Disclosures

- Amarin
- Amgen

Patienter med residual kardiovaskulär risk – kan vi bli bättre?

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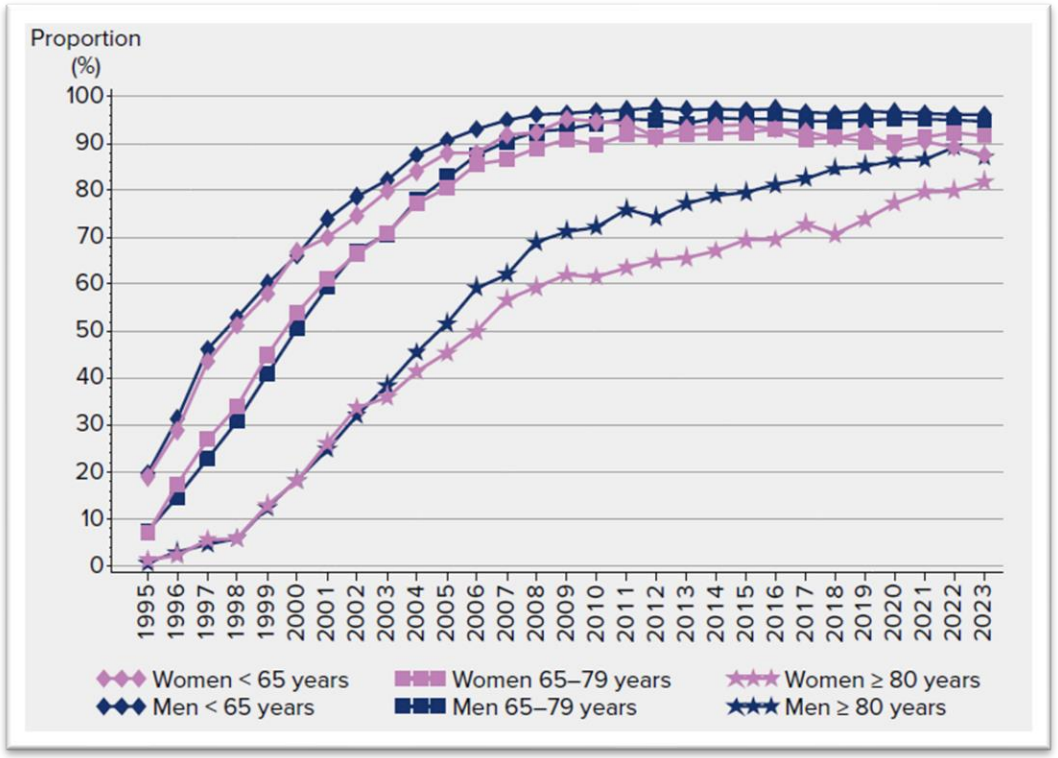
Kardiologiska kliniken
Skånes Universitetssjukhus Lund

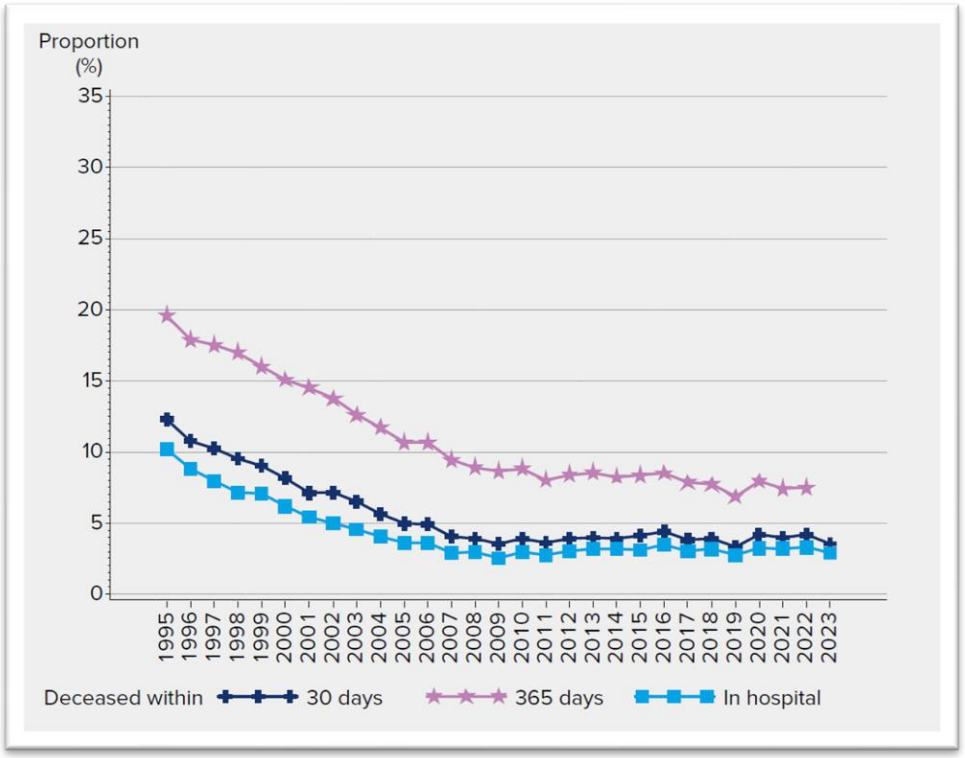
LUND UNIVERSITY

SWEDEHEART annual report 2023[1]

Trends in lipid-lowering therapy at discharge
in MI patients in relation to age

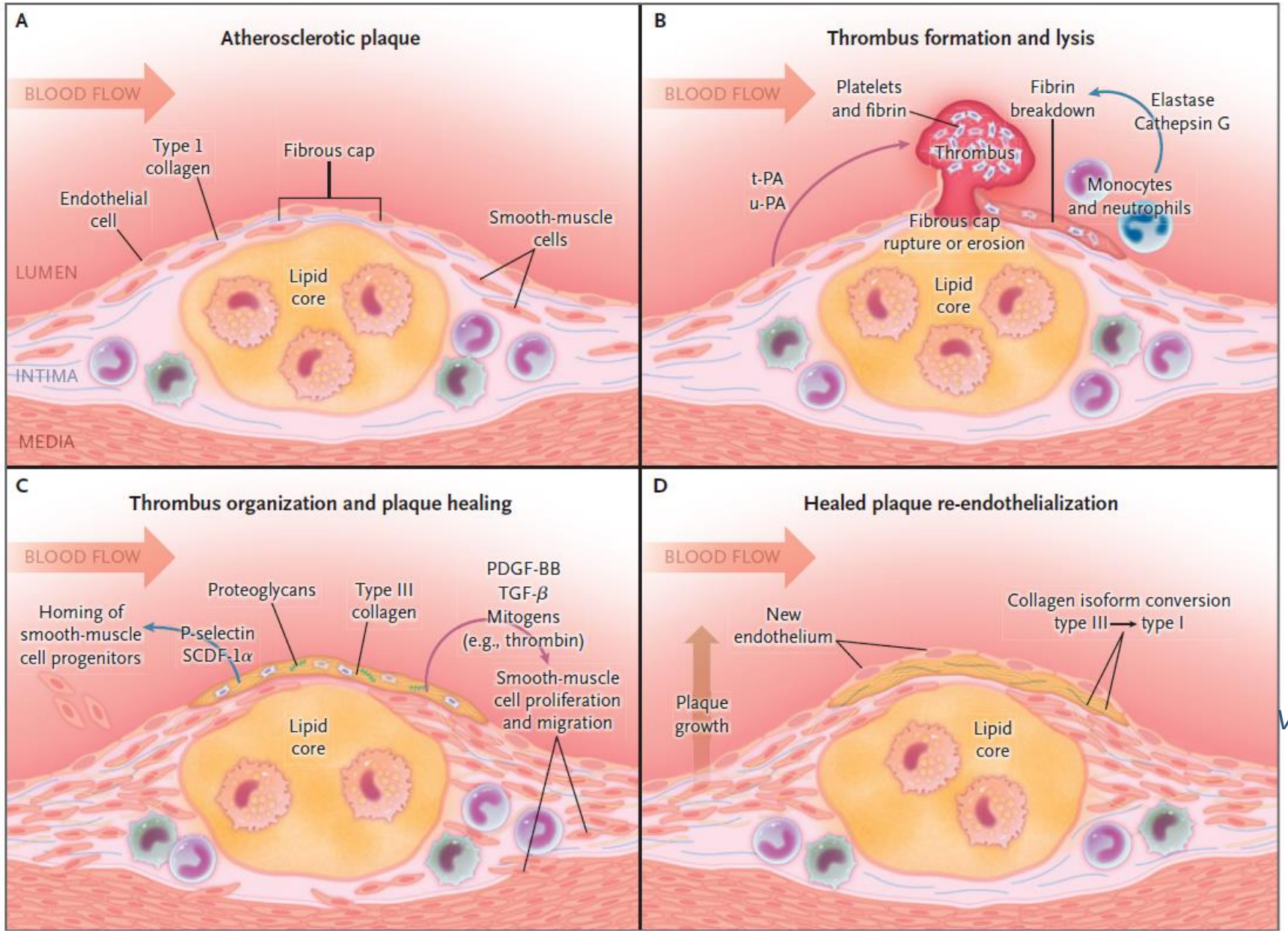


Trends in mortality in MI patient < 80 years.
The mortality reduction appears to have reached a plateau.



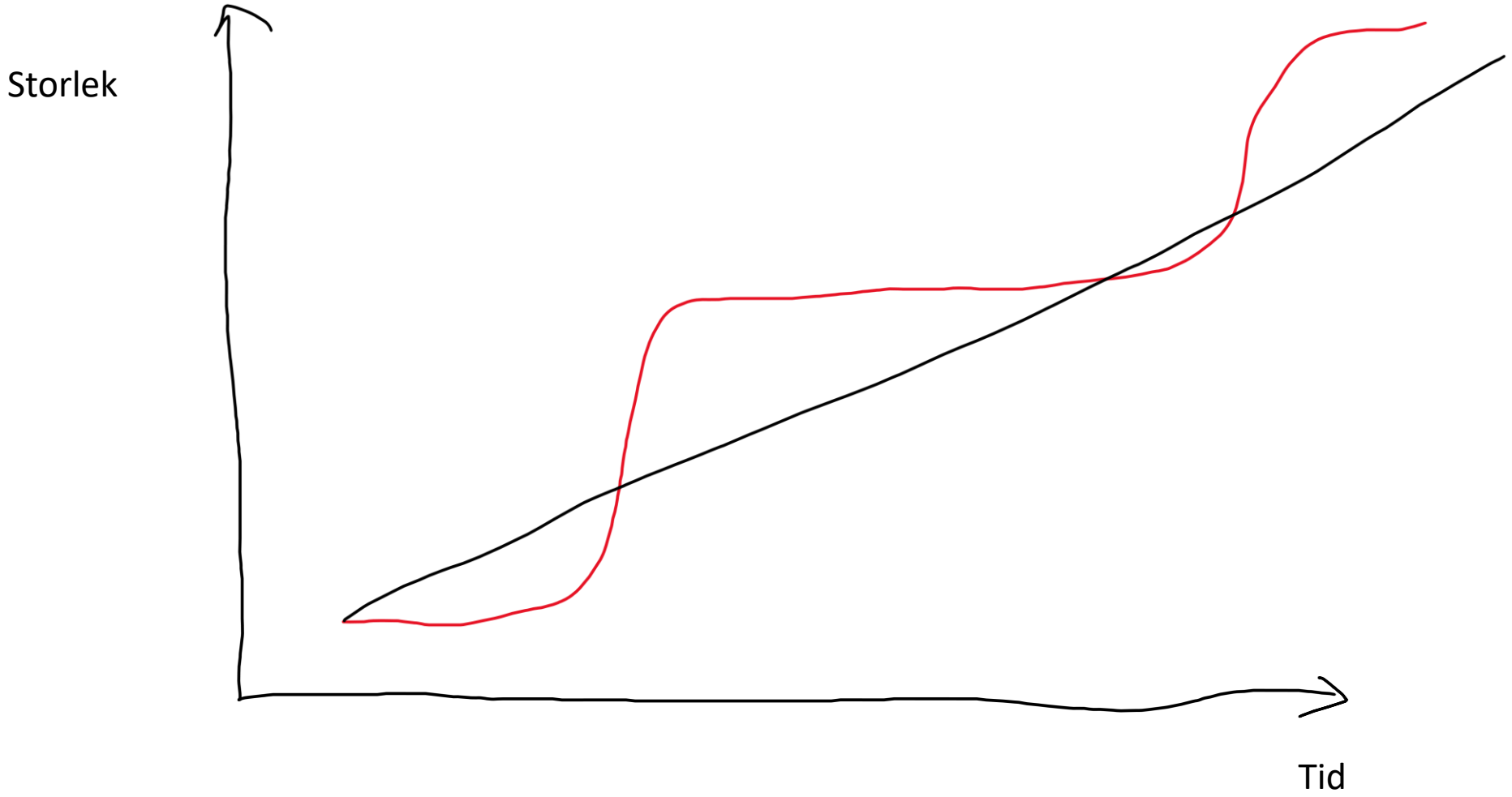
1. Swedeheart, Swedeheart Annual Report 2023 https://www.ucr.uu.se/swedeheart/dokument-sh/arsrapporter-sh

Oerhört komplex process!

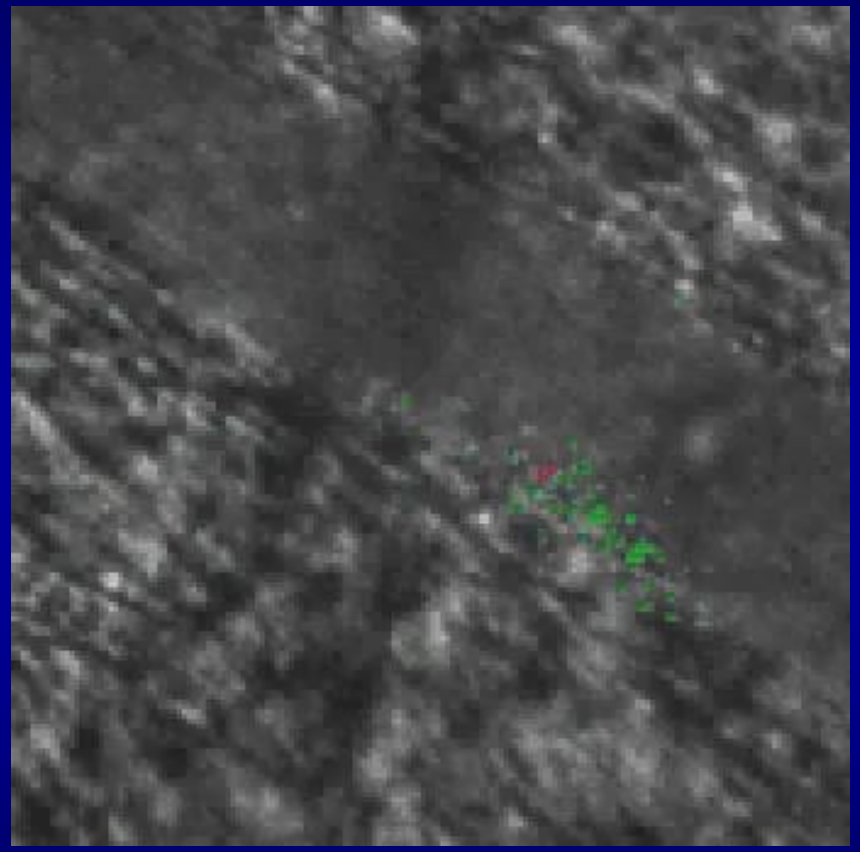


Vergallo et al., NEJM 2020

LUND UNIVERSITY


Storlek
Tid

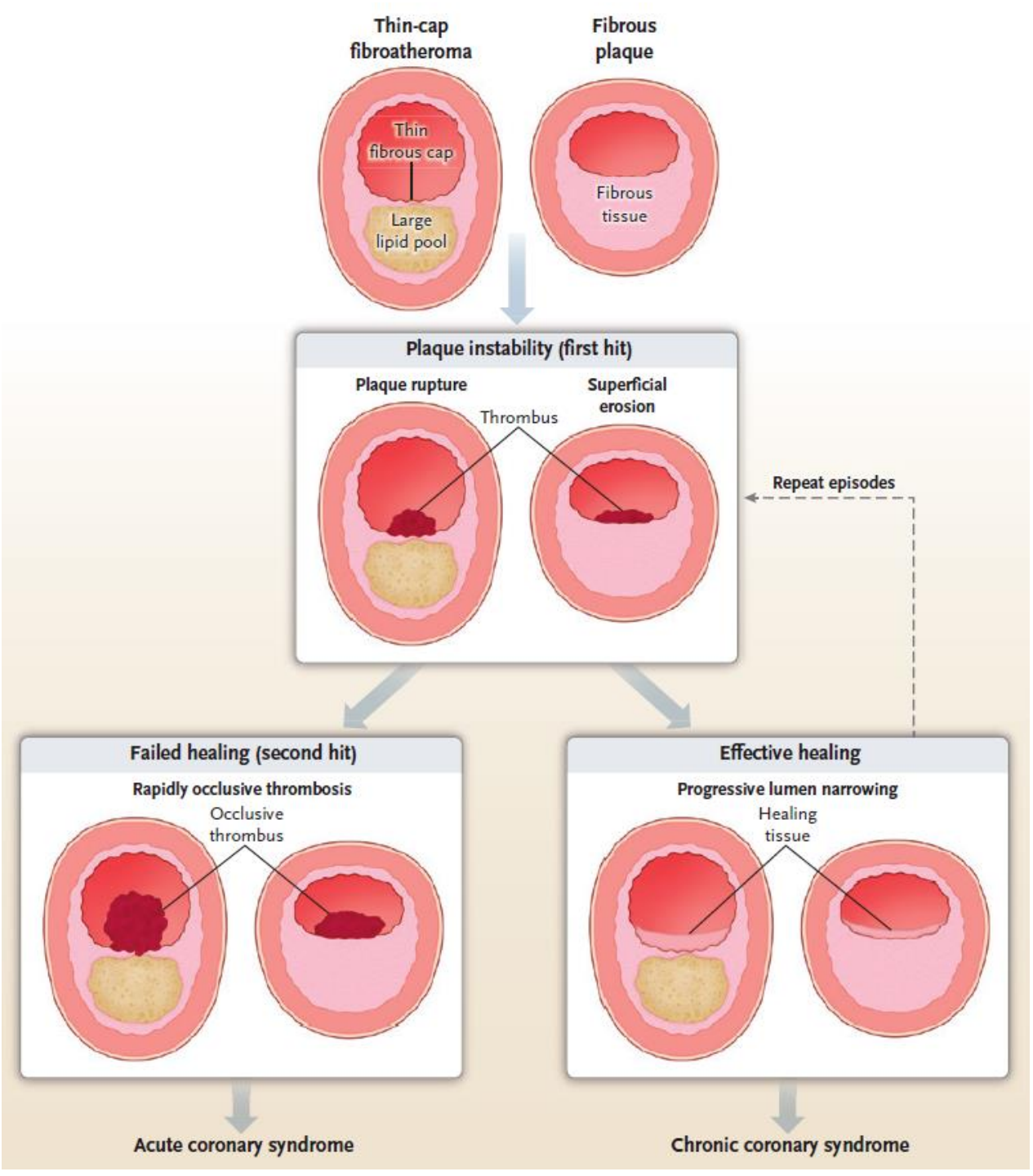
Varför måste vi hämma trombocyter?



platelets (red)
tissue factor (green)
fibrin (blue)
platelets + tissue factor (yellow)
tissue factor + fibrin (turquoise)
platelets + fibrin (magenta)

platelets + fibrin + tissue factor (white)

Falati, S et al (2002). Real-time in vivo imaging of platelets, tissue factor and fibrin during arterial thrombus formation in the mouse. *Nature Medicine*, *8*(10), 1175–1181.



Vergallo et al., NEJM 2020

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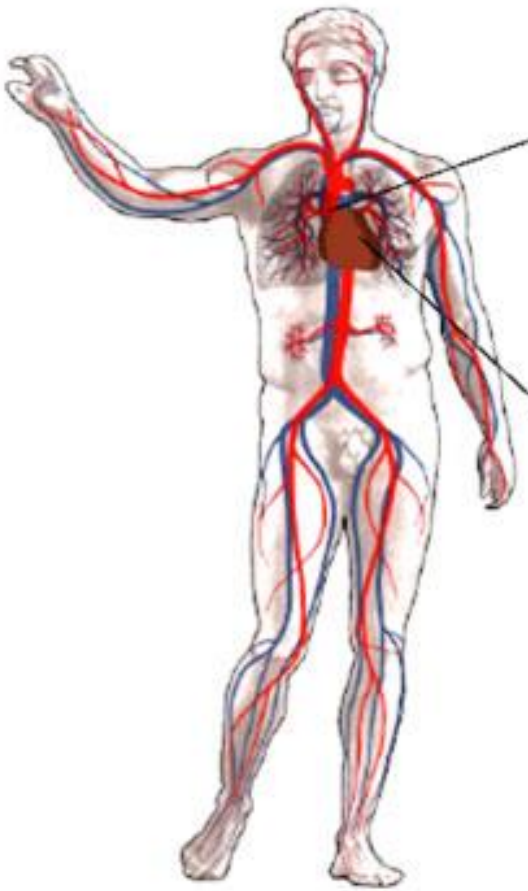
Vulnerable patient

Hypertension
Diabetes
Lipids
Obesity
Thrombosis
Inflammation
Immune response
Gut microbiome

Smoking
Diet
Exercise
Stress
Socieconomics

Omics
Imaging



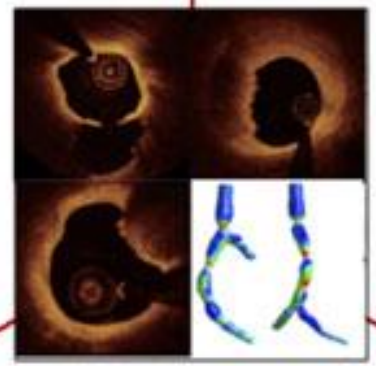
Vulnerable blood

High-risk and culprit plaques

Morphology
Composition

Plaque rupture

Plaque erosion



Calcified nodule

Functional alterations

Biomechanics

Biology
Inflammation

Tomaniak et al., EHJ 2020

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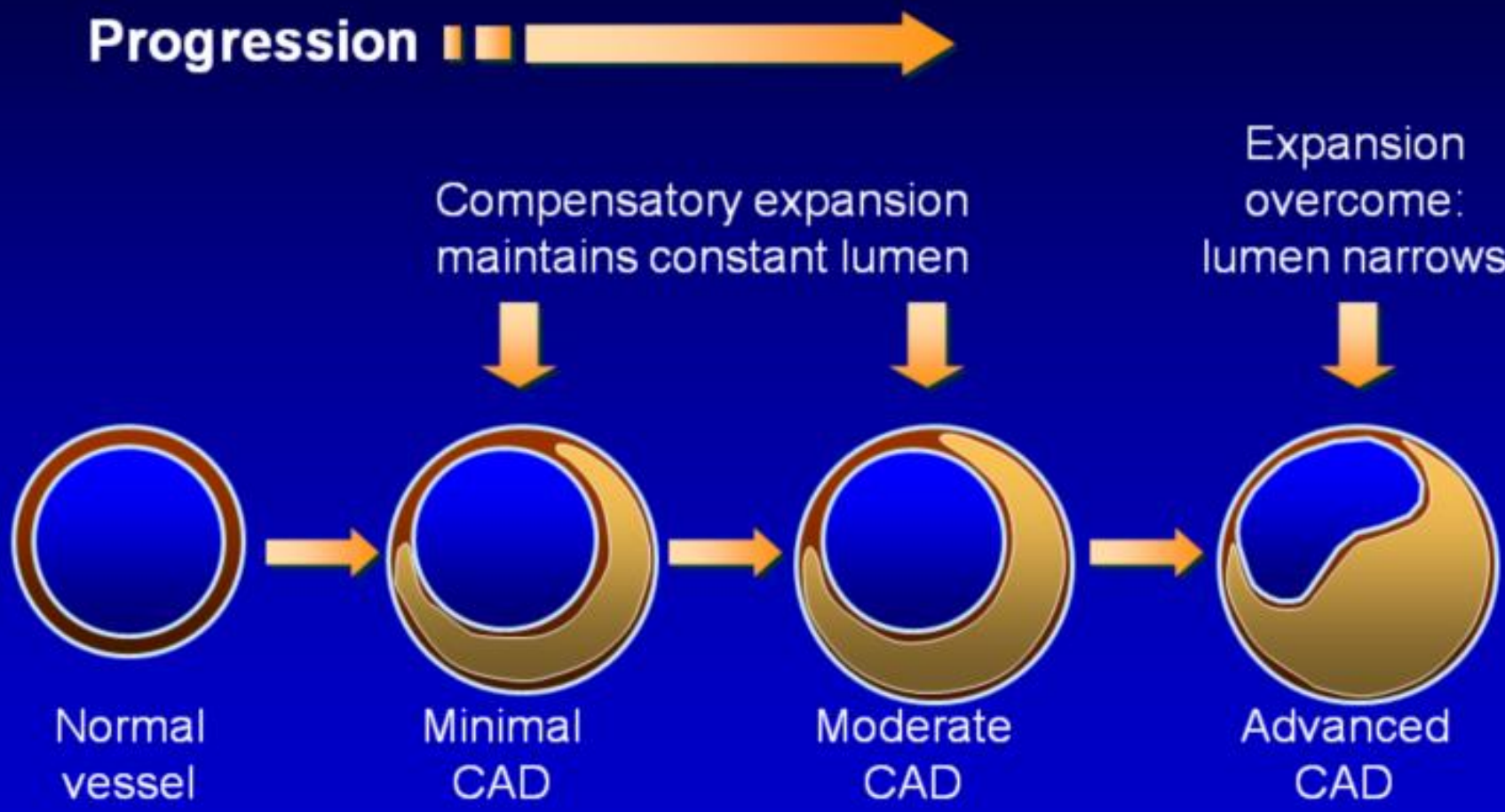
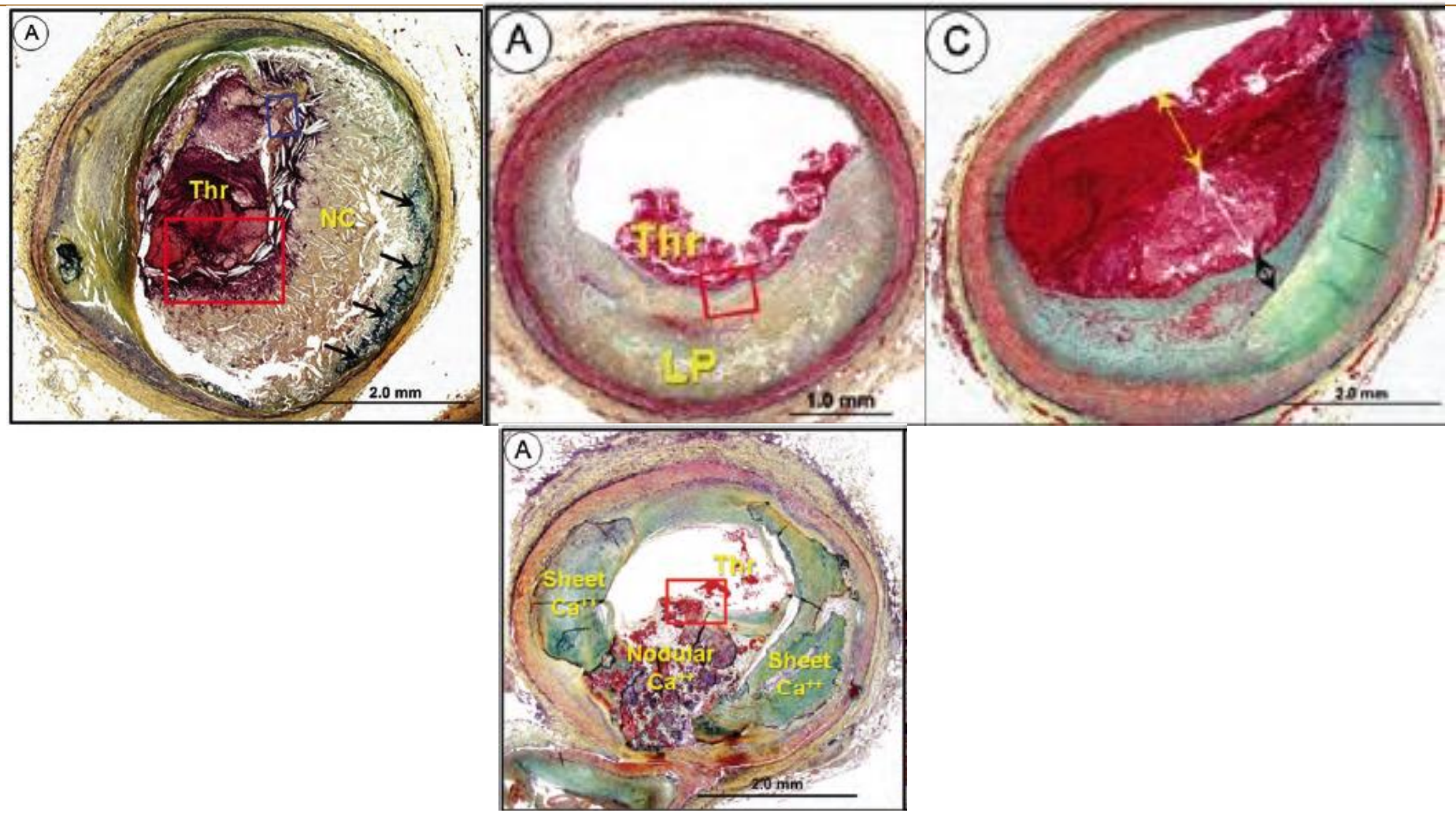
Glagov's Remodelling Hypothesis



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

Många vulnerabla är inte flödesbegränsande innan de ruptur erar



Falk et al., EHJ 2013

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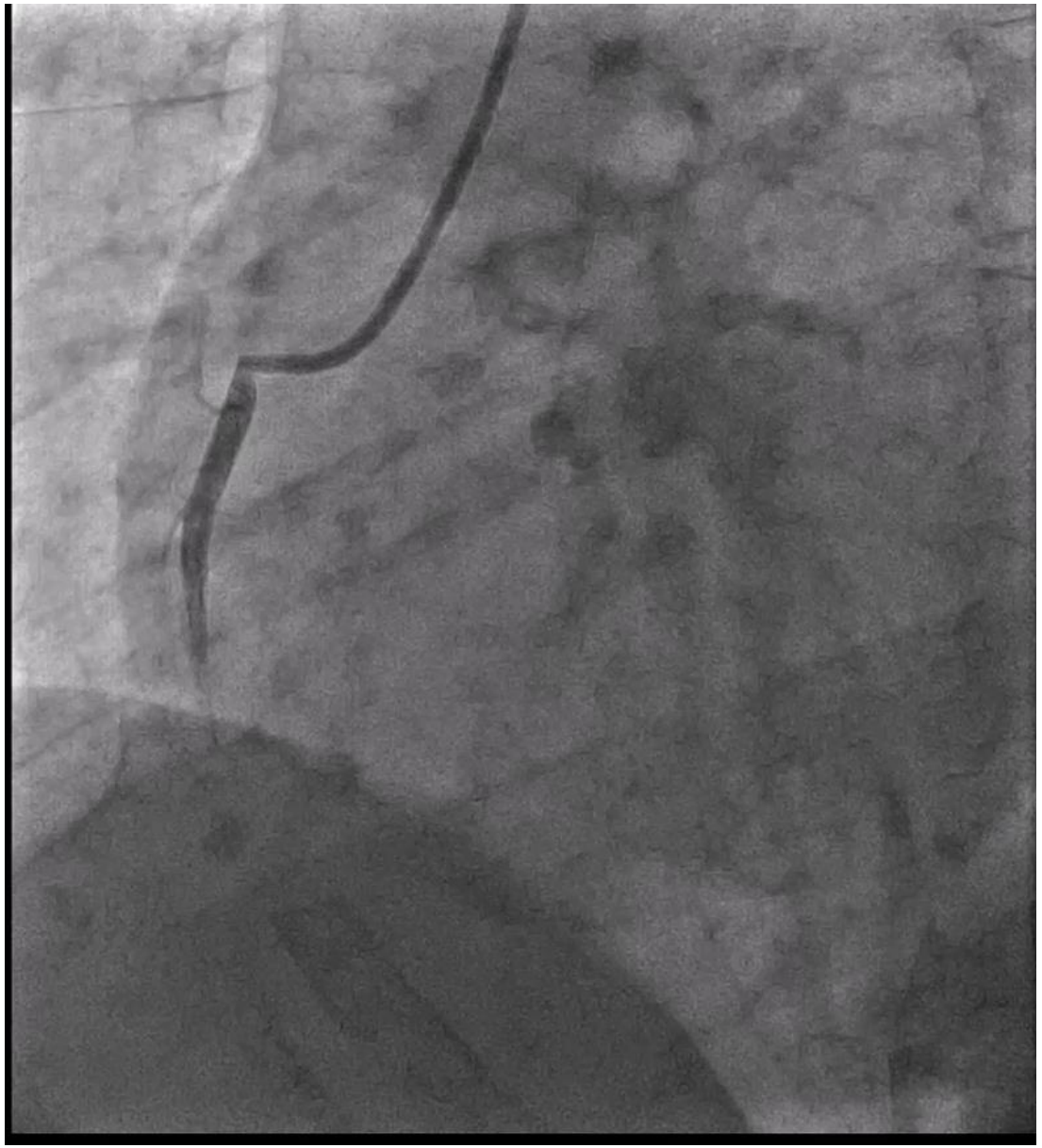
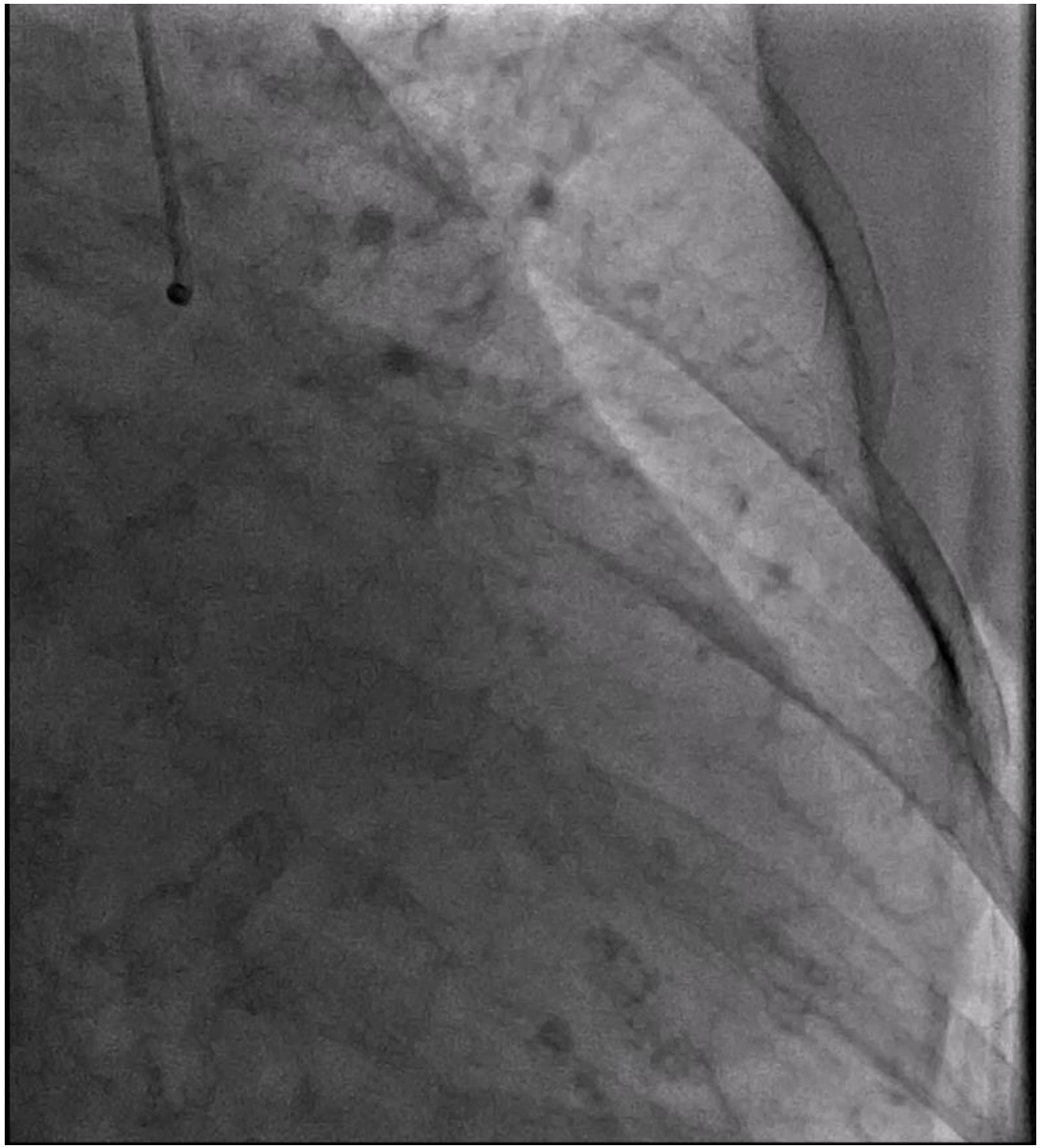
Fallbeskrivning

- Kvinna, 51 år gammal.
- Välreglerad typ 2 diabetes
- Metformin + Atorvastatin 40 mg + kost + motion
- BMI 27 (gått ner 5 kg i vikt på ett år)
- Hb 135, Krea 65, HbA1c 54, LDL 1.6, TG 2.1
- Ingen mikroalbuminuri.
- BT 135/80.

- Inkommer pga oklara bröstsmärtor nov 2022.
- EKO av hjärtat med normal pumpförmåga. Inga klaffel.
- Angiografi utan anmärkning. Fortsatt utredning bekräftar misstanke om perimyokardit. Skrivs ut välmående.

Fallbeskrivning

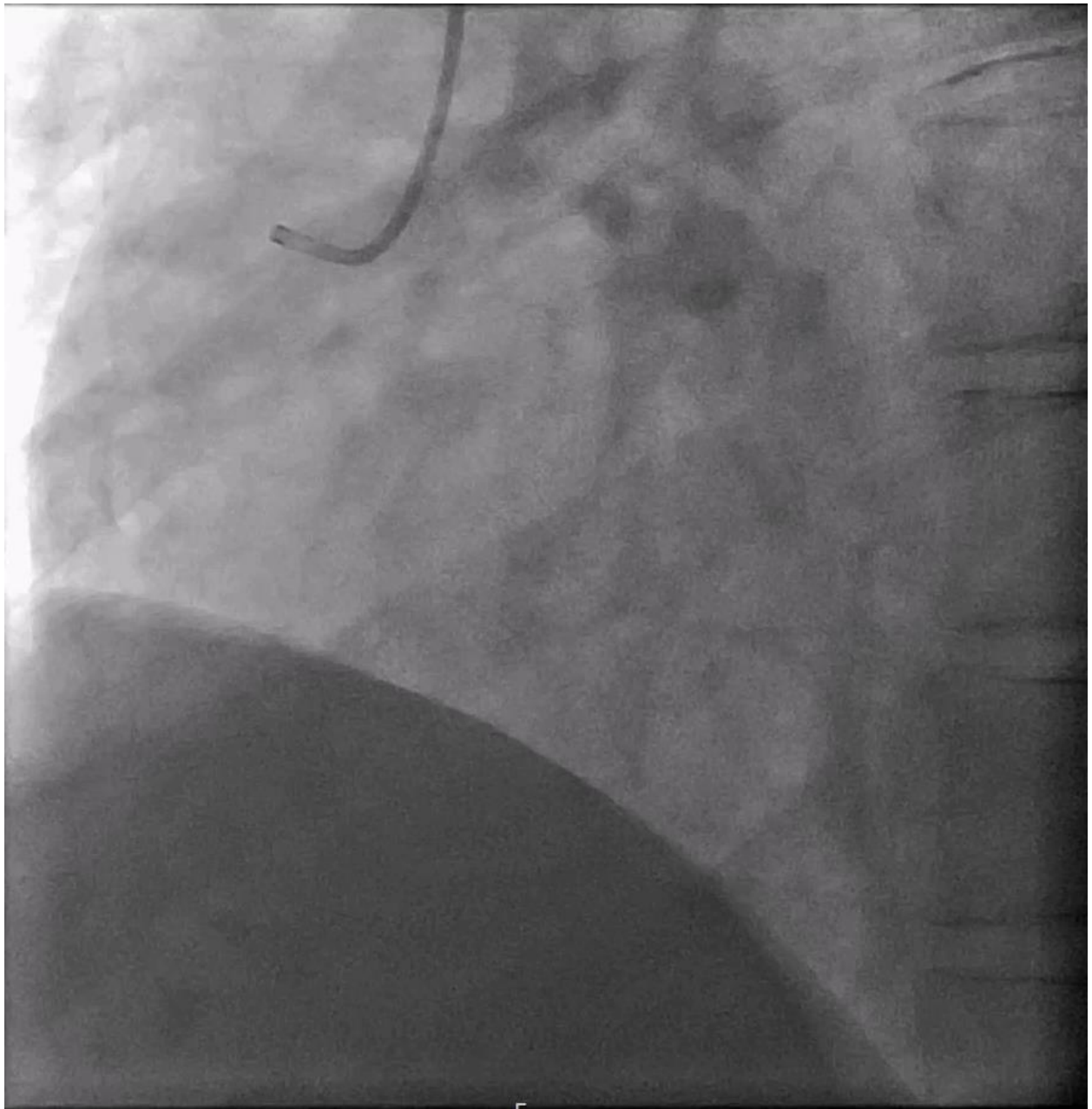


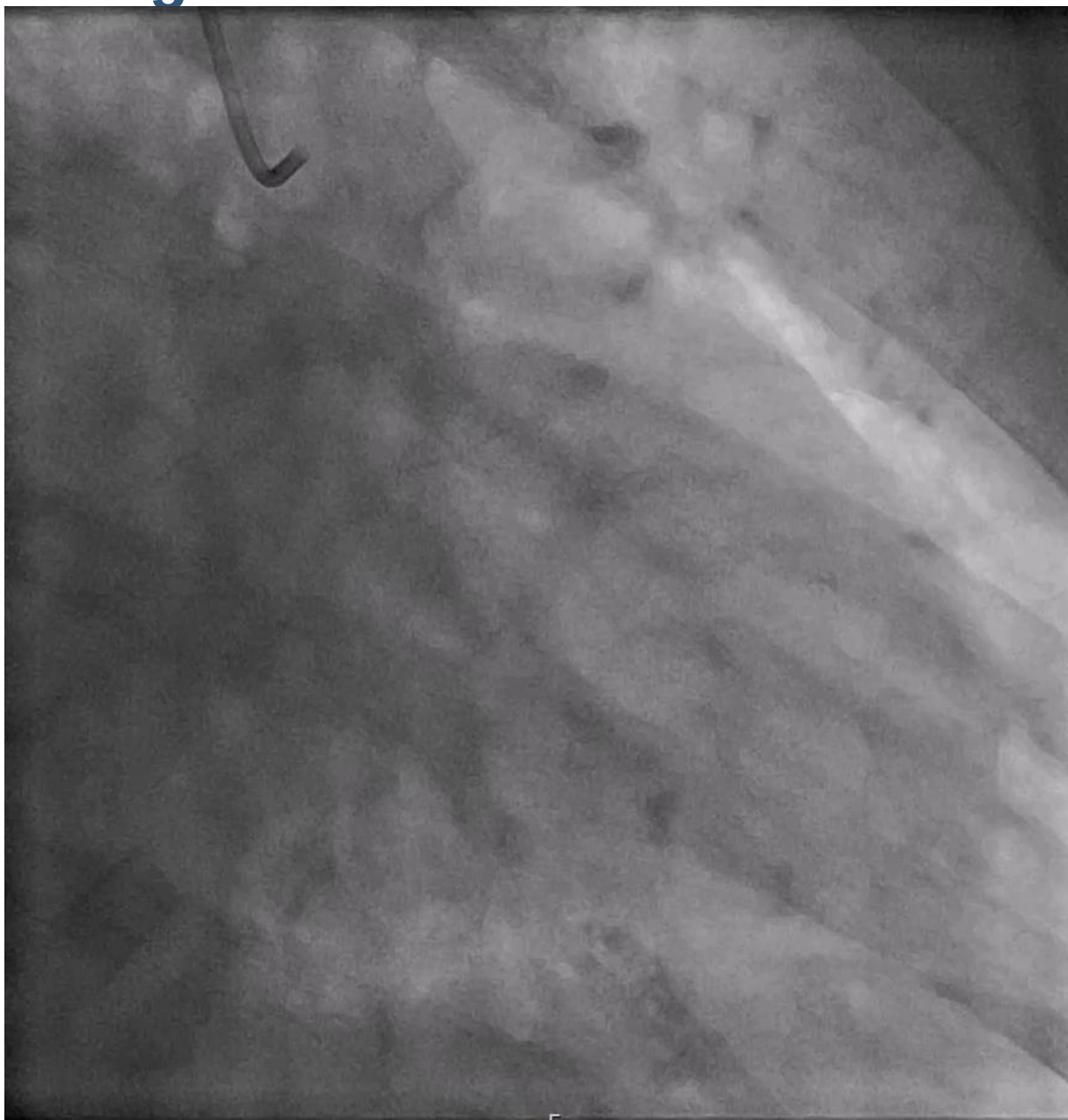


Fallbeskrivning

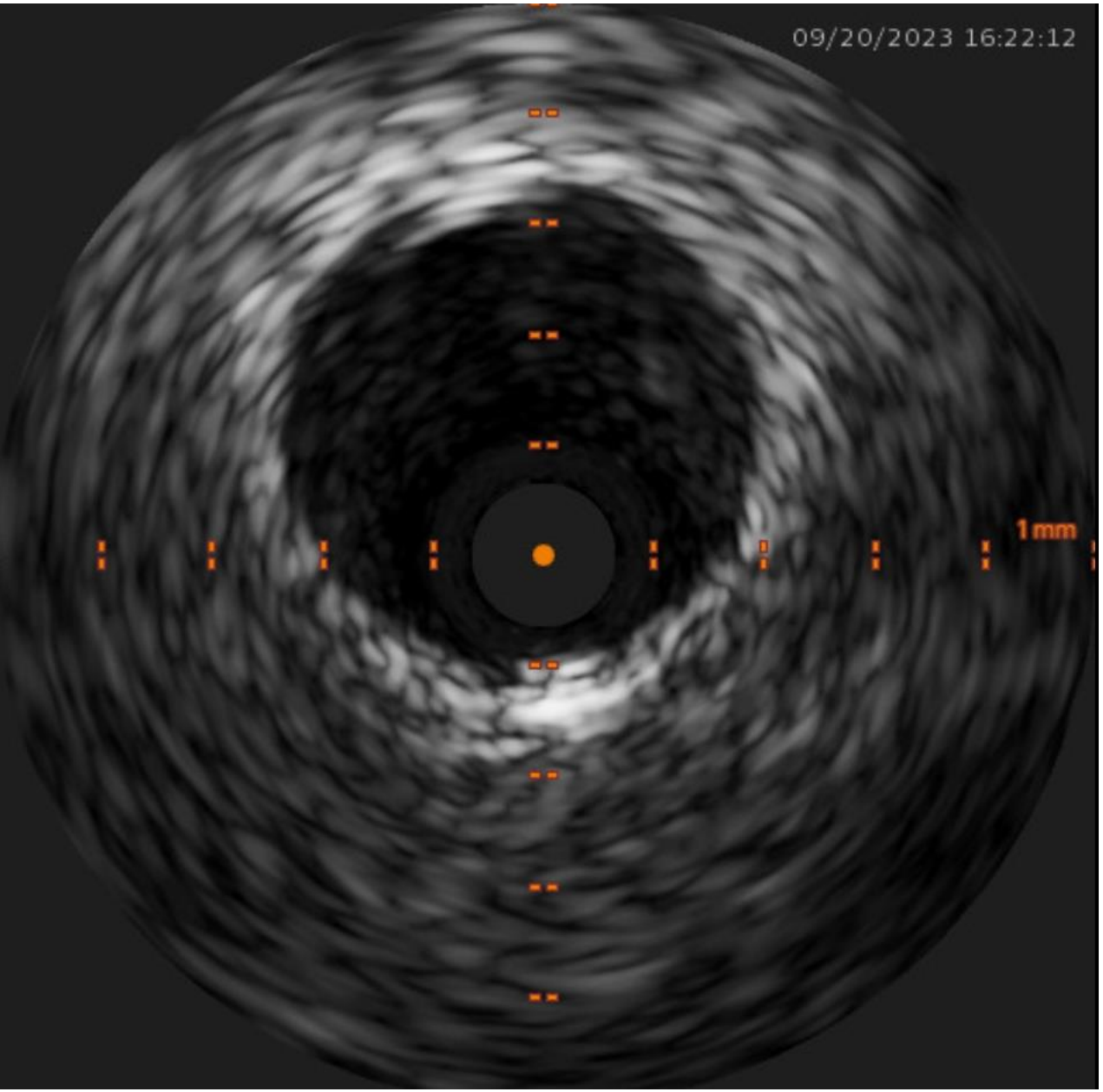
- Senaste månaderna tilltagande bröstsmärta och andfåddhet.
- Söker vård nu i september 2023 pga progredierande bröstsmärta.
- TnI på 50-111-157 (således hjärtskada).
- EKO visar nu nedsatt rörlighet i inferiora hjärtväggen, EF 40-45%.

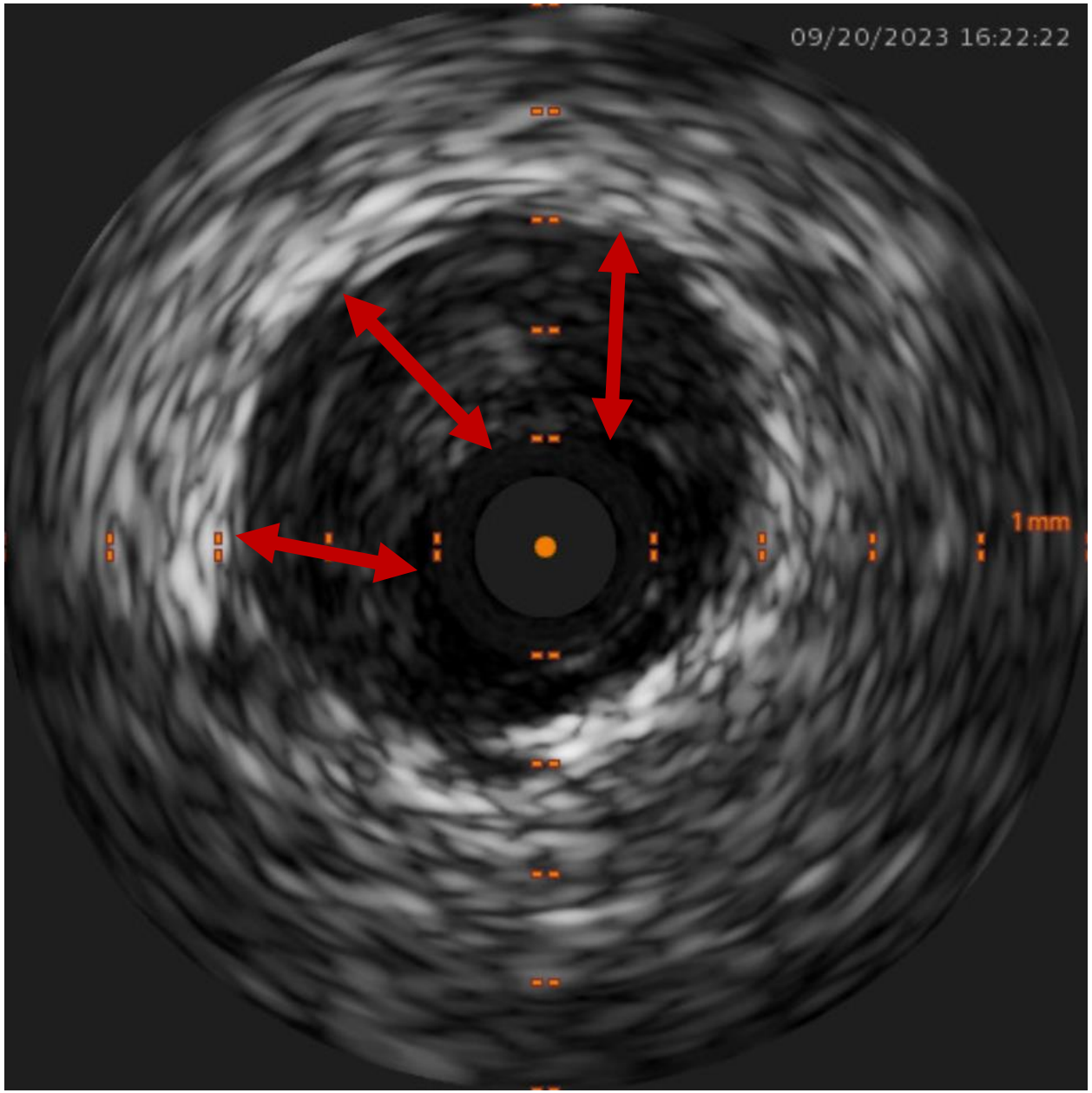
Fallbeskrivning





Fallbeskrivning - IVUS

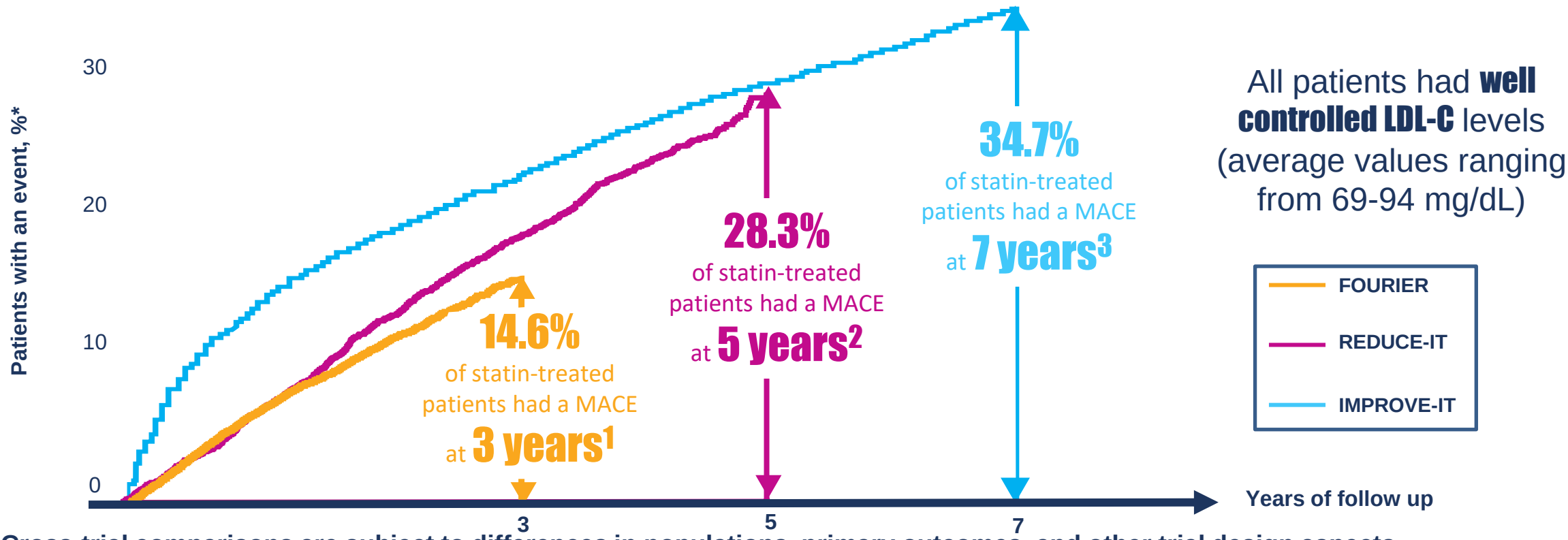




Residual kardiovaskulär risk

Even in Patients Treated With Current Standard-of-Care, P-CVR Remains High and Increases Over Time[1-3]

Several More Recent Trials Show High CV Risk with Statin-Based Standard-of-Care



All patients had **well controlled LDL-C** levels (average values ranging from 69-94 mg/dL)

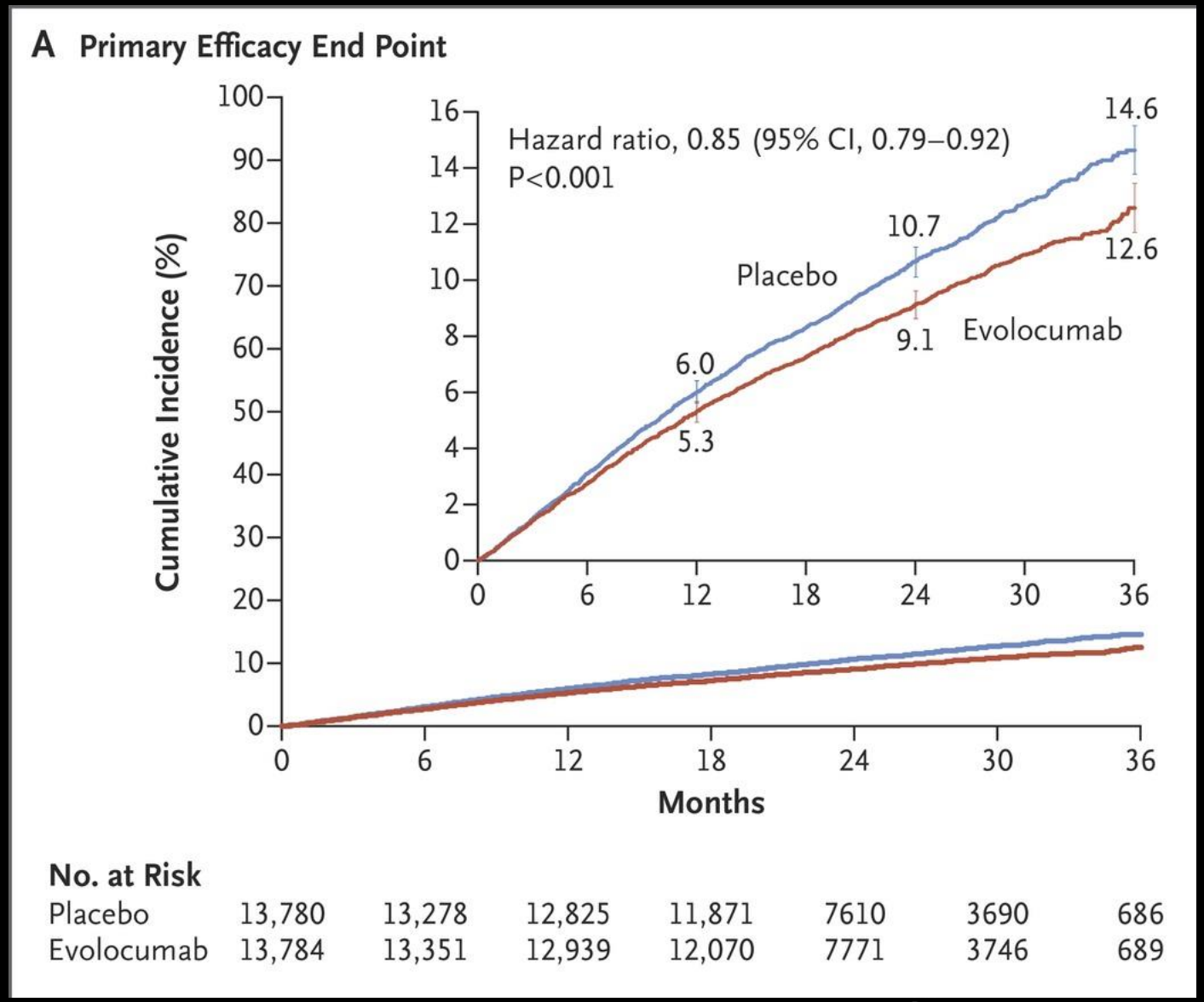
Cross-trial comparisons are subject to differences in populations, primary outcomes, and other trial design aspects

*In FOURIER, REDUCE-IT and IMPROVE-IT, MACE was defined as a 5-point composite of CV death, nonfatal MI, nonfatal stroke, hospitalization for UA, or coronary revascularization. 100% of patients were on a statin, and >80% were taking antiplatelet/anticoagulant, ACEi/ARBs, and a beta-blocker.

ACEi = angiotensin converting enzyme inhibitor; ARB = angiotensin II receptor blocker; CVOT = cardiovascular outcome trial; MACE = major adverse cardiovascular event; MI = myocardial infarction.

1. Sabatine MS, et al. *N Engl J Med.* 2017;376(18):1713-1722; 2. Bhatt DL, et al; for REDUCE-IT Investigators. *N Engl J Med*. 2019;380(1):11-22; 3. Cannon CP, et al. *N Engl J Med.* 2015;372(25):2387-2397.

Cumulative Incidence of Cardiovascular Events
FOURIER (PCSK9-hämmare)

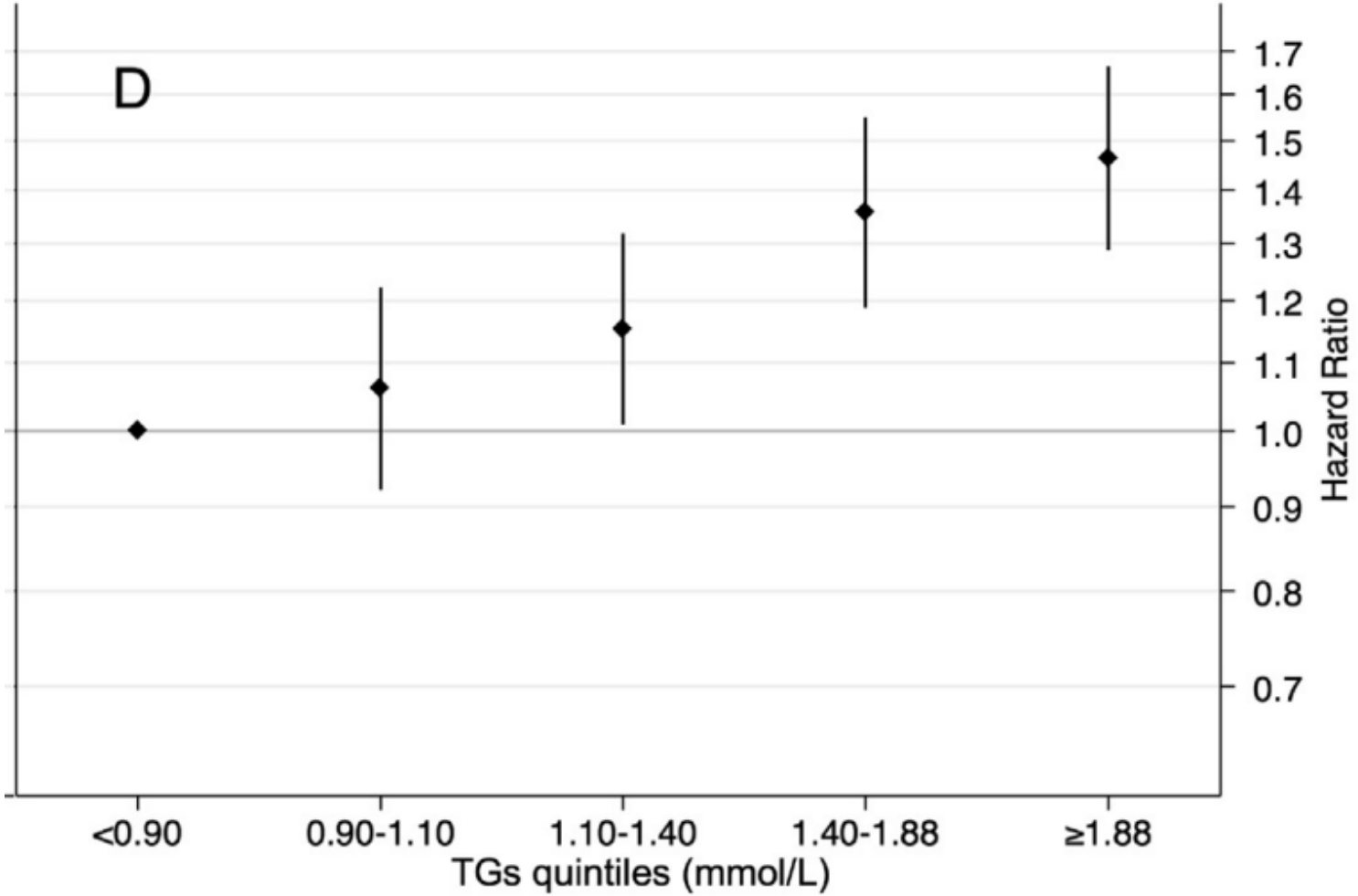


Sabatine MS et al. N Engl J Med ;376:1713-1722

The NEW ENGLAND JOURNAL of MEDICINE

1) Triglyceride

TG – a strong risk marker for new events - SWEDEHEART



International Journal of Cardiology 296 (2019) 1–7

Contents lists available at ScienceDirect

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journal homepage: www.elsevier.com/locate/ijcard

Lipid levels achieved after a first myocardial infarction and the prediction of recurrent atherosclerotic cardiovascular disease

Joel Ohm [a, b, *, 1], Paul Hjemdahl [b, c, 1], Per H. Skoglund [b, d, 1], Andrea Discacciati [e, 1], Johan Sundström [f, 1], Kristina Hambraeus [g, 1], Tomas Jernberg [h, 1], Per Svensson [i, j, 1]

[a] Function of Emergency Medicine Solna, Karolinska University Hospital, Stockholm, Sweden
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[c] Department of Clinical Pharmacology, Karolinska University Hospital, Stockholm, Sweden
[d] Center for Palliative Care, Stiftelsen Stockholms Sjukhem, Stockholm, Sweden
[e] Institute of Environmental Medicine, Unit of Biostatistics, Karolinska Institutet, Stockholm, Sweden
[f] Department of Medical Sciences, Uppsala University, Uppsala, Sweden
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[h] Department of Clinical Sciences, Danderyd University Hospital, Karolinska Institutet, Stockholm, Sweden
[i] Department of Clinical Science and Education, Södersjukhuset, Karolinska Institutet, Stockholm, Sweden
[j] Department of Cardiology, Södersjukhuset, Stockholm, Sweden

SWEDEHEART
First-ever MI survivors aged ≤76 years
attending 4-14 week revisits (2005-2013)
N= 25,643
Follow up mean 4,1 yr
96,9% on statin therapy

Ohm J, Hjemdahl P, Svensson P et al, Lipid levels achieved after a first myocardial infarction and the prediction of recurrent atherosclerotic cardiovascular disease, Int. Journal of Cardiology 296 (2019) 1-7

Kan triglyceridsänkning vara en ”avenue of success”?

Original Article

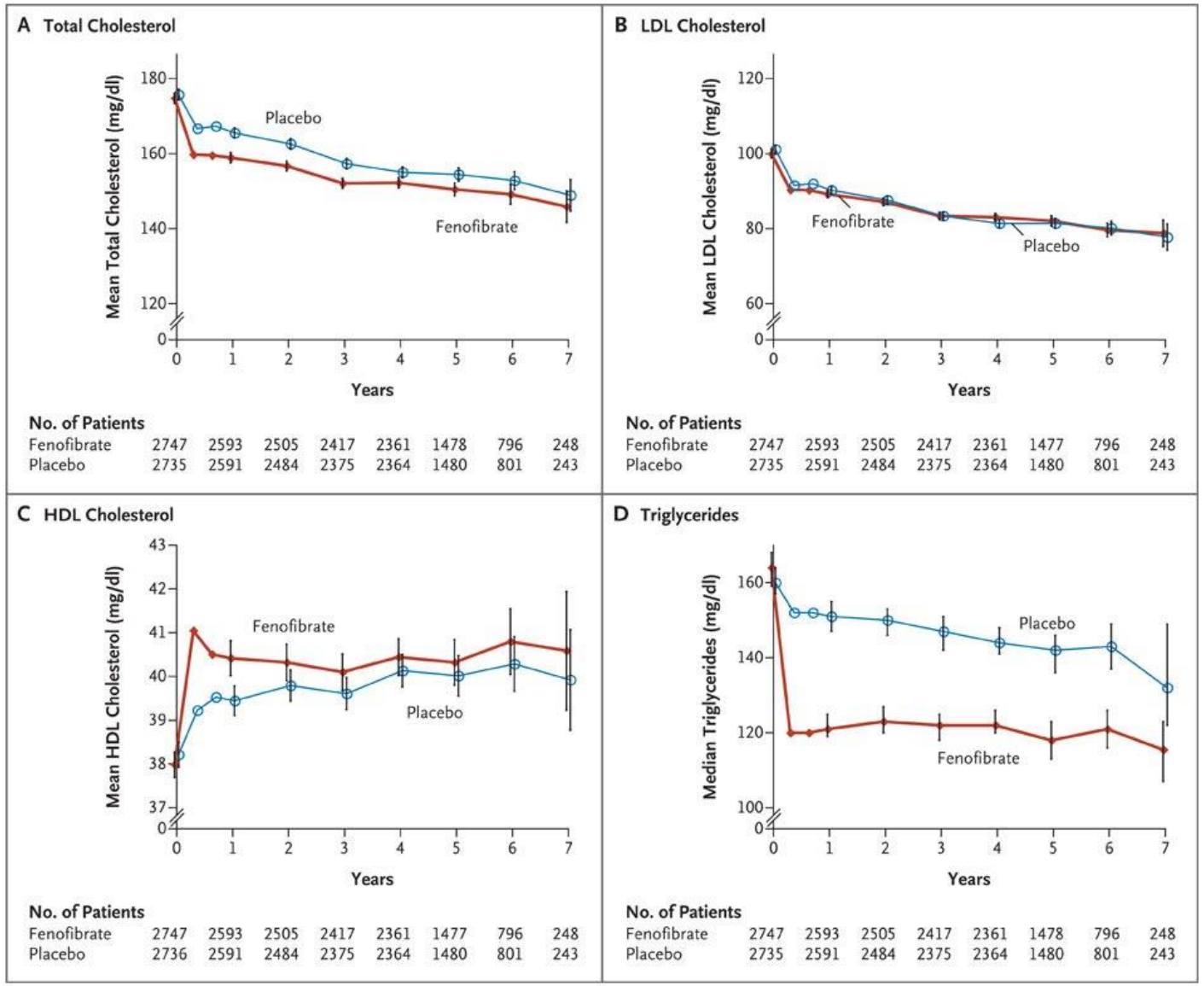
Effects of Combination Lipid Therapy in Type 2 Diabetes Mellitus

The ACCORD Study Group

N Engl J Med
Volume 362(17):1563-1574
April 29, 2010

The NEW ENGLAND JOURNAL of MEDICINE

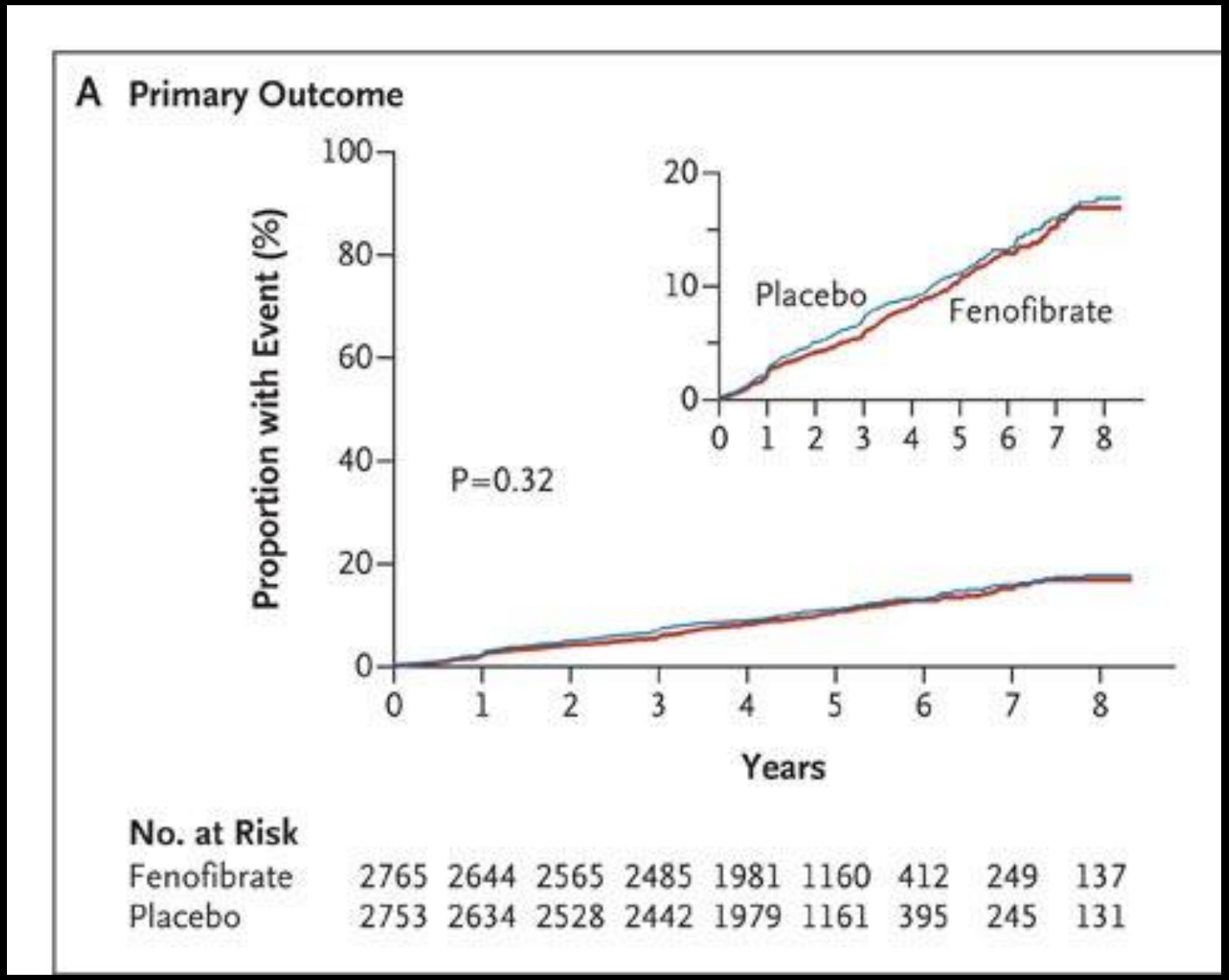
Lipid Values



The ACCORD Study Group. N Engl J Med 2010;362:1563-1574

The NEW ENGLAND JOURNAL of MEDICINE

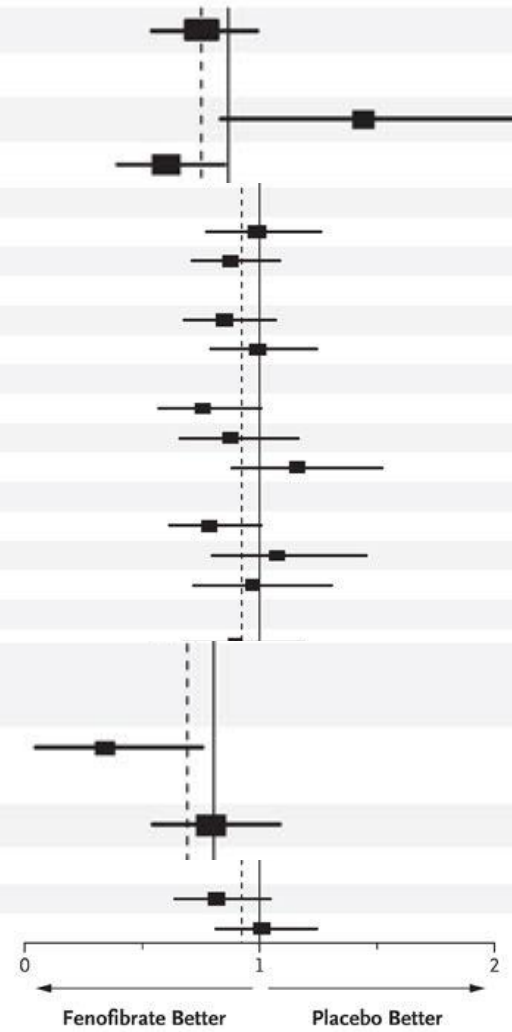
Kaplan-Meier Analyses of the Primary Outcome, Expanded Macrovascular Outcome, and Death



The ACCORD Study Group. N Engl J Med 2010;362:1563-1574

Hazard Ratios for the Primary Outcome in Prespecified Subgroups

Subgroup	Fenofibrate	Placebo	P Value for Interaction
	% of events (no. in group)		
Overall	10.52 (2765)	11.26 (2753)	
Sex			0.01
Female	9.05 (851)	6.64 (843)	
Male	11.18 (1914)	13.30 (1910)	
Previous cardiovascular disease			0.45
No	7.29 (1757)	7.34 (1745)	
Yes	16.17 (1008)	18.06 (1008)	
Glycemia group			0.36
Standard therapy	10.14 (1391)	11.61 (1370)	
Intensive therapy	10.92 (1374)	10.92 (1383)	
LDL cholesterol			0.12
≤84 mg/dl	9.38 (938)	12.23 (891)	
85–111 mg/dl	9.85 (934)	11.17 (922)	
≥112 mg/dl	12.43 (877)	10.57 (927)	
HDL cholesterol			0.24
≤34 mg/dl	12.24 (964)	15.56 (906)	
35–40 mg/dl	10.12 (860)	9.47 (866)	
≥41 mg/dl	9.08 (925)	8.99 (968)	
Triglycerides			0.64
Triglyceride–HDL cholesterol combination			0.06
Triglyceride ≥204 mg/dl and HDL ≤34 mg/dl	12.37 (485)	17.32 (456)	
All others	10.11 (2264)	10.11 (2284)	
Glycated hemoglobin			0.20
≤8.0%	8.69 (1324)	10.56 (1335)	
≥8.1%	12.20 (1435)	11.94 (1415)	



Does TG lowering reduce CV risk?
PROMINENT Study:
Pemafibrate Did Not Reduce Risk of Cardiovascular Events

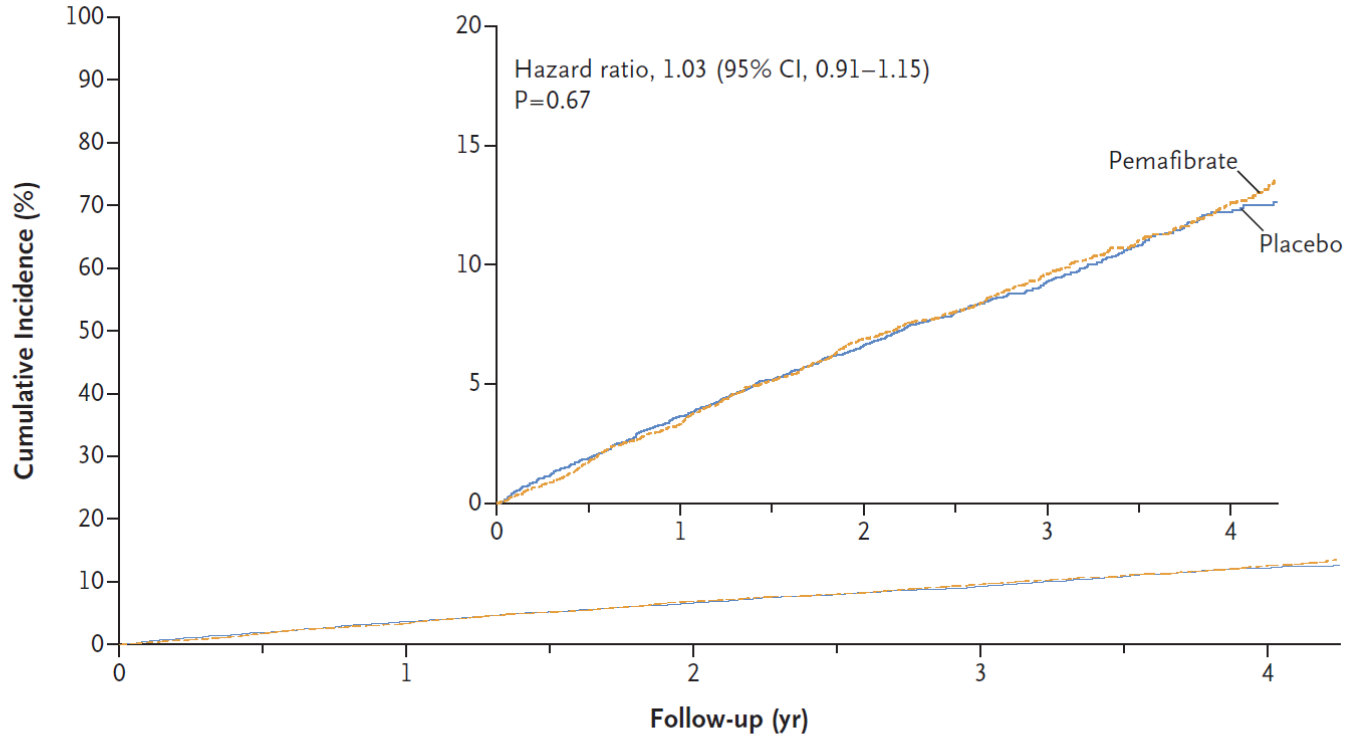


Figure 1. Cumulative Incidence of Cardiovascular Events.

Shown are Kaplan–Meier event curves for the primary trial end point of myocardial infarction, ischemic stroke, coronary revascularization, or death from cardiovascular causes. The inset shows the same data on an expanded y axis.

eks duration

(if secondary prevention

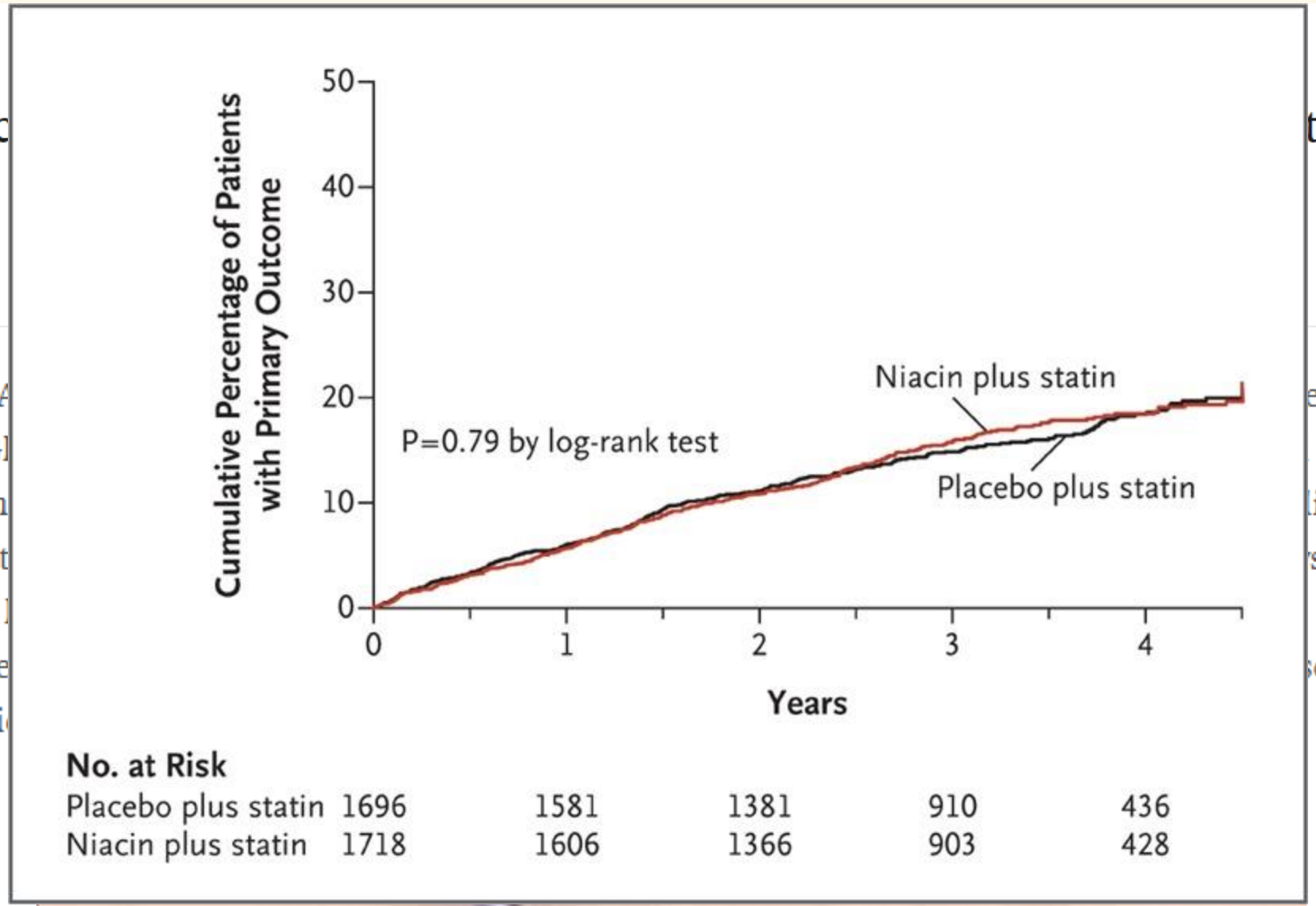
5 mmol/L)

n regimen or ≤ 100 mg/dL

Das Pradhan A. et al, Triglyceride Lowering with Pemafibrate to Reduce Cardiovascular Risk, NEJM 2022 (Online)

Niac tensive

The A es: Impact
on Gl to
inten liovascular
event slipidemia
(low
chole sed risk for
cardi

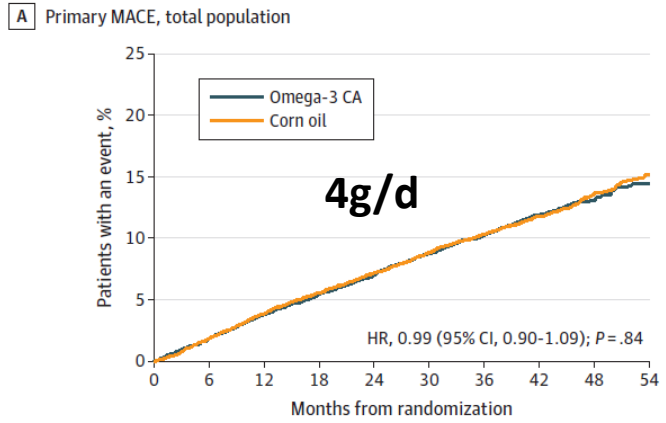


Fiskolja- Omega-3 - EPA – REDUCE-IT

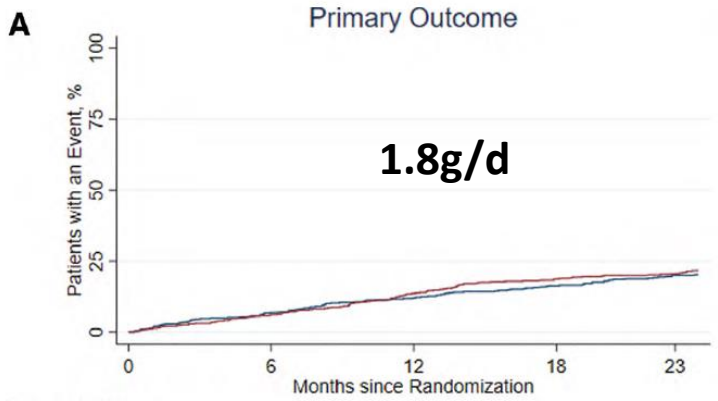
Utfallsstudier med “Omega-3” (mix av EPA+DHA)

Primary Efficacy Outcome:

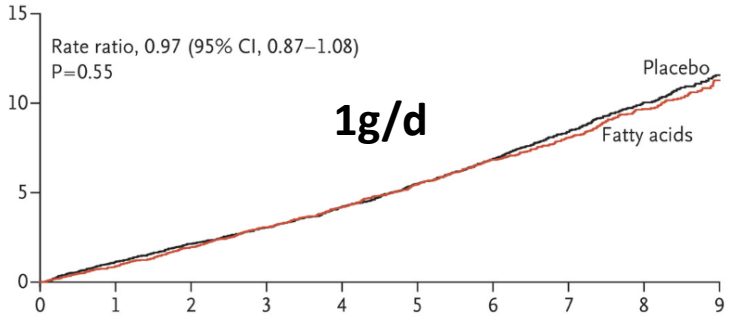
STRENGTH



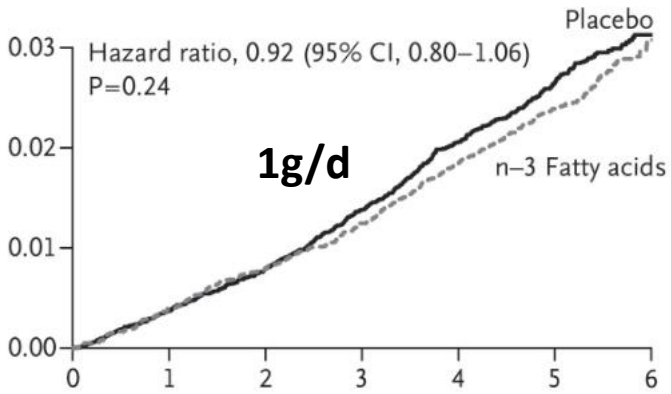
OMEMI



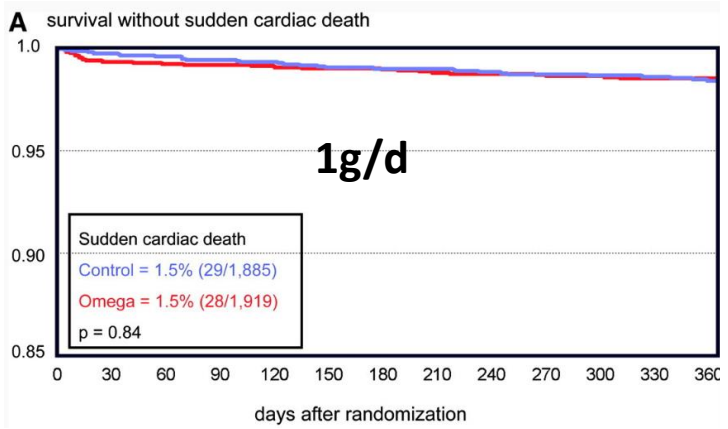
ASCEND



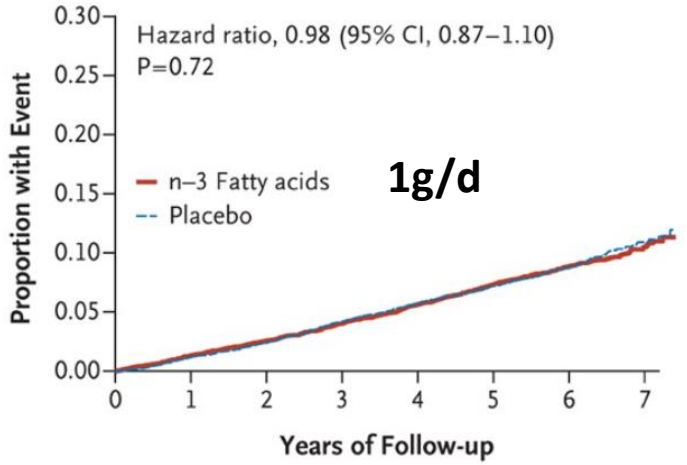
VITAL



OMEGA

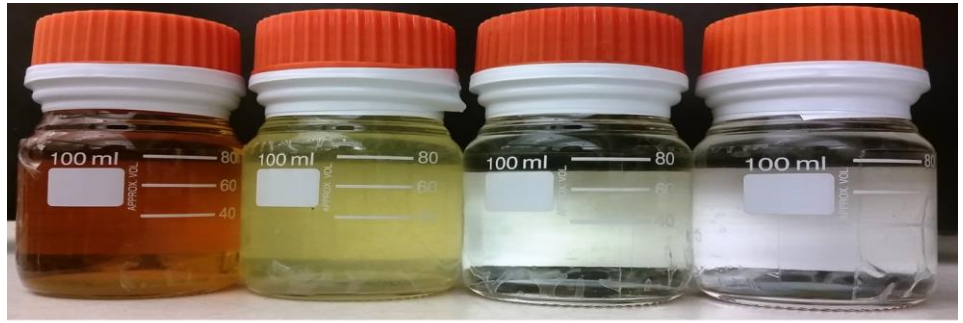


ORIGIN



Rauch et al. Circulation, 2010, OMEGA, Bosch et al. N Engl J Med, 2012, ORIGIN, Bowman et al. N Engl J Med, 2018, ASCEND, Manson et al. N Engl J Med, 2019, VITAL, Kalstad et al. Circulation, 2020, OMEMI, Nicholls et al. JAMA, 2020, STRENGTH.

Manufacturing



Majority of Fish Oil Supplements* EPA 18% DHA 12%

VAZKEPA® (Icosapent Ethyl) ≥EPA 96% DHA Not Detected

*Based on Fish Oil Capsules containing 18% EPA, 12% DHA, and 70% other undisclosed fatty acids

Fish Oil Capsule

Dietary Supplement

Encapsulation

Harvesting, Early Treatment

CRUDE FISH OIL
12% DHA
70% Other
18% EPA

Ethyl Esterification

CRUDE FISH OIL
12% DHA-EE
70% Other
18% EPA-EE

Initial Purification

DHA-EE + Other
45-60% EPA-EE

Intermediate Purification

DHA-EE + Other
75-90% EPA-EE

Final Purification

Icosapent Ethyl (≥96% EPA-EE)

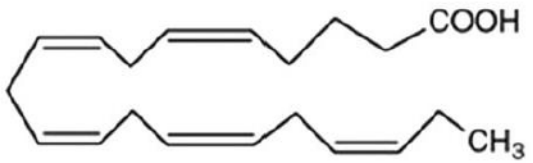
IPE IPE

PURITY

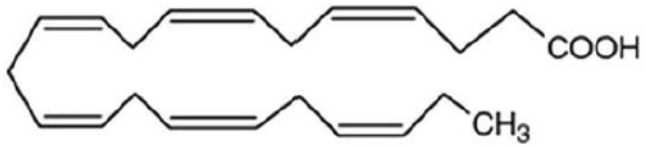
All percents are nominal concentrations based on fish source.

Data on file, Amarin Pharma, inc; 2018
Vazkepa, SmPC Current version

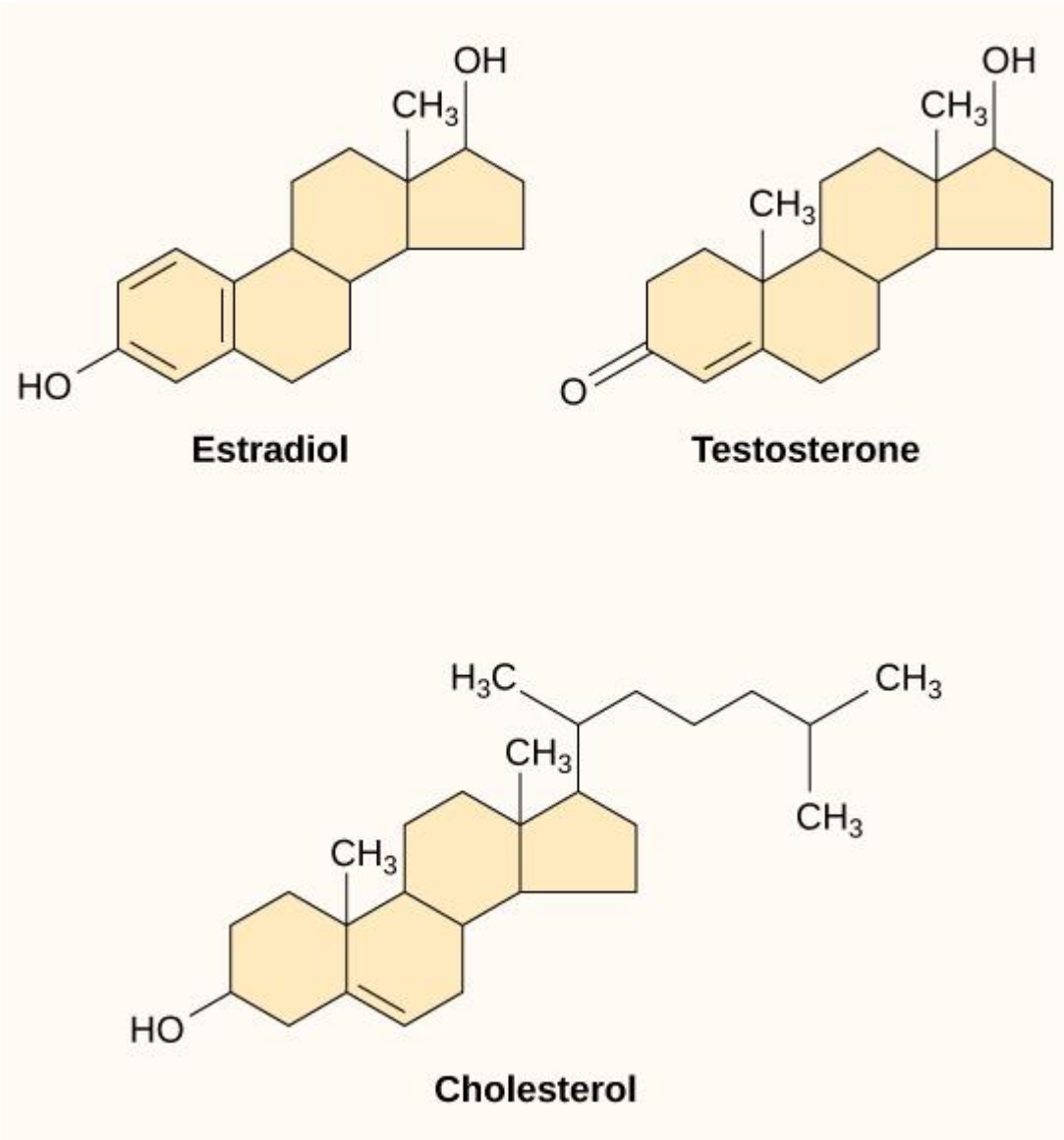
EPA (nytta) vs DHA (skada)



EICOSAPENTANOIC ACID (EPA)



DOCOSAHEXAENOIC ACID (DHA)



REDUCE-IT evaluated statin-treated patients with well-controlled LDL-C and multiple CV risk factors[1,2]

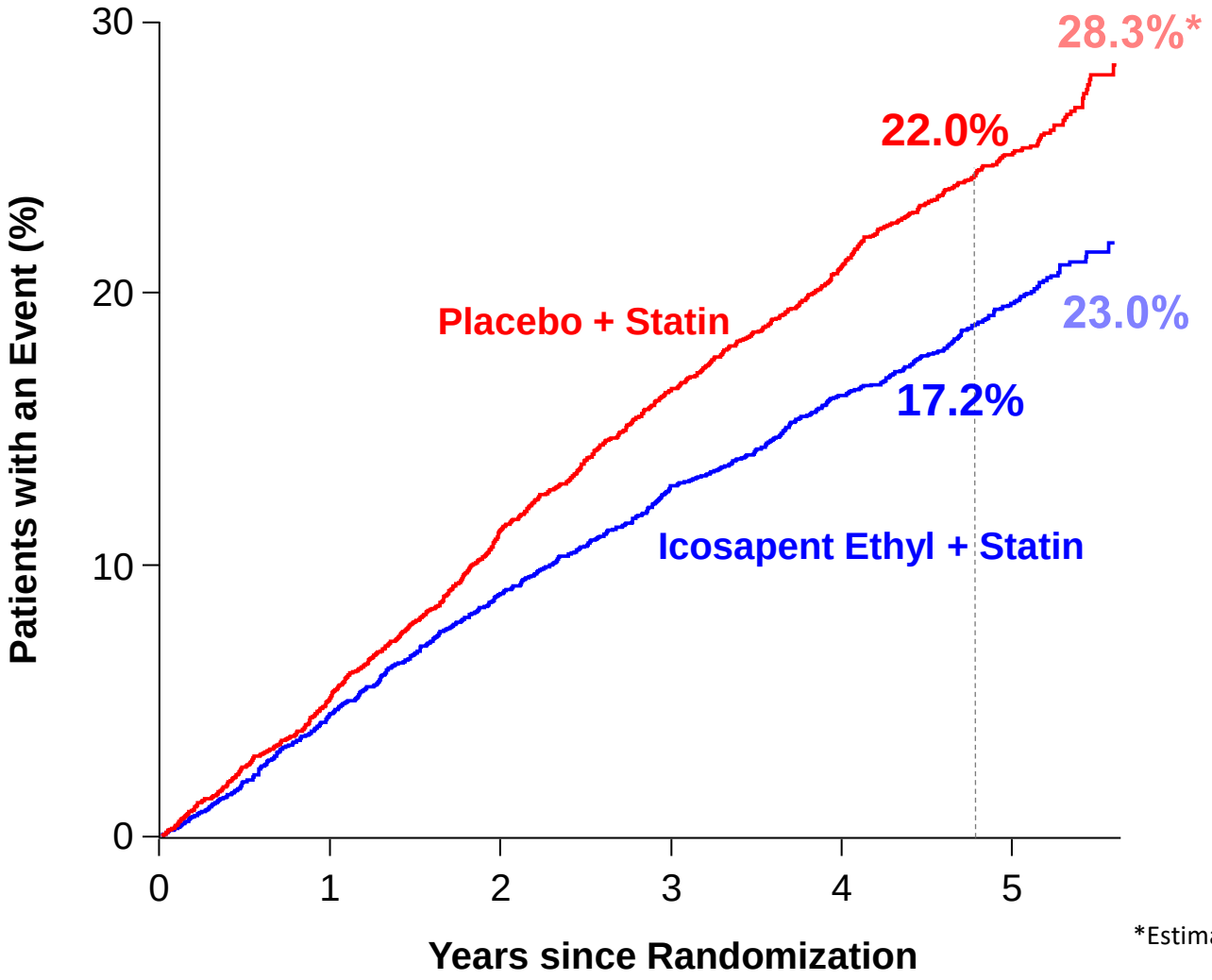
PATIENTS	AT RISK FOR	RANDOMISATION
8179 Patients… ▪ Men and women aged ≥45 years on stable statin therapy +/- ezetimibe ▪ LDL-C 41-100 mg/dL (1.06–2.59 mmol/L); median at baseline 75 mg/dL (1.94 mmol/L)	**At High Risk for CV Events due to:** **Mild to moderate TG elevation** ▪ TG 135 – 499 mg/dL (1.52–5.63 mmol/L); median at baseline 216 mg/dL (2.44 mmol/L) **- AND -** ▪ Established CVD (secondary prevention cohort) **- OR -** ▪ Diabetes mellitus + age ≥50 years + at least 1 risk factor for CVD (primary prevention cohort)	**Randomisation 1:1** **Study Treatment** ▪ Stable statin + IPE 4 g/day (2 g twice daily with meals) **- OR -** ▪ Stable statin + placebo

CV: cardiovascular; CVD: cardiovascular disease; IPE: icosapent ethyl; LDL-C: low density lipoprotein-cholesterol; REDUCE-IT: Reduction of CV Events with Icosapent Ethyl-Intervention Trial; TG: triglycerides.

1. Bhatt DL, et al. *Clin Cardiol*. 2017;40(3). **2**. Bhatt DL, et al. *N Engl J Med*. 2019;380(1).

Primary End Point:

CV Death, MI, Stroke, Coronary Revasc, Unstable Angina



Hazard Ratio, 0.75
(95% CI, 0.68–0.83)

RRR = 24.8%

ARR = 4.8%

NNT = 21 (95% CI, 15–33)

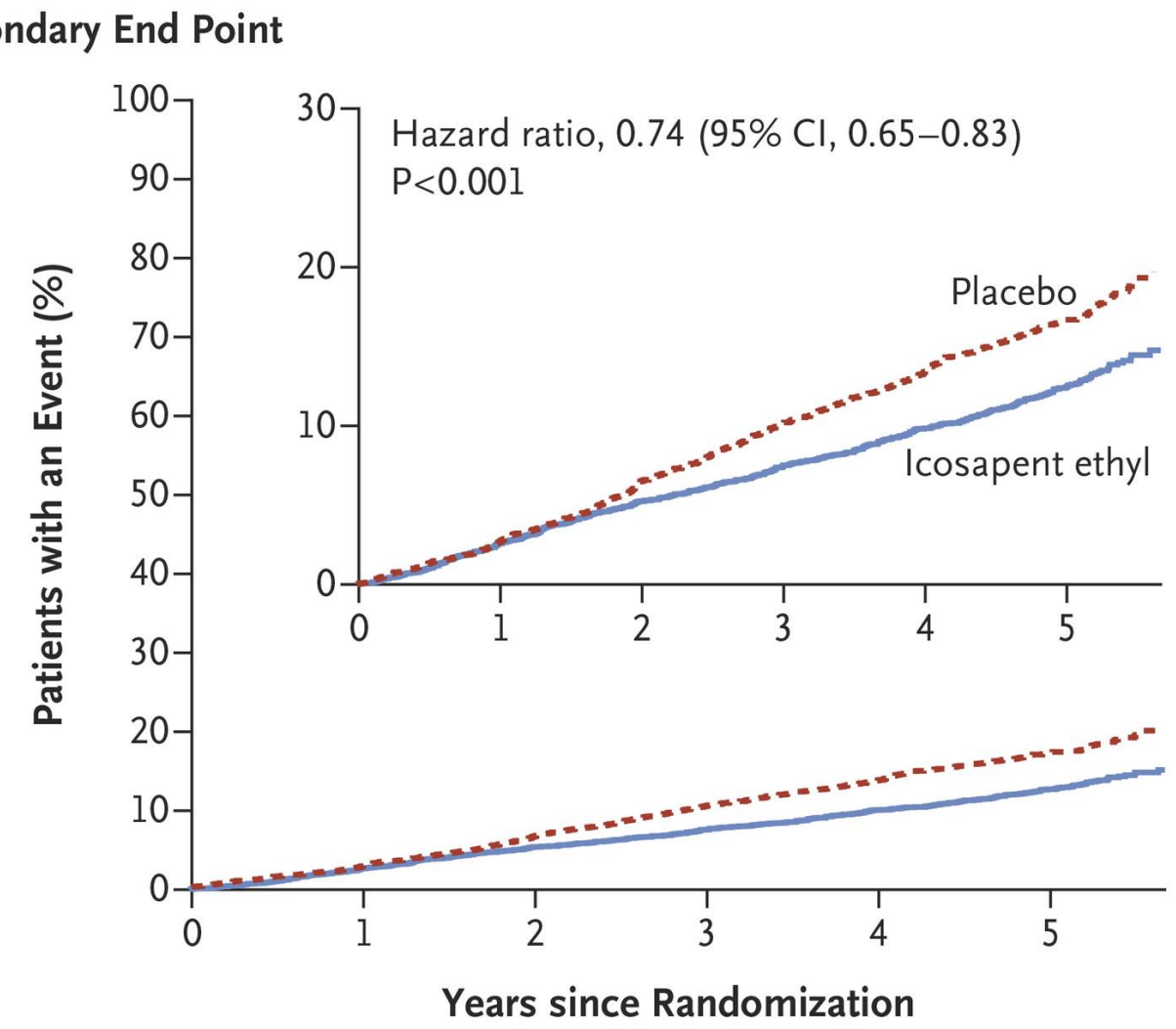
P=0.00000001

*Estimated Kaplan-Meier event rate at approximately 5.7 years

*Presented at the Endocrinologic and Metabolic Drugs Advisory Committee FDA Meeting, November 14, 2019. https://www.fda.gov/media/132768/download
Modified from D. Bhatt et al. N Engl J Med 2019;380:11-22.

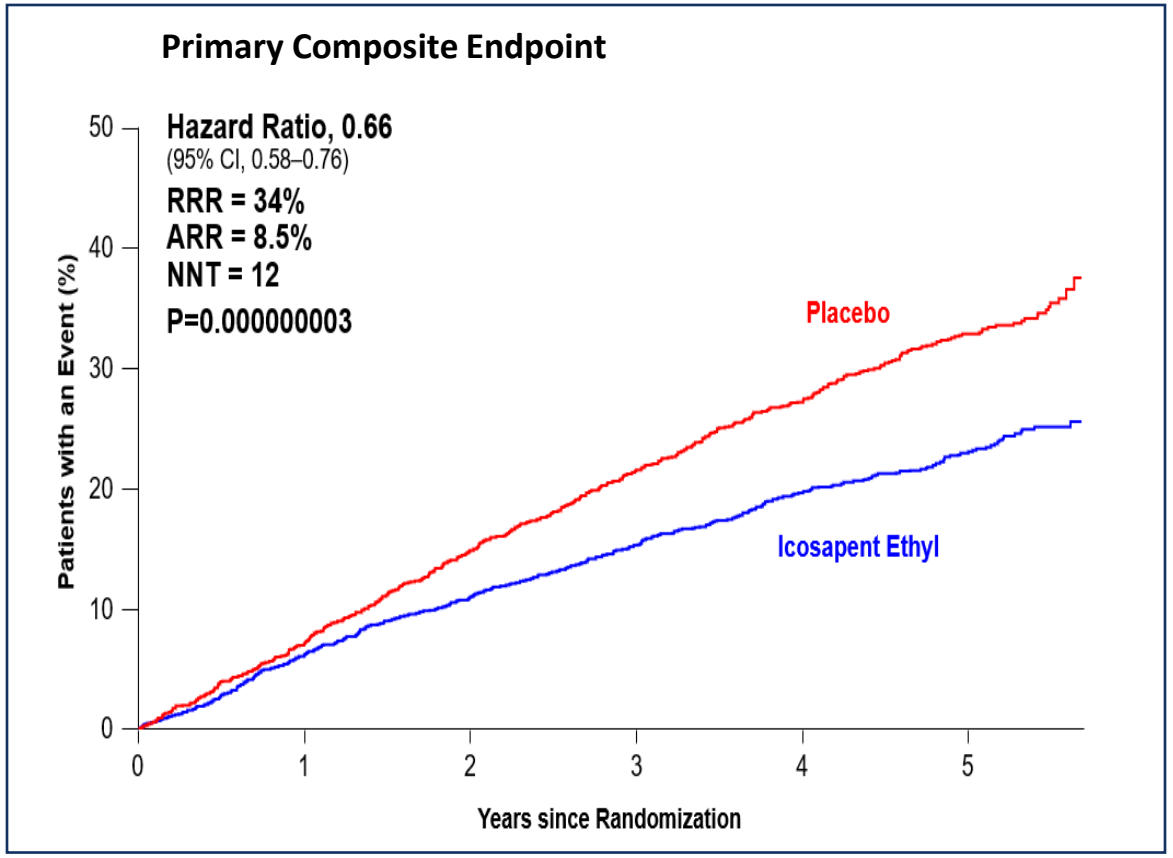
Secondary End Point:

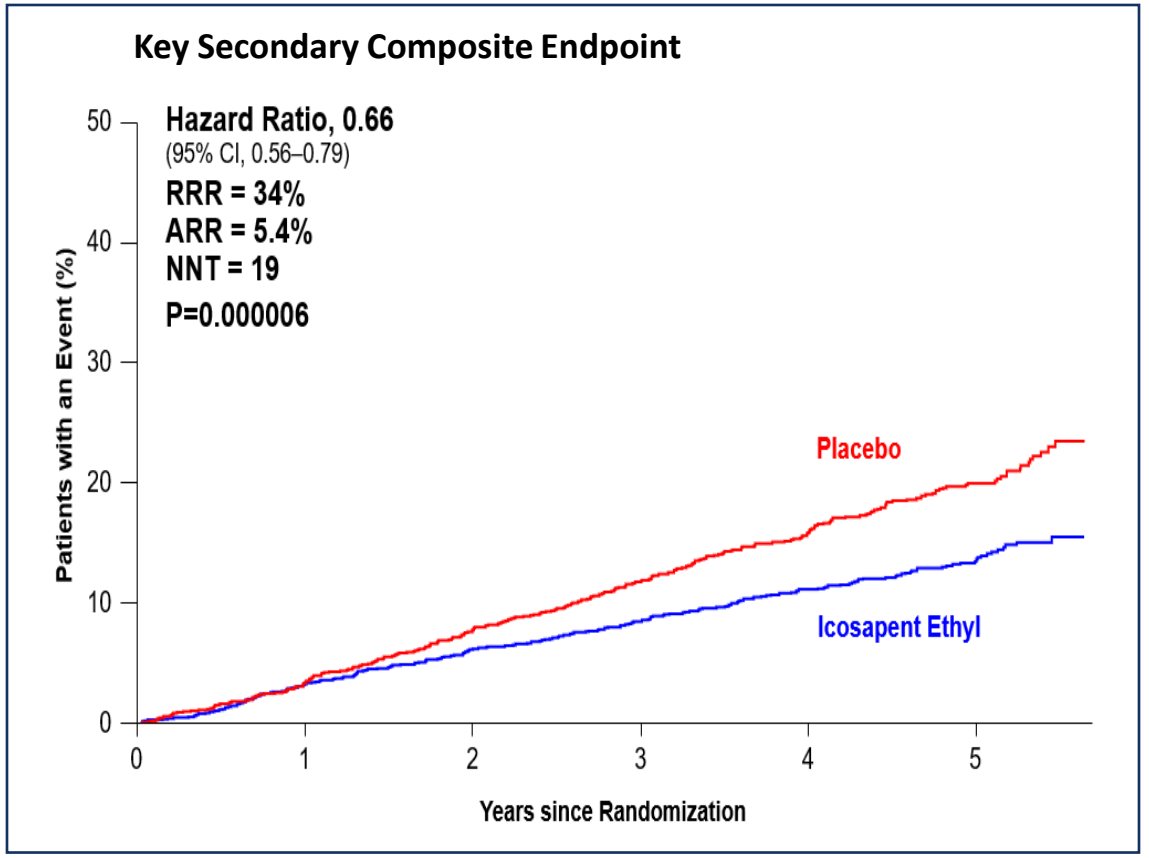
CV Death, MI, Stroke



D. Bhatt et al. N Engl J Med 2019;380:11-22.

Patients with a history of PCI* (N=3408) : Icosapent ethyl reduced Primary and secondary endpoints





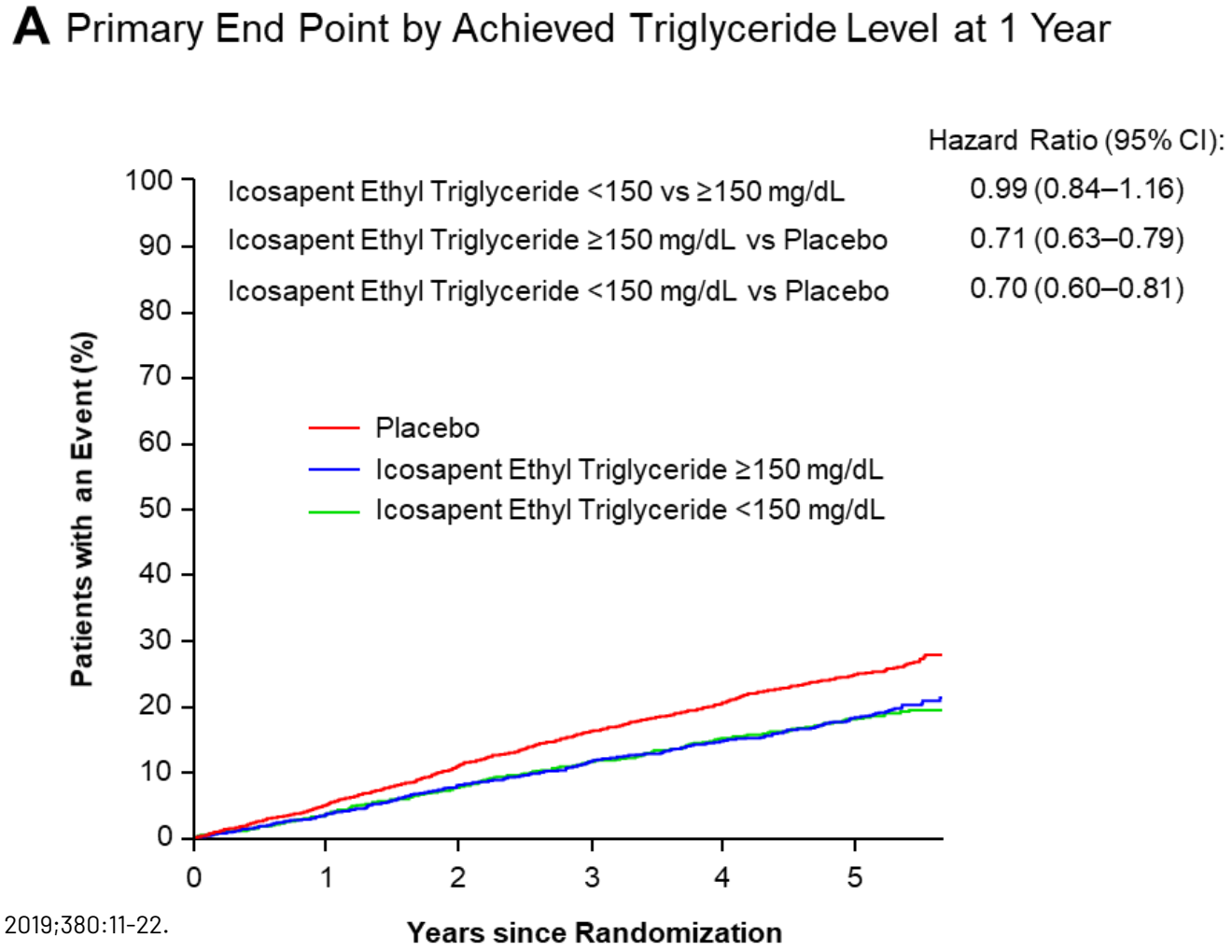
* Post hoc analysis

Peterson BE et al. JAHA 2022;11.

Correlation between TG respons to cardiovascular effect?

"TG reduction appears to provide only a minor contribution to the reduction in risk of cardiovascular events with icosapent ethyl" –EPAR

In REDUCE-IT, no association was seen between baseline or achieved levels of TG or LDL and CV risk reduction, but with steady state EPA levels in serum.



AMARIN D. Bhatt et al. N Engl J Med 2019;380:11-22.

CV benefit of IPE independent of baseline TG levels

Primary endpoint in subgroups*	Icosapent ethyl n/N (%)	Placebo n/N (%)	HR (95%CI)	Int P value
Baseline TGs				
• ≥200 mg/dL (2.3 mmol/L)	430/2481 (17.3%)	559/2469 (22.6%)	0.73 (0.64–0.83)	0.45
• <200 mg/dL (2.3 mmol/L)	275/1605 (17.1%)	342/1620 (21.1%)	0.79 (0.67–0.93)	
Baseline TGs				
• ≥150 mg/dL (1.7 mmol/L)	640/3674 (17.4%)	811/3660 (22.2%)	0.75 (0.68–0.83)	0.83
• <150 mg/dL (1.7 mmol/L)	65/412 (15.8%)	90/429 (21.0%)	0.79 (0.57–1.09	
Baseline TGs ≥200 mg/dL (2.3 mmol/L) and HDL ≤35mg/dl (0.9 mmol/l)				
• Yes	149/823 (18.1%)	214/794 (27.0%)	0.62 (0.51–0.77)	0.04
• No	554/3258 (17.0%)	687/3293 (20.9%)	0.79 (0.71–0.88)	

* Prespecified subgroups

Bhatt DL et al. *N Engl J Med.* 2019;380(1)

CV benefit of IPE independent of baseline LDL-C levels

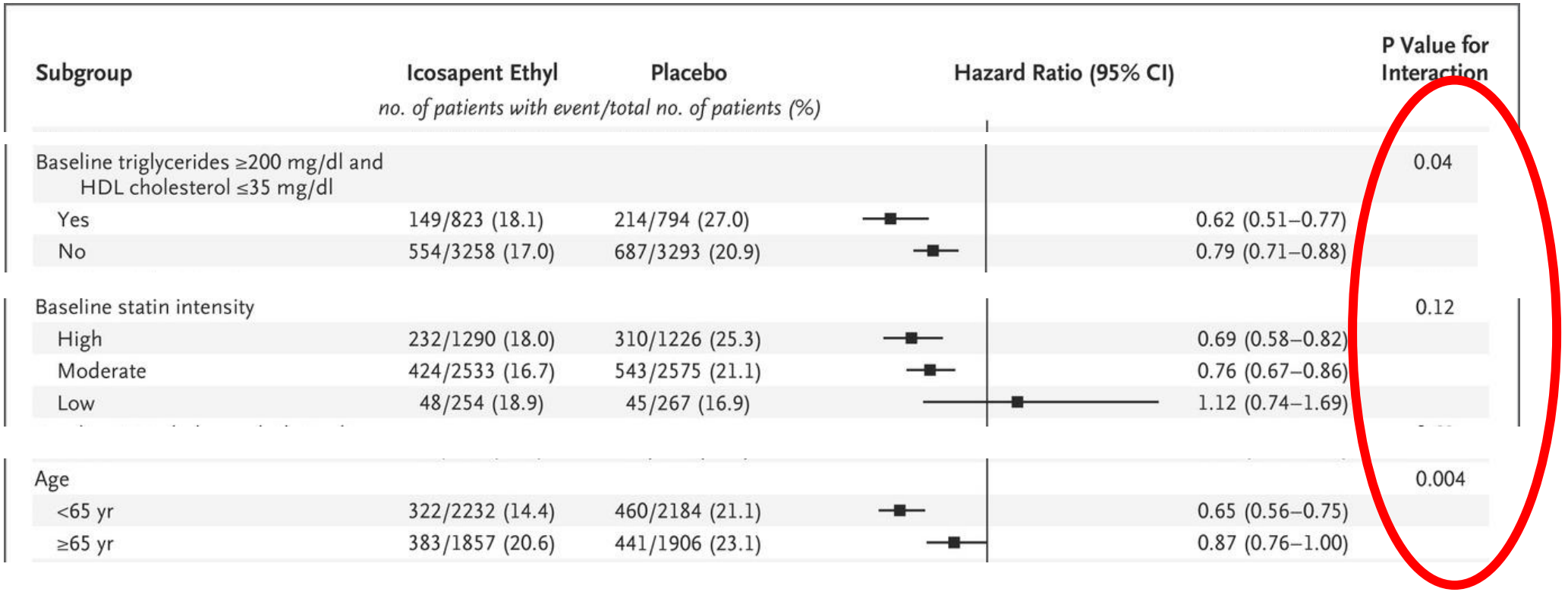
Primary endpoint in subgroups*	Icosapent ethyl n/N (%)	Placebo n/N (%)	HR (95%CI)	Int P value
Baseline LDL-C				
• ≤67 mg/dL (1.73 mmol/L)	244/1481 (16.5%)	302/1386 (21.8%)	0.72 (0.61–0.85)	0.62
• >67 and ≤84 mg/dL (1.73 and ≤2.17 mmol/L)	248/1347 (18.4%)	307/1364 (22.5%)	0.81 (0.68–0.96)	
• >84 mg/dL (2.17 mmol/L)	213/1258 (16.9%)	292/1339 (21.8%)	0.74 (0.62–0.89)	

CV benefit for IPE reported even among patients in the lowest baseline LDL-C tertile, indicating that CV benefit associated with IPE was independent of LDL-C levels

* Prespecified subgroups

Bhatt DL et al. *N Engl J Med.* 2019;380(1)

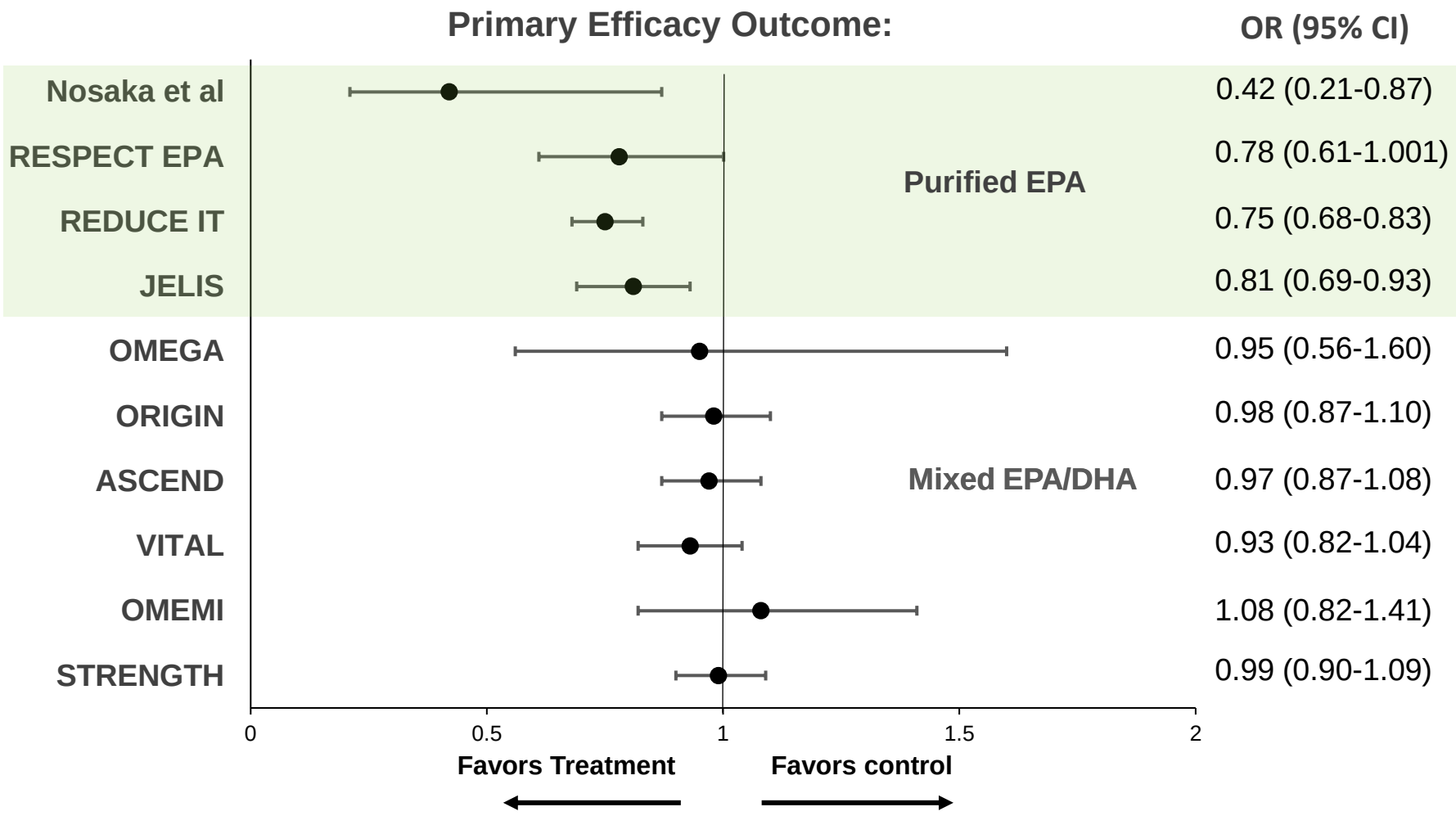
Primary Endpoint Across Baseline Subgroups



Subgroup	Icosapent Ethyl	Placebo	Hazard Ratio (95% CI)	P Value for Interaction
	no. of patients with event/total no. of patients (%)			
Baseline triglycerides ≥200 mg/dl and HDL cholesterol ≤35 mg/dl				0.04
Yes	149/823 (18.1)	214/794 (27.0)	0.62 (0.51–0.77)	
No	554/3258 (17.0)	687/3293 (20.9)	0.79 (0.71–0.88)	
Baseline statin intensity				0.12
High	232/1290 (18.0)	310/1226 (25.3)	0.69 (0.58–0.82)	
Moderate	424/2533 (16.7)	543/2575 (21.1)	0.76 (0.67–0.86)	
Low	48/254 (18.9)	45/267 (16.9)	1.12 (0.74–1.69)	
Age				0.004
<65 yr	322/2232 (14.4)	460/2184 (21.1)	0.65 (0.56–0.75)	
≥65 yr	383/1857 (20.6)	441/1906 (23.1)	0.87 (0.76–1.00)	

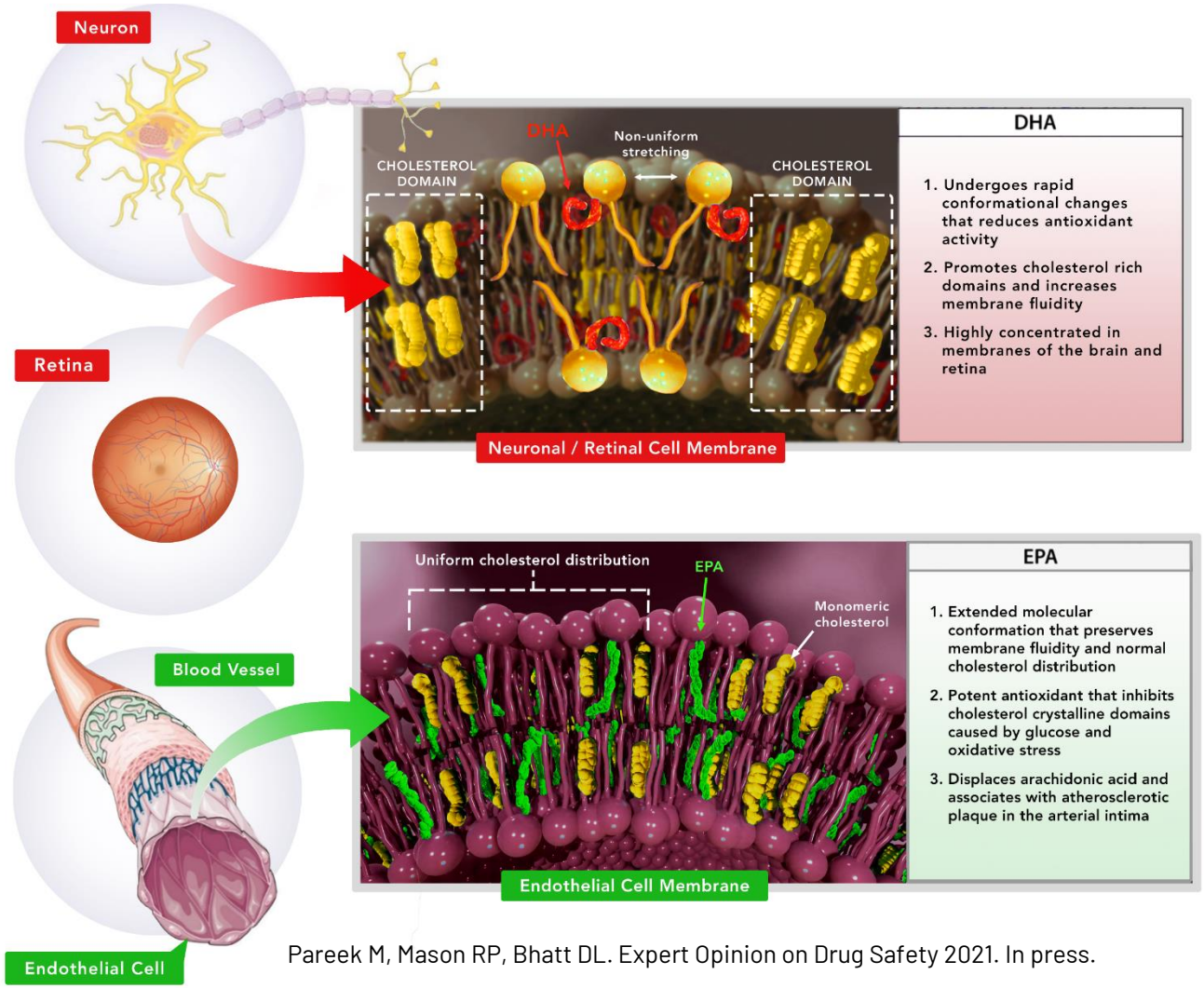
EPA vs DHA

Recent CVOTs with EPA and EPA/DHA mixtures on top of statins



Bernhard Rauch et al. Circulation, 2010, OMEGA, Jackie Bosch et al. N Engl J Med , 2012, ORIGIN, Louise Bowman et al. N Engl J Med, 2018, ASCEND, JoAnn E. Manson et al. N Engl J Med, 2019, VITAL, Are A Kalstad et al. Circulation, 2020, OMEMI, Nicholls et al. JAMA, 2020, STRENGTH, Yokoyama, et al. Lancet, 2007 , JELIS, Bhatt et al. N Engl J Med, 2019, REDUCE-IT, Budoff et al. Eur Heart J, 2020, EVAPORATE, Daida, Presented at AHA, Chicago 2022, RESPECT-EPA, Nosaka et al. Intyernational Journal of Cardiology 228 (2017) 173–179

Contrasting Effects of DHA and EPA



Mechanism of Action	EPA	DHA
Does not raise LDL in pts with very high TGs[1,2,3]	+	–
Reduces hsCRP in patients with elevated TGs[4,5,6]	+	–
Maintains membrane cholesterol distribution[7]	+	–
Preserves membrane stability[7,8]	+	–
Inhibits cholesterol domains[9,10]	+	–
Enhances endothelial function with statin[11]	+	–
Inhibits sdLDL, LDL, VLDL oxidation[9,10,12]	+	–
Inhibits HDL oxidation[13]	+	–

Pareek M, Mason RP, Bhatt DL. Expert Opinion on Drug Safety 2021. In press.

AMARIN

1Bays HE et al. Am J Cardiol.2011; 108:682-690; 2Jacobson T A et al. J Clin Lipidol. 2012; 6:5-18; 3Goldberg AC et al. Clin Ther.1989;11(1):69-83; 4Bays HE et al. Am J Cardiol.2013; 13:37-46; 5Dunbar RL et al. Lipids in Health and Disease. 2015; 14:98; 6Belf ort R et al. J Clin EndocrinMetabol. 2010; 95:829-836; 7Mason RP et al. Biochimicaet BiophysicaActa. 2016; 1858:3131-3140; 8Sherratt SC and RP Mason. Chem Phys Lipid. 2018; 212:73-79; 9Sherratt SC et al. Biochimicaet BiophysicaActa. 2020; 1862(7); 10Mason RP and RF Jacob. Biochimicaet BiophysicaActa.2015; 1848:502-509;; Sherratt SCR, Juliano R and Mason RP, BiochimBiophysActa doi.org/10.1016/j.bbamem.2020.183254;11Mason RP et al. Biomed Pharmacother. 2018; 103:1231-1237; 12Mason RP et al. J Cardiovasc Pharmacol. 2016; 68:33-40; 13Sherratt SC and RP Mason. BiochemBiophysRes Comm. 2018; 496:335-338. 14Jacobs et al. *Biophysical Journal* 2021;120:2317-2329

Mekanismer?

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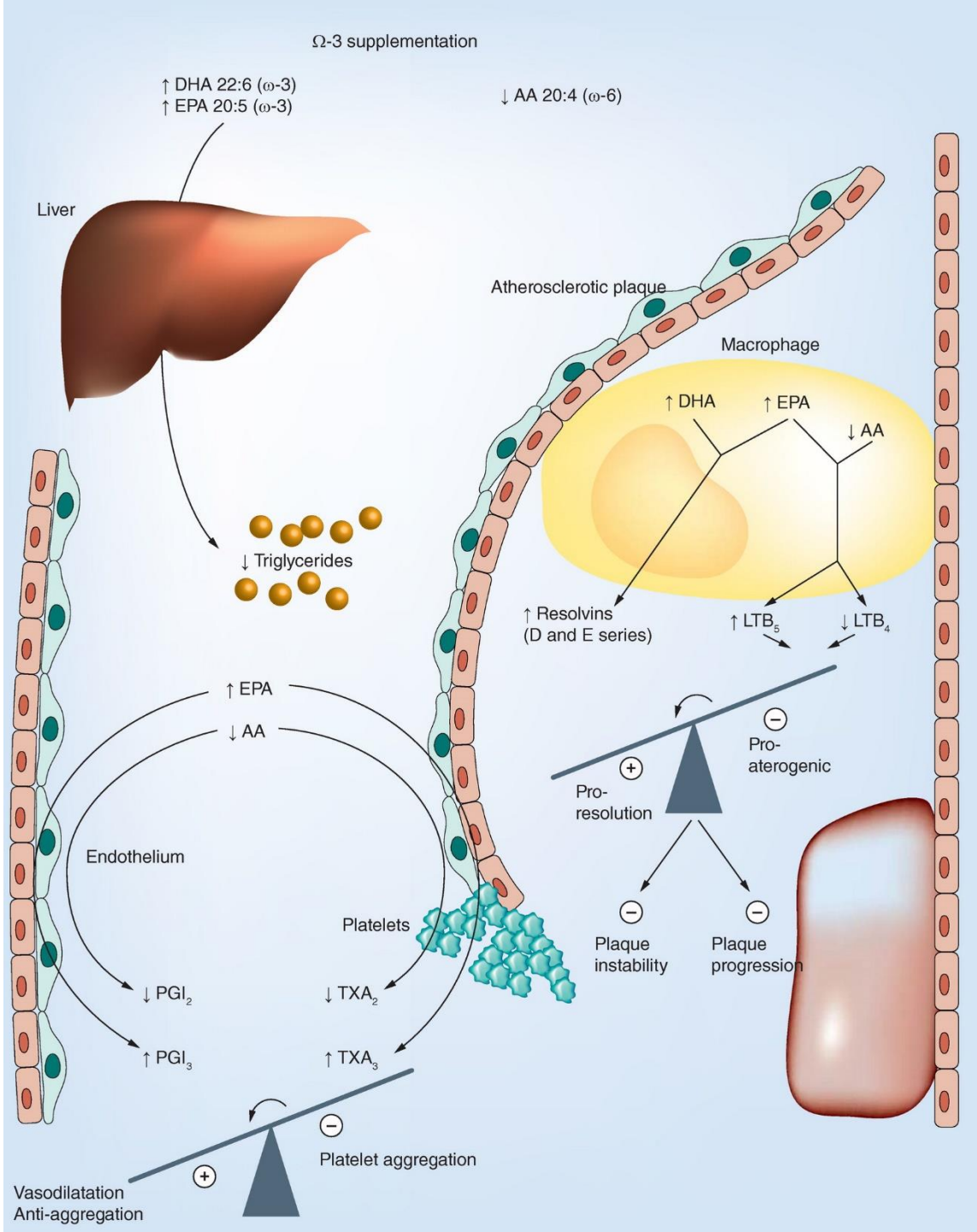

Magnus Bäck, FUTURE SCIENCE 2017, VOL. 3, NO. 4

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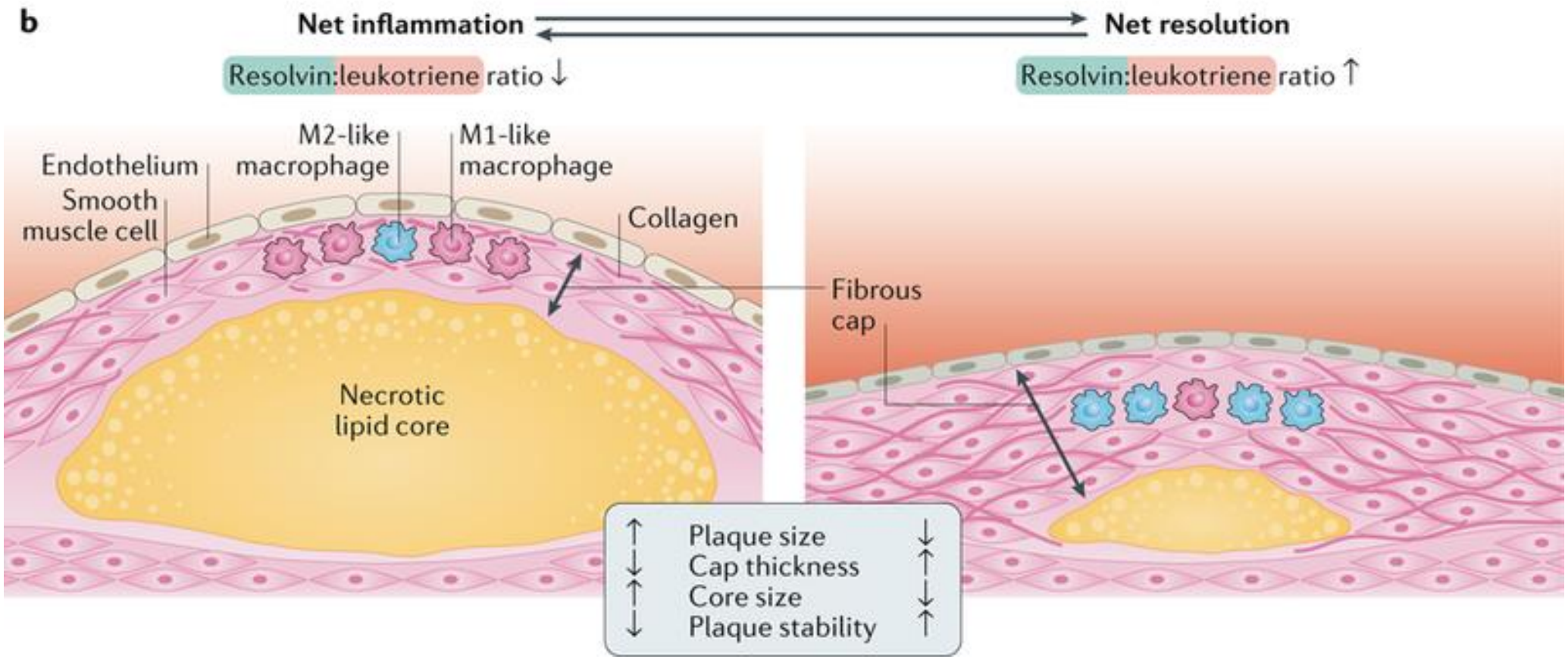

Magnus Bäck et al, NATURE REV CARDIOLOGY,2019

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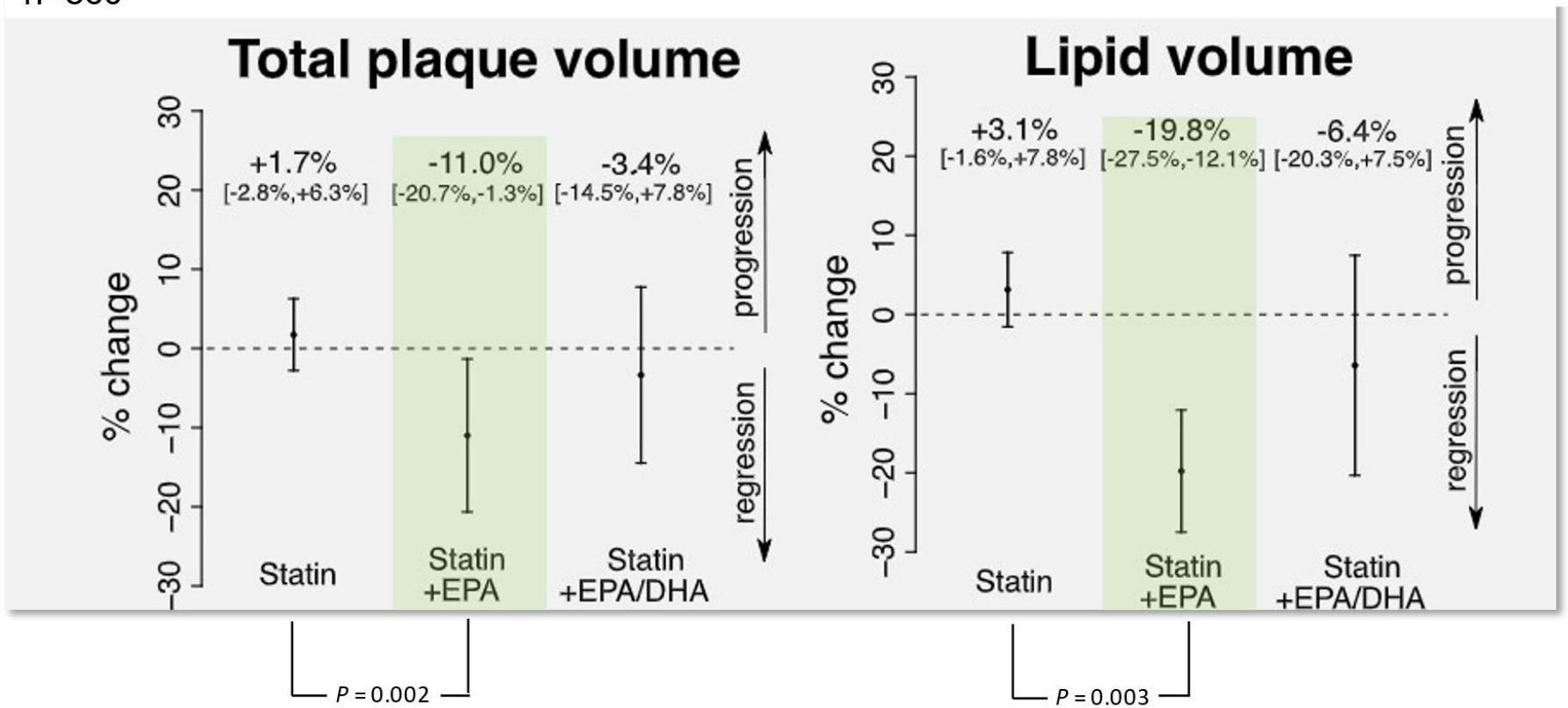
EPA vs Mixed EPA/DHA added to Statin for Reducing Plaque Volume in Coronary Atherosclerosis

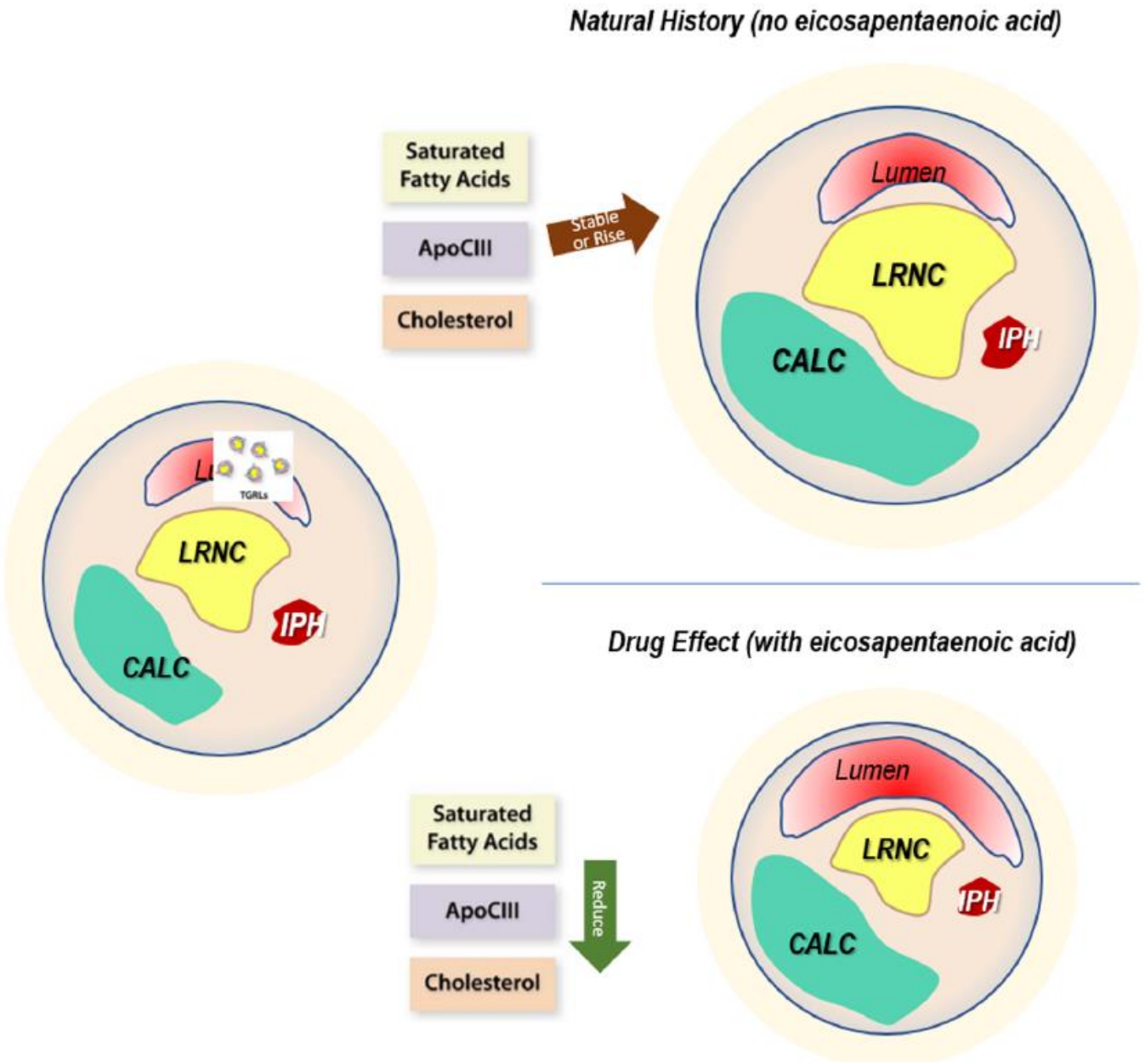
Metaanalysis of 10 prospective randomized trials
n=860



Sheppard JP, et al. Circulation. 2021;144:A10328
https://www.ahajournals.org/doi/10.1161/circ.144.suppl_1.10328

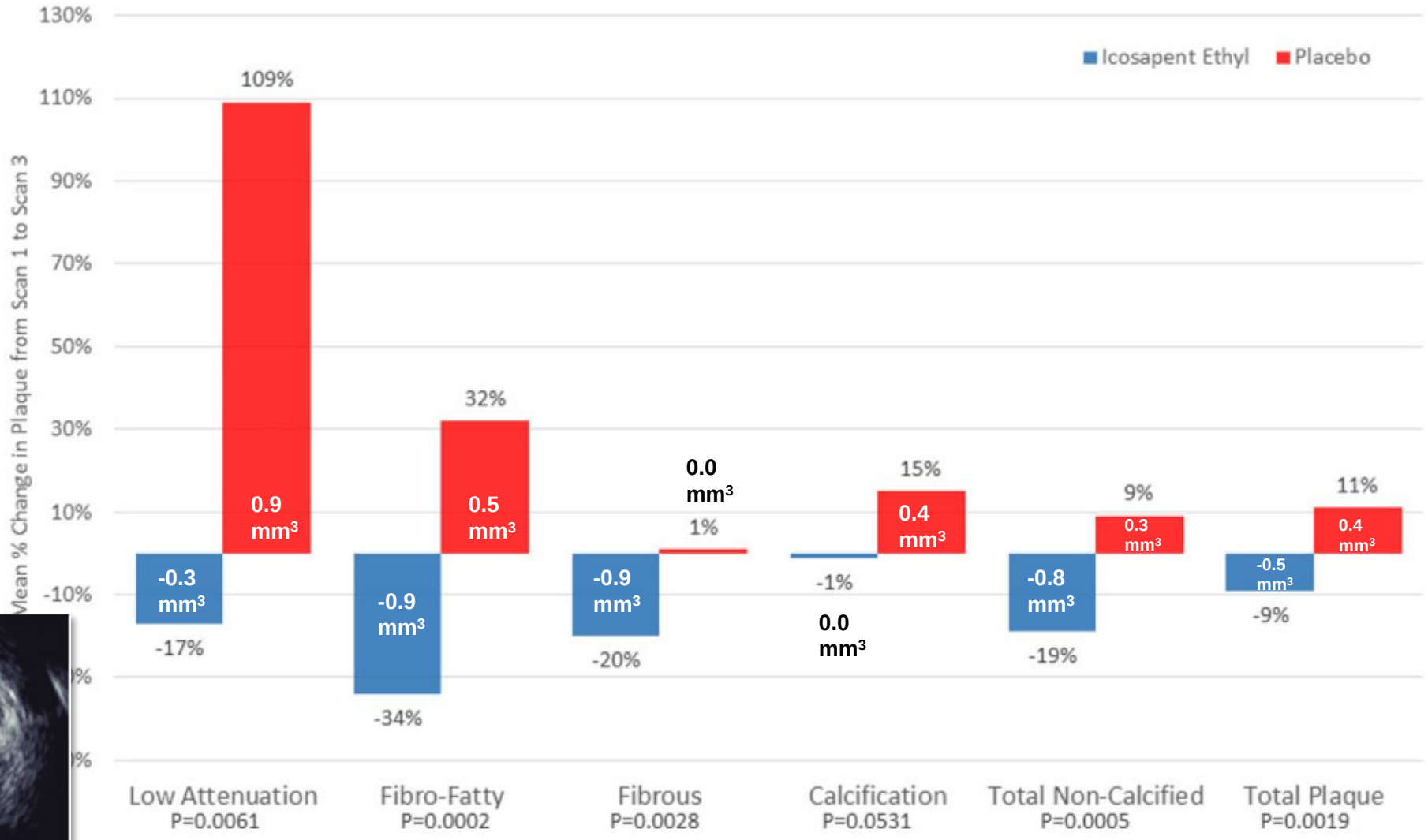
Påverkan på plack

Buckler et al, Frontiers in Cardiovascular Medicine, 2023

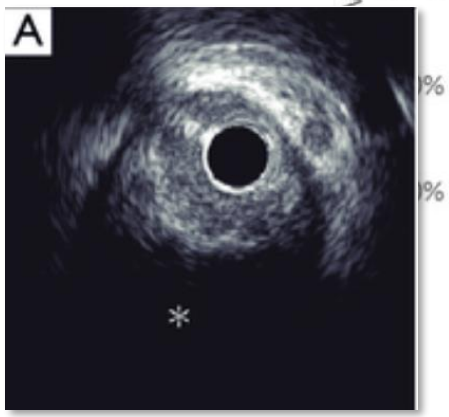


RCT, n=80

Plaque Volume Changes at 18 Months (by CT Angio)



Primary Endpoint



Budoff M, et al. *Eur Heart J.* 2020;41(40):3925-3932.

Mechanism of Action

- The mechanisms of action contributing to reduction of cardiovascular events with icosapent ethyl are not completely understood

- The mechanisms are likely multi-factorial including improved lipoprotein profile with reduction of triglyceride-rich lipoproteins, anti-inflammatory, and antioxidant effects, reduction of macrophage accumulation, improved endothelial function, increased fibrous cap thickness/stability, and antiplatelet effects

- Each of these mechanisms can beneficially alter the development, progression, and stabilisation of atherosclerotic plaque, as well as the implications of plaque rupture

Vazkepa, SmPC Current version

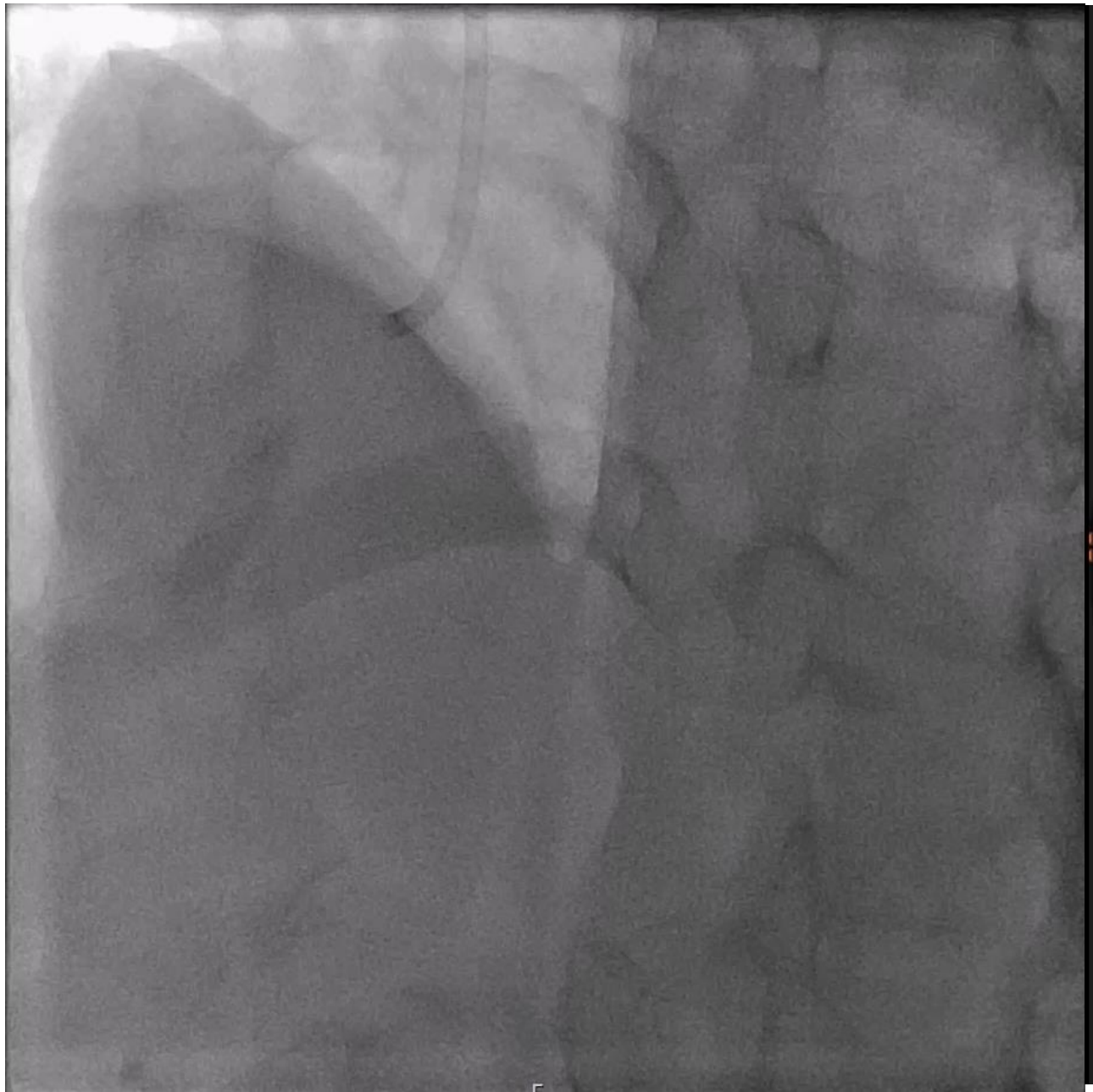
Fallbeskrivning

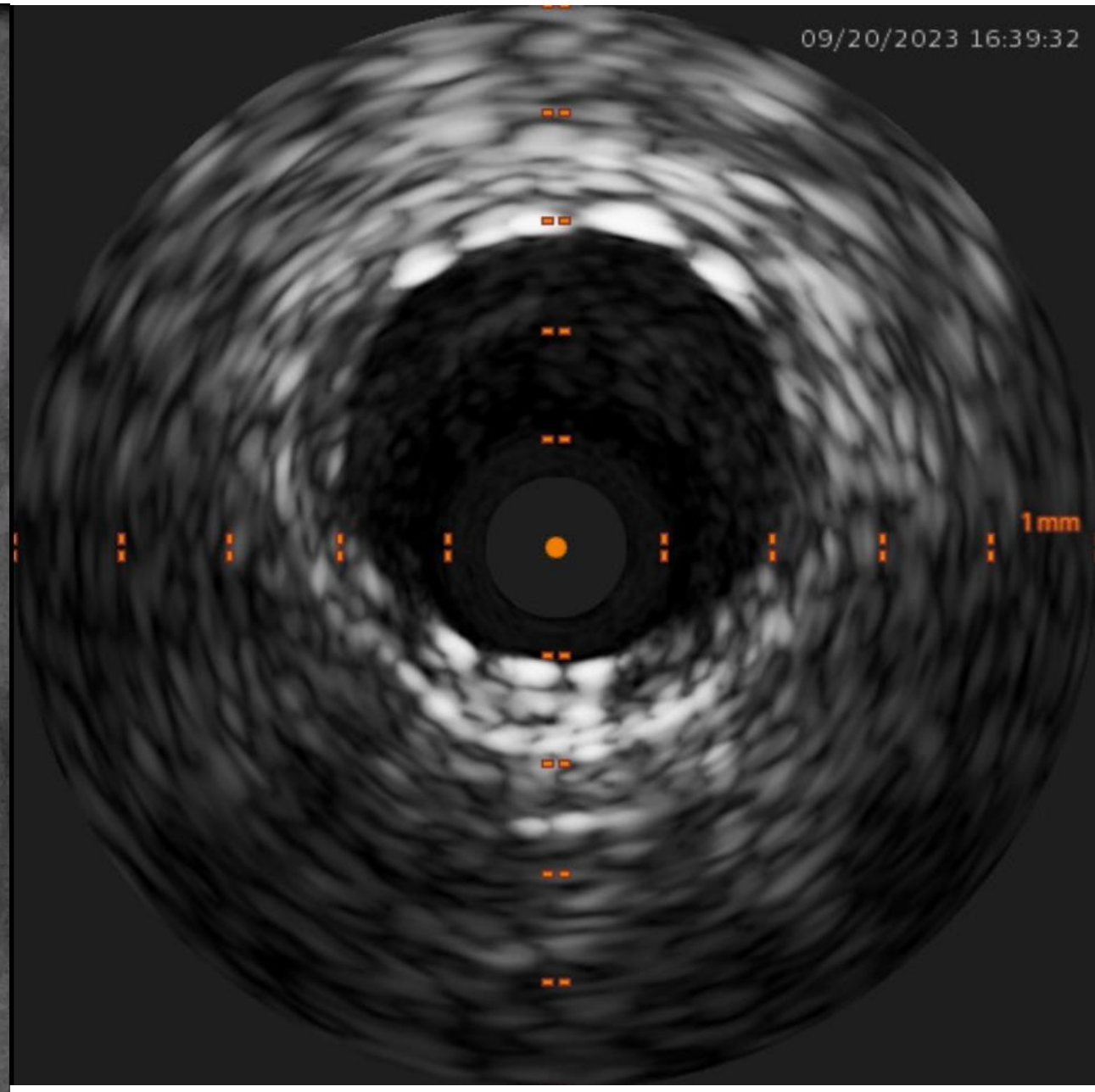
- Kvinna, 51 år gammal.
- Välreglerad typ 2 diabetes
- Metformin + Atorvastatin 40 mg + kost + motion
- BMI 27 (gått ner 5 kg i vikt på ett år)
- Hb 135, Krea 65, HbA1c 54, LDL 1.6, TG 2.1
- Ingen mikroalbuminuri.
- BT 135/80.

- Inkommer pga oklara bröstsmärtor nov 2022.
- EKO av hjärtat med normal pumpförmåga. Inga klaffel.
- Angiografi utan anmärkning. Fortsatt utredning bekräftar misstanke om perimyokardit. Skrivs ut välmående.
- Senaste månaderna tilltagande bröstsmärta och andfåddhet.
- Söker vård nu i september 2023 pga progredierande bröstsmärta.
- TnI på 50-111-157 (således hjärtskada).
- EKO visar nu nedsatt rörlighet i inferiora hjärtväggen, EF 40-45%.

- Angio med nytillkommen stenos med IVUS-verifierad ateroskleroras i höger kranskärl.

Fallbeskrivning slutresultat





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- Angio med nytillkommen stenos med IVUS-verifierad ateroskleroras i höger kranskärl.
- Stentning av höger kranskärl.

Fallbeskrivning fortsättning

- Dubblering av Atorvastatin till 80 mg.
- Diskussion tillägg av Ezetimibe/Vazkepa där valet föll på sistnämnda.
- Sätts in på ASA + Brilique, där patienten efter avslutad Brilique-behandling planeras för ASA + lågdos Xarelto (2,5 mg 1x2).

Skånes universitetssjukhus
En del av Region Skåne

PM

Hypertriglyceridemi

VO HLM
Hjärtmottagning kranskärl
SUS Lund

PM ansvarig: Anders Hansson

Giltigt till: 2024-12-31

ommer ”lysa svart” i
er!

ivt leta/screna!

Hypertriglyceridemi (Förhöjda triglycerider hos patienter med hög och mycket hög kardiovaskulär risk)

- Optimal S-TG nivå är <1,7 mmol/l
- Hypertriglyceridemi föreligger vid S-TG >2,0 mmol/l
- Måttlig hypertriglyceridemi: S-TG 2-10 mmol/l
- Uttalad hypertriglyceridemi: S-TG över 10 mmol/l

Värdena ger ökad risk för hjärt-kärlsjukdom och tillståndet ses vid övervikt, insulinresistens och typ 2 diabetes. Triglyceridvärden bör ingå i riskvärdering efter akut koronart syndrom. Det som ses i studien är att viss riskökning sker redan vid TG >1,2. Vid TG >1,7 är riskökningen väldigt markant.

UTREDNING
Genomgång av livsstilsfaktorer såsom läkemedelskonsumtion, fysisk aktivitet, alkoholkonsumtion och matvanor.

Kontroll av P-glukos, HbA1c, leverstatus, kreatinin, TSH, BMI och bukomfång.

BEHANDLING
Livsstilsförändringar med adekvat kost, ökad motion, minskad alkoholkonsumtion och reducerad övervikt.

Hos utvalda patienter där livsstilsförändringar och statin inte givit önskad effekt kan kapslar Vazkepa (ikosapentetyl) övervägas. Till patienter med hög kadiovaskulär risk baserat på dokumenterad aterosklerotisk sjukdom, som vid uppföljning på hjärtmottagningen trots statinbehandling har kvarstående TG ≥ 1,7 mmol/l.

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Hjärtmottagning kranskärl
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PM ansvarig: Anders Hansson

Giltigt till: 2024-12-31

TG på 1.7 kommer ”lysa svart” i våra journaler!

Vi måste aktivt leta/screna!

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2) Anti-inflammatorisk behandling - colchicine

Anti-inflammatorisk behandling har prövats tidigare - interleukinhämmare

Original Article

Antiinflammatory Therapy with Canakinumab for Atherosclerotic Disease

Paul M Ridker, M.D., Brendan M. Everett, M.D., Tom Thuren, M.D., Jean G. MacFadyen, B.A., William H. Chang, Ph.D., Christie Ballantyne, M.D., Francisco Fonseca, M.D., Jose Nicolau, M.D., Wolfgang Koenig, M.D., Stefan D. Anker, M.D., John J.P. Kastelein, M.D., Jan H. Cornel, M.D., Prem Pais, M.D., Daniel Pella, M.D., Jacques Genest, M.D., Renata Cifkova, M.D., Alberto Lorenzatti, M.D., Tamas Forster, M.D., Zhanna Kobalava, M.D., Luminita Vida-Simiti, M.D., Marcus Flather, M.D., Hiroaki Shimokawa, M.D., Hisao Ogawa, M.D., Mikael Dellborg, M.D., Paulo R.F. Rossi, M.D., Roland P.T. Troquay, M.D., Peter Libby, M.D., Robert J. Glynn, Sc.D., for the CANTOS Trial Group

N Engl J Med
Volume 377(12):1119-1131
September 21, 2017

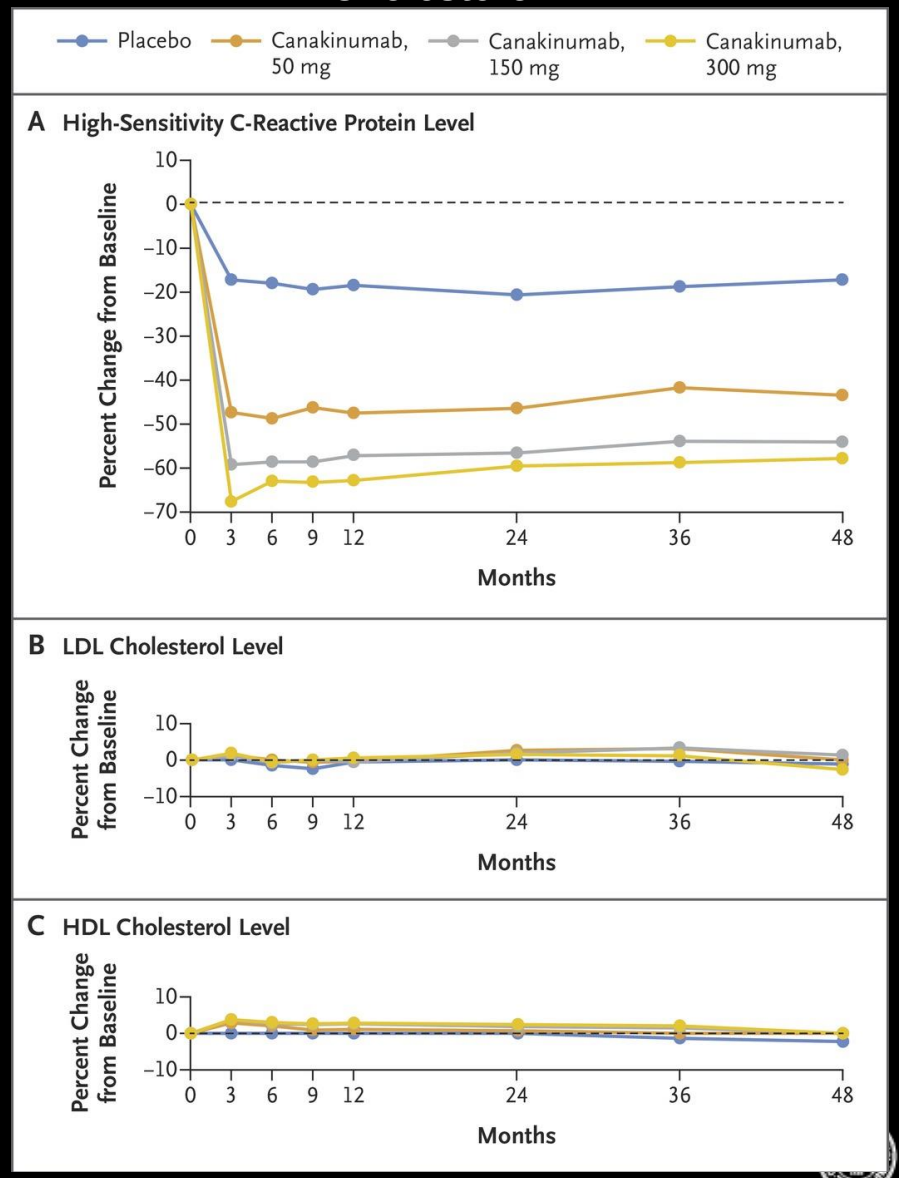
The NEW ENGLAND JOURNAL of MEDICINE

Study Overview

- Patients with myocardial infarction and a high-sensitivity CRP level of 2 mg or more per liter were assigned to one of three canakinumab doses or placebo.
- The 150-mg dose, but not the 50-mg or 300-mg dose, led to a lower incidence of recurrent cardiovascular events.

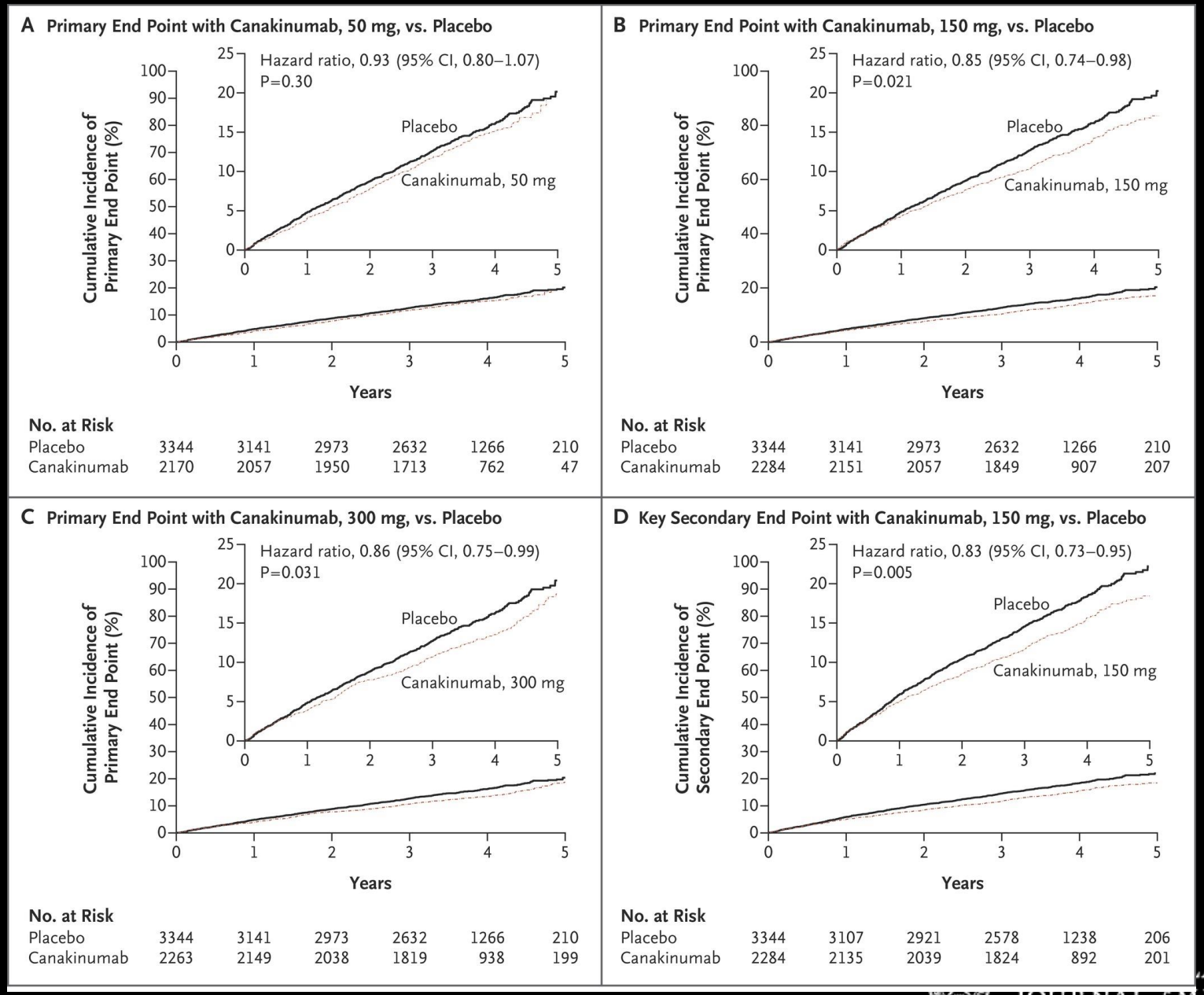
The NEW ENGLAND JOURNAL of MEDICINE

Effects of Canakinumab, as Compared with Placebo, on Plasma Levels of High-Sensitivity C-Reactive Protein, Low-Density Lipoprotein (LDL) Cholesterol, High-Density Lipoprotein (HDL) Cholesterol



Ridker PM et al. N Engl J Med ;377:1119-1131

Cumulative Incidence of the Primary End Point and the Key Secondary Cardiovascular End Point.



Ridker PM et al. N Engl J Med ;377:1119-1131

LAND JOURNAL of MEDICINE

Table 2. Incidence Rates and Hazard Ratios for Major Clinical Outcomes and All-Cause Mortality.*

Clinical Outcome	Placebo Group (N=3344)	Canakinumab				P Value for Trend across Doses vs. Placebo
		50-mg Group (N=2170)	150-mg Group (N=2284)	300-mg Group (N=2263)	All Doses (N=6717)	
Cardiovascular death, confirmed						
Incidence rate per 100 person-yr (no. of patients)	1.44 (182)	1.18 (94)	1.26 (110)	1.33 (115)	1.26 (319)	0.76
Hazard ratio (95% CI)	1.00	0.80 (0.62–1.03)	0.88 (0.70–1.12)	0.93 (0.74–1.18)	0.87 (0.73–1.05)	
P value	—	0.083	0.30	0.55	0.15	
Cardiovascular death or death of unknown cause						
Incidence rate per 100 person-yr (no. of patients)	1.86 (235)	1.71 (137)	1.65 (144)	1.74 (151)	1.70 (432)	0.62
Hazard ratio (95% CI)	1.00	0.89 (0.72–1.11)	0.90 (0.73–1.10)	0.94 (0.77–1.16)	0.92 (0.78–1.07)	
P value	—	0.30	0.30	0.59	0.28	
Noncardiovascular death, confirmed						
Incidence rate per 100 person-yr (no. of patients)	1.11 (140)	1.14 (91)	1.08 (94)	1.02 (88)	1.08 (273)	0.45
Hazard ratio (95% CI)	1.00	1.02 (0.78–1.34)	0.97 (0.74–1.26)	0.92 (0.70–1.20)	0.97 (0.79–1.19)	
P value	—	0.87	0.81	0.54	0.79	
Death from any cause						
Incidence rate per 100 person-yr (no. of patients)	2.97 (375)	2.85 (228)	2.73 (238)	2.76 (239)	2.78 (705)	0.39
Hazard ratio (95% CI)	1.00	0.94 (0.80–1.11)	0.92 (0.78–1.09)	0.94 (0.80–1.10)	0.94 (0.83–1.06)	
P value	—	0.48	0.33	0.42	0.31	

Ökning av sepsis/fatala infektioner

Hög kostnad

Utveckling stoppades

Anti-inflammatorisk behandling har prövats tidigare - metotrexat

Study Overview

- Patients with coronary artery disease were randomly assigned to either methotrexate (15 to 20 mg weekly) or placebo.
- At a median of 2.3 years, there was no difference between the two groups in the rate of myocardial infarction, stroke, or cardiovascular death.

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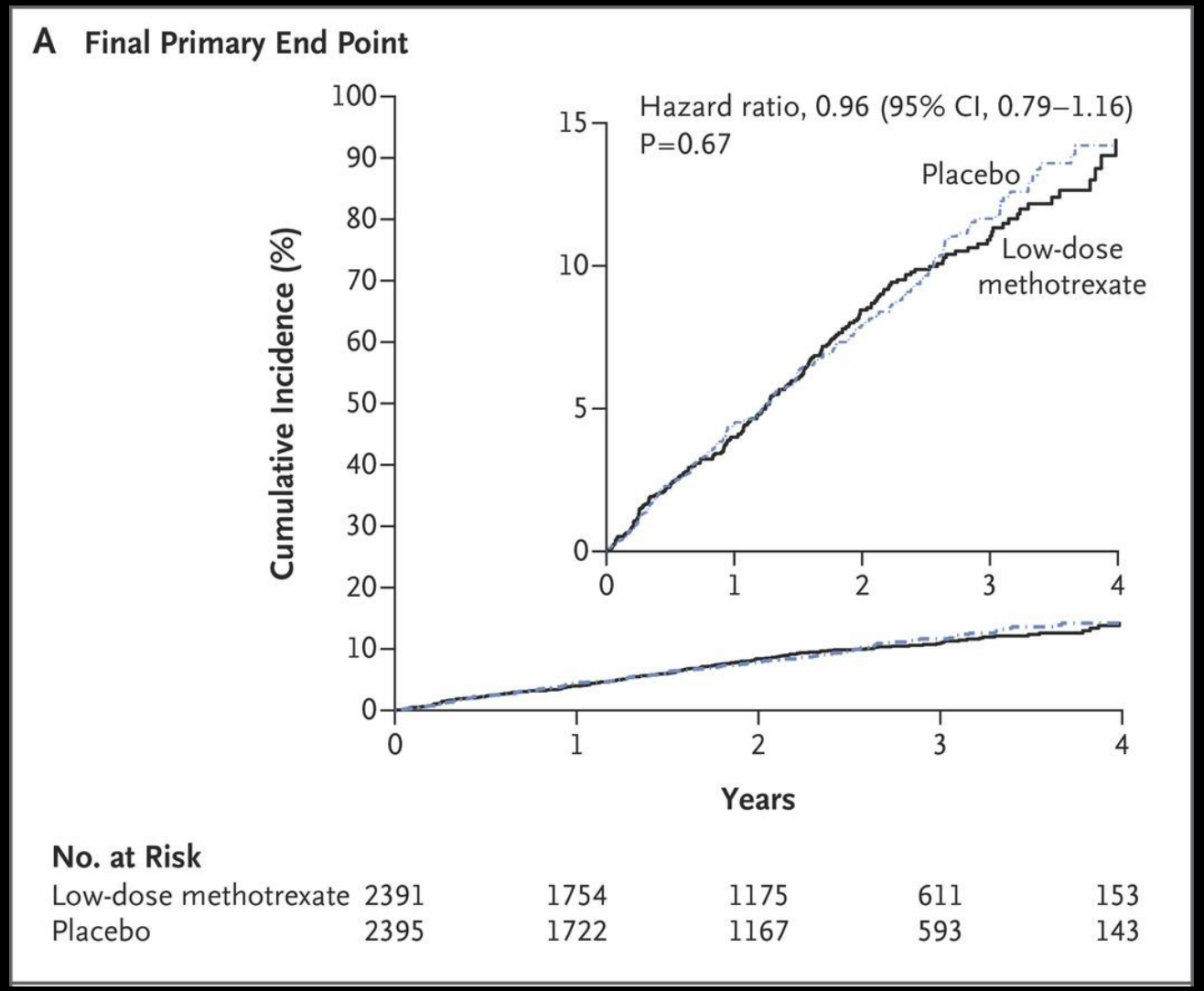
Cumulative Incidence of the Final Primary End Point and the Original Primary End Point.



Ridker PM et al. N Engl J Med2019;380:752-762

Anti-inflammatorisk behandling - colchicine

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Efficacy and Safety of Low-Dose Colchicine after Myocardial Infarction

Authors: Jean-Claude Tardif, M.D., Simon Kouz, M.D., David D. Waters, M.D., Olivier F. Bertrand, M.D., Ph.D., Rafael Diaz, M.D., Aldo P. Maggioni, M.D., Fausto J. Pinto, M.D., Ph.D., +17, and François Roubille, M.D., Ph.D. Author Info & Affiliations

Published November 16, 2019 | N Engl J Med 2019;381:2497-2505 | DOI: 10.1056/NEJMoa1912388

VOL. 381 NO. 26

- Inflammation appears to play a role in atherosclerosis, raising the possibility that treatments that reduce inflammation could prevent cardiovascular events.
- In a randomized, placebo-controlled trial involving 4745 patients with recent myocardial infarction, low-dose colchicine (0.5 mg once daily) prevented ischemic cardiovascular events.

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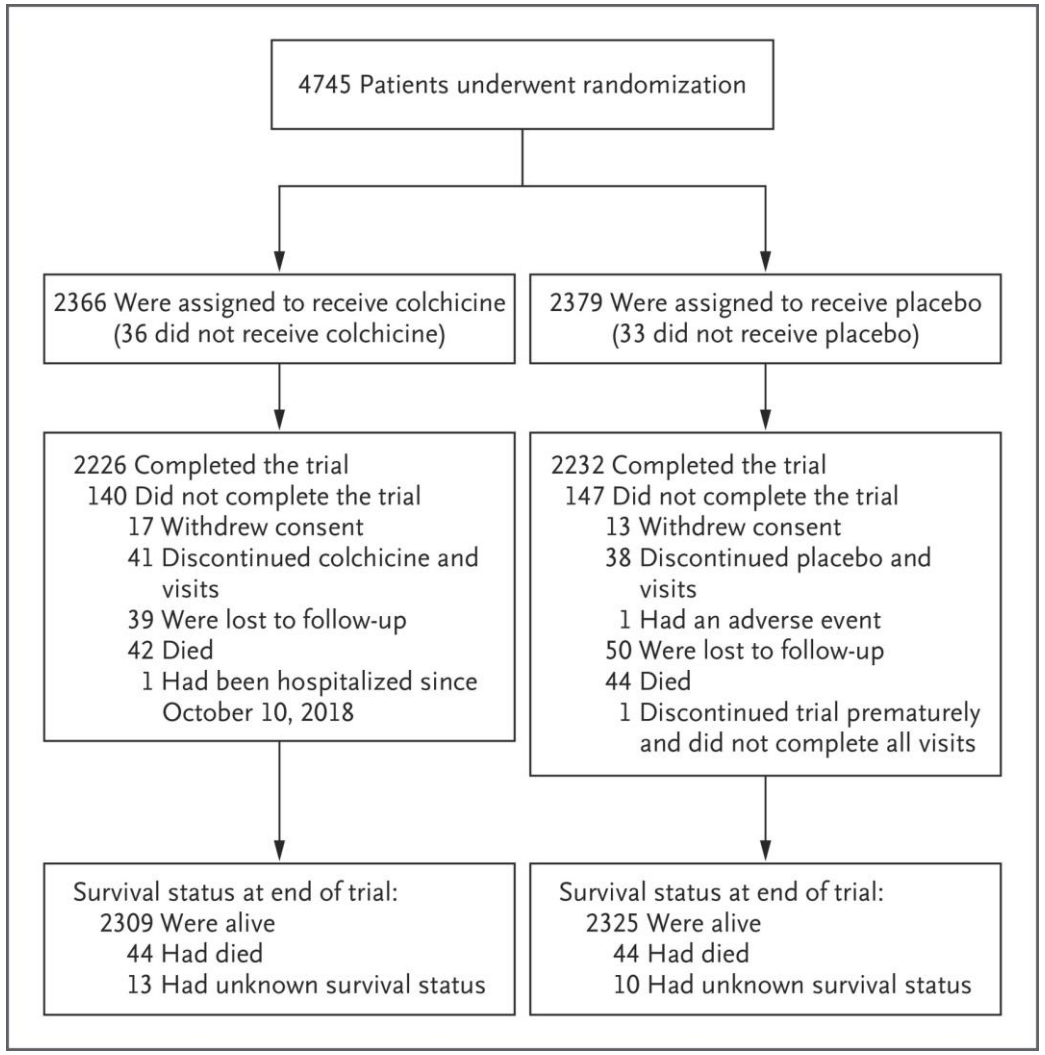
Randomization and Follow-up of the Patients.



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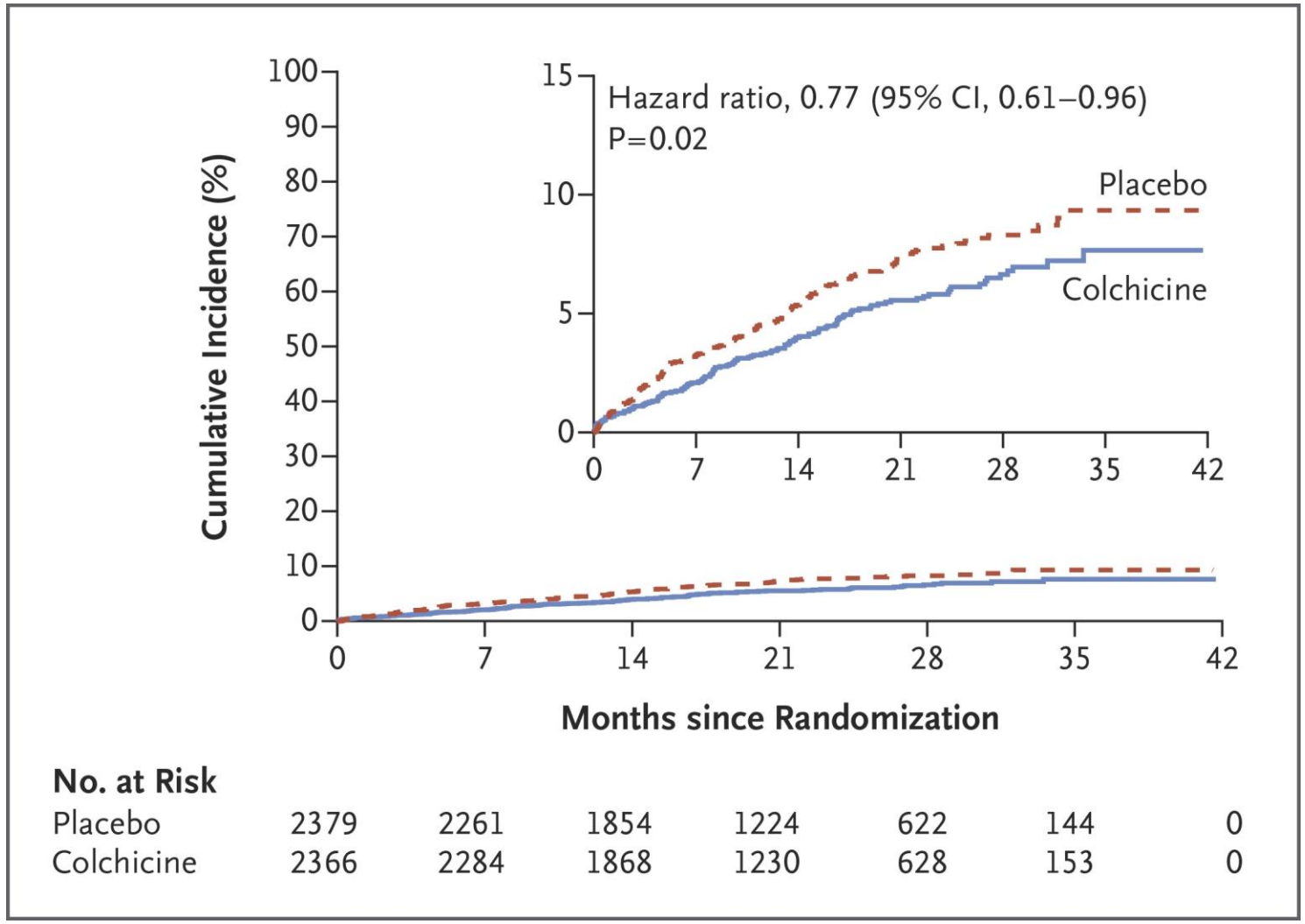
Cumulative Incidence of Cardiovascular Events (Intention-to-Treat Population).



No. at Risk

Placebo	2379	2261	1854	1224	622	144	0
Colchicine	2366	2284	1868	1230	628	153	0

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Major Clinical End Points (Intention-to-Treat Population).

Table 2. Major Clinical End Points (Intention-to-Treat Population).*

End Point	Colchicine (N=2366)	Placebo (N=2379)	Hazard Ratio (95% CI)	P Value
	number (percent)			
Primary composite end point	131 (5.5)	170 (7.1)	0.77 (0.61–0.96)	0.02†
Components of primary end point				
Death from cardiovascular causes	20 (0.8)	24 (1.0)	0.84 (0.46–1.52)	
Resuscitated cardiac arrest	5 (0.2)	6 (0.3)	0.83 (0.25–2.73)	
Myocardial infarction	89 (3.8)	98 (4.1)	0.91 (0.68–1.21)	
Stroke	5 (0.2)	19 (0.8)	0.26 (0.10–0.70)	
Urgent hospitalization for angina leading to revascularization	25 (1.1)	50 (2.1)	0.50 (0.31–0.81)	
Secondary composite end point‡	111 (4.7)	130 (5.5)	0.85 (0.66–1.10)	
Death	43 (1.8)	44 (1.8)	0.98 (0.64–1.49)	
Deep venous thrombosis or pulmonary embolus	10 (0.4)	7 (0.3)	1.43 (0.54–3.75)	
Atrial fibrillation	36 (1.5)	40 (1.7)	0.93 (0.59–1.46)	

* Only the initial event was counted in the analyses of time to first event for the primary composite end point and for the secondary composite end point. In the component analysis, the different types of events were counted separately.

† The log-rank test and the multivariable Cox proportional-hazards model including age, history of diabetes, previous coronary revascularization, and previous heart failure yielded similar P values.

‡ The secondary composite end point included death from cardiovascular causes, resuscitated cardiac arrest, myocardial infarction, and stroke.

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- Among patients with a recent myocardial infarction, colchicine at a dose of 0.5 mg daily led to a significantly lower risk of ischemic cardiovascular events than placebo.

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- Patients with chronic coronary disease were randomly assigned to receive 0.5 mg of colchicine once daily or matching placebo.
- The incidence of the composite end point of cardiovascular death, spontaneous myocardial infarction, ischemic stroke, or ischemia-driven coronary revascularization was significantly lower with colchicine than with placebo.

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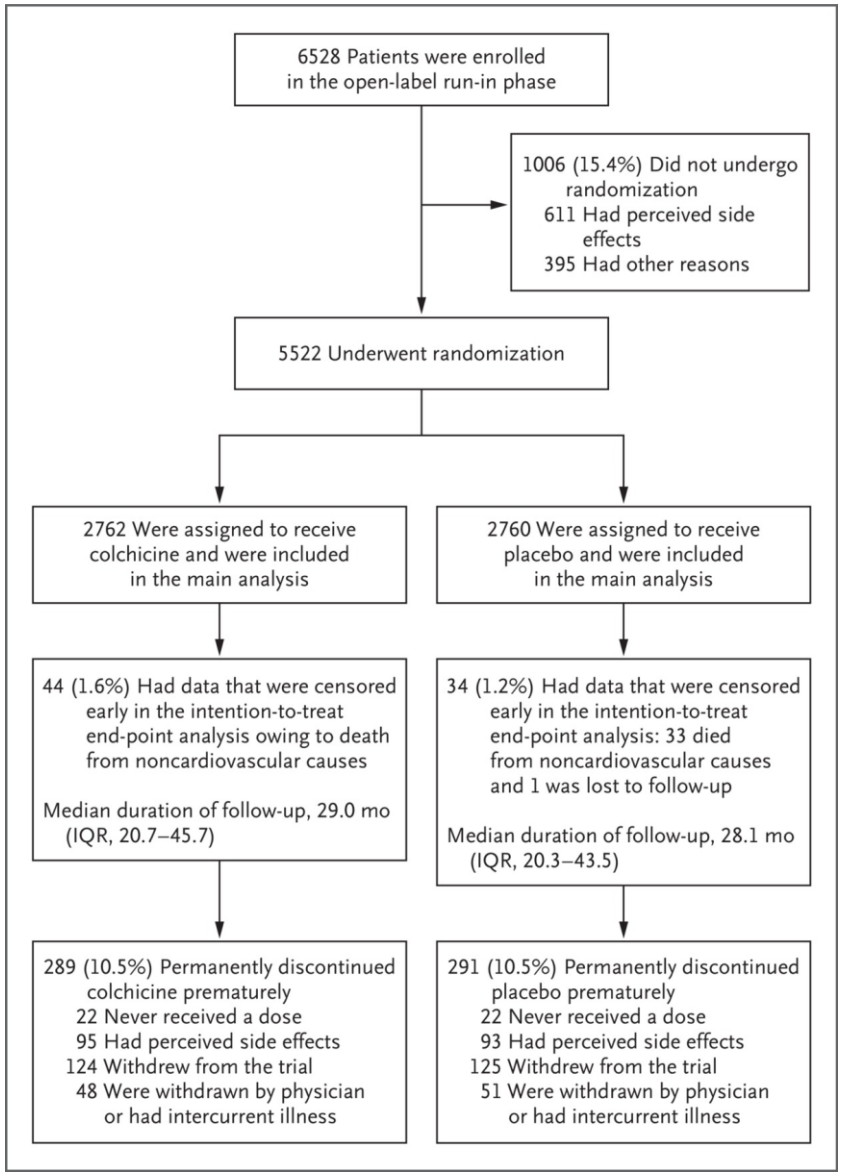
Enrollment, Randomization, and Follow-up.



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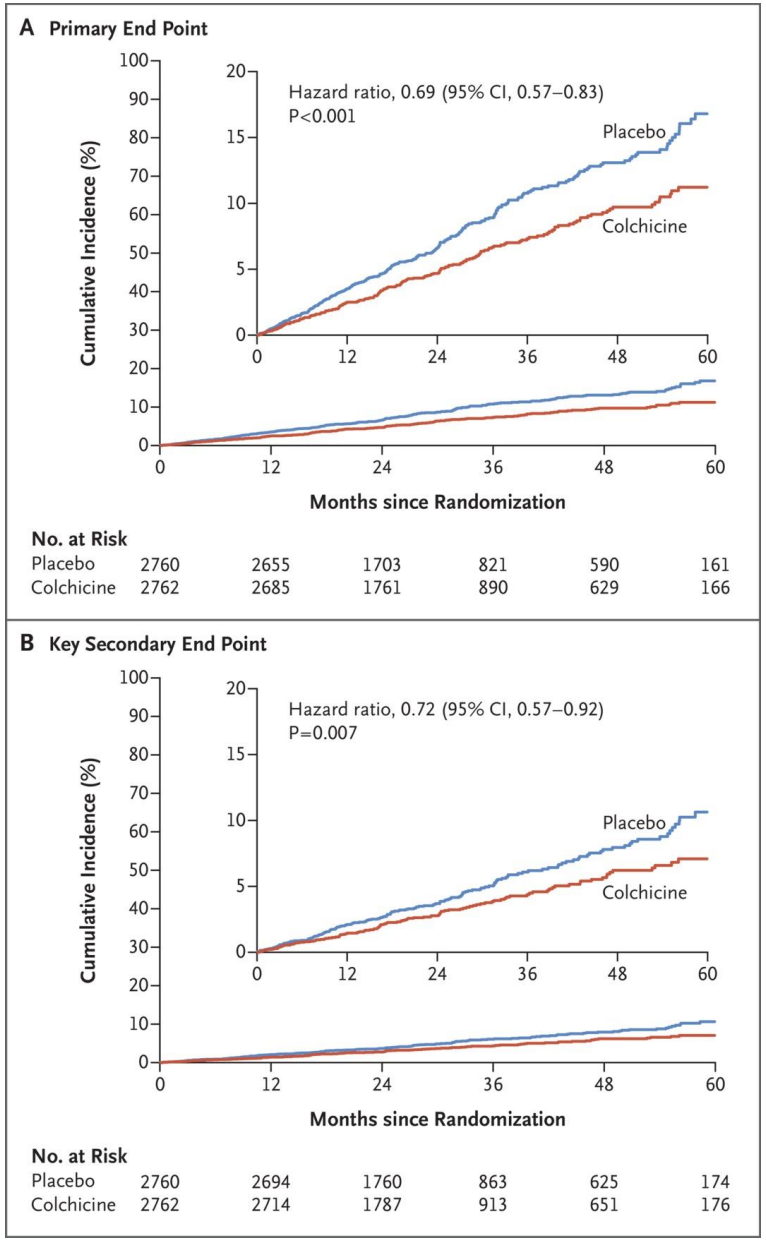
Cumulative Incidence of the Primary End Point and the Key Secondary End Point.



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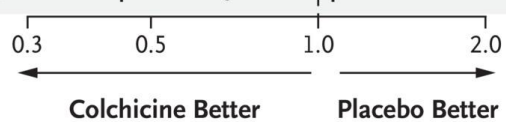
Key End Points and their Components.

End Point	Colchicine (N=2762)		Placebo (N=2760)		Hazard Ratio (95% CI)	P Value
	no. of patients (%)	*no. of events/100 person-yr*	*no. of patients (%)*	*no. of events/100 person-yr*		
Primary end point						
Cardiovascular death, myocardial infarction, ischemic stroke, or ischemia-driven coronary revascularization	187 (6.8)	2.5	264 (9.6)	3.6	0.69 (0.57–0.83)	<0.001
Secondary end points in ranked order						
Cardiovascular death, myocardial infarction, or ischemic stroke	115 (4.2)	1.5	157 (5.7)	2.1	0.72 (0.57–0.92)	0.007
Myocardial infarction or ischemia-driven coronary revascularization	155 (5.6)	2.1	224 (8.1)	3.0	0.67 (0.55–0.83)	<0.001
Cardiovascular death or myocardial infarction	100 (3.6)	1.3	138 (5.0)	1.8	0.71 (0.55–0.92)	0.01
Ischemia-driven coronary revascularization	135 (4.9)	1.8	177 (6.4)	2.4	0.75 (0.60–0.94)	0.01
Myocardial infarction	83 (3.0)	1.1	116 (4.2)	1.5	0.70 (0.53–0.93)	0.01
Ischemic stroke	16 (0.6)	0.2	24 (0.9)	0.3	0.66 (0.35–1.25)	0.20
Death from any cause	73 (2.6)	0.9	60 (2.2)	0.8	1.21 (0.86–1.71)	
Cardiovascular death	20 (0.7)	0.3	25 (0.9)	0.3	0.80 (0.44–1.44)	
Additional end points						
The primary end point in the first LoDoCo trial	201 (7.3)	2.7	290 (10.5)	4.0	0.67 (0.56–0.81)	
New onset or first recurrence in atrial fibrillation or atrial flutter	126 (4.6)	1.7	148 (5.4)	2.0	0.84 (0.66–1.07)	
Deep-vein thrombosis or pulmonary embolism or both	17 (0.6)	0.2	16 (0.6)	0.2	1.06 (0.53–2.10)	
Any myocardial infarctions	85 (3.1)	1.1	117 (4.2)	1.5	0.72 (0.54–0.95)	
New-onset diabetes	34 (1.2)	—	49 (1.8)	—	0.69 (0.44–1.06)	



- In a randomized trial involving patients with chronic coronary disease, the risk of cardiovascular events was significantly lower among those who received 0.5 mg of colchicine once daily than among those who received placebo.

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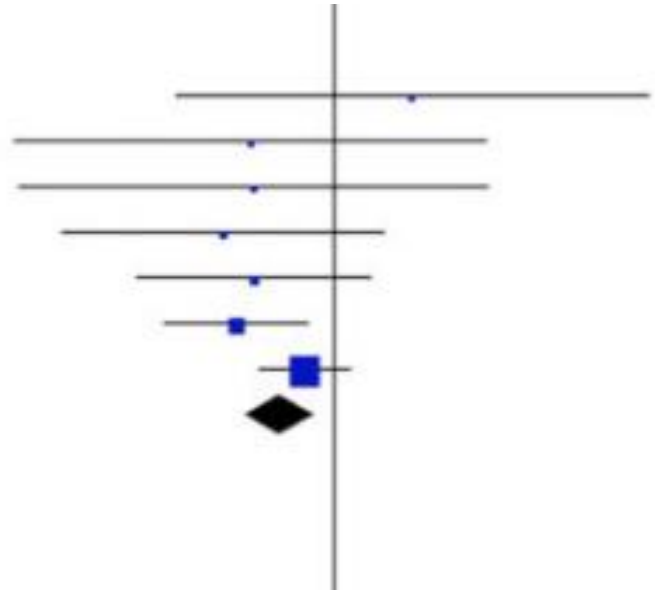
2.3.4 Stroke						
Shah 2020	1	206	0	194	2.2%	2.83 [0.12, 68.96]
Deftereos 2013	0	100	1	96	2.2%	0.32 [0.01, 7.76]
Raju 2012	0	40	1	40	2.2%	0.33 [0.01, 7.95]
Nidorf 2013	1	282	4	250	4.7%	0.22 [0.02, 1.97]
Tong 2020	2	396	6	399	8.8%	0.34 [0.07, 1.65]
Tardif 2019	5	2366	19	2379	23.2%	0.26 [0.10, 0.71]
Nidorf 2020	16	2762	24	2760	56.6%	0.67 [0.35, 1.25]
Subtotal (95% CI)		**6152**		**6118**	**100.0%**	**0.48 [0.30, 0.77]**
Total events	25		55			

Heterogeneity: $Tau^2 = 0.00$; $Chi^2 = 4.43$, $df = 6$ ($P = 0.62$); $I^2 = 0\%$

Test for overall effect: $Z = 3.03$ ($P = 0.002$)



Andreis et al, European Journal of Preventive Cardiology (2021) 28, 1916–1925

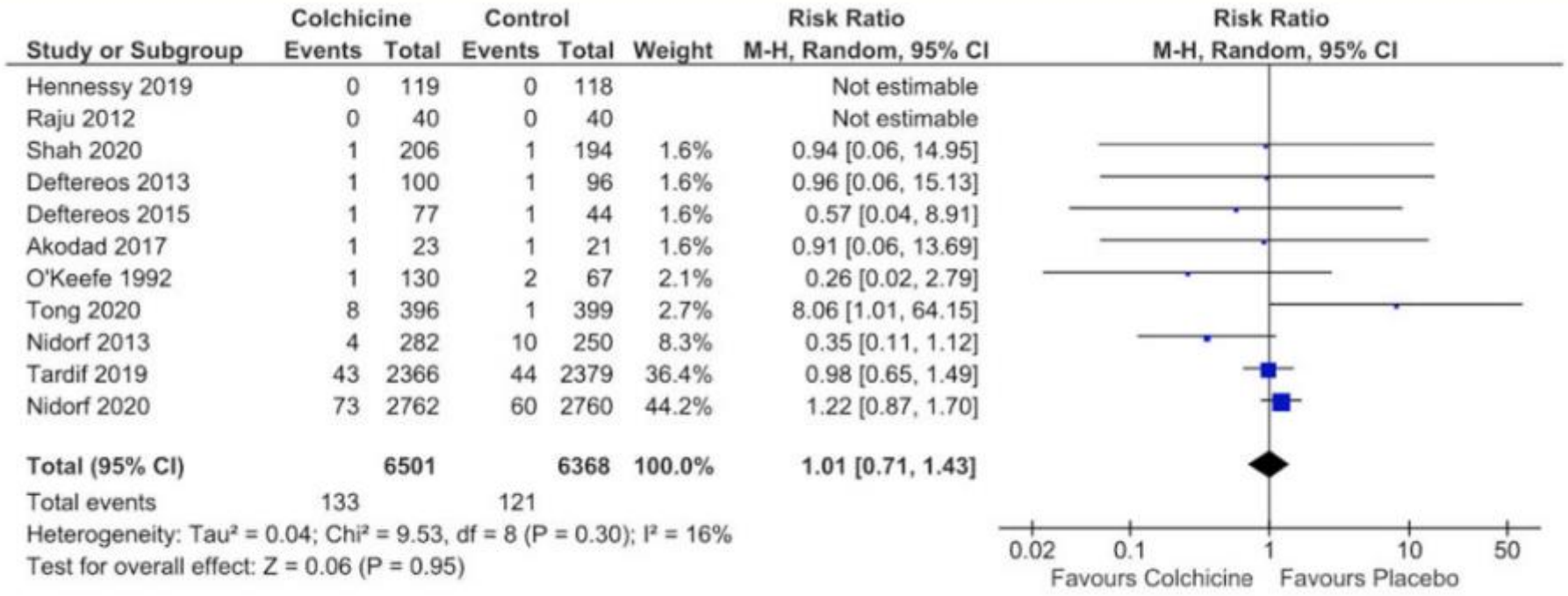


Figure 3 Forest plot showing the estimated relative risk of all-cause death during colchicine treatment compared with placebo.

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

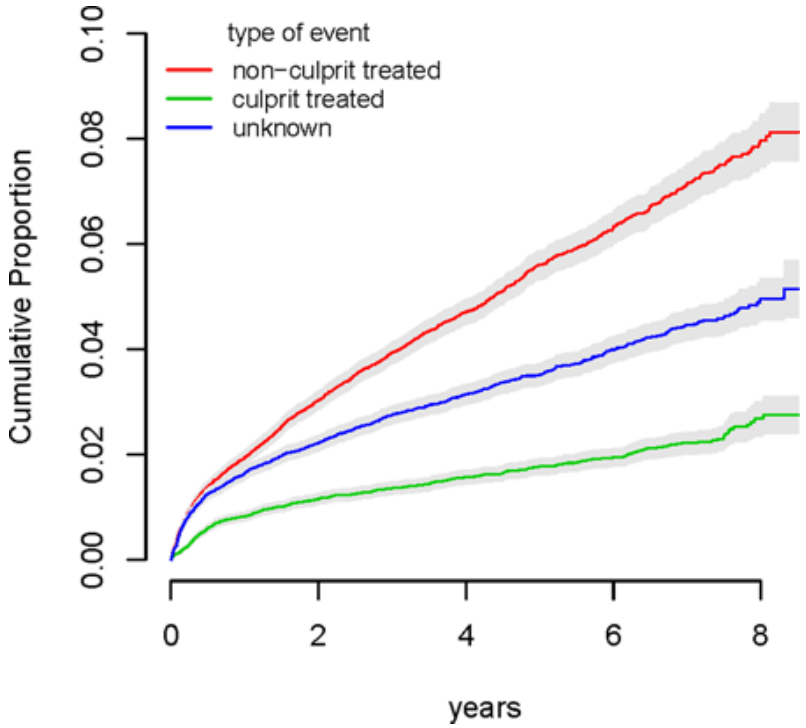
Recommendation	Class[a]	Level[b]
In CCS patients with atherosclerotic CAD, low-dose colchicine (0.5 mg daily) should be considered to reduce myocardial infarction, stroke, and need for revascularization.[714–716]	IIa	A

Andreis et al, European Journal of Preventive Cardiology (2021) 28, 1916–1925

3) Förlängd anti-trombotisk behandling
- Lågdos Brilique som tillägg till ASA

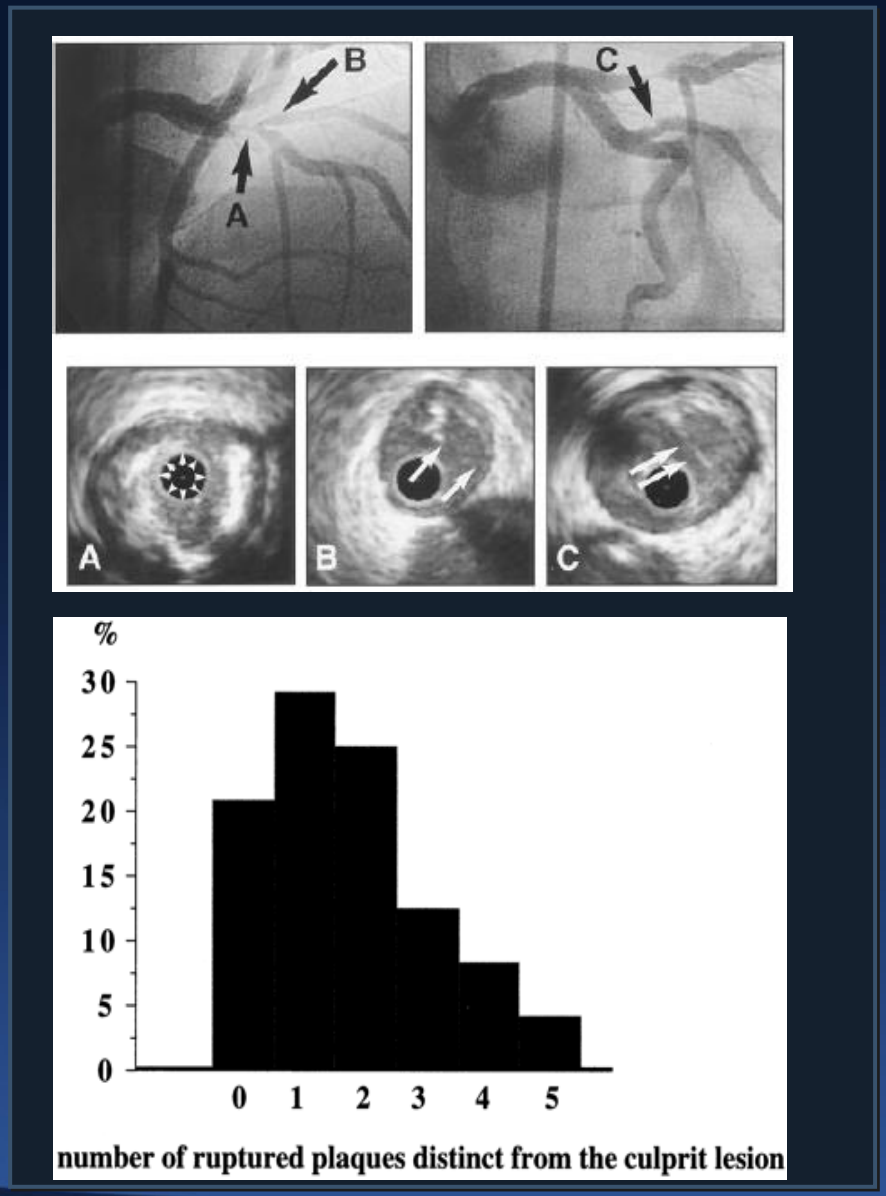
Risk of re-MI originating from a non-culprit lesion was 3 times higher than the risk of lesions originating from a previously stented culprit lesion

Prospective cohort study in 99,546 first-occurrence MI patients, undergoing PCI, enrolled in SWEDEHEART between 1 Jan 2006 and 31 Dec 2014



Varenhorst et. al. Circulation. 2016;134:A13206

Non-culprit ulcerated plaques are common



- *Ulcerated plaques are ubiquitous and multiple in patients with acute coronary deaths (Frink 1994)*
- *80% of ACS patients have >1 ulcerated plaque (Rioufol 2002)*

Frink J Invas Cardiol 1994; Rioufol et al. Arch Mal Coeur Vaiss 2002 (Circulation 2002 retracted due to replication)

The PEGASUS-TIMI 54 study

The NEW ENGLAND JOURNAL of MEDICINE

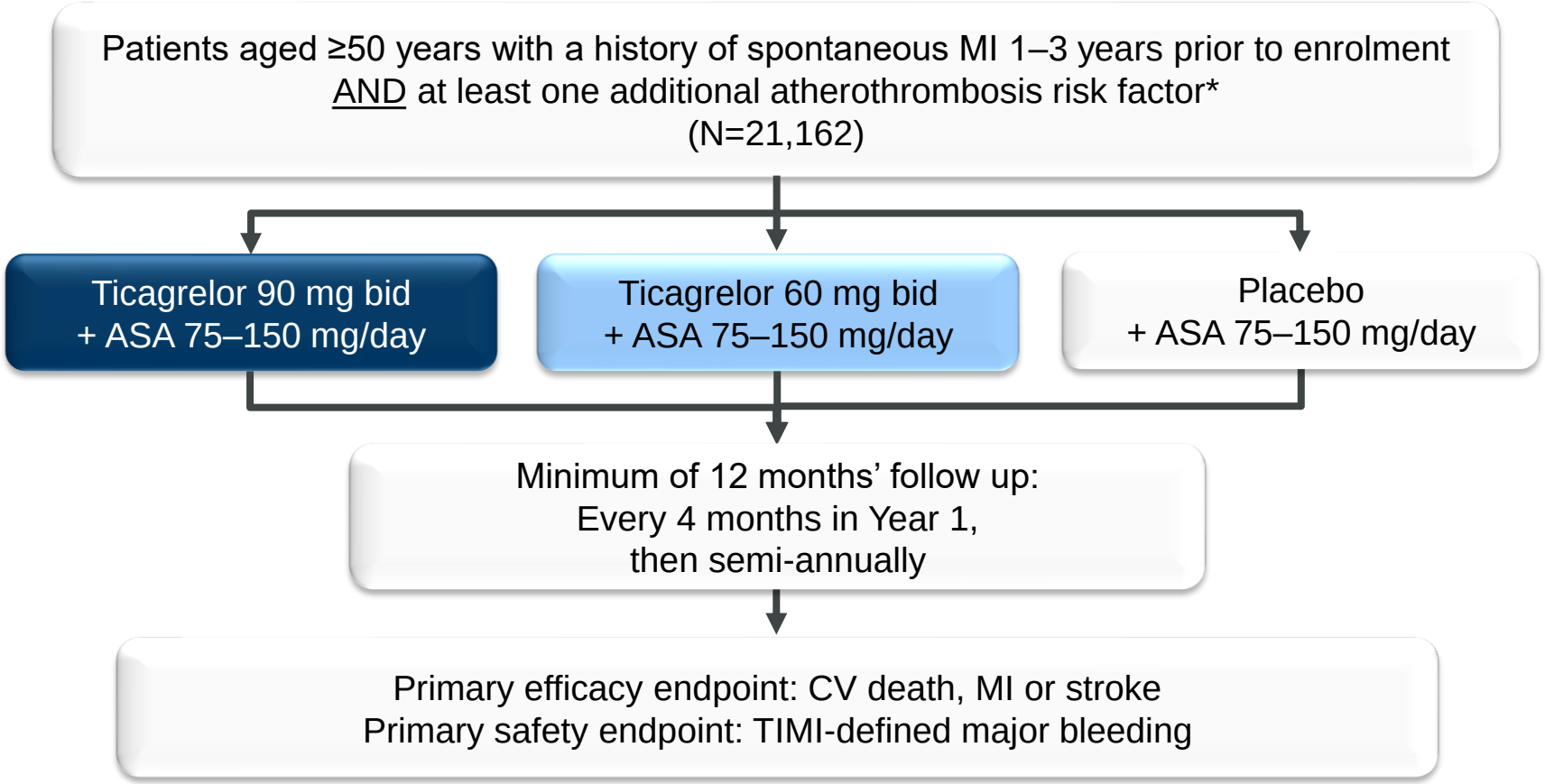
ORIGINAL ARTICLE

Long-Term Use of Ticagrelor in Patients with Prior Myocardial Infarction

Marc P. Bonaca, M.D., M.P.H., Deepak L. Bhatt, M.D., M.P.H., Marc Cohen, M.D., Philippe Gabriel Steg, M.D., Robert F. Storey, M.D., Eva C. Jensen, M.D., Ph.D., Giulia Magnani, M.D., Sameer Bansilal, M.D., M. Polly Fish, B.A., Kyungah Im, Ph.D., Olof Bengtsson, Ph.Lic., Ton Oude Ophuis, M.D., Ph.D., Andrzej Budaj, M.D., Ph.D., Pierre Theroux, M.D., Mikhail Ruda, M.D., Christian Hamm, M.D., Shinya Goto, M.D., Jindrich Spinar, M.D., José Carlos Nicolau, M.D., Ph.D., Robert G. Kiss, M.D., Ph.D., Sabina A. Murphy, M.P.H., Stephen D. Wiviott, M.D., Peter Held, M.D., Ph.D., Eugene Braunwald, M.D., and Marc S. Sabatine, M.D., M.P.H., for the PEGASUS-TIMI 54 Steering Committee and Investigators*

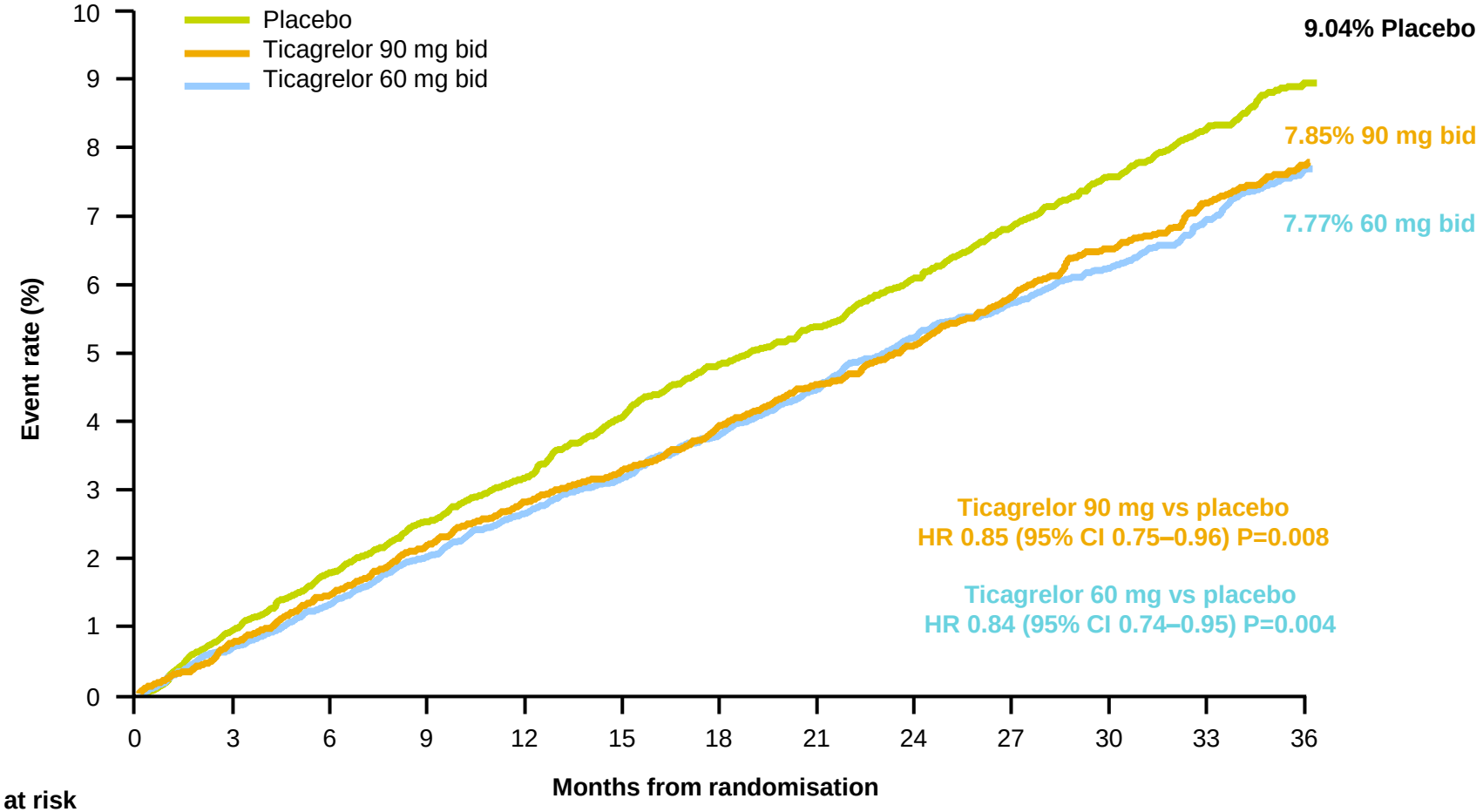
Bonaca MP *et al*. *N Engl J Med* 2015;372:1791–1800

PEGASUS-TIMI 54:
Study Design



*Age ≥65 years, diabetes mellitus, second prior MI, multivessel CAD or chronic non-end stage renal disease
bid, twice daily; CAD, coronary artery disease; TIMI, Thrombolysis in Myocardial Infarction
Bonaca MP *et al. Am Heart J* 2014;167:437–444, Bonaca MP *et al*. *N Engl J Med* 2015;372:1791–1800

PEGASUS-TIMI 54: Primary Endpoint

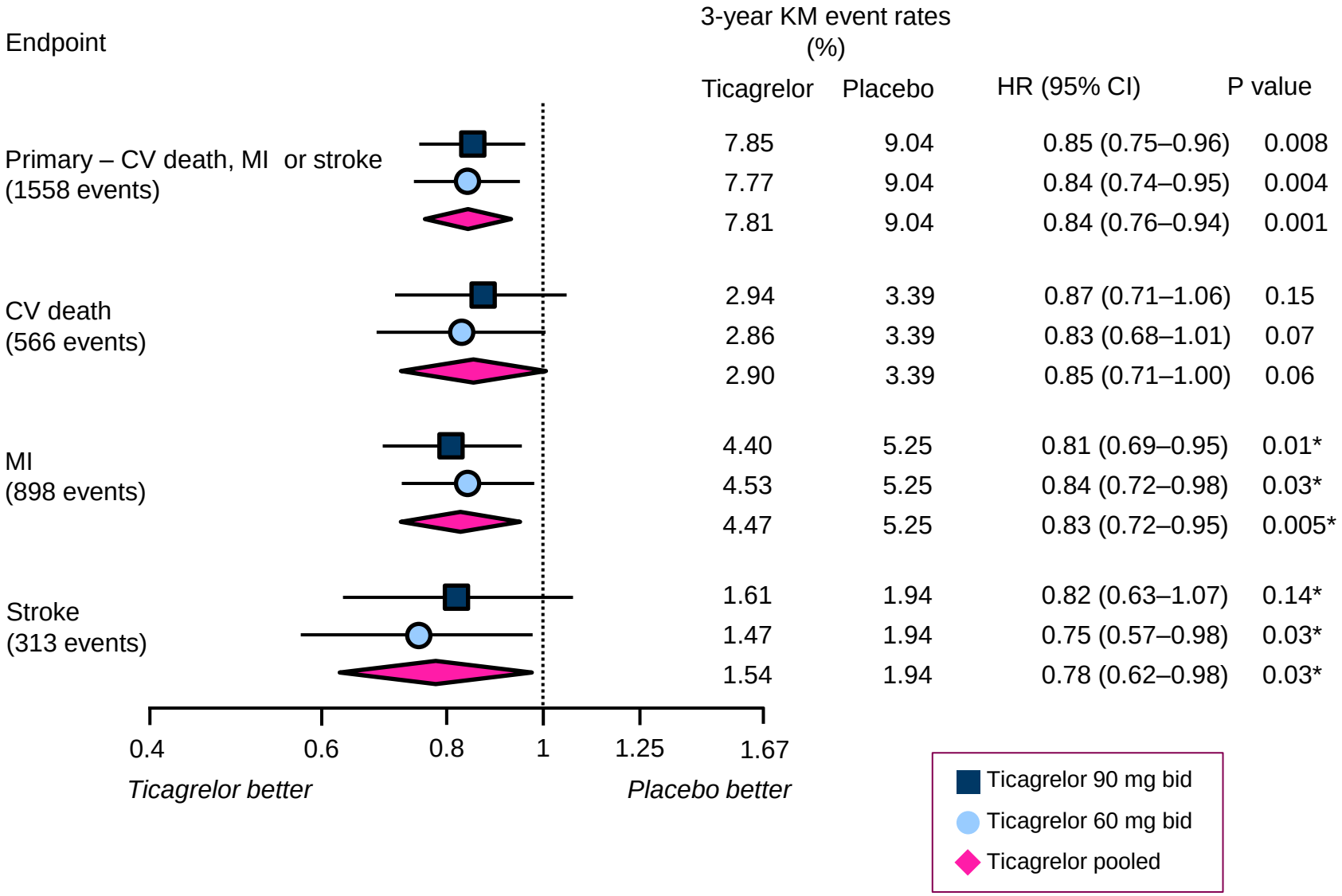


No. at risk													
Placebo	7067	6979	6892	6823	6761	6681	6508	6236	5876	5157	4343	3360	2028
90 mg bid	7050	6973	6899	6827	6769	6719	6550	6272	5921	5243	4401	3368	2038
60 mg bid	7045	6969	6905	6842	6784	6733	6557	6270	5904	5222	4424	3392	2055

P<0.026 indicates statistical significance; CI, confidence interval; HR, hazard ratio

Bonaca MP *et al*. *N Engl J Med* 2015;372:1791–1800

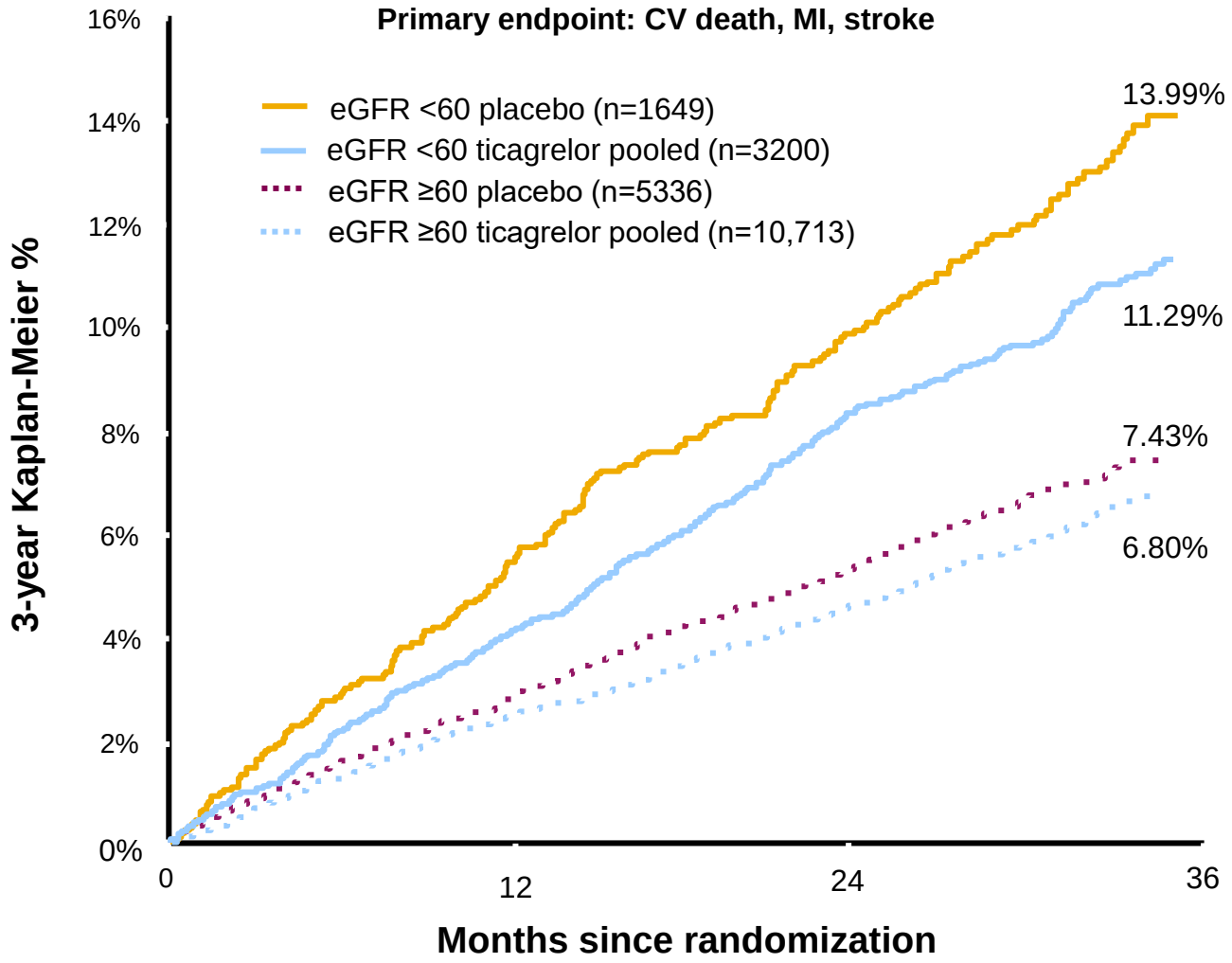
PEGASUS-TIMI 54: Efficacy Endpoints



*Indicates nominal P value; P<0.026 indicates statistical significance

Bonaca MP *et al*. *N Engl J Med* 2015;372:1791–1800

Efficacy of ticagrelor by eGFR



HR (95% CI)
0.81 (0.68, 0.96)
ARR = 2.70%

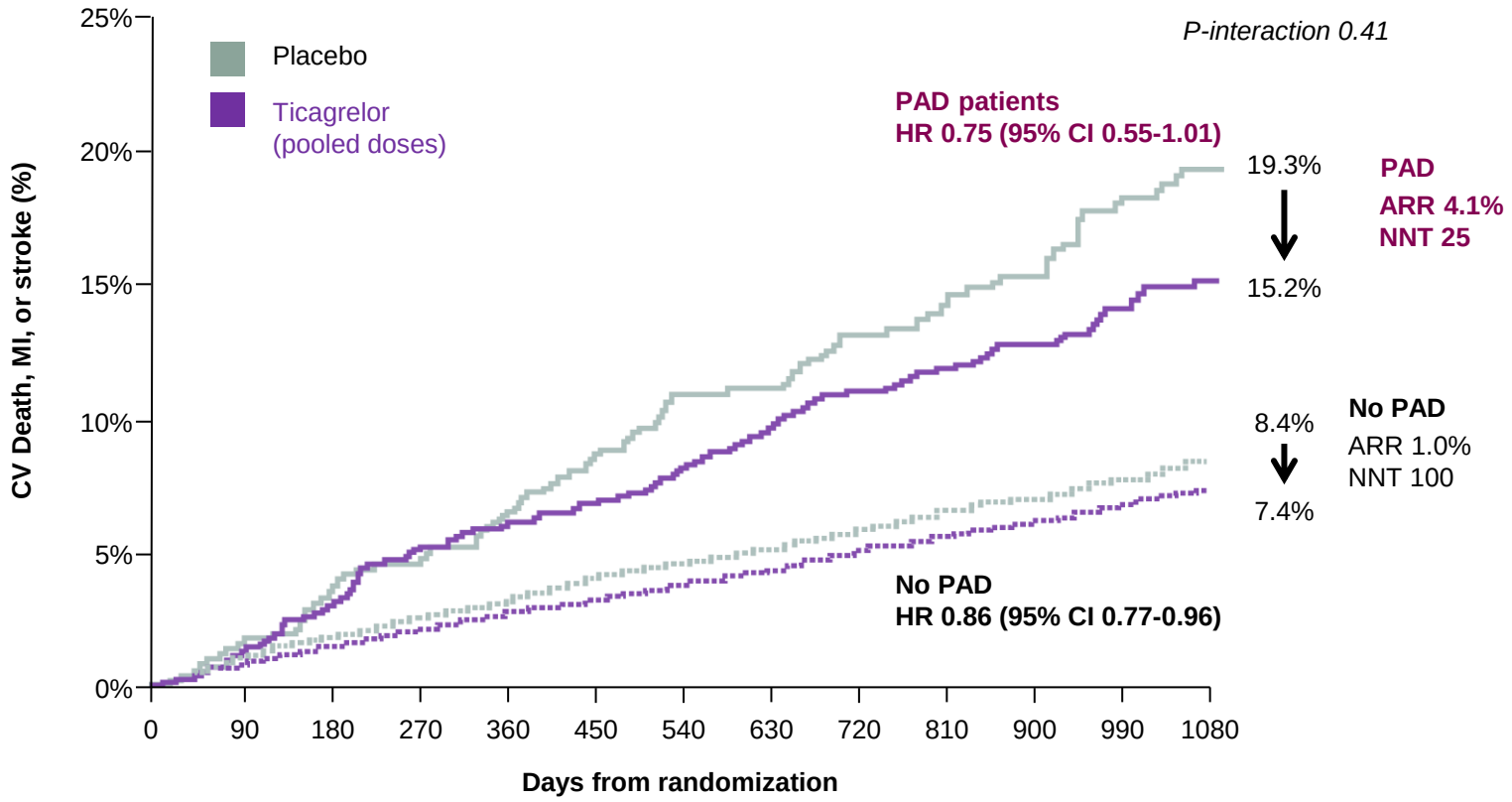
HR (95% CI)
0.88 (0.77, 1.00)
ARR = 0.63%

ARR, absolute risk reduction

Magnani G. *et al,* Eur Heart J, doi:10.1093/eurheartj/ehv482

PEGASUS-TIMI 54 – PAD

MACE with ticagrelor by PAD at baseline



Bonaca M et al., JACC 2016, 10.1016/j.jacc.2016.03.524

3) Förlängd anti-trombotisk behandling
- Lågdos Xarelto som tillägg till ASA

COMPASS

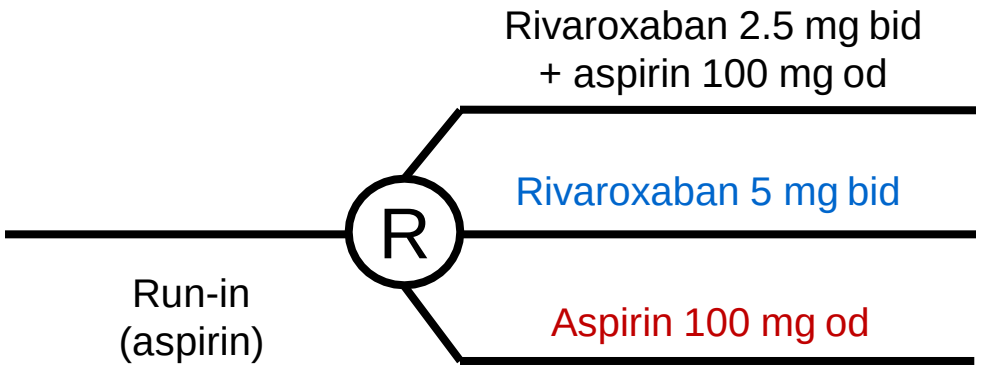
Population Health Research Institute
HEALTH THROUGH KNOWLEDGE

August 27, 2017

Rivaroxaban with or without aspirin in stable cardiovascular disease

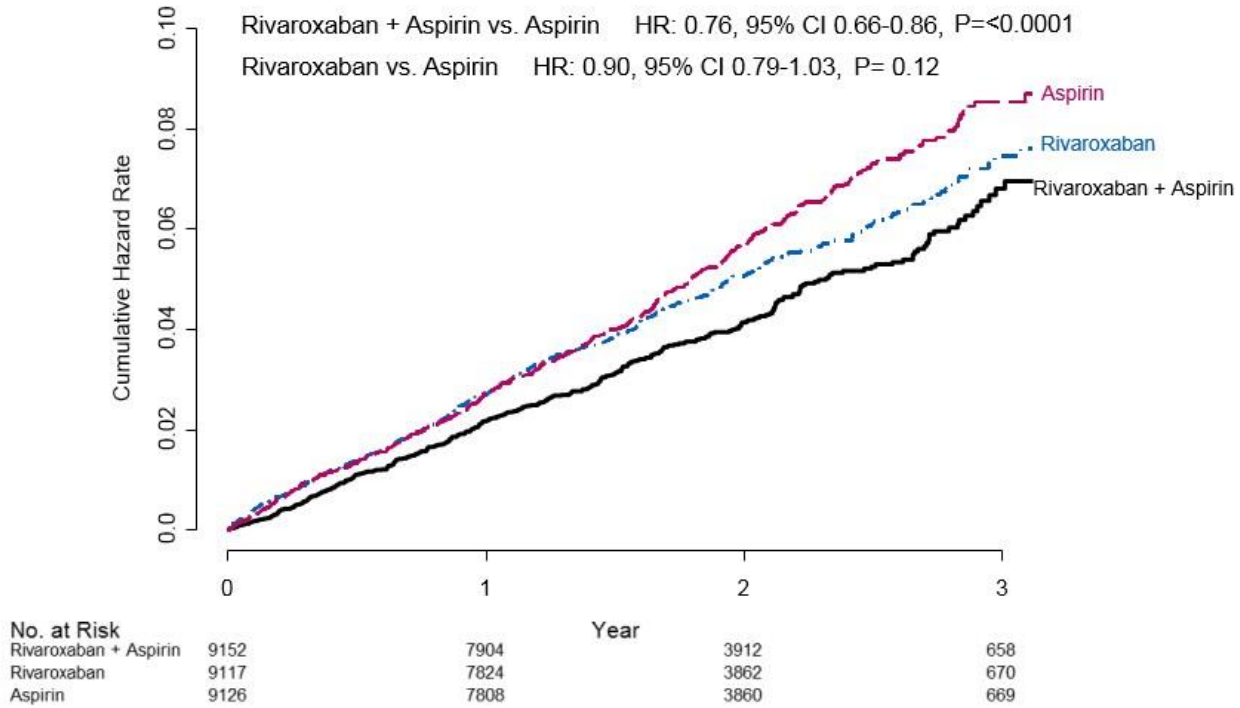
COMPASS design

Stable CAD or PAD
2,200 with a primary outcome event



Expected follow up
3-4 years

Primary: CV death, stroke, MI

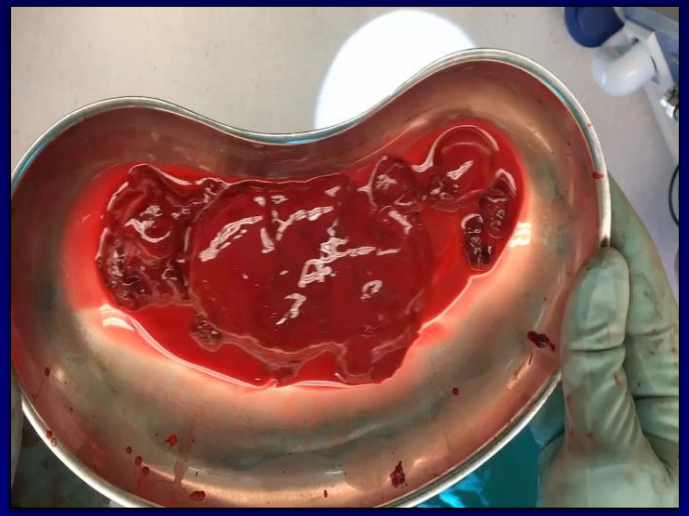


Secondary outcomes

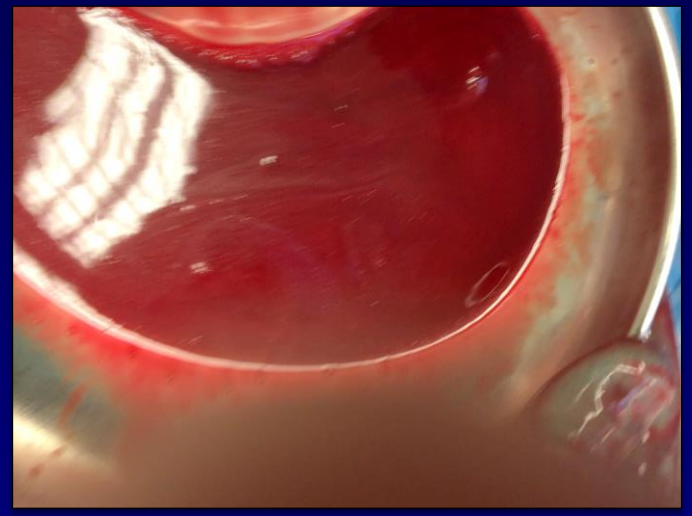
Outcome	R + A N=9,152	A N=9,126	Rivaroxaban + Aspirin vs. Aspirin	
	N (%)	N (%)	HR (95% CI)	P*
CHD death, IS, MI, ALI	329 (3.6%)	450 (4.9%)	0.72 (0.63-0.83)	<0.0001
CV death, IS, MI, ALI	389 (4.3%)	516 (5.7%)	0.74 (0.65-0.85)	<0.0001
Mortality	313 (3.4%)	378 (4.1%)	0.82 (0.71-0.96)	0.01

* pre-specified threshold P=0.0025

Hemostasis and platelet P2Y12-inhibition



Elective aortic aneurysm repair



Acute aortic dissection with clopidogrel

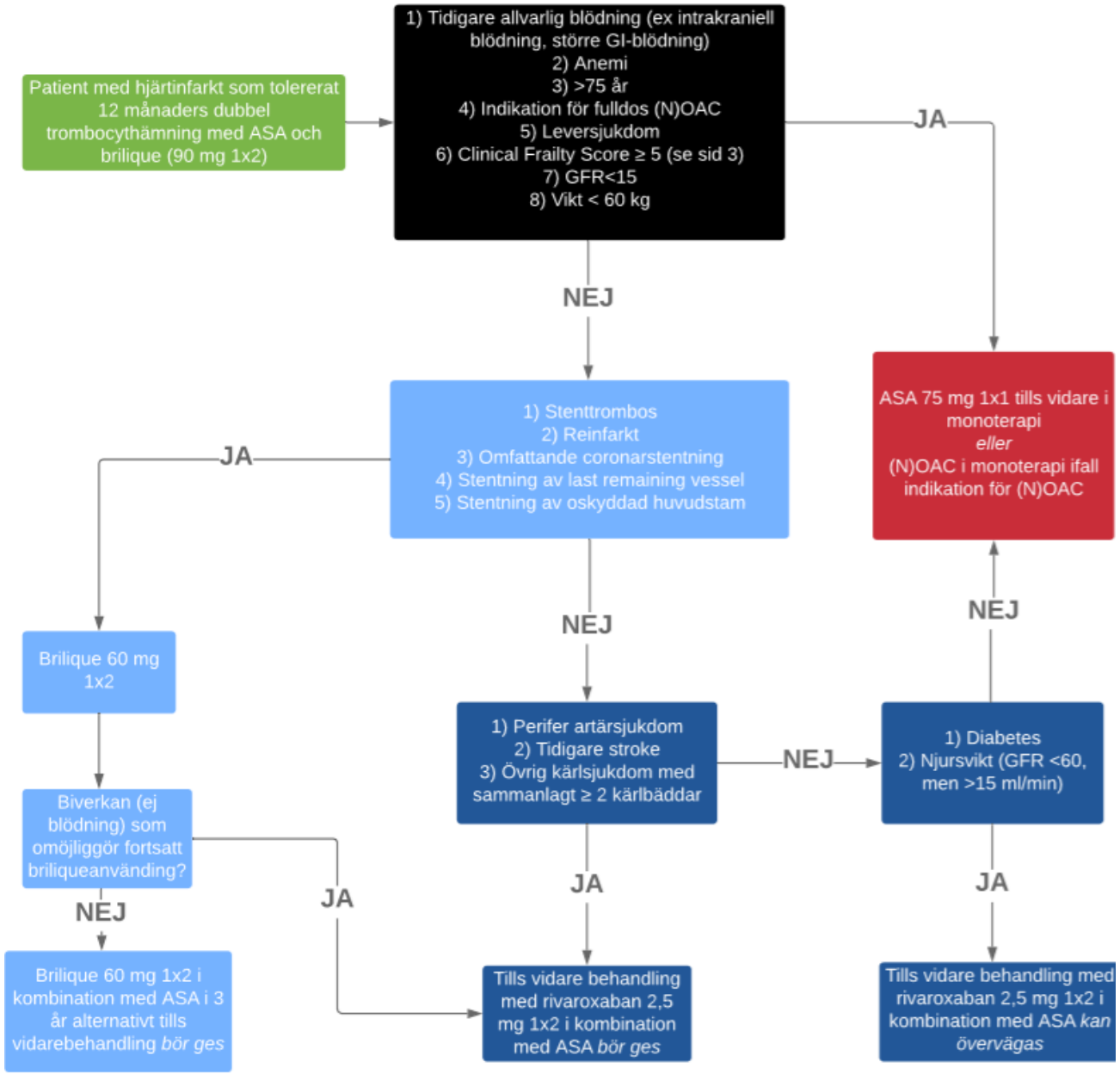
Blood from swabs after weaning of cardio-pulmonary bypass

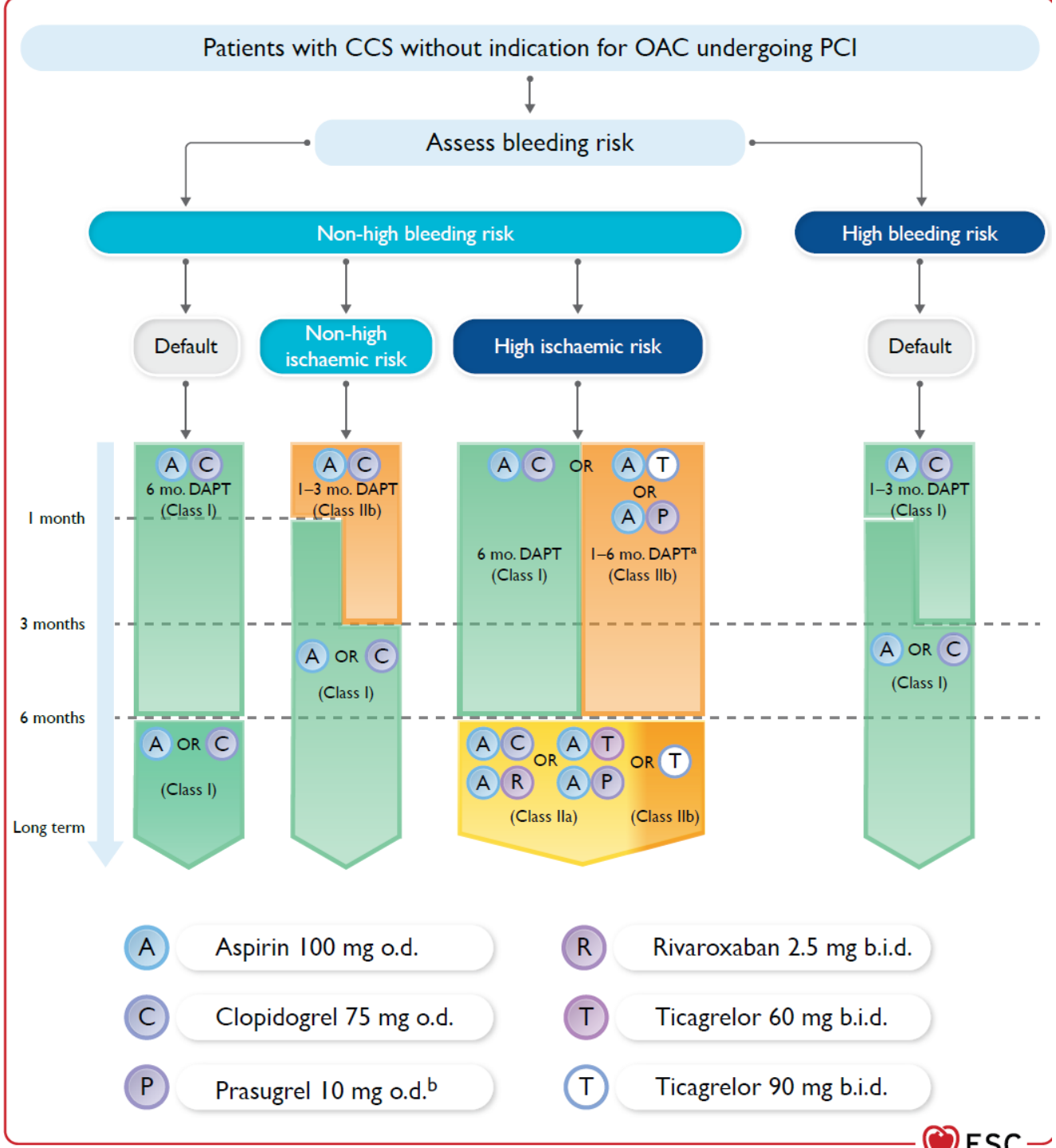
Image courtesy of Dr Emma Hansson, footage by Dr Carl Johan Malm

Adding a second antithrombotic agent to aspirin for extended long-term secondary prevention should be considered in patients at enhanced ischaemic risk[c] and without high bleeding risk[d] (options and definitions in *Table 8* and in the Supplementary data online, *Tables S2 and S3*).[592–594]	IIa	A

Långtidsbehandling med anti-hemostatiska läkemedel vid AKS efter avslutande av 12 månaders Brilique

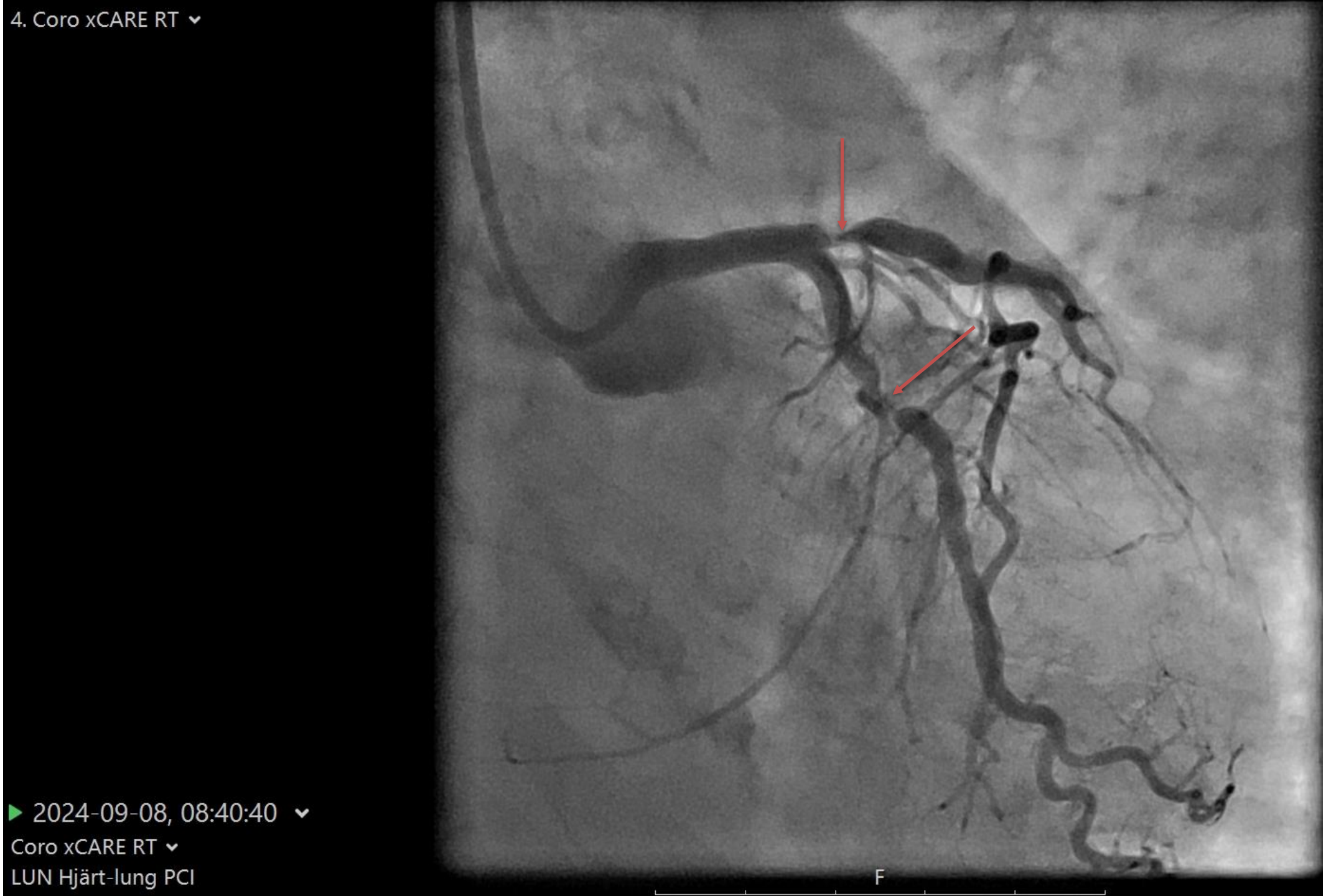
Nedan är endast en rekommendation, där det alltid är läkarens totala bedömning som gäller.

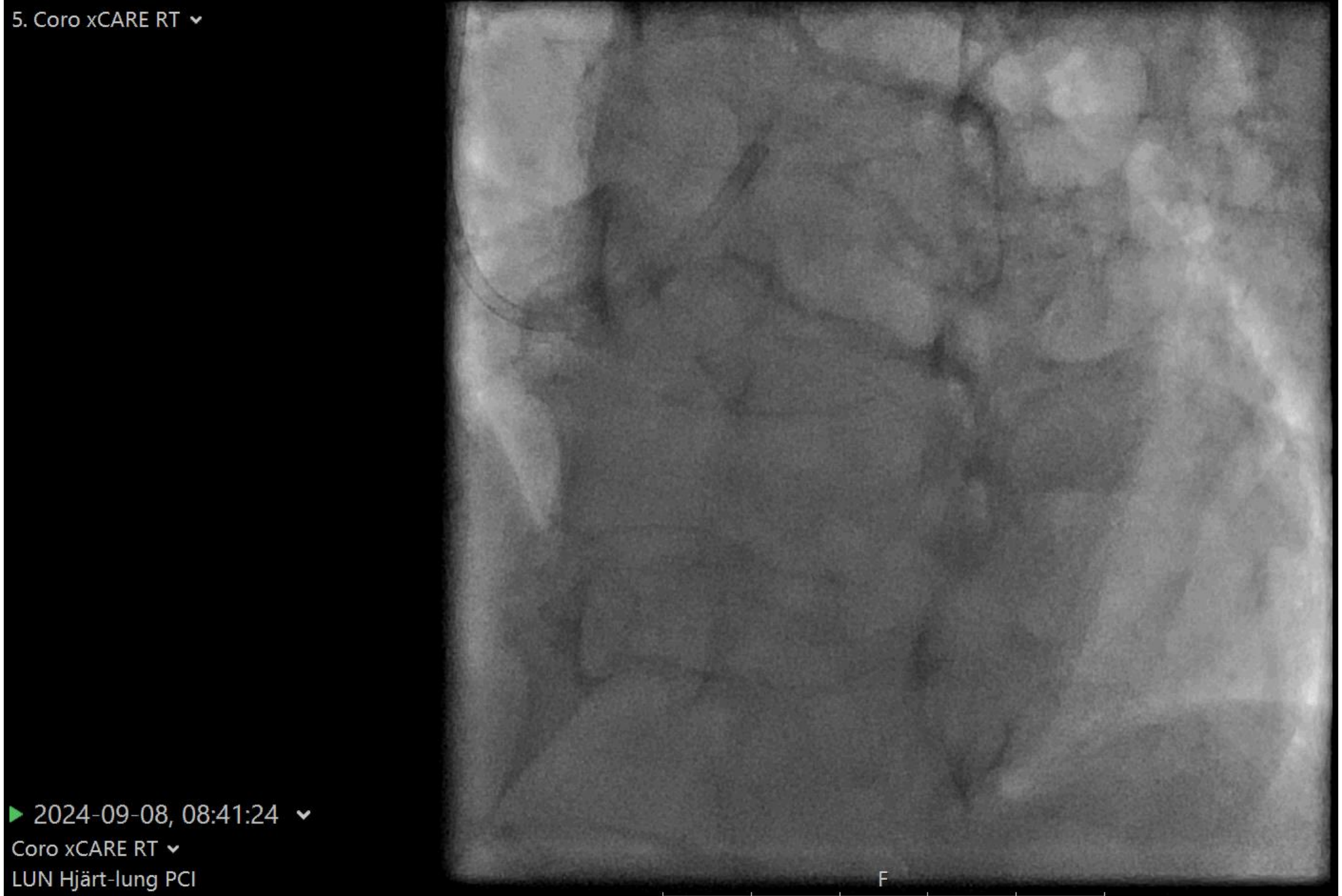



Patients with CCS without indication for OAC undergoing PCI
Assess bleeding risk
Non-high bleeding risk
High bleeding risk
Default
Non-high ischaemic risk
High ischaemic risk
Default
1 month
3 months
6 months
Long term
A C
6 mo. DAPT
(Class I)
A C
1–3 mo. DAPT
(Class IIb)
A OR C
(Class I)
A C OR A T OR A P
6 mo. DAPT
(Class I)
1–6 mo. DAPT[a]
(Class IIb)
A C OR A T A R OR A P
(Class IIa)
OR T
(Class IIb)
A C
1–3 mo. DAPT
(Class I)
A OR C
(Class I)
A OR C
(Class I)
A Aspirin 100 mg o.d.
C Clopidogrel 75 mg o.d.
P Prasugrel 10 mg o.d.[b]
R Rivaroxaban 2.5 mg b.i.d.
T Ticagrelor 60 mg b.i.d.
T Ticagrelor 90 mg b.i.d.

Patientfall

- Kvinna, 72 år gammal.
- Lateral STEMI
- Hypertoni. Hypotyreos.
- LDL 3.1, TG 2.3, HDL 1.1 noteras i lablistan från endokrin.


4. Coro xCARE RT
2024-09-08, 08:40:40
Coro xCARE RT
LUN Hjärt-lung PCI
F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2024-09-08, 08:41:24
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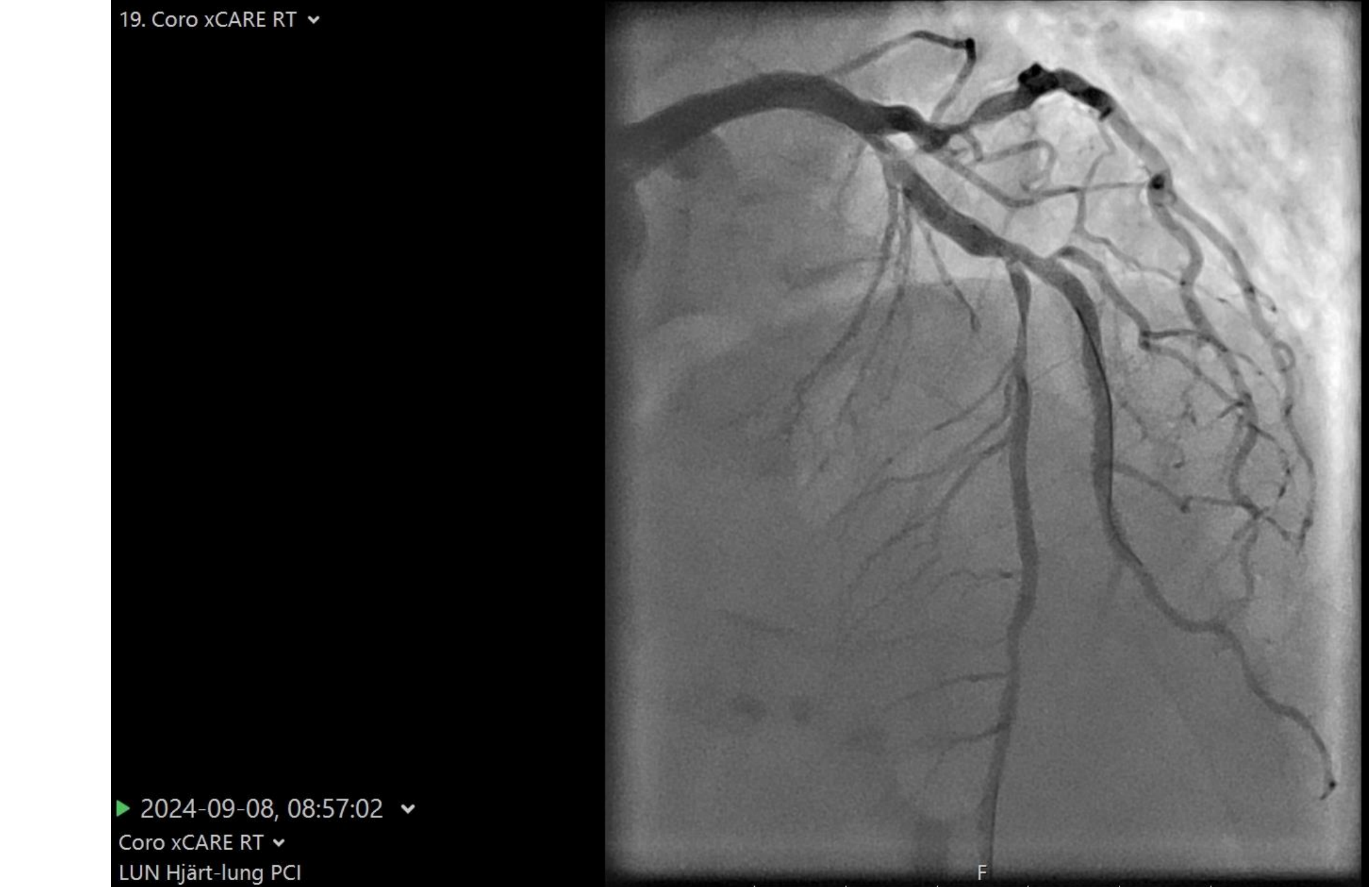
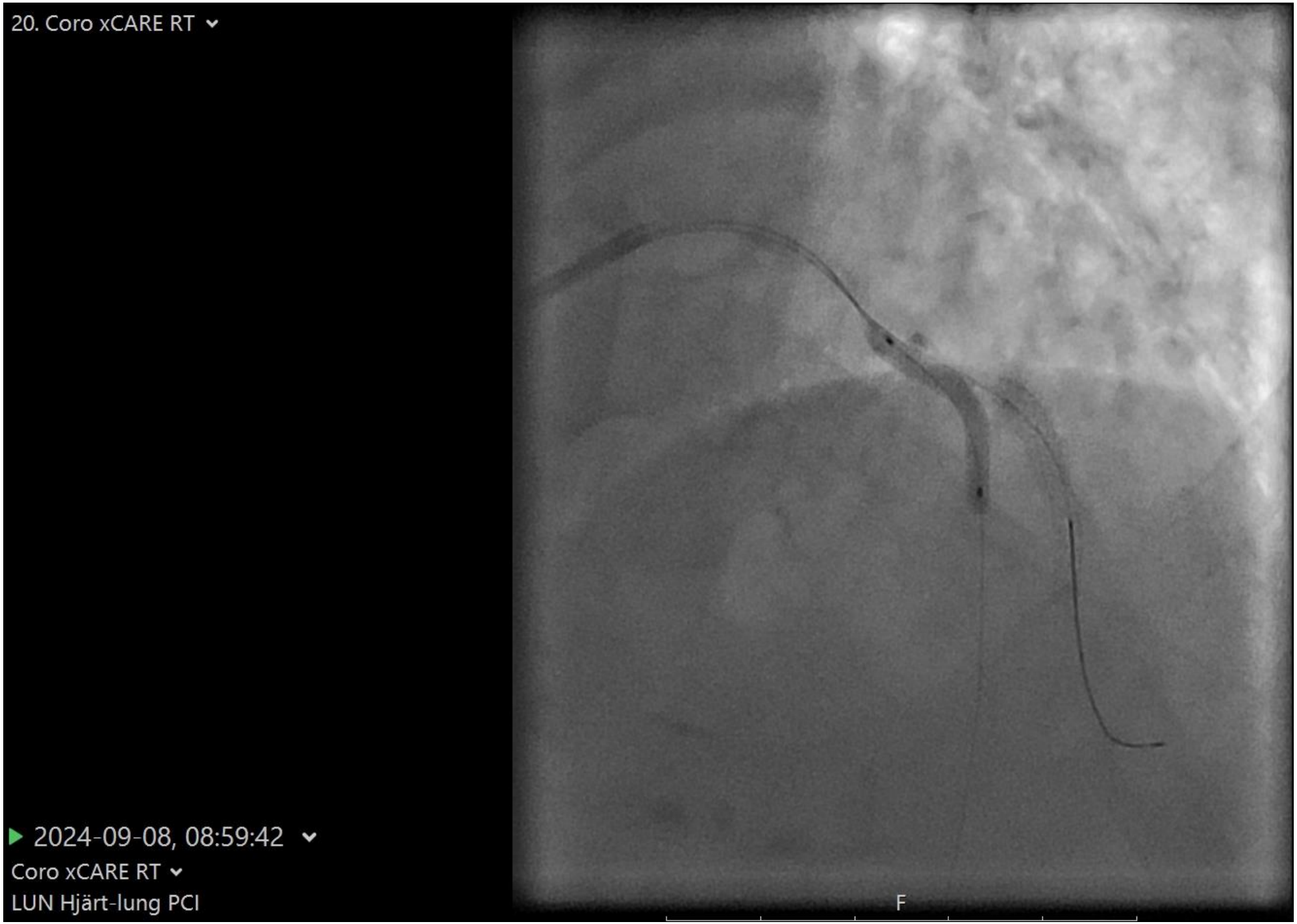
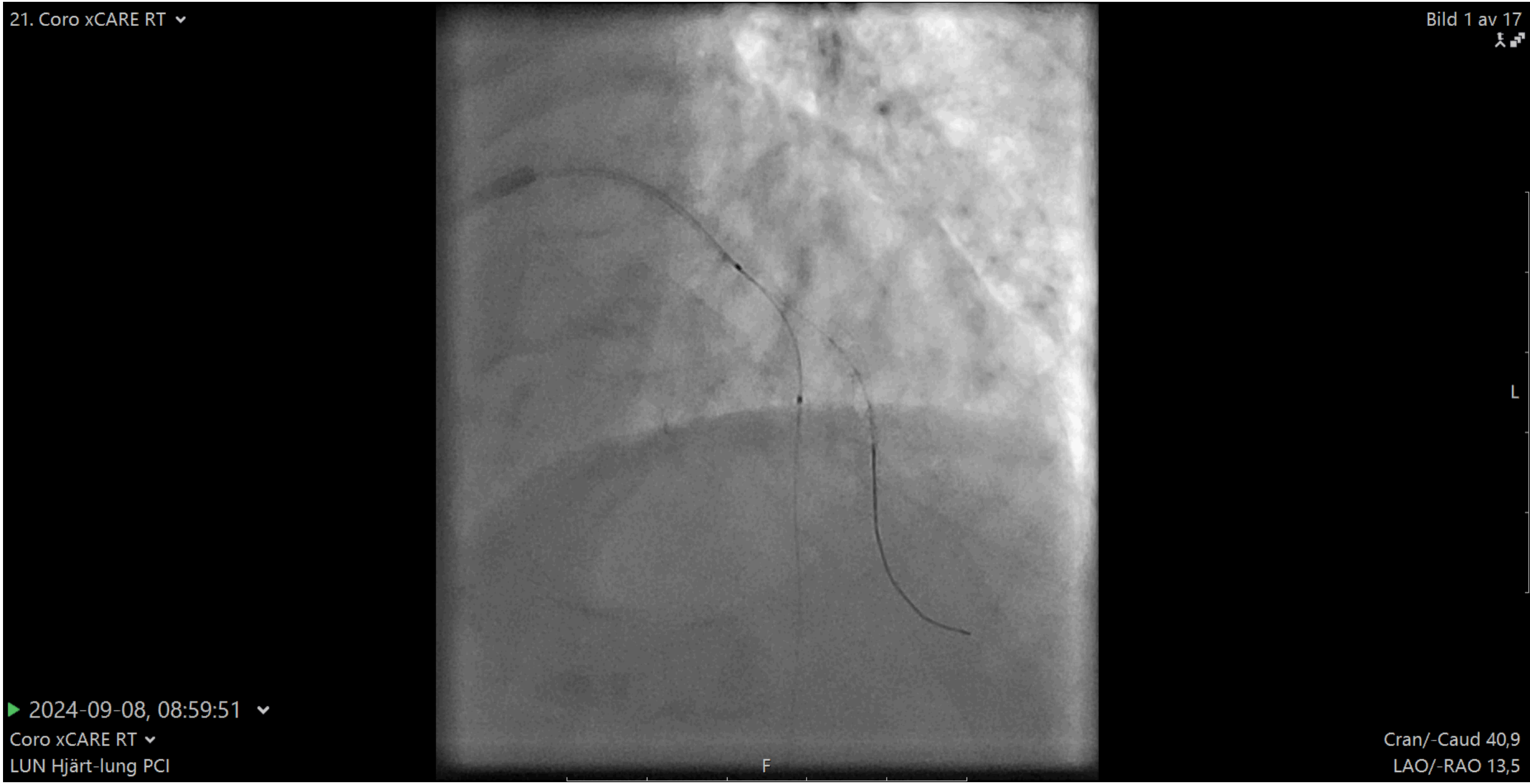
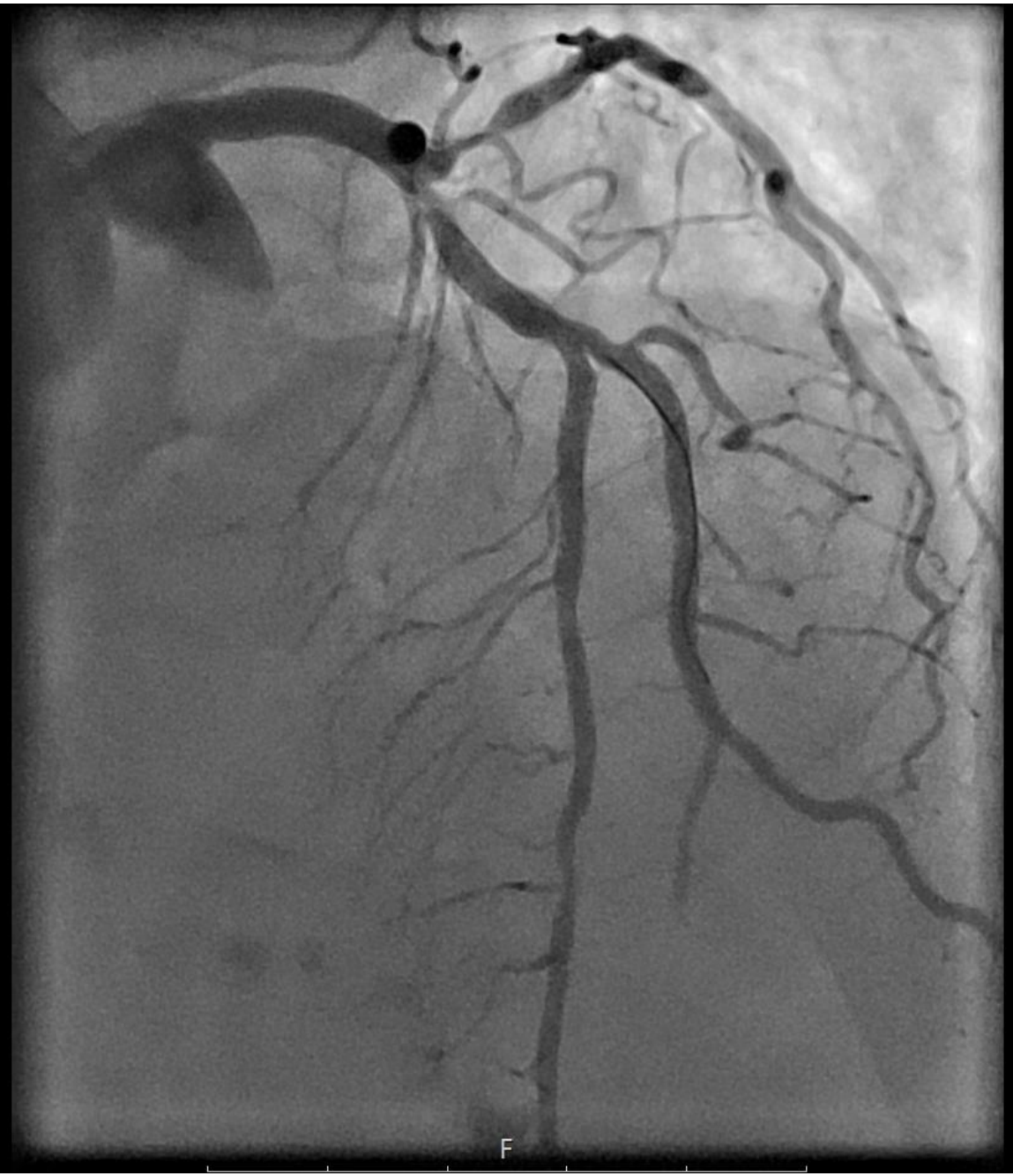
LUN Hjärt-lung PCI
F


19. Coro xCARE RT
2024-09-08, 08:57:02
Coro xCARE RT
LUN Hjärt-lung PCI
F

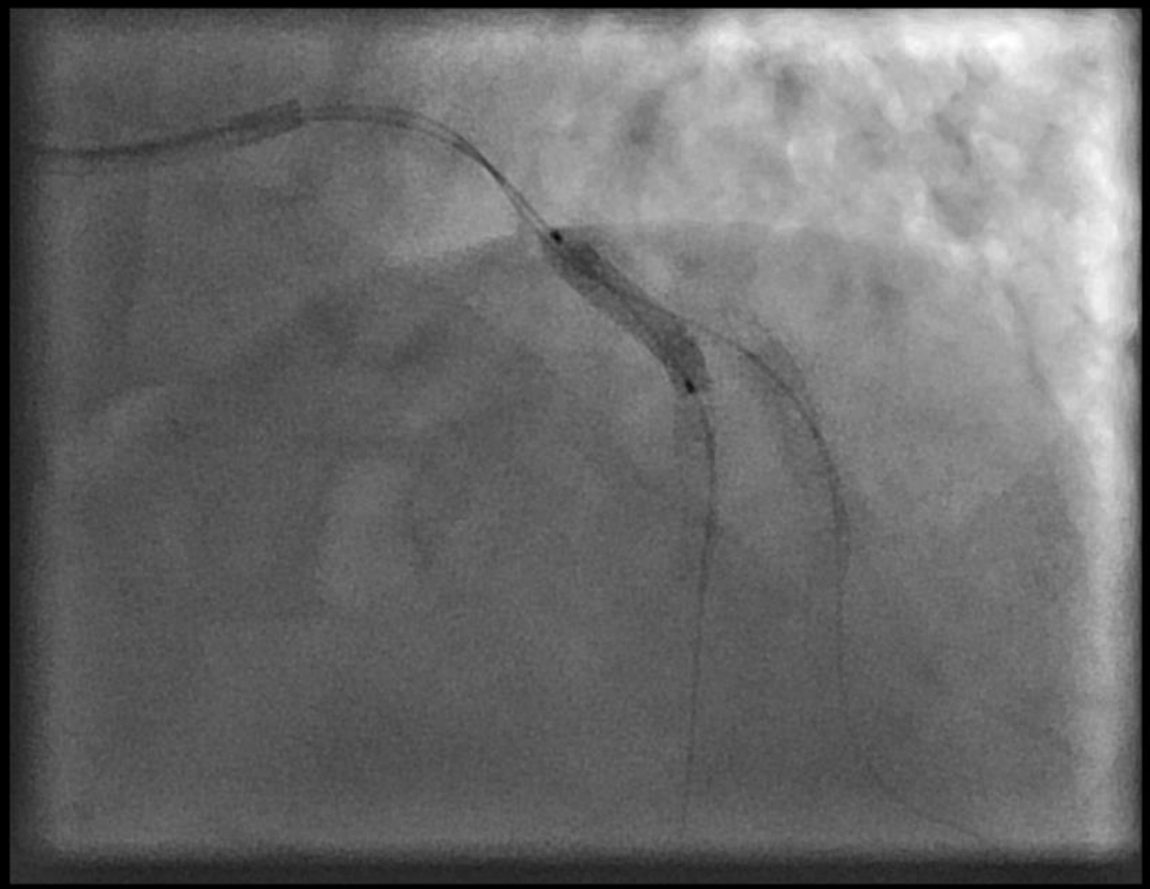

20. Coro xCARE RT
2024-09-08, 08:59:42
Coro xCARE RT
LUN Hjärt-lung PCI
F


21. Coro xCARE RT
Bild 1 av 17
L
2024-09-08, 08:59:51
Coro xCARE RT
LUN Hjärt-lung PCI
F
Cran/-Caud 40,9
LAO/-RAO 13,5


26. Coro xCARE RT
Bild 16 av 3
2024-09-08, 09:05:19
Coro xCARE RT
LUN Hjärt-lung PCI
F
Cran/-Caud 40,
LAO/-RAO 13,

39. Coro xCARE RT

Bild 3 av 4



2024-09-08, 09:16:14

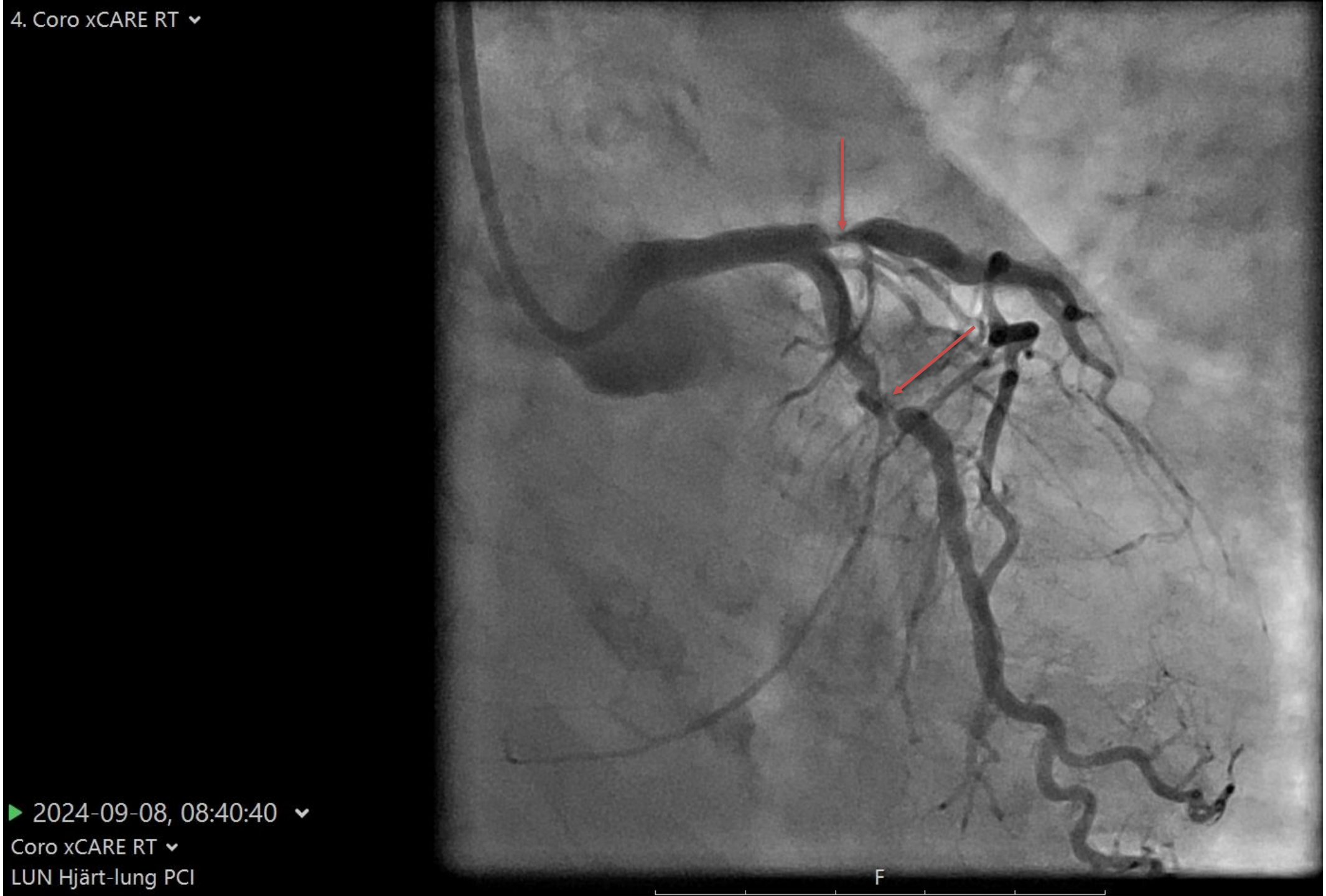
Coro xCARE RT

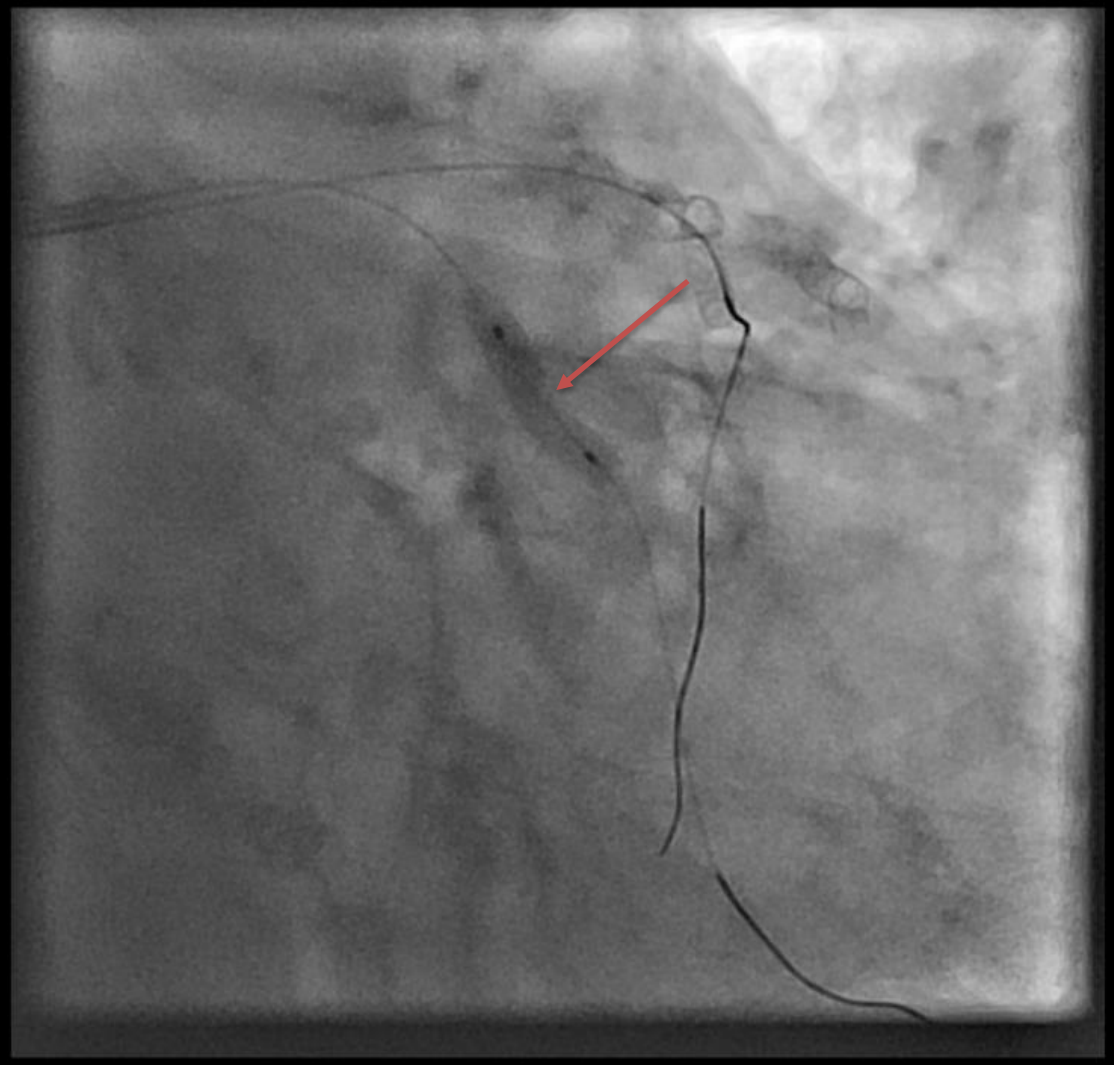
LUN Hjärt-lung PCI

F

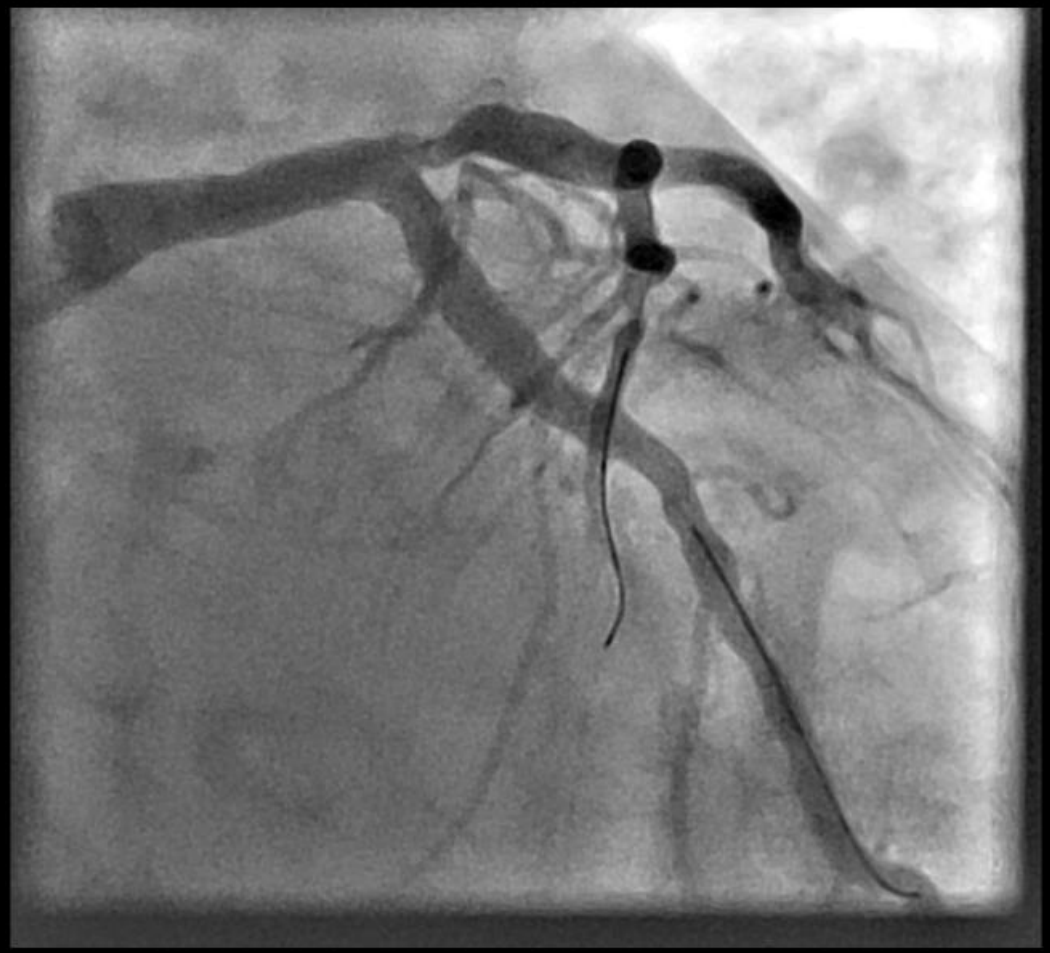
Cran/-Caud 40,9

LAO/-RAO 13,5


4. Coro xCARE RT
2024-09-08, 08:40:40
Coro xCARE RT
LUN Hjärt-lung PCI
F

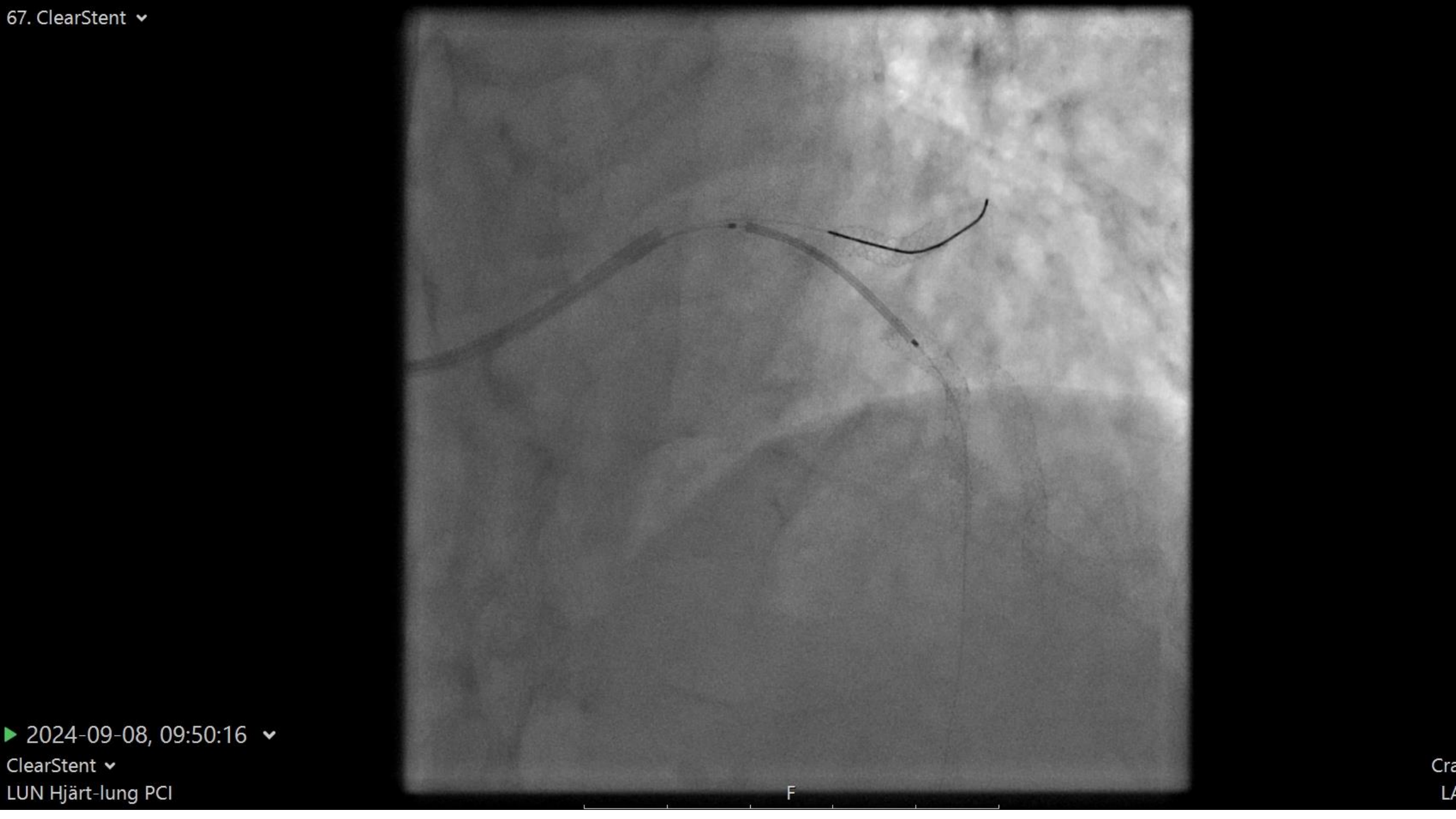


59. Coro xCARE RT

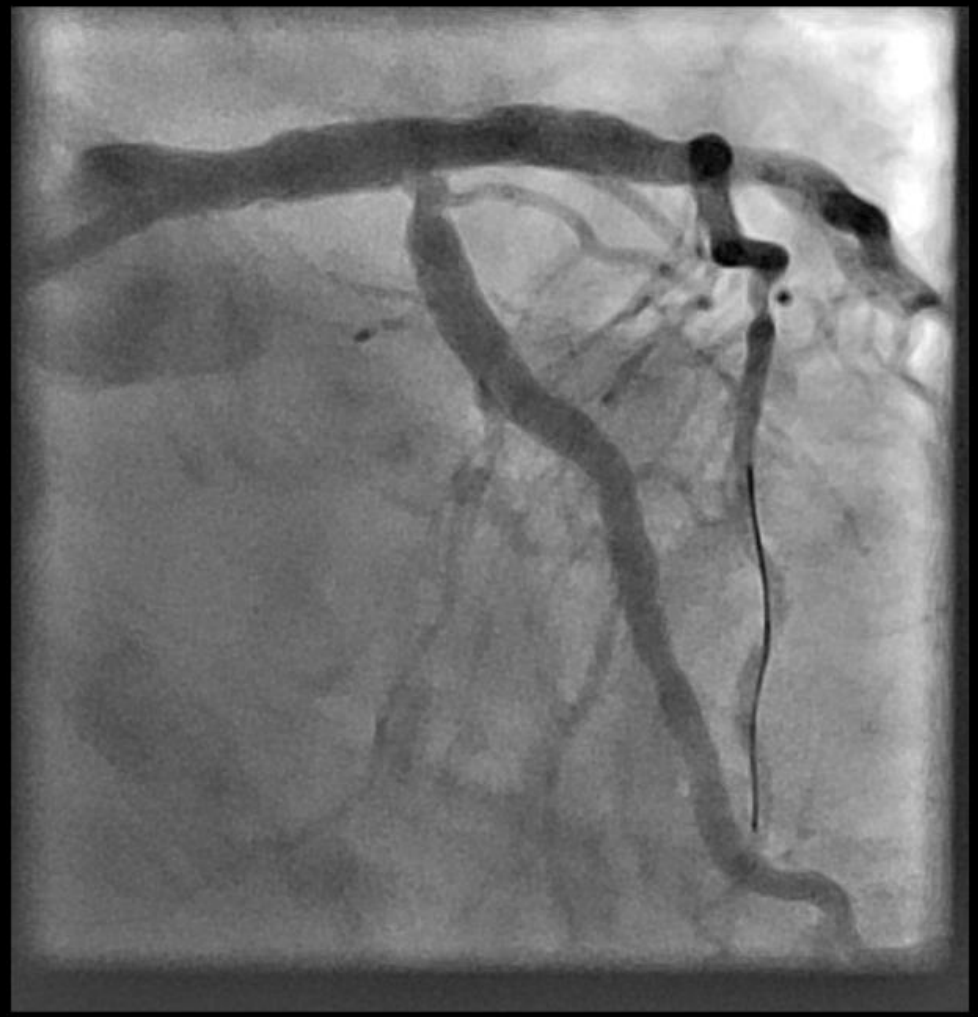


2024-09-08, 09:41:31

Coro xCARE RT


67. ClearStent
2024-09-08, 09:50:16
ClearStent
LUN Hjärt-lung PCI
F

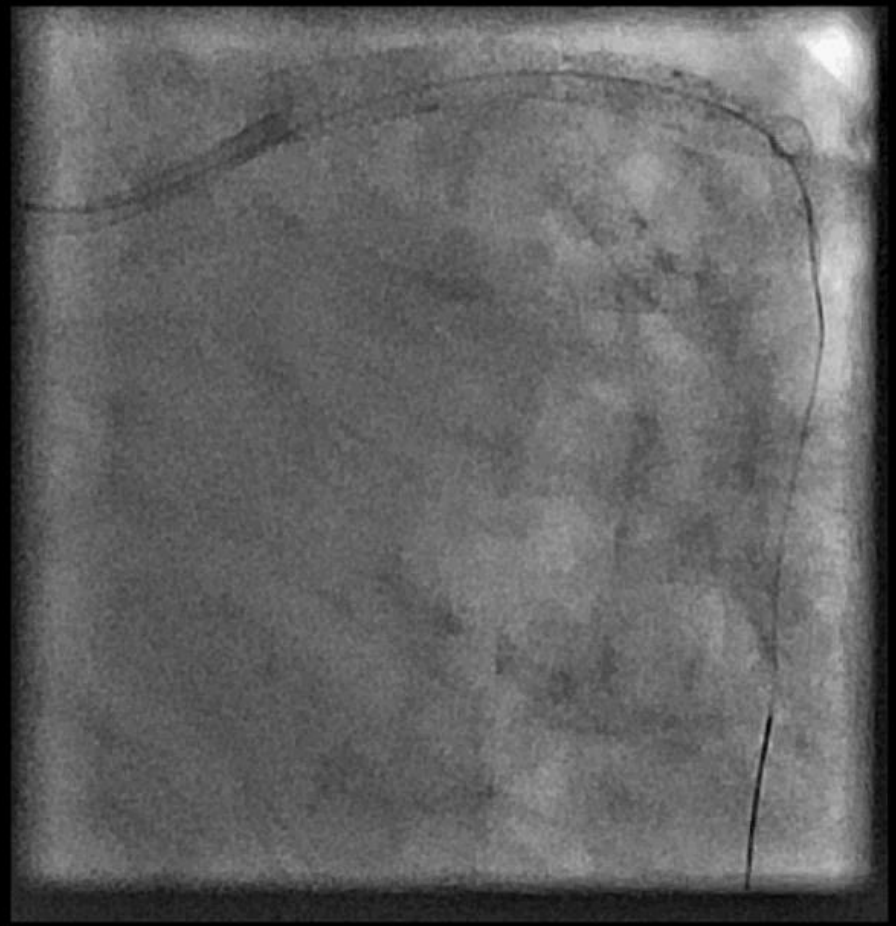
75. Coro xCARE RT



2024-09-08, 09:55:06

Coro xCARE RT

81. Coro xCARE RT

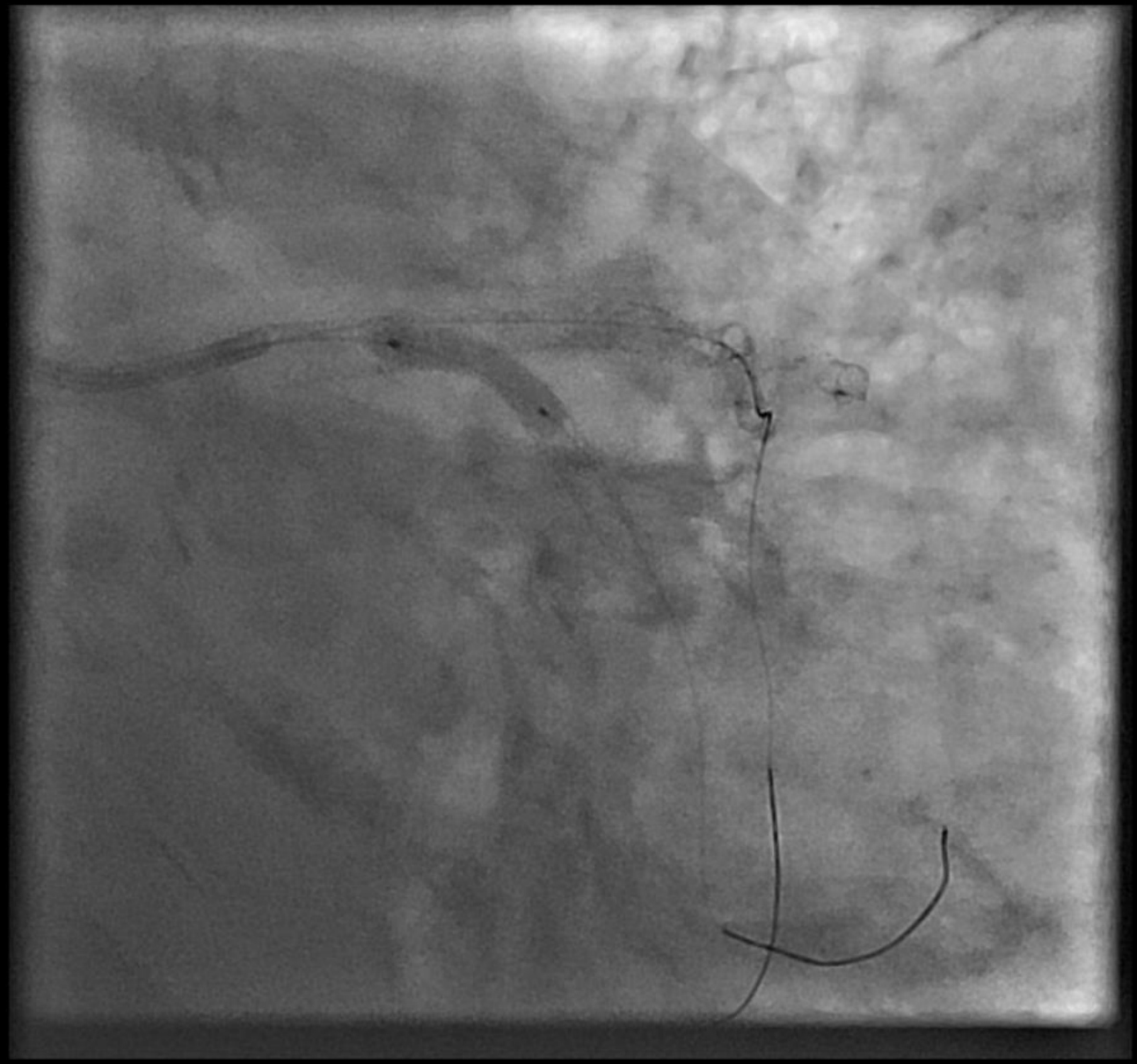


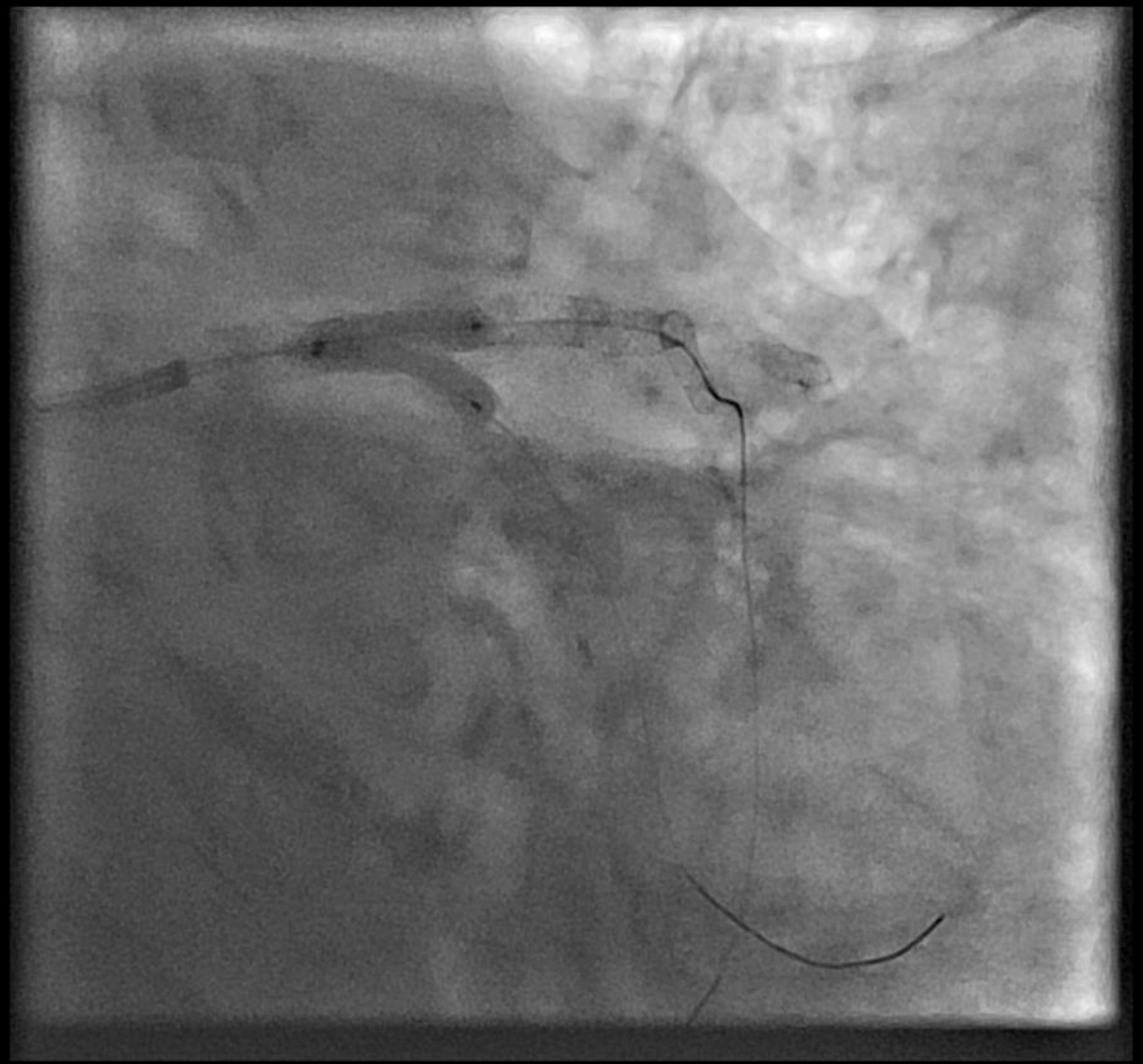
2024-09-08, 09:59:35

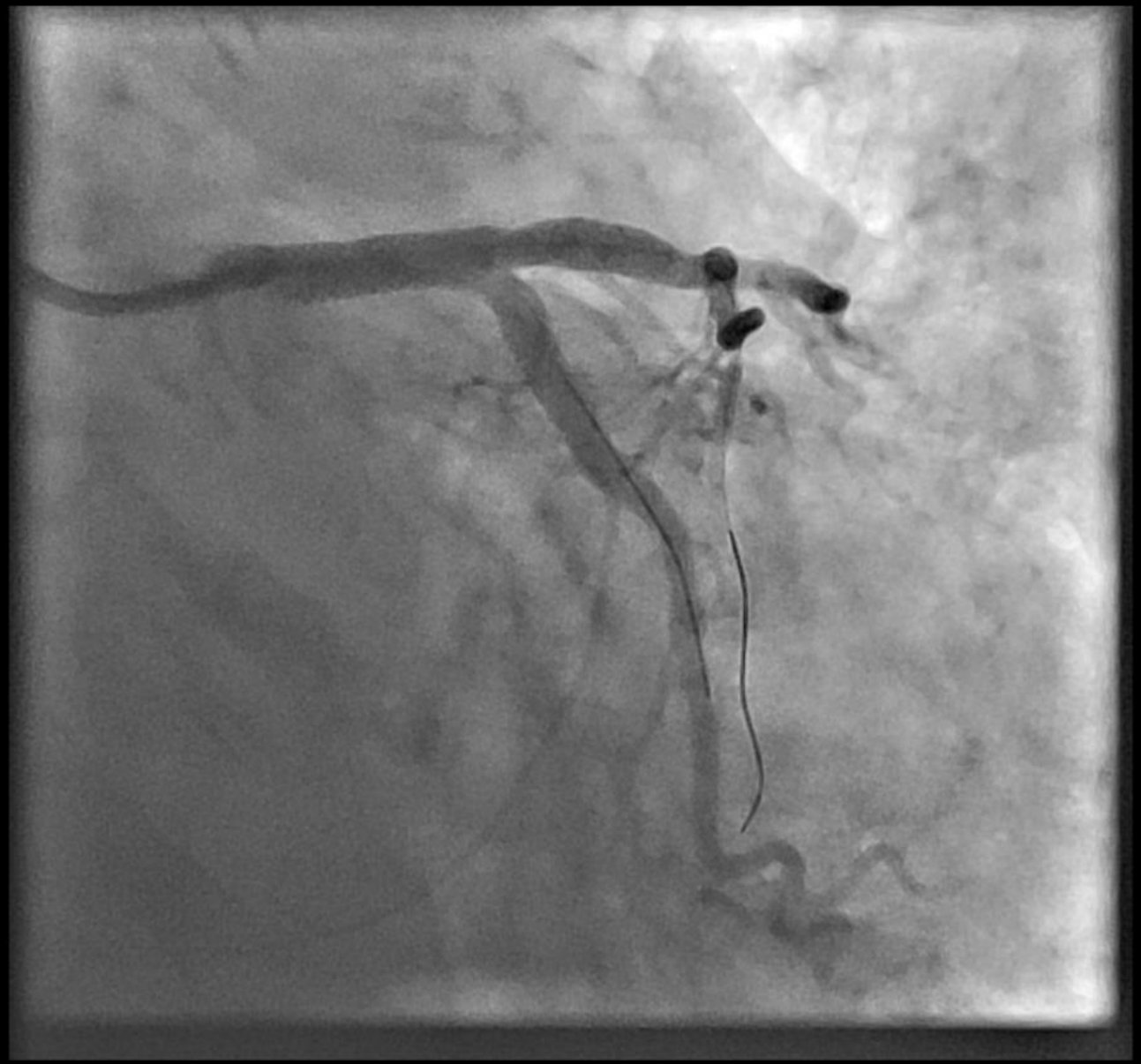
Coro xCARE RT

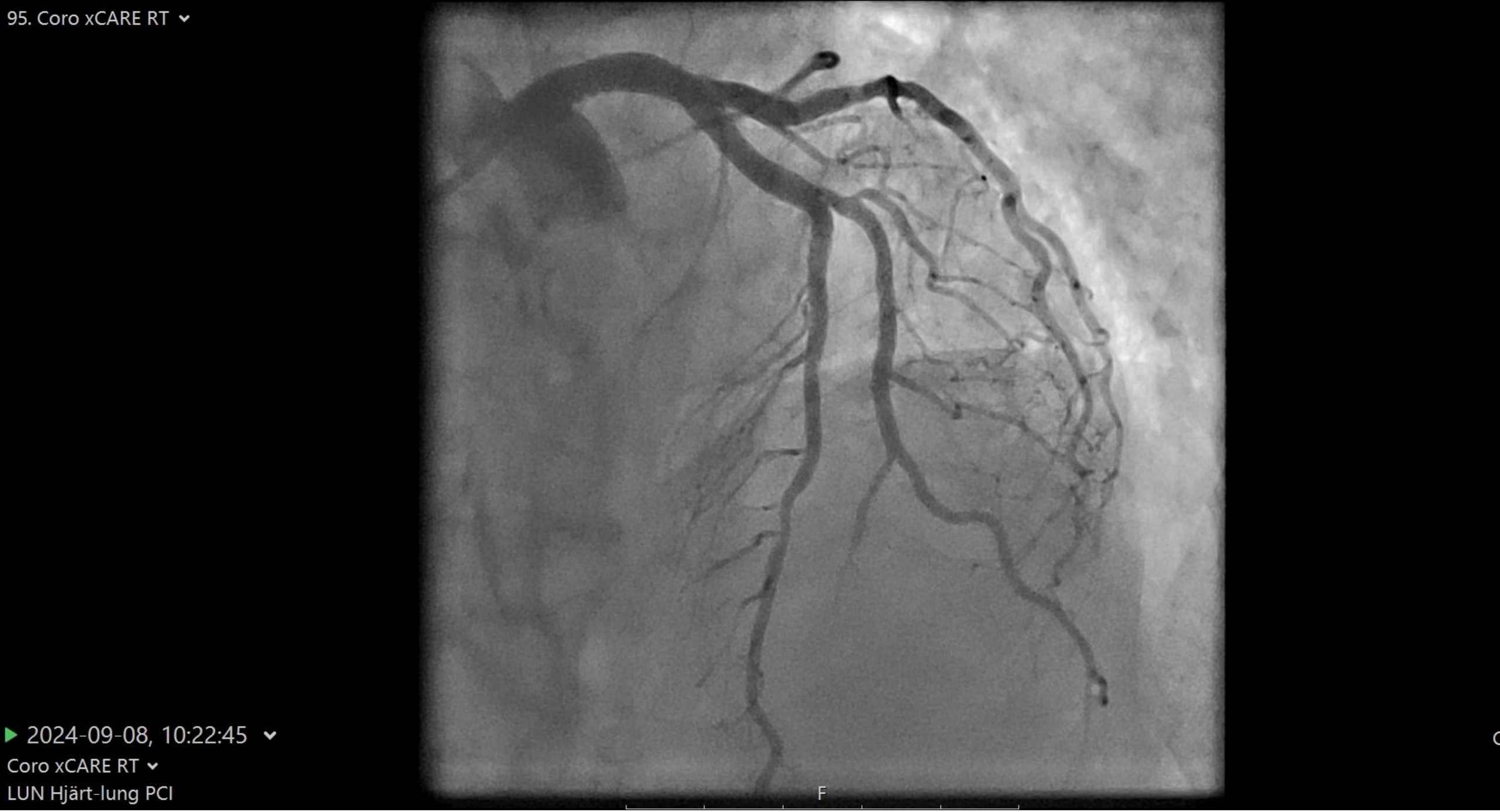
LUN Hjärt-lung PCI

F








95. Coro xCARE RT
2024-09-08, 10:22:45
Coro xCARE RT
LUN Hjärt-lung PCI
F

LM: Ua.

LAD: Tät stenos x flera. Ockluderad D1.

Cx: Tät stenos.

RCA: Ateromatos.

Komplex PCI med stentning mot D1 + mellersta + proximala LAD ut i LM samt stent i Cx + DEB i Cx-ostiet.

Heparin + Efient. Sion blue ledare ut i LAD + D1. Ballongvidgning av D1 med 1.5 + 2.0 mm ballong. Stentning med Onyx 2.5x26.

Ballongvidgning av mellersta LAD med 2.5 mm ballong. Stetnning med 2.5x18. Noterar att en liten underskänkel till D1 har distal emboli. Lätter ut ledare i denna. Försiktig ballongvidgning med 1.25 mm ballong. Får dock inget flöde och lämnar kärlet därhän.

Därefter stentning av proximala LAD över D1 med Onyx 2.75x12. POT med 4.0 mm ballong.

IVUS som inte kommer ut distalt utan hakar i kalk/stentkant men vi får en uppfattning om proximala LAD + LM.

Därefter sion blue ledare ut i Cx. Ballongvidgning med 3.0 mm ballong. IVUS. Cutting balloon 3.0 mm.

Stentning med Onyx 3.5x18. POT med 4.0 mm NC-ballong. Därefter ballongvidgning av LM ut i LAD med 4.0 mm NC-ballong. God expansion. Stentning av LM ut i LAD med Onyx 4.0x26. POT med 5.0 mm NC-ballong. Diskret kantdissektion och förlänger i LM ut i ostiet med Onyx 5.0x12.

Viss plackskifte i proximala Cx som också uppvisar mycket kraftig rörlighet. Vill därmed undvika stentning där om jag kan. Rewirar Cx med ny sion blue ledare. Ballongvidgning av Cx-ostiet samt kissing balloon LAD/Cx med 3.5/3.5 mm ballong.

Därefter DEB av Cx-ostiet med 4.0x15 Agent. Avslutar med kissing balloon LAD/Cx med 3.5/3.5 mm. Gott resultat.

Rekommenderar förlängd DAPT-behandling (c:a 3 år avhängigt av hur pat tål behandling). Aggressiv lipidsänkning där Vazkepa som tillägg kan övervägas polikliniskt.

Ett multidisciplinärt omhändertagande!!
PCI-lab - HIA - Mottagning

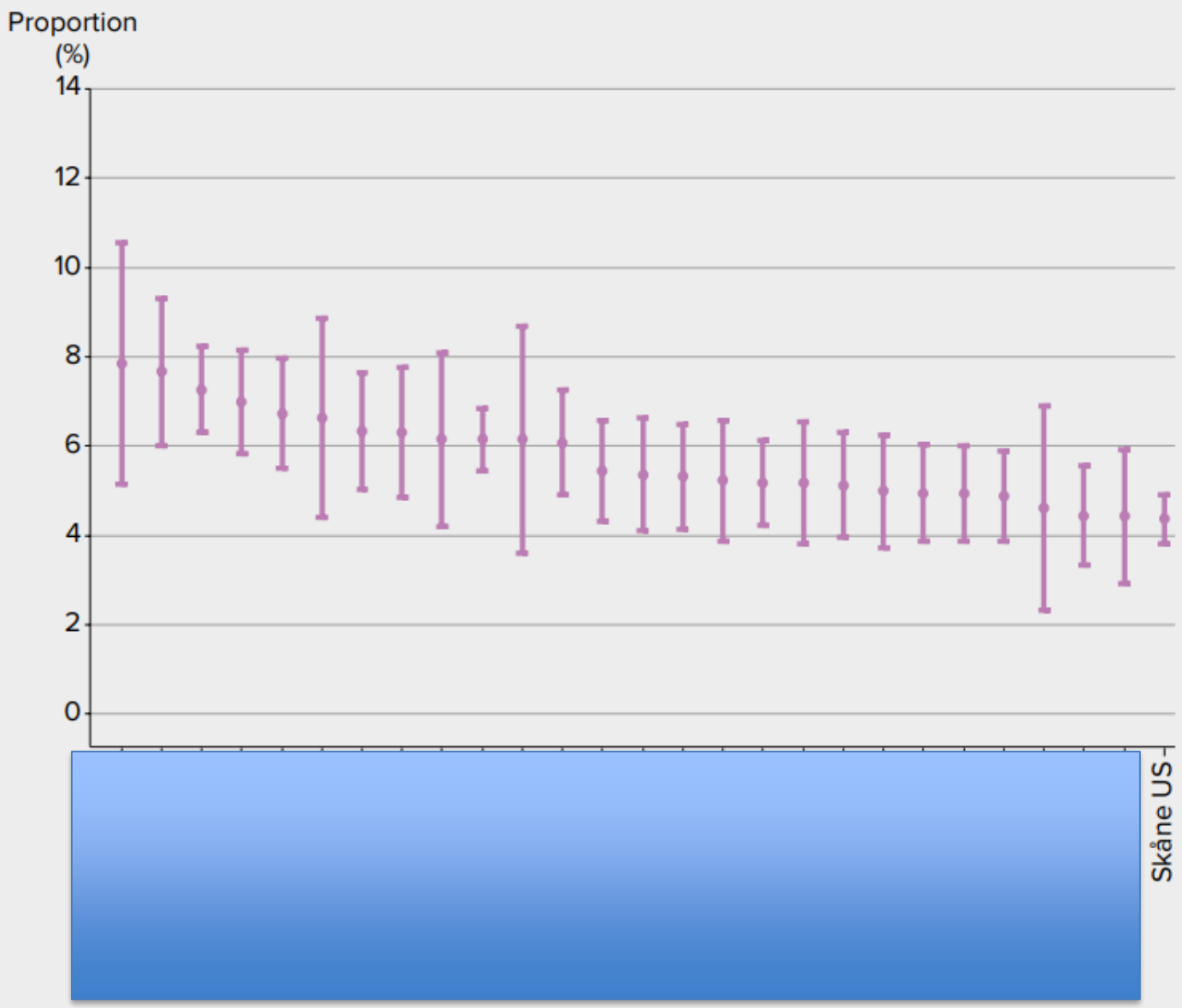


Figure 13. Thirty-day mortality after PCI in STEMI patients, per hospital, 2014–2023 (mean value and 95 % CI).

Sammanfattning

Plackbiologi är komplicerat!

Triglycerider – även modesta stegringar – signalerar en patient med hög kardiovaskulär risk!

Trots modern behandling och optimering av konventionella riskfaktorer så kvarstår en “residual cardiovascular risk” hos många patienter.

Utöver konventionell sekundärprevention, kan nya terapeutiska behandlingsmöjligheter kan erbjuda förbättrade outcomes hos högriskpatienter. Dessa inkluderar ikosapentetyl, colchicine samt förlängd dubbel anti-trombotisk behandling.

Multidisciplinär approach!

TACK!