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The Protective Effect of Peritoneal Dialysate-derived Mesenchymal Stem Cells on Peritoneal Fibrosis by Inhibiting Oxidative Stress

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Objectives : Mesenchymal stem cells (MSCs) exert regenerative and anti-fibrotic effects largely through paracrine actions, including inhibition of epithelial-to-mesenchymal transition (EMT). The EMT of mesothelial cells (MCs) is an early mechanism of peritoneal dysfunction during peritoneal dialysis (PD). We investigated whether peritoneal dialysate-derived MSCs (PD-MSCs) suppress TGFβ-induced EMT in human peritoneal mesothelial cells (HPMCs) and evaluated its mechanism.
Methods : PD-MSCs were isolated from the dialysate of early-stage PD patients and characterized by MSC markers expression (real-time PCR, FACS) and trilineage differentiation. EMT was evaluated by changes in morphology and epithelial/mesenchymal markers. Oxidative stress was assessed by DCF-DA and MitoSox staining. Antioxidant enzymes and anti-fibrotic proteins in PD-MSCs were compared with adipose (AD)-derived MSCs and bone marrow (BM)-derived MSCs. An animal model of PD was established by daily infusion of 4.25% glucose-based dialysate with methylglyoxal for 3 weeks via intraperitoneal catheter. PD-MSCs ($1.0x10^6$ cells, i.p. or i.v.) were injected at 14 days, and peritoneal tissue was isolated 7 days after PD-MSC injection. Markers of oxidative stress and anti-fibrotic were evaluated with peritoneal equilibrium test and histologic analysis.
Results : PD-MSCs expressed characteristic MSC markers and demonstrated trilineage differentiation. PD-MSCs inhibited TGFβ-induced EMT (increased E-cadherin; decreased Fibronectin and αSMA) and reduced ROS generation. PD-MSCs exhibited higher expression of catalase, GPx, and SOD, and higher HGF and BMP-7, than AD-/BM-MSCs. PD-MSC also restored TGF-β-induced suppression of antioxidant enzymes and anti-fibrotic factors in HPMCs. In vivo, PD-MSC-treated rats had a higher D_2/D_0 glucose and a lower D_2/P_2 creatinine ratio. PD-MSCs alleviated EMT and peritoneal fibrosis with an amelioration of oxidative stress and NLRP3 inflammasome, as evidenced by decreased expression of nitrotyrosine, along with increased SOD.
Conclusions : PD-MSCs derived from early-stage PD patients represent a clinically accessible autologous cell source that mitigates EMT and oxidative stress while restoring anti-fibrotic factors, supporting their potential to preserve peritoneal membrane function in PD patients.