Isolated Hypoglossal Nerve Palsy After Cervical Manipulation at the Dentist

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INTRODUCTION

Isolated and unilateral hypoglossal nerve palsy secondary to head and neck trauma is uncommon. The reasons for its infrequency include anatomy, clinical presentation, and mechanism of injury. The hypoglossal nerve is intimately associated with other critical structures of the neck and skull base, making isolated injury difficult to achieve. Because it is a purely motor nerve, the only presenting symptom will be weakness of half the tongue. It may not declare itself immediately as the other side is able to compensate. Finally, the forces required to produce this injury are often significant. A hypoglossal palsy can easily be missed due to the prominence of other symptoms seen with concomitant lower body damage.

There is very little documentation of minor cervical trauma as the cause of this type of injury. We present a case report demonstrating a unique diagnosis of inflammatory neuritis after a dental visit.

CASE PRESENTATION

The patient is a 72 year old male with no relevant medical history who went for a routine dental appointment in June of 2016. During the visit his dentist had difficulty clearing a bridge from his left jaw, rapidly snapping his neck from extension into flexion. A week later he noted tongue swelling and went to an outside hospital where he was evaluated for a stroke. He had an MRI, MRA and CT scan at that time which showed no evidence of infarct or vascular abnormality .

He presented to our tertiary care facility one month later. His primary complaint was dysphagia, with difficulty chewing on the left side of his mouth. He denied dysarthria. Review of systems was positive for back pain.

On exam, the left half of his tongue was atrophic and deviated to the ipsilateral side with protrusion. This can be seen in Figure 1. He had mild prominence of lingual tonsillar tissue on the left, but no evidence of infiltrative tumor. Flexible endoscopy was performed and the nasopharynx, oropharynx, and hypopharynx were clear. Both vocal cords were mobile bilaterally. The remainder of cranial nerves II through XI were intact.

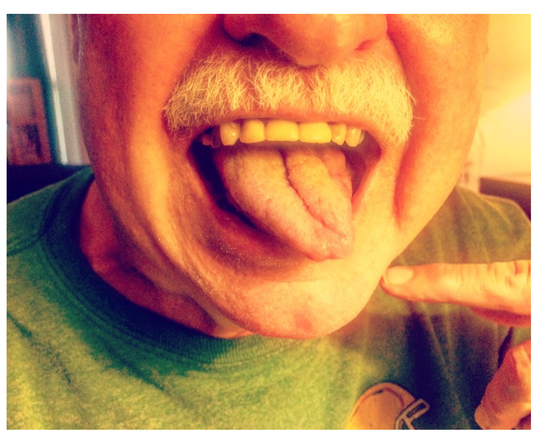

Figure 1. Initial clinical photo of patient. Atrophy of left hemitongue with associated deviation.

RADIOLOGY REVIEW

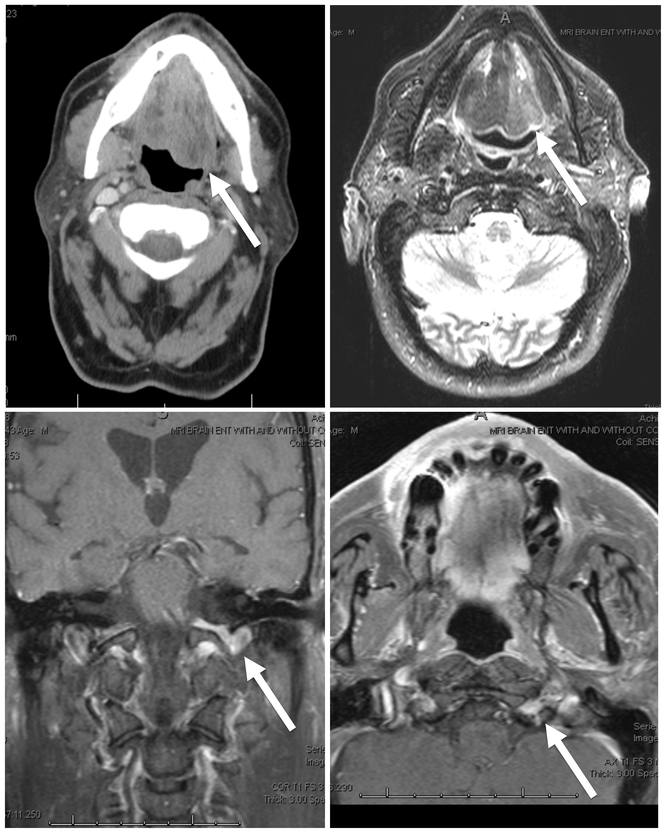

Figure 2 (top left) Axial view of CT scan. Arrow points to asymmetric protrusion of left tongue base into oropharynx. Figure 3 (top right) Axial view of MRI showing T2 phase. Arrow points to denervation atrophy with hyperintense signal of the left hemitongue. Figure 4 (bottom right) Axial view of MRI in T1 post contrast phase. Figure 5 (bottom left) Coronal cut of MRI in T1 post contrast phase. Arrows point to asymmetric enhancement of the left hypoglossal nerve within the canal.

The imaging performed at the outside institution was reviewed by our radiologist. He noted asymmetry of the tongue with inflammation on the left side representative of myositis. There was also asymmetric protrusion of the left tongue base into the oropharynx. This is apparent on the CT scan shown in Figure 2. On MRI, the left side of the tongue demonstrated denervation atrophy with hyperintense T2 signal as seen in Figure 3. No lesions were visible at the skull base or along the course of the hypoglossal nerve. There was osteophytosis throughout the cervical spine but upon further review, the osteophytes were distant from the entire length of the hypoglossal nerve.

Because the quality of the previous scans was limited, it was decided to repeat the MRI with finer cuts and closer attention to the skull base. A scan in August showed similar changes to the soft tissue of the tongue. However, we also found the left hypoglossal nerve enhancing throughout the entire course of the canal. This is shown in the axial cut on Figure 4 and coronal view on Figure 5. The canal itself was not enlarged or distorted. The proximal and distal portions of the nerve appeared normal. Again there were no masses or lesions found.

A follow-up MRI was performed in November. Interestingly, the tubular enhancement of the left hypoglossal canal was no longer present.

DISCUSSION

The most cited mechanism of hypoglossal nerve palsy due to head and neck trauma is fractures of the occipital condyle. Due to anatomic proximity, with the hypoglossal canal located superior to the occipital condyle, it is vulnerable to injury. It is also theorized that hyperextension of the atlanto-occipital joint makes the hypoglossal nerve susceptible to harm[123]. The skull rests on the spinal atlas at the occipital condyle and excessive traction at this junction can impinge on the canal above. Our patient describes a story that mimics whiplash and his imaging is found to support this rational.

There is no established protocol for the evaluation of isolated hypoglossal nerve palsy, with CT and MRI both frequently performed. The radiologic findings of the denervated tongue in our case are consistent with reports in the literature[4]. In general, there is a phase progression of muscle denervation that corresponds to characteristic findings on an MRI. The early acute phase shows paradoxical hypertrophy and muscle edema, corresponding to T2 hyperintensity as in Figure 3. The late acute phase has a mixture of edema and atrophy. The chronic phase involves only atrophy and fatty infiltration, represented by T1 hyperintensity.

There are a few cases that describe spinal osteophytes compressing on the nerve or canal to cause this palsy[56]. Our patient suffered from chronic back pain and had evidence of bony abnormalities on neuroimaging. While there was no relation of these osteophytes to the area of interest he did suffer from cervical spine disease. We cannot rule out a component of underlying arthritis with a stenotic canal contributing to this case.

The most unique feature involves the hypoglossal nerve within the canal. The combination of nerve swelling, high T2 signal and contrast enhancement confirm the diagnosis of inflammatory neuritis.

CONCLUSION

The etiology remains unknown as to whether this was a traction injury, an arthritic flare or a combination of both. This is the first case report to describe hypoglossal inflammatory neuritis developing as a result of minor cervical manipulation. It supports the idea that excessive traction at the atlanto-occipital joint can lead to impingement of the hypoglossal nerve within the canal. The literature supports a good prognosis for patients with benign, isolated, unilateral hypoglossal nerve palsy.

He was most recently seen in April 2017, nine months after the initial incident. His atrophy and deviation had significantly improved. Tongue mobility was almost back to normal. His exam is portrayed in Figure 6. We will continue to follow him clinically with serial MRI scans and predict a full recovery in function.

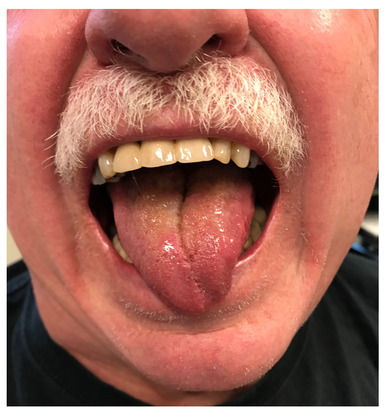

Figure 6. Recent clinical photo of patient. Improved atrophy and deviation.

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