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【 CASE REPORT 】

Variant Angina with Spontaneously Documented Ischemia- and Tachycardia-induced "Lambda" Waves

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Abstract:
In a patient with variant angina of the proximal left anterior descending coronary artery, myocardial ischemia changed the QRS-ST-T configurations without J-waves into those resembling "lambda" waves at maximal ST-segment elevation, and couplets or triplets of supraventricular extrasystole (SVE) changed the ischemia-induced "lambda" waves into QRS-ST-T configurations resembling a "tombstone" morphology or "monophasic QRS-ST complex." At the resolution phase of coronary spasm, the QRS-ST-T configurations returned to those without J-waves and were changed by SVE into "lambda" waves. Interestingly, neither ischemia- nor SVE-induced "lambda" waves or SVE-induced "tombstone" morphology or "monophasic QRS-ST complex" were complicated by ventricular tachyarrhythmia.

Key words: variant angina, lambda wave, transmural conduction delay, proximal left anterior descending coronary artery, left septal fascicular block, left anterior fascicular block

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Introduction

An electrocardiogram (ECG) pattern with both an upsloping-QRS and end-QRS slurring concomitant with steep downward ST-segment elevation landing on a negative T-wave in a case of Brugada syndrome reported by Riera et al. (1), which resembles the Greek letter "λ (lambda)," was dubbed the "lambda" wave by Gussak et al. (2).

"Lambda" waves, which are relatively uncommon ECG findings, are considered J-wave variants leading to malignant ventricular arrhythmia, particularly in acute ST-segment elevation myocardial infarction (STEMI) (3) or variant angina (VA) (4). Furthermore, the ECG pattern with a massive ST-segment elevation in which the limits of the QRS complex and the T-wave with or without negativity are frequently undefinable, referred to as a "tombstone" morphology or "monophasic QRS-ST complex," is observed in STEMI or VA (5). A "tombstone" morphology or "monophasic QRS-ST complex" is also associated with fatal ventricular arrhythmia.

We herein report a case in which both ischemia-induced and supraventricular extrasystoles (SVE)-induced QRS-ST-T configurations resembling "lambda" waves occurred, and those showing a "tombstone" morphology or "monophasic QRS-ST complex" developed only at the onset of SVE without ventricular tachyarrhythmia (6).

Case Report

An 80-year-old Japanese man was admitted to our hospital with a 10-day history of left anterior chest pain lasting several minutes without associated symptoms, such as diaphoresis. Multiple episodes occurred daily in the left lateral decubitus position (LLDP) at 9 PM when he went to bed and at 1 AM during sleep.

He had not smoked or consumed alcohol. His medical history included a 1-year history of hypertension and chronic renal disorder, and he had been prescribed 2.5 mg enalapril maleate daily at another medical clinic.

On admission, his temperature was 36.7°C, pulse rate was 76 beats per minute with regular rhythm, systemic blood

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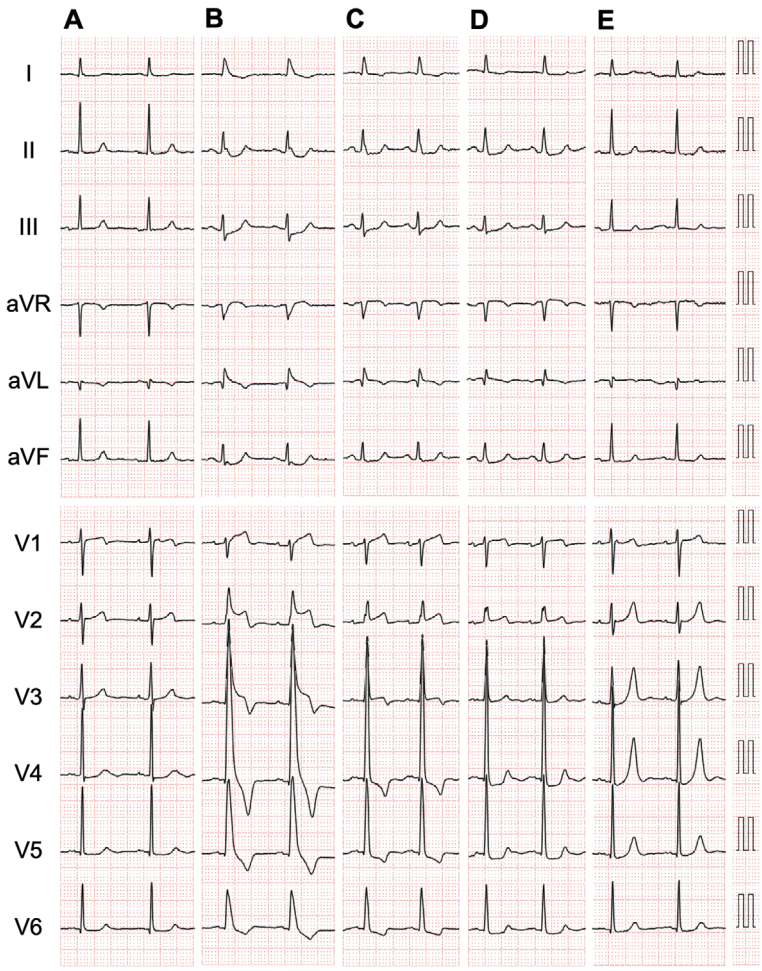

Figure 1. Electrocardiograms (ECGs) recorded on admission (A) and during silent angina attack (B-E). The ECG on admission (Panel A) reveals a sinus rhythm with ST-segment depressions of 0.1 mV in leads V4 through V6. Negative U-waves, although shallow, are also found in leads V4 and V5, but no J-waves are observed. During angina without chest pain (Panel B-E), ST-segment elevations in leads aVR and V1 through V2 and "lambda" waves in leads I, aVL, and V3 through V6, associated with reciprocal ST-segment depressions in leads II, III, and aVF, and QRS-axis deviations to the left in the frontal plane of -23° are seen, as well as an increase in R-wave amplitudes in leads V2 through V4 (Panel B). These ECG findings subside spontaneously, and negative U-waves and tall T-waves appear (Panel C-E). The duration of Panels B to E is approximately 2 minutes.

pressure was 159/88 mmHg, and oxygen saturation on room air measured using a pulse oximeter was 100%. On auscultation, there were no heart murmurs or rales in the lung fields. The liver, kidneys, and spleen were not palpable. No pretibial edema was found. Blood tests revealed a high-sensitivity cardiac troponin I level of 15.1 pg/mL, which was below the reference range (≤ 34.0 pg/mL). His estimated glomerular filtration rate was 39 mL/min/1.73 m^2, and his brain natriuretic peptide level was 75.1 pg/mL (reference range, ≤ 18.4 pg/mL). A radiograph of the chest did not detect pulmonary congestion or cardiomegaly. An ECG revealed sinus rhythm with ST-segment depressions of 0.1 mV in leads V4 through V6 (Fig. 1A) Negative U-waves, although shallow, were also found in leads V4 and V5, and no J-waves were observed.

Approximately 2 hours after the first ECG was recorded, echocardiography was performed in the LLDP. The left ventricular wall motion was almost normal, but severe hypokinetic wall motions occurred in the basal to apical anteroseptal and anterior regions without chest pain after approximately 2 minutes. ECG recording was therefore quickly performed again in the supine position (Fig. 1B-E and Fig. 2; the recordings in Fig. 2 are obtained between Fig. 1B and Fig. 1C; the duration between Fig. 1B and E is approximately 2 minutes). ST-segment elevations in leads aVR and V1 through V2 and QRS-ST-T configurations resembling "lambda" waves in leads I, aVL, and V3 through V6, associated with reciprocal ST-segment depressions in leads II, III, and aVF, and QRS-axis deviations to the left in the frontal plane of -23° were seen, as well as an increase in R-wave amplitudes in leads V2 through V4 (Fig. 1B). Immediately after the recording in Fig. 1B, several couplets or triplets of SVE appeared for approximately 20 seconds. These SVEs (Fig. 2A-C) changed the "lambda" waves into QRS-ST-T

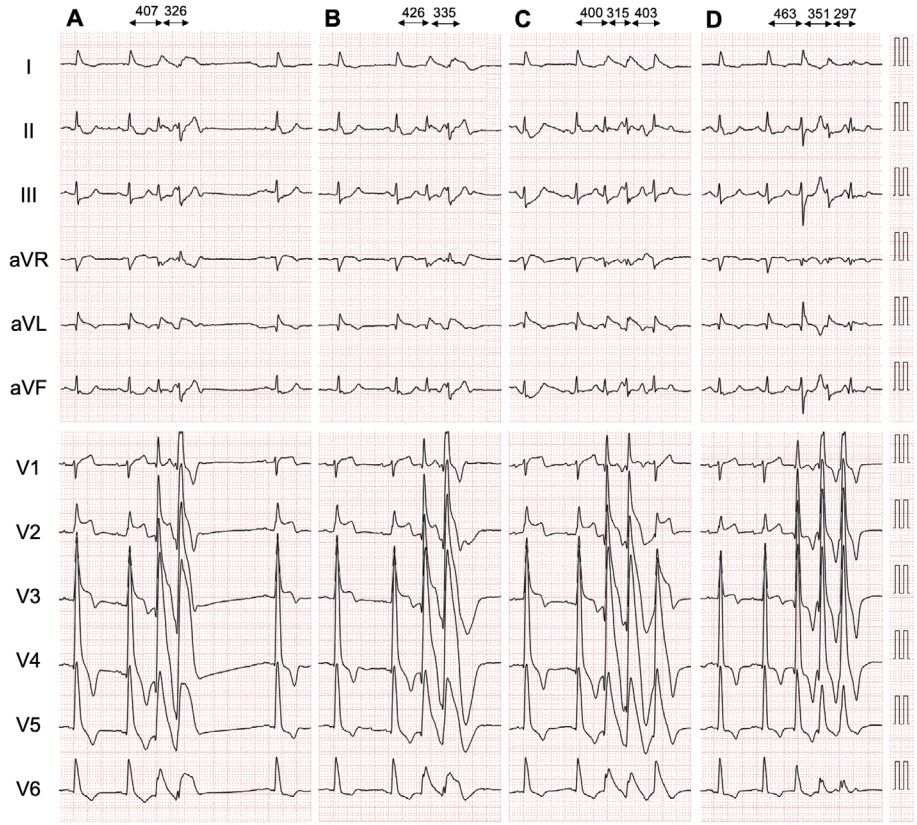

Figure 2. Electrocardiograms recorded during silent angina attack. Immediately after the recording in Fig. 1B, several couplets or triplets of supraventricular extrasystoles (SVEs) appear for approximately 20 seconds. These SVEs (Panel A-C) change the "lambda" waves into "monophasic QRS-ST complex" and "tombstone" morphology. During the resolution period of ST-segment elevations (Panel D), the QRS-ST-T configurations develop prolongation of R-waves but no apparent J-waves and are changed by SVEs into "lambda" waves, concomitant with the fragmentation in the fourth and fifth beats of leads I and V6. Furthermore, the SVEs augment leftward QRS-axis shifts in the frontal plane and prominent anterior QRS forces inappropriate to the degree of ST-segment elevation, particularly in Panel D, together with Q-waves in leads V1 through V5. Numbers indicate RR intervals (ms).

configurations resembling "monophasic QRS-ST complex" and "tombstone" morphologies. During the resolution period of ST-segment elevation (Fig. 2D), the QRS-ST-T configurations developed prolonged R-waves but no apparent J-waves and were changed by SVEs into "lambda" waves, concomitant with fragmentation in the fourth and fifth beats of leads I and V6. Furthermore, SVE augmented leftward QRS-axis shifts in the frontal plane and prominent anterior QRS forces (PAF) inappropriate to the degree of ST-segment elevations, particularly in Fig. 2D, together with Q-waves in leads V1 through V5. These ECG findings subsided spontaneously, and negative U-waves and tall T-waves appeared (Fig. 1C-E). Given the above, the patient was diagnosed with silent myocardial ischemia within the territory of the proximal left anterior descending coronary artery (LAD) that had resulted in total or nearly total transient occlusion.

Nicorandil was administered intravenously at a rate of 4 mg/h along with a single dose of 20 mg of oral prasugrel hydrochloride and 324 mg of oral aspirin. The patient was immediately referred for percutaneous coronary intervention (PCI). Coronary angiography revealed severe narrowing without thrombi or washout delay of contrast medium at the proximal LAD, but no significant stenosis was found in the right coronary artery or left circumflex coronary artery (Fig. 3A-C). Isosorbide dinitrate (2 mg) was then injected into the left coronary artery with no change in the degree of coronary stenosis in the proximal LAD. Coronary artery spasm in combination with a fixed atheromatous plaque of the proximal LAD was suspected. Intravascular ultrasonography (IVUS) revealed eccentric growth of the fibrous plaque away from the lumen, which was indicative of positive vascular remodeling at the culprit lesion (Fig. 4). An IVUS-guided everolimus-eluting platinum chromium coronary stent was directly deployed to the lesion and post-dilated with a non-compliant balloon. The stent was successfully deployed without residual stenosis (Fig. 3D). An ECG was not performed during PCI.

Serum cardiac enzymes, such as creatine kinase, were not elevated over the 1-day follow-up. Repeat ECG performed on the second hospital day revealed T-wave abnormalities in

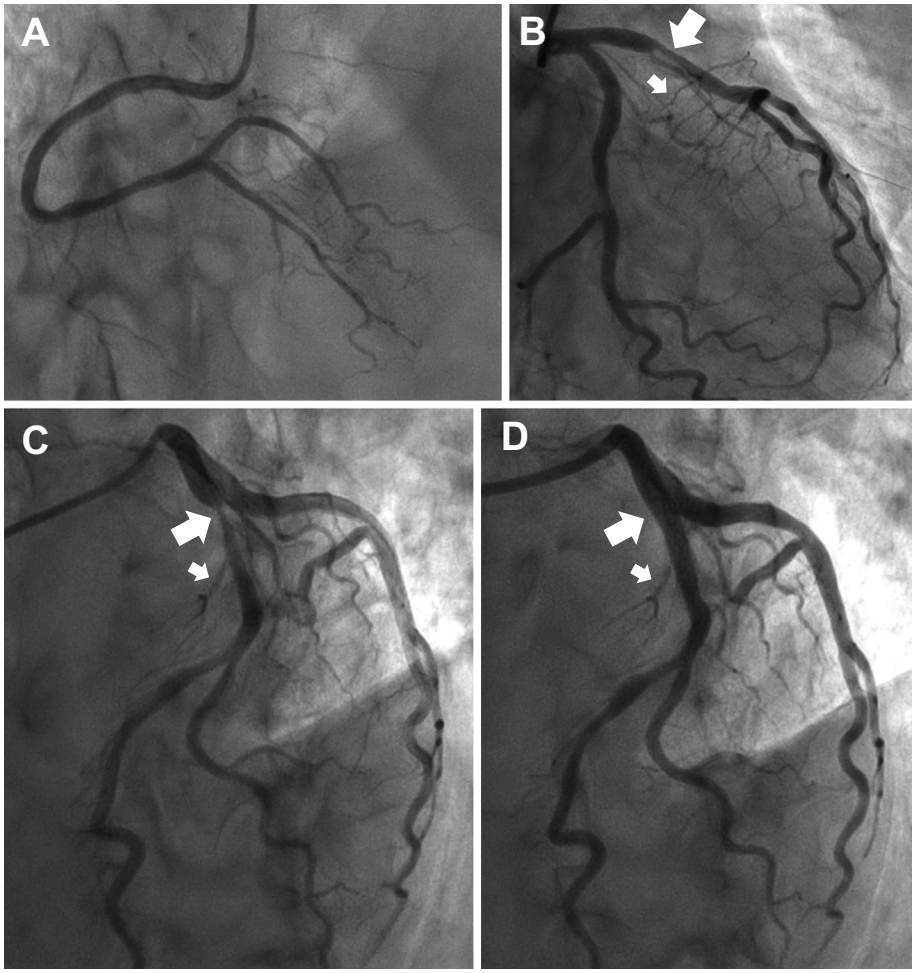

Figure 3. **Coronary angiograms. Coronary angiograms reveal severe narrowing without thrombi or washout delay of the contrast medium at the proximal left anterior descending coronary artery (LAD), but no significant stenosis is found in the right coronary artery or left circumflex coronary artery (Panel A-C). A coronary stent is successfully deployed to the lesion with no residual stenosis (Panel D). The large arrows indicate severe stenosis in the proximal LAD, and the small arrows indicate the first septal perforating branch.**

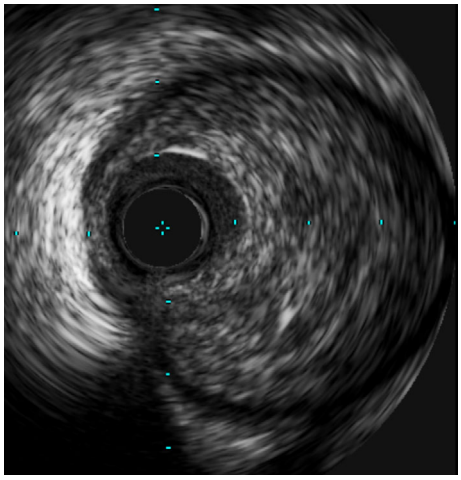

Figure 4. **Intravascular ultrasound (IVUS) imaging at the culprit lesion. A grayscale cross-sectional IVUS image shows eccentric growth of the fibrous plaque away from the lumen.**

lead I and all precordial leads, suggesting Wellens syndrome (Fig. 5A), and an echocardiograph did not reveal wall motion abnormalities in the left ventricle. Intravenous nicorandil was discontinued after the patient started 40 mg of oral nifedipine, and no silent angina re-attacks were confirmed by continuous ECG monitoring. On the sixth hospital day, the ECG patterns normalized, except for the notched or slurred J-waves in leads I, II, and V4 through V6 (Fig. 5B). A provocative test for coronary vasospasm using intracoronary injection of ergometrine maleate (64 μg to the left coronary artery and 40 μg to the right coronary artery) as the provocative stimulus was performed without discontinuing nifedipine and showed a negative test result for coronary vasospasm. Approximately 10 months later, the patient was quite well with no lasting angina.

Discussion

We encountered a case in which ischemia-induced and tachycardia-induced “lambda” waves spontaneously occurred

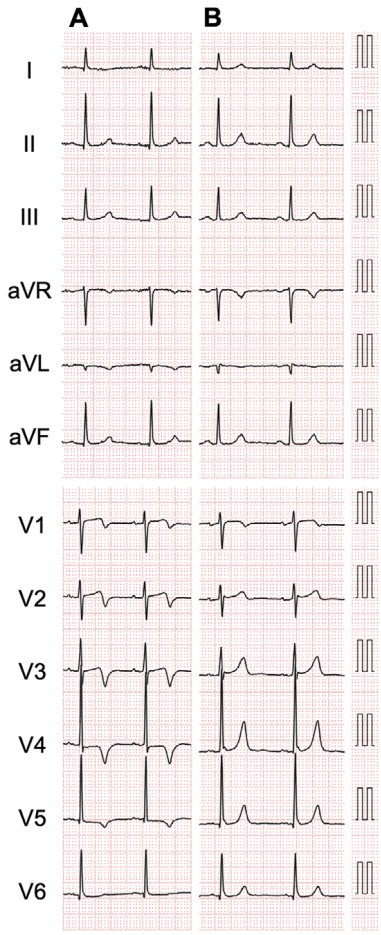

Figure 5. Follow-up electrocardiograms (ECGs). ECG patterns recorded on the second hospital day reveal T-wave abnormalities in lead I and all precordial leads, suggesting Wellens syndrome (Panel A). On the sixth hospital day, the ECG results are normalized, except for the notched or slurred J-waves in leads I, II, and V4 through V6 (Panel B).

during VA in the territory of the proximal LAD. During the period of maximal ST-segment elevation, myocardial ischemia changed the QRS-ST-T configurations without J-waves into "lambda" waves, and couplets or triplets of SVEs changed the ischemia-induced "lambda" waves into a "tombstone" morphology and "monophasic QRS-ST complex." During the resolution phase of coronary spasm, the QRS-ST-T configurations returned to those with persistent prolongation of R-waves but no apparent J-waves and were changed by the SVEs into "lambda" waves. Furthermore, the SVEs augmented ischemia-induced leftward QRS-axis shifts in the frontal plane, which was thought to be the left anterior fascicular block (LAFB), and PAF in the horizontal plane, which was thought to be the left septal fascicular block (LSFB). During angina attack, no ventricular tachyarrhythmia was documented.

Acute myocardial ischemia during VA may affect all components of the electrical activation of the heart, including the P-wave, PR interval, QRS complex, ST-segment, and T- and U-waves (7). The most frequent and typical ECG changes are related to repolarization resulting from progressive ischemia. Early-stage ischemia is very often limited to the subendocardial area, and the first ECG change is often a tall, symmetrical, and usually peaked T-wave accompanied by a mild increase in the QT interval, although this was not observed in the present case. Subsequently, the ischemia becomes transmural. In most cases, the peaked T-wave is followed by an increase in the height and width of the R-wave caused by transmural conduction delays (8), a coincident decrease in the magnitude or disappearance of the S-wave together with ST-segment elevation in leads facing the ischemic region, which is a classical hallmark of acute transmural myocardial ischemia, and reciprocal ST-segment depression in the leads facing the anatomically opposite myocardial segments. The presence of ST-segment elevation coincides with the duration for which coronary vasospastic angina is present (usually several minutes). During the resolution period of ST-segment elevation, the T-waves often become transitionally peaked, positive, and symmetric again and then return to normal. Subsequently, however, deep negative T-waves, which are transient and related to reperfusion, can be seen. These negative T-waves, known as Wellens syndrome if seen in the precordial leads, are dependent on the severity and duration of the preceding ischemia and may last from a few minutes to several hours or days (9). Negative U-waves are detected, particularly in nearly 80% of LAD spasms (10).

J-waves, which have end-QRS notches or slurs occurring on the final 50% of the R-wave downslopes (11), are often revealed in patients with STEMI and even in those with VA. J-waves precede ST-segment elevations as well as morphological changes in T-waves (4) and sometimes become prominent. On serial ECG recording, "monophasic QRS-ST complex" follows a newly appeared "lambda" wave, and the initial part of the ST-segments begin at the halfway point of the descending limbs or close to the top of the R-waves, resulting in "lambda" wave or "monophasic QRS-ST complex", respectively (12). A peculiar pattern, called "tombstone" morphology, is also detected in STEMI or VA (5). In "lambda" wave, "monophasic QRS-ST complex," or "tombstone" morphology, particularly the latter two patterns, it might be difficult to identify the J-waves exactly because of the deformation of the QRS-ST-T configurations. These ECG patterns are considered to have prominent J-waves, although end-QRS notch or slur with a peak of >50% of the R-wave amplitude is regarded as fragmentation, not J-wave (11). In this patient, the "lambda" waves definitely have J-waves with end-QRS slur. The presence of a prominent transient outward current (Ito)-mediated action potential notch in the ventricular epicardium, but not endocardium, leads to the development of a transmural voltage gradient manifesting as a J-wave or a J-point elevation on the surface ECG (13). In contrast, a step delay in impulse transmission across the ventricular wall from the endocardium to the epicardium results in ischemia-induced "lambda" wave, "monophasic QRS-ST complex," or "tombstone" morphology (5, 12).

Ito-mediated J-waves have characteristic features with both pause- or bradycardia-dependent augmentation and tachycardia-dependent attenuation. Nakayama et al. reported that some end-QRS slurs and notches may be augmented or induced at short RR intervals, such as SVEs, particularly in patients with coronary heart disease, which are most often accompanied by changes in the QRS morphology (14). Conduction delay is suspected as the underlying mechanism of such J-waves, which Nakayama et al. labeled as conduction delay-induced J-waves. In our patient, QRS-ST-T configurations, which were nearly normal but had remaining transmural conduction delay as suggested by the continuous R-wave prolongation, were changed by SVEs into "lambda" waves, as shown in Fig. 2D, so these "lambda" waves are likely to include conduction delay-induced J-waves. Fragmentation in the fourth and fifth beats of leads I and V6 is generally believed to be the result of conduction delay (15) and supports the mechanism. Our present patient uniquely showed "monophasic QRS-ST complex" and "tombstone" morphology only at short RR intervals produced by SVEs. These ECG patterns are also considered to be caused by conduction delays. In our patient, both ischemia-induced and tachycardia-induced conduction delays change QRS-ST-T configurations into a "tombstone" morphology and "monophasic QRS-ST complex," but only one of those delays lead to "lambda" waves. "Lambda" waves, "monophasic QRS-ST complex," and "tombstone" morphology may be a series of morphological changes in QRS-ST-T configurations due to transmural conduction delay, and the degree of conduction delay may then lead to various waveforms.

The last part of the QRS-ST-T morphology is modified by T-waves, with or without negativity. "Tombstoning" of the ST-segment, reported first by Wimalaratna (16), or the "triangular QRS-ST-T waveform", reported by Cipriani et al. (17), has no negative T-waves. The impulse transmission across the ventricular wall from the endocardium to the epicardium is blocked in the mid-myocardium, leading to failure of epicardial activation and loss of the negative T-waves, as shown in the fourth beat of leads I, aVL, and V4 to V6 (Fig. 2A). In contrast, negative T-waves are the consequence of a delay in epicardial activation, causing its repolarization to outlast that of the endocardial action potential response (5). The degree of deformation in whole QRS-ST-T waveforms resulting from conduction delays or blocks varies, making it difficult to describe those waveforms reported thus far, including "tombstone" morphology, "tombstoning" of the ST-segment, "monophasic QRS-ST complex," "triangular QRS-ST-T waveform," and "lambda" wave, among others.

Ventricular arrhythmias, which occur during the resolution phase of coronary spasm as well as during the period of maximal ST-segment elevation, are frequently documented (7). In particular, ischemia-induced prominent J-waves with ST-segment elevations, such as "lambda" wave, "monophasic QRS-ST complex," or "tombstone" morphology, may be a warning sign of the development of ventricular tachyarrhythmias in myocardial ischemia as well as in J-wave syndrome, Brugada syndrome, or early repolarization syndrome (4, 12). In contrast, SVEs, which generally occur during the period of ischemia, are not very common. Tachycardia-induced J-waves due to conduction delay are thought to be different from J-wave syndrome (6, 14). Our patient had no ventricular tachyarrhythmia, although both ischemia-induced and tachycardia-induced "lambda" wave, tachycardia-induced "monophasic QRS-ST complex," and tachycardia-induced "tombstone" morphology occurred.

The most likely explanation for the increase in the height of the R-waves in leads V3 and V4 of Fig. 1B-D and in leads V1 through V4 of the third and fourth beats of Fig. 2A-C and the third, fourth, and fifth beats of Fig. 2D is the transient manifestation of PAF secondary to an LSFB (18, 19). Transient PAF in the horizontal plane meets the criteria for LSFB, although the QRS duration reached 0.12 seconds in the fourth beats in Figs. 2A and B and the fourth and fifth beats in Fig. 2D, together with the leftward QRS-axis shift in the frontal plane that was attributed to LAFB (20). Anatomopathological studies have shown that the left septal fascicle (LSF) has diverse morphologies and considerable variability in its structure. The fascicles of the left bundle branch cascade in a trifascicular fashion down the smooth left ventricular septal surface. The most superior fascicle, the left anterior fascicle (LAF), extends toward the superolateral papillary muscle of the mitral valve, while the left posterior fascicle (LPF) runs toward the inferoseptal muscle. The middle or LSF descends between the two, forming a mesh-like network on the septal surface, although this fascicle is absent in approximately 15-40% of human hearts. ECG changes resulting from conduction abnormalities of the LAF and LPF are commonly diagnosed, mainly by the QRS-axis shift in the frontal plane.

In contrast, the existence of conduction defects of the LSF remains controversial. Pérez-Riera et al. reported several cases of transient PAF thought to be caused by LSFB in the setting of proximal LAD disease and have stated that the characteristic ECG feature of LSFB is PAF on the horizontal plane (21, 22). These ECG findings should be distinguished from other conditions that also produce PAF, such as right ventricular enlargement, lateral myocardial infarction, right bundle branch block, Wolff-Parkinson-White syndrome, hypertrophic cardiomyopathy, and Chagas cardiomyopathy in Latin America. The transient nature of PAF excludes all other known causes of ECG changes and is decisive in the differential diagnosis. In our patient, incomplete or complete right bundle branch block due to aberrant conduction might have occurred in the third and fourth beats of Fig. 2A-C and the third, fourth, and fifth beats of Fig. 2D. However, the use of ECG findings alone cannot explain the apparent PAF.

Transient PAF in the horizontal plane, thought to be due to LSFB, and leftward QRS-axis shifts in the frontal plane, thought to be due to LAFB (Fig. 1) are a consequence of proximal LAD disease. In addition, short coupled SVEs augmented the ischemia-induced LSFB and LAFB, as

shown in Fig. 2. However, LAFB in the fifth beat of Fig. 2D appeared attenuated in spite of a shorter duration of the preceding RR interval, in general, leading to a more severe conduction block and an accentuation of LAFB. We believe that recovery from myocardial ischemia during the phase depicted in Fig. 2D prevented further LAFB in the present case, although the degree of LSFB was thought to have peaked by that time. The transient appearance and disappearance of fascicular blocks may be influenced by additional factors, such as latent intrinsic lesions of the conduction system (20, 23, 24).

In conclusion, we encountered a very rare case in which ischemia- and SVE-induced "lambda" waves spontaneously occurred during VA in the territory of the proximal LAD, along with an SVE-induced "tombstone" morphology or "monophasic QRS-ST complex" and the augmentation of ischemia-induced LAFB and LSFB by SVEs. The present patient had no ventricular tachyarrhythmia.

The authors state that they have no Conflict of Interest (COI).

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