CASE REPORT

Rate-Dependent Bundle Branch Block: Not Only Phase 3 Exists

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ABSTRACT

Rate-dependent bundle branch blocks are often related with increased heart rate (tachycardia-dependent or phase-3 bundle branch block). Less often, they are caused by heart rate reduction (bradycardia-dependent or phase-4 bundle branch block). Phase-3 block is the most common type of paroxysmal intraventricular conduction disturbances documented in several publications. However, few articles describe phase-4 bundle branch block, and both mechanisms rarely coexist in the same patient. We report the case of a patient admitted with acute myocardial infarction, presenting with both tachycardia- and bradycardia-dependent paroxysmal intraventricular conduction disturbances.

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Key words > Bundle branch block; Bradycardia; Tachycardia; Acute myocardial infarction.

Abbreviations >

AMI Acute myocardial infarction	**HR** Heart rate
CPK Creatine phosphokinase	**IVCD** Intraventricular conduction disturbances
ECG Electrocardiogram	**LBBB** Left bundle branch block

INTRODUCTION

Rate-dependent bundle branch blocks are often related with increased heart rate (HR) (tachycardia-dependent or phase-3 bundle branch block). Less often, they are caused by heart rate reduction (bradycardia-dependent or phase-4 bundle branch block).

Phase-3 block is the most common type of paroxysmal intraventricular conduction disturbances (IVCD) documented in several publications. However, few articles describe phase-4 bundle branch block, and the coexistence of both mechanisms in the same patient has rarely been described.

We report the case of a patient admitted with acute myocardial infarction (AMI), presenting both paroxysmal tachycardia- and bradycardia-dependent IVCD.

CASE REPORT

A 76 year-old man with hypertension, diabetes mellitus and dyslipidemia, was referred to our hospital due to chest pain consistent with unstable angina. He was asymptomatic upon arrival. The first ECG showed sinus rhythm with HR around 95 bpm (RR interval of 620 ms), PR interval of 200 ms and left bundle branch block (LBBB), without established criteria for AMI with ST-segment elevation (Figure 1A). For these reasons, and because the patient remained asymptomatic, we decided to transfer the patient to the coronary care unit under acute medical treatment, and to schedule a coronary angiography within the next 48 hours. Troponin T was slightly elevated (0.4 ng/ml), while CPK was normal.

Heart rate-dependent ECG changes were observed in the following hours prior to the angiography. Thus, with a HR of 60 bpm (RR: 1000 ms) the LBBB disappeared, showing a narrow QRS of 80 ms (Figure 1B). Later, when the patient experienced an episode of asymptomatic bradycardia of uncertain origin, with a HR of 35 bpm (RR: 1620 ms), the QRS widened again (Figures 1B & 2). All the beats showing LBBB during this transient HR reduction were preceded by P waves and PR intervals similar to the other beats. The patient did not experience chest pain or ST-segment variations suggestive of ischemia-dependent LBBB during bradycardia.

The coronary angiography showed three-vessel disease with non-revascularizable anterior descending and right coronary arteries. An angioplasty was performed and a drug-eluting stent was implanted on a subtotal occlusion in the circumflex artery. No complications or new episodes of IVCD were observed in ECG monitoring following revascularization.

Phase-3 IVCD seems to occur due to critical shortening of the RR interval. This is the most common presentation of paroxysmal IVCD. The electrophysiological

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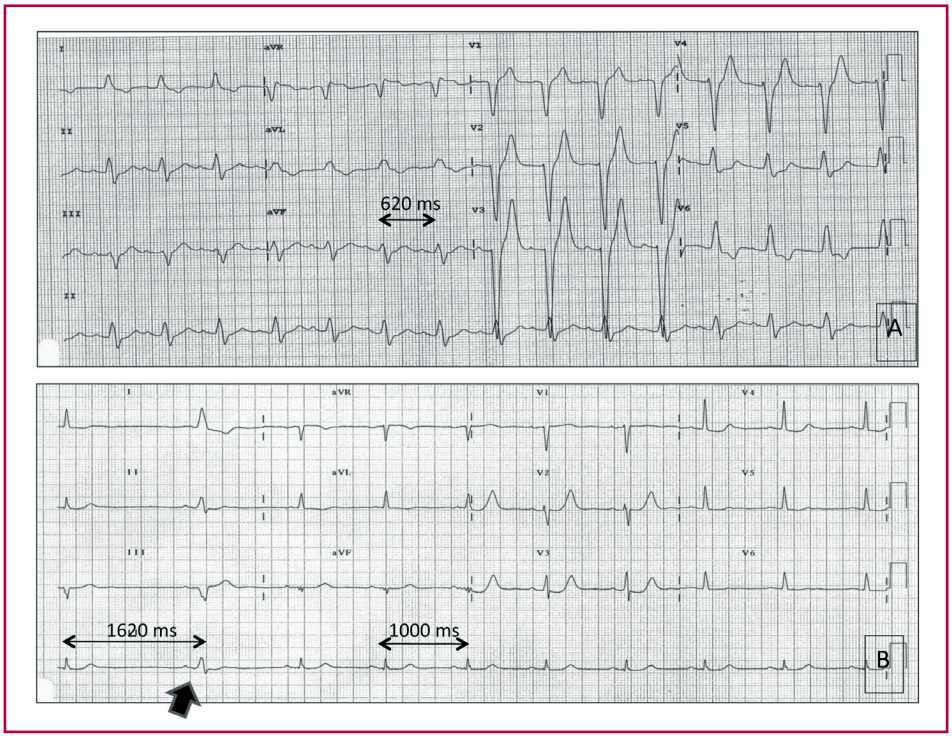

Fig. 1. A: First electrocardiogram (ECG) showing sinus rhythm with HR around 95 bpm (RR: 620 ms), PR of 200 ms, and left bundle branch block (LBBB).
B: ECG showing sinus rhythm with HR 60 bpm (RR: 1000 ms), PR of 200 ms and no LBBB (QRS: 80 ms). The second beat (arrow), following the longest RR interval (1620 ms), shows the same LBBB as the first ECG.

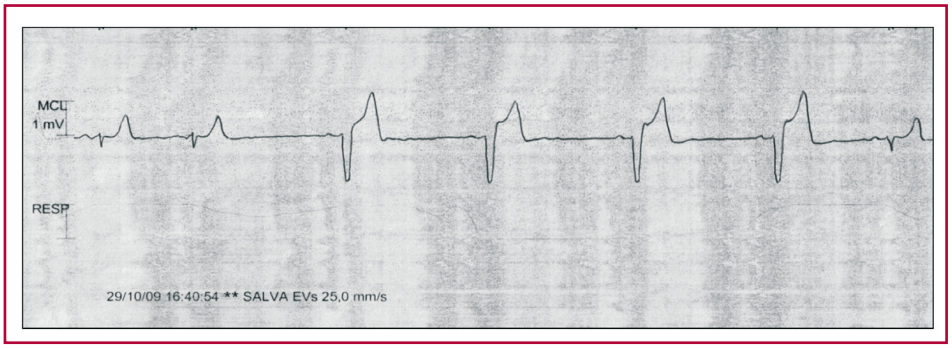

Fig. 2. Telemetry registry with heart rate around 35 bpm, shows a similar phenomenon with a new complete LBBB.

factors causing it are the length of the branch refractory period, its variation compared with the previous cycle, and HR. (1) Although we were unable to document the exact moment of LBBB disappearance with HR reduction, it is reasonable to assume that the QRS narrowed at some point between 95 bpm (Figure 1A) and 60 bpm (Figure 1B). It should also be pointed out that the occurrence of LBBB in case of slightly accelerated HR is a rare finding, although in some cases similar to the one presented here, it has been reported as a phenomenon associated with myocardial ischemia. (2)

According to Singer's works, bradycardia-dependent IVCD is attributed, (3) to the inherent capacity of one or another branch to spontaneous diastolic depolarization ("spontaneous diastolic depolarization" or "diastolic hypopolarization") during phase 4 of the action potential. (4) The next impulse from the atria or the atrioventricular node would arrive at the branch during the refractory period, in which the cell would be hypopolarized, not reaching the triggering threshold. (2) El-Sherif confirmed the relationship between the increased slope of diastolic depolarization and decreased responsiveness of the cell membrane associated with a variation of the threshold potential towards zero. (5)

A continuous electrocardiographic monitoring in our patient showed QRS widening when HR was reduced to 35 bpm (Figure 2). All wide QRS beats were preceded by a P wave and a reasonable PR interval, similar to previous and posterior beats, so we can presume they were conducted from the atria to the ventricles. According to the criteria proposed by Massumi (2), there is no other reasonable explanation for this phenomenon, so it can be assumed these ECGs show the rare finding of a phase-4 LBBB shortly after a phase-3 LBBB.

The most common context in which these disorders occur is acute myocardial ischemia, and it is predictive of bad prognosis in AMI. (6, 7) Therefore, although many factors of poor AMI outcome have been documented, knowledge of rate-dependent IVCD may provide additional predictive information to perform a

more aggressive management in these patients as well as achieving better differential diagnosis from other arrhythmic complications.

RESUMEN

Bloqueo de rama frecuencia-dependiente: no sólo existe la fase 3.

Los bloqueos de rama frecuencia-dependiente suelen estar relacionados con el aumento de la frecuencia cardíaca (bloqueo de rama taquicárdico-dependiente o en fase 3). Con menor frecuencia son causados por la reducción de la frecuencia cardíaca (bloqueo de rama bradicárdico-dependiente o en fase 4). El bloqueo en fase 3 es la presentación más frecuente de los trastornos paroxísticos de la conducción intraventricular documentados en varias publicaciones. Sin embargo, no son tan numerosos los artículos que describen el bloqueo de rama en fase 4 y, raras veces, ambos mecanismos coexisten en el mismo paciente. Se presenta un paciente ingresado con un infarto agudo de miocardio en el que se detectaron trastornos de conducción intraventricular paroxísticos tanto taquicárdico-dependientes como bradicárdico-dependientes.

Palabras clave > Bloqueo de rama; bradicardia; taquicardia; infarto agudo de miocardio.

Conflicts of interest:
None declared.

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