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Subclinical Ventricular Electrical Remodeling In Young Adults With Adiposity: A Cross-Sectional Analysis Of The Ventricular Activation Time

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ABSTRACT

Background: Obesity has become more common in recent years, especially in young adults. Obesity is known to increase cardiovascular risk, but the early subclinical effects on cardiac electrical activity in healthy young individuals are not well understood. This study examined Ventricular Activation Time (VAT) – a novel ECG marker – and its association with general obesity (BMI) and central adiposity (WHR) in healthy young adults.
Methods: We carried out this cross-sectional community-based study in a small South Indian town over 5 months on healthy young adults (n=150, 18-30 years) and divided them equally into three groups (n=50 each) according to Asia-Pacific BMI criteria: normal weight (18.5-22.9 kg/m²), overweight (23-24.9 kg/m²), and obese (≥25 kg/m²). All three groups were matched for age and sex. VAT was measured from Lead II of a standard 12-lead ECG. BMI and WHR were recorded. Data were analyzed using ANOVA, Pearson's correlation, and Tukey's post-hoc test.
Results: VAT increased progressively with higher BMI. Normal weight, overweight, and obese groups had mean VAT values of 36.78±3.00 ms, 37.54±3.05 ms, and 41.22±3.09 ms, respectively ($F = 30.21$, $p < 0.001$). The obese group had significantly longer VAT than both normal weight and overweight groups ($p < 0.001$ for each), while no difference was observed between normal weight and overweight groups ($p = 0.42$). Both BMI ($r = 0.464$, $p < 0.001$) and WHR ($r = 0.324$, $p < 0.001$) showed significant positive correlations with VAT.
Conclusion: Even in healthy young adults, obesity is associated with early changes in ventricular electrical activity detected by VAT. Both overall adiposity and central obesity contribute to this effect. VAT is a low-cost, non-invasive ECG parameter that may serve as an early screening tool to identify obese young individuals at risk and to motivate weight management.

Keywords: Ventricular Activation Time, Obesity, Body Mass Index (BMI), Waist-to-Hip Ratio (WHR), Electrocardiography (ECG), Young adults.

INTRODUCTION

Overweight and obesity are now major global health problems and contribute significantly to non-communicable disease burden and mortality [1]. Several anthropometric indices are used to quantify body fat. Among them, Body Mass Index (BMI) reflects overall obesity, while Waist-to-Hip Ratio (WHR) specifically indicates central or abdominal adiposity [2]. The World Health Organization has defined standard BMI categories. However, evidence shows that Asian populations develop cardiometabolic complications at lower BMI thresholds [3,4]. Therefore, many researchers now use the Asia-Pacific BMI criteria for Asian individuals: normal weight 18.5-22.9 kg/m², overweight 23-24.9 kg/m², and obese ≥25 kg/m² [5,6].
Obesity is an established risk factor for cardiac arrhythmias and sudden cardiac death [7]. Conventional ECG parameters such as QT interval have been studied in obesity [8], but less is known about Ventricular Activation Time (VAT) in young healthy populations. VAT measures the time taken for the electrical impulse to travel from the His bundle to the Purkinje fibres and is obtained from the onset of the QRS complex to the peak of the R wave [9]. Prolonged VAT has been reported as a marker of subclinical cardiac damage in hypertensive patients, even before the development of left ventricular hypertrophy [10-13]. Body surface potential mapping studies have also shown that VAT can detect intraventricular conduction abnormalities when the standard ECG appears normal [17,20,21].

Given the limited data on early electrical changes in healthy young individuals, we conducted this study to investigate whether BMI and WHR are associated with VAT. We hypothesized that higher adiposity would be linked to longer VAT.

AIM AND OBJECTIVES

Aim: To find out how obesity markers affect VAT in healthy young adults.

Objectives:

1. To compare VAT readings across three groups – normal weight, overweight, and obese – in young adults.
2. To determine the correlation between BMI and VAT.
3. To determine the correlation between WHR and VAT.

MATERIALS AND METHODS

Study design and setting: This was a descriptive cross-sectional study done in a South Indian town. It lasted five months.
Participants: A total of 150 healthy young adults aged 18-30 years were randomly selected from the community. Based on Asia-Pacific BMI criteria [6], they were equally allocated into three groups (n=50 each): normal weight (18.5-22.9 kg/m²), overweight (23-24.9 kg/m²), and obese (≥25 kg/m²). All three groups were matched for age and sex (25 males and 25 females per group). Only healthy individuals without any known acute or chronic illness were enrolled.
Exclusion criteria: Individuals with a history of smoking, tobacco chewing, alcohol use, or any diagnosed illness – including hypertension, diabetes mellitus, cardiac, pulmonary, liver, thyroid, or neoplastic disorders – were excluded. Those with prior myocardial infarction, stroke, peripheral vascular disease, or those taking medications that could alter cardiac or pulmonary function were also excluded.
Ethical approval and informed consent: The Institutional Ethical Committee gave its approval for the study. All participants gave written consent after we explained the study's aims, how it would be done, and how their data would be kept confidential.
Methodology: Height (cm) and weight (kg) were measured using a stadiometer and digital weighing scale, respectively. BMI was calculated as weight/height² (kg/m²). Waist and hip circumferences (cm) were measured with a non-flexible tape, and WHR was calculated. A standard 12-lead resting ECG was recorded in the supine position after 10 minutes of rest. Paper speed was 25 mm/s (1 mm = 0.04 s) and calibration was 10 mm/mV. VAT was measured in Lead II from the first deflection after the P wave to the peak of the R wave [9]. All measurements were performed by a single blinded investigator to minimize bias.
Statistical analysis: Data were analyzed using SPSS version 23. Continuous variables are presented as mean ± standard deviation (SD). One-way ANOVA was used to compare VAT across the three groups, followed by Tukey's post-hoc test for pairwise comparisons. Pearson's correlation coefficient (r) was calculated to assess linear relationships between obesity markers (BMI, WHR) and VAT. A p-value <0.05 was considered statistically significant. Effect size (η^2) was also reported.

RESULTS

Participant characteristics: Table 1 shows the baseline characteristics of the 150 participants. Age and sex were well matched across groups. BMI and WHR progressively increased as expected.

Table 1: Baseline Characteristics of Study Participants (N=150)

Variable	Normal-Weight (n=50)	Overweight (n=50)	Obese (n=50)
Age (years)	23.34 ± 4.07	24.12 ± 3.74	24.06 ± 3.86
Sex (Male, n, %)	25 (50%)	25 (50%)	25 (50%)
Body Mass Index (BMI, kg/m²)*	20.77 ± 1.2	24.01 ± 0.65	29.02 ± 2.49
Waist-Hip Ratio (WHR)*	0.86 ± 0.07	0.93 ± 0.09	1.11 ± 0.21

*Data are mean ± SD unless otherwise specified. *p < 0.001 for inter-group differences by study design.*

VAT across BMI groups: Table 2 presents VAT values. There was a significant difference among the three groups (F = 30.21, $p < 0.001$, η^2=0.29), indicating that adiposity level explains 29% of the variance in VAT.

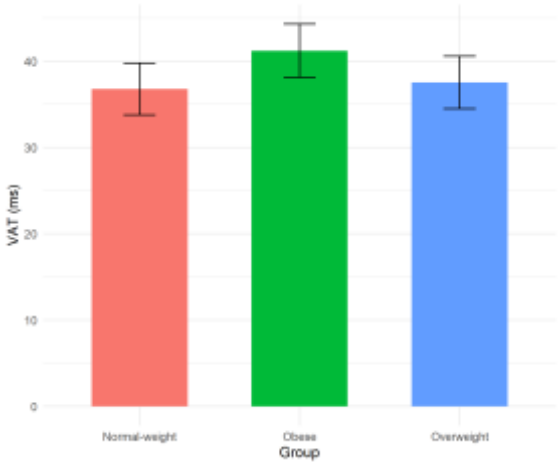

*Figure 1- Mean VAT (± SD) in normal-weight, overweight, and obese young adults. **p < 0.001 vs. normal-weight and overweight (one-way ANOVA, Tukey post-hoc).*

Table 2: Comparison of Ventricular Activation Time (VAT) across Groups

Variable	Normal-Weight (n=50)	Overweight (n=50)	Obese (n=50)	F-value	p-value*	Effect Size (η^2)
VAT (ms)	**36.78 ± 3.00**	**37.54 ± 3.05**	**41.22 ± 3.09**	**30.21**	**<0.001**	**0.29**

Data are mean ± SD. One-way ANOVA. ms = milliseconds.

Post-hoc comparisons: Table 3 shows pairwise comparisons. The obese group had significantly longer VAT than both the normal weight ($p < 0.001$) and the overweight ($p < 0.001$) groups. No difference was found between the normal weight and the overweight groups ($p = 0.42$).

Table 3: Post-Hoc Analysis (Tukey) for VAT

Comparison (VAT)	Mean Difference (ms)	95% CI for Difference	p-value*
Overweight vs. Normal-weight	**0.76**	**-0.90 to 2.42**	**0.42**
Obese vs. Normal-weight	**4.44**	**2.78 to 6.10**	**<0.001**
Obese vs. Overweight	**3.68**	**2.02 to 5.34**	**<0.001**

Tukey's post-hoc test. CI = Confidence Interval. Statistically significant comparisons are bolded.

Correlations: Table 4 shows a moderate positive correlation between BMI and VAT ($r = 0.464$, $p < 0.001$). Table 5 shows a weaker but still significant positive correlation between WHR and VAT ($r = 0.324$, $p < 0.001$).

Table 4: Correlation of BMI with VAT (N=150)

Variable 1	Variable 2	Pearson's r	p-value*
Body Mass Index (BMI)	**Ventricular Activation Time (VAT)**	**0.464**	**<0.001**

Pearson's Correlation

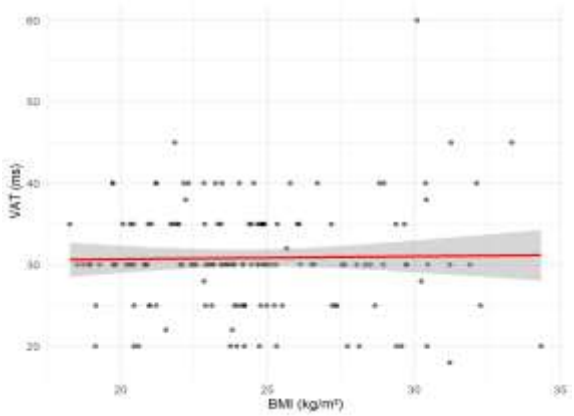

Figure 2- Scatter plot showing positive correlation between BMI and VAT (r = 0.464, p < 0.001). The red line represents the linear regression line with 95% confidence interval.

Table 5: Correlation of WHR with VAT (N=150)

Variable 1	Variable 2	Pearson's r	p-value*
Waist-to-Hip Ratio (WHR)	**Ventricular Activation Time (VAT)**	**0.324**	**<0.001**

Pearson's Correlation.

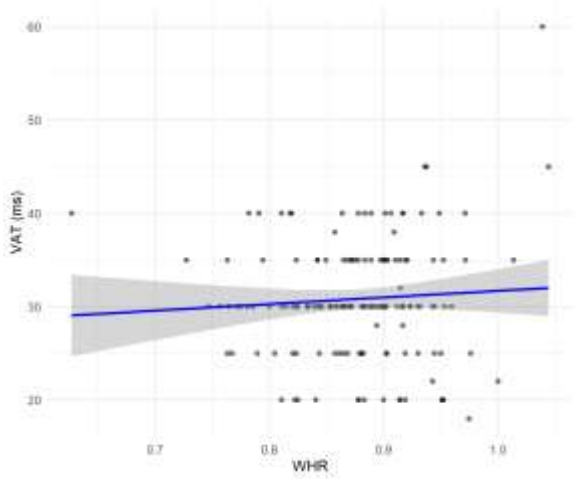

Figure 3- Scatter plot showing positive correlation between WHR and VAT (r = 0.324, p < 0.001). The blue line represents the linear regression line with 95% confidence interval.

DISCUSSION

This cross-sectional study of healthy South Indian young adults shows that both general obesity (BMI) and central adiposity (WHR) are associated with early electrical changes in the ventricles, measured as VAT. The key finding is that VAT was significantly longer in the obese group compared to the normal weight and overweight groups, even though all values remained within clinically normal limits. This suggests that the threshold for detectable subclinical electrical remodeling is crossed when an individual progresses from overweight to obesity, based on Asia-Pacific criteria.

Our results are consistent with earlier reports. Previous studies have shown that cardiac electrical remodeling, manifesting as increased QRS duration, occurs in hypertension and left ventricular hypertrophy [24,25]. Diastolic dysfunction is known to precede structural changes such as left ventricular hypertrophy [26-28]. Boles et al. [12] demonstrated a positive correlation between prolonged VAT and diastolic dysfunction in early hypertension. Our study extends this observation to young, otherwise healthy obese individuals, independent of any diagnosed comorbidity. This supports the idea that electrical remodeling can occur before structural remodeling [30].

Experimental models have shown that left ventricular hypertrophy initially affects conduction velocity before measurable changes in QRS amplitude, further supporting a sequence of electrical then structural remodeling [19,20]. The presence of subclinical myocardial injury, measured by high-sensitivity troponin, is more common in higher BMI categories, irrespective of metabolic syndrome [18].

Several mechanisms may explain the prolonged VAT in obesity. Epicardial adipose tissue can infiltrate the myocardium and create inert electrical barriers [26,28]. Obesity also causes inflammation in the body. Levels of cytokines like TNF-alpha and IL-6 go up [27]. These inflammatory substances can change how heart cells connect to each other and make electrical signals move slower [30]. More fat also means the heart has to work harder. This extra work puts stress on the heart wall and leads to remodeling [31,32]. Excess fat around the heart has also been linked to atrial fibrillation, showing that body fat affects many parts of heart function [33].

Limitations and future directions: The cross-sectional design prevents causal inferences. We did not perform echocardiography to exclude subclinical left ventricular hypertrophy, which could influence VAT. Future studies should incorporate echocardiography and advanced metabolic profiling to distinguish metabolically healthy obesity from metabolically unhealthy obesity [34,35]. Longitudinal studies are needed to determine whether VAT prolongation in obese youth predicts future cardiovascular events.

CONCLUSION

In healthy young adults, obesity is associated with significant prolongation of ventricular activation time, indicating early subclinical ventricular electrical remodeling. Both overall adiposity (BMI) and central adiposity (WHR) are positively correlated with VAT. The simple, non-invasive, and cost-effective measurement of VAT from a routine ECG can serve as

a screening tool for early cardiac risk stratification in young obese populations. This provides an opportunity for early lifestyle interventions aimed at weight reduction, which may reverse or arrest the progression of these changes.

Institutional ethical approval: obtained.
Conflict of interest: none.

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