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DIFFERENTIAL DIAGNOSTICS AND OPTIMIZATION OF TREATMENT OF TONIC SYNDROME OF THE NECK MUSCLES AND RADIUTAL PAIN SYNDROME

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Annotation: Currently, based on etiopathogenesis, pain syndromes are divided into three main groups: somatogenic (nociceptive), neurogenic (neuropathic) and psychogenic pain. Pain syndromes that arise as a result of activation of nociceptors during injury, inflammation, ischemia, tissue damage (skin, bones, joints, muscles, etc.) are referred to as nociceptive pain. Neuropathic pain occurs due to damage or dysfunction of structures of the peripheral and/or central nervous systems at various levels, which is supported by disturbances in the processing of sensory information in the nervous system.

Keywords: Pain, vertebral neurological syndromes, segment.

Pain is an unpleasant experience affecting the sphere of sensations and emotions, associated with real or perceived tissue damage, and at the same time a reaction of the body that mobilizes functional systems to protect it from the effects of a pathogenic factor.

Pain is not only a symptom of various acute and chronic pathological processes, but also a complex psychophysiological phenomenon, including mechanisms of regulation and formation of emotions, motor, humoral and hemodynamic manifestations. Neurophysiological mechanisms of pain include, in turn, the participation of peripheral, segmental and suprasegmental structures [2, 4, 14].

A special group consists of psychogenic pain, which can occur regardless of somatic, visceral or neuronal damage and is largely determined by psychological and social factors. One of the mechanisms for the formation of this type of pain is reflex muscle tension caused by emotional factors, leading to the development of painful discomfort [4, 8].

Neck and back pain can be acute or chronic. Acute pain is a normal reaction to injury tissues and usually goes away as they heal. Chronic pain can be caused by constant irritation of nociceptors in the area of tissue damage. It gradually loses the protective function inherent in physiological pain, signaling value and activating effect on the mechanisms of eliminating the algogenic factor. According to M.L. Kukushkin [8], pain that has a pathogenic effect and causes maladaptation is called “pathological pain.”

Neck and back pain are among the most important and practically significant problems of modern neurology, which is determined by a number of factors. Firstly, this group of diseases is the most common non-infectious human pathology - according to domestic and foreign researchers, their prevalence in the general population reaches 30-40%. Secondly, pain in the neck and back has a pronounced tendency to be chronic and recurrent, often leading to temporary and permanent disability. These two factors explain the

huge primary and secondary costs, as well as the medical and social significance of the problem, since it leads to a pronounced decrease in the quality of life of patients [10, 13].

The causes of neck and back pain are very varied and numerous. As a rule, they are divided into two large groups - vertebrogenic and non-vertebrogenic origin. Pain in the neck and back and clinical manifestations that occur with degenerative-dystrophic changes in the spine are referred to in the domestic literature as vertebral neurological syndromes [1, 10, 13]. Foreign authors describe such disorders as benign musculoskeletal back pain, dorsalgia and dorsopathy [21, 22].

Pain in the neck and back of a vertebrogenic nature

In the vast majority of cases, pain in the neck and back is etiologically associated with degenerative-dystrophic lesions of the spine, which include osteochondrosis (protrusion or prolapse of the intervertebral disc, dystrophic damage to the intervertebral disc and adjacent vertebral bodies), spondyloarthrosis (arthrosis of the facet joints) and spondylosis, manifested by bone formation under the anterior longitudinal ligament. We should not forget about rarer, but practically very important (due to the seriousness of the prognosis in the absence of adequate treatment) such diseases of the spine as infectious lesions (tuberculosis, brucellosis, epidural abscess), autoimmune pathology (ankylosing spondylitis, Reiter's syndrome, rheumatoid arthritis), metabolic lesions (osteoporosis, osteomalacia), neoplasms and injuries, as well as diseases of internal organs, accompanied by referred pain.

From the point of view of the modern pathogenetic classification of pain, which takes into account the leading mechanism of its formation, vertebrogenic pain syndromes should be classified as having a mixed nature (at least in most cases). Indeed, pain in vertebrogenic pathology includes both nociceptive (irritation of pain receptors located in the fibrous ring of the intervertebral disc, posterior longitudinal ligament, areas of the dura mater, facet joints, muscles, fascia, etc.) and neuropathic component (effect on the root, spinal nerve or spinal ganglion). In addition, since vertebrogenic pain tends to be chronic, it is not surprising that at a certain stage of the disease a psychogenic component also appears, which later often becomes dominant.

It is important to note that the source of pain in vertebrogenic pathology can be both elements of the spinal motion segment and neural structures, as well as extravertebral formations , primarily muscles; Moreover, when certain structures are damaged, the pain syndrome will have its own characteristics. From this point of view, in the structure of vertebrogenic pain syndromes it is necessary to distinguish: 1) myogenic; 2) arthrogenic; 3) discogenic; 4) spondylogenic; 5) neuropathic (radicular) pain. Naturally, this division of pain is arbitrary, since in the same patient they are usually combined; at the same time, identification of the dominant form is necessary for a differentiated approach to therapy [1, 13, 14].

The features of myogenic pain, which invariably accompanies muscular-tonic and neurodystrophic syndromes of spinal osteochondrosis, include aggravation when the muscle is tense, especially in a shortened state, when the muscle is passively stretched, when the trigger zone is compressed, when the affected muscle is in a shortened state for a long time. In connection with the latter, the clinic often observes a pathognomonic phenomenon of increased pain during the first movements after rest, and with continued physical activity, the pain significantly decreases or disappears. The pain intensifies with local mild cooling, which often affects the next day and is qualified by the patient as “blown neck, lower back, etc.” Myogenic pain decreases after short rest, slow passive stretching of the affected muscle, using local heat, and after light movements [12, 7].

Discogenic pain also has its own clinical characteristics. The first characteristic sign is increased pain with movement, decreased with rest. This is most demonstrably observed in the pathology of the lumbar discs. As walking (movements) continues, the patient notes a progressive increase in pain, often localized in the midline or with slight lateralization , and the appearance of scoliosis (or worsening of existing scoliosis).

The nature of the pain is pressing, bursting or cerebral. But if, with protrusion of the lumbar discs, a horizontal position is optimal, then patients with cervical discogenic pain often experience increased pain in the supine position, which forces them to sleep half-sitting. A characteristic sign may also be sclerotomal irradiation of pain [10, 24, 20].

Spondylogenic pain occurs when the vertebral bodies and their processes are damaged. The causes of spondylogenic pain can be both dystrophic changes, leading to increased intraosseous pressure and irritation of intraosseous receptors, and vertebral tumors (primary and metastatic), tuberculosis, angiomas, osteomyelitis.

Arthrogenic pain in the neck and back is a consequence of damage to the intervertebral joints. The intervertebral joints do not bear a supporting load; they guide movements. When the height of the disc decreases, the articular surfaces of the intervertebral joints may begin to contact each other, resulting in the occurrence of spondyloarthrosis. Algic syndrome with spondyloarthrosis does not have a characteristic clinical picture. As a rule, the arthrogenic component of the formation of pain syndrome is established on the basis of characteristic changes in the joints detected by X-ray computed tomography (X-ray computed tomography). Acute arthrogenic pain can occur when the meniscoid of the joint is pinched, with the development of blocking in the joint. Resolution of joint blockade is possible through mobilization or manipulation techniques [18, 16, 19, 23].

The most common cause of neuropathic pain is compression of a root or spinal nerve. Compression is usually accompanied by pain, radiating along the corresponding dermatome or sclerotome . The most common causes of compression are herniated discs and hypertrophy of the ligamentum flavum. Less commonly, the cause of radiculopathy is true or false spondylolisthesis . To identify the cause, the patient should definitely undergo magnetic resonance imaging (MRI) of the spine and spinal cord, and, if necessary, X-ray CT. As a rule, pain increases with movement and decreases with rest. Impaired sensitivity in the dermatome occurs when two or more roots are compressed due to overlapping zones that “mask” sensory disorders in monoradiculopathy .

Non -vertebrogenic neck and back pain

Currently, the structure of pain in the neck and back of a non-vertebral nature is represented by the following forms: myofascial pain syndromes, fibromyalgia , psychogenic pain, referred pain in diseases of the visceral organs (heart, lungs, gastrointestinal tract, genitourinary tract), tumors of the intra- and extraspinal , epidural abscess, metastatic tumors, syringomyelia, retroperitoneal tumors, osteoarthritis . The most relevant for general medical practice are the following first two forms of non-vertebrogenic back pain, as they are the most common in clinical practice [5, 7, 11, 12].

Myofascial pain syndrome (MPS) is one of the most common causes of chronic pain, but its reality (due to the lack of objective signs) is still controversial. MPS is based on the formation of trigger points in the muscles (this is an area of local longitudinal compaction measuring from 2 to 5 mm, located in the direction of the muscle fibers), the irritation of which causes not only local, but also referred pain (in an area remote from this point). The trigger point (TP) corresponds to a zone of local muscle compaction, palpation of which not only reproduces the pain experienced by the patient, but also causes local muscle contraction. TTs can be active or latent. Active points cause spontaneous pain, often in the reflected zone, and limit the contractile capabilities of the muscle in which they were formed. Latent points cause local muscle tension and muscle dysfunction, but not pain. Although focal neurological symptoms are not detected, patients often experience “numbness” associated with limited mobility, increased fatigue, and muscle tension. Active TTs can “metastasize”, contributing to the formation of secondary TTs and the transformation of regional pain syndrome into a more diffuse one.

The cause of the formation of TT can be trauma, muscle overload (for example, during a long stay in an uncomfortable position, with skeletal asymmetry, scoliosis), metabolic disorders, malnutrition, rheumatological diseases (osteoarthritis, rheumatoid arthritis, SLE), neurological diseases (radiculopathies , tunnel neuropathies , polyneuropathy, plexopathy , multiple sclerosis). An important role is played by vertebrogenic influences and especially psychological factors (emotional stress, anxiety, depression, conscious or unconscious desire to obtain moral or material benefit from one's illness).

Clinical manifestations of MS MS depend on the location of the point, for example, headache can be associated with TT in the sternocleidomastoid , suboccipital , temporal, scalene muscles, posterior neck muscles, and facial pain simulating temporomandibular joint syndrome can be caused by trigger points in the masticatory muscles [11, 12, 14].

The problem with diagnosing MBS is that a relatively large number of muscles could potentially be involved in the development of MBS, and, accordingly, the doctor must be proficient in the technique of manual examination of various muscles. Difficulties are associated with the different constitution of patients, the thickness of the subcutaneous fatty tissue, the depth of the muscles, especially the muscles of the legs and gluteal region (deep-lying piriformis muscle) that are difficult to palpate.

Fibromyalgia is characterized by a chronic, widespread pain syndrome that symmetrically involves different areas in the upper and lower half of the body. The pain intensifies under the influence of various factors (weather changes, physical activity, stress). It occurs 10 times more often in women than in men. Clinical criteria for fibromyalgia include: 1) the presence of chronic pain in various areas of the trunk and limbs on both sides above and below the waist; 2) detection with moderate palpation of multiple painful trigger points in at least 11 of 18 standard zones: occipital region (at the site of attachment of the suboccipital muscles); neck (anterior sections of the spaces between the transverse processes (Sui); trapezius muscle (middle of the upper edge); sternocostal joint at the level of the second rib; region of the angle of the scapula (attachment site of the periosteal muscle); external epicondyles of the shoulder (2 cm distal); the upper outer quadrant of the buttock (along the anterior edge of the muscle); the greater trochanter; the inner surface of the knee joint (medial fat pad).

Unlike MPS in fibromyalgia, stimulation of painful points does not cause local muscle contraction or irradiation of pain (therefore, these points are not trigger points), and the pain syndrome itself is diffuse, not focal. In addition, the pain syndrome in fibromyalgia is bilateral, often symmetrical, while in MBS the pain is often unilateral. At the same time, MBS can transform over time into fibromyalgia , so it is not always possible to clearly separate these two conditions.

Peripheral pain syndrome in fibromyalgia is often accompanied by tension headaches, sleep disturbances, constant daytime fatigue, emotional disorders, especially depression, hyperventilation syndrome, autonomic crises, irritable bowel syndrome and other psycho-vegetative disorders.

The pathogenesis remains unclear. In a significant proportion of cases, one can detect muscle detraining , depression, neuroendocrine changes, as well as peculiar changes in the EEG during sleep (the alpha rhythm, characteristic of relaxed wakefulness, appears in patients of this category in deep slow-wave sleep). All these changes indicate dysfunction of the central neurotransmitter systems.

Thus, fibromyalgia may be the result of an interaction between psychological and organic factors. However, the absence of objective clinical or laboratory signs that could confirm the diagnosis serves as a basis for doubting the reality of this disease. Sometimes fibromyalgia syndrome occurs with rheumatoid arthritis, SLE or other connective tissue diseases - secondary fibromyalgia [6].

Psychogenic pain (psychalgia) is diagnosed in the absence of an organic disease or in the case when the latter cannot explain the nature and severity of the pain syndrome. Psychogenic pain is always chronic and

occurs against the background of mental disorders: depression, anxiety, hypochondria, hysteria, phobia. In a significant proportion of patients, psychosocial factors play an important role (dissatisfaction with work, desire to obtain moral or material benefit). Particularly strong links exist between chronic pain and depression. Differentiating purely psychogenic pain from chronic nociceptive or neuropathic pain can be difficult, since in these types of pain psychogenic factors play a significant (although not dominant) role. In diagnosis, it is important to take into account the discrepancy between the patient's complaints and standard pain syndromes, the absence of objective signs of pain, its non-localized nature ("the whole back, the whole leg, the whole body hurts"), the constant migration of pain, the ineffectiveness or poor tolerability of treatment, the presence of numerous "crises" , whimsicality or amorphism in describing the nature and location of pain. A significant role in the diagnosis of psychogenic pain is played by the identification of other symptoms of the disease, for example, demonstrative personality traits, rental attitudes, functional neurological disorders - in hysteria, melancholy, anxiety and mood swings - in depression. Occasionally, psychogenic pain is a manifestation of senestopathic disorders in schizophrenia. Some cases of psychogenic pain are accompanied by severe autonomic disturbances (for example, of a vasomotor nature) in the pain area, which serves as a reason for the erroneous diagnosis of reflex sympathetic dystrophy [3, 9, 15, 22].

A good placebo response may not be a hallmark of psychogenic pain; it is observed in approximately one third of patients suffering from pain of any origin. However, persistent response to placebo or psychotherapy is a strong argument in favor of psychogenic pain.

An integrated approach to treating neck and back pain

Before moving on to a discussion of methods of treating pain syndromes in the neck and back, it is advisable to briefly dwell on the issue of surgical treatment. According to Ya.Yu. Popelyansky , only 0.3% of patients with intervertebral disc herniations require surgical intervention. The absolute indications for surgical treatment are acute compression of the cauda equina and spinal cord, manifested by pelvic disorders, bilateral pain and paresis. In addition, surgical treatment is probably justified in the presence of large sequestered disc herniations. Relative indications are the severity and persistence of radicular symptoms in the absence of the effect of adequate conservative therapy carried out for more than 3-4 months, however, even in this case, the issue of surgical intervention should be decided strictly individually and preference should be given to conservative methods of treatment.

Drug therapy

Currently, many analgesics, non-steroidal anti-inflammatory drugs (NSAIDs) and other drugs are available for pain relief, which often leads to polypharmacy with all the ensuing adverse consequences. In this regard, it should be emphasized once again that drug therapy must be strictly individual and differentiated. It is important to take into account the severity, course and basic pathogenetic mechanisms of pain. As already mentioned, in most cases, pain syndromes in the neck and back are mixed, however, one should always try to isolate the dominant component (nociceptive , neuropathic or psychogenic).

For acute, predominantly nociceptive pain, the drugs of choice are NSAIDs - diclofenac, ketoprofen (ketorol), dexketoprofen (dexalgin). The course of therapy should not be long; it is advisable to limit it to 5-7 days. If longer-term therapy is necessary, it is advisable to use selective COX-2 inhibitors, such as meloxicam (movalis , mirlox , arthrozan), celecoxib (celebrex), arthrozan , nimesulide (nimesil , nise). In case of severe pain syndrome, the effect of NSAIDs may not be enough; in such cases, it is advisable to use more powerful analgesics, of which the combination drug of paracetamol and tramadol , zaldiar , has proven itself well . Usually a short course of zaldiar therapy (1-3 days) followed by NSAIDs is sufficient. For both acute and chronic pain, the selective activator of neuronal potassium channels flupirtine (

catadolone), which also has a muscle relaxant effect, is effective; the drug is used as monotherapy or in combination with NSAIDs.

In case of pronounced muscle-tonic reactions and the presence of painful muscle spasms, NSAIDs and analgesics should be combined with centrally acting muscle relaxants, such as tolperisone (mydocalm), tizanidine (sirdalud), baclofen (baclosan).

In addition to systemic therapy, you can use local agents - ointments with a local irritant, distracting and anti-inflammatory effect. The transdermal therapeutic system with lidocaine, Versatis , is highly effective for neck and back pain ; its use is especially advisable in cases where there is a neuropathic component of pain, even in the absence of obvious signs of radicular syndrome.

For acute pain with a pronounced neuropathic component (vertebrogenic radiculopathy), prescribing only NSAIDs or analgesics is not enough; it is necessary to use drugs that eliminate central sensitization . A number of anticonvulsants, such as gabapentin (Neurontin , Tebantin), pregabalin (Lyrica), topiramate (Topamax), are highly effective for radicular pain of vertebrogenic etiology. To reduce irritation of the affected root (which, in addition to mechanical compression, can also be caused by edema due to a local inflammatory process), dehydration therapy (short course loop or osmotic diuretics) or epidural blockades using a mixture of local anesthetic and glucocorticoid solutions can be used.

Therapy aimed at restoring the function of the affected root (or spinal nerve) is important. For this purpose, the prescription of vasoactive [aminophylline, pentoxifylline (Trental), vinpocetine (Cavinton)], as well as metabolic [B vitamins (milgamma), thioctic acid (thiogamma , berlition , thioctacid)] and neuroprotective drugs (actovegin) is justified , Cerebrolysin).

General principles for the treatment of chronic pain

For chronic pain of any origin, it is important to influence all possible pain factors using physical, medicinal, and psychotherapeutic means. For example, in the treatment of a suffering patient with vertebrogenic lumbar ischialgia , it is advisable to have a local effect on altered structures of the spine, reflex muscle-tonic syndrome, neuropathic pain associated with compression of the root, but no less important is an attempt to change its motor pattern and psychological mood. Drug therapy for chronic pain syndromes, in addition to analgesics and NSAIDs, includes a number of additional drugs that are especially effective for neuropathic pain.

Antidepressants have a moderate effect on chronic pain of any origin, which is not directly related to their antidepressant effect and occurs at lower doses. Antidepressants are more effective for persistent burning pain than for paroxysmal pain. The most widely used is amitriptyline (initial dose 10 mg at night, then the dose is gradually increased to 50-100 mg at night), however, excessive sedation, pronounced anticholinergic effects, and adverse effects on the heart limit the use of the drug. Patients sometimes tolerate other tricyclic or tetracyclic antidepressants better, in particular doxepin , 25-100 mg at night, desipramine , 50-100 mg/ day , mianserin (Lerivon), 30-90 mg/ day . Apparently, antidepressants that block the reuptake of not only serotonin, but also norepinephrine, are more effective. Selective serotonin reuptake inhibitors (for example, fluoxetine, 10-40 mg/ day , or sertraline , 25-100 mg/ day), although they cause fewer side effects, have a less distinct and analgesic effect and are indicated mainly in cases where the pain is associated with depression, and the patient does not tolerate tricyclic antidepressants well. In some situations, it is possible to combine low doses of tricyclic antidepressants with selective serotonin reuptake inhibitors, which have a relatively short half-life (for example, sertraline).

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