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Nogo-B may mediate the glomerular endothelial cell injury of hypertensive nephropathy by enhancing the inflammatory phenotype

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Objectives : Nogo-B is an endoplasmic reticulum resident protein, which is mostly expressed in vascular endothelial cells and smooth muscle cells. Previous studies have elucidated the relationship between hypertension and Nogo-B in endothelial cells, but whether Nogo-B is involved in glomerular endothelial cell injury in hypertensive nephropathy and its mechanism is not clear.
Methods : The localization of Nogo-B was proved by immunohistochemistry (IHC) and immunofluorescence (IF) staining in renal tissue of hypertensive patients, and the relationship between it and systolic blood pressure and UACR was analyzed. In animal experiments, IHC, IF, PAS, Masson, qRT-PCR and Western blot were employed to prove the localization of Nogo-B and analyze the relationship between its expression level and the degree of renal injury in Ang II-induced Nogo-B KO and control mice. In the vitro, the expression level of Nogo-B in endothelial cells (Ea.hy926) was knocked down by small interfering RNA (siRNA), and the correlation between Nogo-B expression and endothelial cell functions and phenotypes were explored.
Results : Nogo-B is expressed in glomerular endothelial cells and increased in hypertensive nephropathy. It is positively correlated with the patients' systolic blood pressure and UACR. In animal experiments, the renal pathological damage of Nogo-B KO mice treated with hypertension was mild and the biochemical indexes of renal function were improved. The expression of Nogo-B in endothelial cells is up-regulated in hypertensive environment, and the proliferation and tube formation of endothelial cells are increased. Knockdown of Nogo-B by siRNA inhibits the proliferation and tube formation, and reduces the inflammatory phenotype of endothelial cells in hypertensive environment.
Conclusions : Nogo-B is expressed in glomerular endothelial cells and may mediate the glomerular endothelial cells injury of hypertensive nephropathy by enhancing the inflammatory phenotype.