

Dr. Raimundo Barbosa Case report (courtesy)

English

Dear friend Prof Andrés sees what nice case!

Patient feminine 78years old was admitted with typical clinical picture of an Acute Coronary Syndrome (ACS).The ECG(first ECG) shows ST segment elevation in inferior lead <12 hs evolution. Submitted to primary angioplasty with success (stent placement in culprit Right Coronary Artery (RCA). Additionally, the angiocoronarygraphy study shows in association a significative proximal obstruction in left anterior descending coronary artery (LAD). The strategy was then to carry through an elective angioplasty in second time. After three weeks she returns with several episodes of angina and acute pulmonary edema. After stabilization of the clinical picture the patient was submitted to the new angioplasty successfully of the LAD.

ECG 1 (first admission)

ECG2 (during angina episode). Which is the diagnosis?

Later I will send the ECG carried through after the procedure to prove the evolution this

ECG evolution

Hug

Raimundo Barbosa

Andrés Ricardo Pérez Riera analysis

Portuguese

Paciente 78anos, do sexo feminino, admitida com síndrome coronária aguda (SCA) com supra desnivelamento do segmento ST na parede inferior com < 12h de evolução. Submetida com sucesso à angioplastia primária + stent da artéria culpada (CD). Na ocasião o CATE revelava oclusão total de CD e lesão significativa proximal da artéria DA. Foi programada ATC eletiva para esta artéria, porém a paciente solicitou alta hospitalar retornando após três semanas com episódios de angina associado a quadro de edema agudo de pulmão.

Após estabilização, a paciente foi submetida à nova angioplastia com sucesso da artéria DA.

ECG 1 (primeira admissão);

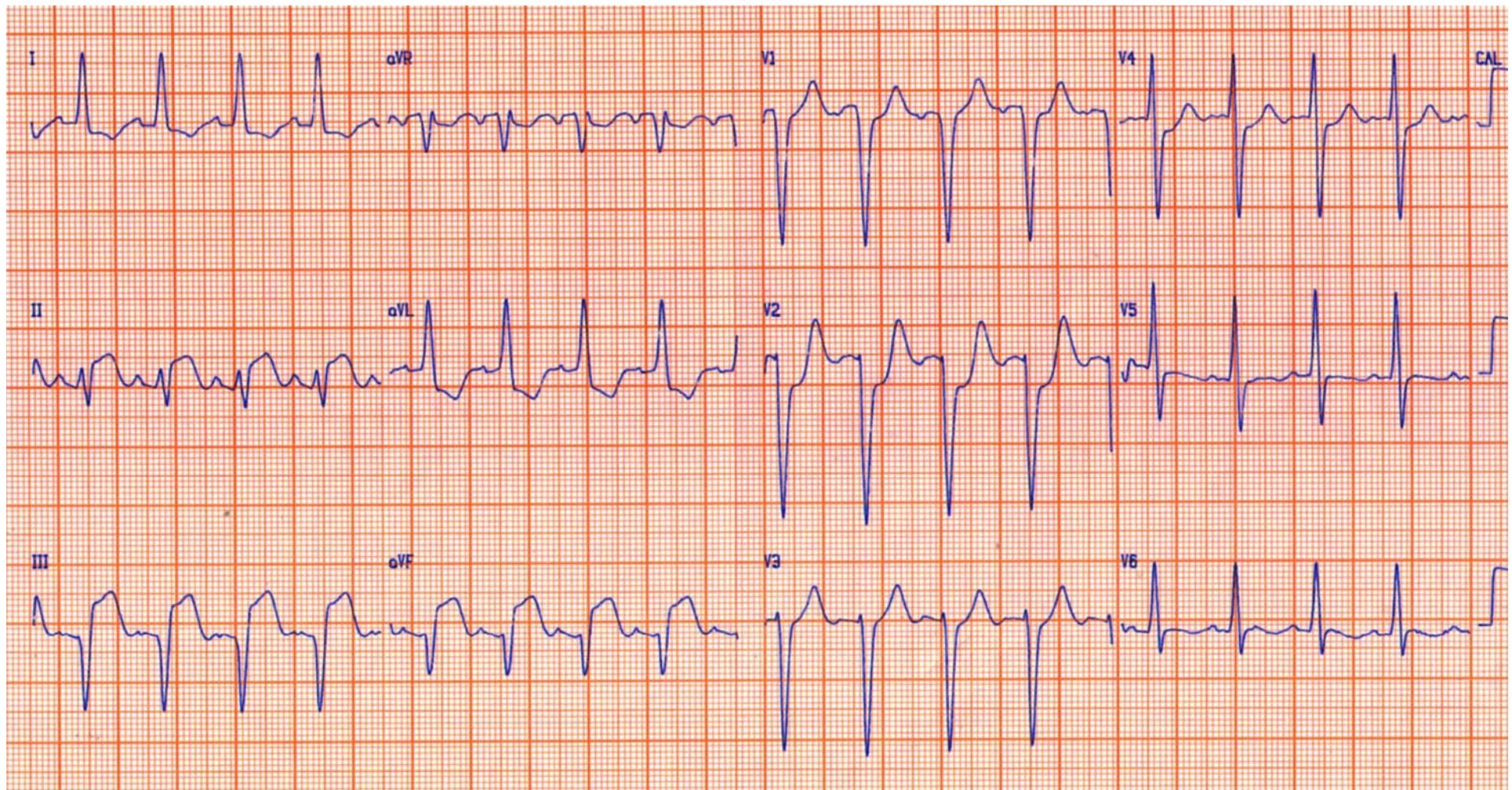
ECG2(segunda internação durante episódio de angina). Qual o diagnóstico?

Posteriormente enviarei ECG pós procedimento para comprovar a evolução do segundo traçado.

Um abraço

Raimundo.

ECG 1 FIRST ADMISSION < 12h EVOLUTION PAIN

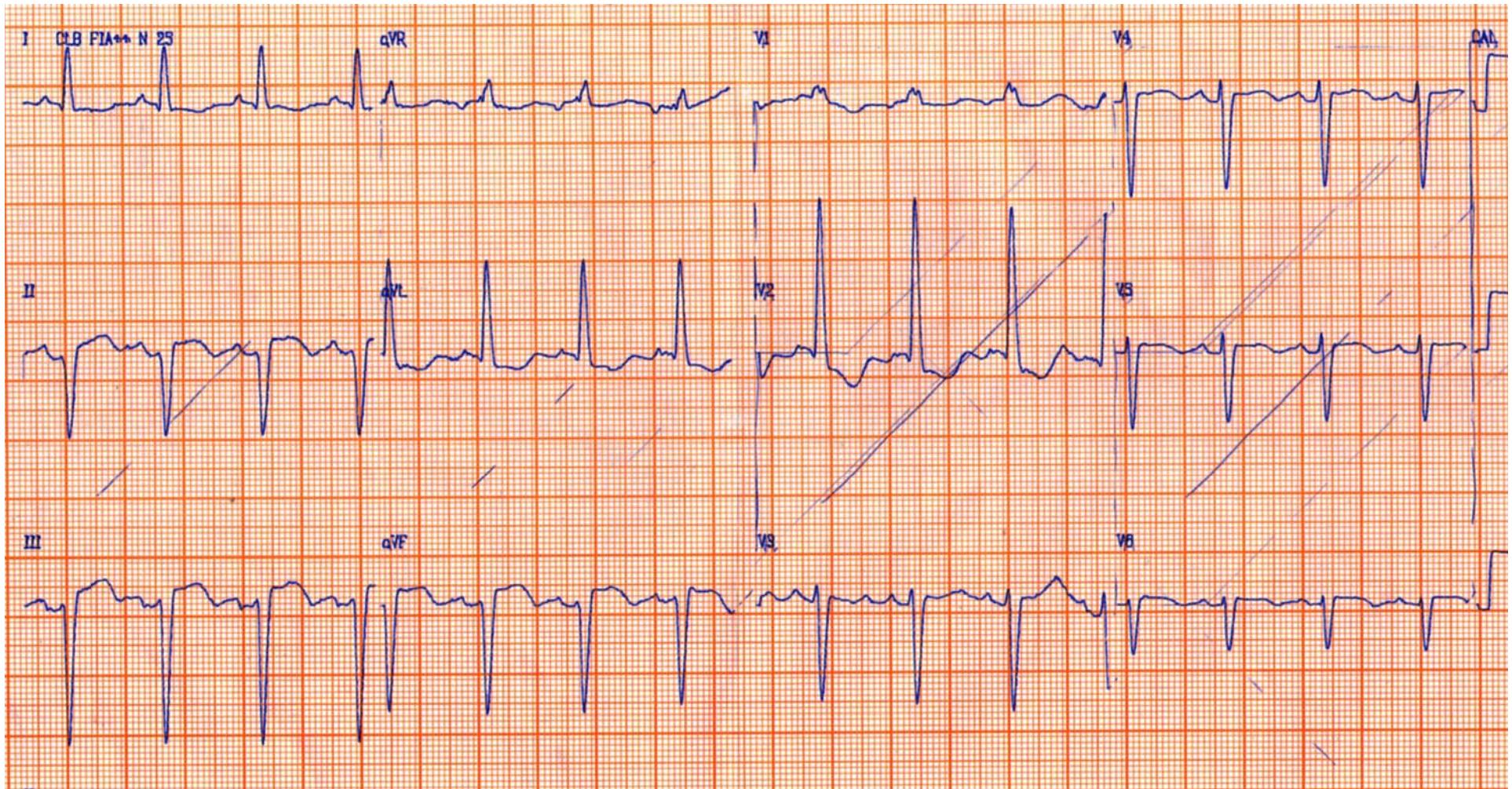


ECG diagnosis: Sinus tachycardia, QRS axis -30°. QS pattern in III and aVF followed by ST segment elevation convex to the top (subepicardial injury current).

Mirror image from V1-to V4: ST segment depression conspicuous in V2.

Conclusion: Acute inferior myocardial infarction.

ECG2 (during second angina episode).



ECG diagnosis: Sinus tachycardia, extreme left axis QRS deviation - 70°, QRSd: 115ms, qR in I and VL, rS pattern in II, III and aVF, SIII>SII followed by minimal ST segment elevation convex to the top (subepicardial injury current). rS pattern from V3 to V6: **Left Anterior Fascicular Block LAFB**.

r wave in V1 and qR pattern in V2 (R wave 25mm tall!): **Left Septal Fascicular Block LSFB**.

Conclusion: Left bifascicular block **LAFB + LSFB**. Injury current in inferior leads. Initial embryonic r wave in all inferior leads.

Addendum:

LAFB Rosembaum type IV: association of LAFB +LVE or LVH. Why? Because S III > 15 mm, Inverted T wave in one or more of the left leads: D1, aVL, V5 and V6.

LEFT INTRAVENTRICULAR HIS SYSTEM BLOOD SUPPLY(1)

- The blood supply to most of the human His bundle and its proximal branches is dual in origin, with anastomosis principally within the His bundle.

I) HIS BUNDLE BLOOD SUPPLY

It is dually supplied by the AV node artery from the right coronary artery (RCA) and the first septal branch of the left anterior descending artery (LADA) in 90% of the cases, but entirely by the AV node artery in 10%;

II) PROXIMAL RIGHT BUNDLE BRANCH BLOOD SUPPLY

It is supplied by both the AV node artery and the septal branch in 50% of the cases, only the septal branch in 40%, and the AV node artery alone in one (10%).

III) LEFT BUNDLE BRANCH BLOOD SUPPLY

It is dually supplied by the AV node artery (ramus septi fibrosi) from the right coronary artery (RCA in 90% of the cases) and ramus septi ventriculorum superior and ramus critae. Branches from LADA: ramus limbi sinistri.

IV) BLOOD SUPPLY OF THE LEFT BRANCH FASCICLES OR DIVISIONS

- **Branches of the LADA:**.....LAF: 40%; LPF: 10%; LSF: **100%**
- **Double irrigation (LADA & RCA):** LAF: 50%; LPF: 40%; LSF: **0%**
- **RCA branches:**.....LAF: 10%; LPF: 50%; LSF: **0%**
- **LADA – Anterior Descending Artery; RCA – Right Coronary Artery; LAF: Left Anterior Fascicle; LPF: Left Posterior Fascicle; LSF: Left Septal Fascicle.**

1. Frink RJ, James TN. *The Normal blood supply to the human His bundle and proximal branches Circulation* 1973; 47: 8-18.

The LSF is irrigated exclusively by the septal perforating branches of the LADA. Critical lesions of the LADA before the first perforating septal branch (S1) constitute the main cause of LSFB in developed countries, and it is a major determinant of R wave amplitude during acute myocardial ischemia. The LSB eventually is exercise-induced, transient or intermittent and sometimes it originates giant R waves (1; 2; 3;4; 5; 6).

Coronary sources for LSFB at the InCor service of São Paulo are 18% of the total.

The appearance of LSFB in critical LADA lesions, speaks in favor of the proximal lesion, and therefore, of a worse prognosis.

Intermittent LSFB secondary to critical lesion of LADA was described for us, and recently during ergometer test(7).

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2. Tranches J, Moffa PJ, Pastore CA, et al. Block of the antero-medial division of the left bundle branch of His in coronary diseases. Vectrocardiographic characterization. Arq Bras Cardiol 1979; 32:355-360.
3. Cesar LAM, Carvalho VB, Moffa PJ, et al. - Bloqueio da divisão ântero-medial do feixe de His e obstrução da arteria coronária descendente anterior. Correlação eletro-cinecoronariográfica. Rev Latina de Cardiol 1980; 1:57-63.
4. Hassapoyannes CA, Nelson WP. Myocardial ischemia-induced transient anterior conduction delay. Am Heart J 1991; 67:659-660.
5. Madias JE.: The “giant R waves” ECG pattern of hyperacute phase of myocardial infarction. J Electrocardiol 1993; 26:77-80.
6. Moffa PJ, Pastore CA, Sanches PCR et al. The left-middle (septal) fascicular block and coronary heart disease. In Liebman J, ed. *Electrocardiology' 96 – From the cell to body surface*. Cleveland, Ohio, Word Scientific, 1996; 547-550.
7. Uchida AH, Moffa PJ, Pérez Riera AR, et al. Exercise-induced Left septal Fascicular Block: An Expression of Severe Myocardial Ischemia. Indian Pacing and Electrophysiology Journal; 2006; 6: 135-138. Online:<http://www.ipej.org/0602/uchida.htm>).

ETIOLOGIES AND INCIDENCE OF LSFB

Literature and our own experience, show us the following etiological causes for LSFB:

- 1.Chronic Chagasic Cardiomyopathy(1)
- 2.Coronary Artery Disease (CAD): critical lesion of LADA(2) and/or its septal perforating branches before the first septal (S1) one. We observe the first case associated with Wellens syndrome(3)
- 3.Non-Obstructive Hypertrophic Cardiomyopathy (NO-HCM)
- 4.Obstructive Hypertrophic Cardiomyopathy (O-HCM)
- 5.Diabetes Mellitus
- 6.Papillary Muscle Dysfunction
- 7.Kearns-Sayre syndrome(5)

1. Riera AR, Uchida AH, Schapachnik E, Dubner S, Zhang L, Filho CF, Ferreira C, Ferrara DE, de Luna AB, Moffa PJ. The History of Left Septal Fascicular Block: Chronological Considerations of a Reality Yet to be Universally Accepted. Indian Pacing Electrophysiol J. 2008 Apr 1;8:114-128.
2. Uchida AH, Moffa PJ, Pérez Riera AR, et al. Exercise-induced Left septal Fascicular Block: An Expression of Severe Myocardial Ischemia. Indian Pacing and Electrophysiology Journal; 2006; 6: 135-138. Online:<http://www.ipej.org/0602/uchida.htm>).
3. Riera AR, Ferreira C, Ferreira Filho C, Dubner S, Schapachnik E, Uchida AH, Moffa P, Zhang L, de Luna AB. Wellens syndrome associated with prominent anterior QRS forces: an expression of left septal fascicular block?J Electrocardiol. 2008 Nov-Dec;41:671-674.
4. Magnacca M, Valesano G, Rizzo G, et al. Diagnostic value of electrocardiogram in septal fascicular conduction disorders of the left branch in diabetics Minerva Cardioangiologica 1988; 36:361-363.
5. Riera AR, Kaiser E, Levine P, Schapachnik E, Dubner S, Ferreira C, Ferreira Filho C, de Luna AB, Zhang L. Kearns-Sayre syndrome: electro-vectorcardiographic evolution for left septal fascicular block of the his bundle. J Electrocardiol. 2008 Nov-Dec;41:675-678.

ECG-VCG CHARACTERIZATION OF LSFB (PROPOSAL FOR CRITERIA) ELECTROCARDIOGRAPHIC

- Normal QRS duration or with a discrete increase (up to 110 ms). When associated with other fascicular or bundle blocks it could be ≥ 120 ms
- FP leads with no modifications: normal QRS
- Increased ventricular activation time or intrinsic deflection V1 and V2: ≥ 35 ms
- R wave voltage of V1 \geq than 5 mm
- R/S ratio in V1 > 2
- R/S ratio in V2 > 2
- S wave depth in V1 < 5 mm
- Possible small (embryonic) q wave in V2, V3 or V1 and V2
- R wave of V2 > 15 mm
- RS or Rs pattern in V2 and V3 (frequent rS in V1) with R wave "in crescendo" from V1 through V3 and decreasing from V5 to V6
- Absence of q wave in left precordial leads V5, V6 and DI (by absence of vector 1AM). Absence of ILBBB, CLBBB, WPW is necessary;
- Intermittent PAF during hyperacute phase of myocardial infarction (68), exercise stress testing in patients with severe myocardial ischemia (71; 72; 77; 78) and during early atrial extrastimuli.
- Apparition of intermittent, rate-dependent q wave in V1 and V2.

Vectocardiographic characterization (all in the HP)

- QRS loop in the HP with an area predominantly located in the left anterior quadrant (> 2/3 of the loop facing the orthogonal X lead: 0° to $\pm 180^\circ$)
- Absence of normal convexity to the right of the initial 20 ms of the QRS loop
- Discrete dextro-orientation with moderate delay of the vector from 20 ms to 30 ms
- Anterior location of the vector from 40 to 50 ms
- Posterior location with a reduced magnitude of the vector from 60 to 70 ms
- Maximal vector of the QRS loop located to the right of $+30^\circ$
- Intermittent anterior displacement of QRS loop (79)
- T loop with posterior orientation tendency (useful for the differential diagnosis with posterior MI)
- The QRS loop rotation may be:
Counterclockwise: incomplete LSFB.
Clockwise: advanced or complete LSFB or in association with CRBBB, LAFB, or LPFB.

First Answer

Interesting case, Dr. Barbosa. The first electrocardiogram corresponds to inferior acute coronary syndrome with a certain degree of evolution (in the leads of the inferior side, the negative terminal portion of T can be seen, which guides us at the beginning of reperfusion, as well as Q waves in aVF and III). Since there are no right chest leads, the presence of isoelectric ST in V1 guides us to the occlusion being probably proximal to the right ventricular artery.

In my modest opinion, the second ECG suggests acute coronary syndrome by proximal occlusion of ADA (before the first septal) and block of the septal fascicle of the left branch, originating high voltage in V1, and mostly in V2. After three weeks ST elevation persists in inferior leads. Since the occlusion is proximal, it is unlikely that this elevation originates in the ischemic symptoms, and ventricular aneurysm should be ruled out.

Best regards,

Interesante caso Dr. Barbosa. El primer electrocardiograma se corresponde con un síndrome coronario agudo inferior con cierto grado de evolución (se puede observar en las derivaciones de la cara inferior la porción terminal negativa de la T que nos orienta comienzo de la reperfusión, así como ondas Q en aVF y III). Al no disponer de derivaciones torácicas derechas, la presencia de ST isoeléctrico en V1 nos orienta a que la oclusión será posiblemente proximal a la arteria ventricular derecha.

En mi modesta opinión, el segundo ECG sugiere síndrome coronario agudo por oclusión proximal de la DA (antes de la primera septal) y bloqueo del fascículo septal de la rama izquierda originando voltaje alto en V1 y sobre todo en V2. Después de tres semanas persiste ascenso del ST en derivaciones inferiores. Al ser la oclusión proximal no es probable que este ascenso sea originado por el cuadro isquémico y habría que descartar aneurisma ventricular.

Un afectuoso saludo,

Javier García-Niebla