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PR INTERVAL VARIATIONS IN ASYMPTOMATIC OBESE YOUNG ADULTS: A COMPARATIVE ECG ANALYSIS

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ABSTRACT

Background: This study investigates PR interval variations in asymptomatic obese young adults to assess their potential cardiovascular risk and predict future cardiovascular diseases. A total of 150 obese individuals (BMI ≥ 30 kg/m²), aged 18–39 years, were compared with 150 age- and sex-matched healthy controls (BMI < 22 kg/m²). Anthropometric indices including Body Mass Index (BMI), Body Surface Area (BSA), and Waist-Hip Ratio (WHR) were calculated. Vital signs such as pulse rate, systolic blood pressure (SBP), and diastolic blood pressure (DBP) were recorded. A 12-lead ECG was performed after 10 minutes of rest, and PR interval was measured in milliseconds. Data were expressed as mean ± standard deviation and analyzed using an unpaired t-test. Results showed significant increases in BMI, BSA, WHR, SBP, and DBP in the obese group compared to controls. Notably, the PR interval was prolonged in the obese group and demonstrated high statistical significance ($P < 0.001$). These findings suggest that asymptomatic obesity is associated with prolonged PR intervals, indicative of conduction abnormalities that may contribute to heightened cardiovascular risk. In conclusion, routine monitoring of PR interval and anthropometric indices in obese young adults is essential to mitigate long-term cardiovascular risks and prevent potential conduction-related complications.

Keywords: Electrocardiogram; PR interval; Obesity; Body Mass Index.

INTRODUCTION

Obesity is a significant global public health challenge that is closely linked to cardiovascular comorbidities and an increased risk of sudden cardiac death. This epidemic affects both developed and developing countries, highlighting the urgent need for early detection of subclinical cardiovascular changes. The rising prevalence of obesity, particularly among young adults, necessitates the immediate implementation of preventive strategies.

Electrocardiography (ECG) is a non-invasive and cost-effective diagnostic tool widely used to assess cardiovascular abnormalities. Obesity can lead to specific ECG changes, including leftward shifts in the axes of the P-wave, QRS, and T-wave, alterations in P-wave morphology, low QRS voltage, and a higher prevalence of conduction delays.[1] Among these changes, the

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PR interval, which measures atrioventricular conduction, is particularly noteworthy. It serves as a key indicator of subtle cardiac electrical remodeling, even in asymptomatic individuals. A deviation from the normal range of the PR interval (120–200 ms) may signal potential cardiac issues. However, the variations of the PR interval in obese individuals, especially in asymptomatic young adults who are often overlooked in cardiovascular risk assessments, are not well understood.

Factors such as increased adipose tissue, altered autonomic tone, and systemic inflammation could influence cardiac conduction in obesity. There has been considerable debate regarding the nature and extent of this relationship, as reflected in cardiology and electrocardiography textbooks and various journal reports.[2] Some of this controversy may stem from improperly categorizing patients as obese or non-obese without considering the degree or severity of obesity, as well as potentially confounding factors such as age, sex, and blood pressure.

Existing literature predominantly focuses on clinical obesity, with limited emphasis on the anthropometric correlations with PR interval changes in this population. This study aims to address this gap by examining PR interval variations in asymptomatic obese young adults through a comparative approach. By concentrating on this demographic, the research seeks to enhance our understanding of the early electrophysiological changes associated with obesity and their potential as markers for future cardiovascular risk.

MATERIAL AND METHOD

This cross-sectional comparative study included 150 asymptomatic obese young adults aged 18–39 with a body mass index (BMI) ≥ 30 kg/m². Age and sex-matched 150 healthy individuals with a BMI < 22 kg/m² were recruited from the general population as controls. We adhered to standardized protocols for anthropometric and physiological measurements, ensuring methodological rigor. Body weight was recorded in kilograms and height in meters, with BMI calculated as weight in kilograms divided by the square of height in meters. Additional indices such as body surface area (BSA) and waist-to-hip ratio (WHR) were derived using established equations.

Pulse rate, systolic blood pressure (SBP), and diastolic blood pressure (DBP) were measured using validated digital sphygmomanometers, ensuring the reliability of our data. A 12-lead electrocardiogram (ECG) was recorded for all participants after 10 minutes of rest in a supine position. The PR interval, expressed in milliseconds, was calculated from the ECG tracings. Participants with pre-existing cardiac conditions, medication affecting cardiac conduction, or metabolic syndromes were excluded.

Data were expressed as mean ± standard deviation (SD). The normality of the data distribution was assessed using the Shapiro-Wilk test. An unpaired Student's t-test was used for group comparisons of the PR interval, anthropometric indices, and hemodynamic parameters, assuming equal variances. Nonparametric alternatives, such as the Mann-Whitney U test, were employed for variables not meeting the normality assumption. A p-value < 0.001 was considered statistically significant.

The sample size was calculated to ensure a power of 80% and an alpha error of 0.05 based on prior studies evaluating ECG parameters in obese individuals. Statistical analyses were performed using SPSS software (version 26.0), ensuring robust data handling and interpretation. The study was conducted with the utmost ethical considerations, with approval obtained from the institutional review board, and informed consent secured from all participants before enrolment.

RESULTS

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The results of the study highlight significant differences in anthropometric, hemodynamic, and electrophysiological parameters between asymptomatic obese young adults and their non-obese counterparts.

The mean BMI of the obese group (33.6 ± 3.5 kg/m²) was significantly higher than that of the non-obese group (21.6 ± 3.3 kg/m²) ($p < 0.001$). This reflects the distinct separation of the groups based on body fat percentage and weight status, as defined by the study inclusion criteria.

The mean BSA was higher in the obese group (1.60 ± 0.12 m²) compared to the non-obese group (1.49 ± 0.10 m²) ($p < 0.001$). This indicates that obese individuals generally have a larger surface area, a parameter relevant to cardiac workload and metabolism.

WHR was slightly but significantly higher in the obese group (0.96 ± 0.02) compared to the non-obese group (0.95 ± 0.02) ($p < 0.001$). Although the difference appears minor, it may indicate a more excellent central fat distribution in obese individuals linked to cardiometabolic risk.

The mean SBP was significantly elevated in the obese group (122.3 ± 4.4 mmHg) compared to the non-obese group (120.9 ± 4.3 mmHg) ($p < 0.001$). This suggests increased vascular resistance and cardiac workload in obesity, even in asymptomatic individuals. Similar to SBP, the obese group exhibited higher DBP (83.9 ± 6.7 mmHg) compared to the non-obese group (81.74 ± 5.1 mmHg) ($p < 0.001$). This finding further supports subtle cardiovascular changes associated with obesity.

Table 1: Comparison of Anthropometric, hemodynamic and electrophysiological findings among asymptomatic obese young and non obese subjects

Parameters	Obese	Non-Obese	P-Value
BMI (kg/m^2)	33.6 ± 3.5	21.6 3.3	< 0.001*
BSA (sq. m)	1.60 0.12	1.49 0.10	< 0.001*
WHR	0.96 0.02	0.95 0.02	< 0.001*
SBP (mm Hg)	122.3 4.4	120.9 4.3	< 0.001*
DBP (mm Hg)	83.9 6.7	81.7 5.1	< 0.001*
PR interval	140.6 17.8	135.1 17.7	< 0.001*

(* p value highly significant)

The PR interval was significantly prolonged in the obese group (0.210 ± 17.8 seconds) compared to the non-obese group (0.135 ± 17.7 seconds) ($p < 0.001$). This indicates a delay in atrioventricular conduction in obese individuals, suggesting early subclinical electrical remodeling of the heart.

DISCUSSION

The findings of this study highlight significant differences in anthropometric, hemodynamic, and electrophysiological parameters between asymptomatic obese young adults and non-obese controls. One of the key observations was the prolonged PR interval in the obese group, which aligns with existing evidence indicating a relationship between obesity and delayed atrioventricular conduction.

The prolonged PR interval in obese individuals observed in our study is consistent with prior findings by Frank *et al.*[3] and Alpert *et al.* [4], who reported a progressive increase in PR interval duration with increasing obesity severity. Notably, this prolongation was independent of age, sex, and blood pressure. Additionally, Alpert *et al.* demonstrated that a 10% increase in obesity was associated with a 0.5 ms increase in PR interval duration, indicating a dose-dependent effect of obesity on atrioventricular conduction delay. Similarly, Pipberger *et al.*[5] noted slight

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but statistically significant increases in PR interval with increasing weight, further supporting the correlation between excess adiposity and conduction abnormalities.

Prolonged PR interval has significant clinical implications, as demonstrated in the Framingham Heart Study, a long-term, ongoing cardiovascular cohort study[6] [7], where it was associated with an increased risk of heart failure, atrial fibrillation, pacemaker implantation, and all-cause mortality. These findings underscore the subclinical cardiovascular risk posed by even modest changes in conduction times, particularly in the setting of obesity. Additionally, prolonged PR interval has been linked to endothelial dysfunction and activation of vascular repair mechanisms, which are thought to contribute to adverse cardiovascular outcomes.[8]

The observed prolongation of the PR interval in asymptomatic obese young adults may reflect early electrophysiological remodeling secondary to obesity. This remodeling could result from mechanical stress, systemic inflammation, and metabolic changes, including insulin resistance and adipokine dysregulation. Given the association of prolonged PR interval with adverse cardiovascular outcomes, these findings emphasize the need for early monitoring and targeted interventions in the obese population, even in the absence of overt clinical symptoms.

In conclusion, the prolonged PR interval observed in our study reaffirms the impact of obesity on cardiac conduction. It highlights the need for vigilance in identifying and managing subclinical cardiovascular risks in this population. Further longitudinal studies are warranted to explore the causal mechanisms and assess the prognostic significance of these findings.

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