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Alpha-1 Antitrypsin Protects Against Cisplatin-Induced Acute Kidney Injury by Restoring Redox and Mitochondrial Homeostasis

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Objectives : Cisplatin is an effective chemotherapeutic agent, but yet its clinical utility is limited by dose-dependent nephrotoxicity. Alpha-1 antitrypsin (AAT) has cytoprotective, anti-inflammatory, and anti-apoptotic properties, but its therapeutic potential in cisplatin-induced acute kidney injury (AKI) remains unclear.
Methods : A murine cisplatin–AKI model was used to evaluate whether AAT (80 mg/kg) ameliorates renal injury. Renal function, oxidative stress, NADPH oxidase (NOX) isoforms, mitochondrial metabolism, inflammatory mediators, apoptosis, and fibrosis-related markers were assessed using biochemical, histological, immunohistochemical, and Western blot analyses
Results : Cisplatin markedly impaired renal function,Cisplatin markedly impaired renal function and inducedinduced tubular injury , and increased NGAL expression; meanwhile, whereas AAT significantly reversed these changes. Cisplatin also induced severe oxidative stress and disrupted NOXthe isoformbalance balanceof NOX isoforms, whereas; AAT restored redox homeostasis. Cisplatin induced maladaptive mitochondrial changes, with upregulated CPT1A/PDK4 upregulation and suppressed CPT2, UCP3, PGC1α, and DRP1, inducing maladaptive mitochondrial changes suppression, indicating impaired β-oxidation and defective mitochondrial dynamics. ; AAT reversed these alterations, and restoring normalized mitochondrial metabolismc pathways. IL-1β, IL-6R, OPN, and F4/80 expression, recovery of the Bax/Bcl-2 ratio, and MAPK activation were reduced, indicating decreased Inflammation inflammation and apoptosis were reduced, as indicated by lower expression of IL-1β, IL-6R, OPN, and F4/80, recovery of the Bax/Bcl-2 ratio, and decreased MAPK activation. P; profibrotic markers were also reduced.
Conclusions : AAT confers multifaceted protection against cisplatin-induced AKI by restoring redox balance, mitochondrial homeostasis, and inflammatory and apoptotic signaling. These findings support AAT as a promising therapeutic strategy agent for preventing cisplatin nephrotoxicity.