Cardiac imaging is of special value in the diagnosis and management of hypertrophic obstructive cardiomyopathy. Echocardiography and magnetic resonance imaging, in particular, have been used to diagnose the disorder, to elucidate its pathophysiology, and to guide and document the results of treatment.

Case Presentation

A 41-year-old woman was evaluated for treatment of symptomatic hypertrophic obstructive cardiomyopathy. Because of a strong family history of hypertrophic and dilated cardiomyopathies, in some cases associated with sudden death, an automatic implantable cardioverter defibrillator was implanted. Despite treatment with metoprolol succinate, verapamil, and finally metoprolol plus diltiazem, she had New York Heart Association Class III dyspnea and fatigue; symptoms were worse after a meal. Echocardiography showed asymmetrical septal hypertrophy, systolic anterior motion of the mitral valve, and moderate tricuspid and mitral valves regurgitation (right ventricular systolic pressure 50 to 60 mm Hg) (online-only Data Supplement Movies I to III).

A multidetector computerized tomography (MDCT) study of the heart and coronary arteries was done to delineate 1) the degree and exact location of interventricular septal hypertrophy; 2) the presence and degree of systolic anterior motion of the mitral valve, and moderate tricuspid and mitral valves regurgitation (right ventricular systolic pressure 50 to 60 mm Hg) (online-only Data Supplement Movies I to III).

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Electrocardiogram (ECG)-gated contrast enhanced MDCT scan was performed using a Siemens SOMATOM Definition AS 128-slice computerized tomography scanner (Siemens Medical Solutions, Forchheim, Germany). Imaging was performed from the ascending thoracic aorta to the level of the upper abdomen during a 6-second breath hold at end inspiration. We used a triphasic IV regimen: 60 mL of iodinated contrast (Isovue [Iopamidol] 370 mg I/mL, Bracco Diagnostics, Princeton, NJ) followed by 40 mL of 50/50% contrast/saline mixture and finally a flush of 50 mL of normal saline. The injection rate was 5 mL/s for all phases. For timing purposes, an automated bolus tracking software was employed, which started the scan automatically 6 seconds after contrast density in the ascending aorta reached a predefined threshold of 120 HU. The following acquisition parameters were employed: ECG pulsing, gantry rotation time of 300 milliseconds; 100 kV, 190 ref mAs, collimation 2×64×0.6 mm. The calculated radiation dose for this examination was 4 mSv.

ECG-gated MDCT depicted marked asymmetrical thickening of the basal portion of the interventricular septum up to 2.5 cm during diastole (Figure 1A). By reconstructing the volumetric data set every 5% of the cardiac cycle, we were able to document a continued anterior mitral valve leaflet-septal contact over 7 consecutive phases throughout mid and late systole. Because the heart rate during imaging acquisition was 65 beats per minute, the total duration of anterior mitral valve leaflet-septal contact was 32 ms and lasted over 35% of the cardiac cycle, indicating significant dynamic subaortic left ventricular outflow tract obstruction (Figure 1B and online-only Data Supplement Movie IV). MDCT also showed near complete obliteration of the left ventricular lumen in systole, with a measured ejection fraction of 73%. Three septal arterial branches, measuring approximately 1 mm in diameter, were delineated originating from the proximal anterior descending coronary artery (Figure 2A).

After discussing treatment options including medical therapy, myectomy, and percutaneous transcoronary alcohol septal ablation (PTASA), the patient preferred PTASA, partially because of religious beliefs prohibiting transfusion of blood products. Left heart catheterization was done with a Langston dual-lumen pigtail catheter (Vascular Solutions, Minneapolis, MN). At rest, there was a left ventricular outflow tract systolic gradient of 88 mm Hg (LV 192/21; Aorta 104/64). There was pulmonary arterial hypertension (right ventricular systolic pressure 63 mm Hg). Coronary arteriography confirmed 3 proximal septal branches of the left anterior descending coronary artery (Figure 2B and online-only Data Supplement Movie V).
The distributions of these branches were mapped with transthoracic echocardiography during injection of Definity (Bristol-Myers Squibb Medical Imaging, Billerica, MA) contrast through a subselective and occlusive angioplasty balloon (Maverick 1.5x9 mm, Boston Scientific, Natick, MA) (online-only Data Supplement Movie VI). Selective alcohol ablation was done in the 2nd and 3rd branches, using 1cc desiccated ethanol in each branch, and guided by real-time 2-dimensional echocardiography to the enhancement saturation of each vascular bed. On repeat left heart catheterization, the left ventricular outflow tract systolic gradient was 4 mm Hg.

Five hours following PTASA, a nonenhanced, prospectively triggered diastolic sequential MDCT scan was performed with the following acquisition parameters: gantry rotation time of 300 milliseconds; 100 kV, 205 ref mAs, collimation 128x0.6 mm and the center of the triggering window set at 70% of the cardiac cycle (R-R interval). The calculated radiation dose for this examination was 2 mSv. There was a hyperdense ablation lesion centered in the basal aspect of the hypertrophied interventricular septum. Its mean density was 96 HU in comparison to 46 HU of the posterior basal myocardium (Figures 3B and 4B).

Comparing equivalent mid diastolic MDCT derived multiplanar reformats of the interventricular septum in 3 chamber and short axis views prior to and following the PTASA, we were able to allocate the hyperdense ablation lesion to the point of maximal thickening of the basal interventricular septum, suggesting optimal localization of the PTASA (Figures 3 and 4).

Discussion
This report documents the use of ECG-gated MDCT in noninvasive dynamic evaluation of hypertrophic obstructive cardiomyopathy patient prior and following PTASA. As noted, ECG-gated MDCT offers an alternative to and magnetic resonance imaging in assessment of HCOM by enabling adequate depiction of the degree and exact location of interventricular septal hypertrophy, systolic anterior motion of the mitral valve, and mitral valve leaflet-septal contact. It also provides accurate delineation of the septal arterial anatomy, which is of paramount importance in guiding therapy before PTASA. Furthermore, in contrast to and magnetic resonance imaging,

Figure 1. ECG-gated MDCT 3-chamber view of the heart at end diastole (A) and at peak systole (B). Note the asymmetrical thickening of the basal portion of the interventricular septum up to 2.5 cm during diastole (*). Also note the anterior mitral valve leaflet (black arrows) that demonstrates systolic anterior motion with mitral valve leaflet-septal contact (white arrow).

Figure 2. Diastolic ECG-gated MDCT 2-chamber view of the heart at the level of the interventricular septum (A) and equivalent right anterior oblique view of an invasive catheter coronary angiography (B) demonstrate 3 minute septal arteries (white arrows) originating from the proximal left anterior descending artery.

Figure 3. Equivalent diastolic ECG-gated MDCT 3-chamber views of the heart prior to (A) and following (B) alcohol septal ablation. Note the asymmetrical thickening of the basal portion of the interventricular septum (*) starting 6.5 cm from the left ventricular apex on the preablation image. Note the hyperdense ablation lesion within the basal portion of the interventricular septum (white arrow) also starting 6.5 cm from the left ventricular apex on the post ablation image, confirming that the ablation was centered within the most thickened basal portion of the interventricular septum.

Figure 4. Equivalent diastolic ECG-gated MDCT short axis views of the heart base prior to (A) and following (B) alcohol septal ablation. Note the asymmetrical thickening of the basal portion of the interventricular septum (*) on the preablation image. Note the hyperdense ablation lesion within the basal portion of the interventricular septum (white arrow), confirming that the ablation was centered within the most thickened basal portion of the interventricular septum. The dotted arrow indicates automatic implantable cardioverter defibrillator right ventricular lead.
ECG-gated MDCT also allows feasible, safe, and accurate evaluation of the cardiac anatomy and function in patients with automatic implantable cardioverter defibrillator devices such as the presented patient.

This case also documents for the first time prospective ECG-triggered MDCT analysis of PTASA in the immediate/early post procedure period, by depiction and accurate localization of a hyperdense ablation defect, relative to the point of maximal thickening of the interventricular septum as disclosed on the preprocedural ECG-gated MDCT. We believe the ablation defect hyperdensity results from focal accumulation of iodinated contrast in the ablated/infracted myocardium following alcohol-induced microvascular occlusion, which causes lack of iodinated contrast washout.

Disclosures

None.

References

Multidetector Computerized Tomography Can Guide and Document Alcohol Septal Ablation in Hypertrophic Obstructive Cardiomyopathy
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