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THE IMPACT OF VALSARTAN ON VASCULAR ENDOTHELIAL CELLS IN PLAQUE WITH APOE KNOCKOUT MICE

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Background To investigate whether Valsartan plays a role in vascular endothelial cells in plaque and hs-CRP with ApoE knockout mice, thus improving plaque stability.

Materials and Methods Animals were divided into control group (wild-type C57/BL mice), AS (atherosclerosis) group and the Valsartan group. Blood lipids and hs-CRP were measured in all animals. After 14 weeks, aortic specimens were given HE staining and immunohistochemical examination. CD34 expression of atherosclerosis lesions was measured by image analysis obtaining the IOD (integrated optical density) value.

Results HDL-C in the Valsartan group (0.78±0.56 mmol/l) was significantly lower than that in the control group

(1.24±0.09 mmol/l), p<0.01 hs-CRP in AS group (1.11±0.99, mg/l) and the Valsartan group (0.84±0.46, mg/l) was significantly higher than that in the control group (0.63±0.10, mg/l); hs-CRP in the Valsartan group was significantly lower than that in the AS model group, p<0.01. Plaque areas in AS group (22050.015084.63 μm^2) and the Valsartan group (8505.51±4388.27 μm^2) was significantly higher than that in the control group (2628.21±1224.64 μm^2); Plaque areas in the Valsartan group was significantly lower than that in the AS model group, p<0.01. Aortic wall of mice in the control group expressed no CD34, the expression of AS lesion IOD for the CD34 in Valsartan group of mice (22778.43±4007.19) was significantly reduced compared with that in AS group (126329.9±26261.98), p<0.01.

Conclusions Valsartan may inhibit plaque vascular endothelial cell proliferation and reduce the level of hs-CRP, thus improving plaque stability.