Original Article

Assessment of heart rate variability indices in overweight and obese Indian population

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Abstract

Background and Aim: Due to the increased incidence of overweight and obesity, its associated co-morbidities have also been reported to be escalating. Resting tachycardia, increased rate pressure product (RPP) and decreased heart rate variability (HRV) contribute to cardiovascular (CV) risks. Therefore, in the present study HRV indices and CV risks in overweight and obese subjects have been assessed.

Methods: Body mass index (BMI), waist circumference, waist-to-hip ratio, waist-to-height ratio (WHtR) and basal CV parameters were assessed in control (*N* = 50), overweight (*N* = 50) and obese (*N* = 50) group subjects. Spectral analysis of HRV was performed in all these groups using Biopac system.

Results: The basal CV parameters were significantly increased in overweight and obese subjects compared to controls. Low-frequency to high-frequency ratio (LF-HF ratio), marker of sympathovagal imbalance (SVI) was increased in overweight and obese subjects compared to controls. Total power (TP) and high frequency component expressed as normalized unit (Hfnu) which reflects the overall vagal drive to heart, was significantly decreased and low frequency component expressed as normalized unit (Lfnu) which reflects the sympathetic cardiac modulation, was significantly increased, in overweight and obese subjects compared to controls. These CV risks were predominately more in obese compared to overweight subjects.

Conclusion: Increased resting heart rate, RPP and decreased HRV due to sympathetic activation and vagal withdrawal observed in overweight and obese subjects might contribute to the increased CV risk in these high risk populations.

Key words: Cardiovascular risk, heart rate variability, obesity, overweight

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INTRODUCTION

Obesity with its associated metabolic diseases has reached epidemic proportions worldwide.[1] In Asian population, death as a result of being overweight or obese is about 300,000. Overweight/obesity is usually characterized by excess adiposity, which has been reported to produce number of cardiovascular (CV) dysfunctions.[2,3] Resting tachycardia, increased waist-to-height ratio (WHtR) and rate pressure product (RPP) has been reported to increase CV risks in overweight and obese subjects. Spectral analysis of heart rate variability (HRV) is a useful tool to measure cardiac autonomic activity and sympathovagal balance.[4] Obesity has been reported to produce dysfunctions of autonomic nervous system (ANS) either independently[5,6] or in association with its other co-existing morbidities such as insulin resistance, diabetes, hypertension and dyslipidemia.

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Sympathovagal imbalance (SVI) due to increased sympathetic and decreased parasympathetic activity has been reported in obesity.[7-9] However, the nature and magnitude of SVI in overweight subjects has not been adequately studied. Also, the progression in the intensity of SVI from overweight to obesity and the increased CV risks in these otherwise healthy overweight subjects have not been elucidated. Therefore, in the present study we have assessed the SVI and CV risks in overweight and obese subjects.

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MATERIALS AND METHODS

This cross-sectional study was conducted in the Department of Physiology, Jawaharlal institute of postgraduate medical education and research (JIPMER), Puducherry, India. After obtaining approval of the project plan from research and ethics committees of JIPMER, 150 healthy young adults were recruited from the Medicine OPD, JIPMER. Based on the body mass index (BMI) classification of WHO for Asian population[10] these subjects were divided into following three groups:

Control group: Normal healthy subjects having BMI 18.5-22.9 ($N = 50$)

Overweight group: Healthy subjects having BMI 23-27.4 ($N = 50$)

Obese group: Healthy subjects having BMI 27.5 or above ($N = 50$)

Written informed consent was obtained from all the participants prior to initiation of the study. Subjects on antihypertensive therapy or receiving any medication, with history of smoking and/or alcoholism, with acute or chronic ailments and known cases of diabetes mellitus, hypertension, cardiac diseases, kidney disease or any endocrinal disorder were excluded from the present study. As the level of physical fitness is a major determinant of vagal tone, subjects performing regular athletic activities, body-building exercises and yoga[11] were also excluded from the study.

Brief procedure

Height was measured to the nearest millimeter by a wall mounted stadiometer and weight was measured with digital weighing balance to the nearest 0.1 kg. Waist circumference (WC) was measured as the circumference of the abdomen at its narrowest point between the lower costal (10^{th} rib) border and the top of the iliac crest. Obesity indices such as waist-to-hip ratio (WHR), WHtR and BMI were calculated. BMI was calculated using the formula weight in kilograms divided by square of height in meters. After 10 minutes of supine rest, basal heart rate (BHR), systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded by oscillometric method using automated blood pressure monitor Omron MX3 (Omron Healthcare Co. Ltd, Kyoto, Japan). RPP, a determinant of myocardial oxygen consumption and work load was calculated using the formula, RPP = (BHR × SBP) $\times 10^{-2}$.[12] Mean arterial pressure (MAP) was also calculated. For recording of short-term HRV, recommendation of the Task Force on HRV was followed.[11] For this purpose, electrocardiography (ECG) electrodes were connected and Lead II ECG was acquired at a rate of 1000 samples/second during supine rest using BIOPAC MP 100 data acquisition system (BIOPAC Inc., USA). The data was transferred from BIOPAC to a windows-based PC with Acqkowledge software version 3.8.2. Ectopics and artefacts were removed from the recorded ECG. RR tachogram was extracted from the edited 256s ECG using the R wave detector in the Acqkowledge software and saved in ASC-II format which was later used offline for short term HRV analysis. HRV analysis was done using the HRV analysis software version 1.1 (Bio-signal Analysis group, Finland). Different frequency domain indices such as total power (TP), low frequency (LF) component expressed as normalized unit (LFnu), high frequency (HF) component expressed as normalized unit (HFnu) and LF/HF ratio were recorded.

Statistical analysis of data

SPSS version 19 (SPSS Software Inc., Chicago, IL, USA) was used for statistical analysis. All the data were presented as mean ± SD. Normality of data was tested by Kolmogorov Smironov test. The level of significance between the groups was tested using one-way ANOVA and post-hoc by Tukey-Krammer test.

RESULTS

There was no significant difference in age ($P = 0.838$) between the subjects of control group, overweight and obese group [Table 1]. Obesity indices such as BMI, WC, WHR and WHtR were significantly ($P < 0.001$) higher in overweight and obese group compared to that of control group [Table 1].

The BHR, SBP, DBP, MAP and RPP of overweight and obese group subjects were significantly ($P < 0.001$) increased compared to that of control group subjects [Table 1]. Among the frequency domain indices of HRV, TP and HFnu were significantly reduced ($P < 0.001$) and LFnu and LF-HF ratio were significantly increased ($P < 0.001$) in overweight and obese group subjects, and in obese group compared to overweight group subjects [Table 2].

DISCUSSION

In the present study, LF-HF ratio, the sensitive marker of SVI,[4] was significantly increased [Table 2] in overweight and obese subjects compared to controls and the increase in LF-HF ratio was more significant in obese group. As increased LF-HF ratio in resting supine condition indicates increased sympathetic and decreased vagal activity,[5,13] it is evident that the cardiac sympathetic drive was more in both overweight and obese subjects, which was more augmented in obese subjects. Increase in sympathetic activity in the overweight and obese subjects was further demonstrated by increase in LFnu, as it reflects the sympathetic drive to the heart.[5,13]

Table 1: Age and basal cardiovascular parameters in control, overweight and obese subjects

Parameters	Control group (*N*=50)	Overweight group (*N*=50)	Obese group (*N*=50)	*P* value
Age (years)	28.58±6.75	27.74±6.23	27.98±8.65	0.838
Height (meters)	1.62±0.09	1.61±0.09	1.63±0.12	0.613
Weight (kg)	56.47±6.15	64.96±7.39***	76.51±11.01***,###	<0.001
BMI (kg/m^2)	21.34±0.91	24.87±1.15***	31.16±2.13***,###	<0.001
WC (cm)	81.24±5.29	88.33±7.12***	91.74±9.90***	<0.001
WHR	0.82±0.08	0.90±0.06***	0.94±0.08***,#	<0.001
WHtR	0.50±0.04	0.54±0.04**	0.58±0.08***,##	<0.001
Basal CV parameters				
BHR (per min)	69.40±9.75	76.44±11.03**	80.98±10.69***	<0.001
SBP (mmHg)	107.46±8.61	112.54±8.37**	119.90±7.81***,###	<0.001
DBP (mmHg)	67.86±7.41	72.86±6.89**	80.12±7.87***,###	<0.001
MAP (mmHg)	81.06±7.33	84.82±9.93	88.47±7.11***	<0.001
RPP (mmHg/min)	74.61±12.24	86.13±14.76***	97.22±15.27***,###	<0.001

The values are expressed as Mean±SD; statistical analysis was done by one-way ANOVA. The *P* values<0.05 was statistically considered significant. BMI: Body mass index, WC: Waist circumference, WHR: Waist-to-hip ratio, WHtR: Waist-to-height ratio, BHR: Basal heart rate, RPP: Rate pressure product, MAP: Mean arterial pressure. The * mark indicates comparison with control group and the # mark indicates comparison with overweight group. *P<0.05; **P<0.01; ***P<0.001; #P<0.05; ##P<0.01; ###P<0.001

Table 2: Frequency domain indices of heart rate variability in control, overweight and obese groups

Parameters	Control group (*N*=50)	Overweight group (*N*=50)	Obese group (*N*=50)	*P* value
FDI of HRV				
TP (ms^2)	935.12±284.38	837.64±228.45	529.26±213.76***,###	<0.001
LFnu	40.51±14.74	48.49±17.46*	61.95±14.02***,###	<0.001
HFnu	59.48±14.75	51.51±17.46***	38.08±14.03***,###	<0.001
LF:HF	0.69±0.28	1.13±0.80**	1.55±0.79***,###	<0.001

The values are expressed as Mean±SD; statistical analysis was done by one-way ANOVA. The *P* values<0.05 was statistically considered significant. TP: Total power, LF: Low frequency component; HF: High frequency component, LFnu: Normalized low frequency component, HFnu: Normalized high frequency component. The * mark indicates comparison with control group and the # mark indicates comparison with overweight group. *P<0.05; **P<0.01; ***P<0.001; #P<0.05; ##P<0.01; ###P<0.001

Decrease in parasympathetic activity in overweight and obese subjects was further confirmed by the decrease in TP and HFnu [Table 2], as it represents vagal modulation to the heart.[5,13] Our findings are in agreement with the reports of earlier studies that SVI in obesity is due to increased sympathetic and decreased vagal activities[7-9] and its associated CV risks.[12]

This was further supported by significantly high BHR, SBP, DBP, MAP and RPP in these overweight and obese subjects [Table 1]. Since, BHR is an index of vagal tone,[13] resting tachycardia in overweight and obese subjects could be a potential CV risk in these subjects.[14] The increased blood pressure in these subjects further contributes to the augmented CV risk.[15] RPP, an index of myocardial work load and oxygen consumption has been recently reported as a CV risk,[16] was found to be significantly increased in overweight and obese subjects [Table 1]. Thus, findings of present study indicate increased CV risks in overweight and obese subjects. However, the association and independent contribution of HRV indices with CV risks in overweight and obese subjects has not been assessed in the present study.

Recently, it has been reported that CV risks is quite prominent in overweight and borderline obese subjects.[2,3] It has been reported that overweight subjects have resting tachycardia, which is a known CV risk.[14] Resting tachycardia has been defined as an etiology of all cause mortality.[14] However, no study was conducted till date to understand the physiological basis of increased CV risks in these subjects. Findings of the present study of increased sympathetic activity in the form of increased LFnu and decreased parasympathetic activity in the form of decreased HFnu depict the nature of alteration in the symapthovagal balance as the mechanism for risk of CV diseases in overweight population. Further, increased RPP, which is an established CV risk was prominately high in overweight population. This indicates that resting tachycardia and increased RPP could be due to the influence of SVI in these subjects. However, studies should be done in a larger sample size to determine the association of SVI with CV risks in subjects with borderline range of obesity.

Limitations of the study

Due to moderate sample size, we have not performed multiple regression analysis to determine the association of CV risks in overweight and obese populations.

CONCLUSION

Present study indicates the presence of SVI in the form increased sympathetic and decreased parasympathetic activity in overweight and obese subjects. Increased basal HR, RPP, BP and LF-HF ratio, and decreased TP in overweight and obese subjects make them susceptible to augmented CV risks, which was more intense in obese subjects. However, due to the small sample size in the present study, the cause and association of autonomic imbalance, and its link to increased CV risk in overweight and obese subjects could not be performed. Therefore, future studies in larger sample size should be conducted to assess the association of HRV indices with CV risks in this high risk population.

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