CRT-721

The Cryopreserved Mitral Homograft Valve: 19 Years Experience

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Objective: The aim of this study was to evaluate the long term fate of the cryopreserved mitral homograft focusing on structural valve deterioration (SVD).
Methods: Homograft replacement of the mitral valve was performed in 106 patients. The causes of mitral disease were: rheumatic disease (n=75), endocarditis (n=24) and others (n=7). There were 40 partial homografts and 66 total homografts.
Results: Mean follow-up was 9.3 + 4.7 years (up to 17.8yrs). There were 5 early (< 3months) and 15 late deaths. There have been 5 early (<3 months) and 30 late reoperations. Five patients had endocarditis and 5 had ischemic/haemorrhagic event. As compared to baseline, follow-up echography showed progression of MR grade (from 0.4 to 1.3 $p<0.001$) with stenosis (elevated gradient: from 3.9 to 7.0 mmHg $p<0.001$ and decreased valve area: from 2.3 to 1.7 cm^2 $p<0.001$). Freedom from SVD was 90%, 76% and 65% at 5 years, 10 years and 15 years respectively. SVD was more frequent in total homografts ($p=0.018$ vs partial homografts) and in case of pregnancy ($p=0.016$ vs no pregnancy). Stenosis related to SVD was more pronounced for age<40 years ($p=0.03$) and ring size $\leq$30 mm ($p=0.002$). Pathological analysis of the explanted homografts almost invariably showed dense fibrosis with calcification and no cellularity.
Conclusion: Mitral homografting could be accomplished with early echographic results similar to those of valve repair. SVD produced mixed stenosis with insufficiency and its incidence was comparable to that of bioprostheses SVD. An improvement in the preservation mode of valvular homografts is warranted.

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All Degrees of Mitral Regurgitation Found During Invasive Ventriculography are Associated with All-Cause Mortality

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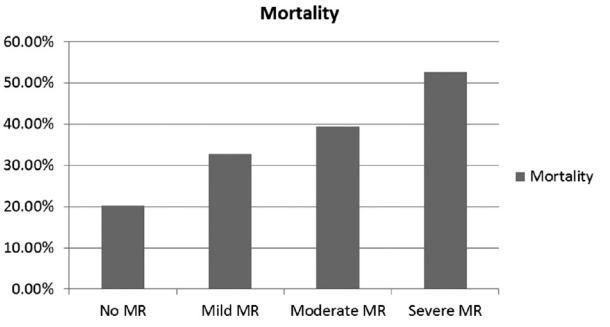
Background: Using a large data base of patients who underwent coronary angiography for clinical reason, we evaluated association between reported degrees of mitral regurgitation (MR) with all-cause mortality.
Method: Using retrospective angiographic data of 1771 patients between 1993 to 1997 from the VA Long Beach Health Care System with documented ventriculography, we evaluated any association between reported degree of MR and all-cause mortality. We performed uni- and multi variant analysis adjusting for age and ejection fraction.
Results: Any degree of MR was associated with all-cause mortality. Total mortality was 20.2 % (296/1465) in patients with no MR vs. 32.7% in patients with mild MR (64/196), p<0,001. Similar to mild MR, any degree of MR was independently associated with all-cause mortality [all MR, 35.1%, (108/306) vs. no MR, 20.2 % (296/1465), p<0.001]. After adjustment for age and ejection fraction, any degree of MR remained independently associated with all-cause mortality. (Multivariate adjusted OR 1.7, CI 1.2-2.3, P<001).
Conclusion: The presence of any MR documented on invasive ventriculography is associated with increased total mortality independent of age or ejection fraction. Our finding suggests that even mild MR has negative prognostic significant.



Valve & Structural Heart

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Is the Sporadic Thoracic Aortic Aneurysm the Result of an Inflammatory Process?

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Background: Sporadic thoracic aortic aneurysm (S-TAA) is potentially devastating with severe morbidity and mortality. The histopathologic underlying abnormality of both ascending aortic aneurysm and dissection is medial degeneration, a pathological entity initially described as no inflammatory lesions of smooth muscle cells and elastic fibres. Accordingly, this study sought to determine whether inflammation characterize medial degeneration and the onset and progression of S-TAA.
Methods: Aortic specimens were obtained from patients (31 men and 11 women, whose median age 66.16 $\pm$ 5.87 years) undergoing surgical repair of TAA (n=24) and TAD (n=18). Histo-pathological and immunoistochemical aorta examinations were executed. Furthermore, genotyping of ten SNPs (single nucleotide polymorphisms) in cases and controls was performed. Plasma inflammatory molecules were also detected in patients and controls using ELISA technique.
Results: A significant inflammatory/immune CD3+CD4+CD8+CD68+CD20+ cellular infiltrate mainly in vasa vasorum of adventitia was observed in case aortas, suggesting its possible migration from these vessels into media and its role in destroying of all components of extracellular matrix and vascular smooth muscle cells (VSCM)s. Consistent of these data, significant higher plasma levels of systemic inflammatory mediators characterized the cases. Different aorta abnormalities, apoptosis of VSCMs and severe MMP-9 amounts were also found in S-TAA aortas. In addition, five very significant associations with S-TAA risk were detected. Of these, D/I ACE (Angiotensin Converting Enzyme) and -1562 C/T MMP-9 (Metalloproteinases-9) SNPs are independent risk factors for S-TAA. Higher tissue and plasma levels of MMP-9 were also observed in -1562T MMP-9 allele carriers. A high S-TAA risk genotype was also detected significantly associated with high levels of systemic inflammatory mediators, immune/inflammatory cells and hypertension.
Conclusion: Results obtained are encouraging and lead to suppose that inflammation also is a shared pathological mechanism for S-TAA. On the other hand, they are in agreement with the emerging evidence suggesting the role of inflammation in several aorta diseases, such as S-TAA.