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The importance of the differential diagnosis between pulmonary thromboembolism and acute coronary syndrome

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ABSTRACT

Introduction: Pulmonary thromboembolism (PTE) occurs when there is an obstruction of one of the pulmonary arteries, thus preventing normal blood circulation. Pulmonary thromboembolism is often associated with various diseases. Clot formation results from hypercoagulability, blood stasis (reduced blood flow) and damage to the endothelium of blood vessels, a set of alterations known as Virchow's triad. Objectives: To understand the role of the electrocardiogram in the diagnosis of pulmonary thromboembolism in an emergency setting. Clinical Case: A 79-year-old female patient with a personal history of chronic bronchitis came to the Emergency Department with dyspnea, chest pain, a cough that was not very productive and fatigue on slight exertion. An ECG revealed sinus rhythm with profound inversion of the T wave in V1-V4. The cardiologist was contacted and confirmed the diagnosis of subacute coronary syndrome. In order to complement the diagnosis, a transthoracic echocardiogram was carried out, which revealed a rectified movement of the interventricular septum in relation to right-sided overload and severe pulmonary hypertension, echocardiographic findings suggestive of pulmonary thromboembolism. Conclusion: In conclusion, although the S1Q3T3 pattern is the best known for diagnosing PTE, it is not always present on the ECG. It is therefore necessary to raise awareness of other electrocardiographic findings present in this condition.

Keywords: Pulmonary thromboembolism, Electrocardiography, Acute coronary syndrome.

INTRODUCTION

Pulmonary thromboembolism (PTE) occurs when there is an obstruction of one of the pulmonary arteries, thus preventing normal blood circulation. Pulmonary thromboembolism is often associated with several diseases. Clot formation results from hypercoagulability, blood stasis (decreased blood flow) and damage to the endothelium of blood vessels, a set of changes known as Virchow's triad. The diagnosis of PTE can be tricky, since common clinical signs such as tachypnea, dyspnea, and hypoxemia are not specific to the disease. Initial evaluation of PTE should include arterial blood gas analysis and chest x-ray. When there is a strong clinical suspicion of PTE, specific additional tests are indicated, such as measurement of the biological marker D-Dimer, thromboelastography, and more advanced imaging tests.

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In acute coronary syndrome, the electrocardiogram (ECG) provides important information about the presence, extent, and severity of myocardial ischemia. Sometimes the changes are typical and clear. In other cases, the changes are subtle and can be recognized only when the ECG recording is repeated after changes in symptom severity. ECG interpretation is a crucial part of the initial evaluation of patients with suspected symptoms of myocardial ischemia, along with medical history and other specific tests such as echocardiography. One of the equivalents of acute myocardial infarction is called *Wellens syndrome*. This syndrome represents the pre-infarction period involving the proximal left anterior descending artery, which can lead to extensive anterior myocardial infarction and even death if there is no intervention quickly.

The importance of cardiopulmonary technicians and cardiologists in the interpretation of electrocardiograms is well known, especially in differential diagnoses so that decisions can be made faster, and more complications can be avoided.

OBJECTIVE

To understand the role of the electrocardiogram in the diagnosis of a pulmonary thromboembolism in context of urgency.

CLINICAL CASE

A 79-year-old female patient with a personal history of chronic bronchitis was admitted to the Emergency Department due to dyspnea, chest pain, unproductive cough and fatigue on minor exertion. She reported chest pain that improved after rest without interference in her daily life, in the 4 days before being admitted to the emergency room the pain worsened, with radiation to the neck. An ECG revealed sinus rhythm with deep inversion of the T wave at V1-V4 (Figure 1). Analyses were performed, where a troponin value higher than normal was verified, but due to its value being less than 1000, acute myocardial infarction was not diagnosed. When the cardiologist who confirms the diagnosis of Coronary Syndrome in the subacute phase is contacted, he proposes the patient for coronary angiography, which does not perform it, because in order to complement the diagnosis, a transthoracic echocardiogram was performed where the existence of a rectified movement of the interventricular septum in relation to right overload was verified (*Dshape*) and severe pulmonary hypertension (94 mmHg), echocardiographic findings suggestive of pulmonary thromboembolism (Figure 2 and Figure 3). After the echocardiogram, the patient also underwent a transthoracic computed tomography (CT) scan, which confirmed signs of acute central and bilateral pulmonary thromboembolism, with repletion defects at the distal level of the right and left arteries and extending to the various lobar branches of both lungs, and finally other clinical analyses were performed that demonstrated elevated dimers (>3000).

Figure 1 – Electrocardiogram performed in an emergency context.

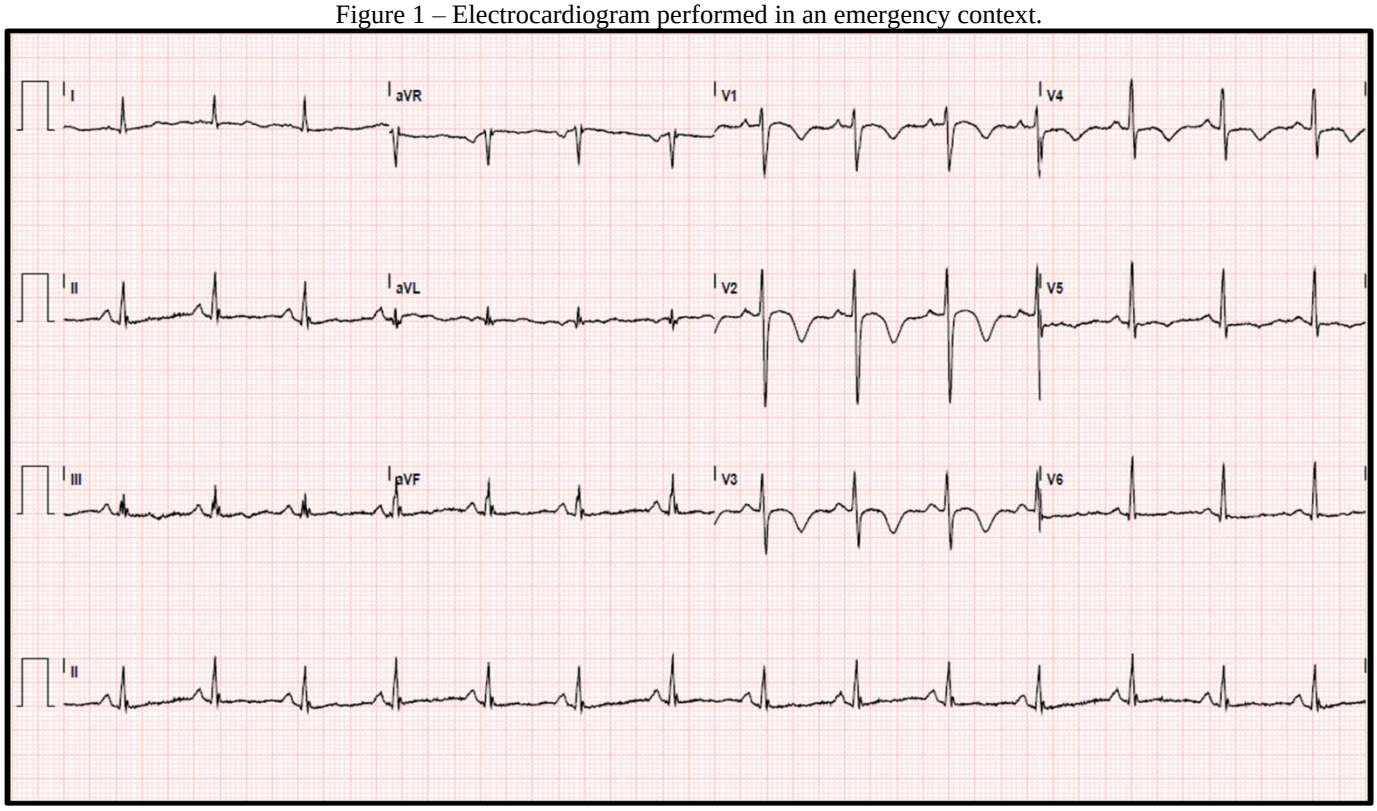


Figure 2 – Echocardiogram, measurement of pulmonary artery systolic pressure (PASP).

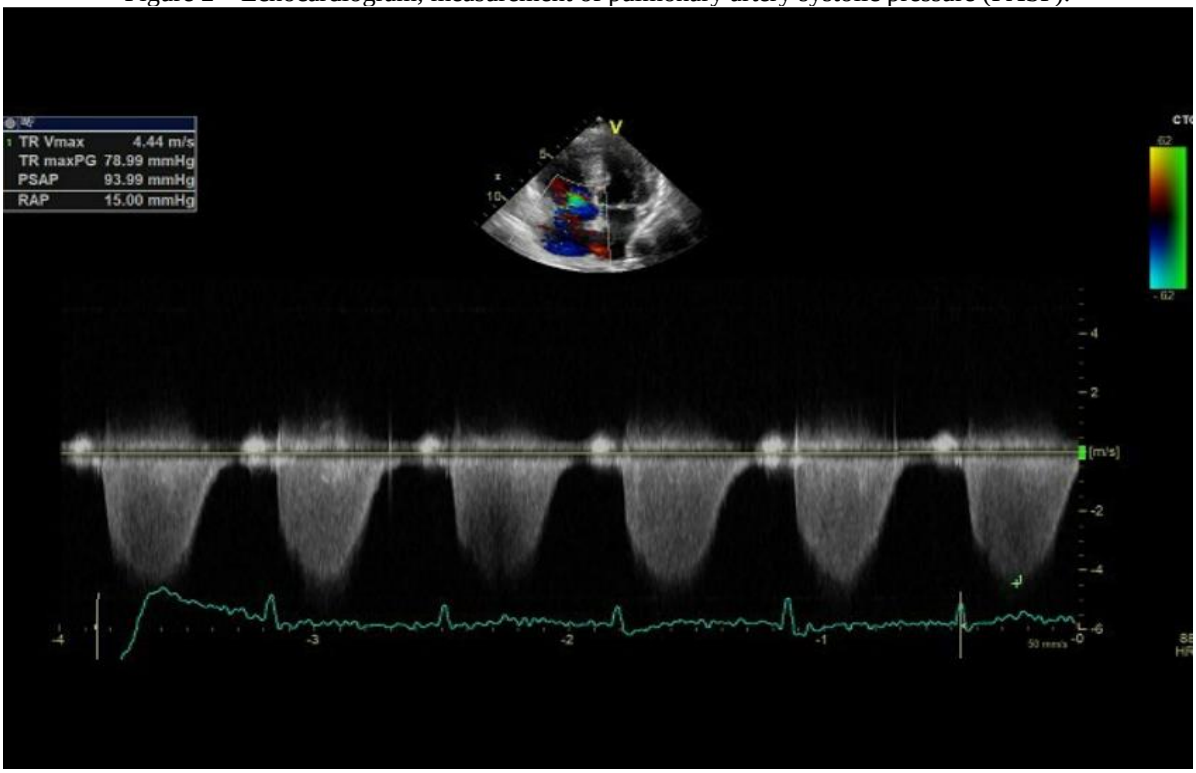
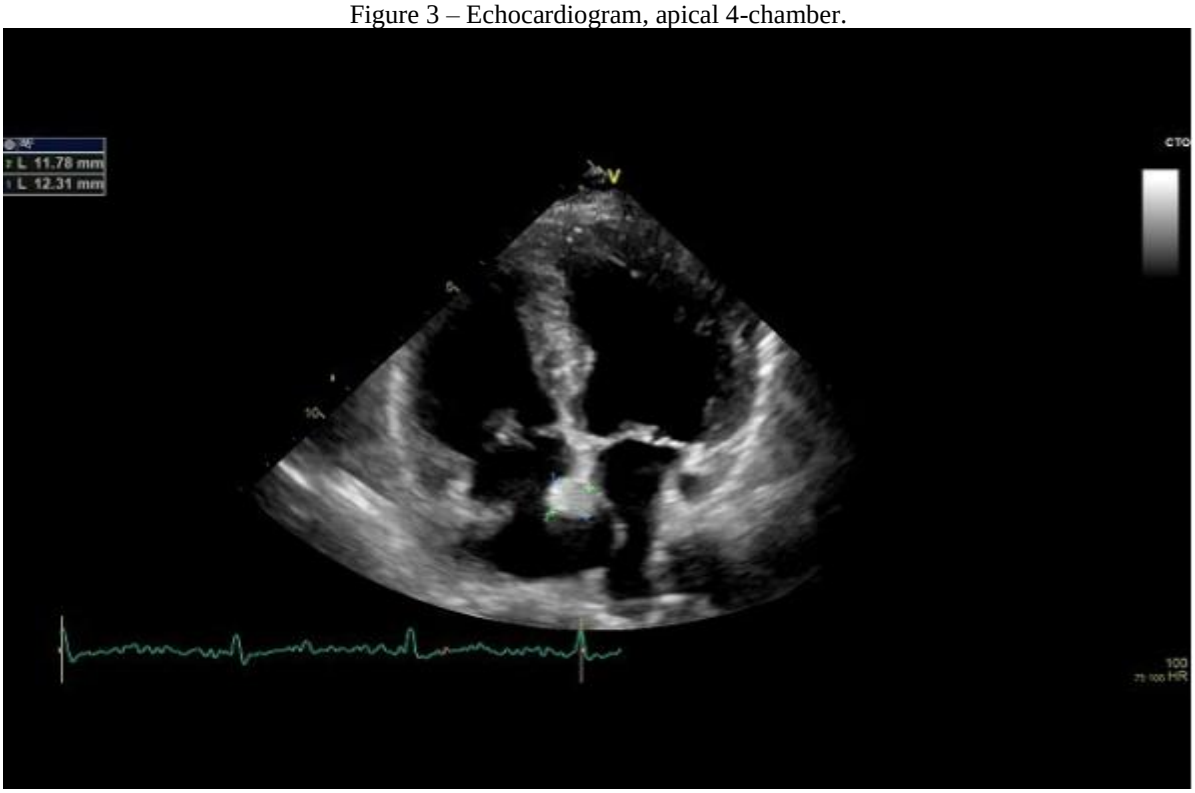


Figure 3 – Echocardiogram, apical 4-chamber.



DEVELOPMENT

Electrocardiographic anomalies are common in pulmonary thromboembolism, with sinus tachycardia being one of the most frequent, but not specific to PTE. According to several studies, the ECG is abnormal in more than 70% of patients with PTE, with sinus tachycardia being the most prevalent anomaly. There are other ECG findings that are frequently related to the impact of PTE on the pressure and function of the right ventricle and the right atrium, namely, right bundle branch block (RBB), wide R wave in V1, right axis deviation, T wave inversion in V1-V4 and P-pulmonary and/or atrial arrhythmias6-9. The combination of an S wave in DI, a Q wave and an inverted T wave in DIII forms the S1Q3T3 pattern, is suggestive of right ventricular overload and although this pattern is the best known in the diagnosis of PTE it is only present in 3.7% of patients, however the electrocardiographic characteristic with the highest incidence in cases of PTE is the inversion of the T wave. found in about 17% to 68% of cases3.

Although all of these ECG findings are quite important, they are not specific to a diagnosis of PTE. A study conducted in the 2000s demonstrated 30 different ECG abnormalities in 246 patients with suspected PTE and concluded that tachycardia and incomplete right bundle branch block (BIRD) were the only significantly more common ECG findings in patients with confirmed PTE.

The positive predictive value of tachycardia was 38%, while that of IBRD was 100%. However, acute changes in right ventricular overload may be observed in other pathologies, such as pneumonia or

acute exacerbation of chronic obstructive pulmonary disease. After several studies, it has been observed that the electrocardiographic abnormalities present in patients with PTE return to normal after treatment, and this normalization of ECG abnormalities after treatment is a positive indicator, suggesting a recovery of cardiac function and a decrease in pressure in the right ventricle.

Patients with ST-segment elevation on the electrocardiogram and symptoms consistent with ischemia/acute myocardial infarction should be referred to the hemodynamics unit. However, it is important to highlight that a considerable number of patients may have ST-segment elevation without having acute coronary syndrome.

ST-segment depression with positive T waves is increasingly recognized as a sign of subendocardial ischemia associated with severe obstruction of the left anterior descending coronary artery. Generalized ST-segment depression, often associated with inverted T waves and ST-segment elevation in the aVR lead during episodes of chest pain, may represent diffuse subendocardial ischemia caused by severe coronary artery disease.

Wellens syndrome is characterized by T-wave changes on the electrocardiogram during the pain-free period in a patient with intermittent angina. This syndrome contains significant diagnostic and prognostic value, as it represents a pre-infarction period involving the proximal left anterior descending artery, which can cause anterior myocardial infarction and even death without coronary angioplasty. Since the treatment of PTE fundamentally involves anticoagulants, such as heparin, and acute coronary syndrome can be treated through angioplasty, the differential diagnosis between both pathologies is crucial, in order to have a good management of human resources, materials, transport of patients and additional treatments.

Heparin accelerates the action of antithrombin III, prevents the further formation of fibrinolysis thrombi, and allows the clot to dissolve. Heparin also promotes the endothelialization of the thrombus and decreases the likelihood of its embolization. Patients with suspected PTE should start taking anticoagulant drugs quickly and intensively.

Therefore, it is crucial that all physicians and cardiopulmonary technicians recognize all the electrocardiographic characteristics that this syndrome transmits so that immediate interventions can be taken to try to reduce the mortality and morbidity of acute myocardial infarction5.

FINAL THOUGHTS

In conclusion, although the S1Q3T3 pattern is the most well-known for diagnosing PTE, it is not always present on the ECG. Therefore, it is necessary to sensitize to other electrocardiographic findings present in this condition. In the clinical case presented, the ECG showed alterations in ventricular repolarization, namely inversion of T waves from V1 to V4, characteristics that could be present in the

context of an Acute Coronary Syndrome. Although these alterations may have an ischemic etiology, with the use of different diagnostic means such as echocardiography, it was possible to make the differential diagnosis of PTE.

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