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Poster Presentation

Secondary Demyelination in Spinal Cord Injury

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Abstract

The pathology of spinal cord injury (SCI) comprises multiple processes characterized by extensive cell death and glial scar formation, which together limits axonal regeneration. In particular, variations in chronic demyelination of the lesion have been found to be largely independent of injury intensity, suggesting a secondary pathologic origin. This chronic demyelination results in progressive tissue degeneration and constitutes a significant component of the pathology in many cases of SCI. Widespread demyelination is resulted from oligodendrocyte apoptosis which has been seen after ischemic or traumatic injury to the central nervous system which also could be found within remote degenerating fiber tracts. Both secondary degeneration at the site of SCI and the chronic demyelination of tracts away from the injury appear to be due in part to apoptosis. Furthermore, secondary pathology is coincident with dense invasion of the lesion by macrophages and their phagocytosis of the membranous debris remaining from the initial hemorrhagic necrosis. Prolonged demyelination enhances axonal vulnerability to degeneration, and is thereby thought to contribute to the axonal degeneration that underlies the permanent functional losses associated with SCI. Recovery of conduction in demyelinated axons may permit recovery of function and can be mediated by several mechanisms including remyelination by oligodendrocytes and Schwann cells transplantation alone or in co-transplantation with other cellular types such as bone marrow mesenchymal cells or neural stem cells. In addition, combination cellular transplantation strategy with the complementary administration of neuro-protective agents/growth factors, improves the effect of cellular transplantation after SCI.

Keywords: Demyelination, Apoptosis, Spinal Cord Injury, Transplantation.

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