CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Electrocardiogram Quiz – Case 18

A 68-year-old man with no previous medical history presented with angina pectoris of sudden onset at rest of half an hour duration. The patient's 12-lead surface ECG at the emergency department is depicted in figure 1. The treatment team decided to mobilize the Cath Lab for an emergency coronary arteriography, with ST-segment elevation acute myocardial infarction (STEMI) as the working diagnosis.

Questions

- a. Do you think that the treatment team's decision to mobilize the Cath Lab was wise?
- b. What is the differential diagnosis in the presented patient?

Comment

Pericarditis is a common disease but frequently subclinical. There are a number of causes, including infection, systemic disease, metabolic disease, post-myocardial infarction, medication, neoplasm, and iatrogenic. The most common presentation is chest pain and electrocardiographic findings of diffuse ST-segment elevation. However, on occasion, these changes can mimic a presentation of acute myocardial infarction ARCHIVES OF HELLENIC MEDICINE 2014, 31(4):511–512 ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2014, 31(4):511–512

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(AMI). Therefore, these patients may erroneously receive thrombolytic treatment or undergo an urgent coronary arteriography.

Typically, the acute ECG change in acute pericarditis (AP) is a concave-upward ST-segment elevation in all leads, except aVR and V1. In AMI, this ST-segment elevation has a convex configuration and occurs in leads corresponding to specific coronary artery territories. Further ECG clues to help differentiate AP from AMI include the following: In AP, T-wave inversion usually occurs after ST-segment elevation returns to baseline, while in AMI the former occurs spontaneously with ST-segment elevation. In AP, ST-segment axis ranges from 30 to 60 degrees, whether in AMI from 100 to 120 degrees. In AP, there can







be PR-segment depression (sensitivity 64%), while in AMI there is no PR-segment depression. Finally, in AP there is no evolution of Q-wave, something that could be the case in AMI. Other differential diagnoses of the above ECG patterns include early repolarization and ventricular aneurysm. The ST-segment elevation as a result of early repolarization is more commonly observed in young black males. It is also usually J-point elevation as opposed to the true ST-segment elevation. Moreover, it is persistent and occurs in the absence of chest pain. Further differential diagnoses of ST-segment elevation, with or without clinical syndrome, include: Coronary vasospasm (Printzmetal's angina), left bundle branch block, left ventricular hypertrophy, Brugada syndrome, ventricular paced rhythm, and raised intracranial pressure. Especially Printzmetal's angina causes a pattern of ST-segment elevation that is very similar to STEMI; i.e. localized ST-segment elevation with reciprocal ST-segment depression occurring during episodes of chest pain. However, unlike STEMI, the ECG changes are transient, reversible with vasodilators and not usually associated with myocardial necrosis. It may be impossible to differentiate these two conditions based on the ECG alone.

Interestingly, in our patient's initial ECG, Spodick's sign, described in 1974, was present. The observed TP-segment (or entire QRS-TP) downsloping is suggestive of pericarditis, with an 80% sensitivity. A recent study demonstrated that patients with ST-segment elevation AMI, but not those with AP, show prolongation of QRS-complex and shortening of QT-interval in ECG leads with ST-segment elevation. These new findings may improve the differential diagnostic yield of the classical ECG criteria.

Our patient underwent an uneventful coronary arteriography that revealed normal coronary arteries. The 12-lead surface ECG a few hours after admission is shown in figure 2. Furthermore, cardiac enzymes were within normal limits throughout the patient's hospitalization. A transthoracic echocardiogram was also normal, showing no hypokinetic ventricular wall regions and no pericardial effusion. The patient was prescribed ibuprofen and was discharged with instructions for a repeat transthoracic echocardiogram.

References

- 1. GINZTON LE, LAKS MM. The differential diagnosis of acute pericarditis from the normal variant: New electrocardiographic criteria. *Circulation* 1982, 65:1004–1009
- OMAR HR, FATHY A, RASHAD R, ELGHONEMY M. Acute perimyocarditis mimicking transmural myocardial infarction. *Int Arch Med* 2009, 2:37
- PETROU E, TSIPIS A, BOUTSIKOU M, VARTELA V, COKKINOS DV. Electrocardiogram quiz – case 17: Left ventricular aneurysm (Goldberger's sign). Arch Hellen Med 2014, 31:380–381
- SPODICK DH. Electrocardiogram in acute pericarditis. Distributions of morphologic and axial changes by stages. *Am J Cardiol* 1974, 33:470–474
- ROSSELLO X, WIEGERINCK RF, ALGUERSUARI J, BARDAJÍ A, WORNER F, SUTIL M ET AL. New electrocardiographic criteria to differentiate acute pericarditis and myocardial infarction. *Am J Med* 2014, 127:233–239

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