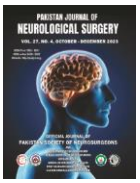

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EDITORIAL

Dear Readers

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Targeting cytokine- therapy to alleviate neuropathic pain caused by the spinal damage

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Damage to the spinal cord that results in paralysis and varied degrees of motor and/or sensory abnormalities is known as spinal cord injury (SCI). Both nociceptive and neuropathic chronic pain are prevalent and worse quality of life is a result of both. SCI has a complicated pathophysiology. Following the initial physical damage to the spinal cord, which may result in vascular rupture, bleeding, mechanical destruction to neural structures, and necrosis, a number of biomolecular cascades are activated, causing secondary harm. A pro-inflammatory condition has been linked to neuropathic pain. Neuroinflammation is a prominent factor in subsequent damage following severe SCI. Many clinical investigations have called into question the efficacy of existing treatments for neuropathic pain following SCI due to life-threatening adverse effects such as addiction. Growing data shows that chronic inflammatory responses following primary SCI cause an imbalance between anti- and pro-inflammation, leading in the etiology and maintenance of neuropathic pain. Interferons (IFNs) play important roles in the pathology as well as they are beneficial for the nervous system. Allison et al. examined the potential of an anti-inflammatory intervention for treating neuropathic pain after SCI. They concluded that changes in proinflammatory cytokines (interleukins -2) and interferons (IFN-γ) were responsible for the shift in sensory neuropathic pain. They strongly supported the concept of targeting inflammation as a means of treating neuropathic pain in SCI with following a mechanism related to reducing proinflammatory cytokines and PGE 2(Prostaglandin E2).

Iwatsuki et al. evaluated the effectiveness of anti-tumor necrosis factor-α medication in promoting functional recovery and reducing hypersensitivity following peripheral nerve crush damage. The anti-TNF medication lowered interleukin-6 and monocyte chemotactic and activating factor-1 expression in the crushed sciatic nerve. These results show that anti-TNF medications can improve functional recovery while also decreasing hypersensitivity following nerve compression. Park et al. studied long-term local immunomodulation utilizing lentiviruses expressing the anti-inflammatory cytokine IL-10 or IL-4 and supplied via multichannel bridges. Anti-inflammatory cytokines were thought to modify the pronociceptive inflammatory niche and stimulate axonal regeneration, resulting in neuropathic pain reduction. It is concluded that immunomodulatory strategies target several barriers to reduce secondary inflammation and neuropathic pain following SCI. It has been reported that IFN-α can be a viable target for medication repurposing. Monoclonal antibodies against IFN-α or its receptor might cure acute brain or spinal cord damage. Also, other pro inflammatory (Interleukins 1β/6/17) and anti-inflammatory (interleukins 4/10 & TGF β) are also under investigations. Conversely, depending on the concentration at which they are released and whether or not IFN or its receptors are targeted—either by neutralizing antibody administration or genetic deletion of either mediator or receptor—interferons may have conflicting effects on the injured central nervous system. Genetic models have shown either favorable or negative outcomes, despite the fact that IFN-γ may improve the prognosis of severe spinal cord injuries. A thorough investigation into the administration of IFN-β and IFN-γ for brain trauma and spinal cord injuries is necessary, since the precise treatment and administration remain to be validated.

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