ST-segment Elevation in Lead Augmented Vector Right may also be Caused by Diffuse Left Main Coronary Artery Vasospasm Without Fixed Stenosis

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ABSTRACT

ST-segment deviation in lead augmented vector right (aVR) is useful for evaluating patients with acute coronary syndrome (ACS). The ST-segment elevation in this aVR in the patient with clinically suspected acute coronary syndrome suggests a strong possibility of left main coronary artery (LMCA) obstruction due to fixed stenosis. In this article, we report the first case, to our knowledge, of ST-segment elevation in lead aVR due to diffuse LMCA spasm.

Key words: vasospasm, left main coronary artery, ST-segment elevation, lead augmented vector right

Introduction

ST-segment deviation in lead augmented vector right (aVR) is useful for evaluating patients with acute coronary syndrome (ACS). The ST-segment elevation in this lead has been associated with fixed left main coronary artery (LMCA) lesions in patients with unstable angina or ST-segment elevation acute myocardial infarction.^{1–3} Although acute obstruction of the LMCA is not frequently encountered,⁴ prediction of LMCA obstruction is important with regard to selecting the appropriate treatment strategy, since acute LMCA obstruction usually causes severe hemodynamic deterioration resulting in a less favorable prognosis.

Coronary artery spasm is an infrequent but important cause of unstable angina. Its incidence among patients undergoing diagnostic coronary angiography for suspected ischemic syndromes is about 3–4%.⁵ But, spasm of the LMCA is very rare.^{6,7} In this article, we report the first case, to our knowledge, of ST-segment elevation in lead aVR due to diffuse LMCA spasm without fixed stenosis.

Case Report

A 53-year-old man presented to our department with New York Heart Association class III–IV dyspnea, fatigue, and cough. He had history of closed mitral valvulotomy 23 years ago and persistent atrial fibrillation for the last 2 years. The patient was an ex-smoker and was receiving only aspirin (100 mg/day). On physical examination, his blood pressure was 120/70 mmHg, and pulse rate 160 beats per min, arrhythmic. The patient appeared orthopneic and tachypneic. Examination of the heart revealed a rapid rhythm.

A grade 2/6 holosystolic murmur that radiated from the apex to the axilla and diastolic rumble at the apex were heard. Crackles were heard throughout in both lungs. His electrocardiogram (ECG) revealed atrial fibrillation with rapid ventricular response. There were no pathological findings on the laboratory examinations. A chest radiograph showed pulmonary edema. Moderate mitral stenosis (valve area 1.4 cm², mean gradient 5 mmHg), moderate mitral regurgitation, severe tricuspid regurgitation, and moderate pulmonary hypertension (systolic pulmonary artery pressure 50 mmHg) were detected on transthoracic echocardiography. Left ventricular ejection fraction was 65%. Left atrial and left ventricular end-diastolic diameter were 52 mm and 33 mm, respectively. His symptoms resolved following treatment for heart failure including digoxin and furosemide. After having been compensated, he was started on 50 mg of metoprolol for the purpose of ventricular rate control. From the third day of therapeutic switch, his effort angina developed in the morning hours. His ECG taken during angina pectoris, showed a 1-mm ST-segment elevation in lead aVR and up to 3-mm ST-segment depression in leads II, III, augmented vector foot (aVF), and V3-6 (Figure 1). Examination of blood biochemistry including cardiac enzymes revealed no abnormal findings. Coronary angiography with the 6 Fr JLA catheter was performed upon recurrence of angina pectoris despite nitrate and metoprolol and fixed stenosis was not observed in the coronary arteries. However, the LMCA revealed diffuse vasospasm (Figure 2). Electrocardiographic changes were similar during the documented episode of vasospasm and during spontaneous angina. A total of 200 µgm of intracoronary nitroglycerin

Figure 1: Twelve lead ECG, which showed ST-segment elevation in lead aVR and ST-segment depression in leads II, III, augmented vector foot (aVF), and V3-6.

was administered. A subsequent angiogram revealed that the LMCA spasm was completely relieved (Figure 2). Electrocardiographic findings and angina pectoris resolved after intracoronary nitroglycerin (Figure 3). Then, metoprolol was tapered and eventually discontinued. The patient has been free from angina for 6 months on a regimen of 240 mg of diltiazem, 40 mg of isosorbide mononitrate, and warfarin.

Discussion

Coronary spasm is an important cause of chest pain and myocardial ischemia. Vasospasm of the LMCA may be spontaneous (as an uncommon cause of variant angina) or iatrogenic (catheter- or guidewire-induced). Catheter-induced spasm of the LMCA has been previously reported,⁸ however, left main spasm during routine diagnostic catheterization is a relatively rare occurrence. We did not think the catheter-induced spasm in our case, since chest pain was present before coronary angiography. Although the ECG during vasospasm is classically described as showing ST elevation, ST-depression can be rarely documented as in our

case.⁹ The average QRS axis in the presence of LMCA obstruction also shows left axis deviation and this indicates an injury current toward the right shoulder induced by severe anterior and posterolateral ischemia.¹⁰ So the QRS axis was horizontal (around -10°) during acute ischemia and more vertical after the relieving of vasospasm in our case.

When the coronary angiography is normal or shows only nonobstructive plaques and if transient ST-segment deviation can be demonstrated at the time at which the patient has the discomfort, the presumptive diagnosis of variant angina can be made and no further tests are necessary.¹¹ The role of provocative testing in the clinical diagnosis of coronary spasm is controversial. Furthermore, most patients can be often diagnosed clinically by a triad of chest pain with associated ST-segment changes and symptomatic response to calcium channel blockers. But, LMCA vasospasm was confirmed during coronary angiography in our case. Widespread use of these tests in patients with undiagnosed chest pain syndromes should not be recommended currently.¹² Therefore, we did not perform provocative testing.

Figure 2: Twelve lead ECG recording after administration of intracoronary nitroglycerin.

Figure 3: Coronary angiography in right anterior oblique (RAO) view, showing diffuse left main coronary artery vasospasm (A) and resolution of the spasm after administration of intracoronary nitroglycerin (B).

The ST-segment elevation in lead aVR in the patient with clinically suspected ACS suggests a strong possibility of LMCA obstruction. Yamaji et al.³ noted the relationship between acute LMCA obstruction and ST-segment elevation greater than 0.05 mV in lead aVR. From the study, the sensitivity of ST-segment elevation in lead aVR for predicting LMCA obstruction was determined to be 81% and the specificity to be 80%. Kosuge et al.¹ found that ST-segment elevation greater than 0.5 mm in lead aVR to be the strongest predictor of LMCA or three-vessel disease, superior to the presence of ST-segment depression in other leads in patients with non-ST-segment elevation-ACSs who underwent coronary angiography in the acute phase. Statistical analysis of this finding revealed a sensitivity of 78%, a specificity of 86%, a positive predictive value of 57%, and a negative predictive value of 95%. This reasonably high sensitivity allows the clinician to rule-in LMCA in the ACS patient with lead aVR abnormality.

In addition to aiding in the diagnosis of LMCA involvement in patients presenting with ACS, the finding of ST-segment elevation in lead aVR also has prognostic significance. In his second study on the topic, Kosuge et al.¹³ studied numerous ECG findings and a variety of biochemical markers, only ST-segment elevation greater than 0.5 mm in lead aVR on the admission ECG and elevated troponin T levels were determined to be independent predictors of adverse clinical events at 90 days. Additional prognostic information is obtained from lead aVR electrocardiographic presentations, as noted by the work of Barrabes et al.¹⁴ They reported that ST-segment elevation in lead aVR on admission was superior to ST-segment depression in other leads for predicting the risk of death in a first non-ST-segment elevation acute myocardial infarction.

In conclusion, it should be kept in the mind that ST-segment elevation in lead aVR might be due to vasospasm of LMCA without fixed stenosis in the patients with non-ST-segment elevation-ACS because of differences between the therapeutic strategies. It is especially important that lead aVR not be ignored as is common fashion, because it can provide essential diagnostic information in a range of clinical presentations, as highlighted by the example of LMCA vasospasm as well as fixed stenosis.

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