Features of Chronic Pain Syndrome in Radiculopathies

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ABSTRACT
The article deals with the features of chronic pain syndrome in radiculopathies. Causes of radiculopathy is a disease of the peripheral nervous system of a person, which occurs as a result of damage, inflammation or pinching of the roots of the spinal nerves. Radiculopathy symptoms, diagnosis and how radiculopathy can be treated.

KEYWORDS: Radiculopathy, syndrome, diagnosis, osteochondrosis, pathology

INTRODUCTION
Against the background of lumbar osteochondrosis, a disease such as radiculopathy of the lumbosacral spine very often develops. This is a clinical syndrome that occurs due to compression and damage to the nerve roots. Radiculitis is synonymous with radiculopathy.

Radiculopathy is a disease of the peripheral nervous system of a person, which occurs as a result of damage, inflammation or pinching of the roots of the spinal nerves. This is a widespread disease, more than 10% of the population of our planet is over 40-50 years old, and in the last decade sciatica has become younger, occurs in the young age group from 25 to 35 years old, people involved in professional sports, as well as those who are while sitting at a computer or driving a car.

Causes of radiculopathy
The cause of the development of radiculopathy is the pathological processes that occur in the spine, namely:

- Degenerative-dystrophic processes - osteochondrosis and its complications: protrusion and herniated intervertebral discs.
- Malformations of the musculoskeletal system, accompanied by changes in muscle tone.
- Posture disorders, curvature of the spine - scoliosis.
- Incorrect distribution of physical loads on the spinal column during professional human activities (when driving a car, for office workers and loaders - with a long stay in one position, loaders).
- A sedentary lifestyle, in which blood supply to the muscles of the lumbar region is gradually increasing.
- Improper drinking regime throughout the day and for several months.
- Deficiency of vitamins and minerals leads to osteoporosis and trophic lesions of the nerve fiber.
- Tuberculosis, osteomyelitis, chronic syphilis and other types of spinal cord infections.
- Regular injuries of the vertebrae and surrounding soft tissues (impacts, sharp twisting, compression when jumping, sprains of the ligamentous apparatus).

- Overweight with obesity 1-2 degrees and the presence of bad habits such as smoking, abuse of tea, coffee and alcoholic beverages.

Symptoms of radiculopathy
The first manifestation and the main permanent symptoms of radiculopathy of the lumbosacral spine are pain, impaired sensitivity and muscle weakness.

The affected area corresponds to the innervation area. Depending on the affected area, pain can be observed in the lumbar and gluteal regions, posterolateral and front of the thigh, along the antero-out surface of the lower leg, on the back of the foot, big toe, in the gastrocnemius muscle, in the area of the external ankle and heel.

Lumbosacral radiculopathy is often chronic, with acute relapses. Lumbosacral radiculopathy is characterized by an increase in pain with a sharp change in the body from a horizontal position to a vertical one or vice versa, as well as when engaging in an active lifestyle (sports, walks, fitness classes).

Exacerbation of pain syndrome in radiculopathy of the lumbosacral region is noted when the inter-root nerve is compressed by an intervertebral hernia.

According to the types of radiculopathy of the lumbosacral spine, there are:

- Lumbago (lumbodynia) is an acute pain in the lower back during abrupt physical activity, as well as provoked by overheating or hypothermia of the body. The painful attack itself can last in different ways: from several minutes to several hours or even days. The main reason for this type of radiculopathy is overstrain of the muscles of the lumbar region, intervertebral hernias or displacement of the vertebrae relative to each other.

- Sciatica (sciatica) - in this type of radiculopathy, pain is localized in the buttock, on the back of the thigh and lower leg, and can reach the foot. Sometimes, in addition to pain, muscle weakness is noted. This is due to damage or irritation of the sciatic nerve, the largest nerve in the body. Pain in sciatica - shooting, like an electric shock, burning, tingling, "chills" and numbness are also possible. Pain sensations of varying degrees of intensity are possible: from mild to very intense, such that the patient cannot sleep, sit, stand, walk, bend over or turn.

- Lumboischialgia - back pain radiating to the leg or legs. With this type of radiculopathy, pain spreads mainly along the buttoc and along the posterior outer surface of the leg, not reaching the toes, most often it is aching, burning, growing pain.
Diagnostics
For the diagnosis of radiculopathy, the presence of tension symptoms is important. For example, the Lasègue symptom is characteristic - when you try to raise a straight leg while lying on your back, lower back pain increases.

An MRI or CT scan of the lumbar-sacral spine is important for making the correct diagnosis, because only these studies will allow the attending physician to detect the presence of diseases such as intervertebral hernias or other diseases of the spine (spondylitis, compression fractures, neoplasms).

Treatment of radiculopathy
The modern approach to the treatment of acute and chronic pain in radiculopathy of the lumbar-sacral spine is to use non-steroidal anti-inflammatory drugs and muscle relaxants. The mechanism of their action: reduction of inflammation, pain intensity, removal of muscle spasm. Also, during the period of remission of the disease, physiotherapeutic treatment is carried out (electrophoresis, ampuliplex, darsenolization).

Radiculopathy resulting from pathological changes in the spine is one of the most severe forms of neurological manifestations of vertebrogenic pathology of the peripheral nervous system. In the case of compression of the spinal root, the duration of disability is significantly increased, and expensive examination and treatment are required. With an inadequate assessment of the existing symptoms of degeneration, permanent disability with a significant neurological deficit can occur. With a high prevalence of low back pain, symptoms of spinal root compression are observed in 3-5% of people in the population. The peak incidence in men is at the age of 40-50 years, in women - 50-60 years [4-7].

The leading mechanism of the pathogenesis of spondylogenic radiculo- and myelopathies is the compression effect on the arterial and venous vessels of the spinal root. Coarser compression leads to mechanical damage to the fibers. This occurs when space is limited at the site of the passage of the spinal root in the intervertebral foramen - at the level of the lateral pocket (in most cases, with a herniated disc). A stenosing effect can also be observed with calcification of the ligamentous apparatus with the formation of osteophytes, hypertrophy of the articular facets, or capsular segmental processes. A combination of these changes is possible, especially with a significant decrease in the height of the intervertebral discs [7, 8].

Radiculopathy is a medical, social and economic problem that not only reduces the quality of life, activity and performance, but also causes disability of patients. The urgency of this problem is due to the prevalence and duration of the disease. The term "radiculopathy" comes from the Latin words "radícula" and "pathía", which means a lesion of the spinal nerve. This is not an independent disease of the peripheral nervous system, but a complex of symptoms of damage to the spinal nerves, a relatively common pathological condition, typical for an average of 10-15% of people over the age of 45. According to statistics, there is no gender difference - men and women get sick equally.

The spinal nerve is formed by the fusion of the anterior axons of peripheral motor neurons that exit through the anterior lateral clef of the spinal cord, the number of which is equal to the number of spinal cord segments, with the sensory neurons of the spinal cord. After fusion, the spinal nerve (SMN) includes motor, sensory and autonomic fibers and exits the spinal canal through the spinal foramen.

The roots of the spinal cord lie in the subarachnoid space, filled with cerebrospinal fluid, between the arachnoid and pia mater, the latter of which is tightly attached to the spinal cord and fuses with it. SMN can be damaged by osteophyte, protrusion, hernia, surrounding tissues, such as ligaments, muscles, in rare cases by a tumor and other mechanical factors. For the development of radiculopathy, a provoking factor is needed, which can be lifting of weight, violation of a motor stereotype, hypothermia, stress, prolonged stay in a fixed, especially vertical, posture, bending, especially forward [1].

Pain can be caused by irritation or compression of one or, less commonly, several spinal nerves. The most common risk factor for radiculopathy is herniated disc. Hernias at the cervical and lumbar level are quite common. The intervertebral disc undergoes dehydration early enough, cracks, the outer wall of the annulus fibrosus becomes thinner, stretches, the nucleus pulposus shifts from the center to the periphery, which leads to its bulging. This process can lead to localized pain and moderate muscle tension. With the further process, the bulging increases by several millimeters and a protrusion forms. Clinically, this is manifested not only by local pain, but also by a disorder of sensitivity, asymmetry of tendon reflexes. After rupture of the posterior longitudinal ligament, a hernia or extrusion is formed, which, depending on the localization, can compress the SMN. The median hernia practically does not put pressure on the root, paramedian and foraminal (lateral) hernias are significant. Clinically, this is manifested by a pronounced pain syndrome, sensory and motor disorders in the area of localization of the corresponding root. However, the size of the hernia and the severity of the pain syndrome do not have a strict interdependence [1, 2].

At the moment, there is no single point of view regarding the mechanism of development of compression radiculopathy. The direct effect of the compressing agent on the spinal root (SMC), artery or vein, causing the development of an inflammatory reaction with the release of prostaglandin E2, interleukins, tumor necrosis factor, nitric oxide and other substances, is considered as a triggering factor. Venous discirculatory disorders develop. The pathogenesis of pain syndrome is quite complicated, it is not only mechanical compression of the MCS, but also is chemic, since the arteries and veins of the MCS are subjected to compression. Pain in radiculopathy manifests itself sharply, intensely, with lumbago in the extremities and even in the head like cervicocranialgia.

The pain syndrome is of a mixed nature: nociceptive as a result of irritation of peripheral nociceptors of the surrounding tissues, neuropathic - due to damage to the spinal root, psychogenic - with chronic pain. The role of inflammation, reflex ischemia of the root, hemorrhage in the root is also not excluded [3]. Violation of venous blood flow in neurological manifestations of radiculopathy plays a
significant role. Even small disc prolapses can cause complete epidural vein block [4]. Indirect confirmation of these data was obtained when examining 9640 patients with back pain or lumboschialgia: 13 (0.13%) patients had radicular symptoms caused not by a herniated disc or stenosis of the spinal canal, but by occlusion of the inferior vena cava leading to the expansion of the epidural veins; 12 of them managed to restore blood flow through the inferior vena cava, which led to the disappearance of radicular symptoms [5, 6].

However, the theory of compression impact cannot explain all cases of radiculopathy with disc herniation, since sometimes the severity of the symptoms of neurological deficit does not correspond to the size of the hernial protrusion. An inflammatory theory in the genesis of radiculopathy caused by herniated discs has found numerous confirmations. It is believed that inflammation can be either autoimmune, triggered by antigens of the nucleus pulposus, or caused by the release of inflammatory mediators directly from the nucleus pulposus. In this case, the mediators of inflammation can be phospholipase A2, cytokines, especially interleukin 1, 6, prostaglandin E2, nitric oxide, immunoglobulins. Phospholipase A2 regulates the release of arachidonic acid from cell membranes and has a direct damaging effect on cell membranes. With a herniated disc, stagnation and thrombosis in local venous vessels with thickening of the basement membrane and fibrosis of the endothelium are found, accompanying severe perineural and endoneural fibrosis and atrophy without signs of inflammation. These pathological deviations occur due to impaired venous outflow. Edema and root damage increase with increasing speed and degree of compression, and sensory fibers may be more vulnerable than motor fibers [7, 8].

Conservative therapy is ineffective only in a small group of patients; in this case, surgical treatment is required [9]. These findings are supported by neurophysiological observations. In a chronic process, there is demyelination of the SMC.

Herniation at several levels is rare, therefore biradiculopathy syndrome in hemias is explained mainly by the compression of two roots at the same time by large hernias. Reactive adhesion phenomena may develop in the roots, epidural tissue, the membranes of the spinal cord, which may be associated with autoimmune inflammation in a herniated disc.

Cervical, thoracic and lumbar radiculopathy is distinguished depending on the level of the lesion - they are equally characterized by certain symptoms:

1. pain of varying intensity and frequency, radiating along the course of the SMN;
2. Violation of sensitivity. With prolonged lesion of SMN, there is a decrease in tone in the surrounding tissues, hypo- or muscle atrophy;
3. With a pronounced lesion of the SMN, peripheral paresis develops.

Conclusion
The currently available evidence of the high efficacy of pregabalin for the treatment of NBS, both in monotherapy and in combination with other drugs, makes it possible to use it as a drug of choice or a first-line agent, including in radiculopathy. The combination of pregabalin with other drugs, in particular NSAIDs, contributes to more effective relief of pain syndrome, reduces the severity of anxiety-depressive disorders, and also improves the quality of life of patients with radiculopathy.

Literature