HEART FAILURE

COMPLEX CASE STUDY

Severe Mitral Regurgitation and Heart Failure in a Structurally Normal Heart Induced by Rate-related Left Bundle Branch Block and Improved with Cardiac Resynchronization Therapy

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ABSTRACT. Cardiac resynchronization therapy (CRT) has been proposed to improve hemodynamics in patients with heart failure and left bundle branch block (LBBB) by resynchronization of left ventricular (LV) dyssynchrony. This case report describes a patient with otherwise no structural heart disease in whom exercise-induced, acceleration-dependent LBBB resulted in acute heart failure secondary to induction of LV dyssynchrony and severe mitral regurgitation. CRT successfully prevented the acute consequences of the functional LBBB in this patient and improved functional capacity. These findings suggest that transient rate-related LBBB should be considered in evaluation of patients with heart failure symptoms, with or without apparent heart disease, and the hemodynamic effect of LV dyssynchrony observed; its relationship to symptoms and cardiac function should be evaluated.

KEYWORDS. cardiac resynchronization therapy, functional mitral regurgitation, heart failure, left bundle branch block.

Case presentation

A 62-year-old female patient with no prior illnesses presented with several months’ history of exertional dyspnea and fatigue that had been significantly limiting her functional capacity. On the day of presentation to the emergency room, the patient experienced severe dyspnea that started while working in her yard and progressed quickly, prompting her to call emergency medical service. On presentation, physical examination demonstrated sinus tachycardia at 115 bpm, blood pressure of 146/74, oxygen saturation of 89%, diffuse bilateral pulmonary crackles, and a long systolic murmur consistent with mitral regurgitation (MR). There was no peripheral edema. The electrocardiogram (ECG) showed sinus tachycardia, wide (160 ms) QRS complex with left bundle

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branch block (LBBB) configuration, and occasional premature ventricular complexes (PVCs). Chest radiography was consistent with pulmonary alveolar edema. Transthoracic echocardiography showed hyperdynamic left ventricular (LV) systolic function (ejection fraction of 75%), moderately severe MR, mildly increased right ventricular systolic pressure, and no LV hypertrophy. The patient’s symptoms improved quickly with initiation of furosemide, carvedilol, and lisinopril. She underwent extensive work-up over the following few days: SPECT myocardial perfusion imaging was negative for myocardial ischemia. Coronary angiography excluded the presence of coronary artery disease and revealed normal LV end diastolic pressure. Enhanced spiral computed tomography of the chest showed no evidence of pulmonary embolism or parenchymal disease. Pulmonary function test was normal. Transesophageal echocardiography (TEE) demonstrated normal LV systolic function and “intermittent” severe MR. The patient was subsequently referred for consideration of mitral valve surgery. Upon evaluation at our facility, the patient was still experiencing exertional dyspnea. An ECG showed normal
sinus rhythm at 55 bpm with no intraventricular conduction abnormality. Upon review of TEE images, the mitral valve was morphologically normal; however, intermittent MR was observed, with severe (3–4/4) MR present during aberrantly (LBBB pattern) conducted sinus beats and minimal MR during slower heart rate (60–70 bpm) with no aberration (Figure 1). Treadmill stress echocardiography was performed using the modified Bruce protocol. Baseline sinus rate was 54 bpm, and peak rate was 135 bpm. LBBB developed once the sinus rate accelerated to above 65–70 bpm. The patient exercised for only 6 min, and the stress was stopped due to fatigue and dyspnea. Transthoracic echocardiography showed minimal mitral regurgitation at baseline and severe mitral regurgitation both at moderate and peak stress levels after development of LBBB, with subsequent complete resolution of MR once the heart rate decreased and LBBB disappeared. Holter monitoring also revealed rate-related LBBB at heart rates faster than 65–70 bpm (Figure 2).

On the basis of the reproducibility of the acceleration-dependent LBBB at relatively slow sinus rates and induction of acute LV dyssynchrony associated with severe mitral regurgitation accompanied by severe dyspnea, the patient underwent implantation of a biventricular pacemaker. The LV lead was positioned in the left marginal vein. Following biventricular pacemaker

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**Figure 1:** Transesophageal echocardiographic images of the mitral valve during ventricular systole. (a) Severe mitral regurgitant flow is observed during sinus rhythm with LBBB. (b) Minimal mitral regurgitation in observed sinus rhythm with normal QRS duration. Both images were acquired during the same session.

**Figure 2:** Holter monitor tracing showing normal sinus rhythm with normal QRS complexes at rates of 60–70 bpm and rate-related left bundle branch block at sinus rates faster than 70 bpm.
implantation, transthoracic echocardiogram was performed. Atrial pacing (AAI) at 90 bpm induced LBBB aberrancy and severe mitral regurgitation (Figure 3); on the other hand, biventricular pacing (DDD) at 90 bpm was associated with narrowing of the QRS duration (from 160 to 132 ms) as well as significant reduction of the degree of mitral regurgitation (from grade 3–4/4 to grade 1/4). The pacemaker was programmed to DDD mode at 50–140 bpm with an AV delay of 100 ms. At the 3-month follow-up, the patient reported a dramatic improvement in her functional capacity and activity tolerance with resolution of her dyspnea, a benefit that was maintained over a 2-year follow-up period.

**Discussion**

Functional MR is an important component in the pathophysiology of dilated cardiomyopathy and heart failure, and is characterized by the absence of structural abnormalities of the mitral valve leaflets, chordae, and papillary muscles. The most common secondary cause of functional MR is dilated cardiomyopathy (idiopathic or ischemic). LV conduction delay or its equivalent in the form of right ventricular pacing can precipitate or aggravate varying degrees of functional MR in patients with cardiomyopathy with or without heart failure. LV contraction initiated by LBBB alters papillary muscle function with resultant derangement of the time sequence of activation of the mitral valve apparatus, leading to altered force balance on the leaflets with impaired coaptation. CRT often reduces the varying degrees of functional MR, and such improvement occurs immediately after the initiation of therapy in patients with poor LV systolic function and LV dyssynchrony related to either spontaneous or right ventricular pacing-induced intraventricular conduction delay. CRT was shown to reduce functional MR by increasing the maximal rise of LV systolic pressure [LV+dP/dT (max)] secondary to a more coordinated LV contraction. The effective regurgitation orifice was decreased by as much as 50%, an effect directly related to the increase in LV contractility. The closing force of the mitral regurgitation was increased and so was the transmural gradient, factors facilitating mitral valve closure. To our knowledge, this case report is the first to describe that exercise-induced, acceleration-dependent LBBB resulting in acute heart failure secondary to induction of LV dyssynchrony and severe mitral regurgitation in a patient with otherwise no structural heart disease. Transient rate-related LBBB aberrancy developed at relatively slow heart rates (65–70 bpm) during exercise or atrial pacing and, therefore, resulted in severe activity intolerance. It was also demonstrated that CRT could prevent the acute consequences of the functional LBBB in this patient and improve functional capacity. These findings suggest that transient rate-related LBBB should be considered in the evaluation of patients with heart failure symptoms, with or without apparent heart disease. Such a diagnosis can potentially be missed if cardiac evaluation is performed during periods of rest and normal intraventricular conduction, in patients undergoing pharmacologic nuclear stress testing, and even in patients undergoing exercise or pharmacologic echocardiographic stress testing without echocardiographic Doppler evaluation of the mitral valve. Therefore, it seems reasonable to perform exercise stress testing and, if rate-related LBBB is observed, to evaluate the hemodynamic effect of LV dyssynchrony and its relationship to symptoms and cardiac function.

**References**