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Bundle Branch Block: Right and Left Prognosis Implications

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Abstract

An increasing number of papers, most based on epidemiology, have shown a strong association between bundle branch block (BBB) and cardiovascular disease, more specifically hypertension, cardiomegaly, coronary artery disease, and heart failure. Left BBB (LBBB) has been associated with cardiovascular disease complications in a much larger number of cases if compared to right BBB (RBBB). RBBB is usually associated to benign findings in healthy and asymptomatic individuals; nevertheless it can indicate affection of the right side of the heart through cor pulmonale, myocardial ischemia/infarction, pulmonary embolism, myocarditis, or congenital heart disease.

LBBB and RBBB prognosis strength are taking new positions over the time. Both have shown its relation with patient's mortality and prognosis implications. Therefore, in this review, we intend to make an approach of the BBB and its prognosis relevance based on the latest studies about this role.

Physicians should keep their patients with heart failure, myocardial infarction, and other abnormalities associated with BBB, under watch. Even the asymptomatic ones with coronary risk factors, should be carefully followed, once they have been associated with an increased mortality if compared with those who are not affected by bundle branch blocks.

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Introduction

The bundle branch block (BBB) is an alteration of the ventricular conduction that may lead into a ventricular dyssynchrony and to heart failure (HF) [1]. Its usual to see these abnormal ventricular conduction with HF, since BBB and HF most of the time share the same ethyology [2]; about one third of the patients with this comorbidity have a BBB identified with by your QRS complex criteria, and most of the cases there is a left bundle branch block (LBBB) associated [3,4].

In the general population, BBB is not as common as in the population with HF. The LBBB in general population ranges from 0.1-0.8% [5], and the Right Branch Bundle Block (RBBB) is around 1.9-24.3 per thousand [6,7], and in the general population over 52 years old with any complete BBB had 3.7% of prevalence [7]. Besides, the prevalence rate rises and is strongly related with sex and age group (men and older individuals) [8-10].

Anatomy and physiology

It is well known that the intraventricular septum contains the his bundle who is most common divided in 3 original fascicles: the right pathway, with one branch; and the left bundle with 2 pathway, the left anterior fascicle and the left posterior fascicle [11,12]. In this review we adopted the usual presentation of trifascicular intraventricular conduction system, however the anatomical variances of more or less pathways doesn't change the physiological concept of the hemiblocks [12].

The left bundle branch (LBB) and the right bundle branch (RBB) does not trade stimuli by a mechanism between the right and the left pathways: a "physiological barrier". This barrier is very important to maintain the synchronism between the left and right ventricles and for the role of the BBB [13]. When a complete

BBB is established, the ventricular activation occurs firstly on the opposite side of the blocked way; initiating the depolarization on its septum and the ventricular mass [14]. Then, before completely activate the ventricle of the non-blocked bundle, the depolarizing wave overleaps the septum physiological barrier by the ordinary myocardial cells and reaches the Purkinje system and the septum, activating the remaining ventricle in an anomalous way and with an delay of 0.02-0.04s in the RBBB and over 0.06s for the LBBB [13,15].

Clinical significance

The BBB usually arises from a degenerative process of the heart's conduction system and it is substantiated by its close relation with cardiovascular diseases that degenerate any of the ventricles: HF, myocardial infarction (MI), cor pulmonale, Brugada's syndrome, or anything that alters the function of the ventricle. Although, it also can occur in patients without any underlying heart diseases [16-18]. Common causes of RBBB include MI, hypertensive heart disease, and pulmonary diseases, such as pulmonary embolism and chronic obstructive lung disease [16].

In patients who have pulmonary embolism, RBBB can be found in 6%-67% of the cases [19]; a new RBBB is also usually related to a larger anterior MI and it happens in 3%-7% of the MI cases [16,20]. In Brugada's Syndrome, the block of the right branch is one of the criteria whose underlying pathophysiology; seeming to be related with a defect in the cardiac sodium channel. The clinical significance of identifying this syndrome lies in its main cause of death: sudden cardiac death caused by ventricular tachycardia [16].

In LBBB, the most common causes include coronary artery disease, hypertension, and cardiomyopathy. LBBB also can be seen during cardiac pacing. The pacing wire usually abuts the right ventricle and induces an LBBB-like morphology on the ECG [17]. Among patients with chest pain and suspect of MI, LBBB ranges between 1% and 9%; LBBB mostly obscures acute MI diagnosis in the ECG criteria, masking the elevation of the S-T segment [17]. LBBB also has a close relation with HF, associated with approximately 25% of the cases, and it is known as a worsen factor of the left ventricular fraction ejection [2].

Diagnosis

ECG is considered the gold standard for noninvasive diagnosis of conduction disturbances and arrhythmias. Its sensitivity and specificity is higher for the diagnosis of arrhythmias and conduction disorders than for structural or metabolic changes [21]. RBBB, in most of the cases, has a pathological cause, but it can also be found in healthy individuals. On the other hand, LBBB is most commonly caused by coronary artery disease, hypertensive heart disease, or dilated cardiomyopathy. It is unusual for LBBB to exist in the absence of organic disease. So, in order to completely evaluate the patient for suspected associated abnormalities that may be the cause of BBB, physicians may run other tests, as physical examination, chest X-ray and echocardiography. Recently, Strauss et al. has suggested changes in the criteria for definition of LBBB. He considers that about one-third of patients diagnosed by current criteria, based on the duration of the QRS interval ≥120 ms, do not really have full LBBB **(Table 1)**. He's proved that with his new proposed criteria, specificity could rise to maximum.

Prognosis and Mortality

Left bundle branch block

An increasing number of papers, most based on epidemiology, have shown a strong association between LBBB and cardiovascular disease, more specifically hypertension, cardiomegaly, coronary artery disease, and heart failure. Left BBB has also been associated with more complications for cardiovascular disease than right BBB. The prevalence of LBBB is dependent on the population studied; it is lower than 0.5% in healthy young individuals and increases up to 25% in patients with chronic heart failure [23,24]. LBBB causes some damage on the mechanical function of the LV secondary to asynchronous myocardial activation, which sequentially, may trigger ventricular remodelation and bad prognosis.

Zannad et al. proposed a sequence for the development of HF in patients with LBBB: intra-ventricular asynchrony-reduced pump function-neurohormonal activation-asymmetric hypertrophy-dilatation [25]. The dyssynchrony has been shown to accelerate disease progression in HF. The more pronounced the dyssynchrony the more the mortality in individuals with HF. The Framingham Heart Study has shown that patients with acquired BBB were more likely to have, or to develop, advanced cardiovascular manifestations-especially the male population with LBBB [26]. Also, sudden death as the first manifestation of heart disease was 10 times higher in male individuals with LBBB than in those without the condition [27]. LBBB and an abnormal Q wave are risk factors of cardiovascular mortality in end-stage hypertrophic cardiomyopathy and provide new evidence for early intervention [28]. According to Khalil et al. Isolated LBBB occurring in the setting of young, clinically healthy men conveys a benign prognosis. However, in older patients, LBBB usually indicates an underlying progressive degenerative disease of the ventricular myocardium [29].

Some new studies have shown associations between LBBB and adverse prognosis in patients with preserved LV systolic function after myocardial infarction. Lewinter et al. affirm that univariable analysis showed that both LBBB and RBBB were associated with increased mortality compared with patients without BBB, and supports the association between LBBB and prognostic importance in patients with preserved LV systolic function. Lewinter et al. also brings up on his study that the prognostic importance of RBBB and LBBB adjusted for interactions with wall motion index was independent of infarct location, diabetes, gender, hypertension, renal function, COPD, and treatment with fibrinolysis. Sensitivity analyses showed a difference in mortality between 1, 5, 10, and 15 years follow-up of mortality between non-BBB, RBBB, and LBBB (LBBB with 47% in 1 year, 75% in five years, 86 in 10 years and 95% in 15 years). Overall, LBBB had the highest proportion of mortality during the whole follow-up period, with the rate of death increasing remarkably until 5 years, after which the trend slowed down.

Baldasseroni et al. in a study with 5517 patients, brings the idea that LBBB is an indicator of poor prognosis in MI and congestive HF mainly in short- and medium-term follow-ups. Recently, LBBB was demonstrated to be an independent predictor of mortality also in long-term (1 year) followed-up patients without severe HF after hospital admission of acute HF [30]. Just as Baldasseroni, some other authors also established relationship between LBBB and risk factors for mortality in the form of increased age, history of MI and HF, and in-hospital complications such as reduced LVEF, ventricular and atrial fibrillation.

Some have also been discussed in the literature about induced LBBB. Poels et al. brings that prognostic implication of TAVI-induced LBBB is an independent predictor of all-cause mortality. On their study the excess mortality in the LBBB group was mainly caused by an increase in fatal cardiovascular events, indicating that this might be caused by (dyssynchrony-induced) heart failure. Therefore, heart failure hospitalization should be considered an important study endpoint in TAVI-related research in addition to mortality [24].

Right bundle branch block

The RBBB was associated to a benign finding in healthy and asymptomatic individuals in the past [31,32]. Although, RBBB can indicate affection of the right side of the heart through cor pulmonale, myocardial ischaemia/infarction, pulmonary embolism, myocarditis, or congenital heart disease. And among patients whom undergo with HF and RBBB associated, new studies have shown worse prognosis and mortality than it in the past [33,34]. A recent study from Bussink et al. have warned us about the asymptomatic individuals with the RBBB condition. In this study, individuals with that condition were associated with approximately 30% increased mortality risk mainly due to Cardiovascular Diseases and increased risk of all-cause mortality and adverse cardiovascular outcomes with similar associations in both genders. On the other hand, the incidence of chronic HF, atrial fibrillation, or chronic obstructive pulmonary disease (COPD) was not different for the RBBB group when compared with normal individuals [34].

In the other hand, patients who have RBBB and hospitalized with systolic HF, showed the worst prognosis compared to groups with LBBB and no BBB in 48 months. At 4 years of follow up of 1,888 patients, mortality rates were highest in patients with RBBB (69%), intermediate in those with LBBB (63%), and lowest in those without BBB (50%, $p<0.001$). The results demonstrated a significant (36%) increased mortality risk in patients with RBBB compared to no BBB individuals ($p=0.002$).

In 2014, Supariwala et al. studied the correlation of Stress Echocardiography (SE) test with complete BBB, whether the result of the SE was normal or not and following up these patients for a long term period 9 ± 4 years. The mortality rates were 4.5%/year for patients with LBBB, 2.5%/year for patients with RBBB, and 1.9%/year for patients without BBB ($P<0.001$) for patients with abnormal SE test. Corroborating with Biagini et al. who has also determined the RBBB prognosis through SE test. In this study were followed up 163 RBBB patients, and after 4.3 years the mortality was of 57 deaths (35%), which 37 (23%) were caused by cardiac causes. And both studies reviled that the abnormal SE exam is a strong predictor of a subsequent cardiac event and worst prognosis in patients with RBBB [35,36].

In patients who had suffered of acute MI and have RBBB, the prognosis is really poor. A study with 6676 patients who experienced acute MI, 260 (4%) had RBBB and 39% of this group died at the first year, followed of 61% of the total in 5 years, 79% in 10 years and 89% in 15 years. If compared to LBBB with 47% in 1 year, 75% in five years, 86 in 10 years and 95% in 15 years, the difference between BBB groups was not significant [29].

Another recent study with MI compared the prognostic rate of new BBB and preexisting BBB with a control group. In this multicentre Cohort study, with 5,570 patients, revealed that a new BBB was associated worst prognosis if compared to preexistent ones. The new permanent RBBB group showed in a short-term (30 days), mortality of 62.7%, 2.01 on the calculated hazard ratio (HR) with adjustments (adjusted by age, gender, coronary risk factors, comorbidities, type of myocardial infarction, anterior location, Killip class, heart rate, systolic blood pressure, glycaemia on admission, peak of CK-MB, reperfusion and left ventricle ejection fraction) compared with the control (no BBB group); Vs. only 1.07 on the adjusted HR in the group of preexisting RBBB compared to the control. And in the long-term (7 years) mortality this difference rises. In this study a new permanent RBBB was declared as an independent mortality predictor and need to have its attention before infer the prognosis of a MI patient [37].

Treatment

Cardiac resynchronization therapy (CRT) has proved to be a very effective treatment for patients with depressed left ventricular (LV) function, symptomatic congestive heart failure (HF), and abnormal QRS width. It is able to induce LV reverse remodeling with improvement of LV function and reduction of heart failure symptoms, boosting not only quality of life and heart function, but also remarkably changing prognosis, reducing HF-related hospitalization and mortality [23,24].The study of individuals with LBBB and its mechanisms of intraventricular conduction abnormalities brought the idea of CRT as a number one therapy for symptomatic patients. Some predictable changes in the LV activation sequence, such as abnormal activation of the septum and markedly delayed activation of the lateral LV with dyssynchronous electromechanical activation leads to increased cardiac work, less efficient cardiac contraction, and lower cardiac output [38,39].

The knowledge that a device can, electric and mechanically, change preactivation of the late-activating LV region and improve almost all LBBB complications has set CRT as a prerequisite for successful clinical response in LBBB patients [38-40].

Although the use of CRT is deeply set for patients with LBBB, it's use in individuals with RBBB remains unclear. There's not enough evidence to prove if whether outcomes with RBBB are worse because of the compounding effects of adverse predictors and disease severity or decreased efficacy of (or maybe harm from) CRT [38].

Table 1 ECG criteria for diagnosis of RBBB, LBBB and Strauss strict criteria for LBBB [22].

Diagnostic criteria for right bundle branch block	Diagnostic criteria for left bundle branch block	Diagnostic Strict Criteria for left bundle branch block by Strauss
QRS duration >0.12 s	QRS duration of >0.12 s	QRS duration ≥0.14s for men and ≥0.13s for women
A secondary R wave (R') in V1 or V2	Broad monophasic R wave in leads 1, V5, and V6	QRS duration ≥0.14s and mid-QRS (beginning after 40 ms) notching/slurring in at least two of the leads V1, V2, V5, V6, I, and/or aVL]
Associated feature	Absence of Q waves in leads V5 and V6	QS or rS in V1
ST segment depression and T wave inversion in the right precordial leads	Associated features	
	Displacement of ST segment and T wave in an opposite direction to the dominant deflection of the QRS complex (appropriate discordance)	
	Poor R wave progression in the chest leads	
	RS complex, rather than monophasic complex, in leads V5 and V6	
	Left axis deviation-common but not invariable finding	

Conclusion

Both LBBB and RBBB prognosis strength are taking new positions over the time. With this review, we intend to warn physicians to keep their patients with HF, MI, and other abnormalities associated with BBB, under watch. Even the asymptomatic ones with coronary risk factors, should be carefully followed, once they are associated with increased mortality compared with those who do not have BBB [29,34-37]. Future perspectives of changes in the clinical practice await in this role.

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