Role of Optical Coherence Tomography in Diagnosis and Treatment of Patients with Acute Coronary Syndrome

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Abstract

Acute coronary syndrome (ACS) is the main cause of death worldwide and the leading cause of disease burden in high-income countries. ACS refers to a constellation of clinical symptoms that are compatible with acute myocardial ischemia. It describes a spectrum of clinical manifestations that result from a common pathophysiological process. The most common cause of ACS are rupture of an atherosclerotic lesion containing a large necrotic core and a thin fibrous cap followed by acute luminal thrombosis. It was thought that a high-resolution imaging modality would be ideal to detect high-risk plaques before their disruption and the formation of an occlusive thrombus. Optical coherence tomography has proven to be an invaluable tool in early detection of high-risk plaques and particularly in the understanding of ACS. This review focuses on the current evidence for the role of optical coherence tomography in the diagnosis and treatment of patients with ACS.

Keywords: acute coronary syndromes; optical coherence tomography

Introduction

Acute coronary syndrome (ACS) remains a significant global health problem and the leading cause of disease burden in high-income countries [1, 2].

Coronary artery thrombosis plays a central role in the development of ACS: plaque rupture, plaque erosion, and calcified nodules are the commonest plaque morphology responsible for the development of coronary thrombosis [3–5]. Optical coherence tomography (OCT) is a high-resolution (10–15 μm) intracoronary imaging modality that has allowed visualization of the key components of the atherosclerotic plaque in the coronary artery [6–9].

Acute Coronary Syndrome

ACS refers to a constellation of clinical symptoms that are compatible with acute myocardial ischemia.

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It describes a spectrum of clinical manifestations that result from a common pathophysiological process [10]. Pathologically, coronary artery thrombosis plays a central role in the development of ACS, and the commonest cause of coronary thrombosis is plaque rupture, followed by plaque erosion and infrequently calcified nodule [11, 12]. Plaque rupture has been defined as the presence of a luminal thrombus in continuity with the large necrotic core and an overlying thin interrupted fibrous cap, measuring less than 65 μm and heavily infiltrated by inflammatory cells (macrophages and T lymphocytes). It has also been defined by Virmani et al. [11] as an area of fibrous cap disruption whereby the overlying thrombus is in continuity with the underlying necrotic core. The rupture of an atherosclerotic lesion containing a large necrotic core and a thin fibrous cap followed by acute luminal thrombi [11, 12]. Plaque erosion is characterized by a luminal thrombus in direct contact with the intimal plaque without rupture of a lipid pool; the underlining luminal surface beneath the thrombus in plaque erosion shows proteoglycan-rich and smooth muscle–rich cells [13, 14]. Jia et al. [7], using OCT to characterize the morphological features of plaque, divided plaque erosion into definite erosion (defined as the definite presence of an attached thrombus overlying an intact and visualized plaque) and probable erosion (defined as an intact fibrous cap without a thrombus with an irregular lumen, or the presence of a thrombus without superficial lipid or calcification). Calcified nodule refers to a lesion with fibrous cap disruption and thrombi associated with eruptive, dense, calcific nodules [11, 15–17]. The luminal plaque of calcified nodule shows the presence of breaks in the calcified plate, bone formation, and interspersed fibrin with an overlying thrombus [11]. After rupture or erosion of an atherosclerotic plaque, intraluminal thrombosis partially or completely obstructs the coronary artery, and the ruptured thin fibrous cap allows contact of the platelets with the highly thrombogenic necrotic core. The process is complicated by encroachment of the disrupted coronary plaque into the vessel lumen, by embolization of fragments of the thrombus into the distal coronary circulation, and by changes in vascular tone [18]. On the basis of these different features of plaque morphology in ACS, vulnerable plaques, which are identified as thrombosis-prone plaques and plaques with a high probability of undergoing rapid progression, thus becoming culprit

plaques, are proposed by the following pathological criteria: active inflammation, a thin cap with a large lipid core, endothelial denudation with superficial platelet aggregation, fissured/injured plaque, severe stenosis, superficial calcified nodules, intraplaque hemorrhage, and positive remodeling [12, 19–23]. Although most of these features are not identified in vivo by other imaging modalities, OCT allows the visualization of some of these characteristics in detail in vivo in patients, and is the best tool to assess vulnerable plaques in vivo in humans [24–26]. The clinical manifestations of ACS depend on the volume of myocardium affected and by the severity of ischemia. Thus the spectrum ranges from unstable angina with ischemia, but without detectable myocyte necrosis, to ACS with variable degrees of myocyte necrosis. The latter encompasses patients with a typical clinical syndrome accompanied by ECG changes and increased levels of cardiac markers (troponins or creatine kinase MB) through to those with extensive infarction complicated by hemodynamic compromise and other major complications [27, 28].

OCT and Plaque Assessment

The feasibility of OCT to access atherosclerotic plaque in vitro was shown in early studies. OCT achieves high resolution, can image through highly calcified tissue, and has high dynamic range [29]. A study has shown the ability of OCT to characterize human atherosclerotic plaques correlated with histological findings [30]. In that study, 357 diseased atherosclerotic arterial segments obtained at autopsy were correlated, and three types of plaque were formulated: fibrous plaques, fibrocalcific plaques, and lipid-rich plaques. Fibrous plaques were defined as homogeneous, signal-rich regions (i.e. highly backscattering plaque interiors void of OCT signal–poor regions). Fibrocalcific plaques revealed signal-poor regions with sharply delineated upper and/or lower borders. Lipid-rich plaques showed diffusely bordered, signal-poor regions (lipid pools) with overlying signal-rich bands, corresponding to fibrous caps. Sensitivity and specificity ranging from 71 to 79% and ranging from 97 to 98% respectively for fibrous plaques, ranging from 95 to 96% and 97% respectively for fibrocalcific plaques, and ranging from 90 to 94% and ranging from 90 to 92% respectively for lipid-rich plaques were obtained with

OCT compared with histology. The interobserver and intraobserver agreements for characterization of plaque type by use of OCT were good ($\kappa = 0.88$ and $\kappa = 0.91$ respectively). This study shows that OCT is highly sensitive and specific for characterizing different types of atherosclerotic plaques.

Jang et al. [8] performed the first in vivo study of detailed coronary plaque morphology in patients with various clinical coronary presentations. This study also confirmed the feasibility and safety of intravascular OCT for in vivo investigation of coronary atherosclerosis. Of 57 patients in whom OCT was successful before coronary intervention, 20 patients presented with recent acute myocardial infarction, 20 patients presented with ACS, and 17 patients presented with stable angina pectoris. The findings of this study shows a trend toward a higher frequency of lipid-rich plaque in patients with acute myocardial infarction or ACS compared with those with stable angina pectoris, but the differences were not statistically significant. A significant difference was found in the thinnest fibrous cap thicknesses among the groups. The frequency of a thin-cap fibroatheroma was also significantly different among the groups. This launched OCT as an imaging tool for the detection of the thin-cap fibroatheroma, which is considered the prototype of vulnerable plaque and a precursor of plaque rupture. Other studies confirmed the ability of OCT to assess in vivo coronary plaque morphology through enrollment of patients with ACS [28]. Tanaka et al. [31] studied the relationship in patients with ACS between the morphology of a ruptured plaque and the patient's activity at the onset of ACS using OCT. They found that the thickness of the broken fibrous cap correlated positively with activity at the onset of ACS. The study suggested that a thin-cap fibroatheroma is a lesion predisposed to rupture both at rest and during the patient's day-to day activity, and some plaque rupture may occur in thick fibrous caps depending on exertion levels. Another study found that the incidence of plaque rupture, thin-cap fibroatheroma, and red thrombus was significantly higher in ST-segment elevation myocardial infarction (STEMI) compared with non–ST-segment elevation ACS (NSTEACS) (70 vs. 47%, P = 0.033, 78 vs. 49%, P = 0.008, and 78 vs. 27%, P < 0.001 respectively). Further, OCT showed that a ruptured plaque of which the aperture was open wide against the direction of coronary flow was more frequent in STEMI than in NSTEACS (46 vs. 17%, P = 0.036) [32]. Another study selected 55 myocardial patients and documented culprit plaque rupture by OCT (*n* = 30 with STEMI; *n* = 25 with non-ST-segment elevation myocardial infarction [NSTEMI]). The study authors reported that the site of plaque rupture was the minimal lumen in only 34.5% of patients, whereas 69% of the ruptures occurred at the plaque shoulder. In 96% of cases, the ruptured cap thickness was 90 μm or less. Patients with NSTEMI presented with a greater minimal luminal area (P < 0.001), less lipid content (P = 0.01), and shorter rupture length (P < 0.001) and length of the missing fibrous cap (P < 0.05) compared with patients with STEMI [33]. OCT has proven to be the ideal intravascular imaging modality and enables us to accurately evaluate plaque morphologies; specifically, thin fibrous cap, macrophages, and intracoronary thrombus. More importantly, OCT potentially help us understand the underlying mechanism behind the abrupt transition from stable atherosclerotic disease to ACS by investigating the natural evolution of coronary atherosclerotic plaques, which may ultimately allow us to discover new diagnostic algorithms and therapeutic targets [5, 34–40].

OCT Assessment of Percutaneous Coronary Intervention

OCT has undoubtedly played a critical role in our understanding of the underlying mechanisms and ultimately the treatment of ACS. With use of OCT, pathological findings of culprit plaque in ACS were validated. The agreement between pathological and OCT findings was high, and interobserver reliability and intraobserver reliability were good [11, 28, 30]. Our group recently provided unique insights in patients with ACS using OCT by evaluating the morphological characteristics of OCT-determined plaque erosion and OCT-determined calcified nodules in culprit lesions [7]. In that study, the incidence of culprit plaque rupture, plaque erosion, and calcified nodule was 44, 31, and 8% respectively; Plaque erosions were more commonly observed in younger patients and had less severe stenosis. Higuma et al. [41] reported a comprehensive in vivo evaluation of culprit plaque in patients with

acute STEMI using OCT. They found that the incidence of plaque rupture, plaque erosion, and calcified nodule in STEMI patients was 64.3, 26.8, and 8.0% respectively; plaque erosion was characterized by fewer features of plaque vulnerability and was associated with less microvascular damage after percutaneous coronary intervention (PCI). Calcified nodule was characterized by superficial calcium sheets and negative remodeling. Current therapeutic strategies for ACS patients are prioritized to either catheter-based coronary revascularization or conservative management (antiplatelet agents with or without anticoagulation agents to resolve the thrombus), with all patients receiving medical therapy [14, 42]. On the basis of the pathological features associated with plaque erosion (intact fibrous cap, larger residual lumen, and platelet-rich thrombus) [14, 43], Jia et al. [44] prospectively investigated the safety of antithrombolytic therapy without stent implantation in patients with ACS caused by plaque erosion. This study reflects a potential paradigm in the treatment of ACS patients: thrombus volume reduced in 55 patients treated with antithrombolytic therapy and followed up by OCT at 1 month. The clinical management of plaque erosion may differ from that of plaque rupture. Plaque erosion has an entity distinction: most eroded lesions are deeply seated in the necrotic core, and when present, the core does not communicate with the lumen because of a thick fibrous cap [7, 14, 45], lesions shows minor lumen narrowing, and the luminal thrombosis has been attributed to apoptosis [46–48]. Therefore, these unique features of plaque erosion indicate that after thrombus, removal treatment with delay or avoidance of stent deployment and effective antithrombotic therapy may potentially restore coronary artery patency and allow healing of the endothelial layer [44, 49].

Restoration of coronary blood flow by PCI has dramatically improved the prognosis of patients with ACS during the past few decades [50]. OCT-guided PCI can provide detailed information during PCI (no-reflow phenomenon, stent malapposition, tissue protrusion, coronary dissection) and at follow-up examinations [51–56]. Tanaka et al. [57] showed that OCT can predict no reflow after PCI in patients with ACS. The lipid contents of a culprit plaque may play a key role in damaging the microcirculation after PCI.

Conclusion

OCT continues to provide great insights into the pathophysiology of ACS and visualization of unstable lesion morphologies in vivo that have been demonstrated in histology examinations. Thus OCT examination helps us to understand the appropriate patient-specific therapeutic approach and clinical outcomes of patients with ACS. OCT is a novel tool, with the capacity to examine structures of the artery wall before or after PCI, and is superior to other imaging modalities, such as angiography and intravascular ultrasonography.

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Conflict of Interest

The authors declare that they have no conflicts of interest.

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