Ultrasound Characteristics of Adherent Thrombi in the Common Carotid Artery

As neurologists, we read with interest the report by Schlachetzki et al regarding ultrasound findings of a mobile thrombus in the common carotid artery (CCA), and we would like to share our similar experience regarding this rare phenomenon.

We recently examined, by ultrasound, 2 cases of focal adherent thrombus in the CCA that led us to conclusions similar to those of Schlachetzki et al. In our 2 cases, we identified 4 ultrasonographic characteristics of an adherent thrombus in the CCA, which is a rare finding in this large and nonturbulent vessel. (1) The thickness of the intima-media was unaffected, which also seems to be the case in the report of Schlachetzki et al. (2) The CCA thrombus had a “cigar-shaped filling defect” appearance, as was previously described by conventional angiography and as was seen in the case of Schlachetzki et al. (3) The focal adherent thrombus in the CCA was compressible, suggesting a soft thrombus rather than a hard atherosclerotic plaque; however, the 3 CCA thrombi had different echogenic patterns. That reported by Schlachetzki et al seemed to be hyperechoic, but without acoustic shadowing, whereas one of ours was isoechoic and the other was hypoechoic. (4) There was no significant increase in blood flow velocities measured by ultrasound, despite luminal narrowing; this may be explained by either the length of the stenosis or the soft structure.

The last 2 criteria (compressibility and the hemodynamic effect) cannot be discussed in detail because Schlachetzki et al did not provide this information in their report. It is important to note that the presumed source of the CCA thrombus in both Schlachetzki et al’s case and our own first case was cardioembolic (atrial septal aneurysm and aortic valvular strands, respectively). A MEDLINE search found a similar report by Kimura and Uchino about duplex carotid ultrasound findings in 2 patients who had emboli with a cardiac source in whom a mobile thrombus was found in the CCA. Although the risk factors and potential causes of CCA thrombus formation in the absence of significant atherosclerosis are currently only speculative (eg, embolic occlusion from a cardiac source), our first case and that of Schlachetzki et al provide further evidence for a CCA thrombus of cardioembolic origin.

One of our 2 cases, Kimura and Uchino’s 2 cases, and Schlachetzki et al’s 1 case have similar features. Therefore, we can confirm that a thrombus of cardiac embolic origin can be lodged in a large and nonturbulent vessel, such as the CCA, and can be assessed by duplex ultrasound.

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Response

We appreciate the interest of Dr Kemény and colleagues in our ultrasound finding of a mobile atheroma in the common carotid artery (CCA). In their letter describing findings from 2 similar cases, they discuss various important aspects.

All reported thrombi have a “cigar-shaped filling defect.” We think this may be attributed to the reversed flow behind the luminal narrowing, as seen physiologically within the carotid bulb; this area of reversed flow is referred to as the area of boundary layer separation. In pathological conditions, it may be referred to as the zone of poststenotic turbulence. This reversed flow may cause a washout defect at the distal base of the atheroma, leading to this bizarre shape of the thrombus.

In contrast to Dr Devuyst and colleagues, we did not perform any compression tests of the CCA because of the potential embolic risk involved. The real-time image sequences are not suggestive of any compressibility; however, our intraoperative findings confirmed a highly fragile thrombus. Under these circumstances, we would not have dared a compression test, and we would definitely advise against anyone doing so. Blood clot echogenicity depends on the hematocrit, fibrin content, and ultrasound frequency applied, and it undergoes various phases from echogenic to echolucent and back as organization and lamination proceed. Our B-mode investigation revealed an echogenic thrombus with areas of low echogenicity. However, ultrasound was performed using a tissue harmonic imaging sequence (transmit frequency, 3.5 MHz; receiving frequency, 7 MHz), which results in a higher contrast resolution. Further studies must be performed to evaluate whether tissue harmonic imaging is superior to conventional B-mode sonography in detecting echolucent thrombi.

Like Devuyst et al, we only found a moderate, nonsignificant velocity increase within the area of stenosis. An increase of flow velocity in moderate-resistance vessels, such as the CCA, will occur only with a diameter reduction >50% that is also equivalent to a >75% reduction in cross-sectional area, which was not apparent in our case.

We strongly agree with Kemény and colleagues on the importance of the cardioembolic origin of the CCA thrombi described thus far. To date, the reason for this kind of thrombus remains obscure. We believe, however, that in routine diagnostic ultrasound, these thrombi may often be overlooked when in the isogenic or hypoechogenic state or due to minor color-filling defects. This may well have an impact on therapeutic strategies in stroke patients. In the future, thrombus-targeting ultrasound contrast agents, like those described by Takeuchi et al, may help visualize the source of embolization and better detect isogenic or hypoechogenic thrombi.

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