Līdzfinansē Eiropas Savienība
Nacionālais attīstības plāns 2027
Veselības ministrija
PAULA STRADIŅA KLĪNISKĀ UNIVERSITĀTES SLIMNĪCA
Metodiskās vadības institūcija kardioloģijā

Eiropas Sociālā fonda Plus projekts Nr. 4.1.2.7/1/24/I/001 ''Pilnveidot pacientu drošību un aprūpes kvalitāti''

Kāpēc akūts koronārs sindroms ir svarīgs? Patofizioloģisks skatījums

Asoc. prof. Kārlis Trušinskis,
PSKUS, LU

LATVIJAS HIPERTENSIJAS UN ATEROSKLEROZES BIEDRĪBA
dzīve ir tā vērta

Neprogresējošas un progresējošas aterosklerotiskas pangas

Neprogresējoša panga

Progresējoša panga

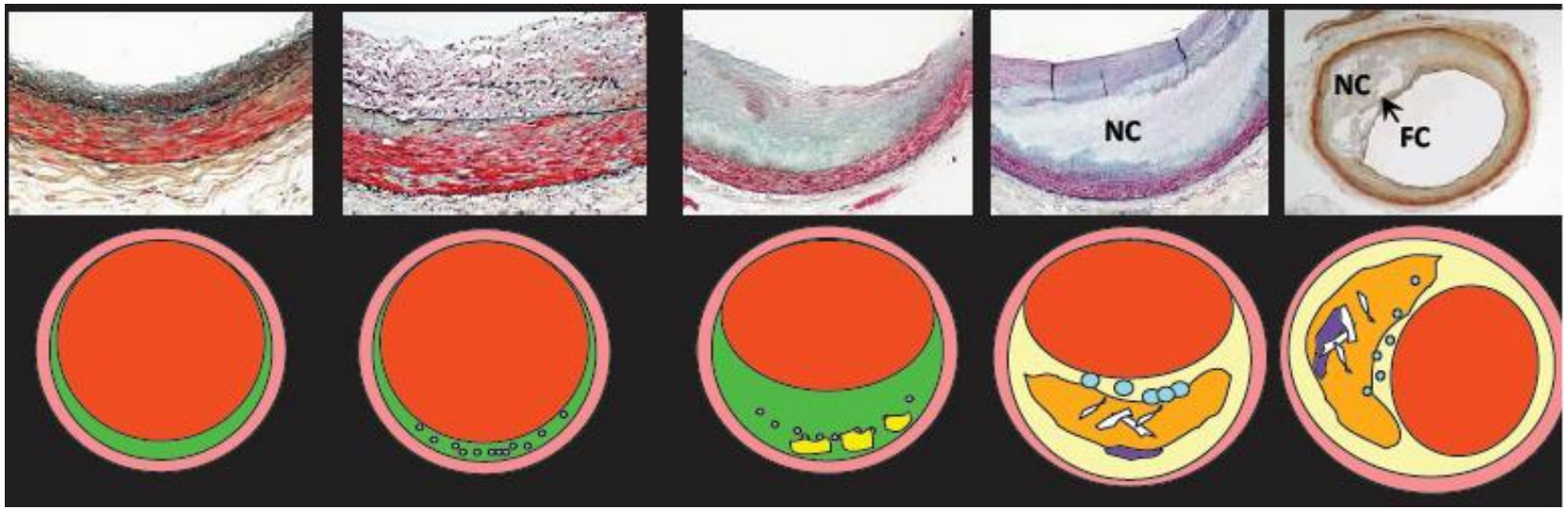
Intīmas sabiezējums

Intīmas ksantoma

Patoloģisks intīmas sabiezējums

Fibroateroma

Plānas kapsulas fibroateroma



Lipīdu ieslēgumi

Nekrotiskie audi

agrīna ▷ vēlīna nekroze

Adaptēts no R.Virmani, AHA 2011

JACC FOCUS SEMINAR: HISTORICAL AND CONCEPTUAL CHANGES OF CORONARY ARTERY DISEASE (1980–2020)

JACC STATE-OF-THE-ART REVIEW

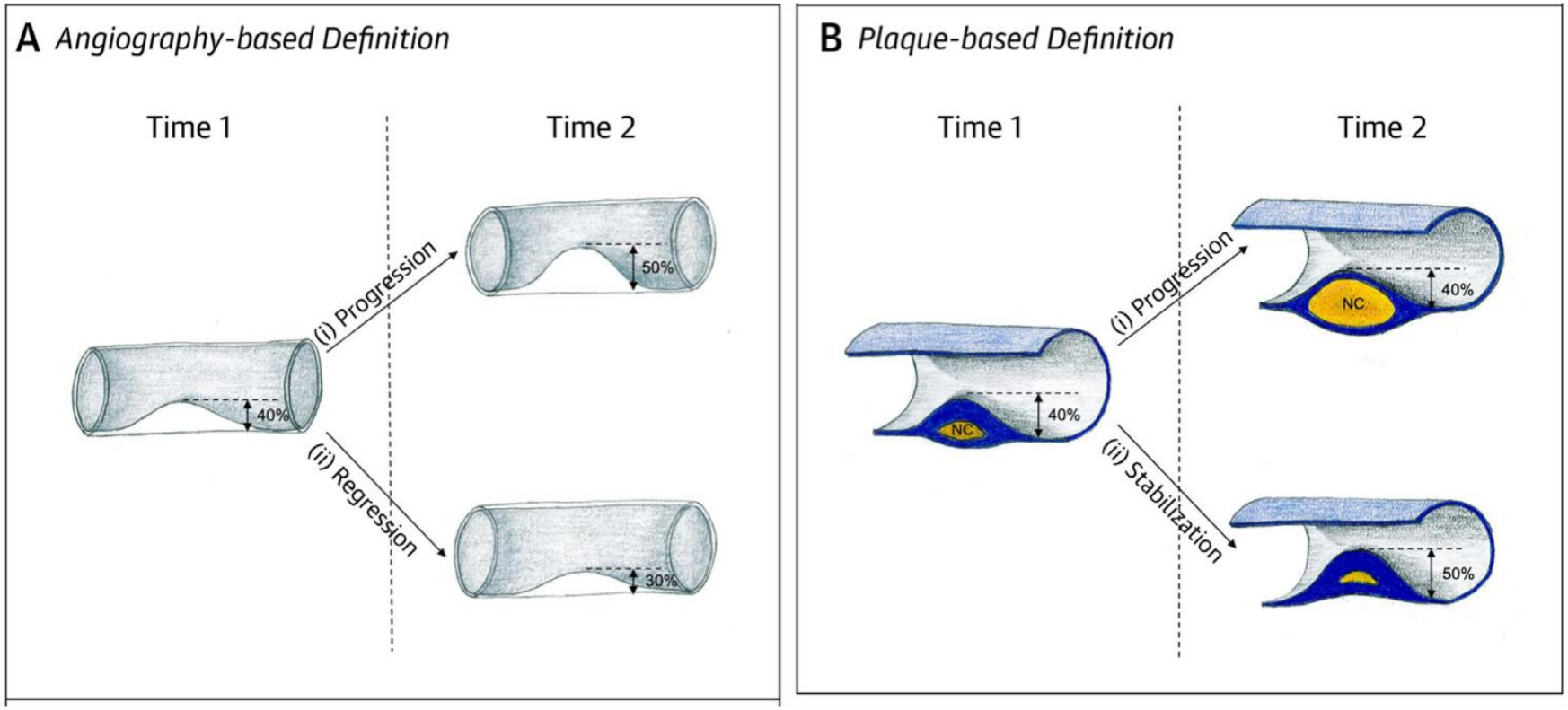
From Subclinical Atherosclerosis to Plaque Progression and Acute Coronary Events

JACC State-of-the-Art Review

Amir Ahmadi, MD,[a,b] Edgar Argulian, MD,[c] Jonathon Leipsic, MD,[b] David E. Newby, MD,[c] Jagat Narula, MD, PhD[a]

Aterosklerozes progresija

FIGURE 2 What Is Plaque Progression and Regression?



In routine clinical practice, plaque progression and regression are defined by changes in luminal stenosis by an invasive coronary angiogram **(A)**. However, with advances in invasive and noninvasive imaging, one could consider the change in the overall plaque volume and its morphology in defining progression and regression, especially because it is primarily the plaque morphology that determines the fate of a lesion **(B)**. An increase in necrotic core volume with significant positive remodeling and fibrous cap thinning, regardless of a change in the luminal stenosis, is demonstrated in **B(i)**. This could be considered as plaque progression. A decrease in necrotic core volume with a resultant increase in the fibrous cap thickness with or without calcification, regardless of a change in the luminal stenosis, is demonstrated in **B(ii)**; a mild increase in luminal stenosis can take place because of negative remodeling. This represents plaque stabilization or lesion regression.

JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY
VOL. ■, NO. ■, 2025

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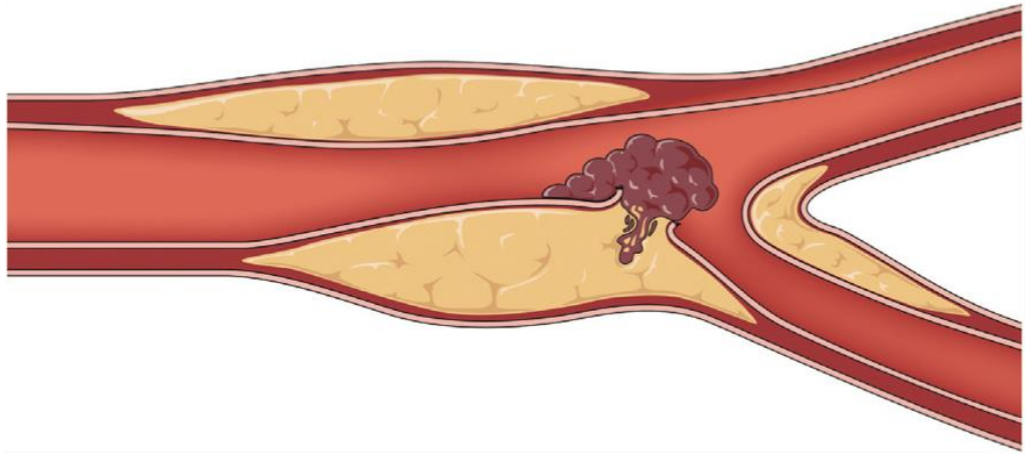
CLINICAL PRACTICE GUIDELINE

2025 ACC/AHA/ACEP/NAEMSP/SCAI Guideline for the Management of Patients With Acute Coronary Syndromes

Akūta koronāra sindroma vadlīnijas

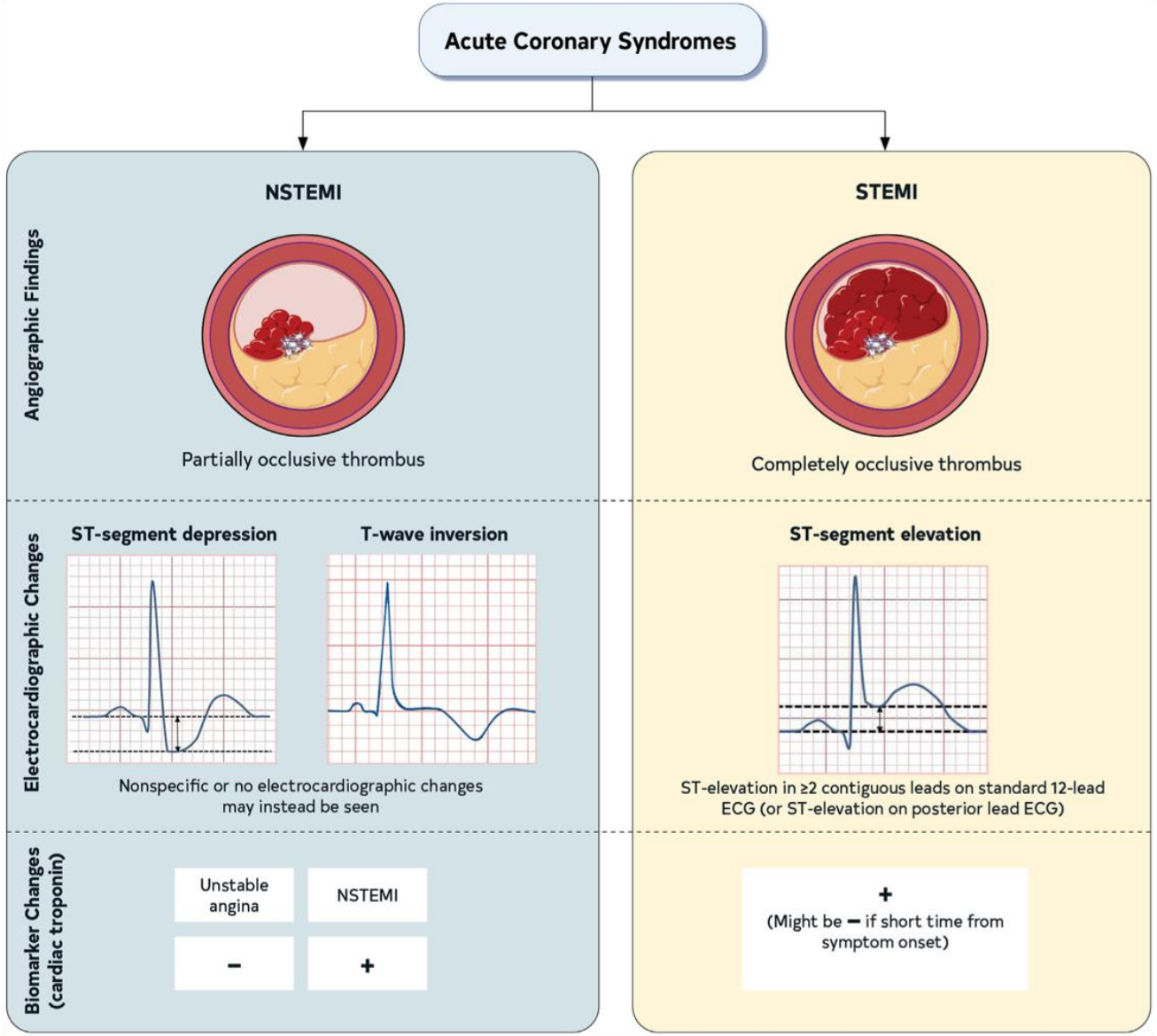
FIGURE 1 Pathobiology of a Type 1 Myocardial Infarction Due to Atherosclerotic Plaque Disruption



Progressive lipid accumulation and inflammation within an atherosclerotic plaque may lead to plaque instability. Rupture or erosion of the atherosclerotic plaque and exposure of plaque contents to the circulation may then culminate in activation of the coagulation cascade and subsequent thrombosis. When this occurs in the epicardial vessels of the coronary circulation, the presence of thrombus may compromise flow to the myocardium, leading to myocardial ischemia and eventual myonecrosis. Illustration by Patrick Lane, ScEYEnce Studios. Copyright 2025 American College of Cardiology Foundation, and American Heart Association, Inc.

Akūta koronāra sindroma vadlīnijas

FIGURE 2 Types and Classification of Acute Coronary Syndromes



NSTEMI indicates non-ST-segment elevation myocardial infarction; and STEMI, ST-segment elevation myocardial infarction. Illustration by Patrick Lane, ScEYEnce Studios. Copyright 2025 American College of Cardiology Foundation, and the American Heart Association, Inc.

Plaque Erosion: A Distinctive Pathological Mechanism of Acute Coronary Syndrome

published: 28 September 2021
doi: 10.3389/fcvm.2021.711453

Xing Luo [1,2†], Ying Lv [1,2†], Xiaoxuan Bai [1,2†], Jinyu Qi [1,2], Xiuzhu Weng [1,2], Shaoyu Liu [2,3], Xiaoyi Bao [1,2], Haibo Jia [1,2] and Bo Yu [1,2]*

[1] Department of Cardiology, 2nd Affiliated Hospital of Harbin Medical University, Harbin, China, [2] Key Laboratory of Myocardial Ischemia, Ministry of Education, Harbin Medical University, Harbin, China, [3] Bin Xian People's Hospital, Harbin, China

Aterosklerotiskās pangas ruptūra vs erozija

1/3 no ST elevāciju miokarda infarkta pacientiem

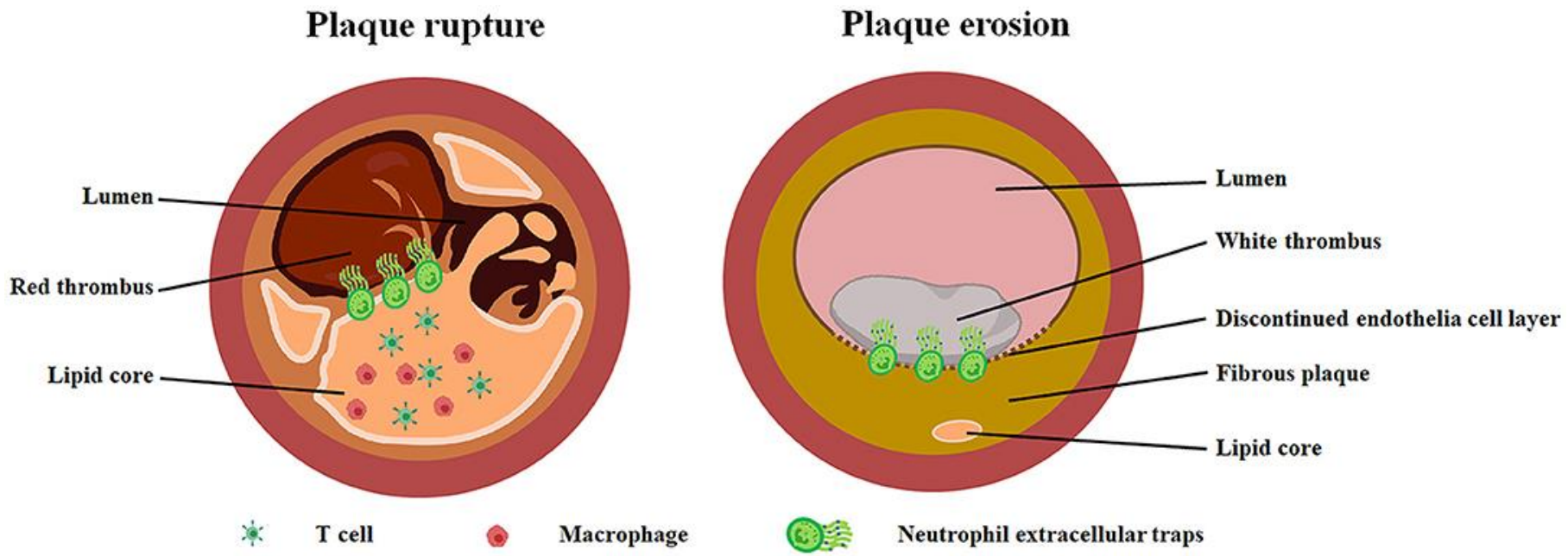
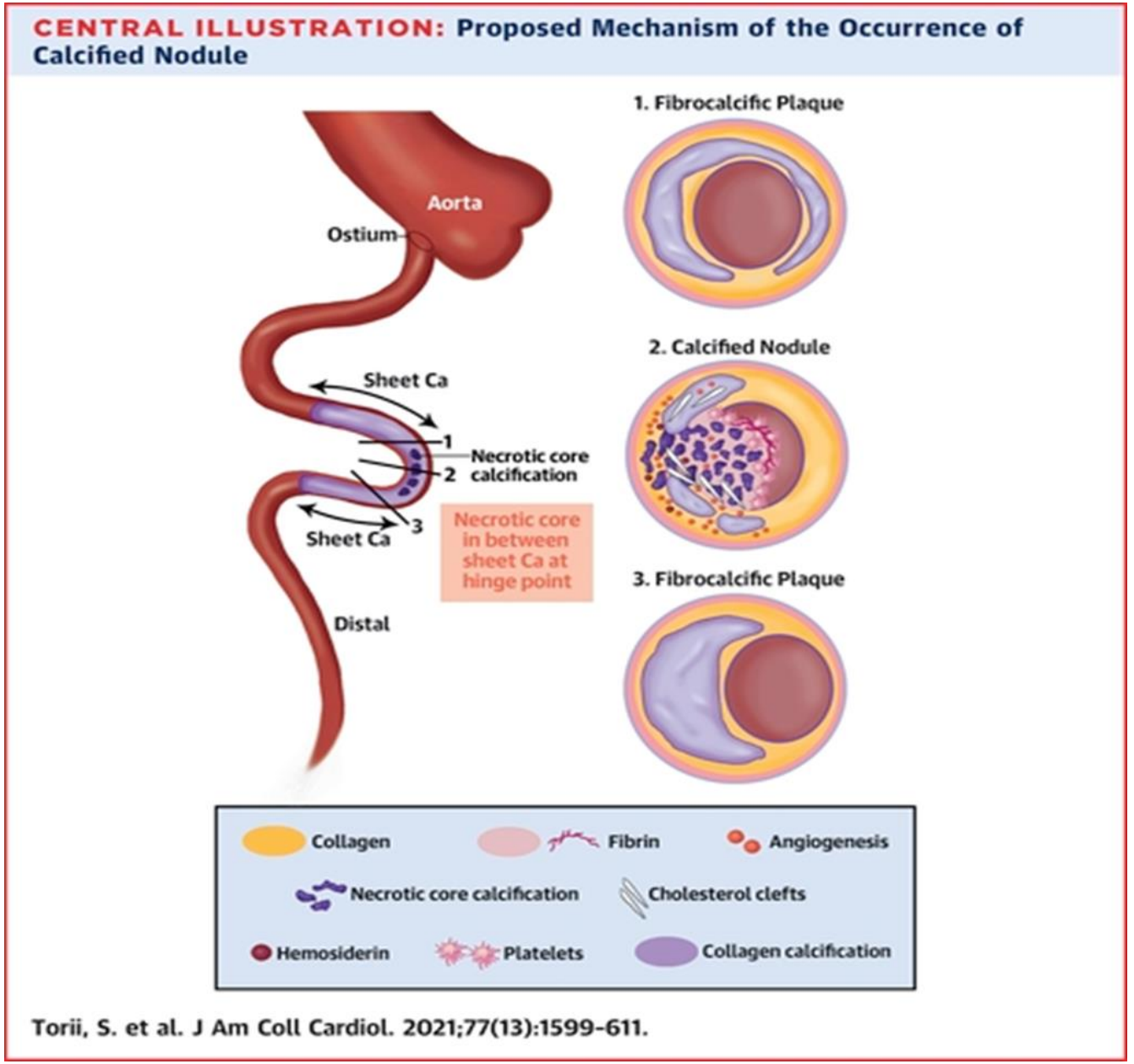


FIGURE 1. Pathological characteristics of plaque rupture and plaque erosion. Ruptured plaque (left image) is featured with a larger lipid core containing abundant macrophages and T cells.

Red thrombus was observed in the small lumen. Eroded plaque (right image) has a large lumen with white thrombus and fibrous plaque tissue characterized by little or no lipid deposition.

In particularly, there is discontinuous endothelial cell layer in eroded plaque. Neutrophil extracellular traps was found at the junction of plaque tissue and thrombus in both eroded and ruptured plaque.



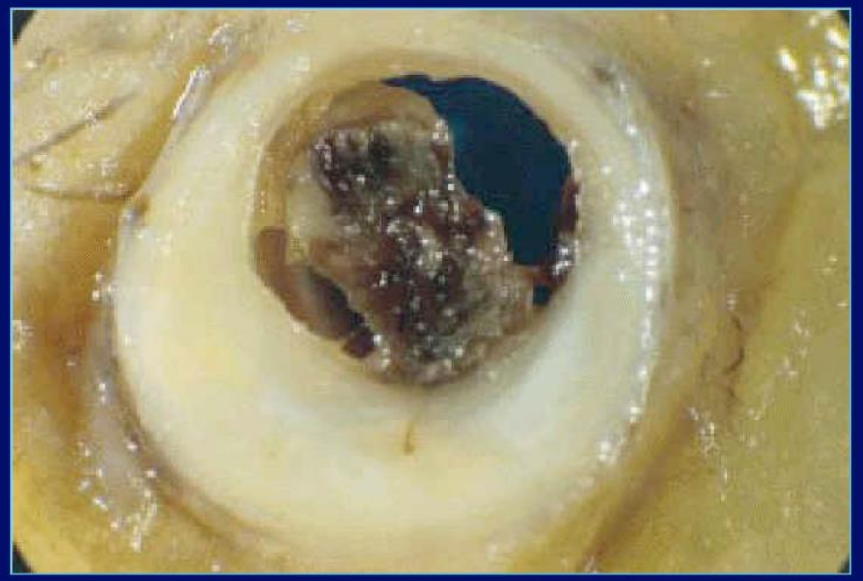
Sho Torii et al. *JACC* 2021; 77:1599-1611.

JACC JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY

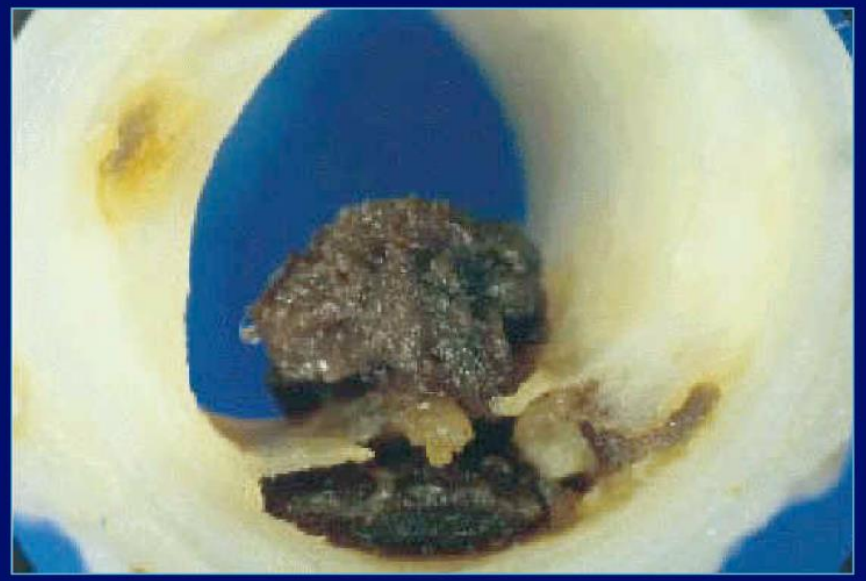
Endotēlija erozija vs pangas ruptūra

- Thrombi result from endothelial erosion or plaque rupture[Davies 2000:C,F]

Endothelial erosion and intact plaque[Davies 2000:D]



Plaque rupture[Davies 2000:E]



Davies MJ. *Heart*. 2000;83:361-366.

ESC European Society of Cardiology
European Heart Journal (2019) 00, 1–71
doi:10.1093/eurheartj/ehz425

ESC GUIDELINES

2019 ESC Guidelines for the diagnosis and management of chronic coronary syndromes

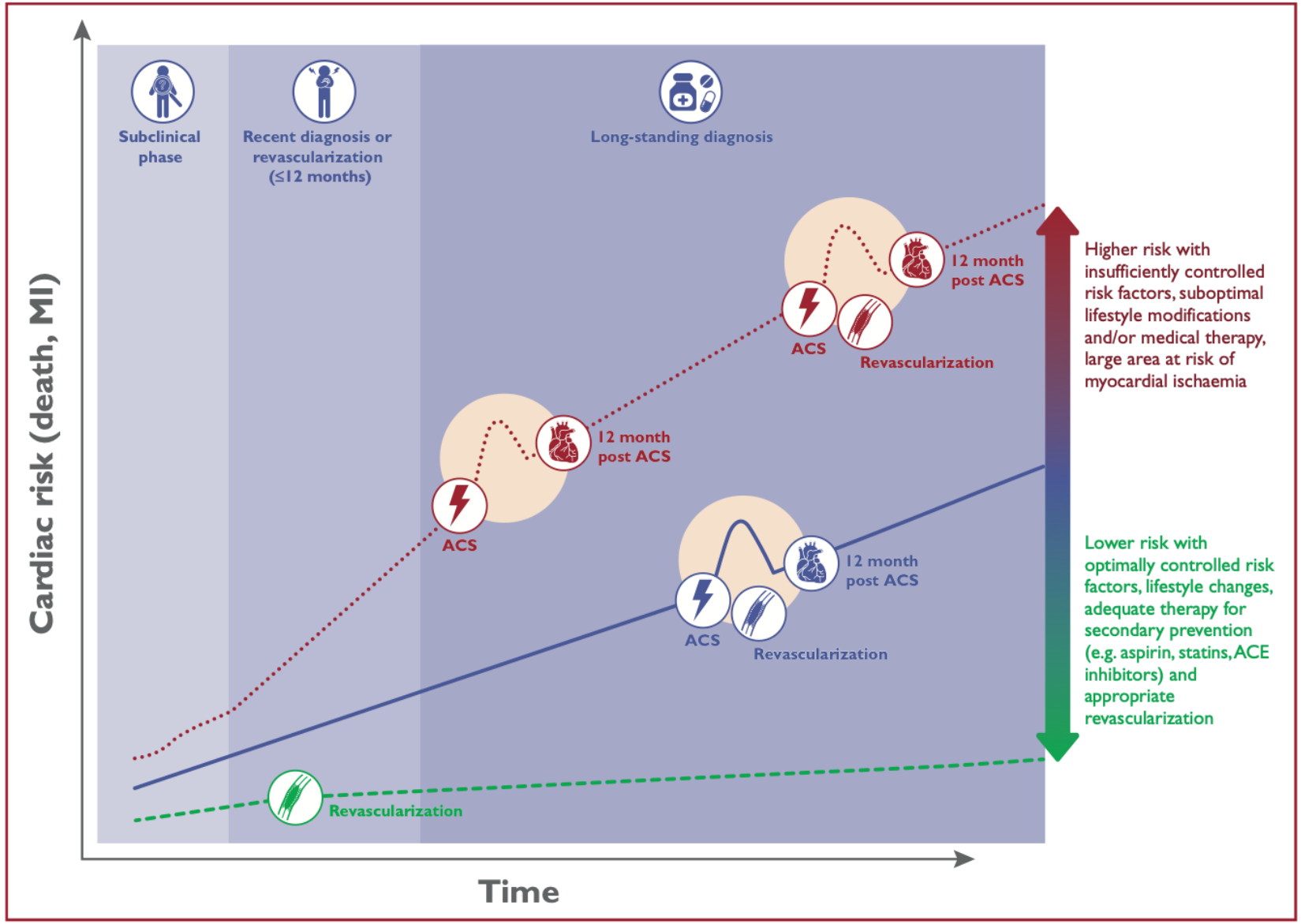
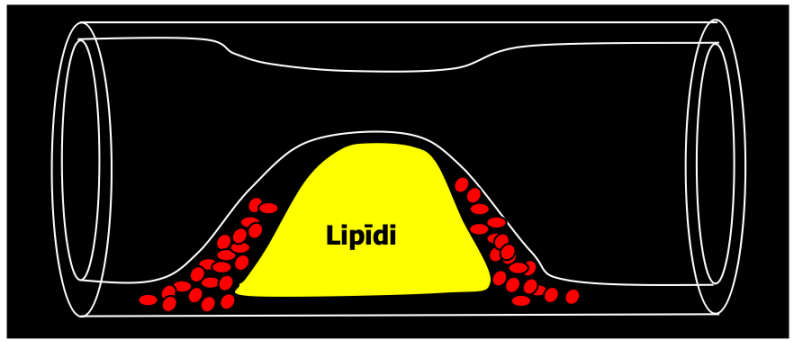
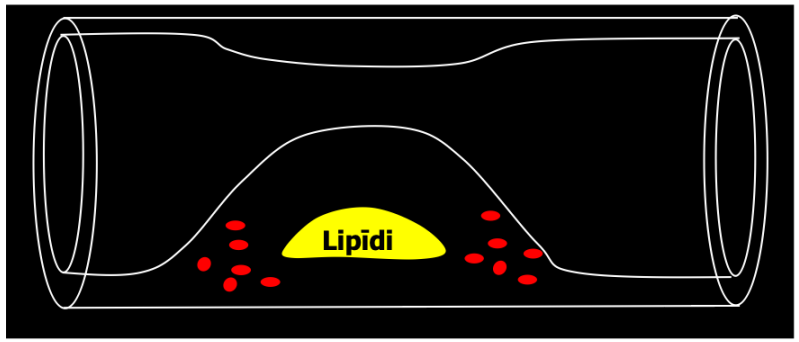


Figure 1 Schematic illustration of the natural history of chronic coronary syndromes. ACE = angiotensin-converting enzyme; ACS = acute coronary syndromes; CCS = chronic coronary syndromes; MI = myocardial infarction.

Kāpēc pangas rupturē?



Nestabila panga



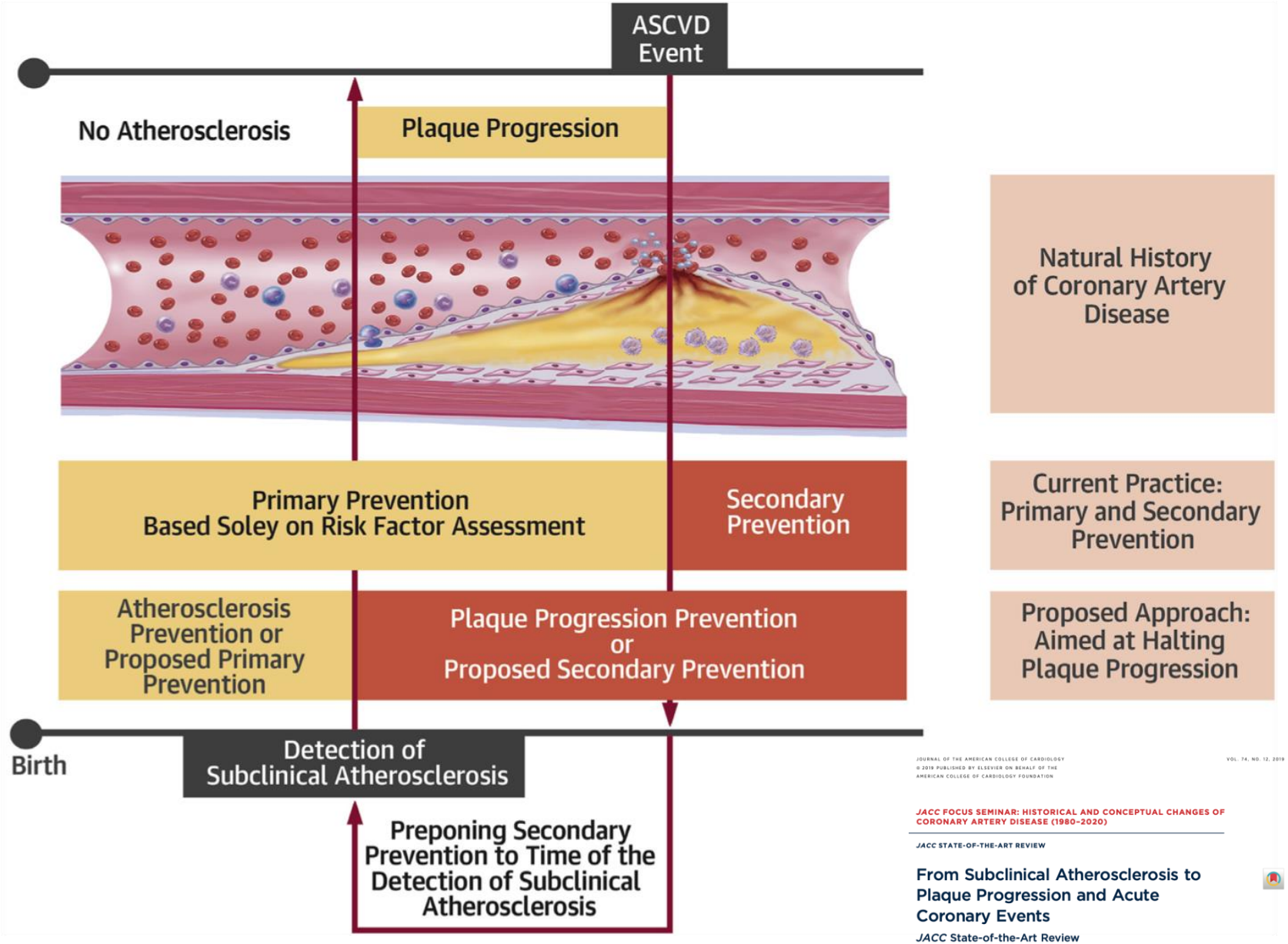
Stabila panga

+++	Lipīdi	+
plāna < 150 μm	Fibrozā kapsula	bieza
+++	Makrofāgi, T-limfocīti	+
+	Gludās muskuļu šūnas	+++
+++	Vasa vasorum adventīcijā	+
rupturējusi	Iekšējā elastiskā membrāna	neskarta

+ biomehāniskie faktori

Davies MJ et al. Circulation 1996;94:2013-20.

CENTRAL ILLUSTRATION **Prevention Based on Detection of Subclinical Atherosclerosis Should Result in Reduced Coronary Events**



JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY VOL. 74, NO. 12, 2019

JACC FOCUS SEMINAR: HISTORICAL AND CONCEPTUAL CHANGES OF CORONARY ARTERY DISEASE (1980–2020)

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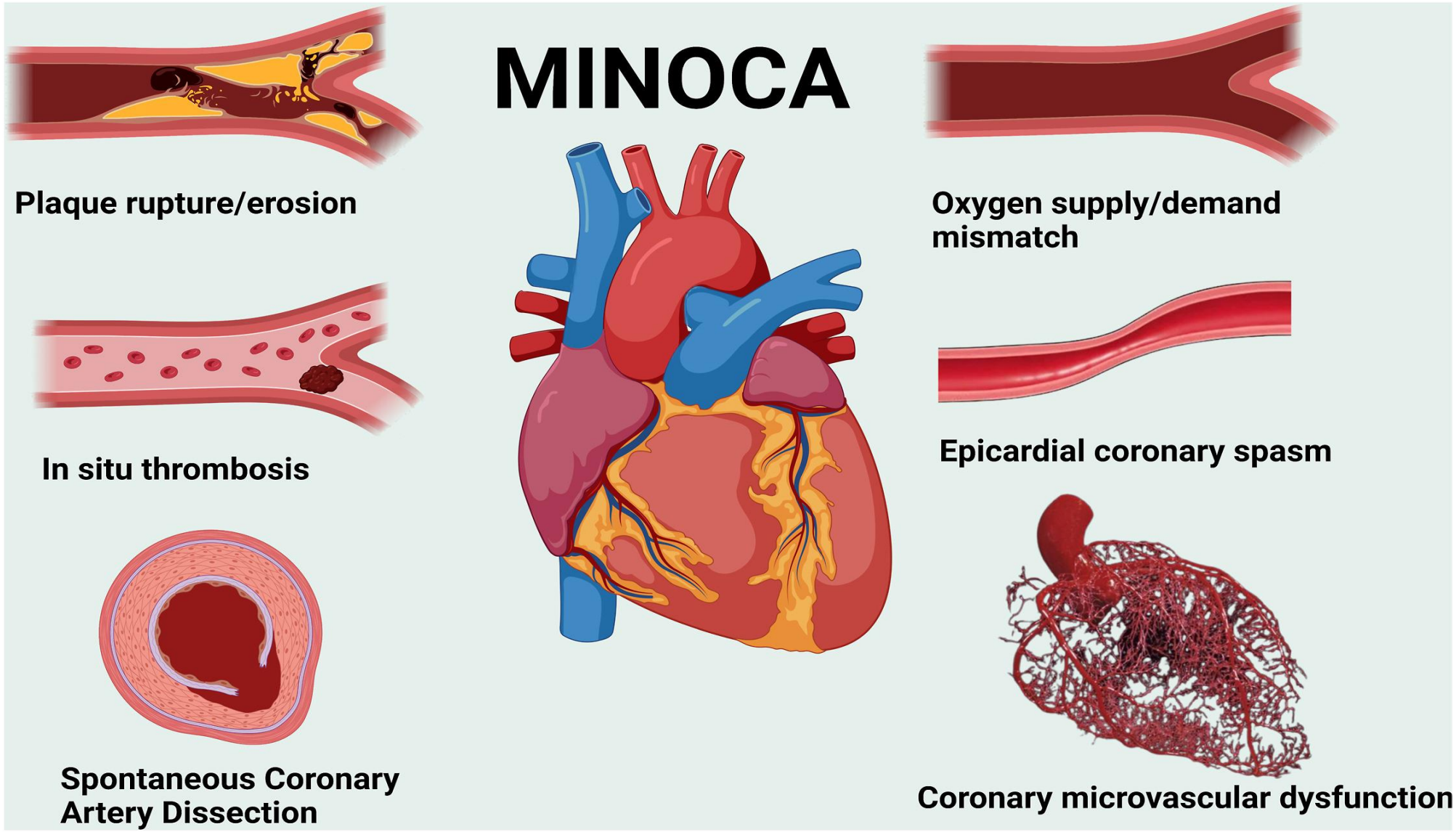
From Subclinical Atherosclerosis to Plaque Progression and Acute Coronary Events

JACC State-of-the-Art Review

Amir Ahmadi, MD,[a,b] Edgar Argulian, MD,[a] Jonathon Leipsic, MD,[b] David E. Newby, MD,[c] Jagat Narula, MD, PhD[a]

Ahmadi, A. et al. J Am Coll Cardiol. 2019;74(12):1608–17.

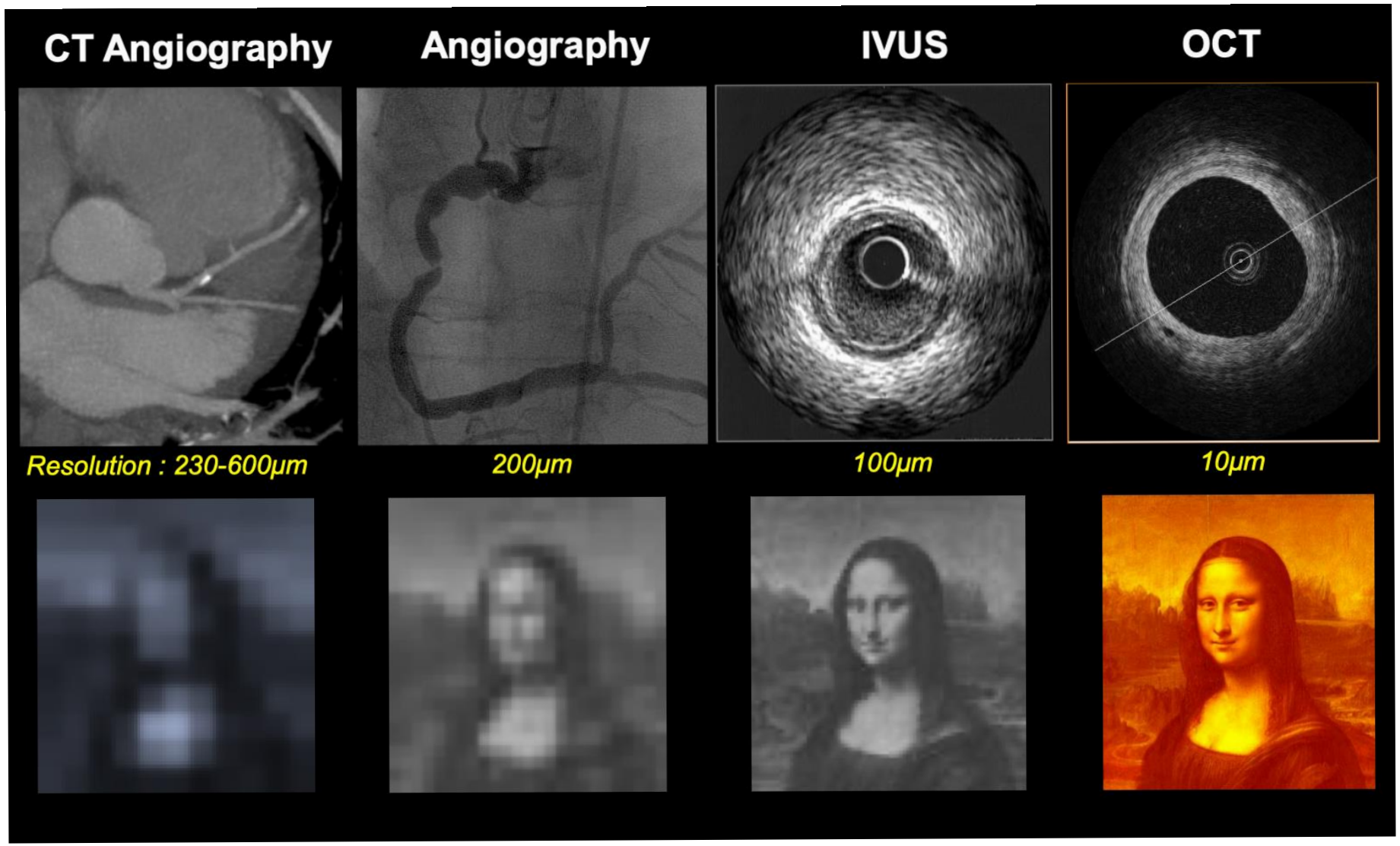
Miokarda infarkts bez koronārās artērijas obstrukcijas



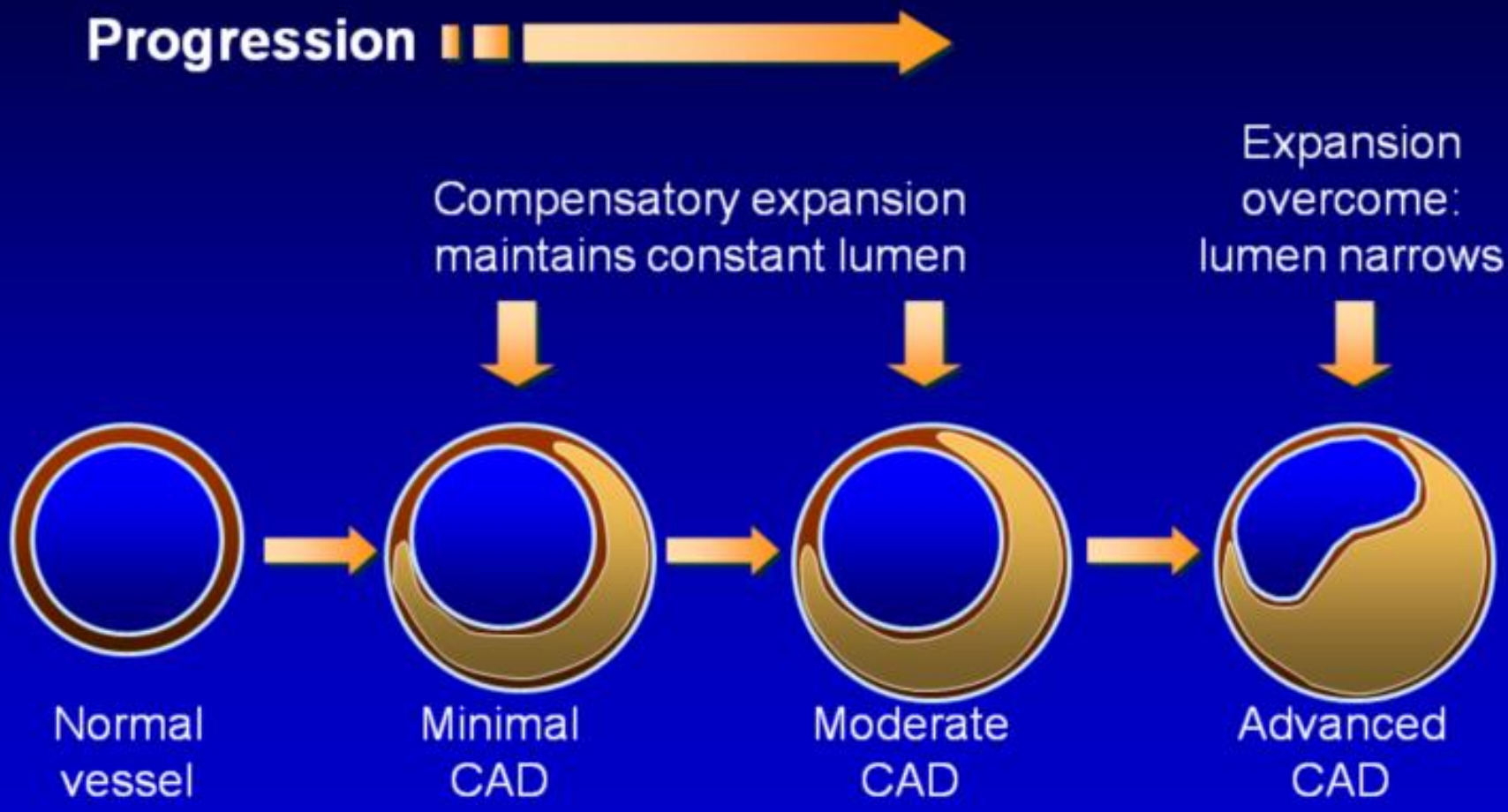
Boivin-Proulx et al.
Pathophysiology of MINOCA

CJC Open 6 (2024) 380–390

Koronāro artēriju attēldiagnostikas izšķirtspēja

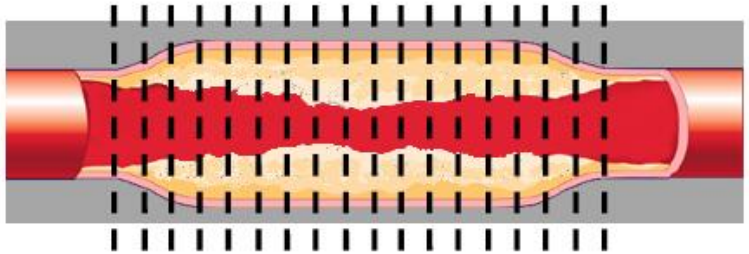


Glagov's Remodelling Hypothesis

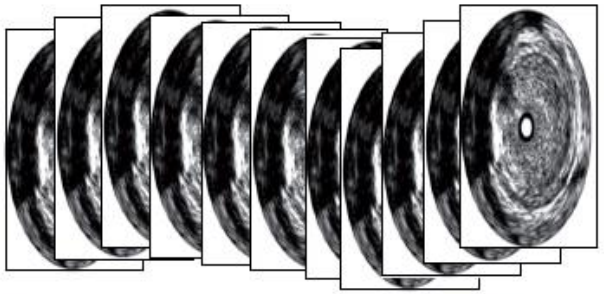


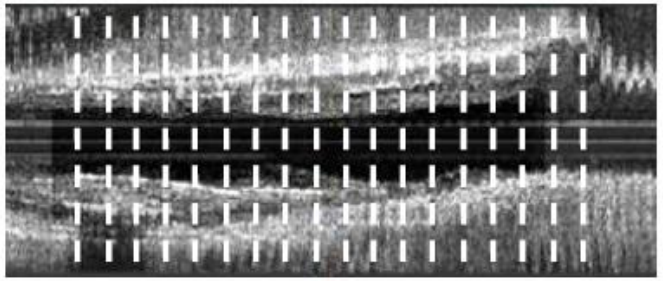
Glagov S, et al. *N Engl J Med*. (1987) 316:1371-1375.

Intravaskulārās ultraskaņas metode



$S_0\ S_1\ S_2\ S_3 - S_{n-1}$





$$\text{Tilpums} = \sum_{i=0}^{n} S_i = S_0 + S_1 + \cdots\cdots + S_{n-1} + S_n$$

Tilpuma indekss = tilpums / garums (mm^3/mm)

Tilpuma aprēķini:

EEM, lūmens, stents, neointīma

European Heart Journal (2016) 37, 524–535
doi:10.1093/eurheartj/ehv642

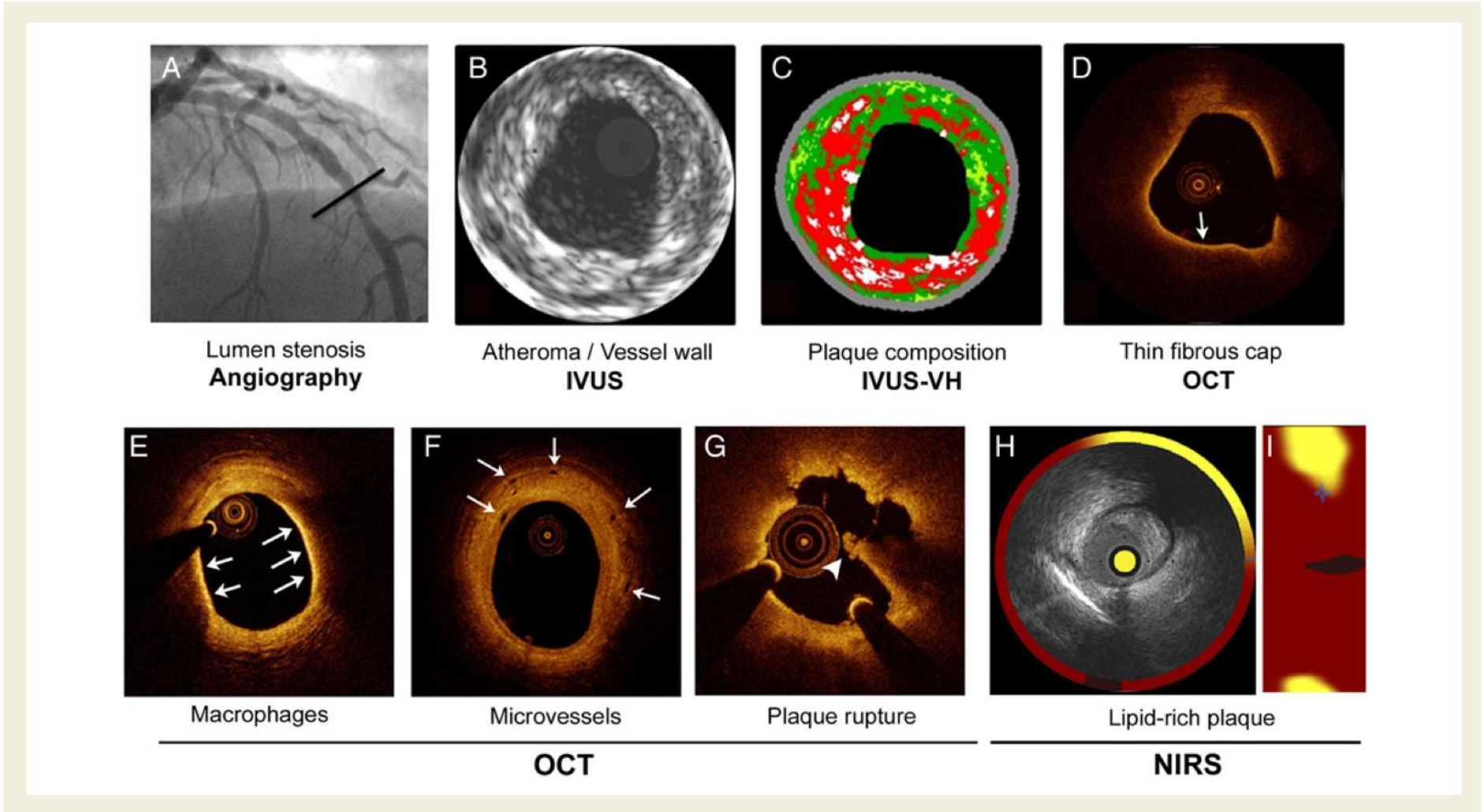
REVIEW

Imaging

Intracoronary imaging of coronary atherosclerosis: validation for diagnosis, prognosis and treatment

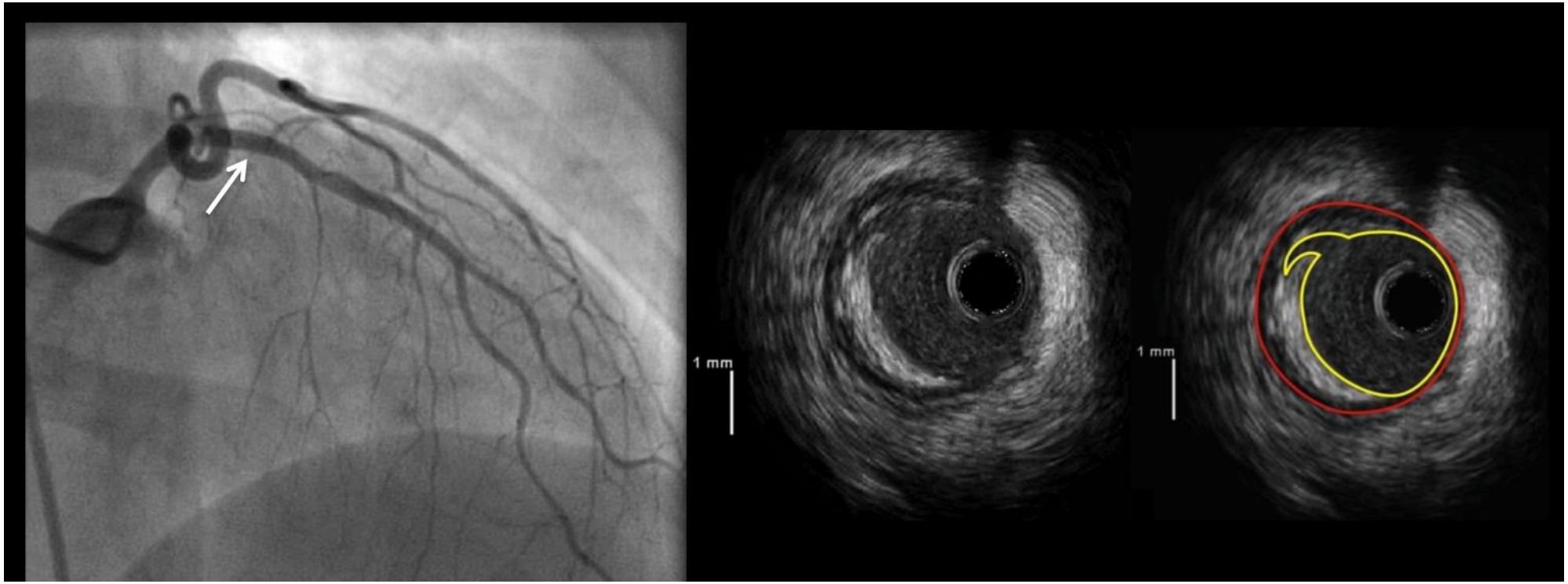
Konstantinos C. Koskinas[1], Giovanni J. Ughi[2], Stephan Windecker[1], Guillermo J. Tearney[3,4], and Lorenz Räber[1]*

Aterosklerozes attēldiagnostika



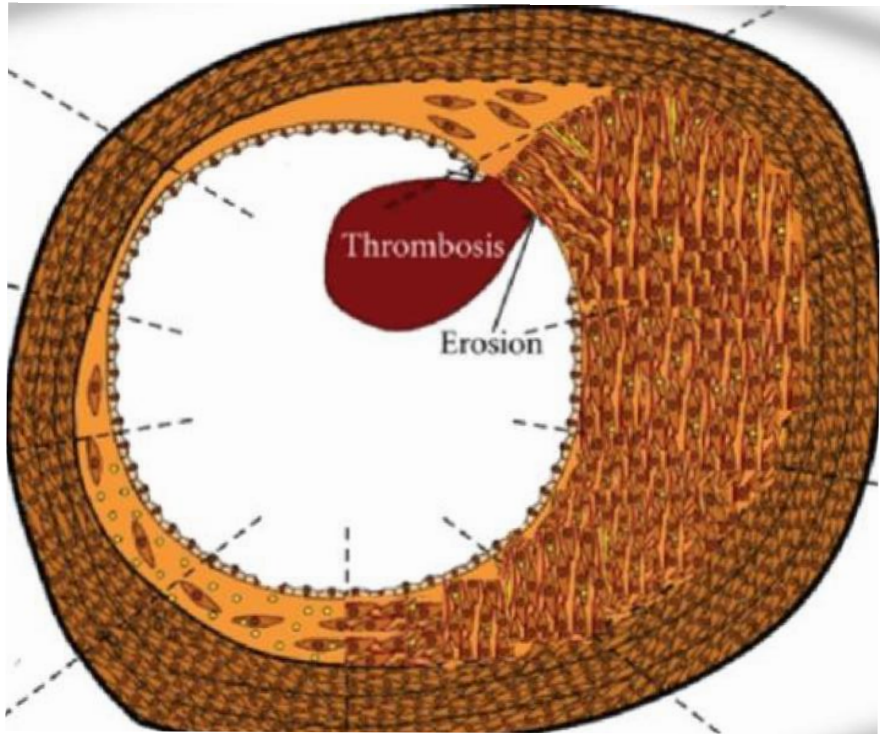
Aterosklerotiskās pangas ruptūra bez asinsvada lumena obstrukcijas

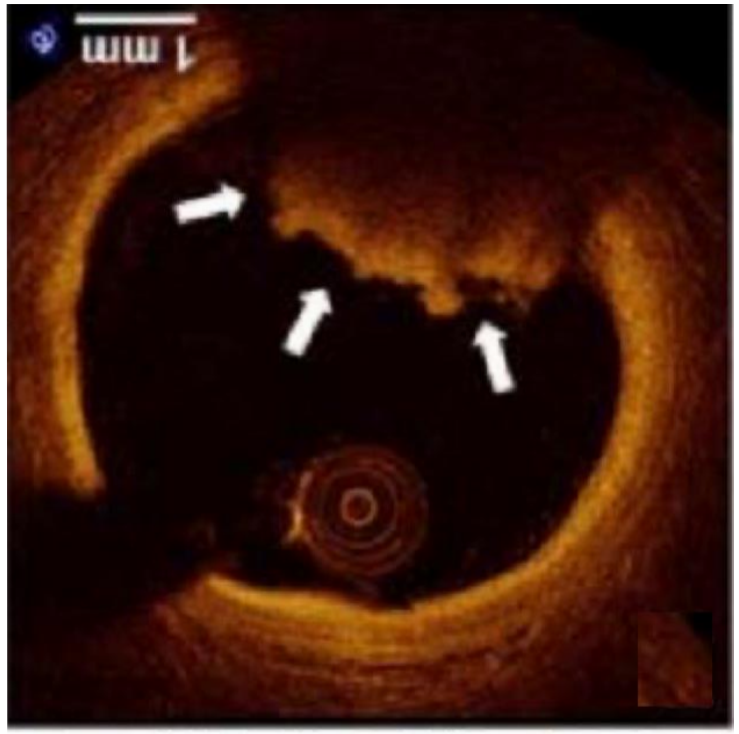
Intravaskulārās ultraskaņas izmeklējums



Aterosklerotiskās pangas erozija ar piesienas trombozi

Optiskās koherences tomogrāfijas izmeklējums





Akūta koronāra sindroma vadlīnijas

ESC European Society of Cardiology
European Heart Journal (2023) **00**, 1–107
https://doi.org/10.1093/eurheartj/ehad191

ESC GUIDELINES

2023 ESC Guidelines for the management of acute coronary syndromes

Developed by the task force on the management of acute coronary syndromes of the European Society of Cardiology (ESC)

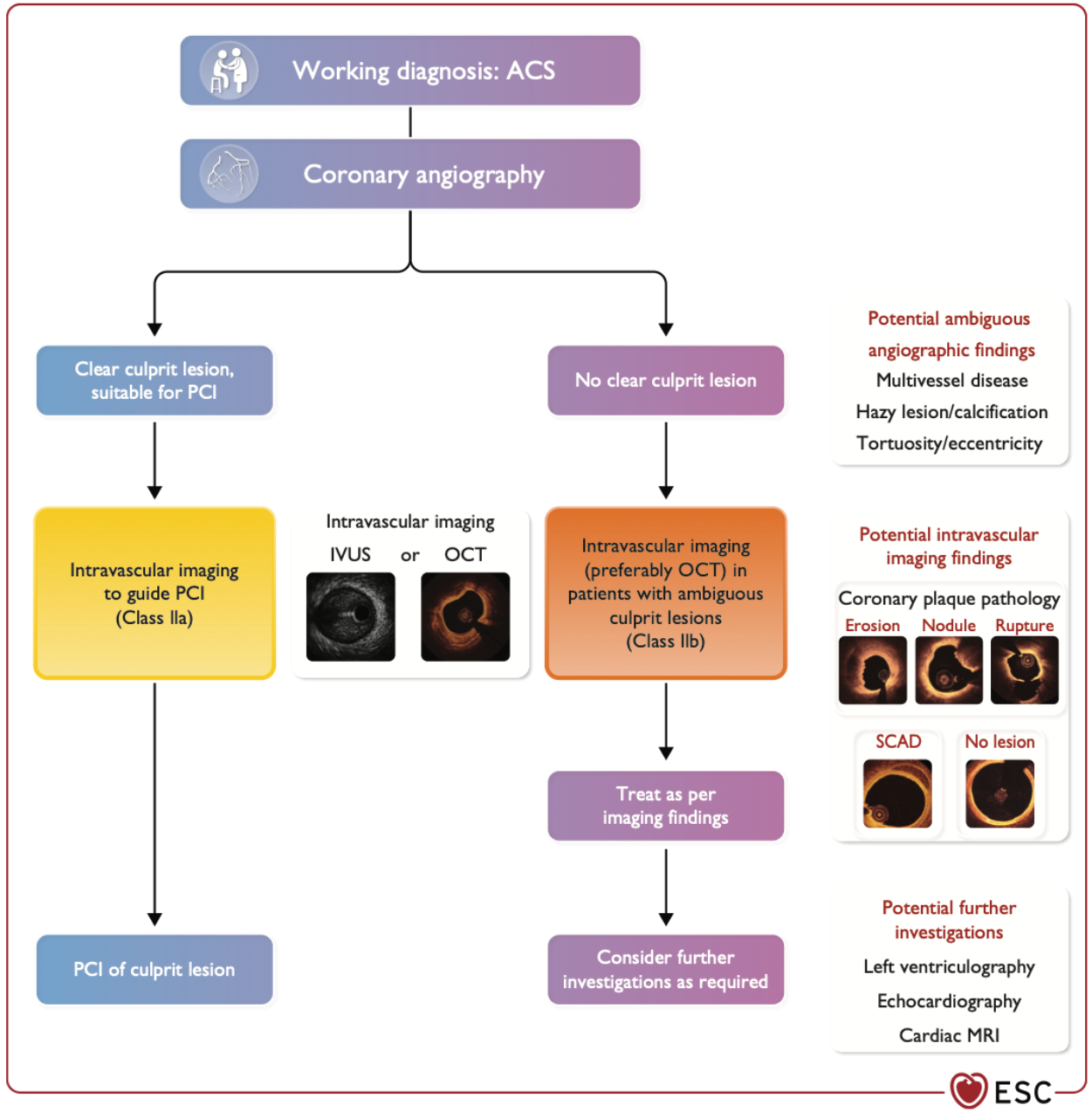


Figure 13 A practical algorithm to guide intravascular imaging in acute coronary syndrome patients. ACS, acute coronary syndrome; IVUS, intravascular ultrasound; MRI, magnetic resonance imaging; OCT, optical coherence tomography; PCI, percutaneous coronary intervention; SCAD, spontaneous coronary artery dissection.

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VOL. ■, NO. ■, 2025

CLINICAL PRACTICE GUIDELINE

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A Report of the American College of Cardiology/American Heart Association
Joint Committee on Clinical Practice Guidelines

Akūta koronāra sindroma vadlīnijas

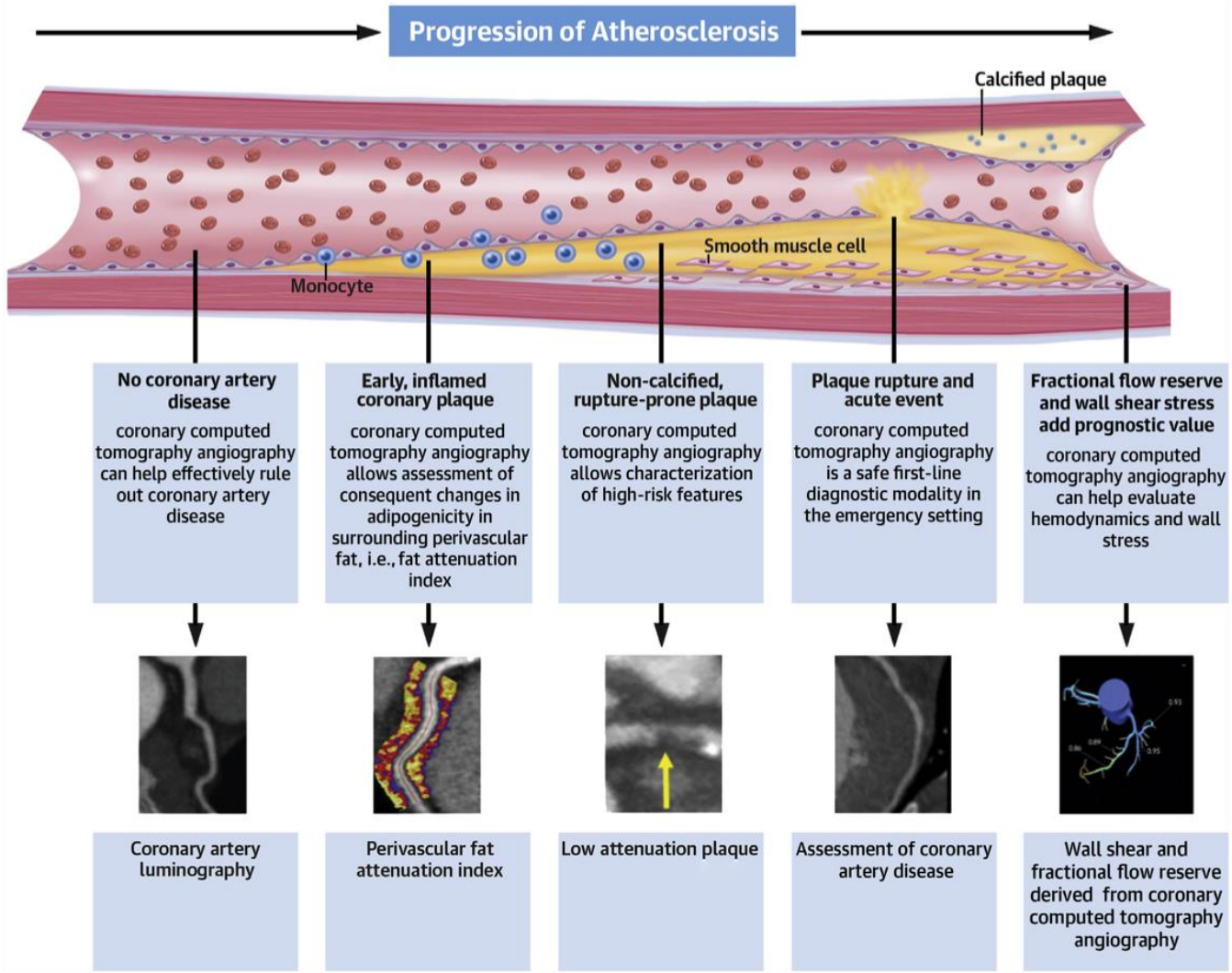
7.3. Use of Intracoronary Imaging

**Recommendation for Use of Intracoronary Imaging
Referenced studies that support recommendation are summarized in the Evidence Table.**

COR	LOE	RECOMMENDATION
1	A	1. In patients with ACS undergoing coronary stent implantation in left main artery or in complex lesions, intracoronary imaging with intravascular ultrasound (IVUS) or optical coherence tomography (OCT) is recommended for procedural guidance to reduce ischemic events.*[1-11]

Attēldiagnostika dažādās ateroģenēzes stadijās

CENTRAL ILLUSTRATION Utility of Coronary Computed Tomography Angiography in Coronary Artery Disease



Abdelrahman, K.M. et al. J Am Coll Cardiol. 2020;76(10):1226–43.

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Akūta koronāra sindroma vadlīnijas

TABLE 4 Types of Acute Myocardial Infarction According to the Universal Definition of Myocardial Infarction

Type 1*	**Caused by acute coronary atherothrombosis, usually precipitated by atherosclerotic plaque disruption (rupture or erosion) and often associated with partial or complete vessel thrombosis.**
Type 2	Caused by an imbalance between myocardial oxygen supply and demand unrelated to acute coronary atherothrombosis.
Type 3	Cardiac death, with symptoms of myocardial ischemia and presumed ischemic electrocardiographic changes or ventricular arrythmia, before blood samples for cardiac biomarkers can be obtained or increases in cardiac biomarkers can be identified and/or in whom MI is identified by autopsy.
Type 4	4a: Peri-PCI MI caused by a procedural complication and detected ≤48 h after PCI. 4b: Post-PCI MI caused by coronary stent or stent scaffold thrombosis. 4c: Post-PCI MI caused by coronary stent restenosis.
Type 5	Peri-CABG MI caused by a procedural complication detected ≤48 h after CABG surgery.

Adapted with permission from Thygesen et al.[4] Copyright 2018 The European Society of Cardiology, American College of Cardiology Foundation, American Heart Association, Inc., and the World Heart Federation.

*This guideline focuses on the management of type 1 AMI. The diagnostic evaluation of chest pain and the management of type 2 MI, SCAD, and MINOCA are covered in separate documents.[5-8]

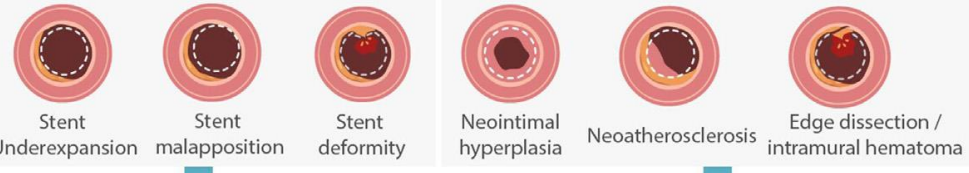
AMI indicates acute myocardial infarction; CABG, coronary artery bypass grafting; MI, myocardial infarction; MINOCA, MI with nonobstructive coronary artery disease; PCI, percutaneous coronary intervention; and SCAD, spontaneous coronary artery dissection.

SCAI Expert Consensus Statement on Management of In-Stent Restenosis and Stent Thrombosis

STENT THROMBOSIS

Assessment: Intravascular Imaging

Mechanism of stent failure



- Balloon angioplasty (NC Balloon)
- Cutting/scoring balloons
- Laser angioplasty
- Lithotripsy
- Thrombectomy
- Pharmacotherapy

- Balloon angioplasty (NC Balloon)
- Cutting/scoring balloons

Assess for further intervention with coronary stenting with drug eluting stent

IN-STENT RESTENOSIS

Assessment: Intravascular Imaging

Mechanism

- Predilation
- NC Balloon
- Cutting/scoring balloons

Unsuccessful

- Very high pressure balloon
- Laser angioplasty
- Lithotripsy
- Rotational atherectomy
- Vascular brachytherapy (consider for previous ISR therapy or multiple layers of stent)

Successful

- PCI DES
- Diffuse ISR
- Suboptimal gain

European Heart Journal (2016) 37, 524–535
doi:10.1093/eurheartj/ehv642

REVIEW

Imaging

Intracoronary imaging of coronary atherosclerosis: validation for diagnosis, prognosis and treatment

Konstantinos C. Koskinas[1], Giovanni J. Ughi[2], Stephan Windecker[1], Guillermo J. Tearney[3,4], and Lorenz Räber[1]*

Aterosklerozes intrakoronārā attēldiagnostika: terapijas efekts

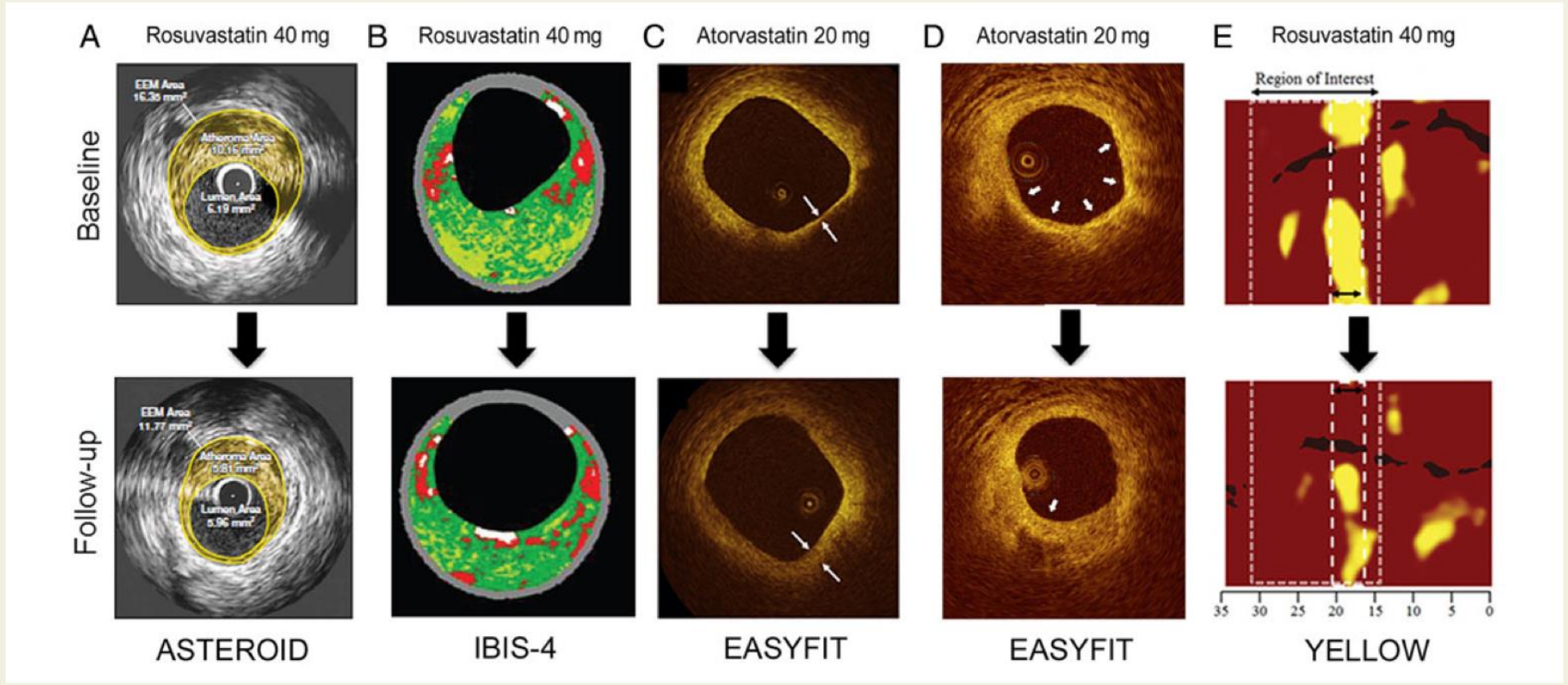


Figure 3 Representative examples of serial atheroma regression by grayscale intravascular ultrasound in the ASTEROID trial[39] (*A*); no change of necrotic core by intravascular ultrasound-virtual histology (*B*) in the IBIS-4 study[40]; increase of fibrous cap thickness (*C*) and macrophage signal decrease by optical coherence tomography (*D*) in the EASY-FIT study[48]; and reduction of lipid-core burden index by near-infrared spectroscopy (*E*) in the YELLOW trial[49] associated with statin treatment.

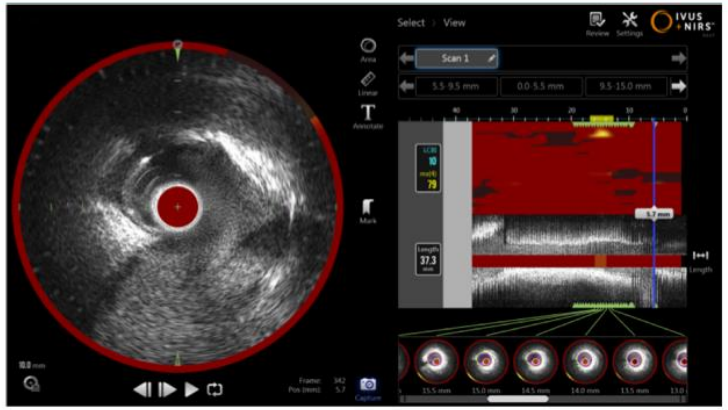
Tuvā infrasarkanā spektroskopija - NIRS

- Tuvā infrasarkanā spektroskopija (ang.val. *near infrared spectroscopy,* NIRS)
- Intravaskulāra sistēma, kas apvieno:
  - **NIRS** (tuvo infrasarkano staru spektroskopiju)
  - **IVUS** (intrvaskaulāro ultraskaņu)

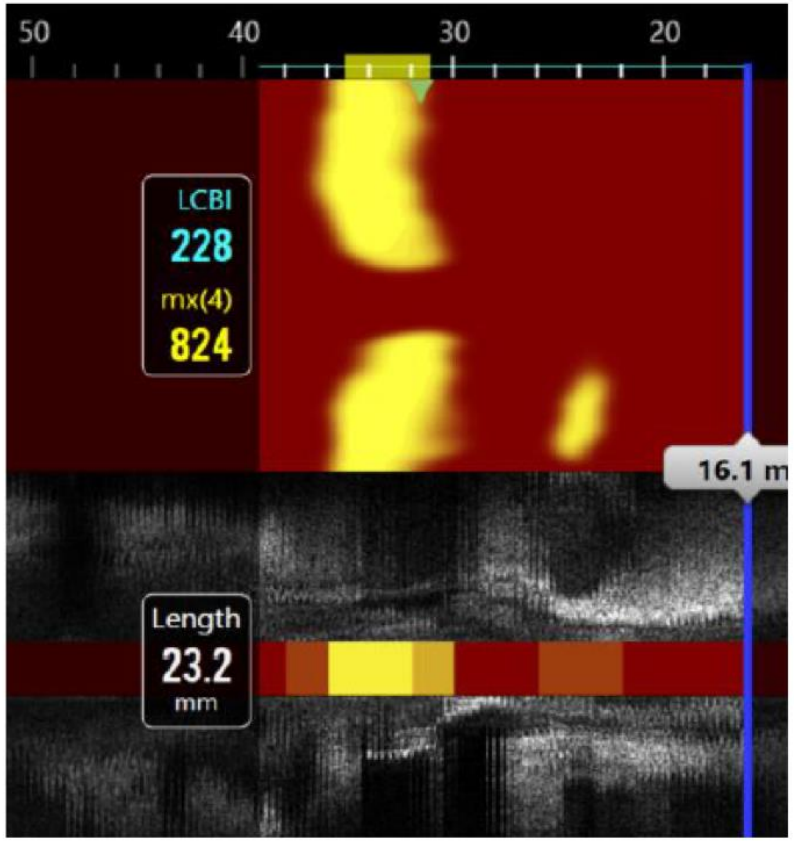
- Nosaka lipīdu kodolu koronāro artēriju pangās
- Lipīdiem bagātas pangas asociējas ar akūtu koronāru sindromu

Waxman S. et al. In vivo validation of a catheter-based near-infrared spectroscopy system for detection of lipid core coronary plaques: initial results of the SPECTACL study. *JACC Cardiovascular imaging*. 2009.2(7):

NIRS



- Hemogramma
  - Identificē LRP
    - Nav LRP
    - Ir LRP
- Lipid Core Burden Index (LCBI) ir kvantitatīvs mērījums no skenētā reģiona
  - LCBI ir no 0 – 1000
  - LCBI = dzeltenie pikseļi/visu pikseļu skaitu x 1000
- $maxLCBI_{4mm}$ = maksimālais LCBI četros milimetros noteiktā zonā



OCT-NIRS Imaging for Detection of Coronary Plaque Structure and Vulnerability

James Muller[1]* *and Ryan Madder*[2]

[1] *Brigham and Women's Hospital, Harvard Medical School, Boston, MA, United States,* [2] *Spectrum Health, Grand Rapids, MI, United States*

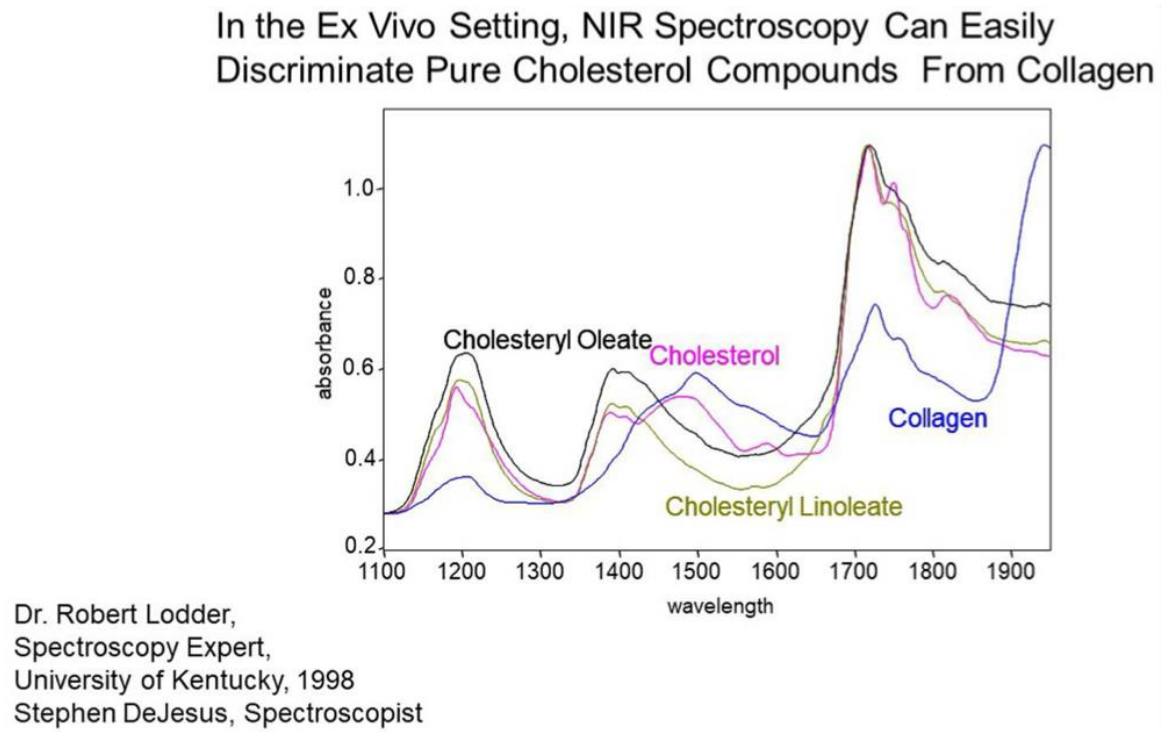
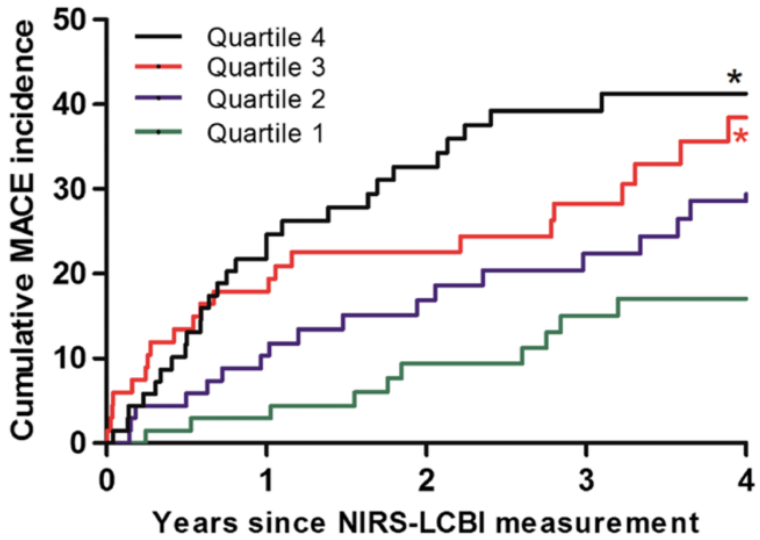


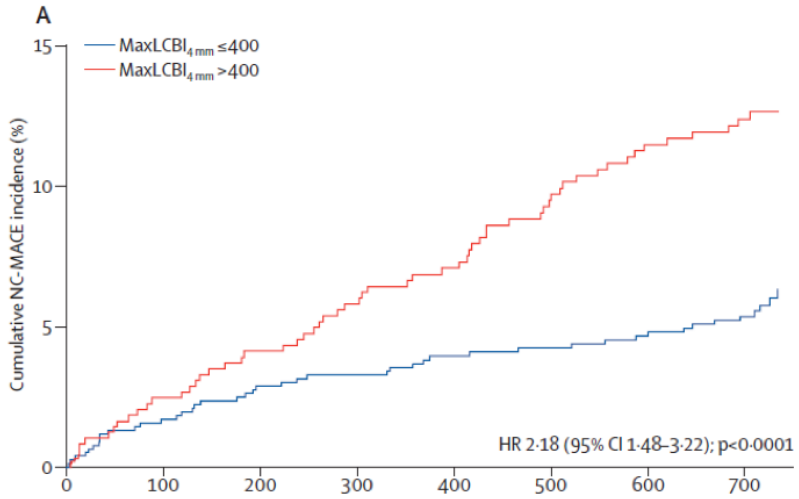
FIGURE 4 | Measurement of pure chemicals by spectroscopy in the absence of blood flow and motion. Substances of interest, such as collagen and cholesterol, are easily identified by their variable absorbance at different near-IR wavelengths.

LCBI pētījumos



No. at risk					
LCBI Q1(<83.0)	68	66	52	43	29
LCBI Q2 (≥83.0-227.0)	68	61	47	40	31
LCBI Q3 (≥227.0-360.0)	67	55	42	35	22
LCBI Q4 (≥360.0)	70	53	41	30	25

Schuurman A-S, et al. Near-infrared spectroscopy-derived lipid core burden index predicts adverse cardiovascular outcome in patients with coronary artery disease during long-term follow-up. European heart journal. 2017;39(4):295-302.



Number at risk								
MaxLCBI4mm ≤400	778	748	733	728	689	682	676	664
MaxLCBI4mm >400	493	473	457	448	426	409	399	392

Waksman R, et al. Identification of patients and plaques vulnerable to future coronary events with near-infrared spectroscopy intravascular ultrasound imaging: a prospective, cohort study. Lancet (London, England). 2019;394(10209):1629-37.

Cardiology

CAD and AMI

Cardiology
DOI: 10.1159/000543463

Received: July 14, 2024
Accepted: December 28, 2024
Published online: January 17, 2025

Role of Bailout Gene-Silencing Therapy in Plaque Lipid Reduction: Intravascular Imaging Study

Karlis Trusinskis[a, b] Baiba Kokina[b, c] Maris Lapsovs[a, b] Mairita Karantajere[b] Evija Kanasniece[b, d] Laima Caunite[b, c] Sanda Jegere[a, b] Inga Narbute[b] Dace Sondore[b] Alona Grave[b] Indulis Kumsars[a, b] Andrejs Erglis[a, b]

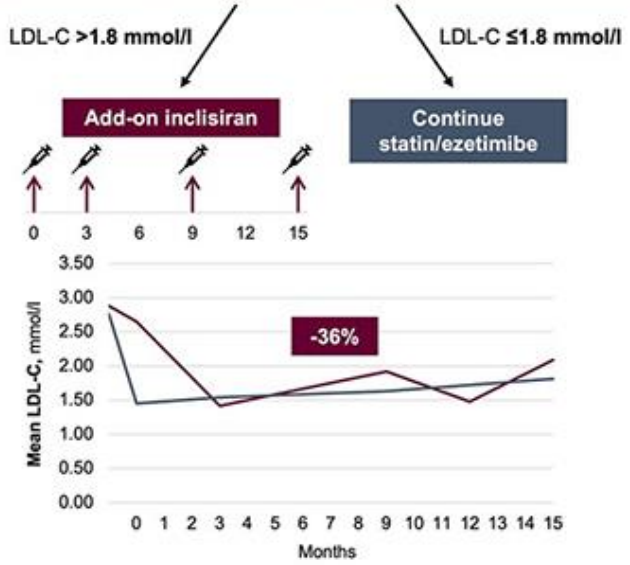
[a]Faculty of Medicine and Life Sciences, University of Latvia, Riga, Latvia; [b]Latvian Centre of Cardiology, Pauls Stradins Clinical University Hospital, Riga, Latvia; [c]Department of Residency, Riga Stradins University, Riga, Latvia; [d]Department of Internal Diseases, Riga Stradins University, Riga, Latvia

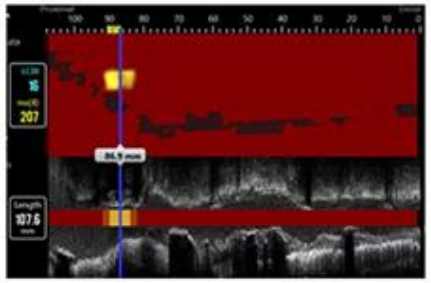
Cardiology. Published online January 17, 2025. doi:10.1159/

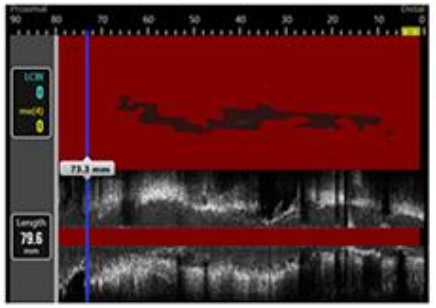
Cardiology

10.1159/000543463

- Stable CAD patients having a nonobstructive atherosclerotic plaque 20-50% in the proximal/middle third of a coronary artery → NIRS of the segment of interest
- Maximum tolerated dose of statin/ezetimibe therapy for 4-6 weeks



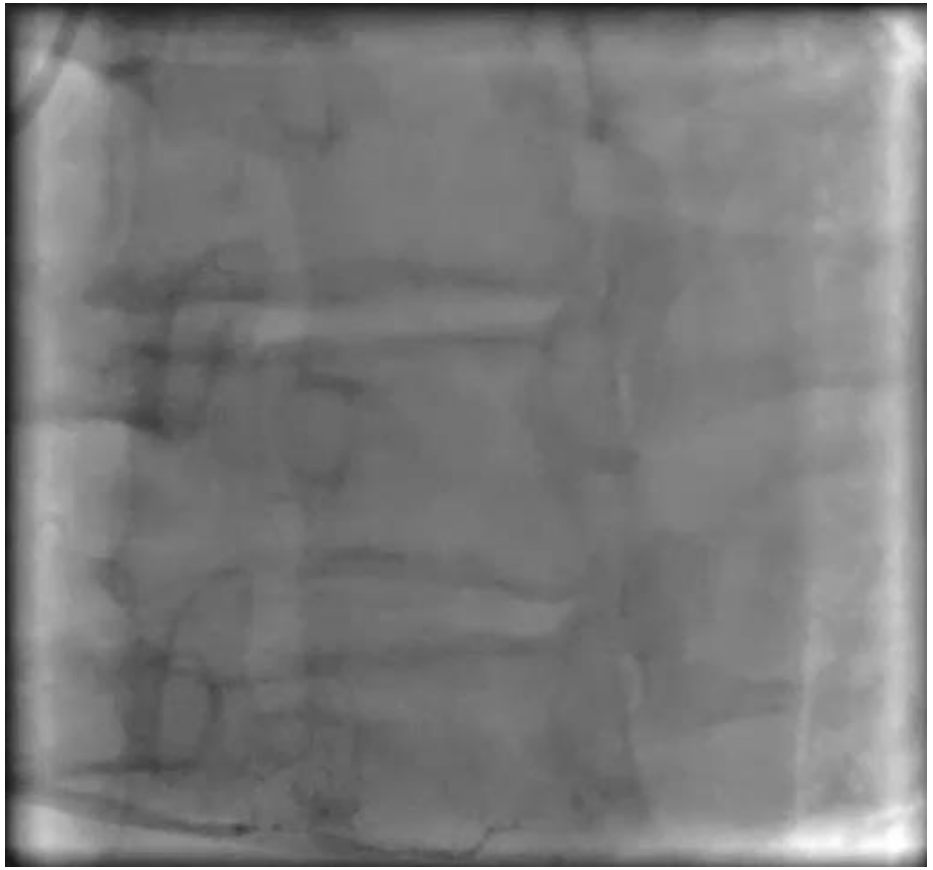


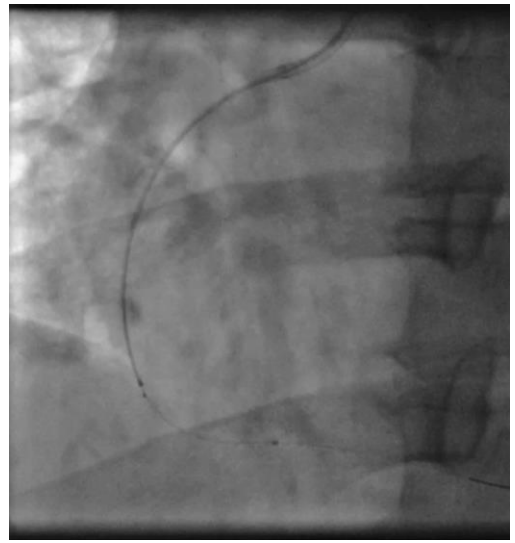


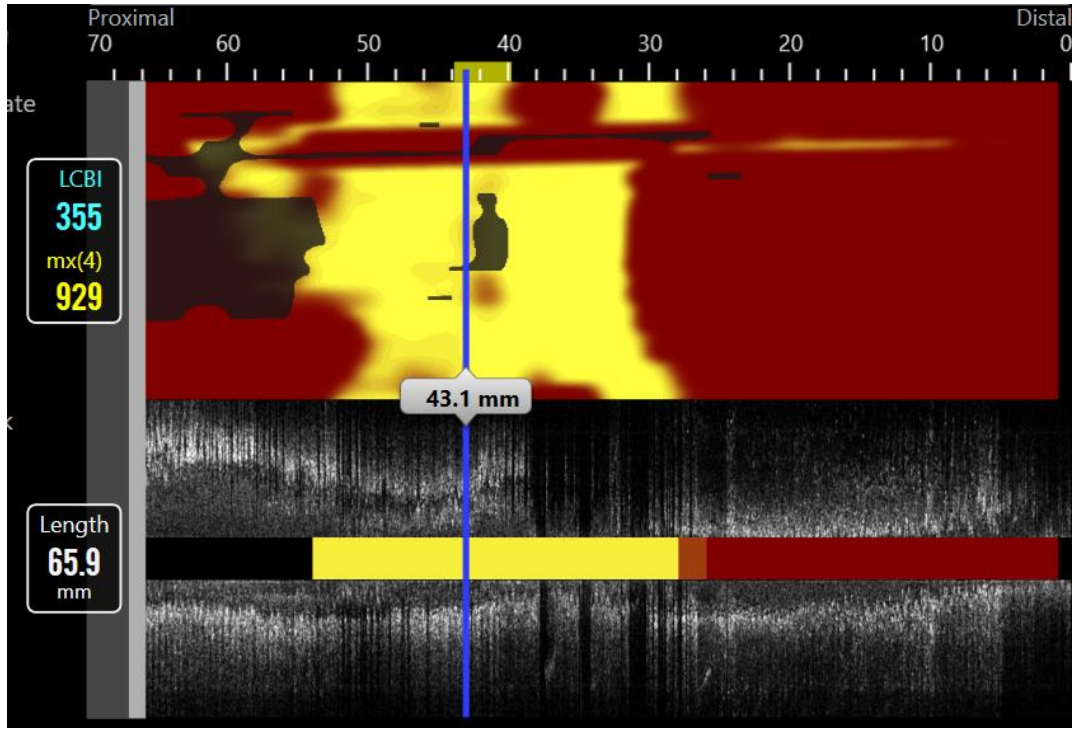
- **Add-on inclisiran** resulted in a **signficant LDL-C decrease of 26.42% (*p*=0.006)** after 15 months of therapy
- Intravascular imaging results demonstrated a **significant reduction in maxLCBI4 mm in the inclisiran (*p*=0.004) and statin/ezetimibe groups (*p*=0.004)** when comparing baseline and the 15-month follow-up results, with no statistically significant difference between the groups (*p*=0.213)

Tuvo infrasarkano staru spektrometrija akūta koronāra sindroma gadījumā

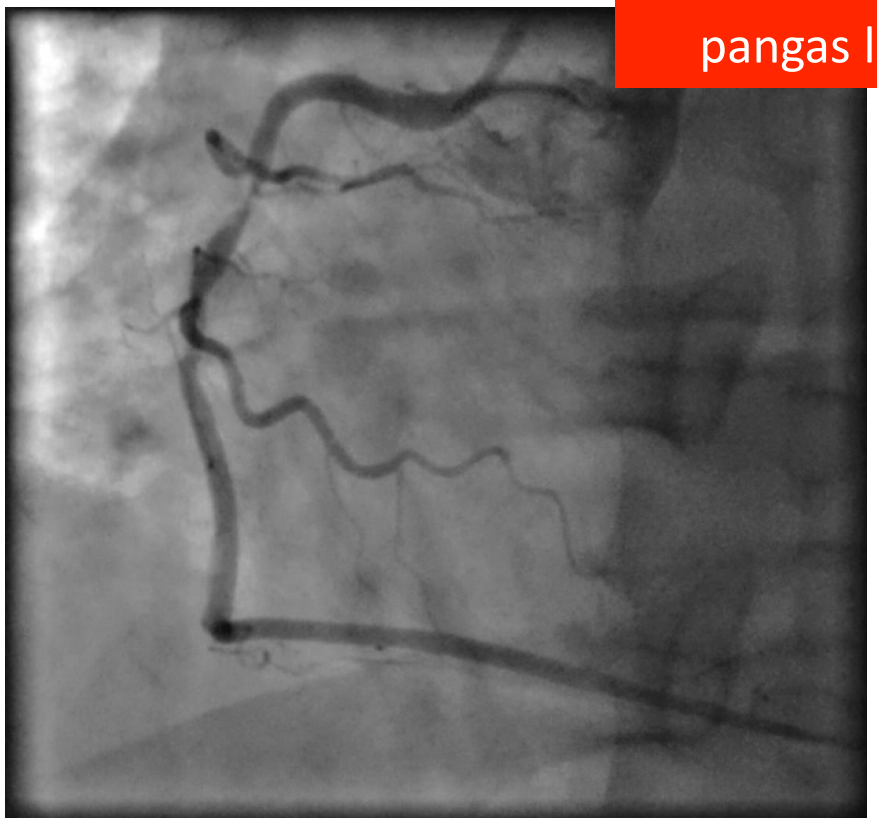
- 49 gadi, vīrietis
- Koronārā sirds slimība
- Akūts miokarda infarkts ar Q kreisā kambara apakšējā sienā, labā kambara infarkts (23.01.2024.)
- Perkutāna koronāra intervence RCA ar 2 bioabsorbējošiem stentiemS (23.01.2024.)

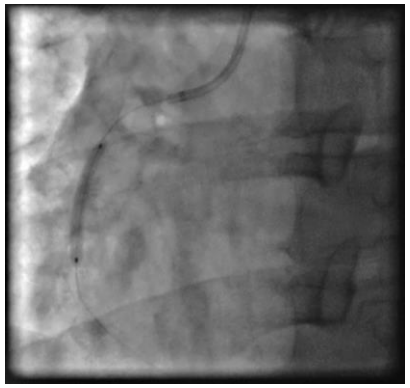


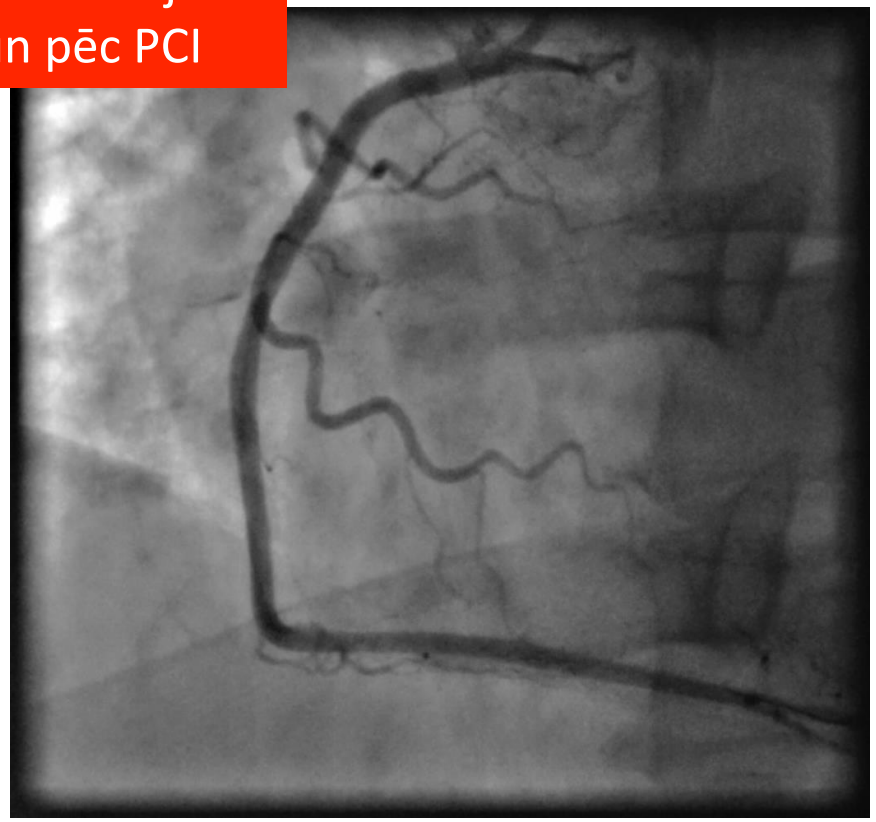


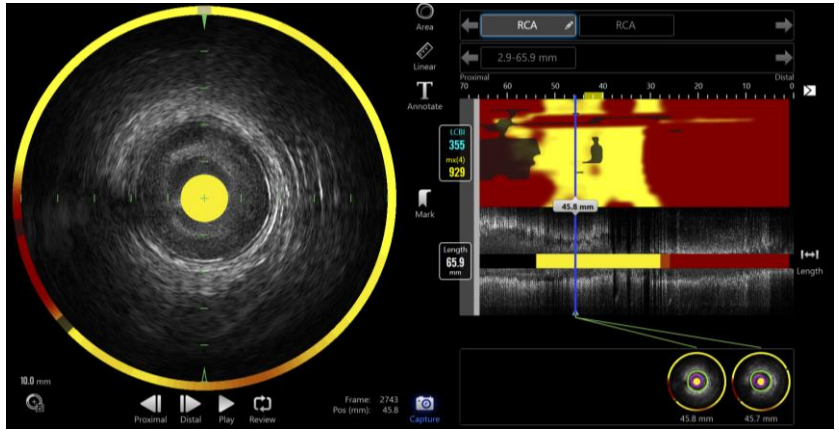


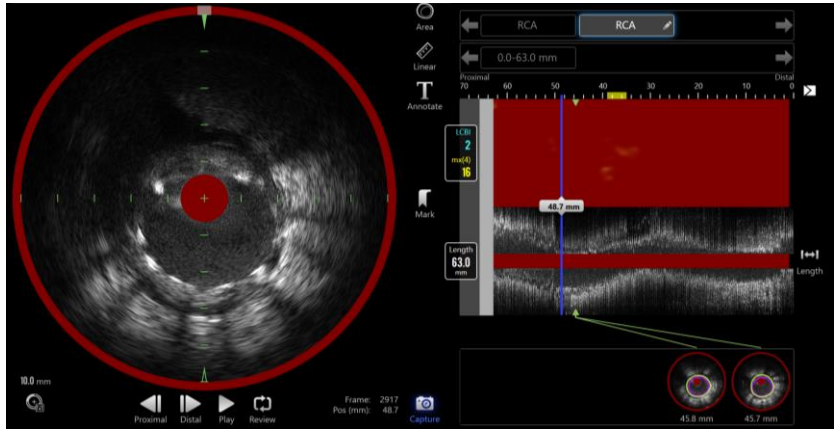
Tuvo infrasarkano staru spektrometrija: pangas lipīdu sastāvs pirms un pēc PCI











Preventive percutaneous coronary intervention versus optimal medical therapy alone for the treatment of vulnerable atherosclerotic coronary plaques (PREVENT): a multicentre, open-label, randomised controlled trial

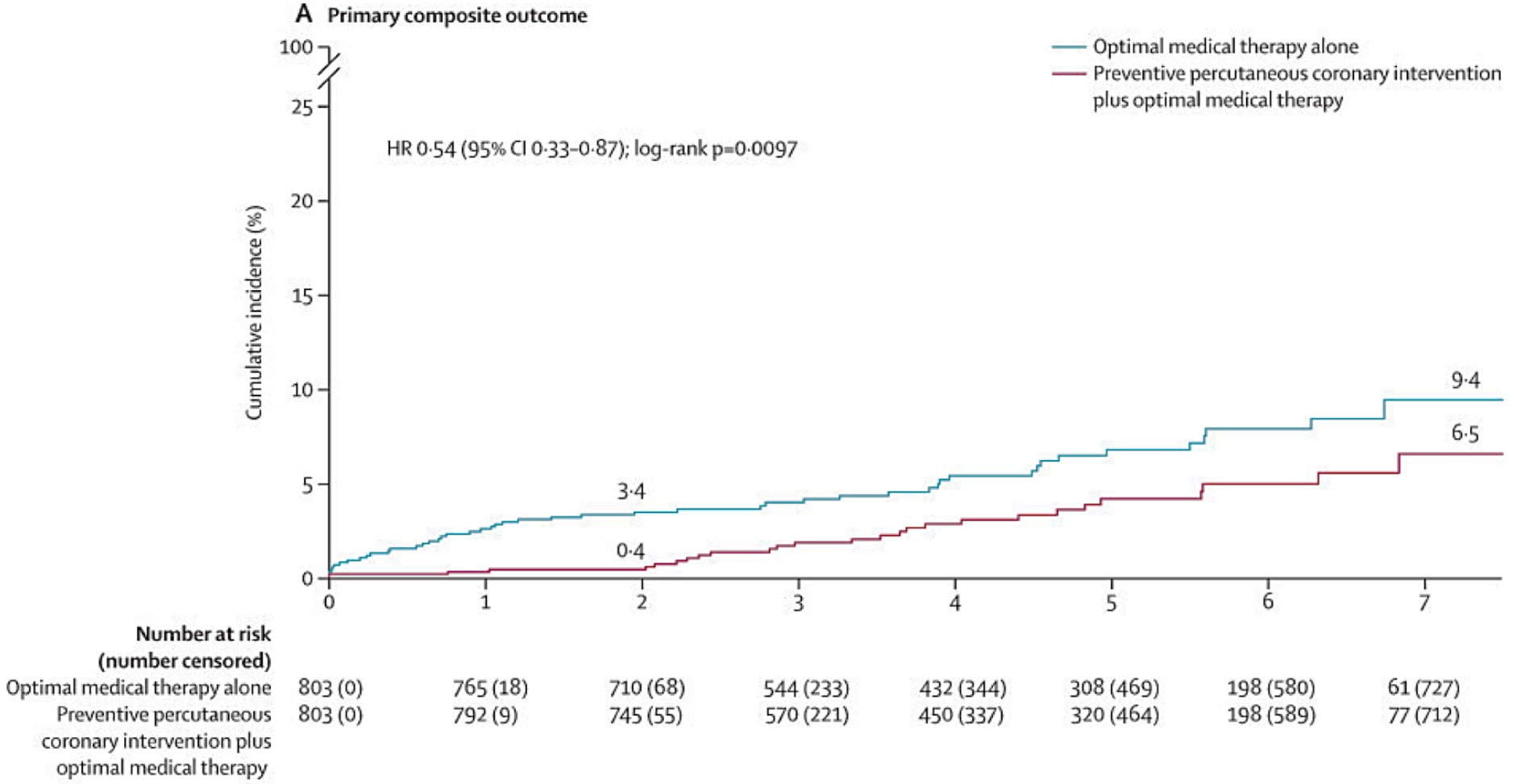
Seung-Jung Park, Jung-Min Ahn*, Do-Yoon Kang, Sung-Cheol Yun, Young-Keun Ahn, Won-Jang Kim, Chang-Wook Nam, Jin-Ok Jeong, In-Ho Chae, Hiroki Shiomi, Hsien-Li Kao, Joo-Yong Hahn, Sung-Ho Her, Bong-Ki Lee, Tae Hoon Ahn, Ki-Yuk Chang, Jei Keon Chae, David Smyth, Gary S Mintz, Gregg W Stone, Duk-Woo Park, for the PREVENT Investigators†*

PREVENT Background

- ACS and SCD are often due to rupture and thrombosis of lipid rich plaque ("vulnerable plaques")
- Non-flow limiting plaques can be vulnerable plaques

Does ***PCI*** for ***non-flow limiting vulnerable plaques*** improve outcomes when compared to ***medical therapy alone***?

PREVENT pētījums: primārais galamērķis



The primary endpoint of target vessel failure at 2 years (composite of death from cardiac causes, target vessel myocardial infarction [TV-MI], ischemia-driven target-vessel revascularization [ID-TLR], or hospitalization for unstable or progressive angina) for PCI + OMT vs. OMT alone, was: 0.4% vs. 3.4% (hazard ratio [HR] 0.11, 95% confidence interval [CI] 0.03-0.36, p = 0.0003).

Target vessel failure at 7 years: 6.5% vs. 9.4% (HR 0.54, 95% CI 0.33-0.87, p = 0.0097)

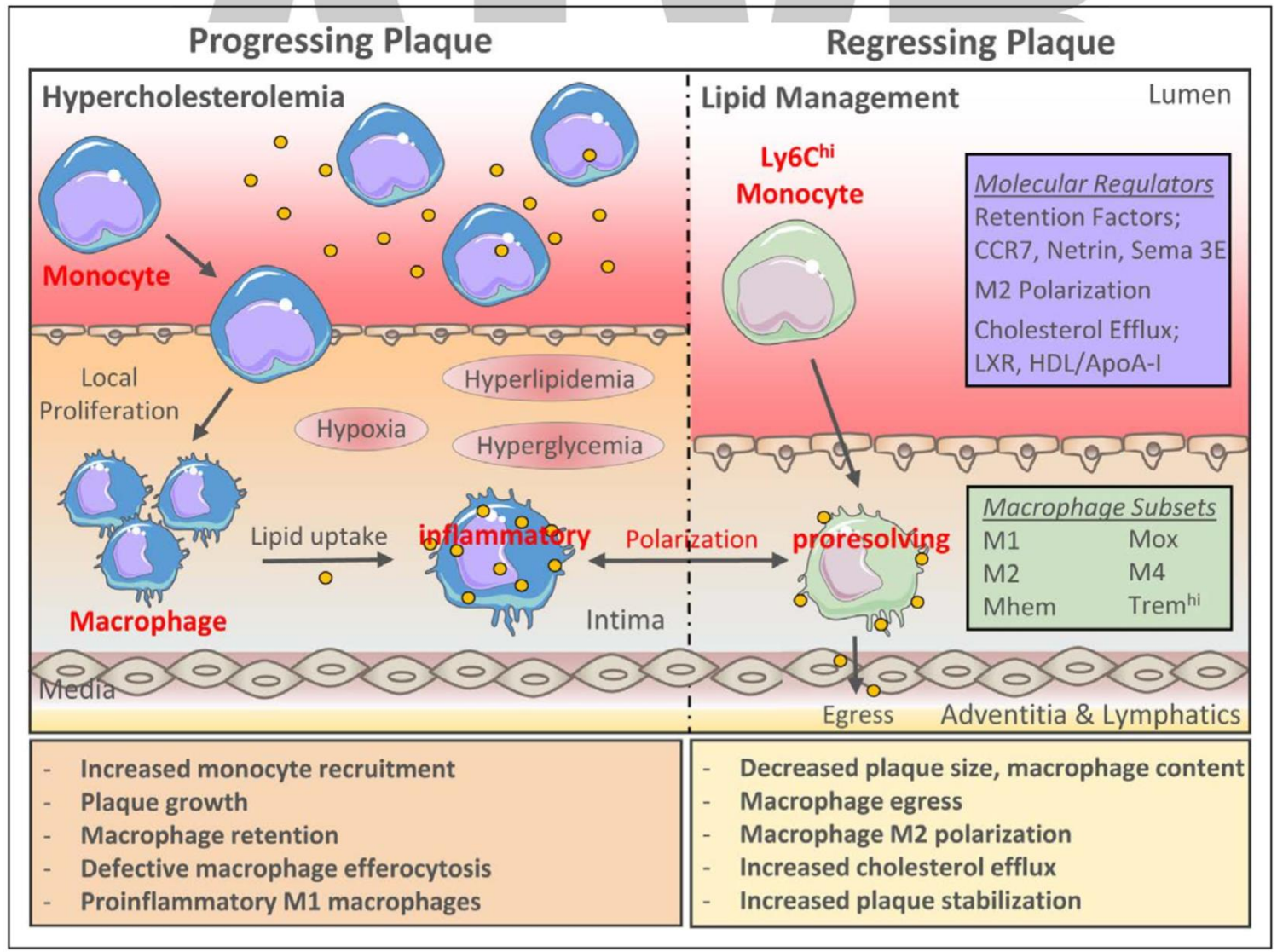
Arterioscler Thromb Vasc Biol. 2020

ATVB IN FOCUS:
The Science of *ATVB* Early Career Committee

Series Editor: Robert A. Hegele

Macrophages in Atherosclerosis Regression

Tessa J. Barrett

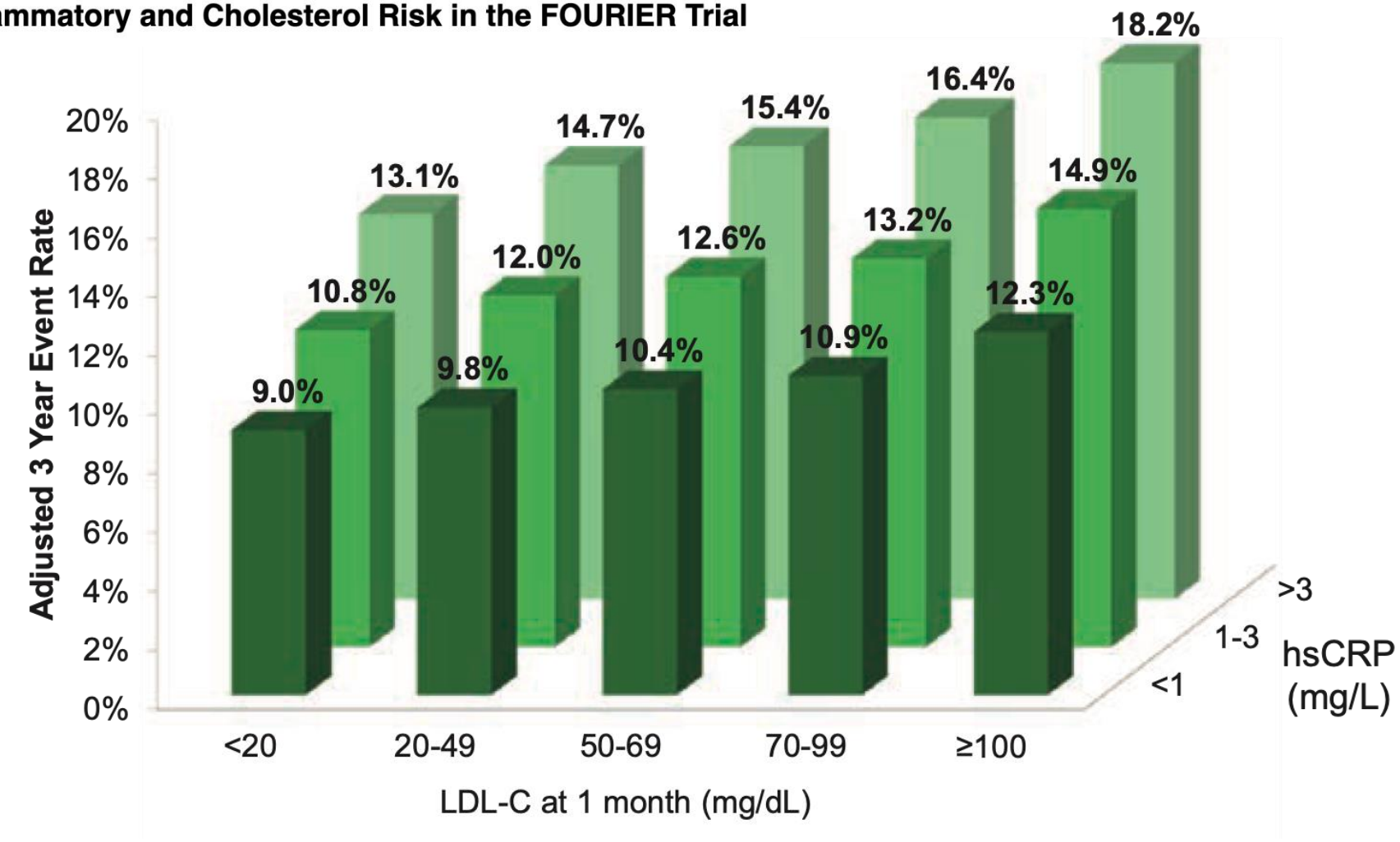


Circulation
Volume 138, Issue 2, 10 July 2018; Pages 131-140
https://doi.org/10.1161/CIRCULATIONAHA.118.034032

Iekaisuma un holesterīns līmeņa ietekme uz KV risku

ORIGINAL RESEARCH ARTICLE

Inflammatory and Cholesterol Risk in the FOURIER Trial



0.5 mmol/l **1.3 mmol/l** **2.6 mmmol/l**

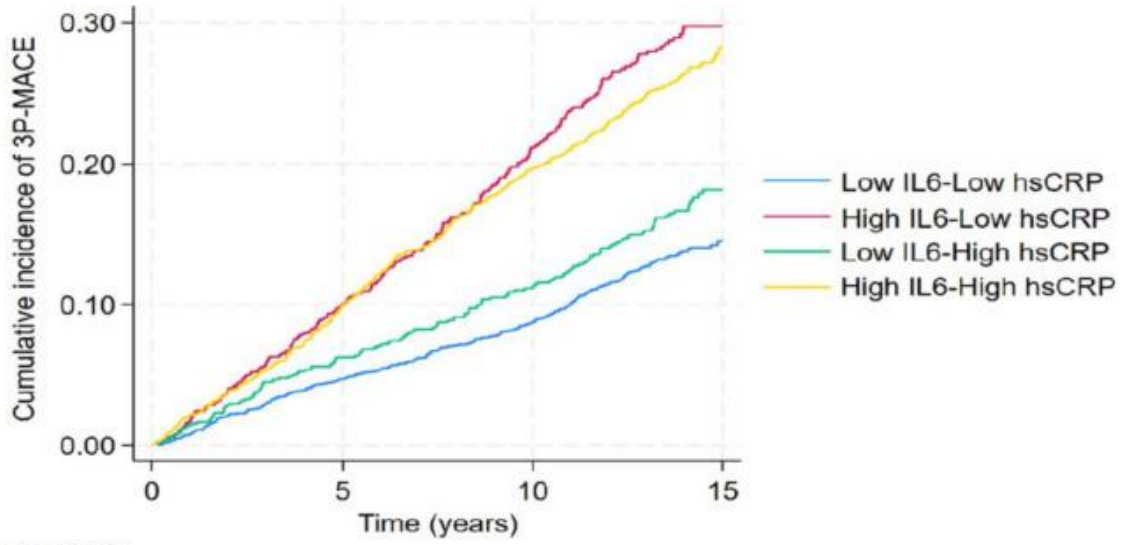
Interleikīns - 6

Current Atherosclerosis Reports (2025) 27:12
https://doi.org/10.1007/s11883-024-01259-7

REVIEW

IL-6 and Cardiovascular Risk: A Narrative Review

Kardiovaskulārā mirstība, insults, miokarda infarkts



Number at risk			
Low IL6-Low hsCRP 2328	2104	1885	223
High IL6-Low hsCRP 1002	834	651	55
Low IL6-High hsCRP 975	867	760	101
High IL6-High hsCRP 2288	1880	1511	180

- Mehta NN, deGoma E, Shapiro MD. IL-6 and Cardiovascular Risk: A Narrative Review. *Curr Atheroscler Rep*. 2024;27(1):12. Published 2024 Nov 26. doi:10.1007/s11883-024-01259-7

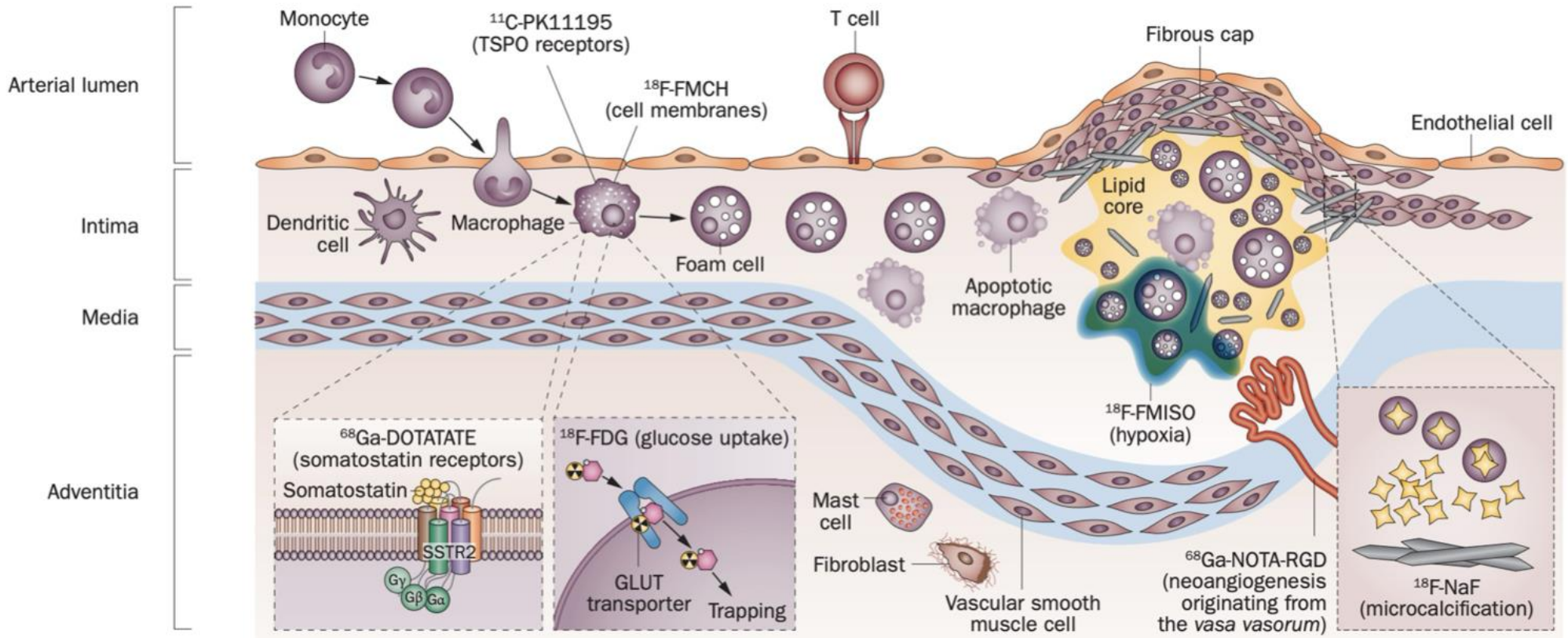
Aterosklerozes iekaisuma attēldiagnostika

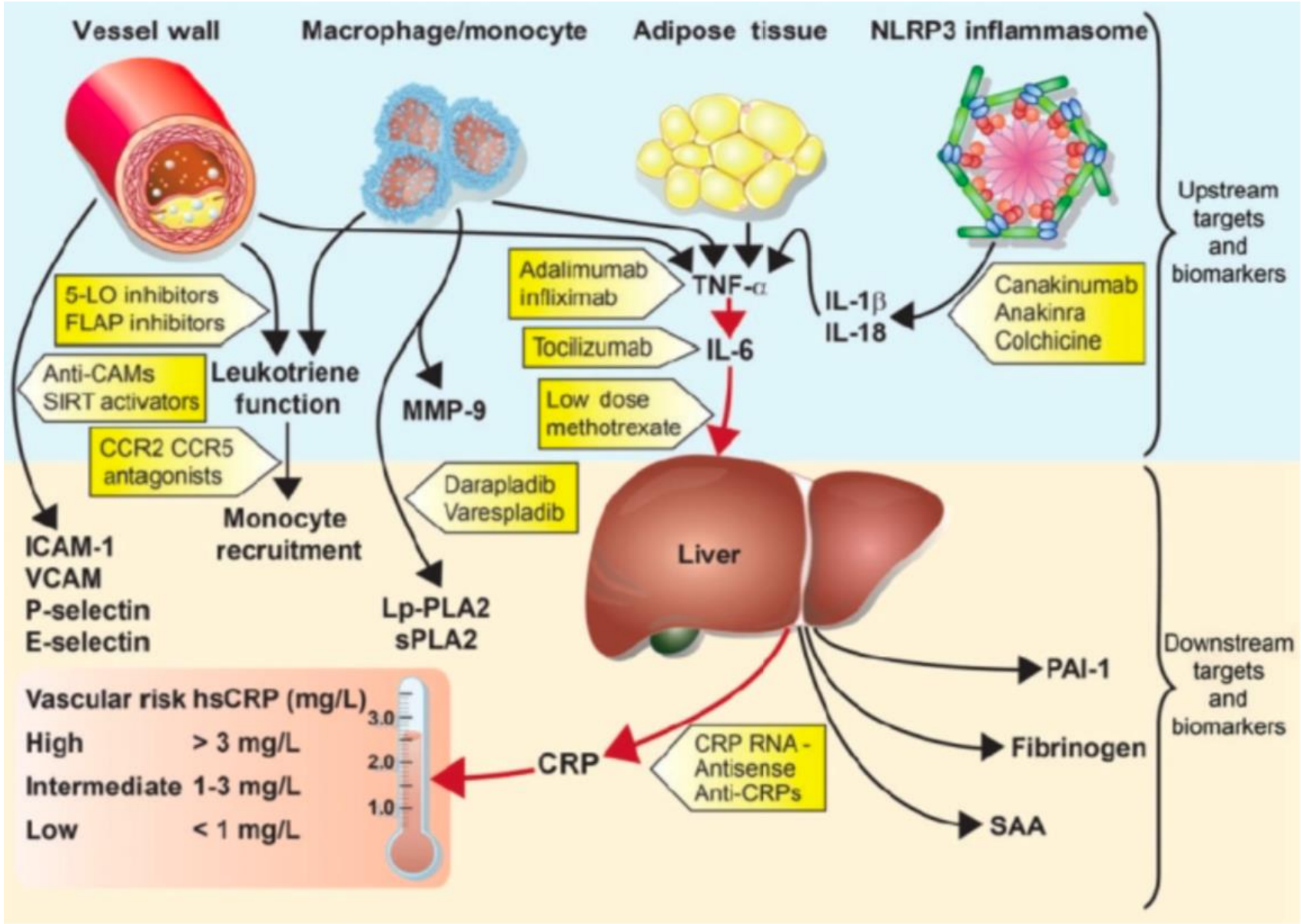
nature > nature reviews cardiology > review articles > article

Published: 10 June 2014

PET imaging of inflammation in atherosclerosis

Jason M. Tarkin, Francis R. Joshi & James H. F. Rudd





European Heart Journal. 2014;35:1782–1791

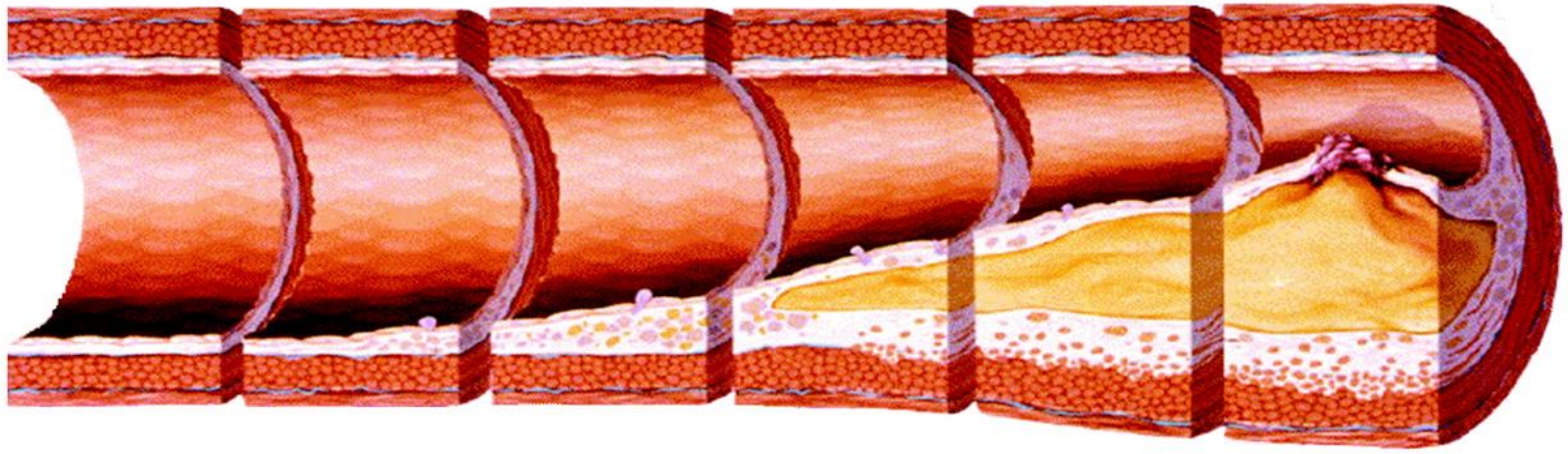
Recommendation Table 18 — Recommendations for lipid-lowering drugs in patients with chronic coronary syndrome (see also Evidence Table 18)

Recommendations	Class[a]	Level[b]
Lipid-lowering treatment with an LDL-C goal of <1.4 mmol/L (55 mg/dL) and a ≥50% reduction in LDL-C vs. baseline is recommended.[64,670,671]	I	A
For patients with a recurrent atherothrombotic event (not necessarily of the same type as the first event) while taking maximally tolerated statin therapy, an LDL-C goal of <1.0 mmol/L (<40 mg/dL) may be considered.[675,676]	IIb	B

Recommendation Table 20 — Recommendations for anti-inflammatory drugs in patients with chronic coronary syndrome (see also Evidence Table 20)

Recommendation	Class[a]	Level[b]
In CCS patients with atherosclerotic CAD, low-dose colchicine (0.5 mg daily) should be considered to reduce myocardial infarction, stroke, and need for revascularization.[714–716]	IIa	A

Asins biomarķieri dažādās ateroģenēzes stadijās

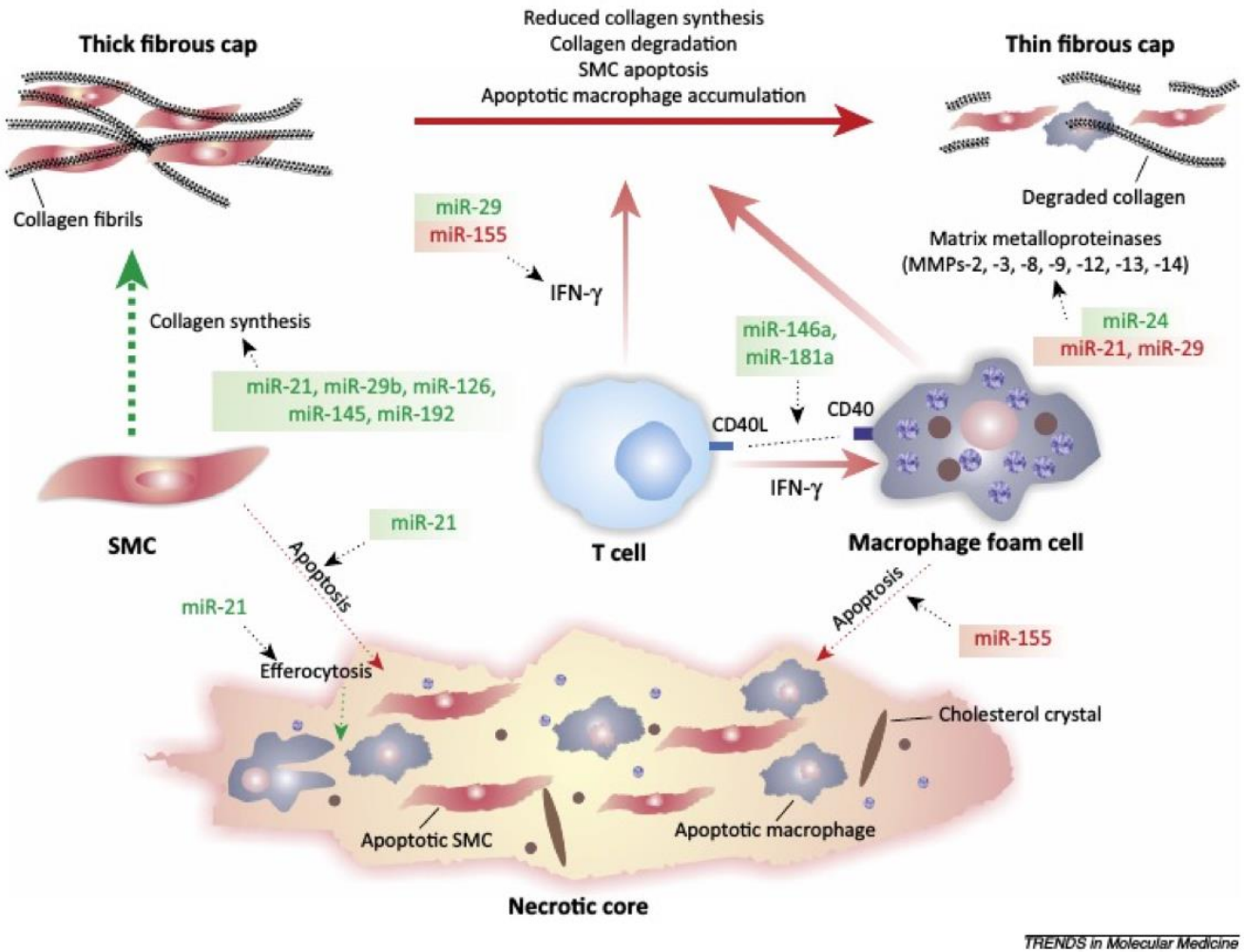


1°& Messenger Inflamm. Cyto/Chemokines		Cellular Adhesion Molecules	Plaque Destabilization		Plaque Rupture
IL-1	IL-6*	sICAM	IL-18*	MPO*	PAPP-A*
TNF-α	IL-18*	sVCAM	oxLDL*	MMPs *	sCD40L*
	MCP-1*	sSelectins	Lp-PLA_2*	MCP-1*	
			GPx-1*	PlGF*	

Acute Phase Reactants

CRP*, sPLA_2*, SAA, Fibrinogen, WBCC

MikroRNS un pangas destabilizācija



Green miRNAs indicate those with stabilizing properties, while red miRNAs indicates those with destabilizing properties.

Ioannis Andreou et al. Trends in Molecular Medicine May 2015, Vol. 21, No. 5
http://dx.doi.org/10.1016/j.molmed.2015.02.003

ESC European Society of Cardiology
European Heart Journal - Cardiovascular Imaging (2025) **26**, 444–460
https://doi.org/10.1093/ehjci/jeae314

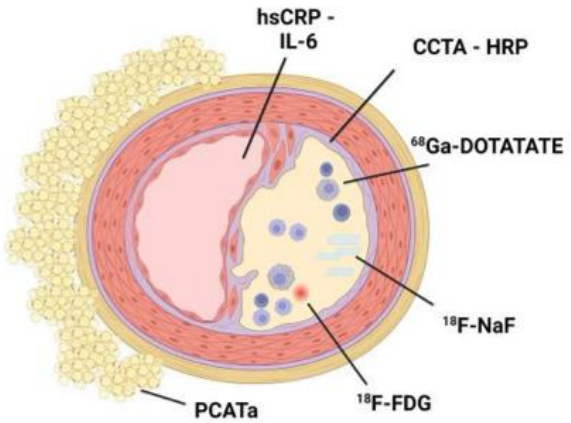
REVIEW

Estimating inflammatory risk in atherosclerotic cardiovascular disease: plaque over plasma?

Maxim E. Annink [1†], Jordan M. Kraaijenhof [1†], Cheyenne Y.Y. Beverloo [1], Reindert F. Oostveen [1], Hein J. Verberne [2], Erik S.G. Stroes[1], and Nick S. Nurmohamed [1,3]*

[1]Department of Vascular Medicine, Amsterdam UMC, University of Amsterdam, Meibergdreef 9, 1105AZ Amsterdam, The Netherlands; [2]Department of Radiology & Nuclear Medicine, Amsterdam UMC, University of Amsterdam, Meibergdreef 9, 1105AZ Amsterdam, The Netherlands; and [3]Department of Cardiology, Amsterdam UMC, Vrije Universiteit Amsterdam, De Boelelaan 1117, 1081HV Amsterdam, The Netherlands

Inflammation in ASCVD



hsCRP IL-6	Systemic (non)specific inflammation
^{68}Ga-DOTATATE	M1 Macrophages
^{18}F-NaF	Microcalcifications
^{18}F-FDG	Metabolic activity
CCTA-HRP	High-risk plaque
PCATa	Pericoronary inflammation

Associated with clinical events
And may be used for:

- ASCVD risk assessment
- Medical therapy efficacy

From a population-based approach to personalized medicine

Population-based strategy
Current standard of care

- Systemic plasma markers
- While affordable and easily obtained, lacks specificity and discriminating capacity

Personalized medicine strategy
Visualizing and identifying inflammation

- Imaging disease phenotype
- May provide improvement in risk prediction and selection and monitoring of therapy

Estimating inflammatory risk: from population-based plasma markers to personalized medicine. ASCVD, atherosclerotic cardiovascular disease; hsCRP, high-sensitivity C-reactive protein; IL-6, interleukin 6; ^{18}F-FDG, 18F-fluorodeoxyglucose; ^{68}Ga-DOTATATE, gallium-68 DOTATATE; ^{18}F-NaF, 18F-sodium fluoride; PCATa, pericoronary adipose tissue attenuation; CCTA–HRP, coronary computed tomography angiography–high-risk plaque.

The *Lancet* Commission on rethinking coronary artery disease: moving from ischaemia to atheroma

Sarah Zaman, Jason H Wasfy, Vikas Kapil, Boback Ziaeian, William A Parsonage, Sira Sriswasdi, Timothy J A Chico, Davide Capodanno, Róisín Colleran, Nadia R Sutton, Lei Song, Nicole Karam, Reecha Sofat, Chiara Fraccaro, Daniel Chamié, Mirvat Alasnag, Takayuki Warisawa, Nieves Gonzalo, Walid Jomaa, Shamir R Mehta, Elizabeth E S Cook, Johan Sundström, Stephen J Nicholls, Leslee J Shaw, Manesh R Patel, Rasha K Al-Lamee

Lancet 2025; 405: 1264–312

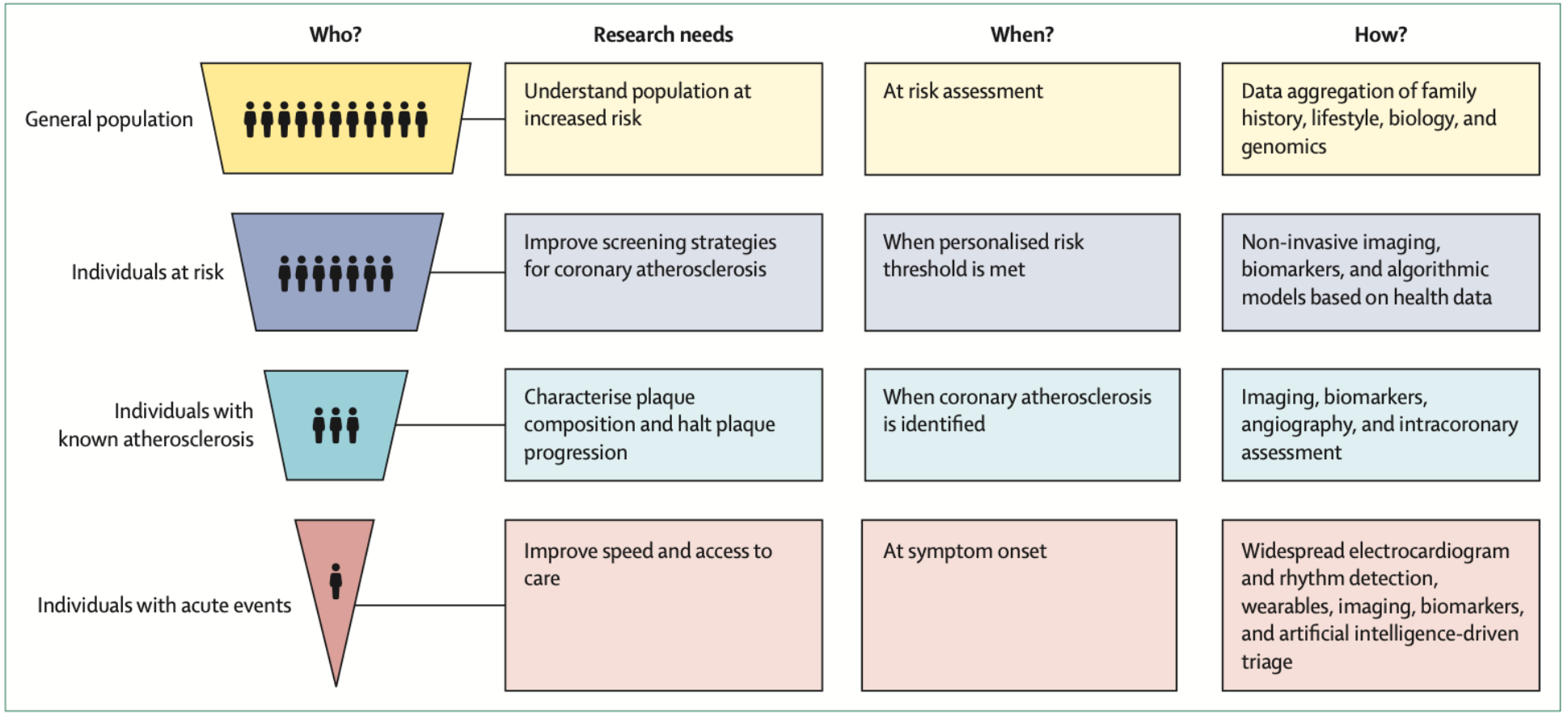
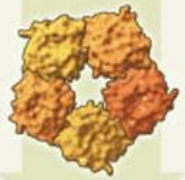


Figure 11: **Guidance on screening and investigation of atherosclerotic coronary artery disease**

Reziduālais risks

Despite contemporary evidence-based therapies*, residual risk of ASCVD events persists

Biological Issue	Residual Cholesterol Risk	Residual Inflammatory Risk	Residual Thrombotic Risk	Residual Triglyceride Risk	Residual Lp(a) Risk	Residual Diabetes Risk
						
Critical Biomarker	LDL-C ≥100 mg/dL	hsCRP ≥2mg/L	No simple biomarker	TG ≥150mg/dL	Lp(a) ≥50mg/dL	HbA1c Fasting glucose
Potential Intervention	Targeted LDL/Apo B Reduction	Targeted Inflammation Reduction	Targeted Antithrombotic Reduction	Targeted Triglyceride Reduction	Targeted Lp(a) Reduction	SGLT2 Inhibitors GLP-1 Agonists
Randomized Trial Evidence	IMPROVE-IT FOURIER SPIRE ODYSSEY	CANTOS COLCOT *LoDoCo2* *OASIS-9*	PEGASUS COMPASS THEMIS	REDUCE-IT *PROMINENT*	Planned	EMPA-REG CANVAS DECLARE CREDENCE LEADER SUSTAIN-6 REWIND

- Lawler PR, Bhatt DL, Godoy LC, et al. Targeting cardiovascular inflammation: next steps in clinical translation. *Eur Heart J*. 2021;42(1):113-131. doi:10.1093/eurheartj/ehaa099