

In Vivo Lipid Core Plaque Modification With Percutaneous Coronary Revascularization: A Near-Infrared Spectroscopy Study

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ABSTRACT: Background. Coronary revascularization is associated with no-reflow phenomenon and elevation of cardiac biomarkers. This may occur due to plaque modification. We used near-infrared spectroscopy (NIRS) to evaluate lipid core plaque (LCP) modification with coronary revascularization and its correlation with periprocedural myocardial infarction. **Methods.** Patients presenting to the cardiac catheterization laboratory who underwent NIRS, NIRS/intravascular ultrasound (IVUS) were reviewed and their lipid core burden index (LCBI) was assessed. Using fuzzy c-means clustering algorithm, the coronary was divided into three zones and the lipid burden was recalculated. Its correlation to postprocedure troponin elevation and outcomes with a mean follow-up of 42 months were studied. **Results.** A total of 77 coronaries were evaluated. There was an overall decrease in the LCBI after percutaneous revascularization ($P < .0001$). Using fuzzy c-means clustering algorithm, there was always a decrease in the lipid burden at the site of the percutaneous revascularization ($P < .0001$). Postprocedure troponin elevation was only noted in patients with an axial shifting of the LCP. There was no difference in long-term outcomes due to the degree of reduction of lipid burden or its axial. **Conclusions.** Plaque modification may be performed successfully using interventional methods and can be evaluated with NIRS. Axial plaque shifting is an acute prognostic marker for postprocedure myocardial infarction.

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Key words: near-infrared spectroscopy (NIRS), no reflow, lipid core plaque (LCP)

It is well known that thrombotic occlusion of a high-risk coronary plaque is the leading cause of major morbidity and mortality. Lipid core plaques (LCPs) may be identified with near-infrared spectroscopy (NIRS). Coronary revascularization is associated with the slow and no-reflow phenomenon along with troponin release.¹⁻⁹ This is especially noted in patients with a lipid core burden index (LCBI) of ≥ 500 .¹ Evacuated craters have been identified under stent planes and remain unhealed after drug-eluting stent implantations.^{10,11} One would therefore surmise that LCP modification would occur alongside coronary revascularization with the release of lipid contents and atherosclerotic debris.

To address this issue, we employed NIRS before and after intervention to see the manipulation of LCPs with angioplasty and stenting.

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The outcomes in terms of increase and decrease in lipid burden after stent placement and the relationship to postprocedure troponin elevations were assessed. These patients were then followed for any target lesion, vessel revascularization, recurrent angina, repeat angiography, myocardial infarction, and/or death with a mean follow-up of 42 months.

Methods

Patients presenting to the cardiac catheterization laboratory who underwent NIRS, NIRS/intravascular ultrasound (IVUS) (Infra-RedX)¹²⁻¹⁵ were evaluated after Institutional Review Board approval was obtained. Patients were presented to the cardiac catheterization laboratory for a variety of indications including acute coronary syndromes (ST-elevation myocardial infarction, non-ST elevation myocardial infarction, and unstable angina) and coronary artery disease with angina and abnormal stress tests.

The NIRS and NIRS/IVUS catheter is a 3.2 Fr short-monorail sheath-based design similar to rotating IVUS catheters. Within the short-monorail imaging sheath, the imaging core pulls back at 0.5 mm/s and rotates at 240 rpm. Raw spectra are acquired at a rate of about 40 Hz (1 spectrum every 25 ms), with successive spectra taken after about 0.01 mm of pullback motion and 36° of rotary motion. Spatial filtering and image processing of the raw data produced an image with data points every 0.1 mm and 1°.

Anatomic coregistration of the near-infrared spectroscopy with angiography was performed, with each scan starting from a predefined fiducial branch.

LCBI is a quantitative summary metric of the LCP content in the chemogram and is a fraction of the chemogram image pixels above the probability of 0.6.¹²

We then proceeded with segmentation of the chemograms into three zones, which was achieved by using MATLAB software and the fuzzy c-means clustering algorithm.^{16,17} Each image was converted to a gray-scale image and segmented into two classes, with the lower-intensity value class representing absence of lipid and higher-intensity value class representing lipid. Zone 1 was the segment where the percutaneous revascularization was performed, zone 2 was the region proximal to zone 1, and zone 3 was distal to zone 1. Pre- and poststenting segmented scans were then evaluated for lipid burden using the fuzzy c-means clustering algorithm.

Results

A total of 77 coronaries were evaluated. A significant percentage of patients had comorbid conditions (Table 1). One

Table 1. Demographics (n = 77).

Male	70%
Female	30%
Dyslipidemia	85%
Hypertension	65%
Peripheral arterial disease	5%
Diabetes	40%
COPD	5%
End-stage renal disease	5%
Cerebrovascular accident	5%
Carotid stenosis	5%
Smoking history	20%
GERD	5%
COPD = chronic obstructive pulmonary disease; GERD = gastroesophageal reflux disease.	

patient developed a dissection from the percutaneous revascularization and developed no-reflow and was sent for emergency surgical revascularization. The remaining patients underwent successful percutaneous coronary revascularization with TIMI 3 flow at the completion of the procedure.

Upon evaluating the chemograms, the LCBI had decreased significantly ($P<0.0001$) (Figure 1). Preprocedure mean LCBI was 177.51 and postprocedure mean LCBI was 109.69 (Figure 1). However, there were some chemograms where the LCBI had increased, and upon visual examination of the chemogram, lipid was noted after stenting where there was none prior to the stenting (Figure 6). When the chemogram was segmented to the three zones, there was always a decrease in the lipid burden at the site of the percutaneous revascularization ($P<.0001$) (Figures 2, 5, and 6). In instances where there seemed to have been an increase in the total LCBI, there was always a decrease in lipid burden in zone 1 and an increase in zones 2 or 3 (Figures 3 and 4). This was suggestive of the LCP shifting axially along the coronary artery.

Postprocedure troponin elevation was noted in 3 (3.8%). All these patients had LCP shift proximally and distally. The maximum troponin elevation was 1.5 ng/mL. Patients who had no plaque shifting axially had no rise in their postprocedure troponins.

At a mean follow-up of 42 months, the all-cause mortality rate was 5.1% (n = 4). Myocardial infarction occurred in 1 (1.2%). Target lesion revascularization occurred in 4 (5.1%) and target vessel revascularization in 2 (2.5%). Recurrent angina and abnormal stress tests occurred in 12 (15.58%). None of these variables correlated with the degree of decrease in plaque burden and LCP shifting proximal and distal to zone 1.

Discussion

The NIRS, NIRS/IVUS^{1,13-15} system provides an accurate identification of lipid-core plaques. Plaque modification may

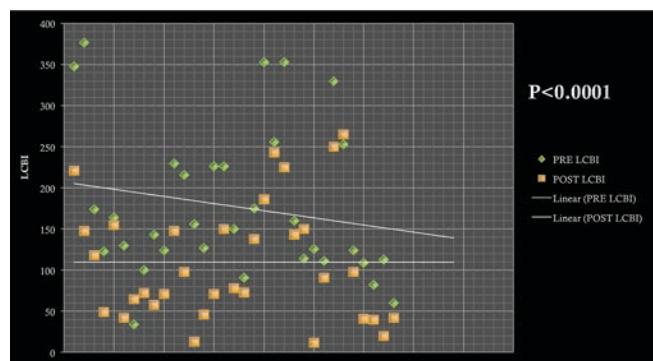


Figure 1. Decrease in coronary LCBI with percutaneous revascularization.

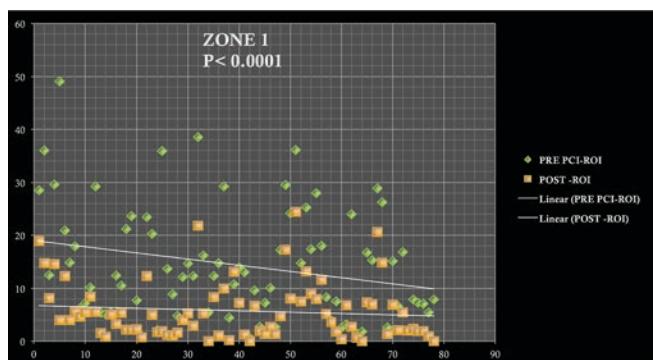


Figure 2. Decrease in lipid burden in zone 1.

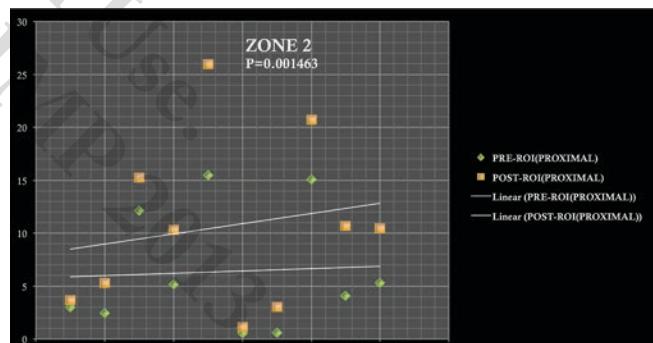


Figure 3. Increase in lipid burden in zone 2.

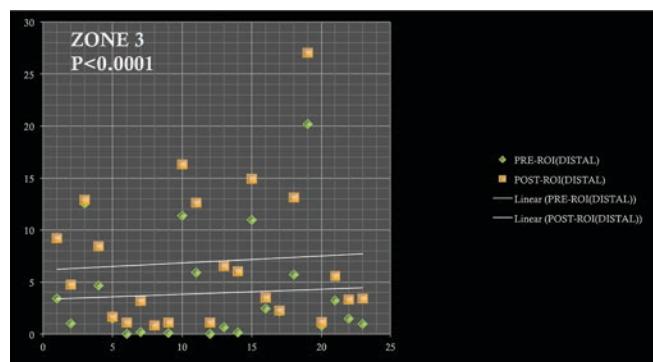


Figure 4. Increase in lipid burden in zone 3.

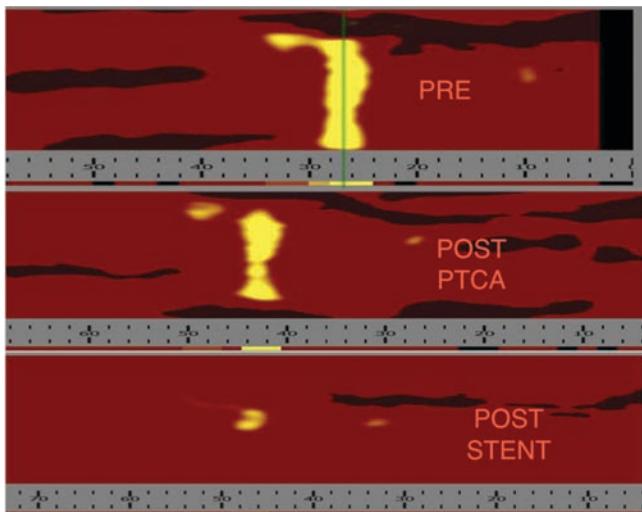


Figure 5. Decrease in lipid pool at region of interest (the zone where revascularization was performed) in zone 1.

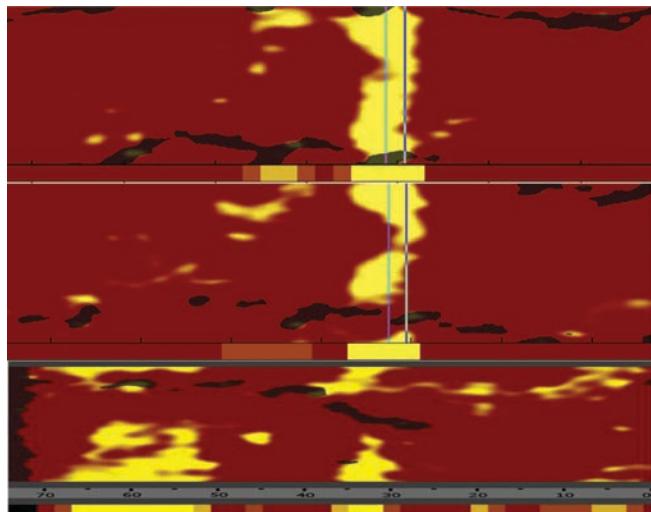


Figure 6. Decrease in lipid burden in zone 1 with an increase in zones 2 and 3.

be performed by using interventional methods and our study suggests that lipid burden at the site of revascularization always decreases. On occasion, there was an increase in the proximal and distal zones. This shifting of the LCP appears to be an acute prognostic marker for postprocedure troponin elevations, as noted in our study. These LCPs therefore may be the true high-risk plaques. Learning the pathophysiology of this active mechanical modification, therefore, is important such that hypotheses and techniques may be developed to identify these high-risk LCPs.

It is noteworthy that on long-term follow-up, the LCP shift or the degree of lipid burden change did not appear to have any prognostic significance. Larger studies are needed to further validate our study findings.

Conclusion

LCP modification may be performed and is an acute prognostic marker for postprocedure myocardial infarction when there is evidence of axial shifting of the LCP.

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