Transient Hemiglossal Denervation during Acute Internal Capsule Infarct in the Setting of Dysarthria—Clumsy Hand Syndrome

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SUMMARY: A case of MR imaging—documented transient unilateral tongue denervation presenting during acute internal capsule infarction is described. Understanding the corticolingual pathway innervation of the hypoglossal nucleus is essential for explaining these findings. Awareness of the findings in this case will facilitate appropriate diagnosis, provide neuroanatomic explanation, and prevent misdiagnosis.

Case Report

A 73-year-old man with chronic hypertension presented with acute dysarthria and right arm weakness. Physical examination also revealed right facial droop and right arm incoordination. The tongue and uvula were midline without fasciculations or atrophy. The clinical diagnosis was DCHS. MR imaging performed at 1.5T revealed a focal area of restricted diffusion at the posterior limb of the left internal capsule, indicating acute infarction (Fig 1A). Additionally, the right hemitongue was markedly diffusely swollen and hyperintense on T2-weighted imaging with right-sided posterior prolapse into the oropharynx (Fig 1B). Fatty atrophy was not identified on T1-weighted images. The brain stem was normal.

Repeat MR imaging with gadolinium performed 2 days later to assess possible tongue mass revealed persistence of restricted diffusion in the posterior limb of the left internal capsule but with resolved right hemiglossal swelling and T2 hyperintensity and resolved posterior prolapse (Fig 2). There was no abnormal tongue enhancement or brain stem abnormality.

After 1 year, the patient had persistent ataxic right hemiparesis with spasticity. The tongue was midline with spasticity.

Discussion

Hypoglossal nerve dysfunction, often with tongue atrophy, is well known to occur secondary to lower motor neuron injury, and hypoglossal palsy due to supranuclear infarction has rarely been reported. We report a case of transient reversible hemiglossal denervation edema on MR imaging, contralateral to acute internal capsule infarction in the clinical setting of DCHS. A neuroanatomic basis is discussed.

While permanent hypoglossal denervation is well known to occur subsequent to lower motor neuron injury, hypoglossal palsy due to supranuclear infarction has rarely been reported. We report a case of transient reversible hemiglossal denervation edema on MR imaging, contralateral to acute internal capsule infarction in the clinical setting of DCHS. A neuroanatomic basis is discussed.
The transient nature of the tongue edema in this case presumably represented a reversible or compensated event that could be explained by reperfusion, by partial denervation with recruitment or re-establishment of latent neural pathways, or by transient voluntary inaccessibility of ipsilateral uncrossed corticofacial projections during the acute phase of the infarct.\(^5\)\(^6\)

**Conclusions**

Hemiglossal denervation is not always due to lower motor neuron injury, can occur with supranuclear infarction, and can be transient and reversible. Awareness of the existence of individually variable bilateral crossed and uncrossed corticofacial projections facilitates a neuroanatomic explanation for these findings. In the future, one could postulate a potential role for diffusion tensor fiber tractography and functional MR imaging in the assessment of normal and impaired corticofacial pathways and lingual motor response.

**References**


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**Fig 1.** A 73-year-old man with acute onset of right arm weakness and dysarthria. *A,* Diffusion-weighted image (b=1000) reveals focal hyperintensity at the posterior limb of the left internal capsule, indicating acute infarction. Hypointensity was present on an apparent diffusion coefficient map (not shown). *B,* Axial T2-weighted image obtained at the time of acute infarction shows diffuse right hemiglossal edema with swelling and posterior prolapse.

**Fig 2.** Axial T2-weighted image obtained 2 days later shows resolved right hemiglossal swelling and edema and resolved posterior prolapse.