YOUNG INVESTIGATOR AWARD SESSION – CLINICAL SCIENCE

442

Left bundle branch block and coronary artery disease in coronary ct angiography

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Purpose: Left bundle branch block (LBBB) is considered an unfavourable prognostic marker in heart disease. Testing for coronary artery disease (CAD) is often prompted by incidental LBBB finding, but published studies disagree about an association between LBBB and CAD. Thus, we assessed the association of LBBB with CAD in patients undergoing coronary CT angiography (CCTA).

Methods: We enrolled 818 patients (106 LBBB patients and 712 controls) without known CAD, cardiomyopathy, valvular or congenital heart disease who underwent CCTA. Image quality was assessed on a 4-point scale for each coronary segment. After exclusion of non-diagnostic studies, comparison of CAD prevalence (≥50% coronary stenosis) was performed using triple case-matching for pre-test probability (based on age, gender and typicality of symptoms) in 101 LBBB patients and 303 matched controls.

Results: Mean age was 57.2 ± 11.1 years. CAD prevalence did not differ between LBBB patients and matched controls: 15% vs. 16% (p=0.86). Similarly, no significant differences were found in cardiovascular risk factors (CVRF), stenosis severity, CAD extent, non-obstructive disease and vessel-based analysis. In multivariate analysis, age, gender, typical angina and CVRF, but not LBBB (p=0.94), emerged as significant and independent predictors of obstructive CAD. Image quality was very high in LBBB and controls (3.8 versus 3.9). LBBB did not affect image quality in multivariate analysis including technical factors (p=0.70).

Conclusion: CAD prevalence is similar in LBBB patients at low-to-moderate pre-test probability compared to controls matched for age, gender, symptom typicality and CVRF. LBBB does not affect image quality in CCTA, which is therefore a useful imaging modality in LBBB patients.

444

Arhgap24, a first gene for fibro elastic deficiency mitral valve prolapse? A phenotypic study

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Purpose By contrast to Barlow-mitral valve prolapse (MVP), fibro-elastic deficiency (FED) disease is considered a pure degenerative condition. Filaggrin, encoded by ARHGP24 (Chr. 4), is involved in mechanical transaction with Filamin-A (FLNA), the first gene of MVP. We hypothesized that ARHGP24 mutations could elicit MVP.

Methods: Four probands with ARHGP24 mutations were identified among 95 MVP. By familial echocardiography screening, a total of 13 adults (>16 years old) with an ARHGP24 mutation (Mutated) were enrolled and matched with 39 healthy adults (Control). Anterior (AML) and posterior (PML) mitral leaflets, length and thickness were measured. The coaptation point position was measured by the ratio of coaptation height to the systolic annulus diameter. MVP (displacement > 2 mm above the annulus line), minimal systolic displacement (MSD, displacement < 2 mm) and abnormal antero-posterior coaptation position (AARC, ratio < 60%) were assessed. The conjunction of MSD and AARC defines a prodromal form (MVPProd).

Results: Baseline characteristics were similar in the 2 groups. We noted a frequent alteration of the mitral valve in our mutated population: 93% of our ARHGP24 population had a mitral regurgitation (versus 38% in our Control, P=0.0007), 93% had an abnormal mitral valve, 70% had a MVP and 23% a MVPProd. This phenotype is characterized by an elongation of the PML (8.2 ± 1.6 vs 6.0 ± 1.2 mm²/m2, P=0.0003), leading to an anterior displacement of the coaptation point in our mutated group (51 ± 11 vs 66 ± 7%, P=0.0003). Of interest the AML is not elongated and the leaflets are thin in the 2 groups (PML: 2.7 ± 1.8 vs 2.2 ± 0.5 mm in Control, P=0.21, AML: 2.5 ± 0.9 vs 2.1 ± 0.4 mm, P=0.25) suggesting a FED-MVP phenotype. Two probands were operated for severe MR related to chordal rupture. The aspect of FED was confirmed by surgical and histological examination with thin leaflets, excepted at the level of the flail posterior scallop (P2).

Conclusions: ARHGP24 is the first gene for autosomal dominant inherited MVP. Our limited series of patient exhibit typical features of FED-MVP. Our results could change the paradigm of a pure degenerative disease for FED-MVP.

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Abstract 442 Figure. Prevalence of CAD by severity

443

Focal myocardial fibrosis and abnormal left ventricular strain in patients with sarcoidosis without clinical evidence of cardiac disease

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Background: Cardiac involvement in systemic sarcoidosis occurs in 20-25% of patients; and is associated with poor outcome and reduced survival. The identification of cardiac sarcoidosis is challenging in asymptomatic patients using conventional methods including echocardiography. We aimed to assess the role of CMR for detecting subclinical cardiac sarcoidosis (CSc) in patients with demonstrated pulmonary sarcoidosis and without cardiac symptoms.

Methods: We included consecutive patients with pulmonary sarcoidosis referred for CMR study from a specialized sarcoidosis ambulatory clinic in a tertiary university hospital. Inclusion criterion was the diagnosis of sarcoidosis and absence of clinical signs of cardio-vascular disease. Exclusion criteria included the presence of atrial fibrillation, more than mild valvular heart disease, ischemic heart disease and general contra-indications to CMR and/or gadolinium. All patients underwent CMR at 3.0T. CMR study included cine CMR (short-axis and long-axis planes) for LV function, T2-weighted imaging, tagging, late gadolinium enhancement (LGE). Patients underwent Holter monitoring for arrhythmias.

Results: 38 patients were included, 54 ± 17 year-old, 17 male. A control group of 18 healthy individuals were assessed by tagging. ECG and conventional echocardiograms were normal in all. LV end-diastolic volume and ejection fraction were normal in all patients (72 ± 11 ml/m2 and 59 ± 6% respectively). No myocardial signal changes were found on T2-weighted imaging. Focal LGE was found in 11 patients (21%), predominantly involving the midwall and or subepicardial of the basal septum and lateral myocardial segments. Regional strain analysis obtained from tagging, patients with sarcoidosis had significantly lower LV peak longitudinal strain than the controls (-14.4 ± 1.8 versus -19.0 ± 1.1, p=0.005). In 22 patients, including 11 patients with focal LGE and other 11 without LGE, strain values were abnormal, with a mean value of -13.5 ± 2.1. Patients with LGE showed significantly more frequent ventricular arrhythmias, which were present in all patients with LGE, in comparison with the ones without LGE (p=0.02) in the Holter monitoring.

Conclusion: In patients with systemic sarcoidosis and absence of clinical cardiac involve-ment, CMR showed subclinical involvement in a substantial proportion, with focal LGE and abnormal longitudinal strain probably due to more widespread myocardial disease. LGE was associated with a more frequent occurrence of ventricular arrhythmias with potential impact on prognosis.
Advantage of using ASE/EACVI criteria for detection of subclinical cardiotoxicity in breast cancer patients undergoing anthracycline and trastuzumab therapy

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Purpose: The present study aimed at assessing the advantage of using ASE/EACVI 2D Speckle Tracking Echocardiography (STE) criteria in diagnosing subclinical cardiotoxicity (CTX) in breast cancer patients undergoing anthracycline (ANT) and trastuzumab (TRS) chemotherapy.

Methods: Sixty consecutive women (mean age = 48 years) with breast cancer were treated by sequential protocols including ANT (epirubicin, cumulative dose = 489 ± 74 mg/m²) + cyclophosphamide and/or 5-fluorouracil for 3-4 cycles, followed by 18 cycles of TRS (mean cumulative = 877.4 ± 306 mg/Kg) combined with taxanes (80 mg/m²) in the first three months. At baseline, after ANT (3 months) and during (3-6-9 months) and after TRS completion (12 months), they underwent a complete standard echocardiogram with determination of ejection fraction (EF) and 2D STE derived global longitudinal strain (GLS). The detection of subclinical CTX according to ASE/EACVI criteria (GLS drop > 15% in comparison with baseline) was applied to start cardiac therapy with beta-blockers and ACE-inhibitors. Overt CTX (defined according to 2D EF < 53%) induced the therapy interruption.

Results: Fifty out of the 60 enrolled patients (83.3%) completed the cycles of chemotherapy without showing subclinical and/or clinical CTX. 2D STE was feasible in all the patients, before, during and after any cycle of therapy. Ten patients showed subclinical CTX but, by with the help of cardiac drugs, 7 completed successfully the chemotherapy cycles. Among the 3 patients developing overt CTX during the following TRS cycles despite the use of cardiac drugs, 2 withdraw permanently whereas one could start over TRS after 3-month interruption and complete successfully. In the 58 patients finally completing ANT + TRS (96.6%), 2D EF (baseline: 63 ± 4%, ANT 62 ± 5%, TRS: 61 ± 5%) did not differ significantly but GLS was lower after both ANT (-21.7 ± 2.5%) (p < 0.02) and TRS (-20.8 ± 2.7% (p < 0.01) than at baseline (-23.0 ± 2.8%).

Conclusions: The use of ASE/EACVI criteria for detection of subclinical CTX, based on GLS monitoring, presents the important advantage of an early beginning of cardiac therapy in breast cancer patients undergoing ANT + TRS. This allows to complete successfully the chemotherapy in the majority of the patients. GLS is superior to EF in detecting cardiotoxicity.