Echocardiographic Changes in Systemic Hypertension

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Abstract

Background: Older patients with hypertensive blood pressure have a higher risk of cardiovascular complication when compared to younger hypertensives. Echocardiography is the non invasive procedure of choice in the evaluation of the cardiac effects of systemic hypertension.

Objective: To study the echocardiographic changes resulting from systemic hypertension in age group between 61-70 years.

Methods: This is an observational study which included 70 individuals presented to the Echocardiography Unit in Ain Shams University Hospital between January 2008 & July 2009. Subjects were divided into 2 groups: Group A included 50 adult hypertensive patients aged between 61-70 years not complaining of any other cardiac disease and Group B included 20 healthy non hypertensive individuals of the same age group (control group). Echocardiographic study was done for all selected individuals using 2-D, M-mode and Doppler flow.

Results: There was no significant difference between both groups regarding age, sex and risk factors except for body mass index (BMI) which was significantly higher in hypertensive patients. Duration of hypertension ranged from 4-19 years (10.78±3.67). Hypertensive patients had statistically significant higher IVS thickness (11.41 vs 9.34mm) (p=0.0001), LV posterior wall thickness (11.47 vs 9.29mm) (p=0.0001), left ventricular mass index (LVMI) (114.74 vs 84.95g/m²) (p=0.0001) & larger LA size whether measured in antero-posterior (38.4 vs 35.1mm) (p=0.013), supero-inferior (50.63 vs 46.3mm) (p=0.008) or medio-lateral axis (37.15 vs 34.4mm) (p=0.023). Mitral valve calcification [4 (8%) vs 0 (0%)] (p=0.013), mild MR [5 (10%) vs 0 (0%)] (p=0.022), aortic valve calcification [6 (12%) vs 0 (0%)] (p=0.019), sclerosis [12 (24%) vs 1 (5%)] (p=0.001) & mild AR [2 (4%) vs 0 (0%)] (p=0.012) were statistically more affecting hypertensive patients. Size of ascending aorta was significantly larger in hypertensive patients (30.18 vs 27.74mm) (p=0.009). Also, pulmonary artery pressure was significantly elevated in hypertensive patients when measured by PAT (112.8 vs 121.0 msec) (p=0.041) or mean PAP (28.24 vs 24.55mmHg) (p=0.041). There was a statistically significant correlation between the duration of hypertension and LVMI (r=0.846).

Conclusion: Hypertension is associated with enlargement of left atrium, dilatation of ascending aorta, development of pulmonary hypertension and increase in left ventricular mass which is correlated to the duration of hypertension.

Key Words: Systemic hypertension – Echocardiographic changes.

Introduction

HYPERTENSION, the most treatable form of cardiovascular disease, has been identified as a major risk factor for coronary heart disease [1]. Prevention and treatment of hypertension and target organ disease remain important public health challenges that must be addressed [2].

Older people are more likely than younger people to have hypertension [3]. There was a wide spread belief that a rising blood pressure with age was inevitable and harmless but observational data in several populations have demonstrated that this is not so [4]. Older patients with hypertensive blood pressure have a higher risk of cardiovascular complications when compared to younger hypertensives [5] and are more likely to benefit from treatment to bring blood pressure down [3].

Abbreviations:

AR : Aortic regurgitation.
AS : Aortic stenosis.
BMI : Body mass index.
BP : Blood pressure.
DM : Diabetes mellitus.
DT : Deceleration time.
EF : Ejection fraction.
HTN : Hypertension.
IVS : Interventricular septum.
IVST : Interventricular septal thickness.
LA : Left atrium.
LV : Left ventricle.
LVEDD : Left ventricular end-diastolic diameter.
LVESD : Left ventricular end-systolic diameter.
LVID : Left ventricular internal dimension.
LVMI : Left ventricular mass index.
LVPW : Left ventricular posterior wall.
MR : Mitral regurgitation.
MS : Mitral stenosis.
PAP : Pulmonary artery pressure.
PAT : Pulmonary acceleration time.
PWT : Posterior wall Thickness.
RVSP : Right ventricular systolic pressure.
Echocardiography is the non invasive procedure of choice in the evaluation of the cardiac effects of systemic hypertension, the most common cause of LV hypertrophy and congestive heart failure in adults \[1\]. So, our aim is to study the echocardiographic changes resulting from systemic hypertension in age group between 61-70 years.

**Material and Methods**

This is an observational study in which we enrolled 70 individuals presented to the Echocardiography Unit in Ain Shams University Hospital during the period between January 2008 & July 2009. They were divided into 2 groups: Group A included 50 adult hypertensive patients aged between 61-70 years not complaining of any other cardiac disease and Group B included 20 healthy non hypertensive individuals of the same age group (control group).

Patients were included in group A if they had chronic hypertension under treatment or with systolic B.P $\geq 140$ and/or diastolic B.P $\geq 90$mmHg aged between 61-70 years.

Patients were considered ineligible for the study if they had any other cardiac disease as: Ischaemic heart disease, valvular heart disease, rheumatic heart disease, congenital heart diseases or arrhythmias.

All included subjects were subjected to thorough history taking including duration of hypertension and antihypertensive medications used, general and local cardiac examination. Blood pressure was measured according to the recommended guidelines \[6\].

**Echocardiographic study was done for all selected individuals by use of 2-D, M-mode and Doppler flow to study the following:**

A- Left Ventricle (LV) for assessment of LV wall thickness (reference values: IVS: 6-10mm & LVPW: 6-10mm) \[7\], LV dimensions (reference values: LVEDD: 42-59mm & LVESD: 24-42mm) \[7\], LV systolic function \[8\] (EF of 50% or more is generally considered normal \[9\]). LV diastolic function using pulsed wave Doppler across mitral valve inflow in the apical four-chamber view \[10\] [Diastolic dysfunction was present if: E/A ratio is less than 1 and deceleration time (DT) of E wave is more than 240 msec (Impaired relaxation pattern) or E/A ratio more than 1 and DT 160-220 msec (pseudonormal pattern) or E/A ratio more than 1.5 and DT less than 160 msec (restrictive pattern)], LV mass in grams which was calculated by the following equation: $LVM = 1.04 \left( \left( LVID + PWT + IVST \right)^3 - LVID^3 \right) x 0.8 + 0.6$ \[11\].

B- Left Atrium (LA) for assessment of LA dimensions (antero-posterior, longitudinal & transverse diameter) \[7\].

C- Mitral Valve & Annulus for assessment of mitral valve calcification and function (MR, MS).

D- Aortic Valve for assessment of aortic valve sclerosis, calcification and function (AS, AR).

E- Aorta for assessment of annular, sinotubular junction & ascending aorta size (reference values: Annular size: 20-31mm, Sinutubular size: 22-36mm, Ascending aorta: 22-36mm) \[12\].

F- Pulmonary artery pressure using pulmonary acceleration time (PAT) (reference value: PAT >120 msec). Then, mean pulmonary artery pressure (Mean PAP) was calculated from Mahan’s equation: Mean PAP = 79 – (0.45x PAT) (reference value: Mean PAP <20mmHg) \[13\].

**Statistical analysis:**

Statistical analysis was performed using SPSS statistical package version 15. Mean and standard deviation were calculated. Analysis of Variance (ANOVA) or T-tests were used for comparing continuous variables, and chi square test was used for comparing categorical variables. Significance was considered to be achieved for a two-sided probability of chance ($p$-value) $<0.05$.

**Results**

We enrolled 70 individuals presenting to the Echocardiography Unit at Ain Shams University Hospital in the period between January 2008 & July 2009. They were divided into two groups: Group A (included 50 adult hypertensive patients aged between 61-70 years) and group B (control group). There was no significant difference between both groups regarding age, gender and risk factors. Body mass index (BMI) was significantly higher in hypertensive patients. In group A, duration of hypertension ranged from 4-19 years with a mean of 10.78 $\pm$ 3.67 years (Table 1).

**Echocardiographic parameters:**

Hypertensive patients had statistically significant thicker inter-ventricular septum (IVS) (11.41 $\pm$ 1.10 vs 9.34$\pm$0.95mm) ($p=0.0001$), LV posterior wall (11.47 $\pm$0.90 vs 9.29 $\pm$0.94mm) ($p=0.0001$) & higher LVMI (114.74 $\pm$ 19.74 vs 84.95 $\pm$ 16.22g/m$^2$) ($p=0.0001$) while there were no significant difference between both groups as regard LVEDD, LVESD & LV diastolic dysfunction. Left ventricular systolic function was...
Hypertensive patients showed significantly larger LA dimensions whether measured in antero-posterior (38.4±5.50 vs 35.1±5.37mm) (p=0.013), supero-inferior (50.63 ± 6.5 vs 46.3 ± 6.9mm) (p=0.008) or medio-lateral axis (37.15 ± 5.1 vs 34.4±5.2mm) (p=0.023).

As regard the mitral valve, calcification [4 (8%) vs 0 (0%)] (p=0.013) & mild MR [5 (10%) vs 0 (0%)] (p=0.013) were statistically more affecting hypertensive patients. The same for aortic valve calcification [6 (12%) vs 0 (0%)] (p=0.019), sclerosis [12 (24%) vs 1 (5%)] (p=0.001) & mild AR [2 (4%) vs 0 (0%)] (p=0.012) which were more associated with hypertension.

Despite the size of ascending aorta was within normal limits in both groups but it was significantly larger (30.18±3.6 vs 27.74±4.4mm) (p=0.009) in hypertensive patients. Also, pulmonary artery pressure was significantly elevated with hypertension when measured using PAT (112.8±18.85 vs 121.0±14.10 msec) (p=0.041) & mean PAP (28.24±8.48 vs 24.55±6.35mmHg) (p= 0.041) (Table 2).

### Effects of duration of hypertension:

There was a statistically significant correlation between the duration of hypertension and the left ventricular mass index (r=0.846, p=0.0001), while this correlation could not be proved with left atrial size (Table 3).

<p>| Table (1): Baseline demographic and clinical characteristics. |</p>
<table>
<thead>
<tr>
<th>Hypertensive group (n=50)</th>
<th>Control group (n=20)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (years)</strong></td>
<td>64.98±2.94</td>
<td>65.25±2.59</td>
</tr>
<tr>
<td><strong>Gender (n, %):</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>31 (62%)</td>
<td>8 (40%)</td>
</tr>
<tr>
<td>Females</td>
<td>19 (38%)</td>
<td>12 (60%)</td>
</tr>
<tr>
<td><strong>DM (n, %)</strong></td>
<td>10 (20)</td>
<td>3 (15)</td>
</tr>
<tr>
<td><strong>Dyslipidaemia (n, %)</strong></td>
<td>9 (18)</td>
<td>2 (10)</td>
</tr>
<tr>
<td><strong>Smoking (n, %)</strong></td>
<td>11 (22)</td>
<td>2 (10)</td>
</tr>
<tr>
<td><strong>BMI (kg/m²)</strong></td>
<td>29.71±2.19</td>
<td>26.15±2.10</td>
</tr>
<tr>
<td><strong>Duration of HTN (years)</strong></td>
<td>10.78±3.67</td>
<td></td>
</tr>
</tbody>
</table>

Results are mean±standard deviation for quantitative data and number (%) for qualitative data.

<p>| Table (2): Echocardiographic findings. |</p>
<table>
<thead>
<tr>
<th>Hypertensive group (n=50)</th>
<th>Control group (n=20)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left ventricle:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IVS (mm)</td>
<td>11.41±1.10</td>
<td>9.34±0.95</td>
</tr>
<tr>
<td>LVPW (mm)</td>
<td>11.47±0.90</td>
<td>9.29±0.94</td>
</tr>
<tr>
<td>LVESD (mm)</td>
<td>32.03±4.61</td>
<td>30.12±3.43</td>
</tr>
<tr>
<td>LVEDD (mm)</td>
<td>50.76±5.07</td>
<td>49.01±5.36</td>
</tr>
<tr>
<td>EF (%)</td>
<td>66.26±4.96</td>
<td>68.6±4.77</td>
</tr>
<tr>
<td>LVMI (g/m²)</td>
<td>114.74±19.74</td>
<td>84.95±16.22</td>
</tr>
<tr>
<td>LV diastolic dysfunction</td>
<td>50 (100%)</td>
<td>19 (95%)</td>
</tr>
<tr>
<td><strong>Left atrial size:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antero-posterior diameter (mm)</td>
<td>38.4±5.50</td>
<td>35.1±5.37</td>
</tr>
<tr>
<td>Supero-inferior diameter (mm)</td>
<td>50.63±6.5</td>
<td>46.3±6.9</td>
</tr>
<tr>
<td>Medio-lateral diameter (mm)</td>
<td>37.15±5.1</td>
<td>34.4±5.2</td>
</tr>
<tr>
<td><strong>Mitrail valve:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcification</td>
<td>4 (8%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Mild MR</td>
<td>5 (10%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td><strong>Aortic valve:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcification</td>
<td>6 (12%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Sclerosis</td>
<td>12 (24%)</td>
<td>1 (5%)</td>
</tr>
<tr>
<td>Mild AR</td>
<td>2 (4%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td><strong>Aorta:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Annular size (mm)</td>
<td>21.7±3.6</td>
<td>20.3±2.4</td>
</tr>
<tr>
<td>Sinotubular size (mm)</td>
<td>28.81±4.1</td>
<td>27.52±2.5</td>
</tr>
<tr>
<td>Ascending aorta (mm)</td>
<td>30.18±3.6</td>
<td>27.74±4.4</td>
</tr>
<tr>
<td>Tricuspid Regurge (mild)</td>
<td>10 (20%)</td>
<td>2 (20%)</td>
</tr>
<tr>
<td>RVSP (mmHg)</td>
<td>34.0±8.84</td>
<td>35.0±0.0</td>
</tr>
<tr>
<td>PAT (msec)</td>
<td>112.8±18.85</td>
<td>121.0±14.10</td>
</tr>
<tr>
<td>Mean PAP (mmHg)</td>
<td>28.24±8.48</td>
<td>24.55±6.35</td>
</tr>
</tbody>
</table>

Results are mean±standard deviation for quantitative data and number (%) for qualitative data.

<p>| Table (3): Correlation between duration of hypertension and some variables. |</p>
<table>
<thead>
<tr>
<th>Duration of hypertension#</th>
<th>r</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVMI (g/m²)</td>
<td>0.846 (**)</td>
<td>0.0001</td>
</tr>
<tr>
<td>LA antero-posterior diameter</td>
<td>0.161</td>
<td>0.265</td>
</tr>
<tr>
<td>LA supero-inferior diameter</td>
<td>0.087</td>
<td>0.546</td>
</tr>
<tr>
<td>LA medio-lateral diameter</td>
<td>0.167</td>
<td>0.248</td>
</tr>
</tbody>
</table>

**Correlation is significant at the 0.01 level.
Hypertension, the most treatable form of cardiovascular disease, has been identified as a major risk factor for coronary heart disease [1]. Older people are more likely than younger people to have hypertension [3].

Further emphasis has been given to identification of target organ damage, since hypertension-related subclinical alterations in several organs indicate progression in the cardiovascular disease continuum which markedly increases the risk beyond that caused by the simple presence of risk factors [14].

Older patients more frequently have other risk factors, target organ damage and associated cardiovascular or non cardiovascular clinical conditions than younger ones [15].

Echocardiography is more sensitive than electrocardiography in diagnosing left ventricular hypertrophy as well as other cardiac effects of systemic hypertension [16] and predicting cardiovascular risk [17] and may help in the more precise stratification of overall risk and in determining therapy [18]. So our goal was to study the echocardiographic changes in hypertensive patients aged between 61-70 years.

We found that the left ventricular wall thickness and left ventricular mass index were significantly higher in hypertensive group. The identification of such finding, which is met by other studies [19], is important because concentric left ventricular hypertrophy, which is one of target organ affection in hypertensive patients, has been identified as the cardiac structural parameter that more markedly increases cardiovascular risk [6].

Also, it is well known that the relation between left ventricular mass index and cardiovascular risk is continuous. Concentric hypertrophy, eccentric hypertrophy and concentric remodeling all predict an increased incidence of cardiovascular disease, but concentric hypertrophy has consistently been shown to be the condition which most markedly increases the risk [20,21]. The same was proved by a large sized study such as LIFE which has been able to show that a treatment-induced reduction in left ventricular mass is significantly and independently associated with a reduction of major cardiovascular events, stroke and cardiovascular and all-cause mortality [22].

In our study, left ventricular mass was directly correlated to the duration of hypertension. This was proved by another study which stated that the duration of hypertension is one of the main predictors of hypertensive cardiac damage [23]. Also, all our hypertensive patients, except one, showed increased LVMI which ranged between 92.1-162.4g/m^2 with a mean of 114.74 ± 19.74g/m^2 in contrast to another study in which only 62% of hypertensive patients had increased LVMI which could be attributed to the difference of the population age between both studies as our patients were older with longer duration of hypertension [24].

Left ventricular systolic function which could be assessed by echocardiography has been proposed as possible additional predictors of cardiovascular events [25,26]. In our study, left ventricular systolic function was normal in all hypertensive patients although it was lower than the control group and this normal systolic function may be the result of excessive increase in left ventricular wall thickness in hypertensive patients. This result was similar to a study done by Miguel et al., [24] but in contrary to another study done by Akintunde et al., which found that there is reduced systolic function in hypertensive patients but the latter study was done on 188 hypertensive patients aged between 32 and 86 years with mean age 55.95 ± 10.71 years and the lower ejection fraction occurred among the long term hypertensive subjects suggesting that eccentric hypertrophy heralds a progressive prevalence of congestive heart failure among hypertensive subjects [27].

Left ventricular diastolic filling (a measure of the so-called “diastolic function”) can also be assessed by Doppler measurement of the ratio between the E and A waves of transmitial blood flow velocity [28]. These measurements are of great current interest because it is now recognized that a considerable proportion (about 50%) of heart failure may be explained by “diastolic dysfunction”, with no or little impairment of systolic function, and that so called “diastolic heart failure” is an ominous condition [29]. Also, there is evidence that diastolic dysfunction increases the risk of atrial fibrillation [30]. Furthermore, other studies have reported that diastolic dysfunction predicts subsequent heart failure [26] and is associated with an increased incidence of all cause mortality [31]. Alterations of diastolic function are frequent among hypertensives, and in elderly individuals with elevated blood pressure at least one in four patients may be affected [32].

In our study, there was no significant difference in left ventricular diastolic dysfunction between both groups & this was also proved by other studies.
which go with the fact that the age is one of the predictors of diastolic dysfunction [33]. Yet, all our hypertensive patients had impairment of left ventricular diastolic function which increases their risk of development of atrial fibrillation and subsequent diastolic heart failure.

Also, echocardiography provides some information on the presence and degree of left atrial enlargement, which is related to the risk of atrial fibrillation, cardiovascular disease and death [34-36]. In our study, we had measured the left atrial linear dimensions by M-mode and 2-D echocardiography which are the most common measurements for assessment of left atrial size. LA dimensions, whether measured in supero-inferior, medio-lateral or antero-posterior axis, were found to be significantly larger in hypertensive patients. These results were similar to a study done by Miller et al., which revealed that mean left atrial dimension was 40.4±3.0mm in hypertensive patients versus 34.6±3.0mm in normal individuals [37] as well as another study done by Tsifoufis et al., which revealed that there was significant difference between hypertensive patients and normotensives regarding LA diameter (39mm versus 36mm) [38]. This significant increase in left atrial diameter in association with hypertension could be explained by the development of left ventricular hypertrophy and changes in both left ventricular diastolic and systolic functions.

As regard the relation between the duration of hypertension and LA size, no significant correlation could be proved in our study. The same was found in the study carried by Miller et al, which signifies that the echocardiographic finding of increase in left atrial size found in hypertensive patients may be an early sign of hypertensive heart disease in those with no other discernible cause of left atrial enlargement [37]. On the contrary, another study revealed that LA enlargement is not an early echocardiographic finding but this study was conducted on younger age group with a mean age of 46±12 years and did not exclude hypertensive patients with other cardiovascular diseases or abnormalities such as patients with atrial fibrillation [39].

The presence of mitral valve calcification and mild MR were significantly encountered in our hypertensive patients. This could be attributed to the ability of hypertension to accelerate the degenerative process in the cardiovascular fibrous skeleton. This result was similar to the study done by Arthur et al., which proved that hypertension is strongly and significantly associated with mitral valve calcification [40] and the study done by Jagmeet et al., which proved that hypertension is associated with mitral valve regurgitation [41].

Also, aortic valve calcification and sclerosis were significantly affecting our hypertensive patients when compared to control. This finding which was proved in many studies could be also explained by the ability of hypertension to cause degenerative process in the cardiac fibrous skeleton [40,42,43].

In our study, there was no significant difference between both groups when the aortic annular size or the aortic sino-tubular size were measured while the ascending aortic diameter was significantly larger in hypertensive patients. These results are met with the study done by Michael Kim et al., which stated that aortic diameters increased with increasing quartiles of diastolic and systolic pressures, particularly at the supra-aortic ridge and ascending aorta [44].

An interesting finding of a significantly higher mean pulmonary artery pressure in our hypertensive patients was similar to other studies which proved that hypertension is likely contributing to diastolic dysfunction and the resulting pulmonary hypertension [45].

Our study met some limitations as the patients included were from a single medical center and not a community sample, the sample size was relatively small and the study was done on a specific age group from 61-70 years and not compared to the other age groups. Also, we measured only left atrial diameters and we did not estimate the left atrial volume, in addition, we did not grade the diastolic dysfunction in our patients.

Based on the results of the current study we recommend that (1) Screening and early treatment of hypertension is important to prevent many of the cardiac changes complicating hypertension, (2) A more detailed method is recommended for measurement of left atrial enlargement other than simple measurement of left atrial diameters, (3) It is important to estimate the left ventricular mass, left ventricular diastolic function and pulmonary artery pressure in hypertensive patients.

**Conclusion:**

Finally, we can conclude from our study that the heart is a target organ that is greatly affected by hypertension in the form of increase in left ventricular mass which is affected by the duration
of hypertension, enlargement of left atrial size, dilatation of ascending aorta and the development of pulmonary hypertension.

Conflict of interest: Nothing to be declared.

References


