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Carotid Plaque Assessment

A Bumpy Road to Improved Risk Prediction

We congratulate Nambi et al. (1) on their study of improved prediction of coronary heart disease (CHD) risk by including the information on carotid plaque presence in the ARIC (Atherosclerosis Risk In Communities) study. Inclusion of plaque in the risk prediction model reclassified over 10% of individuals into the higher risk category beyond the levels of carotid intima-media thickness (CIMT) and traditional risk factors. The study is significant because the ARIC investigators acknowledge that small, nonstenotic carotid plaque might be a different phenotype of atherosclerosis, carrying an important contribution to the vascular risk beyond CIMT. Plaque presence was defined if 2 of the following 3 criteria were met: CIMT >1.5 mm, abnormal wall shape, and abnormal wall texture. This is a somewhat novel approach, because Dr. Ward A. Riley (a reputable CIMT and ARIC investigator who unfortunately is no longer with us) believed that “whatever is between intima and media represents CIMT” (Dr. Ward A. Riley, personal communication, 2001). In the recent CIMT meta-analysis (2) very little is mentioned regarding the difference between CIMT and plaque and the prognostic importance of carotid plaque. Carotid plaque is a distinctive phenotype of atherosclerosis, most likely is not a simple continuum of CIMT progression, and predicts stroke and CHD risk better than CIMT (3).

Interestingly, the ARIC investigators report that slightly more subjects were reclassified to a lower risk group (approximately 12%) than to a higher risk group (approximately 11%) after adding CIMT and plaque information. No one was reclassified from the low-risk group (<5% estimated 10-year CHD risk) to the high-risk group (>20% estimated 10-year CHD risk). In the NOMAS (Northern Manhattan Study)—a prospective, multi-ethnic, urban, population-based cohort—the presence of small, nonstenotic carotid plaque reclassified 44% of the low-risk individuals (<10% estimated 10-year CHD risk) to the intermediate-risk category (10% to 20% estimated 10-year risk) (4). In addition, approximately 12% of subjects in a lower risk category had a 10-year estimated risk of 25%, which reclassified these individuals to high risk (>20% estimated 10-year risk). None of the individuals was reclassified to a lower risk category after adding information on plaque presence—as opposed to ARIC. The NOMAS results are, however, hardly ever cited, possibly because they appeared in *Neurology*, a journal mostly neglected by non-neurologists. Therefore, “the intriguing hypothesis” raised by Stein and Johnson in the Editorial Comment (5) that “carotid ultrasound could be used to identify persons at lower than apparent risk who might be candidates for *less* intensive interventions” might be simply rejected if data from NOMAS and others (6) are considered.

Nevertheless, less intensive intervention should not be advised according to ultrasound imaging data for individuals otherwise estimated at intermediate-to-high vascular risk on the basis of traditional vascular risk factors. We believe these individuals should be treated aggressively, irrespective of a possible lower risk according to the information obtained by the levels of biomarkers, either imaging or soluble.

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Reply

We thank Drs. Rohatgi and Berry and Rundek and Salameh for their interest in our communication regarding the use of carotid intima media thickness (CIMT) and plaque to improve coronary heart disease (CHD) risk prediction in the ARIC (Atherosclerosis Risk In Communities) study (1).

It is important to note that our study tested whether CIMT and plaque can help better predict CHD risk, but it does not have the ability to offer guidance on treatment strategies on the basis of such a risk prediction scheme. Therefore, we completely agree with Drs. Rundek and Salameh that, on the basis of our data alone, one should not decide on decreasing interventions. However, we feel that such a strategy should be prospectively tested, as has been suggested by Drs. Stein and Johnson in the editorial that accompanied our publication (2). Drs. Rundek and Salameh also discuss their excellent contribution from the NOMAS (Northern Manhattan Study) (3), in which they examined the value of adding plaque to the Framingham risk prediction score (FRS). However, some important facts/differences need to be considered.