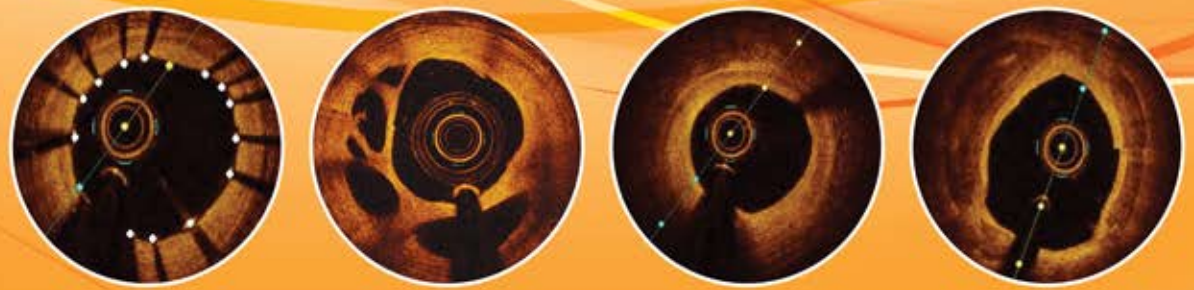
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Coronary OCT

A Case-based Approach

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- Image → Interpret → Implement
- Best way to become an expert in OCT



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Forewords
Takashi Akasaka
Rajesh M Dave

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CHAPTER 7

ST-elevation Myocardial Infarction: Acute Coronary Syndrome

ABSTRACT

Acute coronary syndromes (ACS) are associated with high morbidity and mortality, develop from acute thrombus formation secondary to plaque rupture (55–60% of cases) or plaque erosion (30–35%) or eruptive calcium nodule (2–7%). About 17–20% cases of acute coronary syndromes are due to other etiologies such as subocclusive stenosis, recanalized thrombus, spontaneous coronary dissection, coronary embolism, and coronary spasm. Optical coherence tomography (OCT) is useful in assessing morphological features of plaques to differentiate various subsets of ACS thereby guides in the appropriate management decisions.

INTRODUCTION

Optical coherence tomography (OCT) is very useful for acute coronary syndrome (ACS) patients with acute onset of angina and SOB, mainly to assess plaque morphology and etiology of ACS nature. ACS is mainly due to plaque rupture, plaque erosion, eruptive calcific nodule with thrombus, spontaneous coronary artery dissection (SCAD), recanalized thrombus, etc. it is useful to detect the site of lesion, assessing the mean luminal areas, quantification of thrombus, and helpful in differentiating white thrombus and red thrombus. Management of ACS depends upon underlying etiology **(Box 1, Flowcharts 1 and 2, and Figs. 1A and B and Fig. 2A)**.[1]

PATHOLOGY OF CULPRIT LESIONS IN ACUTE CORONARY SYNDROME (FIGS. 1 AND 2A)

Plaque rupture- of ST-elevation myocardial infarction (STEMI) is seen as a presence of fibrous-cap discontinuity with underlying cavity with large lipid-rich plaque with thin-cap fibroatheroma (TCFA). These are associated with a greater extent of cap disruption and a smaller minimal lumen area compared with non-STEMI patients with ruptured plaques. Patients of STEMI is more often associated with "proximal-type" of rupture of the fibrous cap, contrary to "distal-type" of ruptures of non-STEMI patients.

Plaque erosion: Plaque erosion (PE) is visible as evidence of thrombi with an irregular luminal surface. The characteristic of OCT features of erosions is a thick intima without cap rupture and a small or absent lipid core. If the lipid core is present, it does not communicate with luminal thrombi.[2]

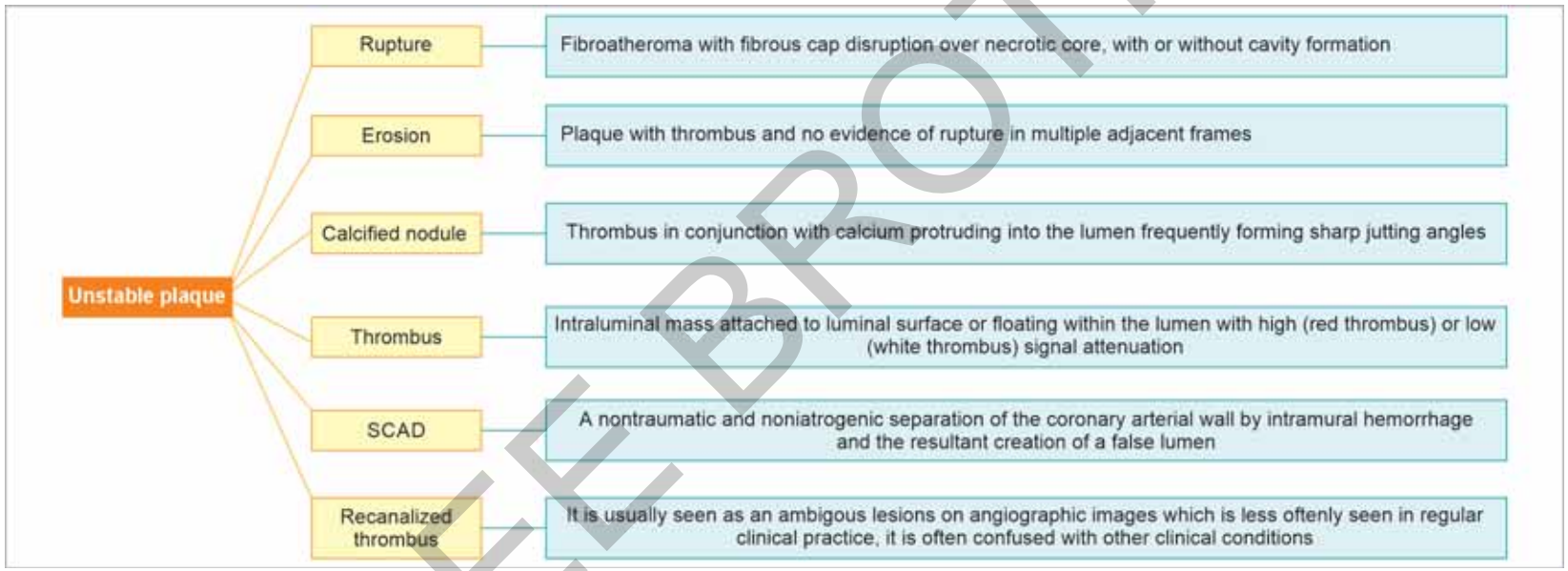
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BOX 1: OCT for ACS.

- Detects and quantifies thrombus at the culprit site.
- Defines plaque morphology in ACS and guides the appropriate therapy.
- Very useful in ambiguous or borderline lesions to diagnose rare causes such as SCAD, recanalized thrombus, MINOCA and embolism, etc.

(ACS: acute coronary syndrome; MINOCA: myocardial infarction with nonobstructive coronary arteries; OCT: optical coherence tomography; SCAD: spontaneous coronary artery dissection)

Flowchart 1: Unstable plaque morphology by optical coherence tomography (OCT).



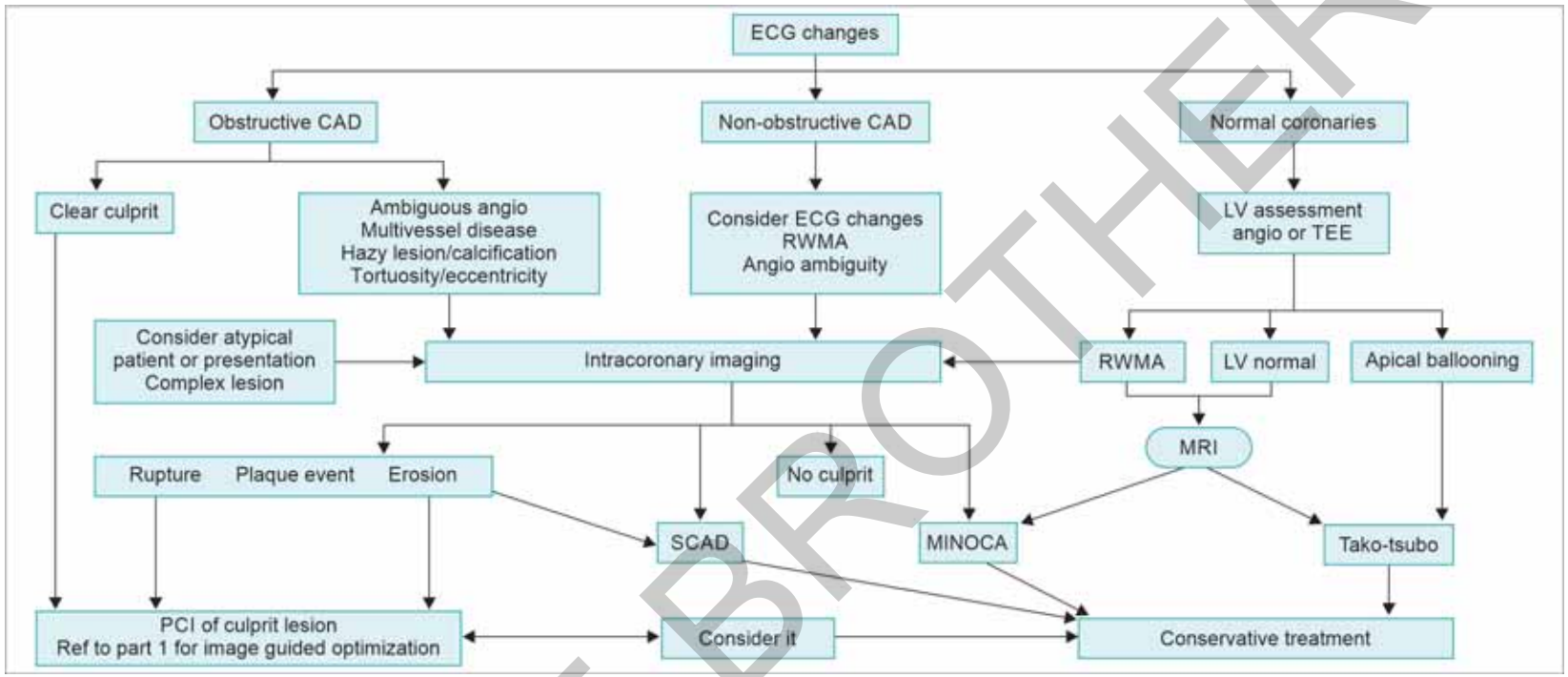
(SCAD: spontaneous coronary artery dissection)

Calcified nodule: OCT shows it as a protrusion of a signal—poor or heterogeneous region with a sharply delineated border. These are more often present with non-STEMI and seen in more often patients with advanced age, chronic kidney disease (CKD), longstanding uncontrolled diabetes, and postcoronary artery bypass grafting (CABG) status.[3,4]

Plaque rupture releases prothrombogenic substrates leading to recurrent local thrombosis and distal embolization, decreases coronary microcirculation, and these are usually associated with worse outcomes after stenting and higher frequency of distal embolization. Patients of PE are associated with less severe obstruction, a less superficial necrotic core, and a smaller plaque burden. However, thrombus in PE is generally due to apoptosis or denudation of superficial endothelial cells and is rich in platelets **(Fig. 2B)**.

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Flowchart 2: A treatment algorithm to guide the use of intravascular OCT imaging in patients presenting with acute coronary syndromes.



(CAD: coronary artery disease; LV: left ventricular; MINOCA: myocardial infarction with nonobstructive coronary arteries; OCT: optical coherence tomography; PCI: percutaneous coronary intervention; RWMA: regional wall motion abnormalities; SCAD: spontaneous coronary artery dissection; TEE: transesophageal echocardiography)

THROMBUS EVALUATION

OCT assessment of red thrombus is more accurate, and it can be differentiated from platelet-rich white thrombus. It is defined as intraluminal tissue >0.25 mm in diameter and either high backscattering with high attenuation (red-cell-rich thrombus) less back scattering with homogeneous low attenuation (platelet-rich thrombus) or a mixture of both. Thrombus can be quantified by sum up of the total number of quadrants of lumen containing thrombus. The maximum thrombus area is calculated by tracing the outline of the thrombus if the outline is visible or subtracting the minimum area within the thrombus from the adjacent reference lumen area not having thrombus if the outline of thrombus is not visible.

Thrombus burden can be measured quantitatively by planimetry and semi-quantitatively by the OCT thrombus score **(Figs. 3A to F)**. OCT can differentiate between red and white thrombi with high sensitivity (90%) and specificity (80%) (Kume et al.), and mixed thrombus shows intermediate features of both white and red thrombus. Sometimes, it is difficult to differentiate between in-stent plaque prolapse, thrombus protrusion, and acute stent thrombosis by OCT.[5]

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Thin-cap fibroatheroma (TCFA) ***(Figs. 4A and B)****:* A fibrous cap is identified as a signal-rich homogenous region overlying a lipid core, which is characterized by a signal—poor region. TCFA is defined as a plaque with a fibrous cap measuring <65 μm.

DETECTION OF VULNERABLE PLAQUE IN VIVO BY OPTICAL COHERENCE TOMOGRAPHY

OCT is the best tool to identify vulnerable plaque **(Fig. 5)** by detecting 7–8 out of 10 criteria except for glistening yellow color, endothelial dysfunction, and intraplaque hemorrhage (**Tables 1 and 2 and Box 2**).

Major Criteria

- Active inflammation (monocyte/macrophage and sometimes T-cell infiltration)
- Thin cap with large lipid core
- Endothelial denudation with superficial platelet aggregation
- Fissured plaque
- Stenosis >90%.

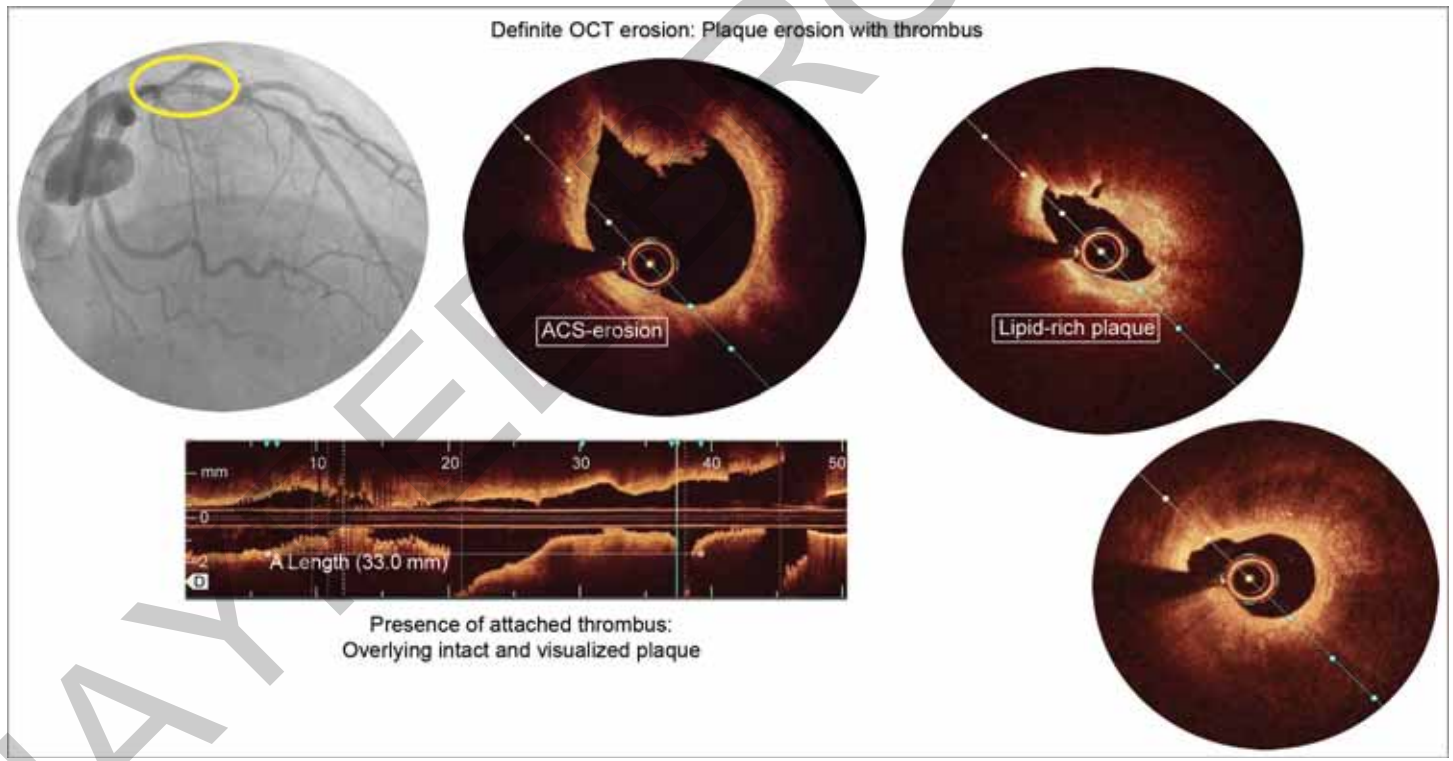


Fig. 1A

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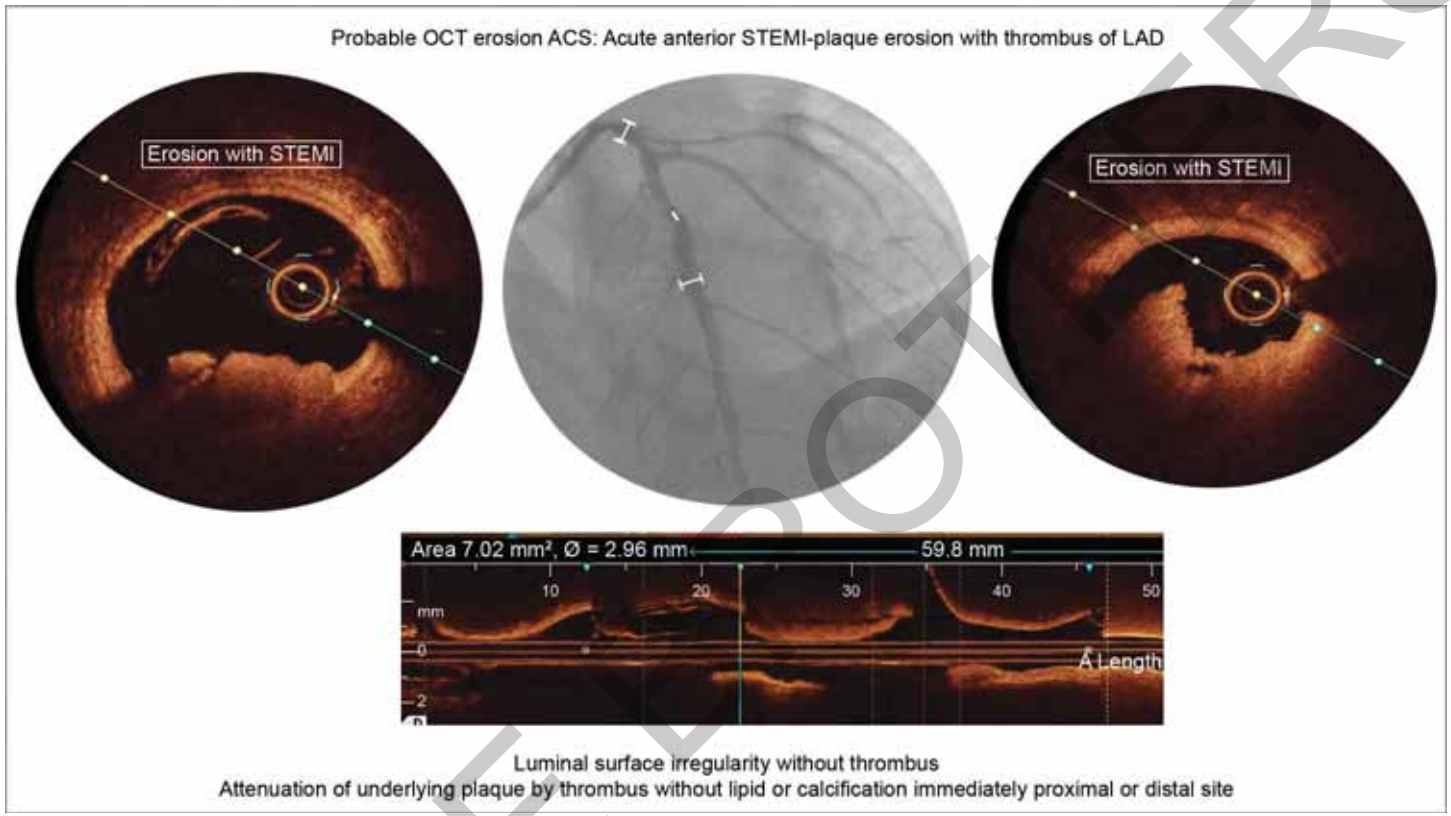


Fig. 1B

Figs. 1A and B: (A) Definite OCT erosion: Plaque erosion with thrombus; (B) Probable OCT erosion ACS: Acute anterior STEMI-plaque erosion with thrombus of LAD. (ACS: acute coronary syndrome; LAD: left anterior descending; OCT: optical coherence tomography; STEMI: ST-elevation myocardial infarction)

Minor Criteria

- Superficial calcified nodule
- Glistening yellow
- Intraplaque hemorrhage
- Endothelial dysfunction
- Outward (positive) remodeling.

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SPOTTY CALCIFICATION

A small amount of calcium is characteristic of vulnerable plaque, and it contributes to plaque instability. Patients with STEMI with PR are more often associated with small and discrete calcifications within an arc of <90°, the presence of a fibro-fatty plaque, and positive remodeling of the culprit arterial segment.[6]

MACROPHAGE INFILTRATION

On OCT, macrophages are seen as signal rich, distinct, or confluent punctuate regions that exceed the intensity of background speckle noise, and positive correlation between histological and OCT data with respect to the density of macrophages on the fibrous cap of the plaque was observed.

CHOLESTEROL CRYSTAL

Phagocytosis of cholesterol crystals by macrophages causes and advances an inflammation in the atherosclerotic plaques **(Figs. 6A to H)**. OCT can visualize cholesterol crystals as a thin linear region of high density without attenuation.

VASA VASORUM (NEOVASCULARIZATION)

Vasa vasorum is seen as a sharply distinct tiny black holes (50–100 μm), signal-free voids located on the intimal side of lipid-rich plaques with TCFA, and is usually seen in at least three successive cross-sections **(Figs. 6A to H)**. Microchannels are located as small black holes in the outer plaque near the adventitia and in the thickened intima.

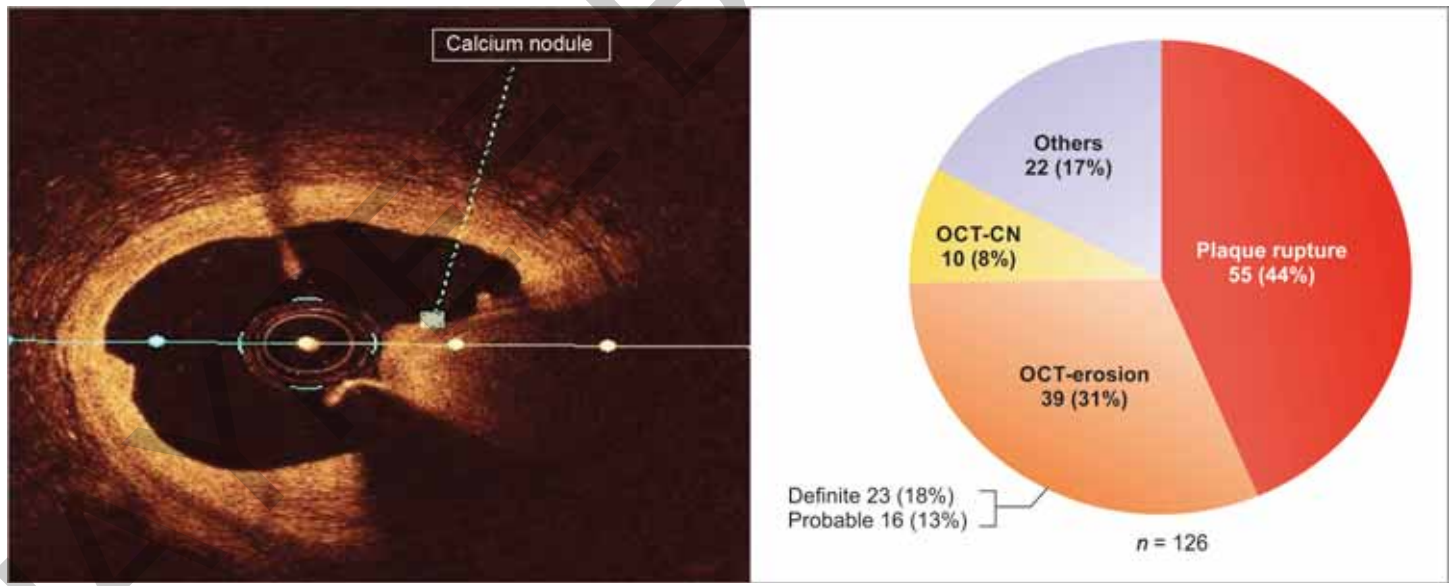


Fig. 2A: Unstable Plaque morphology by OCT.

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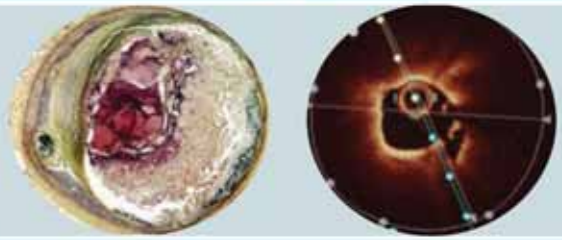
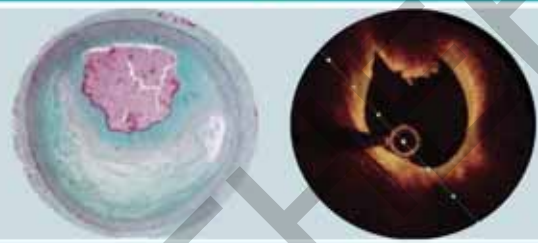
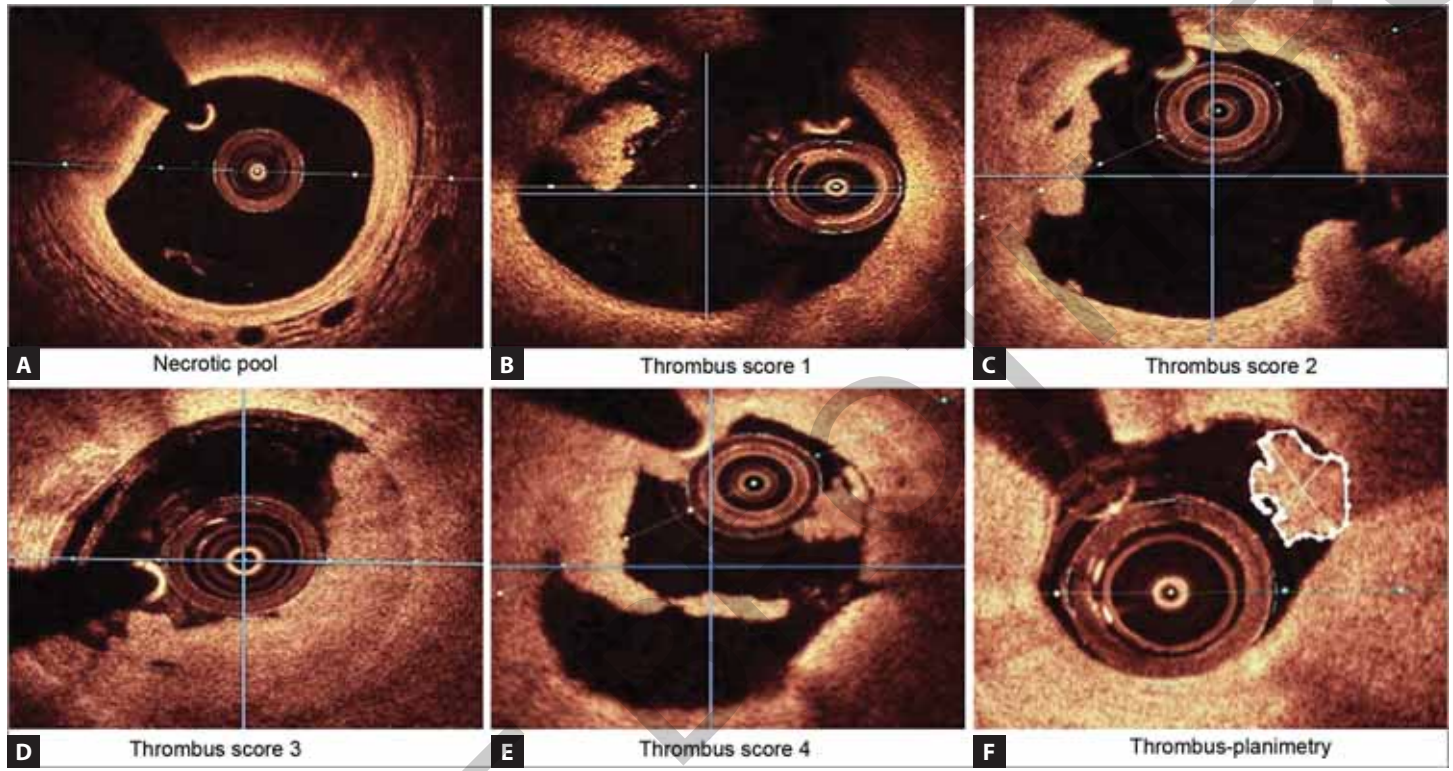
Rupture	Erosion
	
Ruptured thin fibrous cap with empty cavity	Deficiency of endothelium
Smooth muscle cells apoptosis	Endothelial cell apoptosis
Thin fibrous cap	Thick fibrous cap
Large lipid-rich plaque	Less plaque burden
More inflammation	Less inflammation
Majority presents with acute STEMI	Majority presents with NSTEMI and USA
More often in male	More often in female
Majority requires PCI with stenting	Majority can be treated with medical management without intervention

Fig. 2B: Difference between plaque erosion and plaque rupture. (PCI: percutaneous coronary intervention; NSTEMI: non-ST-elevation myocardial infarction)

Vasa vasorum promotes growth of the plaque by increasing blood flow, thereby supplying lipids, inflammatory cells, and cytokines into the plaque, and is frequently associated with positive remodeling and elevated high-sensitive CRP levels. Sometimes, it may cause intraplaque hemorrhage with sudden increase in plaque size and rupture of the plaque, manifest as ACS and sudden death depending upon the acute luminal compromise with reduction in the forward flow of culprit vessel.

Large lipid-rich plaques with TCFA are more often associated with high incidence of slow flow/no reflow, decreased (TIMI) flow, and myocardial blush grade. TCFA is a predictor for type IV (periprocedural) MI and increases the chances of medial edge dissection by six-fold. The presence of ruptured plaque should prompt for an aspiration procedure, followed by implantation of drug eluting stents and an aggressive medical therapy (in particular statins and antiplatelet therapy) and thrombus aspiration may not be useful for patients with plaques containing intact fibrous caps. Plaques with TCFA are seen in about 72% in acute myocardial infarction (AMI), 50% in unstable angina pectoris (UAP), and 20% in SAP. The frequency of OCT-detected plaque

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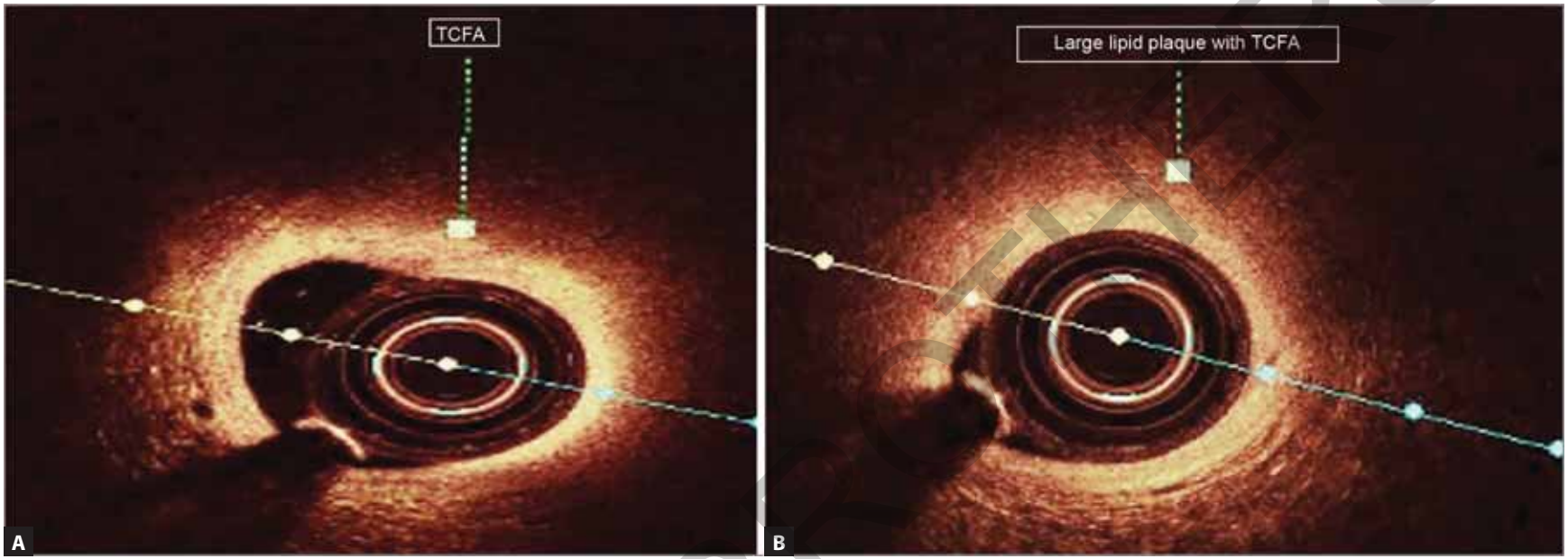
Figs. 3A to F: Thrombus evaluation.

rupture, erosion, and thrombus is similar to that of the pathological reports. ACS may be due to single or multiple culprit large lipid plaques with TCFA and sometimes associated with many nonculprit lesions of culprit or nonculprit vessels which may progress rapidly contributing to increased MACE during follow-up, and is more frequently noticed in diabetic patients.

NEOATHEROSCLEROSIS AND THE VULNERABLE PLAQUE

Neoatherosclerosis with neointimal rupture is one of the most common causes of late and very late drug-eluting stent thrombosis. Neointimal rupture occurs in regions with high-necrotic core content and thin fibrous cap as same as vulnerable plaques of native atherosclerosis. Similarly, it shows high

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Figs. 4A and B: Thin-cap fibroatheroma (TCFA).

incidence of macrophage infiltration and vasa vasorum. Lipid-rich plaque or coronary thrombus causes OCT signal attenuation, which interferes with visualization of deep layers of coronary artery wall especially in segments with positive remodeling.[8]

OCT can be used safely in patients with STEMI. However, in the case of complete vessel occlusion (TIMI 0–1 flow), restoration of vessel patency with thrombectomy needs to be obtained before the OCT pulls back to ensure adequate clearing of the vessel lumen during contrast injection so that adequate images can be obtained.[9,10]

OCT runs of longitudinal view and three-dimensional (3D) reconstruction with angio co-registration is useful in assessing quantification of thrombus and relationship between the thrombus and the location of the major side branches to select the appropriate stent size and the length with IC nitroglycerine or nicorandil injections.

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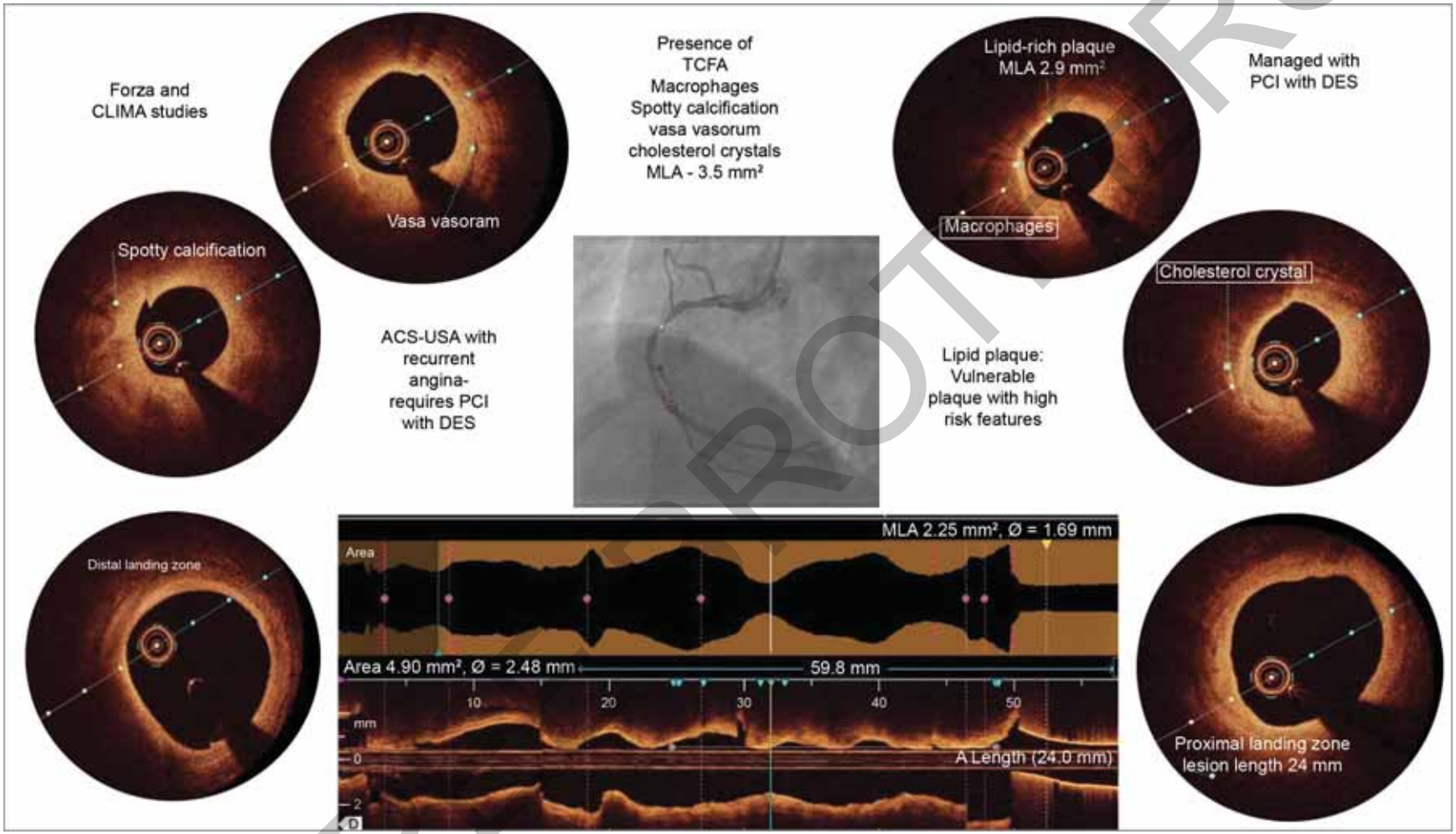


Fig. 5: Detection of vulnerable plaque by OCT imaging. (ACS: acute coronary syndrome; DES: drug eluting stent; MLA: minimal lumen area; PCI: percutaneous coronary intervention; TCFA: thin-cap fibroatheroma)

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TABLE 1: Comparison of OCT with IVUS for identification of vulnerable plaques in ACS.

Plaque morphology	***OCT***	***IVUS***
Plaque rupture	3	2
Fibrous cap	3	0
Necrotic core	2	0±1
Thrombus	3	1
Spotty calcification	3	2
Calcified nodule	3	1
Intimal vasculature	1	0
Macrophages	1	0
Remodeling	1	3
Plaque burden	1	3

(0: not visible; 1: less visible; 2: visible; 3: more visible). (ACS: acute coronary syndrome; IVUS: intravascular ultrasound; OCT: optical coherence tomography)

TABLE 2: Studies showing criteria for detecting high-risk vulnerable plaques with event rates.

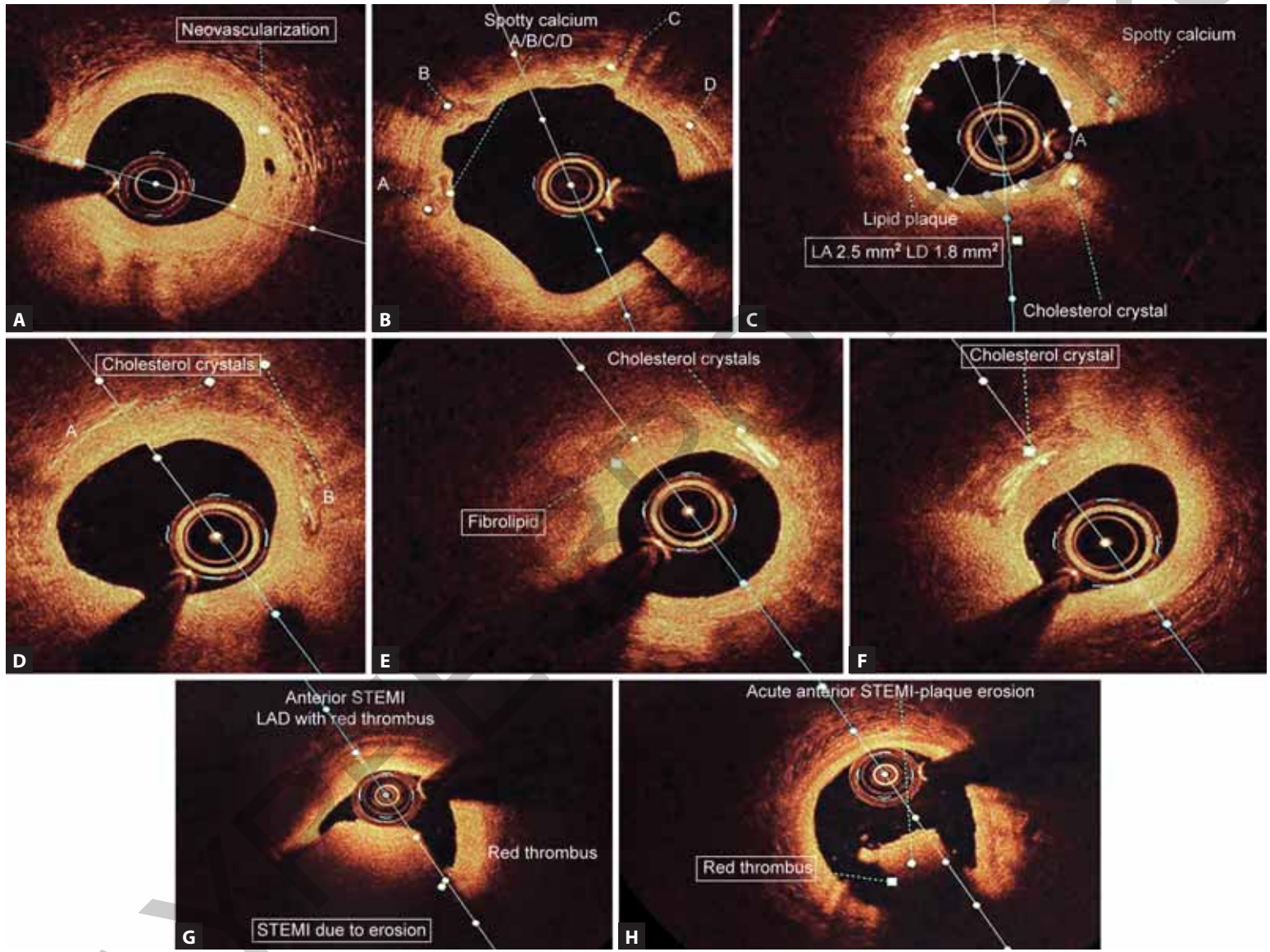
Study	***Modality***	***Variables***	***Endpoint***	***Follow-up***	***PPV***	***NPV***
PROSPECT ($n = 697$)	IVUS and VH-IVUS	PB >70% and MLA <4 mm^2 and VH-IVUS TCFA	MACE (CD, cardiac arrest, MI, re-hospitalization due to AP)	3 years	17%	98%
ATHEROREMO IVUS ($n = 581$)	IVUS and VH-IVUS	PB >70% and MLA <4 mm^2 and VH-IVUS TCFA	MACE (non-CL related or indeterminate death, ACS, unplanned revascularization)	1 year	23%	93%
PREDICTION ($n = 506$)	IVUS and ESS	PB >58% and low ESS <1.0Pa	PCI	1 year	41%	92%
CLIMA ($n = 1,003$) **(Figs. 6A to H)**	OCT	MLA <3.5 mm^2 and TCFA and lipid arc >180° and macrophage	MACE (CD and TV-MI)	1 year	19%	97%

(ACS: acute coronary syndrome; AP: angiina pectoris; ESS: endothelial shear stress; IVUS: intravascular ultrasound; MACE: ; MLA: minimal lumen area; NPV: negative predictive value; OCT: optical coherence tomography; PB: plaque burden; PPV: positive predictive value; TCFA: thin-cap fibroatheroma; VH-IVUS: virtual histology intravascular ultrasound)

BOX 2: Role of imaging in vulnerable plaque detection and risk stratification.

- Large lipid plaque (>60–70%) indicates proneness for plaque rupture and associated with higher incidence of MACE.
- OCT identifies high-risk vulnerable plaques and is also superior to CTCA in predicting future adverse events.
- Identification of high-risk plaque is helpful to differentiate high-risk patients who would benefit from an aggressive therapy for plaque regression.

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Figs. 6A to H: Phagocytosis of cholesterol crystals by macrophages.

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CASE 1

PCI with stenting LMCA and mid LAD—Ambiguous lesion.



CLINICAL HISTORY

A 74-year-old female (no comorbidities), presented with complaints of chest pain associated with grade II SOB on and off. ECG–ACS–evolved AWMI. 2D Echo–RWMA in LAD territory with mild LV dysfunction–EF-47%.

Investigations: Hb-10.4 g/dL, RBS-96 mg/dL, Serum Cr-1.01 mg/dL, eGFR-55 mL/min, and NT-proBNP-480 pg/mL.

CAG showed ostial LMCA 70–80% lesion, distal LAD 70% lesion, normal LCX and RCA.

LMCA engaged with EBU 3.5–6 Fr guiding catheter, LAD lesion crossed with 0.014″ Run through wire and 0.014″ BMW wire placed in LCX. Prestenting OCT was done which significant plaque in ostial LAD-LM diameter found to be fibrolipid plaque **(Figs. 7A to C)**.



PROCEDURE

Distal LAD stenting was done with 2.5 × 23 mm Xience Xpedition stent. Then, LMCA stenting is done with 4.0 × 18 mm Xience Xpedition stent. LAD wire recrossed and POT was done with 4.5 × 08 mm NC Trek balloon. Poststenting OCT showed well-expanded stent struts with no edge dissection in LM to LAD stent, underexpansion in distal LAD stent suggestive of postdilatation which was done with 2.75 × 12 mm NC Trek balloon **(Figs. 8 and 9)**. The final result was good with TIMI III flow with no complications.

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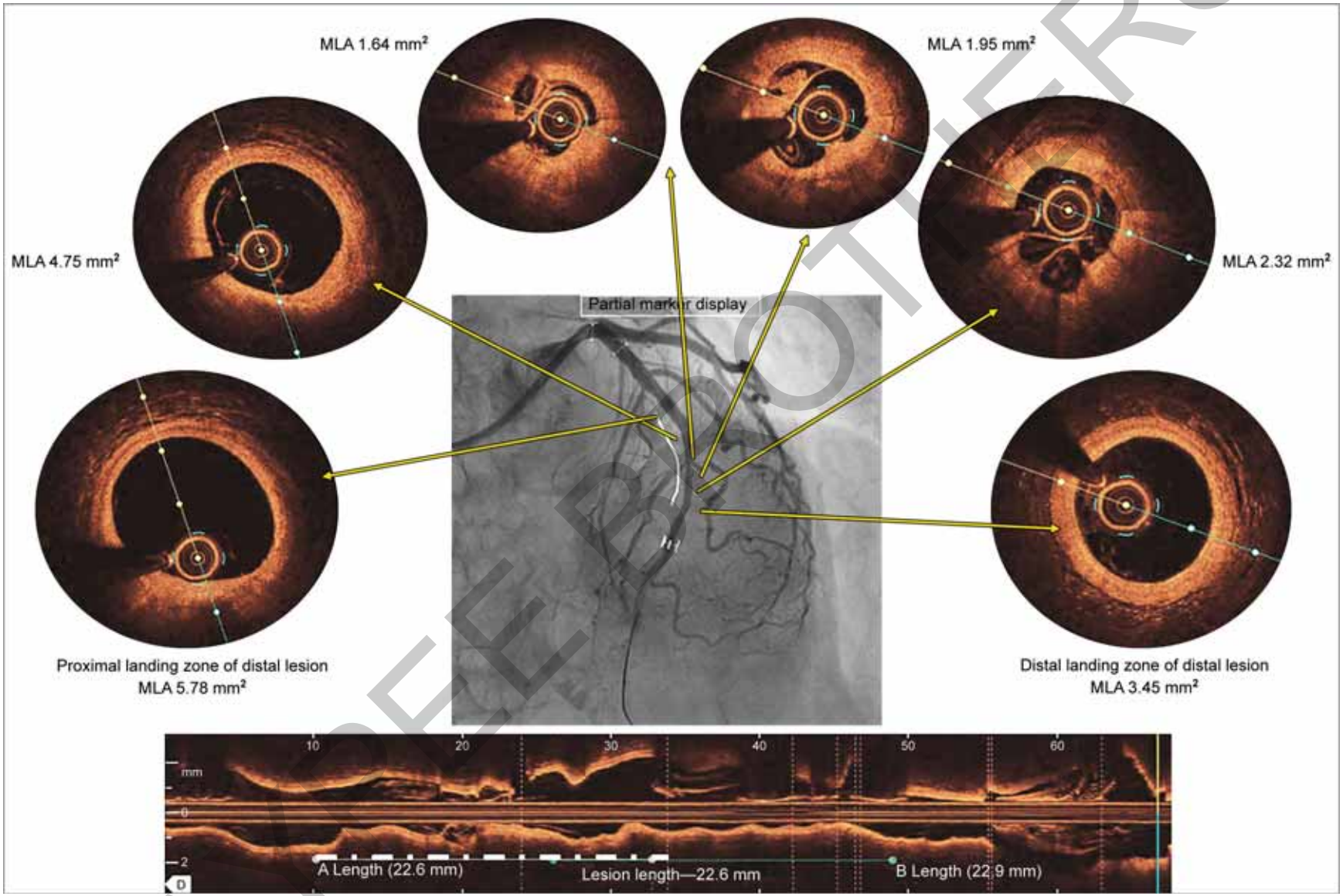


Fig. 7A

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Coronary OCT: A Case-based Approach

This book illustrates many case-based detailed descriptions of Angio and OCT images with attached video files for a comprehensive understanding of the concept of intravascular OCT imaging while performing coronary interventional procedures. The authors took extreme care in choosing cases to narrate the importance of OCT imaging in assessing plaque morphology, lesion length, and diameter for each and every case to use an appropriate pre-PCI modification strategy. Moreover, it highlights the importance of detecting medial edge dissections and malapposition to improve future DES implantation outcomes. This book also emphasizes the usefulness of imaging in various complex high-risk subsets of imaging in addition to simple PCI like ISR, ST, bifurcation lesions, multivessel PCI, PCI of diffuse long lesions, plaque modification and PCI of calcified coronary arteries. This book mainly focuses on developing concepts in reading and interpreting OCT images to make it a more user-friendly manual for those who are inclined to learn and practice OCT imaging in cathlab.



Sridhar Kasturi MD DM FACC FESC FSCAI FICC is the Head in the Department of Cardiology and Cardiothoracic Surgery, KIMS Sunshine Hospitals, Begumpet, Hyderabad, hails from Dharmapuri, Telangana, India. He did his MBBS and MD (General medicine) from Kakatiya Medical College, Warangal, Telangana, and DM (Cardiology) from Gandhi Medical College, Hyderabad, Telangana, India. He was the Chief Medical Director, Senior Interventional Cardiologist, Chairman and Managing Director in Remedy Super Specialty Hospital, Medical Director and Senior Interventional Cardiologist in SHK Super Specialty Hospital and Visiting Professor and Head–Cardiology in Chalmeda Anand Rao Institute of Medical Sciences Karimnagar, Hyderabad, Telangana, India. He was the former Organizing Secretary for Cardiological Society of India – National Interventional Council (2018 and 2023) and President for Cardiological Society of Hyderabad, Telangana Chapter (2017). He is the Course Director for Hyderabad Live, Chairman for Hyderabad Interventional Council and President for Interventional Cardiologists Forum.

He has been performing high-risk complex interventional procedures for the past three decades and performed more than 60,000 angiograms and more 25000 angioplasties like

- Primary PCI
- CHIP CTO
- LM and non-LM bifurcation PCI
- PCI under the guidance of Imaging (OCT and IVUS) and physiology (FFR, and RFR)
- PCI using plaque modification devices like ROTA pro, IVL and orbital atherectomy
- PCI with mechanical circulatory support like IABP, ECMO and IMPELLA
- Noncoronary interventions like PTA of carotid arteries with and without embolic protection devices, PTA of renal and peripheral vessels like iliofemoral, below knee and upper limb vessels, transfemoral stent grafting of aortic aneurysms
- Structural interventions like TAVR, TMVR and TRIC valve

Achievements and Awards:

- He is the first cardiologist in united andhra pradesh to perform bifurcation stent grafting of abdominal aortic aneurysm through a percutaneous approach and device atherectomy for peripheral vascular disease
- He won the 2nd best case award for endovascular repair of a long total occlusion of the thoracic aorta—Takayasu aortitis in EURO PCR-2014 and for the case "Bifurcation PCI-Provisional stenting of LAD/D1in ACS" in EBC-2020
- He is the members of the National and International Faculty of the National Heart Center—Singapore, CAD-ASIA Summit, BVS Summit, India Live, NIC, TCTAP, Bifurcation club, ASIA PCR, EURO PCR, and TCT
- He received fellowships from the European Society of Cardiology, the American College of Cardiology, the Society of Cardiovascular Angiography and Interventions and the Indian College of Cardiology
- He published many review articles and case reports, contributed many clinical studies and multicenter registries.

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