Editorial

Left ventricular hypertrophy in patients with acute type A aortic dissection: a formidable challenge

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Hypertension is the most common risk factor for acute aortic dissection and is a major determinant of left ventricular (LV) remodeling, including LV hypertrophy (LVH) (1). Alterations in LV geometry result in high arterial load and vascular damage, elevated levels of neuroendocrine components, reduced coronary flow reserve, and high risk of LV dysfunction (2,3). The association between LVH and overall mortality is well established; Haider *et al.* analyzed data from 3,661 Framingham Heart study participants and found that LVH was associated with an increased risk of sudden cardiac death: 2.16-fold for any degree of LVH, and 1.45-fold for every 50 gm of LV mass (4).

In their study titled "Prognostic value of left ventricular hypertrophy in postoperative outcomes in Type A acute aortic dissection", Zuo and colleagues (5) from Wuhan, China analyzed the utility of LVH in predicting postoperative outcomes in patients with acute aortic Type A dissection (ATAAD). This was a retrospective, single-center analysis of 193 patients who underwent ATAAD surgical repair over a nearly four-year period. Patients were classified as having or not having LVH according to their echocardiographically measured LV mass index (LVMI) and were propensity-score matched. LVH was noted in 28.5% of the patient cohort. The LVH patients were more likely to be female, to present with cardiac tamponade, and to have lower LV ejection fraction (LVEF). There was no significant difference between groups in the prevalence of hypertension, although the severity of hypertension and number of baseline antihypertensive medications were not known. The incidence of composite major outcomes (CMO), which included operative mortality, stroke, paraplegia, dialysis, and adverse cardiac events, was 30.9% in patients with LVH *vs.* 15.2% in patients without LVH. The LVH group had higher operative mortality (18.2% *vs.* 7.2%) and higher incidences of postoperative stroke, low cardiac output syndrome, and ventricular arrhythmias (5).

Multivariable logistic regression identified three independent risk factors for postoperative CMO: LVH (OR 2.6, P=0.04), hyperlipidemia (OR 3.0, P=0.01), and emergency surgery (OR 2.8, P=0.02). LVMI (as a continuous variable) predicted postoperative CMO with an OR of 1.2 (P=0.02) for every 10 g/m^2 of LVMI. The need for postoperative dialysis, the need for tracheostomy for respiratory failure, and atrial fibrillation were also associated with greater LVMI. Interestingly, although patients with LVH had higher rates of reduced LVEF, logistic regression analysis found no significant association between lower LVEF and postoperative CMO (OR 2.6, P=0.21). Both the LVH and LVMI models showed good clinical utility and consistency in predicting postoperative CMO and adverse cardiac events.

Although LVH is a common echocardiographic finding in patients with aortic dissection, its clinical significance is unclear. In a study by Rocha *et al.*, LVH was unrelated to 1-year mortality in patients with type A aortic dissection (6). Others have found that LVH may be a biomarker for early

mortality after type B aortic dissection (7).

The important questions this study raises are the extent to which LVH contributes to acute aortic dissection and whether treating LVH could improve clinical outcomes. Iarussi *et al.* retrospectively reviewed demographic and echocardiographic data from 63 patients who underwent ATAAD repair over a 10-year period; hypertension was present in 76% and LVH in 79%. These patients were compared to 16 healthy volunteers matched for age, gender, heart rate, and blood pressure. The investigators found that the ATAAD patients had significantly larger LV mass than the control patients, and LVMI was independently associated with aortic arch and descending thoracic aortic diameter (8). Weiner *et al.* identified elevated LVMI as a strong independent predictor of perioperative mortality after all types of adult cardiac surgery (9).

In LVH patients, the increase in LV mass leads to myocardial fibrosis that impairs LV diastolic function. It also changes the intramyocardial coronary vasculature and impairs subendocardial blood flow secondary to increased LV end-diastolic pressure. These changes predispose the heart to increased oxygen demand preoperatively and increased risk of suboptimal myocardial protection intraoperatively, which may result in postoperative myocardial dysfunction and dysrhythmias (10,11). Moreover, LVH may be a surrogate for a more advanced stage of hypertension and associated end-organ dysfunction or vulnerability to injury (neurologic, renal, peripheral vascular, among others), leading to worse outcomes. It may also be an indicator of a malignant form of hypertension with cellular processes that predispose to the development of ATAAD. Whether increasing severity of LVH should lower the aortic diameter threshold for surgery is a potential area of investigation.

Detecting LVH early and treating it with guideline-directed medical therapy may help mitigate these longer-term adverse effects (12). LVH regression is associated with reduced risk of arrhythmias, mortality, and sudden cardiac death, and such regression can be induced with angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, calcium channel blockers, and β-blockers (13).

In terms of intraoperative management, this was a single-institution retrospective study with standardized cooling and cardioplegia strategies and no control group for comparison. Whether LVH in patients undergoing ATAAD would benefit from modification of cardioplegia or myocardial cooling temperature remains to be seen. Perhaps the target mean arterial pressure during or immediately after surgery needs to be recalibrated.

The authors should be commended for investigating the prognostic value of LVH in this patient population, as the association is poorly defined in the literature. However, more work needs to be done to further characterize the impact of LVH on ATAAD and to determine whether current perioperative strategy should be modified to improve outcomes.

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