American Heart Journal Plus: Cardiology Research and Practice 39 (2024) 100367

Contents lists available at ScienceDirect

American Heart Journal Plus: Cardiology Research and Practice

journal homepage: www.sciencedirect.com/journal/american-heart-journal-plus-cardiology-research-and-practice

Review Article

Clinical course of adults with co-occurring hypertrophic cardiomyopathy and hypertension: A scoping review

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ARTICLE INFO

Keywords:
Hypertrophic cardiomyopathy
Hypertension
Scoping review

ABSTRACT

Introduction: Hypertension affects approximately 50 % of patients with hypertrophic cardiomyopathy (HCM) but clinical course in adults with co-occurring HCM and hypertension is underexplored. Management may be challenging as routine anti-hypertensive medications may worsen obstructive HCM, the most common HCM phenotype. In this scoping review, we sought to synthesize the available literature related to clinical course and outcomes in adults with both conditions and to highlight knowledge gaps to inform future research directions.
Methods: We searched 5 electronic databases (PubMed, CINAHL, Scopus, Embase, Web of Science) to identify peer-reviewed articles, 2011–2023. We followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses-Scoping Review (PRISMA-ScR) guideline.
Results: Eleven articles met eligibility. Adults with both conditions were older and had higher rates of obesity and diabetes than adults with HCM alone. Results related to functional class and arrhythmia were equivocal in cross-sectional studies. Only 1 article investigated changes in medical therapy among adults with both conditions. Hypertension was a predictor of worse functional class, but was not associated with all-cause mortality, heart failure-related mortality, or sudden-death. No data was found that related to common hypertension-related outcomes, including renal disease progression, nor patient-reported outcomes, including quality of life.
Conclusions: Our results highlight areas for future research to improve understanding of co-occurring HCM and hypertension. These include a need for tailored approaches to medical management to optimize outcomes, evaluation of symptom burden and quality of life, and investigation of hypertension-related outcomes, like renal disease and ischemic stroke, to inform cardiovascular risk mitigation strategies.

1. Introduction

Hypertrophic cardiomyopathy (HCM) is the most common inherited cardiac disorder, affecting at least 1:500 individuals [1,2]. While associated with sudden death, HCM is increasingly viewed as a manageable chronic condition due to advances in diagnostic methods, higher public awareness of the disease, and HCM treatment [3,4]. Thus, there is a growing need to better understand and manage common comorbidities that impact individuals with HCM. Hypertension is the most common chronic condition in the US, affecting 47.3 % of the population overall, with noted racial disparities in prevalence and outcomes [5,6]. It is the major risk factor for coronary artery disease, renal disease and stroke, and is associated with an increased risk of mortality [7]. Hypertension co-occurs in approximately 40–60 % of adults with HCM [8–11]. Yet, clinical course and outcomes in adults with co-occurring HCM and hypertension are underexplored.

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https://doi.org/10.1016/j.ahjo.2024.100367
Received 4 December 2023; Received in revised form 22 January 2024; Accepted 29 January 2024
Available online 2 February 2024

Co-management of HCM and hypertension may present a challenge. Clinical guidelines for HCM advise against use of medications with vasodilatory properties in those with left ventricular outflow tract (LVOT) obstruction, a phenotype that occurs in approximately 70 % of individuals with HCM, because of their potential to worsen obstruction and exacerbate symptoms including dyspnea and exercise intolerance [4]. While the recommendations are based on limited data and primarily rely on expert consensus, they impact the use of first- and second-line anti-hypertensive medications because of their vasodilatory effects (e.g., angiotensin converting enzyme inhibitors, angiotensin receptor blockers, dihydropyridine calcium channel blockers) [4,7,12–14]. Vasodilators are not contraindicated in individuals with non-obstructive HCM [4]. However, confirmation of the lack of obstruction is important and requires a thorough work-up, including provocative non-invasive and invasive maneuvers, like echocardiographic imaging after eating or after amyl nitrite administration [15,16]. Recent advances in the treatment of obstructive HCM have not shown differences between those with and without hypertension, though the science is still evolving [17]. Management of co-occurring HCM and hypertension is underexplored, constraining clinical decision-making and potentially limiting optimization of outcomes [18–20].

With this scoping review, we sought to systematically synthesize the existing literature on the management, clinical profile, and disease course of adults with co-occurring HCM and hypertension. Knowledge and evidence gaps identified through the review process will help inform future research directions related to improved understanding and clinical management of adults with both conditions to optimize outcomes. A preliminary search of the literature yielded limited results, supporting the methodologic choice of undertaking a scoping review [21,22].

2. Methods

This review is registered in the Open Science Framework https://osf.io/cy8qb/?view_only=fbb1dc1b2fe441d0b5dd4e18a78f42c3 and the review protocol methods, including search strategy, is published elsewhere [23]. In brief, we conducted a literature search in 5 electronic data bases, PubMed, CINAHL, Embase, Web of Science, and SCOPUS, and reference lists of published articles, to identify eligible articles. Included articles were those that were peer-reviewed, published in English, and comprised of adult (age $\geq$ 18 years) cohorts with a confirmed HCM diagnosis via standard HCM diagnostic criteria [4]. Furthermore, eligible articles analyzed either: 1) medical management; 2) clinical course; and/or 3) outcomes in adults with co-occurring HCM and hypertension. Search terms and yield for each database are presented in **Supplemental Table 1**. We excluded abstracts, case studies/reports, and editorials as they present incomplete data, low level of evidence, or opinion [24]. We also excluded articles where only hypertension prevalence was reported or where there were no association analyses between hypertension and other variables. The time frame for articles eligibility was from January 1, 2011 to June 30, 2023. Identification of eligible articles occurred in 2 stages: 1) title and abstract review; and 2) full-text review. Two study team members conducted independent screening at both stages of review, with disagreements related to eligibility of articles resolved by a senior member of the study team or by consensus. Ethical approval was not sought as the review examined secondary, published data and did not meet the criteria for human subjects research.

3. Results

Eleven articles met review criteria. The PRISMA-ScR flowchart showing the article selection process is presented in **Supplemental Fig. 1**. The articles comprise cohorts from Asia (China, Korea), Europe (Spain, United Kingdom, and the European Society of Cardiology Cardiomyopathy and Myocarditis Registry), and the United States. All were observational, 3 prospective [25–27], and 8 retrospective [8,10,18,20,28–31]. One study specifically recruited related individuals from a pool of families with HCM, including carriers of HCM-related variants within the MYBPC3 and MYH7 genes [25], whereas other recruited unrelated individuals, or did not specify whether participants were related. One study focused on adults with apical HCM [28]; 2 focused on adults with obstructive HCM only [27,29], and the rest included adults with varied subtypes of HCM. Six studies defined hypertension and used Joint National Congress 7 (JNC7) criteria, systolic blood pressure (SBP) $\geq$140 mmHg, diastolic blood pressure (DBP) $\geq$ 90 mmHg, or taking anti-hypertensive medication [10,20,22,31,34,35]. Article details are presented in **Table 1**.

3.1. Clinical profile of adults with co-occurring HCM and hypertension

Clinical profile characteristics of adults with co-occurring HCM and hypertension, compared to adults with HCM alone, are presented in **Table 2**. Adults with co-occurring HCM and hypertension were consistently older across studies compared to those with HCM alone [10,22,31–35]. Co-occurring hypertension was more prevalent among Black adults, compared to White adults, with HCM, though only one study reported race [10]. Hypertension prevalence was largely equally distributed by sex [31,32,34,35], though one study noted differences in prevalence by sex when age was stratified, there was a higher prevalence of hypertension in men age $\leq$37 (first quartile) and women age $\geq$ 75 (last quartile),in a cohort with a median age of 55 [8]. Adults with HCM and hypertension were more likely to have other comorbidities, including diabetes, obesity, and hyperlipidemia compared to adults with HCM alone [22,30–32,34,35]. A minority of articles examined history of stroke/transient ischemic attack or renal function among their participants, though they found that adults with HCM and hypertension had worse renal function and more history of stroke/transient ischemic attack compared to those with HCM alone [22,31,32,35]. A history of syncope was generally lower among those with co-existing hypertension [10,22,31–35]. Findings related to history of arrhythmia, including atrial fibrillation or ventricular arrhythmias, were equivocal [10,22,31–35].

3.2. Medical management of adults with HCM and hypertension

Adults with HCM and hypertension were more likely to be on direct vasodilators (angiotensin converting enzyme inhibitors [ACE-I], or angiotensin receptor blockers [ARB]) and calcium channel blockers (both dihydropyridines and non-dihydropyridines) compared to adults with HCM only [22,34,35]. There was no difference in beta blocker administration between HCM patients with versus without hypertension [18,25].

Only two articles addressed blood pressure control over time. Sheikh et al. reported that 81.5 % of their cohort maintained SBP $<$ 140 mmHg or DBP $<$ 90 mmHg over mean (SD) 44.1 (23.9) months (range 5–120 months). However, the authors did not specify which medications constituted anti-hypertensive therapy and did not specify when and how

Table 1
Overview of articles included in this scoping review.

First author (year)	Study design and funding source	Study location (time period)	Study aim	Sample size (+ HTN)	Inclusion criteria *All studies used standard diagnostic criteria for HCM unless otherwise noted	Exclusion criteria	Criteria to define HTN	HCM subtypes (e.g., obstructive, non-obstructive, mid-ventricular obstruction, apical) included in the study cohorts
Wang et al. 2023	Retrospective Cohort Funding from Sichuan Science and Technology Program, China (2022YFS0186); National Natural Science Foundation of China (81600299).	Chengdu, China (2008–2018)	To investigate the impact of HTN on the prognosis of HCM patients	$N = 468$ + HTN $N = 149$	Hospitalized patients with a discharge diagnosis of HCM	Amyloid CM, restrictive CM, dilated CM, myocarditis	JNC7 criteria or taking anti-hypertensive medications	Not specified
Zhang et al. 2023*	Prospective Cohort Funding from National Natural Science Foundation of China (81870286); CAM Innovation Fund for Medical Science (2022-I2M-1-005 & 2020-I2M-C&T-A-006)	Beijing, China (not specified -July 2020)**	To elucidate if concomitant HTN may affect the disease trajectory and morbidity of HCM in the septal reduction therapy cohort and non-septal reduction therapy cohort separately	$N = 696$ + HTN $N = 150$ ($N = 50$ in SRT cohort and $N = 100$ in non-SRT cohort)	HCM, confirmed with genetic testing	None specified	JNC7 criteria or taking anti-hypertensive medications	Obstructive and non-obstructive HCM; did not specify type of obstruction (LVOT or mid-ventricular)
Lopes et al. 2023	Cross-sectional Funding from British Heart Foundation, GOSH NIHR BRC, Medical Research Council Clinical Academic Partnership award	EUR Observational Research Program -cardiomyopathy registry (2012–2016)	To report the prevalence of cardiovascular risk factors in patients with HCM and to determine their association with clinic phenotype	$N = 1739$ + HTN $N = 648$	Age > 18; diagnosis of HCM	None specified	Not defined	Not specified
Luo et al. 2020	Case-control Funding from National Natural Science Foundation of China (81670420); Natural Science Foundation of Hunan Province of China (2018JJ1045)	Hunan, China (2014–2018)	To study the clinical features, cardiac structure, functional changes and prognosis of HCM patients with HTN	$N = 262$ + HTN $N = 90$	Diagnosed with HCM between 2014 and 2018	Myocardial hypertrophy due to amyloidosis, aortic stenosis, hypothyroidism	JNC7 criteria or taking anti-hypertensive medications	Not specified Sub-group analysis of adults with apical HCM and mid-ventricular obstruction HCM performed
Zhou et al. 2020*	Cross-sectional Funding from internal institutional sources	Beijing, China (2013–2016)	To investigate different roles of systemic HTN on left ventricular remodeling in male and female patients with obstructive HCM	$N = 453$ + HTN N = 150	Obstructive HCM (LVOT gradient not specified); controlled HTN (criteria for controlled HTN not specified)	coronary artery disease, cardiac valve disease; left ventricular EF <50 %; secondary HTN; infection; pregnancy; connective tissue disease; neoplasm; percutaneous transluminal septal myocardial ablation; septal myectomy; permanent mechanical device implantation	JNC7 criteria or taking anti-hypertensive medications	Obstructive HCM (with LVOT obstruction) only
Smith et al. 2018	Retrospective cohort Funding from National Institutes of Health (R25HL092621; HL12663); American Heart Association (18POST3990251)	Site Unspecified*** (1995–2016)	To determine the relationship between demographic factors, comorbidities, and echocardiography indices with the compromised VO2 peak in obstructive HCM patients	$N = 1177$ + HTN $N = 582$	Obstructive HCM (LVOT gradient ≥30 mmHg)	None specified	Not defined	Obstructive HCM (with LVOT obstruction) only
Perez-Sanchez	Prospective cohort Funding from Fundación Espanola del Corazon-Coca-Cola LTD (2007);	Spain (time not specified)§	To determine whether factors such as sex, systemic HTN, or physical activity are modifiers of disease	$N = 183$ from a pool of 72 families with	Participants recruitment from an initial pool of 72	Do not carry a family HCM-related variant	Not defined	Not specified

(*continued on next page*)

Table 1 (*continued*)

First author (year)	Study design and funding source	Study location (time period)	Study aim	Sample size (+ HTN)	Inclusion criteria *All studies used standard diagnostic criteria for HCM unless otherwise noted	Exclusion criteria	Criteria to define HTN	HCM subtypes (e.g., obstructive, non-obstructive, mid-ventricular obstruction, apical) included in the study cohorts
et al. 2018	Fundación para la Formacion e Investigacion Sanitaria de la Region de Murcia; Carlos III Health Institute (RIC; RD12/0042/0049)		severity and to establish their role in age-related penetrance of HCM	HCM + HTN N = 58	families; met diagnostic criteria for HCM			
Geske et al. 2017***	Retrospective cohort Funding source not specified	Minnesota, USA 1975–2012	To characterize sex differences in a large HCM referral center population	*N* = 3673 + HTN *N* = 1690	Adults with HCM; index evaluation at study center between 1975 and 2012	None specified	Not defined	Not specified
Sheikh et al. 2016	Retrospective cohort Funding from the British Heart Foundation and charitable foundation "Cardiac Risk in the Young"	London, UK (2001–2014)	To address differences in the clinical phenotype, risk factor profile, and outcome of HCM between Afro-Caribbean and White patients	*N* = 425 + HTN *N* = 178	Diagnostic criteria for HCM *Participants with a history of HTN, HCM was diagnosed with LVH $\geq$ 20 mm or $\geq$ 15 mm and additional criteria: 1. Gene mutation 2. Family history of HCM or sudden cardiac death in first-degree relative 3. Non-concentric LVH, segmental patterns of LVH confined to apical segments 4. Systolic anterior motion of the mitral valve leaflets	None specified	JNC7 criteria or taking anti-hypertensive medications	Not specified
Argulian et al. 2013	Retrospective cohort Funding source not specified	New York, USA (1995–2011)	Assess the impact of a stepwise, symptom-oriented approach to treating concurrent HCM and HTN on LVOT obstruction, HCM-symptoms, HTN control, and clinical outcomes	*N* = 115 + HCM N = 115	Co-occurring HCM and HTN; referred to study site program between 1995 and 2011	• Hypertrophied nondilated left ventricle$\geq$15 mm inappropriate for the degree of HTN. AND 1 of the 3 criteria: 1. Dynamic LVOT obstruction due to systolic anterior motion of the mitral valve and mitral-septal contact at rest or with physiologic provocation 2. Apical or apical-mid hypertrophy sparing the base, marked asymmetric hypertrophy with systolic anterior motion but with gradients <30 mmHg, or severe asymmetric hypertrophy with mild HTN. 3. In patients with nonobstructive HCM, ancillary supportive criteria were a family history of HCM in a first-degree family member or genotype analysis showing an HCM-related sarcomeric protein mutation	JNC7 criteria or taking anti-hypertensive medications	Obstructive HCM (with LVOT obstruction), non-obstructive HCM (including apical and mid-ventricular LVH)

(*continued on next page*)

Table 1 (*continued*)

First author (year)	Study design and funding source	Study location (time period)	Study aim	Sample size (+ HTN)	Inclusion criteria *All studies used standard diagnostic criteria for HCM unless otherwise noted	Exclusion criteria	Criteria to define HTN	HCM subtypes (e.g., obstructive, non-obstructive, mid-ventricular obstruction, apical) included in the study cohorts
Moon et al. 2011	Case-control Funding source not specified	Seoul, Republic of Korea 2003–2009	To investigate the prognosis of patients with apical HCM undergoing treatment at a large-volume referral center and to determine the clinical and echocardiographic predictors of a poor outcome	$N = 454$ + HTN $N = 232$	Apical HCM; diagnosed between 2003 and 2009	EF <50 %; significant valvular disease; significant coronary artery disease • Asymmetric LVH confined predominantly to the left ventricular apical portion with an apical wall thickness of 15 mm Ratio of maximal apical to left ventricular posterior wall thickness of 15 mm at end-diastole assessed using standard 2-dimensional transthoracic echocardiography	Not defined	Apical HCM

Abbreviations: CM – cardiomyopathy; EF – ejection fraction; HCM- hypertrophic cardiomyopathy, HTN- hypertension, JCN7- The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure – hypertension defined as systolic blood pressure ≥ 140 mm HG, diastolic blood pressure ≥ 90 mm HG; LVH – left ventricular hypertrophy; LVOT – left ventricular outflow tract; SRT – septal reduction therapy.

*Zhou et al. (2020) and Zhang et al. (2023) have the same study location, Fuwai Hospital National Center for Cardiovascular Diseases, Beijing, China. We cannot ascertain whether the study cohorts overlap.

**Zhang et al. (2022) did not specify beginning date for data collection; median follow-up for SRT cohort is reported as 4.9 years-no interquartile range reported; median follow-up for non-SRT cohort is reported as 5.9 years – no interquartile range reported.

***Smith et al. (2018) did not specify a study location, authors are affiliated with Mayo Clinic Rochester, MN, USA and report a single center study. Geske et al. (2017) analyzed patient data from the same location. We cannot ascertain whether the study cohorts overlap.

§Perez-Sanchez et al. (2018) did not specific time frame for data collection; mean reported follow-up was 5.5 ± 3.3 years.

Table 2
Clinical profile of adults with HCM and hypertension compared to adults with HCM alone.

Article	HCM and hypertension compared to HCM alone	Effect
Age (years)		
Wang et al. 2023	65 [57.0;73.0] vs. 52 [41.5;63.0]	↑
Zhang et al. 2023	SRT: 51.1 ± 8.3 vs. 39.4 ± 14.7 Non-SRT: 57.3 ± 12 vs. 43.5 ± 14.4	↑
Lopes et al. 2022*	60 [51.0;67.0] vs. 44 [32.0;55.0]	↑
Luo et al. 2020	55 ± 12.5 vs. 47 ± 16.2	↑
Zhou et al. 2020	55.8 ± 9.3 vs. 45.2 ± 12.8	↑
Perez-Sanchez et al. 2018*	66.9 ± 10.8 vs. 46.9 ± 14.7	↑
Sheikh et al. 2016	59.5 ± 13.3 vs. 44.7 ± 16.7	↑
Sex (%male with HCM and hypertension compared to %male with HCM alone)		
Wang et al. 2023	75 (50.3 %) vs. 140 (43.9 %)	↑
Zhang et al. 2023	SRT: 34 (68 %) vs. 172 (68.8 %) Non-SRT: 61 (61 %) vs. 186 (62.8 %)	↔
Lopes et al. 2022	57.3 % (371) vs. 60.2 % (657)	↔
Luo et al. 2020	61 (68 %) vs. 101 (59 %)	↔
Zhou et al. 2020	81 (54 %) vs. 171 (56 %)	↔
Perez-Sanchez et al. 2018	34 (58.6 %) vs. 87 (69.6 %)	↓
Sheikh et al. 2016	100 (56.2 %) vs. 182 (73.7 %)	↑
Race (Black compared to White)		
Sheikh et al. 2016	95 (53.4 %) vs. 68 (27.5 %)	↑
Systolic blood pressure (mm HG)		
Wang et al. 2020	Median [IQR] 136 [122;150] vs.116 [105;127.5]	↔
Zhou et al. 2020	Mean (SD) 128.0 (18.8) vs. 112.6 (14.2)	↑
Diastolic blood pressure (mm HG)		
Wang et al. 2020	Median [IQR] 78 [70;85] vs 70 [64; 80]	↔
Zhou et al. 2020	Mean (SD) 76.7 (11.0) vs. 69.3 (9.1)	↑
Body Mass Index (kg/m^2)		
Zhang et al. 2023	SRT: 26.2 ± 3.5 vs. 24.2 ± 3.3 Non-SRT: 26.5 ± 3.4 vs. 24.8 ± 3.5	↑
Lopes et al. 2022	27.8 [25.1;31.1] vs. 25.7 [23.2;28.4]	↑
Zhou et al. 2020	27.1 ± 3.5 vs. 24.5 ± 3.4	↑
Diabetes		
Wang et al. 2023	24 (16.1 %) vs. 15 (4.7 %)	↑
Lopes et al. 2022	116 (17.9 %) vs. 47 (4.3 %)	↑
Zhou et al. 2020	21 (14 %) vs. 10 (3.3 %)	↑
Hyperlipidemia		
Lopes et al. 2022	369 (56.9 %) vs. 266 (24.4 %)	↑
Luo et al. 2020	31 (38 %) vs. 35 (22 %)	↑
Zhou et al. 2020	87 (58 %) vs. 66 (21.8 %)	↑
Coronary artery disease		
Luo et al. 2020	27 (33 %) vs. 35 (22 %)	↑
NYHA functional class (higher NYHA class)**		
Wang et al. 2023	51 (34.2 %) vs. 111 (34.8 %)	↔
Lopes et al. 2022	107 (20 %) vs. 138 (16 %)	↑
Zhou et al. 2020	50 (33 %) vs. 129 (42.6 %)	↔
Perez-Sanchez et al. 2018	12 (21 %) vs. 10 (8 %)	↑
Lower max VO2 peak (ml/kg/min)		
Lopes et al. 2022	17.1 [14.0;20.5] vs. 21.0 [17.5;27.1]	↑
Smith et al. 2018 (VO2 peak <21.51 ml/kg/min)***	432 (48.9 %) vs. 451 (51.1 %)	↔
History of syncope		
Wang et al. 2023	32 (21.5 %) vs. 114 (35.7 %)	↓
Zhang et al. 2023	SRT: 7 (14 %) vs. 58 (23 %) Non-SRT: 8 (8 %) vs. 29 (10 %)	↔
Luo et al. 2020	7 (8 %) vs. 37 (22 %)	↓
Zhou et al. 2020	22 (14.6 %) vs. 93 (30.7 %)	↓
Lopes et al. 2022	85 (15.4 %) vs. 178 (19.8 %)	↓
Perez-Sanchez et al. 2018	13 (23 %) vs. 12 (10 %)	↑
Sheikh et al. 2016	32 (18 %) vs. 48 (19.4 %)	↔
History of atrial fibrillation		
Wang et al. 2023	31 (20.8 %) vs. 52 (16.3 %)	↑
Zhang et al. 2023	SRT: 12 (24 %) vs. 36 (14 %) Non-SRT 23 (23 %) vs. 63 (21 %)	↔
Lopes et al. 2022	205 (31.6 %) vs. 258 (23.7 %)	↑
Luo et al. 2020	31 (34 %) vs. 50 (29 %)	↔
Zhou et al. 2020	22 (15 %) vs. 42 (14 %)	↔
Perez-Sanchez et al. 2018	24 (47 %) vs. 20 (19 %)	↑
History of non-sustained ventricular tachycardia		
Zhang et al. 2023	SRT: 1 (2 %) vs. 14 (5.6 %) Non-SRT: 5 (5 %) vs. 20 (6.8 %)	↔
Lopes et al. 2022§	77 (17.9 %) vs. 160 (21.9 %)	↔
Luo et al. 2020	8 (9 %) vs. 21 (12 %)	↔
Zhou et al. 2020	12 (18 %) vs. 32 (20 %)	↔
Perez-Sanchez et al. 2018§	25 (57 %) vs. 22 (24 %)	↑
Sheikh et al. 2016	45 (26.2 %) vs. 57 (24.9 %)	↔
Renal dysfunction		
Wang et al. 2023	85 [71.0;101.90] vs.77 [66.0;90.0] creatinine μmol/L	↔
Lopes et al. 2022	103 (15.9 %) vs. 55 (5.04 %) renal impairment	↑
Luo et al. 2020	4 (5 %) vs. 1 (0.6 %) chronic renal failure	↑
Zhou et al. 2020	95 ± 29.3 vs. 104 ± 28.6 eGFR (ml/min/1.73m^2)	↑
History of stroke/transient ischemic attack		
Lopes et al. 2022†	57 (8.8 %) vs. 54 (4.95 %)	↑
Luo et al. 2020‡	7 (8 %) vs. 3 (2 %)	↑

↑ or ↓ reflect significant ($p < 0.05$) positive or negative differences between those with concurrent HCM+ hypertension compared to those with HCM alone; ↔ reflects a non-significant difference between those with HCM+ hypertension compared to those with HCM alone.
Abbreviations: HCM- hypertrophic cardiomyopathy; HTN – hypertension; SRT-septal reduction therapy; NYHA- New York Heart Association; eGFR – estimated glomerular filtration rate.
*Age at first HCM evaluation.
**Wang et al., Zhou et al., Perez-Sanchez et al. reported likelihood of having NYHA class III/IV, whereas Lopes et al. reported likelihood of having NYHA class >II among patients with HCM + HTN versus those with HCM alone.
***Smith et al. reported stratified VO2 peak (ml/kg/min) categories among those with HCM + HTN and those with HCM alone, VO2 peak ≤14.14 (144 (49 %) versus 150 (51 %)); between 14.15 and 17.70 (139 (47 %) vs.155 (53 %)); between 17.71 and 21.51 (149 (50 %) vs. 146 (50 %)), $p = 0.80$.
§Holter monitor, †Stroke, etiology not reported, ‡Transient ischemic attack.

blood pressure was measured (in office or hospital) [10]. Argulian et al. reported a clinical medication management pathway for adults with co-occurring HCM and hypertension. The treatment strategy constituted of reducing the use of direct vasodilators and dihydropyridine calcium channel blockers and optimizing β_1-selective beta blockers, non-dihydropyridine calcium channel blockers, with use of disopyramide for relief of left ventricular outflow tract obstruction in patients with obstructive HCM. Clonidine, a central nervous system agent, was utilized as needed for blood pressure control in those with uncontrolled hypertension. Over a mean (SD) follow up of 36 (32) months, range (1–192 months), the authors reported improvement in blood pressure control (SBP 137 to 131 mmHg, $p = 0.01$), reduction in left ventricular

Table 3
Multivariate associations* between hypertension and outcomes in adults with HCM and hypertension compared to adults with HCM alone.

Article	Hazard ratio	95%CI	Effect
Functional class (higher NYHA class)			
Lopes et al. 2022	1.41	1.03–1.94	↑
Perez-Sanchez et al. 2018**	10.39	1.71–19.06	↑
Atrial fibrillation			
Perez-Sanchez et al. 2018**	4.87	−3.19 -12.92	↔
Stroke-related death			
Wang et al. 2023	1.55	0.44–5.51	↔
Sudden death			
Perez-Sanchez et al. 2018**	14.3	−7.27-35.85	↔
Wang et al. 2023	0.53	0.15–1.89	↔
Heart-failure related death			
Wang et al. 2023	0.71	0.30–1.67	↔
Composite cardiac outcome***			
Perez-Sanchez et al. 2018	4.18	−2.41-10.77	↔
Wang et al. 2023	0.77	0.35–1.71	↔
Zhang et al. 2023	SRT 0.229	0.033–1.54	↔
	Non-SRT 0.394	0.17–0.92	↓
Sheikh et al. 2016	2.02	1.05–3.88	↑
Moon et al. 2011	2.25	1.435–3.514	↑
All-cause mortality			
Zhang et al. 2023	SRT 0.314	0.077–1.29	↔
	Non-SRT 0.29	0.1–0.9	↓
Geske et al. 2017	0.92	0.83–1.02	↔

↑ or ↓ reflect significant ($p < 0.05$) positive or negative differences between those with concurrent HCM+ hypertension compared to those with HCM alone; ↔ reflects a non-significant difference between those with HCM+ hypertension compared to those with HCM alone.
Abbreviations: HCM – hypertrophic cardiomyopathy; HTN – hypertension; SRT – septal reduction therapy; NYHA – New York Heart Association.
*Covariates for multi-variate analysis: Lopes et al.: age, sex, maximal left ventricular wall thickness, atrial fibrillation, left atrial diameter, proband vs. relative status, genotype positive vs. negative; Perez-Sanchez et al.: age, sex, physical activity, indexed maximal wall thickness [mm/m^2], left atrial diameter [mm], presence of left ventricular outflow tract obstruction [>30 mmHg] and type of gene; Wang et al.: sex, age, family history of HCM, chest pain, pre/syncope, SBP, DBP, prior TE, vascular disease, diabetes, atrial fibrillation, ACEI, ARB, dihydropyridines, hydrochlorothiazide, aspirin, statins, intervention of obstruction, devices, creatinine, uric acid, HDL-C, left ventricular end diastolic volume, interventricular septum (mm), left ventricular posterior wall (mm), maximal left ventricular wall thickness, resting left ventricular outflow tract obstruction ≥30 mmHg, LV apical aneurysm and SAM; Zhang et al.: age, gender, BMI, atrial fibrillation, syncope, non-sustained ventricular tachycardia, family history, duration of HCM, maximal left ventricular thickness, left ventricular end-diastolic diameter, left ventricular ejection fraction, left atrial diameter, left ventricular outflow tract obstruction, genetic characteristics were well balanced by inverse probability of treatment weighting; Sheikh et al.: age, Black ethnicity, syncope, left atrial size, left ventricular outflow tract obstruction, history of non-sustained ventricular tachycardia; Moon et al.: age, gender, diabetes, mixed-type apical HCM, left ventricular ejection fraction, left atrial volume index, early mitral inflow velocity, mitral annular early diastolic velocity, mitral annular systolic velocity, early mitral inflow/mitral annual early diastolic ratio, right ventricular systolic pressure; Geske et al.: age, female sex, NYHA Class III – IV, atrial fibrillation, coronary artery disease, implantable cardiac defibrillator implantation, beta blocker use
**Perez-Sanchez et al. used Laplace estimates to analyze associations between hypertension and outcome.
***Definitions of composite cardiac outcome: Perez-Sanchez et al. 2018: atrial fibrillation, stroke, NYHA Class III-IV, sudden death; Wang et al. 2023, Zhang et al. 2023: heart failure-related death, stroke-related death, sudden cardiac death; Sheikh et al. 2016: end-stage heart failure, stroke, sudden cardiac death, non-HCM related cardiovascular deaths, survived cardiac arrest, implantable cardiac defibrillator therapy; Moon et al. 2011: death secondary heart failure, stroke, myocardial infarction, or intractable arrhythmia; heart failure requiring unplanned hospitalization, stroke.

outflow gradient at rest 48 to 14 mmHg, $p < 0.01$ (some required invasive treatment for obstruction reduction), and functional status (New York Heart Association [NYHA] class 2.4 to 1.8, $p < 0.01$) among obstructed HCM patients. Among patients with HCM without LVOT obstruction, there was no significant improvement in SBP (130 to 124 mmHg, $p = 0.26$); however functional status did improve (NYHA class 1.8 to 1.5, $p = 0.03$) [16].

3.3. Activity limitations in adults with HCM and hypertension

Functional (NYHA) class was neutral to higher in those with HCM and hypertension compared to those with HCM alone [22,32,33,35]. One study identified a significant univariate association between hypertension and low peak VO2 max in patients with HCM, an objective measure of exercise capacity. However, this association lost statistical significance in multivariate analysis [29].

3.4. Outcomes

Follow-up varied across studies, from 3.5 to 10.9 years [20,25,30]. Lou et al. (2020) examined cardiovascular-related death among those with HCM and hypertension compared to those with HCM alone and did not identify significant differences, 3 (38 %) compared to 5 (71 %), $p > 0.05$ [28], whereas Moon et al. (2011) found that among adults with apical HCM and adverse cardiovascular events ($N = 110$), defined as unplanned hospitalization due to heart failure, stroke, or cardiovascular mortality, hypertension was more prevalent than in adults with apical HCM and no adverse cardiovascular events ($N = 344$), 76 (69 %) compared to 156 (46 %), $p = 0.0001$ [26]. Luo et al. (2020) and Sanchez-Perez et al. (2018) examined sudden death outcomes and found no difference among those with HCM and hypertension compared to those with HCM alone, though sudden death outcomes were overall low [23,28]. Over half of articles examined the association of hypertension with a variety of outcomes among those with HCM, though few reported on disaggregated outcomes. We present multivariate associations between hypertension and outcomes in **Table 3**. Overall, all-cause mortality was not higher among those with co-occurring HCM and hypertension compared to HCM alone. Results related to composite cardiac outcomes were varied across studies and there was some variability in how different authors defined composite cardiac outcomes (**Table 3**). Etiology of stroke (ischemic or embolic) was generally not reported. We also did not identify any articles that examined common hypertension-related outcomes, like progression of renal disease.

4. Discussion

In this scoping review of the literature, we identified several notable findings and knowledge gaps related to the clinical profile and clinical course of individuals with co-occurring HCM and hypertension. Adults with HCM and hypertension were older and had more co-morbidities like obesity, diabetes, hyperlipidemia, and coronary artery disease compared to adults with HCM alone. It is well documented that hypertension prevalence increases with greater age [5]. However, in the setting of HCM, additional issues should be considered, including that hypertension may be a diagnostic confounder for HCM leading to delays in diagnosis. The diagnostic criterion for HCM is unexplained left ventricular hypertrophy (LVH) ≥15 mm in those without family history of the condition and ≥13 mm in those with family history [4]. Hypertension is the most common cause of LVH [32]. Thus, an overlap may occur in adults with both conditions. While asymmetric hypertrophy is often seen in HCM, concentric LVH, a hallmark of hypertension, can also be observed in HCM. Suboptimal imaging may often preclude accurate quantification of LV magnitude and pattern, thus making a diagnosis

challenging. Cardiac magnetic resonance imaging techniques are increasingly used to differentiate between HCM and hypertension, but such imaging may not be widely available in community settings [33,34]. Patients may see a variety of providers and be diagnosed with other conditions, before reaching an HCM specialist, and the age at which individuals present to HCM care has increased over time [35,36]. Five of the 11 articles in this review did not explicitly define hypertension criteria within their cohorts, and none reported data relating to the duration of hypertension as an HCM co-morbidity. Thus, future research efforts should focus on elucidating the role of hypertension as a diagnostic confounder of HCM. Furthermore, implementation strategies are needed to improve HCM recognition and distinguish between HCM and hypertension in community settings that may not have the resources to regularly utilize cardiac magnetic resonance. Recent advances in artificial intelligence and machine learning in detecting HCM via electrocardiogram or echocardiogram, both more accessible than CMR, have shown promise in accurately detecting HCM [37–39].

Sheikh et al. (2016) noted that hypertension prevalence was higher among Black adults with HCM who also experience diagnostic disparities and delays in HCM care, compared to White adults [10,40]. Differences in the prevalence of hypertension among Black and White adults with HCM have been equivocal in the literature but findings are limited as Black adults are underrepresented in HCM clinical care and research overall [11,41,42]. Even in our review, none of the United States-based articles reported race other than White, if race was reported at all. Black adults have a higher prevalence of hypertension overall in the United States and have more adverse cardiovascular outcomes related to hypertension compared to all other groups [43]. Thus, better understanding of co-occurring HCM and hypertension in this population is of high importance, given that dihydropyridine calcium channel blockers (CCBs) and diuretics are recommended as first-line anti-hypertensive therapy and are also effective at reducing cardiovascular complications in Black adults [7]. However, these medications may worsen LVOT obstruction in HCM, leading to increased symptom burden and a need for invasive obstruction-relieving procedures, which carry their own inherent risks [44].

Medical regimen among those with HCM and hypertension compared to HCM alone is overall underexplored, including in the sparsity of primary literature informing HCM clinical guideline recommendations. Medical regimen was addressed in a minority of the articles we reviewed. While direct vasodilators, including angiotensin converting enzyme inhibitors and angiotensin receptor blockers, were more common among individuals with both conditions, only 1 article reported a medication treatment strategy in patients with obstructive and non-obstructive HCM, finding that reduction of vasodilators and prioritization of β_1-selective beta blockers, non-dihydropiridine CCB (verapamil) and use of clonidine, contributed to improvement in functional class in both HCM-types and a reduction in both obstruction and systolic blood pressure among those with obstructive HCM [18]. The study suggested that therapy aligned with HCM goals (e.g. reduction of obstruction) may also be beneficial for blood pressure control in patients with obstructive HCM. However, this strategy did not improve blood pressure in those with non-obstructive HCM and the overall sample size was relatively small, $N = 114$. Future studies should test these strategies in larger sample sizes with diverse patient populations.

Recent advances in obstructive HCM treatment, specifically cardiac myosin inhibitors have shown positive results in reducing LVOT obstruction [45]. Preliminary secondary analyses of trial data have not shown differences in the effect of these therapies related to quality of life or health status in patients with obstructive HCM and hypertension compared to those with obstructive HCM alone [17]. Future analyses may focus on how these therapies may impact patients with non-obstructive HCM and hypertension, as ongoing trials are currently testing the effect of these medications on adults with non-obstructive HCM [46].

In our review, cross-sectional analyses varied in whether those with HCM and hypertension had worse functional class (NYHA class) and exercise capacity than those with HCM alone, while 2 studies identified hypertension as an independent predictor for higher NYHA class [20,25,27,29,31]. We did not identify any articles that reported symptoms or quality of life measures among adults with HCM and hypertension. The lack of patient-reported data is a limitation as NYHA class is a subjective determination by the clinical provider and studies have shown discordance between provider-assigned NYHA class and patient-reported health status [47–49]. Furthermore, evaluation of patient-reported outcomes is critical to understanding the everyday experiences of patients, which has wider implications related to disease self-management and overall self-care.

A notable finding in our review was that even though adults with HCM and hypertension had a less optimal clinical profile than adults with HCM alone, older and with more cardiovascular co-morbidities, outcomes, including all-cause mortality and cardiovascular outcomes, disaggregated and in composite, were largely similar between groups. There was an overall preponderance of no significant associations between hypertension and outcomes in adjusted analyses. This discordance may be explained in several ways. It is possible that once patients get to specialty HCM care, treatment care and follow-up may be more individualized and thorough with providers more readily responding to changes in health status [50]. It is also possible that there are unique mechanistic factors underlying hypertension among those with HCM, including higher cardiac contractility versus peripheral vascular resistance. Future work should focus on examining the mechanistic underpinnings of hypertension in HCM, which will help to identify suitable blood pressure control targets overall and that consider the dynamic phenomenon of LVOT and mid-ventricular obstruction. Another opportunity for future research investigation is the association of hypertension with surrogate markers, including Brain Natriuretic Peptide (BNP), among adults with HCM, which could help tailor treatment more precisely.

Another gap in the existing literature related to outcomes among those with HCM and hypertension is that common hypertension-related outcomes are underexplored. For example, only 2 articles discussed blood pressure control, though one did not discuss a hypertension-specific regimen [10,18]. This is important to understand in order to inform treatment strategies. Additionally, articles that reported strokes as outcomes did not specify their etiology. Adults with HCM are overall at high risk for developing atrial fibrillation, and embolic stroke, if atrial fibrillation is left untreated [51]. This is in contrast with ischemic or hemorrhagic strokes, which are more commonly associated as hypertension sequelae [52]. While some articles reported variable cross-sectional results related to renal dysfunction among those with HCM and hypertension compared to those with HCM alone, none reported renal-related outcomes. Both distinction in stroke etiology and renal function outcomes are critical to elucidate in order to inform prevention and treatment priorities related to blood pressure for those with HCM.

4.1. Limitations

The review had several potential limitations. First, scoping reviews are geared towards synthesizing the available literature rather than appraising it. Thus, we did not appraise the quality of the literature. As a result, we may have included studies with methodologic flaws, though we noted heterogeneity in statistical methods and outcome definition in the tables. Second, we focused our review on full-length peer-reviewed articles, though we included relevant conference abstracts in our discussion section. As all studies we identified were observational with most retrospective, results should be interpreted in the context of the level of evidence they represent.

5. Conclusions

We methodically examined the clinical course and outcomes in

adults with HCM and hypertension, which is understudied but has high clinical implications. We used a robust operational framework to conduct this scoping review and followed PRISMA-ScR guidelines. We found that even though adults with co-occurring HCM and hypertension were more likely to have an adverse clinical profile, including older age and more cardiometabolic comorbidities, hypertension was not associated with adverse outcomes. However, in examining the available literature, we identified multiple knowledge gaps where future research may focus in order to ultimately optimize patient well-being and outcomes.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ahjo.2024.100367.

Ethical statement

This scoping review did not meet the criteria for Human Subject Research as we analyzed publicly available published work that did not contain identifiable information. Thus, the review did not undergo institutional ethical approval.

Contributors

All authors have read and approved the manuscript. All authors contributed significantly to the final manuscript.

Funding

The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

CRediT authorship contribution statement

Milla Arabadjian: Writing – review & editing, Writing – original draft, Validation, Supervision, Project administration, Methodology, Investigation, Conceptualization. **Sophie Montgomery:** Writing – review & editing, Visualization, Formal analysis. **Mitchell Pleasure:** Writing – review & editing, Visualization, Formal analysis. **Barnaby Nicolas:** Writing – review & editing, Validation, Methodology. **Maxine Collins:** Writing – review & editing. **Maria Reuter:** Writing – review & editing, Formal analysis. **Daniele Massera:** Writing – review & editing, Methodology. **Daichi Shimbo:** Writing – review & editing. **Mark V. Sherrid:** Writing – review & editing, Methodology.

Declaration of competing interest

MA reports advisory board fees from Bristol-Meyers Squibb; DM reports advisory board/consulting fees from Bristol-Meyers Squibb; advisory board/consulting/speaker fees from Sanofi, advisory board/consulting fees from Tenaya Therapeutics; MVS reports consulting fees from Pfizer, Inc. The rest of the authors report no competing interests.

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