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Interatrial Block and Ventricular Repolarization Parameters in Young Patients with Mild Acute Myocarditis

Hafif Akut Miyokarditli Genç Hastalarda İnteratriyal Blok ve Ventriküler Repolarizasyon Parametreleri

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ABSTRACT

Objective: To evaluate ECG findings regarding interatrial block and ventricular repolarization and certain biochemical parameters in young patients diagnosed as acute myocarditis with normal ventricular functions.

Materials and Methods: 405 patients under the age of 35 who underwent diagnostic coronary angiography between January 2014 and January 2020 were retrospectively analyzed. Patients whose ECG records could not be accessed or quality were not suitable for evaluation, with sudden cardiac death, cardiomyopathy, ejection fraction <50%, diabetes mellitus, hypertension, chronic kidney and liver failure were excluded. Patients who underwent diagnostic angiography for the differential diagnosis of acute myocarditis were assigned to myocarditis group (n: 35), and age– and sex–matched subjects with normal coronary circulation and underwent coronary angiography for any other reason were assigned to control group (n: 35).

Results: Heart rate, P wave duration, P wave peak time, PR interval, QRS, QT, and Tp–e interval, QTc, Tp–e/QT and Tp–e/QTc ratios did not differ from each other. Whereas troponin (p<0.001), glucose (p=0.004), LDL (p=0.015), AST (p<0.001), ALT (p<0.026), CRP (p<0.001) levels and neutrophil count (p=0.003) were markedly higher in myocarditis group, HDL was lower (p<0.001).

Conclusions: Although biochemical parameters display differences in myocarditis group, ECG findings did not differ.

Keywords: ECG, myocarditis, Tp–e interval, troponin, ventricular repolarization

ÖZ

Amaç: Ventrikül fonksiyonları normal olan akut miyokardit tanısı almış genç hastalarda interatriyal blok ve ventriküler repolarizasyon ile ilgili EKG bulgularını ve bazı biyokimyasal parametreleri değerlendirmektir.

Materyal ve Metot: Ocak 2014 ile Ocak 2020 arasında tanısal koroner anjiyografi yapılan 35 yaş altı 405 hastanın verileri geriye dönük olarak incelendi. EKG kayıtlarına ulaşılamayan veya kayıt kalitesi değerlendirmeye uygun olmayan, ani kardiyak ölüm, kardiyomiyopati, ejeksiyon fraksiyonu <%50, diabetes mellitus, hipertansiyon, kronik böbrek ve karaciğer yetmezliği olan hastalar çalışma dışı bırakıldı. Akut miyokardit ayırıcı tanısı için tanısal anjiyografi yapılan hastalar miyokardit grubuna (n: 35) ve yaş ve cinsiyet açısından uyumlu, koroner dolaşımı normal olan ve başka bir sebepten ötürü anjiografi yapılan hastalar kontrol grubuna dahil edildi (n: 35).

Bulgular: Kalp hızı, P dalgası süresi, P dalgası tepe zamanı, PR aralığı, QRS, QT ve Tp–e aralığı, QTc, Tp–e/QT ve Tp–e/QTc oranları gruplar arasında farklılık göstermedi. Troponin (p<0,001), glukoz (p=0,004), LDL (p=0,015), AST (p<0,001), ALT (p<0,026), CRP (p<0,001) düzeyleri ve nötrofil sayısı (p=0,003) miyokardit grubunda belirgin olarak yüksek iken, HDL daha düşüktü (p<0,001).

Sonuç: Biyokimyasal parametreler miyokardit grubunda farklılık göstermsine rağmen, EKG bulguları farklılık göstermedi.

Anahtar Kelimeler: EKG, miyokardit, Tp–e aralığı, troponin, ventriküler repolarizasyon

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INTRODUCTION

Myocarditis is a kind of myocardial damage that occurs either by various microorganisms or a primary inflammatory event. Viral myocarditis is a widely accepted cause of arrythmias and sudden cardiac death (SCD).

Myocarditis might progress to dilated cardiomyopathy (DCM) in approximately 30%.[1] Patients often present with chest pain mimicking myocardial ischemia, palpitations, myalgia, arthralgia, fever, rash, fatigue, signs of heart failure (HF), or arrhythmic complications, most of which begin a few days after respiratory or gastrointestinal tract viral infection. Clinical findings and course of the disease vary according to the etiologic agent, the degree of inflammation, the presence and severity of complications such as HF and arrhythmia.[2] Acute myocarditis in young adults very typically might be presented with chest pain suggestive of pericarditis or myocardial infarction, commonly accompanied by elevated serum troponin levels.[3]

Abnormal QRS complex and left bundle branch block findings are found to be associated with more advanced stage and poor prognosis.[4] The most common ECG finding in myocarditis is supraventricular tachycardia, and are the others nonspecific ST segment and T wave changes.[5] Interatrial block (IAB) and atrial fibrillation (AF) expose DCM patients to an increased risk of life–threatening arrhythmias (LTA).[6]

In this study, existence of IAB along with certain ventricular repolarization parameters on ECG recording, such as QT interval, QTc (corrected value according to heart rate extremes), Tp–e (time between the peak and end of the T wave), and Tp–e/QT and biochemical parameters were analyzed in young acute myocarditis patients.

MATERIALS AND METHODS

Ethics Committee Approval: Ethical approval was provided by Adıyaman University Non–interventional Clinical Research Ethical Committee (Date: 20.10.2020, decision no: 2020/9–17). The whole process was carried out in accordance with the Declaration of Helsinki. All study–related data are available upon reasonable request.

Subjects: 405 patients under the age of 35 who underwent diagnostic coronary angiography between January 2014 and January 2020 were retrospectively analyzed. Patients whose ECG records could not be accessed or whose recording quality were not suitable for evaluation, those with SDC, cardiomyopathy, ejection fraction <50%, diabetes mellitus, hypertension, chronic kidney and liver failure were excluded. We calculated a sample size of 27 in each group with an alpha error of 5% and power of 95. Patients who underwent diagnostic angiography for the differential diagnosis of acute myocarditis and diagnosed as acute myocarditis in conformity with criteria determined by working group of European Society on Myocardial and Pericardial Diseases were assigned to the myocarditis group (n: 35), and age– and sex–matched subjects with normal coronary circulation, but also underwent an angiography procedure for any other reason were assigned to the control group (n: 35).[1]

Analyses: ECG recordings taken at the first admission were scanned, and then analyzed using ImageJ (imagej.nih.gov/ij) and CardioCaliper programs at 300% magnification. Heart rate, P wave duration, P wave peak time and PR interval were measured from the inferior leads. QRS, QT, and Tp–e intervals were measured from chest leads, then QTc (calculated by Bazett's formula) and Tp–e/QTc ratio was calculated. IAB was defined according to the diagnostic criteria as a P–wave ≥120 ms. A notched P wave in the inferior leads and P wave duration ≥120 ms in lead DII was considered as partial IAB. Advanced IAB was considered to be present when a P–wave ≥120 ms and a negative terminal part was detected in one of the inferior leads II, III or aVF (biphasic or positive–negative).[7]

Statistical Analyses: Data was appraised with the SPSS–24. Kolmogorov–Smirnov test was used for normality testing. When the descriptive statistics of groups had a normal distribution, variables were expressed as mean ± standard deviation (SD). Independent student's t test was used to make comparisons between quantitative variables of groups. A two –sided p value of <0.05 was accepted as the level of significance.

RESULTS

Neither parameters such as mean age, male ratio, body mass index (BMI), smoking rate, blood pressure values, creatinine, total cholesterol, triglyceride, albumin, Na+ level, hemoglobin, lymphocyte count and neutrophil/lymphocyte ratio (Table 1) nor ECG measurements which were heart rate, P wave duration in DII, P wave peak duration in DII, PR interval, QRS interval, QT interval, QTc interval, Tp–e interval, Tp–e/QT and Tp–e/QTc ratios differed significantly from each other between groups (Table 2). Pericarditis was detected in 5% of group 1. Troponin levels were significantly higher in group 1. Actually, troponin was not found to be elevated at all in group 2 patients. Whereas glucose level, low–density lipoprotein (LDL), C–reactive protein (CRP), white blood cell count (WBC), neutrophil count, CRP/Albumin ratio, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were markedly higher, high–density lipoprotein (HDL) was signifi-

Table 1. Demographic characteristics and laboratory parameters of the patients.

	Group 1 (Acute myocarditis) (n: 35)	Group 2 (Control) (n: 35)	p value
Age, years	26.61 ± 4.91	26.93 ± 2.42	0.688
***Male n (%)**	32 (91.4%)	32 (91.4%)	1.0
***Pericarditis (%. n)**	14.28 %. 5	0. 0	-
BMI (kg/m2)	26.41 ± 2.12	26.73 ± 1.83	0.541
***Smoking (%. n)**	37.14 %. 13	34.28 %. 12	0.803
Systolic blood pressure (mmHg)	115.60 ± 7.35	116.70 ± 9.74	0.345
Diastolic blood pressure (mmHg)	77.20 ± 3.45	77.80 ± 2.82	0.473
Troponin (µg/mL)	1.85 ± 2.22	0	0.000
Glucose (mg/dL)	104 ± 18	94 ± 16	0.004
Creatinine (mg/dL)	0.90 ± 0.20	0.80 ± 0.10	0.076
Total cholesterol (mg/dL)	163.50 ± 29.32	166.40 ± 52.37	0.920
Triglyceride (mg/dL)	151.10 ± 52.32	136.70 ± 62.90	0.056
HDL (mg/dL)	32.60 ± 7.20	39.40 ± 10	0.001
LDL (mg/dL)	96.70 ± 14.10	89.70 ± 17	0.015
Albumin (g/dL)	3.90 ± 0.30	4.10 ± 0.10	0.184
AST U/L	49.70 ± 22.50	25.50 ± 11.20	0.000
ALT U/L	31.80 ± 12.80	25 ± 10.70	0.026
Na (mEq/L)	138.70 ± 2	139.10 ± 2.40	0.477
K (mEq/L)	4 ± 0.50	4.30 ± 0.30	0.005
Ca (mg/dL)	9.20 ± 0.14	9.29 ± 0.10	0.001
C–reactive protein (CRP) mg/dL	4.20 ± 1.50	0.10 ± 0.10	0.000
Wight blood cell × 103/µL	9.80 ± 2.70	8.60 ± 1.70	0.001
HGb (mg/dL)	15.51 ± 0.90	15.61 ± 1.10	0.562
Platelet × 10^3/µL	235.70 ± 65	240.20 ± 59.38	0.765
Neutrophil (N) × 10^3/µL	6.52 ± 2.54	4.96 ± 1.55	0.003
Lymphocyte (L) × 10^3/µL	2.34 ± 0.79	2.57 ± 0.62	0.190
N/L ratio	2.71 ± 1.43	2.12 ± 0.84	0.090
CRP/Albumin	1 ± 0.40	0.01 ± 0.01	0.000

Continuous variables are represented as mean ± SD, with the exception of percentage of male, subjects who smoke and pericarditis cases in groups.

Table 2. Comparison ECG measurements of acute myocarditis and control groups.

	Group 1 (Acute myocarditis; n: 35) (mean ± SD)	Group 2 (Control; n: 35) (mean ± SD)	p value
Heart rate (beats/min)	75 ± 13.20	76 ± 15.40	0.892
DII P wave duration (ms)	98 ± 17.20	96 ± 14.30	0.778
DII P wave peak time (ms)	54 ± 15.80	50 ± 12.70	0.240
PR interval (ms)	147 ± 26.10	135 ± 27.50	0.066
QRS interval (ms)	93 ± 13.70	91.80 ± 15	0.817
QT interval (ms)	372 ± 46.50	371 ± 42.50	0.985
QTc interval (ms)	410 ± 42.60	413 ± 33.60	0.198
Tp-e interval (ms)	91 ± 16.90	92 ± 24.40	0.754
Tp-e/QT ratio	0.25 ± 0.05	0.25 ± 0.05	0.923
Tp-e/QTc ratio	0.22 ± 0.05	0.22 ± 0.06	1.0

Tp-e: Tpeak–Tend interval, time between the peak and end of the T wave; c = Corrected value according to heart rate extremes.

cantly lower, in group 1. As for electrolytes, K^+ and Ca^{++} levels were markedly higher in group 2 (Table 1). IAB was not encountered at all in both groups.

DISCUSSION AND CONCLUSION

It seems that because of the differences occurred in determination of the control groups make the findings of our and the previous studies not to confirm each other. In one of previous studies, it was observed that the prognosis of patients with fulminant myocarditis who had needed intensive hemodynamic support, but most of them not requiring mechanical support, was better during the course of the disease than patients with acute non–fulminant myocarditis in long–term follow–up.[8] So, the question, "How do we determine the prognosis?" stands still to be elucidated.

IAB predisposes to AF. This relationship gets stronger as the duration of the P wave gets longer. On the other hand, AF give rise to a vicious cycle of LTA and SCD. It is revealed that AF is associated with LTA and SCD independently of other factors. In case of additional considerations of patient, like advanced age, heart failure, vascular diseases, arrythmias or structural heart diseases, IAB even much vigorously predisposes to atrial fibrillation/flutter.

The importance of IAB and AF comes from the emergence of a thrombogenic sequence due to delayed left atrial excitation. Particularly, advanced IAB is a secure predictor of AF, as this is the case for Chaga's cardiomyopathy.[9–11] Uçar et al. declare that patients with clinically diagnosed as acute myocarditis display significantly higher Tp–e interval and Tp–e/QT and Tp–e/QTc ratios. The Tp-e interval is considered to be an indicator of transmural dispersion, although it is somewhat controversial.[12,13] An increase or prolongation in the Tp–e interval or Tp–e/QT ratio is considered an index of arrhythmogenesis in all cases of long, normal, or short QT intervals, or in acquired or congenital channelopathies.[14] Although Uçar et al. found the Tp –e interval or Tp–e/QT ratio to be significantly higher in the group of acute myocarditis patients compared to the control group, it should be noted that the control group in their study is consisted of completely healthy volunteers. The normal value of the Tp–e/QT ratio is around 0.192 ± 0.35.[13-15] Since our study did not involve completely healthy controls, we cannot say whether the mean value found is statistically different from the healthy control values. Güneş et al. also declare that QT interval, Tp–e interval, Tp–e/QT ratio and Tp–e/QTc ratio values are found to be markedly prolonged in myocarditis group when compared to control group.[16]

In patients with fulminant myocarditis, a decreasing trend in high–sensitivity cardiac troponin I in the first 24 hours is found to be associated with a lower incidence of in–hospital mortality.[17] As myocarditis is an inflammatory disease, CRP, WBC and neutrophil count were higher in myocarditis group in our study, as expected. High CRP level and erythrocyte sedimentation rate are among the most important parameters that allow us to accurately monitor the course of the disease and response to treatment. Although lymphocytosis often accompanies leukocytosis in myocarditis, only the neutrophil count was significantly higher in the myocarditis group in our study.[1,18]

High HDL and triglyceride levels are found to be associated with lower mortality in the group of HF patients, the majority of whom had idiopathic dilated, ischemic, or hypertensive cardiomyopathy, and Chagas' disease.[18] This is somewhat consistent with our study because we encountered significantly lower HDL and higher LDL levels in the myocarditis group. Since cholesterol is a substance that cannot be broken down and only converted, body cells have various efflux mechanisms that are responsible for removing cholesterol. It is known that increased cholesterol induces apoptosis in a myriad of cells, such as smooth muscle cells, pancreatic β–cells and macrophages.[20–22] An important component of acute myocarditis is apoptosis seen in cardiomyocytes, which is considered to be the main pathology leading to DCM or HF.[23] In an experimental autoimmune myocarditis study, it is observed that the cholesterol efflux mechanisms of cardiomyocytes are impaired. The inflammatory process is observed to be regressed and cardiac functions improved, following draining of cholesterol out of the cardiomyocytes by a cholesterol depleting agent, called methyl –β–cyclodextrin.[20] In a previous study, it is observed that troponin I, ALT and AST, which are found to be high during an acute myocarditis attack, decrease significantly after clinical remission is achieved.[24] In our study, ALT and AST levels were markedly higher in the myocarditis group compared to the control.

In conclusion, novel studies, which will be conducted by stratifying myocarditis patients according to the severity and even etiology of the disease, are required to be able to specify biomarkers or short–long term predictors in myocarditis.

Ethics Committee Approval: Ethical approval was provided by Adıyaman University Non–interventional Clinical Research Ethical Committee (Date: 20.10.2020, decision no: 2020/9–17).

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