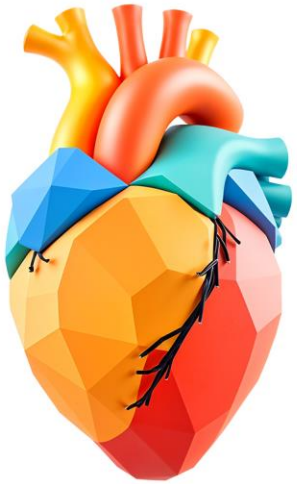

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12th Annual Tehran Heart Center Congress

7th CRITICAL CARDIOVASCULAR CARE

دوازدهمین کنگره سالیانه مرکز قلب تهران

2025

۲۵ و ۲۶ بهمن ماه ۱۴۰۳

13 & 14 February

Tehran Heart Center

Tehran, Iran

Management of stent thrombosis

Management of stent thrombosis

Maryam Mehrpooya MD,
Associate professor of cardiology

Fellowship of Interventional Cardiology
Tehran university of medical science
Imam Khomeini Hospital

Nightmare for cardiologist

Panel B: Complication of Coronary intervention

Moderator: Dr. Haji Zeinali

Panelists: Dr. Amirzadegan, Dr. Eslami, V, Dr. Taghavi, Dr. Khalilpour, Dr. Ghorbanzadeh

9:30 -10:15	Stent thrombosis management	Dr. Mehrpouya
	Coronary dissection & perforation management	Dr. Moezi
	No reflow & slow flow management	Dr. Rouzi Talab

Introduction

- An important cause of acute MI is stent thrombosis
- Primary PCI for stent thrombosis is less effective (76%–80% successful reperfusion) than primary PCI for native artery occlusion
- STEMI caused by stent thrombosis is also associated with an increased risk for long-term MI (23%) and repeat stent thrombosis (15%) compared with STEMI caused by native artery occlusion

JACC: CARDIOVASCULAR INTERVENTIONS

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STATE-OF-THE-ART REVIEW

Stent Thrombosis

A Clinical Perspective

Bimmer E. Claessen, MD, PhD,* José P.S. Henrique
Roxana Mehran, MD,‡§ Jan J. Piek, MD, PhD,* Geo

TABLE 1 Definition of Stent Thrombosis According to the Valve Academic Research Consortium

Level of Certainty	Timing
Definite	**Early**
Angiographic or pathological confirmation of partial or total thrombotic occlusion within the peri-stent region	Acute (<24 h)
AND at least 1 of the following additional criteria:	Subacute (24 h to 30 days)
Acute ischemic symptoms	
Ischemic electrocardiogram changes	
Elevated cardiac biomarkers	
Probable	**Late**
Any unexplained death <30 days of stent implantation	31 days to 1 yr
Any myocardial infarction related to documented acute ischemia in the territory of the implanted stent without angiographic confirmation of stent thrombosis and in the absence of any other obvious cause	
Possible	**Very Late**
Any unexplained death beyond 30 days	>1 yr

Introduction

An Intraprocedural stent thrombosis (IPST) and acute stent thrombosis (<24 hours from implantation) ***are rare events, occurring 0.8% to 1.2% and 0.1% to 0.9% of cases,*** **respectively**

TABLE 3 Predictors of Early and (Very) Late Stent Thrombosis

	Early Stent Thrombosis	(Very) Late Stent Thrombosis
Patient	Malignancy, heart failure, peripheral artery disease, diabetes mellitus, acute coronary syndromes, nonadherence to dual-antiplatelet therapy, genetic polymorphisms, thrombocytosis	End-stage renal disease, smoking, STEMI, nonadherence to dual-antiplatelet therapy (unknown for very late ST)
Lesion	Bifurcation lesion, LAD, vessel size, lesion length, thrombus, saphenous vein grafts	LAD, incomplete endothelialization, delayed healing, previous brachytherapy, vein graft stenting
Procedural	Stent undersizing, stent underexpansion, stent malapposition, dissection, no pre-procedural thienopyridine administration, bivalirudin as anticoagulant in STEMI patients, stent length	DES (compared with BMS), permanent polymer DES (compared with bioresorbable polymer DES), overlapping DES
Post-procedural	Discontinuation of antiplatelet therapy	Discontinuation of antiplatelet therapy (unknown for very late ST), late acquired stent malapposition

Procedure

Small vessels
Edge dissections
Bifurcation
Multiple/long stenting
Slow flow/no reflow

Plaque

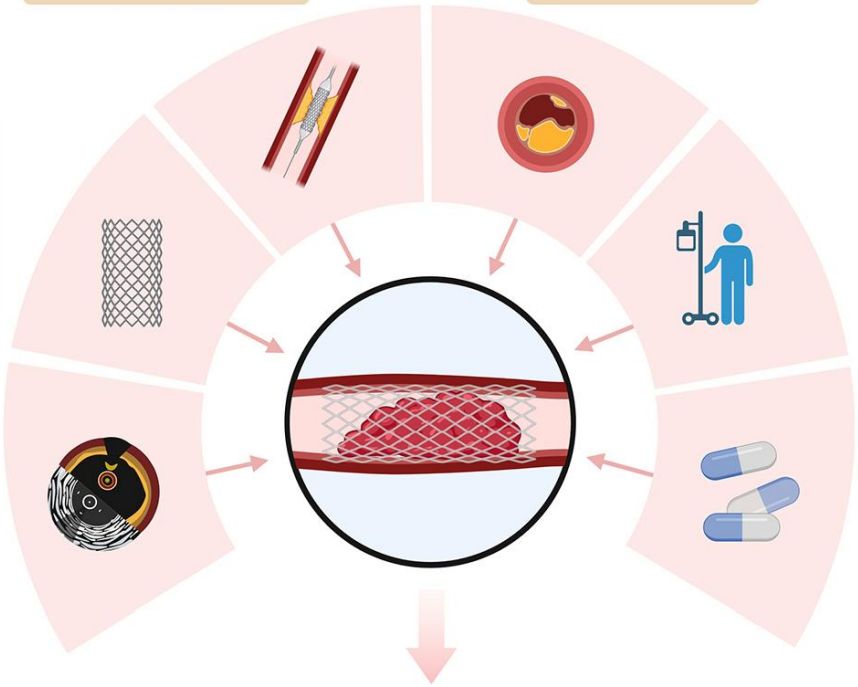
Lipid rich plaque
Big necrotic core
Severe calcification
Plaque ulceration

Stent

First generation DES
Underexpansion
Undersizing
Strut thickness
Fracture
Malapposition

Patient

ACS
Diabetes
CKD
COPD
Malignancy
Individual susceptibility
AHF at index procedure



IV imaging

Major residual dissection
Plaque protrusion
Major malapposition
Plaque at stent edges
Incomplete rehendotelization
Thrombus at index procedure

DAPT

Contraindication to DAPT
Early interruption
Genetic plymorphisms
Non-response to P2Y12 inhibitors

Stent thrombosis

Treatment for ST and the role of intravascular imaging

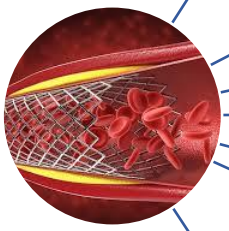
Prevention

- Avoiding stent thrombosis requires operators to be meticulous about anticoagulation and stent implantation

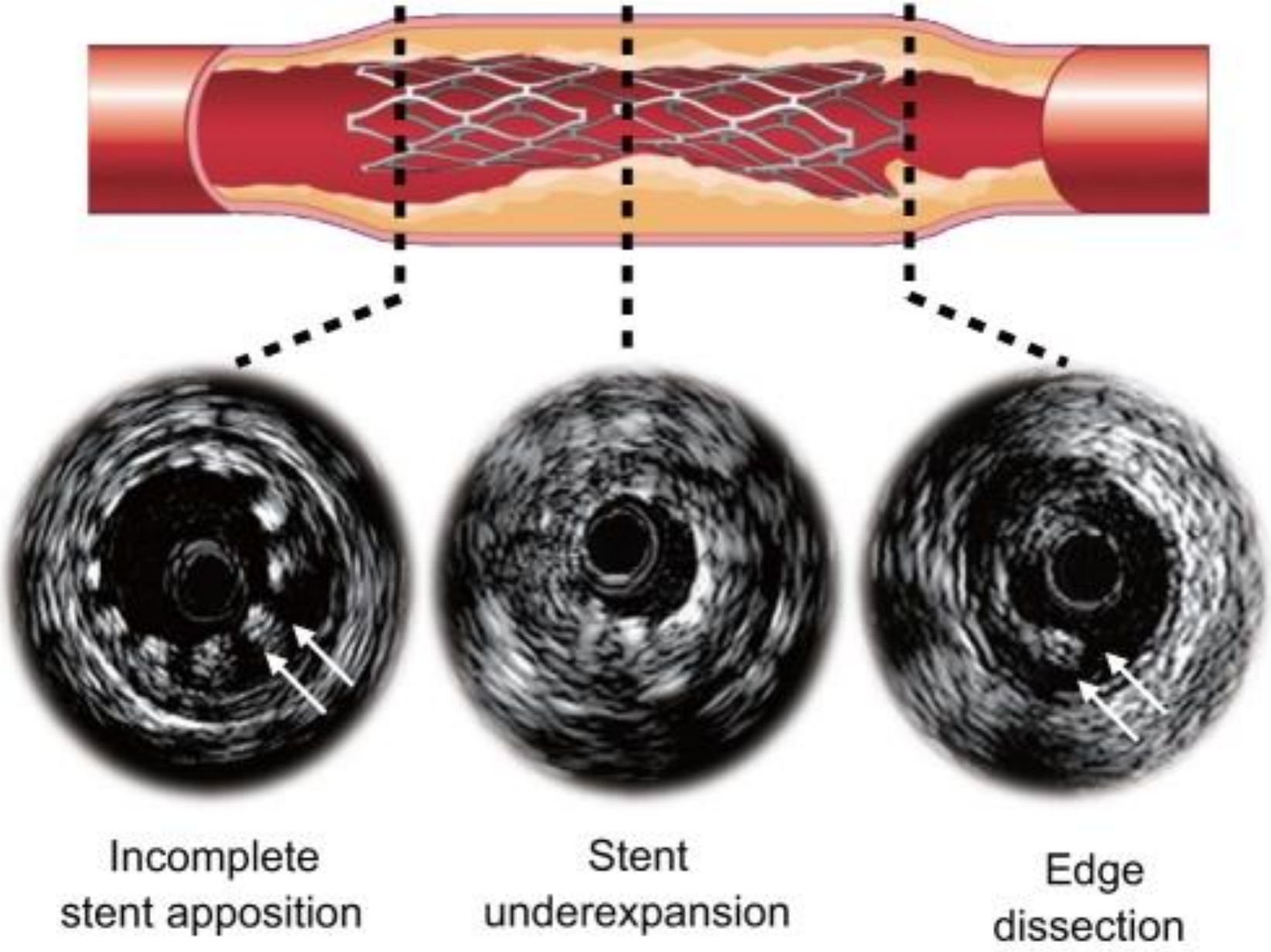
- A fundamental axiom is confirming adequate outflow prior to stent implantation

- Placing a stent in an artery with no-reflow due to microvascular obstruction or a vessel with poor distal runoff is risky and may increase the likelihood of stent thrombosis

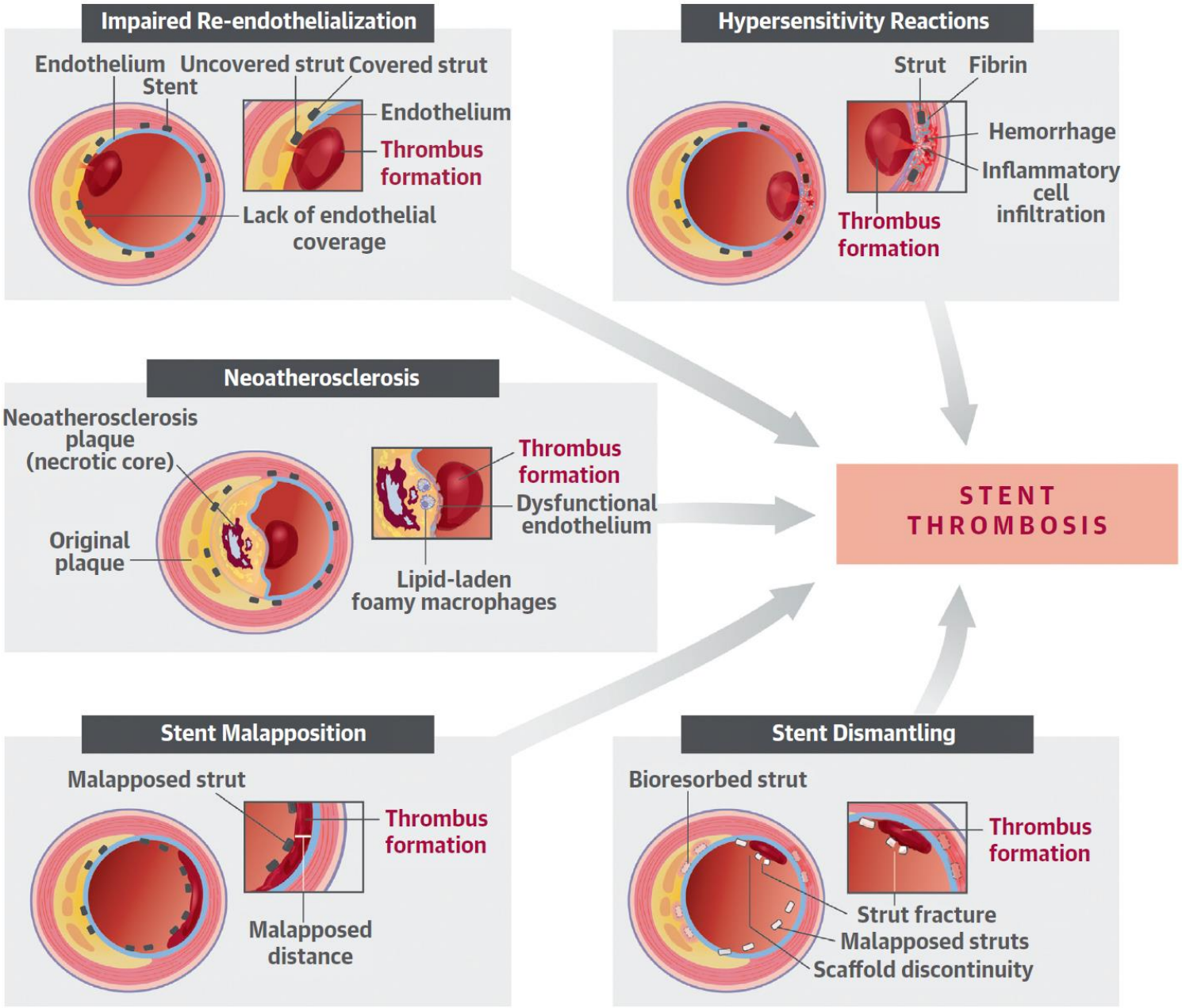
Procedural Mechanisms of ST



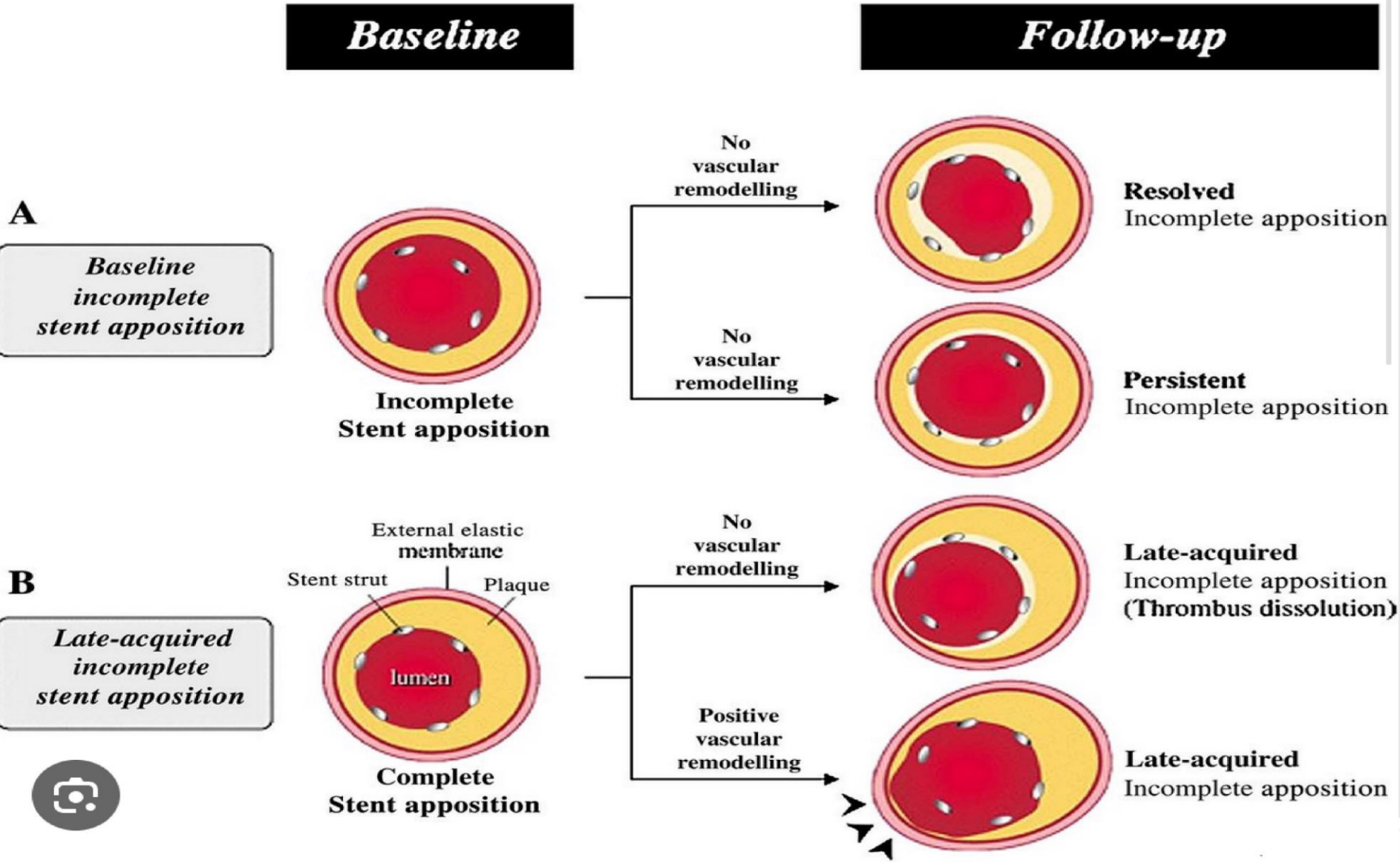
- Stent underexpansion
- Stent malapposition
- Dissections
- New plaque rupture
- Stent fracture
- Negative remodeling at the stent edge
- Neoatherosclerosis


Incomplete
stent apposition
Stent
underexpansion
Edge
dissection

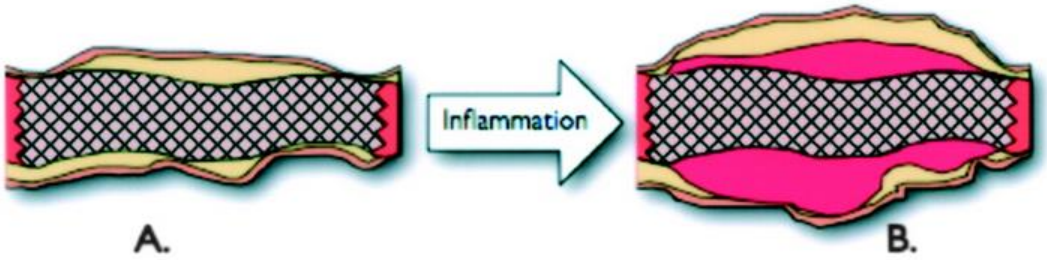
CENTRAL ILLUSTRATION Pathogenic Mechanisms of Late ST/Very Late ST



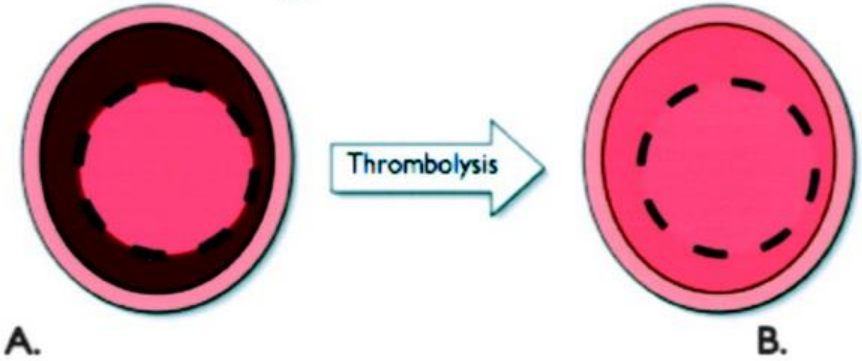
Torrado, J. et al. J Am Coll Cardiol. 2018;71(15):1676–95.


Baseline
Follow-up
A
Baseline incomplete stent apposition
Incomplete Stent apposition
No vascular remodelling
Resolved
Incomplete apposition
No vascular remodelling
Persistent
Incomplete apposition
B
Late-acquired incomplete stent apposition
External elastic membrane
Stent strut
Plaque
lumen
Complete Stent apposition
No vascular remodelling
Late-acquired
Incomplete apposition
(Thrombus dissolution)
Positive vascular remodelling
Late-acquired
Incomplete apposition

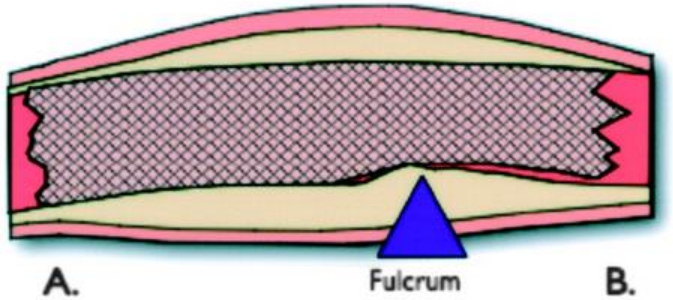
I. Incomplete stent apposition due to positive vessel remodeling

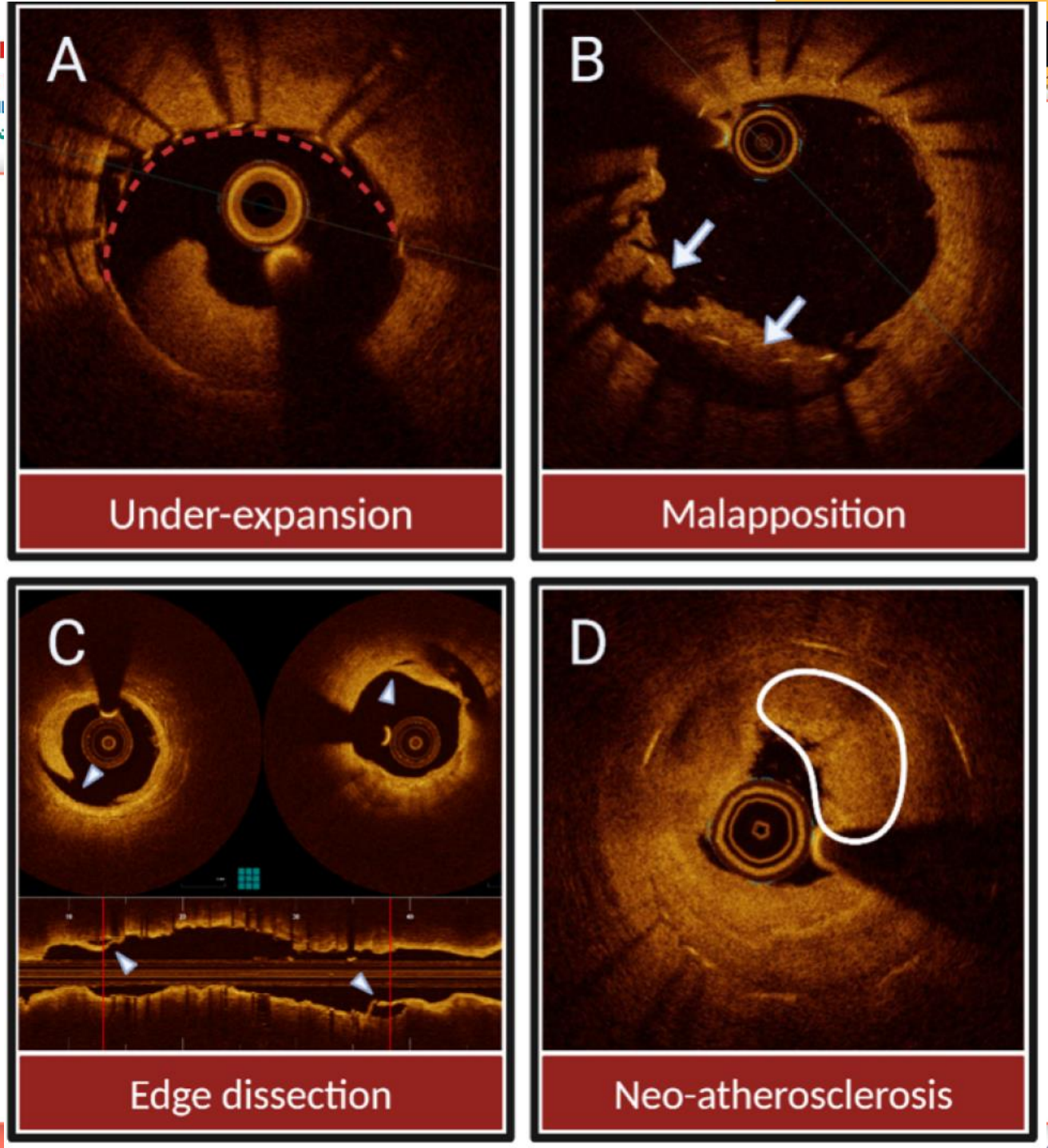


II. Incomplete stent apposition due to thrombus dissolution

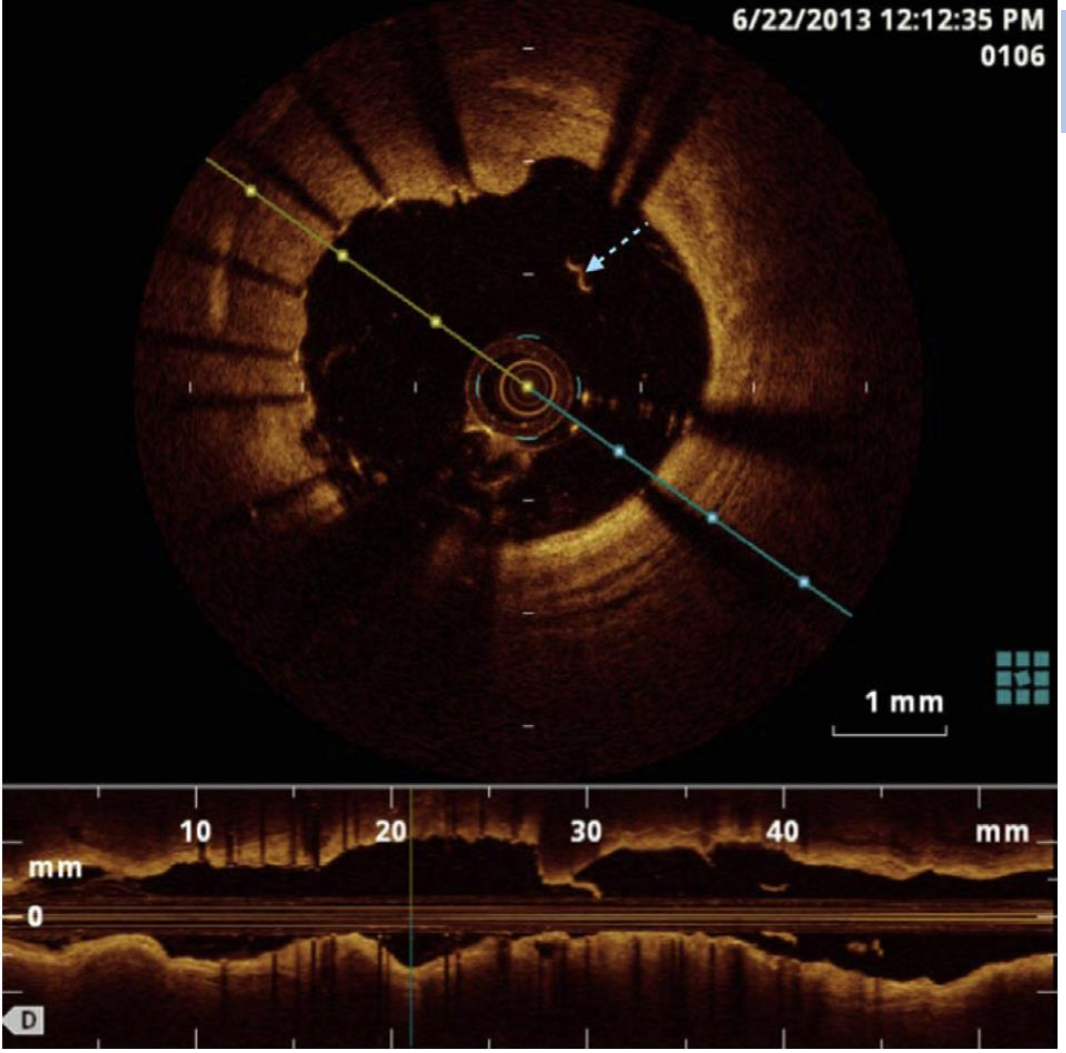


III. Incomplete stent apposition due to stent underexpansion



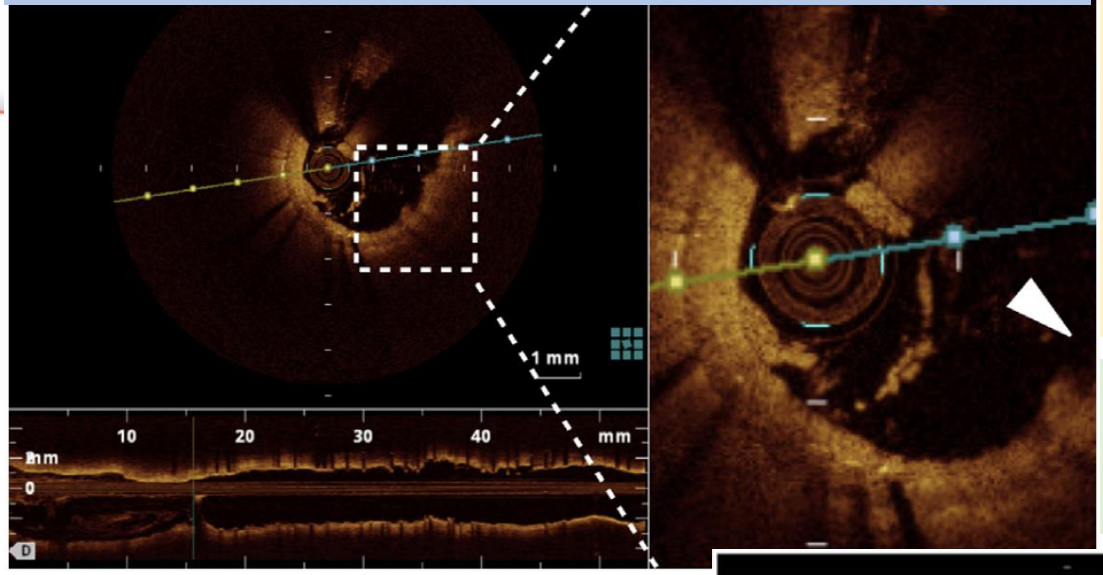

A
Under-expansion
B
Malapposition
C
Edge dissection
D
Neo-atherosclerosis

Stent Malapposition Detected by OCT Imaging



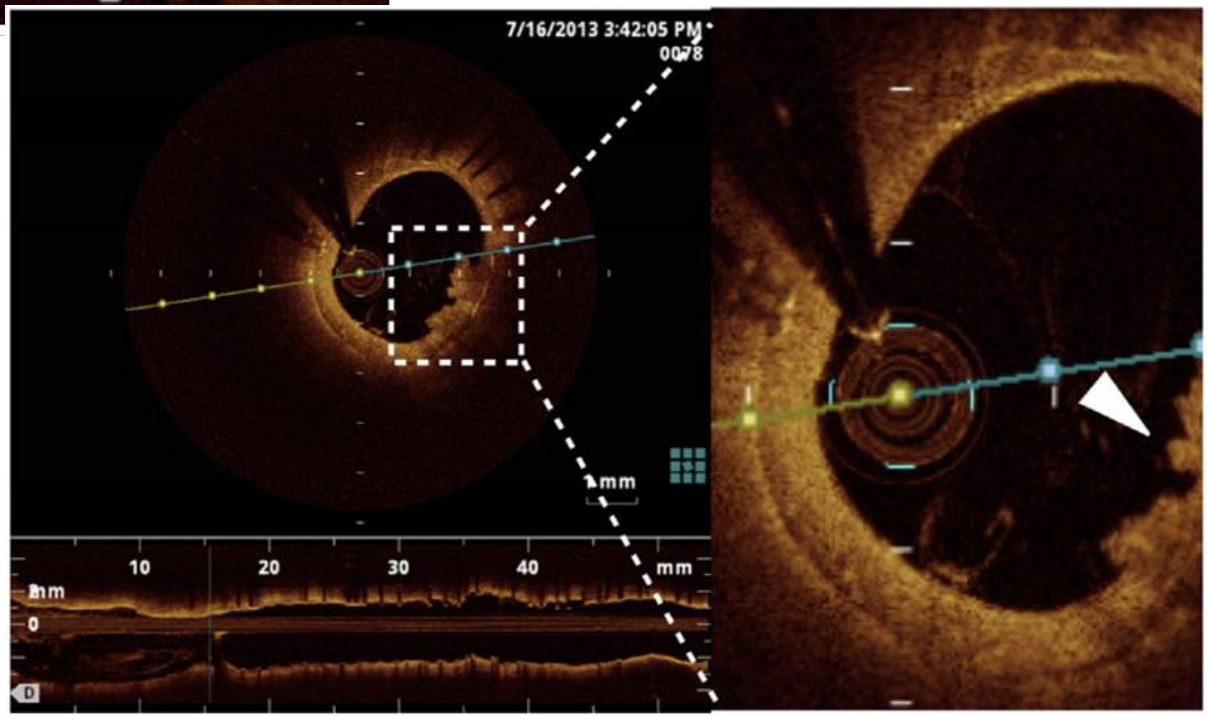
- (Top) The axial image shows an OCT catheter in the center of RCA artery
- At the 2-o'clock position, bright, reflective stent struts are visualized and are not in contact with the intima of the artery *(blue dotted arrow)*
- There is a gap of 0.6 mm between these stent struts and the artery
- This finding indicates *stent malapposition*, a mechanical, stent-based risk factor for late stent thrombosis

Neoatherosclerosis as a Cause of Very Late Stent Thrombosis of a DES Placed in an SVG



- Three years after DES placement to the distal body of an SVG to the RCA, this patient presented with unstable angina
- SVG angiography revealed a severe, hazy filling defect in the SVG stent
- He was placed on IV heparin
- The following day, OCT of the SVG was performed
- Two sequential OCT frames are presented with full and magnified images (dotted white box regions)

OCT of the stent reveals mild neointima within the stent, but at the 4-o'clock position, there is evidence of cap disruption and a residual crater (white arrowhead,



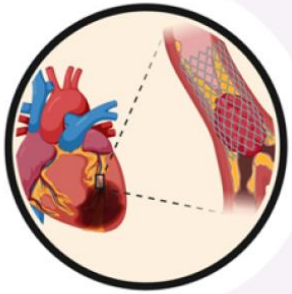
- (Bottom) In the adjacent OCT frame, the same region *at the 4-o'clock position demonstrates an irregular contour consistent with thrombus within the stent*
- Stent struts are noted covered by neointima (small arrowheads at the 2-o'clock position)
- These findings indicate plaque rupture of neoatherosclerosis within a stent, a recently appreciated mechanism of stent thrombosis

Treatment

- When confronted with acute or IPST, one must be very prompt at restoring perfusion to minimize myocardial damage and the approach to treatment should include both pharmacologic and mechanical optimization

- Certainly, if intracoronary thrombus develops, ***prompt aspiration thrombectomy or angioplasty*** should be performed immediately to restore patency

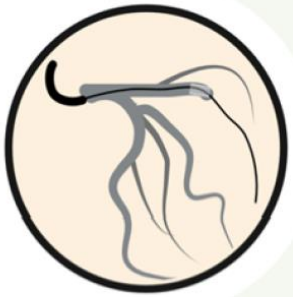
- ***Therapeutic anticoagulation*** should be confirmed and more potent antiplatelet therapy considered, as both ***IV cangrelor and glycoprotein inhibition*** were both associated with less IPST.



Stent thrombosis

- Antiplatelet drug check
- Consider onset timing

Urgent PCI



Guidewire advancement and angiographic evaluation

- High thrombus amount → TA and/or i.c. GPIIb/IIIa inhibitors
- Low thrombus amount → i.v cangrelor (in P2Y12 inhibitor naive patients)

Obtain vessel patency and adequate flow

Index procedure or second staged procedure
(if patient's conditions do not allow further investigations during index PCI)

Identify the mechanism

IVI and/or stent enhancement techniques

- Stent-related issues
  - Underexpansion
  - Malapposition
  - Fracture
- Procedure-related issues
  - Edge dissection
  - Plaque missing
  - Plaque protrusion
- In-stent restenosis
  - Neoatherosclerosis
  - Neointimal hyperplasia

Individualized treatment

Treatment

- Bailout glycoprotein inhibitor, loading with either prasugrel or ticagrelor, or switching to cangrelor to rapidly reach maximal steady state platelet inhibition may be helpful if not already using these medications

- If there is suspicion the patient could have heparin-induced thrombocytopenia (HITT), switching to a direct-thrombin inhibitor such as bivalirudin should be considered

- Intravascular imaging with either IVUS or OCT should be used to determine stent apposition, expansion, and presence of edge dissections

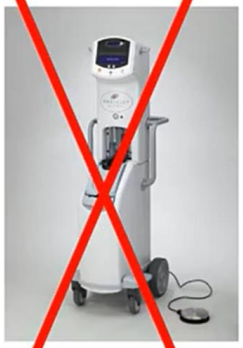
Treatment

- **Optimization of stent deployment with appropriate postdilation and treatment of edge dissections with additional stents will be imperative to prevent repeat stent thrombosis**

- **Additional stent implantation should be done judiciously because each millimeter of stent increases the probability of IPST**

Thrombectomy

Mechanical



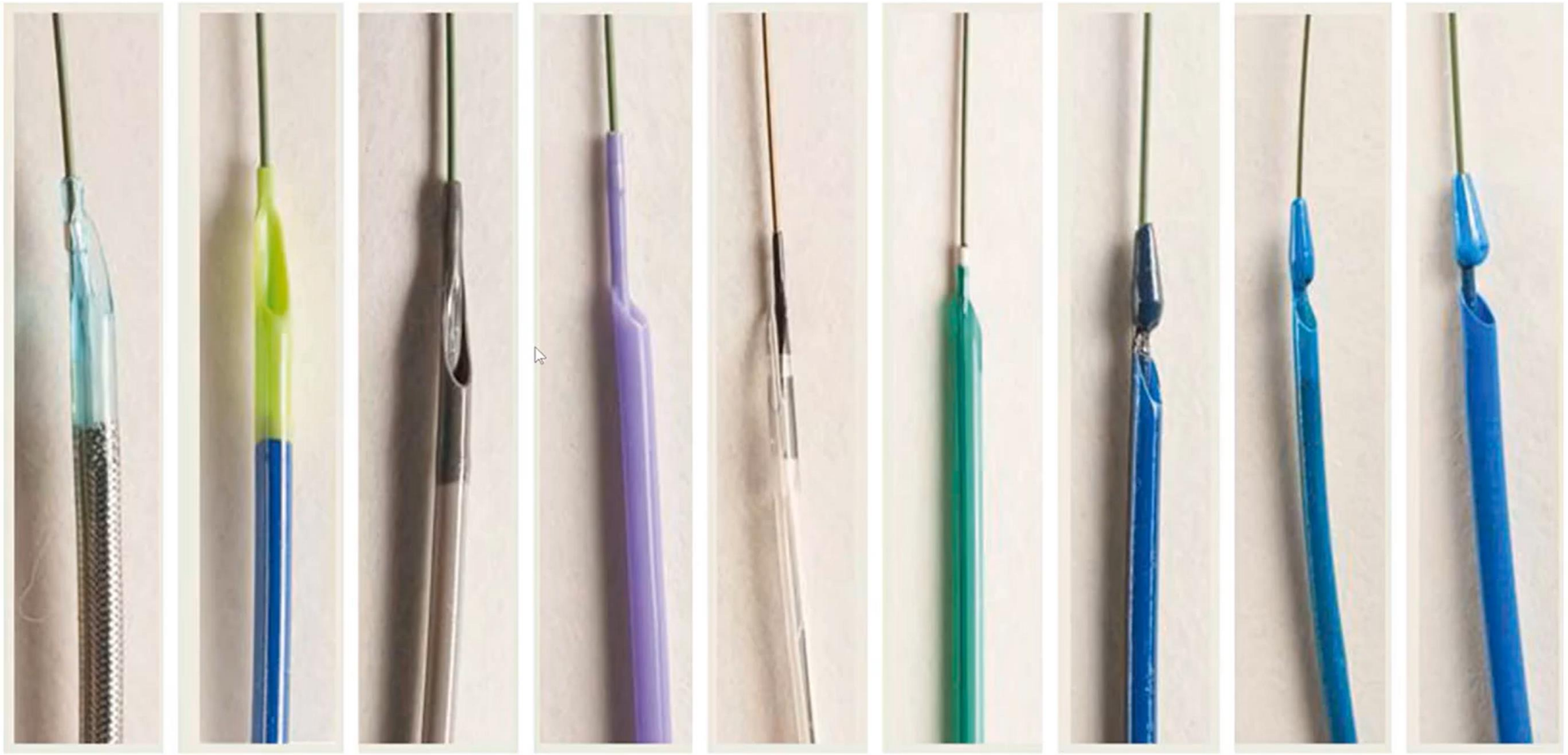
Rheolytic thrombectomy (AngioJet)

Aspiration

Export

Penumbra

Through guide/guide extension



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Bavry T. Cardiovascular Intervention: A Companion to Braunwald's Heart Disease 2015

Procedural Mechanisms of ST

- Stent underexpansion
- Stent malapposition
- Dissections
- New plaque rupture
- Stent fracture
- Negative remodeling at the stent edge
- Neoatherosclerosis

Procedural Mechanisms of ST

❖Stent underexpansion:

- If confirmed by IVUS/OCT, high-pressure balloon angioplasty with noncompliant balloons sized according to the normal adjacent reference segment can be attempted

- A new stent can be particularly deleterious in treating stent underexpansion if the reason is due to a highly rigid (e.g., calcified) segment because multiple layers of stent metal will be present in an underexpanded lesion

- If balloon expansion is ineffective, then the patient should be temporarily treated with balloon angioplasty and return for definite therapy after the acute ST presentation has

Procedural Mechanisms of ST

❖Stent malapposition :

- This can be verified by the existence of a space filled with blood between the stent struts and the vessel wall
- The extent of this low-flow area has been associated with ST
- Angioplasty with an appropriately sized balloon as determined by the IVUS/OCT-derived measurement of the arterial wall diameter at the culprit cross sections typically suffices to ameliorate this problem

❖**Dissections :** Stent edge dissections can provoke ST and are easily detectable by IVUS/OCT, despite being sometimes dubious angiographically. A new stent can typically cover them and restore adequate

❖**New plaque rupture:** This condition essentially means the absence of ST per se and supports the extension of thrombosis within the stent after initiation at the adjacent plaque disruption area. A new stent may be needed to treat the culprit new lesion area.

Procedural Mechanisms of ST

❖Stent fracture :

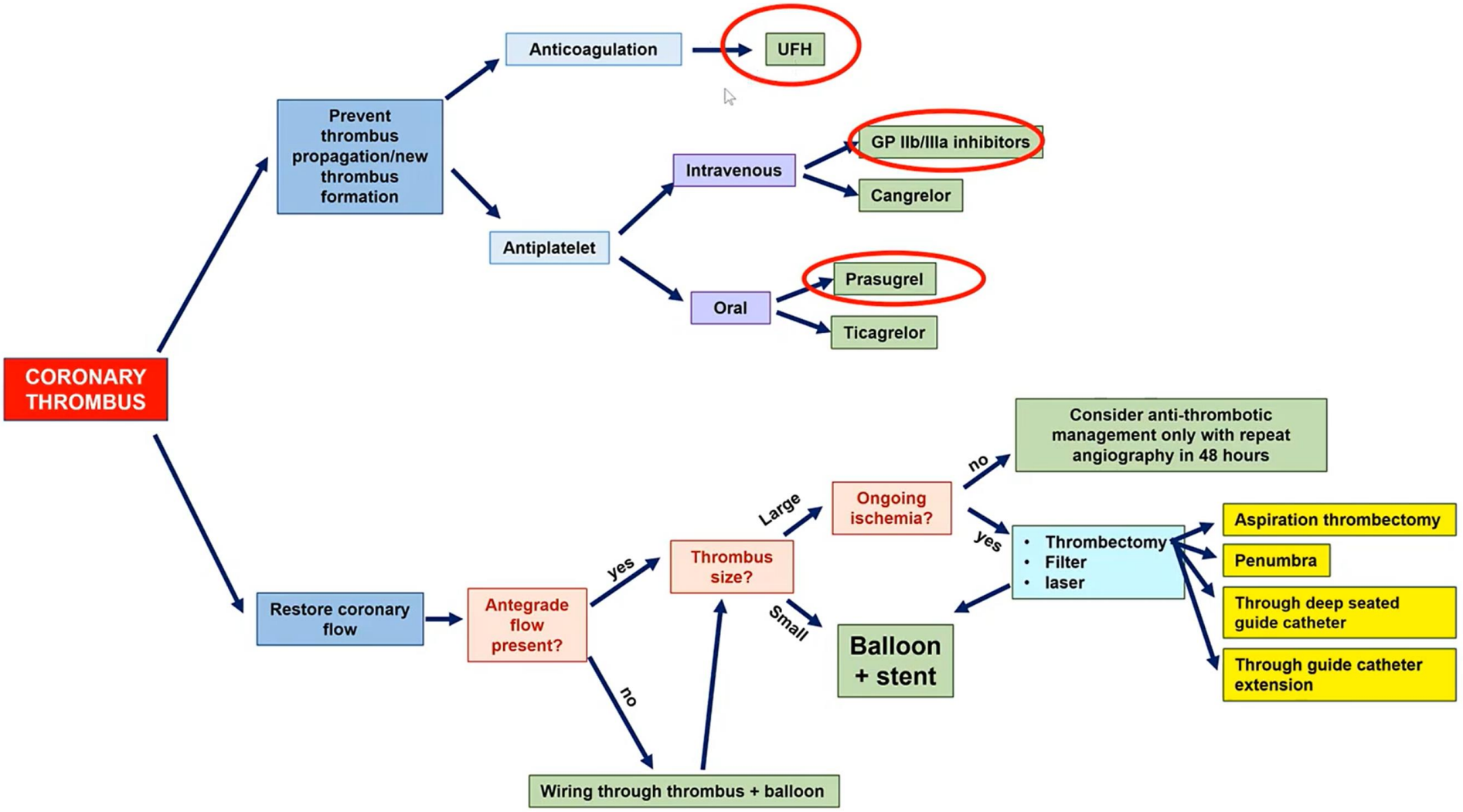
- RCA lesions, excessive tortuosity or angulation of the vessel, overlapping stents, and longer stents have been associated with an increased risk of stent fracture
- A stent fracture may cause mechanical damage to the endothelium that could subsequently lead to ST and can be treated with implantation of a short stent to cover the area of the stent fracture.

❖Negative remodeling at the stent edge

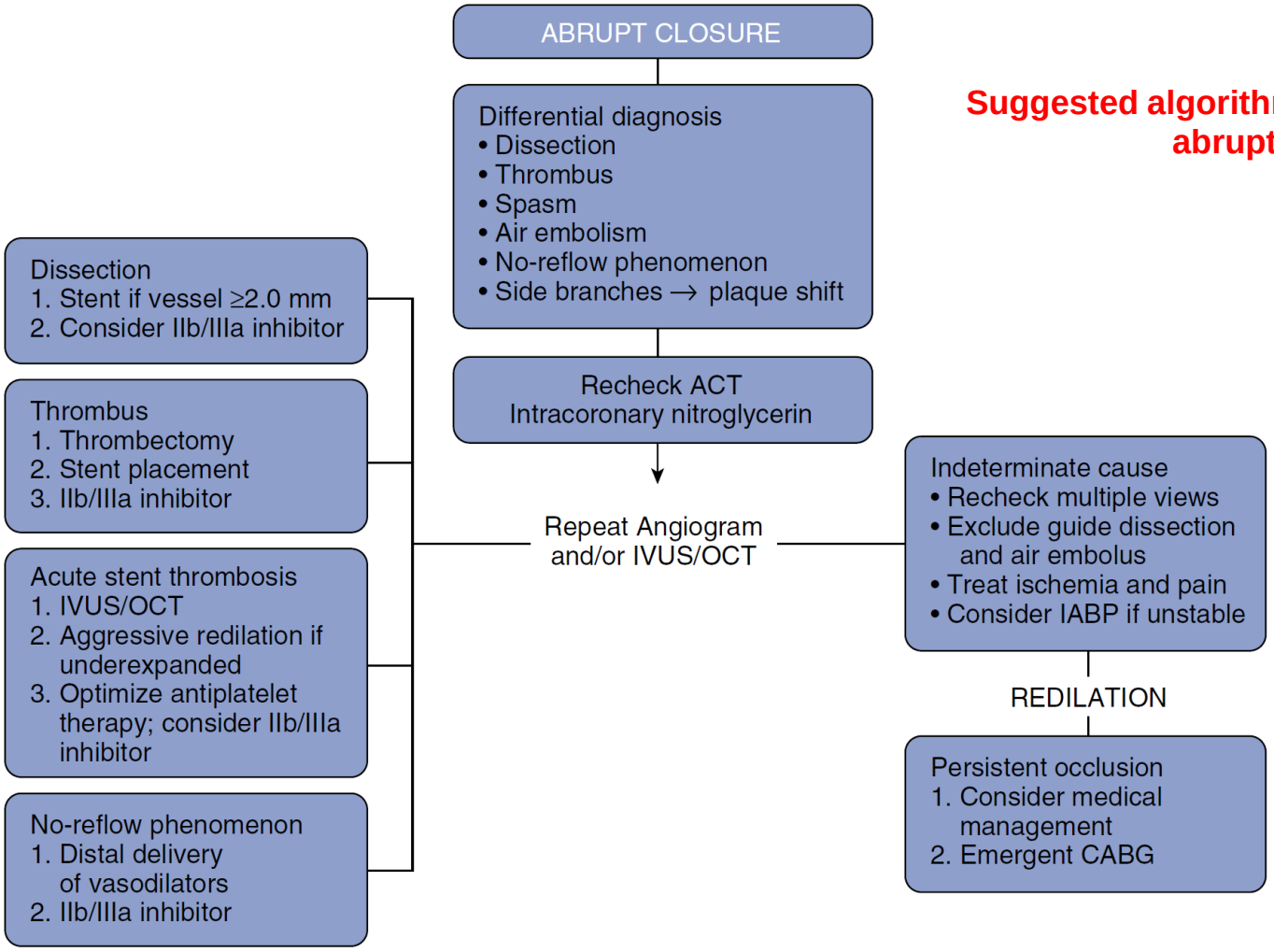
This can explain abrupt lumen compromise distal to a stent that might have predisposed to flow reduction

❖Neoatherosclerosis

The development of an ACS caused by rupture or erosion of a neoatherosclerotic plaque in a previously stented lesion has been acknowledged as a potential culprit in very late ST


CORONARY THROMBUS
Prevent thrombus propagation/new thrombus formation
Anticoagulation
UFH
Antiplatelet
Intravenous
GP IIb/IIIa inhibitors
Cangrelor
Oral
Prasugrel
Ticagrelor
Restore coronary flow
Antegrade flow present?
yes
no
Thrombus size?
Large
Small
Ongoing ischemia?
no
yes
Consider anti-thrombotic management only with repeat angiography in 48 hours
Thrombectomy
Filter
laser
Aspiration thrombectomy
Penumbra
Through deep seated guide catheter
Through guide catheter extension
Balloon + stent
Wiring through thrombus + balloon

Suggested algorithm for management of abrupt closure



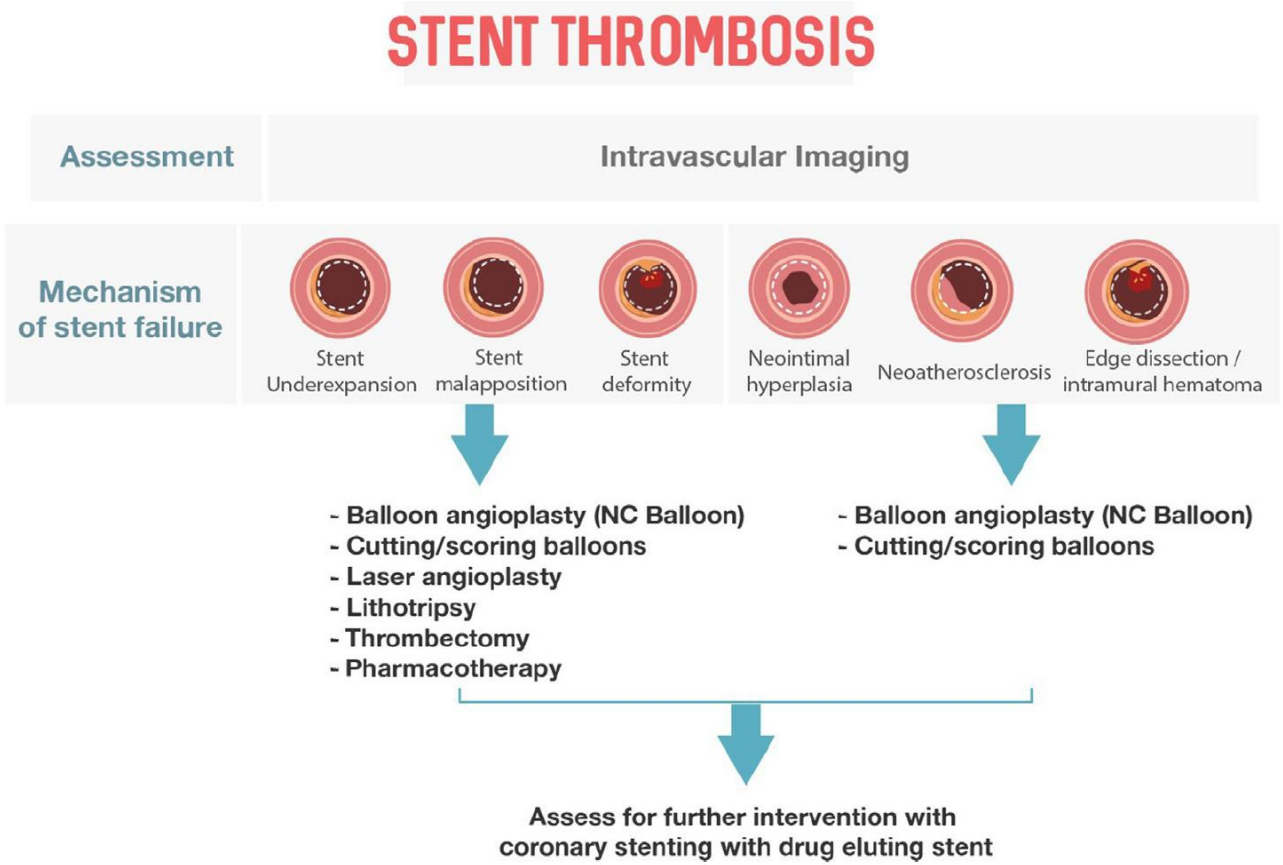


Figure 6.
SCAI algorithmic approach to stent thrombosis.

Take home messages

- A comprehensive approach encompassing precise diagnosis, urgent procedural interventions, and tailored pharmacological treatments is essential for optimizing patient outcomes during ST

- Intracoronary antithrombotic therapies combined with TA and IVI guidance play pivotal roles in achieving optimal outcomes, particularly in cases with a high thrombus burden and complex stent-related issues

- When ISR or ST is encountered, imaging should be strongly considered to optimize the subsequent approach to these challenging cases.

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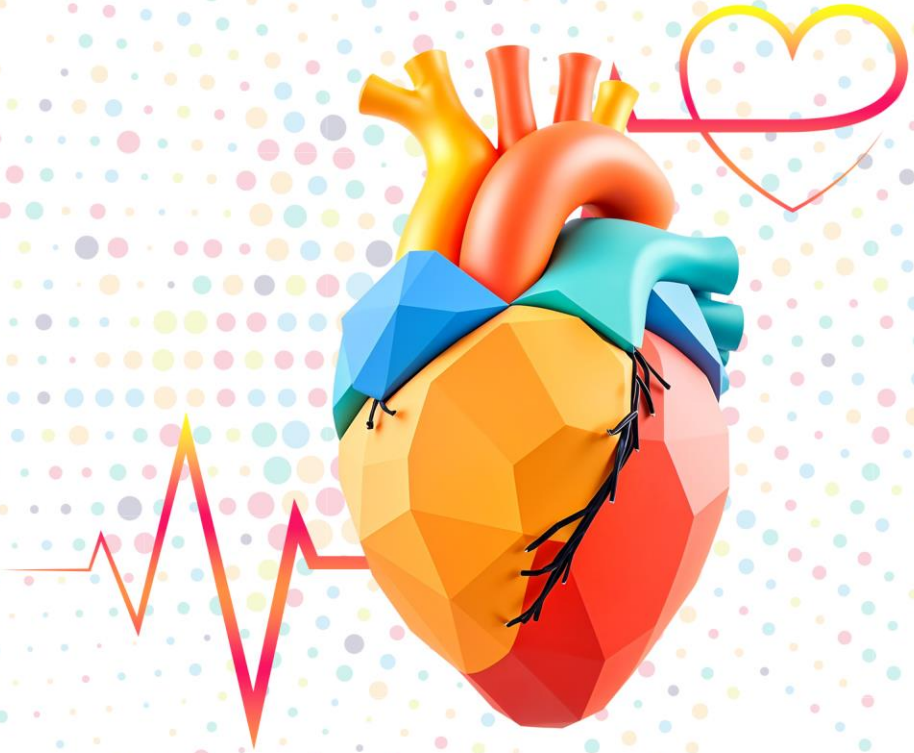

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