Development clinical-electro-vectorcardiographic deterioration of an acute anteroseptal infarction with anterolateral ischemia

Deterioro clínico-eletro-vetorcardiográficos evolutivo de um infarto agudo ântero-septal com isquemia anterolateral

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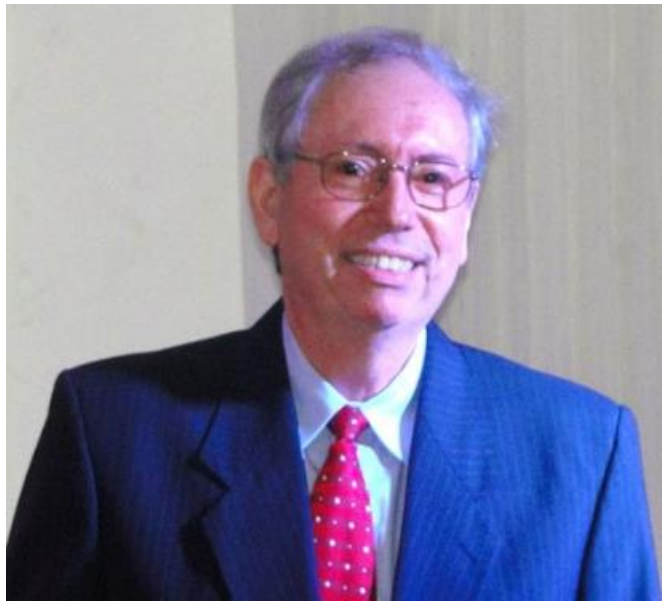

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English Case report

ARS, male, white, bank teller, 58 years old, type 2 diabetes and dyslipidemic. Six months ago he was admitted with symptoms of ST segment elevation acute myocardial infarction in anterior wall (ECG1 performed at hospital discharge). At the time, angioplasty was attempted unsuccessfully, as the guidewire did not go through the lesion (total obstruction of the middle third). Subocclusive lesion was observed in the first diagonal branch. The right coronary artery (RCA) is dominant and left circumflex artery(LCX) reached the middle third of the AV sulcus, both being normal He received the discharge being stable and asymptomatic.

Four months after the discharge he returned with symptoms of congestive heart failure, and gallop rhythm B3; BP = 13/80 mmHg.

He was medicated with carvedilol 25 mg 2 x day, enalapril 20 mg 2 x day, aspirin 10 mg, atorvastatin 40 mg, spironolactone 25 mg x day, furosemide 40 mg x day.

Echo = segmentary hypokinesis of anterior wall with left ventricular ejection fraction = 38%.

Questions:

1. **What is the electrocardiographic diagnosis of ECG1?**

2. **What is the diagnosis of the current ECG2/VCG?**

3. **How do you explain the changes that occurred?**

Reporte de caso (Português)

ARS, masculino, branco, bancario, 58 anos, diabético tipo 2 e dislipidêmico. Há 6 meses deu entrada com quadro de infarto agudo de miocárdio com elevação do segmento ST de parede anterior (ECG-1 realizado na alta hospitalar). Não ocasião, foi tentado angioplastia sem sucesso pois o fio guia não ultrapassou a lesão (obstrução total no terço médio). Se observou lesão sub-oclussiva da primeira diagonal. A artéria coronária direita era dominante e a circunflexa esquerda alcançava o terço médio do surco AV Ambas eram normais. Recebeu alta estável e assintomático.

Quatro meses depois da alta retorna com quadro de insuficiência cardíaca congestiva, ritmo com cadência de galope B3; PA= 13 /80 mm de Hg

Medicado com carvedilol 25mg 2 x dia, enalapril 20mg 2 x dia, aspirina 10omg, atorvastatina 40mg, espironolactona 25mg x dia ,furosemida 40mg x dia

Ecocardiograma transtorácico: hipocinesia segmentar da parede anterior com fração de ejeção do ventrículo esquerdo de 38%

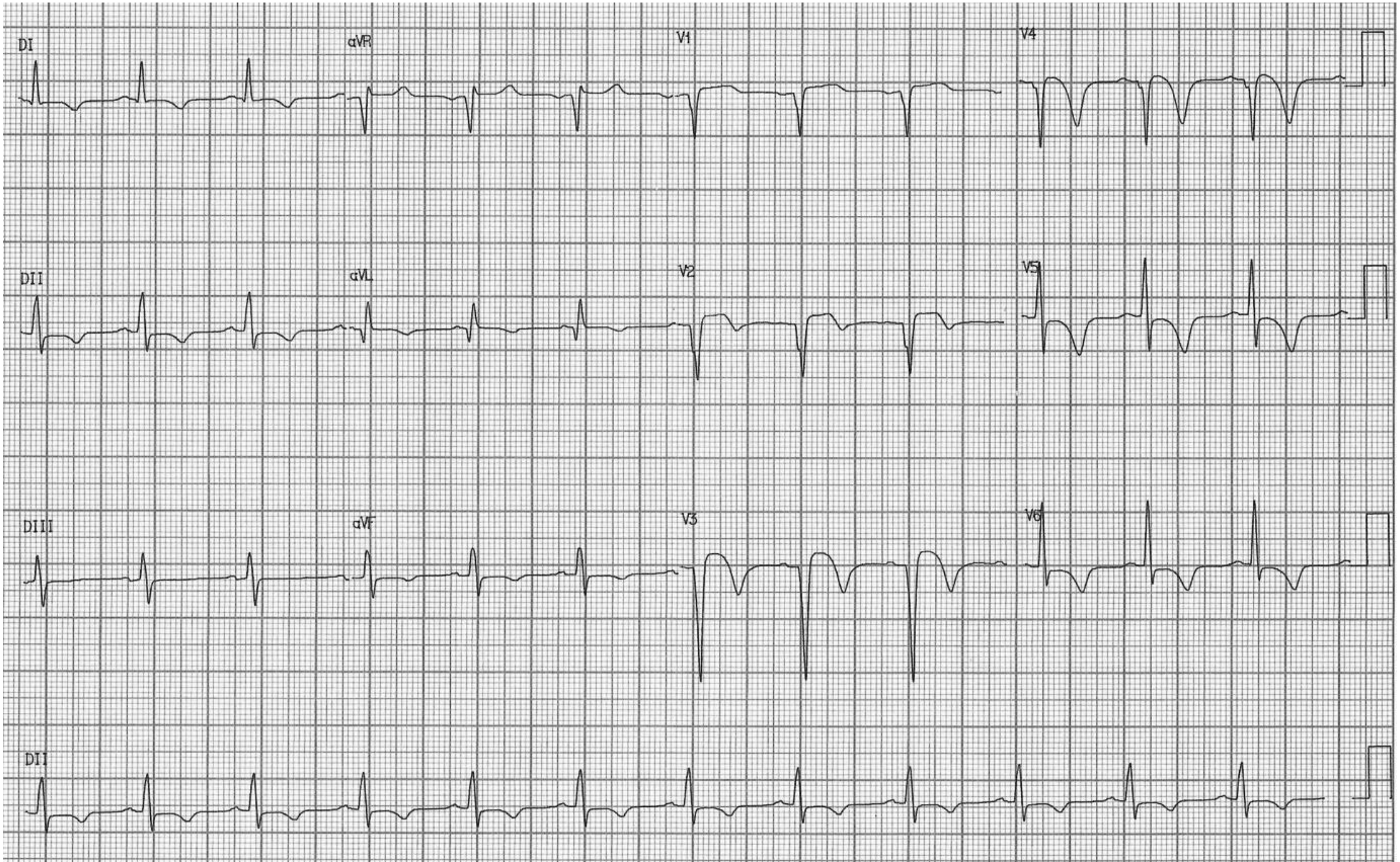
Perguntas

1. Qual o diagnóstico eletrocardiográfico do ECG-1?

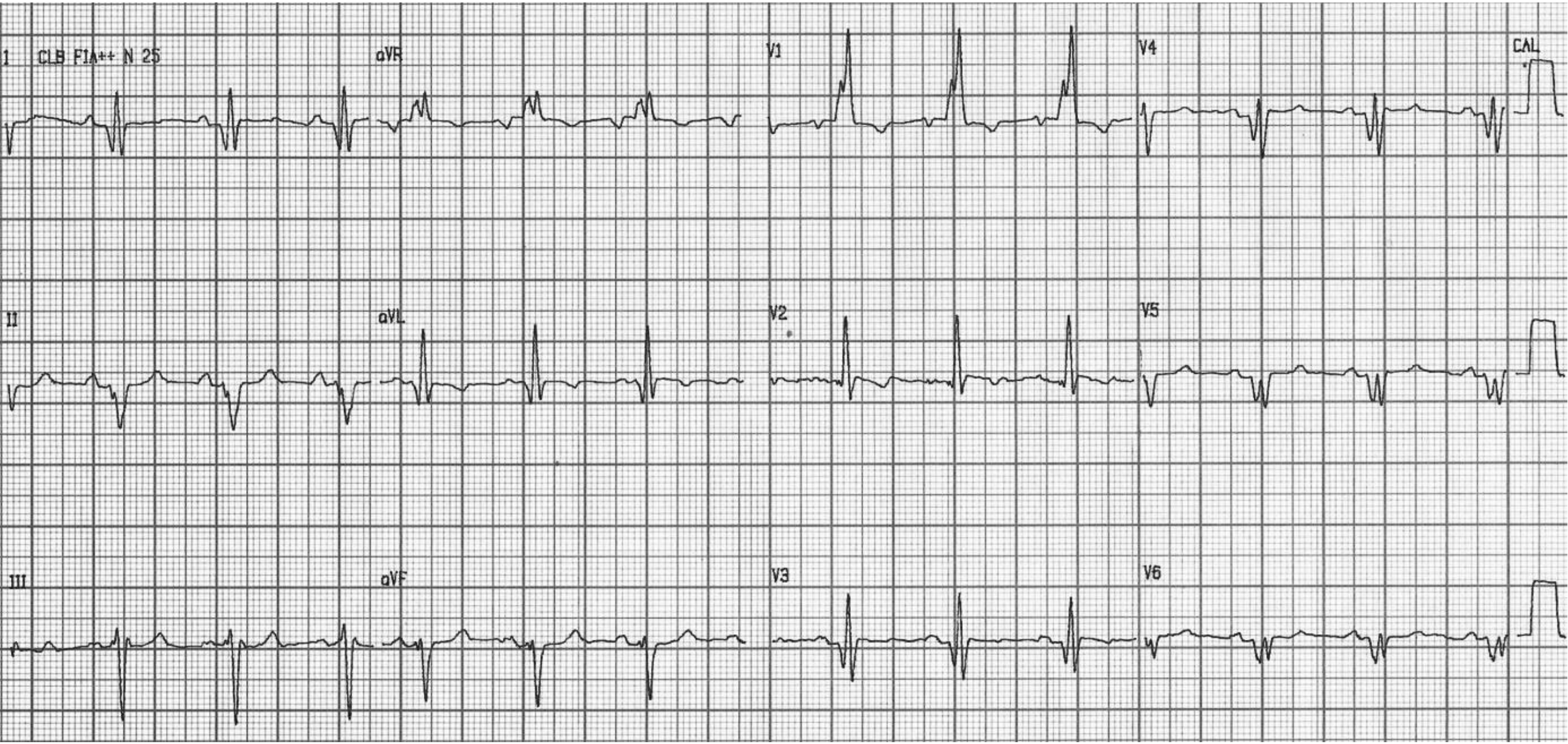
2. Qual o diagnóstico do ECG-2/VCG atuais?

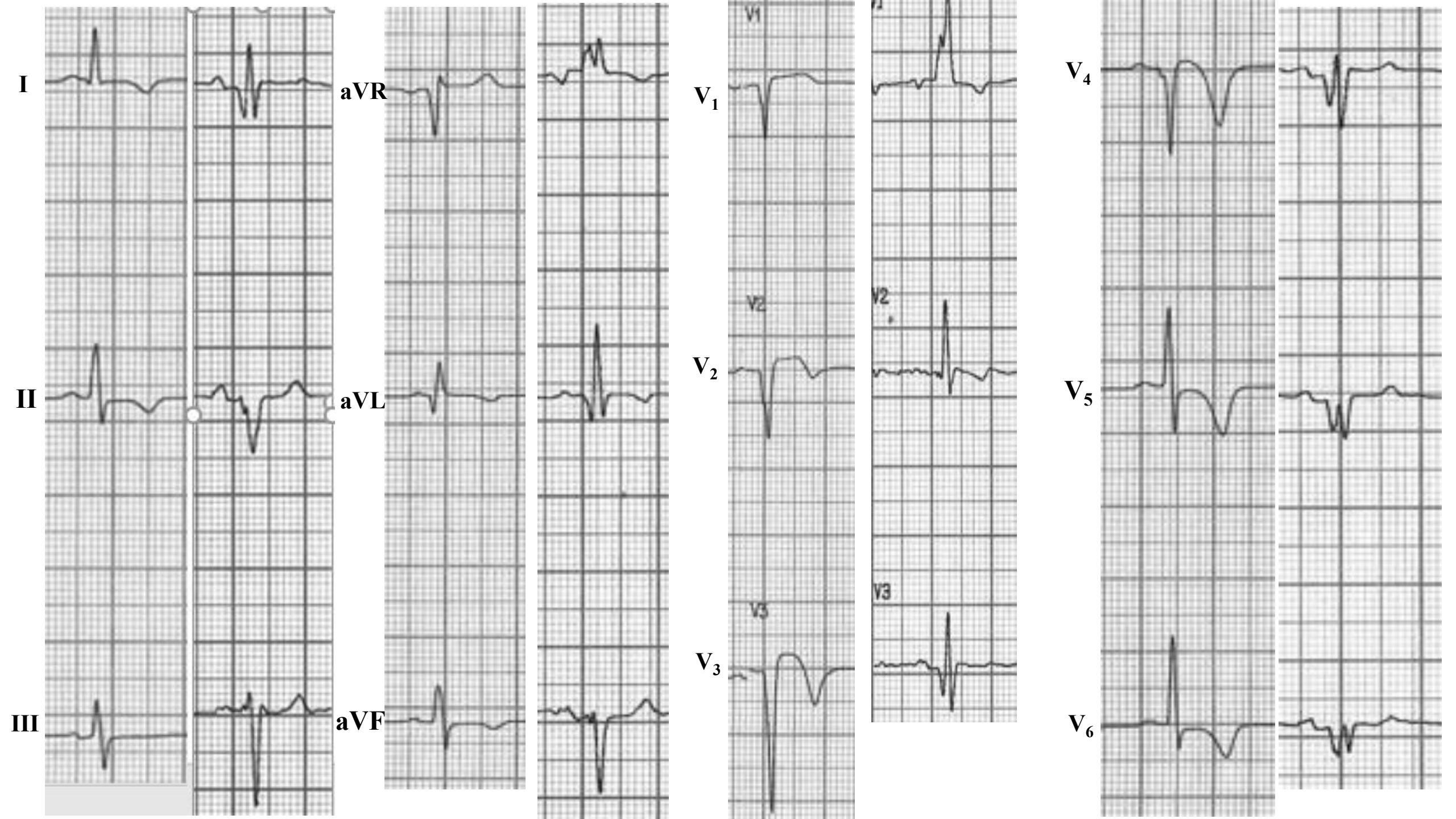
3. Como explica as mudanças eletro-vetorcardiográficas ocorridas?

ECG-1 Realizado na alta hospitalar ECG performed at hospital discharge



ECG-2 Atual: realizado 4 meses após a alta hospitalar/ECG-2 Current: performed 4 months after discharge




I
II
III
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V1
V2
V3
V4
V5
V6

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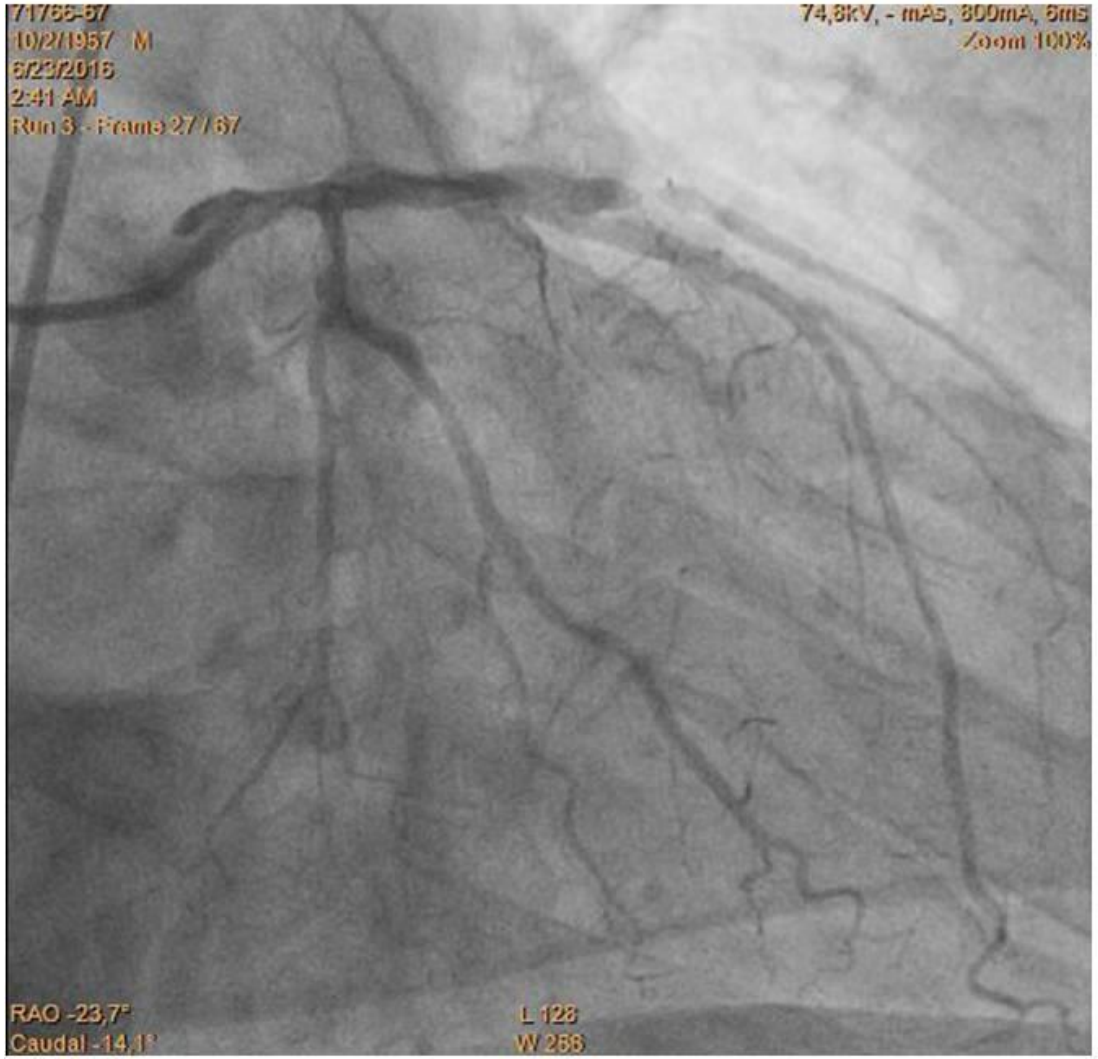
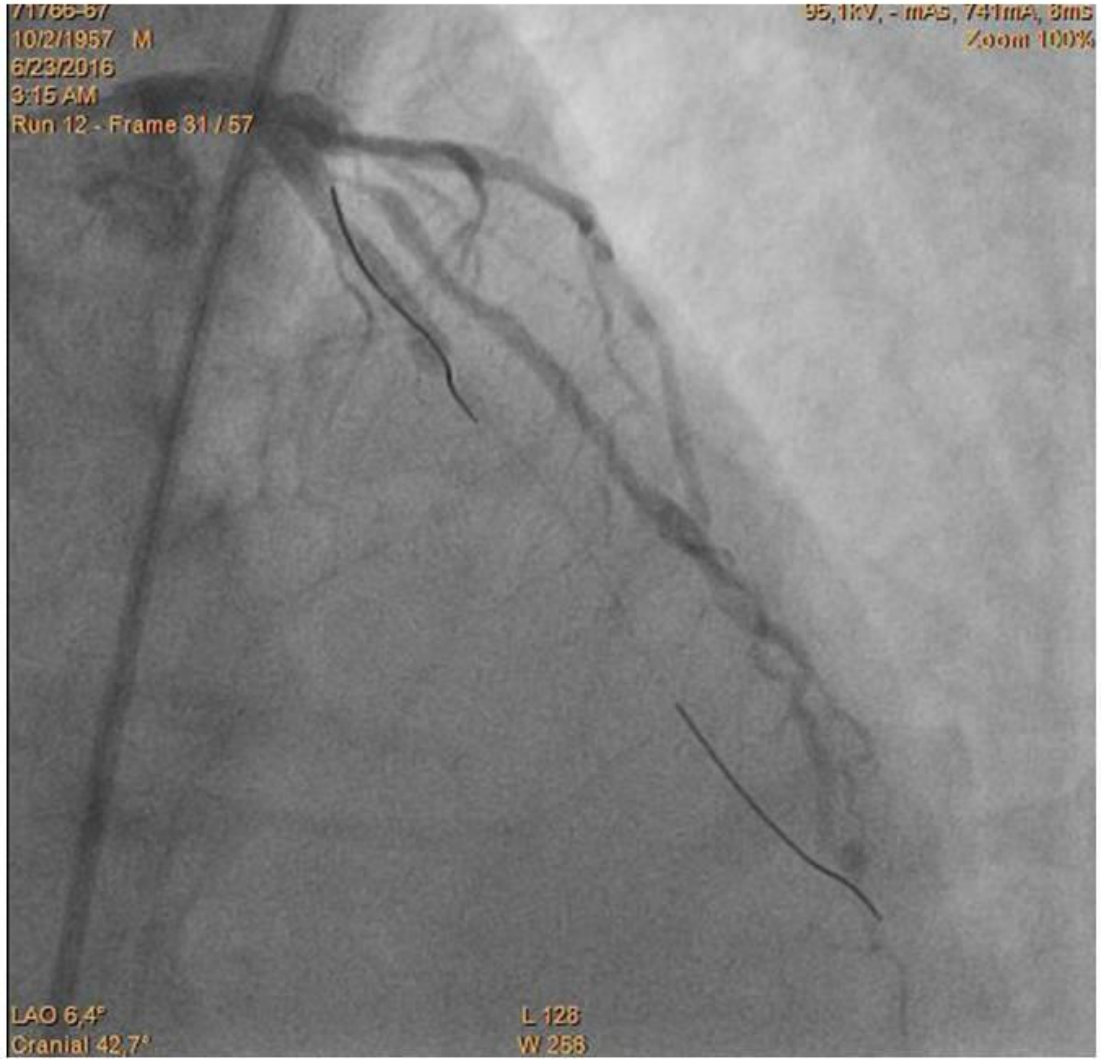
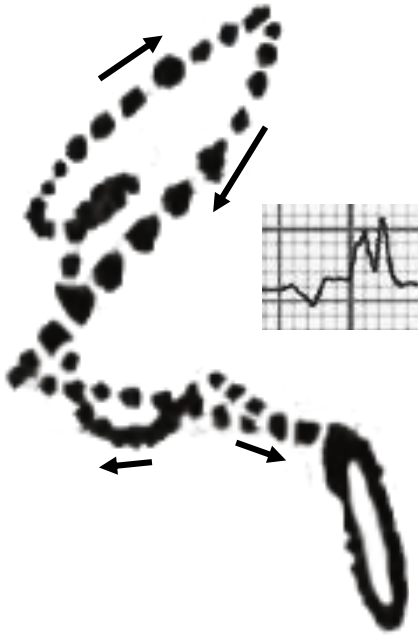
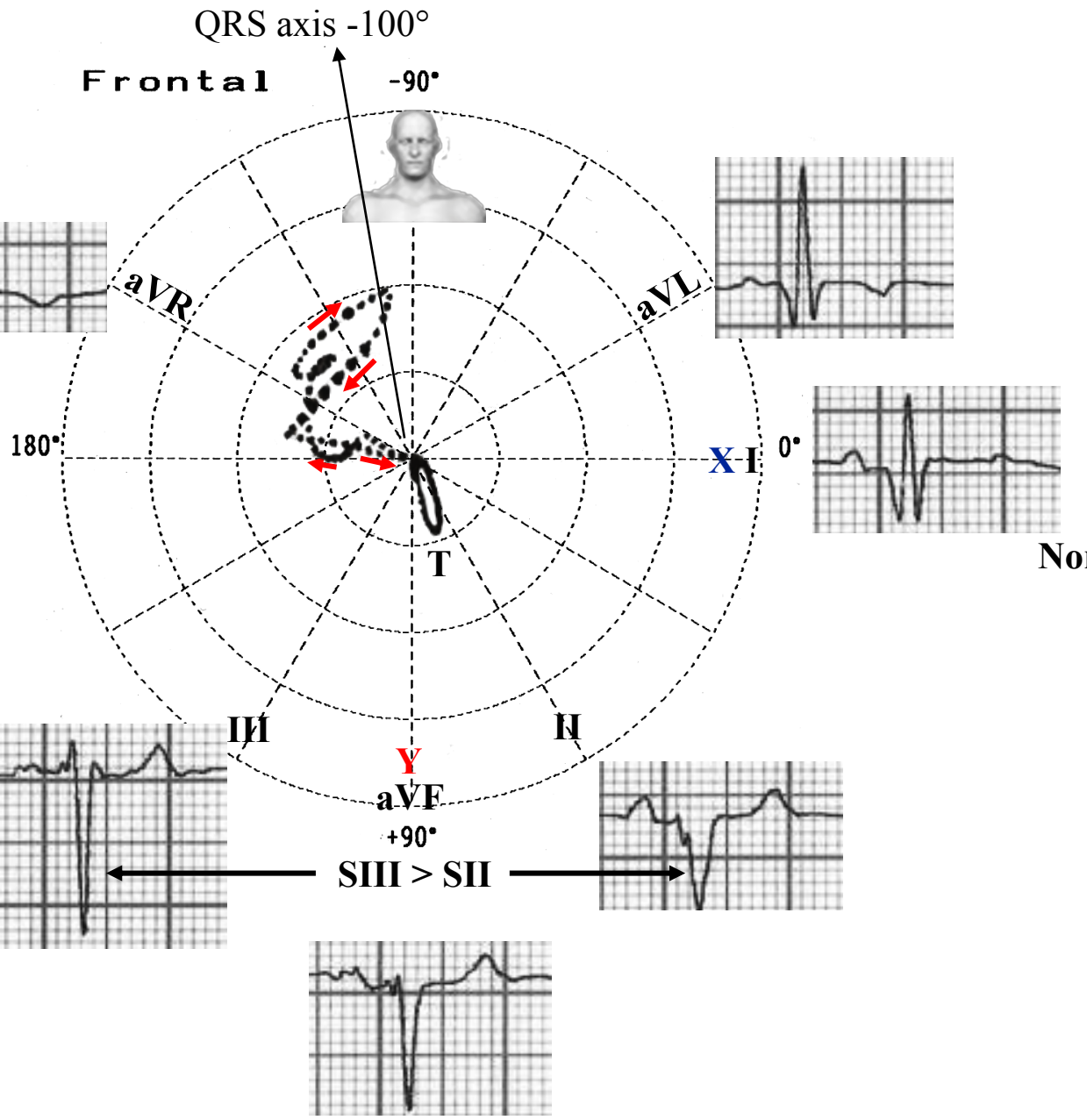


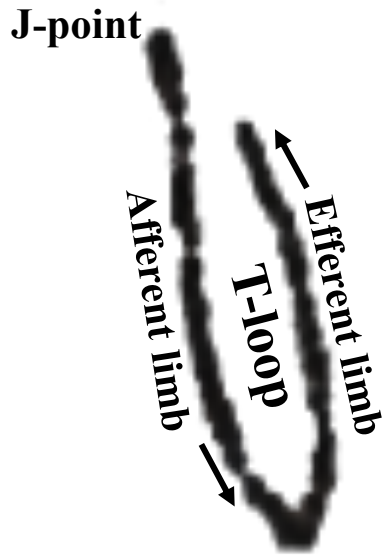


ECG/VCG correlation in the Frontal Plane

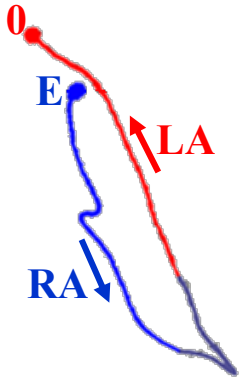




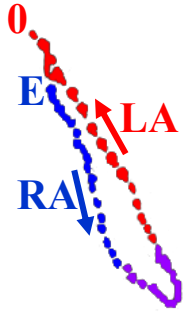
Magnified T-loop



Normal P-loop rotation



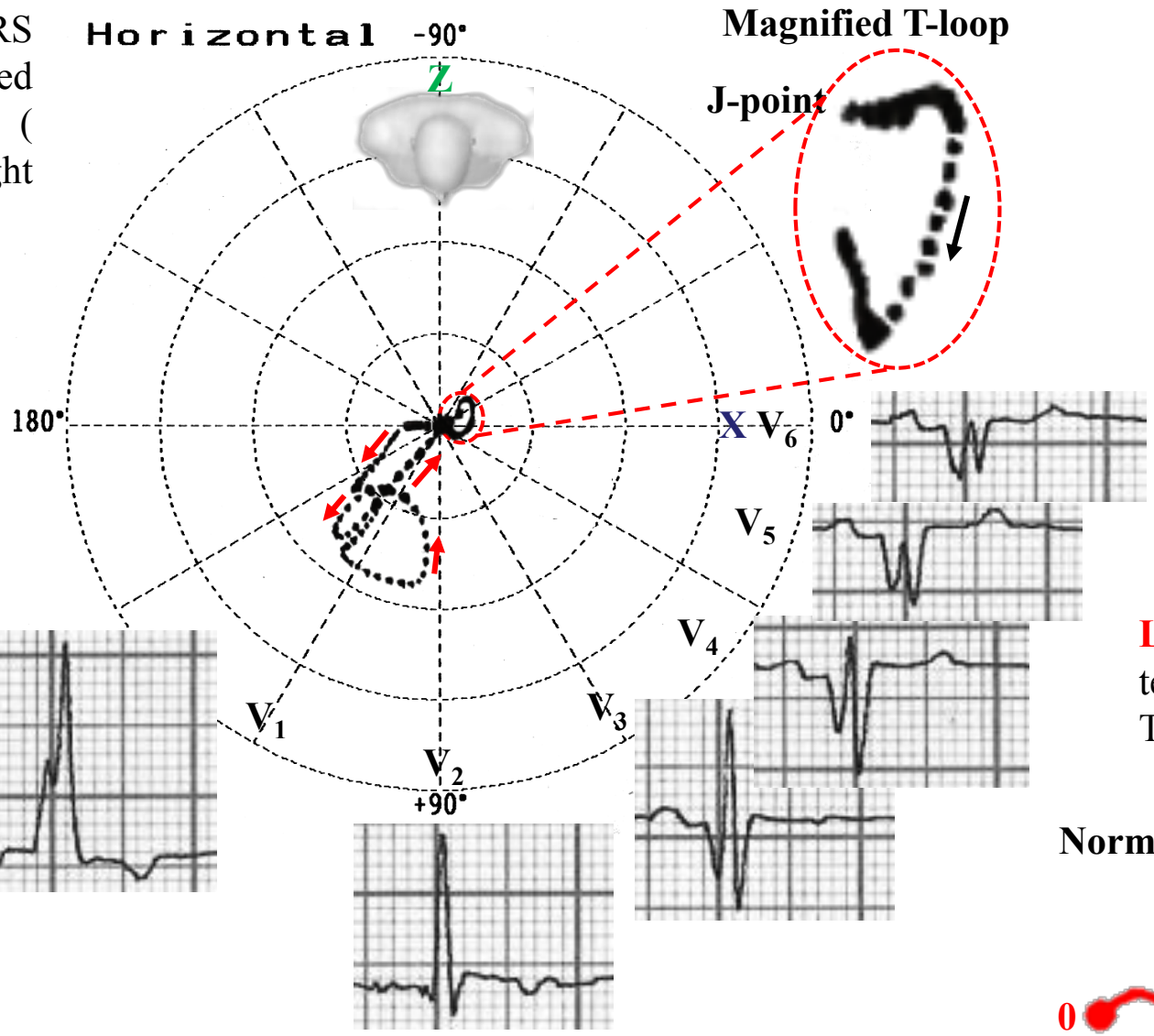
Magnified P-loop



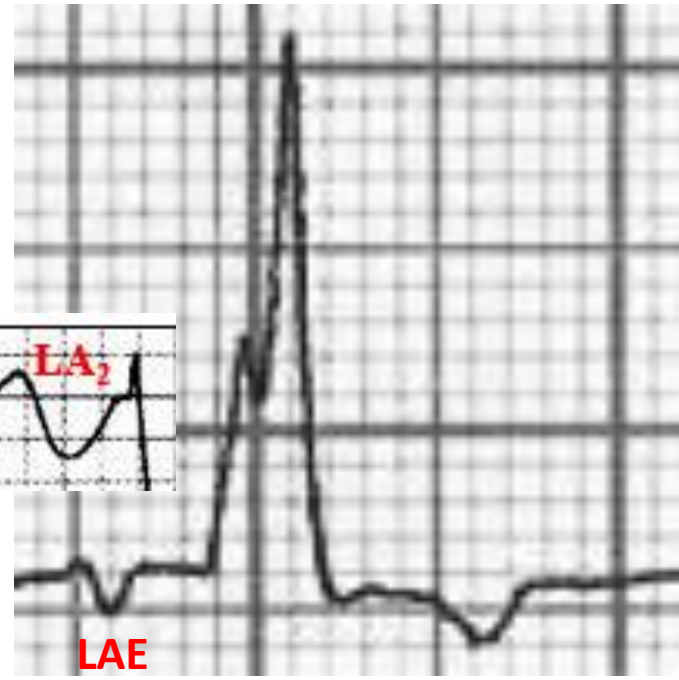
Normal P-loop

ECG/VCG correlation in the Horizontal Plane

QRSd = 121ms, prominent R wave in V1(= 15mm) QRS loop in eight, and dislocated to front and rightward (totally located on right anterior quadrant)
Anterolateral MI.



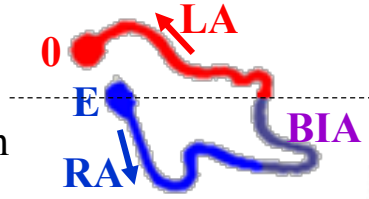
Magnified T-loop



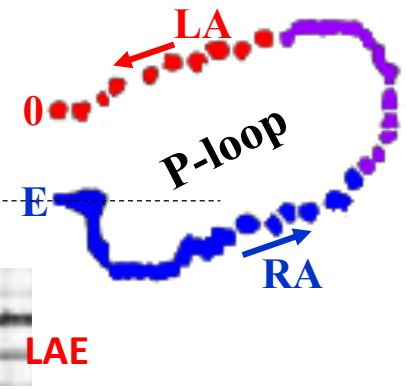
QRSd = 121ms

LAE: Positive Morris index (**Morris 1964**) (P terminal force in V1 mm/s) (0.04 mm/s). Terminal mode negative of P in V1 > 40 ms

Normal P-loop

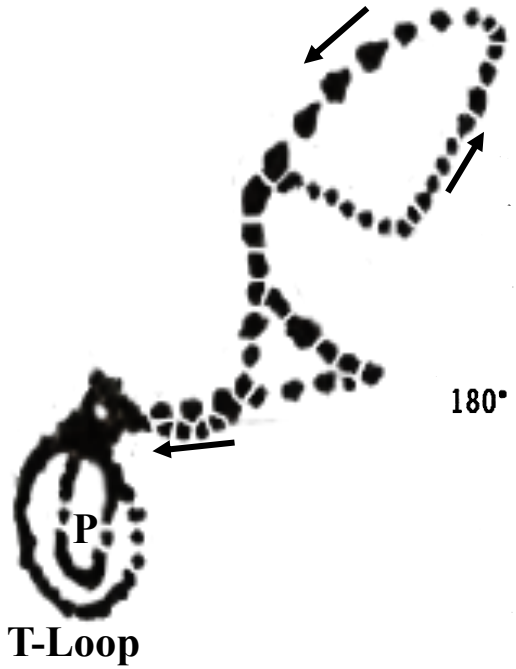


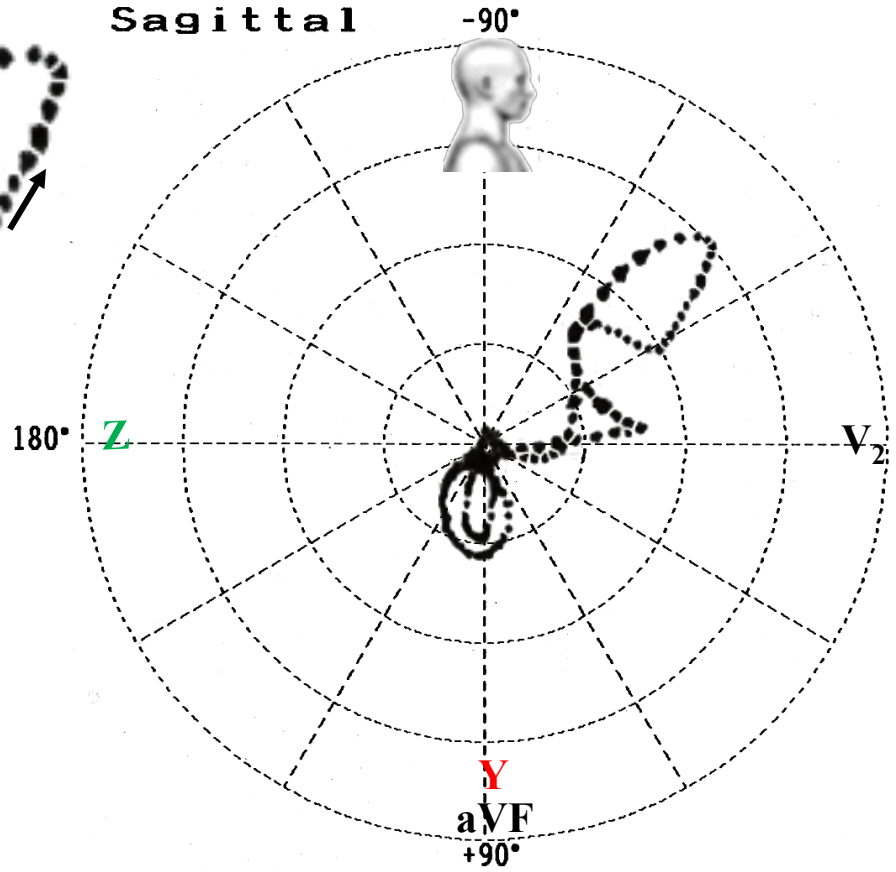
P-loop in the present case

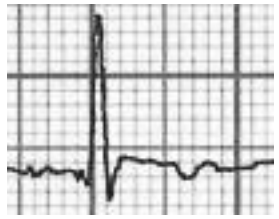


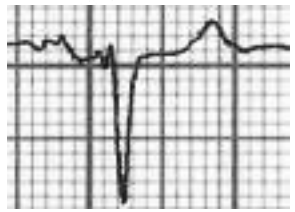
LAE: P-terminal force (PTF-V1) exceeding 0.04 mm/s. This is the terminal, negative part of the P wave in lead V1 expressed as the multiplication of its depth in millimeters and width in seconds (mm/s). The normal PTF-V1 does not exceed 0.04 s wide and 1mm deep, i.e., 0.04 mm/s.

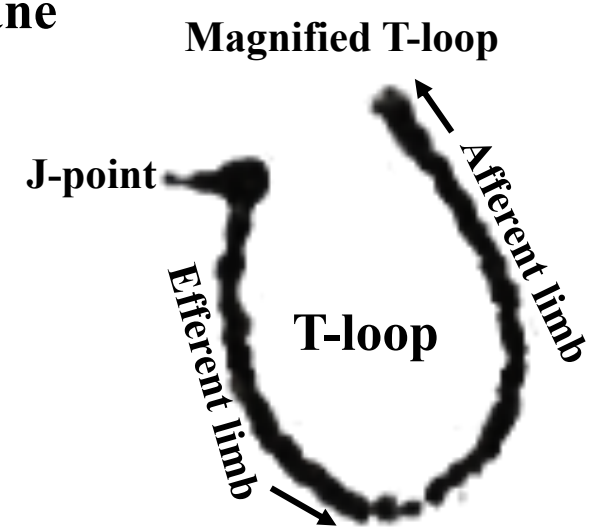
ECG/VCG correlation in the Right Sagittal Plane



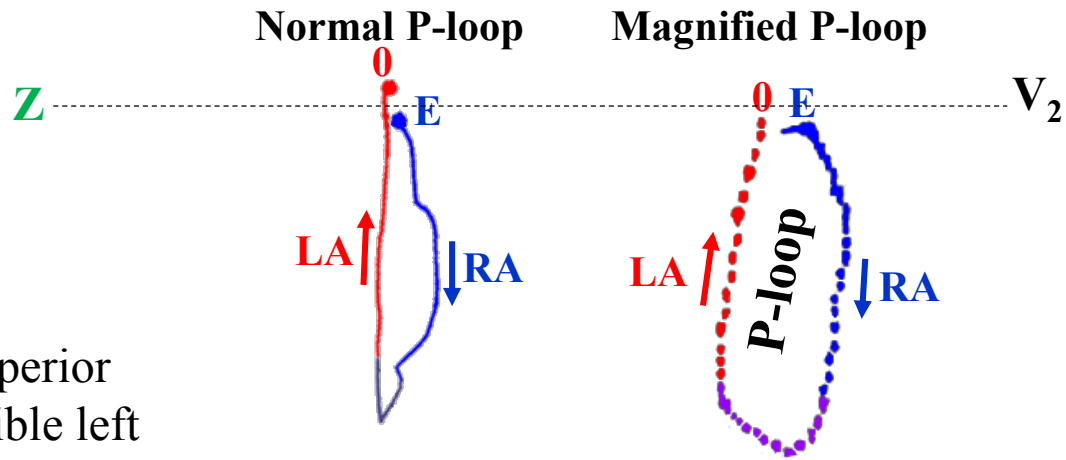








Primary T-loop: wide, rounded, small and with slow conduction efferent and afferent limbs with slow conduction(comets very close one to another)



Bizarre aspect of QRS loop with anterior (prominent anterior forces) and superior shift may be caused by association of lateral infarction + CRBBB, and possible left anterior fascicular block(LAFB).

Hello, here is my interpretation.

ECG 1. Anterior Q-wave MI with unfavorable ECG signs regarding the development of persistent left ventricular remodeling: 1) Elevated ST segment V1-V4 (dyskinesis of the anterior wall, typically the apex), 2) Lateral ST depression with T inversions (“strain pattern”), 3) P terminal force in V1. This is not an ECG pattern that we like to see at patient discharge. These patients will develop LV remodeling with dilatation and dysfunction. They should probably be treated with betablocker, ACE-inhibitor/ATR antagonist and mineralocorticoid inhibitor, but unfortunately no one has done a prospective study randomizing the patient to different kinds of medication (standard therapy vs. “heavy” anti-remodeling medication) based on the ECG, independently of the LV function.

ECG 2. Worsening of LV dysfunction (prominent PTF), new RBBB which masquerades the septal Q waves, lateral extension of the infarct (Q waves in lateral leads, high R/S ratio in V1-V2). Also LAFB. There can be ischemia in the lateral wall, because there is significant stenosis in a large diagonal branch and this is a border zone to the necrotic anterior wall and therefore, this area has increased demand for contraction (hyperkinesis in the region that is not infarcted). A new angiography could be valuable.

Best regards

Kjell Nikus

Portugues

Olá, aqui está a minha interpretação:

ECG 1. IM anterior com sinais eletrocardiográficas desfavoráveis por desenvolvimento de remodelamento ventricular esquerdo persistente:

1) Segmento ST elevado V1-V4 (disquinesia da parede anterior, tipicamente do ápice)
2) Depressão lateral do ST com inversões de T "Strain pattern"
3) Onda P com força terminal em V1 profunda e lenta: SAE.

Este não é um padrão de ECG que nós gostamos de ver na alta do paciente. Estes pacientes desenvolverão remodelamento do VE com dilatação e disfunção. Eles provavelmente deveriam ser tratados com β-bloqueador, inibidor da ECA / antagonista da ATR e inibidor dos mineralocorticóides, mas infelizmente até agora não temos um estudo prospectivo randomizando comparativo para os diferentes tipos de medicação (terapia padrão versus medicação "anti-remodelação" pesada) com base no ECG, independentemente da função do LV.

ECG 2. Piora da disfunção do VE (PTF proeminente), novo RBBB que mascara as ondas Q septais, extensão lateral do infarto (ondas Q em derivações laterais, alta relação R / S em V1-V2). Também se observva LAFB. Pode haver isquemia na parede lateral, pois há estenose significativa em um grande ramo diagonal e esta é uma zona limítrofe para a parede anterior necrótica e, portanto, esta área tem aumento da demanda por contração (hipercinesia na região que não é infartada) . Uma nova angiografia pode ser valiosa.

Cumprimentos

Kjell Nikus Finland

Final comments

How to explain this considerable change that occurred in ECG-2 in regard to ECG-1?

In the first ECG, a transmural anteroseptal or anterior myocardial infarction with ischemia in this wall is observed, associated to lateral wall ischemia(V5-V6-I and aVL). The anterior myocardial infarction is the consequence of obstruction in the middle third of LADA. But additionally, the report on hemodynamics tells us that the first diagonal branch was critically obstructed and was not treated. Probably, in this hiatus between the discharge and his return, the first diagonal branch that had a critical obstruction suffered total occlusion, causing additional transmural lateral infarction, which justifies prominent QRS anterior forces in V1 and V2, with QRS complex broadening by addition of CRBBB. We believe that there was an inadequate management. Instead of the discharge, he should have been proposed to undergo ADA and Dg revascularization procedure with Coronary Artery Bypass Grafting (CABG or "cabbage"). In ECG2, the bizarre aspect of QRS loop with clear right (lateral MI), anterior (prominent anterior forces) and superior shift may be caused by association of lateral infarction + CRBBB, and possible left anterior fascicular block(LAFB) based on the superior shift, initial q waves in I and aVL, and notch in the descending slope of S in the inferior wall. In brief:

Left atrial enlargement (LAE)

Prominent Anterior QRS Forces(PAF): caused by lateral MI + CRBBB (See in next slides the possible causes of QRS PAF). The presence of prominent the R wave in V1 is due to the lateral MI and not to the involvement of inferobasal segment of inferior wall (old posterior wall) (**Goldwasser 2015)** The inexistence of the dorsal wall is the end of an electrocardiographic dogma (**Bayés de Luna 2015**).

Right shift of QRS forces: by loss of left lateral wall (lateral MI).

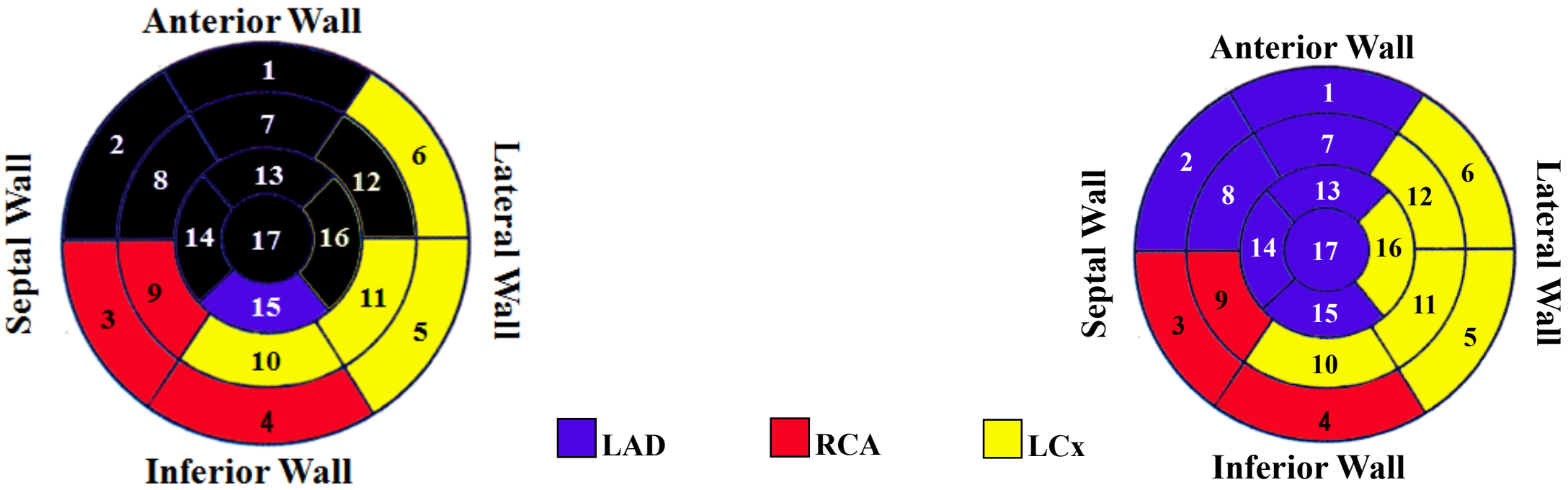
Upward shift of QRS forces: consequence of additional LAFB. This diagnosis is reinforced by SIII>SII, rIII > rII (it indicates CCW rotation in the FP), qR pattern in I and aVL, and very prolonged R-wave peak-time in aVL (>45 ms). In this case, 80 ms.

Bifascicular block (RBBB+ LAFB).

Conclusion diagnosis on ECG2:

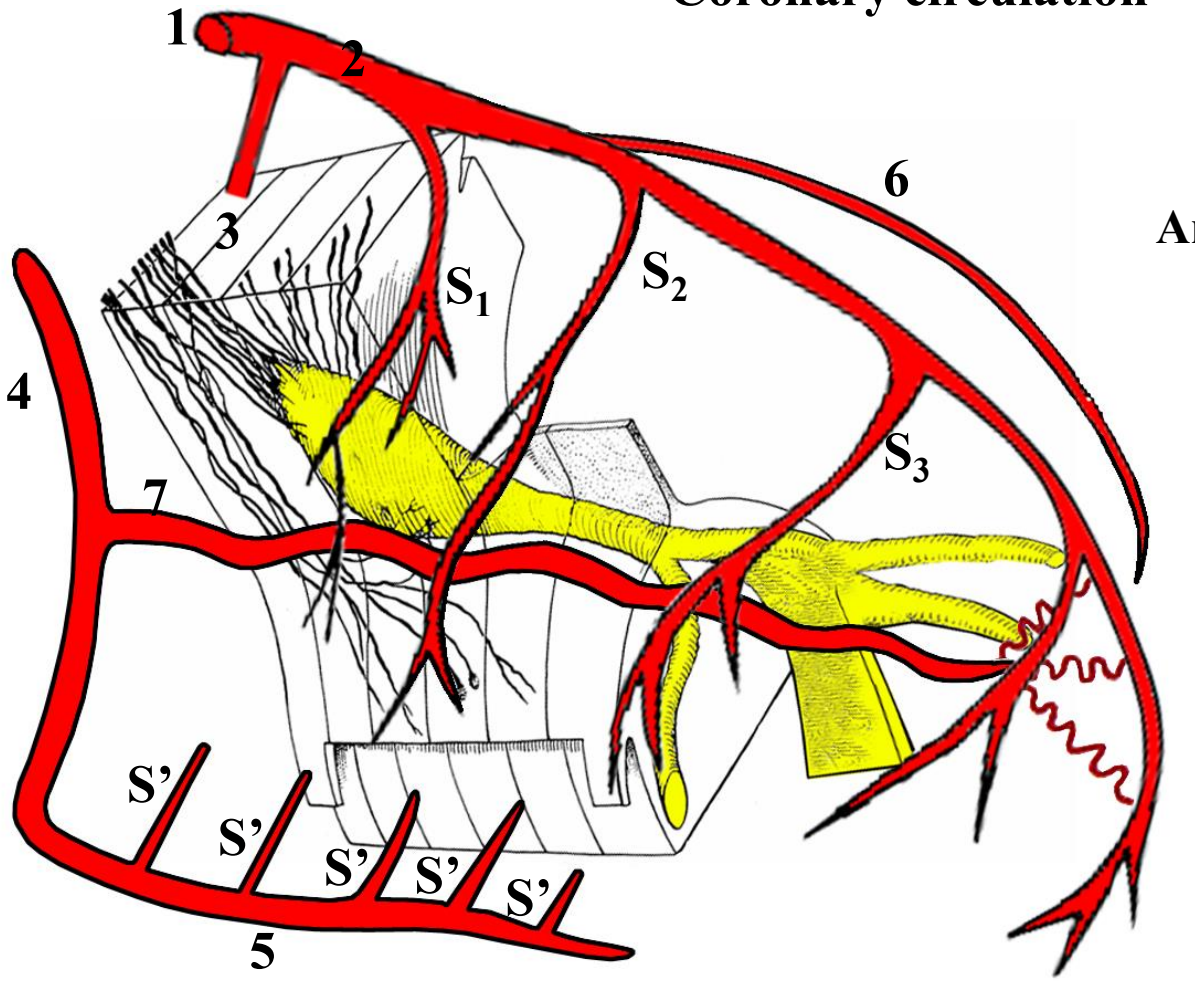
1) LAE
2) Anterolateral MI
3) PAF by association of lateral infarction, and CRBBB; QRS loop of bizarre aspect located at the front (lateral MI + RBBB), the right (lateral MI) and up (associated to LAFB).
4) LAFB: It is the most common type of intraventricular conduction defect seen in acute anterior MI, and the LADA is usually the culprit vessel (**Miller 1973; Chandrashekhar 1991**).
5) Bifascicular block: RBBB + LAFB
6) Ischemic primary T-wave/T-loop: symmetric, wide, rounded, small and with slow conduction of efferent and afferent limbs (comets very close)

Segments affected in the present case



Observation: when first diagonal artery is large some segments of lateral wall are affected in territories of LCx. See next slide.

Coronary circulation



Anterior Septal Perforator Branches

S_1: First Septal Perforator branch
S_2: Second Septal Perforator
S_3: Third Septal Perforator

S': Posterior Septal Perforators

1. Left Main Coronary Artery (**LMCA)**
2. Left Anterior Descending Artery (**LAD)**
3. Left Circumflex Coronary Artery (**LCX)**
4. Right Coronary Artery (**RCA)**
5. Posterior Descending Artery (**PDA**). In this case is supplied by the RCA, then the coronary circulation can be classified as "right-dominant“
6. First Diagonal (**Dg**)
7. Acute Marginal (**A. Mg)**

Considerations about the presence of LAFB in this case

As early as 1937, Ashman and Hull ascribed left axis deviation to coronary artery disease(CAD) and LVH (**Ashman 1937**), and a decade later Wilson reinforced the latter concept (**Wilson 1947**). Shortly thereafter, the idea of "peri-infarction block" was introduced," and attention was called to Wilson's comments on intraventricular block (**Wilson 1944**). The notion of pen-infarction block emphasized the importance of CAD as a cause of left axis deviation, and provided a seemingly rational explanation for it (**First 1950**). In the vast majority of instances it can be differentiated from bundle branch block and from intraventricular block of other types. The importance of the lesion is emphasized, as the name implies, by its association with (usually) an old MI.

The relationship of the leftward shift of the frontal plane(FP) QRS axis to pathologic changes of myocardial fibrosis was reported latter by Grant in 1956 (**Grant 1956**) and confirmed by others. It is generally agreed that left axis deviation is an ECG conduction disturbance which connotes the presence of underlying organic heart disease, usually myocardial fibrosis or degenerative secondary mainly to CAD, cardiomyopathies, myocarditis(example Chagas myocarditis), Lenègre and Lev disease, congenital heart disease such as endocardial cushion defect or ostium primum atrioventricular septal defects (**Caro 2015**), tricuspid atresia (**Calabró 1981**), and occasionally, pulmonary emphysema are also associated with extreme left axis deviation on the FP. Mauricio Rosenbaum (**Rosenbaum 1970**) has established that a lesion which involves the left anterior superior fascicle/division of the left bundle branch accounts for the development of severe LAD (QRS axis ≥45°), and has suggested myocardial fibrosis secondary to CAD as its most common cause. If a sufficient number of fibers of the LAF are interrupted, the sequence of electrical activation will be altered in such a manner that the initial QRS forces will be shifted inferiorly and to the right, and the terminal forces will be oriented superiorly and to the left. Notwithstanding when LAFB is associated with lateral MI the QRS axis on the frontal plane (FP) is dislocated to the right superior quadrant such as in the present case (QRS axis -100°). This is to be distinguished from the ECG of patients with inferior MI in whom the loss of inferior forces results in unopposed superiorly directed forces, and consequently, the QRS axis on FP is deviated to the left and superiorly. Precise correlation of left axis deviation in a resting ECG with specific myocardial lesions and subsequent valid prognostic information has been tenuous. In patients with suspected CAD referred for stress testing, LAFB is associated with increased risk of cardiac death. This risk is persistent after adjustment for major clinical data and abnormalities on the stress echocardiogram. Therefore, isolated LAFB should not be considered a benign electrocardiographic abnormality in these patients (**Biagini 2006**). A significant proportion of patients with this ECG finding to have an abnormal response to graded submaximal exercise testing, indicating the presence of factors causing a disparity between myocardial oxygen supply and demand." One of these factors is CAD. Additionally, a positive correlation between exercise stress testing and anatomic abnormalities demonstrated by selective coronary arteriography has been reported." If, however, the results of an exercise stress test are suggestive of CAD, additional investigation with selective coronary arteriography and left ventricular angiography seems warranted.

Possible Causes for Prominent QRS Anterior Forces

In the presence of PAF in the anterior wall (tall R waves) in the right and/or middle precordial leads V 1 through V 3 or V 4, the following differential diagnosis should be excluded clinico-electro-vectorcardiographically (**Zema 1990**).

I. PAF are observed in only 1 % of normal subjects (**Mattu 2001**). There are two main types: Normal variant with marked counterclockwise rotation of the heart around the longitudinal axis of the heart resulting in a shifting of the transition area (R = S) early, i.e., to the right of the precordial lead V2 (**Yanagisawa 1981 ; Mori 1992 ; Paparella 1987**). Athlete's heart (**Ferst and Chaitman 1984**).
II. Misplaced precordial leads as cause of PAF (**MacKenzie 2004; Mattu 2001**)
III. MI previously known as strictly posterior, dorsal, high posterobasal. Currently lateral MI (**Bayés de Luna 2006**).
IV. Right ventricular hypertrophy (RVH): vectorcardiographic types A (**Brohet 1990; Suzuki 1978; Hugenholtz 1964**) and B (**Ellison and Restieaux 1972a, b**)
V. Diastolic, volumetric or eccentric left ventricular hypertrophy (LVH).
VI. Secondary to septal hypertrophy (magnitude of increase of 1AM vector) and CCW heart rotation around the longitudinal axis (**Cabrera 1960; Donoso 1955**).
VII. Combined or biventricular hypertrophy (**Elliott 1963**).
VIII. Complete RBBB (**Baydar. 1965;Chen 1980**)
IX. Pre-excitation variant of Wolff-Parkinson-White syndrome, with accessory anomalous pathways (Kent fibers), located in a posterior location (Type A): right posterior, right and left posterior paraseptal and left posteriorparaseptal and left posterior pre-excitation (**Chung 1965**).
X. HCM: both obstructive and non-obstructive forms (**Pérez-Riera 2013**);
XI. Progressive muscular dystrophy of childhood (Duchenne's cardiomyopathy), Duchenne's muscular dystrophy, X-linked muscular dystrophy, pseudohypertrophic muscular dystrophy, childhood muscular dystrophy (**Secchi 1982; Yotsukura 1999**)
XII. Endomyocardial fibrosis (**Tobias 1992**)
XIII. Dextroposition. Example: left pneumonectomy (**Pérez Riera 2011**).
XIV. Left Septal Fascicular Block
XV. A combination of the above, such as the present case: lateral MI associated with RBBB.

References

1. Ashman R, Hull E: Essentials of Electrocardiography for the Student and Practitioner of Medicine. New York, MacMillan, 1937, p 83
2. Baydar ID, Walsh TJ, Massie E. A vectorcardiographic study of right bundle branch block with the Frank lead system. Clinical correlation in ventricular hypertrophy and chronic pulmonary disease. Am J Cardiol. 1965;15:185–94.
3. Bayés de Luna A, Wagner G, Birnbaum Y, International Society for Holter and Noninvasive Electrocardiography, et al. A new terminology for left ventricular walls and location of myocardial infarcts that present Q wave based on the standard of cardiac magnetic resonance imaging: a statement for healthcare professionals from a committee appointed by the International Society for Holter and Noninvasive Electrocardiography. Circulation. 2006;114(16):1755–60.
4. Bayés de Luna A, Rovai D, Pons Llado G, et al. The end of an electrocardiographic dogma: a prominent R wave in V1 is caused by a lateral not posterior myocardial infarction-new evidence based on contrast-enhanced cardiac magnetic resonance-electrocardiogram correlations. Eur Heart J. 2015;36(16):959-64.
5. Biagini E, Elhendy A, Schinkel AF, et al. Prognostic significance of left anterior hemiblock in patients with suspected coronary artery disease. J Am Coll Cardiol. 2005;46(5):858-63.
6. Brohet CR. Special value of the vectorcardiogram in pediatric cardiology. J Electrocardiol. 1990;23(Suppl):58–62.
7. Cabrera E, Gaxiola A. Diagnostic contribution of the vectorcardiogram in hemodynamic overloading of the heart. Am Heart J. 1960;60:296–317.
8. Calabrò R, Elia LR, Marsico L, Marsico F. Electrocardiographic and vectorcardiographic aspects of tricuspid atresia. G Ital Cardiol. 1981;11(5):569-76.
9. Caro M, Conde D, Pérez-Riera AR, de Almeida AP, Baranchuk A. The electrocardiogram in Down syndrome. Cardiol Young. 2015;25(1):8-14.
10. Chandrashekhar Y, Kalita HC, Anand IS. Left anterior fascicular block: an ischaemic response during treadmill testing. Br Heart J. 1991;65(1):51-2.
11. Chen CH, Kawai C, Sakurai T, Fujita M, Nobuyoshi M. The RSR' pattern in right chest leads in hypertrophic cardiomyopathy: vectorcardiographic analysis. Jpn Circ J. 1980;44(9):734–9
12. Chung KY, Walsh TJ, Massie E. Wolff-Parkinson-White syndrome. Am Heart J. 1965;69:116–33.
13. Donoso E, Sapin SO, Braunwald E, Grishman A. A study of the electrocardiogram and vectorcardiogram in congenital heart disease. II. Vectorcardiographic criteria for ventricular hypertrophy. Am Heart J. 1955;50(5):674–93.

14. Elliott LP, Taylor WJ, Schiebler GL. Combined ventricular hypertrophy in infancy: vectorcardiographic observations with special reference to the Katz-Wachtel Phenomenon. Amer J Cardiol. 1963;11:164.
15. Ellison RC, Restieaux NJ. Chapter 7: Vectorcardiography in congenital heart disease a method for estimating severity. Philadelphia/London/Toronto: W. B. Saunders Company; 1972a. p. 75–85.
16. Emery JL, Mithal A. Weights of cardiac ventricles at and after birth. Br Heart J. 1961;23:313-6.
17. Ferst JA, Chaitman BR. The electrocardiogram and the athlete. Sports Med. 1984;1(5):390–403.
18. First SR, Bayley RH, Bedford DR. Peri-infarction block; electrocardiographic abnormality occasionally resembling bundle branch block and local ventricular block of other types. Circulation. 1950;2(1):31-6.
19. Goldwasser D, Senthilkumar A, Bayés de Luna A, et al. Lateral MI Explains the Presence of Prominent R Wave (R ≥ S) in V1. Ann Noninvasive Electrocardiol. 2015;20(6):570-7.
20. Grant RP. Left axis deviation; an electrocardiographic-pathologic correlation study. Circulation. 1956;14(2):233-49.
21. Hugenholtz PG, Gamboa R. Effect of chronically increased ventricular pressure on electrical forces of the heart. A correlation between hemodynamic and vectrocardiographic data (frank system) in 90 patients with aortic or pulmonic stenosis. Circulation. 1964;30:511–30.
22. MacKenzie R. Tall R, wave in lead V1. J Insur Med. 2004;36(3):255–9.
23. Mattu A, Brady WJ, Perron AD, Robinson DA. Prominent R wave in lead V1: electrocardiographic differential diagnosis. Am J Emerg Med. 2001;19(6):504–13.
24. Miller AB, Naughton J, Gorman PA. Left axis deviation: diagnostic contribution of exercise stress testing. Chest. 1973;63(2):159-64.
25. Mori H, Kobayashi S, Mohri S. Electrocardiographic criteria for the diagnosis of the left septal fascicular block and its frequency among primarily elderly hospitalized patients. Nihon Ronen Igakkai Zasshi. 1992;29(4):293–7.
26. Morris JJ Jr, Estes EH Jr, Whalen RE, Thompson HK Jr, Mcintosh HD. P-wave analysis in valvular heart disease. Circulation. 1964;29:242-52
27. Paparella N, Alboni P, Cappato R, et al. Prominent anterior QRS forces: clinical, electrocardiographic and prospective study. J Electrocardiol. 1987;20(3):233–40.
28. Pérez Riera AR, Ferreira C, Ferreira Filho C, et al. Electrovectorcardiographic diagnosis of left septal fascicular block: anatomic and clinical considerations. Ann Noninvasive Electrocardiol. 2011;16(2):196–207.
29. Pérez-Riera AR, de Lucca AA, Barbosa-Barros R, et al. Value of electro-vectorcardiogram in hypertrophic cardiomyopathy. Ann Noninvasive Electrocardiol. 2013;18(4):311–26.
30. Rosenbaum MB. The hemiblocks: diagnostic criteria and clinical significance. Mod Concepts Cardiovasc Dis. 1970;39(12):141-6.

31. Secchi MB, Wu SC, Obbiassi M, Oltrona L, Folli G. Electro-vectorcardiographic study in Duchenne de Boulogne progressive muscular dystrophy. Arch Mal Coeur Vaiss. 1982;75(11): 1297–309.
32. Suzuki K, Toyama S. Vectorcardiographic criteria of high posterior infarction: differentiation from normal subjects, right ventricular hypertrophy and primary myocardial disease. J Electrocardiol. 1978;11(2):159–63.
33. Tobias NM, Moffa PJ, Pastore CA, et al. The electrocardiogram in endomyocardial fi brosis. Arq Bras Cardiol. 1992;59(4):249–53.
34. Wilson FN, Johnston FD, Rosenbaum FF, et al. The precordial electrocardiogram. Am Heart J. 1944;27:19-85.
35. Wilson FN, Rosenbaum FF, Johnston FD: The interpretation of the ventricular complex in the electrocardiogram. Adv Intern Med 1947;2:1-63.
36. Yanagisawa N, Honda M, Watanabe H, Nakamura F, Higuchi Y, Wada T. Counterclockwise rotation of the heart: a correlative study with tomographic echocardiography. J Cardiogr. 1981;11(3):881–9.
37. Yotsukura M, Yamamoto A, Kajiwara T, et al. QT dispersion in patients with Duchenne-type progressive muscular dystrophy. Am Heart J. 1999;137(4 Pt 1):672–7.
38. Zema MJ. Electrocardiographic tall R waves in the precordial leads. Comparison of recently proposed ECG and VCG criteria for distinguishing posterolateral myocardial infarction from prominent anterior forces in normal subjects. J Electrocardiol. 1990;23(2):147–56.