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Ultrasound

Case study

Characterizing carotid artery stenosis using high-resolution imaging

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Patient history and findings

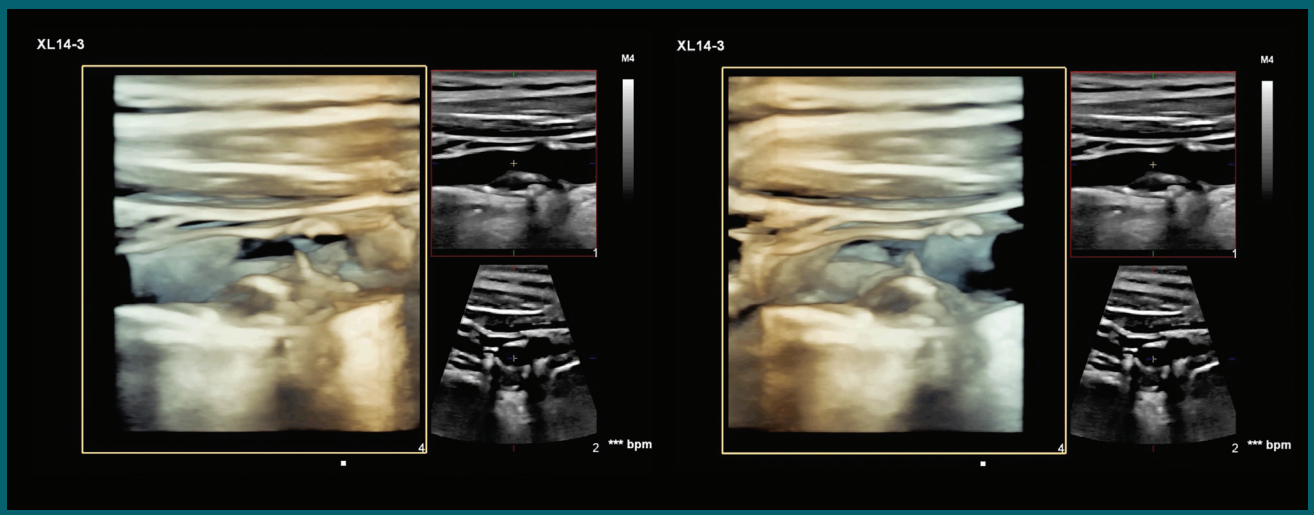
A 64-year-old woman has a past medical history of hypertension, hyperlipidemia, and a former 20-pack-per-year history of tobacco use. In June 2020, she had symptoms of transient left visual loss with aphasia, both of which resolved within four hours. An MRI of her brain at that time did not reveal any abnormalities. She was diagnosed with a transient ischemic attack. A CT angiogram of her head and neck did not reveal any significant intracranial or extracranial stenosis. The CT angiogram revealed a mild atherosclerotic plaque in the right internal carotid artery with approximately 40% stenosis. A carotid duplex ultrasound exam was performed using a Philips EPIQ 7 ultrasound system with both the L12-3 and XL14-3 transducers to assess the characteristics of the plaque.

Delineating fine-structure architecture

Both transducers were able to show the complexity and the calcifications of the right internal carotid plaque; however, the XL14-3 grayscale evaluation was much better able to delineate the fine-structure architecture compared to the L12-3 transducer. Looking at these high-resolution images, one can easily see that this plaque shows a thin cap with some calcific elements and a lucent area behind it, which likely represents softer elements such as lipid-laden plaque or non-calcified thrombus. At the apex of the plaque on the posterior wall of the artery, the slightly less bright portions of the plaque above the lucent area may represent partially calcified thrombus, but this is only a conjecture.

What is clear is that this is a highly complex plaque that shows at least some of the characteristics associated with plaque activity and instability. There is also deep calcium seen at one spot, with acoustic shadowing behind the deep calcium that is clearly distinct from the acoustically non-reflective portion of the more superficial plaque. Pulse Doppler evaluation of the right internal carotid artery revealed a peak systolic velocity of 63 cm/s and an end diastolic velocity of 24 cm/s. This is consistent with a "non-significant" (<50%) stenosis and reminds one that, despite the clinical approach generally taken and earlier evidence showing that stroke incidence goes up in higher grade stenoses, not all strokes come from a tight narrowing within the arterial lumen.

In this case, the clinical presentation with transient aphasia and left-sided visual field loss suggests a contralateral hemispheric transient ischemic attack. Language dominance has been seen to be left-hemispheric in nearly 95% in early clinical reports; however, studies using fMRI have shown that 7.5% of patients overall may have right-dominant language sidedness and that among the left-handed, 27% may have right-dominance.[1,2] Therefore, this plaque may well have been responsible for her symptoms. Given the high-risk appearance of this plaque, the decision was made to escalate antiplatelet therapy to a P2Y12 inhibitor (clopidogrel) and she was started on high-dose statin.



Figures 1 and 2
3D imaging with the XL14-3 transducer.

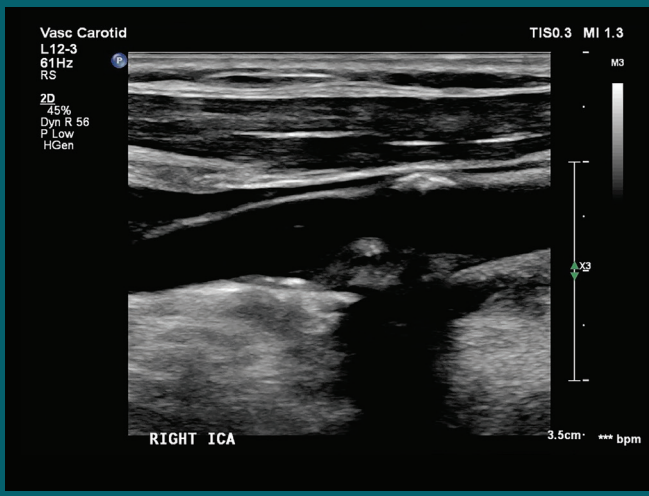


Figure 3
Right internal carotid artery (ICA) imaging with the L12-3 transducer.

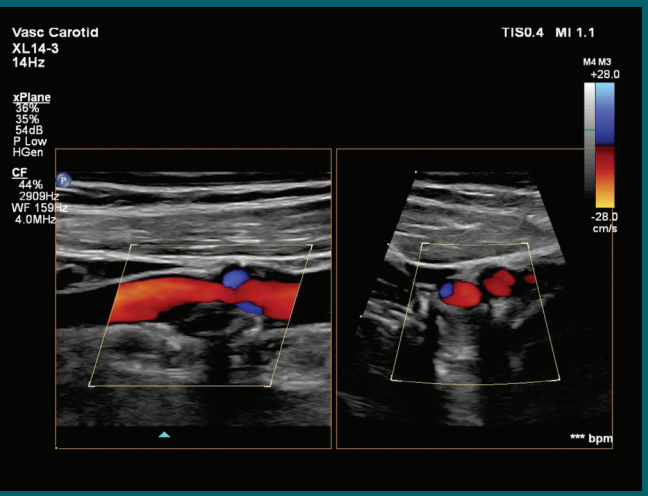


Figure 4
xPlane and color flow Doppler imaging with the XL14-3 transducer.

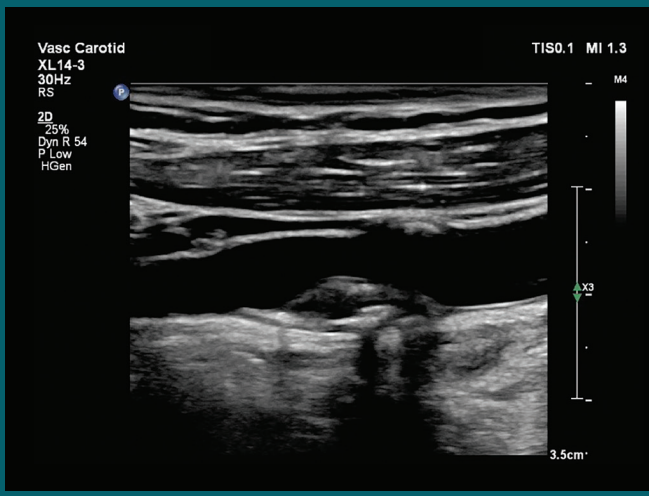


Figure 5
2D imaging with the XL14-3 transducer.

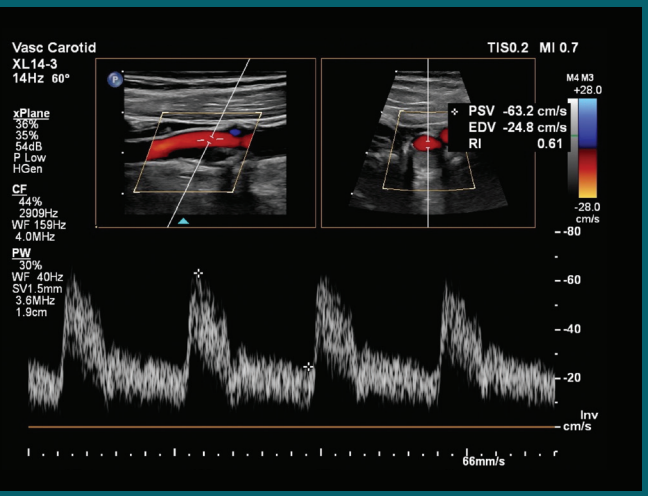


Figure 6
Imaging including pulse wave Doppler with the XL14-3 transducer.

Evaluating carotid artery stenosis

The prevalence of asymptomatic carotid artery stenosis (CAS) is low. In a meta-analysis, the prevalence of asymptomatic CAS (≥50% stenosis) for males and females aged <50 years was 0.2% and 0%, respectively. The prevalence for males and females aged ≥80 years was 7.5% and 5.0%, respectively.[3] Given these low rates, general population screening of asymptomatic patients has not been recommended. However, incidental detection of CAS may occur if carotid artery imaging is done for the evaluation of a carotid bruit, of patients with documented other atherosclerotic disease coronary artery disease (CAD), peripheral artery disease (PAD) or for detection of subclinical atherosclerosis. Once CAS is detected, patients should be evaluated for signs or symptoms of a prior stroke or transient ischemic attack. All of these patients should be screened for modifiable risk factors for cardiovascular disease.[4]

The dreaded clinical consequence of carotid artery stenosis is an ischemic stroke. The risk of an ischemic stroke in an asymptomatic patient with a >50% carotid artery stenosis remains low, at <1% per year.[5]

Asymptomatic carotid artery stenosis is nonetheless a marker of significantly increased risk of a cardiovascular event and should be considered the equivalent of the finding of cardiovascular disease.[6]

Studies have suggested that progression of the degree of CAS over time may be related to increased stroke risk.[7] Evidence of silent embolic strokes, as seen on MRI or CT, is another marker of increased risk of stroke.[8] Some data suggests that plaque morphology is another marker for increased risk of stroke.[9] Use of ultrasound or MRI allows for evaluation of plaque morphology. Ulceration, plaque area, intraplaque hemorrhage and plaque echogenicity may be useful to identify patients with asymptomatic CAS who are at higher risk of stroke.[10] More recently, the ongoing BIOVASC study[11] using metabolic activity scanning of plaque with an FDG-PET/CT co-registration technique suggests that the finding of active plaque on one side is meaningfully associated with contralateral activity.

Conclusion

The risk factors for carotid artery stenosis are equivalent to those for cardiovascular disease and put the patient at higher risk for future cerebrovascular and cardiovascular disease. Therefore, it is important to focus on intensive medical therapy to reduce future risk. The risk of stroke in patients with asymptomatic carotid artery stenosis on intensive medical therapy is low enough that many disagree with the need for revascularization and opt for intensive medical therapy.[11] Intensive medical therapy includes the use of a high dose statin, antiplatelet therapy with aspirin, strict blood pressure control, glycemic control, tobacco cessation, healthy diet, weight loss and regular exercise.

The role for revascularization in asymptomatic patients with carotid artery stenosis remains debatable. Ongoing clinical trials are continuing to compare revascularization with modern intensive medical therapy in asymptomatic patients. Clearly, based on early trials such as ACAS, stenoses less than 50% do not warrant revascularization in symptomatic or asymptomatic patients. Stenosis between 50-69% in asymptomatic patients are generally treated with intensive medical therapy alone. Patients should be surveilled for progression of stenosis. For CAS of 70-99% in asymptomatic patients, the current guideline (where the patient is medically stable and has a life expectancy of greater than five years) recommends either revascularization along with medical therapy, or intensive medical therapy alone.[12]

The ongoing CREST-2 trial is working to compare these two strategies.[13] There is no role for revascularization to prevent stroke for a chronic total carotid artery occlusion. Information on the complexity of a plaque may influence the decision as to medical therapy or anatomic correction.

The choice of mode for revascularization remains controversial. The options are surgical carotid endarterectomy or carotid artery stenting. The original trials showing the benefit of revascularization in high-degree stenosis were done with carotid artery endarterectomy. The short-term periprocedural-related stroke and death rate were reported to be higher with transfemoral carotid artery stenting than with surgical endarterectomy; however, the outcomes with carotid artery stents continue to improve, and the recent 10-year CREST trial results suggest long-term outcome equivalency between the two methods.[14]

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