Parapneumonic ST segment elevation

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SOUHRN

Přítomnost elevace úseku ST na 12svodovém EKG záznamu pacienta se symptomy připomínajícími infarkt myokardu musí vyvolat úsilí o rychlé stanovení diagnózy infarktu myokardu s elevacemi úseku ST a následnou urgentní intervenci s cílem obnovit perfuzi koronárních tepen, obvykle formou perkutánní koronární intervence. Akutní infarkt myokardu nebo obraz elevací úseku ST na elektrokardiogramu však může ukazovat na několik různých onemocnění a stavů. Popisujeme klinický případ pacienta hospitalizovaného nejdříve s diagnózou pneumonie, u něhož došlo na EKG záznamu *de novo* k elevacím úseku ST naznačujícím anteroseptální infarkt myokardu, který však zároveň uváděl bolest připomínající transmurální infarkt myokardu. Urgentní koronarografie vyloučila ischemickou chorobu srdeční a další vyšetření vedla ke stanovení nečekané diagnózy.

Keywords:
Differential diagnosis
Electrocardiogram
Pleural effusion
Pneumococcal pneumonia
ST-segment elevation

ABSTRACT

The presence of ST-segment elevation on the 12-lead electrocardiogram in a patient with suggestive symptoms of myocardial infarction should trigger an early diagnosis of ST-segment elevation myocardial infarction and urgent intervention to restore coronary perfusion, usually by percutaneous coronary intervention. However, several diseases and conditions can mimic an acute myocardial infarction or the ST-segment elevation pattern on the electrocardiogram. We report a clinical case of a patient hospitalized with the first diagnosis of pneumonia, who developed a de novo anteroseptal ST-segment elevation on electrocardiogram and clinical pain suggestive of transmural myocardial infarction. Emergent coronary angiography excluded coronary heart disease, and further investigation led to an unexpected final diagnosis.

Introduction

ST-segment elevation myocardial infarction (STEMI) is an acute manifestation of coronary artery disease and is associated with significant morbidity and mortality. In the clinical scenario of ischemia, the presence of ST-segment elevation (STE) in the 12-lead electrocardiogram (ECG) should trigger a prompt STEMI diagnosis and rapid and successful revascularization, usually by percutaneous coronary intervention. However, several diseases and conditions can mimic an acute myocardial infarction or the STE pattern on ECG.[1–3] Here, we describe a case of unusual ST segment elevation in a patient admitted with a first diagnosis of pneumonia.

Case report

A 64-year-old male with heavy smoking habits was admitted with global respiratory failure and obnubilation. There was no other relevant medical history, including other cardiovascular risk factors. On physical examination, the patient was normotensive, with normal cardiac auscultation, febrile (axillary temperature of 38.3 °C), presenting rapid and shallow breathing, and dependent on oxygen inhalation by nasal catheter to achieve optimal peripheral oxygen saturation. Pulmonary crackles on the left hemithorax and diminished vesicular murmur were heard on pulmonary auscultation. Laboratory analysis of the patient's blood showed neutrophilic leukocytosis, elevated inflammatory markers, and normal renal and hepatic function. An initial ECG revealed *sinus* tachycardia (Fig. 1), and a chest X-ray showed hypotransparency in the lower half of the left hemithorax, suggestive of pleural effusion and passive pulmonary atelectasis, and small bilateral cotton-wool opacities (Fig. 2). The diagnosis of pneumococcal pneumonia was done, and the patient was placed under empirical antibiotic therapy with amoxicillin/clavulanic acid and clindamycin.

On the day two of admission, due to *de novo* abdominal pain and tachycardia (140–150 beats per minute [bpm]), an ECG was requested, revealing atrial fibrillation with a rapid ventricular response (140 bpm), a *de novo*

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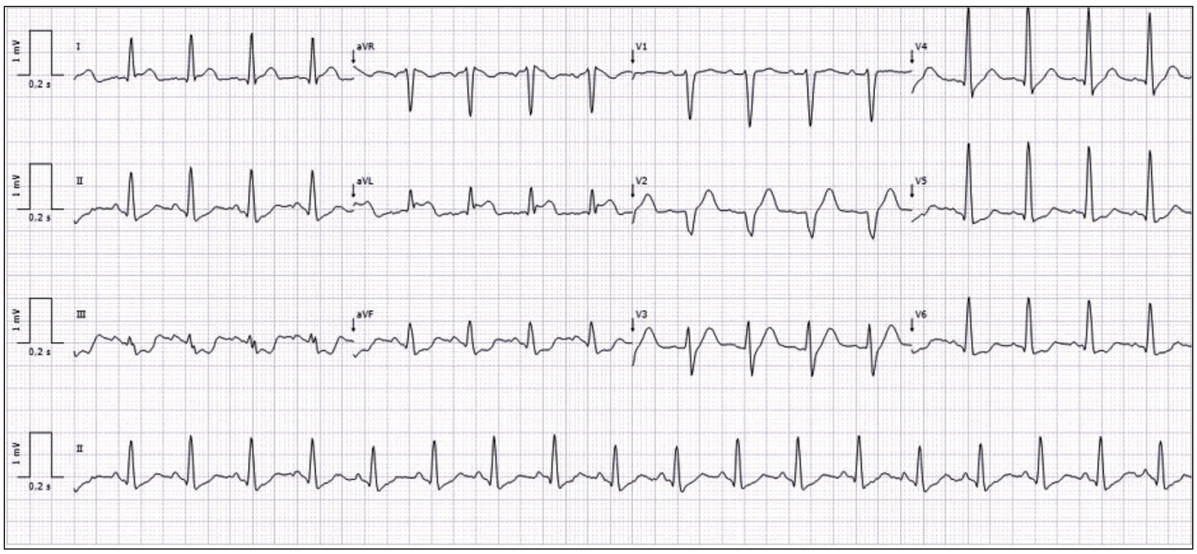

Fig. 1 – Initial electrocardiogram: sinus tachycardia, 110 beats per minute, nonspecific changes in ventricular repolarization.

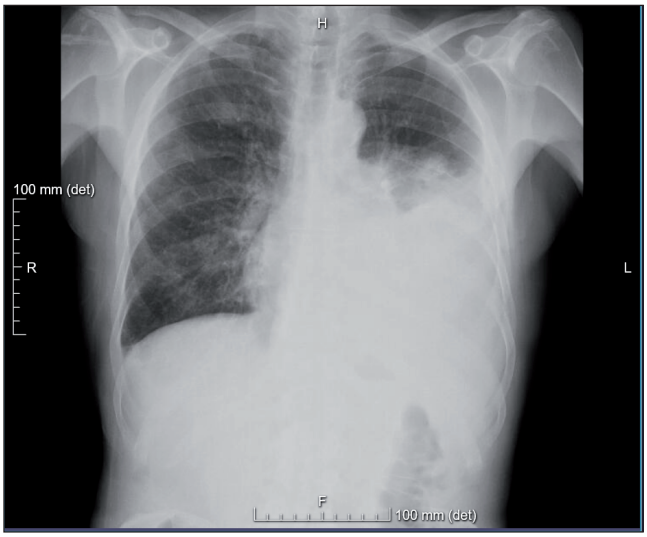

Fig. 2 – Chest X-ray: hypotransparency in the lower half of the left hemithorax, suggestive of pleural effusion and passive pulmonary atelectasis, and small bilateral cotton-wool opacities.

ST-segment elevation in leads V_1 to V_3, and poor R-wave progression in the right precordial leads (Fig. 3). By suspicion of anteroseptal myocardial infarction, antitrombotic drugs (600 mg of clopidogrel, 300 mg of acid acetylsalicylic and 5 000 units of non-fractionated heparin) were administrated and an emergent coronary angiography was performed that excluded coronary heart disease. A later abdominal echo was normal, with the exception of hepatic steatosis and repeated troponin values were normal, thus, acute coronary syndrome was excluded.

A bedside echocardiogram was performed showing preserved global systolic function of both ventricles, absence of wall motion abnormalities, valvulopathies or pericardial effusion. This exam also revealed on its pulmonary window a large loculated pleural effusion adjacent to the pericardium with thickening and hyperecogenicity of the pleura and pericardium beneath (Fig. 4 and Video 1).

Amiodarone infusion was initiated in order to achieve rhythm control and the *de novo* STE ECG alterations were interpreted as secondary to localized inflammation of the

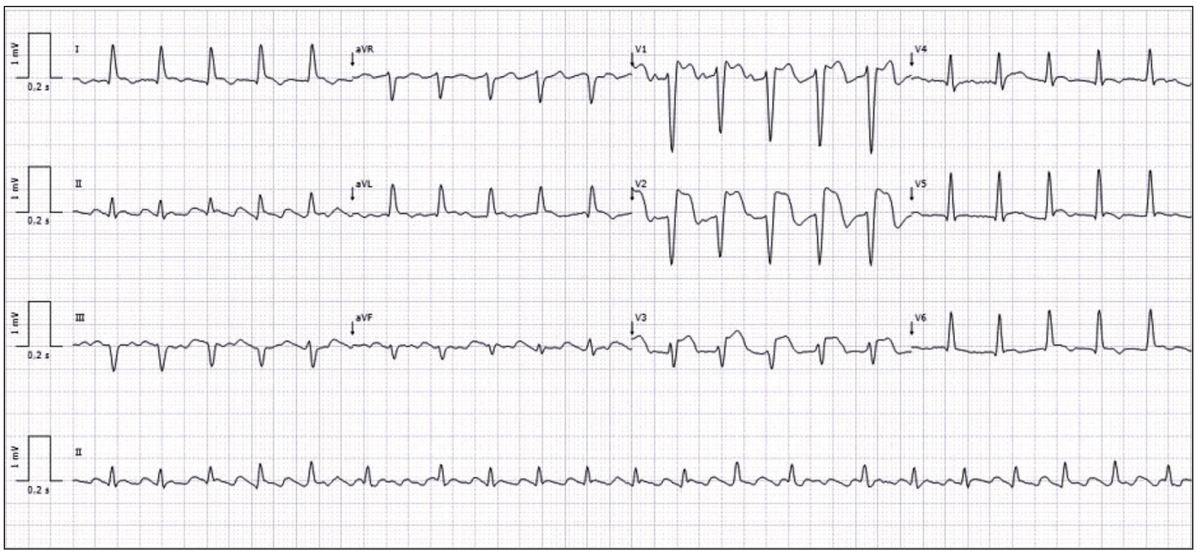

Fig. 3 – Fibrillation with a rapid ventricular response (140 beats per minute), ST-segment elevation in leads V_1 to V_3 and poor R-wave progression in the right precordial leads.

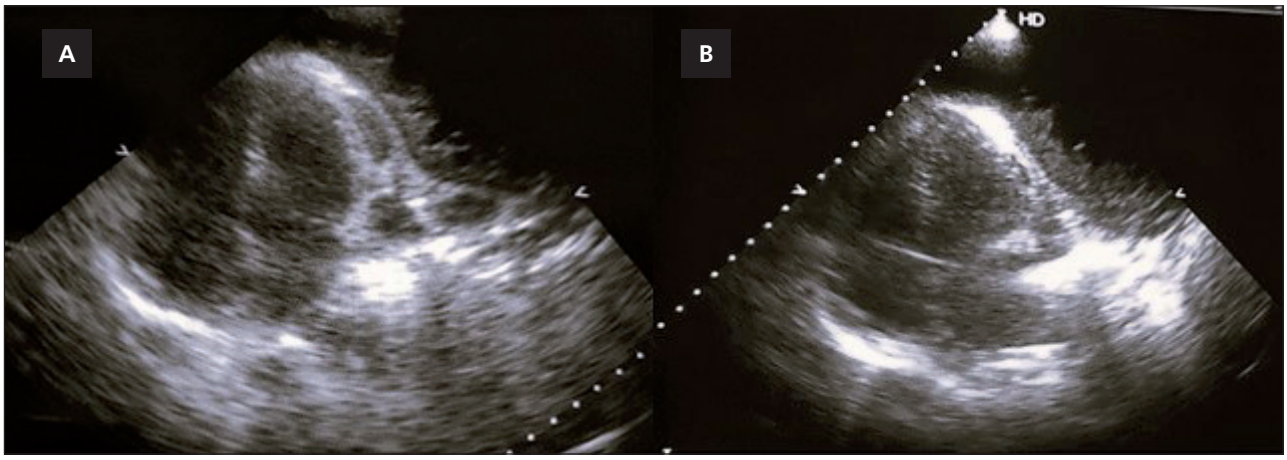

Fig. 4 – Bedside echocardiogram 4-chamber view (A and B): large loculated pleural effusion adjacent to the pericardium with thickening and hyperechogenicity of the pleura and pericardium.

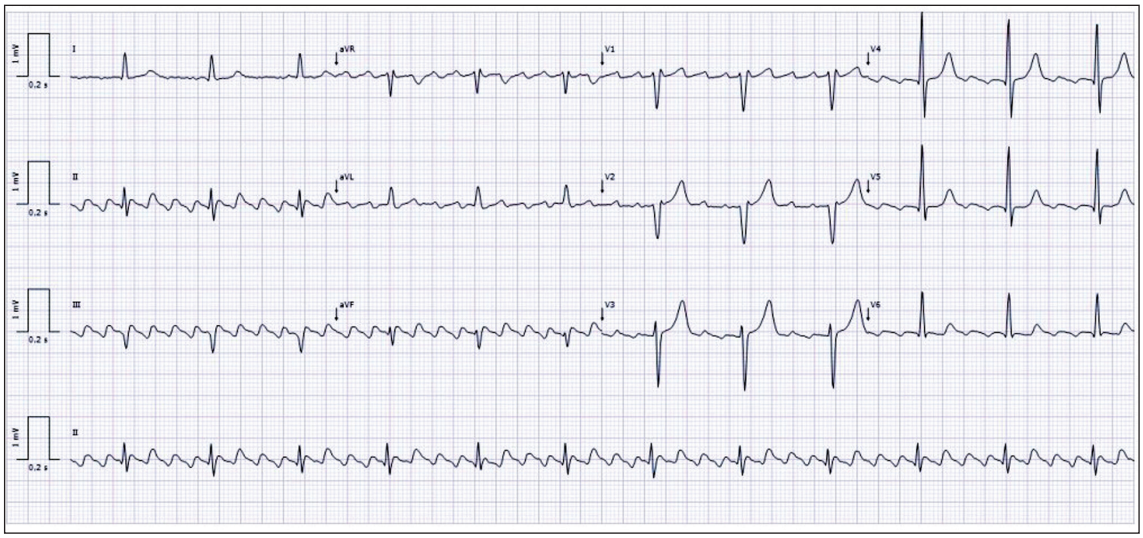

Fig. 5 – Atrial flutter 75 bpm; with complete resolution of ST segment elevation.

pericardium by direct contact with a complicated parapneumonic pleural effusion.

After 18 days of antibiotic therapy and thoracocentesis to drain the loculated pleural effusion, the patient achieved favorable clinical, analytical, and radiologic outcomes, being discharged fully recovered. On the last ECG, the STT alterations had resolved (Fig. 5).

Discussion

In case of a suspected STEMI diagnosis, reperfusion therapy with primary coronary intervention to restore the coronary blood flow to the myocardium is essential to save cardiac tissue and decrease mortality and morbidity. The reperfusion therapy must be offered promptly and without delay, even before blood analysis or other complementary exams, keeping with the maxim "time is myocardium".[1–3]

In this clinical case, epigastric pain (a possible anginal equivalent) and *de novo* electrocardiographic changes were compatible with an anteroseptal STEMI (new STE in V_1–V_3, ≥2 mm amplitude, in contiguous leads). An emergent coronary angiography with the objective of restoring myocardial perfusion was performed accordingly with present recommendations in acute care of STEMI patients. Once coronary disease was excluded, other causes of STE on ECG were investigated.

Many conditions other than acute myocardial infarction can cause STE on an ECG, mimicking an acute myocardial infarction. The differential diagnosis of STE includes vasospastic angina, left ventricular hypertrophy, left bundle branch block, acute pericarditis, early repolarization, preexcitation pattern, hyperkalemia, pulmonary embolism, and Brugada syndrome. The clinical setting and cardiac imaging diagnostic workout often allow identification of the cause. Specific electrocardiographic criteria and morphologies are decisive clues for the differential diagnosis.[3,4]

Normally, the ST segment on an ECG represents an electrically neutral area of the complex between ventricular depolarization (QRS complex) and repolarization

(T wave). During acute ischemia, cardiac cells are unable to maintain the physiologic ion gradient across the cell membranes, resulting in an earlier cellular depolarization with transmural ischemia. That is recorded as an injury current and STE on an overlying recording electrode between the boundaries of the normal (polarized) and injured tissue (depolarized).[5,6]

Subepicardial cells are near the ventricle surface and local inflammatory conditions at the subepicardial level will provoke altered depolarization and can produce STE in the leads facing the inflammation.

General pericardial inflammation in acute pericarditis provokes STE as a result of abnormal repolarization. The typical features are an upwardly concave STE configuration (against convex appearance in STEMI) and a diffuse STE pattern not corresponding to any specific arterial territory. In these cases, there are no reciprocal ST-T changes, Q waves or T-wave inversion, which classically occurs with acute myocardial infarction. Loss of R-wave progression may occur with acute myocardial infarction, but this feature is not present with acute pericarditis.[6,7]

In this case, the STE was interpreted as secondary to localized inflammation of the pericardium by the direct contact with a complicated parapneumonic pleural effusion, by a mechanism similar to acute pericarditis, which results in a STE pattern very suggestive of STEMI and limited to a certain territory (anteroseptal STE).

Recent data suggest a rate of myocardial infarction of 7 to 8% among patients who were hospitalized for pneumococcal pneumonia, including type 1 (acute coronary occlusion related to atherosclerotic plaque disruption and superimposed thrombosis, triggered by the proinflammatory, prothrombotic and procoagulant state and endothelial dysfunction) and type 2 (when the metabolic demands of myocardial cells exceed the capacity of the blood to supply oxygen to the cells) myocardial infarction. The risk of myocardial infarction associated with pneumonia peaks at the onset of infection and is proportional to the severity of the illness. Most studies of acute infection have not distinguished between type 1 and type 2 myocardial infarction, but demand ischemia should explain only a small proportion of infection-related myocardial infarction events that occur in the short-term postinfection period and none beyond that period.[8–10]

In the presence of a possible clinical scenario of ischemia (epigastric pain), de novo STE that fulfilled STEMI ECG criteria, and acknowledging that severe proinflammatory states could trigger myocardial infarction, the invasive strategy with urgent coronariography was the correct one.

Conclusion

In conclusion, the clinical context is preponderant for the diagnosis of acute myocardial infarction with ST-segment elevation and not all cases of ST elevations necessarily reflect transmural infarction. In some cases, it can occur in a nonischemic context, as in the case of localized inflammation above the epicardial surface like in complicated parapneumonical effusion.

Conflict of interest
No conflict of interest.

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Supplementary material
The supplementary material is available online.

References

1. Asatryan B, Vaisnora L, Manavifar N. Electrocardiographic Diagnosis of Life-Threatening STEMI Equivalents: When Every Minute Counts. JACC Case Rep 2019;1:666–668.
2. de Bliek EC. ST elevation: Differential diagnosis and caveats. A comprehensive review to help distinguish ST elevation myocardial infarction from nonischemic etiologies of ST elevation. Turk J Emerg Med 2018;18:1–10.
3. Wang K, Asinger RW, Marriott HJL. ST-segment elevation in conditions other than acute myocardial infarction. N Engl J Med 2003;349:2128–2135.
4. Akar JG, Akar FG. Regulation of ion channels and arrhythmias in the ischemic heart. J Electrocardiol 2007;40:S37–S41.
5. Kashou AH, Basit H, Malik A. ST Segment. [Updated 2023 Aug 14]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK459364/
6. Imazio M, Spodick DH, Brucato A, et al. Diagnostic issues in the clinical management of pericarditis. Int J Clin Pract 2010;64:1384–1392.
7. Ramirez J, Aliberti S, Mirsaeidi M, et al. Acute Myocardial Infarction in Hospitalized Patients with Community Acquired Pneumonia. Clin Infect Dis 2008;47:182–187.
8. Musher DM, Abers MS, Corrales-Medina VF. Acute Infection and Myocardial Infarction. N Engl J Med 2019;380:171–176.
9. Kim J, Park SJ, Choi S, et al. Hospitalization for acute coronary syndrome increases the long-term risk of pneumonia: a population-based cohort study. Sci Rep 2021;11:9696.