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EDITORIAL

Hypertrophic cardiomyopathy and left ventricular non-compaction: Distinct diseases or variant phenotypes of a single condition?

Natalia Przytuła, Ewa Dziewięcka, Mateusz Winiarczyk, Katarzyna Graczyk, Agnieszka Stępień, Paweł Rubiś

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Abstract

Hypertrophic cardiomyopathy (HCM) is a genetically determined myocardial disease characterized by an increased thickness of the left ventricle (LV) wall that cannot be solely attributed to abnormal loading conditions. HCM may present with an intraventricular or LV outflow tract obstruction, diastolic dysfunction, myocardial fibrosis and/or ventricular arrhythmias. Differentiating HCM from other diseases associated with LV hypertrophy, such as hypertension, aortic stenosis, or LV non-compaction (LVNC), can at times be challenging. LVNC is defined by excessive LV trabeculation and deep recesses between trabeculae, often accompanied by increased LV myocardial mass. Previous studies indicate that the LVNC phenotype may be observed in up to 5% of the general population; however, in most cases, it is a benign finding with no impact on clinical outcomes. Nevertheless, LVNC can occasionally lead to LV systolic dysfunction, manifesting as a phenotype of dilated or non-dilated left ventricular cardiomyopathy, with an increased risk of thrombus formation and arterial embolism. In extreme cases, where LVNC is associated with a very thickened LV wall, it can even mimic HCM. There is growing evidence of an overlap between HCM and LVNC, including similar genetic mutations and clinical presentations. This raises the question of whether HCM and LVNC represent different phenotypes of the same disease or are, in fact, two distinct entities.

Key Words: Left ventricle hypertrabeculation; Hypertrophic cardiomyopathy; Left ventricle non-compaction; Left ventricle hypertrophy; Left ventricle obstruction

Core Tip: Hypertrophic cardiomyopathy (HCM) is a genetic myocardial disease marked by increased left ventricle (LV) wall thickness in the absence of abnormal loading conditions. Differentiating HCM from other conditions with LV hypertrophy, such as hypertension, aortic stenosis, or LV non-compaction (LVNC), can be challenging. LVNC is characterized by excessive LV trabeculation and deep recesses, affecting up to 5% of the general population. While typically benign, LVNC can occasionally lead to systolic dysfunction and arterial embolism. The overlap between HCM and LVNC, including genetic mutations and clinical features, raises the question of whether they are distinct diseases or variations of the same condition.

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INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is the most common form of primary, genetically determined cardiomyopathy (CMP)[1]. Caused by mutations in genes encoding sarcomere proteins, it results in structural changes in myocardial tissue, manifesting as pathological left ventricular hypertrophy (LVH) in terms of either an increase in left ventricular (LV) mass, LV wall thickness, or both (Figure 1A). The HCM phenotype can be diagnosed using transthoracic echocardiography (TTE) or cardiac magnetic resonance imaging (CMR) [2]. The diagnostic criterion for HCM is the presence of LVH in any myocardial segment measuring at least 15 mm, not attributable to excessive LV load[1]. Although HCM is estimated to have a prevalence of 0.2%, most patients remain undiagnosed due to the oligo-symptomatic nature of the disease[1]. However, in instances of more pronounced LVH, alongside diastolic dysfunction, arrythmias, or even systolic dysfunction, patients may present with shortness of breath, chest pain, palpitations, and signs indicative of heart failure [3,4].

HCM is primarily associated with LV muscle thickening; however, morphological changes also commonly involve the entire mitral valve apparatus. These changes can include hypertrophy and/or an increased number of papillary muscle heads, atypical papillary location, and/or elongation of the mitral leaflets (Figure 1B)[5]. Additionally, LVH can present with varying degrees and morphological traits, even among members of the same family or individuals with the same mutation[6]. Typically, LVH is classified into concentric or eccentric forms, with further differentiation based on the location of LV thickening, such as asymmetric septal hypertrophy, symmetric hypertrophy, or apical hypertrophy (Figure 1C and D). The apical form of HCM, predominantly found in individuals of Asian descent, occurs in approximately 2% of Caucasians[7]. Due to its frequently asymptomatic course, apical HCM is often detected incidentally during the evaluation of heart failure symptoms or through cascade screening of relatives.

PATHOLOGY OF LV NON-COMPACTION *VS* HYPERTRABECULATION

Approximately 15 days after fertilization, the heart tube is formed, primarily composed of cardiomyocytes. Around three weeks later, it develops into the four-chambered heart[8]. Initially, the heart muscle consists of trabeculae, which elongate and thicken during subsequent stages, ultimately merging into a uniform, compact muscle. Disruptions in gene expression due to a congenital or spontaneous mutation can impede this process, resulting in increased LV trabeculation or a 'spongy' myocardial structure. While numerous genes involved in LV development have been identified in mice, most have yet to be confirmed in humans[8].

LV non-compaction (LVNC) was first described in the 1990s, and its definition has evolved over the years[9]. Current definitions include specific and distinct diagnostic criteria for CMR imaging, with a noncompacted/compacted layer ratio ≥ 2.3, and for TTE, with a noncompacted/compacted layer ratio > 2[10,11]. The American Heart Association continues to classify LVNC as a distinct genetically determined CMP. However, the latest guidelines from the European Society of Cardiology (ESC) in 2023 introduced significant changes. The ESC replaced the term 'LVNC' with 'hypertrabeculation', while retaining the previous diagnostic criteria[1,12]. The authors of the ESC guidelines argue that the high prevalence of 'hypertrabeculation' (or 'LVNC') in the general population, including healthy individuals[13], supports the notion that 'hypertrabeculation' should be considered an anatomical variant of LV morphology rather than a structure which is pathological in nature.

There are also many other general medical conditions that can cause cardiac muscle hypertrophy, including arterial hypertension, aortic stenosis, myocarditis, or hyperthyroidism, complicating the diagnosis of primary or secondary hypertrophy. In all patients, the most common causes of secondary hypertrophy should be ruled out first. Additionally, the closest family should be included in the extended cardiological diagnostics. Genetic tests also appear to be helpful, but they are not widely available[14,15].

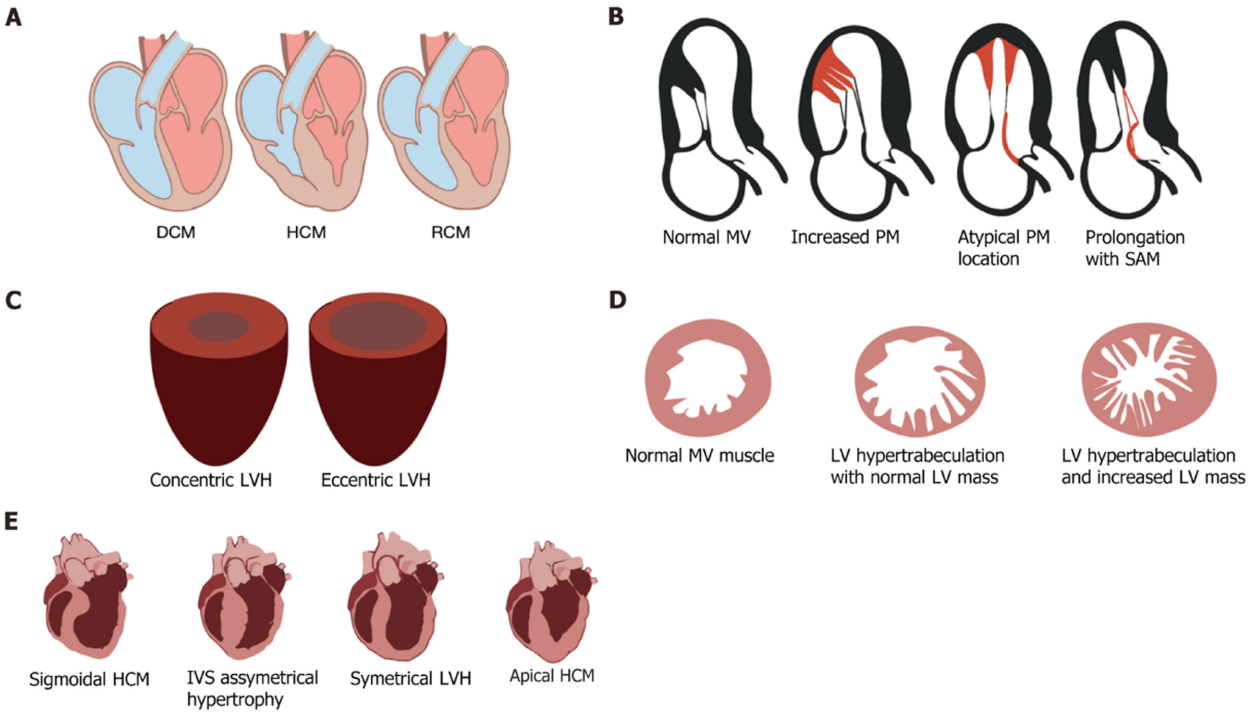

Figure 1 Different morphological changes in hypertrophic cardiomyopathy. A: Different cardiomyopathy (CMP) phenotypes. Dilated cardiomyopathy: Dilated CMP with left ventricle (LV) enlargement and hypokinesia. Hypertrophic cardiomyopathy (HCM): With increased LV mass or thickness. Restrictive cardiomyopathy: Restrictive CMP with enlarged atria; B: Morphological changes in mitral apparatus in HCM. Increased number and mass of papillary muscle heads, their atypical location, and elongation of the mitral leaflets; C and E: Different types and localization of LV hypertrophy; D: Phenotypic continuum between normal LV mass and structure, LV hypertrabeculation with normal LV mass [fulfilled diagnostic criteria for LV non-compaction (LVNC)], and hypertrabeculation with increased LV mass (fulfilled diagnostic criteria for LVNC and HCM). DCM: Dilated cardiomyopathy; HCM: Hypertrophic cardiomyopathy; IVS: Intraventricular septum; LV: Left ventricle; LVH: Left ventricular hypertrophy; MV: Mitral valve; PM: Papillary muscle; RCM: Restrictive cardiomyopathy; SAM: Systolic anterior motion.

LV HYPERTROPHY AND HYPERTRABECULATION

Despite the clear definition of HCM, complexities arise in patients exhibiting an atypical "spongy" myocardial structure. Over the years, discussions have centered on the continuum of 'hypertrabeculation' observed in healthy individuals with normal LV mass, or in those with coexisting increased LV mass in HCM, despite the presence of normal levels of thickness in the compacted LV muscle (Figure 1E). According to the definition, patients with increased LV mass, regardless of the LV muscle structure (compacted *vs* non-compacted), can also meet the criteria for an HCM diagnosis. Therefore, it is crucial to distinctly evaluate both the compacted and non-compacted LV layers, including the mass of trabeculae and papillary muscles, when assessing LV muscle thickness or LV mass in CMR and TTE (Figure 1F). Experts have long debated whether 'hypertrabeculation' in HCM (or other CMP phenotypes, including dilated CMP-DCM) significantly affects disease progression. A limited number of reports suggest that hypertrabeculation is associated with an increased arrhythmia frequency, LV systolic dysfunction, or an increased risk of thromboembolic complications[16].

Here, we report the case of a 58-year-old woman admitted to a tertiary cardiology clinic due to exercise-induced dyspnea over the preceding couple of months. Her previous medical history included arterial hypertension and smoking, and, at admission, she was on ramipril 5 mg and amlodipine 5 mg. TTE revealed HCM with intraventricular obstruction, a maximal wall thickness of 20 mm, LV mass of 86 mg/cm^2, and a resting LV intracavitary gradient of 76 mmHg (Figure 2A and D). CMR showed increased LV trabeculation, meeting LVNC criteria (C/NC layer ratio 6 mm:13 mm) (Figure 2C and D), and identified a thrombus in the LV apex extending to the papillary muscles (dimensions 32 mm × 12 mm × 16 mm) (Figure 2E), causing LV cavity obliteration.

Echocardiographic assessment of normal LV muscle thickness in these patients can be challenging. The 'spongy' myocardial structure can hamper accurate thickness measurement, raising the question of whether to measure the thickness of both the compacted and non-compacted layers, or merely the former, in the HCM diagnosis. In this case, the intracavity thrombus, mimicking the LV muscle, may also lead to an overestimation of LVH (Figure 2B). The thrombus formation was likely promoted by the spongy structure of the LV muscle and intracavitary narrowing, causing 'blood stasis' in the apex (Figures 2D and E)[17,18]. Due to the increased risk of systemic embolism with thrombus detection, oral anticoagulant therapy (warfarin or direct oral anticoagulants) should be considered for 3-6 months, guided by repeat imaging examinations[19]. In light of this, the patient was prescribed dabigatran 150 mg b.i.d.

In HCM, regardless of the presence of thrombus or LV hypertrabeculation, recommended therapy for intraventricular or LV outflow tract obstruction typically includes beta-blockers or non-dihydropyridine calcium channel blockers. If these are ineffective, novel therapy with mavacamten (if available) should be considered[1]. In this case, 5 mg of bisoprolol was initiated. Follow-up TTE showed a decrease in intraventricular obstruction at the apex with a resting gradient of 24 mmHg and 32 mmHg after the Valsalva maneuver and a reduction in LV thrombus. Consequently, the

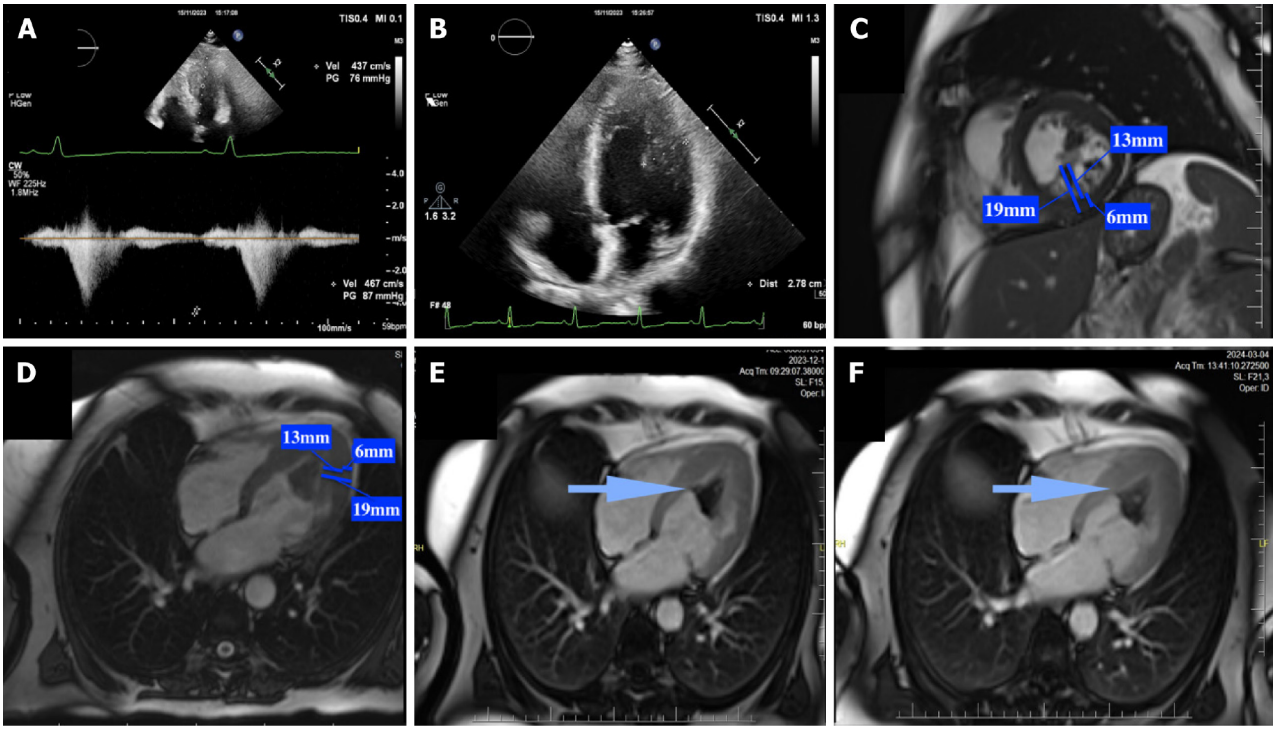

Figure 2 Images from transthoracic echocardiography and cardiac magnetic resonance imaging of a 58-year-old woman with coexistence of left ventricle hypertrophy and hypertrabeculation leading to intracavitary obstruction with heart failure symptoms, and left ventricle thrombus formation with subsequent transient ischemic attack. A: Pulse-wave Doppler from apical 4-chamber transthoracic echocardiography (TTE) view showing intracavitary obstruction at the level of the papillary muscles (maximal resting gradient of 76 mmHg); B: Left ventricle (LV) apical hypertrophy in apical 4-chamber view in TTE; C and D: LV hypertrabeculation in cardiac magnetic resonance short axis view and 4-chamber view (ratio of thickness of non-compacted to compacted layers 2.1: 13 mm and 6 mm); E and F: LV thrombus entering between the LV trabeculae and recesses in short axis view and 4-chamber view (blue arrow).

gradient reduction improved LV blood flow, including outflow from the apex, which likely decreased the risk of thrombus formation and propagation, while thrombus dissolution led to reduced LV obstruction.

CONCLUSION

Historically, LVNC was identified as a distinct form of CMP with a somewhat unpredictable course. However, advancements in imaging quality and the increased availability of CMR have revealed that 'hypertrabeculation' is prevalent even among healthy individuals, occurring independently of symptomatic cardiomyopathies. Emerging data highlight genetic mutations associated with 'LVNC', particularly in patients with coexisting CMP, such as HCM or DCM. In these patients, 'hypertrabeculation' can significantly worsen disease progression, increasing the risk of LV thrombus formation, LV systolic and diastolic dysfunction, and exacerbating mid-cavitary obstruction. Moreover, hypertrabeculation significantly complicates the accurate assessment of actual LV muscle mass and volume, thereby challenging the establishment of a reliable diagnosis of HCM and DCM.

FOOTNOTES

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