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Meeting abstract

406 CMR assessment of the mitral valve: technical considerations

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Introduction

CMR is increasingly used in the assessment of mitral valve disease, especially mitral regurgitation. Although quantification of the severity of mitral regurgitation using CMR is well established, assessment of the mechanism of mitral regurgitation by CMR i.e. mitral valve leaflet anatomy and dysfunction is often suboptimal. Additional imaging planes to the standard 2-chamber, 4-chamber and LVOT cines are necessary in order to visualise each component of the mitral valve leaflet (A1–A3, P1–P3) and determine valve dysfunction such as prolapse or restriction. In addition, assessment of regional and global left ventricular (LV) function, contractile reserve and viability is necessary in functional mitral regurgitation, e.g. secondary to ischemic heart disease. We describe our technique for assessing the mitral valve using CMR and illustrate it with case examples of degenerative, ischemic and rheumatic mitral valve disease.

Methods

Following standard imaging sequences, including cines of 2-chamber, 4-chamber, LVOT, and a short axis stack, the mitral valve is thoroughly imaged. A basal short axis slice is selected which shows the mitral valve. From this, a stack of oblique slices all parallel to the LVOT long axis plane are taken of the mitral valve starting from the inferior commissure and at intervals of 5 mm up to the superior commissure. A pair of further slices are taken at each commissure perpendicular to the plane of coaptation of the leaflets. From this, each component of the mitral valve (A1–A3, P1–P3) is identified and any dysfunction defined. We use SSFP end-expiratory breath hold cines, retrospective ECG gating, high temporal resolution, repetition time 1.86 ms, echo time 1.13 ms, in plane pixel size 1,7 × 1.7 mm, flip angle 80 degrees, acquisition time 16 heart beats, slice thickness 5 mm. Velocity mapping of aortic flow is next performed to allow quantification of mitral regurgitation. In patients with ischemic mitral regurgitation, we further assess regional LV viability and contractile reserve using low dose dobutamine stress at 5 and 10 mcg/kg/min followed by gadolinium.

Conclusion

Using this examination technique, each component of the mitral valve (A1–A3, P1–P3) is clearly identified and any dysfunction determined. The severity of mitral regurgitation is quantified. Regional and global LV function and viability is defined. CMR is able to provide a detailed and comprehensive assessment of the mitral valve leaflets and dysfunction and of LV size, function, and viability in a single examination. When used optimally, CMR may complement existing imaging modalities such as echocardiography in the assessment of mitral valve disease.