British Cardiovascular Society
'Promoting excellence in cardiovascular care'

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

Banya Aung Myint M.B., B.S, MRCP(UK)

Specialist registrar in Cardiology

Queen Elizabeth Hospital Birmingham

Introduction

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.

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.

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 mm[1]. Its prevalence varies based on demographic and physiological factors, such as race, obesity, hypertension, and athletic training[2]. The Dallas Heart Study[3] 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 events[8].

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 mortality[9]. 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 TAVR[10]. 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 HCM[15] 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 disease[16]. 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 changes[18]. 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) consensus[20] 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.

References

(1) Robinson S, Rana B, Oxborough D, Steeds R, Monaghan M, Stout M, Pearce K, Harkness A, Ring L, Paton M, Akhtar W, Bedair R, Bhattacharyya S, Collins K, Oxley C, Sandoval J, Schofield MBChB R, Siva A, Parker K, Willis J, Augustine DX. A practical guideline for performing a comprehensive transthoracic echocardiogram in adults: the British Society of Echocardiography minimum dataset. Echo Res Pract. 2020 Dec;7(4):G59-G93. doi: 10.1530/ERP-20-0026. PMID: 33112828; PMCID: PMC7923056.

(2) Avelar E, Cloward TV, Walker JM, Farney RJ, Strong M, Pendleton RC, Segerson N, Adams TD, Gress RE, Hunt SC, Litwin SE. Left ventricular hypertrophy in severe obesity: interactions among blood pressure, nocturnal hypoxemia, and body mass. Hypertension. 2007 Jan;49(1):34-9. doi: 10.1161/01.HYP.0000251711.92482.14. Epub 2006 Nov 27. PMID: 17130310.

(3) Drazner MH, Dries DL, Peshock RM, Cooper RS, Klassen C, Kazi F, Willett D, Victor RG. Left ventricular hypertrophy is more prevalent in blacks than whites in the general population: the Dallas Heart Study. Hypertension. 2005 Jul;46(1):124-9. doi: 10.1161/01.HYP.0000169972.96201.8e. Epub 2005 Jun 6. PMID: 15939807

(4) Basavarajaiah S, Boraita A, Whyte G, Wilson M, Carby L, Shah A, Sharma S. Ethnic differences in left ventricular remodelling in highly-trained athletes' relevance to differentiating physiologic left ventricular hypertrophy from hypertrophic cardiomyopathy. J Am Coll Cardiol. 2008 Jun 10;51(23):2256-62. doi: 10.1016/j.jacc.2007.12.061. PMID: 18534273.

(5) J. Neil Chapman, Jamil Mayet, C. Lan Chang, Rodney A. Foale, Simon A.McG Thom, Neil R. Poulter, Ethnic Differences in the identification of left ventricular hypertrophy in the hypertensive patient, American Journal of Hypertension, Volume 12, Issue 5, May 1999, Pages 437–442, https://doi.org/10.1016/S0895-7061(99)00027-8

(6) Verdecchia P, Schillaci G, Borgioni C, et al. Prognostic significance of serial changes in left ventricular mass in essential hypertension. Circulation 1998; 97:48-54.

(7) Aronow WS. Hypertension and left ventricular hypertrophy. Ann Transl Med 2017;5(15):310. doi: 10.21037/atm.2017.06.14

(8) Verdecchia P, Angeli F, Borgioni C, Gattobigio R, de Simone G, Devereux RB, Porcellati C. Changes in cardiovascular risk by reduction of left ventricular mass in hypertension: a meta-analysis. Am J Hypertens. 2003 Nov;16(11 Pt 1):895-9. doi: 10.1016/s0895-7061(03)01018-5. PMID: 14573325.

(9) Stein EJ, Fearon WF, Elmariah S, Kim JB, Kapadia S, Kumbhani DJ, Gillam L, Whisenant B, Quader N, Zajarias A, Welt FG, Bavry AA, Coylewright M, Piana RN, Mallugari RR, Clark DE, Patel JN, Gonzales H, Gupta DK, Vatterott A, Jackson N, Huang S, Lindman BR. Left Ventricular Hypertrophy and Biomarkers of Cardiac Damage and Stress in Aortic Stenosis. J Am Heart Assoc. 2022 Apr 5;11(7):e023466. doi: 10.1161/JAHA.121.023466. Epub 2022 Mar 18. PMID: 35301869; PMCID: PMC9075421.

(10) Gonzales H, Douglas PS, Pibarot P, Hahn RT, Khalique OK, Jaber WA, Cremer P, Weissman NJ, Asch FM, Zhang Y, Gertz ZM, Elmariah S, Clavel MA, Thourani VH, Daubert M, Alu MC, Leon MB, Lindman BR. Left Ventricular Hypertrophy and Clinical Outcomes Over 5 Years After TAVR: An Analysis of the PARTNER Trials and Registries. JACC Cardiovasc Interv. 2020 Jun 8;13(11):1329-1339. doi: 10.1016/j.jcin.2020.03.011. PMID: 32499024.

(11) Hatani T, Kitai T, Murai R, et al. Associations of residual left ventricular and left atrial remodelling with clinical outcomes in patients after aortic valve replacement for severe aortic stenosis. J Cardiol 2015 Oct 30.

(12) 7. Chau KH, Douglas PS, Pibarot P, Hahn RT, Khalique OK, Jaber WA, Cremer P, Weissman NJ, Asch FM, Zhang Y, Gertz ZM, Elmariah S, Clavel MA, Thourani VH, Daubert M, Alu MC, Leon MB, Lindman BR. Regression of Left Ventricular Mass After Transcatheter Aortic Valve Replacement: The PARTNER Trials and Registries. J Am Coll Cardiol. 2020 May 19;75(19):2446-2458. doi: 10.1016/j.jacc.2020.03.042. PMID: 32408979.

(13) Rawlins J, Bhan A, Sharma S. Left ventricular hypertrophy in athletes. Eur J Echocardiogr. 2009 May;10(3):350-6. doi: 10.1093/ejechocard/jep017. Epub 2009 Feb 26. PMID: 19246500.

(14) Maron, Barry & Doerer, Joseph & Haas, Tammy & Tierney, David & Mueller, Frederick. (2009). Sudden Deaths in Young Competitive Athletes Analysis of 1866 Deaths in the United States, 1980-2006. Circulation. 119. 1085-92. 10.1161/CIRCULATIONAHA.108.804617.

(15) Authors/Task Force members; Elliott PM, Anastasakis A, Borger MA, Borggrefe M, Cecchi F, Charron P, Hagege AA, Lafont A, Limongelli G, Mahrholdt H, McKenna WJ, Mogensen J, Nihoyannopoulos P, Nistri

S, Pieper PG, Pieske B, Rapezzi C, Rutten FH, Tillmanns C, Watkins H. 2014 ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy: the Task Force for the Diagnosis and Management of Hypertrophic Cardiomyopathy of the European Society of Cardiology (ESC). Eur Heart J. 2014 Oct 14;35(39):2733-79. doi: 10.1093/eurheartj/ehu284. Epub 2014 Aug 29. PMID: 25173338.

(16) Moura B, Aimo A, Al-Mohammad A, Keramida K, Ben Gal T, Dorbala S, Todiere G, Cameli M, Barison A, Bayes-Genis A, von Bardeleben RS, Bucciarelli-Ducci C, Delgado V, Mordi IR, Seferovic P, Savarese G, Čelutkienė J, Rapezzi C, Emdin M, Coats A, Metra M, Rosano G. Diagnosis and management of patients with left ventricular hypertrophy: Role of multimodality cardiac imaging. A scientific statement of the Heart Failure Association of the European Society of Cardiology. Eur J Heart Fail. 2023 Sep;25(9):1493-1506. doi: 10.1002/ejhf.2997. Epub 2023 Sep 4. PMID: 37581253.

(17) Aimo A, Tomasoni D, Porcari A, Vergaro G, Castiglione V, Passino C, Adamo M, Bellicini MG, Lombardi CM, Nardi M, Palamara G, Varrà GG, Saro R, Allegro V, Merlo M, Sinagra G, Metra M, Emdin M, Rapezzi C. Left ventricular wall thickness and severity of cardiac disease in women and men with transthyretin amyloidosis. Eur J Heart Fail. 2023 Apr;25(4):510-514. doi: 10.1002/ejhf.2824. Epub 2023 Mar 15. PMID: 36919654.

(18) Ota S, Izumiya Y, Kitada R, Nishi T, Taruya A, Wada T, Takahata M, Ozaki Y, Kashiwagi M, Shiono Y, Kuroi A, Takemoto K, Tanimoto T, Kitabata H, Fukuda D, Tanaka A. Diagnostic significance of paradoxical left ventricular hypertrophy in detecting cardiac amyloidosis. Int J Cardiol Heart Vasc. 2023 Oct 13;49:101279. doi: 10.1016/j.ijcha.2023.101279. PMID: 37859641; PMCID: PMC10582063.

(19) Hao-Chih Chang, Ling Kuo, Shih-Hsien Sung, Dau-Ming Niu, Wen-Chung Yu, Prognostic Implications of Left Ventricular Hypertrophy and Mechanical Function in Fabry Disease: A longitudinal Cohort Study, Journal of the American Society of Echocardiography, Volume 37, Issue 8, 2024, Pages 787-796, ISSN 0894-7317, https://doi.org/10.1016/j.echo.2024.04.010.

(20) Elena Arbelo, Alexandros Protonotarios, Juan R Gimeno, Eloisa Arbustini, Roberto Barriales-Villa, Cristina Basso, Connie R Bezzina, Elena Biagini, Nico A Blom, Rudolf A de Boer, Tim De Winter, Perry M Elliott, Marcus Flather, Pablo Garcia-Pavia, Kristina H Haugaa, Jodie Ingles, Ruxandra Oana Jurcut, Sabine Klaassen, Giuseppe Limongelli, Bart Loeys, Jens Mogensen, Iacopo Olivotto, Antonis Pantazis, Sanjay Sharma, J Peter Van Tintelen, James S Ware, Juan Pablo Kaski, ESC Scientific Document Group , 2023 ESC Guidelines for the management of cardiomyopathies: Developed by the task force on the management of cardiomyopathies of the European Society of Cardiology (ESC), European Heart Journal, Volume 44, Issue 37, 1 October 2023, Pages 3503–3626, https://doi.org/10.1093/eurheartj/ehad194

Abbreviations

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

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