FEATURES OF THE COURSE OF PAIN SYNDROMES IN POST-STROKE PATIENTS

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Summary: Vascular diseases of the brain continue to be one of the most important medical and social problems of modern society. Strokes are one of the most common causes of severe and long-term disability in most countries of the world. One of the complications of the post-stroke period is central post-stroke pain (CPS), which develops during the first year after stroke and occurs in 8% of patients.

Key words: central post-stroke pain, post-ischemic stroke, post-hemorrhagic stroke, localization of pain.

CPB can develop both after hemorrhagic and after ischemic lesions of the central nervous system (CNS). Some studies report a greater incidence of central pain after hemorrhagic strokes, which may be due to more frequent involvement of the thalamic region in hemorrhagic strokes.

Pain after a stroke can be classified in various ways. According to modern concepts, it is conditionally divided into three types of pain syndromes:

- central post-stroke pain;
- pain associated with damage to the joints of the paretic limbs - "pain shoulder syndrome";
- pain syndrome associated with painful spasm of the muscles of the paretic extremities [7].

The term "central post-stroke pain" refers to pain and some other sensory disturbances resulting from a previous stroke. CPPS is a neuropathic pain syndrome that develops after an acute cerebrovascular accident [5]. Loss of sensitivity, the presence of signs of hyper- or hypesthesia in the area of pain in patients with CPB indicates a combination of deafferentation with the subsequent development of increased neuronal excitability. central pain can also occur with extrathalamic lesions. The causes of these disorders are most often

heart attacks, hemorrhages, in particular due to rupture of arteriovenous malformations [2]. Despite the large representation of antinociceptive systems in the brain stem, its damage is rarely accompanied by pain. The frequency of development of CPB depends on the size, nature and localization of the focus. The localization of the central post-stroke pain can vary from a small (arm) to a large (half of the body) area. In patients with brain stem lesions, pain can be localized on half of the face and the opposite side of the trunk or extremities, and periorbital localization of pain has also been described [1].

The distribution of pain according to the hemitype is characteristic of a thalamic lesion. The defeat of the bridge and the lateral parts of the medulla oblongata (Wallenberg syndrome) more often than other structures, is accompanied by algic manifestations. However, the thalamus (posterior -ventral part) and the brainstem are the parts of the brain that are most often accompanied by CPB in stroke. When the focus is localized in the lower-lateral part of the thalamus, the frequency of development of CPB is relatively low - 12%. Age, gender, and side of the lesion are not constant predictors of CPB .

Clinically, CPSP is similar to neuropathic pain syndromes (both central and peripheral). There are no specific signs or common symptoms typical for the onset, clinical manifestations and intensity of CPB, and its characteristics and description by patients differ significantly, however, the nature of the patients' sensations may also contain important diagnostic information. Neuropathic pain is often perceived as a burning and/or shooting pain associated with an unusual sensation of tingling, crawling, or electrical shocks (dysesthesia). Patients describe spontaneously occurring constant pain as “burning”, “aching”, “stabbing”, “chilling” and “squeezing”, and paroxysmal - as “piercing” or “shooting”. Descriptions of the affective side of pain include characteristics such as "excruciating", "irritating", and "debilitating".

Clinical manifestations of central post-stroke pain and other central and peripheral neuropathic pain syndromes are similar. There are no pathognomonic characteristics or intensity measures for central post-stroke pain, and patient descriptions of pain vary significantly. Central post-stroke pain can be spontaneous or induced. Dysesthesia is noted in 85% of patients, and the average intensity on a ten-point scale varies from 3 to 6 [2]. In a

number of studies, the intensity of pain was higher when the lesion was localized in the brainstem or thalamus, however, according to other data, the severity of central post-stroke pain in thalamic and extrathalamic lesions did not differ [4]. The intensity of spontaneous pain often varies and may increase under the influence of internal or external factors, such as stress or cold, and decrease with rest or distraction. Constant spontaneous pain is described more often as burning, tingling, cooling and pressing, and periodic pain is tearing and shooting in nature [2,5]. It has been shown that central post-stroke pain worsens the quality of life of stroke patients, hinders rehabilitation, adversely affects sleep, and can lead to self-mutilation and suicide. Neuropathic pain, as a rule, does not prevent the patient from falling asleep, while he may suddenly wake up from intense pain. In the area of pain, trophic changes in the skin, subcutaneous tissue, hair, nails, impaired muscle tone, or local autonomic disorders can be observed.

Pain is a highly subjective experience that patients describe according to their specific symptoms. Therefore, standardized screening tools such as the Neuropathic Pain Inventory (Pain Detect , ID- Pain and DN4) are designed to classify pain based on patients' verbal description of pain characteristics. Most of these questionnaires include questions about the presence of burning pain, paresthesia, pain attacks, mechanical and thermal hypersensitivity and numbness [6].

Difficulties in diagnosing central post-stroke pain are due to a diverse clinical picture, the presence of different types of pain in one patient, and the lack of clear diagnostic criteria. Diagnosis should be based on the history taking, the results of a clinical sensitivity study, the use of neuroimaging methods (computed tomography or magnetic resonance imaging), and other clinical indicators (table) [3]. Previous stroke should be confirmed by neuroimaging data describing the nature, location, and size of the lesion, and other causes of central neuropathic pain should be excluded. In the process of taking an anamnesis, it is necessary to find out the characteristics of pain, the presence of dysesthesia or allodynia , as well as the exact localization of pain. Clinical examination should include sensitivity assessment to confirm and localize sensory disturbances and rule out other causes of pain.

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