

ST-Segment Elevation: Not Always an Acute Coronary Syndrome

Érico Costa ¹, Elsa Meireles ¹, Catarina Rodrigues ², Tiago Teixeira ^{3,*}, Dulce Silva ¹

¹ Internal Medicine Department, Centro Hospitalar Entre Douro e Vouga, Santa Maria da Feira, Portugal

² Oncology Department, Centro Hospitalar Entre Douro e Vouga, Santa Maria da Feira, Portugal

³ Cardiology Department, Centro Hospitalar Entre Douro e Vouga, Santa Maria da Feira, Portugal

* Corresponding author: Tiago Teixeira, Cardiology Department, Centro Hospitalar Entre Douro e Vouga, Santa Maria da Feira, Portugal. E-mail: tiagoteixeiranoc@gmail.com

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Abstract

Cardiac tumors can be primary or metastatic, the latter being more frequent and usually of pulmonary or hematologic origin. These patients' clinical signs are non-specific and the electrocardiogram (ECG) can assume many patterns, among which, ST-segment elevation. Nevertheless, associated occlusion of the coronary arteries is rare in these situations.

We present a 79-year-old woman with a history of pulmonary neoplasia who was admitted to the emergency department due to prolonged atypical chest pain, cough and worsening dyspnea in the previous 3 days. The ECG revealed an ST-segment elevation in the lateral leads, despite normal blood work, with only residually elevated troponin I. Due to the disparity between the patient's symptoms and the ECG findings, a decision was made not to proceed to primary angioplasty, but to further investigate with echocardiography, which revealed a mass localized in the anterolateral and inferolateral left ventricle walls, confirmed by computed tomography. The patient was admitted in the medical ward for symptomatic management. Her clinical condition gradually deteriorated due to the disease's natural evolution and she died two weeks later.

This case highlights the importance to keep in mind differential diagnoses to acute coronary syndromes, when a ST-segment elevation is encountered on a ECG.

INTRODUCTION

The electrocardiogram (ECG) is one of the most frequently used diagnostic exams in the medical daily basis. Despite being broadly available for more than a century, its composition remains unchanged, as does its place as a first line diagnostic exam in the approach of many cardiac diseases, being irreplaceable in the evaluation of thoracic pain syndromes. However, like in every diagnostic exam, the clinical context must never be dismissed, as many situations other than acute myocardial infarction (normal variant, pericarditis, hyperkalemia, and pulmonary embolism) may cause ST-segment elevation [1, 2]. This clinical interpretation, being decisive for the immediate treatment of the ST-segment elevation myocardial infarction (STEMI), becomes of paramount importance in hospitals without conditions for primary angioplasty. Even though it's a rare presentation, tumoral myocardial invasion may be responsible for many ECG abnormalities including bradycardia, atrial fibrillation, atrial or ventricular extrasystoles or even ST-segment elevations, the latter mimicking a STEMI [3, 4].

CASE REPORT

A 79-year-old, caucasian woman, partially dependent on her activities of daily life due to physical deterioration, but with preserved cognition, presented to the emergency department. She complained of 3-day worsening dyspnea, nonproductive cough and right sided thoracic pain radiating to the right shoulder. The pain had an intensity of 3 in 10, had no aggravating or improving factors and was described as a persistent pressure on the right side of the chest that had had few or none free from any pain moments during this period. She had a history of arterial hypertension and stage IIIB undifferentiated big cell lung cancer (diagnosed 8 years before), under palliative care as the patient had refused chemotherapy. She was medicated with Paracetamol/Codeine 500 + 30 mg, Furosemide 40 mg, Ramipril 2.5 mg and Bromazepam 1.5 mg. On admission, she was alert and fully oriented, her blood pressure was 111/84 mmHg, pulse 155 beats per minute, temperature 36.7°C, oxygen saturation 94% without supplementary oxygen, and with a respiratory rate of 23 breaths

per minute. There were bilateral basal crackles on the lungs, and the heart sounds were normal. The arterial blood gas revealed a respiratory alkalosis (pH 7.53, pCO₂ 29, pO₂ 64, HCO₃- 24.2, SatO₂ 94%) and the ECG a sinus tachycardia with a 2.5 mm ST-segment elevation on leads DI, DII, aVL and V5-V6, without Q waves, and reciprocal ST-segment depression on leads DIII, aVR and V1-V3 (Fig 1).

Considering the relative discordance between patient's symptoms and the ECG findings, on top of the patient's general condition, a decision was made not to proceed to primary angioplasty, but to gather further information. Transthoracic echocardiogram showed a mass with a maximal thickness of 21 mm, localized in the antero-lateral and infero-lateral walls (Figs 2 and 3), rendering them akinetic, without other segmental contractility abnormalities, but already originating moderately depressed left ventricular function. Blood analysis identified a normocytic normochromic anemia, leukocytosis with neutrophilia, increased C reactive protein, hyponatremia, and increased D-dimers and troponin I (Table 1). A computed tomography (CT) scan (requested to exclude pulmonary thromboembolism) confirmed the presence of a pericardial mass involving the left ventricle (Fig 4), and showed signs of pulmonary distal pulmonary embolism as well as a bilateral pleural effusion, multiple atelectasis and nodular lesions suggesting neoplastic dissemination and destruction of the costal arches.

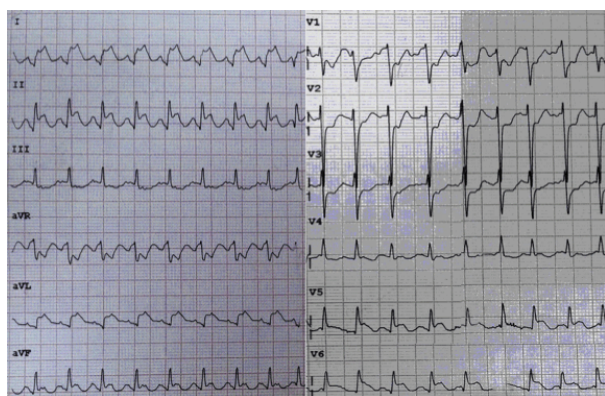


Figure 1: Presentation Electrocardiogram Showing ST-segment Elevation in the Lateral Leads, without Clear Q waves, and Reciprocal ST-segment Depression in the Antero-septal Leads.

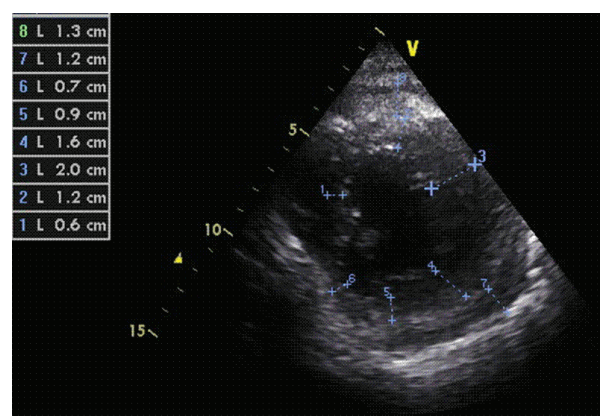


Figure 2: Echocardiographic Parasternal Short Axis View at the Left Ventricle Mid Level, with Wall Thickness Measured on AHA Segments from Antero-septal (1) to Infero-septal (6). Despite low image quality, it is visible asymmetrical hypertrophy in the lateral (3) and posterior (4) walls. Mild pericardial effusion (12 mm).

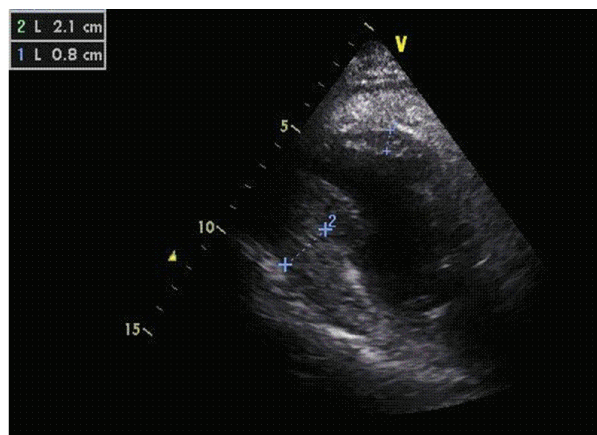


Figure 3: Echocardiogram in the Parasternal Long Axis View. Confirmation of significant hypertrophy of the posterior wall, reaching 21 mm (2).

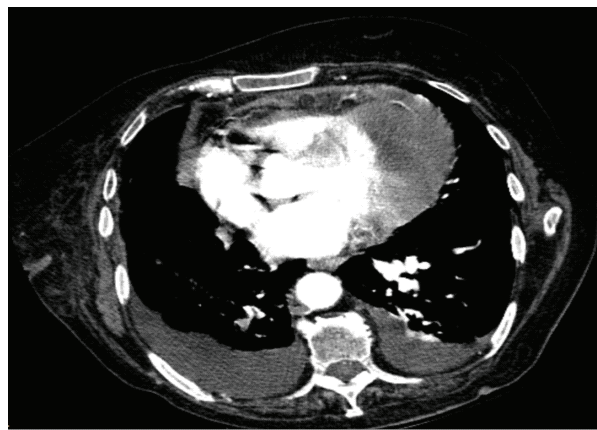


Figure 4: Thoracic Computed Tomographic Scan, requested on the diagnosis doubt of concomitant pulmonary thromboembolism, showing a pericardial mass involving the left ventricle, along with bilateral pleural effusion.

During a 24-hour period of vigilance in the emergency department no increase in troponin I was documented. Afterwards, the patient was admitted to a medical ward for symptomatic control, with the diagnosis of left ventricle invasion by lung carcinoma, pulmonary embolism, upper respiratory tract infection and a probable syndrome of inappropriate secretion of antidiuretic hormone. The pain was controlled with intravenous morphine, the infection treated with Amoxicillin/Clavulanic acid, the heart rate controlled with Carvedilol 12.5 mg every 12 hours and hipocoagulation pursued with enoxaparin. The ECG pattern remained unchanged for the duration of the admission. Despite all measures, the patient's clinical condition progressively deteriorated, and she died 2 weeks after admission.

DISCUSSION

Seventy-five percent of primary cardiac tumors are benign, mixoma being the most frequent [5]. Malignant tumors are majorly secondary, originating in the lung, breast and hematological system, being their composite about 20 times more frequent than primary Malignant cardiac tumors [6, 7]. Pulmonary mesothelioma and melanoma have a particular

Table 1: Resumed Blood Work		
Blood Work	Result	Reference Values
Hemoglobin g/dL	10.6	12.0 - 16.0
Leucocytes (mm ³)	26.1	4.0 - 11.0
Neutrophils (mm ³)	23.0	1.8 - 8.0
Sodium (mmol/L)	128	136 - 145
D-dimers (ng/mL)	1600	< 230
Troponin I (ng/mL)	0.254	< 0.012
Myoglobin (ng/mL)	67.4	< 140
B Natriuretic Peptide (ng/mL)	1425	< 100
Creatinine Kinase (U/L)	21	29 - 168
C Reactive protein (mg/L)	117	< 5.0

tendency for cardiac invasion [8]. The pericardium is the most frequently involved site of invasion, being the myocardium and endocardium progressively rare [8]. This involvement, by metastatic cancer, can occur through lymphatic, hematologic or direct invasion and is indicative of a poor short term prognosis [6].

Patients' clinical presentation is normally with indolent dyspnea, cough and thoracic pain resembling stable angina, and about 50% of patients will have the diagnosis of malignancy only when the cardiac symptoms ensue [4]. The ECG may show bradycardia, atrial fibrillation, T wave inversion, bundle branch block or low voltage, among others. However, the most characteristic pattern in cardiac metastatic cancer is a persistent ST-segment elevation without Q waves [2, 4, 9]. Different pathophysiologic explanations have been proposed for the ECG changes, like inflammatory response, ionic transfer of potassium from necrotic tissue to the adjacent myocardium or muscle fibers overstretching. However no consensus has been reached [4]. Occlusion of a coronary artery is very rare though, and has no role in the repolarization changes [3]. In our case, the patient had already a known neoplasia and a non-acute thoracic pain with associated dyspnea. Her ECG was compatible with the classic pattern associated with cardiac tumor invasion and, as expected, remained immutable throughout the hospitalization. Despite the exact mechanism responsible for the ECG changes being unclear, those changes seem to be dependent upon the tumor's location. Suga T et al. reported that in cases of posterolateral, anterolateral or apical/anterolateral invasion, the ST-segment elevation is present in the lateral and precordial leads, respectively [4]. In his report only one patient had diffuse pericardial metastases affecting all cardiac chambers, having symptoms of constrictive pericarditis and diffuse precordial ST-segment elevation. Our patient's ECG shown ST-segment elevation in DI, DII, aVL and V5-V6, congruent to the location of the tumor invasion and in concordance with usual findings [4]. The echocardiogram and the CT were the chosen methods to determine the tumor's location and delineate its margins. Echocardiogram's noninvasiveness and availability make it an excellent first line exam to characterize the tumor's morphology and cardiac functional compromise [10]. The CT and magnetic resonance have further therapeutic implications, as they stage the neoplasia [10]. The reported mortality of this situation is high, at a reported 50% short-term mortality (2 to 3 months) [4]. Other reviews did not report outcomes.

Despite being rare, secondary cardiac tumor involvement produces characteristic ECG changes. When confronted with ST-segment elevation, a clinic must confirm the presence of an acute coronary syndrome, without automatically dismiss other differential diagnosis. In cancer patients with this ECG pattern and atypical thoracic pain, cardiac metastization should be considered among the diagnostic possibilities, considering the immediate therapeutic implications.

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