

Acute Inferior Myocardial Infarction with Right Ventricular Involvement resembling Acute Anteroseptal Myocardial Infarction

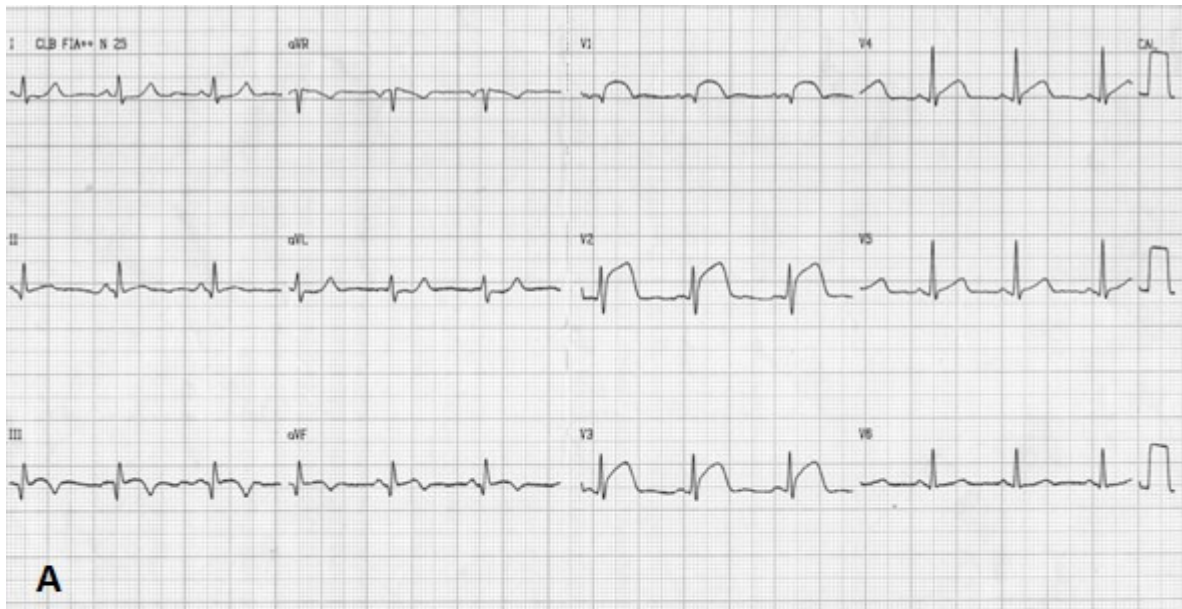
A 39-year-old woman with history of smoking since age 17 was admitted to the ER with symptoms of “stabbing” chest pain radiating into the upper left arm and accompanied by dyspnea, sweating and dizziness. The family history revealed that her mother died at age 49 from “infarction”.

On admission the physical examination was normal with a blood pressure of 130/90 mmHg. The initial electrocardiogram (**Figure 1A**) showed sinus rhythm with convex-upwards ST segment elevation (transmural injury) decreasing from V1 to V3 and acute inferior myocardial infarction pattern with Q waves and ST segment elevation of transmural injury in leads III and aVF. The patient was medicated with aspirin, clopidogrel (300 mg) and enoxaparin, and sent to the catheterization lab. Angiography revealed total proximal occlusion of the right coronary artery (RCA) (**Figure 1B**) and discrete irregularities in the LAD and LCx. (**Figure 1C**).

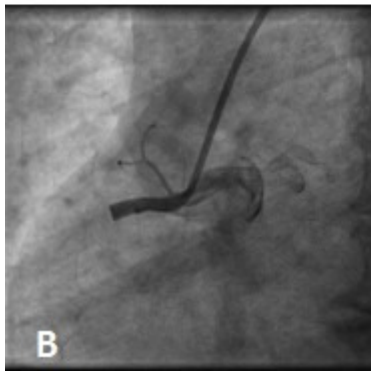
A successful percutaneous coronary intervention with stent implantation in the RCA was performed (**Figure 1D**).

The ECG obtained after the procedure (**Figure 2**) showed normalization of ST segment elevation in the anteroseptal leads with Q waves and evolving ST-T wave changes in leads III and aVF.

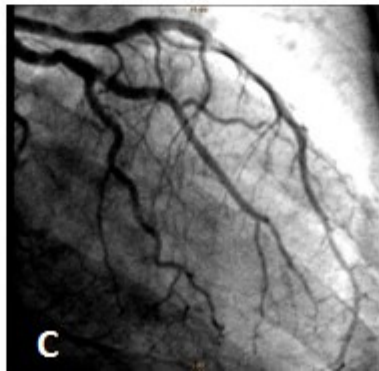
Figure 1A



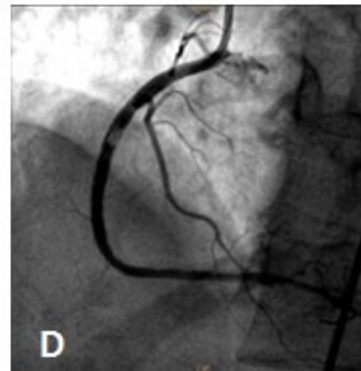
A



B

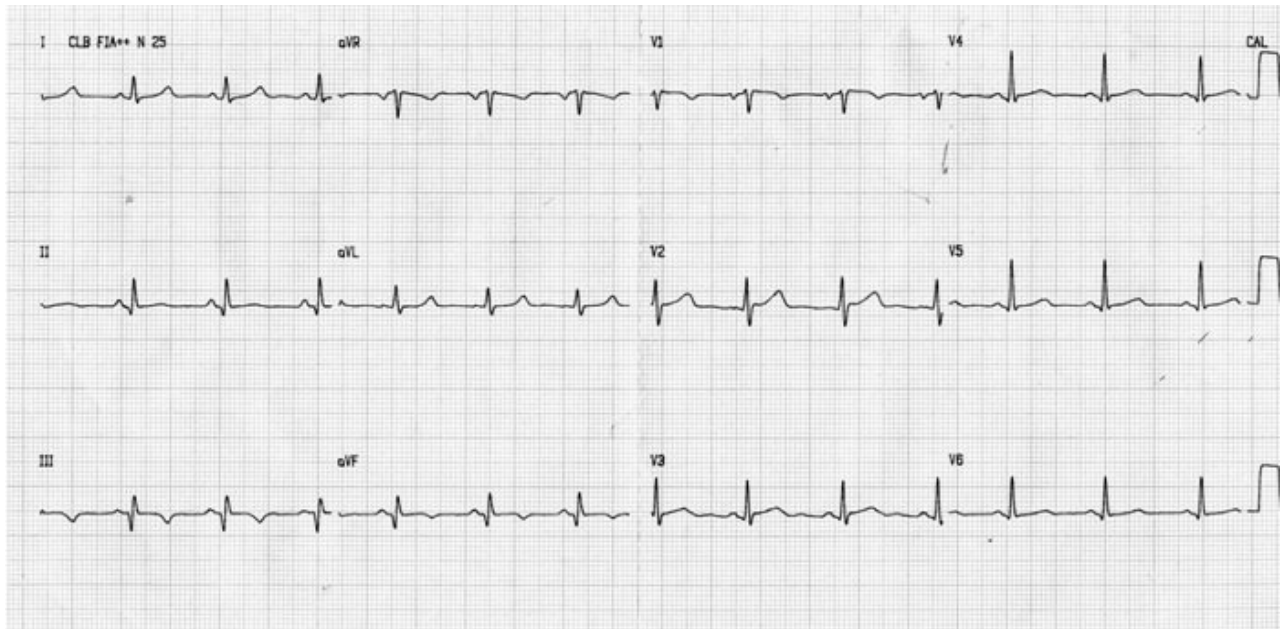


C



D

Figure 2



Electrocardiographic diagnosis: Normalization of ST segment elevation in the anteroseptal leads, with Q wave and subepicardial ischemia (negative broad base symmetrical T waves) persisting in III and aVF.

Colleagues Opinions

This is a wonderful case. The anterior ST elevation can be seen with RV infarction but is usually limited to leads V1 and V2. The presence of ST elevation in leads V3 and V4 with minimal ST elevation in the inferior leads would have made me believe that this would have been an LAD territory STEMI. The angiogram confirms that the RV branch was involved in this RCA distribution MI.

Thanks,

Tim

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Spanish

Interesante caso amigos. La paciente como bien muestra la coronariografía oclusión proximal de la arteria CD que presenta ascenso del ST de V1-V3 con discretos cambios (sub-agudos) en III y aVF. Esto se debe a que estamos ante una CD muy corta. Los cambios son similares a los que ocurren cuando se ocluye de forma aislada la arteria del VD.

Un saludo:

[Javier García-Niebla](#)

English

Interesting case dear friends. The patient as well shows proximal occlusion of the RCA having ST elevation in V1-V3 with discrete changes (sub-acute) in III and aVF in coronary angiography. This is because we have a very short RCA. The changes are similar to those that occur occlusion when isolated RV artery.

Discussion:

In the clinical setting of acute coronary syndrome ST segment elevation in the anterior precordial leads generally implies acute transmural injury in the anteroseptal wall of the left ventricle. However, there are several reports in literature of right ventricular (RV) myocardial infarction presenting with similar ECG findings. Unlike anteroseptal MI, when the RV is affected in the setting of RCA occlusion ST segment elevation from V1 to V3 has a pattern of *decreasing* magnitude from right to left as seen in this case (1,2,3). These ECG findings can be explained by the proximity of the RV to the right precordial leads. The RV is more properly explored by the following ECG leads:

- RV trabecular region: V2
- Right paraseptal inferior region: V3 and V4

- RV free wall from V1 to V4 (antero-septal)
- RV outflow tract, infundibular region: aVR, V_{1H}, V_{2H} and V_{3H}
- RV inflow tract: aVR, V_{4R} and V_{5R}.

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:

- 1) *Increased jugular venous pressure and passive liver congestion*
- 2) *Clean lungs*
- 3) *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.

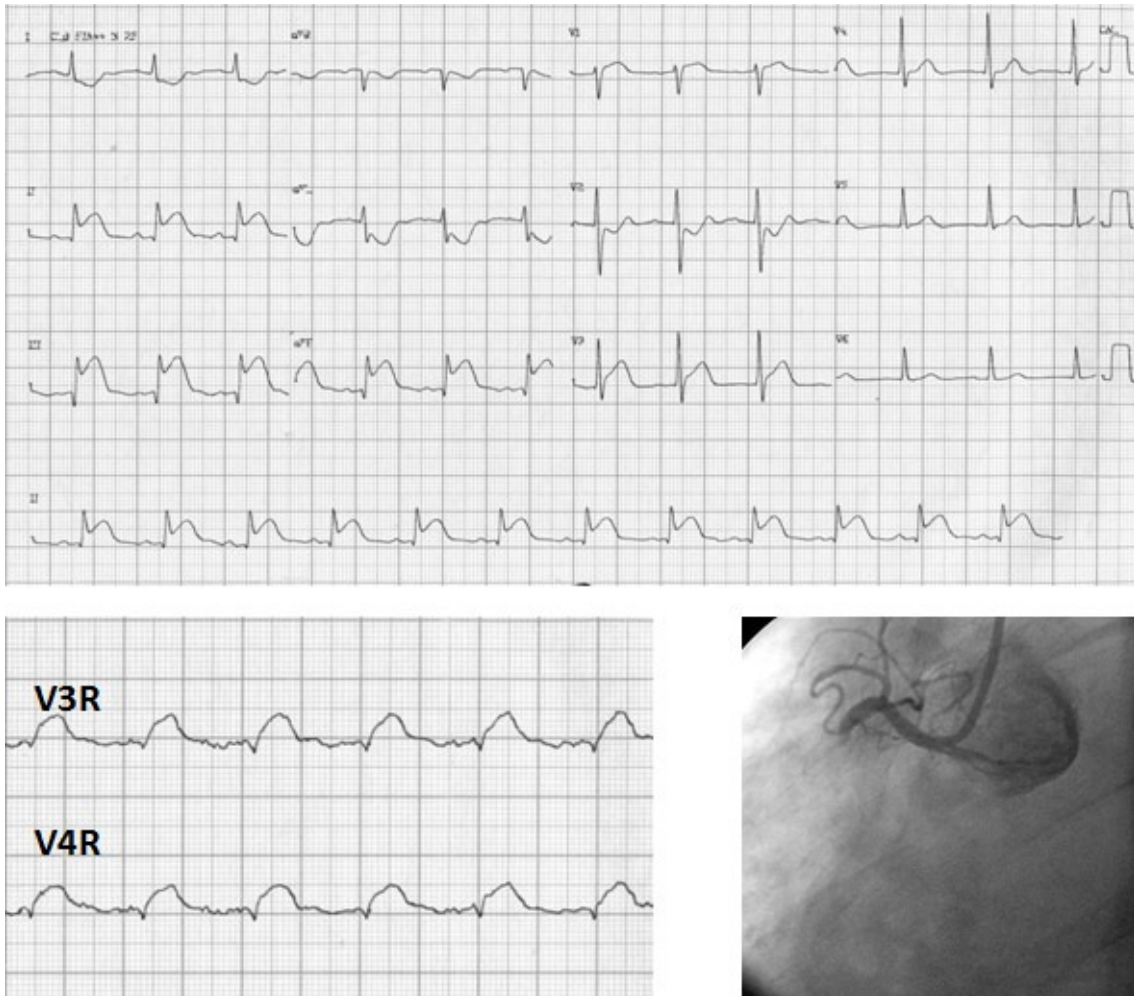
Table 1 shows the main clinical and ECG differences between acute inferior wall myocardial infarction with or without acute right ventricular myocardial infarction.

Table 1(5)

	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 3**. These ECG findings are transient and disappear, on average, within 10 hours after the event in 50% of the patients.

Figure 3



Electrocardiographic diagnosis: another typical case of acute inferior infarction associated with acute RV MI. In the frontal plane the injury vector is pointing towards $+120^\circ$ 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 2 below shows the sensitivity and specificity of the ST segment elevation >1 mm in V1, V3R and V4R (4).

Table 2

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

Main electrocardiographic criteria for Acute Right Ventricular Myocardial Infarction

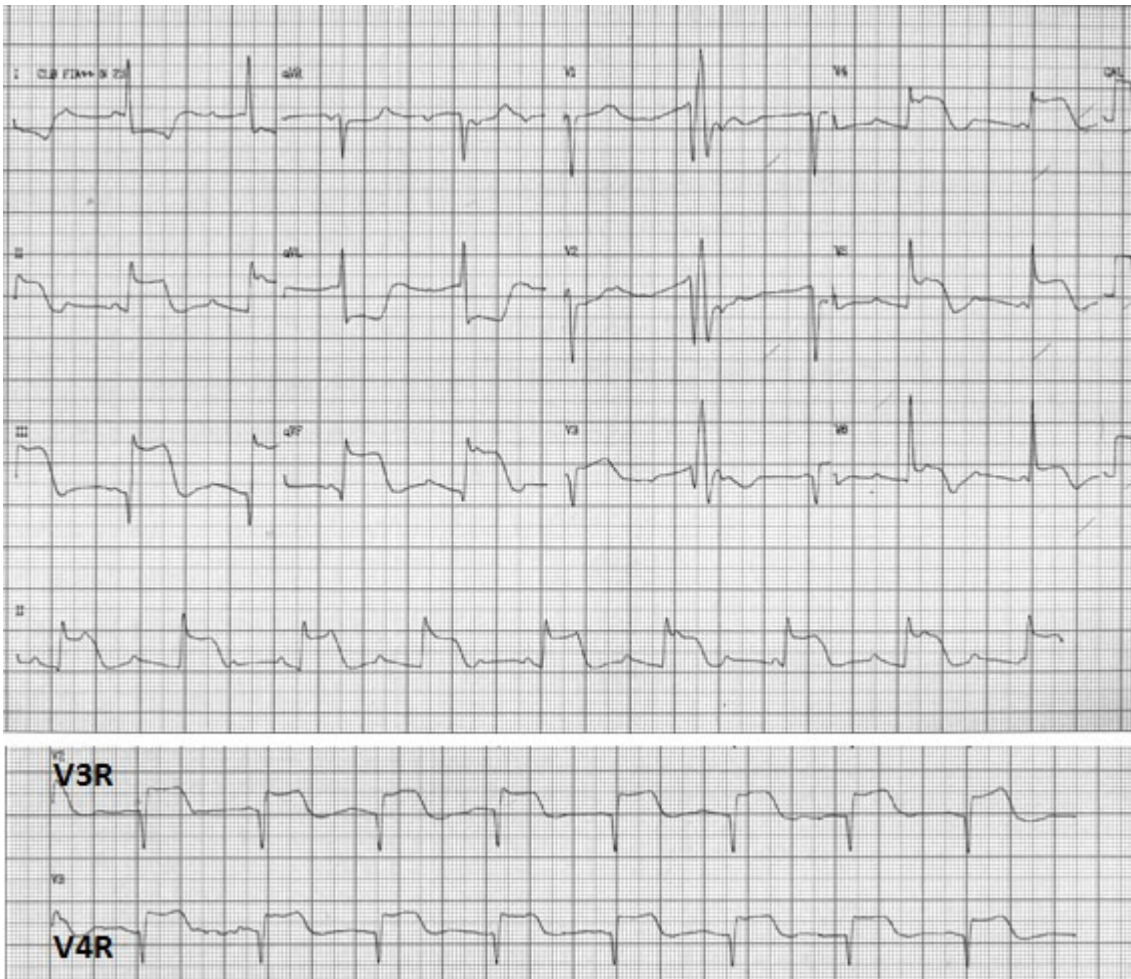
- **Rhythm:** frequent atrial fibrillation, atrial flutter, multifocal atrial pacemakers, and junctional rhythms due to atrial infarction ($\frac{1}{3}$ of the patients present concomitant atrial infarction).
- **P wave:** right atrial enlargement (RAE) pattern may be seen as a consequence of increased 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 V_{4R} to V_{6R} 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 V₁ to V₃, 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 V₁ to V₃.
- 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.

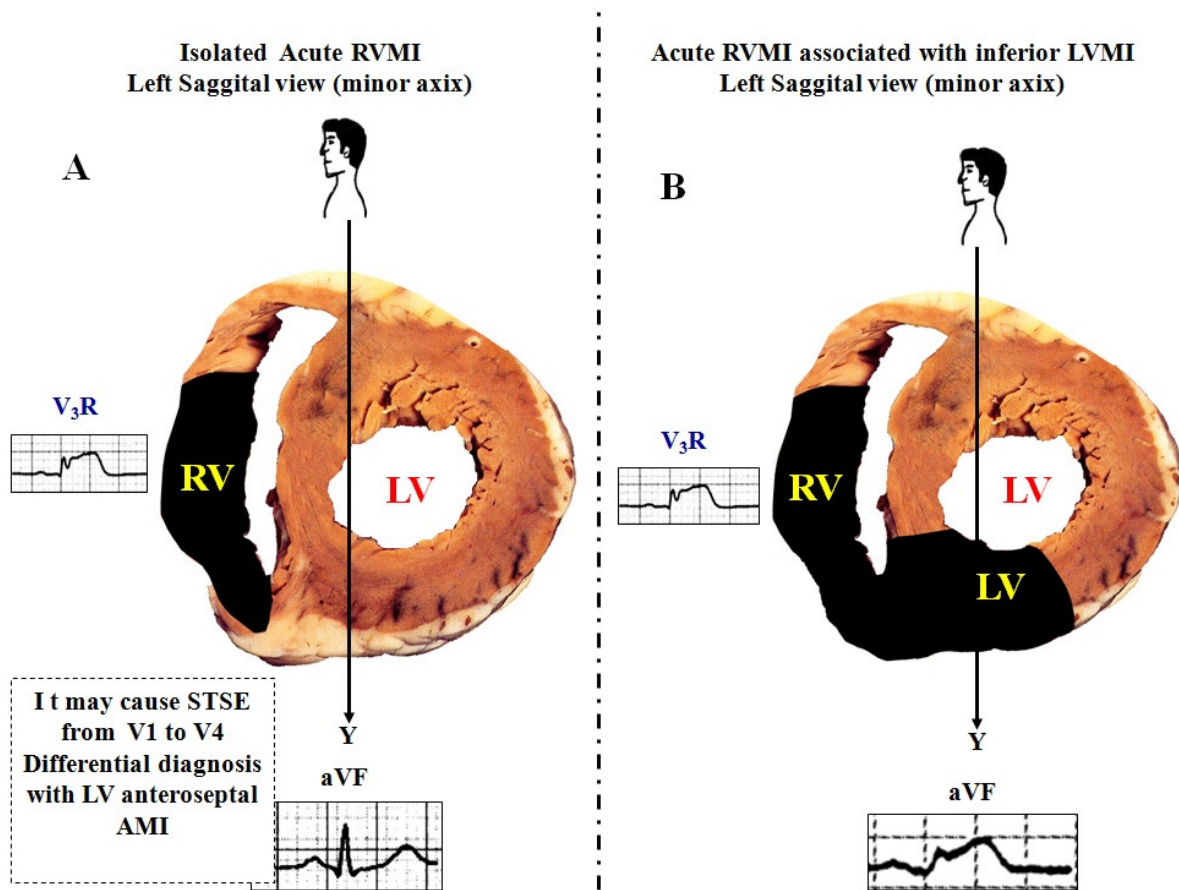
Patients with inferior wall infarction with additional RV involvement have a greater incidence of complete AV block which leads to worsening hemodynamics due to atrioventricular dyssynchrony. The ischemic RV with reduced compliance depends to a large extent on efficient atrial contractions to fill adequately.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

Figure 4



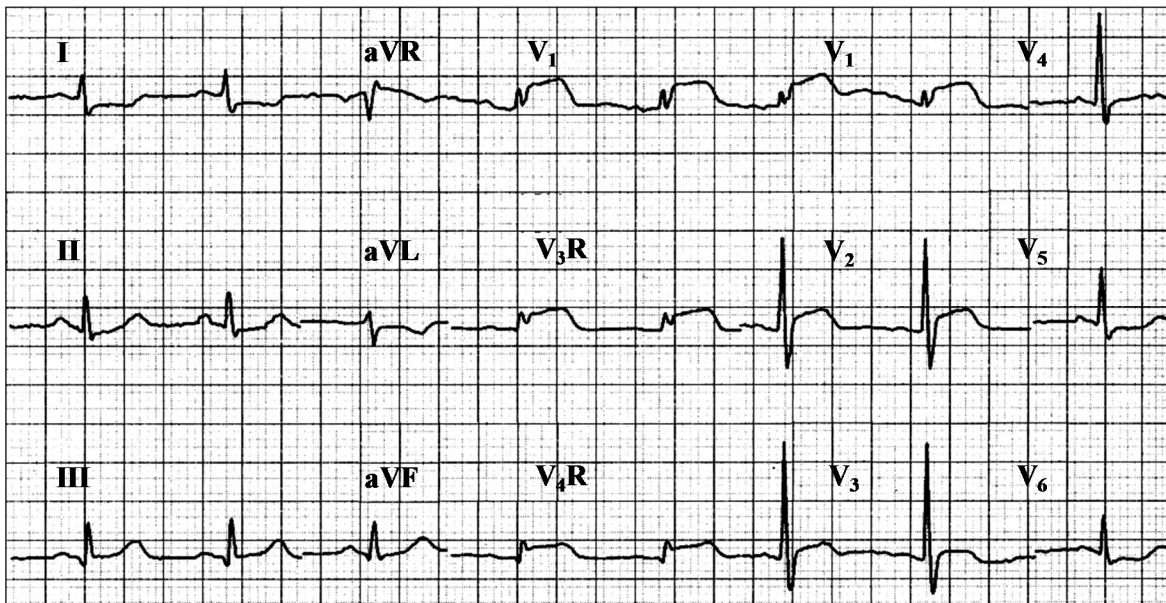
Electrocardiographic diagnosis: The tracing shows acute infero-lateral MI complicated with complete AV block. In the frontal plane STE III is greater than STE II (ST injury vector pointing to III). QS or QR waves associated with ST segment elevation in the right precordial leads V3R and V4R indicate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

Figure 5



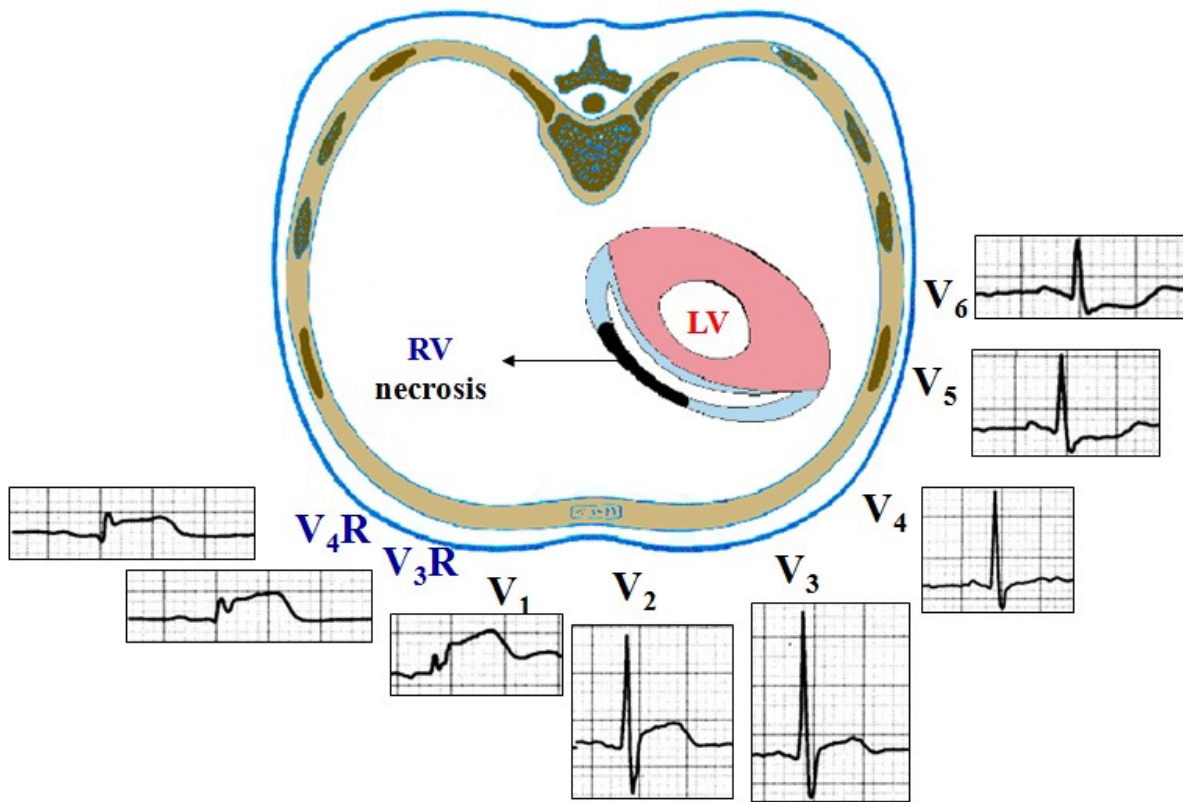
The figure shows electro-anatomical differences between isolated right ventricle acute myocardial infarction (A) and RV MI associated with diaphragmatic or inferior acute myocardial infarction of the left ventricle (B). In figure 5A ST segment elevation (transmural injury) is seen only in anteroseptal wall leads (V1 to V3) and right accessory leads (V₃R and V₄R) without changes in the inferior leads.

Figure 6



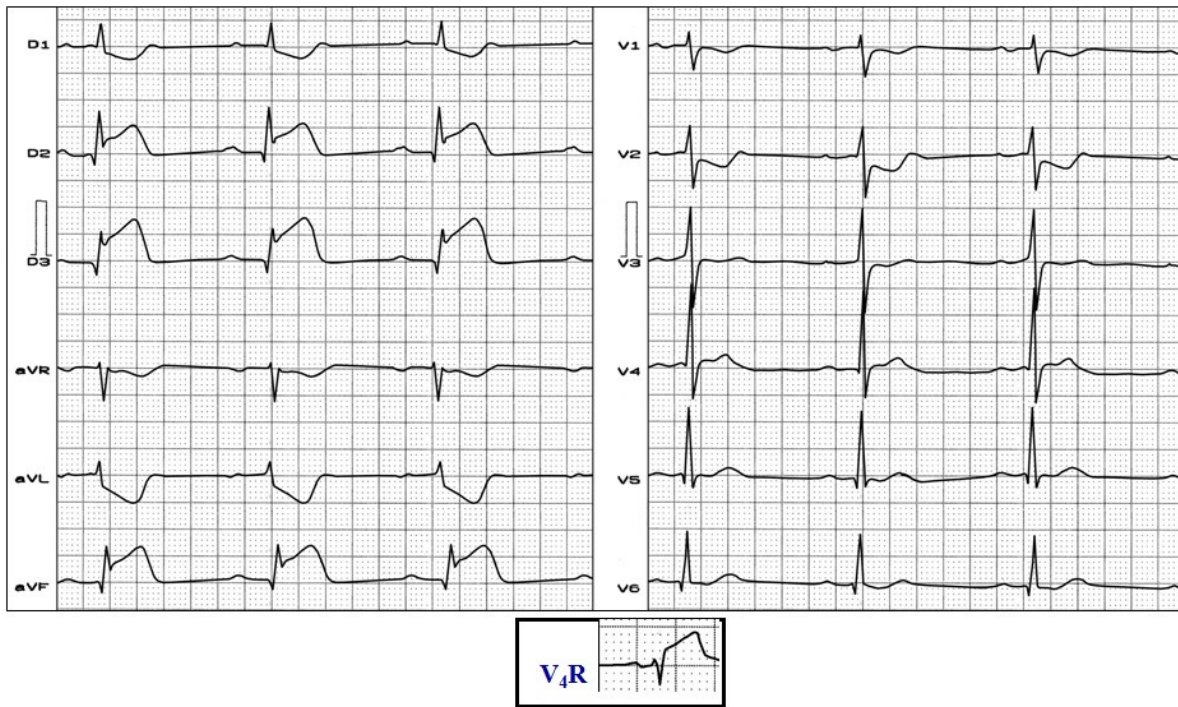
Electrocardiographic diagnosis: ST segment elevation reflecting transmural injury in leads V3R, V4R, V1 and V2. This ECG is a rare case of isolated acute right ventricular myocardial infarction without inferior wall left ventricle involvement.

Figure 7



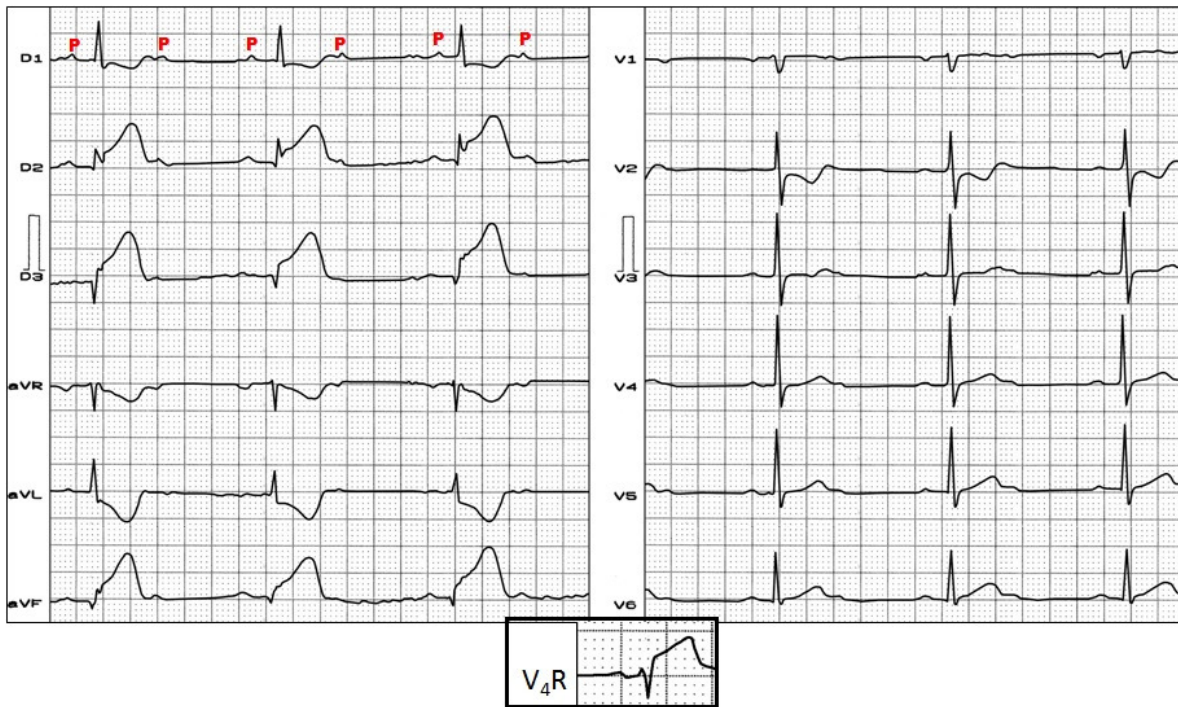
The figure 7 shows the six conventional precordial leads and the accessory right precordial leads V3R, V4R in acute isolated right ventricular myocardial infarction. ST segment elevation is seen in in V3R, V4R, and from V1 to V3.

Figure 8



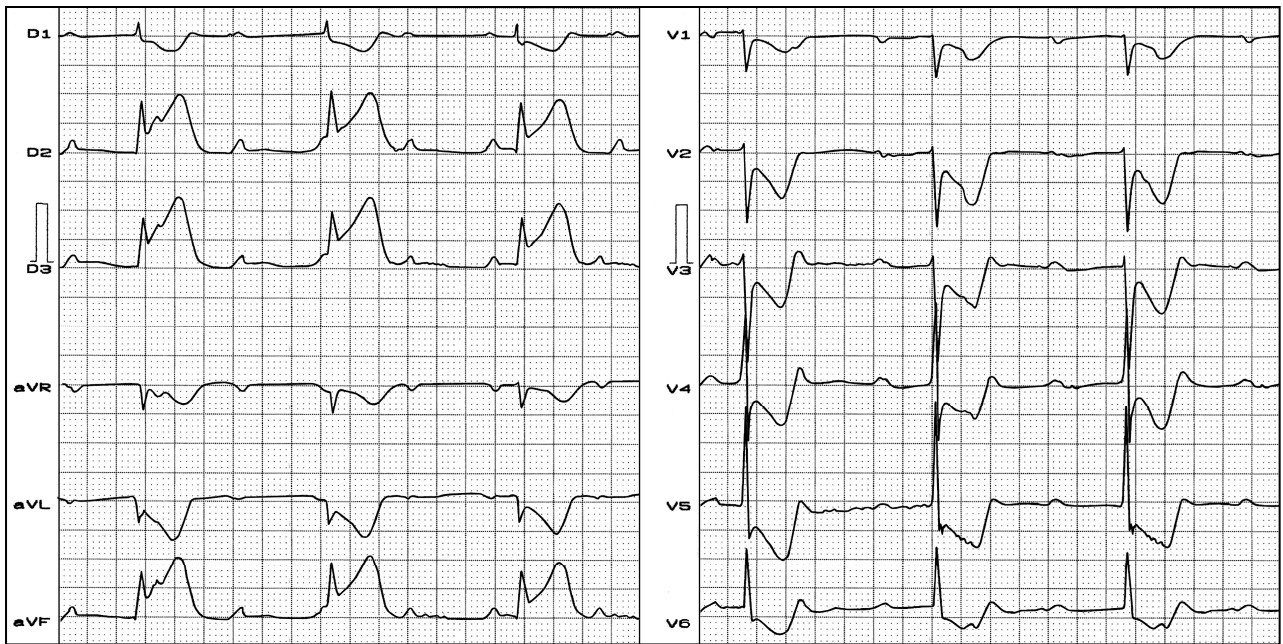
Electrocardiographic diagnosis: sinus bradycardia, first-degree AV block, ST segment elevation in the inferior leads with $ST_{III} > ST_{II}$ (the ST injury vector pointing to III). This pattern is compatible with acute inferior myocardial infarction resulting from proximal RCA occlusion with additional 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 9



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 10



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 (6,7,8). 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 (9,10,11). 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.

References

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