Commentary: Clear as blood: Anticoagulation, false lumen patency, and outcomes in acute aortic dissection

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We pursue certain principles when operating on those with acute type I aortic dissection (ATIAD). Foremost is to relieve the patient of his or her imminent mortality. But we also want to leave a competent aortic valve, resect as much diseased aorta as is safely possible, correct malperfusion, resect the primary intimal tear, attempt to restore flow into the true lumen, and obliterate the false lumen. All of this is done to minimize aortic-related death, need for reintervention, and morbidity. However, evidence for this is not robust and is at times divergent.1-5 Take for instance obliteration of the false lumen.

Vendramin and colleagues from the University Hospital of Udine1 report their retrospective study assessing the influence of oral anticoagulation (OAC) therapy on false lumen patency and outcomes following ATIAD repair. There was a 24% operative mortality for all acute type A aortic dissections over the study period. In 188 ATIAD operative survivors-to-discharge, 127 had sufficient imaging to assess the false lumen over time. OAC using warfarin was used in 39 of the patients and the other 88 comprised the no-OAC group. OAC was not a randomized treatment and mechanical aortic valve replacement was the most common indication for OAC.

The authors conclude that OAC with warfarin favored false lumen patency. However, this is analogous to a glass half full or half empty. A partially thrombosed lumen is also partially patent and whether or not the patency or the thrombosis is afforded precedence will alter perception. The OAC group had 54% patent false lumen compared with 38% in the no-OAC group. Statistically, these rates were not significantly different. Complete false lumen thrombosis was also not significantly different; 38% for...
OAC and 34% for no-OAC. The only difference was seen in those with partial false lumen thrombosis/partial patency. And here, it was less commonly seen in the OAC group (8% vs 28% in the no-OAC group). Looking at this another way, if patent or partially patent were an any-patency group the proportions would be 62% in the OAC cohort versus 66% in the no-OAC cohort. But if thrombosed or partially thrombosed were an any-thrombosis group, then this would have occurred in 46% of the OAC patients and 62% of the no-OAC patients. Thus, it is not that OAC favors false lumen patency per se, but that it disfavors thrombosis.

Extension of the repair seemed to induce false lumen thrombosis in this study. False lumen patency was 44% in the hemiarch (HA) patients and 34% in those extended beyond a hemiarch repair (ER). Complete thrombosis was 31% in HA and 48% in ER. Even looking at patent or partially patent as a group, it was 68% in HA and 50% in ER, whereas thrombosis or partial thrombosis was 55% in HA and 64% in ER. All of this points toward extending the repair slightly favoring false lumen thrombosis and slightly disfavoring false lumen patency.

What should be the goal? Preserving patency, inducing thrombosis, or simply avoiding a partially thrombosed state? And why does this matter? Returning to the original point about divergence of evidence, false lumen status—whether full, empty, half full, or half empty—has been suggested to influence outcome and to not influence outcome. The study by Vendramin and colleagues falls into the camp that it does not influence outcome, adding to the divergence of evidence. But it does show that use of OAC was assessed to not influence mortality or reintervention rates and this alone has value.

References