ST Segment Elevation During Adenosine Pharmacological Stress Testing in a Patient with Coronary Artery Disease

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Case history: a 76-year-old African-American female presented to a community hospital with complaints of chest pain. Her medical history was significant for hypertension, gastroesophageal reflux disease (GERD), hyperlipidemia, and bipolar disorder. On arrival at the emergency department, electrocardiogram showed acute inferior wall myocardial infarction (MI). The patient received thrombolytic therapy with tenecteplase (TNKase™) with successful reperfusion. On arrival at our hospital, the patient denied any chest pain and her electrocardiogram showed sinus bradycardia with small Q-waves in the inferior leads II, III, and AVF (see *Figure 1*). She was treated with aspirin, heparin, lisinopril, simvastatin, and clopidogrel. Beta-blocker was not started as the patient was having bradycardia. The patient was scheduled to undergo myocardial perfusion scan 48 hours following her uncomplicated inferior wall MI.

At rest, the patient received 12.0mCi of technetium 99m-sestamibi. Seventy-five minutes later, rest myocardial perfusion images were obtained by non-gated single photon emission computed tomography (SPECT). Then she received adenosine infusion at 140µg/kg/minute for six minutes. Blood pressure and 12-lead electrocardiogram were obtained every minute and the patient's symptoms were recorded. Resting heart rate was 67. Heart rate three minutes into the adenosine infusion was 73, and heart rate six minutes into the adenosine infusion was 85. Resting blood pressure was 152/69. Blood pressure three minutes into the adenosine infusion was 128/54, and blood pressure six minutes into the adenosine infusion was 70/47. Resting electrocardiogram showed normal sinus rhythm, small inferior Q-waves, and ST–T changes. There was an additional 1–2mm ST segment depression in the electrocardiographic leads I, AVL, and V2, plus 1–3mm additional ST segment elevation was noted in II, III, and AVF during and up to three minutes-post adenosine infusion (see *Figure 2*). There were occasional premature ventricular complexes and premature atrial complexes before, during, and post-adenosine infusion. The patient did not report chest pain during or after adenosine infusion.

Three minutes into the adenosine infusion, 44mCi of technetium 99m sestamibi was injected intravenously. One hour and 50 minutes later, the stress (adenosine) myocardial perfusion images were obtained by gated SPECT.

SPECT images were acquired with a dual-head gamma camera using the step-and-shoot detector rotation, obtaining 32 projections over 180º arcs (45º right anterior oblique to 45º left posterior oblique). The camera was equipped with a low-energy, high-resolution collimator. Rest and stress images were acquired with a 20% window centered over the 140KeV photo peak. Gated acquisitions were obtained using eight frames per cardiac cycle with a 40% acceptance window. Acquisition times were 40 seconds per projection. The summed projection data sets were filtered with a butterworth filter (order 5 cut-off 0.33). Short-, vertical-, long-, and horizontal-axis images were evaluated for transient, partially reversible, and fixed perfusion defects.

The rest and stress (adenosine) myocardial perfusion images revealed normal-sized left ventricle and mild to

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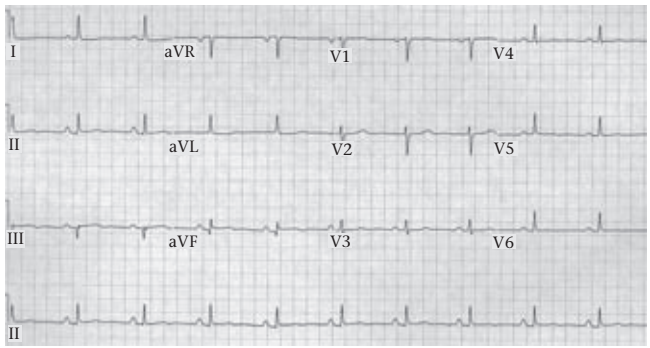
Figure 1: Initial Electrocardiogram Showing Q-waves in the Inferior Leads



moderate transient myocardial perfusion defect (mild to moderate ischemia) superimposed on mild fixed myocardial perfusion defect (superimposed on mild scar) involving the inferior, inferoapex, and inferolateral wall of the left ventricle (see *Figure 3*). There were hypokinetic inferior, inferoapex, and inferolateral walls of the left ventricle, with resting global left ventricular ejection fraction (LVEF) of 40%. The right ventricle was normal in size and function.

Following the adenosine myocardial perfusion scanning, the patient underwent cardiac catheterization. Cardiac catheterization showed normal left main, left anterior descending, and circumflex arteries. The right coronary artery showed a 75% stenosis in the mid segment (see *Figure 4*). The patient had a 3.5x16mm stent deployed without any complications. Her post-procedure course in the hospital was uneventful and she was discharged the following day in a stable condition.

Discussion

Adenosine acts via its four subtypes (A1, A2A, A2B, and A3) of receptors in the coronary artery.[1] A2A adenosine receptor is the predominant subtype on the endothelium and smooth muscle of the coronary blood vessels that causes coronary vasodilatation.[1,2] However, A2B subtype has also been reported in the coronary vessels and may play a role in coronary vasodilatation but to a lesser extent in normal than in pathophysiological situations.[1,3] On the other hand, both A1 and A3 adenosine receptors have been shown to play an inhibitory role in the regulation of coronary blood flow.[4,5]

In this case history, transient myocardial perfusion defect with corresponding ST segment elevation has been noted with adenosine infusion induced ischemia.[6,7] This has been postulated to be secondary to myocardial ischemia that occurs during adenosine infusion, which causes a fall in regional coronary flow below resting levels

Figure 2: Electrocardiogram Obtained During Adenosine Infusion Showing ST Segment Elevation in the Inferior Leads

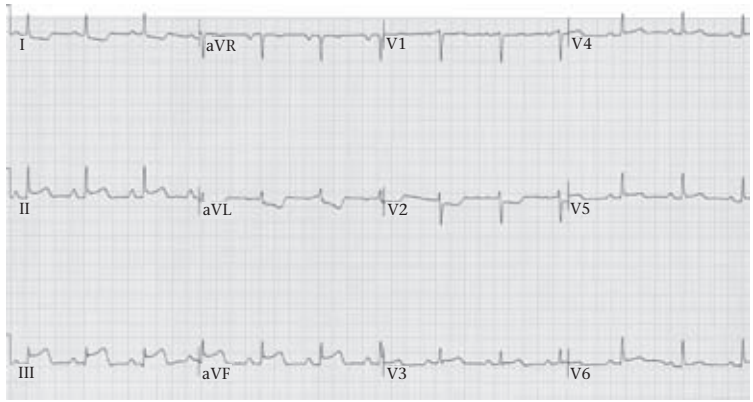


Figure 3: Stress Myocardial Perfusion Polar Map Depicting Flow Defect in the Region of the Right Coronary Artery

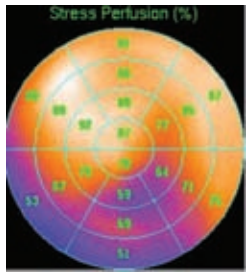
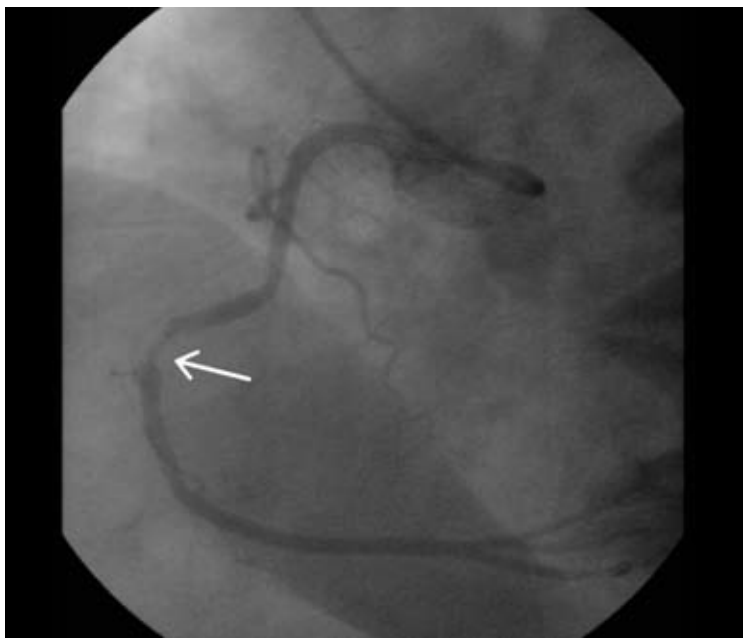


Figure 4: Left Anterior Oblique View of the Right Coronary Artery



Arrow points to the site of stenosis.

caused by a steal phenomenon.[8] When these patients were treated with anti-anginal medications and re-studied by echocardiography, wall motion abnormality and ST depression were less frequent.[9] Rare cases of MI after

adenosine stress testing have been reported.[10] However, adenosine myocardial perfusion scanning has been safely performed within 24 hours after an uncomplicated MI.[11] We reported a case of adenosine-induced coronary vasospasm with significant ST elevation in a patient with normal coronary arteries.[12] Although there could be similarities between this case and the one reported earlier without significant coronary artery disease where A1 receptor activation by adenosine in the vascular smooth muscles may provide an explanation, other possible explanations should be considered. These include the overexpression of A1 receptors compared with A2A/B receptors in the damaged endothelium and/or underlying smooth muscle and/or the increase in sympathetic tone due to adenosine infusion causing vasospasm. Finally, unless we fully understand the pharmacology of adenosine receptors in diseased coronary artery, these are mere speculations based on recent work that shows more heterogeneity of the adenosine receptor population in animal models than previously appreciated.[1] ■

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