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CLINICAL CASE OF REHABILITATION OF CENTRAL POST-STROKE NEUROPATHIC PAIN - DEJERINE ROUSSY SYNDROME.

Rasulova D.K.
RakhimbaevaG.S
Rasulova M.B.
Tashkent Medical Academy
Department of Neurology and Medical Psychology
Tashkent, Uzbekistan
Contact person: Rasulova Munisa
dmrasulova04@gmail.com

Abstract: One of the complications of acute cerebrovascular accident is "central post-stroke neuropathic pain" - CPB. In the article, we decided to describe a clinical case of Dejerine Russy's CPB syndrome, which was characterized by a syndrome of five hemi: hemianesthesia, hemiataxia, hemihyperpathia, causalgia, and a characteristic "thalamic hand". The goal of rehabilitation of post-stroke neuropathic pain is to reduce the intensity of causalgia as much as possible. Pain intensity was assessed using the VAS scale. Gabanerv was used for rehabilitation.

Keywords: Stroke, central post-stroke neuropathic pain, neurorehabilitation, VAS scale, Gabanerv.

Relevance of the problem: Stroke is one of the most important problems of modern society, being the main cause of deep and prolonged disability of the population. [1,2,4,5]. A stroke often leaves behind severe consequences in the form of motor, speech, cognitive, prorioceptive disorders, significantly worsening the social significance of patients and worsening their quality of life. One of the complications of acute cerebrovascular accident is central post-stroke neuropathic pain. The term "central post-stroke pain" (CPB) refers to pain and some other sensory disturbances resulting from a previous stroke. Dejerine and Roussy (1906) described intense intolerable pain in the form of a thalamic syndrome (superficial and deep hemianesthesia, sensitive ataxia, moderate hemiplegia, mild choreoathetosis) after infarcts in the region of the thalamic thalamus. Pain syndrome can develop soon after a stroke or after a certain time. In 50% of patients, pain occurs within 1 month after a stroke, in 37% - in the period from one month to two years after a stroke, in 11% - after two years [5]. In addition to gross neurological deficits, post-stroke pain significantly worsens the quality of life of patients, exacerbates depressive manifestations and makes it difficult to carry out rehabilitation measures, while significantly slowing down social adaptation. Pain, as a rule, is a heavy burden for the patient, even when its intensity is low. Complicating rehabilitation, disrupting sleep, CPB significantly impairs the quality of life and can lead to depression and even suicide. 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.

The frequency of development of CPB depends on the size, nature and localization of the focus. The defeat of the bridge and the lateral parts of the medulla oblongata (Wallenberg's syndrome) is more often than other structures accompanied by algic manifestations. However, the thalamus (posterior - ventral part) and the brainstem are the parts of the brain, the defeat of which in stroke is most often accompanied by CPB. 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 [1,2,4,5].

CPB belongs to the group of chronic pain disorders, which are combined under the concept of "central neuropathic pain" of the CNP, since the pain is caused by a focal lesion or dysfunction of the central nervous system of the CNS [2]. Given the complexity of the differential diagnosis of this syndrome and other pain disorders associated with CNS disorders, an alternative definition of central neuropathic pain has been proposed: pain that occurs as a direct consequence of damage or disease affecting the central somatosensory system [5]. Finnerap et al., as well as some others, described CPPS as a central neuropathic pain syndrome that occurs after a stroke in a part of the body corresponding to the focus and is characterized by pain and other sensory disorders. The International Association for the Study of Pain defines CNS as pain associated with CNS disease. Although some types of peripherally induced pain can also affect the activity of the CNS, the term "central post-stroke neuropathic pain" strictly means pain that is based on a primary process in the CNS [1].

According to the new classification of CNP, which can be used in stroke patients suffering from central neuropathic pain-CPNP and those whose pain is peripheral, the criteria for assessing each case are defined. They are based on the classification of neuropathic pain proposed by Treede et al. [2,4]. A case is defined as "possible CPNB" if criteria 1, 2, 3 are met, as "probable CPNB" if criteria 1, 2, 3 plus criteria 4 or 5 are met, and as "confirmed CPNB" if criteria 1-5 are met. The criteria are presented below.

1.Exclusion of other potential causes of pain.

2.The pain has a clear and anatomically substantiated localization (unilateral to the focus in the CNS on the body and/or face or unilaterally on the body with contralateral involvement of the face).

3.History of stroke (neurological symptoms developed suddenly, pain appeared simultaneously with the stroke or later).

4.Identification of clear and anatomically substantiated disorders during a clinical neurological examination (disturbance of sensitivity with a positive or negative sign in the painful area, pain is localized in the zone of sensory disorders, and the location of the zone of sensory disorders corresponds to the localization of the lesion in the CNS).

5.Identification of the corresponding vascular focus using neuroimaging methods (with CT or MRI, a focus is visualized, which can explain the localization of sensory disturbances).

Given the importance of knowing the symptoms of central post-stroke neuropathic pain in the rehabilitation of post-stroke disorders, we decided to describe the clinical case that we identified, described below.

The purpose of the study: To describe a clinical case of central post-stroke neuropathic pain - Dejerine Sott's syndrome in a patient with a stroke and its rehabilitation.

Patient B.A. born in 1963 59 years old entered the neurological department on 07.09.2022. disabled person 2. diagnosed with CVD. Consequences of repeated strokes (March 2016, March 03/17/2022), in the MCA basin on the right. Donkey: Left-sided spastic hemiparesis.

Complaints: weakness of the left arm and leg, numbness, burning pain on the left arm, feeling of coldness on the left limb, causalgia, hypersensitivity to mechanical and thermal stimuli on the left arm, inability to walk in the dark, cannot immediately recall familiar objects , confuses names, decreased memory and concentration.

From the anamnesis vitae: married, has children, a mathematics teacher at school, denies bad habits. Before the stroke, he was not registered with any doctors. Did not take antihypertensive drugs.

Anamnesis Morbi: He suffered his first hemorrhagic stroke at the age of 53, in 2016, on March 12, against the background of high blood pressure, weakness of the left limbs suddenly developed, he could not walk for a month. Retired on disability. After 6 years, the second stroke - on 03/17/2022 in the morning, weakness and numbness of the left side of the body suddenly appeared, he could not raise his left arm, dizziness was noted. Crawling in the kitchen took aspirin. Relatives called the ambulance, he entered the department of intensive neurology of the multidisciplinary clinic of the Tashkent Medical Academy. On the first day of hospitalization, the disorder of consciousness is in the form of stupor. Recovery after the second stroke in 2 months, that is, he began to walk slowly. In September 2022, he enters the department for rehabilitation treatment.

The general condition of the patient is moderate. In consciousness, adequate, oriented in place and space, signs of semantic aphasia are noted: difficulties in naming familiar objects. Central paresis of the VII and XII cranial nerves on the left, in the motor area left-sided spastic hemiparesis, with muscle strength on the left limbs, 3 points each. With outstretched arms forward in the paretic hand, athetoid hyperkinesis of the fingers, and the characteristic posture of the "thalamic hand" were noted. Tendon reflexes were elicited by BR, TR D<S high left, PR, AR D<S. Pathological reflex Babinski on the left, reflexes of oral automatism Marinescu-Rodovici and proboscis on both sides are called. In the sensitive area, left-sided hemihypesthesia, left hemihyperpathy, causalgia, sensitive ataxia. "Thalamic hand". Symptom Neglekt. With neuropathic pain, the patient revealed an area of impaired sensitivity and hypersensitivity in the affected area, which was combined with a skin zone of loss of sensitivity. Routine neurological examination of a patient with neuropathic pain included responses to touch, prick, pressure, cold, hot, and vibration. The reaction was assessed as normal, reduced or enhanced. Tactile sensitivity (response to touch) was assessed by lightly touching the skin with a piece of paper, the reaction to a prick was assessed by pricking the skin with a neurological pin. Deep sensitivity was investigated - the joint-muscular feeling of pressure on the muscles and joints. Vibrating - a tuning fork. Surface temperature sensitivity was assessed by the response to sensations of cold or hot in response to thermal stimuli. In the patient, the sensitivity test was accompanied by neurological symptoms such as hyperesthesia, dysesthesia, causalgia, hyperpathia, i.e., a feeling of numbness, changes in sensitivity to heat, cold and touch.

Data of laboratory and instrumental methods of examination of the patient: 1. Complete blood count: Hb - 110g/l; Erythrocytes - 3.9x1012 / l; Leukocytes - 5.4x109 / l; T / I - 2%, S / I - 51%, Monocytes - 5%, Lymphocytes - 42%, ROE - 6 mm / hour. VSK: 3.40-4.32.

2.General analysis of urine: quantity - 80 ml, color - yellow, transparent, relative density - 1010, protein - 0.033, Leukocytes - 12-14/1, Urates ++.

3.Coagulogram: Hemocrit number - 60%, heparin tolerance to plasma - 4.20, Fibrinogen - 353, Reference test - negative, Thrombotest - V.

4.Blood biochemistry: ALT - 34 U / l, AST - 17 U / l, total bilirubin - 32.8 μmol / l, bound - 16.2 μmol / l, free - 16.6 μmol / l, Urea - 8.8 mmol / l, Creatinine - 118.4 μmol /l, total oxygen strength - 78.7g/l.

5.RW, HbsAg, HCV - negative.

6.blood sugar: 4.8mmol/l.

7.Lipid spectrum: total cholesterol - 4.1mmol/l, triglycerides - 2.5mmol/l, HDL-C - 0.97mmol/l, LDL-C - 1.98mmol/l, VLDL-C - 1.14u, cholesterol coefficient - 3.2mmol / l.

8.ECG (09/17/2022): Sinus rhythm with a heart rate of 63 bpm. EOS is deflected to the left. P-mitrale. Incomplete blockade of the right leg of the Hiss bundle in combination with an incomplete blockade of the anterior branch of the left leg of the Hiss bundle. Signs of left ventricular hypertrophy with some circulatory disorders in the myocardium of the apical-lateral wall.

9.Chest x-ray (17.09.2022): Chronic bronchitis.

10.Examination of an ophthalmologist: vascular angiopathy.

11.Examination by a cardiologist: IHD. Stable exertional angina FS III. IKKS (EKG bjyich). Hypertension III stage. Arterial hypertension of the 1st degree. Risk IV (very high). Complication: SUE II A. FS III (according to NYHA).

12.Examination by a hepatologist: chronic hepatitis, unclear etiology, moderate activity. Fatty hepatosis II degree.

To determine the intensity of the pain syndrome, a visual analog rating scale was used - VAS, the objectification of the severity of which was important not only at the diagnostic stage, but also during the treatment process to assess the effectiveness of therapeutic measures. The patient described CPNB as long-term pain, which was documented. Pain in the study of CPNB was spontaneous due to the action of stimuli. When assessed on a scale from 0 to 10, the average pain intensity ranged from 3 to 6 points. According to the results of some studies, if the foci are localized in the brain stem or thalamus, the pain is more intense than when other areas are affected. According to the patient, the intensity of pain increased under the influence of stress or cold, and decreased after rest or distraction. Although nervous system involvement underlies all neuropathic pain syndromes, the pattern of sensory disturbances in the cutaneous lesion may vary between pathologies or even between patients with the same disease. Some patients experience spontaneous pain, paresthesias, and an electric shock sensation, while others experience hypersensitivity to touch or thermal stimuli in the affected area of the body. It is most likely that the individual pattern of sensory disturbances reflects the underlying mechanisms of pain generation and may also determine the cause of the differentiated and individual response to treatment.

In the classic thalamic syndrome described by Dejerine and Roussy, short-term hemiplegia or hemiparesis (paralysis or paresis of one half of the body) is observed, sometimes with subsequent incoordination of movements on this side after the disappearance of paresis, which was observed in our patient, a pronounced violation of the skin and deep sensitivity on the same side, and at the same time - unbearable, burning, diffuse, extremely intense pain in this half of the body, unilateral hyperpathy (the perception of any touch and other usually painless tactile sensations as extremely painful), as well as pronounced mood swings or depression up to suicidal thoughts or attempts.

Starting the patient's rehabilitation program, we identified all the patient's problems that he had, these are: motor, sensory, pain, speech and cognitive. Since the topic of our consideration of the clinical case is the rehabilitation of central post-stroke neuropathic pain - Dejerine Roussy syndrome, we will describe only the rehabilitation of pain. 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". The pain symptom we identified according to the classification of neuropathic pain proposed by Treede et al. Meets all criteria 1, 2, 3, 4 and 5, i.e. "confirmed CPNB". The patient's pain symptom has a clear and anatomically substantiated localization (on the extremities of the body and face, with a contralateral lesion); the anamnesis indicates repeated strokes (neurological symptoms developed suddenly, pain appeared simultaneously with a stroke or later); clear and anatomically justified disorders were revealed during a clinical neurological examination (disturbance of sensitivity of hemihypesthesia, hemihyperpathy, causalgia in the painful area, pain is localized in the zone of sensory disorders, and the location of the zone of sensory disorders corresponds to the localization of the lesion in the CNS); brain CT neuroimaging data - a focus is

visualized, which can explain the localization of sensory disturbances).

Rehabilitation - the treatment of the described clinical case of central post-stroke neuropathic pain - Dejerine Roussy syndrome, presented a difficult problem: in some patients with this pathology, it is not possible to achieve a pronounced reduction in pain. These difficulties in treatment are associated with the described heterogeneity of the mechanisms underlying neuropathic pain, as well as the frequent presence of psychological and emotional aspects of pain. At the first stage, we carried out the correct diagnosis, which helps to establish the cause of the pain. Starting curation of a patient with CPB, we informed him about the pathophysiology of the pain syndrome and the features of neuropathic pain. Together with the patient, realistic goals of therapy were determined - a decrease in the severity of pain by 30% was regarded as a clinically significant effect. This made it possible to save the patient from false expectations and set him up for the result.

The Neuropathic Pain Special Interest Group (NeuPSIG) was established by the International Association for the Study of Pain (IASP) to develop evidence-based guidelines for the pharmacological treatment of neuropathic pain. The recommendations of this group have been endorsed by the American, Canadian, Finnish and Mexican Pain Societies [5], the Federation of Latin American IASP chapters. Additional guidelines for the pharmacological treatment of neuropathic pain have been proposed concurrently by the Canadian Pain Society [2] and the European Federation of Neurological Societies [3,4,5,6]

According to the NeuPSIG recommendations published to date, tricyclic antidepressants, pregabalin, and gabapentin should be used as first-line therapy in patients with central neuropathic pain. The NeuPSIG guidelines support that a combination of drugs to treat neuropathic pain may be more effective than monotherapy, but it is associated with more side effects, less adherence to treatment, risk of drug interactions, and higher financial costs. Nevertheless, it should be taken into account that in ongoing studies, a satisfactory reduction in pain is achieved in less than 50% of cases, so sometimes a combination of drugs is necessary. Such combinations have been included in the recommendations for stepwise therapy for patients with a partial response to first-line treatment. The authors of the recommendations note that the choice of a particular drug in each individual case determines a combination of various factors, including potential risks of side effects, treatment of comorbidities (for example, depression and sleep disorders), the risk of drug interactions, the possibility of overdose or drug abuse, and cost. . For a patient with CPNB, we suggested the use of tricyclic antidepressants and Gabaner 300 mg, 1 tab x 1 time per day at 21.00. A week later, the pain decreased and was assessed on the VAS scale by 2 points.

Conclusion

In the rehabilitation of post-stroke movement disorders, knowledge and symptoms of central post-stroke neuropathic pain is important. It is one of the complications of acute cerebrovascular cerebral accident. The nature of the pain is described as Dejerine Sotta's five hemi syndrome: hemianesthesia, hemiataxia, hemihyperpathia, causalgia, thalamic arm. The task of rehabilitation therapy of post-stroke patients with motor, speech, pain, psychological disorders is the maximum possible improvement of impaired motor functions of paretic limbs, speech, reduction of pain syndrome with subsequent readaptation of patients to the environmental conditions that have changed for them. It is important to classify neuropathic pain according to the criteria proposed by Treede et al. As "confirmed CPNB", and assessed on the VAS scale to determine the effectiveness of therapy.

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