◆ CASE REPORT —

Recurrent Laryngeal Nerve Injury Following Uncomplicated Carotid Angioplasty and Stenting

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Purpose: To describe a unique instance of cranial nerve injury related to uncomplicated carotid artery stenting (CAS).

Case Report: A 74-year-old woman with a history of expressive aphasia and right upper/ lower extremity weakness underwent staged CAS procedures under local anesthesia and conscious sedation. After routine predilation with a 4-mm balloon, a tapered $7 \times 10 \times 30$ -mm Acculink stent was placed and dilated with a 5-mm balloon. At 1 month after the second procedure, the carotid stents were patent bilaterally, but the patient reported voice fatigue and hoarseness along with dysphagia to liquids that started 2 days after her second procedure. Brain scans ruled out stroke. Direct laryngoscopy showed left vocal cord paralysis and a mobile right vocal cord; computed tomography revealed adduction of the left vocal cord consistent with a left recurrent laryngeal nerve injury. Radiography did not show any evidence of stent fracture. Electromyography was suggestive of right recurrent laryngeal nerve paralysis and only mild abnormalities on the left. A repeat laryngoscopy performed 4 months after the initial evaluation revealed persistent left vocal fold paralysis and no abnormalities on the right. The patient was referred for voice therapy; at 18 months, the stents were patent, and her vocal symptoms had significantly improved.

Conclusion: While minimally invasive endovascular techniques evolve for management of vascular disease, the anatomical structures at risk during open procedures may be injured with endovascular approaches as well.

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Key words: carotid artery, stent, carotid artery stenting, complication, recurrent nerve injury, vocal cord paralysis

Most of the scientific literature pertaining to cranial nerve injuries and carotid proceduresfocuses on technical aspects of open endarterectomy that can result in cranial and

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cervical nerve injury. One of the purported advantages of carotid artery stenting (CAS) is the theoretical decreased risk of cranial nerve injury.^{1,2} Potential injury to the cranial nerves during open cases is usually related to direct trauma from dissection, transient injury from blunt trauma or retraction, electrocautery thermal injury, or direct disruption of the blood supply to the nerve. Recent reviews of the literature examining the incidence of cranial nerve injury following open carotid endarterectomy suggest that cranial nerves

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XII, X, and VII are the most commonly involved.^{1,3–5} The actual incidence of temporary recurrent laryngeal nerve injury in the setting of open carotid endarterectomy ranges between 0.5% and 4%.^{6–8} Permanent vocal cord paralysis, defined as lasting longer than 6 months, rarely occurs in the few series examining the incidence of recurrent laryngeal nerve injury following open carotid endarterectomy. Spontaneous recovery of vocal cord function occurs in a majority of cases, and significant symptoms related to vocal cord paralysis are rare.⁶

Although the incidence of cranial nerve injuries with CAS is expected to be negligible, 3 (1.1%) cranial nerve injuries were recorded in a multicenter randomized trial of endarterectomy versus stenting in patients with symptomatic severe carotid stenosis (EVA-3S).² One case of Horner's syndrome was likely precipitated by a carotid dissection during angioplasty. The other 2 patients were converted to open procedures; it is unclear whether the hypoglossal nerve palsies they experienced were recognized before or after open conversion.

In this report, we present what may be the first case of symptomatic recurrent laryngeal nerve injury after uncomplicated carotid stent placement.

CASE REPORT

A 74-year-old woman with a history of a transient ischemic attack (TIA) consisting of expressive aphasia and right upper and lower extremity weakness was diagnosed with bilateral internal carotid stenosis on duplex ultrasound. A severe (70%-99%) stenosis was noted on the right, with a peak systolic velocity (PSV) of 292 cm/s and end diastolic velocity of 103 cm/s. There was a moderately severe (50%-69%) stenosis on the left, with a PSV of 207 cm/s and end diastolic velocity of 65 cm/s. Risk factors included symptomatic coronary artery disease, hyperlipidemia on statin therapy, hypertension, and a history of smoking. Aspirin and clopidogrel therapy was initiated after the TIA.

Given her symptoms and findings, the patient was advised to undergo carotid angioplasty and stenting and was enrolled in CAPTURE 2 (Post Approval Study of the Guidant Carotid Stent Systems and Embolic Protection SysJ ENDOVASC THER 2009;16:345–348

tems) using the Accunet/Acculink system (Abbott Vascular, Santa Clara, CA, USA). The symptomatic left-sided lesion was treated first using cerebral protection; the patient was discharged the next day without any complications. A follow-up duplex at 1 month revealed a widely patent left carotid stent without any hemodynamically significant stenosis and an unchanged severe right carotid stenosis. The right side was similarly treated 6 weeks later in an uncomplicated procedure. Both procedures were performed under local anesthesia and conscious sedation and consisted of routine predilation with a 4-mm balloon, placement of a tapered $7 \times 10 \times 30$ -mm Acculink stent, and postdilation with a 5-mm balloon. Follow-up at 1 month after the second procedure revealed patent carotid stents bilaterally. Although asymptomatic immediately following the second procedure, the patient reported voice changes including voice fatigue and hoarseness along with dysphagia to liquids starting 2 days after discharge. Neurological examination and computed tomography (CT) of the brain ruled out any strokes.

The symptoms persisted and prompted further evaluation. The patient denied any previous intubations, neck operations, or previous voice changes. Direct laryngoscopy showed left vocal cord paralysis and a mobile right vocal cord. The patient was placed on a proton pump inhibitor for reflux. A CT scan of the neck revealed adduction of the left vocal cord (Figure, A), which was consistent with a left recurrent laryngeal nerve injury. Plain radiographs (Figure, B) of the cervical spine did not show any evidence of stent fracture. A needle electromyogram (EMG) to further evaluate vocal cord function was performed. Interestingly, it was suggestive of right recurrent laryngeal nerve paralysis and only revealed mild abnormalities on the left. A repeat laryngoscopy performed 4 months after the initial evaluation revealed persistent left vocal fold paralysis and no abnormalities on the right. The patient was referred to a voice specialist to pursue voice therapy and possible future vocal cord injection. Follow-up at 18 months showed patent stents and significantly improved vocal symptoms, including voice fatigue without any odynophagia or dysphagia. The patient did not agree to further workup

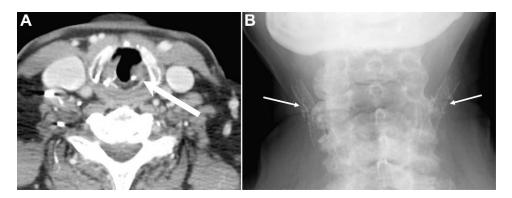


Figure \blacklozenge (**A**) Adduction of the left vocal cord (arrow) on a CT scan of the neck. (**B**) Plain radiograph of the stents (arrows) without evidence of stent fracture.

or interventions with laryngoscopy, EMG studies, voice therapy, or vocal cord augmentation given her improved symptoms.

DISCUSSION

This is an unusual case of a cranial nerve injury following staged bilateral CAS. There are several postulated mechanisms by which this injury may have occurred. Stretching of the carotid artery with balloon angioplasty could result in localized tissue inflammation and edema, which could lead to local compressive effects within the carotid sheath and resultant vagal nerve and recurrent nerve dysfunction. Also, hematoma formation secondary to microperforation could lead to similar consequences, although none was visualized on duplex ultrasound in this case. A carotid sheath hematoma may have resolved by the time the patient was re-imaged. CT imaging did not show any evidence of aneurysm formation resulting in local compression. Neuropraxia of the vagus nerve or its branches from this type of local tissue injury would be expected to result in only a transient hoarseness. Endotracheal intubation injury with resultant vocal cord paralysis has been described and may be due to local edema from cuff inflation or direct trauma to the vocal fold. However, both procedures in this case were performed under conscious sedation, which is typical of most CAS patients.

A less likely etiology would be a potential embolic phenomenon from plaque within the carotid artery resulting in nerve ischemia. The time interval between the last stent placement and almost immediate onset of symptoms may indicate a minor acute injury to the right recurrent laryngeal nerve from balloon angioplasty, possibly in the setting of an undiagnosed chronic left recurrent laryngeal nerve injury. The left cord paralysis may have been chronic but asymptomatic and not related to nerve conduction, so a minor CAS-induced injury on the right was therefore enough to cause symptoms. This would be the most likely explanation of a visualized left vocal cord paralysis with EMG findings favoring a dominant right recurrent nerve injury. Her complaints of dysphagia, cough, and globus sensation occurring after the (second) right-sided procedure would be explained by a compromised right recurrent nerve's sensory function as the primary sensory nerve supplying the glottis.

Local compression on the left recurrent larvngeal nerve has been described in the setting of transcatheter coil closure of a patent ductus arteriosus in infants.^{9,10} Similar mechanisms of overstretching of the vessel or placement of too large a stent for the vessel size and impingement on the nerve could be implicated, although the stents were appropriately sized in this case. Moreover, stent fractures could cause strut injury to the surrounding structures by creating a microperforation, although this was not documented in this patient. Alternatively, while reaction to nitinol is theoretically possible, this has been poorly described in the literature and does not explain the patient's unilateral symptoms. While there exist several other neurological etiologies of vocal cord paralysis

(e.g., infection, local primary tumor, central nervous system pathology, peripheral neuropathies, collagen vascular diseases), none applies to the current case.

Few series have examined patients in a prospective manner undergoing open endarterectomy with preoperative and postoperative direct vocal cord examination.¹¹ In these reports, a significant subset of asymptomatic patients have evidence of vocal cord immobility on examination.^{6,12} Thus, the actual incidence of vocal fold paralysis may be underestimated in CAS since only symptomatic patients are evaluated with direct laryngoscopy.

Conclusion

This is an unusual case of a cranial nerve injury following uncomplicated CAS. As endovascular interventions in various vascular beds continue to evolve, it is important to note that the anatomical structures at risk during open procedures can be vulnerable during noninvasive interventions as well. While one of the reported advantages of CAS is the decreased risk of nerve injury, this is likely underreported due to a low index of suspicion. Several mechanisms can explain cranial nerve injury during CAS. Systematic patient evaluation is needed, however, to better characterize the pathophysiology involved.

REFERENCES

1. Coward LJ, Featherstone RL, Brown MM. Safety and efficacy of endovascular treatment of carotid artery stenosis compared with carotid endarterectomy: a Cochrane systematic review of the randomized evidence. *Stroke*. 2005;36:905–911.

- Mas JL, Chatellier G, Beyssen B, et al. Endarterectomy versus stenting in patients with symptomatic severe carotid stenosis. N Engl J Med. 2006;355:1660–1671.
- 3. Maroulis J, Karkanevatos A, Papakostas K, et al. Cranial nerve dysfunction following carotid endarterectomy. *Int Angiol.* 2000;19:237–241.
- Sajid M, Vijaynagar B, Singh P, et al. Literature review of cranial nerve injuries during carotid endarterectomy. *Acta Chir Belg.* 2007;107: 25–28.
- Zannetti S, Parente B, De Rango P, et al. Role of surgical techniques and operative findings in cranial and cervical nerve injuries during carotid endarterectomy. *Eur J Vasc Endovasc Surg.* 1998;15:528–531.
- Curran A, Smyth D, Sheehan S, et al. Recurrent laryngeal nerve dysfunction following carotid endarterectomy. *J R Coll Surg Edinb*. 1997;42: 168–170.
- Espinoza F, Macgregor F, Doughty J, et al. Vocal fold paralysis following carotid endarterectomy. *J Laryngol Otology*. 1999;113:439–441.
- 8. Cunningham E, Bond R, Mayberg M, et al. Risk of persistent cranial nerve injury after carotid endarterectomy. *J Neurosurg.* 2004;101:445– 448.
- Hwang M, Su W. latrogenic cardiovocal syndrome caused by transcatheter coil closure of patent ductus arteriosus. *Acta Pediatr.* 2005; 94:372–374.
- Liang C, Ko S, Huang S, et al. Vocal cord paralysis after transcatheter coil embolization of patent ductus arteriosus. *Am J Heart*. 2003; 146:367–371.
- Ballotta E, DaGiau G, Renon L, et al. Cranial and cervical nerve injuries after carotid endarterectomy: a prospective study. *Surgery*. 1999; 125:85–91.
- Cahill L, Murdoch B, McGahan T, et al. Perceptual and instrumental evaluation of voice and tongue function after carotid endarterectomy. *J Vasc Surg.* 2004;39:742–748.