CASE REPORT

Acute Myocardial Infarction with a Left Main Trunk Lesion and Documented Lambda-like J Waves

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Abstract

We herein describe a case of a myocardial infarction, in which Lambda-like J waves were documented. The patient was referred to our hospital due to ventricular fibrillation. The twelve-lead electrocardiogram (ECG) on admission showed prominent J waves in the lateral and precordial leads. Coronary angiography revealed 99% stenosis with a delay in the left anterior descending artery, 75% stenosis in the left main trunk, and possible ischemia in the conus branch. Our report addresses the possibility that ischemic J waves can be used as an important marker for lethal arrhythmias in patients with acute myocardial infarction.

Key words: J wave, ventricular fibrillation, myocardial infarction

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Introduction

Recently, attention has been focused on “J wave syndrome” (1-3) since the association between the early repolarization patterns observed in the electrocardiograms and risk of idiopathic ventricular fibrillation (VF) was first reported by Haïssaguerre et al. (4). In 2009 the concept of ischemic J waves was introduced by Jastrezebski (5), whose paper presented ECGs from seven patients with ischemia-induced VF and J waves during transient coronary spasms and acute myocardial infarctions. Similar observations had been already reported in several papers (6-8). More recently, the relationship between the J-wave dynamics and arrhythmias during myocardial ischemia in patients with vasospastic angina was also systemically investigated (9).

On the other hand, from the basic electrophysiological point of view, Yan et al. reported that acute myocardial ischemia caused ST-segment elevation with a prominent J wave, which was caused by a heterogeneous loss of the transient outward potassium current (Ito)-mediated epicardial action potential dome, leading to the development of phase 2 reentry and resultant VF (10). In addition, more recently, a genetic disorder underlying J wave syndrome was discovered in some patients, and one of the possible candidates has been shown to be the ATP sensitive potassium (IK,ATP) channel (11-13). Since the IK,ATP channel can be related to an early repolarization pattern and Brugada syndrome in certain situations, there may also be a link between ischemic J waves and primary or secondary IK,ATP channelopathy.

We experienced a patient with acute myocardial infarction (MI) whose culprit lesion was thought to be the left anterior descending artery (LAD) associated with the possible involvement of the left main trunk (LMT) and conus branch, and in whom prominent ischemic J waves were documented in the lateral and precordial leads. We herein discuss the clinical implications of Lambda-like J waves induced by ischemia based on the present case.

Case Report

A 72-year-old man presented with a sudden loss of consciousness with a moan while reading a newspaper on the sofa early in the morning on January, 2012. A bystander

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started resuscitation efforts right away and called an ambulance. When the rescue team arrived at his home, he was found to be in VF, and defibrillation was performed. He was transferred to Asahikawa Medical University Hospital by ambulance. On admission, ventricular tachycardia at a rate of 140/min was documented and a successful defibrillation was performed. At that time, his ECG exhibited ST elevation with prominent J waves in the lateral and precordial leads (what we call Lambda-like ST segment elevation) and reciprocal ST depression in the inferior leads (Fig. 1). He had no family history of sudden cardiac death. The initial laboratory data revealed an increased white blood cell count (9,830/μL) and the creatine phosphokinase (CPK)-MB level was 59 IU/L. The potassium level after resuscitation was 3.5 mEq/L. Emergency coronary angiography revealed 99% stenosis with a severe delay in segment 7 in the LAD, 75% stenosis in the LMT, and possible ischemia in the conus branch supplied by the left circumflex artery (LCX) (Fig. 2).

Since the culprit lesion was thought to be the LAD, a stent was performed. At that time, his ECG exhibited ST elevation in aVR, V1-V3 and J waves in the inferior leads, compatible with an antero-septal MI (Fig. 3). The maximum CPK-MB value was 541 IU/L. Echocardiography revealed a left ventricular ejection fraction (LVEF) of 59%, with hypokinesia of the left ventricular (LV) apex. Although his vital signs gradually became stable without any inotropic support, the patient remained unconscious, and hence, the hypothermic therapy was continued. In the chronic stage at one month after admission, the J waves in the inferior leads could still be observed (Fig. 4). Unfortunately, the patient has not yet regained consciousness at six month after admission.

An ECG recorded the next day exhibited a QS pattern in V1-V6 and J waves in the inferior leads, compatible with an antero-septal MI (Fig. 3). The maximum CPK-MB value was 541 IU/L. Echocardiography revealed a left ventricular ejection fraction (LVEF) of 59%, with hypokinesia of the left ventricular (LV) apex. Although his vital signs gradually became stable without any inotropic support, the patient remained unconscious, and hence, the hypothermic therapy was continued. In the chronic stage at one month after admission, the J waves in the inferior leads could still be observed (Fig. 4). Unfortunately, the patient has not yet regained consciousness at six month after admission.

Discussion

We herein describe a case of acute antero-septal MI with an episode of VF in which global ischemic J waves were documented. The J-wave-like ST segment elevation pattern, similar to a grade-III ischemia pattern (14), which was observed after cardioversion, was considered to be a J wave, since no initial upsloping phase of the ST-T complex was observed (5). Furthermore, this feature of the J wave was considered to be what we call a “Lambda wave,” as described by Riera et al. (15), which was subsequently named by Gussak and recently reported by the Jastrzebski and Aizawa groups (9, 16, 17).

Originally, the “Lambda wave” was named for a case with primary idiopathic cardiac asystole whose ECG showed a nonischemic “action-potential-like” QRS-ST complex generated by a remarkably slow slope of its descending portion, followed by negative T waves seen predominantly in the inferior leads. More recently, the Jastrzebski group reported a Lambda-like ST segment elevation in three cases of acute myocardial infarction (17). In their cases, an “action-potential-like” QRS-ST complex was observed in all cases, however, the leads in which the Lambda wave appeared differed from case to case depending on the culprit lesion, and negative T waves were not always present. Those characteristics were similar to those observed in our case.

Considering the mechanism responsible for the Lambda-like ST segment elevation in the present case, not only the ischemia in segment 7 in the LAD, but also the 75% stenosis in the LMT might have played a critical role. The mechanisms underlying the development of this condition were considered as follows. It has been said that it is sometimes difficult to interpret ECG findings in patients with LMT infarctions due to the presence of bundle branch block and/or lethal arrhythmias such as VF or atrio-ventricular block. Hirano et al. (18) classified the ECG features of LMT infarctions into two main groups; right bundle branch block with a remarkable left axis deviation (RBBB+ LADEV type) and ST segment elevation in leads V2-V6, I and aVL without any abnormal axis deviation (LAD type). According to the classification, our ECG was similar to the first group, an RBBB+ LADEV type. Furthermore, although it has been generally accepted that ST segment elevation in leads V1 and aVL and ST segment changes in various leads are specific features of a LMT lesion, they further summarized the specific features of LMT lesions as follows; [1] relative left axis deviation, [2] prolongation of the QTc interval, [3] prolongation of the QRS interval, [4] ST segment elevation in aVL, [5] ST segment elevation in extensive precordial leads and [6] newly emerged abnormal Q waves (18). In our case, in addition to the main culprit lesion at segment 7 in the LAD, a 75% stenotic lesion in the LMT might also have played a critical role in the genesis of the ECG changes, since the specific features as described by Hirano et al. almost matched our case. On the other hand,

**Figure 1.** ECGs obtained just after cardioversion. Note that prominent J waves (arrow heads) can be observed in leads I, aV1, and all the precordial leads.
recent reports have provided evidence that ischemia of the conus branch may induce Brugada-type ECG changes in patients with coronary artery disease (19-22). In our case, the Brugada-like ECG pattern may have been caused by a LMT lesion leading to possible conus branch ischemia. To the best of our knowledge, this is the first report to describe the J wave elevation in the precordial leads induced by possible ischemia of the conus branch supplied by the LCX. In addition, the excessive ST segment elevation in I, aV_{L}, V_{1}-V_{6}, may also be explained by the LMT stenosis, as well as the severe delay in the LAD.

Recently, an association between J waves and myocardial ischemia has also been reported (9, 23, 24). According to those reports, the global ischemia induced by the right coronary artery (RCA) and/or left coronary artery (LCA) tended to generate J waves mainly in the inferolateral leads and rarely in the precordial leads. In our case, the J waves in the inferior leads were persistently documented, suggesting a possible link between J waves and an ischemic event, as reported in a recent clinical study (25).

It has also been proposed that some patients who suffer from an acute MI have genetic abnormalities predisposing them to lethal ventricular arrhythmias, which may be caused by channelopathies, and these patients are at high risk of developing VF during acute ischemia (17, 26, 27). Yan and Anzelevitch proposed that a prominent I_{to}-mediated action potential notch in the ventricular epicardium, but not in the endocardium, creates a transmural voltage gradient producing a J wave or J-point elevation on the ECG (28). Furthermore, an increase in the net repolarizing current, due to either a decrease in inward Na^{+} and Ca^{2+} currents, or augmentation of outward currents, such as I_{to} and IKATP, is postulated to accentuate the notch leading to the J wave and its augmentation (3). In ischemic heart disease, the inhibition of Na^{+} and Ca^{2+} currents and enhancement of the I_{to} and IKATP is expected to occur. The peculiar ST-segment elevation pattern that we call Lambda, which was seen in our case, would be explained by such a mechanism. A basic study reporting that

Figure 2. Coronary angiography findings on admission. There was 99% stenosis with a severe delay in segment 7(a), 75% stenosis in the left main trunk (b), 50% stenosis in segment 1, and 99% stenosis with a severe delay in 4PL (c), as well as possible ischemia in the conus branch supplied by the left atrial branch originating from the left circumflex artery (d, e).

Figure 3. ECGs obtained the day after admission. Note the QS pattern in leads V_{1}-V_{3} and J waves in leads II, III and aV_{L}.
The authors state that they have no Conflict of Interest (COI).