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Effect of Obesity on Sympathovagal Activities in Hypertensive Indian Population

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Summary: Decreased physical activity, increased psychosocial stress and work stress have contributed to the increased prevalence of obesity and Hypertension (HTN). Irrespective of the aetiology, sympathetic over activity has been recognized as the main pathophysiologic mechanism in the genesis of obesity and HTN. Sympathovagal imbalance or dysregulation of autonomic functions owing to sympathetic over activity and vagal withdrawal is reported to be the basis of many clinical disorders. Obesity, hypertension and diabetes mellitus are known to be associated with dysregulation of autonomic functions independently. Heart Rate Variability (HRV) has emerged as a practical, non-invasive tool to quantitatively investigate cardiac autonomic dysregulation. The present study was undertaken to ascertain whether obesity has any effect on further disruption of autonomic functions particularly in hypertensive patients. A total of 96 male and female adults aged between 40-50 years visiting the Primary Health Centre, Yelwala, Mysuru district, India were recruited for this study. They were grouped in to 3 (n=32) as Groups I (Obese hypertensive), II (non-obese hypertensive) and II (non-obese normotensive, control), HRV was determined using the One minute during deep breathing method. Data were presented as Mean ± SD, inferential statistics was by One Way ANOVA and Tukey's Post Hoc test p value <0.005 at α0.05 HRV was significantly decreased in obese hypertensive patients compared to the non-obese hypertensive patients. Our present study supports that obesity and hypertension probably has additive effect in causing autonomic dysregulation.

Keywords*:* Autonomic functions, HRV, Hypertension, Obesity.

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INTRODUCTION

Modernization, Urbanization and industrialization has decreased Physical activity, increased psychosocial stress and work stress have contributed to the increased prevalence of obesity and hypertension (HTN). Autonomic nervous system (ANS) plays important role in maintaining homeostasis, as it regulates most of the visceral functions in the body through its two major divisions, sympathetic and parasympathetic nervous system (Guyton and Hall, 2010). Sympathetic over activity has been recognized as the main pathophysiologic mechanism in the genesis of obesity and HTN. Sympathovagal imbalance or dysregulation of autonomic functions owing to sympathetic over activity and vagal withdrawal is reported to be the basis of many clinical disorders like obesity (Rajalakshmi et al., 2012), hypertension (Narhare et al., 2011), and diabetes mellitus (Fareedabanu et al., 2011).

Since ANS plays important role in cardiovascular health, it is not surprising that there has been great interest in the measurements of human sympathetic and vagus nerve traffic as tools that might inform physiological and pathophysiological mechanisms (Dwain et al., 1997)

Heart Rate Variability (HRV) is a term used to describe variations in both instantaneous heart rate and R-R interval set (Malik et al., 1996). There are regular fluctuations in heart rate, which are primarily due to changing level of both parasympathetic and sympathetic neural control of the heart (Arai et al., 1989); HRV is a non-invasive electrocardiographic marker reflecting the activity of sympathetic and vagal components of the ANS on sinus node of the heart. It expresses the total amount of variations of both instantaneous heart rate and R-R intervals (Stein et al., 1994; Van Ravenswaaij-Arts et al., 1993). Thus, HRV analyses the tonic baseline autonomic functions. In normal heart with an intact ANS, there will be

continuous physiological variations of sinus cycles reflecting a balanced sympathovagal state and normal HRV. In damaged heart which suffered necrosis, the change in activity of afferent & efferent fibers of the ANS and on local neural regulation will contribute to the resulting sympathovagal imbalance reflected by a diminished HRV.

HRV with deep breathing (HRVdb) is highly sensitive measure of cardiovagal or sympathetic cardiac function and defined as the difference in beats/minute between the shortest and the longest heart rate interval measured by electrocardiographic recording during six cycles of deep breathing. Therefore, HRVdb is a reliable and sensitive clinical test for early detection of cardio vagal dysfunction in a wide range of autonomic disorders (Dwain, 1997; Shields, 2009). This is defined as the difference in beat per minute between the shortest and the longest heart rate interval measured by electrocardiographic recording during six cycles of deep breathing in one minute. The shortest interval will be during inspiratory phase and longest being in expiratory phase of respiration. HRV reflects the balance between sympathetic and parasympathetic tone: when sympathetic tone is dominant HRV is low and vice-versa (VanRavenswaaij-Arts et al., 1993).

Obesity, hypertension and diabetes mellitus are known to be associated with dysregulation of autonomic functions independently. A previous study reported that there was no significant difference in the autonomic function of the obese and non-obese hypertensive groups (Amjad et al., 2016) which might be due inadequate sample size of the obese subjects and the non-inclusion of control group. The present study is therefore designed to ascertain whether obesity with hypertension has additive effect in further disruption of autonomic functions, so that an early intervention could decrease mortality, morbidity and the health risks related to hypertension and obesity.

MATERIALS AND METHODS

Study site and Ethical consideration

Institutional ethical committee approval was obtained and study was carried out at primary health centre Yelwala, Mysuru. Informed and written consent was taken from the subjects participating in the study.

Study design

A total of 96 participants were included in this study, they were grouped into 3 (n=32 each) as Groups I (Obese hypertensive), II (non-obese hypertensive) and II (non-obese normotensive, control). Sample size was based on the estimation by previous studies of similar kind at power of 80% and alpha error of 5%.

Inclusion criteria

Patients of both gender, aged 40-50years visiting the Primary Health Centre, having BMI of $\geq 25 kg/m^2$ were considered obese, based on BMI cut off for Indian population. Hypertensive on treatment, smokers, alcoholics and patients who had coffee, tea and cola drinks in preceding 48 hours were excluded from our study sample. Average BP measuring - $\geq 140/90$ mm of Hg and $\leq 130/80$ mm of Hg after 3 readings on different days considered hypertensive and normotensive respectively.

Heart rate variability measurement

HRV was measured using a simple bedside test of 1 minute HRV during forced deep breathing. The test was performed using CARDIART 6108-T electrocardiograph. ECG was taken every morning between 10 to 11 am. The subjects were made to rest quietly in supine posture and standard twelve lead ECG monitoring connected. Rhythm strip using lead II at speed of 25mm/sec was selected for our study purpose. After baseline recording, the patients were instructed to breathe deeply at 6-8 breaths/min. The HRV interval (R-R intervals between two adjacent QRS complex) was measured manually with a slide calliper. R-R intervals surrounding premature ventricular contractions were excluded. One minute HRV was calculated as the difference in beats/minute between the shortest and the longest heart rate interval. (Fareedabanu et al., 2011).

Statistical analysis

Data are presented as Mean ± SD, inferential statistics was by One Way ANOVA and Tukey's Post Hoc test p value <0.005 was considered significant. Data were entered in Microsoft Excel and analysed using SPSS® Version 23.0.

RESULTS

There was no significant difference in the mean age among the groups. The mean age among the groups ranged between 53 – 55 yrs. The mean weight in obese was 66.69±7.5, among Non –obese group was 55.9±6.8 and among controls was 58.4±4.9. The mean Height in Obese was 1.54±0.76, among Non-obese was 1.6±0.1, and among control group was 1.64±0.09. Mean BMI among obese was 28.04±2.2, in Non-obese it was 21.8±2.1 and among control group it was 21.7±1.8.

On applying One way ANOVA it was found that there was a significant difference in the recorded mean Maximum Heart Rate f(2,) = 31.208,P= 0.0001, mean Minimum Heart Rate f (2,) = 100.05, p= 0.0001 as well as the HRV f(2,) = 213.27,p=0.0001 among the three groups. Tukey's Post Hoc revealed significant lower recording of Maximum Heart Rate among the Obese Hypertensives (86.83±1.44) and Non –obese Hypertensives (88.69±1.05) compared to Controls (Non-obese and Non-Hypertensive) (90.03± 2.17). There is also a significant difference in the Max HR recorded between obese and Non-obese Hypertensives. With respect to Minimum HR, in

Table 1: Descriptive statistics of the patient characteristics and study variable

Variables	Group	Mean	Std. Deviation	95% Confidence Interval for Mean	
				Lower Bound	Upper Bound
Max HR (beat/minute)	I	86.8334	1.44746	86.3116	87.3553
	II	88.6913	1.05372	88.3113	89.0712
	III	90.0313	2.17424	89.2474	90.8151
Min HR (beat/minute)	I	71.9344	.96916	71.5850	72.2838
	II	67.6850	1.76150	67.0499	68.3201
	III	66.2516	2.08287	65.5006	67.0025
HRV	I	14.90	1.465	14.37	15.43
	II	21.01	1.640	20.41	21.60
	III	23.76	2.101	23.00	24.52
Age (years)	I	54.84	3.539	53.57	56.12
	II	54.22	3.599	52.92	55.52
	III	53.41	3.378	52.19	54.62
Weight (Kg)	I	66.69	7.541	63.97	69.41
	II	55.91	6.864	53.43	58.38
	III	58.41	4.996	56.61	60.21
Height (m)	I	1.54	.076	1.51	1.57
	II	1.60	.115	1.56	1.64
	III	1.64	.098	1.60	1.68
BMI (Kg/m^2)	I	28.0438	2.29107	27.2177	28.8698
	II	21.8138	2.11247	21.0521	22.5754
	III	21.7516	1.89416	21.0686	22.4345

Table 2: Shows the difference in the means in Maximum HR, Minimum HR and HRV among the Obese Hypertensives, Non-Obese Hypertensives and Control group respectively

		Sum of Squares	Degree of freedom	Mean Square	F-Ratio	P-Value
Max HR	Between Groups	165.046	2	82.523	31.208	.000*
	Within Groups	245.916	93	2.644		
	Total	410.962	95			
Min HR	Between Groups	559.000	2	279.500	100.054	.000*
	Within Groups	259.796	93	2.794		
	Total	818.796	95			
HRV	Between Groups	1315.250	2	657.625	213.274	.000*
	Within Groups	286.764	93	3.083		
	Total	1602.014	95			

comparison with the Controls (66.25± 2.08), there is a significant higher recording of Minimum HR among Obese (71.93± 0.96) and Non-obese hypertensive (67.68± 1.76). Significant difference is also noted between the mean of Min HR between obese and Non-obese Hypertensives. The mean one minute HRV showed a significant lower recording in the obese and non-obese hypertensives compared to controls (23.76±2.1). There is significant difference in the mean one minute HRV among the Obese (14.9±1.46) and Non-obese Hypertensives (20.01±1.64) as well.

DISCUSSION

The arterial pressure of hypertensive and obese individuals is more labile to various forms of physical stresses than those of normotensive and normal weight individuals, indicating altered baroreceptor reflex i.e. autonomic system which can be detected by HRV. This study was carried out to know the HRV response in newly diagnosed Non-Obese hypertensives and obese hypertensives along with controls to know the presence of additive effects of HRV variations in obese hypertensive patients. If obesity can change the HRV response as well as hypertension then, obese hypertensive patients should have more HRV alterations which can be dangerous to the health. To investigate this hypothesis, HRV was measured and the result showed the reduction in HRV in obese & non

obese hypertensives compared with normal healthy individuals, also reduction in HRV was observed more in obese hypertensives than non-obese hypertensives.

Prevalence of overweight, obesity as well as hypertension in India is showing an increase trend because of lifestyle changes among the population. Obesity is also on the rise with excessive consumption of processed foods and high fat diets. WHO had reported that prevalence of obesity is increasing worldwide including the developing countries (Jeong et al., 2005). Overweight and obesity itself are the independent risk factors for developing many diseases. Hypertension presents mainly because of genetic alterations and environmental effects. Both obesity and hypertension have higher sympathetic tone which is proved from elevated catecholamine levels (Schroeder et al., 2003). When sympathetic stimulation is high it releases renin from the kidney which in turn increases the angiotensin levels. Angiotensin II has positive effect on sympathetic ganglion and adrenal medulla while the negative effect on vagal outflow to the heart (Virtanen et al., 2003) which will decrease the HRV.

The present study shows that HRV is uniformly reduced in newly diagnosed hypertensive patients. There is slightly altered max HR and gross variations in min HR between obese and non-obese hypertensives, thus indicating the autonomic instability with decreased HRV, which is in concordance with the study carried out by Virtanen et al (2003**).** Not only was freshly diagnosed HTN associated with decreased HRV but also the association between HRV and blood pressure was present across the full range of blood pressure which is similar to the study by Shroeder et al (2003). Previous studies have shown reduced HRV in patients with hypertension alone (Mohd.Urooj et al., 2011) and in persons with obesity alone (Rajalakshmi et al., 2012; Windham et al., 2012) . Our study revealed a first of its findings that significant reduced HRV with gross variations in max HR and min HR between obese hypertensives and control indicating alteration in both parasympathetic and sympathetic outflow, which is evident by the more positive correlation of HRV with obese hypertensives compared to non-obese hypertensives, putting these patients in high risk category for early development of coronary artery disease. Short term regulation of heart rate is predominantly by sympathetic & parasympathetic system, so autonomic imbalance can be known by heart rate fluctuations (Rajalakshmi et al., 2012). Our study shows that HR was higher in obese hypertensives when compared to non-obese hypertensives & control group but the association of blood pressure with HRV was independent of HR**.**

There are studies that have proven that autonomic nervous system dysregulation precedes the development of clinical hypertension and obesity independently (Stein et al., 1994; Palatini and Julius, 2009) Decreased Parasympathetic activity in obesity was reported by Wu et al (2008). Obesity is associated with impaired glucose tolerance and hyperinsulinemia contributing to low cardiac vagal activity. Thus, obese individuals suffer from increased mortality risk due to cardiovascular disorder (Rajalakshmi et al., 2012) which gets worse if they develop Hypertension along with that.

In this century, we have amassed new knowledge about physical activity and associated positive implications on health. Almost all physiological process will be benefited by the regular physical activity. In fact, now a days it has taken first line of management in treating several chronic illnesses and also in rehabilitation. Different types of exercise have its own effects on the human body most important one being prevention of obesity. A working party of the Royal College of Physicians, convened in 1989, examined this evidence, recognized its importance and based a series of recommendations on it (Fentem, 1994).So not only identifying the autonomic imbalance through HRV can reduce the risk of developing hypertension and obesity but also by early intervention of life style changes and exercise can have a remarkable health improvement in the population.

In conclusion, regular monitoring of HRV and detection of autonomic instability and rehabilitative measures can not only reduce the incidence hypertension and obesity but may be diabetes also where the prevalence has crossed >50% in Indian population. The limitation of this study was that the duration of obesity was not estimated. Prospective studies are needed to find out whether reduced heart rate variability identifies hypertensive subjects with increased risk of cardiac mortality. Updated version of HRV analyser will be better than conventional methods of HRV measurements which have been used in the present study.

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