

Paciente femenina de 73 años con cuadro isquémico agudo de 3 horas de evolución.

Dr. Raimundo Barbosa Barros

Portuguese

Prezado Dr Andrés estou enviando mais um caso para sua análise. Trata-se de uma paciente do sexo feminino, 73 anos de idade, que deu entrada na emergência com sintomatologia isquêmica iniciada 3 horas antes da admissão. Hemodinamicamente estável. Como a sala de hemodinâmica não estava disponível a paciente foi submetida à terapia fibrinolítica com sinais de reperfusão (desaparecimento da dor, resolução > 50% do supra de ST e reversão do quadro de dissociação AV).

Questionamentos:

- 1- IAM inferior e VD associado à BRD? Parede posterior? (antigo IAM dorsal)
- 2- Existe bloqueio divisional (BDAM?)

Posteriormente enviarei a coronariografia

Atenciosamente
Raimundo Barbosa

English

Dear Dr Andrés I am sending a case for your analysis. One is about a patient of the feminine gender, 73 years of age, that gave entered in the emergency room with ischemic symptomatology initiate 3 hours before the admission.

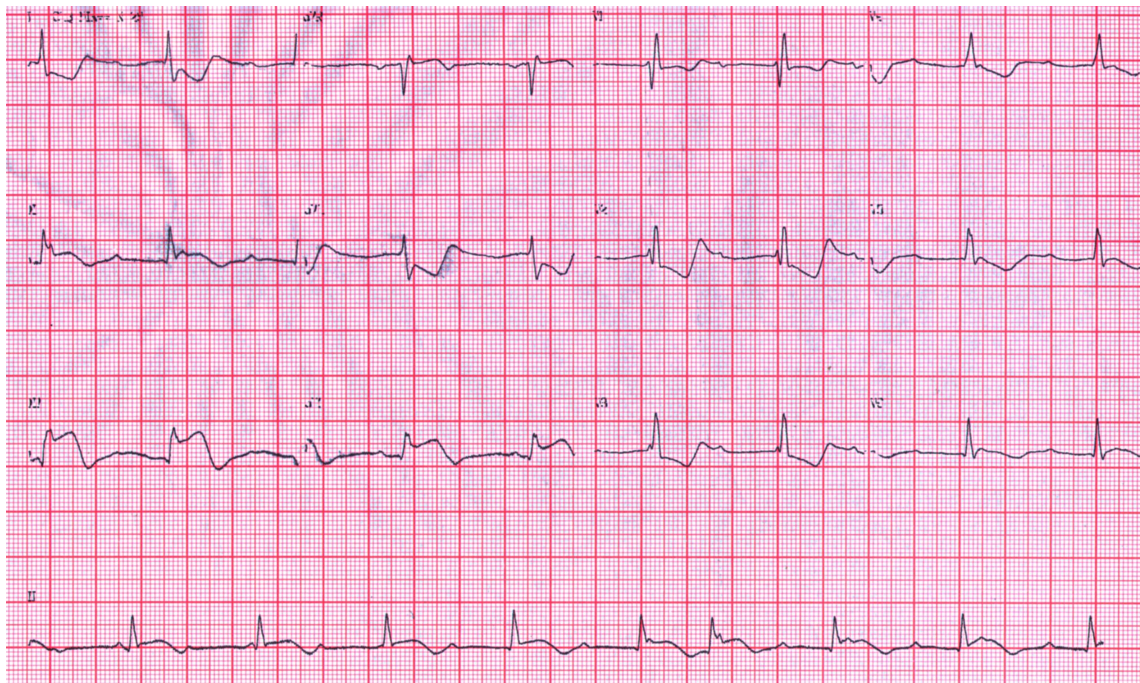
Steady Hemodically. As the hemodinamic room was not available the patient submitted to the fibrinolytic therapy with reperfusion signals (disappearance of pain, resolution > 50% ST segment elevation and reversion of the picture of AV dissociation).

Questionings:

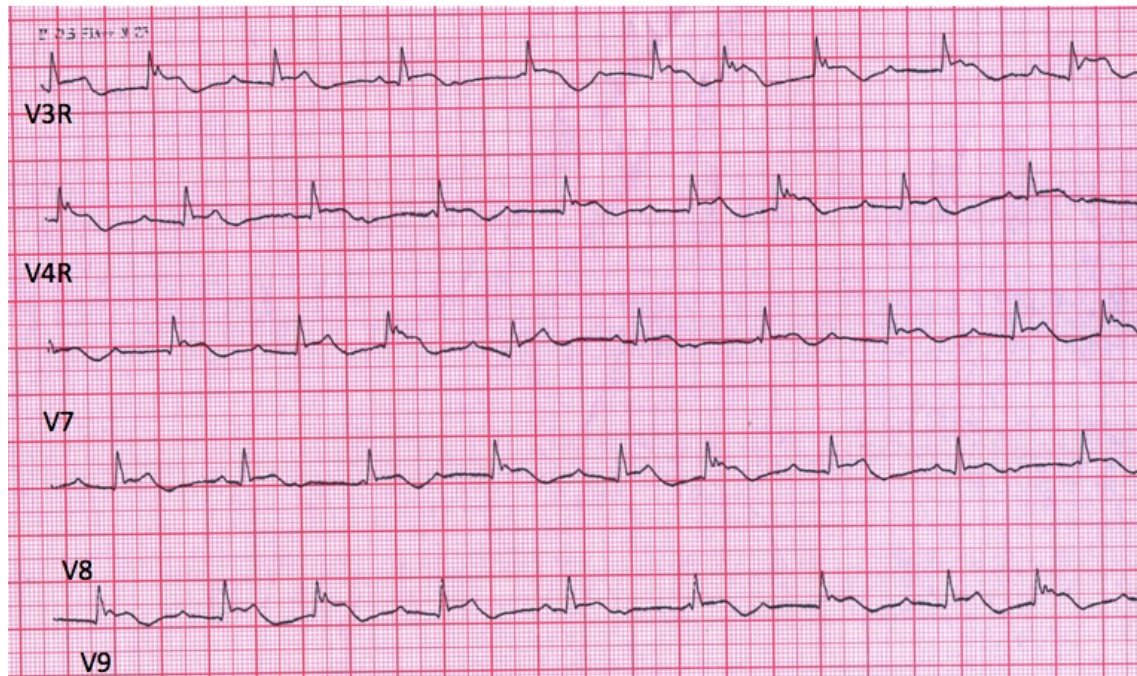
1. Inferior AMI and RV associated with the RBBB? Or posterior wall acute MI? (old dorsal infarction)
2. Exist left septal fascicular block??

Later I will send the coronariography

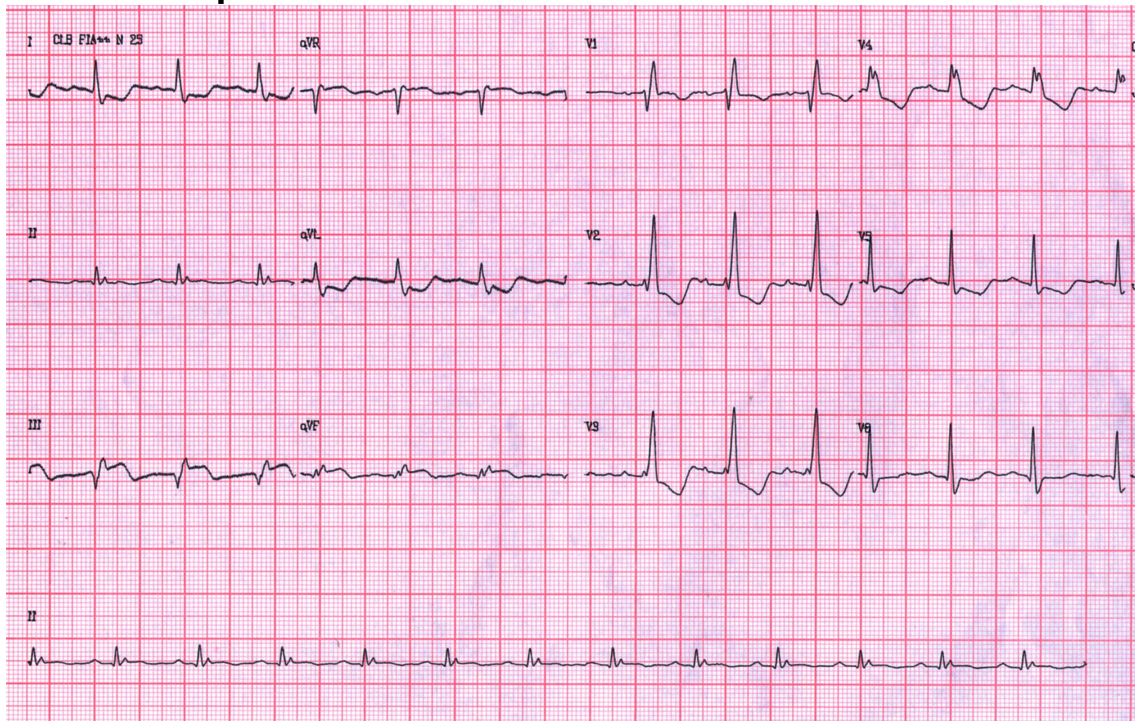
ECG N° 1



ECG N° 2



ECG N° 3. Após ESTREPTOQUINASE



OPINIONES DE COLEGAS

Estimado Raimundo

Que bonito caso!

ECG 1: IAM infero-postero-lateral con signos indirectos de compromiso eléctrico de VD (ST elevation III>II), ST depresión V2>V1), disociación AV sugiriendo activación de reflejo de Bezold Jarisch (mecanorreceptores), RRRB.

ECG 2: Signo directo de compromiso de VD (ST elevación V3R)

ECG 3: isquemia por reperfusión de la división septal de la rama derecha (Ver Riera et al), produciendo LSFB (V2>15mm), PAF.

Lo saludo muy atentamente y gracias por compartir esta secuencia tan bonita.

Adrián Baranchuk

Querido Adrián:

Entiendo que en el ECG 1 manifiestas que existe bloqueo de la rama derecha, aunque se lee RRRB mas es RBBB de la sigla inglesa Right Bundle Branch Block? Pienso que fue un error de digitación. Es así?

En el ECG 3 manifiestas que hay isquemia por reperfusión de la división septal de la rama derecha (Ver Riera et al). Pienso que quisiste decir isquemia por reperfusión de la división septal de la rama izquierda y no de la derecha. Es así?

Finalmente, en este trazado (ECG 3) no manifiestas si existe RBBB. ¿Existe a tu criterio bloqueo de rama derecha en el ECG 3?

Después de tu respuesta y la de otros haremos nuestro análisis del caso.

Andrés R. Pérez Riera

Dear Andres and friends,

My interpretation is an acute inferoposterior MI (old terminology; now probably called inferolateral) with additional RV infarction evidenced by the ST elevation in right-sided chest leads. The lateral (or posterior) component is evidenced by the marked ST depression in V2-3 and ST elevation in V7-8. The greater ST elevation in lead III compared to lead II along with the RV MI suggests a high-grade RCA occlusion. There's also a RBBB. The rhythm is sinus

tachycardia (~100 bpm) along with high-grade AV block, incomplete AV dissociation (occasional captures) and a junctional escape rhythm (~55 bpm).

Frank Yanowitz

Si a todas tu preguntas (RBBB) y LSFB (Subdivision rama izquierda) era tarde, y los dedos fueron más rápido que la mente.

Entiendo tu explicación de por qué NO es un LSFB (solo ramas de LAD irrigan la rama septal izquierda). Es posible que en persona con oclusión previa de LAD, la oclusión aguda de la RCA produzca LSFB por compromiso de circulación colateral heterocoronaria?

Por favor, explica una vez más, la interpretación de PAF en ausencia de LSFB (tú mencionas expansión del VI...eso genera una R más alta?). Si entendí bien, por favor sugiere alguna referencia para leer al respecto ya que me resulta fascinante que la expansión del VI se refleje en el ECG como R alta.

Como siempre, gracias por tu clase. Frank también ha demostrado ser uno de los grandes maestros de la electrocardiografía (Thanks Frank!).

Adrian Baranchuk

Dear Frank: Thank a lot for your valuable opinion.
Only one question: what do you think about the Prominent Anterior Forces in ECG-3? Why in ECG-1 R' voltage V2 lead = 8mm and R' V2 of ECG-3 = 18mm?

Thank in advance dearest friend

Andrés R. Pérez Riera.

Prezado Frank: Obrigado pela sua valiosa opinião.
Apenas uma pergunta: O que você pensa acerca das Forças Anteriores Proeminentes no ECG-3. Porque no ECG-1 a voltagem da R' de V2 é apenas de 8 mm e a R' de V2 do ECG -3 é de 18mm?

Obrigado queridíssimo amigo

Andrés R. Pérez Riera.

Efectivamente, se trata de una oclusión proximal de la coronaria derecha con afectación por tanto del ventrículo derecho. Ante este tipo de oclusión habitualmente encontraremos ascenso del ST en II, III y aVF con ascenso en III>II, ascenso del ST en precordiales derechas (V3R,V4R...) y en V1 ascenso del ST o un ST isoelectrico. Incluso en ocasiones podemos registrar ascenso de V1-V3 con V1>V3. Por ejemplo, en la oclusión de una CD corta encontraremos este tipo de patente.

En este caso, el ascenso en II, III y aVF (III>II) se explica por oclusión de la CD, el ascenso en V1-V3 por afectación del VD, de V3-V4 por la gran dilatación del VD que se expone fuertemente a precordiales intermedias y el ascenso de V5-V6 se explica por ser CD muy dominante llegando a irrigar la pared lateral baja del VI.

Un ECG parecido podemos registrar en la oclusión de la DA proximal que da la vuelta a la punta cardiaca e irriga la los segmentos basales que habitualmente pertenecen a la CD.

Un saludo a todos,

Javier García Niebla.

Versión castellana

Efectivamente como ya lo mostrara Edgardo, Fiol y col. desarrollaron un algoritmo secuencial basado en la elevación o depresión del segmento ST en diferentes derivaciones para predecir la arteria ocluida (si la derecha o la circunfleja) en casos de infartos agudos infero-posteriores.

Infarto agudo de miocardio (IMA) de la pared infero-dorsal es causado por oclusión de la arteria coronaria derecha o la circunfleja. El pronóstico depende de la arteria ocluida. Consecuentemente, la presunción de la arteria ocluida basado en el ECG realizado en la admisión posee importancia clínica.

Los autores analizaron el ECG y la angiografía de 63 pacientes consecutivos en fase aguda de infarto con elevación del segmento

ST en las derivaciones inferiores (II, III, y aVF) y con oclusión de vaso único. Estudiaron especificidad, sensibilidad y los valores predictivos positivos y negativos de los diferentes criterios del ECG (elevación o depresión del ST) en forma individual y en conjunto para encontrar un algoritmo que mejor pudiese predecir la arteria ocluida.

Los siguientes criterios de ECG fueron incluidos en un algoritmo de tres pasos sucesivos:

- 1) Mudanzas en el ST en la derivación DI
- 2) Relación entre la elevación del ST en DIII/DII
- 3) Relación de la suma de las depresiones del ST en las precordiales de V1 a V3 con la suma de las elevaciones de los ST en las inferiores (II, III, y aVF).

La aplicación de este algoritmo en pasos sucesivos reveló la arteria responsable en más del 95% de los pacientes. Los pocos pacientes en que el algoritmo falló obedecía a la presencia de una circunfleja muy dominante con depresión del ST ≥ 0.5 mm en DI.

Los autores concuyeron que un cuidadoso análisis del ECG en casos de infarto agudo inferoposterior puede llevar a identificar la arteria responsable.

English version

Acute myocardial infarction (AMI) of the inferoposterior wall is due to occlusion of the (RCA) or the LCx coronary artery. The outcome of patients depends mainly on the culprit artery. Therefore, the presumptive prediction of a culprit artery based on the ECG recorded at admission is of clinical importance.

Fiol et al develop a sequential algorithm based on the "ups and downs" of the ST segment in different leads to predict the culprit artery (RCA vs LCx) in cases of inferoposterior AMI.

The authors analyzed ECG and angiographic findings of 63 consecutive patients with an evolving AMI with ST elevation in the inferior leads (II, III, and aVF) and a single-vessel occlusion.

Specificity, sensitivity, and positive and negative predictive values of different ECG criteria (ups and downs of the ST segment) were studied individually and in combination to find an algorithm that would best predict the culprit artery.

The following ECG criteria were included in the 3-step algorithm:

- 1) ST changes in lead I
- 2) The ratio of ST elevation in lead III to that in lead II
- 3) The ratio of the sum of ST depression in precordial leads to the sum of ST elevation in inferior leads (summation operator downward arrow ST in leads V(1) to V(3))/(summation operator upward arrow ST in leads II, III, and aVF)].

Application of this sensitive algorithm suggested the location of the culprit coronary artery (RCA vs LCx) in 60 of 63 patients (>95%). The few patients in whom this algorithm did not work were those with a very dominant LCx that presented ST depression of ≥ 0.5 mm in lead I.

The authors conclude that a , careful sequential analysis of an ECG of an inferoposterior AMI with ST elevation may lead to the identification of a culprit artery.

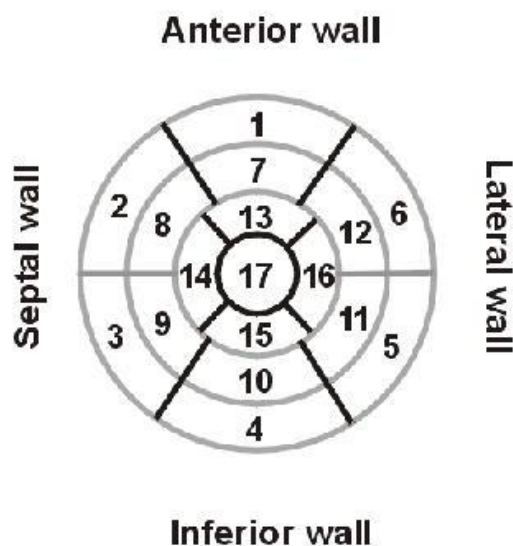
- 1) Fiol M, Cygankiewicz I, Carrillo A, Bayés-Genis A, Santoyo O, Gómez A, Bethencourt A, Bayés de Luna A. Value of electrocardiographic algorithm based on "ups and downs" of ST in assessment of a culprit artery in evolving inferior wall acute myocardial infarction *Am J Cardiol.* 2004 Sep 15;94(6):709-714.
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Creo que el trazado sugiere la existencia de un infarto agudo de miocardio posteroinferior con afectación del VD y el trastorno del ritmo ya mencionado. Dos consideraciones: una ¿hasta qué punto el BRD distorsiona la repolarización en las precordiales? y otra que los clínicos recordamos siempre que vemos un infarto inferior y aún cuando no se hayan hecho las precordiales derechas, algo muy frecuente, hemos de pensar en el VD y la tríada de hipotensión, ingurgitación yugular y campos pulmonares limpios, amén de la auscultación cardíaca, pueden estar presente y nos guiarán para el tratamiento.

Cordialmente Feliciano Pérez C.

Our diagnosis:

- 1) Acute posterolateral and inferior myocardial infarction associated to right ventricle compromising: in classical denomination In posterobasal MI the posterobasal wall of left ventricle is affected. When associated to inferior MI inferior wall is compromised.
- 2) In the new ECG terminology for Q-waves infarctions based on the correlation with CE-CMR this MI is located in inferolateral zone of polar map short axis in “bull’s-eye”, named B3: **4, 10, 15, 5, 6, 11, 12, and 16** ^{1; 2; 3; 4; 5,6;7; 8}.



INFEROLATERAL ZONE

- Inferolateral
- Type: B-3
- Most likely site of occlusion: RCA or dominant LCx
- ECG pattern: signs of inferior (Q in II, II, VF: B2) and/or lateral infarction (RS in V1). When preexistent RBBB prominent anterior forces are observed.
- Segments compromised by infarction in CE-CMR: **4, 10, 15, 5, 6, 11, 12, and 16**
- Sensibility: 73%.
- Specificity: 98%.

.We agree with Prof Frank Yanovitz that the artery obstruction suggests a high-grade RCA occlusion or proximal right coronary artery (RCA). I think that in this case the Prominent Anterior Forces (PAF) are not secondary to left septal fascicular block (LSFB)

because these fibers are irrigated exclusively by anterior descending artery^{9; 10; 11}

So, how we can explain the PAF in ECG-3 of Raimundo's case?

R wave amplitude increases significantly in precordial leads (V(2)-V(6)) and limb leads (I, II, aVL) of the surface ECG during brief episodes of transmural ischemia. The increase in R wave amplitude might be consistent with the expansion of the left ventricular cavity during ischemia and/or alterations in conduction that are intrinsic to the myocardium¹².

Right bundle branch block has been experimentally observed in dogs in all the cases of isolated right ventricular AMI and this dromotropic disorder has been found clinically, too. As RBBB is rare in inferior infarction, its presence may indicate associated involvement of the right ventricle. Both left bundle branch block and right bundle branch block (RBBB) have been associated with increased inhospital and long-term mortality in patients with acute ST elevation myocardial infarction (STEMI). Unlike RBBB in STEMI, RBBB in NSTEMI is not an independent predictor of inhospital and long-term mortality¹³.

In chronic phase of posterolateral (classical nomenclature) or inferolateral

(new nomenclature) is characterized by tall, wide R wave in lead V1 or V1-V2 and broad deep Q wave in lead V6, V7, V8 and V9(left lateral accessory leads) II, III and VF. Dorsal infarction (actual lateral) may be recognized directly just by the accessory leads located between the left shoulder blade and the spine V7, V8 and V9 leads. Robert Zalenski, professor of emergency medicine of the Wayne State University Of Detroit et al., published that using ECG with 15 leads with V4R, V8 and V9 routinely, significantly increases sensitivity to diagnose coronary syndromes, especially to detect myocardial infarction^{14;15}.

In this case we observe ST segment elevation of ≥ 1 mm (0.1 mV) at least one of the right precordial leads V3R, V4R. The sensitivity of the ST elevation sign in V4R is 100% and specificity is 70%. The right precordial leads should always be mapped in patients with diaphragmatic or inferolateral infarction and suggestive clinical symptoms. ST segment elevation ≥ 1 mm or 0.1 mV in one or more leads from V4 to V6 presents a high sensitivity (90%) and specificity to identify RV acute myocardial infarction¹⁶.

A complete ECG thoracic circle allows exploring some heart structures not explored by the conventional ECG. It provides a direct

indication on the location of the damaged myocardium. Posterolateral infarctions can be limited to the inferior third of the left ventricle or can cover the entire free left ventricular wall from the base up to the heart apex and can be univentricular or biventricular. On the other side, the unipolar thoracic leads and the high abdominal leads MD, ME, MI show the evolution of the signs of injury, characteristic of the acute stage of infarction, toward necrosis.

The moderate elevation of the RS-T segment from V9R to V7R indicates the presence of subepicardial injury in the high posterior regions of the right ventricular wall. These ECGs data were confirmed by the radioactive isotope study and, definitively, by the anatomical findings¹⁷.

Thirty to 50% of patients experiencing an inferior wall infarct may also have involvement of the RV. RVs seldom exist alone they are almost always seen with an inferior infarct. The coronary artery involved is usually an occluded RCA¹⁸.

Early recognition of RV myocardial infarction, namely by means of right electrocardiographic leads in acute myocardial infarction, may have prognostic value. Whether or not this recognition will permit improvement of outcomes through more aggressive percutaneous coronary intervention would need to be tested in future studies¹⁹.

Successful thrombolysis should not be considered completed reperfusion therapy. Thrombolytic therapy in this category of patients is accompanied by high frequency of ischemia recurrence, which is connected with critical lesion RCA and ADA, which participate in RV blood supply with collateral vessels. The success of primary and deferred transcatheter intervention in the infarction-associated, including patients after thrombolytic therapy, shows that invasive interventions in such patients are methods of choice; they provide the completeness of reperfusion therapy and improve the course and prognosis of the disease significantly²⁰.

Andrés R. Pérez Riera

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