

# Case Report

# Exercise-Induced Bundle Branch Block - an Infrequent Incidence

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# **ABSTRACT**

We describe an elderly woman who underwent a treadmill test preoperatively and was found to have Exercise-induced Left Bundle Branch Block without chest pain. Coronary angiogram was done which revealed a 70% stenosis in the first diagonal branch of Left Anterior Descending Artery and a 40% lesion in proximal Right Coronary Artery. Available literature attributes rate-dependent transient conduction defects to obstructive lesions of coronary arteries, slow arterial blood flow, vasospasms as well as pathology in the Bundle of His.

Keywords: Bundle branch block; Exercise; Left bundle branch block; Transient; Treadmill test

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## INTRODUCTION

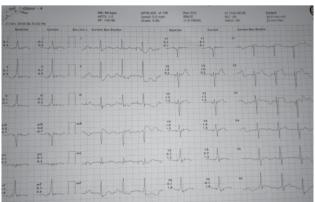
Exercise-induced bundle branch block (BBB) has been reported to occur in 0.2% to 1.1% of the population according to different series. <sup>1-3</sup> Exercise induced left bundle branch block (LBBB) has been found in association with and without underlying structural heart disease. <sup>4</sup> It has also been associated with slow arterial coronary flow <sup>5</sup> and coronary vasospasm which is relieved with calcium channel blocker. <sup>6</sup>

## CASE REPORT

A 72 years old female with known cardiovascular risk factors of Hypertension (HTN) and Type 2 Diabetes Mellitus (T2DM), under oral hypoglycemic drugs along with a beta-blocker since 6 years for frequent ventricular premature contractions noted on her 12 lead electrocardiogram (ECG), was evaluated for preoperative cardiac assessment before an exploratory laparotomy to relieve her of a suspicious overgrowth in the terminal ileum and caecum. She had limited physical activity due to shortness of breath aggravated with exertion. On examination, her Body mass index was 22.89kg/m² with a resting Blood pressure of 120/80mmHg and a Heart Rate of 80bpm. She had no cardiac murmurs or signs of heart failure. Her prior ECGs revealed Premature Ventricular complexes while the recent one had normal sinus rhythm. Her Echocardiogram was normal except for a Left ventricular diastolic dysfunction. In view of her high pre-test probability for Coronary Artery Disease (CAD), she was taken up

for a Treadmill test(TMT) after holding on her beta-blockers for 48 hours.

Her ECG tracings showed a normal sinus rhythm with baseline HR of 90bpm. (fig. 1A) After 1 minute 14 seconds into Stage 1 of Bruce protocol, her ECG converted to a complete Left Bundle Branch Block pattern at HR of 121bpm which was 81% of her maximum predicted heart rate. (fig.1B)



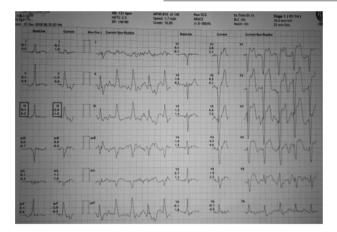


Figure 1: A) Electrocardiogram showing normal sinus rhythm with a baseline heart rate of 90 b/min. B) Electrocardiogram after 1 minute 14 seconds into Stage 1 of Bruce protocol, showing a complete Left Bundle Branch Block pattern at a heart rate of 121 b/min.

She was asymptomatic and exercise was continued. Target Heart Rate was achieved and exercise was stopped in early Stage 2 at 3 minutes and 6 seconds of exercise due to fatigue and shortness of breath. Her BP at Peak exercise was 140/80mmHg. The LBBB persisted for a little over 1 minute into recovery with a decrease in QRS width from 120ms during peak exercise to 80ms at 92bpm (fig.2) finally reverting to baseline morphology at 85bpm after 2 minutes into recovery. A few ventricular premature contractions were noted during the recovery period thereafter.

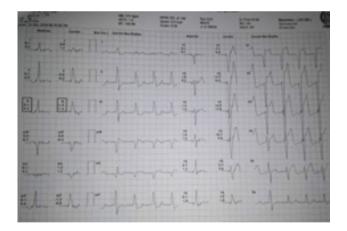


Figure 2: Electrocardiogram showing persistence of LBBB for over 1 minute into recovery with a decrease in QRS width from 120ms during peak exercise to 80ms at 92b/min.

Because of the ECG changes on TMT, Coronary Angiogram (CAG) was undertaken which revealed a 70% stenosis in the first diagonal branch of Left Anterior Descending Artery (LAD) and a 40% lesion in proximal Right Coronary Artery (RCA) with minor plaques in mid LAD and Left Circumflex artery (LCx). She was planned for conservative management for her CAD and underwent exploratory laparotomy with intraoperative betablocker and nitroglycerine to control her HR and BP. The post-operative period remained uneventful and the patient was started on a regimen of antiplatelet, statins, and beta-blockers after she regained her bowel movements.

### DISCUSSION

Lewis et al first described transient BBB in 1913 in a 32-year-old bookbinder who had been experiencing shortness of breath for six months before seeking medical advice. The observation of Right Bundle Branch Block (RBBB) was presumed to be due to an infection invading the myocardium as the patient was febrile during the observation. The RBBB had subsided the next day and reverted to physiological type with subsidence of the fever soon after that. In 1976, Vieweg et al described a 46-year-old man who developed rate-dependent LBBB and angina pectoris with effort reproducible with treadmill exercise testing who had a normal coronary angiogram.

A triad of exertional chest discomfort, transient rate-dependent LBBB and normal coronary arteries were reported in 7 consecutive patients by Virtanen et al in 1982 which noted that the onset of pain was always abrupt and took place simultaneously with the appearance of LBBB induced by physical exercise in all the patients along with local non radiating nature of the pain, palpitation and walk through phenomenon.<sup>3</sup>

Vasey in 1985 concluded in his study that patients developing rate-dependent LBBB during treadmill tests who present with atypical chest pain are significantly less likely to have CAD than patients presenting with classic angina. Exercise-induced LBBB at higher heart rates of >125bpm correlated with the presence of normal coronaries regardless of patient presentation. In his study 9 out of 18 patients with angina who developed chest pain and LBBB during exercise had normal coronary arteries. But Hertzeanu et al concluded in their 1992 study that an EI-LBBB at a heart rate below 125 bpm does not by itself constitute a sign of CAD. 9

Heinsimer in 1987 followed 15 patients who developed exercise-induced LBBB, 7 who had normal coronaries and 8 with CAD for an average of 6.6 years. 8 of them developed permanent LBBB, seven of these eight had underlying CAD. During his follow up, 4 patients had died, 3 had significant CAD and 1 had depressed LV function. He concluded that the development of permanent LBBB in patients with exercise-induced LBBB is related to the presence or absence of underlying CAD or myocardial disease. In the absence of underlying heart disease, patients who present with LBBB did not progress to conduction disturbances and the prognosis was excellent. 10

Bounhoure et al in 1991 observed 332 complete BBB during 16,500 exercise stress tests for 15 years. There were 7 RBBB and 25 LBBB cases in his study. All the patients underwent coronary angiogram. He concluded that complete RBBB appearing during exercise stress testing was constantly associated with atherosclerotic coronary artery disease. The predictive value of complete LBBB on effort was 72% and complete LBBB occurring at HR <120bpm was frequently associated with proximal stenosis of the LAD artery.<sup>2</sup> Similar conclusion was also given by Williams et al in 1988. The study followed 37 EI-LBBB patients and 13 EI-RBBB patients. Significant CAD was diagnosed in 70% of EI-LBBB patients and 100% of RBBB. Data demonstrated a high incidence (85%) of LAD lesion and a 46% incidence of coronary events. 11 This finding can be corroborated by the incidence of the 70% stenosis noted in the first diagonal branch of LAD in our case who developed EI-LBBB at a heart rate of around 120 bpm

as well without typical angina chest pain.

Neuss, Thormann, and Schlepper in their 1974 publication on electrophysiological findings in frequency-dependent LBBB suggest the underlying mechanism of the observed phenomena might be a frequency-dependent reduction of the upstroke velocity of action potentials. <sup>12</sup> Narula O S first suggested that LBBB and/or axis deviation can be the result of a focal lesion or altered refractoriness in the Bundle of His which could be normalized by stimulation distal to the lesion. <sup>13</sup> Recent studies have shown that His Bundle Pacing (HBP) induces significant QRS narrowing in most patients with LBBB and even normalization in patients with shorter baseline QRS duration <sup>14</sup> supporting the theory of functional longitudinal dissociation in the His Bundle. HBP has also been shown to effectively alleviate symptoms in exercise-induced painful LBBB<sup>15</sup> which suggests a pathology in the His bundle with altered refractoriness in a rate dependent mechanism.

In 2002, Candell et al<sup>4</sup> reported reviewing 9318 exercise stress studies which included 20 patients with EI-LBBB in which CAG had been performed. 8/20 had normal coronaries and 12 had CAD. Permanent LBBB appeared in 5 patients who had normal coronary arteries but no deaths were reported in this group. In the group who had CAD, there were 3 deaths and 2 patients had acute myocardial infarction during follow up. One patient in each group

developed AV block and required pacemaker implantation. They concluded that in contrast with LBBB and CAD, the prognosis in patients with painful LBBB and normal CAG is good. However the development of permanent LBBB is frequent. AV block, although rare, may occur irrespective of underlying coronary artery status.<sup>4</sup>

### **CONCLUSIONS**

Exercise-induced Left bundle branch block is more common than the right bundle branch block and around 70% of EI-LBBB and 100% of EI-RBBB have been associated with significant coronary artery disease. If these abnormalities in conduction are detected on exercise stress tests, coronary angiogram should be undertaken to determine the presence and extent of the lesion. In the event of painful EI-LBBB and normal CAG, early intervention with His Bundle Pacing can be explored. Despite the good prognosis associated with exercise-induced LBBB without CAD, some patients can develop an AV block. Therefore all patients regardless of CAD status should be closely followed up for possible acute coronary syndrome, development of permanent LBBB, and/or AV block in the future which can develop even years after the initial diagnosis.

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