ORIGINAL RESEARCH

Bagcilar Med Bull
DOI: 10.4274/BMB.galenos.2025.18894

The Impact of Systemic Inflammation and Electrolyte Imbalance on Electrocardiogram Parameters in Acute Pancreatitis: A Clinical Research Article

Akut Pankreatitte Sistemik Enflamasyon ve Elektrolit Dengesizliğinin Elektrokardiyogram Parametreleri Üzerindeki Etkisi: Klinik Bir Araştırma Makalesi

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Abstract

Objective: Acute pancreatitis (AP) frequently results in hospitalization and is associated with substantial morbidity and mortality, and may lead to various complications, including electrocardiographic (ECG) abnormalities. Cardiovascular manifestations in AP are often underrecognized, despite their potential clinical significance. We aimed to analyze ECG changes in patients with AP and examine their associations with systemic inflammatory response and electrolyte imbalances.

Method: We retrospectively reviewed data from 89 patients with a diagnosis of AP and 49 healthy controls. ECG parameters and laboratory data were analyzed. Comparative statistical analyses were performed to assess differences in ECG findings and laboratory results between the AP and control groups.

Results: Patients with AP demonstrated significant prolongation of P_{max}, P dispersion, P wave peak time in lead D2, and V1, QT_{max}, QTc_{max}, and Tp-e intervals compared to controls. These ECG alterations were associated with lower serum potassium and calcium levels in the AP group. Inflammatory markers, particularly C-reactive protein, were significantly

Öz

Amaç: Akut pankreatit (AP), önemli morbidite ve mortaliteye yol açabilen, sık görülen bir hastaneye yatış nedenidir ve elektrokardiyografik (EKG) değişiklikler dahil olmak üzere çeşitli komplikasyonlara neden olabilir. AP'de kardiyovasküler bulgular, klinik olarak anlamlı olabilmelerine rağmen sıklıkla göz ardı edilmektedir. Bu çalışmanın amacı, AP'li hastalarda EKG değişikliklerini değerlendirmek ve bu değişikliklerin sistemik enflamatuvar yanıt ve elektrolit dengesizlikleriyle ilişkisini incelemektir.

Yöntem: Bu retrospektif çalışmada AP tanısı almış 89 hasta ile 49 sağlıklı kontrol grubu değerlendirildi. EKG parametreleri ve laboratuvar verileri analiz edildi. AP grubu ile kontrol grubu arasındaki EKG bulguları ve laboratuvar sonuçlarındaki farkları değerlendirmek için karşılaştırmalı istatistiksel analizler yapıldı.

Bulgular: AP'li hastalarda, kontrol grubuna kıyasla P_{maks}, P dispersiyonu, D2 ve V1 derivasyonlarında P dalga pik zamanı, QT_{maks}, QTc_{maks} ve Tp-e aralıklarında anlamlı uzamalar saptandı. Bu EKG değişiklikleri, AP grubunda düşük serum potasyum ve kalsiyum düzeyleri ile ilişkiliydi. Enflamatuvar belirteçlerden özellikle C-reaktif protein düzeyleri anlamlı

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Received: 12.05.2025 **Accepted:** 27.08.2025 **Epub:** 29.08.2025

Cite this article as: Çavdar VC, Öz A, Akdeniz E, Akbaş F, Arıç M, Tanrıverdi FN, et al. The impact of systemic inflammation and electrolyte imbalance on electrocardiogram parameters in acute pancreatitis: a clinical research article. Bagcilar Med Bull. [Epub Ahead of Print].

Abstract

elevated and showed a positive correlation with ECG abnormalities. Multivariate analysis revealed that P_{max} and QTc_{max} durations remained prolonged significantly and independently in the AP group.

Conclusion: ECG monitoring should be considered in patients with AP, as ECG changes may reflect underlying systemic inflammation and electrolyte disturbances, which could have prognostic value.

Keywords: Acute pancreatitis, ECG parameters, electrolyte imbalance, inflammation

Öz

şekilde yüksekti ve EKG anormallikleriyle pozitif korelasyon gösterdi. Çok değişkenli analizde, P_{maks} ve QTc_{maks} sürelerinin AP grubunda anlamlı ve bağımsız şekilde uzamış olduğu görüldü.

Sonuç: AP'li hastalarda EKG izlem dikkate alınmalıdır; çünkü EKG değişiklikleri altta yatan sistemik enflamasyonu ve elektrolit bozukluklarını yansıtabilir ve prognostik değer taşıyabilir.

Anahtar kelimeler: Akut pankreatit, EKG parametreleri, elektrolit inbalansı, enflamasyon

Introduction

Pancreatitis accounts for a significant portion of hospital admissions for gastrointestinal diseases in the United States, and due to its high morbidity and mortality rates, it represents a major healthcare concern; it also imposes a substantial socioeconomic burden (1).

In patients presenting to the emergency department with abdominal pain, acute pancreatitis (AP) holds substantial percentage of cases. A thorough medical history, a well-conducted physical examination, along with laboratory findings and imaging results, is generally sufficient for diagnosis. The diagnosis of pancreatitis requires at least two out of three diagnostic criteria: Increased pancreatic enzyme levels, typical abdominal pain, and imaging findings supportive of the diagnosis (2).

To grasp the concept of pancreatitis, it is crucial to explore its fundamental pathophysiological processes. Over time, considerable progress has been made in elucidating the molecular mechanisms of acute pancreatitis. Studies have demonstrated how calcium (Ca) contributes to acinar cell injury and death, particularly by triggering Ca influx pathways and causing mitochondrial membrane disruption. Furthermore, the beneficial effects of the unfolded protein response and autophagy in alleviating prolonged endoplasmic reticulum stress, apoptosis, and necrosis have been clearly established. The significant role of unsaturated fatty acids in triggering pancreatic organ failure has also been extensively investigated (3).

AP is caused by numerous etiological factors. The primary causes include gallstones, ethanol consumption, hypertriglyceridemia, medications, endoscopic retrograde cholangiopancreatography, trauma, hypercalcemia, viral infections, tumors, anatomical variations, cardiac bypass surgery, scorpion stings, and organophosphate poisoning (4).

AP can affect multiple organ systems, with the extent of involvement largely depending on the severity of inflammation. Cardiovascular changes in AP are often overlooked but can present with life-threatening complications. Several studies have explored the mechanisms by which pancreatitis affects the cardiovascular system. It was predicted that severe hypotension in AP could reduce coronary artery perfusion, thereby precipitating myocardial ischemia (5). In addition, vagal reflexes triggered by parasympathetic stimulation increase the secretion of proteolytic enzymes from the pancreas, which are believed to directly contribute to myocardial and cardiac damage (6,7). It was shown that certain electrolyte imbalances observed in pancreatitis could trigger ischemic changes on the electrocardiographic (ECG) (8). Myocardial injury may occur in patients with AP, irrespective of the disease's severity. In some cases, pancreatitis can mimic the clinical manifestations of acute coronary syndrome, despite the absence of significant coronary artery obstruction (9). AP patients presenting to the emergency department might exhibit atrial extrasystole, bradycardia, T wave and/or ST segment changes, intraventricular conduction abnormalities, left anterior hemiblock, complete left bundle branch block, incomplete right bundle branch block, and first-degree atrioventricular block (10). Moreover, there is a significant association between atrial fibrillation (AF) and AP. In patients with AF, the risk of developing AP is 1.5 times that of those without AF (11). Furthermore, the occurrence of AF in patients with AP has been associated with unfavorable clinical outcomes (12). In addition to AF, a case report was presented in which a patient with AP developed ventricular extrasystoles and ventricular tachycardia (13). Given the clinically meaningful association between AP and cardiac arrhythmias, both in terms of potential causality and prognostic implications, the evaluation of ECG parameters to predict arrhythmias holds considerable importance. Such an approach may contribute to more accurate

risk stratification and improved patient management, ultimately enhancing clinical outcomes.

Given the growing evidence of cardiovascular involvement in AP and the potential prognostic implications of ECG changes, this research aimed to comprehensively investigate alterations in specific ECG parameters at the time of hospital admission in patients with AP compared to healthy controls. Furthermore, the study sought to evaluate whether these parameters, particularly P wave indices, QT and QTc intervals, and ventricular repolarization markers, could serve as indicators of arrhythmic risk in this patient population, thereby contributing to improved risk stratification and early clinical management strategies.

Materials and Methods

A total of 89 patients diagnosed with acute pancreatitis between January 2023 and January 2024 and admitted to the Department of Internal Medicine of University of Health Sciences Turkey, İstanbul Training and Research Hospital were included in our retrospective study. Medical records, including complete blood count (CBC) and biochemical data, were obtained from the hospital's electronic health information system. ECG parameters were derived from measurements taken from ECG recordings obtained at the time of hospital admission. The CBC, biochemical, and ECG data from these patients were then compared to those of a control group made up of 49 healthy individuals of similar age. The control subjects had no documented history of cardiovascular disease, chronic systemic illness, or the use of medications known to influence ECG parameters or laboratory findings. The inclusion criteria were being over 18 years old and being admitted to the internal medicine department with a diagnosis of AP. Exclusion criteria included having type 1 or type 2 diabetes, end-stage renal disease, liver cirrhosis, a background of bypass surgery or operative treatment for heart valve disorders, recent ST-elevation myocardial infarction, newly diagnosed heart failure with reduced ejection fraction, atrial or ventricular arrhythmias, and bundle branch block detected on ECG. Electrolyte imbalance was not excluded, as it is a common component of the natural course of AP. Any observed ECG changes could, at least in part, be attributed to electrolyte disturbances secondary to the underlying disease process.

The Ethics Committee of University of Health Sciences Turkey, İstanbul Training and Research Hospital granted approval for this study (approval no: 15, date: 05.07.2024). Written informed consent was obtained from all participants prior to their inclusion in the study. All study procedures complied with the ethical standards of the 1964 Helsinki Declaration and its later amendments.

ECG Evaluation

Upon admission to the hospital, every patient underwent a standard 12-lead ECG, performed at a paper speed of 25 mm/s with signal amplitude standardized to 10 mm per millivolt. The durations of the P wave at its longest and shortest were recorded, and P wave dispersion was determined by subtracting the shortest duration from the longest. The amplitude of the P wave in lead DI was measured in millivolts. We defined the P wave peak time (PWPT) as the time from the start of the P wave to its peak, measured in leads DII and VI. For cases with a biphasic P wave morphology, the PWPT was calculated as the time between the initiation of the P wave and its maximal downward deflection. The P wave morphology in lead V1 was categorized as positive, negative, or biphasic. Adjustment of the QT interval was performed according to Bazett's correction method. The lead displaying the highest T wave amplitude was used to measure R-R intervals, as well as the maximum and minimum QT intervals. The termination of the T wave was determined by locating the intersection between the isoelectric line and the tangent drawn along its steepest descending slope. QT and QTc dispersions were calculated by subtracting the shortest QT measurement from the longest. The JTp interval was described as the interval from the start of the J wave to the peak of the T wave, and the Tpe interval as the interval from the peak of the T wave to its endpoint (Figure 1). Amplitude and time interval measurements on the ECG recordings were performed using the EP Calipers software (EP Studios, London, UK). PWPT was assessed by two independent observers who were unaware of the patients' clinical data.

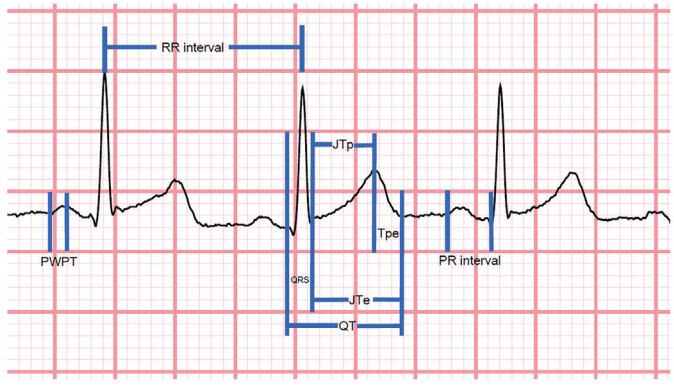

Figure 1. Image related to ECG measurements

ECG: Electrocardiographic, PWPT: P wave peak time, JTp: J to T peak

The inter-observer reliability for PWPT measurements was high, with a Pearson correlation coefficient of 0.88 ($p<0.001$).

Statistical Analysis

Findings were summarized using descriptive statistics, including mean, standard deviation, median, minimum, maximum, frequency, and percentage values. Normality of the variables was assessed using the Kolmogorov-Smirnov and Shapiro-Wilk tests. Parametric tests (Independent samples t-test) were conducted for variables with normal distributions, while non-parametric methods (Mann-Whitney U test) were utilized for variables without normal distributions. Qualitative independent variables were assessed through the chi-square test. Logistic regression models, both univariate and multivariate, were applied to assess the magnitude of the effect. Statistical analyses were performed with SPSS software (version 27.0).

Results

The study included 138 participants, comprising 89 patients with AP and 49 healthy controls. The overall mean age was 49.5±9.7 years, 49±9.8 years in the pancreatitis group and 50.5±9.5 years in the control group. Among the study participants, 61.6% (n=85) were male and 38.4% (n=53) were female. In the pancreatitis group, 67.4% (n=60) were male and 32.6% (n=29) were female.

When examining the etiology of the 89 pancreatitis patients, 28.3% (n=39) were found to have idiopathic pancreatitis, 14.5% (n=20) had pancreatitis secondary to hypertriglyceridemia, 13% (n=18) had biliary pancreatitis, 5.8% (n=8) had alcoholic pancreatitis, 2.2% (n=3) had drug-induced pancreatitis, and 0.7% (n=1) had pancreatitis of infectious origin.

The laboratory parameters of 89 pancreatitis patients and 49 healthy controls at hospital admission are collectively presented in Table 1. Table 2 presents the results of collective measurements obtained from the ECG' s of all patients.

Table 3 presents the laboratory parameters of the pancreatitis patients and the control group. Age and gender distributions were comparable between the control and case groups, with no statistically significant differences observed ($p>0.05$). In the case group amylase, lipase, aspartate aminotransferase, alanine aminotransferase, C-reactive protein (CRP), and white blood cell (WBC) levels were significantly increased compared to the control group ($p<0.05$). Conversely, potassium (K) and Ca levels were significantly decreased in the case group than in the control group ($p<0.05$) (Table 3).

Table 4 presents the ECG parameters of the pancreatitis patients and the control group together. In the case group, P_{max}, P dispersion, PWPT-D2, and PWPT-V1 values were significantly higher ($p<0.05$) compared to the control group. The biphasic P wave in lead V1 was significantly lower ($p<0.05$) in the case group compared to the control group. Furthermore, the control and case groups showed no statistically significant differences in minimum and mean QT intervals ($p>0.05$). However, the maximum and QT dispersion values were significantly higher ($p<0.05$) in the case group compared to the control group. Similarly, there were no significant differences ($p>0.05$) in minimum or mean QTc values between the groups, but maximum QTc and QTc dispersion values were significantly higher ($p<0.05$) in the case group. In the case group, Tp-e, Tp-e/QT_{mean}, Tp-e/QTc_{mean}, and Tp-e/JTp values were significantly higher ($p<0.05$) compared to the control group (Table 4).

In the univariate model, significant ($p<0.05$) effectiveness of amylase, K, Ca, CRP, WBC, P_{max}, P dispersion, PWPT-D2, PWPT-V1, P wave morphology in lead V1, QT_{max}, QT dispersion, QTc_{max}, QTc dispersion, Tp-e interval, Tp-e/QT_{mean}, Tp-e/QTc_{mean}, Tp-e/ JTp values, was observed in distinguishing between control and case group patients. In the multivariate model, a significantindependent ($p<0.05$) association of amylase, WBC, P_{max}, and QTc_{max} values was observed in the distinction between control and case group patients (Table 5).

Discussion

In our study evaluating the ECG changes in patients admitted to the hospital with AP, the main ECG changes we observed were prolongation of P_{max}, P dispersion, PWPT-D2, PWPT-V1, QT_{max}, QT dispersion, QTc_{max}, and QTc dispersion. Also, Tp-e, Tp-e/QT_{mean}, Tp-e/QTc_{mean}, and Tp-e/JTp values were found to be prolonged in patients with pancreatitis. Moreover, the biphasic P wave in lead V1 was markedly lower in pancreatitis patients than in the control group. In the multivariate analysis, P_{max} and QTc_{max} durations were found to be significantly and independently longer in patients with AP.

In the present study, the maximum duration of the P wave (P_{max}), which corresponds to atrial depolarization in the ECG, was found to be prolonged in patients with pancreatitis. A possible mechanism may involve P_{max} prolongation. Firstly, AP is an acute inflammatory disease that begins in the pancreas but is significantly influenced by systemic inflammatory mediators. The importance of

inflammatory mediators in the morbidity and mortality of the disease is substantial (14). Lazzerini et al. (15) conducted a study investigating the effects of inflammation on atrial electrical remodeling; it was found that the P_{max} duration was associated with levels of inflammatory mediators, leading to the conclusion that the pro-inflammatory process may prolong P_{max} duration. This result is consistent with our study. The second possible mechanism may be related to alterations in electrolyte levels. Severi et al. (16) explored the relationship between electrolyte levels and P wave durations in patients undergoing haemodialysis. They identified an inverse relationship between K levels and P wave durations. It was shown that after hemodialysis, K levels decreased while P wave durations increased (16). In our study, K levels in the AP group were likely to be lower as compared to the control group. This mechanism can be another factor contributing to the prolonged P_{max} period in our patients with AP. Other possible myocardial involvement mechanisms are immune-mediated myocyte damage, disruptions in myocardial microcirculation, and autonomic dysfunction, in AP (9). As a result of the factors outlined above, atrial electrophysiology may be altered, causing a prolongation of the P wave.

Another statistically significant ECG change in our study was the prolongation of QTc duration. The QT interval on the ECG reflects the time required for ventricular depolarization and repolarization, corresponding to the duration of the cardiac action potential (17). Since the QT duration varies with heart rate, the QT value measured on

Table 1. Classification of all patients in the study

		Min-max	Median	Mean ± SD/n-%	
Age (years)		19.0-78.0	50.0	49.5±9.7	
Gender	Male			85	61.6%
	Female			53	38.4%
Pancreatitis etiology	Idiopathic			39	28.3%
	Hypertriglyceridemia			20	14.5%
	Biliary			18	13.0%
	Alcoholic			8	5.8%
	Drug related			3	2.2%
	Infection			1	0.7%
Control group				49	35.5%
Glucose (mg/dL)		73.0-156.0	94.0	99.9±19.1	
HbA1c (%)		4.3-6.4	5.6	5.6±0.4	
Amylase (U/L)		26.0-3909.0	145.7	479.1±766.6	
Lipase (U/L)		12.0-8472.6	261.8	926.6±1523.6	
AST (U/L)		10.0-604.0	23.0	51.3±83.9	
ALT (U/L)		10.0-421.0	23.0	43.1±61.8	
Urea (mg/dL)		11.0-67.1	29.0	29.1±9.4	
Creatinine (mg/dL)		0.41-1.5	0.8	0.8±0.2	
GFR (mL/min/1.73 m^2)		13.4-134.3	98.0	95.9±18.1	
Na (mmol/L)		122.0-148.0	139.0	138.4±4.1	
K (mmol/L)		3.21-5.5	4.2	4.2±0.4	
Mg (mg/dL)		1.0-3.6	2.0	2.0±0.3	
Albumin (g/L)		3.9-56.1	44.0	42.8±7.0	
Ca (mg/dL)		7.8-10.6	9.4	9.3±0.6	
CRP (mg/L)		0.3-412.0	6.6	36.3±68.6	
WBC (10^9/L)		4.9-31.3	9.0	10.1±4.3	
Hb (g/dL)		8.9-18.2	14.0	13.9±1.8	
PLT (10^9/L)		79.0-448.0	256.5	260.6±78.5	

Values are presented as minimum, maximum, medain, mean ± standard deviation and number (%)
SD: Standard deviation, HbA1c: Glycated haemoglobin, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, e-GFR: Estimated glomerular filtration rate, Na: Sodium, K: Potassium, Mg: Magnesium, Ca: Calcium, CRP: C-reactive protein, WBC: White blood cell, Hb: Hemoglobin

Table 2. ECG characteristics of all patients in the study

		Min-max	Median	Mean ± SD/n-%	
P_{max} (ms)		57.3-164.8	102.9	104.9±16.3	
P_{min} (ms)		26.3-85.0	56.9	58.2±12.3	
P_{disper} (ms)		13.4-100.8	43.9	46.9±17.8	
P wave D1 amplitude time (mm)		0.70-3.08	1.30	1.40±0.42	
PR interval (ms)		70.6-280.5	142.6	143.9±26.0	
PWPT in lead DII (ms)		17.3-77.9	52.1	50.3±13.0	
PWPT in lead V1 (ms)		13.5-65.5	31.6	32.0±9.4	
P-wave morphology in lead V1	Positive			45	32.6%
	Negative			63	45.7%
	Biphasic			30	21.7%
QRS duration (ms)		62.0-196.0	84.0	85.8±14.2	
Heart rate (/min)		46.0-126.0	79.0	79.9±13.9	
P axis (o)		-4.0-89.0	55.0	51.5±18.6	
R axis (o)		-63.0-101.0	28.0	28.1±30.9	
T axis (o)		-27.0-200.0	43.5	44.2±27.1	
QT_{min} interval (ms)		175.9-428.6	325.4	325.8±37.6	
QT_{max} interval (ms)		317.7-490.8	382.0	383.4±32.4	
QT_{mean} interval (ms)		265.2-459.7	352.5	357.4±39.7	
QT_{disper} interval (ms)		6.6-214.4	52.0	57.6±33.2	
QTc_{min} interval (ms)		204.0-483.0	376.5	372.4±39.3	
QTc_{max} interval (ms)		346.0-546.0	436.0	438.3±32.0	
QTc_{mean} interval (ms)		298.5-514.5	407.0	408.2±43.4	
Qtc_{disper} interval (ms)		9.0-252.0	58.0	66.0±38.6	
Tp-e interval (ms)		34.8-124.0	64.4	65.6±15.4	
JTp interval (ms)		116.1-294.2	197.8	201.0±31.0	
JTe interval (ms)		154.0-388.5	267.0	266.7±35.0	
Tp-e/QT_{mean} (ms)		0.09-0.36	0.18	0.18±0.04	
Tp-e/QTc_{mean} (ms)		0.07-0.35	0.15	0.16±0.04	
Tp-e/JTp (ms)		0.15-0.72	0.31	0.33±0.10	
RR interval (ms)		446.9-1301.0	765.2	778.8±145.8	

Values are presented as minimum, maximum, medain, mean ± standard deviation and number (%). SD: Standard deviation, disper: Dispersion, PWPT: P wave peak time, QTc: Corrected QT, Tp-e: Tpeak-Tend interval, JTp: J to T peak, JTe: J to T end

the ECG needs to be corrected to obtain the QTc. Various formulas have been developed for this purpose (18). The compelling evidence of relationship between QTc and ventricular arrhythmias establish QTc as a highly significant parameter (19,20). While the exact mechanism of QTc max prolongation in AP patients remains unclear, several potential pathophysiological processes are suggested. One mechanism may be the systemic inflammatory response caused by AP. Because it has been shown that there is a relationship between active inflammation, pro-inflammatory cytokine levels, and QTc duration in different clinical scenarios, such as myocarditis, inflammatory bowel disease, and rheumatoid arthritis (21-24), these associations warrant further investigation. Since AP is a clinical condition that generates a strong inflammatory response, the prolongation of QTc_{max} duration in our study may have been secondary to the inflammatory process. Consistent with our findings, Ates et al. (25) observed that patients with acute biliary pancreatitis had a significantly prolonged QTc_{max} compared to healthy controls. Another possible scenario for the prolongation of QTc_{max} durations in the AP group could be related to Ca and K levels, which in the AP group tended to be lower than those in the healthy control group. Pickham et al. (26) showed that hypocalcemia and hypokalemia were predictors of QT prolongation in acutely ill patients. One more potential mechanism that could lead to the prolongation of QTc is the use of certain medications. Pain management constitutes a fundamental component

Table 3. Comparison of control group and case group

		Control group (n=49)			Case group (n=89)			p
		Mean ± SD/n-%		Median	Mean ± SD/n-%		Median	
Age (years)		50.5±9.5		50.0	49.0±9.8		50.0	0.782[m]
Gender	Male	25	51.0%		60	67.4%		0.058[X²]
	Female	24	49.0%		29	32.6%		
Glucose (mg/dL)		96.2±14.3		92	101.9±21.1		96.0	0.076[m]
HbA1C (%)		5.60±0.3		5.5	5.6±0.4		5.6	0.594[m]
Amylase (U/L)		65.7±18.6		67.0	706.7±875.9		260.1	**0.000**[m]
Lipase (U/L)		38.2±13.6		38.0	1415.8±1712.5		644.7	**0.000**[m]
AST (U/L)		23.2±7.4		22.0	67.0±101.2		23.4	**0.000**[m]
ALT (U/L)		24.1±14.3		22.0	53.8±74.4		22.0	**0.000**[m]
Urea (mg/dL)		29.4±8.5		29.0	28.9±9.9		29.0	0.78[m]
Creatinine (mg/dL)		0.8±0.14		0.8	0.83±0.20		0.80	0.304[m]
GFR (mL/min/1.73 m²)		93.3±15.3		95.0	97.4±19.4		100.9	0.090[m]
Na (mmol/L)		139.4±2.7		139.0	137.9±4.7		139.0	0.198[m]
K (mmol/L)		4.41±0.41		4.47	4.13±0.44		4.13	**0.001**[m]
Mg (mg/dL)		1.96±0.15		1.98	1.95±0.34		1.98	0.593[m]
Albumine (g/L)		42.1±8.5		44.0	43.1±6.1		43.9	0.727[m]
Ca (mg/dL)		9.6±0.5		9.6	9.2±0.6		9.2	**0.001**[t]
CRP (mg/L)		3.6±1.6		4.0	54.3±80.0		22.1	**0.000**[m]
WBC (10^9/L)		7.4±1.5		7.2	11.6±4.5		10.9	**0.000**[m]
Hb (g/dL)		13.7±1.5		13.4	14.0±1.9		14.1	0.342[t]
PLT (10^9/L)		277.0±69.1		261.0	251.5±82.2		254.0	0.105[m]

[t]: Independent sample t-test, [m]: Mann-Whitney U test, X^2: Chi-square test
Values are presented as mean ± standard deviation, median and number (%).
SD: Standard deviation, HbA1c: Glycated haemoglobin, AST: Aspartate aminotransferase, ALT: Alanine transaminase, e-GFR: Estimated glomerular filtration rate, Na: Sodium, K: Potassium, Mg: Magnesium, Ca: Calcium, CRP: C-reactive protein, WBC: White blood cell, Hb: Hemoglobin

of AP treatment. Non-steroidal anti-inflammatory drugs or opioids used for this purpose can cause QTc prolongation (27).

To summarize, ECG changes can be observed in patients with AP. In some cases, these changes may even include ST-segment elevation or mimic the presentation of acute coronary syndrome. ECG changes are not exclusively caused by AP; they can also be observed in a variety of other clinical conditions (28-31).

Table 4. Comparison of ECG features of the control group and the case group

		Control group (n=49)			Case group (n=89)			p
		Mean ± SD/n-%		Median	Mean ± SD/n-%		Median	
P_{max} (ms)		93.8±13.7		93.8	110.9±14.3		109.3	**0.000**[m]
P_{min} (ms)		57.6±11.8		56.7	58.5±12.6		56.9	0.676[t]
P_{disper} (ms)		36.3±12.4		38.3	52.6±17.7		49.1	**0.000**[m]
P wave D1 amplitude time (mm)		1.32±0.25		1.30	1.44±0.49		1.30	0.461[m]
PR interval (ms)		140.2±21.0		142.0	146.0±28.3		146.0	0.171[m]
PWPT in lead DII (ms)		47.4±12.2		48.0	52.0±13.2		53.1	**0.045**[t]
PWPT in lead V1 (ms)		28.3±8.5		28.4	34.1±9.2		33.0	**0.001**[m]
P-wave morphology in lead V1	Positive	15	30.6%		30	33.7%		**0.001**[X^2]
	Negative	15	30.6%		48	53.9%		
	Biphasic	19	38.8%		11	12.4%		
QRS duration (ms)		83.2±9.9		82.0	87.2±16.0		84.0	0.152[m]
Heart rate (/min)		78.7±13.0		78.0	80.6±14.4		79.0	0.460[t]
P axis (o)		51.1±18.2		55.0	51.7±18.9		54.5	0.704[m]
R axis (o)		25.1±25.1		22.0	29.7±33.7		30.0	0.406[t]
T axis (o)		44.1±18.8		44.0	44.3±30.8		43.0	0.613[m]
QT								
QT_{min} interval (ms)		331.0±24.8		326.6	323.0±42.9		324.6	0.426[m]
QT_{max} interval (ms)		373.1±25.1		376.7	389.1±34.6		383.6	**0.017**[m]
QT_{mean} interval (ms)		358.6±48.9		348.6	356.7±34.0		353.8	0.786[t]
QT_{disper} interval (ms)		42.1±17.3		42.0	66.1±36.7		56.0	**0.000**[m]
QTC								
QTc_{min} interval (ms)		375.3±23.6		376.0	370.8±45.8		377.0	0.897[m]
QTc_{max} interval (ms)		422.9±20.9		418.0	446.8±33.9		447.0	**0.000**[m]
QTc_{mean} interval (ms)		407.0±56.9		402.0	408.8±34.1		410.5	0.060[m]
Qtc_{disper} interval (ms)		47.6±19.3		49.0	76.1±42.7		64.0	**0.000**[m]
Tp-e interval (ms)		59.5±11.5		59.1	68.9±16.3		66.6	**0.000**[t]
JTp interval (ms)		200.5±23.7		198.4	201.3±34.5		197.2	0.864[t]
JTe interval (ms)		260.0±25.3		258.5	270.4±39.0		268.8	0.094[t]
$Tp\text{-}e/QT_{mean}$ (ms)		0.17±0.03		0.16	0.19±0.05		0.19	**0.000**[m]
$Tp\text{-}e/QTc_{mean}$ (ms)		0.15±0.03		0.15	0.17±0.04		0.17	**0.001**[m]
Tp-e/JTp (ms)		0.30±0.07		0.29	0.35±0.10		0.33	**0.003**[m]
RR interval (ms)		789.7±126.4		803.1	772.8±155.8		758.6	0.366[m]

[t]: Independent sample t-test, [m]: Mann-Whitney U test, X^2: Chi-square test
Values are presented as minimum, maximum, median, mean ± standard deviation and number (%).
SD: Standard deviation, disper: Dispersion, PWPT: P wave peak time, QTc: Corrected QT, Tp-e: Tpeak-Tend interval, JTp: J to T peak, JTe: J to T end, ECG: Electrocardiographic

Table 5. Univariate and multivariate analysis of biochemistry values and ECG findings

	Univariate model			Multivariate model		
	OR	95% CI	p	OR	95% CI	p
Amylase (U/L)	1.072	1.037-1.107	**0.000**	1.117	1.020-1.222	**0.017**
Lipase (U/L)	2.756	0.000->100	0.900			
K (mmol/L)	0.440	0.215-0.900	**0.025**			
Ca (mg/dL)	0.333	0.170-0.650	**0.001**			
CRP (mg/L)	1.362	1.154-1.608	**0.000**			
WBC (10^9/L)	1.744	1.406-2.164	**0.000**	2.627	1.077-6.408	**0.034**
P_{max} (ms)	1.122	1.074-1.173	**0.000**	1.105	1.005-1.215	**0.040**
P_{disper} (ms)	1.078	1.044-1.114	**0.000**			
PWPT in lead DII (ms)	1.028	1.000-1.057	**0.048**			
PWPT in lead V1 (ms)	1.085	1.035-1.137	**0.001**			
P-wave morphology in lead V1	0.569	0.347-0.931	**0.025**			
QT_{max} interval (ms)	1.018	1.005-1.031	**0.007**			
QT_{disper} interval (ms)	1.049	1.025-1.074	**0.000**			
QTc_{max} interval (ms)	1.030	1.015-1.046	**0.000**	1.098	1.006-1.199	**0.037**
Qtc_{disper} interval (ms)	1.047	1.024-1.070	**0.000**			
Tp-e interval (ms)	1.048	1.019-1.078	**0.001**			
Tp-e/QT_{mean} (ms)	>100	450.230->100	**0.002**			
Tp-e/QTc_{mean} (ms)	>100	361.366->100	**0.002**			
Tp-e/JTp (ms)	>100	7.736->100	**0.005**			

Logistic regression (forward LR)
OR: Odds ratio, CI: Confidence interval, K: Potassium, Ca: Calcium, CRP: C-reactive protein, WBC: White blood cell, disper: Dispersion, PWPT: P wave peak time, QTc: Corrected QT, Tp-e: Tpeak-Tend interval, JTp: J to T peak, ECG: Electrocardiographic

Conclusion

In conclusion, significant changes in ECG parameters may be observed in patients with AP. Various factors, such as the cardiac effects of the systemic inflammatory response, fluctuations in electrolyte levels, and the medications used, can contribute to these ECG changes.

Ethics

Ethics Committee Approval: The Ethics Committee of University of Health Sciences Turkey, İstanbul Training and Research Hospital granted approval for this study (approval no: 15, date: 05.07.2024).

Informed Consent: Informed consent was obtained from each participant in compliance with applicable ethical regulations.

Footnotes

Authorship Contributions

Concept: V.C.Ç., A.Ö., E.A., F.A., B.B., Design: V.C.Ç., A.Ö., E.A., F.A., M.A., F.N.T., B.B., Data Collection or Processing: V.C.Ç., M.A., F.N.T., Y.G., Analysis or Interpretation: V.C.Ç., A.Ö., E.A., M.A., Y.G., Literature Search: V.C.Ç., A.Ö., E.A., F.A., M.A., F.N.T., Y.G., B.B., Writing: V.C.Ç., E.A.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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