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CE - LETTER TO THE EDITOR

Anti-hypertensive drugs and left ventricular hypertrophy: a clinical update

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We read with great interest the article by Milan et al. [1], which briefly gives a clinical update about anti-hypertensive drugs and left ventricular hypertrophy (LVH). The authors remark on how LVH seems to benefit from all classes of anti-hypertensive drugs; however, antagonists of the renin-angiotensin-aldosterone system (RAAS) have demonstrated an additional benefit in the inhibition and reversal of myocardial interstitial fibrosis.

Recently, aliskiren has emerged as the first direct renin inhibitor (DRI) available for clinical use [2]. By inhibiting the conversion of angiotensinogen to angiotensin I and by decreasing PRA, aliskiren may provide a more complete blockade of the RAAS and offers a new opportunity to explore multistep RAAS blockade.

Initial studies in patients with diabetic nephropathy, LVH, and congestive heart failure (CHF) have shown promising effects on surrogate markers. In this regard, ALLAY (aliskiren in left ventricular hypertrophy) [3] was a 36-week study that compared the effects of aliskiren 300 mg and losartan 100 mg alone, and in combination on left ventricular hypertrophy (assessed by magnetic resonance imaging) in 465 overweight, hypertensive patients. Aliskiren monotherapy was statistically the same as losartan in reducing left ventricular mass index (4.9 g/m^2 for aliskiren versus 4.8 g/m^2 for losartan). The aliskiren–losartan combination resulted in a greater reduction in left ventricular mass index (5.8 g/m^2) compared with losartan monotherapy, although the difference was not statistically significant. These results were independent of BP decrease. Safety and tolerability were similar across all the treatment groups.

Conflict of interest None.

References

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