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CRT-721

The Cryopreserved Mitral Homograft Valve: 19 Years Experience

Francesco Nappi
Centre Cardiologiche du Nord, Paris, France

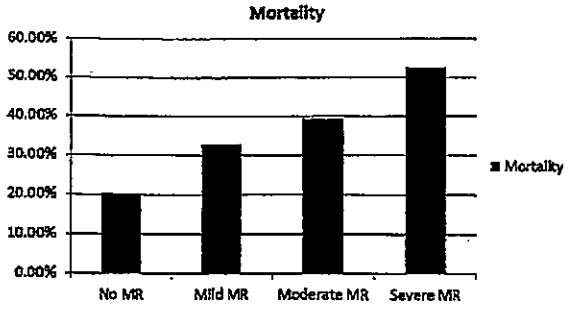
Objective: The aim of this study was to evaluate the long term fate of the cryopreserved mitral homograft focusing on structural valve deterioration (SVD).
Methods: Homograft replacement of the mitral valve was performed in 106 patients. The causes of mitral disease were: rheumatic disease (n=75), endocarditis (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 ≤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.

CRT-722

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

Mohammad Reza Movahed,[1] Kusum Lata[2]
[1]CareMore Arizona and University of Arizona Sarver Heart Center, Tucson, AZ;
[2]University of Arizona Sarver Heart Center, Tucson, AZ

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

CRT-723

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

Calogera Pisano, Carmela Rita Balistreri, Rosalba Franchino, Stefano Borselline, Daniele Merlo, Oreste Fabio Triolo, Eduardo Tulumello, Gianfranco Filippone, Francesco Damiani, Cesira Palmeri, Giovanni Ruvolo
University of Palermo, Palermo, Italy

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 ± 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.

STRUCTURAL HEART

CRT-724

Can the Aortic Wall Communicate with Us?

Calogera Pisano, Carmela Rita Balistreri, Daniele Merlo, Sara Vacirca, Giuseppe Vite, Tommaso Delisi, Salvatore Ocello, Oreste Fabio Triolo, Vincenzo Argano, Cesira Palmeri, Giovanni Ruvolo
University of Palermo, Palermo, Italy

Objective: Association between aortic aneurysm wall and risk of rupture or dissection.
Methods: Aortic specimens were obtained from 73 patients (51 men and 22 women, whose median age 61.7± 10.7 years) undergoing surgical repair of thoracic ascending aneurysm (TAA). Histopathological and immunohistochemical analyses were performed using adequate tissue specimens, appropriate techniques and criteria. Furthermore, genetic risk factors were also investigated.
Results: We identified three phenotypes of TAAs with different quality of aortic wall at the time of operation: phenotype I (normal wall); phenotype II (moderate wall thickness); phenotype III (thin and weak wall). No significant differences were detected in term of demographic and clinical data, co-morbidity conditions and pharmacological treatments. In contrast, significant statistical differences were observed by comparing abnormalities of extracellular matrix components among three phenotypes (fibrosis $p<0.005$; elastic fragmentation $p=0.002$; medionecrosis $p=0.004$; cystic necrosis $p=0.07$; apoptosis $p<0.0001$; MMP-9 amount $p=0.004$). In addition, significant differences both in genotype distributions and allele frequencies were observed for following SNPs (Single Nucleotide Polymorphism): -1562C/T MMP-9 (Metalloproteinases-9), -786T/C eNOs (endothelial Nitric Oxide Synthase) and D/I ACE (Angiotensin Converting Enzyme).
Conclusion: Aneurysm with thin and weak wall at the time of operation should seem genetically and mainly associated with extracellular matrix disorders of aorta wall and consequently with aorta aneurysm complications (rupture and dissection).

CRT-725

The Largest Case Series of Thrombolytic Therapy for Right Sided Mechanical Pulmonic and Tricuspid Valve Thrombosis Showing Very High Success Rate

Maryam Taherkhani,[1] Reza Hashemi Hashemi,[1] Manouchehr Hekmat,[2] Morteza Safi,[1] Adineh Taherkhani,[3] Mohammad Reza Movahed[3]
[1]Cardiovascular Research Center, Modarres Hospital Shahid Beheshti University, Tehran, Iran, Islamic Republic of; [2]CareMore Arizona, and University of Arizona Sarver Heart Center, Tehran, Iran, Islamic Republic of; [3]CareMore Arizona, and University of Arizona Sarver Heart Center, Tucson, AZ

Introduction: Treatment data using thrombolytic in the setting of right sided mechanical valve thrombosis are almost nonexistent and all the guidelines are based on a very small case series. The goal of this manuscript was to perform survival analysis of larges case series of patients with right sided mechanical valve thrombosis treated with thrombolytic therapy.
Method: We reviewed in-hospital and long term outcome data for survival analysis of patients presenting with right sided mechanical pulmonic and tricuspid valve thrombosis treated with thrombolytic therapy from September 2005 until Jun 2012 retrospectively.
Result: A total of 16 patients with definite thrombotic mechanical valve obstruction in tricuspid or pulmonary position were identified (8 in pulmonary and 8 tricuspid position) who underwent thrombolytic therapy. All the patients except one had suptherapeutic INR. All 8 pulmonic mechanical valve thrombosis were in children with 100% respond rate to thrombolytic therapy. In hospital survival rate of pulmonic valve thrombosis treated with thrombolytic therapy was 100%. One year survival rate of successful treated mechanical valve pulmonic valve thrombosis was 87.5%.
Conclusion: Based on our data, we strongly recommend that thrombolytic therapy should remain the first line therapy for the right sided mechanical valve thrombosis in adults or children including children with complex congenital heart disease and patients with mechanical pulmonic valve thrombosis. Surgery should be reserved for patients who fail this treatment.

CRT-727

Percutaneous Balloon Atrial Septostomy for Direct Left Heart Decompression in Patients on Extra Corporeal Membrane Oxygenation

Pradeep K. Yadav, Giselle A. Baquero, Mark Kozak, Christoph Brehm
Pennsylvania State University, Hershey, PA

Background: Severe myocardial dysfunction and added afterload from the Extra Corporeal Membrane Oxygenation (ECMO) arterial cannula may lead to significant rise in left ventricular (LV) end-diastolic and left atrial (LA) pressures. This may result in ischemia, lower likelihood of ventricular recovery and hence raise the already high mortality in this group. Various mechanical approaches have been suggested but available literature in adults is limited.
Methods: We retrospectively reviewed all patients on ECMO that underwent Percutaneous Balloon Atrial Septostomy (PBAS) for left heart decompression at our institution over 3 years. Left heart decompression based on echocardiography, pulmonary edema on chest radiograph and improvement in LA pressure, as well as mortality at 30 days were analyzed.
Results: Six patients, (age 54±18 years; 3 males) were identified. LA pressure improved immediately post septostomy in 4 (80%) / 5 patients. Pulmonary edema improved within 24 hours in 5 (83%) patients. LV decompression on echocardiography was felt in 3 (50%) patients. Four out of six patients died due to multiorgan failure.
Conclusion: Percutaneous Atrial Septostomy appears to decompress left heart in patients on ECMO and profound shock. However, due to multi-organ involvement, mortality in this critically sick group remains high.

Patient	Etiology	Cardiac arrest	Time from ECMO to Septostomy (hours)	Left atrial pressure (mmHg) Pre / Post Septostomy	Pulmonary edema on radiograph at 24 hours	LV decompression on echo	Survival	Time from septostomy to death	Cause of death
1	LMCA MI	No	72	15 / 9	Improved	No	No	Day 17	Multiorgan failure
2	LMCA MI	Yes	456	20 / 20	Improved	No	Yes	-	Left ventricular assist device
3	LMCA dissection	Yes	72	Not available	Improved	Yes	No	Day 9	Multiorgan failure, multiple ischemic stroke
4	Influenza	No	30	32 / 24	Improved	Yes	Yes	-	Discharged on day 31
5	ARDS	Yes	20	60 /30	Not improved	No	No	8 hours	Multiorgan failure
6	MI	Yes	240	16 / 9	Improved	Yes	No	52 hours	Multiorgan failure

LMCA = Left Main Coronary Artery, MI = Myocardial Infarction, ARDS = Acute Respiratory Distress Syndrome.