UNIVERSITÀ DEGLI STUDI DI FERRARA
- EX LABORE FRUCTUS -

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Cosa è l'infarto?

ESC European Society of Cardiology

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EXPERT CONSENSUS DOCUMENT

Fourth universal definition of myocardial infarction (2018)

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Universal definitions of myocardial injury and myocardial infarction

Criteria for myocardial injury

The term myocardial injury should be used when there is evidence of elevated cardiac troponin values (cTn) with at least one value above the 99th percentile upper reference limit (URL). The myocardial injury is considered acute if there is a rise and/or fall of cTn values.

Criteria for acute myocardial infarction (types 1, 2 and 3 MI)

The term acute myocardial infarction should be used when there is acute myocardial injury with clinical evidence of acute myocardial ischaemia and with detection of a rise and/or fall of cTn values with at least one value above the 99th percentile URL and at least one of the following:

- Symptoms of myocardial ischaemia;
- New ischaemic ECG changes;
- Development of pathological Q waves;

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- Symptoms of myocardial ischaemia;
- New ischaemic ECG changes;
- Development of pathological Q waves;
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic aetiology;
- Identification of a coronary thrombus by angiography or autopsy (not for types 2 or 3 MIs).

Post-mortem demonstration of acute athero-thrombosis in the artery supplying the infarcted myocardium meets criteria for *type 1 MI*.
Evidence of an imbalance between myocardial oxygen supply and demand unrelated to acute athero-thrombosis meets criteria for *type 2 MI*.
Cardiac death in patients with symptoms suggestive of myocardial ischaemia and presumed new ischaemic ECG changes before cTn values become available or abnormal meets criteria for *type 3 MI*.

Criteria for coronary procedure-related myocardial infarction (types 4 and 5 MI)

Criteria for coronary procedure-related myocardial infarction (types 4 and 5 MI)

Percutaneous coronary intervention (PCI) related MI is termed *type 4a MI*.
Coronary artery bypass grafting (CABG) related MI is termed *type 5 MI*.
Coronary procedure-related MI ≤ 48 hours after the index procedure is arbitrarily defined by an elevation of cTn values > 5 times for *type 4a MI* and > 10 times for *type 5 MI* of the 99th percentile URL in patients with normal baseline values. Patients with elevated pre-procedural cTn values, in whom the pre-procedural cTn level are stable (≤ 20% variation) or falling, must meet the criteria for a > 5 or > 10 fold increase and manifest a change from the baseline value of > 20%. In addition with at least one of the following:

- New ischaemic ECG changes (this criterion is related to *type 4a MI* only);
- Development of new pathological Q waves;
- Imaging evidence of loss of viable myocardium that is presumed to be new and in a pattern consistent with an ischaemic aetiology;
- Angiographic findings consistent with a procedural flow-limiting complication such as coronary dissection, occlusion of a major epicardial artery or graft, side-branch occlusion-thrombus, disruption of collateral flow or distal embolization.

Isolated development of new pathological Q waves meets the *type 4a MI* or *type 5 MI* criteria with either revascularization procedure if cTn values are elevated and rising but less than the pre-specified thresholds for PCI and CABG.
Other types of 4 MI include *type 4b MI* stent thrombosis and *type 4c MI* restenosis that both meet *type 1 MI* criteria.
Post-mortem demonstration of a procedure-related thrombus meets the *type 4a MI* criteria or *type 4b MI* criteria if associated with a stent.

Criteria for prior or silent/unrecognized myocardial infarction

Any one of the following criteria meets the diagnosis for prior or silent/unrecognized MI:

- Abnormal Q waves with or without symptoms in the absence of non-ischaemic causes.
- Imaging evidence of loss of viable myocardium in a pattern consistent with ischaemic aetiology.
- Patho-anatomical findings of a prior MI.

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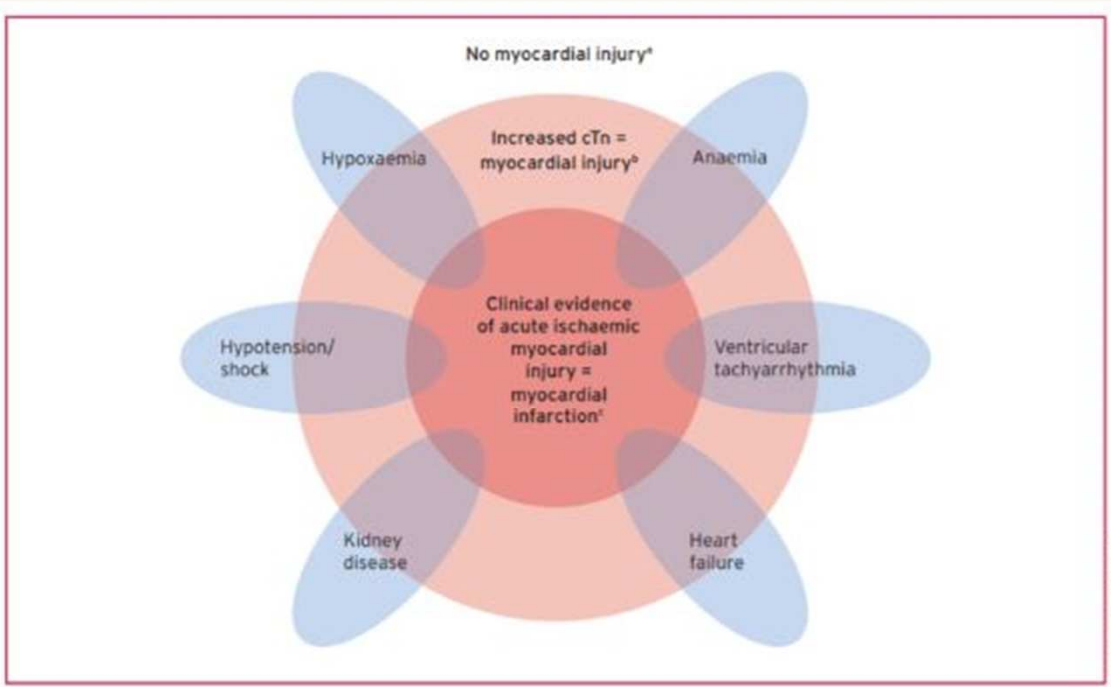


Figure 2 Spectrum of myocardial injury, ranging from no injury to myocardial infarction. Various clinical entities may involve these myocardial categories, e.g. ventricular tachyarrhythmia, heart failure, kidney disease, hypotension/shock, hypoxaemia, and anaemia. cTn = cardiac troponin; URL = upper reference limit. [a]No myocardial injury = cTn values ≤ 99th percentile URL or not detectable. [b]Myocardial injury = cTn values > 99th percentile URL. [c]Myocardial infarction = clinical evidence of myocardial ischaemia and a rise and/or fall of cTn values > 99th percentile URL.

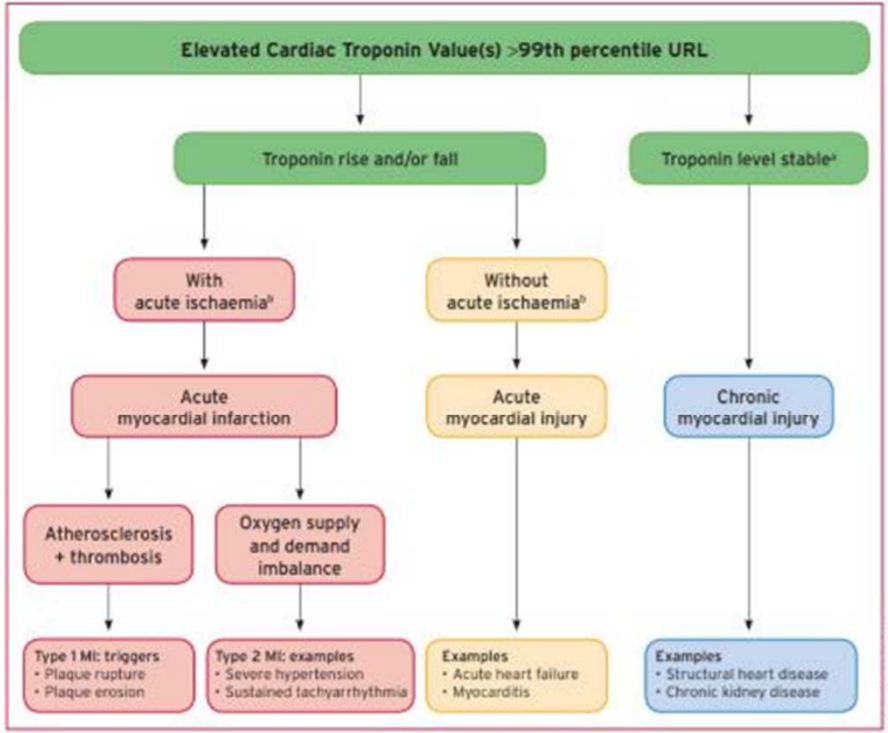
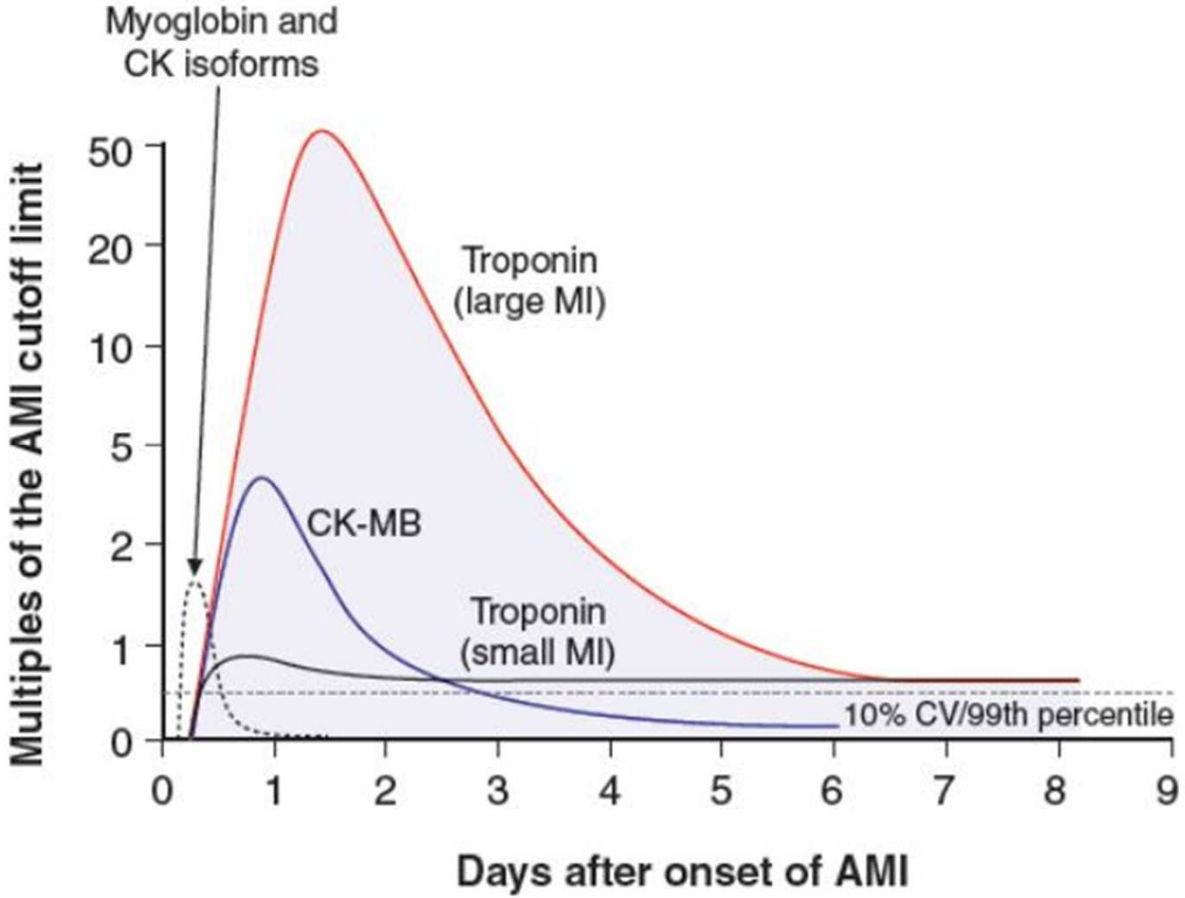


Figure 6 A model for interpreting myocardial injury. Ischaemic thresholds vary substantially in relation to the magnitude of the stressor and the extent of underlying cardiac disease. MI = myocardial infarction; URL = upper reference limit. [a]Stable denotes ≤ 20% variation of troponin values in the appropriate clinical context. [b]Ischaemia denotes signs and/or symptoms of clinical myocardial ischaemia.

Table 3 Clinical implications of high-sensitivity cardiac troponin assays

Compared with standard cardiac troponin assays, high-sensitivity assays:
• Have higher negative predictive value for acute MI.
• Reduce the "troponin-blind" interval leading to earlier detection of acute MI.
• Result in a ~4% absolute and ~20% relative increase in the detection of type 1 MI and a corresponding decrease in the diagnosis of unstable angina.
• Are associated with a 2-fold increase in the detection of type 2 MI.
Levels of high-sensitivity cardiac troponin should be interpreted as quantitative markers of cardiomyocyte damage (i.e. the higher the level, the greater the likelihood of MI):
• Elevations beyond 5-fold the upper reference limit have high (>90%) positive predictive value for acute type 1 MI.
• Elevations up to 3-fold the upper reference limit have only limited (50–60%) positive predictive value for acute MI and may be associated with a broad spectrum of conditions.
• It is common to detect circulating levels of cardiac troponin in healthy individuals.
Rising and/or falling cardiac troponin levels differentiate acute from chronic cardiomyocyte damage (the more pronounced the change, the higher the likelihood of acute MI).

MI = myocardial infarction.


Myoglobin and
CK isoforms
Troponin
(large MI)
CK-MB
Troponin
(small MI)
10% CV/99th percentile
Multiples of the AMI cutoff limit
50
20
10
5
2
1
0
0
1
2
3
4
5
6
7
8
9
Days after onset of AMI

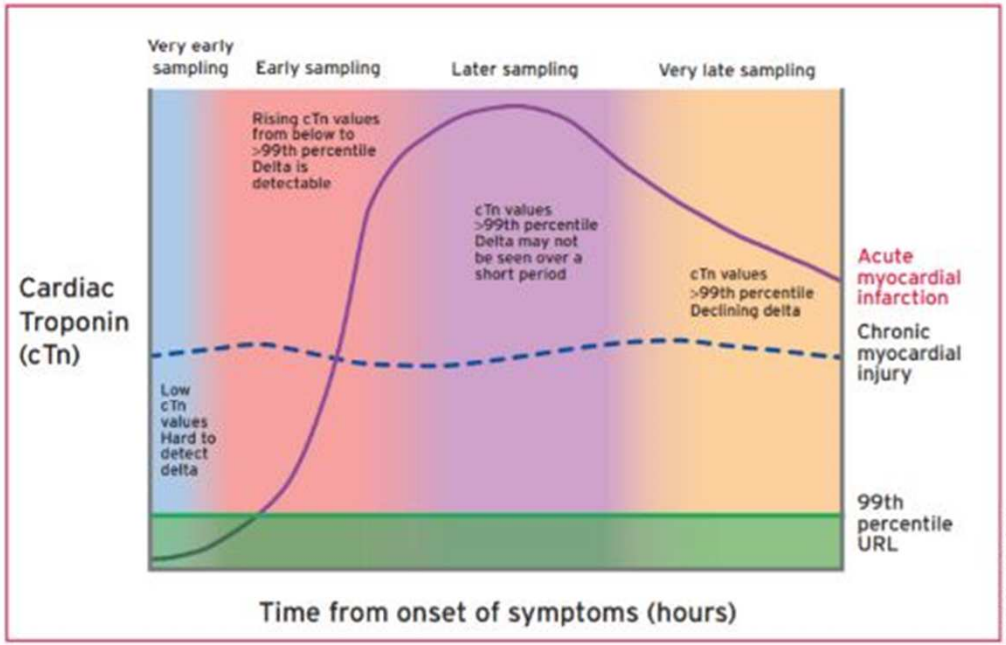


Figure 7 Illustration of early cardiac troponin kinetics in patients after acute myocardial injury including acute myocardial infarction. The timing of biomarker release into the circulation is dependent on blood flow and how soon after the onset of symptoms samples are obtained. Thus, the ability to consider small changes as diagnostic can be problematic. In addition, many comorbidities increase cTn values and, in particular, hs-cTn values, so that elevations can be present at baseline even in those with myocardial infarction who present early after the onset of symptoms. Changes in cTn values or deltas can be used to define acute compared with chronic events, and the ability to detect these is indicated in the figure. Increased cTn values can often be detected for days after an acute event. cTn = cardiac troponin; URL = upper reference limit.

Table 1 **Reasons for the elevation of cardiac troponin values because of myocardial injury**

Myocardial injury related to acute myocardial ischaemia
Atherosclerotic plaque disruption with thrombosis.
Myocardial injury related to acute myocardial ischaemia because of oxygen supply/demand imbalance
Reduced myocardial perfusion, e.g. • Coronary artery spasm, microvascular dysfunction • Coronary embolism • Coronary artery dissection • Sustained bradyarrhythmia • Hypotension or shock • Respiratory failure • Severe anaemia
Increased myocardial oxygen demand, e.g. • Sustained tachyarrhythmia • Severe hypertension with or without left ventricular hypertrophy
Other causes of myocardial injury
Cardiac conditions, e.g. • Heart failure • Myocarditis • Cardiomyopathy (any type) • Takotsubo syndrome • Coronary revascularization procedure • Cardiac procedure other than revascularization • Catheter ablation • Defibrillator shocks • Cardiac contusion
Systemic conditions, e.g. • Sepsis, infectious disease • Chronic kidney disease • Stroke, subarachnoid haemorrhage • Pulmonary embolism, pulmonary hypertension • Infiltrative diseases, e.g. amyloidosis, sarcoidosis • Chemotherapeutic agents • Critically ill patients • Strenuous exercise

For a more comprehensive listing, see[9–41]

Criteria for type 1 MI

Detection of a rise and/or fall of cTn values with at least one value above the 99th percentile URL and with at least one of the following:

- Symptoms of acute myocardial ischaemia;
- New ischaemic ECG changes;
- Development of pathological Q waves;
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic aetiology;
- Identification of a coronary thrombus by angiography including intracoronary imaging or by autopsy.[a]

cTn = cardiac troponin; ECG = electrocardiogram; URL = upper reference limit.

[a]Post-mortem demonstration of an atherothrombus in the artery supplying the infarcted myocardium, or a macroscopically large circumscribed area of necrosis with or without intramyocardial haemorrhage, meets the type 1 MI criteria regardless of cTn values.

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ESC GUIDELINES

2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC)

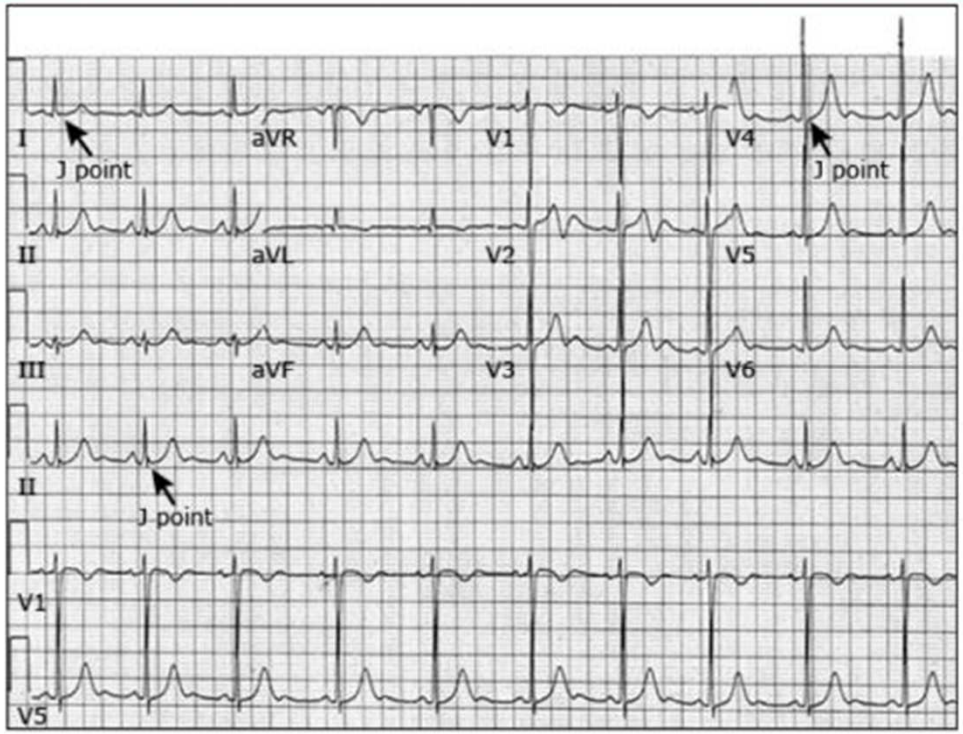
Authors/Task Force Members: Borja Ibanez* (Chairperson) (Spain), Stefan James* (Chairperson) (Sweden), Stefan Agewall (Norway), Manuel J. Antunes (Portugal), Chiara Bucciarelli-Ducci (UK), Héctor Bueno (Spain), Alida L. P. Caforio (Italy), Filippo Crea (Italy), John A. Goudevenos (Greece), Sigrun Halvorsen (Norway), Gerhard Hindricks (Germany), Adnan Kastrati (Germany), Mattie J. Lenzen (The Netherlands), Eva Prescott (Denmark), Marco Roffi (Switzerland), Marco Valgimigli (Switzerland), Christoph Varenhorst (Sweden), Pascal Vranckx (Belgium), Petr Widimský (Czech Republic)

Document Reviewers: Jean-Philippe Collet (CPG Review Coordinator) (France), Steen Dalby Kristensen (CPG Review Coordinator) (Denmark), Victor Aboyans (France),

ST elevation

New ST elevation at the J point in two contiguous leads with the cut-points: ≥0.1 mV in all leads other than leads V_2–V_3 where the following cut points apply: ≥0.2 mV in men ≥40 years; ≥0.25 mV in men <40 years, or ≥0.15 mV in women.

J point



The J point is the junction between the end of the QRS and the beginning of the ST segment.

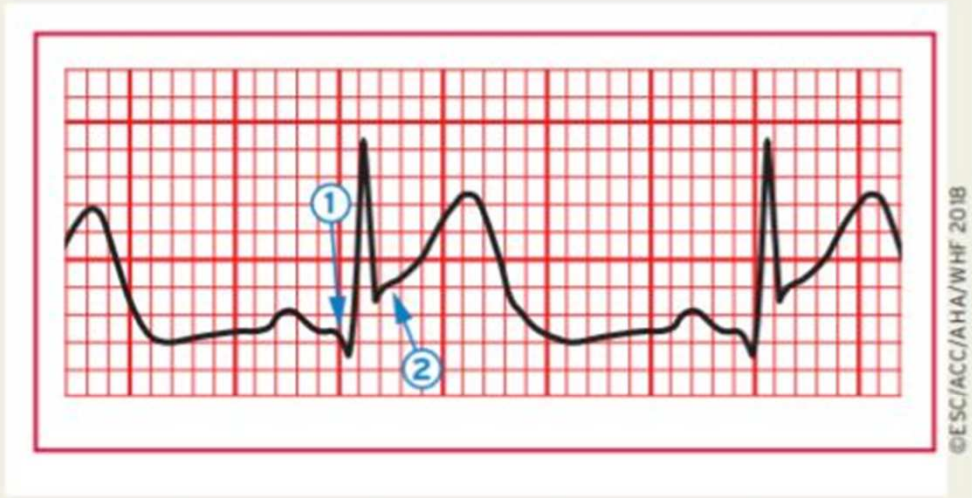
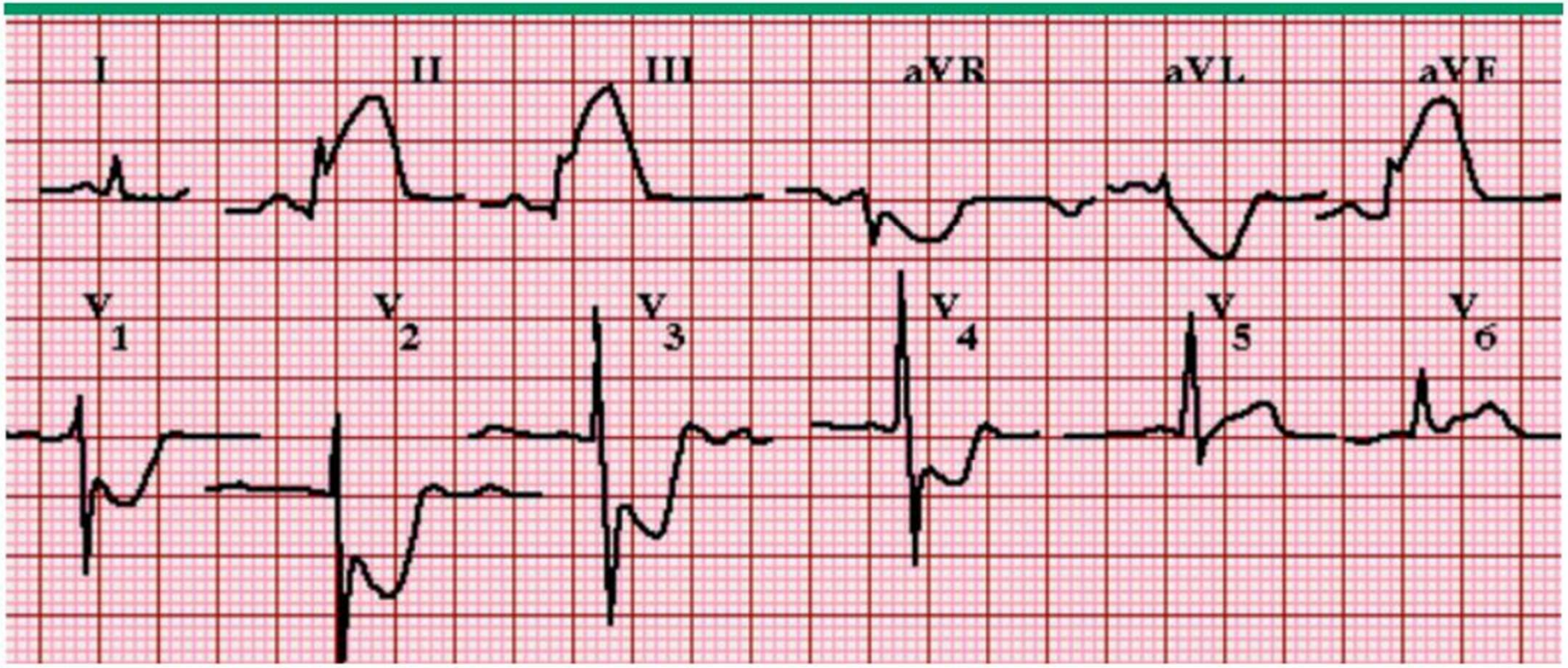
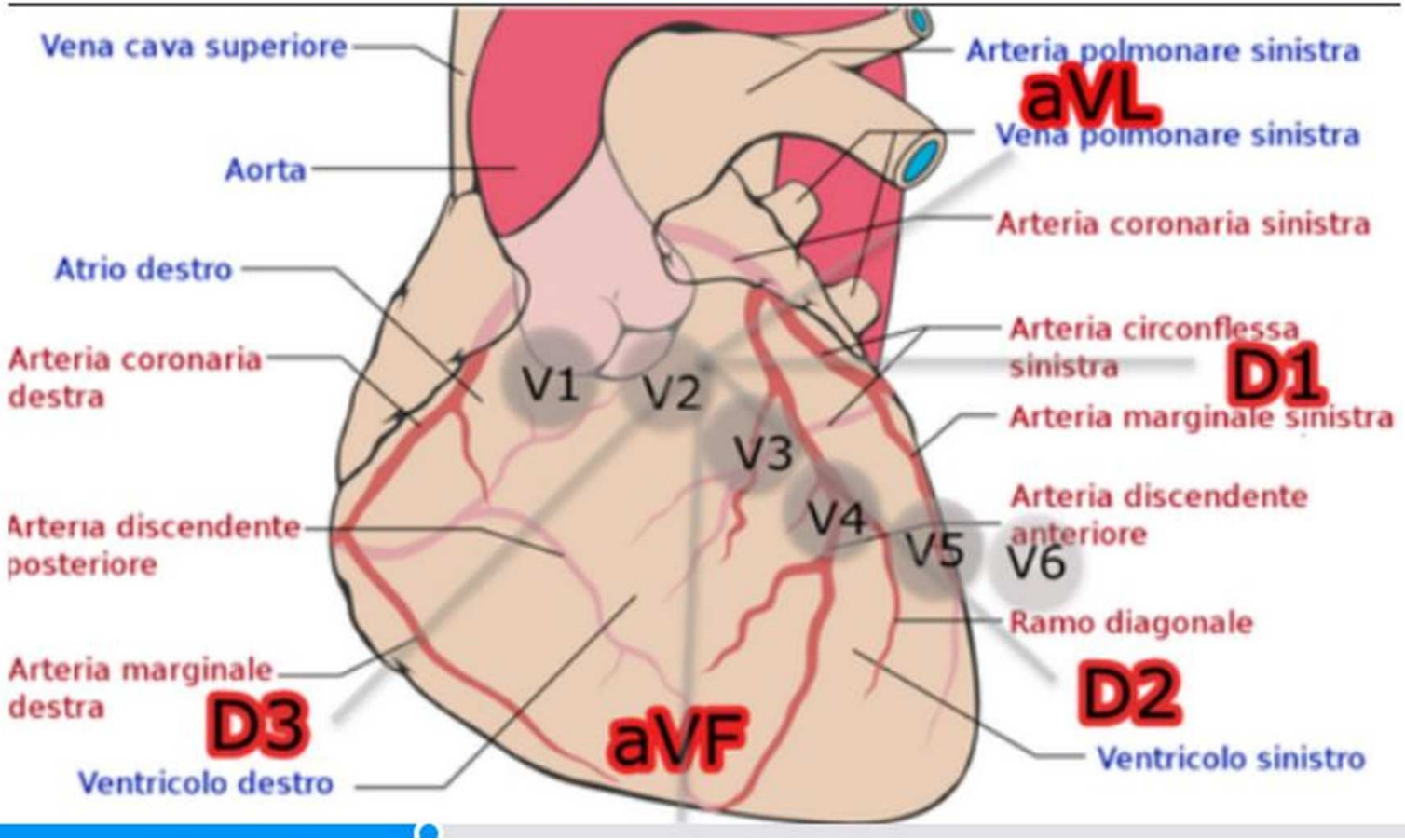


Figure 8 Electrocardiogram example of ST-segment elevation. The initial onset of the Q wave shown by arrow 1 serves as the reference point and arrow 2 shows the onset of the ST-segment or J-point. The difference between the two identifies the magnitude of displacement. Measurements of both arrows should be made from the top of the electrocardiogram line tracing.


I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6


Vena cava superiore
Aorta
Atrio destro
Arteria coronaria destra
Arteria discendente posteriore
Arteria marginale destra
D3
Ventricolo destro
aVF
Arteria polmonare sinistra
aVL
Vena polmonare sinistra
Arteria coronaria sinistra
Arteria circonflessa sinistra
D1
Arteria marginale sinistra
Arteria discendente anteriore
Ramo diagonale
D2
Ventricolo sinistro
V1
V2
V3
V4
V5
V6

Incidenza

- La principale causa singola di decesso, con incidenza globale in crescita
- Almeno 1.8 milioni di morti ogni anno, di cui 20% in Europa
- STEMI: incidenza di 58 casi ogni 100.000 persone/ anno

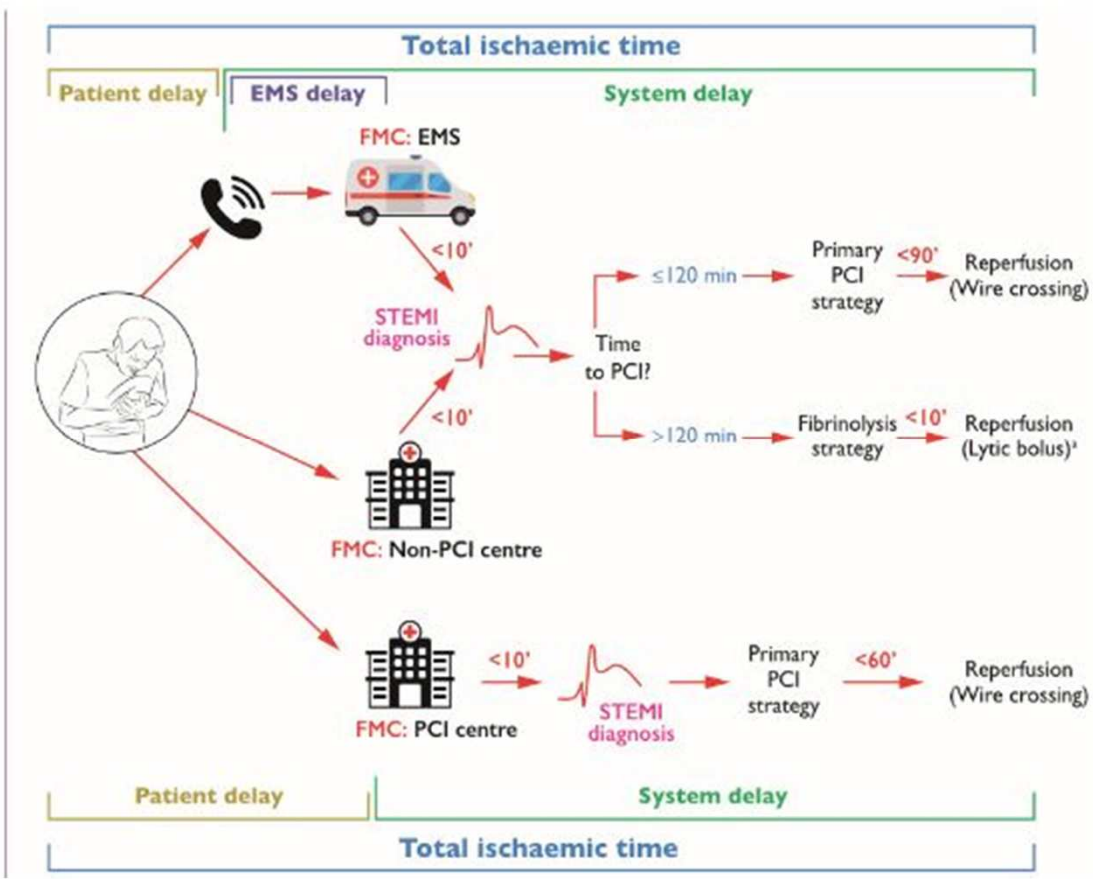
Mortalità influenzata da:

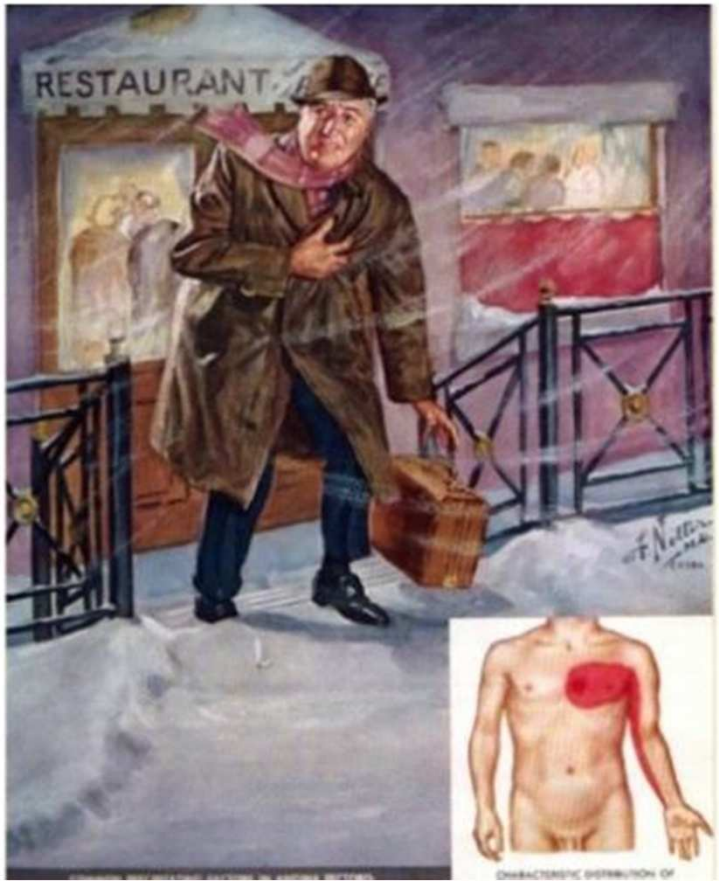
- Età avanzata
- Classi Killip
- Ritardo nel trattamento
- Strategia di trattamento
- Storia di IMA
- Diabete Mellito
- Insufficienza Renale Cronica

Classificazione di Killip e Kimball

Class		Findings
I	No heart failure	Absence of rales over the lung fields and absence of S3.
II	Heart Failure	Rales over 50% or less of the lung fields or the presence of an S3 and signs of pulmonary venous hypertension
III	Severe Heart Failure	Rales over more than 50% of the lung fields.
IV	Cardiogenic Shock	Hypotension (SBP < 90 mmHg for at least 30 minutes or the need for supportive measures), end-organ hypoperfusion (cool extremities or a urine output of less than 30 ml/h, and a heart rate of greater than or equal to 60 beats per minute); cyanosis.

Killip T & Kimball JT, Am J Cardiol 1967; 20(4):457–464.

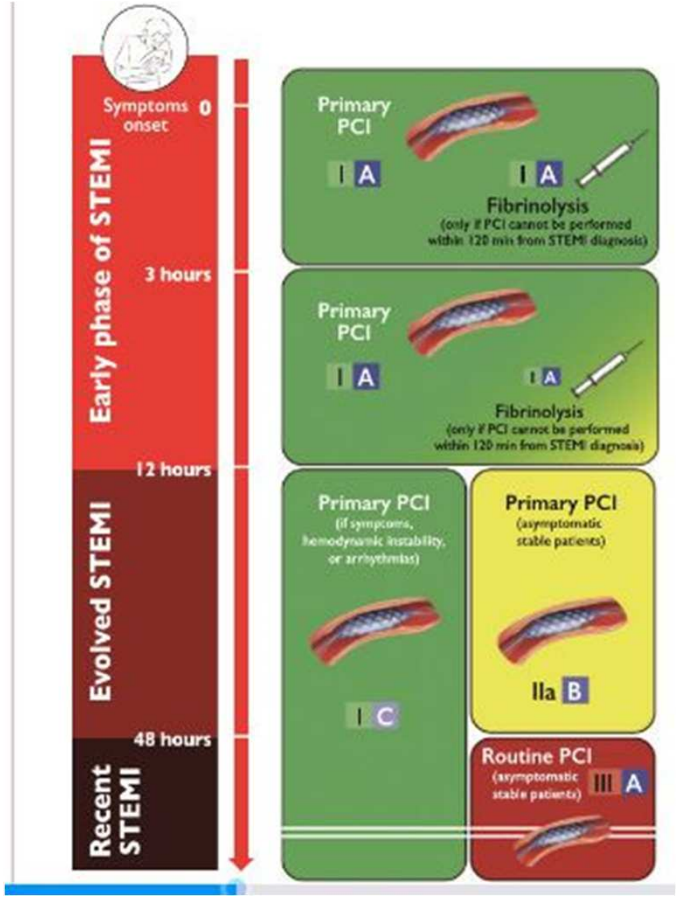

Total ischaemic time
Patient delay
EMS delay
System delay
FMC: EMS
<10'
STEMI diagnosis
<10'
Time to PCI?
≤120 min
Primary PCI strategy
<90'
Reperfusion (Wire crossing)
>120 min
Fibrinolysis strategy
<10'
Reperfusion (Lytic bolus)[a]
FMC: Non-PCI centre
FMC: PCI centre
<10'
STEMI diagnosis
Primary PCI strategy
<60'
Reperfusion (Wire crossing)
Patient delay
System delay
Total ischaemic time

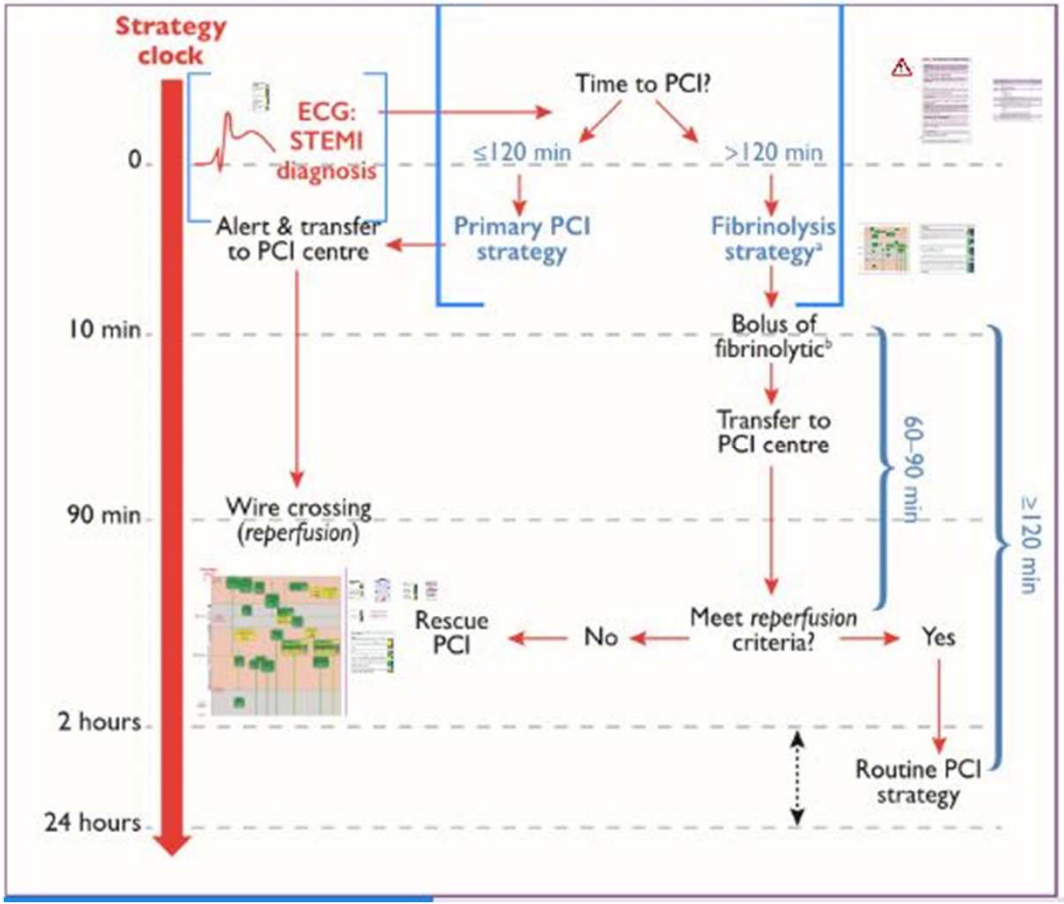

RESTAURANT

Localizzazione	punti
Retrosternale, Precordiale,	+ 3
Emitorace Sn, Collo, Mandibola, Epigastrio	+2
Apice	+1
Carattere	
Oppressione, Morsa, Lacerazione / Strappamento	+3
Pesantezza, Restringimento	+2
Puntorio, Pinzettatura, Pleurico	+1
Irradiazione	
Braccia, spalla, posteriore, collo, mandibola	+1
Sintomi Associati	
Dispnea, Sudorazione, Nausea	+2

Score <4 dolore probabilmente ATIPICO **con bassa probabilità di origine coronarica**

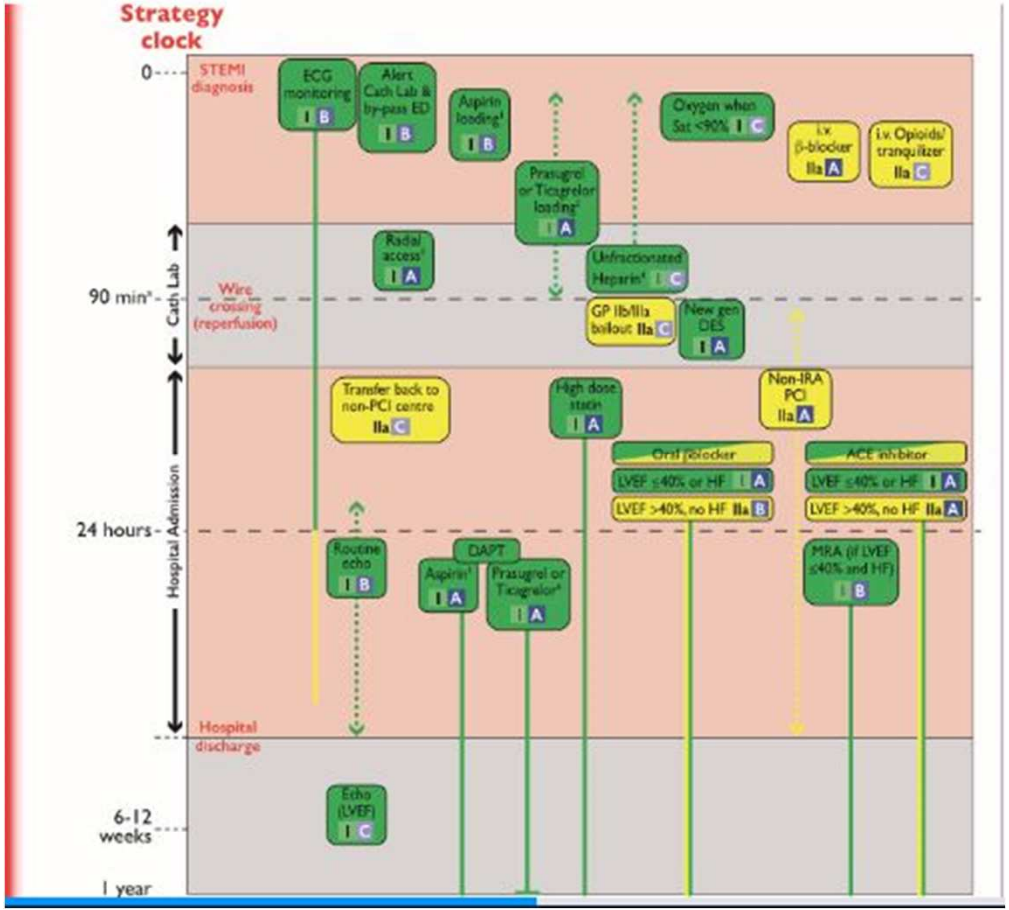
Score >4 dolore TIPICO **con probabilità di origine coronarica**


Symptoms onset
0
Early phase of STEMI
3 hours
12 hours
Evolved STEMI
48 hours
Recent STEMI
Primary PCI
I A
I A
Fibrinolysis
(only if PCI cannot be performed within 120 min from STEMI diagnosis)
Primary PCI
I A
I A
Fibrinolysis
(only if PCI cannot be performed within 120 min from STEMI diagnosis)
Primary PCI
(if symptoms, hemodynamic instability, or arrhythmias)
I C
Primary PCI
(asymptomatic stable patients)
IIa B
Routine PCI
(asymptomatic stable patients)
III A


Strategy clock
0
10 min
90 min
2 hours
24 hours
ECG: STEMI diagnosis
Time to PCI?
≤120 min
>120 min
Alert & transfer to PCI centre
Primary PCI strategy
Fibrinolysis strategy[a]
Bolus of fibrinolytic[b]
Transfer to PCI centre
60–90 min
≥120 min
Wire crossing (reperfusion)
Meet reperfusion criteria?
No
Rescue PCI
Yes
Routine PCI strategy

Recommendations for initial diagnosis

Recommendations	Class[a]	Level[b]
ECG monitoring		
12-lead ECG recording and interpretation is indicated as soon as possible at the point of FMC, with a maximum target delay of 10 min.[36,38]	I	B
ECG monitoring with defibrillator capacity is indicated as soon as possible in all patients with suspected STEMI.[44,45]	I	B
The use of additional posterior chest wall leads (V_7–V_9) in patients with high suspicion of posterior MI (circumflex occlusion) should be considered.[8,46–49]	IIa	B
The use of additional right precordial leads (V_3R and V_4R) in patients with inferior MI should be considered to identify concomitant RV infarction.[8,43]	IIa	B
Blood sampling		
Routine blood sampling for serum markers is indicated as soon as possible in the acute phase but should not delay reperfusion treatment.[8]	I	C


Strategy clock
0
STEMI diagnosis
ECG monitoring I B
Alert Cath Lab & by-pass ED I B
Aspirin loading* I B
Prasugrel or Ticagrelor loading* I A
Oxygen when Sat <90% I C
I.v. β-blocker IIa A
I.v. Opioids/ tranquilizer IIa C
Radial access* I A
Unfractionated Heparin* I C
90 min*
Cath Lab
Wire crossing (reperfusion)
GP IIb/IIIa bailout IIa C
New gen DES I A
Transfer back to non-PCI centre IIa C
High dose statin I A
Non-IRA PCI IIa A
Oral β-blocker
LVEF ≤40% or HF I A
LVEF >40%, no HF IIa B
ACE inhibitor
LVEF ≤40% or HF I A
LVEF >40%, no HF IIa A
24 hours
Hospital Admission
Routine echo I B
DAPT
Aspirin* I A
Prasugrel or Ticagrelor* I A
MRA (if LVEF ≤40% and HF) I B
Hospital discharge
Echo (LVEF) I C
6-12 weeks
1 year

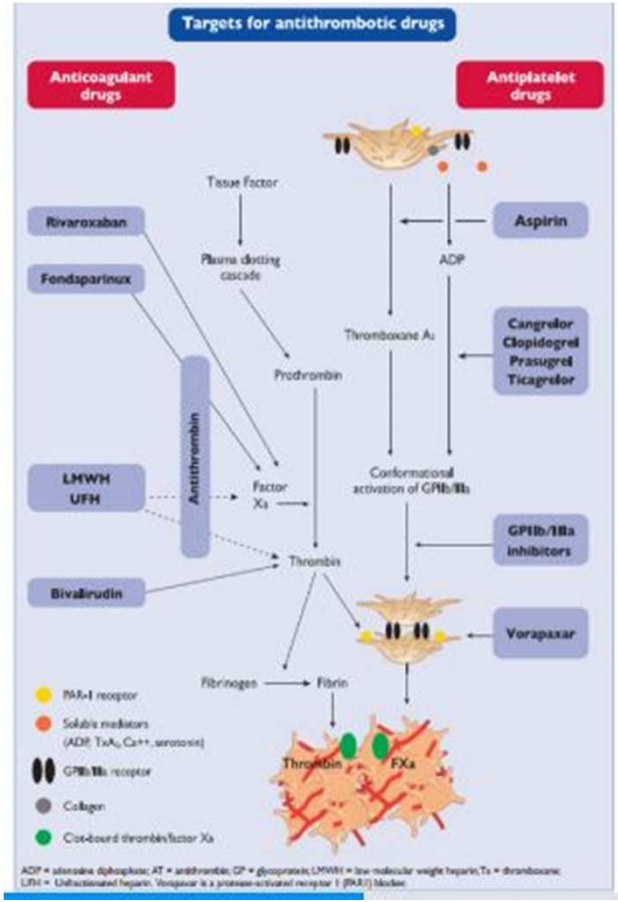
Relief of hypoxaemia and symptoms

Recommendations	Class[a]	Level[b]
Hypoxia		
Oxygen is indicated in patients with hypoxaemia (SaO_2 < 90% or PaO_2 < 60 mmHg).	I	C
Routine oxygen is not recommended in patients with $SaO_2 \geq 90\%$.[64–66]	III	B
Symptoms		
Titrated i.v. opioids should be considered to relieve pain.	IIa	C
A mild tranquillizer (usually a benzodiazepine) should be considered in very anxious patients.	IIa	C

i.v. = intravenous; PaO_2 = partial pressure of oxygen; SaO_2 = arterial oxygen saturation.

[a]Class of recommendation.

[b]Level of evidence.

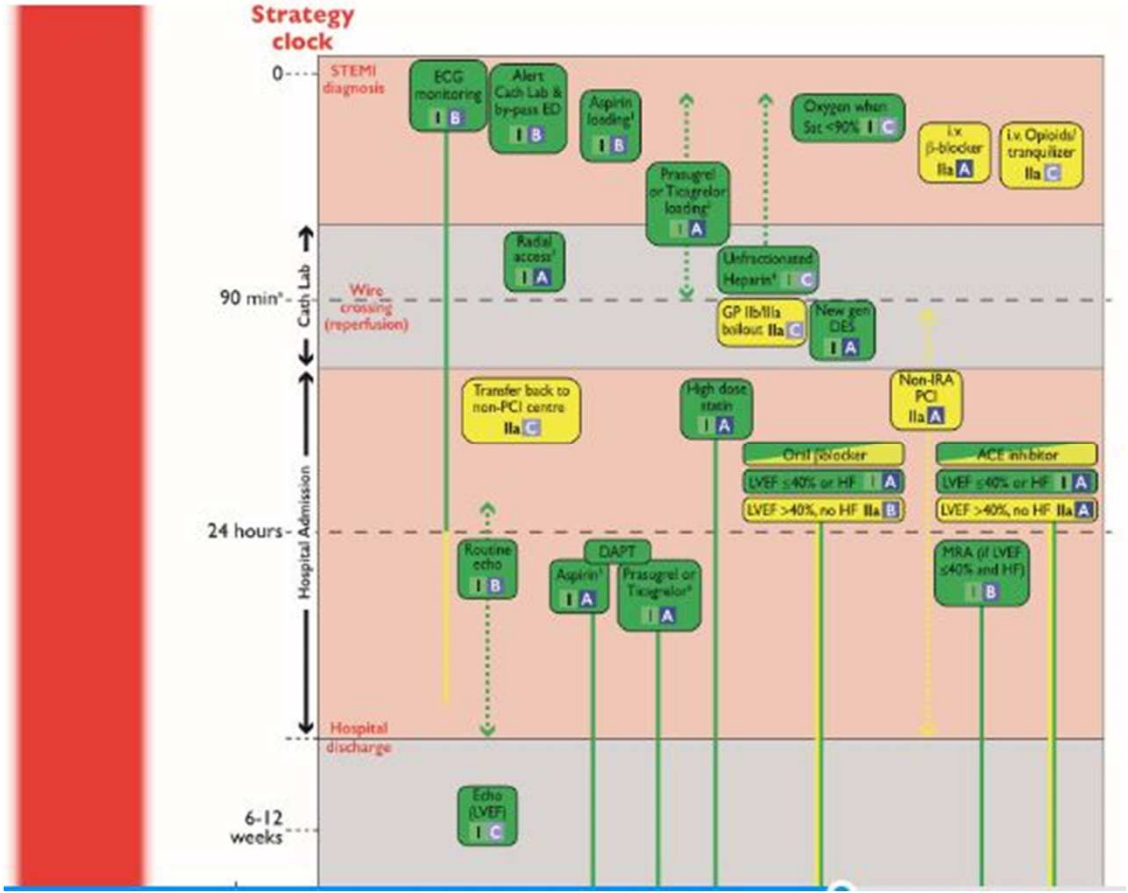

Targets for antithrombotic drugs
Anticoagulant drugs
Antiplatelet drugs
Tissue Factor
Rivaroxaban
Fondaparinux
Plasma clotting cascade
Aspirin
ADP
Cangrelor
Clopidogrel
Prasugrel
Ticagrelor
Thromboxane A2
Prothrombin
Antithrombin
LMWH
UFH
Factor Xa
Conformational activation of GPIIb/IIIa
GPIIb/IIIa inhibitors
Thrombin
Bivalirudin
Vorapaxar
Fibrinogen
Fibrin
PAR-1 receptor
Soluble mediators (ADP, TxA2, Ca++, serotonin)
GPIIb/IIIa receptor
Collagen
Clot-bound thrombin/factor Xa
Thrombin
FXa
ADP = adenosine diphosphate; AT = antithrombin; GP = glycoprotein; LMWH = low-molecular weight heparin; TxA2 = thromboxane; UFH = Unfractionated heparin. Vorapaxar is a protease-activated receptor 1 (PAR1) blocker.

Recommendations	Class[b]	Level[c]
Antiplatelet therapy		
A potent $P2Y_{12}$ inhibitor (prasugrel or ticagrelor), or clopidogrel if these are not available or are contraindicated, is recommended before (or at latest at the time of) PCI and maintained over 12 months, unless there are contraindications such as excessive risk of bleeding.[186,187]	I	A
Aspirin (oral or i.v. if unable to swallow) is recommended as soon as possible for all patients without contraindications.[213,214]	I	B
GP IIb/IIIa inhibitors should be considered for bailout if there is evidence of no-reflow or a thrombotic complication.	IIa	C
Cangrelor may be considered in patients who have not received $P2Y_{12}$ receptor inhibitors.[192–194]	IIb	A

Doses of antiplatelet and parenteral anticoagulant co-therapies in primary PCI	
Antiplatelet therapies	
Aspirin	Loading dose of 150–300 mg orally or of 75–250 mg i.v. if oral ingestion is not possible, followed by a maintenance dose of 75–100 mg/day
Clopidogrel	Loading dose of 600 mg orally, followed by a maintenance dose of 75 mg/day
Prasugrel	Loading dose of 60 mg orally, followed by a maintenance dose of 10 mg/day In patients with body weight ≤60 kg, a maintenance dose of 5 mg/day is recommended Prasugrel is contra-indicated in patients with previous stroke. In patients ≥75 years, prasugrel is generally not recommended, but a dose of 5 mg/day should be used if treatment is deemed necessary
Ticagrelor	Loading dose of 180 mg orally, followed by a maintenance dose of 90 mg *b.i.d.*
Abciximab	Bolus of 0.25 mg/kg i.v. and 0.125 μg/kg/min infusion (maximum 10 μg/min) for 12 hours
Eptifibatide	Double bolus of 180 μg/kg i.v. (given at a 10-min interval) followed by an infusion of 2.0 μg/kg/min for up to 18 hours
Tirofiban	25 μg/kg over 3 min i.v., followed by a maintenance infusion of 0.15 μg/kg/min for up to 18 hours

	Anticoagulant therapy		
→	Anticoagulation is recommended for all patients in addition to antiplatelet therapy during primary PCI.	I	C
→	Routine use of UFH is recommended.	I	C
	In patients with heparin-induced thrombocytopenia, bivalirudin is recommended as the anticoagulant agent during primary PCI.	I	C
→	Routine use of enoxaparin i.v. should be considered.[200–202]	IIa	A
	Routine use of bivalirudin should be considered.[209,215]	IIa	A
	Fondaparinux is not recommended for primary PCI.[199]	III	B

Parenteral anticoagulant therapies	
UFH	70–100 IU/kg i.v. bolus when no GP IIb/IIIa inhibitor is planned 50–70 IU/kg i.v. bolus with GP IIb/IIIa inhibitors
Enoxaparin	0.5 mg/kg i.v. bolus
Bivalirudin	0.75 mg/kg i.v. bolus followed by i.v. infusion of 1.75 mg/kg/hour for up to 4 hours after the procedure


Strategy clock
0
STEMI diagnosis
ECG monitoring I B
Alert Cath Lab & bypass ED I B
Aspirin loading¹ I B
Prasugrel or Ticagrelor loading² I A
Oxygen when Sat <90% I C
i.v. β-blocker IIa A
i.v. Opioids/ tranquilizer IIa C
Radial access³ I A
Unfractionated Heparin⁴ I C
90 min*
Cath Lab
Wire crossing (reperfusion)
GP IIb/IIIa bailout IIa C
New gen DES I A
Transfer back to non-PCI centre IIa C
High dose statin I A
Non-IRA PCI IIa A
Oral β-blocker
LVEF ≤40% or HF I A
LVEF >40%, no HF IIa B
ACE inhibitor
LVEF ≤40% or HF I A
LVEF >40%, no HF IIa A
24 hours
Hospital Admission
Routine echo I B
DAPT
Aspirin⁵ I A
Prasugrel or Ticagrelor⁶ I A
MRA (if LVEF ≤40% and HF) I B
Hospital discharge
Echo (LVEF) I C
6-12 weeks

Routine therapies in the acute, subacute, and long-term phases: beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, mineralocorticoid receptor antagonists, and lipid-lowering treatments after ST-elevation myocardial infarction

Recommendations	Class[a]	Level[b]
Beta-blockers		
Oral treatment with beta-blockers is indicated in patients with heart failure and/or LVEF ≤40% unless contraindicated.[317–361]	I	A
Intravenous beta-blockers should be considered at the time of presentation in patients undergoing primary PCI without contraindications, with no signs of acute heart failure, and with an SBP >120 mmHg.[246–248,350,403]	IIa	A
Routine oral treatment with beta-blockers should be considered during hospital stay and continued thereafter in all patients without contraindications.[361,364–366,403–405]	IIa	B
Intravenous beta-blockers must be avoided in patients with hypotension, acute heart failure or AV block, or severe bradycardia.[311]	III	B
Lipid lowering therapies		
It is recommended to start high-intensity statin therapy* as early as possible, unless contraindicated, and maintain it long-term.[365,366,368]	I	A
An LDL-C goal of < 1.8 mmol/L (70 mg/dL) or a reduction of at least 50% if the baseline LDL-C is between 1.8–3.5 mmol/L (70–135 mg/dL) is recommended.[367,369,376,382]	I	B
It is recommended to obtain a lipid profile in all STEMI patients as soon as possible after presentation.[369,406]	I	C
In patients with LDL-C ≥1.8 mmol/L (≥70 mg/dL) despite a maximally tolerated statin dose who remain at high risk, further therapy to reduce LDL-C should be considered.[376,382]	IIa	A
ACE inhibitors/ARBs		
ACE inhibitors are recommended, starting within the first 24 h of STEMI in patients with evidence of heart failure, LV systolic dysfunction, diabetes, or an anterior infarct.[383]	I	A
An ARB, preferably valsartan, is an alternative to ACE inhibitors in patients with heart failure and/or LV systolic dysfunction, particularly those who are intolerant of ACE inhibitors.[356,407]	I	B
ACE inhibitors should be considered in all patients in the absence of contraindications.[384,385]	IIa	A
MRAs		
MRAs are recommended in patients with an LVEF ≤40% and heart failure or diabetes, who are already receiving an ACE inhibitor and a beta-blocker, provided there is no renal failure or hyperkalaemia.[387]	I	B

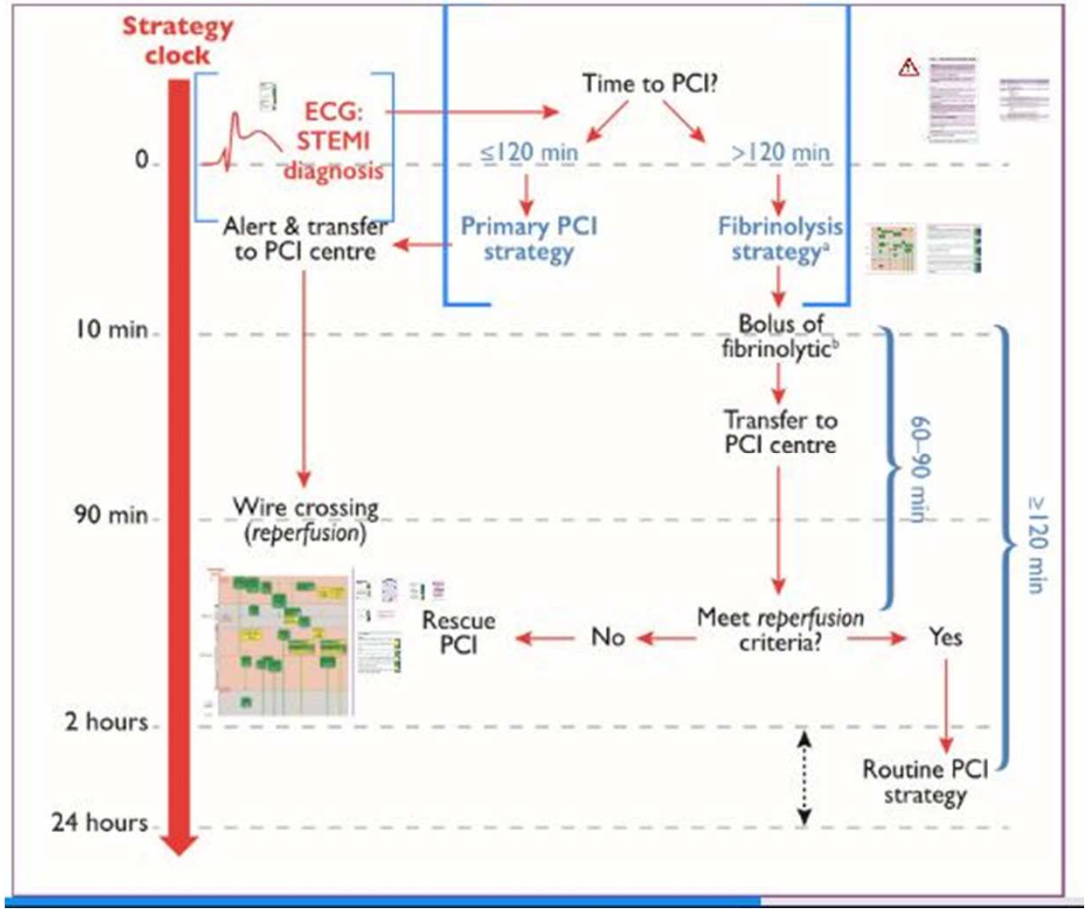
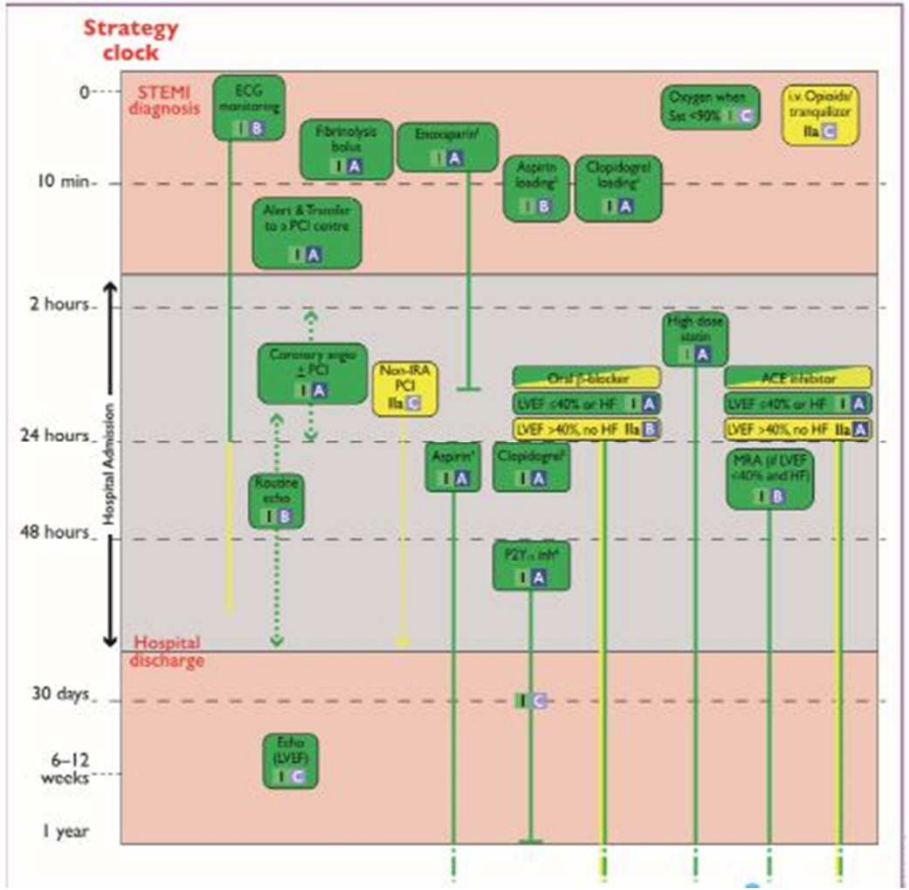

Strategy clock
0
10 min
90 min
2 hours
24 hours
ECG: STEMI diagnosis
Time to PCI?
≤120 min
>120 min
Primary PCI strategy
Fibrinolysis strategy[a]
Alert & transfer to PCI centre
Bolus of fibrinolytic[b]
Transfer to PCI centre
Wire crossing (reperfusion)
Meet reperfusion criteria?
No
Yes
Rescue PCI
Routine PCI strategy
60–90 min
≥120 min

Table 8 **Contra-indications to fibrinolytic therapy**

Absolute
Previous intracranial haemorrhage or stroke of unknown origin at anytime
Ischaemic stroke in the preceding 6 months
Central nervous system damage or neoplasms or arteriovenous malformation
Recent major trauma/surgery/head injury (within the preceding month)
Gastrointestinal bleeding within the past month
Known bleeding disorder (excluding menses)
Aortic dissection
Non-compressible punctures in the past 24 hours (e.g. liver biopsy, lumbar puncture)
Relative
Transient ischaemic attack in the preceding 6 months
Oral anticoagulant therapy
Pregnancy or within 1 week postpartum
Refractory hypertension (SBP >180 mmHg and/or DBP >110 mmHg)
Advanced liver disease
Infective endocarditis
Active peptic ulcer
Prolonged or traumatic resuscitation

DBP = diastolic blood pressure; SBP = systolic blood pressure.

Drug	Initial treatment	Specific contra-indications
Doses of fibrinolytic therapy		
Streptokinase	1.5 million units over 30–60 min i.v.	Previous treatment with streptokinase or anistreplase
Alteplase (tPA)	15 mg i.v. bolus 0.75 mg/kg i.v. over 30 min (up to 50 mg) then 0.5 mg/kg i.v. over 60 min (up to 35 mg)	
Reteplase (rPA)	10 units + 10 units i.v. bolus given 30 min apart	
Tenecteplase (TNK-tPA)	Single i.v. bolus: 30 mg (6000 IU) if <60 kg 35 mg (7000 IU) if 60 to <70 kg 40 mg (8000 IU) if 70 to <80 kg 45 mg (9000 IU) if 80 to <90 kg 50 mg (10000 IU) if ≥90 kg It is recommended to reduce to half-dose in patients ≥75 years of age.[121]	
Doses of antiplatelet co-therapies		
Aspirin	Starting dose of 150–300 mg orally (or 75–250 mg intravenously if oral ingestion is not possible), followed by a maintenance dose of 75–100 mg/day	
Clopidogrel	Loading dose of 300 mg orally, followed by a maintenance dose of 75 mg/day. In patients ≥75 years of age: loading dose of 75 mg, followed by a maintenance dose of 75 mg/day.	
Doses of anticoagulant co-therapies		
Enoxaparin	In patients <75 years of age: 30 mg i.v. bolus followed 15 min later by 1 mg/kg s.c. every 12 hours until revascularization or hospital discharge for a maximum of 8 days. The first two s.c. doses should not exceed 100 mg per injection. In patients ≥75 years of age: no i.v. bolus; start with first s.c. dose of 0.75 mg/kg with a maximum of 75 mg per injection for the first two s.c. doses. In patients with eGFR <30 mL/min/1.73 m^2, regardless of age, the s.c. doses are given once every 24 hours.	
UFH	60 IU/kg i.v. bolus with a maximum of 4000 IU followed by an i.v. infusion of 12 IU/kg with a maximum of 1000 IU/hour for 24–48 hours. Target aPTT: 50–70 s or 1.5 to 2.0 times that of control to be monitored at 3, 6, 12 and 24 hours.	
Fondaparinux (only with streptokinase)	2.5 mg i.v. bolus followed by a s.c. dose of 2.5 mg once daily up to 8 days or hospital discharge.	


Strategy clock
0
STEMI diagnosis
ECG monitoring I B
Fibrinolysis bolus I A
Enoxaparin I A
Oxygen when Sat <90% I C
i.v. Opioids/ tranquilizer IIa C
Aspirin loading I B
Clopidogrel loading I A
10 min
Alert & Transfer to a PCI centre I A
2 hours
Hospital Admission
Coronary angio ± PCI I A
Non-IRA PCI IIa C
Oral β-blocker
LVEF ≤40% or HF I A
LVEF >40%, no HF IIa B
High dose statin I A
ACE inhibitor
LVEF ≤40% or HF I A
LVEF >40%, no HF IIa A
24 hours
Routine echo I B
Aspirin I A
Clopidogrel I A
MRA (if LVEF ≤40% and HF) I B
48 hours
P2Y12 inh* I A
Hospital discharge
30 days
I C
Echo (LVEF) I C
6–12 weeks
1 year

Fibrinolytic therapy

Recommendations	Class[a]	Level[b]
When fibrinolysis is the reperfusion strategy, it is recommended to initiate this treatment as soon as possible after STEMI diagnosis, preferably in the pre-hospital setting.[76,90,123,222]	I	A
A fibrin-specific agent (i.e. tenecteplase, alteplase, or reteplase) is recommended.[223,224]	I	B
A half-dose of tenecteplase should be considered in patients ≥75 years of age.[121]	IIa	B
Antiplatelet co-therapy with fibrinolysis		
Oral or i.v. aspirin is indicated.[213]	I	B
Clopidogrel is indicated in addition to aspirin.[225,226]	I	A
DAPT (in the form of aspirin plus a $P2Y_{12}$ inhibitor[c]) is indicated for up to 1 year in patients undergoing fibrinolysis and subsequent PCI.	I	C
Anticoagulation co-therapy with fibrinolysis		
Anticoagulation is recommended in patients treated with lytics until revascularization (if performed) or for the duration of hospital stay up to 8 days.[199,234,237–239] The anticoagulant can be:	I	A
• Enoxaparin i.v. followed by s.c. (preferred over UFH).[237–242]	I	A
• UFH given as a weight-adjusted i.v. bolus followed by infusion.[234]	I	B
• In patients treated with streptokinase: fondaparinux i.v. bolus followed by an s.c. dose 24 h later.[199,233]	IIa	B
Transfer after fibrinolysis		
Transfer to a PCI-capable centre following fibrinolysis is indicated in all patients immediately after fibrinolysis.[121,124,126–130,234]	I	A
Interventions following fibrinolysis		
Emergency angiography and PCI if indicated is recommended in patients with heart failure/shock.[124, 235]	I	A
Rescue PCI is indicated immediately when fibrinolysis has failed (<50% ST-segment resolution at 60–90 min) or at any time in the presence of haemodynamic or electrical instability, or worsening ischaemia.[121,124,236]	I	A
Angiography and PCI of the IRA, if indicated, is recommended between 2 and 24 h after successful fibrinolysis.[125–128,234]	I	A
Emergency angiography and PCI if needed is indicated in the case of recurrent ischaemia or evidence of reocclusion after initial successful fibrinolysis.[124]	I	B

Logistical issues for hospital stay

Recommendations	Class[a]	Level[b]
It is indicated that all hospitals participating in the care of STEMI patients have a CCU/ICCU equipped to provide all aspects of care for STEMI patients, including treatment of ischaemia, severe heart failure, arrhythmias, and common comorbidities.	I	C
Transfer back to a referring non-PCI hospital		
Same day transfer should be considered appropriate in selected patients after successful primary PCI, i.e. those without ongoing myocardial ischaemia, arrhythmia, or haemodynamic instability, not requiring vasoactive or mechanical support, and not needing further early revascularization.[263]	IIa	C
Monitoring		
It is indicated that all STEMI patients have ECG monitoring for a minimum of 24 h.	I	C
Length of stay in the CCU		
It is indicated that patients with successful reperfusion therapy and an uncomplicated clinical course are kept in the CCU/ICCU for a minimum of 24 h whenever possible, after which they may be moved to a step-down monitored bed for an additional 24–48 h.	I	C
Hospital discharge		
Early discharge (within 48–72 h) should be considered appropriate in selected low-risk patients[c] if early rehabilitation and adequate follow-up are arranged.[257,259–262,264,265]	IIa	A

Maintenance antithrombotic strategy after ST-elevation myocardial infarction

Recommendations	Class[a]	Level[b]
Antiplatelet therapy with low-dose aspirin (75–100 mg) is indicated.[329]	I	A
DAPT in the form of aspirin plus ticagrelor or prasugrel (or clopidogrel if ticagrelor or prasugrel are not available or are contraindicated), is recommended for 12 months after PCI, unless there are contraindications such as excessive risk of bleeding.[186,187]	I	A
A PPI in combination with DAPT is recommended in patients at high risk of gastrointestinal bleeding[c].[335–337]	I	B
In patients with an indication for oral anticoagulation, oral anticoagulants are indicated in addition to antiplatelet therapy.[5]	I	C
In patients who are at high risk of severe bleeding complications, discontinuation of $P2Y_{12}$ inhibitor therapy after 6 months should be considered.[332,339,340]	IIa	B
In STEMI patients with stent implantation and an indication for oral anticoagulation, triple therapy[d] should be considered for 1–6 months (according to a balance between the estimated risk of recurrent coronary events and bleeding).[5]	IIa	C
DAPT for 12 months in patients who did not undergo PCI should be considered unless there are contraindications such as excessive risk of bleeding.	IIa	C
In patients with LV thrombus, anticoagulation should be administered for up to 6 months guided by repeated imaging.[341–343]	IIa	C
In high ischaemic-risk patients[e] who have tolerated DAPT without a bleeding complication, treatment with DAPT in the form of ticagrelor 60 mg twice a day on top of aspirin for longer than 12 months may be considered for up to 3 years.[333]	IIb	B
In low bleeding-risk patients who receive aspirin and clopidogrel, low-dose rivaroxaban (2.5 mg twice daily) may be considered.[338]	IIb	B
The use of ticagrelor or prasugrel is not recommended as part of triple antithrombotic therapy with aspirin and oral anticoagulation.	III	C

Routine therapies in the acute, subacute, and long-term phases: beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, mineralocorticoid receptor antagonists, and lipid-lowering treatments after ST-elevation myocardial infarction

Recommendations	Class[a]	Level[b]
Beta-blockers		
Oral treatment with beta-blockers is indicated in patients with heart failure and/or LVEF ≤40% unless contraindicated.[257–261]	I	A
Intravenous beta-blockers should be considered at the time of presentation in patients undergoing primary PCI without contraindications, with no signs of acute heart failure, and with an SBP >120 mmHg.[346–348,350,403]	IIa	A
Routine oral treatment with beta-blockers should be considered during hospital stay and continued thereafter in all patients without contraindications.[344,354–356,404,406]	IIa	B
Intravenous beta-blockers must be avoided in patients with hypotension, acute heart failure or AV block, or severe bradycardia.[344]	III	B
Lipid lowering therapies		
It is recommended to start high-intensity statin therapy[c] as early as possible, unless contraindicated, and maintain it long-term.[364,366,368]	I	A
An LDL-C goal of < 1.8 mmol/L (70 mg/dL) or a reduction of at least 50% if the baseline LDL-C is between 1.8–3.5 mmol/L (70–135 mg/dL) is recommended.[367,369,376,382]	I	B
It is recommended to obtain a lipid profile in all STEMI patients as soon as possible after presentation.[369,406]	I	C
In patients with LDL-C ≥1.8 mmol/L (≥70 mg/dL) despite a maximally tolerated statin dose who remain at high risk, further therapy to reduce LDL-C should be considered.[376,382]	IIa	A
ACE inhibitors/ARBs		
ACE inhibitors are recommended, starting within the first 24 h of STEMI in patients with evidence of heart failure, LV systolic dysfunction, diabetes, or an anterior infarct.[383]	I	A
An ARB, preferably valsartan, is an alternative to ACE inhibitors in patients with heart failure and/or LV systolic dysfunction, particularly those who are intolerant of ACE inhibitors.[216,407]	I	B
ACE inhibitors should be considered in all patients in the absence of contraindications.[394,395]	IIa	A
MRAs		
MRAs are recommended in patients with an LVEF ≤40% and heart failure or diabetes, who are already receiving an ACE inhibitor and a beta-blocker, provided there is no renal failure or hyperkalaemia.[397]	I	B

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ESC GUIDELINES

2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation

Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC)

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(2) Patients with acute chest pain but no persistent ST-segment elevation.

ECG changes may include transient ST-segment elevation, persistent or transient ST-segment depression, T-wave inversion, flat T waves or pseudo-normalization of T waves or the ECG may be normal.

The clinical spectrum of non-ST-elevation ACS (NSTE-ACS) may range from patients free of symptoms at presentation to individuals with ongoing ischaemia, electrical or haemodynamic instability or cardiac arrest. The pathological correlate at the myocardial level is cardiomyocyte necrosis [NSTE-myocardial infarction (NSTEMI)] or, less frequently, myocardial ischaemia without cell loss (unstable

2.1.2 Unstable angina in the era of high-sensitivity cardiac troponin assays

Unstable angina is defined as myocardial ischaemia at rest or minimal exertion in the absence of cardiomyocyte necrosis. Among unselected patients presenting with suspected NSTE-ACS to the emergency department, the introduction of high-sensitivity cardiac troponin measurements in place of standard troponin assays resulted in an increase in the detection of MI (~4% absolute and 20% relative increase) and a reciprocal decrease in the diagnosis of unstable angina.[7–10] Compared with NSTEMI patients, individuals with unstable angina do not experience myocardial necrosis, have a substantially lower risk of death and appear to derive less benefit from intensified antiplatelet therapy as well as early invasive strategy.[2–4,6–13]

3.1 Clinical presentation

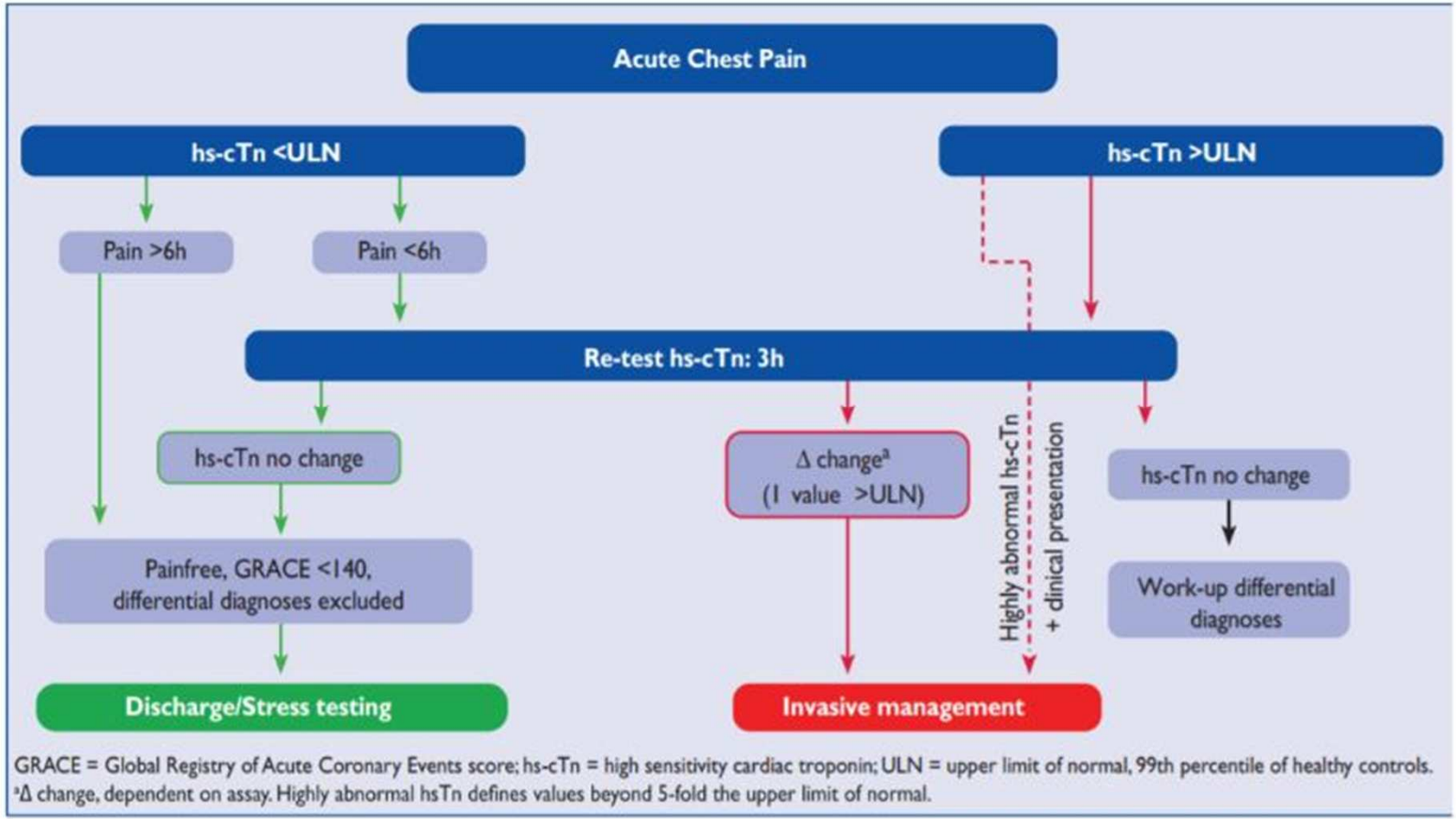
Anginal pain in NSTE-ACS patients may have the following presentations:

- Prolonged (>20 min) anginal pain at rest;
- New onset (de novo) angina (class II or III of the Canadian Cardiovascular Society classification);[21]
- Recent destabilization of previously stable angina with at least Canadian Cardiovascular Society Class III angina characteristics (crescendo angina); or
- Post-MI angina.

Diagnosi differenziale:

- 5-10% STEMI
- 15-20% NSTEMI
- 10% AI
- 15% altre cardiache
- 50% NON CARDIACHE

Cardiac	Pulmonary	Vascular	Gastro-intestinal	Orthopaedic	Other
Myopericarditis Cardiomyopathies[1]	**Pulmonary embolism**	**Aortic dissection**	**Oesophagitis, reflux or spasm**	**Musculoskeletal disorders**	**Anxiety disorders**
Tachyarrhythmias	**(Tension)-Pneumothorax**	Symptomatic aortic aneurysm	Peptic ulcer, gastritis	Chest trauma	Herpes zoster
Acute heart failure	Bronchitis, pneumonia	Stroke	Pancreatitis	Muscle injury/ inflammation	Anaemia
Hypertensive emergencies	Pleuritis		Cholecystitis	Costochondritis	
Aortic valve stenosis				Cervical spine pathologies	
Tako-Tsubo cardiomyopathy					
Coronary spasm					
Cardiac trauma					



GRACE = Global Registry of Acute Coronary Events score; hs-cTn = high sensitivity cardiac troponin; ULN = upper limit of normal, 99th percentile of healthy controls.
[a]Δ change, dependent on assay. Highly abnormal hsTn defines values beyond 5-fold the upper limit of normal.

The HEART Score for Chest Pain Patients in the ED		
History	• Highly Suspicious • Moderately Suspicious • Slightly or Non-Suspicious	• 2 points • 1 point • 0 points
ECG	• Significant ST-Depression • Nonspecific Repolarization • Normal	• 2 points • 1 point • 0 points
Age	• ≥ 65 years • > 45 - < 65 years • ≤ 45 years	• 2 points • 1 point • 0 points
Risk Factors	• ≥ 3 Risk Factors or History of CAD • 1 or 2 Risk Factors • No Risk Factors	• 2 points • 1 point • 0 points
Troponin	• ≥ 3 x Normal Limit • > 1 - < 3 x Normal Limit • ≤ Normal Limit	• 2 points • 1 point • 0 points
Risk Factors: DM, current or recent (<one month) smoker, HTN, HLP, family history of CAD, & obesity		
Score 0 – 3: 2.5% MACE over next 6 weeks → Discharge Home **Score 4 – 6**: 20.3% MACE over next 6 weeks → Admit for Clinical Observation **Score 7 – 10**: 72.7% MACE over next 6 weeks → Early Invasive Strategies		

Recommendations for anti-ischaemic drugs in the acute phase of non-ST-elevation acute coronary syndromes

Recommendations	Class[a]	Level[b]	Ref.[c]
Early initiation of beta-blocker treatment is recommended in patients with ongoing ischaemic symptoms and without contraindications.	I	B	119
It is recommended to continue chronic beta-blocker therapy, unless the patient is in Killip class III or higher.	I	B	126
Sublingual or i.v. nitrates are recommended to relieve angina;[d] i.v. treatment is recommended in patients with recurrent angina, uncontrolled hypertension or signs of heart failure.	I	C	
In patients with suspected/confirmed vasospastic angina, calcium channel blockers and nitrates should be considered and beta-blockers avoided.	IIa	B	127

Recommendations	Class[a]	Level[b]	Ref.[c]
Oral antiplatelet therapy			
Aspirin is recommended for all patients without contraindications at an initial oral loading dose[d] of 150–300 mg (in aspirin-naive patients) and a maintenance dose of 75–100 mg/day long-term regardless of treatment strategy.	I	A	129–132
A $P2Y_{12}$ inhibitor is recommended, in addition to aspirin, for 12 months unless there are contraindications such as excessive risk of bleeds.	I	A	137, 148, 153
• Ticagrelor (180 mg loading dose, 90 mg twice daily) is recommended, in the absence of contraindications,[e] for all patients at moderate-to-high risk of ischaemic events (e.g. elevated cardiac troponins), regardless of initial treatment strategy and including those pretreated with clopidogrel (which should be discontinued when ticagrelor is started).	I	B	153
• Prasugrel (60 mg loading dose, 10 mg daily dose) is recommended in patients who are proceeding to PCI if no contraindication.[e]	I	B	148, 164
• Clopidogrel (300–600 mg loading dose, 75 mg daily dose) is recommended for patients who cannot receive ticagrelor or prasugrel or who require oral anticoagulation.	I	B	137
$P2Y_{12}$ inhibitor administration for a shorter duration of 3–6 months after DES implantation may be considered in patients deemed at high bleeding risk.	IIb	A	187–189, 192

Recommendations for anticoagulation in non-ST-elevation acute coronary syndromes

Recommendations	Class[a]	Level[b]	Ref.[c]
Parenteral anticoagulation is recommended at the time of diagnosis according to both ischaemic and bleeding risks.	I	B	227
Fondaparinux (2.5 mg s.c. daily) is recommended as having the most favourable efficacy–safety profile regardless of the management strategy.	I	B	218, 228, 229
Bivalirudin (0.75 mg/kg i.v. bolus, followed by 1.75 mg/kg/h for up to 4 h after the procedure) is recommended as an alternative to UFH plus GPIIb/IIIa inhibitors during PCI.	I	A	205, 222, 223
UFH 70–100 IU/kg i.v. (50–70 IU/kg if concomitant with GPIIb/IIIa inhibitors) is recommended in patients undergoing PCI who did not receive any anticoagulant.	I	B	219, 229
In patients on fondaparinux (2.5 mg s.c. daily) undergoing PCI, a single i.v. bolus of UFH (70–85 IU/kg, or 50–60 IU/kg in the case of concomitant use of GPIIb/IIIa inhibitors) is recommended during the procedure.	I	B	219
Enoxaparin (1 mg/kg s.c. twice daily) or UFH are recommended when fondaparinux is not available.	I	B	218, 230

Table 11 Dosing of anticoagulants in patients with normal and impaired renal function

Drug	Recommendations		
	Normal renal function or stage 1–3 CKD (eGFR ≥30 mL/min/1.73m²)	Stage 4 CKD (eGFR 15–29 mL/min/1.73m²)	Stage 5 CKD (eGFR <15 mL/min/1.73m²)
Unfractionated heparin	• Prior to coronary angiography: 60–70 IU/kg i.v. (max 5000 IU) and infusion (12–15 IU/kg/h) (max 1000 IU/h), target aPTT 1.5–2.5x control • During PCI according to ACT or 70–100 IU/kg i.v. in patients not anticoagulated (50–70 IU/kg if concomitant with GPIIb/IIIa inhibitors)	No dose adjustment	No dose adjustment
Enoxaparin	1 mg/kg s.c. twice a day	1 mg/kg s.c. once a day	Not recommended
Fondaparinux	2.5 mg s.c. once a day	Not recommended if eGFR <20 mL/min/1.73m²	Not recommended
Bivalirudin	Bolus 0.75 mg/kg i.v., infusion 1.75 mg/kg/h*	Not recommended	Not recommended

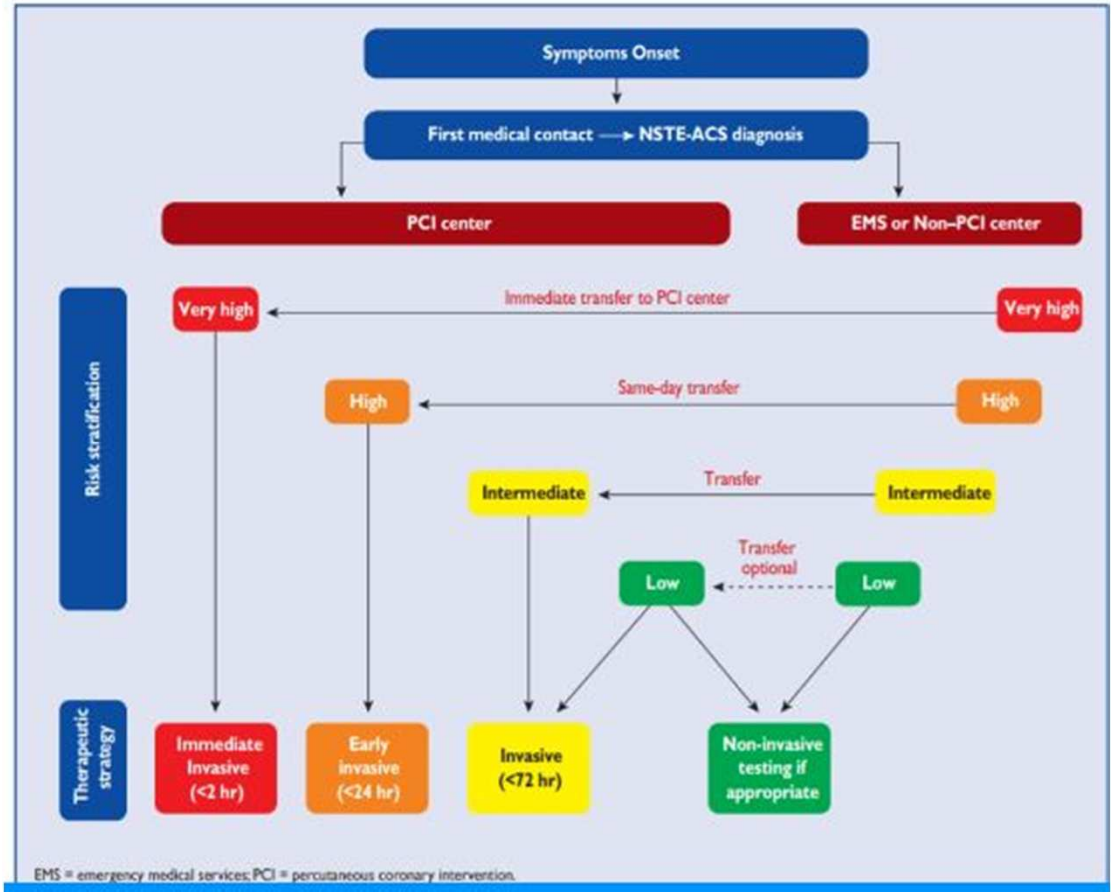
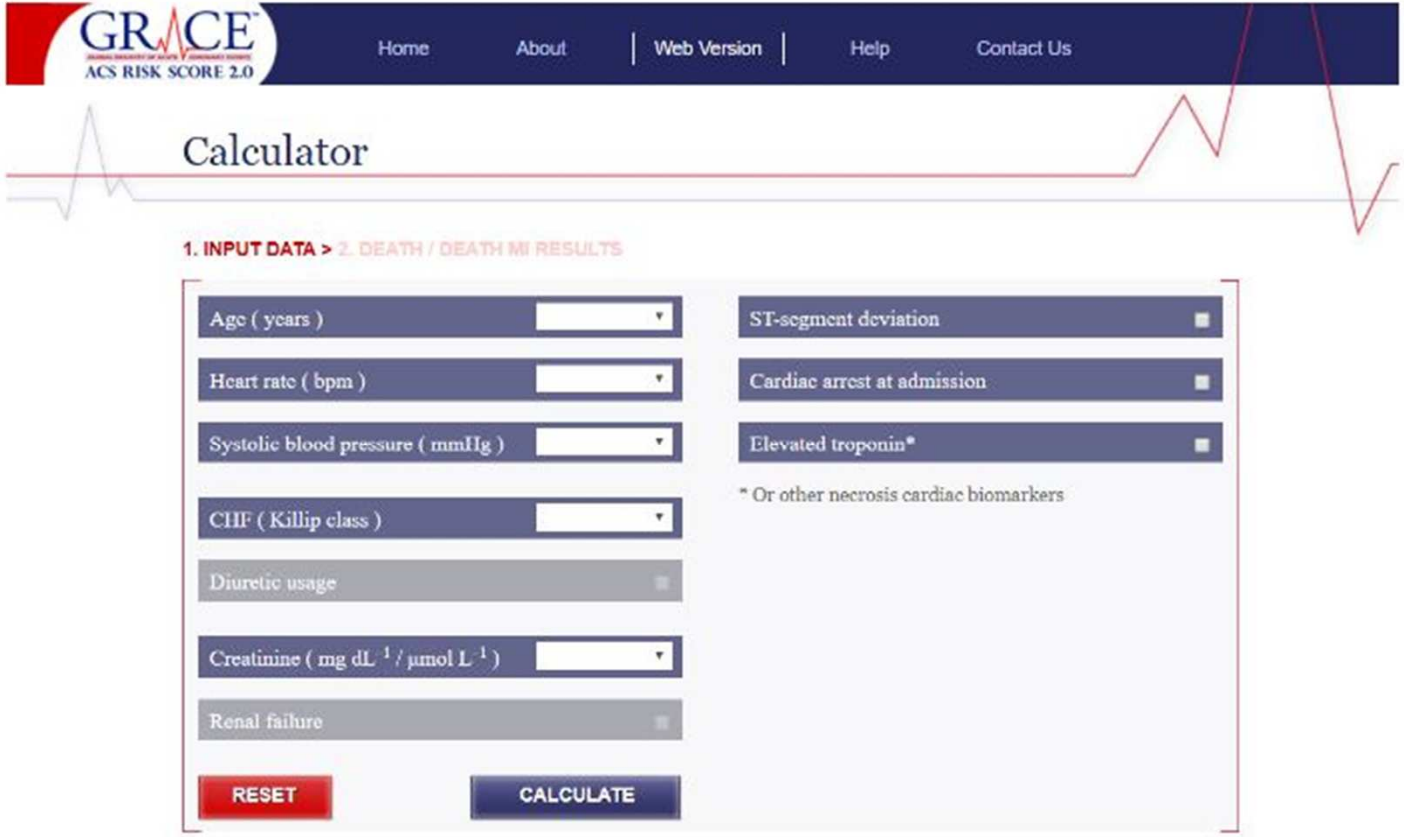

Symptoms Onset
First medical contact → NSTE-ACS diagnosis
PCI center
EMS or Non–PCI center
Risk stratification
Very high
Immediate transfer to PCI center
Very high
High
Same-day transfer
High
Intermediate
Transfer
Intermediate
Transfer optional
Low
Low
Therapeutic strategy
Immediate Invasive (<2 hr)
Early invasive (<24 hr)
Invasive (<72 hr)
Non-invasive testing if appropriate
EMS = emergency medical services; PCI = percutaneous coronary intervention.

Table 13 **Risk criteria mandating invasive strategy in NSTE-ACS**

Very-high-risk criteria
• Haemodynamic instability or cardiogenic shock
• Recurrent or ongoing chest pain refractory to medical treatment
• Life-threatening arrhythmias or cardiac arrest
• Mechanical complications of MI
• Acute heart failure
• Recurrent dynamic ST-T wave changes, particularly with intermittent ST-elevation
High-risk criteria
• Rise or fall in cardiac troponin compatible with MI
• Dynamic ST- or T-wave changes (symptomatic or silent)
• GRACE score >140
Intermediate-risk criteria
• Diabetes mellitus
• Renal insufficiency (eGFR <60 mL/min/1.73 m^2)
• LVEF <40% or congestive heart failure
• Early post-infarction angina
• Prior PCI
• Prior CABG
• GRACE risk score >109 and <140
Low-risk criteria
• Any characteristics not mentioned above


GRACE
ACS RISK SCORE 2.0
Home
About
Web Version
Help
Contact Us
Calculator
1. INPUT DATA > 2. DEATH / DEATH MI RESULTS
Age (years)
Heart rate (bpm)
Systolic blood pressure (mmHg)
CHF (Killip class)
Diuretic usage
Creatinine (mg dL -1 / µmol L -1)
Renal failure
ST-segment deviation
Cardiac arrest at admission
Elevated troponin*
* Or other necrosis cardiac biomarkers
RESET
CALCULATE

Recommendations for the management of elderly patients with non-ST-elevation acute coronary syndromes

Recommendations	Class[a]	Level[b]	Ref.[c]
It is recommended to tailor antithrombotic treatment according to bodyweight and renal function.	I	C	
Elderly patients should be considered for an invasive strategy and, if appropriate, revascularization after careful evaluation of potential risks and benefits, estimated life expectancy, comorbidities, quality of life, frailty and patient values and preferences.	IIa	A	408, 414–418
Adjusted dosing regimens of beta-blockers, ACE inhibitors, ARBs and statins should be considered to prevent side effects.	IIa	C	

Recommendations for long-term management after non-ST-elevation acute coronary syndromes

Recommendations (for the recommendations on antithrombotic treatment, see sections 5.2.9 and 5.3.3)	**Class**[a]	**Level**[b]	**Ref.**[c]
It is recommended to advise all patients on lifestyle changes (including smoking cessation, regular physical activity and a healthy diet).	I	A	536, 537
It is recommended to start high-intensity statin therapy as early as possible, unless contraindicated, and maintain it long term.	I	A	522, 527, 528
An ACE inhibitor is recommended in patients with LVEF ≤40% or heart failure, hypertension or diabetes, unless contraindicated. An ARB provides an alternative, particularly if ACE inhibitors are not tolerated.	I	A	478–481, 530, 531, 538
Beta-blocker therapy is recommended in patients with LVEF ≤40%, unless contraindicated.	I	A	482–486
Mineralocorticoid receptor antagonists, preferably eplerenone, are recommended in patients with LVEF ≤35% and either heart failure or diabetes after NSTE-ACS but no significant renal dysfunction or hyperkalaemia.[d]	I	A	487, 488, 525
A diastolic blood pressure goal of <90 mmHg is recommended (<85 mmHg in diabetic patients).	I	A	539, 540