

## **In Vivo Assessment of Culprit Lesion Morphology in Acute Coronary Syndrome Using Optical Coherence Tomography**

Konstantina Bouki, MD, Thomas Apostolou, MD

*Second Department of Cardiology  
General Hospital of Nikea, Piraeus*

Autopsy studies suggest that acute myocardial infarction is provoked by sudden disruption of thin-cap fibroatheromas, known as vulnerable plaques followed by subsequent thrombosis<sup>1-3</sup>. The pathological characteristics of vulnerable plaques include a thin fibrous cap with macrophage infiltration and a large lipid pool<sup>1-3</sup>. These findings are based largely on postmortem studies because it has previously not been possible to accurately define coronary plaque morphology *in vivo*.

Optical coherence tomography (OCT) is an optical analogue of intravascular ultrasound (IVUS) that has recently been proposed as a high-resolution imaging method for plaque characterization<sup>4</sup>. Its resolution is approximately 10 to 20  $\mu\text{m}$ , which is about 10 times higher than IVUS. The histology-controlled studies have shown that OCT can evaluate the characteristics of culprit lesions such as fibrous cap thickness, fibrous cap macrophage density, lipid core and intracoronary thrombus<sup>5</sup>.

In this case report we used OCT for *in vivo* assessment of culprit lesion morphology in a patient with acute coronary syndrome.

---

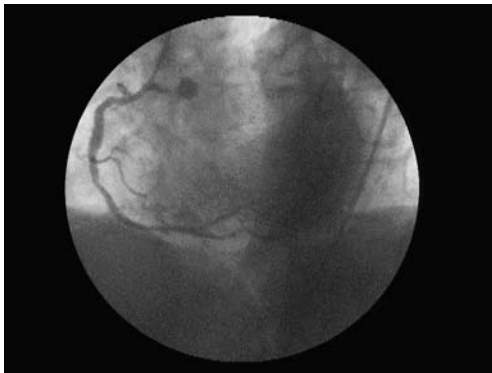
### **CASE REPORT**

---

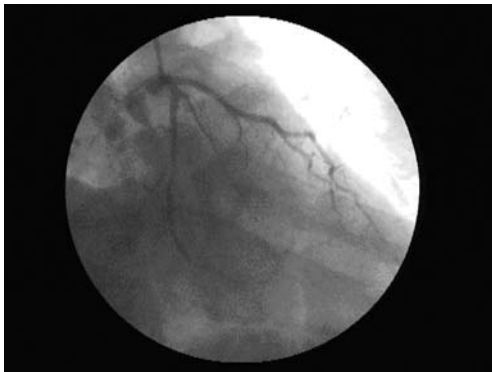
A 74-year-old man, smoker with a history of hypertension, was admitted to our Hospital because of continuous chest pain lasting one hour. The 12-lead electrocardiogram (ECG) showed sinus rhythm at 90 bpm and ST-segment elevation 0.2 mV in leads II, III and AVF, and thus the diagnosis of acute inferior wall myocardial infarction was established. The patient was hemodynamically stable with blood pressure 140/90 mm Hg. Immediately he received fibrinolysis and then he was transferred to the intensive coronary care unit where he continued therapy with enoxaparin, aspirin, clopidogrel, and beta blockers. One hour later the pain was relieved and the repeat ECG showed resolution of ST-segment elevation in the inferior leads. Blood tests showed moderate elevation of cardiac enzymes (max CK=980 iu/l, CK-MB=67 iu/l and max troponin=35.6 ng/ml). However, the third day of hospitalization the patient presented post-infarction angina and subsequently underwent coronary angiography which revealed a moderate stenosis in the mid-right coronary artery (Figure 1) and a severe stenosis in the distal circumflex artery with TIMI flow grade III (Figure 2).

Based on these findings, it was hypothesized that the culprit lesion was the lesion in the circumflex coronary artery and OCT was used for assessment of the morphology of this lesion. A 6-F guide catheter was engaged into the left coronary artery and a

*Address for correspondence:*  
E-mail address: epapak@tee.gr



**FIGURE 1.** Left anterior oblique view in 300 showing moderate stenosis in the mid-right coronary artery

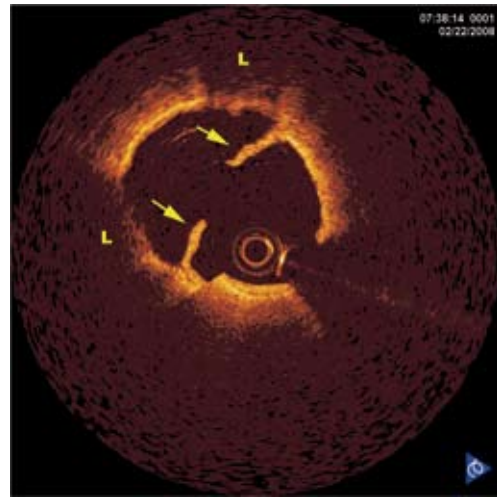


**FIGURE 2.** Right anterior oblique caudal view showing the severe stenosis in the distal circumflex

floppy guidewire crossed the lesion. Then a 0.016-inch OCT catheter (ImageWire, LightLab Imaging) was advanced to the distal end of the culprit lesion through a Twin pass catheter, over the guide wire. In order to remove the blood from the field of view, 10 ml of contrast agent was infused into the coronary artery from the guide catheter. The entire length of the culprit lesion was imaged during the contrast infusion, with automatic pullback device moving at 3 mm/sec. The images were stored for subsequent analysis.

Optical coherence tomography identified the presence of a lipid rich plaque with severe disruption (Figure 3). Multiple thrombi superimposed on the plaque disruption were also visualized (Figure 4). With use of the OCT method, the thickness of the fibrous cap was measured at the thinnest part of the flap and was found to be 35  $\mu$ m.

After OCT imaging and analysis, percutaneous coronary intervention (PCI) for the circumflex artery was performed. A bare metal stent (3.0/18 mm, Prokinetic, Biotronic) was implanted successfully in the culprit lesion, without balloon predilatation. Good apposition of the stent was confirmed by another OCT image acquisition (Figure 5).



**FIGURE 3.** Fibrous cap disruption with a flap (arrows) protruding into lumen. L=Lipid rich plaque

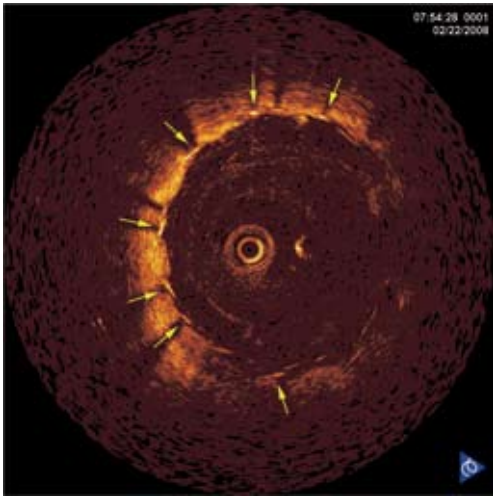


**FIGURE 4.** Severe plaque disruption with thrombi. T= thrombus, F=flap, L=Lipid rich plaque

**DISCUSSION**

In the present case, OCT allowed us to evaluate accurately the culprit lesion morphology in a patient with acute coronary syndrome. The high resolution of OCT enabled visualization of the lipid rich plaque, the fibrous cap disruption and the intracoronary thrombi.

There are several potential applications of this new imaging modality. With its high resolution and unique characteristics it provides histology-grade definition of the microstructure of coronary plaque in vivo and allows a greater understanding of the mechanisms of coronary artery disease. Moreover, it offers detailed structural information before and after PCI, with greater accuracy compared with IVUS<sup>6</sup>.



**FIGURE 5.** Successful stent apposition (arrows show the stent struts)

An inherent limitation of OCT imaging is that imaging is attenuated by blood<sup>4</sup>. It is necessary to achieve a blood-free imaging zone which can be achieved through intermittent saline or contrast flushes through the coronary guide catheter. However, even with this flushing technique, the image acquisition time is only a few seconds, which preclude imaging of long arterial segments. A second limitation<sup>4</sup> is the relatively

shallow penetration (2 mm) through the arterial wall, but because the most important morphological determinants of plaque vulnerability are superficial, the region of greatest interest is within the imaging range of current OCT systems. Next generation OCT systems are expected to eliminate many of the technical limitations.

#### REFERENCES

1. Fuster V, Fayad Z, Badimon J. Acute coronary syndromes: biology. *Lancet* 1999;353: 115-119
2. Burke A, Farb A, Malcom G, Liang Y, Smialek J, Virmani R. Coronary risk factors and plaque morphology in men with coronary disease who died suddenly. *N Engl J Med* 1997;336:1276-82
3. Virmani R, Burke A, Kolodgie F, Farb A. Vulnerable plaque: the pathology of unstable coronary lesions. *J Interv Cardiol* 2002;15:439-46
4. Jang I, Tearney G, MacNeill B, et al. In vivo characterization of coronary atherosclerotic plaque by use of optical coherence tomography. *Circulation* 2005;111:1551-55
5. Yabushita H, Bourma B, Houser S, et al. Characterization of human atherosclerosis by optical coherence tomography. *Circulation* 2002;106:1640-45
6. Kubo T, Imanishi T, Takarada S, et al. Assessment of culprit lesion morphology in acute myocardial infarction. *J Am Coll Cardiol* 2007;50:933-9