INFARTO AGUDO DE MIOCARDIO
IAM -- SCACEST

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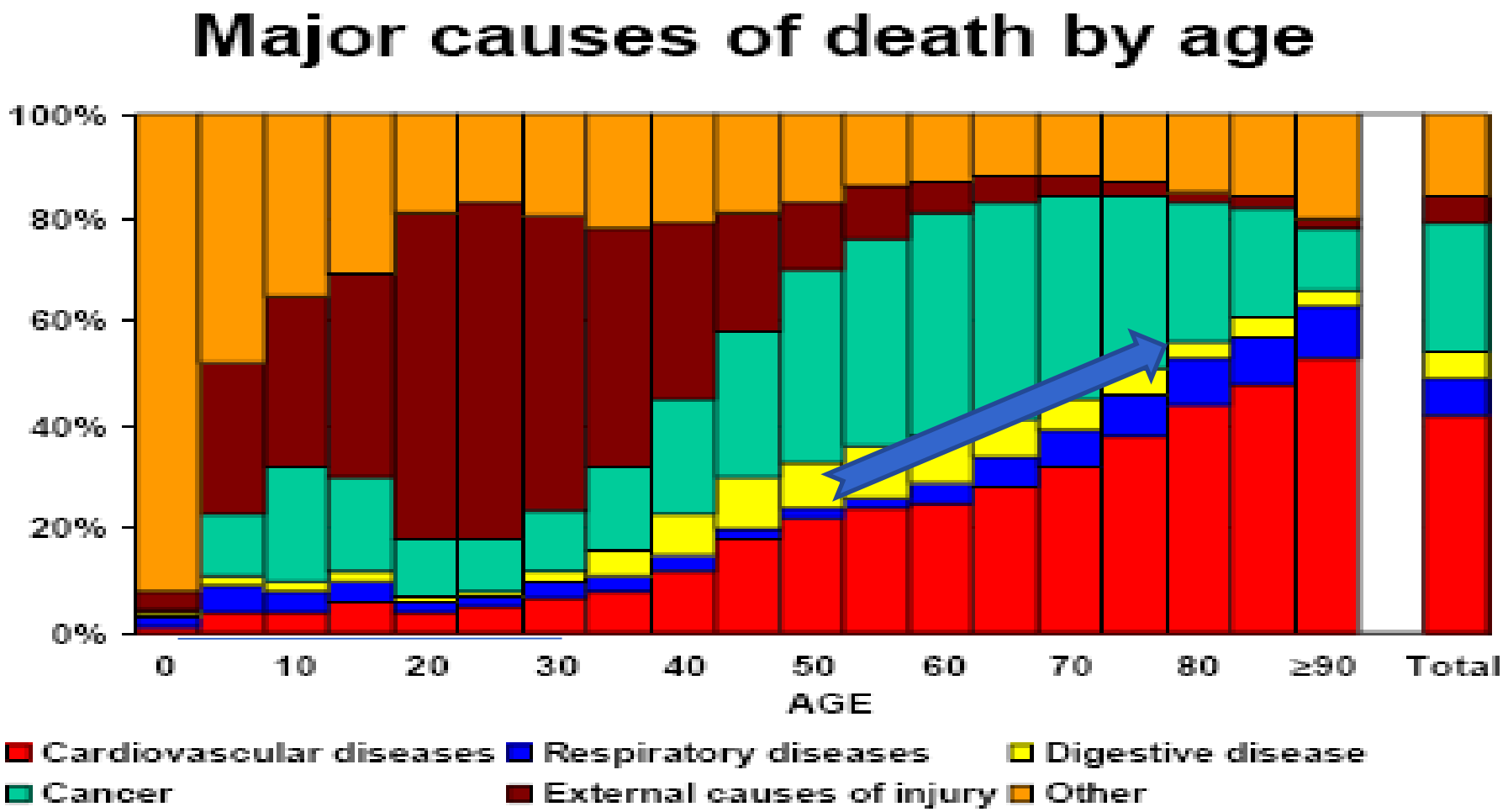
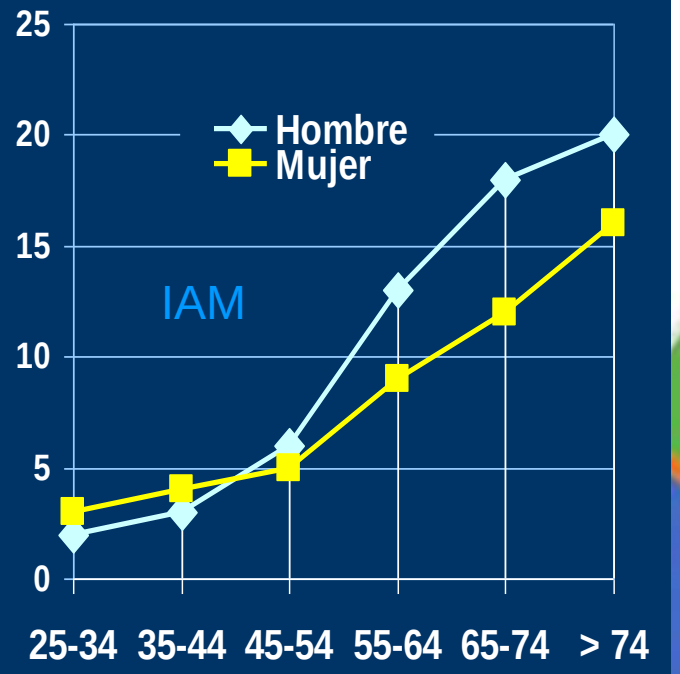
Causas de Muerte /EDAD




25
20
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Hombre
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35-44
45-54
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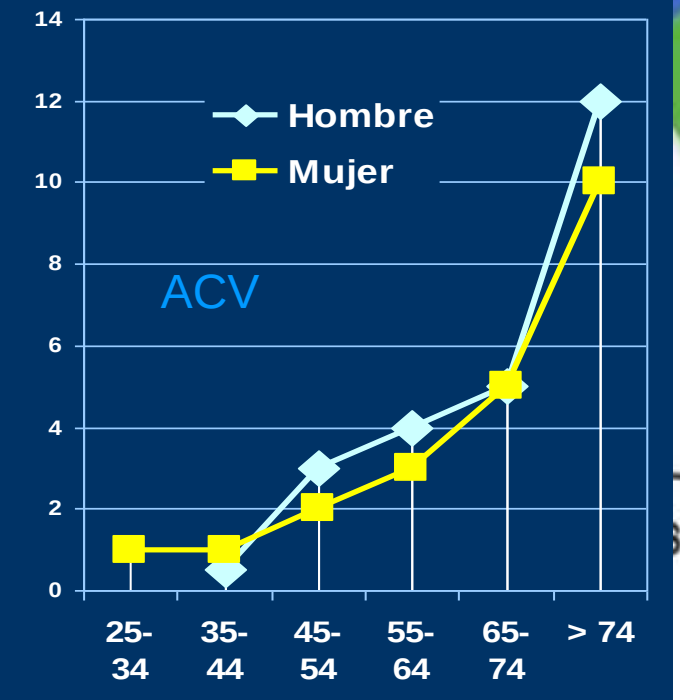
14
12
10
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Hombre
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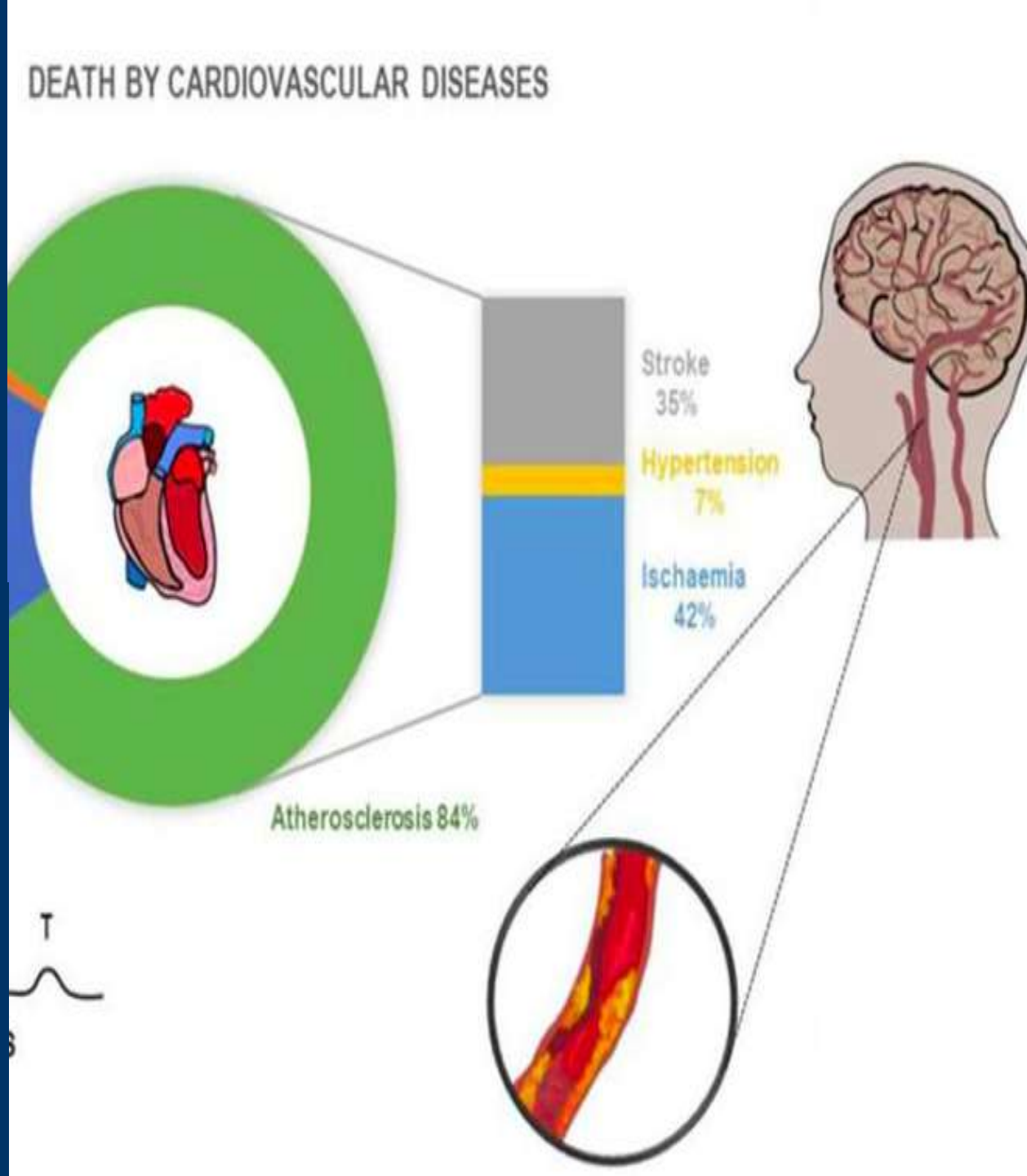
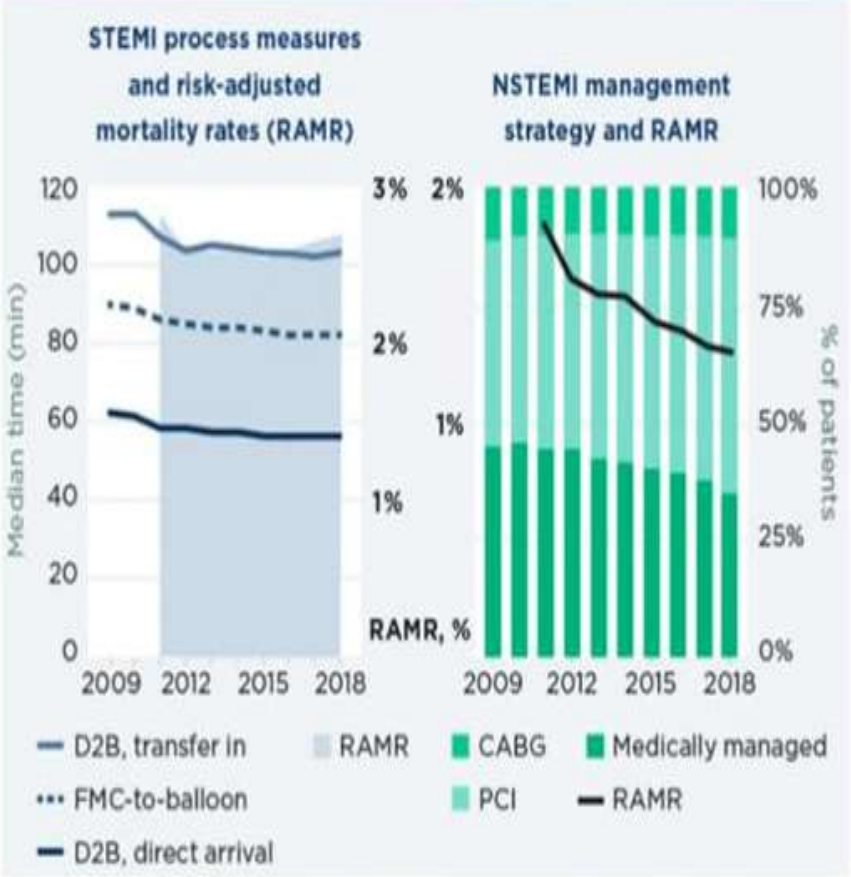
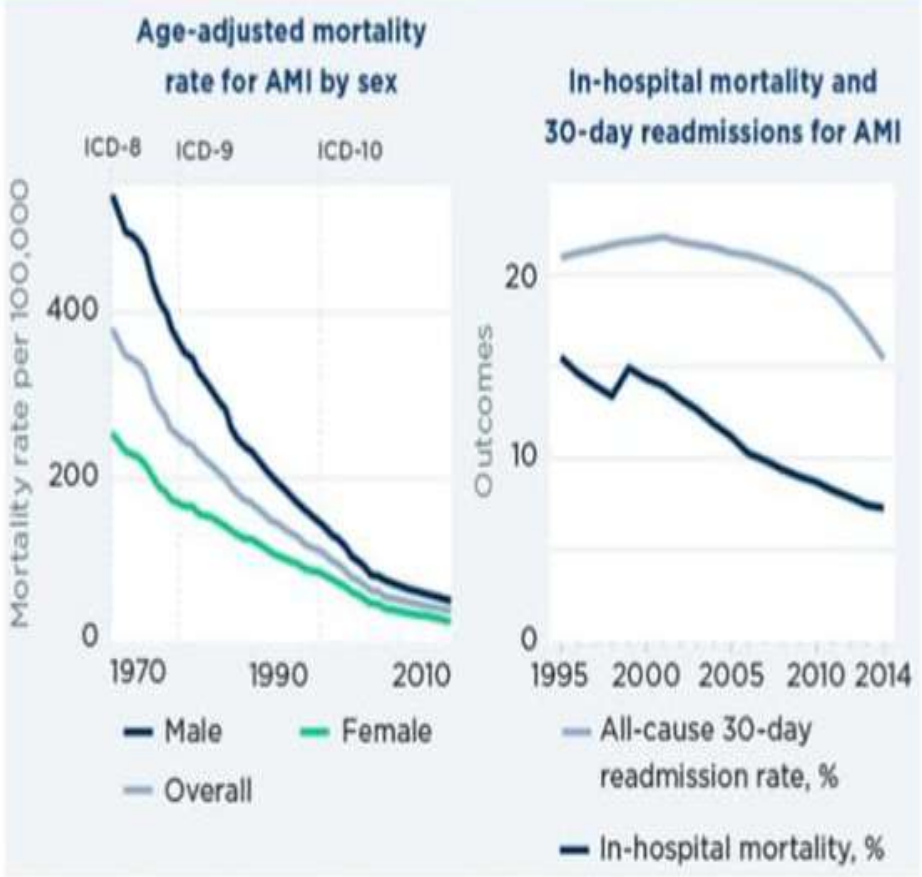

DEATH BY CARDIOVASCULAR DISEASES
Stroke 35%
Hypertension 7%
Ischaemia 42%
Atherosclerosis 84%

AMI Quality of Care and Mortality in the US
2026 JACC Statistics



Between 2010 and 2017, the proportion of patients receiving defect-free AMI care increased across all sociodemographic groups. But nearly 1 in 4 patients still did not receive optimal comprehensive care by 2017



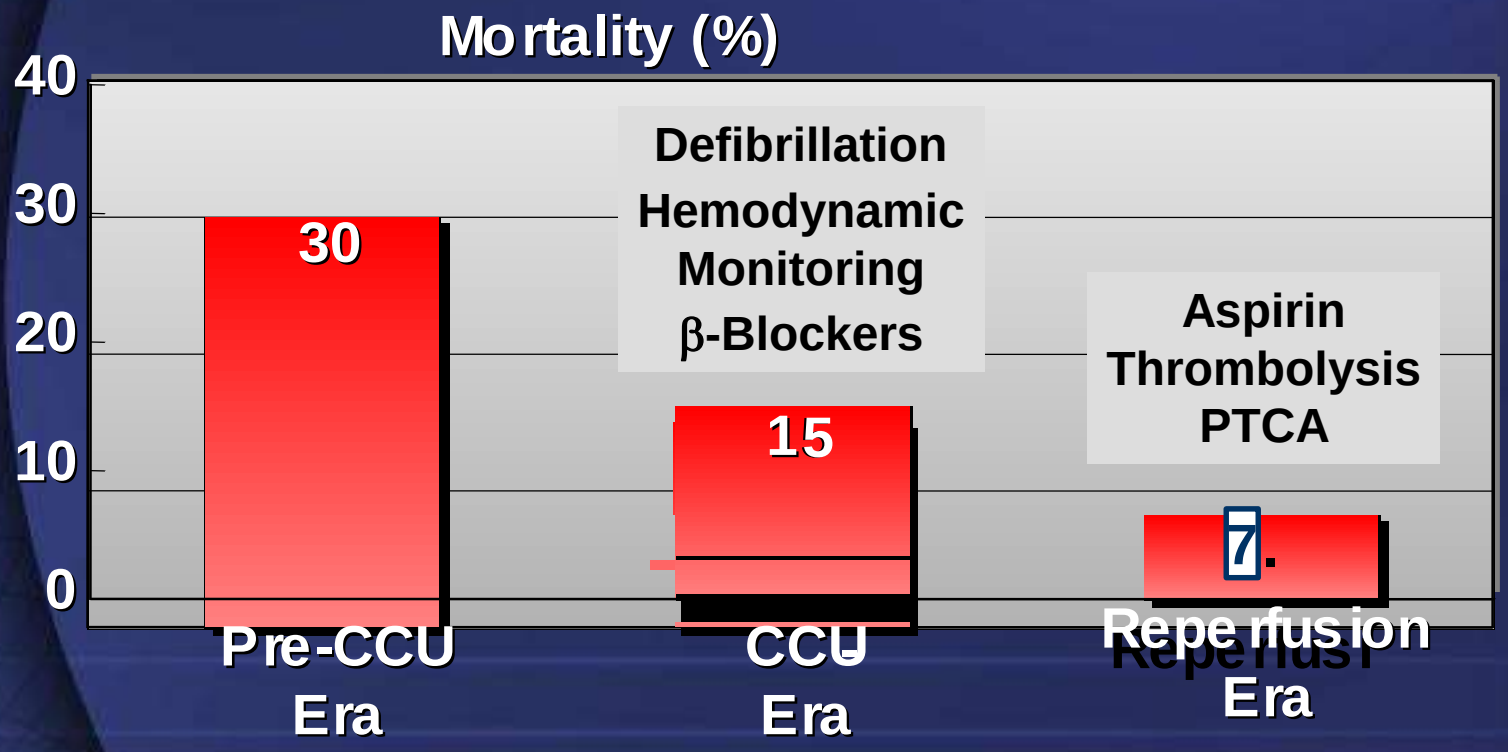
Age-adjusted mortality rates declined sharply between 1968 and 2019. In-hospital mortality and 30-day readmissions among Medicare beneficiaries declined markedly from 1995 to 2014

CABG, coronary artery bypass grafting; D2B, door-to-balloon; FMC, first medical contact; JACC, Journal of the American College of Cardiology; NSTEMI, non-ST-elevation myocardial infarction; PCI, percutaneous coronary intervention; STEMI, ST-elevation myocardial infarction.

Wadhera RK, et al. J Am Coll Cardiol. 2026;87(11):1094-1134.

INFARTO DE MIOCARDIO

Impacto de la UCO en la mortalidad



Adaptado de Antman, Braunwald In:Braunwald ed. Heart Disease p 1184.

Hospitalizaciones en Argentina SCACEST

- 2021 Argentina .Aprox. 40 - 45.000 hospitalizaciones por año por IAM

- 10.8 casos por cada 10.000 habitantes

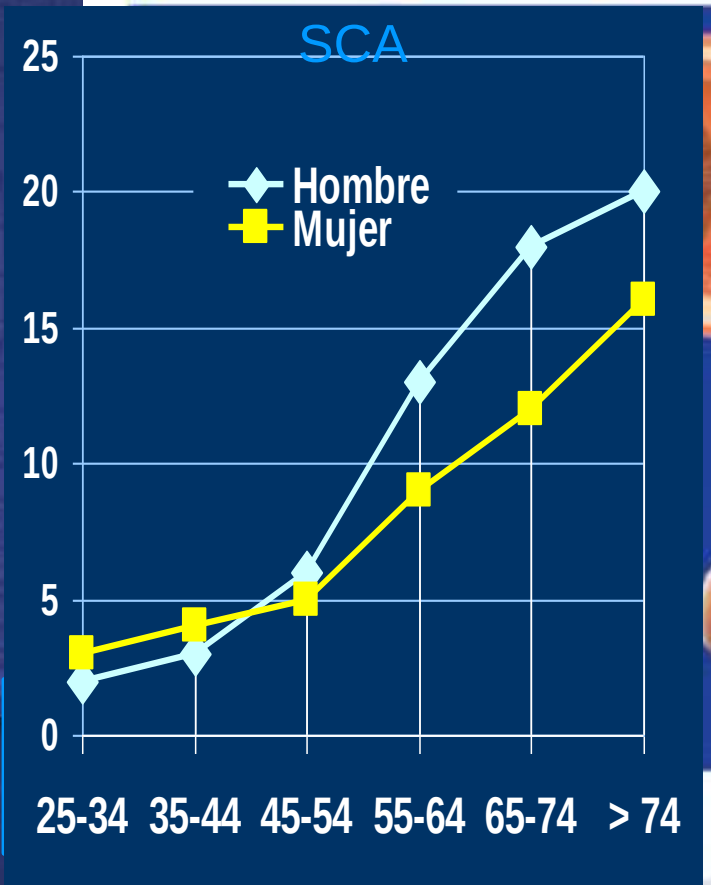
Acute Coronary Syndromes AMI

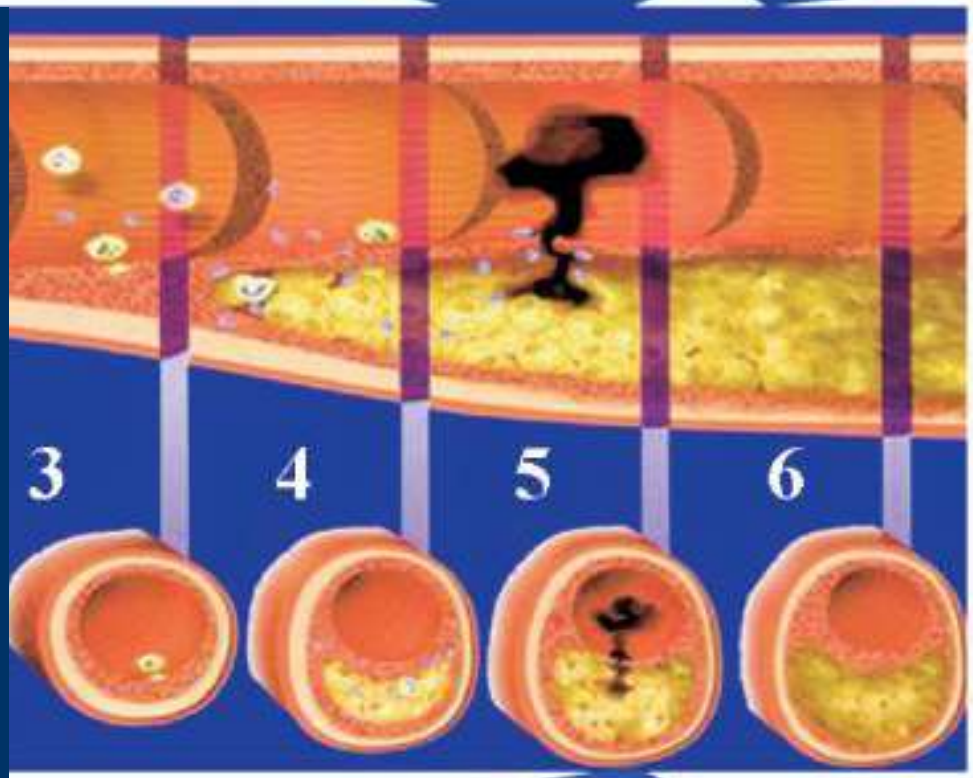
Onset of NSTE-ACS
-Initial recognition and management in the ED by first responders or ED personnel
-Risk stratification
-Immediate management

Hospital Management
-Medication
-Conservative versus invasive strategy
-Special groups
-Preparation for discharge

Secondary Prevention/ Long-Term Management

Management Prior to NSTE-ACS





Acute Coronary Syndromes

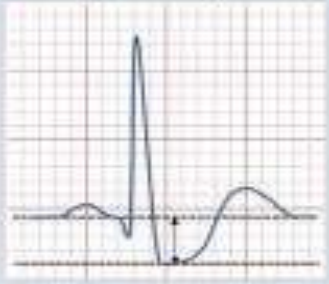
NSTEMI

Angiographic Findings

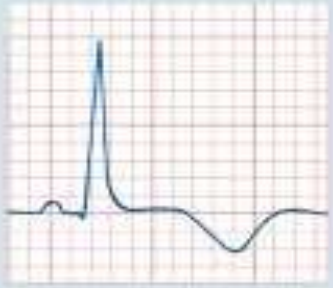
Partially occlusive thrombus

Electrocardiographic Changes

ST-segment depression



T-wave inversion



Nonspecific or no electrocardiographic changes may instead be seen

Biomarker Changes (cardiac troponin)

Unstable angina	NSTEMI
−	+

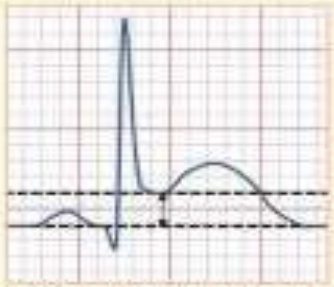
STEMI

Angiographic Findings

Completely occlusive thrombus

Electrocardiographic Changes

ST-segment elevation



ST-elevation in ≥2 contiguous leads on standard 12-lead ECG (or ST-elevation on posterior lead ECG)

Biomarker Changes (cardiac troponin)

\+

(Might be − if short time from symptom onset)

Stress Circunferencial
HTA

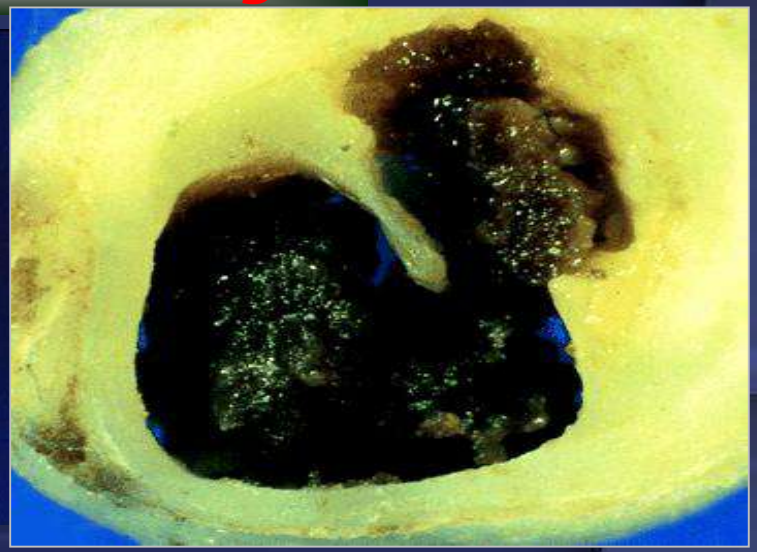
TBQ
HTA
DBT
↑LDL
Genetica

Inmunocompl
Infecciones?
Inflamación

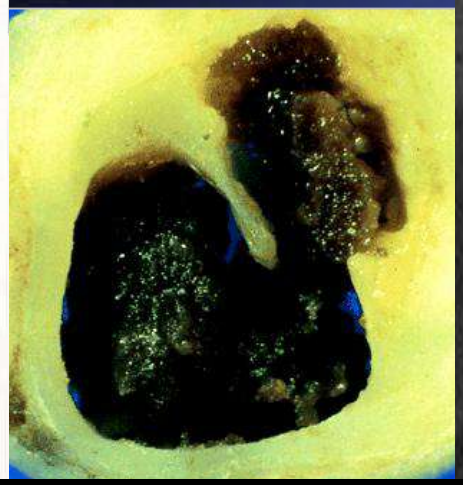
Injuria endotelial mínima

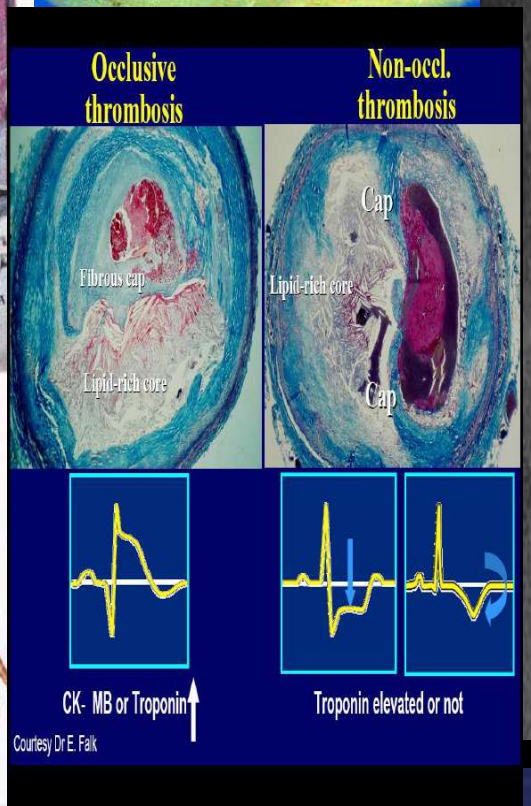
Acumulación de lípidos
Migración de monocitos/macrófagos

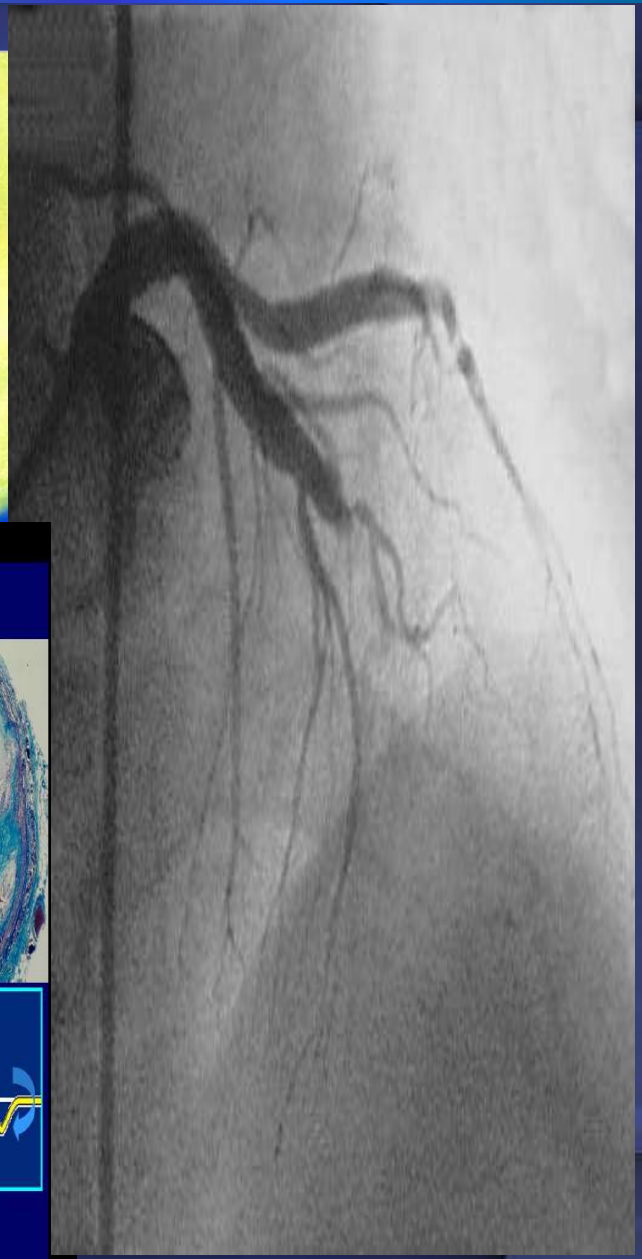
Placa Vulnerable

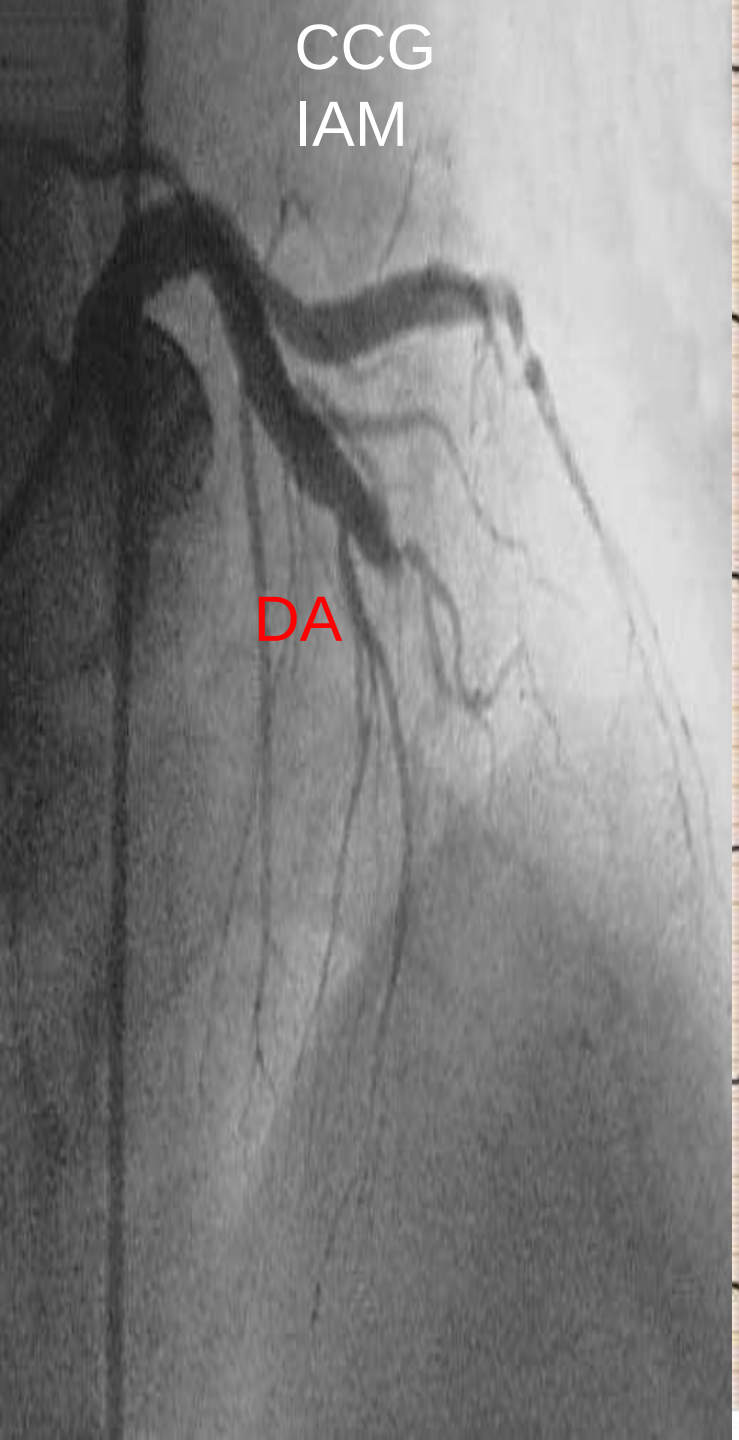


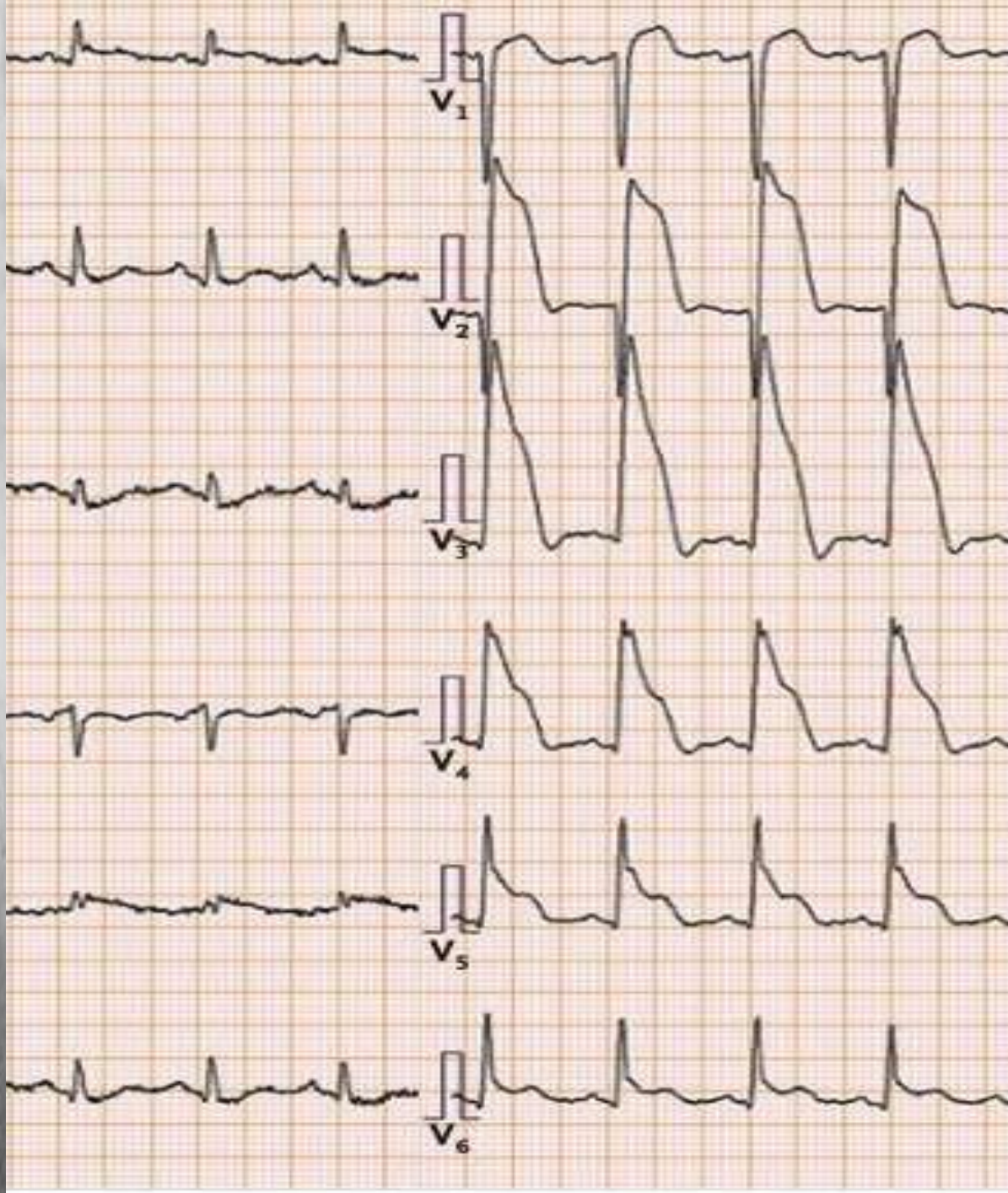
Infarto Agudo de Miocardio



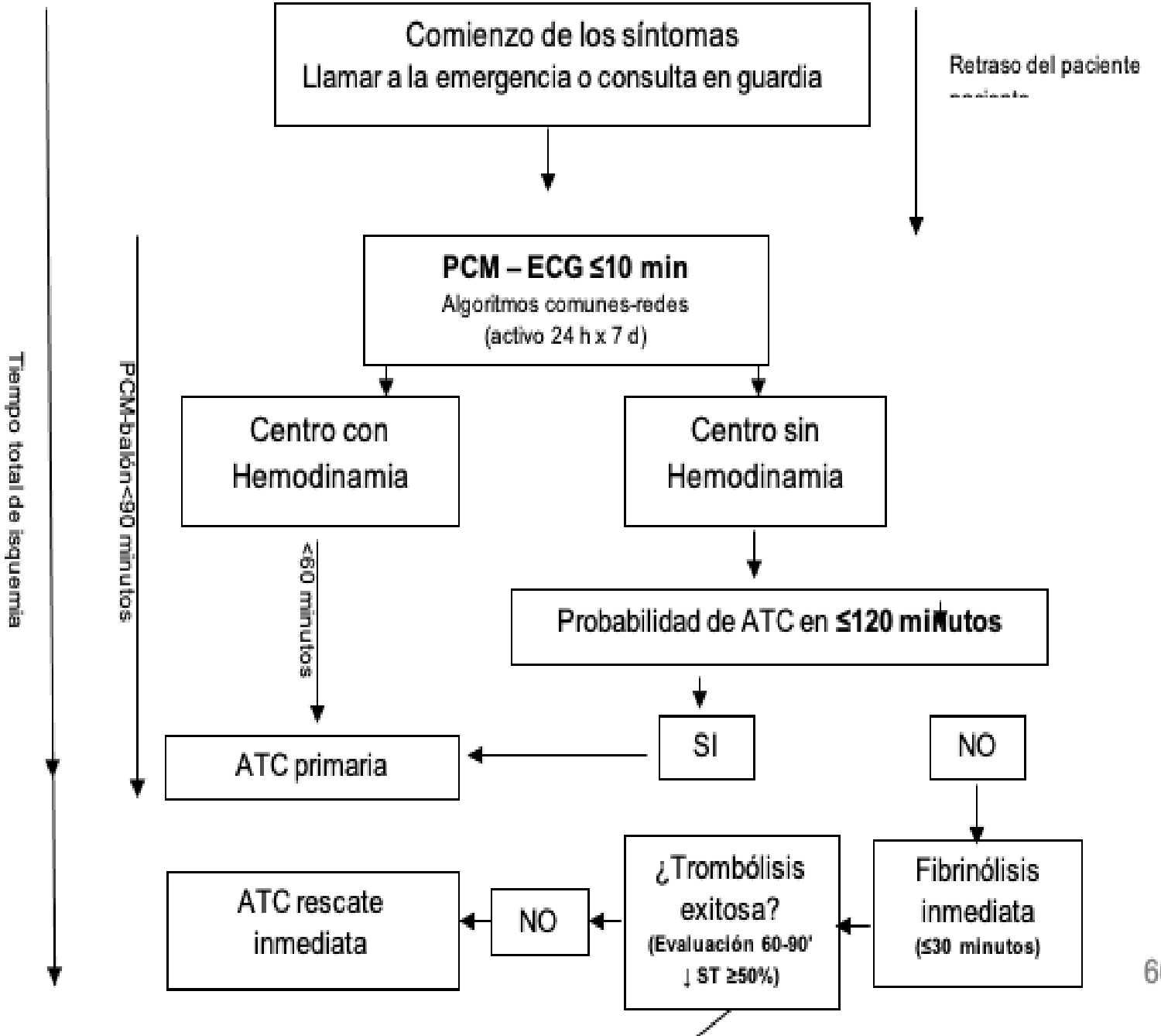





CCG
IAM
DA


V1
V2
V3
V4
V5
V6

ESTRATEGIA DE REPERFUSION



4 - DEFINICION IAM (Tipo1)

Daño miocárdico + evidencia de isquemia

Criterios para diagnosticar IAM

#Síntomas=> isquemia miocárdica-**Angor Disnea**
#-Aumento y descenso de cTn con al menos 1 valor > percentil 99 .
#-Cambios nuevos en el ECG isquémicos: ST, onda T, BRI nuevo ,Ondas Q patológicas nuevas
#-Imagen con Pérdida de miocardio viable o alteración de la motilidad→causa isquémica
#-Angiografía: Trombo coronario identificado

PRO

Poor operationalization limits clinical uptake and utility.

Type 2 MI - Encompasses heterogeneous entities, significant potential for misdiagnosis, and inaccurate MI categorization - management implications.

No cutoff to discriminate ischaemic/other causes of myocardial injury and necrosis.

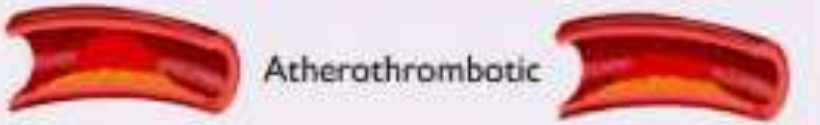
Prognostic significance of Type 4a and 5 MI remains uncertain. Arbitrary threshold values for cTn elevation in Type 4a and 5 MI.

FLAWED

Should be substantially modified

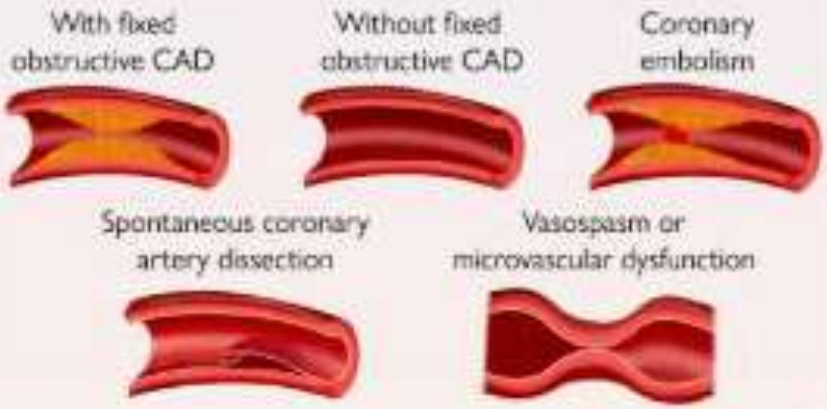
Myocardial infarction types

Type 1 MI



Type 2 MI

Supply - demand mismatch



Type 3 MI

Cardiac death

Type 4 MI

Post-PCI

Type 5 MI

Post-CABG

CON

General acceptance by the clinical community.

Current Type 1 MI (atherothrombotic) aligns with management with PCI or fibrinolysis.

Type 2 MI category helps focus on causes of supply-demand mismatch. Moving non-atherothrombotic aetiologies out of Type 2 MI may add confusion.

Distinguishing Types 1 and 2 MI informs therapies and clinical trials.

Operationalization - ICD codes based on UDMI.

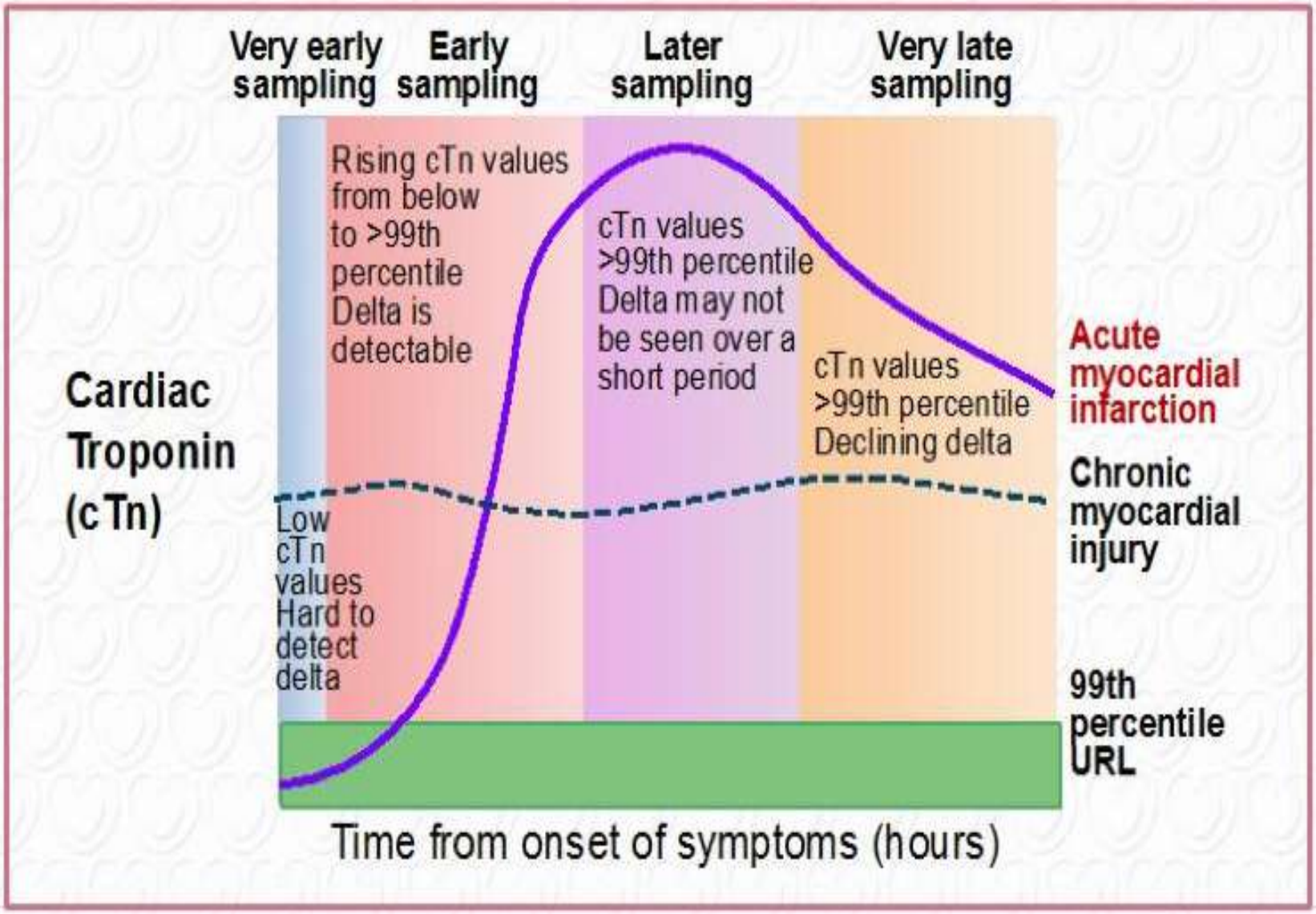
USEFUL

Retain and modify slightly

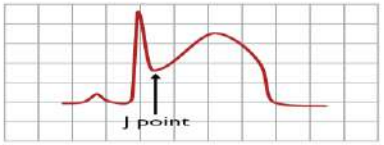
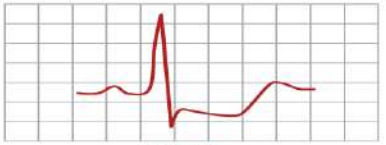
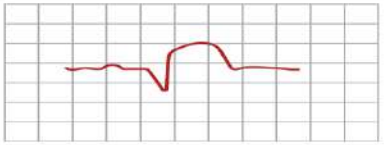
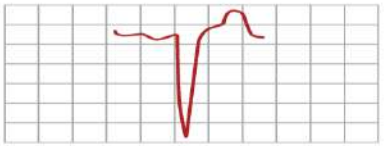
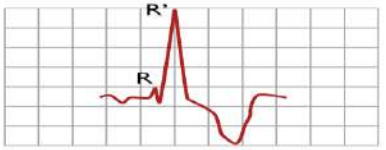
CABG, coronary artery bypass grafting; CAD, coronary artery disease; cTn, cardiac troponin; ICD, International Classification of Diseases; IHD, ischaemic heart disease; MI, myocardial infarction; PCI, percutaneous coronary intervention; UDMI, universal definition of myocardial infarction

Conceptual Illustration of Troponin Kinetics after Acute Myocardial Injury and Infarction

ESC
European Society of Cardiology

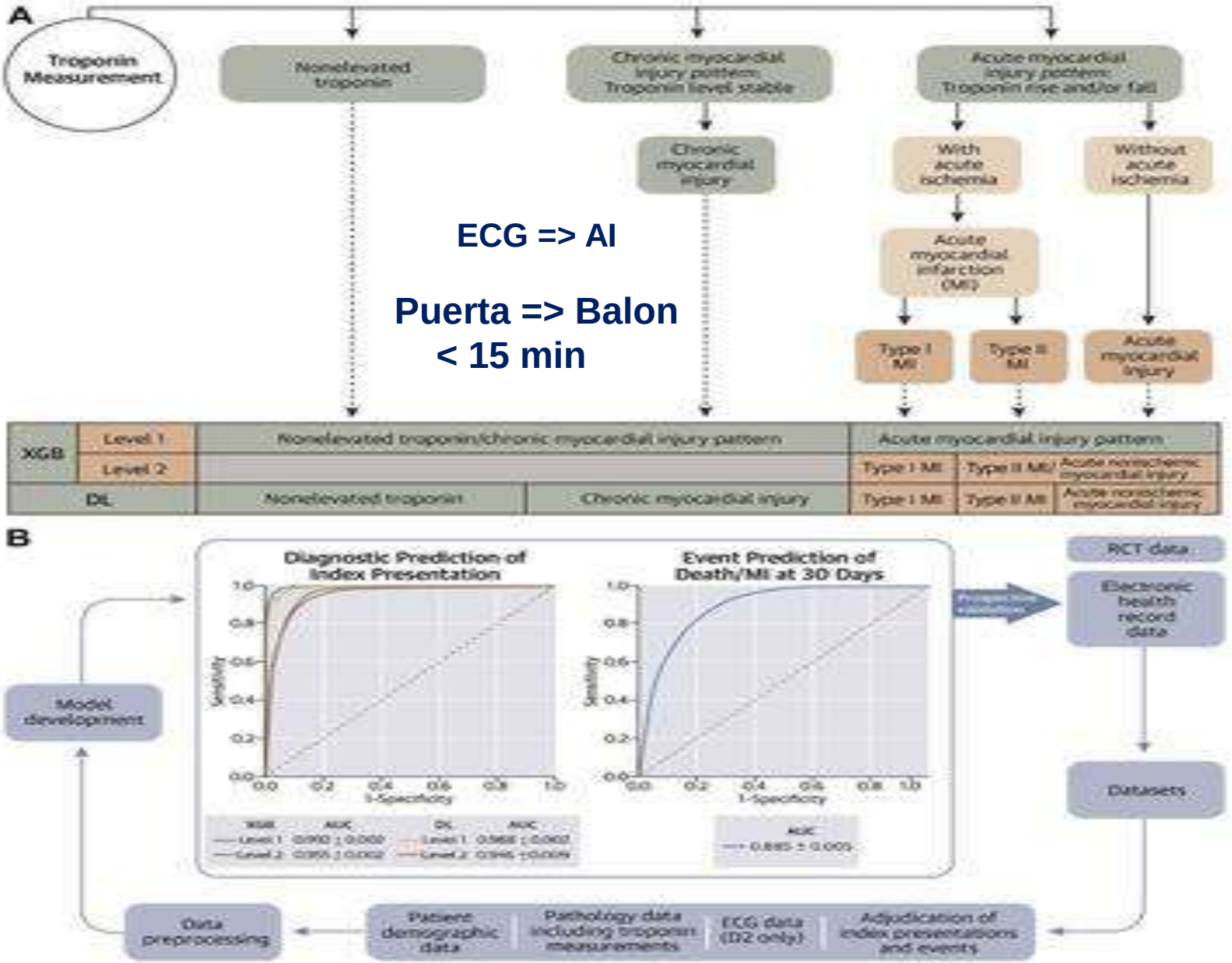


ECG IAM REQUIERE RAPIDA REPERFUSION

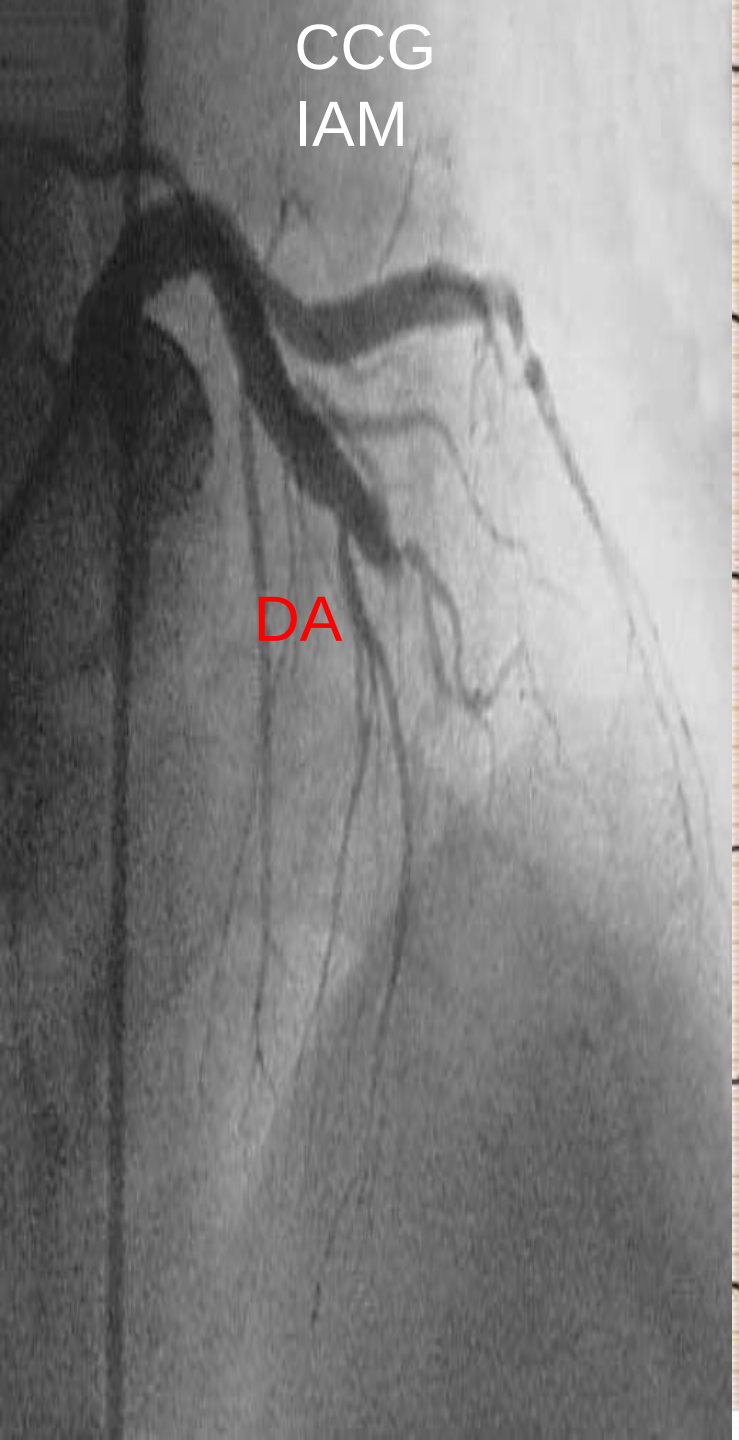
ECG pattern	Criteria	Signifying	Figure
i STEMI	New ST-elevation at the J-point in ≥ 2 contiguous leads[a] ≥2.5 mm in men <40 years, ≥2 mm in men ≥40 years, or ≥1.5 mm in women regardless of age in leads V2–V3 and/or ≥1 mm in the other leads (in the absence of LV hypertrophy or left bundle branch block) [a]Including V3R and V4R	Ongoing acute coronary artery occlusion	
ii Posterior STEMI	ST-segment depression in leads V1–V3, especially when the terminal T-wave is positive (ST-segment elevation equivalent), and concomitant ST-segment elevation ≥0.5 mm recorded in leads V7–V9	Posterior STEMI	
iii LCx occlusion/ right ventricular MI	ST-segment elevation in V7–V9 and V3R and V4R, respectively	Left circumflex (LCX) artery occlusion or right ventricular MI	
iv Multivessel ischaemia/ left main obstruction	ST depression ≥1mm in six or more surface leads (inferolateral ST depression), coupled with ST-segment elevation in aVR and/or V1	Multivessel ischaemia or left main coronary artery obstruction, particularly if the patient presents with haemodynamic compromise	
v Left bundle branch block/ paced rhythm	QRS duration greater than 120 ms Absence of Q wave in leads I, V5 and V6 Monomorphic R wave in I, V5 and V6 ST and T wave displacement opposite to the major deflection of the QRS complex	Patients with a high clinical suspicion of ongoing myocardial ischaemia should be managed in a similar way to STEMI patients	
vi Right bundle branch block	QRS duration greater than 120 ms rsR' "bunny ear" pattern in the anterior precordial leads (leads V1-V3) Slurred S waves in leads I, aVL and frequently V5 and V6	Patients with a high clinical suspicion of ongoing myocardial ischaemia should be managed in a similar way to STEMI patients	

ESC

CENTRAL ILLUSTRATION: Machine Learning Models for Phenotyping and Prognostication of Myocardial Infarction and Injury in Suspected Acute Coronary Syndrome



Khan E, et al. JACC Adv. 2024;10.1016/j.jacadv.2024.101011


CCG
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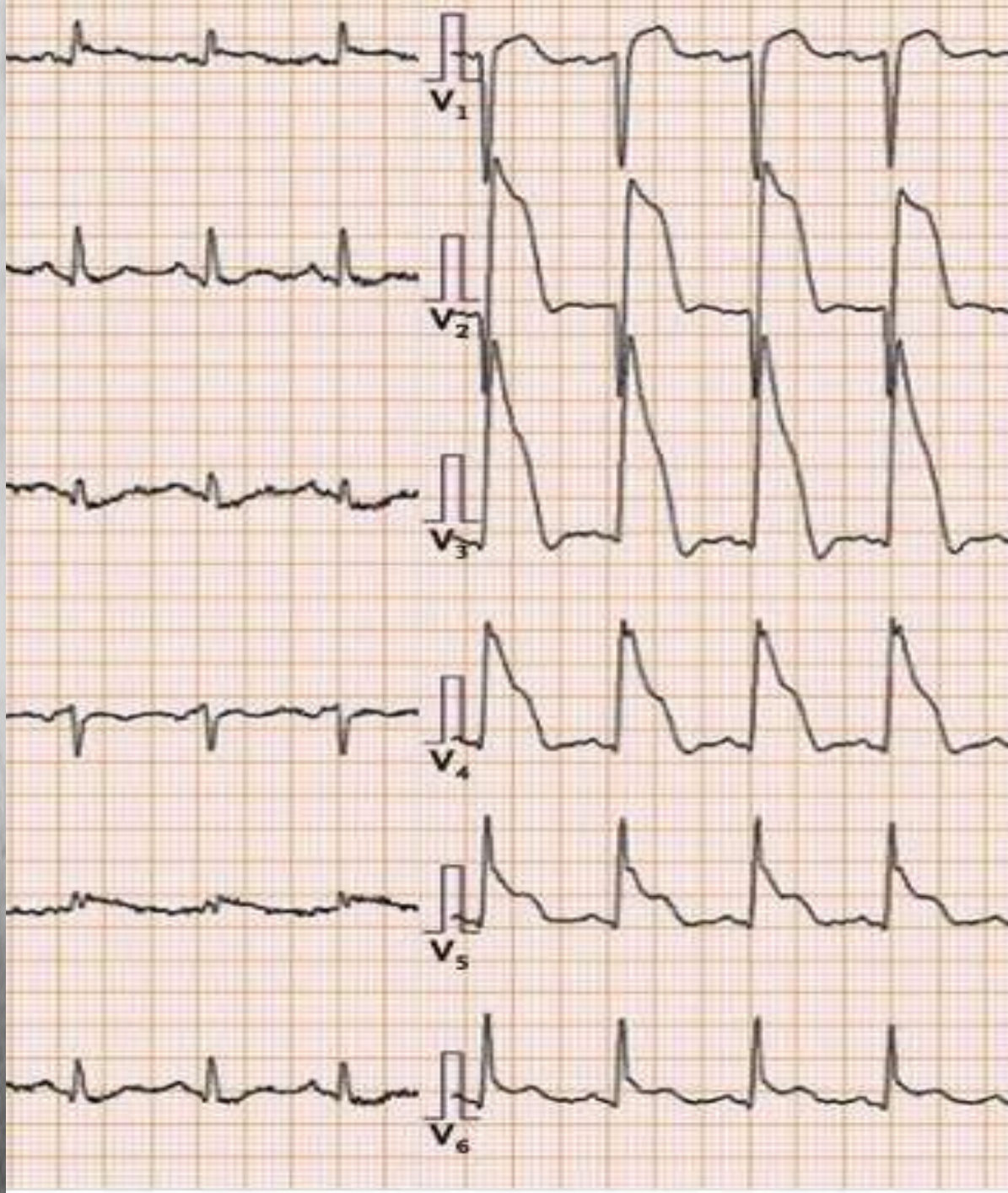
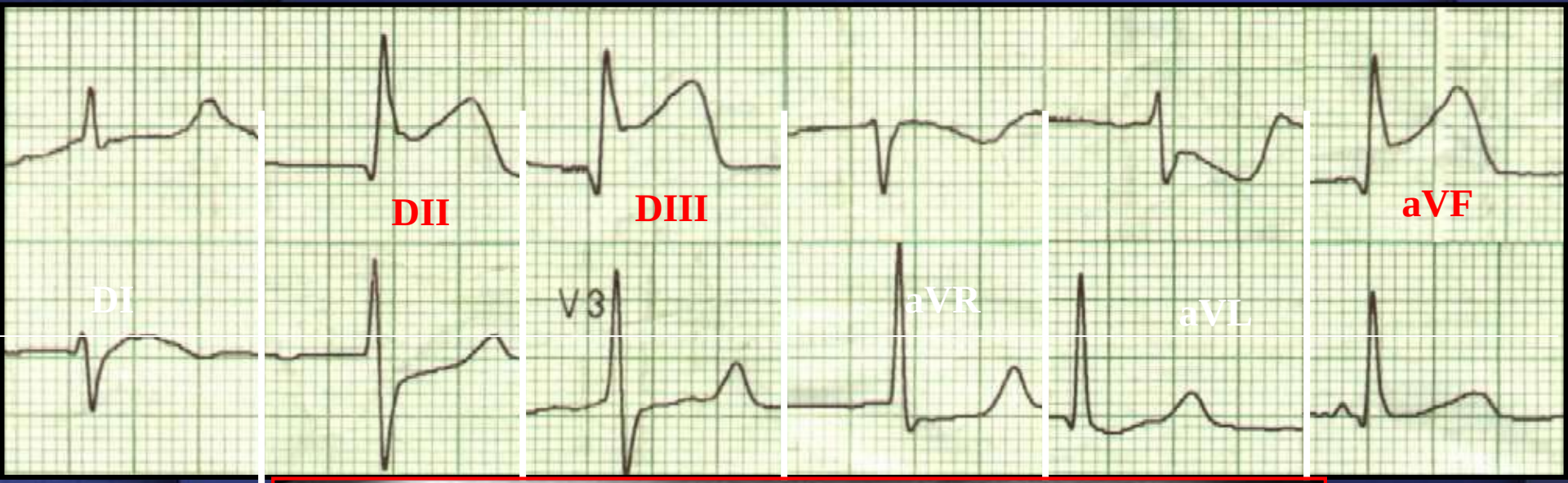
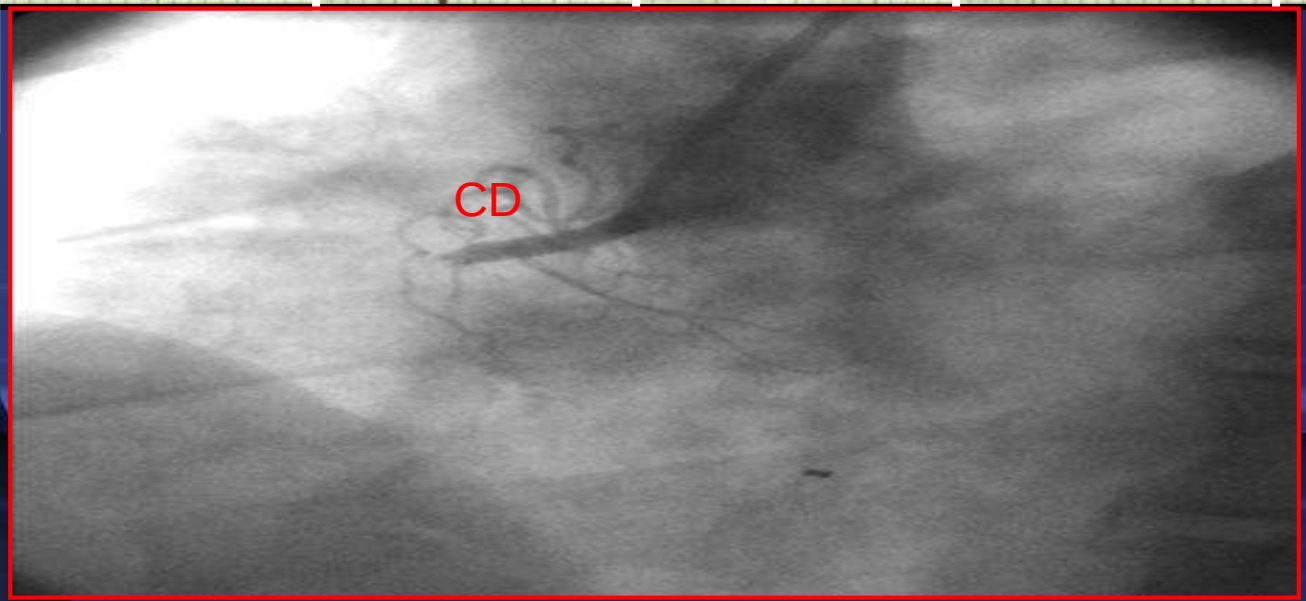

V1
V2
V3
V4
V5
V6

Infarto de Miocardio

IAM Inferior - Inferoposterior

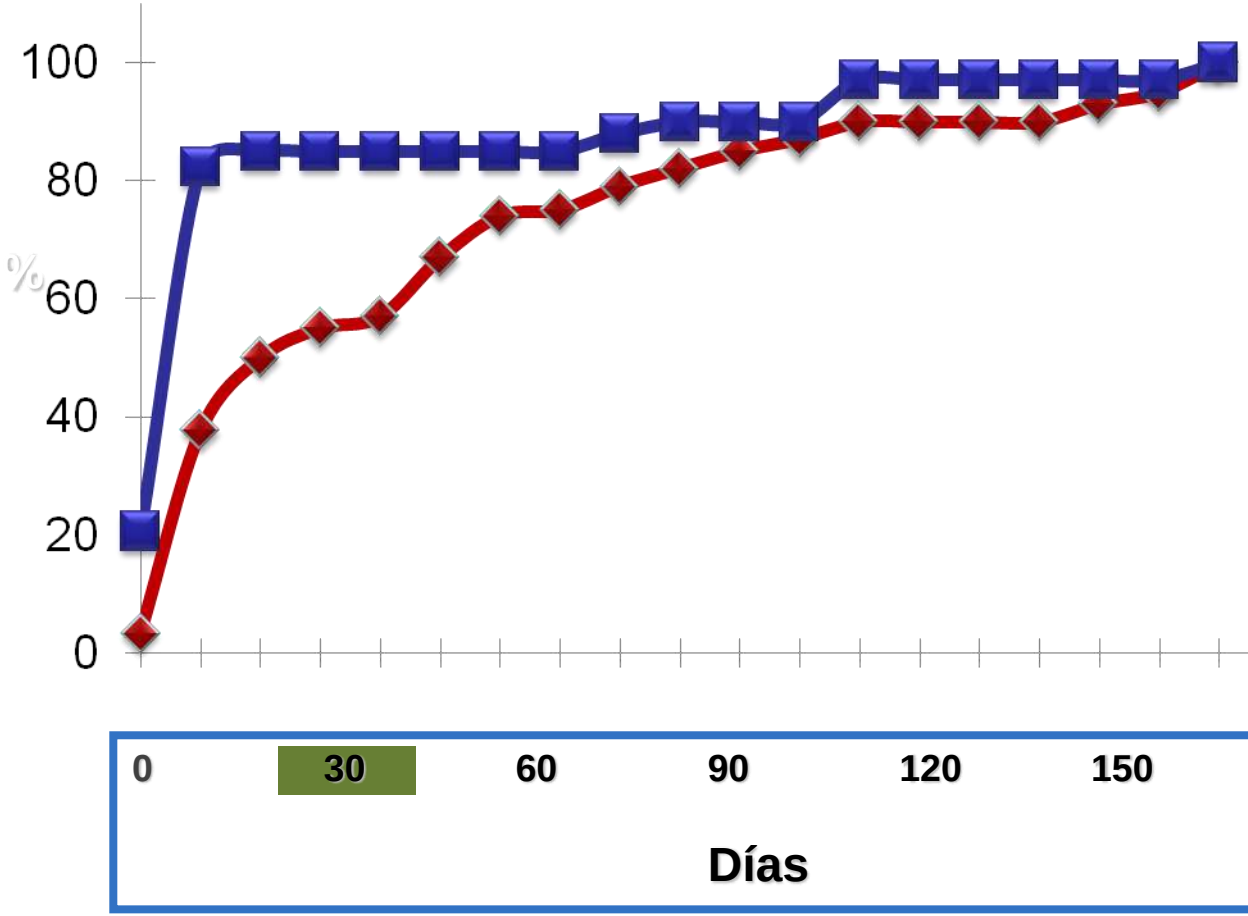




SELECCIÓN DE LOS PACIENTES CANDIDATOS A REPERFUSIÓN

Eventos Clínicos Mayores

PACS: 1483 pacientes
IAM / MORT.9.5%



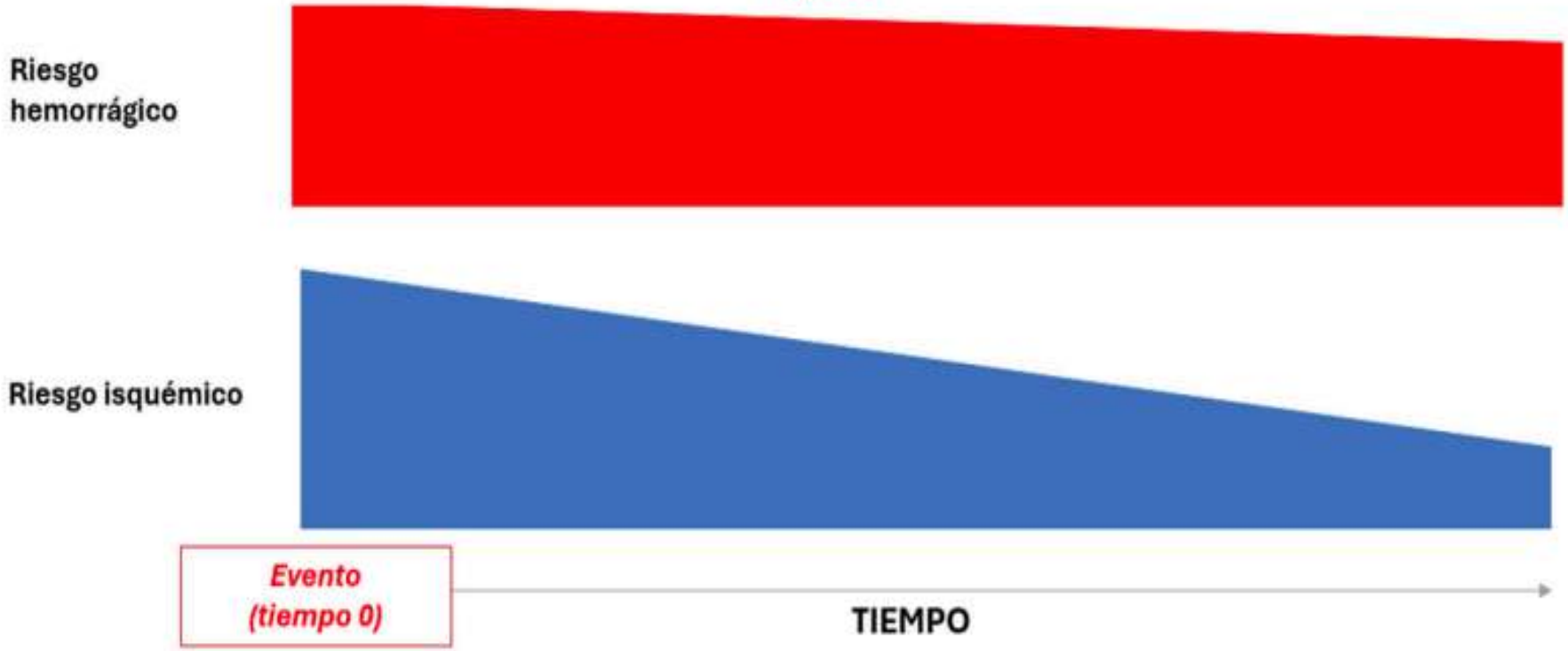
Se observa la curva de eventos mayores (infarto/muerte) .
La mayoria se acumulan en los primeros días (Historia Natural)

---- IAM
---- Muerte

Eur.Heart J.2005 .26,
BAZZINO O., HIRSCHSON PRADO A et al..

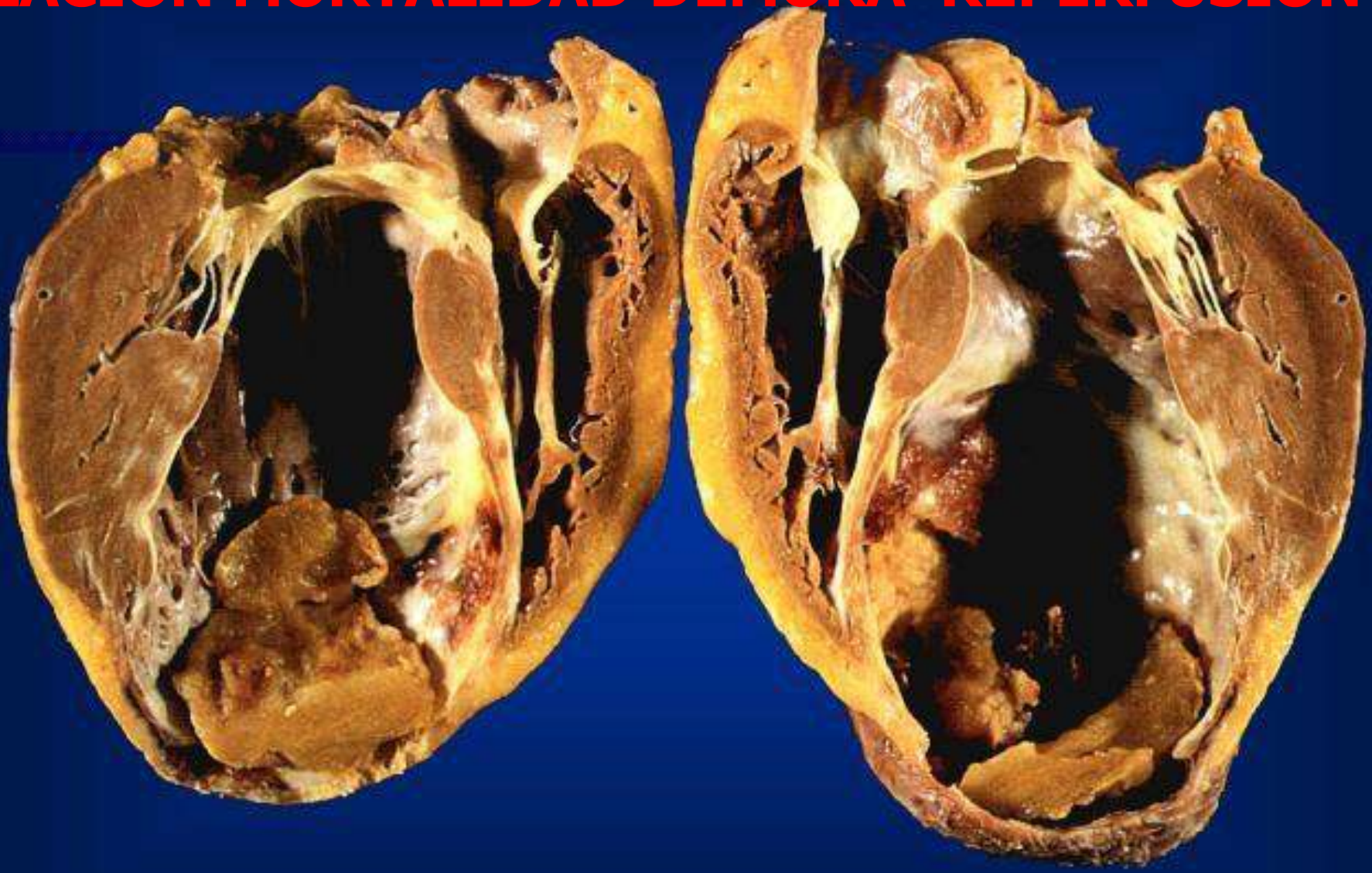
Continuo isquemia – sangrado
INDIVIDUALIZAR al PACIENTE

Procedimiento (ATC)		Clínica del paciente		Scores	
Acceso femoral, IP2Y12 mas potentes	EMV, Bif, nº *stents*, longitud de *stents*, OTC, *revasc completa*	ACO, sangrado previo, anemia, plaquetopenia, fragilidad, hepatopatía, fragilidad	SCA, DM, IRC, historia de trombosis del *stent*, IAM previo, EVP, IMC>30	PRECISE-DAPT, ARC-HBR, DAPT, CREDO-Kyoto, HASBLED	GRACE, TIMI, GUSTO, SYNTAX I y II

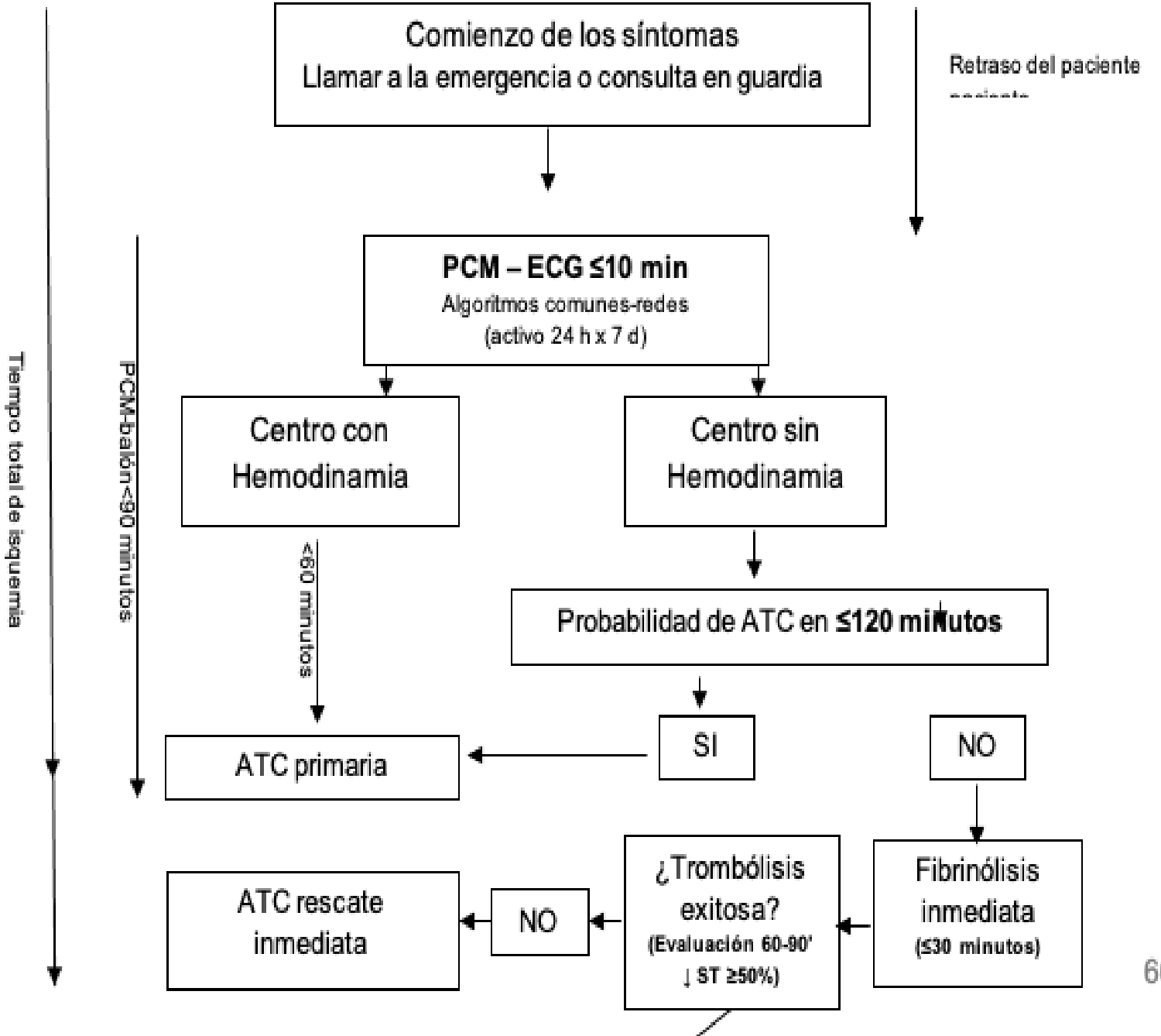


ATC: Angioplastia coronaria; iPX12: inhibidores de los receptores P2Y12; EMV: enfermedad de múltiples vasos; Bif: lesiones en bifurcación; OTC: oclusión total

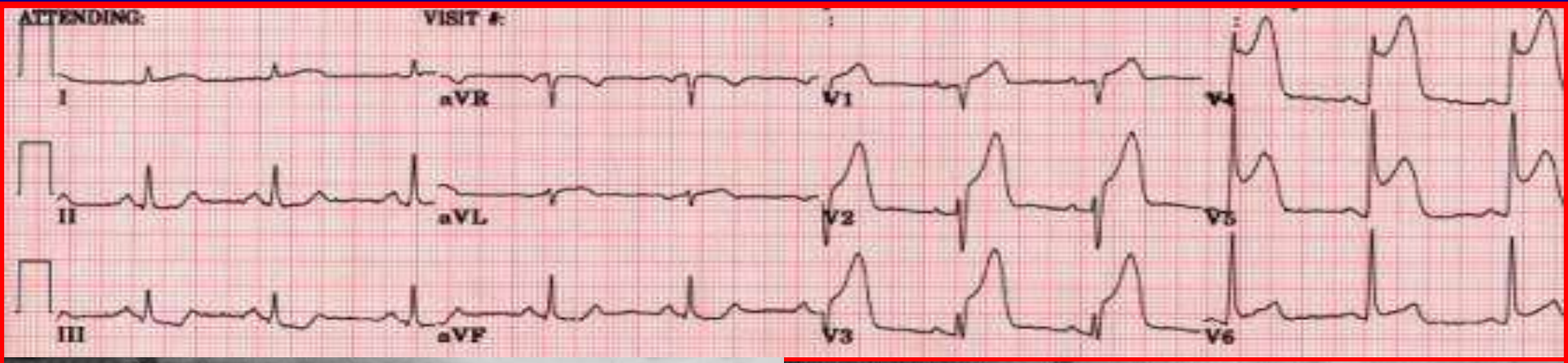
INFARTO de MIOCARDIO
RELACION MORTALIDAD DEMORA REPERFUSION

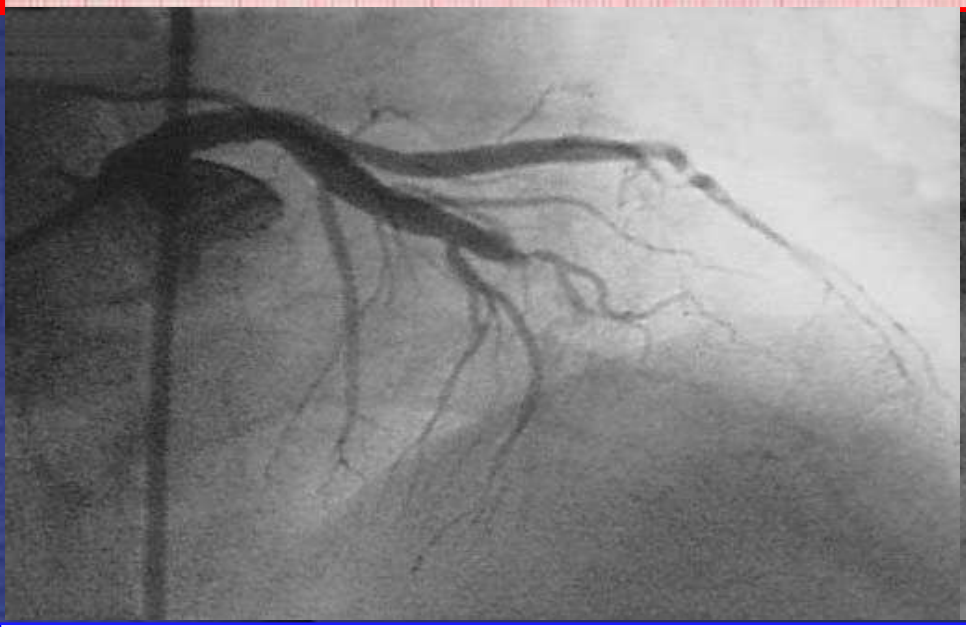


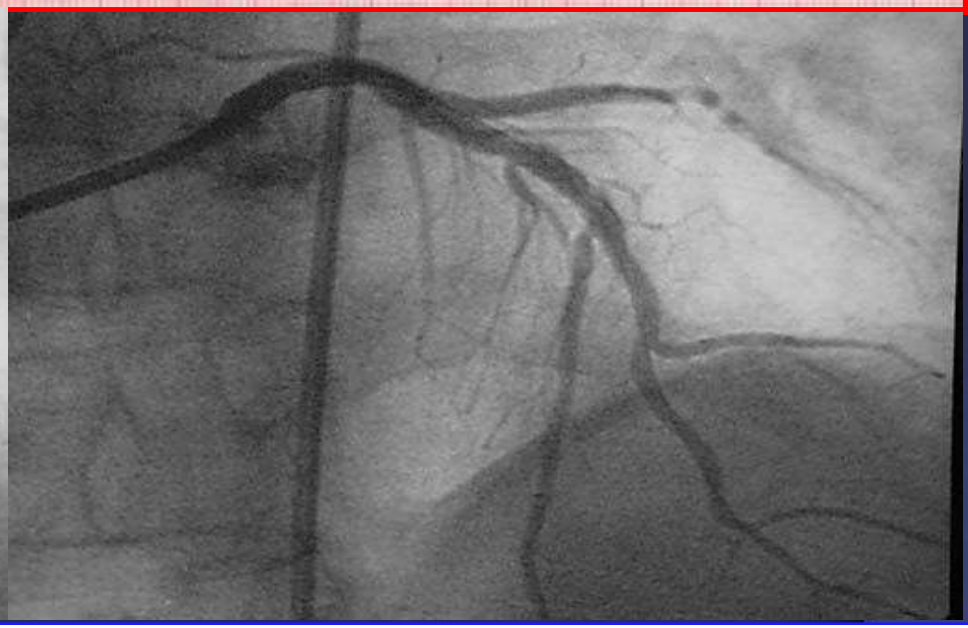
ESTRATEGIA DE REPERFUSION



INFARTO AGUDO DE MIOCARDIO



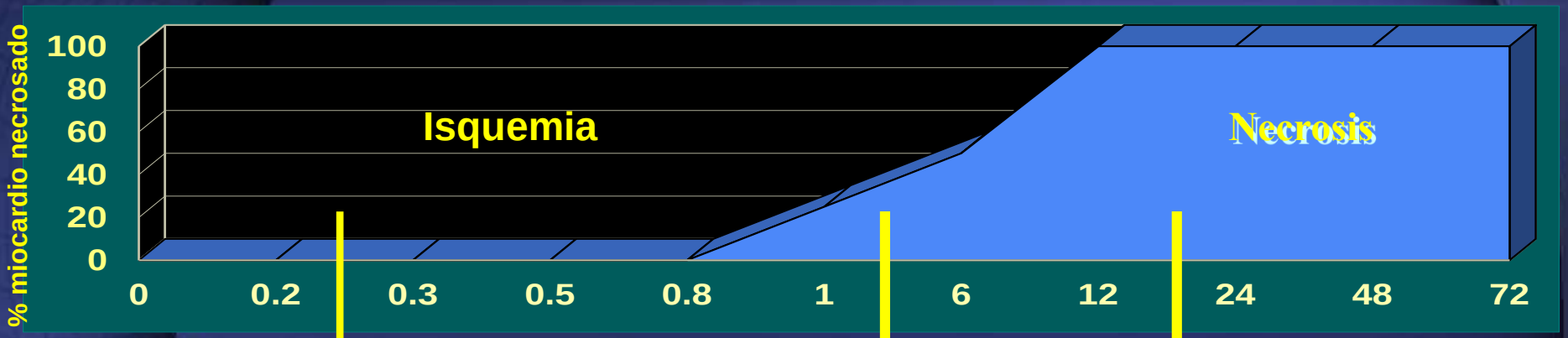


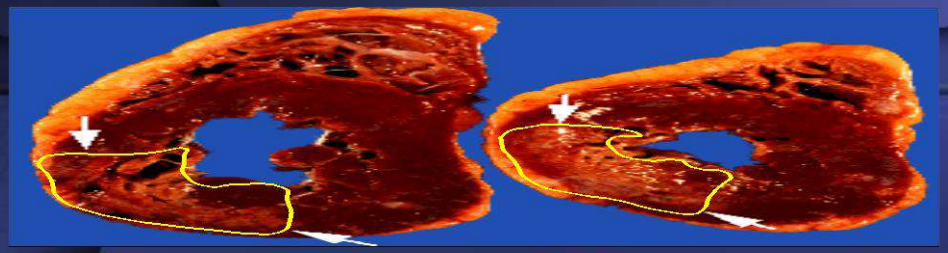


Objetivo del tratamiento de la arteria responsable del IAM

Recanalizar Precoz Completa Sostenida

Compromiso miocárdico en relación al tiempo de oclusión coronaria





9 – 12 horas

Metas de la Reperfusión en el IAM

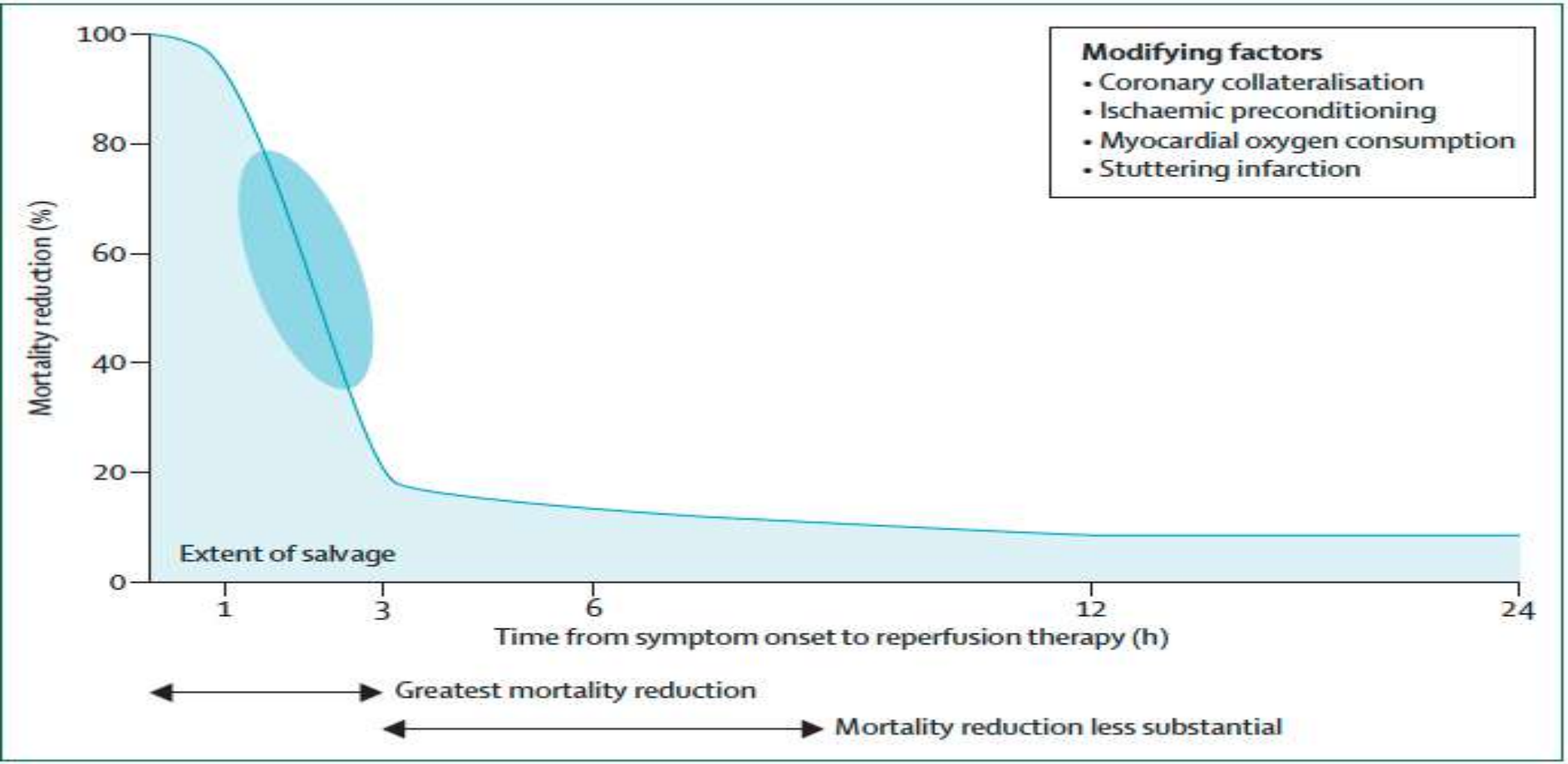
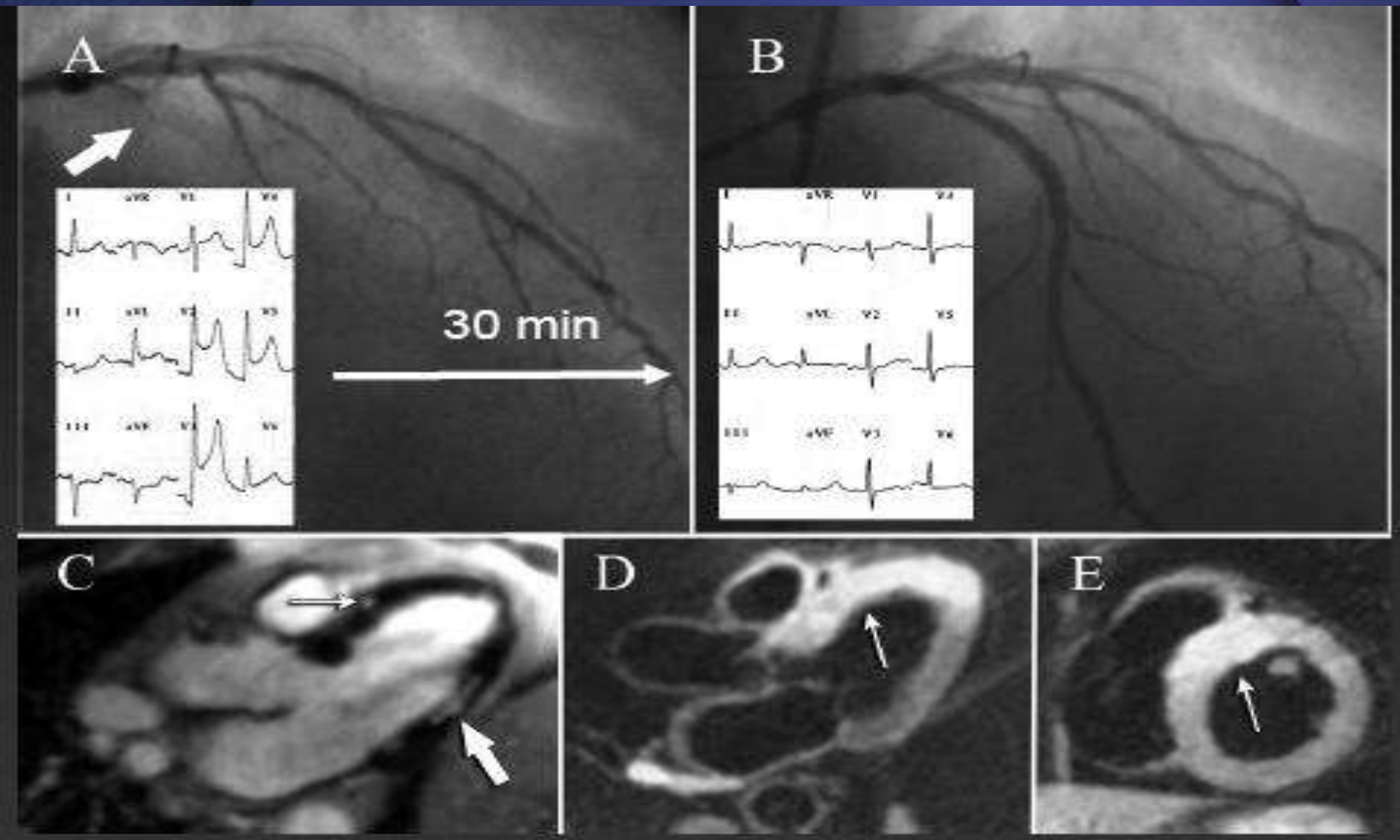


Figure 2: **The association between time to treatment, reduction in mortality, and extent of myocardial salvage**

- **Reducir el tiempo total de isquemia**

Infarto de Miocardio Abortado
Correlación entre Reperfusión, Angiografia y, RMN

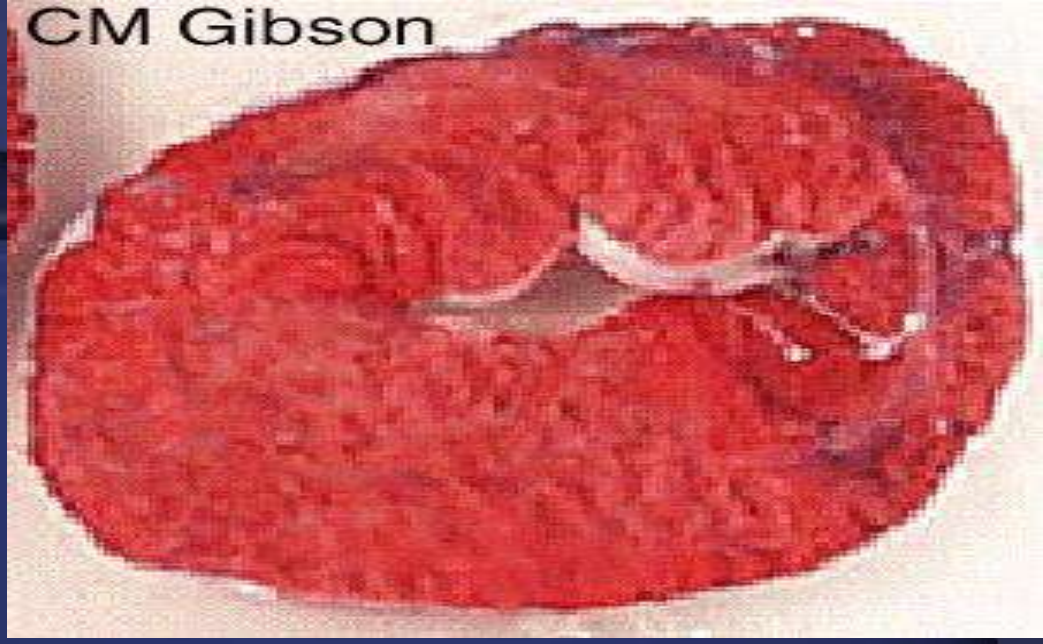


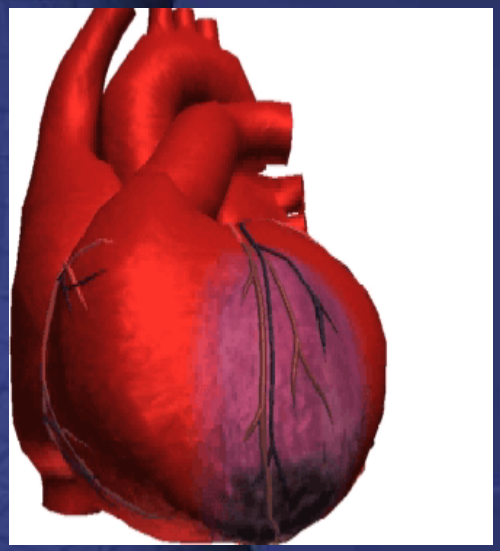
IAM

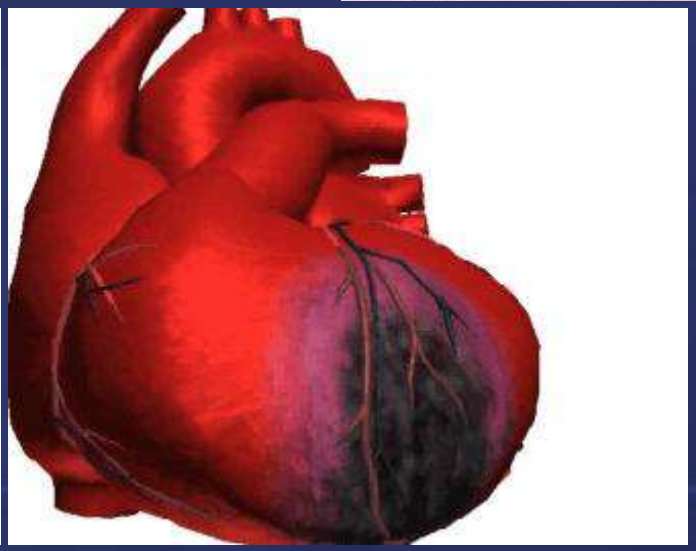
Critico=>tiempo de oclusion

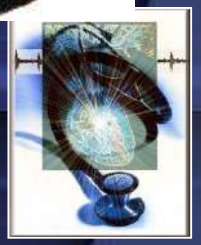
Tiempo desde la oclusión

1 – 2 hs **4 -8 hs**

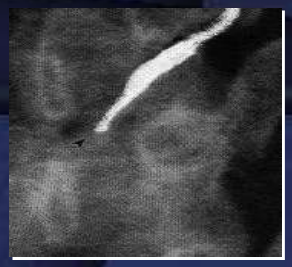




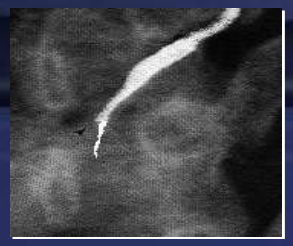




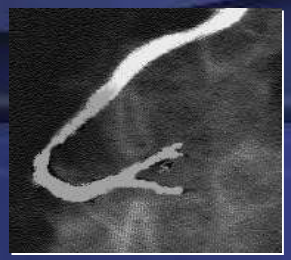
Relación entre grado de flujo TIMI y mortalidad



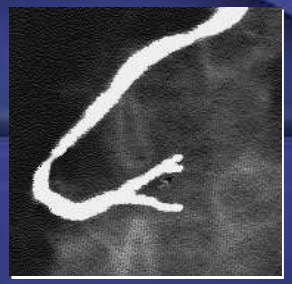
TIMI 0
Oclusión



TIMI 1
Penetración



TIMI 2
Slow Flow



TIMI 3
Flujo Normal

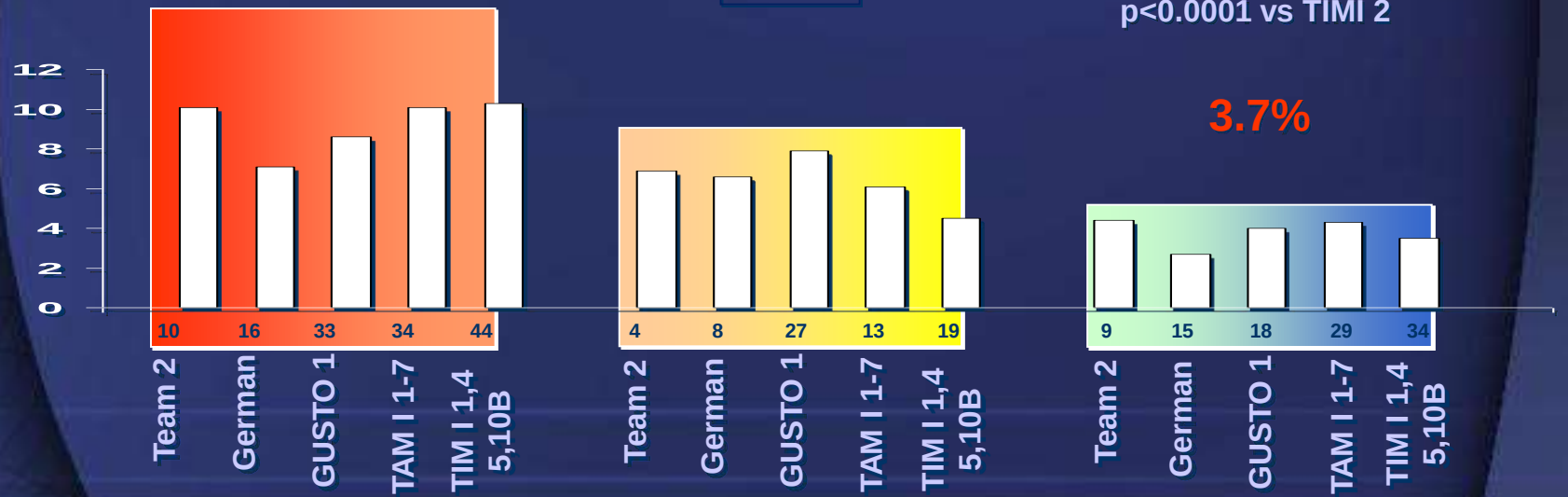
9.3%

P=0.003 vs TIMI 0/1

6.1%

p<0.0001 vs TIMI 0/1
p<0.0001 vs TIMI 2

3.7%



Tamaño de la muestra del análisis combinado: *5,498*

Gibson 1998

Tratamiento del IAM

OBJETIVOS

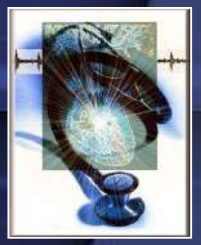
1. **Reperfusión del vaso Ocluido**

   Limitación del tamaño del IAM

   1. Fibrinolíticos — **FARMACOINVASIVA**
   2. Angioplastia primaria

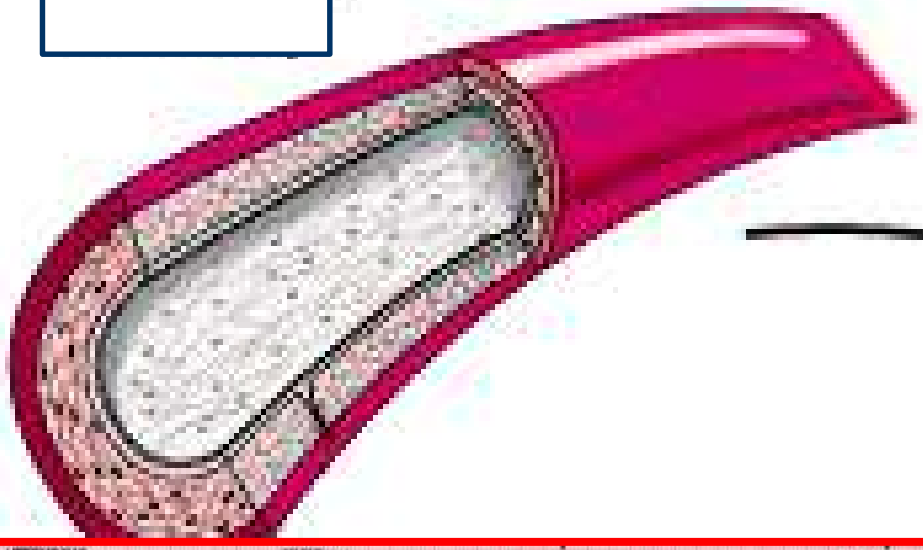
1. **Tratamiento coadyuvante**

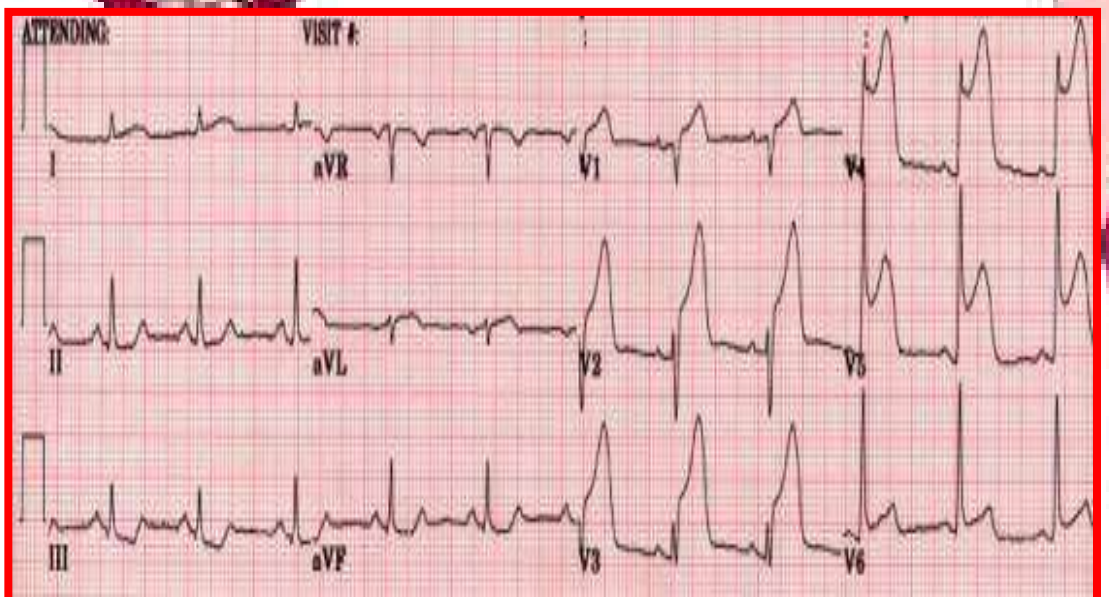
   1. Medidas generales (sedo-analgesia, O_2)
   2. AAS - (Guardia antes de derivar 500 mg)
   3. Nitritos iv
   4. β-bloqueantes oral.
   5. IECAs=Estatinas (24 hs)
   6. Antiplaquetarios, anticoagulantes

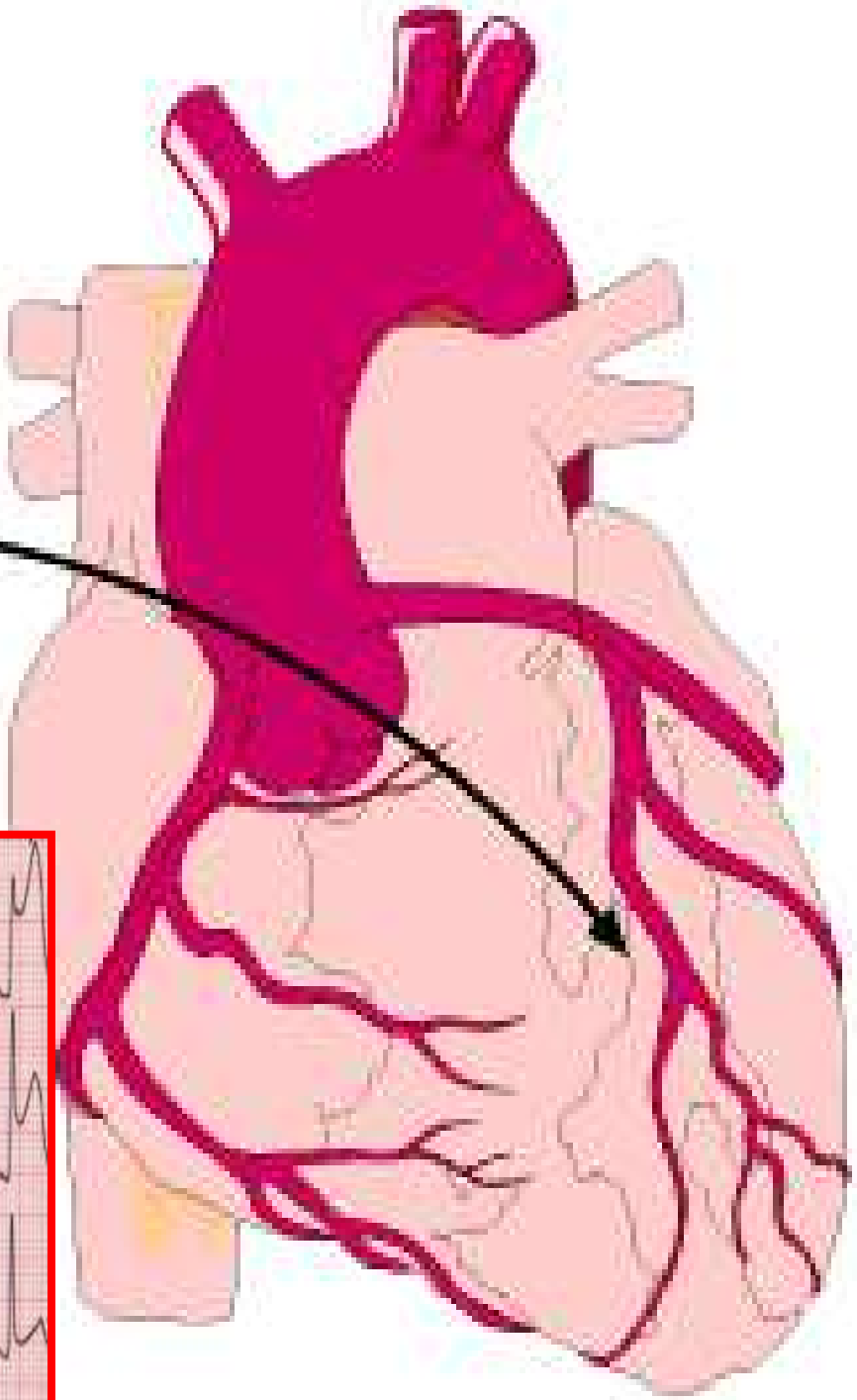


IAM TIEMPO CRITICO

= REPERFUSION =

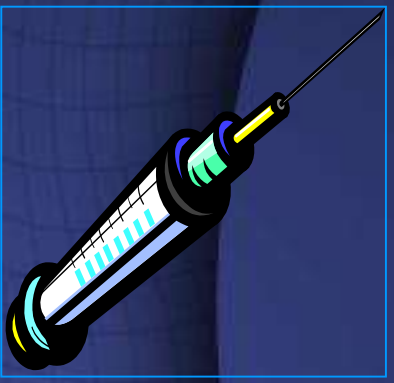




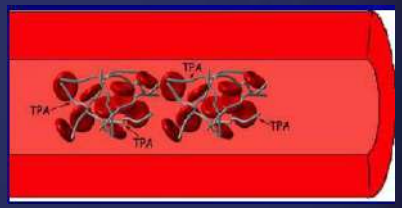


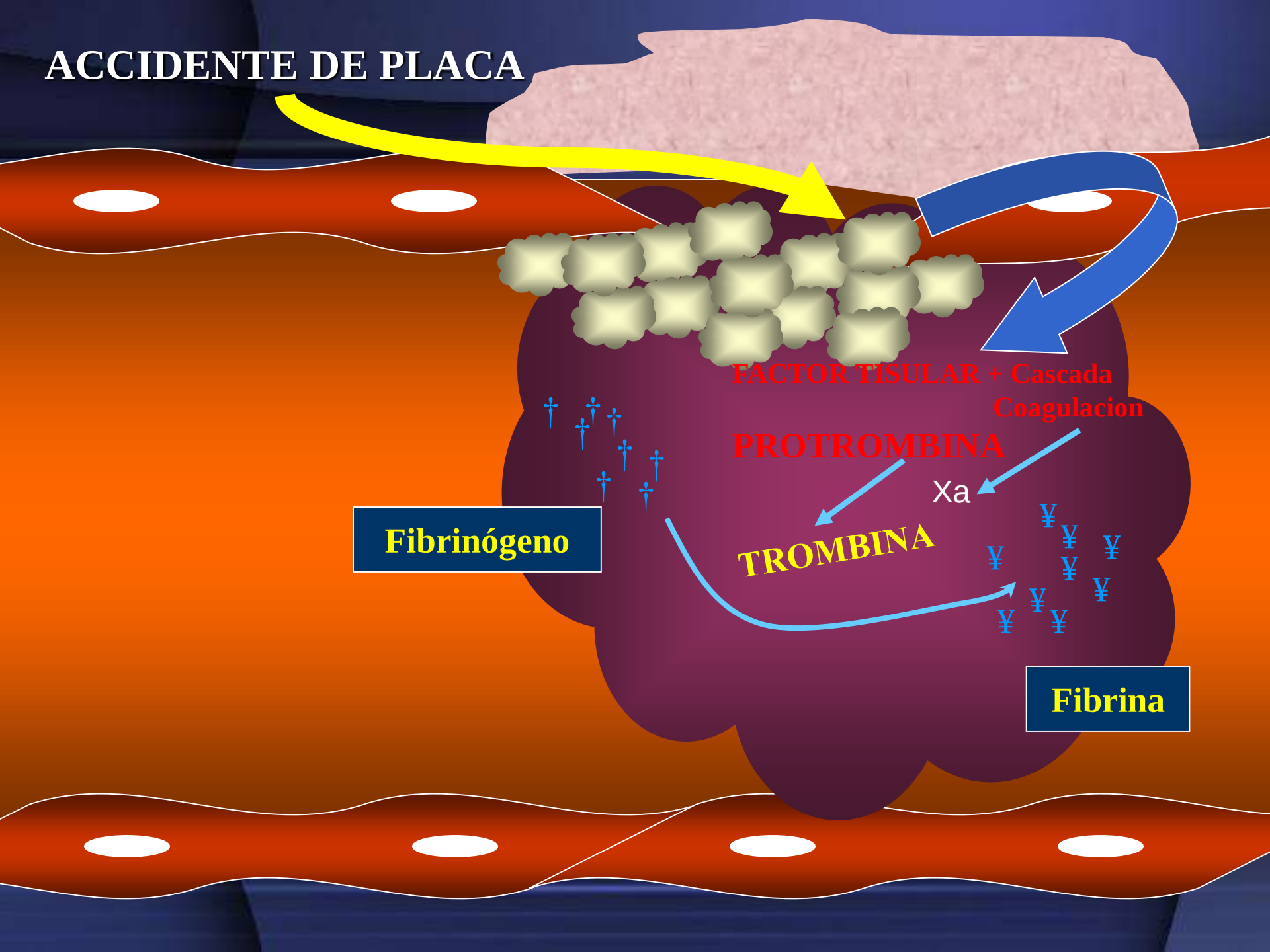
INFARTO AGUDO DE MIOCARDIO

ESTRATEGIAS DE REPERFUSION



FIBRINOLITICOS IV

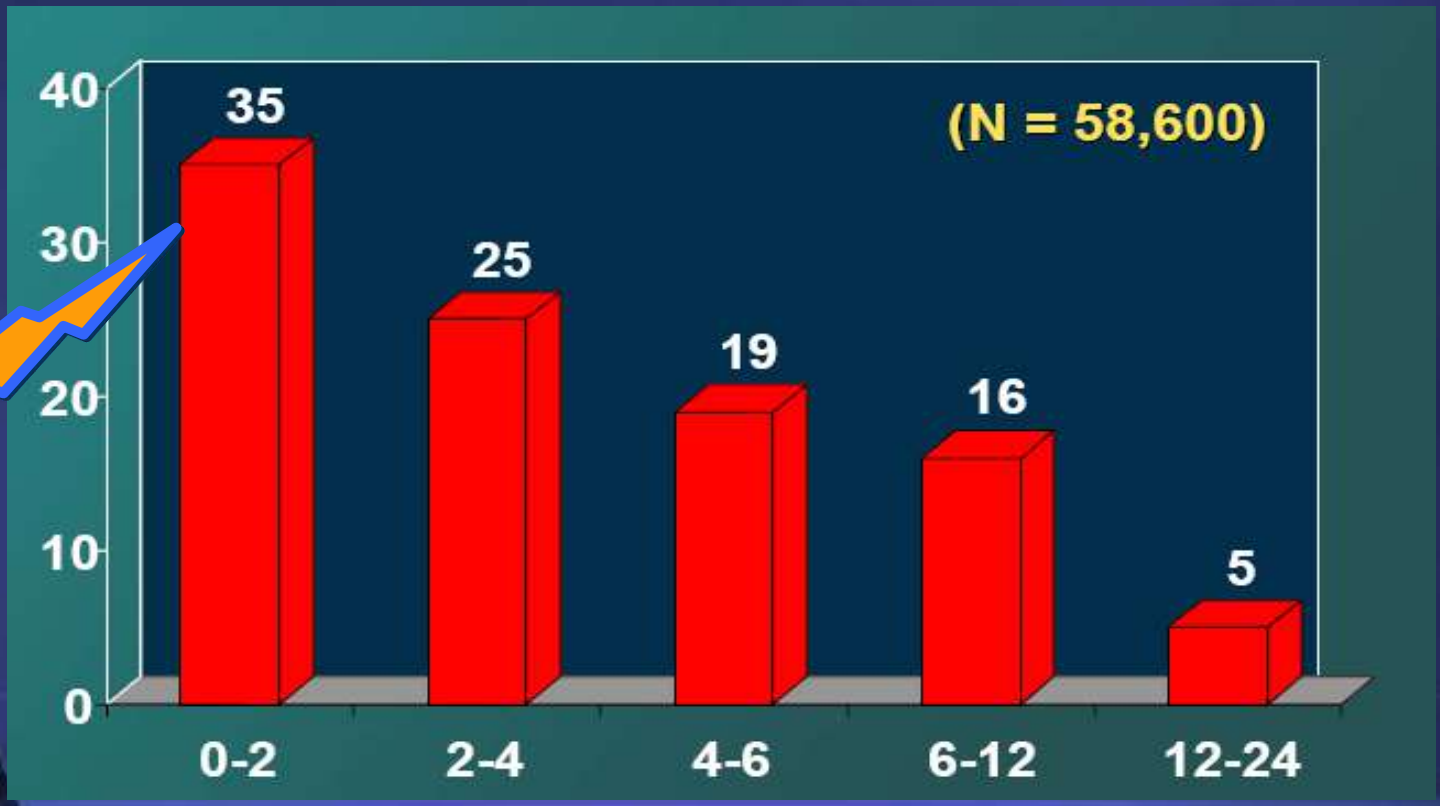



ACCIDENTE DE PLACA
FACTOR TISULAR + Cascada Coagulacion
PROTROMBINA
Xa
TROMBINA
Fibrinógeno
Fibrina

FTT

Variación del número de vidas salvadas según la demora del tratamiento con líticos

9 estudios randomizados



Tiempo total (Hs)

Vidas salvadas por cada 1000 ptes. tratados
(a 35 d)

Lancet 1994;343:311

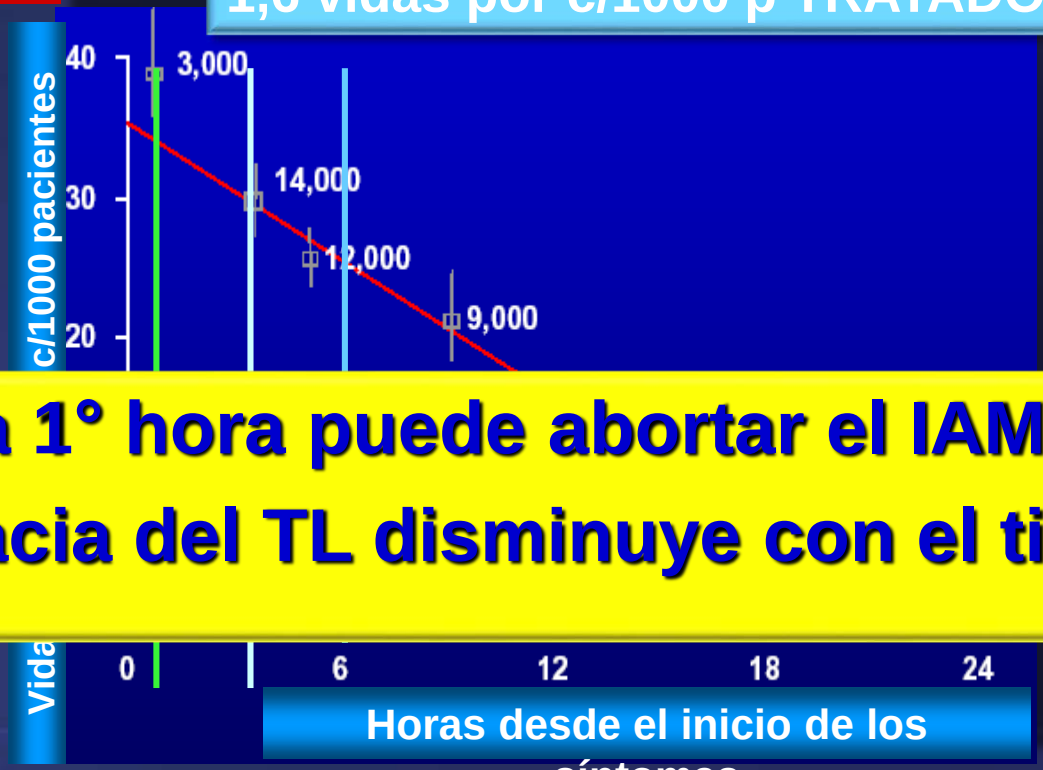
INFARTO AGUDO DE MIOCARDIO

Reducción de la Mortalidad según demora al TL

9 trials-58600p

Perdida de beneficio por cada hora de retraso: 1,6 vidas por c/1000 p TRATADOS

FTT



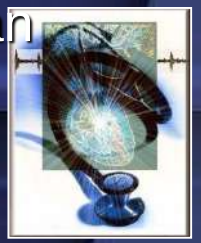
- **TL en la 1° hora puede abortar el IAM**
- **La eficacia del TL disminuye con el tiempo**

Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. *Lancet*. 1994;343:311

Trombolíticos

Trombolítico --Beneficios

- Han demostrado una limitación del tamaño del infarto.< 4hs
- Mejora de la función ventricular izquierda.
- Reducción de la mortalidad.
- Fácil y rápida administración.Ampliamente disponibles.
- No requieren un costoso laboratorio de cateterismo y personal altamente entrenado.
- Son igual de eficaces en un hospital rural y en un gran centro de referencia.



Trombolíticos

Contraindicaciones absolutas

- ACV hemorrágico o ACV de origen desconocido en cualquier momento

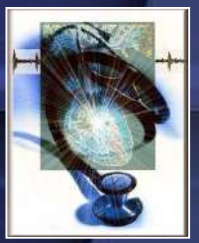
- Traumatismo o neoplasia en el sistema nervioso central

- Traumatismo/cirugía/daño encefálico reciente importante (durante las 3 semanas precedentes)

- Sangrado gastrointestinal durante el último mes

- Alteración hemorrágica conocida

- Disección aórtica

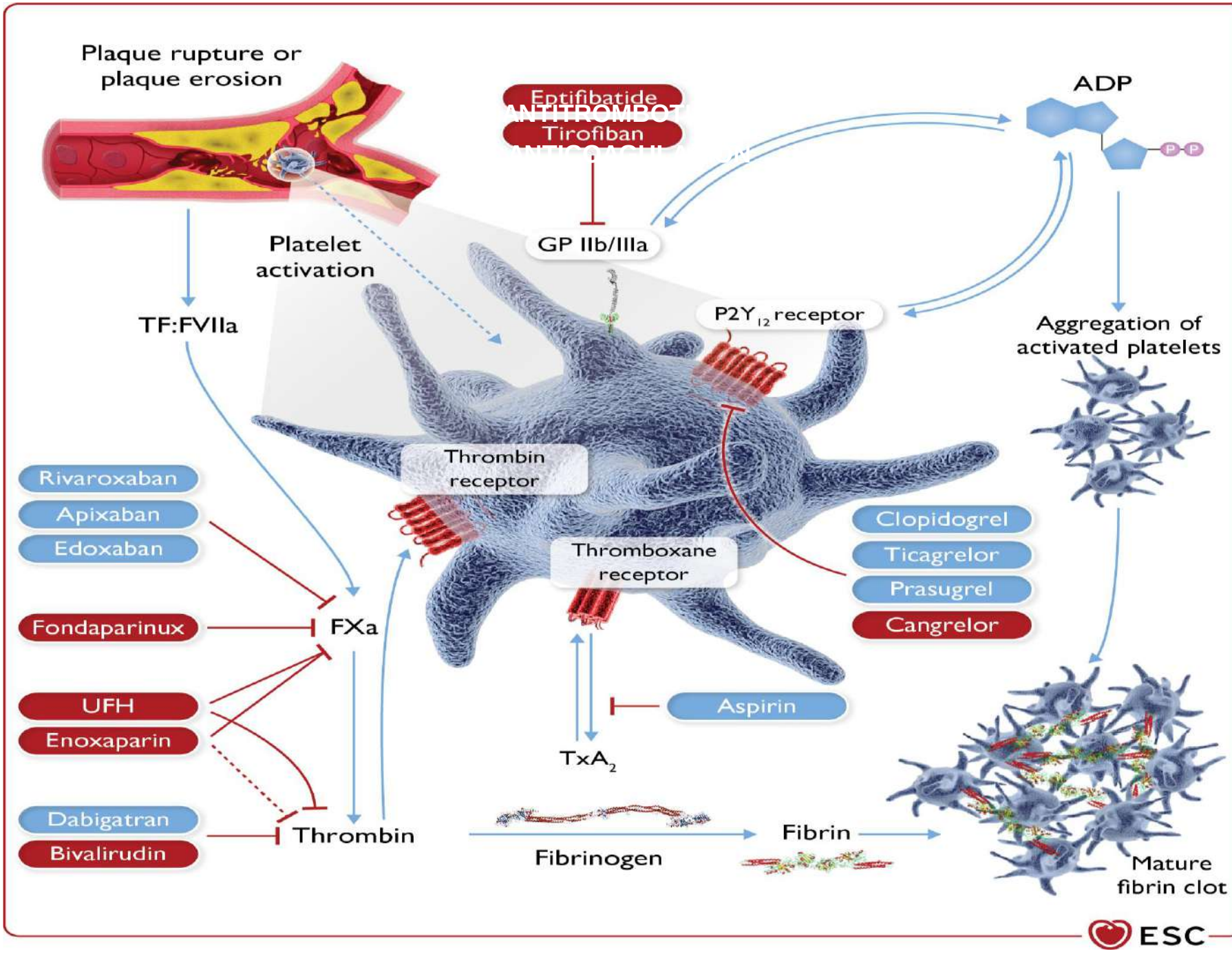


Se recomienda el uso de fibrinolíticos fibrinoespecíficos sobre no fibrinoespecíficos.(98,226)	I	A
Se sugiere preferir tenecteplasa (TNK) por eficacia equivalente a t-PA, menor sangrado no cerebral y administración en bolo único ajustado por peso, lo que simplifica la práctica y reduce tiempos y errores.	I	B
En pacientes ≥75 años, si se indica fibrinólisis, usar TNK a mitad de dosis solo dentro de una estrategia farmacoinvasiva	IIa	B
En sistemas sanitarios con capacidad para implementarlos, se recomienda la fibrinólisis prehospitalaria con fibrinolíticos fibrinoespecíficos en bolo sobre la fibrinólisis hospitalaria.(177)	I	B

Doses of fibrinolytic agents and antithrombotic co-therapies

ESC
European Socie
of Cardiology

Drug	Initial treatment	Specific contra-indications
Doses of fibrinolytic therapy		
Streptokinase	1.5 million units over 30–60 min i.v.	Previous treatment with streptokinase or anistreplase
Alteplase (tPA)	15 mg i.v. bolus 0.75 mg/kg i.v. over 30 min (up to 50 mg) then 0.5 mg/kg i.v. over 60 min (up to 35 mg)	
Reteplase (rPA)	10 units + 10 units i.v. bolus given 30 min apart	
Tenecteplase (TNK-tPA)	Single i.v. bolus: 30 mg (6000 IU) if <60 kg 35 mg (7000 IU) if 60 to <70 kg 40 mg (8000 IU) if 70 to <80 kg 45 mg (9000 IU) if 80 to <90 kg 50 mg (10000 IU) if ≥90 kg It is recommended to reduce to half-dose in patients ≥75 years of age.	Es el fármaco de elección para iniciar reperfusión . Si ATC > 90 -120 min. Efectividad: 85% flujo TIMI 3 ACV isquémico agudo: aprobado disponible en centros con unidad de Stroke Las ventajas clave la SAC la recomienda: Bolo único IV en <10 segundos. No hace falta bomba de infusión **ºtiempo es músculo=vida"**

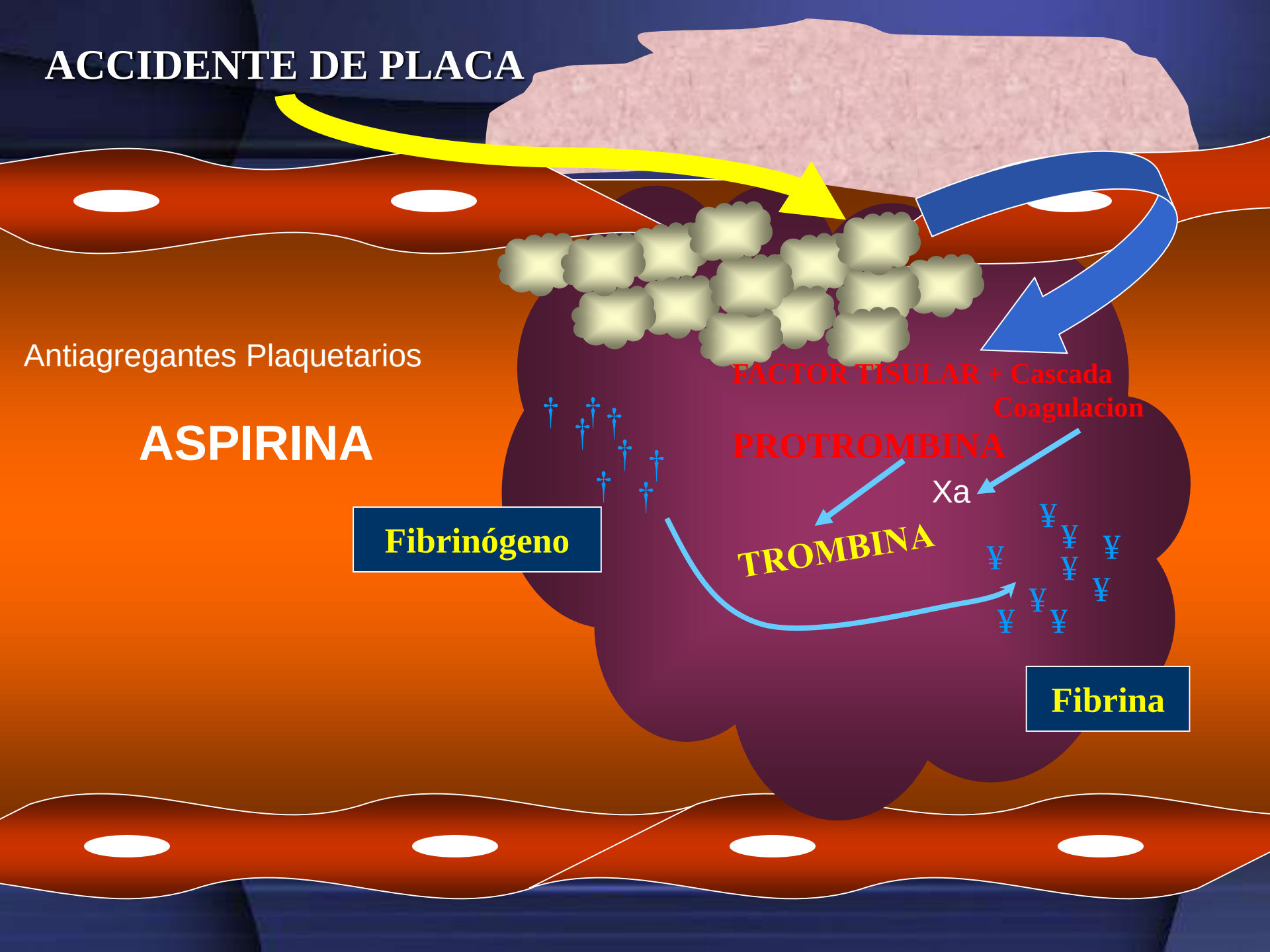

Plaque rupture or plaque erosion
Eptifibatide
Tirofiban
ADP
GP IIb/IIIa
Platelet activation
TF:FVIIa
P2Y12 receptor
Aggregation of activated platelets
Thrombin receptor
Rivaroxaban
Apixaban
Edoxaban
Clopidogrel
Ticagrelor
Prasugrel
Cangrelor
Thromboxane receptor
Fondaparinux
FXa
UFH
Enoxaparin
Aspirin
TxA2
Dabigatran
Bivalirudin
Thrombin
Fibrinogen
Fibrin
Mature fibrin clot
ESC

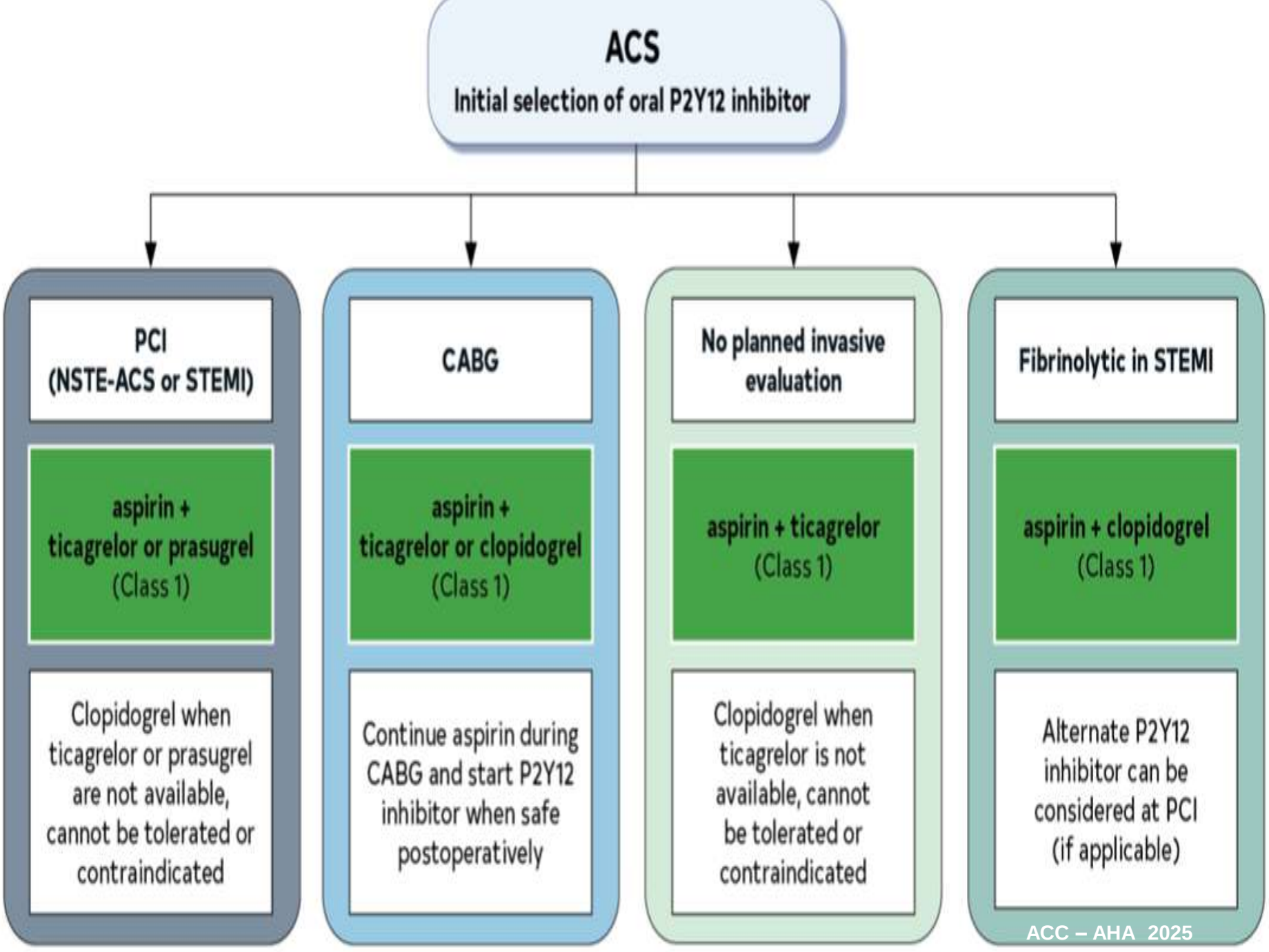
ASPIRINA

- ***Indicacion Clase I A***

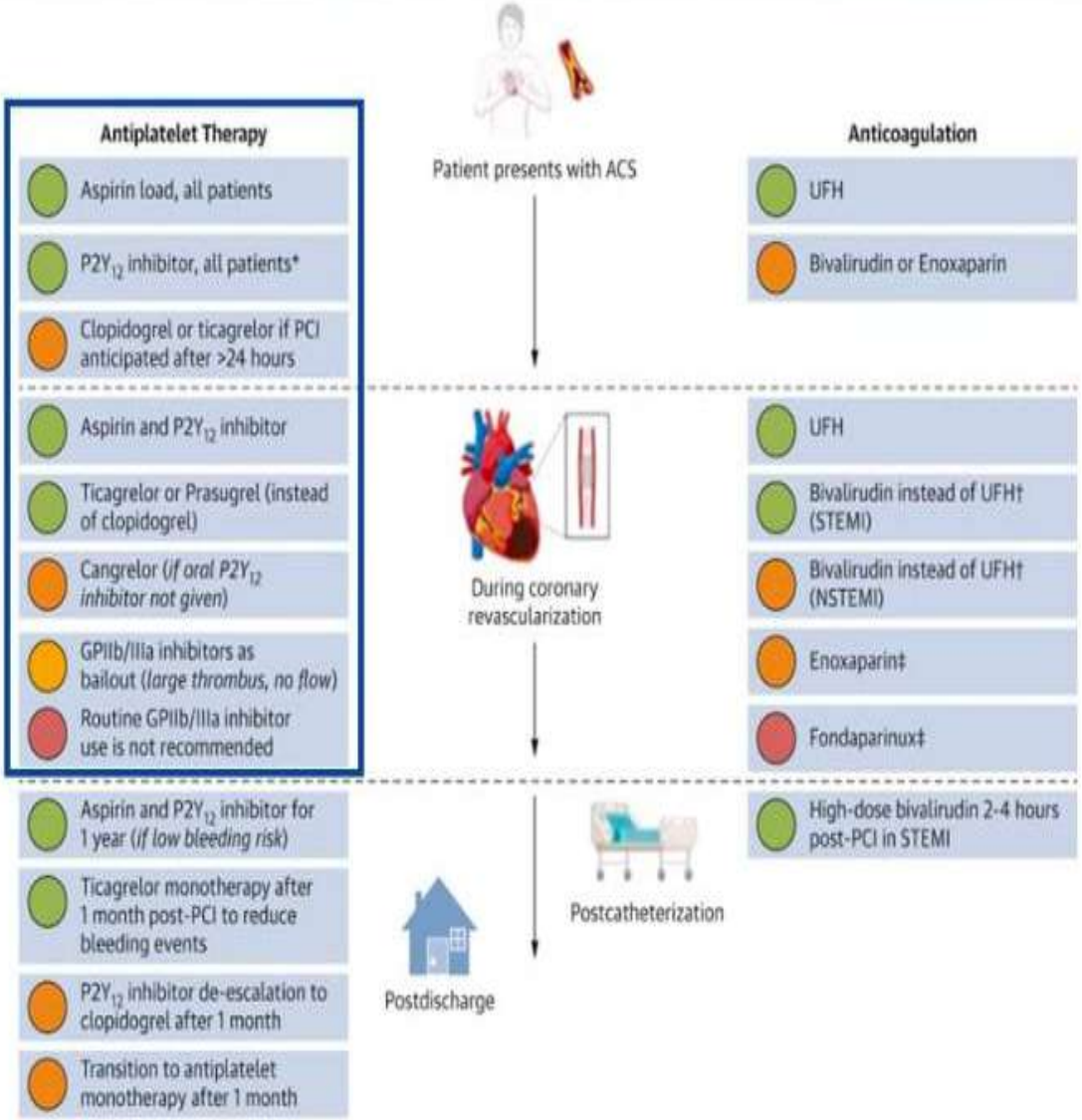
- **AAS debe ser administrada masticada en una dosis de 500 mg (GUARDIA) y continuar 100 mg - dia.**

- ISIS II (1988) Solo adminitrando AAS se reduce un 23%(RR) + STK(25%) un 42% (Ambas) la mortalidad a 35 dias .


ACCIDENTE DE PLACA
Antiagregantes Plaquetarios
ASPIRINA
FACTOR TISULAR + Cascada Coagulacion
PROTROMBINA
Xa
TROMBINA
Fibrinógeno
Fibrina


ACS
Initial selection of oral P2Y12 inhibitor
PCI (NSTE-ACS or STEMI)
aspirin + ticagrelor or prasugrel (Class 1)
Clopidogrel when ticagrelor or prasugrel are not available, cannot be tolerated or contraindicated
CABG
aspirin + ticagrelor or clopidogrel (Class 1)
Continue aspirin during CABG and start P2Y12 inhibitor when safe postoperatively
No planned invasive evaluation
aspirin + ticagrelor (Class 1)
Clopidogrel when ticagrelor is not available, cannot be tolerated or contraindicated
Fibrinolytic in STEMI
aspirin + clopidogrel (Class 1)
Alternate P2Y12 inhibitor can be considered at PCI (if applicable)
ACC – AHA 2025

Antiplatelet and Anticoagulant Therapy for ACS
2025 ACC/AHA Guidelines



UFH, unfractionated heparin.
*If planned for PCI within 24 hours, P2Y12 inhibitor administration can be deferred unt coronary anatomy has been defined.

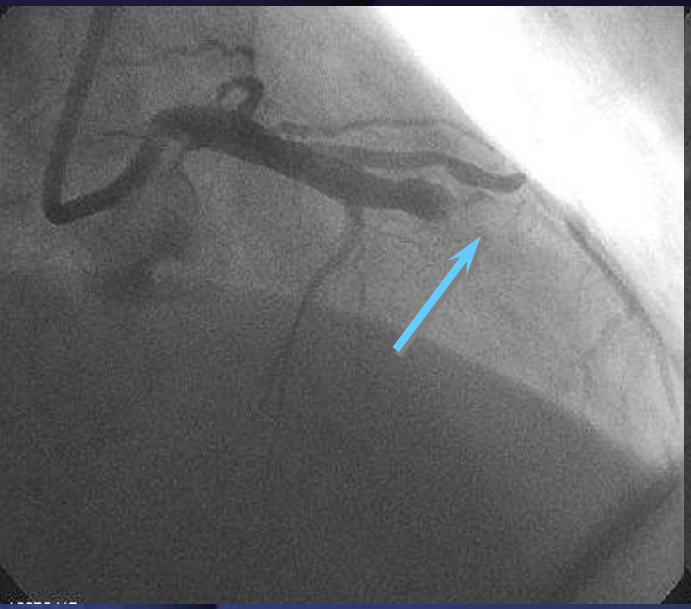
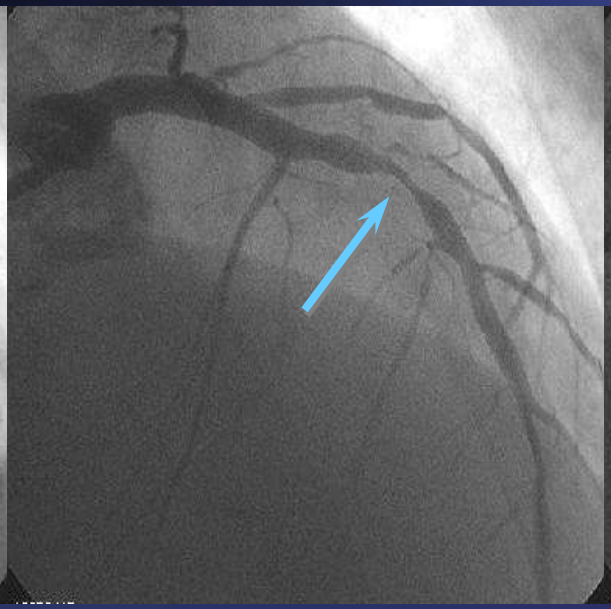
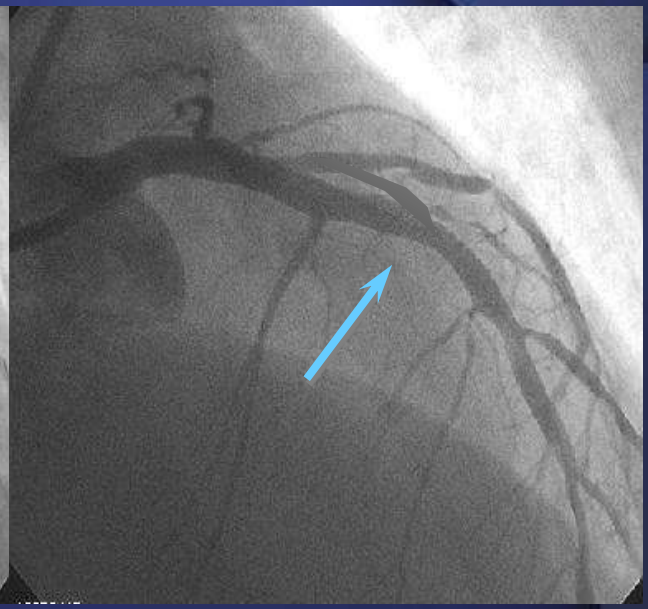
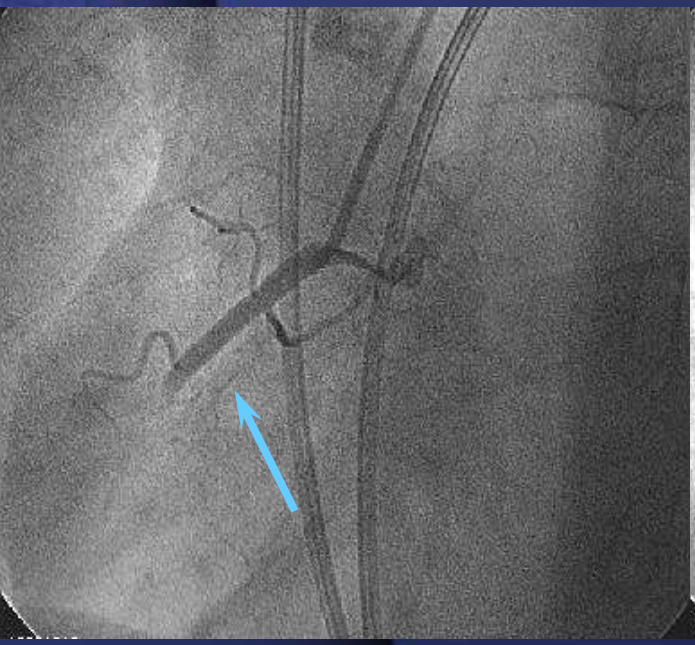
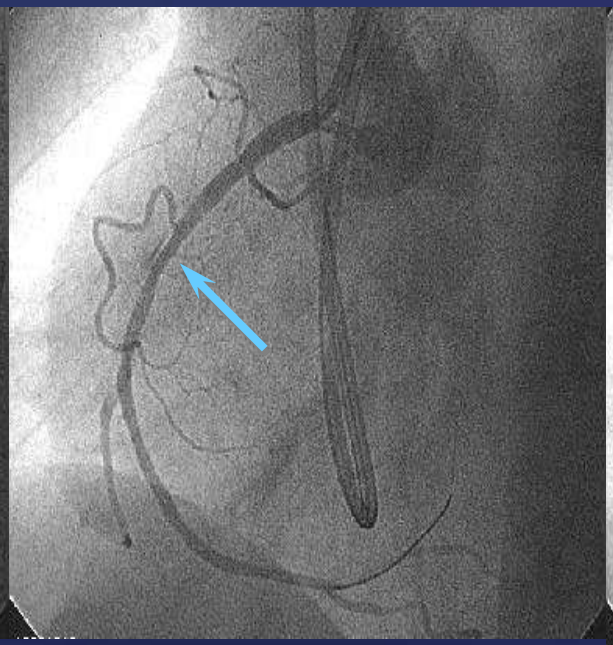
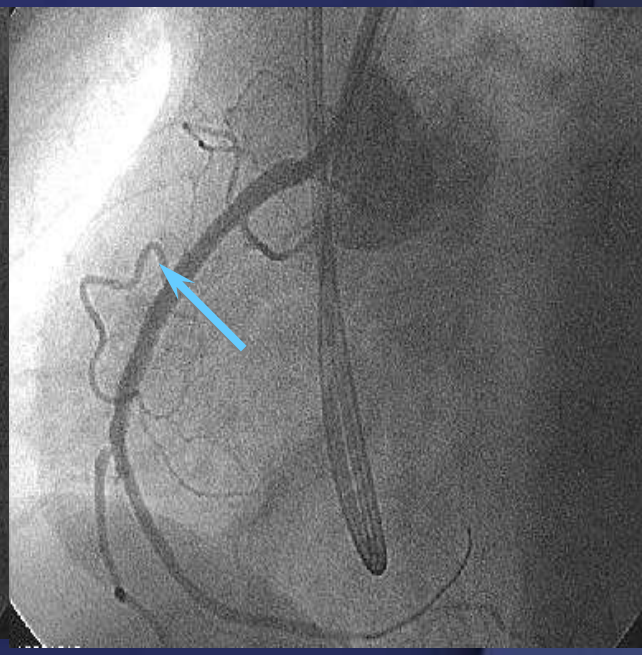
Bikdeli B, et al. J Am Coll Cardiol. 2025;85:2074-2078.

Doses of anti-thrombin co-therapies

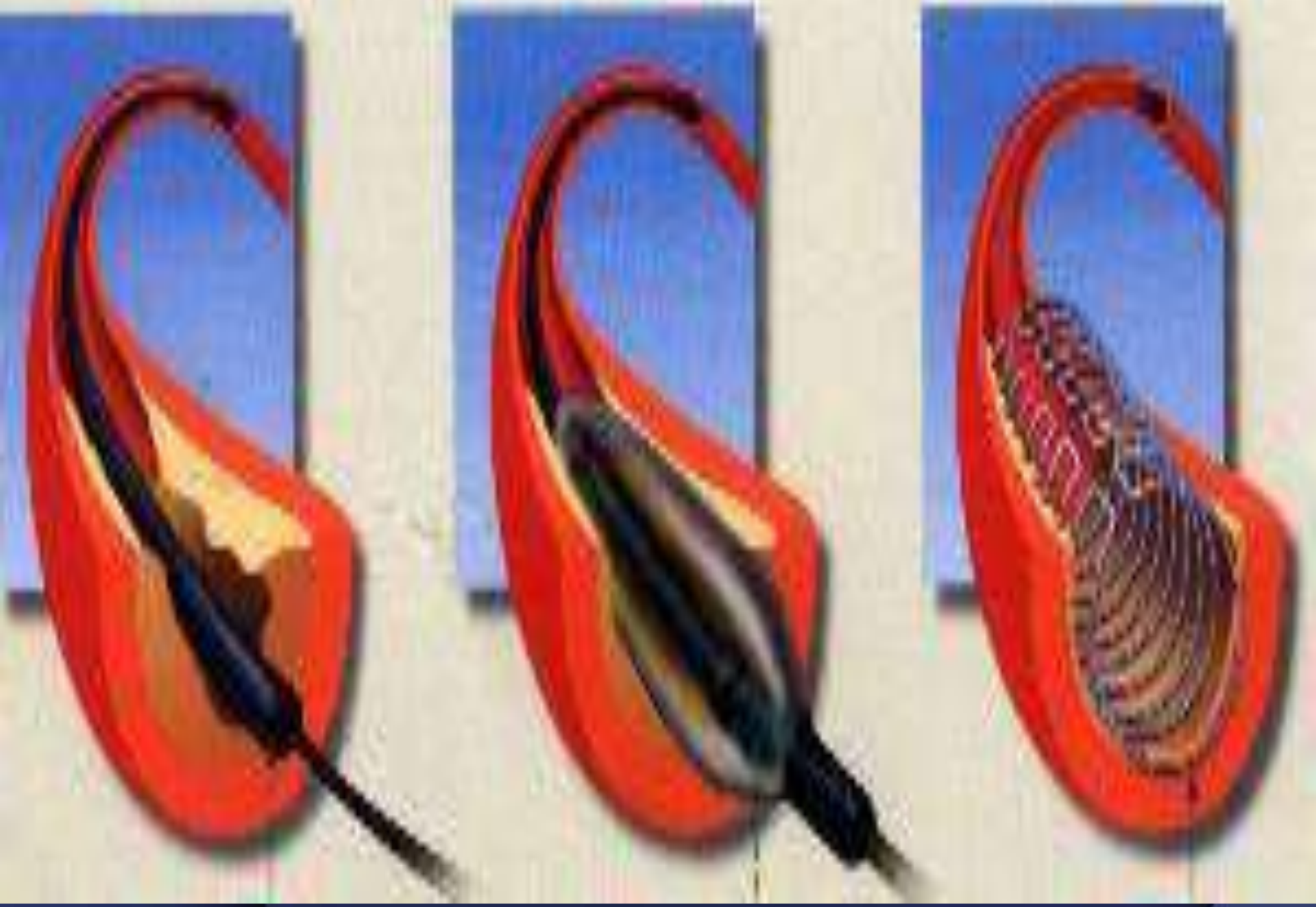
Doses of antithrombin co-therapies	
ANGIOPLASTIA	
Unfractionated heparin	70-100 U/kg i.v. bolus 50-60 U/kg i.v. bolus
Enoxaparin	0.5 mg/kg i.v. bolus.
Bivalirudin	0.75 mg/kg i.v. bolus followed by i.v infusion of 1.75 mg/kg/h for up to 4 h after the procedure as clinically warranted. After cessation of the 1.75 mg/kg/h infusion, a reduced infusion dose of 0.25 mg/kg/h may be continued for 4-12 h as clinically necessary.
ESTRATEGIA CONSERVADORA	
Unfractionated heparin	60 U/kg i.v. bolus with a maximum of 4000 U followed by an i.v. infusion of 12 U/kg with a maximum of 1000 U/h for 24-48 h. Target aPTT: 50-70 s or 1.5 to 2.0 times that of control to be monitored at 3, 6, 12 and 24 h.
Enoxaparin	In patients < 75 years of age: 30 mg i.v. bolus followed 15 min later by 1 mg/kg s.c. every 12 h until hospital discharge for a maximum of 8 days The first two doses should not exceed 100 mg. In patients > 75 years of age: no i.v. bolus; start with first s.c. dose of 0.75 mg/kg with a maximum of 75 mg for the first two s.c. doses. In patients with creatinine clearance of < 30 mL/min, regardless of age, the s.c. doses are given once every 24 h.
Fondaparinux	2.5 mg i.v. bolus followed by a s.c. dose of 2.5 mg once daily up to 8 days or hospital discharge.

Anticoagulación parenteral asociado al uso de fibrinolíticos.(99,244)	I	A
En pacientes sin estrategia invasiva (angioplastia de rescate o farmacoinvasiva) se recomienda enoxaparina durante 8 días o hasta el alta.	I	A
Fondaparinux como alternativa a la enoxaparina	IIb	C
En pacientes con estrategia invasiva que requieren régimen corto de anticoagulación se recomienda enoxaparina o HNF.	I	C
En pacientes en los que se realiza una angioplastia, suspender anticoagulantes luego del procedimiento.	I	C

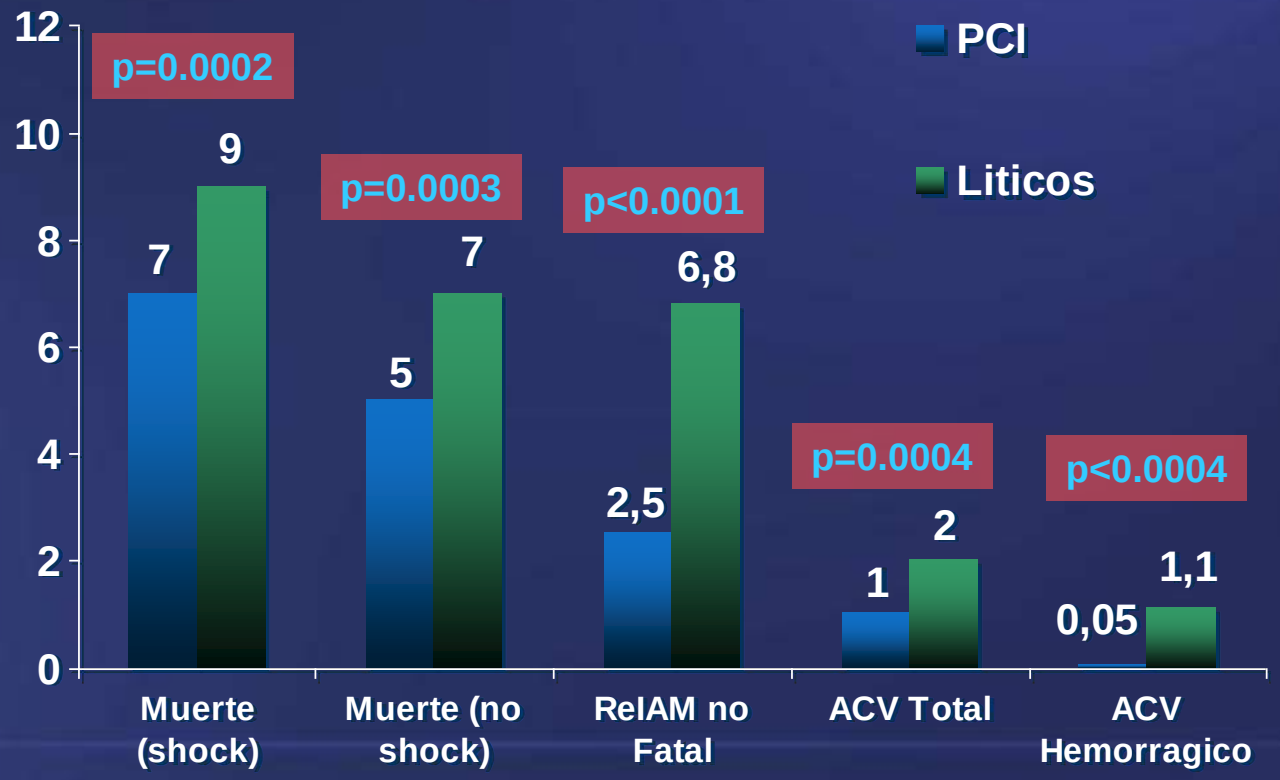
ANGIOPLASTIA 1RIA

ATC - STENT



Metanalisis de 23 Ensayos Randomizados sobre PCI vs Líticos (n=7739 p).

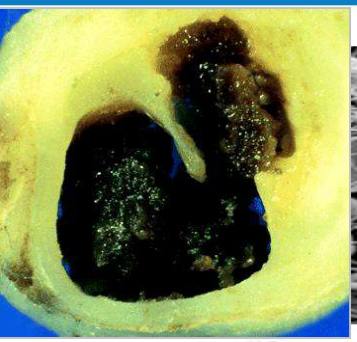


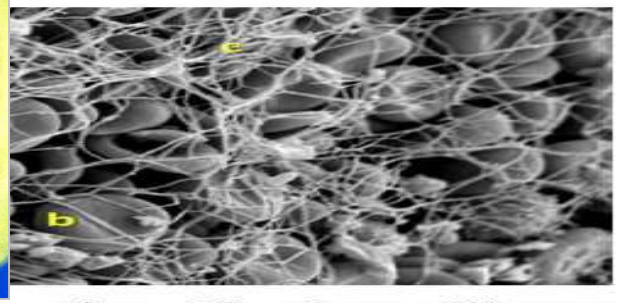
Keeley EC, Boura JA, Grines CL. Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials. Lancet. 2003 Jan 4;361(9351):13-20

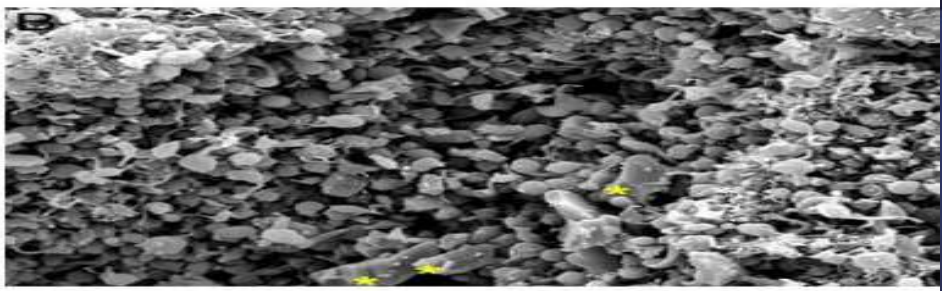
Se recomienda la revascularización por angioplastia sobre los fibrinolíticos en pacientes que se presentan dentro de las 12hs, siempre que pueda realizarse dentro de los 120 minutos desde el diagnóstico.	I	A
Pacientes que se presentan con una ventana >12hs y persistencia de los síntomas, inestabilidad hemodinámica o arritmias graves deben ser considerados para revascularización por angioplastia	I	C
Se sugiere la revascularización por angioplastia en pacientes que se presentan entre las 12 y 24 del comienzo de los síntomas.	IIa	B
En pacientes que se presentan entre la 24 y 72hs que se encuentran asintomáticos, sin insuficiencia cardíaca o arritmias ventriculares se podría realizar angioplastia.	IIb	C

Eficacia Liticos depende del Tiempo vs. ATC

> 3 horas	< 3 horas

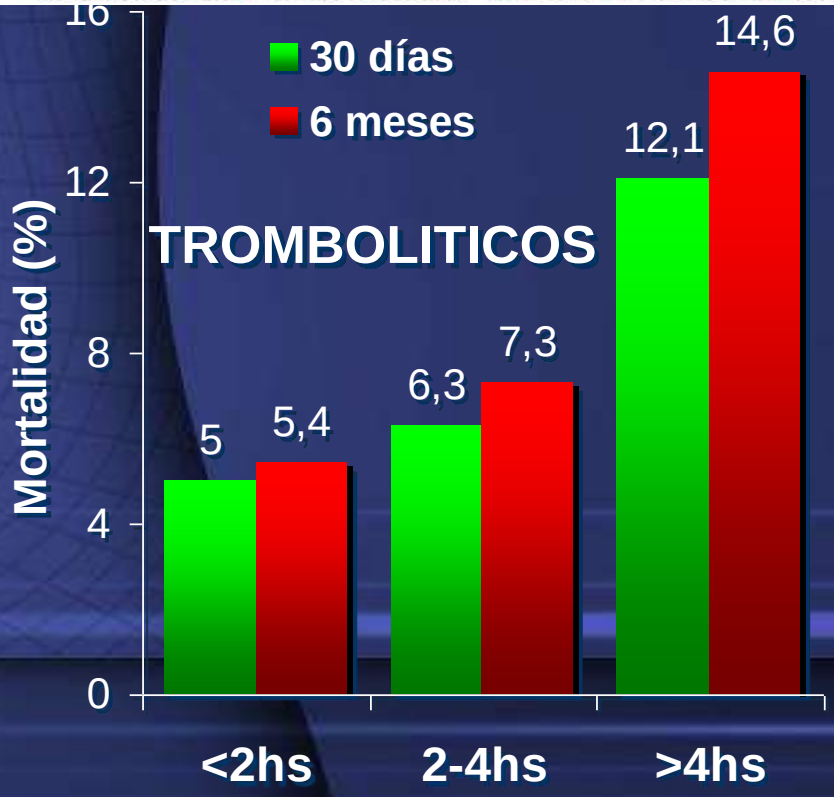


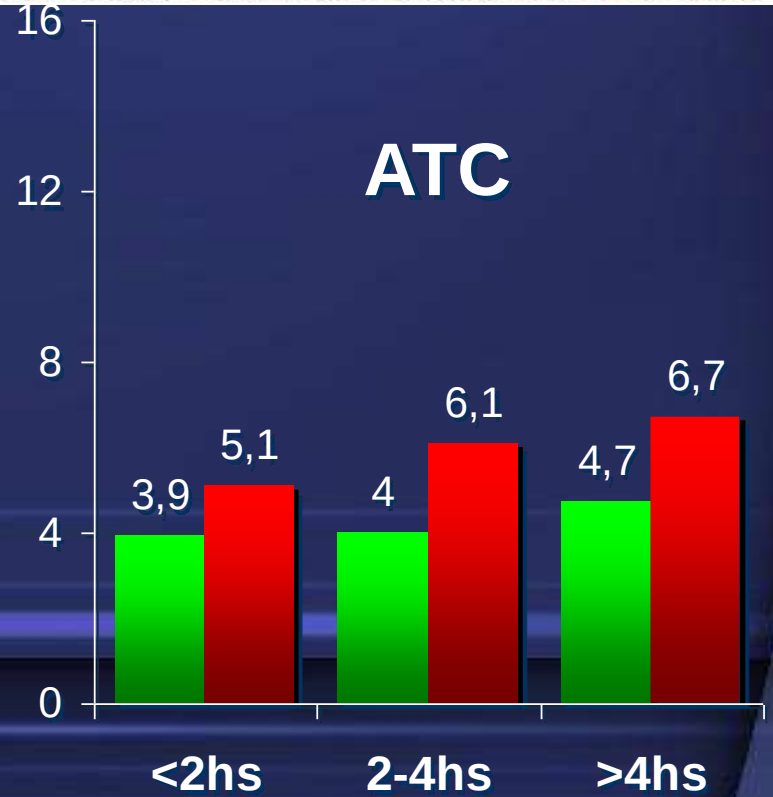




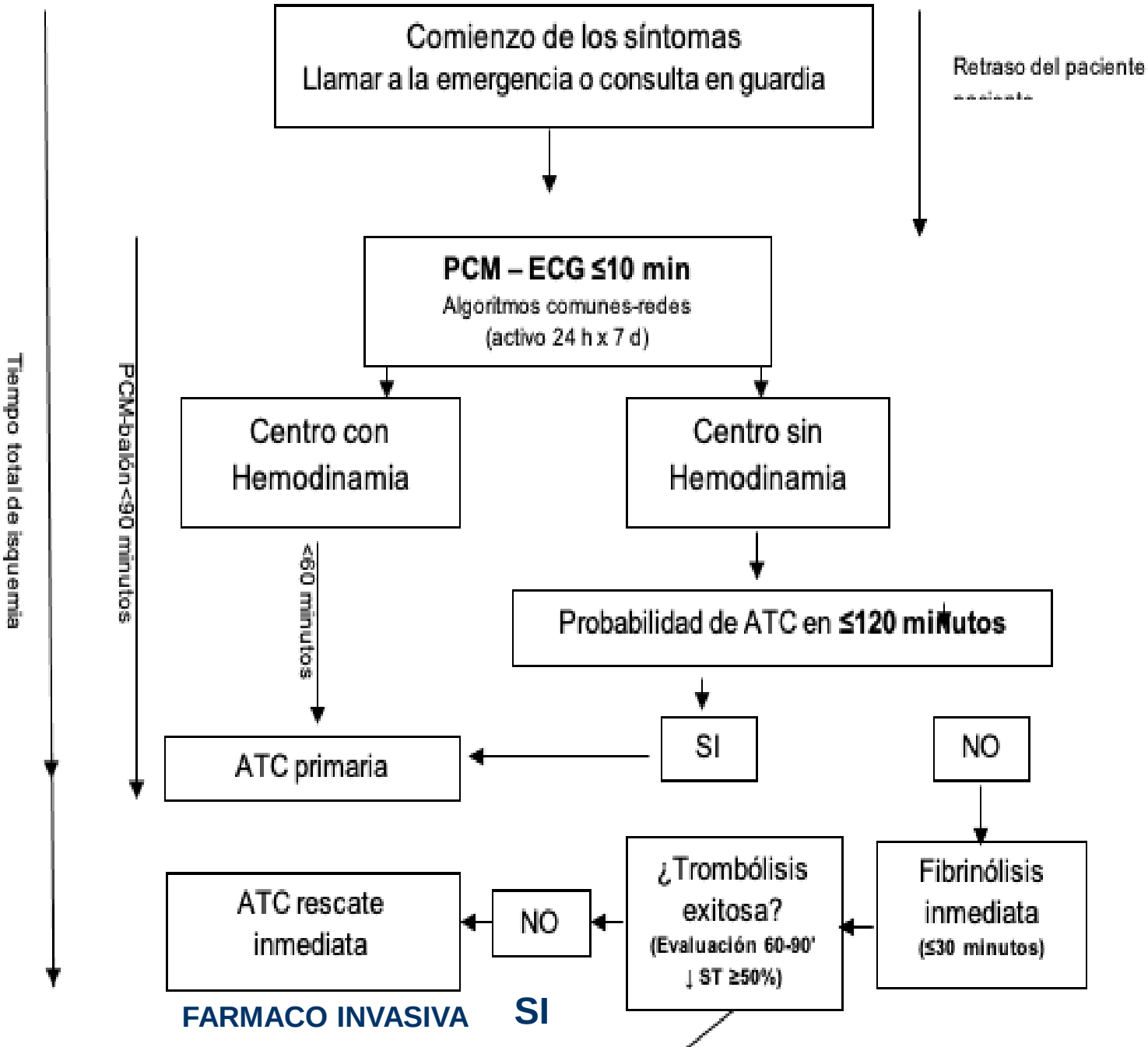
Scanning Electron Micrograph at 3,000x Magnification

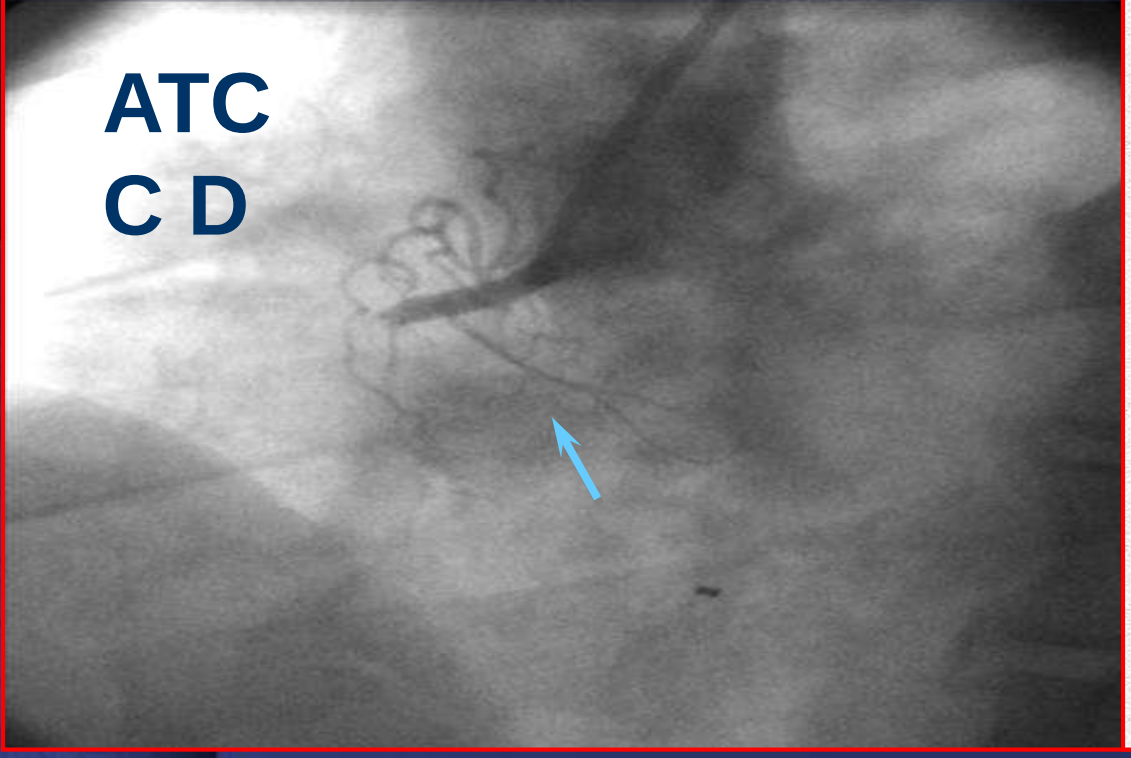
Composition of Coronary Thrombus in Acute Myocardial Infarction J Am Coll Cardiol 2011;57:1359–67

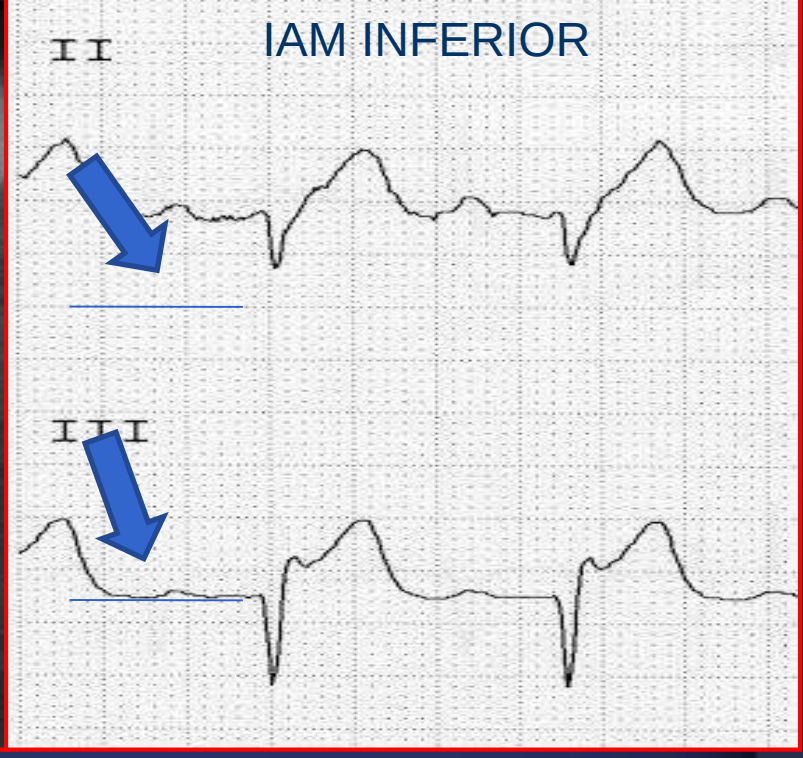


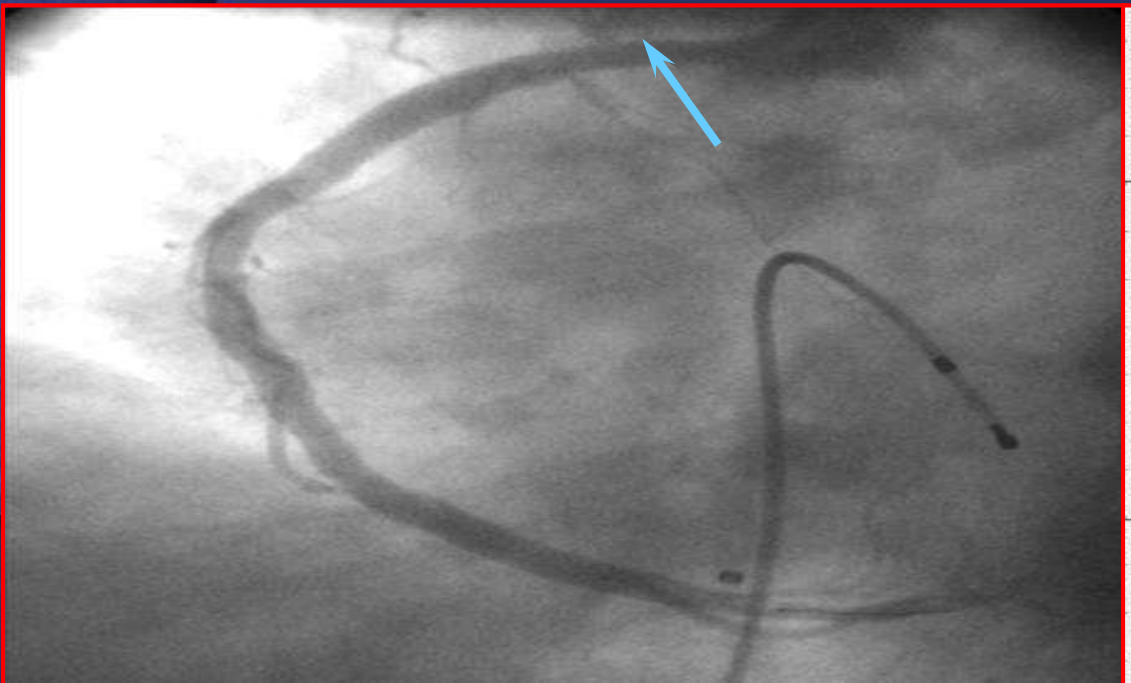


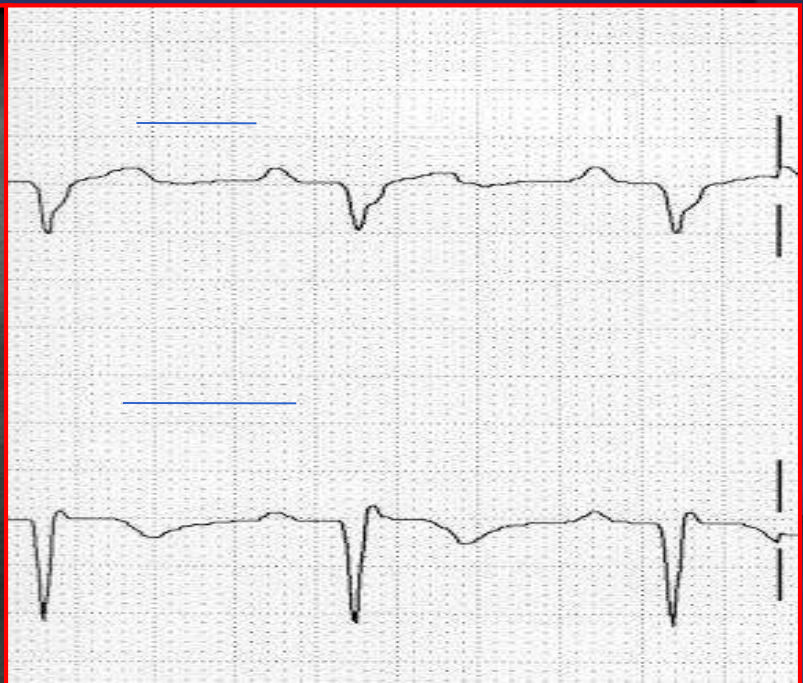
ESTRATEGIA DE REPERFUSION



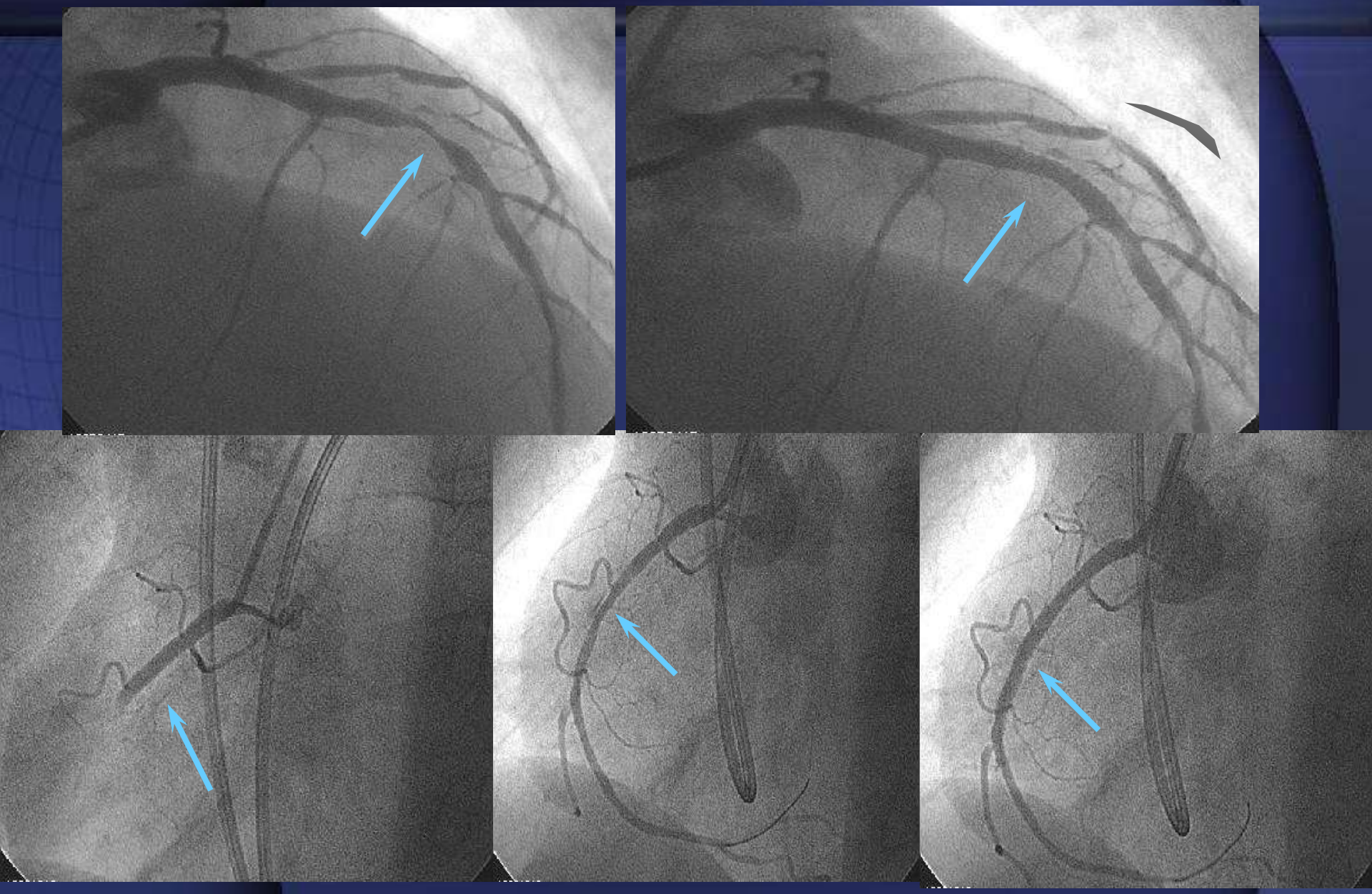

ATC
C D


II
IAM INFERIOR
III

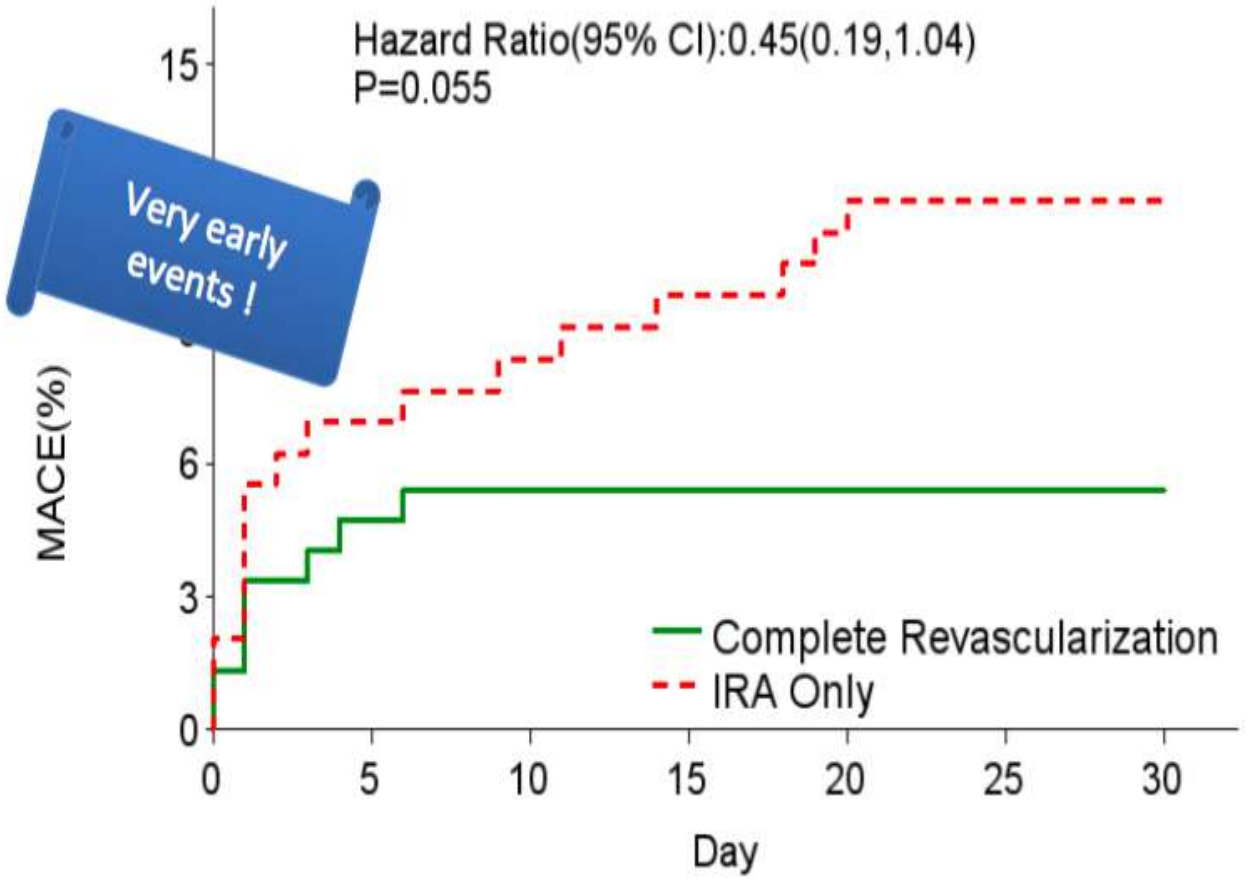




ENFERMEDAD de MULTIPLES VASOS



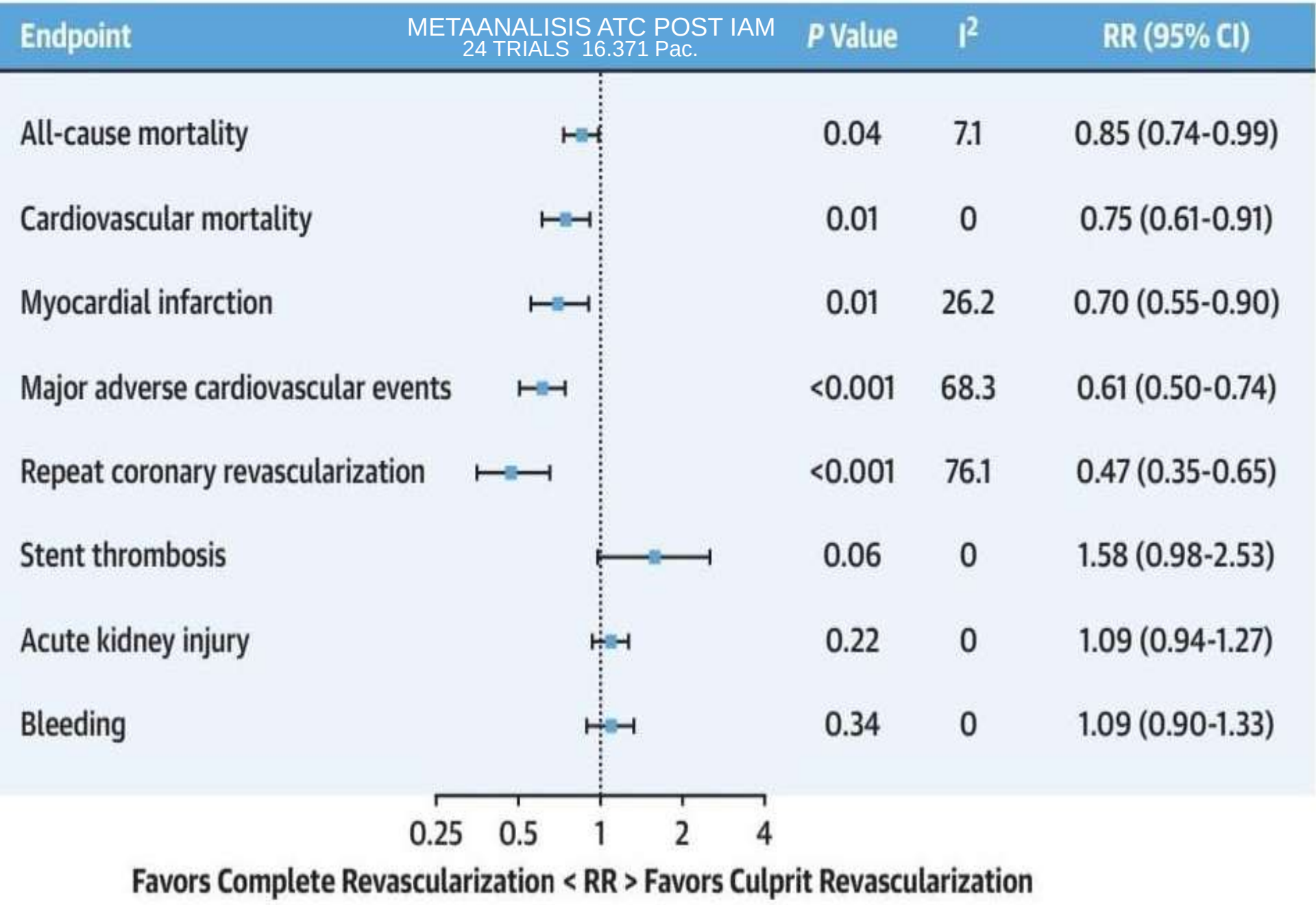
MACE to 30 days



Number at risk:						
Complete:150	136	133	133	133	133	133
IRA Only:146	131	128	126	124	123	123

J Am Coll Cardiol. 2015 Mar 17;65:963-72.

METAANALISIS ATC POST IAM
24 TRIALS 16.371 Pac.



y RK, et al. J Am Coll Cardiol. 2024;84(3):276-294.

MULTIPLES VASOS

SAC 2025

Recomendación	Clase	Nivel de Evidencia
Se recomienda la revascularización completa luego de un IAMCEST en pacientes hemodinámicamente estables.	I	A
El momento de tratar las lesiones no culpables puede ser en el mismo procedimiento o diferido (durante la internación o hasta 45 días), sin diferencias en mortalidad o reinfarto; decidir según estabilidad, anatomía, función renal y carga de contraste.	IIa	B

Guías ACC/AHA

ATC post Trombolíticos

(Farmacoinvasiva)

Clase I
A

- En pacientes con anatomía favorable

  - Evidencia de Lesion severa post TL

Isquemia miocárdica espontánea o provocada durante la evolución de un IAM.

ATC de RESCATE

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Fibrinolytic therapy *(continued)*

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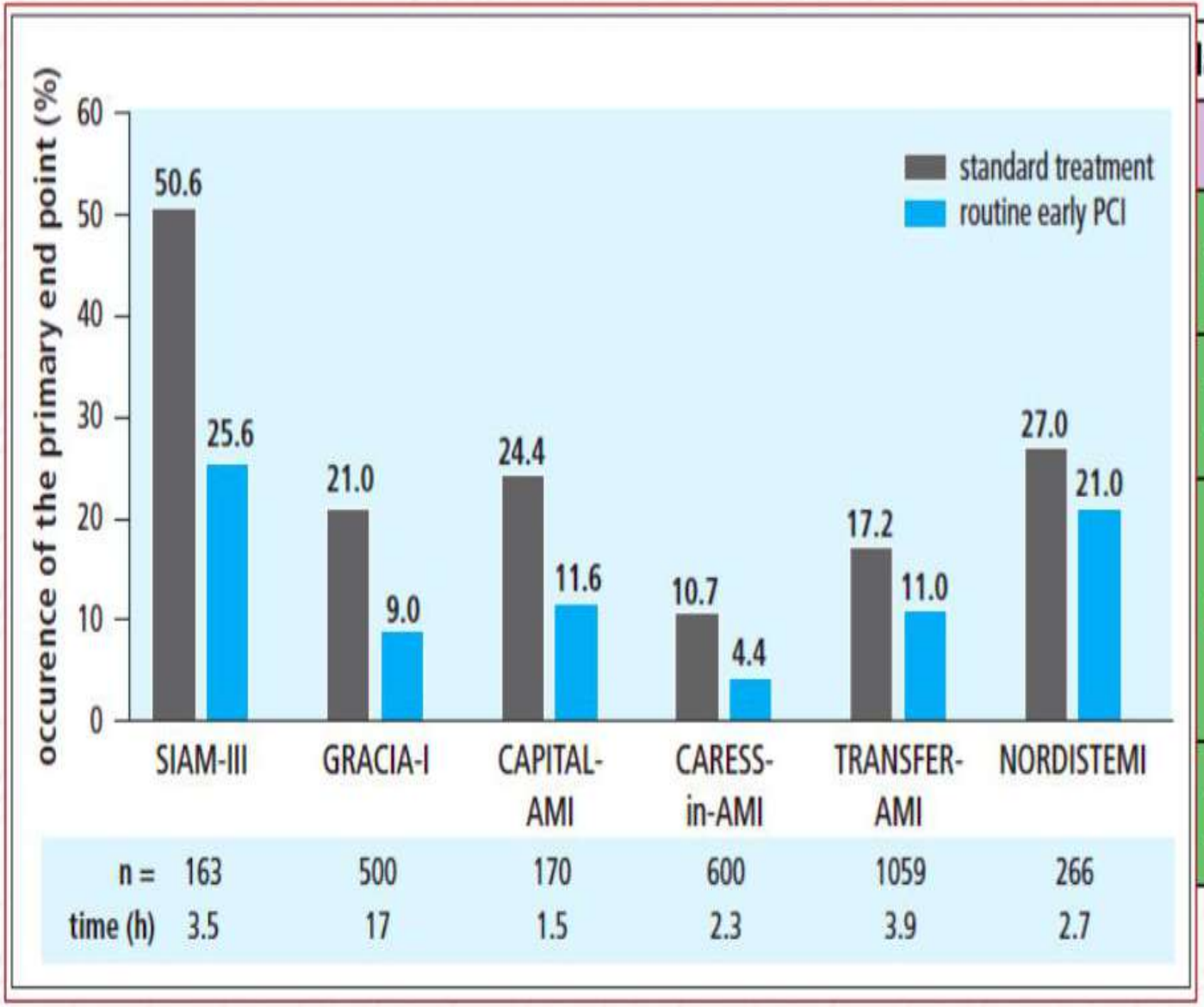


lass	Level
I	A
I	A
I	A
I	A

From 3-24 h to 2-24 h

INFARTO AGUDO DE MIOCARDIO

Estrategia Fármaco Invasiva

Combinación de los trombolíticos seguida por CCG de rutina y ATC en las lesiones severas, entre las 2 y las 24 hs., en pacientes con trombolísis exitosa.

CLASE I A

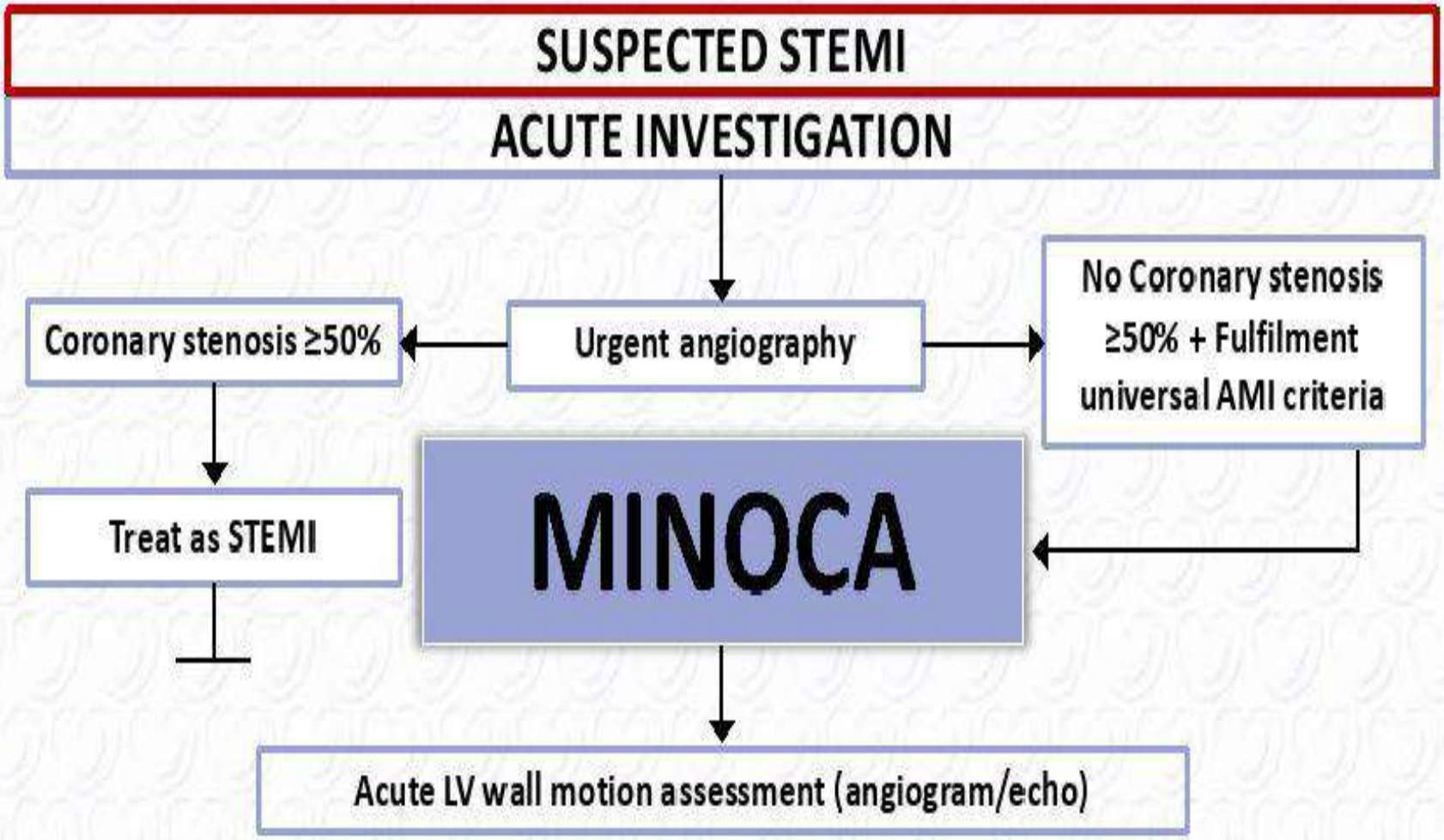
Estrategia Fármaco Invasiva

Conclusiones

Comparada con Angioplastia Primaria: IGUAL EFICACIA

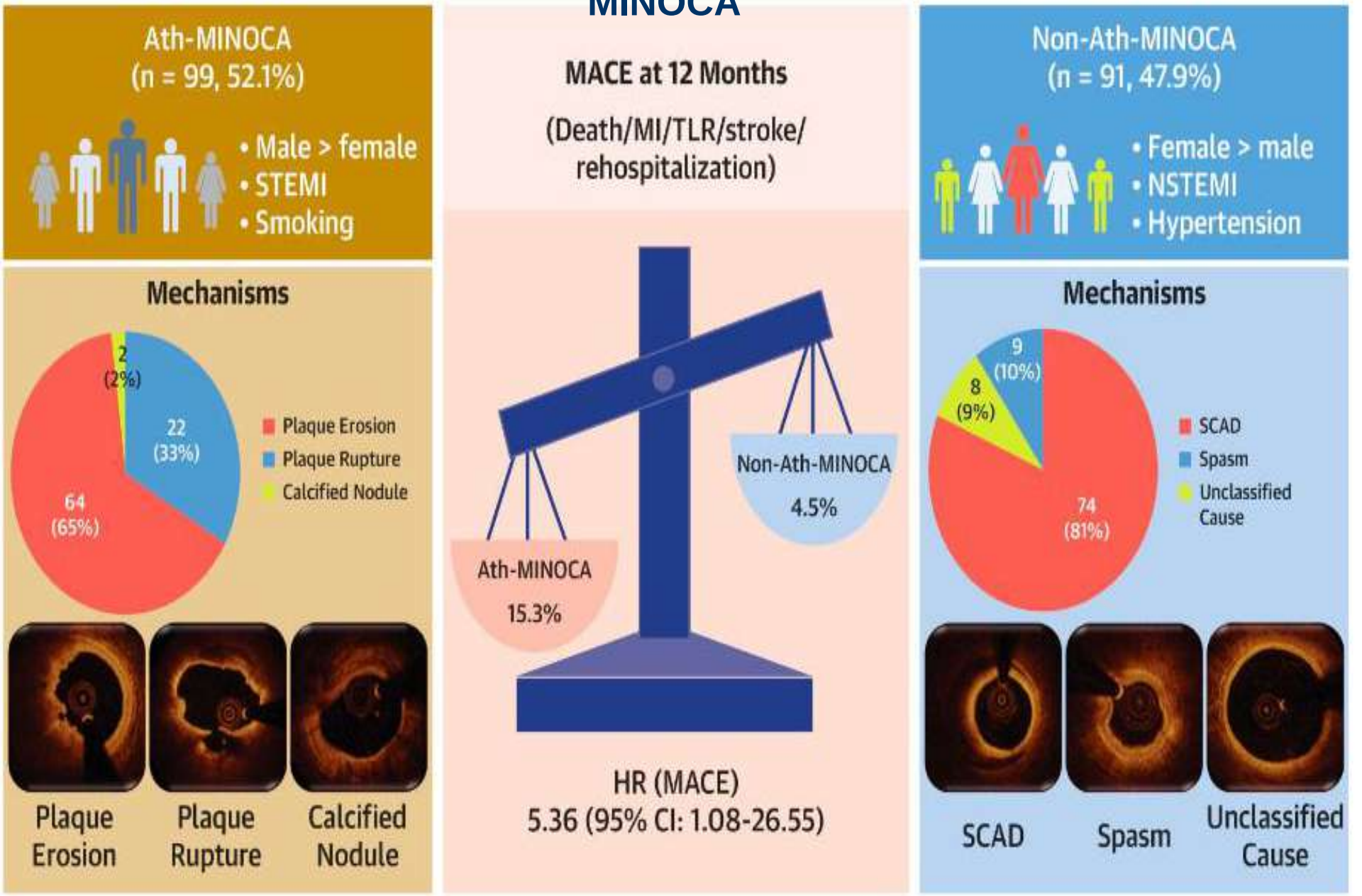
Diagnostic test flow chart in MINOCA

ESC
European Society of Cardiology

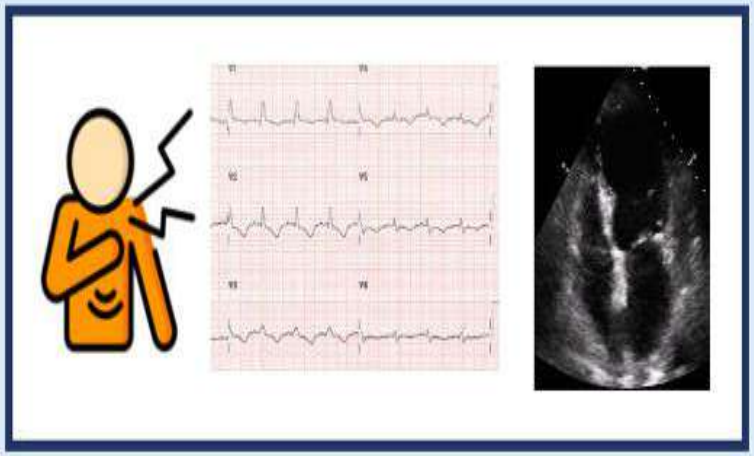



MINOCA

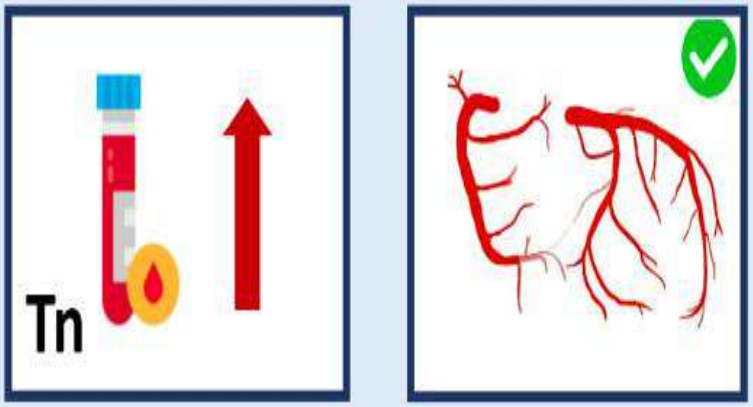
MINOCA



Zeng M, et al. J Am Coll Cardiol Img. 2023;16(4):521-532.



+ IAM 10%



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MINOCA

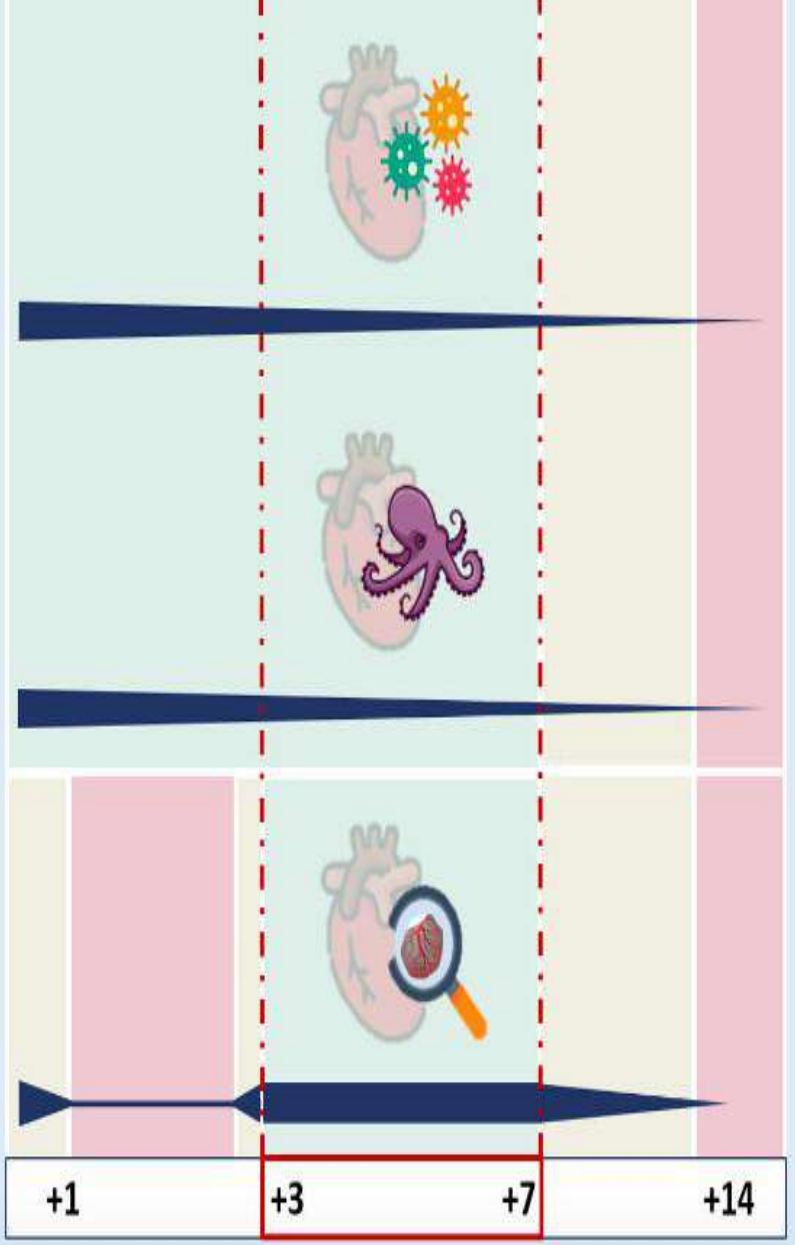


Figura 1 En la parte izquierda de la figura se resumen los criterios para el diagnóstico inicial de infarto de miocardio con enfermedad coronaria no obstructiva (MINOCA): a) síntomas de isquemia miocárdica, alteraciones electrocardiográficas compatibles con isquemia y/o alteraciones en la contractilidad segmentaria en el ecocardiograma; b) aumento y/o disminución del valor de troponina con al

Realizar IVUS u OCT en pacientes con sospecha de MINOCA en el cateterismo diagnóstico inicial para identificar rotura, erosión de placa o trombosis.(338,339)(centro con disponibilidad).	IIa	B
Realizar IVUS u OCT para identificar rotura o erosión de placa en pacientes en los que la RMC sugiere infarto por compromiso de vaso epicárdico. (338-340)	IIa	B
Por su mayor definición, la OCT es preferible al IVUS en la caracterización del endotelio coronario. (341)	IIb	B
Realizar prueba de vasorreactividad intracoronaria en pacientes con sospecha de vasoespasmo coronario, descartando otras causas de MINOCA (DCE, trombosis o embolias) luego de 48 h	IIa	B

PREVENCION SECUNDARIA

FACTORES DE RIESGO MODIFICABLES

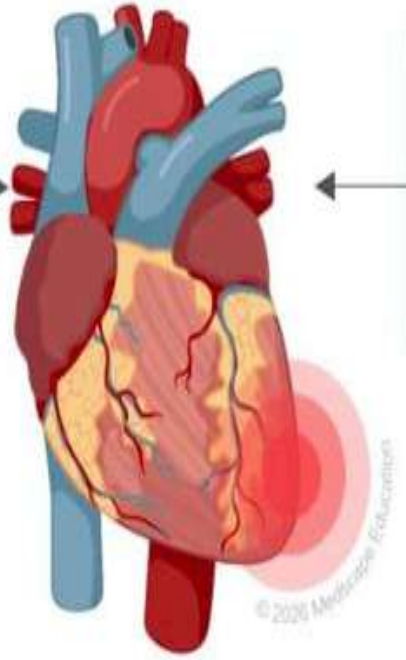
- Evitar el tabaco
- Colesterol total <200 mg/ dl **(LDL <60)**
- Presion arterial <120/80 mmHg
- Glucosa serica <100 mg/dl
- IMC < 25 kg/m2
- Dieta baja en grasas saturadas y carbohidratos refinados
- Actividad fisica regular
- Dormir adecuadamente

Menos del 5% de la población mantiene una salud cardiovascular ideal

Recommended Medical Therapies for ACS

Lipid-lowering therapy
- High-intensity statin[a]
- + ezetimibe and/or PCSK9i
- Icosapent ethyl (↑ TGs)

Antithrombotics
- Aspirin[a]
- P2Y12 inhibitors[a]
- Oral anticoagulants (AF, LV thrombus)



Patients with LV dysfunction
- Beta blocker[b]
- ACEI/ARBs (or with diabetes)
- MRA

[a]All patients. [b]Or significant residual coronary artery disease with angina.

ROUTINE MAINTENANCE THERAPIES:

ESC
European Society of Cardiology

Antiplatelet therapy

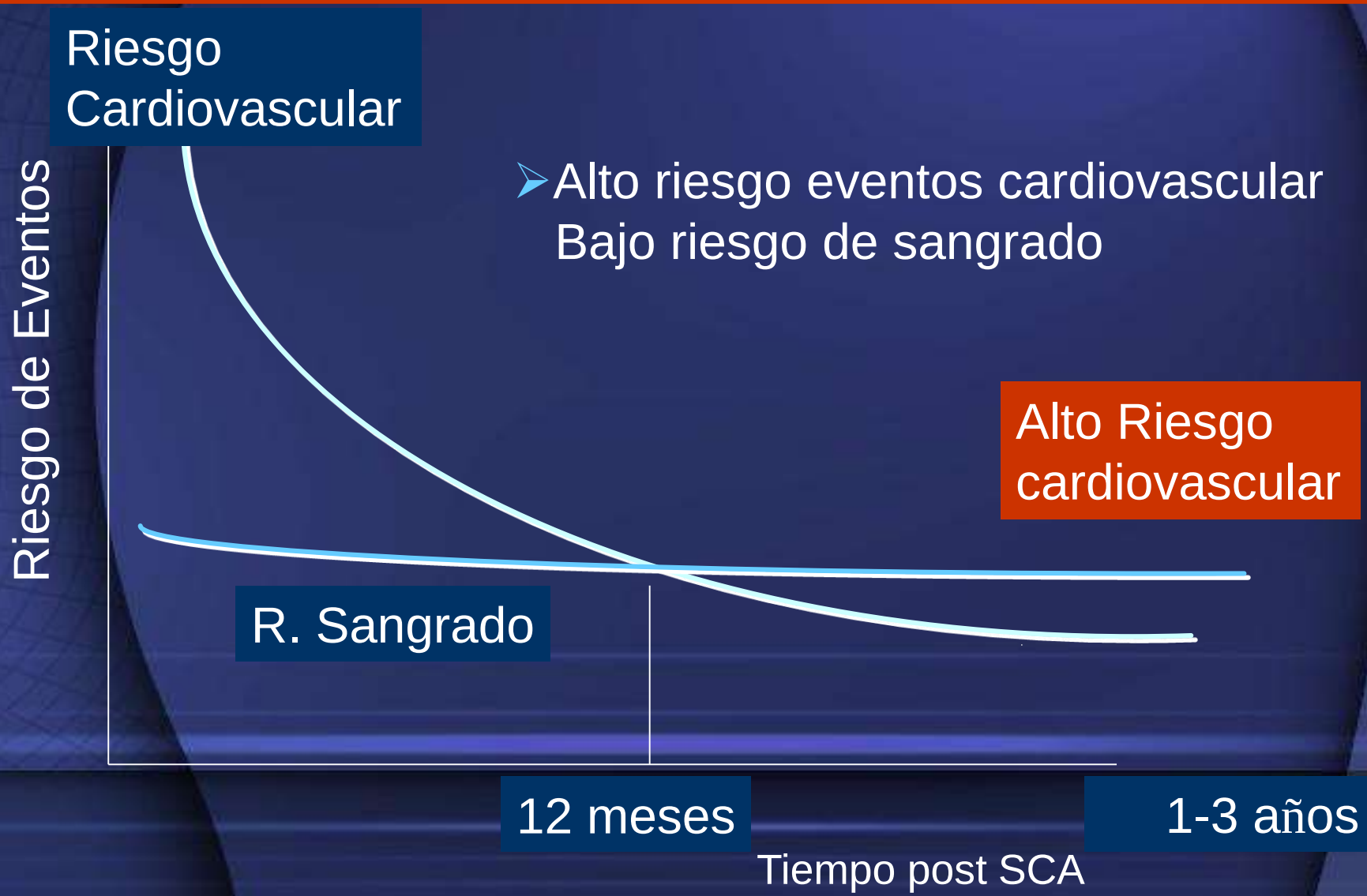
Lipid lowering therapy

Beta-blockers

ACE inhibitors/ARBs

MRA

Toma de Decisiones de acuerdo a Riesgos despues de un SCA

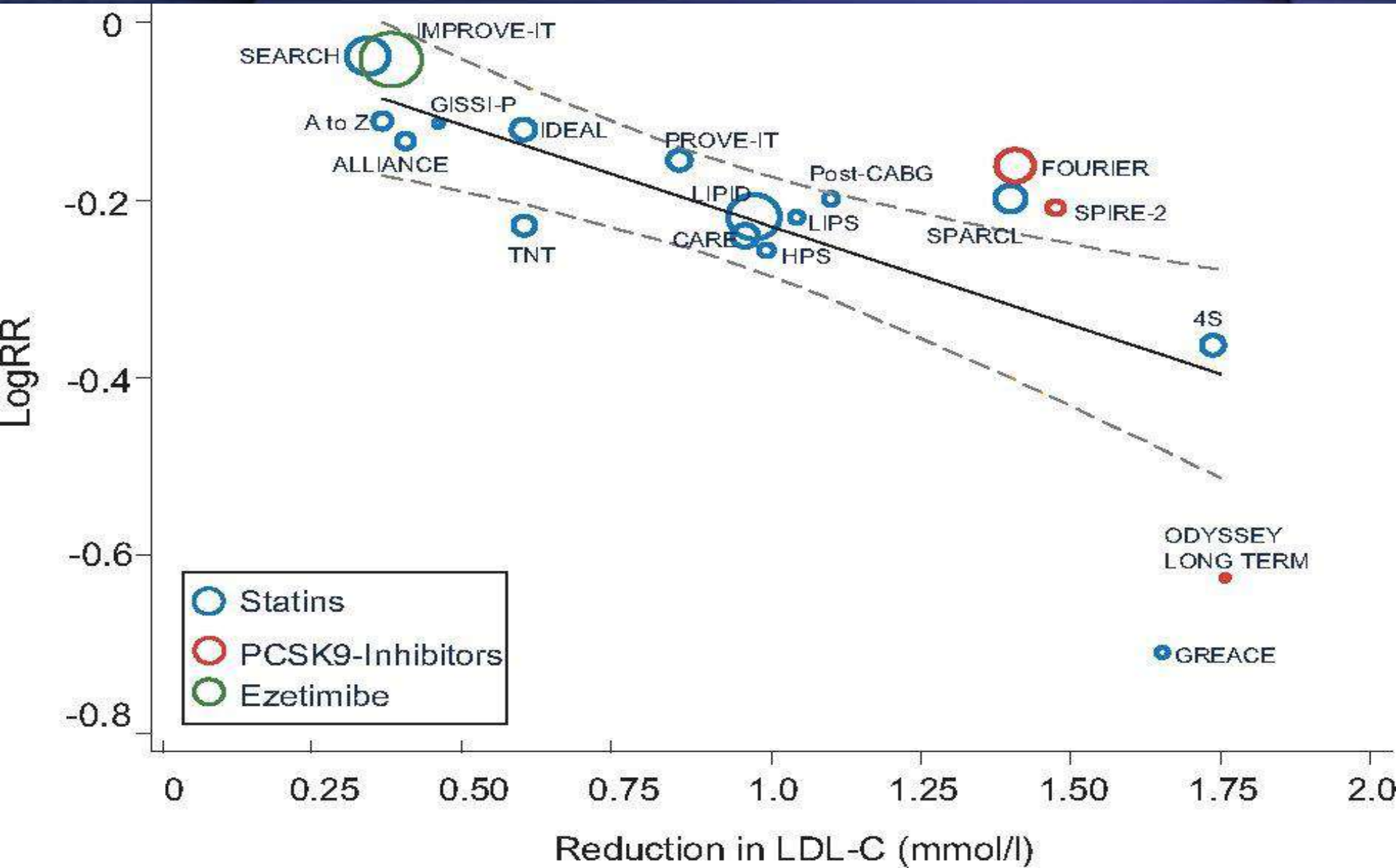


Antiplatelet and Anticoagulant Therapy for ACS Guideline Comparisons (cont)

		ACS: Antiplatelet and Anticoagulant Therapy	ACC/AHA 2025	ACC/AHA 2013-2014	ESC 2023	Evidence Summary
After PCI	Antiplatelet	DAPT with aspirin and $P2Y_{12}$ inhibitors for 12 months in patients without high bleeding risk	✓	✓	✓	Supported by the CLARITY-TIMI 28, COMMIT, and CURE trials
		Preference of ticagrelor and prasugrel over clopidogrel in patients proceeding to PCI	✓¶	?#	✓¶	TRITON-TIMI 38 & PLATO trials: ↓ MACE but ↑ major bleeding with prasugrel or ticagrelor vs clopidogrel
		Preference of prasugrel over ticagrelor in patients who proceed to PCI	?	?	✓	ISAR-REACT-5 trial: ↓ MACE without increased major bleeding with prasugrel vs ticagrelor
		Clopidogrel when prasugrel/ticagrelor are unavailable, cannot be tolerated, or are contraindicated	✓	?	✓‖	Supported by the CLARITY-TIMI 28, COMMIT, and CURE trials
		De-escalation from DAPT to SAPT with ticagrelor after 1 month in patients at high bleeding risk post PCI	✓	?	✓**	TWILIGHT-ACS, ULTIMATE-DAPT, T-PASS, TICO trials: ↓ bleeding without excess in MACE with ticagrelor alone (vs continued DAPT) after 1-3 months

✓ Recommended as Class 1 ✓ Suggested as Class 2a ✓ Considered as Class 2b ● Not Recommended (Class 3) ? Not Addressed

Prevencion 2ria Niveles LDL



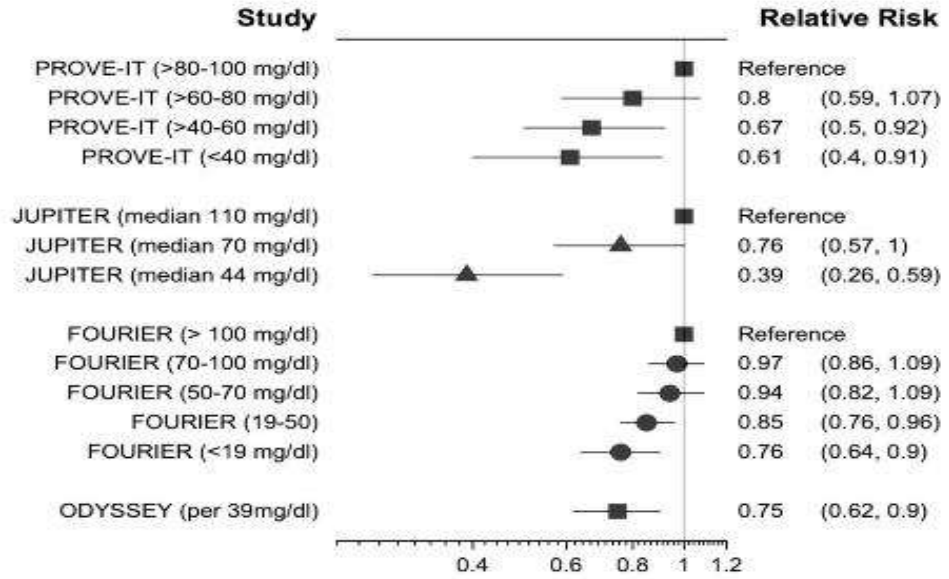
Endothelial function

Oxidative stress

Inflammation

LDL-C reduction

The ealier the better, the lower the better for longer



- Stroke/TIA
- Myocardial infraction
- Mesenterie ischemia
- Lower extremity
- Amputation
- Erectile dysunction
- Ischemic cardiomyopathy
- Renal ischemia

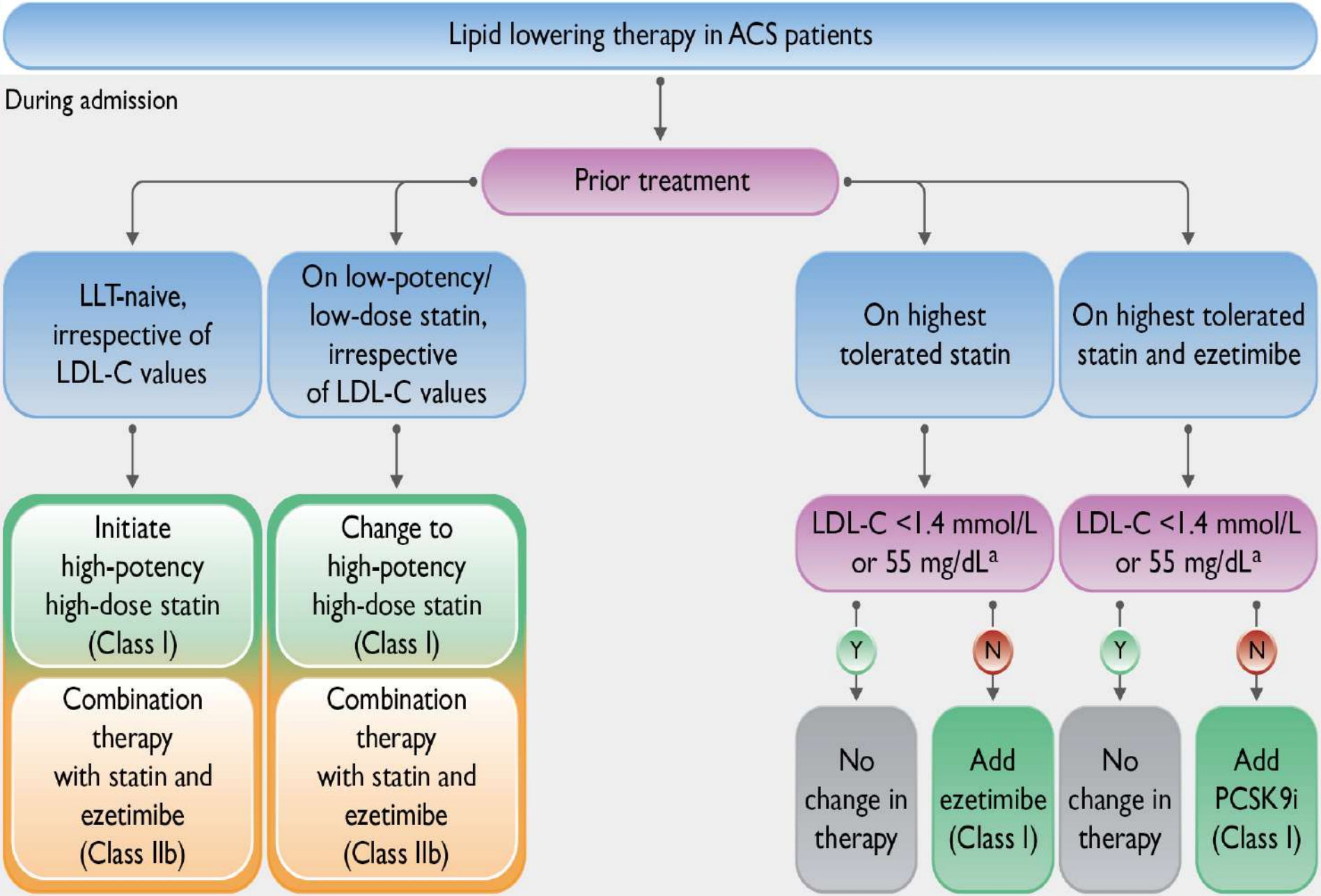
- Neurodegenerative disorders
- Vascular dementia
- Alzheimer's disease
- Parkinson's disease

ATHEROSCLEROSIS
(coronary artery diseases; peripheral vascular disease; renal artery discase; mesenteric artery disease; cerebrovascular disease)

Aortic aneurysms, aortic valve sclerosis

HIPOLIPEMIANTES 2026

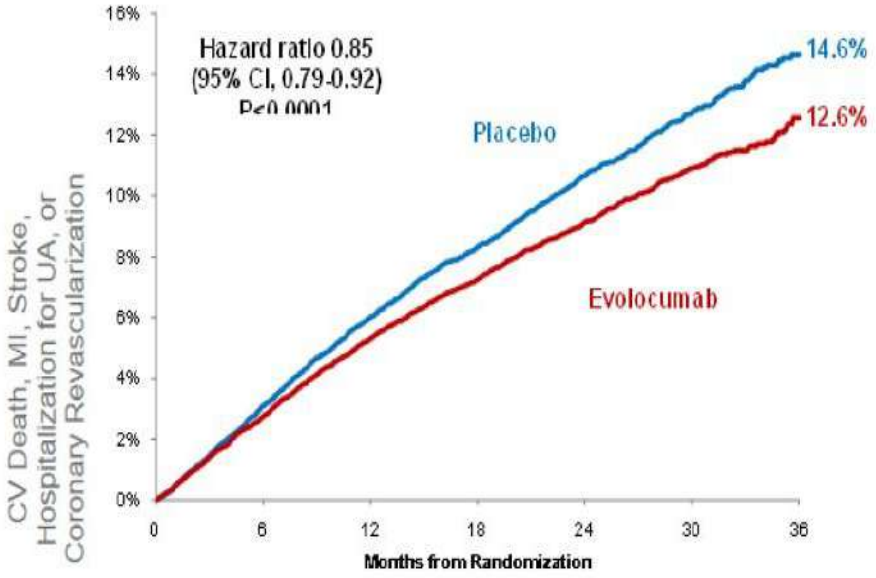
	Small molecules	Monoclonal antibody	Antisense Therapy		Macrocyclic Peptides
			ASO	siRNA	
Example	Statins	Evo/Alirocumab	Pelacarsen	Inclisiran	MK-0616
Structure	Organic compound	Protein (mAb)	RNA **simple** chain	RNA **double** chain	Protein (Macrocytic Peptide
Mechanism	Block enzyme	Block protein	Block RNAm translation		Block interaction protein-protein
"Off target" AE	Yes	No			
Immunogenicity	No	Low			
Eficacy	50% LDLc	60% LDLc	90% Lp(a)	50% LDLc	60% LDLc
Response variability	High	Low			Medium
Administration	Oral	Subcutaneous			Oral
Frequency	Daily	Every 2 weeks	Every 4 weeks	Every 6 months	Daily


Lipid lowering therapy in ACS patients
During admission
Prior treatment
LLT-naive, irrespective of LDL-C values
On low-potency/ low-dose statin, irrespective of LDL-C values
On highest tolerated statin
On highest tolerated statin and ezetimibe
Initiate high-potency high-dose statin (Class I)
Combination therapy with statin and ezetimibe (Class IIb)
Change to high-potency high-dose statin (Class I)
Combination therapy with statin and ezetimibe (Class IIb)
LDL-C <1.4 mmol/L or 55 mg/dL[a]
LDL-C <1.4 mmol/L or 55 mg/dL[a]
Y
N
Y
N
No change in therapy
Add ezetimibe (Class I)
No change in therapy
Add PCSK9i (Class I)

FOURIER and ODYSSEY Outcomes
CV Risk Reduction With PCSK9 Inhibitors

FOURIER: Benefit of evolocumab in patients with stable ASCVD + moderate- or high-intensity statin (N = 27,564)[a]

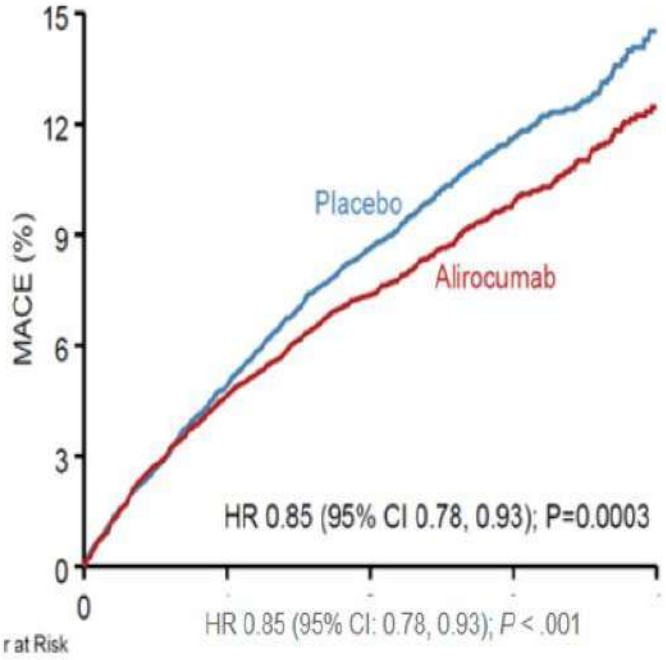
Primary endpoints: CV death, MI, stroke, coronary revascularization, or hospital admission for UA



Limitation: Short duration of follow-up (median 2.2 y); infrequent use of ezetimibe for CV efficacy

ODYSSEY OUTCOMES: Benefit of alirocumab in patients with recent ACS + maximal statin therapy (N = 18,924)[b]

Primary endpoint: MACE



Limitation: Infrequent use of ezetimibe for CV efficacy

a. Sabatine MS, et al; for the FOURIER Steering Committee and Investigators. N Engl J Med. 2017;376:1713-1722; b. Schwartz GG, et al; for the ODYSSEY OUTCOMES Committees and Investigators. N Engl J Med. 2018;379:2097-2107.

Comprehensive Review of Statin-intolerance and the practical application of Bempedoic Acid

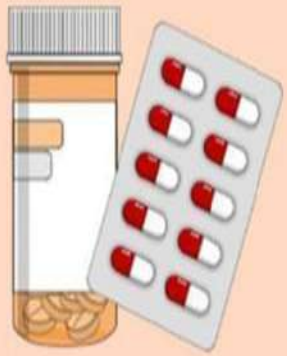
Bempedoic acid, an ATP citrate lyase inhibitor plays a crucial role in lowering LDL-c levels.

Bempedoic Acid significantly reduced the risk of major cardiovascular events.

Bempedoic acid should be explored as an additional therapy to achieve required LDL-C levels in statin-intolerant patients.

Heart disease is leading casuse of death in the United States

- Elevated LDL-C have been consistently associated with increased risk of CVD
- Statins are most commonly prescribed medication for LDL-C reduction and have shown significant efficacy in reducing LDL-C Levels
- 7-29% patients reported adverse effects due to statins



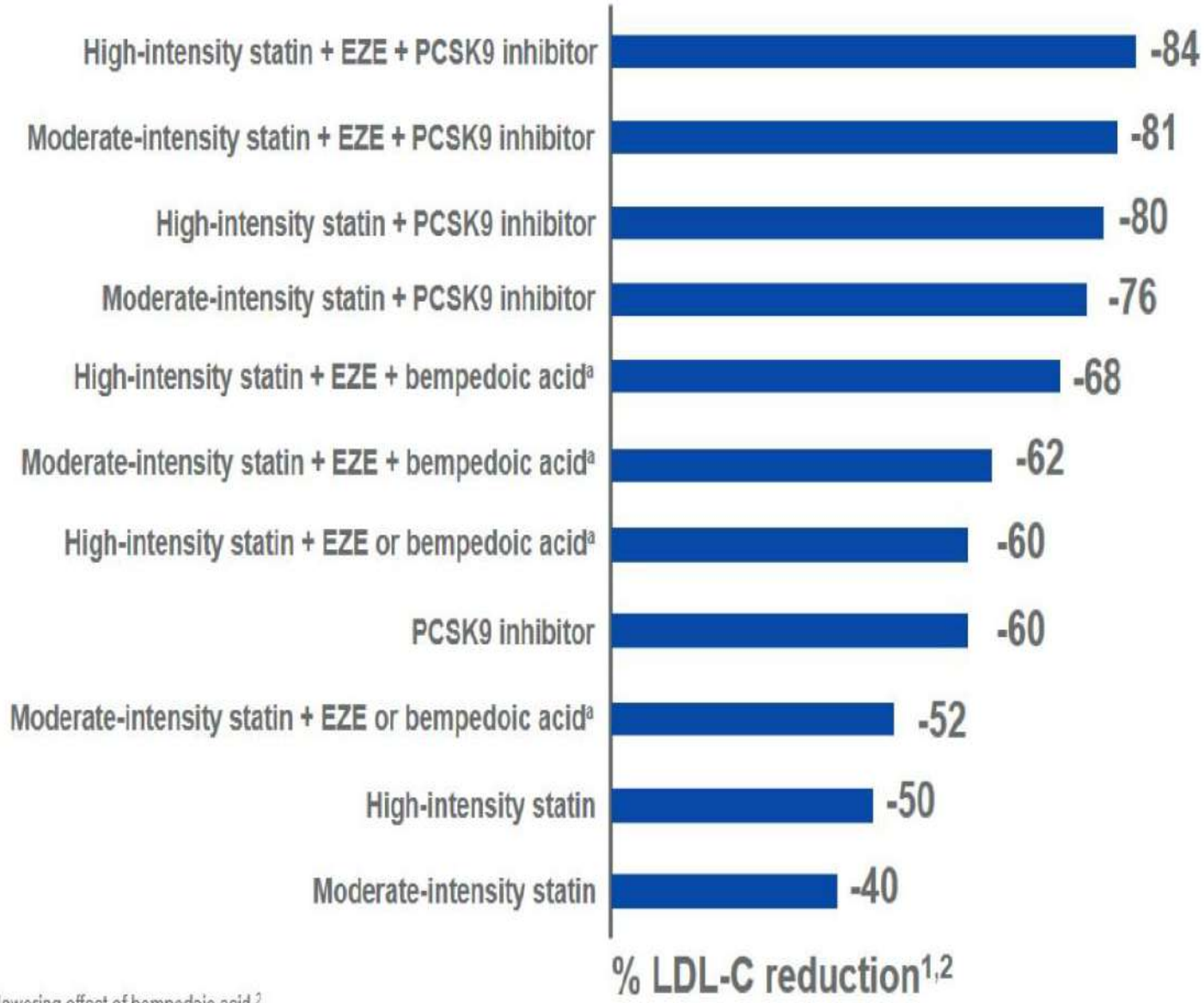
FDA approved Bempedoic Acid (BA), reduces hepatic cholesterol, raises LDL receptor expression and increases LDL cholesterol clearance from circulation

- Bempedoic acid (ETC-1002), is an oral agent with:
- newer first-in-class mechanism inhibits adenosine triphosphate (ATP)-citrate lyase inhibitor leading to lower LDL-C
- Due to similarity in the mechanism of action, like statins, BA decreases hepatic generation of cholesterol, upregulates the LDL receptor expression on liver and clears the circulating LDL-C from the blood

Due to absence of ACSVL1 in skeletal muscle, bempedoic acid potentially avoids the most common side effect encountered with statin, which is myotoxicity.

- Bempedoic acid significantly lowered LDL-C in 4 phase 3 placebo RCTs.
- Bempedoic acid is associated with increase in uric acid and of Gout.
- New-onset diabetes was lower with bemepdoic acid than placebo

Combination Therapy to Reach Guideline-Recommended Goals



[a]Based on estimation of 20% lowering effect of bempedoic acid.[2]

1. Masana L, et al. Curr Cardiol Rep. 2020;320:122-128; 2. Nissen SE, et al. N Engl J Med. 2023;388:1353-1364.

2024 ESC Guideline Recommendations for Lipid-Lowering Drugs in Patients With CCS

2024 ESC Guideline Recommendations for Lipid-Lowering Drugs in Patients With CCS		
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	Class I	Level A
A **high-intensity statin** up to the highest tolerated dose to reach the LDL-C goals is recommended for **all patients with CCS**	Class I	Level A
If a patient's goal is not achieved with maximum tolerated dose of statin, **combination with ezetimibe** is recommended	Class I	Level B
For patients who are statin intolerant and do not achieve their goal on ezetimibe, **combination with bempedoic acid** is recommended	Class I	Level B
For patients who do not achieve their goal on a maximum tolerated dose of statin and ezetimibe, **combination with a PCSK9 inhibitor** is recommended	Class I	Level A
For patients who do not achieve their goal on a maximum tolerated dose of statin and ezetimibe, combination with bempedoic acid should be considered	Class IIa	Level C

Vrints C, et al. Eur Heart J. 2024. doi: 10.1093/eurheartj/ehae177. [Epub ahead of print].

Recomendaciones **DISFUNCION DE VI POST IAM SAC 2025**	Clase	Nivel de evidencia
En pacientes con fracción de eyección ≤ 40%, insuficiencia cardíaca, hipertensión, diabetes mellitus se recomienda el uso de IECA o BRA (129,130,134,135) **B. Bloqueantes**	I	A
En pacientes con síndromes coronarios agudos y una fracción de eyección ≤ 40% con síntomas de insuficiencia cardíaca y/o diabetes mellitus, se recomiendan los antagonistas de la aldosterona (138,139)	I	A

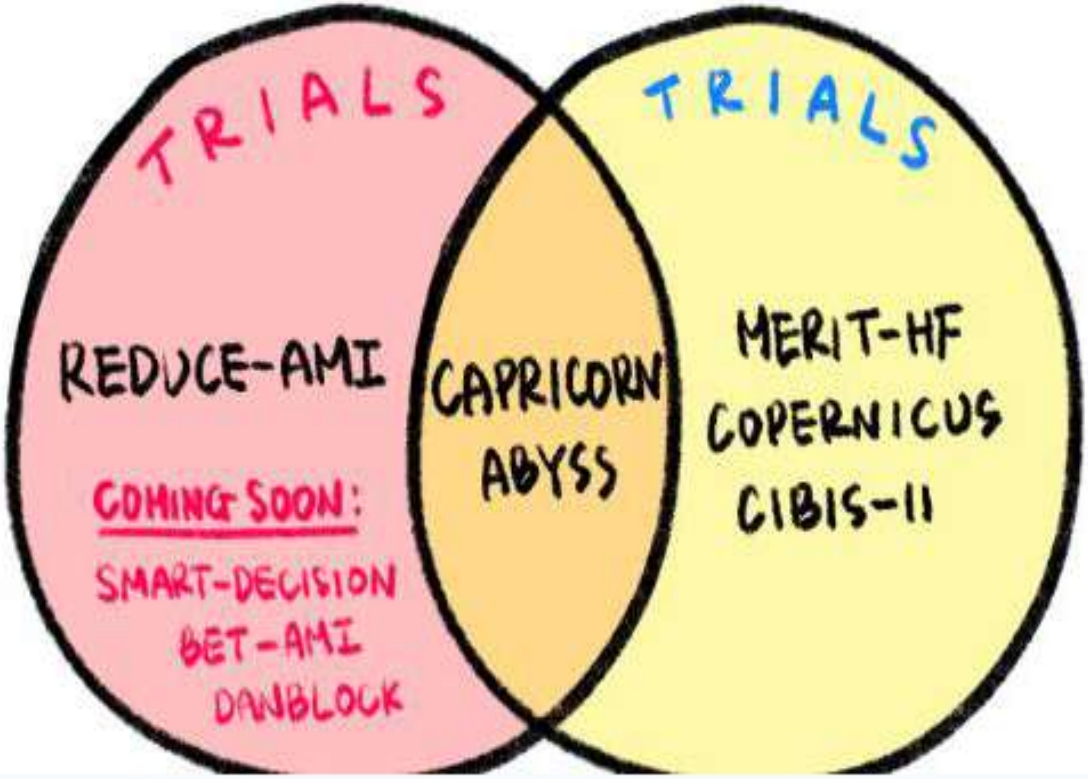
CORE IM

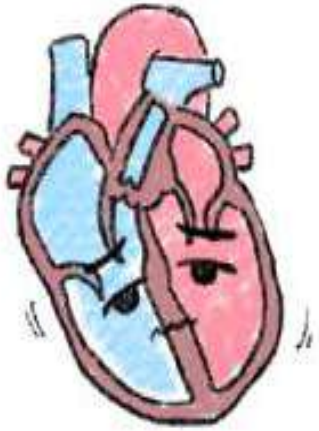
DOES THIS PATIENT NEED BETA BLOCKERS?

@JIMINHWANGMD

POST-ACUTE CORONARY SYNDROME

HEART FAILURE WITH REDUCED EJECTION FRACTION

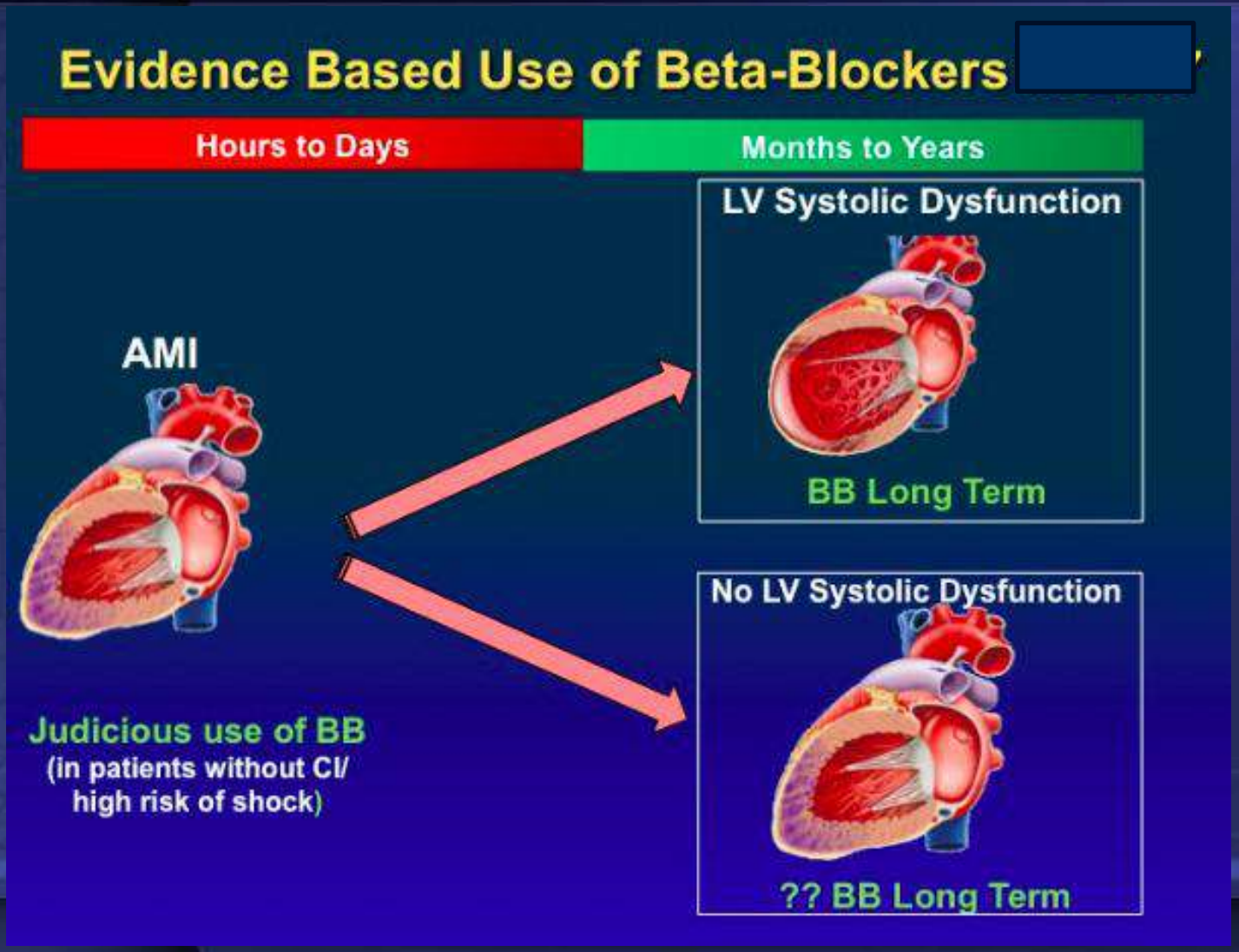




<40%

Beneficio de la adherencia a largo plazo

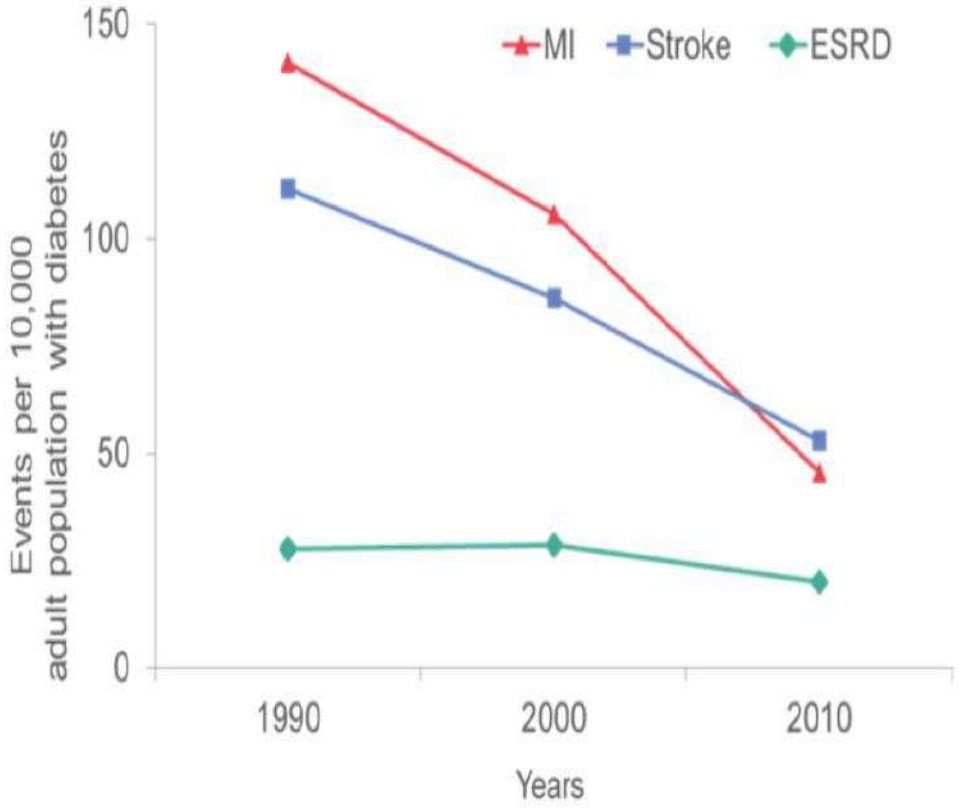
Betabloqueantes



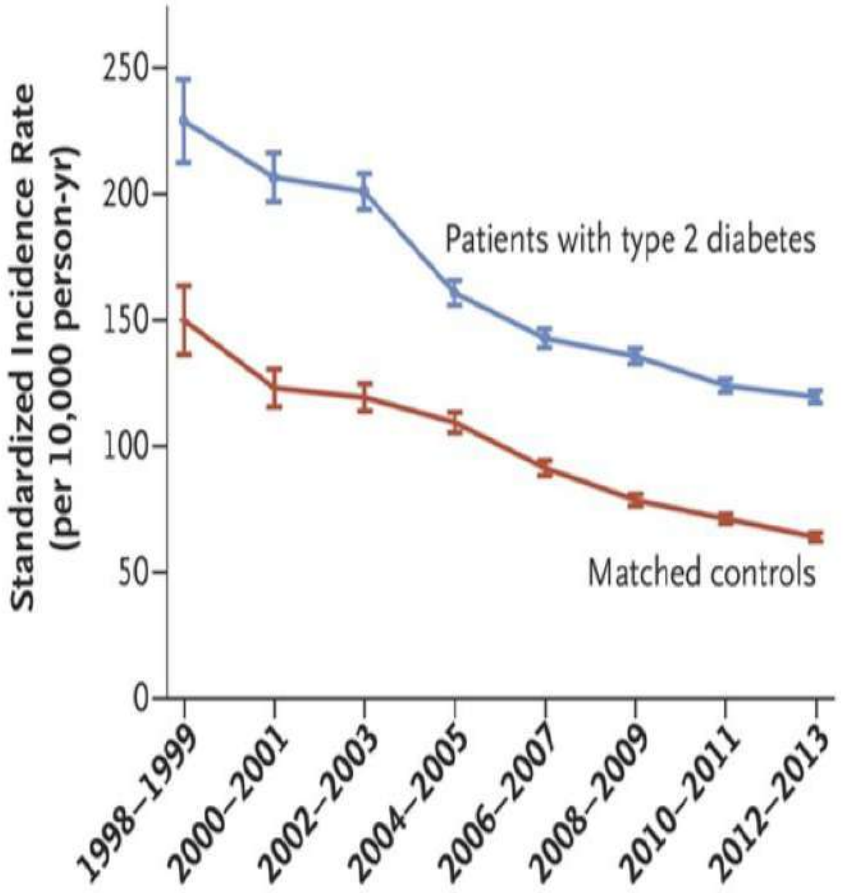
Fey < 40% -- Isquemia Residual

CV Risk in Type 2 Diabetes

Reduction of MI and Stroke in Diabetes[1]



CV Death in Diabetes[2]



ESRD, end-stage renal disease; MI, myocardial infarction.

1. Adapted from Gregg, et al. N Engl J Med. 2014;370:1514-1523.; 2. Rawshani A, et al. N Engl J Med. 2017;376:1407-1418.

Glucose-Lowering Medications for Patients With Type 2 Diabetes to Reduce CV Risk

Risk assessment for patients with Type 2 Diabetes based on the presence of ASCVD/Severe TOD and 10-year CVD risk estimation via SCORE2-Diabetes

Metformin (Class IIa)		Metformin SGLT2 inhibitor GLP-1 RA (Class IIb)	GLP-1 RA[a] SGLT2 inhibitor[b] (Class 1)
Low risk	Moderate risk	High risk	Very high risk
No ASCVD or severe TOD SCORE2-Diabetes < 10%		No ASCVD or severe TOD SCORE2-Diabetes ≥ 10%	ASCVD

GLP-1 RA, glucagon-like peptide-1 receptor agonist; SGLT2, sodium-glucose cotransporter 2.

a. GLP-1 RAs with proven CV benefit: liraglutide, semaglutide (subcutaneous), dulaglutide, efpeglenatide; b. SGLT2 inhibitors with proven CV benefit: empagliflozin, canagliflozin, dapagliflozin

GLIFLOZINS: MECHANISTIC INSIGHTS

Pleiotropic effects

Systemic levels

- ↓ Inflammation
- ↓ Oxidative stress
- ↑ Autophagy
- ↑ Mitochondrial function
- ↓ Insulin resistance
- ↓ NHE activity
- ↓ Fibrosis
- ↓ Ferritin
- ↓ Hepcidin

Vasculature

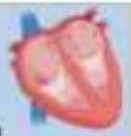
- ↓ Endothelial dysfunction
- ↓ Arterial stiffness
- ↓ Blood pressure
- ↓ Atherogenesis

Liver

- ↑ SIRT1
- ↑ HIF-2α
- ↑ Erythropoietin

Heart

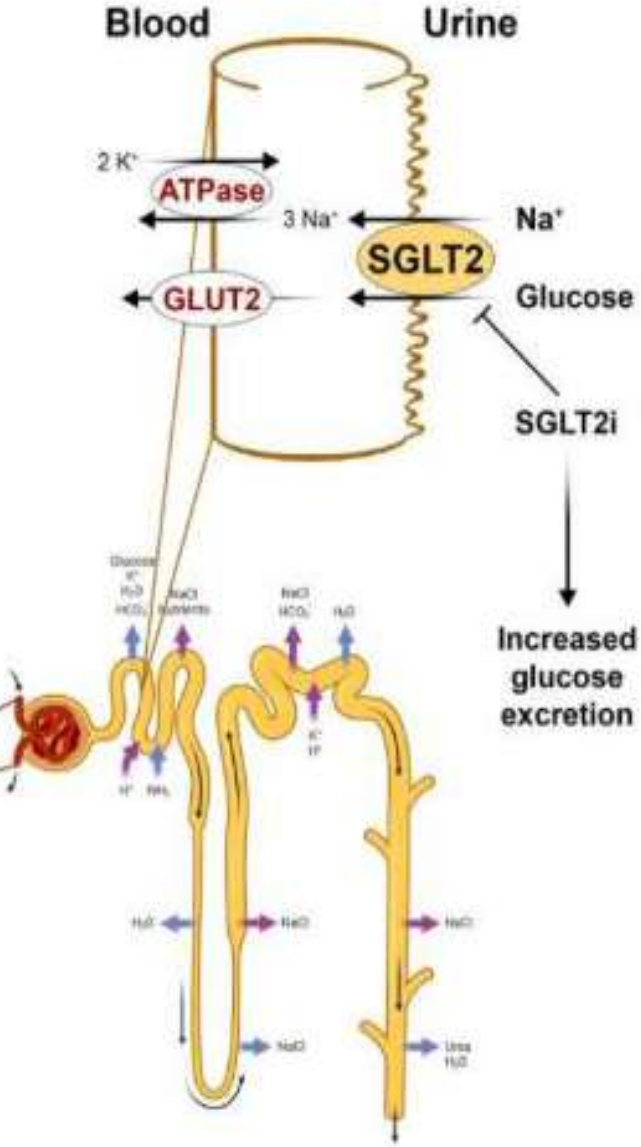
- ↑ Energetics
- ↓ Epicardial fat
- ↓ Pre- and afterload
- ↑ Ca^{2+} homeostasis
- ↑ Myocardial contractility
- ↑ Ketone body utilization
- ↑ Left-ventricular function



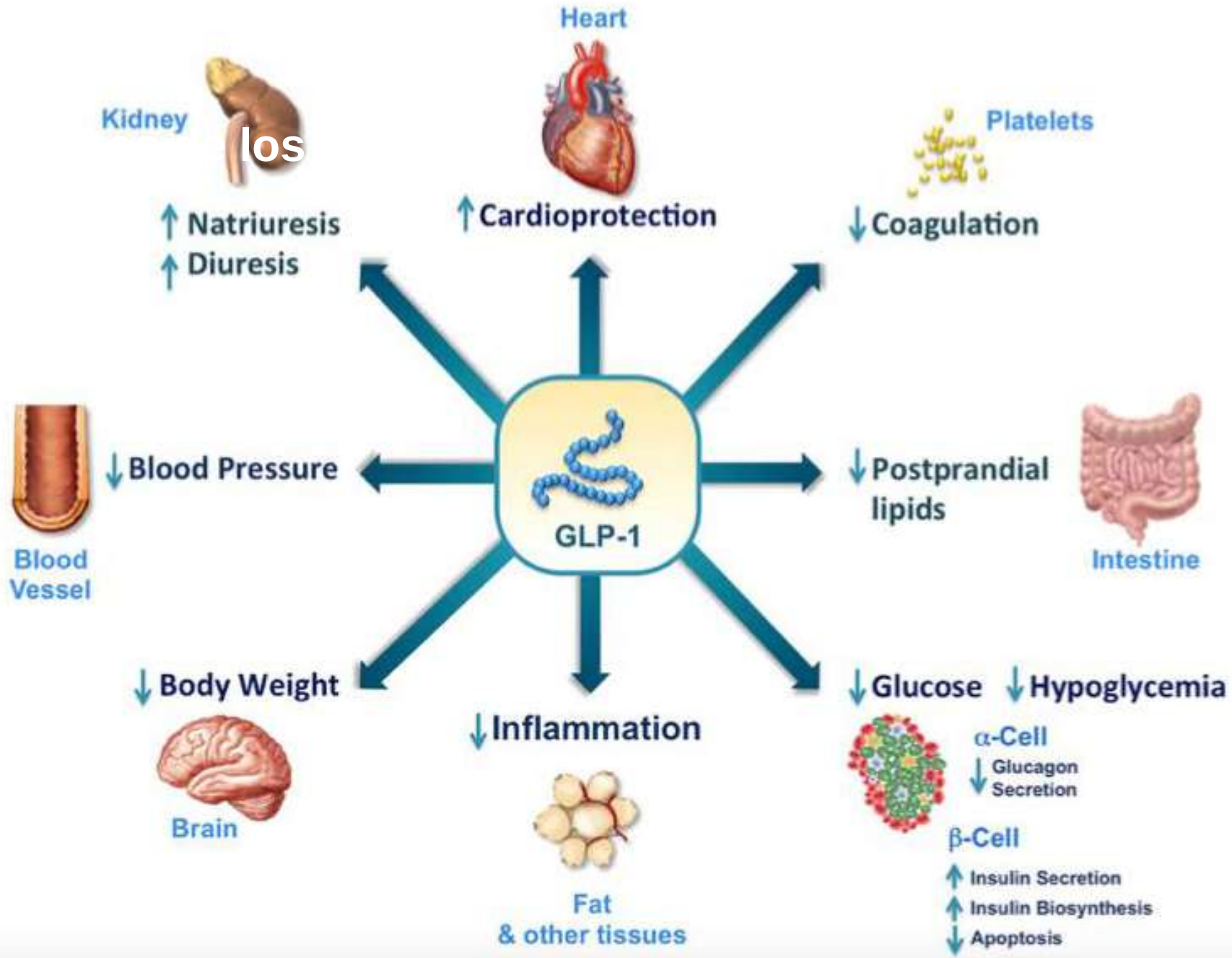
Kidney

- ↑ Glycosuria
- ↑ Natriuresis
- ↑ Diuresis
- ↓ Albuminuria
- ↓ Glomerular pressure
- ↑ Tubuloglomerular feedback
- ↓ Intrarenal RAAS activation

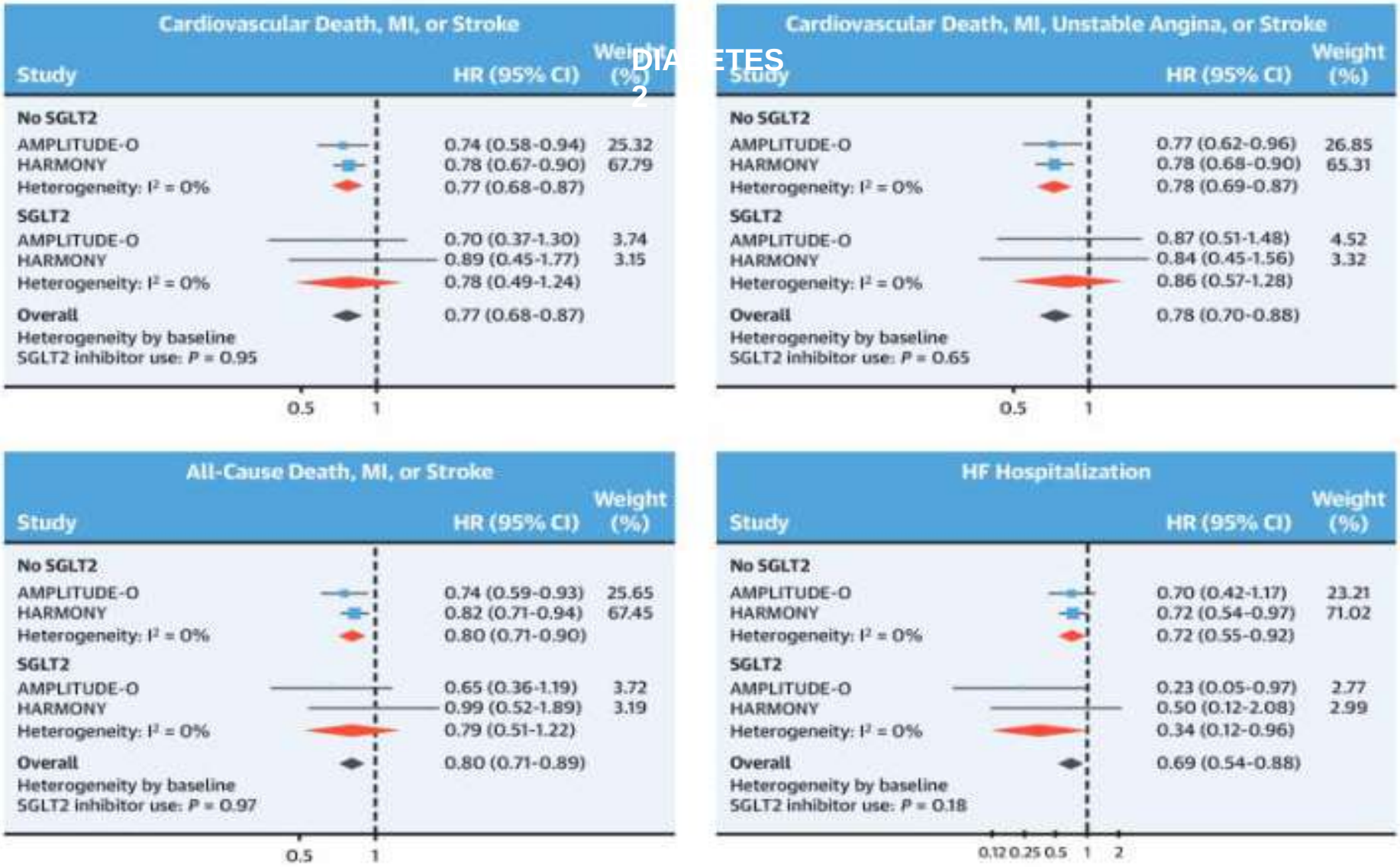
Glucose-lowering effects



Trials in Heart Failure and a Reduced Ejection Fraction (With or Without Diabetes)		
	DAPA-HF (dapagliflozin)	EMPEROR-Reduced (empagliflozin)
Cardiovascular death or hospitalization for heart failure	0.75 (0.65 – 0.85) [877 events]	0.75 (0.65 – 0.86) [823 events]
First hospitalization for heart failure	0.70 (0.59 – 0.83) [549 events]	0.69 (0.59 – 0.81) [588 events]
Renal composite endpoint	0.71 (0.44 – 1.16) [67 events]	0.50 (0.32 – 0.77) [88 events]
Cardiovascular death	0.82 (0.69 – 0.98) [500 events]	0.92 (0.75 – 1.12) [389 events]
Trials in Type 2 Diabetes (With or Without Heart Failure)		
GLIFOZINAS EFECTO CV	DECLARE-TIMI58 (dapagliflozin)	EMPA-REG OUTCOME (empagliflozin)
Cardiovascular death or hospitalization for heart failure	0.83 (0.73 – 0.95) [913 events]	0.66 (0.55 – 0.79) [463 events]
First hospitalization for heart failure	0.73 (0.61 – 0.88) [498 events]	0·65 (0·50 – 0·85) [221 events]
Renal composite endpoint	**0.53 (0·43 – 0·66)** [365 events]	**0·54 (0·40 – 0·75)** [152 events]
Cardiovascular death in patients with prior myocardial infarction	**0.92 (0.61 – 1.23)** [183 events]	**0.59 (0.44 – 0.79)** [183 events]

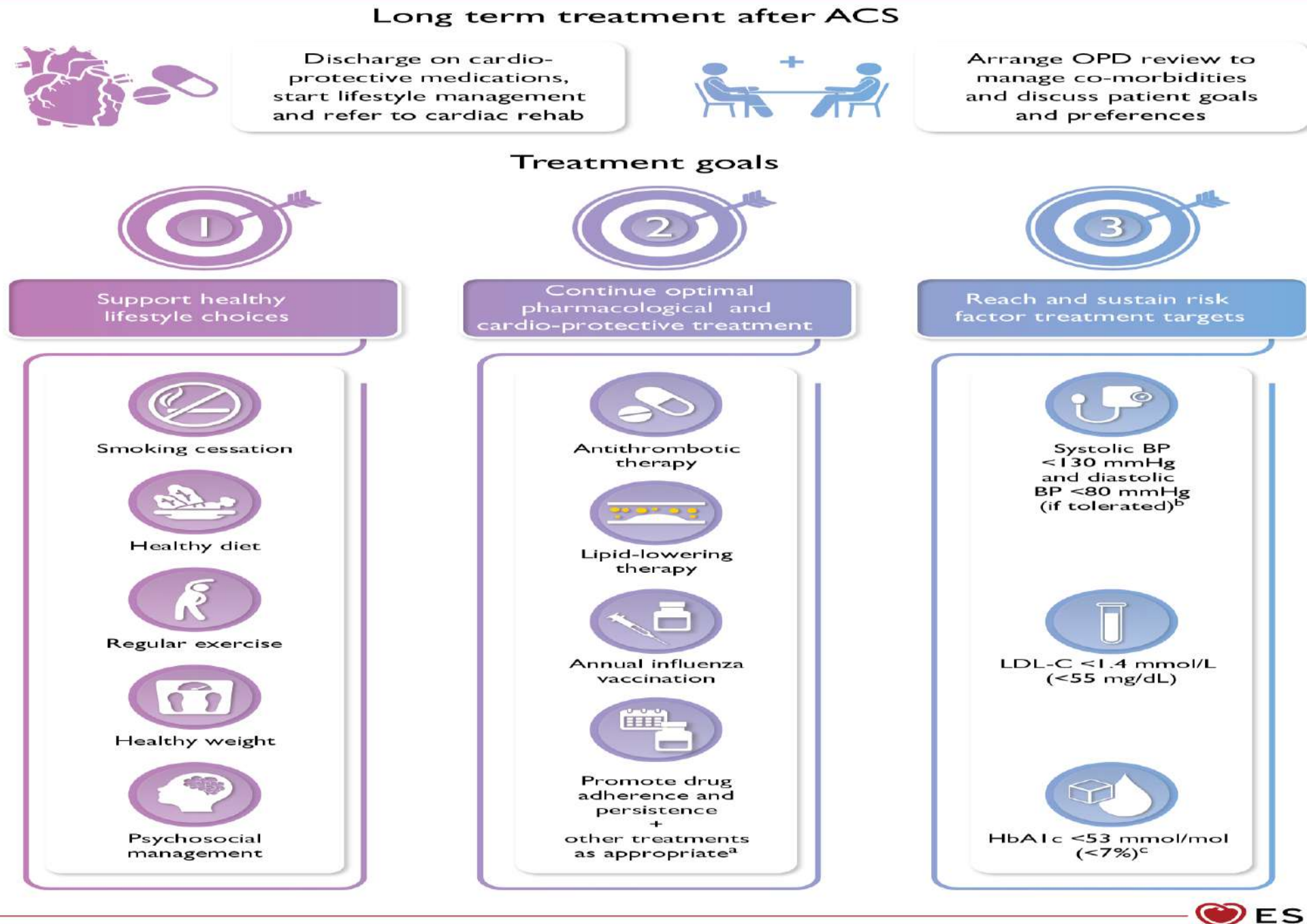

Heart
Kidney
los
Platelets
↑ Natriuresis
↑ Diuresis
↑ Cardioprotection
↓ Coagulation
↓ Blood Pressure
Blood Vessel
GLP-1
↓ Postprandial lipids
Intestine
↓ Body Weight
Brain
↓ Inflammation
Fat & other tissues
↓ Glucose
↓ Hypoglycemia
α-Cell
↓ Glucagon Secretion
β-Cell
↑ Insulin Secretion
↑ Insulin Biosynthesis
↓ Apoptosis

CENTRAL ILLUSTRATION: Meta-analysis of Cardiovascular Outcomes of Glucagon-Like Peptide 1 Receptor Agonist by Baseline Sodium-Glucose Cotransporter 2 Inhibitor Use

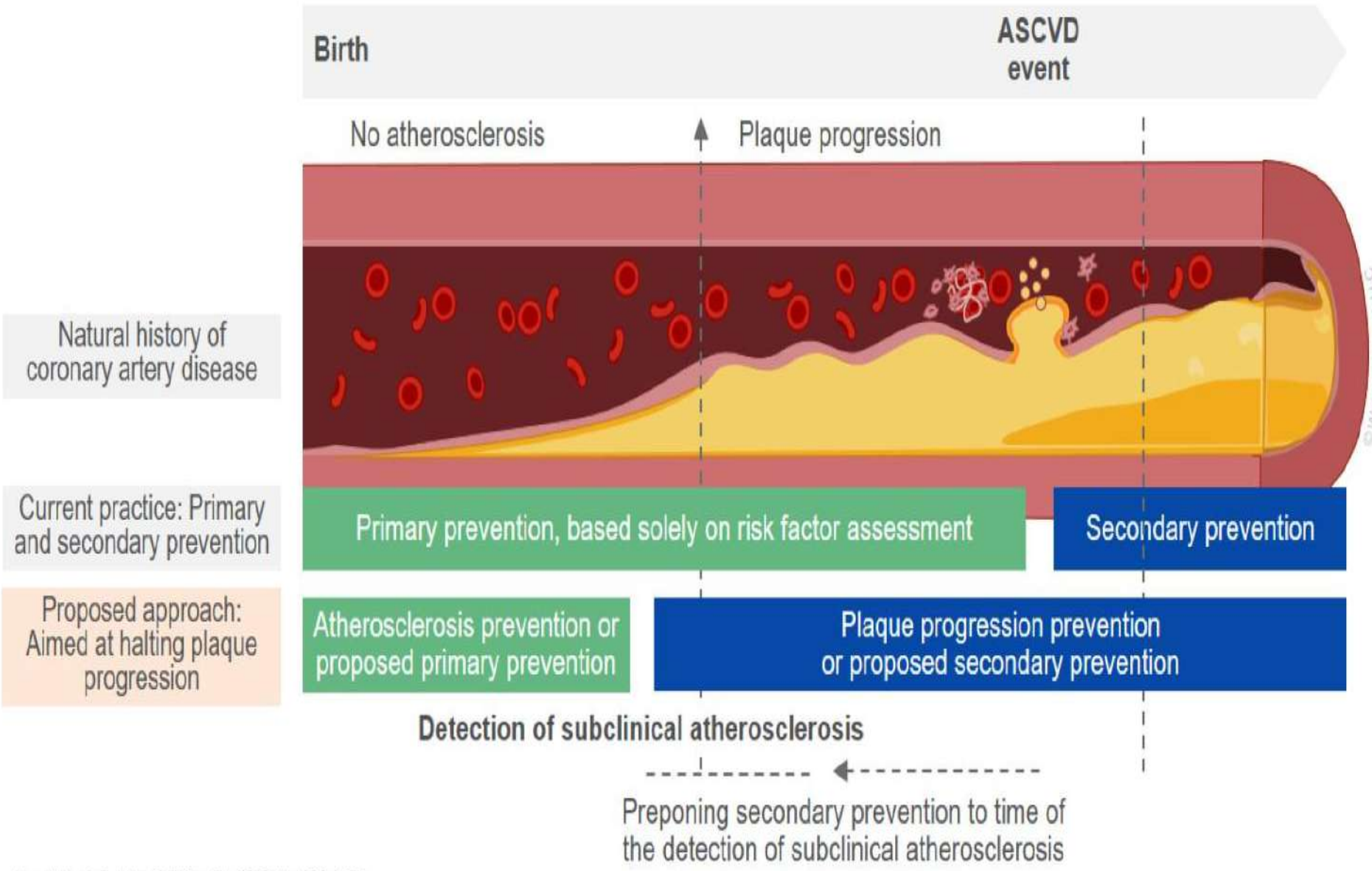


Neves JS, et al. J Am Coll Cardiol. 2023;82(6):517–525.

RESUMEN PREVENCION 2ria



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

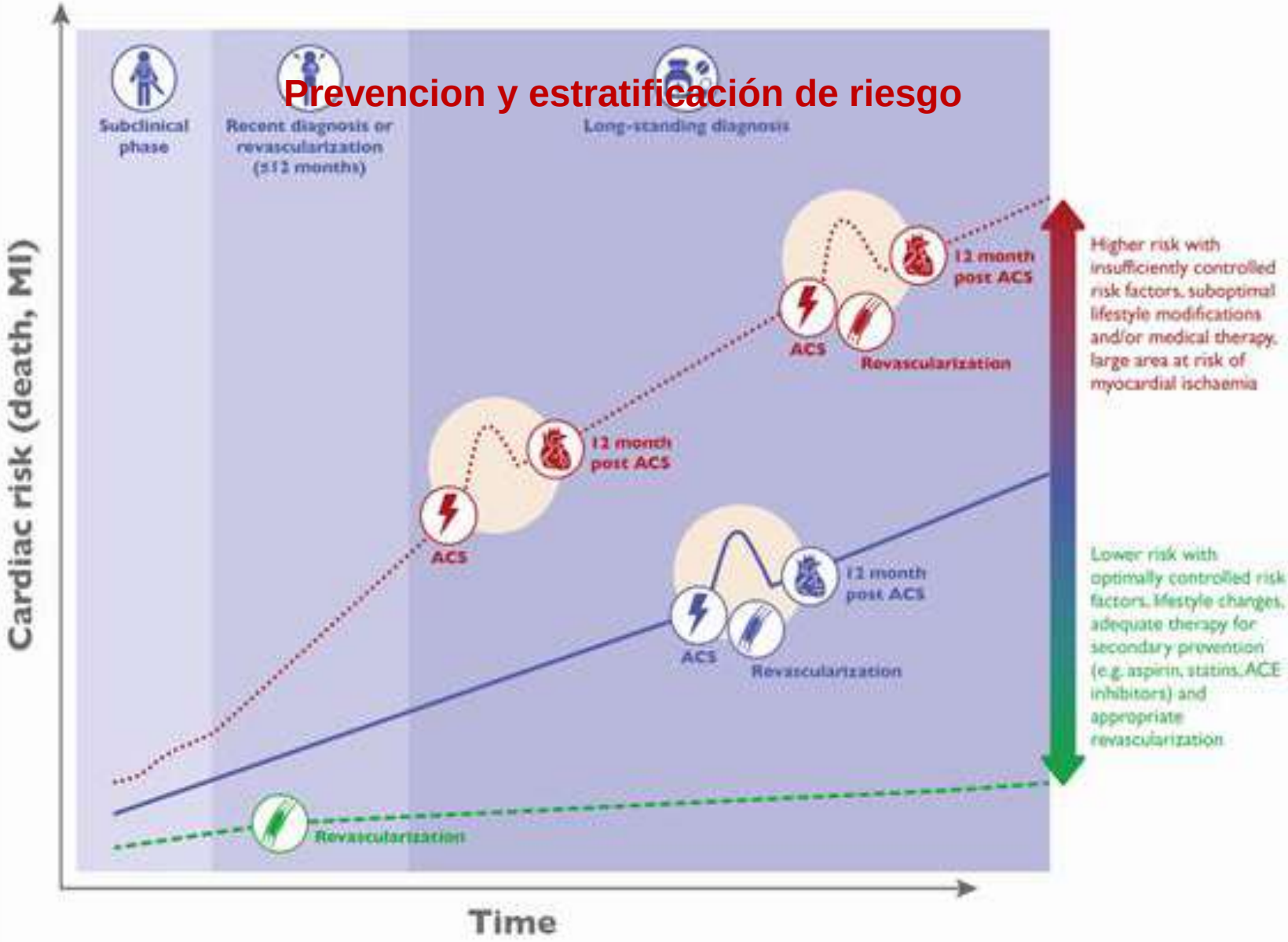


Ahmadi A, et al. J Am Coll Cardiol. 2019;74:1608-1617.

Natural history of chronic coronary syndromes

A dynamic process

Prevencion y estratificación de riesgo



#Trombolíticos (21%) 165 min Isquemia ➔ Mort **5.2%**

#Farmaco Invasiva 191 min. Isquemia ➔ Mort **4.9%**

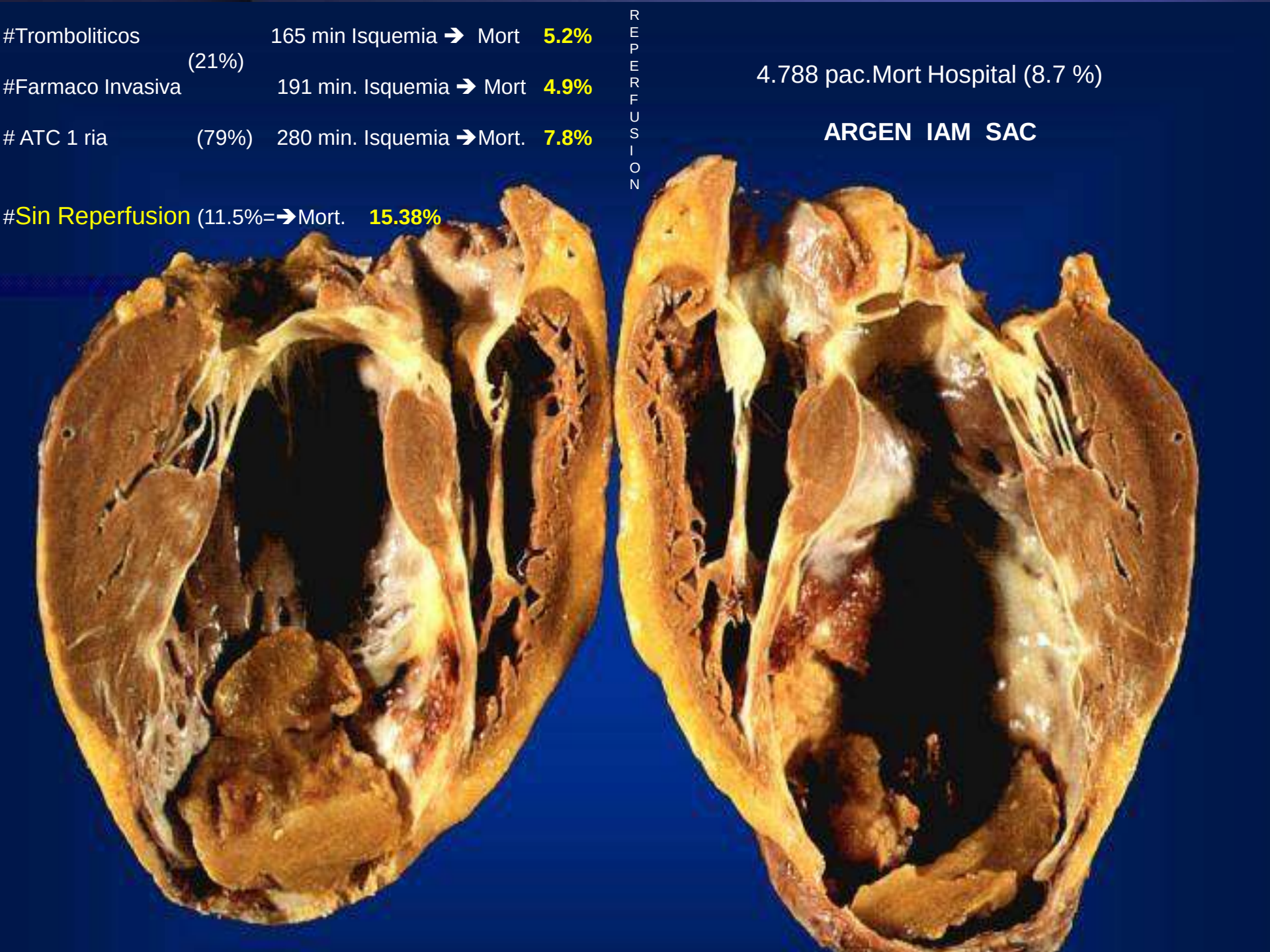
ATC 1 ria (79%) 280 min. Isquemia ➔Mort. **7.8%**

#Sin Reperfusion (11.5%=➔Mort. **15.38%**

REPERFUSION

4.788 pac.Mort Hospital (8.7 %)

ARGEN IAM SAC



CONCLUSIONES

(SCACEST IAM)

OBJETIVOS

REPERFUSION PRECOZ

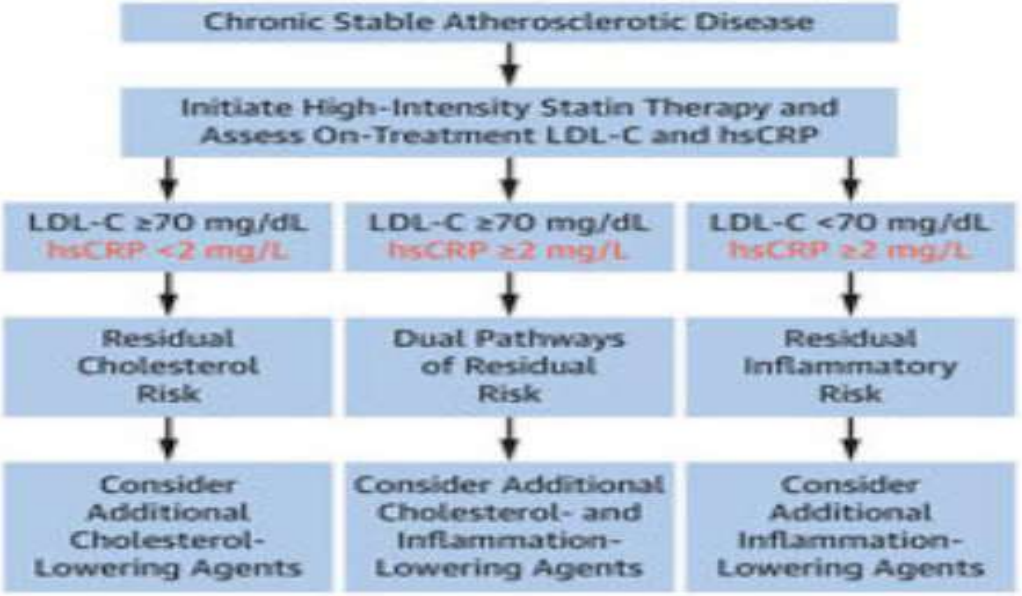
(Fibrinolíticos, FARMACO INVASIVA o ATC)

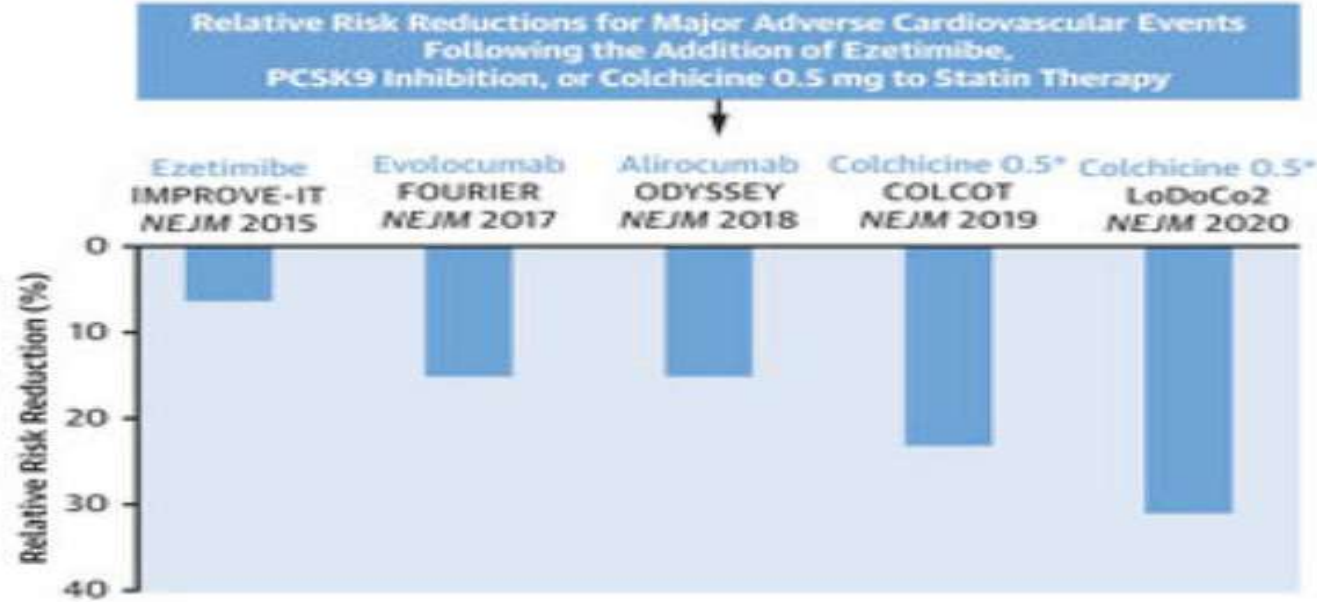
(REDUCCION DE MORBIMORTALIDAD)

PREVENCION SECUNDARIA =>MEJORA PRONOSTICO

(ATEROTROMBOSIS Y DISFUNCION VI)

CENTRAL ILLUSTRATION: Managing Residual Inflammatory Risk and Residual Cholesterol Risk in Stable Coronary Disease





Nelson K, et al. J Am Coll Cardiol. 2023;82(7):648-660.

WHY COLCHICINE?

Inflammation has been associated with increased cardiovascular event risk (CVE), regardless of LDL-C Level.

The CANTOS Trial evaluated the use of canakinumab (Interleukin-1b inhibitor) vs. placebo in 10,061 atherosclerosis patients with residual inflammatory risk (hsCRP >2 mg/L) despite statin therapy. CANTOS found a reduction in the composite endpoint of MI, stroke, or CV death. Limitations of canakinumab therapy include cost of agent, lack of mortality benefit, and increase in mortality due to infection seen in CANTOS trial.

Colchicine is a cheaper alternative to canakimumab. In patients with coronary artery disease, colchicine reduces inflammation and low-attenuation plaque volume.

DOSING AND FORMULATIONS

Dosing for Secondary Prevention:
Colchicine 0.5 mg PO daily

Branded Formulation	Generic Formulation
Colchicine 0.5 mg tablet	Colchicine 0.6 mg tablet
AWP $19.80/tablet	AWP $5.76-8.20/tablet

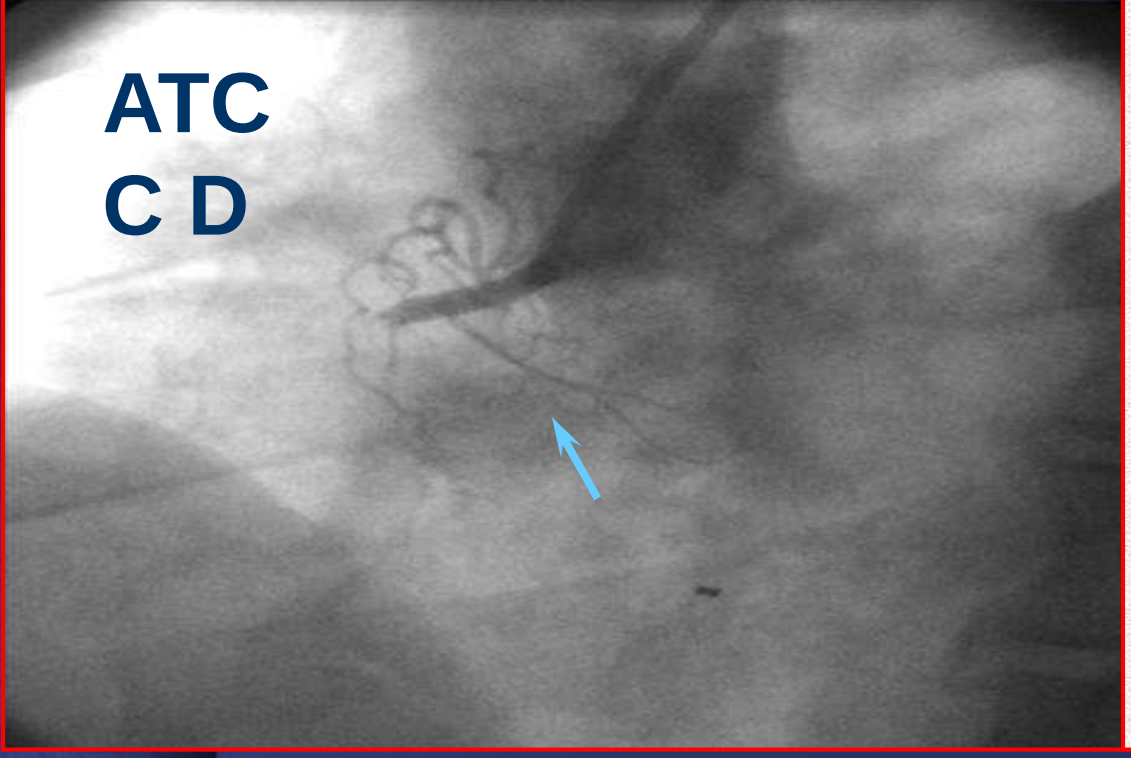
20% increase in dose when using generic formulation vs. branded formulation

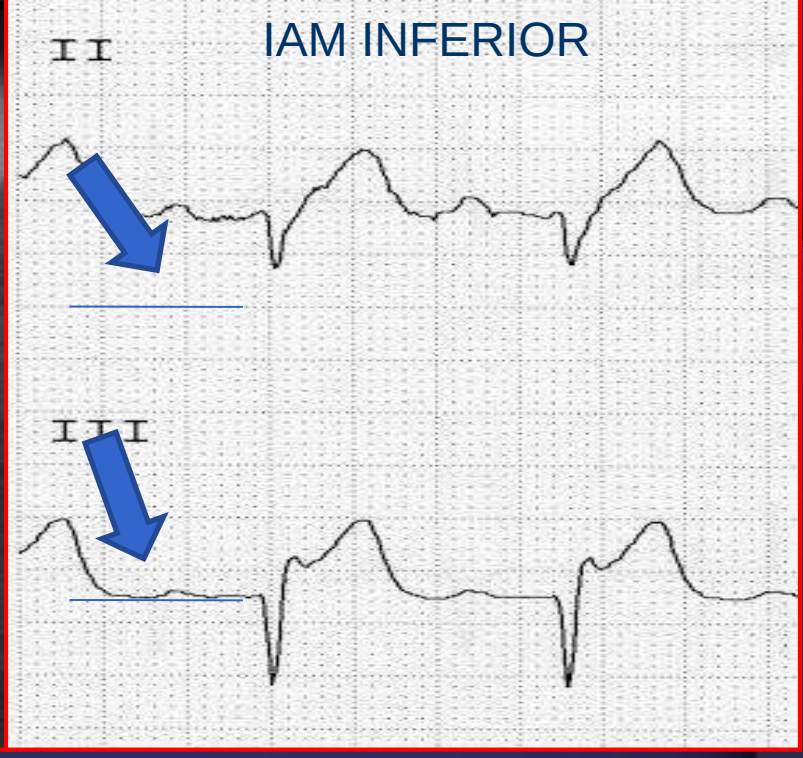
SUMMARY OF TRIALS

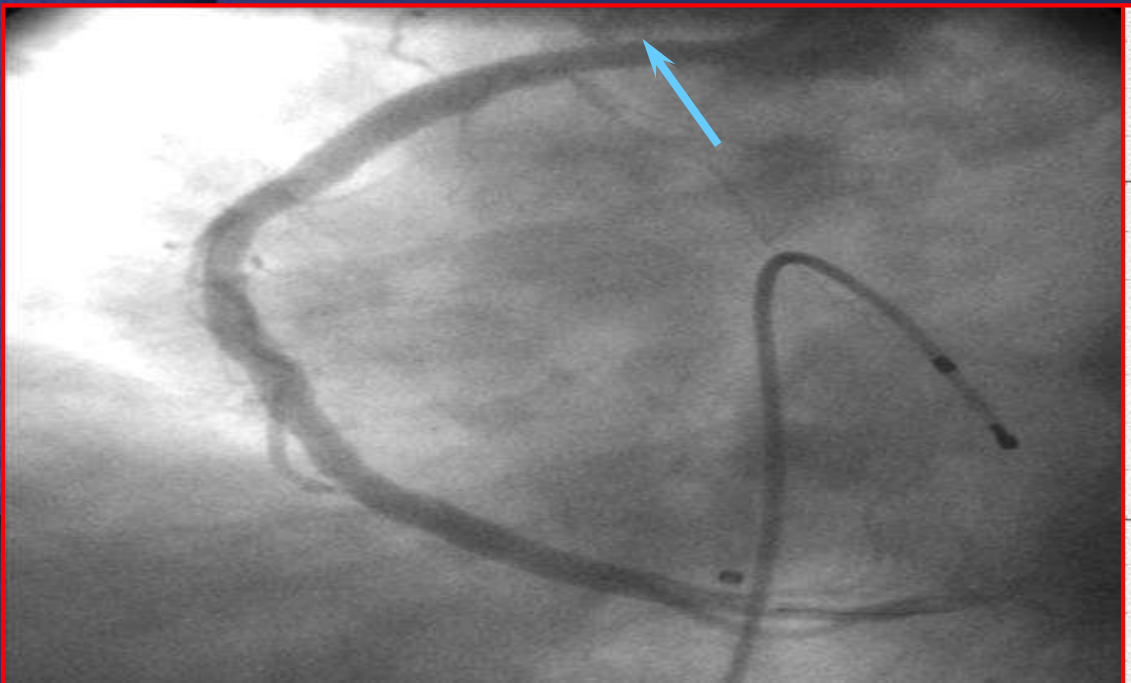
Trial	Population	Key Exclusion Criteria	Intervention	Primary Outcomes	Endpoints: Primary Efficacy	Endpoints: Safety
LoDoCo 2013	Clinically stable CAD for > 6 months	CABG < 10 years prior, PCI after CABG	Colchicine 0.5 mg daily (n=282) vs. placebo (n=250)	Composite of ACS, cardiac arrest, or ischemic stroke	5.3% vs. 16% p < 0.0001	
COLCOT 2019	Patients with MI within the last 30 days and completion of all intended coronary revascularization	CABG < 3 years prior, Severe HF, LVEF < 35%, Severe renal or hepatic disease	Colchicine0.5 mg daily (n=2,366) vs. placebo (n=2,379)	CV death, MI, stroke, resuscitated cardiac arrest, or urgent hospitalization for unstable angina leading to revascularization	5.5% vs. 7.1% P= 0.02	All Cause Morality 1.8% vs. 1.8% HR 0.98 (0.64–1.49)
LoDoCo2 2020	Clinically stable CAD for > 6 months	Severe heart failure, Moderate-to-severe renal impairment	Colchicine0.5 mg daily (n=2,762) vs. placebo (n=2,760)	Composite of CV death, spontaneous MI, ischemic stroke, or ischemia-driven coronary revascularization	6.8% vs. 9.6% p <0.001	Non-CV Morality 1.9% vs. 1.3% HR 1.51 (0.99–2.31)
COPS 2020	Patients with acute coronary syndromes (ACS) during index hospitalization	CAD requiring CABG, Severe renal or hepatic insufficiency	Colchicine 0.5 mg BID x1 month then 0.5 mg daily (n=396) vs. placebo (n=399)	Composite of death, ACS, ischemia-driven urgent revascularization and non-cardioembolic ischemic stroke	6.1% vs. 9.5% P=0.10	GI symptoms

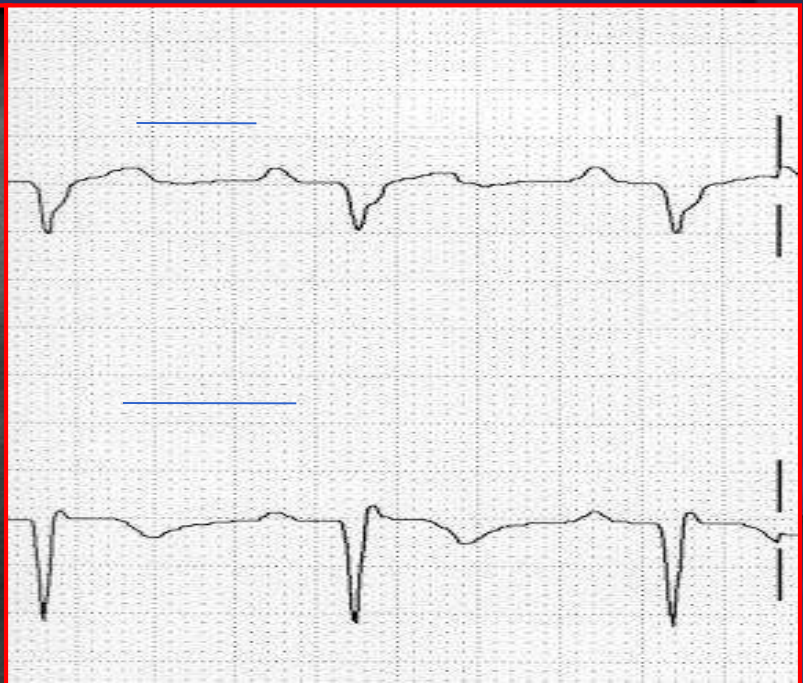
Recommendations for reperfusion therapy and timing of invasive strategy (1)

Recommendations	Class	Level
Recommendations for reperfusion therapy for patients with STEMI		
Reperfusion therapy is recommended in all patients with a working diagnosis of STEMI (persistent ST-segment elevation or equivalents) and symptoms of ischaemia of ≤12 h duration.	I	A
A PPCI strategy is recommended over fibrinolysis if the anticipated time from diagnosis to PCI is <120 min	I	A
If timely PPCI (<120m) cannot be performed in patients with a working diagnosis of STEMI, fibrinolytic therapy is recommended within 12 h of symptom onset in patients without contraindications.	I	A
Rescue PCI is recommended for failed fibrinolysis (i.e. ST-segment resolution <50% within 60–90 min of fibrinolytic administration) or in the presence of haemodynamic or electrical instability, worsening ischaemia, or persistent chest pain.	I	A


ATC
C D

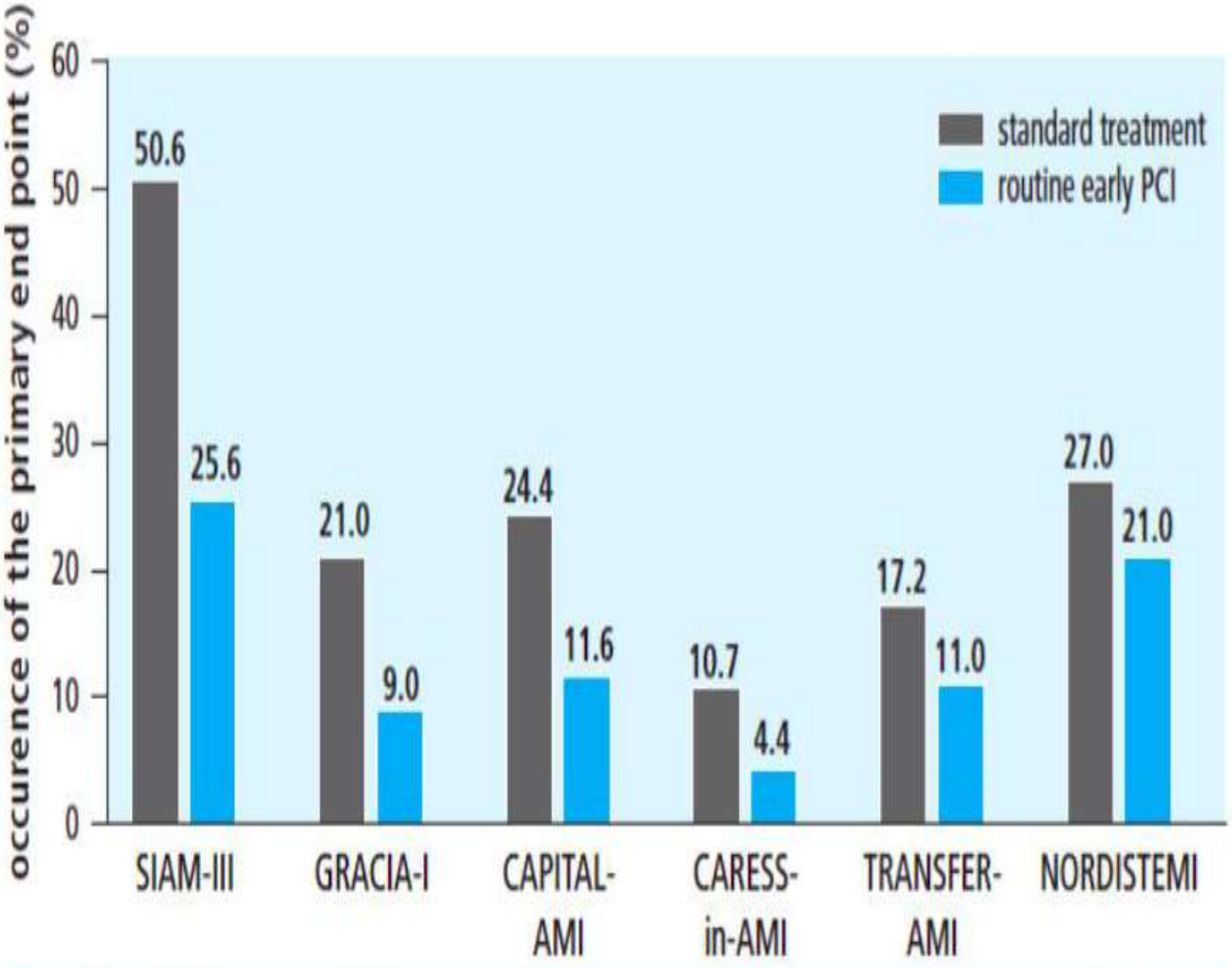

II
IAM INFERIOR
III





Fibrinolytic therapy *(continued)*

ESC
European Society of Cardiology



lass	Level
I	A
I	A
I	A
I	A

From 3-24 h to 2-24 h

Metaanalisis de Revascularizacion Completa

Además, en comparación con la revascularización completa por etapas, la revascularización completa en un solo ajuste redujo lo siguiente:

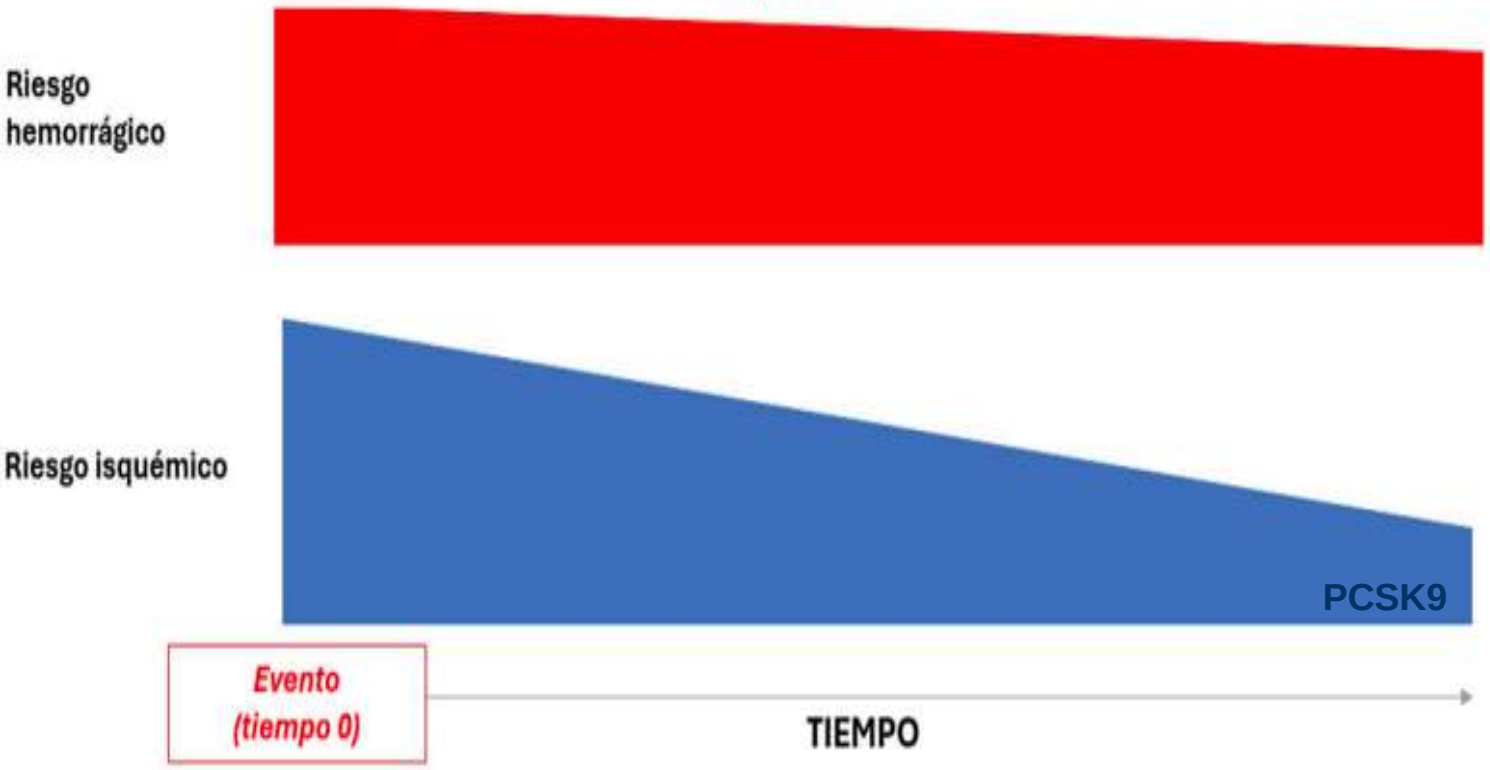
- Mortalidad cardiovascular/IM: OR 0,70, IC del 95 % 0,55-0,91
- Mortalidad por todas las causas/IM: OR 0,67, IC del 95 % 0,52-0,85
- IM solo: OR 0,53, IC del 95% 0,36-0,77
- MACE: OR 0,67, IC del 95 % 0,50-0,91

El Dr. Sripal Bangalore, MHA, de la Facultad de Medicina Grossman de la Universidad de Nueva York en la ciudad de Nueva York, y sus colegas descubrieron que la revascularización completa en un solo entorno era la mejor opción para todos los resultados, seguida de la revascularización completa en etapas y, en tercer lugar, la revascularización de la arteria culpable. Su informe se publicó en *Circulation: Cardiovascular Interventions*.

INDIVIDUALIZAR al PACIENTE

ALGORITMO DE DISLIPEMIA

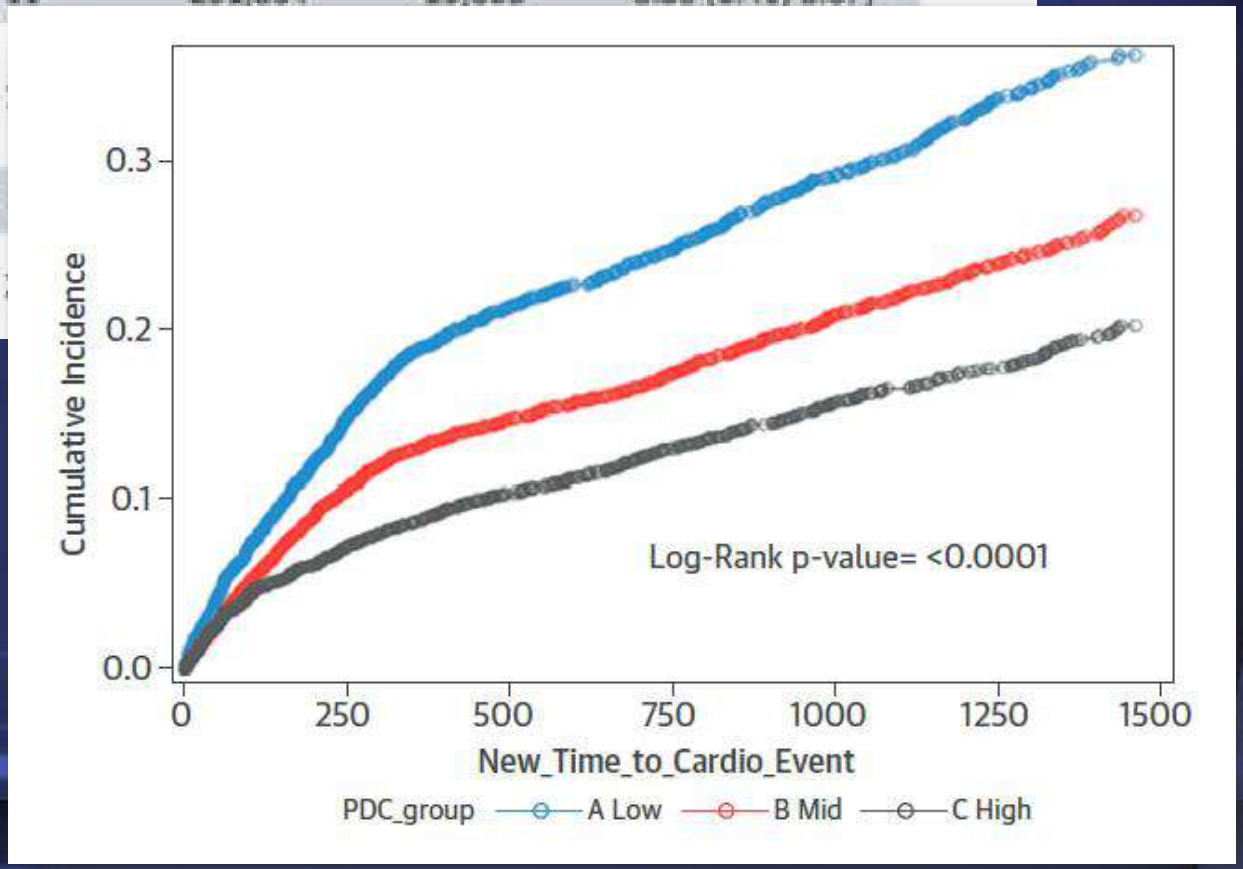
Procedimiento (ATC)		Clínica del paciente		Scores	
Acceso femoral, IP2Y12 mas potentes	EMV, Bif, nº *stents*, longitud de *stents*, OTC, *revasc completa*	ACO, sangrado previo, anemia, plaquetopenia, fragilidad, hepatopatía, fragilidad	SCA, DM, IRC, historia de trombosis del *stent*, IAM previo, EVP, IMC>30	PRECISE-DAPT, ARC-HBR, DAPT, CREDO-Kyoto, HASBLED	GRACE, TIMI, GUSTO, SYNTAX I y II



ATC: Angioplastia coronaria; iPY12: inhibidores de los receptores P2Y12; EMV: enfermedad de múltiples vasos; Bif: lesiones en bifurcación; OTC: oclusión total

Beneficio de la adherencia a largo plazo

	No. of Studies	No. of Participants	No. of Deaths	Proportion (95% CI)
Adherence to statins	11	291,864	29,605	0.55 (0.46, 0.67)
Adherence to antihypertensive agents				
Adherence to aspirin				
Adherence to any CVD medication				



Chronic coronary syndromes in specific circumstances

Diabetes mellitus

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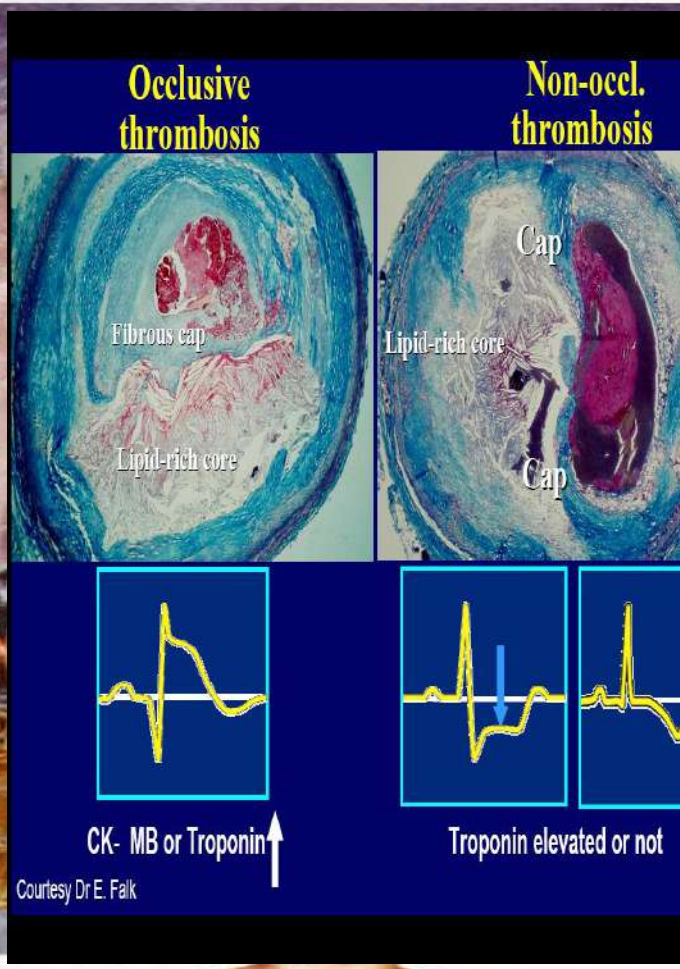
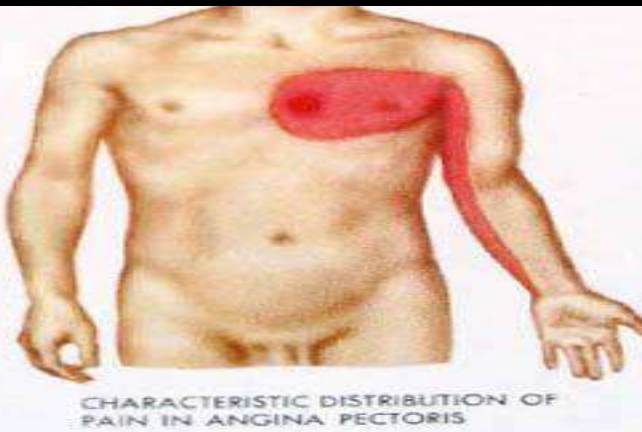
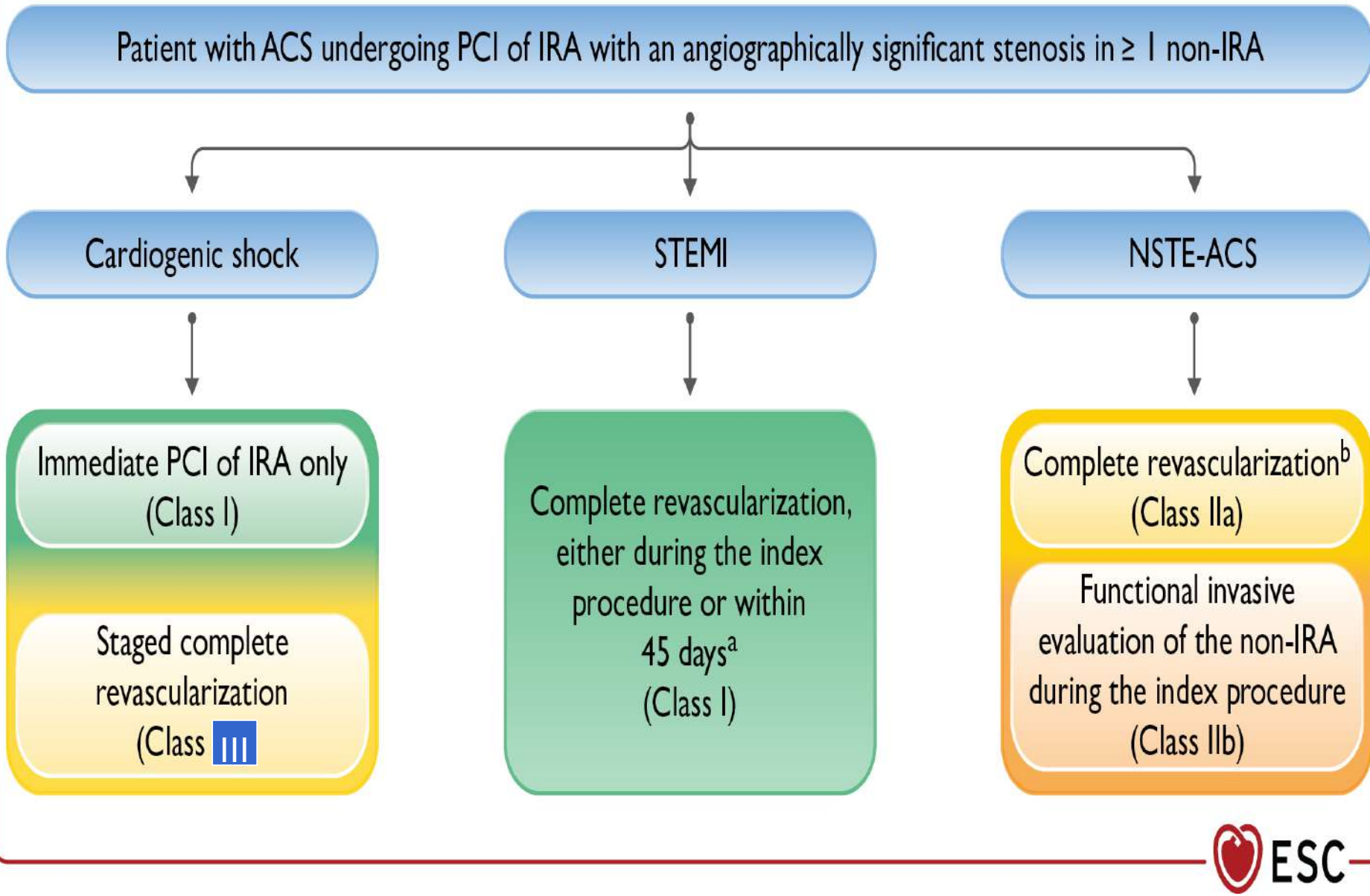
Recommendations	Class	Level
Risk factor (BP, LDL-C, and HbA1c) control to targets is recommended in patients with CAD and diabetes mellitus.	I	A
In asymptomatic patients with diabetes mellitus, a periodic resting ECG is recommended for cardiovascular detection of conduction abnormalities, AF, and silent MI.	I	C
ACE inhibitor treatment is recommended in CCS patients with diabetes for event prevention.	I	B
The sodium-glucose co-transporter 2 inhibitors empagliflozin, canagliflozin, or dapagliflozin are recommended in patients with diabetes and CVD.	I	A
A glucagon-like peptide-1 receptor agonist (liraglutide or semaglutide) is recommended in patients with diabetes and CVD.	I	A
In asymptomatic adults (age >40 years) with diabetes, functional imaging or coronary CTA may be considered for advanced cardiovascular risk assessment.	IIb	B



ANGOR > 20 Minutos



SCA y MULTIPLES VASOS



IAM RESUMEN DATOS DE ARGENTINA
SAC ARGEN IAM ST (2015-2024)

4.788 pac.Mort Hospital 8.7 %

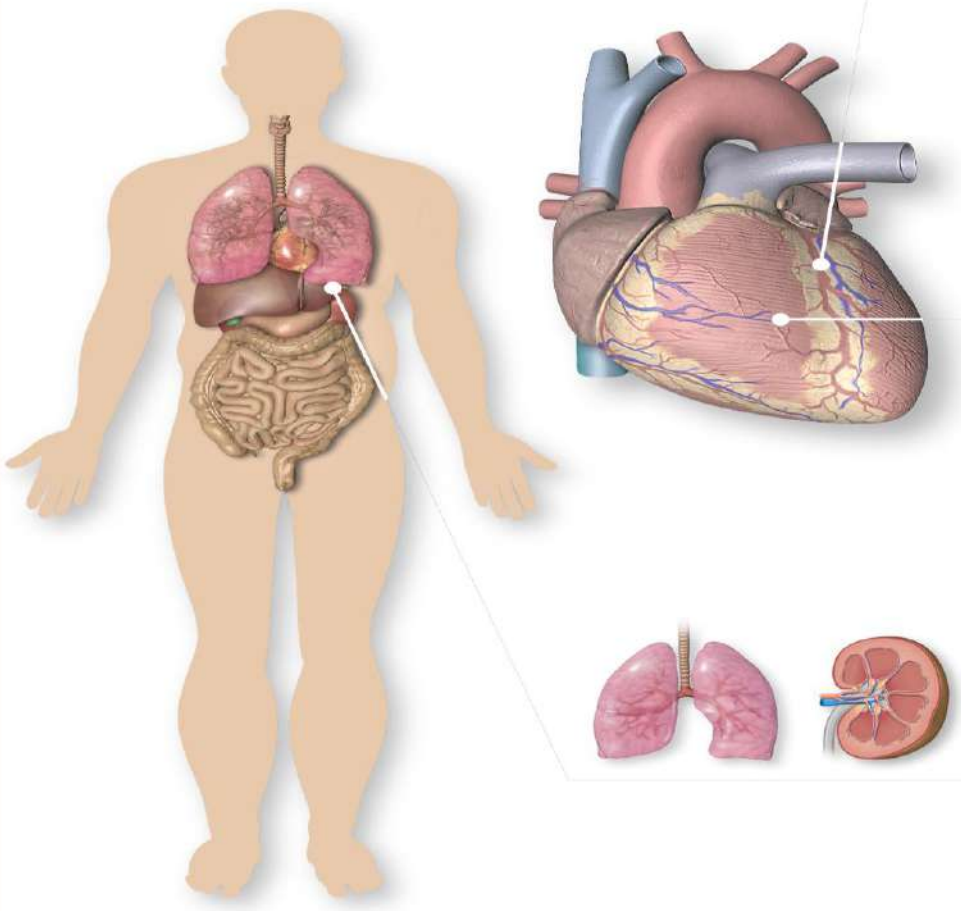
#Trombolíticos (8.5%) 165 min Isquemia ➔ Mort 5.2%

#Farmaco Invasiva (2.98%) 191 min. Isquemia ➔ Mort 4.9%

ATC 1 ria (88.5%) 280 min. Isquemia ➔Mort. 7.8%

#Sin Reperfusion (11.5%) =➔Mort. 15.38%

CAUSAS DE IAM
SIN OBSTRUCCION CORONARIA



Coronary causes

- Coronary embolism
- Coronary microvascular dysfunction
- Coronary spasm
- Coronary thrombosis
- Myocardial bridging
- Plaque rupture/erosion
- Spontaneous coronary artery dissection

Non-coronary, cardiac causes

- Cardiac trauma
- Cardiomyopathy
- Cardiotoxins
- Myocarditis
- Strenuous exercise
- Takotsubo cardiomyopathy
- Transplant rejection

Non-cardiac causes

- Acute respiratory distress syndrome
- Allergic/hypersensitivity reactions
- End-stage renal failure
- Inflammation
- Pulmonary embolism
- Sepsis
- Stroke

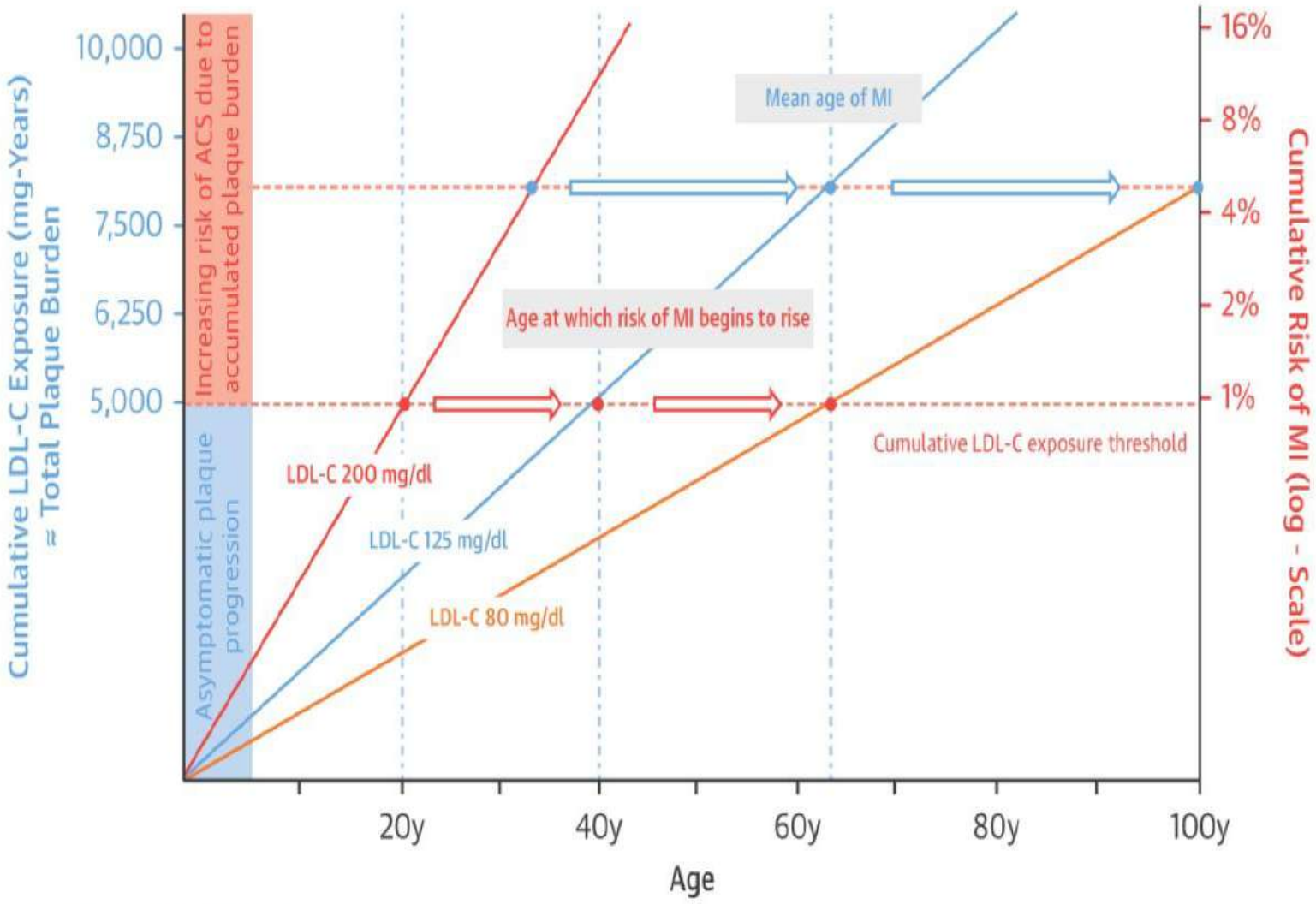
ESC

MINOCA

SAC 2025

Recomendación	Clase	Nivel de evidencia
Sospecha o evidencia de accidentes de placa o erosión: comenzar con antiagregantes de acuerdo con las guías nacionales de síndromes coronarios agudos. (76,345)	I	C
Estatinas a dosis intensiva en todos los pacientes con MINOCA. (346,347)	I	B
En los casos de evidencia o alta sospecha de vasoespasmo de arterias coronarias epicárdicas o espasmo microvascular, se recomienda el uso de bloqueantes cálcicos (diltiazem, verapamilo) y nitratos (endovenosos y orales). (348-350)	I	B
En los casos de evidencia o alta sospecha de disfunción microvascular, se recomienda el uso de betabloqueantes (nebivolol), bloqueantes cálcicos, estatinas y IECA y ARA II, salvo contraindicaciones o intolerancia.	IIa	C

Modifying Disease Course by Slowing Atherosclerosis Progression



Ference BA, et al. J Am Coll Cardiol. 2018;72:1141-1156.

ACS encompasses a spectrum

Unstable angina — NSTEMI — STEMI

1 Think 'A.C.S.' at initial assessment

- **A** — Abnormal ECG?
- **C** — Clinical context?
- **S** — Stable patient?

2 Think invasive management

STEMI	Very high-risk NSTE-ACS	High-risk NSTE-ACS
Primary PCI OR Fibrinolysis (If timely primary PCI not feasible)	Immediate angiography ± PCI	Early (<24 h) angiography should be considered

3 Think antithrombotic therapy

Antiplatelet therapy	AND	Anticoagulant therapy
Aspirin + $P2Y_{12}$ inhibitor		UFH OR LMWH OR Bivalirudin OR Fondaparinux

4 Think revascularization

Based on clinical status, co-morbidities, and disease complexity	Aim for complete revascularization	Consider adjunctive tests to guide revascularization
PCI OR CABG		Intravascular imaging; Intravascular physiology

5 Think secondary prevention

- Antithrombotic therapy
- Lipid lowering therapy
- Smoking cessation
- Cardiac rehabilitation
- Risk factor management
- Psychosocial considerations

ESC