Abnormal Chords of Left Ventricle as Cardiac Manifestations of Connective Tissue Dysplasia Syndrome

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Abstract: The article highlights the major problems of diagnostics and clinical relevance of such minor structural heart abnormalities as left ventricular abnormal chords. Possible hemodynamic changes, clinical manifestations and cardiovascular complications associated with abnormal chords are presented. Abnormal chords of the ventricles (VAC) are connective tissue and muscular formations, which have ectopic attachment unlike normal chords. Abnormal chords are the most common cardiac manifestations of non-differentiated connective tissue dysplasia syndrome (CTDS). Commonly, they are associated with mitral valve prolapse as well as with other phenotypical manifestations of connective tissue dysplasia – joint hypermobility, increased skin extensibility, scoliotic posture, duplication and ptosis of kidneys etc. Modern views concerning the prevalence of this pathology, its clinical manifestations, diagnostic peculiarities as well as prognostic value of different topical and quantitative variants of abnormal chords are given. The effect of abnormal chords on arrhythmogenesis of left ventricle, development of cardialgia and myocardial ischemia development, heart cavity remodeling, systolic and diastolic function of the left ventricle are discussed. Pathogenetic relationship between multiple abnormal chords and such serious hereditary disease as left ventricular non-compaction is emphasized. The article focuses on the statement that the patients with left ventricular abdominal chords like all patients with minor structural heart abnormalities should be placed into "the risk group" because of possible development of ventricular arrhythmias, heart remodeling and chronic heart failure.

Keywords: Left ventricular abnormal chords, minor structural heart abnormalities, connective tissue dysplasia syndrome, left ventricular non-compaction.

Abnormal chords of the ventricles (VAC) are connective tissue and muscular formations (trabeculae, chords, "bundles"), which have ectopic attachment unlike normal chords. Real life-time diagnostics of abnormal chords became possible due to improvement of echocardiographic technique. Abnormal chords are visualized as echo-dense thin linear formations not connected with valvular apparatus of the heart Figure **1** (Figures **1**, **2**, **3**).

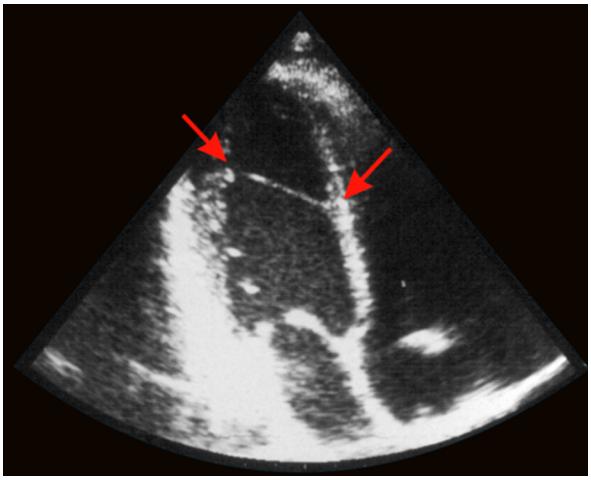

Figure 1: Abnormal transverse middle chord in the left ventricular.

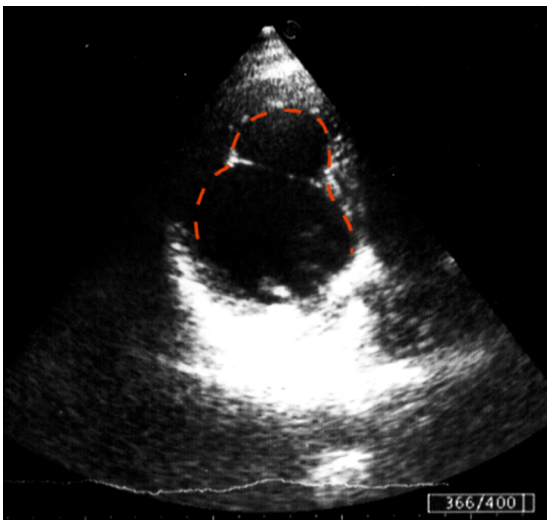

Figure 2: Deformation of the left ventricular cavity of abnormal chord.

Abnormal chords are the manifestation of connective tissue dysplasia syndrome (CTDS), its cardiac marker. The problem of connective tissue dysplasia syndrome attracts the attention of investigators because of high detection frequency of its signs in population as well as the risk for severe complications involving various organs and systems, but primarily the heart.

Mitral valve prolapse (MVP), left ventricular abnormal chords (LVAC) and their combinations are

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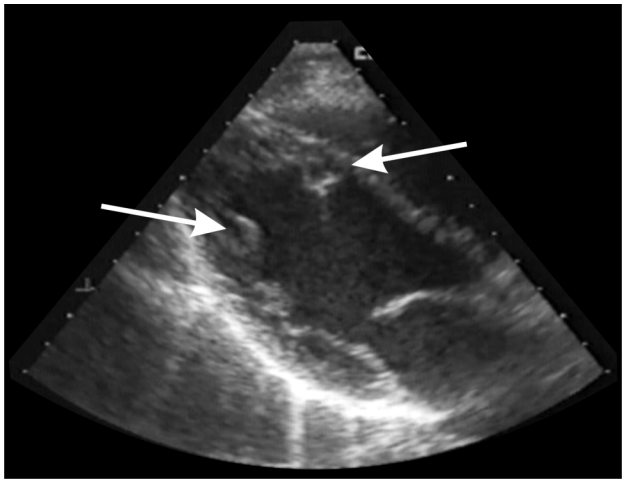

Figure 3: The broken abnormal chord of left ventricle.

the best studied cardiac manifestations of this syndrome. Prolapse of other valves, isolated aortic regurgitation, aortic annulus bulging, aneurism of pulmonary artery and interatrial septum (without bypass grafting) etc. are referred to the syndrome of non-differentiated connective tissue dysplasia as well. All these conditions are defined as "minor structural heart abnormalities" (MSHA). It should be mentioned that bicuspid aortic valve is not a minor structural abnormality despite its pathogenesis similarity to MSHA. It is referred to the list of congenital heart defects, hence there are differences in their prognosis.

The basis of connective tissue dysplasia is a hereditary determined defect of tissue structures manifested by the decrease of certain collagen content as well as imbalance in their proportions. This leads to the "weakness" of connective tissue "framework" of an organ or organs. These changes are usually manifested by the combination of phenotypical signs, which can be diagnosed at the first examination of the patient. They are as follows: body asymmetry (long upper and lower extremities), scoliosis, high degree of plat-foot, joint hypermobility and others.

Detection frequency of ventricular abnormal chords in population is 16% by autopsy findings and it is 1-68% according to echocardiographic findings [2, 3]. Such differences, undoubtedly, is the result of various methodologic approaches to the detection of abnormal chords and interpretation of the results obtained. Abnormal chords are located predominantly in the left ventricular cavity (95% of cases). In 5% of cases they are found in the right ventricle [4]. They may be isolated, comprising 62% of cases, and multiple, occurring in 38% of cases. LVAC are diagnosed more frequently in men (17-71%), than in women (17-30%), with no significant age differences. LVAC occur more often in young people and adolescents than in middle aged people and have more evident symptoms associated with hypersympathicotony [2, 4, 5]. LVAC are commonly diagnosed in patients with ischemic heart disease and dilated cardiomyopathy (25-43%), rheumatic heart lesions (38%), ventricular septum defects and other congenital heart diseases (15%) [6].

LVAC may be isolated or combined with other MSHA. According to many authors MSHA are associated with MVP in 53-68% [3, 5]. This implies the pathogenetic relationship between LVAC and MVP, allows to agree with the term offered by Glesbi M. "mass"-phenotype (combined phenotype) in such patients as well as to modify the "phenotypical range" in the following way: normal variant – LVAC – MVP – Marfan or Ehlers-Danlos syndrome [7].

No generally accepted classification of abnormal chords is available at present. In echocardiographic practice simplified classification is often used. According to it there are multiple and isolated LVAC, which, in their turn, are subdivided into apical, medial, transverse and less frequently – longitudinal and diagonal (depending on the localization in left ventricular cavity).

Ventricular abdominal chords are most commonly associated with such "markers" of connective tissue dysplasia as asthenic constitution type, kyphoscoliosis, chest deformity, increased skin extensibility, plat-foot, thumb and wrist sign, joint hypermobility, primary tracheobronchial dyskinesia, kidney disorders, venous valve insufficiency of lower extremity [2, 8, 9].

There may be lifelong absence of evident clinical manifestations or they may be presented as auscultation symptoms, pre-excitation syndrome, early ventricular repolarization syndrome, arrhythmia and dysfunction of the left ventricle.

LVAC usually cause systolic murmur predominantly at the heart apex, which is recorded in 72-100% of cases and can vary at the change of body position or on physical exertion [3, 7]. Murmur origin is likely to be associated with increased blood flow, blood flow turbulence and LVAC vibration if they are located at ventricular inflow and outflow tracts.

In 58-96% of patients with LVAC vegetative dysfunction symptoms are diagnosed [4, 5, 7, 8, 10, 11]. It is still unclear whether vegetative dysfunction is primary or whether it develops as adaptive response of

cardio-vascular system to minor structural abnormalities, as well as specific hemodynamics and connective tissue metabolism in these patients. Vegetative dysfunction is supposed to have constitutional, genetically determined character, mediated by hypothalamus influence, which has a dominant role both in collagen synthesis and neuro-humoral coordination and organization of adaptive behavior.

In patients with LVAC cardialgia is recorded in 42-65% of cases, palpitation - in 35-55%, arrhythmia - in 39-55%, weakness and rapid fatigue - in 60-65%, dizziness and loss of consciousness - in 10 -15%, dyspnea on exertion - in 15-25% [12]. The patients with isolated LVAC often have hyperventilation (55%) and syncopal syndromes (55%), termoregulation disturbances (50%), vascular abnormalities in the extremities (40%), nausea (38%), “lump in the throat” sensation (30%) [13]. Hemorrhagic syndrome in the patients with LVAC may be presented as nose bleeding tendency, gingival haemorrhage and bruises formation, being the evidence of mesenchymal dysplasia as well [14]. Psychopathic (panic) syndrome occurs predominantly in young patients and presents as nervous prostration, anxiety- phobic and affective disorders (hypochondria, depression, hysteria, psychasthenia) [10]. Such neurotic disorders are supposed to be related to excessive activity of hypothalamo-pituitary-adrenal axis and dysregulation of sympathoadrenal system.

There are several suggestions as to occurrence of LVAC associated with cardialgia: local ischemia caused by congenital coronary vessels abnormality, excessive extension and abnormal stretch of papillary muscles associated with hypersympathicotonia, pathologic sensitivity to adrenergic action of coronary vessels, decreased diastole duration associated with physical and emotional exertion as a result of tachycardia, deformity of left ventricular cavity by abnormal chords, sub-endocardial ischemia and microthromboembolism at the places of cardiac muscle thickening where LVAC goes etc. [3, 15]. According to Holter ECG monitoring (HMECG), LVAC of any localization are associated with increased maximal heart rate and circadian index, suggesting hypersympathicotonia in patients with MSHA, including those with LVAC, and followed by negative influence of hypercatecholaminemia on the myocardium [16]. HMECG findings in patients with LVAC concerning ST segment indicate possible relationship between pain syndrome and ischemic myocardium: ST segment depression is noted in 30.2% of patients with LVAC, the highest frequency rate is recorded in patients with multiple (64.3%) and isolated medial (46.4%) LVAC. Close correlation between duration of maximal episode of ST segment depression, calcinosis and thickening of LVAC ($r = 0.64$ and $r = 0.58$, respectively, $p < 0.01$), being determined predominantly in patients with multiple (64.3%) and thickened medial LVAC (78.6%), emphasizes the role of anatomical and degenerative characteristics of LNAC structure in the development of ischemic pain syndrome.

There is no single academic view at to the role and severity of electrocardiographic changes in patients with LVAC – it ranges from acknowledgement of adverse “arrhythmic” prognosis because of chords arrhythmogenicity [2, 5, 14-16] to flat denial of LVAC role in arrhythmia development [59]. Some authors acknowlege the only combination - “mitral valve prolapse – abnormal chord” as arrhythmogenic one, giving the dominant role to mitral valve prolapse and its severity [17], LVNC is being considered a normal variant. This ambiguous predictive valuation of LVAC is suggested to be the result of shallow analysis of this pathology without taking into account their localization, quantity and structural character.

Pre-excitation syndrome in LVAC is recorded in 12-28% of cases [13, 18]. The cause of ventricular pre-excitation development is thought to be the presence of conductance heart muscle fibers and Purkinje cells in LVAC. Therefore ventricular abnormal chords may become an additional channel of accelerated atrioventricular conduction with likely functioning of re-entry mechanism. Early ventricular repolarization syndrome (EVRS) is the most frequent ECG-syndrome in LVAC occurring in 70-72% of children and adolescents and 19-25% of adults [18, 19]. Early ventricular repolarization syndrome is considered to be the evidence of nonstable functioning of additional atriofascicular tract. It is commonly seen in patients with multiple LVAC. It is suggested that due to one of multiple LVAC the impulse is conducted from the upper third of interventricular septum to the apex and then to the anterior upper branch of His bundle, creating conditions for premature myocardial excitation.

Right His bundle branch block commonly associated with pre-excitation syndrome and early ventricular repolarization syndrome is the most frequent conduction disorder in patients with LVAC particularly in those with medial and multiple chords [18]. High additional conducting channels rate in

patients with MVP and LVAC is believed to be risk factor for arrhythmia development.

A number of authors found such LVAC complications as ventricular tachycardia or ventricular fibrillation being the indications for operative intervention [20, 21]. Strong direct correlation relationship between QT interval dispersion value and the rate of high gradation ventricular extrasystoles ($r = 0.52$, $p < 0.01$) and ventricular tachycardia episodes ($r = 0.58$, $p < 0.01$) was found in patients with MSHA, including those with LVAC [16, 18]. Increased QT interval dispersion, most commonly recorded in patients with multiple chords as compared to those with isolated LVAC of any localization (medial, apical, diagonal, longitudinal), may be regarded as ventricular arrhythmia predictor.

Premature ventricular contraction is the most clinically significant manifestation of LVAC. Extrasystolic arrhythmia, according to many authors, occurs in 70-100% of LVAC cases [2, 4, 14, 16, 22]. There is no agreement of opinions concerning the mechanisms of arrhythmia development in patients with LVNC. Some investigators state that LVAC are additional channels of excitation conduction leading to uncoordinated excitation of various left ventricular portions and contributing to ventricular arrhythmia development [5, 6, 20]. Other researchers consider that changes in electrophysiological characteristics of smooth muscular cells, as a result of chord deformity by turbulent blood flow because of their abnormal location at the outflow tract, are the likely cause of arrhythmia development mechanism [21, 22]. In arrhythmias the patients with LVAC permanently have an increased activity of sympathetic nervous system. The “central” arrhythmia origin should not be excluded as well, considering neurotic maladjustment type in patients with CTDS [10]. Additionally, one must note not identical degree of arrhythmogenic activity in patients with LVAC depending on their localization and number: the most severe ventricular arrhythmias are observed in patients with multiple chords, rather frequently – in patients with isolated, particularly thickened medial chords, less frequently – in patients with isolated LVAC of other localizations [16]. Ventricular extrasystoles (ES) of II-IY class by Lown classification, double ventricular ES and “couplets” are diagnosed predominantly in patients with multiple (35.3%) and medial (23.5%) LVAC. Arrhythmogenic activity of multiple and medial LVAC was manifested also by their high frequency rate (92-100%) and by daily number of supraventricular premature beats. Wandering pacemaker occurring three times as often in patients with LVAC as in patients without such pathology, is a likely cause of increased supraventricular arrhythmia rate.

To stratify arrhythmias and changes of left ventricular repolarization processes in LVAC the following increasing sequence may be suggested: normal variant – apical, diagonal and longitudinal isolated LVAC – medial LVAC – multiple LVAC – combination of LVAC and MVP [16].

Changes in connective tissue framework of the heart lead not only to abnormalities, but they are also the basis for the formation of special heart geometry, which is frequently observed in patients with LVAC and causes the symptoms of heart failure. Impaired left ventricular diastolic function in the presence of abnormal chords, according to many authors, is caused by the abnormal ventricular relaxation due to LVAC in its cavity [5, 7]. In young people with CTDS and multiple LVAC remodeling of left ventricle in the form of its dilation and lower LV contractile function are noted by contrast to young people of control group; it is followed by the symptoms of chronic heart failure in about half of the patients (46%) [23]. In isolated abnormal chords of any localization left ventricular geometry undergoes no significant changes. According to our data changes in LV diastolic function depend largely on the number of abnormal chords and their localization: 12% of patients with multiple chords have restrictive type of diastolic dysfunction, in 28% of patients with thickened medial transverse LVAC the changes are related to impaired relaxation dominate. The causes of recorded hemodynamic abnormalities in patients with LVAC may be various. They are supposed to be associated primarily with the changes of heart geometry. In its turn, the changes of heart geometry may be primary and conditioned by congenital defects of heart connective tissue framework, which progress together with the growth of the organism and acquire definite clinical significance. The markers of this pathologic process are LVAC and other MSHA. On the other hand, remodeling of heart cavities may be caused by local myocardial hypertrophy at the attachment sites of abdominal chords as well as by impaired myocardial relaxation interfered by LVAC, thickened transverse and medial ones in particular. The location of LVAC at the ventricular outflow tract can result in its partial obstruction with the development of gradient and excitation and contraction asynergy [24]. The chords themselves, being independent anatomical structures,

may induce remodeling processes in the left ventricle resulting in its dysfunction.

Prolonged and progressive left ventricular diastolic dysfunction with underlying connective tissue dysplasia syndrome may cause overload and dilation of the left atrium, leading to arrhythmia and increased pressure in pulmonary artery. "Multiplicity" of LVAC typically co-exists with dilation of left ventricular cavity associated with restriction of transmitral flow and, in a definite way, determines it [23]. One of the adverse events and potential consequences of left ventricular remodeling in patients with LVAC is simultaneous contrast syndrome, which proceeds thrombi formation and indicates the risk of such complication as thromboembolism. It should be noted that this phenomenon is recorded in patients predominantly with multiple LVAC as well as with a great number of visual dysembryogenic stigma, abnormal development of lower extremity veins, kidneys and gallbladder etc., that is with severe connective tissue dysplasia syndrome [25].

Simultaneous contrast effect may appear in multiple abnormal chords, because the chords themselves presumably become an obstacle on the way of blood flow, forming turbulent whirls and leading to thrombosis. We found in the literature the evidences of cerebral stroke and myocardial infarction occurrence in the patients with LVAC [26, 27]. According to some authors, the defect of heart connective tissue structures - abnormal chords in the left ventricle being its marker - may be associated with dysplastic aortic structure, which, in its turn, is the major cause of ascending aorta aneurism in young people [28, 29].

Until recently all cases of abnormal chords of heart ventricles were regarded exclusively as the manifestation of dysplasia of connective tissue heart structures, that is the variant of MSHA. Combination of LVAC and MVP with other MSHA confirmed this suggestion. However, these combinations occur in less than one third of patients with LVAC. Besides, histopathological examination of abnormal chords, notably multiple ones, detects not only connective tissue, but also muscular fibers with occasional predominance of the latter [3].

In the literature there are reports of multiple LVAC as characteristic sign and even diagnostic criterion of severe impairment of cardiac embryogenesis – the syndrome of left ventricular myocardial non-compaction or non-compacted left ventricle (NCLV).

Considering the fact that both connective tissue dysplasia syndrome and left ventricular non-compaction syndrome are proved hereditary diseases associated with impaired embryogenesis as a whole and cardioembryogenesis, these diseases are supposed to be the manifestations of one and the same pathologic process with various degrees of heart damage. But in both cases there is a common feature – multiple LVAC, which, on the one hand, are the marker of pathologic process, on the other – the pathogenic link between the appearance of both manifestations and complications.

The main distinctive feature of non-compacted myocardium is the presence of numerous bridges and trabeculae in the left ventricle and intratrabecular recesses between them lined with endocardium and communicating with left ventricular cavity. Multiple bridges and trabeculae form a wide non-compacted spongy layer of cardiac muscle, while the layer of true solid myocardium remains thin. Such abnormal structure leads to increased worsening of cardiac contractility and eventually to fatal heart failure.

Isolated non-compaction of left ventricle is a heart disease with rather definite morphologic and clinical manifestations. Recent increase of this pathology detection indicates not so much real LVNC prevalence, as the fact that often it remained undiagnosed, and its complications were established only in the terminal stage. Even after introduction of the term "left ventricular non-compaction" in 1990 this pathology was most frequently reported in analyzing the causes of sudden death, fatal ventricular arrhythmias and systemic thromboembolisms. Advances in echocardiography techniques, introduction of magnetic resonance imaging (MRI) and multispiral computed tomography allowed to diagnose this pathology even in asymptomatic patients.

Ultrasound examination of the heart is the main method of LVNC diagnostics. The presence of multiple abnormal chords in the LV cavity (three or more) is one of defining diagnostic echocardiographic criteria of LVNC [30].

Unfavourable prognosis and high mortality rate in this disease require its recognition on early stages as well as differentiated approach to the choice of treatment using modern, mainly surgical methods.

Thus, the patients with LVNC like all patients with minor structural heart abnormalities should be included

into the “risk group” because of possible development of high gradation ventricular extrasystoles, paroxysmal ventricular tachycardia, heart remodeling and chronic heart failure. Early diagnostics of abnormal chords and degree of manifestation of connective tissue dysplasia syndrome as well as the number of abdominal chords, their localization and anatomic structure allow to evaluate the prognosis and to develop the program of prevention and treatment of these patients.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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