

Carotid Imaging: Ensuring Optimal Imaging Before, During, and After Revascularization

A review of the contemporary imaging algorithm for carotid stenosis and its role in guiding revascularization strategies.

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Carotid imaging is essential before revascularization to inform procedural planning and strategy, guide intraoperative decision-making, facilitate periprocedural management and postprocedural surveillance, and assess treatment efficacy. In patients with incidentally diagnosed asymptomatic carotid stenosis, serial Doppler ultrasonography may be appropriate, with CTA or contrast-enhanced MRA obtained when stenosis exceeds 70% to further characterize the lesion and inform revascularization planning. In contrast, patients with symptomatic carotid stenosis require emergent, comprehensive noninvasive vascular imaging at presentation, including Doppler ultrasonography and CTA or MRA, regardless of whether emergent endovascular therapy is anticipated, to define disease extent and severity and guide management. Brain MRI, when available, is appropriate to define embolic stroke burden in symptomatic disease. In severe stenosis approaching $\geq 90\%$, CT perfusion (CTP) imaging may identify isolated hemispheres resulting from an incompetent circle of Willis, raising concern for hyperperfusion hemorrhage and the need for higher-level postprocedural care and aggressive blood pressure (BP) management. Importantly, early randomized trials of carotid revascularization in symptomatic carotid stenosis, as well as trials including both asymptomatic and symptomatic carotid stenosis, were conducted when CTA was increasingly used to characterize symptomatic lesions, but perfusion-based imaging metrics were not incorporated into patient selection or treatment decisions.¹⁻⁶ With technologic advances and an evolving understanding of carotid anatomic complexity, this article reevaluates the contemporary imaging algorithm for carotid stenosis and its role in guiding revascularization strategies.

PREPROCEDURAL IMAGING CONSIDERATIONS

Asymptomatic carotid stenosis is typically identified incidentally by auscultation of a neck bruit, Doppler ultrasonography performed for unrelated indications (including preoperative assessment for coronary artery bypass grafting or evaluation of syncope or incidental CTA of the neck for other indications), or during surveillance after prior treatment of contralateral carotid stenosis. The elective nature of revascularization permits greater flexibility in the timing and extent of preprocedural imaging. Prior trials of revascularization for asymptomatic carotid stenosis, including ACST-1, ACST-2, and SPACE-2, relied predominantly on Doppler ultrasonography thresholds for enrollment, whereas later trials, including CREST and CREST-2, incorporated multimodality imaging with CTA, MRA, or digital subtraction angiography (DSA) alongside Doppler criteria. In contrast, SAPHIRE relied on Doppler or DSA assessment for trial eligibility. At our center, patients with asymptomatic carotid stenosis undergo serial Doppler ultrasonography. When stenosis is $> 70\%$, CTA or MRA of the head and neck is obtained to characterize the extracranial and intracranial carotid circulation and define disease extent. Neck MRI may additionally be obtained to characterize plaque morphology. In patients with near-total carotid occlusion on Doppler ultrasonography, CTP is obtained with CTA to characterize baseline cerebral perfusion.

Patients with symptomatic carotid stenosis require emergent imaging, and at our center, the endovascular neurosurgery service is engaged at presentation. Patients with suspected acute stroke undergo immediate noncontrast head CT, thin-slice CTA of the head and neck, and CTP using a second contrast bolus. Imaging is urgently

reviewed for large (LVO) or medium vessel occlusion (MeVO), cerebral ischemia, and cervical carotid disease to determine candidacy for emergent mechanical thrombectomy or carotid artery stenting (CAS). Although most patients do not require emergent intervention, this imaging establishes the anatomic framework for subsequent management. Carotid duplex ultrasonography is subsequently correlated with CTA, and preintervention MRI with diffusion-weighted imaging (DWI) and FLAIR (fluid-attenuated inversion recovery) is obtained when appropriate to confirm the diagnosis and define infarct burden.

Comprehensive noninvasive imaging for asymptomatic and symptomatic carotid stenosis provides a procedural roadmap for carotid endarterectomy (CEA), CAS, and transcarotid artery revascularization (TCAR). Cervical CTA defines aortic arch and proximal great-vessel anatomy, plaque burden and calcification, carotid bifurcation and lesion morphology, and distal cervical internal carotid anatomy, thereby characterizing extracranial carotid disease. A hostile arch, defined by severe atherosclerosis, “shaggy” morphology, or type III tortuosity, precludes transfemoral (TF) CAS. We next assess lesion morphology: Severe bifurcation angulation that prevents safe stent delivery or deployment favors CEA, whereas in its absence, TCAR is considered. In the absence of a hostile arch or severe lesion angulation, lesions may be favorable for TF-CAS. Historically, severe, concentric, heavily calcified lesions were considered a relative contraindication to stenting because of concerns regarding inadequate stent expansion and increased procedural risk. However, as we and others have reported, intravascular lithotripsy has expanded the range of lesions amenable to endovascular treatment, allowing heavily calcified lesions to be treated with stenting through either TCAR or TF-CAS.⁷

Patient-specific clinical factors further inform revascularization strategy. The ability to tolerate dual antiplatelet therapy (DAPT) is an important consideration for CAS and TCAR; a history of gastrointestinal bleeding, coagulopathy, or other contraindications to DAPT may favor CEA. Neck CTA characterizes plaque morphology, including calcific versus soft plaque, and defines stenosis severity. It also confirms the anatomic suitability for TCAR, including sufficient distance between the clavicle and carotid bifurcation (ideally 7 cm; minimum, 5 cm) and a common carotid artery diameter ≥ 6 mm. Intracranial CTA evaluates LVO and MeVO and concomitant intracranial atherosclerotic disease⁸; severe extracranial stenosis with ipsilateral disease may prompt concomitant or staged intracranial angioplasty and stenting rather than isolated cervical revascularization, depending on the severity of intracranial stenosis and associated perfusion profile.

Trials of revascularization for symptomatic carotid stenosis have relied predominantly on luminal stenosis thresholds, excluding patients with subthreshold disease and giving limited consideration to cerebral hemodynamics. Although perfusion imaging has been studied, primarily to predict hyperperfusion after CAS, perfusion parameters have not been systematically incorporated into revascularization decisions. Cerebral hypoperfusion, characterized by reduced cerebral blood flow, increased cerebral blood volume, and prolonged time to peak, indicates perfusion failure and identifies patients at increased ischemic and postrevascularization hyperperfusion risk. In severe stenosis, increased cerebral blood volume with reduced flow may reflect compensatory arteriolar vasodilation and exhausted cerebrovascular reserve, indicating maximal autoregulatory compensation and vulnerability to perfusion-pressure changes. Revascularization of a chronically hypoperfused hemisphere may markedly increase cerebral blood flow and predispose to hyperperfusion and intracranial hemorrhage, particularly with near occlusion, pronounced distal vasodilation, and reliance on collateral flow. Severe unilateral stenosis or near occlusion combined with impaired circle of Willis compensation and marked perfusion delay may therefore identify patients at increased risk for hyperperfusion-related hemorrhage after CAS; a preprocedural time to peak > 1.22 seconds has been associated with this risk.⁹ Perfusion severity may also inform treatment urgency. Marked time-to-peak prolongation with reduced flow and a significant penumbra on CTP warrants emergent treatment, analogous to LVO thrombectomy. Similarly, fluctuating symptoms with concerning perfusion abnormalities may warrant urgent or emergent intervention. Thus, revascularization strategy should integrate vascular anatomy, cerebral perfusion, plaque characteristics, and clinical and hemorrhagic risk.¹⁰

Additional considerations apply to near-total and complete carotid occlusion. Patients with near-total occlusion who receive preprocedural heparin to reduce intraluminal thrombus and allow stabilization or washout of thrombus or soft plaque, thereby reducing distal thromboembolism risk before definitive revascularization, are monitored in the intensive care unit (ICU) with close hemodynamic surveillance. Systolic BP is maintained at 160 to 180 mm Hg to optimize cerebral perfusion. At 48 hours, repeat CT stroke imaging, including noncontrast CT, CTA, and CTP, reassesses carotid patency and cerebral perfusion. CAS is subsequently planned when clinically appropriate based on interval imaging and clinical status. In complete carotid occlusion, we additionally perform an acetazolamide challenge to assess cerebrovascular reserve and a transcranial Doppler breath-holding test to assess cerebral vasomotor reactivity using the breath-holding index. Although most

patients do not have hemodynamic failure, a subset develop clinically significant cerebral hypoperfusion and may be considered for endovascular recanalization of the chronic total occlusion or superficial temporal artery-to-middle cerebral artery bypass (extracranial-to-intracranial bypass).

PROCEDURAL IMAGING CONSIDERATIONS

For CAS, our protocol mandates dual embolic protection: proximal balloon occlusion with continuous back bleeding to prevent antegrade flow before lesion crossing, combined with a distal embolic filter to capture emboli when flow is restored. We previously demonstrated universal intracranial flow reversal using a balloon guide catheter (BGC) positioned in the common carotid artery.¹¹ This approach approximates TCAR flow reversal and provides an additional layer of proximal protection beyond distal embolic protection.

We prefer angioplasty with a balloon sized to the nominal internal carotid artery diameter before stent deployment to minimize the potential “cheese-grater” effect of stent tines traversing the plaque, compared with the more uniform force of balloon angioplasty. This may further reduce distal embolization. We preferentially use second-generation stents with micromesh or small-cell designs to limit plaque protrusion and microembolization, including Neuroguard (Contego Medical, Inc.), Roadsaver (Terumo Interventional Systems), and CGuard (InspireMD).

After stent deployment and before restoring antegrade flow, intravascular ultrasound (IVUS) is routinely performed to assess stent expansion and apposition and identify filling defects or plaque protrusion; post-dilatation is performed only when indicated. Routine angiography is avoided to minimize procedure-related distal embolization. If IVUS identifies intraluminal debris, it is aspirated through the BGC before angiography and balloon deflation. Once optimal stent apposition and absence of plaque protrusion are confirmed, the BGC is deflated, antegrade flow restored, and cervical and cranial angiography performed. The distal embolic filter remains in place until complete stent deployment, absence of filling defects, and BGC deflation are confirmed, after which it is retrieved. During TCAR, IVUS is similarly performed after angioplasty and stent deployment to confirm apposition, absence of filling defects, and lack of distal embolization, followed by discontinuation of flow reversal and confirmatory angiography.

POSTPROCEDURAL IMAGING CONSIDERATIONS

After carotid revascularization, patients are monitored in the neuroscience step-down unit or neuro ICU with neurologic assessments at least every 2 hours.

Systolic BP is maintained at ≤ 140 mm Hg. Patients with severe near-occlusive disease and preintervention perfusion evidence of maximal cerebral vasodilation are managed in the neuro ICU, with systolic BP maintained at < 110 mm Hg and liberalized by 10 mm Hg daily until normotensive. This strategy mitigates hyperperfusion syndrome, which may be predicted by CTP findings of vasodilation, increased cerebral blood volume, and reduced flow. Postoperative ultrasonography establishes a new baseline for the treated vessel.

We routinely perform noncontrast MRI after carotid revascularization to detect periprocedural cerebral embolic events, which are more frequently clinically silent but detectable on DWI. CEA remains the benchmark for embolic protection, with DWI lesion rates of 18.1% (95% CI, 14%-22.7%) to 27.1% (95% CI, 25.1%-28.9%).^{12,13} Hybrid TCAR approaches employing flow reversal demonstrate similarly low DWI lesions rates.¹⁴ With contemporary embolic protection systems, new DWI lesion rates after CAS are reported in 37.8% (95% CI, 24.7%-51%) to 40.5% (95% CI, 35.4%-45.7%) of patients.^{12,13} Small studies suggest lower DWI lesion rates with proximal balloon protection (56%-57%) than distal filter protection (71%-78%).^{15,16} Our ongoing analysis of DWI lesions following carotid revascularization suggests an incidence and burden comparable to or lower than those reported after TCAR and CEA (authors' unpublished data). The clinical significance of post-CAS DWI lesions remains uncertain. Some studies associate DWI positivity with long-term cognitive decline, dementia, and subsequent cerebrovascular events, including a 5-year incidence of 22.8% versus 8.8% in DWI-negative patients,¹⁷ whereas others report no association with long-term stroke risk.¹⁸ Silent DWI lesions may reflect limitations of current embolic protection, as 72% to 90% of particulate debris generated during CAS is smaller than the 100- to 280- μ m pore size of available distal filter,¹⁹ as well as the multistep nature of the procedure and associated device exchanges.

CONCLUSION

Traditional imaging has provided the foundation for individualized carotid revascularization according to operator and institutional practice. We advocate for contemporary multimodal strategies, incorporating CTP, intraprocedural IVUS, and routine postprocedural MRI to better characterize cerebral hemodynamics, enhance procedural safety, detect silent cerebral injury, and enable more precise postprocedural surveillance, with the ultimate goal of improving patient outcomes. ■

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