Acute occlusion of the left main trunk presenting as ST-elevation acute coronary syndrome

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Abstract
Acute obstruction of the left main coronary artery (LMCA) is not frequently encountered. Electrocardiographic findings are important to early diagnosis in determining an acute obstruction of the LMCA, which requires immediate aggressive treatment, in this extremely unstable condition. However, there is no single typical electrocardiographic pattern representing acute occlusion of the LMCA. We describe a rare electrocardiographic finding that suggested ST-elevation acute coronary syndrome of the anterior zone due to left main trunk total occlusion.

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Introduction

An acute obstruction of left main coronary artery (LMCA) is encountered approximately in only 0.5% of acute myocardial infarction (AMI) cases.¹ Acute myocardial infarction due to acute obstruction of LMCA, which has poor prognosis, causes severe left ventricular dysfunction and life-threatening malignant arrhythmias as a consequence of large infarction area.² The electrocardiogram (ECG) predicting an acute obstruction of the LMCA, which requires immediate aggressive treatment, is very important for early diagnosis. Various studies have showed that ECG findings of acute LMCA occlusion may be different and confusing.³

In this case report, we report a rare ECG pattern due to the acute obstruction of the LMCA.

Case report

A heavy-smoker 40-year-old man was admitted to the emergency department with a complaint of typical retrosternal chest pain lasting for 3 hours accompanied by nausea and vomiting. He had a history of diabetes mellitus for 4 years and insulin therapy. The patient had no history of angina pectoris. The patient developed ventricular fibrillation in the emergency department, requiring direct-current cardioversion. On physical examination, his blood pressure was measured at 65/40 mm Hg, and his pulse rate was 110 beats/min and regular. On auscultation, there were crepitant rales in the basal parts of the lungs. The ECG showed sinus rhythm, left anterior hemiblock, and ST-segment elevation in leads L1, aVR, aVL, and V2-V6, besides ST-segment depression in the inferior leads and lead V1 (Fig. 1). It also showed grade 3 ischemia according to Sclarovsky-Birnbaum ischemia grading algorithm; the J point is above 50% of the R wave amplitude in leads V2 to V5, and there are no S waves in leads V2 to V3. The plasma levels of both troponin I and creatinine kinase-MB on admission were high: 19.0 ng/mL (reference value, <0.01 ng/mL) and 25.9 ng/mL, respectively. The other laboratory findings were normal. The patient was referred to the catheterization laboratory for primary percutaneous coronary intervention with the diagnosis of anterior wall AMI and cardiogenic shock. The coronary angiogram revealed a significant thrombus-containing stenosis in the LMCA (Fig. 2). There were no critical lesions in the other major coronary arteries. Primary stenting was performed, with a stent of 4.0 mm in diameter and 9.0 mm in length, and the thrombolysis in myocardial infarction grade 3 flow was achieved. After the coronary stenting, the patient’s ECG showed ST resolution of more than 70%. The echocardiography was performed after the procedure and showed an ejection fraction of 55%, besides wall motion.

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impairment in the anterior segments. The discharge ECG revealed negative T waves in leads L1, aVL, V5, and V6 without any significant Q waves. The patient was discharged on fifth day.

Discussion

There are some ECG criteria demonstrated in various studies in the diagnosis of acute LMCA obstruction for different clinical settings. Gorgels and colleagues$^4$ reported that ST elevation in lead aVR indicated LMCA lesion or 3-vessel disease in patients with unstable angina pectoris. Barrabes and colleagues$^5$ reported that in patients with non-ST-elevation AMI, an ST elevation in lead aVR of 1 mm or greater was associated with LMCA lesion or 3-vessel disease and with adverse outcome. Similar results were also confirmed in several other studies in non-ST-elevation acute coronary syndromes.$^6,7$

The data about ST-elevation AMI are relatively limited. Nikus and Eskola$^3$ emphasized that ST elevation in lead aVR with anterior ST-elevation or ST-segment depression in leads V4 to V6 may indicate left main occlusion. Mahajan et al$^8$ showed in their study that greater ST-segment deviation in lead V6, compared with ST-segment deviation in lead V1, was a useful predictor of acute coronary syndrome resulting from culprit LMCA lesion. Differentiation of the left anterior descending artery (LAD) occlusion from the LMCA lesion is more problematic in ST-elevation AMI. In a study of 16 patients with acute obstruction of the LMCA by Yamaji et al.$^9$ ST-segment elevation in lead aVR with less elevation in lead V1 was proved to be an important predictor of total or subtotal LMCA obstruction. In the same study, the authors reported that ST-segment elevation in lead aVR was

Fig. 1. The ECG revealing ST elevation in leads L1, aVR, aVL, and V2 through V6 besides ST depression in inferior leads and lead V1.
observed in greater amplitude and more frequently in patients with anterior AMI with LMCA disease compared with those without significant LMCA disease.

Both acute LMCA and LAD obstructions generally produce anterior wall ischemia and present as an anterior AMI with ST elevation in precordial leads. Because LMCA disease is associated with both LAD and left circumflex coronary artery (LCx) involvement, involvement of more than 1 coronary artery in the ischemic process may alter the ECG manifestations. In acute occlusion of LMCA, Yamaji et al hypothesized that the electrical force in the posterior (newly termed as “lateral”) wall ischemia counterbalances the ischemia-induced electrical force in the anterior wall. Interestingly, the ECG in this case demonstrates ST-segment depression in lead V1. ST depression in leads V1 and V2 are accepted as the reciprocal changes of lateral wall ischemia that was caused by LCx obstruction. The absence of ST elevation in lead V1 in anterior AMI may be explained in several ways: (i) in the fact that the LAD occlusion is distal to the first septal branch and (ii) the protection of interventricular septum due to a large conus branch arising from right coronary artery. In this case, the conus branch was not large. At first glance, the presence of ST elevation in aVR and the sum of ST depression of 2.5 mm or greater in lead L2 and aVF have been thought that the occlusion was in proximal part of LAD. However, the absence of ST depression in lead V5 or V6 and presence of ST depression in lead V1 were unusual. Therefore, we thought that the ST-segment depression in lead V1 may be explained by the lateral wall ischemia because of LCx involvement that counteracts the ST elevation in lead V1 in the LMCA obstruction.

In conclusion, ST depression in lead V1 may suggest acute occlusion of the LMCA in patients with anterior AMI and without previous anginal symptoms as a result of lateral wall involvement.

References