



Non Invasive Imaging (Echocardiography, Nuclear, PET, MR and CT)

CARDIAC MAGNETIC RESONANCE EVIDENCE OF MYOCARDIAL DIFFUSE FIBROSIS IN PATIENTS WITH MITRAL VALVE PROLAPSE

Poster Contributions

Poster Hall B1

Sunday, March 15, 2015, 9:45 a.m.-10:30 a.m.

Session Title: Parametric Mapping of the Myocardium by CMR

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Background: Quantitative T1 mapping shows promise for the detection of diffuse myocardial fibrosis. Mitral valve prolapse (MVP) is a common valvulopathy with known arrhythmic complications. Whereas papillary muscle fibrosis has been described in MVP, diffuse myocardial fibrosis and its association with complex ventricular arrhythmia (ComVA) in MVP are yet to be investigated.

Methods: A retrospective analysis was performed on 41 consecutive MVP patients referred for CMR between 2006-2011 and 31 age/gender matched controls free of significant cardiac disease. ComVA was defined as Grade III or higher by the Lown and Wolf classification on Holter/event monitor in a subset of MVP patients. CMR images were acquired using a Philips Achieva 1.5 T CMR scanner. Left ventricular (LV) septal T1 times were derived from Look-Locker sequences after administration of 0.2 mmol/kg gadopentetate dimeglumine. Mitral regurgitation (MR) fraction (MRF) was quantified by velocity encoded CMR.

Results: MVP patients had significantly shorter post-contrast T1 times when compared to controls (334 ± 52 vs. 363 ± 58 ms; $p = 0.03$) despite similar LV ejection fraction (61 ± 3 vs. 60 ± 6 %, $p = 0.84$). MVP patients had greater MRF compared to controls (21 ± 24 vs. 0 %, $p < 0.05$). There was no significant difference in post-contrast T1 times between MVP subjects with \leq mild MR (MRF 0-15%) and $>$ mild MR (MRF > 16 %) (347 vs. 334 ms; $p = 0.59$). Among MVP patients with available arrhythmia data ($n = 19$), those with ComVA ($n=12$) had even shorter post-contrast T1 times when compared to controls (322 ± 42 vs. 363 ± 58 ms; $p = 0.016$). In the MVP ComVA subgroup, the majority (11/12 or 91%) had T1 times ≤ 350 ms, but only 5/12 or 41% had papillary muscle LGE, $p = 0.009$.

Conclusion: MVP is associated with reduced post-contrast T1 times despite preserved LV systolic function, suggestive of subclinical diffuse myocardial fibrosis. MR alone is less likely to be the primary contributor to diffuse LV interstitial derangement in MVP. ComVA is observed in MVP patients with reduced post-contrast T1 times even in the absence of papillary muscle LGE. Further studies are needed to clarify if ventricular arrhythmias in MVP are secondary to diffuse rather than localized fibrosis.