Persistent Left Bundle Branch Block in a Patient With Dilated Cardiomyopathy That Improved With Low Dose Carvedilol Therapy

Bunji KAKU,1 MD, Takao SATO,1 MD, Yosuke NAKATANI,1 MD, Shoji KATSUDA,1 MD, Tomio TAGUCHI,1 MD, Yutaka NITTA,1 MD, and Yoshio HIRAIWA,1 MD

SUMMARY

A 43-year-old Japanese woman with dilated cardiomyopathy had complete left ventricular bundle branch block (CLBBB), which had persisted for at least two years. At the time of admission, the serum brain natriuretic peptide (BNP) concentration was 502 pg/mL (normal range, 0-18 pg/mL), the left ventricular diastolic dimension (LVDd) was 59 mm, the left ventricular systolic dimension (LVDs) was 54 mm, the %fractional shortening (FS) was 8%, and the left ventricular ejection fraction (LVEF) was 19.7% by echocardiography. Low dose carvedilol was initiated for the treatment of heart failure. Adverse effects, such as progression of cardiac conduction disturbances, did not occur after initiation of carvedilol therapy. About one year after initiation of carvedilol therapy, the CLBBB disappeared and a significant improvement in left ventricular function was noted. The LVDd was 44 mm, the LVDs was 30 mm, the %FS was 33%, and the LVEF was 61%, and the serum BNP concentration was decreased to 18.5 pg/mL. We describe a case in which low dose carvedilol was effective for treating both CLBBB and left ventricular function. (Int Heart J 2008; 49: 243-248)

Key words: Dilated cardiomyopathy, Complete left bundle branch block, Low dose carvedilol

The nonselective β-receptor antagonist carvedilol is one of the most effective drugs for treating patients with chronic heart failure.1) In Japanese patients, even low dose carvedilol has been shown to improve chronic heart failure.2) We describe a case in which persistent complete left bundle branch block (CLBBB) and impaired left ventricular function improved after low dose carvedilol therapy.
CASE

A 43-year-old Japanese woman was admitted to our hospital with dyspnea on effort in February 2004. The patient was 155 cm tall and weighed 46.5 kg. The electrocardiogram at the time of admission showed CLBBB and a previous electrocardiogram in December 2003 also demonstrated CLBBB (Figure 1). A chest roentgenogram showed a cardio-thoracic ratio of 55%. The serum brain natriuretic peptide (BNP) concentration was 502 pg/mL (normal range, 0-18 pg/mL) (Figure 2) and echocardiography showed diffuse hypokinesis, dilation of the left ventricle, and asynchronous ventricular contraction. The echocardiographic data were as follows: left ventricular diastolic dimension (LVDd): 59 mm; left ventricular systolic dimension (LVDs): 54 mm; %fractional shortening (FS): 8%; left ventricular ejection fraction (LVEF): 19.7%; interventricular septal thickness (IVST): 10 mm; and left ventricular posterior wall thickness (PWT): 9 mm (Figure 3). Doppler echocardiography revealed mild mitral regurgitation and a deceleration time for the left ventricular inflow tract of 240 msec.

Coronary angiography showed no organic stenosis in her coronary arteries and left ventriculography revealed diffuse hypokinesis of the left ventricular wall. $^{123}$I MIBG scintigraphy showed diffusely reduced uptake of the myocardium and the H/M ratio was 1.76 in the early phase and 1.72 in the delayed phase. The washout rate for $^{123}$I MIBG was 20%. These results were compatible with dilated cardiomyopathy and she was treated with an angiotensin receptor antagonist.

![Figure 1](image_url). Serial changes in the electrocardiogram.
From February 2004 to March 2006, complete left bundle branch block (CLBBB) persisted. However, the CLBBB resolved in April 2007.
(losartan, 50 mg/day) and a diuretic (torasemide, 4 mg/day). After administration of these drugs, her symptoms improved. Although we attempted to use carvedilol at this time, she declined use of the $\beta$-receptor antagonist.

After discharge, she was followed as an outpatient and the CLBBB persisted. Although the serum BNP concentration decreased after administration of the angiotensin receptor antagonist and diuretic, the value remained at a modestly high level (Figure 2). Therefore, carvedilol administration was recommended and she agreed to the therapy. She was admitted again for initiation of carvedilol in March 2006 and at this time, the CLBBB was still present and the cardio-thoracic ratio was 56%. Echocardiography was performed on this second admission and the echocardiographic data were as follows: LVDd: 62 mm; LVDs: 51 mm; %FS: 18%; and LVEF: 37% (Figure 3). Doppler echocardiography revealed mild mitral regurgitation and a deceleration time for the left ventricular inflow tract of 233 msec.

Carvedilol administration was initiated at 2.5 mg/day and then increased to 7.5 mg/day. However, because of orthostatic hypotension, carvedilol was decreased to 5 mg/day and this dose was maintained (low dose carvedilol). One month after administering carvedilol, although the CLBBB persisted, no side effects, such as worsening of cardiac conduction defects, were observed. The serum BNP concentration declined after the administration of carvedilol (Figure

![Figure 2. Serial changes in serum brain natriuretic peptide (BNP) concentration. The serum BNP concentration declined after administration of carvedilol.](image-url)
2) About one year after the initiation of carvedilol therapy, the CLBBB, which had persisted for at least two years, resolved (Figure 1) and the cardio-thoracic ratio had improved to 43%. Echocardiography showed normal left ventricular systolic function. The echocardiographic data were as follows: LVDd: 44 mm; LVDs: 30 mm; %FS: 32%; and LVEF: 62% (Figure 3). Doppler echocardiography revealed very mild mitral regurgitation and a deceleration time of 285 msec for the left ventricular inflow tract. Cardiac function and disturbances of the conduction system were dramatically improved one year after initiation of carvedilol therapy.

Figure 3. Serial echocardiographic changes.
A: Before treatment. Diffuse hypokinesis, dilation of the left ventricle, and asynchronous ventricular contraction were observed.
B: After administration of losartan (50 mg/day) and torasemide (4 mg/day).
C: About one year after starting administration of carvedilol (5 mg/day).
Impaired left ventricular function improved after low dose carvedilol therapy.
DISCUSSION

Beta-blocker therapy is one of the most effective treatments for patients with chronic heart failure. Carvedilol is a nonselective β-receptor antagonist that also blocks α₁-receptors and exerts antioxidant effects. It produces an important clinical benefit in patients with mild to severe heart failure. In Japanese patients, even low dose carvedilol has been proven to improve chronic heart failure.

The presence of left bundle branch block is an important prognostic factor, especially in patients with dilated cardiomyopathy. Recently, patients with left bundle branch block who have poor left ventricular systolic function and asynchrony were thought to be candidates for bi-ventricular pacing therapy. However, even in these patients, β-blocker therapy for chronic heart failure was considered before cardiac resynchronization therapy. Beta-blocker therapy for patients with CLBBB sometimes has detrimental effects due to progression of more advanced atrioventricular block. It is therefore important to pay attention to the progression of cardiac conduction system abnormalities after initiation of carvedilol therapy. Fortunately, this adverse effect of β-blocker therapy did not occur in this patient and CLBBB, which had persisted for at least two years, resolved after one year of low dose carvedilol therapy, and was accompanied by an improvement in left ventricular function.

Although the precise mechanism of improvement in the bundle branch block after β-blocker therapy is unknown in this patient, improvement of heart failure by β-blocker therapy may play an important role. It has been proven in some patients that improvement in left bundle branch block results in the improvement in left ventricular function. On the other hand, does improvement in left ventricular function result in the improvement in left bundle branch block? In our patient, it was unclear as to whether the CLBBB or left ventricular function was improved first. However, based on its pharmacological effect, it is difficult to conclude that a direct effect of carvedilol improved the left bundle branch block. Blanc, et al reported that in about 17% of patients with dilated cardiomyopathy who had left bundle branch block and severe heart failure, complete normalization of left ventricular function was obtained 1 year after left ventricular resynchronization pacing. In these good responders, after cessation of left ventricular resynchronization pacing, the intrinsic QRS duration decreased from 182 msec (baseline) to 162 msec (1 year after). From this result, they speculated that left bundle branch block-induced dyssynchrony leads to a form of left ventricular dysfunction which aggravates intraventricular conduction disturbances. Because left bundle branch block itself accelerates left ventricular dysfunction, left bundle branch block and heart failure may have synergistic effects. Carvedilol therapy might halt this vicious cycle.
REFERENCES


