Left-Sided Valvular Dysfunction Preceding Infective Endocarditis

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Introduction: There are limited studies on the risks of acquiring infective endocarditis (IE) according to pre-existing valvular haemodynamics, an important consideration in an aging population with degenerative valve disease.

Aim: To explore pre-existing left-sided native (NV) and prosthetic (PrV) valvular indices of dysfunction in relation to risk of IE.

Methods: Retrospective analysis of 278 patients (2005-2017) with NV and/or PrV left-sided surgical IE (LSE) at a quaternary-level hospital. Echo results accessible prior to IE onset (2001-) were identified (n=70/278 (25%)), and compared between valve sites that did and didn’t subsequently acquire IE. Grade of valve dysfunction and peak velocity (Pvel) data were collected. Effect size was calculated as the difference between mean velocities (85% CIs). Categorical variables were presented as percentages and p-values derived using Fisher’s or Pearson’s chi-square test (p<0.05=significant).

Results: Of 70 patients (64% of AVs [PrV 76%] and 47% of MVs [PrV 64%] acquired IE) with pre-existing echocardiograms (67% TTEs, mean time (echo to IE surgery)=1.4 yrs; mean age: 61 yrs [SD+/-17 yrs]), data were available for 69/70 (99%) AVs and 61/70 (88%) MVs. Of PrAVs, 54% were biological (3% unknown), and 46% PrMVs mechanical. Risk of LSE: OR=1.9 (p=0.37) and OR=3.5 (p=0.07) if pre-existing >mild (vs. ≤mild) regurgitation and stenosis (MV+ AV), respectively. For ≥moderate, OR=4.8 (p=0.07) respectively. For PrAVs (Figures 2 and 4), mean pre-IE Pvel was 2.8m/s versus 2.3m/s (p=0.29), whilst PrMVs, mean Pvel. (E wave) was 1.5m/s versus 1.2m/s (p=0.04), for valves that acquired and those that didn’t acquire IE, respectively. For native AVs, the mean Pvel. was 2.9m/s versus 2.02m/s (p=0.01), whilst for native MVs, mean Pvel. was 1.01m/s versus 0.98 m/s (p=0.4), for valves that acquired and those that remained free from IE, respectively (Figures 1 and 3).

Conclusion: Peak forward flow velocity is summative of valve stenosis, regurgitation and loading conditions, exposing endothelium to shear stresses. Forward flow haemodynamics and risk of IE were most noticeable for the native AV, likely due to higher velocities of LV ejection, compared with typical low inflow velocities across the native MV. In addition, it may be high velocity regurgitant lesions are more contributory at the native MV site. Higher forward flow velocities across left-sided PrVs were associated with elevated risk, though not statistically significant at the AV site, likely due to small cohort numbers. The mean Pvel prior to IE was almost identical for NVs (2.8m/s) and PrVs (2.9m/s), with no known comparative research identified. This equates to a mild-moderately elevated native AV gradient, but falls within normal reference range for a PrV, suggesting even the early stage of elevated haemodynamics in NVs is an important driver associated with IE risk. For PrVs it remains uncertain if this ‘threshold’ velocity (2.8 – 2.9 m/s) is a common risk factor across all valve types, including normally functioning aortic prostheses, above and beyond the inherent risk associated with foreign material. Although risk of IE was on average associated with mild perturbations in forward flow valvular haemodynamics, increasing categorical grades of valve dysfunction (stenosis>regurgitation), were evidently associated with proportionately greater risk of IE acquisition.