Heart

Inappropriate Left Ventricular Mass in Patients With Primary Aldosteronism

Maria Lorenza Muiesan, Massimo Salvetti, Anna Paini, Claudia Agabiti-Rosei, Cristina Monteduro, Gloria Galbassini, Eugenia Belotti, Carlo Aggiusti, Damiano Rizzoni, Maurizio Castellano, Enrico Agabiti-Rosei

Abstract—Assessment of appropriateness of left ventricular mass (LVM) for a given workload may better stratify hypertensive patients. Inappropriate LVM may reflect the interaction of genetic and neurohumoral factors other than blood pressure playing a significant role in myocardial growth. Primary aldosteronism (PA) represents a clinical model useful in assessing the effect of aldosterone increase on LVM. The aim of this study was to evaluate the inappropriateness of LVM in patients with PA. In 125 patients with PA (54 females; adrenal hyperplasia in 73 and adenoma in 52 patients) and in 125 age-, sex-, and blood pressure–matched, essential hypertensive patients, echocardiography was performed. The appropriateness of LVM was calculated by the ratio of observed LVM to the predicted value using a reference equation. In all of the subjects plasma renin activity and aldosterone, as well as clinic and 24-hour blood pressure, were measured. The prevalence of inappropriate LVM was greater in patients with traditionally defined left ventricular hypertrophy (70% and 44%, respectively; \( P = 0.02 \)) but also in patients without left ventricular hypertrophy (17% and 9%, respectively; \( P = 0.085 \)). In PA patients, a correlation was observed between the ratio of observed:predicted LVM and the ratio of aldosterone:plasma renin activity levels (\( r = 0.29; \ P = 0.003 \)) or the postinfusion aldosterone concentration (\( r = 0.44; \ P = 0.004; \ n = 42 \)). In conclusion, in patients with PA, the prevalence of inappropriate LVM is increased, even in the absence of traditionally defined left ventricular hypertrophy. The increase in aldosterone levels could contribute to the increase of LV mass exceeding the amount needed to compensate hemodynamic load. (Hypertension. 2008;52:529-534.)

Key Words: hypertension arterial ■ hypertrophy ■ echocardiography ■ aldosterone ■ inappropriate LV mass

In hypertension, left ventricular (LV) hypertrophy (LVH) is initially a useful compensatory process to abnormal loading conditions, but it is also the first step toward the development of overt clinical disease.1–4 In the attempt to discriminate between normal (compensatory) and abnormal (noncompensatory) increase in LV mass (LVM), it has been recently proposed that researchers evaluate the LVM increase in hypertensive patients, taking into account gender and cardiac loading conditions, together with some measure of body size.5 Patients with inappropriate LVM, exceeding the amount needed to adapt to stroke work for a given gender and body size, tend to cluster with metabolic risk factors.6 More interestingly, the prevalence of low systolic myocardial function, as well as of abnormal relaxation, is greater in hypertensive patients with inappropriate LVM, suggesting that this condition may represent an accelerated phase of transition from compensatory LVH toward heart failure.7,8

Three studies have demonstrated that the presence of inappropriate LVM implies a greater risk of cardiovascular (CV) events, either in the presence or in the absence of traditionally defined LVH,9–11 and changes in LVM appropriateness may add prognostic information, mainly in patients with persistence or development of traditionally defined LVH.9,11

A substantial percentage of LV mass variance, however, remains unexplained, even when the influence of cardiac workload, body size, and gender are taken into account. In fact, other nonhemodynamic (genetic) or neurohumoral (renin-angiotensin-aldosterone system) factors may exert influence on the individual LVM. Recent experimental studies suggest that long-term exposure to increased aldosterone levels contributes to the development of cardiac, vascular, and renal damage,12,13 also independent of blood pressure (BP) increase.

In patients with primary aldosteronism (PA), a higher prevalence and degree of CV structural and functional changes have been demonstrated,14–18 possibly influencing an excess rate of CV events, as suggested by 2 recent longitudinal studies of large cohorts comparing patients with essential hypertension and with PA.19,20 Therefore,
we considered it worthwhile to investigate the appropriateness of LV mass in patients with PA and to evaluate whether the excess of LV mass could be related to aldosterone increase.

Methods

Subjects

A total of 125 consecutive uncomplicated patients with PA, aged 23 to 79 years (71 males and 54 females) were enrolled into the study: 52 of them had an aldosterone-producing adenoma, and in 73, idiopathic hyperaldosteronism was diagnosed. Basal evaluation was performed after discontinuation of any antihypertensive treatment for ≥4 weeks, except for calcium channel and α-adrenoceptor blockers, required in 30% of patients. Patients with hypokalemia had been given oral potassium supplements before the hormonal evaluations.

During the years 1988–1995, only patients with a high suspicion for PA, such as those with spontaneous or diuretic-induced hypokalemia or hypertension and an adrenal mass, were further examined for PA; from 1996 to 2002, the aldosterone:renin ratio (ARR) was progressively used with a cutoff level of 40 ng/dL per ng/mL/h plus an aldosterone level >150 pg/dL. A positive intravenous saline load (ie, posttest aldosterone levels >50 pg/dL)14,15 was considered a confirmatory test. In addition, a computed tomography and/or magnetic resonance of the adrenal glands were used as imaging techniques23,24 (for details, please see the data supplement available online at http://hyper.ahajournals.org).

Patients with PA were carefully matched for age, gender, body size, clinic BP values, and use of antihypertensive treatment at the time of the basal examination with 125 patients with essential hypertension, referred to our outpatient clinic for high BP diagnostic workup, including supine and standing plasma renin activity and aldosterone measurements. Eighteen patients with essential hypertension and 21 patients with PA had received previous treatment for various periods of time with a combination of 2 to 3 drugs (calcium channel blockers, angiotensin-converting enzyme inhibitors, diuretics, or β-blockers). Each subject provided informed consent for the study, which was approved by institutional ethics committee.

Clinical Evaluation

Patients underwent a thorough clinical examination. CV risk factors were carefully assessed, and a documented clinical history was collected. BP was measured using a mercury sphygmomanometer (Korotkoff phase V for diastolic BP), with a regular adult cuff, after the echocardiographic examination in a semirecumbent position and thereafter in a sitting position (3 measurements in each position).

Echocardiography

In all of the patients, a technically optimal M-mode echocardiogram (<2D control) was obtained at baseline and included in the study. M-mode echocardiographic tracings were recorded on a videotape and printed on a strip-chart recorder. Two different readers measured all of the echocardiograms, in a blind manner, and the average of the 2 calculations was considered for the study. The LV internal dimensions, interventricular septum, and posterior wall thicknesses were measured according to the recommendations of the American Society of Echocardiography,25 and derived anatomic variables were calculated26. LVMI index (LVMI) was obtained by normalization of LVM for height to the 2.7 power.27 and LVH was prospectively defined as a value of LVMI ≥50 g/m² in males and 47 g/m² in females.27 End-diastolic relative wall thickness (RWT) was calculated as the ratio of posterior wall thickness to one half of the LV internal dimension, and concentric geometry was defined as RWT ≥0.42.28 Ejection fraction, endocardial, and midwall fractional shortening (FS) were calculated by standard methods.29 In addition, transmitral Doppler flow patterns recorded at the mitral valve tips were used to measure early and late-wave diastolic filling velocities, their ratio (early/late-wave ratio), and early wave deceleration time.30 LV end-diastolic and end-systolic volumes were calculated with the Teicholz’s correction of the cube formula.31,32 Stroke work (SW) was estimated as systolic BP (measured in a semirecumbent position after the echocardiographic study) times stroke volume and converted into gram-meters by multiplying by 0.0144. The theoretical value of LVM was estimated using an equation developed previously:33 Predicted LVM (pLVM)=55.37+6.64 × height (m²)+0.64 × SW−18.07 × gender (where gender was coded as male=1 and female=2).

LVM measured by M-mode tracing25 was divided by the predicted LVM and was expressed as a percentage (observed LVM/predicted LVM). Observed LVM/predicted LVM was categorized using the 97.5th percentiles of the distribution in the normotensive, normal weight reference adult population, and inappropriate LVM was defined as an excess of >35% from the predicted value (ie, observed LVM/predicted LVM >135%).

Statistical Analysis

The statistical significance of differences between the 2 groups (PA patients and controls) was assessed by unpaired t test. χ² statistic was used to assess differences of categorical variables between groups.

Relationships between the ratio of observed:predicted LVM and urinary aldosterone or aldosterone:renin ratio were investigated calculating the Pearson’s coefficient. Stepwise multivariate logistic regression analysis was used to select the variables independently associated with the presence of inappropriate LV mass; entry into the equations was restricted to variables whose partial F test was significant at the level of P<0.05. All of the statistical tests were 2-tailed at the 0.05 P value. Results are expressed as means±SDs. All of the analyses were carried out with the SPSS 13.01 statistical package.

Results

Patients With PA and Essential Hypertension

Demographic characteristics of patients with PA and with essential hypertension are shown in Table 1; by definition, no differences in age, gender distribution, BP, and heart rate were required in 30% of patients. Patients with hypokalemia had been given oral potassium supplements before the hormonal evaluations.

### Table 1. Demographic Characteristics and Laboratory Data of Patients

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Primary Aldosteronism</th>
<th>Essential Hypertension</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>50±11</td>
<td>51±11</td>
<td>0.59</td>
</tr>
<tr>
<td>Gender, male/female, %</td>
<td>71/54</td>
<td>71/54</td>
<td>NS</td>
</tr>
<tr>
<td>Smokers, %</td>
<td>21</td>
<td>18</td>
<td>NS</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>27.4±5</td>
<td>27.6±5</td>
<td>0.70</td>
</tr>
<tr>
<td>SBP/DBP clinic, mm Hg</td>
<td>152±18/96±11</td>
<td>148±16/94±9</td>
<td>0.12/0.09</td>
</tr>
<tr>
<td>HR clinic, bpm</td>
<td>67±10</td>
<td>70±12</td>
<td>0.07</td>
</tr>
<tr>
<td>SBP/DBP 24 h, mm Hg</td>
<td>139±17/88±12</td>
<td>133±16/84±12</td>
<td>0.08/0.11</td>
</tr>
<tr>
<td>HR 24 h, bpm</td>
<td>71±10</td>
<td>73±11</td>
<td>0.08</td>
</tr>
<tr>
<td>Glycemia, mg/dL</td>
<td>97±22</td>
<td>99±20</td>
<td>0.10</td>
</tr>
<tr>
<td>Total cholesterol, mg/dL</td>
<td>207±42</td>
<td>216±43</td>
<td>0.12</td>
</tr>
<tr>
<td>Triglycerides, mg/dL</td>
<td>125±101</td>
<td>137±100</td>
<td>0.44</td>
</tr>
<tr>
<td>Creatinine, mg/dL</td>
<td>0.96±0.27</td>
<td>0.98±0.24</td>
<td>0.5</td>
</tr>
<tr>
<td>Glomerular filtration rate, MDRD, mL/min</td>
<td>84±25</td>
<td>80±19</td>
<td>0.21</td>
</tr>
<tr>
<td>Uric acid, mEq/dL</td>
<td>4.8±1.6*</td>
<td>5.3±1.7</td>
<td>0.04</td>
</tr>
<tr>
<td>Serum K⁺, mEq/dL</td>
<td>3.7±0.57†</td>
<td>4.15±0.28</td>
<td>0.001</td>
</tr>
</tbody>
</table>

HR indicates heart rate; NS, not significant; MDRD, Modification of Diet in Renal Disease Study Group. Data are means±SDs unless otherwise specified. *P<0.05. †P<0.001.
values measured in the clinic were observed between patients with essential hypertension and PA. Heart rate and BP measured by 24-hour ambulatory monitoring were also similar in PA and essential hypertensive subjects.

Serum potassium was lower in patients with PA, as compared with patients with essential hypertension, whereas no significant differences in laboratory parameters were observed. LVMI was significantly higher in patients with PA, as compared with control subjects, whereas no differences in RWT were observed. LVH was observed in 57 of 125 PA patients and in 27 of 125 control subjects (45% and 22%, respectively; \( \chi^2 P<0.001 \)); concentric geometry was observed in 26 of 125 PA patients and in 31 of 125 control subjects (21% versus 25%; \( \chi^2 P=0.45 \)).

Prevalence of inappropriate LVM was greater in patients with PA with respect to those with essential hypertension (71% and 17%, respectively; \( \chi^2 P<0.0001 \)); this was true in patients with traditionally defined LVH (70% and 44%, respectively; Fisher’s exact test \( P=0.02 \)) but also in patients without LVH (17% and 9%, respectively; Fisher’s exact test \( P=0.085 \); Figure). Among patients with PA, LVMI was 49±18 g/m² and 51±16 g/m²; and RWT was 0.36±0.07 and 0.375±0.073 in the 74 patients with aldosterone-producing adenoma and in the 51 patients with adrenal hyperplasia, respectively. The prevalence of inappropriate LVM was 54% in patients with aldosterone-producing adenoma and 42.5% in patients with adrenal hyperplasia.

The left atrium was larger in patients with PA, whereas the early:late-wave ratio, E wave deceleration time, and IVRT did not differ between groups (Table 2). The prevalence of diastolic dysfunction, diagnosed according to the European Society of Cardiology guidelines, was slightly greater in patients with PA as compared with essential hypertensive subjects (40% versus 32%), although the difference did not reach statistical significance.

**Patients With Appropriate and Inappropriate LVM**

In patients with PA, the mean age, body mass index, and systolic and diastolic BP both measured in the clinic and during 24-hour BP monitoring did not differ between those with inappropriate or appropriate LVM (Table 3). Uric acid and the ARR were increased in patients with PA and inappropriate LVM. Relative wall thickness was higher, whereas midwall FS was lower in patients with inappropriate LVM (Table 4).

In essential hypertensive control subjects, systolic and diastolic BPs measured during 24-hour monitoring were higher in patients with inappropriate LVM, whereas no significant differences were observed in fasting glucose concentration, serum uric acid, and the ARR between patients with inappropriate LVM and appropriate LVM (Table 3). Relative wall thickness was higher, whereas midwall FS was lower in patients with inappropriate LVM (Table 4).

In both groups (PA and essential hypertensive control subjects) the glomerular filtration rate was slightly reduced in patients with inappropriate LVM, although differences reached statistical significance only in essential hypertensive subjects (Table 3). In patients with inappropriate LVM, patients with PA had a greater prevalence of diastolic dysfunction (54% versus 30%; \( \chi^2 P=0.06 \)). In patients with PA, LVMI was not related to ARR at univariate analysis \( (r=0.053; P=0.67) \), whereas a low, albeit statistically significant, correlation was observed between the degree of LVM inappropriateness and the ARR \( (r=0.29; P=0.001) \) or urinary aldosterone concentration \( (r=0.19; P=0.05) \). In patients with PA who underwent a saline infusion \( (n=42) \), a significant correlation was found between postinfusion aldosterone levels and LVMI \( (r=0.32; P=0.04) \) and the observed:predicted LVM ratio \( (r=0.44; P=0.004) \); such a correlation explained the 10% and 19% of the variation of LVMI and LV mass inappropriateness, respectively.

**Table 2. Echocardiographic Data of Patients**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Primary Aldosteronism, Mean±SD</th>
<th>Essential Hypertension, Mean±SD</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVMI, g/m²</td>
<td>50±17†</td>
<td>40±11</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RWT</td>
<td>0.37±0.07</td>
<td>0.36±0.07</td>
<td>0.45</td>
</tr>
<tr>
<td>FS endocardial, %</td>
<td>40±7</td>
<td>41±7</td>
<td>0.43</td>
</tr>
<tr>
<td>FS midwall, %</td>
<td>18.5±3*</td>
<td>19±3</td>
<td>0.014</td>
</tr>
<tr>
<td>Left atrium, mm</td>
<td>3.89±0.57*</td>
<td>3.67±0.58</td>
<td>0.04</td>
</tr>
<tr>
<td>Early:late-wave ratio</td>
<td>1.02±0.29</td>
<td>1.08±0.36</td>
<td>0.16</td>
</tr>
<tr>
<td>Early wave deceleration, ms</td>
<td>195±51</td>
<td>185±42</td>
<td>0.13</td>
</tr>
<tr>
<td>IVRT, ms</td>
<td>87±26</td>
<td>83±20</td>
<td>0.19</td>
</tr>
</tbody>
</table>

RWT, isovolumetric relaxation time.  
*\( P<0.05 \).  
†\( P<0.001 \).
Multivariate logistic regression analysis performed in all of the patients (essential hypertensive subjects and PA) showed that only the presence of PA (hazard ratio: 2.33; 95% CI: 1.06 to 3.0; \( P=0.03 \)) and the increase in 24-hour systolic BP (hazard ratio: 1.023; 95% CI: 1.004 to 1.044; \( P=0.02 \)) were significantly related to the ARR and to 24-hour urinary aldosterone. In patients who underwent a saline infusion, the amount of LV mass exceeding the amount needed to compensate for the hemodynamic load, but not LVMI as traditionally measured, was significantly related to the ARR and to 24-hour urinary aldosterone. 

### Discussion

LVH is more severe in PA than in essential hypertension and seems to develop early among all forms of end organ damage.\(^{13-20,34-38}\) Some evidence has accumulated supporting the concept that, in patients with PA, cardiac hypertrophy is not the simple consequence of a raised hemodynamic pressure and volume load. Available data with 24-hour BP monitoring have not supported the concept that a greater 24-hour load or a nondipping pattern could exert an influence on the increase in LV mass.\(^{17,37,38}\)

The results of the present study indicate that the increase in aldosterone levels could contribute to the increase of LV mass exceeding the amount needed to compensate hemodynamic load. First, the prevalence of inappropriate LV mass was greater in patients with PA than in carefully matched essential hypertensive subjects, and this was evident both in patients with traditionally defined LVH and in those without LVH. Second, in patients with PA, 24-hour BP values were similar in patients with appropriate and inappropriate LV mass. Finally, in patients with PA, the amount of LV mass exceeding the amount needed to compensate for the hemodynamic load, but not LVMI as traditionally measured, was significantly related to the ARR and to 24-hour urinary aldosterone. In patients who underwent a saline infusion, the observed/predicted LV mass, and, to a lesser extent, LV mass index, were significantly related to postinfusion aldosterone levels. However, we were not able to find a correlation between the degree of LV mass inappropriateness and plasma aldosterone concentrations in essential hypertensive patients, as shown previously in the Insulin Carotids US Scandinavia Study.\(^{39}\)

Aldosterone stimulates cell growth and cardiomyocyte hypertrophy, which can evoke an increased LVM\(^{12,40,41}\); the chronotropic action of aldosterone, linked to an increase of T-channel activity, could contribute to the deleterious effect of aldosterone on cardiac function.\(^{42}\) Aldosterone also induces profound changes in the extracellular matrix, leading to

### Table 3. Demographic Characteristics and Laboratory Data of Patients With Essential Hypertension and With PA, According to the Presence of Inappropriate or Appropriate LVM (Mass)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>PA, Mean±SD</th>
<th>Essential Hypertension, Mean±SD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Appropriate LVM</td>
<td>Inappropriate LVM</td>
</tr>
<tr>
<td>Age, y</td>
<td>50±11</td>
<td>51±11</td>
</tr>
<tr>
<td>BMI, kg/h²</td>
<td>26.5±4</td>
<td>28.5±5*</td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>153±19</td>
<td>150±18</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>96±11</td>
<td>95±12</td>
</tr>
<tr>
<td>24 h SBP, mm Hg</td>
<td>137±16</td>
<td>141±20</td>
</tr>
<tr>
<td>24 h DBP, mm Hg</td>
<td>88±12</td>
<td>87±11</td>
</tr>
<tr>
<td>Glycemia, mg/dL</td>
<td>95±20</td>
<td>101±24</td>
</tr>
<tr>
<td>Glomerular filtration rate MDRD, mL/min</td>
<td>87±26</td>
<td>80±21</td>
</tr>
<tr>
<td>Uric acid, mEq/dL</td>
<td>4.4±1.3</td>
<td>5.4±1.7†</td>
</tr>
<tr>
<td>Aldosterone:plasma renin activity ratio</td>
<td>127±108</td>
<td>187±161†</td>
</tr>
</tbody>
</table>

MDRD, Modification of Diet in Renal Disease Study Group.

\( ^* P<0.05 \) vs appropriate LVM.

\( ^† P<0.01 \) vs appropriate LVM.

### Table 4. Echocardiographic Data of Patients With Essential Hypertension and With PA, According to the Presence of Inappropriate or Appropriate LVM (Mass)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>PA, Mean±SD</th>
<th>Essential Hypertension, Mean±SD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Appropriate LVM</td>
<td>Inappropriate LVM</td>
</tr>
<tr>
<td>RWT</td>
<td>0.34±0.06</td>
<td>0.41±0.06†</td>
</tr>
<tr>
<td>Endocardial FS, %</td>
<td>74±9</td>
<td>65±8</td>
</tr>
<tr>
<td>Midwall FS, %</td>
<td>20.2±2.6</td>
<td>16.2±2.02†</td>
</tr>
<tr>
<td>Left atrium, mm</td>
<td>3.75±0.54</td>
<td>4.1±0.56</td>
</tr>
<tr>
<td>Early:late-wave ratio</td>
<td>1.04±0.28</td>
<td>1.00±0.32</td>
</tr>
<tr>
<td>Early wave deceleration, ms</td>
<td>186±46</td>
<td>208±55*</td>
</tr>
<tr>
<td>IVRT, ms</td>
<td>84±26</td>
<td>92±24</td>
</tr>
</tbody>
</table>

IVRT, isovolumetric relaxation time.

\( ^* P<0.05 \) vs appropriate LVM.

\( ^† P<0.01 \) vs appropriate LVM.
collagen deposition, and, subsequently, to myocardial fibrosis, arterial stiffening, and vascular alterations in small resistance arteries. \(^{14,43}\) In animal models aldosterone has been demonstrated to influence LV remodeling independent of its impact on systemic BP, as cardiac fibrosis was prevented by treatment with nonantihypertensive doses of spironolactone. One speculation is that the excessive growth of LVM is associated with cardiomyocyte hypertrophy and with changes in myocardial structure, with a disproportionate increase in extracellular matrix and myocardial fibrosis, as observed in postmortem specimens of human hearts from a few patients with autopsy-proven adrenal adenoma. \(^{49}\)

Rossi et al\(^ {46}\) were able to demonstrate that PA patients exhibited greater alterations of videodensitometric indexes of LV myocardial texture compared with demographically and hemodynamically similar essential hypertensive patients. The videodensitometric parameter changes were evident despite the modest increase of LV mass and were inversely related to plasma renin activity, indicating that lower plasma renin activity values are associated with higher collagen deposition in the heart. In our study, impairment of midwall FS and LV filling and relaxation were more frequently documented in patients with PA and with inappropriate LV mass, suggesting that an augmented collagen content could alter the intrinsic contractile properties of the heart, as well as diastolic function, although it cannot be assumed by our study and requires further investigations, whether the inappropriate increase in LV mass is mainly because of abnormal growth of cardiomyocytes or LV fibrosis.

Previous studies have reported an association between metabolic risk factors and inappropriate LV mass in hypertensive patients with low or high CV risk and in normotensive subjects with metabolic syndrome.\(^ {6}\) It may be hypothesized that metabolic risk factors for CV disease, such as abdominal obesity, impaired insulin sensitivity, and dyslipidemia, may increase the hemodynamic load to the heart, through their effect on arterial vascular wall, thus contributing to the development of inappropriate LV mass.

In PA patients, a high prevalence of the metabolic syndrome has been shown recently.\(^ {50}\) Glucose intolerance and reduced insulin sensitivity in PA are probably attributable to a direct effect of aldosterone on insulin receptor function\(^ {51}\) or could be mediated by hypokalemia,\(^ {52}\) although the precise mechanisms of glucose disturbances in PA remain to be elucidated.

In this study, we could not observe any significant difference in metabolic parameters and, in particular, fasting glucose, between patients with PA and essential hypertension, although it should be kept in mind that patients were carefully matched for body size. In patients with PA, those with inappropriate LVM showed a significantly higher BMI and uric acid and a slight, albeit not statistically significant, increase in fasting glucose.

The influence of treatment on the inappropriateness of LV mass in essential hypertensive patients and in PA patients has not yet been established. To this regard, the potential confounding influence of pharmacological treatment on CV structure cannot be completely excluded in the small number of previously treated patients included in the study.

In 2 groups of PA patients,\(^ {15,18}\) either the removal of an adrenal adenoma or antihypertensive treatment with a selective aldosterone antagonist may significantly reduce LV mass, possibly improving CV prognosis in patients with PA\(^ {19,20}\); however, the effect of absence/antagonism of aldosterone cannot be differentiated from the one induced by the control in BP. It could be argued that, in PA patients, the decrease in BP may depend, at least in part, on the degree of concomitant vascular alterations in small resistance arteries, as reported recently.\(^ {53}\)

In conclusion, our results indicate that the increase in aldosterone levels could contribute to the increase of LV mass exceeding the amount needed to compensate for hemodynamic load.

**Perspectives**

The results of this study support the concept that hyperaldosteronism may induce an additional increase of LV mass beyond hemodynamic load and promotes further investigations to evaluate whether the inappropriate increase in LV mass is mainly because of abnormal growth of cardiomyocytes or LV fibrosis. The measurement of inappropriateness of LV mass should raise concern about the diagnosis of a secondary form of hypertension and a more appropriate choice of treatment, in addition to more strict BP control.

**Disclosures**

None.

**References**


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