Diagnosis of Coronary Artery Disease. Part II - Case reports

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Case report number 1
Acute Myocardial Infarction in a Patient with New or Apparently New Complete Left Bundle Branch Block (CLBBB)

Case presentation

The patient is a 69 year old white male with onset of typical chest pain 7 days ago which worsened on the day of admission.

Risk factors: Advanced age and cigarette smoking. There was no history of hypertension, dyslipidemia, or diabetes.

Physical examination: BP = 130/90 mmHg; dyspneic, regular heart rhythm with a fourth heart sound (S4), split second sound (S2), and warm limbs with symmetrical pulses.

He was in Killip Class II with rales in both lung bases.

The admission ECG is illustrated in **Figure 1**.

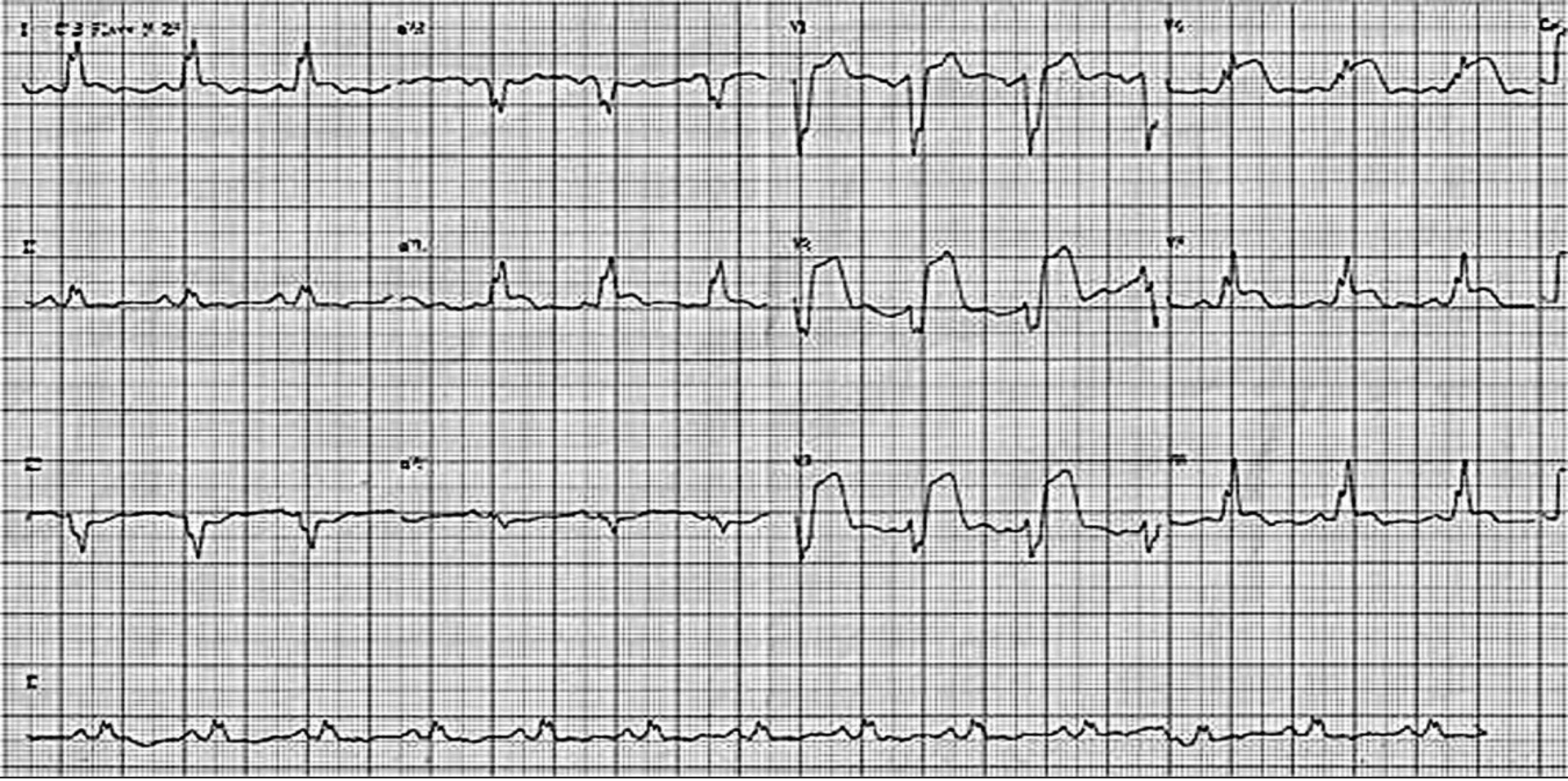
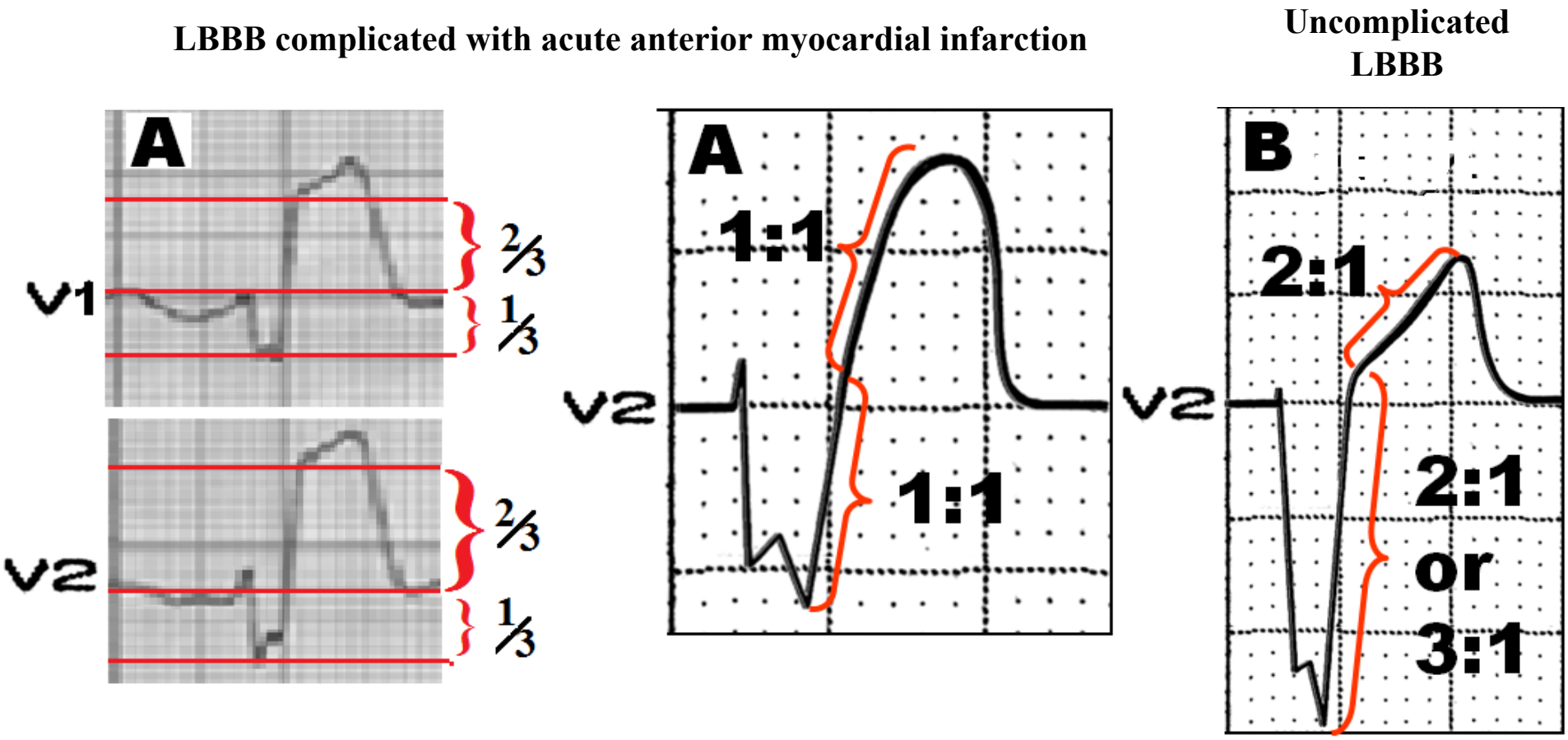


Figure 1 - Electrocardiographic findings: sinus rhythm, HR: 83 bpm, PR interval ≥120 ms, QRS axis 10°, QRS duration >120 ms, predominantly negative QRS complexes (rS) in the right precordial leads (V1, V2 and V3), and monophasic notched R waves in leads I, aVL, V5 and V6. ST segment elevation indicative of anterolateral transmural injury present in leads V1-V6, I and aVL. Marked ST segment elevation (>5 mm) present in V2-V3. There is a decrease in the $^{QRS}/_{ST\text{-}T}$ ratio in V2 and V3. (In the presence of uncomplicated CLBBB, this ratio is usually 2:1 or 3:1 in V2) (**Figure 2**). In CLBBB associated with acute myocardial infarction (AMI), the ratio of $^{QRS}/_{ST\text{-}T}$ voltages is closer to 1:1 as in this case (**Schamroth 1975**). Finally there is concordant ST segment elevation in the same direction as QRS in leads I, aVL, V5 and V6, a finding not seen in uncomplicated CLBBB (**Figure 3**).

Figure 2



In Figure 2A, a decrease in the $^{QRS}/_{ST\text{-}T}$ ratio is observed in V1 and V2; this ratio is <1:1 This is an important electrocardiographic sign of CLBBB complicated with anterior wall infarction (1). A ratio ≤1:1 is due to reduced QRS voltage resulting from the infarcted myocardium.

In Figure 2B, the V2 lead is shown in uncomplicated CLBBB. In this case, the $^{QRS}/_{ST\text{-}T}$ width ratio in V2 is more typically 2:1 to 3:1.

Figure 3

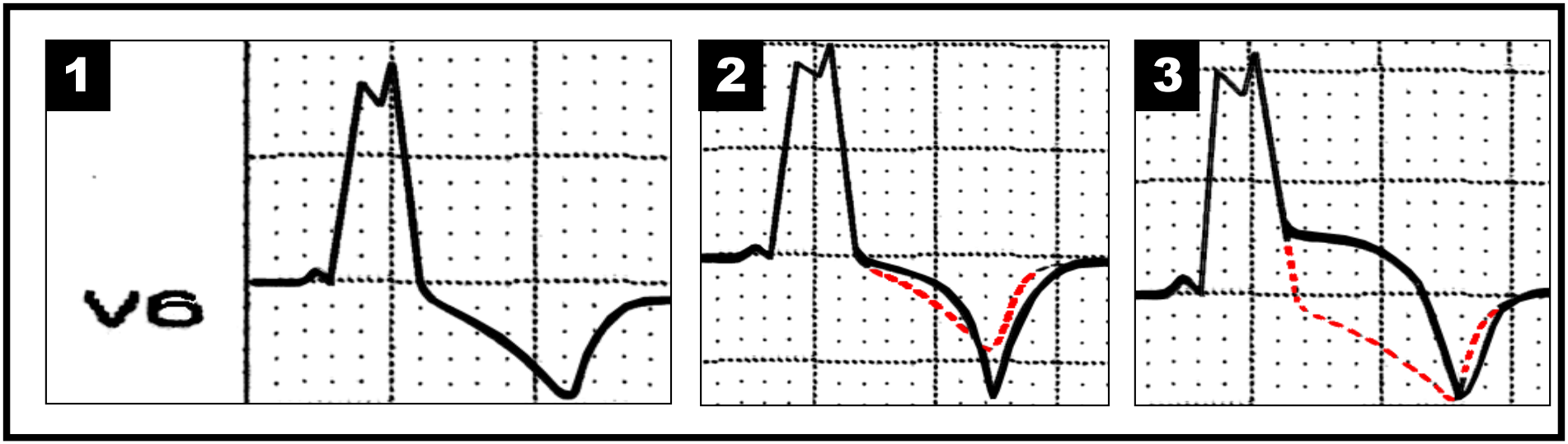


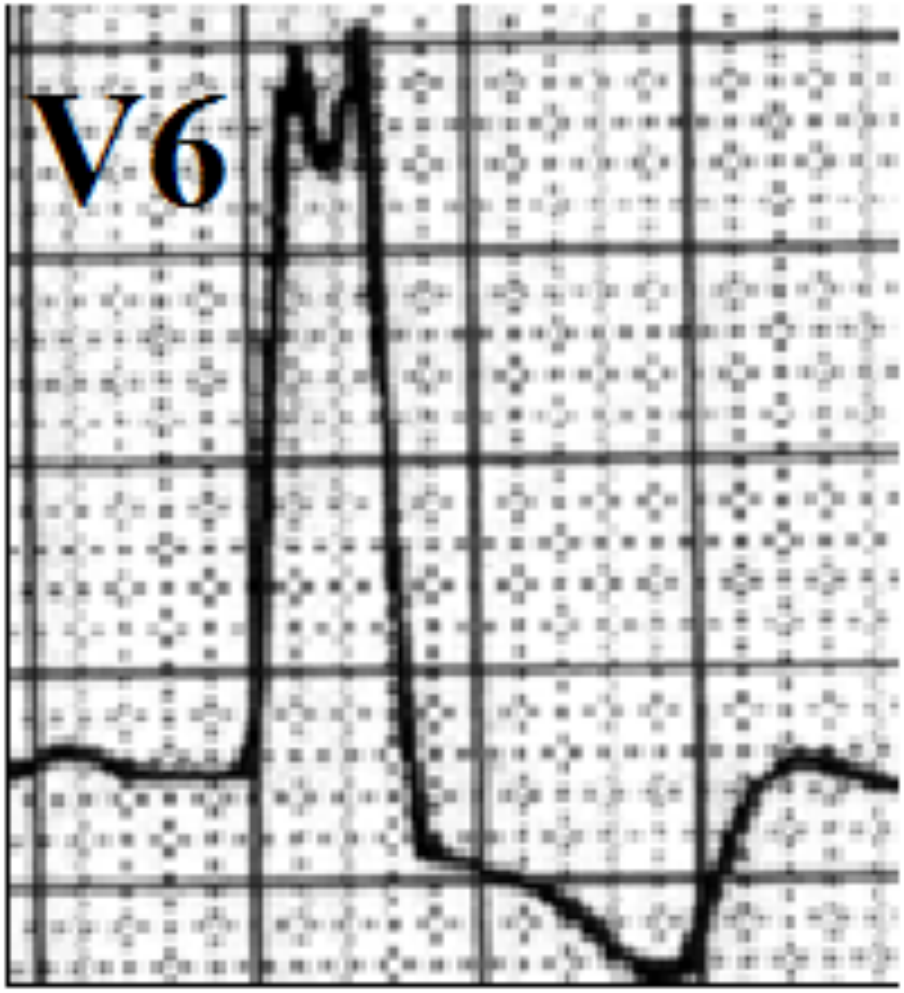
Figure 3 shows the left precordial lead V6 in uncomplicated CLBBB (1) and CLBBB associated with transmural ischemia (**Sgarbossa 1996; Sgarbossa 1996**). Figure 3-3 is similar to leads I, aVL, V5-V6, in the present case illustrated above in Figure 1. The red dotted line in Figure 3B and 3C refers to the normal repolarization in uncomplicated CLBBB noted in Figure 3-1.

ECG features in uncomplicated CLBBB in precordial leads

Ventricular depolarization: In the presence of uncomplicated CLBBB, monophasic and frequently notched R waves are observed in the left leads I, aVL, V5 and V6 with a ventricular activation time (intrinsicoid deflection) in leads I and V6 ≥50 ms. There may be an initial small q wave in I and aVL but never in V5 and V6. Possible Rs or RS patterns are found in V5 and V6 which may indicate late transition association with right ventricular enlargement, left anterior fascicular block, or an electrically inactive area in the LV free wall. In this latter case the final S wave is usually wide (≥4 ms) and notched (so called Q wave equivalent). In the early years of electrocardiography Frank Wilson suggested that the S waves in lead V6 in patients with LV free wall infarction and CLBBB were due to the V6 electrode capturing the intracavitary potential of this ventricle; it was called the "electrical window of Wilson". Later, with the application of vectorcardiography, it was determined that the appearance of S wave in these patients was due to the rightward shift of the afferent limb of the QRS loop in the orthogonal Z plane and not the intracavitary potential. This hypothesis is reinforced by the fact that the S wave appears wider (>40 ms) and notched.

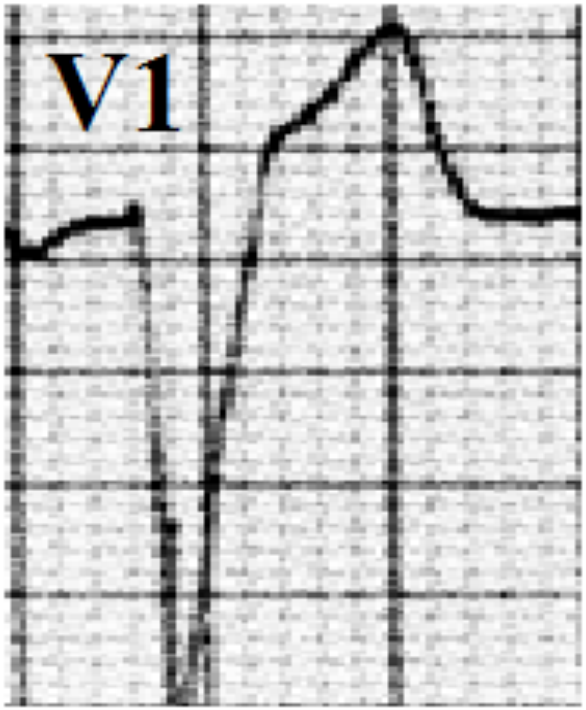
Ventricular repolarization: In uncomplicated CLBBB the J point and ST segments are directed opposite to the deflection of the QRS. In leads with positive QRS complexes (I, aVL, V6) the J point is depressed and the ST segment has upward convexity followed by an asymmetric negative T wave with the initial descending portion being slow and the final ascending part being rapid. This pattern is secondary to the alteration in the sequence of ventricular depolarization caused by the left bundle branch block. The axes of the QRS and ST/T are parallel with opposite directions in a degree close to +180°. In other words, these are alterations secondary to ventricular repolarization with wide QRS-ST-T angle; however the ventricular gradient remains normal. The normal QRS and ST/T pattern in lead V6 in uncomplicated CLBBB is shown in Figure 4.

Figure 4



In the right precordial leads in patients with uncomplicated CLBBB, as well as in patients with right ventricular pacing, the QRS complex is totally (QS) or predominantly negative (rS) and ventricular repolarization is characterized by J point and ST segment elevation of upper concavity followed by asymmetrical positive T waves (appropriate discordance) (Figure 5).

Figure 5



In summary: Uncomplicated CLBBB is characterized by the presence of "appropriate" discordance between ST segments and T waves relative to the QRS polarity. Thus, ST segment elevation with upper concavity is observed, followed by positive and asymmetrical T wave with slow ascending ramp and more rapid descending ramp in precordial leads with complexes of the rS or QS type (right precordial leads); ST segment depression of upper convexity followed by asymmetrical negative T wave with slower descending ramp than the ascending one in the left leads I, aVL, V5 and V6. They are "secondary" changes in ventricular repolarization due to changes in the sequence of depolarization with a wide $^{QRS}/_{ST-T}$ angle but with normal ventricular gradient. This gradient is defined as the algebraic sum (or net difference) of the QRS area and the T area of the ECG.

Electrocardiographic characteristics of CLBBB associated with acute MI

CLBBB with ischemic T waves may be characterized by symmetrical limbs resembling “seagull wings” as illustrated in Figure 3-2. In the present case, however, CLBBB complicated with acute antero-apical infarction is characterized by ST segment elevation with upper convexity in leads I, aVL, and V5-6 as illustrated in Figure 3-3.

Patients with suspected acute coronary syndrome and acute myocardial infarction (AMI) who present with CLBBB represent a particular diagnostic and therapeutic challenge because both intraventricular conduction disorders and infarction affect the first portions of the QRS complex (Q wave). Although current guidelines recommend early reperfusion therapy for patients with new CLBBB, or when the CLBBB is not known to have previously existed, only a minority of patients with CLBBB are finally diagnosed with AMI and many will not have an occluded culprit artery diagnosed in the catheterization laboratory. This approach to treatment may lead to a significant number of patients vulnerable to the risks of fibrinolytic therapy without the probability of benefit. Also there are unnecessary risks and costs of catheterization due to false positive AMI diagnoses. Therefore, alternative strategies are needed to identify AMI in clinically stable patients with CLBBB that do not have ECG findings highly specific to ST segment elevation MI.

Generally, in patients with uncomplicated CLBBB the presence of ST segment elevation in the right precordial leads V1-V3 is a normal consequence of the abnormal sequence of ventricular depolarization which results in an obligatory change in the sequence of ventricular repolarization. The presence of ST segment elevation matching the positive QRS polarity in the left leads I, aVL, V5 and V6 in the admitting ECG in this case (Figure 1) is abnormal. This is also illustrated in Figure 3-3 and in Figure 6.

Figure 6

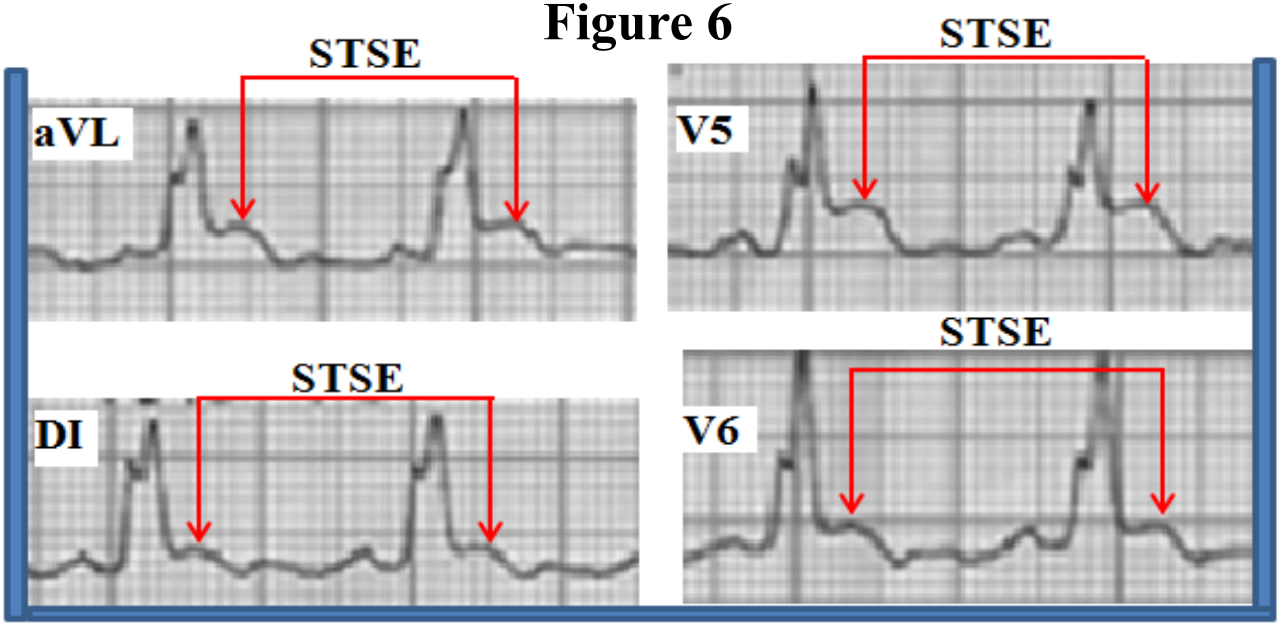


Figure 6 shows ST elevation in left leads I, aVL, V5 and V6 matching the preceding QRS complex. Additionally, the ST segment is upwardly convex. This aspect is always abnormal.

Also, the very unusual mismatching ST segment elevation (>5 mm) in the right precordial leads may indicate the presence of AMI.

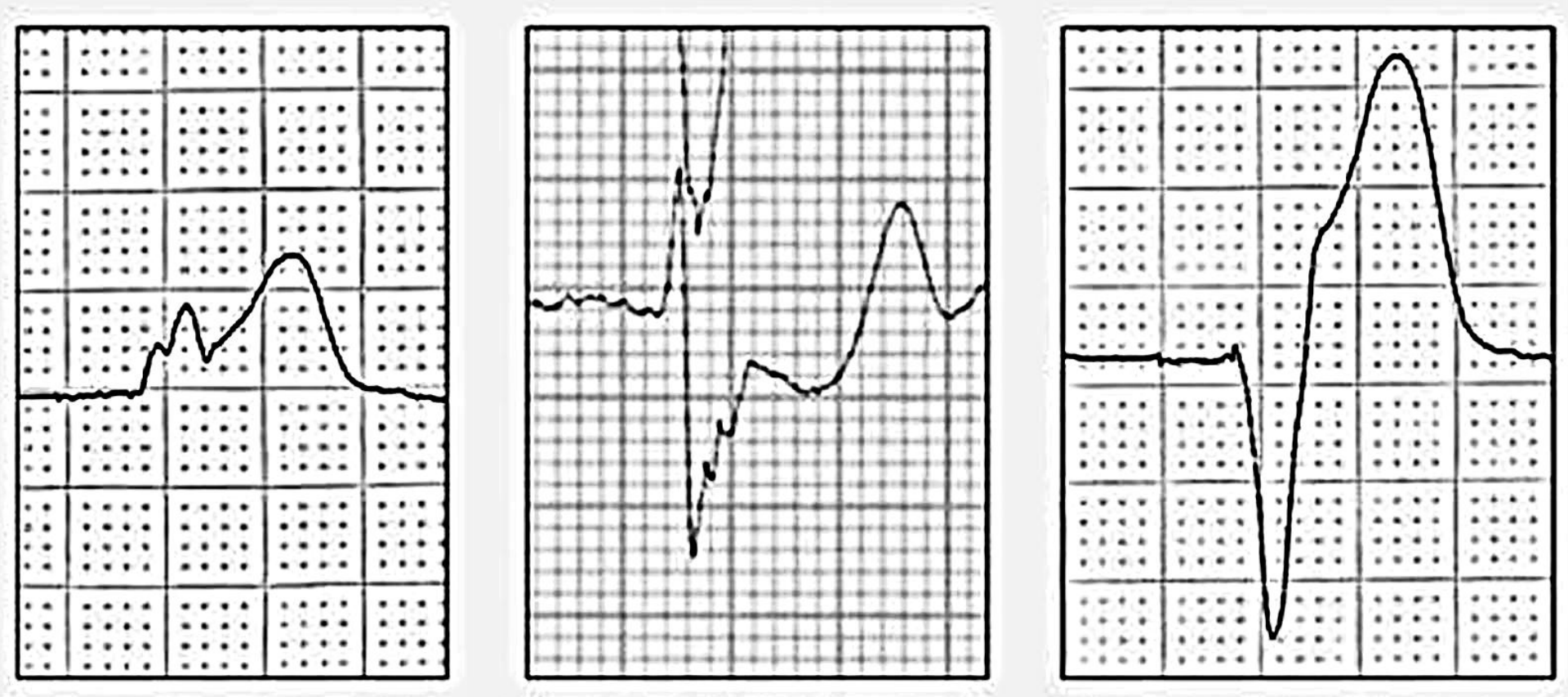
In 1996 Sgarbossa et al (**Sgarbossa 1996; Sgarbossa 1996**) published an ECG scoring system derived from data obtained in the GUSTO study (**G**lobal **U**tilization of **S**treptokinase and **T**issue Plasminogen Activator for **O**ccluded Coronary Arteries) published in 1993 with the aim of assessing the changes in ventricular repolarization in patients with AMI in the presence of *de novo* CLBBB. The following three criteria were included in the score.

•**Criterion A:** ST elevation ≥1 mm in leads with positive QRS complexes (inappropriate concordance): **5 points**. This criterion is most specific for the diagnosis of infarction. Figure 7A.

•**Criterion B:** ST segment depression ≥1 mm in the leads with predominantly negative QRS complexes, V1-V2-V3 (inappropriate concordance): **3 points**. This is the least specific criterion. Figure 7B.

•**Criterion C:** ST segment elevation ≥5 mm in the leads with predominantly (rS) or completely negative (QS) QRS complexes (V1, V2 or V3) (inappropriate discordance): **2 points**.

Figure 7 - Sgarbossa's Criteria



A. Concordant STSE >1 mm

B. Concordant ST depression >1 mm V1,V2, V3

C. Discordant ST-elevation >5 mm or 0.25 QRS

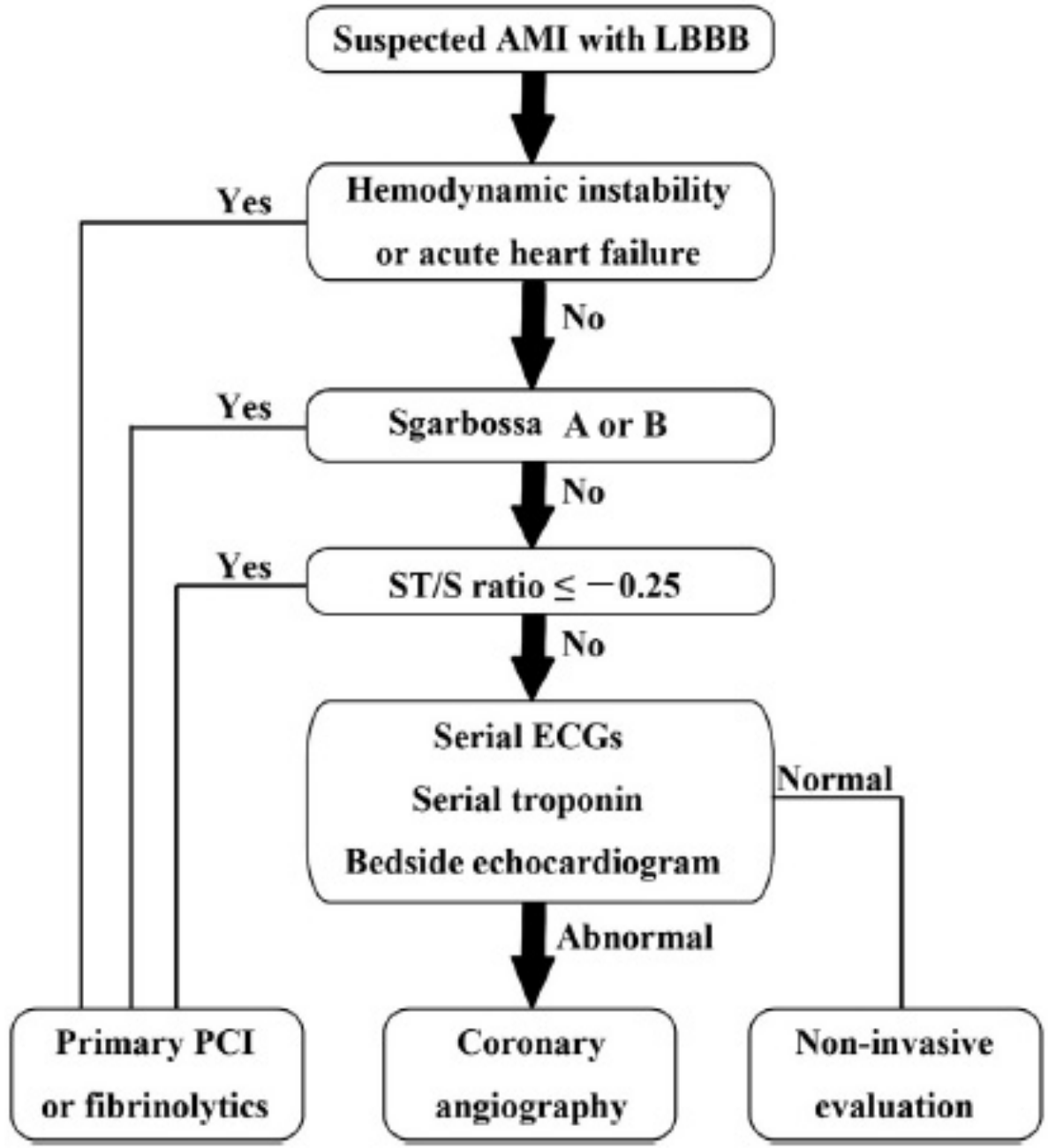
A score of ≥3 points is highly predictive of STEMI and has a high specificity (90%), but a low sensitivity.

The new 2013 STEMI guidelines have made a significant change withdrawing the previous recommendations for early reperfusion therapy in patients with suspicion of ischemia and new or supposedly new CLBBB. The new guidelines do not acknowledge that some patients with suspicion of ischemia and CLBBB have MI, and sparing reperfusion therapy in them could be fatal.

The Sgarbossa ECG criteria are the most validated tool to help in the diagnosis of AMI in the presence of CLBBB. A Sgarbossa score of ≥3 has an excellent specificity (98%) and positive predictive value for acute coronary occlusion confirmed by angiography. Thus, Cai et al (**Cai 2013**) have proposed a new diagnostic algorithm and triage strategy adding the Sgarbossa criteria to quickly identify those patients with chest pain and new or supposedly new CLBBB who have an acute coronary occlusion. This is a high risk population in whom urgent reperfusion therapy would not be applied according to the 2013 guideline. This new algorithm may also significantly reduce the inappropriate use of the catheterization lab and prevent inappropriate fibrinolytic treatments as recommended by the 2004 AMI guidelines.

The algorithm by Cai et al for patients with suspicion of AMI and *de novo* CLBBB is illustrated in Figure 8.

Figure 8


Suspected AMI with LBBB
Hemodynamic instability or acute heart failure
Yes
No
Sgarbossa A or B
Yes
No
ST/S ratio ≤ −0.25
Yes
No
Serial ECGs
Serial troponin
Bedside echocardiogram
Normal
Abnormal
Primary PCI or fibrinolytics
Coronary angiography
Non-invasive evaluation

In a systematic review and meta-analysis made by Tabas et al (**Tabas 2008**), the Sgarbossa scoring system revealed that a score ≥3, which represents ≥1 mm of matching elevation of the ST segment or ≥1 mm of matching depression of ST segment from V1 to V3, is useful for the diagnosis of AMI in patients with CLBBB in the ECG. Also, the Sgarbossa score proved to have a good to excellent interobserver variability (**Sokolove 2000**). A score of 2, representing 5 mm or more of a mismatching ST shift, proved to have less accurate positive probability. The Sgarbossa score of 0 was not useful in ruling out AMI. The great drawback of the Sgarbossa criteria for the diagnosis of AMI in the presence of CLBBB is its low sensitivity (approximately 20%) according to the recently published meta-analysis.

Criterion C, ≥5 mm discordant ST segment elevation in the right precordial leads, is problematic as a criterion because it may be found in patients with CLBBB who do not have AMI, especially when very deep S waves are present in leads V1-V3. Smith et al (**Smith 2012**) suggested that it would be more rational to analyze the proportionality between ST segment elevation and S wave depth (ST/S). According to these authors, the discordance should be proportional to QRS amplitude. The substitution of the measurement of absolute discordant ST segment elevation ≥5 mm in the right precordial leads by an ST/S ratio ≤0.25 significantly improved the diagnostic utility for recognizing acute myocardial infarction. This new criterion is based on the electrophysiological principle that repolarization voltages are always proportional to depolarization voltages. Figure 9 shows an example of this new substitute for the C criterion of Sgarbossa.

Figure 9

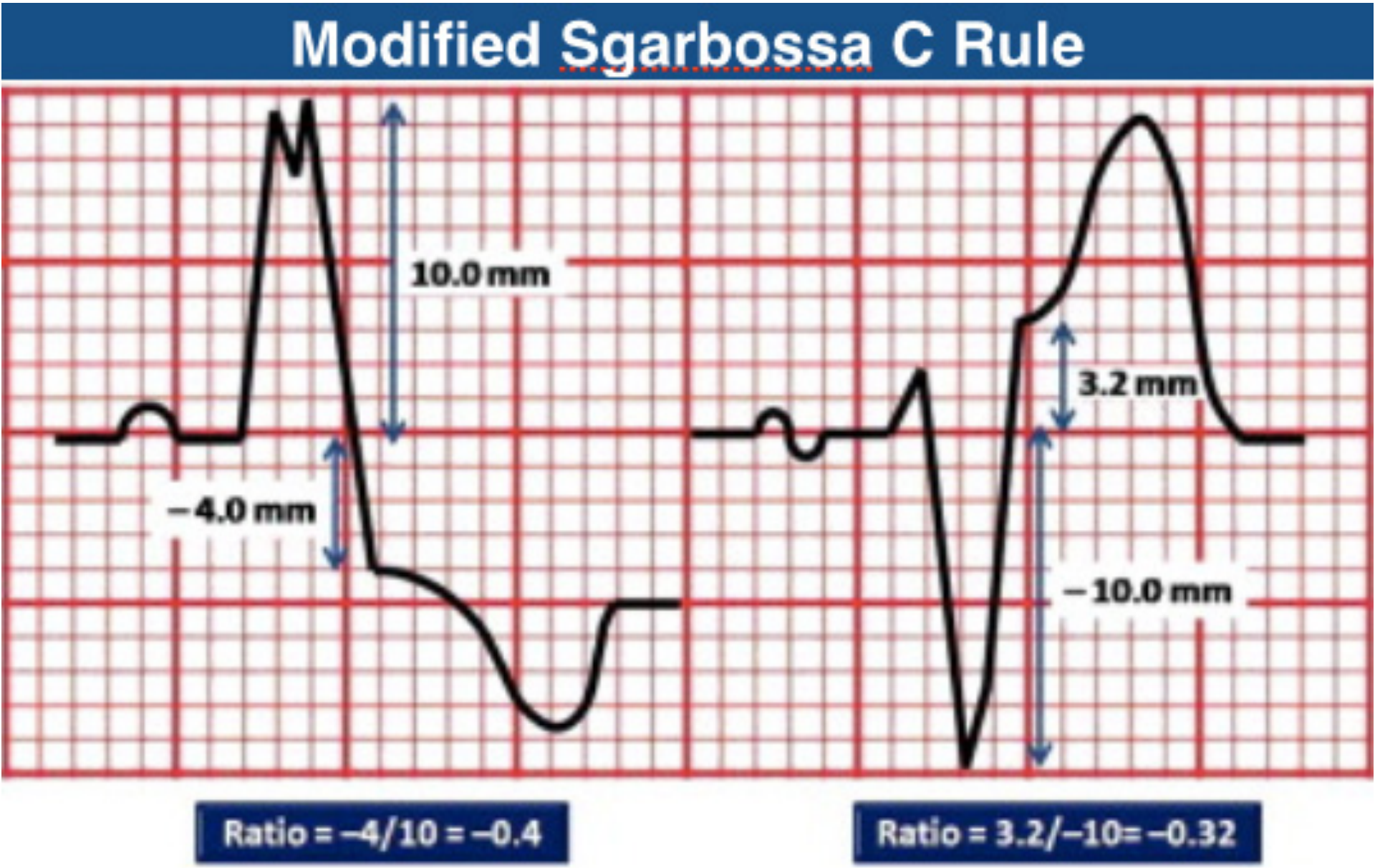
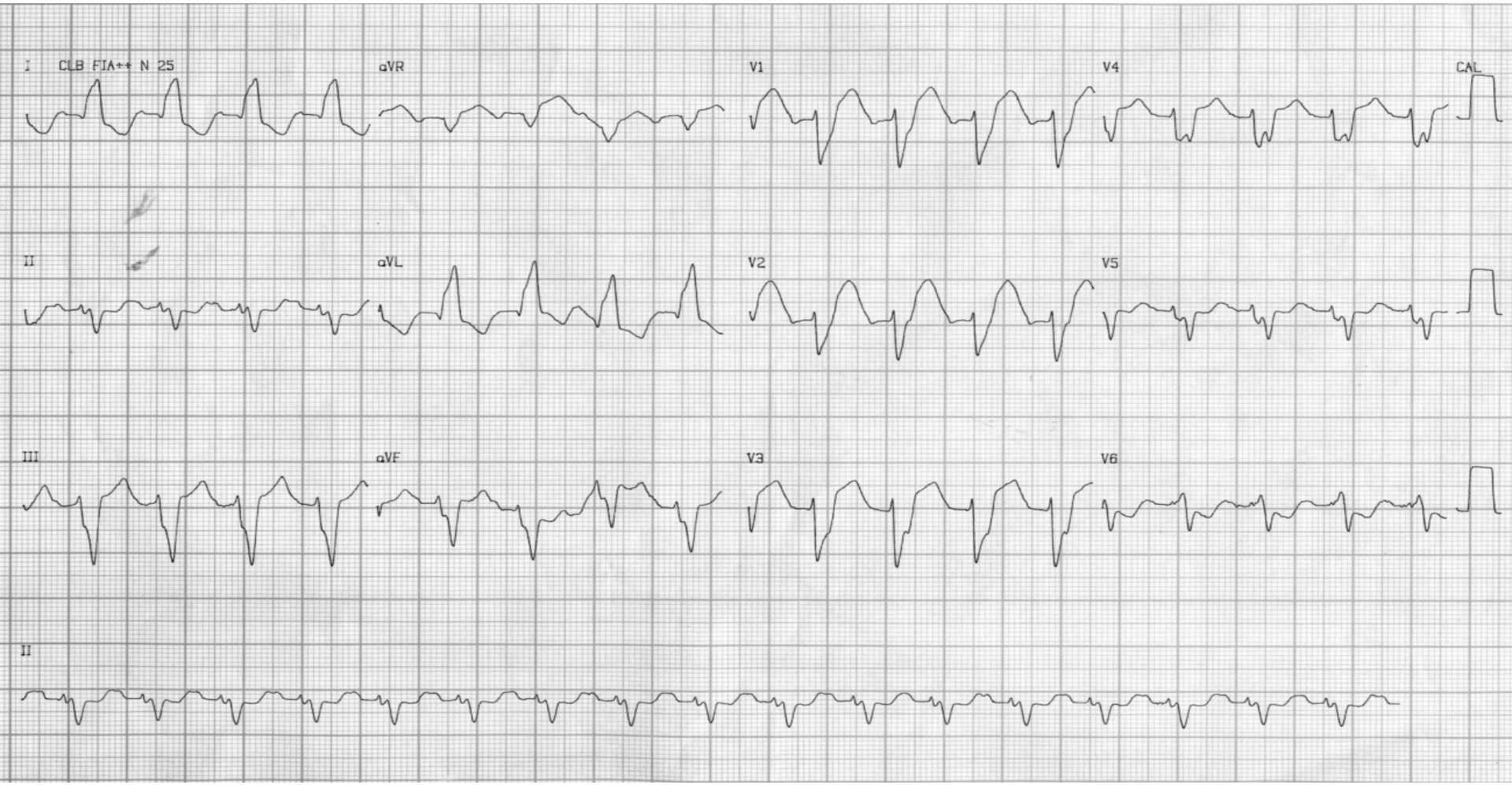

Modified Sgarbossa C Rule
10.0 mm
−4.0 mm
3.2 mm
−10.0 mm
Ratio = −4/10 = −0.4
Ratio = 3.2/−10= −0.32

Figure 10 illustrates this new criterion in a patient with CLBBB who has an AMI due to total occlusion of the left anterior descending coronary artery.

Figure 10



Electrocardiographic diagnosis: Sinus tachycardia, heart rate 115 bpm, extreme left axis QRS in the frontal plane (-60°), QRS duration >120 ms, qR in I and aVL, decrease in $^{QRS}/_{ST\text{-}T}$ ratio in V1 and V2 (close to 1/1). This is an electrocardiographic sign of CLBBB complicated with acute anterior wall infarction. ST segment with upper convexity in the right precordial leads (normal: upwardly concave). A notch of 50 ms is present in the ascending ramp of the S wave in V3 and V4 (Cabrera's sign), and the ST/S ratio in lead V2 is < -0.25.

Studies have indicated that the presence of concordant elevation (Sgarbossa criterion #1) is related to AMI with a culprit artery in 71.4% of the cases. It was also observed that when concordant ST segment elevation is absent, CLBBB is not related to acute coronary occlusion. CLBBB without concordant ST segment elevation should not be a criterion for the activation of the catheterization lab for reperfusion therapy.

In the example in Figure 11, the presence of concordant ST segment elevation is seen in lead II and the ST/S ratio >0.2 in leads III and aVF. Coronary angiography revealed total occlusion of the right coronary artery treated by primary angioplasty.

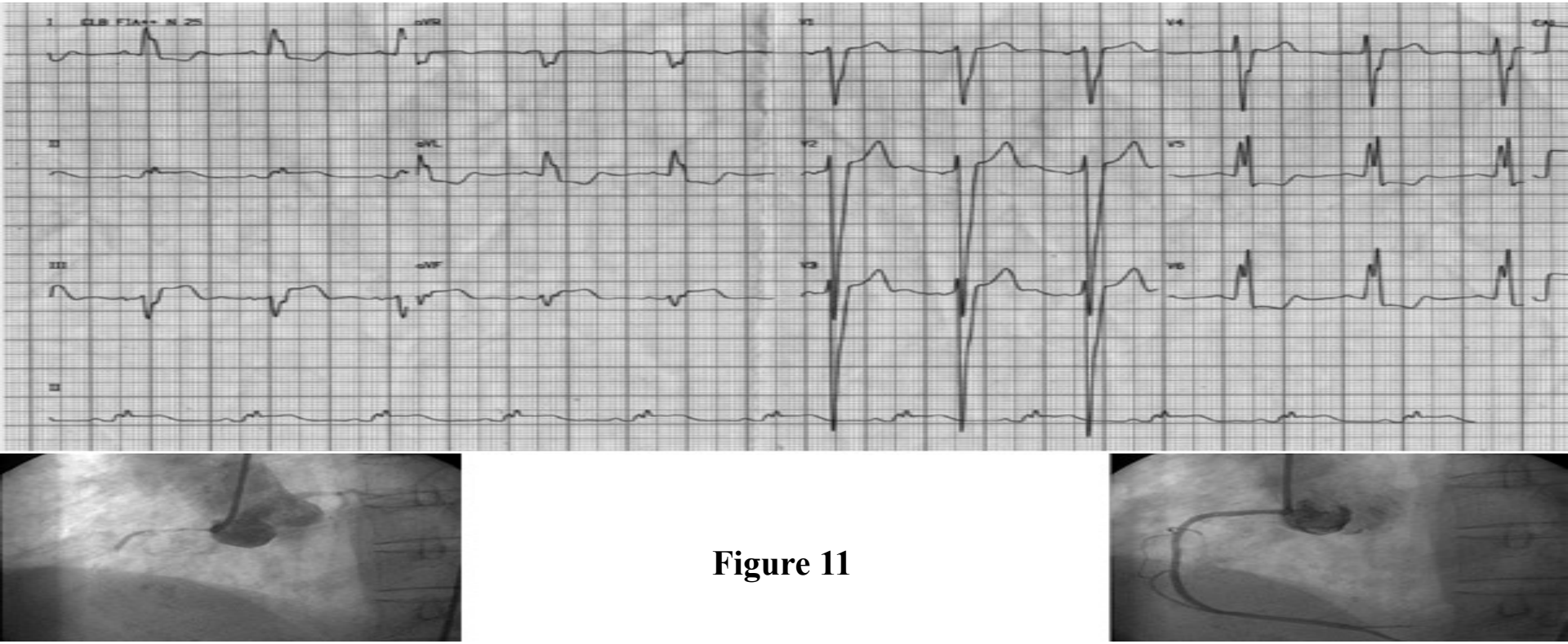


Figure 11

ECG diagnosis: Sinus rhythm, HR 75 bpm, QRS axis -5°, typical CLBBB pattern. ST segment elevation is present in III, II and aVF; ST segment elevation is greater in III than in II, indicating that the injury vector points to +120°, which suggests proximal occlusion of the right coronary artery. There is concomitant ST segment depression in I and aVL and in the left precordial leads because the injury vector moves away from the left superior and apical region of the LV.

Sørensen et al (**Sørensen 2013**) assessed ECGs and mortality in a pre-hospital cohort of 4905 consecutive patients suspected of having AMI. CLBBB was considered to be present when QRS duration ≥ 120 ms associated to classical morphological criteria. Both mortality and angiographic data were measured from a database. The whole population was divided into four groups: with or without AMI and with or without CLBBB. Mortality was evaluated by the Kaplan-Meier analysis and compared to the log-rank statistical analysis. The presence of AMI was diagnosed in 954 patients, from whom 118 also had CLBBB. From 3951 patients without AMI, 436 had CLBBB.

The patients with AMI and CLBBB underwent revascularization less frequently than those patients with AMI without CLBBB (24 vs. 54%, $p<0.001$). AMI with CLBBB was considered as *de novo* in 43 patients; and among them only 2 underwent primary angioplasty. One-year mortality was 47.2% in the group of patients with AMI+CLBBB, 17.5% in patients with AMI without CLBBB, 20.8% in those with CLBBB without AMI, and 8.6% in patients without AMI or CLBBB (log-rank<0.001) respectively. From the patients with AMI and CLBBB, only 25% underwent revascularization and only a few were referred to emergency primary angioplasty. The approach of improving triage and pre-hospital identification of patients at high risk with CLBBB and chest pain may improve the result.

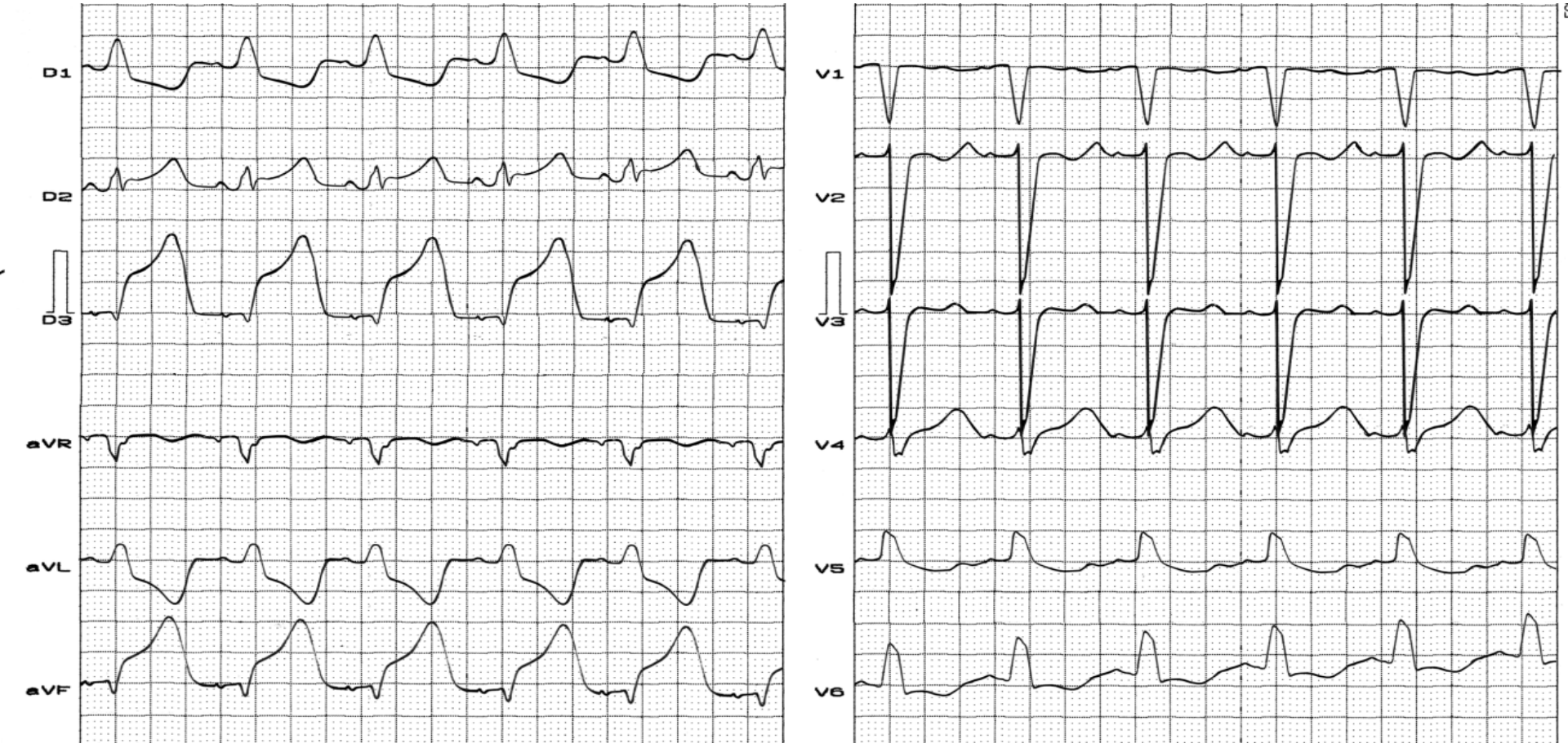
Al-Faleh et al (**Al-Faleh 2006**) validated Sgarbossa's criteria prospectively in a large population. These authors verified that patients with Sgarbossa score ≥ 4 presented a greater mortality when compared to those with score below 3. The authors concluded that these criteria constitute a simple approach and a practice that optimizes risk-benefit and improves the therapeutic approach.

The guidelines recommend urgent reperfusion for patients with new CLBBB (nLBBB), similar to patients with STEMI without nLBBB. However, the comparison between both groups of patients is limited. Both groups were compared by Yeo et al (**Yeo 2012**) in regard to clinical characteristics, type of treatment, and results. In general, patients with nLBBB had a greater percentage of co-morbidities in comparison to those with STEMI without this conduction disorder. In comparison to patients with STEMI, those with nLBBB were less likely to receiving reperfusion therapy in the acute phase (primary percutaneous coronary intervention - PCI) (93.9% vs. 48.3%, $p<0.0001$) and had a greater percentage of door-to-balloon time ≤ 90 minutes (76.8% vs. 34.5%, $p<0.0001$). Mortality rates were greater for the patients with nLBBB in comparison to those with STEMI without nLBBB (13.3% vs. 5.6%, $p<0.0001$). After multivariate adjustment, the patients with nLBBB were not associated with an increased risk of in-hospital mortality. The authors concluded that the patients with nLBBB presented a greater percentage of co-morbidities and were less prone to receiving reperfusion therapy (PCI) in the acute phase. In spite of these differences, adjusted mortality rates were similar between patients with nLBBB and those with STEMI.

In the following sequence, two cases of nLBBB complicated with inferior and anterior infarction in the acute phase are illustrated.

Figure 12

Name: TRS; **Sex:** Male; **Age:** 67 y.o.; **Race:** White; **Weight:** 64 Kg; **Height:** 1.65 m; **Date:** Oct 1, 1998; **Time:** 17:30.



Clinical diagnosis: Acute inferior wall myocardial infarction (AMI) associated with nLBBB evolving after 5 hours.

ECG diagnosis: nLBBB + ST segment elevation in III, aVF and II. Pardee's complex are noted in the inferior wall leads. Typical ECG findings of CLBBB associated with acute inferior wall MI are seen in leads III and aVF resembling monophasic action potentials (Pardee's complex). ST segment elevation in III>II indicates that the injury vector points to +120° and indicates proximal occlusion of the right coronary artery.

Figure 13

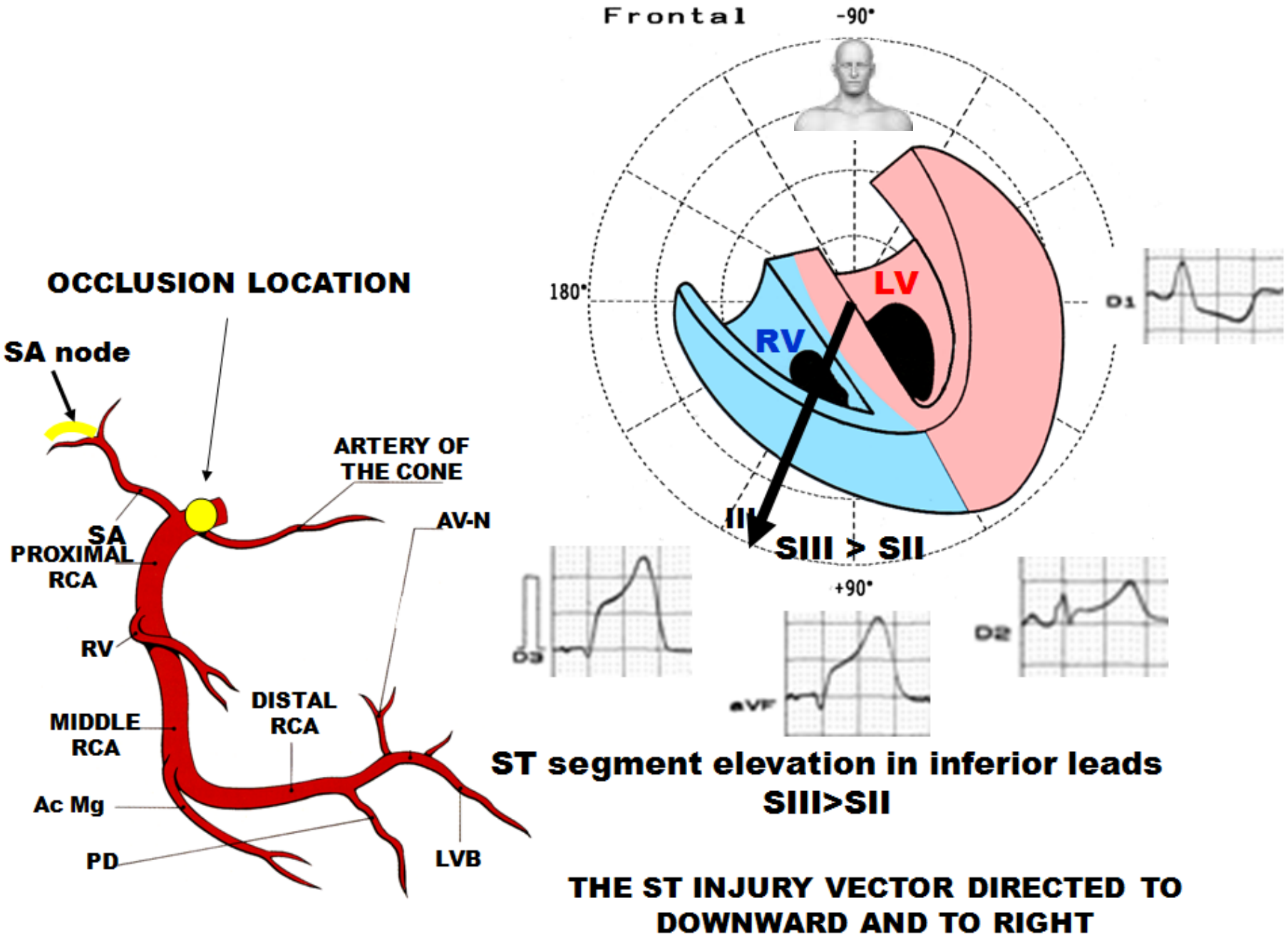
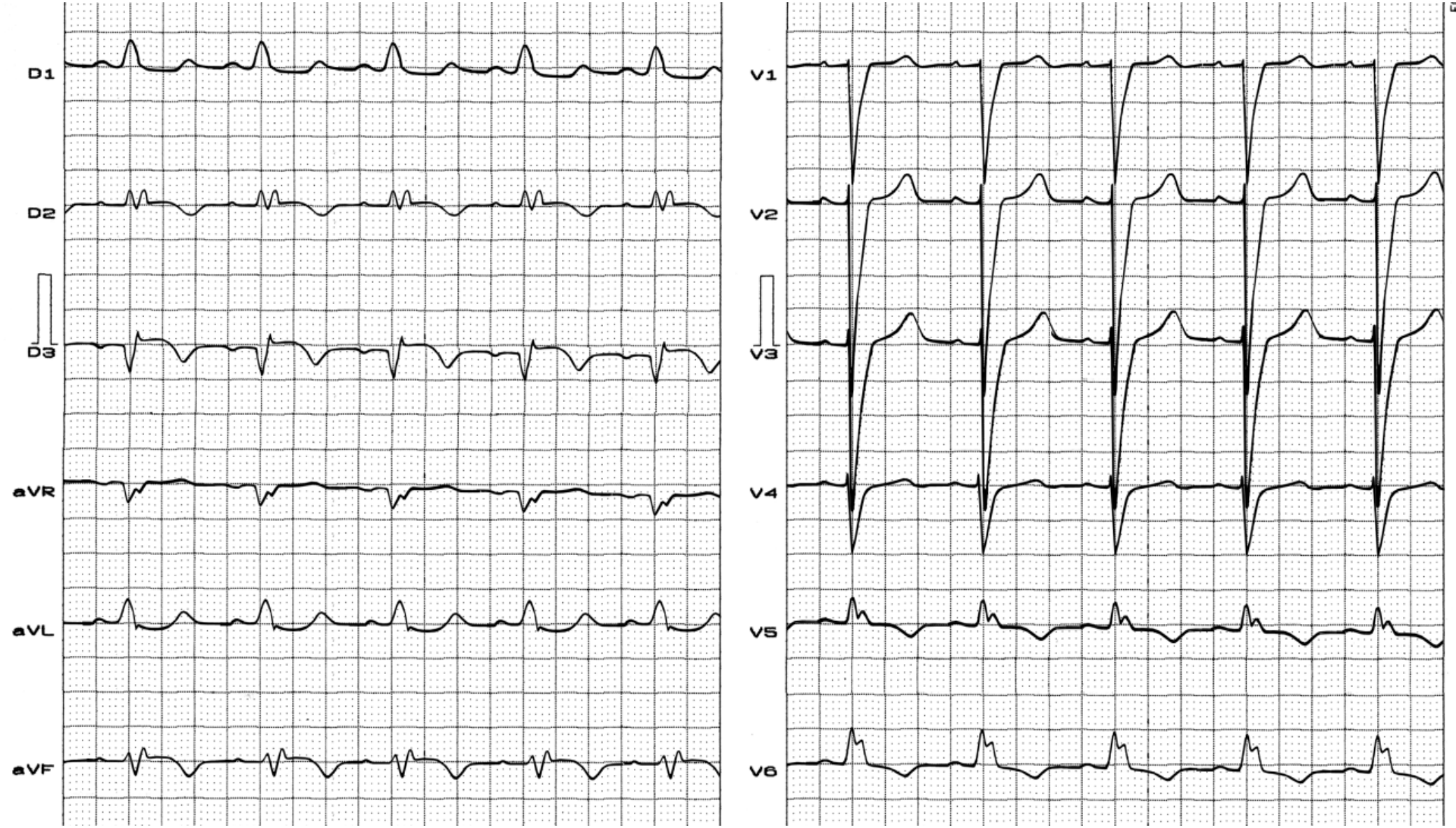

OCCLUSION LOCATION
SA node
ARTERY OF THE CONE
AV-N
SA PROXIMAL RCA
RV
DISTAL RCA
MIDDLE RCA
Ac Mg
PD
LVB
Frontal
-90°
180°
LV
RV
III
SIII > SII
+90°
D1
D2
D3
aVF
ST segment elevation in inferior leads
SIII>SII
THE ST INJURY VECTOR DIRECTED TO DOWNWARD AND TO RIGHT

Figure 14

Name: TRS **Sex:** Male; **Age:** 67 yrs; **Race:** White; **Weight:** 64 Kg; **Height:** 1.65 m; **Date:** Oct 2, 1998; **Time:** 20:30; **Note:** The ECG belongs to the same patient 27 hours later.

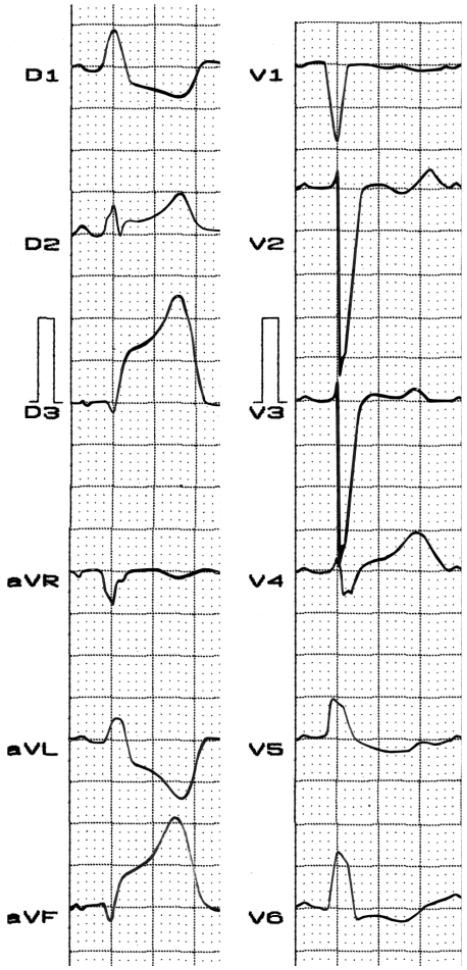


Clinical diagnosis: Inferior subepicardial ischemia.

ECG diagnosis: Q wave of necrosis, only visible in lead III. Inferior subepicardial ischemia. A decrease in R waves voltage is seen in V5-V6, which indicates additional involvement in the LV apical region.

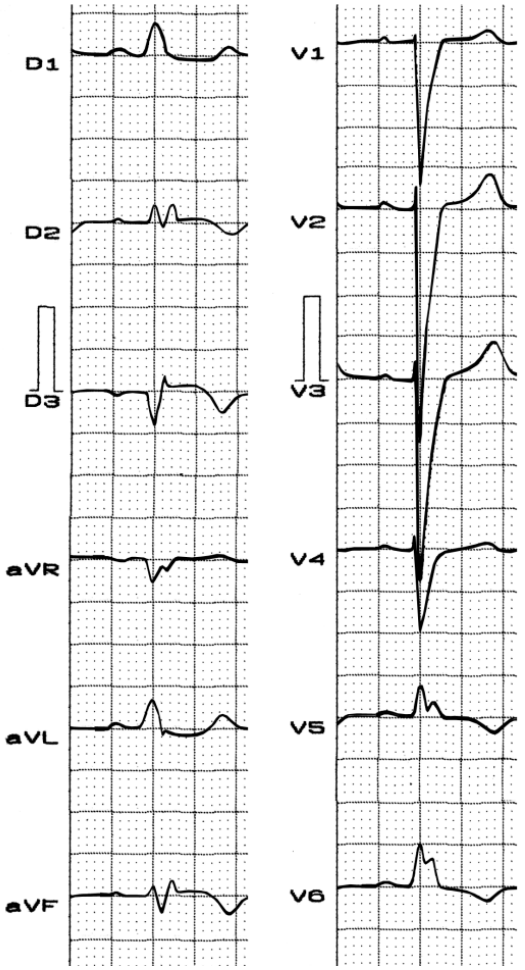
Figure 15

Name: TRS Date: Oct 1, 1998 Time: 17:30



"Pardee's complex" transmural injury current.
Q wave of necrosis only visible in lead III.

Name: TRS Date: **Oct 2,** 1998 Time: **20:30**

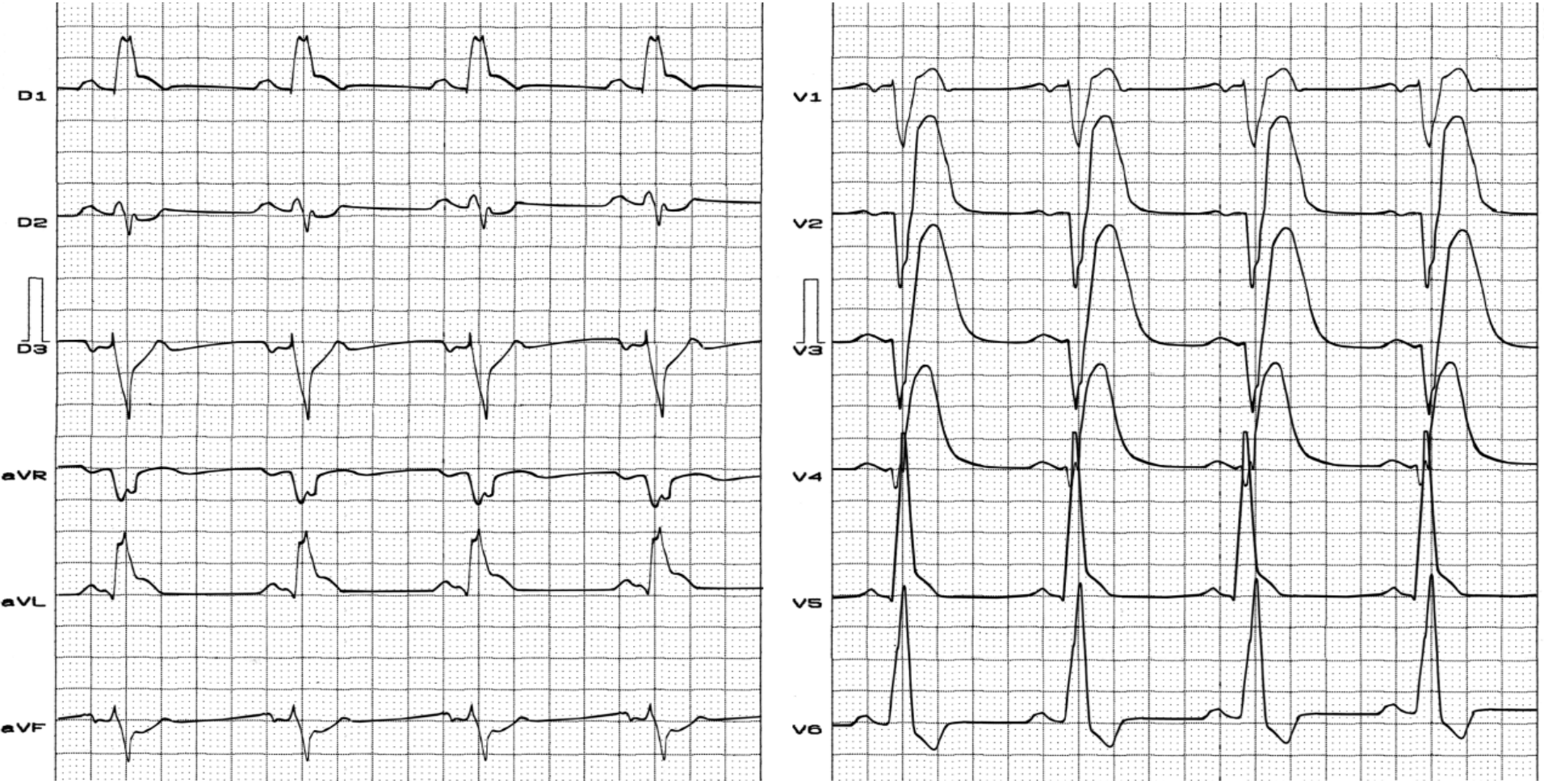


Ischemia in inferior wall (symmetrical, negative T wave, in II, III and aVF). Decrease of R wave voltage in V5-V6, which points out additional involvement in the apical region of the LV free wall (the red dotted line compares the voltage at both times).

The tracing in Figure 16 shows an example of a patient with anterior wall infarction complicated by CLBBB.

Figure 16

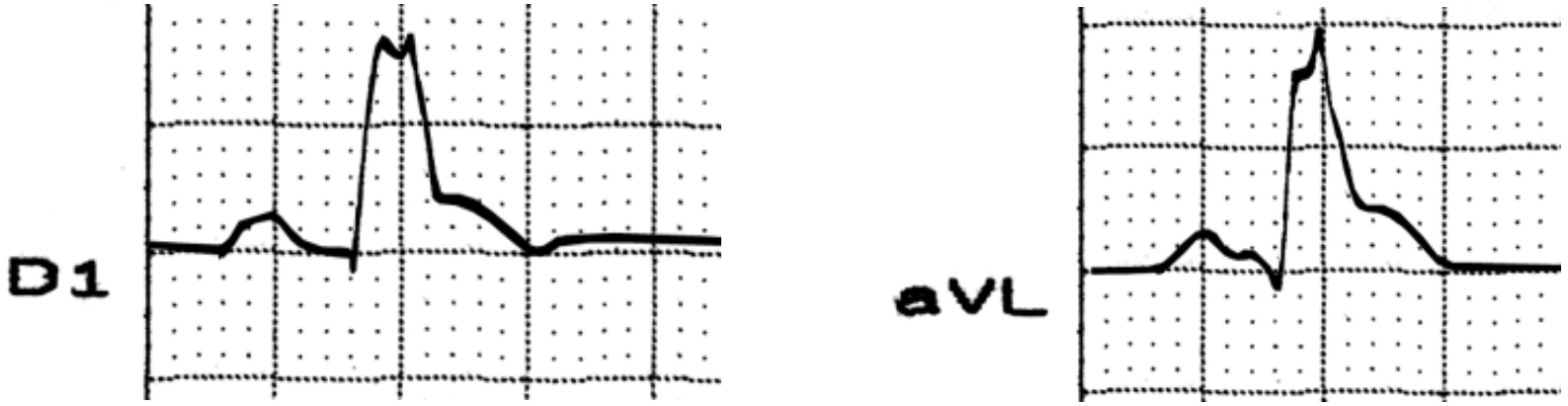
Name: ECA; **Sex:** Male; **Age:** 61; **Race:** White; **Weight:** 69 Kg; **Height:** 1.80 m.



Clinical diagnosis: Hypertension, type II diabetes mellitus, acute myocardial infarction.

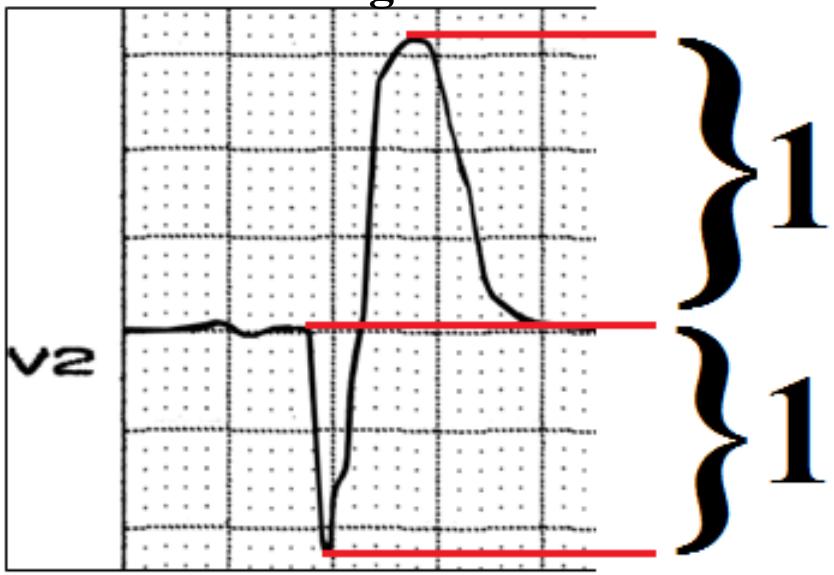
ECG diagnosis: nLBBB associated with anterolateral wall MI. A qR pattern in leads I and aVL is present with significant transmural anterolateral injury current. ST segment elevation with upper convexity matching the QRS complex in lead I and aVL is present. The $^{QRS}/_{ST\text{-}T}$ ratio = or <1 (normal 2:1 or 3:1) (see Figure 17).

Figure 16



Transmural injury current is illustrated in the high lateral wall concordant with QRS, a finding that is always abnormal.

Figure 17



$^{QRS}/_{ST\text{-}T}$ ratio = or <1 (normal 2:1 or 3:1).

Case report number 2
Uremic Pericarditis Resembling Acute Myocardial Infarction

Case Presentation

MAR is a 70 yr old woman with long-standing hypertension treated with an angiotensin converting enzyme inhibitor (ACEI). She was admitted to the emergency department with symptoms of progressive dyspnea on exertion that had worsened over the past 20 days. She was found to be in severe respiratory failure. She had a past history of left nephrectomy for uncertain reasons. On physical examination she appeared very dyspneic with a blood pressure of 160/80 mmHg, heart rate of 115 bpm, and with some premature beats. Laboratory assessment revealed low peripheral oxyhemoglobin saturation (SpO2 86%; normal SpO2 95-100% breathing room air at sea level), and high serum potassium (K^+ 6.2 mEq/L). Her initial ECG (**Figure 1**) revealed peaked T waves consistent with hyperkalemia (hyperpotassemia).

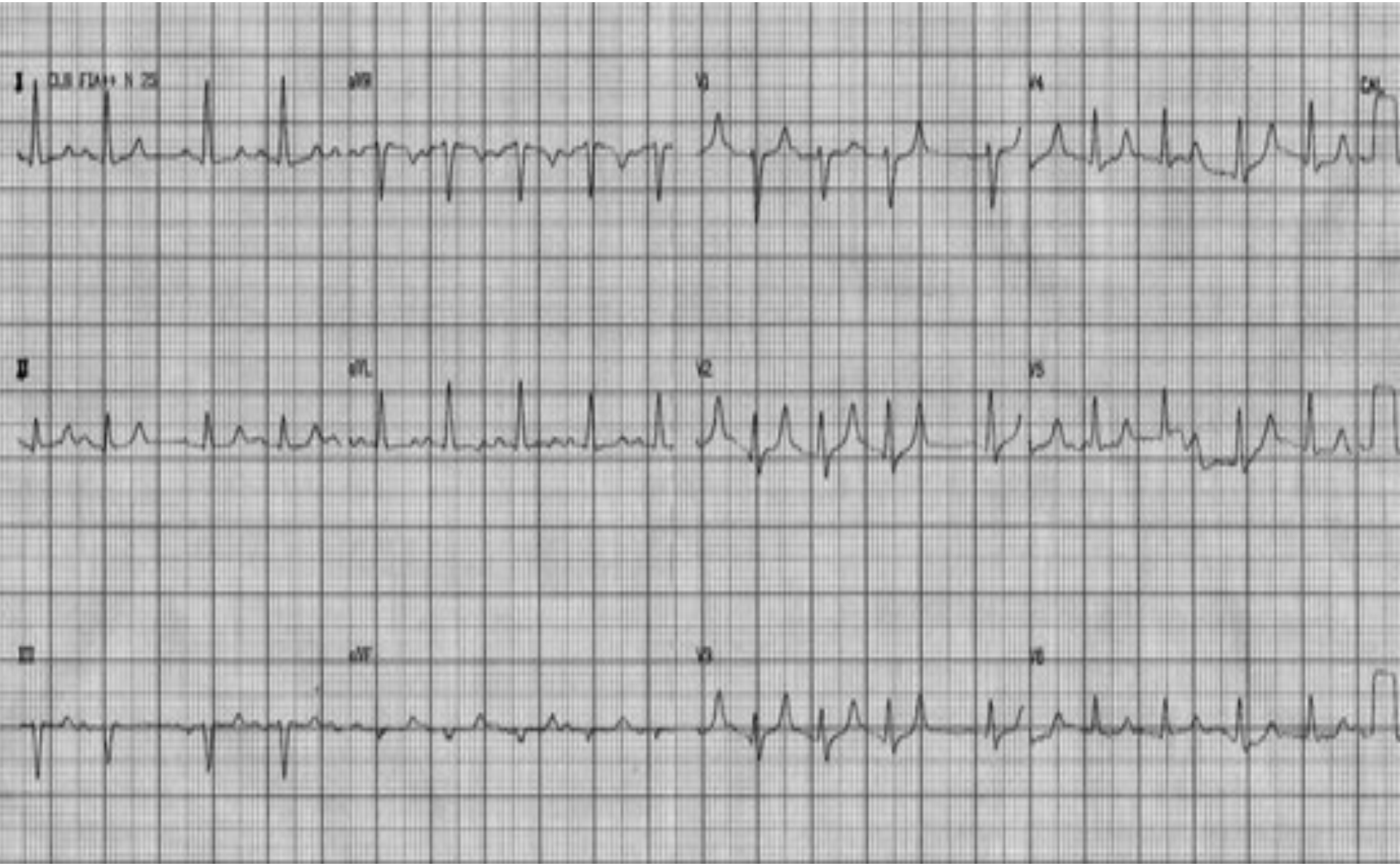
Initial management:

I) Oral tracheal intubation with mechanical ventilation (OTI/MV).

II) Intravenous administration of: 10 ml calcium gluconate (to stabilize the myocardial membrane), sodium bicarbonate (1 mEq/Kg, IV bolus), and regular insulin with glucose (10 U IV + 50 gr of glucose). After these measures, the T wave abnormalities in the ECG improved (**Figure 3**).

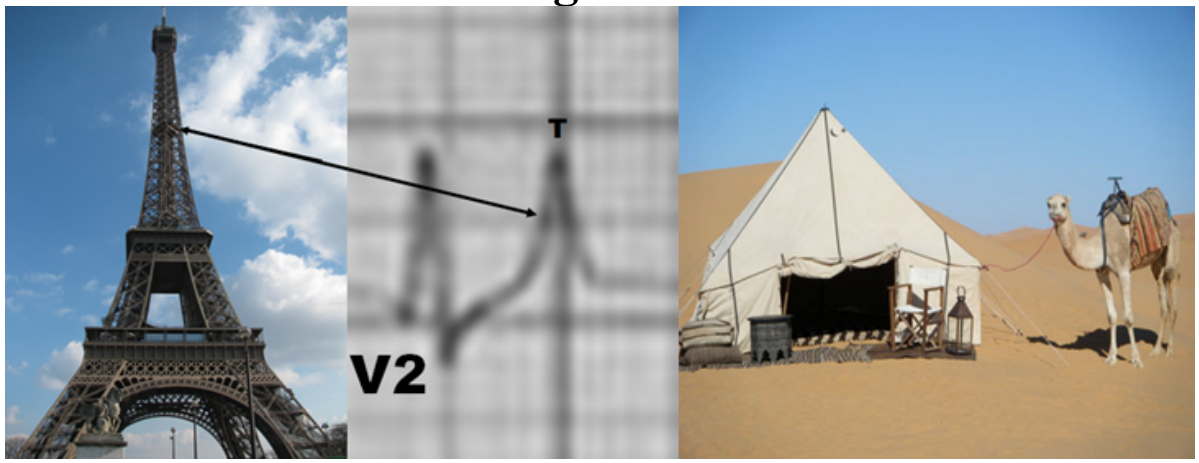
The patient subsequently developed intense chest pain of uncertain etiology, and serial ECGs, illustrated in **Figures 4 and 5**, were obtained. The abnormalities observed in these tracings were initially interpreted as an ST Elevation Myocardial Infarction (STEMI). ECG diagnosis of STEMI requires the presence of >1.0 mm ST segment elevation in at least two contiguous ECG leads in the frontal plane or lateral precordial leads (V4-5), >2.0 mm in the right precordial leads (V1-3), or presumably new complete left bundle branch block. Because of her ECG findings she immediately underwent coronary angiography that revealed normal coronary arteries.

Figure 1



ECG diagnosis: Sinus tachycardia with peaked, symmetrical T waves with narrow base (desert-tent or Eiffel tower-like T waves) (**Figure 2**). These are the earliest repolarization abnormalities when serum potassium levels exceed >5.5mEq/L.

Figure 2



Tall, peaked/pointed/thin T waves resembling a desert tent or Eiffel Tower

Figure 3

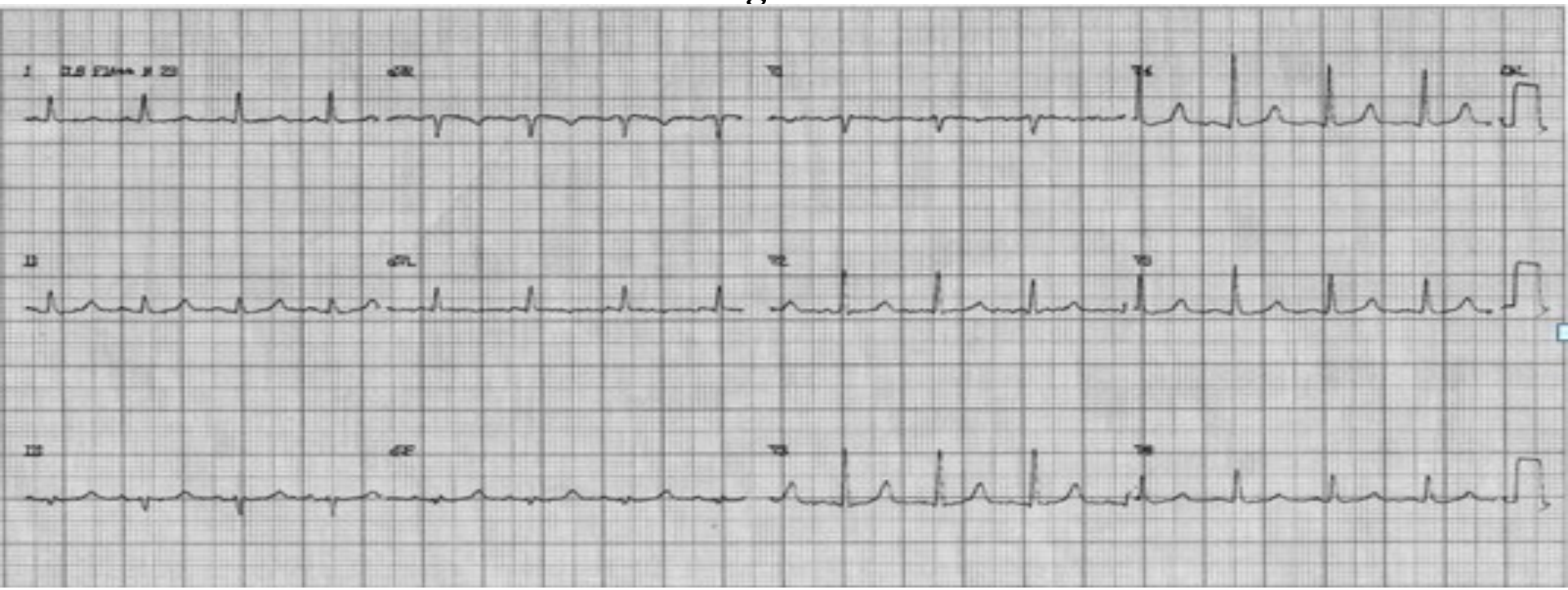


Figure 3. There is regression of the T wave abnormalities after initiating the treatment.

Table 1 describes ECG abnormalities at various serum potassium levels.

Table 1

Serum potassium level	Electrocardiographic patterns found
K^+ > 5.5 mEq/L and < 6.5 mEq/L: Mild hyperkalemia	Repolarization abnormalities: tall, symmetrical and narrow-based T waves resembling a desert tent or the Eiffel tower. Usually this is the earliest sign of hyperkalemia.
K^+ > 6.5 mEq/L and < 7.0 mEq/L: Moderate hyperkalemia	Progressive paralysis of the atria muscle cells results in widening and flattening of P waves due to slow interatrial conduction with eventual disappearance of the P wave. Sinus rhythm, however, is maintained through interatrial conduction pathways (sino-ventricular rhythm). First degree AV block may be present. Decrease in R wave voltage, prolonged QRS duration, and ST segment changes resembling acute myocardial injury, dialyzable injury current or Brugada phenocopy may be seen.
K^+ > 7.0 mEq/L and < 7.5 mEq/L or Severe hyperkalemia	Dromotropic alterations and bradycardia: QRS widening with bizarre morphology, high degree AV block, junctional or ventricular escape rhythms, pseudo CLBBB, CRBBB, fascicular blocks, sinus bradycardia, or atrial fibrillation with slow heart rate response. Wide QRS-T complexes resembling sine waves usually seen in more severe hyperkalemia sometimes occur.
K^+ > 8.0 mEq/L and < 9.0 mEq/L Severe hyperkalemia	Decrease in R wave voltage, prominent S waves, diffuse QRS complex widening resembling CLBBB or CRBBB; extreme SAQRS shifts in the FP suggesting LAFB or LPFB. QRS complex widening is differentiated from true bundle branch blocks where the delay is more in the final or mid-final QRS, while in hyperkalemia the delay is always more diffuse. In the late phase the QRS complex merges with the T wave resulting in a smooth, diphasic sine wave appearance with QT interval prolongation. The intensification of ST segment depression or elevation, known as a dialyzable injury current, may eventually resemble the type 1 Brugada pattern or acute anteroseptal STEMI with absence of R waves from V_1 through V_4 and marked ST segment elevation (transmural injury current).
K^+ > 9.0 mEq/L Extreme hyperkalemia	Wide QRS complexes, sino-ventricular rhythm (absence of P waves), irregular rhythm, pseudo-AF, very wide and bizarre QRS complexes, ventricular tachycardia, ventricular fibrillation, and asystole when potassium concentrations exceed 12 - 14 mEq/L.

Figure 4

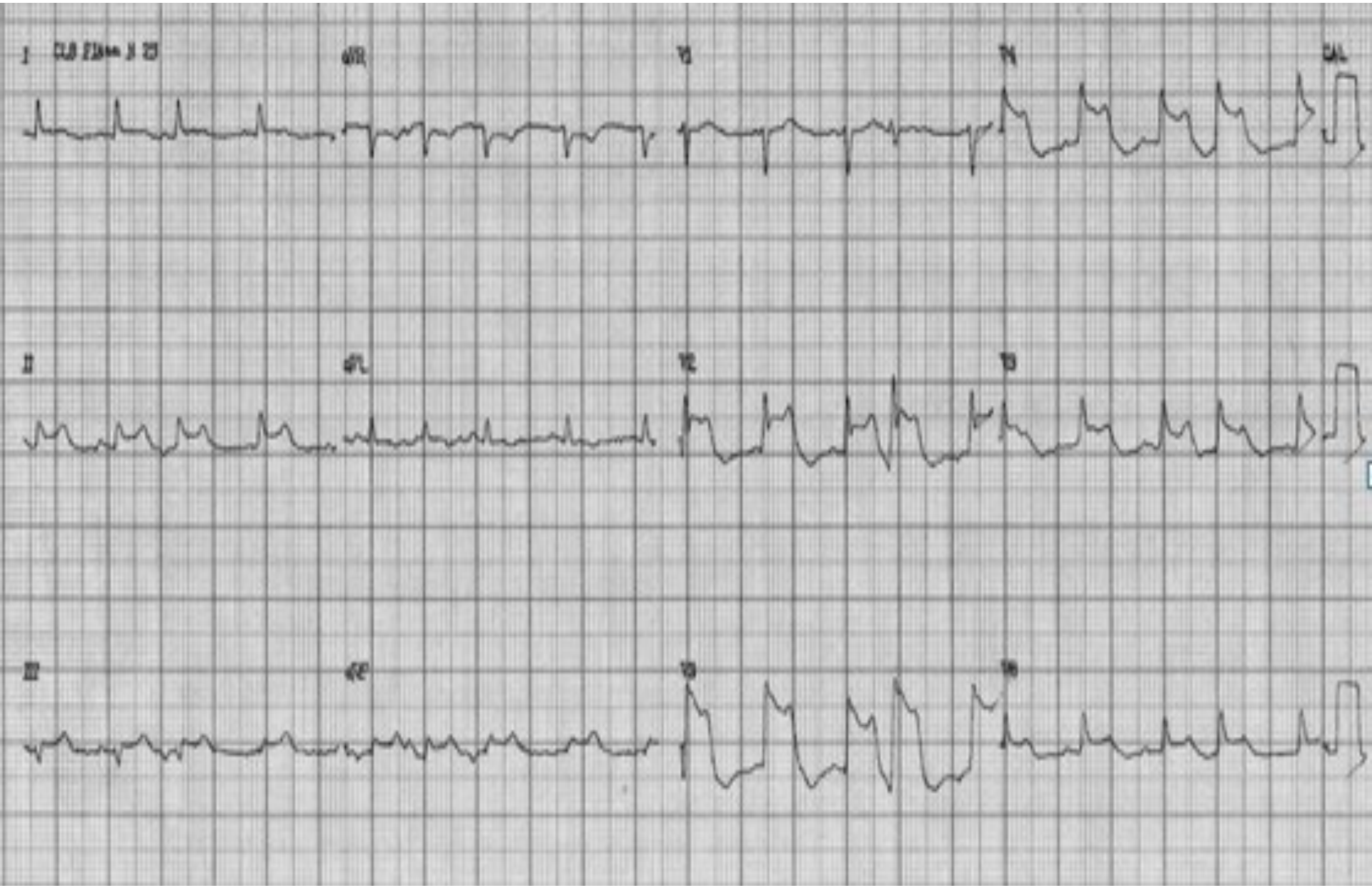


Figure 4. Diffuse concave upwards ST segment elevation with PR segment elevation in lead aVR and premature atrial complexes (during chest pain episode).

Figure 5

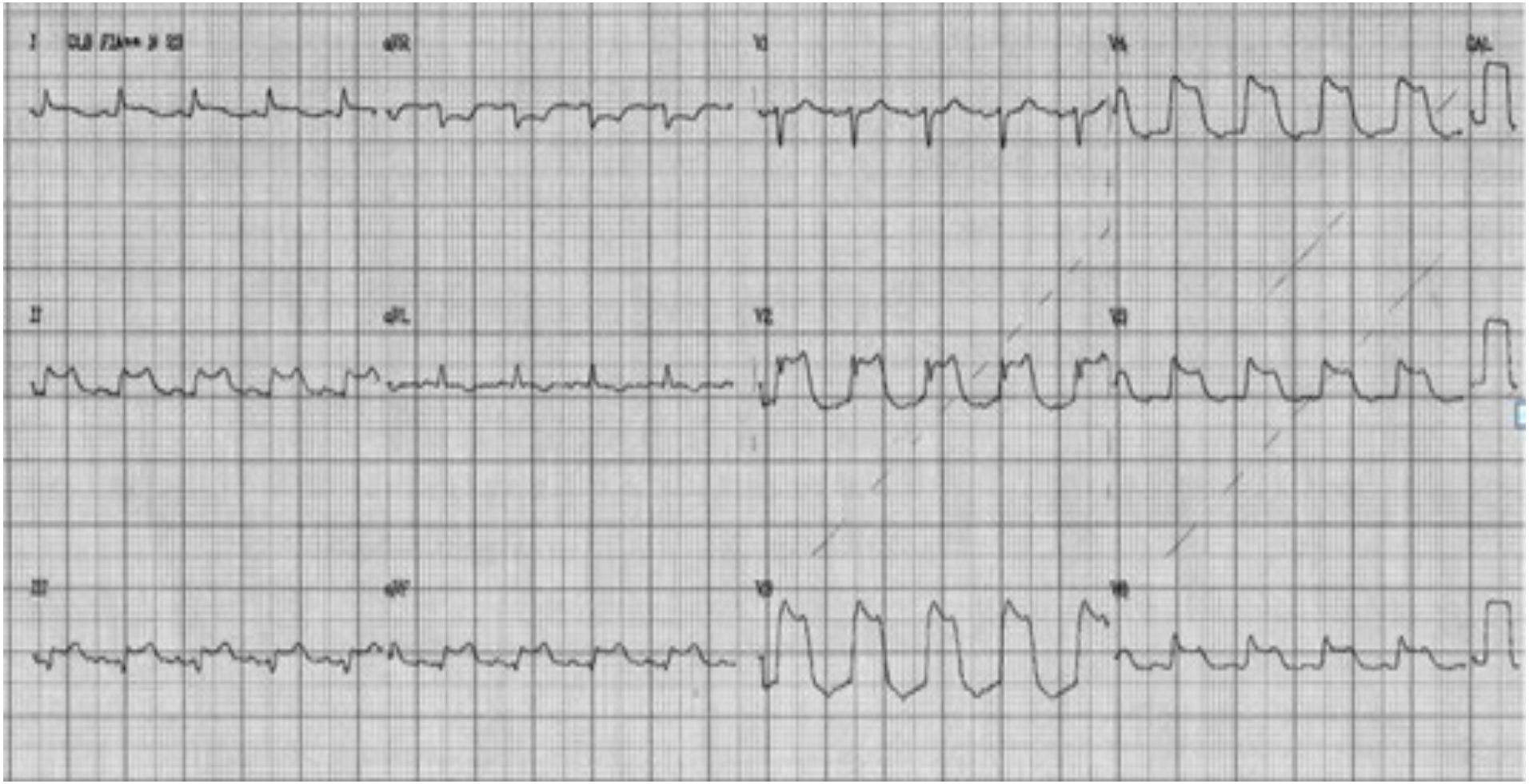


Figure 5. Marked concave upwards ST segment elevation more prominent from V2 through V6, II and III with PR segment elevation in aVR (during chest pain). This pattern in the mid precordial leads resembles an intracellular monophasic action potential (a single polarity) and can be mistakenly diagnosed as an acute STEMI. The combined appearance of ST elevation in both anterior and inferior leads along with PR segment shifts opposite to P polarity may also be suspicious of stage I acute pericarditis **(Bruch 2001; Spodik 2001)**. The typical pattern in acute pericarditis is diffuse ST segment elevation in I, II, aVL, aVF and from V3 to V6. ST segment depression is always seen in lead aVR, frequently in V1, and occasionally in V2 (**Maisch 2004**).

After the cardiac catheterization the patient's precordial pain persisted and radiated into the trapezius muscle region; it improved when sitting up, and it worsened when lying down. On physical examination a triphasic pericardial friction rub was audible along with a tachycardia that was exacerbated when sitting up and leaning forward.

The lab tests were suggestive of acute and chronic renal failure (CRF): **metabolic acidosis**, **hyperkalemia** and **hypocalcemia**.

pH: 7.04; PO2: 126; PCO2: 14; K: 7.0 mEq/L; HCO3: 3.8 mEq/L; Glucose: 134 mg/dL; BUN: 475 mg/dL; Cr: 18.9 mg/dL; Ca: 0.4 mg/dL; Hgb: 6.4 mg/dL; Tp: 0.13 g/dL; CK-MB: 14.1.

ECG changes in chronic renal failure reflecting both hyperkalemia and hypocalcemia include prolonged ST segments often with QT prolongation (hypocalcemia) as well as peaked, symmetrical T waves (hyperkalemia). **Figure 6** illustrates these changes in another case.

Figure 6

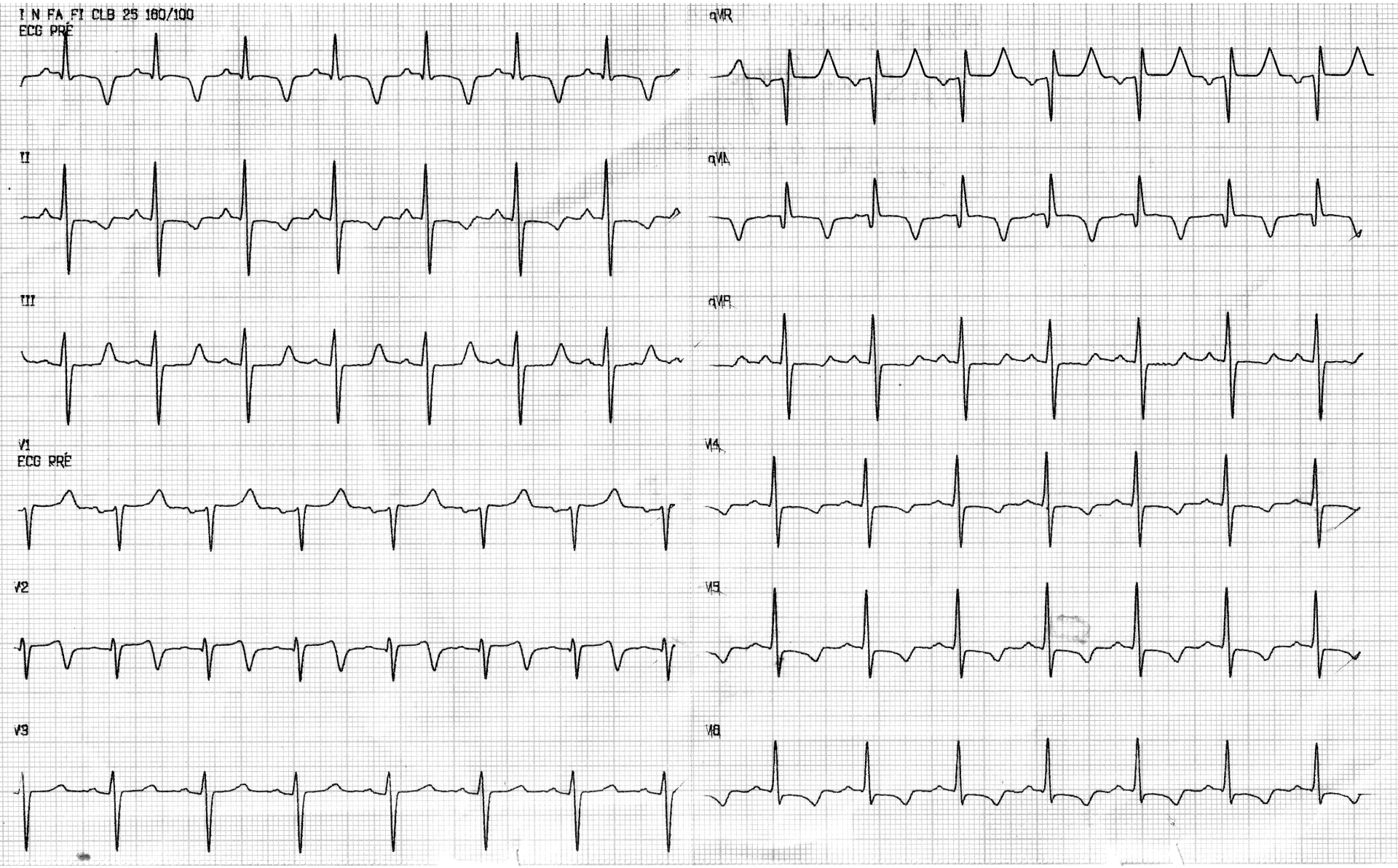


Figure 6. Clinical diagnosis: 47 yr old man with polycystic kidney disease and chronic renal failure (Creatinine 7.0 mg/dL) treated with dialysis for hyperkalemia and hypocalcemia.
ECG diagnosis: Prolonged ST segments, peaked, symmetrical T waves, and QT prolongation.

Figure 7 shows lead V2 in a patient with hypocalcemia and hyperkalemia due to CRF.

Figure 7

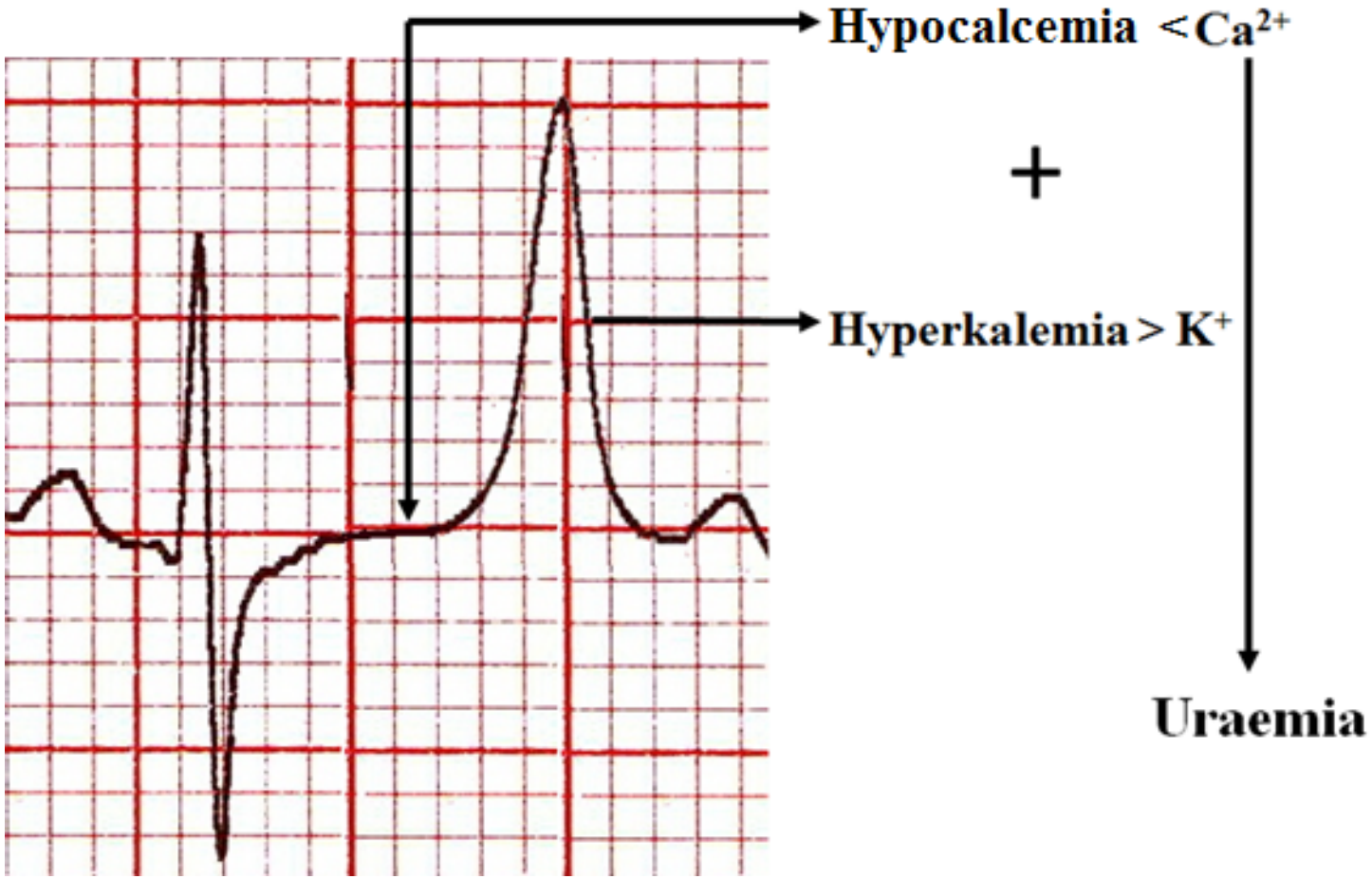


Figure 7. The lead shows typical prolonged ST segment (hypocalcemia) associated with a tall, symmetrical T wave with a narrow base (hyperkalemia).

The patient (MAR) underwent hemodialysis with progressive improvement. After 1 week there was regression of ST segment elevation and new diffuse T wave inversion indicating Stage III ECG evolution of pericarditis (**Figure 8**). At this time the pericardial rub had disappeared.

Figure 8

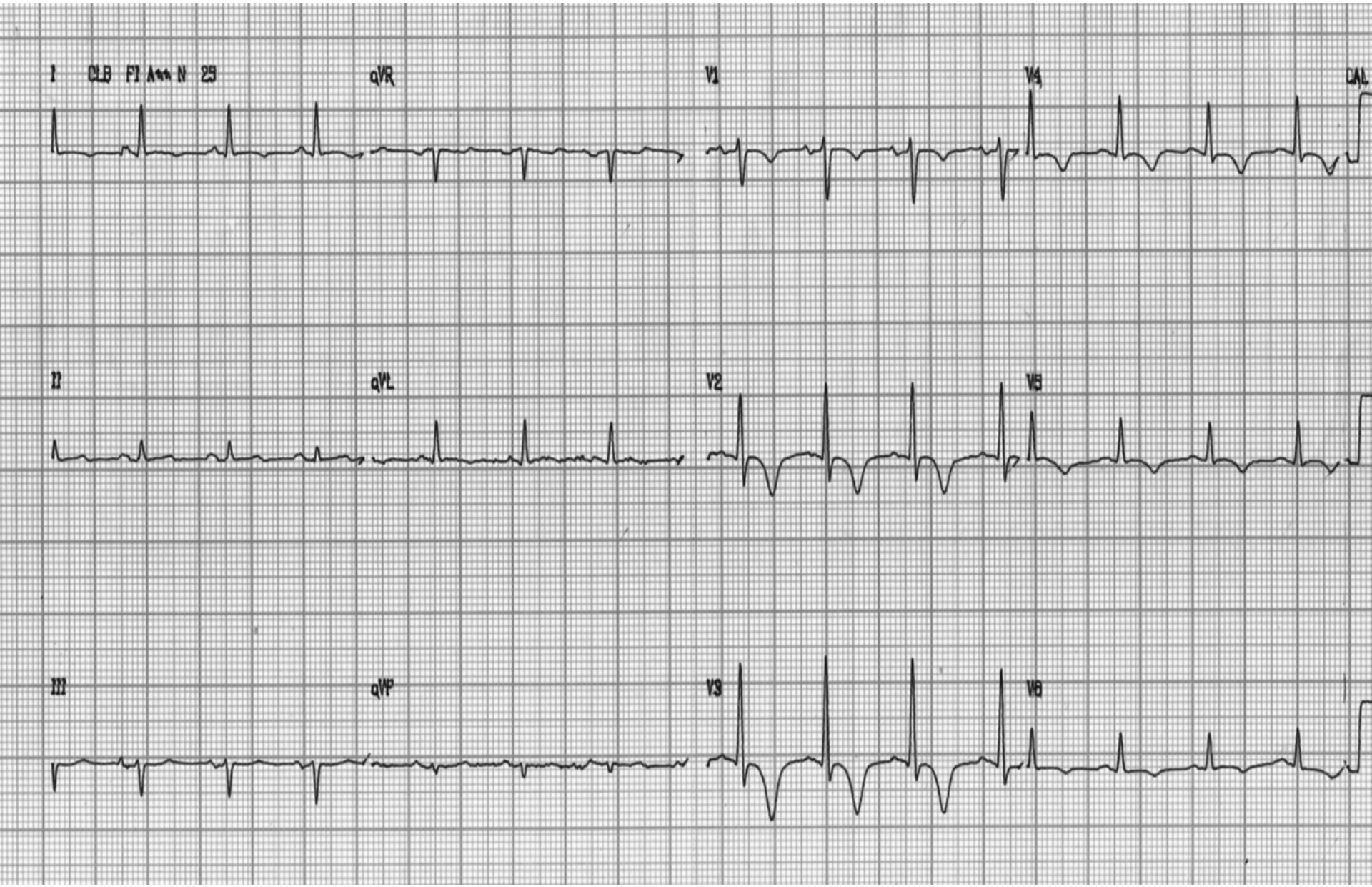


Figure 8. There is resolution of ST segment changes and widespread inversion of T waves indicating Stage III pattern of acute pericarditis.

Introduction

Acute pericarditis is an inflammatory disease of the pericardium with multiple causes. It is the most common form of pericardial disease presenting to the emergency department, and it is often mistakenly diagnosed as an ACS (as in the present case). The real incidence is not known; however in individuals who are not immunosuppressed 90% of cases have a viral or idiopathic etiology (**Lange 2004; Zayas 1995**). Acute pericarditis is found in approximately 5% of the patients admitted in the ER for chest pain not due to acute myocardial infarction. It occurs more frequently in men between age 20 and 50 years. Potential etiologies of acute pericarditis include infection, acute myocardial infarction, medications, chest wall trauma, and systemic diseases such as rheumatoid arthritis and chronic renal failure. However, most often diagnostic evaluations are inconclusive.

Possible etiologies of pericarditis.

I. Infectious:

- Viral: infectious being most frequent.
- Bacterial including tuberculosis.
- Fungal.
- Parasitic.

II. Autoimmune and collagen vascular diseases:

- Lupus (systemic lupus erythematosus)
- Scleroderma
- Dermatomyositis
- Rheumatoid arthritis
- Polyarteritis nodosa
- Ankylosing spondylitis
- Reiter's syndrome

- Familial mediterranean fever.

III. Autoimmune processes:

- Acute polyarticular arthritis or rheumatic fever.
- Post-cardiotomy syndrome.
- Post-myocardial infarction syndrome.
- Chronic autoimmune pericarditis.

IV. Processes in organs adjacent to the pericardium:

- After acute myocardial infarction
- Myocarditis
- Aortic aneurysm
- Pulmonary infarction
- Pneumonia
- Esophageal diseases
- Hydro-pericardium in CHF
- Paraneoplastic

V. Metabolic:

- Uremia (as in the present case) is a frequent cause. It is observed in 8% of patients with chronic or acute renal failure. It relates to the degree of azotemia with BUN levels usually >60 mg/dL. (The reference value for those older than 60 years is 8-23 mg/dL). Pericarditis is often related to inadequate dialysis or fluid overload.
- Myxedema
- Addison's disease
- Diabetic ketoacidosis
- Severe hyperlipidemia.

VI. Gestational:

- Pregnant women (very rare).

VII. Trauma:

- *Direct injuries of the pericardium:* direct penetrating injuries, esophageal perforations, foreign bodies.
- *Indirect injuries of the pericardium:* irradiation, non-penetrating injuries.

VIII.Neoplastic:

Primary Tumors: rare.

Secondary tumors: metastasis of breast, lungs, stomach, colon cancer, sarcoma, lymphoma, leukemia, etc.

IX. Idiopathic: Up to 50% of the cases in some series.

Diagnosis

The diagnosis is based on a history of pleuritic chest pain and the presence of a pericardial rub (present in 85% of the cases (**Snyder 2014**)). Typical ECG changes are characterized by diffuse and widespread concave upwards ST segment elevation often mistakenly confused with STEMI (**Lange 2004; Imazio 2004; Bonnefoy 2000; Salisbury 2009**). At least 2 of 4 criteria are necessary for diagnosis: pleuritic chest pain, pericardial friction rub, diffuse concave upwards ST segment elevation, and pericardial effusion (**Imazio 2010**).

Fever is present in approximately 46% of the cases, and 40% have a recent history of respiratory infection. Classically, the pleuritic chest pain is relieved by leaning forward in a sitting position and worsened in a supine position; it often radiates to the inter-scapular region because the trapezius muscle and the pericardium have the same sensory innervation (**Spodick 2003**). Pain may be minimal or absent in patients with pericarditis due to uremia, neoplasm, tuberculosis, or radiation therapy.

Physical examination

A pericardial rub is virtually 100% specific for the diagnosis; however, sensitivity is variable (16-85%) depending on the frequency of auscultation and etiology (**Lange 2004; Bonnefoy 2000**). The rub is best heard at end-expiration with the diaphragm of the stethoscope placed in the low left sternal border while the patient is sitting forward (**Figure 9)**. Erb's point is the ideal location to hear the rub.

Figure 9

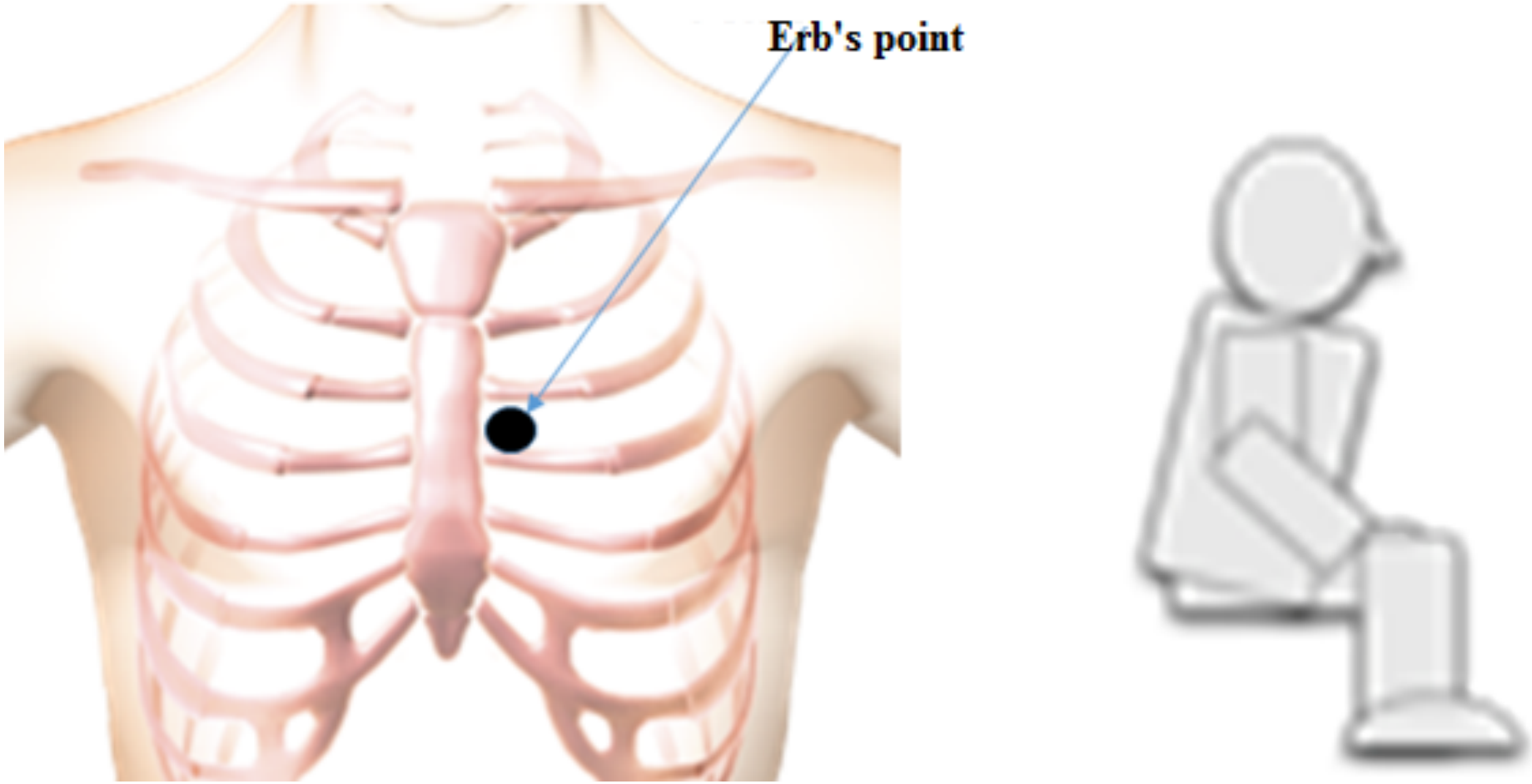


Figure 9. Location of Erb's point and the proper position for pericardial rub auscultation.
The pericardial friction rub has three components that correlate with the phases of the cardiac cycle: a systolic component, an early diastolic and a late diastolic component (triphasic). Often not all three components are audible in a given patient at any particular time.

In patients with uremic pericarditis with autonomous nervous system impairment the heart rate may remain non-tachycardic even in the presence of tamponade, fever, hypotension and anemia (**Tarng 1997**).

Laboratory data

Acute phase inflammatory markers including sedimentation rate, leukocytosis and C-reactive protein (CPR) are elevated in approximately 75% of the patients. Increase in cardiac troponins (TnI) is indicative of myocardial involvement or myopericarditis (**Imazio 2003; Sagristà Sauleda 2005**). Other tests to confirm the etiology should include thyroid function, rheumatologic tests, renal function, and blood cultures when suspicious of bacterial infection. In the presence of a large pericardial effusion, histological analysis and immunohistochemistry of the pericardial fluid should be done to rule out TB (adenosine deaminase) or neoplasia (**Sagristà Sauleda 2005**). In patients that do not respond to therapy after a week, other etiologies should be pursued to rule out autoimmune disease and HIV.

Electrocardiography

The electrocardiographic alterations in pericarditis involve PR segments, ST segments and rhythm according to the time course of pericarditis. The ECG can be normal in approximately 6% of cases. In acute pericarditis the ECG changes evolve over four serial stages (**Spodic 1973; Bruce 1980**).

Stage I: concave upwards ST segment elevation in multiple leads except aVR and V1 where ST depression may occur; symmetrical and peaked T waves with narrow base and mild increase in voltage; and PR segment depression (except in aVR, where there is PR segment elevation). These alterations are observed in more than 80% of the cases (**Bruce 1980; Baljepally 1998**). (**Figures 10, 11, 12**).

Figure 10

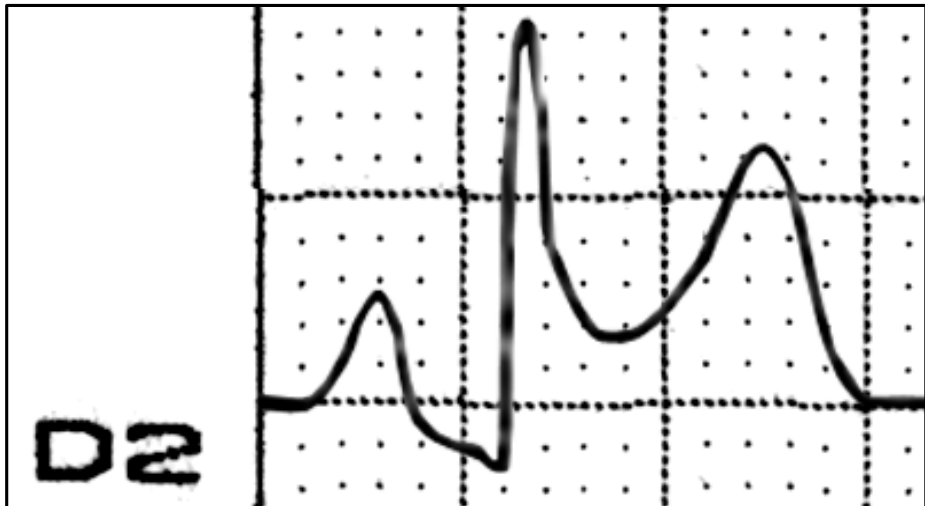


Figure 10. ST segment elevation.

Figure 11

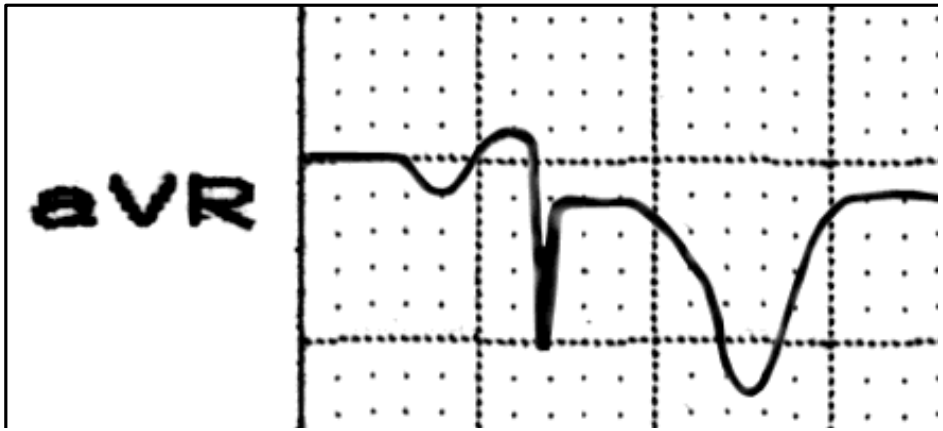


Figure 11. Reciprocal alterations in aVR.

Figure 12

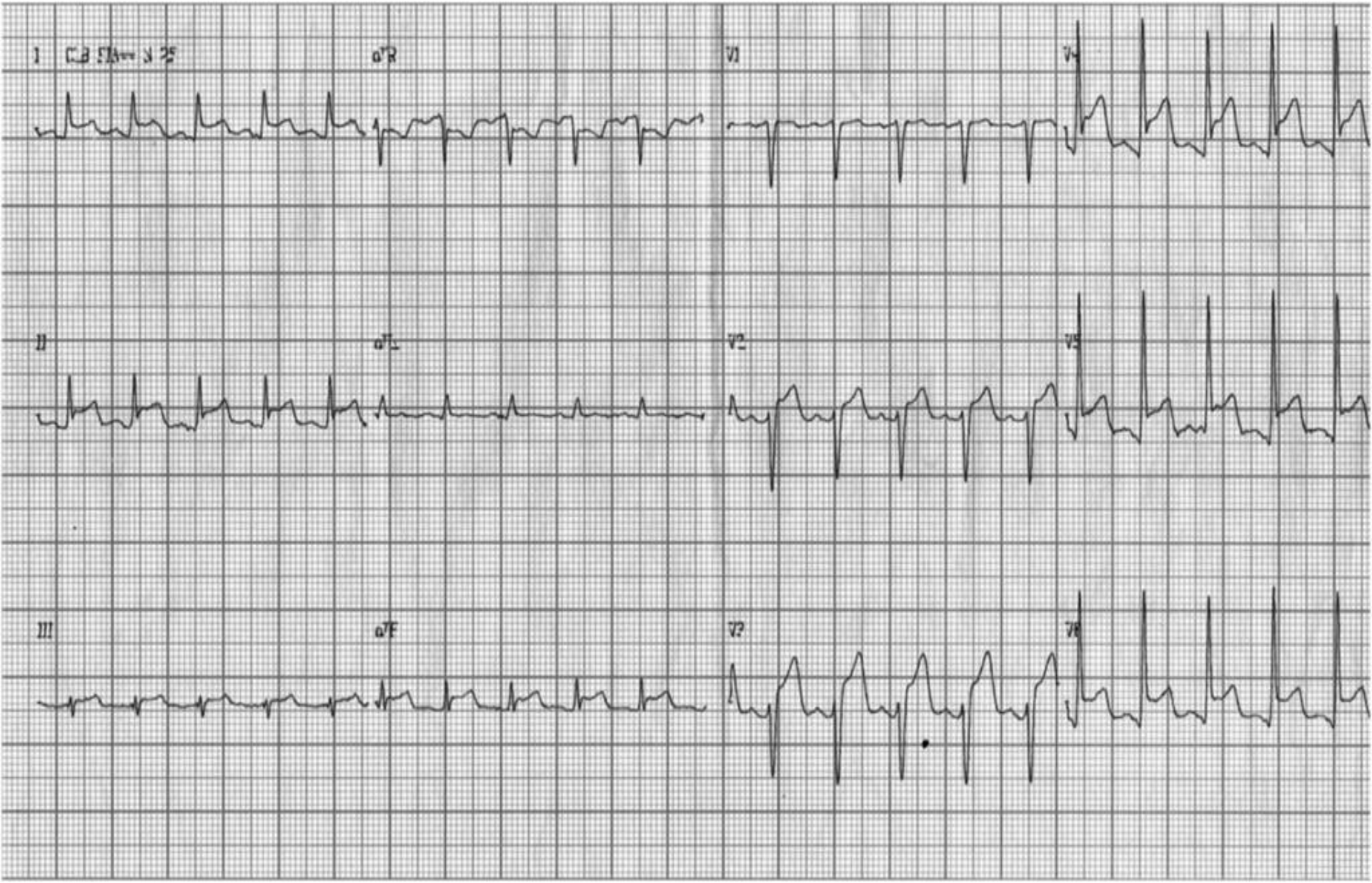


Figure 12. Stage 1 of acute pericarditis. Diffuse concave upwards ST segment elevation and PR segment elevation is shown in aVR.

Another useful ECG finding in Stage I acute pericarditis is **Spodik's sign** (**Chaubey 2014**), the slightly downward sloping TP segment best seen in lead II as illustrated by the black arrow in **Figure 13**.

Figure 13

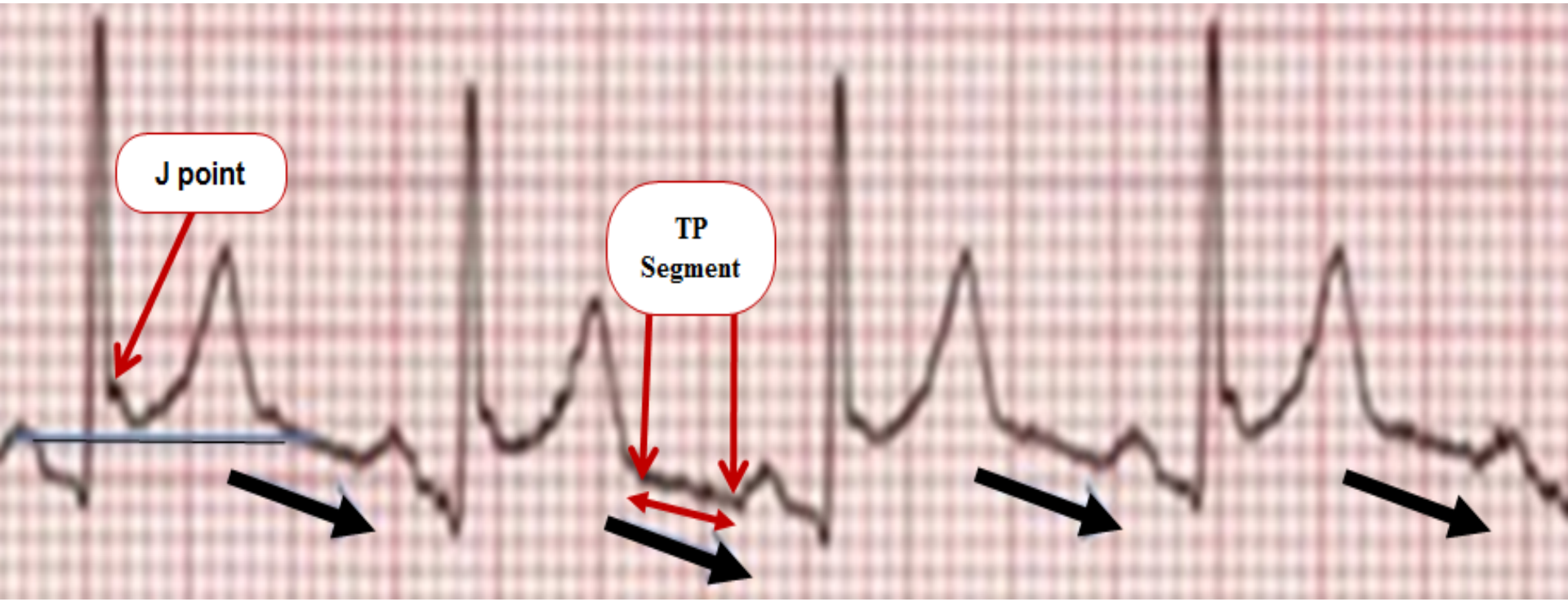


Figure 13: Lead II shows Spodik's sign characterized by downward TP segment, indicated by the black arrows.

Table 2 modified from (**Yahalom 2013**) lists the differential diagnosis of ST segment elevation.

Table 2 - Main causes of ST segment elevation
➢ Acute Myocardial infarction
➢ Acute myocarditis
➢ Acute pericarditis
➢ Takotsubo cardiomyopathy or stress-induced (apical ballooning) cardiomyopathy
➢ Left ventricular hypertrophy
➢ Left ventricular aneurism
➢ Hypertrophic Cardiomyopathy
➢ Acute aortic dissection
➢ Pulmonary embolism
➢ Arrhythmogenic Right Ventricular Cardiomyopathy/Dysplasia
➢ Left Bundle Branch Block
➢ **Hyperkalemia**
➢ Post electrical cardioversion
➢ Ventricular paced rhythm
➢ Vasospastic or Prinzmetal´s angina
➢ Brugada Syndrome
➢ Benign Early repolarization pattern
➢ Inferolateral Early Repolarization (ER)
➢ Idiopathic ventricular fibrillation
➢ Congenital Short QT syndrome
➢ Hypothermic J-wave
➢ Hypercalcemia
➢ Acute cerebral hemorrhage. J-wave in acute nervous system injuries; i.e. subarachnoid hemorrhage. cardiac arrest, etc.
➢ Dysfunction of the cervical sympathetic system

Stage II: Reversion of ST and PR segments shifts along with T wave flattening (after some days).

Stage III: Diffuse T wave inversion resembling myocardial ischemia. Absence of Q waves indicative of myocardial necrosis is a useful clue to differentiate this stage of pericarditis from the diagnosis of myocardial infarction.

Stage IV: T wave abnormalities resolve to more normal morphology; this may take weeks to months after the onset of acute pericarditis.

Other ECG alterations: Alterations in rhythm may occur during any stage and include sinus tachycardia and various atrial arrhythmias (**Spodick 1976**). In the presence of pericardial effusion the most typical finding is low QRS voltage (**Jung 2010**) which improves after pericardiocentesis. In the setting of pericardial tamponade electrical alternans of QRS voltage may be seen. Low QRS voltage is defined as complexes of less than 5 mm (0.5 mV) in the frontal plane leads and less than 10 mm in the precordial leads. QRS alternans is the beat by beat variation in axis, amplitude and/or morphology. Low voltage may also be present in other diseases including emphysema, infiltrative myocardial disease, and pneumothorax. After pericardial drainage the QRS voltage does not normalize immediately, suggesting a continued inflammatory process. Electrical alternans is the result of the heart moving around in a large effusion. This pattern is more visible in the precordial leads. Isolated QRS alternans is not specific to pericardial effusion, but the presence of *both* P and QRS alternans is pathognomonic.

The most frequent type of alternans involves only the QRS. When P, QRS, and T waves are included it is called total alternans and is observed in case of cardiac tamponade. Beck's triad (jugular vein distention, hypotension and muffled sounds) is a feature of cardiac tamponade. **Figure 14** illustrates electrical alternans of the QRS.

Figure 14

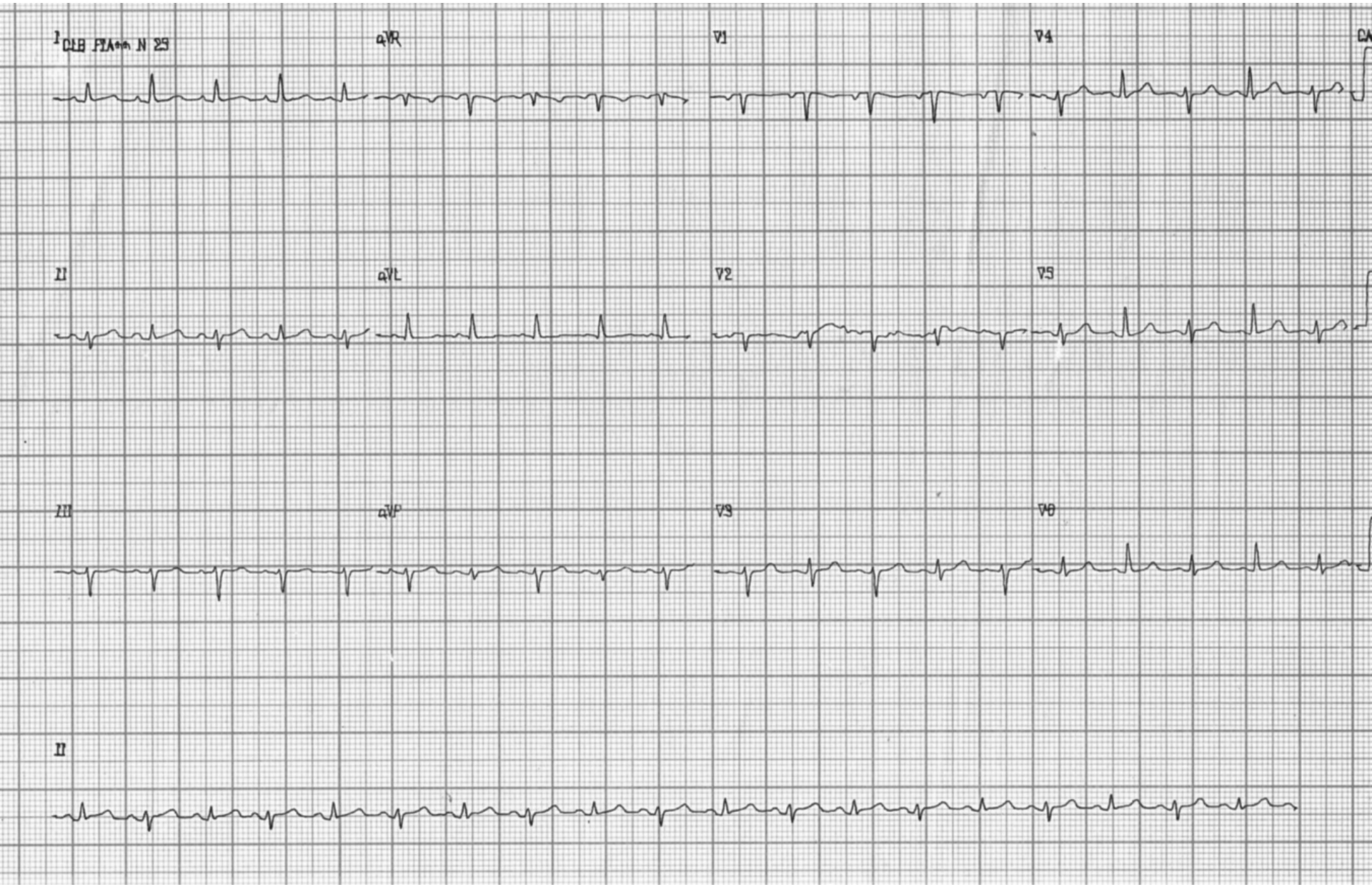


Figure 14. Patient with TB pericarditis and cardiac tamponade. ECG shows low, diffuse voltage and QRS electrical alternans.

Differential diagnosis of pericarditis

Tables 3 and 4 indicate ECG features that differentiate acute pericarditis from early repolarization syndrome and acute myocardial infarction.

Table 3

	Early repolarization syndrome	Acute pericarditis in early stage/stage I
$^{ST}/_{T}$ ratio in V_6	<0.25	Variable.
T wave	Always wide, positive and persistent. Greater voltage.	It decreases its width in hours. Less voltage. Only increased in the early stage.
ST	Significant elevation only in precordial leads. Reciprocal depression only in aVR.	Diffuse elevation in multiple leads. There may be reciprocal depression in aVR.
Response to exercise	Frequent return of ST to the baseline. T wave may normalize.	No change in ST segment elevation.
Hyperventilation	The polarity of T may be modified.	It does not modify the polarity of T.
Presentation	Stable.	Transient.
HR	Frequent bradycardia.	Frequent tachycardia.
Clinical	Asymptomatic.	Symptomatic.
Age range	20 to 40 years.	Predominance 40 or more years.

The electrocardiographic features of early repolarization that help in the differentiation with acute pericarditis in stage I include:

- ✓ Slow heart rate: bradycardia.
- ✓ QRS electrical axis, ST segment and T wave pointing in the same direction in the frontal plane.
- ✓ Frequent deep and narrow Q waves followed by R waves with great voltage in the left precordial leads.
- ✓ Notch or slurring in the downward slope of R.
- ✓ Sudden change in precordial QRS transition.
- ✓ J point and ST segment elevation; usually J <2 mm (it may exceptionally be >5 mm) with upper concavity in intermediate and left leads, and sometimes in inferior leads, followed by wide and positive T wave;
- ✓ Before sympathetic stimulus and sympathomimetic drugs, J point and ST segment elevation is observed.
- ✓ Absence of reciprocal or mirror image, except in aVR.
- ✓ Pseudo-symmetrical T waves with great width and polarity matching their corresponding QRS complexes.

Table 4

	STE-ACS /STEMI	Acute pericarditis
Number of involved leads	Segmentary and located	Greater (diffuse) and extensive
Intensity of phenomena	Greater	Lower
Reciprocal or mirror effect	When present, it occurs in several leads	Only in aVR (**Chenniappan 2013**).
QRS complex prolongation	Yes	No
QT interval shortening in leads with ST segment elevation.	Yes	No
QT dispersion (Rossello 2014).	Greater	Lower

Chest X-rays

A chest X-ray is mandatory in all patients with suspicion of pericarditis. The presence of cardiomegaly occurs only when there is more than 200 ml of fluid in the pericardial sac with the cardiac outline acquiring a globular shape (**Figure 15**, a patient with purulent pericarditis). The chest X-ray may also be of great help in identifying the etiology by showing concomitant pulmonary infection or masses in the mediastinum. This tool has a moderate sensitivity (70%) but low specificity (41%) for the diagnosis of pericardial effusion (**Eisenberg 1993**). The presence of pericardial calcification (**Figure 16**) strongly suggests constrictive pericarditis, although it is only present in 25% of these patients (**Ling 2000**).

Figure 15

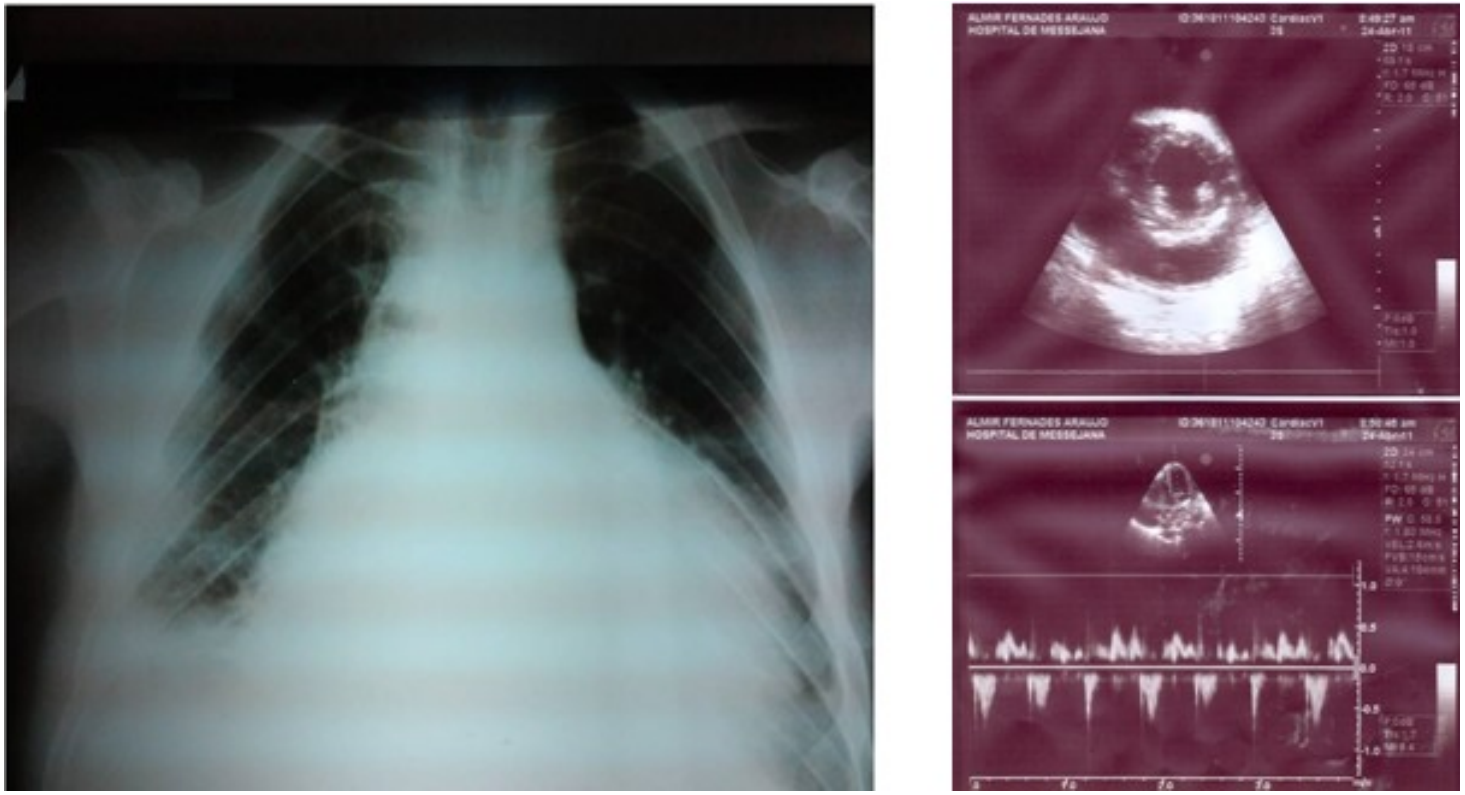


Figure 15: X-rays shows globular increase of the cardiac area with pleural effusion at the right. Echo showing pericardial effusion.

Figure 16

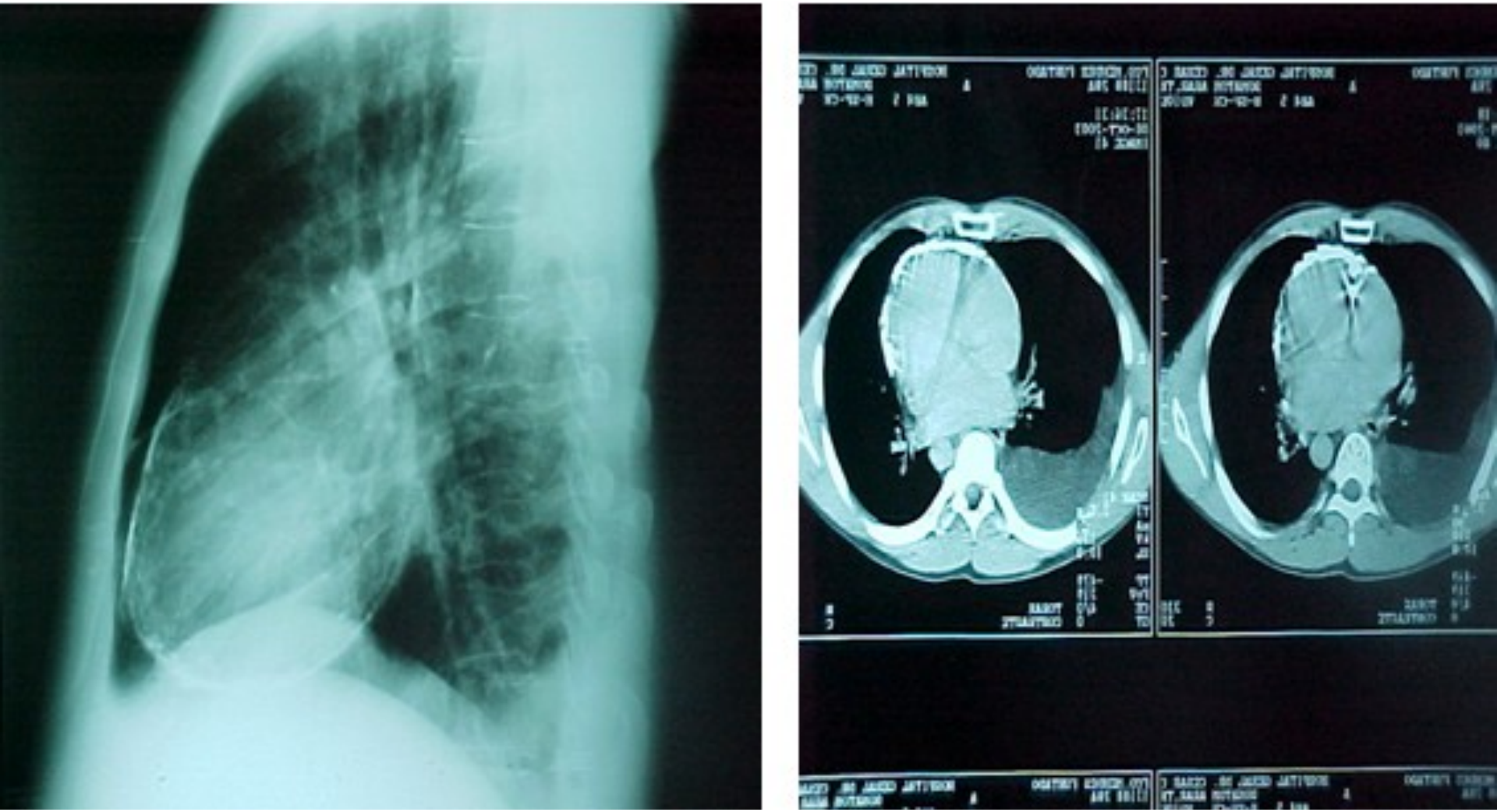


Figure 16. Typical pericardial calcification in the chest X-rays, confirmed by CT.

Echocardiogram

Cardiac ultrasound should be performed in all cases of suspected pericarditis to assess the presence of pericardial effusion (**Maisch 2004**). The echocardiogram may offer clues to the etiology as it allows characterizing the nature of the liquid (transudate or exudate) and verifying if there is fibrin (as in TB) or calcium (**Figure 15**). The collapse of the RA is the most sensitive sign of tamponade; however collapse of the RV is more specific. Doppler may also indicate the echocardiographic expression of a paradoxical pulse.

CT and NMR

Computerized tomography and nuclear magnetic resonance are two advanced imaging modalities that more accurately evaluate the pericardium. They are useful jointly with the echo to better characterize effusion and tamponade. They should not be done in critically ill patients who require immediate treatment. They are especially useful in detecting loculated effusions or when associated with pleural effusions. Pericardial thickening may be evaluated by both methods enabling assessment of chronicity and severity (**Figure 16**). Density of fluid similar to water suggests transudate; greater than water suggests malignancy, blood or pus (**Verhaert 2010**).

Analysis of pericardial fluid and biopsy

In cases of refractory pericarditis with effusion analysis of the pericardial fluid should be done in order to determine the etiology, although the definitive cause is only obtained in 20% of cases.

Treatment

Most cases of acute pericarditis have a viral etiology and do not need to be hospitalized. They respond very well to therapy with non-steroidal anti-inflammatory drugs (NSAIDs). Complicated patients should be admitted for diagnosis, treatment and for the detection of cardiac tamponade, the most feared complication (**Imazio 2007**). High risk indicators include increase in positive troponins, fever above 38°C with leukocytosis, voluminous pericardial effusions with or without cardiac tamponade, immunosuppressed patients, patients on oral anticoagulation, myocardial dysfunction by echo suggesting myopericarditis. These markers indicate the need of hospital admission for etiological evaluation and therapeutic interventions.

NSAIDs should be used in anti-inflammatory doses: acetylsalicylic acid (ASA), 500 to 750 mg every 6 or 8 hours for 7 to 10 days, followed by gradual reduction of 500 mg per week, for three weeks; ibuprofen, 400 to 800 mg every 6 or 8 hours, for 14 days; indomethacin is sometimes used, because it may reduce scar formation. The duration of the treatment with NSAIDs is usually around 14 days and guided by serum levels of C-reactive protein.

Colchicine has been shown in controlled clinical trials to be more effective than NSAIDs in relieving symptoms and preventing recurrence (**Imazio 2010; Imazio 2005; Adler 1998; Imazio 2005**). Colchicine is generally well tolerated, with a low incidence of adverse side effects (**Norrid 2014**). A multicenter, double-blind, placebo-controlled and randomized study concluded that colchicine, added to conventional anti-inflammatory treatment, significantly reduced the rate of subsequent recurrences of pericarditis in patients with multiple recurrences. Taken as a whole with the results of other studies, colchicine should be considered a first-line treatment for acute or recurrent pericarditis in the absence of contraindications or specific indications (**Imazio 2014**). The dose of colchicine is 0.5 mg BID or 0.5 mg once daily in patients <70 kg for three months during the first event or 6 months in recurrent pericarditis.

The routine use of corticosteroids should be avoided, since it is associated with an increase in the rate of recurrence. They may be considered indicated in the following situations:

- Pericarditis refractory to NSAIDs and colchicine.
- In cases in which the above mentioned drugs are contraindicated.
- Autoimmune etiology or connective tissue diseases.

Prednisone, when used, is dosed at 1 mg/Kg/day for at least 1 month before tapering; there is evidence that a lower dose of 0.5 mg/Kg/day has the same efficacy with fewer side effects or recurrences (**Imazio 2008**). To prevent recurrence or reactivation of acute pericarditis, slow weaning and addition of colchicine (1 mg per day) should be done.

Bacterial pericarditis requires pericardial drainage and appropriate antibiotic therapy. TB pericarditis requires a multidrug management approach. If there is a neoplastic etiology, appropriate chemotherapy is required, although there is a high rate of recurrence. The patients with uremic pericarditis require intensive hemodialysis.

Pericardiocentesis and pericardiectomy

Pericardial drainage is mainly indicated in the cases of cardiac tamponade, purulent pericardial effusion, or large symptomatic effusions (**Sagristà Sauleda 2005**). Pericardial drainage is made either by percutaneous technique with placement of a drainage catheter or by open surgical drainage (pericardial window). It may also be done using video-assisted pericardioscopy. Echo guided percutaneous pericardiocentesis decreases complications and increases the likelihood of success (**Uramoto 2010**). Patients with constrictive pericarditis refractory to clinical treatment should undergo pericardiectomy (**Maisch 2004**).

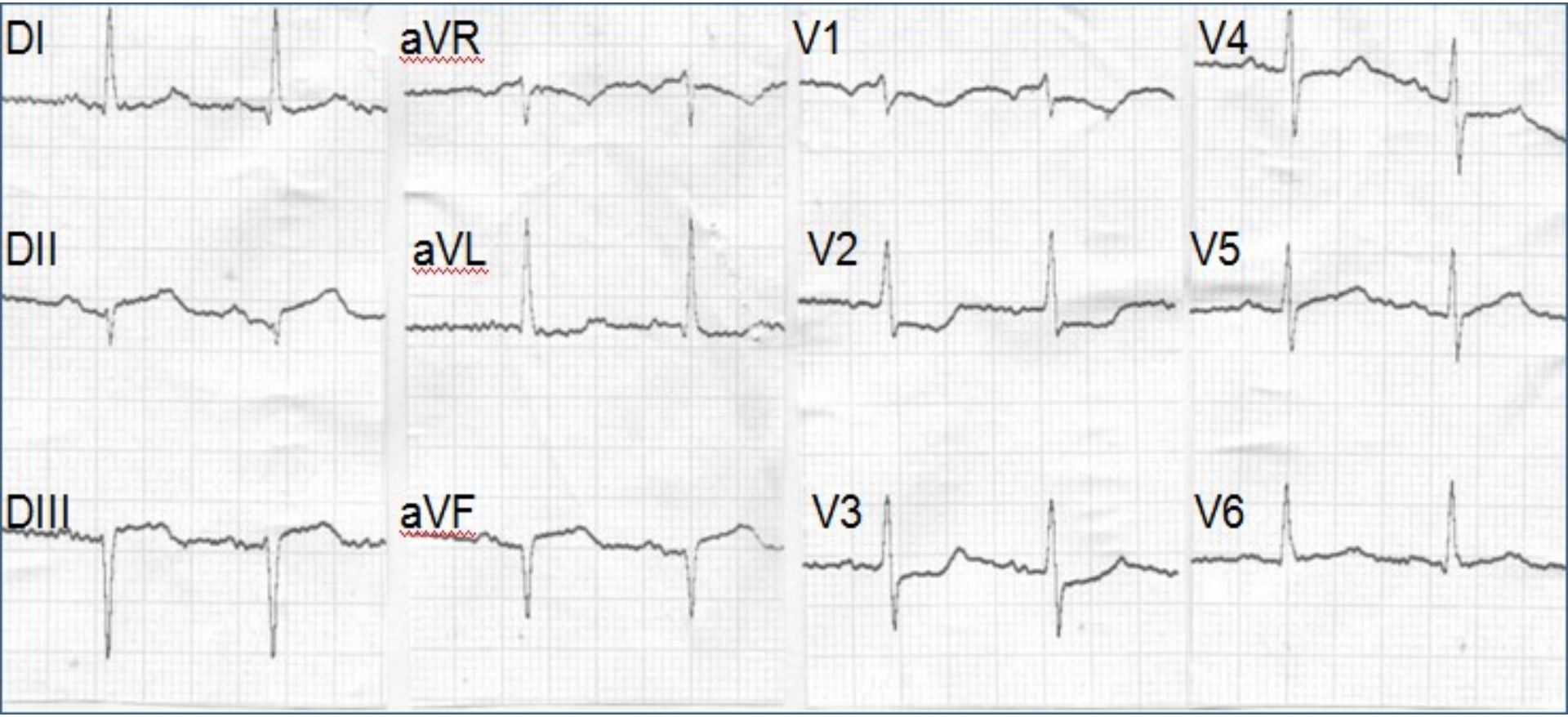
Case report number 3
Acute Myocardial Infarction Due To Acute Total Occlusion Of The Circumflex Coronary Artery Treated as non-ST Segment Elevation Acute Coronary Syndrome (NSTE-ACS)

A seventy six-year-old man presented to the Emergency Department with oppressive retrosternal chest pain radiating down both arms. Risk factors for coronary artery disease (CAD) included systemic hypertension, diabetes mellitus, and dyslipidemia.

Physical examination: Hemodynamically stable. BP = 130/90 mmHg; HR = 88 bpm and regular; clear lungs; S4 heart sound; absence of murmur.

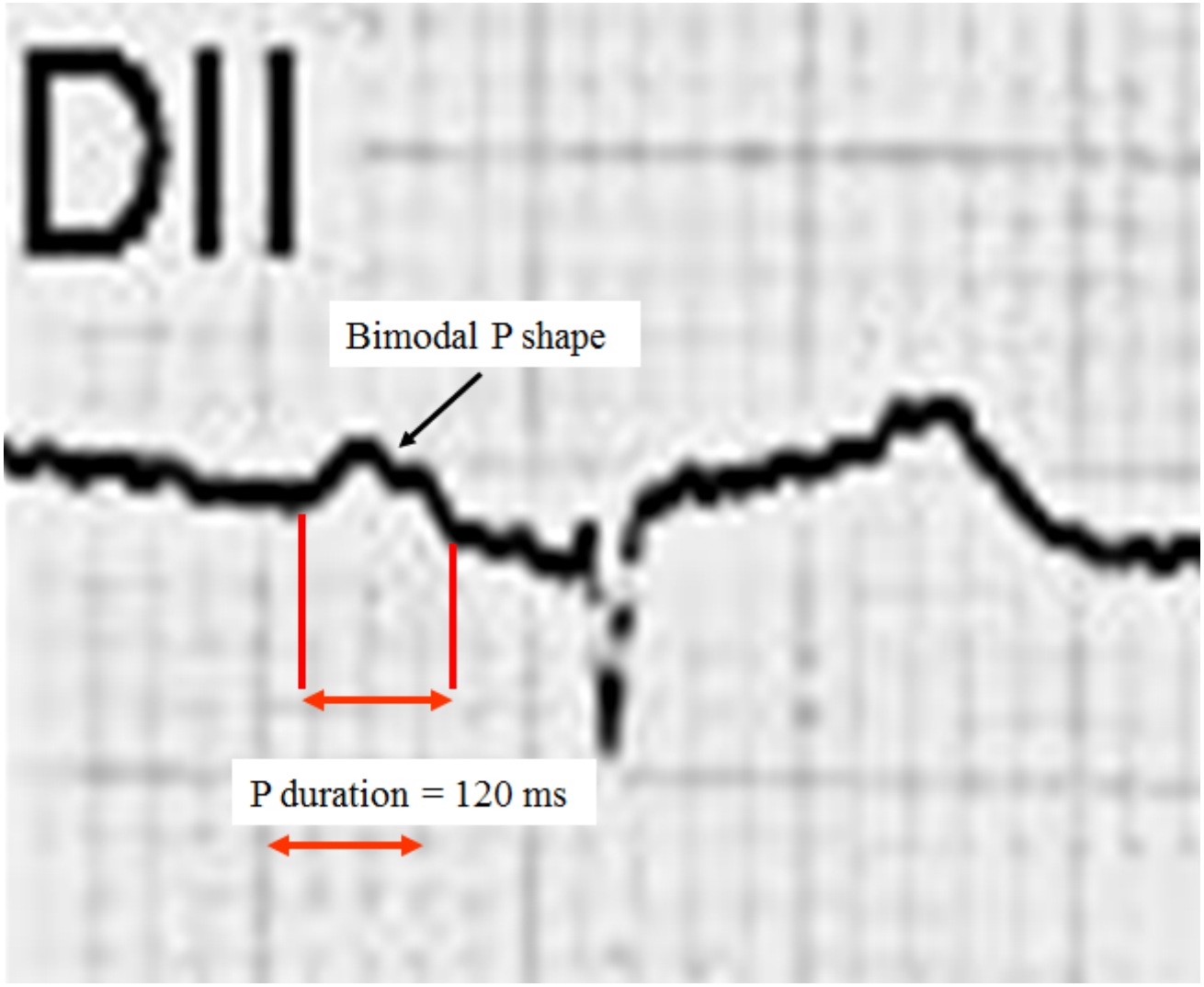
The 12-lead ECG was obtained without recording of posterior accessory leads (**Figure 1**). Even with slight ST segment elevation in the inferior leads, a therapy based on non-ST segment elevation acute coronary syndrome (NSTE-ACS) was provided. After 48 h coronary angiography was performed revealing complete occlusion of the circumflex (LCx) artery (**arrow, Figure 3**). It was decided not to perform percutaneous coronary intervention due to the prolonged time of evolution. The echocardiogram showed akinesis in the infero-lateral LV wall.

Figure 1



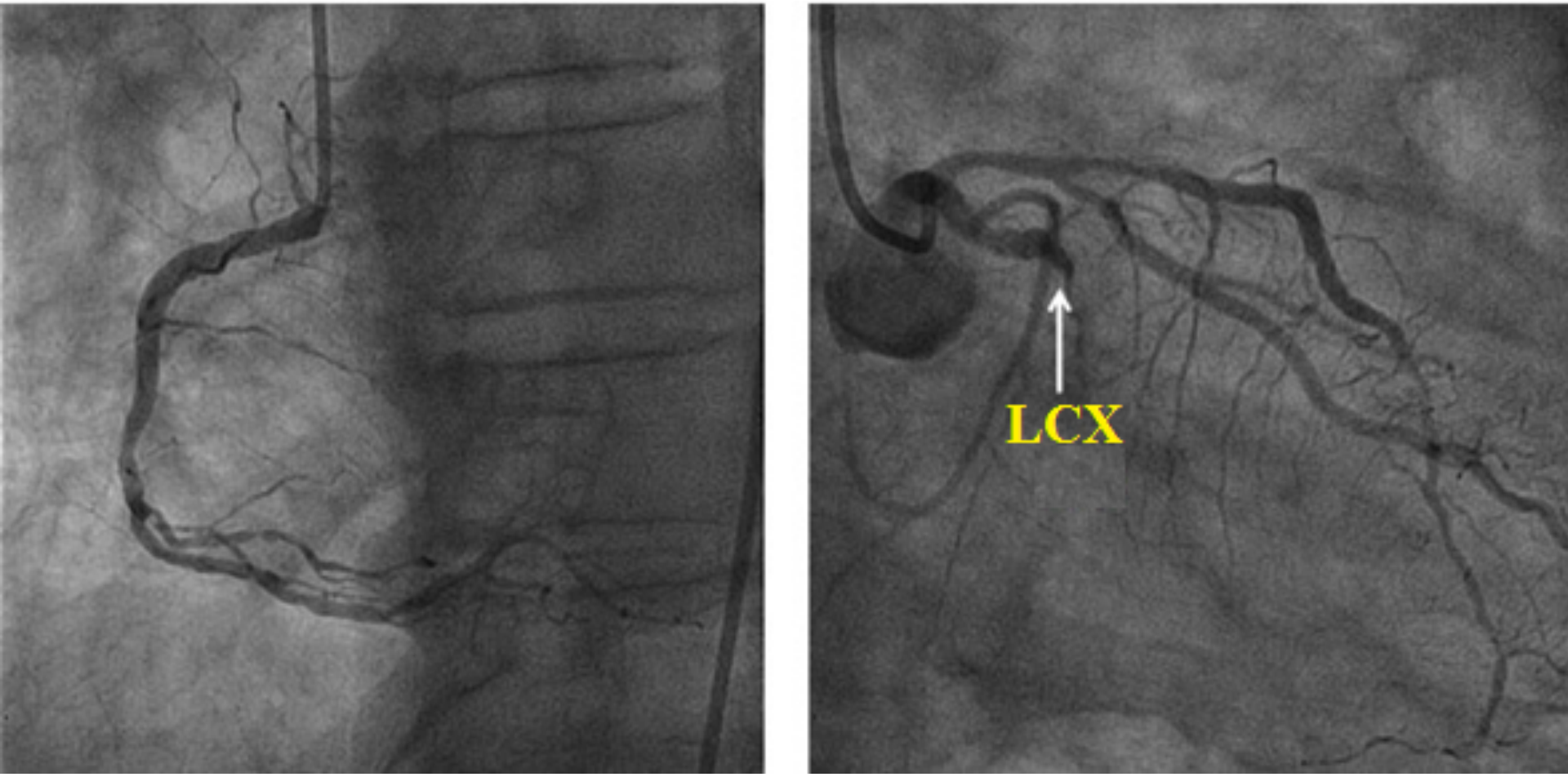
ECG diagnosis: Sinus rhythm, HR 83 bpm. P wave: SAP +60° and posterior; duration 120 ms (prolonged: incomplete interatrial block, LAE, or both?), bimodal P wave in lead II (**Figure 2)** and V3-5; normal PR interval; left axis deviation (SQRS -40°); qR in I and aVL, rS or QS in inferior leads with SIII>SII (LAFB); rs pattern in V1 and Rs in V2 with Q waves in inferior leads and ST depression from V1 to V3; this corresponds to infero-lateral infarction according to the new terminology for Q-wave infarctions based on correlation with cardiac nuclear magnetic resonance (lateral wall: segments 6, 3, 11, 12 and 15 and inferior segments 15, 10 and 4 "bullseye" [bullseye heart magnetic resonance image]) (**Figure 5C**). ST segment depression is present in V1-V3, and ST segment elevation is present in the inferior leads suggestive of an infero-lateral ST segment elevation AMI (STEMI)**.**

Figure 2



The P wave duration (120 ms) is prolonged suggesting incomplete first degree interatrial block and/or left atrial enlargement (LAE). LAE and interatrial block are frequently related. First degree interatrial block is common and is related to an increased incidence of atrial fibrillation with an increase in overall and cardiovascular mortality (**Bayés de Luna 2012**).

Figure 3



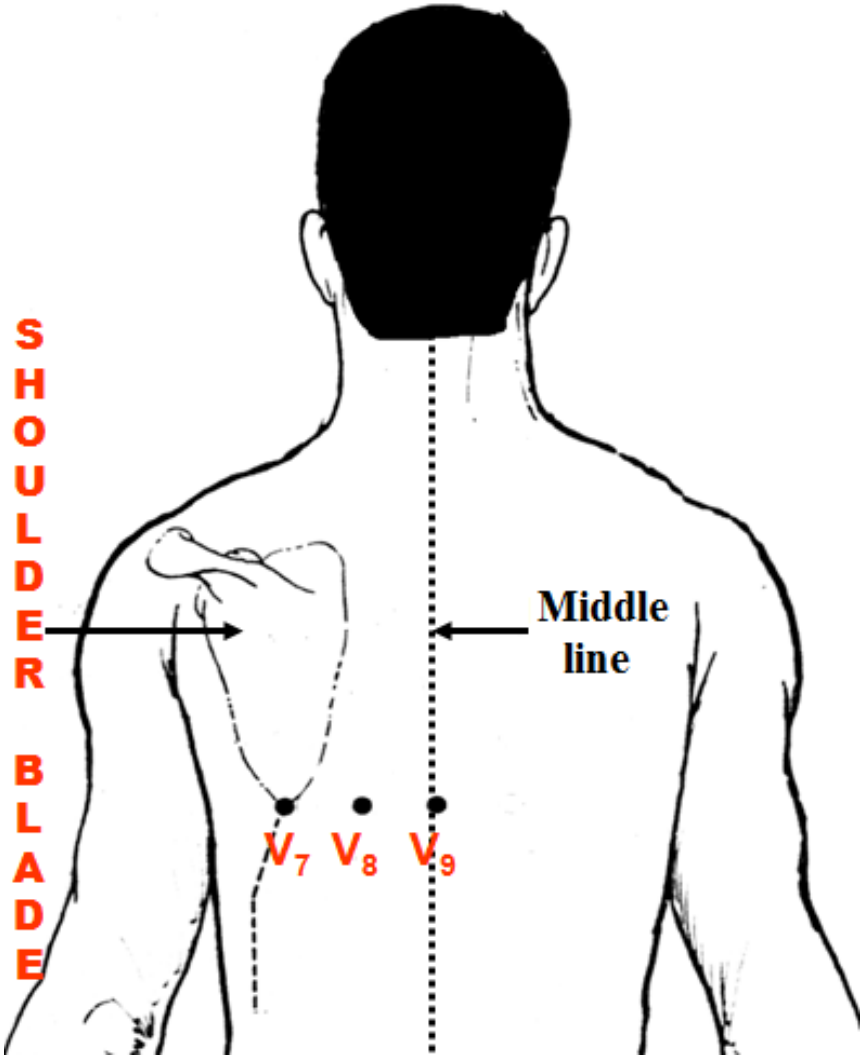
Coronary angiography indicates obstructive lesions in the distal RCA, total occlusion of the LCx artery, and 70% proximal LDA occlusion with distal portion sharpening.

ST segment elevation is the *sine qua non* condition for the diagnosis of transmural AMI resulting from total acute coronary occlusion; however, when the culprit artery is the LCx, ST segment elevation is only observed in approximately 50% of cases (**O'Keefe 1995; Schmitt 2001; Stribling 2011**). The LCx artery originates from the left main coronary artery and follows a trajectory below the left atrium in the AV groove; it gives off marginal branches that perfuse the LV lateral wall and the left posterior ventricular branch perfusing the infero-basal LV wall (formerly called the dorsal or true posterior wall). The terms "posterior" or dorsal, used in the International Society for Holter and Noninvasive Electrocardiography (ISHNE) guidelines, should be discontinued (**Bayes de Luna 2006**). Studies of cardiac NMR-ECG correlation showed that the so-called posterior wall actually corresponds to the basal portion of the inferior wall or segment 4 in the bullseye view (**Figure 5**). Q waves never appear in an infarction in segment 4 (basal-inferior) because activation in this region occurs later during the middle and final portions of the QRS (between the 30 and 100 ms); i.e., in the second half of the QRS. Pathologic Q or QS waves occur during the first 40 ms of the QRS. The infarction of the basal region of the LV causes a late anterior shift of the QRS loop in the horizontal plane because the QRS loop area is located at least 50% anteriorly (in the front of the orthogonal X). In 40% of the cases this anterior shift of the middle-to-final portion of the QRS loop may cause a triphasic QRS complex (rSr', rSR' or rsR') in V3R or V1 resembling IRBBB (pseudo-IRBBB). The septal depolarization vectors, vectors 1 (initial septal vector 10-20 ms) and 2 (vector of the low portion of the septum from 20-40 ms), are not affected; only the anterior shift of vector 3 of the LV and RV free walls and vector 4 of the basal wall are significant.

According to the National Cardiovascular Data Registry (NCDR) data, approximately one-third of patients with AMI due to acute LCx occlusion present as NSTE-ACS (**Sribling 2011**). Current guidelines for patients presenting with ST segment depression in V1-V4 associated with high R waves and positive T waves consider this to be an infero-lateral STEMI equivalent according to the universal definition of AMI (**Steg 2012; Thygesen 2007**). When ST segment depression extends into other leads (V4-V6), the differential diagnosis should include subendocardial ischemia, making the identification of the culprit vessel more difficult. Since conventional 12-lead ECG is incapable of detecting ST segment elevation in the infero-lateral wall (basal-inferior portion) this differential diagnosis becomes more difficult (**Eisenstein 1985; Matetzky 1999**). Some physicians believe that when the magnitude of the ST segment depression in the V4-V6 leads is much greater than in leads V1-V3, the presence of diffuse subendocardial ischemia and not transmural inferior-lateral ischemia is more likely. One way of resolving this differential diagnosis is the use of accessory posterior leads V7-V9 (**McClelland 2003**). These leads enable the identification of up to 20% of the patients with infero-lateral AMI that could benefit with early reperfusion therapy. In spite of the class IIa recommendation by the European Society of Cardiology, use of these additional leads frequently is not made (**Steg 2012**).

Figure 4 shows the location of these accessory leads.

Figure 4

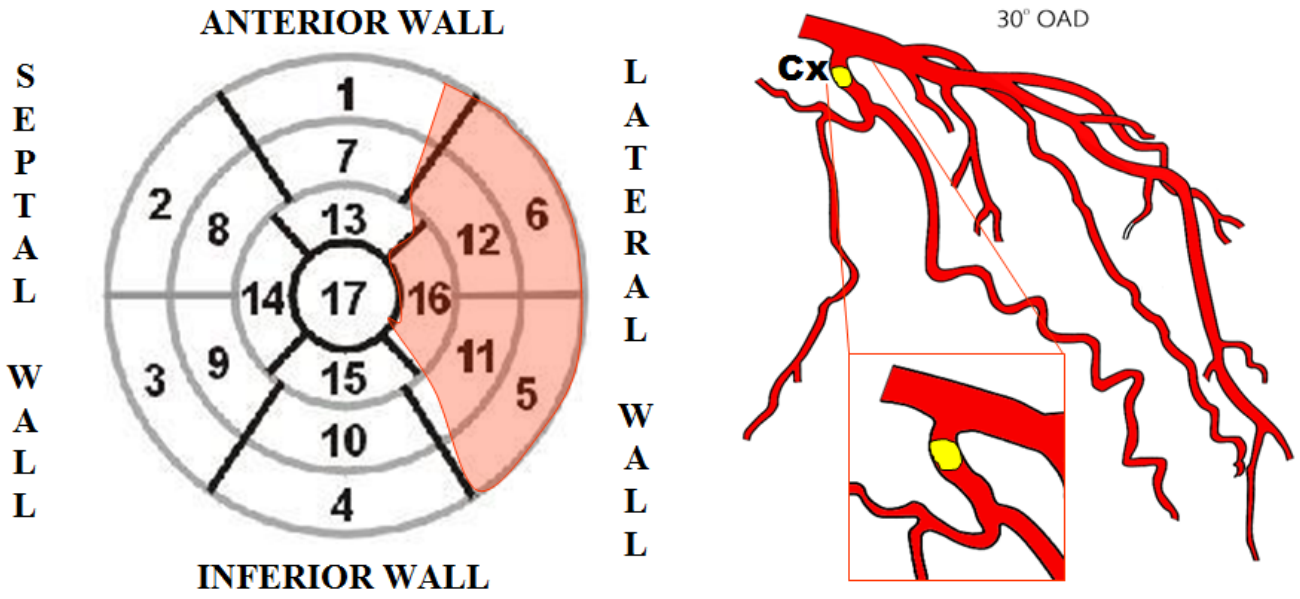


The figure shows the location of the posterior accessory leads V7, V8 and V9.

New electrocardiographic terminology for Q wave infarctions based on the correlation to cardiac NMR in reference to the obstruction in the territory of the left circumflex artery (Bayés de Luna 2006-2007-2007; Luna 2007)

Figure 5A

LATERAL MYOCARDIAL INFARCTION

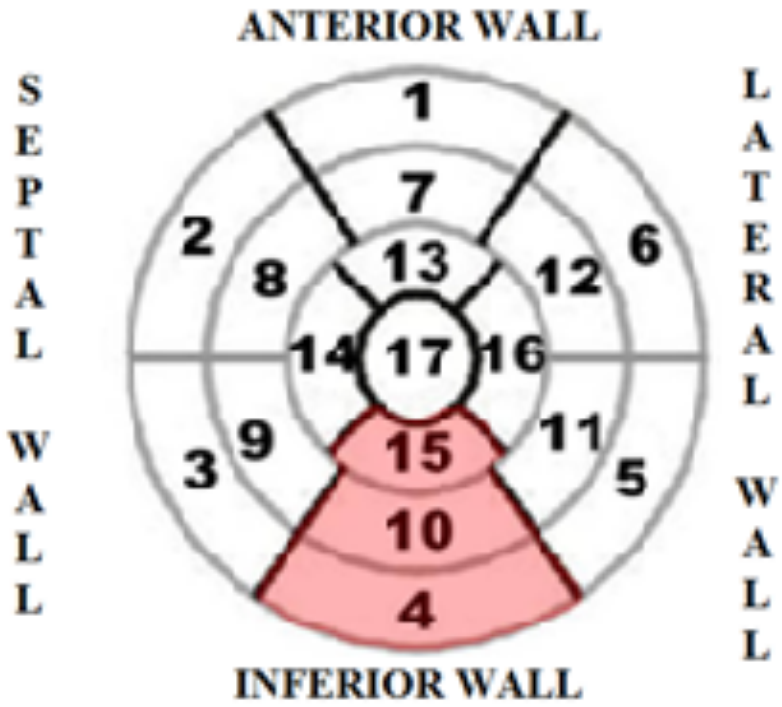


ECG pattern: RS in V1-V2 and/or Q in I, aVL, V5-V6. R voltage with minor amplitude

- **Type:** B-1
- **Most likely site of occlusion:** LCx artery or its marginal oblique branch
- **ECG pattern:** SR in V1-V2 and/or Q in I, VL, V5-V6. Voltage of R wave in V6 of lower amplitude.
- **Segments affected by infarction in cardiac Nuclear Magnetic Resonance (NMR):** 6, 5, 12, 11 and 10.
- **Sensitivity:** 67%
- **Specificity:** 99%.

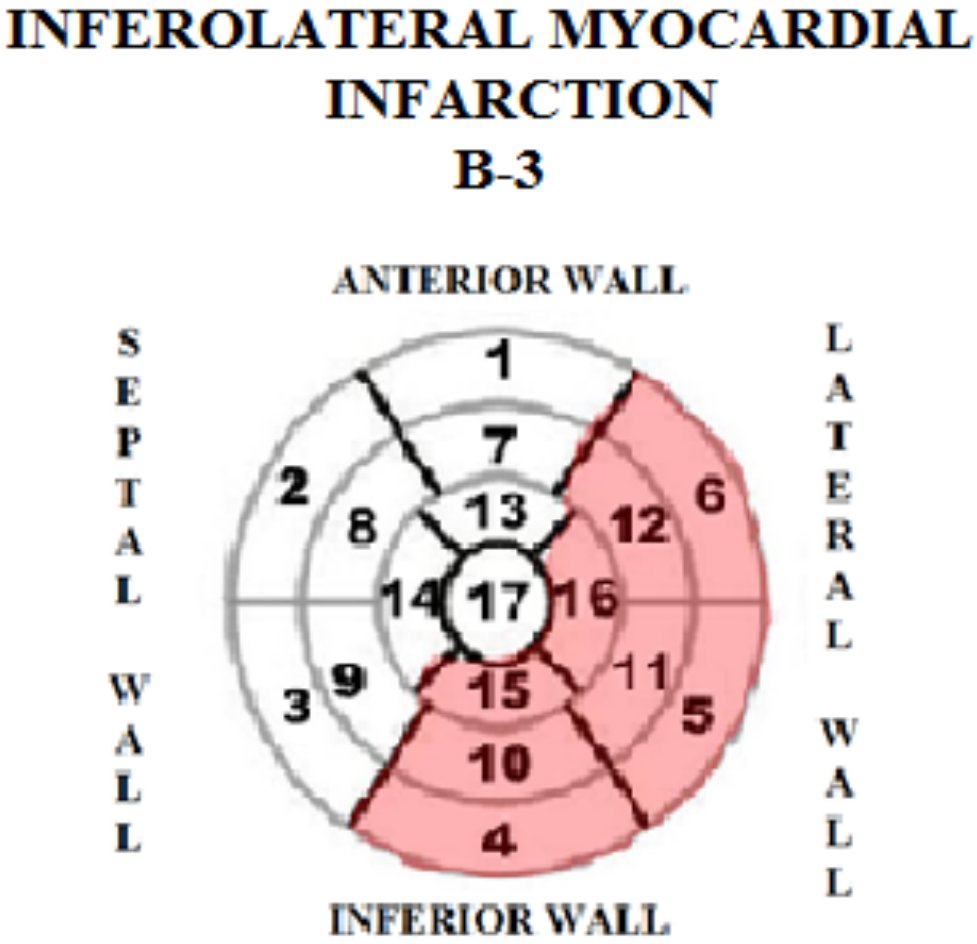
Figure 5B

INFERIOR MYOCARDIAL INFARCTION
B-2



- **Type:** B-2
- **Most likely site of occlusion:** middle third of RCA.
- **ECG pattern:** Q and Qr in II, III and aVF.
- **Segments affected by infarction in cardiac NMR:** 4 and 10.

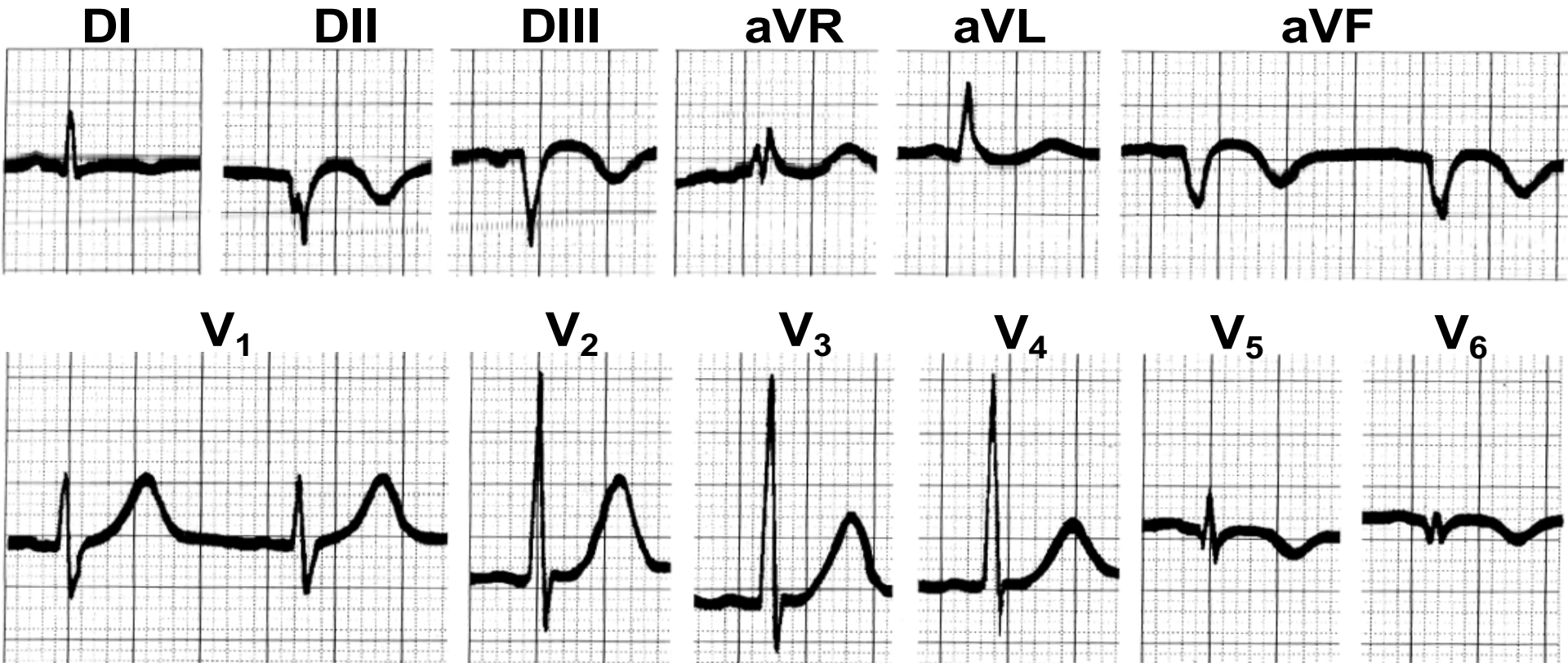
Figure 5C



- **Type:** B-3
- **Most likely site of occlusion:** proximal RCA.
- **ECG pattern:** QS in II, III and aVF and Q in I, aVL, V5-V6.
- **Segments affected by infarction in cardiac NMR:** 15, 10 and 4/5, 6, 12, 11 and 16.

Figure 6 shows a case of late infarction of the B3 type in infero-lateral wall.

Figure 6

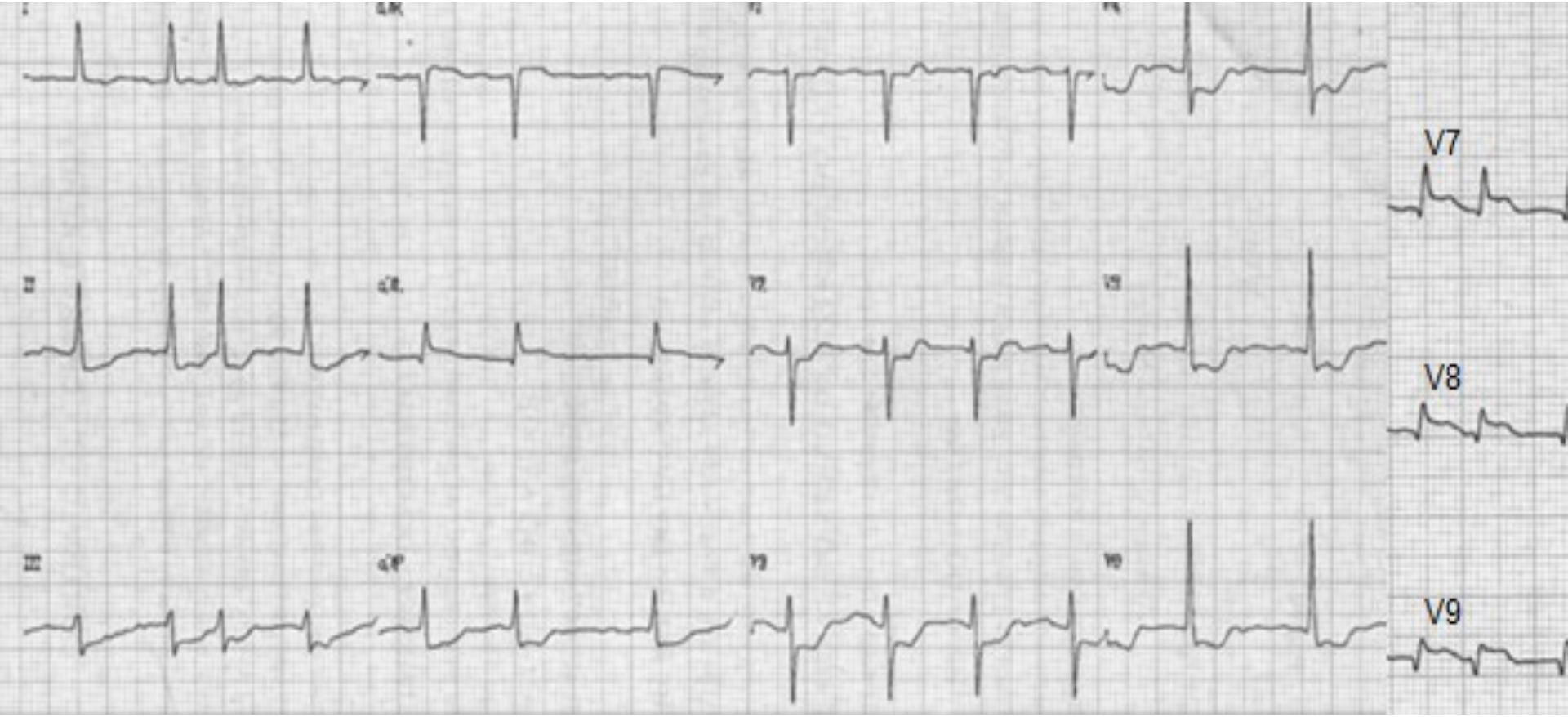


ECG diagnosis: Infero-lateral electrically inactive area corresponding to B3 infarction (Figure 5C) using the new ECG/cardiac NMR correlation classification: QS in II, III and aVF with prominent R in V1-V2 and low voltage r/R in V5 and V6. Inferior infarction is extensive, affecting the entire inferior wall, which explains the absence of final r or R wave in II, III and aVF. Involvement of the low lateral or apical wall represented by small r waves in leads V5 and V6 indicates that the necrosis also extends into this wall.

Prominent R waves in V1-V2 are no longer thought to be caused by true posterior or dorsal infarction but to lateral left ventricular wall infarction. The coronary angiogram in this case revealed total obstruction of the posterior descending branch of a dominant left circumflex artery.

Figure 7 illustrates an example of a patient with symptoms of acute chest pain, in which the posterior accessory leads were essential for the diagnosis of ST segment elevation AMI. Coronary angiography revealed total acute occlusion of the LCx artery.

Figure 7

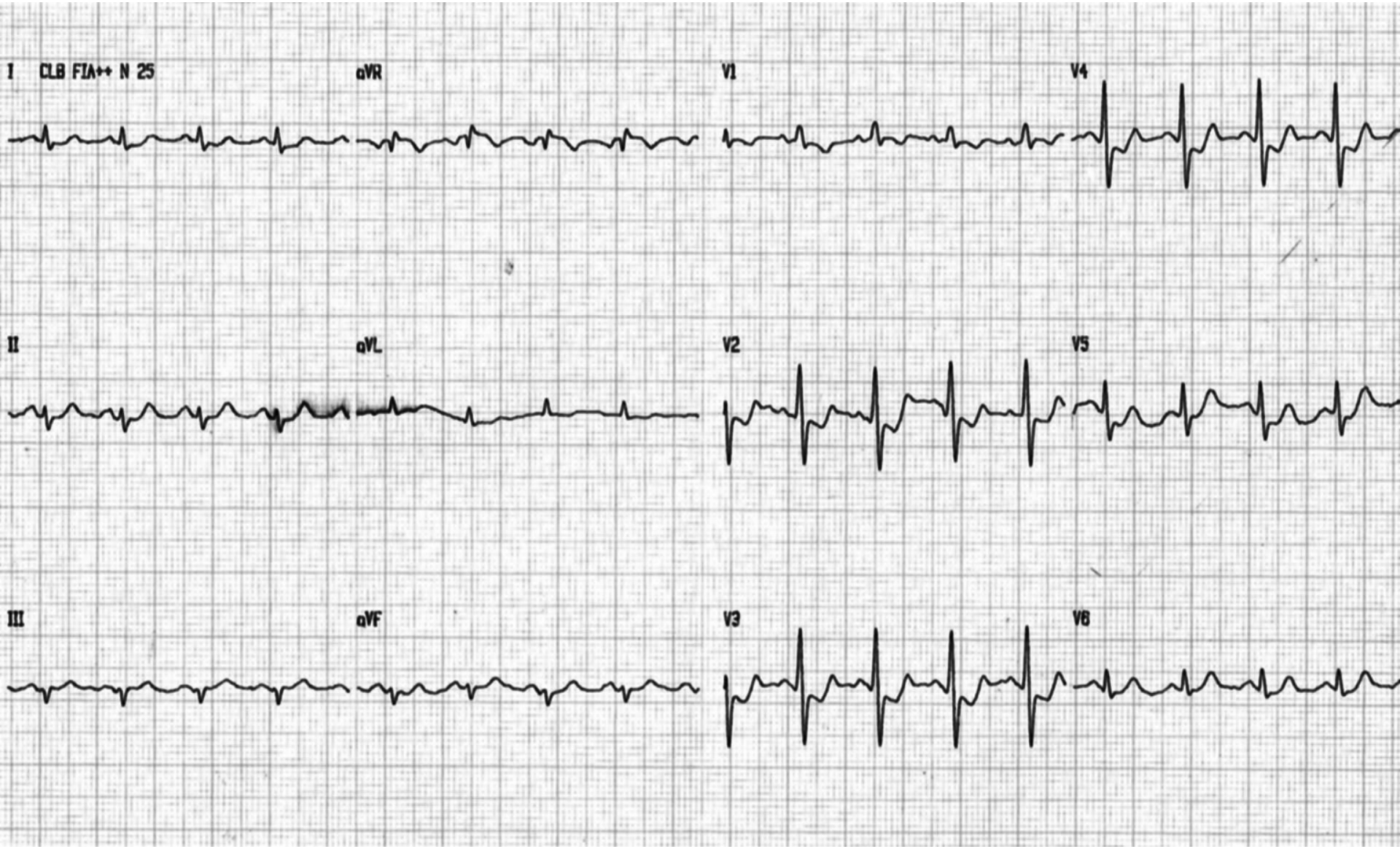


ECG diagnosis: Atrial fibrillation, LVH, and ST segment depression in the inferior leads and leads V2-V6. Only the posterior accessory leads V7-9 revealed pathologic Q waves and ST segment elevation (STE-ACS).

A recent study was done evaluating the percent of patients presenting with equivocal ECG criteria of ST segment elevation AMI that were mistakenly managed as non-ST segment elevation AMI. The results showed 55% of patients that exhibited ST segment depression from V1 to V6 and inferior ST segment elevation had angiographic evidence of LCx occlusion (**Hamm 2011**).

Figure 8 shows an example of a patient admitted with prolonged precordial pain and ST segment depression from V1 to V4 without ST segment elevation; coronary angiography the next day revealed total occlusion of marginal branch of the LCx artery.

Figure 8

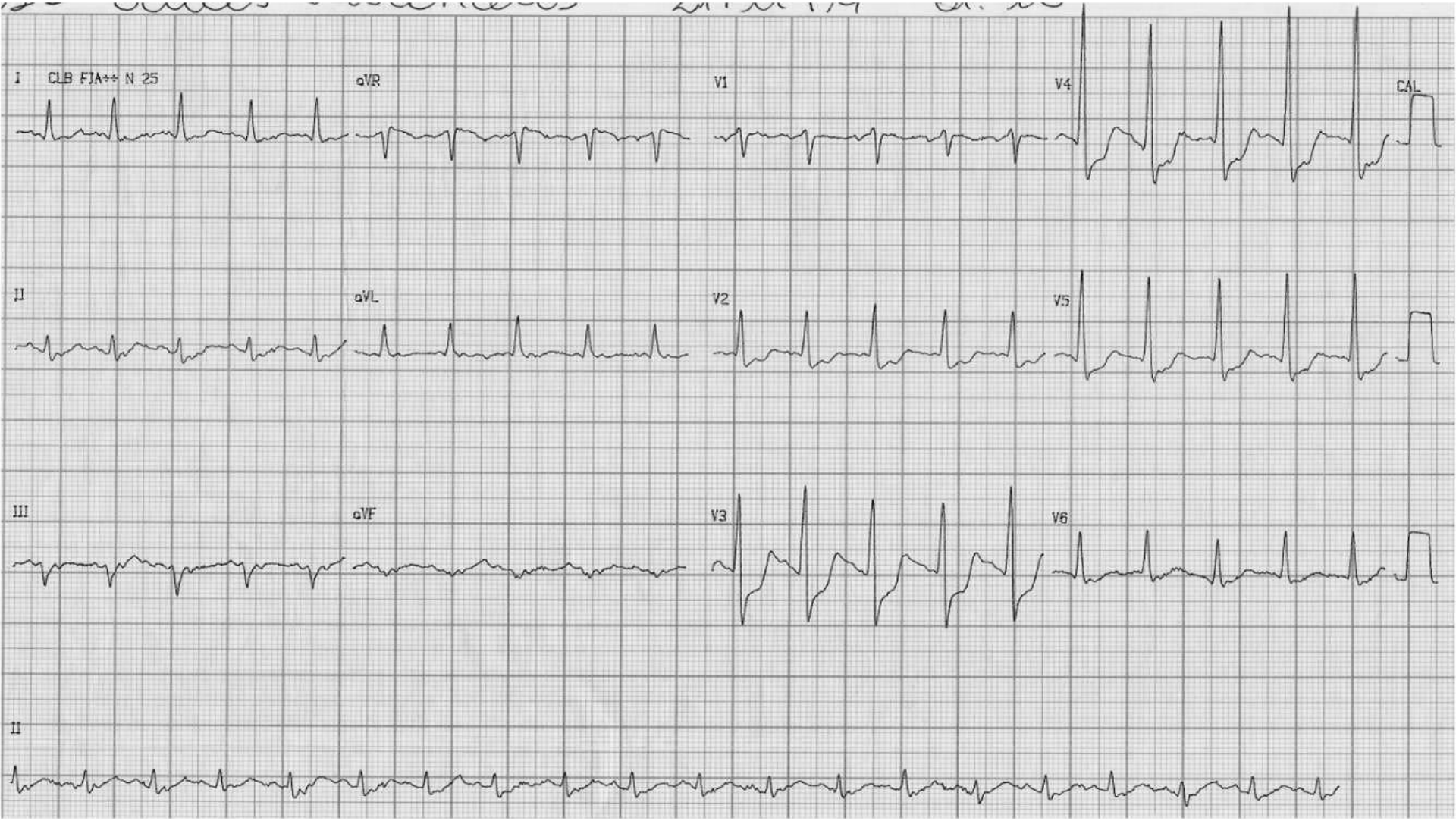


Consequences of not making an early diagnosis

There are no large, randomized studies in literature to analyze the consequences of not recognizing by ECG an acute occlusion of the LCx artery. However, it is known that acute LCx occlusion with inferior or infero-lateral necrosis can result in rupture of the postero-medial papillary muscle with severe hemodynamic consequences due to mitral valve regurgitation, worsening congestive heart failure, acute pulmonary edema, and cardiogenic shock. Often vasoactive drugs and mitral valve replacement are needed in addition to revascularization in the acute stage (**Park 2011**). An early diagnosis and emergency surgery are mandatory before these complications develop (**Yanagi 1998**). Transesophageal echocardiogram has a high sensitivity for this diagnosis. Thus, in the setting of cardiogenic shock associated with acute inferior or infero-lateral infarction, severe mitral valve regurgitation should be suspected and confirmed with this technique even when a regurgitation murmur is not audible. The sensitivity of transthoracic echo and left ventriculography is lower than that of transesophageal echo (**Iwasaki 2000; Baruzzi 1994**).

Figure 9 below shows an example of a woman mistakenly treated as NSTE-ACS that, on the second day of admission, evolved into acute pulmonary edema. Echocardiogram revealed infero-lateral LV akinesis, EF of 38%, and severe mitral valve insufficiency. Coronary angiography revealed total LCx marginal branch occlusion; 50% lesion in the middle third of the LDA; 70% in the middle third of the RCA and 90% in the posterior ventricular branch. Ventriculography confirmed severe mitral valve insufficiency. The patient was referred to emergency surgery with mitral valve replacement.

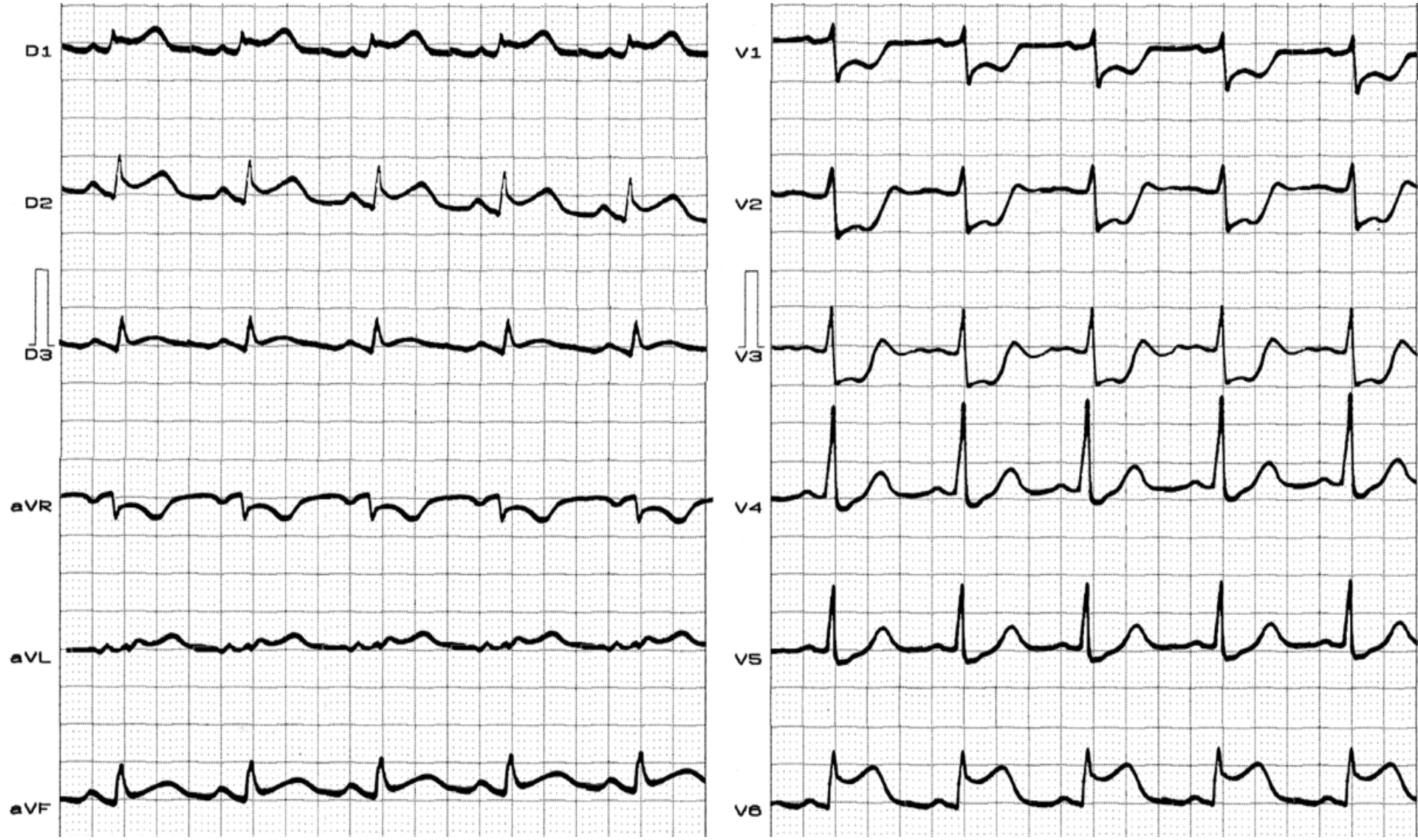
Figure 9



ECG diagnosis: Sinus tachycardia 148 bpm, prominent R in V2 (Rs) and ST segment depression from V2 through V5.

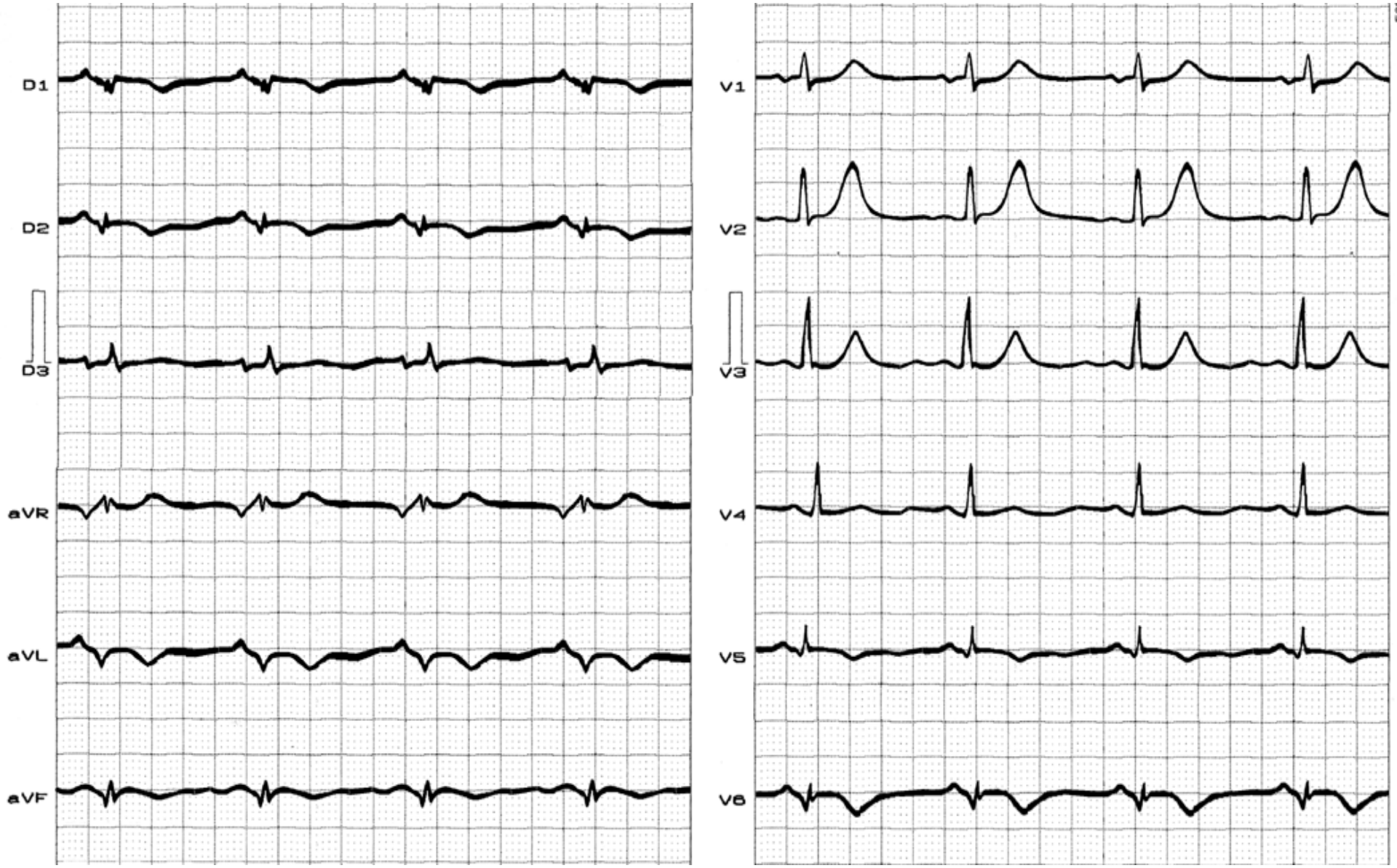
On the occasion of total occlusion of the LCx artery, typical inferior or infero-lateral wall ST segment elevation changes of a STEMI appear, enabling appropriate early management. Figure 10 shows a typical case with these features.

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) required an immediate intervention. When this is not performed the pattern in Figure 11 appears 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 infero-lateral myocardial infarction in the late phase.

Conclusions:

In ACS patients presenting with new ST segment depression in leads V1-V3 who are suspected of acute occlusion in the LCx artery, it is important to add the posterior accessory leads V7-V9. If the ST segment elevation is confirmed in these leads, these patients should undergo emergency myocardial reperfusion therapy according to STEMI guidelines. This approach will often prevent an unfavorable outcome resulting from the lack of identification of circumflex artery occlusion.

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