

The Bitter Taste of Whiplash

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Like most 18-year-olds, Joshua Bohsali* wasn't interested in listening to his parents. Following his recent car accident, his mother claimed his left eye looked "sunken in" and smaller than his right eye, but he was loath to waste a summer's day at the doctor's office. After all, a CT scan of his head in the ER on the night of the accident was negative, and he felt fine. Finally, five days after the accident, and with continued prodding from his mother, he agreed to see the ophthalmologist.

We Get a Look

On examination, his vision was 20/20 bilaterally, with intraocular pressures of 18 mmHg on the right and 16 mmHg on the left. His pupils were slightly asymmetric in size (Fig. 1) but were reactive and without an afferent defect. Closer inspection showed the right pupil to be 3.5 mm and the left pupil to be 3.0 mm in normal room light, and in darkened conditions the right and left pupils increased to 5.0 mm and 4.0 mm, respectively. Both pupils reacted briskly to light. Hertel

exophthalmometry was normal, and the adnexal exam was remarkable only for a slight 1-mm left ptosis without any associated inverse ptosis. The remainder of his slit-lamp and dilated fundus exams were unremarkable.

Differential Diagnosis

We were mindful that Horner's syndrome from an internal carotid artery (ICA) dissection in a recent motor vehicle accident was a possibility, but we were largely unimpressed by his equivocal exam findings. Mr. Bohsali's

anisocoria was well within

physiologic range, and a single drop of apraclonidine instilled in both eyes produced no perceptible dilation of either pupil. His mild left ptosis was also consistent with his recent trauma, which can often result in a transient ptosis. In addition, Mr. Bohsali did not complain of the head or neck pain often seen in cervical ICA dissections. Finally, his lack of significant headache argued against cluster headaches, which can occasionally be associated with Horner's syndrome.

We counseled Mr. Bohsali and his mother that the additional workup we were contemplating would likely be unrevealing. However, to complete a thorough history, we asked Mr. Bohsali if he'd noticed any unusual tastes in his mouth since the accident. He was surprised at the question, but he surprised us even more with his response. For the past several days, he had experienced a "foul, bitter taste" in his mouth that he couldn't explain. We cautioned that the finding may be incidental, but our suspicions were heightened; we ordered a CT angiogram of the head and neck.

Imaging

Later that day, we got a call from the neuroradiologist. He said the CT angiogram was highly consistent with a left ICA dissection (Figs. 2 and 3). Mr. Bohsali was admitted to the hospital that day and received a stat neurosurgery consultation. He was given load-

What's Your Diagnosis?



WE GET A LOOK. (1) We noted a mild left anisocoria and left ptosis.



WE REVIEW THE ANGIOGRAMS. (2) We note the narrowing of our patient's left ICA near the jugular foramen. (3) The left ICA lumen (solid arrow), while lacking the classic double lumen or crescent sign of dissection, shows an approximately 50 percent stenosis relative to the right ICA (broken arrow). (4) The cervical/cerebral catheter angiogram confirms a left ICA dissection. Marked attenuation is seen at the posterior genu of the ICA as the vessel enters the skull base.

ing doses of aspirin and clopidogrel bisulfate (Plavix) and was monitored in the neurosurgical ICU. The next day, a cervical/cerebral catheter angiogram confirmed the diagnosis (Fig. 4).

Neuroanatomy

Dysgeusia, the abnormal perception of taste, can be associated with a variety of toxic, neoplastic and traumatic disorders. The special sensory innervation of taste is divided among cranial nerves VII (anterior two-thirds of tongue), IX (posterior third of tongue) and X (pharynx). Although some reports had suggested that dysgeusia in patients with ICA dissection was caused by chorda tympani (cranial nerve VII) involvement, newer studies indicate that the lesion may be along cranial nerve IX.¹ The proposed mechanism is thought to be the compression of cranial nerve IX against the expanding wall of the dissecting artery.²

The combination of dysgeusia (due to a cranial nerve lesion) and Horner's syndrome (due to a sympathetic pathway lesion) demonstrates the close anatomic relationship of vital structures in the neck and localizes the site of the lesion to the jugular foramen. Cranial nerve IX crosses the ICA medially at the level of the second cervical vertebra soon after the nerve exits the skull base.² As the third-order neuron

of Horner's sympathetic pupillary pathway also travels along the upper cervical ICA, a lesion at this junction can result in the presentation seen in our patient.^{1,2}

Our Patient

Mr. Bohsali's presentation was fascinating for several reasons. ICA dissection-induced Horner's syndrome is typically painful, yet Mr. Bohsali noted no significant head or neck pain. His presentation was also subtle enough to escape detection at his initial ER evaluation. In fact, his symptoms were so mild that he continued to defer evaluation until finally convinced by his observant mother. Furthermore, the cause of his anisocoria was obscured by a falsely negative apraclonidine drop test. Apraclonidine, commonly used as an accessible substitute for cocaine in the detection of Horner's syndrome, has often been ineffective in diagnosing acute presentations.³

Treatment

Our patient's dissection involved the junction of the petrous and cervical portions of the ICA and involved a 50 percent stenosis. After careful consideration, neurosurgery decided against surgical intervention, noting that stenting the mobile cervical carotid against the immobile petrous carotid

could result in further dissection or occlusion of the ICA. Mr. Bohsali was placed on a heparin drip and started on warfarin. After achieving a therapeutic INR (international normalized ratio), he was discharged from the hospital on long-term anticoagulation. A repeat CT angiogram one month after his hospital course showed an improvement in ICA luminal caliber, and he reports a near-total improvement in his Horner's syndrome symptoms.

Conclusion

ICA dissection is a life-threatening condition that can present to any ophthalmologist. Mr. Bohsali's case demonstrates the importance of 1) taking a thorough history and 2) having a good knowledge of the anatomy (next page).

* Patient name is fictitious.

1 Bhatti, M. T. and I. Schmalzfuss. *Acta Ophthalmol Scand* 2002;80:562-564.

2 Taillibert, S. et al. *J Neurol Neurosurg Psychiatry* 1998;64:691-692.

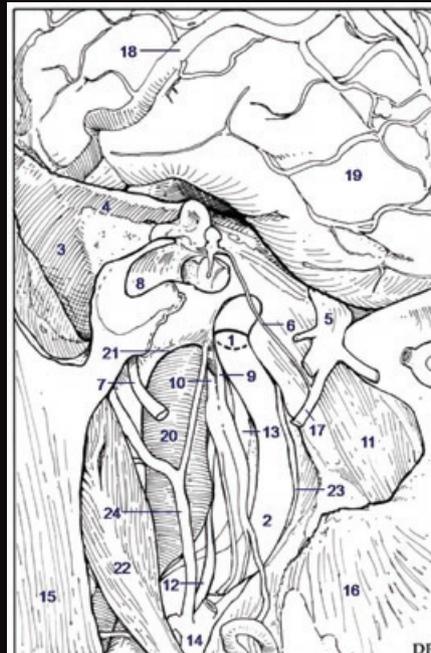
3 Dewan, M. A. et al. *Can J Ophthalmol* 2009;44:109-110.

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Refresh Your Knowledge of the Anatomy

Lateral view, at the level of the jugular foramen showing the relationship of the internal carotid artery to the lower cranial nerves. The styloid process, carotid canal and temporal bone are missing. (Courtesy of Takaya Inoue, MD, and Albert L. Rhoton Jr., MD, University of Florida)

- 1 Foramen lacerum.
- 2 Internal carotid artery.
- 3 Sigmoid sinus.
- 4 Superior petrosal sinus.
- 5 Third division of trigeminal nerve.
- 6 Chorda tympani nerve.
- 7 Facial nerve.
- 8 External auditory meatus.
- 9 IX cranial nerve.
- 10 X cranial nerve.
- 11 Tensor veli palatini muscle.
- 12 XII cranial nerve.
- 13 Sympathetic trunk.
- 14 External carotid artery.
- 15 Sternocleidomastoid muscle.
- 16 Buccinator muscle.



- 17 Lingual nerve.
- 18 Vein of Labbé.
- 19 Temporal lobe.
- 20 Jugular vein.

- 21 Jugular foramen.
- 22 Posterior belly of digastric muscle.
- 23 Ascending pharyngeal artery.
- 24 Occipital artery.

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