IMAGES IN CARDIOLOGY

Left Bundle Branch Block (LBBB) Induced Angina / Painful LBBB Syndrome

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Abstract

A case of painful left bundle branch block (LBBB) syndrome is presented where anginal symptoms developed during periods of rate-dependent LBBB associated with stress or exertion. Rhythmos 2020; 15(4):78-79.

Key Words: left bundle branch block; painful LBBB; LBBB-induced angina; dyssynchrony

Abbreviations: CAD = coronary artery disease; ECG = electrocardiogram; LBBB = left bundle branch block

A 66-year-old gentleman, ex-smoker, with history of hypertension and hypercholesterolemia under treatment, was recently admitted to another hospital for symptoms of unstable angina. The electrocardiogram (ECG) on admission showed a left bundle branch block (LBBB) (Fig. 1). A myocardial infarction was ruled out by cardiac enzyme assay. He reported experiencing angina-like symptoms during periods of exertion and stress. His medications included metoprolol 25 mg bid, atorvastatin 40 mg qd, ramipril 2.5 mg qd and aspirin 100 mg qd. An echocardiographic examination was reported normal with a left ventricular ejection fraction of 60%.

He was thus referred for coronary angiography, which showed only mild atherosclerotic plaques noted in the mid segment of a dominant left circumflex coronary artery. However, during the procedure he was noted to have a rate-dependent LBBB and when the sinus rate was >80 bpm he complained of angina (Fig. 2); symptoms resolved when the heart rate dropped below 80 bpm when he was also noted to normalize the QRS width (Fig. 3). He also had an HV interval measured, which was found to be at 40 ms during the narrow-QRS rhythm and at 70 ms during LBBB. Infusion of small dose of isoproterenol 1 mcg/min increased the heart rate to 90 bpm that promptly provoked anginal symptoms, which resolved upon discontinuation of isoproterenol and heart rate lowering to <77 bpm.

Prior ECGs when patient was free of symptoms had shown a narrow QRS at lower sinus rates (~60 bpm) (Fig. 4).

The patient was re-assured about his coronary anatomy, was advised to control coronary risk factors and was prescribed higher doses of beta-blocker therapy to keep lower heart rates where there was no LBBB noted; ranolazine was also added to his regimen to possibly target regional coronary blood flow in areas of LBBB-induced myocardial ischemia. With this regimen the patient remained symptom-free at a 3-month follow-up.

LBBB, apart from heart failure, can also cause angina-like symptoms. Painful LBBB syndrome is a rare syndrome of angina-like chest pain caused by intermittent LBBB in the absence of significant coronary artery disease (CAD). The syndrome has been previously reported and attributed to disordered afferent neural network responsible for introception (awareness of heartbeat) that...
may be abnormally activated during aberrant ventricular conduction in some patients, or to disordered left ventricular activation causing dyssynchrony and ensuing ischemia. The issue of true ischemia has been refuted by some investigators. On the other hand, patients with LBBB often show abnormal images on exercise thallium scintigraphy without evidence of significant CAD. Investigators have suggested that abnormal perfusion images are more common in the septum in patients with LBBB, partially caused by impaired septal wall thickening during systole; abnormal wall motion may reduce coronary blood flow to the septum with ensuing ischemia; others have argued for impaired systolic thickening and augmented intramyocardial pressure, rather than ischemia, explaining the septal perfusion defect. Another possible etiology of LBBB-induced chest pain and ischemia has been reported and relates to the association of LBBB with the coronary slow flow phenomenon. Of course, it appears plausible that the painful LBBB syndrome may coexist with CAD, in which cases symptoms may be accentuated during rate-dependent LBBB, however, the suspicion may be stronger if the chest pain persists after all suspect coronary lesions are successfully revascularized.

Due to intermittent rate-dependent LBBB causing the anginal symptoms, therapy often aims at heart rate slowing with use of beta-blocker and/or ivabradine to prevent rate acceleration and emergence of LBBB and development of symptoms, as was done in our case. Exercise training has also been recommended as a successful nonpharmacologic strategy that may delay the onset of rate-related LBBB and chest pain and avoid the need for beta blocker therapy. Other investigators have reported the "walk through" phenomenon in some of these patients.

If dyssynchrony is the culprit for the LBBB-induced angina, cardiac resynchronization therapy (CRT) might be of value and has occasionally been employed in patients with symptoms refractory to beta-blocker and/or anti-anginal therapy; however, this is a major procedure with its attendant cost and risks. Alternatively, His bundle or LBBB area pacing has been applied in similar cases and reported to resolve chest pain symptoms.

Finally, in a double-blind crossover study comprising 191 patients with stable CAD randomly assigned to a 4-period of placebo and ranolazine at various doses for 1 week each, it was shown that compared with placebo, ranolazine ameliorated exercise-induced ischemia. The authors concluded that the progressive magnitude of ischemia reduction on ranolazine was proportionally more substantial than the minor reductions in heart rate or rate-pressure product, suggesting that ranolazine's beneficial anti-ischemic mechanism could be likely primarily due to an improvement in regional coronary blood flow in areas of myocardial ischemia. This was the impetus to employ ranolazine in our patient in addition to beta-blocker therapy, since LBBB is known to induce regional dyssynchrony and possibly ischemia in the absence of coronary lesions.

References