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Human Dental Pulp Stem Cells Attenuate Streptozotocin-Induced Parotid Gland Injury in Rats

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Abstract : Background: Diabetes mellitus causes severe deteriorations of almost all the organs and systems of the body, as well as significant damage to the oral cavity. The oral changes are mainly related to salivary glands dysfunction characterized by hyposalivation and xerostomia, which significantly reduce diabetic patients' quality of life. Human dental pulp stem cells represent a promising source for cell-based therapies, owing to their easy, minimally invasive surgical access, and high proliferative capacity. It was reported that the trophic support mediated by dental pulp stem cells can rescue the functional and structural alterations of damaged salivary glands. However, potential differentiation and paracrine effects of human dental pulp stem cells in diabetic-induced parotid gland damage have not been previously investigated. Our study aimed to investigate the therapeutic effects of intravenous transplantation of human dental pulp stem cells (hDPSCs) on parotid gland injury in a rat model of streptozotocin (STZ)-induced type 1 diabetes. Methods: Thirty Sprague-Dawley male rats were randomly categorised into three groups: control, diabetic (STZ), and transplanted (STZ+hDPSCs). hDPSCs or vehicle was injected into the tail vein 7 days after STZ injection. The fasting blood glucose levels were monitored weekly. A glucose tolerance test was performed, and the parotid gland weight, salivary flow rate, oxidative stress indices, parotid gland histology, and caspase-3, vascular endothelial growth factor (VEGF), and proliferating cell nuclear antigen (PCNA) expression in parotid tissues were assessed 28 days post-transplantation. Results: Transplantation of hDPSCs downregulated blood glucose, improved the salivary flow rate, and reduced oxidative stress. The cells migrated to, survived, and differentiated into acinar, ductal, and myoepithelial cells in the STZ-injured parotid gland. Moreover, they downregulated the expression of caspase-3 and upregulated the expression of VEGF and PCNA, likely exerting pro-angiogenetic and antiapoptotic effects and promoting endogenous regeneration. In addition, the transplanted cells enhanced the parotid nitric oxide (NO) -tetrahydrobiopterin (BH4) pathway. Conclusions: Our results show that hDPSCs can migrate to and survive within the STZ-injured parotid gland, where they prevent its functional and morphological damage by restoring normal glucose levels, differentiating into parotid cell populations, and stimulating paracrine-mediated regeneration. Thus, hDPSCs may have therapeutic potential in the treatment of diabetes-induced parotid gland injury.

Keywords : dental pulp stem cells, diabetes, streptozotocin, parotid gland
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