Commentary: Stent-graft induced new entry: Is it just too much radial force?

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Thoracic endovascular aortic repair (TEVAR) for type B aortic dissection has made many advancements but are still associated with several shortcomings, either due to incomplete seal of the primary entry tear, persistent false lumen perfusion or incomplete false lumen thrombosis, progressive aneurysmal dilatation or evolving dissection flap dynamics, adverse events from the deployed stent grafts, or a combination. Stent graft-induced new entry (SINE) has emerged as an important complication related to the persistent radial expansion of the stent graft against the dissection flap, causing a new dissection tear and acute change in aortic bifurmation. They found that the expansion mismatch ratio was a predictor for dSINE and that the complete thrombosis of the false lumen lowered its risk. The authors found no significant difference in the incidence of dSINE when comparing acute versus chronic type B dissection, although the analysis was really underpowered to determine this.1

Although the authors accounted for several differences between groups, type B aortic dissection is a very heterogeneous disease with many clinical, radiographic, and anatomic presentations. Thus, it is likely that there were many unmeasurable confounders inherent to this retrospective analysis that limit the interpretation and generalizability of this study. Having said that, this article brings forward 4 important considerations. First, it highlights the need for better understanding of the mechanical properties of the stent graft and its interaction with the vulnerable dissection flap and distal aorta both early and late in the disease process of patients with type B aortic dissection. Second, it emphasizes the need for proactive/preemptive approaches to prevent the occurrence of SINE, such as the authors’ suggestion to reduce the stent graft expansion mismatch ratios with tapered stent graft designs or combined stent grafts with restrictive bare metal stents. Perhaps more aggressively tapered stent grafts or novel stent graft designs that avoid excessive distal radial forces may further reduce SINE. Third, it elucidates the importance of defining treatment success and failure. In fact, is dSINE an acceptable complication of TEVAR for type B dissection, or is it a preventable adverse event that, when it occurs, reflects a
failure of the primary intervention? Fourth, it stresses the critical importance of lifelong annual clinical and surveillance imaging follow-up for these patients.

TEVAR for type B dissection is far from being a definitive treatment and requires continued advancements in stent graft design and technical proficiencies. Li and colleagues should be congratulated for helping us better understand the risks and consequences of dSINE.

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