Hyperaldosteronism and Left Ventricular Hypertrophy

To the Editor:

In their prospective study, Gaddam et al.1 have examined the effects of spironolactone on cardiac structure in 34 patients with resistant hypertension who were separated according to their plasma aldosterone levels. At baseline, cardiac volume overload was detected in patients with higher aldosterone levels that was significantly and rapidly reduced by spironolactone, whereas left ventricular (LV) mass was comparable at baseline and decreased significantly after treatment in both patients with higher and lower plasma aldosterone.

Detection of comparable baseline LV mass even in the presence of significant volume overload is difficult to explain. As pointed by the authors, several cross-sectional echocardiographic studies have demonstrated excess LV hypertrophy in hypertensive patients with hyperaldosteronism independent of cardiac workload. In a study that examined 125 patients with primary aldosteronism, Muiesan et al.2 reported that the prevalence of inappropriate LV mass was greater than in matched controls with essential hypertension even in the absence of traditionally defined LV hypertrophy, suggesting that aldosterone contributes to the increase of LV mass exceeding the amount needed to compensate for the hemodynamic load.

The crucial point in Gaddam’s study, however, is related to the reversal of LV hypertrophy that occurred rapidly after spironolactone treatment. In fact, LV mass decreased in both patients with higher and lower plasma aldosterone, but the degree of LV mass regression tended to be greater in the former group (21 versus 12%), suggesting, in this case, a direct involvement of aldosterone in the development of LV hypertrophy. These findings are in partial agreement with those of a long-term prospective study,3 in which we reported significant reduction of LV mass to occur in 30 patients with primary hyperaldosteronism who were treated with spironolactone and in whom a multivariate analysis demonstrated that pretreatment aldosterone levels predict treatment-induced LV mass decrease independent of blood pressure change. In our study, however, decrease in LV mass was much slower than that in Gaddam’s study, reaching statistical significance only after a few years, and occurred in the absence of significant changes in cardiac and systemic markers of the volume status. We wonder whether the rapid effect of spironolactone on LV mass observed by Gaddam et al could not be simply due to the marked and rapid decrease in blood pressure obtained with addition of spironolactone to treatment.

Knowledge of the effects of mineralocorticoid receptor antagonists on LV mass is important, because LV hypertrophy is an important predictor of major cardiovascular events. In our patients with hyperaldosteronism, reversal of LV hypertrophy with spironolactone treatment was associated with significant reduction of cardiovascular events.4 These benefits could be relevant not only in patients with primary aldosteronism, but also in patients with lesser degrees of aldosterone excess, and this hypothesis will have to be tested in future studies.

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Disclosures

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