Diastolic Dysfunction and Hypertension

Wilson Nadruz, MD, PhD[a,b], Amil M. Shah, MD, MPH[a], Scott D. Solomon, MD[a,*]

KEYWORDS

• Hypertension • Diastolic dysfunction • Heart failure • Left ventricular hypertrophy

KEY POINTS

- Hypertension is the leading etiology for diastolic dysfunction, which is ubiquitous in elderly individuals and contributes to the development of heart failure.
- In addition to hypertension, diabetes, renal function, salt intake, and others contribute to the development and progression of diastolic dysfunction in hypertensive individuals.
- Lowering blood pressure improves diastolic dysfunction, but it is uncertain whether this improvement is translated into better cardiovascular outcome in hypertensive patients.

INTRODUCTION

Left ventricular (LV) diastolic dysfunction (LVDD) is characterized by alterations in LV diastolic filling, which may include impairments in myocardial relaxation and abnormal distensibility of the myocardium.[1,2] It is commonly seen in community settings, especially among elderly individuals, and is a strong predictor of cardiovascular events and incident heart failure (HF).[2] Several risk factors, including hypertension, coronary artery disease, obesity, and diabetes mellitus, are implicated in the development of LVDD.[2] Hypertension has been reported as the most important risk factor for LVDD in the community and a major contributor to the development of HF.[3,4] Importantly, LVDD is considered a critical link between hypertension and HF, particularly in individuals with HF and preserved ejection fraction (HFpEF),[1] which is quite prevalent, accounting for up to one-half of patients with HF, and is associated with substantial morbidity and mortality.[5] The prevalence of HFpEF has progressively increased over the last decades, but death rates have not changed substantially.[6] Even though various therapies improve survival in patients with HF and a reduced ejection fraction, no pharmacologic therapy has been shown to effectively reduce mortality in HFpEF patients.[5] These trends highlight the importance of understanding the pathophysiologic alterations that precede the development HFpEF, particularly hypertension-induced LVDD.

Conflicts of Interest: None.

[a] Cardiovascular Division, Brigham and Women's Hospital, 75 Francis Street, Boston, MA 02115, USA; [b] Department of Internal Medicine, University of Campinas, Campinas, Brazil
* Corresponding author.
E-mail address: ssolomon@bwh.harvard.edu

Med Clin N Am 101 (2017) 7–17
http://dx.doi.org/10.1016/j.mcna.2016.08.013 **medical.theclinics.com**
0025-7125/17/© 2016 Elsevier Inc. All rights reserved.

MECHANISMS OF LEFT VENTRICULAR DIASTOLIC DYSFUNCTION IN HYPERTENSION

Diastole includes isovolumic relaxation and the 3 filling phases (rapid filling, diastasis, and atrial contraction) of the cardiac cycle. Diastolic dysfunction refers to slow or delayed relaxation, abnormal LV diastolic distensibility, and impaired filling of the myocardium.[1,2] In LVDD, the LV cannot fill with enough blood at low pressures and the chamber filling is slow or incomplete in the absence of increases in the left atrial pressure. Consequently, LV filling becomes more dependent on left atrial contraction and higher atrial pressures.[7] Impairment in relaxation may result from any mechanism that influences the removal of calcium from the cytosol and actin–myosin cross-bridge detachment,[8] whereas reduced LV chamber compliance may be related to alterations in myocardial composition, including interstitial fibrosis, alterations in titin phosphorylation, and increases in microtubules content in cardiomyocytes.[2,5]

Hemodynamic and Nonhemodynamic Factors

Hypertension may induce LVDD through several potential mechanisms, including hemodynamic and nonhemodynamic factors, and myocardial ischemia (**Fig. 1**). The strong association between hypertension and LVDD[3,9] supports the notion that pressure overload plays a major role in the development of LVDD. Indeed, casual blood pressure measurements are consistently associated with markers of impaired diastolic function.[3,9] In addition, 24-hour blood pressure measurements, which are more representative of the hemodynamic load imposed by hypertension, show a stronger association with LVDD than casual blood pressure.[10] The use of ambulatory blood pressure monitoring is particularly useful to identify individuals with masked hypertension, among whom the prevalence of LVDD is similar to those with sustained hypertension.[11]

Hypertension induces stiffening of larger arteries and hemodynamic influences derived from these vascular alterations have also been suggested to influence LV diastolic function.[12] Stiffening of the aorta produces an earlier return of wave reflection from the periphery to the proximal aorta with consequent augmentation of aortic systolic pressure, and reductions in diastolic blood pressure. These events result in increases in LV afterload during systole and reductions in LV coronary perfusion during diastole, which may favor the development of LVDD.[13] Several studies have

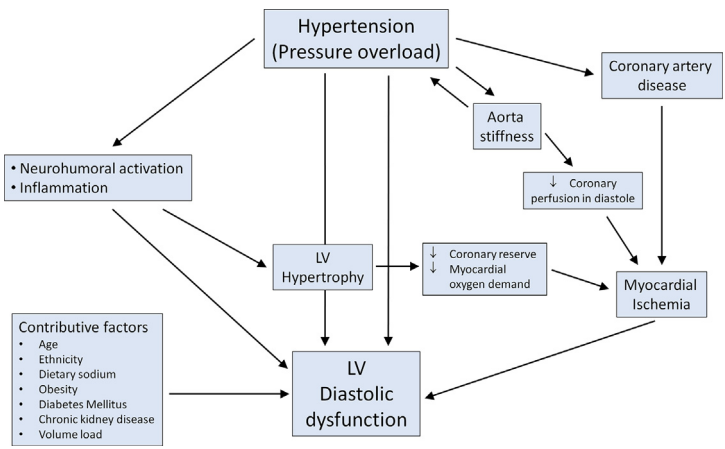

Fig. 1. Pathways of left ventricular (LV) diastolic dysfunction secondary to hypertension.

Download English Version:

https://daneshyari.com/en/article/5680477

Download Persian Version:

https://daneshyari.com/article/5680477

Daneshyari.com