Objective  To investigate the influence of pyridoxamine on the pre-iteration of rat vascular smooth muscle cells induced by Angiotensin (A) in vitro and its mechanism.

Methods  Primary VSMCs were cultivated from the thoracic aortas of spontaneously hypertensive rats, and which from the 3th to 5th generation at exponential phase of growth were selected for the experiments and induced by A (10^{-7} mol/l). The proliferation of VSMCs after intervention with pyridoxamine (0.1, 1 and 10 mmol/l) was determined by MTT. The levels of advanced glycation end-products in cellular supernatant was determined by enzyme-linked immunosorbent assay (ELISA) and the intracellular level of reactive oxygen species (ROS) was detected by flow cytometry. The mRNA expressions of RAGE, NF-κB-P65, NADPH oxidaseP47phox were detected by Real-time PCR.

Results  The proliferation of VSMCs induced by A was inhibited by pyridoxamine in dependent-concentration manner (0.1, 1 and 10 mmol/l). The cellular supernatant AGEs level in pyridoxamine (1 mmol/l, P1) and pyridoxamine (10 mmol/l, P10) group was significantly lower (p<0.001) as compared with A group. It was decreased more in P10 group than that in P1 group (p<0.001); the intracellular ROS level significantly was decreased in P1 and P10 group as compared with that in A group (p<0.001), and less in P10 group than that in P1 group (p<0.001); Compared with A group, The mRNA expressions of advanced glycation end-products receptor (RAGE), NF-κB-P65, NADPH oxidaseP47phox and MMP-9 mRNA in P1 and P10 group all were significantly lower (p<0.01), and they were much decreased in P10 group than that in P1 group (p<0.01).

Conclusions  The pyridoxamine inhibits the proliferation of VSMCs induced by A in dependent-concentration manner, and the potential mechanism of which pyridoxamine may reduce the expressions of RAGE by NF-κB pathway signals and inhibiting AGEs expressing and oxidative stress.