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УДК. 616.81678/76.54

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TRANSIENT COMPLEX REGIONAL PAIN SYNDROME AGAINST THE BACKGROUND OF CENTRAL POST-STROKE PAIN

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ANNOTATION

Pain syndrome occurs in about half of stroke patients. Despite this prevalence, it is an underestimated consequence of stroke. According to the place of generation, central and peripheral pain are distinguished. More often in stroke there is peripheral pain resulting from arthropathies of large joints of paralyzed limbs, skeletal-muscular pain, painful spasticity. Central post-stroke pain is less common in stroke patients and complex regional pain syndrome is rarely described.

Keywords: complex regional pain syndrome, central post-stroke pain, strombotic venous syndrome, systemic treatment approach

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Dilbar Tadjievna Xodjieva
Madina Zafarovna Oxunjanova
Buxoro davlat tibbiyot instituti

KEYINGI MARKAZIY OG'RIQ FONIDA VAQTINCHALIK KOMPLEKS MINTAQAVIY OG'RIQ SINDROMI

ANNOTATSIYA

Og'riq sindromi insult bilan og'rigan bemorlarning taxminan yarmida kuzatiladi. Bunday keng tarqalganligiga qaramay, bu insultning kam baholangan oqibatidir. Insult markaziy va periferik og'riqlari bilan ajralib turadi. Ko'pincha qon tomirida falaj bo'lgan oyoq-qo'llarning katta bo'g'imlarining artropatiyasi, mushak-skelet tizimining og'rig'i va og'riqli spastisit natijasida periferik og'riq kuzatiladi. Insultdan keyingi Markaziy og'riqlar insultdan keyingi bemorlarda kamroq uchraydi va murakkab mintaqaviy og'riq sindromi juda kam bemorlarda uchraydi.

Kalit so'zlar: murakkab mintaqaviy og'riq sindromi, insultdan keyingi markaziy og'riq, post-trombotik venoz sindrom, tizimli davolash usuli.

Дилбар Таджиевна Ходжиева
Мадина Зафаровна Охунджанова
Бухарский государственный медицинский институт

ПРЕХОДЯЩИЙ КОМПЛЕКСНЫЙ РЕГИОНАРНЫЙ БОЛЕВОЙ СИНДРОМ НА ФОНЕ ЦЕНТРАЛЬНОЙ ПОСТИНСУЛЬТНОЙ БОЛИ (ЛИТЕРАТУРНЫЙ ОБЗОР)

АННОТАЦИЯ

Болевой синдром наблюдается примерно у половины пациентов, перенесших инсульт. Несмотря на такую распространенность, является недооцененным последствием инсульта. По месту генерации выделяют центральную и периферическую боль. Чаще при инсульте наблюдается периферическая боль, возникающая в результате артропатий крупных суставов парализованных конечностей, скелетно -мышечной боли, болезненной спастичности. Реже у пациентов, перенесших инсульт, встречается центральная постинсультная боль и совсем редко описывается комплексный регионарный болевой синдром.

Ключевые слова: комплексный регионарный болевой синдром, центральная постинсультная боль, постромботический венозный синдром, системный подход к лечению.

Introduction. Central post-stroke pain (CPPS) refers to the neuropathic type of pain, which according to the International Association for the Study of Pain (IASP) is caused by a primary lesion or disease of the somatosensory nervous system [6]. True central neuropathic pain in the post-stroke period averages 8%. CPIB can develop when the spino-thalamo-cortical pathways are affected at any level. However, the leading place belongs to thalamic neurons, which can, under certain conditions, form a central pain generator and organize a pathological algic system. The main clinical manifestations of CPPS are hemialgia (intense pain in half of the body), hyperalgesia (increased response to the source of pain), allodynia (occurrence of pain sensations to stimuli that usually do not cause pain - passive movements, touch, temperature exposure). The treatment of CPPS is recommended to start as early as possible.The main directions of treatment of the analyzed pain syndrome are pharmacotherapy and psychotherapy. It should be noted that this type of pain is not sensitive to analgesics and nonsteroidal anti-inflammatory drugs. Pharmacological treatment of CPIB involves anticonvulsants (lamatridine, pregabalin, gabapentin, carbamazepine, phenytoin) and tricyclic antidepressants (amitriptyline). Amitriptyline (at a daily dose of 75 mg) and lamotrigine (up to 200 mg/day) are among the first-line therapies for central post-stroke pain 11a B. Their efficacy in CPIB has been proven in placebo-controlled randomized clinical

trials. Pregabalin, gabapentin, carbamazepine, and phenytoin may be recommended as second-line therapy for IIb B central post-stroke pain. The efficacy of these medications has not been sufficiently studied in CPPS, but they have been used successfully in other types of neuropathic pain [2]. Another variant of neuropathic pain that can be observed after a stroke is complex regional pain syndrome (CRPS) [4]. A distinctive feature of this pain syndrome is the presence of pronounced autonomic and trophic disorders caused primarily by microcirculatory disorders . Trophic disorders in cADS are observed in the skin and its appendages (edema, color changes, hair growth, nails), bones (osteoporosis development), joints (arthrosis and arthritis formation), muscles (up to muscle contractures formation), which leads to additional movement disorders. CPS developing during stroke does not meet the criteria for type I CPS (without signs of significant nerve damage) or type II CPS (with signs of significant nerve damage). The issue of distinguishing type III CPS remains open, and it is supposed to include diseases of the brain and spinal cord accompanied by CPS, including stroke [2, 5]. It is believed that in cADS, primary peripheral disorders are the triggering as well as supporting factors in the formation of pathophysiological transformations of spinal and cerebral levels. Central mechanisms play the main role in the implementation of CRPS [6]. The CNS dysfunction in cPSS is evidenced by the possibility of development in a number of patients of symptoms of the type of lack of sensory recognition, reminiscent of similar hemisensory disorders after a stroke. More than half of the patients with CRPS agreed with the following statements: "I feel like my painful leg is not part of my body" or "I have to concentrate completely on my painful limb to get it to move the way I want it to." The results of brain research using positron emission tomography and functional MRI in patients whose clinical manifestations of CRPS, combined with the above-mentioned disorders of perception and limb control, showed decreased activity of the thalamus (contralateral to the injured limb) [6]. Antidepressants and anticonvulsants are used for pain management, the leading syndrome-forming indicator of CRPS, just as in CPIB. There was no doubt about the neuropathic nature of the patient's pain, since there were all 4 signs that met the diagnostic criteria for neuropathic pain (see table). There also seemed to be no doubt that this pain was of central origin, arising after a stroke. However, to diagnose CPPS, in addition to the above-mentioned signs, an additional criterion is introduced - the exclusion of other potential causes of pain. T.M. Cherenko, in her review of the literature on post-stroke pain syndrome, notes that it is quite difficult to exclude other possible causes of pain, as often stroke is preceded by various chronic pain syndromes. A similar situation took place in our patient. Two foci capable of generating pain were identified in him. The substrate of one of them is cerebral disorders - a post-stroke cyst in the right thalamus, the structure modulating pain sensations. The second nidus causing peripheral nociception disorders is localized in the proximal parts of the left femur and is caused by tissue changes around the endoprosthesis and strombotic venous disorders. In spite of the fact that the mentioned foci form different types of pain - neuropathic and nociceptive, respectively, these pain generators cannot help interacting with each other. There are indications in the literature that even in peripheral post-stroke pain syndromes there are central pain mechanisms [5]. Somatogenic (nociceptive) pain is characterized by the formation of primary and secondary hyperalgesia zones. Primary hyperalgesia is based on sensitization - increase of nociceptors sensitivity to the action of damaging stimuli. The development of primary hyperalgesia induces central sensitization and the emergence of secondary hyperalgesia zones. Peripheral injuries trigger a whole cascade of pathophysiological processes affecting all nociceptive system from tissue receptors to cortical neurons, shifting the balance between anti- and nociceptive systems towards the latter, which is manifested by persistent pain [1, 2]. Permanent pain as a manifestation of persistent sensitization can arise only in case of severe damage of peripheral tissues or when the mechanisms of control of nociceptive signals conduction by the antinociceptive system are impaired. From the presented observation, we can see that the patient has both pronounced peripheral tissue damage in the left leg and damage to the central integrative structures that control and analyze pain sensations. On the other hand, we cannot exclude the contribution of the stroke to the existing peripheral changes in the paretic leg. Thrombosis of the femoral veins quite often complicates the course of stroke both in the acute phase and in the post-stroke period. Bones in the paralyzed limbs also undergo changes with the development of hemiosteoporosis. It is possible that in our patient, in addition to endoprosthesis, post-stroke bone disorders were also involved in the process of pronounced restructuring of the femur bone. The data presented suggest that a stroke may have contributed to the creation and maintenance of a peripheral focus of pain hyperactivation. The association between deep vein thrombosis, heterotopic ossification of the hip, and CRPS in the paralyzed leg 4 months after a hemorrhagic stroke has also been reported by other authors [3]. Addition of autonomic-trophic disorders to typical clinical manifestations of CPPS (burning pain, hyperalgesia, allodynia) indicated the development of CPS [4]. In the literature, there are indications of the formation of post-stroke CRPS in one of the paralyzed limbs (arm or leg), in our observation, autonomic-trophic disorders spread not only to both left limbs, but also to the trunk. The appearance of a complex of disorders with hemithypical mapping corresponding to the revealed cystic transformation of the right thalamus indicated that this was post-stroke CRPS. It is known that in the acute period of stroke, especially with a severe course of the disease, autonomic-trophic disorders in the paretic limbs are revealed. They can be without pain or accompanied by algic syndrome, as a rule, in large joints. In fact, these changes can be considered as CRPS. In our patient a recurrent stroke was excluded, but probably there was a phenomenon of locus minoris resistentiae as a reflection of trace reaction - one of the typical pathophysiological processes of the nervous system [1,4]. As G.N. Kryzhanovsky notes, this phenomenon can be realized under the action of new pathogenic factors, which disrupt compensatory mechanisms and intensify latent pathological changes. In our observation, such provoking factors were most likely the signs of inflammation (occurrence of hyperthermia and acceleration of CRP) of unspecified genesis. On the other hand, the development of rare, widespread, full-body CRPS variants, as in our patient, may reflect impaired central regulation of neurogenic inflammation [7]. CPIB frolicking as a result of lacunar infarction in the thalamus is a pathological algic system (PAS), the formation of which is indicative of profound dysregulation in the system of pain sensitivity, combined with insufficiency of the antinociceptive system. Probably, under these conditions one can expect extraordinary responses of the nervous system to additional pathogenic factors. Thus, our patient appeared distorted perception of the left limbs ("as if pumped up with a rubber ball"), without adequate visual assessment of swelling in them. These manifestations most likely indicate additional involvement in the previously formed PAS of the cortical sections of the right parietal lobe of the brain, which are responsible not only for analysis of general sensitivity, but also for perception of one's own body scheme. At the same time, joined CRPS can also cause similar sensations and change the activity of the thalamus [6], contributing to the dysregulation of brain activity. The transient nature of new clinical manifestations that arose in the patient in addition to CPIB remains unclear. Modern methods of functional neuroimaging, which allow assessing the systemic work of the brain by studying the connections (connectivity), have shown the complexity, unpredictability, and remoteness from the primary lesion focus of changes in the brain occurring in different diseases, including those after a stroke [3,4]. It is possible that PAS expansion or creation of a new pathological system on the type of CRPS in our observation was interrupted by both sanogenetic mechanisms and medications. In the complex and still understudied post-stroke period, clinical manifestations can be caused by bizarre interactions of pathophysiological and sanogenetic processes. G.N.Kryzhanovsky showed that physiological trophogens having sanogenetic effect can also cause pathological effects. In particular, repeated administration of the nerve growth factor in increased doses causes hyperalgesia, thermal and mechanical allodynia in rats. And, as the author notes, there is reason to believe that similar effects can occur in humans as well [1]. In this connection, it can be assumed that CPIB is partially caused by hyperreactivity of sanogenetic processes, especially since it is formed, as a rule, in the recovery period of a stroke. In order to relieve post-stroke pain syndrome the patient is recommended to take

anticonvulsants for a long time, if necessary to combine them with antidepressants. Both comorbid, stroke-related and polymorbid problems arising in the post-stroke period are beyond the scope of neurological solutions. Peripheral sanation, in particular, endoprosthesis replacement, is a necessary link in the patient's care; without this surgery he is unlikely to be able to move independently. On the other hand, such surgical intervention is a high risk of venous thrombosis and pulmonary embolism. At the time of discharge from the hospital, the question of surgery was postponed. Dual antithrombotic therapy under control of the clotting system and monitoring of the strombotic process were recommended.

Conclusions: Thus, two pathological foci (central and peripheral), capable of generating pain and inducing each other, were identified in the patient. The interaction of these foci resulted in the formation of a transient complex regional pain syndrome against the background of central post-stroke pain with extensive spread of autonomic-trophic disorders to the whole half of the body and development of impairment of sensory perception and visual control over his affected limbs.

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