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Is multidetector computed tomography a suitable alternative to MR imaging for the assessment of myocardial necrosis after alcohol septal ablation? ☆

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ABSTRACT

Background: To compare magnetic resonance (MR) imaging and multidetector computed tomography (MDCT) for the assessment of myocardial infarction (MI) after alcohol septal ablation (ASA).
Methods: Ten patients (mean age, 60 years ± 16) were examined with both MDCT and 1.5-T MR imaging performed 10 minutes after injection, within 3 days after ASA. Half of them had a temporary pacemaker (PM) during MDCT examination. Global image quality (IQ) and localization of MI were noticed on both MDCT and MR images. Volumes of MI, contrast-to-noise ratios (CNR) and signal-to-noise ratios (SNR) were also calculated. ASA effectiveness was evaluated by echocardiography immediately and 3 months after procedure.
Results: Global IQ was considered adequate for both procedures. In 8 patients, MI reached the basal part of the septum on both MDCT and MR images. The 2 remaining patients exhibited sparing of the basal septum on MDCT and MR images. Volumes of MI were within the same range with the 2 techniques (MDCT: 22.1 ± 8.8 mL; MR imaging: 23.8 ± 9.4 mL) and correlated well each other ($R^2 = 0.85$, $p < 0.002$). The 2 patients with sparing of the basal interventricular septum had persistent gradient on echocardiography 3 months after ASA, suggesting failure of the procedure. The volumes of MI didn't correlate with the reduction of pressure gradient on echocardiography 3 months after ASA ($R^2 = 0.02$, $p < 0.05$).
Conclusions: Evaluation of post ASA MI is feasible with MDCT by comparison with MR imaging. MDCT might serve as an alternative imaging method in case of PM implantation.

1. Introduction

Hypertrophic cardiomyopathy (HCM) is characterized by myocardial hypertrophy in the absence of any other systemic or cardiac disease. Approximately 25% of patients with HCM have a dynamic left ventricular outflow tract (LVOT) obstruction caused by basal interventricular septum narrowing and/or abnormal systolic anterior motion of the mitral valve [1]. This obstructive HCM may or may not be symptomatic. In patients who remain symptomatic despite optimal medication, alcohol septal ablation (ASA) is a reliable technique for relieving obstruction and improving symptoms [2–5]. ASA consists of inducing a localized myocardial infarct (MI) in the basal part of the septum by infusing ethanol into a septal perforatory artery during coronarography. Despite good clinical results, ASA has been reported to fail in 8% of cases, with residual functional symptoms and/or a residual resting LVOT [6].

Because of its high signal-to-noise ratio and high spatial resolution, cardiac MR imaging is considered the gold standard method for assessing non-ischemic cardiomyopathies, detecting MI and determining its location and volume [7–11]. MR evaluation of myocardial necrosis after ASA has been reported by several authors [6,12,13]. Successful post-ASA MI appears as a transmural region of tissue necrosis located in the lower basal part of the septum. In contrast, nontransmural MI, MI sparing the basal septum or located at the middle part of the septum evidenced on MR imaging is predictive of the failure of ASA [13]. However, the use of MR imaging in this setting is partly limited by the fact that up to 27% of patients require permanent pacing after ASA [14,15]. Evaluation of MI with multidetector computed tomography (MDCT) has been shown to be feasible in animal models [16–19] and in humans [20–22], and the results correlate well with those of MR imaging. Moreover, pace maker (PM) implantation does not preclude CT examination. Here we examined whether MDCT might serve as an alternative for post-ASA MI evaluation in comparison to MR imaging, especially in patients with PM.

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2. Materials and methods

2.1. Study population

This prospective study was approved by the Henri Mondor Hospital ethics committee. All the participants gave their written informed consent. Between September 2009 and September 2010, 16 consecutive patients were treated with ASA because of obstructive HCM. Ten of these 16 patients underwent both cardiac MR and CT imaging within 3 days after ASA. Their baseline characteristics are listed in Table 1. Six other patients who underwent ASA during this period were ineligible because of permanent pacemaker implantation immediately after ASA (n = 2), arrhythmia (n = 1), clinical heart failure (n = 2) or renal impairment (n = 1).

2.2. ASA procedure

The indications for ASA were based on the presence of a significant LVOT, documented by echocardiography (>50 mmHg: at rest (n = 8) or after stress provocation (n = 2)), and class II or III dyspnea (New York Heart Association classification) despite medical treatment.

Coronary angiography was used to identify the appropriate septal perforator artery, which was cannulated with a 0.014-inch guidewire. The most proximal septal artery amenable to cannulation was selected as the initial artery. It was occluded with a 2-mm-diameter coronary angioplasty balloon, and an echocardiographic contrast agent (Sonovue; Bracco; Milan; Italia) was injected through the balloon in order to locate the area perfused by the artery. If the perfused area of interventricular septum was inappropriate, another septal perforator artery was cannulated. To create a localized infarct, 2–3.5 mL of absolute ethanol was injected slowly (5 to 15 min) into the septal perforator artery under echocardiographic guidance. The direct pressure gradient between the LV and the aorta was continuously recorded. The procedure was considered complete when the gradient was reduced by at least 50%. A temporary PM was systematically inserted immediately after the procedure and removed 1 to 3 days after implantation. A pacemaker was permanently inserted in one patient on day 5 (2 days after removal of the temporary PM) for complete heart block.

2.3. MR and MDCT imaging

A baseline cardiac MR imaging was performed one day before ASA. Cardiac MR imaging and MDCT were performed within 3 days after ASA. In 5 cases the 2 procedures were performed on day 3, after removal of the temporary PM. In the remaining 5 patients, MDCT was performed first (on day 1), before removal of the temporary PM ablation in order to evaluate the impairment of images induced by PM. MR imaging was performed secondarily on day 3 after removal of the PM.

MDCT examination CT was performed on a 64-section MDCT device (Lightspeed VCT; General Electric; MI; USA), with a rotation time of 330 ms and 64 x 0.6 mm-wide detector collimation. Atenolol (10 mg) was injected intravenously 5 minutes before the acquisition in 7 patients whose heart rate was more than 70 bpm. The average heart rate during the acquisition was 68 ± 12 bpm (range, 54–80). The cardiac CT was performed 10 minutes after injection of 1.5 mL/kg of contrast agent (Iomeron 400, Bracco Imaging, France) flushed by serum. To reduce radiation exposure, a lower tube output was used (80 kV, 700 mAS) and collimation was increased to 1.25 mm. The calculated radiation dose was 6 mSv ± 2. Eight-mm short-axis images were reconstructed throughout the R-R interval at steps of 10% by using retrospective electrocardiographic gating. Motion-free mid-diastolic images were selected for analysis.

MR imaging MR imaging was performed before and after ASA with a clinical 1.5-T system (Magnetom Avanto; Siemens Healthcare; Erlangen; Germany) equipped with a high-performance gradient subsystem (maximum amplitude: 40 mT/m, minimum rise: 200 µs) using an 8-channel phased-array cardiac coil. The following protocol was used. Short axis sections were acquired using Steady State Free Precession (SSFP) cine sequences. Ten minutes after an intravenous bolus of 0.2 mmol/kg body weight gadolinium chelate (gadoterate dimeglumine, Dotarem®; Guerbet, Aulnay-sous-bois, France), a three-dimensional (3D) inversion recuperation (IR) gradient-echo T1-weighted sequence was acquired during the diastolic phase on the short axis, the 2 chambers and the 4 chambers view. A dedicated TI scouting sequence was systematically used to adjust the optimal inversion recovery time (TI). The sequence parameters were as follows: mean TI 220 ± 50 ms, repetition time 3.9 ms, echo time 1.4 ms, flip angle 10°, matrix 192 × 192, field of view 300 × 270 mm, 12 sections, section thickness 4 mm. Image acquisition lasted between 12 and 20 seconds, depending on the heart rate. Two complete sets of contiguous short-axis sections (24 sections), one complete set of contiguous 4-chamber sections (12 sections) and one complete set of contiguous 2-chamber sections (12 sections) were acquired.

2.3.1. MR and MDCT image analysis

MDCT and MR images were analyzed on a dedicated platform (ADW, version 4.4, GE Healthcare, WI, USA) by two independent observers in two consecutive sessions two months apart. The readers were blinded to the clinical data and the results of the other reading session.

2.3.1.1. Qualitative analysis. The readers graded the image quality (IQ) of MDCT and MR acquisitions on a 4-point scale: 1 nondiagnostic; 2 poor; 3 adequate; 4 excellent.

Baseline MR imaging: the presence and the location of high signal areas, consistent with areas of fibrosis or MI, were noted on the late enhanced images, based on the first 16 segments of the AHA classification. Post ASA imaging: the presence and location of hyperenhanced areas (suggesting necrosis) or hypoenhanced areas (suggesting microvascular lesions) on delayed acquisitions was noted both for MDCT and MR imaging, based on the first 16 segments of the AHA classification. A sparing or a global infarction of the basal septum was noted.

2.3.1.2. Quantitative analysis. Hyper and hypoenhanced areas were delimitated on contiguous average 8-mm short-axis MDCT sections and on contiguous short-axis IR T1-weighted 4-mm slices. A dedicated software (CardiQExpress; GE Healthcare; USA) was used to calculate lesion volumes after planimetric analysis. Predefined display settings were used for MDCT images as previously reported [21]: the mean was 160 HU and the window level mean was 130 HU. MDCT attenuation and MR signal intensity were sampled by placing a region of interest (ROI) of at least 50 mm^2 within the infarct on the delayed acquisition (in hyper- and hypoenhanced regions), within the left ventricular wall (remote myocardium) and within the left cavity. Contrast-to-noise ratios (CNR) were calculated for hyper- (CNR_{hyper}) and hypo- (CNR_{hypo}) enhanced areas on delayed acquisitions, as follows: CNR = (SI of abnormal area - SI of remote myocardium) / standard deviation of SI_{noise}. Signal-to-noise ratios (SNR) were calculated for hyper- (SNR_{hyper}) and hypo- (SNR_{hypo}) enhanced areas on delayed acquisitions, as follows: SNR = SI of abnormal area / standard deviation of $SI_{noise.}$

2.4. Echocardiography

The LVOT gradient was measured by Doppler echocardiography immediately and 3 months after ASA. A gradient > 50 mm Hg at rest or after stress provocation was considered as a failure of the procedure.

2.5. Statistical analysis

SPSS version 15.0 software (Chicago; Ilinois; USA) was used for statistical analysis. Values are reported as means ± SD. Volumes of hyper- and hypoenhanced areas measured on MDCT and MR images had a normal distribution and were compared by using Pearson's correlation coefficient and Bland-Altman plots. Wilcoxon's test was used to compare mean values. P values <0.05 were considered to denote statistical significance. Between-reader and between-method agreement on semiquantitative measurements was determined with the kappa test. Interobserver reliability was assessed by calculating the 2-way random single-measure intraclass correlation coefficient (ICC). Intraobserver reliability was assessed with a 1-way random 2-measure ICC.

Table 1
Characteristics of the patients.

Age (y)*	60 ± 16 (34–88)
Men/Women	7/3
NYHA functional class*	2.5 ± 0.52
II	5
III	5
Medication	
Beta-blockers	9
Anti-aldosterone	2
ACE inhibitor	5
Echocardiographic parameters (baseline)	
LVOT gradient (mm Hg)*	59.5 ± 19
IVS thickness (mm)*	19 ± 2.6
Family history of HCM	0
Risk profile	
Diabetes	2
Hypertension	4
Hyperlipidemia	4
Current nicotine use	5
History of coronary disease	1
Family history of coronary disease	0
Body mass index (Kg/m^2)*	27 ± 3.4 (22–31)

* Mean ± SD.
NYHA = New York Heart Association; LVOT = left ventricular outflow tract; IVS = interventricular septum; ACE inhibitor = angiotensin-converting-enzyme inhibitor.

3. Results

3.1. ASA procedure

Ten perforating arteries (one per patient) were injected. The target artery was the first and second explored perforator artery in