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ECG stress test induced atrial ischemia in a patient with old inferior myocardial infarction due to a distal coronary artery lesion

András Vereckei[1,*], Gábor Katona[1], Zsuzsanna Szelényi[2], Edit Takács[3], Pál Maurovich-Horvat[2], Dávid Becker[2]

[1]*3rd Department of Medicine, Budapest, Hungary*
[2]*MTA-SE Cardiovascular Imaging Research Group, Heart and Vascular Center, Budapest, Hungary*
[3]*Center of Nuclear Medicine, Semmelweis University, Budapest, Hungary*

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A 78-year-old man with a history of mitral valve prolapse underwent echocardiography during his cardiological check-up examination in 2011 in a symptom-free stage. Echocardiography revealed akinesis of the inferior septum and inferobasal free wall as a novel finding suggesting a distal right coronary artery (RCA) lesion (Figure 1). The systolic left ventricular function was normal. Earlier echocardiographies did not show wall motion abnormalities. The patient informed us that several months before the echocardiography he had a stronger chest pain. The stress myocardium perfusion imaging showed only fixed perfusion defects corresponding to the akinetic myocardial segments (Figure 2). The patient was symptom-free, therefore coronary angiography was not performed and secondary prevention therapy was initiated due to the history of myocardial infarction. Since the diagnosis of myocardial infarction, he was essentially symptom-free and attended yearly check-up echocardiography and ECG stress test examinations. The follow-up echocardiographies were identical to that performed in 2011 and the ECG stress tests were normal. In October 2014, the patient came to a regular out-patient check-up examination. His physical examination was unremarkable with normal heart rate and blood pressure. The echocardiography findings were unchanged. The ECG recorded before the start of the ECG treadmill stress test was also identical to the resting ECG tracing recorded in 2011, showing signs of old inferior myocardial infarction (pathological Q wave in lead III and borderline pathological Q wave in lead aVF by voltage criterion, Q wave in lead II, rSr' complex in lead aVR) (Figure 3A). Figure 3B demonstrates the ECG tracing recorded during stage II of the ECG treadmill stress test. The most important alteration is a 0.075 mV PR segment elevation associated with a positive P wave in lead III. Other significant alterations are the 0.175 mV and 0.125 mV horizontal PR segment depressions, resulting in sharp-angled P wave distal limb-PR segment junction, associated with positive P waves in leads I and aVL, respectively and a 0.1 mV sharp-angled PR segment elevation in lead aVR associated with a negative P wave. In leads III and aVF 0.175 mV horizontal and 0.125–0.15 mV mildly ascending ST segment depressions are present respectively. These alterations did not progress or became less conspicuous at later stages of the treadmill test and the PR and ST segment alterations completely disappeared in the recovery stage. The patient did not have chest pain or arrhythmia during the stress test and showed a normal heart rate and blood pressure response.

The ECG alterations that appeared during stage II of the treadmill test may be consistent with atrial infarction or atrial ischemia, however, because they were not present in the pre-test resting ECG and did not persist i.e., completely disappeared during the recovery period, they most likely correspond to atrial ischemia and not to atrial infarction. The atrial repolarization is normally reflected by the T wave of atrial repolarization (Ta wave) and the P-Ta segment. The Ta wave is normally opposite in direction to the P wave representing atrial depolarization and is most frequently hidden in the following QRS complex. The P-Ta segment, which is analogous to the ST segment of ventricular repolarization, is also masked by the superimposition of the QRS complex, the only part of it, which is usually visible is the PR or PQ interval.[1] The P-Ta segment normally slopes

***Correspondence to:** vereckei@kut.sote.hu

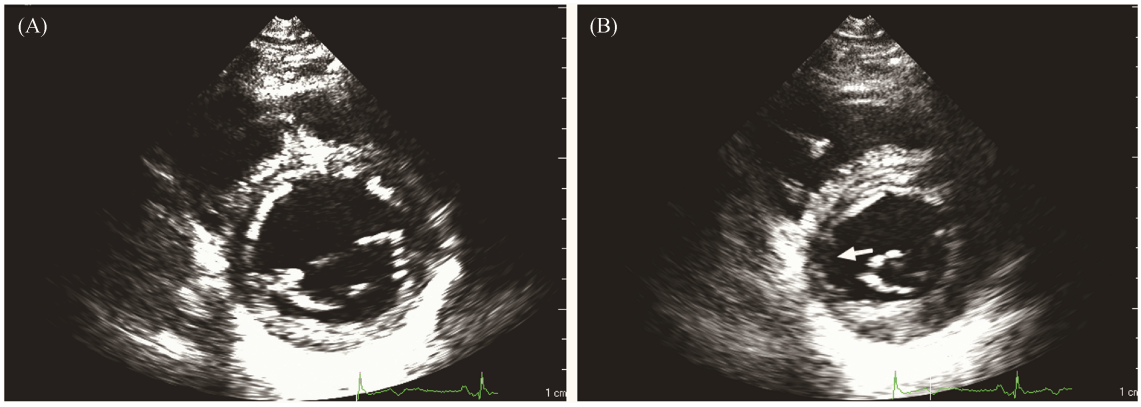

Figure 1. Echocardiographic end diastolic (A) and systolic (B) parasternal short axis basal views. Arrow shows in panel B that the inferior septum and inferobasal free wall being akinetic fails to thicken during systole.

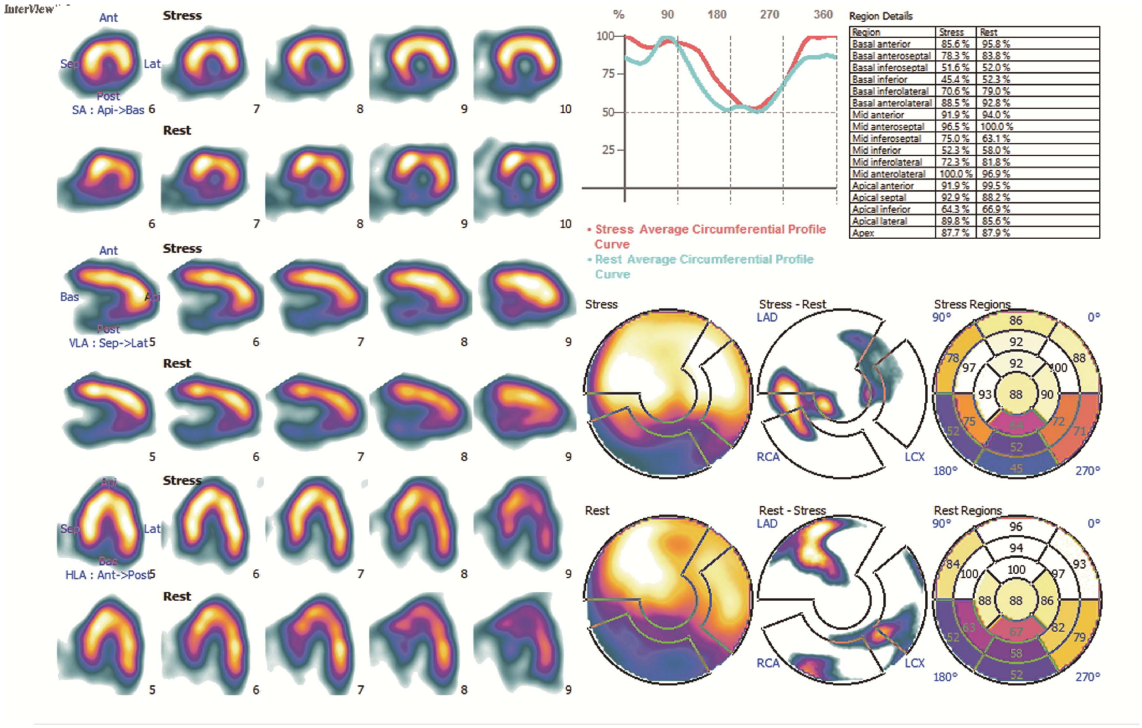

Figure 2. Myocardial perfusion SPECT study viewed in slices and polar maps under stress and rest. Severe irreversible perfusion defect is visualized in the middle and distal segments of the inferior wall extending to the inferolateral and inferoseptal regions. Ant: anterior; Api: apical; Bas: basal; HLA: horizontal long axis; LAD: left anterior descending; LCX: left circumflex coronary artery; Lat: lateral; Post: posterior; RCA: right coronary artery; SA: short axis; Sep: septal; VLA: vertical long axis.

gently in the opposite direction to the P wave from the distal limb of the P wave. Any displacement (elevation or depression) of the PR segment may indicate atrial infarction or atrial ischemia.[1–3] The PR segment elevation in the presence of an upright P wave in lead III is definitely abnormal, because normally a PR segment depression should accompany an upright P wave. The PR segment depressions associated with an upright P wave in leads I and aVL and the PR segment elevation associated with a negative P wave in lead aVR might be normal findings, as the polarity of the PR segment is normally opposite to the P wave it belongs to. However, normally the PR segment tends to merge smoothly and imperceptibly to the distal limb of the P wave, but if there is a sharp-angled, horizontal depression or elevation, as is the case in our patient, it should be considered an abnormal finding suggesting atrial infarction or ischemia, although this is a less reliable sign than a PR segment displacement of opposite than expected polarity.[1] In summary,

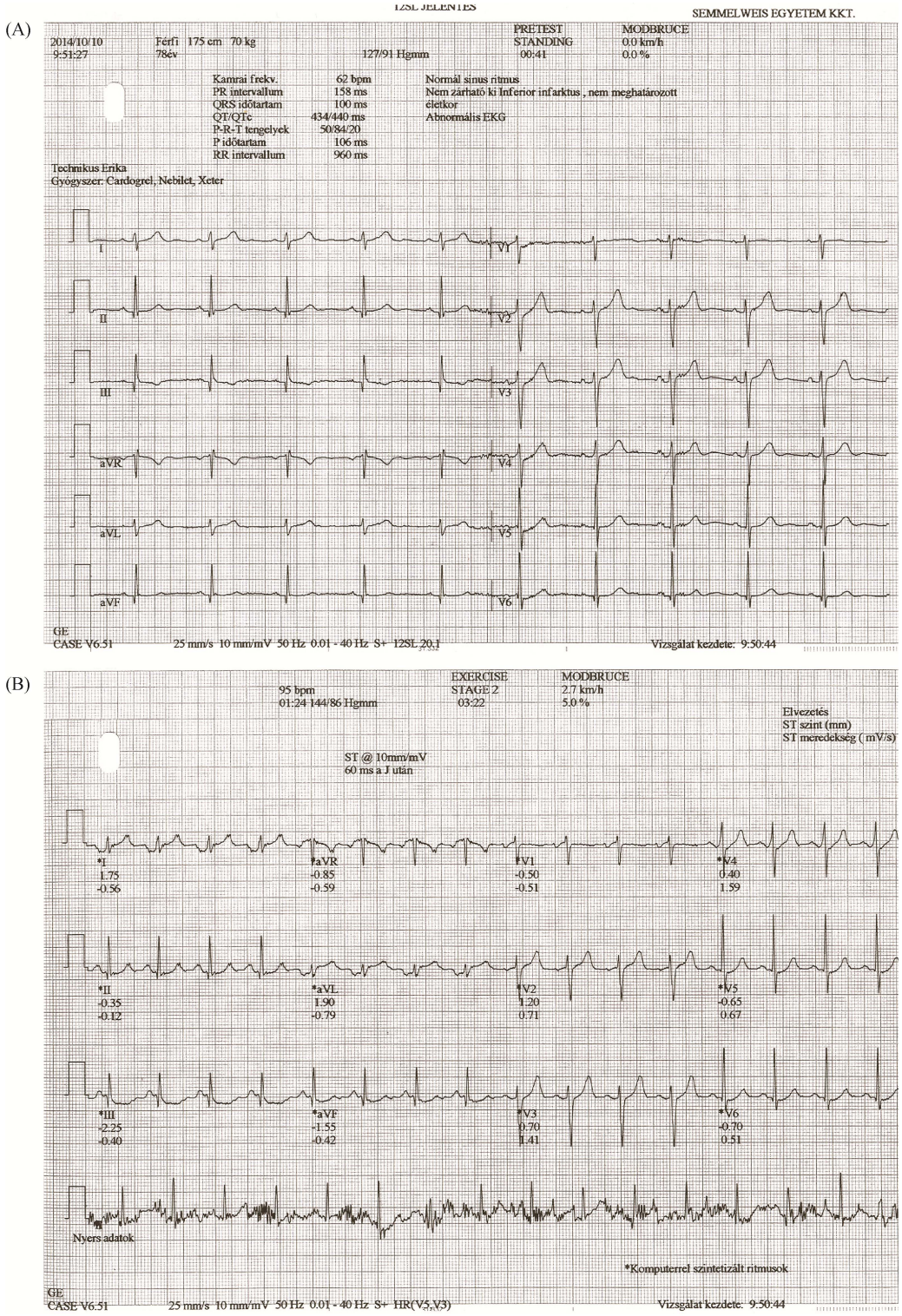

Figure 3. Representative ECGs recorded during the ECG stress test. (A): Pretest resting ECG; (B): ECG recorded during stage II of the stress test. For further explanation see text.

the ECG stress test in our patient suggested the presence of atrial ischemia. No other signs of atrial infarction or ischemia such as supraventricular arrhythmias, sinus bradycardia, sinus arrest, sinoatrial block or P wave abnormalities were present in our patient.[1–3] The borderline significant-significant horizontal ST depressions in leads III and aVF sug-

gested inferior ventricular myocardial ischemia as well. Exaggerated Ta waves may also produce spurious depression of ST segments during exercise stress test, because with exercise-induced tachycardia both the P wave and Ta wave amplitudes increase, the PR interval shortens shifting the Ta wave toward the ST segment. This phenomenon may be suspected when there is a prominent P wave together with a short sharply down-sloping PR segment, especially notable in the inferior leads.[4,5] However, this is not the case in our patient, because the PR segment is horizontal and not down-sloping and in lead III the PR segment is elevated.

The presence of these unconventional ECG signs of ischemia have great importance in this patient, because atrial ischemia can be the consequence of a significant stenosis in the proximal RCA before giving rise to the sinoatrial branch or in the sinoatrial branch itself, because the sinoatrial branch arises from the RCA in approximately 60% and from the left circumflex artery in approximately 40% of cases.[6] The presence of inferior septum and inferobasal free wall akinesis in the absence of associated right ventricular infarction on echocardiography and myocardial perfusion imaging indicated a distal RCA lesion, i.e., an RCA lesion located after the arising of the right ventricular branch. Thus, the novel finding of atrial ischemia during ECG stress test in the symptom-free patient suggested the potential development of a novel significant proximal RCA lesion on top of the known old distal RCA lesion in this patient, warranting further work-up.

The coronary CT revealed severe atherosclerosis, medium grade stenosis in the proximal-mid left anterior descending (LAD) and proximal circumflex (CX) arteries, and severe stenosis in the proximal RCA, medium grade stenosis in the mid-RCA and segmental occlusion of the RCA before the crux (not shown). The coronary angiography revealed a dominant RCA with a complex, significant lesion right after its orifice and another significant stenosis in the proximal RCA slightly more distally, a borderline significant stenosis in the mid part of the RCA and a gracile RCA outflow with occlusion of the posterolateral branches (Figure 4A). The significant proximal RCA stenoses were responsible for the right atrial ischemia and the occlusion of the posterolateral branches for the old inferior myocardial infarction. At the territory of left main coronary, LAD and CX only diffuse coronary atherosclerosis, gracile branches with vessel wall irregularities were visualized. Circumscribed significant stenoses were visualized only on gracile secondary branches (not shown). The blood flow of the RCA outflow tract was provided through collaterals. Percutaneous coronary intervention of the proximal RCA stenosis was performed with the deposition of a drug eluting stent with an excellent result (Figure 4B). During a follow-up ECG stress test performed after the intervention atrial ischemia could no longer be provoked (Figure 5), right now the patient is in a good clinical condition and symptom-free.

During the evaluation of exercise stress test, attention must be paid to unconventional ECG signs of myocardial ischemia, such as atrial ischemia, precordial peaking of the T waves, lead strength calculations, ST elevation in lead aVR, increase in P-wave duration and ST depression in premature ventricular complexes, in order to avoid overlooking important pathology.[7]

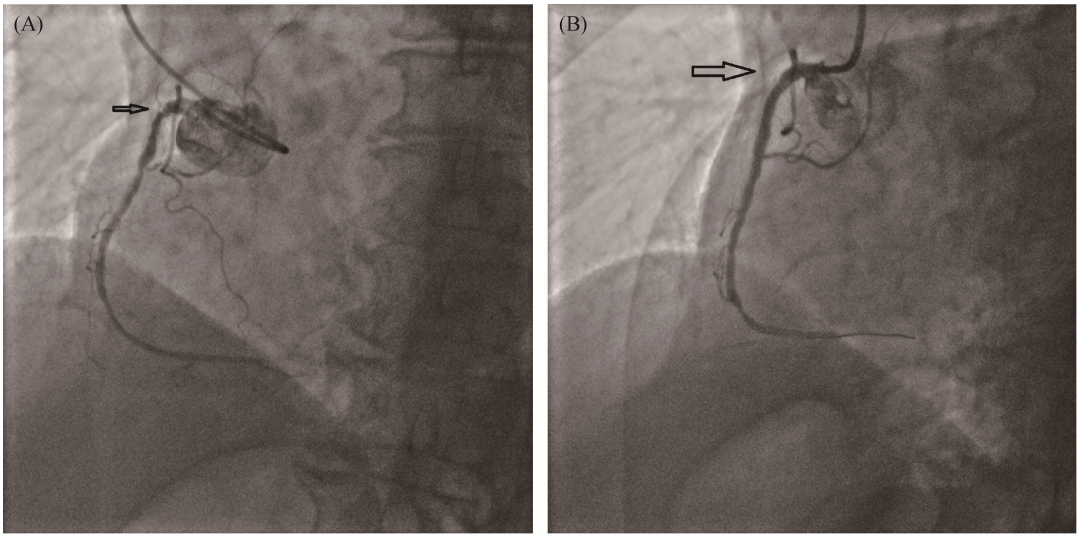

Figure 4. Coronary angiogram in the left anterior oblique view. (A): Demonstrates the significant proximal right coronary artery stenosis denoted by an arrow; (B): shows the result of the percutaneous coronary intervention (direct stent implantation marked by an arrow). For further explanation see text.

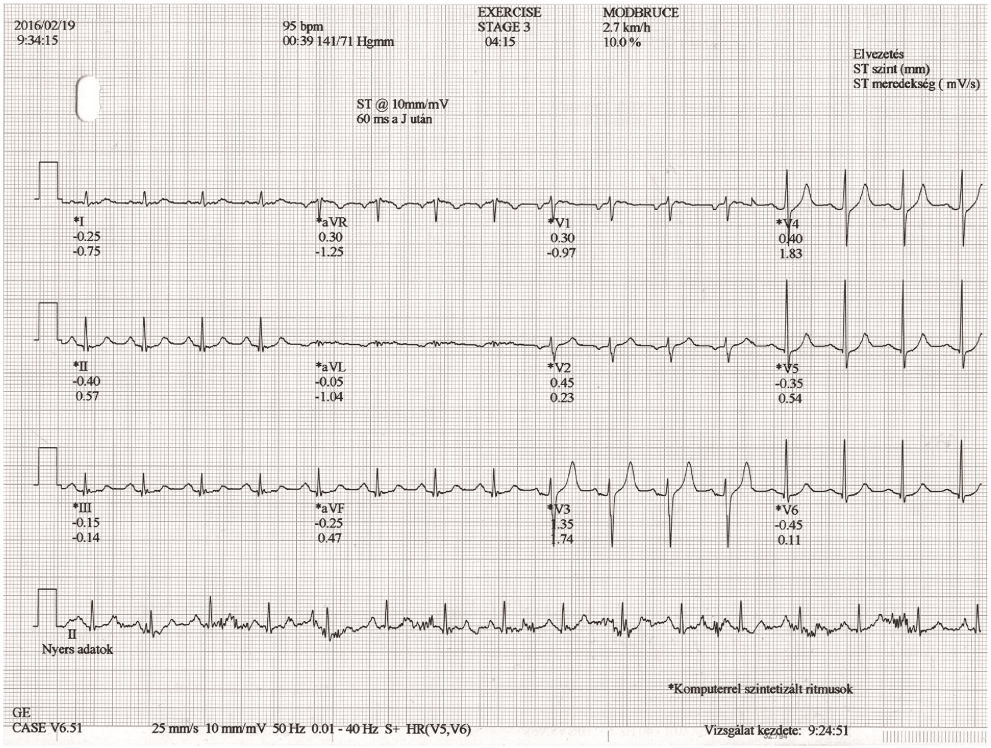

Figure 5. Representative ECG recorded during the post percutaneous coronary intervention follow-up ECG stress test. At the same heart rate, when ECG signs of atrial and inferior ventricular ischemia was induced during the first stress test, no signs of atrial and ventricular ischemia appeared.

Acknowledgements

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