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The Relationship between Visceral fat and Heart Rate Variability in Normotensives and Prehypertensives

F B Irani[1 ID], B V Shinde[2 ID], S V Mulkhede[2], S R Phatale[2]

[1] Associate Professor, Dept. Physiology, MGM Medical College Aurangabad,
[2] Associate Professor, Dept. Physiology, MVP'S Dr.Vasantrao Pawar Medical college Nashik.
[2] Assistant Professor, Dept. Physiology, MGM Medical College Aurangabad,
[2] Professor, Dept. Physiology, MGM Medical College Aurangabad,

Corresponding author : FB Irani[1],
Associate Professor, Dept. Physiology, MGM Medical College Aurangabad,
driranifb@gmail.com
Mobile no 9922208436

Abstract

Introduction: Asian Indians are at risk of diabetes, hypertension(HT) and coronary diseases due to genetic background. Changing lifestyle, obesity also has correlation with HT and coronary damages. Autonomic imbalance with sympathetic hyper, parasympathetic hypoactivity leads to hypertension, which is also noted in prehypertensives obese.

Heart rate variability (HRV) is a noninvasive method to evaluate sympathovagal balance.

Visceral fat (VF) also correlates with sympathovagal imbalance (SVI), and is a sensitive indicator, as

compared to BMI to assess obesity.

With paucity of data that shows that autonomic imbalance slowly change normotensives to prehypertensives in obese Present study evaluate link between visceral fat and autonomic health in normotensives and prehypertensives.

Material & method: This study was conducted under the supervision of the Dept. of Physiology, MGM Medical College Aurangabad after Institutional ethical clearance was obtained. 80 consented health care students in the age group of 18-25 years of either sex, satisfying inclusion and exclusion criteria were enrolled. Group1 (n=40): Normotensives, with BMI < 25;Group 2(n=40): Prehypertensives with BMI ≥ 25. Anthropometric data was collected, i.e. height, weight, BMI, waist/hip ratio. VF was measured by Body fat analyzer (Omron HBF 375) based on BIA. ANS functions (HRV) were analyzed by a Diabetes risk profiler.

Statistical analysis: Level of significance was analyzed by using unpaired student t- test ($p < 0.001$). Correlation between VF and HRV parameter by Pearson correlation coefficient rejection at $p < 0.01$.

Results & Conclusion: Obese group showed significantly increased BMI, WHR, VF, basal HR and BP as compared to non obese. LFnu and LF:HF were more and HFnu was less in obese group. There was increased sympathetic activity and decreased vagal activity. VF showed significant positive correlation with LFnu & LF:HF ratio and negative correlation with HFnu with sympathovagal imbalance in obese subjects. To conclude that, obese, prehypertensives may suffer from CVD, related to either lowered parasympathetic or higher sympathetic activity & SVI.

Keywords: cardiac autonomic activity, heart rate variability, obesity, visceral fat

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INTRODUCTION: Asian Indians are at risk of diabetes, hypertension(HT) and coronary diseases due to genetic background. Changing lifestyle and obesity also has correlation with HT and coronary damages. Autonomic imbalance with sympathetic hyperactivity, which leads to vasoconstriction along with parasympathetic hypoactivity leads to hypertension. similar changes in relation to coronary damage and cardiovascular diseases are also noted in prehypertensives obese [1.]

Heart rate variability (HRV) is variation in time interval between each heartbeat due to variation in cardiac cycle length. HRV is mainly influenced by vagal activity. It is a sensitive indicator of cardiac autonomic function. HRV is a noninvasive quantitative method to evaluate sympathovagal balance. Obesity due to energy imbalance is associated with much comorbidity like hypertension, diabetes, coronary disease. Cardiovascular diseases with obesity are due to autonomic imbalance. There are many anthropometric indices included to measure obesity like Body mass index (BMI), waist-hip-ratio (WHR), waist circumference (WC),hip circumference, waist stature ratio, neck circumference (NC). Central (abdominal) obesity is more related with autonomic dysfunction. Visceral fat (VF) is adipose tissue around the internal organ, and is a sensitive indicator to assess obesity. VF is also correlated with sympathovagal imbalance and associated with increased cardiovascular diseases [2,3]

With paucity of data that shows that autonomic imbalance slowly change normotensives to prehypertensives in obese. Present study evaluate link between visceral fat and autonomic health in normotensives and prehypertensives.

MATERIAL & METHOD: This is a cross-sectional, case control study conducted under the supervision of the Dept. of Physiology. MGM Medical college Aurangabad India. Institutional ethical committee clearance was taken.

80 Consented health care students of different streams from campass in the age group of 18-25 years of either sex, satisfying inclusion and exclusion criteria were enrolled.

Inclusion criteria were healthy, normal weight subjects and obese subjects willing to enroll in the program.

Exclusion criteria were non cooperative, anxious, suffering with medical ailments, H/o medication, smoking, addiction to tobacco, alcohol.

Subjects were divided into two groups. Group1(n=40):Normotensive, with BMI < 25, (SBP100-110mmHg, DBP 60-79mmHg); Group 2(n=40): Prehypertensive with BMI ≥ 25, (SBP120-139mmHg, DBP 80-89mmHg)[4].

Anthropometric data was collected, i.e. height was measured by stadiometer, weight by digital weighing scale, waist circumference, hip circumference, by measuring tape and BMI, waist/hip ratio was calculated, BP was recorded by digital sphygmomanometer. Body fat was measured by Body fat analyser (Karade HBF- 375) based on Bioelectrical Impedance analysis (BIA). ANS functions (HRV) were analyzed by a Diabetes risk profiler and analysis machine.

All data was recorded in an autonomic research laboratory. First heart rate and blood pressure was recorded after five minutes of rest. Subjects were instructed to remain fasting for 2 hours before the test, avoid coffee, nicotine or alcohol 24 hrs prior to test and not to practice any form of physical exercise.

Analysis of HRV, ECG was recorded in supine position for 5 min after 15 min of rest. Subject was instructed to close eyes and avoid movements. R-R tachogram was plotted using the R-R intervals in the five minute lead II ECG. HRV was analyzed by frequency domain method.

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Power spectrum is divided into three frequency bands: VLF (0.0 to 0.04) Hz, LF (0.04 to 0.15)Hz, HF (0.15 to 0.4)Hz. Frequency domain indices such as total power(TP), normalized low frequency power (LF nu), normalized high frequency power (HF nu), and LF: HF ratio were calculated.[5]

STATISTICAL ANALYSIS: Level of significance was analyzed by using two tailed unpaired student t- test (0.001). Correlation between visceral fat and HRV parameters was tested by using Pearson's correlation coefficient; null hypothesis was rejected at < 0.01.

RESULT: 80 subjects Group A normotensives, non obese (n-40) and group B prehypertensives, obese (n- 40) that have satisfied the inclusion and exclusion criteria were selected

Table: 1 Comparison of Anthropometric data between the groups

Parameters	**Group 1 (mean ± SD)**	**Group 2 (mean ± SD)**	**P- value (significance)**
Age (years)	20.43 ± 1.91	21.35 ± 2.14	0.0448 (NS)
Height (cm)	164.80 ± 8.46	167.15 ± 5.99	0.1558 (NS)
Weight (Kg)	65.83 ± 5.86	75.90 ± 7.95	< 0.0001(S)
BMI (Kg/m2)	21.97 ± 3.71	26.98 ± 1.66	< 0.0001(S)
WHR	0.82 ± 0.035	0.87 ± 0.032	< 0.0001(S)
RHR (bpm)	77.08 ± 3.43	81.25 ± 12.71	< 0.048 (S)
SBP (mm Hg)	110.78 ± 7.01	123.95 ± 22.98	< 0.001(S)
DBP (mm Hg)	71.75 ± 6.31	84.58 ± 3.36	< 0.001(S)

(RHR - resting heart rate, SBP - systolic blood pressure, DBP - diastolic blood pressure, S- significant, NS - non significant)

Table: 1 shows there is no significant difference between age & height between group 1 and 2. Obese group showed significantly higher (p< 0.001) weight, BMI, WHR, resting HR and BP as compared to the non obese group.

Table: 2 Comparison of Body fat distribution between the groups

Parameters	**Group 1 (mean ± SD)**	**Group 2 (mean ± SD)**	**P-value (Significance)**
TBF (%)	22.50 ± 4.42	28.1 ± 5.20	<0.001(S)
SCF (%)	15.5 ± 2.46	20.00 ± 3.80	<0.001(S)
VF (%)	4.50 ± 1.77	11.87 ± 5.00	<0.001(S)

(TBF- Total body fat, SCF- Subcutaneous fat, VF - Visceral fat)

Table:2 shows obese group has significantly higher TBF, SCF and VF than the non obese group

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Table: 3 Comparison of Frequency domain parameters of HRV

Parameters	**Group 1 (mean ± SD)**	**Group 2 (mean ± SD)**	**P-value** (Significance)
LF nu (%)	41.48 ± 15.70	62.00 ± 13.92	<0.001(S)
HF nu (%)	65.25 ± 13.72	41.93 ± 15.11	<0.001(S)
LF:HF	0.7532 ± 0.67	1.272 ± 1.072	<0.001(S)
TP (ms2)	872.63 ± 405.54	496. 60 ± 256 ± 64	<0.001(S)

(LF nu: Normalized low frequency, HFnu: Normalized high frequency, LF:HF; Low frequency component : High frequency component, TP: total power)

Table0: 3 shows significant decrease in TP and HFnu and significant increase in LF nu and LF:HF ratio in obese group rather than non obese group.

Table: 4 Correlation between frequency domain parameters and visceral fat in obese

Parameters	**R-value**	**P-value (< 0.01)**
.LF nu	**0.47**	**< 0.01(S)**
HF nu	**-0.93**	**< 0.01(S)**
LF:HF ratio	**0.99**	**< 0.01(S)**

Table: 4 shows Significant positive correlation between LF nu and LF:HF ratio with visceral fat. And significant negative correlation between HFnu and visceral fat in obese pre-hypertensives

DISCUSSION: Heart rate variability is the cardiac beat -to-beat variation, mainly due to variation in cardiac activity during the respiratory sinus arrhythmia at rest. Other physiological factors affecting HRV are exercise, circadian rhythm and temperature. HRVmainly affected by vagal tone.[6] HRV acts as a noninvasive, quantitative tool to estimate cardiac autonomic function, mainly sympathovagal balance, which predicts severity of hypertension and acts as cardiovascular risk marker.

In our study heart rate and blood pressure was significantly higher in obese group, which marked increased sympathetic activity.[7] The study also showed decrease inTP and HFnu in prehypertensives obese than normotensives indicates decrease in HRV and decrease in vagal activity in heart.[8,9]

Increased LFnu in prehypertensive obese compared to normotensives indicates increased sympathetic activity[10] LF: HF ratio sensitive tool to represent sympathovagal balance.24 increased ratio represents sympathovagal imbalance. In our study LF:HF was increased in prehypertensives obese as compared to normotensives indicating sympathovagal imbalance and risk of cardiovascular diseases.

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Study showed LFnu and LF: HFratio of HRV were significantly positively correlated and HFnu was negatively correlated with visceral fat in obese. Which indicate relation between visceral fat and sympathovagal imbalance.

Studies have reported that obesity is associated with hyperinsulinemia and insulin resistance. Hyperinsulinemia leads to increased sympathetic tone, later leading to proinflammatory changes in blood vessels noted by increased cytokine marker (IL-6). Inflammation -mediated arterial rigidity leads to cardiovascular risk in obese

CONCLUSION: study concludes that, obese prehypertensive may suffer from cardiovascular disease, related to lowered parasympathetic activity and higher sympathetic activity and sympathovagal imbalance. These changes are seen earlier before any cardiovascular disease symptoms are observed. Hypertension once established cannot be reversed, but prehypertension can be reversed.

So early implementation of interventional programs (weight reduction, lifestyle changes and physical exercise) to reduce the risks by prehypertensives. And regular assessment of HRV measures can be used for early detection and management of cardiovascular disease.

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