preexercise				
SBP	122.55 (8.47)	113.95 (11.44)	−3.437	0.001
DBP	82.95 (5.99)	76.15 (5.66)	−4.467	0.001
Immediate after exercise				
SBP	148.60 (7.87)	143.10 (9.43)	−2.558	0.011
DBP	91.90 (4.32)	87.45 (5.49)	−3.769	0.001
1 min after exercise				
SBP	135.60 (7.76)	129.20 (8.14)	−3.404	0.001
DBP	85.65 (4.15)	82.25 (5.38)	−2.870	0.004
5 min after exercise				
SBP	128.90 (7.88)	120.25 (7.85)	−4.155	0.001
DBP	82.00 (5.16)	77.20 (5.02)	−3.957	0.001

Mann–Whitney *U*-test. SBP: Systolic blood pressure; DBP: Diastolic blood pressure; SD: Standard deviation

Table 4: Mean arterial pressure in different stages in the study subjects

MAP (mmHg)	Mean (SD)		*Z*	*P*
	Obese group (case)	Nonobese group (control)		
Preexercise	96.15 (6.45)	88.74 (7.27)	−4.219	0.001
Immediate after exercise	110.80 (5.08)	106.00 (5.84)	−3.846	0.001
1 min after exercise	102.30 (4.92)	97.90 (5.89)	−3.261	0.001
5 min after exercise	97.64 (5.12)	91.55 (5.43)	−4.543	0.001

Mann–Whitney *U*-test. MAP: Mean arterial pressure; SD: Standard deviation

Table 5: QT and corrected QT intervals in different stages in the study subjects

QT and QTc interval (ms)	Mean (SD)		*Z*	*P*
	Obese group (case)	Nonobese group (control)		
Preexercise				
QT	400.00 (15.69)	364.50 (17.24)	−6.679	0.001
QTc	475.75 (13.61)	434.58 (14.23)	−7.452	0.001
Immediate after exercise				
QT	328.50 (18.05)	289.50 (17.53)	−6.737	0.001
QTc	508.65 (25.14)	454.25 (38.55)	−5.756	0.001
1 min after exercise				
QT	359.50 (20.99)	329.50 (17.53)	−5.600	0.001
QTc	519.45 (25.96)	442.18 (25.82)	−7.362	0.001
5 min after exercise				
QT	389.50 (16.94)	365.50 (14.31)	−5.581	0.001
QTc	505.78 (19.93)	448.08 (14.07)	−7.510	0.001

Mann–Whitney *U*-test. QTc: Corrected QT; SD: Standard deviation

Table 6: Rate pressure product in different stages among the study subjects

RPP	Mean (SD)		*Z*	*P*
	Obese group (cases)	Nonobese group (controls)		
Preexercise	10,481.40 (1394.97)	9782.70 (1428.41)	−2.209	0.027
Immediate after exercise	22,430.600 (2806.450)	21,250.200 (3035.248)	−2.806	0.045
1 min after exercise	17,085.15 (2006.36)	14,004.55 (1448.15)	−6.318	0.001
5 min after exercise	13,058.70 (1012.37)	10,863.00 (921.81)	−6.712	0.001

Mann–Whitney *U*-test. RPP: Rate pressure product; SD: Standard deviation

The mean baseline HR in both obese and nonobese subjects was normal with no statistically significant difference ($P = 0.874$). However, immediately after exercise, at 1 min and 5 min after exercise, HR was found to be significantly higher in obese subjects than in nonobese subjects ($P = 0.006$, $P = 0.001$, and $P = 0.001$, respectively). This indicates a delayed HRR in obese subjects after exercise which is a predictor of cardiovascular risk. In the previous studies conducted by Aslan *et al*.[12] and Gondoni *et al*.,[13] a negative association was found between BMI and HRR ($P = 0.001$ and $P = 0.003$, respectively) in obese after exercise due to decreased parasympathetic activity with an increased tendency for CVDs. Itagi *et al*.[14] in their study observed a higher mean HR in obese after exercise than in nonobese ($P < 0.001$) indicating reduced postexercise baroreceptor sensitivity and impaired autonomic regulation, which lead to attenuated HRR in obese. A study conducted by Barbosa Lins *et al*. concluded that impaired HRR was associated with higher BMI ($P = 0.006$) and obesity was associated with vagal nerve dysfunction.[15] Udaya *et al*. in their study found a negative correlation of HRR with increasing BMI (Pearson's correlation coefficient: $r = -0.354$, $P = 0.04$) suggesting increased risk for cardiovascular morbidity and mortality in obese patients.[16]

The mean resting SBP, DBP, and MAP in obese were found to be significantly higher than those in nonobese in this study. Furthermore, these parameters were considerably greater in obese individuals compared to nonobese subjects immediately following exercise, 1 min after exercise, and 5 min after exercise ($P = 0.001$). It is postulated that high sympathetic nervous system activity and activation of the renin-angiotensin-aldosterone system result in sodium and water retention and higher baseline SBP and DBP in obese individuals.[17] Similar findings were reported by Kerhervé *et al*.,[18] who observed that obese individuals had higher SBP, DBP, and MAP in comparison to normal weight ($P = 0.015$, $P = 0.004$, and $P = 0.004$) and overweight individuals ($P = 0.002$, $P < 0.001$ and

$P < 0.001$) at rest and during exercise due to obesity-specific sympathetic nervous system activity dysregulation. Gowdhami *et al*. concluded that after exercise, both SBP and DBP increased significantly in obese subjects compared to nonobese ($P < 0.005$) which may predict the future incidence of hypertension and CVDs.[10] Studies conducted by Martin *et al*.[19] and Chrysohoou *et al*.[20] also concluded that increasing level of obesity is associated with a rise in BP ($P = 0.002$, $P < 0.001$) and can predict the risk of CVDs.

The mean QT interval and QTc intervals at rest, immediately after exercise, at 1 min, and 5 min postexercise were found to be significantly greater ($P = 0.001$ each) in obese patients than in the nonobese controls. Higher QT and QTc intervals in obese subjects indicate delayed cardiac repolarization due to autonomic nervous system dysfunction that might contribute to their raised cardiovascular risk.[21] Itagi *et al*.,[14] Esposito *et al*.,[22] and Curione *et al*.[23] observed similar findings; they found significantly higher QT and QTc intervals in obese subjects when compared to nonobese, indicating an association between QT/QTc intervals and autonomic dysfunction in obese individuals. Another study conducted by Waheed *et al*. also observed a significant positive association between BMI and QTc interval ($P < 0.001$).[24]

The mean values of RPP were significantly higher in the obese group at baseline, immediately after exercise, at 1 min after exercise, and 5 min after exercise than in the nonobese group in this study. This indicates that obesity is a strong predictor of increased myocardial oxygen demand and decreased cardiac efficiency. In a study conducted by Parkhad and Palve, RPP was significantly positively correlated with BMI at rest and after exercise, in both male and female participants (Pearson's correlation coefficient: $r = 0.412$, $r = 0.369$).[25] Jena *et al*. also observed that overweight and obese subjects had higher RPP than normal participants with a significant positive correlation between RPP and anthropometric indices, i.e., BMI, waist circumference, and waist to hip ratio.[26] On the contrary, Thimmappa and Vageesh observed no statistically significant difference in RPP across BMI categories ($P = 0.939$) and no statistically significant association between BMI and RPP ($P = 0.512$) in their study.[27]

Strength of the study

Data regarding the effect of obesity on cardiovascular parameters after exercise are limited from the study region which prompted this study. This study helps us to understand the scenario of autonomic dysfunction in obese individuals which is of fundamental clinical importance. Early assessment of cardiovascular risk in obese individuals can be helpful in clinical practice to motivate and encourage the obese and their families to adopt a healthy lifestyle and other necessary measures to control and treat obesity, thereby preventing the associated long-term CVDs among them.

Limitations of the study

This was a single-center study with a limited sample size.

Conclusion

Being overweight and obese poses an increased risk of developing CVDs in the long run. Due to alteration in autonomic functions with resultant sympathetic hyperactivity, obesity can induce structural and functional abnormalities in the heart. Obese subjects have higher resting BP (SBP, DBP, and MAP), QT interval, RPP, and increased response to submaximal exercise activity. Delayed HRR after exercise is also noted in obese subjects which is a predictor of CVDs. Thus, the submaximal exercise stress test is a simple and noninvasive test that can provide valuable screening data to assess cardiovascular risk in obese individuals. It can be used in clinical practice to motivate and encourage obese individuals and their families to adopt a healthy lifestyle and undertake necessary measures to control and treat obesity, thereby preventing the associated long-term CVDs.

Ethical statement

The study was approved by the Institutional Ethics Committee of Guru Gobind Singh Medical College Faridkot, Punjab vide letter no. GGS/IEC/07 dated April 19, 2021. Written informed consent was obtained from each respondent. All the participants were told about the purpose of the study and were ensured strict confidentiality.

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Conflicts of interest

There are no conflicts of interest.

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