

Electro-vectorcardiographic demonstration of rate-independent transient left posterior fascicular block

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Abstract

Left posterior fascicular block (LPFB) is a rare intraventricular conduction disorder of rare occurrence, especially as an isolated entity. Its transient form is even rarer and may be rate-independent or rate-dependent intermittent LPFB (phase 3 block, tachycardia-dependent and phase 4 block or bradycardia-dependent). We present a case of a young adult male whose baseline ECG/VCG showed the typical LPFB pattern. A treadmill stress test revealed rate-independent intermittent LPFB with random occurrence. Imaging exams ruled out structural heart disease. To our knowledge, this is the first case in the literature of a rate-independent intermittent LPFB with no underlying structural heart disease.

Keywords: Transient left posterior fascicular block; Isolated left posterior fascicular block; Rate-independent intermittent left posterior fascicular block.

Case report

A 33-year-old male was referred to our hospital for a periodic preventive checkup.

Medical history: recent onset mild hypertension controlled with thiazide.

Family history and physical examination: nothing worthy of note.

Due to baseline ECG findings (Figure 1), a vectorcardiogram (VCG) (Figure 2) and treadmill stress test (Bruce protocol, submaximal) (Figure 3) were requested. The VCG revealed asymptomatic random rate-independent intermittent left posterior fascicular block (LPFB).

Two sequential transthoracic echocardiograms were performed. The first one included measures of diastolic function and the second one also strain rate imaging. Echo strain and strain-rate imaging (deformation imaging) for assessment of myocardial function was necessary to rule out early, subclinical myocardial disease due to its ability to differentiate between active and passive movement of myocardial segments, to quantify intraventricular dyssynchrony and to evaluate components of myocardial function, such as longitudinal myocardial shortening.

Cardiac magnetic resonance imaging with late gadolinium enhancement was normal.

Figure 1

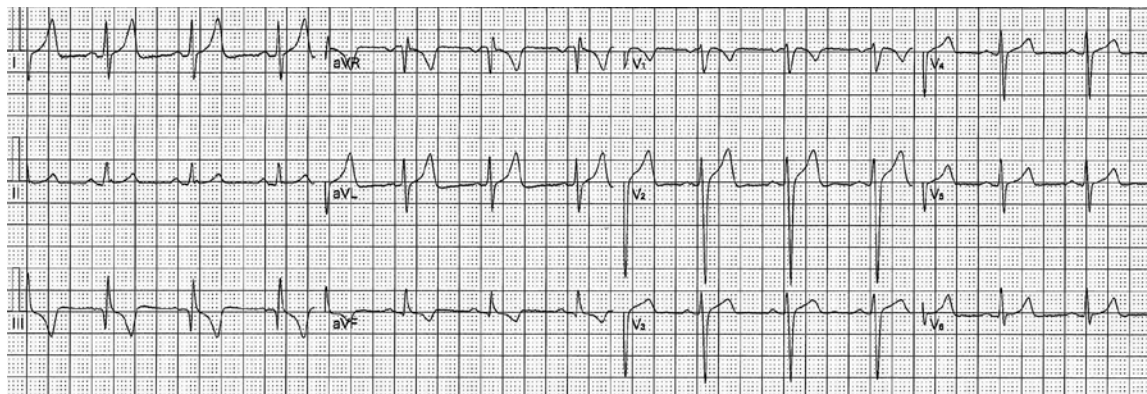


Figure 2

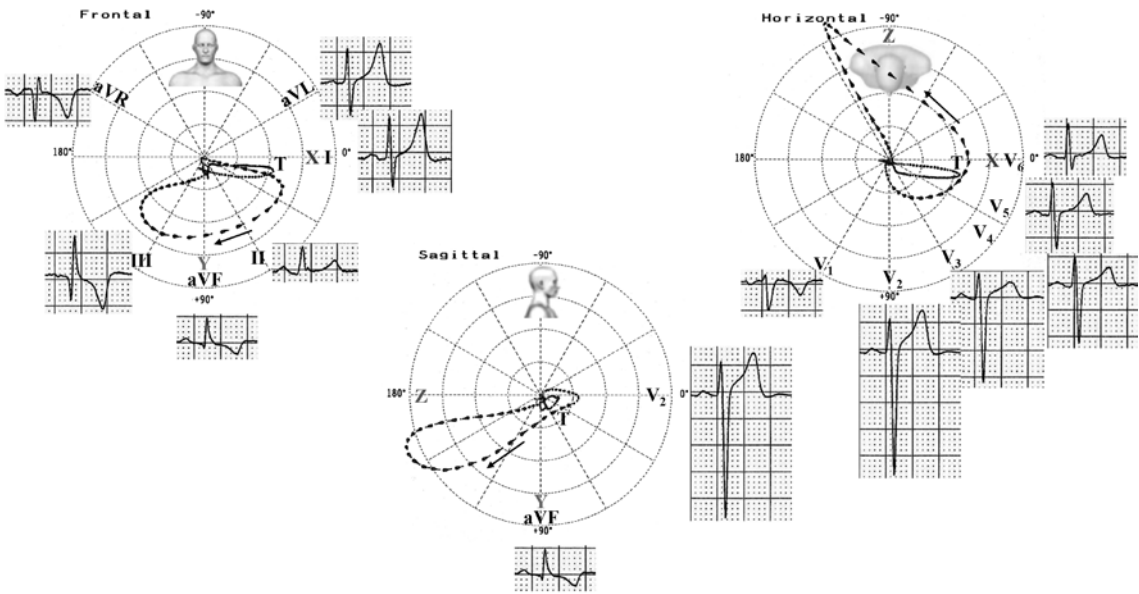
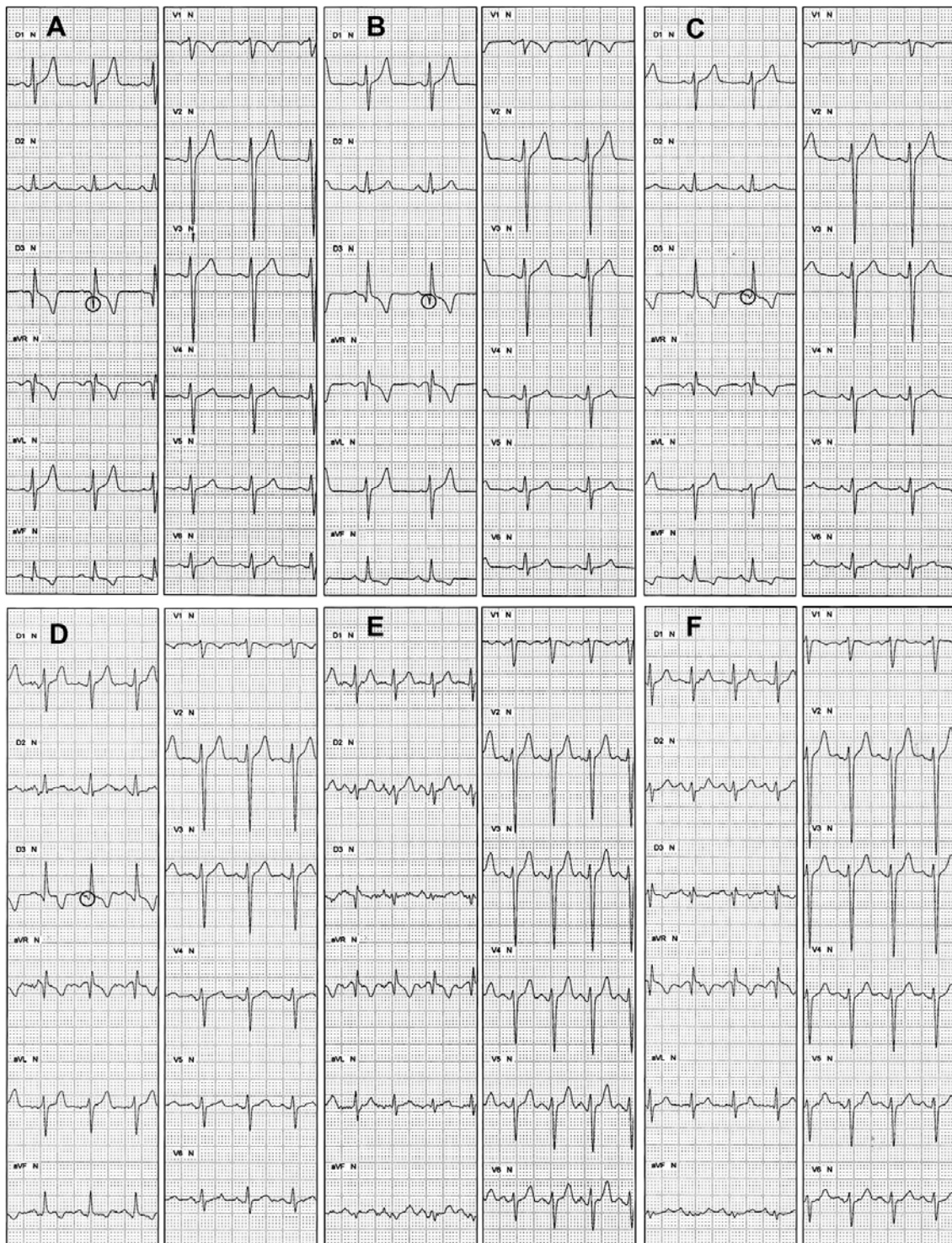


Figure 3



Discussion

Isolated LPFB in the absence of associated right bundle branch block is a very rare electrocardiographic finding. The lower vulnerability of the left posterior fascicle (LPF)

compared to the left anterior fascicle is due to anatomical (greater diameter of the LPF) and electrophysiological factors (shorter duration of the LPF action potential), irrigation (dual blood supply) and localization (the LPF runs through a more protected area, with less mechanic impact or pressure). The term transient LPFB is an aberrant conduction not due to previous QRS abnormalities, accessory pathway conduction, or unwanted drug effects (Issa, Miller, & Zipes, 2009), and transient right axis deviation with a LPFB pattern has been reported during acute myocardial infarction or during exercise stress test in cases of two- or three-vessel or left main disease (Demoulin & Kulbertus, 1979; Madias & Knez, 1999). Transient LPFB during Prinzmetal's angina culminating in acute inferior myocardial infarction was described by Ortega-Carnicer et al (Ortega-Carnicer, Garcia-Nieto, Malillos, & Sanchez-Fernandez, 1983), acute anterolateral myocardial infarction (Ogawa, Kimura, Okada, Ogino, & Katayama, 1976) and induced by exercise in the setting of severe coronary artery disease (Bobba, Salerno, & Casari, 1972).

Transient or intermittent blocks can occur at any level of the His-Purkinje system and may be due to different mechanisms.

According to steadiness, LPFB could be: a) Permanent (most frequent), b) Intermittent or second degree LPFB. The periods of alternating LPFB result from a changing relationship between conduction velocity and refractory period.

Transient LPFB could be:

A) Rate-dependent intermittent LPFB

I. Phase 3 block (tachycardia-dependent) (Chimienti, Salerno, & Tavazzi, 1981).

This is due to electrophysiological modification of tissue during the effective refractory period and can be a physiological or pathological phenomenon. A special form of this block is acceleration-dependent block, which is due to

changes in the heart rate. Phase 3 block is believed to express a pathological increase in the duration of the recovery period of the bundle branch or fascicles.

- II. Phase 4 block (bradycardia-dependent or pause-dependent) is almost always pathological. It occurs after the end of the refractory period due to decreased membrane potential, because of increased His-Purkinje automaticity or partial depolarization of the myocardial lesion. Phase 4 block was best explained on the basis of enhanced phase 4 depolarization of the bundle branch system or fascicles, with inability of excitation if the cardiac cycle is particularly prolonged.

Note: phase 3 and phase 4, often coexist. Supernormal conduction is in relation with the presence of a phase of supernormal excitability experimentally demonstrated in the late phase of repolarization of cardiac cells, and supernormal and alternating conduction are related phenomena. The evidence on ECG of alternating conduction through a pathological branch or an atrioventricular accessory pathway can be considered as a marker of the presence of supernormal conduction through the structure.

B) Rate-independent intermittent LPFB

Mechanisms: Mobitz type I; Mobitz type II by Wenckebach phenomenon; and by significant hypopolarization. Hidden conduction or heart rate-independent, which is defined as the propagation of an impulse within the specific conduction system and can only be recognized by its effect on the impulse, the interval, or the following cycles (Demoulin & Kulbertus, 1972). As indicated by its name, this phenomenon cannot be observed on surface ECG. According to Moe et al. (Moe, Childers, & Merideth, 1968) there are alternative explanations for electrocardiographic abnormalities suggestive of supernormal conduction or the Wenckebach phenomenon. The gap phenomenon in electrophysiology arises from the differences in refractory periods at two or more levels

of the AV conduction system (Gallagher et al., 1973). One explanation for the gap phenomenon is conduction delay in the proximal part of the AV conduction system, causing recovery of its more distal portion. Moe and coworkers defined the conduction gap as a period of the cardiac cycle during which premature atrial beats did not propagate to the ventricles, while atrial impulses of greater and lesser prematurity were able to activate the ventricles. The conduction “gap” can occur between any two portions of the AV conducting system from the atrium to the distal Purkinje system. It is due to functional differences of conduction and/or refractoriness in at least two regions of the conducting system. To support the diagnosis of supernormal conduction it is indispensable to exclude the presence of a conduction gap that may create the conditions for a better impulse propagation of premature beats as they arrive to the bundle branches after the end of the refractory period. In the present case, it is not possible to describe the exact mechanism of the observed ECG changes with certainty; an electrophysiological study would have been necessary to be absolute certain about a variable intrahisian conduction delay that could have resulted in different aberrant QRS complexes as well as normally conducted beats mimicking supernormal conduction.

Conclusion

We present a very unusual case of a rate-independent transient LPFB in a patient without structural heart disease. It is important to point out that the block did not appear during the effort phase, being of an apparently random character with variable degrees of LPFB.

Conflicts of interest

None.

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Figure legends

Figure 1 Basal ECG

QRS duration <120 ms, QRS axis $+85^\circ$, rS pattern in leads I and aVL, qR pattern in III and aVF, $q_{III} > q_{aVF}$ (in LPFB, the q wave in III is always greater than the q wave in II and aVF), time of appearance of R-wave apex in aVF ≤ 35 ms, rS pattern in V_1 and V_2 , very deep S wave in V_2 - V_3 by posterior dislocation to the right of the terminal forces, disappearance of q wave in V_5 - V_6 , and R-wave peak time ≥ 45 ms in these leads. Conclusion: LPFB.

Figure 2 VCG/ECG correlation

Frontal plane: Initial 10 to 20 ms vector heading superiorly and to the left (near -45°) with delay (initial 10 to 25 ms); broad QRS loop, with clockwise rotation; maximal vector $\approx +110^\circ$; almost all the QRS loop located below the X orthogonal line predominantly in the inferior quadrants; QRS loop duration 110 ms; T loop with clockwise rotation, heading below and to the left near $+10^\circ$.

Horizontal plane: Initial 10 to 20 ms vector heading to the front; QRS loop shape very similar to type C right ventricular hypertrophy; QRS loop of counterclockwise rotation; greater area of QRS loop located in the left posterior quadrant; maximal vector of QRS around -80° ; final portions with delay located in the right posterior quadrant ($>20\%$ of the area of the QRS loop located in the right posterior quadrant); T loop minimally directed to the front and the left ($+10^\circ$) with clockwise rotation.

Right sagittal plane: Initial 10 to 20 ms vector heading to the front and above with delay; most of the QRS loop located in the inferoposterior quadrant; QRS loop of clockwise rotation; maximal vector around $+160^\circ$; end QRS delay; T loop heading to the front and below with clockwise rotation.

Figure 3 Treadmill stress test sequence

Variable degrees of LPFB are observed: major degree at rest (**A**); lower degree during pre-stress laying down (**B**); and minimal degree during pre-stress standing (**C**). Note the progressive decrease of the q-wave depth in lead III indicating variable degree of LPFB (circles). During stress test at 2.7 km/h (**D**) LPFB is present. During stress test at a heart rate of 128 bpm (**E**) and after the stress test at 109 bpm (**F**) LPFB disappears. The PR interval was normal during the test. No significant ST changes were observed.

Note: the $\hat{S}\hat{A}QRS$ in A is $\approx +125^\circ$ and in C $\approx +118^\circ$, which would indicate a higher degree of LPFB in C than in A. However, when we analyze the depth of the q wave in III, q_{III} in A $>$ q_{III} in C, which indicates that the initial portions of ventricular activation are more significant in A than in C, consequently, a higher degree of LPFB in A in the initial phase and a higher degree in C in the mid-final phase.