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 diuretics, 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 mitral valve leaflet-septal contact; and 3) the anatomy of the coronary arterial supply to the hypertrophied interventricular septum.

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 128×0.6 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). By 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,
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
多排螺旋 CT 引导酒精消融厚性梗阻性心肌病

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心脏影像在诊断和管理肥厚性梗阻性心肌病中有专门的价值。尤其是超声心动图和磁共振，已经广泛应用于诊断疾病，明确病理生理学，引导治疗和证实治疗效果。

病例介绍

一位 41 岁的女性为治疗有症状的肥厚性梗阻性心肌病而接受评估。因为有明确的肥厚性和扩张性心肌病家族史，其中一些病例与猝死有关，患者植入埋藏式自动心脏复律除颤器。尽管使用琥珀酸美托洛尔、维拉帕米，最后使用美托洛尔加地尔硫卓，她的心功能仍为美国心脏病学会 III 级，表现为呼吸困难和乏力，进食后症状加重。超声心动图显示室间隔不均匀增厚，二尖瓣收缩期前叶活动，中度三尖瓣和二尖瓣反流（右心室收缩压 40–60mmHg）。

我们决定为病人进行心脏和冠脉多排螺旋 CT(MDCT) 检查以便描绘①室间隔肥厚的程度和精确定位；②收缩期二尖瓣前叶活动的存在和程度以及二尖瓣和室间隔的连接；③冠状动脉供应肥厚室间隔 ECG 隔的解剖。

使用西门子 SOMATOM128 薄层高分辨率 CT 进行心电图（ECG）门控对比增强 MDCT 扫描，在吸气末屏气 6 秒，从升主动脉扫描到上腹部。我们使用三相静脉注射方法：60ml 碘对比剂（碘帕醇 370mg I/ml），随后 40ml 50/50% 碘剂/盐水混合物，最后注射 50ml 盐水。注射速度均为 5ml/秒。为了体现实时监测，使用一个自动弹丸跟踪软件，在注射 6 秒后升主动脉内对比剂密度达到预先确定的 120HU 的阈值时自动开始扫描。随后获得并使用的参数为：ECG 脉冲，构架旋转时间 300 毫秒，100kV，190mA，准直 2×64×0.6mm。这项检查的计算辐射量为 4mSv。

ECG 门控 MDCT 显示出室间隔基底部显著地非对称肥厚，舒张期 2.5cm（图 1）。重建容积数据设定每 5% 心脏循环，我们可以证实持续的二尖瓣前叶间隔连接超过收缩中期和晚期 7 个连续相。因为心脏节律在获取图像时间为 65 次/分，总的二尖瓣前叶和间隔连接持续时间为 32ms，持续 35% 的心动周期，提示明显的动态主动脉下左心室流出道梗阻（图 1B）。MDCT 也显示左心室腔收缩期几乎完全闭塞，估计射血分数为 73%。三条间隔动脉供应肥厚室间隔 ECG 隔的解剖。

在讨论治疗方案，包括药物治疗、肌切除术和经皮介入酒精消融（PTASA）之后，因为宗教信仰禁止输血，病人选择 PTASA。左心导管插入在朗斯顿双腔猪尾导管，休息时，左心室流出道收缩梯度 88mmHg (LV 192/21; AO 104/64)，存在肺动脉高压（右心室收缩压 63mmHg），冠状动脉造影显示左前降支的 3 条近端间隔支（图 2B）。这些分支的血流分配通过经胸超声心动图在注射 Definity 对比剂及亚选择性闭塞血管成形球囊获得。选择性酒精消融在第二和第三支进行，每支使用 1cc 无水乙醇，并使用实时 2 维超声心动图增强血管床的色饱和度。重复左心导管检查，左心室流出道收缩期压力梯度为 4mmHg。

PTASA 后 4 小时，进行一次非增强的，预设的触发心脏舒张器连续 MDCT 扫描，随后获得的参数是：门架循环时间 300 毫秒；100kV；205ref mA；准直 128×0.6mm，中心触发窗设置在 70%心动周期（RR 期间）时。此项检查的计算辐射量为 2 mSv。在肥厚的间隔基底部有一个高密度的消融缺损中心，密度为 96HU，而作为对比，较后的基底部心肌密度为 46HU（图 3B 和 4B）。通过对比等收缩中期 MDCT 导出的 PTAS 之前和之后
后的多平面三腔短轴室间隔重建，我们可以发现高密度消融病损是室间隔基底部最厚的部位，提示为 PTASA 的最佳位置（图 3 和 4）。

讨论

该病例是使用 ECG 门控 MDCT 在非侵入性动态评估肥厚性梗阻性心肌病患者 PTASA 之前和之后的报告。如前指出，除磁共振成像外 ECG 门控 MDCT 为评估肥厚性梗阻性心肌病提供了另一个可选择的检查方式，使我们能够充分描绘室间隔肥厚的程度和精确定位，收缩期二尖瓣前叶活动和二尖瓣瓣叶与间隔的连接。这也提供了准确的描述间隔动脉解剖的条件，这对 PTASA 之前指导治疗非常重要。进一步说，与磁共振成像相比，这项检查也允许对植入自动复律除颤器的患者，如我们的这个患者，进行安全、准确地评估心脏解剖和功能。

本例也图示了预期 ECG 触发 MDCT 第一时间分析 PTASA 即刻和术前早期过程。对高密度消融部位与室间隔最厚点的关系的描述和准确定位，都能在预处理 ECG 门控 MDCT 上揭示。我们认为消融点高密度是因为酒精使微血管梗阻引起消蚀/坏死心肌缺乏对碘对比剂的冲刷作用，碘对比剂向灶性积聚。

利益冲突

无

参考文献

图 1。ECG 门控 MDCT 舒张末期 (A) 和收缩峰期 (B) 心脏 3 腔切面。注意室间隔基底部不对称增厚，舒张末期达 2.5cm(*)。也要注意二尖瓣叶 (黑箭)，证实收缩期前叶活动，和二尖瓣瓣叶间隔连接 (白箭)。

图 2。书张启 ECG 门控 MDCT 心脏室间隔水平 2 腔切面 (A) 和同期右前斜位心导管冠脉造影 (B) 显示三个微小的间隔动脉 (白箭) 起源于左前降支。
图 3。等舒张期 ECG 门控 MDCT 心脏 3 腔切面，乙醇消融前 (A) 和后 (B)。注意非均匀性室间隔基底部增厚 (*), 在消融前图像中距离左心室尖为 6.5cm。注意消融后图像中高密度消融点位于室间隔基底部 (白箭) 距离心尖也是 6.5cm, 证明消融中心是在间隔增厚的最厚部位。

图 4。等舒张期 ECG 门控 MDCT 心脏底部短轴切面乙醇消融前 (A) 和后 (B) 图像。注意消融前非均匀的室间隔增厚 (*), 室间隔基底部高密度消融点 (白箭) 证实消融中心位于室间隔增厚最严重的部位。点箭指出的部位是埋藏式自动复律除颤器的右心室电极。