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Paclitaxel ameliorates Palmitate-induced podocyte injury

Seung seob SON, Jeong suk KANG, Ah reum JOENG, *Eun young LEE

Internal Medicine, Soonchunhyang University Cheonan Hospital, Korea,South

Objectives : Diabetic Nephropathy has become an important health problem worldwide. Podocyte injury is relevant to diabetic nephropathy. Recently, it has been reported that paclitaxel has an anti-fibrosis and anti-inflammatory effects on kidney disease. However, the mechanism of paclitaxel on diabetic kidney disease is unknown. In this study, we investigated the effects of paclitaxel on palmitate-induced podocyte injury.

Methods : We used immortalized mouse podocytes for in vitro system. Palmitate showing increased levels in patients with diabetic mellitus used to induce diabetic mimic condition. Podocytes were divided into four groups; Bovine serum albumin, Palmitate, Palmitate+1nM paclitaxel, and Palmitate+5nM paclitaxel. The effects of paclitaxel were analyzed by western blot and real time-PCR.

Results : ER-stress marker, ATF-6α expression was significantly increased in Pamlitate-treated podocytes compared to control, which was decreased by paclitaxel treatment. Elevated Nox4 expression in podocytes stimulated by palmitate was ameliorated by paclitaxel. Paclitaxel also restored Nrf-2 down-regulated expression and up-regulated expression of inflammatory markers, TNF-α and MCP-1 in palmitate-treated podocytes. Increased expression of fibronectin and TGF-β1 molecules was restored by paclitaxel. Palmitate-induced podocyte apoptosis was ameliorated by paclitaxel by reducing the expression of BAX and caspase-3 levels.

Conclusions : These results suggest that paclitaxel has therapeutic effects on palmitate-induced podocyte injury by inhibiting fibrosis.

Keywords : Paclitaxel(Paclitaxel), Kidney (신장), Fibrosis(섬유화), Palmitate(지방산)