Abnormal Motion of the Interventricular Septum after Coronary Artery Bypass Graft Surgery: Comprehensive Evaluation with MR Imaging

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Objective: To define the mechanism associated with abnormal septal motion (ASM) after coronary artery bypass graft surgery (CABG) using comprehensive MR imaging techniques.

Materials and Methods: Eighteen patients (mean age, 58 ± 12 years; 15 males) were studied with comprehensive MR imaging using rest/stress perfusion, rest cine, and delayed enhancement (DE)-MR techniques before and after CABG. Myocardial tagging was also performed following CABG. Septal wall motion was compared in the ASM and non-ASM groups. Preoperative and postoperative results with regard to septal wall motion in the ASM group were also compared. We then analyzed circumferential strain after CABG in both the septal and lateral walls in the ASM group.

Results: All patients had normal septal wall motion and perfusion without evidence of non-viable myocardium prior to surgery. Postoperatively, ASM at rest and/or stress state was documented in 10 patients (56%). However, all of these had normal rest/stress perfusion and DE findings at the septum. Septal wall motion after CABG in the ASM group was significantly lower than that in the non-ASM group (2.1 ± 5.3 mm vs. 14.9 ± 4.7 mm in the non-ASM group; $p < 0.001$). In the ASM group, the degree of septal wall motion showed a significant decrease after CABG (preoperative vs. postoperative = 15.8 ± 4.5 mm vs. 2.1 ± 5.3 mm; $p = 0.007$). In the ASM group after CABG, circumferential shortening of the septum was even larger than that of the lateral wall (-20.89 ± 5.41 vs. -15.41 ± 3.7, $p < 0.05$)

Conclusion: Abnormal septal motion might not be caused by ischemic insult. We suggest that ASM might occur due to an increase in anterior cardiac mobility after incision of the pericardium.

Accurate assessment of left ventricular (LV) performance is crucial for patients who will undergo cardiac surgery. However, abnormal septal motion (ASM), which is defined as either decreased excursion or paradoxical anterior movement of the interventricular septum toward the right ventricle during systole with normal thickening, frequently develops even after uncomplicated cardiac surgery; however, its etiology is still uncertain (1–3). Several studies using gated myocardial single photon emission tomography (SPECT) or echocardiography have proposed that ASM might be related to ischemic injury to the septum during coronary bypass graft surgery (CABG), to increased anterior motion of the entire heart as a result of pericardiotomy, or to restriction of right ventricular contraction from the chest wall (4–8).

However, determination of whether or not ASM is truly deteriorated is often

difficult when using routine echocardiography or gated myocardial SPECT, as either the window is sometimes limited or the regional contractile function is estimated qualitatively by visual assessment. Magnetic resonance imaging (MRI) has been used for assessment of LV function, perfusion, and viability, as well as to track movement of radiofrequency tissue tags within the myocardium for quantitative calculation of myocardial strain in any given region without limitation of the window (9, 10). Therefore, in this study, using various MRI techniques, we attempted to determine which mechanism is associated with postoperative ASM.

MATERIALS AND METHODS

Study Population

Among 63 patients with coronary artery disease who had been scheduled for elective CABG, 18 patients (mean age, 58 ± 12 years; M:F = 15:3) were enrolled in this study, as they had demonstrated normal regional wall motion and rest/stress perfusion in the septum without evidence of non-viable myocardium on baseline MRI. All patients underwent follow-up MRI three months after CABG. Patients were excluded if they were medically unstable, had a recent myocardial infarction (less than two weeks earlier), or had any contraindication to MRI (e.g., pacemaker or internal defibrillator) or to an MR paramagnetic contrast agent (e.g., hypersensitivity, allergy, etc.), or to adenosine (e.g., asthma, or heart block). Institutional review board approval was obtained for our retrospective review of patients' medical records and all patients provided informed consent for participation in the MRI study.

MR Imaging

All patients underwent cardiac MR imaging using a 1.5-T unit (Intera CV release 10; Philips Medical Systems, Best, Netherlands) equipped with high-performance gradients for cardiac MRI (maximum slew rate, 150 T/m/sec; gradient strength, 40 mT/m) and a five-element phased-array cardiac coil. MRI was performed with rest/stress perfusion, rest cine images, and delayed enhancement imaging before surgery. Electrocardiographic gating and triggering were performed using a vectorcardiographic method. Fast survey images were acquired for determination of the true short axis of the left ventricle.

Following acquisition of rest cine scans in the standard views, adenosine (Denosin, Kukdongpharm Inc., Korea) was intravenously infused at a dose of 0.14 mg/kg/min for up to six minutes. During adenosine infusion, electrocardiographic activity was continuously monitored, and

patients' blood pressure and heart rate were measured every minute. Within the last minute of infusion, an adenosine stress MR perfusion scan was performed in order to visualize the three short-axis geometries using 40 dynamic acquisitions. Perfusion images were acquired simultaneously with injection of gadodiamide (Omniscan; GE Healthcare, Princeton, NJ), using a power injector (Spectris; Medrad, Indianola, PA), into an antecubital vein at a dose of 0.1 mmol per kilogram of body weight and an injection rate of 4 ml/sec, followed by a 20 mL-saline flush. Identical MR scan was performed with a delay of 15 minutes after stress MR perfusion in order to allow adequate clearance of the first bolus of contrast agent. Following confirmation of dissipation of the contrast agent in the myocardium, rest MR perfusion was additionally performed with a new injection of the contrast agent using the same imaging parameters used in the stress study. DE-MRI was acquired 10–15 min after the second contrast injection using an inversion recovery fast gradient-echo pulse sequence. The look-locker sequence was used for selection of an appropriate inversion time (approximately 200–350 msec) in order to nullify the signal intensity of the normal myocardium.

Follow-up MRI was performed with rest/stress perfusion, rest cine images, delayed enhancement imaging, and myocardial tagging at three months after surgery. Tagged MRI was performed using an ECG-gated segmented k-space gradient echo pulse sequence with complementary spatial modulation of magnetization (CSPAMM) with spiral readout. Following localization of the heart, three tagged short-axis cine images of the LV were prescribed using the same imaging locations and slice thickness as used in perfusion MRI. Sequences of 20 to 26 images were acquired serially over a period of 16 to 24 seconds during breath-holding at end expiration until completion of the cardiac cycle. Imaging parameters included a repetition time of 32 ms per cine-segment, echo time of 2.5 ms, excitation angle of 25°, slice thickness of 8 mm, and a 256 × 256-pixel matrix size. Fields of view were 300 × 300 mm^2 and 400 × 400 mm^2 for short- and long-axis images, respectively.

Image Analysis

Two experienced investigators (one observer with five years of experience in cardiac MR imaging, and the other observer with 12 years of experience in cardiac MR imaging) evaluated MR images by consensus. MR imaging data analysis for cine MRI, perfusion, and DE-MRI was performed using commercially available software on a commercially available workstation (View Forum, release 3.2, Cardiac Package; Philips Medical Systems). Presence

or absence of a perfusion defect or delayed hyperenhancement in the septum was qualitatively assessed on follow-up images of stress perfusion and DE-MRI. Endocardial and epicardial contours seen on short-axis images of cine MRI were traced using the semi-automated border technique for quantitative evaluation of global and regional systolic functions, such as the ejection fraction, LV end-diastolic volume, LV end-systolic volume, regional wall thickening, and regional wall motion. Presence or absence of ASM on cine MRI was determined using qualitative visual and quantitative assessment, and the final diagnosis was made by consensus. Septal wall motion was expressed as endocardial inward movement (in millimeters) from the end-diastolic to the end-systolic phase on cine MRI (11). For qualitative analysis of the ASM, we calculated the average value of regional wall motion in the septal segments (Fig. 1).

Statistical Analysis

Continuous variables were expressed as means $\pm$ standard deviation (SD) or as a median with interquartile range, whereas categorical variables were presented as absolute values and percentages. Wilcoxon's rank-sum test was performed for comparison of septal wall motion after CABG between the ASM group and the non-ASM group. Wilcoxon's signed-rank test was used for comparison of data for septal wall motion before and after CABG in the ASM group. Finally, using Wilcoxon's signed-rank test, we compared circumferential strain after CABG for both the septal and lateral walls in the ASM group. SPSS version 13 (SPSS, Inc., Chicago, IL) was used for statistical analyses. Probability values of < 0.05 were considered statistically significant.

RESULTS

Demographic data for the 18 patients enrolled in this study are summarized in Table 1. The median time to follow-up was 96 days after CABG. Postoperatively, 10 patients (56%) had ASM after CABG (Fig. 2). However, all patients showed normal rest/stress perfusion and viable myocardium at the septal wall.

Statistical differences were observed with respect to septal wall motion after CABG for both the ASM group and the non-ASM group, which was 2.1 ± 5.3 mm in the ASM group and 14.9 ± 4.7 mm in the non-ASM group ($p < 0.001$). The degree of septal wall motion decreased significantly after CABG (preoperative vs. postoperative = 15.8 ± 4.5 mm vs. 2.1 ± 5.3 mm; $p = 0.007$) in the ASM group. Circumferential shortening of the septum was even larger than that of the lateral wall in the ASM group after CABG (-20.89 ± 5.41 vs. -15.41 ± 3.7, $p < 0.05$) (Fig. 3).

DISCUSSION

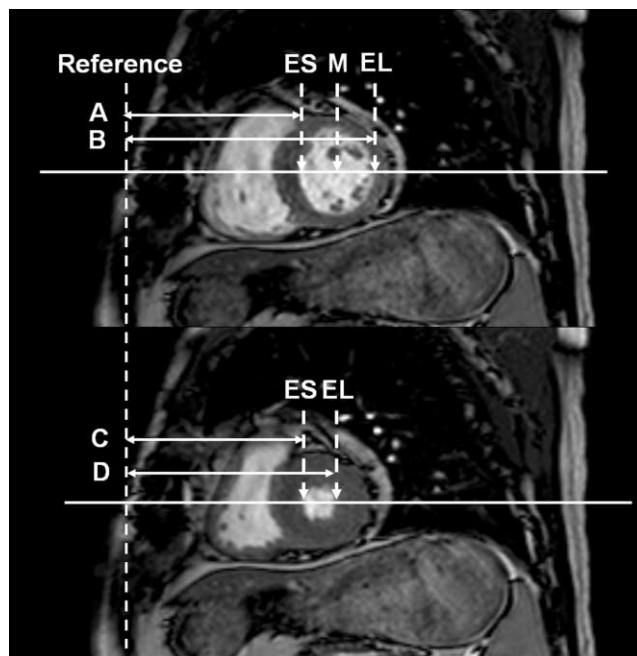The main finding of this study is that although no signifi-

Fig. 1. Schematic measurement of abnormal septal motion during cardiac cycle after coronary artery bypass graft surgery (CABG). Predefined landmarks on heart were chosen for qualitative evaluation of wall motion on end-diastole (upper panel) and end-systole (lower panel) in same post-CABG patient. Distances were measured from stationary anterior reference point. Septal wall motion was calculated as endocardial inward movement (C-A in millimeters) from end-diastolic to end-systolic phase. Note that septal wall motion was markedly decreased compared to wall motion in lateral wall (B-D in millimeters), suggesting abnormal septal motion after CABG. ES = endocardial border of interventricular septum, EL = endocardial border of lateral wall, M = midpoint of left ventricular cavity.

Table 1. Demographic Data for 18 Patients for Both Abnormal Septal Motion and Non-Abnormal Septal Motion Groups after Coronary Artery Bypass Graft Surgery

	ASM (+) (n = 10)	ASM (−) (n = 8)
Age (y)	59 ± 11	57 ± 14
M/F	8/2	7/1
LVEDV (ml)	96 ± 21	94 ± 24
LVESV (ml)	36 ± 18	34 ± 12
EF (%)	54 ± 6	56 ± 9
2/3 VD	2/8	2/6
No. of grafts	3.1 ± 1.2	3.4 ± 1.3

Note.— ASM = abnormal septal motion, CABG = coronary artery bypass graft surgery, EF = ejection fraction, LVEDV = left ventricular end diastolic volume, LVESV = left ventricular end systolic volume, No. = number, VD = vessel disease
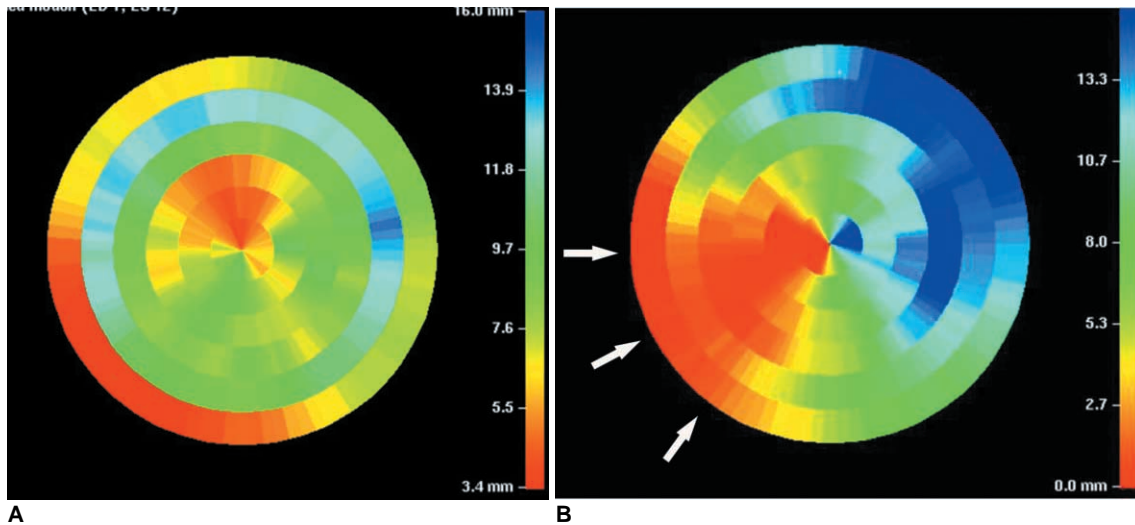
Fig. 2. Bulls eye diagrams before (**A**) and after (**B**) coronary artery bypass graft surgery. Note decreased wall motion at septum, indicating abnormal septal motion (white arrows), compared with lateral wall.
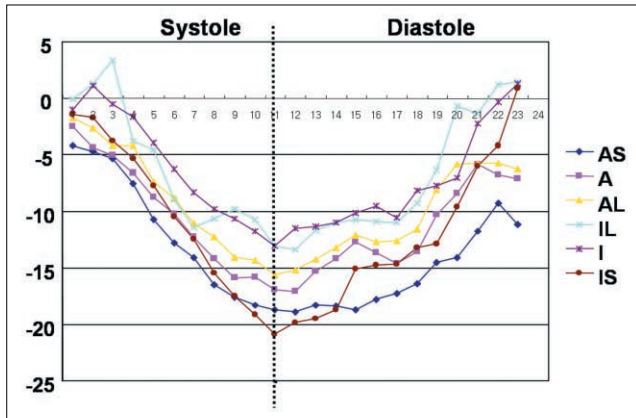

Fig. 3. Regional circumferential shortening in patients with abnormal septal motion after coronary artery bypass graft surgery. Note that circumferential stain of septum was significantly larger than that of lateral wall throughout systole.
A = anterior, AL = anterolateral, AS = anteroseptal, I = inferior, IL = inferolateral, IS = inferoseptal

cant differences were observed in wall thickening after CABG between the ASM group and the non-ASM group, the ASM group showed decreased septal motion and increased circumferential thickening. In addition, no perfusion abnormality or infarction in the septum was observed during evaluation using comprehensive MRI techniques. ASM after cardiac surgery has been noted since the early days of echocardiography (12), and marked ASM may be mistaken for septal dyskinesia, a possible manifestation of myocardial infarction or ischemia (3). There are two prevailing theories regarding the possible mechanism of ASM. The first is that ASM occurs due to an increase in anterior cardiac mobility after incision of the pericardium, as it has been noted that ASM may occur

whenever the pericardium is incised (13). Moreover, ASM is a typical finding in patients with congenital absence of the pericardium (14); therefore, it might be suggested that surgery releases the restraint of the pericardium on anterior cardiac mobility, thereby resulting in forward motion of the entire heart during systole. The other theory regarding the possible mechanism of ASM involves ischemic injury to the septum during surgery. Contractility would be expected to be abnormal after any injury to the septum (6). In our study, although septal wall motion decreased significantly after CABG in the ASM group, there was no perfusion abnormality or infarction in the septum after CABG in the ASM group. Furthermore, there were no differences in septal wall thickening before and after CABG. Therefore, our results may support the former theory concerning increased anterior cardiac motion by released pericardium, rather than the latter theory concerning ischemic injury to the septum after surgery.

To the best of our knowledge, this is the first study to examine ASM using comprehensive MRI techniques, including cine MRI, perfusion, DE-MRI, and tagged MRI. Recently, using cine MRI after CABG, Joshi et al. (15) insisted that the entire left ventricle translocated anteriorly during systole, and this could be suggested as the potential reason for larger circumferential shortening of the septum compared to that of the lateral wall, as seen in our study. However, they used only cine and DE-MRI for evaluation of changes in the motion of the heart during the cardiac cycle after CABG. Using thallium scans, Okada et al. (16) also reported that ASM was usually associated with normal septal perfusion and viability, which is consistent with our result. Using strain rate imaging with echocardiography,

Toyoda et al. (17) demonstrated that ASM in patients with CABG was caused by relatively reduced systolic myocardial velocity in the septum, while the systolic contractile function of the septum had not actually deteriorated.

Although our study population consisted of patients who underwent CABG only, ASM is also commonly seen after heart valve surgery (3). We believe that the same mechanism may be responsible for ASM in both CABG and heart valve surgery, as it occurs soon after surgery, and usually tends to resolve with time, although in some patients it can persist indefinitely (1, 16). Wranne et al. (5) suggested that the persistence or disappearance of ASM in the late postoperative period depends on factors such as recovery of right heart function with restoration of tricuspid annular motion, development of retrosternal adhesions, and development of left or right ventricular volume overload. This abnormal cardiac mobility may be related to sternotomy and pericardiotomy with anteromedial translation of the heart during systole (11).

A number of potential limitations must be addressed. First, as the total number of ASM patients was too small to allow generalization of the results, further studies will be needed on a larger scale. Second, as our results were analyzed three months after surgery, our study cannot exclude the possibility of transient ASM, which may occur during the early period after surgery and may be caused by ischemic injury to the interventricular septum during surgery.

In conclusion, ASM is a frequent finding after CABG. However, there is no evidence of ischemia or infarction in the septum on post-operative comprehensive cardiac MR imaging. Therefore, ASM might not be caused by ischemic insult. We suggest that ASM might occur due to an increase in anterior cardiac mobility after incision of the pericardium.

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