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Carotid intima-media thickness and coronary atherosclerosis: weak or strong relations?

We would like to respond to a recent article by Bots et al. We agree with the authors’ conclusion that there is a relationship between carotid intima-media thickness (CIMT) and coronary atherosclerosis. However, significant progress has been made in the analysis of CIMT and lesions since the time many of these studies were conducted. Furthermore, the predictability of event risk has evolved from large epidemiological prediction to individual prediction.

In the beginning, this group recognized the importance of CIMT by itself to predict stroke and myocardial infarction on a population scale. Nevertheless, in 2007, the analysis method has clearly evolved since the quoted studies were performed. For example, the authors previously published an article on a related topic and the letter to the editor by Barth et al. addressed similar issues. The response to that letter by Bots et al. stated that ‘our CIMT measurement predicts future disease in a magnitude similar to that of population based studies that use either manual tracings or automated edge detection tracings.’ A fully automated individualized analysis method is now possible and may, given a long-term sequential database, lead to an individual predictability that was not previously available.

Additionally, the fact that the authors are not dealing with all aspects of carotid ultrasound and coronary angiography and the incomplete use of the literature in their meta-analysis may explain, in part, their conclusions. Coronary angiography focuses on the lumen and is generally performed in symptomatic/advanced disease populations, whereas with IMT HeartScan, lesion detection and tissue typing are usually performed in an asymptomatic population. Only considering what is happening in the lumen to assess the disease and not the wall is debatable. Further, the importance of lesion detection as indicated by Spence further underscores that, although in large population studies manual or automated edge detection tracings may demonstrate a relationship, it fails to assess lesions or plaque composition, resulting in low confidence of event predictability on an individual basis. Measuring the area of such lesions, particularly when assessing progression, is much more informative than measuring the thickness alone, because plaque progresses along the carotid artery ~2.4 times faster than it thickens. In a prospective study, a risk score based on age, blood pressure, smoking, and cholesterol predicted only 32% of patients with vascular events over a 5-year period, whereas 77% of events occurred among patients in the top quartile of plaque area.

Finally, the clinical relevance and long-term follow-up in different ethnic and age groups of IMT measurements in combination with plaque formation underscores the importance of current advances in IMT technology. Our large database can reliably predict on an individual basis the likelihood of a cardiovascular complication within several years if no intervention is performed. The SHAPE report highlights the importance of an initial IMT measurement for clinical follow-up. Quantitative IMT in combination with lesion detection and plaque composition assessment is used widely in clinical settings with great predictability on an individual basis for cardiovascular outcomes.

References

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Discrepancy in guidelines for the prevention of thrombo-embolism in patients with prosthetic heart valves

We have read with interest and became aware of diverse therapeutic approaches in several recently published guidelines, referring to patients with prosthetic heart valves and atrial fibrillation.

In the latest European guidelines on the management of valvular heart disease, life-long oral anticoagulation is recommended for all patients with mechanical valves and for those patients with bioprostheses who have additional indications for anticoagulation such as atrial fibrillation, heart failure, or impaired left ventricular function. Indications for the addition of antiplatelet therapy include concomitant arterial disease, in
particular coronary artery disease and other significant atherosclerotic disease or at least one embolic episode in spite of adequate INR.

In the ACC/AHA 2006 guidelines for the management of patients with valvular heart disease, we are guided to prescribe aspirin 75-100 mg once daily for all patients with prosthetic heart valves: aspirin alone in patients with bioprostheses and no risk factors and aspirin combined with warfarin in patients with mechanical heart valves and high-risk patients with bioprostheses.

Risk factors for thrombo-embolism are: atrial fibrillation, left ventricular dysfunction, previous thrombo-embolism, and a hypercoagulable condition. In other words, a patient with any type of prosthetic heart valve and atrial fibrillation should always receive aspirin combined with warfarin.

Lastly, in the ACC/AHA/ESC 2006 guidelines for the management of patients with atrial fibrillation, published just 15 days after the Valvular Heart Disease guidelines, it is clearly recommended for the patients who additionally have any type of prosthetic heart valve to receive exclusively oral anticoagulation, with a target INR related to the type of the prosthesis. As a result, it is not clear whether a patient with a prosthetic heart valve, suffering from atrial fibrillation should receive a combination of anticoagulant and antiplatelet therapy or anticoagulation only.

Obviously, considering the diversity of the above strategies, the therapeutic decision will be eventually based on which guidelines are more familiar to the attending physician and not to pure scientific knowledge.

Without doubt the recommendations of these recent practice guidelines will generate a relative confusion and thus a consensus revision paper between the writing committees might be necessary.

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Discrepancy in guidelines for the prevention of thrombo-embolism in patients with prosthetic heart valves: reply

We read with interest the comments from Dr Stefanidis et al., which point out some discrepancies between the recommendations in the recent guidelines on the management of patients with valvular heart disease and in particular those on the management of patients with atrial fibrillation.

Guidelines are supposed to represent the best of our current knowledge, thus, they can only be supported by the knowledge existing in the field today. Unfortunately, in valvular disease, the level of evidence is more limited than in other domains and recommendations usually rely on expert consensus.

These limitations apply to the use of antiplatelet drugs on top of anticoagulation in patients with valve prostheses. Available trials show that antiplatelet drugs can be beneficial in patients with vascular disease alone as well as in those with vascular disease and prosthetic valves. The decrease in thrombo-embolic risk observed with the addition of antiplatelet drugs seems to be more related to a decrease in complications of coronary artery disease (myocardial infarction, heart failure, and sudden death) rather than to all embolic events. It has not been demonstrated that this benefit is also present in those without vascular disease. In addition, all trials show consistently that when added to anticoagulation these agents increase the risk of bleeding. Thus, the ESC Task Force on Valvular Heart Disease propose to tailor this combined therapy to specific indications and not to expand to all patients.

We recommend tailoring the degree of anticoagulation to the thrombo-embolic risk of the specific prosthesis and the patient. In a given patient with atrial fibrillation, the degree of anticoagulation is higher than those in sinus rhythm with no other risk factors.

Finally, there is no evidence to support the long-term use of antiplatelet agents in patients with bioprostheses who have no other indications.

These recommendations are also that of the ACC/AHA/ESC guidelines on atrial fibrillation, but are different to those in the ACC/AHA guidelines on valvular heart disease.

However, it should be noted that fortunately the ACC/AHA and the ESC guidelines on valve disease are overall consistent in most domains and convey the same messages, the differences being in some topics where evidence is lacking.

Generally speaking, we agree that the large number of published guidelines, even more so if their recommendations are discordant, may well cause confusion for the reader and therefore limit their use in clinical practice, which is the final goal. The comments made by Dr Stefanidis should support the performance of further trials to provide better evidence and also that of consensus papers between the different Task Forces on a given field to underline the main messages and explain the potential discrepancies.