

Acute, Severe Mitral Regurgitation May Mask Left Bundle Branch Block–Related Dyssynchrony



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INTRODUCTION

Left ventricular (LV) systolic dysfunction can develop insidiously in patients with severe mitral regurgitation (MR), and its persistence following successful mitral valve surgery is a dreaded outcome. Such persistent systolic dysfunction is poorly understood and has been ascribed to latent contractile dysfunction masked by favorable loading conditions in chronic compensated MR.

We present a case of LV dysfunction that appeared early after successful mitral valve repair (MVR). This LV dysfunction was not apparent on the preoperative study, when the patient had severe MR. We believe that this postoperative LV dysfunction was not due to latent contractile dysfunction but rather to left bundle branch block (LBBB)–related dyssynchrony. Importantly, the patient had LBBB before and after MVR, with no change in QRS duration. Accordingly, we hypothesize that the LBBB-related dyssynchrony was masked by the loading conditions present when the patient had severe MR and manifested only when the MR was abolished. Furthermore, we believe that speckle-tracking echocardiography provided evidence that supports this hypothesis.

CASE PRESENTATION

A 64-year-old man with hemochromatosis had a murmur detected during an annual physical examination. He was hemodynamically stable and appeared well. His cardiac examination was significant for a grade 2/6 holosystolic murmur best heard at the apex with a normal rate and regular rhythm. There was no evidence of volume overload, and the remainder of his examination was unremarkable.

Electrocardiography showed normal sinus rhythm and LBBB with a QRS duration of 168 msec (Figure 1A). The patient underwent transthoracic echocardiography, which showed posterior leaflet flail, most likely related to myxomatous degeneration, and severe MR. LV size was at the upper limit of normal (LV internal diameter in diastole 55 mm), LV ejection fraction (LVEF) was 65%, and global longitudinal strain (GLS) was -20% (Figure 2A, Video 1). Transesophageal echocardiography confirmed P2 flail and normal LVEF (Video 2).

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The patient underwent successful MVR with a quadrangular resection of P2, sliding posterior leaflet resection, and placement of an annuloplasty band. Immediate postoperative transesophageal echocardiography showed total abolition of MR but a clear drop in LVEF. There was now markedly abnormal septal motion consistent with LBBB dyssynchrony (Video 3). Subsequent transthoracic echocardiography 3 months later showed borderline LV dilation (LV end-diastolic volume index 74 mL/m^2), worsened LVEF of 26%, persistent LV dyssynchrony (Video 4), trace MR, and a transmitral peak gradient of 11 mm Hg and mean gradient of 4 mm Hg. GLS was now -11.2% (Figure 2B), compared with the preoperative value of -20% ; LBBB morphology on electrocardiography was unchanged (Figure 1B).

In view of the systolic dysfunction, guideline-directed medical therapy was begun with an angiotensin-converting enzyme inhibitor and a β -blocker. Implantable cardioverter-defibrillator placement and cardiac resynchronization therapy were deferred until a trial of medical therapy was attempted, in accordance with the patient's preference. He continued to have regular follow-up for heart failure and serial echocardiography, which showed improvement in LVEF at 6-month follow-up to 45%, with improved GLS to -15% (Figure 3).

DISCUSSION

The mechanisms underlying a reduced LVEF in MR are complicated.¹⁻³ In the acute setting, LVEF is supported, if not enhanced, by suddenly reduced afterload (the so-called low-impedance leak into the left atrium), suddenly increased preload, and increased contractility.¹⁻³ In the chronic state, these factors all change, and reduced contractility may supervene, leading to suboptimal return of LVEF following surgery.¹⁻³ A significant drop in LVEF from preoperative values is a dreaded consequence of mitral valve surgery and, in the past, was attributed to poor LV preservation during surgery, complications of coronary heart disease, or removal of the low-impedance leak.² It is now well appreciated that MV repair surgery with preservation of the mitral valve apparatus, including the chordae tendineae, is associated with better postoperative LVEF compared with instances when the surgical procedure involves chordal transection.¹

We add a new entity to the differential diagnosis of reduced LVEF following MVR: unmasking of LBBB-related dyssynchrony. There is 0.1% to 0.9% prevalence of LBBB in the general population, which increases with advanced age or with heart failure.⁴ LBBB is an established cause of ventricular dyssynchrony due to delayed conduction within the ventricle and sometimes leads to hemodynamic deterioration. LBBB can be associated with deterioration of LVEF because dyssynchronous ventricular activation leads to dyssynchronous contraction. Diastolic dysfunction can accompany this drop in LVEF because dyssynchrony leads to prolonged isovolumic

VIDEO HIGHLIGHTS

Video 1: Apical four-chamber view taken from preoperative study. Ejection fraction is hyperdynamic, and there is evidence of a flail posterior leaflet.

Video 2: Composite two-dimensional and color flow Doppler transesophageal echocardiographic image, midesophageal window, 133° view, showing posterior leaflet flail (*left*) and severe MR (*right*).

Video 3: Two-dimensional view obtained at 0° plane, midesophageal window, immediately after MVr, showing significant rightward septal motion in systole, indicative of dyssynchronous contraction.

Video 4: Apical four-chamber view taken from postoperative study. Note the significant systolic septal expansion and apical “rocking” motion, typical of systolic dyssynchrony.

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contraction and relaxation times and thereby a shortened time for LV filling. Such deterioration in LV function, when accompanied by heart failure, provides a rationale for cardiac resynchronization therapy.⁵

Speckle-tracking imaging is useful for detecting LBBB-related dyssynchrony, with a recent study showing that impaired GLS measured on two-dimensional speckle-tracking echocardiography has significant and independent association with the occurrence of cardiovascular events in patients with LBBB.⁴ These results suggest that measurement of GLS can provide better risk stratification than LVEF in patients with LBBB.⁴ Critical to recognizing LV dysfunction in LBBB are the phenomena of septal flash (early, rapid short septal inward motion) and apical rocking (a short-lived early systolic septal motion of the apex and a predominantly lateral motion during ejection).^{6,7} Early systolic pressure generation in the septum prestretches the lateral wall, which is still relaxed. This augmented lateral preload leads to robust contraction and an outward motion of the septum in late systole.

Interestingly, in our patient, there was no change in QRS morphology or width over time, but significant LV mechanical dys-

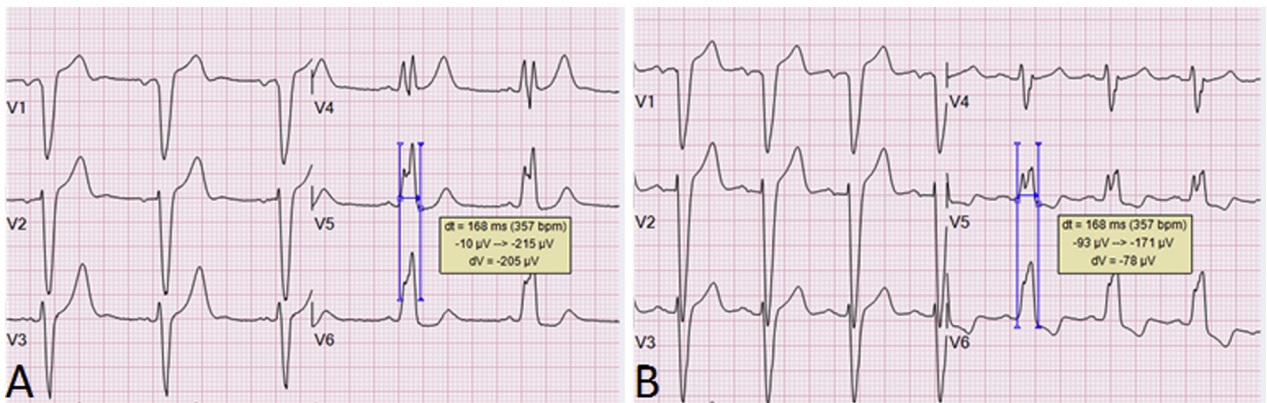


Figure 1 (A) Preoperative electrocardiogram with LBBB; QRS duration 168 msec. (B) Postoperative electrocardiogram with LBBB; QRS duration 168 msec. The electrocardiograms are essentially no different despite significant differences in hemodynamics and LV systolic function.

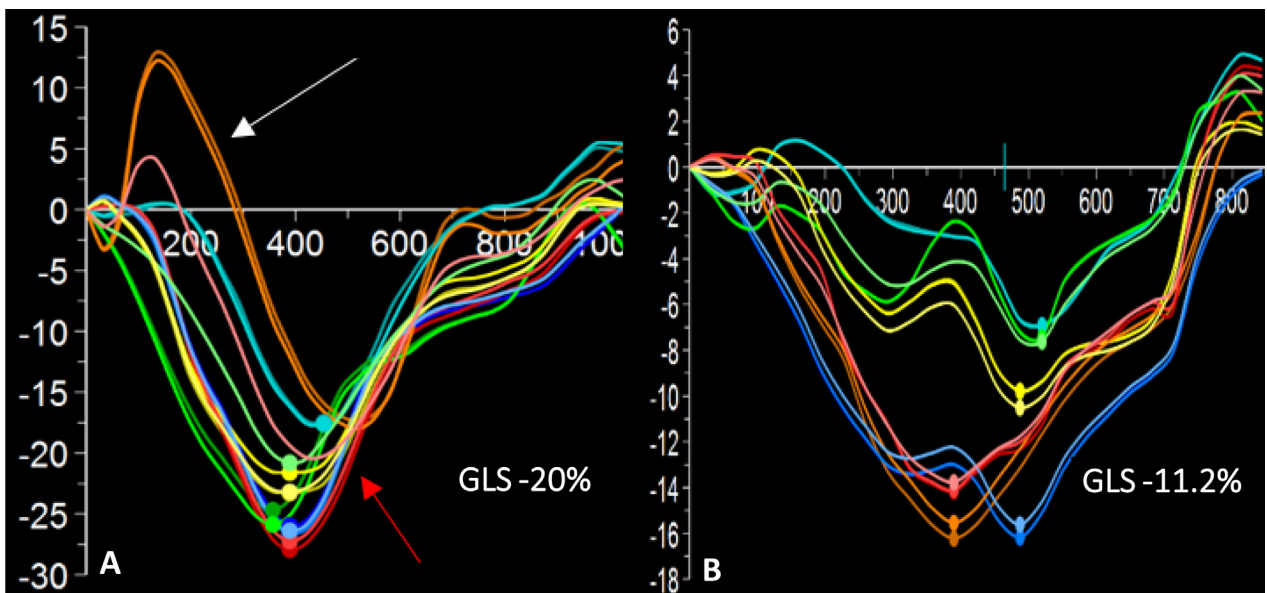


Figure 2 (A) Preoperative segmental strain curves, showing prestretching of the lateral wall (*white arrow*) and late systolic peak of the lateral segments (*red arrow*). However, there was relatively little dispersion of time to peak strain. (B) Postoperative segmental strain curves showing greater dispersion of time to peak strain (*orange-red curves vs blue curves*).

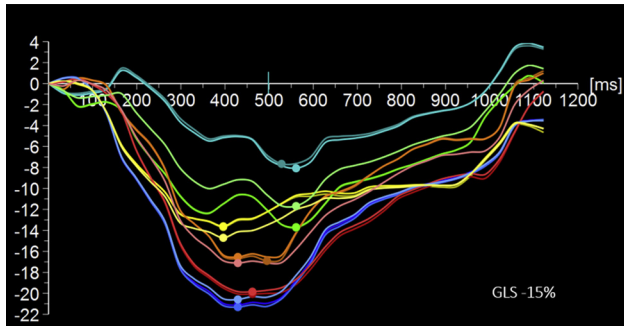


Figure 3 Follow-up GLS after goal-directed medical therapy. Segmental strain curves with decreased dispersion of time to peak strain compared with early postoperative imaging (Figure 2). GLS in this study was -15% .

synchrony seen on the postoperative study, which was not apparent on the preoperative study. We explain the appearance of dyssynchrony on the postoperative study as follows: when this patient with LBBB developed MR because of leaflet flail, early systolic unloading of the left ventricle during isovolumic contraction eliminated any apical rocking by reducing prestretching of the lateral wall. However, once the MR was repaired, there was no longer early systolic unloading, and, we speculate, the lateral wall prestretch was re-established. In this way, the “latent” LBBB dyssynchrony was unmasked. To be clear, we do not believe that the MVR created the LV dysfunction but, rather, abolished the regurgitant leak that had masked the dyssynchrony.

CONCLUSION

Severe MR can mask mechanical dyssynchrony due to LBBB, which can later be revealed after MVR. The appearance of typical dyssynchronous pattern, with apical rocking and septal flash, should serve as an

alert to this pathophysiology. Better awareness of this syndrome may allow clinicians to better understand postoperative reduction in LVEF and lead to more effective therapy.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.case.2020.05.017>.

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