

Septal Radiofrequency Ablation for Sigmoid Ventricular Septum-Related Dynamic Obstruction: First Mechanistic Evidence of Left Bundle Branch Block as a Therapeutic Collaborator

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Case Report

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Abstract

Background

Sigmoid ventricular septum (SVS) can cause dynamic left ventricular outflow tract obstruction (LVOTO) during stress. Radiofrequency ablation (RFA) has been used for septal reduction in hypertrophic obstructive cardiomyopathy, but its application in SVS is limited and the underlying mechanisms remain poorly understood.

Results

A 77-year-old woman with symptomatic dynamic LVOTO secondary to SVS underwent endocardial RFA after failing medical therapy. Dobutamine stress echocardiography (DSE) before ablation provoked severe obstruction (peak gradient 210 mmHg) with systolic anterior motion (SAM) of the mitral valve and a large systolic vortex on blood speckle imaging (BSI). After targeted RFA of the basal septum, the patient developed new-onset complete left bundle branch block (CLBBB). Repeat DSE demonstrated resolution of SAM and obstruction (peak gradient 14 mmHg), with BSI showing restoration of laminar flow. Strain analysis revealed reduced, delayed, and post-systolic contraction of the basal septum. At 3-month follow-up, the patient remained asymptomatic.

Conclusions

This case provides the first mechanistic evidence that CLBBB may act as a therapeutic collaborator in RFA treatment for SVS-related dynamic LVOTO.

Background

Sigmoid ventricular septum (SVS) is characterized by basal septal thickening, an increased basal-to-mid septal thickness ratio (≥ 1.3), and a reduced aortoseptal angle ($< 120^\circ$) [1, 2]. During stress, SVS can provoke dynamic LVOTO through systolic anterior motion (SAM) of the mitral valve, causing angina, dyspnea, or syncope [3, 4]. Beta-blockers are first-line therapy, but some patients remain refractory [5]. Surgical myectomy and alcohol septal ablation are established for hypertrophic obstructive cardiomyopathy, but their application to SVS is limited by the thinner mid-septum and absence of suitable septal perforators [6, 7]. RFA has been used for septal reduction in obstructive cardiomyopathy [8], but its use in SVS has only been reported in two cases [9], and the role of iatrogenic conduction disturbances remains unexplored. We present the first mechanistic evaluation of RFA for SVS, using multimodality imaging (strain, post-systolic index, time-to-peak strain, and blood speckle imaging) to demonstrate that new-onset CLBBB may act as a therapeutic collaborator rather than a mere complication.

Case presentation

A 77-year-old woman presented with exertional dyspnea and syncope. Blood pressure during episodes dropped to 60/40 mmHg. Past history included 25 years of hypertension. Physical examination was unremarkable. Baseline echocardiography showed LVEF 61%, basal septal thickness 15 mm, and aortoseptal angle $< 120^\circ$ (Fig. 1A). No resting SAM or LVOT obstruction was detected (peak gradient 4 mmHg). Coronary angiography was normal.

Dobutamine stress echocardiography (DSE, 20 $\mu\text{g/kg/min}$) induced SAM with leaflet-septal contact, peak gradient 210 mmHg (Fig. 1B), causing dizziness and hypotension (84/50 mmHg). Irbesartan was discontinued and bisoprolol 5 mg daily started. Despite 5 months of therapy, symptoms persisted with one additional syncopal episode. Repeat DSE confirmed inducible SAM (peak gradient 171 mmHg). Septal RFA was performed. The procedural endpoint was the development of a left bundle branch block pattern on surface electrocardiography (Figs. 1C, 1D). QRS duration increased from 68 ms to 138 ms (new-onset CLBBB).

Post-procedural day 2: resting gradient 2 mmHg; DSE (30 $\mu\text{g/kg/min}$) showed no SAM (peak gradient 14 mmHg).

Transesophageal BSI (Fig. 2) before ablation at mid-ejection (C2) showed SAM with a large systolic vortex (yellow arrows). Post-ablation DSE demonstrated laminar flow with no SAM or vortex (C3) (Supplemental Videos 1, 2).

Strain analysis (Fig. 3) showed pre-ablation hypercontractility of the basal anterior septum (ANT_SEPT – 29%) with preserved synchrony (time-to-peak 296 ms) and minimal post-systolic shortening (PSI 7%). Post-ablation, targeted segments showed reduced strain (-9% to -13%), prolonged time-to-peak (296–329 ms), and elevated PSI (8–25%). The basal inferior wall (INF) remained normal (strain – 15%, time-to-peak 163 ms, PSI 0%). To account for inter-study variations in heart rate and loading conditions, intra-study comparisons were performed using INF as an internal reference. Table 1 summarizes the relative changes using INF as internal reference.

Table 1
Summary of Basal Septal Mechanical Changes Relative to Basal Inferior Wall (INF)

Parameter	Pre-Ablation (SEPT/ANT_SEPT vs INF)	Post-Ablation (SEPT/ANT_SEPT vs INF)	Mechanistic Interpretation
Peak Longitudinal Strain	Stronger (-0% / -7%)	Weaker (+ 6% / +2%)	RFA eliminates regional hypercontractility
Time-to-Peak Strain	Synchronous (+ 1 ms / +33 ms)	Markedly delayed (+ 133 ms / +166 ms)	CLBBB shifts contraction out of early systole
Post-Systolic Index (PSI)	Similar (-6% / -4%)	Markedly increased (+ 8% / +25%)	Contraction occurs after aortic valve closure
Values are differences between septal segments and INF during the same DSE study. Negative values indicate greater/higher in septal segments.			

Follow-Up

At 3 months, the patient was asymptomatic off beta-blockers. LVEF was 58%. DSE (40 µg/kg/min) showed mild SAM without leaflet-septal contact; peak gradient 30 mmHg. No recurrent hypotension or syncope occurred.

Discussion

This case provides the first mechanistic evidence that iatrogenic CLBBB may act as a therapeutic collaborator in RFA for SVS-related LVOTO. Successful ablation was associated with reduced, delayed, and post-systolic septal contraction.

RFA reduces regional septal contractility.** The basal anterior septum was hypercontractile pre-ablation (-29% vs. INF - 22%). After RFA, strain decreased to -13% while INF remained preserved (-15%), indicating successful targeted ablation, similar to our previous SVS series [10]

CLBBB induces delayed and post-systolic septal contraction—a therapeutic collaboration.** Using INF as internal reference, septum-to-inferior time-to-peak delay increased from near-synchronous pre-ablation (+ 33 ms) to markedly delayed post-ablation (+ 166 ms). PSI increased from - 4% to + 25%. This delayed and post-systolic contraction shifts septal shortening beyond the early-systolic window—the critical period when SAM is initiated—rendering it incapable of propelling the anterior leaflet into the LVOT. Remarkably, as early as 1995, Dalvi hypothesized that RFA-induced LBBB could treat hypertrophic cardiomyopathy by altering septal contraction timing [11]. Our case provides the first clinical support for this concept, demonstrating that CLBBB—traditionally viewed as a complication—may serve as a therapeutic collaborator when combined with targeted septal RFA.

Safety of CLBBB in this context. While new-onset CLBBB raises concern for potential progression to heart failure, large cohort studies have demonstrated that post-operative LBBB after septal myectomy

does not adversely affect long-term survival [12]. The risk of developing CLBBB-induced cardiomyopathy is low in patients with preserved baseline LVEF, and progression to overt heart failure is slow (average 12 years) when it occurs [13]. Our patient remained asymptomatic at 3 months, consistent with a benign short-term course.

Comparison with surgical myectomy. The risks of surgical myectomy in SVS are highlighted by Fujita et al., who reported catastrophic complications including complete atrioventricular block requiring permanent pacing, iatrogenic ventricular septal defect, and acute pulmonary edema [6]. In contrast, our patient developed only CLBBB without hemodynamic compromise, suggesting RFA may offer a safer alternative for selected SVS patients.

Limitations. This is a single case report; findings not generalizable. Relative contributions of direct RFA injury versus CLBBB-induced dyssynchrony cannot be separated. Long-term outcomes beyond 3 months unknown. Risk of late atrioventricular block, while low, requires informed consent.

---Patient Perspective

"Before the procedure, I could not walk a few steps without shortness of breath and dizziness, and I fainted once. After the ablation, I feel much better. I can do housework and go for walks. I no longer take heart medications. I am very grateful."

Abbreviations

ANT_SEPT

Basal anterior septum

BSI

Blood speckle imaging

CLBBB

Complete left bundle branch block

DSE

Dobutamine stress echocardiography

INF

Basal inferior wall

LVEF

Left ventricular ejection fraction

LVOTO

Left ventricular outflow tract obstruction

PSI

Post-systolic index

RFA

Radiofrequency ablation

SAM

Systolic anterior motion
SVS
Sigmoid ventricular septum

Declarations

****Ethics approval and consent to participate****

Reviewed and approved by the Ethics Committee of Clinical Medical College & Affiliated Hospital of Chengdu University

****Consent for publication****

Written informed consent obtained from the patient.

****Availability of data and materials****

All data supporting the conclusions are included in the article and supplementary material. De-identified data available from corresponding author upon reasonable request.

****Competing interests****

None declared.

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****Authors' contributions****

SH: Conceptualization, writing, review & editing. KF: Data analysis, writing. XW: Follow-up, investigation. JJ: Methodology, review. All authors approved final version.

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Figures

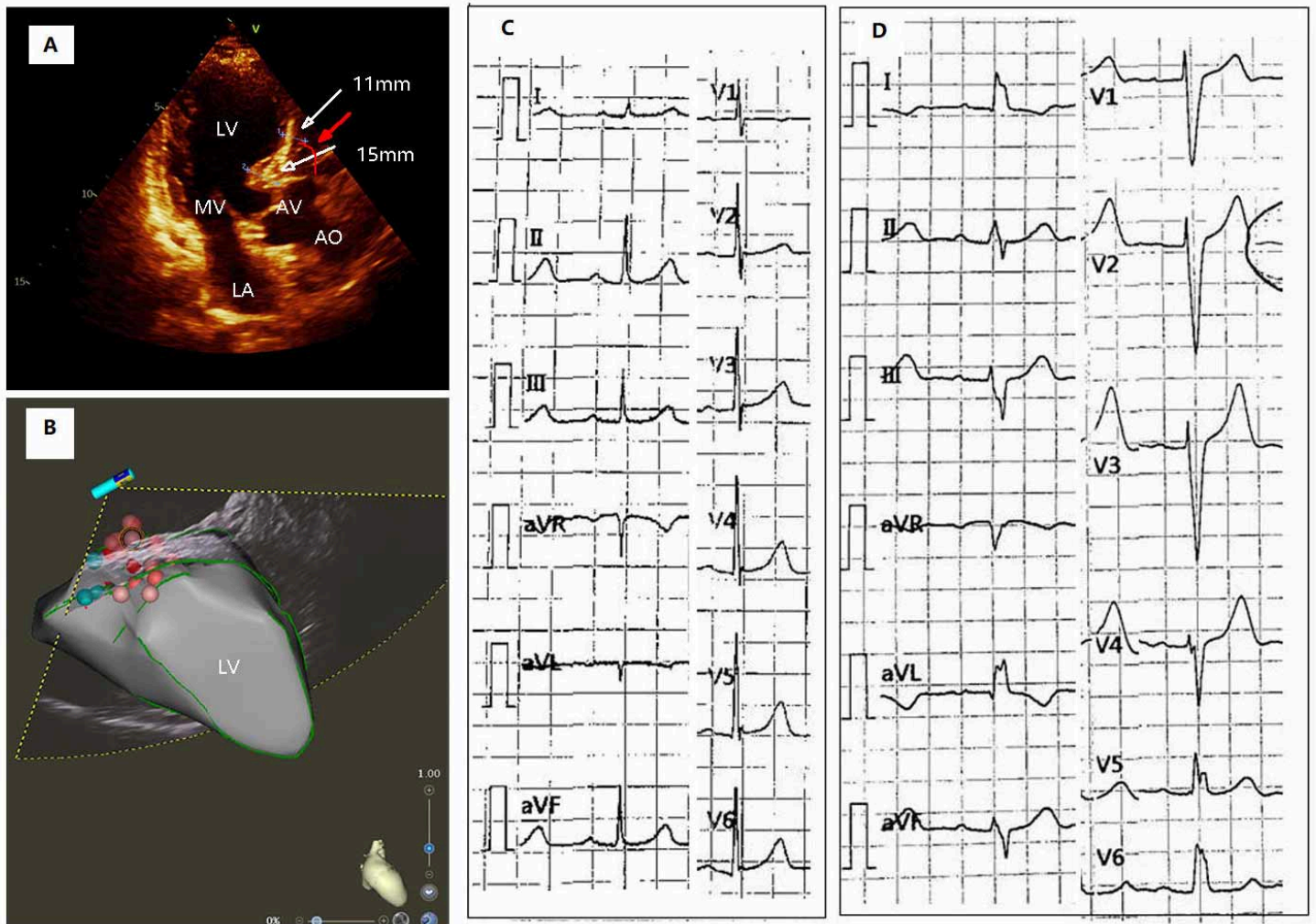


Figure 1

Transthoracic and intracardiac echocardiography-guided ablation. (A) Basal septal hypertrophy (15 mm) and aortoseptal angle $<120^\circ$. (B) ICE mapping guiding septal ablation. (C) Pre-ablation ECG. (D) Post-ablation ECG: new-onset CLBBB (QRS 138 ms).

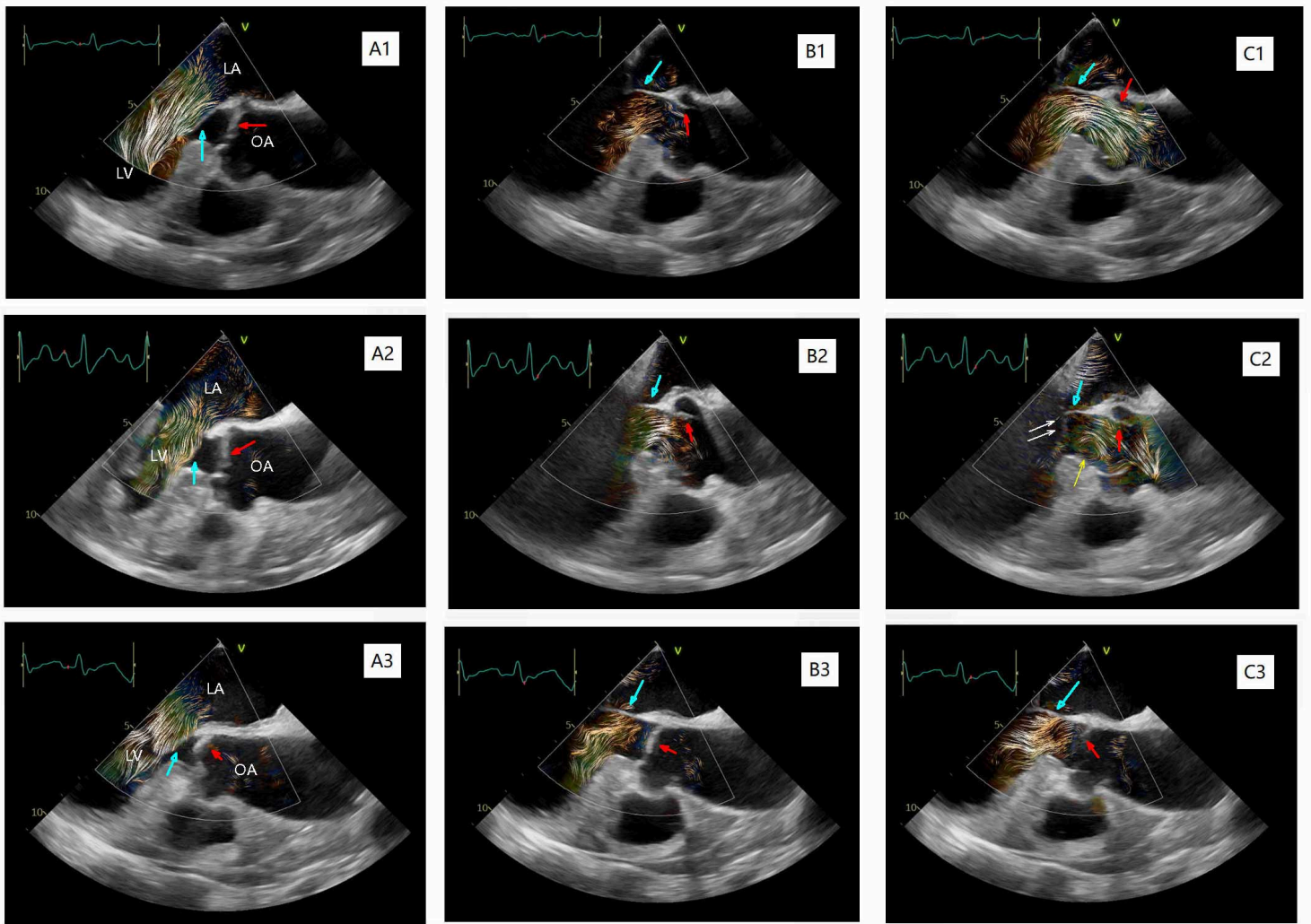


Figure 2

Blood speckle imaging (BSI) and systolic anterior motion (SAM). Rows: resting (top), pre-ablation DSE (middle), post-ablation DSE (bottom). Columns: early filling (left), early ejection (middle), mid-ejection (right). Pre-ablation DSE at mid-ejection (C2) shows SAM (white arrow) with large systolic vortex (yellow arrows). No SAM or vortex post-ablation (C3).

pre-ablation

post-ablation

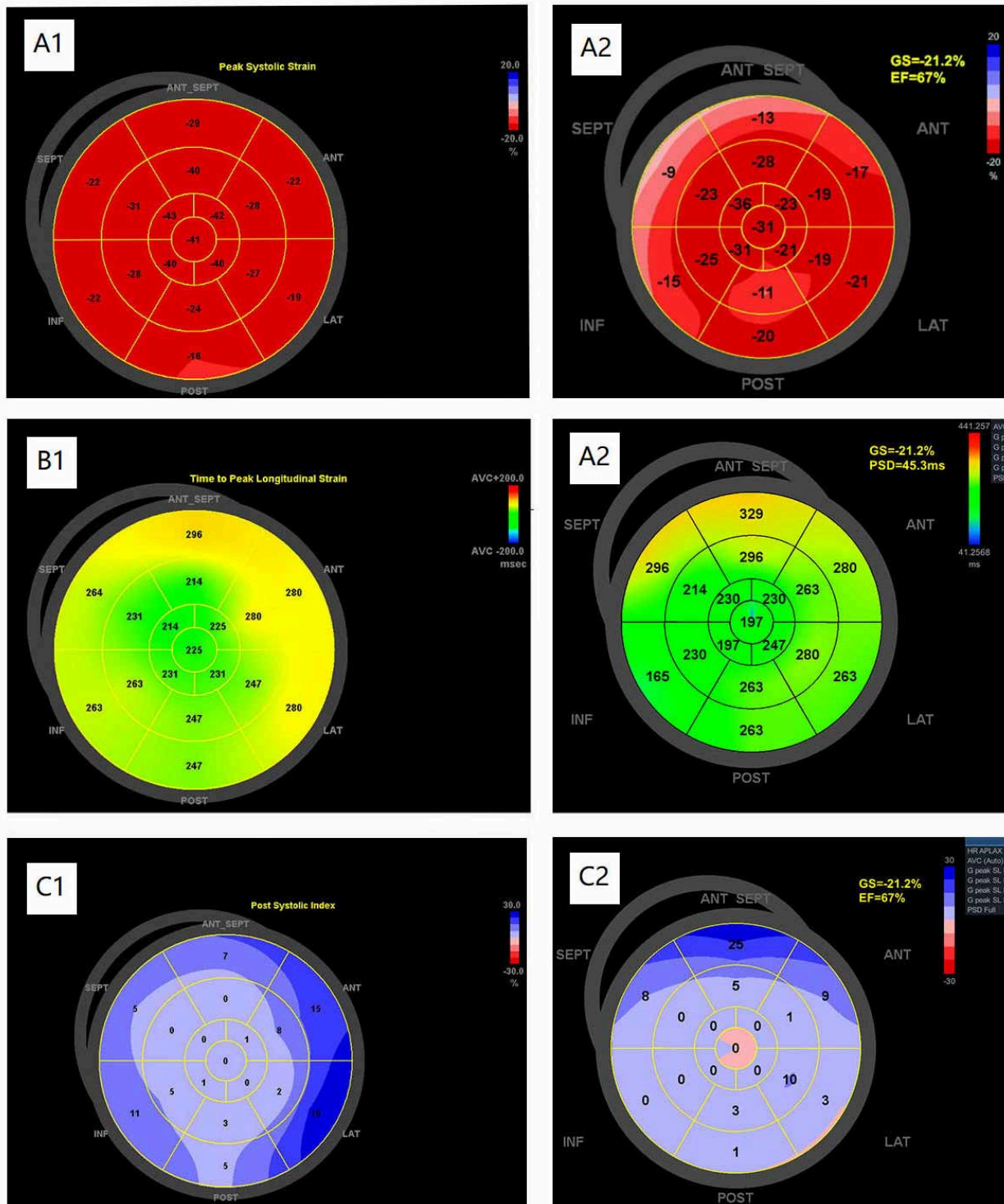


Figure 3

Dobutamine stress speckle-tracking echocardiography pre- vs post-ablation. Bull's-eye maps of longitudinal strain (A), time-to-peak strain (B), and PSI (C). Post-ablation, basal anterior septum (arrow) shows reduced strain (A2), prolonged time-to-peak (B2), and elevated PSI (C2, 25%). Basal inferior wall remains normal.

Supplementary Files

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