INTERMEDIATE AND NON-ATHEROSCLEROTIC CEREBROVASCULAR IMAGING

INTRODUCTION

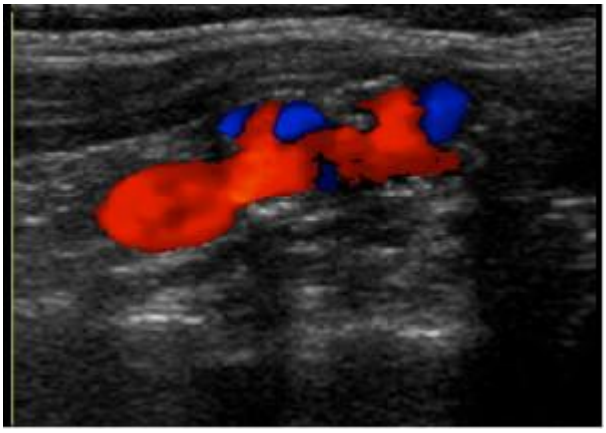
The basic tenets of carotid and cerebrovascular duplex are relatively simple and reflect the basic physiologic and hemodynamics of any medium-sized vascular bed. Simple atherosclerotic disease behaves generally predictably, and duplex criteria founded on fairly solid evidence is available for the evaluation of simple lesions in the carotid distribution. However, not all atherosclerotic disease appears or manifests following this simple pattern, and the vascular technologist and physician reader will be called on to scan and diagnose more complex cerebrovascular disorders of both atherosclerotic and non-atherosclerotic origin on a routine basis. In addition, other local or systemic conditions may impact the interpretation of a study and must be taken into account when suspicion for these external factors is high.

ATHEROSCLEROTIC CAROTID DISEASE

While the diagnosis of atherosclerotic stenoses is often simple following prescribed evidence-based and institutional guidelines, other factors may affect either the interpretation or eventual clinical management.

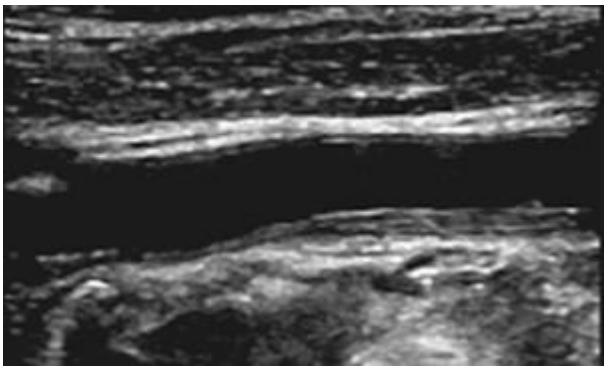
Plaque Morphology

The assessment of carotid atherosclerotic disease typically relies on evaluation of stenotic lesion by spectral doppler criteria. However, the qualitative assessment of carotid plaque using grayscale imaging can be a useful adjunct. Atherosclerotic plaque is thought to cause stroke by an embolic mechanism as compared to limiting flow. Plaque ulceration can lead to hemorrhage, causing distal embolization of thrombus or plaque material, progression of plaque growth, or both. Identification of so-called “vulnerable” or “unstable” plaques is thought to be improved by qualitative assessment of plaque morphology on B-mode ultrasound.



Plaque ulceration demonstrated in B-mode and color Doppler.

The depth of carotid plaque can be assessed using the combined width of the plaque-free intima and media and used to determine the presence of disease. This intima-media thickness increases linearly with age, but thickens more quickly in response to early plaque. Measurement of the intima-media thickness is done in a longitudinal view in the CCA and proximal ICA. A value of less than 0.9mm is considered to represent normal thickness of these layers. Echolucency is another hallmark of a vulnerable or unstable plaque(1) , as echolucent plaque is thought to represent soft or unstructured plaque at risk of embolization(2). In contrast, plaque echogenicity represents calcification or structured plaque that is at less risk.



Thickening of the intima-media layer in atherosclerotic disease.

Evaluation of plaque morphology is much more subjective than assessment of stenosis by spectral doppler criteria. The plaque must be assessed from both longitudinal and transverse orientations in order to view as much of the area as possible. Qualitative measurements such as minimal, moderate, or severe are suggested for measuring plaque encroachment on the arterial lumen.
The degree of echolucency should likewise be qualitatively assessed and documented. Several classification systems have been utilized for uniform reporting of plaque morphology. The international classification system [3] is one such system that groups plaque into five groups depending on the subjective amount of echogenic material present in the plaque [Table].
Type/ Description
1/ Uniformly echolucent
2/ Predominantly echo lucent (>50%)
3/ Predominantly echogenic (>50%)
4/ Uniformly echogenic
5/ Unable to classify
In this system, plaque types 1 and 2 are generally considered unstable or vulnerable and should be reported as such. Plaque types 3 and 4 are likely composed of higher amounts of fibrous plaque or calcification and so are considered more benign.
Some authors have recommended utilizing intravenous contrast for further definition of plaque(4). Contrast, consisting of ultrasound-dense microbubbles, is injected through an IV during carotid imaging. The contrast is thought to gravitate to new growth microvessels within an unstable plaque. A high concentration of micro bubble contrast may be useful as an adjunct to diagnosis of unstable plaque, although evidence is not conclusive.
Occlusion and Near Occlusion

The management of carotid occlusion and near occlusion has evolved as the understanding of carotid-related stroke has improved, with greater recognition of the role of emboli and microemboli in transient ischemic attack and stroke. Intervention on truly occluded internal carotid arteries has diminished due to demonstrated low risk of future stroke after occlusion. However, near-occlusion of the internal carotid continues to carry a significant stroke risk and both symptomatic and asymptomatic patients with near-occlusion generally warrant
intervention.

It is often difficult to distinguish between an occlusion and a near occlusion, and sometimes multiple imaging modalities are needed. CT angiogram in particular tend to be difficult to interpret in these situations due to artifact from plaque calcification. While angiography is an accurate diagnostic mechanism, it is limited by its invasive nature and a small but concrete risk of stroke from the procedure itself. Duplex has emerged as a useful tool for the identification of near-occlusion.

Ruling out near-occlusion is difficult, but because of the continued stroke risk posed to the patient, several different points must be considered. Because there is low flow in the carotid in a near-occlusion, visualization of active flow on color or spectral doppler may be difficult, especially as echogenic material may not be visible in the lumen even with a complete occlusion. The area of suspicion must be interrogated by color, power, and spectral doppler. Adjunct findings for a complete ICA occlusion include a “water hammer” pulsed spectral doppler signal in the CCA with a sharp systolic upstroke and no flow in diastole, or a low-resistance antegrade ECA trunk flow indicating distal external-to-internal collateralization. Adjunct findings for a complete CCA occlusion may include a patent ICA due to retrograde ECA filling via neck collaterals.

NON-ATHEROSCLEROTIC CAROTID DISEASE

Several other non-atherosclerotic disease mechanisms can cause carotid pathology. Many of these are recognizable by duplex, which can often be utilized as a noninvasive screen or confirmation of diagnosis. These pathologies include carotid dissection, pseudo aneurysm, arteriovenous fistula, and carotid body tumor.

Carotid Dissection

Carotid dissection is caused by a tear in the wall of the carotid artery, often between the media and the adventitia. This process can result from iatrogenic injury, trauma, as a result of an arteriopathy or collagen vascular disease, as an extension from an aortic arch dissection, or spontaneously without any clear etiology.

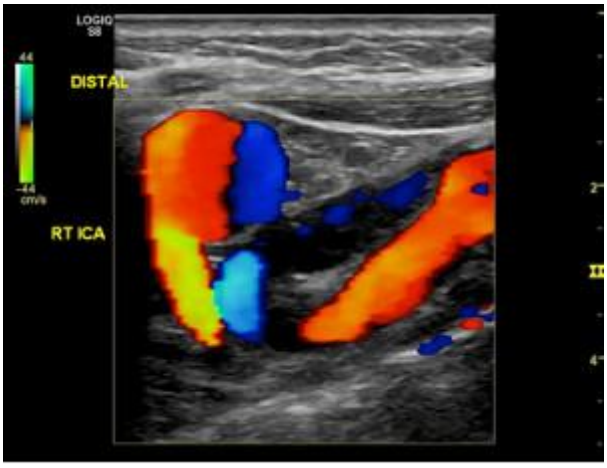
Duplex imaging of a carotid dissection is classically demonstrated as a dual-lumen image on transverse B-mode imaging. Following the dissection flap distally may show either a blind end, or a distal tear with reconnection of the lumens. Dissection may also present with intramural thrombus if the false lumen is thrombosed due to low flow.

Dissection may occur in the CCA, ICA or both. The classic ICA dissection occurs in 35-50 year old females, with trauma or spontaneously. Clinical symptoms include pain, hemispheric stroke symptoms, and facial nerve palsy or Horner’s syndrome. Dissections of the ICA often present with the false lumen occluded with thrombus on duplex imaging. Other B-mode findings may include a mobile intimal flap or a smooth tapering of the ICA but without visualized plaque on a longitudinal view. If a dissection of the ICA is suspected, patency, flap characteristics, flow direction in both lumens, and any stenosis caused by the dissection should be documented by the technologist in order to assist with decision making by the primary physician.

Carotid Pseudoaneurysm

Carotid pseudoaneurysm (PSA) is an extravascular hematoma contained by external tissue and with a patent connection to the artery. PSA is most often caused iatrogenically via catheterization or due to trauma. Less common etiologies include dissection and arteriopathies. Patients typically present with a neck mass or cranial palsy. Due to the formation of thrombus in the pseudoaneurysm, embolization and subsequent stroke is a concern.

Duplex findings of carotid PSA are similar to diagnosis of PSA anywhere else in the arterial system. Findings on B-mode ultrasound include a spherical or oblong extravascular cavity with active flow on color doppler, connected to the artery by a narrow “neck”. Often a “yin-yang” sign will be seen on color doppler in a transverse view as blood flow circulates within the PSA. Spectral doppler may show a classic “to-and-fro” pattern in the neck of the PSA due to active flow into and out of the contained space. Although limited data exists on the clinical significance of PSA characteristics, the diameter, neck length, and area of active blood flow compared to thrombus in the PSA should be documented for reference.



Carotid pseudoaneurysm showing “yin-yang” sign and arterial connection.

Small pseudoaneurysms may thrombose spontaneously, requiring no interventional management; therefore, serial duplex examinations may be required. Clear documentation of PSA characteristics may influence clinical decision making if the PSA is spontaneously thrombosed, or alternately if the neck and sac remain patent.

Arteriovenous Fistula

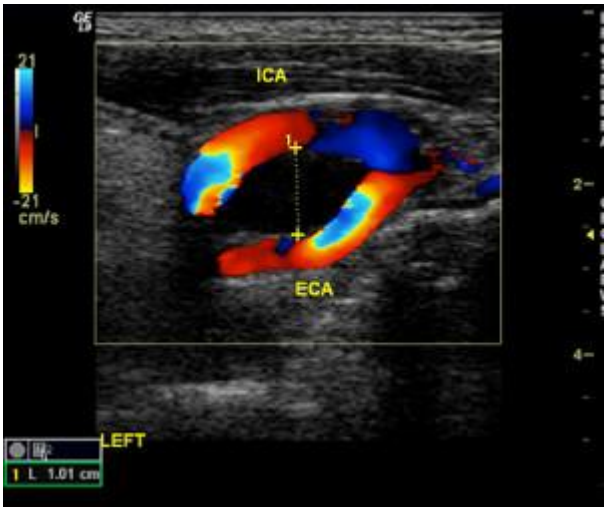
A carotid arteriovenous fistula (AVF) refers to an anomalous connection between the carotid artery and a venous structure, usually the accompanying internal jugular vein. Iatrogenesis or trauma is usually the cause of these abnormalities. In an AVF, blood flows from the high pressure arterial system into the low pressure venous system, resulting in characteristic properties on duplex examination.

Duplex ultrasound findings for diagnosis of an AVF may include turbulent flow in the area leading up to the fistula. Pulsatile, accelerated, or turbulent flow may be seen in the vein at the site of the fistula, and a high-velocity jet may be seen. A connecting channel between the artery and vein is seen in some situations. However, AVFs are not uniform and may not always be visualized despite high clinical suspicion.

Carotid Body Tumor

Carotid body tumors are paragangliomas that develop in the notch at the bifurcation of the common carotid, often splaying the internal and external carotid arteries apart. Presenting symptoms may include

headache, neck pain, and neck mass. Duplex is often used as a diagnostic modality or for preoperative planning. Findings of a carotid body tumor include a highly vascular structure with many feeding vessels at the bifurcation of the common carotid. Vascularity of the mass can be identified using color doppler. Identification of the extent of the tumor and any encasement of the ICA or ECA trunk can be helpful in preoperative planning.



Carotid body tumor at the CCA bifurcation.

Duplex is used extensively for vessel surveillance after carotid intervention. In the short-term, duplex is useful for identifying early restenosis or undiagnosed technical issues such as incomplete endarterectomy or stent deformation in vessels that have been recently intervened upon. However, the post-intervention duplex images can often look different due to disruption of normal anatomy.

After Carotid Endarterectomy

After carotid endarterectomy, duplex exam of the vessel will show the absence of the normal intima-media stripe, as this portion has been removed during the operation. Sutures in the patch or closure may be echogenic and must be distinguished from pathologic structures such as retained plaque. In a recent postoperative patient, attention should be paid to the endpoint of the endarterectomy to verify that no significant plaque has been retained and the intima is not significantly disrupted. On color doppler flow should be generally laminar and free of large areas of disturbance; significant turbulent flow may indicate intimal flaps, plaques, or residual stenosis.

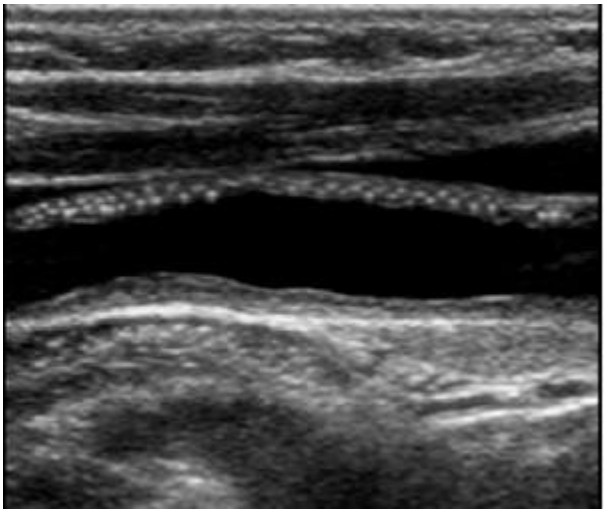
Post carotid endarterectomy showing echogenic suture line

The endarterectomy process triggers neointima growth in the carotid wall. This neointima may manifest as wall thickening of the entire endarterectomized area; however, this thickening is benign unless associated with significant luminal narrowing. Spectral doppler flows are usually within normal limits using typical criteria even at the first follow-up period.

At long-term follow-up of 1-2 years, intimal hyperplasia may play a significant role in recurrent carotid disease in addition to development of new plaque. Intimal hyperplasia manifests on B-mode ultrasound as a smooth focal or diffuse narrowing of the endarterectomized area. Standard criteria should be used to judge the severity of these areas of stenosis. Recurrent stenosis greater than two years from the operation may also be due to recurrent atherosclerotic disease.

After Carotid Stenting

Postoperative duplex after carotid stenting should show laminar flow throughout the stent. Occasionally spectral doppler may detect flow changes due to difference in caliber between the distal vessel and the stent. Surveillance of stents follows a similar pattern to endarterectomy. Criteria for the diagnosis of severe restenosis after carotid stenting remain debated, but authors have advocated a luminal loss of >75%, PSV > 300 cm/s, EDV > 125, or a ratio of >4 as potential criteria [5].



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