

# COPING WITH POST-STROKE PAIN

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Central post-stroke pain (CPSP) is a neuropathic pain syndrome characterized by constant or intermittent pain occurring after stroke and associated with sensory abnormalities in the painful body part. Sensory symptoms almost always develop on the side contralateral to the CNS lesion. Exceptionally, unpleasant sensations occur (with delayed onset) on the side ipsilateral to the lesion.

CPSP occurs in about 2-8% (1, 2) of patients following a stroke. A higher incidence of this type of pain was registered in aged subjects after a completed stroke (3). Central pain in stroke patients may begin with a different delay after a cerebrovascular accident. It is usually constant, but some patients may suffer from paroxysmal or episodic pain. CPSP is usually referred to as having a burning, aching, lancinating, pricking, lacerating, or pressing quality (1, 2). Its intensity is affected by external (e.g. changes in temperature) and internal events, body movements, visceral stimuli, emotions, and changes in mood. Anxiety and depression aggravate CPSP.

The cerebrovascular lesions causing central neuropathic pain are usually located in the thalamus, in the suprachiasmatic area, and in the lower brain-stem. Traditionally, the thalamic lesion has been considered to be the most frequent cause of CPSP. Apart from thalamic lesions, cerebrovascular lesions in many other locations have been shown to cause this type of pain, the most important being the posterior limb of the internal capsule, the subcortical and cortical zones in the postcentral gyrus, the insular region, and the lateral medulla oblongata (Wallenberg syndrome) (2). It has been generally agreed that damage to spinothalamic

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pathways plays a crucial role in the pathogenesis of CPSP. In strokes involving the brain-stem, a similar role is played by a lesion of the quintothalamic pathway. Several hypotheses have been formulated on the cause of central pain (4).

**Key words:** *post-stroke pain, central neuropathic pain, coping strategies*

Apart from pain, an abnormal temperature, touch and pain sensibility, and hyperpathia on the affected part of the body in patients with CPSP may be revealed (1). A rather neglected fact is the presence of allodynia together with central pain in about one quarter to more than half of patients with post-stroke pain syndrome (1, 5). In some cases, the type of allodynia depends on the lesion's location within the thalamus (5).

Pharmacotherapeutic options for CPSP have been limited till now. Drugs used for this complaint belong to groups of anticonvulsants, antiarrhythmics, opioids, and N-methyl-d-aspartate (NMDA) antagonists (6). Central pain shows a low sensitivity to opioids. Amitriptyline has been considered for a long time to be the drug of first choice. As for its alternative, an anticonvulsant lamotrigine was referred to as a well tolerated and moderately effective treatment for CPSP (7). In some subjects with CPSP who failed to respond to a variety of oral analgesics, gabapentin significantly reduced their pain (8). Tricyclic antidepressants suppress especially the continuous (constant) component of the pain, whereas anticonvulsants bring some effect in the treatment of brief, stabbing pain episodes. It was described that ketamine provided (by means of the blockade of NMDA receptors) significant pain relief to patients suffering from CPSP (9).

The use of neurosurgical methods is limited to subjects with resistant pain, who do not respond to pharmacotherapy. Deep brain stimulation (DBS) belonged to the scope of surgical management of chronic, refractory central neuropathic pain. Meta-analysis of this therapeutical

approach for pain suppression has revealed that the best results have been reached after the stimulation of the periventricular grey matter. The successful alleviation of central pain following DBS has been reported in up to 50-70% of post-stroke patients (10, 11). However, this method has been recently replaced by motor cortex stimulation (MCS) (12). MCS suppressed central pain in approximately 50% of patients with CPSP (13).

Pharmacological testing revealed that ketamine and thiamylal-sensitive (the latter is a member of the barbiturate group) and morphine-resistant cases displayed long-lasting pain reduction with chronic motor cortex stimulation (MCS) therapy (14). Therefore, it has been suggested that the pharmacological assessment of CPSP could be useful for predicting the effects of chronic motor cortex stimulation therapy.

Another type of post-stroke pain which has been repeatedly reported is a shoulder pain in hemiplegia. It has been described that about 40-60% of stroke patients suffer from hemiplegic shoulder pain (15). To prevent and alleviate this type of pain, therapeutic effort should be directed toward the proper positioning of the shoulder, range of motion activities, and the avoidance of immobilization. Transcutaneous electrical nerve stimulation (TENS) and functional electrical stimulation (FES) may be used for preventing and treating post-stroke shoulder pain, too (16).

Furthermore, another different type of post-stroke pain which has been described in paralyzed limbs is complex regional pain syndrome (CRPS) type 1. A wide range of its incidence (between 2% and 40%) has been described (17, 18). Its onset may be prevented by early inpatient rehabilitation.

Different coping strategies have been recommended and used for post-stroke pain suppression. It involved both the medical, and the psychological approaches. As with other chronic pain syndromes, psychological factors play an important role in the intensity of the CPSP. It is recommended that patients with CPSP consult with a psychologist specializing in the evaluation and treatment of chronic pain.

Generally, the most common were making the pain comprehensible, planning of activities, taking medication, communicating, and distractions (19).

## **Literature**

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## Streszczenie

Ośrodkowy ból poudarowy (Central post-stroke pain - CPSP) jest zespołem bólu neuropatycznego charakteryzującym się stałym lub okresowym bólem występującym po udarze mózgu z towarzyszącymi im zaburzeniami czucia. Objawy czuciowe prawie zawsze rozwijają się po stronie przeciwnej w stosunku do uszkodzenia OUN. W wyjątkowych przypadkach te nieprzyjemne sensacje (z opóźnionym początkiem) pojawiają się po stronie ipsilateralnej.

Dla zmniejszenia bólu poudarowego rekomendowane i używane są różne strategie. Obejmują one zarówno podejście medyczne jak psychologiczne. Tak jak w innych zespołach bólu przewlekłego czynniki psychologiczne grają ważną rolę w intensywności ośrodkowego bólu poudarowego. Zaleca się aby pacjenci z ośrodkowym bólem poudarowym konsultowali się z psychologami specjalizującymi się w badaniach i leczeniu bólu przewlekłego.

Generalnie najbardziej rozpowszechnione postępowanie to: zrozumienie istoty bólu, planowanie zajęć, zażywanie leków, informowanie i odwracanie uwagi od bólu.

**Słowa kluczowe:** *ból neuropatyczny, ośrodkowy ból poudarowy, strategie zwalczania*