GW23-e0745 **ALDOSTERONE IS A VASCULAR CALCIFICATION PROMOTING FACTOR**

doi:10.1136/heartjnl-2012-302920a.123

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Objectives Aim: To observe aortic and cardiac aldosterone expression and its receptor changes in rats with vascular calcification, and exogenous aldosterone effects on vascular calcification, so as to explore the significance of aldosterone in vascular calcification.

Methods Male SD rats were randomly divided into five groups. (1) Normal control group; (2) Aldosterone group: rats were received aldosterone subcutaneously for 6 weeks (20 μgin 0.1 ml ethanol, 1/d); (3) Calcification group: rats were received intramuscular injection with vitamin D3 (300 000 IU/kg) and anintragastric dose of nicotine (25 mg/kg, in peanut oil) at 8:00 on the first day; nicotine was gavaged again at 18:00 on the same day. (4) Calcification +aldosterone group: Rats were treated as the calcification group, and also received aldosterone subcutaneously for 6 weeks (20 μg in 0.1 mlethanol, 1/days). (5) Calcification+spironolactone group: Rats were treated as the calcification group, with the exception of oral gavage of spironolactone (40 mg/kg/d)] for 6 weeks. The control group received normal saline injection, oral gavage of peanut oil, and ethanol treatment. At the end of the experiment, the extent of aortic calcification was confirmed by Von Kossa staining and measurement of calcium content. Alkaline phosphatases activity was also evaluated. The deposition of collagen in cardiovascular tissues was measured by masson staining. The content of aldosterone and urotensin II in plasma and aorta were determined by radioimmunoassay. The immunoactivity of mineralocorticoid receptor and urotensin II receptor were evaluated by immunohistochemistry. The content of C-reactive peptide, interleukin-6, tumour necrosis factor-α and monocyte chemoattractant protein-1 in the serum were determined by radioimmunoassay.

Results Von Kossa staining showed significant aortic calcium deposition in calcified rats. Aortic calcification was further aggravated when aldosterone was added. It was decreased in calcification +spirolactone group as compared with the calcification group, and the calcification+aldosterone group. In addition, aortic calcium content in aldosterone group increased, but not statistically significant ($p>0.05$), compared with the control group. In calcification group, the aortic calcium content increased by63.45% ($p<0.01$), and 27.55% ($p<0.05$), respectively, compared with the controls and the aldosterone group. In calcification+aldosterone group, it was elevated by 121.72% ($p<0.01$), 73.02% ($p<0.01$), and 35.65% ($p<0.01$), respectively, compared with the controls, the aldosterone group, and the calcification group. However, in calcification+spironolactone group, it decreased by 41.31% ($p<0.01$), and 56.73% ($p<0.01$), than the calcification group and calcification+aldosterone group respectively. Alkaline phosphatase activity showed a similar trend. Vascular and myocardial collagen staining showed that the most obvious staining is seen in the calcification +aldosterone group. It was slightly reduced in the calcification +spironolactone group than the calcified rats. Immunohistochemical staining showed that mineralocorticoid receptor and urotensin II receptor the immunoactivity was higher, in the aldosterone group, the calcification group, and the calcification+aldosterone group, respectively, than the control group, with the most obvious staining in the calcification+aldosterone group. However, the immunoactivity in the calcification +spironolactone group was lower than the calcification group. Serum inflammatory factors, including C-reactive protein, interleukin-6, tumour necrosis factor-αand monocyte chemoattractant protein-1, increased significantly in the aldosterone group, calcified alone group, and calcification+aldosterone group, than the controls, especially in the calcification+aldosterone group. However, these inflammatory factors decreased in the calcification+spironolactone group, similar to the controls. In addition, aortic urotensin II contents increased in the aldosterone group, the calcification group, and the aldosterone +calcification group, especially in the aldosterone group and aldosterone +calcification group. The aortic aldosterone content in the calcified group was significantly higher than the controls.

Conclusions This study suggests that aldosterone is promoting factors contributing to vascular calcification, probably by stimulating inflammatory factors and urotensin II upregulation, in an autocrine/paracine manner.