GLS (HR 1.4 95% CI 1.1-1.6, p<.001) were significantly predictive of the primary endpoint, only LV GLS remained independently associated on multivariate analysis (HR 1.3, 95% CI 1.0-1.7, p=0.02). Those with LV GLS >-17.3% (median) were significantly more likely to be free of the primary event at follow-up compared to those with LV GLS >=-17.3% (log rank p=0.02).

Conclusions: In ECS patients, LV GLS is reduced suggesting subclinical cardiac dysfunction, despite absence of clinical or conventional echocardiographic evidence of cardiac disease. Furthermore, LV GLS is independently associated with the occurrence of adverse events and/or future development of accepted evidence for CS.

P2972 | BEDSIDE
Apical hypertrophic cardiomyopathy: chest pain and myocardial perfusion defects result from regional diastolic persistence of hyperdynamic cardiac contractility
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Background: Apical hypertrophic cardiomyopathy (HCM) is commonly associated with drug-refractory chest pain. We sought to determine whether, in apical HCM, coronary perfusion time is abbreviated by the diastolic persistence of contractility into diastole

Methods: 62 apical HCM patients had cardiac magnetic resonance (CMR) scans assessed for stress perfusion (myocardial perfusion reserve index (MPRI)), late gadolinium enhancement (LGE; % of myocardial volume) left ventricular (LV) volumes and LV contractile persistency (% total cardiac cycle) at the LV apex and base. Radial and circumferential strain were assessed. Patients were divided into three groups on the basis of severity of contractile persistency. The interval between earliest and latest systolic peaks was measured from strain data from each of the apical segments.

Results: Compared to subjects with the least contractile persistency (C1), those in the most (C3) were more likely to have chest pain (94% vs 63%, p<0.05) and lower MPRI (0.90±0.24 vs 1.43±0.50, p<0.05). Multiple regression analyses included contractile persistency, LVH, %LGE, age and gender. Contractile persistency was independently associated with chest pain (0.4 per 10% cardiac cycle, CI 95%: 0.1 to 0.8, p<0.05) and a reduction in apical MPRI (p<0.10 per 10% cardiac cycle, CI 95%: -0.04 to -0.15, p<0.01). There were striking differences in systolic strain between C1 and C3. First, radial strain was almost absent in C3, with only post-systolic contraction detected. Second, temporal dispersion in circumferential strain was greater in C3 than C1 (230±101ms vs 114±44ms, p<0.05). Using the convention >130ms as a threshold, circumferential dysynchrony was present in 25% of C1 and 81% of C3 patients (p<0.001) and radial dysynchrony in 65% of C1 and 95% of C3 patients (p<0.05). In patients with radial dysynchrony, the earliest peak was most often in the inferior or anterior segments (60%) and the latest in the lateral segment (33%). In patients with circumferential dysynchrony, the earliest peak was most often in the inferior or anterior segments (59%) and the latest in the lateral segment (41%).

Conclusion: In apical HCM, regional persistency of contractility into diastole causes myocardial ischaemia and chest pain. This is the first description of contractile persistency and dysynchrony as a mechanism for myocardial perfusion abnormalities and presents novel therapeutic opportunities for drug-refractory chest pain in apical HCM.

P2973 | BENCH
Characterization of myocardiopathy deformation in hypertrophic cardiomyopathy using speckle tracking: comparison with physiological hypertrophy
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Background: In young competitive athletes, the differential diagnosis between apical hypertrophic cardiomyopathy (HCM) and nonpathological changes in cardiac morphology associated with training (commonly referred to as ‘athlete’s heart’) and certain cardiac diseases with potential for sudden death as Hyperthrophic Cardiomyopathy (HCM), is an important & not uncommon clinical problem.

Objective: In young competitive athletes, the differential diagnosis between apical hypertrophic cardiomyopathy and physiological hypertrophy using speckle tracking: comparison with physiological hypertrophy. The aim was to determine the association between cardiopulmonary exercise test (CPET), non-invasively-determined hemodynamic variables, and clinical and echocardiographic variables and outcomes in patients with HCM.

Methods: The study comprised 22 patients with HCM. 81% had asymmetric septal hypertrophy, 34 athletes with septal thickness >12 mm & 28 age matched healthy subjects as a control group. Apical four chamber view was displayed; speckle tracking was used to measure longitudinal peak systolic strain (syst), peak systolic strain rate (SRsys), time to peak (t) [TPP], post systolic strain (pSS) and intra-ventricular systolic delay (Intra-V delay). These parameters were quantified in 3 segments (C1, C2, C3) of apical LV segments.

Results: Regional Myocardial deformation of LV segments was significantly reduced in comparison to corresponding segments in athletes & control (P<0.001). The systolic function of the basal (48.86±4.74, ±74.40±0.41 sec) and mid (4.3.6±6.9%, -0.57±0.48 sec) segments were significantly lower than apical septal (21.6±8.2%, -1.6±2.04 sec) & all lateral segments (-14.4±6.9%, -1.12±0.44 sec), -11.7±4.8%, -0.77±0.42 sec, -16.7±7.3%, -0.8±0.34 sec respectively, P<0.001 in HCM patients, while myocardial deformation was normal & was homogeneous in athletes. The later showed homogeneous systolic activation of the ventricular walls. Conversely, HCM group showed significant increase of the interval between earliest and latest systolic peaks which is more prominent in septal lateral wall (P<0.001). While averaged s was negatively correlated to NYHA functional class and E/Em ratio (r=-0.5, n=0.46 respectively, P<0.01), intraventricular delay showed direct relationship to LVM (r=0.63, P<0.01). A cut-off value of basal and mid septum <13%, <12% differentiated between HCM and athletes with 91, 95% sensitivity and 91, 100% specificity respectively.

Conclusion: The non-uniform distribution & magnitude of LV hypertrophy in HCM, is associated with disorganized contraction & regional heterogeneity of myocardial systolic function. Deformation analysis using speckle tracking is a novel ultrasonic technique that helps to differentiate mechanical dysfunction in HCM from myocardial adaptations in physiologic hypertrophy.

P2974 | BEDSIDE
Two-dimensional speckle tracking echocardiography in early detection and prediction of cardiotoxicity during epirubicine based chemotherapy
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Objective: To investigate whether alterations of myocardial strain and high sensitive cardiac Troponin T (cTnT) could predict future cardiac dysfunction in patients treated with epirubicine exposure.

Methods: Sixty-five patients aged 52.46±13.58 years with newly-diagnosed large B-cell non-Hodgkin lymphoma treated with epirubicin were studied. Blood collection and echocardiography were performed at baseline, 1 day after the third cycle, and 1 day after chemotherapy completion. After 4-6 months of chemotherapy, patients were studied using echocardiography. cTnT was detected with a highly sensitive assay. Longitudinal (LS), Circumferential (CS) and Radial Strain (RS) were calculated using two-dimensional speckle tracking echocardiography. Left Ventricular Ejection Fraction (LVEF) was analyzed by real-time 3D echocardiography. Cardiotoxicity was defined as a reduction of the Left Ventricular Ejection Fraction (LVEF) <5% to <50% or the appearance of symptoms of heart failure or an asymptomatic reduction of the LVEF >10% to <55%.

Results: LVEF remained stable and within normal limits in the whole course of chemotherapy, however decreased from 65.13±3.65% at baseline to 60.58±5.6% following up (p=0.002). Twelve patients (18.46%) developed cardiotoxicity 4-6 months after treatment. Global LS (-18.56±1.69%) vs. -15.79±1.53%), CS (-20.88±2.67%) vs. -19.23±2.31), RS (39.32±6.36% vs. 34.78±6.15%) were markedly reduced and cTnT elevated from 0.010±0.002ng/ml to 0.0072±0.0035ng/ml (p all<0.01) at the completion of chemotherapy compared with baseline values. A >15% decrease in longitudinal strain (sensitivity: 86%, specificity: 75%) and a >0.004ng/ml elevation in cTnT levels (sensitivity: 79%, specificity: 64%) from baseline to the third cycle of chemotherapy predicted later cardiotoxicity.

Conclusions: Longitudinal strain combined with high sensitive cTnT may provide a reliable and non-invasive method to predict cardiac dysfunction in patients receiving anthracycline-based chemotherapy.
Cardiomyopathies: from pathogenesis to treatment

P2976 | BEDSIDE
Diffuse and regional patterns of myocardial fibrosis in hypertrophic cardiomyopathy occur independently of each other and have distinct clinical associations

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Purpose: Previously, in hypertrophic cardiomyopathy (HCM), only dense regional fibrosis could be identified by speckle magnetic resonance (SMR) imaging. A novel myocardial fibrosis scoring method (MFSEM) with late gadolinium enhancement (LGE) sequences. Recently, post-contrast T1 mapping has also enabled quantification of diffuse myocardial fibrosis. We sought to explore significant between both patterns of myocardial fibrosis and several clinical parameters in a typical cohort of patients with HCM.

Methods: We performed contrast-enhanced CMR on 123 patients (67 male, mean age 51 years) with documented HCM. Regional myocardial fibrosis was evaluated by LGE sequences, and diffuse fibrosis by post-contrast T1 mapping. Correlations between both patterns of fibrosis and patients’ clinical characteristics, CMR-derived LV volumetric data and echocardiographic findings were investigated with univariate and multivariate analysis.

Results: LGE consistent with HCM was observed in 88% of patients with a mean quantity of 4.6±6.3% of total LV mass, and mean-post contrast myocardial T1 time was 566±81 ms. LGE extent correlated with LV ejection fraction (r = -0.48, p < 0.01) whereas T1 time did not. Conversely, T1 time correlated with E/E’ (r = -0.46, p < 0.01) whereas LGE extent did not. Patients with LV outflow tract obstruction had less LGE (3.1±5.6% vs. 6.9±7.5%, p < 0.05), but similar T1 times. Patients with exertional dyspnoea had shorter T1 times (485±64 ms vs. 521±71 ms, p < 0.05), but similar LGE extent. No relationship was observed between LGE and post-contrast T1 time.

Conclusions: In HCM, regional and diffuse patterns of myocardial fibrosis occur independently of each other and exhibit distinct clinical associations. Regional fibrosis is associated with reduced LV systolic function and the absence of LV outflow tract obstruction, whereas diffuse fibrosis is associated with higher LV filling pressures and the presence of exertional dyspnoea.

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Diffuse myocardial fibrosis in Alstrom syndrome: an early marker of disease progression

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Purpose: Alström syndrome (ALMS) is a rare autosomal recessive genetic disorder with progressive multisystem involvement. Idiopathic dilated cardiomyopathy (ICM) occurs in approximately 45% of patients with high rates of recurrence in adolescence. Myocardial fibrosis is present on post-mortem and on MRI as patchy diffuse late gadolinium enhancement (LE) in an older cohort of ALMS patients. We hypothesise that subclinical diffuse fibrosis in young patients with ALMS precedes any change in conventional parameters of ventricular function or overt scarring on LE.

Methods: Seventeen patients with ALMS (mean age 31 years (9), 64% male, 24 hour ABPM 134 (162)±80 (mmHg)) were compared to gender and age matched newly diagnosed borderline hypertensive patients (24 hour ABPM 137 (9) (7 mmHg)) and healthy volunteers. All subjects underwent cardiac MRI (1.5T). Myocardial extracellular volume (ECV) was assessed using T1-mapping pre and (7) mmHg) and healthy volunteers. All subjects underwent cardiac MRI (1.5T). Myocardial extracellular volume (ECV) was assessed using T1-mapping pre and (7) mmHg) and healthy volunteers. All subjects underwent cardiac MRI (1.5T). Myocardial extracellular volume (ECV) was assessed using T1-mapping pre and post contrast. The American College of Cardiology/European Society of Cardiology (ACC/ESC) guidelines for the diagnosis and management of cardiovascular disease in the setting of non-ischaemic cardiomyopathy are used.

Results: Overall females had significantly increased septal myocardial ECV compared with males (0.30 (0.03) vs. 0.26 (0.03), p < 0.01). Septal myocardial ECV was higher in ALMS than hypertensive and controls (0.29±0.04 vs. 0.26±0.02 vs. 0.25±0.02, p < 0.05) and was correlated with LV EF (r = -0.64, p < 0.05). Four ALMS patients without a history of infarct CM had patchy diffuse LE in non coronary artery territories with an increased ECV compared to remote “normal” myocardium (ECV 0.40 (0.08) vs. 0.27 (0.02), p < 0.05). The mean LV mass index (LVMI) in ALMS and hypertension was increased compared to controls (151±21 (g/m²) vs. 116±15 (g/m²), p < 0.005) and MAPSE was reduced (12 (2) vs. 12 (3) vs. 16 (3), p < 0.05). There were no differences in LV ejection fraction or LA volumes. Increased septal myocardial ECV was inversely corre-