By examining the cervical arteries, we sought to identify high-risk atheromatous plaques that could cause future stroke. Currently, risk stratification of carotid atheromatous plaques is basically centered on the degree of stenosis that it causes in the carotid bifurcation and in the internal carotid artery, being measured by angiography and Doppler.

Despite the importance that this traditional model attributes to the vessel lumen size, hypoflow is not a frequent cause of stroke. About 90% of infarction caused by carotid atheromatosis are due to plaque rupture and consequent distal embolization.

Carotid atheromatous plaque is considered unstable when it has a hemorrhagic component (Figure 1, for example), greasy core >40% and thin or broken capsule. Carotid atheromatous plaques were first studied in vivo by magnetic resonance imaging in 1996 by Toussaint et al. Since then, several studies have been conducted and it is now well established that magnetic resonance imaging is the most accurate method to identify the constituents of plaque instability and the presence of these elements significantly increases the risk of stroke.

Despite the usefulness of magnetic resonance imaging, it takes time to be incorporated into clinical practice, as it demands specific high-cost equipment and a long scanning time. About two years ago, the black-blood 3D sequences (T1 FSE with variable flip angle) became available. These sequences significantly increased the spatial definition of vessel wall images and allowed us to identify the characteristics of atheromatous plaques using conventional equipment (neurovascular coil). This change made the test accessible and enabled it to be incorporated into the clinical routine.

The tests of choice for carotid assessment are still Doppler ultrasound, computed tomography angiography and magnetic resonance angiography. When these methods reveal atheromatous plaques causing stenosis above 50%, magnetic resonance imaging of the plaques is recommended.

**Figure 1** – Magnetic resonance imaging. T1 3D black-blood sequence. The plaque to the right on the axial plane (A) presents hypersignal in T1 compatible with hemorrhage, while the left side plaque presents hyposignal, compatible with protein/collagen component. Image on the sagittal plane of the right-side plaque (B) demonstrates the extent of plaque hemorrhage.
The characterization of instability components in magnetic resonance imaging suggests that the plaque is of high risk and favors the choice of surgical and endovascular procedures or even close clinical follow-up.

Even when Doppler ultrasound, computed tomography angiography or magnetic resonance angiography do not find stenoses above 50%, the carotid plaque may be the cause of a stroke or transient ischemic attack of undetermined origin. When the plaque grows out of the vessel lumen (positive remodeling), the luminal repercussion is small and does not cause any relevant stenosis that can be assessed by angiographic methods. In these cases, magnetic resonance imaging can identify the plaque, as it sees the tissue around the vessel and not just the lumen. If the plaque is hemorrhagic or lipid-derived, stroke or transient ischemic attack are no longer cryptogenic, and the etiology turns out to be the carotid artery.

At present, it is possible to use magnetic resonance imaging to distinguish hemorrhagic and lipid (unstable) plaques from fibrous plaques (stable), and to know whether the plaque actually represents high risk for a future stroke or even the cause of stroke hitherto considered cryptogenic.

References