

Facial Numbness and Dysesthesia

New Features of Carotid Artery Dissection

Kenneth R. Francis; Douglas P. Williams, MD; B. Todd Troost, MD

• Facial numbness and dysesthesia have not been emphasized as presenting features in spontaneous internal carotid artery dissection. Progressive facial pain, accompanied by oculosympathetic paresis, altered taste, and facial numbness suggest the possibility of basal skull neoplasm. We describe a patient, with previously undiscovered fibromuscular dysplasia, who presented with severe neck and face pain, dysgeusia, oculosympathetic paresis, and markedly reduced facial sensation due to a spontaneous vascular dissection. Altered facial sensation should now be included in the symptomatology of internal carotid artery dissection.

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Headache, neck and scalp pain, bruits, and oculosympathetic paresis are the cardinal presenting symptoms of internal carotid artery dissection.¹⁻⁵ The condition may be unsuspected in a relatively young patient who presents in an unusual manner. We present a patient with a previously undescribed sign—reduced facial sensation—who also had dysgeusia, oculosympathetic paresis, and severe unilateral facial pain.

REPORT OF A CASE

A 46-year-old mildly hypertensive woman developed "lightning pains" that radiated to her face from the left side of her neck. The following day the pain had become dull and was localized behind her left eye. She noted slight drooping of her left eyelid, a strange persistent metallic taste, and discomfort over the left side of her forehead. Her unilateral neck and face pain then increased in intensity, and the entire left side of her face became "numb and disagreeable." Her blood pressure was 130/80 mm Hg. Her left pupil was 2 mm and her right 3.5 mm in normal room illumination, and she had 2 mm of left-sided ptosis, with normal facial sweating (oculosympathetic paresis). She had a marked decrease in sensation to light touch and pinprick over all three divisions of the left trigeminal nerve. Testing facial

sensation evoked an unpleasant sensation. The remainder of the cranial nerves were intact, and the patient had neither subjective nor objective cervical or intracranial bruits. The hydroxyamphetamine hydrobromide (Paredrine) test was consistent with a third-order oculosympathetic defect.⁶ Results of computed tomography and magnetic resonance imaging of the head and neck were normal, but cerebral angiography demonstrated a dissection of the left internal carotid artery extending intracranially to the cavernous sinus (Figure). There was also a 2-cm dissection of the left vertebral artery. The patient initially underwent anticoagulation with heparin and thereafter for a three-month period with coumadin. Following anticoagulation, her pain disappeared within 48 hours, but she had persistent left facial numbness, facial dysesthesia, and abnormal subjective taste sensation for two weeks, after which sensation returned to normal. The oculosympathetic defect was permanent.

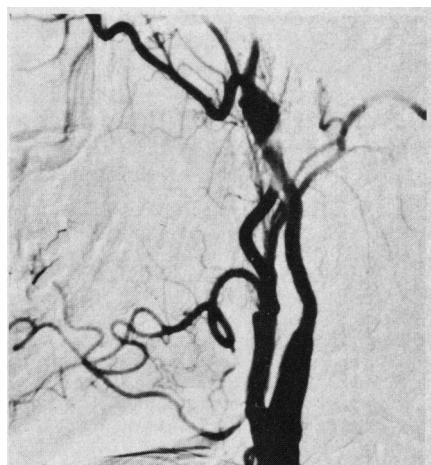
COMMENT

Symptoms and signs described with carotid artery dissection include headache, facial pain, subjective bruit, oculosympathetic paresis, and, more rarely, dysgeusia.¹⁻⁵ A most unusual facet of this patient's presentation was the consistent decrease in sensation over all three divisions of the left trigeminal nerve, accompanied by dysesthesia. Rarely, patients with carotid artery dissections have described a "rubbery feeling,"¹ but, to our knowledge, decreased facial sensation has not previously been described. It is believed that vascular dissection causes interruption of the vasa nervorum supplying the sympathetic trunk traveling with the internal carotid artery. The ischemia produces an oculosympathetic paresis, with miosis and ptosis; however, facial sweating remains intact because sympathetic fibers to the face travel with the external carotid artery not involved in the dissection. Referred facial pain is well described in internal carotid artery dissection, presumably due to referred pain in the distribution of the fifth nerve, as trigeminal fibers supply sensation to the carotid artery in the anterior and middle fossa.⁷ The sensation of perturbed taste and the dysgeusia described by the patient cleared after two weeks. Dysgeusia is a rare complaint in patients with internal caro-

tid artery dissections, and one might posit an alteration in the blood supply to the seventh cranial nerve or branches of the chorda tympani at the base of the skull. It is surprising that if the trigeminal nerve is, in fact, responsible for the perception of pain (which is the most frequent complaint in carotid artery dissection), objective involvement of its peripheral sensory distribution has not been previously reported.

Fibromuscular hyperplasia, a vascular condition associated with spontaneous vascular dissection,⁸ was evident on other angiographic views and was the most likely underlying vascular pathologic feature responsible for the combined carotid and vertebral dissection in our patient. So-called Raeder's paratrigeminal syndrome describes patients with persistent unilateral facial pain and an oculosympathetic paresis with or without associated cranial nerve deficits. When there are associated cranial nerve deficits, such as trigeminal nerve dysfunction, an underlying neoplasm must always be ruled out. The clinical presentation of a patient who has unilateral face and neck pain associated with sympathetic, seventh, and fifth nerve dysfunction should prompt a consideration of a basal skull nerve neoplasm such as nasopharyngeal carcinoma. Also, in patients in whom neuroimaging tests

Lateral view of left carotid artery showing tapered narrowing of left internal carotid artery.



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From the Department of Neurology, Bowman Gray School of Medicine, Wake Forest University, Winston-Salem, NC.

Reprint requests to Department of Neurology, Bowman Gray School of Medicine, Wake Forest University, 300 S Hawthorne Rd, Winston-Salem, NC 27103 (Dr Troost).

such as computed tomography and magnetic resonance imaging do not lead to the correct diagnosis, strong diagnostic consideration should be given to a vascular dissection, which must be demonstrated by arteriography.

We believe that facial numbness and dysesthesia represent previously unemphasized features of internal carotid artery dissection. Spontaneous vascular dissections should be considered in the differential diagnosis of patients presenting with unilateral head and neck pain, oculosympa-

thetic paresis, dysgeusia, and facial numbness.

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Loss of Deep Dyslexic Reading Ability From a Second Left-Hemispheric Lesion

David P. Roeltgen, MD

• It has been hypothesized that the residual reading ability in people with deep dyslexia (an acquired dyslexia in which the subjects make semantic paralexias, eg, child read as boy) utilizes right hemispheric structures. A patient who was deep dyslexic following an initial left hemispheric stroke was studied. Following a new left hemispheric stroke, he lost his residual reading ability. In this patient, deep dyslexic reading abilities were dependent on left hemispheric structures.

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Subjects with deep dyslexia produce semantic paralexias when reading aloud. Other characteristics, including visual errors, function word substitutions, derivational errors, demonstration of an inability to derive phonology from print and reading high imagery words better than low imagery words, nouns better than adjectives, adjectives better than verbs, and nouns better than function words, also occur as part of the syndrome.¹ Although there is agreement

on the general clinical presentation of patients with deep dyslexia, there is considerable controversy regarding potential anatomic substrates underlying their residual reading ability. We addressed this issue by assessing the residual reading ability in a patient with deep dyslexia after he had a second cerebral lesion. Consequently, we were able to draw some conclusions regarding the anatomic substrate for his reading abilities following his first stroke.

REPORT OF A CASE

A 54-year-old right-handed man had a left cerebral infarction in 1981. He had a transient right-sided paresis and mild stable aphasia. He was studied 2½ years following his stroke and except for a mild aphasia, results of an examination were normal. His spontaneous speech was mildly nonfluent with good articulation. His comprehension for conversation and commands was good, but deteriorated with increased complexity of syntax. Naming to confrontation was good. Based on history and results of the Western Aphasia Battery (WAB)² (Table 1), he could best be described as a recovered Broca's aphasic. He was considered deep dyslexic because he made semantic errors when reading aloud, especially when reading paragraphs.

When reading single words (n = 420) he made 58 errors: 3% semantic ("clothing" for "fabric"), 14% derivational ("confident" for "confidence"), 43% visual ("melt" for "man"), and 4% function word substitutions ("should" for "such"). When reading words in paragraphs (n = 387) he made 55 errors: 11% semantic, 9% deriva-

tional, 20% visual, and 35% function word substitutions. Analysis of his ability to read single words is presented in Table 2.

The patient had adequate comprehension of single words and moderately good comprehension of paragraphs, with a raw score of 65 on the paragraph comprehension subtest of the Peabody Individual Achievement Test.³ On phonological reading tasks, he had difficulty reading nonwords (14 [23%] of 60 correct) but repeated them well (20 of 20) (Battery of Linguistic Analysis of Writing and Reading)⁴, (Battery of Adult Reading Function [L. J. Rothi, H. B. Coslett, K. M. Heilman, unpublished data, 1983]). He also made errors on a visually presented rhyming task when asked to say whether two words rhymed (16 of 20 correct; chance equals ten of 20) and on a picture-pointing task for nonwords that were homophonic for real words (pseudohomophones) (Battery of Adult Reading Function; 36 of 40 correct; chance equals 13 of 40) (Table 3).

In September 1984, the patient sustained a new stroke, with a new right hemiparesis and marked worsening of his language performance. The results of the rest of the examination were unchanged. On examination, his fluency and comprehension were more impaired than previously, and his repetition was limited to approximately two word phrases. Naming ability declined such that he was able to name only rare objects with phonemic cues. Clinical evaluation and results from the WAB² (Table 1) suggested that he could be best classified as having a mixed aphasia.

His language performance was studied at two, four, and six weeks following his second stroke, and results from clinical and formal examinations were constant. He had fairly consistent scores on the WAB² (Table 1).

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From the Department of Neurology, University of Missouri-Columbia and the Harry S Truman Memorial Veterans Hospital, Columbia, Mo.

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Reprint requests to Department of Neurology, M741 Health Science Center, University of Missouri-Columbia, Columbia, MO 65212 (Dr Roeltgen).