Thalamic or central pain states poststroke

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Central poststroke pain (CPSP) has received a significant

CASE PRESENTATION

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Thalamic or central pain states are generally regarded as rare in stroke, occurring in fewer than 2% of patients. However, a recent study suggests that they may be more common, occurring in up to 8% of unselected stroke patients. Cerebrovascular lesions leading to central pain states do not necessarily involve the thalamus, but can occur following lower brainstem and supratthalamic lesions. Damage to the spinothalamocortical tract appears to be a prerequisite to the development of central poststroke pain (CPSP). Development of CPSP is likely related to denervation hyperexcitability of third or fourth order, thalamic or cortical neurons. Central pain is often described as a 'burning' sensation in association with an unpleasant association of tingling, pins and needles, or numbness. Spontaneous or evoked dysesthesia and allodynia/hyperalgesia are common. Central or thalamic pain is generally intractable to most therapeutic interventions. One case is presented to illustrate the typical clinical presentation of thalamic pain states and the difficulties in treating this pain.

Key Words: *Central pain, Deafferentation, Stroke*

Les états douloureux d'origines thalamique ou centrale à la suite d'un accident vasculaire cérébral

RÉSUMÉ : En général, on considère que les états douloureux d'origines thalamique ou centrale sont rares dans les cas d'accident vasculaire cérébral (AVC) et qu'ils se produisent chez moins de 2 % des patients. Pourtant, une étude récente laisse croire que ces douleurs seraient en fait plus courantes et qu'elles se produisent chez au moins 8 % des patients non sélectionnés ayant subi un AVC. Les lésions cérébrovasculaires qui entraînent des états douloureux d'origine centrale n'impliquent pas nécessairement le thalamus mais peuvent aussi survenir à la suite de lésions touchant le tronc cérébral inférieur et la région supra-thalamique. Les lésions touchant le faisceau spinothalamocortical semblent être une condition préalable au développement de la douleur d'origine centrale suivant un AVC. Le développement de ce type de douleur est vraisemblablement lié à une hyperexcitabilité de dénévation de troisième ou quatrième ordre de neurones corticaux ou thalamiques. La douleur d'origine centrale est souvent décrite comme une sensation de « brûlure » associée à une sensation désagréable de picotements, de fourmillements ou d'engourdissement. Des dysesthésies spontanées ou déclenchées et des hyperalgies-allodynies sont fréquentes. La douleur thalamique ou centrale est en général réfractaire à la plupart des interventions thérapeutiques. On présente un cas qui illustre le tableau clinique typique des états douloureux d'origine thalamique ainsi que les difficultés à traiter ce type de douleur.

amount of attention recently (1-9). One study reported that as many as 8% of all stroke patients had some form of CPSP. However, given the multitude of clinical impairments, disabilities or both suffered by stroke patients, it is not unusual for pain to be ignored or placed on the back burner (7). The original thalamic pain syndrome described by Dejerine and Roussy (10) characterized the thalamic pain syndrome as a thalamic lesion with slight hemiplegia, abnormal sensation, hemiataxia, hemiasoereognosia, intolerable pain and choreoathetoid movements (8). Thalamic or central pain states poststroke are generally characterized by burning, stabbing, knife-like pains and dysesthesia with allodynia and hyperalgesia frequently present.

A 65-year-old woman with a history of diabetes suffered a right-sided stroke that led to a minimal left hemiparesis and sensory loss. Motor difficulties associated with stroke completely resolved within weeks. However, almost immediately after the stroke she developed an uncomfortable 'cold spot' in the palm of her left hand. It spread to involve her entire hand and remained in that location for several months, after which it progressed to involve the entire left side of her body where it remained unchanged. She complained of an ice-cold sensation, which she described as like putting her hand in dry ice. The pain was described as burning, constant and steady; it was most severe in her hand, next most severe in her rib cage, followed by her thigh and then her ankle and foot. It bothered her least of all in her face, where it was located primarily in her cheek and a small patch on her forehead. She also described frequent intermit-

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CASE REPORT

tent stabbing pains; these came on spontaneously, lasting from seconds to minutes, and tended to be confined to the palm of her hand, left elbow and left rib cage. Light touch, such as clothing or even a light wind, caused discomfort along the entire left side of her body.

On examination there was some impairment of fine finger and fine toe movements on the left side, although strength was grossly normal and only minimally impaired. Over the entire left side of her body appreciation of light touch was altered. She found light touch uncomfortable over the left leg and arm, with the trunk less involved and the face least involved. Pin prick sensation was hyperpathic, tended to outlast the stimulus and had a burning, unpleasant quality to it; it involved the entire left side of the body with the exception of the cheek and forehead. Magnetic resonance imaging demonstrated a small infarct in the right upper midbrain and in the posterior and medial most aspect of the thalamus (Figure 1). Diagnosis was made of a thalamic pain syndrome. Treatment involved amitriptyline, carbamazepine and baclofen, as well as pain clinic-initiated intravenous xylocaine infusions. None of the treatments was successful, and she was subsequently treated with a mild narcotic with minimal pain relief. She continued to live at home independently.

DISCUSSION

Pathophysiology

CPSP is generally regarded as rare, occurring in fewer than 2% of cases (11-13), although a recent study reported an 8% incidence among unselected stroke patients with 5% reporting moderate to severe pain (8). Central pain resulting from a stroke is often referred to

as 'thalamic pain' despite that, in many patients with CPSP, the cerebrovascular lesions do not involve the thalamus (4,14-18). Leijon et al (4) noted that central pain states occurred following lower brainstem, thalamic and supratthalamic cerebrovascular events. CPSP is invariably associated with a lesion involving the spinothalamic pathway with a disturbance in temperature and pain sensation (8).

The pathophysiology of CPSP states remains unknown. It is becoming increasingly clear that damage to the spinothalamic pathway is a necessary prerequisite (1,7-10), although not all patients with damage to this pathway experience pain (8). CPSP is always associated with deficits in cold and warm stimuli and to pin prick sensation; these somatosensory functions are mediated by the spinothalamic tract (1,2,9). However, other sensory deficits, such as touch, two-point discrimination and vibration sense, generally regarded as mediated by lemniscal pathways in the central nervous system, although often involved in CPSP states, may also be intact (1,9). Vestergaard and colleagues (9) reported that lemniscal system lesions are not necessary for CPSP development.

Most, but not all, cases of CPSP are associated with hyperalgesia, allodynia or both. This paradoxical presence of a sensory deficit in combination with hyperalgesia in that part of the body deafferented by the stroke lesion suggests a central sensitization of third and fourth order central nervous system neurons as a result of loss of spinothalamic (or thalamocortical) input (9). Hyperexcitability of thalamic or cortical neurons could then evoke the perception of pain. Vestergaard et al (9) noted that this hypothesis shares many features thought to be characteristics of other neuropathic pain syndromes, such as those associated with peripheral nerve lesions where spinal cord neurons that have lost their afferent input develop a central hyperexcitability (19-21).

Clinical picture

Central pain is often described as a 'burning' sensation in association with an unpleasant association of tingling, pins and needles, or numbness (13). It often is described in terms such as ripping, tearing, pressing, twisting, aching, pricking and lacerating (1,4,8,13). Leijon et al (4), in their study of 23 patients with CPSP secondary to a known cerebrovascular lesion, noted little difference in the character of the pain in relation to the site of the lesion, with the exception that 'burning' pain was more commonly described with brainstem and supratthalamic lesions while 'lacerating' pain was seen more with the thalamic lesions. In their study of 16 patients with CPSP, Anderson and co-workers (8) noted no relation between size or location of the stroke and the presence of CPSP. CPSP pain is generally constant and often associated with spontaneous paroxysms of pain (1,4,13). It also can be exacerbated by physical movement, emotional stress, loud noises or voices, changes in the weather, cold and light touch (1,4,13).

Virtually all patients with CPSP report spontaneous or evoked paresthesia and/or dysesthesia (4,8). Spontaneous dysesthesia occur in the majority of CPSP patients while almost all demonstrate some hypersensitivity to an external somatic stimuli (4). Hence, the spontaneous pain seen in central pain states may be accompanied by further unpleasant effects induced by somatosensory stimuli known as hyperalgesia, allodynia and dysesthesia. Dysesthesia are defined as unpleasant sensations, either spontaneous or evoked (8). Allodynia

refers to an abnormally unpleasant somatosensory experience, often poorly localized, elucidated by normally non-nociceptive stimuli (8). Hyperalgesia is defined as an increased pain response to a normally painful stimulus (8). Initially hypersensitivity to sensory stimuli may make it difficult to differentiate CPSP from other pain entities, such as reflex sympathetic dystrophy, in the upper extremity. Anderson et al (8) noted that nine of 16 CPSP patients (56%) reported allodynia to cold stimulation while another nine (56%) reported allodynia to touch.

As discussed above, all patients with CPSP generally have some kind of sensory abnormality on the affected side (4). Decreased pin prick and threshold abnormalities to temperature detection are invariably present. However, touch, vibration and two-point discrimination are more variably involved. Allodynia and hyperalgesia have been discussed. Paralysis is not necessarily a feature of CPSP patients. Leijon et al (4) found that 14 of 27 patients (52%) with CPSP had no paresis, ten (37%) had moderate paresis and only four (15%) had severe paresis.

CPSP does not necessarily begin immediately after the stroke. In their review of 16 CPSP patients, Andersen et al (8) noted that 10 (63%) reported pain onset within one month of the stroke, three (19%) within one to six months and three (19%) after more than six months.

Treatment

Central pain is generally intractable to most therapeutic interventions. Narcotic and non-narcotic analgesics consistently fail to provide adequate pain relief (22). Tricyclic antidepressants have been shown to have a beneficial effect on central pain states (23,24). In the mechanisms through analyses of the sensory abnormalities. Pain 1989;37:173-85.

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one controlled study, amitriptyline was shown to have some pain ameliorating effect on CPSP patients (5). Phenothiazines (chlorpromazine) (25) and anticonvulsants (phenytoin [26,27] and carbamazepine [5]) are only minimally effective in reducing pain (17). Apomorphine has been reported to be effective but associated with significant adverse effects and a tendency to lose its effectiveness over time (28). Transcutaneous electrical nerve stimulation has proved to be effective in some CPSP patients (6). Sympathetic blockade in the form of stellate ganglion and lumbar sympathetic blocks or local venous guanethedine blocks may provide some temporary pain relief (15). A variety of operative treatments have been tried for central pain states. These include neurosurgical brain lesions (29-32), brain stimulation (33-34) and even stereotaxic chemical hypophysectomy (35). Overall, neurosurgical ablative procedures have demonstrated a 25% effectiveness in permanently relieving central pain states but are associated with a significant risk of brain injury (36).

CONCLUSIONS

A case of central or thalamic poststroke pain is presented. These conditions may not be as rare as previously thought. Damage to the spinothalamocortical tract appears to be necessary, with denervation hyperexcitability of cortical or thalamic neurons the most popular hypothesis for the pain. Spontaneous or evoked dysesthesia and allodynia/hyperalgesia are very common. The majority of cases are intractable to treatment.

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