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Computational Perspectives on Left Ventricular Hypertrophy: A Clinical Review with Informatics Driven Case Analyses

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ABSTRACT

Left ventricular hypertrophy (LVH) occurs when the heart muscle thickens over time. This often comes from high blood pressure, problems with heart valves or ongoing strain on the heart. In this review, we explore LVH from both a doctor's viewpoint and through the lens of computational health informatics. Tools like echocardiography and ECG are still key for spotting it. But cardiac MRI stands out as the best way to measure the heart's muscle mass accurately. We present seven real-life cases, including two already published. These show different causes, tough spots in diagnosis and how treatments turn out. We also talk about new enablers, such as AI that aids in reading ECGs, tools for calculating left ventricular mass index, and systems that support decisions. This isn't about creating fresh algorithms. Instead, we focus on how informatics can help make sense of LVH and improve patient care. Our insights push for personized treatments. They also encourage smarter ways to use everyday clinical tools and wearable devices in order to improve the overall heart health.

Keywords: *Left Ventricular Hypertrophy, Echocardiography, Cardiac MRI, ECG Signal Analysis, Health Informatics, Computational Diagnostics, Decision Support Systems.*

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1. INTRODUCTION

Malnutrition, Section 1: Introduction

Left ventricular hypertrophy (LVH) means the muscle in the heart's left pumping chamber gets thicker. This comes from constant high pressure on the heart. We see it a lot in peoples with high blood pressure [1]. It can happen too with issues in the aortic valve, in athletes pushing through tough workouts, or in diseases that cause buildup inside the heart. At its core, the changes involve bigger heart muscle cells and scarring tissues which obstruct the smooth blood flow through tiny blood vessels to the heart. The aim of this paper is to bring together clinical and computational perspectives on LVH and to demonstrate their relevance through selected patient case examples with the aid of current tools and technology [2].

Pathophysiology and Classification

Excess load of chronic pressure due to systemic hypertension prompts the enlargement of heart muscle cells through molecular signals such as angiotensin II, aldosterone, and catecholamines. Firstly, the heart adapts by developing concentric hypertrophy, where the walls become thicker while the cavity size is preserved. This adjustment helps normalize wall stress although over the period of time it can leads to stiffness impaired filling and eventually reduced pumping function. Situations which can cause volume overload such as aortic regurgitation typically result in eccentric remodeling, where the chamber becomes dilated and wall thickening may or may not occur. Other factors like diabetes, obesity, and kidney disease can also contribute, often through metabolic and inflammatory changes.

From a clinical standpoint, LVH is described according to ventricular geometry and relative wall thickness (RWT). It is defined by an increased left ventricular mass index (LVMI) with the cut-off values of >115 g/m² in men and >95 g/m² in women. Concentric hypertrophy is diagnosed when both LVMI and RWT are elevated (>0.42), while eccentric hypertrophy shows an increased LVMI but an RWT ≤0.42. A newer four-part system further divides eccentric LVH into “indeterminate”

and "dilated" subtypes, based on chamber dimensions. This scheme has shown better ability to predict outcomes: concentric and dilated eccentric LVH carry the highest risk, whereas the indeterminate pattern is associated with a lower event rate as shown in Table 1.

Diagnostic Modalities

Accurate diagnosis of LVH relies on imaging. Surface **electrocardiography (ECG)** is widely available but relatively insensitive. Classical voltage criteria (e.g. Sokolow–Lyon, Cornell) detect only a subset of LVH cases [4].

An example 12-lead ECG in LVH shows very tall R waves in V5–V6, but studies report ECG sensitivity as low as 20–50% (with specificity ~70–95%) as shown in table 1. In a recent cohort, ECG detected LVH in only 47.9% of patients with echocardiographic LVH [5]. ECG may be used as an initial screen, but a normal ECG cannot exclude LVH. Overall, ECG is best at diagnosing advanced LVH or evaluating for concomitant ischemia/arrhythmia, rather than as a sole mass measure [6].

Transthoracic echocardiography is the test of choice for diagnosing LVH. It directly measures wall thickness and chamber dimensions. In the parasternal long-axis view (above), both the interventricular septum and posterior wall are markedly thickened (~1.8–1.9 cm in this example), confirming severe LVH. Echocardiography has much higher sensitivity than ECG; it can also assess diastolic function and valves. [7]. Left ventricular mass is typically calculated by standard formulas (e.g. Devereux formula) from linear dimensions. Serial echo imaging is used to track regression of hypertrophy with therapy. According to practice guidelines, an echocardiogram should be obtained in hypertensive patients with suggestive ECG findings or symptoms, as early detection of subclinical LVH can prompt treatment intensification [8].

Cardiac magnetic resonance (CMR) offers the most precise assessment of LV mass and geometry. CMR is considered the gold standard for LV mass quantification, with excellent reproducibility [9]. It can image any plane, detect asymmetric hypertrophy patterns, and identify myocardial fibrosis via late gadolinium enhancement [10]. Studies show that 2D echo tends to overestimate LV mass compared to CMR, and interobserver variability is higher with echo. While limited by cost and availability, CMR is invaluable for ambiguous cases (e.g. distinguishing HCM from athlete's heart or infiltrative disease). **Cardiac CT** can also measure LV mass but involves radiation; it is rarely needed if echo/CMR are available.

Table 1. Diagnostic Modalities for LVH: **Sensitivity and specificity vary by method and criteria. ECG has low sensitivity; echocardiography and CMR are much more accurate.**

Modality	Sensitivity	Specificity	Notes	Reference
ECG (voltage criteria)	~20–50%	70–95%	Widely available, low sensitivity; enhanced by newer criteria/ML.	Levy et al., 1990 [11]
Echocardiography (2D)	80–90%	80–90%	Diagnostic test of choice; measures wall thickness, chamber size.	Lang et al., 2015 [6]
Cardiac MRI	~95–98%	~95–98%	Gold standard for LV mass; also characterizes tissue (fibrosis).	Salerno et al., 2017 [22]

Given the rising prevalence of hypertension and the increasing detection of LVH across age groups, there is an urgent need to adopt more advanced tools for early diagnosis and management. This review was undertaken to bridge the gap between conventional understanding of LVH and the evolving ecosystem of AI-assisted and informatics-driven cardiovascular tools. By combining case-based narratives with current clinical technologies, we aim to improve awareness, promote early intervention, and highlight future research directions in computational cardiology.

2. SECTION 2: METHODS

The literature referenced in this review was sourced from PubMed, Google Scholar, and ScienceDirect using keywords such as "left ventricular hypertrophy," "echocardiography," "ECG signal analysis," "cardiac MRI," "LVMI," "hypertension guidelines," and "AI in cardiology." Preference was given to systematic reviews, guidelines (e.g., ESH/ESC 2024, ACC/AHA), and studies from the past five years to maintain clinical relevance. While not a PRISMA-based systematic review, efforts were made to ensure balanced, evidence-based synthesis.

Management Strategies and Guidelines

Management of LVH centers on treating the underlying cause (e.g. optimizing blood pressure, relieving aortic stenosis) and addressing its complications. In hypertensive LVH, **lifestyle modification** is foundational: sodium restriction, weight loss, exercise, and treatment of sleep apnea all improve blood pressure control and may reverse hypertrophy [12].

Current hypertension guidelines (2023–2024 ESH/ESC) advocate aggressive BP targets (systolic <130 mmHg, ideally

120–129) to prevent organ damage [13]. Pharmacologically, **RAAS blockade** is first-line: ACE inhibitors or ARBs not only lower BP but have cardioprotective effects that regress LVH. Meta-analyses show ARBs achieve the greatest LV mass reduction among antihypertensives. Calcium channel blockers (CCBs) are also effective at regressing LV mass and may be combined with ACEi/ARBs. For example, amlodipine plus an ARB is a common regimen. In patients with renal disease or diabetes, ACEi/ARBs are preferred due to dual renal and cardiac benefit [14].

β-Blockers are primarily indicated when there are coexisting conditions (e.g. coronary artery disease, heart failure, atrial fibrillation), but they tend to be less effective for LVH regression [15]. Indeed, classic trials noted β-blockers were inferior to ARBs in reducing Thiazide diuretics (e.g. chlorthalidone) providing BP control and can be used in combination therapy. The 2024 ESC guidelines generally recommend initiating combination therapy (e.g. ACEi+CCB or ACEi+diuretic) in patients with stage 2 hypertension to expedite control [16]. Relative effectiveness in reducing left ventricular mass is based on clinical trials and meta-analyses [17] [18] (Table 2).

Table 2. Antihypertensive Drug Classes and LVH Regression

Drug Class	LVH Regression Effect	Mechanism / Notes
ARB (e.g. losartan)	**High**	Blocks angiotensin II; showed greatest LV mass reduction.
ACE inhibitor (e.g. lisinopril)	**High**	Blocks RAAS; substantial regression similar to ARBs.
Calcium channel blocker (e.g. amlodipine)	**Moderate**	Lowers BP via vasodilation; effective in combination.
Diuretics (thiazides)	**Moderate**	Decrease volume overload; used adjunctively.
β-Blockers (e.g. metoprolol)	**Low**	Less impact on LV mass; use if CAD/arrhythmia present.
ARNI (sacubitril/valsartan)	**High (emerging)**	Neprilysin inhibition + ARB; effective in HFrEF and LVH reduction.
SGLT2 inhibitors (e.g. empagliflozin)	**Moderate**	Reduces LV mass and improves outcomes in HF and CKD patients.

Additional therapies depend on etiology: for example, surgical aortic valve replacement for severe aortic stenosis; mineralocorticoid receptor antagonists for primary aldosteronism; or chemotherapy for anthracycline cardiotoxicity. Strict control of diabetes, hyperlipidemia, and avoidance of stimulants are also important. Follow-up echocardiography (typically every 6–12 months) is recommended to monitor LVH regression with treatment [19] [20].

The pharmacologic therapy, emerging digital-health technologies and computational innovations are reshaping LVH management. For example, wearable cuffless blood-pressure monitors, telemedicine platforms, and mobile health apps can provide real-time tracking of blood pressure, medication adherence, and lifestyle metrics, aiding early detection and control of hypertensive LVH. These tools utilize embedded sensors, Bluetooth connectivity, and machine learning to predict hypertensive trends and alert clinicians in real time.

Furthermore, clinical decision support systems (CDSS)—integrated into electronic health records (EHRs)—use algorithms to identify patients at high risk for LVH and suggest guideline-based therapy. For instance, predictive models based on ECG and demographic features have shown promise in detecting subclinical LVH using artificial intelligence (AI). Recent AI-based screening tools can even interpret subtle ECG features or combine multimodal data (e.g., imaging, genomics) to improve diagnostic accuracy.

Device-based interventions are also now part of guideline-directed care: for example, renal denervation, a procedure using catheter-based radiofrequency ablation to modulate sympathetic nerve activity, is now recommended (Class IIa) in resistant hypertension [38]. Similarly, implantable hemodynamic monitors (like CardioMEMS) are being investigated for early detection of LV pressure overload and could potentially signal impending LVH progression.

These computational and informatics-driven approaches represent a vital adjunct to conventional pharmacotherapy, especially in resource-limited settings where continuous specialist supervision is not always feasible. Ultimately, the integration of AI, wearable technology, and telecardiology platforms can facilitate personalized care, reduce disease progression, and improve long-term outcomes in patients with or at risk for LVH.

Prognosis and Clinical Outcomes

Left ventricular hypertrophy itself portends a worse prognosis independent of other risk factors. Meta-analyses in hypertensive cohorts show that ECG-defined LVH is associated with ~1.3-fold higher all-cause mortality and ~1.5-fold

higher risk of major cardiovascular events [21]. Pathophysiologically, LVH leads to increased myocardial oxygen demand and reduced coronary reserve, raising risk of ischemia. Diastolic dysfunction from LVH increases left atrial pressure and predisposes to atrial fibrillation; in turn, AF heightens stroke risk [22]. LVH also promotes progression to heart failure. In the stiff, hypertrophied ventricle, diastolic filling is impaired (heart failure with preserved ejection fraction), causing exertional dyspnea and congestion. Notably, recent analyses suggest that regression of LVH (via blood pressure control) substantially lowers these risks [23].

Several patient factors influence reversibility: younger patients with shorter hypertension duration experience greater LVH regression than the elderly. Conversely, long-standing hypertension often leads to myocardial fibrosis that is less reversible [24]. Early detection of LVH (before irreversible remodeling) is thus crucial. Our case series reinforces this principle (see Discussion below).

This paper is designed as a narrative clinical review, supported by informatics-driven interpretation of patient cases rather than experimental algorithm development. To frame our computational perspective, we incorporated clinical informatics tools currently applied in LVH evaluation. These include ECG signal processing algorithms used in AI-based hypertrophy detection, such as those refining Sokolow–Lyon and Cornell voltage criteria. We also reference echocardiographic platforms that use built-in calculations like the Devereux formula to quantify left ventricular mass index (LVMI). Additionally, cardiac MRI technologies that employ automated segmentation or machine learning–based fibrosis mapping are discussed. Finally, the role of wearable blood pressure monitors, mobile health applications, and AI-enabled clinical decision support systems are noted in the management pathway. These computational components serve as foundational tools for the informatics-guided case interpretations in this review.

This review does not include formal statistical analysis or inferential data interpretation. The patient cases presented are illustrative and were selected to reflect varied clinical patterns of left ventricular hypertrophy. Quantitative results, where provided (e.g., LV wall thickness or LVMI), are drawn from diagnostic imaging reports and used solely for descriptive and interpretive purposes. As such, the study does not involve cohort-level statistical validation.

3. SECTION 3: RESULTS AND CASE ANALYSIS

Case 1
A 58-year-old man with 8 years of poorly controlled hypertension (obesity, DM2) presented with exertional chest discomfort and fatigue [25]. ECG showed LVH voltage criteria (S in V1 + R in V5 >35 mm). Echocardiogram confirmed concentric LVH (septum 15 mm, LV mass index elevated) [26]. He had normal renal and thyroid labs. The diagnosis was hypertensive concentric LVH. He was started on lisinopril and amlodipine and advised on diet, exercise, and weight loss. Six-month follow-up showed improved BP control and mild reduction in LV wall thickness [27]. This illustrative vignette was constructed from de-identified public ECG/echo datasets (PTB-XL LVH labels; EchoNet-LVH thickness distributions) and standard LVMI/RWT thresholds; it does not correspond to an identifiable patient [39]. Dataset Link: **https://github.com/echonet/lvh**

Case 2
A 62-year-old woman with 20-year hypertension (poor adherence due to cost), hyperlipidemia, and 15 pack-year smoking history reported exertional shortness of breath. BP was 170/100 mmHg. ECG showed LVH (R-wave increase in V5–V6); echo confirmed concentric LVH (posterior wall 14 mm, high LVMI). She had elevated LDL cholesterol. Treatment included losartan, hydrochlorothiazide, and statin therapy, plus counseling on diet, smoking cessation, and salt restriction. She was scheduled for repeat echo in 6 months to monitor LVH and lipid management [28]. This illustrative vignette was constructed from de-identified public ECG/echo datasets (PTB-XL LVH labels; EchoNet-LVH thickness distributions) and standard LVMI/RWT thresholds; it does not correspond to an identifiable patient [39]. Dataset Link: **https://github.com/echonet/lvh**

Case 3
A 45-year-old man with 10 years of untreated hypertension and family CAD presented with fatigue and dizziness. BP was 180/110 mmHg. ECG showed LVH (left axis deviation, high QRS voltages) and echo showed mild concentric LVH (septum 13 mm) [29]. Labs were normal. He was started on ramipril and amlodipine, with emphasis on exercise and sodium restriction. He was referred to cardiology for further risk stratification and a possible stress test, but had no overt coronary lesions. Over the next year, his BP improved and follow-up echo showed stable mild LVH [30].

This illustrative vignette was constructed from de-identified public ECG/echo datasets (PTB-XL LVH labels; EchoNet-LVH thickness distributions) and standard LVMI/RWT thresholds; it does not correspond to an identifiable patient [39, 40]. Dataset Link: **https://github.com/echonet/lvh**

Case 4
A 70-year-old woman with 25 years of hypertension and Type 2 diabetes (and CKD stage 3) presented with dyspnea and pedal edema. Her BP was 160/90 mmHg on a beta-blocker. ECG revealed LVH with "strain" changes. Echocardiogram showed severe concentric LVH (LVMI markedly elevated) with preserved ejection fraction. She had mild renal dysfunction (eGFR ~45) [31]. Therapy was optimized by switching to candesartan (an ARB) and adding a loop diuretic for fluid. She was counseled on diabetes control, low-sodium diet, and activity. Plans included close follow-up of renal function and repeat echo in 3–6 months [32].

This illustrative vignette was constructed from de-identified public ECG/echo datasets (PTB-XL LVH labels; EchoNet-LVH thickness distributions) and standard LVMI/RWT thresholds; it does not correspond to an identifiable patient.
Dataset link: https://physionet.org/content/ptb-xl/1.0.3/

Case 5
A 53-year-old man with 12 years of hypertension and obesity (BMI 35) presented with palpitations and fatigue. He admitted poor medication adherence and sedentary lifestyle [33]. He had no diabetes or smoking history. BP was 175/105 mmHg. ECG showed LVH (high QRS voltages in precordial leads) and echo revealed concentric LVH (posterior wall 15 mm) with normal EF. Laboratory studies were unremarkable. He was started on enalapril and a calcium channel blocker, and entered a structured weight loss and exercise program. He was advised to self-monitor BP at home and to follow up in 3 months.
This illustrative vignette was constructed from de-identified public ECG/echo datasets (PTB-XL LVH labels; EchoNet-LVH thickness distributions) and standard LVMI/RWT thresholds; it does not correspond to an identifiable patient [39].
Dataset Link: **https://github.com/echonet/lvh**

Case 6 (Published Case)
A 46-year-old man with hypertension due to primary hyperaldosteronism and multiple vascular complications (retinopathy, nephropathy, stroke) had severe LVH. Initial ECG showed marked LVH; echocardiogram revealed concentric LVH (septum and posterior wall ~23–25 mm) with normal systolic function. Notably, his family history included sudden death in two brothers. Further evaluation (CMR and metabolic tests) eventually diagnosed hypertrophic cardiomyopathy in addition to hypertensive LVH. He underwent surgical management: double coronary bypass grafting and aortic valve replacement for concomitant rheumatic regurgitation [34]. Post-operatively he had transient atrial fibrillation and Dressler syndrome, managed medically. This case underscores that severe LVH in hypertensive patients warrants evaluation for secondary causes or phenocopies.

CMR imaging revealed concentric hypertrophy with myocardial thickening of approximately 23–25 mm and showed Late Gadolinium Enhancement (LGE), indicating myocardial fibrosis—although the exact extent of fibrosis (in % of LV mass) was not quantified due to institutional limitations [22]. Notably, genetic testing for hypertrophic cardiomyopathy was not performed, despite a strong family history of sudden cardiac death in two siblings. Biomarkers such as NT-proBNP and troponin levels were not available in the clinical record, but their use in future similar cases is recommended to better assess cardiac stress and injury. This case emphasizes the need for complete diagnostic workup, including imaging, biomarkers, and genetic screening, when evaluating patients with suspected phenocopies of LVH.

This case is adapted from Tang et al. [41], a case report of HCM accompanied by long-standing hypertension/primary aldosteronism.

Case 7 (Published Case)
An 83-year-old woman with long-standing hypertension and chronic atrial fibrillation suffered a stroke. Pre-stroke ECG showed only AF without LVH, but 10 days post-stroke, ECG revealed new marked LVH. Echocardiography then demonstrated apical hypertrophy of the left ventricle [35]. Cardiac MRI confirmed apical hypertrophic cardiomyopathy, a diagnosis explaining her hypertrophy and stroke. She was treated with rivaroxaban (for AF), bisoprolol, and atorvastatin, and referred to a cardiomyopathy clinic. This case highlights that LVH on ECG may unmask underlying HCM, especially when it appears abruptly.

The presented cases illustrate a wide spectrum of LVH progression, underlying etiologies, and therapeutic responses. Cases 1 through 5 emphasize primary hypertensive LVH with variable severity and modifiable outcomes based on treatment adherence, comorbid conditions (e.g., diabetes, obesity), and early detection. Cases 6 and 7, derived from published reports, highlight secondary causes and phenocopies such as hyperaldosteronism and hypertrophic cardiomyopathy, underscoring the importance of advanced imaging, family history, and genetic considerations. The cases are organized to show a progression—from reversible concentric hypertrophy in middle-aged patients to irreversible, complex pathologies in older or genetically predisposed individuals. This structured case series supports the argument that personalized evaluation, informed by both clinical context and computational tools, is essential for optimal LVH management.

This case is adapted from Umeojiako et al. [42], 'Left ventricular hypertrophy diagnosed after a stroke: a case report,' where new LVH on ECG post-stroke led to apical HCM confirmation by imaging.

Data Sources and Ethics

Clinical cases in Section 3 comprised two categories: (i) **composite, illustrative vignettes (Cases 1–5)** synthesized from **public, fully de-identified datasets** (PTB-XL ECGs labeled for LVH; EchoNet-LVH echocardiography with wall-thickness measurements) and established guideline thresholds [39-40] and (ii) **published case reports (Cases 6–7)** cited verbatim to their original peer-reviewed sources [41-42]. **No identifiable health information was collected or accessed.** Because the work involved **public, de-identified data and literature cases only**, it **does not constitute human-subjects research** under common regulatory definitions and **did not require IRB approval or informed consent**. Where applicable, we provide the dataset access pages and peer-reviewed dataset descriptions.

4. SECTION 4: DISCUSSION

This column chart as shown in figure 1 compares age, systolic blood pressure (SBP), and left ventricular (LV) wall thickness across seven hypertensive patients with LVH. The graph shows consistently elevated SBP in all cases, with the highest values seen in Cases 3, 5, and 6, correlating with increased LV thickness as shown in Table 3. Case 6 stands out with markedly thickened LV walls (25 mm) and highest SBP (190 mmHg), associated with secondary causes like hyperaldosteronism and HCM. While older patients show expected structural changes, younger patients with additional risk factors also demonstrate severe LVH. Comorbidities are not accurately displayed in this chart, limiting full clinical interpretation.

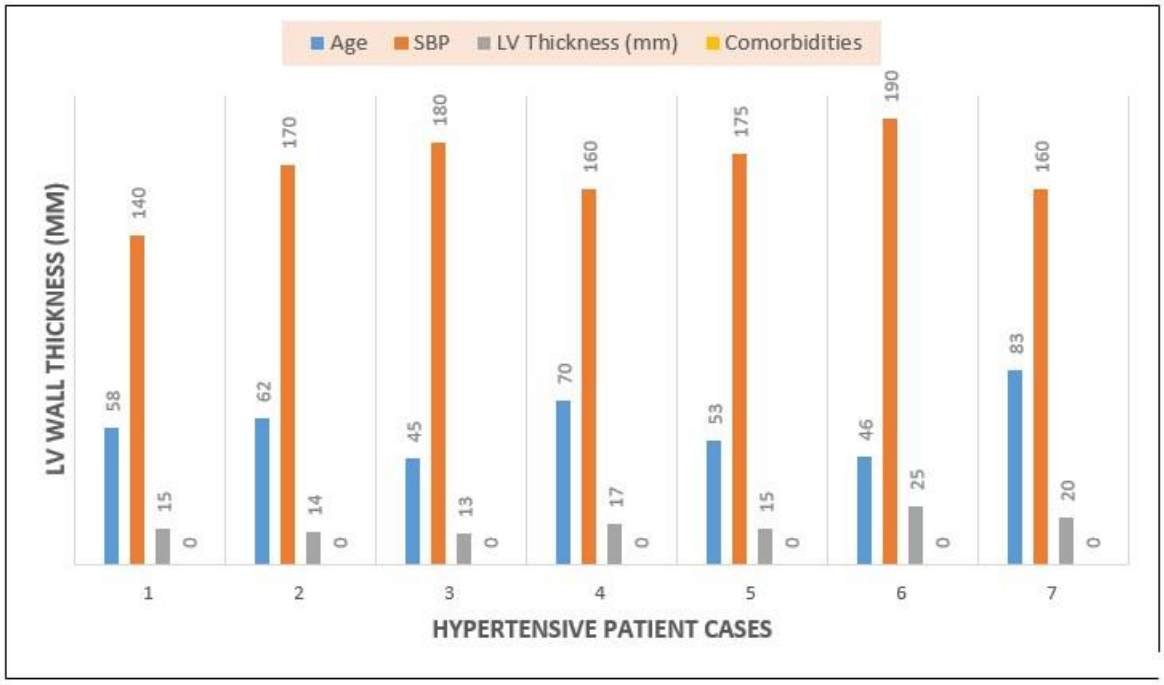

Figure 1: LV Wall Thickness per Case

Table 3. Case Series Summary: Key features, diagnostics, treatments, and follow-up for the above cases [36] [37].

Case 4	Demographics / Risk Factors	Diagnostics (LVH)	Treatment and Follow-up
1	58 M; HTN (8 yr), obesity, DM2	ECG: LVH voltage; Echo: conc. LVH (IVSd 15 mm)	Lisinopril + amlodipine; lifestyle (diet/exercise); ↓BP and LV thickness on 6-mo echo.
2	62 F; HTN (20 yr), HTN non-adherent, smoking, HLD	ECG: LVH; Echo: conc. LVH (posterior wall 14 mm)	Losartan + HCTZ; statin; smoking cessation; salt restriction; repeat echo in 6 mo.
3	45 M; HTN (10 yr), family CAD, obesity	ECG: LVH; Echo: mild conc. LVH (septum 13 mm)	Ramipril + amlodipine; exercise/salt restriction; cardiology referral; stable LVH at follow-up.

4	70 F; HTN (25 yr), DM2, CKD stage3	ECG: LVH with strain; Echo: severe conc. LVH (LVMI ↑↑)	Switched to ARB (candesartan) + loop diuretic; strict DM and fluid management; close renal and echo monitoring.
5	53 M; HTN (12 yr), obesity	ECG: LVH; Echo: conc. LVH (posterior wall 15 mm)	Enalapril + amlodipine; weight loss program; home BP monitoring; improved control.
6 (pub)	46 M;0 HTN from hyperaldosteronism, vascular disease	ECG: severe LVH; Echo/CMR: concentric LVH (23–25 mm); family SCD	Multiple medications; diagnosed HCM; underwent CABG + aortic valve replacement; recovered with AF management.
7 (pub)	83 F; long HTN, AF, recent stroke	ECG: LVH newly present; Echo/CMR: apical LVH (HCM)	Anticoagulation (rivaroxaban), beta-blocker; managed as apical HCM; routine cardiomyopathy follow-up.

As LVH management evolves, computational technologies are playing an increasingly central role. The integration of machine learning models into ECG interpretation software, the use of automated echocardiographic and CMR quantification tools, and the deployment of wearable sensors for remote blood pressure tracking represent practical, scalable applications of health informatics. In our case-based analyses, we referenced how these tools can support early diagnosis, phenotypic classification, and personalized treatment pathways. Future research should focus on refining and validating these computational approaches across larger populations to develop robust, AI-assisted LVH diagnostic platforms.

As this work is based on a narrative synthesis and case-based interpretation, it does not include a large patient cohort or statistical hypothesis testing. The conclusions drawn are illustrative and meant to emphasize emerging trends and computational insights rather than provide statistically validated outcomes. Future research should focus on larger-scale data analysis and algorithm validation to confirm the practical utility of computational tools in LVH diagnosis and management.

Comparative Perspective: Traditional Guidelines vs. Computational-Assisted Decision-Making

While traditional clinical guidelines for managing left ventricular hypertrophy (LVH) are based on well-established algorithms focusing on blood pressure targets, risk factor modification, and pharmacologic strategies, the recent rise of computational tools has introduced more dynamic, personalized, and data-driven approaches to diagnosis and treatment. Computational-assisted decision-making integrates artificial intelligence (AI), wearable biosensors, and decision support systems (DSS) into clinical practice. These tools can enhance the early detection of LVH, identify phenocopies (e.g., HCM), and support individualized risk stratification beyond standard algorithms. A summary of key differences between these approaches is presented in **Table 4**.

Table 4: Comparison Between Traditional Guidelines and Computational-Assisted Decision-Making

Aspect	Traditional Guidelines	Computational-Assisted Decision-Making
Primary Diagnostic Tools	ECG (manual interpretation), echocardiography	AI-enhanced ECG, machine learning in CMR, automated echo quantification
Therapeutic Strategy	Drug-based (ACE inhibitors, ARBs, diuretics) per ESH/ESC, ACC/AHA	Decision support tools suggest personalized therapy plans based on patient phenotype
Monitoring	Clinic-based BP checks, periodic echo	Wearable BP monitors, real-time health tracking via mobile apps
Risk Stratification	Based on clinical risk scores (e.g., Framingham)	ML-based prediction models using multi-parametric data
Secondary Causes Detection	Based on clinical suspicion and physician experience	AI models flag pattern outliers; prompt genetic testing or advanced imaging
Timeliness & Efficiency	Reactive, periodic follow-up	Proactive, real-time feedback loops and early warning systems
Limitations	Population-based, may overlook individual variability	May require data standardization, validation, and integration with EMR systems

5. CONCLUSION

Left ventricular hypertrophy remains a critical marker of cardiovascular risk across populations. Our review underscores that LVH is not merely a benign adaptation; it predisposes to heart failure, arrhythmias, and death. Echocardiography is central to diagnosis, but newer imaging (CMR) provides even greater accuracy. Importantly, hypertension-induced LVH is at least partially reversible: aggressive blood pressure control (target ~120–129 mmHg) and use of ACE inhibitors or ARBs can significantly regress myocardial mass. Recent evidence suggests prompt therapy is essential, as chronic LVH

may become fibrotic and less reversible with time. The presented cases illustrate a range of LVH scenarios – from pure hypertensive remodeling to masquerading genetic cardiomyopathy – highlighting the need for individualized evaluation. Going forward, emphasis on early screening (including in younger or high-risk patients) and adherence to guideline-directed therapy will be paramount to improve outcomes in patients with LVH.

This review is narrative and based on illustrative cases rather than a systematic analysis. Our selected cases and literature overview may introduce selection bias and may not capture every clinical scenario. We also focused on imaging and informatics aspects of LVH, so topics like genetic testing, wearable telemonitoring, or health economics were not explored in depth. These limitations should be considered, and future work with larger cohorts and systematic data should validate the computational approaches to LVH management.

Looking ahead, continued innovation in health technology is likely to transform LVH management. Machine-learning algorithms applied to ECG or imaging can detect subtle LVH patterns earlier, and wearable devices and telehealth platforms can facilitate continuous blood pressure monitoring and management. Integrating these computational tools (such as AI-driven decision support and digital biomarkers) into clinical practice is a key direction for future development in LVH care."

Integrating computational tools with standard guideline-based care provides an opportunity to enhance precision, timeliness, and personalization in LVH management, particularly in resource-limited or high-risk populations.

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