Thalamic or central pain states poststroke

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Central poststroke pain (CPSP) has received a significant 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, hemiastereognosia, 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-
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 clininc-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 suprathalamic cerebrovascular events. CPSP is invariably associated with a lesion involving the spinothalamocortical pathway with a disturbance in temperature and pain sensation (8).

The pathophysiology of CPSP states remains unknown. It is becoming increasing clear that damage to the spinothalamocortical 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 hyperalgasia, allodynia or both. This paradoxical presence of a sensory deficit in combination with hyperalgasia in that part of the body deafferentated 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, prickling 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 suprathalamic 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 parasthesia 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 hyperalgasia, allodynia and dysesthesia. Dysesthesia are defined as unpleasant sensations, either spontaneous or evoked (8). Allodynia
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 ofstellate 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 sterotaxic 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 spinothalamicorcolateral 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.

REFERENCES

1. Boivie J, Leijon G, Johansson I. Central post-stroke pain – a study of
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