Go big or go home: But think small

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Central Message

Type A dissection repair is a big procedure. Understanding the histology of the process might improve our understanding as well as our treatment of this disease process.

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Acute type A dissection has a very high mortality without treatment and remains one of the most challenging aortic surgical cases that can face the surgeon with a high surgical mortality in the International Registry of Acute Aortic Dissection.¹ The surgical correction of an acute type A dissection presents a number of challenging decisions as well as technical challenges. Can the root be spared? If not, can the valve be spared with a valve-sparing root replacement? Does the arch need to be replaced in part or whole? Should one debranch at least the innominate artery to plan for potential future endograft need? The tissue in dissection generally ranges from bad to worse. How should the surgeon reinforce their suture lines? The surgeon facing the acute type A dissection is presented with big decisions and a big procedure. To gain success the surgeon must go big or go home. But like many big procedures we have mastered as surgeons this is not the whole story.

The more we understand the biology of a disease process, generally the better we can treat it. In this issue of *The Journal of Thoracic and Cardiovascular Surgery*, Leone and colleagues² investigate the biology of the aorta in acute type A dissection to describe the histopathology substrate and provide clinical correlation.² Pathologic specimens were available for study in 158 patients who had repair of an acute type A dissection at their aortic center. They found 2 histologic patterns with 122 (77%) showing degenerative changes and 36 (23%) showing a mixed degenerative-atherosclerotic pattern. No difference between the groups was seen in the clinical events of mortality, major aorta-related events, or cardiovascular non-aorta-related events. A clinical difference in long-term non-aortic-related events was seen with more in the mixed group ($P = .046$). Histopathologic differences were seen with the degenerative group showing more intralamellar-mucoid extracellular matrix accumulation (86% vs 66.7%, $P = .017$) and less translamellar collagen increase (9.8% vs 50%; $P < .001$). These results just reinforce what surgeons already know

about the poor tissue strength in degenerative aortas and the more systemic nature of the atherosclerotic process. At first blush the clinician might ask how does this information affect my clinical decisions in acute type A dissection? My honest answer is that it likely does not at this time but that does not make this information unimportant. Other disease such as cancer has taught us to go from the anatomy of the tumor to the tissue histology to the cell physiology as we seek better treatments. This approach of thinking smaller eventually going to the cellular level might well also pay dividends in aortic dissection treatment and this study is a good stepping stone in that direction.

Even in the clinical realm, approaches to type A dissection are moving in a less invasive “smaller” direction with the use of stent grafts. Open surgical repair for the moment remains the standard in type A dissection. Forward-thinking surgeons might currently go big or go home with open repair but continue to think small as we move toward a better understanding of the biology of dissection as well as “smaller” repair techniques.

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