CASE REPORT

Transient Left Septal Fascicular Block: An Electrocardiographic Expression of Proximal Obstruction of Left Anterior Descending Artery?

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left septal fascicular block; proximal left anterior descending obstruction; transient fascicular block;
prominent anterior QRS forces

INTRODUCTION

Trifascicular activation of the left ventricle is a controversial concept. An important element supporting the existence of an anatomical left septal fascicle is the demonstration of the transient (or intermittent) appearance of the left septal fascicular block (LSFB). There are few published cases documenting intermittent LSFB, either in association with myocardial ischemia^{1–4} or as a consequence of a bradycardia-dependent (phase 4 block) mechanism.⁵ The following clinical vignette reveals an electrocardiographic phenomenon—transient prominent anterior QRS forces (PAF) likely related to a LSFB—occurring in the setting of a high-grade lesion of the proximal left anterior descending (LAD) artery.

CASE DESCRIPTION

A 78-year-old Caucasian female patient was diagnosed in the emergency room with an acute inferoposterior ST-elevation myocardial infarction

(STEMI). Within the first 12 hours of chest pain onset, a standard 12-lead ECG was done (Fig. 1A). The patient was immediately referred to the cardiac catheterization lab for consideration of primary percutaneous transluminal coronary angioplasty (PTCA). Significant lesions in the proximal right coronary artery (RCA) and in the LAD were found. Of note, the LAD lesion was extending over segments located both proximally and distally to the first septal perforator branch (S₁). A two-staged stent-assisted angioplasty strategy was adopted. First, a stent was successfully deployed in the RCA, which was thought to be the infarct-related artery (Fig. 2). The second angioplasty—coronary recanalization of the LAD—was to be done on an elective basis. A few days after discharge, the patient presented with another episode of typical angina chest pain, with a completely different 12-lead ECG (Fig. 1B). The new ECG changes seen in Figure 1B resolved (Fig. 3A) after successful PTCA to the LAD (Fig. 3B).

How can be explained the transient ECG changes?

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Figure 1. (A) ECG on admission before PTCA to the proximal RCA. (B) ECG 3 days post-PTCA to the RCA. New onset chest pain. (Refer to text for details).

DISCUSSION

The electrophysiological mechanism that would best explain the ECG changes between Figures 1A and B is the transient manifestation of PAF secondary to a LSFb.

LSFB is a conduction disorder whose electrocardiographic pattern is not yet universally accepted in the literature. A recent international consensus stresses that the most frequently accepted criterion for identifying LSFb is the transient or permanent appearance of PAF, defined as "RS in leads V₁–V₂ with RS in lead V₆."⁴ However, the identification of PAF warrants the search for alternative causes

such as: right bundle branch block (either troncular or peripheral), ventricular conduction disturbances (VCD) of the LV distal Purkinje network, right ventricular hypertrophy, acute lateral myocardial infarction, hypertrophic cardiomyopathy (either obstructive or nonobstructive), type A Wolff-Parkinson-White syndrome (left-sided accessory pathway), left ventricular hypertrophy, Duchenne muscular dystrophy, endomyocardial fibrosis, dextroposition, misplaced precordial leads, and normal variant.^{4,6}

The single-artery blood supply to the left septal fascicle by the LAD artery (via its septal perforator branches)⁷ underlines that an LSFb should be

Figure 2. Angiography before and after successful stent implantation to the RCA.

Figure 3. (A) ECG post-PTCA to LAD. (Refer to text for details). (B) Angiography before and after stent implantation to the proximal LAD.

suspected in the setting of PAF and proximal LAD occlusion demonstrated by coronary angiography. Moreover, sudden onset of intermittent PAF in the right and/or middle precordial leads should alert the physicians of the possibility of a significant proximal obstruction of the LAD manifested as LSFB.^{4,6}

The first ECG (Fig. 1A) reveals sinus tachycardia at 125 bpm, a QRS axis at -30° (minor degree of LAFB), pathological QS waves in leads III and aVF, followed by ST-segment elevation ≥ 1 mm in the inferior leads and ST-segment depression (≥ 1 mm reciprocal changes) in the lateral leads. ST-segment elevation in lead III $>$ II, combined with a mirror image in I and aVL, has a high-positive predictive value for RCA obstruction.

On the ECG (Fig. 1A) there is not an initial q wave in the left leads because the patient has a systolic left ventricular hypertrophic pattern. (strain in leads I, aVL, V₅, and/or V₆). Classically, this pattern is characterized by absence of initial q wave associated with strain pattern of repolarization: ST-segment depression followed by asymmetric negative T wave in the left-side leads. The strain pattern has been associated with underlying coronary heart disease as in the index case. Additionally, the strain pattern is also associated with cardiovascular risk factors, such as hypertension, diabetes, older age, and male gender.

The second ECG (Fig. 1B) shows sinus rhythm at 94 bpm, QRS axis at -70° , QRS duration of 115 ms, qR pattern in lead I and aVL, rS pattern

in inferior leads with S wave in lead III > II (S wave in lead III > 15 mm) and rS pattern in the lateral precordial leads V₅–V₆ (Rosenbaum's type IV LAFB). Inverted T waves in the left leads (I, aVL, V₅, and V₆) are probably ischemia-related. The combination of LSFB + LAFB should be considered a bifascicular block (Fig. 1B).⁸ The presence of high R wave amplitude in lead V₂ (>15 mm), in conjunction with the absence of initial q wave in the left precordial leads, is one of the hallmark representations of LSFB.^{1,2,4,6,8}

On ECG 1B there is a small embryonic initial q wave followed by a very tall R wave in lead V₂ (R wave voltage = 26 mm). Both conduction disorders (incomplete right bundle branch block—IRBBB—and LSFB) may show PAF but they have different ECG patterns: In IRBBB, the activation of the right ventricle (RV) is delayed because depolarization has to spread across the septum from the left ventricle (LV). The LV is normally activated, meaning that the early part of the QRS complex is unchanged. The delayed RV activation produces a secondary wide R' in the right precordial leads, a broad and slurred S wave in the left lateral leads and a broad final R wave in aVR. Additionally, the QRS loop of VCG in the horizontal plane (HP) is located predominantly on right anterior quadrant. On the other hand, in LSFB the QRS loop in the HP is located predominantly in the left anterior quadrant. PAF is characterized by:

- I. Increased ventricular activation time in leads V₁–V₂;
- II. Frequent embryonic small initial q wave in lead V₂ or V₂–V₃;
- III. R wave voltage in lead V₂ >15 mm;
- IV. Sharp-point R wave in lead V₂ or V₂–V₃;
- V. Absence of q wave in the lateral left leads consequence of the inexistence of first septal vector, vector 1, first anteromedial vector (1_{AM}) or Peñaloza and Tranchesi vector;
- VI. Decreasing R waves from V₄ to V₆.

Consequently, in the LSFB, unlike the RBBB, the early part of the QRS complex is changed.

The third ECG (Fig. 3A) displays sinus rhythm with a QRS axis at –65°, qR pattern in leads I and aVL, rS pattern in the inferior leads (S wave

III > II) and RS pattern in the left precordial leads, all consistent with LAFB. New negative T wave inversions in the anterolateral leads are also seen as a manifestation of residual ischemia. Following PTCA to the LAD, the characteristic PAF pattern is no longer present, which supports that the transient PAF (LSFB) was caused by the proximal LAD obstruction.

CONCLUSION

This case relates the transient appearance of PAF, most likely the expression of an underlying LSFB, in association with ischemic chest pain and angiographic evidence of intracoronary thrombosis of the proximal LAD before S₁. Coronary recanalization of the proximal LAD led to the resolution of the presumed LSFB. In clinical practice, recognition of this ECG pattern appears particularly useful in the setting of acute myocardial ischemia, as there is growing evidence that transient LSFB may be a marker of severe coronary artery disease.

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