Delayed spontaneous reversibility of left bundle branch block in non-ischemic cardiomyopathy: a case report

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Left bundle branch block (LBBB) causes a delay in left ventricular contraction with an unsynchronized ventricular systole. LBBB is an independent determinant of morbi-mortality mainly when associated with cardiomyopathy and left ventricular dysfunction.[1]

LBBB due to non-ischemic cardiomyopathy is considered non-reversible. Such irreversibility occurs because LBBB and cardiomyopathy act in a synergic manner in order to maintain both situations. However, there are a few reports in the literature showing that some patients have had an improvement in cardiac function with normalization of QRS and have experienced a reverse remodelling with pharmacological therapy only.[2–4]

The present report shows a case of a non-ischemic dilated cardiomyopathy with a wide QRS which progressed with a normalization of QRS duration and left ventricular function without the use of cardiac resynchronization therapy (CRT).

A 68 year-old woman came to our clinic after having been referred to CRT, but she did not accept this therapy and wanted a second opinion. Previously, she had hypertension, but she did not have diabetes. She never smoked or drank alcohol. She was in class III NYHA heart failure (HF) and LBBB with a QRS width of 156 ms.

At the moment of her first visit, her blood pressure was 118/82 mmHg and her heart rate was 56 beats/min. She had been taking losartan 100 mg/day, atorvastatin 20 mg/day, furosemide 40 mg/day, and carvedilol 25mg/day for three and a half months, but she had not experienced improvement yet, and still persisted in Class III NYHA.

The initial ECG showed QRS duration of 156 ms, a broad, notched wave in leads I, aVL and V5-V6 with ST-T in the opposite direction to the QRS complex (Figure 1).

Echocardiography + Doppler showed an enlargement of the left atrium (LA) and the left ventricle (LV), a mild/moderate mitral regurgitation, and a left ventricular ejection fraction (LVEF) of 29.92%, Teicholz (Figure 2).

Since she complained of chest discomfort, she was sent to coronary angiography, which showed no significant artery obstruction. Thus, the aetiology of her cardiomyopathy was unknown.

Magnetic resonance imaging showed an increase in volume of the left ventricle with an important diffuse hypokinesia and a paradoxical motion of interventricular septum and desynchronization of septum, as well as lateral wall to septum.

After analysis of the clinical presentation, as well as physical and imaging exams, we confirmed that the CRT had been properly recommended. However, once again, she did not accept this procedure and wanted to be treated with medicine only, and without any devices. Since the only treatment option was drug therapy, we changed the angiotensin receptor blocker to enalapril 10 mg/day and the doses of spironolactone was raised to 50 mg/day. The doses of carvedilol was not raised, as the heart rate was about 56 beats/min. Recommendations were also made to take short walks on even ground, as well as maintaining a low salt diet. It was requested that she should be on out-patient care with a cardiologist appointment every three months, and at each clinic visit a 12 leads rest ECG was made. Every six months a transthoracic echocardiogram was also done.

Gradually, the patient’s clinical situation became better and her functional capacity improved day by day (the capacity to walk was increasing, as well as bathing without dyspnea). The echocardiogram made six months after being referred to CRT showed an improvement in LVEF and a decrease in left ventricular end diastolic diameter (LVEDD) and left ventricular end systolic diameter (LVESD).
Figure 1. ECG at the first visit to the clinic showing complete LBBB with market long duration (156 ms) of the QRS complex. The absence of the septal Q wave is noted in leads I, VL and V6, typical pattern of left bundle branch block. LBBB: left bundle branch block.

On the patient’s ninth visit to the clinic, about 27 months after she was referred to CRT, it was observed that the LBBB had disappeared (Figure 3). Therefore, a transthoracic echocardiogram was done, which showed that the LVEF had an important improvement to 60.28% and that the LVEDD and LVESD had returned to normal ranges (Figure 4).
Figure 3. ECG at ninth visit to the clinic (27 months after the patient’s first visit) showing an important shortening in QRS-complex (156 to 108 ms).

Figure 4. M-mode tracing of the LV (27 month after patient’s first visit to the clinic) obtained from parasternal long-axis view showing diminution of diastolic and systolic diameter. DIVEd: Left ventricular diameter in diastole; DIVEs: left ventricular diameter in systole; EF: ejection fraction; LV: left ventricular.
LBBB causes diastole and systole desynchronization between right and left ventricles leaving to left ventricular dysfunction by shortening the filling time, decreasing the septal contribution to LV ejection, and a globally depressed EF compared with normal matched controls.[5]

LBBB is associated with cardiomyopathies, arterial hypertension, coronary artery disease (CAD) and valvular heart disease, although relatively little is known about the establishment of LBBB because its onset is usually silent.[6]

When LBBB is related to non-ischemic cardiomyopathy, it is assumed that it will be permanent, as HF and LBBB act synergistically and CRT is indicated earlier in order to avoid desynchronization and to better LV contraction.[5] Interestingly, scholarly literature has shown that LBBB can sometimes be reversed only with medical treatment of HF.[5-4]

The guidelines indicate that patients with HF + LBBB and persistence of symptoms consistent with NYHA functional class III and IV, despite optimal pharmacological therapy, must be referred to CRT. Guidelines established three months on optimal medical therapy but, lengthen this time-frame before referral to CRT has been demanded.[8,9]

The present case report shows that, despite CRT not being used, the patient had an exciting improvement in her clinical situation, in echocardiogram parameters and, in the narrowing of the QRS complex only with appropriated medical treatment. However, about 27 months were required for the reverse remodelling to be completed, allowing for the QRS narrowing. Table 1 shows the changes in ECG and echocardiographic parameters.

The mechanism to explain the QRS narrowing could be the disappearance of the oedematous state of the congestive heart failure with the HF treatment, since the oedema has an influence on QRS duration.[10]

The QRS narrowing can be due to either changes in specialized cardiac conduction system, or changes in intra-myocardial impulse transmission. If the change is a result of improved impulse transmission in the specialized conduction tissue, this could be the result of improved hemodynamic, or secondary to reverse remodelling, or even electrical regeneration of the conduction tissue.[5] More efficient intra-myocardial impulse transmission could also be possible as a result of an improvement of the mechanical and hemodynamic reverse remodelling of the heart with smaller chamber sizes.[4]

Furthermore, cardiomyopathy can lead to changes in ionic currents, autonomic state, myocardial structure and conduction velocity. Likewise, cardiomyopathy may induce changes in density, distribution, and the characteristics of gap junctions channels which lead to electrical and, possibly, to mechanical cell uncoupling.[5] ACEI may reverse the process of cell uncoupling and have a beneficial effect on the gap junctions, so on the base of conduction block.[3,11]

The present case shows the achievement of a delayed complete reverse remodelling provided by long-term optimal medical therapy. It was not possible to determine whether the patient had not been on optimal therapy when she was first referred to CRT (losartan + carvedilol + spironolactone + furosemide). Essentially, the patient had been on optimal when she was first referred to CRT (losartan + carvedilol + spironolactone + furosemide). Certainly, the change in medication perhaps was not the reason for the patients’ improvement, but continuing the medication for a longer period of time was the reason whereby reverse remodelling and QRS narrowing were achieved. Perhaps waiting more than three months before referring to CRT may be safe in some cases. A study of Jin, et al.[12] emphasized the great significance of HF medication despite the patients being on the base of CRT implantation. These authors found that the use of ACEI/ARB is the strongest predictor of super-response to CRT. These findings demonstrate the importance of medicines to reverse remodelling and also to LBBB. A recent report shows four cases of QRS narrowing achieved in a mean of 866 days on medication base (range: 538–1211).[13] Thus, a clearer idea of what is optimal medical therapy is needed at present to treat HF, especially after the emerging of drugs such as ivabradine and sacubitril-valsartan.

Table 1. Changes in QRS duration and in the echocardiographic characteristics at the first visit to the clinic and at 27 months later the patient was referred to CRT.

| Electrocardiographic parameter | 1st visit to the clinic | 27th months latter |
|-------------------------------|------------------------|-------------------|
| QRS width, ms                 | 156                    | 108               |
| Echocardiographic parameters  |                        |                   |
| LVEDD, mm                     | 60.7                   | 43.3              |
| LVESD, mm                     | 52.0                   | 29.5              |
| Ejection fraction             | 29.92%                 | 60.28%            |
| Grade mitral regurgit          | Moderate               | Mild              |

Ao max: maximal aorta velocity; CRT: cardiac resynchronisation therapy; E/A ratio: early wave of diastole/late wave of atrial systole; LA: left atrium; LV: left ventricle; LVEDD: left ventricular end diastolic diameter; LVESD: left ventricular end systolic diameter.

References

1. Zannad F, Huvelle E, Dickstein K, et al. Left bundle branch block as a risk factor for progression to heart failure. *Eur J Heart Fail* 2007; 9: 7–14.
2. Kaku B, Sato T, Nakatami Y, et al. Persistent left bundle branch block patient with dilated cardiomyopathy that improved with low dose carvedilol therapy. *Int Heart J* 2008; 49: 243–248.
Van der Heijden R, Res JCJ, Jordaens LJ. Reversible left bundle branch block induced congestive heart failure. A case report. *J Electrocard* 2007; 40: 348–351.

Kloosterman M, Rienstra M, Van Gelder IC, Maass AH. Spontaneous resolution of the left bundle branch block and biventricular stimulation lead to reverse remodeling in dysynchronopathy. *J Electrocard* 2016; 49: 696–698.

Grines CL, Bashore TM, Boudoulas H, et al. Functional abnormalities in isolated left bundle branch block. The effect of intraventricular asynchrony. *Circulation* 1989; 79: 845–853.

Surkova E, Badano LP, Bellu R, et al. Left bundle branch block: from cardiac mechanics to clinical and diagnostic challenges. *Europace* 2017; 19: 1251–1271.

Ogano M, Iwasaki Y, Tanable H, et al. Cardiac resynchronization therapy restored ventricular septal myocardial perfusion and enhanced ventricular remodeling in patients with non-ischemic cardiomyopathy presenting with left bundle branch block. *Heart Rhythm* 2014; 11: 836–841.

DeFilippis E, Butler J, Vaduganathan M. Waiting period before implantable cardioverter-defibrillator implantation in newly diagnosed heart failure with reduced ejection fraction—A window of opportunity. *Circ Heart Fail* 2017; 10: e004478.

Linde C, Braunenschweig F. Cardiomyopathy and left bundle branch block – A farewell to drugs. *JACC* 2018; 71: 318–320.

Madias JE. The impact of changing oedematous states on the QRS duration: implications for cardiac resynchronization therapy and implantable cardioverter/defibrillator implantation. *Europace* 2005; 7: 158.

De Mello WC. Heart failure: how importante is cellular sequestration? The role of renin-angiotensin-aldosterone system. *J Mol Cell Cardiol* 2004; 37: 33–41.

Jin H, Gu M, Hua W, et al. Predictors of super-response to cardiac resynchronization therapy: the significance of heart failures medication, pre-implant left ventricular geometry and high percentage of biventricular pacing. *J Geriatr Cardiol* 2017; 14: 737–742.

Kucerová A, Doskar P, Dujka L, et al. Heart rate reduction after ivabradine might be associated with reverse electrical remodeling in patients with cardiomyopathy and left bundle branch block. *J Int Med Res* 2018; 46: 4825–4828.