Case Report

Cardiac resynchronization therapy for heart failure induced by left bundle branch block after transcatheter closure of ventricular septal defect

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Abstract

A 54-year-old female patient with congenital heart disease had a persistent complete left bundle branch defect three months after closure by an Amplatzer ventricular septal defect occluder. Nine months later, the patient suffered from chest distress, palpitation, and sweating at daily activities, and her 6-min walk distance decreased significantly (155 m). Her echocardiography showed increased left ventricular end-diastolic diameter with left ventricular ejection fraction of 37%. Her symptoms reduced significantly one week after received cardiac resynchronization therapy. She had no symptoms at daily activities, and her echo showed left ventricular ejection fraction of 46% and 53%. Moreover, left ventricular end-diastolic diameter decreased 6 and 10 months after cardiac resynchronization therapy, and 6-min walk distance remarkably increased. This case demonstrated that persistent complete left bundle branch block for nine months after transcatheter closure with ventricular septal defect Amplatzer occluder could lead to left ventricular enlargement and a significant decrease in left ventricular systolic function. Cardiac resynchronization therapy decreased left ventricular end-diastolic diameter and increased left ventricular ejection fraction, thereby improving the patient’s heart functions.

Keywords: Ventricular septal defect; Amplatzer occluder; Left bundle branch block; Heart failure; Cardiac resynchronization therapy

1 Introduction

Application of Amplatzer occluder has become an effective and conventional way in percutaneous treatment of congenital heart disease such as ventricular septal defect (VSD). There are rare complications reported, although sometimes cardiac conduction abnormalities, for example, may occur. We met a case of congenital heart disease, got persistent complete left bundle branch block (CLBBB) which leaded to increased left ventricle and decreased left ventricular systolic function after VSD treatment by Amplatzer occluder, and finally the patient got heart function restored by cardiac resynchronization therapy (CRT).

2 Case Report

A 54-year old female with perimembranous ventricular septal defect was hospitalized for transcatheter closure of the defect. Echocardiogram showed left ventricular end-diastolic diameter (LVEDD) of 53 mm, left atrium of 44 mm, left ventricular ejection fraction of 65%, congenital heart disease, and perimembranous ventricular septal defect of 4 mm with the formation of aneurysm of the membranous septum (left to right shunts). Electrocardiogram (ECG) showed sinus rhythm and normal QRS wave. Transcatheter perimembranous ventricular septal defect closure with Amplatzer device was performed as described previously.[1] After the procedure, ECG showed sinus rhythm and CLBBB. Venous methylprednisolone was administered to the patient for three days, and ECG showed that the CLBBB disappeared and QRS wave was normal. Echocardiography showed LVEDD of 53 mm, left atrium of 45 mm, and left ventricular ejection fraction of 70%. The patient was followed up in an out-patient clinic. ECG at more than two months after the procedure showed sinus rhythm, normal QRS wave, and left electrical axis (Figure 1A). However, three months after the procedure, ECG showed CLBBB and premature ventricular contraction (Figure 1B). After three months, ECG showed persistent CLBBB (QRS wave width, 176 ms), (Figure 1C).
Figure 1. Electrocardiogram results. (A): Two months after closure of perimembranous ventricular septal defect showing normal sinus rhythm, normal QRS wave, and left electrical axis; (B): three months after closure of perimembranous ventricular septal defect showing sinus rhythm, complete left bundle branch block, occasional premature ventricular contraction, and left electrical axis; and (C): more than four months after closure of perimembranous ventricular septal defect showing sinus rhythm, complete left bundle branch block (QRS wave width, 176 ms), and left electrical axis.
Nine months after persistent CLBBB, the patient was admitted into our hospital because of her chest distress, palpitation, and sweating at daily activities. After admission, ECG showed sinus rhythm, CLBBB (QRS wave width, 174 ms), and left electrical axis. Cardiac echo showed left atrium (45 mm) and LVEDD of 68 mm and left ventricular end-systolic diameter (LVESD) of 56 mm with reduced left ventricular ejection fraction of 37% (Figure 2). The 6-min walk distance was 155 m, and the blood brain natriuretic peptide (BNP) level was 480 pg/mL. The patient was recommended to receive CRT because she exhibited CLBBB and reduced left ventricular ejection fraction. Moreover, beta-blocker metoprolol and angiotensin-converting enzyme inhibitor Benazepril were administered to the patient.

A CRT implant was performed under the consent of the patient. Coronary sinus angiography showed that the lateral cardiac vein was a good target to place the left ventricular lead. We employed Attain StarFix® model 4195 (Medtronic, Inc., Minneapolis, MN, USA), an active fixation electrode,

Figure 2. Cardiac echo results. (A): Left ventricular end-diastolic diameter of 68 mm with reduced left ventricular ejection fraction of 37% nine months after persistent complete left bundle branch block; and (B) left ventricular end-systolic diameter of 56 mm nine months after persistent complete left bundle branch block.
with deployable lobes to fix the lateral vein. The StarFix acute pacing threshold was 0.7 V, lead impedance was 554 ohms, and slope was 1.2. We observed neither diaphragmatic muscle nor phrenic nerve stimulation at 10 V pacing. An active lead (CapSureFix Novus model 5076, Medtronic, Inc.) was placed in the right ventricular apex, with a pacing threshold of 0.6 V, impedance of 868 ohms, R wave amplitude of 10.5 mV, and slope of 0.8, without phrenic nerve stimulation (PNS) at 10 V pacing. A J-shaped atrial lead (CapSure Sense model 4574, Medtronic, Inc.) was placed at the right atrial appendage, with measured pacing threshold of 0.4 V, slope of 0.2, P wave amplitude of 2.0 mV, and lead impedance of 512 ohms. The CRT device was implanted with Syncra model C2TR01 (Medtronic, Inc.), (Figure 3).

The patient’s symptoms on palpitations and chest distress after the procedure improved compared with those before the procedure. ECG showed double ventricle pacing rhythm and QRS wave width of 136 ms at 87 beats/min (Figure 4). One week after CRT, echocardiography showed an enlarged left atrium and left ventricle, as well as left ventricular ejection fraction of 41%. Six months after CRT, the patient was reviewed in our out-patient clinic. No palpitation, sweating, and chest distress at daily activities were observed. The echo showed left atrium of 44 mm, LVEDD of 61 mm, LVESD of 45 mm, and left ventricular ejection fraction of 50% (Figure 5). Ten months after CRT, the echo showed left atrium of 44 mm, LVEDD of 61 mm, LVESD of 43 mm, and left ventricular ejection fraction of 53%. The 6 min walking distance was 325 m, and the blood BNP level was 81 pg/mL.

3 Discussion

Conduction block after transcatheter closure of VSD may result in some different types of conduction blocks, includ-

![Figure 3. X-ray after cardiac resynchronization therapy. (A): posterior-anterior; (B): RAO 30 degree; and (C): LAO 45 degree. LAO: left anterior oblique; RAO: right anterior oblique.](image)

![Figure 4. ECG after cardiac resynchronization therapy showing sinus rhythm, double ventricle pacing, QRS wave width of 136 ms, and left electrical axis.](image)
Cardiac echo results. (A): left ventricular end-diastolic diameter of 61 mm with left ventricular ejection fraction of 50% six months after cardiac resynchronization therapy; (B): left ventricular end-systolic diameter of 45 mm six months after cardiac resynchronization therapy.

Left bundle branch block. Bundle branch block and atrioventricular block occurs in 3.5% to 8.6% of cases.[2–4] Most atrioventricular block and left bundle branch block are transient. Left bundle branch block has been proven to induce left ventricular remodeling and cause heart failure.[5,6] For CLBBB patients, the left ventricular motion shows significant delay and is uncoordinated. Simultaneously, the EF value and ejection time of the left ventricle decrease, suggesting that the systolic function of the left ventricle is impaired.[7] Vaillant, et al.[8] reported that heart failure may develop over a mean of 11.6 years of left bundle branch block. In our case of persistent CLBBB for nine months, the patient exhibited the symptoms of heart failure and a significant decrease in the left ventricular enlargement with left
ventricular ejection fraction after nine months. Left bundle branch block-induced left ventricle enlargement is referred to as left bundle branch block-induced cardiomyopathy.\(^9\)

CRT is an effective treatment method in typical CLBBB patients with heart failure.\(^{10,11}\) Multi-center randomized clinical research MIRACLE and its follow-up study confirmed that both left ventricular end-systolic and end-diastolic volumes decrease significantly six months after CRT, as demonstrated by Doppler echocardiography. The effect of CRT on cardiac structure and function can improve the symptoms of heart failure and survival rate.\(^{12}\) After CRT, ECG of our patient showed narrowing of the QRS wave width. Doppler echocardiography showed a decrease in LVEDD and blood BNP levels, and increase in left ventricular ejection fraction and 6 min walking distance. Moreover, the symptoms of heart failure in the patient disappeared.

This case demonstrated that persistent CLBBB for nine months after transcatheter closure with VSD Amplatzer occluder might lead to left ventricular enlargement and a significant decrease in left ventricular systolic function. CRT corrected the left and right ventricular dysynchrony complicated by CLBBB, decreased LVEDD, and increased left ventricular ejection fraction, thereby improving the patient’s heart function.

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