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Objectives To investigate the role of TGF-β1/Smads pathway in carotid artery remodelling in renovascular hypertensive rats and the prevention of enalapril and amlodipine.

Methods The renovascular hypertensive rats (RHR) developed by ‘two-kidney and one-clip’ method were treated consecutively with distilled water (model group, n=6), enalapril (10 mg/(kg/d), n=6), amlodipine (50 mg/(kg/d), n=6) for 6 weeks. Six sham-operated rats were used as controls. Carotid artery morphology and structural changes in the media were observed by HE staining, immunohistochemical staining and Masson staining. Media thickness (MT), lumen diameter (LD), media thickness and lumen diameter ratio (MT/LD) and collagen fibre area percentage of carotid arteries were measured. In addition, the immunohistochemical staining was applied to detect the expression of α-smooth muscle actin (α-SMA), proliferating cell nuclear antigen (PCNA), TGF-β1, p-Smad2/3 and Smad7.

Results MT, LD, MT/LD, α-SMA, PCNA and collagen fibre area percentage of carotid arteries in the model group were higher than those in the sham-operated group (p<0.01), and TGF-β1 and p-smad2/3 were significantly increased compared to sham-operated group, Smad7 was much lower in the model group (p<0.01). Single therapy of enalapril or amlodipine decreased MT, MT/LD and the protein expression of TGF-β1, p-Smad2/3, and increased the expression of Smad7. The combination treatment of enalapril and amlodipine was significantly better than that in single amlodipine group (p<0.05), but not in single enalapril group.

Conclusions In RHR, TGF-β1/Smads pathway may participate in the mechanism of carotid artery remodelling. The enalapril or amlodipine could attenuate carotid remodelling of RHR through the intervention in TGF-β1/Smads pathway. The combination of enalapril and amlodipine is better than amlodipine therapy.