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For complete biographies of the NSPC team of physicians, please visit **nspc.com**.

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Neurovascular Surgery Case Studies

An Unusual Indication for Carotid Stenting Over Endarterectomy

by **Jonathan L. Brisman, M.D.**

This 77 year old man with a history of stroke in 2013 presented to a small community hospital with aphasia upon awakening. Given the unclear timing of onset of his symptoms he was not deemed a candidate for intravenous thrombolytic. His NIHSS was 2 in the emergency room. He was admitted to the hospital where a CT (**Figure 1**) and CTA were performed showing a left frontal small infarct and severe extracranial carotid stenosis on the left. MRI could not be performed because of the patient's pacemaker. Examination was significant for an awake gentleman with normal cranial nerve function and normal motor exam but significant expressive aphasia. He was placed on Aspirin.

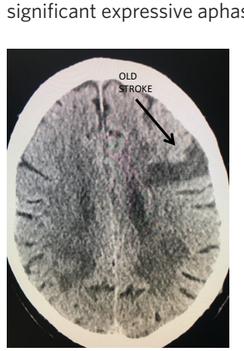

Figure 1

The patient was brought to the operating room for a left carotid endarterectomy (CEA) after cardiologic evaluation deemed him a suitable candidate. After placing the patient under general anesthesia and positioning him with slight neck extension and head turned 30 degrees to the right a significant drop in somatosensory potentials of the left side of his body was noted. As this was the side ipsilateral to the carotid stenosis, an ischemic etiology was felt unlikely. Multiple attempts at repositioning failed to improve the potentials and the procedure was therefore aborted in favor of carotid angioplasty and stenting (CAS) under local anesthesia. He was transferred to our tertiary care regional facility for this procedure and loaded with Plavix in addition to his aspirin. Assays confirmed antiplatelet inhibition of both agents.

Catheter angiography prior to angioplasty revealed critical stenosis (**Figure 2**). Angioplasty was therefore performed under local anesthesia with an embolic protection device deployed to catch any dislodged debris (**Figure 3**). After angioplasty, a stent was opened across the lesion to maintain long-term patency (**Figure 4**). Post-stent angiography showed smooth dilatation of the lesion. The patient's aphasia continued to improve at his 6 week follow-up visit and duplex revealed no significant stenosis through the stent.

Discussion:

It is now considered Class I evidence (i.e. strongly in favor of benefit vs. risk) for patients experiencing non-disabling strokes or TIAs (including amaurosis fugax) who demonstrate greater than 70% carotid stenosis by non-invasive imaging, to undergo carotid revascularization to reduce the chance of further strokes (2 year reduction of 26% recurrent stroke down to 9%).[1-3] Revascularization is recommended by experienced surgeons only and includes carotid angioplasty and stenting (Level 2 evidence) or carotid endarterectomy (Level 1 evidence). American Heart Association guidelines recommend revascularization within 2 weeks preferably and no later than 6 months after the event in order to to have the best chance of stroke reduction.[3]

The technical details of how I perform these procedures has been published previously and will not be recounted here.[3] In general I recommend CEA for standard risk patients and reserve CAS for surgically high risk patients as defined by the SAPPHIRE trial. These include patients

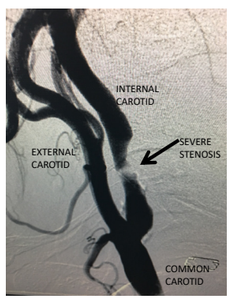

Figure 2

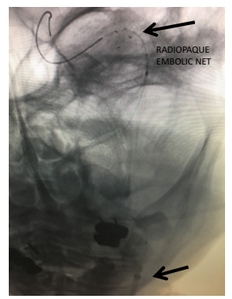

Figure 3

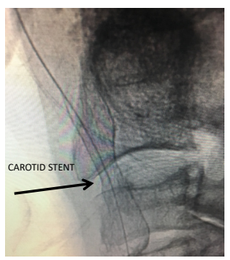

Figure 4

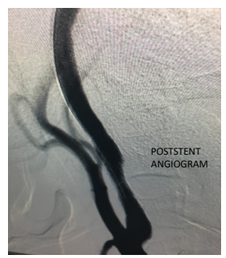

Figure 5

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with radiation stenosis, very high bifurcations, contralateral occlusion, severe pulmonary or cardiac disease or hostile surgical field as is the case with patients who have recurrent disease after prior CEA. Advanced age has been reconfigured as a surgical risk factor and generally I feel that CEA is safer in those patients older than 75 years, assuming they have no additional high risk features. Based on the recent CREST trial, the patient described here was considered a good candidate for either procedure. Given the sensitivity of the monitoring potentials to positioning for CEA, this patient was treated with CAS successfully under local anesthesia. He was premedicated with Plavix and Aspirin and will continue on the Plavix for three months post-stenting and Aspirin indefinitely.

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3. Brisman, JL and Mayberg MR: **Indications For Carotid Endarterectomy In Patients With Symptomatic Stenosis,** *in Stroke-Pathophysiology, Diagnosis and Management*, Fifth Edition, pp. 1398-1402 Elsevier Saunders, Philadelphia, PA, 2011

Central Retinal Artery Occlusion (CRAO) – Advancing Therapies for Ophthalmologic Emergencies

by **Sundeep Mangla, M.D.**

This patient is a 65-year-old male with a past medical history of gastric ulcers and smoking, who awoke normally one morning. At 8:45 a.m., while watching TV, he noticed sudden onset of blurred vision, which rapidly progressed to complete loss of vision in his RIGHT eye. Upon presentation to the Emergency Department, his initial exam confirmed acute right, monocular vision loss, with only light perception and no perception of movement or objects.

Clinical and fundoscopic examination confirmed right central retinal artery occlusion (CRAO) diagnosed at approximately 11:45 a.m. (3 hours from onset). A normal retina is offered for reference in **Figure 1A**. CRAO pre therapy is seen in **Figure 1B**.

In collaboration with our multi-disciplinary team of neurointerventionalists, neurologists, ophthalmologists, and hyperbaric medicine specialists, several therapeutic options were considered, including IV tPA (Tissue Plasminogen Activator, or tPA, is administered through an IV in the patient's arm), IA tPA (an Intra-Arterial Thrombolysis delivers the tPA directly to the area around the clot), and hyperbaric oxygen therapy (HBOT). We decided to proceed to IA tPA therapy based on this very early presentation (<4-6 hours). It was also felt that using IAt PA would have the best benefit. Additionally, a regimen of hyperbaric oxygen therapy to follow thrombolysis was proposed for this patient with severe, advanced vision loss, in the right eye.

Neuro-Interventional Therapy and Management

We performed emergent angiography of the right internal carotid artery and right ophthalmic artery, demonstrating normal origin of the ophthalmic artery with a poor choroidal blush confirming the diagnosis of retinal ischemia (**Figure 2A & 2B**). Superselective angiography of the ophthalmic artery was performed followed by intra-arterial infusion of tPA at a concentration of 0.4 mg/cc for a total dose of 2 mg over 5 minutes. The patient then received post interventional hyperbaric (HBOT) oxygen therapy (2.0 – 2.6 atmospheres, 2 hours duration) for 4 sessions within 48 hours of diagnosis. Upon serial examinations, he experienced early improvement in vision, with ability to distinguish light, movement, objects and color within right lateral fields, while central fields remained obscured. A follow-up fundoscopic examination demonstrates partial early recanalization of retinal branches (**Figure 1 C**). He was discharged home for continued outpatient therapy and management.

Summary and Discussion

Central Retinal Artery Occlusion (CRAO) represents a neuro-ophthalmologic emergency, which can lead to irreversible retinal damage secondary to ischemia of a terminal vessel (without collaterals). It is characterized by a sudden, unilateral and painless loss of vision. Embolism is the most common cause of CRAO, the major source of which is

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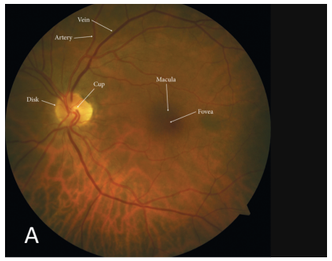

Figure 1 A: *Normal Retina*

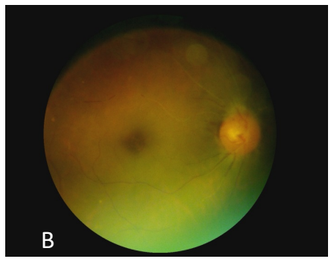

Figure 1 B: *CRAO pre therapy*

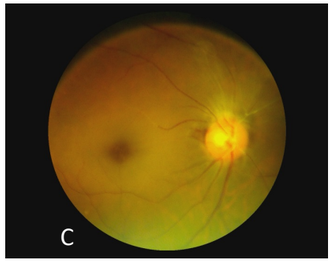

Figure 1 C: *Post IA tPA and HBOT, early reperfusion (24 hours)*

(normal reference in A, The Atlas of Emergency Medicine, 4th ed; patient fundoscopic exam, B and C, courtesy of Marlon Seliger, M.D.)

Central Retinal Artery Occlusion (CRAO)

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carotid artery disease. The natural history for spontaneous recovery or improvement with conservative measures (including paracentesis, acetazolamide, anticoagulation and/or antiplatelets) remains poor, ranging from 20-30% for some measures of functional visional improvement. In studies comparing selective thrombolysis with conservative therapy, Schmidt et al. reported 58% of the interventional therapy group compared with 29% of the control group demonstrated partial improvement in visual acuity, with 77% vs. 26% if treated within 6 hours.[1] Additional studies delivering lower doses of tPA within 4 hours of onset published by Aldrich and colleagues demonstrated significant improvement in visual acuity (at least 1 line on the Snellen chart) in 76% of patients receiving intra-arterial therapy versus 33% of patients in the control group. The Interventional group was 13 times more likely to have improvement in visual acuity of 3 lines or more and 4.9 times more likely to have a visual acuity of 20/200 or better.[2]

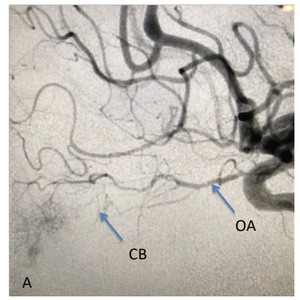

Figure 2 A: *Right ICA Angiogram, OA-Ophthalmic Artery, CB-Choroidal Blush.*

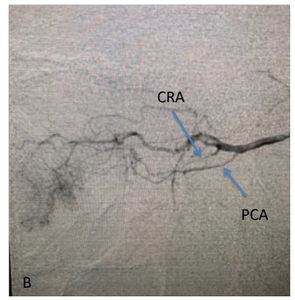

Figure 2 B: *PCA-Posterior Ciliary Artery, CRA-Central Retinal Artery – original visualized*

Hyperbaric oxygen therapy (HBOT) has been associated with visual improvement in retrospective studies.[3] HBOT can maintain oxygenation of the retina through the choroidal blood supply, decrease edema and preserve compromised tissue adjacent to ischemic area. Important key factors for improvement include early therapy (<4-12 hours), degree of vessel occlusion, type of vessel occluded, and presence of an adequate PaO2 of oxygen.[4]

Our patient experienced the most severe form of CRAO with complete vision loss with only light perception. Despite this critical presentation, with a combination of early neurointerventional therapy with intra-arterial thrombolysis directed to provide primary revascularization and Hyperbaric oxygen therapy to improve collateral and retinal perfusion, he was able to achieve early functional visual improvement of movement, objects, and color in his lateral fields. CRAO represents an ocular emergency with devastating outcomes. Poor outcomes are more commonly observed with delayed presentation, complete vision loss, and conservative management. Early recognition and potential multi-disciplinary treatment plans may offer patients an opportunity for improved functional outcomes and restoration of vision for these ophthalmologic emergencies and impending strokes of the eyes.

Acknowledgments:

Thanks to Scott Gorenstein, M.D. (Hyperbaric Medicine) and Marlon Seliger, M.D. (Stroke Neurology) for their contribution to this case discussion.

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Brain Stem Cavernous Malformation

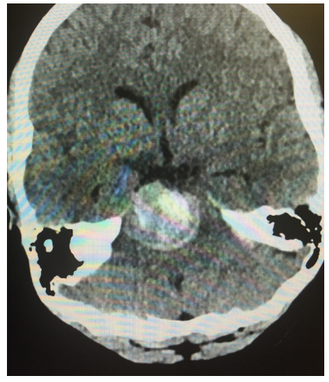
by **John A. Grant, M.D.**

A 13 year old boy fell while playing soccer and had a head ache. He was taken to an outside ER and was sent home.

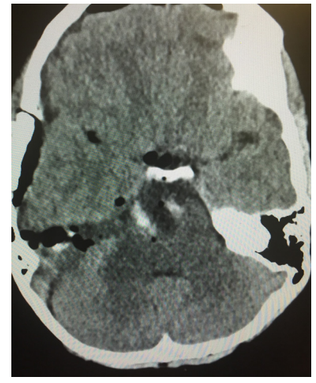
Over the next week his mother noted a facial weakness and brought him to another ER where a CT scan was done which showed a bleed in his brainstem from a Cavernous malformation unrelated to his fall.

He was transferred to our care and over the next week or so the bleed increased and he developed worsening weakness and slow heart rate. We took him to the OR and, operating under the temporal lobe to approach the brainstem from in front, removed the clot and the cavernous malformation.

Now, 2 months post operatively, he is normal.



Before Treatment



After Treatment

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