Increased levels of oxidative stress, subclinical inflammation, and myocardial fibrosis markers in primary aldosteronism patients

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Abstract

Background: Patients with primary aldosteronism experience greater left ventricular hypertrophy and a higher frequency of cardiovascular events than do essential hypertensive patients with comparable blood pressure levels. Aldosterone has been correlated with increased oxidative stress, endothelial inflammation, and fibrosis, particularly in patients with heart disease. Aim: To evaluate oxidative stress, subclinical endothelial inflammation, and myocardial fibrosis markers in patients with primary aldosteronism and essential hypertension. Design and individuals: We studied 30 primary aldosteronism patients and 70 control essential hypertensive patients, matched by age, sex and median blood pressure. For all patients, we measured the serum levels of aldosterone, plasma renin activity, malondialdehyde (MDA), xanthine oxidase, metalloproteinase-9, ultrasensitive C-reactive protein and amino terminal propeptides of type I (PINP), and type III procollagen. We also evaluated the effect of PA treatment in 19 PA individuals. Results: PA patients showed elevated levels of MDA (1.70 ± 0.53 versus 0.94 ± 0.65 μmol/l, P <0.001) and PINP (81.7 ± 50.6 versus 49.7 ± 27 mg/l, P = 0.002) compared with essential hypertensive controls. We found a positive correlation between MDA, PINP, and the serum aldosterone/plasma renin activity ratio in primary aldosteronism patients. Clinically, treating primary aldosteronism patients decreased MDA and PINP levels. Conclusion: We detected higher levels of MDA and PINP in primary aldosteronism patients, suggesting increased oxidative stress and myocardial fibrosis in these individuals. Treating primary aldosteronism patients reduced MDA and PINP levels, which may reflect the direct effect of aldosterone greater than endothelial oxidative stress and myocardial fibrosis, possibly mediated by a mineralocorticoid receptor.

Keywords

Aldosterone, Endothelial inflammation, Myocardial fibrosis, Oxidative stress, Primary aldosteronism.