

Commentary: Aortic aneurysms are not created equal



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Disclosures: Authors have nothing to disclose with regard to commercial support.

Received for publication Sept 13, 2019; revisions received Sept 13, 2019; accepted for publication Sept 13, 2019; available ahead of print Sept 20, 2019.

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J Thorac Cardiovasc Surg 2020;160:e261-2

0022-5223/\$36.00

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<https://doi.org/10.1016/j.jtcvs.2019.09.013>



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Central Message

A comparison of aneurysms of differing etiologies reveals microstructural differences.

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Potentially lethal complications, such as rupture and dissection, are more common as aortic aneurysm size increases.¹ It is therefore universally accepted that replacement of the aneurysmal aorta is a preventive treatment for these complications. Determining the size threshold at which to intervene is a more complicated issue; furthermore, patients with genetic aortopathies, such as Marfan syndrome are recommended to undergo earlier intervention because dissection can occur at smaller aortic diameters.^{2,3} The lack of a more personalized approach to aortic aneurysmal disease commonly leads to everyday clinical dilemmas as to when intervention should be recommended, especially for patients with congenital heart diseases (eg, tetralogy of Fallot and bicuspid aortic valve [BAV]) that may be associated with inherently large aortic roots and aortas.^{4,5} Clearly, a more data-driven approach to the dilated aorta is needed to guide clinical decision making on an individual patient basis.

In this month's edition of the *Journal*, Chim and colleagues⁶ present a novel micromechanical and microstructural approach to differentiate BAV aortopathy from idiopathic aneurysmal aortopathy. The data are limited by sample size (N = 39) and the lack of an optimal control group; nevertheless, this study indicates that BAV aortopathy is associated with reduced vessel wall compliance relative to idiopathic aneurysmal aortopathy and non-aneurysmal aortas. As such, Chim and colleagues⁶ postulate that BAV aortopathy may require specific guidelines for intervention, because its microstructure is inherently different from that of idiopathic aneurysmal aortopathy. Though there are limitations to their data, their analysis with nanoindentation demonstrates that aortopathies do possess specific biomechanical signatures.

The goal of all studies that determine the biomechanical properties and important biomarkers of aortopathy is to predict which patients would benefit from earlier intervention—and, conversely, which patients would benefit from delayed intervention and continued surveillance. The next step in the clinical translation of this

study by Chim and colleagues⁶ is to determine how these microstructural changes and changes in stiffness actually influence the failure mechanics of aortic aneurysmal tissue. Specifically, studies would need to measure directly either the aneurysmal tissue toughness⁷ or the amount of energy required to cause irreversible failure. This is not clinically practical, however, because it requires direct testing of resected aortic tissue. Alternatively, understanding how specific biomarkers and non-failure-related biomechanical parameters relate to toughness and other failure-related biomechanical properties would have the most clinical impact.

Although treatment and fundamental understanding of aortopathies have seen significant progress during the past few decades, the key issue of predicting aneurysmal complications on an individual patient basis remains unanswered. Continued research and advances in imaging, structural mechanics and characterization, biomarkers, and genetics are therefore needed to address this unmet medical need.

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