DOI: 10.4081/aiua.2025.14073

ORIGINAL PAPER

P-wave dispersion in patients with premature ejaculation

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Summary

***Background:** Premature ejaculation (PE) is one of the common male sexual disorders and is associated with autonomic nervous system imbalance. P-wave dispersion (PWD), a marker of atrial electrical heterogeneity and a predictor of atrial arrhythmias, has not been previously investigated in the PE population. This study aimed to investigate PWD in patients with PE and to evaluate whether the subtypes of PE, namely acquired (APE) and lifelong (LLPE), differ in terms of atrial conduction parameters.*

***Methods:** Seventy-eight male patients were included in the study. The distribution of patients was 40 healthy controls, 21 patients with APE, and 17 patients with LLPE. All patients underwent 12-lead electrocardiography to evaluate PWD. Echocardiographic, hormonal, and biochemical parameters were recorded to exclude confounding factors. PWD was defined as the difference between the maximum (Pmax) and minimum P wave durations (Pmin).*

***Results:** PWD was significantly increased in both PE groups compared to the control group (APE: 44.4 ± 4.1 ms; LLPE: 48.1 ± 2.1 ms; control: 38.2 ± 3.4 ms; $p < 0.001$). Pmin was significantly lower in the PE groups, while Pmax remained comparable among all groups. There was no significant difference in PWD between the PE groups ($p = 0.38$). All patients had normal echocardiographic and hormonal parameters, and there were no significant differences in age, body mass index, blood pressure, or heart rate between the groups.*

***Conclusions:** PWD was significantly higher in patients with PE. This indicates that the patients of PE are at a higher risk of developing cardiovascular diseases like atrial fibrillation.*

KEY WORDS: Premature ejaculation; P-wave dispersion; Atrial fibrillation; Cardiovascular disease; Electrocardiography.

Submitted 12 June 2025; Accepted 10 July 2025

INTRODUCTION

Premature ejaculation (PE) is one of the most common male sexual disorders, affecting approximately 20-30% of men (1, 2). It is typically defined as ejaculation occurring either within one minute of vaginal penetration in lifelong cases (LLPE), or within approximately three minutes in acquired forms (APE) (3, 4). The exact pathogenesis of PE has not yet been fully elucidated. While psychological factors, genetic predisposition, endocrine abnormalities, alterations in penile sensitivity, and chronic prostatitis have all been suggested as potential contributors, definitive evidence linking these conditions to PE remains lacking (4). Studies have indicated that autonomic nervous system dysregulation, particularly heightened sympathetic activity, may play a central role in its underlying mechanisms (5). This hypothesis is supported by heart rate variability analyses in men with PE, which consistently demonstrate increased sympathetic tone alongside diminished parasympathetic activity (6). Given that autonomic imbalance is also a recognized factor in atrial conduction disturbances, its role in PE may extend beyond sexual function, potentially implicating broader cardiovascular implications.

P-wave dispersion (PWD), one of the non-invasive electrocardiographic markers reflecting atrial conduction abnormalities, is defined as the difference between the longest and shortest P wave durations recorded on a standard 12-lead ECG (7). Increased PWD reflects heterogeneity in atrial depolarization and is considered a reliable indicator of atrial structural remodeling (8). Various studies have demonstrated that the duration and dispersion of the P wave are influenced by autonomic nervous system activity (9). Increased PWD has been identified as an independent risk factor for the development of *atrial fibrillation* (AF), the most commonly sustained arrhythmia in the general population. Moreover, increased PWD is associated with a higher incidence of cardiovascular events and overall mortality, contributing to a reduced quality of life (8, 10).

Although several studies have reported increased PWD in various systemic and neuropsychiatric conditions, including lichen planus, acute pancreatitis, psoriasis, schizophrenia, thalassemia, polycystic ovary syndrome, and erectile dysfunction, the association between PWD and PE remains unexplored (11-17). Considering the established role of autonomic nervous system dysregulation in the pathophysiology of PE, it is reasonable to hypothesize that these patients may exhibit altered atrial conduction. Therefore, the present study aimed to evaluate PWD in patients with both LLPE and APE compared to healthy controls, with the hypothesis that increased PWD in the PE group may reflect a greater likelihood of atrial conduction abnormalities, such as AF.

MATERIALS AND METHODS

Study design and participants

This prospective observational study was conducted with 78 male participants aged between 25 and 55 years who visited the outpatient clinic either with or without com-

plaints of PE. Participants were divided into three groups as follows:
Group 1: Patients diagnosed with APE (n = 21)
Group 2: Patients with LLPE (n = 17)
Control group: Age-matched healthy men without any PE symptoms (n = 40).
Participants were recruited consecutively from the urology and cardiology outpatient clinics of our institution. Controls were selected among healthy volunteers attending the clinics for routine health check-ups or minor urological issues not associated with autonomic or cardiovascular conditions.
The diagnosis of PE was made based on detailed clinical history and self-reported symptoms, in line with the guidelines of the *International Society for Sexual Medicine* (ISSM). Individuals with a known history of psychiatric disorders, cardiovascular disease, diabetes mellitus, hypertension, or those on medications that might affect autonomic or cardiac function were excluded from the study. In addition, patients with abnormal thyroid function tests were excluded to eliminate potential confounding effects on both PE and PWD.
All participants provided written informed consent prior to enrolment. The study protocol was reviewed and approved by the *Clinical Research Ethics Committee of Alanya Alaaddin Keykubat University* (Decision No: 16-14, Date: 13/02/2020).

Clinical and laboratory evaluation
Demographic and clinical characteristics, including age, *body mass index* (BMI), *systolic* and *diastolic blood pressure* (SBP and DBP), and resting *heart rate* (HR), were recorded for all participants. Blood samples were collected in the morning following an overnight fast of 8-12 hours. The laboratory tests included fasting glucose, total cholesterol, *low-density lipoprotein* (LDL), triglycerides, creatinine, hemoglobin, prolactin, and total testosterone levels.

Echocardiographic assessment
All subjects underwent transthoracic echocardiography following standardized imaging protocols. The following cardiac parameters were measured: *left ventricular end-diastolic diameter* (LVDD), *left ventricular end-systolic diameter* (LVSD), *interventricular septum thickness* (IVS), *posterior wall* thickness (PW), *left atrial diameter* (LAD), and *ejection fraction* (EF). Diastolic function was assessed using the E/A ratio derived from pulsed-wave Doppler measurements of mitral inflow.

Electrocardiographic measurements
Standard 12-lead *electrocardiograms* (ECGs) were recorded with participants in the supine position after a 10-minute rest. ECGs were obtained at a paper speed of 25 mm/s and amplitude of 10 mm/mV, then digitized for analysis.
The onset of the P-wave was defined as the point where it first deviated from the isoelectric line, and the end was marked by its return. The longest (Pmax) and shortest (Pmin) P-wave durations across all leads were measured using the software Cardio Calipers (*Iconico Inc., New York USA*). PWD was calculated as PWD = Pmax - Pmin.
All ECG measurements were independently analysed by two cardiologists who were blinded to group allocation. Interobserver variability was assessed and found to be within acceptable limits, with a difference of less than 5% between observers.

Statistical analysis
Data were analysed using SPSS version 21.0 (*IBM Corp., Armonk, NY, USA*). Continuous variables were expressed as mean ± *standard deviation* (SD). Comparisons between the three groups were made using *analysis of variance* (ANOVA), followed by Tukey's post-hoc test for multiple comparisons. A p-value less than 0.05 was considered statistically significant.

RESULTS

Demographic and clinical characteristics
A total of 78 male subjects were included: 40 healthy controls, 21 with acquired premature ejaculation (Group 1), and 17 with lifelong premature ejaculation (Group 2). There were no statistically significant differences among the three groups in terms of age, BMI, blood pressure, heart rate, or laboratory values including glucose, lipid profile, creatinine, hemoglobin, testosterone, and prolactin (p > 0.05 for all) (Table 1).

Table 1.
Demographic, clinical, and echocardiographic variables of study group.

Parameter	Control (n = 40)	Group 1 - APE (n = 21)	Group 2 - LPE (n = 17)	P-value
Age (years)	39.6 ± 7.3	38.7 ± 8.5	38.5 ± 8.3	0.69
BMI (kg/m²)	25.9 ± 4.7	24.9 ± 5.7	25.0 ± 5.1	0.51
Glucose (mg/dL)	88.9 ± 7.0	87.4 ± 7.9	86.8 ± 8.2	0.95
Total cholesterol (mg/dL)	193.1 ± 37.7	186.7 ± 33.7	185.2 ± 35.1	0.73
Triglycerides (mg/dL)	176.1 ± 86.7	179.4 ± 79.3	180.1 ± 80.6	0.39
LDL (mg/dL)	117.0 ± 33.5	121.1 ± 28.2	120.5 ± 27.9	0.67
Creatinine (mg/dL)	0.83 ± 0.2	0.92 ± 0.2	0.91 ± 0.3	0.51
Hb (g/dL)	13.5 ± 1.4	14.7 ± 0.9	14.4 ± 1.0	0.49
Prolactin (ng/mL)	10.6 ± 3.4	11.2 ± 3.1	11.3 ± 2.8	0.18
Testosterone (ng/dL)	520.4 ± 85.2	498.7 ± 79.6	501.2 ± 78.3	0.27
SBP (mmHg)	117.8 ± 7.8	121.3 ± 8.9	121.1 ± 9.2	0.27
DBP (mmHg)	78.3 ± 5.6	74.3 ± 4.1	74.1 ± 4.6	0.37
HR (bpm)	76.8 ± 6.5	79.3 ± 6.5	79.7 ± 6.9	0.15
LVDD (cm)	47.7 ± 5.1	48.5 ± 4.4	48.3 ± 4.2	0.26
LVSD (cm)	28.7 ± 3.5	28.7 ± 3.3	28.6 ± 3.4	0.72
EF (%)	64.2 ± 3.4	63.7 ± 5.2	63.5 ± 4.8	0.18
IVS (cm)	9.5 ± 1.1	9.6 ± 0.9	9.5 ± 0.8	0.42
PW (cm)	9.4 ± 1.0	9.2 ± 1.4	9.3 ± 1.1	0.14
LAD (cm)	33.4 ± 3.3	34.1 ± 2.8	34.0 ± 3.0	0.22
E/A ratio	±0.4	± 0.2	± 0.2	0.36

Data are presented as mean ± standard deviation values.
BMI: Body mass index; LDL: Low-density lipoprotein; Hb: Hemoglobin; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; HR: Heart rate; LVDD: Left ventricular diastolic diameter; LVSD: Left ventricular systolic diameter; EF: Ejection fraction; IVS: Interventricular septum; PW: Posterior wall; LAD: Left atrial diameter; E/A: Early-to-late mitral inflow velocity ratio.

Table 2.
Electrocardiographic variables of study group.

Parameter	Control (n = 40)	Group 1 - APE (n = 21)	Group 2 - LPE (n = 17)	P-value
Pmax (ms)	108.4 ± 4.2	107.1 ± 5.1	109.3 ± 3.3	0.47
Pmin (ms)	70.5 ± 3.2	63.8 ± 3.1*	61.4 ± 3.3**, ***	< 0.001
PWD (ms)	38.2 ± 3.4	44.4 ± 4.1*	48.1 ± 2.1**, ***	< 0.001

Data are presented as mean ± standard deviation values.
Pmax: Maximum P-wave duration; Pmin: Minimum P wave duration; PWD: P wave dispersion.
**: Significant difference group 1 vs. Control (p < 0.001).*
***: Significant difference group 2 vs. Control (p < 0.001).*
****: Group 1 vs. Group 2 (p = 0.38).*

Echocardiographic parameters such as LVDD, LVSD, EF, IVS, PW, LAD, and E/A ratio also showed no significant group differences.

Electrocardiographic findings

The Pmax was similar across all groups ($p = 0.47$), while Pmin was significantly reduced in both PE groups compared to controls ($p < 0.001$), being lowest in the LLPE group. Consequently, PWD was significantly increased in Group 1 and especially in Group 2 compared to controls ($p < 0.001$).
Although PWD was numerically higher in Group 2 than Group 1, this difference was not statistically significant ($p = 0.38$) (Table 2).

Discussion

We demonstrated that PWD is significantly increased in men with PE compared to healthy controls. After stratifying the PE cohort into APE and LLPE subtypes, no significant differences were found between these subgroups in demographic, hormonal, or echocardiographic parameters, confirming comparability. Both APE and LLPE groups demonstrated significantly higher PWD values compared to controls, indicating that PE, regardless of subtype, is associated with similar degrees of atrial conduction delay.
This prolongation of PWD was primarily driven by a reduction in Pmin, while the Pmax remained comparable across groups. All participants had similar age, body mass index, blood pressure, and echocardiographic findings, including normal left atrial size and systolic function. Additionally, serum testosterone and prolactin levels, which influence sexual function and autonomic regulation, were statistically similar between groups. These findings indicate that the observed changes in atrial conduction are not attributable to structural heart disease or hormonal imbalances but are more likely related to functional alterations in autonomic control.
PWD is a well-recognized electrocardiographic marker for atrial arrhythmogenic risk. The significant PWD prolongation observed in our PE patients suggests a potential subclinical predisposition to atrial arrhythmias. These findings are consistent with previous studies reporting similar PWD prolongation in disorders associated with autonomic or endothelial dysfunction. For instance, men with ED have been shown to exhibit prolonged PWD compared to healthy individuals, with the increase attributed to a shortened Pmin while Pmax remained unchanged. Moreover, PWD in ED patients was inversely correlated with erectile function scores, implying that more severe dysfunction is associated with greater atrial conduction heterogeneity (17). As ED is a known marker of endothelial and autonomic dysfunction, the parallels with PE support a shared pathophysiological basis.
Prolonged PWD has also been reported in several systemic conditions. Patients with schizophrenia demonstrate significantly higher PWD compared to age-matched controls, despite having no structural heart disease (14). Chronic inflammatory diseases such as psoriasis are similarly associated with prolonged PWD, and values tend to rise with increasing disease severity (13). Women with polycystic ovary syndrome, a condition characterized by endocrine and autonomic imbalance, show higher PWD along with prolonged atrial conduction intervals (16). Furthermore, hypertensive disorders of pregnancy like preeclampsia have been linked to increased PWD, supporting a broader role of systemic disturbances in atrial conduction abnormalities (18).
However, not all conditions with autonomic involvement result in increased PWD. For example, fibromyalgia, despite its association with autonomic dysfunction and cardiovascular symptoms, was not found to significantly differ from controls in terms of PWD (19). This suggests that the link between autonomic imbalance and atrial conduction heterogeneity may depend on disease-specific mechanisms. It is possible that pro-arrhythmic effects of sympathetic overactivity in fibromyalgia are counterbalanced by other regulatory processes, unlike in PE or ED.
Although our study did not directly assess mechanisms, autonomic dysregulation remains the most plausible explanation for the observed findings. Prior work has shown that men with lifelong PE display elevated sympathetic and reduced parasympathetic activity based on heart rate variability analysis, reflecting increased sympathovagal balance (5). Elevated sympathetic tone and circulating catecholamines are known to alter atrial electrophysiology by shortening refractory periods and increasing conduction velocity, thereby promoting atrial conduction heterogeneity.
Autonomic influences have been shown to modulate P-wave indices, with sympathetic activation typically associated with PWD prolongation (20). In PE patients, chronic stress, anxiety, or serotonergic dysfunction may further exacerbate this imbalance. Given that ejaculation is primarily under sympathetic control, a predisposition to PE might reflect a generalized sympathetic dominance. The comparable extent of PWD prolongation in both APE and LLPE supports the idea that once established, PE may lead to persistent autonomic alterations and cardiac electrical remodelling regardless of onset type.
Clinically, the increased PWD values in young, otherwise healthy men with PE are noteworthy. A PWD threshold above 40 ms is commonly considered predictive of AF (10). In our PE cohort, average PWD values exceeded this threshold, while control values remained within the upper normal limit. Although no arrhythmic events were documented, this finding suggests a latent electrical vulnerability that may have long-term relevance.

Whether this predisposition translates into clinical arrhythmias over time remains uncertain. If sustained, autonomic imbalance could contribute to atrial remodelling. Our findings underscore that PE, typically viewed as a psychosexual disorder, may also carry electrophysiological relevance. Recognizing this association can support a more holistic clinical approach that includes evaluation of autonomic and cardiac function in men presenting with PE.

This study has several limitations that should be acknowledged. First, the sample size of the PE subgroups was relatively modest. However, it was sufficient to detect statistically significant differences in PWD compared to controls, suggesting that the effect size was strong enough to demonstrate the relationship between PE and atrial conduction. Nonetheless, larger cohorts are warranted to confirm our findings and to better clarify whether subtle distinctions exist between PE subtypes. Moreover, although the study groups were well matched for potential confounders, future studies with larger samples would benefit from multivariate analyses to further adjust for unmeasured variables.

Second, the cross-sectional design of the study limits causal inference. While we observed a significant association between PE and increased PWD, the temporal sequence or directionality of this relationship cannot be determined.

Finally, although autonomic nervous system involvement is strongly supported by previous literature and aligns with our clinical rationale, we did not directly evaluate autonomic function using objective physiological measurements.

Despite these limitations, our study provides novel evidence that premature ejaculation, a common psychosexual disorder, may be associated with subclinical alterations in atrial conduction. These findings emphasize the potential value of incorporating basic electrocardiographic assessment into the broader evaluation of sexual dysfunction and highlight the need to consider cardiovascular dynamics in patients presenting with PE.

Conclusions

This study is the first to demonstrate a significant increase in PWD among patients with PE, including both APE and LLPE subtypes. These findings suggest that PE is associated with increased atrial conduction heterogeneity, most likely related to autonomic dysregulation rather than structural cardiac abnormalities or traditional cardiovascular risk factors.

Although no arrhythmic events were detected in our cohort, the elevated PWD values, frequently exceeding thresholds predictive of AF, may reflect a latent electrophysiological susceptibility. Recognizing PE as a condition with potential cardiovascular relevance could help promote more integrative clinical assessment strategies. Larger-scale studies are needed to determine whether addressing PE or its autonomic underpinnings can mitigate future cardiac risk.

Declarations

Ethical approval and consent for participate: *This study was approved by the Alanya Alaaddin Keykubat University Faculty of Medicine Clinical Research Ethics Committee (ALKÜ-KAEK), approval number: 16-14, dated 13/02/2020.*

Consent for publication: *Not applicable.*

Availability of data and material: *The datasets used and/or analysed during the current study are not publicly available but are available from the corresponding author on reasonable request.*

Competing interests: *The authors declare that they have no competing interests.*

Funding: *The authors report no funding.*

Authors' contributions: *AA: study concept, data collection and analysis related to premature ejaculation; CRÖ and CK: interpretation of cardiological parameters; AA, CRÖ and CK: contributed equally to manuscript drafting and revision. All authors read and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.*

Acknowledgments: *Not applicable.*

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