Analysis of coronary ulcers: are they all the same? When to treat them?

Análisis de las úlceras coronarias: ¿Son todas iguales? ¿Cuándo tratarlas?

A Matías Rodriguez-Granillo, MD
Centro de Estudios en Cardiología Intervencionista (CECI)
Departamento de Cardiología Intervencionista, Sanatorio Otamendi, Buenos Aires, Argentina
@matiasrg

Declaración de conflictos de interés

Speaker (Honorarios)

    Novo Nordisk

    Bagó

SOLACI CACI`24
ARGENTINA
BUENOS AIRES HILTON
BUENOS AIRES, ARGENTINA
AUGUST
7 - 9
2024

Objetivo

Discutir el rol de las imágenes intravasculares en las lesiones coronarias no obstructivas, y reconocer cuando deben ser tratadas.

Cuando producen síntomas.

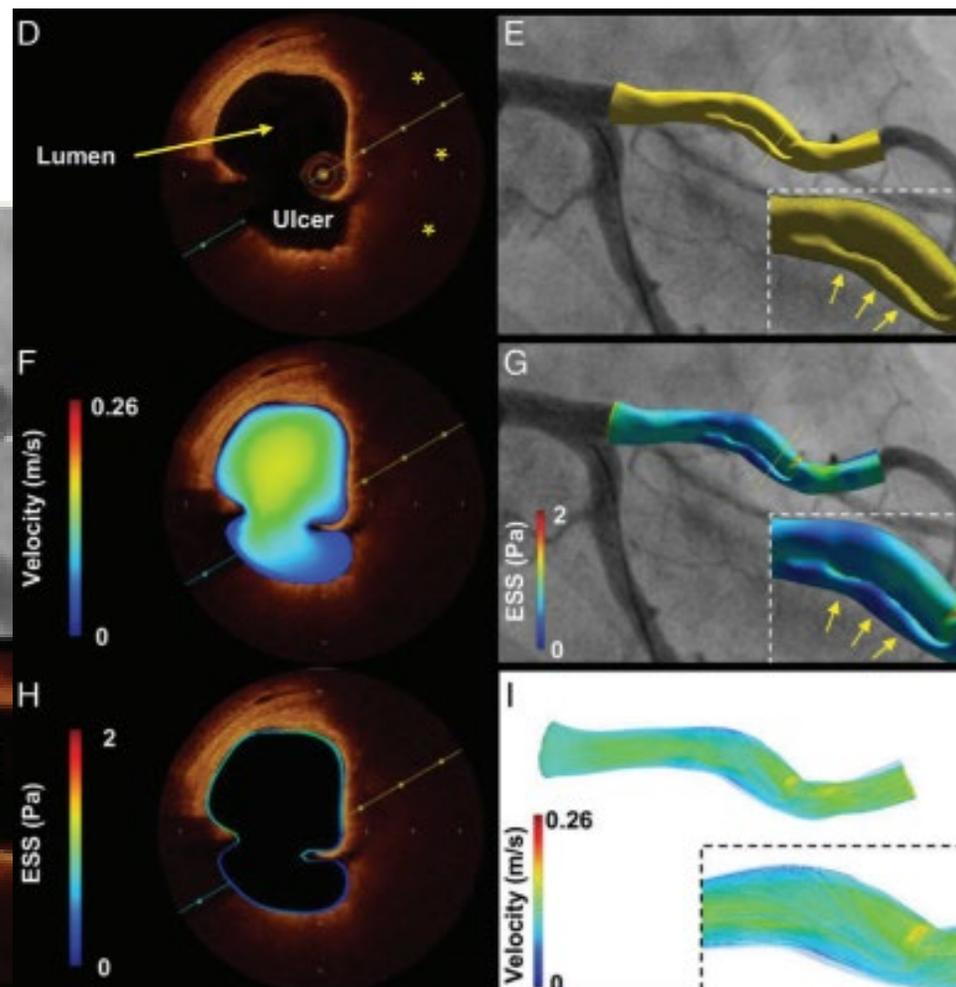# Muchas gracias!

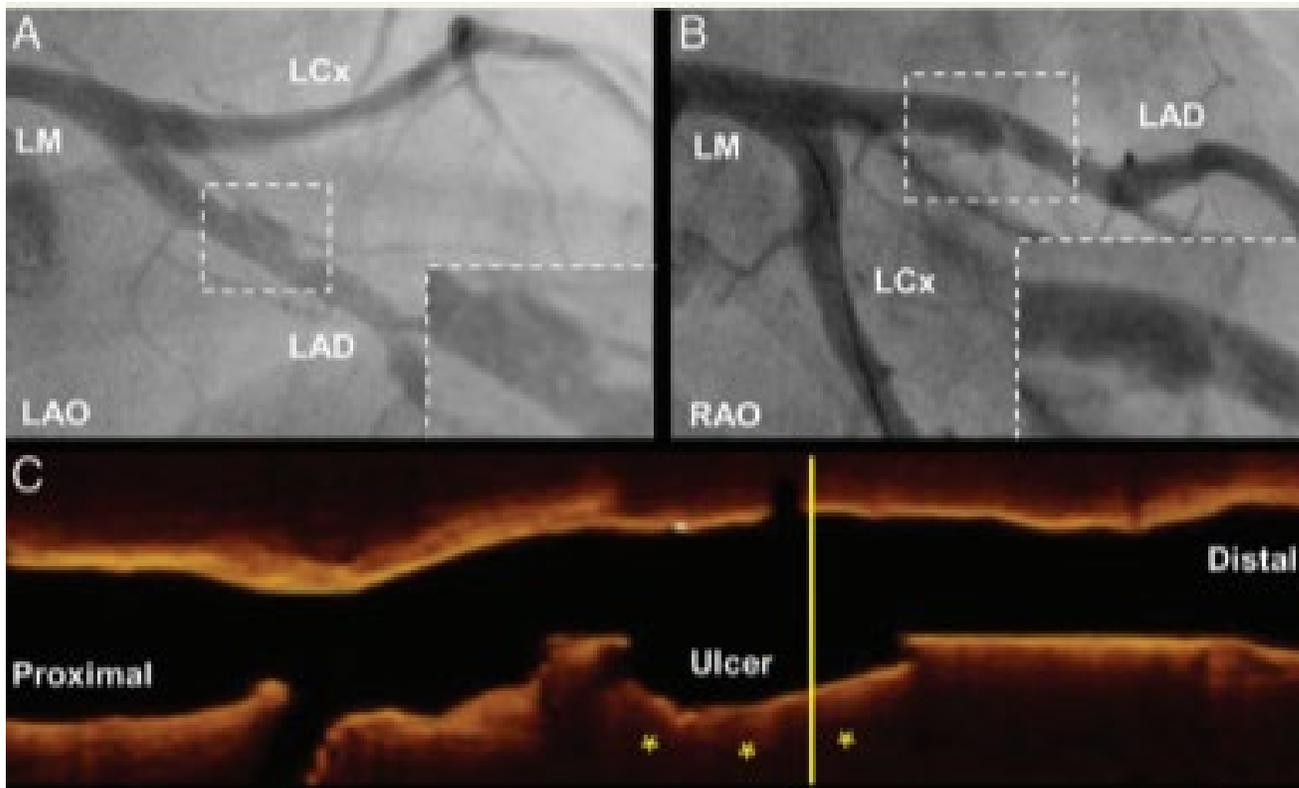A Matías Rodriguez-Granillo, MD
Centro de Estudios en Cardiología Intervencionista (CECI)
Departamento de Cardiología Intervencionista, Sanatorio Otamendi, Buenos Aires, Argentina
@matiasrg

SOLACI
CACI`24
ARGENTINA
BUENOS AIRES HILTON
BUENOS AIRES, ARGENTINA

AUGUST
7 - 9
2024

CACI

XXIX ANNUAL CONGRESS OF LATIN AMERICAN SOCIETY OF INTERVENTIONAL CARDIOLOGY
XXXIV NATIONAL ANNUAL CONGRESS OF THE ARGENTINE COLLEGE OF INTERVENTIONAL CARDIOANGIOLOGY

www.solacicongress.org

Definir de que estamos hablando…

Se calculó el shear stress por dinámica de flujo computacional mediante OCT

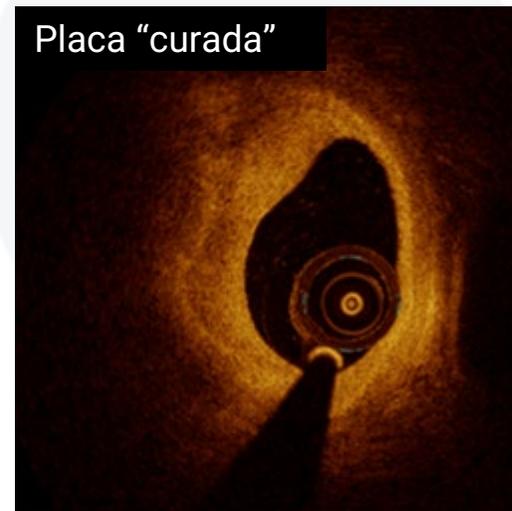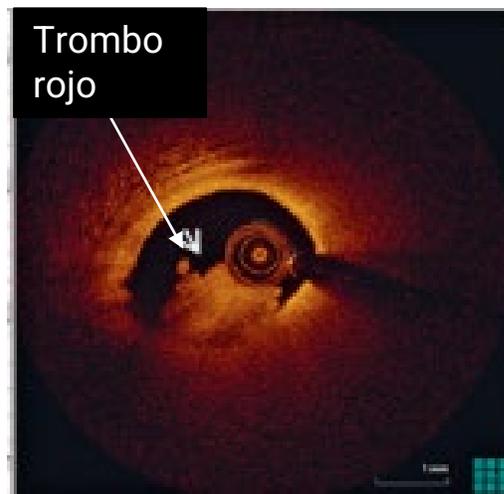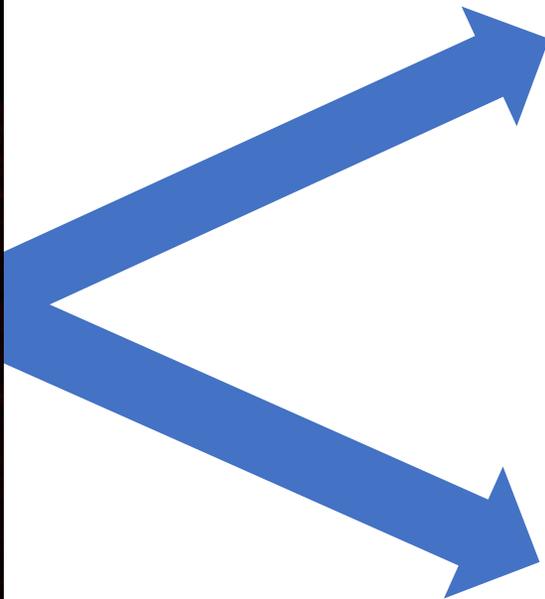Giannopoulos AA et al. Eur Heart J Cardiovasc Imaging. 2015 Sep;16(9):1042.

Definir de que estamos hablando…
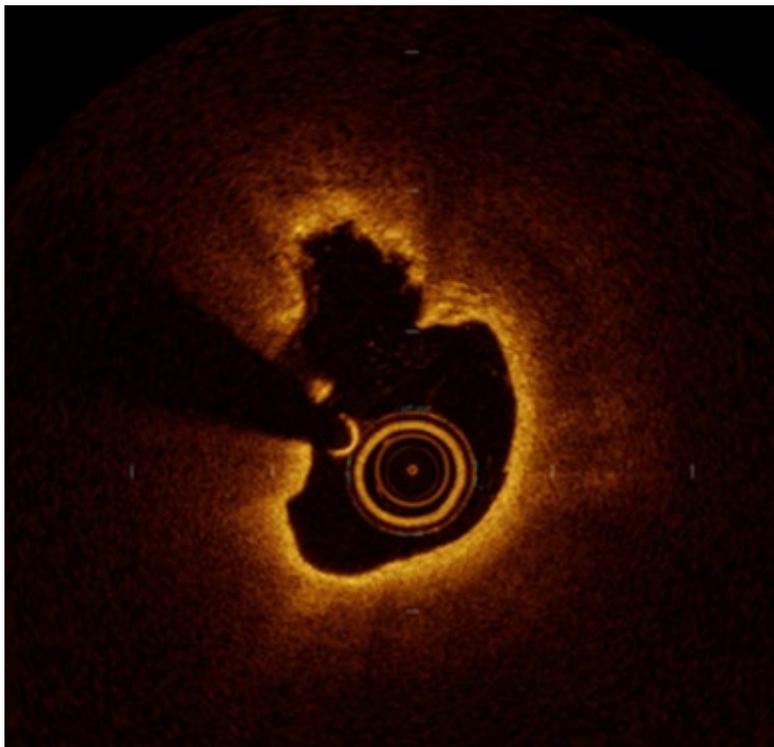
Úlcera de placa lipídica, con o sin formación de trombo.
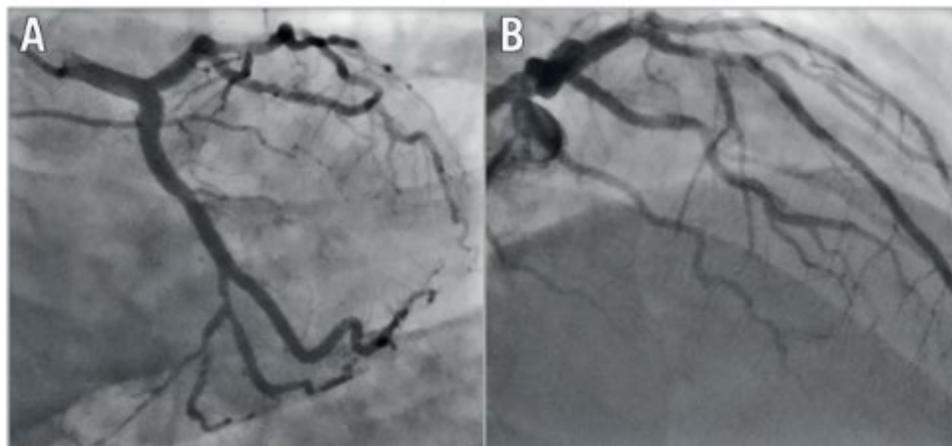
Trombo rojo

Placa "curada"

Si hay síntomas y lesión identificada, no hay dudas!

Definir de que estamos hablando…

SCACEST ANTERIOR

CCG

OCT

Trombo rojo

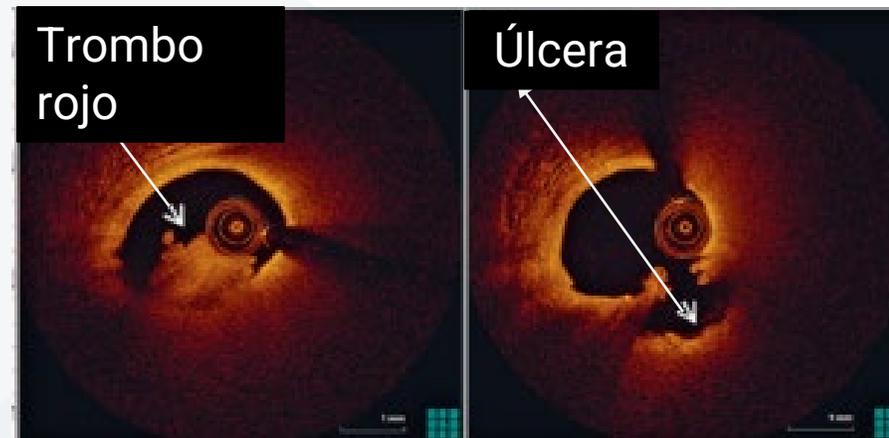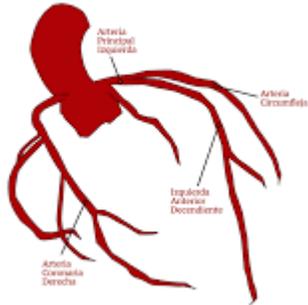Úlcera
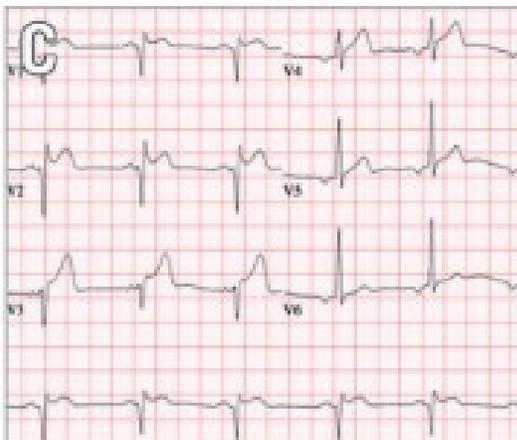
Lesiones no obstructivas ni culpables del cuadro que sucitó el estudio, que por imagenes no invasivas o invasivas tienen características de vulnerabilidad.

?

Table 2. Kaplan–Meier Estimates for Cumulative Rates of Major Adverse Cardiovascular Events at 3 Years.*

Event	Events Related to Culprit Lesions	Events Related to Nonculprit Lesions	Indeterminate Events	All Events
	percent (number of patients)			
Composite cardiac events†	12.9 (83)‡	11.6 (74)	2.7 (17)	20.4 (132)
Death from cardiac causes, cardiac arrest, or myocardial infarction	2.2 (14)	1.0 (6)	1.9 (12)	4.9 (31)
Death from cardiac causes	0.2 (1)	0	1.8 (11)	1.9 (12)
Cardiac arrest	0.3 (2)	0	0.2 (1)	0.5 (3)
Myocardial infarction	2.0 (13)	1.0 (6)§	0.3 (2)	3.3 (21)
Rehospitalization for unstable or progressive angina	11.5 (74)	10.8 (69)	0.8 (5)	17.5 (113)
Other events				
Revascularization	10.9 (70)	10.5 (67)	0	17.1 (110)
Stent thrombosis¶	2.0 (13)	0	1.3 (8)	3.3 (21)
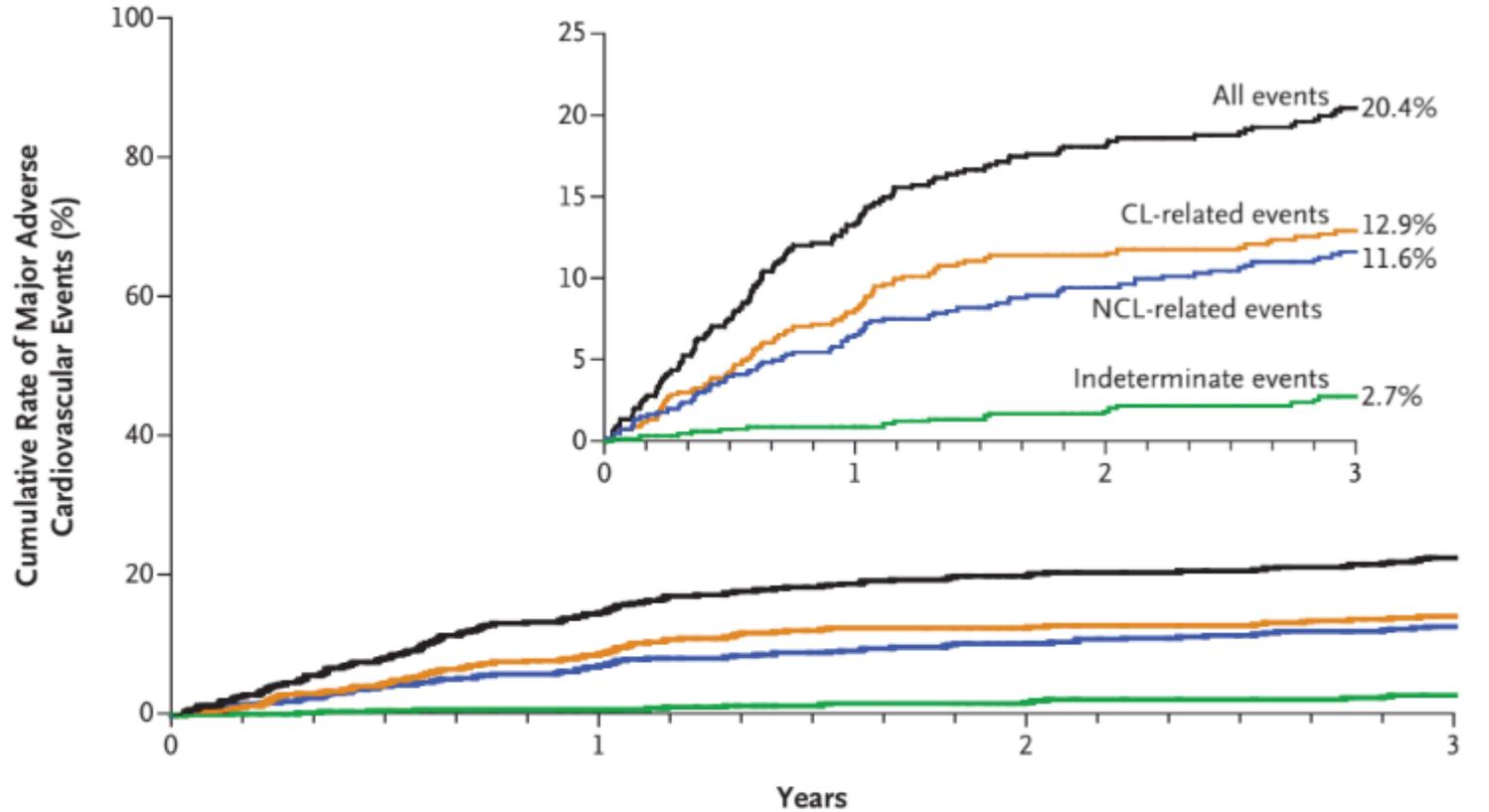
Stone et al. PROSPECT study. N Engl J Med 2011;364:226-35.

SCA

697
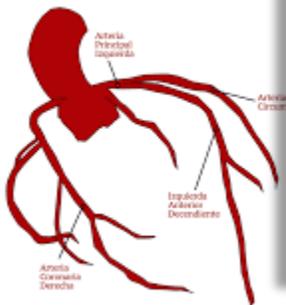
Event	Events Related to Nonculprit Lesions	Indeterminate Events
	percent (number of patients)	
Composite cardiac events†	11.6 (74)	2.7 (17)
Death from cardiac causes, cardiac arrest, or myocardial infarction	1.0 (6)	1.9 (12)
Death from cardiac causes	0	1.8 (11)
Cardiac arrest	0	0.2 (1)
Myocardial infarction	1.0 (6)§	0.3 (2)
Rehospitalization for unstable or progressive angina	10.8 (69)	0.8 (5)
Other events		
Revascularization	10.5 (67)	0
Stent thrombosis¶	0	1.3 (8)

Present Absent

20

16.4

18.2

1.7

1.9

FA+PB ≥70%

TCFA+PB ≥70%+ MLA ≤4 mm²

3 (5.55–21.10)   11.05 (4.39–27.82)
<0.001   <0.001
10.1   4.2
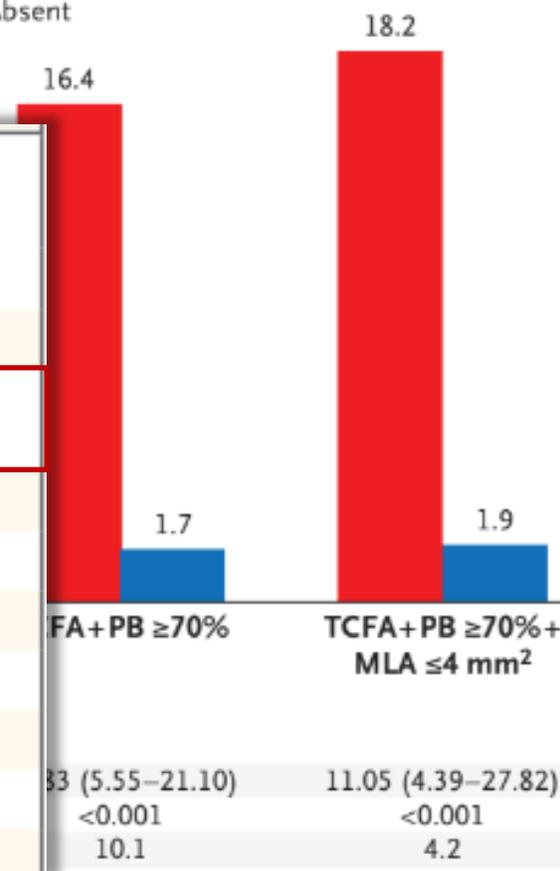
620 les ALM <4.0 mm2
283 les Carga de placa >
70%
596 les TCFA

Stone et al. PROSPECT study. N Engl J Med 2011;364:226-35.
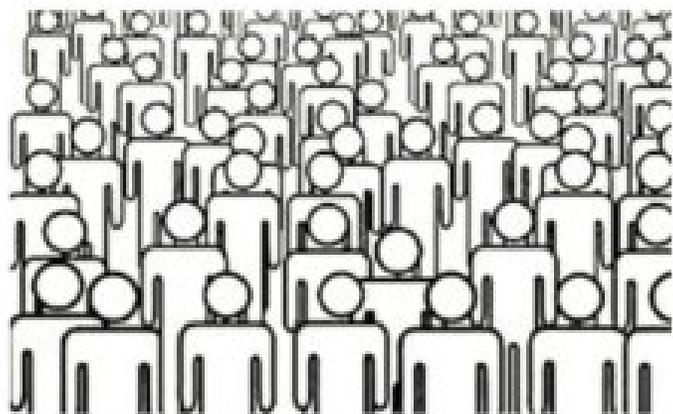
OCT a DA (n=1003 pts)

Relationship between coronary plaque morphology of the left anterior descending artery and 12 months clinical outcome: the CLIMA study

Francesco Prati[1,2]*, Enrico Romagnoli [2,3], Laura Gatto[1,2], Alessio La Manna[4], Francesco Burzotta[3], Yukio Ozaki[5], Valeria Marco[2], Alberto Boi[6], Massimo Fineschi[7], Franco Fabbiocchi[8], Nevio Taglieri[9], Giampaolo Niccoli[3], Carlo Trani[3], Francesco Versaci[10], Giuseppe Calligaris[8], Gianni Ruscica[4], Alessandro Di Giorgio[11], Rocco Vergallo[3], Mario Albertucci[1,2], Giuseppe Biondi-Zoccai [12,13], Corrado Tamburino [4], Filippo Crea[3], Fernando Alfonso[14], and Eloisa Arbustini[15]; on behalf of CLIMA Investigators

| ALM < 3.5mm2 | Macrófagos | FCT < 75 um | Arco lipídico circ >180° |

LAS 4 CARACTERISTICAS X OCT

Prati F et al. CLIMA study. European Heart Journal (2020) 41, 383−391

Resultados clínicos al año

	All population (1003)	Plaques with MLA <3.5 mm², FCT <75 µm, lipid arc >180° and macrophages (36)	Plaques without MLA <3.5 mm², FCT <75 µm, lipid arc >180° and macrophages (967)	P-value
Cardiac death or target LAD segment MI (%)	37 (3.7)	7 (19.4)	30 (3.1)	<0.001 HR 7.54 (3.1–18.6)
Death (%)	34 (3.4)	4 (11.1)	30 (3.1)	0.015 HR 3.90 (1.3–11.7)
Non-cardiac death (%)	9 (0.			
Cardiac death (%)	25 (2.			

MLA <3.5mm² + FCT <75µm + Lipid arc circumferential extension >180° + OCT defined macrophages

En el 18.9% de los ptes que tuvieron el endpopint combinado tuvieron las 4 características HR 7.54, IC 95% 3.1-18.6

...mbination of the 4 95% 3.1-18.6).

Valor predictivo

OCT variable	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Minimum lumen area <3.5 mm²	56.8	61.2	5.3	97.4
Minimum fibrous cap thickness <75 µm	51.4	81.5	10.4	97.6
Maximum lipid arc extension >180°	64.9	56.5	5.9	97.5
Presence of macrophages	81.1	38.3	5.2	98.0
Cholesterol crystal	32.4	77.6	5.7	96.5
Calcified nodules	27.0	82.4	5.6	96.7
Intra-plaque intimal vasculature	56.8	48.6	4.1	96.7
Layered tissue	16.2	82.5	3.4	96.3
Simultaneous presence of minimum lumen area <3.5 mm², fibrous cap thickness <75 µm, lipid arc >180°, and macrophages	18.9	97.5	19.4	96.9

Prati et al. CLIMA study- European Heart Journal (2020) 41, 383–391

Características basales de los pacientes

	Population (1003)			HR (95% CI)	P-value
Cardiac risk factors					
Age (years)[b]	66 (56–74)	69 (58–81)	66 (56–74)	1.05 (1.0–1.1)	0.028
Female gender (%)	247 (24.6)	12 (32.4)	235 (24.3)	1.48 (0.7–2.9)	0.263
Left ventricle ejection fraction (%)[b]	55 (48–60)	55 (40–60)	55 (48–60)	0.96 (0.8–0.9)	0.034
Hypertension (%)	678 (67.6)	31 (83.8)	647 (67.0)	2.46 (1.1–5.9)	0.044
Smoking habit (%)	279 (27.8)	11 (29.7)	268 (27.7)	1.07 (0.5–2.2)	0.846
Family history of CAD (%)	246 (24.5)	7 (18.9)	239 (24.7)	0.71 (0.3–1.6)	0.421
Prior MI (%)	207 (20.6)	10 (27.0)	197 (20.4)	1.37 (0.7–2.8)	0.391
Prior PCI (%)	300 (29.9)	12 (32.4)	288 (29.8)	1.12 (0.6–2.2)	0.741
Diabetes mellitus (%)	223 (22.2)	10 (27.0)	213 (22.0)	1.31 (0.6–2.7)	0.462
CKD (GFR <60 mL/min/1.73 m^2) (%)	149 (14.9)	11 (29.7)	138 (14.3)	2.47 (1.2–5.0)	0.012
Hypercholesterolaemia (%)	606 (60.4)	19 (51.4)	587 (60.8)	0.68 (0.4–1.3)	0.241
Total cholesterol (mg/dL)[b]	177 (151–207)	161 (147–193)	177 (152–208)	0.99 (0.9–1.0)	0.275
LDL (mg/dL)[b]	110 (85–137)	107 (80–126)	110 (85–137)	1.00 (0.9–1.0)	0.509
HDL (mg/dL)[b]	42 (35–51)	39 (31–51)	42 (35–51)	0.98 (0.9–1.0)	0.229
Triglycerides (mg/dL)[b]	119 (88–169)	116 (80–155)	120 (89–169)	1.00 (0.9–1.0)	0.174
High-sensitivity C-reactive protein (mg/L)[b]	4.65 (1.5–12.1)	7.10 (1.7–12.9)	4.60 (1.5–12.0)	0.99 (0.9–1.1)	0.565
Diagnosis					
Acute coronary syndrome (%)	536 (53.4)	17 (45.9)	519 (53.7)	0.72 (0.4–1.4)	0.305
STEMI (%)	199 (19.8)	8 (21.6)	191 (19.7)	1.08 (0.5–2.4)	0.853
NSTEMI (%)	199 (19.8)	7 (18.9)	192 (20.0)	0.95 (0.4–2.2)	0.907
Unstable angina (%)	138 (13.8)	2 (5.4)	136 (14.0)	0.36 (0.2–1.4)	0.143
Stable angina (%)	467 (46.6)	20 (54.1)	447 (46.3)	1.40 (0.7–2.7)	0.305
CAD-vessel disease					
No significant disease	66 (6.5)	2 (5.4)	64 (6.6)	0.83 (0.2–3.5)	0.800
1-vessel significant disease	481 (48.0)	10 (27.0)	471 (48.8)	0.40 (0.2–0.8)	0.012
2-vessel significant disease	297 (29.6)	15 (40.6)	282 (29.2)	1.62 (0.8–3.1)	0.150
3-vessel significant disease	159 (15.9)	10 (27.0)	149 (15.4)	2.01 (1.0–4.2)	0.059
Left main significant disease	61 (6.1)	1 (2.7)	60 (6.2)	0.41 (0.1–3.0)	0.380

Prati et al. CLIMA study- European Heart Journal (2020) 41, 383−391

Características basales de los pacientes

	Population (1003)	Patients with clinical event[a] (37)	Patients without clinical event[a] (966)	HR (95% CI)	P-value
Cardiac risk factors					
Age (years)[b]	66 (56–74)	69 (58–81)	66 (56–74)	1.05 (1.0–1.1)	0.028
Female gender (%)	247 (24.6)	12 (32.4)	235 (24.3)	1.48 (0.7–2.9)	0.263
Left ventricle ejection fraction (%)[b]	55 (48–60)	55 (40–60)	55 (48–60)	0.96 (0.8–0.9)	0.034
Hypertension (%)	678 (67.6)	31 (83.8)	647 (67.0)	2.46 (1.1–5.9)	0.044
Smoking habit (%)	279 (27.8)	11 (29.7)	268 (27.7)	1.07 (0.5–2.2)	0.846
Family history of CAD (%)	246 (24.5)	7 (18.9)	239 (24.7)	0.71 (0.3–1.6)	0.421
Prior MI (%)	207 (20.6)	10 (27.0)	197 (20.4)	1.37 (0.7–2.8)	0.391
Prior PCI (%)	300 (29.9)	12 (32.4)	288 (29.8)	1.12 (0.6–2.2)	0.741
Diabetes mellitus (%)	223 (22.2)	10 (27.0)	213 (22.0)	1.31 (0.6–2.7)	0.462
CKD (GFR <60 mL/min/1.73 m^2) (%)	149 (14.9)	11 (29.7)	138 (14.3)	2.47 (1.2–5.0)	0.012
Hypercholesterolaemia (%)	606 (60.4)	19 (51.4)	587 (60.8)	0.68 (0.4–1.3)	0.241
Total cholesterol (mg/dL)[b]	177 (151–207)	161 (147–193)	177 (152–208)	0.99 (0.9–1.0)	0.275
LDL (mg/dL)[b]	110 (85–137)	107 (80–126)	110 (85–137)	1.00 (0.9–1.0)	0.509
HDL (mg/dL)[b]	42 (35–51)	39 (31–51)	42 (35–51)	0.98 (0.9–1.0)	0.229
Triglycerides (mg/dL)[b]	119 (88–169)	116 (80–155)	120 (89–169)	1.00 (0.9–1.0)	0.174
High-sensitivity C-reactive protein (mg/L)[b]	4.65 (1.5–12.1)	7.10 (1.7–12.9)	4.60 (1.5–12.0)	0.99 (0.9–1.1)	0.565
Diagnosis					
Acute coronary syndrome (%)	536 (53.4)	17 (45.9)	519 (53.7)	0.72 (0.4–1.4)	0.305
STEMI (%)	199 (19.8)	8 (21.6)	191 (19.7)	1.08 (0.5–2.4)	0.853
NSTEMI (%)	199 (19.8)	7 (18.9)	192 (20.0)	0.95 (0.4–2.2)	0.907
Unstable angina (%)	138 (13.8)	2 (5.4)	136 (14.0)	0.36 (0.2–1.4)	0.143
Stable angina (%)	467 (46.6)	20 (54.1)	447 (46.3)	1.40 (0.7–2.7)	0.305
CAD-vessel disease					
No significant disease	66 (6.5)	2 (5.4)	64 (6.6)	0.83 (0.2–3.5)	0.800
1-vessel significant disease	481 (48.0)	10 (27.0)	471 (48.8)	0.40 (0.2–0.8)	0.012
2-vessel significant disease	297 (29.6)	15 (40.6)	282 (29.2)	1.62 (0.8–3.1)	0.150
3-vessel significant disease	159 (15.9)	10 (27.0)	149 (15.4)	2.01 (1.0–4.2)	0.059
Left main significant disease	61 (6.1)	1 (2.7)	60 (6.2)	0.41 (0.1–3.0)	0.380

Prati et al. CLIMA study- European Heart Journal (2020) 41, 383–391

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†

Todas las lesiones no culpables RVD (EV) >50% → FFR >0·80
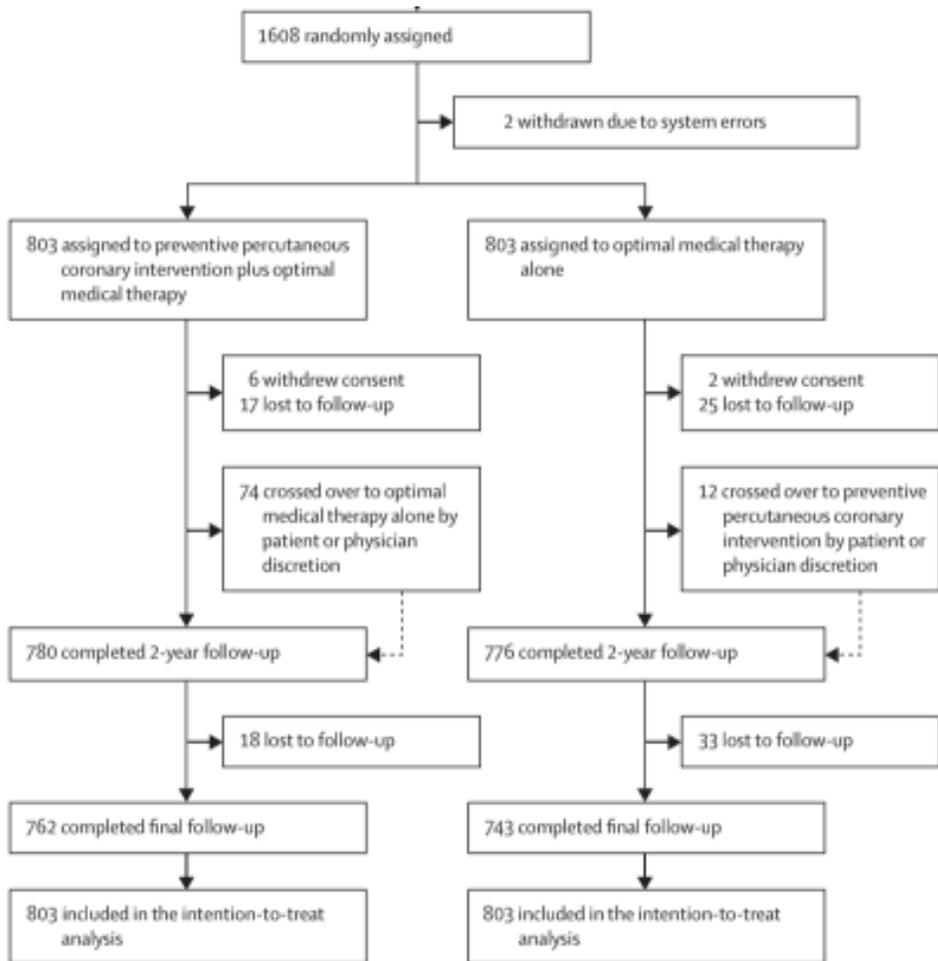
ALM < 4.0 mm2 (IVUS u OCT)
Carga de placa > 70% (IVUS)
Core Lipídico
TCFA < 65 Um (IVUS u OCT)

Al menos 2

ATC preventiva + TMO   TMO

MACE (2 años)
Muerte CV/TV-IAM/iTVR/Internación x AI

Park SJ et al. Lancet. 2024 May 4;403(10438):1753-1765.

Left table:

	Preventive percutaneous coronary intervention plus optimal medical therapy (n=803 [831 lesions])	Optimal medical therapy alone (n=803 [841 lesions])
Age, years	64 (58–71)	65 (59–71)
Clinical presentation		
Stable angina or silent ischaemia	670 (83%)	677 (84%)
Unstable angina	106 (13%)	91 (11%)
Non-ST-elevation myocardial infarction	18 (2%)	28 (3%)
ST-elevation myocardial infarction	9 (1%)	7 (1%)
Left ventricular ejection fraction†		
Number of target lesions (vulnerable plaques) per patient		
Qualifying criteria for target lesions††		
Minimal luminal area <4·0 mm² by grey-scale IVUS or OCT		
Plaque burden >70% by grey-scale IVUS	792/815 (97%)	805/831 (97%)
Large lipid-rich plaque by NIRS (maxLCBI₄ₘₘ >315)	99/348 (28%)	94/369 (26%)
Thin-cap fibroatheroma defined by OCT or radiofrequency IVUS	39/571 (7%)	40/679 (6%)
Target lesion location		
Left anterior descending artery	416/831 (50%)	400/841 (48%)

Right table:

	Preventive percutaneous coronary intervention plus optimal medical therapy (n=803)	Optimal medical therapy alone (n=803)	Difference in event rates, percentage points (95% CI)	Hazard ratio (95% CI)*
Primary composite outcome†	..	..	..	0·54 (0·33 to 0·87)
At 2 years (primary timepoint)	3 (0·4%)	27 (3·4%)	−3·0 (−4·4 to −1·8)	0·11 (0·03 to 0·36), p=0·0003
At 4 years	17 (2·8%)	37 (5·4%)	−2·6 (−4·7 to 0·4)	
At 7 years	26 (6·5%)	47 (9·4%)	−2·9 (−7·3 to 1·5)	
Death from cardiac causes	..	..	..	0·87 (0·31 to 2·39)
At 2 years	1 (0·1%)	6 (0·8%)	−0·6 (−1·3 to 0·02)	..
At 4 years	5 (0·8%)	7 (0·9%)	−0·1 (−1·1 to 0·9)	..
At 7 years	7 (1·4%)	8 (1·3%)	0·1 (−1·4 to 1·5)	..
				0·79 (0·40 to 1·55)
			−0·5 (−1·7 to 0·6)	..
			−0·1 (−1·5 to 1·4)	..
			−1·2 (−3·4 to 1·0)	..
				0·62 (0·20 to 1·90)
			−0·6 (−1·3 to 0·02)	..
			−0·3 (−1·3 to 0·6)	..
At 7 years	5 (1·0%)	8 (1·4%)	−0·3 (−1·7 to 1·1)	
Ischaemia-driven target-vessel revascularisation	..			0·44 (0·25 to 0·77)
At 2 years	1 (0·1%)	19 (2·4%)	−2·3 (−3·4 to −1·2)	
At 4 years	10 (1·7%)	29 (4·4%)	−2·7 (−4·6 to −0·8)	
At 7 years	17 (4·9%)	38 (8·0%)	−3·2 (−7·4 to 1·1)	
Hospitalisation for unstable or progressive angina	..	..		0·19 (0·06 to 0·54)
At 2 years	1 (0·1%)	12 (1·5%)	−1·4 (−2·3 to −0·5)	
At 4 years	4 (0·7%)	16 (2·4%)	−1·7 (−3·0 to −0·4)	
At 7 years	4 (0·7%)	21 (4·9%)	−4·2 (−7·2 to −1·4)	

En pacientes con placas vulnerables no significativas, la ATC preventiva redujo el MACE, comparado con el TMO

Park SJ et al. Lancet. 2024 May 4;403(10438):1753-1765.

	Preventive percutaneous coronary intervention plus optimal medical therapy (n=803 [831 lesions])	Optimal medical therapy alone (n=803 [841 lesions])
Age, years	64 (58–71)	65 (59–71)
Clinical presentation		
Stable angina or silent ischaemia	670 (83%)	677 (84%)
Unstable angina	106 (13%)	91 (11%)
Non-ST-elevation myocardial infarction	18 (2%)	28 (3%)
ST-elevation myocardial infarction	9 (1%)	7 (1%)
Left ventricular ejection fraction†	63 (60–66)	63 (60–66)
Number of target lesions (vulnerable plaques) per patient	1 (1–1)	1 (1–1)
Qualifying criteria for target lesions††		
Minimal luminal area <4.0 mm² by grey-scale IVUS or OCT	809/831 (97%)	817/841 (97%)
Plaque burden >70% by grey-scale IVUS	792/815 (97%)	805/831 (97%)
Large lipid-rich plaque by NIRS (maxLCBI$_{4mm}$ >315)	99/348 (28%)	94/369 (26%)
Thin-cap fibroatheroma defined by OCT or radiofrequency IVUS	39/571 (7%)	40/679 (6%)
Target lesion location		
Left anterior descending artery	416/831 (50%)	400/841 (48%)

	Preventive percutaneous coronary intervention plus optimal medical therapy (n=803)	Optimal medical therapy alone (n=803)	Difference in event rates, percentage points (95% CI)	Hazard ratio (95% CI)*
Primary composite outcome†	..	..	..	0.54 (0.33 to 0.87)
At 2 years (primary timepoint)	3 (0.4%)	27 (3.4%)	–3.0 (–4.4 to –1.8)	0.11 (0.03 to 0.36), p=0.0003
At 4 years	17 (2.8%)	37 (5.4%)	–2.6 (–4.7 to 0.4)	..
At 7 years	26 (6.5%)	47 (9.4%)	–2.9 (–7.3 to 1.5)	..
Death from cardiac causes	..	..	..	0.87 (0.31 to 2.39)
At 2 years	1 (0.1%)	6 (0.8%)	–0.6 (–1.3 to 0.02)	..
At 4 years	5 (0.8%)	7 (0.9%)	–0.1 (–1.1 to 0.9)	..
At 7 years	7 (1.4%)	8 (1.3%)	0.1 (–1.4 to 1.5)	..
All myocardial infarctions	..	..	..	0.79 (0.40 to 1.55)
At 2 years	9 (1.1%)	13 (1.7%)	–0.5 (–1.7 to 0.6)	..
At 4 years	14 (2.0%)	15 (2.0%)	–0.1 (–1.5 to 1.4)	..
At 7 years	15 (2.4%)	19 (3.5%)	–1.2 (–3.4 to 1.0)	..
Target-vessel-related myocardial infarction	..	..	..	0.62 (0.20 to 1.90)
At 2 years	1 (0.1%)	6 (0.8%)	–0.6 (–1.3 to 0.02)	..
At 4 years	4 (0.6%)	7 (10%)	–0.3 (–1.3 to 0.6)	..
At 7 years	5 (1.0%)	8 (1.4%)	–0.3 (–1.7 to 1.1)	..
Ischaemia-driven target-vessel revascularisation	..	..	..	0.44 (0.25 to 0.77)
At 2 years	1 (0.1%)	19 (2.4%)	–2.3 (–3.4 to –1.2)	..
At 4 years	10 (1.7%)	29 (4.4%)	–2.7 (–4.6 to –0.8)	..
At 7 years	17 (4.9%)	38 (8.0%)	–3.2 (–7.4 to 1.1)	..
Hospitalisation for unstable or progressive angina	..	..	..	0.19 (0.06 to 0.54)
At 2 years	1 (0.1%)	12 (1.5%)	–1.4 (–2.3 to –0.5)	..
At 4 years	4 (0.7%)	16 (2.4%)	–1.7 (–3.0 to –0.4)	..
At 7 years	4 (0.7%)	21 (4.9%)	–4.2 (–7.2 to –1.4)	..

Park SJ et al. Lancet. 2024 May 4;403(10438):1753-1765.

Atherosclerosis in Patients With Acute Myocardial Infarction
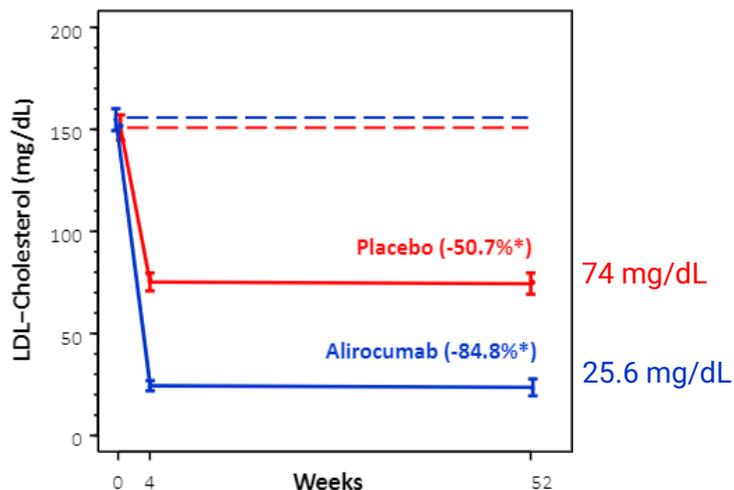The PACMAN-AMI Randomized Clinical Trial
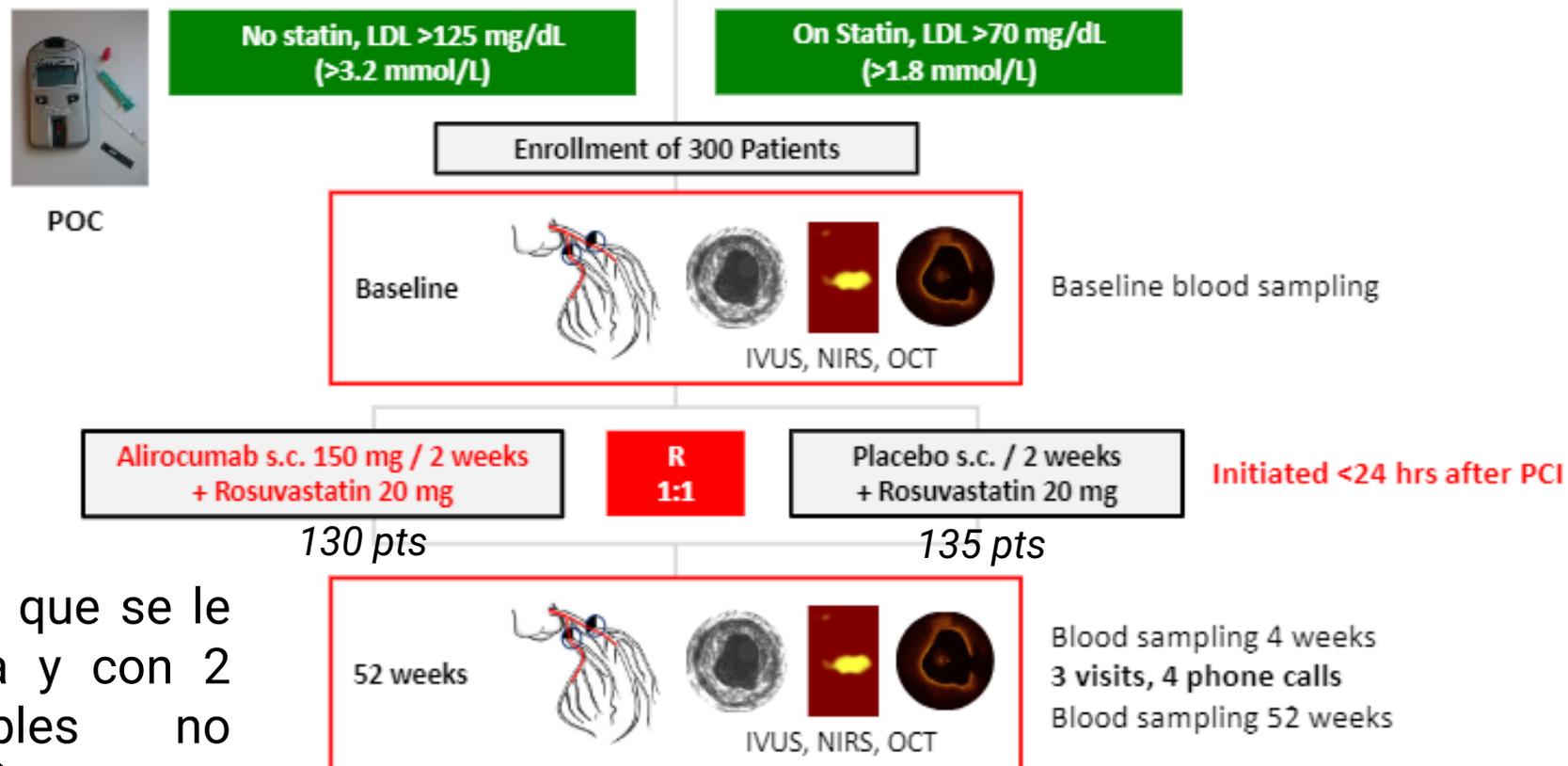
Pacientes con IAM a los que se le realizó una ATC exitosa y con 2 lesiones no culpables no significativas (20-50% DR)

Raber L et al. PACMAN Trial. JAMA. 2022;327(18):1771-1781

En pacientes con antecedentes de IAM, la adición de Alirocumab + estatinas de alta intensidad (vs placebo) resultó en una mayor regresión de placa al año.
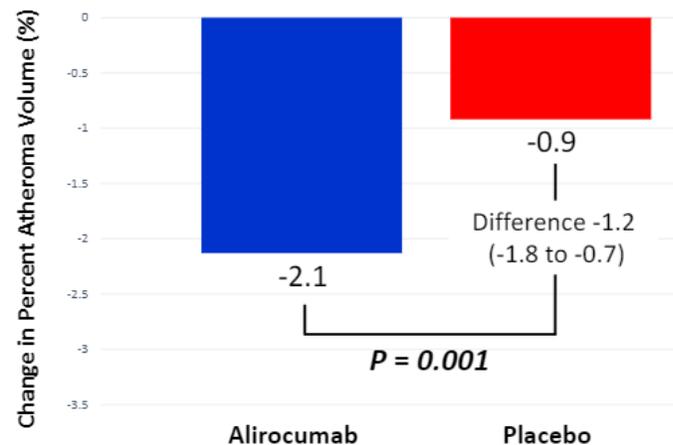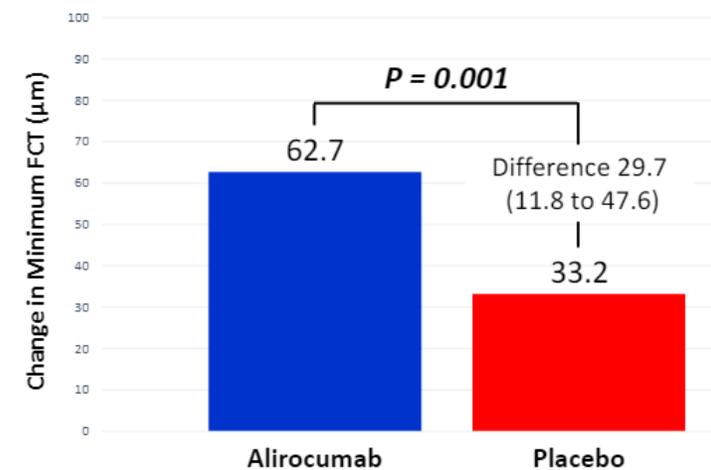
Cambios en LDLc

Cambios en el grosor de la capa fibrosa (OCT)

Cambios en el porcentaje de volumen de placa (IVUS)

Raber L et al. PACMAN Trial. JAMA. 2022;327(18):1771-1781

Mensajes para llevar a casa

- No hay una respuesta clara – <u>todavía</u>.

- La <u>enfermedad aterosclerótica es dinámica</u>, un continuo. Una placa vulnerable puede estabilizarse y viceversa.

- <u>Podemos identificar placas vulnerables</u> por métodos <u>invasivos</u> (IVUS, OCT, NIRS) y <u>no invasivos</u> (MSCT)

- Las <u>lesiones no culpables de alto riesgo</u> (área luminal mínima <4 mm2, Core lípidico y tamaño de placa) tienen que tratarse con el <u>mejor tratamiento medico</u> hasta que nuevos ECR evaluando terapias localizadas puedan inequivocamente <u>demostrar seguridad y eficacia.</u>

Muchas gracias!

A Matías Rodriguez-Granillo, MD
Centro de Estudios en Cardiología Intervencionista (CECI)
Departamento de Cardiología Intervencionista, Sanatorio Otamendi, Buenos Aires, Argentina
@matiasrg