Electrocardiogram in Hypertrophic Cardiomyopathy Revisited: Does ECG Pattern Predict Phenotypic Expression and Left Ventricular Hypertrophy or Sudden Death?

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Background: left ECG hypertension (LHV). Electrocardiographic studies have recently shown magnitude of LHV to be directly related to prognosis, specifically as a predictor of sudden death (SD) based on maximum LV wall thickness (30 mm).

Methods: Therefore, we investigated whether ECG patterns, specifically standard and precordial leads voltages accurately reflect phenotypic expression and magnitude of LHV as well as clinical outcome in HCM. ECG voltages were compared with echocardiographic LV wall thickness and outcome is shown with consecutive HCM patients from a largely unselected regional cohort. Ages were 46 ± 23 years.

Results: Weak correlations were evident between ECG voltages and wall thickness - e.g., correlation between max. LV wall thickness and sum of voltages in all 12 leads was 0.254 (p<0.01), between max. LV wall thickness and max. voltage in any single lead 0.254 (p<0.01) and between LV wall thickness index (sum of wall thicknesses in all LV segments) and maximum voltage in any lead was 0.209 (p<0.01). Among 53 patients with extreme LVH (max. wall thickness ≥ 30 mm), only 24 (45%) had greatly increased max. voltages ≥ 30 mm in any lead. Conversely, of 153 patients with max. voltage ≥ 30 mm, just 24 (16%) had LV wall thickness ≥ 30 mm. There was no relation between basal LV outflow obstruction and ECG voltages. Of 102 patients with outflow gradient ≥ 30 mm Hg, only 43 (42%) had max. voltage ≥ 32 mm in any lead; of 346 patients with gradient < 30 mm Hg, just 13 (3%) had max. voltage > 30 mm. Of the 16 patients who died suddenly, ECGs showed a variety of abnormalities in 17, but only 5 (27%) had markedly increased voltages (max. ≥ 30 mm).

Conclusion: The 12-lead ECG shows abnormalities in most patients with HCM, often with bizarre patterns, but cannot be regarded as a reliable marker for the magnitude of LHV (even massive hypertrophy), outflow obstruction, or likelihood of sudden cardiac death.

Outcome for Children With Lymphocytic Myocarditis Embolized in the National Australian Childhood Cardiomyopathy Study

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Background: The natural history of lymphocytic myocarditis (LM) in children is uncertain. Outcomes for children with LM enrolled in the National Australian Childhood Cardiomyopathy Study (NACCS) were reviewed.

Methods: NACCS is a population-based study of all children <10yrs presenting in Australia from 1987-97 with CM. Study end-points were death or transplantation. Histology was reviewed by a single pathologist.

Results: Histology was available in 70/184 patients with DCM, from endomyocardial biopsy (EMBx) in 30 and autopsy or explantation in 31. LM was present in 25/70 (36%) children with available histology and 13/39 (33%) of those who had undergone EMBx. Of 25 children with LM, 24 presented with sudden death and another 24 died shortly after presentation. Of children with histology, the proportion of those with LM decreased with increasing time from presentation. Of 39 children with EMBx, freedom from death or cardiac transplant at 1 and 5 years after presentation was 100% for those with LM, compared to 80% and 59%, respectively, for those with non-specific histology (p<0.008).

1113/13 (85%) children with LM on EMBx received immune-modulatory therapy. The time constant of chamber stiffness (k, ml-1). RESULTS. Wigle’score was directly related to pulmonary artery wedge pressure (r=0.48, p<0.04), peak atrial pressure (r=0.43, p<0.04), LV end-diastolic pressure (r=0.42, p<0.04), and inversely related to end-diastolic volume (r=-0.49, p<0.04). There were no significant relationship between magnitude of LVH and t. CONCLUSIONS. The magnitude and distribution of LVH influences the impairment in passive LV diastolic function. In fact, Wigle’scores were associated with increased chambers stiffness and LV end-diastolic pressure. Moreover, this led to a rise in left atrial pressure and in pulmonary artery wedge pressure in HCM patients with more severe LVH.

Nitric Oxide Mediates Myocyte Apoptosis in Experimental Acute Chagasic Myocarditis

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Background: Apoptosis has been implicated in the pathogenesis of chagasic myocardiitis. Nitric oxide (NO) has been identified as a trigger for apoptosis. To evaluate the role of NO in the pathogenesis and consequences of myocyte apoptosis in acute chagasic myocarditis, we infected-inducible nitric oxide synthase knock-out (KO) and C57BL/6 129sv wild-type (WT) mice with the Trypanosoma cruzi strain. Methods. Serial transthoracic echocardiographic (TTE) studies were performed on infected animals (n=12) and their corresponding controls (n=13) to measure left ventricular (LV) end-diastolic diameter (EDD), relative wall thickness (RWT) and fractional shortening (FS). The degree of inflammation and myocyte necrosis, and apoptosis (TUNEL assay) were assessed in each at necropsy. Results. At day 19 post-infection, compared with the KO infected mice (n=6), the WT infected mice (n=6) showed larger LV EDD (3.5±0.3 vs 2.4±0.2 mm, p<0.01), lower RWT (0.4±0.5 vs 0.6±0.1, p<0.01), and lower LVFS (43±5 vs 56±4%, p=0.07). The differences in LV EDD, RWT, and SF between infected animals and their corresponding control mice were significantly greater in WT than in the KO mice: EDD (4±0.5 vs 7±1.5%, p<0.001), RWT (42±5 vs 29±1.9, p<0.001) and SF (30±5 vs 74±4%, p<0.01). At necropsy, myocardial inflammation and necrosis were more severe in WT than in KO mice. In addition, higher number of apoptotic LV myocytes were seen in infected WT than in KO mice (1.1±0.2/16 vs 0.6±0.1/00 per HPF 200X, p<0.01). Conclusion. Apoptosis is a major form of myocyte death in acute chagasic myocarditis. It contributes to the adverse functional consequences of the disease and is mediated, in part, by NO.

Risk Markers of Ventricular Arrhythmias and Fourier Phase Analysis of Radionuclide Angioscintigraphy to Evaluate Ventricular Asynchrony and Prognosis in Idiopathic Dilated Cardiomyopathy

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Radionuclide angioscintigraphy is able to evaluate the interventricular and intraventricular LV and RV asynchrony. No information is available about the relationships between LV and RV asynchrony and risk markers of ventricular arrhythmias in idiopathic dilated cardiomyopathy (IDC).

Methods. 103 patients (68% female, LVESF 27 ± 11% ) were studied. Left bundle branch block (LBBB) was present in 25% of the patients and QRS duration was 113 ± 32 ms. Equilibrium radionuclide angiography with Tc99m was performed and Fourier phase analysis were examined in both ventricles. Interventricular delay between the mean phase of RV and LV was assessed. Differences between infected and uninfected mice were evaluated using LAM (p<0.05). The differences in LV EDD, RWT, and SF between infected animals and their corresponding control mice were significantly greater in WT than in the KO mice: EDD (4±0.5 vs 7±1.5%, p<0.001), RWT (42±5 vs 29±1.9, p<0.001) and SF (30±5 vs 74±4%, p<0.01). At necropsy, myocardial inflammation and necrosis were more severe in WT than in KO mice. In addition, higher number of apoptotic LV myocytes were seen in infected WT than in KO mice (1.1±0.2/16 vs 0.6±0.1/00 per HPF 200X, p<0.01). Conclusion. Apoptosis is a major form of myocyte death in acute chagasic myocarditis. It contributes to the adverse functional consequences of the disease and is mediated, in part, by NO.

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