Contrast-enhanced Look-Locker and Delayed-enhancement MRI in Patients with Apical Hypertrophic Cardiomyopathy: Distribution of Myocardial Damages and Its Association with Risk Factors and Cardiac Function

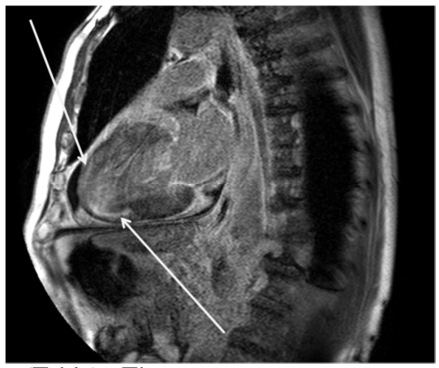
Yasuo Amano[1], Masaki Tachi[1], Hitomi Tani[1], Shinichiro Kumita[1], and Makoto Obara[2]

[1]Nippon Medical School, Tokyo, Tokyo, Japan, [2]Philips Healthcare Asia Pacific, Tokyo, Tokyo, Japan

Target Audience: radiologists, cardiologists, physicists who are interested in cardiomyopathy

Purpose: Apical hypertrophic cardiomyopathy (APH) is a unique phenotype of hypertrophic cardiomyopathy (HCM), which is characterized by its localized myocardial hypertrophy of the left ventricular (LV) apex and a "spade-like" configuration of the LV cavity. One-third of the patients with APH present with ventricular tachyarrhythmia and other serious complications. Myocardial fibrosis can be quantified using measurement of myocardial T1 value, and myocardial scarring is identified using delayed-enhancement MRI (DE-MRI). We sought to determine whether these myocardial damages detected by either contrast-enhanced Look-Locker MRI or DE-MRI is related to risk factors and cardiac dysfunction in APH.

Methods: Twenty-one patients with APH were examined using a 1.5 T or 3.0 T imager. Six patients had the traditional risk factors, such as ventricular tachycardia (n = 4) and family history of sudden cardiac death (n = 2). We evaluated myocardial hypertrophy using cine MRI, measured myocardial T1 value using contrast-enhanced Look-Locker MRI, and assessed myocardial scarring on DE-MRI. We compared the myocardial damages evaluated by either Look-Locker or DE-MRI with the risk factors or cardiac function.



Results: Myocardial scarring on DE-MRI was present in 15 (71.4%) of the 21 patients with APH (scar mass % = 2.7 ± 3.7%; 0 – 13.1%). Myocardial hypertrophy and scarring distributed dominantly in the apical myocardium ($P < 0.01$; Table). Mass percentage of myocardial scarring inversely correlated with LV ejection fraction and was related to a combination of traditional risk factors and family history of HCM ($P < 0.05$; Fig. 1). The mass percentage of scarring significantly differed between the APH patients with and without the traditional risk factors plus family history of HCM ($P < 0.05$). Myocardial T1 value did not differ between apical, midventricular and basal myocardium (Table). There was no association between myocardial T1 value and the risk factors or cardiac function in the APH patients.

Discussion: The apical myocardium showed hypertrophy and scarring in APH particularly. Ischemia of the hypertrophied myocardium and increase in the intracavitary pressure in the LV apex may lead to subendocardial scarring of the apical myocardium. The myocardial scarring was related to reduction of LV ejection fraction and clinical risk factors. Myocardial T1 values measured by contrast-enhanced Look-Locker MRI may reflect diffuse myocardial fibrosis. In APH, however, there were no significant differences in the myocardial T1 value between the non-hypertrophied and hypertrophied myocardium. This result indicates that the modest myocardial fibrosis may occur even in the non-hypertrophied LV myocardium in APH.

Conclusion: The mass percentage of myocardial scarring is associated with the decrease in LV ejection fraction and traditional risk factors plus family history of HCM in APH, but we do not have to quantify myocardial T1 value for the risk stratification of APH.

References: 1. Moon JCC. Heart 2004; 90: 645-9. 2. Iles L. JACC 2008; 52: 1574-80 3. Amano Y. Acta Radiol 2011; 52: 613-8.

Table	**basal**	**mid**	**apical**
wall thickness (mm)	11.7 ±2.8	15.1 ± 2.8	18.0 ± 3.7*
scarred AHA segments	1/126	6/126	24/84*
myocardial T1 value (ms)			
3.0 T	490.1 ± 69.2	467.8 ± 59.1	471.3 ± 67.3
1.5 T	339.1± 21.9	340.3 ± 33.3	333.7 ± 33.2

* significantly greater