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Pathophysiology, mechanisms and applications of mesenchymal stem cells for the treatment of spinal cord injury



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ABSTRACT

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Keywords: Spinal cord injury Transplantation Stem cells Bone marrow Mesenchymal cell Spinal Cord Injury (SCI) is a serious devastating condition associated to the high chances of morbidity and mortality. It involves a primary and a secondary injury, former cause damages to both lower and upper motor neurones and disrupts sensory, motor and autonomic functions while the latter involves various stages of molecular plus cellular incidents which elaborate the original injury. In the treatment of SCI, stem cells possess a good therapeutic potential. Bone marrow, adipose tissue, placenta, amniotic fluid and umbilical cord are the good sources for mesenchymal stem cells. This review article shows the uses of bone marrow derived mesenchymal cells in the treatment of acute and chronic case of SCI and its future scope.

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Contents

1. Introduction

Spinal cord injury (SCI) is well-defined as an injury or lesion that results due to the dysfunction imposed on the spinal cord thereby compromising the major functions of spinal cord viz; sensory, motor, autonomic, and reflex, either completely or partially due to trauma or disease or degeneration (non-trauma) [1,2]. Global incidence for SCI is estimated to be in 40–80 persons in a million population. Amongst these, 90% occurs due to trauma but the occurrence appears to be growing recently for the non-traumatic SCI [3]. The incidence of SCI level is shown in Fig. 1. Cervical region accounts for about 55% of acute SCI, and the rest

* Corresponding author at: Shobhaben Pratapbhai Patel School of Pharmacy and Technology Management, SVKM'S NMIMS, Vile Parle (W), Mumbai, India. *E-mail address:* shendepravin94@gmail.com (P. Shende). three regions i.e. thoracic, thoracolumbar and lumbosacral, each report for about 15% of SCI [4].

The prevalence of SCI is about 54 cases per million population, as per census data in USA, thereby indicating about 17,000 new cases being re-counted each year. Male accounts for 80% of these incidences. When compared to cases of 1970s, the age for incidence has increased from then 29 years to 42 years now, however, the length of stay in hospital has decreased from 24 days to 11 days. When neurological category at the time of discharge of SCI patients is taken into consideration since 2010, 45% accounts for incomplete tetraplegia followed by 21.3%, 20% and 13.3% accounting for incomplete paraplegia, complete paraplegia and incomplete paraplegia respectively. Only 0.4% of SCI cases experience complete recovery on discharge from hospital [5].

SCI is the most serious complication that usually lead to neuronal death and axonal damage resulting in dyskinesia or somatosensory loss [6]. SCI interrupts the nerve connections



Fig. 1. Level of Injury in Adult SCI.

between the brain and the body, and results in paralysis. The pathology of SCI is determined by both the primary mechanical injury and the secondary processes that prevails around hours and days after injury, includes ischemia, anoxia, inflammation, cavity and glial scar formations [7]. Spontaneous regeneration of neural tissue and the efficacy of therapies used for regeneration of axons is also compromised during secondary tissue damages of SCI [8].

Usually, the spinal cord axonal regeneration is subsidized by various factors, some of which includes diminution on inherent growth potential of CNS neurons, damaged CNS myelin that generate inhibitory signals, local astrocytes in reaction to the external stimuli forming glial scars and the absence of nerve growth factors and neurotrophic factors [9]. SCI is a serious damaging condition where patient experience significant sensory and functional loss, along with financial, social and emotional problems. SCI patients are at bigger risk of cardiovascular complexities, deep venous thrombosis, bed sores, osteoporosis, neuropathic pain and autonomic dysreflexia. SCI involves various complexities involved in its mechanism along with the failure on



Fig. 2. Mechanisms of damage after SCI in the cellular level.

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