

LO MEJOR DEL CONGRESO ESC 2013 DE AMSTERDAM



Lo mejor sobre Cardiopatía Isquémica

*Antonio Fernández-Ortiz
Hospital Clínico San Carlos. Madrid*



SOCIEDAD
ESPAÑOLA DE
CARDIOLOGÍA

Casa del Corazón
Madrid, 5 de Septiembre de 2013



TOTAL ATTENDANCE



MUNICH 2012

27 407

AMSTERDAM 2013

29 990

Don't Miss

Amsterdam - Central Village

• 08:30

Clinical Trial Update Hot Line III: Risks and outcome



Study Outcomes

- Improve understanding of the importance of the study to the community and the wider world
- Increase awareness of the study's findings and the impact of the research
- Provide a platform for the study's findings to be shared with the public

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European Heart Journal Advance Access published August 30, 2013



EUROPEAN
SOCIETY OF
CARDIOLOGY

European Heart Journal
doi:10.1093/eurheartj/ehs296

ESC GUIDELINES



2013 ESC guidelines on the management of stable coronary artery disease

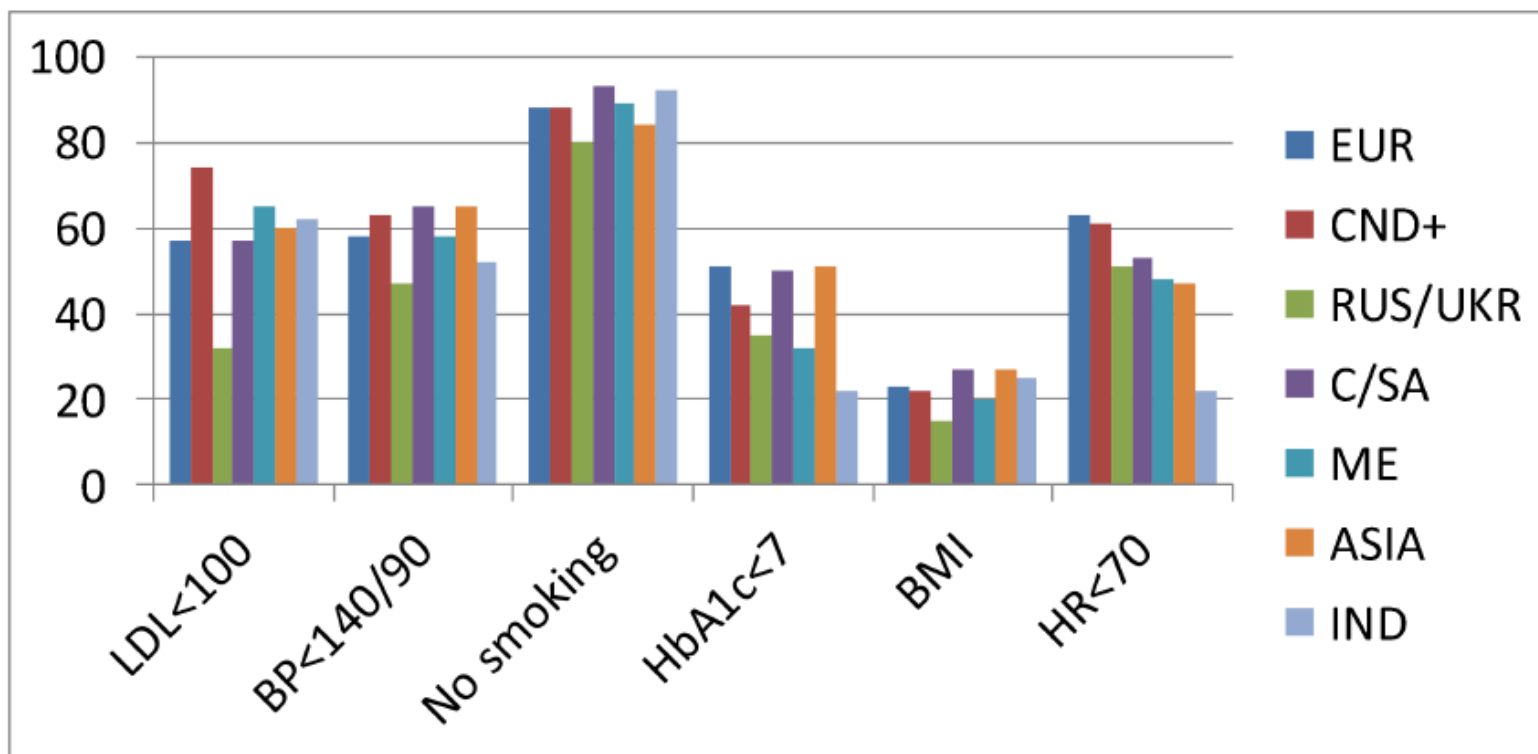
**The Task Force on the management of stable coronary artery disease
of the European Society of Cardiology**

Task Force Members: Gilles Montalescot* (Chairperson) (France), Udo Sechtem* (Chairperson) (Germany), Stephan Achenbach (Germany), Felicita Andreotti (Italy), Chris Arden (UK), Andrzej Budaj (Poland), Raffaele Bugiardini (Italy), Filippo Crea (Italy), Thomas Cuisset (France), Carlo Di Mario (UK), J. Rafael Ferreira (Portugal), Bernard J. Gersh (USA), Anselm K. Gitt (Germany), Jean-Sebastien Hulot (France), Nikolaus Marx (Germany), Lionel H. Opie (South Africa), Matthias Pfisterer (Switzerland), Eva Prescott (Denmark), Frank Ruschitzka (Switzerland), Manel Sabaté (Spain), Roxy Senior (UK), David Paul Taggart (UK), Ernst E. van der Wall (Netherlands), Christiaan J.M. Vrints (Belgium).

... is new as compared to 2006?

CLARIFY

Proportion of patients reaching target values



Angina relief

1st line

Short-acting Nitrates, *plus*

- **Beta-blockers** or **CCB-heart rate** ↓
- Consider **CCB-DHP** if low heart rate or intolerance/contraindications
- Consider **Beta-blockers + CCB-DHP** if CCS Angina > 2

May add or switch (1st line for some cases)

2nd line

Ivabradine
Long-acting nitrates
Nicorandil
Ranolazine^a
Trimetazidine^a

+ Consider Angio → PCI – Stenting or CABG

Event prevention

- Lifestyle management
- Control of risk factors

+ Educate the patient

- Aspirine^b
- Statins
- Consider ACEI or ARBs

Treatments in SCAD patients (1)

Angina/ischaemia^d relief

Short-acting nitrates are recommended.

I B 3,329

First-line treatment is indicated with β -blockers and/or calcium channel blockers to control heart rate and symptoms.

I A 3,331

For second-line treatment it is recommended to add long-acting nitrates or ivabradine or nicorandil or ranolazine, according to heart rate, blood pressure and tolerance.

IIa B 177,307,3,199,284,286,308,319-321,328,364

For second-line treatment, trimetazidine may be considered.

IIb B 313,315

According to comorbidities/tolerance it is indicated to use second-line therapies as first-line treatment in selected patients.

I C -

In asymptomatic patients with large areas of ischaemia (>10%) β -blockers should be considered.

IIa C -

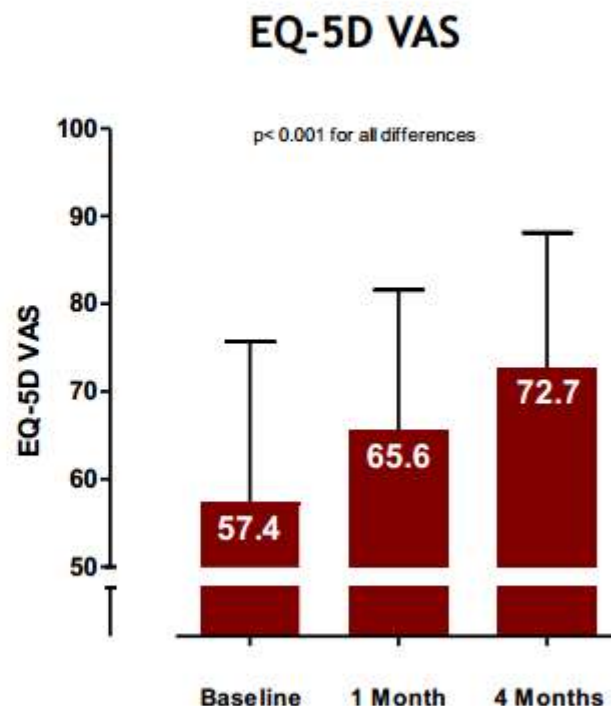
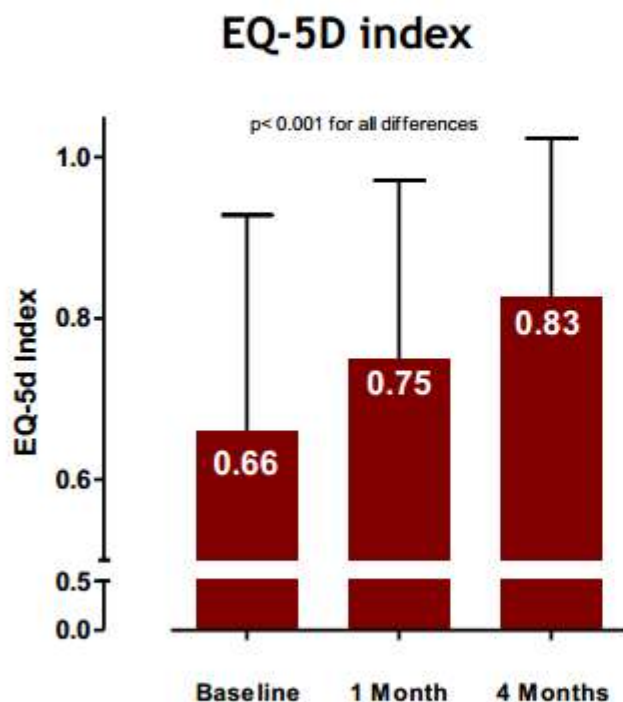
In patients with vasospastic angina, calcium channel blockers and nitrates should be considered and beta-blockers avoided.

IIa B 3,365

Second line treatments (1)

ADDITIONS
The addition of second-line treatment

Quality of life



Survey of swedish population : Angina pectoris EQ-5D index: 0.70



Table 29 Treatment in patients with microvascular angina

Recommendations	Class ^a	Level ^b	Ref. ^c
It is recommended that all patients receive secondary prevention medications including aspirin and statins.	I	B	371
β -blockers are recommended as a first line treatment.	I	B	372
Calcium antagonists are recommended if β -blockers do not achieve sufficient symptomatic benefit or are not tolerated.	I	B	367
ACE inhibitors or nicorandil may be considered in patients with refractory symptoms.	IIb	B	368
Xanthine derivatives or non-pharmacological treatments such as neurostimulatory techniques may be considered in patients with symptoms refractory to the above listed drugs.	IIb	B	373–375

SCAD b

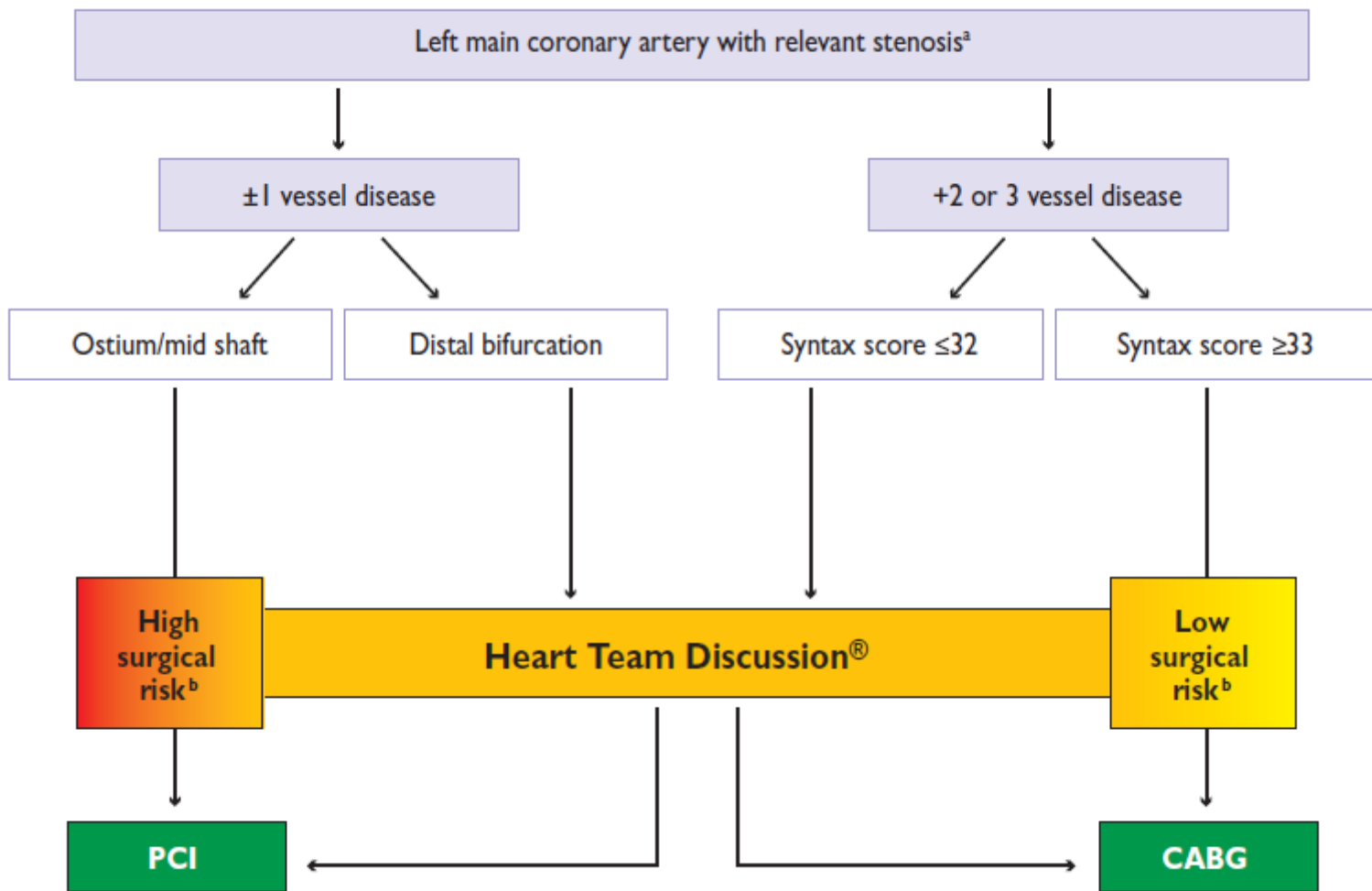
• Micro

• Vaso



Ilippo CREA
Roma - (IT)

365
C CONGRESS





Abbott

Promise for Life

Structural Heart

ECG

max



UCR

Uppsala Clinical
Research Center



Thrombus Aspiration in ST- Elevation myocardial infarction in Scandinavia (*TASTE* trial)

Main results at 30 days

Ole Fröbert, MD, PhD - on behalf of the *TASTE* investigators
Department of Cardiology
Örebro University Hospital
Sweden

Amalation

11 23



Ole FRÖBERT
(Örebro - SE)

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ESC Congress 365

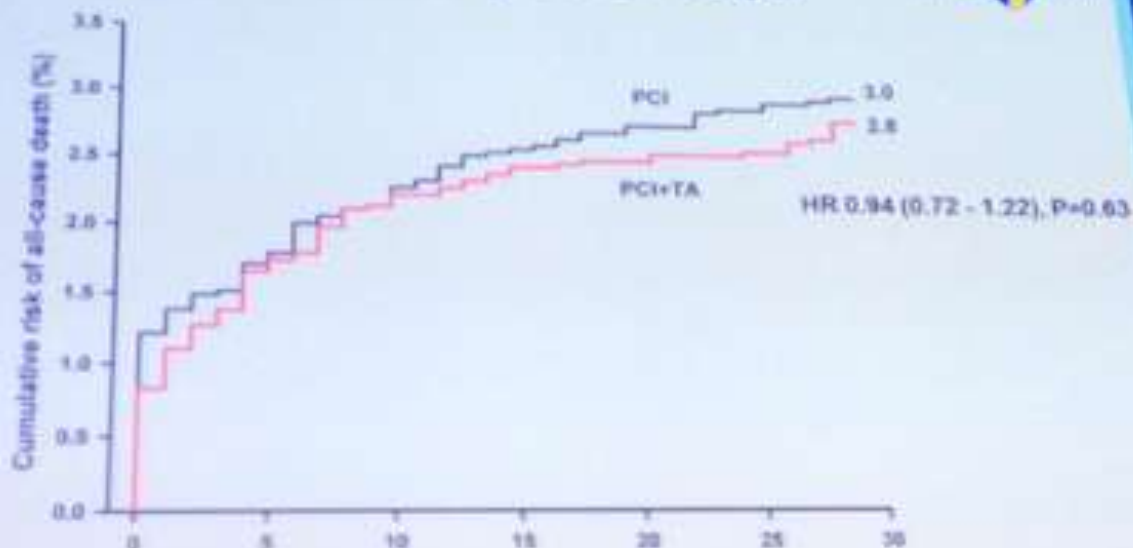
www.esccongress.org

All-cause mortality at 30 days



Amsterdam

11.26



Dr. FRANCESCO
FRONEO (Director - ESC)

No. at Risk

PCI+TA	3821	3568	3540	3522	3528	3524	3519
PCI	3823	3867	3545	3520	3523	3517	3513

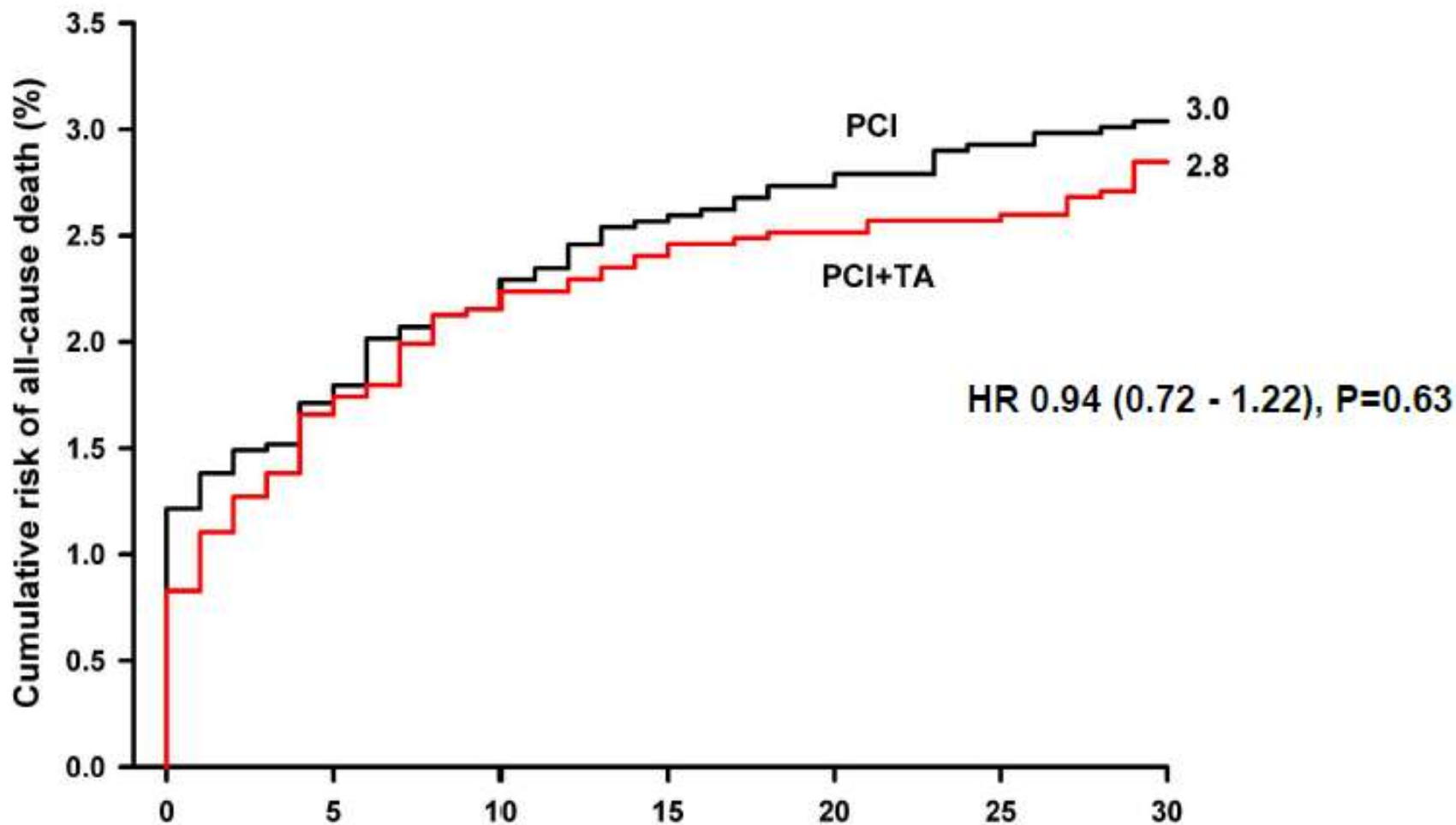
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TASTE

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All-cause mortality at 30 days



No. at Risk

PCI+TA	3621	3568	3540	3532	3526	3524	3519
PCI	3623	3567	3545	3530	3523	3517	3513

TASTE



Treatment of Acute Coronary
Syndromes with Otamixaban

TAO : Treatment of Acute Coronary Syndromes with Otamixaban

Philippe Gabriel Steg* on behalf of the TAO investigators

*DHU-FIRE, Hôpital Bichat, Assistance Publique – Hôpitaux de Paris, Université Paris –
Diderot, INSERM U-698, Paris, France

*Disclosures: Research grants (to INSERM U698): NYU school of Medicine, Sanofi, Servier. Speaking or consulting:
Amarin, AstraZeneca, Bayer, Boehringer Ingelheim, Bristol-Myers Squibb, Daiichi-Sankyo-Lilly, GlaxoSmithKline,
Medtronic, Novartis, Otsuka, Pfizer, Sanofi, Servier, The Medicines Company, Virvax. Stockholding: Aterova.

The TAO trial was supported by SANOFI

clinicaltrials.gov : NCT01076764

Amsterdam

14.30



Philippe Gabriel STEG
(Paris Cedex 18 - F14)

14-18 SEPTEMBER 2013
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Treatment of Acute Coronary Syndrome with Otamixaban

Moderate- to high-risk NSTEMI-ACS with planned early invasive strategy (n=13,220)

Aspirin + ADP receptor antagonist
at or before randomization

R

Otamixaban
0.08+0.10
(n=1969)

Otamixaban
0.08+0.140
(n=1969)

UFH +
eptifibatide
(n=1969)

Interim analysis
One dose goes
forward*

*Selected by DSMB while
maintaining the blind

Double-blind, triple-dummy study

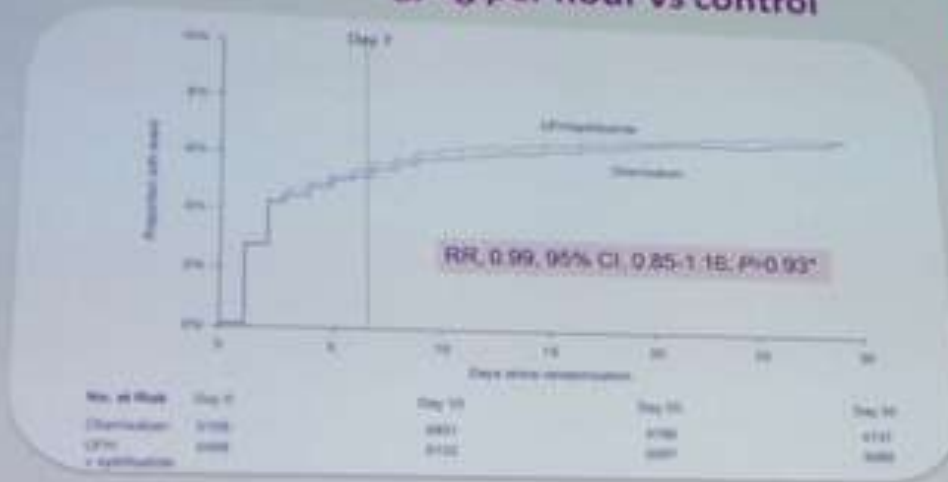
(n=5625)

(n=5625)

Primary efficacy endpoint: death/MI at day 7
Primary safety endpoint: TIMI major +minor bleeds at day 7



Primary efficacy outcome for otamixaban 0.140 mg/kg per hour vs control



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11/25



Philippe Gabriel STEG
(Pars Codex 18 - FR)

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*Fisher's exact test



Primary efficacy outcome for otamixaban
0.140 mg/kg per hour vs control

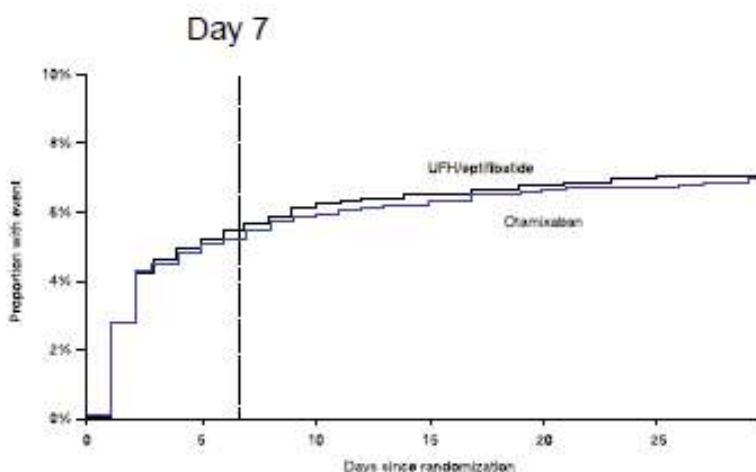




Treatment of Acute Coronary Syndrome with Otamixaban

Primary efficacy and safety outcomes for otamixaban 0.140 mg/kg/hr vs control

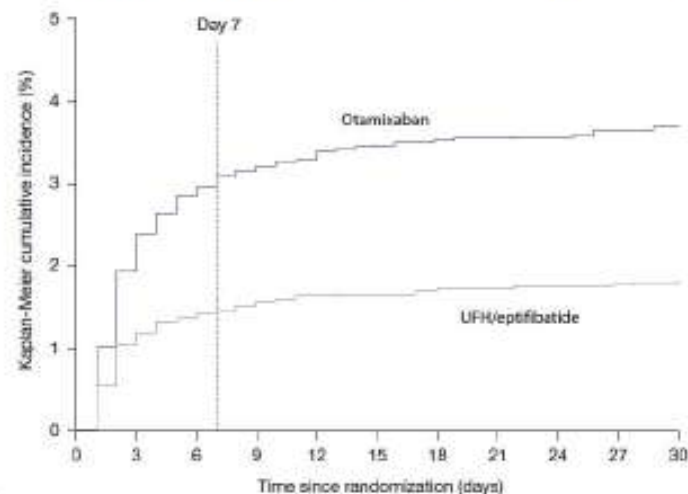
Efficacy Death or MI



No. at Risk	Day 0	Day 10	Day 20
Otamixaban	5105	4801	4766
UFH + eptifibatid	5466	5132	5097

RR, 0.99, 95% CI, 0.85-1.16; P=0.93*

Safety TIMI major or minor bleed



No. at Risk	Day 0	Day 7	Day 15	Day 30
Otamixaban	2657	2552	2533	2431
UFH + eptifibatid	5466	5293	5257	5086

RR, 2.13, 95% CI, 1.63-2.78

Preventive **A**ngioplasty in **M**yocardial **I**nfarction Trial

PRAMI Trial

Randomised multicentre single-blind trial conducted
in five UK cardiac centres

Attendees



Preventive Angioplasty in Mycocardial Infarction Trial

PRAMI Trial

Randomised multicentre single-blind trial conducted
in five UK cardiac centres

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Randomized Trial of Preventive Angioplasty in Myocardial Infarction

David S. Wald, M.D., Joan K. Morris, Ph.D., Nicholas J. Wald, F.R.S.,
Alexander J. Chase, M.B., B.S., Ph.D., Richard J. Edwards, M.D.,
Liam O. Hughes, M.D., Colin Berry, M.B., Ch.B., Ph.D.,
and Keith G. Oldroyd, M.D., for the PRAMI Investigators*

N Engl J Med September 1st 2013;369. DOI: 10.1056/NEJMoa1305520

Cardiac Death, Nonfatal MI or Refractory Angina
in patients having infarct-artery PCI

Hazard Ratio 0.35
(95% CI 0.21 to 0.58),
 $p < 0.001$

Risk Reduction 65%

21

53

Preventive
n=231

No Preventive PCI
n=231

Cardiac Death, Nonfatal MI or Refractory Angina in patients having infarct-artery PCI

Hazard Ratio 0.35
(95% CI 0.21 to 0.58),
 $p < 0.001$

Risk Reduction 65%

21



Group	n	Events
Preventive PCI	234	21
No Preventive PCI	231	53

Preventive PCI
n=234

53

No Preventive PCI
n=231

Conclusion

Substantial benefit of Preventive PCI in patients with ST elevation myocardial infarction

65% ↓ cardiac death / nonfatal MI / refractory angina

64% ↓ cardiac death / nonfatal MI



ACCOAST

A Comparison of prasugrel at the time of percutaneous
Coronary intervention **O**r as pretreatment **A**t the time
of diagnosis in patients with non-**ST**-elevation MI

G Montalescot, L Bolognese, D Dudek, P Goldstein, C Hamm, JF Tanguay,
JM ten Berg, DL Miller, TM Costigan, J Goedicke, J Silvain, P Angioli,
J Legutko, M Niethammer, Z Motovska, JA Jakubowski, G Cayla,
LO Visconti, E Vicaut, P Widimsky for the ACCOAST investigators

COI DISCLOSURE FOR P

are available @ <http://www.action-coeur.org>

Amsterdam



ACCOAST design

NSTEMI + Troponin • 1.5 times ULN local lab value
Clonidogrel naive or on long term clonidogrel 75 mg

n~4100 (event driven)

Randomize 1:1
Double-blind

Prasugrel 30 mg

Placebo

**Coronary
Angiography**

**Coronary
Angiography**

Prasugrel 30 mg

Prasugrel 60 mg

PCI

PCI

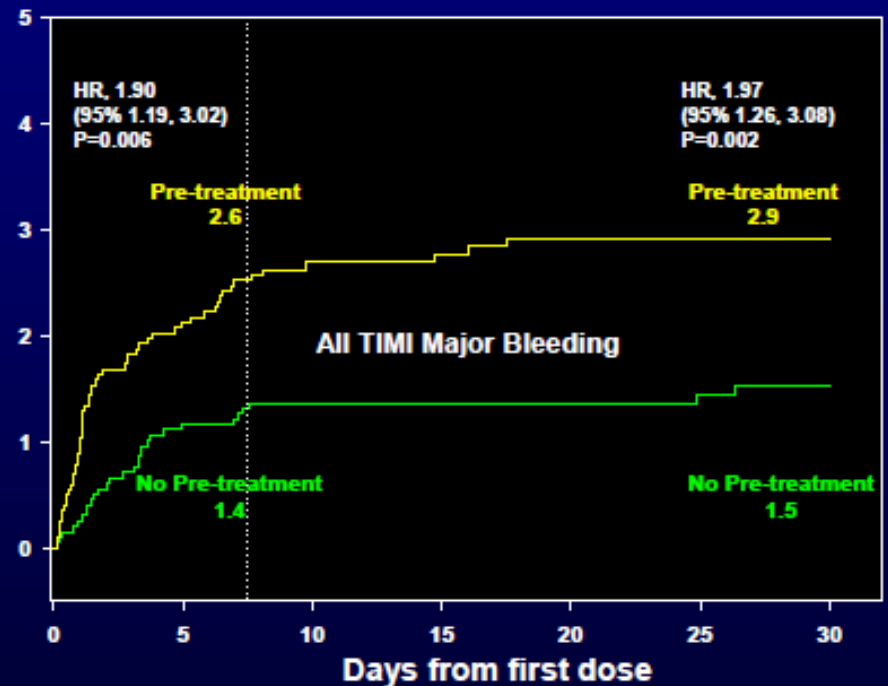
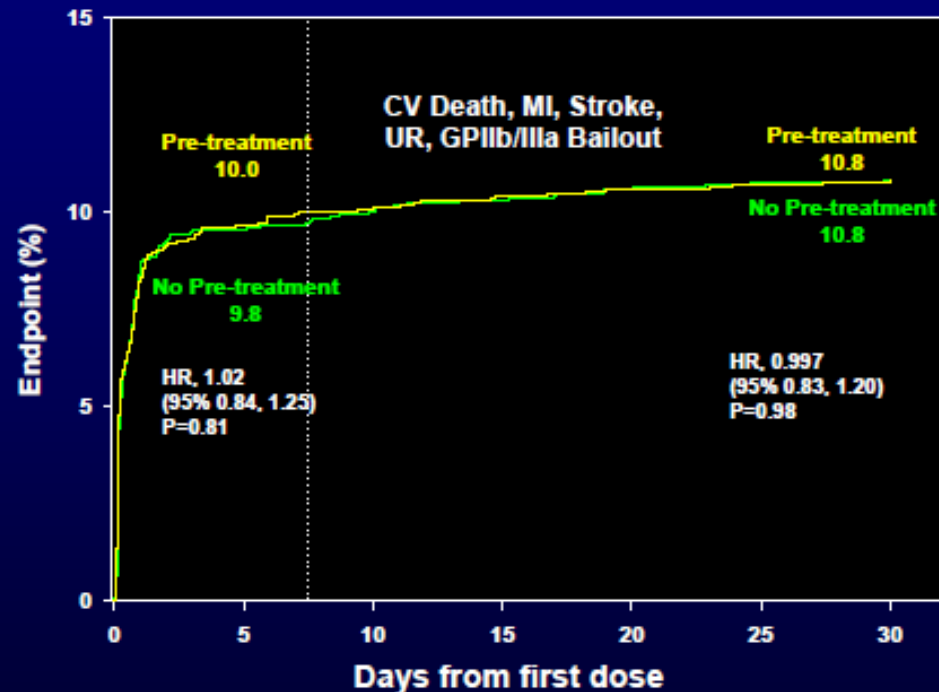
CABG
or
Medical
Management
(no more prasugrel)

CABG
or
Medical
Management
(no prasugrel)

Prasugrel 10 mg or 5 mg (based on weight and age) for 30 days

1° Endpoint: CV Death, MI, Stroke, Urg Revasc, GP IIb/IIIa inh. Bailout, at 7 days

Primary Efficacy and Safety Endpoints (All Patients)





UPPSALA
UNIVERSITET

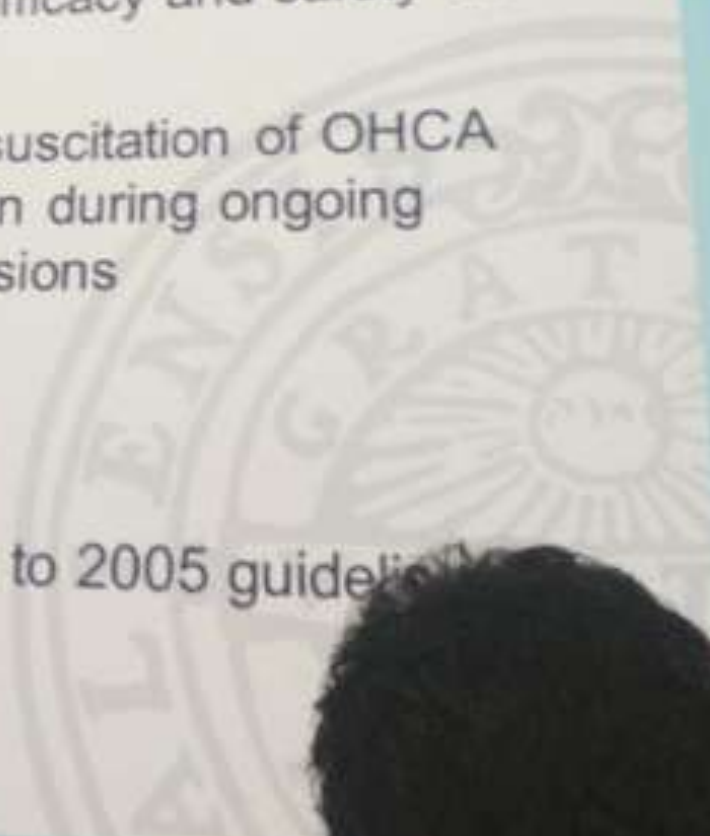
What is LINC?

A multicenter, randomized, controlled trial designed to evaluate the efficacy and safety of:

LUCAS concept for resuscitation of OHCA including defibrillation during ongoing compressions

vs.

manual CPR according to 2005 guidelines



LUCAS 2™



Mechanical Compression- Decompression

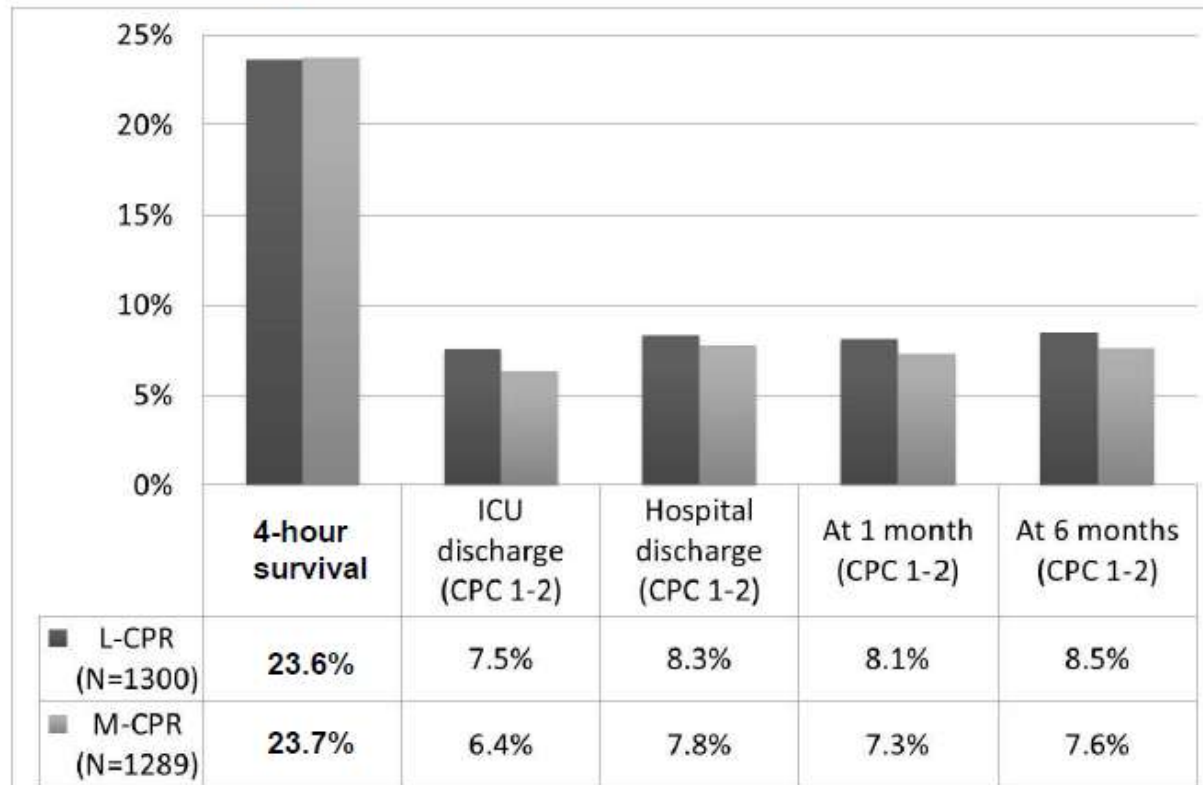
- Electricity-battery
- 100 compressions/min
- 4-5 cm compression depth
- Complete chest recoil
- 50/50 duty cycle
- Allows defibrillation running



UPPSALA
UNIVERSITET

4-hour survival:
Risk difference -0.05%
95% C.I. -3.32 – 3.23, p=1.00

Outcome



LINC study





ACCA A new association in the ESC family

*Peter Clemmensen MD, DMSC, FESC
ACCA President*

Moscow



Peter Michael Clemmensen
(Copenhagen)

ESC C

ESC Congress 36

Current management of ACS: An international perspective from the EPICOR study



Hector BUENO
(Madrid - ES)

Héctor Bueno, MD, PhD, FESC, FAHA
Associate Professor of Medicine
Universidad Complutense de Madrid
Head, Clinical Cardiology & CCU
Department of Cardiology
Hospital General Universitario Gregorio Marañón
Madrid (SPAIN)



Hospital General Universitario
Gregorio Marañón

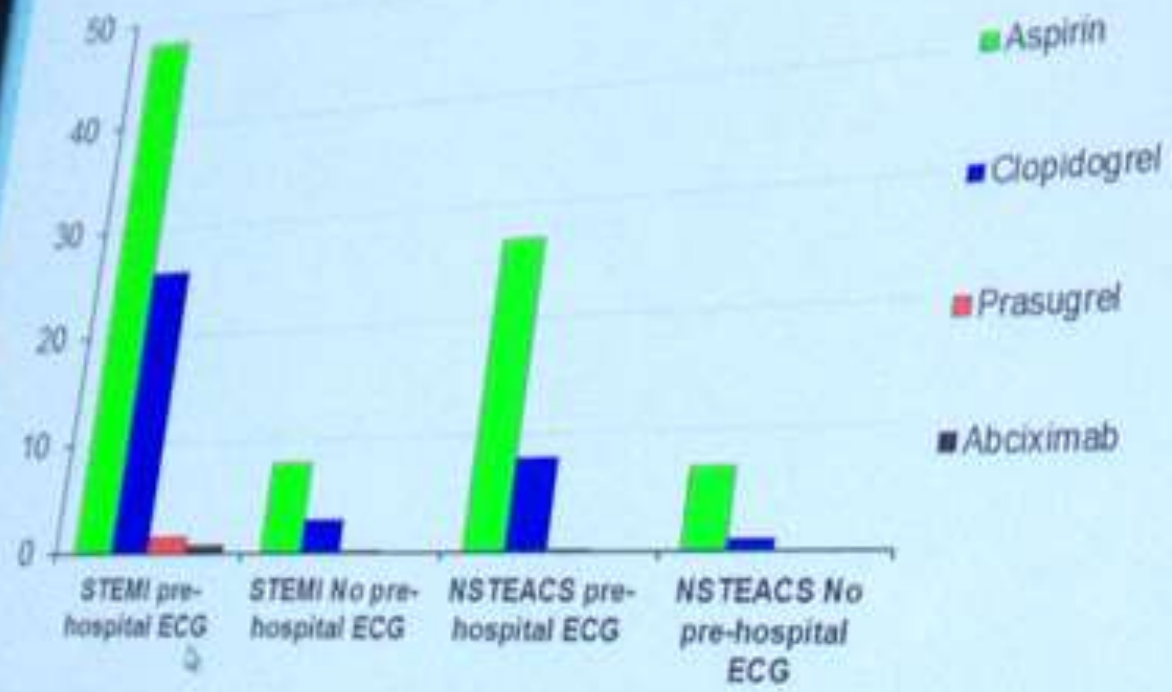
ambulatory
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www.esc-congress.org/2013

EPICOR: Pre-hospital use of antithrombotic drugs



Hector BUENO
(Madrid - ES)

ESC CONGRESS

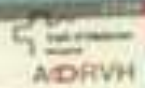
ESC Congress 365





SERVIER

IN
RESEARCH



Primary results of the PROMISE trial: myocardial protection with intracoronary adenosine given before reperfusion in patients with STEMI. (Clin. Trial gov NCT 00781404)

D. Garcia-Dorado, I. Otaegui, JF. Rodriguez Palomares, A. Evangelista, V. Pineda, R. Ruiz Salmeron, F. Gimeno, F. Fernandez Aviles, A. San Roman, B. Garcia Del Blanco
On behalf of the PROMISE investigators

Hospital Universitari Vall d'Hebron, Cardiology Department, Barcelona, Spain, University Hospital of Virgen Macarena, Seville, Spain, Clinic University Hospital of Valladolid, ICICOR, Valladolid, Spain, University Hospital Gregorio Maranon, Department of Cardiology, Madrid, Spain

No conflict of interest

Funded by Carlos III Health Institute, Ministry of Science of Spain, EC07/90511

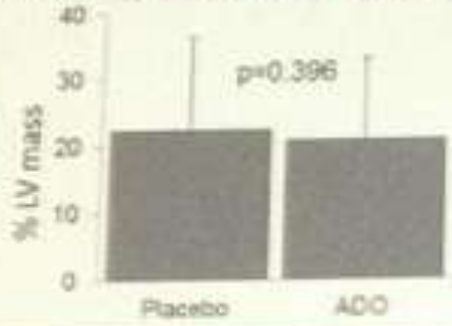


David GARCIA-DORADO (Barcelona - ES)

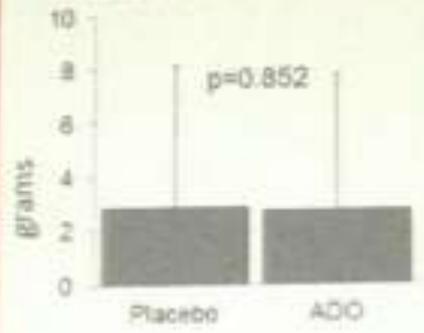


Results-3: total study population

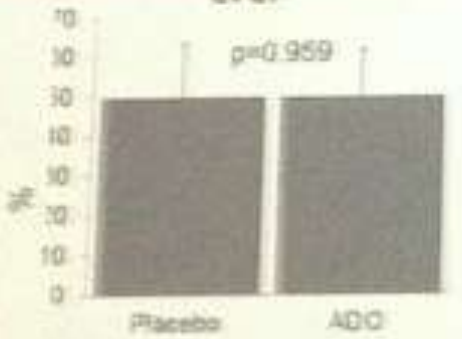
Primary end-point: Infarct mass



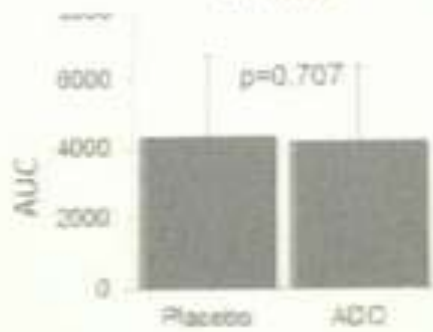
Microvascular obstruction mass



LVEF



CK-MB



David GARCIA-DORADO
(Barcelona - ES)



Non-intensity Trial

EVEREST 2: randomized clinical trial

Study design

20 patients enrolled at 17 sites

Supportive care

Randomized 1:1

Standard of care (SOC)

Control Group (Supportive care + treatment X)

Primary (P) by: Effectiveness

Clinical benefit (Mortality, health-related quality of life, and patient-reported outcomes)

Secondary (S) by: Effectiveness

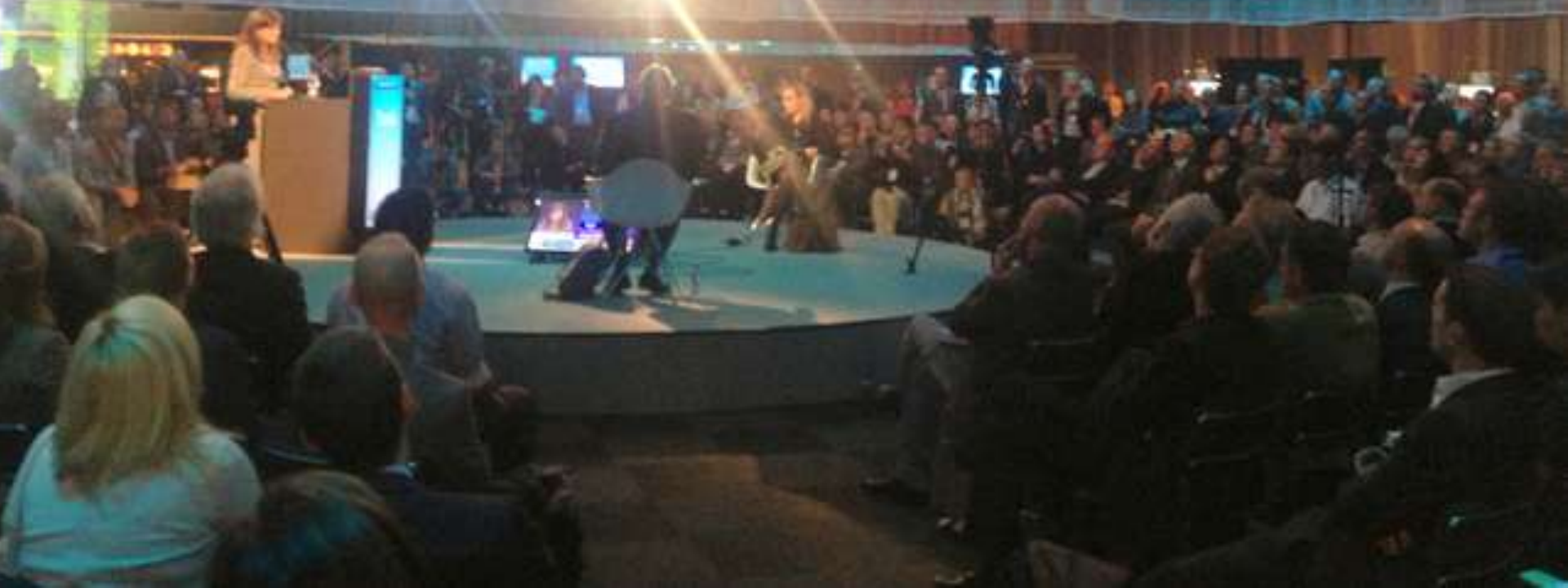
Health-related quality of life (HRQL), patient-reported outcomes (PRO), and patient-reported burden of care (PRBC)

Statistical significance

HRQL, PRO, and PRBC

Statistical significance

HRQL, PRO, and PRBC





P3997

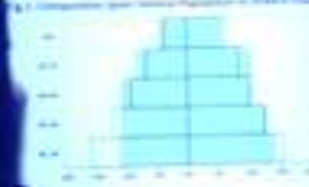
Abstract
COR - ACC

Stable angina in Spain. Results of the OFRECE study

Muñoz J, Anguita M, Rodríguez-Rodríguez J, López C, Remón J, Díez-Tejedor E, et al. Sociedad Española de Cardiología. Spanish Society of Cardiology Spain

RESULTS

The study included 30,000 patients with stable angina pectoris who were treated with 44 drugs and 2,000 of them with 4 or more drugs. The study aimed at: (1) characterizing the clinical presentation and (2) determining the prevalence of each drug in the population of stable angina patients.



Basic characteristics of the study population	
Age (years)	58 ± 13
Gender (n / %)	57 / 19
MI	38 (13.0)
Cholesterol (mmol/L)	4.1 ± 0.8
Smoking (%)	31.1
Diabetes (%)	25.1
Family history (%)	22.3
History of CAD (%)	41.1
Coronary artery disease (%)	7.8
Coronary artery bypass grafting (%)	1.1

Drug	n	%	95% CI
ASA	11,573	38.6	37.9-39.3
CCB	11,400	38.0	37.3-38.7
Beta-blockers	10,827	36.1	35.4-36.8
Diltiazem	10,754	35.8	35.1-36.5
Clonidine	10,123	33.7	33.0-34.4
Nitroglycerin	9,512	31.7	31.0-32.4
Nitroglycerin sublingual	9,401	31.3	30.6-32.0
Total	29,977	100.0	

Prevalence of drug use in the population of stable angina patients	
Number of drugs	n (%)
1	2,000 (6.7)
2	10,000 (33.3)
3	15,000 (50.0)
4	2,500 (8.3)
5	400 (1.3)
6	70 (0.2)
7	10 (0.0)
8	0 (0.0)

Effect of ischemic postconditioning on microvascular obstruction in reperfused myocardial infarction. Results of a randomized multicenter study in patients and data of an experimental model.

C. Bonanad¹, JM. Ruiz-Noda², E. Fedu¹, J. Nunez¹, J. Sanchez¹, JM. Martinez-Ehriza², G. Mirana², JV. Monmeneu¹, F.J. Chorro¹, V. Bodí Pons¹
 (1) Hospital Clínic Universitario, IBC3 IVA, Universitat de València, Valencia, Spain (2) General University Hospital of Alicante, Alicante, Spain

PURPOSE

Ischemic Postconditioning (PCON) appears as a potentially beneficial tool to complement primary angioplasty in ST-segment elevation myocardial infarction (STEMI). We evaluated the impact of PCON on Microvascular Obstruction (MVO) both in patients and in a highly controlled swine model.

METHODS

In a multicenter study, 101 patients with a first STEMI were randomized to undergo primary angioplasty followed by PCON or primary angioplasty alone (non-PCON). MVO was quantified in late enhancement cardiac magnetic resonance imaging.

In an anterior STEMI swine model based on a 90-min angioplasty balloon coronary occlusion and 3-day reperfusion, MVO was defined as a lack of thioflavin-S staining in the core of the infarcted area (with triphenyltetrazolium-chloride staining).

The extent of MVO (% of left ventricular mass) was quantified in a core laboratory.

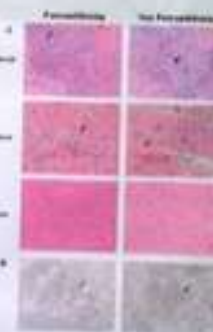
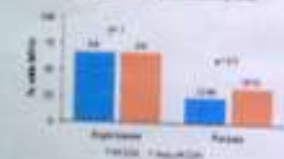
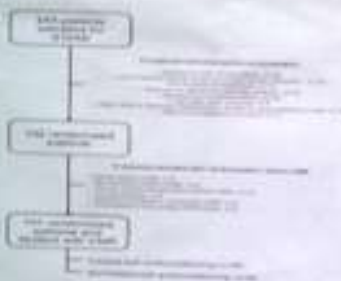


RESULTS

Patients treated with (n=49) and without PCON (n=52) were well matched in terms of baseline characteristics and time to revascularization.

MVO (> 1 segment in the 17 segments model) occurred in 12 (24%) PCON and in 18 (35%) non-PCON patients, p=0.3. PCON did not significantly reduce MVO (1.4±1.9% vs. 1.9±3.6% of LV mass, p=0.3). IS was similar in PCON and non-PCON patients (18±13 vs. 21±14% of LV mass, p=0.2).

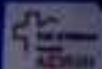
No significant differences were observed between PCON and non-PCON patients in LV volumes, ejection fraction or the extent of hemorrhage. In the swine model, MVO occurred in 4/6 (67%) PCON and in 4/6 (67%) non-PCON pigs, p=1. The extent of MVO (7.8±5.5% vs. 5.6±5.4% of LV mass, p=0.5) and IS (22±14 vs. 24±10% of LV mass, p=0.8) were not reduced in PCON compared with non-PCON pigs.



CONCLUSION

Ischemic postconditioning does not reduce microvascular obstruction in STEMI.

Determinants of infarct mass, assessed by cardiac magnetic resonance, in STEACS revascularized by primary-PCI: Findings of the PROMISE randomized clinical trial



Ignacio Ferreira-González¹, Bruno García-del-Blanco¹, José Barrabés¹, Sergio Moral¹, Gerard Martí¹, Inmanol Otaegui¹, Enric Domingo¹, Jaime Elizaga¹, Hipólito Gutiérrez², and David García-Dorado¹ (presenter). 1: Cardiology Department, Vall d'Hebron Hospital, Barcelona. 2: Cardiology Department, Hospital Gregorio Marañón, Madrid. 3: Cardiology Department, Hospital de Valladolid.

Abstract

Objective: The randomized, placebo controlled, PROMISE trial assessed the effect of intracoronary adenosine given at the time of primary percutaneous coronary intervention (PCI). Determinants of relative infarct mass (RIM), assessed by magnetic resonance imaging (MRI), are presented.

Methods and results: 177 patients with a first STEMI receiving PCI within 6 hours of symptoms onset (SO) who underwent MRI 2-7 days after reperfusion were included. RIM, the percentage of total myocardial mass revascularized, was quantified by late Gadolinium enhancement. Myocardium at risk (MAR), quantified by T2 weighted sequences. Demographics, risk factors, angiographic characteristics, reperfusion time, and intra-procedure features were potential determinants of RIM to be assessed by linear regression modeling. TIMI 3 flow was achieved in 162 (91.5%) patients, a mean of 211 minutes (SD: 64.17, IQR: 105 - 195) after SO. Grade 2-3 Rentrop collateral flow was present in 36 (21.5%) patients. Percentage of total MAR was 20% (SD15%, min 0%, max 78%), and the final RIM was 21.6% (SD13.1%, min 0%, max 53.3%, mean salvaged myocardium 38%). The best model (Table) predicted 70.6% of the total variability of RIM. MAR had the most impact on the final RIM, whereas the impact of the rest was modest. Rentrop 2-3 did not predict lower RIM but it attenuated the effect of MAR on the final RIM (significant interaction). Higher body mass index (BMI) was associated with lower RIM.

Conclusions: More than 50% of MAR, the variable with the highest impact on final RIM, is not salvaged in spite of the best available treatments. The low impact of late SO-TIMI 3 flow on final RIM probably reflects that most patients arrive too late to influence RIM substantially. A paradoxical relationship between BMI and RIM was found.

Objectives

The randomized, placebo controlled, PROMISE trial assessed the effect of intracoronary adenosine given at the time of primary percutaneous coronary intervention (PCI).

In this sub-study, determinants of the percentage of total myocardial mass with necrosis (PTMN), assessed by cardiac magnetic resonance (CMR), are presented.

Methods

Patient population:

- 177 patients with a first STEMI receiving PCI within 6 hours of symptoms onset (SO) who underwent CMR 2-7 days after reperfusion were included.

- TIMI 3 flow was achieved in 162 (91.5%) patients, a mean of 211 minutes (SD: 64.17, IQR: 105 - 195) after SO. Grade 2-3 Rentrop collateral flow was present in 36 (21.5%) patients.

MRI measurements:

- The percentage of total myocardial mass with necrosis (PTMN), was quantified by late Gadolinium enhancement.

- Myocardium at risk (MAR) was quantified by T2 weighted sequences.

Analytical procedures: demographics, risk factors, angiographic characteristics, reperfusion time, and intra-procedure features were potential determinants of PTMN to be assessed by linear regression modeling.



Results

- Percentage of total MAR was 35% (SD13%; min 12%, max 78%).

- The final PTMN was 21.6% (SD13.1%; min 0%, max 53.3%; mean salvaged myocardium 38%).

- The best model (Table) predicted 70.6% of the total variability of PTMN.

- MAR had the most impact on final RIM.

- Rentrop 2-3 did not predict lower PTMN but it attenuated the effect of MAR on the final PTMN (significant interaction).

- Higher body mass index (BMI) was associated with lower PTMN.

	Non-adjusted	95% CI	P	Contribution
Myocardium at risk (MAR)	4.51	(4.01 - 5.02)	<0.001	70%
Age (yr)	-0.13	(-0.19 - -0.07)	0.002	5%
Myocardium at risk at baseline	0.74	(0.29 - 1.20)	0.003	13%
Log time to TIMI 3	0.75	(0.36 - 1.14)	0.001	11%
Final TIMI 3	-11.02	(-11.48 - -10.56)	<0.001	11%
Body mass index (BMI) (kg/m ²)	-0.38	(-0.73 - -0.03)	0.03	1%

To the variability of total RIM in the model. Compared to being with baseline value.

Conclusions

1. More than 50% of myocardium at risk, the variable with the highest impact on the final infarct size, is not salvaged in spite of the best available treatments.
2. The low impact of the time onset of symptoms-TIMI III probably reflects that most patients arrive too late to PCI.
3. A paradoxical relationship between body mass index and infarct size was found.

Authors declare no potential conflict of interests

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Functional mitral regurgitation after a first non-ST elevation acute coronary syndrome: very long term follow-up, prognosis, and contribution to left ventricular enlargement and atrial fibrillation

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PURPOSE:

Functional mitral regurgitation (MR) is frequent after a myocardial infarction. Our aim was to assess the relationship between onset functional MR after a non-ST segment elevation acute myocardial syndrome (NSTSEACS) and long-term prognosis, ventricular remodeling and further development of atrial fibrillation.

RESULTS:

MR was detected in 95 cases (40.1%), and became an independent risk factor for the development of heart failure and MACE (Per MR degree, HR_{HF} 1.71, CI 95% 1.138-2.588, p=0.01; HR_{MACE} 1.43, CI 95% 1.156-1.921, p=0.002), see figure 1. LV diastolic (grade I: 12.7±40.7, grade II: 26.8±12.4, grade III: 46.3±50.9 cc, p=0.01) and systolic (grade I: 10.4±37.3, grade II: 10.12±12.7, grade III: 36.8±46.0) cc, p=0.02) mean volumes were higher after follow-up in patients with MR, proportionally to the initial MR degree.

In the rhythm analysis (125 patients, previously excluding those with any history of atrial fibrillation) after follow-up, 11.4% degree I MR patients developed AF, 14.3% degree II and 75% degree III, while only 5.1% degree 0 patients developed AF, p=0.001.

METHODS:

We prospectively studied 237 patients consecutively discharged in NYHA class I-II (74% men, mean age 66.1) after a first NSTSEACS.

All underwent an electrocardiogram the first week after admission and were echocardiographically and clinically followed up (median: 6.95 years).



Figure 1. Event free Survival probability (Kaplan Meier) after an STEMI regarding the presence (A-B) degree (C-D) of MR at admission echocardiogram.

CONCLUSION:

MR is frequent after a NSTSEACS. The presence and degree of MR confers a worse long-term prognosis after a first NSTSEACS.

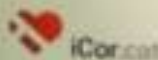
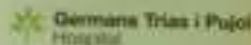
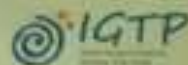
In part, this can be explained by an increased negative remodeling and increased occurrence of atrial fibrillation.

Declaration of interest: None

Effect of a cell-based bioactive smart patch after myocardial infarction in swine

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Purpose
 Here we show in the first study of acute myocardial infarction (AMI) and size of the acute infarction, the effect of a cell-based bioactive smart patch (ATDPC) on myocardial infarction. The objective of this work was to determine the effect of a cell-based bioactive smart patch with ATDPCs on myocardial infarction and on the infarct size. www.igtp.upc.edu



Fig. 1. Application of the cell-based bioactive smart patch (ATDPC) on the infarcted area of the heart. The patch is applied on the infarcted area of the heart. The patch is applied on the infarcted area of the heart. The patch is applied on the infarcted area of the heart.

Methods
 Decellularized human pericardium was used as a biological scaffold filled with 2x10⁶ GFP-ATDPCs embedded in Poly(lactide-co-glycolide) (PLGA) and 3D network, biodegradable polymer. A left lateral MI was induced by coronary artery ligation in 22 pigs divided in treated (n=11) and control (n=11) groups. 30 minutes after MI induction, the smart patch with or without GFP-ATDPCs was implanted covering the infarcted area. In 2 treated and 1 control pigs the smart patch was connected to a biopotential monitoring system to analyze the myocardial scar evolution. Electrical impedance tomography (EIT) measurements in the range 1 kHz-200 kHz were obtained every 5 minutes during 7 months by a custom made EIT system with telemetry. Pigs were sacrificed after 7 months to obtain samples for histological analysis. Scar size and inflammation were evaluated on post-mortem sections.

Results
 Histopathology analysis confirmed the presence of GFP-ATDPCs in infarcted myocardium (Figure 2) and vessel formation in the bioactive smart patch (Figure 3). Vascular density was similar in myocardial infarct, border and distal zones in both groups. Inflammation state assessed by CD45/CD31 ratio showed significant differences between treated and control groups (0.4x10⁴ and 0.2x10⁴ respectively, P<0.005) (Figure 4). Impedance magnitudes at low and high frequency were initially unpaired and converge after a period of 4.5-7 days, which is coherent with the scar formation. However, no significant differences were found between groups.

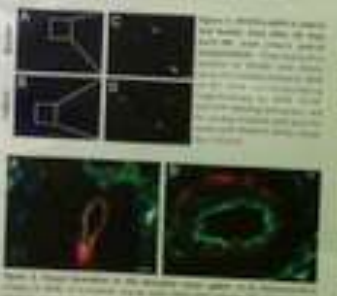


Fig. 2. GFP-ATDPCs in the infarcted myocardium. The images show GFP-ATDPCs in the infarcted myocardium. The images show GFP-ATDPCs in the infarcted myocardium.

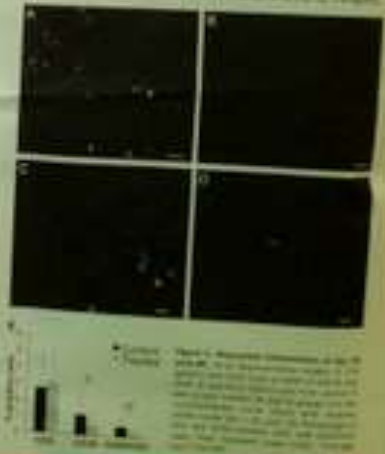


Fig. 4. Inflammation state assessed by CD45/CD31 ratio. The images show CD45 (green) and CD31 (red) in various sections of the heart. The bar graph shows the CD45/CD31 ratio for treated and control groups.

Conclusions
 Decellularized human pericardium is an effective biological scaffold for ATDPCs delivery into the myocardial scar with new integrated vessels in vivo. ATDPCs minimize the inflammation in myocardial ischemia. This is the first study demonstrating on-line EIT measurements in vivo.

Clinical impact of a chronic total occlusion in a non-infarct-related coronary artery in patients with ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention

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BACKGROUND

In spite of recent advances in the treatment of ST-segment elevation acute myocardial infarction (STEMI), some groups of patients still experience considerable morbidity and mortality after a STEMI. Previous reports showed conflicting results regarding the prognostic impact of a chronic total occlusion (CTO) in a non-infarct-related coronary artery.

RESULTS

1179 patients were included in the study. 125 (10.6%) had at least one CTO in a non-infarct-related artery. Mean follow-up was 329 days.

1. BASELINE CHARACTERISTICS

	CTO (n=125)	No CTO (n=1054)	p
Age	65.3 (12.2)	61.4 (12.2)	0.002
Male	69 (55.2%)	594 (56.2%)	0.908
Hypertension	64 (51.2%)	594 (56.2%)	0.002
Cholesterol elevation (preMI)	61.8 (50.2%)	56.1 (52.3%)	0.002
Previous stroke	12 (9.6%)	38 (3.6%)	0.002
Peripheral vascular disease	25 (20.0%)	11 (1.0%)	0.002
Previous myocardial infarction	58 (46.4%)	41 (3.9%)	0.002
Previous PCI	14 (11.2%)	11 (1.0%)	0.002
Previous CABG	3 (2.4%)	3 (0.3%)	0.002

PURPOSES

To analyze the prevalence and the clinical impact of a CTO in a non-infarct-related artery in patients with STEMI treated with primary percutaneous coronary intervention (PCI).

METHODS

We prospectively included all patients with STEMI that underwent primary PCI at our institution from October 2009 to April 2012.

2. SEVERITY OF INDEX HOSPITALIZATION

	CTO (n=125)	No CTO (n=1054)	p
Killip class at admission			0.002
I	79 (63.2%)	660 (62.4%)	
II	22 (17.6%)	120 (11.3%)	
III	11 (8.8%)	21 (2.0%)	
IV	9 (7.2%)	21 (2.0%)	
Left ventricular dysfunction at discharge (%)	64.8 (51.8%)	54.3 (51.4%)	0.002
ICU	46 (36.8%)	40 (3.8%)	0.002
Mechanical ventilation	10 (8.0%)	10 (1.0%)	0.002
Open-Heart surgery	0 (0.0%)	20 (1.9%)	0.002
In-hospital mortality	3 (2.4%)	20 (1.9%)	0.002

3. MORTALITY DURING FOLLOW-UP: Patients with a CTO had a significantly higher mortality, specially due to non-cardiac causes. We did not find a significant association between CTO and cardiac mortality in our patients. In the multivariate analysis, including Killip class at admission and left ventricular systolic fraction, the presence of a CTO was not associated with increased total mortality.

Kaplan-Meier analysis of survival



Multivariate analysis: predictors of all-cause mortality

Predictor	HR (95%CI)	p
CTO	1.34 (0.64-2.82)	0.199
Age	1.09 (1.05-1.13)	0.007
Anterior STEMI	1.54 (0.98-2.71)	0.130
Number of treated infarct-related arteries	1.35 (1.01-1.80)	0.045
LVDF (%)	0.93 (0.90-0.96)	0.001
Cholesterol elevation (preMI)	0.97 (0.94-0.99)	0.001

CONCLUSIONS

- In non-selected patients with STEMI treated with primary PCI, the presence of a CTO in a non-infarct-related artery identifies a group of patients at higher risk, with worse morbidity and worse clinical outcome.
- The higher mortality observed in patients with CTO was mainly due to non-cardiac causes.
- The presence of a CTO was not an independent predictor of mortality in our series.



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