

Electrocardiographic Changes in Patients with Inflammatory Cardiomyopathy.

Ahmedova D.M¹;Hojakuliyev B.G²;Muhammedov M.B²
Turkmen State Medical University (Ashgabat ,Turkmenistan)

Abstract

Purpose: studying variability of changes on an electrocardiogram due to localization of inflammatory process.

Material and methods : We analysed 53 electrocardiograms of patients with an inflammatory cardiomyopathy (middle age 35,01±1,0). Patients were divided into two groups – 1) 21 patients - with acute forms of inflammatory cardiomyopathy (AIC), and 2) – 32 patients with a chronic form of inflammatory cardiomyopathy (CIC). The diagnosis was established according to recommendations of WHO experts. The control group consisted of 30 almost healthy people (middle age 32,61±1,1).

Results : The analysis of resting ECG (table 1) at the time of hospitalization found widespread violations of the process of repolarization in the form of reduction in the amplitude of the positive wave T and the appearance of smooth, isoelectric or low positive T wave (5 class of the Minnesota code).

Conclusion: In patients with severe forms of inflammatory cardiomyopathy , ECG examination may reveal repolarization breaking of T wave, decrease the voltage of wave R, elongation and depression interval QT.

Keywords: cardiomyopathy, heart failure, depression, hypertrophy.

I. Introduction

Inflammatory cardiomyopathy (IC) constitutes an important class of noncoronary myocardial diseases takes resulting from exposure to infectious agents, chemical and physical factors, as well as appearing on the background of allergic and autoimmune processes. According to estimates by different studies, frequency of inflammatory cardiomyopathy reaches 4-11% of all cardiovascular diseases. Although cardiomyopathy (C) is a major cause of morbidity and mortality of among young age cardiac patients, it is a less studied problem. Diseases of the cardiovascular system are one of the main causes of early morbidity and mortality. Among these, inflammatory disease of the myocardium is particularly relevant; and presents challenges for the intern doctors in terms of verification of the causes, diagnosis and treatment strategies [2,9].

For the noncoronary of damages of a myocardium, the ambiguity of an etiological factor and uniformity of clinical manifestations, including a cardiomegaly, decrease in pump function of heart, the progressing HF causing a high lethality, disturbances of rhythm and conduction of heart, and also very frequent tromboembolic episodes [1,3, 6] is often peculiar. In general, there is considerable amount of research on studying the features of ECG of a picture and heart rhythm disturbances (HRD) and conductions at a myocarditis [5, 10, 12]. However their detailed analysis is complicated by that cases of myocardites of absolutely various etiology are considered, at different stages of a disease, in many works there is no morphological confirmation of the diagnosis, various diagnostic approaches concerning HRD [4,13] are noted. Comparative studies of the characteristics of HRD and conductions in patients with clinical and morphologic diagnosis of myocarditis are extremely rare.

Abnormal electrocardiographic changes in inflammatory cardiomyopathy are non-specific and they are associated not only with inflammatory infiltration but with degeneration of cardiomyocytes and myocardial cardiosclerosis [8,11,14].

Purpose: studying variability of changes on an electrocardiogram from localization of inflammatory process.

II. Material And Methods

We analysed 53 electrocardiograms from patients with an inflammatory cardiomyopathy (middle age 35,01±1,0). Patients were divided into two groups: 21 patients with acute forms of inflammatory cardiomyopathy (AIC), and 32 patients with a chronic form (CIC). The diagnosis was established according to recommendations of WHO experts. The control group was made of 30 healthy people (middle age 32,61±1,1). Assessment of activity of heart it was carried out by means of an electrocardiogram. The electrocardiogram was registered on the electrocardiograph for every patient on the first days of hospitalization of patients in the morning. Necessarily made the electrocardiogram by Neb and Slopak, for identification of cicatricial changes. For this purpose, we used quantitative electrocardiographic criteria: the size of teeth of RI Ravl, RV2-5, SIII, Sv, TI, TavI, TV5-6, RI+SIII syndrome, RV5-6+SVI syndrome, TVI-TV6 syndrome, and also some qualitative

criteria of Qv5-6, Rv5-6 and Rv4 the increased T tooth in assignments of V5-6. Along with it criteria of a hypertrophy of the right ventricle are applied: $R/S_{VI} > 1$, $R_{VI}+S_{V6} > 10,5\text{mm}$, $R_{V5-6} < 5\text{mm}$, $S_{VI} > 22\text{mm}$, $R/S_{V5-6} < 1$, $S_{V5-6} > 7\text{mm}$. The comparative analysis of the electrocardiograms was conducted by taking into account changes of teeth P, lengthening of an atrio-ventricular interval, changes of QRS, shifts of ST [7,15].

III. Results

The analysis of resting ECG (table 1) at the time of hospitalization found widespread violations of the process of repolarization as a reduction in the amplitude of the positive wave T and the appearance of smooth, isoelectric or low positive T wave (5 class of the Minnesota code). In 21.4% of patients in group 1 and 5.2% of patients in group 2, we did not find significant differences in changes of T wave between the studied groups. In 1st group, low-voltage curve and segment depression of ST complex was more common (4 class of the Minnesota code ($p < 0,05$)). In this group of patients, Makruz index was significantly lower ($1,5 \pm 0,06$), as well as more common measurable lengthening of the interval QT which reached a maximum 0.08 seconds. ($p < 0,05$).

Table 1

ECG parameters	1 st group	2 nd group	Class of Minnesota code
Repolarization breaking of T-wave	16	10	5-3
Diffuse decrease of R-wave voltage	10	3*	9-1
QT prolongation	14	2**	
ST depression intervals	12	3*	4-1-2

Note: * $p < 0,05$ ** $p < 0,001$

Infringements of myocardium repolarization as the changes in the terminal portion of the ventricular complex were identified in 26 (43.3%) patients with IC. P wave duration did not differs significantly between the surveyed groups (Fig. 1). Averages of P wave in 1st group were within $0,09 \pm 0,001$ seconds, the 2nd group - $0,10 \pm 0,001$ seconds. And the averages of segment PQ were in the 1st group - $0,10 \pm 0,001$ seconds, in the 2nd group - $0,13 \pm 0,002$ seconds ($p < 0,05$). In the 1st group of patients, QT prolongations were identified due to the expansion of wave T in 5.1%, high P wave in II and III standard leads in 6.3% and elongation of atrioventricular interval PQ in 8.9% of cases.

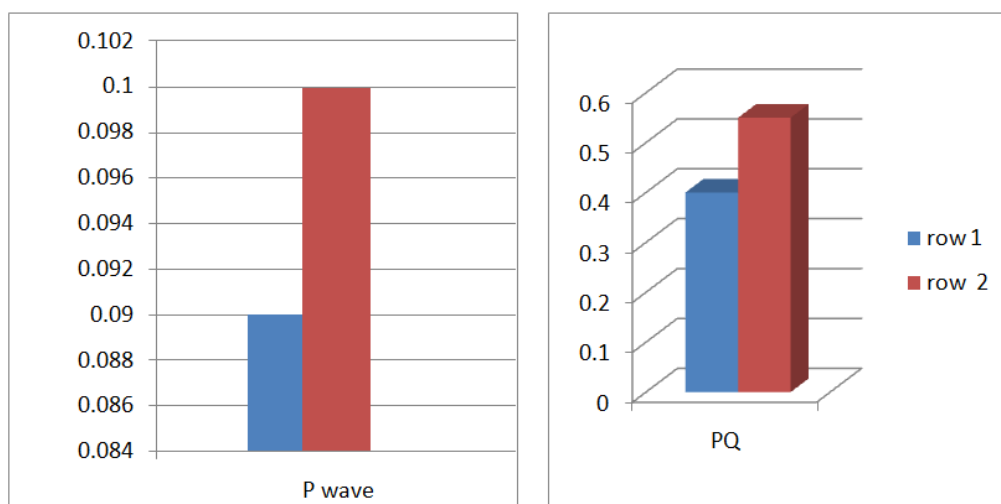


Fig. 1. The duration of P wave and PQ interval depending on the activity of inflammation

These changes are obviously related to the stage of the inflammatory process. QRS complex in 5.8% of patients has been expanded with the increase in the amplitude of the I and II standard leads to the displacement of the electrical axis to the left. In 3.2% of patients examined, electrical axis of the heart was rejected to the right with high P wave in II and III leads which indicates the overvoltage of the pulmonary circulation. At 7.3% of patients, T wave flattening was identified in I and II standard leads as well as in V4. 3.1% of cases registered negative T waves and in 6.3% of cases detected pathological Q wave and 16.7% noted lengthening the duration of complex QRS.

Analysis of the duration of intervals PQ, QT and QTc showed that in patients with inflammatory cardiomyopathy the whole width of the QRS complex was significantly different from the respective figures in healthy individuals (table 2). Thus, the duration of atrioventricular conduction QT and QTc intervals is statistically significantly higher in IC patients in comparison with the control group ($p < 0,05$).

Table 2 Electrocardiographic parameters in patients with inflammatory cardiomyopathy and control group

Indicators	Inflammatory cardiomyopathy n=53	Control group n=30	P
PQ ms	184,2±4,2	160,5±3,6	<0,05
QT ms	476,8±17,4	380,5±15,8	<0,05
QT ms	510,1±11,1	430,1±9,3	<0,05
QRS ms	103,7±3,0	89,1±1,9	<0,05

p - significance of differences between the IC and control

ECG signs of left ventricular hypertrophy (LVH) were identified in more than half of patients with inflammatory cardiomyopathy 41 (52.6%) patients. Along with left ventricular hypertrophy, we observed almost equally often combined ventricular hypertrophy - in 32 (41.0%) patients. ECG signs of right ventricular hypertrophy (RVH) and hypertrophy of the interventricular septum (IVS) were rare. RVH was detected in 3 (3.8%) patients and the IVS hypertrophy in 2 (2.6%) of patients with IC (**Fig. 2**)

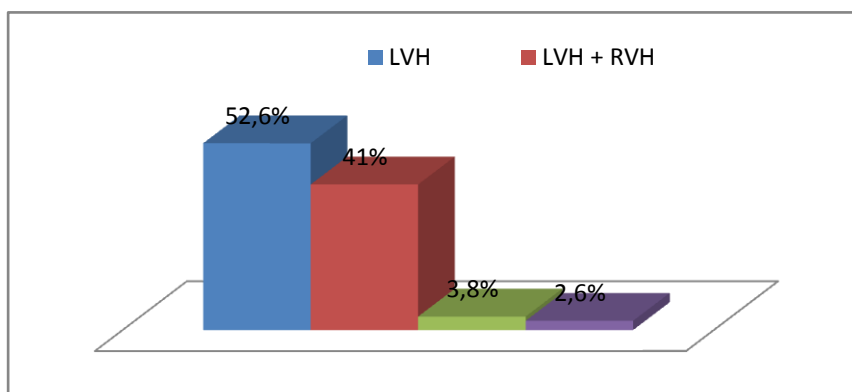


Fig. 2. The frequency of hypertrophy various parts of the heart in patients with inflammatory cardiomyopathy

Cicatricial changes on an ECG create significant differential diagnostic difficulties. Published data indicate that the ECG of patients with noncoronary myocardial diseases very often revealed focal changes in the left ventricle. Cicatricial changes in the myocardium according to commonly accepted ECG leads were identified in 21 patients with chronic inflammatory cardiomyopathy (CIC) and localization in all parts of the left ventricle

(**Fig. 3**).

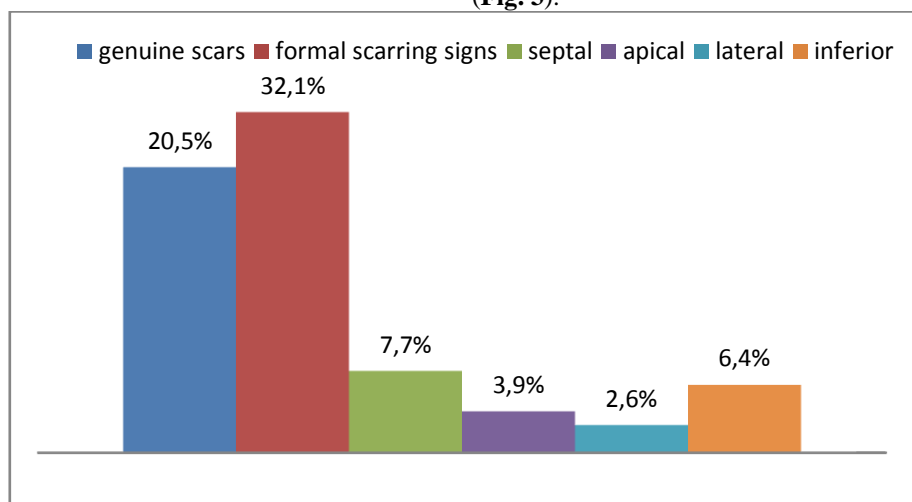


Fig. 3 Frequency of heart attack similar changes on an ECG in 12-leads

IV. Discussion

In our analysis of the number of identified focal myocardial injury does not correspond to the absolute number of patients because in one patient can be combined lesion of various localization such as the front-side, front-apical and septal area. In such situation, the count was conducted separately in each area of the lesion, without combining them into the categories as comprehensive, front and common.

The most frequently recorded formal signs of myocardial cicatricial changes was in the form of small teeth «r» in precordial leads in 25 (32.1%) patients. True myocardial cicatricial changes according to 12-leads of ECG were observed in 16 (20.5%) patients CIC. They had the following localization - septal – in 6 (7.7%) cases, the apical – in 3 (3.9%) cases, lateral – in 2 (2.6) cases, inferior – in 5 (6.4%) cases. In 5 (6.4%) cases recorded deep scar S on V1-V6, wave q on V1-V6 was observed in 2 (2.6%) cases. In 3 (3.9%) patients CIC was found “frozen” ECG which indicate about left ventricular aneurysm.

V. Conclusion

Thus, ECG examination in patients with severe forms of inflammatory cardiomyopathy can commonly reveal repolarization breaking of T wave, decrease the voltage of wave R, elongation and depression interval QT. At the same time in patients with chronic forms of IC - hypertrophy of various parts of the heart and changes similar to heart attack can be observed.

Conflict of interest statement:

The other authors have no conflicts of interest to declare.

Acknowledgements

This study was supported by the Turkmen State Medical University

Literature

- [1]. Belyavsky E.A., Zyakov K.A., Narusov O. Yu., Masenko V.P., A.A., Shchedrin A.Yu. Starlings. Inflammatory cardiomyopathy: Current state of problems.//Therapeutic archive, 2010. No. 8, page 62-71.
- [2]. Fighters S. A., Deryugin M. V. Non rheumatic myocardites / Guide to cardiology: Book. A grant in 3 t.//Under G. I. Storozhakov, A.A. Gorbachenkov's edition. - M.: Geotar-media, 2008. – T. 2. – page 116-145.
- [3]. Gurevich M. A. Chronic heart failure. M.: MIA,-2005. Page 280.
- [4]. Kurbanov R. D., Abdullaev T.A. Myocardites.//Guide to clinical cardiology, 2007 of Page 223-237.
- [5]. Moiseyev V. S., Kiyakbayev G.K.Patogenesis, clinic and diagnostics of a cardiomyopathy and myocardites.//In book: Cardiomyopathy and myocardites. Kiev., 2011. - C127-230.
- [6]. Ryabenko D. V. Inflammatory cardiomyopathy: questions of terminology and treatment.//Rational Pharmacology scientific and practical magazine. 2008.-№1. - Page-16-22.
- [7]. Ryabykina G. V. Holter monitoring of an electrocardiogram in detection of noncoronary pathology.//Messenger aritmologii.2005.- №4.C.14-19.
- [8]. Paleev N. R., Paleev F.N. Classification of noncoronary diseases of a myocardium//Cardiology, 2008; 9 Pages 53-58.
- [9]. Abramson S.V., Burke J.F., Kelly J.J. et al. Pulmonary hypertension predicts mortality and morbidity in patients with dilated cardiomyopathy //Ann.Intern. Med. 2002. - V. 116. - № 11. - P. 888-895.
- [10]. Baldeviano G.C., Barin J.G., Taylor M.V. et al. Interleukin-17A is dispensable for myocarditis but essential for the progression to dilated cardiomyopathy // Circ. Res. – 2010. – Vol. 106. – P. 1646–1655.
- [11]. Blauwet L.A., Cooper L.T. Myocarditis // Prog. Cardiovasc. Dis. – 2010. – Vol. 54 (2). – P. 274–288.
- [12]. Gussak I., Chaitman B.,Kopecky S., Nerborne J. Rapid ventricular repolarization in roden: electrocardiographic manifestation, molecular mechanisms, and clinical insights. J Electrocardiol 2000; 33(2): 159-170.
- [13]. Kawai C. From myocarditis to cardiomyopathy: mechanisms of inflammation and cell death // Circulation. – 2006. – Vol. 99. – P. 1091–1100.
- [14]. Maisch B., Portig I., Ristic A. et al. Definition of inflammatory cardiomyopathy (myocarditis): on the way to consensus // Herz. – 2000. – V. 25, №3. – P. 200–209.
- [15]. Silka M. Ambulatory electrocardiographic methods for the evaluation of cardiac arrhythmias in children. ACC Current Journal Review. 1999 8 Issue 5(10) p.61 63.