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EFFECT OF PYRIDOXAMINE AND TELMISARTAN ON TUBULAR EPITHELIAL CELLS PROLIFERATION AND ITS MECHANISM

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Objective The purpose of this study was to explore the protective effects of telmisartan and pyridoxamine on tubular epithelial cells (HK-2) in early renal damage.

Methods Cultured HK-2 cells were divided into HK-2 control, angiotensin II (10^{-6} mol/l) group (Ang II), telmisartan (10^{-6} mol/l, T) group, pyridoxamine group (1 mmol/l, P1), pyridoxamine group (10 mmol/l, P10), and T (10^{-6} mol/l)+P (10 mmol/l) group (T+P). Methyl thiazolyl tetrazolium (MTT) was used to measure for cell proliferation. Reactive oxygen species (ROS) generation in cellular supernatant was detected by flow cytometry. Real-time quantitative PCR was performed to measure the mRNA expression of transforming growth factor β 1 (TGF β 1) and receptor for advanced glycation end-products (RAGE).

Results The OD values of HK-2 were inhibited in Ang II ($p < 0.05$), 10 mmol/l P group and TP group ($p < 0.01$) after both 24 h and 48 h intervention. Synergy of inhibition showed in TP group after 48 h intervention ($p < 0.01$). The level of ROS was reduced in P1, P10 and TP group ($p < 0.01$). There was no statistically significant difference among the three groups ($p > 0.05$). Compared with the Ang II, mRNA expression of RAGE and TGF β 1 were decreased in T, P and TP group ($p < 0.05$). Synergy decreased of TGF β 1 expression was showed in TP group ($p < 0.01$).

Conclusion There is a synergistic effect of inhibiting HK-2 cells proliferation by combined use of telmisartan and

pyridoxamine. It may be related to reduce RAGE and TGF β expression and alleviate oxidative stress on early renal damage.