Unraveling the Natural History of Aneurysms by Exploiting Clinical Images: Insightful Follow-up of Localized Aneurysm Characteristics

Eleni Metaxa, PhD¹, Nikolaos Kontopodis, MD², Konstantinos Tzirakis, PhD¹, Christos Ioannou, MD, PhD², and Yannis Papaharilaou, PhD¹

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In addition to abdominal aortic aneurysm (AAA) size, which is the major clinical predictor of rupture risk, growth rate is also being used to support decision making in the clinical management of these lesions. Rapid growth may reflect undesirable remodeling that leads to weakening of the sac wall and may indicate a high rupture potential.1

Currently in clinical practice, AAA growth is determined by the change in maximum diameter over time, and intervention is indicated when the expansion rate exceeds 10 mm/year.2 Although maximum diameter progression can be easily recorded through 2-dimensional computed tomography (CT) imaging and simple multiplanar reconstruction, which is readily available in most vascular institutes, such measurements may not be in accord with the pathophysiology and natural history of aneurysmal disease. Specifically, it has been found that aneurysm enlargement is accompanied by high inhomogeneity in the distribution of wall mechanical properties,3,4 such as stiffness, thickness, and strength throughout the aneurysmal surface.5 Subsequently, AAA expansion is expected to be nonuniform, characterized by significant spatial variability. Thus, a metric that quantifies regional growth may be more appropriate to describe this process compared with the maximum diameter, which may fail to highlight regions of rapid growth. Moreover, rupture, which is the target event to be predicted, is by nature a rather localized phenomenon resulting from material failure of the degenerated aortic wall.6,7 It is therefore reasonable during the evaluation of AAA rupture risk to consider indices that describe both regional (strength, distensibility,4,8 and growth) and global (diameter, sac volume) AAA characteristics. The former are expected to improve rupture risk stratification by complementing maximum diameter measurements. In this context, growth rate should also be evaluated based on local parameters.

The major challenge in quantifying local growth comes from the complexity of the aneurysm enlargement process. In fact, each AAA is characterized by a unique, complex geometric configuration that alters continuously during disease progression. In this regard, the aneurysm may elongate, become tortuous, and present significant torsion and/or bulge toward either side. Therefore, matching corresponding wall regions in serial follow-up scans is not straightforward. To overcome this difficulty, Martufi et al⁹ have recently generated a surrogate model that consists of a center line and 100 consecutive cross sections between the lowest renal artery and the aortic bifurcation. By comparing the diameter of each cross section with its follow-up diameter at the same relative centerline position, the authors showed that neither maximum diameter nor volume measurements over time are able to measure the largest diameter growth of the aneurysm sac. They concluded that localized spots of large diameter growth can be detected through multiple centerline-based diameter measurements over the entire aneurysm sac.

In this issue of the JEVT, Martufi and colleagues10 have gone one step further with this technique and searched for local correlations between the initial outer diameter, thrombus thickness, and wall stress. Such an investigation could provide significant insight into a long-debated subject concerning the
“driving forces” of aneurysm growth. Does a thick or thin local thrombus deposition predispose to aneurysm growth? Is thrombus or peak wall stress location related to high regional growth? And if all these are of high importance, how can they be prioritized or integrated in a statistical model for reinforcing the quality of aneurysm surveillance programs? These questions have already been posed in the literature,11–13 but a definite, convincing answer has not yet been provided.

One of the interesting findings is that a thin intraluminal thrombus layer slows down the local AAA expansion rate, whereas a thick layer accelerates it. They also postulated that stress-mediated expansion weakens as aneurysm progresses, and once the wall is covered by a thick thrombus layer, the mechanical sensitivity of the wall is diminished and no longer leads to an increment in growth. All these observations provide valuable evidence for future hypothesis-driven experimental studies that will further enlighten the natural history of AAAs.

The next step for following and correlating co-localized AAA characteristics would advance from a cross section to a surface-based analysis. This would help improve our understanding of aneurysmal disease evolution and support clinical decision making in AAA clinical management. Our group is currently working toward this goal, with the aim of introducing an aneurysm surface growth estimation method based on local surface deformation analysis.

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