Impact of Pulmonary Vein Isolation on Left Bundle Branch Block Following Tachycardia-induced Cardiomyopathy in a Patient with Persistent Atrial Fibrillation

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Abstract

A 61-year-old man was referred to our hospital with exertional dyspnea. Electrocardiography showed atrial fibrillation (AF) with a heart rate of 116 bpm and left bundle branch block (LBBB). Chest radiography demonstrated pulmonary congestion and cardiomegaly with a cardiothoracic ratio of 57%. Transthoracic echocardiography revealed a severely reduced left ventricular systolic function (ejection fraction: 32%), suggesting tachycardia-induced cardiomyopathy (TIC) due to AF. Following treatment for congestive heart failure and complete isolation of each pulmonary vein, the LBBB disappeared, with a complete recovery of the cardiac systolic function. This report describes a case of transient reversible LBBB associated with systolic dysfunction treated with catheter ablation.

Key words: pulmonary vein isolation, left bundle branch block, tachycardia-induced cardiomyopathy, persistent atrial fibrillation

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Introduction

Transient bundle branch block is defined as an intraventricular conduction defect that subsequently returns, if only temporarily, to normal conduction. It is considered to be evidence of the presence of organic heart disease (1). Left ventricular (LV) strain with ischemia, absolute or relative, of the dilated and hypertrophied myocardium appears to be the most common background leading to the development of left bundle branch block (LBBB) (2). Currently, pulmonary vein isolation (PVI) is effective in patients with LV dysfunction and it is associated with the reversal of atrial fibrillation (AF) tachycardia-induced cardiomyopathy following successful PVI (3). However, to the best of our knowledge, little has previously been reported regarding the effectiveness of PVI for improving LBBB. We herein report the case of a patient with reversible LBBB following tachycardia-induced cardiomyopathy that was treated with PVI, without cardiac resynchronization therapy (CRT).

Case Report

A 61-year-old man with a three-year history of AF categorized as being in New York Heart Association (NYHA) functional class IV with dyspnea was hospitalized at our institution. He had a past history of hypertension and hyperuricemia, for which he had been receiving medical therapy. On admission, electrocardiograms showed AF with a rapid ventricular response and wide QRS duration (134 ms) associated with a LBBB pattern (Fig. 1A). The level of brain natriuretic peptide was 231 pg/mL. Chest radiography revealed pulmonary congestion and cardiomegaly, with a cardiothoracic ratio of 57%. Transthoracic echocardiography was performed to assess the LV function due to the presence of electrocardiographic abnormalities. The left ventricular ejection fraction (LVEF), calculated using biplane Simpson’s rule, was decreased to 32% with diffuse severe hypokinesis (left ventricular end-diastolic diameter [LVEDD]: 56 mm, and left ventricular end-systolic diameter [LVESD]: 48 mm). Treatment with intravenous human atrial natriuretic peptides
and diuretics was initiated, and the dose of beta-blockers was carefully increased for the treatment of congestive heart failure and rapid AF. One week later, the patient’s symptoms were relieved, with heart rate control and persistent LBBB (Fig. 1B). Coronary angiography revealed normal coronary arteries, and the etiology of acute heart failure was considered to be tachycardia-induced cardiomyopathy caused by persistent AF with a rapid ventricular response. Thereafter, we performed PVI for the recurrence of AF two days after electric shock conversion to a sinus rhythm. After obtaining the patient’s informed consent, electrophysiological studies and catheter ablation for PVI were performed. RF energy was applied at the ostia of all four pulmonary veins (PVs) in the power control mode starting at 25 W with an irrigation flow rate of 17 mL/min and a maximal temperature of 42°C for 120 seconds, using a 4-mm extratipped ablation catheter (EZ Steer Thermocool NAV, Biosense Webster, Diamond Bar, USA). All four PVs were isolated from the LA using RF application during the procedure with no evidence of localized tachycardia inside the PV. The bidirectional conduction block between each PV and LA was confirmed by both the disappearance of the PV potentials and the circumferential pacing at the PV ostium using a Lasso catheter. AF could not be induced by the administration of isoproterenol and adenosine following PVI.

After the procedure, an electrocardiogram (ECG) continued to exhibit LBBB (QRS duration: 138 ms), even though the patient’s heart rate was relatively low at 80 bpm with a sinus rhythm (Fig. 1C). Surprisingly, two weeks later, an ECG showed the disappearance of the LBBB with a return to a normal sinus rhythm and QRS duration (80 ms) (Fig. 1D). Three months later, the patient’s symptoms were well controlled (NYHA 1), with the normalization of the brain natriuretic peptide level (33 pg/mL), chest radiography findings, including a cardiothoracic ratio of 51%, the echocardiographic cardiac function (LVEF: 57%) and the LV diameter (LVEDD: 52 mm, and LVESD: 34 mm), thus all suggested the presence of reversible, transient LBBB due to tachycardia-induced cardiomyopathy (Fig. 2). One year later, the patient had not experienced any further episodes of recurrent AF or LBBB on electrocardiograms.

**Discussion**

We herein present a rare case of reversible LBBB following AF tachycardia-induced cardiomyopathy after PVI. The
underlying pathology of transient bundle branch block in many cases is related to ischemic heart disease. Most patients with LBBB suffer from hypertension or ischemic heart disease, and, although either condition alone may be accompanied by this defect, the combination of the two is most consistently complicated by LBBB (4, 5). Not infrequently, however, the appearance and disappearance of bundle branch block is unaccompanied by any recognizable changes in the patient’s physical condition. When transient LBBB is observed in association with AF, it is usually caused by aberrancy that is rate dependent. However, aberrancy was excluded as a cause of the transient LBBB in this case because the wide QRS duration with the LBBB did not change under adequate rate control in the acute phase. Therefore, the transient LBBB observed in this patient was clearly associated with a reduction in the global systolic function of the left ventricle resulting from the AF tachycardia-induced cardiomyopathy.

The mechanisms underlying the development of transient bundle branch block remain obscure. Intraventricular conduction defects can be produced by a number of disturbances (6), such as the anatomic or pathologic interruption of a conducting bundle, ventricular enlargement and strain with dilatation of the appropriate chamber and functional or neurogenic depression, with or without underlying pathologic lesions in the conducting tissues (7). It can be speculated that a combination of these factors is of importance in the pathogenesis of transient bundle branch block. In this case, mechanical damage to the LBBB most likely caused the transient LBBB associated with tachycardia-induced cardiomyopathy. When the left ventricle expands with a high end-diastolic pressure in patients with heart failure, the LBBB may be stretched, and the subsequent mechanical damage may cause a conduction disturbance in the LBBB. Hence, with the elimination of persistent exposure to AF following catheter ablation, these mechanisms may occur in the opposite direction and thereby encourage the contractile properties of the myocardium. A potential explanation for LBBB and LVEF improvement also includes the enhancement of atrial contractility, the maintenance of synchronous atrioventricular contractions and the prevention of a high ventricular rate. Although it is unclear whether PVI for AF directly leads to an improved LBBB, we believe that our case implies that PVI results in the reversal of the cardiac function and conduction defects in patients with AF-induced cardiomyopathy.

Several analyses have demonstrated that AF and LBBB are useful clinical variables that can be used to identify populations at higher risk of total and sudden death among those with congestive heart failure (8, 9). While Leclercq et al. reported that biventricular pacing stimulation is particularly effective in patients with congestive heart failure and that a wide QRS is associated with AF (10), our patient showed LBBB and LV dysfunction associated with tachycardia-induced cardiomyopathy (TIC) that completely recovered following PVI. AF, heart failure and LBBB compose a wide spectrum of disease; therefore, treatment should be tailored based on a close observation of each patient’s background.

We herein reported a case of reversible transient LBBB and TIC that was treated with PVI. This case emphasizes the significance of PVI for reverse remodeling of the LV and LBBB in AF patients.

The authors state that they have no Conflict of Interest (COI).

References