Case Report

# Simultaneous right coronary artery spasm in a patient with Anterior ST-Segment Elevation Myocardial Infarction: a case report

**Zhiva Taherpour[1]*, Seyed Masoud Seyedian[2], Mohammad Alasti[3]**

[1] Department of Cardiology, Golestan Hospital, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran
[2] Department of Cardiology; Cardiovascular Research Center; Golestan Hospital; Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran
[3] Cardiovascular research center; Golestan Hospital; Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran

## Abstract

Simultaneous occlusion of two vessels causing infarction at different territories is an uncommon finding. We report simultaneous right ventricular and anterior ST-segment elevation myocardial infarction in a previously healthy young man.

The angiographic results demonstrated the simultaneous occlusion of the right and left coronary arteries because of simultaneous occlusion of left anterior descending artery (LADA) and spasm of right coronary artery (RCA). In this patient, we found simultaneous ST elevations in right and precordial leads so everyone should be careful about all leads of the surface electrocardiogram for decision making in the management of a patient.

**Keywords:** ST Segment Elevation Myocardial Infarction, Coronary Artery Spasm, Right Ventricle

***Corresponding Author:** Zhiva Taherpour, Department Of Cardiology, Golestan Hospital, Golestan Blvd, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran, E- mail: zh_taherpour@yahoo.com

## Introduction

Right ventricular (RV) myocardial infarction (MI) usually occurs in association with inferior MI. We present a case who suffered RV MI in association with anterior MI. In this case coronary angiography revealed simultaneous occlusion of left anterior descending artery (LADA) and spasm of right coronary artery (RCA).

**Case presentation:**

A previously healthy 45-year-old man presented to the emergency department (ED) with retrosternal chest pain. His chest pain was sudden in onset and had started 20 minutes before coming to the ED. It had awakened him in the early morning. He described it as a heavy pain that was severe in intensity, with radiation to his left shoulder and arm. He also noted diaphoresis and nausea, and denied any shortness of breath.

The patient's family history was significant for Coronary Artery Disease (CAD), in his 50-year-old brother. On physical examination, his pulse was regular, with a rate of 88 bpm; his blood pressure was 100/60 and oral temperature was 37°C.

The examination of the head and neck was unremarkable. The neck examination showed no jugular venous distension. His cardiovascular examination revealed normal heart sounds. The chest examination showed good respiratory effort, with normal breath sounds. The peripheral pulses were strong and symmetric in all four extremities. The laboratory evaluation included the following:

Hb: 12.6g/dl; WBC: 7000/mm3; Creatinin: 0.7mg/dl; BUN: 14mg/dl; BS: 101mg/dl; K: 4.6mmol/lit; Na: 141mmol/lit, CRP: Negative; Cardiac Troponin I: positive; TG: 88mg/dl; LDL: 64mg/dl; HDL: 35mg/dl; PTT and INR were normal.

Electrocardiography (ECG) showed sinus rhythm with ST-segment elevation of 2-4 millimeters in all precordial leads also in V4R and V5R (Figure 1), without ST-segment elevation in inferior leads. ST-segment elevation in v2 and v3 was higher than in v1.

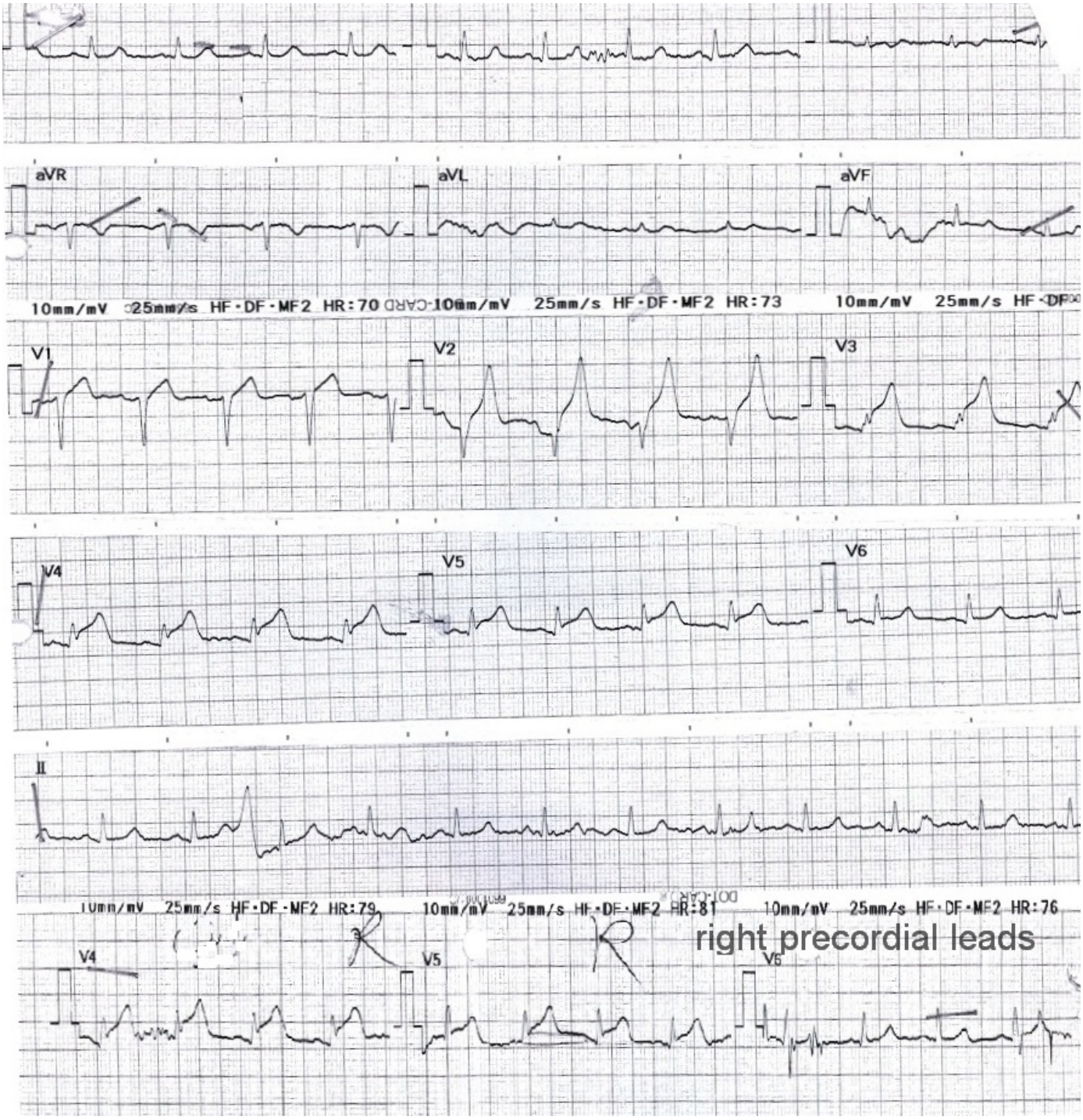


**Figure 1: Electrocardiography obtained at presentation**

The results of ECG were consistent with acute myocardial injury in anterior wall and right ventricle. As in our hospital or near it there was not any center for primary percutaneous coronary interventional procedure, so Immediately after coming to the ED and recording the ECG, fibrinolytic therapy was started. After fibrinolytic therapy, the patient's chest pain was relatively relieved and elevations of the ST-segments were reduced.

Transthoracic echocardiography revealed hypokinesia at mid inferior and anteroseptal walls and reduced right ventricular function; with left ventricular ejection fraction estimated at 35%, accompanied by trivial mitral regurgitation and grade 2 diastolic dysfunction.

Because of low left ventricular ejection fraction, coronary angiography was performed and revealed a significant lesion near the proximal part of LADA, and also during RCA angiography the spasm of the proximal portion of RCA occurred, which relieved after using Nitroglycerin directly in RCA (Figure 2). ECG changes in right leads during RCA spasm mimicked the initial changes at presentation time and returned to baseline after intracoronary nitroglycerin.

After performing myocardial perfusion scan with Single Photo Emission Tomography method and demonstrating viable tissue in LADA territory, the patient was recommended for stenting on LADA.

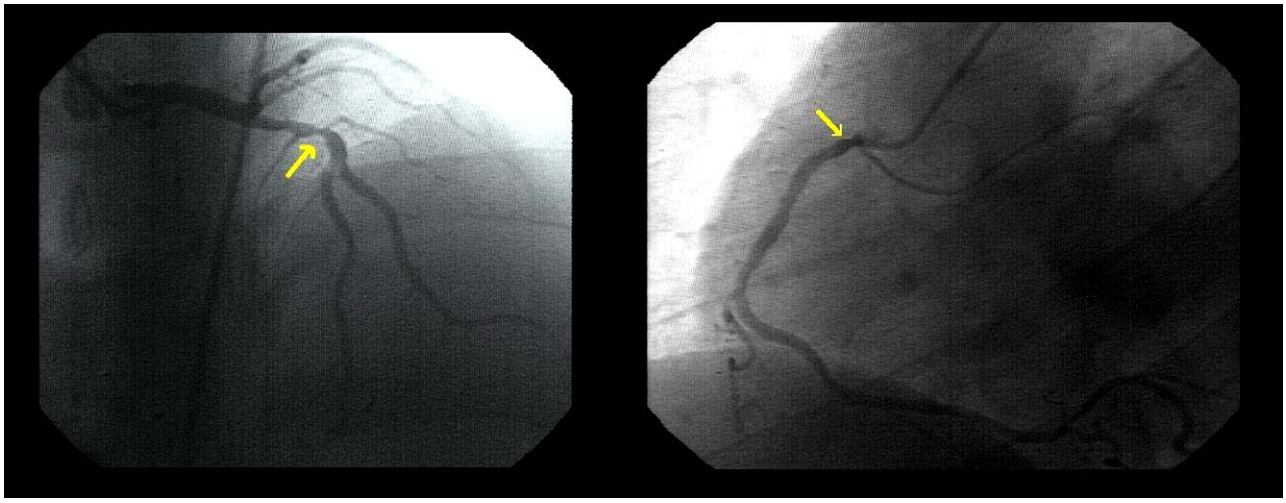

**Figure 2: Angiographic results**

## Discussion

This patient presented with chest pain and ST-segment elevation, a combination that invokes a differential diagnosis that clinicians must recognize to initiate appropriate therapy. The distribution and shape of the ST-segment elevation and associated electrocardiographic findings provide important clues to diagnosis [1]. Differential diagnosis of ST-segment elevation on ECG listed in the Table 1.

In myocardial infarction caused by occlusive thrombus, ST elevations are typically convex in shape and occur in a localized anatomical distribution [2]. Widespread ST-segment elevations across the precordials and limb leads, as well as upward concave ST-segments and PR-segments depression are highly characteristic of pericarditis. ECG pattern of myocarditis is indistinguishable from that of pericarditis [1].

Prinzmetal's angina is another differential diagnosis for ST-segment elevation. When an epicarial artery is completely "pinched off" as a result of spasm, the ST-segment becomes elevated in the leads facing the affected area, reflecting transmural ischemia. In this condition, the spasm is usually brief and the ST-segment returns to normal, with no resultant myocardial damage. The ST-segment elevation in prinzmetal's angina and in acute infarction is indistinguishable, since they reflect to the same pathophysiological process: transmural ischemia from occlusion of an epicardial artery by

transient spasm in the first condition and by persistent thrombus in the second, if the spasm lasts long enough, infarction results [3]. In this patient there was ST-segment elevation in precordials and RV leads. In distinct RV MI-related precordial ST-elevation pattern, V2 shows the highest STE which decreases towards more left sided leads or STE in V1 > V3 with absence of progression of STE from V1 to V3 that may facilitate differentiation from anterior MI [4], where almost exclusively V2-V4 shows the greatest STE and STE in V3 is higher than in V1 [5-7]. In our case, we found STE in V1-V5 and V4R, V5R, which was higher in V3 than V1.

**Table 1: Some causes of ST-segment elevation in Electrocardiogram**

| Cause | Features |
|---|---|
| **Myocardial infarction** | Convex ST segment typically limited to a single coronary artery territory RCA:II≥III , aVF ; LCX:I,aVL,V5,V6; LADA:V1-V3 |
| **Acute pericarditis** | Diffuse concave ST segment elevation of precordial and limb leads & ST depression in aVR ; ST:T ratio≥0.24 in V6 |
| **Myocarditis** | Same as myocardial infarction or pericarditis may be associated with atrial or Ventricular arrhythmia and/or heart block |
| **Prinzmetal's angina** | Typically limited to a single coronary artery distribution |
| **Pulmonary embolism** | Mimicking right ventricular infarction ( III,aVF,V1) sinus tachycardia, incomplete or complete RBBB,S1Q3T3 pattern |
| **Aortic dissection (involving coronary ostium)** | RCA ostium involvement is more common than left main coronary involvement; III≥II, aVF |
| **Myocardial contusion** | Right ventricular involvement V1,V2 |
| **Normal (male pattern)** | Seen in approximately 90% of healthy young men; therefore, normal elevation of 1-3mm most marked in V2 concave |
| **Early repolarization** | Most marked in V4, with notching at J point; tall, upright T waves; reciprocal ST depression in aVR, not in aVL, when limb leads are involved |
| **ST elevation of normal variant** | Seen in V3-V5 with inverted T waves Short QT, high QRS voltage |
| **Left ventricular hypertrophy** | Concave; other features of left ventricular hypertrophy |
| **Left bundle branch block** | Concave; ST segment deviation discordant From the QRS |
| **Acute pericarditis** | Diffuse ST segment elevation ; reciprocal ST segment depression in aVR, not in aVL; seldom≥5mm; PR depression elevation |
| **Hyperkalemia** | Other features of hyperkalemia present: widened QRS and tall, peaked, tented T waves Low amplitude or absent P waves ST segment usually down sloping |
| **Brugada syndrome** | rSR′ in V1 and V ST segment elevation in V1& V2, typically downsloping |
| **Pulmonary embolism** | Changes simulating myocardial infarction Seen often both in inferior and anteroseptal leads |
| **Cardioversion** | Striking ST segment elevation often≥10mm, but lasting only a minute or two immediately after direct-current shock |
| **Prinzmetal's angina** | Same as ST segment elevation in infarction, but transient |
| **Acute myocardial infarction** | ST segment with a plateau or shoulder or upsloping reciprocal behavior between aVL and III |

## Conclusion

Simultaneous RCA and LAD artery involvement producing combined STE in V1-V5 and right leads is possible. But simultaneous complete occlusion of two major coronary arteries is rare. It has been suggested that coronary spasm may lead to infarction [8], so another possibility is the occlusion of one of them and spasm in the other one; as coronary arteriography confirmed this possibility.

His coronary arteriography showed near proximal (after small S1, before D1) Significant LADA lesion, and there was no obstructive lesion but spasm at proximal part of RCA which relieved after direct administration intracoronary nitroglycerin.

The goal of the initial management of ST-elevated myocardial infarction includes swift restoration of flow within the occluded coronary artery, prevention of early infarction and avoidance of complications of reperfusion therapy. In this patient, we found simultaneous ST elevation in right and precordial leads so everyone should be careful about all leads of the surface electrocardiogram for decision making for the management of such patients.

## References


1. Lewis GD, Holmes CB, Holmvang G, Butterton JR. Case records of the Massachusetts General Hospital. Case 8-2007. A 48-year-old man with chest pain followed by cardiac arrest. N Engl J Med. 2007 Mar 15;356(11):1153-62.
2. Zimetbaum PJ, Josephson ME. Use of the electrocardiogram in acute myocardial infarction. N Engl J Med. 2003 Mar 6;348(10):933-40.
3. Wang K, Asinger RW, Marriott HJ. ST-segment elevation in conditions other than acute myocardial infarction. N Engl J Med. 2003 Nov 27;349(22):2128-35.
4. Muhammad KI, Kapadia SR. Anterior ST-segment elevation with right coronary artery occlusion: a unique case of isolated right ventricular infarction. Angiology. 2008 Oct-Nov;59(5):622-4.
5. Andreou AY, Ioannides MA, Georgiou GM, Avraamides PC. Diffuse precordial ST-segment elevation in inferior-right myocardial infarction. Cardiol J. 2010;17(6):628-31.
6. Halkett JA, Commerford PJ, Millar RS. Right ventricular infarction mimicking extensive anterior infarction. Chest. 1986 Oct;90(4):617-9.
7. Geft IL, Shah PK, Rodriguez L, Hulse S, Maddahi J, Berman DS, et al. ST elevations in leads V1 to V5 may be caused by right coronary artery occlusion and acute right ventricular infarction. Am J Cardiol. 1984 Apr 1;53(8):991-6.
8. Vincent GM, Anderson JL, Marshall HW. Coronary spasm producing coronary thrombosis and myocardial infarction. N Engl J Med. 1983 Jul 28;309(4):220-3.