1er FORO DE RESIDENTES DE MEDICINA INTENSIVA DE CASTILLA LA MANCHA

INTRODUCCION AL PACIENTE CRÍTICO.

Dr Rafael Blancas. Hospital del Tajo

Síndrome coronario agudo

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SOCIEDAD DE MEDICINA INTENSIVA Y UNIDADES CORONARIAS DE CASTILLA-LA MANCHA

Síndrome coronario agudo sin elevación persistente del segmento ST

Recomendaciones

ACC/AHA Guideline Revision

ACC/AHA 2007 Guidelines for the Management of Patients With Unstable Angina/Non–ST-Elevation Myocardial Infarction: Executive Summary

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients With Unstable Angina/Non–ST-Elevation Myocardial Infarction)

Developed in Collaboration with the American College of Emergency Physicians, the Society for Cardiovascular Angiography and Interventions, and the Society of Thoracic Surgeons

Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation and the Society for Academic Emergency Medicine

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rican Heart Association, Inc.

FULL TEXT (with 2002 edits highlighted)

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Infarction

of Cardiology/American Heart Association
(Committee on the Management of Patients

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istent ST-segment elevation

nt of Acute Coronary Syndromes of the
ciety of Cardiology*

Simoons, Keith A. A. Fox, Lars C. Wallentin,
ne McFadden, Pim J. De Feyter,
cchia, Witold Ruzyllo

Introduction

The clinical presentations of ischaemic heart disease include stable angina pectoris, silent ischaemia, unstable angina, myocardial infarction, heart failure, and sudden death. For many years, unstable angina has been considered as an intermediate 'syndrome' between chronic stable angina and acute myocardial infarction. In recent years, its physiopathology has been clarified and there have been major advances in management.

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Síndrome coronario agudo sin elevación persistente del segmento ST

Definición (ESC): *dolor torácico y alguno de los siguientes*

ECG

- **Elevación transitoria del segmento ST (menos de 20 minutos)**
- **Descenso del segmento ST**
- **Cambios en la onda T**
- **Pseudonormalización de la onda T**
- **Sin cambios ST/T**

Síndrome coronario agudo sin elevación persistente del segmento ST

Definición (ESC): *dolor torácico y alguno de los siguientes*

Troponina (marcadores cardiacos)

- **Elevación: IAM sin elevación de ST**
- **Normal: Angina inestable**

Síndrome coronario agudo sin elevación persistente del segmento ST

Troponina

TABLA 3. **Enfermedades no coronarias con elevación de las troponinas**[68]

- Insuficiencia cardiaca congestiva grave: aguda y crónica
- Disección aórtica, valvulopatía aórtica, o miocardiopatía hipertrófica
- Contusión cardiaca, ablación, estimulación cardiaca, cardioversión o biopsia endomiocárdica
- Enfermedades inflamatorias como, por ejemplo, miocarditis o extensión miocárdica de endocarditis/pericarditis
- Crisis hipertensiva
- Taquiarritmias o bradiarritmias
- Embolia pulmonar, hipertensión pulmonar severa
- Hipotiroidismo
- Síndrome del *apical ballooning*
- Disfunción renal crónica o aguda
- Enfermedad neurológica aguda, como accidente cerebrovascular o hemorragia subaracnoidea
- Enfermedades infiltrativas, como amiloidosis, hemocromatosis, sarcoidosis o esclerodermia
- Toxicidad farmacológica, como adriamicina, 5-fluorouracilo, herceptina, venenos de serpiente
- Quemaduras, cuando afectan a más del 30% de la superficie corporal
- Rabdomiolisis
- Pacientes críticos, especialmente con insuficiencia respiratoria o sepsis

Síndrome coronario agudo sin elevación persistente del segmento ST

Diagnóstico diferencial

TABLA 4. **Enfermedades cardiacas y no cardiacas que pueden semejar síndromes coronarios agudos sin elevación del segmento ST**

Cardiacas	Pulmonares	Hemáticas	Vasculares	Gastrointestinales	Ortopédicas
Miocarditis	Embolia pulmonar	Anemia falciforme	Disección aórtica	Espasmo esofágico	Discopatía cervical
Pericarditis	Infarto pulmonar		Aneurisma aórtico	Esofagitis	Fractura de costilla
Miopericarditis	Neumonía, pleuritis		Coartación aórtica	Úlcera péptica	Daño o inflamación muscular
Miocardiopatía	Neumotórax		Enfermedad cerebrovascular	Pancreatitis	Costocondritis
Valvulopatía				Colecistitis	
Apical ballooning (síndrome de Tako-Tsubo)					

1er FORO DE RESIDENTES DE MEDICINA INTENSIVA DE CASTILLA LA MANCHA

SINDROME CORONARIO AGUDO

Dr Rafael Blancas. Hospital del Tajo

Clasificación del riesgo

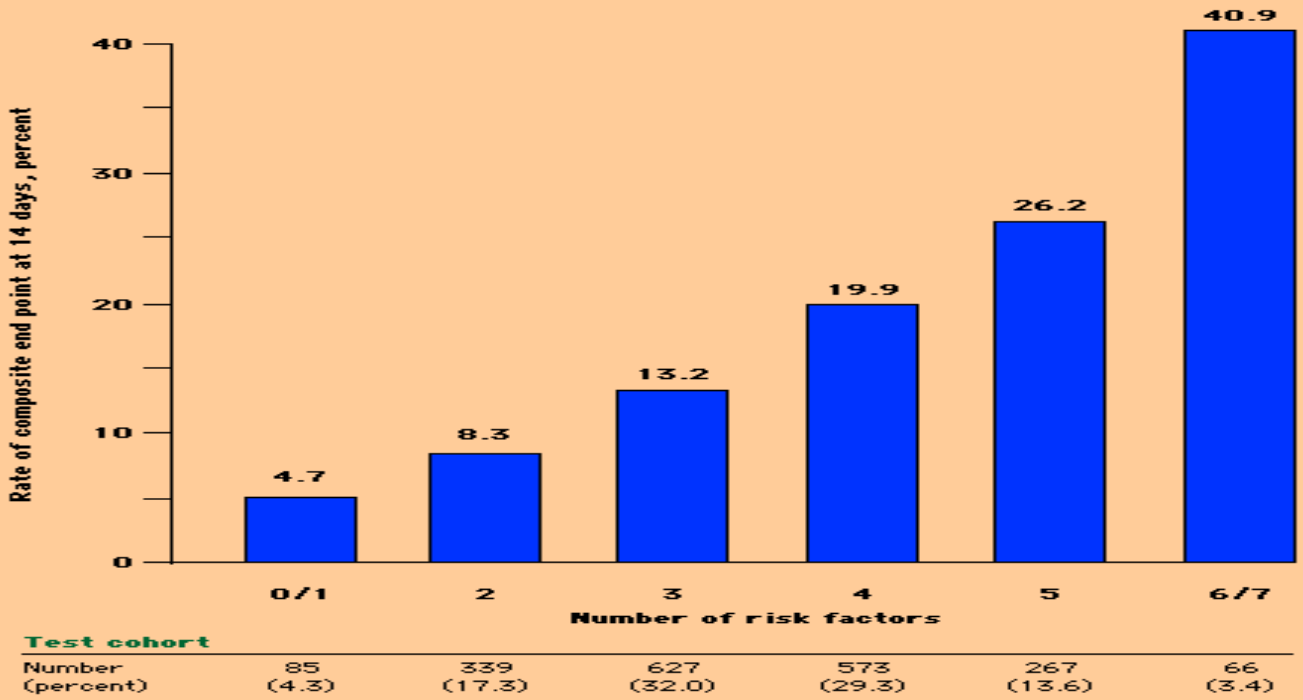
Riesgo TIMI

- ✓ Edad > 65 años
- ✓ Al menos tres factores de riesgo de enfermedad cardiovascular de entre:
  - Fumador activo
  - Hipercolesterolemia
  - HTA
  - Historia familiar de enfermedad coronaria
  - Diabetes
- ✓ Historia previa de cardiopatía isquémica (IAM, ACTP o cirugía)
- ✓ Marcadores cardiacos elevados
- ✓ Desviación de ST
- ✓ Uso de AAS en los 7 días previos
- ✓ Dos o más episodios de angina de reposo en las últimas 24 h

Alto	>5 puntos
Medio	3-5 “
Bajo	<3 “

Síndrome coronario agudo sin elevación persistente del segmento ST

Riesgo TIMI



TIMI risk score for non-ST elevation ACS Rates of all-cause mortality, myocardial infarction, and severe recurrent ischemia prompting urgent revascularization **at 14 days** after randomization according to the number of risk factors among patients with a non-ST elevation acute coronary syndrome (ACS) in TIMI 11B and ESSENCE. The risk factors were age ≥65 years; presence of at least three risk factors for coronary disease; prior coronary stenosis of ≥50 percent; presence of ST segment deviation on admission ECG; at least two anginal episodes in prior 24 hours; use of aspirin in prior seven days; and elevated serum cardiac biomarkers. Event rates increased significantly as the TIMI risk score rose. Patients are considered to be at low risk with a score of 0 to 2; intermediate risk with a score of 3 to 4; and high risk with a score of 5 to 7. (Adapted from Antman, EM, Cohen, M, Bernink, PJ, et al, JAMA 2000; 284:835.)

Síndrome coronario agudo sin elevación persistente del segmento ST

Riesgo GRACE

Risk Calculator for 6-Month Postdischarge Mortality After Hospitalization for Acute Coronary Syndrome

Record the points for each variable at the bottom left and sum the points to calculate the total risk score. Find the total score on the x-axis of the nomogram plot. The corresponding probability on the y-axis is the estimated probability of all-cause mortality from hospital discharge to 6 months.

Medical History

① Age in Years	Points
≤29	0
30–39	0
40–49	18
50–59	36
60–69	55
70–79	73
80–89	91
≥90	100

② History of Congestive Heart Failure	24
③ History of Myocardial Infarction	12

Findings at Initial Hospital Presentation

④ Resting Heart Rate, beats/min	Points
≤49.9	0
50–69.9	3
70–89.9	9
90–109.9	14
110–149.9	23
150–199.9	35
≥200	43

⑤ Systolic Blood Pressure, mm Hg	
≤79.9	24
80–99.9	22
100–119.9	18
120–139.9	14
140–159.9	10
160–199.9	4
≥200	0

⑥ ST-Segment Depression	11

Findings During Hospitalization

⑦ Initial Serum Creatinine, mg/dL	Points
0–0.39	1
0.4–0.79	3
0.8–1.19	5
1.2–1.59	7
1.6–1.99	9
2–3.99	15
≥4	20

⑧ Elevated Cardiac Enzymes	15
⑨ No In-Hospital Percutaneous Coronary Intervention	14

Points

① _____
② _____
③ _____
④ _____
⑤ _____
⑥ _____
⑦ _____
⑧ _____
⑨ _____

Total Risk Score _____ (Sum of Points)

Mortality Risk _____ (From Plot)

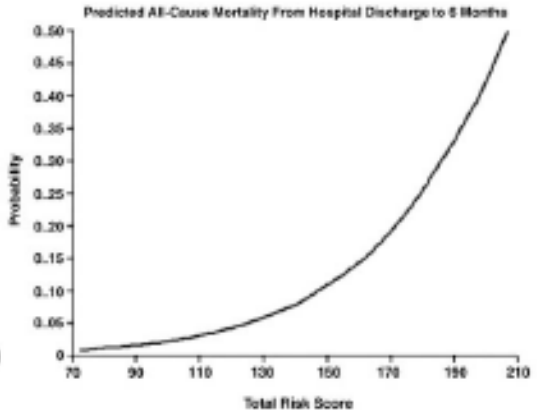


Figure 3. GRACE Prediction Score Card and Nomogram for All-Cause Mortality From Discharge to 6 Months

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Riesgo GRACE

TABLA 5. **Mortalidad intrahospitalaria y a los 6 meses tras el alta en las categorías de riesgo bajo, intermedio y alto de los registros poblacionales según la clasificación de riesgo GRACE**[8,117]

Categoría de riesgo (terciles)	Clasificación de riesgo GRACE	Muertes intrahospitalarias (%)
Bajo	≤ 108	< 1
Intermedio	109-140	1-3
Alto	> 140	> 3
Categoría de riesgo (terciles)	**Clasificación de riesgo GRACE**	**Muertes hasta 6 meses tras el alta (%)**
Bajo	≤ 88	< 3
Intermedio	89-118	3-8
Alto	> 118	> 8

Para los cálculos, véase http://www.outcomes.org/grace.

www.outcomes.org/grace

Síndrome coronario agudo sin elevación persistente del segmento ST

Alto riesgo coronario

- Ascenso transitorio del segmento ST (>0,1 mV)
- Descenso del segmento ST (>0,1 mV)
- Cambios en onda T
- Elevación de marcadores cardíacos (trop Ic > 0,5, trop I< 0,1)
- Angina postIAM
- Inestabilidad hemodinámica
- TV recidivante o FV (en relación con angor)
- Diabetes mellitus
- ECG que impida valorar el segmento ST

Síndrome coronario agudo sin elevación persistente del segmento ST

Tratamiento

SCASEST

- Antiagregantes
- IGP IIb/IIIa
- Anticoagulantes

- Betabloqueantes
- Ca antagonistas
- Nitratos
- IECAs
- ARA II

Medidas generales

O2: en las primeras 6 h. Continuar si satO2 <90% o taquipnea

Reposo: en cama las primeras 12 h. Luego pasar a sillón

Dieta: absoluta durante 12 h. Luego, iniciar baja en sal y grasas

AINEs: suspender durante el ingreso (salvo AAS) por riesgo de reIAM, muerte, fallo ventricular, HTA y ruptura cardiaca

Estatinas: al ingreso, para obtener cLDL< 100 mg/dl (posteriormente, conseguir cLDL < 70 mg/dl)

Protectores gástricos: ranitidina, omeprazol, sucralfato

Medidas generales

Analítica: al ingreso y a las 6, 12, 24, 48 h. Obtener hemograma, coagulación, bioquímica. Perfil lipídico antes del alta.

ECG: ingreso, diario y si cambios clínicos (desaparición/ reaparición de dolor, arritmias, shock, etc)

Rx tórax: al ingreso y si cambios clínicos

ECOcardio: en función de la situación clínica (si disponible, antes de alta a planta)

Betabloqueantes

ESC: Recomendados en ausencia de fallo ventricular, asma, historia de hiperreactividad bronquial o bloqueo AV (I-B)

ACC/AHA: iniciarlos de forma oral en las primeras 24 h, si no existen las contraindicaciones mencioandas (I-B)

Antagonistas canales del Ca

ESC: diltiazem o verapamilo; nifedipino en angina vasoespástica (I-B)

ACC/AHA: si contraindicación a betabloqueantes o angina recurrente (I-B)

Nitratos

Contraindicados en pacientes que toman sildenafilo, vardenafilo, tadalafilo.

ESC: recomendados para mejora de síntomas (I-C)

ACC/AHA: en caso de angina recurrente. Administrar IV si isquemia persistente, fallo cardiaco o HTA (I-B)

Síndrome coronario agudo sin elevación persistente del segmento ST

Anticoagulación

HNF

Enoxaparina: rev. sistemática

JAMA, 2004

Conclusion In a systematic overview of approximately 22000 patients across the spectrum of ACS, enoxaparin is more effective than unfractionated heparin in preventing the combined end point of death or MI.

Fondaparinux: OASIS-5

N Eng J Med, 2006

CONCLUSIONS

Fondaparinux is similar to enoxaparin in reducing the risk of ischemic events at nine days, but it substantially reduces major bleeding and improves long term mortality and morbidity. (ClinicalTrials.gov number, NCT00139815.)

Bivalirudina: ACUITY,

N Eng J Med, 2006

CONCLUSIONS

In patients with moderate- or high-risk acute coronary syndromes who were undergoing invasive treatment with glycoprotein IIb/IIIa inhibitors, bivalirudin was associated with rates of ischemia and bleeding that were similar to those with heparin. Bivalirudin alone was associated with similar rates of ischemia and significantly lower rates of bleeding. (ClinicalTrials.gov number, NCT00093158.)

Anticoagulación

A. Estrategia invasiva urgente

HNF, enoxaparina o bivalirudina

B. Estrategia invasiva precoz o conservadora:

Fondaparinux mejor perfil eficacia/seguridad

Administrar enoxaparina sólo si bajo riesgo hemorrágico

Anticoagulación

a. En ACTP/stent mantener el mismo tipo de anticoagulación inicial
b. Si se ha utilizado fondaparinux, durante ACTP/stent debe administrarse un bolo HNF de 50-100 UI/Kg
c. Tras ACTP/stent, la anticoagulación se mantendrá durante 24 h.
d. En estrategia conservadora, se mantendrá anticoagulación con fondaparinux, enoxaparina o bivalirudina hasta el alta o máximo de 8 días.

Anticoagulación

Fondaparinux: 2,5 mg/día sc

Enoxaparina: 1 mg/Kg/12 h

HNF: 60-70 UI/Kg en bolo, con máximo de 5.000 UI. Infusión a 12-15 UI/Kg/h (máximo 1.000 UI/h) para aPTT 1,5-2 veces el control.

Bivalirudina: bolo de 0,1 mg/Kg. Infusión de 0,25 mg/Kg/h. Antes de ACTP, bolo adicional de 0,5 mg/Kg y aumentar infusión hasta 1,75 mg/Kg/h.

Aspirina

ESC: dosis inicial de 160 a 325 mg, mantenimiento con 75-100 mg. Mantener de forma indefinida

ACC/AHA: administrar de forma indefinida. Recomiendan administrar protectores de la mucosa digestiva

En caso de intolerancia o contraindicación, sustituir por clopidogrel

Síndrome coronario agudo sin elevación persistente del segmento ST

Clopidogrel

The New England Journal of Medicine

EFFECTS OF CLOPIDOGREL IN ADDITION TO ASPIRIN IN PATIENTS WITH ACUTE CORONARY SYNDROMES WITHOUT ST-SEGMENT ELEVATION

THE CLOPIDOGREL IN UNSTABLE ANGINA TO PREVENT RECURRENT EVENTS TRIAL INVESTIGATORS*

ABSTRACT

Background Despite current treatments, patients who have acute coronary syndromes without ST-segment elevation have high rates of major vascular events. We evaluated the efficacy and safety of the antiplatelet agent clopidogrel when given with aspirin in such patients.

Methods We randomly assigned 12,562 patients who had presented within 24 hours after the onset of symptoms to receive clopidogrel (300 mg immediately, followed by 75 mg once daily) (6259 patients) or placebo (6303 patients) in addition to aspirin for 3 to 12 months.

Results The first primary outcome — a composite of death from cardiovascular causes, nonfatal myocardial infarction, or stroke — occurred in 9.3 percent of the patients in the clopidogrel group and 11.4 percent of the patients in the placebo group (relative risk with clopidogrel as compared with placebo, 0.80; 95 percent confidence interval, 0.72 to 0.90; P<0.001). The second primary outcome — the first primary outcome or refractory ischemia — occurred in 16.5 percent of the patients in the clopidogrel group and 18.8 percent of the patients in the placebo group (relative risk, 0.86, P<0.001). The percentages of patients with in-hospital refractory or severe ischemia, heart failure, and revascularization procedures were also significantly lower with clopidogrel. There were significantly more patients with major bleeding in the clopidogrel group than in the placebo group (3.7 percent vs. 2.7 percent; relative risk, 1.38; P=0.001), but there were not significantly more patients with episodes of life-threatening bleeding (2.1 percent vs. 1.8 percent, P=0.13) or hemorrhagic strokes.

Conclusions The antiplatelet agent clopidogrel has beneficial effects in patients with acute coronary syndromes without ST-segment elevation. However, the risk of major bleeding is increased among patients treated with clopidogrel. (N Engl J Med 2001;345: 494-502.)

THROMBOSIS caused by a ruptured or eroded atherosclerotic plaque is the usual underlying mechanism of acute coronary syndromes.[1] Aspirin and heparin reduce the risk of death from cardiovascular causes, new myocardial infarction, and recurrent ischemia,[2,3] but there is still a substantial risk of such events in both the short term and the long term. Intravenous glycoprotein IIb/IIIa receptor blockers have been shown to reduce the incidence of early events, mainly among patients who are treated according to an invasive strategy,[4,5] but long-term oral therapy with glycoprotein IIb/IIIa receptor blockers is not beneficial and may even increase mortality.[6] Similarly, continuing treatment with low-molecular-weight heparin beyond one week has not been shown to be effective.[7] Although the long-term use of oral anticoagulants may be useful, no convincing evidence of their benefit is yet available.[8] Therefore, there is a need to reduce further the risk of ischemic events in a broad spectrum of patients both when they first present with acute coronary syndromes and in the long term.

The thienopyridine derivatives, ticlopidine and clopidogrel, are antiplatelet agents that inhibit the platelet aggregation induced by adenosine diphosphate, thereby reducing ischemic events.[9] Combining one of these drugs with aspirin, which blocks the thromboxane-mediated pathway, may have an additive effect. In patients who are undergoing percutaneous transluminal coronary angioplasty (PTCA) with stenting, short-term aspirin treatment plus a thienopyridine derivative results in a substantially lower rate of myocardial infarction than does either aspirin alone or warfarin.[10] However, the role of long-term combined therapy with aspirin and an antiplatelet agent in a broader group of patients at high risk for cardiovascular events is unknown. We therefore designed the Clopidogrel in Unstable Angina to Prevent Recurrent Events (CURE) trial to compare the efficacy and safety of the early and long-term use of clopidogrel plus aspirin with those of aspirin alone in patients with acute coronary syndromes and no ST-segment elevation.

METHODS

Study Design

We undertook a randomized, double-blind, placebo-controlled trial comparing clopidogrel with placebo in patients who presented with acute coronary syndromes without ST-segment elevation. The design and rationale of the study have been reported previously.[9]

Study Patients

Patients were eligible for the study if they had been hospitalized within 24 hours after the onset of symptoms and did not have ST-segment elevation. Initially, patients older than 60 years

The Manuscript Writing Committee (Salim Yusuf, D.Phil., F.R.C.P.C., Feng Zhao, M.Sc., Shamir R. Mehta, M.D., F.R.C.P.C., Susan Chrolavicius, B.Sc., Gianni Tognoni, M.D., and Keith K. Fox, M.D., F.R.C.P.) assumes responsibility for the overall content of the manuscript. Address reprint requests to Dr. Yusuf at the Canadian Cardiovascular Collaboration Project Office, Population Health Research Institute, McMaster University, Hamilton General Hospital, 237 Barton St. E., Hamilton, ON L8L 2X2, Canada, or at yusufs@mcmaster.ca.

*The Clopidogrel in Unstable Angina to Prevent Recurrent Events (CURE) trial investigators are listed in the Appendix.

CURE

12.500 pacientes

Disminución de eventos cardiovasculares y ACV

N Engl J Med 2001

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Síndrome coronario agudo sin elevación persistente del segmento ST

Clopidogrel en ACTP

Articles

Effects of pretreatment with clopidogrel and aspirin followed by long-term therapy in patients undergoing percutaneous coronary intervention: the PCI-CURE study

*Shamir R Mehta, Salim Yusuf, Ron J G Peters, Michel E Bertrand, Basil S Lewis, Madhu K Natarajan, Klas Malmberg, Hans-Jürgen Rupprecht, Feng Zhao, Susan Chrolavicius, Ingrid Copland, and Keith A A Fox, for the Clopidogrel in Unstable angina to prevent Recurrent Events trial (CURE) Investigators**

Summary

Background Despite the use of aspirin, there is still a risk of ischaemic events after percutaneous coronary intervention (PCI). We aimed to find out whether, in addition to aspirin, pretreatment with clopidogrel followed by long-term therapy after PCI is superior to a strategy of no pretreatment and short-term therapy for only 4 weeks after PCI.

Methods 2658 patients with non-ST-elevation acute coronary syndrome undergoing PCI in the CURE study had been randomly assigned double-blind treatment with clopidogrel (n=1313) or placebo (n=1345). Patients were pretreated with aspirin and study drug for a median of 6 days before PCI during the initial hospital admission, and for a median of 10 days overall. After PCI, most patients (>80%) in both groups received open-label thienopyridine for about 4 weeks, after which study drug was restarted for a mean of 8 months. The primary endpoint was a composite of cardiovascular death, myocardial infarction, or urgent target-vessel revascularisation within 30 days of PCI. The main analysis was by intention to treat.

Findings There were no drop-outs. 59 (4·5%) patients in the clopidogrel group had the primary endpoint, compared with 86 (6·4%) in the placebo group (relative risk 0·70 [95% CI 0·50–0·97], p=0·03). Long-term administration of clopidogrel after PCI was associated with a lower rate of cardiovascular death, myocardial infarction, or any revascularisation (p=0·03), and of cardiovascular death or myocardial infarction (p=0·047). Overall (including events before and after PCI) there was a 31% reduction cardiovascular death or myocardial infarction (p=0·002). There was less use of glycoprotein IIb/IIIa inhibitor in the clopidogrel group (p=0·001). At follow-up, there was no significant difference in major bleeding between the groups (p=0·64).

Interpretation In patients with acute coronary syndrome receiving aspirin, a strategy of clopidogrel pretreatment followed by long-term therapy is beneficial in reducing major cardiovascular events, compared with placebo.

Lancet 2001; **358:** 527–33
See Commentary page 520

Introduction

Antiplatelet therapy is an important adjunctive treatment that reduces ischaemic complications in patients undergoing percutaneous coronary intervention (PCI).[1–3] Ischaemic events after PCI are mainly the result of a platelet-dependent process that results in thrombosis at the site of mechanical plaque disruption and distal embolisation of platelet thrombi into the coronary microcirculation.[4–9] Although treatment with aspirin before PCI (pretreatment) reduces cardiac events, a substantial risk still remains. Therefore, there is a need to develop more effective antiplatelet strategies that can be given before PCI, with the goal of reducing events after the procedure.

In addition to pretreatment, long-term oral administration of antiplatelet therapy after PCI might also be beneficial because atherothrombosis is a generalised vascular disease that affects not only the target coronary lesion, but also other vascular territories. Despite the beneficial effects of long-term treatment with aspirin after PCI, there remains a significant risk of major cardiovascular events.[10–13] Although the intravenous glycoprotein IIb/IIIa inhibitors improve clinical outcomes when given for a short time,[5,14] longer term oral administration of these agents has not been effective, and might even be harmful.[12,15] Whether other, more effective long-term antiplatelet regimens can add to the beneficial effects of aspirin after PCI is currently unknown.

Clopidogrel is an oral antiplatelet agent of the thienopyridine class, which selectively and irreversibly inhibits the platelet ADP receptor. When clopidogrel is given with aspirin, the antiplatelet effect is synergistic.[16–18] This observation has been confirmed clinically in patients receiving intracoronary stents, in whom ticlopidine plus aspirin given after PCI for about 4 weeks was superior to aspirin alone or aspirin plus oral anticoagulation.[19]

The PCI CURE study was designed to test the hypothesis that, in addition to aspirin, treatment with clopidogrel before PCI is superior to placebo in preventing major ischaemic events afterwards. The second objective of

*For a full list of CURE investigators, please see *Eur Heart J* 2000, **21:** 2033–41. Study slides can be obtained at www.theCureStudy.com

Division of Cardiology, the Population Health Research Institute and the Canadian Cardiovascular Collaboration Project Office, McMaster University, Hamilton, Canada (S R Mehta MD, Prof S Yusuf DPhil, M K Natarajan MD, F Zhao MSc, S Chrolavicius BA, I Copland); **Academic Medical Center, Amsterdam, Netherlands** (R J G Peters MD); **Department of Cardiology, Hôpital Cardiologique, Lille, France** (Prof M E Bertrand MD); **Lady Davis Carmel Hospital, Haifa, Israel** (B S Lewis MD); **Karolinska Hospital, Stockholm, Sweden** (K Malmberg MD); **Klinikum der Johannes-Gutenberg-Universität, Mainz, Germany** (Prof H-J Rupprecht MD); **and Royal Infirmary of Edinburgh, UK** (Prof K A A Fox MBChB)

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PCI-CURE

2.650 pacientes

Disminuicón de eventos cardiovasculares y de necesidad de revascularización posterior

Lancet 2001

somiucam

Clopidogrel: dosis de carga

ISAR-REACT 2: abciximab + clopidogrel (600 mg al menos 2 h antes de ACTP/stent) se asocia a reducción de muerte, IAM o revascularización urgente a los 30 días, en pacientes con SCASEST y elevación de troponina (JAMA, 2006)

Clopidogrel

ESC: dosis de carga de 300 mg, seguida de 75 mg/día. Si cate en menos de 12 h la dosis de carga puede ser 600 mg.

ACC/AHA: dosis de carga y mantenimiento

Mantener clopidogrel durante al menos 1 año

IGP

TACTICS: *Conclusions*

In patients with unstable angina and myocardial infarction without ST-segment elevation who were treated with the glycoprotein IIb/IIIa inhibitor tirofiban, the use of an early invasive strategy significantly reduced the incidence of major cardiac events. These data support a policy involving broader use of the early inhibition of glycoprotein IIb/IIIa in combination with an early invasive strategy in such patients.

N Eng J Med 2001.

TARGET: ***Conclusions***

Although the trial was intended to assess the noninferiority of tirofiban as compared with abciximab, the findings demonstrated that tirofiban offered less protection from major ischemic events than did abciximab.

N Eng J Med 2001

Síndrome coronario agudo sin elevación persistente del segmento ST

IGP

Tirofibán o eptifibatide, como IGP inicial.

Abciximab o eptifibatide si ACTP/stent previstos por anatomía conocida

Chest, 2004

Antithrombotic Therapy for Coronary Artery Disease

The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy

Robert A. Harrington, MD; Richard C. Becker, MD, FCCP; Michal Eisenstein, MD; Theresa W. Meade, DM, FCCP; Christopher M. O'Connor, MD; David A. Verchleitner, MD; and Gordon H. Guyatt, MD, FCCP

This chapter about antithrombotic therapy for coronary artery disease (CAD) is part of the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy: Evidence Based Guidelines. Grade 1 recommendations are strong and indicate that the benefits do, or do not, outweigh risks, burden, and costs. Grade 2 suggests that individual patients' values may lead to different choices (for a full understanding of the grading see Guyatt et al, *CHEST* 2004; 126:179S–187S). Among the key recommendations in this chapter are the following: For patients presenting with non-ST-segment elevation (NSTE) acute coronary syndrome (ACS), we recommend immediate and then daily oral aspirin (Grade 1A). For patients with an aspirin allergy, we recommend immediate treatment with clopidogrel, 300-mg bolus po, followed by 75 mg/d indefinitely (Grade 1A). In all NSTE ACS patients in whom diagnostic catheterization will be delayed or when coronary bypass surgery will not occur until > 5 days, we recommend clopidogrel as bolus therapy (300 mg), followed by 75 mg/d for 9 to 12 months in addition to aspirin (Grade 1A). In NSTE ACS patients in whom angiography will take place within 24 h, we suggest beginning clopidogrel after the coronary anatomy has been determined (Grade 2A). For patients who have received clopidogrel and are scheduled for coronary bypass surgery, we recommend discontinuing clopidogrel for 5 days prior to the scheduled surgery (Grade 2A). In moderate- to high-risk patients presenting with NSTE ACS, we recommend either eptifibatide or tirofiban for initial (early) treatment in addition to treatment with aspirin and heparin (Grade 1A). For the acute treatment of NSTE ACS, we recommend low molecular weight heparins over unfractionated heparin (UFH) [Grade 1B] and UFH over no heparin therapy use with antiplatelet therapies (Grade 1A). We recommend against the direct thrombin inhibitors as routine initial antithrombin therapy (Grade 1B). For patients after myocardial infarction, after ACS, and with stable CAD, we recommend aspirin in doses from 75 to 325 mg as initial therapy and in doses of 75 to 162 mg as indefinite therapy (Grade 1A). For patients with contraindications to aspirin, we recommend long-term clopidogrel (Grade 1A). For primary prevention in patients with at least moderate risk for a coronary event, we recommend aspirin, 75 to 162 mg/d, over either no antithrombotic therapy or vitamin K antagonist (VKA) [Grade 2A]; for patients at particularly high risk of events in whom the international normalized ratio (INR) can be monitored without difficulty, we suggest low-dose VKA (target INR, 1.5) [Grade 2A].

(*CHEST* 2004; *126*:513S–548S)

Key words: acute coronary syndrome; antithrombotic; aspirin; clopidogrel; coronary artery disease; prophylaxis

Abbreviations: ACC = American College of Cardiology; ACS = acute coronary syndrome; ACEI = angiotensin-converting enzyme inhibitors; ACT = activated clotting time; ADP = adenosine diphosphate; AHA = American Heart Association; AMI = acute myocardial infarction; APRICOT = Antithrombotics in the Prevention of Reocclusion in Coronary Thrombolysis; aPTT = activated partial thromboplastin time; ASPECT = Anticoagulation in the Secondary Prevention of Events in Coronary Thrombosis; CABG = coronary artery bypass grafting; CAD = coronary artery disease; CAPRIE = Clopidogrel vs Aspirin in Patients at Risk of Ischemic Events; CARS = Coumadin Aspirin Reinfarction Study; CHF = congestive heart failure; CI = confidence interval; CrCl = creatinine clearance; CRP = C-reactive protein; CURE = Clopidogrel in Unstable Angina to Prevent Recurrent Events; DTI = direct thrombin inhibitor; ESSENCE = Efficacy and Safety of Subcutaneous Enoxaparin in Non-Q-Wave Coronary Events; ESTREM = Efficacy and Safety of the Oral Direct Thrombin Inhibitor Ximelagatran in Patients With Recent Myocardial Damage; FRIC = Fragmin in Unstable Coronary Artery Disease; FRISC = Fragmin During Instability in Coronary Artery Disease; GP = glycoprotein; GUSTO = Global Use of Strategies to Open Occluded Coronary Arteries; HIT = heparin-induced thrombocytopenia; HOT = Hypertension Optimal Treatment; IHD = ischemic heart disease; INR = international normalized ratio; ISIS = International Studies of Infarct Survival; LMWH = low-molecular-weight heparin; MI = myocardial infarction; NS = not significant; NSTE = non-ST-segment elevation; OASIS = Organization to Assess Strategies for Ischemic Syndromes; OR = odds ratio; PARAGON = Platelet IIb/IIIa Antagonism for the Reduction of Acute Coronary Syndrome Events in a Global Organization Network; PCI = percutaneous coronary intervention; PRISM = Platelet Receptor Inhibition in Ischemic Syndrome Management; PRISM-PLUS = Platelet Receptor Inhibition in Ischemic Syndrome Management in Patients Limited by Unstable Signs and Symptoms; PURSUIT = Platelet Glycoprotein IIb/IIIa in Unstable Angina: Receptor Suppression Using Integrilin Therapy; RCT = randomized controlled trial; RR = relative risk; RRR = relative risk reduction; SC = subcutaneous; TIA = transient ischemic attack; TIMI = Thrombolysis in Myocardial Infarction; TPT = Thrombosis Prevention Trial; UA = unstable angina; UFH = unfractionated heparin; VKA = vitamin K antagonist; WARCEF = Warfarin Versus Aspirin in Reduced Cardiac Ejection Fraction; WARIS = Warfarin-Aspirin Reinfarction Study; WASH = Warfarin/Aspirin Study in Heart Failure; WATCH = Warfarin Antiplatelet Trial and Chronic Heart Failure

Antithrombotic therapies, both antiplatelet as well as anticoagulant, have become the mainstays of treatment for coronary artery diseases (CADs). Based on the important role of thrombosis in the pathogenesis and complications of the atherosclerotic process, antithrombotic therapy has become essential treatment for both

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somiucam

IGP

ESC:

Tirofibán o eptifibatide como tratamiento inicial. Estos IGP deben mantenerse durante y después de la ACTP/stent.

Cuando no se han empleado los anteriores o cuando al anatomía coronaria es conocida y se va a realizar ACTP/stent, iniciar abciximab.

AHA/ACC:

Abciximab si la angiografía es inmediata o ACTP/stent es previsible; en caso contrario, tirofibán o eptifibatide.

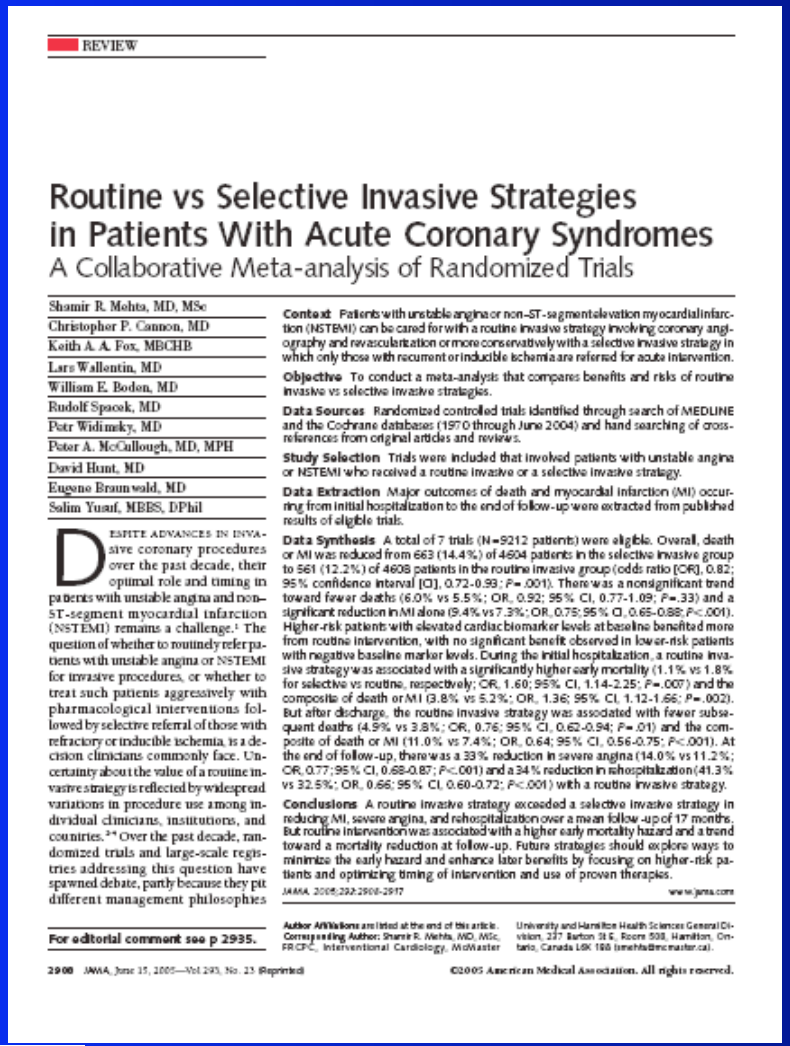
Dosis antitrombóticos

TABLA 6. **Uso clínico del tratamiento antitrombótico**

Tratamiento antiplaquetario oral	Dosis inicial de aspirina: 160-325 mg formulación no entérica, seguida por 75-100 mg diarios
	Clopidogrel 75 mg/día después de una dosis de carga de 300 mg (600 mg cuando se quiere tener un inicio rápido de su acción)
Anticoagulantes	Fondaparinux* 2,5 mg/día subcutáneo
	Enoxaparina*, 1 mg/kg/12 h subcutánea
	Dalteparina*, 120 U/kg/12 h
	Nadroparina*, 86 U/kg/12 h
	HNF, bolo intravenoso 60-70 U/kg (máximo 5.000 UI) seguido por infusión de 12-15 U/kg/h (máximo 1.000 U/h) titulado a un TTPa 1,5-2,5 veces el control
	Bivalirudina*, bolo intravenoso de 0,1 mg/kg e infusión de 0,25 mg/kg/h. Bolos intravenosos adicionales de 0,5 mg/kg e infusión aumentada a 1,75 mg/kg/h antes de la ICP
Inhibición de la GP IIb/IIIa*	Abciximab, 0,25 mg/kg bolo intravenoso seguido de infusión de 0,125 µg/kg/min (máximo 10 µg/min) durante 12-24 h
	Eptifibatida, 180 µg/kg bolo intravenoso (segundo bolo a los 10 min para ICP) seguido de infusión de 2 µg/kg/min durante 72-96 h
	Tirofibán, 0,4 µg/kg/min intravenoso durante 30 min seguido de infusión de 0,10 µg/kg/min durante 48-96 h. Algunos estudios clínicos están probando un régimen de dosis más altas (bolo de 25 µg/kg e infusión de 0,15 µg/kg/min durante 18 h)

HNF: heparina no fraccionada; TTPa: tiempo de tromboplastina parcial activada.

Intervencionismo coronario



REVIEW

Routine vs Selective Invasive Strategies in Patients With Acute Coronary Syndromes

A Collaborative Meta-analysis of Randomized Trials

Shamir R. Mehta, MD, MSc
Christopher P. Cannon, MD
Keith A. A. Fox, MBCHB
Lars Wallentin, MD
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Rudolf Spacek, MD
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Peter A. McCullough, MD, MPH
David Hunt, MD
Eugene Braunwald, MD
Salim Yusuf, MBBS, DPhil

Context Patients with unstable angina or non–ST-segment elevation myocardial infarction (NSTEMI) can be cared for with a routine invasive strategy involving coronary angiography and revascularization or more conservatively with a selective invasive strategy in which only those with recurrent or inducible ischemia are referred for acute intervention.

Objective To conduct a meta-analysis that compares benefits and risks of routine invasive vs selective invasive strategies.

Data Sources Randomized controlled trials identified through search of MEDLINE and the Cochrane databases (1970 through June 2004) and hand searching of cross-references from original articles and reviews.

Study Selection Trials were included that involved patients with unstable angina or NSTEMI who received a routine invasive or a selective invasive strategy.

Data Extraction Major outcomes of death and myocardial infarction (MI) occurring from initial hospitalization to the end of follow-up were extracted from published results of eligible trials.

Data Synthesis A total of 7 trials (N=9212 patients) were eligible. Overall, death or MI was reduced from 663 (14.4%) of 4604 patients in the selective invasive group to 561 (12.2%) of 4608 patients in the routine invasive group (odds ratio [OR], 0.82; 95% confidence interval [CI], 0.72-0.93; *P*=.001). There was a nonsignificant trend toward fewer deaths (6.0% vs 5.5%; OR, 0.92; 95% CI, 0.77-1.09; *P*=.33) and a significant reduction in MI alone (9.4% vs 7.3%; OR, 0.75; 95% CI, 0.65-0.88; *P*<.001). Higher-risk patients with elevated cardiac biomarker levels at baseline benefited more from routine intervention, with no significant benefit observed in lower-risk patients with negative baseline marker levels. During the initial hospitalization, a routine invasive strategy was associated with a significantly higher early mortality (1.1% vs 1.8% for selective vs routine, respectively; OR, 1.60; 95% CI, 1.14-2.25; *P*=.007) and the composite of death or MI (3.8% vs 5.2%; OR, 1.36; 95% CI, 1.12-1.66; *P*=.002). But after discharge, the routine invasive strategy was associated with fewer subsequent deaths (4.9% vs 3.8%; OR, 0.76; 95% CI, 0.62-0.94; *P*=.01) and the composite of death or MI (11.0% vs 7.4%; OR, 0.64; 95% CI, 0.56-0.75; *P*<.001). At the end of follow-up, there was a 33% reduction in severe angina (14.0% vs 11.2%; OR, 0.77; 95% CI, 0.68-0.87; *P*<.001) and a 34% reduction in rehospitalization (41.3% vs 32.5%; OR, 0.66; 95% CI, 0.60-0.72; *P*<.001) with a routine invasive strategy.

Conclusions A routine invasive strategy exceeded a selective invasive strategy in reducing MI, severe angina, and rehospitalization over a mean follow-up of 17 months. But routine intervention was associated with a higher early mortality hazard and a trend toward a mortality reduction at follow-up. Future strategies should explore ways to minimize the early hazard and enhance later benefits by focusing on higher-risk patients and optimizing timing of intervention and use of proven therapies.

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For editorial comment see p 2935.

Con intervencionismo precoz:

1. Reducción mortalidad no significativa a los 17 meses
2. Reducción significativa IAM
3. Aumento mortalidad e IAM durante hospitalización inicial
4. Disminución de angina y rehospitalización

JAMA, 2005

Intervencionismo coronario

Articles

5-year outcome of an interventional strategy in non-ST-elevation acute coronary syndrome: the British Heart Foundation RITA 3 randomised trial

K A A Fox, P Poole-Wilson, T C Clayton, R A Henderson, T R D Shaw, D J Wheatley, R Knight, S J Pocock

Summary

Background The long-term outcome of an interventional strategy in patients with non-ST-elevation acute coronary syndrome is unknown. We tested whether an interventional strategy (routine angiography followed by revascularisation) was better than a conservative strategy (ischaemia-driven or symptom-driven angiography) over 5 years' follow-up.

Methods In a multicentre randomised trial, 1810 patients (from 45 hospitals in England and Scotland, UK) with non-ST-elevation acute coronary syndrome were randomly assigned to receive an early intervention (n=895) or a conservative strategy (n=915) within 48 h of the index episode of cardiac pain. In each group, the aim was to provide the best medical treatment, and also to undertake coronary arteriography within 72 h in the interventional strategy with subsequent management guided by the angiographic findings. Analysis was by intention to treat and the primary outcome (composite of death or non-fatal myocardial infarction) had masked independent adjudication. RITA 3 has been assigned the International Standard Randomised Control Trial Number IS RCTN07752711.

Findings At 1-year follow-up, rates of death or non-fatal myocardial infarction were similar. However, at a median of 5 years' follow-up (IQR 4·6–5·0), 142 (16·6%) patients with intervention treatment and 178 (20·0%) with conservative treatment died or had non-fatal myocardial infarction (odds ratio 0·78, 95% CI 0·61–0·99, p=0·044), with a similar benefit for cardiovascular death or myocardial infarction (0·74, 0·56–0·97, p=0·030). 234 (102 [12%] intervention, 132 [15%] conservative) patients died during follow-up (0·76, 0·58–1·00, p=0·054). The benefits of an intervention strategy were mainly seen in patients at high risk of death or myocardial infarction (p=0·004), and for the highest risk group, the odds ratio of death or non-fatal myocardial infarction was 0·44 (0·25–0·76).

Interpretation In patients with non-ST-elevation acute coronary syndrome, a routine invasive strategy leads to long-term reduction in risk of death or non-fatal myocardial infarction, and this benefit is mainly in high-risk patients. The findings provide support for national and international guidelines in the need for more robust risk stratification in acute coronary syndrome.

Introduction

In patients that present with acute coronary syndrome without ST-segment elevation, the pathophysiological mechanisms include plaque disruption, thrombus formation, and microembolisation.[1] Aneplatelet and antithrombin therapy affect acute and subsequent outcome, but the long-term effect of an interventional strategy is currently unknown. The rationale for an interventional strategy, in addition to a pharmacological approach for non-ST-elevation acute coronary syndrome, is based on evidence indicating that disrupted architecture of the plaque contributes to ischaemia and progression of coronary disease. Revascularisation procedures (percutaneous coronary intervention [PCI] and coronary artery bypass grafting surgery [CABG]) improve coronary perfusion, reduce ischaemia, and could reduce the likelihood of presentation with a further coronary event.

Meta-analysis of all the published trials of a selective strategy versus a routine invasive strategy in acute coronary syndrome suggests a net reduction in risk of death or myocardial infarction with a routine interventional strategy (odds ratio 0·82, 95% CI 0·72–0·93).[2] However, there was an early net increase in the risk of death during the index hospital admission (1·60, 1·14–2·25, p=0·007). The defining of long-term outcomes in patients with acute coronary syndrome is of critical importance. Experience from trials of surgical revascularisation in patients with stable coronary heart disease shows that the early hazards of surgery are outweighed by benefits that emerge after 2 to 5 years of follow-up.[3]

The RITA 3 study was designed to investigate whether a strategy of early angiography and revascularisation (as clinically indicated) is more effective than a conservative strategy in patients with non-ST-elevation acute coronary syndrome.[4] At 4 months, 10% of patients in the intervention group had died, had a myocardial infarction, or had an episode of refractory angina compared with 15% in the conservative group (risk ratio 0·66, 95% CI 0·51–0·85, p=0·001). This difference was mainly due to a halving of the frequency of refractory angina in the intervention group. Rates of death or non-fatal myocardial infarction were similar in both treatment groups at 1 year (7·6% [intervention] vs 8·3%

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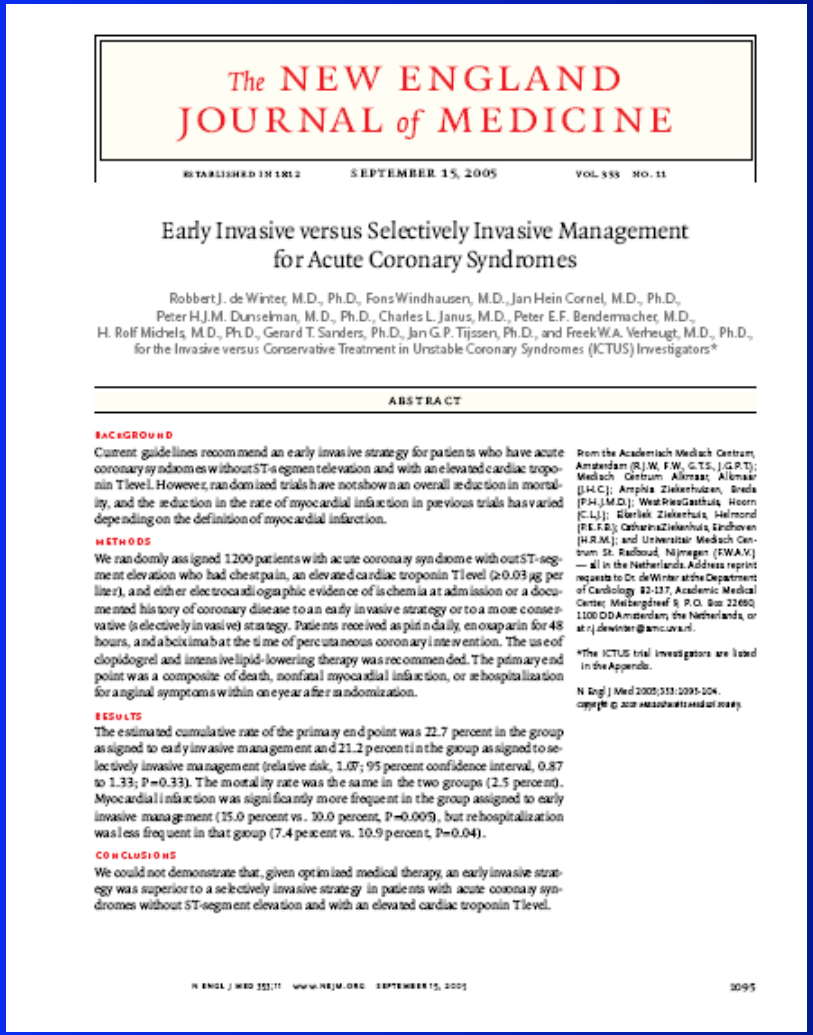
In patients with non-ST-elevation acute coronary syndrome, a routine invasive strategy leads to longterm reduction in risk of death or non-fatal myocardial infarction, and this benefit is mainly in high-risk patients.

Lancet 2005

Intervencionismo coronario



The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 SEPTEMBER 15, 2005 VOL. 353 NO. 11

Early Invasive versus Selectively Invasive Management for Acute Coronary Syndromes

Robbert J. de Winter, M.D., Ph.D., Fons Windhausen, M.D., Jan Hein Cornel, M.D., Ph.D., Peter H.J.M. Dunselman, M.D., Ph.D., Charles L. Janus, M.D., Peter E.F. Bendermacher, M.D., H. Rolf Michels, M.D., Ph.D., Gerard T. Sanders, Ph.D., Jan G.P. Tijssen, Ph.D., and Freek W.A. Verheugt, M.D., Ph.D., for the Invasive versus Conservative Treatment in Unstable Coronary Syndromes (ICTUS) Investigators*

ABSTRACT

BACKGROUND

Current guidelines recommend an early invasive strategy for patients who have acute coronary syndromes without ST-segment elevation and with an elevated cardiac troponin T level. However, randomized trials have not shown an overall reduction in mortality, and the reduction in the rate of myocardial infarction in previous trials has varied depending on the definition of myocardial infarction.

METHODS

We randomly assigned 1200 patients with acute coronary syndrome without ST-segment elevation who had chest pain, an elevated cardiac troponin T level (≥0.03 μg per liter), and either electrocardiographic evidence of ischemia at admission or a documented history of coronary disease to an early invasive strategy or to a more conservative (selectively invasive) strategy. Patients received aspirin daily, enoxaparin for 48 hours, and abciximab at the time of percutaneous coronary intervention. The use of clopidogrel and intensive lipid-lowering therapy was recommended. The primary end point was a composite of death, nonfatal myocardial infarction, or rehospitalization for anginal symptoms within one year after randomization.

RESULTS

The estimated cumulative rate of the primary end point was 22.7 percent in the group assigned to early invasive management and 21.2 percent in the group assigned to selectively invasive management (relative risk, 1.07; 95 percent confidence interval, 0.87 to 1.33; P=0.33). The mortality rate was the same in the two groups (2.5 percent). Myocardial infarction was significantly more frequent in the group assigned to early invasive management (15.0 percent vs. 10.0 percent, P=0.009), but rehospitalization was less frequent in that group (7.4 percent vs. 10.9 percent, P=0.04).

CONCLUSIONS

We could not demonstrate that, given optimized medical therapy, an early invasive strategy was superior to a selectively invasive strategy in patients with acute coronary syndromes without ST-segment elevation and with an elevated cardiac troponin T level.

From the Academisch Medisch Centrum, Amsterdam (R.J.W., F.W., G.T.S., J.G.P.T.); Medisch Centrum Alkmaar, Alkmaar (J.H.C.); Amphia Ziekenhuizen, Breda (P.H.J.M.D.); West Friesgasthuis, Hoorn (C.L.J.); Elkerliek Ziekenhuis, Helmond (P.E.F.B.); Catharina Ziekenhuis, Eindhoven (H.R.M.); and Universitair Medisch Centrum St. Radboud, Nijmegen (F.W.A.V.) — all in the Netherlands. Address reprint requests to Dr. de Winter at the Department of Cardiology B2-137, Academic Medical Center, Meibergdreef 9, P.O. Box 22660, 1100 DD Amsterdam, the Netherlands, or at r.j.dewinter@amc.uva.nl.

*The ICTUS trial investigators are listed in the Appendix.

N Engl J Med 2005;353:1095-104.

We could not demonstrate that, given optimized medical therapy, an early invasive strategy was superior to a selectively invasive strategy in patients with acute coronary syndromes without ST-segment elevation and with an elevated cardiac troponin T level.

N Engl J Med, 2005.

Intervencionismo coronario

ESC:
La angiografía coronaria debe planificarse lo antes posible (estrategia invasiva urgente) en pacientes con angina severa, cambios importantes en el ECG o dinámicos, arritmias mayores o inestabilidad hemodinámica en el momento del ingreso o después. Estos pacientes son un 2-15% de los ingresados con SCASEST. En pacientes con características de riesgo intermedio a alto pero sin las características antes mencionadas y con riesgo vital, se han probado como estrategias alternativas la angiografía coronaria precoz (en las primeras 72 h) seguida de revascularización cuando sea posible y esté indicado, o la estabilización médica inicial y la realización selectiva de angiografía coronaria basada en el curso clínico.
En pacientes de bajo riesgo, la evaluación no invasiva de isquemia inducible debe realizarse antes del alta. Si es positiva, se debe realizar una angiografía coronaria

Intervencionismo coronario

AHA/ACC
CLASS I
1. An early invasive strategy (i.e., diagnostic angiography with intent to perform revascularization) is indicated in UA/NSTEMI patients who have refractory angina or hemodynamic or electrical instability (without serious comorbidities or contraindications to such procedures). *(Level of Evidence: B)*
2. An early invasive strategy (i.e., diagnostic angiography with intent to perform revascularization) is indicated in initially stabilized UA/NSTEMI patients (without serious comorbidities or contraindications to such procedures) who have an elevated risk for clinical events *(Level of Evidence: A)*

Intervencionismo coronario

AHA/ACC

Table 5. Selection of Initial Treatment Strategy: Invasive Versus Conservative Strategy

Preferred Strategy	Patient Characteristics
Invasive	Recurrent angina or ischemia at rest or with low-level activities despite intensive medical therapy
	Elevated cardiac biomarkers (TnT or TnI)
	New or presumably new ST-segment depression
	Signs or symptoms of HF or new or worsening mitral regurgitation
	High-risk findings from noninvasive testing
	Hemodynamic instability
	Sustained ventricular tachycardia
	PCI within 6 months
	Prior CABG
	High risk score (e.g., TIMI, GRACE)
	Reduced left ventricular function (LVEF less than 40%)
Conservative	Low risk score (e.g., TIMI, GRACE)
	Patient or physician preference in the absence of high-risk features

CABG = coronary artery bypass graft surgery; GRACE = Global Registry of Acute Coronary Events; HF = heart failure; LVEF = left ventricular ejection fraction; PCI = percutaneous coronary intervention; TIMI = Thrombolysis In Myocardial Infarction; TnI = troponin I; TnT = troponin T.

Intervencionismo coronario

URGENTE:

1. Angina refractaria
2. Inestabilidad hemodinámica
3. Inestabilidad eléctrica: arritmias ventriculares graves

PRECOZ (< 72 h)

Pacientes de riesgo alto o intermedio sin las anteriores características

Síndrome coronario agudo sin elevació persistente del segmento ST

Errores frecuentes

Considerar que no se puede tratar al paciente si no existe laboratorio de hemodinámica en el hospital

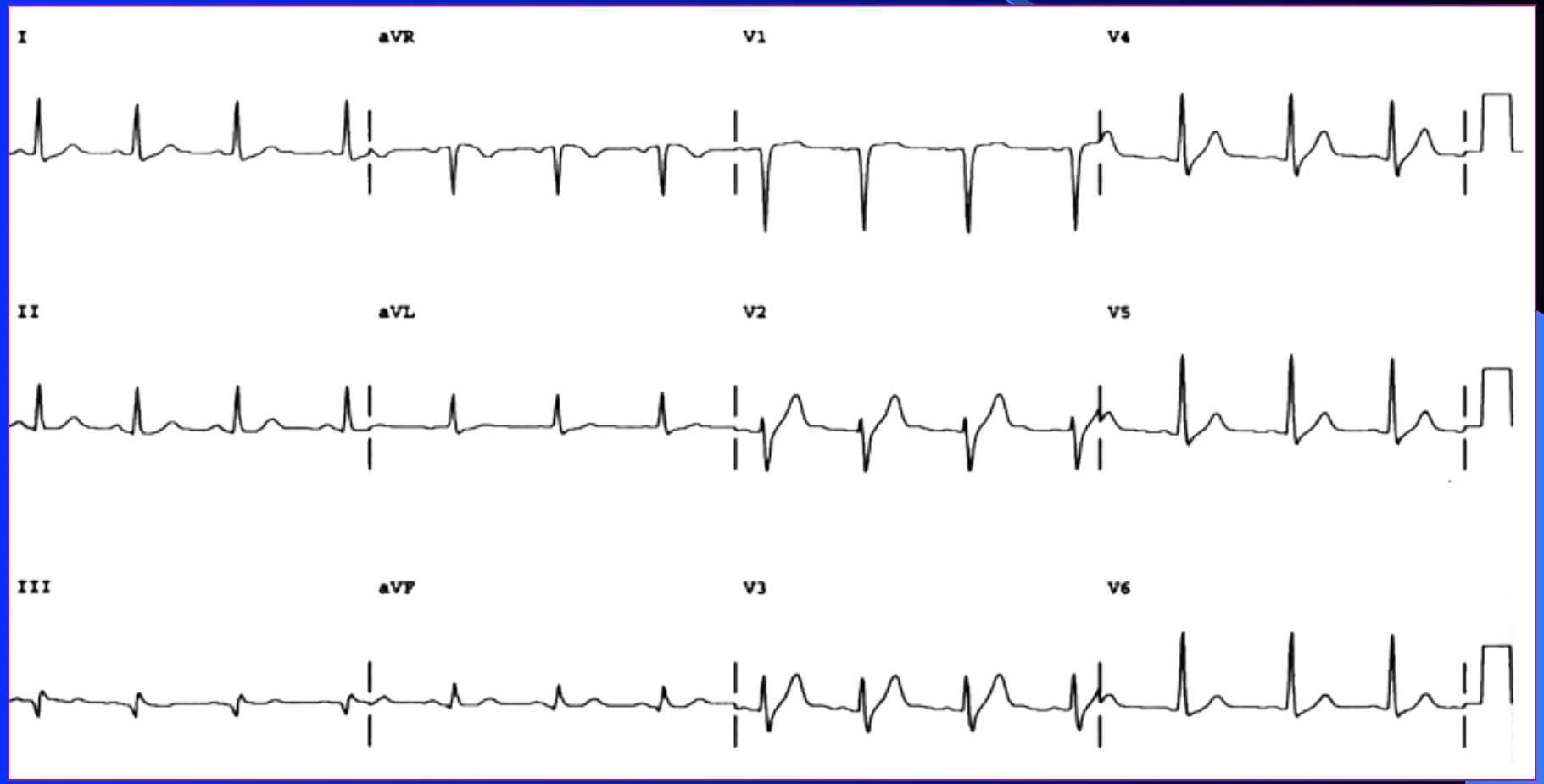
Esperar a los marcadores de daño miocárdico sin valorar el riesgo del paciente por otros parámetros

Retrasar la administración de betabloqueantes

Mantener nitratos sin indicación

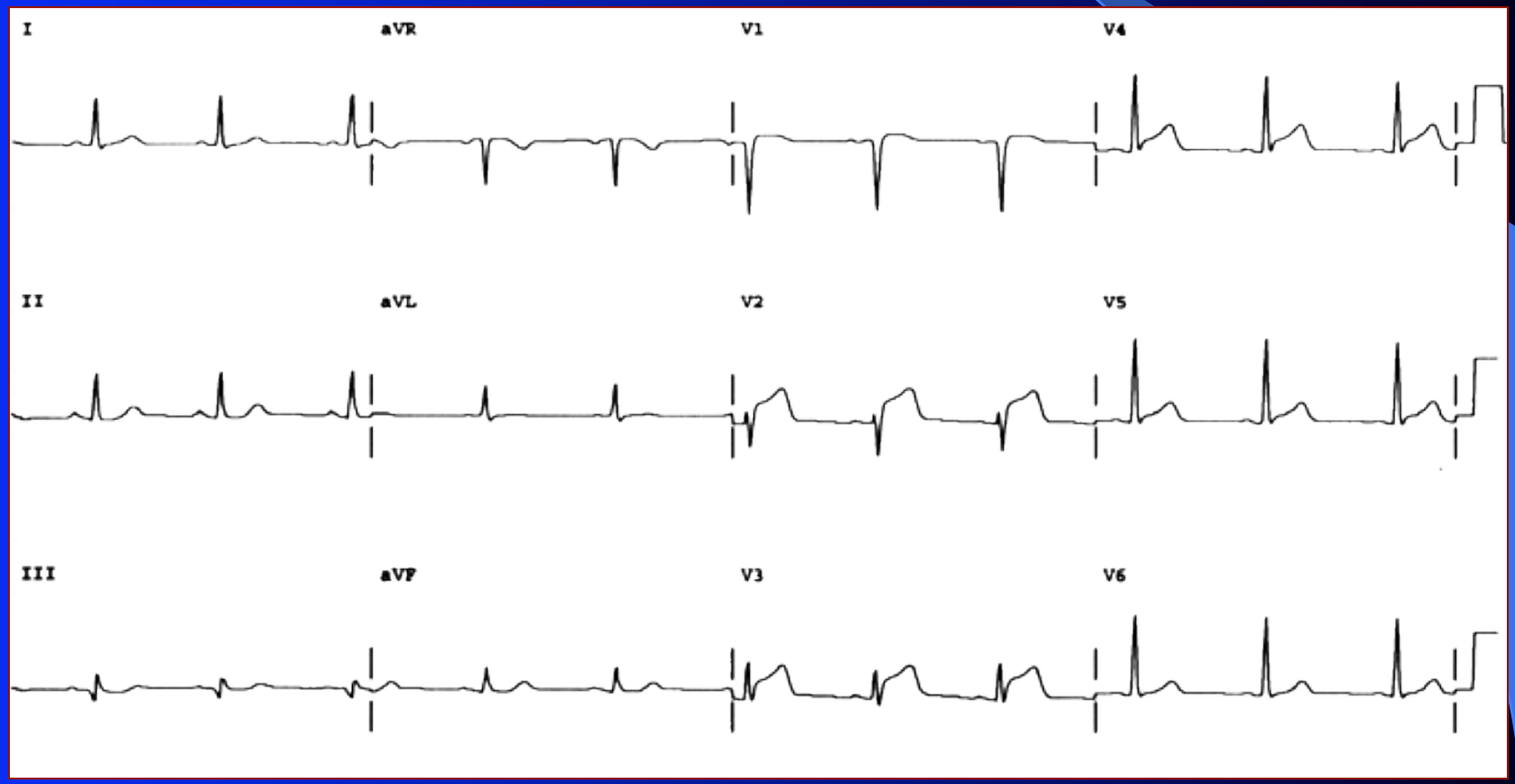
Síndrome coronario agudo SIN elevación persitente del segmento ST

SIN



Síndrome coronario agudo CON elevación del segmento ST

CON



Síndrome coronario agudo con elevación del segmento ST

1er FORO DE RESIDENTES DE MEDICINA INTENSIVA DE CASTILLA LA MANCHA

SINDROME CORONARIO AGUDO

Dr Rafael Blancas. Hospital del Tajo

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

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2007 Focused update of the ACC/AHA 2004 Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

Elliott M. Antman, Mary Hand, Paul W. Armstrong, Eric R. Bates, Lee A. Green, Lakshmi K. Halasyamani, Judith S. Hochman, Harlan M. Krumholz, Gervasio A. Lamas, Charles J. Mullany, David L. Pearle, Michael A. Sloan and Sidney C. Smith, Jr

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ractice Guidelines

e Management of Patients With
nfarction—Executive Summary
llege of Cardiology/American Heart
ice Guidelines (Writing Committee to
e Management of Patients With Acute
ial Infarction)
the Canadian Cardiovascular Society

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SOCIEDAD DE MEDICINA INTENSIVA Y UNIDADES CORONARIAS DE CASTILLA-LA MANCHA

Síndrome coronario agudo con elevación del segmento ST

CRITERIOS ELECTROCARDIOGRÁFICOS

1. **Elevación del segmento ST (0,1 mV) en dos o más derivaciones contiguas, durante al menos 20 minutos.**
2. **Aparición de nuevo BRIHH.**

Síndrome coronario agudo con elevación del segmento ST

Medidas iniciales

O2

2 Vías periféricas

ECG 12 derivaciones y V3R, V4R, V7, V8

Analítica: hemograma, bioquímica general, coagulación completa, marcadores enzimáticos.

¡Evitar gasometría arterial!

Exploración: pulsos, masas abdominales pulsátiles, soplos diastólicos, roces pericárdicos

Rx tórax: datos de sospecha de disección aórtica

Tratamiento farmacológico (Urgencias-UCI)

- **Antiagregación (AAS y clopidogrel)**
- **NTG sl/iv**
- **Analgesia**
- **Reperfusión**
- **Betabloqueantes**
- **IECA/ARA2**

Síndrome coronario agudo con elevación del segmento ST

NITROGLICERINA

Class I

1. Los pacientes con dolor isquémico deben recibir NTG sl. (0.4 mg) cada 5 minutos hasta un total de tres dosis. Posteriormente se empleará IV. (Nivel de evidencia C)

2. NTG IV está indicada para control de dolor, control de la HTA o IC (Nivel de evidencia C)

Class III

1. No debe administrarse nitratos si:
   - TA < 90 mm de Hg o 30 mm de Hg por debajo de su TA basal
   - Bradicardia severa (<5 lpm)/ taquicardia (> 100 lpm)
   - Sospecha de IAM de VD

(Nivel de evidencia C)

2. No deben administrarse nitratos en paciente que hayan tomado inhibidores de la fosfodiesterasa en las últimas 24 h (48 h si se trata de tadalafil).

(Nivel de evidencia B)

Síndrome coronario agudo con elevación del segmento ST

ANALGESIA

MORFINA

Clase I

- Sigue siendo el tto de elección (aunque en el SCASEST ha descendido a la *Clase IIa*) Nivel de evidencia C
- 2- 4 mg que se puede aumentar a 2-8 mg cada 5-15 minutos

AINES

Clase I

- Suspender el tto con AINES, excepto la aspirina, de todos aquellos que lo estén tomando en el momento del IAM (tanto de los inhibidores selectivos de la COX-2 como los no selectivos)

(Nivel de evidencia C)

Clase III

No administrar AINES, excepto la aspirina, durante la hospitalización (tanto de los inhibidores selectivos de la COX-2 como los no selectivos)

(Nivel de evidencia C)

Síndrome coronario agudo con elevación del segmento ST

ANTIAGREGACIÓN

-AAS

Clase I

- Se debe administrar a todos los pacientes que no lo tomaran previo al IAM
- La dosis inicial debe ser de 162 mg (Nivel de evidencia A) a 325 mg (N. de evidencia C)
- Las formulaciones con protectores entéricos retrasan la absorción.
-Posteriormente se mantendrá un tto indefinido con 75-162 mg /día orales (N. de evidencia A)

- THIANOPIRIDINAS (Clopidogrel)

CLARITY-TIMI 28 Y COMMIT

Clase I

- 75 mg al día + AAS +/- Fibrinolisis
- 14 días
- si Qx suspenderlo> 5 días

Clase IIa

- < 75 años dar dosis de carga de 300 mg
- Dar 75 mg/día durante un año.

Síndrome coronario agudo con elevación del segmento ST

BETABLOQUEANTES

Clase I

1. De forma oral, deben comenzarse tan pronto como sea posible, en ausencia de contraindicaciones:

   - IC

   - Bajo gasto, alto riesgo de shock cardiogénico (> 70 años, TAS <120, TQ>120 o BQ <60 lpm o llevar mucho tiempo sintomático)

2. Reevaluar tras pasar 24 h

Class IIa

1. Es razonable emplearlos de forma iv inicialmente, en pacientes sin contraindicaciones, especialmente si existe taquiarritmia o hipertensión.

Síndrome coronario agudo con elevación del segmento ST

REPERFUSIÓN

-Los pacientes con síntomas característicos de IAM de < 12 h de duración deben ser evaluados de forma rápida , tras su primer contacto con el sistema sanitario, para saber cual es la estrategia de reperfusión más adecuada. (Nivel de evidencia A):

- Se realizará angioplastia primaria a todos aquellos SCACEST si es viable realizarla dentro de los primeros 90 minutos tras el primer contacto sanitario.

- Si se trata de un hospital sin servicio de hemodinámica o no puede ser trasladado a otro hospital que disponga de éste, dentro de los primeros 90 minutos tras el primer contacto sanitario, se realizárá tratamiento fibrinolítico preferíblemente en los primeros 30 minutos) si no está contraindicado

Síndrome coronario agudo con elevación del segmento ST

FIBRINOLISIS

Clase I

1. En pacientes con síntomas de menos de 12 h de duración, se administrarán fibrinolíticos si presentan elevación de ST de al menos 0.1 mV en dos derivaciones contiguas (precordiales) o adyacentes (derivaciones de las extremidades). (Nivel de evidencia*: A*)
2. En pacientes con síntomas de menos de 12 h de duración, se administrarán fibrinolíticos si presentan un nuevo BRIHH. (Nivel de evidencia*: A*)

Clase IIa

1. Es razonable administrar fibrinolíticos a pacientes con síntomas de menos de 12 h de duración y hallazgos en el ecg compatibles con un verdadero IAM posterior. (Nivel de evidencia*: C*)
2. Es razonable administrar fibrinolíticos a pacientes con síntomas de 12 a 24 horas de duración, con síntomas persistentes de isquemia y elevación de ST de al menos 0.1 mV en dos derivaciones contiguas o adyacentes. (Nivel de evidencia*: B*)

Clase III

1. Los fibrinolíticos no deben administrarse a pacientes cuyos síntomas empezaron más de 24 h antes ni a aquellos que presentan únicamente descenso de ST (salvo IAM posterior).

CONTRAINDICACIONES DE LA FIBRINOLISIS

Absolutas

Cualquier sangrado craneal previo

Lesiones vasculares intracraneales

Neoplasias intracraneales

ACV isquémico en los 3 meses previos (salvo los ocurridos en las 3 h previas)

Disección aórtica

Traumatismo facial o craneal significativo en los 3 meses anteriores

Relativas

Historia de HTA mal controlada

TAs>180 ó TAd>110

ACV previo hace más de 3 meses, demencia u otra patología intracraneal

RCP de más de 10 minutos

Cirugía mayor en las 3 semanas previas

Sangrado en las últimas 2-4 semanas

Punciones vasculares no compresibles

Embarazo

Ulcera péptica activa

Uso de anticoagulantes: a mayor INR mayor riesgo de sangrado.

Empleo de estreptoquinasa/anistreplasa hace más de 5 días o alergia a los mismos (emplear tenecteplasa)

1er FORO DE RESIDENTES DE MEDICINA INTENSIVA DE CASTILLA LA MANCHA
SINDROME CORONARIO AGUDO
Dr Rafael Blancas. Hospital del Tajo

FIBRINOLISIS PREHOSPITALARIA

1. Tiempo de traslado al hospital de referencia mayor de 1 hora
2. Posibilidad de comunicación con la Unidad Coronaria de referencia
3. Personal médico entrenado específicamente en el manejo del enfermo coronario agudo
4. Posibilidad de transmisión de ECG a la Unidad Coronaria de referencia
5. Establecimiento de protocolo de actuación instituido por la Unidad Coronaria de referencia y aprobado por la Institución pertinente

Síndrome coronario agudo con elevación del segmento ST

ANGIOPLASTIA DE RESCATE

1. *Case I*
   - <75 años con shock cardiogénico y susceptibles de revascularización
   - IC severa (Killip III)
   - Arritmias ventriculares con compromiso hemodinámico
2. *Clase II a*
   - > 75 años con shock cardiogénico
   - Inestabilidad hemodinámica o eléctrica y/o isquemia persistente (2004)
   - Fallo de fibrinolisis + área de alto riesgo

Síndrome coronario agudo con elevación del segmento ST
ANTICOAGULANTES

- ***Clase I*** **(>48 h tas la fibrinolisis y preferiblemente *8 días)***

1. - Tras fibrinolisis

Durante un periodo >48 h (Nivel de evidencia C), preferiblemente durante la hospitalización hasta 8 días (Es recomendable no utilizar durante tanto tiempo Heparina no fraccionada por el riesgo de inducir trombopenia)

A - Heparina no fraccionada.

- bolo 60 U/kg (maximo 4000 U) seguido de infusión de 12 U/kg/h (maximo 1000 U/h) ajustando el tiempo de cefalina a 1.5 a 2.0 veces el control (50 a 70 segundos).

*** (G. 2004) Los pacientes que reciben :**

- alteplasa, reteplasa, or tenecteplasa

- Fibrinolíticos no selectivos (estreptoquinasa, anistreplasa, o uroquinasa) en riesgo de embolia sistémica (IAM anterior, FA, embolismo previo, o trombo intraventricular conocido).

- Fibrinolíticos no selectivos sin riesgo de embolia *(ClaseIIb)*

Síndrome coronario agudo con elevación del segmento ST
ANTICOAGULANTES

- ***Clase I***

B. Enoxaparina

- En pacientes con menos de 75 años, sin disfunción renal significativa (creatinina mayor de 2.5 mg/dL en hombres o 2.0 mg/dl en mujeres). 30 mg IV bolo seguida de 1.0 mg/kg subcutánea cada 12 h en combinación con tenecteplasa. (Nivel de evidencia A)

- > 75 años: no dar bolo y luego dar 0,75 mg/Kg/12h

- CCr <30 ml/min dar 1 mg/24 h

- 8 días

***C.* Fondaparinux**

- Si Cr < 3 mg/dl dar bolo IV 2,5 mg y luego 2,5 mg/sc/24 h

- 8 días

Síndrome coronario agudo con elevación del segmento ST

INHIBICIÓN DEL SISTEMA RENINA-ANGIOTENSINA-ALDOSTERONA

Inhibición del sistema renina-angiotensina-aldosterona

Clase I

1. Administrar un IECA en las primeras 24 h a pacientes con IAM anterior, IC izda o FE<40% en ausencia de hipotensión (TAs < 100 mmHg o menos de 30 mmHg de su TAs habitual)

2. Administrar ARA 2 en las mismas circunstancias, si existe intolerancia a IECAs. Valsartan y candesartan han demostrado su eficacia en esta recomendación .

Clase IIa

1. Los IECAs pueden ser beneficiosos en ausencias de las anteriores circunstancias aunque, en este caso, el beneficio es menor que cuando se administran en pacientes con disfunción del VI.

Magnesio

Clase IIa

- Es razonable administrar Mg a aquellas personas con déficit documentado de especialmente aquellos que estuvieran tomando diuréticos previamente (Nivel de evidencia C)

-Es razonable administrar suplementos de Mg en presencia de TQ ventricular tipo Torsade de pointes asociado a QT largo. 1-2 g de Mg /IV en 5 minutos. (Nivel de evidencia C)

Clase III

1. En ausencia de déficits documentado de Mg o TV tipo Torsade de pointes no debe administrarse Mg. (Nivel de evidencia A)

Síndrome coronario agudo con elevación del segmento ST

Antagonistas del Ca

Clase IIa

- Es razonable administrar verapamil o diltiazem para el control de la isquemia o de la respuesta ventricular en el flutter o la FA en aquellos casos en los que los betabloqueantes sean ineficaces o estén contraindicados en ausencia de disfunción de VI .

Clase III

-Están contraindicada la administración de diltiazem y verapamil en pacientes con IAM y disfunción de VI asociada o Shock cardiogénico. (Nivel de evidencia A)

- Nifedipina (de absorción rápida) está contraindicado en el tratamiento del IAM por la taquicardia refleja y la hipoTA asociada a su uso (Nivel de evidencia B).

Síndrome coronario agudo con elevación del segmento ST

Nivel de actividad

Clase IIa

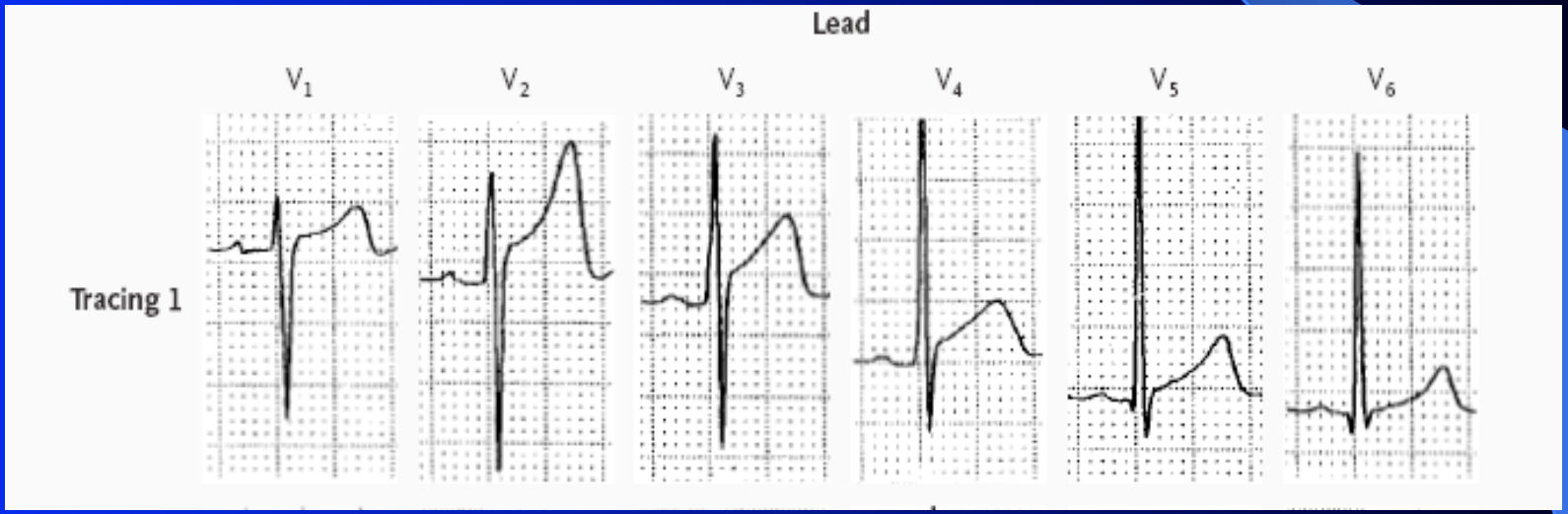
-Después de 12-24 h, es razonable que aquellos pacientes con inestabilidad hemodinámica o persistencia de la isquemia continúen encamados. (Nivel de evidencia C)

Clase III

- No mantener el reposo en cama más de 12-24 h a aquellos pacientes con IAM en ausencia de isquemia recurrente, síntomas de fallo cardiaco, o arritmias severas (Nivel de evidencia C)

Síndrome coronario agudo con elevación del segmento ST

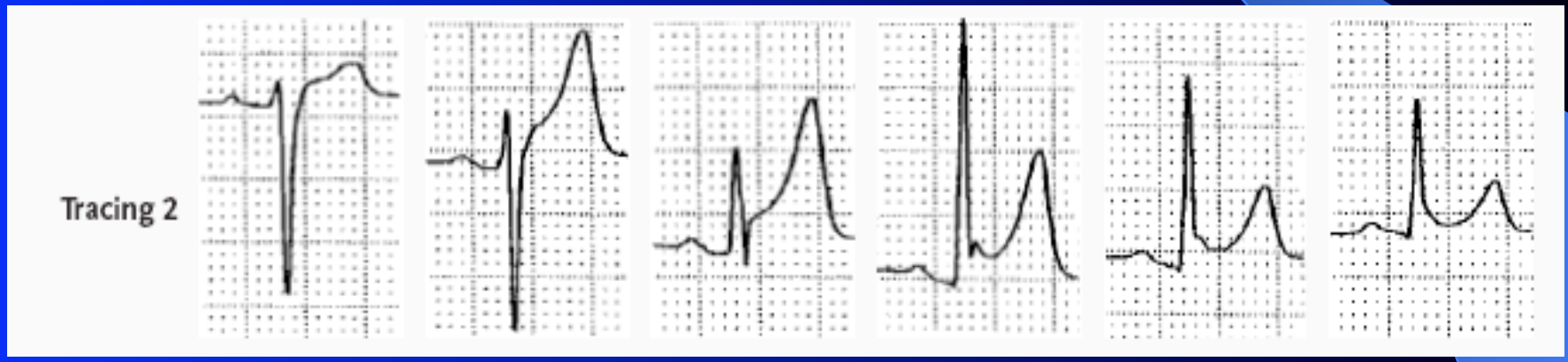
Otras causas de elevación de ST



Trazado normal en paciente joven

Síndrome coronario agudo con elevación del segmento ST

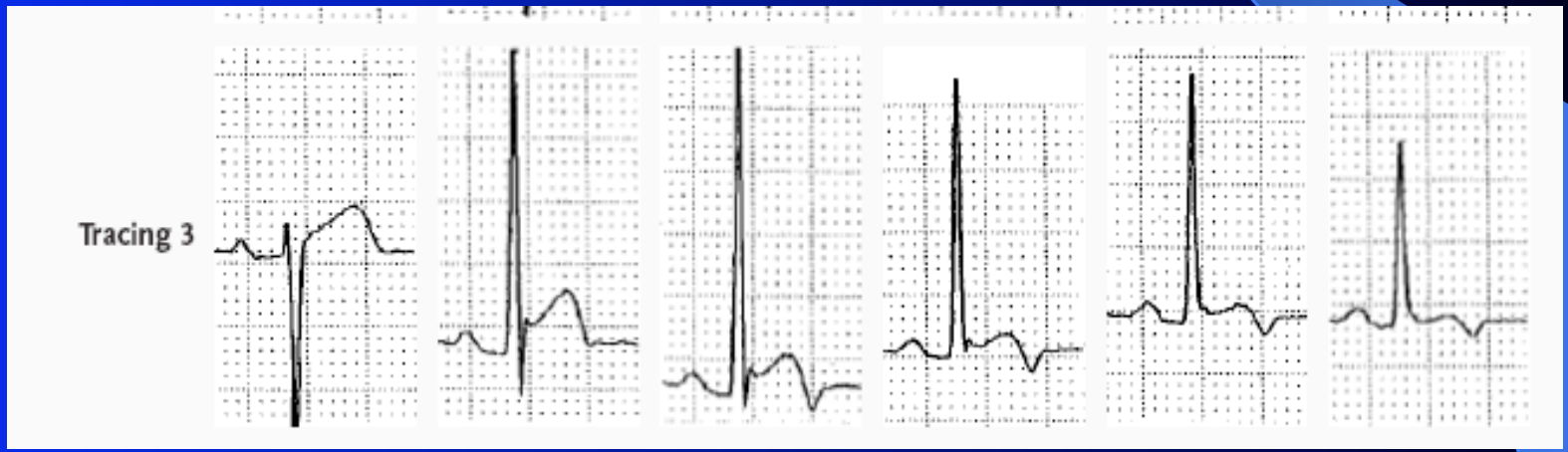
Otras causas de elevación de ST



Repolarización precoz con elevación de punto J

Síndrome coronario agudo con elevación del segmento ST

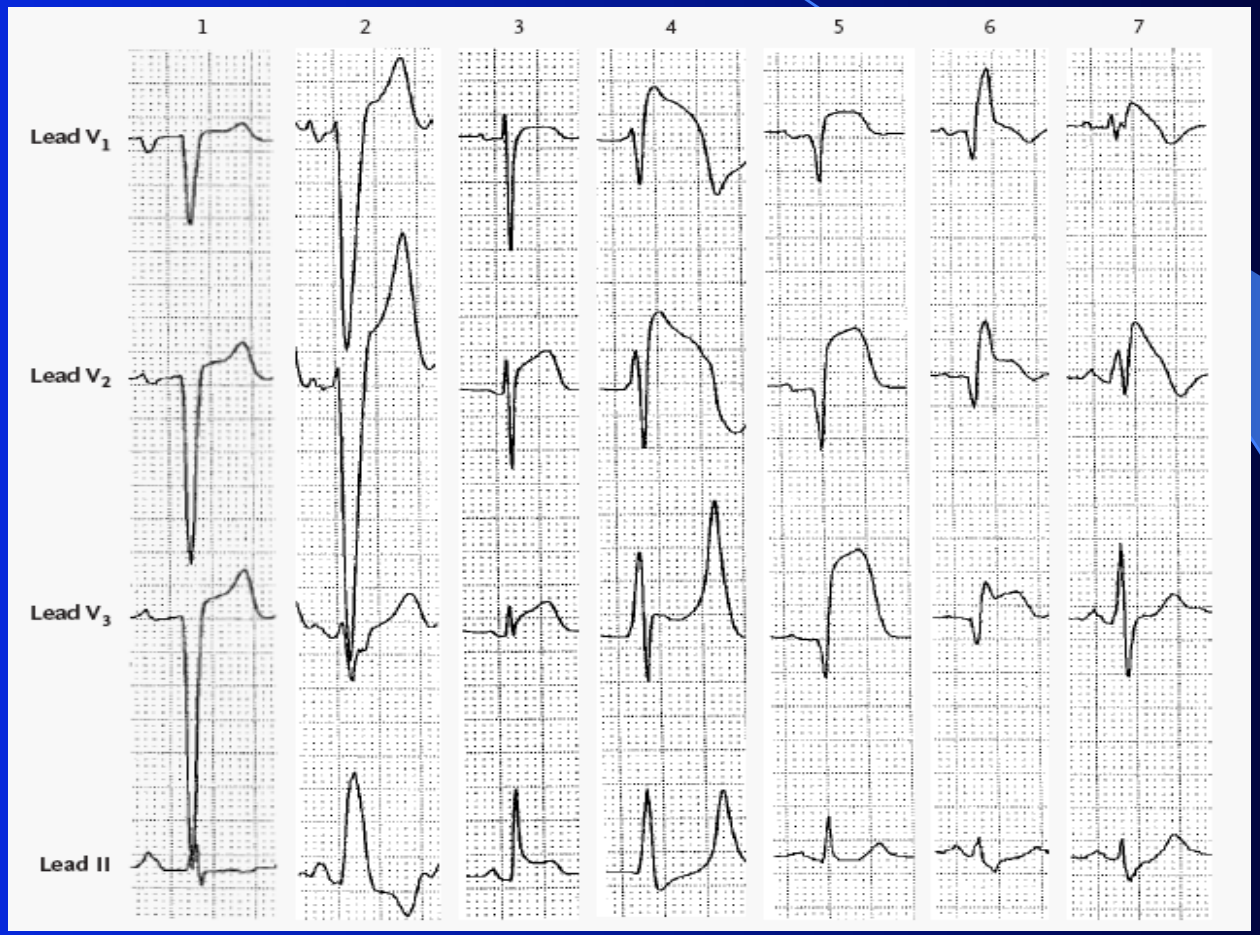
Otras causas de alteraciones de la repolarización



Variante normal con inversión de onda T

Síndrome coronario agudo con elevación del segmento ST

Otras causas de elevación de ST



HVI | BRI | pericar | HiperK | IAM ant-septal | IAM + BRD | Brugada

Errores frecuentes

Retrasar la reperfusión: fibrinolisis vs ACTP

Mantener NTG en ausencia de indicación

Valorar el descenso del ST especular

No descartar pericarditis o disección aórtica

Extraer gasometría arterial

Gracias