

Case Report

Visualization of Coronary Plaque Rupture Using Optical Coherence Tomography

KONSTANTINA P. BOUKI, DIONISSIOS N. CHATZOPOULOS, ELENI K. SAKKALI, STAVROULA N. PSYCHARI, THOMAS S. APOSTOLOU

2nd Cardiology Department, General Hospital of Nikea, Piraeus, Greece

Key words: Acute coronary syndromes, plaque detection.

Optical coherence tomography (OCT) is an optical analogue of intravascular ultrasound that has recently been proposed as a high-resolution imaging method for plaque characterization. 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. We describe a case where OCT was used to evaluate the culprit lesion morphology in a patient with acute myocardial infarction. The patient was treated with stent implantation. OCT was also used to confirm good stent apposition.

Autopsy studies suggest that acute myocardial infarction is provoked by sudden disruption of thin-cap fibroatheromas, known as vulnerable plaques, followed by subsequent thrombosis.¹⁻³ The pathological characteristics of vulnerable plaques include a thin fibrous cap with macrophage infiltration and a large lipid pool.¹⁻³ 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.⁴ Its resolution is approximately 10 to 20 μm , which is about 10 times higher than IVUS. 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.^{5,6}

In the case reported here we used OCT for the *in vivo* assessment of culprit

lesion morphology in a patient with acute coronary syndrome.

Case presentation

A male patient, 74 years old, a smoker with hypertension, was admitted to our hospital because of continuous chest pain lasting one hour. The 12-lead electrocardiogram (ECG) showed sinus rhythm at 90 beats/min and ST-segment elevation 0.2 mV in leads II, III and aVF, so the diagnosis of acute inferior myocardial infarction was established. The patient was hemodynamically stable with blood pressure 140/90 mmHg. He received fibrinolysis immediately and was then transferred to the intensive coronary care unit, where he continued his therapy with enoxaparin, aspirin, clopidogrel, and beta blockers. One hour later the pain was relieved and the new ECG showed resolution of the ST-segment elevation in the inferior leads. Blood tests showed moderate elevation of the cardiac enzymes (max CK 980 IU/L, CKMB 67 ng/ml, max troponin 35.6 ng/ml). On the third day of hospitalization the patient developed post-infarction an-

Manuscript received:
February 12, 2009;
Accepted:
July 30, 2009.

Address:
Konstantina P. Bouki

20 Yakinthou St.
15123 Athens, Greece
e-mail: epapak@tee.gr

gina 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).

We hypothesized that the culprit lesion was the one in the circumflex artery and we used OCT for assessment of the morphology of this lesion. A 6 F guide catheter was introduced into the left coronary artery and a floppy guide wire crossed the lesion. Then a 0.016-inch OCT catheter (ImageWire, Light-Lab 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 en-

tire length of the culprit lesion was imaged during the contrast infusion using an automatic pullback device moving at a speed of 3 mm/s. The images were stored for subsequent analysis.

OCT identified a very severe stenosis with almost no residual lumen (Figure 3), and just proximal to that the presence of a lipid-rich plaque with disruption (Figure 4). Multiple thrombi superimposed on

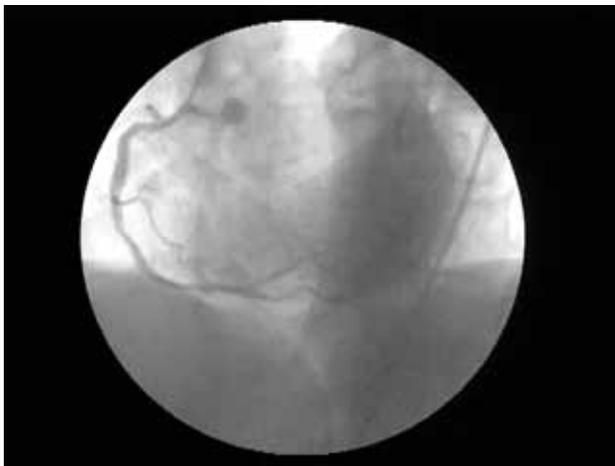


Figure 1. Left anterior oblique view at 30°, showing the moderate stenosis in the mid-right coronary artery.



Figure 2. Right anterior oblique caudal view showing the severe stenosis in the distal circumflex artery.

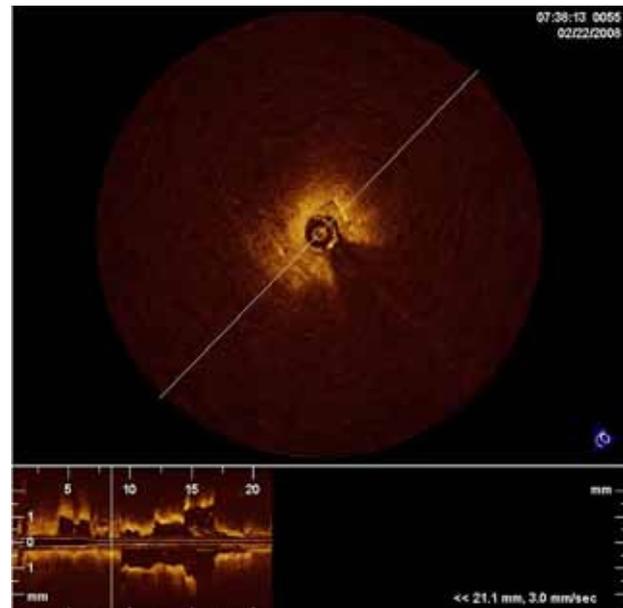


Figure 3. Very severe stenosis with almost no residual lumen.

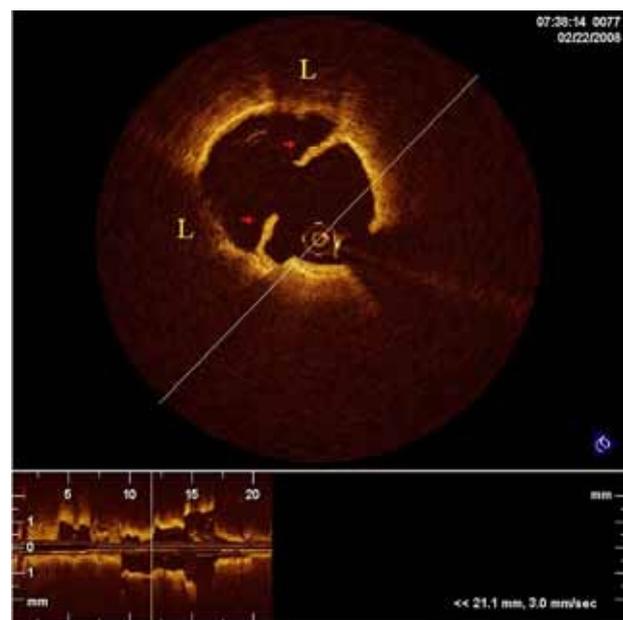


Figure 4. Fibrous cap disruption with flaps (arrows) protruding into the lumen. L – lipid-rich plaque.

the plaque disruption were also visualized (Figure 5). The thickness of the fibrous cap was measured at the thinnest part of the flap and was found to be 35 μm .

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 6).

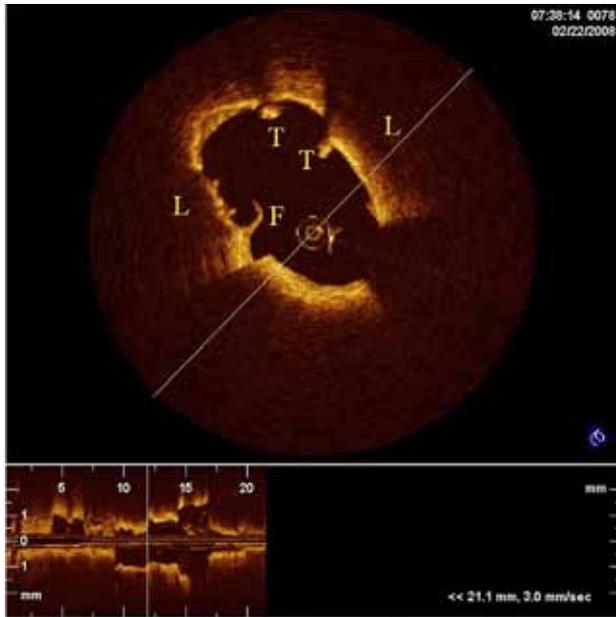


Figure 5. Severe plaque disruption with thrombi. T – thrombus; F – flap; L – lipid-rich plaque.

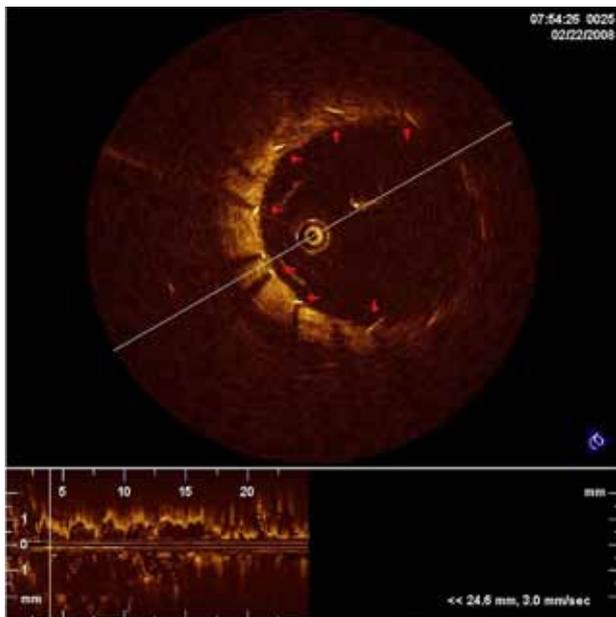


Figure 6. Successful stent apposition. Arrows show the stent struts.

Discussion

In the present case OCT allowed us to accurately evaluate the culprit lesion morphology in a patient with acute coronary syndrome. The high resolution images obtained by 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 better understanding of the mechanisms of coronary artery disease. Moreover, it offers detailed structural information before and after PCI, and greater accuracy compared with IVUS.⁷

An inherent limitation of the OCT technique is that imaging is attenuated by blood.⁴ A blood-free imaging zone is necessary and can be achieved by 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 precludes imaging of long arterial segments. A second limitation⁴ 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 aforementioned technical limitations.

References

1. Fuster V, Fayad Z, Badimon J. Acute coronary syndromes: biology. *Lancet*. 1999; 353 Suppl 2: S115-119
2. Burke AP, Farb A, Malcom GT, Liang YH, Smialek J, Virmani R. Coronary risk factors and plaque morphology in men with coronary disease who died suddenly. *N Engl J Med*. 1997; 336: 1276-1282.
3. Virmani R, Burke AP, Kolodgie FD, Farb A. Vulnerable plaque: the pathology of unstable coronary lesions. *J Interv Cardiol*. 2002; 15: 439-446.
4. Jang I-K, Tearney GJ, MacNeill B, et al. In vivo characterization of coronary atherosclerotic plaque by use of optical coherence tomography. *Circulation*. 2005; 111: 1551-1555.
5. Yabushita H, Bouma BE, Houser SL, et al. Characterization of human atherosclerosis by optical coherence tomography. *Circulation*. 2002; 106: 1640-1645.
6. Van Velzen JE, Schuijff JD, De Graaf FR, et al. Imaging of atherosclerosis: invasive and noninvasive techniques. *Hellenic J Cardiol*. 2009; 50: 245-263.
7. Kubo T, Imanishi T, Takarada S, et al. Assessment of culprit lesion morphology in acute myocardial infarction. *J Am Coll Cardiol*. 2007; 50: 933-939.