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CLINICAL, ANAMNESTIC, AND INSTRUMENTAL CHARACTERISTICS OF ANOMALOUS LEFT VENTRICULAR CHORDS IN CHILDREN AND EVALUATION OF THEIR ARRHYTHMOGENIC POTENTIAL

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Abstract. Left ventricular anomalous chords (LVAC) are picked up surprisingly often in children, usually as a "by-the-way" finding on echocardiography, yet we still don't fully understand what they mean for the child's future health. The main unresolved question is whether these structures are just harmless anatomical quirks or whether, in some children, they help create an arrhythmogenic substrate, especially when they coexist with undifferentiated connective tissue dysplasia (CTD) and a history of perinatal stress. In this article, we pull together current data on how common LVAC are, how they are shaped and located, and how they appear on echocardiography, and we look more closely at the mechanisms that have been proposed to link them with repolarization changes, conduction disturbances, and ventricular arrhythmias. CTD is treated as a systemic "background condition" that may favor the formation of aberrant chords and subtle myocardial remodeling, making the ventricular myocardium electrically more vulnerable, at least in a subset of patients. We also consider how adverse perinatal factors such as intrauterine hypoxia, infection, or prematurity might nudge myocardial maturation and autonomic regulation in an unhealthy direction. On this basis, we outline an integrative, and admittedly still evolving, approach to risk stratification that combines chordal characteristics, clinical markers of CTD, perinatal history, and documented rhythm disturbances to plan more individualized follow-up rather than assuming that all LVAC can be safely dismissed as benign.

Keywords: Left ventricular false chords, minor cardiac developmental anomalies, undifferentiated connective tissue dysplasia, arrhythmias, WPW syndrome.

Introduction. Left ventricular false chords (LVFC) - sometimes called anomalous chords or abnormal fibrous strands are extra bands of connective or muscle-connective tissue that stretch across the cavity of the left ventricle [1,2,3,5]. What makes them "false" is where they attach. Instead of linking the atrioventricular valve leaflets to the papillary muscles like normal chordae, these strands latch onto the free wall of the ventricle or the interventricular septum.

Morphologically, clinicians usually describe them by where they sit and how they run through the cavity: along the long axis of the LV (longitudinal), across it (transverse), or diagonally. They are also classified by thickness, type, and where exactly they attach - closer to the base, the mid-ventricle, or the apex [1,2,4,5,7].

Additional left ventricular chordae (ACLV) are among the most frequent "minor anomalies" of cardiac development (MARS). Because they show up so often on echocardiography, we can't just shrug and ignore them - we have to keep asking what they actually mean clinically and what they imply for long-term prognosis [1,3,4-6]. A lot of recent work links ACLV to undifferentiated connective tissue dysplasia (UCTD), where they appear as one of the common cardiac signs [1,5,9,10]. UCTD is a systemic problem: the extracellular matrix doesn't form quite right, and that can produce structural and functional abnormalities not

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only in the heart (for instance, mitral valve prolapse) but also in the musculoskeletal system, kidneys, and other organs [10].

The clinical weight of left ventricular false tendons (LVFT) is still being argued about. For years many authors treated them as a basically benign anatomical quirk that doesn't raise the risk of ventricular arrhythmias or sudden cardiac death [1,5,6]. However, as more data have piled up, that simple picture has started to fray. LVFT may participate in pathological remodeling of the left ventricle, including both systolic and diastolic dysfunction, and can create a substrate for arrhythmias [1,2,3,7,8]. That possibility pushes us to view children with LVFT as a "risk group" for later chronic heart failure and ventricular arrhythmias [1,7,8].

In the cohort discussed here, potentially dangerous electrophysiological findings - including Wolff-Parkinson-White (WPW) syndrome - were identified. That immediately raises the stakes and makes a careful analysis of the clinical and functional profile of these children essential if we want to rethink how we stratify their arrhythmic risk [2,3,7,8,11]. If LVFT are one manifestation of a broader connective tissue dysplasia, then it is quite plausible that the dysplastic process involves both the working myocardium and the specialized conduction system of the left ventricle [2,3,9,10].

Objective of the study: to describe the clinical, historical (anamnestic), phenotypic, hemodynamic, and electrophysiological features in children with left ventricular false tendons, and to assess their clinical and prognostic significance, with particular attention to risk factors for arrhythmogenesis.

Materials and methods. The work was descriptive and retrospective-analytical. Data from 10 children (5 boys and 5 girls) aged 1-10 years were analyzed. All of them had a confirmed diagnosis of left ventricular apical hypertrophy (LVAH). All were born at term with normal birth weight, so we are not dealing with extreme prematurity or severe intrauterine growth restriction as confounders.

The medical histories were reviewed in detail. For the mothers, attention was paid to the course of pregnancy: anemia, acute viral illnesses, toxicosis, threat of miscarriage, and documented intrauterine infections such as herpes and cytomegalovirus (CMV). For childbirth we noted asphyxia, umbilical cord entanglement around the neck, weak labor, and premature birth.

Clinical examination included standard auscultation and systematic collection of complaints. Many of the reported symptoms fit the picture of vegetative-vascular dystonia (VVD): emotional lability, fatigue or poor tolerance to physical exertion, headaches, and dizziness. Cardiac-focused complaints were considered separately: chest pain, palpitations, and hyperventilation episodes.

Because the suspected problem is systemic connective tissue dysplasia (CTD), a phenotypic assessment was performed. The team looked for asthenic body habitus, skin hyperelasticity, joint hypermobility, and small developmental anomalies such as shortened fifth fingers or unusual ear shape.

Two-dimensional echocardiography was the main non-invasive tool to confirm left ventricular false tendons: thin, linear, usually longitudinal structures crossing the LV cavity, clearly not attached to the mitral valve leaflets. At the same time, standard dimensional parameters were measured - end-diastolic and end-systolic dimensions (EDD, ESD), wall thickness (posterior wall and interventricular septum, PW and IVS), and the size of the right ventricle and right atrium (RV, RA). Functional status was assessed via ejection fraction (EF) and left ventricular end-diastolic volume (LV EDV).

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A standard 12-lead ECG was used to pick up rhythm disorders (e.g., extrasystoles, WPW), disorders of automaticity (sinus tachycardia or bradycardia), and conduction disturbances (first-degree AV block, incomplete right bundle branch block, IRBBB). Particular focus was given to changes in ventricular repolarization.

Results. A striking finding in this small group was how often the obstetric and perinatal histories were complicated. The overall etiologic "load" was high and seemed to point toward an increased risk of connective tissue dysplasia.

During pregnancy, anemia and acute viral infections were present in all mothers (100%), pregnancy toxicosis in 70%, and threat of miscarriage in 50%. Intrauterine infections (herpes and CMV) were documented in 40% of cases.

Problems during labor were also frequent: asphyxia in 60%, the umbilical cord around the neck in 50%, and weak labor in 30% of births (Table 1). Taken together, the fact that all children in the cohort experienced significant prenatal stress strongly suggests that the observed chordal and connective tissue anomalies are, at least in part, a consequence of disrupted connective tissue development under adverse intrauterine conditions. This strengthens the proposed pathogenetic link between perinatal pathology and systemic dysplasia, even though, with only 10 patients, we should be cautious about over-generalizing.

Table 1. Maternal obstetric and gynecological history and childbirth characteristics (n=10).

Factor	Absolute number	%
Pregnancy pathology (total)	10	100
Anemia during pregnancy	10	100
Acute viral diseases	10	100
Pregnancy toxicosis	7	70
Threat of miscarriage	5	50
Presence of intrauterine infections (herpes, CMV)	4	40
Birth asphyxia	6	60
Umbilical cord wrapped around neck	5	50
Weak labor	3	30

Systemic signs of CTD were obvious in many children. The most frequent phenotypic markers were skin hyperelasticity (60%) and joint hypermobility (50%) (Table 2). An asthenic body type was noted in 40%. These features support the idea that what we are seeing is not just an isolated cardiac oddity but a systemic connective tissue disorder in which left ventricular false tendons are simply one visible, cardiac "marker."

Table 2. Phenotypic manifestations of connective tissue dysplasia in children with LCHD (n=10).

Sign	Abs	%
Asthenic constitution	4	40
Skin hyperelasticity	6	60
Joint hypermobility	5	50
Short pinky finger	2	20
Flatfoot	1	10
Changes in the shape of the auricle	1	10

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Complaints were dominated by symptoms of autonomic dysfunction. Emotional instability was recorded in 80% of children, poor tolerance of physical exertion in 60%, and headaches and dizziness in 40% each - a clinical combination that sits squarely in the vegetative-vascular dystonia (VVD) spectrum. Cardiac complaints included palpitations (40%) and chest pain (30%). The pain was stabbing, appeared after physical activity, and did not require medication. Put together, the overall clinical profile - autonomic dysfunction on the background of CTD - encourages us to view LV false tendons not as a standalone defect but as part of a broader systemic picture.

On auscultation, every child had a systolic murmur of the so-called "chordal squeak," with a maximal point at the apex of the heart. In practical terms, this is useful: such a murmur may serve as a relatively sensitive signal that an instrumental examination (especially echocardiography) is warranted.

Echocardiography confirmed thin, linear, longitudinal structures crossing the LV cavity without attachment to the mitral valve leaflets. Systolic function by ejection fraction was preserved in all children, with LVEF values between 56% and 73%.

The volumetric parameters, however, told a more subtle story. Left ventricular end-diastolic volume ranged from 11 to 51 ml (Table 3). In several children, LV EDV exceeded 20 ml despite a normal ejection fraction. This pattern was interpreted as a mild impairment of diastolic function and may represent an early marker of LV remodeling. That interpretation fits with what is known about how left ventricular apical hypertrophy or aneurysm can alter the functional state of the ventricle, even before systolic function visibly falls.

Table 3. Average echocardiography indicators in children with left ventricular apical aneurysm.

Indicators	**Left ventricular apical aneurysm (Range)**
EDD (mm)	17-37
ESD (mm)	11-26
LVPW (mm)	4/5-6/8
IVS (mm)	4-8
Heart rate	85-179
EF (%)	56-73
LV EDV (ml)	11-51
RV (mm)	11-17
RA (mm)	14-28
PA annulus (mm)	10-17

ECG findings were also far from trivial. There were not only autonomically driven rhythm variants but also problems with potentially serious implications. Disorders of automaticity included sinus tachycardia in 80% of children and sinus bradycardia in 20%. Conduction disorders were less common but still present: first-degree AV block in 10% and incomplete right bundle branch block in 10%.

The most worrying result was the very high rate of myocardial repolarization abnormalities (90%) and the detection of WPW syndrome in 10% of patients (Table 4). WPW is clinically significant because it carries a real risk of life-threatening arrhythmias, including ventricular fibrillation.

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Table 4. Frequency of rhythm and conduction disorders on ECG in children with left ventricular apical aneurysm (n=10).

ECG finding	Abs	%
Sinus tachycardia	8	80%
Sinus bradycardia	2	20%
Extrasystoles	3	30%
WPW syndrome	1	10%
1st degree AV block	1	10%
Incomplete right bundle branch block	1	10%
Myocardial repolarization abnormality	9	90%

The high frequency of repolarization changes was interpreted as a sign of unstable function of an additional atriofascicular pathway. The presence of WPW in a child with LV false tendons suggests that the false tendons are not always an innocent bystander. Instead, they may be part of a structurally and electrically unstable substrate that predisposes to arrhythmias.

Discussion. Looking at the histories and phenotypes together, LV false tendons in these children seem less like a random anatomical extra and more like one manifestation of a connective tissue disorder acquired very early in ontogenesis. Maternal anemia, viral infections, and other pregnancy complications, combined with a high frequency of birth asphyxia, point to significant hypoxic and infectious impacts during critical periods of organ development. These factors can disrupt embryogenesis, especially the formation of the extracellular matrix, and lead to systemic connective tissue dysplasia.

Because the conduction system, valvular apparatus, and myocardium are all rich in specialized connective tissue, dysplastic changes in them are a predictable extension of this systemic process. In that context, LV false tendons can be seen as a morphological reflection of this "immaturity" of the connective tissue framework.

Clinically, the dominance of autonomic dysfunction symptoms (emotional lability, sinus tachycardia) is typical of undifferentiated connective tissue dysplasia (UCTD). These symptoms hint at disturbed autonomic regulation, which is often closely intertwined with dysplastic connective tissue changes (for example, increased joint laxity, venous tone abnormalities, and so forth).

From a hemodynamic standpoint, the preserved ejection fraction could look reassuring if taken in isolation. However, the increased LV end-diastolic volumes point toward early, subclinical diastolic dysfunction. Reduced ventricular compliance could be due to the mechanical influence of the false chord itself (acting as an internal "guy-wire" that restricts motion) or to more diffuse myocardial dysplasia with areas of interstitial fibrosis. If left ventricular apical hypertrophy is indeed triggering early diastolic changes, that would fit with the broader idea that diastolic impairment can be a more sensitive marker of unfavorable remodeling than systolic dysfunction and may precede overt chronic heart failure.

The electrophysiological findings, particularly WPW syndrome in 10% of this small cohort, are central for prognosis. WPW arises from an additional conduction pathway between the atria and the ventricles. In the wrong circumstances - say, a child develops atrial fibrillation during a high fever - this accessory pathway can transmit impulses very rapidly to the ventricles, precipitating ventricular fibrillation and sudden cardiac death.

This observation pushes back against the convenient notion that additional left ventricular chords are always benign. The same dysplastic process that produced the false tendons may also

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have altered the cardiac conduction system, resulting in both anatomical anomalies (LV false tendons) and electrophysiological ones (WPW and repolarization abnormalities). In that sense, the presence of false tendons may serve as a visible marker of a deeper, more global electrical vulnerability rather than a harmless curiosity.

The extremely high rate of repolarization disturbances (90%) suggests that the myocardium's electrical behavior is, at the very least, unstable. One can speculate about local ischemia or tension forces from the chordae as contributors, but in the setting of WPW many authors lean toward an explanation centered on the functional instability of accessory conduction pathways. Regardless of the exact mechanism, these findings together make it hard to classify additional left ventricular chords in children with CTD and ECG changes as purely benign.

Of course, there are limits here. The series is small, retrospective, and drawn from a group already selected for having cardiac pathology. We cannot assume that every child with an incidental false tendon and no other CTD features carries the same risk. Still, the pattern is concerning enough to justify more cautious follow-up.

Conclusions. Abnormal left ventricular chords in children are closely linked to undifferentiated connective tissue dysplasia and significant perinatal stress. Clinically, they most often present against the background of autonomic dysfunction, and a characteristic auscultatory sign is the systolic "chordal squeak" at the apex. Even with preserved systolic function, mild disturbances of left ventricular diastolic function are detectable, suggesting early myocardial remodeling and arguing for long-term hemodynamic monitoring rather than one-time reassurance. Frequent electrophysiological abnormalities, especially repolarization changes and the presence of WPW syndrome - support classifying children who have both additional left ventricular chords and signs of CTD and/or ECG changes as a high-risk group. For these patients, more detailed risk stratification and closer follow-up are justified to reduce the likelihood of severe, potentially fatal arrhythmias.

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