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Letter to Editor **Journal of MAR Neurology (Volume 3 Issue 4)**

Nervous System, Inflammation and Glial Scar

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This letter to editor is resulted of a search for information about nervous system, inflammation and glial scar. In this way, pubmed and Google scholar were used to access publications concerned this subject.

According to the literature, inflammation on nervous system may occur as a result of a traumatic, vascular, infectious or autoimmune event.

Traumatic injuries to the brain or spinal cord, for example may cause tissue damage by at least three mechanisms: mechanical disruption of neurons, biochemical or metabolic changes and through reactive inflammatory or gliotic changes[2]. The direct or mechanical impact to the nervous tissue results in a primary lesion. This lesion produces activation of the immune system in consequence of the trauma leading to the development of the inflammatory process or secondary lesion **[**4,5**]**.

Strategies have been shown trying to solve these issues and they consider: treatments immediately following an accident (limiting initial degeneration and treating inflammation) and long-term treatments (stimulating axonal growth, promoting new growth through substrate or guidance molecules, blocking molecules that inhibit regeneration, supplying new cells to replace lost ones and building bridges to span the 5 lesion cavity.

Although different experimental animal models that simulate the traumatic lesion to the nervous system seen in human have been described in the current scientific literature, some experiments have estimated

the time-dependent repercussion of hemodynamic parameters on sensory and motor activities and systemic consequences due to the **[**6**]** injury.

Propagation of damage is a common occurrence after any nervous system insult and the lesion not only involves primary degeneration of the directly injured neurons, but also initiates inflammation and a selfdestructive process that leads to secondary lesion[3]. A great deal of research has been to limiting the extent of the secondary lesion and thereby improving functional recovery from nervous system injury. All forms of nervous system inflammation would do more harm than good and, hence, the less immune natural intervention the better. So, evidences indicate that some forms of immune-system 7 modulation may help to protect or restore the nervous function and its integrity.

The problem is that the inflammation process may result in scar formation on nervous tissue. Then, glial scar is considered to be a tertiary lesion and behaves as the most 4 important inhibitor factor to neuroregeneration . The injury induces a complex cascade of events of inflammatory and pathological processes, culminating in formation of a scar in the nervous tissue **[**[1]**]**.

On the one hand, if it is not possible to control the inflammatory process at the beginning, immune-modulation strategy has to be considered to stop the destruction of the nervous tissue. One possibility could be through the control of pro-inflammatory cytokines levels and how they act. On the other hand, if the inflammation has been established, the next step certainly will be the prevention of the glial scar. Stopping the development of the inflammation and/or controlling the formation of the scar, a functional recovery will be possible. In addition, the use of steam cells or neuronal grown factors in an appropriate environment into the nervous tissue will create an interesting possibility to neuroregneration and the function's recovery **[**4,5**]**

Therefore, to bring back the function control after an injury on the nervous system inflammatory-modulation and glial scar prevention are considered mandatory.

Reference

1. Bradbury EJ, Burnside ER. Moving beyond the glial scar for spinal cord repair: review article. Nature Communications 2019;10:3879. https://doi.org/10.1038/s41467-019-11707-7 (www.nature.com/naturecommunications)

2. Faden AI. "Experimental neurobiology of central nervous system trauma". Crit Rev Neurobiol 1993;7(3-4):175-186

3. Hauben E, Agranov E, Gothilf A, Nuevo U, Cohen A, Smirnov I, Steinman L, Schwartz M.

"Posttraumatic therapeutic vaccination with modified myelin selfantigen prevents complete paralysis while avoiding autoimmune disease". J Clin Invest. 2001;108(4):591-599

4. Leal Filho MB. "Spinal cord injury: from inflammation to glial scar". Surg Neurol Int 2011;2:112. doi: 10.4103/2152-7806.83732. (www.surgicalneurologyint.com)

5. Leal Filho MB. Spinal Cord injury and potential anti-inflammatory treatment: technical report written in 2006 and published in 2014. doi: 10.13140/2.1.3788.4160. https://www.researchgate.net/publication/265846507

6. Leal Filho MB, Morandin RC, de Almeida AR, Cambiucci EC, Metze K, Borges G, Gontijo AJR. "Hemodynamic parameters and neurognic pulmonary edema following spinal cord injury: an experimental model". Arq Neuropsiquiatr 2005;63(4):990-996

7. Schwartz M, Moalem G, Leibowitz-Amit R, Cohen IR. "Innate and adaptive immune responses can be beneficial for CNS repair". Trends Neurosci. 1999;22(7):295-299