

Review

Hypertensive Left Ventricular Hypertrophy: Pathogenesis, Treatment, and Health Disparities

Sherldine Tomlinson 

School of Health, Science and Wellbeing, Staffordshire University, Leek Road, Stoke on Trent ST4 2DF, UK;
sherldine.tomlinson@research.staffs.ac.uk

Abstract

Hypertensive left ventricular hypertrophy (LVH) is an ominous cardiovascular sequel to chronic hypertension, marked by structural and functional alterations in the heart. Identified as a significant risk factor for adverse cardiovascular outcomes, LVH is typically detected through echocardiography and is characterized by pathological thickening of the left ventricular wall. This hypertrophy results from chronic pressure overload (increased afterload), leading to concentric remodelling, or from increased diastolic filling (preload), contributing to eccentric changes. Apoptosis, a regulated process of cell death, plays a critical role in the pathogenesis of LVH by contributing to cardiomyocyte loss and subsequent cardiac dysfunction. Given the substantial clinical implications of LVH for cardiovascular health, this review critically examines the role of cardiomyocyte apoptosis in its disease progression, evaluates the impact of pharmacological interventions, and highlights the necessity of a comprehensive, multifaceted treatment approach for the prevention and management of hypertensive LVH. Finally, we address the health disparities associated with LVH, with particular attention to the disproportionate burden faced by African Americans and other Black communities, as this remains a key priority in advancing equity in cardiovascular care.

Keywords: apoptosis; left ventricular hypertrophy; cardiomyocyte



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1. Introduction

Hypertension remains a leading modifiable risk factor for cardiovascular morbidity and mortality globally, affecting over 1.28 billion people worldwide, and is the most significant contributor to mortality and morbidity [1,2]. Studies have shown that patients with elevated blood pressure (BP) are up to six times more likely to experience myocardial complications compared to those with normal blood pressure levels [2]. One of these major complications is left ventricular hypertrophy (LVH), a condition characterized by pathological thickening of the left ventricular wall due to chronic pressure overload (e.g., increased afterload) leading to concentric hypertrophy, or hyper diastolic filling (increased preload), which results in an abnormal hypertrophic response [2–8]. Over time, this adaptation reduces myocardial elasticity, progressive fibrosis, and ultimately, cardiac dysfunction [3]. Moreover, LVH was identified as a significant risk factor for high BP in the landmark Framingham Heart Study with the assistance of echocardiography [9]. Since then, it has become a primary focus of cardiovascular research and has been strongly associated with adverse cardiovascular outcomes [8,9].

Despite extensive investigations, the precise mechanisms driving the LVH development remain unknown. However, both historical and emerging evidence suggests that LVH

arises as part of a compensatory remodelling process that alters the heart’s size, shape, and function [2–8]. While this remodelling can serve a protective role in physiological contexts such as sustained athletic training it becomes detrimental when uncontrolled leading to adverse outcomes, including ventricular dysfunction, myocardial infarction (MI), stroke, congestive heart failure (CHF), and sudden cardiac death [3].

A key factor in pathological remodelling is the loss of cardiomyocytes through apoptosis, a controlled, non-inflammatory form of programmed cell death triggered by several factors, including chronic mechanical overload, oxidative stress, and inflammatory signalling cascades [9–13]. In contrast to other cell death like necrosis, which results in unregulated cell rupture and inflammation, apoptosis preserves tissue homeostasis [12,13]. Figure 1 illustrates forms of cardiomyocyte cell death, each with its key features.

Forms of Cardiomyocyte Cell Death

Apoptosis	Necrosis	Autophagy-Associated Cell Death
Features • Cell shrinkage • Chromatin condensation • Membrane blebbing	Features • Cell swelling • Membrane rupture • Uncontrolled release of contents	Features • Formation of autophagosomes • Organelle degradation
Outcome • Programmed cell death • Minimal inflammation • Gradual loss of cardiomyocytes	Outcome • Uncontrolled cell death • Induces inflammation • Tissue damage	Outcome • Initially protective • Excessive activation • Leads to cell death

Figure 1. Forms of cardiomyocyte cell death.

Given the substantial clinical implications of LVH for cardiovascular health, this review critically examines the role of cardiomyocyte apoptosis in its disease progression, evaluates the impact of pharmacological interventions, and highlights the necessity of a comprehensive, multifaceted treatment approach for the prevention and management of hypertensive LVH. Finally, we address the health disparities associated with LVH, with particular attention to the disproportionate burden faced by African Americans and other Black communities as this remains a key priority in advancing equity in cardiovascular care.

2. Cardiomyocyte Apoptosis in Hypertension

Apoptosis, a form of programmed cell death, significantly influences the pathogenesis of hypertensive left ventricular hypertrophy (LVH) through cardiomyocyte loss and progression toward various heart conditions [4,5,9]. First discovered in 1972, this energy-dependent and tightly regulated process is essential for maintaining cell growth and tissue homeostasis, involving a well-defined series of steps that culminate in the controlled dismantling of the cell [5]. Key morphological features of the process include the condensation of chromatin, the fragmentation of the nucleus, cell shrinkage, and the formation of apoptotic bodies [2]. This process is primarily mediated by a family of enzymes known as caspases, with caspase-3 being a central caspase activated through both intrinsic (mitochondrial) and extrinsic apoptotic (death receptor) pathways [2,9,10]. The intricate details of these pathways are further illustrated in Table 1. The intrinsic pathway is activated

by internal cellular stressors including oxidative stress, hypoxia, and DNA damage [10]. These signals lead to mitochondrial outer membrane permeabilization and cytochrome c release, which then activate caspase-9 and downstream caspase-3, ultimately leading to cell death [9,11,12]. Genetic mutations, particularly those affecting sarcomeric proteins, may increase susceptibility to intrinsic apoptosis [9]. While essential for tissue remodelling during embryonic development, where it shapes organ architecture by removing excess cells, uncontrolled intrinsic apoptosis contributes to disease [4–6].

Table 1. Roles of caspases in the intrinsic and extrinsic apoptotic pathways.

Apoptotic Pathway	Initiator Caspase(s)	Executioner Caspase(s)	Function of Caspases
Intrinsic (Mitochondrial)	Caspase-9	Caspase-3, Caspase-7	Caspase-9 is activated by the apoptosome complex following cytochrome c release from mitochondria. It activates caspase-3 and -7, leading to DNA fragmentation and cell dismantling.
Extrinsic (Death Receptor)	Caspase-8	Caspase-3, Caspase-7	Caspase-8 is activated upon ligand binding to death receptors (e.g., Fas, TNFR). It activates executioner caspases and cleaves Bid to tBid, linking to the intrinsic pathway.
Convergent Point	-----	Caspase-3	Caspase-3 is the key executioner caspase common to both pathways. It cleaves structural proteins, DNA repair enzymes, and nuclear components, resulting in controlled cell death.

In contrast, the extrinsic pathway is triggered by the binding of pro-inflammatory cytokines, such as tumour necrosis factor- α (TNF- α) and Fas ligand (FasL), to their respective death receptors on the cardiomyocyte membrane [9,10]. This interaction activates caspase-8 and converges on the same executioner caspase cascade as the intrinsic pathway [10]. In the myocardium, apoptosis, particularly via the extrinsic pathway, is considered pathological when activated under diseased conditions like chronic hypertension or ischemia, leading to the removal of damaged cardiomyocytes and contributing to cardiac dysfunction [5,9,10]. Therefore, a deeper understanding of these molecular pathways offers more promising therapeutic targets for mitigating LVH progression.

Notably, components of the renin–angiotensin–aldosterone system RAAS, particularly angiotensin II, are potent regulators of cardiomyocyte apoptosis. This system plays a central role in regulating BP, fluid balance, and cardiac modelling [14,15]. It achieves this by controlling blood volume, electrolyte homeostasis, and vascular tone, influencing both short-term hemodynamics and long-term structural adaptations in the heart [16]. Hypertension is often characterized by chronic overactivity of the RAAS, which promotes vasoconstriction and fluid retention, thereby increasing BP. In hypertensive heart disease, the delicate balance of cellular signalling within the RAAS is disrupted, leading to maladaptive changes [15,16]. Angiotensin II, a key hormone within the RAAS, exerts its effects mainly through the angiotensin type 1 receptor (AT₁R) and the angiotensin type 2 receptor (AT₂R) [14]. Under normal conditions, a balance between these receptors regulates vascular tone and tissue homeostasis. However, in the context of hypertension, persistent overstimulation of AT₁R promotes pathological processes such as vasoconstriction, pump dysfunction, cardiomyocyte hypertrophy, and inflammation, all of which contribute to adverse cardiac remodelling. Meanwhile, the protective and growth-inhibitory effects

mediated by AT₂R are weakened [14,15]. Over time, progressive pump failure can occur as the heart gradually loses its ability to maintain adequate cardiac output to meet the body's metabolic demands. Clinically, this can manifest as fatigue, shortness of breath, reduced exercise tolerance, and fluid retention, ultimately leading to heart failure and further advancing maladaptive cardiac remodelling [16]. These symptoms significantly impair a patient's quality of life, limiting daily activities and often requiring hospitalization. Shortness of breath and fluid retention, for instance, results from the heart's inability to pump effectively, leading to congestion in the lungs and peripheral tissues.

Studies using Spontaneously Hypertensive Rats (SHR) have been instrumental in demonstrating fluid imbalance within the RAAS system, supporting the development of targeted therapies such as angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs) to manage blood pressure and prevent cardiovascular damage [15,16]. Beyond its role in fluid homeostasis, the RAAS system is also intricately linked to cellular processes involved in cardiac remodelling and apoptosis. Concurrently, other pathways, such as those involving caspases, have been identified as key contributors to cardiomyocyte death during cardiac stress. Consequently, preclinical data exploring inhibition of these pathways, for example, using caspase inhibitors or RAAS modulating agents, has suggested that these interventions can preserve cardiomyocyte integrity, thus delaying conditions like heart failure onset and improving long-term cardiovascular outcomes [14]. While preclinical data is encouraging, the outcomes in human trials have been variable, suggesting differences in response or optimal targeting strategies.

3. Advanced Approaches to Understanding and Managing LVH Progression

Technological innovations have provided researchers with more powerful tools to investigate the complex mechanisms behind cardiovascular diseases and their complications. One significant breakthrough is the improved ability to detect programmed cell death using techniques, such as the TUNEL assay and electron microscopy [4]. Advanced methods like these have allowed scientists to identify and characterize apoptosis and study the condition more effectively, leading to better insights into heart disease progression.

Beyond mechanistic understanding, artificial intelligence (AI) is also revolutionizing diagnostics. For example, an AI-driven model called LVH-fusion was developed to help clinicians distinguish between hypertrophic cardiomyopathy and hypertension-related left ventricular hypertrophy (LVH). This model analyzes data from both 12-lead electrocardiograms (ECGs) and echocardiogram videos, incorporating patient electronic health records, clinical blood pressure measurements, and expert diagnoses from leading specialists [17]. Identifying key diagnostic markers, such as lateral T-wave inversion on ECGs and proximal septal hypertrophy in echocardiograms LVH-fusion enhances diagnostic accuracy, ensuring timely interventions [17].

Early detection and precise diagnosis are, therefore, essential in preventing severe cardiovascular complications, including heart failure, stroke, coronary artery disease, and sudden cardiac death. These diagnostic advancements, coupled with deeper mechanistic insights, highlight the promise of targeted therapies. Indeed, experimental studies, summarized in Table 2, demonstrate how various interventions can reduce apoptosis in LVH cases, revealing potential strategies to preserve heart function and slow disease progression.

Table 2. Experimental mechanisms demonstrating the reduction of apoptosis in LVH.

Intervention	Model Used	Apoptosis Reduction	Authors
Qingda Granule	Spontaneously Hypertensive Rats (SHR)	↓ Caspase-3, ↑ Bax, ↑ Bcl-2	Cheng, Y.; Shen, A.; Wu, X.; Shen, Z.; Chen, X.; Li, J.; Liu, L.; Lin, X.; Wu, M.; Chen, Y.; Chu, J.; Peng, J. Qingda granule attenuates angiotensin II-induced cardiac hypertrophy and apoptosis and modulates the PI3K/AKT pathway [18].
ASK1 Inhibitor	Angiotensin II-induced hypertrophy	↓ JNK pathway activation, ↓ cleaved caspase-3	Savira, F.; Cao, L.; Wang, L.; Yang, W.; Huang, K.; Hua, Y.; Jucker, B.M.; Willette, R.N.; Huang, L.; Krum, H.; Li, Z.; Fu, Q.; Wang, B.H. Apoptosis signal-regulating kinase 1 inhibition attenuates cardiac hypertrophy and cardiorenal fibrosis induced by uremic toxins: Implications for cardiorenal syndrome [19].
Thyroxine (T4)	Isoproterenol-induced cardiac hypertrophy in rats	↓ TUNEL+ cells, ↑ anti-apoptotic genes	Wang, Y.; Jiao, B.; Guo, W.; Che, H.L.; Yu, Z.B. Excessive thyroxine enhances susceptibility to apoptosis and decreases contractility of cardiomyocytes. <i>Mol. Cell. Endocrinol.</i> 2010, 320 (1–2), 67–75 [20].
Ivabradine	Pressure-overloaded rats	↓ apoptotic index, ↑ mitochondrial stability	Yu, Y.; Hu, Z.; Li, B.; Wang, Z.; Chen, S. Ivabradine improved left ventricular function and pressure overload-induced cardiomyocyte apoptosis in a transverse aortic constriction mouse model [21].
Rapamycin	Transverse aortic constriction (TAC) in mice	↓ mTOR activity, ↓ cardiomyocyte death	Gao, G.; Chen, W.; Yan, J.; Liu, J.; Luo, H.; Wang, C.; Yang, P. Rapamycin regulates the balance between cardiomyocyte apoptosis and autophagy in chronic heart failure through mTOR and ER stress pathways [22].
Hydroxysafflor Yellow A (HSYA)	Myocardial infarction in rats	↓ Bax, ↑ Bcl-2, ↓ myocardial injury	Ye, J.; Wang, R.; Wang, M.; Fu, J.; Zhang, Q.; Sun, G.; Sun, X. Hydroxysafflor yellow A ameliorates myocardial ischemia reperfusion injury by inhibiting apoptosis and oxidative stress [23].
Serelaxin	Ischemia–reperfusion injury in mice	↓ Caspase-9 and -3 activity, ↓ TUNEL staining	Wilhelmi, T.; Xu, X.; Tan, X.; Hulshoff, M.S.; Maamari, S.; Sossalla, S.; Zeisberg, M.; Zeisberg, E.M. Serelaxin alleviates cardiac fibrosis through inhibiting endothelial-to-mesenchymal transition via Notch signaling pathway [24].

↓ decrease ↑ increase.

The critical impact of cardiomyocyte apoptosis on patient health cannot be understated, as it is a major contributor to the high mortality and morbidity rates seen in cardiovascular conditions. Individuals with poorly managed hypertension thus face an increased risk of serious outcomes. Recognizing LVH as a predictor of future cardiovascular complications is therefore crucial. Beyond simply controlling blood pressure, healthcare providers must take a broader approach, addressing the causal mechanisms driving LVH to optimize treatment and improve patient cardiovascular outcomes.

4. Clinical Implications

The primary strategy for managing hypertension and the complications from the condition, including LVH, is antihypertensive drug therapy. These pharmacologic treatments help to regulate factors that trigger apoptosis, mitigate apoptosis signalling, and improve patient survival rates. As noted, ACE inhibitors and ARB agents are among the classes of known antihypertensive drugs. ACE inhibitors help to prevent the conversion of angiotensin I to angiotensin II and have demonstrated cardio protective effects by inhibiting apoptosis-mediated cardiac remodelling [25]. Typically, these agents interfere with the breakdown of bradykinin, a peptide that plays a key role in vasodilation (widening of blood vessels), by inhibiting the enzyme responsible for its degradation [25,26]. When this occurs, bradykinin levels increase, enhancing blood vessel relaxation and reducing blood pressure, which supports the outcomes of cardiac patients [25,26].

Similarly, ARBs block angiotensin II from binding to AT1 receptors [27,28]. However, a key difference is that ARBs do not inhibit ACE, and typically, these agents do not lead to an increase in bradykinin levels. As such, this often translates to a lower risk of adverse

results, such as a cough, a common side effect associated with ACE inhibitors [26]. Thus, ARB medications provide an effective alternative for patients who are unable to tolerate ACE inhibitors, while offering similar long-term effectiveness and cardiovascular benefits in the context of hypertensive LVH.

The clinical relevance of the RAAS pathway extends to emerging health challenges, such as the impact of viral infections. Moreover, recent evidence highlighted the role of angiotensin II in the pathophysiology of COVID-19, as the SARS-CoV-2 virus interacts with angiotensin-converting enzyme 2 (ACE2), a key modulator of the RAAS system [29]. The interaction induces dysregulation of angiotensin II signalling, exacerbating inflammation, oxidative stress, and cardiovascular complications, particularly in patients with pre-existing hypertension and LVH [30]. The overlap between angiotensin II pathway convergence in LVH and COVID-19 emphasizes the need for targeted therapeutic intervention (e.g., lifestyle and non-clinical intervention such as dietary modification, exercise, and stress reduction) to avoid cardiovascular damage in high-risk populations.

Well established clinical trials demonstrated the efficacy of antihypertensive medications, forming the foundation for developing effective treatments for cardiovascular conditions. For example, the Study of Left Ventricular Dysfunction (SOLVD) and the CONSENSUS research were pivotal clinical trials demonstrating the efficacy of ACE inhibitor enalapril in patients with left ventricular dysfunction. The SOLVD assessment found a 29% reduced risk of patients developing symptomatic heart failure ($p < 0.001$), though with no statistically significant reduction in all-cause mortality (8% relative risk reduction; $p = 0.30$) [31]. Likewise, CONSENSUS showed that patients who received either enalapril (2.5–40 mg/day) or placebo reduced six-month mortality by 40% (26% vs. 44%) and one year mortality by 31% [32]. Further, the Survival and Ventricular Enlargement (SAVE) and Trandolapril Cardiac Evaluation (TRACE) trials showed that these inhibitors could lower death by 19% in patients with systolic dysfunction following MI [33,34]. The Losartan Intervention for Endpoint (LIFE) experiment explained that the losartan agent was better than the beta-blocker atenolol at reducing left ventricular hypertrophy and cardiovascular morbidity and mortality in patients with hypertension [35] (See Table 3). Finally, a 2023 meta-analysis study concluded that ARB therapy was associated with a marked reduction in left ventricular mass and systolic blood pressure in patients with hypertrophic cardiomyopathy [36].

Table 3. Antihypertensive drug clinical trials.

Clinical Trial	Number of Participants	Treatments Arms	Key Outcomes	References
Study of Left Ventricular Dysfunction (SOLVD)	n = 2569 patients with congestive heart failure symptoms and reduced left ventricular ejection fraction ($\leq 35\%$). 4228 asymptomatic patients who had reduced left ventricular function but no overt heart failure symptoms at enrollment.	Enalapril versus Placebo	Enalapril reduced mortality by approximately 16% compared to placebo. Fewer hospitalizations due to heart failure were seen among enalapril-treated patients. Long-term therapy with enalapril improved survival and reduced hospitalization, a milestone in the management of heart failure.	Yusuf S, Pitt B, Davis CE, Hood WB, Cohn JThN. The SOLVD Investigators. Effect of enalapril on survival in patients with reduced left ventricular ejection fractions and congestive heart failure [31].
Cooperative North Scandinavian Enalapril Survival Study (CONSENSUS) 1987	253 patients with severe CHF, classified as New York Heart Association (NYHA) Class IV	253 patients with severe CHF, classified as New York Heart Association (NYHA) Class IV	Enalapril to conventional therapy can reduce mortality and improve symptoms. It was suggested that enalapril therapy should be taken in low doses, specifically in high-risk individuals.	CONSENSUS Trial Study Group. Effects of enalapril on mortality in severe congestive heart failure. N Engl J Med. 1987 Jun 4;316(23):1429–35. doi: 10.1056/NEJM198706043162301 [32].

Table 3. Cont.

Clinical Trial	Number of Participants	Treatments Arms	Key Outcomes	References
Survival and Ventricular Enlargement (SAVE)	2231 patients aged 21 to 80 years, with a left ventricular ejection fraction (LVEF) of less than 40%, enrolled within 3 to 16 days post-myocardial infarction.	Captopril	Treatment with captopril caused a 19% reduction in all-cause mortality from placebo ($p = 0.019$). In the patients on captopril, there was a strong reduction in the number of major cardiovascular events. Relative risk reduction of 37% ($p < 0.001$) for severe heart failure. Hospitalization for heart failure was 22% relative risk reduction ($p = 0.019$). Recurrent myocardial infarction was 25% relative risk reduction ($p = 0.015$).	Pfeffer MA, Braunwald E, Moyé LA et al. Effect of captopril on mortality and morbidity in patients with left ventricular dysfunction after myocardial infarction [33].
Trandolapril Cardiac Evaluation (TRACE)	6676 patients across 27 centres in Denmark, enrolling 1749 patients with an echocardiographic ejection fraction (EF) of less than 35%	Trandolapril	Trandolapril administration led to a substantial 22% reduction in all-cause mortality compared with placebo (relative risk [RR] = 0.78; 95% confidence interval [CI], 0.67–0.91; $p = 0.001$). There was a 25% reduced cardiovascular cause mortality in the trandolapril arm (RR = 0.75; 95% CI, 0.63–0.89; $p = 0.001$). Trandolapril decreased the incidence of severe heart failure (RR = 0.71; $p = 0.003$). While recurrent AMI events were reduced, the reduction was not statistically significant (RR = 0.86; 95% CI, 0.66–1.13; $p = 0.29$).	Køber L, Torp-Pedersen C, Carlsen JE et al. A clinical trial of the angiotensin-converting-enzyme inhibitor trandolapril in patients with left ventricular dysfunction after myocardial infarction [34].
Losartan Intervention for Endpoint (LIFE) 2002	9193 patients aged 55 to 80 years with essential hypertension and electrocardiographically confirmed LVH.	Losartan 50 mg (n = 457) Atenolol 50 mg (n = 459)	Losartan demonstrated a relative risk reduction of 13% for the composite endpoint of cardiovascular death, myocardial infarction, or stroke compared with atenolol ($p = 0.021$). The losartan group had a notable 25% decrease in the risk of stroke ($p = 0.001$). Losartan was also associated with a 25% reduced incidence of new-onset diabetes mellitus compared to atenolol. Losartan-treated patients experienced greater regression of LVH by electrocardiographic criteria compared with atenolol-treated patients.	Dahlöf B, Devereux RB, Kjeldsen SE et al. Cardiovascular morbidity and mortality in the Losartan Intervention For Endpoint reduction in hypertension study (LIFE): a randomised trial against atenolol. Lancet [35].

All this evidence with positive outcomes from clinical trials reinforces the importance of targeting the RAAS in hypertrophic cardiomyopathy. The results support the continued exploration of ARBs as an improvement therapy in managing both structural and functional cardiac abnormalities. Collectively, these landmark trials have shaped current clinical guidelines, firmly establishing the role of renin-angiotensin system inhibitors and angiotensin receptor blockers as cornerstone therapies in managing hypertension, heart failure, and the regression of LVH. Importantly, ongoing research continues to explore novel agents and combination therapies to optimize cardiovascular outcomes in high-risk populations further.

5. Health Disparities in Hypertensive LVH

Health disparities significantly contribute to the burden of hypertension and other cardiovascular diseases and sequelae, including LVH, in those of African origin compared to

other racial and ethnic groups [36–40]. For example, in the United States, the age-adjusted hypertension prevalence among non-Hispanic Black adults is approximately 58.0%, significantly higher than that of non-Hispanic White adults (around 44.5% overall), contributing to a disproportionately higher burden of associated complications like LVH [39]. These disparities are attributed to several factors, including [40]. A study by Katz and colleagues described several biological pathways theorized to be implicated in the pathophysiologic cascade of LVH and new-onset heart failure (HF) [40]. Certainly, understanding these pathways helps researchers and clinicians identify potential biomarkers for early detection and develop targeted therapies to prevent or treat LVH and HF. Further studies are needed to confirm the above findings and fully elucidate the mechanisms.

There are studies in the United States suggesting that African Americans and other black populations respond differently to specific antihypertensive agents. For example, some studies described that ACE inhibitors and ARBs may be less effective in this population due in part to differences in the RAAS pathway and the higher risk for angioedema [39–42]. Accordingly, despite the higher prevalence of hypertension in Black populations, race-based guidelines recommend different pharmacological approaches for treatment compared to other demographic groups [43]. The guidelines established by the Eighth Joint National Committee (JNC8) in 2014, the American College of Cardiology/American Heart Association (ACC/AHA) in 2017, and the European Society of Cardiology in 2018 advocate a distinct method to initial hypertension therapy for Black and African American individuals with hypertension. For those without comorbidities, thiazide diuretics or calcium channel blockers (CCBs) are advised as first-line treatments. In contrast, angiotensin-ACE inhibitors and ARBs are recommended only for individuals with specific comorbidities. Meanwhile, non-Black individuals can receive any of these three medication classes regardless of the presence of comorbidities [43]. These guidelines were even suggested in the International Society of Hypertension in Blacks (ISHB) consensus statement [43].

An individualized approach to hypertension management is more appropriate and preferable to race-based treatment strategies. Thus, clinicians should carefully evaluate Black patients' risks and benefits, implement personalized monitoring strategies, and consider complementary therapies or adjustments in medication dosage [43]. Ensuring culturally sensitive education and close follow-up can optimize treatment outcomes and mitigate potential risks. Likewise, clinicians ought to carefully consider the potential drug costs when treating African American patients, as economic barriers could influence adherence and access to medication, potentially compromising treatment outcomes [43]. Extensive research is necessary to clarify the clinical outcomes and potential unintended harms of guidelines on the very individuals they aim to benefit.

Furthermore, disparities in targeting efforts require a complex plan or more community-based interventions that focus on social determinants of health. Several targeted programmes have shown promise in improving outcomes among historically underserved populations, particularly African Americans. For example, the Barbershop Hypertension Trial done by authors Victor and colleagues demonstrated significant reductions in blood pressure among Black men who received pharmacist-led interventions in community barbershops [44]. Similarly, the “Check. Change. Control” program by the American Heart Association engages community partners and uses self-monitoring tools to empower hypertensive individuals in managing their blood pressure [38]. Faith-based health initiatives, mobile health clinics, and culturally tailored educational programmes have also been effective in improving medication adherence and lifestyle changes [45,46]. These efforts leverage trust, accessibility, and cultural relevance factors often missing from traditional clinical settings. Community-based settings are particularly effective for this, as they offer culturally

sensitive education on a variety of health topics, e.g., nutrition, exercise, and medication compliance. Interventions that facilitate access to medical care, lower medication costs, or build on cultural factors are those that are required to reduce the excess LVH burden among African American communities. Expanding and investing in such initiatives enhances health equity and creates scalable models for chronic disease management in high-risk communities. Future research could consider including implementing population-specific guidelines for treatment and carrying out pharmacogenomic studies to clarify biological determinants of health disparities in cardiovascular disease. This would support improving tailored interventions and enhancing healthcare outcomes for diverse populations.

6. Lifestyle Modification

A combination of pharmacological treatments and lifestyle modifications (LM) is highly recommended. With minimal side effects, LM serves as an early treatment before starting drug therapy, is a step down to drug treatment, and is a medication withdrawal for those patients who have achieved and sustained lifestyle changes [47]. Studies have highlighted the effectiveness and recommendation of lifestyle approaches in managing and preventing hypertension and hypertensive-related conditions including LVH. For instance, the DASH (Dietary Approaches to Stop Hypertension) has shown positive to reduce these heart diseases, with the diet emphasizing the consumption of fruits, vegetables, whole grains, lean proteins, and low-fat dairy products while reducing sodium intake [48–50]. Supplements with fish oils, calcium, magnesium, potassium vitamin C, and antioxidants may offer additional benefits, although evidence varies [51]. Moreover, exercise training as an LM is optimal for increasing functional capacity and psychological well-being [47,52]. In African American cohorts, community-based structured physical activity interventions integrating faith-based therapeutic lifestyle modifications and nutritional education have been found to significantly lower BP and improve participants' quality of life [53]. Therefore, healthcare professionals need to adopt and support a multi-component, community-based approach to effectively address health outcomes and promote well-being within African American communities.

A meta-analysis comparing the effects of high-intensity interval training (HIIT) and moderate-intensity continuous training (MICT) on BP in hypertensive patients found that both HIIT and MICT effectively reduced systolic and diastolic BP [54]. Notably, HIIT showed a greater reduction in daytime systolic BP and improved flow-mediated vasodilation compared to MICT. Integrating structured physical activity into hypertension education enhances both clinical outcomes and patient engagement, making it a cornerstone of effective BP and related conditions management strategies. When implemented consistently, these behavioural changes help reduce cardiac workload, significantly improve function, alleviate symptoms, and may even reverse structural abnormalities such as LVH. Clinicians should, therefore, prioritize and strongly advocate for lifestyle interventions as foundational elements in the management of cardiac conditions. Various innovative and tailored approaches to implementing LM in LVH treatment and management are summarized in Table 4.

Table 4. Approaches to treating and managing hypertensive LVH.

Studies	Non-Clinical Approaches	Key Benefits
D’Andrea, A.; Carbone, A.; Ilardi, F.; Pacileo, M.; Savarese, C.; Sperlongano, S.; Di Maio, M.; Giallauria, F.; Russo, V.; Bossone, E.; Picano, E. Effects of High Intensity Interval Training Rehabilitation Protocol after an Acute Coronary Syndrome on Myocardial Work and Atrial Strain [55].	High-Intensity Interval Training (HIIT)	Reverse adverse cardiac remodelling, improve LV function, myocardial efficiency and aerobic capacity.
Goessler, K.F.; Buys, R.; VanderTrappen, D.; Vanhumbeeck, L.; Cornelissen, V.A.J. A randomized controlled trial comparing home-based isometric handgrip exercise versus endurance training for blood pressure management [56].	Isometric Resistance Training	Isometric Resistance Training is a viable and effective strategy for reducing blood pressure and may be beneficial for both arterial stiffness and left ventricular function.
Lightner, J.S.; Collinson, S.; Grimes, A. Cost Analysis of a Culturally Appropriate, Community-Delivered Intervention to Increase Physical Activity [57].	Dance-Based Aerobic Exercise	Culturally tailored cardiovascular intervention for ethnic minority populations.
Murugesan, P. Yoga and Cardiovascular Diseases—A Mechanistic Review [58].	Yoga & Tai Chi	Lowers sympathetic nervous system activity and improves autonomic function.
Burlacu, A.; Brinza, C.; Popa, IV.; Covic, A.; Floria, M. Influencing Cardiovascular Outcomes through Heart Rate Variability Modulation: A Systematic Review [59].	HRV Training	Reduces stress and cardiac workload.
Schneider, R.H.; Myers, H.F.; Marwaha, K.; Rainforth, M.A.; Salerno J.W.; Nidich, S.I.; Gaylord-King, C.; Alexander, C.N.; Norris, K.C. Stress Reduction in the Prevention of Left Ventricular Hypertrophy: A Randomized Controlled Trial of Transcendental Meditation and Health Education in Hypertensive African Americans [60].	Transcendental Meditation	Stress reduction with TM was effective in preventing LVMI progression and thus may prevent LVH.
Yau, K.K.; Loke, A.Y. Effects of forest bathing on pre-hypertensive and hypertensive adults: a review of the literature [61].	Green Prescriptions & Nature Therapy	Reduces stress hormones and improves cardiovascular function.
Mohol, P.; Ghosh, A.; Kulkarni, S. Blue Zone Dietary Patterns, Telomere Length Maintenance, and Longevity: A Critical Review [62].	Blue Zones-Inspired Lifestyle Adaptations	Promotes longevity through strong social networks, natural movement, and stress reduction, which correlates with lower rates of hypertension and cardiovascular disease.
Gentile, F.; Orlando, G.; Montuoro S; Ferrari Chen YF; Macefield V.; Passino, C.; Giannoni, A.; Emdin M. Treating heart failure by targeting the vagus nerve [63].	Transcutaneous Vagus Nerve Stimulation (tVNS)	Cardio-vagal baroreflex gain in CHF patients with reduced LVEF, without causing discomfort or physiologic disturbance and reduces cardiac stress.
Tagashira, H.; Abe, F.; Sakai, A.; Numata T. Shakuyaku-Kanzo-To Prevents Angiotensin II-Induced Cardiac Hypertrophy in Neonatal Rat Ventricular Myocytes [64].	Acupuncture & Traditional Chinese Medicine (TCM)	Modulates autonomic function and reduces LVH-related complications.
Mu, L.; Li, C.; Zhao, W.; Li, A.; Zhao, D.; Zhang, B. Association between Sleep Duration and Left Ventricular Hypertrophy for Patients with Type 2 Diabetes Mellitus [65].	Optimizing Sleep Patterns	Reduces cardiac remodelling and hypertension-related stress.

Table 4. Cont.

Studies	Non-Clinical Approaches	Key Benefits
Gumz, M.L.; Shimbo, D.; Abdalla, M.; Balijepalli, R.C.; Benedict, C.; Chen, Y.; Earnest, D.J.; Gamble, K.L.; Garrison, SR; Gong, M.C.; Hogenesch, J.B. Toward Precision Medicine: Circadian Rhythm of Blood Pressure and Chronotherapy for Hypertension—2021 NHLBI Workshop Report [66].	Circadian Rhythm Regulation	Supports blood pressure control through synchronized sleep and eating patterns.

7. Conclusions

In summary, apoptosis is a physiological cell death process that, if deregulated, can contribute to the excess loss of cardiomyocytes and induce heart failure. The involvement of apoptosis in hypertensive LVH underscores the importance of applying specific pharmacologic interventions. Antihypertensive treatment, particularly ACE inhibitors and ARBs, represents a promising direction for inhibiting apoptosis, reducing cardiac remodelling, and improving patient outcomes.

Redressing health inequities is also paramount in mitigating the disproportionate burden of LVH among populations at risk, such as African Americans. Culturally sensitive healthcare delivery, tailored pharmacologic therapeutic strategies, and expanded access to preventive services can reduce the gap in cardiovascular equity. Further exploration of social determinants of health and pharmacogenomics will be essential in the quest for equitable outcomes in treating hypertensive LVH. Correspondingly, pharmacologic therapy adjuncts and novel non-clinical interventions, such as lifestyle modifications, offer effective multidimensional strategies for the treatment of LVH. These approaches can further improve outcomes and potentially reverse pathological cardiac changes.

Despite advances in understanding the molecular mechanisms of cardiomyocyte apoptosis in hypertensive heart disease, several gaps remain. In particular, the translational relevance of many experimental interventions, such as caspase inhibitors or natural compounds, requires further validation in clinical settings. Additionally, most existing studies lack population specific data, overlooking ethnic and genetic variability in treatment response. Future research should focus on clinical trials that evaluate these therapeutic agents in diverse populations and mechanistic studies that explore long-term effects and safety profiles. Bridging these gaps is essential for developing targeted, equitable interventions to effectively reduce the burden of LVH and heart failure.

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