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Relationship Between Attenuated Plaque Identified by Intravascular Ultrasound and Thrombus Formation After Excimer Laser Coronary Angioplasty

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ABSTRACT

Background: Excimer laser coronary angioplasty (ELCA) has been reported to be a safe and effective atherectomy device in percutaneous coronary intervention (PCI). However, thrombotic complications after ELCA have been occasionally observed. In this study, we evaluated the impact of attenuated plaque on thrombus formation and transient no-reflow after ELCA.

Methods: This study enrolled 58 lesions in 56 patients who underwent PCI with ELCA. It was a retrospective observational study at a single center. All lesions were imaged by intravascular ultrasound (IVUS) before and immediately after ELCA. On the plaque with ultrasound attenuation, attenuation angle per millimeter and attenuation length were measured. ELCA-induced thrombus was detected by IVUS, and transient no-reflow after ELCA was recorded.

Results: Thrombus was detected in 14 lesions (30 %), and transient no-reflow occurred in 3 lesions (5 %). Lesions with thrombus had a higher mean attenuation angle (median [interquartile range] 142° [112°-152°] vs. 64° [0°-115°]; $p = 0.001$), maximum attenuation angle (209° [174°-262°] vs. 86° [0°-173°]; $p < 0.001$), and longer attenuation length (12 mm [8 mm–17 mm] vs. 2 mm [0 mm–5 mm]; $p < 0.001$). Lesions with thrombus leading to transient no-reflow had a longer lipid length and a significantly higher troponin I level after PCI.

Conclusions: IVUS-identified attenuated plaque was strongly correlated with ELCA-induced thrombus. Furthermore, attenuation length may predict transient no-reflow.

1. Introduction

Excimer laser coronary angioplasty (ELCA) is a recognized adjunctive therapy used in percutaneous coronary intervention (PCI) for complex lesions. ELCA has shown particular effectiveness in the treatment of in-stent restenosis (ISR) [1], saphenous vein graft lesions [2], chronic total occlusion (CTO) [3], and thrombotic lesions [4]. The safety of ELCA has been demonstrated in clinical trials, and the overall complication rate has declined over time because of technical improvements and appropriate case selection [5–7]. On the other hand, a recent large registry study confirmed an increased risk of dissection, perforation, and slow flow with increased use of ELCA compared with non-ELCA interventions [8]. A previous large study reported that the incidence of angiographic thrombus formation after ELCA was 4.8 % and associated with ulcerated lesions, which were identified by coronary angiography [9]. However, there is a lack of sufficient intracoronary imaging data. Intravascular ultrasound (IVUS) is a useful tool to evaluate coronary lesion characteristics and detect thrombus formation. Some IVUS studies showed that attenuated plaque (hypoechoic or mixed atheroma with ultrasound attenuation but without calcification) is associated with a high rate of transient deterioration in coronary flow during PCI and a larger infarct size [10,11]. Those results suggest that atherothrombotic embolization might occur more often during PCI in patients with IVUS-detected attenuated plaque. We hypothesized that attenuated plaque is associated with thrombus formation and thrombotic events after ELCA. The study's aim was to evaluate the effect of attenuated

Abbreviations: CTO, chronic total occlusion; EEM, external elastic membrane; ELCA, excimer laser coronary angioplasty; ISR, in-stent restenosis; IVUS, intravascular ultrasound; PCI, percutaneous coronary intervention; TIMI, thrombolysis in myocardial infarction.

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plaque assessed by IVUS on ELCA-induced thrombus and transient no-reflow.

2. Materials and methods

2.1. Study design and population

We retrospectively reviewed a coronary angiography database of 120 consecutive lesions in 115 patients who underwent PCI with ELCA at Hiroshima University Hospital from January 2014 to December 2020. A study flow chart is reported in Fig. 1. We excluded lesions without IVUS before or after ELCA (n = 54 lesions) and lesions with thrombus before ELCA (n = 8 lesions). We analyzed IVUS imaging in the remaining 58 lesions in 56 patients. Eligible lesions were all imaged by IVUS before and immediately after ELCA. Lesions were divided into the ELCA-induced thrombus group (thrombus group) and the no-thrombus group (Fig. 1). This study was approved by the ethics committee of Hiroshima University Hospital. We were not required to obtain written informed consent from the participants because this was a retrospective observational study using data collected previously as part of routine clinical care.

2.2. Catheter procedure

Anticoagulation during PCI was achieved with intravenous unfractionated heparin boluses to maintain an activated clotting time ≥ 300 s. All ELCA procedures used the CVX-300 excimer laser system (Spectranetics, Colorado, CO, USA), consisting of a pulsed xenon–chlorine laser catheter capable of delivering excimer energy densities from 30 to 80 mJ/mm^2 at pulse repetition rates of 25 to 80 Hz. The operators chose to treat the patients with 0.9-, 1.4-, or 1.7-mm excimer laser catheters on the basis of the intracoronary imaging findings. The excimer energy densities and repetition rates were determined by the operators. The operators advanced the laser catheter at a speed of 0.5 mm/s while an assistant injected saline at 2–3 ml/s [12]. After ELCA, angiography and IVUS were first performed, followed by PCI using a standard technique. We occasionally administered intracoronary injection of vasodilators, such as nitroprusside and nicorandil, to improve coronary slow-/no-reflow. ELCA-induced transient no-reflow was defined as decreased thrombolysis in myocardial infarction (TIMI) flow grade immediately after ELCA, and a TIMI flow grade of 3 at completion of the procedure [13].

2.3. IVUS imaging and analysis

IVUS imaging used a mechanical IVUS system (VISIWAVE for Terumo Corporation or VISICUBE for Ueda Japan Radio Corporation, Japan) with 40-MHz (ViewIT) or 60-MHz (AltaView) imaging catheters (both by Terumo Corporation, Tokyo, Japan). An automated pullback device was used at a rate 0.5–3.0 mm/s to perform pullback IVUS imaging. Computerized analysis (VISIATLAS, Terumo Corporation, Tokyo, Japan) was used to perform off-line quantitative IVUS analysis. Lesions subjected to ELCA were analyzed. The lumen area and external elastic membrane (EEM) were measured, and the plaque area was calculated as follows: EEM area − lumen area. The cross-sectional area per millimeter was measured for these variables. The minimum lumen area site was determined by visual screening of all contiguous frames. Lesion length was defined as the cross-sectional area with a plaque burden >40 %. In the plaque with ultrasound attenuation, the angle of the attenuation (attenuation angle) per millimeter was measured and summed to create an overall attenuation angle. The mean attenuation angle was calculated as the overall attenuation angle divided by the attenuation length. The length of attenuation was calculated from the number of frames in which attenuation was identified (Fig. 2). Thrombus was an intraluminal mass having a layered or lobulated appearance, evidence of blood flow within the mass, and speckling or scintillation [14]. A representative case with ELCA-induced thrombus is shown in Fig. 3.

2.4. Statistical analysis

Statistical analyses were performed with the JMP statistical software (version 16.0, SAS Institute, Inc. Cary, NC, USA). Data were analyzed at the patient and lesion levels according to the thrombus formation. The χ^2 test or Fisher's exact test, as appropriate, was used to compare categorical variables. The Wilcoxon rank-sum test was used to compare continuous variables and presented as the median (interquartile range). A p-value < 0.05 was considered to be indicative of statistical significance.

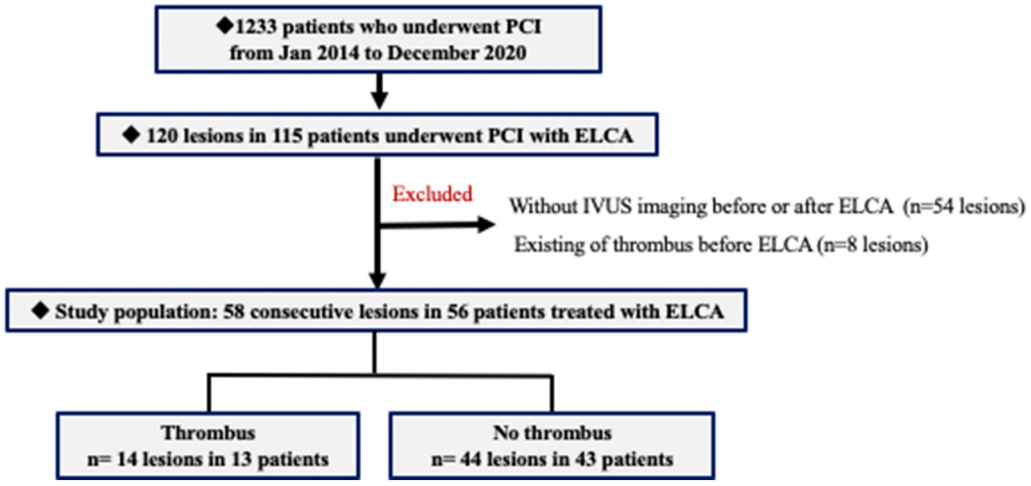

Fig. 1. Study flowchart.
We retrospectively reviewed coronary angiography database of 120 consecutive lesions in 115 patients who underwent PCI with ELCA at Hiroshima University Hospital from January 2014 to December 2020. We excluded lesions without IVUS before or after ELCA (n = 54 lesions) and lesions with thrombus before ELCA (n = 8 lesions). We analyzed images of IVUS in the 58 lesions in 56 patients. Of 58 lesions, 14 lesions with thrombus were compared with 44 lesions with no thrombus.
Abbreviations ELCA: excimer laser coronary angioplasty, IVUS: intravascular ultrasound, PCI: percutaneous coronary intervention.

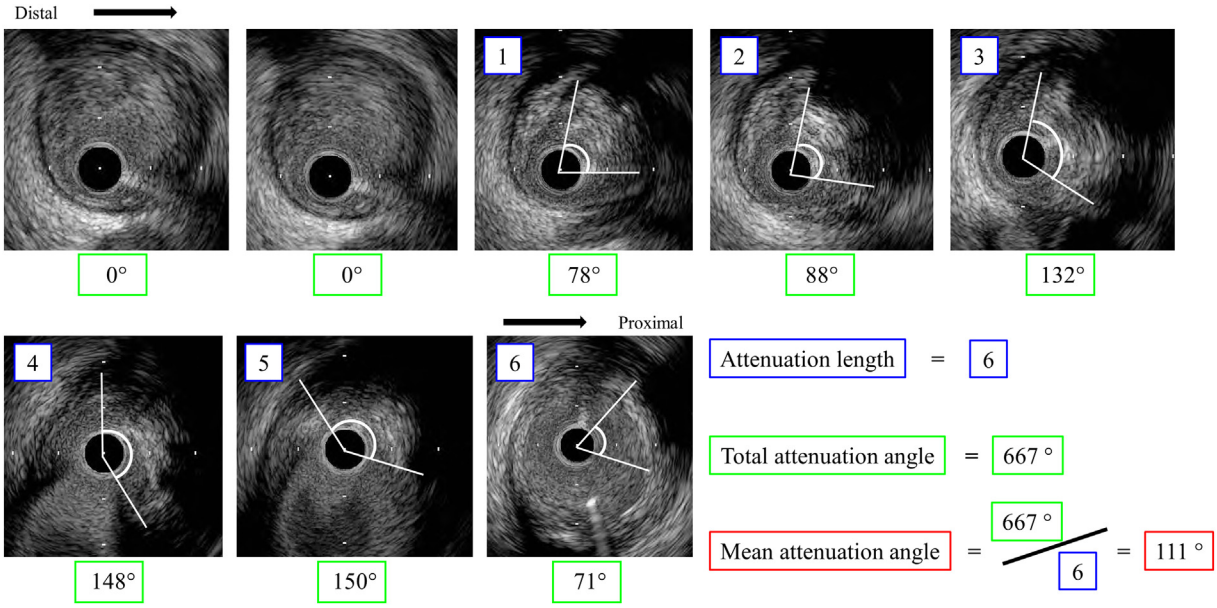

Fig. 2. Measurement of attenuation parameters in the analysis of intravascular ultrasound.
Measurement of lipid parameters in the analysis of intravascular ultrasound.
In the plaque with ultrasound attenuation, the attenuation length is calculated based on the number of frames in which attenuation is identified (blue frames). The attenuation length in this case was six. The angle of the attenuation (attenuation angle) per millimeter is measured and summed to create an overall attenuation angle (green frames). The total attenuation angle in this case was 667° (78° + 88° + 132° + 148° + 150° + 71°). The mean attenuation angle is calculated as the overall attenuation angle divided by the attenuation length (red frames). The mean attenuation angle in this case was 111°.

3. Results

Of the total 58 lesions, ELCA-induced thrombus was detected in 14 (30 %) lesions (Fig. 1). In addition, thrombus with transient no-reflow occurred in 3 (5 %) lesions. Patient characteristics were comparable except for left atrial diameter measured by transthoracic echocardiography (Table 1). Lesion and procedural characteristics according to the ELCA-induced thrombus are shown in Table 2. There were no significant differences in the lesion and procedural characteristics, including ELCA catheter size, maximum energy densities, and repetition rate. The frequency of thrombus formation tended to be higher after PCI of the right coronary artery.

Lesions with ELCA-induced thrombus had a higher mean attenuation angle (median [interquartile range] 142° [112°–152°] vs. 64°

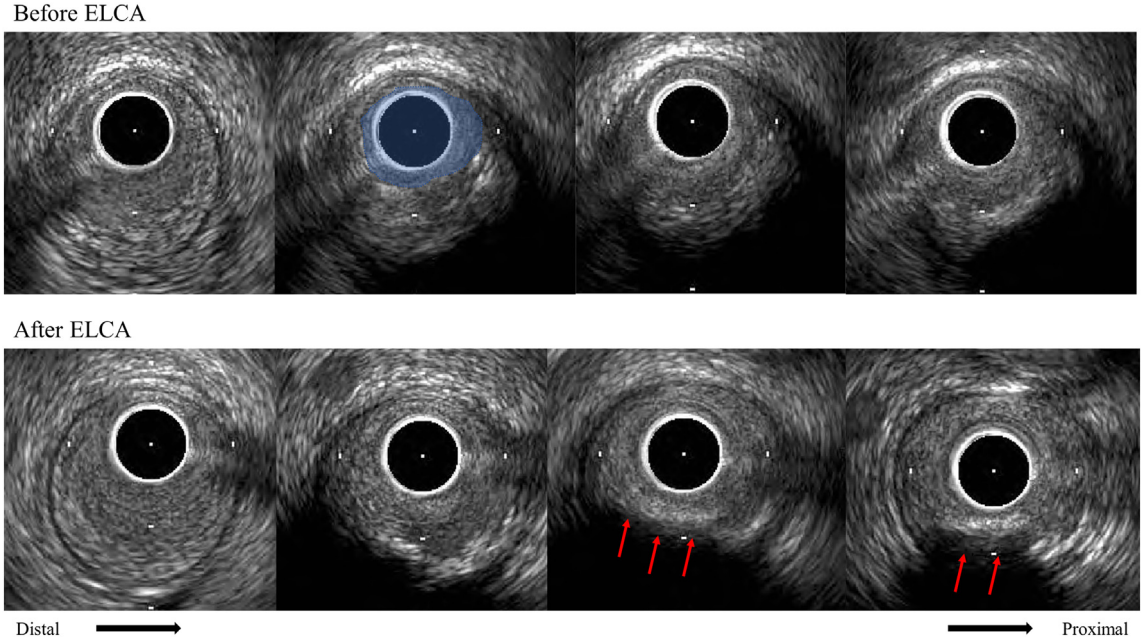

Fig. 3. A representative patient with an ELCA-induced thrombus.
Attenuated plaque detected by IVUS before ELCA. IVUS showed plaque disruption and thrombus after ELCA (red arrow). The blue area distinguishes a flap of the plaque.
Abbreviations ELCA: excimer laser coronary angioplasty, IVUS: intravascular ultrasound.

Table 1
Baseline clinical characteristics of the study patients.

	Thrombus (n = 13)	No thrombus (n = 43)	p-Value
Age (years)	71 (62–78)	76 (66–82)	0.35
Male	11 (85)	32 (73)	0.66
Body mass index (kg/m^2)	21.2 (18.4–26.3)	23.2 (21.5–26.0)	0.39
Clinical history			
Hypertension	13 (100)	38 (88)	0.19
Diabetes mellitus	8 (62)	28 (65)	0.81
Dyslipidemia	11 (85)	34 (79)	0.65
Current smoker	14 (61)	16 (37)	0.12
Family history of CAD	4 (31)	6 (14)	0.16
Hemodialysis	0 (0)	5 (12)	0.19
Medications			
Dual antiplatelet therapy	12 (92)	41 (95)	0.66
Aspirin	11 (85)	40 (93)	0.35
Clopidogrel	4 (31)	19 (44)	0.38
Prasugrel	9 (69)	23 (53)	0.31
Anticoagulant therapy	5 (38)	7 (16)	0.08
ACEIs or ARBs	7 (54)	23 (53)	0.98
Beta blockers	9 (69)	27 (63)	0.67
Statins	11 (85)	37 (86)	0.89
Calcium channel blocker	4 (31)	16 (37)	0.67
Laboratory data			
Platelet	176 (160–241)	194 (152–233)	0.80
Hemoglobin (g/dl)	13.3 (11.9–14.2)	11.9 (11.2–14.1)	0.21
NT-proBNP (pg/ml)	683 (231–1917)	684 (169–3154)	0.93
Serum creatinine (mmol/L)	0.99 (0.85–1.43)	1.06 (0.82–1.71)	0.71
Estimated GFR (ml/min/1.73 m^2)	43 (39–69)	49 (30–70)	0.64
CRP (mg/dl)	0.63 (0.07–0.92)	0.09 (0.05–0.48)	0.18
Hemoglobin A1c (%)	7.1 (5.8–7.3)	6.5 (6.0–7.1)	0.34
LDL cholesterol (mg/dL)	89 (68–96)	73 (62–84)	0.09
HDL cholesterol (mg/dL)	42 (38–46)	50 (36–60)	0.08
Triglycerides (mg/dL)	121 (86–173)	128 (92–157)	0.81
CK-MB (ng/ml)	9 (8–13)	9 (7–12)	0.59
Troponin I (pg/ml)	0.04 (0.02–0.69)	0.03 (0.02–0.05)	0.45
Echocardiographic data			
LVEF (%)	57 (38–59)	59 (48–65)	0.17
LVDd (mm)	51 (44–53)	49 (45–52)	0.69
LAD (mm)	41 (38–48)	37 (35–42)	0.03

Notes: Data are presented as mean ± standard deviation, median (interquartile range), or count (percentage).
Abbreviations: ACEI, angiotensin converting-enzyme inhibitor; ARB, angiotensin receptor blocker; CAD, coronary artery disease; CK-MB, creatine kinase–myocardial band; CRP, C-reactive protein; GFR, glomerular filtration rate; HDL, high-density lipoprotein; LAD, left atrium diameter; LDL, low-density lipoprotein; LVDd, left ventricular diameter at end diastole; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-brain natriuretic peptide.

Table 2
Baseline lesion and procedural characteristics according to the thrombus formation.

	Thrombus (n = 14)	No thrombus (n = 44)	p-Value
Culprit lesion			0.07
RCA	8 (57)	9 (20)	
LADA	4 (29)	21 (48)	
LCx	2 (14)	13 (30)	
LMCA	0 (0)	1 (2)	
Acute coronary syndrome	1 (7)	2 (5)	0.71
In-stent restenosis	4 (29)	22 (50)	0.16
Chronic total occlusion	4 (29)	10 (23)	0.65
Initial TIMI flow grade			0.44
Grade 0	4 (29)	13 (30)	
Grade 1	2 (14)	3 (7)	
Grade 2	2 (14)	9 (20)	
Grade 3	6 (43)	19 (43)	
Use of two size of ELCA catheter	3 (21)	10 (23)	0.91
ELCA catheter			
0.9-mm ELCA catheter	2 (14)	11 (25)	0.40
1.4-mm ELCA catheter	9 (64)	25 (57)	0.62
1.7-mm ELCA catheter	6 (43)	18 (41)	0.89
0.9 + 1.4-mm ELCA catheter	0 (0)	3 (7)	0.31
0.9 + 1.7-mm ELCA catheter	1 (7)	3 (7)	0.96
1.4 + 1.7-mm ELCA catheter	2 (14)	4 (9)	0.57
Maximum energy densities (mJ/mm^2)	60 (55–60)	60 (55–60)	0.52
Repetition rate (Hz)	40 (25–40)	40 (30–40)	0.23
Drug eluting stent	9 (64)	21 (48)	0.28
Drug coated balloon	4 (29)	23 (52)	0.12

Notes: Data are presented as mean ± standard deviation, median (interquartile range), or count (percentage).
Abbreviations: ELCA, excimer laser coronary angioplasty; LADA, left anterior descending artery; LCx, left circumflex artery; LMCA, left main coronary artery; RCA, right coronary artery; TIMI, thrombolysis in myocardial infarction.

[0°–115°]; p = 0.001), maximum attenuation angle (209° [174°–262°] vs. 86° [0°–173°]; p < 0.001), and a longer attenuation length (12 mm [8 mm–17 mm] vs. 2 mm [0 mm–5 mm]; p < 0.001) (Table 3 and Fig. 4A). Lesions with ELCA-induced thrombus that led to transient no-reflow had a longer attenuation length (12 mm [5 mm–18 mm] vs. 3 mm [0 mm–9 mm]; p = 0.04) (Fig. 4B). Changes in myocardial enzymes during PCI are shown in Table 4. There was no significant difference between the thrombus and no-thrombus groups in terms of the change of myocardial enzymes. However, after PCI, thrombus leading to transient no-reflow was associated with a significantly higher troponin I level in patients with thrombus and no-reflow (10.90 pg/ml [1.23 pg/ml–16.90 pg/ml]) than in patients with no thrombus and thrombus without no-reflow (0.49 pg/ml [0.21 pg/ml–1.41 pg/ml]; p = 0.04) and a significantly greater change of troponin I level in patients with thrombus and no-reflow (10.84 pg/ml [1.11 pg/ml–16.84 pg/ml]) than in patients with no thrombus and thrombus without no-reflow (0.38 pg/ml [0.11 pg/ml–1.27 pg/ml]; p = 0.02).

4. Discussion

The principal study finding was that attenuated plaque detected by IVUS was strongly associated with thrombus formation immediately after ELCA. In addition, a significant interaction was observed between attenuation length and thrombus formation leading to transient no-reflow after ELCA. Patients with thrombus and transient no-reflow after ELCA had a significantly higher troponin I levels after the procedure. To our knowledge, this is the first report to show relationships between plaque features assessed by IVUS and ELCA-induced thrombus formation and transient no-reflow in patients who underwent PCI using ELCA.

Intraprocedural thrombotic events were relatively infrequent by angiography, occurring in <1 % of patients undergoing PCI, but were strongly associated with subsequent ischemic events [15]. A previous study reported that the incidence of thrombus formation during PCI

Table 3
Findings on intravascular ultrasound.

	Thrombus (n = 14)	No thrombus (n = 44)	p-Value
Reference (proximal)			
EEM CSA (mm^2)	16.0 (13.4–17.6)	15.3 (11.5–19.6)	0.59
Lumen CSA (mm^2)	7.4 (5.8–9.8)	6.0 (5.0–9.4)	0.24
Plaque CSA (mm^2)	8.6 (7.6–9.0)	8.5 (5.3–10.5)	0.80
Reference (distal)			
EEM CSA (mm^2)	14.1 (10.1–17.5)	12.1 (7.4–16.3)	0.34
Lumen CSA (mm^2)	7.1 (5.3–10.5)	4.8 (3.0–6.6)	0.01
Plaque CSA (mm^2)	5.7 (3.9–7.9)	6.5 (3.6–8.9)	0.68
Lesion at MLA			
EEM CSA (mm^2)	13.4 (10.9–16.5)	12.8 (10.6–20.9)	0.57
Lumen CSA (mm^2)	2.0 (1.7–2.5)	1.8 (1.5–2.5)	0.55
Plaque CSA (mm^2)	11.3 (9.1–14.6)	11.0 (8.2–13.8)	0.56
Lesion length (mm)	18.3 (13.4–27.6)	15.2 (11.4–23.4)	0.29
Attenuation parameter			
Mean attenuation angle (°)	142 (112–152)	64 (0–115)	0.001
Maximum attenuation angle (°)	209 (174–262)	86 (0–173)	<0.001
Attenuation length (mm)	10 (6–14)	2 (0–5)	<0.001

Notes: Data are presented as mean ± standard deviation, median (interquartile range), or count (percentage).
Abbreviations: CSA, cross-sectional area; EEM, external elastic membrane; MLA, minimum lumen area.

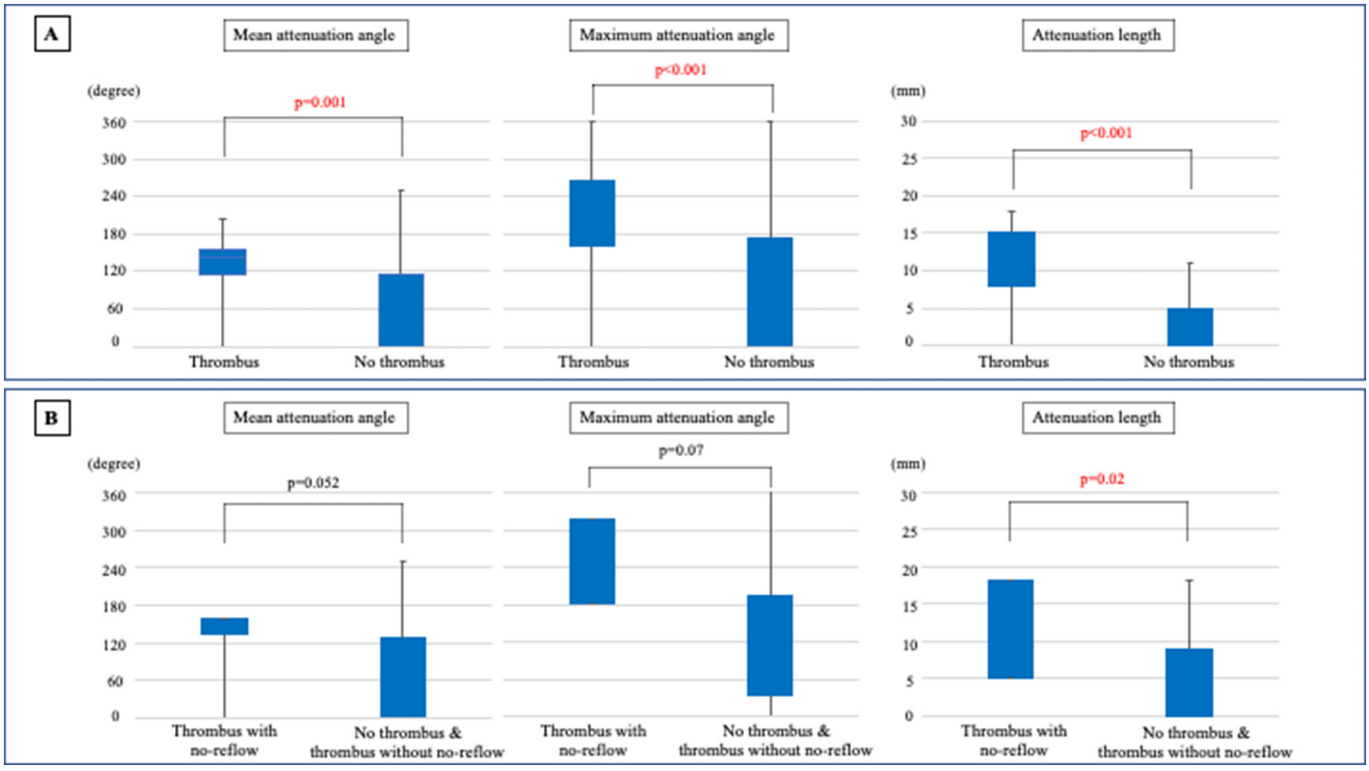

Fig. 4. A Attenuation parameters according to the occurrence of thrombus formation.
Lesions with thrombus had a higher mean attenuation angle, maximum attenuation angle, and longer attenuation length.
B Attenuation parameters according to the occurrence of no-reflow.
Lesions with thrombus that led to transient no-reflow show longer attenuation length (12 mm [5 mm–18 mm] vs. 3 mm [0 mm–9 mm]; p = 0.04).

using ELCA was detected by angiography in 4.8 % [9]. In the present study, thrombus formation just after ELCA was detected in 30 % of patients by IVUS. The results suggested that the frequency of thrombus formation during PCI using ELCA might be relatively high. To prevent intraprocedural thrombotic events, it would be very useful to detect lesion characteristics indicating the possibility of an intraprocedural thrombotic event after ELCA, especially assessed by intracoronary imaging.

Table 4
Periprocedural myocardial enzymes.

	Thrombus (n = 13)	No thrombus (n = 43)	p-Value
CK-MB pre PCI (ng/ml)	9 (8–13)	9 (7–12)	0.59
CK-MB post PCI (ng/ml)	13 (9–27)	12 (8–15)	0.37
ΔCK-MB (ng/ml)	3.0 (0–7.0)	2.0 (0–4.0)	0.64
Troponin I pre PCI (pg/ml)	0.04 (0.02–0.15)	0.03 (0.02–0.05)	0.52
Troponin I post PCI (pg/ml)	1.11 (0.40–8.21)	0.49 (0.17–1.45)	0.05
ΔTroponin I (pg/ml)	0.38 (0.24–3.37)	0.43 (0.11–1.38)	0.53

	Thrombus with no-reflow (n = 3)	No thrombus & thrombus without no-reflow (n = 53)	p-Value
CK-MB pre PCI (ng/ml)	9 (8–12)	9 (7–13)	0.94
CK-MB post PCI (ng/ml)	34 (9–70)	13 (8–15)	0.18
ΔCK-MB (ng/ml)	25 (1–58)	2 (0–4)	0.10
Troponin I pre PCI (pg/ml)	0.06 (0.06–0.12)	0.03 (0.02–0.05)	0.07
Troponin I post PCI (pg/ml)	10.90 (1.23–16.90)	0.49 (0.21–1.41)	0.04
ΔTroponin I (pg/ml)	10.84 (1.11–16.84)	0.38 (0.11–1.27)	0.02

Abbreviations: CK-MB, creatine kinase–myocardial band; PCI, percutaneous coronary intervention.

The current study demonstrated the close relationship between ELCA-induced thrombus formation and the lesion characteristics of attenuated plaque assessed by IVUS. IVUS-detected attenuated plaque is common in acute coronary syndrome and is recognized as representing vulnerable atherosclerotic lesions. A virtual histology IVUS study confirmed that IVUS-detected attenuated plaques were associated with a large amount of necrotic core [16]. In a multidetector computed tomography study, IVUS-detected attenuated plaques typically presented as low-density lipid-rich tissue [17]. Lipid-rich plaques are friable and easily disrupted during PCI, predisposing to intracoronary thrombus [18]. We speculate that these mechanisms of ELCA-induced thrombus formation are as follows. Physical stimulation of an ELCA catheter or energy from an excimer laser can destroy a fibrous cap, leading to the exposure of lipids and subsequent thrombus formation. In addition to injuries of the fibrous cap, laser-independent consequences of deep-vessel wall injury might be a cause of thrombus formation [19]. ELCA reportedly vaporizes thrombus and plaque [20], reduces the risk of distal embolization [21], and suppresses platelet aggregation [22]. ELCA has been used in vessels with a large thrombus and plaque burden with the expectation of achieving those potentially beneficial effects. However, caution may be needed when ELCA is performed on lesions with attenuated plaques, especially those with a greater attenuation length.

In the present study, patients with thrombus leading to transient no-reflow had significantly higher troponin I levels after PCI than patients without thrombus and transient no-reflow. Of the total 58 lesions, thrombus with transient no-reflow occurred in 3 (5 %) lesions. We observed no incidence of persistent no-reflow after ELCA in our study. A multicenter cohort study showed that the incidence of no-reflow occurred in 4.8 % of patients, and the no-reflow phenomenon during PCI was strongly associated with adverse clinical outcomes, regardless of whether the phenomenon was transient or persistent [23]. In some lesions with ELCA-induced thrombus and transient no-reflow, we simultaneously found ruptured plaques that could not be identified by

intracoronary imaging before ELCA (Fig. 3). Distal atherothrombotic embolization is considered to be the main reason of ELCA-induced transient no-reflow. In the present study, lesions with thrombus leading to transient no-reflow had a longer attenuation length. A previous study that calculated the lipid index as the average lipid angle multiplied by the lipid length as shown on IVUS and optical coherence tomography demonstrated that a larger maximum lipid angle and lipid index were associated with no-reflow after primary PCI [24]. When the vascular wall is injured during PCI, a large amount of lipid content can flow into the coronary artery, increasing local thrombogenicity [25]. Assessments of plaque morphology with intracoronary imaging prior to ELCA may have an important role in the detection of lesions having high risk of no-reflow.

In the present study, ISR lesions had a lower mean attenuation angle, maximum attenuation angle, and attenuation length in the IVUS (Supplemental figure). Neoatherosclerosis can be detected by intracoronary imaging as a signal attenuation [26]. A previous autopsy study demonstrated that in-stent neoatherosclerosis was associated with vulnerability and intraluminal thrombus [27]. In the current study, no ISR lesions had transient no-reflow, and all ISR lesions with an ELCA-induced thrombus had some degree of attenuated plaque detected by IVUS. Even in ISR lesions, the attenuation parameters, possibly indicating neoatherosclerosis, may predict thrombotic complications during the ELCA procedure.

4.1. Study limitations

There were some study limitations that should be considered. First, this was a single center, retrospective, observational study. Only 58 lesions were evaluated. The study's ability to detect the relationship between attenuated plaque and thrombus formation after ELCA was therefore limited. A future study using a larger sample size is needed to confirm our findings. Second, we used 40-MHz IVUS and 60-MHz IVUS. IVUS frequency can affect the degree of signal penetration. Furthermore, given the limited resolution of IVUS, the incidence of thrombus might be underestimated. Optical coherence tomography is more sensitive for detecting thrombus in vivo. Third, because ELCA was performed at the operator's discretion, selection bias might have influenced the results. Fourth, the baseline patient and lesion characteristics between the two groups were not comparable, such as left atrial diameter and the culprit lesion, which could have affected the relationship between attenuated plaque and thrombotic complications. Fifth, elevation in periprocedural myocardial enzymes can be caused not only by ELCA but also by other procedures such as balloon inflation and stent implantation. However, transient no-reflow associated with periprocedural myocardial injury in the present study was most likely caused by ELCA because it was defined as a decrease in TIMI flow grade immediately after ELCA. Finally, various conditions such as ISR, CTO, and acute coronary syndrome were included in this study. In-stent neoatherosclerosis is histologically characterized by an accumulation of lipid-laden foamy macrophages with or without necrotic core formation and/or calcification within the neointima. There is no communication between the lesion within the neointima and the underlying native atherosclerosis [26]. In future research evaluating the effect of ELCA, it would be useful to separately analyze neoatherosclerosis and atherosclerosis in native coronary arteries.

5. Conclusions

Attenuated plaque identified by intracoronary imaging was strongly correlated with ELCA-induced thrombus. Furthermore, attenuation length may predict thrombus formation leading to transient no-reflow. ELCA-induced transient no-reflow might contribute to the elevation of troponin I levels after PCI. Further studies should be advocated to investigate the clinical prognostic value of the relationship between plaque features in culprit lesions assessed by IVUS and ELCA-induced thrombus formation in patients who have undergone PCI using ELCA and to select better therapeutic options to reduce cardiovascular events.

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CRediT authorship contribution statement

Dr. HI had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Study conception and design: HI, TN. Analysis and interpretation of data: TN, HI, AT, YM, TH, NW, YS and YN. Drafting of the manuscript: TN. Critical revision of the manuscript for important intellectual content: HI. Final approval of manuscript: TN, HI, AT, YM, TH, NW, YS and YN.

Declaration of competing interest

None of the authors have any conflicts of interest or financial relationships related to this study.

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None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.carrev.2022.12.010.

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