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Review Article

Hypertension, Left Ventricular Hypertrophy, and Heart Failure

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Abstract

Left ventricular hypertrophy (LVH) is a manifestation of arterial hypertension and is an independent risk factor for cardiovascular disease morbidity and mortality. Both concentric and eccentric LVH independently increase risk of sudden cardiac death, coronary artery disease, arrhythmias, as well as congestive heart failure (CHF). Hypertension precedes the diagnosis of heart failure in the majority of patients with newly diagnosed CHF and remains the most important cause of diastolic heart failure. Treatment aimed at reducing left ventricular (LV) mass improves outcomes in such patients. Treatment with angiotensin converting enzyme inhibitors, angiotensin receptor blockers, angiotensin receptor neprilysin inhibior, and aldosterone receptor blockers have been shown to significantly decrease LV mass. SGLT2 Inhibitors are emerging as a new class of medications that have been shown to improve cardiac outcomes likely through their effects on LV remodeling and diastolic function. In this review article we will focus on LVH and cardiovascular outcomes.

Keywords: Hypertension, Left Ventricular Hypertrophy, Heart Failure Preserved ejection fraction, Sodium Glucose Co-Transporter 2 Inhibitors

Introduction

Left ventricular hypertrophy (LVH) is an independent risk factor for cardiovascular disease morbidity and mortality.[1] Both concentric and eccentric LVH independently increase risk of sudden cardiac death (SCD),[2] coronary artery disease (CAD), arrhythmias,[3] as well as heart failure.[4] Recent evidence shows up to 30% increased risk of cardiovascular events in patients with hypertensive target organ damage (TOD).[5] Hypertension-induced TOD encompasses both microvascular injury, namely, nephropathy or retinopathy, and macrovascular injury, namely, coronary or peripheral artery disease causing myocardial infarction or stroke. LVH is another manifestation of TOD from uncontrolled blood pressure and will remain the focus of this article.

Arterial hypertension leads to organ damage on much of the body, with LVH a well-known complication. In fact, LVH is the cardinal manifestation to increased hemodynamic afterload in hypertension.[6] Remodeling of the left ventricle (LV) occurs in response to chronic hemodynamic overload from hypertension. Recently, published data from over 6000 Framingham study patients showed that a one standard deviation increase in mean arterial pressure and central pulse pressure was associated with 37%–45% increased incidence of LVH.[5] Cuspidi *et al.* showed that up to 41% of hypertensive patients and 77% with hypertension and a prior history of diabetes mellitus or cardiovascular disease exhibit echocardiographic evidence of LVH.[7] Insulin resistance is in itself a mediator of LVH, with up to 70% of type two diabetics exhibiting increased LV mass.[8,9]

Increased aortic pulsatile load ultimately leads to increased LV wall stress and LVH.[10] Indeed, the maladaptive mechanisms involved in the developed of LVH are the first step toward overt cardiovascular disease such as congestive heart failure (CHF), ischemic heart disease, arrhythmias, and stroke.[2,11] LVH increases myocardial oxygen consumption while reducing coronary blood flow – predisposing the patient to angina pectoris, myocardial infarction, and sudden cardiac death. Untreated hypertension along with LVH leads to impaired diastolic filling due to LV stiffness.[10] Hypertension precedes the diagnosis of heart failure in up to 91% of patients with newly diagnosed CHF and remains the most important cause of diastolic heart failure.[12,13]

It has been shown that long-standing and untreated hypertension leads to heart failure. Pressure overload caused

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by untreated hypertension leads to cardiac remodeling, which can manifest as LV diastolic dysfunction and concentric LVH. With sustained pressure overload, there is progressive diastolic dysfunction and heart failure with preserved ejection fraction (HFpEF) ensues. End-stage hypertensive disease presents with both pressure and volume overload and there is eccentric remodeling that leads to dilated cardiomyopathy and heart failure with reduced ejection fraction (HFrEF).[14]

Regression of LVH and Outcomes

Regression of LVH has been shown to be an effective way to reduce cardiovascular events.[15] This finding has held true independent of blood pressure lowering.[15] Investigators in the HOPE trial showed that reduction of LVH with the ramparil versus placebo significantly lowered the incidence of cardiovascular death and myocardial infarction.[16] Investigators also found that hospitalizations for CHF and unstable angina as well as the risk for the development of CHF were significantly lowered.[16] Investigators in the LIFE trial showed a greater reduction of LVH with losartan at 1 year compared to patients treated with beta-blockers.[17] Reduction in LVH corresponded with a reduction in cardiovascular morbidity, mortality, new-onset atrial fibrillation, and sudden cardiac death.[18-20] More recently, it was shown that sacubitril/valsartan was superior to olmesartan in reduction of LV mass and mass index.[21] Consistent throughout these studies, it has been shown that a reduction in the left ventricular mass reduces cardiac events independent of differences in blood pressure lowering.[16,21] This lends credence to the idea that cardiac myocytes may be affected directly by an inhibitor of the renin angiotensin aldosterone system (RAAS).[22-24]

Despite the positive outcomes in trials showing that reduction in LVH leads to better outcomes, no therapy has been shown to improve mortality in HFpEF, the most common outcome in patients with hypertension and LVH.[25,26] The I-PRESERVE trial and CHARM-PRESERVED failed to show a reduction in death or hospitalization from cardiovascular causes with the use of irbesartan and candesartan, respectively.[25,27] In the I-PRESERVE trial, 30% of patients randomized to the irbesartan arm had LVH at baseline,[25] compared to ~50% of patients in the CHARM-PRESERVED trial.[27,28] Lund *et al.* failed to show a reduction in combined all-cause mortality and CHF hospitalization in patients treated with beta-blockers, of which 43% had cardiomegaly.[26] The TOPCAT trial failed to show that spironolactone significantly reduced death from cardiovascular causes or hospitalization for CHF in patients with HFpEF,[29] of which ~50% had LVH.[28,29]

Interestingly, new data are now available looking at outcomes for HFpEF based on phenotype.[30] From a pathophysiologic standpoint, HFpEF is likely a far more diverse syndrome than HFrEF.[31] Despite this broad heterogeneity, Shah *et al.* were able to show three specific, mutually exclusive, phenotypes of HFpEF.[32] Individuals in phenotype 1 were younger with moderate diastolic dysfunction and relatively normal brain natriuretic peptide; phenotype 2 were obese, diabetic patients with high prevalence of obstructive sleep apnea and worse LV relaxation; and phenotype 3 were older patients with chronic kidney disease and significant LV remodeling.[32] Overall, individuals in phenotype 2 and 3 were significantly more likely to have diastolic dysfunction, elevated mean left atrial pressures and LVH than those in phenotype 1.[32] Outcomes in the trial were striking – with individuals in phenotypes 2 and 3 being 4–7 times more likely to have heart failure hospitalizations or death.[32]

Certainly, there is some amount of heterogeneity behind that pathophysiology of HFrEF as well. Yet, patients have proven to respond to a "one size fits all" approach. For HFpEF, it may well be necessary to separate patients into clinically relevant phenotypes to tailor a beneficial treatment. In fact, recently published data Cohen *et al.* show that patients in the TOPCAT study with more concentric LVH were more likely to respond to spironolactone and had an improved prognosis.[30]

Effect of insulin resistance and SGLT2 inhibitors on LVH

In addition to traditional therapy involved angiotensin converting enzyme inhibitors (ACE-inhibitor), angiotensin receptor blockers (ARB), beta-blockers, and aldosterone receptor antagonists, a promising development in recent years has been the substantial evidence that sodium glucose transporter 2 inhibitors (SGLT2) inhibitors reduce LV mass and improve LV diastolic function and overall lower cardiovascular morbidity and mortality and hospitalizations for heart failure.[33-37] Data from the EMPA-REG Outcome Trial showed a significant reduction in LV mass and improvement in diastolic function at 6 months in empagliflozin versus placebo in patients with type 2 DM and coronary artery disease.[35,38] The authors also found that change in 24-hour ambulatory blood pressure did not associate with changes in LV mass, thus similar to inhibition of RAAS, suggesting that there are mechanisms other than blood pressure control to explain reduction in LV mass.[35] Pabel *et al.* showed that empagliflozin causes direct pleotropic effects on myocardium by improving LV stiffness, which, in turn, improves the diastolic LV function.[39] These effects were observed independent of diabetic conditions.[39] Early data from the ongoing DAPA-LVH trial show that dapagliflozin also has a significant reduction of the left ventricular mass.[33] Matsutani *et al.* showed that canagliflozin reduces LV mass and improves LV diastolic function.[40]

Several reasons have been postulated for why SGLT2 inhibitors have had such an impact on cardiac outcomes, including heart failure. Potential mechanisms include significant lowering of systolic and diastolic blood pressure[41] as well as changes to vascular endothelial function, arterial stiffness, vascular resistance, and myocardial fibrosis.[42,43] Through natriuresis, SGLT2 inhibitors can reduce intravascular volume and total body sodium, leading to a reduction in preload and afterload, alleviating cardiac work, and improving LV

function.[44,45] Furthermore, despite its use as a diabetes medication, based on the available evidence, it is less likely that SGLT2 inhibitor's reduction in CVD and HF is mediated by their hemoglobin A1c-lowering effects.[46] A much more likely explanation is that SGLT2 protective effects on LV mass and LV diastolic function as well as possible direct protective effects on cardiomyocytes and anti-inflammatory properties explain their significant cardiac benefits.[35,40,46]

While insulin resistance in itself is a risk factor for LVH,[9] there are significant data indicating that those with LVH and diastolic dysfunction due to hypertension could also benefit from SGLT2 inhibitors. Already there is evidence to suggest that patients without type 2 diabetes mellitus will derive a similar benefit from SGLT2 inhibitors when compared to people with diabetes. Dapagliflozin has been shown to reduce the likelihood of death from cardiovascular causes as well as heart failure admissions in patients with HFrEF with and without diabetes.[47] In non-diabetic rat models, empagliflozin has shown to significantly reduce LV end-diastolic pressure, LV mass, and posterior wall thickness.[48] In humans, empagliflozin has shown to improve diastolic function regardless of diabetes status.[39] Ongoing studies include the EMPEROR-PRESERVED trial, looking at effects of empagliflozin in patients with and without T2DM and HFpEF.[49] Primary outcomes in that study include CV mortality as well as heart failure hospitalizations.[49] The EMPA-TROPISM trial is investigating similar outcomes in non-diabetic patients with LV ejection fraction ≤50%.[50] The ongoing DELIVER trial (NCT03619213) is evaluating Dapagliflozin versus placebo in reducing CV mortality and HF events in patients with HFpEF and evidence of cardiac structural abnormalities (LVH or left atrial enlargement on echocardiogram) with and without diabetes mellitus.

Conclusion

Left ventricular hypertrophy is a cardinal manifestation of arterial hypertension and significantly increases the risk of heart failure, ischemia, arrhythmia, and cardiac death. It is clear that regression in the left ventricular mass in those with hypertensive heart disease significantly improves outcomes. The recent trend in evidence-based medicine raises the question – do patients with predominately LVH and diastolic dysfunction phenotype of HFpEF derive a significant mortality benefit from a treatment approach that leads to regression of LVH? Indeed, a drugs effect on LV remodeling is the best surrogate to predicate outcomes such as survival and hospitalizations.[51]

Treatment with ACE inhibitors, ARBs, sacubitril/valsartan, and spironolactone has been shown to significantly decrease LV mass. Interestingly, these effects were independent of blood pressure control and they point toward alternative mechanism of improvement in LV remodeling. SGLT2 inhibitors are emerging as a new class of medications that have been shown to improve cardiac outcomes – in both diabetic and non-diabetic patients – likely through their effects on LV remodeling and diastolic function.

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