Clinical Case

Transient left septal fascicular block in acute coronary syndrome. An entity to consider?

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ABSTRACT

The left branch of the conduction system has a complex distribution in the left ventricle, resulting in great anatomical variability. A transient conduction disorder with prominent anterior forces in the right precordial leads has recently been described. This disorder was called left septal fascicular block and occurs when there is an obstruction/occlusion of the anterior descending artery. We present the case of an acute coronary syndrome with ST elevation due to occlusion of the anterior descending artery that was associated with transient left septal fascicular block, which disappeared with percutaneous revascularization. This electrocardiographic finding, which is rarely observed and has limited electrophysiological evidence, should not go unnoticed in the presence and absence of ST elevation, although the presence of ST elevation beyond the conduction disorder always indicates urgent coronary revascularization.

INTRODUCTION

Rosenbaum MB et al, determined more than 50 years ago that the left branch splits into the anterior and posterior fascicles, which can get blocked, and since then the terms anterior hemiblock and left posterior fascicular block are accepted in electrocardiography, determining the trifascicularity of the conduction system.[1,2] However, anatomical studies suggested that the left branch end is more complex than just a bifurcation. Tawara S. in 1906 pointed out that this branch has fibers coming from it on the surface of the interventricular septum.[3] Demoulin and Kulbertus in 1972, in an elegant histological reconstruction of the left conduction system, proved the existence of a third division of the left branch in 20 normal hearts. This structure had a trajectory through the septal region and emerged from the left branch trunk, from the anterior fascicle, posterior fascicle or a complex of ramifications from both fascicles, producing a great anatomical variability as shown in *Figure 1*.[4,5]

Recently, Perez Riera AR et al, showed a transient conduction disorder by electrocardiogram (ECG) and vectorcardiogram, with prominent anterior forces in precordial leads (V1-V2) in different clinical scenarios, and they called it left septal fascicular block (LSFB).[6,7] This conduction disorder is rare, hard to identify electrocardiographically due to its transitoriness, and with limited electrophysiological evidence.

CLINICAL CASE

Eighty-three-year old patient, who after moderate effort presented oppressive retrosternal pain, with 8/10 intensity, and no radiation or neurovegetative symptoms. Forty-five minutes after the onset of symptoms, he consulted to the hospital where ECG was performed, showing ST-segment

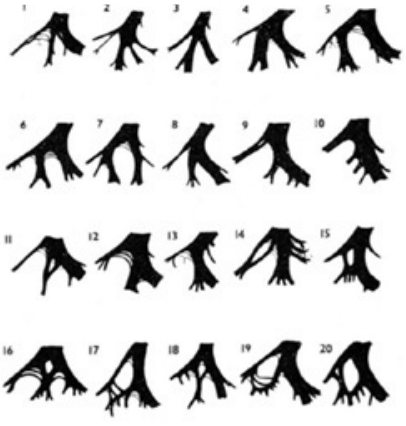

FIGURE 1.
Outline of the left ventricular conduction system observed in 20 normal hearts. The diagrams show a septal or mid fascicle that emerges from the central area of the left branch trunk (diagrams 1-4), from the left anterior fascicle (diagrams 5-7), the left posterior fascicle (diagrams 8-14) or fibers originating from both fascicles (diagrams 15-20).[4]

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elevation acute coronary syndrome (STEACS). He was referred to this hospital, and upon admission we verified ST elevation from V1 through V3, left anterior hemiblock (LAHB) and prominent R waves in right precordial leads. He underwent emergency coronary angiography, showing proximal occlusion of the anterior descending artery (ADA) with TIMI 0 epicardial flow as culprit artery for the acute myocardial infarction *(Figure 2A).* Next, primary percutaneous coronary intervention (PCI) was conducted, with implant of 2 drug-eluting stents of 3.0 x 13 mm, achieving post-PCI TIMI 3 epicardial flow.

The patient had a history of hypertension, treated with losartan 50 mg/day and amlodipine 5 mg/day.

Upon admission to the coronary unit, he presented BP 90/48 mmHg, HR 48 bpm, respiratory rate of 14 bpm, temperature 36°C and oxygen saturation 99%. In physical examination, he had a holosystolic murmur 2/6 in aortic focus. Laboratory tests upon admission showed the following abnormal measures: AST 122 U/L, CPK 520 U/L, LDH 299 U/L, hs-cTnT 959 pg/ml.

ECG upon admission to the coronary unit showed ST-elevation decrease, LAHB and absence of prominent R in V1-V2 *(Figure 2B).*

DISCUSSION

The case presented shows a rare disorder in the conduction system: transient LSFB associated to LAHB during STEACS.

The LSFB is mentioned in literature and is defined as when R wave voltage is observed in V1 ≥5 mm, R/S ratio in V1 and V2 ≥2, R width in V2 ≥15 mm, S wave ≤5 mm and characteristic in crescendo voltage of r/R wave in V1-V3 and decreasing in V5-V6, and absence of q wave in V5, V6 and I.[6]

This electrocardiographic pattern manifests when there is proximal obstruction of the ADA before the septal perforating branches or by subocclusion of the left main coronary artery, leading to ischemia in the upper 2/3 of the interventricular septum and the apical portion of the left ventricle, in the trajectory of the septal fascicle.[8] In this case, early revascularization determined the transitoriness of LSFB, not so of LAHB, which was considered to be chronic due to degenerative phenomenon caused by age and/or hypertension.

This electrocardiographic pattern, appearing transitorily or intermittently, is observed more frequently over the course of acute myocardial infarction with occlusion of severe proximal obstruction in the ADA. However, the presence of this conduction disorder over the course of acute ischemic syndrome may resemble two very well established clinical situations; i.e. Wellens syndrome and Winter syndrome, involving coronary angiography and emergency revascularization of ADA.[9]

As in any clinical-electrocardiographic analysis, differential diagnoses have to be made, excluding alterations that may resemble LSFB: for instance, the normal variant,

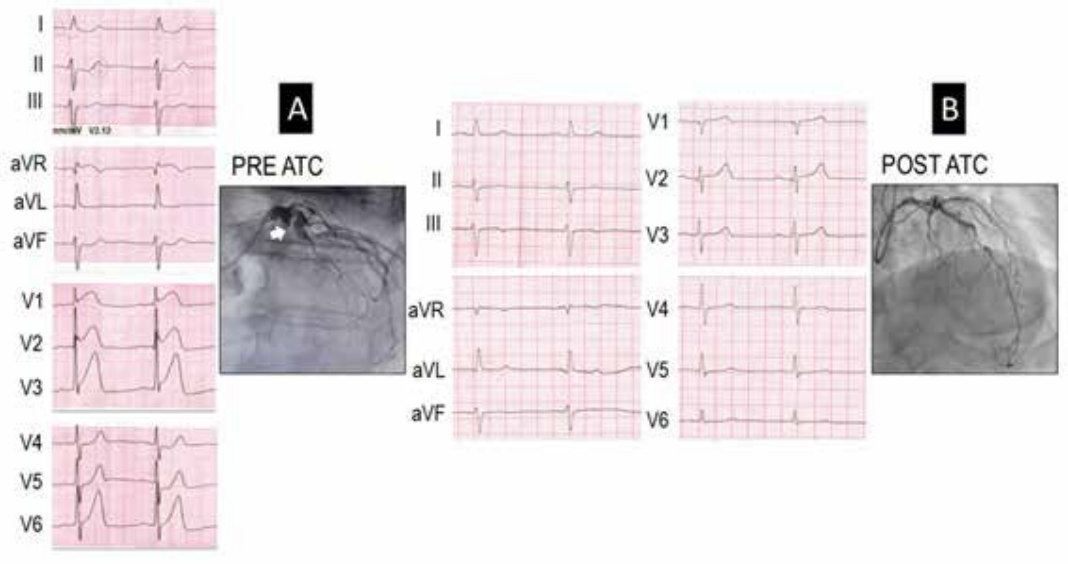

FIGURE 2.
Electrocardiogram with ST elevation in V1-V3, associated to left anterior hemiblock (LAHB) and left septal fascicular block (LSFB) due to proximal occlusion of the anterior descending artery. A. Electrocardiogram during the course of ST-elevation acute coronary syndrome: sinus rhythm, HR 55 bpm, PR 0.20 sec, QRS 100 msec, electrical superior QRS axis (LAHB) associated to LSFB, determined by R wave in V1 of 7 mm, R/S ratio greater than 2, R width in V2 of 14 mm, growth of R from V1 through V3 (prominent anterior forces) and decrease of R from V4 to V5, and absence of Q in V5, V6 and I. Coronary angiography showing proximal occlusion of the anterior descending artery before the septal perforating arteries with TIMI 0 flow in left anterior oblique view. B. Electrocardiogram after revascularization where LAHB persists, with disappearance of ST elevation and prominent right precordial forces, determining transient LSFB. Coronary angiography after stent implant with TIMI 3 flow in the anterior descending artery in cranial front view.

by marked counterclockwise rotation of the heart on its longitudinal axis (≈1% of normal individuals), athlete's heart, error in positioning precordial electrodes, lateral infarction, right ventricular hypertrophy, right bundle branch block, ventricular preexcitation, hypertrophic cardiomyopathy, cardiomyopathy associated to Duchenne muscular dystrophy, and so on.[6]

To conclude, the appearance of prominent R waves in the right precordial leads in acute coronary syndromes may indicate the existence of LSFB, which could be transient, and be determined by ADA obstruction/occlusion. This electrocardiographic finding, rarely observed, of limited electrophysiological evidence, should not go unnoticed, although the existence of ST elevation beyond conduction disorder always requires an emergency coronary revascularization.

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