KSN 2019
Seoul, Korea · May 23 - 26

Abstract Type : Poster
Abstract Submission No. : PO-1061

Therapeutic Potential of Natural Compound 2, 3, 5, 4'-tetrahydroxystilbene-2-O-β-D glucoside in Focal Segmental Glomerulosclerosis

Uyanga Bayarsengee
Department of Internal Medicine, Khan-Uul secondary hospital, Mongolia

Objectives: Focal segmental glomerulosclerosis is the worst prognosis primary glomerular diseases with progressive glomerular scarring and a clinical presentation of nephrotic syndrome in children and adults. More than half of patients with FSGS complicate end-stage renal disease (ESRD). FSGS is defined by heavy proteinuria and edema in clinical findings, but entity diagnosed by sclerotic lesions which occur part (segmental) of the glomerular capillaries in a minority (focal) of glomeruli in histological findings. Oxidative stress, depleted antioxidant ability and inflammation play most critical role in the pathogenesis. 2, 3, 5, 4'-tetrahydroxystilbene-2-O-β-*D* glucoside (THSG) is strong anti-oxidant compound which is extracted from Chinese traditional herbal. In this study, we evaluated these nature antioxidant compounds renoprotective effect in a mouse model of AD-induced FSGS. Furthermore, we tried to elucidate the underlying mechanism.

Methods: In vivo study was intended for prevention. We established female BALB/C mice to induce FGSG model by Adriamycin 11mg/kg body weight, a single dose. We treated with THSG for 21 consecutive days at a daily dose of 120mg/kg body weight by oral gavage. The serum parameters, urine protein were measured and histopathology was examined. Antioxidant enzymes and oxidative stress damage were determined in all groups. The certain fibrotic and inflammatory genes mRNA were analyzed by qPCR. In vitro study, we used CRL1927 mouse mesangial cell lines and treatment with 20μg/ml THSG.

Results: We found potential effect of THSG in ameliorating proteinuria progression and improve renal function in FSGS mouse model. The mechanism involved in the renoprotective effect of THSG on the FSGS were mainly an activated Nrf2 dependent signaling pathway. THSG greatly reduced fibrosis and inflammation in renal tissue. THSG induced HO-1 expression through Nrf2 signaling pathway in vitro.

Conclusions: THSG ameliorated proteinuria progression and kidney injury in AD induced FSGS mouse model by stimulating antioxidant enzymes through Nrf2 dependent signaling pathway.