IMPAIRED RIGHT VENTRICULAR DIASTOLIC FILLING IN TETRALOGY OF FALLOT WITH MULTIPLE LEVELS OF RIGHT SIDED OBSTRUCTION.

Adel M. Zaki, Sameh Bakhoum, Wafaa El-Arousy, Fawzan Shaltout, Yahya S. Mikhael. Cardiology Department, Cairo University, Egypt.

The clinical presentation , hemodynamic and surgical results of tetralogy of Fallot (TOF), varies in relation to the sites and severity of right sided obstruction. The Doppler diastolic tricuspid flow (DDTF) was studied in 20 Pts with angiographically proven TOF. Ten Pts had only infundibular right ventricular outflow tract obstruction (group-A). The rest (group-B), had additional levels of stenosis (pulmonary valvular and/or supra-valvular). The catheter hemodynamic RVEDP was elevated, with higher value in group-B (9.6 ± 2.3, vs., 13.1 ± 3.0 mmHg, $p < 0.001$). The ratio of the late to early DDTF peak velocities (A/E) and their areas, were 0.77 ± 0.15 and 0.51 ± 0.13, in group-A, while in group-B, it were significantly higher, 1.28 ± 0.2, and 1.29 ± 0.27, $p < 0.001$.
Conclusions: These data show impaired RV diastolic function in TOF, which is more prominent with multiple levels of right side obstruction and that DDTF can forecast its severity before hemodynamic study and may explain the occasional bad surgical results.

FIBROBLAST GROWTH FACTORS ARE EXPRESSED IN CORONARY LESIONS OF PATIENTS WITH UNSTABLE ANGINA PECTORIS AND THOSE WHO HAVE POST ANGIOPLASTY RESTENOSIS

Moshe Y. Flugelman, Rosaly Correa, Zu-Xi Yu, Gad Keren, Martin B. Leon, Lowell F. Satler, Kenneth M. Kent, Ward Casscells, Stephen E. Epstein. NHLBI, Bethesda, MD

Coronary atherectomy specimens from 20 pts were analyzed by immunostaining for cellular expression of acidic and basic fibroblast growth factors (a&bFGF) in order to investigate the relevance of a&bFGF in unstable coronary syndromes and post angioplasty (PTCA) restenosis. Four pts presented with stable angina pectoris (AP), 11 with unstable AP, and 5 with post PTCA restenosis (4 days to 3 months). Specific polyclonal antibodies for a&bFGF were used for immunostaining, while normal rabbit serum and antibodies preadsorbed with a&bFGF peptides served as controls.
Results: Three pts with stable AP had no cellular a&bFGF expression in their atherectomy specimen while one pt had heterogenous cellular a&bFGF experession. Of the 11 pts with unstable AP, 3 had cells homogeneously expressing a&bFGF and 8 pts had heterogenous cellular expression of a&bFGF. Three pts with post PTCA resteosis showed prominent cellular a&bFGF expression. Another 2 pts with post PTCA restenosis had inconclusive staining due to a high background staining.
Conclusion: Cellular expression of a&bFGF commonly occurs in coronary lesions of pts with unstable AP and post PTCA restenosis. This observation supports the concept that a&bFGF may play a role, through their mitogenic effects, in the genesis or exacerbation of unstable AP and restenosis following PTCA.

IN SITU HYBRIDIZATION OF HUMAN ATHEROSCLEROTIC MATERIAL FROM CORONARY ARTERIES AND SAPHENOUS VEIN GRAFT BIOPSIED WITH AN ATHERECTOMY CATHETER.

Guy Leclerc, Lawrence Weir, Michael Simons, Robert D. Safian, Marianne Kearney, Donald S. Baim and Jeffrey M. Isner, St.Elizabeth's Hospital, Boston, MA.,and Beth Israel Hospital, Boston, MA.

The purpose of this study was: 1) to determine whether atherosclerotic (ATH) material from human coronary arteries (CA) and saphenous vein grafts (SVG) obtained percutaneously using a mechanical atherectomy device (Atherocath) could be satisfactorily analyzed at the level of gene transcription; and 2) to assess in different types of ATH lesions the expression of an isoform of a human nonmuscle myosin heavy chain (NMMHC-B) mRNA, which may have a role in cytokinesis. Twenty-four 5μm frozen sections were prepared from three fresh stenotic lesions removed by directed atherectomy in three patients with symptomatic coronary artery disease. Two lesions (16 sections) were primary coronary stenoses; one lesion (8 sections) was a SVG restenosis lesion. All twenty-four sections were examined by in situ hybridization (ISH) using a RNA antisense (A) probe specific for NMMHC-B mRNA. The specificity of the hybridization was controlled by using a noncomplementary sense (S) probe. All sections of the restenotic lesion showed strong hybridization to the A probe with clustering of ≥20 grains/cell nucleus in ≥ 90% of total cells on 100x mag fields selected at random and analyzed using a semi-quantitative scoring system. In contrast, primary plaques showed minimal hybridization (<20% of cells hybridized, at an intensity level of <20 grains/cell nucleus). Conclusions: 1) The Atherocath is a satisfactory tool for obtaining ATH material from CA and SVG that can be processed for analysis of gene transcription; 2) these preliminary findings suggest that activation of NMMHC-B may play a role in the development of restenosis lesions.

Monday, March 4, 1991
Poster Displayed: 2:00PM–5:00PM
Author Present: 3:00PM–4:00PM
Hall F, West Concourse
Pediatric Cardiology

THE NATURAL EVOLUTION OF HEART DISEASE BEFORE BIRTH: GROWTH OF THE CARDIAC CHAMBERS AND GREAT ARTERIES IN PRENATALLY DIAGNOSED CONGENITAL HEART DISEASE (CHD).

Lisa K. Hornberger, David J. Sahn, Kathryn Reed. Univ. Calif San Diego, CA; Univ. Arizona, Tucson, AZ.

To assess cardiac chamber and great artery (GA) growth in fetuses with CHD, we reviewed 64 echocardiograms from 25 serially studied fetuses with prenatally diagnosed CHD (≥17 wks gestation). (Follow-up = 5 to 17 wks; mean 9wks). Six fetuses with a hypoplastic RV (2) or LV (4) had some growth of the hypoplastic structures, which nonetheless remained <30% of the size of the unaffected ventricle and its GA, but in 3 others (2 HLV, 1 HRV) the hypoplastic structures failed to grow at all. Three fetuses with dysplastic TV and tricuspid insufficiency (1 initially with a normal, symmetric 4 chamber view) developed massive RV size with an RV to LV diameter ratio of 2/1. In 3 fetuses with PA obstruction (1 D-transposition, 2 double outlet RV [DORV]) and in 2 of the fetuses with TV dysplasia who later developed PA atresia, the PA grew but remained smaller than the AO. The PA did not grow at all in 3 fetuses with tetralogy of Fallot or in 2 others with single ventricle and sub PS. All structures grew normally in 3 fetuses with ventricular septal defects. Narrowing of the ascending AO and branch PAs was not apparent at 21 wks but obstruction progressed from 17-34 wks in a fetus with autosomal dominant supravalve AS. In 1 fetus with truncus arteriosus and truncal stenosis, the RV and LV grew equally and in proportion to the truncus. Finally in 2 fetuses (1 DORV, 1 single ventricle) with a small AO, the AO arch did not grow and coarctation anatomy became apparent in the third trimester. The variable patterns of flow or function related growth of the cardiac chambers and GAs in fetuses with CHD necessitate serial study for determining prognosis and planning postnatal management.