Case report challenge

English

50-year-old Caucasian man in the emergency room with typical constrictive precordial chest.

Patient with typical constrictive retrosternal precordial pain that began three hours before in the early morning hours. He referred symptoms of fatigue, chest discomfort, and malaise in the days preceding this episode.

Personal antecedents: sedentary, centripetal obesity (grade II: BMI = 37 kg/m^2; waist circumference = 110 cm), smoker (20 cigarettes/day),

Physical

Cold sweats, pallor, low blood pressure = 80 mmHg, jugular vein and liver congestion with clean lungs, fourth noise with ventricular gallop that increases with inspiration, jugular distension at deep inspiration, and paradoxical pulse.

ECG at admission performed on April 23, 2015 – 7:17A.M.

Questions:

1. Which is the diagnosis? And why?
2. Which is the appropriate approach?

Português:

Homem caucasiano 50 anos de idade se apresentou na sala de emergência com dor precordial constritiva típica retrosternal que começou três horas antes nas primeiras horas da madrugada. Ele referiu fadiga, desconforto no peito, mal-estar nos dias que precederam este evento.

Antecedentes pessoais: sedentário, obeso centrípeto grau II (IMC = 37 kg/m^2; circunferência da cintura = 110 cm), tabagista (20 cigarros / dia).

Exame físico

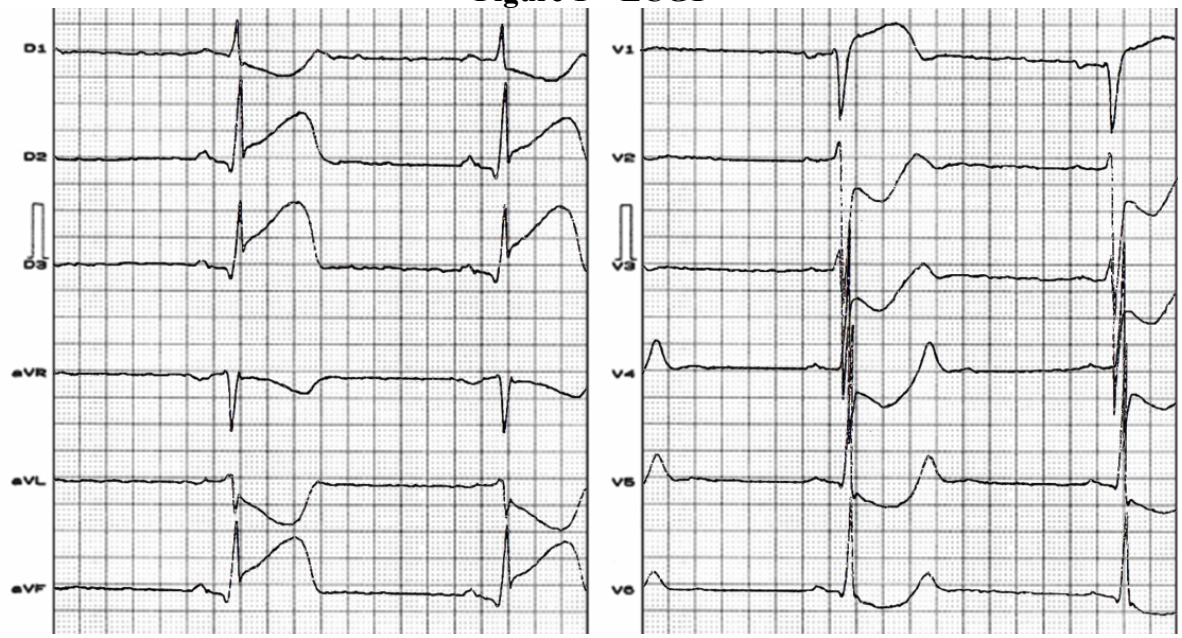
Suor frio, palidez, pressão arterial baixa = 80 mmHg, jugulares e fígado congestos com pulmões limpos. Na ausculta quarto ruído com cadência de galope ventricular que aumenta com a inspiração, distensão jugular a inspiração profunda e pulso paradoxal.

ECG na admissão realizada em 23 de abril de 2015 - 07:17 A.M.

Perguntas:

1. Qual é o diagnóstico? E por quê?
2. Qual é a abordagem adequada?

Figure 1 – ECG1



Colleague's opinions

Hi
This is not my area of expertise, but here I go:
1. Sinus bradycardia
2. Infero-posterior STEMI: ST elevation in inferior leads with "mirror image" + ST depression on the anterior chest leads.
3. RV Infarct" ST elevation in lead III> II, ST depression in lead V2 > than in lead V1

Management
1. Urgent PCI +/-stent
2. Fluid resuscitation as per RV infarct. If possible, avoid chronotropic drugs. See response to fluids first.

Thanks for sharing the case,

Adrian Baranchuk, M.D. FACC FRCPC
Associate Professor of Medicine and Physiology - Cardiac Electrophysiology and Pacing - Director, EP Training Program - Kingston General Hospital - FAPC 3, 76 Stuart Street K7L 2V7, Kingston ON Queen's University - Canada

Looks like acute inferior posterior MI. The ST segment in lead 3 looks a bit higher than in 1 compatible with a right coronary occlusion. The marked ST depression in leads 1 and aVl may be reciprocal or indicate obstruction of the left Circumflex. He needs right sided and posterior leads as well as urgent angioplasty.

Professor Mevin M. Sheinmann
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Portuguese

Andrés

O evento coronariano existe. Ele é indiscutível. Infarto agudo inferior comprometendo a coronária direita com bradicardia (FC média de 30 bpm).
Existe colapso cardiovascular com pulso paradoxal sugerindo tamponamento cardíaco e talvez corroborando com as alterações difusas da repolarização.
Penso em delaminação de Ao com dissecção retrograda obliterando ostio da artéria coronária direita, ocasionado infarto de miocárdio agudo inferior e hemopericardio com tamponamento. Se houver possibilidade de ecocardiograma de urgência na sala de emergência e se conformação, cirurgia de emergência (se der tempo)!

Claudio Pinho

English

The coronary event is a reality. It is undisputed. Acute inferior myocardial infarction (AIMI) compromising the right coronary artery (RCA) with bradycardia (HR average = 30 bpm).
There is cardiovascular collapse with cardiac tamponade paradoxical pulse and perhaps corroborating the diffuse changes in repolarization.
I think of delamination with retrograde dissection obliterating ostium of the RCA, caused inferior AMI and haemopericardium with cardiac tamponade. If there is a possibility of emergency echocardiography in the emergency room in addition, conformation, emergency surgery (if you give time)!

Claudio Pinho M.D. Campinas São Paulo Brazil

Dear Andrés**,** this looks like a right coronary occlusion, where the occlusion is proximal to the right ventricular branches (ST elevation also in V1). The ECG indicates right ventricular transmural ischemia with impending right ventricular infarction. The right coronary artery is probably dominant. Lead V2-V3 ST depressions indicate impending "mirror-image" STEMI (previously named posterior, now lateral MI). The widespread ST depressions in the precordial leads could be caused by concomitant 2- or 3-vessel disease.

Immediate reperfusion therapy is indicated, preferably with PCI. If angiography cannot be performed within 2 hours, thrombolysis is a good alternative. The occlusion is recent (according to Samuel Sclarovsky the "preinfarction syndrome": only ST elevation, no Q waves or inverted T waves) and thrombolysis could be effective. Intravenous fluid should be administered aggressively, maybe many liters to compensate for the decreased contractions of the right ventricle and, thereby, decreased filling of the left ventricle. Nitrates may worsen the hemodynamic state.

Best regards

Kjell Nikus M.D. Tampere University Hospital (TAUH). Finland
http://www.researchgate.net/profile/Kjell_Nikus2/publications

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This is a patient with inferior wall MI and RV infarct. Right-sided precordial leads would add more information. ST depression in precordial leads V2-6 are mirror images of ST elevation in right sided leads. Hypotension, liver enlargement, distended neck veins, increased S4 during inspiration and paradoxical pulse confirm the diagnosis. Patient needs fluid administration and avoidance of nitrates.

Thank you for this interesting case,

Mario D. Gonzalez M.D.

Conclusions by Andrés Ricardo **Pérez-Riera &** Raimundo **Barbosa-Barros**

Answer to the questions

1. Which is the diagnosis? In addition, why?
2. Which is the appropriate approach?

Which is the diagnosis? In addition, why? The diagnosis is hiperacute phase of myocardial infarction on inferobasal wall and right ventricle myocardial infarction. The ECG shows sinus bradycardia at 47bpm with findings compatible with **acute Q wave inferobasal myocardial infarction with right ventricular acute MI extension** because the ST segment elevation in V1 is likely due to right ventricular infarction. In addition, because the clinic is always sovereign in the interrogatory we wrote:" low blood pressure = 80 mmHg, jugular vein and liver congestion with clean lungs, fourth noise with ventricular gallop that increases with inspiration, jugular distension at deep inspiration, and paradoxical pulse".

The classic diagnosis of acute right ventricle MI is characterized by the triad:

1) *Increased jugular venous pressure and passive liver congestion*
2) *Clean lungs*
3) *Hypotension:* blood pressure below 90 mmHg or clinical shock.

Present in this case.

Other valuable physical examination findings are:

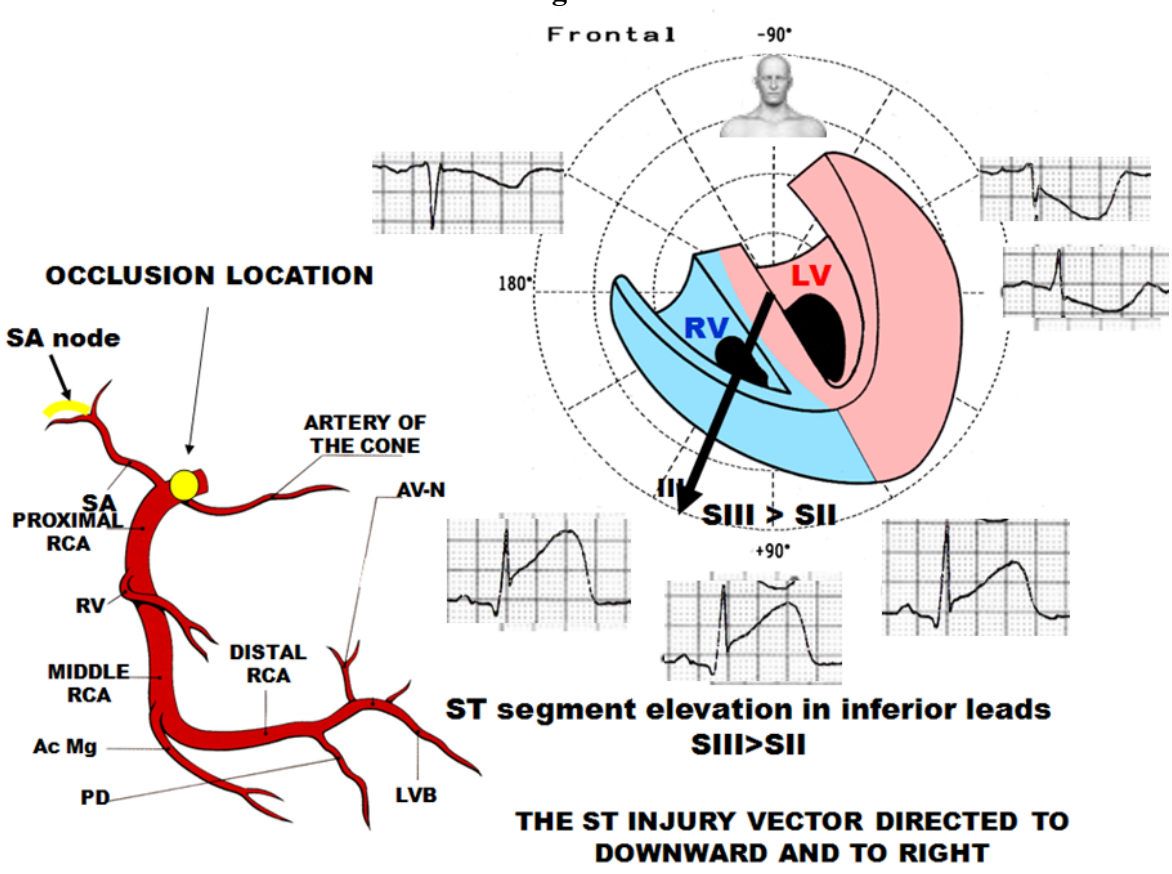
- S4 heart sound with right ventricular gallop increasing with inspiration;
- Possible Kussmaul sign: jugular venous distension with deep inspiration;

- Paradoxical pulse.

Present in this case.

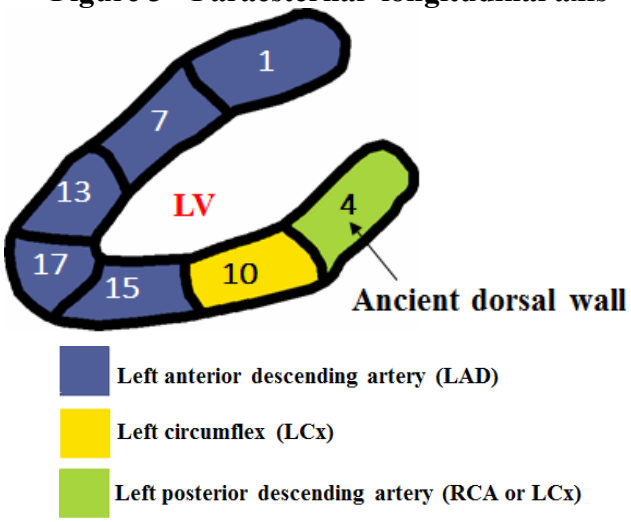
The culprit artery is the right coronary artery (RCA) in proximal location because the ST injury vector is pointed to downward and right near +120º. See figure 2.

Figure 2



We observe the earliest finding of this phase: peaking of the T wave or "hyperacute T waves" (see V4-V5). These are often missed since they occur very early in the course of the event and they are transient. ST and T wave abnormalities suggesting myocardial injury.
The Q waves in III and II and the R wave in V1 do not yet meet criteria for inferobasal MI although they probably would within an hour or two after this tracing)
Summary: Inferobasal and right ventricle acute myocardial infarction during hyperacute phase.
Why not hyperarcute inferior-dorsal infarction? Answer: because for more than 50 years the terms true or strictly dorsal or posterior infarction, injury or ischemia have been applied when the basal portion of the inferior wall was affected (segment 4).

Figure 3 - Paraesternal longitudinal axis



However, for consistency Bayes de Luna in successive manuscripts have to suppress the word "posterior" and just call "inferior" the wall that lies on the diaphragm and "inferobasal segment" the classically named "true posterior wall. **The dorsal wall does not exist!!**

Figure 4 - New terminology for the wall of the heart (4 walls) polar map short axis in "Bull's-eye"

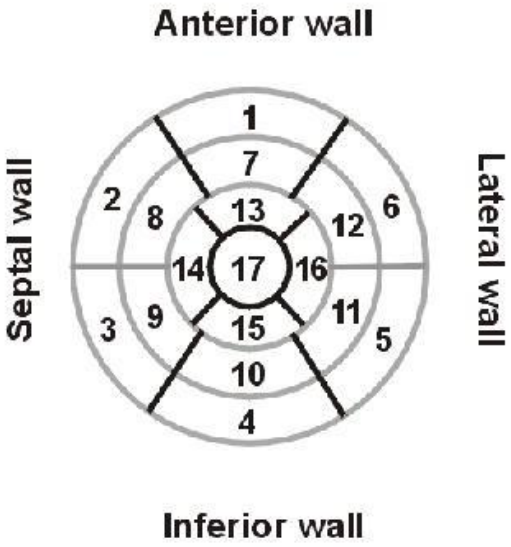
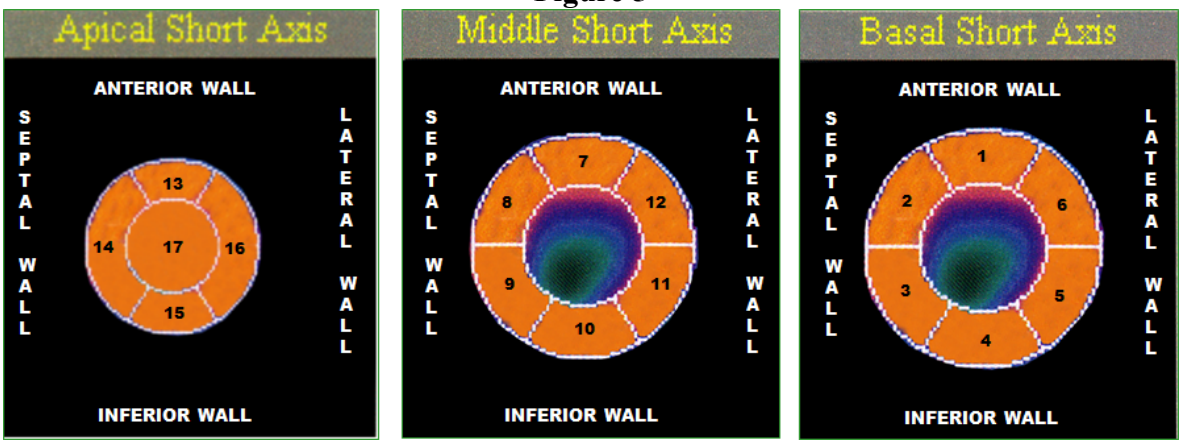


Figure 5



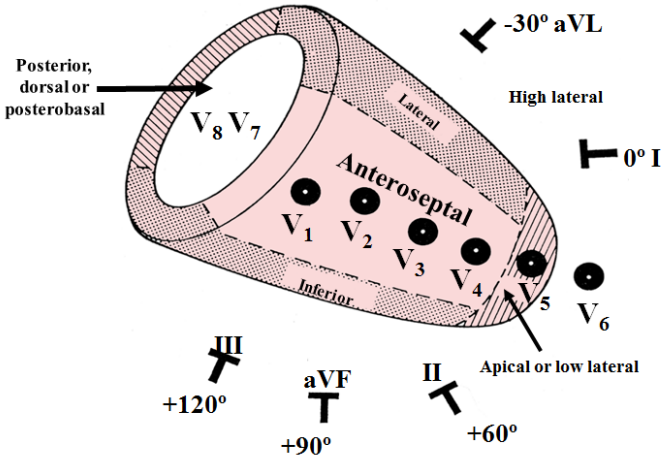
Observation: Number 4 in the basal short axis corresponds to ancient dorsal or posterior wall.

Table 1

Walls	Segments

Anterior	1,7,13
Septal	2,8,3,9,14
Inferior	4,10, 15
Lateral	5, 6, 11, 12, 16
Apex	17

Figure 6 - Topographic classification of heart attacks in cardiac cone (old classic unmodified concept)



Leads and the wall representation

I Lateral	aVR	V1 Septal	V4 Anterior
II Inferior	aVL Lateral	V2 Septal	V5 Lateral
III Inferior	aVF Inferior	V3 Anterior	V6 Lateral

(Burch 1958)

The contiguous leads shown in the same color:

- Pink — aVR (this lead does not have contiguous lead. It is an isolated lead)
- Orange — septal leads (V1, V2)
- Yellow — inferior leads (II, III, aVF)
- Green — lateral leads (I, aVL, V5, V6)
- Blue — anterior leads (V3, V4)

Limitations of the classical classification

a) Name given to different walls.
b) Technical problems; specially related with placement of V3-V6.
c) Different body built
d) Anatomical variants of coronary arteries
e) Structure of left ventricle.
f) Coexisting heart diseases
g) Cancellation of vectorial forces
h) Specific characteristics of anteroseptal and inferolateral zone.
I) Lack of value given to aVR and other additional leads

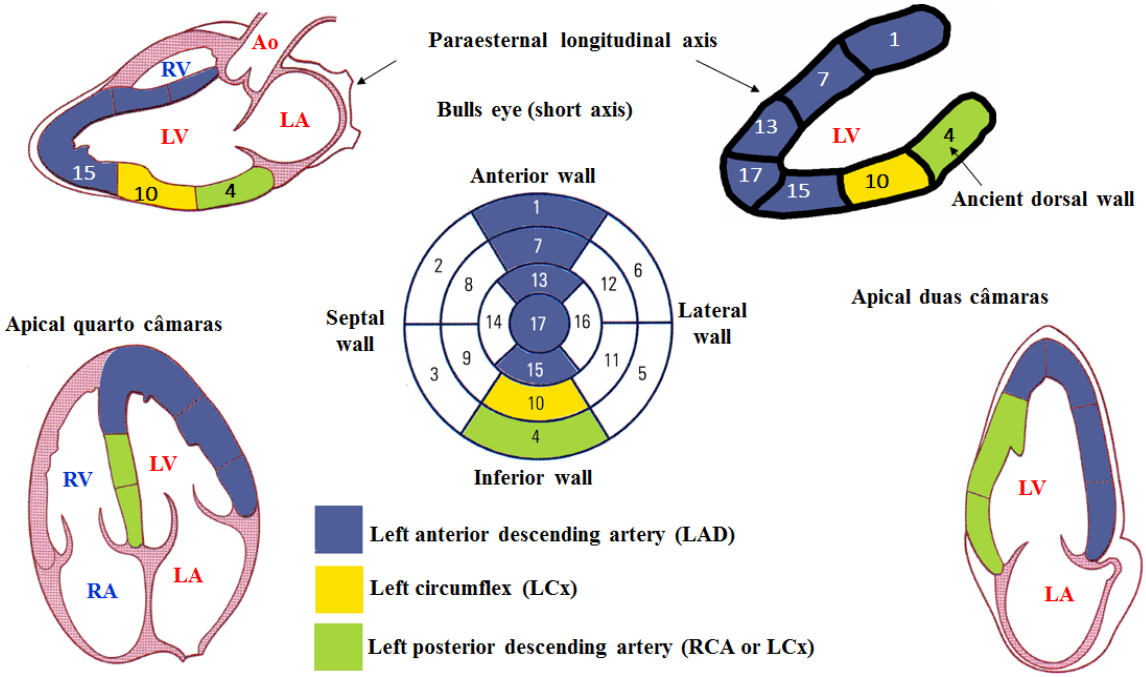
Actually, the CE-CMR is the Gold-Standard technique not only for:

- Infarct identification
- Transmurality characterisation

But also for:

- Infarct location
- Ideal to correlate with ECG patterns

Figure 7 - Current model of ventricular segmentation and artery responsible for irrigation. The color marks the culprit artery by irrigation

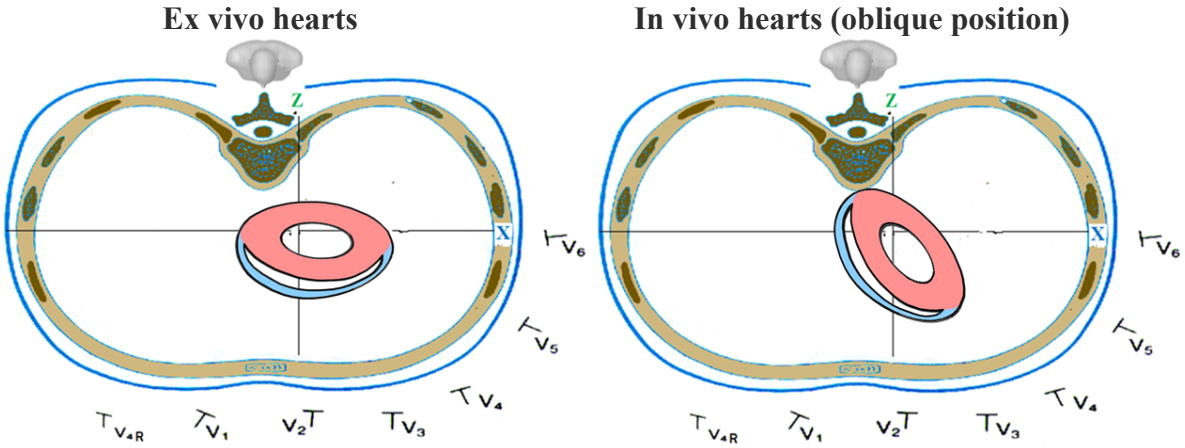


In chronic phase, the involvement of the erroneously and classically named dorsal wall or posterior wall (actually inferobasal segment of inferior wall) produces RS morphology in V3-V4 instead of V1-V2, because the necrosis vector pointed to the intermediates precordial leads V3-V4. On the other hand, lateral MI may produce RS morphology in V1 since the necrosis vector point ≈ to +120º (V1 lead). Lateral MI explains the presence of prominent R wave (R ≥ S) in V1.(**Goldwasser 2015**). With this new concept, we reach not only an agreement between cardiologists devoted to electrocardiography, but also a consensus with the terminology used by experts in magnetic cardiac imaging for both the Q-wave and non-Q-wave myocardial infarctions that may have great advantages for clinical practice (**Arai 2004**)

. This is a new heart wall terminology and new electrocardiographic classification of Q-wave myocardial infarction based on correlations with magnetic resonance imaging**. (Bayés de Luna 2007; 2007).** This concept has been endorsed by a statement for healthcare professionals from a committee appointed by the International Society for Holter and Noninvasive Electrocardiography**.(Bayés de Luna 2006)**

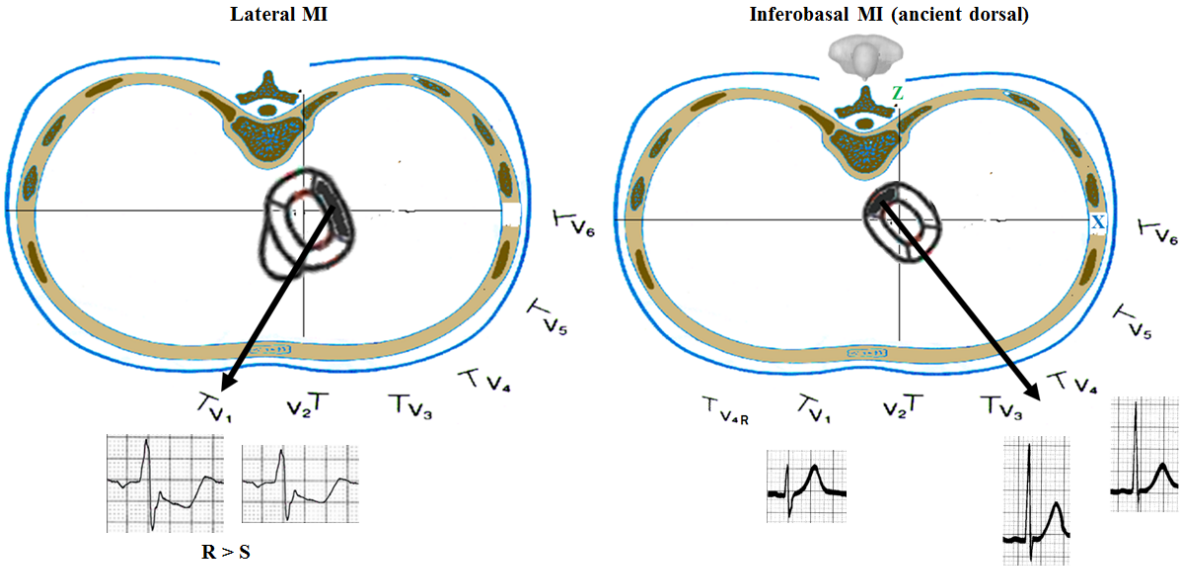
ST-segment depression in leads V_2, and V_3 that occurs in association with an inferior wall MI may be caused by occlusion of either the RCA or the LCx. This ECG pattern has been termed *posterior* or *posterolateral* ischemia since the recommendations of Perloff **(Perloff 1964**) and Horan et al (**Horan 1971**) and is based on anatomic and pathological studies of ex vivo hearts. However, recent **in vivo** imaging techniques, including echocardiography and magnetic resonance imaging, have demonstrated the **oblique position** of the heart within the thorax. These studies demonstrated that the region referred to as the posterior wall was lateral rather than posterior and led to the suggestion that the designation lateral (**Cerqueira 2002**) replace the term posterior.

Figure 8



Bayés de Luna et al correlated the ECG patterns of healed myocardial infarctions to their anatomic location as determined by magnetic resonance imaging. They reported that the most frequent cause of abnormally tall and broad R waves in leads V_1 and V_2 in patients known to have experienced a recent acute infarction was involvement of the lateral and not the posterior wall of the left ventricle.

Figure 9

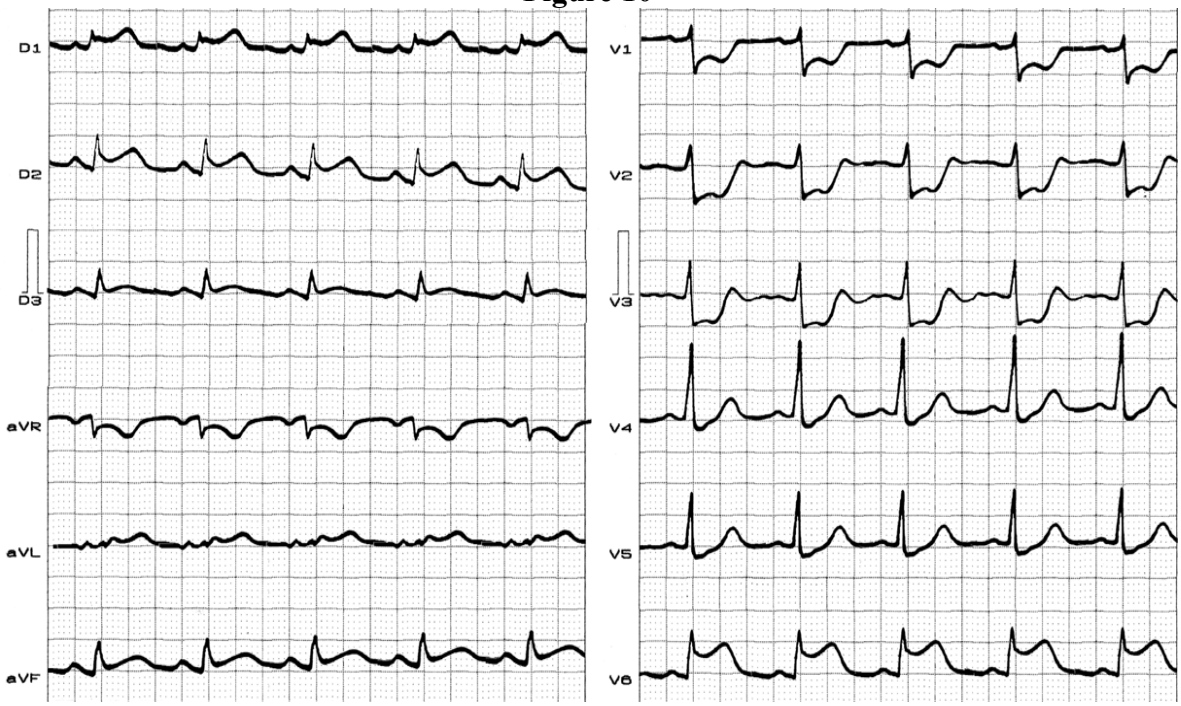


Abnormal tall and broad R waves in leads V_1 and V_2

They suggested that the terms *posterior ischemia* and *posterior infarction* be replaced by the terms *lateral*, *inferolateral*, or *basal-lateral* depending on the associated changes in II, III, aVF, V_1, V_5, and V_6. The International Society has endorsed such terminology for Holter and Noninvasive Electrocardiography **(Bayés de Luna 2006)** and by the last consensus of American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; American College of Cardiology Foundation; Heart Rhythm Society. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram about acute ischemia/infarction:

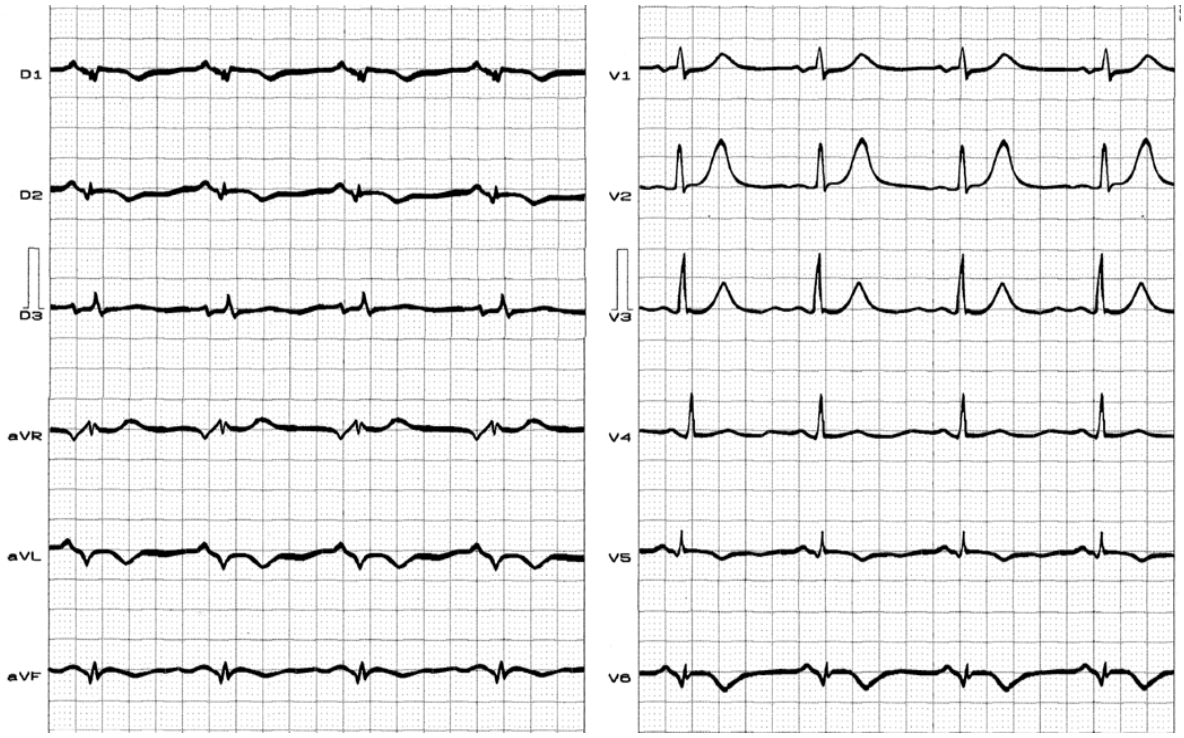
a scientific statement Endorsed by the International Society for Computerized Electrocardiology.(ISCE) **(Wagner 2009)**

Figure 10



ECG diagnosis: Significant ST segment elevation is observed in the infero-lateral wall leads and significant ST segment depression from V1 to V3. Infarction in the hyperacute phase with ST segment elevation in the infero-lateral region (STEMI-ACS) requiring an immediate intervention. When this was not performed the pattern in the figure 11 appeared in the late phase.

Figure 11



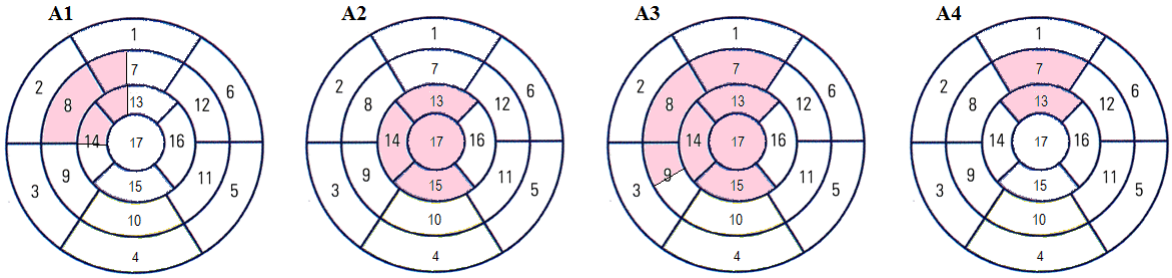
This ECG is from the same patient, made 20 days after the event. Q waves appeared in the inferior wall, prominent R waves from V1 to V3 with positive and symmetrical T waves, low voltage of R in V5-V6 and QS in I and aVL. These changes represent inferolateral myocardial infarction in the late phase.

ECG criteria for MI

1) Any Q ≥30 ms in inferior leads.
2) Q ≥ 40 ms in I /VL.
3) Q wave in ≥ 2 contiguous precordial leads.
4) Any Q-wave in V1-V2 or R ≤ 0.1 mV in V2 .
5) Q-wave equivalents: RS / R in V1 ≥40 ms, and "qr" or "r" <5 mm in V6.

Figure 12 - Current classification of heart walls

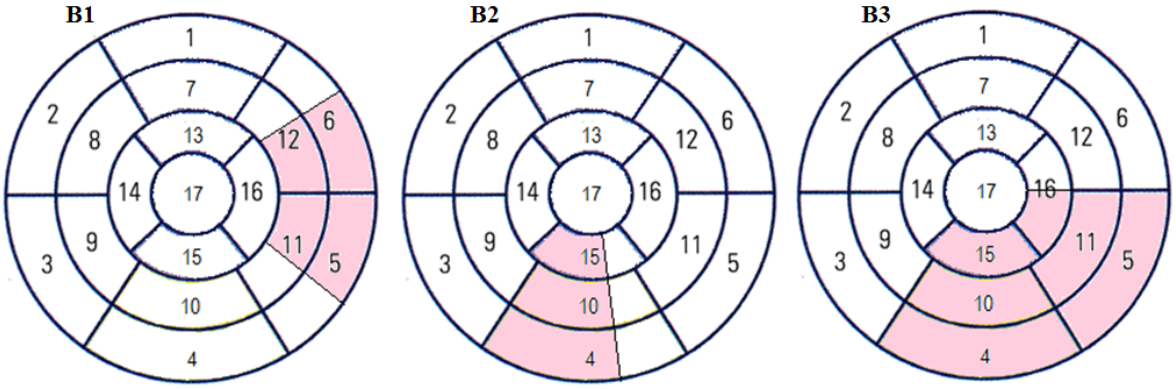
I – Anteroseptal zone



A1 – QS in V1 and V2. Septal. SE: 86%. SP: 98%

A2 – QS in V1 and V2 to V4-V6. Anteroseptal and anteroapical. SE: 86%. SP: 98%
A3 – QS in V1-V2 to V4-V6, I and aVL. Extensive anterior. SE: 83%. SP: 98%
A4 – Q, qs or qr in aVL and eventually in V2-V3. Limited anterior. SE: 75%. SP: 100%

II – Inferolateral zone



B1 – Q, qr or r in I, aVL, V5-V6 and/or RS in V1. Lateral. SE: 50%. SP:98%
B2 – Q in II, III and aVF. Inferior. SE: 87,5%. SP: 98%
B3 – Q in II, III, aVF + Q in I, aVL, V5-V6 and/or RS in V1. Inferolateral. SE: 70%. SP: 100%. This is the present case.

Theoretical consideration about right ventricular MI

Identification of RV myocardial infarction is important because it is associated with greater short term morbidity and mortality. Acute total occlusion of the proximal RCA may result in necrosis in a large area of the RV in addition to the damage to the inferior wall of the left ventricle. The different segments of the right ventricle are perfused as followed:

- **RV free wall:** RCA trunk, except for the anterior border.
- **RV lateral wall:** acute marginal branch (Ac Mg) or ramus marginalis dexter.
- **RV anterior wall:** right ventricular branch of the RCA.
- **RCA conal branch:** part of the septum.
- **Posterior descending artery:** (RCA branch in 86% of the cases; LCx branch in 14%); RV posterior wall.

Note: On rare occasions, the branches from a long left anterior descending artery (LAD) or type IV anatomy (those that wrap around the apex of the heart) perfuse part of the RV.

Clinical identification of Acute Right Ventricular Myocardial Infarction

The classic diagnostic clinical triad is characterized by:

4) *Increased jugular venous pressure and passive liver congestion*
5) *Clean lungs*
6) *Hypotension:* blood pressure below 90 mmHg or clinical shock.

Other valuable physical examination findings are:

- S4 heart sound with right ventricular gallop increasing with inspiration;
- Possible Kussmaul sign: jugular venous distension with deep inspiration;
- Paradoxical pulse.

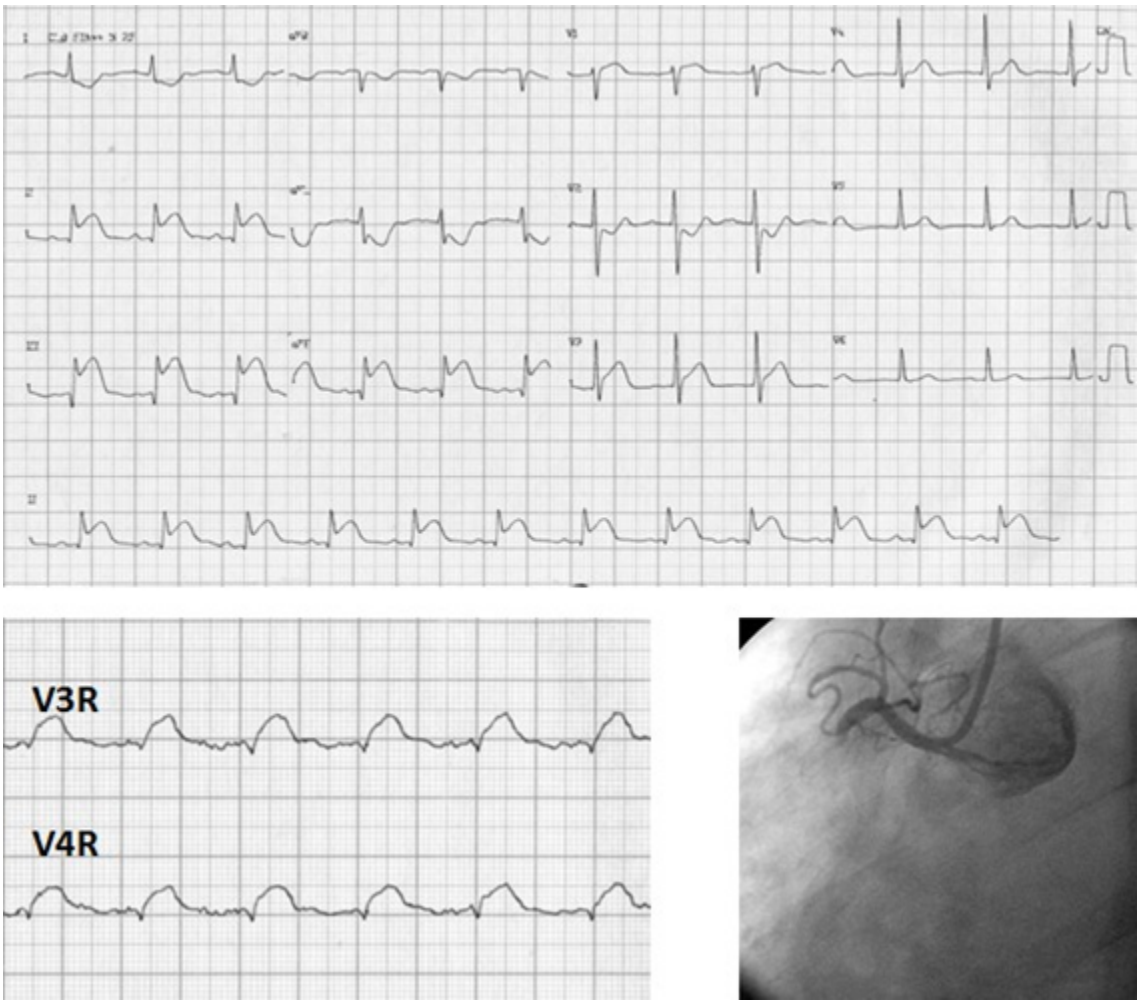
Table 2 shows the main clinical and ECG differences between acute inferior wall myocardial infarction with or without acute right ventricular myocardial infarction.

Table 2 (Braat 1984)

	Inferior MI without RV infarction	Inferior MI with RV infarction
RV heart failure signs	No	Frequent
Kussmaul's sign	Negative	Positive
High grade AV block	48%	13%
ST segment elevation from V4R to V6R	Absent	Present

ST segment elevation in V4R is the strongest predictor of RV involvement and is always associated with acute total proximal RCA occlusion as shown in the case illustrated in **Figure 13**. These ECG findings are transient and disappear, on average, within 10 hours after the event in 50% of the patients.

Figure 13



Electrocardiographic diagnosis: another typical case of acute inferior infarction associated with acute RV MI. In the frontal plane the injury vector is pointing towards +120° resulting in greater ST elevation in lead III than in lead II with reciprocal depression in leads I and aVL. In the right precordial leads V3R and V4R ST segment elevation with positive T waves are seen indicating RV involvement.

Table 3 below shows the sensitivity and specificity of the ST segment elevation >1 mm in V1, V3R and V4R (Klein 1983).

Table 3

Lead	Sensitivity (%)	Specificity (%)
V1	28	92
V3R	69	97
V4R	93	95

Main electrocardiographic criteria for Acute Right Ventricular Myocardial Infarction

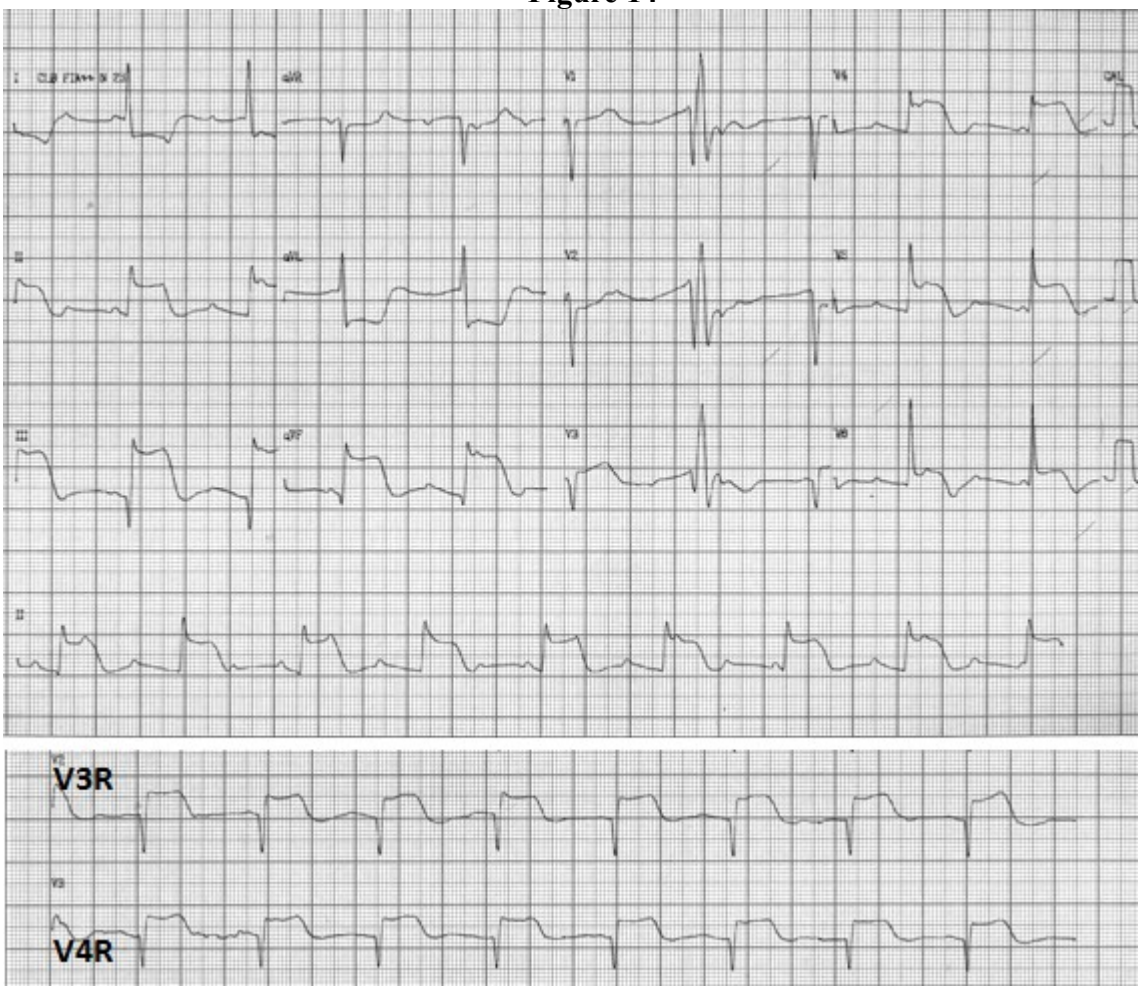
- **Rhythm:** frequent atrial fibrillation, atrial flutter, multifocal atrial pacemakers, and junctional rhythms due to atrial infarction ($^1/_3$ of the patients present concomitant atrial infarction).
- **P wave:** right atrial enlargement (RAE) pattern may be seen as a consequence of increased pressure in the right atrium due to increased RV end-diastolic pressure.
- **PR interval:** depression or elevation resulting from acute atrial infarction.
- **ST segment:** transient ST segment elevation of ≥1 mm (0.1 mV) in at least one of the right precordial leads V_3R, V_4R, V_5R, V_6R. The sensitivity of ST elevation in V_4R is 100% with specificity of 70%. The right precordial leads should always be recorded patients with inferior wall myocardial infarction and clinical findings of RV-MI. Acute RV myocardial infarction is generally associated with left ventricular inferior wall infarction. ST segment elevation ≥1 mm or 0.1 mV in one or more leads from V4R to V6R has a high sensibility (90%) and a reasonable specificity to identify acute RV myocardial infarction.
- ST segment elevation of ≥1 mm in the CR lead (fifth right intercostal space in the midclavicular line).
- Occasional decreasing ST segment elevation from right to left in V_1 to V_3, mainly when the LV lesion is minimal.
- ST elevation usually disappears in average after 10 hours.
- ST segment depression with negative, symmetrical, deep T waves with a wide base, have been observed from V_1 to V_3.
- QS or QR waves associated with ST segment elevation in the right precordial leads.
- Q wave appears in RV dorsal-lateral necrosis (sensitivity 100% in V_4R-low specificity).
- QS complexes in V1 may be normal, especially in the elderly. In V_3R and V_4R, the occurrence of this type of complex is highly suggestive of RV MI.
- ST segment elevation of 1.5 mV in RV intracavitary unipolar lead with the electrode located in the tip of the RV.
- Right bundle branch block has been observed experimentally in dogs with RV AMI in isolation, and clinically this conduction disorder may also be found. Since RBBB is rare in inferior infarction, its presence may indicate associated RV involvement.
- Total AV block; when present, it is associated with a greater mortality.
- High grade AV block is present in almost half of the cases.
- The chronic phase of RV infarction cannot be diagnosed by ECG.

Isolated RV MI is extremely rare and may be interpreted mistakenly as LV anteroseptal infarction due to ST segment elevation in V1-V4.

Patients with inferior wall infarction with additional RV involvement have a greater incidence of complete AV block which leads to worsening hemodynamics due to atrioventricular dyssynchrony. The ischemic RV with reduced compliance depends to a large extent on efficient atrial contractions to fill adequately. **Figure 4** shows a case of

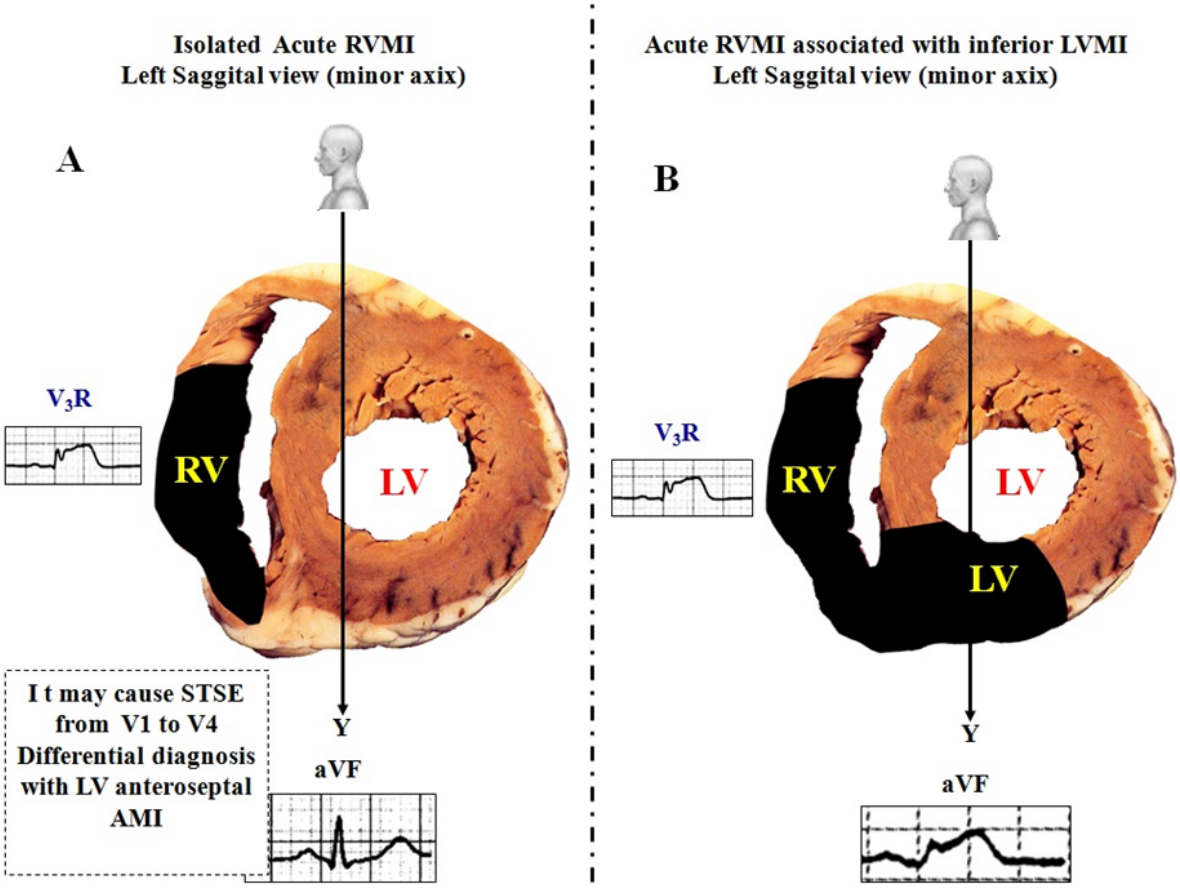
inferior MI with RV involvement complicated by complete AV block that evolved into death from cardiogenic shock.

Figure 14



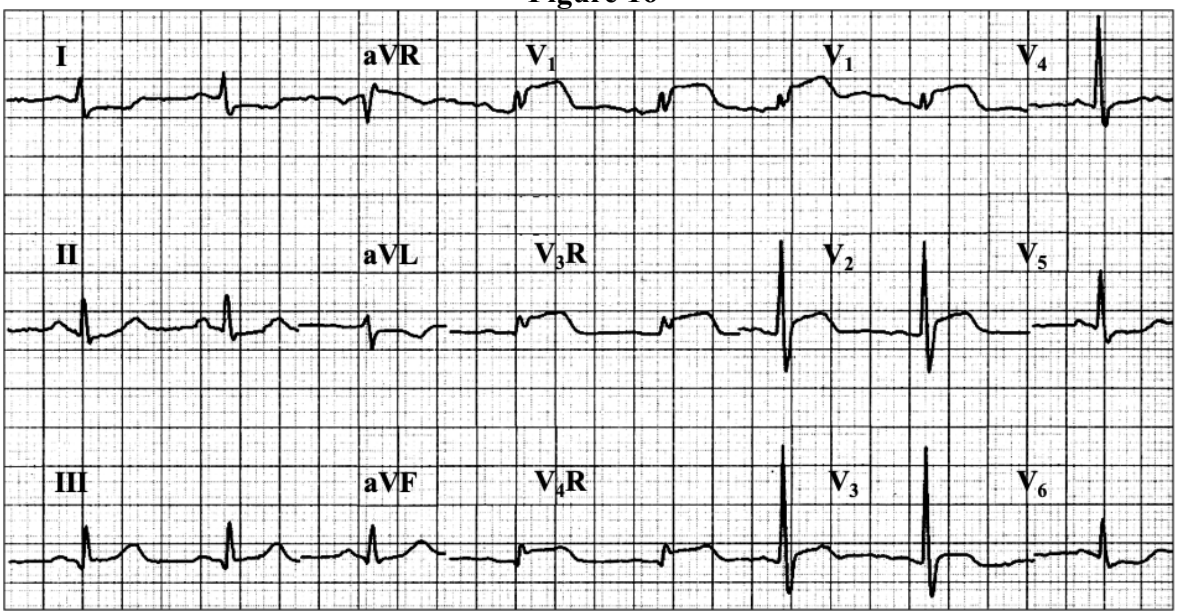
Electrocardiographic diagnosis: The tracing shows acute infero-lateral MI complicated with complete AV block. In the frontal plane STE III is greater than STE II (ST injury vector pointing to III). QS or QR waves associated with ST segment elevation in the right precordial leads V3R and V4R indicate RV involvement.

Figure 15



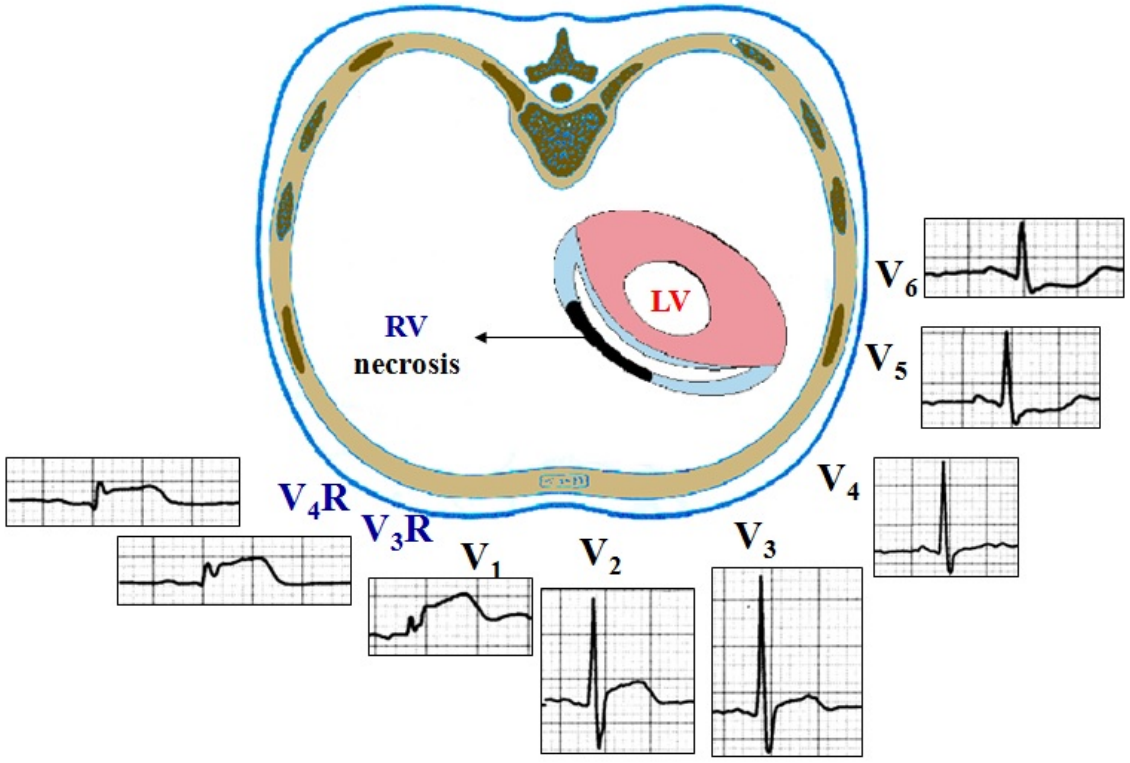
The figure 15 shows electro-anatomical differences between isolated right ventricle acute myocardial infarction **(15A)** and RV MI associated with diaphragmatic or inferior acute myocardial infarction of the left ventricle **(15B)**. In figure **15A** ST segment elevation (transmural injury) is seen only in anteroseptal wall leads (V1 to V3) and right accessory leads (V_3R and V_4R) without changes in the inferior leads.

Figure 16



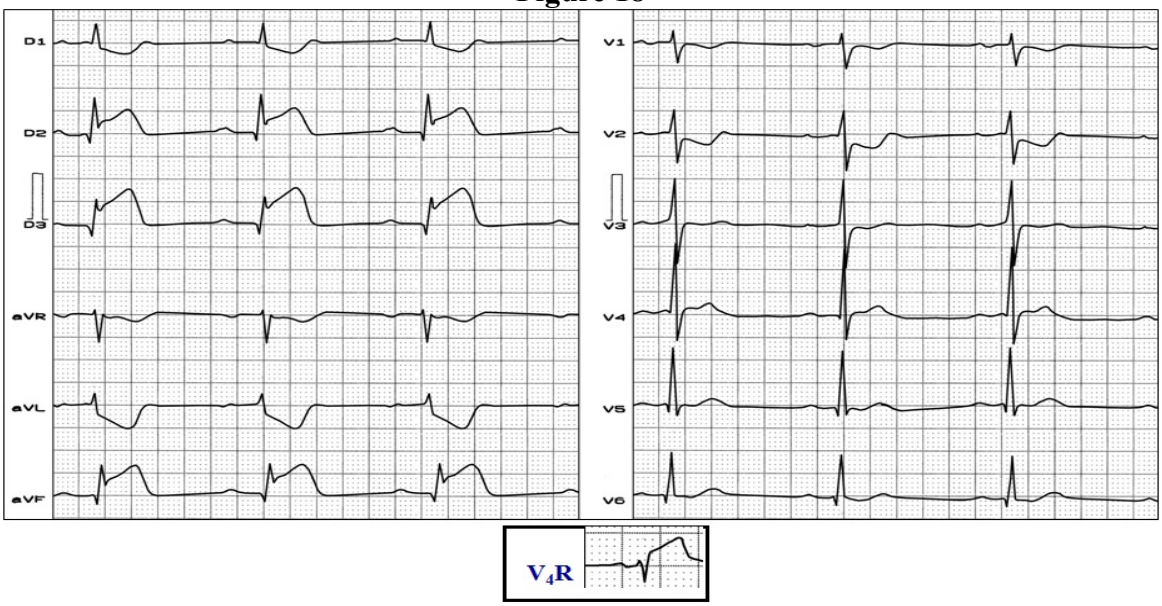
Eletrocardiographic diagnosis: ST segment elevation reflecting transmural injury in leads V_3R, V4R. V_1 and V_2, This ECG is a rare case of isolated acute right ventricular myocardial infarction without inferior wall left ventricle involvement.

Figure 17



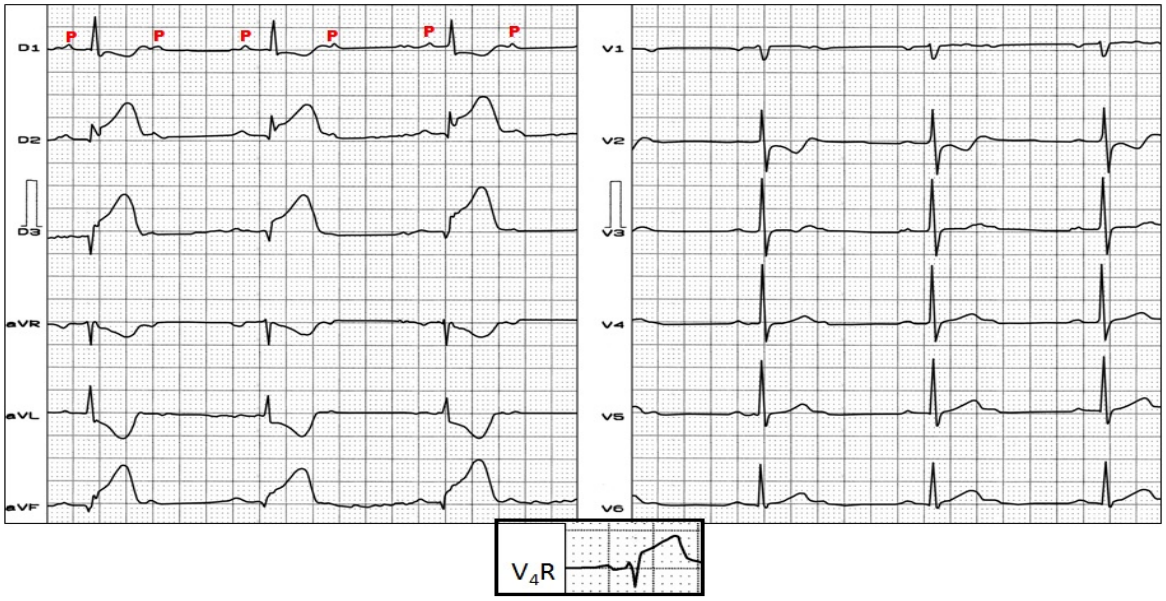
The figure 17 shows the six conventional precordial leads and the accessory right precordial leads V3R, V4R in acute isolated right ventricular myocardial infarction. ST segment elevation is seen in V3R, V4R, and from V1 to V3.

Figure 18



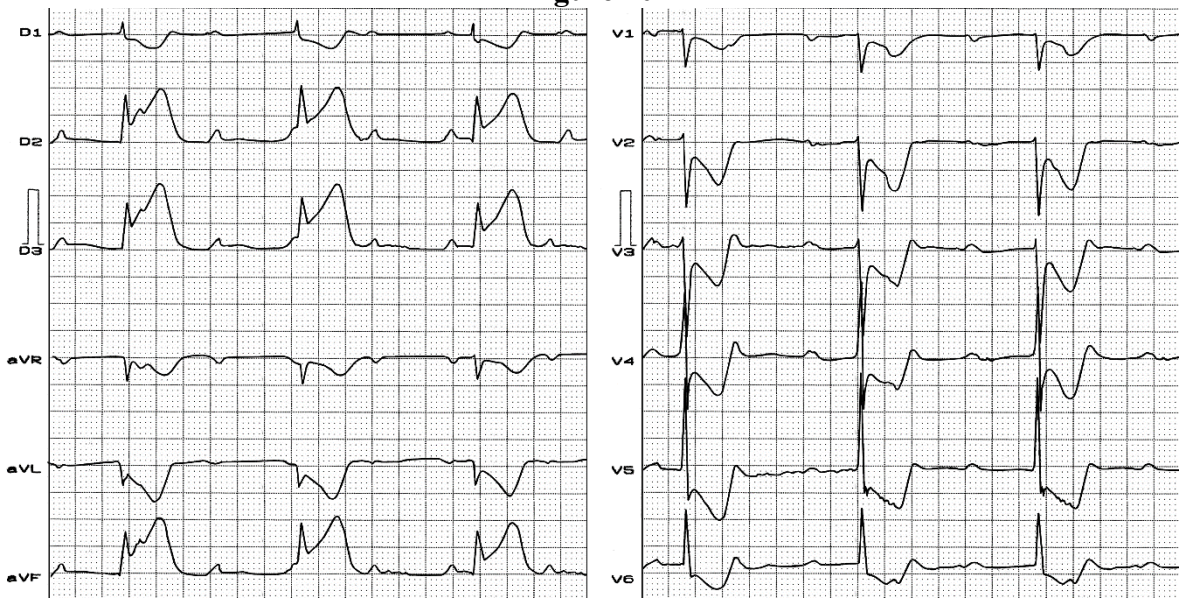
Electrocardiographic diagnosis: sinus bradycardia, first-degree AV block, ST segment elevation in the inferior leads with $ST_{III} > ST_{II}$ (the ST injury vector pointing to III). This pattern is compatible with acute inferior myocardial infarction resulting from proximal RCA occlusion with additional RV involvement: ST segment elevation with positive T wave in V_4R. ST-segment elevation in lead V_4R is the single most powerful predictor of right ventricle acute myocardial infarction. Additionally, depression of ST segment in I, aVL, V1-V2 (are mirror or reciprocal changes on the high lateral and posterior wall).

Figure 19



Electrocardiographic diagnosis: acute inferior myocardial infarction complicated with 2:1 AV block and right ventricular involvement: ST segment elevation in V4R followed by positive T wave. This ECG pattern is a consequence of proximal RCA occlusion.

Figure 20



Electrocardiographic diagnosis: acute inferior myocardial infarction complicated by third degree AV block due to proximal RCA occlusion. QRS complexes are narrow indicating suprahisian escape rhythm. Extensive mirror image or reciprocal changes are registered across precordial leads (V1 to V6), I and aVL.

Therapeutic approach of RV myocardial infarction

Volume resuscitation with saline solution is the therapeutic aim of maintaining pre-load based on prior studies. This has been adopted for several years now as an initial therapeutic

measure (Lopez-Sendon 1981; Baigrie 1983; Goldstein 1983). However, recent studies have shown adverse effects related to excess of volume resuscitation. Several publications, including two prospective studies, have indicated that volume overload increases right sided filling pressure without improving cardiac output (Dell'Italia 1984; Siniorakis 1994; Ferrario 1994). The strategy of early reperfusion preferentially percutaneous coronary intervention or fibrinolytic therapy should be used similar to STEMI of the LV with the aim of reversing the low cardiac output syndrome which is present in 15-20% of cases. Electrical stabilization including proper control of HR and atrioventricular synchrony is another key factor to maintain cardiac output in this subset of patients.

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