

An Editorial on Adipose Stem Cell Therapy and Chronic Pancreatitis

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Editorial

Chronic pancreatitis (CP) is a long-term inflammation of the pancreas that causes it to lose its natural shape and function. It is challenging to create an effective treatment for CP due to the lack of well-defined criteria for early diagnosis and the disease's multi-factorial nature. Stem cell therapy has emerged as a viable treatment for autoimmune illnesses, and it could be used to treat CP. The infusion of adipose-derived mesenchymal stem cells (ASCs) into an experimental mouse model of CP prevented the progression of CP, dramatically reduced pancreatic damage, and reduced fibrosis and cell death, according to a study published in this issue of Molecular Therapy. The infused ASCs most likely developed into acinar-like cells that inhibited inflammation and fibrosis, reducing pancreatic damage, according to the investigators. The study's findings could have a significant impact on people with CP who now lack appropriate treatment alternatives.

"A pathologic fibro-inflammatory syndrome of the pancreas in individuals with genetic, environmental, and/or other risk factors who develop persistent pathologic reactions to parenchymal injury or stress," according to the definition of CP. Due to recurring episodes of acute pancreatitis and chronic inflammation, CP is defined by persistent and irreversible inflammation of the pancreas, which leads to a progressive loss of exocrine and endocrine function. The pathophysiology of CP is characterised by a necrosis-fibrosis loop caused by severe acute pancreatitis, followed by inflammatory cell activation and recruitment, and activation of pancreatic stellate cells (PSCs), myofibroblast-like cells found in the exocrine pancreas. PSCs move to injury sites after activation and contribute in the regeneration process. The production of oxidative stress causes acinar cell necrosis, inflammation, and fibrosis during this phase. Finally, ductal dysfunction causes protein plugs to develop and upstream ductal blockage. Due to its multimodal characteristics, the main therapeutic options for CP have thus far centred on treating the accompanying symptoms and pain management.

In a mouse model of CP, a single low-dose ASC infusion slows disease development and protects the injured pancreas. The diminution of collagen deposition and α -SMA (smooth muscle actin) expression, which are critical factors to the fibrosis process, demonstrated that systemic injection of ASCs inhibited pancreatic fibrosis. When compared to untreated mice, additional results demonstrated reduced inflammation, as seen by reduced infiltration by inflammatory macrophages, and continued protection against pancreatic cell death. A single low-dose ASC infusion slows disease development and

protects the damaged pancreas in a mouse model of CP. The diminution of collagen deposition and α -SMA (smooth muscle actin) expression, both critical components to the fibrosis process, demonstrated that systemic injection of ASCs inhibited pancreatic fibrosis. When compared to untreated animals, additional results demonstrated reduced inflammation, as seen by lower inflammatory macrophage infiltration, and prolonged protection against pancreatic cell death.

Because of their anti-inflammatory qualities, MSCs have become a popular cell source for the treatment of chronic invalidating diseases (such as diabetes, liver diseases, and pancreas and kidney disorders). ASCs have emerged as a promising emerging therapy platform for CP due to their ease of collection and availability. Their ability to specifically homing to injury areas and local differentiation into acinar-like cells makes them an appealing therapeutic tool, but the present study does not investigate whether ASCs have direct regenerative function. Furthermore, the likelihood of negative outcomes creates a possible safety risk. ASCs, like MSCs, may provide a tumorigenic risk, owing to their prolonged in vitro proliferation, which increases the chance of chromosomal instability and malignant transformation [1-6].

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