MSC transplantation: a promising therapeutic strategy to manage the onset and progression of diabetic nephropathy

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Resumen

Currently, one of the main threats to public health is diabetes mellitus. Its most detrimental complication is diabetic nephropathy (DN), a clinical syndrome associated with kidney damage and an increased risk of cardiovascular disease. Irrespective of the type of diabetes, DN follows a well-known temporal course. The earliest detectable signs are microalbuminuria and histopathological changes including extracellular matrix deposition, glomerular basement membrane thickening, glomerular and mesangial expansion. Later on macroalbuminuria appears, followed by a progressive decline in glomerular filtration rate and the loss of glomerular podocytes, tubulointerstitial fibrosis, glomerulosclerosis and arteriolar hyalinosis. Tight glycemic and hypertension controls remain the key factors for preventing or arresting the progression of DN. Nevertheless, despite considerable educational effort to control the disease, a significant number of patients not only develop DN, but also progress to chronic kidney disease. Therefore, the availability of a strategy aimed to prevent, delay or revert DN would be highly desirable.

In this article, we review the pathophysiological features of DN and the therapeutic mechanisms of multipotent mesenchymal stromal cells, also referred to as mesenchymal stem cells (MSCs). The perfect match between them, together with encouraging pre-clinical data available, allow us to support the notion that MSC transplantation is a promising therapeutic strategy to manage DN onset and progression, not only because of the safety of this procedure, but mainly because of the renoprotective potential of MSCs.

Palabras clave

Palabras clave de autor: Regenerative medicine; Diabetes mellitus; Diabetic nephropathy; Multipotent mesenchymal stromal cells; Mesenchymal stem cells
KeyWords Plus: MESENCHYMAL STEM-CELLS; ACUTE-RENAL-FAILURE; MARROW-DERIVED CELLS; MULTIPOTENT STROMAL CELLS; ACUTE KIDNEY INJURY; BONE-MARROW; OXIDATIVE STRESS; MESANGIAL CELLS; PARACRINE MECHANISMS; METABOLIC SYNDROME