論　文　内　容　要　旨

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

（エキシマレーザー冠動脈形成術後の血栓形成と血管内超音波で検出される Attenuated plaque との関連性）

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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. The aim of this study was to evaluate the impact of attenuated plaque on thrombus formation and transient no-reflow after ELCA.

Methods

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. We excluded lesions without IVUS before or after ELCA (n = 54 lesions) and lesions with thrombus before ELCA (n = 8 lesions). We analyzed IVUS imagings 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 and the no-thrombus group. 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. After ELCA, angiography and IVUS were first performed followed by PCI using a standard technique. In the plaque with ultrasound attenuation, attenuation angle per mm 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). Patients with thrombus leading to transient no-reflow had significantly higher amount of change of troponin I level (10.84 pg/ml [1.11 pg/ml–16.84 pg/ml] in patients with thrombus and no-reflow vs. 0.38 pg/ml [0.11 pg/ml–1.27 pg/ml] in patients with no thrombus and thrombus without no-reflow, $p = 0.02$).

Discussion

A previous study reported that the incidence of thrombus formation during PCI using ELCA was detected by angiography in 4.8%. 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.

IVUS-detected attenuated plaque is common in acute coronary syndrome and recognized as representing vulnerable atherosclerotic lesions. A virtual histology IVUS study confirmed that IVUS-detected attenuated plaque were associated with a large amount of necrotic core. In a multidetector computed tomography study, IVUS-detected attenuated plaque presented typical low density of lipid-rich tissue. Lipid-rich plaques are friable and easily disrupted during PCI, predisposing to intracoronary thrombus. We speculate that the 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 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. ELCA reportedly vaporized thrombus and plaque, reduced the risk of distal embolization, and suppressed platelet aggregation. ELCA has been used in vessels with a large thrombus and plaque burden, with the expectation of these potentially beneficial effects. However, caution may be needed when ELCA is performed in lesions with attenuated plaque, especially longer 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. 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. 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. 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. When the vascular wall is injured during PCI, a large amount of lipid content can flow into the coronary artery, increasing local thrombogenicity. 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.

Conclusions

Attenuated plaque identified by IVUS strongly correlated with ELCA-induced thrombus. Furthermore, Attenuation length may predict transient no-reflow.