Electrocardiographic Manifestations of Neuroleptic Cardiomyopathy in its Different Clinical Stages

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Abstract

406 electrocardiograms recorded in 58 men and 23 women with schizophrenia receiving antipsychotics were studied retrospectively. 12 patients had no cardiac pathology, 44 ones had neuroleptic cardiomyopathy in the latent stage and 25 ones had the manifested disease (developed and terminal clinical stages). The frequency and lability of eight most common electrocardiographic signs are considered. It is established that conduction disorders and diffuse muscle changes are the most frequent and labile electrocardiographic signs observed in neuroleptic cardiomyopathy regardless of its stage.

Keywords: Neuroleptic Cardiomyopathy; Electrocardiographic Changes; Frequency and Lability Ones

Introduction

Neuroleptic cardiomyopathy (NCMP) is one of the most serious vitally dangerous complications of antipsychotic therapy [1–7], due to the side cardiotoxic effect of antipsychotic (neuroleptic) drugs [1–3, 5, 7–9].

The disease belongs to the secondary specific metabolic dilated cardiomyopathies [2, 4, 10, 11]. It is characterized by diffuse myocardial lesions, a sharp decrease in contractile function of cardiac muscle and, as a consequence, development of chronic heart failure [4, 6, 7, 12]. In its development NCMP passes three clinical stages: I – latent, II – deployed (manifesting) and III – terminal [4, 13].

Many aspects of the pathogenesis, morphology, clinic and diagnosis of NCMP are still almost completely unexplored. Among others, it is of practical interest to trace the dynamics of changes in the electrocardiogram (ECG) during the development of NCMP. This issue is still without due attention. However, to date, the method ECG is quite informative marker of the morphofunctional state of a heart, its measurement is usually not difficult, it is a routine method of examination of patients. Thus, for clinical practice, the study of ECG changes is extremely important, since they are one of the early signs of any developing heart disease [14–16].

In order to fill the existing gap at least partially, the present study has been undertaken.

Material and Methods

The data of medical records of 81 patients with schizophrenia who died (men – 58, women – 23) were retrospectively studied, almost two thirds of them were aged from 41 to 60 years.

Three groups of observations were identified: group I (comparison) – 12 patients receiving antipsychotic therapy, but did not have a cardiac pathology; group II – 44 patients with NCMP in the latent stage; group III – 25 patients with manifesting disease (developed and terminal clinical stages). The presence of NCMP was verified at autopsy. 406 ECG were analyzed in groups 53, 282 and 71, respectively. In each group, eight pathological electrophysiological parameters (ECG signs) were ranked by their frequency (MX). One of them is the corrected Qt interval (QTc) calculated by the formula H. C. Bazett [14, 15, 17].

To determine the degree of lability of one or another ECG sign, the difference index (MΔ) was determined, expressed as a percentage and

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obtained when comparing the values of MX in group I with those in subsequent groups of the study.

The received results were statistically processed (by the nonparametric Mann-Whitney’s U-criterion) with significance level of distinctions 95% and more (p≤0.05).

Pathological changes in ECG parameters due to the side cardiotoxic effect of AP reflect the processes of tissue changes in the myocardium of dystrophic-degenerative, sclerotic and compensatory-adaptive nature, which unfold in the heart muscle during the formation of the clinical and morphological picture of NCMP [18, 19, 20]. In short, these ECG changes are presented as follows (table 1).

**Table 1.** The frequency of ECG signs (MX) [%] in the course of morphogenesis NCMP.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Rhythm disturbances</th>
<th>Extension of the QTc interval</th>
<th>Conduction disturbances</th>
<th>Diffuse muscle changes</th>
<th>Overload of the right parts</th>
<th>Left ventricular hypertrophy</th>
<th>Reduction of myocardial electrical activity</th>
<th>EAH deviation to the left</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>72.3</td>
<td>5.7</td>
<td>5.2</td>
<td>2.6</td>
<td>3.1</td>
<td>2.2</td>
<td>1.3</td>
<td>5.0</td>
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<td>** ***</td>
<td>** ***</td>
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</tr>
<tr>
<td>II</td>
<td>64.5</td>
<td>8.5</td>
<td>21.6</td>
<td>26.2</td>
<td>11.7</td>
<td>8.9</td>
<td>8.2</td>
<td>14.2</td>
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<td>*</td>
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<td>*</td>
</tr>
<tr>
<td>III</td>
<td>67.6</td>
<td>35.2</td>
<td>50.7</td>
<td>32.4</td>
<td>31.0</td>
<td>7.0</td>
<td>8.5</td>
<td>19.7</td>
</tr>
<tr>
<td></td>
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<td>*** **</td>
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</tbody>
</table>

**Note:** * - statistically significant difference with the group I; ** - statistically significant difference with the group II; *** - statistically significant difference with the group III.

In the comparison group of observations on the ECG recorded primarily cardiac arrhythmias – 72.3%, mainly (69.8%) only in the form of sinus tachycardia. Only 2.5% of its background revealed a variety of types of extrasystole. Quite rarely there was an extension of the Qtc interval (5.7%), conduction disturbances (5.2%), mainly in the form of an incomplete blockade of the right leg of the GIS beam, the deviation of the electric axis of the heart (EAH) to the left (5.0%). Even less common are signs of overload of the right parts of heart (3.1%), diffuse muscle changes (2.6%), left ventricular hypertrophy (2.2%) and a decrease in electrical activity of the myocardium (1.3%).

At the first stage of the morphogenesis of NCMP, with stable functional compensation and the absence of a clinic of the disease (latent stage), the most common signs of this pathology, in addition to arrhythmias, are diffuse muscle changes, conduction disorders, deviation of EAH to the left.

In the presence of clinical manifestations of NCMP (developed and terminal stage of disease), also excluding arrhythmia, conduction disturbance, prolongation of the QTc interval and diffuse muscle changes were registered most often.

The frequency of rhythm disorders, although significantly predominates over that of other ECG signs, in all stages of the NCMP is not statistically different from that observed in the comparison group. In other words, arrhythmia, mainly sinus tachycardia, is a permanent consequence of antipsychotic treatment. This fact is confirmed in the literature [2, 3, 21, 22].

However, the MΔ of one or another ECG sign doesn’t fully show the dynamics of changes in its severity, that is, the degree of lability in the process of morphogenesis of the NCMP. This task is performed by MΔ.

The ranking of MΔ in order of increasing in the latent stage of NCMP gives the following picture (table 2): 1) arrhythmias, 2) prolongation of the QTc interval, 3) deviation of EOS to the left, 4) overload of the right parts, 5) left ventricular hypertrophy, 6) conduction disturbances, 7) decrease in electrical activity of the myocardium, 8) diffuse muscle changes.

In the manifest course of the disease MΔ of ECG-signs is ranked in ascending order: 1) rhythm disturbances, 2) left ventricular hypertrophy, 3) EAH deviation to the left, 4) extension of the QTc interval, 5) reduction of myocardial electrical activity, 6) conduction disturbances, 7) overload of the right parts, 8) diffuse muscle changes.

Thus, conduction disorders and diffuse muscle changes are the most frequent and the most labile ECG signs observed in NCMP regardless of its stage.
### Table2. The lability (МΔ) [%] of ECG signs in the course of morphogenesis NCMP.

<table>
<thead>
<tr>
<th>ECG signs</th>
<th>Rhythm disturbances</th>
<th>Extension of the QTc interval</th>
<th>Conduction disturbances</th>
<th>Diffuse muscle changes</th>
<th>Overload of the right parts</th>
<th>Left ventricular hypertrophy</th>
<th>Reduction of myocardial electrical activity</th>
<th>EAH deviation to the left</th>
</tr>
</thead>
<tbody>
<tr>
<td>M_1</td>
<td>1</td>
<td>10,8</td>
<td>49,1</td>
<td>315,4</td>
<td>907,7</td>
<td>277,4</td>
<td>304,5</td>
<td>530,8</td>
</tr>
<tr>
<td>Rank_MΔ</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>6</td>
<td>8</td>
<td>4</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>M_1</td>
<td>II</td>
<td>6,5</td>
<td>517,5</td>
<td>875,0</td>
<td>1146,1</td>
<td>900,0</td>
<td>218,2</td>
<td>553,8</td>
</tr>
<tr>
<td>Rank_MΔ</td>
<td>II</td>
<td>1</td>
<td>4</td>
<td>6</td>
<td>8</td>
<td>7</td>
<td>2</td>
<td>5</td>
</tr>
</tbody>
</table>

### Conclusion

The pathological changes in ECG, found in patients with schizophrenia, for a long time receiving antipsychotic therapy, reflect the processes of deep tissue changes, unfolding in the heart muscle during the formation of clinical and morphological picture of the NCMP.

It should be emphasized that in psychopharmacological therapy of schizophrenia there is a high risk of developing NCMP due to the side cardiotoxic effects of antipsychotics.

One of the most important elements of detection of cardiotoxic action of neuroleptics and prevention of this iatrogenic disease is a regular electrocardiographic study of such patients.

### References


