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Renoprotective Effects of Autotaxin Inhibitor in Folic Acid-induced Chronic Kidney Disease Mouse Model

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Objectives : Autotaxin (ATX) converts lysophosphatidylcholine into lysophosphatidic acid (LPA), promoting cell migration, proliferation, and fibrogenesis. Although ATX has been studied in kidneys, the therapeutic implications and molecular mechanisms of ATX inhibition in chronic kidney disease (CKD) remain unclear. Therefore, this study aimed to elucidate the therapeutic potential of ATX inhibition in CKD by investigating the antifibrotic effects of an ATX inhibitor (ATXi).
Methods : In this study, a folic acid (250 mg/kg IP)-induced CKD mouse model was used to evaluate the renoprotective effects of ATXi (30 mg/kg/day PO) for 21 days. Renal ATX expression, fibrosis, and lipid accumulation were assessed by Immunohistochemisty (IHC) and histological staining. Renal function (BUN, cystatin C), urine LPA levels (ELISA), mitochondrial structure (electron microscopy), and LPA receptor, Phosphoglycerate mutase family member 5 (PGAM5) expression (qPCR, IHC) were analyzed.
Results : BUN and cystatin C levels were elevated in the CKD group compared with controls but were markedly reduced by ATXi treatment, indicating improved renal function. Histological analysis showed increased tubular dilatation and interstitial fibrosis in CKD kidneys, both significantly attenuated by ATXi. Expression of α-SMA, COL1α1, and Ki67 was upregulated in CKD but decreased following ATXi treatment. Electron microscopy revealed fewer mitochondria with intact cristae in CKD kidneys which were restored by ATXi. Mitochondria damage maker, Phosphoglycerate mutase family member 5 (PGAM5) expression were increased in the CKD group and decreased in the ATXi-treated group. LPA1 and LPA6 mRNA expression and lipid accumulation were significantly increased in CKD, whereas ATXi suppressed lipid deposition and reduced LPA1 and LPA6 levels. These findings suggest that ATX inhibition ameliorates CKD and may serve as a therapeutic strategy for renal fibrosis.
Conclusions : This study provides evidence that ATXi improves fibrosis in CKD through the blockade of the LPA signaling pathway, suggesting that ATX inhibition may serve as a potential therapeutic strategy in renal fibrotic diseases.