Case Report

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Case Report

Left Ventricular Hypertrabeculation (LVHT) In Athletes: Negligible Finding?

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Abstract: Left ventricular hypertrabeculation (LVHT) used to be a rare phenotypic trait. With advances in diagnostic imaging techniques, LVHT is being recognised in an increasing number of people. The scientific data shows a possibility of over-diagnosing this cardiomyopathy in a population of people who have very high physical activity. We described the case of a young athlete with no medical history who presented with syncope during a marathon running race. Initial evaluation showed elevated troponin I, transthoracic echocardiography showed a trabeculated ventricle and subsequent cardiac magnetic resonance (CMR), revealed left ventricular hypertrabeculation (LVHT). During subsequent evaluation by tilt table testing, vasovagal syncope was identified as the likely aetiology of syncope. The patient was advised to cease sports and stimulants like caffeine use. At the 29-month follow-up, CMR showed normalisation of the non-compacted to compacted myocardial ratio and improvement in left ventricular function, with no further syncopal episodes reported. This is an example of physiologic hypertrabeculation of the LV apex in a recreational endurance athlete with normalisation of non-compacted to compacted myocardial layer ratio after detraining. Physiologic hypertrabeculation, a benign component of exercise-induced cardiac remodelling, must be differentiated from noncompaction cardiomyopathy and other pathologies causing syncope. This case underscores the importance of distinguishing physiologic hypertrabeculation from pathological LVHT in athletes, highlighting that exercise-induced cardiac remodelling can normalise with detraining.

Keywords: left ventricular hypertrabeculation; athlete's heart; sports medicine; cardiac magnetic resonance imaging; non-compaction

1. Introduction

Left ventricular hypertrabeculation (LVHT) or non-compaction (LVNC) is characterised by prominent left ventricular trabeculae and deep intertrabecular recesses [1]. The American Heart Association considers LVNC as a genetic cardiomyopathy [2]. Until the release of the 2023 ESC Guidelines for the management of cardiomyopathies [3], the European Society of Cardiology categorised LVHT (LVNC) as unclassified cardiomyopathy [4]. Currently, The Task Force does not classify left ventricular non-compaction as cardiomyopathy but as a phenotypic trait that may occur alone or with other conditions, such as developmental abnormalities, ventricular hypertrophy, dilation, or systolic dysfunction. Due to the absence of morphometric evidence for ventricular compaction in humans [5,6], the term "hypertrabeculation" is preferred, especially when the condition is transient or appears in adulthood. Non-familial and sporadic forms have been described in highly trained athletes [7].

The clinical presentation of LVHT varies from asymptomatic patients to patients with ventricular arrhythmias, thromboembolism, heart failure, and sudden cardiac death. However, there is increasing data about over-diagnosing this cardiomyopathy in an athletic population due to the physiologic adaptation to the extreme preload and afterload conditions characteristic of intense athletic participation [8].

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Gold standard diagnostic criteria for LVHT are currently lacking, and proposed imaging-based criteria have created an epidemic of overdiagnosis in low-risk populations. Athletes suffer significant false-positive rates, and current evidence suggests that this entity may be related to cardiac adaptation to increased preload. We present a clinical case of physiological hypertrabeculation of the left ventricle in an athlete and the contemporary knowledge and current controversies regarding left ventricular hypertrabeculation.

2. Case Presentation

A 21-year-old male, with no prior medical history, was hospitalised after a syncope that occurred during the marathon race. He lost consciousness after running 8 kilometres. Currently, the patient does not take any medications regularly, but during adolescence, he took various supplements while exercising. He denies any recent viral illness. There is no family history of premature cardiovascular deaths. Physical examination on admission was within normal limits, while electrocardiogram (ECG) showed the partial right bundle branch block. Additionally, blood tests indicated elevated cardiac biomarkers (troponin I, creatine kinase MB fraction, B-type natriuretic peptide, myoglobin), which could suggest myocyte and muscle injury as a consequence of exercise-induced stress (Table 1).

Table 1. Results of laboratory tests on admission and at follow-up. RV - reference value used in Vilnius University Hospital Santaros Clinics (VUL SK) laboratory, Vilnius, Lithuania. CK - creatine kinase, CK-MB - creatine kinase MB fraction isoenzyme, BNP - B-type natriuretic peptide, AST/GOT - aspartate aminotransferase/glutamate oxaloacetate transaminase, ALT/GPT - alanine aminotransferase/glutamate pyruvate transaminase.

Laboratory test	Results at presentation	Follow-up after 24 hours	Follow-up after 72 hours	Reference value (male)
CK (U/L)	545.0	679.0	-	25.0-195.0
CK-MB (μg/L)	6.14	-	-	<5.2
Myoglobin, (μg/L)	455.9	-	-	<155.0
Troponin I (ng/L)	4456.4	2733.4	-	≤35.2
BNP (ng/L)	173.8	66.6	-	<100.0
D-dimers (μg/L)	500.0	-	-	<500.0
AST/GOT (U/L)	-	376.0	95.0	<40.0
ALT/GPT (U/L)	-	428.0	368.0	<41.0
Potassium (mmol/L)	4.3	4.5	5.0	3.5–5.1
Sodium (mmol/L)	140.0	140.0	141.0	136.0–145.0
Creatinine (μmol/L)	121.0	98.0	99.0	62.0–115.0

Initially, transthoracic echocardiography was performed (Supplemental Videos 1-3). It showed prolapse of both mitral valve cusps with mild mitral insufficiency. Additionally, Ist degree left atrial dilatation and nondilated left ventricle with a normal ejection fraction of around 61% (as calculated by biplane Simpson) with an increased trabeculation of both left and right ventricles was noticed.

Video I. Transthoracic echocardiography: 4 chamber heart view. **Video II.** Transthoracic echocardiography: modified 4 chamber heart view with zoomed both ventricles. **Video III.** Transthoracic echocardiography: short axis at the midventricular level.

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To exclude coronary artery pathology or anomalies computed tomography angiography was performed (Supplemental Video 4). No changes in coronary vessels were found. Cardiac magnetic resonance (CMR) was performed to assess structural and functional cardiac changes and to exclude cardiomyopathies. The latter test showed slight LV dilatation (left ventricular end-diastolic volume index (EDVI) 104 ml/m² (normal range <92), left ventricular end-systolic volume index (ESVI) 47 ml/m² (normal range <30), borderline LV systolic function (55 %, normal range >55%), and left signs of ventricular non-compaction. Non-compacted to compacted myocardium ratio was up to 2.6 in diastole (Supplemental Videos 5-7, Figure 1 A-C). According to Petersen's criteria, a normal ratio of non-compacted to compacted myocardium should be <2.3 in diastole [9]. A nonspecific focus of fibrosis was also detected on late gadolinium enhancement images at the inferior left and right ventricle insertion points (Figure 1C, white arrow).

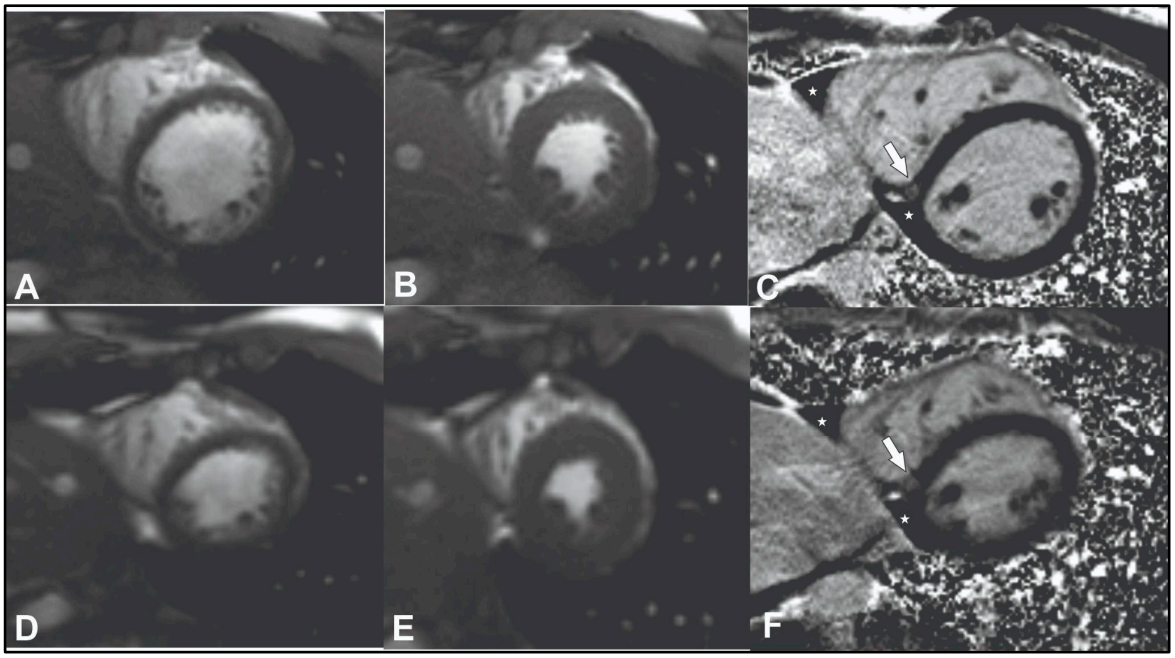

Figure 1. CMR images of the patient at admission (A-C) and follow-up (D-F). Cine short-axis images at the midventricular level at diastole (A) and systole (B), representing left ventricular hypertrabeculation. respective midventricular slice in inversion recovery sequence 10-15 minutes after contrast media (late gadolinium enhancement sequence) injection (arrow pointing to the fibrotic focus at inferior left and right ventricle insertion point, asterisk represents traces of fluid in the pericardial space at inferior cardiac wall). Follow-up cine short-axis images at the midventricular level at diastole (A) and systole (B), representing normalization of left ventricular hypertrabeculation and a decrease in left ventricle volumes after detraining. On a late gadolinium enhancement image the same findings as in C were noticed.

Video IV. Coronary artery computed tomography angiography representing normal coronary arteries

After a comprehensive review of the patient's medical history, it was determined that the patient had experienced positional syncope. As a result, a tilt table test was performed, resulting in the diagnosis of mixed-type vasovagal syncope. 24-hour Holter monitoring was performed and showed sinus rhythm with a heart rate (HR) of 28 – 91 beats per minute (bpm). Short bradycardia episodes with HR less than 25 bpm were observed. The average daily HR was 41 bpm.

Video V. Cardiac magnetic resonance cine (SSFP) sequence at admission: 4 chamber heart view representing left ventricular and right ventricular hypertrabeculation. **Video VI.** Cardiac magnetic resonance cine (SSFP) sequence at admission: short axis view at midventricular to an apical level representing an increase in left ventricular noncompacted to compacted layer ratio in lateral and inferior walls. **Video VII.** Cardiac magnetic resonance cine (SSFP) sequence at admission: short axis

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view at an apical level representing an increase in left ventricular noncompacted to compacted layer ratio in all walls except septum.

At that time, it was concluded that the patient may have physiological remodelling of the left ventricle due to his high activity in endurance sports. Also, it was strongly recommended to discontinue the use of various stimulants such as caffeine and reduce his physical activity from high to moderate or/and mild in order to reduce the incidence of syncope. No anticoagulants or antiaggregants were prescribed.

After 2 years of follow-up, the patient underwent follow-up examination and additional CMR which revealed reverse remodelling of the left ventricle with a decrease in left ventricular dilatation (EDVI from 104 to 97 ml/m² (normal range <92), ESVI from 47 to 40 ml/m² (normal range <30), LV ejection fraction within the normal range (58%) and left ventricular myocardium with normal non-compacted to compacted myocardium ratio in diastole – 1.9 (normal range <2.3) (Figure 1 D-F, Supplemental Videos 8-10). The patient reported no recurrence of syncopal episodes.

Video VIII. Cardiac magnetic resonance cine (SSFP) sequence at 2 years follow-up: 4 chamber heart view representing a decrease in left ventricular end-diastolic diameter and noncompacted to compacted layer ratio. **Video IX.** Cardiac magnetic resonance cine (SSFP) sequence at 2 years follow-up: short axis view at midventricular to an apical level representing a decrease in left ventricular end-diastolic diameter and noncompacted to compacted layer ratio. **Video X.** Cardiac magnetic resonance cine (SSFP) sequence at 2 years follow-up: short axis view at an apical level representing a decrease in left ventricular end-diastolic diameter and noncompacted to compacted layer ratio.

4. Discussion

Left ventricular hypertrabeculation (LVHT) is a complex and under-researched heart condition. The diagnosis of LVHT has increased significantly due to advancements in imaging techniques, yet there remains considerable debate over its diagnostic criteria and management [10]. Clinicians and scientists around the globe have advanced our understanding of the genetics, diagnostics, therapeutics, and outcomes for adult and pediatric patients with LVHT. Recent studies have shown that LVHT, previously underestimated in prevalence, is more common than thought, affecting 0.14% to 0.27% of the general population and 9.5% of children with cardiomyopathies [10]. The condition is diagnosed more frequently due to improved awareness and imaging technology. Isolated LVHT has been found in up to 8% of athletes, suggesting a potential overlap with physiological adaptations in this group [10].

Clinically, left ventricular hypertrabeculation (LVHT) can present with a wide range of symptoms, from none to heart failure, palpitations, chest pain, and, in rare cases, arrhythmias or sudden cardiac death [8]. Syncope with exertion is particularly common, possibly due to the increased demands on the body, especially during elite athletic events. On physical examination, vital signs may show bradycardia, hypotension, or tachyarrhythmias. A cardiac examination is often normal but may reveal arrhythmias, murmurs, signs of left ventricular hypertrophy (LVH), or congestive heart failure [8]. In some cases, isolated LVHT could be an adaptive response to training and may not require further evaluation or restrictions from sports if the patient is asymptomatic and has no concerning findings (e.g., arrhythmias, abnormal stress test, or depressed left ventricular function). However, symptomatic patients should be excluded from sports and closely monitored for potential risks such as heart failure, sudden cardiac death, and thromboembolism.

The prognosis of left ventricular hypertrabeculation (LVHT) remains uncertain, despite it being recognised as a clinical condition for over 30 years. Recent studies indicate that overall survival is lower in patients with LVHT compared to the expected survival of age- and sex-matched individuals from the general U.S. population. However, patients with preserved left ventricular ejection fraction and isolated apical noncompaction have a survival rate similar to that of the general population [11,12].

Guidelines from the American Heart Association (AHA) and American College of Cardiology (ACC) recommend that asymptomatic athletes with normal systolic function and no significant

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arrhythmias may participate in competitive sports [13]. However, those with impaired function or arrhythmias should be limited to low-intensity sports until more data are available.

A recent study involving 1,492 Olympic elite athletes across various sports disciplines who underwent electrocardiograms, echocardiograms, and exercise stress tests found that left ventricle trabeculations (LVT) were common, occurring in 29% of participants, particularly in male, Afro-Caribbean, and endurance athletes [14]. LVTs in this population were interpreted as a manifestation of adaptive remodelling associated with elite athletic training. The study concluded that in the absence of clinical abnormalities such as left ventricular systolic or diastolic impairment, electrocardiogram repolarization abnormalities, or a family history of cardiomyopathy, LVTs in athletes are of benign clinical significance and do not require further investigation. In addition, this prospective study suggested that recreational marathon running does not increase left ventricular trabeculation [15]. However, further investigations are needed to confirm this hypothesis.

Applying cut-off values from published LV hypertrabeculation criteria to young, healthy individuals has a potential risk for overdiagnosis. It has previously been shown that younger individuals possess greater amounts of apical trabeculation [16,17] but age-specific normative or cut-off values for pathological LV trabeculation do not currently exist. In this sample of healthy subjects, excessive trabeculation was found predominantly at the LV apex, which is recognized as the most commonly non-compacted segment [9] and was detected with greater sensitivity by Chin [18] and Captur [19] using apical fractal dimension (FD) criteria. A higher prevalence of positive Chin [18] as compared to Jenni [20] criteria has previously been reported in Olympic athletes with prominent trabeculation [21]. The fractal dimension (FD) quantifies how thoroughly a complex structure fills space. Its value is constrained by the structure's topological dimension. For example, in two-dimensional imaging, endocardial borders are more complex than straight lines, giving them an FD greater than 1. However, since they don't completely occupy the two-dimensional space, their FD remains below 2. Thus, the FD for an endocardial border consistently falls between 1 and 2, representing a non-integral value. In left ventricular hypertrabeculation (LVHT), excessive trabeculations result in a highly irregular endocardial border. Fractal analysis of these borders in LVHT is expected to produce a higher FD compared to normal hearts.

Studies in athletes have reported prominent trabeculation, raising concerns about a diagnostic grey zone between LVHT and exercise-induced remodelling [22]. Severe cases in clinical practice suggest LVHT might be a distinct pathology, as the extent of trabeculation exceeds what could arise from adaptation alone. While adult myocardium can remodel via myocyte hypertrophy, its limited proliferative capacity makes extensive de novo trabeculation unlikely. Most cases likely reflect normal trabeculation variations influenced by ventricular geometry and loading conditions. Clinical evaluations, including symptoms, family history, function, arrhythmias, and CMR findings, suggest only 0.1% of cases align with LVHT, with most being normal or non-cardiomyopathic. This supports the idea that prominent trabeculation in athletes is often exercise-induced remodelling.

In this case, insertion point fibrosis was detected on late gadolinium enhancement images at the inferior left and right ventricles (Figure 1C, white arrow). Insertion point fibrosis is frequently observed in athletes irrespective of age [23,24]. One hypothesis suggests this pattern may result from pressure or volume overload in the right ventricle during intense exercise, causing microinjuries that manifest as late gadolinium enhancement (LGE) [25]. Generally, it is considered to be benign and non-prognostic [26]. In healthy elderly individuals, insertion point fibrosis may represent a normal ageing process and is often deemed an incidental finding when unaccompanied by other signs of cardiac damage [27].

Increased cardiac troponin levels following exercise have been documented in athletes across various sports [28,29]. These increases are temporary and generally return to baseline within 48-72 hours after exercise [30]. While the exact mechanisms behind exercise-induced troponin release remain unclear [31], recent research suggests that activities such as marathon running may impact cardiomyocyte integrity [32], potentially leading to the leakage of cytosolic troponin fragments [33]. Further investigation is required to determine whether specific groups (based on factors such as age,

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sex, or sports discipline) with exercise-induced troponin elevations might be at heightened cardiovascular risk [34].

In this case, LV hypertrabeculation reached its maximum expression at the peak of physical conditioning and decreased two years after the detraining period. This suggests that LV hypertrabeculation occurred during the marathon training period as a response to the intensity and volume of the training load. These changes represent adaptive mechanisms that can regress during detraining.

5. Conclusions

Isolated LVHT may be an adaptive mechanism to training and may not require further evaluation or restriction from sports if the patient is asymptomatic and there are no other high-risk features (e.g., depressed LV function). Symptomatic patients should be barred from sports participation and followed closely for risk of heart failure, sudden cardiac death, and thromboembolism. Latest data suggests that left ventricular hypertrabeculation can be induced by exercise and it represents a normal physiological adaptation in the athletic heart. In this case, an increased preload resulted in eccentric left ventricular (LV) remodelling, accentuating trabeculations. After the detraining period, eccentric LV remodelling was reverted to normal (as evidenced by a decrease in the end-diastolic volume and its index), and the trabeculations collapsed at their base. Consequently, the increased non-compacted to compacted myocardium ratio can no longer be measured. Isolated hypertrabeculation, without associated clinical or imaging risk factors, is not recognised as a definitive indicator of elevated cardiac risk. Meanwhile, caution should be exercised before diagnosing LVHT in an athlete based solely on trabeculation appearance.

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Informed Consent Statement: Informed consent was obtained from all subjects involved in the study. Written informed consent has been obtained from the patient(s) to publish this paper**.**

Data Availability Statement: The data presented in this study are available on request from the corresponding author.

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