

Significance of fibronectin 1 expression in gestational hypertension and its effect on human umbilical vein endothelial cells

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Abstract

Abstract Objective To explore the role of fibronectin 1(FN1) in hypertensive disorder complicating pregnancy(HDCP) and human umbilical vein endothelial cells(HUVECs). **Methods** Plasma FN1 of 80 HDCP women and 40 healthy pregnancy were detected by ELISA and its correlation with the clinical data of HDCP were analyzed. Lentivirus vectors was transfected into HUVECs to induce the overexpression of FN1. The levels of proinflammatory cytokines TNF- α , IL-6 were determined by ELISA and the activity of MMP9 and MMP2 were detected by Gelatin zymography assay. Apoptosis rate was analyzed by TUNEL, and Ad-mCherry-GFP-LC3 was transfected to observe the autophagy. The expression of IKK- α , p-P65, nuclear P65, EMT marker proteins: α -SMA, E-Cadeherin, Bcl2, Bax, caspase-9, autophagy marker proteins:LC3II, ATG5 and BENC1were detected by western blot. **Results** Plasma FN1 in HDCP patients was high expressed and it was increased with the development of HDCP. Overexpression of FN1 increased the expression of TNF- α , IL-6, IKK- α , p-P65, nuclear P65, α -SMA and E-Cadeherin and the activity of MMP9 and MMP2. Overexpression of FN1 induced apoptosis, upregulated the expression of caspase-9, Bax, and downregulated Bcl-2. Overexpression of FN1 upregulated the expression of LC3II, ATG5 and BENC1, induced autophagosome formation and inhibited autophagic degradation. MHY1485 could activate mTOR, to reverse the induction of autophagy caused by overexpression of FN1. **Conclusion** Plasma FN1 was related to the pathogenesis and progress of HDCP. Overexpression of FN1 in HUVECs increased the inflammation, induced the degradation of ECM, EMT and apoptosis, induced autophagosome formation and inhibited autophagic degradation by inhibiting activation of mTOR.

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