Non-surgical septal myocardial reduction by coil embolization for hypertrophic obstructive cardiomyopathy: early and 6 months follow-up

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Aims
To evaluate the feasibility and the incidence of complete heart block (CHB) after non-surgical septal myocardial reduction by coil embolization in hypertrophic obstructive cardiomyopathy (HOCM).

Methods and results
Twenty patients with HOCM and drug-refractory symptoms underwent non-surgical myocardial septal reduction by coil embolization with detachable coils. Occlusion of septal perforator branches was successfully performed in all patients. We detected neither ventricular tachycardia nor CHB. One patient presented an interventricular septal defect after the procedure, and died 19 days later. Cardiac magnetic resonance imaging showed, in all patients, an increase in areas of hyperenhancement in the interventricular septum (IVS) compared with baseline. At 6-month follow-up, NYHA functional class and peak oxygen consumption were significantly improved compared with baseline (14.8 ± 4.5 vs. 18.5 ± 4.5 mL/kg/min; P = 0.001, respectively). Echocardiography showed a significant reduction of the IVS thickness and left ventricular outflow tract gradient (21 ± 4 mm vs. 17 ± 4 mm, P < 0.0001; 80 ± 29 to 35 ± 29 mmHg, P < 0.0001, respectively).

Conclusion
The results of this pilot non-randomized study suggest that non-surgical septal myocardial reduction by coil embolization in HOCM is feasible and does not induce CHB. Larger studies, ideally with a randomized comparison between coil embolization and alcohol septal ablation, are warranted.

Keywords
Hypertrophic obstructive cardiomyopathy  •  Coil  •  Embolization  •  Alcohol  •  Complete heart block

Introduction
Alcohol septal ablation (ASA) has been proposed as an alternative to surgical myectomy for the treatment of hypertrophic obstructive cardiomyopathy (HOCM) in drug-refractory patients.¹⁻¹⁶ This catheter-based approach involves alcohol injection into a septal perforator branch of the left anterior descending coronary artery in order to produce a myocardial infarction within the proximal interventricular septum (IVS). ASA clearly improves clinical symptoms and reduces left ventricular outflow tract gradient (LVOTG).¹⁻¹⁶ However, ASA frequently induces permanent complete heart block (CHB) requiring pacemaker implantation in 7–30%, although the risk of CHB might decrease with smaller alcohol injection.¹⁷⁻⁻²¹ CHB is probably related to the unpredictable diffusion of alcohol within the capillary bed into the myocardium inducing unexpected lesions into the conductive tissue. Female gender, bolus injection of ethanol, injecting more than one septal artery, left bundle branch block, and first-degree atrioventricular (AV) block are independent predictors of CHB after ASA.¹⁷ Moreover,

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myocardial contrast echocardiographic (ECG) quantification of risk area before ASA may predict post-procedural complications, since it has been reported that large septal infarction risk area is associated with a high incidence of CHB.32

We therefore proposed an alternative strategy inducing non-surgical septal myocardial reduction by coil embolization of perforator septal branch in order to avoid the direct alcohol toxicity upon the conductive tissue.33 Coils are routinely used to treat refractory severe bleedsings, intracranial aneurysms, and congenital vascular formations.34–36 We aimed to perform a pilot study to evaluate the feasibility, the efficacy, and the incidence of CHB after non-surgical septal myocardial reduction by coil embolization of perforator septal branch in HOCM.

**Methods**

**Patient population**

This cohort study was conducted in consecutive patients with HOCM referred to the cardiology department at Georges Pompidou European Hospital, Paris, France, between 2004 and 2006 for non-surgical myocardial septal reduction. All patients presented with drug-refractory symptoms (dyspnoea with NYHA class III or IV and/or chest pain with Canadian Cardiovascular Society angina (CCSA) class III or IV) despite optimal medical therapy, interventricular septal myocardial hypertrophy (≥18 mm), and LVOTG of at least 50 mmHg on continuous wave Doppler ECG. Patients with significant coronary artery stenosis (≥50%) and/or ejection fraction <50% and/or major mitral valvular structural abnormality were excluded. The study protocol recommended not to modify medical therapy throughout the study. All patients gave informed consent before the procedure, and the study was approved by the Hospital Ethics Committee.

**Percutaneous transluminal septal coil embolization**

A selective coronary angiogram was performed with a 6F guiding catheter in order to localize septal perforating branch arteries and to verify the absence of significant stenosis. A 7F pacemaker lead was systematically placed in the right ventricle except in two patients who already had a permanent pacemaker. A 4F pigtail catheter was positioned into the left ventricular (LV) cavity to monitor LVOTG throughout the procedure. Heparin (70 IU/kg intravenously) and aspirin (250 mg intravenously) were administered. A coaxial 1.5–2.5 mm balloon catheter was introduced over a 0.014 in. guidewire into the target septal perforator branch of the left anterior descending coronary artery through the 6F guiding catheter. The balloon was then inflated, and ultrasonographic contrast agent (Levovist®, 1–2 mL within 1 min) was injected through the balloon catheter during simultaneous registration of transthoracic two-dimensional ECG to determine the part of the myocardium supplied by the targeted septal artery. The target contrast perfusion area was the site of contact of the anterior mitral valve leaflet with the septum, adjacent to the region of LV outflow tract colour turbulence.32 Thereafter, embolization of the selected septal perforator branches was performed with spiral detachable coils (Cook), and the occlusion of the branches was monitored. If no sufficient reduction in the LVOTG was observed (i.e. <50%), occlusion of another septal perforator branch was performed.26 Procedural success was defined by LVOTG decrease >50%, as previously described.1–16

**Echocardiographic analysis**

In all patients, the diagnosis of HOCM was confirmed by the presence of two-dimensional ECG end-diastolic LV wall thickness >15 mm with no LV enlargement in the absence of other causes of hypertrophy. Indication of non-surgical myocardial septal reduction was based on the presence of severe functional limitation despite maximal medical therapy, with ECG upper septal hypertrophy (>18 mm) with subaortic obstruction (>50 mmHg at rest) because of systolic anterior motion of the mitral valve without major structural mitral valve abnormality. Twenty four hours before the procedure, at day 1 and 7, and 1, 3, and 6 months after coil septal embolization, LVOTG was determined at rest by continuous wave Doppler ECG. LV end-diastolic and systolic diameters, LV ejection fraction, and IVS thickness were also evaluated.

**Cardiac magnetic resonance imaging analysis**

All cardiac magnetic resonance imaging (CMRI) examinations were performed using a 1.5-T MR equipment (Sigma LX, GE Medical Systems, Milwaukee, WI, USA) and the same established techniques of cine MRI for function, of first-pass gradient-echo MRI for myocardial perfusion after injecting a bolus of 0.075 mmol gadoterate meglumine (Guerbet, Roissy, France) per kilogram of body weight, and inversion-recovery delayed contrast material enhancement MRI after injection of a total amount of 0.02 mmol gadoterate meglumine per kilogram for infarct sizing.27–32 All images were analysed by an independent observer. End-diastolic wall thickness, LV end-diastolic volume, end-systolic volume, ejection fraction, and total and septal myocardial mass were quantified with the MASS software package (MEDIS). Microvascular obstruction was identified on first pass perfusion images as an area of persistent myocardial hypoenhancement with signal intensity more than 2 SDs lower than that of the surrounding hyperenhanced myocardium. Infarct size after the septal coil embolization was measured after manual tracing in all short axis images of new hyperenhanced area, which was defined as the area within the septal myocardium with pixel signal intensity (SI) values more than 4 SD of remote, non-enhanced myocardium, as previously described.27–29

**Follow-up**

All patients were monitored for 48 h in coronary care unit, and the following 5 days in the cardiology department with telemetry. Telemetry and ECG were recorded daily until discharge in order to detect ventricular arrhythmia, bundle branch, or auriculo-ventricular blocks. A Holter ECG was also repeated before discharge, and at 1, 3, and 6 months after the procedure. Troponin I and creatine kinase (CK) were measured at baseline and 24 h after the procedure. Clinical evaluation, electrocardiogram, and ECG were repeated at discharge, and 1, 3, and 6 months after the procedure. Metabolic exercise testing was performed before and 6 months after septal coil embolization. Finally, CMRI was performed at baseline, and repeated 7 days and 3 months after the procedure. During the six-month follow-up, medical therapy was not modified.

**Statistical analysis**

We hypothesized that the proportion of AV block would be 1% in our study population and 20% under the null hypothesis (corresponding to ASA). Based on 80% power to detect a significant difference (two-sided test and type I error risk 5%), 20 patients were required.

Data were expressed as mean ± SD or median ± interquartile range, when appropriate. For peak oxygen consumption and exercise duration, 6-month measurements were compared with baseline values using Student’s paired t-tests. For LVOTG and IVS thickness, repeated
measures over time were analysed using analysis of variance with a modelling covariance structure within subjects (a spatial power structure for unequally spaced time points was used); two-by-two comparisons of follow-up measurements vs. baseline values were performed using the adjustment method of Dunnett in order to address the problem of multiplicity. The risk of moderate to severe mitral regurgitation (grade 2+) was estimated using the generalized estimating equation method for repeated binary-response data. Pearson coefficients were computed to assess correlation between infarction size and troponin I and CK elevation. Statistical analysis was performed using SAS statistical software version 8.2 (Cary, NC 27513, USA). All statistical tests were two-sided and significance was assumed when P was <0.05.

**Results**

**Baseline characteristics of patients**

Twenty-three patients with HOCM and drug-refractory symptoms were admitted to our department during the study period (2004–2006). Three patients did not meet the inclusion criteria because their symptoms and their LVOTG were improved after modification of their medical therapy. No patients presented with exclusion criteria or refused to give their consent. Therefore, 20 consecutive patients in whom two or more drugs were previously attempted were included and underwent the procedure. Their baseline characteristics are summarized in Table 1. All patients had NYHA class III dyspnoea except one patient who presented invalid chest pain (CCSA class IV) despite optimal blockade medical therapy, and one patient in NYHA class IV. All patients had an LVOTG >50 mmHg. Two patients were previously treated by a dual-chamber pacemaker without improvement of symptoms or LVOTG. One patient had permanent atrial fibrillation.

### Table 1 Baseline characteristics of the patients

<table>
<thead>
<tr>
<th>Variables</th>
<th>n = 20 patients</th>
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<tbody>
<tr>
<td>Age (years)</td>
<td>54 ± 14</td>
</tr>
<tr>
<td>Male sex, n (%)</td>
<td>11 (55)</td>
</tr>
<tr>
<td>Drugs, n (%)</td>
<td></td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>16 (80)</td>
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<tr>
<td>Calcium-blockers</td>
<td>6 (30)</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>1 (5)</td>
</tr>
<tr>
<td>Permanent pacemaker, n (%)</td>
<td>2 (10)</td>
</tr>
<tr>
<td>NYHA functional class III</td>
<td>18 (90)*</td>
</tr>
<tr>
<td>Permanent atrial fibrillation, n (%)</td>
<td>1 (5)</td>
</tr>
<tr>
<td>Maximal septal left ventricular wall thickness (mm)</td>
<td>21 ± 3</td>
</tr>
<tr>
<td>Basal LVOTG (mmHg)</td>
<td>80 ± 29</td>
</tr>
<tr>
<td>Peak VO2 (mL/kg/min)</td>
<td>14.8 ± 4.5</td>
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</table>

NYHA, New York Heart Association; LVOTG, left ventricular outflow tract gradient; VO2, oxygen consumption.

*Data are summarized using mean ± SD, unless stated otherwise.

**Non-surgical septal myocardial reduction by coil embolization**

Occlusion of septal perforator branches was successfully performed in all patients with detachable coils (median number per patient: 4, interquartile range 3–5). The first perforator septal branch was only used in 60% (n = 12), whereas 35% (n = 7) and 5% (n = 1) had two and three perforator septal branches embolization, respectively. Procedural success was obtained in 90% (n = 18). An example of coil embolization of one perforator septal branch is shown in Figure 1. Septal perforator branches occlusion induced a limited myocardial infarction of basal IVS with an increase in both CK and troponin I levels after the ablative procedure (mean absolute change: 386 ± 538 IU/L and 11.5 ± 11.6 ng/mL, respectively). A 74 year-old female patient died 19 days following coil septal embolization. This patient presented with drug-refractory symptoms (NYHA class IV), IVS hypertrophy (18 mm, type I of Maron), and LVOTG at 160 mmHg. Obliteration of the first septal perforator artery was performed with two detachable coils. At the end of the procedure, LVOTG decreased to 10 mmHg. Neither ventricular arrhythmia nor CHB occurred. The peak of CK was high (2042 IU/L). An IVS defect was detected 24 h after the procedure by echocardiographic examination which was successfully treated by surgical repair 3 days later. Unfortunately, she died of a refractory sepsis 16 days after surgery.

**Clinical and cardiopulmonary exercise testing analysis**

Functional class was evaluated at baseline and 6 months after the procedure in only 18 out of 20 patients, since one patient died after the procedure and one patient did not present any dyspnoea, but only invalid chest pain (see above). All of these patients were in NYHA class III at baseline. Two patients were not improved with a persistent NYHA class III. A coronary angiogram confirmed sustained occlusion of the perforator septal branches. One of these patients was treated by dual-chamber pacing 4 months after coil septal ablation with a transient improvement. Peak oxygen consumption and exercise duration were significantly improved at 6 months compared with baseline 
 
(14.8 ± 4.5 vs. 18.5 ± 4.5 mL/kg/min, P = 0.0001; 7.1 ± 3.5 vs. 9.0 ± 3.8 min, P = 0.01, respectively). All the other patients were improved (i.e. one NYHA class (n = 6 patients) or two NYHA classes (n = 10

[Figure 1] Coronary angiographic projection of the left anterior descending artery with first and second septal branches (arrow) before (A) and after (B) coil embolization.
patients). The patient presenting with invalid chest pain before the procedure was free of symptoms during the follow-up.

**Electrocardiographic and Holter ECG analysis**

We detected neither sustained ventricular tachycardia nor CHB during the follow-up. Two patients presented with a permanent bundle branch block (one right and one left bundle branch block). We also never detected sustained ventricular tachycardia on Holter ECG (data not shown).

**Echocardiographic analysis**

Echocardiographic analyses are summarized in Table 2. The distribution of LVOTG at baseline, and 7 days, 1, 3, and 6 months after the procedure is shown in Figure 2A. Analysis of repeated measurements showed that there was evidence of LVOTG reduction overtime ($P < 0.0001$). It decreased significantly 7 days after the procedure when compared with baseline (45 ± 34 vs. 80 ± 29 mmHg, adjusted $P = 0.0015$) and it remained stable from day 7 to month 6, but significantly decreased when compared with baseline. Six months after the procedure, 5 patients had a persistent LVOTG ≥ 50 mmHg. However, 3 out of these 5 patients had an improvement of both NYHA functional class and peak oxygen consumption. Furthermore, LVOTG < 50 mmHg was achieved in 60% for the first 10 patients, and in 90% for the 10 latest patients suggesting a learning curve effect.

Six-month mitral regurgitation (MR) severity decreased from baseline by at least one grade in 10 patients. Moderate to severe MR (grade 2+) concerned 75% of patients at baseline and 39% at month 6. Accordingly, the risk of moderate to severe MR (grade 2+) was significantly reduced 6 months after the procedure when compared with baseline (odds ratio from the generalized estimating equation method 0.24, 95% CI 0.06–0.9, $P = 0.04$).

The distribution of end-diastolic IVS thickness across the study period is shown in Figure 28. There was evidence of IVS thickness reduction overtime from baseline to month 6 ($P < 0.0001$). In contrast with LVOTG, this decrease persisted from day 7 to month 6 ($P = 0.03$) and the baseline IVS thickness value was highly associated with subsequent measurements ($P < 0.0001$).

**Cardiac magnetic resonance imaging analysis**

CMRI was performed in 90% at baseline ($n = 18$), since 10% ($n = 2$) had already a pace-maker. Left IVS hypertrophy was confirmed in all patients. Hypokinesia of the left IVS was present in 65% ($n = 13$). First-pass imaging was normal in all patients. Patterns of hyperenhancement on delayed contrast-enhanced images were classified as confluent and limited in 45% ($n = 9$), and diffuse in 15% ($n = 3$) in the IVS. CMRI was repeated 7 days after septal ablation in 16 out of 18 patients. Kinetic worsening of the left IVS was noted in 50%. First-pass imaging showed a persistent area of hyperenhancement (i.e., microvascular obstruction) in the proximal septal region in 37.5%. Delayed contrast-enhanced images showed in all patients an extension of patchy areas of hyperenhancement in the IVS (< 20% in 2 patients, 20–50% in 6 patients, 50–75% in 4 patients, and > 75% in 4 patients). These IVS patchy areas were located on the LV side in 37.5%, and to the RV side in 62.5%. No patient had evidence of infarct-related hyperenhancement outside the target area. An example of delayed contrast-enhanced MRI is shown in Figure 3. The estimated mean of myocardial infarction size was $3.3 ± 2.9$ g (range 1–12 g), involving 3.8 ± 3.0% (range 0.4–11.2%) of the septal myocardial mass. We also found a significant positive correlation between MRI estimated myocardial infarction size and troponin I or CK elevation (Figure 4). CMRI showed a significant reduction of the IVS thickness ($19.6 ± 3.4$ vs. $17.6 ± 4.4$ mm, $P = 0.02$), and no significant variation of LV end-diastolic and systolic diameters. First-pass perfusion imaging showed a persistent hyperenhancement in the proximal septal region in 5 patients. Delayed contrast-enhanced images showed similar size of myocardial infarction in the IVS when compared with the second MRI.

**Discussion**

ASA successfully relieves obstruction and improves symptoms of patients with HOCM. However, this treatment is limited by the high incidence of permanent CHB requiring permanent pacing. The aim of this study was to evaluate the feasibility, the efficiency, and the incidence of CHB after non-surgical septal myocardial reduction by coil embolization of perforator septal arteries in patients presenting with HOCM refractory to optimal medical therapy.

**Non-surgical septal myocardial reduction by coil embolization and obstruction**

In the present study, coil septal embolization was performed under the evaluation of the myocardium supplied by perforator septal branches using contrast ECG. Septal perforator branches

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**Table 2 Echocardiography analysis**

<table>
<thead>
<tr>
<th></th>
<th>Baseline ($n = 20$)</th>
<th>1 week ($n = 19$)</th>
<th>1 month ($n = 19$)</th>
<th>3 months ($n = 19$)</th>
<th>6 months ($n = 19$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV end-diastolic diameter (mm)</td>
<td>46.5 ± 5</td>
<td>48.1 ± 5.1</td>
<td>46.7 ± 5.7</td>
<td>47.1 ± 5.3</td>
<td>48.5 ± 4.9</td>
</tr>
<tr>
<td>LV end-systolic diameter (mm)</td>
<td>25.4 ± 5.7</td>
<td>27.3 ± 6.5</td>
<td>25.2 ± 6.2</td>
<td>26.3 ± 5.3</td>
<td>26.1 ± 6.5</td>
</tr>
<tr>
<td>LV ejection fraction (%)</td>
<td>69.9 ± 5.2</td>
<td>69.7 ± 5.8</td>
<td>70.6 ± 5.9</td>
<td>72.8 ± 6.5</td>
<td>70.3 ± 6.3</td>
</tr>
<tr>
<td>Interventricular septal thickness (mm)</td>
<td>20.9 ± 3.1</td>
<td>19.2 ± 3.0</td>
<td>18.3 ± 3.4</td>
<td>17.5 ± 3.6</td>
<td>17.4 ± 4.0</td>
</tr>
<tr>
<td>LV outflow tract gradient (mmHg)</td>
<td>80 ± 25</td>
<td>45 ± 34</td>
<td>37 ± 29</td>
<td>45 ± 33</td>
<td>36 ± 29</td>
</tr>
</tbody>
</table>

Results are expressed as mean ± SD. LV, left ventricular.
occlusion was successfully performed in all the treated patients, and induced a limited myocardial infarction of the basal IVS. A procedural success was achieved in 90%. The infarct size seemed to be smaller than those reported with alcohol regarding the peak of CK and CMRI. Indeed, 50% of patients had a two-fold or higher increase of CPK values. Moreover, the peak of CK was lower than those reported with alcohol.\textsuperscript{1–16} CMRI confirmed that myocardial infarction size was lower with coil than those reported with alcohol (4 vs. 15–20 g), and that infarcts were non-transmural in all patients.\textsuperscript{27–29} However, most of these studies were performed with a high volume of ethanol.\textsuperscript{27,28} We reported a similar improvement of symptoms (including NYHA functional class and cardiopulmonary exercise testing) when compared with ASA. In contrast, the reductions of LVOTG and IVS thickness seemed to be lower than to those previously reported with alcohol.\textsuperscript{1–16} At 6-month follow-up, 10% of the patients had a persistent NYHA functional class III, and 25% had an LVOTG >50 mmHg. In these patients, we cannot exclude the development of collateral vessels supplying the septum, as reported with covered stent septal ablation, since coronary angiograms were not systematically repeated during the follow-up.\textsuperscript{27–29} Interestingly, small studies evaluating the effect of occlusion of perforator septal branch with other devices than alcohol including glue, gelatin sponge, microcoils, or covered stents have been previously published.\textsuperscript{33–39} All of these studies reported a significant improvement of the symptoms and a reduction of LVOTG. However, in most of these small studies, long-term follow-up was missing.\textsuperscript{33–39}

Therefore, our results suggest that non-surgical myocardial septal reduction by coil embolization of perforator septal arteries in patients with HOCM is feasible and associated with limited myocardial infarction, but resulted in significant improvement of symptoms in 90% of cases and LV obstruction in 75% of cases.

**Non-surgical septal myocardial reduction by coil embolization and adverse events**

Immediately after the procedure, pain related to the septal infarction was transient and mild, and successfully limited by analgesia. We did not observe any severe ventricular arrhythmia during and after the procedure. Interestingly, CHB was absent in all patients. We only observed 10% of bundle branch blocks. This result is particularly promising when compared with ASA.\textsuperscript{1–16} Non-surgical septal myocardial reduction by coil embolization could therefore be a good alternative in patients with left or right bundle branch block or prolongation of the PQ-interval (AV block I) or those treated with amiodarone. This group of patients has an increased risk for complete AV block with alcohol ablation and may profit from a treatment with low risk for AV block. One patient died in our study secondary to an IVS defect. This old patient presented with drug-refractory symptoms (NYHA class IV) and severe subaortic obstruction (LVOTG at 160 mmHg) before the procedure. Embolization of the first perforator septal was performed with two coils after guidance with contrast ECG. After the procedure, LVOTG was dramatically decreased. However, coil septal embolization induced the largest myocardial infarction of this study with a peak of CK at 2092 IU/L, and IVS defect at the infarct site. This major adverse event has been also previously reported with alcohol.\textsuperscript{30} We therefore believe that this IVS defect was related to large septal myocardial infarction secondary to perforator septal branch occlusion. The frequency of CHB requiring permanent pacing after ASA varies from 5 to 33%.\textsuperscript{1–16} The lack of deleterious effects of coil septal embolization to the AV conduction system in the present study may be explained by the smaller size of myocardial septal infarction.
when compared with ASA. However, it can be also hypothesized that CHB after ASA is related to a direct toxic effect of ethanol on the conduction tissue, since it has been reported that the amount of ethanol and the injection of more than one septal artery, and not the infarct size are independent predictors of CHB after ASA.\textsuperscript{17,21} Injection of smaller doses of ethanol (1–2 mL) has been shown to decrease the incidence of CHB.\textsuperscript{19,20} In our study, we detected a transient microvascular obstruction only in 37.5% on first-passing imaging by MRI analysis, whereas it is present in all patients treated by ASA.\textsuperscript{41,42} Again, these results suggest a direct toxic effect of alcohol on the microvascular system which could explain the high rate of deleterious effect on the conduction tissue. Interestingly, it has also been reported in small studies that septal ablation performed with other devices than alcohol including polyvinyl alcohol foam particles, glue, gelatin sponge, microcoils, or covered stents did not induce CHB.\textsuperscript{33–39} Therefore, a ‘pure’ ischaemic infarction produced by coils or other device might be responsible for the lack of CHB.

Figure 3 Cardiac magnetic resonance imaging. Example of extension of delayed contrast-enhanced images of patchy areas of hyperenhanced myocardial in the interventricular septum 7 days (B, E) and 3 months (C, F) when compared with baseline (A, D). (A–C) Short axis view, (D–F) three-chamber view

Figure 4 Correlations between magnetic resonance imaging estimated myocardial infarction size and troponin I (A) and creatine kinase (B) elevation
Study limitations
We performed a small pilot non-randomized monocentric study to evaluate feasibility and safety of non-surgical septal myocardial reduction by coil embolization of perforator septal arteries in 20 consecutive patients with HOCM refractory to medical therapy. Larger studies, ideally with a randomized comparison between coil embolization and ASA are mandatory to further evaluate this new percutaneous approach to reduce hypertrophic septal myocardial obstruction in symptomatic patients.

In conclusion, non-surgical myocardial ablation by coil embolization of perforator septal arteries appears as a feasible strategy in patients with severe HOCM refractory to medical therapy. Further studies are warranted to compare the effectiveness and the safety of septal myocardial reduction by coil embolization and ASA in HOCM.

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References
Coil septal ablation and HOCM