The Hemodynamic Benefit of Biventricular Pacing Therapy on Mitral Regurgitation as Demonstrated in a Patient with Ischemic Cardiomyopathy and Intermittent Left Bundle Branch Block

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Abstract

We report a 75-year-old man with ischemic cardiomyopathy who had mitral regurgitation which was increased markedly by intermittent left bundle branch block (LBBB). He complained of angina-like chest pain that was preceded by episodes of LBBB. During LBBB, a marked elevation of the V wave in the pulmonary capillary wedge pressure was shown, and an increase in mitral and tricuspid regurgitation was observed with color Doppler echocardiography. Biventricular pacing (BVP) therapy was selected so as to protect the patient from episodes of LBBB. After BVP, the patient did not experience chest pain or dyspnea. This case sheds valuable light on the ongoing investigation of the hemodynamic benefit of BVP.

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Key words: intermittent left bundle branch block, mitral regurgitation, biventricular pacing

Introduction

The efficacy of biventricular pacing (BVP) therapy for drug-refractory congestive heart failure associated with a prolonged QRS interval which can include a left bundle branch block (LBBB) has been widely recognized (1-5). The hemodynamic benefit of BVP is thought to be achieved by the correction of left ventricular (LV) dyssynchrony. Such cardiac resynchronization achieved by BVP may lead to decreased mitral regurgitation (MR) and forms part of the possible mechanism which improves congestive heart failure (2, 6). Functional MR frequently coincides with severe LV dysfunction as a result of mitral annular dilatation and/or papillary muscle dysfunction. LBBB may prolong MR by increasing pre-ejection and relaxation times (7). Here, we report an elderly patient with ischemic cardiomyopathy whose MR was markedly increased by intermittent LBBB and who was effectively treated with BVP.

Case Report

A 75-year-old man was admitted to our hospital for further follow-up examination of chest pains which had occurred on April 3, 2001. His prior medical history included: 2 episodes of anterior and inferior myocardial infarction at 66 years of age; a percutaneous coronary intervention because of unstable angina pectoris at 69 years of age in another hospital; and chest pains 1 month prior to this admission. His chest pain occurred mainly at rest in the morning. On physical examination, a grade II high-pitched systolic murmur was detected at the apex. There were no abnormal findings except for slight elevations of creatinine (1.4 mg/dl) and triglyceride (222 mg/dl) levels. The cardiothoracic ratio on the chest X-ray was 52% with no pulmonary congestion. The electrocardiographic findings on admission showed a low voltage in the limb leads, a flat T wave, a small Q wave in aVL, and a mild ischemic ST depression in V5-6. However, with chest pain, his electrocardiogram (ECG) documented a complete LBBB with a QRS duration of 160 msec. Coronary angiography showed that no significant coronary stenosis was present; thus coronary
stenosis was ruled out as a possible cause of the patient’s myocardial ischemia. Left ventriculography revealed severe diffuse hypokinesis with an LV ejection fraction of 27%.

The 24-hour ambulatory ECG revealed that LBBB with a sinus tachycardia occurred 8 minutes before the onset of chest pain (Fig. 1). Therefore, we considered that a rate-dependent LBBB was the likely cause of the chest pain. To prove this hypothesis, we observed intracardiac pressure changes using incremental right atrial pacing. LBBB was induced by a high atrial pacing rate (more than 120 beats per minutes), and the pulmonary capillary wedge pressure (PCWP) was elevated with the LBBB. Figure 2A shows the continuous recording of the PCWP curve when the LBBB disappeared with the discontinuation of the high rate atrial pacing. The marked elevation of the V wave amplitude in the PCWP, which suggests MR, was lowered from 63 mmHg to 42 mmHg with the disappearance of the LBBB. An increase in MR along with increased tricuspid regurgitation was confirmed by Doppler echocardiography (Fig. 3A, B). The pressure gradient between the right atrium and the right ventricle as calculated by the continuous Doppler method increased from 20 mmHg to 70 mmHg with the LBBB. As well, insufficient contact of both mitral leaflets was observed with M-mode echocardiography when LBBB was present (Fig. 3C, D). Color Doppler echocardiography documented that the direction of MR was towards the posterior wall of the left atrium.

The patient was first treated with a β-blocker (Carvedilol 10 mg) for rate control, in addition to drugs, such as diuretics, digitalis, and an angiotensin converting enzyme inhibitor, for heart failure. The patient’s LBBB-associated chest pain was transiently inhibited with a β-blocker during outpatient follow-up, but the patient was readmitted to our ward for chest pain and an aggravation of his heart failure on March 30, 2002. Intravenous administration of diuretics cleared his heart failure. A hemodynamic study with BVP was performed using a temporary pacing system. Although one PCWP V wave during BVP showed 18 mmHg, it was

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Figure 1. The 24-hour ambulatory ECG recorded just before admission. The occurrence of a wide QRS (LBBB) with sinus tachycardia is shown at 8 minutes before the onset of chest pain.
Mitral Regurgitation Increased by LBBB

Figure 2. Pulmonary capillary wedge pressure curve. (A) The PCW pressure curve recorded continuously when the LBBB disappeared. The marked elevation of the V wave suggesting mitral regurgitation was lowered from 63 to 42 mmHg with the disappearance of the LBBB. (B) The PCW pressure curve recorded during and immediately following temporary BVP. Although the V wave during BVP showed 18 mmHg, it increased to 40 mmHg immediately after pacing was turned off during the LBBB. PCW: pulmonary capillary wedge pressure, CLBBB: complete left bundle branch block, BVP: biventricular pacing.

elevated to 40 mmHg immediately after the pacing was turned off when the ECG showed LBBB (Fig. 2B).

Consequently, we selected BVP therapy to protect the patient from the LBBB that increased his MR. An atrial lead (Medtronic 5554-45a) was implanted in the right atrial appendage, and 2 ventricular leads (Medtronic 5054-52a, 4023) were implanted in the right ventricular apex and in a posterolateral tributary vein. Two ventricular leads were connected to a Y-adapter (Medtronic 5866-38M) and inserted into a dual chamber pulse generator (Medtronic KDR733) with the DDD mode. We set an atrioventricular delay of 80 msec, as it resulted in the lowest value of the PCWP V wave. After BVP was initiated, the patient’s MR on Doppler echocardiography obviously decreased, and his high-pitched systolic murmur disappeared. Thereafter, the patient did not experience chest pain or dyspnea.

Discussion

In the case presented, the patient’s cardiac condition was diagnosed as an ischemic cardiomyopathy induced by 2 episodes of myocardial infarction and frequent myocardial ischemic episodes. His chest pain which led to admission was first suspected to be angina pectoris, but no significant coronary artery stenosis was shown on coronary angiography. The patient’s episodes of chest pain occurred mainly at rest in the morning and were of a comparatively long duration. They were accompanied by a sinus tachycardia following LBBB. During such episodes an increase in a high-pitched systolic murmur at the apex was observed. The findings of intracardiac pressure with and without LBBB suggested that the MR was obviously increased immediately after the occurrence of LBBB. This was also documented by Doppler echocardiography. As shown in Fig. 3D, during the LBBB, the contact between the two mitral leaflets was insufficient. It is likely that the LBBB interfered with the proper closure of the mitral valve by inducing papillary muscle dysfunction or asynchronous motion of the LV. Consequently, PCWP and pulmonary artery pressures were elevated. Some patients with intermittent LBBB develop angina that is coincident with the onset of LBBB, although it is uncertain if the angina occurs as a result of myocardial ischemia or ventricular asynergy (7). This patient’s chest pain may have been caused by the pulmonary artery pressure elevation. It was thought that the short duration of the LBBB episodes in our patient prevented the patient from developing the typical signs and symptoms of heart failure, though with one episode he did develop heart failure. The patient’s episodes of chest pain were completely inhibited with BVP.

Recent large-scale prospective trials have proven the efficacy of BVP in congestive heart failure associated with an intraventricular conduction disturbance (1, 3). Most of these patients have LBBB, whereas to date the usefulness of BVP has been reported only in patients with right bundle branch block (8). The optimization of atrioventricular intervals is thought to contribute to the success of BVP (9). BVP also improves heart failure in patients with chronic atrial fibrillation (3, 4). Even though BVP is recognized as being effective, LV pacing alone is also considered to be worthwhile (6, 7, 9). In fact, the main mechanism of BVP action is thought to be the mechanical resynchronization of LV wall motion (10). However, the beneficial effect of BVP therapy on MR must not be overlooked. Etienne et al (6) assessed the long-term effects of LV-based pacing on LV function and MR in patients with refractory congestive heart failure and LBBB. They reported that the color Doppler MR jet area was sig-
YONEKURA et al

Figure 3. Color Doppler echocardiography on 4-chamber view (A, B) and M-mode echocardiography (C, D). The volumes of TR and MR are shown in (A) and (B) by white arrows. Both regurgitant flows were markedly increased with LBBB (A). The closures of anterior and posterior leaflets in mitral valve are shown in (C) and (D) by white arrows. The contact of both leaflets is insufficient in the presence of LBBB (D). LBBB: left bundle branch block, RV: right ventricle, LV: left ventricle, TR: tricuspid regurgitation, MR: mitral regurgitation.

significantly decreased along with a significant improvement of LV systolic function. Of particular interest, Reuter et al (2) reported that having no significant MR involvement is an independent predictive factor in patients for identifying nonresponders to BVP therapy. In the present case, cardiac resynchronization by BVP diminished the MR that was markedly increased by intermittent LBBB, and this led to symptomatic improvement. This case sheds valuable light on the investigation of the hemodynamic benefit of BVP.

References
