Chapter 2

Near-Infrared Spectroscopy (NIRS): A Novel Tool for Intravascular Coronary Imaging

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Additional information is available at the end of the chapter

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Abstract

Acute coronary syndrome (ACS) arising from plaque rupture is the leading cause of mortality worldwide. Near-infrared spectroscopy (NIRS) combined with intravascular ultrasound (NIRS-IVUS) is a novel catheter-based intravascular imaging modality that provides a chemogram of the coronary artery wall, which enables the detection of lipid core and specific quantification of lipid accumulation measured as the lipid-core burden index (LCBI) in patients undergoing coronary angiography. Recent studies have shown that NIRS-IVUS can identify vulnerable plaques and vulnerable patients associated with increased risk of adverse cardiovascular events, whereas an increased coronary plaque LCBI may predict a higher risk of future cardiovascular events and periprocedural events. NIRS is a promising tool for the detection of vulnerable plaques in CAD patients, PCI-guidance procedures, and assessment of lipid-lowering therapies. Previous trials have evaluated the impact of statin therapy on coronary NIRS defined lipid cores, whereas NIRS could further be used as a surrogate end point of future ACS in phase II clinical trials evaluating novel anti-atheromatous drug therapies. Multiple ongoing studies address the different potential clinical applications of NIRS-IVUS imaging as a valuable tool for coronary plaque characterization and predictor of future coronary events in CAD patients.

Keywords: near-infrared spectroscopy (NIRS), intravascular ultrasound (IVUS), thin-cap fibroatheroma (TCFA), acute coronary syndrome (ACS), vulnerable plaque

1. Introduction

Coronary artery disease (CAD) is the leading cause of global mortality and the rupture of an unstable atherosclerotic plaque precedes the majority of acute coronary syndromes (ACS) [1, 2]. Autopsy studies have shown that the putative substrate for most ACS and many cases of sudden

cardiac death (SCD) is the rupture of a thin-cap fibroatheroma (TCFA), the so-called "*vulnerable plaque,*" which is defined by a large lipid-rich necrotic core (NC) infiltrated with abundant macrophages and separated from the bloodstream by a thin fibrous cap [3, 4]. The ability to accurately detect index lesions using intravascular imaging is a potential attractive strategy, although it still remains a challenge in daily practice. Conventional coronary angiography (CCA) has been and continues to be an invaluable tool for epicardial coronary stenoses assessment and treatment [5]. Since the coronary angiogram provides a limited "*luminogram*" view of the coronary arteries, it cannot assess the properties of the arterial wall and thus tends to underestimate the true magnitude of plaque burden, especially in early stages of the disease in which positive vascular remodeling leads to a normal lumen caliber appearance on angiography despite substantial vascular wall plaque [6, 7]. Moreover, angiography provides no information in regard to plaque composition and biological activity, whereas intravascular imaging can potentially circumvent those limitations [8]. Several intravascular-imaging modalities, such as angioscopy, intravascular ultrasound (IVUS), virtual histology (VH), optical coherence tomography (OCT), and near-infrared spectroscopy (NIRS), have been developed throughout the quest of vulnerable plaque to characterize plaque composition and progression, to optimize patient risk stratification and for guiding therapy [9].

Near-infrared spectroscopy (NIRS) is a novel intravascular-imaging modality that provides chemical assessment related to the presence of cholesterol esters in lipid cores and

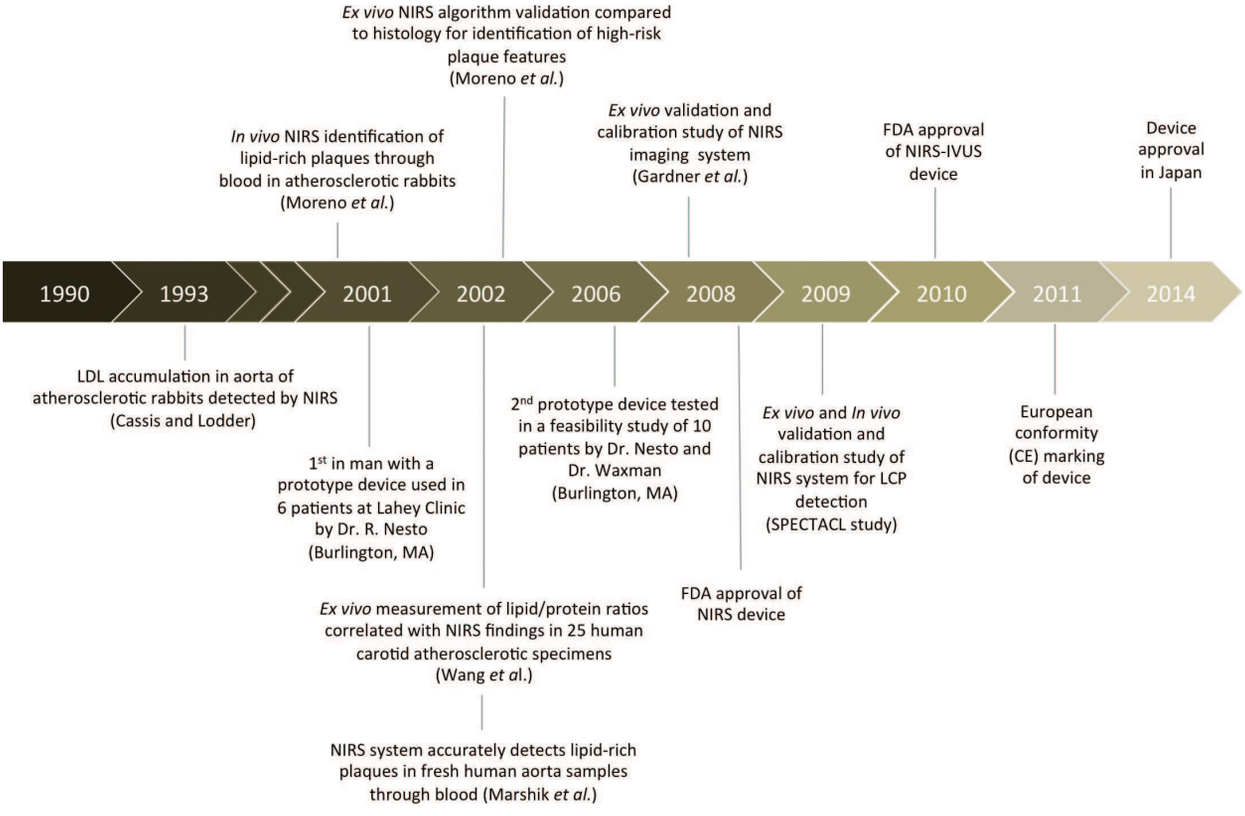

Figure 1. Timeline regarding important steps toward NIRS-IVUS imaging system development and use in clinical applications.

brovascular events (MACCE) [93]. The results showed that the presence of large LCP in a non-stented segment, defined by NIRS maxLCBI$_{4mm}$ ≥400 at baseline, was associated with a significantly increased risk of future MACCE during follow-up (HR 10.2, 95% CI: 3.4–30.6; $P < 0.001$). This study, although single center, underpowered, and with limited follow-up, was consistent with the findings of ATHEROREMO-NIRS study, whereas NIRS detection of lipid burden was associated with patient-level risk of future MACCE [93].

The detection of fibroatheroma could help to identify culprit lesions in ACS patients, predict lesions subject to periprocedural complications, could allow optimal stent selection, and reduce the rate of stent restenosis. Whether the detection of fibroatheroma using NIRS-IVUS will prevent future events is currently being studied in several trials, including the Lipid-Rich Plaque study (LCP; Clinical Trials.org Identifier: NCT02033694), PROSPECT II ABSORB trial (A Multicentre Prospective Natural History Study Using Multimodality Imaging in Patients With acute Coronary Syndromes; Clinical Trials.org Identifier: NCT02171065), and ORACLE-NIRS trial (Lipid cORe Plaque Association With CLinical Events: a Near-InfraRed Spectroscopy Study; Clinical Trials.org Identifier: NCT02265146).

2.6.4. Optimizing percutaneous coronary intervention procedures

Visual estimation of a coronary stenosis on a two-dimensional (2D) angiography or quantitative coronary angiography (QCA) of lesion lengths is often misleading from image foreshortening and underestimation of plaque burden. IVUS offers accurate length measurement during automated pullback, proximal and distal reference diameter of a vessel, and enables to evaluate the presence and extent of calcifications [26]. The ADAPT-DES (Assessment of Dual Antiplatelet Therapy With Drug-Eluting Stents) study, a prospective, multicenter, nonrandomized "all-comers" registry of 8583 consecutive patients, showed that IVUS-guidance PCI, performed in 39% of patients, was associated with reduced 1-year rates of MACE (3.1% vs. 4.7%; adjusted HR, 0.70; 95% CI: 0.55–0.88; $P = 002$), as compared to angiography guidance alone [94]. The benefits of IVUS were observed in patients with ACS and complex lesions, although significant reductions in MACE were present in all patient subgroups, including stable angina and single-vessel disease. Similar results were observed in subsequent meta-analysis [95, 96].

The use of combined NIRS-IVUS imaging may further optimize stent implantation by accurate identification of lipid margins, and thus cover all the segments with high lipid burden. Dixon et al. [97] analyzed 75 lesions with NIRS imaging and demonstrated that lipid-core plaque extended beyond the angiographic margins of the initial target lesion in 16% of cases. Hanson et al. [98] showed that atheroma, defined as plaque burden >40% or LCP, extended beyond angiographic margins in 52 of the 58 lesions analyzed with NIRS-IVUS (90% of lesions), with a mean lesion length that was significantly longer when assessed by NIRS-IVUS as compared with angiography alone (19.8 ± 7.0 vs. 13.4 ± 5.9 mm; $P < 0.0001$). Those results suggests that NIRS-IVUS guidance during PCI procedures, as a "red-to-red" stenting strategy, could optimize complete LCP coverage by a stent with the proper length according to the landing zones and thus reduce the risk of edge dissections, stent failure, and subsequent adverse clinical outcomes [26, 39, 99–101]. Although it seems rationale to implant the edges of a stent in a normal artery segment, the marginal increased risk of stent thrombosis and restenosis with

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longer stents will require future studies to determine if routine use of NIRS-IVUS for proper stent sizing will improve patient outcomes [102].

Detection of lipid core in a lesion has also been used as one of the factors to consider in the decision to implant a bare metal stent (BMS) or a drug-eluting stent (DES). Several studies have demonstrated a greater frequency of stent thrombosis after DES implantation when struts were penetrating into a lipid-rich necrotic core plaque rather than in a non-yellow (fibrous) plaque [103, 104]. The absence of struts coverage by the formation of a neointima layer during vessel's healing process was seen with both DES and BMS implantation in lipid-rich plaques, which is likely the underlying mechanism of stent thrombosis seen in those patients [105, 106]. Neoatherosclerosis is an important contributor to late-stent thrombosis with newer generation DES, as well as late in-stent restenosis. Histologically, neoatherosclerosis is characterized by the accumulation of lipid-laden macrophages within the neointima with or without necrotic core formation and/or calcification and can occur months to years following stent placement [107]. Originally described in postmortem studies, neoatherosclerosis has more recently been detected by intracoronary imaging. Ali et al. [108] used NIRS and OCT to assess the development of neoatherosclerosis in 65 consecutive patients with symptomatic in-stent restenosis. The prevalence of LCP within neointimal hyperplasia segments was 89% using NIRS versus 62% using OCT. Neoatherosclerosis was associated with significantly reduced minimal cap thickness with plaque rupture occurring exclusively in those patients. Moreover, DES had a higher prevalence and earlier occurrence of neoatherosclerosis, thinner cap, and more lipid burden and density. However, LCP identified by NIRS alone was not associated with periprocedural MI during treatment for in-stent restenosis, which reflects the limited ability of NIRS to differentiate lipid located within the neointimal tissue from a lipid core located underneath stent struts. Nevertheless, postmortem imaging and subsequent histology analysis showed that NIRS could correctly characterize lipid despite the presence of metal struts. Similar findings were reported in a study published by Madder et al. [109], whereas NIRS was not reliable for neoatherosclerosis detection when used as the sole imaging modality for LCP detection. The NIRS lipid signal could not distinguish neoatherosclerosis from fibroatheroma underlying the stent. No doubt that NIRS can detect coronary LCP, but it seems unlikely suitable as a standalone technique for accurate neoatherosclerosis detection and that the adjunction of IVUS or OCT will be required to determine the position of NIRS lipid signal relative to the underlying stent struts [110].

It was proposed that the growth of neointima tissue on the top of a vulnerable plaque might increase the thickness of the fibrous cap [103, 110, 111]. Brugaletta et al. [112] reported the ability of bioresorbable vascular scaffold (BVS) implantation to promote the growth of neointimal tissue, which acts as a barrier to isolate vulnerable plaques. An ongoing trial, the PROSPECT II ABSORB sub-study trial (Clinical Trials.org Identifier: NCT021711065), will randomize patients with plaques at high risk of causing future coronary events (plaque burden ≥70%) to receive an Absorb™ BVS (Abbott Vascular, IL, USA) with optimal medical therapy (OMT) versus OMT alone. This sub-study aims to evaluate the changes in the plaque at 2 years follow-up. Clinically, large LCPs have been shown to be associated with MACE, especially periprocedural myocardial infarction [21]. Whether lipid burden influences long-term outcomes following stent implantation remains elusive.

2.6.5. Prevention of periprocedural complications

Approximately 3–15% of percutaneous coronary interventions are complicated by periprocedural myocardial infarction (PPMI) and no-reflow, in part by distal embolization of intraluminal thrombus and/or lipid-core plaque content, which is associated with adverse long-term outcomes [113, 114]. It was reported that periprocedural MIs are associated with increased atherosclerotic burden and large LCPs [115–118]. Indeed, embolization of the lipid core after stent implantation in a plaque with high lipid content has been identified as an important cause of periprocedural no-reflow and MI with and without the presence of intracoronary thrombus [118–120]. A pilot study performed in nine patients using an embolic protection device showed that embolized material consisted in fibrin and platelet aggregates, which reflects the highly thrombogenic content of necrotic core of large atheroma plaques and LCP [98, 120, 121]. In a sub-study of the COLOR (Chemometric Observation of Lipid-Core Plaques of Interest in Native Coronary Arteries) registry, a prospective multicenter observational study aiming to determine a relationship between NIRS-defined high LCBI and periprocedural MI, Goldstein et al. [20] analyzed the cardiac biomarkers of 62 stable patients undergoing PCI. The main findings were that periprocedural MI, defined in the study as a postprocedural elevation above three times the upper limit of normal (ULN) for either creatine kinase-MB (CK-MB) or cTnI measured 4–24 h after PCI, occurred in nine patients (14.5%) and was more common among patients with a $\text{maxLCBI}_{4\text{mm}} \geq 500$ (7 of 14 patients, 50%) versus patients with a $\text{maxLCBI}_{4\text{mm}} < 500$ (2 of 48 patients, 4.2%). The authors concluded that a high LCP, defined as a $\text{maxLCBI}_{4\text{mm}} \geq 500$, was associated with periprocedural events. These results are concordant with the registry study conducted by Raghunathan et al. [21], in which the analysis of 30 patients who underwent pre-procedure NIRS imaging showed a postprocedural increase of CK-MB more than three times the UNL in 27% of patients with a ≥1 yellow blocks ($n = 11$) as opposed to none in the 19 patients without a yellow block within the stented lesion.

Distal embolization, as an important mechanism of periprocedural MI, was further supported by several studies that have demonstrated a significant decrease in the size of LCP after stenting [122–124]. Stone et al. showed in the CANARY trial that LCP measured as LCBI by NIRS in the stented vessels reduces with PCI treatment, with a significant reduction of median LCBI from 143.2 before PCI to 17.9 after PCI ($P < 0.001$) [125]. Moreover, the authors showed that the occurrence of periprocedural MI was associated with higher LCBI, results that are concordant with previous findings [20, 21].

In order to prevent periprocedural MI during PCI, several strategies were proposed during stenting procedures, including aspiration thrombectomy, embolization distal-protection devices, vasodilators, intensive anticoagulation, and antiplatelet therapies. The CANARY (Coronary Assessment by NIR of Atherosclerotic Rupture-Prone Yellow) trial randomized 85 stable angina patients undergoing stent implantation of a single native coronary lesion and pre-procedure NIRS-defined $\text{maxLCBI}_{4\text{mm}} \geq 600$ to PCI with or without distal-protection filter [125]. Among the 31 randomized cases with a $\text{maxLCBI}_{4\text{mm}} \geq 600$, there was no difference in the rates of periprocedural MI with or without the use of distal-protection filter (35.7 vs. 23.5%, respectively; relative risk 1.52; 95% CI: 0.50–4.60, $P = 0.69$). It should be noted that the CANARY trial was ended prematurely due to difficulties in identifying patients suitable

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for randomization to embolic-protection devices and lack of signs of benefits and thus was not adequately powered to detect a difference in MI or other major procedural complications between the two patient groups. An ongoing study, the CONCERTO (Randomized-Controlled Trial of a Combined versus Conventional Percutaneous Intervention for Near-Infrared Spectroscopy Defined High-Risk Native Coronary Artery Lesions; Clinical Trials.org Identifier: NCT02601664) trial, aims to evaluate different strategies for periprocedural MI prevention. Patients undergoing PCI with high-risk native coronary lesion, defined as ≥2 contiguous yellow blocks on the block chemogram, are randomized to combined preventive measures versus conventional PCI. The combined preventive measures consist of pre-PCI administration of an intracoronary vasodilator and a glycoprotein IIb/IIIa inhibitor, in addition to the use of an embolic-protection device if technically feasible and a complete coverage of the LCP if technically feasible.

Thrombectomy is often used to aspirate thrombus and restore blood flow in the culprit vessel during primary PCI in STEMI patients. The clinical benefits of routine thrombus aspiration remain a matter of debate, since the TAPAS (Thrombus Aspiration during Percutaneous Coronary Intervention in Acute Myocardial Infarction) study demonstrated a reduction of mortality while larger studies such as TASTE (Thrombus Aspiration in ST-Elevation Myocardial Infarction in Scandinavia) and TOTAL (Trial of Routine Aspiration Thrombectomy with PCI versus PCI Alone in Patients with STEMI) did not show a reduction of cardiovascular mortality, with an increased rate of stroke at a 30-day follow-up in the TOTAL trial [126–128]. Erlinge et al. [129] performed NIRS-IVUS imaging in 18 ACS patients to examine if aspiration thrombectomy reduced the lipid content of ACS culprit plaques. The culprit lipid content was quantified by NIRS-IVUS before and after thrombectomy as the lipid-core burden index (LCBI), and aspirates were examined by histological staining for lipids, calcium, and macrophages. Culprit lesions were found to have high lipid content prior to thrombectomy, which resulted in a 28% reduction in culprit lesion lipid content (pre-aspiration LCBI 466 ± 141 vs. post-aspiration 335 ± 117, $P = 0.0001$).

As aforementioned, the use of intracoronary NIRS-IVUS imaging for accurate identification of LCP lesions prone to embolize, as well as different treatment strategies, for periprocedural MI prevention are attractive approaches, however their clinical benefits on myocardial salvage and prevention of embolization remains to be demonstrated in future studies.

2.6.6. Monitoring effects of lipid-lowering therapies

It is well known that statin therapy reduces rates of cardiovascular events in secondary prevention. The pharmacological effects of specific lipid-reducing agents that reduce free and esterified cholesterol could be evaluated with NIRS, as it informs on the lipid content of coronary artery plaques over time. The demonstration of markedly reduced LCBI values in a patient after 1 year of high-dose rosuvastatin therapy was the first indication that NIRS-IVUS could be used to evaluate the effect of systemic anti-atherosclerotic medical therapy [130]. In the YELLOW (Reduction in Yellow Plaque by Aggressive Lipid-Lowering Therapy) trial, Kini et al. [22] prospectively randomized 87 patients with multivessel coronary artery disease undergoing PCI with one culprit and one nonculprit hemodynamically significant

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