

Medial Medullary Infarct Aggravates Central Poststroke Pain Caused by Previous Lateral Medullary Infarct

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Key Words

Lateral medullary infarction · Medial medullary infarction · Central poststroke pain, pathogenesis

Abstract

Background: Although the pathogenesis of central poststroke pain (CPSP) remains obscure, interactions between the spinothalamic tract and lemniscal sensory tract may be involved in its pathogenic mechanism. **Case Description:** A patient developed lateral medullary infarction, which produced contralateral spinothalamic sensory deficits and subsequent CPSP. The CPSP gradually improved until the development of ipsilateral medial medullary infarction 26 months later, which was associated with mild hemiparesis and lemniscal sensory deficits. The ipsilateral medial medullary infarction immediately aggravated the CPSP to its previous level. **Conclusion:** This observation has implications regarding the pathogenesis of CPSP. CPSP may be related to hyperexcitation of the spinothalamic pathway by the reticulospinal system, which in turn is modulated by the medial lemniscus pathway.

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Approximately 25% of patients with lateral medullary infarction (LMI) develop central poststroke pain (CPSP) [1], an uncomfortable, frequently persistent pain or par-

esthesia after a stroke [2]. We describe here a patient who developed CPSP after LMI. The CPSP gradually improved until ipsilateral medial medullary infarction (MMI) developed, which aggravated the CPSP to its previous level. The pathogenesis of CPSP is discussed based on observations in this patient.

Case Description

A 55-year-old hypertensive man suddenly developed vertigo, diplopia and gait ataxia. Examination showed right-sided Horner sign, nystagmus, skew deviation, hoarseness and gait ataxia. Temperature and pinprick senses were decreased in the left arm, trunk and leg, to approximately 50% of those on the right side. Vibration and position sensations were normally perceived, and trigeminal sensation was spared. MRI showed an infarct in the right caudal lateral medulla (fig. 1A). MR angiogram showed severe stenosis of the right distal vertebral artery. Over time, his dizziness and gait ataxia gradually improved.

Two months later, the patient began complaining of cold and tingling sensations in his left arm and leg. Upon touch, dysesthesia (electrical sensation) was noted. The symptoms gradually worsened, and 2 months later, these uncomfortable sensations were scored on a visual analogue scale as 8 in the arm and 4 in the leg. The symptoms were severest in the left forearm, where he had increased perception of cold sensation. These symptoms were aggravated by cold weather, and he had to wear a glove while sleeping. He was prescribed amitriptyline 10 mg b.i.d., which partially improved the symptoms. One year after the onset of stroke, amitriptyline was discontinued due to his snoring and sleep apnea. Although he still had uncomfortable paresthesia in the arm and

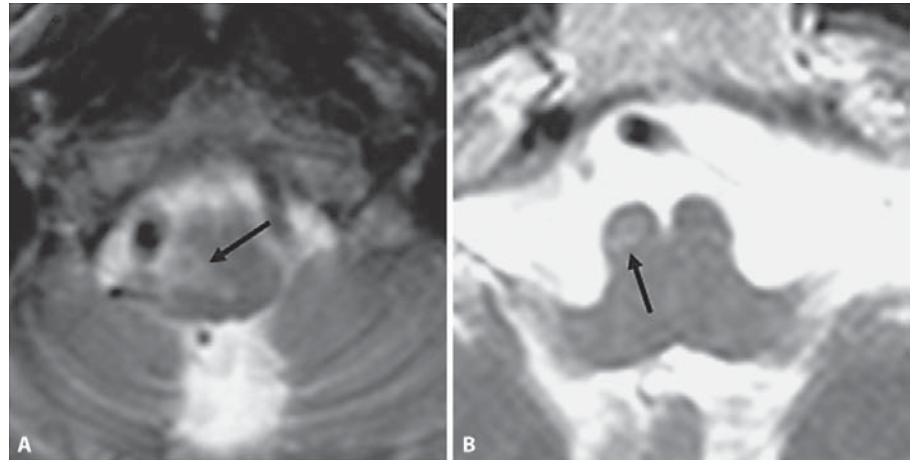


Fig. 1. T₂-weighted MRI shows an infarct in the left caudal lateral medulla (**A**, arrow) and rostral medial medulla (**B**, arrow).

leg, this was tolerable. He could sleep without wearing a glove. At this time, his sensations of discomfort were scored on a visual analogue scale as 4 in the arm and 2 in the leg.

The patient's sensory symptoms remained unchanged until 26 months after the first stroke, when he suddenly developed mild (IV/V) left hemiparesis. The facial and tongue muscles were spared. Muscle tone was normal and deep tendon reflexes were not increased. Sensory examination showed moderately (50% of normal side) decreased pinprick and temperature sensations in the left arm and leg. In addition, perception of position and vibration sensation was mildly impaired. MRI performed 3 days later showed a new infarct in the right rostral, medial part of the medulla (fig. 1B). Except for the shrunken previous lateral medullary lesion, there were no lesions in other parts of the brain including the thalamus or the pons.

Immediately after the new infarction, the patient complained that the cold and tingling sensations became severe in the left arm, trunk and leg. The visual analogue scale score was 8 in the arm and 4 in the leg. The nature and severity of the uncomfortable sensations were comparable to the worst sensory symptoms he had experienced before the second stroke. The motor deficits rapidly improved, and 2 months later, there was only slight clumsiness in the left limbs. However, the sensory symptoms persisted and were severest in the left forearm, where there was increased perception of cold sensations. These symptoms were aggravated by cold weather, and he again had to wear a glove while sleeping. Six months later, his sensory symptoms had not changed.

Discussion

After the LMI, this patient developed CPSP, which gradually improved. When another infarct developed in the ipsilateral medial medulla, however, the CPSP worsened to the previous level. Our observation has implications for the pathogenesis of CPSP.

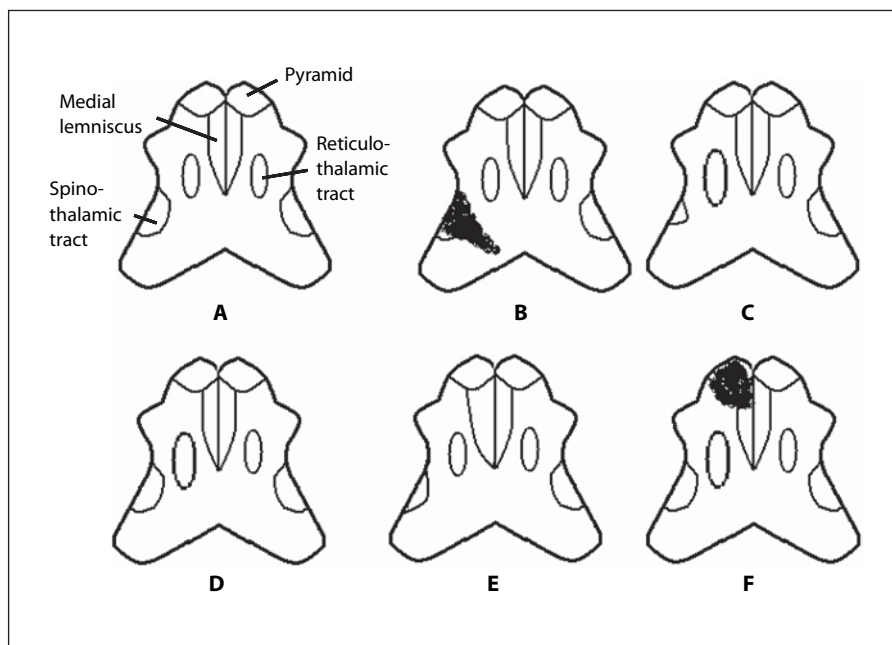
CPSP may be related to the development of hyperexcitation secondary to partially damaged sensory tracts,

damage to central inhibitor pathways or both [2]. Although the structural mediators related to CPSP remain uncertain, one possible candidate is the spinoreticulothalamic sensory system [3]. The spinothalamic tract and the spinoreticulothalamic tract have been shown to be interrelated, in such a way that the deafferentation of the former causes the normally nonexcitable spinoreticular tract to excite the spinothalamic system, producing CPSP [1, 3]. Moreover, there is evidence that the lemniscal sensory pathway inhibits the spinothalamic pathway [4], possibly through the spinoreticulothalamic system [5].

In our patient, the spinothalamic tract, initially damaged by the LMI, may have excited the spinoreticulothalamic system, producing excessive feedback and ultimately leading to CPSP. The gradual improvement of CPSP observed in this patient may be related to modulation of the spinoreticulothalamic system via the intact lemniscal sensory pathway. The subsequent MMI, involving the lemniscal pathway, may have disinhibited the spinoreticulothalamic system, which in turn aggravated CPSP to its previous level (fig. 2). Because the MMI appeared to be located ventrally, the more dorsally located lemniscal pathways may not have been involved. However, the presence of decreased vibration and position sensation at the second admission suggests involvement of the lemniscal pathway.

Because the patient developed hemiparesis, it is possible that concomitant spasticity, joint problems or depression secondary to newly developed motor deficit may have aggravated the CPSP. However, the motor deficit was mild, and no spasticity or muscle contracture was observed. Moreover, the motor deficits rapidly improved while the sensory complaints persisted. Therefore, the aggravated CPSP was unlikely to be related to motor def-

Fig. 2. Schematic drawing of the hypothetical mechanism of aggravated CPSP. **A** Normally, the spinothalamic tract is regulated by the reticulothalamic system, which in turn is inhibited by the lemniscal sensory tract. **B** An initial infarct damages the spinothalamic tract to make it hypofunctional. **C** The reticulothalamic tract is excited, stimulating the spinothalamic tract. **D** With the excitatory input of the reticulothalamic tract, the spinothalamic tract is compensated, but in an abnormal manner, to produce CPSP. **E** The hyperfunctional reticulothalamic tract stimulates the lemniscal pathway, which in turn inhibits the reticulothalamic system. Consequently, the spinothalamic tract becomes less functional and the intensity of central pain decreases. **F** Another stroke damaging the lemniscal tract disinhibits the reticulothalamic tract, and the hyperfunctional reticulothalamic tract aggravates CPSP to the previous level.



icits or concomitant depression. Because CPSP may develop after MMI, the sensory symptoms may have been produced by the MMI itself and may not have been related to the previous CPSP. However, CPSP due to lemniscal pathway involvement is different from that caused by a spinothalamic lesion, in that it is usually expressed as numbness and is rarely aggravated by cold environments or accompanied by increased cold perception [6]. CPSP due to MMI has also been considered a phenomenon of disinhibition of the spinothalamic pathway through the reticulothalamic system [6]. Therefore, in this patient, the disinhibition may have been expressed as an aggravation of the previous CPSP.

Finally, CPSP has been reported to develop when there is a loss of normal inhibition of pain by cold sensations in

the general setting of loss of thermal sensations [7]. Our observations in this patient do not support this hypothesis, because pain and cold sensations were decreased to a similar degree, and there was an increased perception of cold at the time of maximal CPSP.

Acknowledgments

This study was supported by research funding from the Korean Ministry of Health and Welfare (03-PJ1-PG1-CH06-0001) and a grant from the Brain Research Center of the 21st Century Frontier Research Program funded by the Ministry of Science and Technology of Korea (M103KV010010 06K2201 01010).

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