Isolated numbness of the tip of the tongue in hemispheric stroke

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Although the somatosensory afferent of the tip of the tongue runs proximal and parallel along the spinothalamocortical and trigeminothalamocortical pathway up to the sensory cortex, central involvement has been rarely described in cases of nongustatory sensory disturbance at the tip of the tongue. In a hypertensive woman who experienced an acute onset of an isolated numbness at the tip of her tongue, recent small infarctions were found at the postcentral gyrus of the right parietal lobe. Thus, central involvement should not be neglected in the case of sensory disturbance at the tip of tongue. (Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;111:e11-e14)

Nongustatory sensory disturbance (NgS) restricted to the tip of the tongue (TT) is not an unusual complaint in clinical dental practice. Although typically findings restricted to the tip of the tongue have been characterized as gustatory sensory disturbances, the NgS disturbances have not been fully described in the literature. Until now, NgS of TT has been sporadically reported to arise from a broad spectrum of local or systemic conditions, such as allergic response, focal trauma, transoral intubation,1 intraoral postoperation, tongue thrust,2 drugs, intoxication, vitamin deficiency, systemic lupus erythematosus, and others. Conversely, it is rarely mentioned that numbness to the TT can result from central involvement. This rarity conflicts with the neuroanatomical parallelism that is found among 3 cortical tracts (linguotrigeminocortical, spinothalamocortical, and trigeminothalamocortical) in the brain and suggests that there may be an underestimation of NgS of TT in clinical practice. This case report describes a woman encountered in our practice who suffered an isolated numbness of TT and was then diagnosed with a right parietal lobe infarction. The findings of this numbness to the TT reminds clinicians to be alert for the possibility of central neurological involvement when unusual symptoms like this are present.

CASE REPORT

A 50-year-old right-handed woman experienced an acute onset of numbness at her TT 3 days before visiting our hospital. She felt numbness but did not experience any burning sensation or pain, and the numbness was not exacerbated by lingual movement or acidic food intake. The numbness did not spread to the oral mucosa, palate, teeth, or gums. She denied performing any antecedent vigorous activity or having any history of emotional change. There was no other associated discomfort. She could move her limbs freely and speak fluently. At that moment, her blood pressure was 124/76 mm Hg. She had a 2-year history of hypertension, and was regularly receiving antihypertensive medication. She denied having migraine, reversible focal neurological deficit, preceding infection, recent craniofacial injury, vaccination, or an intraoral procedure. She did not consume cigarettes, alcohol, or illicit drugs.

She visited the outpatient department because of a persistent numbness of TT. On presentation, her vital signs were stable. She was conscious and cooperative. Higher cortical function test did not disclose disorientation or other abnormal finding. Her eyegrounds did not show papilloedema. Higher cortical function, speech and language, pronunciation, swallowing, cranial nerve function, brainstem reflexes, motor activity, coordination, and equilibrium were intact. Her tongue consistently showed a significantly decreased sensation to pinprick pain and touch, which extended up to 0.5 from the tip on both sides. However, her responses to tastes like sweetness, saltiness, sourness, and bitterness were symmetric and had not changed. The outer appearance of her tongue was normal. There was no oral ulcer, mass, or color change. Her tongue showed a significantly decreased sensation to pinprick pain and touch, which extended up to 0.5 from the tip on both sides. However, her responses to tastes like sweetness, saltiness, sourness, and bitterness were symmetric and had not changed. The outer appearance of her tongue was normal. There was no oral ulcer, mass, or color change. The proprioceptive and exteroceptive sensation of face, body, and limbs, and cortical sensation were symmetric without any remarkable abnormality. The temporal artery was not tender or palpable, and jaw movement was normal. No synkinetic facial movement was observed. Xerostomia was absent and her mood was stable. Burning mouth syndrome was not favored. Therefore, isolated numbness of TT was interpreted.
Blink reflex showed a normal ipsilateral and contralateral response. Head magnetic resonance imaging (MRI) revealed 2 small diffusion-weighted imaging (DWI), T2-weighted, and fluid-attenuated inversion recovery (FLAIR)-weighted intensities, compatible with recent infarction, in the right parietal lobes located at the postcentral gyrus (Fig. 1). Recent infarction was considered. Stroke risk factor survey did not show any abnormal findings, other than thalassemia minor. Ferric profile was normal. Antibodies to antinuclear factor, ds-DNA, nuclear extract antigen, and beta2-glycoprotein I were within the respective reference ranges. A diagnosis of ischemic stroke was established. After discussion, acetylsalicylic acid was prescribed for prophylaxis. The numbness of her TT subsided progressively within 2 weeks.

DISCUSSION

With regard to neurological involvement, NgS of the TT has only been reported to arise from a few peripheral neurological disorders, such as chronic inflammatory polyneuropathy, autoimmune-mediated sensory neuropathy, auriculotemporal neuropathy, lingual neuropathy, and central neurological disorders, such as stroke, multiple sclerosis, and depression. It may occur either in isolation or in combination with other neurological deficits. The findings in our patient implicate that there is a possibility of central involvement for NgS of TT and these findings can help in making an accurate diagnosis in clinical practice.

Fig. 1. Magnetic resonance imaging of the head showed 2 small DWI (A, C) and FLAIR-weighted intensities (B, D), compatible with recent infarction, at the right parietal lobes located at the postcentral gyrus (lesion 1: A-B, lesion 2: C-D). Lesions are marked by arrows.

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The somatosensory impulse of the tongue is conveyed via the lingual nerve and it reaches the thalamus and insular cortex together via the spinothalamocortical and trigeminothalamocortical pathway. In cases of pontine and thalamic lesions, the sensory disturbance of both the contralateral region of the tongue and TT can occur independently. This sensory presentation suggests that there are 2 types of site-specific somatosensory afferents of the tongue. The first type of afferent carries the somatosensory sensation on 1 side of the tongue from the lingual nerve and then enters into the ipsilateral trigeminal nerve. Thereafter, it follows the trigeminothalamocortical pathway to initially descend into the upper cervical cord, then crosses and ascends into the contralateral thalamus and insular cortex. An involvement of this afferent can cause somatosensory disturbance of the contralateral hemitongue.

The second type of afferent carries TT afferents. Sensory disturbance of the TT involves only a small area of the bilateral anterior tongue tip. Electric stimulation of the insular or parietal cortex in humans factually produces sensory disturbance of the contralateral hemitongue and TT separately in different but nearby areas, and at the perisylvian fissure/postcentral gyrus in both hemispheres. Similar sensory responses have also been identified at the superior temporal gyrus in previous studies. These findings suggest that the TT has an independent representation area in both hemispheres of the brain. Therefore, it is not surprising that a small unilateral infarction at the postcentral gyrus can provoke numbness of TT without any other sensory deficits, as observed in our patient. Thus, physicians should be alert to the possibility of central involvement in cases of isolated NgS of TT, especially when classic risk factors, such as stroke or autoimmune disease are present.

As the TT afferents run proximal and parallel to other somatosensory ascending tracts, there are doubts about the relatively uncommon incidence of NgS of contralateral tongue or TT in comparison with other facial or somatic structures. Previous studies have shown that the sensory representation area of the tongue, including the TT, is disproportionally larger than that of other structures, whereas the threshold of cortical sensory neuron (at the postcentral gyrus) is lower than the cortical motor neuron (at the precentral gyrus) but does not exhibit dermatomal selectivity. In addition, curvation of somatosensory strip and multiple sites of supplemental representation areas of the tongue are also important static factors for a sensory outcome upon incitement. Nevertheless, although these factors may explain the intercooperative model of the cheiro-oral syndrome, they still cannot explain why NgS of the TT is relatively uncommon and occurs in isolation from other dermatomes.

Instead of the static cline suggested by the theory of specific location and corresponding responses, the mosaic theory sketches a dynamic hemostasis of neuronal activity, which is responsible for individual intervariation in response to stimuli. In different individuals, electric stimulation in the same location at the cortex can provoke different sensory patterns in the tongue, such as isolated numbness of the TT, contralateral hemitongue, or the whole tongue. Hemispheric specification or dominance of visceral sensation of the tongue is not unusual. These findings support the existence of individual intervariation for somatosensory expression of the TT. In contrast with the traditional concept, short-term variations in response to cortical stimulations are not only an all-or-none reaction, but they may also factually depend on the complexity of the preexisting condition, local brain states, remote brain effect, and functional brain architecture that is, the neural plasticity. In our patient, the preexisting hypertension may have provoked silent, minor, and undetectable brain damage that may have changed the neural plasticity. In view of these static and dynamic functions and activities of neurons, an isolated numbness of the TT as seen in our patient should not be arbitrarily considered as an incidental finding. In fact, this strengthens the concept of the need for personalized care in both diagnosis and treatment.

REFERENCES

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