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Mini Review

Clinical Signs and Symptoms after Stroke: Who´s to Blame?

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Mini-Review

If we consider the clinical evolution of stroke since its onset, it can be quite obvious that Peripheral Nerves (PN) present an alteration in the ability to transport impulses, due to the continuity of the nervous system concerning the upper and lower motor neurones. Most studies and clinical approaches focus on the relation between the corticospinal tract, among others, to find an explanation for the movement and sensibility disorders which, at the end, determine functionality, independence and even longevity in patients after stroke [1], basing its rationale upon the relearning neuroscience perspective and the normal functioning of the Central Nervous System (CNS) as almost the only prerequisite for motor learning. There is ongoing evidence about PN disorders since the very acute stage of stroke and some even suggest that the PN alterations might be responsible for the muscle wasting that determines patients' ability to manage in daily life [2], not only in the paretic side, in which it was found a morphological degeneration that might be responsible for the altered impulse transport [3], but also entrapment neuropathies in both, the affected and contralateral side [4], suggesting different mechanisms that might be responsible [5], as the origin of this alterations remains uncertain so far, probably due to the multiple factors that might have an influence on PN physiological behaviour. Some studies have found, as an addition to morphological changes in stroke survivors' PN, a correlation between lack of muscle activity and altered compound muscle action potentials [6], suggesting a more peripheral mechanism. Also, it has even been stated that Carpal Tunnel Syndrome (CTS) clinical presentation found in the non-paretic limb of stroke survivors might be due to the overuse of canes [7,8].

Focusing on the immobility process associated with stroke, not only in relation to sedentary behaviour, but also taking into account every joint that is not being moved, it makes sense to think that the PN connective tissues might be responsible for the morphological changes found, for instance, in the Cross Sectional Area (CSA) of the median nerve in stroke patients and the fact that this changes are bigger as time goes by without moving [9]. The CSA changes are a parameter that is being used in other neurological pathologies such as Guillain-Barré, in order to target recovery processes from acute inflammation and its effect on PN [10]. When we consider specific clinical signs such as pain, it has been found that after experimental middle cerebral artery occlusion, some of the wide cited post stroke pain clinical features, might be due to PN disorders that has a determining influence on spinal cord hyperexcitability in pain syndromes [11]. Going deeper into the neurophysiology of entrapment neuropathies as an isolated clinical feature, there's experimental evidence of the ongoing degenerating process that goes on a nerve that is not being moved [12], and the immune interactions associated with this processes, concerning inflammation, both at the site of the entrapment and in the dorsal root ganglia, which can be an explanation for the widespread symptoms found in CTS or sciatica, not only concerning pain but also altered sensation, muscle contraction or coordination. A sustained inflammatory state has a deep impact on PN repair systems and it has been even suggested an anti-inflammatory drug approach for nerve reparation [13]. The immune system has a determining role in the pathogenesis of stroke and though it contributes in a first moment to contain the main aggression. It has been found that inflammatory cytokines such as FNT-α were found post mortem even 15 months after the onset, showing that this inflammatory process increases long-term damage to the brain [14]. Concerning this biological and mechanical environment, it makes sense to think that part of the alterations of the PN found in stroke patients might as well have a peripheral contribution that leads to local nerve inflammation, axonal degeneration and demyelination [15]. If we open the door to this possibility, a part of the solutions concerning clinical aspects in stroke patients, should be seen as neuropathic features and treated in accordance with it. Clinical findings among the stroke population that has an impact on their not only physical, but also psychological and social interactions, concerning body awareness and perception [16], might have a component that comes from altered PN, in what should be considered a secondary problem, that we can hypothesize that is likely to have an impact on the main one and that, due to the neural physiology, can get worse as times goes by with no joint active movement or muscle activity to protect the nerve from compression and ischemic damage.

In addition, is fair to say that there is no precise explanation of the changes that we can find in performance in neurological patients in different environments [17] and how stress affect their body movements. We do know about the biologic interdependence of stress and the Hypothalamic–Pituitary–Adrenal (HPA) axis and stroke outcome [18] or the crucial paper of peripheral immune response and inflammation [19], underlining the hypothesis of another physiological harm to the PN derived from the neurological lesion in stroke, as it happens with muscle degeneration [18]. In summary, if we want to fully understand pain, movement and perceptual behaviours in stroke survivors, it cannot be overseen the mechanical and physiological influence that PN disorders have on them [20]. This features, together with the inflammatory events also present in other neurological conditions like Multiple Sclerosis (MS) [21], should make us include the knowledge about PN fibers in our clinical reasoning in relation to the pathogenesis of spasticity, post-stroke pain, motor, sensory or autonomic alterations frequently seen in the clinical setting.

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