Systolic Anterior Motion of the Mitral Valve

Christopher M. Walker, MD, Gautham P. Reddy, MD, Tan-Lucien H. Mohammed, MD, and Jonathan H. Chung, MD

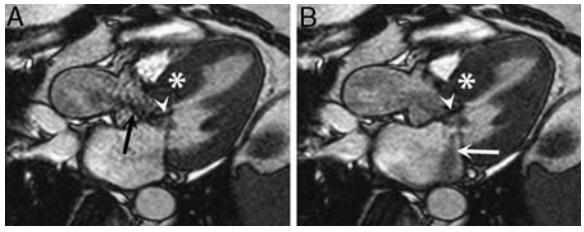

FIGURE 1. Cine steady-state free precession LVOT views in early (A) and late (B) systole show asymmetric septal hypertrophy (asterisk) and a spin dephasing flow void artifact (black arrow) indicating subaortic stenosis secondary to septal hypertrophy and systolic anterior motion of the mitral valve (arrowheads). Note associated mitral regurgitation (white arrow).

Systolic anterior motion

Paradoxical motion of the anterior mitral leaflet towards the LVOT during systole

Differential diagnosis

Hypertrophic cardiomyopathy
Hypertension
Diabetes mellitus
Acute myocardial infarction
Mitral valve repair or dysfunction
D-transposition

Appearance: Systolic anterior motion of the mitral valve (SAM) is a paradoxical motion of the anterior, and occasionally posterior, mitral valve leaflet towards the left ventricular outflow tract (LVOT) during systole (Fig. 1 and see Videos, Supplemental Digital Content 1 and 2, which demonstrate characteristic SAM with subaortic stenosis and mitral regurgitation on cine steady state free precession (SSFP) LVOT images; http://links.lww.com/JTI/A26 and http://links.lww.com/JTI/A27).

Explanation: The exact mechanism of SAM in hypertrophic cardiomyopathy (HCM) is debated, with some investigators postulating that it is a Venturi effect,[1–3] (all references cited in this article can be found at http://links.lww.com/JTI/A29) while others believe that it is secondary to anatomical differences in the positions of the papillary muscles and valve leaflets.[4] According to the Venturi effect, fluid pressure decreases and velocity increases as fluid flows through a region of reduced cross sectional area. Septal hypertrophy in HCM creates the Venturi effect through reduction in the LVOT diameter, which leads to an increased velocity and reduced pressure of the ejected blood in the LVOT. This pressure differential between the left atrium and the outflow tract is thought to lead to deviation of the mitral valve towards the septum.[3] SAM may alternatively be caused by the more anterior and inward location of the papillary muscles which alters chordal tension, resulting in a push of the leaflets towards the ventricular septum at the beginning of systole.[5] The pathogenesis of SAM is likely a combination of these two mechanisms and varies depending on the underlying associated condition.[4–6]

Discussion: SAM is most commonly seen in the asymmetric septal form of HCM but has also been described in hypertensive heart disease, diabetes mellitus, acute myocardial infarction, after mitral valve repair, and even in asymptomatic patients during pharmacologic stress with dobutamine.[4] Approximately 25% of patients with HCM have dynamic subaortic outflow obstruction occurring at rest caused by SAM contacting the hypertrophied interventricular septum.[7] Other individuals experience inducible outflow obstruction during pharmacologic stress or exercise.[1] SAM is frequently associated with mitral regurgitation due to incomplete mitral leaflet apposition during systole.[3] Cine MRI is ideally suited to study SAM and its overall effect on the left ventricle. Significant LVOT obstruction is defined by a pressure gradient greater than or equal to 30 mm Hg.[7] Velocity encoded cine (VENC) phase contrast MRI can be used to calculate the LVOT pressure gradient. Phase contrast MRI is performed in the LVOT plane, but is currently limited in evaluation of the pressure gradient due to the lower temporal resolution of the sequence compared with echocardiography. Current research is focused on developing techniques to improve existing temporal resolution as well as in developing new techniques (eg, real-time Fourier velocity encoding).[1] Although cardiac CT can also show the morphological manifestations of SAM in HCM, it cannot measure pressure gradients. It is important to determine which patients have LVOT obstruction, as they are treated more aggressively. Current therapies aim to reduce the risk of congestive heart failure through reduction of the LVOT pressure gradient. The hallmarks of therapy are medications (eg, β-blockers and calcium channel blockers), septal myectomy, and transcatheter alcohol ablation of the interventricular septum.[7,8] The effectiveness of alcohol ablation is evaluated with delayed gadolinium hyperenhancement and VENC phase contrast sequences.[9] Reduced septal thickness, resolution of SAM, and reduced pressure gradient across the LVOT indicate a response to treatment. It is important to consider a diagnosis of HCM when SAM is identified in conjunction with asymmetric septal hypertrophy and mitral regurgitation.

No financial disclosures.
The authors declare no conflicts of interest.
Supplemental Digital Content is available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's Website, www.thoracicimaging.com.