JACC: CARDIOVASCULAR IMAGING
VOL. 5, NO. 10, 2012

ISSN 1936-878X/$36.00
PUBLISHED BY ELSEVIER INC.

LETTERS TO THE EDITOR

Spontaneous Multivessel Coronary Intramural Hematoma

An Insight With OCT

Spontaneous coronary arterial intramural hematoma is a rarely diagnosed cause of acute myocardial infarction. The underlying pathophysiology is poorly understood. A historical series of intramedial dissecting hematomas, published in 1965 (1), postulated that rupture of the vasa vasorum or cystic medial necrosis, as described in the aorta by Erdheim (2), could be the precipitant. The appearance of angiographic luminal obstruction, without intravascular imaging to investigate its etiology, is likely to have contributed to an under-reporting of this phenomenon. A small number of case reports document

Figure 1. Multivessel Coronary Intramural Haematoma: An Angiographic and OCT Imaging Series

Angiographic assessment of the right coronary artery **(A,** Online Video 1) in a normally fit and well 31-year-old woman complaining of chest pain with electrocardiographic evidence of inferior ST-segment elevation. A long segment of severe "tubular" stenosis in segments 2 and 3 **(arrowhead)** was observed by right coronary angiography with Thrombolysis In Myocardial Infarction (TIMI) flow grade 3 into the distal vessel. A similar angiographic appearance was seen in the mid-left anterior descending artery **(C, arrowhead)** again with TIMI flow grade 3. Optical coherence tomography (OCT) confirmed extensive multivessel intramural hematoma without evidence of intimal dissection in the right coronary artery (**B,** image at the level of a side branch **[white arrow];** Online Video 2). In the left anterior descending artery **(D)**, the lumen is significantly compromised by an intramural filling defect that has detached the intimal and medial layers of the vessel wall **(arrowheads)**. In the absence of any ongoing ischemia, a conservative strategy was continued. Repeat angiographic **(E and G,** Online Video 3) and OCT assessments **(F and H,** Online Video 4) were performed at a 3-month interval. Consistent with Fujikura's intravascular ultrasound–defined report in 2006 (2), the conservative treatment strategy resulted in significant resorption of the hematoma and improved luminal dimensions. Mild residual angiographic stenosis is evident **(E and G, block arrowhead)**. As previously described, the medial disruption is predominantly limited to the outermost cell layers in apposition with the external elastic lamina. However, OCT suggests continuity of hematoma **(block arrowhead)** between medial and adventitial layers **(I).** This finding bears striking resemblance to the original post-mortem specimen presented by Nalbandian et al. (1), demonstrating extensive hematoma **(arrowhead)** at the medial/adventitial transition zone of a left anterior descending artery **(J).** (Reprinted, with permission, from Nalbandian et al. [1]). Association with the vasa vasorum is not clear, although potential vessel structures are evident within the area of interest **(K, block arrowheads)**. In the convalescent phase, there is evidence of persisting adventitial hematoma **(L, block arrowhead).** EEl = external elastic lamina; IEL = internal elastic lamina.

intramural hematoma by intravascular ultrasound; however, the limited resolution of intravascular ultrasound may preclude accurate delineation of the associated arterial deformation and underlying pathological processes (3,4). Optical coherence tomography provides superior resolution on the order of 10 to 15 μm and may allow better characterization of intramural abnormalities, assessment of vessel integrity, and exclusion of atheroma.

Here, we present a case of intramural coronary hematoma in a patient presenting with ST-segment elevation myocardial infarction where the use of optical coherence tomography allowed us to clearly define the underlying cause of luminal obstruction (Fig. 1, Online Videos 1, 2, 3, and 4). Our report suggests that important information can be gleaned from use of optical coherence tomography in addition to angiography, which in some cases may alter medical management.

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http:dx.doi.org/10.1016/j.jcmg.2012.03.020

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APPENDIX

For supplementary videos and their legends, please see the online version of this article.

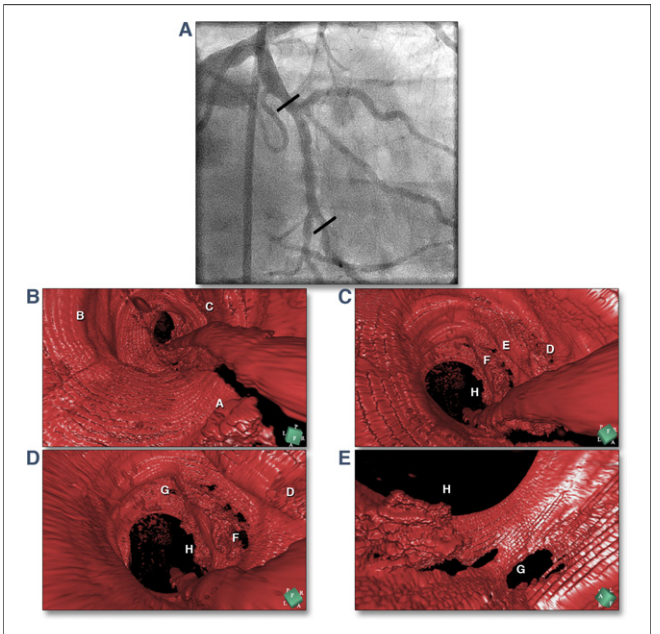

Figure 1. Coronary Angiogram and 3D OCT Renderings

A 57-year old man underwent percutaneous coronary intervention in the left anterior descending artery for stable angina. The left circumflex artery was evaluated for the presence of significant lesions by optical coherence tomography (OCT) (Online Video 1) and subsequent 3-dimensional (3D) reconstruction of OCT images was performed. **(A)** Coronary angiography of the left circumflex artery. **Black lines** indicate the studied segment. **(B to E)** Upstream fly-through view (distal-to-proximal) of 3D reconstruction indicating the sites corresponding to the panels of Figures 2 and 3.

Calcified Nodules

An Underrated Mechanism of Coronary Thrombosis?

Coronary thrombosis, typically presenting as acute coronary syndrome, is commonly considered a result of plaque rupture. However, pathologic studies have also suggested other mechanisms, such as plaque erosion and calcified nodules (1). We present a case of coronary thrombosis in a patient with superficial calcification suggestive of calcified nodules as pathophysiological substrate, as diagnosed by in vivo optical coherence tomography (OCT) imaging (Fig. 1A, Online Video 1). OCT clearly demonstrated protrusion of calcific plaques with absent intimal layer into the lumen, covered by mural thrombus (Fig. 2, Online Video 2). Intravascular ultrasound assessment also revealed thrombus over superficial calcifications (Fig. 3). Three-dimensional reconstruction of OCT images (INTAGE Realia, KGT, Tokyo, Japan) was also performed (Figs. 1D to 1E). Pathologic studies have suggested calcified nodules as a cause of thrombosis, usually associated with plaque progression rather than acute coronary syndrome. Interestingly, the patient had symptoms of stable coronary disease, possibly suggesting the absence of a highly thrombogenic milieu (2), such as necrotic core. Our observations illustrate how OCT imaging can improve diagnosis and offers the potential to improve patient care.

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http:dx.doi.org/10.1016/j.jcmg.2012.04.010

Please note: Dr. Karanasos' work was supported by a grant from the Hellenic Heart Foundation. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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