Jaw claudication in the era of carotid stenting

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Jaw claudication could result from external carotid artery (ECA) occlusive disease. Carotid artery stenting (CAS) has been shown to worsen the disease in the ECA. This could potentially worsen the symptoms in patients with pre-existing jaw claudication undergoing CAS. Meanwhile, ECA endarterectomy is routinely done during internal carotid artery endarterectomy (CEA). This has been shown to alleviate jaw claudication symptoms. We report a case of a high-risk patient for CEA who presented with symptomatic carotid disease as well as bilateral jaw claudication. Both symptoms resolved after CEA. We also present the case of another patient treated for recurrent high-grade carotid disease with CAS resulting in acute ECA occlusion and jaw claudication. High-risk patients with symptomatic carotid disease and jaw claudication should be considered for CEA and not only CAS. (J Vasc Surg 2011;54:526-8.)

Carotid endarterectomy (CEA) has been the traditional operative treatment of carotid occlusive disease. With the advent of endovascular technology, carotid artery stenting (CAS) has since emerged as a less invasive alternative to CEA.[1] Traditionally, CEA involves endarterectomy of both the internal carotid artery (ICA) and the external carotid artery (ECA). With CAS, stents are deployed in the ICA and extend across the bulb to the common carotid artery (CCA) to prevent recurrent stenosis at the bulb. In doing so, the stent traverses the orifice of the ECA, defined as overstenting. Both in vitro[2] and in vivo[3] studies have demonstrated abnormal flow in the ECA when the stent is extended across the orifice of the ECA. In addition, significant progression of stenosis in the ipsilateral ECA compared with the untreated contralateral ECA has been reported.[4]

A branch of the ECA, the maxillary artery, supplies the masseter and temporalis muscles responsible for jaw movement and chewing. Occlusion of the ECA resulting in symptomatic jaw claudication has been previously documented.[5-9] When jaw claudication happens after CAS from ECA occlusion or stenosis, it typically is transient and resolves completely. However, patients with pre-existing jaw claudication will most likely have the symptoms either persist post-CAS, or become worse if the ECA occludes. Jaw claudication is not life-threatening to patients with occlusive carotid disease, but bothersome, resulting in changes in their eating habits on daily basis. We present two cases of jaw claudication in a patient with pre-existing ECA and symptomatic ICA disease, the second patient suffered jaw claudication after acute ECA thrombosis following CAS and angioplasty. In the era of CAS, patients with jaw claudication and severe ECA/ICA atherosclerotic disease should be considered for CEA.

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CASE REPORTS

Patient 1. This patient was a 60-year-old male who presented to the emergency room at the Michael E. DeBakey Veterans Affairs medical center in Houston, Tex, with left ophthalmic transient ischemic attack occurring daily for 3 days. He was admitted to the neurology service for further workup. He was already on aspirin 81 mg daily and Clopidogrel 75 mg twice a day for a history of hypercoagulable state. He has a history of homocysteinemia and factor V Leiden gene mutation resulting in both arterial and venous thrombosis in the past. His medical history is also significant for diabetes, chronic obstructive pulmonary disease, and congestive heart failure with ejection fraction of 15% to 20%, and he is a heavy ex-smoker. His left eye symptoms resolved since he has been an inpatient on heparin drip, and a magnetic resonance imaging brain was negative for evidence of acute stroke. The patient also informed us of his severe bilateral jaw claudication, with the left side worse than the right. He particularly reported left jaw claudication after 10 to 15 bites and after 30 to 40 bites on the right side. This was of concern to the patient as he was avoiding eating solid food or he had to mash the food on his plate before chewing. The carotid ultrasound on admission showed atherosclerotic disease in both the left ICA and ECA (Table). The computed tomography scan showed near occlusion of the left ECA (Fig 1) with disease-free bilateral maxillary arteries.

This patient would have qualified for CAS because of his comorbidities, under the high-risk category.[10] His sedimentation rate was 9 mm/hr (normal <20 mm/hr), so his jaw claudication was related to his ECA near occlusion. After discussion with the patient, we elected to proceed with CEA to help alleviate the jaw claudication symptoms as well as to treat the symptomatic ICA disease. The second day after admission, he underwent left CEA under local anesthesia with ECA eversion endarterectomy. He had focal ICA disease but significant ECA disease that resulted in a long-segment eversion ECA endarterectomy (Fig 2). His postoperative course was uneventful, and he was discharged home 2 days later. His postoperative carotid ultrasound surveillance at 6

Table. Carotid ultrasound result

	Initial presentation and at 6-month follow-up Post-CEA			
	Right side Pre-CEA	*Right side Post-CEA*	*Left side Pre-CEA*	*Left side Post-CEA*
CCA PSV (cm/s)	61	60	60	89
CCA PDV (cm/s)	14	16	15	22
ICA PSV (cm/s)	198	128	230	103
ICA PDV (cm/s)	63	41	84	35
ECA PSV (cm/s)	148	149	409	93

CCA, Common carotid artery; *CEA,* carotid artery endarterectomy; *ECA,* external carotid artery; *ICA,* internal carotid artery; *PDV,* peak diastolic velocity; *PSV,* peak systolic velocity.

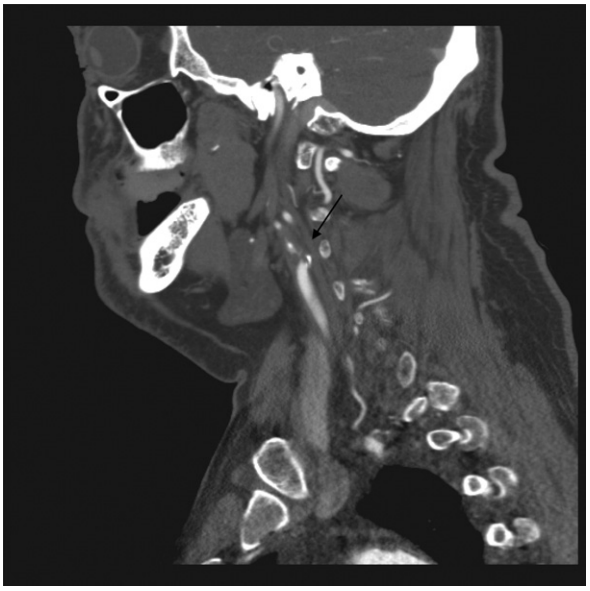

Fig 1. Computed tomography scan showing near occlusion of left external carotid artery (ECA; *arrow*) in a patient with symptomatic left internal carotid artery (ICA) stenosis and jaw claudication.

months shows successful endarterectomy in both the left ICA and ECA (Table). One year later, he remains symptom free without left jaw claudication.

Patient 2. A 72-year-old male presented to our clinic at the same hospital with asymptomatic recurrent high-grade left ICA stenosis. He has a history of multiple risk factors for atherosclerotic disease and underwent left CEA for symptomatic left ICA stenosis 15 years earlier. He underwent a left ICA stent that resulted in acute left ECA occlusion after stent angioplasty (Fig 3). As a result, he suffered from left jaw claudication that lasted for 2 months. He avoided eating solid food (mainly meat) for those 2 months and avoided chewing food on the left side. Carotid ultrasound 2 years later shows no recanalization of the left ECA and no significant disease in the right ECA.

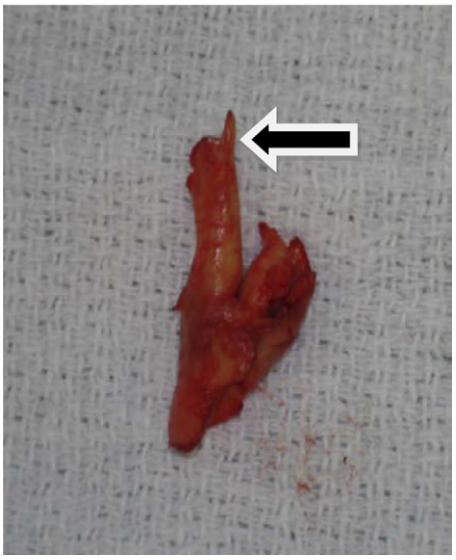

Fig 2. Carotid endarterectomy specimen. *Arrow* points to external carotid artery (ECA) plaque and one of its branches.

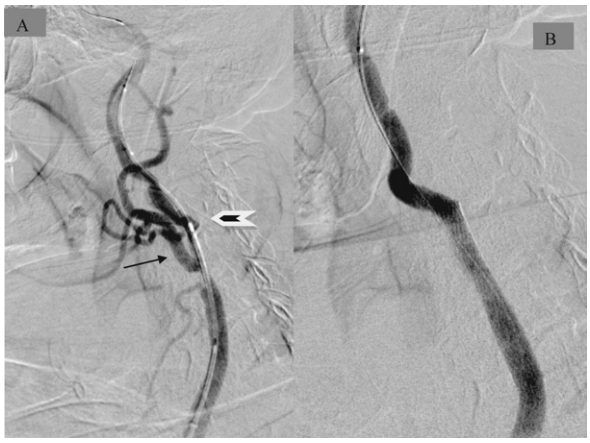

Fig 3. A, Present angiogram of a patient undergoing carotid stent for recurrent high-grade stenosis. Angiogram shows patent internal carotid artery (ICA; *arrowhead*) and patent external carotid artery (ECA; *arrow*). **B,** Completion angiogram showing patent carotid stent and occluded ECA after carotid stenting and angioplasty. This patient complained of unilateral jaw claudication for 2 months.

DISCUSSION

In recent years, CAS has emerged as a less invasive yet comparably effective alternative to CEA in the treatment of carotid artery occlusive disease.[1] However, in CEA the ECA is treated as well, but covered by the stent struts in CAS. Further studies evaluating overstenting of the carotid bifurcation, stent covering the origin of the ECA, have demonstrated a significant progression of ECA stenosis.

Willford-Ehringer et al reported significant progression of atherosclerotic disease at the orifice of the ipsilateral ECA after CAS compared with the contralateral ECA.[11] In one case, occlusion of the ECA due to embolism during the stenting procedure resulted in jaw claudication for up to 10 days. In our case, patient number two had prolonged symptoms of jaw claudication due to acute thrombosis of the ECA rather than embolism. Another explanation for the prolonged symptoms is the time needed for collaterals from the contralateral ECA to form. Others have further assessed overstenting of the bifurcation compared with no overstenting and demonstrated significant disease progression with overstenting.[4] A study by Woo and colleagues compared the CAS and CEA groups for which they performed eversion endarterectomy of the ECA during CEA compared with covering the ECA orifice during CAS. They demonstrated significantly higher ECA velocities at the 1-, 6-, and 12-month intervals following CAS compared with the CEA group.[3] The authors suggested that the stent might be a nidus for atherosclerosis or intimal hyperplasia, resulting in progressive stenosis of the ECA and increased flow velocities. Two ECA occlusions occurred in their study, although no neurological symptoms were reported. Typically, these jaw claudication symptoms are transient.

Few cases of ECA jaw claudication caused by carotid occlusive disease have been documented. There have been no reports on the effect of CAS in a patient with preexisting jaw claudication. But the symptoms might get worse unless the contralateral ECA is normal or treated.[8,12,13] In 1980, Argentino et al reported two cases of jaw claudication. In one case, intermittent jaw claudication was successfully treated with revascularization, indicating that the pain was originally due to ischemia.[9] Additional case reports since then have also documented jaw claudication secondary to severe atherosclerotic disease of ECAs that were successfully treated with revascularization of the ECA stenosis.[7-9,13]

Janssens et al reported a case of bilateral jaw claudication in an 87-year-old patient with bilateral ECA disease that resolved after a unilateral endarterectomy.[13] Schiller et al reported resolution of bilateral jaw claudication after staged bilateral ECA percutaneous transluminal angioplasty (PTA). Symptoms recurred after the first and second PTA, resulting in right carotid endarterectomy and resolution of bilateral symptoms[8] up to a 3-year follow-up. This highlights the importance of collaterals from the ipsilateral ICA and contralateral ECA.

Other causes of unilateral jaw and facial pain include[14] temporal arteritis, temporomandibular joint disease, rheumatoid arthritis, myasthenia gravis, and parotid duct obstruction.

CONCLUSION

Occlusive disease of the ECA should be in the differential diagnosis of patients presenting with jaw claudication. Also, patients with severe bilateral carotid disease need to be evaluated for jaw claudication. Even though there is no evidence to support worsening of jaw claudication after CAS, we wish to alert the interventionalist about this problem that could potentially get worse after CAS, and to consider referring these patients for evaluation for CEA, since external carotid endarterectomy performed during CEA is effective and durable in relieving jaw claudication symptoms.

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