JACC: CARDIOVASCULAR IMAGING
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PUBLISHED BY ELSEVIER INC.
VOL. 8, NO. 2, 2015
ISSN 1936-878X/$36.00
http://dx.doi.org/10.1016/j.jcmg.2014.12.005

REVIEWS

STATE-OF-THE-ART PAPERS

OCT for the Identification of Vulnerable Plaque in Acute Coronary Syndrome

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ABSTRACT

After 2 decades of development and use in interventional cardiology research, optical coherence tomography (OCT) has now become a core intravascular imaging modality in clinical practice. Its unprecedented spatial resolution allows visualization of the key components of the atherosclerotic plaque that appear to confer "vulnerability" to rupture—namely the thickness of the fibrous cap, size of the necrotic core, and the presence of macrophages. The utility of OCT in the evaluation of plaque composition can provide insights into the pathophysiology of acute coronary syndrome and the healing that occurs thereafter. A brief summary of the principles of OCT technology and a comparison with other intravascular imaging modalities is presented. The review focuses on the current evidence for the use of OCT in identifying vulnerable plaques in acute coronary syndrome and its limitations. (J Am Coll Cardiol Img 2015;8:198–209)
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Optical coherence tomography (OCT) is an intravascular imaging modality that uses the reflection of near-infrared light to generate an image. OCT was first described more than 2 decades ago when it was used to image the peripapillary area of the human retina in vitro (1). Eleven years later, OCT was used to image atherosclerotic plaques in human coronary arteries (2). The image resolution achievable with OCT (axial: 10 μm, lateral: 20 to 40 μm) far surpasses that of intravascular ultrasound (IVUS) (100 to 200 μm). Histological studies have shown that certain adverse plaque phenotypes are associated with the onset of an acute coronary syndrome (ACS) (3). With its excellent spatial resolution, OCT is ideally placed to identify vulnerable plaque that could result in ACS.

This review describes the technology underlying OCT, its potential for identifying vulnerable plaques in ACS, and its limitations.

OCT TECHNOLOGY

To generate an image, a low-coherence, near-infrared (wavelength of 1.3 μm) light source is directed at the tissue (**Figure 1**). The light beam is split into 2 arms, a sample arm and a reference arm, by an interferometer. The reference arm is directed to a mirror, which reflects the light directly back to the interferometer. The light of the sample arm is absorbed, refracted, or reflected from the sample tissue, scattering the light at large angles from its surface and sub-surface. Reflected light travels back to the interferometer and

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Manuscript received October 14, 2014; revised manuscript received December 4, 2014, accepted December 9, 2014.

interacts with the reference arm light. The interaction between these 2 light waves determines the OCT image, depending on whether there is constructive or destructive interference between the waves (1). Because red blood cells strongly scatter the light waves and hence attenuate the image, OCT requires a bloodless field. The OCT catheter is connected to a rotary junction, which uses a motor to rotate the optical fiber in the catheter and couples light from this rotating fiber to light from the reference arm. The rotary junction is mounted to an automated pullback device, thus scanning the artery in a helical fashion.

There are 2 types of OCT systems: time domain (**Figure 1A**) and frequency domain (**Figure 1B**). The first-generation time domain–OCT system required sequential measurement of optical echoes from different depths by moving the reference mirror (4). This initially required the use of a balloon to occlude coronary blood flow, and the slow pullback speed of 1 to 5 mm/s led to image acquisition times of 3 to 45 s (4). Subsequently, a blood-free imaging field was obtained by controlled intracoronary infusion of isoosmolar contrast, negating the need for an occlusive balloon (5). This reduced the procedure time, although the length of the analyzed segments of artery were shorter (5). Second-generation frequency domain–OCT systems use a light source that is rapidly swept in time across wavelengths from 1.25 to 1.35 μm, allowing simultaneous recording of reflections from different depths without movement of the reference mirror (6). Depth profiles are then reconstructed by Fourier transformation. This speeds up image acquisition 10-fold, with achievable pullback speeds of up to 40 mm/s and imaging runs of up to 150 mm in length with a 3- to 5-s flush of saline or contrast, without the need for prolonged vessel occlusion (6).

OCT IMAGE FEATURES. The normal coronary artery is seen as a 3-layered structure on OCT (**Figure 2A**) (7). The internal elastic lamina appears as a signal-rich 20-μm-thick band that lies inside the dark band of the media and the further signal-rich band of the external elastic lamina (7). An atherosclerotic lesion is seen on OCT as a mass lesion within the arterial wall, with focal intimal thickening or loss of the normal vessel architecture (8). Fibrous plaque produces a relatively homogenous and highly backscattering signal (8) (**Figure 2B**). Calcified plaques appear as a signal-poor area with sharply delineated borders (**Figure 2C**). However, this only applies to larger regions of calcification; smaller areas and microcalcifications have yet to be validated against histology (8). Necrotic core (and the broader histopathological category of a lipid pool) is seen as a signal-poor region with poorly defined borders and fast OCT signal drop-off (**Figure 2D**) (8). Because light does not penetrate through these areas, OCT cannot be used to measure the depth or volume of lipid pools. Macrophage accumulations can sometimes be seen at the border of the fibrous cap and necrotic core, and can appear as punctate signal-rich spots that exceed the background noise of the image (8). Cholesterol crystals are linear regions of high intensity, often associated with a lipid pool (**Figure 3C**) (8). OCT can differentiate between white and red thrombus (**Figure 4**) due to the high proportion of red blood cells in red thrombi, which causes greater attenuation of the OCT signal and a lower half-width (the distance from peak signal intensity to its half-intensity). Kume et al. (9) showed that a cutoff of 250 μm in the half-width could accurately discriminate between white and red thrombus with a sensitivity of 90% and a specificity of 88%.

HISTOLOGICAL VALIDATION OF OCT. OCT was first validated for plaque characterization in vitro in 2002 (10). Agreement between the histopathological and OCT findings were high ($\kappa = 0.83$ to 0.84), and interobserver and intraobserver reliability were good ($\kappa = 0.88$ and $\kappa = 0.91$, respectively) (10). However, there were a number of false-negative diagnoses of lipid pools, which could be attributed to the limited penetration of OCT, leading to deep lipid pools being misinterpreted as fibrous plaques. Intimal thickness measured by OCT also correlated well with histology ($r = 0.98$, $p < 0.001$) (11). However, OCT images are prone to artifacts; 30.9% of images contained artifacts in 1 study, although this improved with operator experience (12). Seam-line artifacts cause apparent breaks in the lumen contour on the cross-sectional image (6.0% of images); decentration artifacts are caused by eccentric positioning of the imaging catheter within the artery and lead to image attenuation in remote structures (30.9%); caliber artifacts are caused by an arterial diameter greater than the penetration limit of the OCT and are a particular problem in vein grafts and in the left main stem (13) (15.0%); and flow artifacts are caused by failure to clear blood from either the vessel or imaging catheter by flushing (19.6%) (12).

OCT AND VULNERABLE PLAQUES

DEFINITION OF THIN-CAP FIBROATHEROMA. Histopathological studies have identified anatomical characteristics of "vulnerable" plaques that are

ABBREVIATIONS AND ACRONYMS

ACS = acute coronary syndrome

CKD = chronic kidney disease

DM = diabetes mellitus

IVUS = intravascular ultrasound

NIRS = near-infrared spectroscopy

OCT = optical coherence tomography

SCAD = spontaneous coronary artery dissection

STEMI = ST-segment elevation myocardial infarction

TCFA = thin-cap fibroatheroma

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