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Original Article

P-Wave Dispersion in Acute Pancreatitis

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ABSTRACT

Objective: Cardiac complications and electrocardiographic anomalies are frequently seen in acute pancreatitis (AP). Also, an increased P-wave dispersion (PWD) is accepted as a predictor of atrial fibrillation. It was aimed to investigate the P-wave dispersion change in acute pancreatitis and to predict the development of atrial fibrillation.

Methods: Patients diagnosed with acute pancreatitis, treated, and followed up between July 2018 and July 2019 were prospectively evaluated. The diagnosis of acute pancreatitis was based on physical examination and laboratory and imaging methods. The study included patients aged 18-60 who agreed to participate, were diagnosed, treated, and followed up in our hospital, and achieved complete recovery according to the second-month follow-up. The maximum and minimum P-wave durations were measured in milliseconds (ms) using a 12-lead electrocardiography device at the time of the diagnosis of acute pancreatitis and the second month after treatment. The difference between the maximum and minimum values was calculated as PWD. The PWD values were compared between the 2 evaluation times.

Results: The mean age of the patients was 43.2 ± 12.5 (range, 19-60) years. The follow-up period was 2 months. Twenty-six (52%) patients were male and 24 (48%) were female. The maximum P-wave duration and PWD were determined as 98.7 ± 11.4 (60-120) ms and 41.5 ± 9.1 (30-60) ms, respectively, during the acute pancreatitis attack, and 94.1 ± 11.5 (60-110) ms and 37.5 ± 9.3 (20-60) ms, respectively, during the second-month follow-up. The maximum P-wave duration and PWD values were significantly higher during the acute pancreatitis attack compared to those measured after recovery (P = .020 and P = .003, respectively).

Conclusion: In patients with acute pancreatitis, PWD was significantly prolonged compared to post-treatment. We consider that patients with acute pancreatitis have a higher probability of atrial arrhythmia due to prolonged PWD; therefore, appropriate follow-up should be undertaken and preventive measures should be applied.

Keywords: Acute pancreatitis, electrocardiogram, P-wave dispersion, atrial arrhythmia

INTRODUCTION

Acute pancreatitis (AP) is a clinical picture characterized by damage caused by enzymes in the acinar cells of the exocrine pancreas and secondary inflammation. It is characterized by local and systemic responses to inflammation. Acute pancreatitis should be considered in every patient presenting with abdominal pain. Diagnostic criteria are abdominal pain accompanied by an increase in serum amylase-lipase levels 3 times higher than normal and the presence of accompanying imaging findings.[1] Complications of AP can be localized and generalized. Localized complications include fluid accumulations, pseudocyst, acute necrotic collection, necrosis-walled off pancreatic necrosis, venous thrombosis, pseudoaneurysm, and hemorrhage. During the course of AP, problems affecting almost all systems may occur. Systemic complications such as pulmonary, cardiovascular, hematological, renal, metabolic, and central nervous system anomalies may occur.[1]

Electrocardiographic anomalies have been found in approximately 50% of patients with AP.[1] Examples of these anomalies include arrhythmias, conduction

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anomalies, ST elevation, and other electrocardiogram (ECG) findings such as changes in T wave or QT period duration.[2-5]

An increased P-wave dispersion (PWD) is accepted as a predictor of atrial fibrillation.[6] In literature reviews, ECG abnormalities are frequently observed in AP cases. However, although there is a relationship between AP and atrial fibrillation, there are not enough studies examining PWD in AP patients.

In this study, PWD was evaluated using an ECG in terms of its ability to demonstrate atrial conduction delay during an AP attack. The study aimed to investigate the changes in PWD in AP and contribute to the literature. We hypothesize that PWD, which is thought to be an indicator of atrial fibrillation, increases in AP.

MATERIAL AND METHODS

The study was approved by the Ethics Committee of Health Science University Şişli Hamidiye Etfal Training and Research Hospital (approval date and number: June 19, 2018-2013). Patients who were diagnosed with AP, treated, and followed up between July 2018 and July 2019 were prospectively evaluated. Written informed consent was obtained from each patient. The diagnosis of AP was made by acute abdominal pain and tenderness, serum amylase and/or lipase levels at least 3 times the normal level (Siemens Dimension Xpand Plus HM Chemistry Analyzer, Budapest, Hungary; Reference Ranges Amylase 28-100 U/L, Lipase <60 U/L), and typical findings detected by contrast-enhanced computed tomography by an experienced abdominal radiologist.[1]

The study included patients aged 18-60 who agreed to participate, were diagnosed, treated, and followed up in our hospital, and achieved complete recovery according to the second-month follow-up. The exclusion criteria were hypertension, cardiac diseases (cardiomyopathy, valvular disease, heart failure or atrial and/or ventricular hypertrophy on ECG), renal disease [creatinine > 1.0 mg/dL (0.4-1.0 mg/dL)], severe anemia [<10 g/dL (10.0-18.0 g/dL)], alcoholism (alcohol consumption > 60 g/day), hypothyroidism or hyperthyroidism, connective tissue disease, use of any drug known to cause prolonged QT interval, pregnancy, breastfeeding, and smoker patients. Twenty-two patients with hypertension, 17 patients who were smokers, 11 with a body mass index above 35, 7 patients with cardiac disease, 3 with electrolyte imbalance, 4 with thyroid dysfunction, 3 with alcohol dependence, and 1 with connective tissue disease were excluded from the study. As a result, of the total 107 patients diagnosed with AP, 50 who did not have any of the exclusion criteria formed the study group. Sample size calculation was performed before the study (https://www.analytics-toolkit.com/statistical-power-calculator).

MAIN POINTS

- Cardiac complications are frequently seen in acute pancreatitis.
- In patients with acute pancreatitis, P-wave dispersion (PWD) is significantly prolonged compared to post-treatment.
- The patients with acute pancreatitis have a higher probability of atrial arrhythmia due to prolonged PWD.
- For patients diagnosed with acute pancreatitis, appropriate follow-up should be undertaken, and preventive measures should be applied.

The ECGs of the patients were recorded with a standard 12-lead device in the supine position at a speed of 50 mm/sec and an amplitude of 1 mV/cm (ECG 1150, Nihon Kohden, Tokyo, Japan). This procedure was performed by 2 authors using a magnifying glass (5 times magnification) and an electronic ruler capable of measuring at 1/10 mm precision with 0 adjustments, and the arithmetic averages of the 2 measurements were obtained. The difference between the longest (maximum) and shortest (minimum) P-wave duration in 12 leads was calculated as PWD and recorded in ms. P-wave dispersion > 40 ms was considered pathological.[6] The maximum and minimum P-wave durations in ms were measured with a 12-lead ECG device at the time of the AP diagnosis and the second-month follow-up after treatment. The maximum P-wave and PWD values of the patients were compared between the 2 evaluation times (Figure 1).

Statistical Analysis

The data were described using quantitative descriptive statistics, such as mean, SD, median, minimum, maximum, and frequency, as well as qualitative descriptive statistics, including percentage values. The distribution of variables was measured with the Kolmogorov–Smirnov test. The Wilcoxon test was used in the analysis of dependent quantitative data. International Business Machiness (IBM) Statistical Package for the Social Sciences (SPSS) Statistics for Windows, version 22.0 (IBM SPSS Corp.; Armonk, NY, USA), program was used for the statistical analyses. The level of significance was set at $P < .05$ for all statistical tests.

RESULTS

Twenty-six (52%) patients were male and 24 (48%) were female. The mean age of the patients included in the study was 43.2 ± 12.5 (range, 19 to 60) years. The follow-up period was 2 months for all patients. The

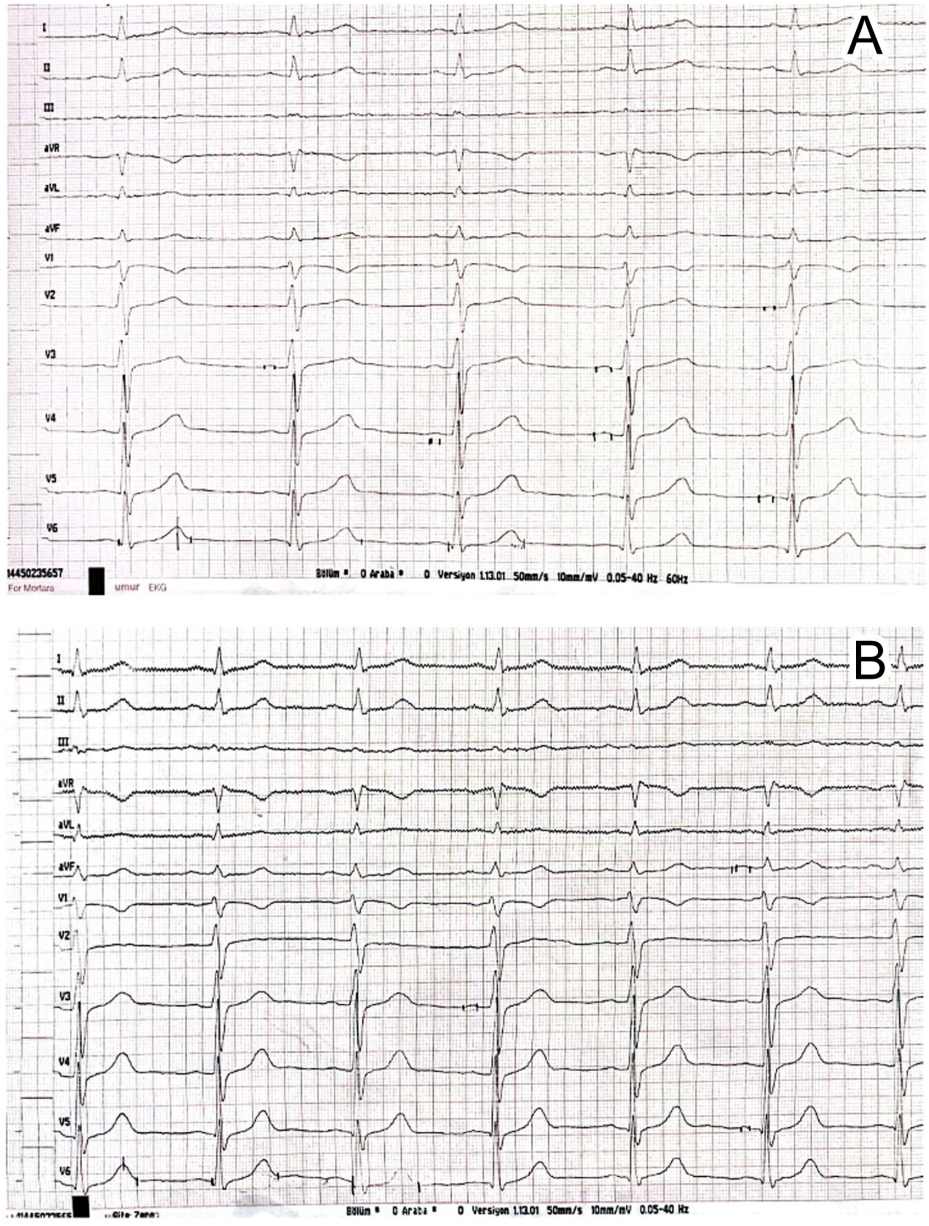


Figure 1. An ECG of one of the patients (A) the ECG at the time AP was diagnosed, (B) the ECG at the second-month follow-up after treatment (AP, acute pancreatitis; ECG, electrocardiography).

demographic characteristics and clinical data of the patients are summarized in Table 1. During the follow-up period, 2 patients were diagnosed with AF, and the treatment was planned and followed up in consultation with a cardiologist.

When the ECGs were evaluated at the first visit, significant (>40 ms) PWD was detected in 42% of the patients. In the first visit undertaken during the AP attack, the average PWD was determined as 41.5 ± 9.1 (range, 30-60) ms. At the last follow-up, significant (>40 ms) PWD was observed in 26% of the patients, and the mean PWD value was 37.5 ± 9.3 (range, 20-60) ms (Figure 2). The PWD value measured during the AP attack was significantly higher compared to the second-month post-attack value ($P = .003$) (Table 2).

When the ECGs were evaluated at the first visit, the mean maximum P-wave duration was 98.7 ± 11.4 (60-120) ms, while the mean maximum P-wave duration (Figure 3). Thus, the mean maximum P-wave duration value was significantly higher during the attack compared to the second-month post-attack value ($P = .002$) (Table 3).

DISCUSSION

The most important aspect of this study is that it is one of the very few studies in the literature examining PWD in patients with AP. In the current study, PWD and the

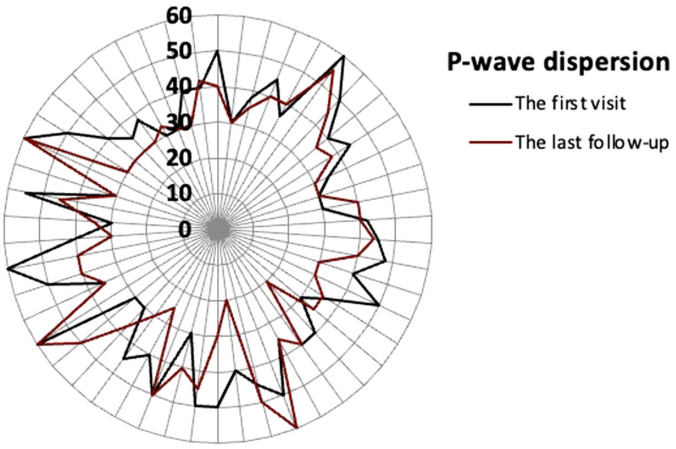


Figure 2. P-wave dispersion values graphs at the first visit and the last follow-up.

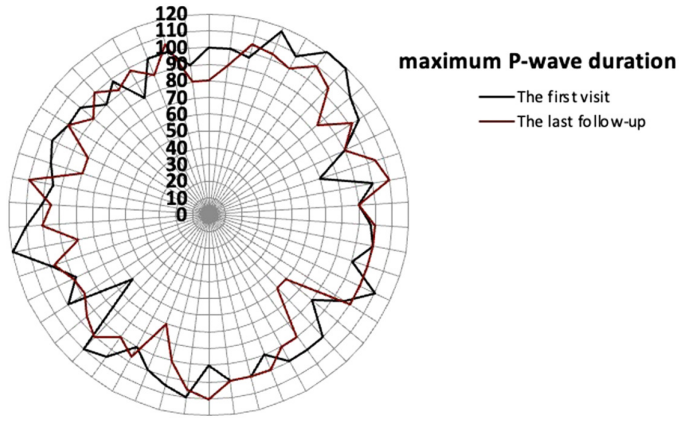


Figure 3. Maximum P-wave duration values graphs at the first visit and the last follow-up.

maximum P-wave durations measured during the AP attack were found to be significantly longer than those obtained at the second-month follow-up after the completion of treatment. This result is an indicator that the risk of developing AF increases in patients with AP.

The critical event in AP is trypsinogen activation in the pancreas. The resulting trypsin activates the complement system and the kallikrein cascade, as well as coagulation and fibrinolysis. Inflammatory cytokines are involved; it is thought to be responsible for the systemic manifestations and complications of the systemic inflammatory response syndrome. During the course of AP, problems affecting almost all organs may occur.[7,8] Cardiovascular complications include shock, hypovolemia, pericardial effusion, and nonspecific ST–T changes on ECG that mimic acute myocardial infarction. The presence of electrocardiographic changes and paroxystic atrial fibrillation has been reported in AP.[9-11]

Increased PWD is considered to be an indicator of an increased risk of developing atrial fibrillation.[12-14] Yoshizawa et al.[15] compared the ECGs of the sinus rhythm in 68 AF patients diagnosed for the first time with 68 controls without AF and found that both the P-wave duration and PWD were longer in the AF group. Gonna et al.[16] calculated the P-wave duration and PWD from the superficial ECGs of 77 patients with persistent AF who underwent electrical cardioversion and observed that a long P-wave duration was associated with the development of recurrent AF within 1 month after electrical cardioversion. In the current study, AF development was observed in 2 (4%) patients during the 2-month follow-up. Although this rate is not high, it is considered important due to the short follow-up period.

In a study by Dedeoğlu et al.,[6] PWD and QTc interval times were significantly prolonged in patients with AP compared to the healthy control group. It was reported that patients with AP might have a higher probability of ventricular or atrial arrhythmia due to the prolonged QTc and PWD durations. In a case report by Bhatt et al.[3] a 47-year-old male patient with AP was observed to have cardiac tamponade and new-onset AF. In this study, the

Table 1. Demographic Characteristics of the Patients Included in the Study

	Minimum-Maximum	Median	Mean ± SD
Age (years)	19.0-60.0	44.5	43.2 ± 12.5
Height (m)	1.6-1.65	1.7	5.0 ± 23.1
Weight (kg)	50.0-127.0	77.5	79.2 ± 13.1
Body mass index	17.3-37.1	27.0	27.8 ± 4.7

Table 2. Evaluation of P-Wave Dispersion at the First Visit and Last Follow-up of the Patients

	Minimum-Maximum	Median	Mean ± SD	*P*
P-wave dispersion				
First visit	30-60	40	41.5 ± 9.1	***.003***[w]
Last follow-up	20-60	36	37.5 ± 9.3	

[w]Wilcoxon test.

Table 3. Evaluation of the Maximum P-wave duration at the First Visit and Last Follow-Up of the Patients

	Minimum-Maximum	Median	Mean ± SD	*P*
Maximum P-wave duration				
First visit	60-120	100	98.7 ± 11.4	***.020***[w]
Last follow-up	60-110	98	94.1 ± 11.5	

[w]Wilcoxon test.

mean PWD was determined as 41.5 ± 9.1 (range, 30-60) ms at the first visit and 37.5 ± 9.3 (range, 20 to 60) ms at the last examination. Similarly, in our study, the PWD value was significantly higher at the first visit during the AP attack compared to the second-month post-attack follow-up.

There are some limitations in this study. The first limitation of the study is the small number of patients and the short follow-up period. In addition, PWD was manually calculated on the ECG paper. Although manual measurement has been scientifically accepted and many studies have been carried out with this method, it is considered that the measurement performed on a high-resolution monitor (with Holter) together with digital ECG recording provides much more accurate and standardized results. The second limitation can be considered as the absence of echocardiography to evaluate the left atrial diameter and left ventricular ejection fraction of the patients since the risk of developing atrial arrhythmia is closely related to the left atrial dimensions. P-wave dispersion is an inexpensive and non-invasive marker of left atrial enlargement and electrical heterogeneity. Increased left atrial dimensions, atrial wall tension, and atrial fibrosis create electrical heterogeneity in the atrial tissue and affect the spread of the impulse from the sinus node. This results in an increase in PWD measured on ECG.

In conclusion, in patients with AP, PWD was significantly prolonged compared to post-treatment. We consider that patients with AP have a higher probability of developing atrial arrhythmia due to their prolonged PWD; therefore, appropriate follow-up should be undertaken, and preventive measures should be applied. However, there is a need for comparative and long-term studies with a sufficient number of patients to provide more accurate information.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Health Science University Şişli Hamidiye Etfal Training and Research Hospital (date: June 19, 2018, number: 2013).

Informed Consent: Written informed consent was obtained from patients who agreed to take part in this study.

Peer-Review: Externally peer-reviewed.

Author Contributions: Concept – A.B.; Design – A.B.; Supervision – F.B.; Resources – G.Z.; Materials – F.B.; Data Collection and/or Processing – G.Z.; Analysis and/or Interpretation – Ö.Y.; Literature Search – U.O.; Writing Manuscript – A.B.; Critical Review – F.B.; Other – U.O., Ö.Y.

Declaration of Interests: The authors have no conflict of interest to declare.

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