Pain in Neurological Patients: A Perspective

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Pain is a leading cause of disability and a major medical and social problem [1]. It is defined by the International Association for the study of Pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage [2]. Thus, it is subjective and may exist even in the absence of tissue damage or any likely pathophysiological cause. Actually, the pain is not only a sensation, but a complex phenomenon which involve emotions and other associated components, thus should be considered according to a biopsychosocial model.

The neurological diseases that most commonly occur with pain are the stroke, the multiple sclerosis and the spinal cord injury [3]. Central neuropathic pain is pain caused by a lesion or disease of the central somatosensory nervous system [4]. This concept should not be confused with central sensitization from chronic peripheral neuropathic pain generators, which results in reversible plastic changes of central nociceptive pathways by up-regulation or wind-up phenomena [3].

Although chronic pain is common in patients with central nervous system diseases, it is usually not central neuropathic pain, but rather musculoskeletal nociceptive pain [5]. For instance, 30 to 40% of stroke patients with arm paresis will present musculoskeletal shoulder pain [6,7]. Neurological patients with gait disorders could also experience low back pain or lower limb pain, in part as a result of biomechanical changes and muscle imbalance. In spinal cord injury (SCI) patients, musculoskeletal pain is also predominant (comprising about 50 to 70% of the patients), but neuropathic pain is usually more intense and incapacitating [8,9]. To be classified as central neuropathic pain, it must be in the body regions affected by the central nervous system disease, although it does not necessarily have to involve the entire affected region [4].

In neurological patients, it is a challenge to distinguish and characterize pain, especially in those with sensory changes, as the pain descriptors are usually vague and the location is poorly defined [3].

The onset of pain is highly variable. It can be a presenting feature (commonly in multiple sclerosis) or it may occur during the acute phase of the neurological disease, but there is usually a delay of weeks to months [10,11]. In SCI, most patients only develop below-level pain after more than 2 years from the onset of the disease [8,9,12].

The interest of correctly diagnosing and classifying the type of pain is because it requires different therapeutic approaches. The treatment of pain in neurological patients is a challenge and should be multimodal, on one side because they may present different types of pain at different locations and on the other hand because patient's cognitive and neuromotor deficits may limit the tolerance to some drugs side effects and the capacity to integrate an intensive rehabilitation program. Functional restoration seems to be the cornerstone of the chronic pain management strategy, not only to improve pain control but also patient's quality of life and participation in family, social, vocational and professional contexts.

Bibliography

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