11:30 a.m.

813-3 Constrictive Pericarditis With Normal Pericardial Thickness on Examination of Histopathology

Deepak R. Talreja, William D. Edwards, Jae Oh, Henry Tazelaar, Gordon Danielson, *Mayo Clinic, Rochester, Minnesota.*

Background: Increased pericardial thickness (PT) has been viewed as an essential diagnostic feature for constrictive pericarditis (CP). While there are reports of clinically-demonstrated CP with normal PT by non-invasive imaging, the evaluation of PT by histopathology in CP has not been described.
Methods: Among 143 pericardial specimens from patients who underwent pericardiectomy for clinically- and/or surgically-proven CP at Mayo Clinic Rochester, 26 (18%) had normal thickness (that is, <= 2 mm). For these 26 patients, surgical pathology and the corresponding microscopic slides were examined for the presence of inflammation, calcification, and fibrosis. Medical records were reviewed to characterize preoperative features including clinical findings, computed tomography (CT) and radiographic imaging, echocardiography and cardiac catheterization.
Results: The 26 patients ranged from 42 to 81 years old (mean, 58) with a striking male preponderance (73%).
No patient had an entirely normal pericardium. Histopathologic abnormalities included fibrosis (85%), inflammation (54%), gross and/or microscopic calcification (35%), fibrin deposition (19%) and focal non-caseating granulomas (7%).
Etiologies for CP included previous cardiac surgery (42%), chest irradiation (19%), postinfarction (7%), idiopathic (7%) and sarcoidosis, chest trauma, and acute viral pericarditis in the remainder (25%). Iatrogenic disease was the most common cause of constriction (61%). The most prevalent symptoms were lower extremity edema (88%), dyspnea (85%), and abdominal distention (77%). The most common physical findings were peripheral edema (85%), increased jugular venous distention (81%) with rapid Y descent (54%), hepatomegaly (54%), ascites (54%), and pericardial knock (38%). This was essentially no different for patients with CP and increased PT.
Conclusions: PT is normal in 18% of patients with surgically-proven constrictive pericarditis although histopathology is abnormal. When clinical or hemodynamic features indicate CP in patients with heart failure, pericardiectomy should not be denied because of normal PT demonstrated by noninvasive imaging.

11:45 a.m.

813-4 The Long-Term Follow-Up Results of Alcohol Septal Ablation for Symptomatic Hypertrophic Obstructive Cardiomyopathy: The Baylor Experience (1996-2001)

Valerian L. Fernandes, Sherif F. Nagueh, Nasser M. Lakkis, Jennifer Franklin, Robert Roberts, William H. Spencer, III, *Baylor College of Medicine, Houston, Texas.*

Background: Alcohol Septal Ablation (ASA) is an accepted procedure for relief of outflow obstruction and symptoms of Hypertrophic Obstructive Cardiomyopathy (HOCM). However, there is still a paucity of data on the long term outcome. Herein we report the long term follow-up from the Baylor College of Medicine HOCM Registry.
Methods: 213 consecutive symptomatic pts (51.2 ± 16.7 yrs, 122 men) who underwent ASA were followed up over 4 years. A mean of 1.35 ± 0.56 septals were injected with 3.0 ± 1.33 cc ethanol per patient. The peak CK post procedure was 1463 ± 884 Units. 33 patients (15%) needed a permanent pacemaker post procedure.
Results: A successful procedure resulted in a significant improvement in symptoms, exercise tolerance and reduction of outflow gradient (table). The initial benefits were maintained at 1, 2, 3 and 4 years post procedure. 24 patients needed a repeat procedure and 5 patients had a myotomy-myomectomy for persistence of symptoms following ASA. 9 deaths (2 procedural, 2 SCD, 5 noncardiac) were recorded during the follow-up.
Conclusion: Alcohol Septal Ablation is an effective procedure with an acceptable risk for patients with symptomatic HOCM. The initial benefits are maintained long term over a 4 year period.

Alcohol Septal Ablation - Follow-up Indices

Indices	Baseline	3 month	1 year	2 year	3 year	4 year
NYHA angina	2.1± 0.7*	1.2± 0.4	1.2± 0.4	1.1± 0.4	1.1± 0.3	1.1± 0.3
NYHA Heart Failure	2.8± 0.6*	1.5± 0.5	1.3± 0.5	1.2± 0.4	1.3± 0.5	1.1± 0.3
Septal Thickness (cm)	2.1± 0.5*	1.6± 0.4	1.3± 0.3	1.2± 0.3	1.2± 0.4	1.2± 0.4
Resting Gradient (mm Hg)	60± 38*	18± 28	9± 19	3± 7	5± 10	8± 14
Provoked Gradient (mm Hg)	95± 56*	54± 66	42± 38	28± 36	21± 20	11± 19
Treadmill duration (sec)	304± 199*	425± 202	451± 210	413± 223	403± 216	405± 161
Ejection Fraction (%)	75± 7*	72± 9	69± 10	67± 10	64± 11	63± 14

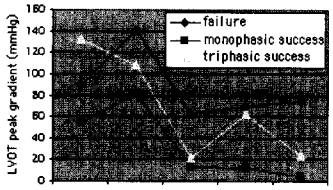
* p value < 0.001 by ANOVA

Noon

813-5 Triphasic Response to Alcohol Septal Ablation

Danita M. Yoerger, Michael H. Picard, Igor F. Palacios, Gus J. Vlahakes, Michael A. Fifer, *Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts.*

Background: Alcohol (EtOH) septal ablation (SA) is an alternative therapy in selected patients with hypertrophic obstructive cardiomyopathy. Acute septal wall motion abnormality followed by later left ventricular outflow tract (LVOT) remodeling are described as mechanisms for early and late relief of LVOT gradient. We have observed that, despite acute reduction in gradient in the cath lab, it may increase in the days after septal ablation. This study characterizes the course of LVOT gradient after SA. **Methods:** 17 patients were assessed by echo at referral (Time 1), immediate pre-ablation (Time 2), immediate post-ablation (Time 3), pre-discharge (Time 4) and at 3 months (Time 5). **Results:** Patients were grouped according to their pattern of LVOT gradient response: monophasic success (n=7), triphasic success (n=7) and failure (n=3). There was no difference in LVOT gradient between the 2 success groups at post-procedure Time 3, but the LVOT gradient in the triphasic success group was significantly increased at time 4, and was not different from that of the failure group at this time point. Groups did not differ in age, baseline upper septal thickness, EtOH amount, percent of upper septum perfused by EtOH or peak CK. **Conclusions:** When a significant decrease in gradient is achieved during SA, predischarge elevation in gradient does not predict failure. This finding supports the mechanisms of acute reversible stunning followed by later long-term LVOT remodeling in relief of gradient after SA.



POSTER SESSION

1110 Heart Failure: Genes Cytokines

Monday, March 18, 2002, Noon-2:00 p.m.
Georgia World Congress Center, Hall G
Presentation Hour: Noon-1:00 p.m.

1110-141 Clusters of Differential Gene Expression of Structural Proteins Between Failing and Nonfailing Hearts: Preliminary Insights From Oligonucleotide Microarrays

W.H. Wilson Tang, Fen-Lai Tan, Gary S. Francis, Christine S. Moravec, Jianbo Li, Carolyn Apperson-Hansen, Patrick M. McCarthy, James B. Young, Meredith Bond, *Cleveland Clinic Foundation, Cleveland, Ohio.*

Background: Simultaneous identification of differential gene expression of structural proteins in the failing heart using microarray technique has not been reported.
Method: Alterations in gene expression were compared between 7 non-failing and 8 failing hearts with idiopathic dilated cardiomyopathy using high-density oligonucleotide microarrays (Hu-6800, Affymetrix Inc, CA) according to conservative criteria developed by our group (Tan et al). 53 known myocardial structural protein components were identified and classified into clusters according to their known functional roles: 1) contractile proteins; 2) sarcomeric proteins; 3) cytoskeletal proteins; 4) membrane-associated proteins (MAP); 5) intercalated disc-associated proteins (IDAP); 6) extracellular matrix (ECM) proteins.
Results: Of the 53 structural protein components identified, gene expression in 20 (38%) were found to be significantly different between failing and non-failing hearts. We observed a consistent trend of altered gene expression in clusters of contractile proteins (down 1.5-fold in α-actin, myosin light polypeptide 2 & 3, α-tropomyosin, troponin T1), clusters of IMAPs (up 1.8-fold in connexin-43) and clusters of ECM proteins (up 1.5- to 5-fold in collagen, fibronectin, fibromodullin, lumican). On the other hand, gene expression of MAPs, sarcomeric and cytoskeletal proteins varied widely within their clusters, but significant changes were observed in α1-actinin (down 3.6-fold), α1-tubulin (down 1.7-fold), α1b-tubulin (up 1.6-fold), β2-tubulin (down 1.5-fold), pinin (up 1.8-fold) and α-spectrin (up 1.5-fold). Discrepancies in gene expression of some MAP/sarcomeric proteins between our study and published reports may warrant further investigation.
Conclusion: Microarray technology provides an alternative strategy whereby numerous myocardial genes can be studied in a simultaneous manner. Clusters of gene expression of structural proteins can be explored according to their functional domains, with genes that control contractile function being consistently downregulated whereas genes associated with structural integrity have a more heterogeneous pattern of expression.

1110-142 Transmyocardial Gradient of Proinflammatory Cytokines in Patients With Decompensated Idiopathic Dilated Cardiomyopathy

Hyuk-Jae Chang, Joon-Han Shin, So-Yeon Choi, Gyo-Seung Hwang, Myeong-Ho Yoon, Han-Soo Kim, Seung-Jea Tahk, Hae-Sim Park, Byung-il W. Choi, *Ajou University School of Medicine, Suwon, South Korea.*

Background: Proinflammatory cytokines such as tumor necrosis factor alpha(TNFa) and interleukin-6(IL-6) have been recently identified as contributors to the syndrome of chronic heart failure. However, the origin of these cytokines in heart failure remains unclear. Therefore, we analyzed concentrations of TNFa, IL-6 and their cognitive receptors in patients with recently(<1 month) decompensated heart failure in peripheral vein(PV) and coronary sinus(CS) simultaneously.
Method: The study group included 24 patients(18 males) with mean age 59±8 years and ejection fraction of 29±16%. NYHA functional class were I: 4, II: 8, III: 2 and IV: 10 during the episode of decompensation. Blood sampling for TNFa, IL-6 and TNFa receptor(TNFaR) 1, 2 was collected from CS and PV to measure the difference as a part of