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Impact of Echocardiographic Left Ventricular Geometry on Clinical Prognosis

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ABSTRACT

Abnormal left ventricular (LV) geometry, including LV hypertrophy (LVH), is associated with increased risk of major cardiovascular (CV) events and all-cause mortality and may be an independent predictor of morbid CV events. Patients with LVH have increased risk of congestive heart failure, coronary heart disease, sudden cardiac death and stroke. We review the risk factors for LVH and its consequences, as well as the risk imposed by concentric remodeling (CR). We also examine evidence supporting the benefits of LVH regression, as well as evidence regarding the risk of CR progressing to LVH, as opposed to normalization of CR. We also briefly review the association of abnormal LV geometry with left atrial enlargement and the combined effects of these structural cardiac abnormalities.

Abnormalities in left ventricular (LV) geometry, including LV hypertrophy (LVH), are targeted responses to chronic arterial hypertension (HTN) and other cardiovascular (CV) disorders; and LVH is an independent risk factor for coronary heart disease (CHD), heart failure (HF), arrhythmias, including sudden cardiac death (SCD), stroke, and other major CV morbidity and mortality.[1] According to Laplace's law, in the short-term, increases in LV mass (LVM) may be beneficial by allowing for a reduction in LV wall stress and hemodynamic compromise. On the other hand, in the long-term, LV geometric abnormalities, particularly LVH, can deteriorate to maladaptive processes and increase the risk of HF and other CV disorders.[2] Clearly, numerous epidemiological studies have demonstrated that LVH is not benign but is associated with very high prevalence of morbid CV events, and in most instances, LVH seems to be a more potent risk factor than other conventional CV risk factors for predicting major CV morbidity and mortality.[1,3–6]

In this review, we discuss the various types of LV geometric abnormalities, including concentric remodeling (CR) and concentric and eccentric LVH (cLVH and eLVH, respectively). We also review the risk factors for LVH, its consequences, and implications for regression. Additionally, we review the more subtle LV geometric abnormality of CR and its predictive value for CV events, as well as implications for preventing the conversion of CR to frank LVH. Finally, we discuss the impact of obesity on LV geometric abnormalities and the association of LV geometric abnormalities with other structural abnormalities, particularly left atrial enlargement (LAE).

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Abbreviations and Acronyms

AF = atrial fibrillation

ASE = American Society of Echocardiography

BP = blood pressure

BSA = body surface area

CFR = coronary flow reserve

CHD = coronary heart disease

CR = concentric remodeling

CV = cardiovascular

DD = diastolic dysfunction

ECG = electrocardiographic

HF = heart failure

HTN = hypertension

LAE = left atrial enlargement

LAVI = left atrial volume index

LV = left ventricular

LVEF = left ventricular ejection fraction

LVH = left ventricular hypertrophy

LVM = left ventricular mass

LVMI = left ventricular mass index

RWT = relative wall thickness

SCD = sudden cardiac death

VEA = ventricular ectopic activity

Risk factors

The major risk factors for LVH are listed in Table 1. Epidemiologic studies have identified age, elevated blood pressure (BP) and weight as independent risk factors for the development of LVH.[1,7] Obviously, BP and weight typically increase with age, not surprisingly markedly increasing the prevalence of LVH in the elderly. LVH is also related with other conditions including diabetes,[8] hyercholesterolemia,[9] myocardial infarction,[10] valvular stenotic and regurgitant lesions,[11] as well as African American race.[12] Additionally, high sodium intake, independent of level of BP, also increases the prevalence of LVH,[1,7] as do other factors listed in Table 1.

Table 1 – Risk factors for LVH.

Major
- Age
- Arterial pressure (including ambulatory, work, and exercise)
- Weight

Other
- Race (higher in African Americans)
- Renin–angiotensin–aldosterone system
- Increased sodium intake
- Diabetes
- Aortic stenosis and regurgitant valvular heart disease
- Catecholamines
- Job strain, including physical and emotional
- Various growth factors

Consequences of LVH

As discussed earlier, although initially LVH may be compensatory and benign, for decades it has been known to be associated with numerous adverse CV consequences (Table 2 and Fig 1). More than 3 decades ago, data from the Framingham Heart Study demonstrated that definite electrocardiographic (ECG) evidence of LVH, both by voltage and with repolarization abnormalities or "strain" pattern on the EGC, was associated with 6-fold and 8-fold increases in CHD and CV mortality, respectively.[10] Clearly, as LVH as assessed by LVM index (LVMI) increases, so does the risk of major CV events.

Coronary flow reserve (CFR)

The ability of the coronary arteries to increase their blood flow under stress is referred to as CFR, which is markedly reduced in patients with LVH due to HTN or aortic stenosis.[1,13]

LV function

Certainly, LVH is associated with development of HF with either preserved or reduced levels of systolic function.[2] Clearly, ECG-LVH with "strain" pattern predicts both new onset HF and increased mortality, with LVH predicting HF events more so than CHD events.[10]

Although the exact mechanism by which LVH impacts LV function is not totally known, LVH and myocardial fibrosis may adversely affect diastolic dysfunction (DD). Although LVM and relative wall thickness (RWT) may both increase DD in HTN, studies have also demonstrated that combined HTN and obesity (discussed below) have significant adverse impact on LV diastolic filling. [1,7] Although LVH is associated more strongly with DD than with systolic dysfunction,[1,7,13] we recently demonstrated that 13% of patients with preserved LV systolic function but with cLVH progressed to LV systolic dysfunction during 3-year follow-up, indicating that LVH impacts diastolic and systolic HF.[2]

Ventricular ectopic activity (VEA)

Messerli and colleagues[14] demonstrated three decades ago that ECG-LVH was associated with the high prevalence of premature and complex VEA, which may partly explain the known association of LVH, especially ECG-LVH, with increased risk of SCD. Several echocardiographic studies have demonstrated increased risk of complex VEA and risk of SCD,[5,15] whereas regression of LVH may be associated with reduced risk of SCD.[16]

Additionally, LVH is a strong risk factor for atrial fibrillation (AF),[17,18] including a 20% increase in the risk of AF for every one standard deviation increase in LVM.[17] Also, ECG-LVH appears to

Table 2 – Consequences of LVH.

- Reduced coronary flow reserve
- Reduced ventricular functional systolic reserve
- Diastolic ventricular dysfunction
- Increased coronary artery disease
- Frequent and complex ventricular arrhythmias
- Increased cardiovascular events
- Increased cardiovascular mortality