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Review article

Symptomatology and pathogenesis of different types of pain in multiple sclerosis

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ABSTRACT

Multiple sclerosis (MS) is a progressive disease of the central nervous system. It is characterized by disseminated foci of demyelination, which are responsible for the diverse clinical picture of MS. Pain is a frequent but underestimated symptom of multiple sclerosis. It is estimated to affect 29–86% of MS patients in various stages of the disease and severely influences rehabilitation and quality of life. The pain experienced by MS patients is generally caused by nervous system damage during the course of the disease process and can usually be characterized as central neuropathic pain (less frequently as peripheral or nociceptive pain). The most frequent symptoms include dysesthetic extremity pain, painful tonic spasms, Lhermitte's sign, trigeminal neuralgia, headaches and low back pain. This paper discusses the probable mechanisms behind the development of pain in MS, the prevalence, classification, types of pain, as well as the most effective treatment methods.

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1. Introduction

Multiple sclerosis is a progressive, inflammatory demyelinating disease of the central nervous system (CNS) of an unknown etiology [1]. Its prominent feature is the presence of disseminated foci of demyelination, mainly in the white matter; in most cases, demyelination occurs periventricularly. The complex pathomechanism of the disease and its disseminated nature make both the clinical picture and the course of MS exceptionally diverse. The symptoms include movement, visual and sensory disturbances, cerebellar symptoms and sphincter control disturbances [1]. Pain is a frequent yet underestimated symptom of multiple sclerosis. For years, multiple sclerosis was thought of as a painless condition; however, as early as 1872, *Charcot* described pains in the shoulder and pelvic girdle region accompanying the disease [2]. In 1924, Lhermitte described the phenomenon of an electrical-like sensation running down the back, which is characteristic of MS [1–3]. However, the theory of a painless course of multiple sclerosis was only definitively invalidated at the close of the XX century. Recently, pain has been recognized as a factor that significantly affects quality of life [3–5]. The study conducted by Warnell [6] showed that among MS

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patients suffering from various types of pain, 40% had difficulty working, 44% had difficulty sleeping and 34% reported troubled interpersonal relationships [6].

2. Prevalence of pain

Pain is rarely observed at the initial stages of MS and is estimated to be its first symptom in only 1% of patients [3,7,8]. However, it frequently accompanies the advanced stages, being a nagging symptom that severely limits the patient's functioning, treatment and rehabilitation. Pain is estimated to occur in approximately 29–86% of patients at different stages of MS [3,8–12]. Pain present in the month preceding the evaluation was diagnosed in 53–79% of patients, whereas pain during the evaluation was present in 43–54% [5,8–11]. In 15% of patients, pain is acute (usually paroxysmal), but it is chronic in the straight majority [6,7,10]. Kalia et al. and Khan et al. estimated that chronic pain is present in 64–69% of MS patients [4,11]. According to the majority of authors, central pain is the most frequent type of pain [3,9–12]. In Osterberg's study, this type of pain was reported by 27.5% of patients [12]. However, Svendsen et al. reported that muscular (39.6% of patients) and articular pain (41.9%) are among the most frequent pain symptoms [5].

In a Polish study, Fryze et al. diagnosed various types of pain in 70.2% of MS patients [2]. In 8% of these patients, it was their first symptom of multiple sclerosis. The most frequently occurring symptoms were dysesthetic extremity pain (45%), back pain (34%) and painful tonic spasms (22%). Chronic pain was diagnosed in 60% of patients [2]. On the other hand, Kwolek et al. found various types of pain in 83% of patients (mainly painful muscle spasms and painful paresthesia) [13].

This large discrepancy in the results stems from the different definitions of pain that were adopted in the studies, different methods of pain assessment, and the different stages of disease progression and disability in the studied populations. It is usually assumed that pain affects approximately half of MS patients, and all studies confirm that it is more frequent in people with multiple sclerosis than in those who are not affected by the disease [3,7–13].

The frequency of pain increases with the progression of MS and may be the dominating syndrome in its advanced stages. However, it should be noted that pain may occur at all stages of MS, and it is reported by as many as 11–23% of newly diagnosed patients [4,9–11]. Risk factors for pain in MS include older age, female gender (F/M = 2.2/1), longer duration of illness, higher EDSS score, concomitant depression or mental disorders, unstable course of illness, lower education and primarily- or secondarily-progressing type of MS [3,4,8,12,14].

3. Pathophysiology of pain in patients with multiple sclerosis

It is believed that most MS-related pain symptoms are of a neuropathic central type [3,8,15,16]. Neuropathic pain caused by peripheral nervous system damage, nociceptive pain caused by irritation of peripheral nerve endings in the nociceptive system and psychogenic pain are less frequent [3,15,16].

Central pain results from a primary CNS injury, but its pathomechanism has not yet been fully elucidated. The demyelination and axonal damage in the brain and spinal cord lead to distinct mechanisms and central hyperexcitability [14–16]. The pain is associated with damage of the thalamus or the parietal cortex, in which projection areas for the sensory tract are located, and it is usually secondary to lesions in the spino-thalamo-cortical pathways [23].

It is postulated that the lack of normal afferent impulses in the area of the lesion causes quantitative and qualitative changes in sodium and calcium channels, which increase neuronal excitability [8,14–17]. The pain symptoms are thought to result from the ephaptic spread of spontaneous ectopic discharges generated by demyelinated axons. Consequently, the spinal cord is constantly inundated with false information about painful stimuli, which in fact are non-existent [17]. The CNS has demonstrated the ability to modify its functions, which causes an enormous variety of pain symptoms depending on the time and place of their occurrence, pathological processes, individual variability, age and many other factors [17,18]. One of the most important roles in these processes is played by NMDA (N-methyl-D-aspartate) receptors for excitatory amino acids and NK-1 neurokinin receptors situated postsynaptically in the posterior horns of the spinal cord [27]. There is evidence that NMDA receptors take part in triggering the long-term excessive pain response to a slight, repetitive irritating impulse, due to the 'wind-up' effect [15–18]. This phenomenon is similar to the kindling effect observed in epileptic foci and is responsible for the creation of abnormal discharges [16,17]. An increased calcium ion concentration in cells leads to a rise of enzymatic activity. This result, for example, in the enhanced synthesis of nitrogen oxide, which takes part in the neuropathic pain mechanisms, most likely via an intensified release of neurotransmitters from presynaptic terminals [18,27].

Central pain involves large areas such as the whole side of the body and is usually one-sided. It is not associated with movement disturbances but may be aggravated by external stimuli. It is characterized by acute, burning or stinging sensations, sensory disturbances and poor (or no) response to standard analgesic therapy [8,18]. Central pain usually lasts for a short time and may take the form of a seizure. However, it may also be chronic with recurring relapses. It is usually moderate in intensity but can aggravate significantly during the attacks and in response to heat, cold or touch. In almost all cases, the pain is associated with other symptoms of CNS damage such as sensory disturbances, paresis or ataxia [8,17,18]. Central pain includes psychogenic and overlapping pain [8].

Peripheral neuropathic pain is less frequent in multiple sclerosis. In most patients, it manifests itself as a chronic, searing, burning pain of a dysesthetic type (unpleasant sensation arising spontaneously or following a stimulus) or as a paroxysmal pain that is usually severe, short-lasting, stinging and ''electric-like'' [16]. Its onset is spontaneous and may be triggered by movement. It is frequently accompanied by mechanical or thermal allodynia (pain in response to touch or hot/cold temperature), hyperalgesia (increased

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