Cerebral Hyperperfusion Syndrome Following Concurrent Left Common Carotid Artery Stenting and Endarterectomy

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All authors contributed equally as co-writers of this report.

Consent
Written informed consent was obtained from the patient for submission of this manuscript for publication.

Conflict Of Interest
None

ABSTRACT

Treatment of carotid artery stenosis has been described as a risk factor for cerebral hyperperfusion syndrome. We present a case wherein an elderly female underwent concurrent stenting of a left common carotid artery origin stenosis and left carotid endarterectomy who presented with headache and left hemispheric symptoms 6 days post-procedure. Cerebral edema was seen on CT. The patient made a full recovery with no evidence of infarction.

CASE REPORT

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A 74-year-old female was referred for treatment of stenoses at the origin of the left common carotid artery and internal carotid artery. Before her procedure, she was experiencing confusion and disorientation approximately once per month for the past year that lasted a few minutes each time. The patient reported isolated incidences of bilateral blurry vision, memory loss, and loss of balance. The patient complained of discomfort during eye movement with spotty visual fields that was alleviated with sleep and/or lying down. Upon physical exam she had a 2+ right carotid pulse with a bruit and the left carotid pulse was non-palpable. The patient had 70% stenosis of the left internal carotid artery and 90% of the left common carotid artery at its origin due to mixed calcified and noncalcified atherosclerotic plaque diagnosed by CT angiogram and arteriography as well as carotid duplex ultrasound. (Figure 1) Of note, the heavy calcification of the common carotid and internal carotid arteries prevented the distinguishing of contrast from calcified plaque. The lumen would of course be seen better during catheter carotid arteriography during the intervention.

The patient underwent a left sided carotid endarterectomy, using Bovine pericardial patch with shunting of blood done during the patch surgical angioplasty. This was done in conjunction with stenting of the origin of the common carotid artery on the left (Figure 2)

The patient underwent left common carotid stenting and carotid endarterectomy in the following sequence to minimize the risk of cerebral microembolization: Surgical cut-down / dissection of the common carotid, internal carotid, and external carotid arteries; Vessel loop control, proximal and distal of the internal and external carotid arteries; catheter Arch aortography via femoral access; Retrograde micropuncture and 8-french sheath placement of the surgically exposed common carotid artery; Stenting of the left common carotid artery origin using arch aortography guidance; Use of Pott's scissors to cut through the plaque in the internal and common carotid arteries; placement of a Pruitt-Inihara shunt; endarterectomy using plaque elevators; bovine pericardial patch angioplasty of the internal and common carotid arteries.

Immediately post intervention an angiogram depicted widely patent left common external and internal carotid arteries. (Figure 3) Ultrasound showed increased blood flow through the internal and external carotid artery. The patient was in stable condition and was neurologically intact. The patient had a protracted hospital course due to 2 episodes of shortness of breath. This was treated with medical management including a pulmonology consult and a 1 unit of packed red blood cell transfusion for a Hgb of 7.7. The patient also had a GI consult due to transient heme positive stool which subsequently became heme negative on the next check. The patient improved significantly and was

discharged five days post procedure on clopidogrel 75 mg and aspirin 81 mg daily.

Management

She presented to the emergency room the next morning, with confusion, right-sided hemiparesis, headache, and aphasia. She also experienced right sided seizures. She was normotensive. Compared to the only available baseline head CT from 1.5 years ago (Figure 4), emergency room CT identified edema in the left hemisphere (Figure 5). She was placed on IV levetiracetam and lorazepam. Exam was suspicious for a left middle cerebral artery stroke. differential diagnosis was middle cerebral artery stroke and hyperperfusion syndrome.

The patient was transferred from the local hospital ER to the original hospital in a nearby city where the original procedure was performed. Upon admission she underwent repeat noncontrast head CT and CTA of the head and neck. CT of the head revealed new 5mm diameter white matter microbleed in the right parietal lobe that was stable on repeat head CT 2 days later. CTA revealed patency of the left common carotid stent and left carotid endarterectomy site with no intracranial arterial abnormalities. It was elected not to perform an MRI due to the recently placed metallic carotid stent. She was started on a stroke program with speech, physical, and occupational therapy per hospital protocol. EEG showed some focal abnormalities in the right hemisphere localized to the parietal region. The patient was alert and oriented, and she stated that the symptoms which brought her to the ER resolved. She had no overnight seizures and her exam improved significantly. She was more lucid and able to converse without difficulty.

Comprehension was also significantly improved. Her only deficits were some occasional word-finding difficulty and slight inaccuracy with coordination (right hand clumsiness), but there was marked improvement. Modified Rankin Score was 2.

Sustained improvement was seen on the following day. She felt well and there were no new complaints. On the third day after readmission, repeat CT showed no new bleed or change.

She was cleared as medically stable and discharged on aspirin 81 mg daily and levetiracetam 1000 mg twice daily with a scheduled follow up. Clopidogrel was discontinued during her inpatient course. No new complications arose.

Follow-up

Patient was followed for many years, and no new complications arose. No changes were seen on MRI imaging in 2010 (18 months pre-procedure) vs 2015. Specifically, attention to Broca's area (in the frontal lobe) and Wernicke's area (in the superior temporal gyrus) showed no evidence of ischemia or infarction. (Figures 6,7)

DISCUSSION

This patient presented with Cerebral Hyperperfusion Syndrome as evidenced by her symptoms (aphasia and headache), prior history of combined left-carotid endarterectomy and stenting of the left common carotid artery origin, CT-findings of cerebral edema, and resolution of her symptoms within hours after presentation, as well as no evidence of infarction in either Broca's or Wernicke's areas for speech on MRIs prior to or following the event. Differential diagnosis includes complicated migraine (unlikely to present with cerebral edema or for deficits to sustain longer than 24 hours), or cerebral venous thrombosis (unlikely to resolve within hours of presentation). Additionally, CHS may manifest as seizure induced Todd's Paralysis mimicking a postoperative stroke. [1] The previous case highlighted a potential complication of treating carotid artery stenosis, cerebral hyperperfusion. This consequence should be monitored after vascular procedures, especially in the elderly due to the loss of autoregulation that occurs with age. Either one or the combination of the two procedures, endarterectomy and stenting may have caused the hyperperfusion syndrome [2].

While relatively rare, CHS can be further complicated by infarct extension or intracerebral hemorrhage (0.5-2%); ICH carries a mortality rate of 36-60% and morbidity of 80% [3]. As such, it should be considered prudent to take measures to anticipate and prevent CHS when managing patients undergoing CEA and/or carotid stenting.

While there is only modest evidence regarding predictors, screening, and effective management, there have been some studies attempting to analyze cases in an effort to form a standard of care. Predictors could be used to initiate prophylactic management in high-risk patients. Some suggested predictors include female sex, older age, history of chronic kidney disease, and treatment of the left carotid artery [4], as well as hypertension, high grade stenosis, decreased cerebral vascular resistance, increased peak flow velocity, contralateral carotid occlusion, contralateral CEA in the past 3 months, and intraoperative ischemia [5].

Certain screening tools have also been suggested to identify high-risk patients for aggressive management. These include transcranial doppler, and cerebral vasoreactivity using single-photon emission computed tomography [5]. Intra-procedural transcranial doppler during common carotid stenting and endarterectomy can be helpful to monitor for microemboli.

There is not currently a standard for management, and multiple treatment options have been suggested with the aim of preventing CHS. Strict blood pressure control may be protective [2]. Free radical scavengers such as edaravone were shown to decrease incidence of CHS in one small case series, likely due to prevention of vascular endothelial injury [5]. A recent strategy to prevent CHS is staged angioplasty. This involves percutaneous

transluminal angioplasty alone of the carotid stenosis followed by stenting in a subsequent procedure 2 weeks later. Multiple case series, as well as a meta-analysis seem to support this technique [6-8]. It may be reasonable to extrapolate and propose a similar staged intervention when there are multiple or tandem sites of stenosis as in our case. Specifically, performing carotid stenting (of the left common carotid artery stenosis) first to improve the inflow, followed by carotid endarterectomy (of the left internal carotid artery stenosis), in a staged fashion.

While the literature is yet wanting, it is clear that there is patient benefit to be found in standardizing the approach to identification and management of patients planning to undergo CEA while at high-risk for CHS. In this patient who is female and 74 years old, with left-sided tandem carotid stenoses (left common carotid origin and internal carotid artery origin) it may have been prudent in retrospect to have performed staging of her left-common carotid artery origin stenting followed two weeks later by left endarterectomy. It has been demonstrated that a staged procedure by stepwise increasing cerebral blood flow may be superior to an abrupt increase in blood flow.

TEACHING POINT

The likelihood of cerebral hyperperfusion may be decreased through the use of staged intervention.

QUESTIONS

1. Which of the following factors is a suggested predictor of cerebral hyperperfusion syndrome?
   a. Female sex (applies)
   b. Younger age
   c. Previous carotid stenosis
   d. Increased cerebral vascular resistance
   e. None of the above

Explanation: Female sex is a suggested predictor of cerebral hyperperfusion syndrome. [Some suggested predictors include female sex, older age, history of chronic kidney disease, and treatment of the left carotid artery , as well as hypertension, high grade stenosis, decreased cerebral vascular resistance, increased peak flow velocity, contralateral carotid occlusion, contralateral CEA in the past 3 months, and intraoperative ischemia.]

2. Which of the following screening tools could be used preoperatively in identifying patients at risk for cerebral hyperperfusion syndrome?
   a. Magnetic resonance imaging
   b. Transcranial doppler (applies)
   c. Computed tomography scan
   d. Echocardiogram
   e. None of the above

Explanation: Transcranial doppler has been suggested as a modality for screening patients at high risk for cerebral hyperperfusion syndrome. [Certain screening tools have also been suggested to identify high-risk patients for aggressive management. These include transcranial doppler, and cerebral vasoreactivity using single-photon emission computed tomography.]

3. Which of the following strategies is described as a recent approach to prevent CHS?
   a. Percutaneous transluminal angioplasty
   b. Immediate carotid endarterectomy
   c. Staged angioplasty and stenting (Applies)
   d. Autoregulation restoration therapy
   e. None of the above

Explanation: Staged angioplasty is a recently suggested strategy in preventing cerebral hyperperfusion syndrome. [A recent strategy to prevent CHS is staged angioplasty.]

4. What is the mortality rate associated with intracerebral hemorrhage as a complication of cerebral hyperperfusion syndrome?
   a. 5-12%
   b. 20-36%
   c. 36-60% (applies)
   d. 73-85%
   e. None of the above

Explanation: Intracerebral hemorrhage carries a 36-60% mortality rate. (While relatively rare, CHS can be further complicated by infarct extension or intracerebral hemorrhage (0.5-2%); ICH carries a mortality rate of 36-60% and morbidity of 80%.)

5. What is the benefit of monitoring predictors of cerebral hyperperfusion in patients?
   a. To prevent carotid artery stenosis
   b. To initiate prophylactic management (applies)
   c. To assess autoregulation restoration
   d. To evaluate the efficacy of carotid stenting
   e. None of the above

Explanation: The primary reason for monitoring patients at risk for cerebral hyperperfusion syndrome is in order to initiate prophylactic management. [Predictors could be used to initiate prophylactic management in high-risk patients.]

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FIGURES

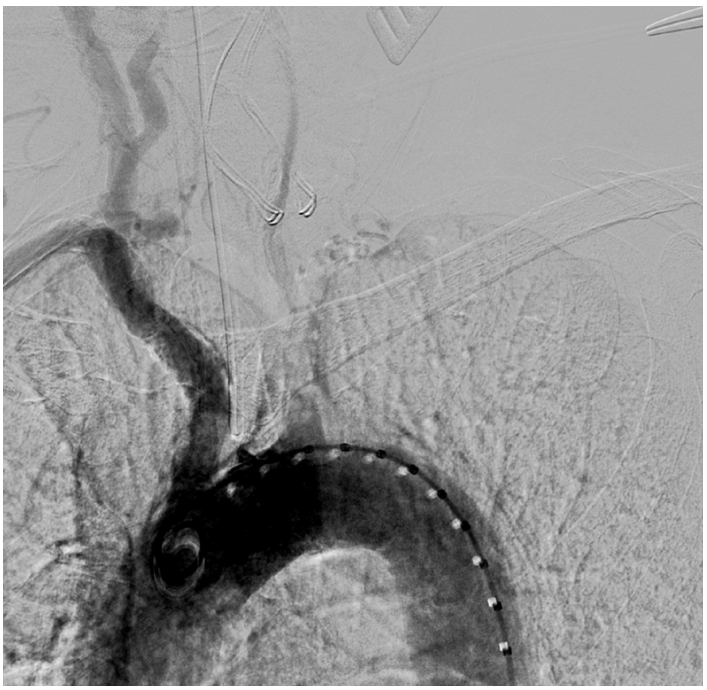

Figure 1: Arch aortogram reveals a critical 90% stenosis of the origin of the left common carotid artery with a difficult to appreciate 70% stenosis of the left internal carotid artery due to poor contrast filling. Incidental note is made of a critical stenosis of the left subclavian artery.

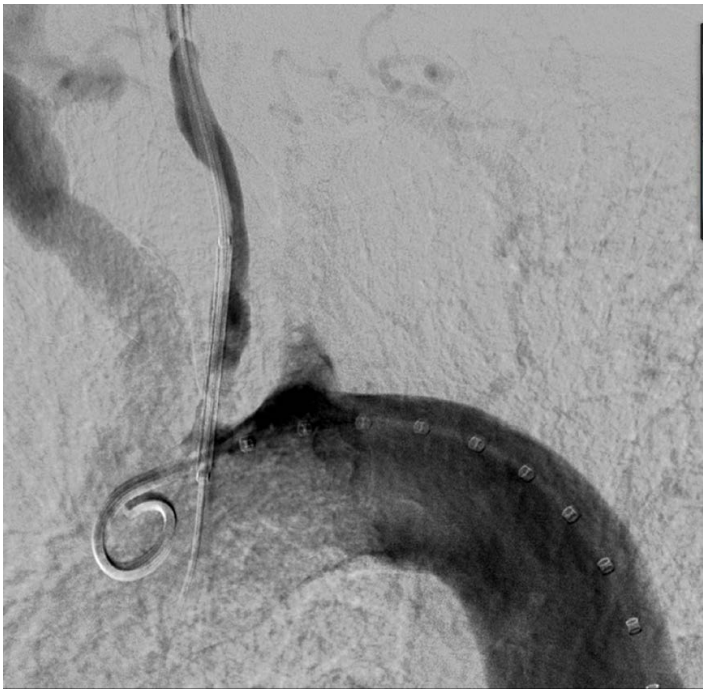

Figure 2: Arch aortogram reveals a critical 90% stenosis of the origin of the left common carotid artery pre-stent deployment (stent is visualized).

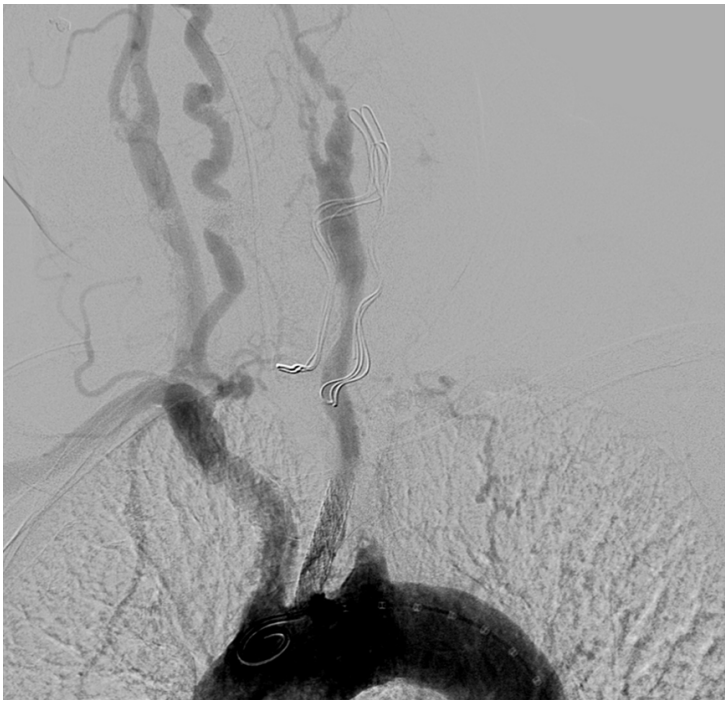

Figure 3: Arch aortogram reveals widely patent left common carotid artery origin post-stenting and left internal carotid artery post-endarterectomy.

Figure 4: Baseline head CT is negative.

Figure 5: Emergency room head CT without contrast shows edema in the left hemisphere.

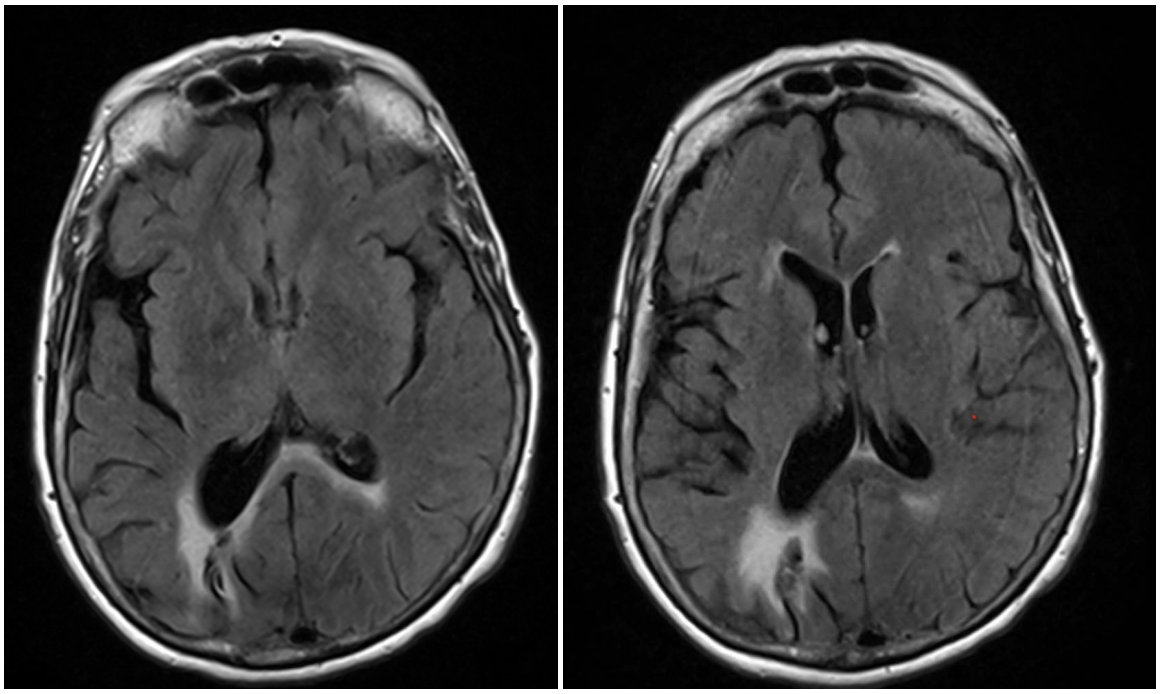

Figure 6 and 7: MRI in 2015 shows no evidence of infarction in either Broca's area or Wernicke's area

KEYWORDS

Cerebral Hyperperfusion Syndrome; Carotid Artery Stenting; Endarterectomy; Aphasia; Headache