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Visceral Adiposity and Autonomic Regulation: A Comparative Study of Normotensive and Prehypertensive Subjects

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KEYWORDS

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ABSTRACT:

Introduction: Indians possess a genetic predisposition to diabetes, hypertension (HT), and coronary artery disease. Lifestyle changes and rising obesity further contribute to the development of HT and cardiovascular complications. Autonomic imbalance characterized by heightened sympathetic and reduced parasympathetic activity is a key mechanism underlying hypertension and is observed even in obese prehypertensive individuals. Heart rate variability (HRV) offers a non-invasive assessment of sympathovagal balance. Visceral fat (VF) has been shown to correlate strongly with autonomic imbalance and is considered a more sensitive obesity indicator than BMI. Given the limited data on how autonomic dysfunction transitions normotensive obese individuals toward prehypertension, the present study aimed to evaluate the relationship between visceral fat and autonomic function in normotensive and prehypertensive subjects.

Materials and Methods: This study was conducted in the Department of Physiology, following approval from the Institutional Ethics Committee. A total of 80 consenting healthcare students aged 18–30 years, of either sex, meeting the inclusion and exclusion criteria were enrolled. Group 1 (n = 100) included normotensive participants with BMI < 25, and Group 2 (n = 100) comprised prehypertensive individuals with BMI ≥ 25. Anthropometric measurements—height, weight, BMI, and waist–hip ratio—were recorded. Visceral fat was assessed using a bioelectrical impedance–based body fat analyzer (Omron HBF 375). Autonomic function was evaluated through HRV analysis using a diabetes risk profiler.

Statistical Analysis: An unpaired Student's t-test ($p < 0.001$) was applied to determine statistical significance. Correlations between VF and HRV parameters were examined using Pearson's correlation coefficient, with significance set at $p < 0.01$.

Results and Conclusion: Obese participants exhibited significantly higher BMI, WHR, VF, basal heart rate, and blood pressure compared to non-obese individuals. They also demonstrated elevated LFnu and LF:HF ratios, along with reduced HFnu, indicating increased sympathetic drive and diminished vagal activity. Visceral fat showed a significant positive correlation with LFnu and LF:HF, and a negative correlation with HFnu, confirming the presence of sympathovagal imbalance in obese subjects. In conclusion, obese prehypertensives are at an increased risk of cardiovascular disease due to reduced parasympathetic activity, enhanced sympathetic dominance, and overall autonomic dysregulation.

INTRODUCTION: Asian Indians have an increased susceptibility to diabetes, hypertension (HT), and coronary artery diseases because of their genetic makeup. Lifestyle changes and rising obesity levels further contribute to the development of HT and coronary complications. An imbalance in autonomic regulationcharacterized by heightened sympathetic activity that causes vasoconstriction and reduced parasympathetic toneplays a major role in the onset of hypertension. Similar autonomic alterations are also observed in obese individuals who are prehypertensive, leading to early coronary and cardiovascular damage.[1]Heart rate variability (HRV) refers to the fluctuations in the time interval between consecutive heartbeats, which reflect variations in the cardiac cycle. Since HRV is primarily modulated by vagal influences,

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it serves as a sensitive marker of cardiac autonomic function. It provides a non-invasive and quantitative assessment of the sympathovagal balance. Obesity, resulting from chronic energy imbalance, is linked to several comorbidities such as hypertension, diabetes, and coronary artery disease. Much of the cardiovascular risk associated with obesity is attributed to autonomic dysfunction.

A variety of anthropometric measures are used to evaluate obesity, including body mass index (BMI), waist-hip ratio (WHR), waist circumference (WC), hip circumference, waist-to-height ratio, and neck circumference (NC). Central or abdominal obesity shows a stronger association with autonomic impairment. Visceral fat (VF), which surrounds internal organs, reliably reflects central obesity. VF has been shown to correlate with sympathovagal imbalance and a higher likelihood of cardiovascular disorders.[2,3]Bioelectrical Impedance Analysis (BIA) provides an affordable and simple method for assessing body composition, especially body fat. It works by sending a low-intensity electrical current through the body and measuring how much resistance the tissues provide. From this resistance, total body water (TBW) is estimated, which is then used to calculate fat-free mass. Body fat can subsequently be derived by subtracting fat-free mass from an individual's total body weight. [4] Although BIA is not considered the definitive standard, studies such as the Framingham Heart Study have shown a strong correlation between BIA and DXA for assessing fat-free mass. Moreover, BIA has been reported to outperform BMI in estimating actual body fat levels. [5]Because there is little evidence showing that autonomic imbalance may slowly move obese normotensive individuals toward prehypertension, this study aims to examine how visceral fat relates to autonomic function in both normotensive and prehypertensive subjects.

MATERIAL & METHOD: This is a cross-sectional, comparative observational study conducted under the supervision of the Dept. of Physiology. ethical committee clearance was taken. 200 Consented health care students of different streams from campus in the age group of 18-30 years of either sex, satisfying inclusion and exclusion criteria were enrolled.

Inclusion criteria: consisted of healthy, normal BMI, normotensive, as well as obese, prehypertensives, to their respective age and sex participants who were willing to take part in the study. **Exclusion criteria:** included individuals who were uncooperative, anxious, or diagnosed with any medical disorders. Participants with a history of medication use, smoking, or dependence on tobacco or alcohol were also excluded.

The participants were categorized into two groups. Group 1 (n = 100) consisted of normotensive individuals with a BMI below 25 and blood pressure values within SBP 100–110 mmHg and DBP 60–79 mmHg. Group 2 (n = 100) included prehypertensive subjects whose BMI was 25 or above, with SBP 120–139 mmHg and DBP 80–89 mmHg.[6]

Anthropometric measurements were obtained for all participants. BMI and the waist–hip ratio (WHR)was calculated. Following a five-minute period of rest, baseline measurements of heart rate and blood pressure were obtained. Body fat percentage was determined using a bioelectrical impedance based body fat analyzer (Karada HBF-375). Heart rate variability (HRV), an indicator of autonomic function, was measured with a diabetes risk profiler and analysis system.

All assessments were conducted in the autonomic research laboratory. Participants were advised to fast for at least two hours before testing, avoid consuming caffeine, nicotine, or alcohol for 24 hours, and refrain from any strenuous physical activity.

For HRV analysis, a 5-minute ECG recording in lead II was obtained while the subject lay in a supine position following a 15-minute rest. Participants were instructed to keep their eyes closed and minimize movement during the recording. An R-R tachogram was generated from the R-R intervals, and HRV was evaluated using frequency-domain analysis.

The power spectrum was segmented into three standard frequency bands: VLF (0.0–0.04 Hz), LF (0.04–0.15 Hz), and HF (0.15–0.40 Hz). From these bands, frequency-domain parametersincluding total power (TP), normalized low-frequency power (LF nu), normalized high-frequency power (HF nu), and the LF/HF ratiowere derived.[7]

Statistical Analysis: Data were analyzed for significance using a two-tailed unpaired Student's t-test

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and relationship between visceral fat and HRV variables was examined using Pearson's correlation coefficient.

RESULTS:A total of 200 eligible participants, as per the inclusion and exclusion criteria, were enrolled in the study.Group A consisted of 100 normotensive, non-obese individuals, while Group B included 100 prehypertensive, obese subjects.

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	0.84 ± 0.04	0.87 ± 0.14	< 0.0001(S)
RHR (bpm)	71.81±6.30	85.32±1.93	< 0.0001(S)
SBP (mm Hg)	112.66 ± 4.98	129.38 ± 6.12	< 0.0001(S)
DBP (mm Hg)	71.81± 6.30	85.32± 1.93	< 0.0001(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**	**Group 2**	**P-value**
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)

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	Group 2	P-value
LF nu	0.43±0.14	0.76±0.10	<0.05(S)
HF nu	0.62±0.12	0.30±0.09	<0.05(S)
LF:HF ratio	0.73±0.27	3.09±4.01	<0.05(S)
TP (ms2)	902.66±511.40	482.96±118.65	<0.05(S)

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

Table: 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	0.218	0.002(S)
HF nu	-0.93	0.01(S)
LF:HF ratio	0.158	0.025(S)
TP	-0.15	0.034(S)

Table: 4 shows Significant positive correlation between LF nu and LF:HF ratio with visceral fat.

And significant negative correlation between HFnu and TP with visceral fat in obese pre-hypertensives.

DISCUSSION:HRV refers to the fluctuation in time intervals between consecutive heartbeats and is primarily influenced by respiratory sinus arrhythmia during rest. Several physiological factors—including physical activity, circadian rhythms, and ambient temperature—also contribute to variations in HRV. Among autonomic influences, vagal tone plays a dominant role.[8] HRV is widely recognized as a non-invasive and quantitative marker of cardiac autonomic regulation, particularly the sympathovagal balance, and serves as a predictor of hypertension severity as well as a cardiovascular risk indicator.

In the present study, both heart rate and blood pressure were significantly elevated in the obese prehypertensive group compared with normotensive non-obese subjects, reflecting heightened sympathetic activation[9,10] A notable reduction in total power (TP) and HFnu values among prehypertensive obese individuals further indicates diminished HRV and reduced parasympathetic (vagal) modulation of the heart.[11,12]

Conversely, an increase in LFnu observed in the prehypertensive obese group signifies enhanced sympathetic drive.[13] The LF:HF ratio, a sensitive index of sympathovagal balance, was also elevated in this group. An increased LF:HF ratio reflects a shift toward sympathetic predominance and impaired autonomic regulation. In our study, this elevated ratio in prehypertensive obese subjects highlights a clear sympathovagal imbalance and suggests a heightened risk for future cardiovascular complications.

The findings of the present study demonstrated that LFnu and the LF:HF ratio showed a significant positive correlation with visceral fat, while HFnu and TP exhibited a negative correlation among obese individuals. These results indicate a clear association between visceral adiposity and sympathovagal imbalance.

Previous research has shown that obesity is frequently accompanied by hyperinsulinemia and insulin resistance. Elevated insulin levels enhance sympathetic

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nervous system activity, which subsequently contributes to pro-inflammatory changes within the vasculature, as reflected by increased cytokines such as IL-6. This inflammation-driven vascular stiffness plays a critical role in increasing cardiovascular risk among obese individuals.

CONCLUSION: The study concludes that obese prehypertensive individuals may be at heightened risk for developing cardiovascular disease due to reduced parasympathetic modulation, increased sympathetic activity, and overall sympathovagal imbalance. These autonomic alterations appear well before the onset of clinical cardiovascular symptoms. While established hypertension is largely irreversible, prehypertension offers a window of opportunity for intervention.

Early adoption of preventive strategies—such as weight reduction, lifestyle modification, and regular physical exercise—can substantially mitigate these risks. Routine evaluation of HRV parameters may serve as an effective tool for early detection, continuous monitoring, and timely management of cardiovascular risk in prehypertensive obese individuals.

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