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Original Article

Correlation of P-wave dispersion with insulin sensitivity in obese adolescents

Ahmet Sert,[1] Eyup Aslan,[2] Muammer Buyukınan,[3] Ozgur Pirgon[4]

[1]*Department of Paediatric Cardiology, Konya Training and Research Hospital, Konya;* [2]*Department of Paediatric Cardiology, Denizli State Hospital, Denizli;* [3]*Department of Paediatric Endocrinology, Konya Training and Research Hospital, Konya;* [4]*Department of Paediatric Endocrinology, Faculty of Medicine, Suleyman Demirel University, Isparta, Turkey*

Abstract *Background:* P-wave dispersion is a new and simple electrocardiographic marker that has been reported to be associated with inhomogeneous and discontinuous propagation of sinus impulses. In the present study, we evaluated P-wave dispersion in obese adolescents and investigated the relationship between P-wave dispersion, cardiovascular risk factors, and echocardiographic parameters. *Methods:* We carried out a case–control study comparing 150 obese adolescents and 50 healthy controls. Maximum and minimum P-wave durations were measured using a 12-lead surface electrocardiogram, and P-wave dispersion was calculated as the difference between these two measures. Echocardiographic examination was also performed for each subject. Multivariate linear regression analysis with stepwise variable selection was used to evaluate parameters associated with increased P-wave dispersion in obese subjects. *Results:* Maximum P-wave duration and P-wave dispersion were significantly higher in obese adolescents than control subjects (143 ± 19 ms versus 117 ± 20 ms and 49 ± 15 ms versus 29 ± 9 ms, $p < 0.0001$ for both). P-wave dispersion was positively correlated with body mass index, waist and hip circumferences, systolic and diastolic blood pressures, total cholesterol, serum levels of low-density lipoprotein cholesterol, triglycerides, glucose, and insulin, homoeostasis model assessment for insulin resistance score, left ventricular mass, and left atrial dimension. P-wave dispersion was negatively correlated with high-density lipoprotein cholesterol levels. By multiple stepwise regression analysis, left atrial dimension (β: 0.252, $p = 0.008$) and homoeostasis model assessment for insulin resistance (β: 0.205; $p = 0.009$) were independently associated with increased P-wave dispersion in obese adolescents. *Conclusions:* Insulin resistance is a significant, independent predictor of P-wave dispersion in obese adolescents.

Keywords: Adolescent; electrocardiogram; obesity; P-wave dispersion

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Obesity is an important and increasingly prevalent public health problem in children and adolescents worldwide. Several studies have shown that obese children present with cardiometabolic risk factors such as high blood pressure, dyslipidaemia, and liver steatosis.[1,2] Obesity in childhood and adolescence has been associated with adverse changes in cardiac geometry, including increased left ventricular mass and left atrial dimension.[3,4] Abnormal left ventricular mass and geometry are well-established risk factors for cardiovascular events.[5] Increased left ventricular mass, hypertension, insulin resistance, and secondary hyperinsulinaemia and dyslipidaemia are recognised cardiovascular complications of obesity.[6] In children with hypertension, excess body weight has been associated with left atrial size, independently of blood pressure,[7] suggesting a potential role of other obesity-related mechanisms including hyperinsulinaemia/insulin resistance.[8] This predisposition to the

Correspondence to: A. Sert, Department of Paediatric Cardiology, Konya Training and Research Hospital, 42080 Konya, Turkey. Tel: +90 332 323 6709; Fax: +90 332 323 6723; E-mail: ahmetsert2@hotmail.com

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development of subclinical cardiovascular alterations early in life is believed to increase cardiovascular morbidity and mortality in adulthood.[9]

P-wave dispersion is a new and simple electrocardiographic marker that has been reported to be associated with inhomogeneous and discontinuous propagation of sinus impulses.[10,11] P-wave duration and P-wave dispersion are the most important non-invasive electrocardiogram markers that have been introduced to assess atrial arrhythmia risk in patients.[11] Previously, P-wave dispersion has been studied in hypertension, anorexia nervosa, asthma, acute rheumatic fever, mitral valve prolapse, and diabetes mellitus in childhood.[12–17]

Several studies have demonstrated that P-wave dispersion values are higher in obese adults.[18–20] At present, the extent of P-wave dispersion in obese adolescents has not been studied. In the present study, we aimed to evaluate P-wave dispersion in obese adolescents and investigate the relationship between P-wave dispersion, cardiovascular risk factors, and echocardiographic variables.

Materials and methods

Study population

In all, 150 obese adolescents (89 girls and 61 boys, mean age: 13.9 ± 1.5 years, age range 12–17 years) were recruited from a subset of obese adolescents admitted to our Paediatric Endocrinology Unit between February, 2013 and July, 2013. Controls (26 girls and 24 boys, mean age: 14 ± 1.3 years, age range 12–17) were selected from non-obese healthy adolescents.

Anthropometric measurements were performed in all patients. Height and weight were measured with an empty bladder in post-absorptive conditions. Height was measured to the nearest 0.5 cm using a standard height board, and weight was determined to the nearest 0.1 kg using a standard physician's beam scale, with the subject dressed only in light underwear without shoes. Body mass index was calculated as weight (in kilograms) divided by height (in metres) squared. Patients with a body mass index $\geqslant$95th percentile according to reference curves for Turkish adolescents were considered as obese.[21] Waist circumference was measured midway between the lowest rib and the top of the iliac crest at the end of gentle expiration. Hip circumference was measured over the great trochanters. Circumferences were measured over the naked skin and noted to the nearest 0.1 cm. Waist/hip ratio was calculated as the ratio of waist and hip circumference measurements.[22] Pubertal development stage was assessed by a single paediatric endocrinologist using Tanner criteria. Staging for sexual maturation was >2 in all patients (Tanner stages II–IV). After resting for at least 5 minutes, systolic and diastolic blood pressures were measured in a sitting position, using a mercury-gravity manometer and a cuff appropriate for body size.

Patients were excluded if they had any systemic disease including type I or type II diabetes mellitus, if they were taking medications, or had a condition known to affect insulin action or insulin secretion – for example, glucocorticoid therapy, hypothyroidism, and Cushing's disease. None of the patients had an accompanying disease, and none were taking drugs known to influence heart rhythm or electrocardiogram results. None of the patients had electrolyte abnormalities. The study protocols were approved by our hospital's Ethics Committee. Signed informed consent forms were obtained from the parents of the adolescents.

Laboratory analyses

Fasting blood samples (at 8:00 am) were obtained to measure serum glucose concentrations, insulin levels, and other parameters in the morning by venepuncture after an overnight fast (at least 12 hours). Glucose level was determined by the hexokinase method. Serum concentrations of total cholesterol, high-density lipoprotein cholesterol, and triglycerides were measured by routine enzymatic methods using an Abbott diagnostics C16000 chemistry analyser (Abbott Diagnostics, Lake Forest, Illinois, United States of America). The value of low-density lipoprotein cholesterol was calculated using Friedwald's equation. Serum insulin levels were measured by Immulite immunoassay (Siemens Healthcare Diagnostics, Camberley, United Kingdom).

Insulin resistance was estimated using the homoeostasis model assessment for insulin resistance: fasting insulin concentration (μU/ml) $\times$ fasting glucose concentration (mmol/L)/22.5.[23]

Electrocardiogram analysis

A 12-lead electrocardiogram with a paper speed of 50 mm/second and 1 mV/cm standardisation was obtained on admission (Nihon Kohden electrocardiogram, Cardiofax GEM, Model 9022K, Tokyo, Japan). Measurements of P-wave duration were performed manually by two investigators blinded to the clinical status of the patients and controls. For greater accuracy, all measurements were performed with callipers and magnifying lenses. All the measurements were repeated twice and average values were recorded. The onset of the P-wave was defined as the

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junction between the isoelectric line and the beginning of the P-wave deflection, and the offset of the P-wave was defined as the junction between the end of the P-wave deflection and the isoelectric line. Heart rate and maximum and minimum P-wave durations were measured from the 12-lead surface electrocardiogram. P-wave dispersion was calculated as the difference between maximum and minimum P-wave duration.[12] An acceptable electrocardiogram was defined by the ability to measure P-wave duration in at least eight of the 12 electrocardiographic leads that were recording simultaneously. All the patients and controls were in normal sinus rhythm during the examination. The intra-observer and inter-observer variations for all measurements were <5%, and therefore insignificant.

Transthoracic echocardiographic examination

All echocardiographic and Doppler assessments were performed by a single, expert paediatric cardiologist, who was blinded to the clinical and laboratory results of the study group. The ProSound Alpha 7 echocardiography equipment (Aloka, Hitachi-Aloka Medical, Tokyo, Japan) with a 3 MHz phased-array transducer was used for each subject. Echocardiographic evaluation from the parasternal long-axis view included left ventricular end-diastolic and end-systolic diameters, septum and left ventricular posterior wall thicknesses in diastole and systole, and left ventricular ejection fraction and fractional shortening. Teichholz's M-mode formula was used to calculate left ventricular ejection fraction and fractional shortening. All data were obtained according to the recommendations of the American Society of Echocardiography.[24]

Left atrial dimension was measured from the parasternal long-axis window in M-mode echocardiography. Left ventricular mass was calculated using the following formula: 0.8{1.04[(left ventricular end-diastolic diameter + left ventricular posterior wall thickness + interventricular septal thickness)^3 – left ventricular end-diastolic diameter^3]} + 0.6 and indexed for height$^{2.7}$.[25] Transmittal flow patterns were obtained by pulsed wave-Doppler echocardiography from an apical four-chamber view. Peak early (E) and late (A) diastolic velocities and E/A ratios were measured.

Statistical analysis

Mean and standard deviations were used as descriptive statistics. Differences in the means of variables were tested using both parametric and non-parametric statistical analyses depending on the distribution of the variables. Correlation was analysed using Pearson's correlation coefficient (r). Stepwise multivariate linear regression analysis was then used to determine parameters that independently explained a significant ($p < 0.05$) fraction of the variance of the dependent variables. A value of $p < 0.05$ was used to indicate statistical significance. Statistical analyses were performed with SPSS for Windows, version 15 (SPSS, Chicago, Illinois, United States of America).

Results

The characteristics of the patients and healthy controls are displayed in Table 1. Both groups were similar with respect to age and sex. The obese group had significantly higher body mass index ($30.7 \pm 3.3\,kg/m^2$ versus $20.1 \pm 1.8\,kg/m^2$, $p < 0.0001$) as well as waist and hip circumferences than the control group. The obese group also had significantly higher total cholesterol, triglycerides, low-density lipoprotein cholesterol, fasting glucose and insulin levels (20 ± 14.6 U/ml versus 7.1 ± 2.4 U/ml, $p < 0.0001$), and homoeostasis model assessment for insulin resistance score (4.66 ± 4.22 versus 1.44 ± 0.49, $p < 0.0001$) and lower high-density lipoprotein cholesterol levels than the control group. Echocardiographic findings of the patients and controls are summarised in Table 2. The obese group had significantly higher left atrial dimension and aortic diameter than the control group. Left ventricular M-mode measurements were significantly higher in the obese group when compared with controls. Measured by echocardiography, left ventricular ejection and shortening fractions were significantly lower in the obese group than the control group. The obese group had significantly lower mitral E-wave and higher mitral A-wave measurements, and consequently lower mitral E/A ratios, compared with controls. The obese group had significantly higher left ventricular mass and left ventricular mass index when compared with control group.

Maximum P-wave durations and P-wave dispersion values were considerably increased in the obese group when compared with the controls (143 ± 19 ms versus 117 ± 20 ms and 49 ± 15 ms versus 29 ± 9 ms, $p < 0.0001$ for both, respectively) (Table 3) (Fig 1).

In the obese group, P-wave dispersion was positively correlated with body mass index, waist and hip circumferences, systolic and diastolic blood pressures, total cholesterol, low-density lipoprotein cholesterol, triglycerides, glucose and insulin levels, homoeostasis model assessment for insulin resistance score, left ventricular mass, left atrial dimension, and aortic diameter and was negatively correlated with high-density lipoprotein cholesterol (Table 4).

To evaluate variables that independently influenced P-wave dispersion, a multiple regression analysis model was performed in the obese group.

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Table 1. Clinical features of controls and obese adolescents.

Variables	Controls	Obese adolescents	p value
(Girls/boys)	50 (26/24)	150 (89/61)	0.364
Age (years)	14 ± 1.3 (12–17)	13.9 ± 1.5 (12–17)	0.824
Body mass index (kg/m^2)	20.1 ± 1.8 (17.5–23.9)	30.7 ± 3.3 (25.1–39.5)	**<0.0001**
Waist circumference (cm)	72 ± 7 (56–88)	97 ± 11 (68–132)	**<0.0001**
Hip circumference (cm)	88 ± 7 (70–106)	108 ± 10 (70–137)	**<0.0001**
Waist/hip circumference ratio	0.81 ± 0.06 (0.67–0.99)	0.89 ± 0.10 (0.58–0.92)	**<0.0001**
Systolic blood pressure (mmHg)	104 ± 7 (80–120)	119 ± 12 (100–150)	**<0.0001**
Diastolic blood pressure (mmHg)	64 ± 6 (50–75)	76 ± 8 (60–100)	**<0.0001**
Total cholesterol (mg/dl)	136 ± 23 (104–178)	172 ± 27 (104–269)	**<0.0001**
Triglycerides (mg/dl)	75 ± 28 (34–144)	130 ± 56 (39–398)	**<0.0001**
Low-density lipoprotein cholesterol (mg/dl)	69 ± 22 (38–106)	104 ± 24 (38–171)	**<0.0001**
High-density lipoprotein cholesterol (mg/dl)	53 ± 7 (41–71)	41 ± 8 (27–69)	**<0.0001**
Fasting glucose (mg/dl)	82 ± 5 (70–92)	91 ± 8 (77–125)	**<0.0001**
Fasting insulin (U/ml)	7.1 ± 2.4 (2–14)	20 ± 14.6 (3–102)	**<0.0001**
Homoeostasis model assessment for insulin resistance score	1.44 ± 0.49 (0.47–2.69)	4.66 ± 4.22 (0.69–36)	**<0.0001**

Values are expressed as mean ± SD with range given in parentheses
Significant p values are shown in bold

Table 2. Echocardiographic findings of controls and obese adolescents.

	Controls (n = 50)	Obese adolescents (n = 150)	p value
M-mode measurements			
Left ventricular end-diastolic diameter (mm)	42.4 ± 3.2 (34–49)	45 ± 4 (36–55)	**<0.0001**
Left ventricular end-systolic diameter (mm)	26.1 ± 2.6 (20–32)	28.3 ± 4.5 (18–40)	**0.002**
Interventricular septum diameter in diastole (mm)	7.2 ± 0.5 (6–9)	8.9 ± 1.4 (6–14)	**<0.0001**
Interventricular septum diameter in systole (mm)	8.4 ± 0.9 (7–10)	9.9 ± 1.5 (7–14)	**<0.0001**
Left ventricular posterior wall diameter in diastole (mm)	8.8 ± 0.9 (4–9)	8.1 ± 1 (6–12)	**<0.0001**
Left ventricular posterior wall diameter in systole (mm)	10.6 ± 1 (8–13)	12.4 ± 2.6 (8–14)	**<0.0001**
Ejection fraction (%)	68.6 ± 4.7 (61–79)	66.9 ± 4.2 (60–83)	**0.019**
Fractional shortening (%)	38.3 ± 4.2 (32–48)	36.7 ± 4.3 (30–51)	**<0.0001**
Left ventricular mass (g)	98 ± 19 (49–141)	136 ± 37 (75–234)	**<0.0001**
Left ventricular mass index (g/m$^{2.7}$)	29 ± 4.1 (16–37)	37.9 ± 9.5 (23–76)	**<0.0001**
Left atrial dimension (mm)	24.1 ± 3 (20–37)	29.8 ± 4.2 (21–41)	**<0.0001**
Aortic diameter (mm)	19.5 ± 2 (16–25)	21.8 ± 2.9 (17–29)	**<0.0001**
Doppler diastolic indices			
Peak early-diastolic wave (m/s)	0.95 ± 0.12 (0.7–1.2)	0.92 ± 0.14 (0.57–1.38)	0.153
Peak late-diastolic wave (m/s)	0.52 ± 0.12 (0.35–0.83)	0.53 ± 0.11 (0.29–0.96)	0.653
Peak early-diastolic wave/peak late-diastolic wave ratio	1.87 ± 0.34 (0.91–2.84)	1.76 ± 0.29 (1.18–2.87)	**0.032**

Values are expressed as mean ± SD with range given in parentheses
Significant p values are shown in bold

Model covariates included body mass index, waist and hip circumferences, systolic and diastolic blood pressures, homoeostasis model assessment for insulin resistance, left ventricular mass, and mitral E/A ratio. After covariate adjustment, variables independently associated with P-wave dispersion included left atrial dimension (β: 0.252, p = 0.008) and homoeostasis model assessment for insulin resistance score (β: 0.205; p = 0.009).

Discussion

In the present study, maximum P-wave duration and P-wave dispersion were detected to be significantly higher in obese adolescents compared with controls. Furthermore, left atrial dimension and homoeostasis model assessment for insulin resistance score were found to be independent parameters associated with increased P-wave dispersion in obese adolescents.

It is recognised that increased P-wave duration and P-wave dispersion are associated with atrial conduction prolongation, left atrial enlargement, and left atrial hypertension. In addition, the autonomic tone, which induces changes in the velocity of impulse propagation, affects P intervals;[10] however, little information exists concerning P-wave duration and P-wave dispersion in obese subjects. Seyfeli et al[18] showed that obesity caused a significant increase in

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Table 3. Electrocardiographic findings of controls and obese adolescents.

Electrocardiographic measurements	Controls (n = 50)	Obese adolescents (n = 150)	p value
P-wave minimum (ms)	87 ± 18 (60–130)	93 ± 16 (65–135)	**0.03**
P-wave maximum (ms)	117 ± 20 (80–160)	143 ± 19 (100–190)	**<0.0001**
P-wave dispersion (ms)	29 ± 9 (15–45)	49 ± 15 (20–85)	**<0.0001**
Heart rate (beats/minute)	89 ± 13 (63–97)	88 ± 11 (58–99)	0.607

Significant p values are shown in bold

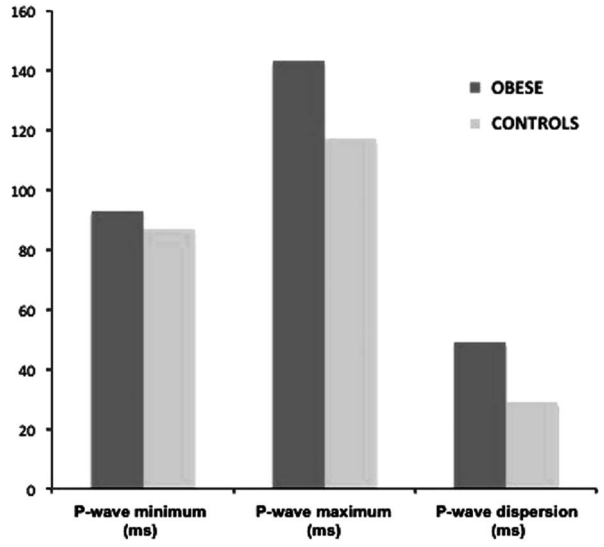


Figure 1.
P-wave measurements in controls and the obese group.

P-wave values, and obese women may be subjected to increased risk of atrial arrhythmias. Liu et al[26] demonstrated that maximum P-wave duration and P-wave dispersion were significantly higher in Chinese obese subjects compared with controls. Our study demonstrated that P-wave dispersion values in obese adolescents are higher than those of controls, which is consistent with the findings of previous studies in adults.[19,26]

Obesity leads to global biatrial endocardial re-modelling, characterised by left atrial enlargement, conduction abnormalities, fractionated electrograms, and interstitial atrial fibrosis.[27] It is well known that adult patients with obesity have a higher prevalence of hypertension, which may lead to left ventricular hypertrophy and left atrial enlargement that may play a role in an alteration of P-wave measurements. In addition, the autonomic control of the heart is abnormal in obese subjects due to a prevalence of sympathetic over parasympathetic limb of the autonomic balance. Therefore, the autonomic imbalance observed in obese subjects may affect intra-atrial and inter-atrial conduction times, and leave them predisposed to developing atrial arrhythmias.[27] The diastolic dysfunction in obese subjects leads to an increase in left atrial pressure and tension in atrial tissue. In the present study, the obese group had a larger left atrial dimension, which is suggestive of early adverse cardiac modelling. In addition, we found that the E/A ratio was lower in the obese group, indicating diastolic dysfunction in obese patients. We demonstrated that P-wave dispersion had a significant correlation with left ventricular mass and left atrial dimension. These results suggest that subclinical left ventricular diastolic dysfunction and left atrial alterations in obese adolescents may contribute to the prolongation of P-wave.

In obese children, insulin resistance is also a key link between excess body weight and the associated risk of cardiovascular events. This metabolic abnormality is not only a risk factor for future development of type 2 diabetes but it is also associated with the presence of other cardiometabolic risk factors such as increased blood pressure, dyslipidaemia, and liver steatosis.[28]

In previous studies, left ventricular diastolic dysfunction and left ventricular re-modelling in obese adolescents have been shown to be linked with insulin resistance.[29] Increased P-wave dispersion may be prolonged because of atrial tissue damage as a result of insulin resistance in the obese group. Peterson et al[30] showed that insulin resistance produces metabolic and proliferative alterations in cardiomyocytes and the interstitium, leading to functional disturbances. Insulin resistance stimulates myocyte growth and interstitial fibrosis, leading to sodium retention and a subsequent blood volume increase. It also activates the sympathetic nervous system, exerting deleterious effects on cardiac performance and altering myocardial metabolism, including progressive increases in fatty acid turnover.[31,32] A recent study showed a positive correlation between insulin resistance and left atrial dimension in obese children. In this study, Marcovecchio et al[5] concluded that the homoeostasis model assessment for insulin resistance score was independently associated with left atrial diameter, area, and volume.

Consequently, insulin resistance may lead to an increased P-wave dispersion by stimulating interstitial fibrosis, worsening diastolic function, and

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Table 4. Pearson's correlations between P-wave dispersion and other parameters in obese adolescents.

	Obese adolescents	
Variables	**R**	**p value**
Anthropometric parameters		
Body mass index (kg/m^2)	0.340	**<0.0001**
Waist circumference (cm)	0.300	**<0.0001**
Hip circumference (cm)	0.320	**<0.0001**
Systolic blood pressure (mmHg)	0.228	**0.005**
Diastolic blood pressure (mmHg)	0.256	**0.002**
Metabolic parameters		
Total cholesterol (mg/dl)	0.335	**<0.0001**
Triglycerides (mg/dl)	0.347	**<0.0001**
Low-density lipoprotein cholesterol (mg/dl)	0.336	**<0.0001**
High-density lipoprotein cholesterol (mg/dl)	−0.277	**0.001**
Fasting glucose (mg/dl)	0.201	**0.014**
Fasting insulin (U/ml)	0.331	**<0.0001**
Homoeostasis model assessment for insulin resistance score	0.309	**<0.0001**
Electrocardiographic parameters		
P-wave minimum	−0.246	**0.002**
P-wave maximum	0.561	**<0.0001**
Echocardiographic parameters		
Left ventricular mass (g)	0.250	**0.002**
Left atrial dimension (mm)	0.359	**<0.0001**
Aortic diameter (mm)	0.255	**0.002**

Significant p values are shown in bold

increasing atrial size. In the present study, we found that the homoeostasis model assessment for insulin resistance values were higher in the obese group, and these increased levels in the obese group were linked with P-wave dispersion. Furthermore, the obese group had higher left ventricular mass and larger left atrial dimension, which are suggestive of early adverse cardiac modelling. We demonstrated that insulin resistance is linked with P-wave dispersion. In addition, there were positive correlations between P-wave dispersion and cardiovascular risk factors and echocardiographic variables in obese adolescents. Our study demonstrated that the homoeostasis model assessment for insulin resistance was an independent predictor of P-wave dispersion in the obese group. Another recent study has also shown that insulin resistance in obese adults is associated with an increased P-wave dispersion.[33] In our study, inclusion of all metabolic and echocardiographic variables in a multivariate regression analysis showed that left atrial dimension and homoeostasis model assessment for insulin resistance score were independent parameters associated with increased P-wave dispersion in obese adolescents. We demonstrated that the presence of insulin resistance may have an additional increasing impact on P-wave dispersion in obese adolescents, which is equivalent to the results of previous publications.[33,34]

Nevertheless, the present study has some limitations. It is a cross-sectional study, and there was no long-term follow-up about whether these factors increase the risk of atrial arrhythmia in obese adolescents. Some further limitations include the fact that the subjects could not be observed prospectively for atrial arrhythmic events, and a 24-hour electrocardiographic examination was not performed to study the presence of atrial arrhythmia in our study population. In addition, we did not use the automatic algorithms for P-wave reconstruction.

Conclusions

It was found that obese adolescents had higher P-wave dispersion values compared with controls. The present study revealed that insulin resistance has a significant, independent impact on P-wave dispersion in obese adolescents; however, long-term follow-up prospective studies are necessary to clarify the potential clinical use of P-wave dispersion in obese adolescents.

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Conflicts of Interest

None.

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Ethical Standards

This study was approved by the local ethics committee of our institution, and parents of the participants provided their written informed consent.

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