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Protective Effects of Urine-Derived Stem Cells on Adriamycin-Induced Podocyte Apoptosis in Chronic Kidney Disease

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Objectives : Podocyte injury and apoptosis are central mechanisms driving progressive glomerular dysfunction in chronic kidney disease (CKD). Urine-derived stem cells (UDSCs) are kidney-proximal multipotent cells with reported renoprotective properties; however, their effects on adriamycin (ADR)-induced podocyte apoptosis and the impact of dosing frequency remain incompletely characterized.
Methods : In vitro, conditionally immortalized human podocytes (CIHP-1) were exposed to ADR (0.1–2.0 μM; up to 48 h) to establish a podocyte apoptosis model. Human UDSCs were isolated from urine, expanded, and characterized. Indirect transwell co-culture was used to evaluate the renoprotective effects of UDSCs. In vivo, BALB/c mice received ADR (10 mg/kg, intravenous) on day 0; UDSCs (1×10^6 cells/mouse) were administered starting on day 7 as either a single dose or three consecutive daily doses. Primary endpoints included urine albumin-to-creatinine ratio (ACR), histology, transmission electron microscopy (TEM), immunostaining, and bulk renal transcriptomics.
Results : ADR induced sequential activation of NF-κB, NLRP3/IL-1β, p53, and caspase-3 in CIHP-1 podocytes. UDSC transwell co-culture attenuated NF-κB, NLRP3, and IL-1β induction, reduced caspase-3 activation and cytochrome-c release, and preserved nephrin expression and F-actin architecture. In vivo, ADR increased ACR and caused glomerular injury with foot-process effacement. Both single and repeated UDSC dosing significantly reduced ACR and ameliorated histologic damage. Compared with a single dose, repeated dosing produced broader transcriptomic suppression of ferroptosis-, ribosome biogenesis-, and senescence/SASP-associated pathways.
Conclusions : UDSCs attenuate ADR-induced podocyte inflammation and apoptosis and promote structural and functional renal recovery. Dosing frequency modulates transcriptomic responses, with repeated dosing eliciting broader anti-senescence signatures. These findings support further mechanistic investigation and translational development of UDSC-based therapy for CKD.