

Neuroleptic Cardiomyopathy: Let's Get Acquainted



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Received: 📅 September 05, 2018; Published: 📅 September 11, 2018

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Mini Review

The presented mini-review summarizes the results of our own research and rather rare literature data on the problem of neuroleptic cardiomyopathy. The aspects of etiology, epidemiology, clinical course, electrocardiographic signs and pathomorphology of the disease are briefly considered. The question of nosological independence of neuroleptic cardiomyopathy is raised. Neuroleptic (antipsychotic) cardiomyopathy (NCMP) is a little known to a wide range of doctors iatrogenic pathology. It is associated with a side cardiotoxic effect of antipsychotic drugs [1-9]. The disease belongs to secondary specific metabolic dilated cardiomyopathies [10-12] and is characterized by diffusion damage of a myocardium, by sharp decrease in its contractile function and by the progressing of congestive chronic heart failure (CHF) [8,10,11,13-17].

Many aspects of the epidemiology, pathogenesis, morphology, clinic and diagnosis of NCMP remain poorly studied. Therefore the purpose of this article, or should I say of mini-review, is to summarize the results of the own researches and some of the very few data of literature on this problem. Thus, although many antipsychotics, as classical, and atypical, can cause NCMP, the most common her cause is clozapine [18-22]. Frequency NCMP associated with taking clozapine is 0,02-0,1% [19, 23].

According to E. Wooltorton [24], 80% of their patients with clozapine-induced NCMP were younger than 50 years. As a rule, the manifestation of NCMP occurs after a sufficiently long reception of antipsychotic drugs [21,25]. According to a systematic literature review conducted by M. Alawami [18], symptoms of a disease appear on average 14.4 months after initiation of clozapine treatment. As showed my researches, in its development NCMP passes three clinical stages: 1) a latent one, it is clinically fully compensated, 2) a full-scale (developed. manifesting) one, when cardiac disorders are clearly detected, but without the expressed signs of CHF and 3) a terminal one, when the clinical picture of CHF comes to the foreground [13-15,26-28]. Mortality from NCMP is 12-18% [17, 24, 29]. The lethal outcome in latent and in developed stages either

comes from the intercurrent diseases or is the sudden cardiac death of arhythmogenic genesis [10,26,28]. The last is observed at 44.2 % of the dead of NCMP according to my data [31,30]. In a terminal stage the progressing CHF serves as an immediate cause of death [10,26,28].

The clinical course of NCMP is similar to that of idiopathic dilated cardiomyopathy [10]. The disease develops slowly and at the beginning is hardly noticeable. In a latent stage it is practically shown by nothing. During this period the complaints of patients have uncertain character or are in general absent. Fatigue and short wind at considerable physical activity is most often noted. Thus it must be kept in mind known difficulties of detection of complaints at mental patients connected both with their inadequate behavior and lack of due criticism to their state and with quite often certain medicament load.

a) In a latent stage of NCMP the findings of the examination are not numerous and aren't specific. The tachycardia serving as almost constant phenomenon at reception of neuroleptics [32] is observed as a rule. De an auscultation in is defined the deafness of cardiac sounds. Poorly the borders of heart are usually changed. An arterial hypo- and a norm tonicity significantly prevail from the arterial pressure. The insignificant arterial hypertension is only approximately in 1/3 cases observed [10,26]. On the electrocardiogram during this period there are most often the following pathological signs:

- a) Diffusion muscular changes;
- b) Different types of violation of conductivity, in particular the blockade of the left leg of Gis's bunch;
- c) Deviation of an electric axis of a heart to the left;
- d) Overload of the right departments of the heart;
- e) Hypertrophy of the left ventricle [10,11,14,15,17,33].

In the developed stage the clinic of NCMP is rather distinctly shown, but the signs of terminal CHF are absent or poorly noticeable. The complaints of patients are more certain: weakness, fatigue, heartbeating, short wind at moderate physical activity, sometimes passing pains in the cardiac region. By the physical examination the deafness of cardiac sounds, some expansion of the borders of a heart, tachycardia, passing breathlessness notes. During this period the steadily normal or labile arterial pressure equally often meets, but there is a tendency to the moderate increase of it [10,26]. The terminal stage of a current of NCMP is characterized by accession to already listed symptoms of the known manifestations of the increasing CHF: short wind at a rest or the small physical activity, orthopnea posture, the increase of a liver, the peripheral and cavitory oedemata, sometimes an anasarka etc. The heart borders are expanded that is confirmed by the roentgen exploration. The cardiac sounds are deaf. There are almost always a tachycardia and an arrhythmia. The moderate arterial hypertension is observed by a little more than at ¼ patients, and the arterial pressure constantly exceeds 150/100 mm of mercury at 13.6 % of patients [10,26].

On an electrocardiogram in developed and terminal stages of NCMP there are such most dangerous phenomena: 1) violations of conductivity; 2) lengthening of an interval of QT in recalculation on Bazett's formula - a correct QT interval (QTc); 3) overload of the right departments of the heart [10,11,14,15,17,33]. The special attention is deserved by monitoring of parameters of QTc as highly informative indicator in the conditions of a decompensation of the heart [34]. According to my morphological studies, HCMP is characterized by a moderate increase in the heart, a noticeable expansion of the ventricles with a certain prevalence of dilatation of the left one, the absence of severe coronary atherosclerosis, especially at the relatively aged persons (45 years and older). At microscopic research of a myocardium the expressed myofibrosis, the chronic interstitial oedema, in the beginning hypertrophic and then dystrophic - degenerative and atrophic changes of cardiomyocytes come to light. Thus NCMP possesses all signs of the concept "illness" that is has own epidemiology, etiology, pathogenesis, clinic and morphology. Proceeding from told about NCMP this pathology is allocated in separate independent nosological unit expediently and quite logical. On ICD-10 it can be carried to the heading I42.7 "The cardiomyopathy caused by influence of medicines and other external factors" with the additional code reflecting an external cause of illness (a class of antipsychotic preparations) - Y49.3-Y49.5 [10,34].

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