ABSTRACTS OF THE 2015 PARIS ECHO CONGRESS

Session 1 – Cardiomyopathies, Heart Failure, Athletes, Hypertension

Thursday May 28 – 10.00 – 11.00

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Can multilayer speckle imaging help in the aetiological diagnosis of LVH?
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Purpose Aetiological diagnosis of LV hypertrophy (LVH) by standard echocardiography is sometimes difficult and the analysis of strain in different myocardial layers using 2D speckle imaging may provide valuable diagnostic information.

Methods Measurements of subendocardial (SEndo), subepicardial (SEpi) and global (G) longitudinal strain (LS) (%) were performed (GE EchoPac) from the 3 apical views recorded in 24 normal pts (Nl), 19 pts with cardiac amyloidosis (CA) and 23 pts with hypertrophic cardiomyopathy (HCM) with preserved LV ejection fraction (ED septal thickness CA 18±3mm vs HCM 21±5mm, ns).

Results A transmural gradient exists in LS with significantly higher values in the subendocardial layer in all pts, but this gradient is significantly less pronounced among CA pts.

All strain values were significantly different by Anova analysis between the 3 groups. Subanalysis by Bonferroni test showed significantly lower values of all strain measurements in pts with CA as compared to both Nl and HCM pts. However, this trend toward lower strain values was not significant between HCM and Nl pts for SendoLS and GLS. Analysis of ROC curves among pts with LVH showed that SendoLS, SEpiLS, GLS and transmural gradient were all good predictors of CA (Areas under Curves respectively 0.84, 0.86, 0.85, 0.78, all p<0.001).

Conclusion Analysis of multilayer longitudinal deformation by 2D speckle imaging confirms the presence of a significant transmural gradient of strain values, significantly less pronounced in CA pts than in Nl and HCM pts. As compared to Nl pts, the decrease of strain values is more severe in CA pts than in HCM pts, despite a non-significant trend toward lower value of septal thickness in CA. Furthermore, global subendocardial LS seems relatively preserved in HCM pts as compared to Nl pts. These findings, along with further analysis of segmental multilayer deformation, are probably interesting adjuncts in the process of the aetiological diagnosis of LVH

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Borderline hypertrophic cardiomyopathy or athlete’s heart: what is the role for imaging and genetic testing in athletes?
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Background Intensive exercise is associated with a modest increase in left ventricular wall thickness (LVWT). In some cases, a doubt may occur between an athlete’s heart and hypertrophic cardiomyopathy (HCM). The aim of the study was to assess the accuracy and role of imaging and genetic testing to distinguish athlete’s heart from borderline HCM and make a decision regarding sport/professional activity.

Methods We studied consecutive athletes (intensive sport >10 hours/weeks; LVWT between 12 to 16mm) with a suspicion of borderline HCM according to the clinical status and cardiac examinations. We studied the accuracy of local cardiac diagnostic assessment versus expert referral center (cardiac assessment blinded to the results of local assessment) versus results of genetic testing (analysis of the 5 main sarcomeric genes). All cardiac local and expert assessments were performed according to ECG, echocardiography, exercise test and CMR (when available) and were blinded to genetic results.

Results 37 athletes (35 men) were enrolled, mean age: 28±12 years, mean LVWT was 13.6±1.2mm. A causative mutation was identified in 27% (10/37) of athletes: among these 10 subjects, 8 had a cardiac diagnosis of suspected HCM and 2 had a suspected diagnosis of athlete’s heart. Genetic testing rectified cardiac assessment in 5.4% of our population. Local cardiac evaluation suspected HCM in 70% (n=26, only 31% with a mutation) versus 46% (n=17, 47% with a mutation) in expert center (p=0.03). In subjects without identified genetic mutation, HCM was suspected in 67% of cases in local center versus 33% in expert center (p=0.01).

Conclusions In borderline HCM/athlete’s heart, imaging should be associated with genetic testing. Genetic testing was able to confirm the diagnosis of HCM in 27%, including the rectification of imaging assessment in 5.4%. Our results suggest that genetic testing may have a role in athletes with a suspicion of borderline HCM.