Review Article

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The Role of Echocardiography in Hypertension

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Abstract

Systemic hypertension leads to hypertensive heart disease. Left ventricular hypertrophy (LVH) is a result of chronic systemic pressure overload. Although systolic function is preserved in early stages, it deteriorates gradually. LVH is symmetrical in hypertension. LVH causes different changes in longitudinal, radial and circumferential mechanics in patients with hypertensive patients. Longitudinal strain is significantly decreased, while radial strain is increased.

Left atrial (LA) dilatation is commonly seen in hypertensive patients. Also, LA strain values are lower in hypertensive patients when compared to normal, irrespective to the presence of LA enlargement or LVH. In addition, LVH may cause early sub clinical RV dysfunction as well.

According to the most recent guidelines, it is stated that initiation or monitoring the response to antihypertensive response is based on clinical parameters. However, periodic evaluation of cardiac function and morphology by echocardiography are necessary because of the progressive characteristics of hypertensive cardiomyopathy.

Keywords: Hypertension; Left ventricular hypertrophy

Abbreviations: LVH: Left Ventricular Hypertrophy; LA: Left Atrium

Introduction

Systemic hypertension causes hypertensive heart disease. Chronic systemic pressure loading results in left ventricular hypertrophy (LVH) in order to maintain normal wall stress. In earlier stages of hypertension systolic function is preserved. But, in later stages or with concomitant coronary artery disease (CAD), systolic function is also impaired [1].

The echocardiographic findings related with hypertension are as follows: Left ventricular hypertrophy, diastolic dysfunction, left atrial dilatation, systolic dysfunction, mitral anular calcification, aortic sclerosis, and aortic root dilatation.

Left Ventricular Hypertrophy is evaluated with echocardiography by measuring left ventricular mass (LVM) and left ventricular (LV) geometry. These parameters are measured:

1) Septal thickness/Posterior wall thickness 2) Relative wall thickness

3) LVM/Body surface area (BSA) 4) LV geometry

ASE/EAE guidelines have set cut off limits for LVH as:

LV septal wall thickness ≥ 0.9 cm for women and ≥ 1.0 cm for men.

Mild LVH as: LV septal thickness 1.-1.2 cm/1.1-1.3 cm, moderate LVH as: 1.3-1.5 cm/1.4-1.5 cm, and severe LVH as: ≥ 1.6 cm/ ≥ 1.7 cm (Figures 1 and 2).
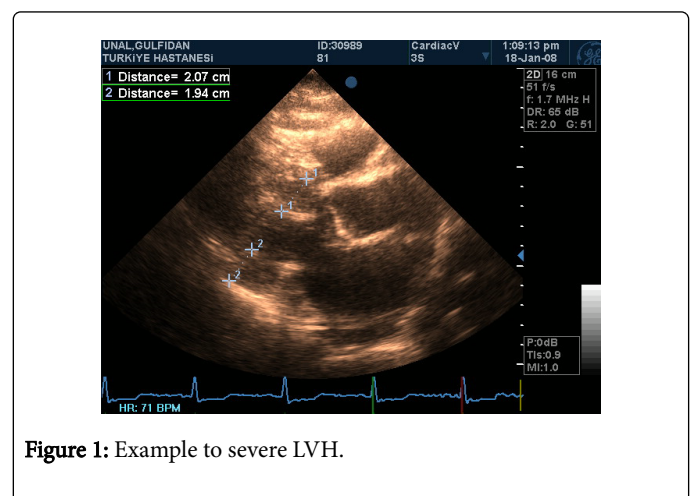

Figure 1: Example to severe LVH.

The formula for relative wall thickness is: (RWT) = (2 × PWTd) / LVIDd

RWT ≥ 0.42 is considered as concentric and RWT <0.42 is considered as eccentric).

LV mass (gram) = 0.8 × 1.04 × ((LVIDd+PWTd+SWTd)^3- LVIDd^3) + 0.6.

The cut of points for LVM/BSA are: 115 g/m^2 for men and 95 g/m^2 for women [1,2].
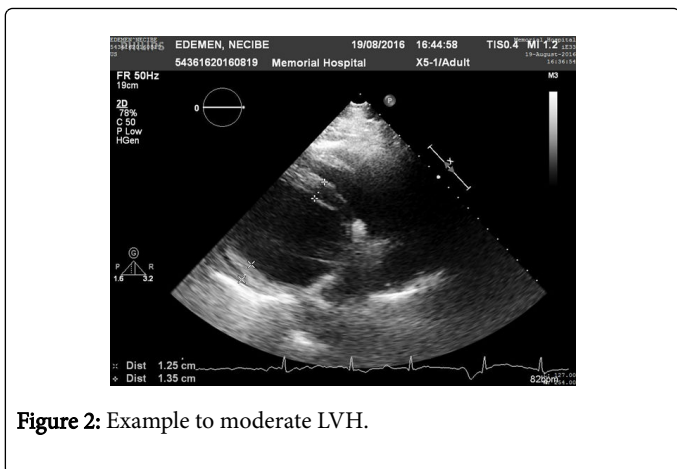
Figure 2: Example to moderate LVH.

Concentric LV hypertrophy is characterized by normal cavity size, symmetrically increased wall thickness and increased LVM. This type of hypertrophy is seen in hypertension. The presence of concentric hypertrophy is associated with increased cardiovascular events.

Long standing hypertension or addition of CAD leads to concentric remodeling: LV shape becomes more rounded and systolic dysfunction occurs. In hypertension, there is pressure overload. Pressure elevation leads to an increase in wall thickness which is known as concentric remodeling.

Eccentric hypertrophy: It is due to volume overload. (e.g. with mitral regurgitation) and it is caused by increased diastolic wall stress. As a consequence, length of the cardiac myocytes is increased. Finally, LV is enlarged (Figure 3) [1,2].

Figure 3: LV Geometric patterns in hypertensive patients according to left ventricular mass and relative wall thickness.

Left ventricular hypertrophy causes different changes in longitudinal, radial and circumferential mechanics in patients with hypertensive patients. Global longitudinal strain is decreased, whereas radial strain is decreased as a compensatory mechanism [3].

Diastolic dysfunction

Most common pattern seen in hypertensive patients with LVH is type I: Early relaxation abnormality. This type of diastolic dysfunction is characterized by using mitral Doppler velocities as low E wave, high A wave, E/A ratio <1 and, increased deceleration time (>200 ms) (Figures 4 and 5).
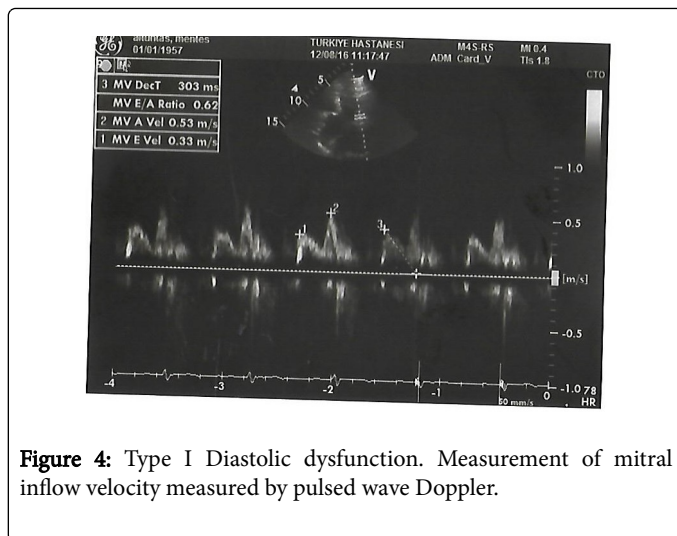

Figure 4: Type I Diastolic dysfunction. Measurement of mitral inflow velocity measured by pulsed wave Doppler.
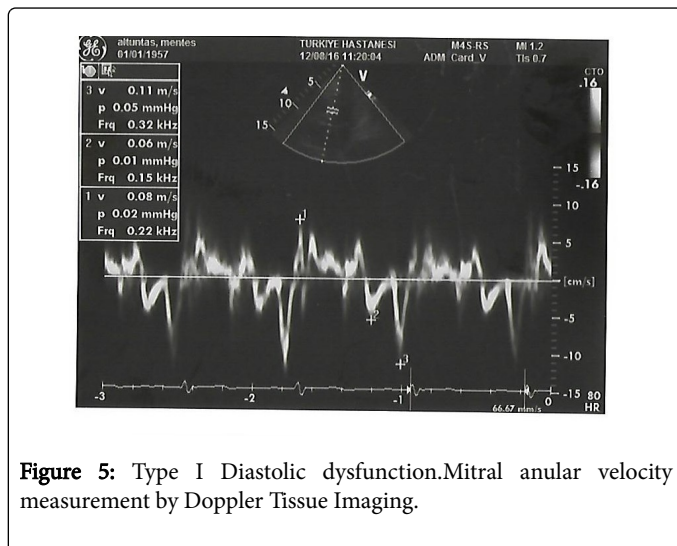

Figure 5: Type I Diastolic dysfunction.Mitral anular velocity measurement by Doppler Tissue Imaging.

As the disease progresses, left ventricular end diastolic pressure (LVEDP) and left atrial pressure increases and pseudonormal pattern is seen where e'/a' ratio is decreased using tissue Doppler imaging (DTİ) (Figures 6 and 7).

Left atrial (LA) size is enlarged in hypertensive patients. Enlarged LA diameter was found >20% of hypertensive patients in a large series [4]. Left atrial dilation is related to diastolic dysfunction. Left atrial size is also a predictor of paroxsismal atrial fibrillation in hypertensive patients [5,6].

LA diameter is measured by M-mode at end ventricular systole when the LA chamber is at its greatest dimension. But enlargement in medial-lateral and superior-inferior diameters alter LA geometry, therefore anteroposterior (AP) dimension may not reflect LA size correctly.

Figure 6: Type II Diastolic Dysfunction. Measurement of mitral inflow velocity by pulsed wave Doppler.
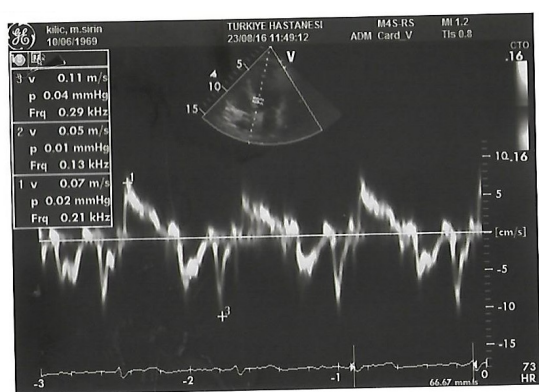

Figure 7: Type II Diastolic Dysfunction. Mitral anular velocity measurement by Doppler Tissue Imaging.

Left atrial area can be planimetred in apical four and two chambered views.

Left atrial volume

Volumetric assessment of LA size is more accurate, and volume indexed to body surface area (LAVi) is even better. According to the ASE guidelines, LAVi $\geq$ 34 ml/m^2 is considered as left atrial enlargement (Figure 8) [7].

Among all these measurements LAVi is most strongly associated with cardiovascular disease and is the most sensitive in predicting cardiovascular outcomes [8]. Currently 2D volume assessment is considered standard for clinical practice.

Measurement of LA volume in 3D may be the preferred method in the future since visualization of LA in 3D shape is more accurate.

LA is not symmetrical, therefore, enlargement may occur non-uniformly, in one direction. Thus, LA size is better evaluated by LA volume. Increased LA size and volume has been associated with poor long term mortality and morbidity [9].
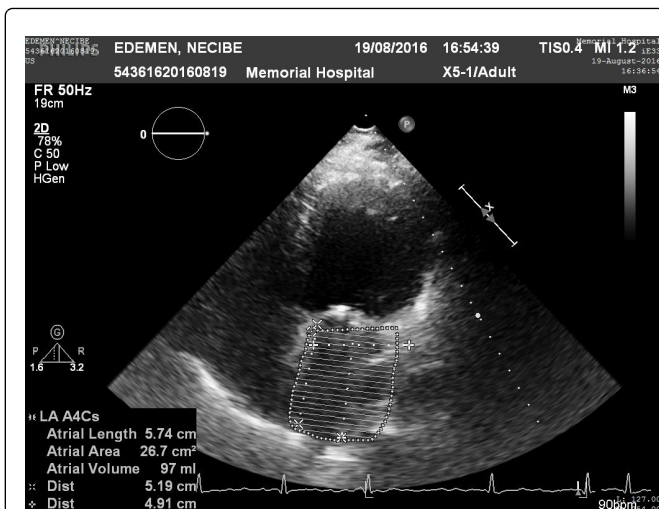

Figure 8: Left Atrial Volume measurement by area length method.

Because of the diastolic dysfunction, reduction in early diastolic emptying is compensated by atrial contraction. Also, elevation of LV filling pressures lead to overfilling of LA. Thus, left atrial enlargement occurs [1,2].

Left atrial function

Recent studies have shown that measurement of left atrial strain (SR) with speckle tracking may be useful in determining left atrial function in hypertensive patients.

According to the studies, it has been shown that LA strain values are lower in hypertensive patients when compared to normal, irrespective to the presence of LA enlargement or LVH. Decrease in SR values are present in all three phases of LA function [10,11].

Reason for LA dysfunction in hypertensive patients is explained by the chronic pressure exposure of the LA, LA pressure rise and reduction of reservoir and conduit functions.

In early hypertensive disease LA stretching causes a temporary enhancement of LA pump function. When compliance is lost and stiffness increases, LA contractility decreases. In later stage, LA mechanics can be depressed in all three phases [12].

Left atrial function assessment may be done using the following methods:

1) Volumetric methods

2) Spectral Doppler

3) Tissue Doppler

4) Deformation analysis (SR imaging).

During the cardiac cycle, LA acts as a reservoir receiving pulmonary venous flow during ventricular systole, as a conduit transferring blood passively during ventricular early diastole, and as an active pump in late diastole.

LA reservoir function corresponds to S' tissue velocity wave and s strain (total),

LA conduit function corresponds to E' tissue velocity wave and e strain (strain positive), and

LA booster pump function corresponds to A' tissue velocity wave and a strain (strain negative) [13].

Systolic dysfunction

In earliest stages of the disease, systolic function is preserved unless coronary artery disease accompanies. Small, hypertrophic left ventricle is typical for this form of the disease. Sometimes small late systolic mid cavity obliteration is seen. The duration of this gradient is smaller than the one seen in hypertrophic cardiomyopathy (HCM).

However, recent studies using 2D speckle tracking strain demonstrate that impaired myocardial systolic deformation occurs in hypertensive patients. Therefore, systolic left ventricular longitudinal strain decreases in the early stages of left ventricular remodeling.

Multidimensional deformation is involved in patients with hypertrophy or those with elevated LV filling pressure. Even though Left ventricular ejection fraction (LVEF) is preserved, left ventricular myocardial deformation starts early. This myocardial deformation has been associated with subendocardial fibrosis [14].

Another issue is the compensatory increase in left ventricular twist in the early phase of systolic dysfunction. LV twists decreases as LV systolic function deteriorates [15]. Mitral annular calcification is frequently seen in chronic hypertensive patients.

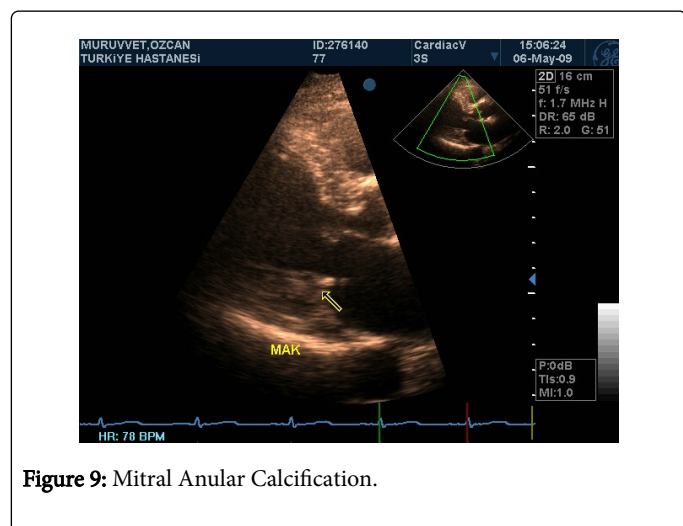It is the reason for mild or moderate mitral regurgitation in these patients (Figures 9 and 10).

Figure 9: Mitral Anular Calcification.

Aortopathy in hypertension

Aortic root dilatation is a common finding for hypertensive patients. Aortic anulus dilatation is seen in progressed disease. According to a meta-analysis, the prevalence of aortic root dilatation in hypertensive patients is about 10%. It may be due to accelerated vascular aging pathway, where increased blood pressure causes fragmentation of elastin [16].

In a recent study, it has been shown that systemic hypertension is positively associated with larger aortic diameter at all considered levels, independent of age, sex and BSA [17].
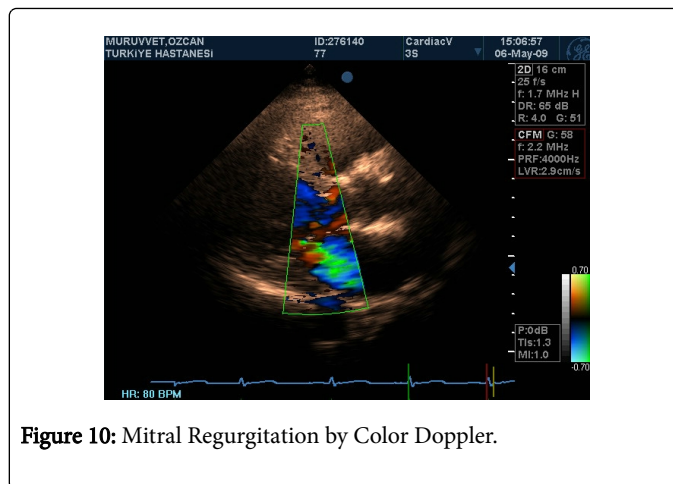

Figure 10: Mitral Regurgitation by Color Doppler.

Right ventricle

Measures of right ventricular deformation are reduced in patients with LVH secondary to hypertension. So, LVH may cause early sub clinical RV dysfunction as well. 2D myocardial deformation is found to be affected by LV geometry in hypertensive patients [18]. In another study, abnormal renin-angiotensin-aldosterone system has been associated with RV dysfunction in systemic hypertension [19].

Dipper and nondipper blood pressure

Non dipper blood pressure pattern is defined as blood pressure decrease in night time is less than10%. Studies have shown that nondipper blood pressure pattern may be associated with increased left ventricular mass, impaired left ventricular and right ventricular function. Therefore, it carries a higher risk for cardiovascular events [20].

Stress echocardiography

LVH is an important reason for false positive results in exercise electrocardiography or SPECT tests. But, it has not been shown to affect the accuracy of stress echocardiography.

In patients with hypertension, a hypertensive response to exercise does not seem to affect the false positive result of exercise echo. Exercise echocardiography might be a good test to perform to detect CAD in hypertensive patients [21].

Conclusion

According to the most recent guidelines, it is stated that initiation or monitoring the response to hypertensive therapy is based on clinical parameters. Therefore, echocardiography is not considered as first line method in all hypertensive patients. However, periodic evaluation of cardiac function or morphology by echocardiography is necessary because of the progressive characteristics of hypertensive cardiomyopathy.

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