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Commentary: Genetic variants in thoracic aortic disease—the root of all evil?

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There are currently 37 known genetic variants or mutations associated with thoracic aortic disease, as was most recently summarized by Vinholo and colleagues,¹ and this total may be the tip of the iceberg. Molecular genetics bolsters the ever-expanding catalogue of culprit genes that contribute to this disease and provide the potential of personalized care to individuals suffering from aortic disease. In this current study in the *Journal*, Yang and colleagues² present a retrospective study comparing the operative treatment strategies for acute aortic dissection with attention to those genetic variants that may require aggressive intervention. Recognizing that genomic variants had a much greater reintervention rate after initial surgery than normal variants, they suggest aggressive root replacement and similar arch management be considered in pathogenic variant carriers at initial repair. This concept enables the idea of tailoring aortic care in a typically silent disease with lethal potential.

As noted by the authors, at the time of the initial aortic event, there is a lack of management guidelines for patients with genetic variants. This should not be surprising, as there is, for the patients in this study, a lack of recognition of patients with genetic variants at the time of presentation, since the authors selected acute type A aortic dissections as their

Acute Stanford type A aortic dissection.

CENTRAL MESSAGE

Preoperative recognition of genetic variants associated with aortic disease may potentially alter the surgical management of these patients.

study cohort. If it were possible to provide the genetic information before taking the patient to the operation, the platform of genetic variant aortic disease would be transformed and the treatment algorithm would be tailored to the needs of this patient population. For patients with aneurysmal disease and/or familial thoracic aortic disease syndromes, this approach would be easier. Given the limitations of preoperative emergent genetic testing, one should hesitate to assume that more aggressive treatment should be applied universally to patients with type A dissections.

The pathogenic group in this study showed a predominance of younger, non-sex dominant, normotensive phenotypes. Beyond that, there are few identifying characters to clearly denote the pathogenic group from the benign group without knowing in advance a history of Loeys–Dietz, Ehlers–Danlos, or Marfan or a family history suggesting familial genetic mutation. The suggestion of aggressive root replacement in patients younger, without smoking history or hypertension is not without merit, considering that the data here identify this group as requiring additional procedures to address the root

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at a point in time after the initial event. It is also important to recognize that timely intervention for a type A dissection in an unstable or critical patient may require a “get-in and get-out” approach. For the highly specialized, this may be second nature to address the root during an emergent procedure. However, for the traditional cardiothoracic surgeon who is not at a specifically high-volume aortic root center, this may not even be a consideration.

Norton and colleagues² have shed light on what may become the future of patient-specific aortic surgery. Although the technology and availability lag, the

authors’ vision to pursue this line of inquiry may help tame the lethality of genetic variants in aortic disease.

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