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Sympathetic Nervous System activity – a new concept of the complicated etiology of low back pain radiates distally at the extremities

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ABSTRACT

Varied and complicated etiology of low back pain radiates distally at the extremities is still causing disagreement and controversies around the issue of its diagnosis and treatment. New research data demonstrated that almost one in five persons with back pain experience symptoms indicative of neuropathic pain component. The neuropathic involvement is not completely understood, and different mechanisms are thought to play important role. A combination of nociceptive and neuropathic pain-generating mechanism is thought to be involved, which established the term mixed pain syndrome. In the pathomechanism of neuropathic pain the lesion, trauma or overloading of the disc is thought to be a primary source of the neuropathic pain but the concept of neuropathic component of pain is more probable for chronic stage than acute. Assessment of neuropathic pain involves a systematic approach which includes a series steps; past and present history, detailed description of pain distribution, quality, pain intensity and neurological examination with emphasis on sensory testing. The sensory examinations need often to be supply neurophysiological testing and quantitate sensory testing. Some groups of the drugs are thought to be useful e.g. tricyclic antidepressant, sodium channel blockers (e.g. carbamazepine), gabapentin, opioids, NMDA (N-methyl-D-aspartate) receptor blockers and others for neuropathic pain treatment. The use of specific kind of the drugs depends on the symptoms and examinations findings.

Key words: sciatica, neuropathic pain, low back pain, quantitative sensory testing.

Varied and complicated etiology of low back pain radiates distally at the extremities is still causing disagreement and controversies around the issue of its diagnosis and treatment. Most of the clinicians are thought that the source of that pain is generally radicular. Some of them postulated the clinical meaning of the sacroiliac joint syndrome which demands injections of lignocaine to the area of that joint for pain release. Lastly, some of the scientist postulated the new concept in the understanding "patients with sciatica". That group of the patients is clinically divided into two sub-groups, namely radicular or pseudoradicular problems [1].

It is widely accepted that acute low back pain is caused by degeneration of intervertebral discs (hernia, bulging). While for acute condition protrusion is stated as the chief reason, for a group in chronic state (approx 10–40%) lateral or foraminal stenosis or tumors [2] and neuropathic component lastly are suggested. One should remember that in the pathomechanism of neuropathic pain the lesion, trauma or overloading of the disc is thought to be a primary source of that pain [3].

New research data from Germany (2009), demonstrated that almost one in five persons with back pain experience symptoms indicative of neuropathic pain component [4]. The neuropathic involvement is not completely understood, and different mechanisms are thought to play important role. A combination of nociceptive and neuropathic pain-generating mechanism is thought to be involved, which established the term mixed pain syndrome [4]. It is indicated that neuropathic pain can occurs via mechanical nerve root compression (mechanical neuropathic root pain), lesions of

nociceptive sprouts within the degenerated disc (local neuropathic pain), or by action of inflammatory mediators such as chemokine's and cytokines, which can originate from the degenerative disc even without any mechanical stress (inflammatory neuropathic root pain) [1]. The clinical symptoms among those two groups of the patients are very similar. It is difficult to carry out full diagnostics of the above mentioned symptoms due to lack of exactly defined golden standards [5, 6].

Nevertheless, the precise examinations can help to diagnose the sympathetic nervous system involvement in the pain thought to be "radicular". The diagnosis of the radicular character of pain thought to be sciatica is done on the basis of clinical examinations, interpretation of the magnetic resonance imagination (MRI) and sometimes electromyography.

It is widely known that, the most common cause of radicular pain in the lower limb is inflammation following nerve compression caused by, for instance, disc herniation, lateral or foraminal stenosis, spondylolisthesis or tumor [7, 8–11]. Due to this fact, patients with low back pain radiates distally at the extremities with positive Lasegue's test and disc hernation confimed by MRI on the level of five and forth lumbar level are often diagnosed as a radicular pain.

However, some studies have shown poor correlation between radiological imagining and clinical symptoms [12]. What is more, in some asymptomatic persons herniated nucleus pulposus was confirmed by MRI (21% for 20–39 years of age, 22% for 40–60 years of age and 36% for above 60 years of age). Taking into consideration disc bulging even higher numbers were obtained for these age groups, respectively 56%, 59% and 79% [13]. There are also reports on patients suffering from confirmed disk pathology or with stenosis with apparent neural compromise i.e. asymptomatic [13–15]. Nevertheless, during MRI evaluation one should consider that in majority of patients with pathology within disc area a strong correlation with pain in the lower limb is visible [16], but sometimes it is possible to observe improvement with no change concerning the disk [17], or the other way round: no improvement in spite of removing the disc protrusion or other reasons of nerve compression [11]. Takashasi et al [11] claim that compression itself causes only loss of function rather than pain, which was firstly postulated by Kelly [8]. It is suggested that processes other than compression are engaged in the development of sciatica and the leading role of inflammation in causing the feeling of strong pain along the sciatic nerve is underlined [9, 18].

In the context of the paper by Freynhagen et al. [1], the above data raise even more doubts as far as the interpretation of MRI is concerned, all the more as in the pathomechanism of neuropathic pain in chronic patients the primary injury of the intervertebral disc, for instance, has been described.

Another test to confirm inflammation of the sciatic nerve is electromyography (EMG). Neurophysiological examinations to support a proximal nerve root lesion include the distal motor latency and the F-wave latency or nerves, which receive their nerve fibers from the affected root. This examination will only show pathological values if motor fibers are involved in the damage. It is important to known that the proximal lesion to the dorsal root ganglion during examinations can give norm of sensory conduction. In general, when we consider the involvement of neuropathic component of pain it is important to realize that conventional electrophysiological techniques assess only the function of myelinated peripheral axonal system [4]. The involvement of the small fibers (neuropathic pain) is possible to assess by Quantitative sensory testing (QST). That system allows to complete assessment of all sensory submodalities, including the large (Aβ) and small (Aδ and C) fibers. Unfortunately, that system is quit new and used in sciences laboratories mainly. What are more QST results should not be the sole criteria utilized to diagnose structural pathology, of either a peripheral or central nervous system (CNS) origin. Abnormalities on QST must be interpreted in the context of a thorough neurologic examination and other appropriate testing such as the EMG, nerve biopsy, skin biopsy, or appropriate imaging studies.

The next of step of the standard diagnosis of radicular pain is bedside examinations on the basis of clinical criteria e.g. positive Lasegue's test, motor sensory, or reflex deficits, apart of MRI value analysis. For many years it was believed that those clinical criteria were specific for radicular pain only. In the study of Freynhagen et al. [1] those common used criteria were confirmed among patients with pseudoradicular pain as well. That situation blurring clinical pictures of those patients and the appropriate distinguishing of CNS involvement is difficult for less experienced physician. On the basis on the new data and the previous findings about disc protrusion importance, some authors postulated that pseudoradiculopathy and radiculopathy is rather a disease continuum, than the different disease entities [4, 18].

Some explanation is needed about the diagnostic value of the Lasegue's test because of the clinical

common use of them. According to scientist the diagnostic accuracy of the neurological signs and tests is unclear [19]. The Lasegue's test is a widely used diagnostic tool for confirming sciatica. Total clinical reliability of this test is questioned, however, as it has no identical application standards or result interpretation. It was even claimed that for diagnostic purposes negative result was more significant than the positive one [12]. Simultaneous use of the Lasegue's test together with a passive ankle dorsiflexion (Bragard's procedure) for more reliable confirmation of radicular pain is suggested [20].

Some of the authors indicate possible distortion of the result for the Lasegue's test by strong hamstrings tension [21]. During the Lasegue's test the patient's description of pain is taken into consideration, which, according to many authors, raises doubts and is not too credible a tool for Lasegue's test verification. According to Backup [22], strong tension of these muscles might simulate the inflammation of the sciatic nerve. In other papers it was proven that basing on the Lasegue's test it was not possible to differentiate between patients who were asymptomatic but had strong tension in the hamstring muscle and patients with sciatica [23]. Mechanisms leading to the increase in the tension of these muscles have not been explored so far.

The crossed Lasegue's test made the diagnosis more specific for hernia thus either crossed Lasegue's test or Bragard's procedure can be used to confirm radicular character of pain in case of positive Lasegue's test. In the cases with the negative Lasegue's test or/with unsure interpretation of the positive one, negative MRI findings we should consider sympathetic nervous system (SNS) activity, especially in chronic stage. Apart of the QST the common used tools are questionnaire: Leeds Assessment of Neuropathic Symptoms and Signs (LANSS), Neuropathic Pain Questionnaire (NPQ), Douleur Neuropathique en 4 questions (DN4), pain DETECT [24]. Clinical examination at bedside includes: pinprick, touch, cold, heat and vibration. Pinprick sensation is assessed by the response to prinprick stimuli; touch is examined by gently stroking the involved skin area with a cotton swab, cold and warm sensation is recorded by measuring the response to a specific cold or warm thermal stimulus. Vibration is assessed by a tuning fork placed at strategic points. At present there is no consensus about the site where such activity should be measured, but it is generally agreed that this is best done in the area as control. For all types of stimuli, the response can simply be graded as: normal, decreased or increased. If the response is increased, it is classified as dysesthetic, hyperalgesic or allodynic. Assessment of neuropathic pain involves a systematic approach which includes a series steps; past and present history, detailed description of pain distribution, quality, pain intensity and neurological examination with emphasis on sensory testing. The sensory examinations needs often to be supply neurophysiological testing and quantitate sensory testing [25]. The distinguishing the radicular and neuropathic component is very important because of the completely different treatment approach. There a lot of cases suffered months or years because of the "chronic sciatica" after different failed therapies. Some groups of the drugs are thought to be useful e.g. tricyclic antidepressant, sodium channel blockers (e.g. carbamazepine), gabapentin, opioids, NMDA receptor blockers and others for neuropathic pain treatment. The use of specific kind of the drugs depends on the symptoms and examinations findings.

We should remember that now clinicians are challenged with a series of possible pathophysiological mechanism of neuropathic pain and the optimal way of the treatment is difficult due to lack of the knowledge. Additionally, excellent work in the basic science of that pain is in contrast with the limitations of inadequate random controlled trials regarding long-term pharmacologic interventions. Complex rational pharmacologic strategies for structural pathology, central pain processes, sites of medication action, and differing routes of administration are delineated [26].

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