

Left ventricular hypertrophy on echocardiogram: When it is more than just physiological

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Introduction

Left ventricular hypertrophy (LVH) is a frequent echocardiographic finding and can represent either an adaptive physiological response or an indicator of underlying disease. Differentiating between these forms is crucial for identifying cardiomyopathies that require further evaluation.

Echocardiography remains the first-line imaging tool for assessing LVH, offering key insights that can guide the need for advanced imaging.

Take Home Messages

- Left ventricular hypertrophy (LVH) exists on a spectrum from physiological adaptation to pathological remodeling.
- LVH in cardiomyopathies can overlap with other causes. Accurate identification is therefore crucial for appropriate management, especially with the emergence of new treatment options.
- Key echocardiographic features, clinical history, and ECG findings are crucial in distinguishing pathological LVH from physiological variants, guiding further evaluation.
- A comprehensive, multimodality approach including the use of advanced imaging, is useful in identifying underlying cardiomyopathies.

LVH: Demographic and Physiological Influences

LVH may result from physiological adaptation or pathological changes associated with increased cardiovascular risk. According to the British Society of Echocardiography (BSE), LVH is defined as a left ventricular mass exceeding 99 g/m² in females and 110 g/m² in males, with wall thickness in diastole above 12 mm¹. Its prevalence varies based on demographic and physiological factors, such as race, obesity, hypertension, and athletic training². The Dallas Heart Study³ found that LVH is two to three times more prevalent in Black individuals compared to White individuals in the general population, and similarly in athletes and hypertensive individuals^{4,5}.



LVH in Hypertension and aortic stenosis

In hypertension, LVH is associated with increased cardiovascular morbidity and mortality^{6,7}.

Notably, regression of LVH has been associated with marked reduction in risk for cardiovascular events⁸.

In aortic stenosis, LVH initially represents an adaptive response but later correlates with myocardial damage and increased mortality^{9,10}. In a multicentre prospective registry of 674 patients with severe aortic stenosis undergoing transcatheter aortic valve replacement (TAVR), elevations in circulating cardiac troponin T (cTnT) and N-terminal pro-B-type natriuretic peptide (NT-proBNP) were more commonly observed in patients with more pronounced left ventricular hypertrophy (LVH), compared to those with no or minimal LVH. These biomarker elevations were associated with increased mortality⁹. Similarly, data from the PARTNER 1, 2, and S3 trials and registries indicate that severe baseline LVH is associated with higher rates of death and rehospitalization at 5 years following TAVR¹⁰. Conversely, regression of LVH following aortic valve replacement has been linked to improved outcomes^{11,12}.

LVH in Athletes

Cardiac remodelling in athletes varies depending on the type and intensity of exercise and ethnicity^{4,5,13}. While physiological LVH is common, the presence of marked hypertrophy may extend beyond normal adaptation. A review of sudden cardiac deaths in competitive athletes (1980–2006, USA) found that hypertrophic cardiomyopathy (HCM) was the most common cause (36%), followed by congenital coronary anomalies (17%)¹⁴.

LVH in cardiomyopathies

The 2014 ESC guidelines define HCM¹⁵ as increased LV wall thickness not solely explained by loading conditions. Primary LVH may result from genetic mutations, metabolic disorders, neuromuscular diseases, amyloidosis, or drug-induced hypertrophy. Genetic mutations in sarcomere proteins (a feature of HCM) account for 40–60% of cases, idiopathic in 25–30% and other genetic and non-genetic causes constitute 5–10%. The 2023 ESC consensus highlights the

role of multimodality imaging in diagnosing LVH-related conditions, including HCM, amyloidosis, and Anderson-Fabry disease¹⁶. Infiltrative diseases such as amyloidosis and Anderson–Fabry disease often show a correlation between the severity of LVH and adverse outcomes [^{17,18,19}].

Clinical and ECG clues in LVH for underlying cardiomyopathy

A comprehensive clinical assessment is vital. Age onset is an important clue for diagnosis in cardiomyopathies. A history of unexplained syncope, exertional dyspnoea, chest pain or a family history of sudden cardiac death or cardiomyopathy -particularly in first-degree relatives- should prompt further evaluation. Equally, non-cardiac history is important especially when there is the possibility of a syndromic or metabolic cause of cardiomyopathy. Moreover, physical examination findings like carpal tunnel syndrome in amyloidosis or neuropathic pain in Anderson-Fabry disease can aid in diagnosis.

ECG abnormalities are frequently seen in cardiomyopathies and may precede echocardiographic changes¹⁸. Although not specific, short PR interval, deep T-wave inversions, Q waves, pseudo-infarction patterns, or progressive conduction delays indicate potential underlying pathology rather than a mere adaptive response, as commonly seen in athletes.

Echocardiographic Clues to Pathological LVH

Several echocardiographic features warrant closer scrutiny, when LVH is detected. Unusual hypertrophy patterns—such as asymmetric septal hypertrophy, apical hypertrophy, or mid-cavity obstruction—raise suspicion for HCM. Myocardial texture abnormalities or increased echogenicity may point toward infiltrative diseases like amyloidosis. Similarly, pattern of LVH, regression of LVH after blood pressure control, presence of right ventricular hypertrophy, doppler and speckle-tracking parameters, including a restrictive filling pattern or impaired longitudinal strain, can provide further insights.

Table (1) Echocardiographic features to aid the diagnosis of LV hypertrophy

Aetiology	Hypertrophy pattern	Morphology and valve function	LVEF	GLS	Diastolic function
Athlete's heart	Eccentric LVH with MWT <14 mm	Balanced LV and RV dilatation	Normal	Preserved	Normal or supernormal
Sarcomeric HCM	Asymmetrical hypertrophy with MWT often >15 mm	Possible LVOTO and/or SAM with secondary MR	Normal/supernormal or reduced	Preserved or abnormal	Abnormal
Hypertensive heart disease	Asymmetrical hypertrophy (>>basal IVS)		Normal or reduced	Preserved or abnormal	Abnormal
Aortic stenosis	Concentric LVH	Valvular cusp calcification with reduced opening, increased valve pressure gradient	Normal or reduced	Preserved or abnormal	Abnormal
Cardiac amyloidosis	Concentric LVH often RV hypertrophy	Progressive reduction in LV volumes; biventricular involvement; bi-atrial enlargement; pericardial effusion	Normal or reduced	Preserved or abnormal, apical sparing	Abnormal
Anderson–Fabry disease	Severe concentric LVH	Progressive increase in LV volume	Normal or reduced	Altered basal LS	Abnormal

* Adapted from scientific statement of the Heart Failure Association of the European Society of Cardiology on Role of multimodality imaging in diagnosis and management of left ventricular hypertrophy

When to Consider Advanced Imaging

The European Society of Cardiology (ESC) consensus²⁰ appraises the critical role of multimodality imaging in LVH evaluation. While specifics are beyond the scope of this

discussion, its guidance emphasizes the need for advanced imaging like cardiovascular magnetic resonance (CMR) or nuclear imaging when echocardiographic and clinical findings indicate potential cardiomyopathy.

CMR, in particular, is invaluable by providing tissue characterization, mass quantification, and early disease detection, helping identify unusual LVH patterns and subtle myocardial abnormalities in HCM gene carriers. It also differentiates hypertrophic cardiomyopathy from other LVH causes, such as amyloidosis or Fabry disease, using late gadolinium enhancement and mapping techniques. Likewise, bone tracer scintigraphy is vital in diagnosing cardiac amyloidosis.

Conclusion

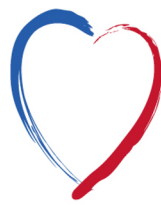
Echocardiography remains the cornerstone for LVH detection, but distinguishing between physiological and pathological forms requires a systematic approach. Recognizing key echocardiographic features, integrating clinical history and ECG findings, and identifying patients who warrant further multimodality imaging are critical steps in diagnosis and optimizing the patient care.

Disclosures

The authors declare no conflicts of interest related to this work.

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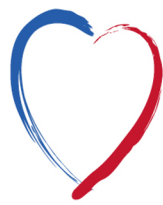


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Abbreviations

BSE	British Society of Echocardiogram
CMR	Cardiac Magnetic Resonance
cTnT	Circulating Troponin T



British Cardiovascular Society

'Promoting excellence in cardiovascular care'

ESC	European Society of Cardiology
HCM	Hypertrophic Cardiomyopathy
LV	Left ventricle
LVH	Left ventricular hypertrophy
LVEF	Left ventricular ejection fraction
LVOTO	Left ventricular outflow tract obstruction
NT-proBNP	N-terminal pro B-type natriuretic peptide
RV	Right ventricle
SAM	Systolic anterior motion
TAVR	Transcatheter Aortic Valve Replacement