Letters to the Editor

Fibrous Cap Thickness and Stability of Carotid Atheromata

To the Editor:

We read with great interest the article by Li et al.1 The authors are to be congratulated for their excellent study. We totally agree that clinical decision-making in patients with carotid disease should take into consideration plaque morphology (eg, fibrous cap thickness) as well as the degree of lumen stenosis. However, we would like to express some concerns on the potential clinical implications of the study.

In our view, the title of the article is somewhat misleading: although the authors intended to construct a flow-(internal carotid) plaque interaction model, they essentially created a model that, despite differences in vessel diameter, would better represent atheromatous disease in the common carotid artery, the distal internal carotid artery, the coronaries or the lower limb arteries. This difficulty to approximate the extremely complicated hemodynamic and anatomic conditions of the human carotid bifurcation is clearly stated by the authors. Therefore, any results of the study implying changes in our current indications for carotid endarterectomy should be interpreted with extreme caution. In particular, the authors state that "when the fibrous cap thickness is <0.1 mm, even a small degree of luminal stenosis (10%) may induce a large plaque stress and could result in plaque rupture. This is consistent with the finding from histological data that when the fibrous cap thickness is <0.1 mm, the plaque can be classified as vulnerable." This is a hyperbole without any solid evidence suggesting that it may pertain to the vast majority of stroke patients. We know for sure that patients with symptomatic carotid stenosis <50% do not benefit from endarterectomy,2 and, therefore, even if a 10% or 20% stenosis (which is hardly seen even with a top quality intra-arterial angiography) is detected in a patient with stroke, certainly endarterectomy will not be offered.

Moreover, a cap <0.1 mm may be thin for a coronary plaque, but we do not hitherto know the critical cap thickness that renders a carotid plaque a "vulnerable" one. This extrapolation of data from the coronaries to the carotids also pertains to the 300 kPa threshold of rupture stress. Using their model, the authors found the stress concentrations at the shoulder regions of the plaque. However, histological data suggest that, in contrast to the coronaries, carotid plaque rupture appears to occur mostly at the midportion of the plaque rather than at the shoulder area.3 Furthermore, luminal surface changes have been shown to occur in the most severely stenotic regions of both symptomatic and asymptomatic carotid plaques, with surface irregularities being notably absent in areas of the plaque distant from the stenosis.4 Once more, the above data undermine that what applies to the coronaries does not necessarily apply to the carotids as well.

We have recently described an ultrasound method of measurement of the fibrous cap of stenosing (>70%) carotid plaques.5 We have demonstrated that carotid plaques with mean fibrous cap thickness <650 μm have a high possibility of being symptomatic. The equivalent value for the minimum cap thickness was <460 μm, but its discriminatory accuracy was much lower because the measured values were quite close to the axial resolution of the ultrasound system. Despite its limitations, the study by Li et al corroborates our results because for a plaque with a cap thickness of 500 μm the degree of luminal stenosis required to reach the (arbitrary) stress threshold for rupture (300 kPa) is 65%. Of note is that in the model by Li et al the cap thickness was kept constant all over the plaque surface, whereas in vivo plaques may have caps that are thin (and perhaps prone to rupture) over much of the necrotic core yet thick over the shoulder regions and vice versa.

Disclosures

None.

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