CAS in a High-Risk Patient

Complex clinical and technical decision making for carotid artery stenting.

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"Luck is what happens when preparation meets opportunity."

—Seneca

Carotid artery stenting (CAS) represents a technically challenging procedure and, in many health care units, has traditionally been offered to patients who are considered to have an increased risk for carotid endarterectomy. Many of these patients have medical comorbidities that do not necessarily affect outcomes with CAS[1] but do require careful periprocedural management.

This article describes a case that represents an unacceptably high risk for carotid endarterectomy on the grounds of hostile anatomy and reflects on a number of factors that also rendered the case high risk for CAS.

A 51-year-old woman with a history of nasopharyngeal carcinoma requiring previous head and neck radiotherapy presented within 1 week of a left hemispheric transient ischemic attack. The patient was referred from a peripheral hospital and had undergone duplex ultrasound imaging of the carotid arteries and contrast-enhanced computed tomography (CT) scanning of the carotid circulation from the arch origins of the great vessels to the Circle of Willis.

Ultrasound revealed high-grade (> 90%), long-segment, bilateral internal carotid artery stenoses with hypoechoic plaque and obvious ulceration. CT confirmed low-attenuation material with dense axial packing of both carotid bulbs (Figures 1 through 4). Furthermore, there was a sizeable ulcer crater at the distal left common carotid artery (CCA), just at the site where one might ordinarily wish to place the CCA occlusion balloon of a proximal embolic protection device (EPD) (Figure 1).

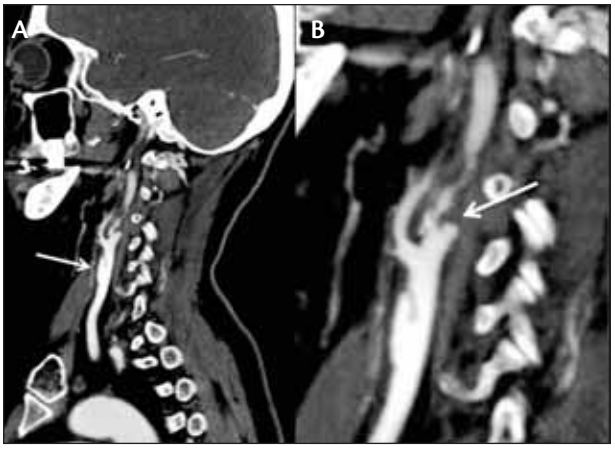

Figure 1. CT angiogram showing the ulcerated left CCA (A) and left internal carotid artery (B).

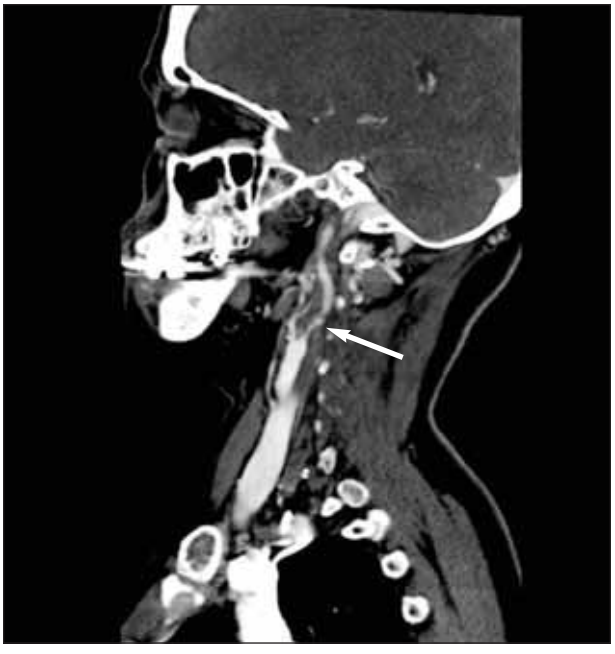

Figure 2. CT angiogram showing right internal carotid artery stenosis (arrow).

CLINICAL DECISIONS

This patient was very recently symptomatic. It is understood that the "numbers needed to treat" to prevent one stroke at 5 years in recently symptomatic patients are significantly lower than in patients with temporally remote

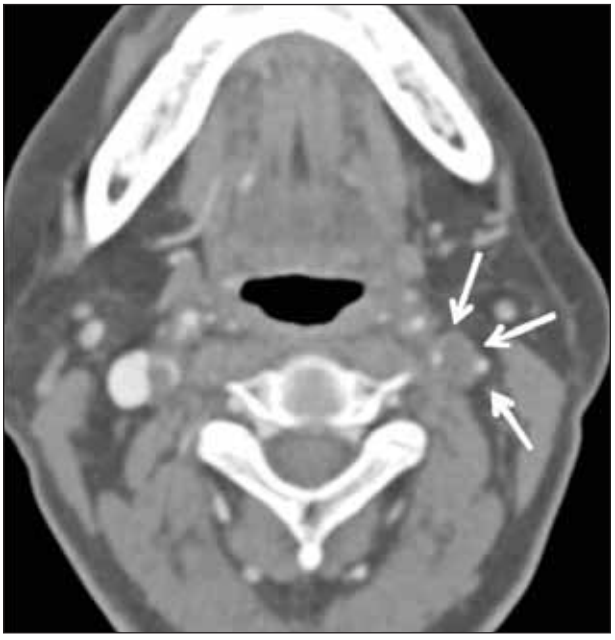

Figure 3. Friable plaque (left bulb) (arrows). Axial packing of the bulb with low-density material.

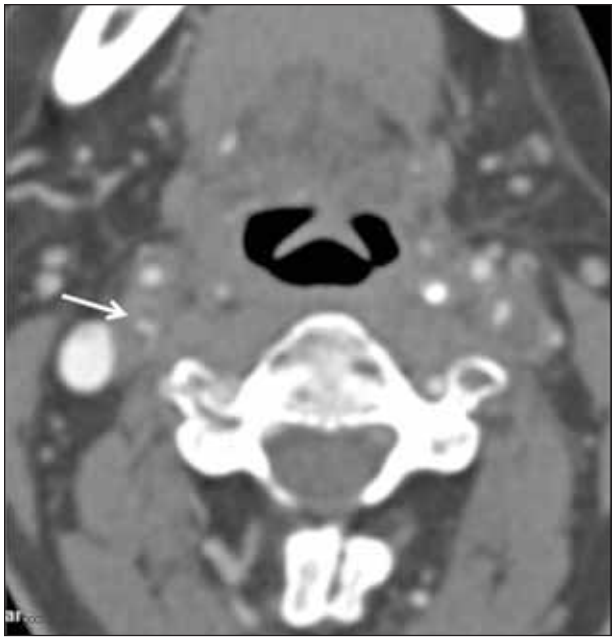

Figure 4. Friable plaque (right bulb) (arrow).

symptoms. These patients have much to gain from early carotid intervention, and although their procedural hazard is likely to be greater, they enjoy a substantially greater net benefit from timely intervention based on pooled data from NASCET and ECST.[2] Although there is no compelling evidence to suggest an unacceptable procedural risk from early intervention by CAS, there are no good datasets to suggest that it is feasible, and many physicians remain unconvinced of the safety of an endovascular option so early in the setting of friable or potentially unstable plaque.

Tolerance of Proximal EPD

This patient's baseline systolic blood pressure was 120 mm Hg on a single antihypertensive agent (angiotensin-converting enzyme [ACE] inhibitor). The patient had bilateral tight lesions, which were considered to be hemodynamically significant (the literature suggests that a carotid stenosis of > 75% is likely to have hemodynamic relevance for the brain). Furthermore, the patient's right vertebral artery was dominant, and it was stenosed at the ostium; the left vertebral was poor (Figure 5). Patients with this pattern of disease can prove challenging to treat with proximal EPD because they may be at increased risk of intolerance to flow reversal/flow arrest.

Dual-Antiplatelet Therapy

There is level 1 evidence from the carotid territory (two randomized trials) supporting the dual-antiplatelet regimen in the periprocedural setting for CAS (aspirin and

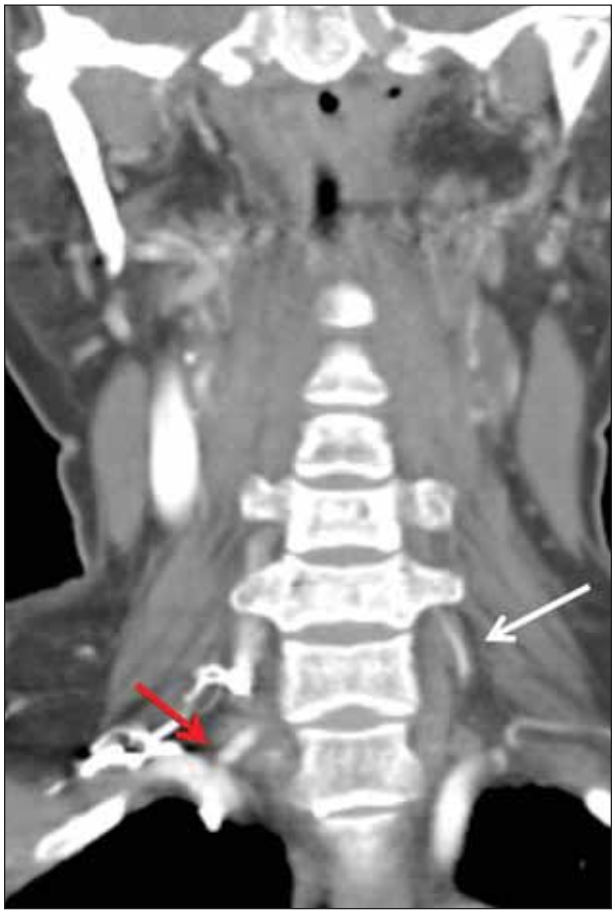

Figure 5. Dominant right vertebral artery, ostial stenosis (red arrow); left vertebral artery (white arrow).

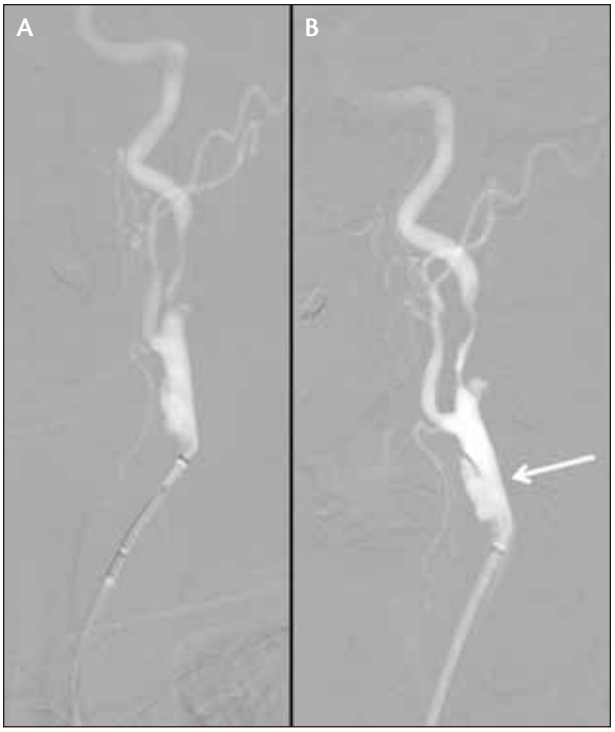

Figure 6. Left internal carotid artery (A). Ulcerated plaque in left internal carotid artery (arrow) (B).

clopidogrel with appropriate preloading of clopidogrel).[3,4] The general understanding is that this regimen should continue for at least 1 month after placement of the stent, after which time, one might expect reasonable endothelialization of a bare-metal stent, although the evidence base for this stipulation is lacking. These recommendations are generally based on de novo atherosclerotic disease; however, this patient's disease was thought to be radiotherapy induced. The likely pathogenesis of her disease process is a combination of radiotherapy-induced periarterial and per arterial inflammatory changes plus accelerated atherosclerosis. Should the dual-antiplatelet regimen be extended in this case? Can we expect the time frame of healing in these lesions to be different from lesions resulting from "standard" atherosclerosis?

Postprocedural Monitoring

It is our unit's policy to monitor patients in a high-dependency unit after CAS to ensure judicious control of blood pressure and thus avoid sustained severe hypotension (< 90 mm Hg systolic for > 24 hours), particularly in patients awaiting cardiac surgery with sustained hypertension (> 20 mm Hg above baseline for > 1 hour, especially if associated with symptoms). Sustained hypotension is significantly associated with increased major adverse clinical events and stroke,[5] while uncontrolled postprocedural hypertension is associated with a risk of hemorrhagic stroke.

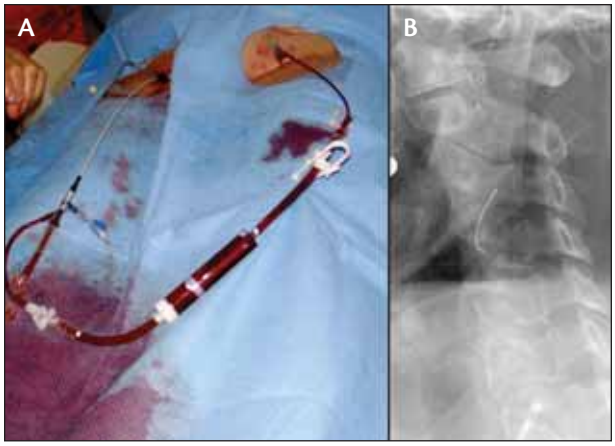

Figure 7. Dual protection using flow reversal and a distal filter (Gore Flow Reversal system [Gore & Associates, Flagstaff, AZ]). No (single) protection device is absolute (A). Emboshield Nav6 bare-wire system (Abbott Vascular, Santa Clara, CA) (B).

TECHNICAL CONSIDERATIONS

The lesions in this case presented a particular challenge for an endovascular approach. They were > 1.5 cm bilaterally, both were ulcerated, and both had features consistent with soft plaque on ultrasound and CT. The Siena risk score for CAS clearly identifies lesion length ≥ 1.5 cm, native lesions (rather than recurrent lesions), and ulceration as important risk factors for CAS and further identified symptom status and the need for predilatation (thought necessary in this case) as additional factors that are associated with a high-risk procedure.[6] In accordance with this scoring system, a composite score of > 15 indicates high risk. This patient's score was 22.

Another important consideration in such a case is the choice of EPD. Proximal protection systems would generally be preferred in a recently symptomatic patient, given the appearance of the lesions on CT and ultrasound, because of the level of security they provide compared to distal protection devices such as filters. Increasingly, the evidence base suggests good control not only of the macroembolic burden but of the microembolic burden of CAS with proximal systems, whereas filters are known to be more permissive, allowing a controlled embolization.[7-13] Furthermore, with proximal protection, the lesion is crossed after protection has been established.

There are two excellent proximal EPDs to choose from: the Mo.Ma device (Medtronic Invatec, Frauenfeld, Switzerland) and the Gore Flow Reversal system. Although both are proximal protection systems, there are some features that would lead interventionists to use one system

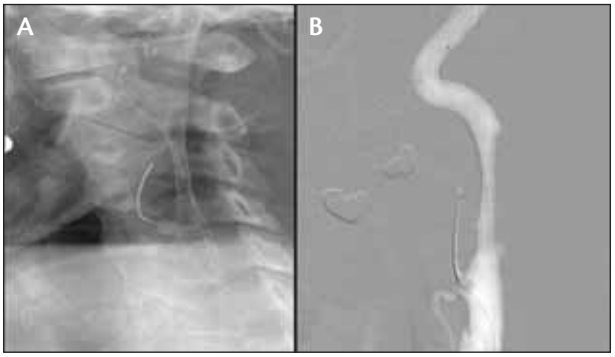

Figure 8. After stenting, no postdilatation was performed. Native image after stent placement (A); angiographic appearances after stent placement (B).

over another in certain circumstances. This patient had gross ulceration of the distal left common carotid artery (Figure 6). Both the Gore Flow Reversal system and the Mo.Ma device utilize balloons inflated in the CCA and the external carotid artery (ECA) to affect flow arrest or flow reversal. However, the CCA occlusion balloon on the Mo.Ma is at a fixed position relative to the occlusion balloon in the ECA, and if this system had been used, the CCA balloon may have been positioned against the large CCA ulcer. In contrast, the Gore Flow Reversal system allows the position of the occlusion balloon in the CCA to be tailored.

In a case that is considered to be high risk on the basis of clinical and lesion parameters, is a single EPD sufficient? Some would argue that because no single protection device is absolute, dual EPD should be employed. Ultimately, the choice was made to use the Gore Flow Reversal system and the Emboshield Nav6 bare-wire (premounted) filter (Figure 7).

The Need for Predilatation and Postdilatation

Each individual interaction with a lesion of this nature is undoubtedly an emboligenic stage, and minimal lesion interaction would make intuitive sense. Predilatation was thought to be important to provide subsequent atraumatic passage of the stent, and a 3-mm X 3-cm ultra-soft SV rapid-exchange 0.014-inch-compatible balloon (Boston Scientific Corporation, Natick, MA) was used for this stage. Postdilatation, one of the riskiest stages of the procedure, was avoided (Figure 8). The patient returned 6 weeks later to have the asymptomatic right carotid lesion stented under proximal EPD. The left-sided stent was noted to have expanded substantially, which was our expectation given that it was a nitinol stent (Figure 9).

Stent Design

There is reasonable evidence that closed-cell stents are associated with better outcomes in symptomatic patients, but the impact of stent design on clinical outcome is less convincing for asymptomatic patients.[14,15] In this case, a 6- to 8-mm X 4-cm closed-cell Xact stent (Abbott Vascular) was deployed because it was believed to provide suitable scaffolding. We considered that it would be appropriate not to stent the ulcer in the left CCA and placed the stent such that its trailing end was positioned just above the CCA ulcer.

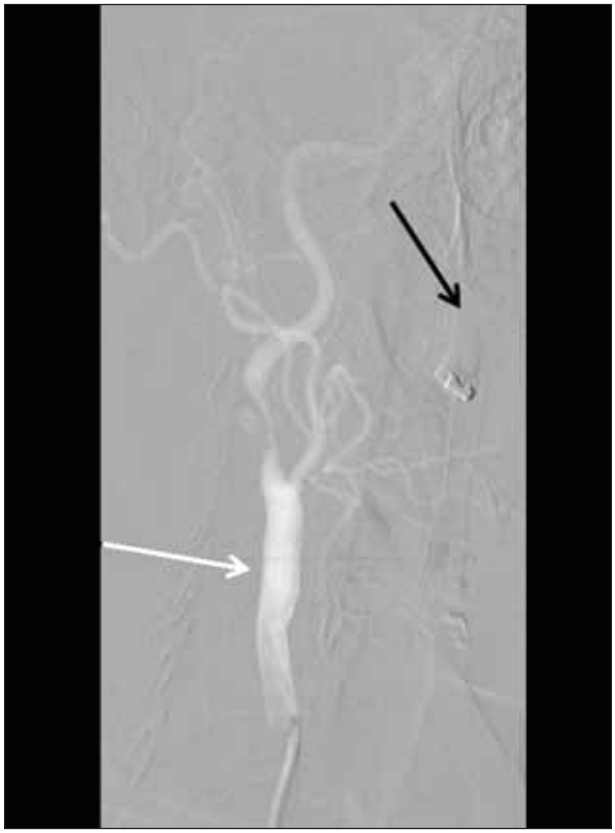

Figure 9. Selective right carotid angiogram obtained when the patient returned for right CAS 6 weeks later (selective angiography of the right carotid artery; white arrow); left-sided stent (black arrow).

Managing Intolerance of Endovascular Clamping

Intolerance tends to occur at two very distinct procedural phases. First, this can occur very early on when the CCA is clamped (after clamping of the ECA), the pressure trace "flatlines," and the stump pressure falls to $\leq$ 40 mm Hg. Aspiration of the standing column, if the Mo.Ma is used, followed by deflation of the CCA or immediate deflation of the CCA balloon with the Gore Flow Reversal system reverses any clinical signs of intolerance (yawning, agitation, seizure, obtundation). Allowing an interval before reinflation of the CCA balloon, during which time the brain is allowed to

"breathe," often results in tolerance through some poorly understood "conditioning" mechanism.

The second phase during which intolerance can occur is when the stent has been deployed. Upon flow reversal or flow arrest, autoregulation results in a compensatory systolic hypertension, which maintains tolerance. Once the stent has been deployed and exerts its influence on the baroreceptors, the autoregulatory mechanisms are overcome, the systolic blood pressure falls, and the patient becomes intolerant. However, at this point in time, the procedure is almost complete, and it makes sense to finish the intervention in a controlled yet timely fashion. Alternative strategies of dealing with intolerance include intermittent clamping at emboligenic stages or the placement of a filter under flow reversal/flow arrest with subsequent release of the CCA balloon, which allows completion of the procedure under antegrade filtered flow.

Generally, any symptomatic intolerance, no matter how unpleasant to witness, is transient and much better tolerated by the brain than an embolic shower. In our practice, patients are not permitted to eat from midnight the night before until we perform CAS with proximal EPD, not because we have any intention of administering general anesthesia, but because we wish to avoid any undue complication of aspiration of stomach contents should seizure occur (although this is acknowledged to be a relatively rare event). Because this patient's baseline blood pressure was low, we withheld her ACE inhibitor on the morning of the procedure. In a personal communication, Dr. Juan Parodi stated that a baseline of 160 mm Hg systolic will, in the majority of cases, support tolerance of endovascular clamping. Despite withholding the ACE inhibitor, the patient's blood pressure at the beginning of the procedure was 130 mm Hg, and she was clearly intolerant as a result of her pattern of disease and hemodynamics. We completed the procedure in a systematic stepwise manner, and there were no clinical sequelae.

Discontinuation of Protection

We decided to advance the retrieval system of the Nav6 through the stent on flow reversal before unclamping the ECA and CCA balloons, allowing the stent to be "washed through" the filter for a short time before filter retrieval. Somewhat remarkably, there was no visible debris in either the Nav6 or the Gore external filter (although thorough examination of the latter is somewhat hampered by its configuration and volume).

CONCLUSION

In our unit, we try to ensure that CAS procedures are performed by two experienced operators. This patient was treated by Dr. Ralph Jackson and myself at the Freeman Hospital in Newcastle upon Tyne, United Kingdom. At the end of the day, although this policy is protective for the patient (and the operator), an optimal result is dependent on treatment in a unit that is used to dealing with complex cases such as this in reasonable volume.[16,17] ■

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