RESEARCH ARTICLE

A cross-sectional study on effect of obesity on autonomic functions in a tertiary care center

Vishrutha K V[1], Pansy Lyall[2]

[1]Department of Physiology, Srinivas Institute of Medical Science and Research Centre, Mangalore, Karnataka, India, [2]Department of Physiology, IQ City Medical College, Durgapur, West Bengal, India

Correspondence to: Pansy Lyall, E-mail: docpansy@gmail.com

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ABSTRACT

Background: Obesity is the most emerging problem in today's time. It is responsible for many metabolic as well as psychological ailments. Obesity and autonomic involvement have been studied, but evidence regarding the same is controversial. Obesity can no longer be considered as a silent epidemic in the new age scenario. Although people consider obesity a problem, it can still be regarded as one of the most overlooked public health issues. **Aims and Objectives:** This study aims to study the relationship between obesity and autonomic function tests. **Materials and Methods:** The present study was carried out in 86 male volunteers in the age group of 18–25 years. 40 of the subjects were included in the obese group, body mass index (BMI >30 kg/m^2) and the other 46 were included in the non-obese group (BMI <30 kg/m^2). Autonomic function tests in the form of heart rate variability and sympathetic skin response (SSR) tests were conducted in both the groups. **Results:** High frequency (HF) was significantly reduced in the obese group (240 ± 3.22, $P < 0.014$). Low frequency/HF was significantly altered in the obese group ($P < 0.05$). SDNN was significantly lesser in obese when compared to non-obese ($P < 0.05$). SSR latencies and amplitudes when compared between obese and non-obese did not reveal statistically significant results. However, both SSR amplitude and latency were lesser in the obese group when compared to non-obese. Tests indicate decreased parasympathetic activity in obese individuals. **Conclusion:** The link between obesity and autonomic functions if detected earlier in the long run will pave the way for a healthier life. This, in turn, may help in preventing cardiovascular morbidity which, in turn, reduces the burden on the society as a whole.

KEY WORDS: Body Mass Index; Autonomic Nervous System; Obesity; Autonomic Function Tests

INTRODUCTION

Obesity is emerging on the forefront as a health-care issue. It is an on-going epidemic heralding a new age crisis to both developed and developing countries. Both genetic and environmental factors interplay are observed in obesity.[1]

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Several factors such as sedentary lifestyle, lack of exercise or lack of motivation to continue exercising, intake of a calorie-dense diet, genetic and environmental factors contribute to the pathogenesis of obesity. Obesity results from a chronic imbalance between intake and energy expenditure. Hemodynamic and metabolic alterations occur in obesity. Obesity and autonomic involvement have been studied from times galore, but evidence regarding the same is controversial. Autonomic nervous system has two divisions sympathetic and parasympathetic. Autonomic system regulates body functions such as heart rate, respiration, urination, sexual function, and pupillary responses. Findings related to obesity and autonomic functions have been found to be inconsistent.[2,3] Hemodynamic instability in the form of increased cardiac output, changes in vascular reactivity, hypertension, diastolic dysfunction, and cardiomyopathy have been documented. Cardiovascular diseases are often linked with obesity. Insulin resistance, hypertension, and reduced high-density lipoprotein have been suggested to cause cardiovascular diseases in obese individuals.[4] However, autonomic instability in obesity cannot be ruled out. Obesity can result in complications manifold, namely hypertension, insulin resistance, dyslipidemia, and coronary heart disease.[5]

Autonomic dysfunctions on a long-term basis pose a high risk for cardiovascular morbidity and mortality. In addition to cardiovascular risks, obesity has also been associated with diabetes, hypertension, cancer, and sleep apnea.[6] This may be the cause for an overall increase in mortality rate linked to obesity. Early detection of autonomic instability may prove as a key factor in promoting weight loss techniques in obese individuals by highlighting the nuances associated with the same, which may improve the quality of living if acted upon.

Various autonomic tests are available to study autonomic dysfunctions including heart rate variability (HRV), sympathetic skin response (SSR), and R-R interval variation. One of the most reliable methods to study cardiovascular autonomic status is HRV in addition to being noninvasive and sensitive. There are not many studies in the Indian scenario pertaining to correlation between obesity and autonomic functions. In addition, evidence concerning the relation of sympathetic skin resistance to obesity has been scarce. Keeping in mind the above facts, the present study has been undertaken to study the relation between obesity and autonomic functions. The study objective was to evaluate the link between autonomic functions and obesity.

MATERIALS AND METHODS

The present study was undertaken in a tertiary care center on 86 male volunteers, in the age group of 18–25 years. Ethical clearance was obtained from the institutional ethical committee. Informed consent was taken. After explaining the procedure to the subjects, anthropometric values were noted down. Obesity criterion based on the WHO cutoffs was added when the body mass index (BMI) was >30 kg/m^2. Group 1 consisted of 40 obese subjects and Group 2 had 46 non-obese subjects who were controls. Mean BMI of obese subjects (32.04 ± 2.66) was higher than that of controls (20.43 ± 2.34). Subjects on medication for cardiovascular and central nervous system disorders were excluded from the study. Subjects with a past/present history of cardiovascular disorders and history of smoking were excluded from the study. Subjects were asked to refrain from consuming tea/coffee/beverages on the day of the study. This was done to avoid any direct influence on cardiac autonomic activity. They were asked to report to the center after a light breakfast with light clothing.

The subjects weight was measured accurately using a digital weighing scale. Standing height was measured using a stadiometer. Both waist circumference and hip circumference were measured with the help of a flexible measuring tape. Measurement of HRV - ambulatory computerized electrocardiogram (ECG) system was used in this study. Frequency domain and time domain methods were used for analysis. ECG was recorded in the supine position for 5 min after 5 min of supine rest. ECG obtained was stored in the computer for analysis as an offline data.

Measurement of SSR - the instrument used in the study was NCV-evoked potential (EP)-electromyography (EMG) machine (RMS EMG EP MARK II, Recorders and Medicare Systems, Chandigarh). Surface disk electrodes were used in the current study. SSR was recorded using supramaximal electrical stimulus. The latency (defined as the time interval between the stimulus and the onset of the SSR waveform) and amplitude (defined as the peak-to-peak amplitude of the SSR wave) of SSR was noted for each of the recordings.

All the values obtained were charted and tabulated as mean ± standard deviation. Comparison between obese and non-obese group was performed using Student's unpaired *t*-test.

RESULTS

A total of 86 male volunteers were included in the study. 40 of them were obese (BMI >30, n = 40) and were grouped as test subjects. The other 46 were control (BMI <30, n = 46). Comparison studies between obese and non-obese group were done using Student's unpaired *t*-test. $P < 0.05$ was considered as statistically significant. Weight, BMI, waist circumference, and hip circumference were significantly higher in Group 1 obese subjects when compared to controls [Table 1]. High frequency (HF) is significantly higher in non-obese controls when compared to obese. Low frequency (LF)/HF when compared showed a significant sympathovagal imbalance in obese. SDNN is significantly lower in obese [Table 2]. Mean values of SSR amplitude and latency are lower in obese when compared to non-obese though no significance is attached [Table 3].

Table 1: Comparison of anthropometric values between Group 1 (obese subjects) and Group 2 (controls)

Parameters	Group 1 (*n*=40)	Group 2 (*n*=46)	*P* value
Age	23.07±7.25	21.48±8.011	0.428
Height in meters	1.57±0.97	1.63±0.10	0.036
Weight in kg	82.39±10.16	60.57±9.38	<0.001*
BMI (kg/m^2)	30.09±2.92	21.67±2.43	<0.001*
Waist circumference (cm)	99.33±13.96	61.98±11.33	<0.001*
Hip circumference (cm)	98.99±13.78	80.97±8.22	<0.001*

*P value<0.05: Significant difference

Table 2: Comparison of HRV indices between Group 1 (obese) and Group 2 (controls)

HRV indices	Group 1 obese subjects (*n*=40)	Group 2 controls (*n*=46)	*P* value
LF (ms^2)	480±3.56	248±3.55	0.063
HF (ms^2)	240±3.22	675±3.48	0.014*
LF nu	52±2.15	42±3.11	0.078
HF nu	44.28±3.92	54.29±3.87	0.078
LF/HF	1.33±3.66	0.78±3.78	0.043*
SDNN	30.65±3.6	42.53±2.08	0.037*

**P* value<0.05: Significant difference, HRV: Heart rate variability, LF: Low frequency, HF: High frequency

Table 3: Comparison of SSR parameters in obese and non-obese group

SSR parameters	Group 1 obese (*n*=40)	Group 2 controls (*n*=46)	*P* value
SSR amplitude	0.27±0.18	0.32±0.42	0.054
SSR latency	0.32±0.19	0.27±0.18	0.052

**P* value<0.05: Significant difference, SSR: Sympathetic skin response

DISCUSSION

Obesity alters autonomic effects on heart. The obese subjects had significantly higher ($P < 0.05$), body weight, BMI, waist circumference, and hip circumference in comparison to the controls. SDNN being a time domain variable, reflecting parasympathetic nerve activity was significantly lower in obese when compared to controls. Among the frequency domain variables, HF power (ms^2) and HF (ms^2) indicating parasympathetic activity were significantly lower in obese individuals. LF/HF ratio when compared between obese and non-obese showed significance indicating sympathovagal imbalance in obesity.

SSR values when compared showed that SSR amplitude was shorter in obese when compared to non-obese individuals. SSR latency was shorter in obese when compared to non-obese. Although no significance was attached to the values obtained through SSR, it can be safely said that parasympathetic activity was found to be reduced in the obese.

In a study by Poliakova *et al*., an independent association between HRV and age, waist circumference, and body fat was found. However, there was no association between BMI and HRV.[7]

All the above findings indicate autonomic imbalance in obese individuals. Parasympathetic activity was lower in obese when compared to controls. This shows poor autonomic control in obese. Our findings are suggestive of decrease in parasympathetic activity in obesity. The test results obtained are akin to that found by other researchers, indicating that parasympathetic activity is altered more than sympathetic in obese individuals.[8] Parasympathetic activity dysfunction is presented in obese when compared to non-obese. Some researchers have suggested insulin resistance as a culprit for the parasympathetic imbalance in obese individuals. Insulin resistance increases with increased body weight causing a state of hyperinsulinemia. This causes low vagal activity in obese individuals.[9] Decrease in vagal activity by itself is a threat to the cardiovascular status.

Nagai *et al*. reported alteration in both sympathetic and parasympathetic systems in obesity.[10] However, certain studies have recorded an increase in sympathetic activity and decrease in parasympathetic activity in an obese individual as age advances. Cardiac sympathetic activity alteration depends on the duration of obesity.[11] In addition, role of hypothalamus in altering the autonomic functions has been put forth. Hypothalamus houses both the satiety center and regulatory center of ANS. Lesions in the hypothalamus may lead onto obesity and autonomic dysfunction. Whether dysfunction is due to obesity or obesity facilitates dysfunction is a point to be pondered on.[12] On the other hand, studies have also detected decrease in sympathetic activity in obese animal models. Laitinen *et al.* have linked central body obesity and total body fat to alteration in autonomic activity.[13]

Although our study found a reduction in parasympathetic activity, other studies have shown autonomic function to be altered in obese based on the duration of obesity. Further cross-sectional studies involving a larger population dealing with obesity in a longer duration basis are warranted for in this regard. The alteration in parasympathetic activity could be owed to the fact that in obese there is a skew in the eating pattern. They usually have a higher carbohydrate intake when compared to fat and protein intake. This may result in a change in parasympathetic activity according to Valensi *et al*.[14] Inadvertent intake of food also increases sympathetic activation.[12]

The strength of the present study is the contribution in understanding the autonomic imbalance associated with obesity in young adults. Obesity is alarmingly increasing in the young adult population in India. In times to come autonomic imbalance along with metabolic disturbances could set in at a younger age. Limitations of the present study are that since the study population involved younger age group, it may be a possibility that noteworthy changes in sympathetic nervous system have not been detected.

CONCLUSION

Obesity and autonomic dysfunction can be correlated. Autonomic dysfunction on a long-term basis is responsible for cardiovascular morbidity and mortality. Autonomic function testing is one way of detecting the possibility of cardiovascular risks in future. If autonomic dysfunction is detected, early obese can be motivated to lose weight. Weight loss may help in balancing the autonomic system which has been thrown off gear due to obesity. Poirier *et al.* in their study found an association between weight loss and parasympathetic activity which definitely paint a positive picture in the long run.[15] Obesity when detected early may help in preventing serious health issues which may occur as a consequence. In addition, it reduces the overburdening of the public health sector. Lifestyle modifications in the form of long-term exercise program, healthy diet is the need of the hour in the present era.

REFERENCES

1. Wang Y, Monteiro C, Popkin BM. Trends of obesity and underweight in older children and adolescents in the United States, Brazil, China, and Russia. Am J Clin Nutr 2002;75:971-7.
2. Fagard RH, Pardaens K, Staessen JA. Influence of demographic, anthropometric and lifestyle characteristics on heart rate and its variability in the population. J Hypertens 1999;17:1589-99.
3. Kuch B, Hense HW, Sinnreich R, Kark JD, von Eckardstein A, Sapoznikov D, *et al.* Determinants of short-period heart rate variability in the general population. Cardiology 2001;95:131-8.
4. Yadav RL, Yadav PK, Yadav LK, Agrawal K, Sah SK, Islam MN, *et al.* Association between obesity and heart rate variability indices: An intuition toward cardiac autonomic alteration-a risk of CVD. Diabetes Metab Syndr Obes 2017;10:57-64.
5. Narayan KM, Fagot-Campagna A, Imperatore G. Type 2 diabetes in children: A problem lurking for India? Indian Pediatr 2001;38:701-4.
6. NIH Guide. Pathophysiologic Mechanism of Obesity Associated Cardiovascular Disease. NHLBI; 2002.
7. Poliakova N, Després JP, Bergeron J, Alméras N, Tremblay A, Poirier P, *et al.* Influence of obesity indices, metabolic parameters and age on cardiac autonomic function in abdominally obese men. Metabolism 2012;61:1270-9.
8. Yakinci C, Mungen B, Karabiber H, Tayfun M, Evereklioglu C. Autonomic nervous system functions in obese children. Brain Dev 2000;22:151-3.
9. Steering Committee. The Asia-Pacific Perspective: Redefining Obesity and its Treatment. Melbourne: International Diabetes Institute; 2000.
10. Nagai N, Matsumoto T, Kita H, Moritani T. Autonomic nervous system activity and the state and development of obesity in Japanese school children. Obes Res 2003;11:25-32.
11. Gutin B, Barbeau P, Litaker MS, Ferguson M, Owens S. Heart rate variability in obese children: Relations to total body and visceral adiposity, and changes with physical training and detraining. Obes Res 2000;8:12-9.
12. Grewal S, Gupta V. Effect of obesity on autonomic nervous system. Int J Curr Bio Med Sci 2011;1:15-8.
13. Laitinen T, Lindström J, Eriksson J, Ilanne-Parikka P, Aunola S, Keinänen-Kiukaanniemi S, *et al.* Cardiovascular autonomic dysfunction is associated with central obesity in persons with impaired glucose tolerance. Diabet Med 2011;28:699-704.
14. Valensi P, Pariès J, Lormeau B, Attia S, Attali JR. Influence of nutrients on cardiac autonomic function in nondiabetic overweight subjects. Metabolism 2005;54:1290-6.
15. Poirier P, Hernandez TL, Weil KM, Shepard TJ, Eckel RH. Impact of diet-induced weight loss on the cardiac autonomic nervous system in severe obesity. Obes Res 2003;11:1040-7.

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