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Right answer 1: E. All choices should be included

Because of the persisting ST elevation on ECG, the patient was quickly transported to the catheterization laboratory. During catheterization, he still had left forearm pain, but coronary angiography was completely normal. We repeated ECG, and it was completely similar to the first one (Fig. 2). In a quick echocardiographic evaluation, there was no pericardial effusion or localized wall motion abnormality.

Because coronary arteries were normal with co-existing ST elevation on ECG, we suspected vasospastic angina. However, we could not perform intracoronary ergonovine test because we did not have ergonovine in our clinic. Meanwhile, we interrogated the pain characteristics again. He expressed the same localized pain but did not mention about any kind of chest pain.

During the physical examination of left arm, as we compressed the patient's wrist, he described a tingling sensation in his palm. The patient was taken to his bed, and control ECG performed that was normal except early repolarization findings (Fig. 3) despite persisting pain and cardiac markers were normal.

Because of the pain characteristics, we referred the patient to the physical treatment and rehabilitation department, and magnetic resonance (MR) was performed on the left arm. MR images were compatible with carpal tunnel syndrome. What is the final diagnosis of the patient?

- A) Pre-excitation syndrome
- B) Vasospastic angina
- C) Acute coronary syndrome
- D) Cervical disc hernia
- E) Carpal tunnel syndrome+Intermittent low atrial rhythm+Early repolarization

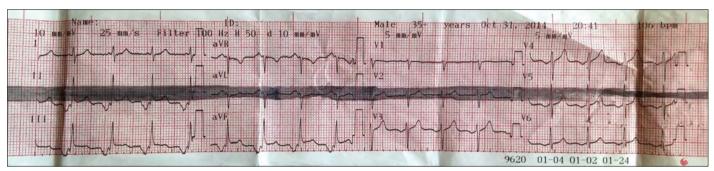


Figure 2. ST-segment elevation and negative P waves in inferior leads (exactly similar to the first ECG)

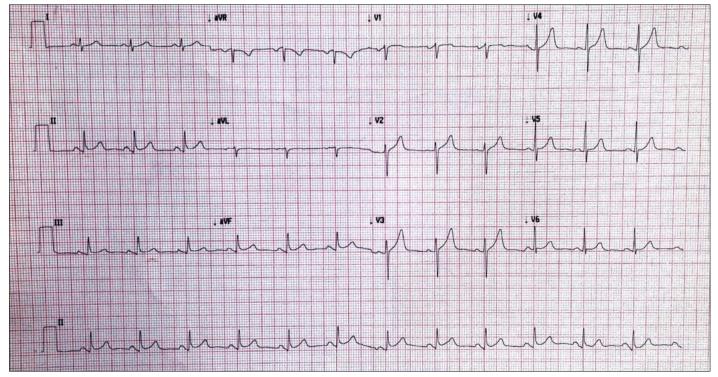


Figure 3. Normal sinus rhythm with early repolarization

Right answer 2: E. Carpal tunnel syndrome+Intermittent low atrial rhythm+Early repolarization

All choices should be included in differential diagnosis according to the first evaluation. However, after detailed evaluation with coronary angiography, electrocardiography, and clinical findings, the most appropriate final diagnosis is carpal tunnel syndrome and pre-existing early repolarization in second question.

If we carefully look at the first 2 ECGs, we can observe negative and prominent P waves, short PR interval (90 msn), and coexisting ST-segment elevation in inferior leads. It is associated with low atrial rhythm ECG findings.

In normal sinus rhythm (SR), P wave represents atrial depolarization. Atrial repolarization (Ta) wave has low amplitude (10–60 μV) and coincide PR segment and QRS complex. Therefore, it is generally not observed in normal ECG. However, in the case of second or third degree atrioventricular block, Ta wave can be easily visualized because QRS complex may not follow every P wave (1). Sprague et al. (2), who first described the Ta wave, noted that the Ta wave polarity is in the opposite side of P wave, and the duration is longer. In low atrial rhythm, P waves are observed inverted on ECG; therefore, repolarization will be on the opposite side.

In normal SR, atrial stimulus originates from the sinoatrial (SA) node and spreads downwards to the ventricles. Therefore, P waves are positively observed in inferior leads. Ta waves of the atria are hidden within QRS complexes; therefore, we can not observe the repolarization of the atria on ECG.

In low atrial rhythm, atrial activation initiates from the ectopic focus different from the SA node, and it spreads from below to upwards in the atria. Thus, in inferior leads, we can observe negative P waves. By antegrade conduction of atrioventricular node, QRS complexes are normally formed, and Ta waves take place in the QRS complexes. However, in some patients with faster heart rate in low atrial rhythm, Ta waves can slide into the ST segment and result in pseudo-ST elevations because of positive of the Ta waves. Yet more in stress electrocardiography, we can see pseudo-ischemic ST depression in inferior leads by the same mechanism as in normal SR.

The causes of ST segment elevation in inferior leads were prominent Ta waves originating from a faster low atrial rhythm and the baseline early repolarization findings in our patient. Therefore, this ECG finding misled us because we supposed it as a ST elevation myocardial infarction.

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