



# Acute Left Circumflex Coronary Artery Occlusion — Diagnostic Problems of Initial Electrocardiographic Changes —

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**Background:** Acute coronary syndrome (ACS) with occlusion of the left circumflex coronary artery (LCX) poses diagnostic problems that may lead to a delay in reperfusion.

**Methods and Results:** From a group of 1,269 consecutive patients with ACS, 138 patients with ACS due to LCX occlusion were analyzed for clinical, electrocardiographic, and angiographic presentations, as well as door-to-balloon (DTB) time. Electrocardiographic changes were classified into 4 patterns: ST-segment elevation in inferior/lateral leads (ST-E); ST-segment depression in V1–V4 (ST-D); no significant ST changes (No-ST); and others. The No-ST group was associated with a longer DTB time ( $P < 0.0001$ ) compared with the ST-E and ST-D groups. Compared with the No-ST and ST-E groups, the ST-D group presented with a more advanced Killip class ( $P = 0.003$ ), greater peak creatine phosphokinase ( $P = 0.007$ ) and peak creatine kinase-MB ( $P = 0.006$ ), more frequent proximal LCX occlusion ( $P = 0.007$ ), and worse 1-year outcomes ( $P = 0.0034$ ).

**Conclusions:** One-third of ACS patients with LCX occlusion showed no ST-segment changes, resulting in significantly longer DTB time. Improving diagnostic accuracy is challenging but critical to avoid delayed reperfusion in these patients without electrocardiographic changes.

**Key Words:** Acute circumflex coronary artery occlusion; Diagnostic problems; Door-to-balloon time; No ST-segment changes

The standard 12-lead electrocardiogram (ECG) performed at the time of first medical contact is the principal determinant of the diagnosis and management of patients presenting with a suspected acute coronary syndrome (ACS). However, ACS with occlusion of the left circumflex coronary artery (LCX) often poses diagnostic problems that may lead to a delay in coronary reperfusion because the ECG findings are often non-diagnostic for ST-elevation ACS.<sup>1–3</sup> Patients without ST-segment changes and those with ST-segment depression in precordial leads are usually not subjected to immediate management with emergency coronary reperfusion.<sup>4</sup> Therefore, in the present study, we assessed and clarified the diagnostic problems of initial ECG changes in patients with acute LCX occlusion in relation to its clinical characteristics and management.

## Methods

### Patient Selection

This study consecutively enrolled 1,269 patients with ACS who underwent emergency coronary angiography, in which a thrombotic occlusion was evident and a culprit lesion could be identified, between January 2015 and December 2019. Of the 1,269 patients with ACS, 1,131 with culprit lesions of the right coronary artery, the left anterior descending coronary artery, and/or the left main trunk were excluded. Thus, 138 patients with a culprit lesion of the LCX were entered into the study (**Figure 1**). A retrospective chart review was performed to assess clinical variables and outcomes, including baseline clinical characteristics, ECG findings at the time of admission, blood tests, angiographic features, door-to-balloon (DTB) time, and in-hospital and short-term (up to 1 year) outcomes.

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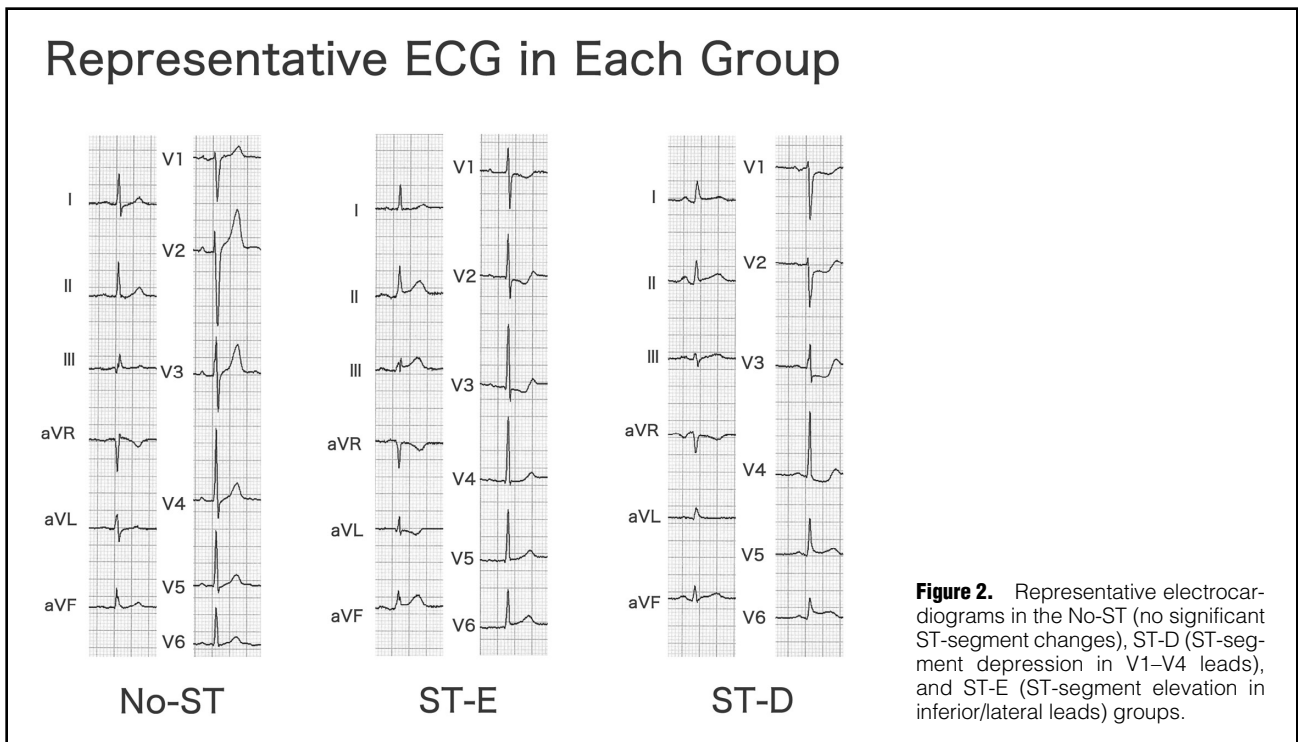
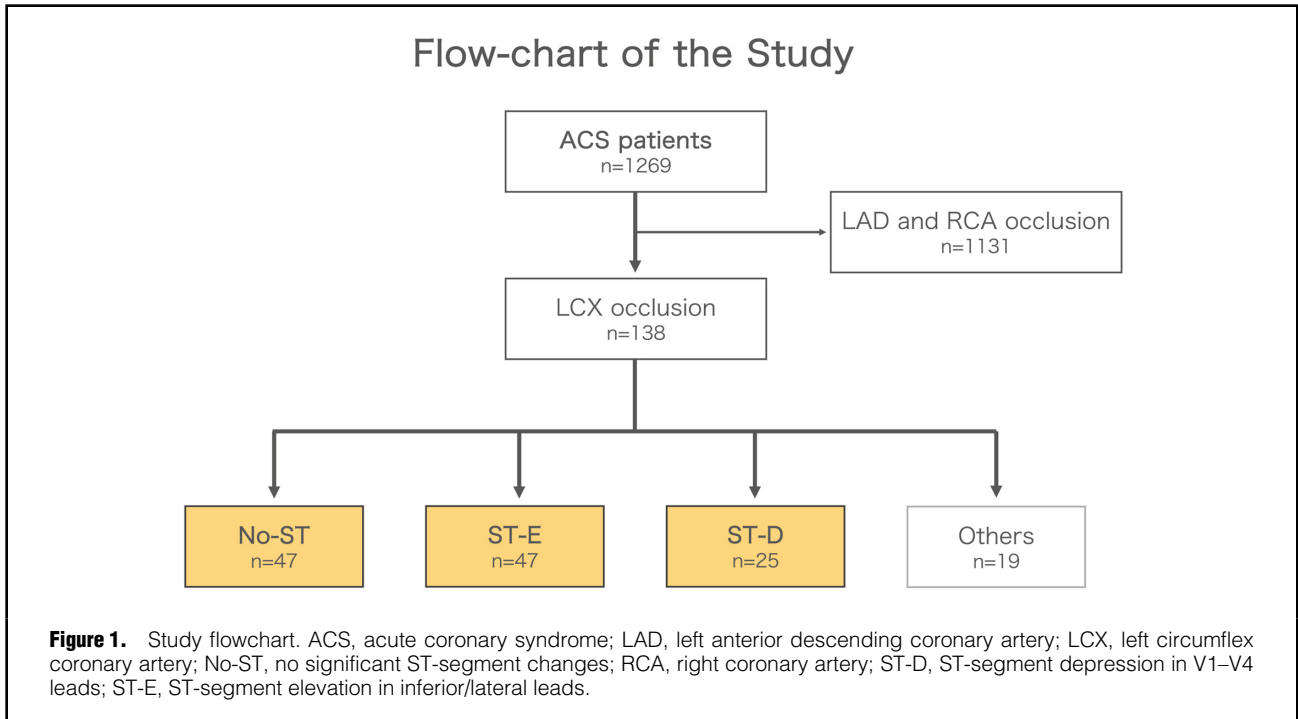
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**Definitions**

**ACS** ACS was defined according to the current universal definition,<sup>5</sup> specifically Type 1 myocardial infarction, elevated high-sensitivity cardiac troponin T (>99th percentile upper reference limit [URL]; >0.014 ng/mL), and angiography features of atherosclerosis and thrombosis.

**Culprit Lesion** All patients underwent coronary angiog-

raphy during hospitalization. The infarct-related artery was identified by the absence of antegrade blood flow and/or by the presence of local intraluminal thrombus. Flow in the culprit coronary segment was graded using the Thrombolysis in Myocardial Infarction (TIMI) trial criteria.<sup>6</sup> Patients with TIMI 0/1 flow in the LCX and those with TIMI >1 flow who presented with ST-elevation and evi-

<b>Table. Baseline Characteristics of Patients in Each of the Three Electrocardiographic Groups</b>				
	<b>No-ST (n=47)</b>	<b>ST-E (n=47)</b>	<b>ST-D (n=25)</b>	<b>P value</b>
<b>Age (years)</b>	70 [59–79]	67 [59–77]	75 [63–81]	0.32
<b>Male sex</b>	32 (68)	33 (70)	12 (48)	0.15
<b>Chest pain</b>	29 (62)	33 (70)	19 (76)	0.42
<b>Coronary risk factors</b>				
Hypertension	29 (62)	27 (57)	18 (72)	0.47
Dyslipidemia	29 (62)	31 (66)	18 (72)	0.68
Diabetes	19 (40)	15 (32)	10 (40)	0.65
Smoker (current or past)	30 (64)	30 (64)	10 (40)	0.10
Family history of CAD	8 (17)	4 (9)	1 (4)	0.19
<b>Past medical history</b>				
Prior MI	3 (6)	3 (6)	4 (16)	0.31
Prior PCI	6 (13)	4 (9)	4 (16)	0.62
Prior CABG	2 (4)	0 (0)	0 (0)	0.21
<b>Baseline medication</b>				
Antiplatelet therapy	9 (19)	8 (17)	5 (20)	0.94
Anticoagulation therapy	2 (4)	2 (4)	0 (0)	0.58
ACEI/ARB	15 (32)	16 (34)	12 (48)	0.37
MRA	1 (2)	0 (0)	1 (4)	0.43
$\beta$ -blocker	4 (9)	5 (11)	3 (12)	0.88
Statin	13 (28)	13 (28)	9 (36)	0.72
Oral antidiabetic agent	9 (19)	4 (9)	4 (16)	0.33
Insulin	0 (0)	0 (0)	0 (0)	–
<b>Killip Class III–IV</b>	5 (11)	3 (6)	9 (36)	0.003
<b>Laboratory findings</b>				
hs-cTnT (ng/mL)	0.118 [0.025–0.445]	0.122 [0.031–0.412]	0.083 [0.028–0.467]	0.84
CPK (U/L)	185 [110–530]	173 [110–396]	165 [99–349]	0.77
CK-MB (IU/L)	18 [12–51]	17 [11–41]	17 [13–43]	0.81
LDH (U/L)	235 [188–294]	219 [194–303]	216 [195–295]	0.88
AST (U/L)	33 [23–64]	35 [23–74]	31 [26–56]	0.98
ALT (U/L)	26 [17–41]	23 [19–39]	22 [17–36]	0.80
BNP (pg/mL)	58 [24–109]	35 [17–112]	71 [27–170]	0.42
LDL-C (mg/dL)	124 [99–150]	122 [105–160]	133 [106–154]	0.78
HbA1c (%)	5.9 [5.6–6.6]	5.9 [5.6–6.5]	6.1 [5.6–6.9]	0.88
Peak CPK (U/L)	697 [291–1,636]	985 [491–2,747]	2,675 [812–3,850]	0.007
Peak CK-MB (IU/L)	73 [21–159]	98 [42–269]	246 [80–341]	0.006
<b>LV asynergy (echo)</b>	31 (66)	38 (81)	22 (88)	0.07
<b>Location of LCX occlusion</b>				0.007
Proximal LCX	16 (34)	6 (13)	13 (52)	
Distal LCX	28 (60)	35 (74)	11 (44)	
Obtuse marginal	3 (6)	6 (13)	1 (4)	
<b>Right dominant</b>	39 (83)	29 (62)	21 (84)	0.03
<b>Multivessel disease</b>	21 (45)	27 (57)	15 (60)	0.38
<b>DTB time (min)</b>	245 [170–562]	93 [83–121]	97 [74–129]	<0.0001
<b>DTB time <math>\leq</math>90 min</b>	5 (11)	21 (46)	10 (43)	0.0004
<b>Outcomes</b>				
In-hospital mortality	1 (2)	1 (2)	1 (4)	0.86
Length of hospitalization (days)	10 [8–13]	13 [9–16]	14 [10–26]	0.0192
MACCE within 1 year	1 (2)	5 (11)	7 (29)	0.0034

Unless indicated otherwise, data are given as the median [interquartile range] or n (%). ACEI, angiotensin-converting enzyme inhibitor; ALT, alanine aminotransferase; ARB, angiotensin receptor blocker; AST, aspartate aminotransferase; BNP, B-type natriuretic peptide; CABG, coronary artery bypass grafting; CAD, coronary artery disease; CK-MB, creatine kinase-MB; CPK, creatine phosphokinase; DTB, door-to-balloon; echo, echocardiography; hs-cTnT, high-sensitivity cardiac troponin T; LCX, left circumflex coronary artery; LDH, lactate dehydrogenase; LDL-C, low-density lipoprotein cholesterol; LV, left ventricular; MACCE, major adverse cardiac and cerebrovascular events; MI, myocardial infarction; MRA, mineralocorticoid receptor antagonist; No-ST, no significant ST-segment changes; PCI, percutaneous coronary intervention; ST-D, ST-segment depression in V1–V4 leads; ST-E, ST-segment elevation in inferior/lateral leads.



dence of LCX intraluminal thrombus were included in the study. With regard to the location of the occlusion and the coronary branch distribution, the proximal LCX segment was considered as the main stem of the vessel extending from its origin to the exit of the first obtuse marginal branch. The coronary segment below this branch was considered as the distal LCX.

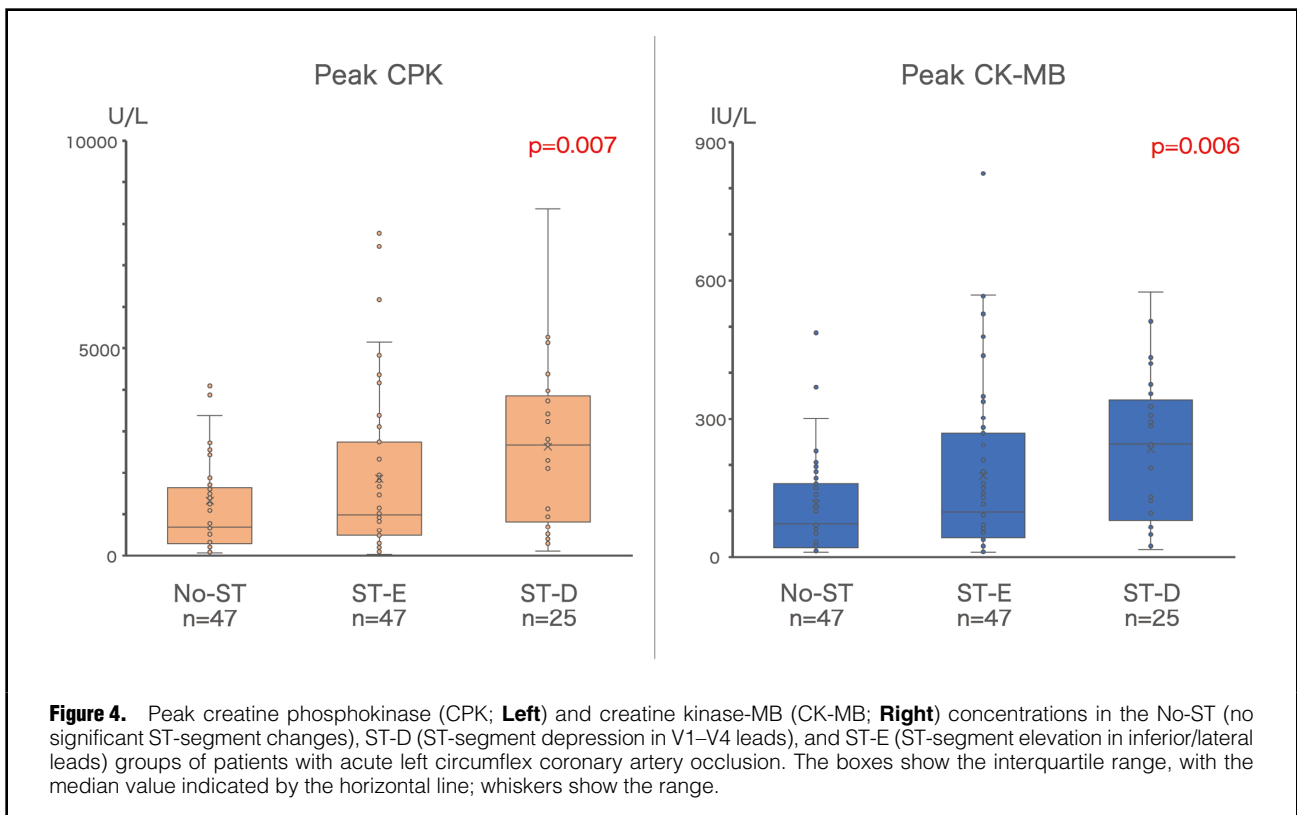
#### Evaluation of ECG Changes

Index ECGs were recorded at the time of arrival at the emergency department using commercially available ECG systems. In each ECG, the basic rhythm, heart rate, and deviation of the ST-segment at the J point with respect to the PR-segment level were analyzed. Accordingly, ECG

changes were classified into 4 patterns: ST-segment elevation of  $>0.1$  mV in at least 2 consecutive leads in inferior/lateral leads (ST-E); ST-segment depression of  $>0.1$  mV in at least 2 consecutive leads from V1 to V4 or  $>0.05$  mV in leads V2 or V3 (ST-D); the absence of ST-segment changes or ST-segment shift  $<0.1$  mV in 2 consecutive leads (No-ST; **Figure 2**); and others. "Other" patterns were those that showed some ST-segment changes but did not fulfill the criteria of ST-E, ST-D, or No-ST. The ECGs were interpreted by 2 investigators who were unaware of the results of coronary angiography.

#### Statistical Analysis

Categorical variables are presented as frequencies and per-



centages. Continuous variables that were not normally distributed are presented as the median and interquartile range (IQR), whereas normally distributed values are presented as the mean  $\pm$  SD. The significance of differences was analyzed using the 2-sample Student's t-test and Chi-squared test, as appropriate. Two-tailed  $P < 0.05$  was considered significant. All analyses were performed using JMP® version 14.2 (SAS Institute, Cary, NC, USA).

## Results

### Characteristics of ACS Patients With LCX Occlusion

Of the 138 ACS patients with acute LCX occlusion, 47 (34%) had no significant ST-segment changes on the ECG (No-ST group), 47 (34%) had ST-segment elevation on the ECG (ST-E group), and 25 (18%) had precordial ST-segment depression (ST-D group; **Figures 1,2**). Nineteen patients (14%) were classified as having an “other” pattern. The characteristics of the 119 ACS patients with LCX occlusion allocated into 3 different electrocardiographic categories are presented in the **Table**. There were no significant differences in the clinical and demographic profiles among the 3 groups. However, compared with the ST-E and the ST-D groups, patients presenting with no ST-segment changes (No-ST group) had a significantly longer DTB time ( $P < 0.0001$ ). A DTB time  $\leq 90$  min was also significantly less frequent in the No-ST group ( $P = 0.0004$ ). In addition, compared with the No-ST and ST-E groups, the ST-D group presented with a more advanced Killip class ( $P = 0.003$ ), greater peak creatine phosphokinase (CPK;  $P = 0.007$ ) and creatine kinase-MB (CK-MB;  $P = 0.006$ ) concentrations, more frequent proximal LCX occlusion ( $P = 0.007$ ), and worse 1-year outcomes ( $P = 0.0034$ ).

### Coronary Risk Factors and Past Medical History

There were no significant differences in the coronary risk factors of hypertension, dyslipidemia, diabetes, current or past smoking, and family history of coronary artery disease among the 3 groups (**Table**). There were also no significant differences in the frequency of prior myocardial infarction, percutaneous coronary intervention, and coronary artery bypass grafting among the 3 groups (**Table**).

### Killip Class

The ST-D group had a significantly higher prevalence of heart failure of Killip Class III–IV on arrival at hospital ( $P = 0.003$ ) compared with the No-ST and ST-E groups (**Figure 3**).

### Baseline Medication

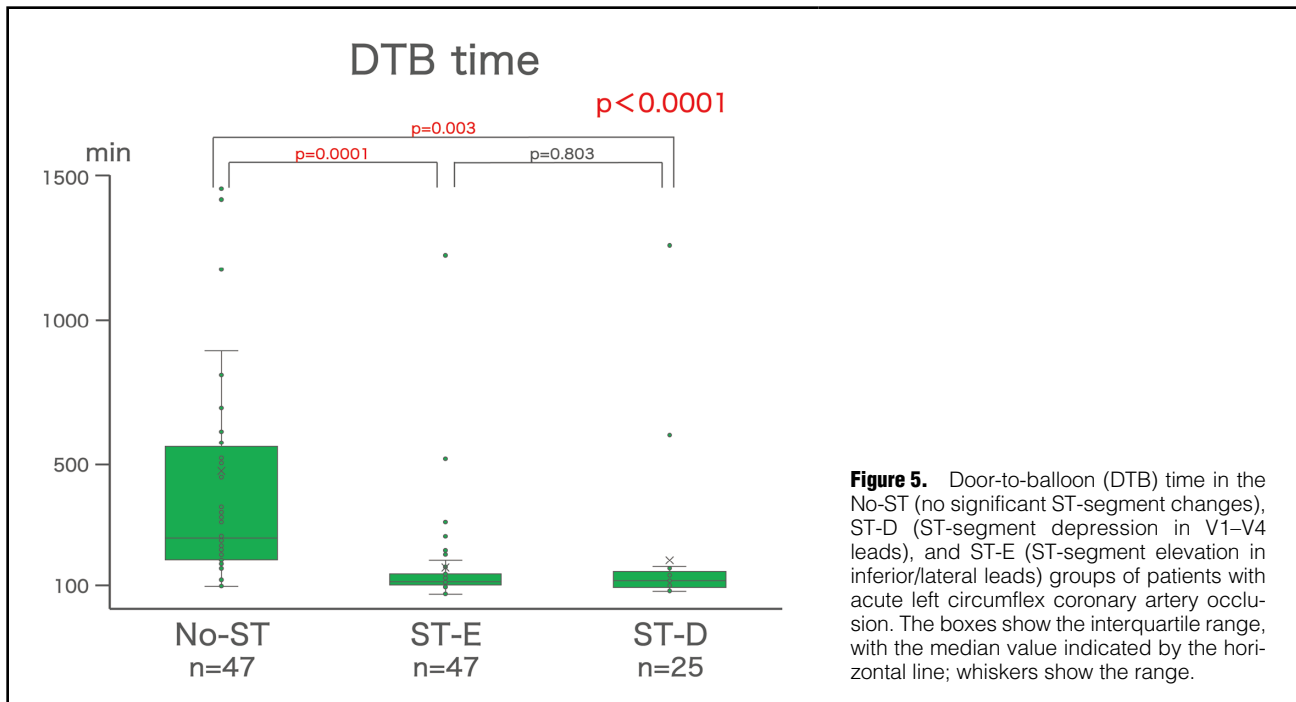
There were no significant differences in the use of antithrombotic therapy and medications for hypertension, dyslipidemia, and diabetes among the 3 groups (**Table**).

### Laboratory Findings

There were no significant differences in the laboratory findings collected at the time of arrival at hospital among the 3 groups. The median value of high-sensitivity cardiac troponin T was higher than the 99th percentile URL ( $> 0.014$  ng/mL) in all 3 groups (0.118 [IQR 0.025–0.445], 0.122 [IQR 0.031–0.412], and 0.083 [IQR 0.028–0.467] ng/mL in the No-ST, ST-E, and ST-D groups, respectively). Peak CPK and peak CK-MB were higher in the ST-D than No-ST and ST-E groups ( $P = 0.007$  and  $P = 0.006$ , respectively; **Figure 4**).

### Echocardiographic Left Ventricular Asynergy

All patients underwent an echocardiographic examination



for the identification of regional left ventricular wall motion abnormality before coronary angiography. Posterior left ventricular asynergy was identified in 31 (66%), 38 (81%), and 22 (88%) in the No-ST, ST-E, and ST-D groups, respectively ( $P=0.07$ ).

#### Location of the LCX Occlusion

There were significant differences in the location of the occluded LCX segment among the 3 groups ( $P=0.007$ ). Proximal LCX occlusion was found more frequently in the ST-D than No-ST and ST-E groups (52% vs. 34% and 13%, respectively; **Figure 3**). Distal LCX occlusion was commonly observed in the ST-E (74%) and No-ST (60%) groups.

#### DTB Time

The DTB time was significantly longer in the No-ST than ST-E and ST-D groups (median [IQR] 245 [170–562], 93 [83–121], and 97 [70–129] min, respectively;  $P<0.0001$ ; **Figure 5**). In addition, a DTB time  $\leq 90$  min was significantly less common in the No-ST than in the ST-E and ST-D groups (11% vs. 46% and 43%, respectively;  $P=0.0004$ ; **Figure 3**).

#### Outcomes

There were no significant differences in in-hospital mortality among the 3 groups (**Table**).

Short-term outcomes up to 1 year of follow-up were evaluated for major adverse cardiac and cerebrovascular events (MACCE), a composite of all-cause mortality, non-fatal myocardial infarction, any repeat coronary revascularization, and stroke. MACCE were found in 1 patient in the No-ST group, in 5 patients in the ST-E group, and in 7 patients in the ST-D group ( $P=0.0034$ ; **Table**).

## Discussion

### No ST-Segment Changes in Acute LCX Occlusion

The 12-lead surface ECG is the initial diagnostic test used

in patients presenting with symptoms suggestive of ACS. Typical ST-segment elevation on ECG is indicative of a coronary artery occlusion with transmural infarction and requires immediate coronary reperfusion.<sup>7,8</sup> ACS patients with ST-segment depression or no significant ST-segment changes are considered to have non-transmural infarction without vessel occlusion and are often not subjected to immediate coronary reperfusion.<sup>4,9</sup> In this regard, ACS with LCX occlusion often poses diagnostic problems, resulting in a delayed recognition of vessel occlusion. The ability to make a diagnosis of acute LCX occlusion by a standard 12-lead ECG is often difficult because over half of acute LCX occlusions present without characteristic ST-segment elevation on ECG.<sup>2,10</sup>

In the present study, one-third of ACS patients with acute LCX occlusion did not have any significant ST-segment changes on ECG, resulting in a significantly longer DTB time compared with patients presenting with ST-segment elevation or ST-segment depression. Despite the delay in reperfusion, prognosis was not worse for patients without ST-segment changes in the present study. It may be postulated that these patients possess more coronary collaterals and the culprit vessel may be supplying less extensive myocardial areas.

### Significance of ST-Segment Depression

Compared with patients without ST-segment changes and those with ST-segment elevation, patients presenting with ST-segment depression had a more advanced Killip class, higher peak levels of cardiac biomarkers, and more frequent proximal LCX occlusion suggestive of large infarcts. This result is consistent with a previous study of 314 patients with LCX occlusion, in which proximal LCX occlusion was most frequently observed in patients with ST-segment depression, whereas the distal LCX occlusion was often the culprit artery in the ST-segment elevation group.<sup>11</sup> It is conceivable that the size and location of the

culprit artery, the presence of coronary collaterals, and the size of the perfusion territory may play a role in the genesis of the different ST-segment patterns in patients with acute LCX occlusion. Therefore, the precordial ST-segment depression pattern should be interpreted with caution, because these patients appear to have large infarct sizes caused by proximal LCX occlusion, resulting in more advanced acute heart failure.

### LCX Occlusion Presenting as Non-ST-Elevation ACS

Many acute LCX occlusions remain undiagnosed because the standard 12-lead ECG is least sensitive for infarcts involving acute LCX occlusions.<sup>1,12,13</sup> A 12-lead surface ECG is able to detect acute LCX occlusion in ACS patients in only one-third to one-half of cases,<sup>2,10</sup> as also demonstrated in the present study. Compared with the occlusion of other major coronary arteries, it seems likely that patients with LCX occlusion have a tendency to present with non-ST elevation ACS (NSTEMI-ACS).<sup>14,15</sup> Furthermore, by subdividing NSTEMI-ACS with LCX occlusion into 2 groups (i.e., No-ST and ST-D groups), this study is the first to show that among patients with NSTEMI-ACS with LCX occlusion, those presenting with ST-segment depression had higher acute risk, such as larger infarct size, advanced acute heart failure, and worse short-term outcomes, than those without ST-segment changes.

### Recommendation to Overcome the Diagnostic Problem

Bedside echocardiography<sup>16</sup> and additional recording of posterior leads (V7–V9)<sup>17</sup> have been proposed to overcome diagnostic problems. These adjunct examinations have been in use for some time in clinical practice and have proved to be useful in the acute emergency setting. Indeed, echocardiographic posterior left ventricular asynergy was identified in 66% of patients without ST-segment changes in the present study. Improving diagnostic accuracy by using bedside echocardiography to identify regional left ventricular asynergy is therefore important when ACS is clinically suspected. It is also critical to avoid delayed reperfusion in patients without electrocardiographic changes. Further, additional recording of posterior leads (V7–V9) may also be of help in improving diagnostic accuracy, as recommended by several guidelines.<sup>18,19</sup> Although a modest improvement in diagnostic accuracy was reported previously,<sup>3</sup> posterior derivation is not routinely performed due to the time-consuming procedures involved, such as electrode repositioning to the patient's back with additional position changes of the patient to a lateral recumbent position. Recently, a synthesized 18-lead ECG, which uses the 12-lead ECG waveforms to mathematically derive the waveforms of the posterior leads and right chest leads, has been developed and started to be used clinically.<sup>20</sup> The reliability of this procedure was already validated, with high correlation between both actual and synthesized leads. Thus, a synthesized 18-lead ECG could be a practical, useful tool for improving the diagnostic accuracy in patients without ST-segment changes. The use of technologies such as body surface mapping,<sup>21</sup> computed tomography coronary angiography,<sup>22,23</sup> and cardiac magnetic resonance imaging<sup>24,25</sup> has been advocated, but these technologies are yet to enter routine practice.

### Study Limitations

First, because this is a retrospective study from a single institution, there may be missing values for study variables.

To avoid these limitations, patients were recruited consecutively and those with no available index ECG or coronary angiography were excluded. Second, the number of patients included in the study was modest. Third, posterior leads (V7–V9) were not routinely recorded as part of the baseline ECG recording in most patients.

### Conclusions

The present study demonstrated that one-third of ACS patients with LCX occlusion showed no significant ST-segment changes, resulting in delayed recognition of vessel occlusion and thus a significantly longer DTB time. Improving diagnostic accuracy by using bedside echocardiography to detect posterior left ventricular asynergy and/or recording of the posterior leads is important to avoid delayed reperfusion in patients without ECG changes. ACS patients presenting with ST-segment depression showed broader myocardial infarction caused by proximal LCX occlusion, more advanced acute heart failure, and worse short-term outcomes than patients without ST-segment changes. Therefore, patients with a precordial ST-segment depression pattern should be managed with caution.

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### Disclosures

The authors have no conflicts of interest to disclose.

### IRB Information

The present study was approved by the Ethics Committee of Chikamori Hospital (Reference no. 444).

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