The Systemic Inflammation after Spinal Cord Injury

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Abstract

Spinal cord injury (SCI) actuates to complex cellular and molecular interactions within the central nervous system in a heave to repair the initial tissue damage. The pathophysiology of acute spinal cord injury (SCI) involves primary and secondary mechanisms. Neuroinflammation is an important secondary injury process in SCI. The local inflammatory microenvironment within the injured spinal cord is a collection of degenerating neurons, damaged endothelial cells, degraded myelin sheath, and this microenvironment produces various kinds of pro-inflammatory mediators. There are many other factors such as dysregulation of the neuroendocrine system and changed neuroimmune regulation that important determinant of the onset and progression of post-SCI systemic inflammation. Epidemiological analyses have unfolded a functional link between systemic inflammation and pathogenesis of post-injury complications. On the other hand cognitive impairment is associated with extensive cerebral inflammation after SCI. SCI triggers systemic inflammatory responses marked by increased circulation of immune cells and pro-inflammatory mediators, which result in the permeation of inflammatory cells into secondary organs and durability of an inflammatory microenvironment that chip in organ dysfunction.

Keywords: Spinal cord injury, Inflammation, Neuroendocrine

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