Isolated Unilateral Hypoglossal Nerve Palsy Due to Vertebral Artery Dissection

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**Abstract**

We report a patient with unilateral tongue weakness secondary due to an isolated lower motor neuron hypoglossal nerve palsy, which was caused by a right vertebral artery dissection in the lower neck. The patient had a boggy tongue with a deviation to right side but an otherwise normal neurological examination. MRA presented narrow lumen of the right vertebral artery in the neck. After initially treating the patient with aspirin in ER and later with warfarin for three months, there was complete recanalization of the right vertebral artery. Only one other case has been reported before with this vertebral artery dissection and twelfth nerve palsy.

**Keywords:** Hypoglossal Nerve, Vertebral Artery Dissection, Tongue Weakness, Lateral Medullary Syndrome, Dysarthria
**Introduction**

The hypoglossal nerve (CN XII) is the motor nerve of the tongue. Isolated hypoglossal nerve paralysis is uncommon. Vascular, inflammatory, traumatic or space-occupying lesions can affect CN XII anywhere in its course from the nucleus in the caudal medulla, through the intracranial extramedullary portion of the nerve, across the hypoglossal canal or into its extracranial termination. The rootlets cross the premedullary cistern superior to vertebral artery and exit the skull base through the hypoglossal canal. This close relationship can cause compression of rootlets intracranially when there is vertebrobasilar pathology or anatomic variants of vertebral artery resulting in paralysis of the nerve. The rootlets join to form one or two trunks before piercing through the dura. The nerve then descends almost vertically to a point corresponding with the angle of the mandible. In the neck it is deeply seated beneath the internal carotid artery, internal jugular vein, and vagus nerve. Lower in the neck the nerve emerges superficially just below the digastric muscle.

**Case Report**

A 20-year-old female presented to the emergency room (ER) with sudden onset of slurring of speech, lasting for about an hour. This was preceded by a two week period of neck pain. The patient took tylenol and ibuprofen with limited relief. There was no history of trauma, nausea, vomiting, headache, blurring of vision, double vision or dysphagia, but she felt dizzy five seconds before symptoms appeared. On examination, the patient was normotensive, dysarthric and afebrile and she was a two on the NIH stroke scale. The dysarthria was due to difficulty in tongue movement. She could not repeat “Methodist Episcopal”, “Huckleberry Finn” and “fifty fifty” but was able to repeat “Mama”, “Tip Top”. She had a boggy tongue, enlarged on left side which led to...
difficulty in movement. Her tongue deviated to the right side on protrusion. With consent a picture of the tongue movement was obtained [Fig. 1A]. The remaining CNs examination and the rest of her neurological examination was unremarkable.

A Laboratory examination with complete blood count and electrolytes assessment was normal. Antithrombin III and lupus anticoagulant were normal. While protein C was lower than expected. A computed tomography of the head showed no intracranial abnormality. The patient was treated with aspirin initially in the ER, and was transitioned to warfarin for 3 months. Diffusion weighted magnetic resonance imaging (MRI) of the brain was normal but magnetic resonance angiography (MRA) of neck showed narrowing of the right vertebral artery. The vertebral artery narrowed approximately 2 cm from its origin until the level of C2 suggesting vertebral dissection [Fig. 2B & E]. A CT angiogram of the neck confirmed right vertebral artery dissection, exhibiting a double lumen, with the true lumen narrowed throughout its course as suggested above. [Fig. 2F]. Both bilateral carotids and left vertebral artery were normal.

**Discussion**

This case is unique as unilateral isolated CN XII palsy is uncommon when vertebral artery dissection occurs outside the premedullary cistern. Upper motor neuron and nuclear lesions are usually bilateral due to the close proximity of CN XII nuclei. In our patient there is no evidence of extension of the extracranial dissection into the intracranial vasculature. Therefore direct compression of CN XII rootlets was unlikely. There was no subarachnoid blood on CT scan and our patient did not want a lumbar puncture. The
more plausible cause for CNXII infarction was distal embolization from extracranial vertebral artery to the vaso nervorum of the distal intracranial arteries.

The patient’s hypoglossal nerve palsy weakness improved [Fig. 1B] with aspirin and warfarin. There was no recurrence of her symptoms in the aftermath of the dissection. No tongue atrophy was seen at one month [Fig. 1B] nor at her three month’s follow up appointments [Fig. 1C]. Our patient did not have lateral medullary syndrome as there was no vertigo. Diffusion weighted image (DWI) showed the lateral portion of medulla oblongata was normal[Fig. 2A, C & D]. She had no sensory deficits affecting the face or the extremities on the contralateral side, no dysphagia or pain on the ipsilateral side of the face and her corneal reflex was intact. There was no Horner syndrome, loss of pain and temperature sensation.

CN XII is closely related to both the carotid and vertebral arteries, and it has been described in the setting of various vascular pathologies.\(^3,4,6\) The proximal, cisternal portion of CN XII is mainly supplied by vertebral branches.\(^4,6,7\) CN XII receives its distal blood supply predominantly from the carotid circulation via the ascending pharyngeal artery, the occipital artery, the external carotid artery and the lingual artery.\(^4,6\)

Most cases of vertebral artery dissection are thought to be spontaneous events. However, dissection can be precipitated by minor trauma, or by spinal manipulation. Connective tissue disorders such as fibromuscular dysplasia, Marfan’s syndrome and Ehlers-Danlos syndrome type IV increases the risk for cervical arterial dissection.\(^4\) Vertebral artery dissection is an important cause of posterior circulation ischemia, particularly in young
and middle-aged patients. Features of lateral medullary syndrome and neck pain, vertigo, nausea and vomiting are common. None of these were present in our subject. Treatment with warfarin may be more effective than aspirin in preventing recurrence of ischemic symptoms.

Conclusion
We describe an unusual case of isolated unilateral hypoglossal nerve palsy due to vertebral artery dissection in the lower neck, which was reported only once before. In our case clinical and neuroimaging evidence led us to conclude that embolization from the extracranial vertebral artery to the vaso nervorum of the distal intracranial arteries resulted in the subject's CN XII infarction.

Learning Points
- This case highlights the need for complete neurological examination in young patients and some knowledge of differential diagnosis.
- In a young patient with cranial nerve palsy, always consider the possibility of stroke.
- Vertebral branches mainly supply the proximal, cisternal portion of hypoglossal nerve.

Diagnosis of dissection of vertebrobasilar artery may reduce complications of posterior fossa infarction and subarachnoid haemorrhage.
References


Figure 1.

A. Tongue Deviated to Right (LMN IX Palsy)

B. Improved Tongue Deviation at 4 weeks

C. Tongue Normal at 3 months

Figure 2.

A. Normal Medulla (DWI)

B. Abnormal Vertebral (R)

C. Normal FLAIR

D. Right Vertebral at Medulla

E. Dissected R. Vertebral Wall (MRI Source)

F. Dissected Right Vertebral (CT Angio)

G. Complete occlusion of the vertebral at 3 months