



Acute Coronary Syndrome

Sindroma Koroner Akut

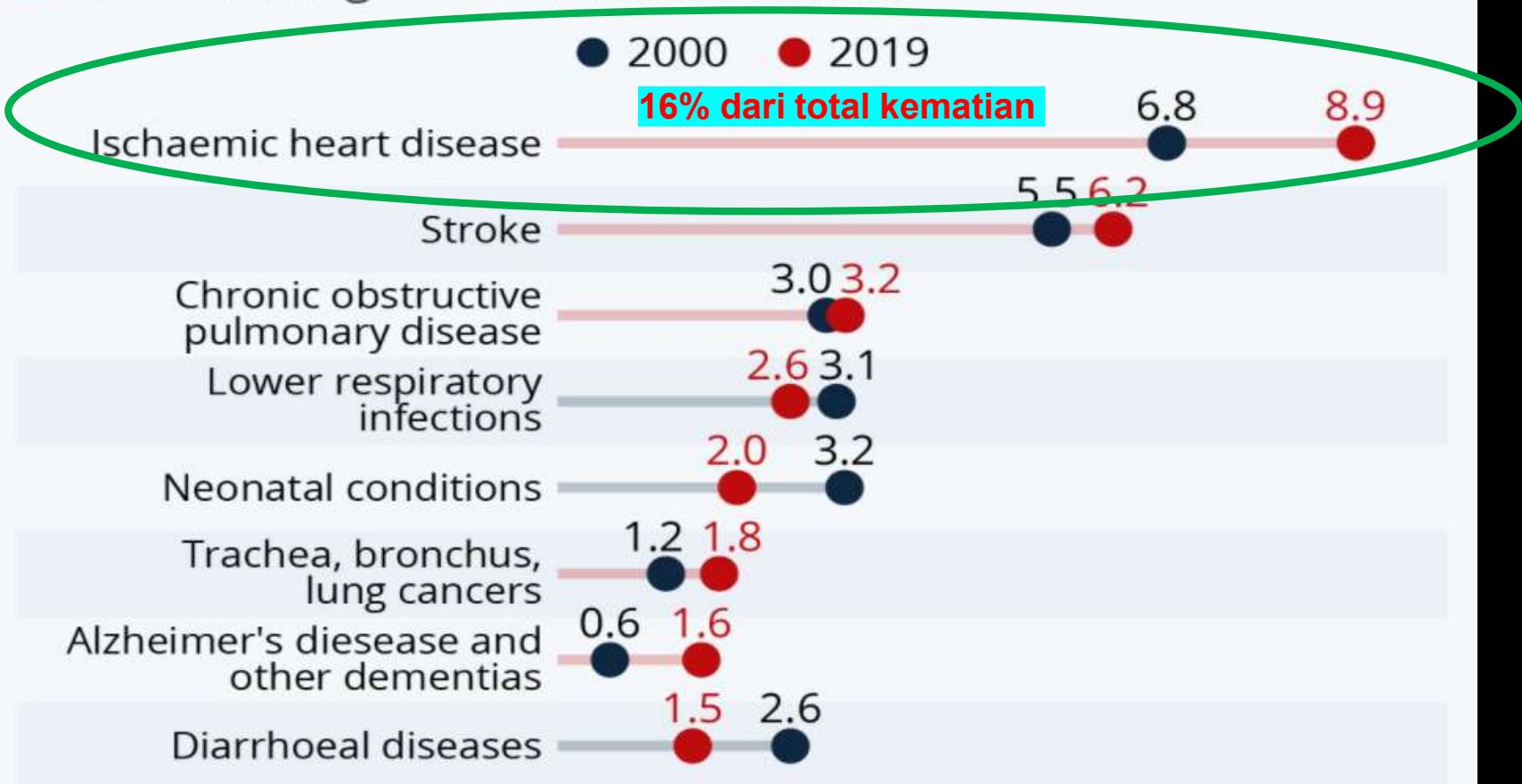
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Fakultas Kedokteran

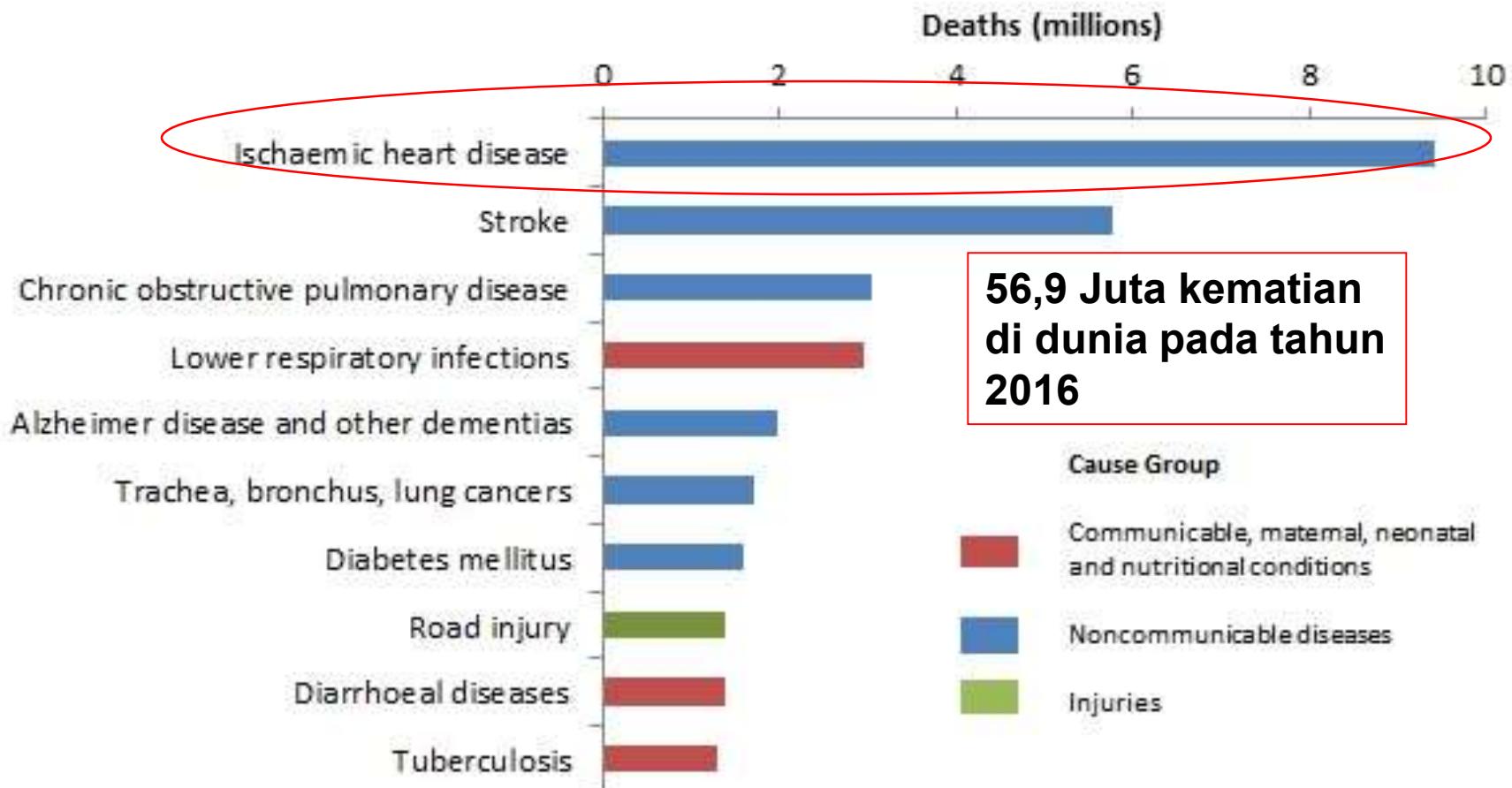
Universitas Muhammadiyah Malang

The World's Leading Causes Of Death

Total number of people who died from the following conditions (in millions)



Top 10 global causes of deaths, 2016



Source: Global Health Estimates 2016: Deaths by Cause, Age, Sex, by Country and by Region, 2000-2016. Geneva, World Health Organization; 2018.

Acute Coronary Syndrome (ACS) A Major Cause of Mortality and Morbidity

UA/NSTEMI

- In-hospital death and re-infarction: 5-10%¹
- Six-month mortality in the GRACE registry² (from admission to 6 months):
 - NSTEMI: 13%
 - UA: 8%

STEMI

- 1/3 of STEMI patients will die within 24 h of the onset of ischemia¹
- In-hospital death and reinfarction: 8-10%³
- One-month mortality: 6-7%⁴

1.Grech & Ramsdale. Acute coronary syndrome : unstable angina and non-ST segment elevation myocardial infarction. BMJ 2003;326:1259-61;

2. Fox. et al. An international on acute coronary syndrome care: Insight from the global registry of acute coronary event

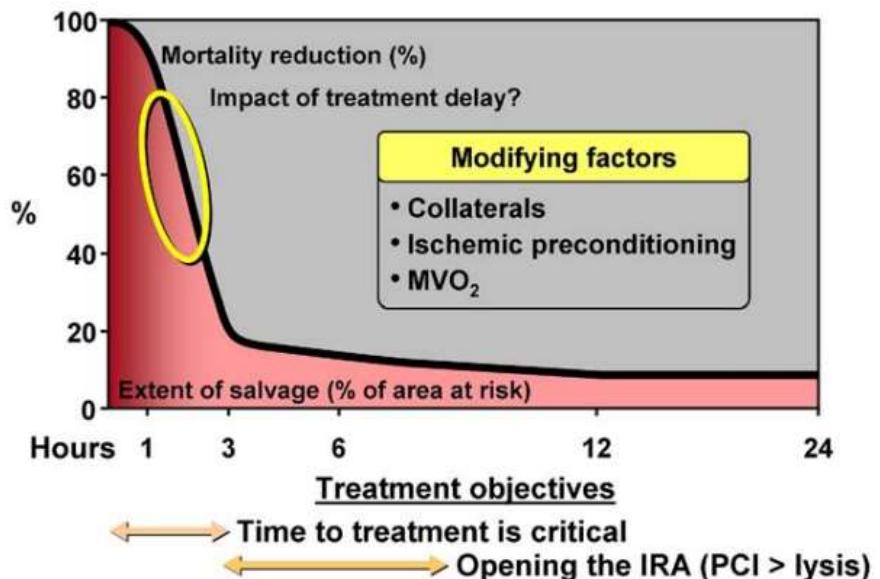
Am Heart. Et al J 2004;148:S40-5;

3.Antman et al. ACC/AHA guideline for the management of patients with ST-Elevation Myocardial infarction. Circulation 2004;110:e82-292;

4.van de Werf et al. Management of acute myocardial infarction in patients presenting with ST-segment Elevation. Eur Heart J 2003;24:28-66

Myocardial Cell Necrosis in MI

- An occlusion **more than 20 minutes** irreversible myocardial cell damage and cell death
- The longer the infarct time the greater the ischemia and necrosis of the myocardium
- Early reperfusion, via fibrinolytic therapy or primary percutaneous coronary intervention (PCI), is crucial for patients with STEMI



TIME SAVED = MUSCLE SAVED

DEFINISI

Suatu sindroma klinik yang menandakan adanya iskemia miokard akut akibat dari ketidakseimbangan antara kebutuhan dan suplai oksigen di miokard , terdiri dari :

- **Infark miokard akut dng elevasi segmen ST (STEMI)**
- **Infark miokard akut tanpa elevasi segmen ST (NSTEMI)**
- **Angina pektoris tidak stabil (UAP)**

PATOGENESIS

- Umumnya disebabkan oleh aterosklerosis koroner
- Plak aterosklerosis ruptur → terbentuk trombus diatas ateroma yang secara akut menyumbat lumen koroner
- Apabila sumbatan terjadi secara total → hampir seluruh dinding ventrikel akan nekrosis

Faktor risiko penyakit jantung koroner

Non modifiable

- **Sex**
- **Hereditary**
- **Race**
- **Age**

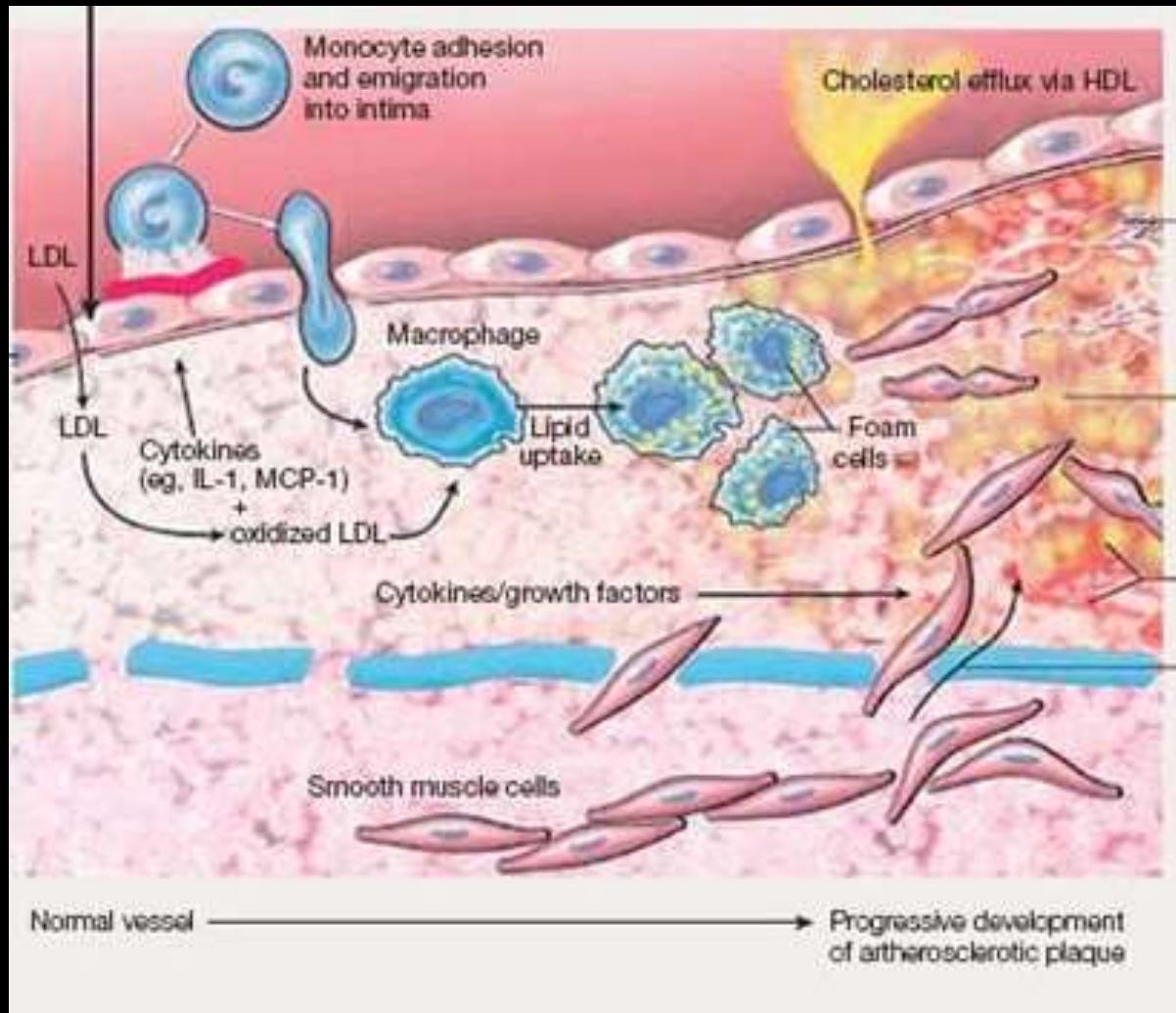
Modifiable

- **High blood pressure**
- **High blood cholesterol**
- **Smoking**
- **Physical activity**
- **Obesity**
- **Diabetes**
- **Stress and anger**

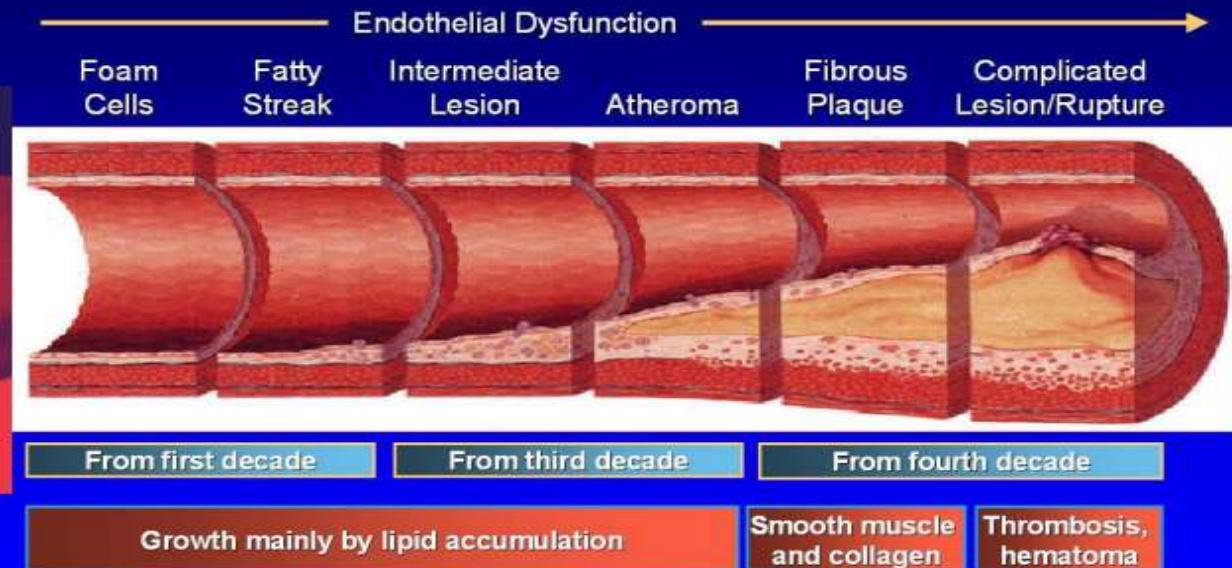
Pathophys (enough to get by..)

Atherosclerosis

- Epithelial injury
- Migration of monocytes/macrophages
- LDL lipids consumed → foam cells
- Growth factors → smooth muscle, collagen, proteoglycans
- Atheromatous plaque forms



Atherosclerosis Timeline



MY HEART,
YOUR HEART

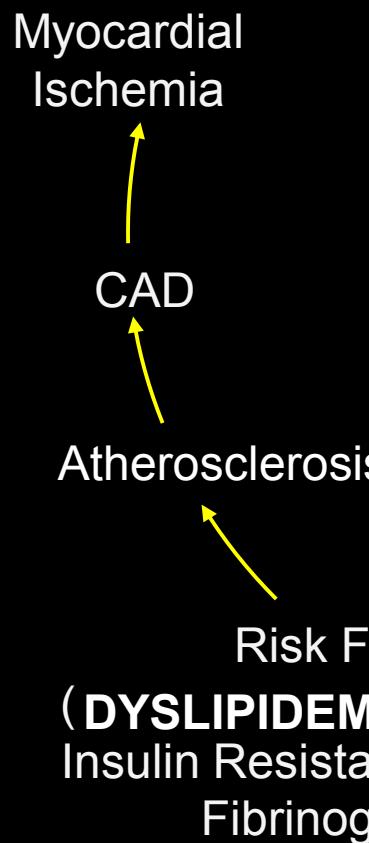
Stary et al. *Circulation*. 1995;92:1355-1374.

Aterosklerosis:

Pengerasan dinding arteri yang diakibatkan oleh adanya ateroma (plak kekuningan yang mengandung lemak, kolesterol, sel-sel, kalsium, dll) pada dinding pembuluh darah arteri arteri.

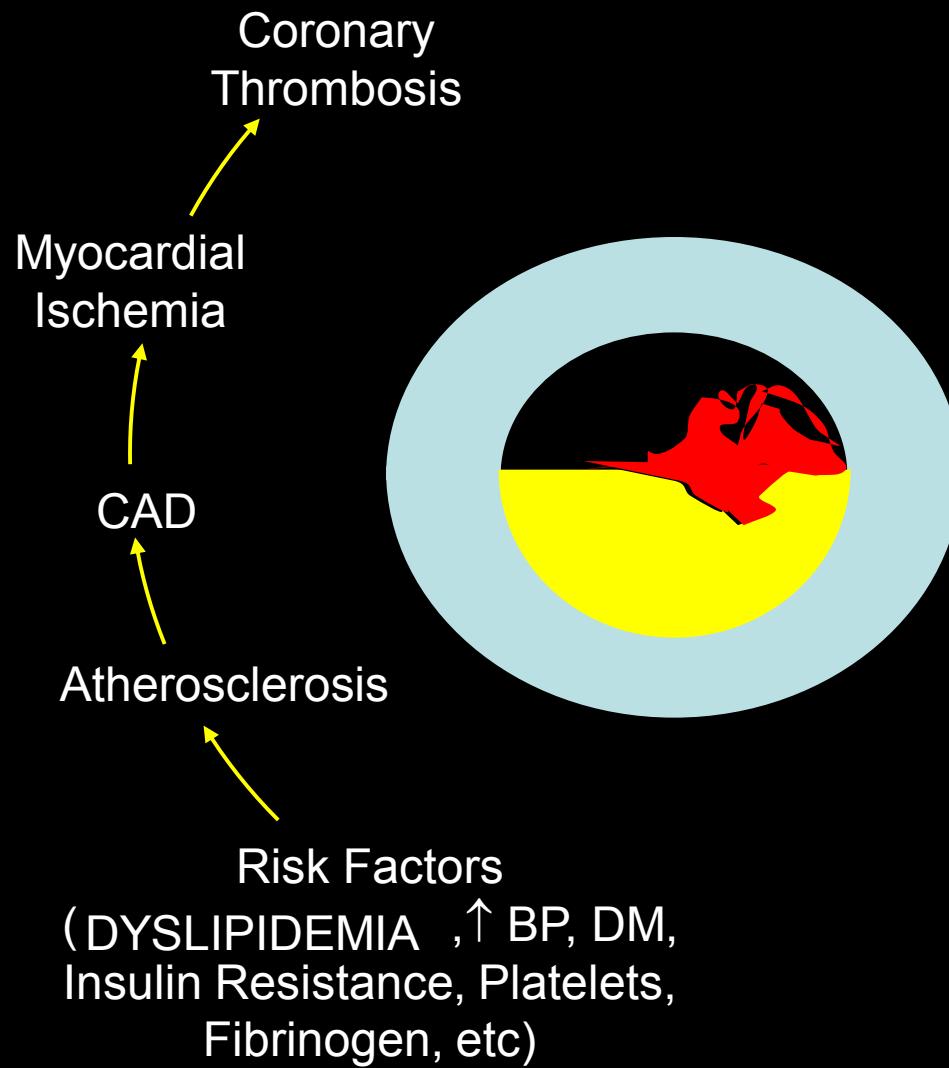
The cardiovascular continuum of events

**Ischemia = imbalance of
oxygen supply and demand**



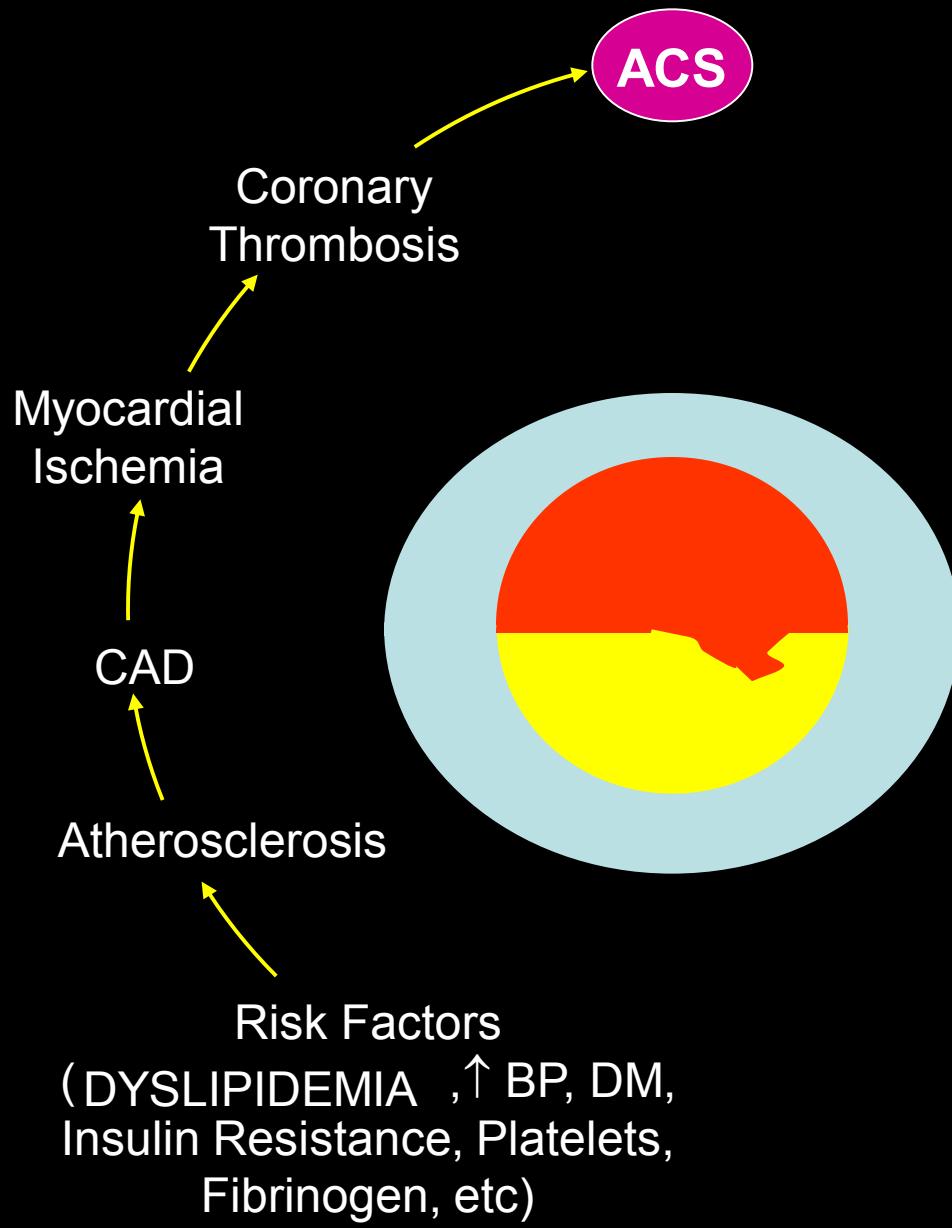
Adapted from
Dzau et al. Am Heart J. 1991;121:1244-1263

The cardiovascular continuum of events



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The cardiovascular continuum of events

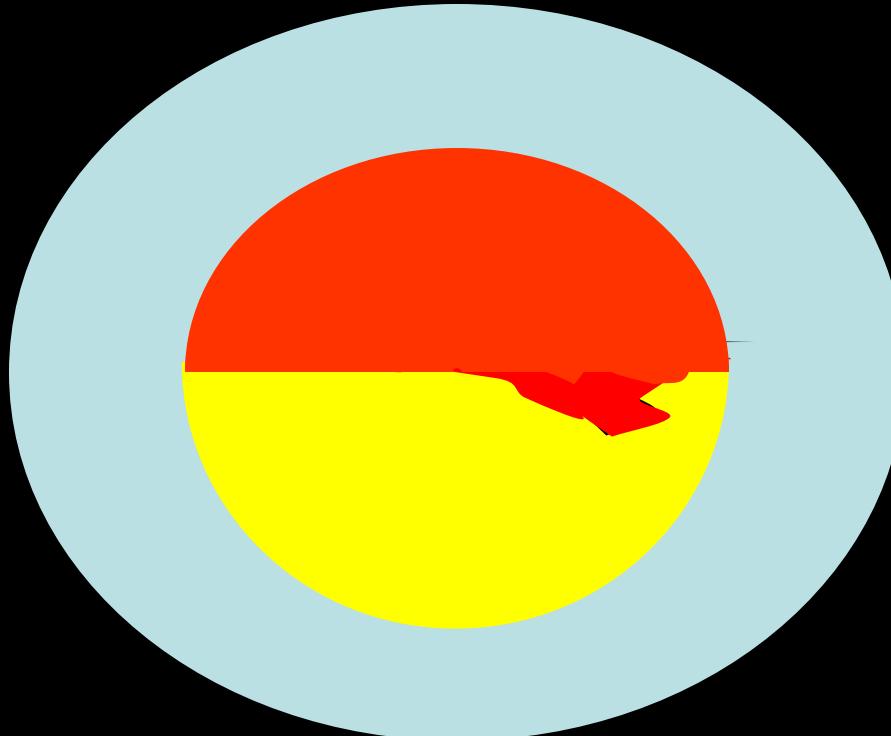


Adapted from
Dzau et al. Am Heart J. 1991;121:1244-1263

Stable angina
Coronary thrombosis

Plaque rupture
UA/NSTEMI

STEMI



Diagnosis

Anamnesis

Pemeriksaan Fisik

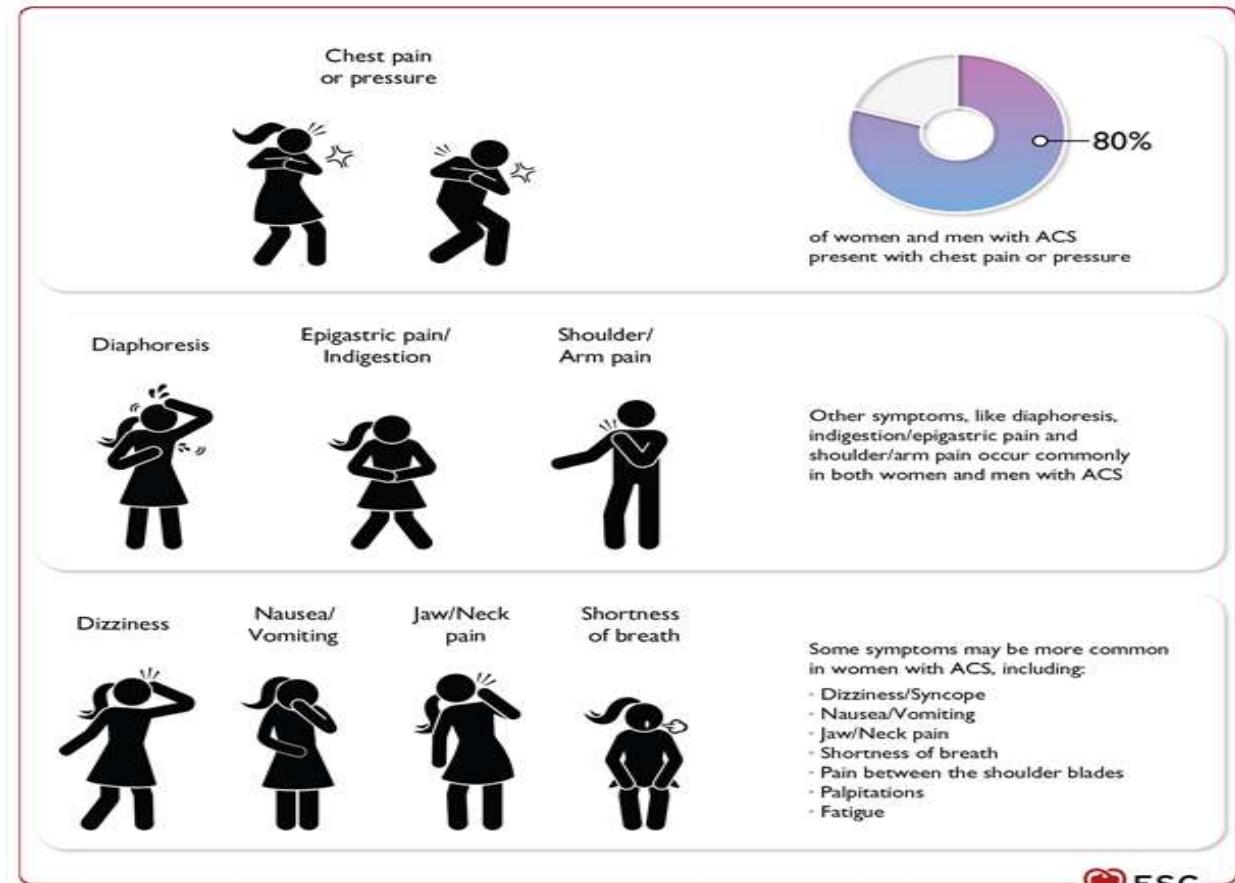
Pemeriksaan Penunjang :

- 1. Laboratorium**
- 2. Elektrokardiografi**
- 3. Thoraks Foto**

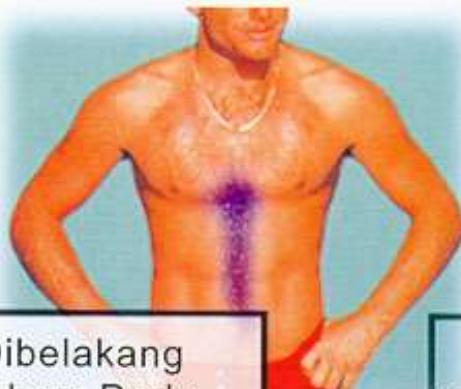
Karakteristik Gejala pada ACS

Pada ACS dapat muncul dengan berbagai macam tanda dan gejala klinis.

Penting untuk **memiliki tingkat kesadaran yang tinggi** baik di masyarakat umum maupun tenaga kesehatan



TEMPAT TEMPAT NYERI PADA GANGGUAN JANTUNG



Dibelakang tulang Dada



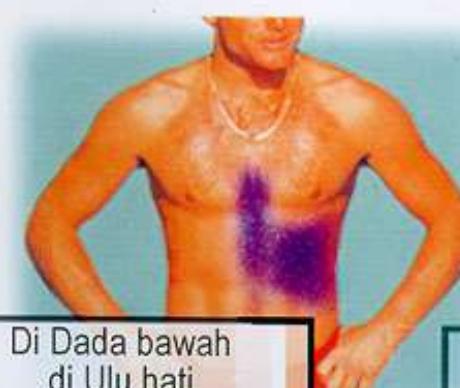
Dibelakang tulang Dadamenjalar ke leher



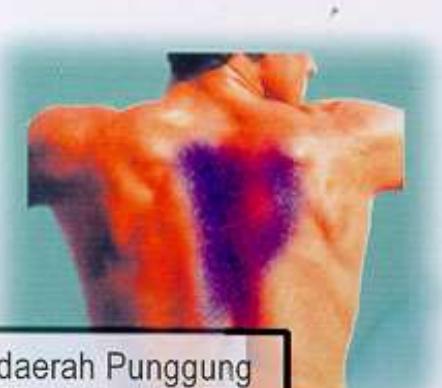
Dari Dada menjalar ke Bahu dan Dada



Dari Dada menjalar ke Rahang



Di Dada bawah di Ulu hati
(sering di tafsirkan sakit Maag)



Di daerah Punggung diantara kedua Belikat

Nyeri Dada Khas Infark

1. Nyeri dada tipikal yang persisten >20 Menit (80%)
2. Nyeri dada angina Pertama Kali (de Novo) dengan tingkatan CCS (*The Canadian Cardiovascular Society*) III
3. Cresendo Angina (makin sering, lebih lama, atau menjadi makin berat, minimal CCS III)
4. Angina Paska Infark (terjadi 2 minggu setelah infark)

Angina Ekuivalen

Pasien mengalami SKA dengan keluhan angina atipikal terutama berhubungan dengan aktivitas, antara lain:

- Gangguan pencernaan (indigesti)
- sesak napas yang tidak dapat diterangkan
- rasa lemah mendadak yang sulit diuraikan.

Pada usia muda (25-40 tahun) atau usia lanjut (>75 tahun), wanita, penderita diabetes, gagal ginjal menahun, atau demensia.

Differential Diagnosis

Cardiac

- MI
- Angina
- Pericarditis
- Aortic dissection

Respiratory

- Pulmonary embolism
- Pneumothorax
- Pneumonia

Chest pain

GI

- Oesophageal spasm
- GORD
- Pancreatitis

Musculoskeletal

- Costochondriasis
- Trauma

PHYSICAL EXAMINATION



GENERAL APPEARANCE

Anxious, considerable distress, restless, fist on chest
(Levine sign)

LV failure & symp. stimulation : cold perspiration, pallor, dyspnea, cough with frothy pink or blood-streaked sputum.

Shock : cool, clammy skin, facial pallor, cyanosis, confusion or disorientation

HEART RATE

Variable depending on underlying rhythm and degree of ventr. failure

Most commonly, HR 100 – 110/min; > 95% patients : VPB's within first 4 hours

BLOOD PRESSURE

Majority normotensive, but syst. BP may decline and diast. BP may rise

± Half of pts with inferior MI → parasympathetic stimulation : hypotension, bradycardia or both (Bezold – Jarisch reflex)

± half of pts with anterior MI, → sympathetic excess : hypertension, tachycardia or both

TEMPERATURE AND RESPIRATION

Most pts with extensive MI → fever within 24-48 hrs, fever resolves by 4th or 5th day

Respiration ↑ due to anxiety and pain, in LV failure : resp. rate correlates with degree of heart failure

JUGULAR VENOUS PULSE

JVP usually normal

RV infarction : marked jug. venous distension

CAROTID PULSE

Small pulse → reduced stroke volume

Pulse alternans : severe LV dysfunction

CHEST

LV failure and/or LV compliance ↓ : moist rales

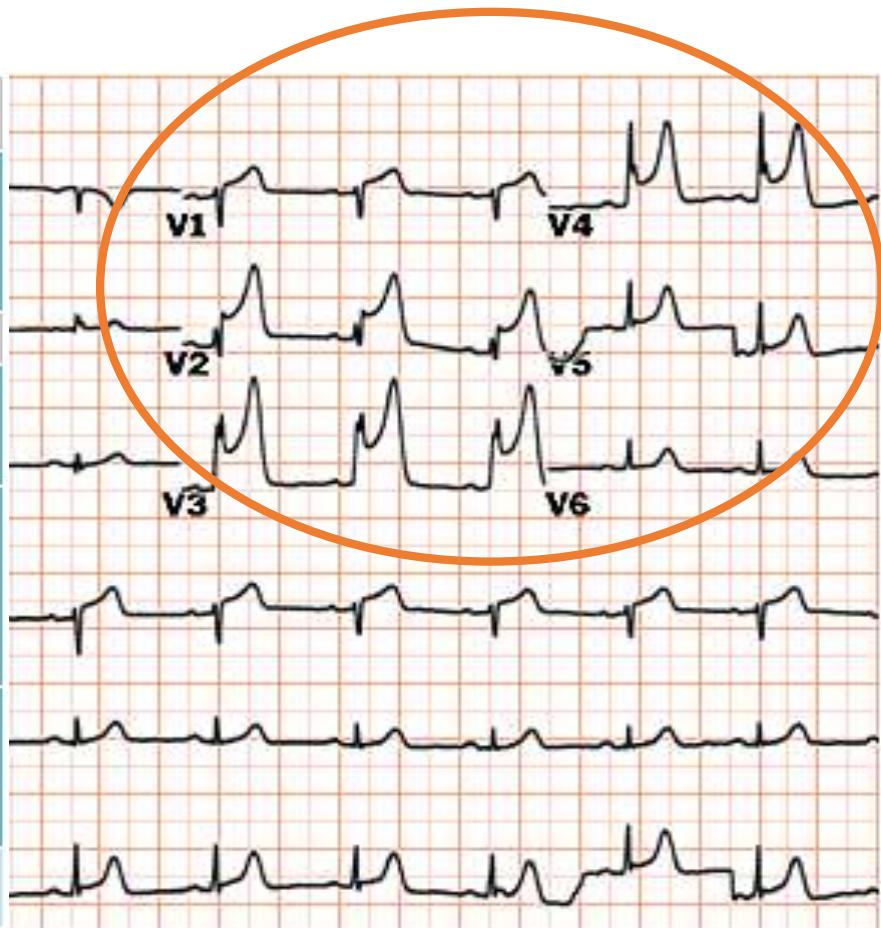
Severe failure : diffuse wheezing, cough + hemoptysis

1967 : Killip & Kimball : prognostic classification

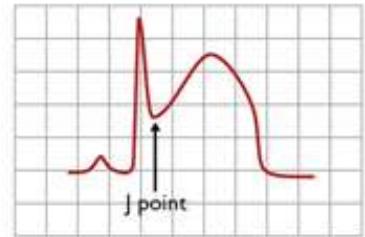
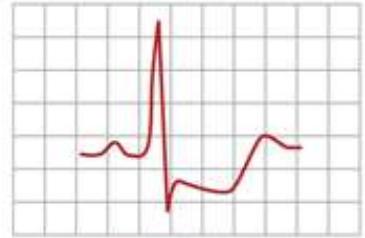
<i>Class</i>	<i>Features</i>	<i>Mortality</i>
<i>Class-I</i>	No signs of pulmonary <i>or</i> venous congestion	0-5%
<i>Class-II</i>	<ul style="list-style-type: none">• Rales at the lung bases• S₃ gallop• Tachypnea, <i>or</i>• Signs of failure of the right side of the heart	10-20%
<i>Class-III</i>	Pulmonary edema	35-45%
<i>Class-IV</i>	<p>Shock with SBP<90 mmHg & evidence of :</p> <ul style="list-style-type: none">• Peripheral vasoconstriction,• Peripheral cyanosis• Mental confusion, and• Oliguria	85-95%

Elektrokardiografi

Recommendations	Class ^a	Level ^b
It is recommended to base the diagnosis and initial short-term risk stratification of ACS on a combination of clinical history, symptoms, vital signs, other physical findings, ECG, and hs-cTn. ^{1,17,18}	I	B
ECG		
Twelve-lead ECG recording and interpretation is recommended as soon as possible at the point of FMC, with a target of <10 min. ^{5,19}	I	B
Continuous ECG monitoring and the availability of defibrillator capacity is recommended as soon as possible in all patients with suspected STEMI, in suspected ACS with other ECG changes or ongoing chest pain, and once the diagnosis of MI is made. ^{20,21}	I	B
The use of additional ECG leads (V3R, V4R, and V7–V9) is recommended in cases of inferior STEMI or if total vessel occlusion is suspected and standard leads are inconclusive. ^{22–24}	I	B
An additional 12-lead ECG is recommended in cases with recurrent symptoms or diagnostic uncertainty.	I	C



ECG STEMI

ECG pattern	Criteria	Signifying	Figure
i STEMI	New ST-elevation at the J-point in ≥ 2 contiguous leads ^a ≥2.5 mm in men <40 years, ≥2 mm in men ≥40 years, or ≥1.5 mm in women regardless of age in leads V2–V3 and/or ≥1 mm in the other leads (in the absence of LV hypertrophy or left bundle branch block) ^a Including V3R and V4R	Ongoing acute coronary artery occlusion	 An ECG strip on a grid background. It shows a normal P wave followed by a sharp QRS complex. An arrow points to the J-point, which is elevated above the baseline. The ST segment is also elevated. The T wave is upright and prominent.
ii Posterior STEMIs	ST-segment depression in leads V1–V3, especially when the terminal T-wave is positive (ST-segment elevation equivalent), and concomitant ST-segment elevation ≥0.5 mm recorded in leads V7–V9	Posterior STEMIs	 An ECG strip on a grid background. It shows a normal P wave followed by a QRS complex. The ST segment in leads V1-V3 is depressed below the baseline. In leads V7-V9, there is a prominent ST-segment elevation. The T waves are upright.
iii LCx occlusion/ right ventricular MI	ST-segment elevation in V7–V9 and V3R and V4R, respectively	Left circumflex (LCX) artery occlusion or right ventricular MI	 An ECG strip on a grid background. It shows a normal P wave followed by a QRS complex. The ST segment is elevated in leads V7-V9. In leads V3R and V4R, there is a prominent ST-segment elevation. The T waves are upright.

ECG STEMI

iv

Multivessel
ischaemia/
left main
obstruction

ST depression ≥ 1 mm in six or more surface leads (inferolateral ST depression), coupled with ST-segment elevation in aVR and/or VI



v

Left bundle
branch block/
paced rhythm

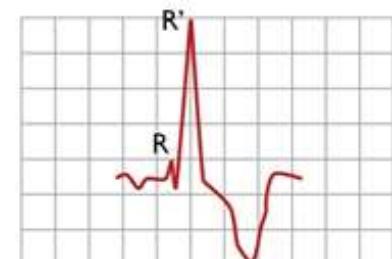
QRS duration greater than 120 ms
Absence of Q wave in leads I, V5 and V6
Monomorphic R wave in I, V5 and V6
ST and T wave displacement opposite to the major deflection of the QRS complex



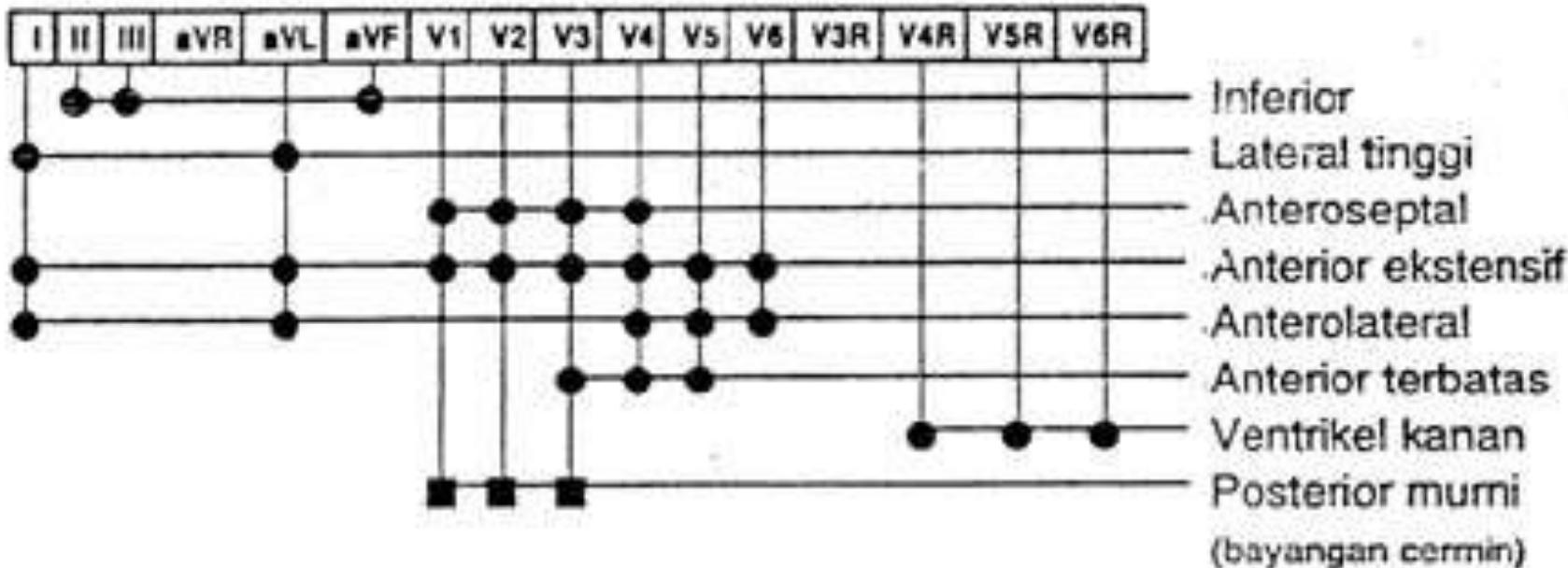
vi

Right bundle
branch block

QRS duration greater than 120 ms
rsR' "bunny ear" pattern in the anterior precordial leads (leads VI-V3)
Slurred S waves in leads I, aVL and frequently V5 and V6



Sandapan dengan ST Elevasi

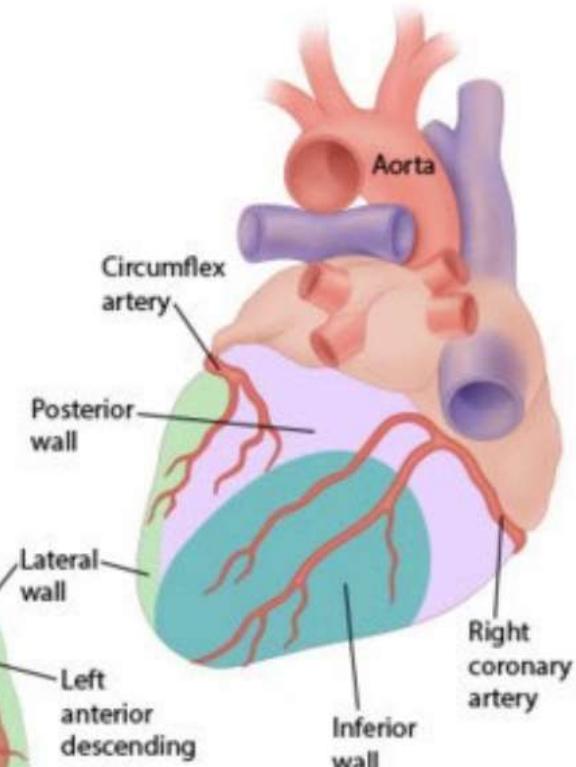
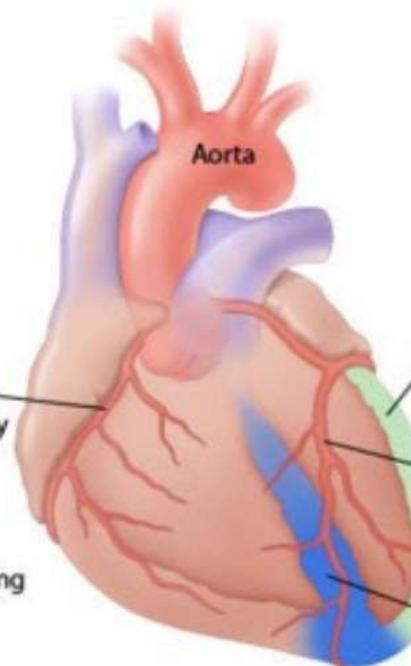
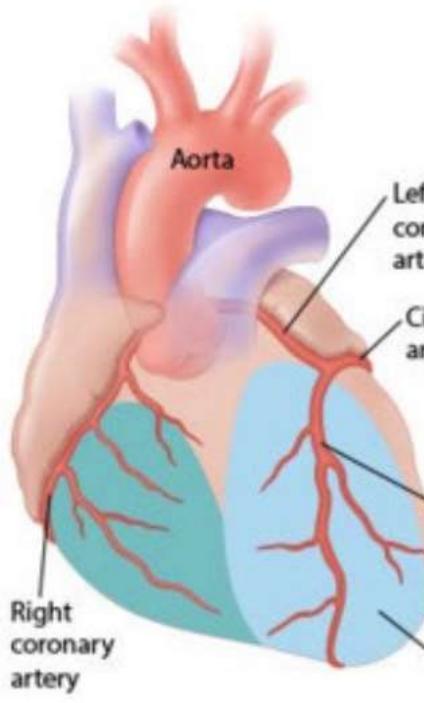


Gambar 46. Lokalisasi Dinding Ventrikel pada EKG.

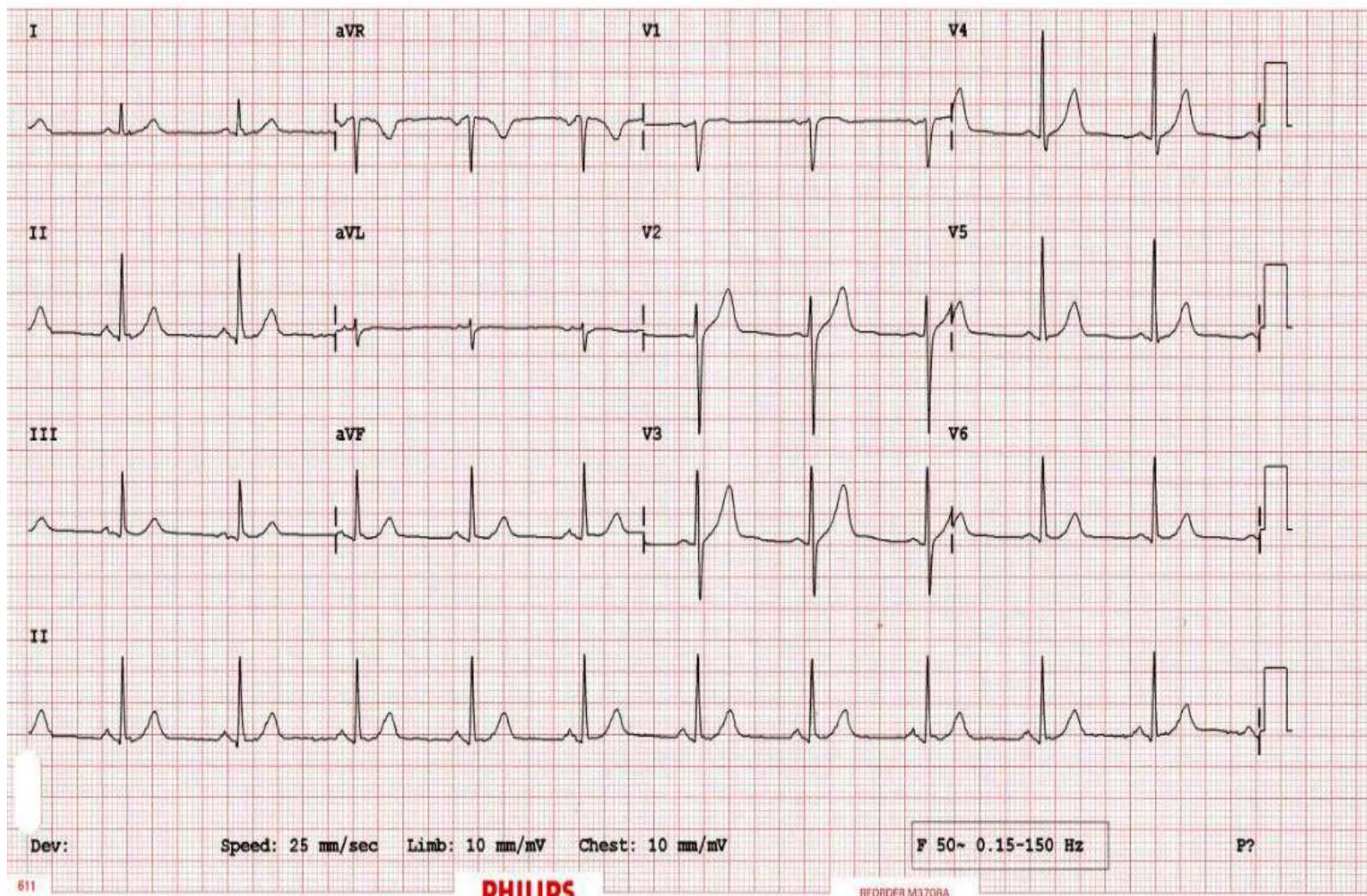
Sandapan dengan ST Elevasi

1	aVR	V ₁	V ₄
II	aVL	V ₂	V ₅
III	aVF	V ₃	V ₆

Inferior: II, III, aVF
Septal: V₁, V₂
Anterior: V₃, V₄
Lateral: I, aVL, V₅, V₆

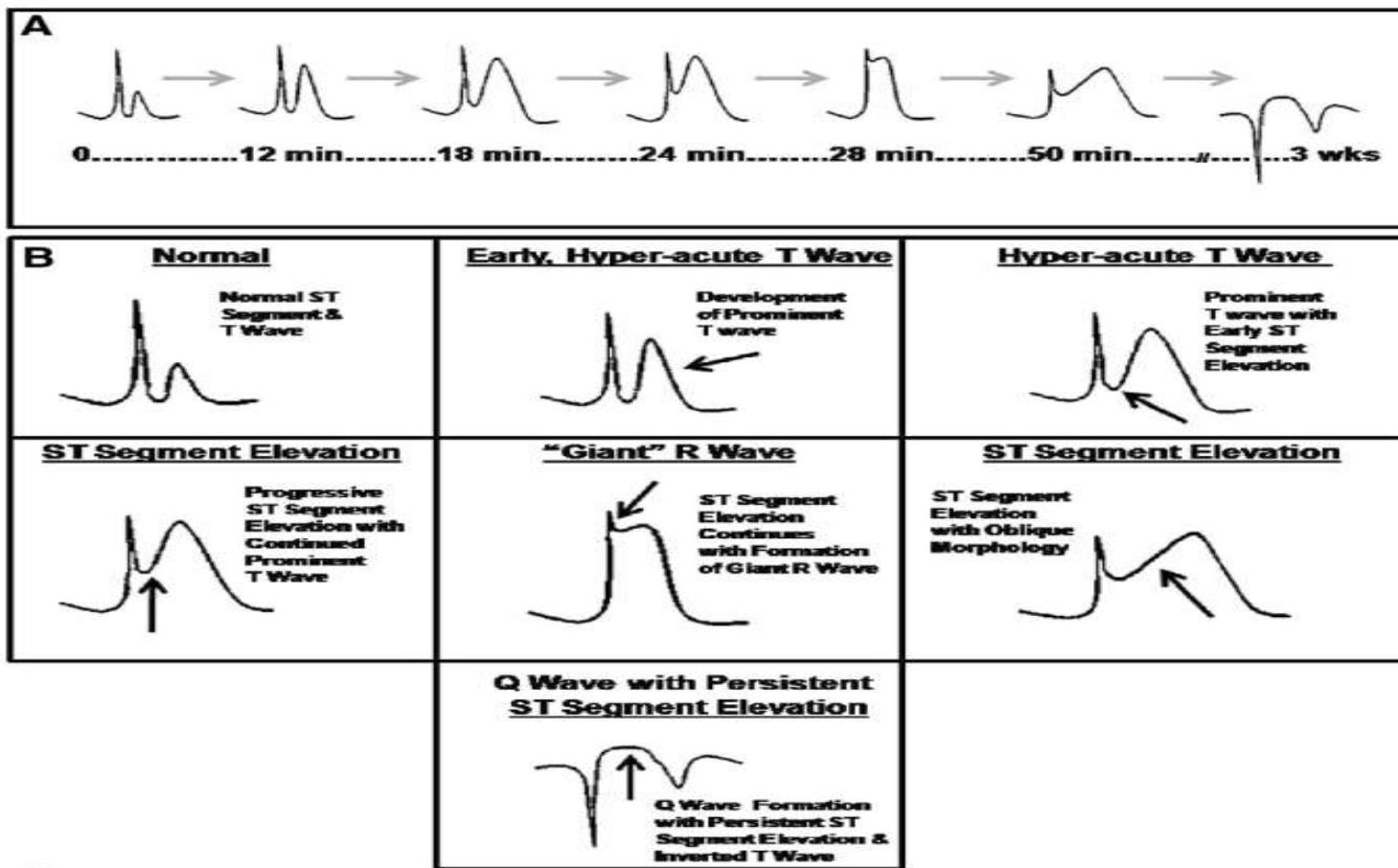


Normal ECG

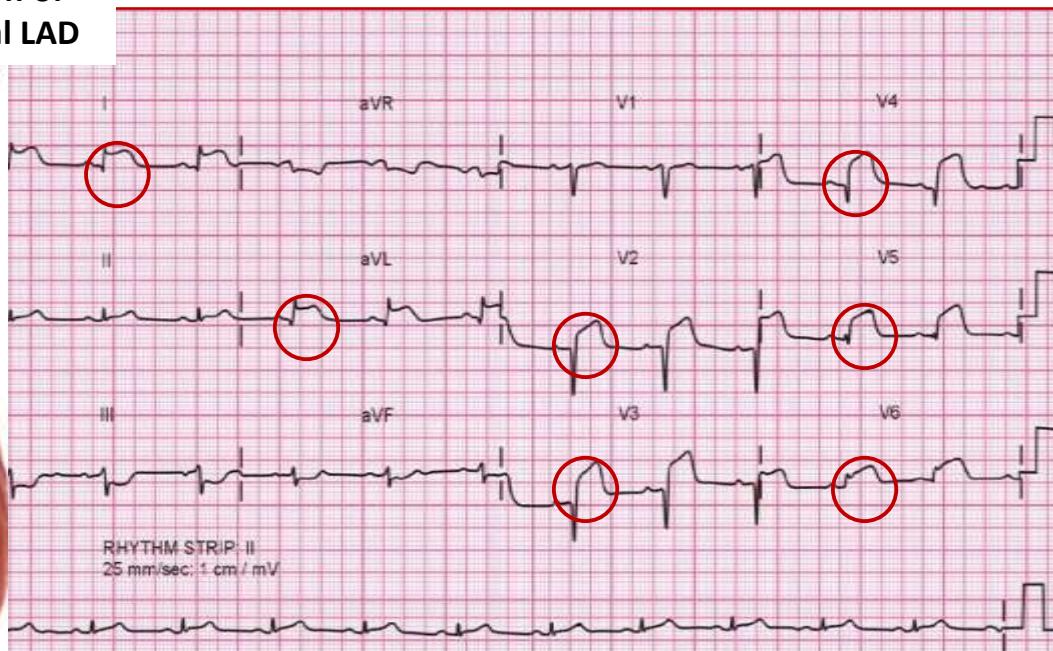
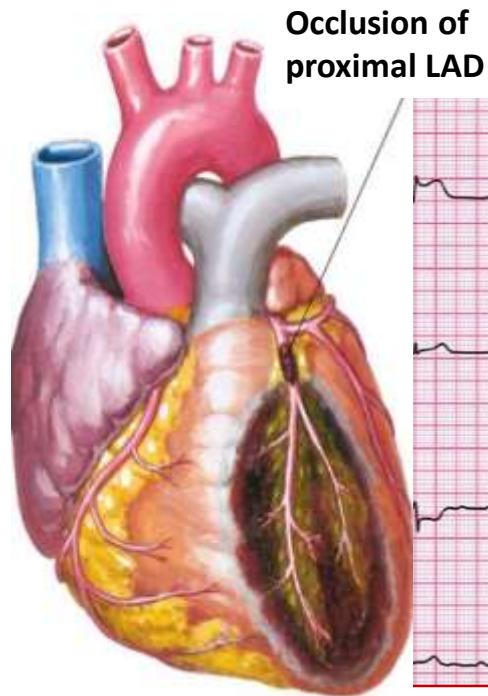


PHILIPS

Evolution of ECG Changes in STEMI



Infark miokardial anteroekstensif



- Maximal ST segment elevation in leads I and aVL and lead V₅ or V₆ or both

Late

Phase 3: Second and Third Day (24 to 72 Hours)

ECG Changes

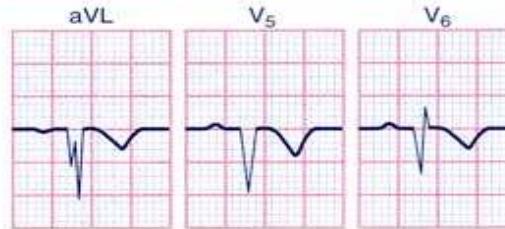
In facing leads I, aVL, and V₅-V₆:

- Abnormal Q waves and small R waves with T wave inversion in leads I and aVL
 - QS waves or complexes and decreased or absent R waves with T wave inversion in lead V₅ or V₆ or both
 - Return of ST segments to baseline
- In opposite leads II, III, and aVF:
- Tall T waves in leads II, III, and aVF
 - Return of ST segments to baseline



**Infarkt
mioKardial
lateral**

EVOLUSI



INFERIOR MYOCARDIAL INFARCTION

Early

**Phase 1: First Few Hours
(0 to 2 Hours)**

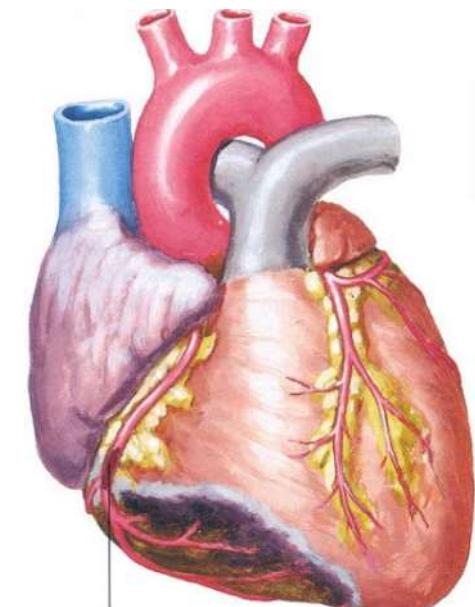
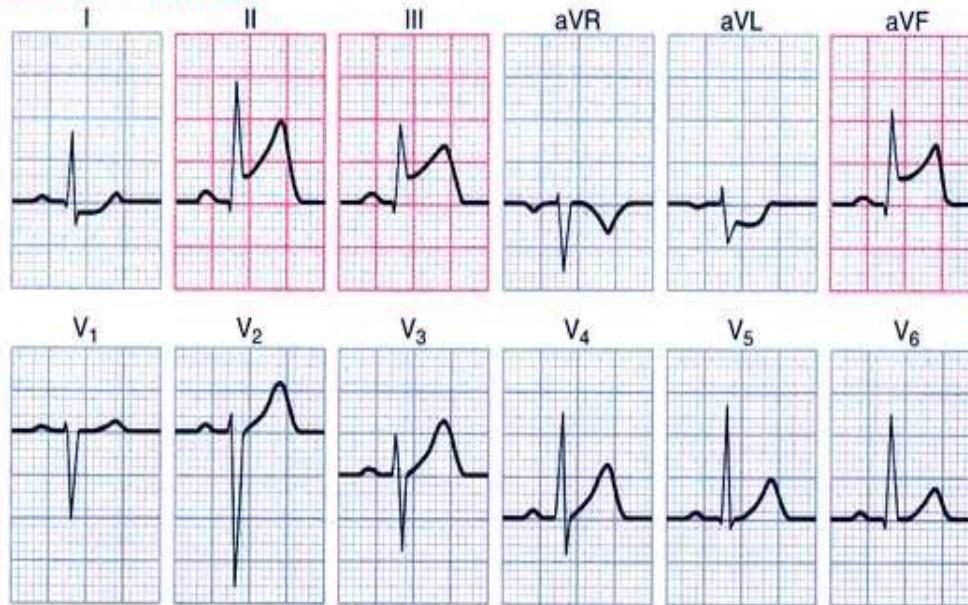
ECG Changes

In facing leads II, III, and aVF:

- ST segment elevation with tall T waves and taller than normal R waves in leads II, III, and aVF

In opposite leads I and aVL:

- ST segment depression in leads I and aVL



Occlusion of right coronary artery

RIGHT VENTRICULAR MYOCARDIAL INFARCTION



POSTERIOR MYOCARDIAL INFARCTION

Early

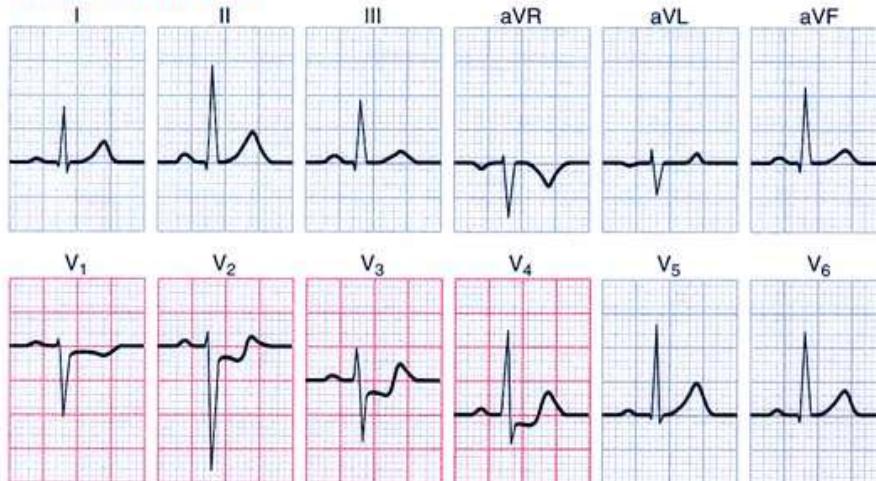
Phase 1: First Few Hours (0 to 2 Hours)

ECG Changes

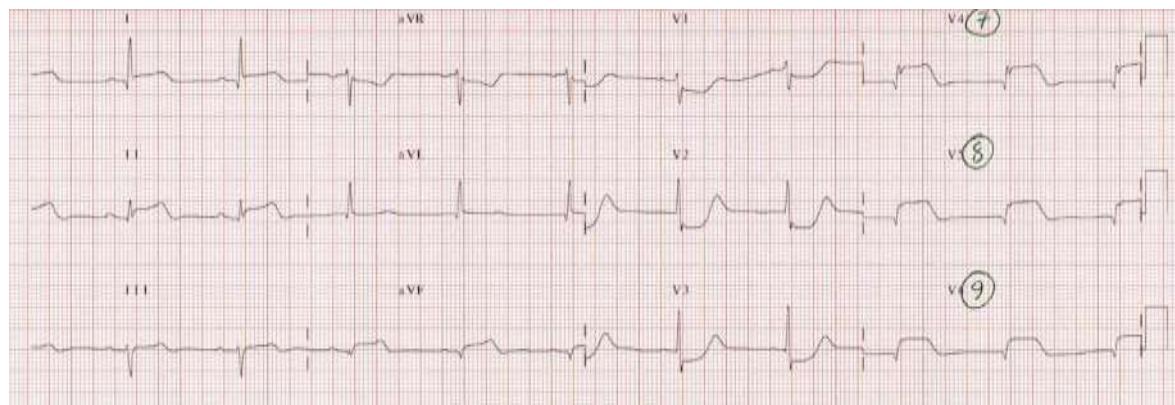
In facing leads: No facing leads present.

In opposite leads V₁-V₄:

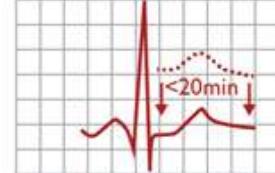
- ST segment depression in leads V₁-V₄
- T wave inversion in V₁ and sometimes V₂



Bayangan
cermin



ECG NSTEMI

ECG pattern	Criteria	Signifying	Figure
a Isolated T-wave inversion	T-wave inversion >1 mm in ≥ 5 leads including I, II, aVL, and V2–V6	Only mildly impaired prognosis	 I, II, aVL, or V2 to V6
b ST-segment depression	J point depressed by ≥ 0.05 mm in leads V2 and V3 or ≥ 1 mm in all other leads followed by a horizontal or downsloping ST-segment for ≥ 0.08 s in ≥ 1 leads (except aVR)	More severe ischaemia	 ≥ 1 leads  ≥ 1 leads
c Transient ST-segment elevation	ST segment elevation in ≥ 2 contiguous leads of ≥ 2.5 mm in men <40 years, ≥ 2 mm in men ≥ 40 years, or ≥ 1.5 mm in women regardless of age in leads V2–V3 and/or ≥ 1 mm in the other leads lasting <20 min	Only mildly impaired prognosis	 ≥ 2 contiguous leads

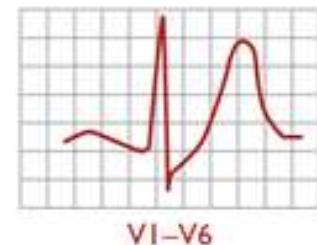
ECG NSTEMI

d

De Winter ST-T

1–3 mm upsloping ST-segment depression at the J point in leads V1–V6 that continue into tall, positive, and symmetrical T waves

Proximal LAD occlusion/
severe stenosis



e

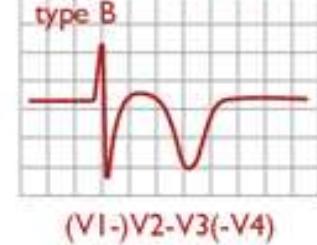
Wellens sign

Isoelectric or minimally elevated J point (<1 mm)
+
biphasic T wave in leads V2 and V3 (type A)
or
symmetric and deeply inverted T waves in leads V2 and V3, occasionally in leads V1, V4, V5, and V6 (type B)

Proximal LAD occlusion/
severe stenosis

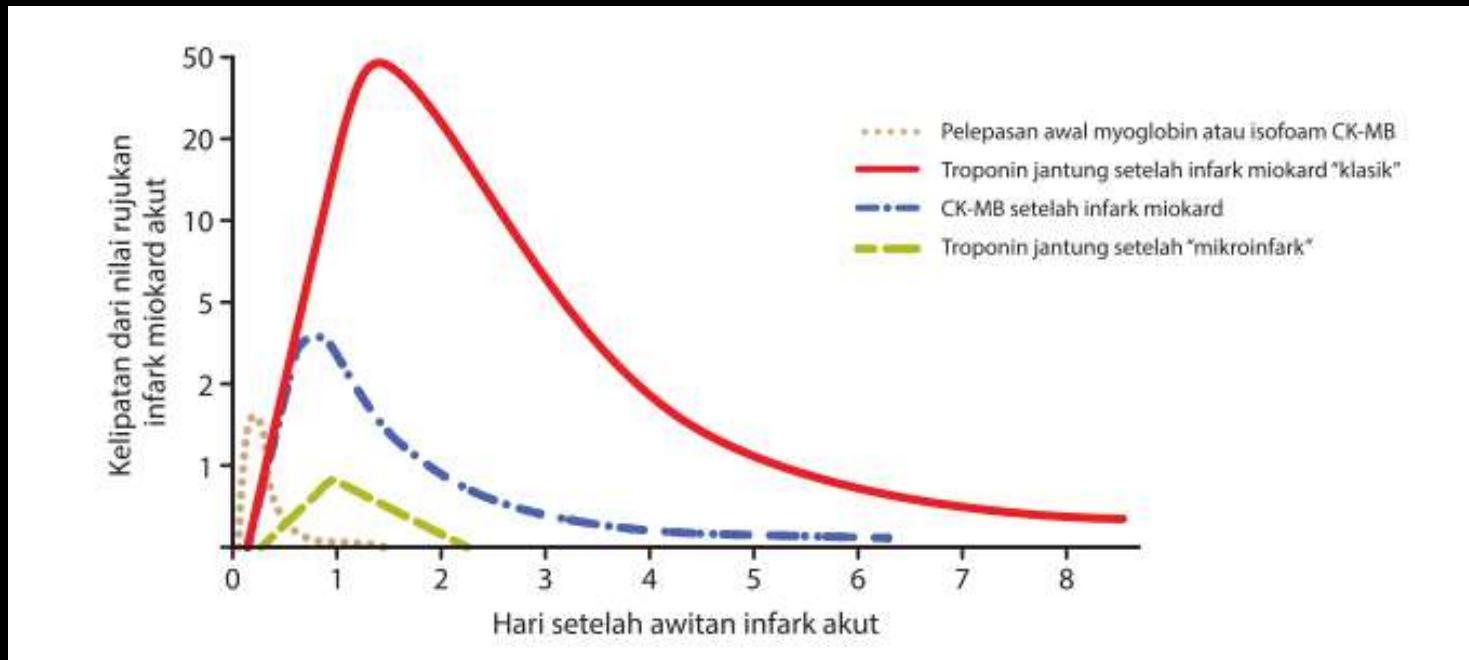


(V1-)V2-V3(-V4)



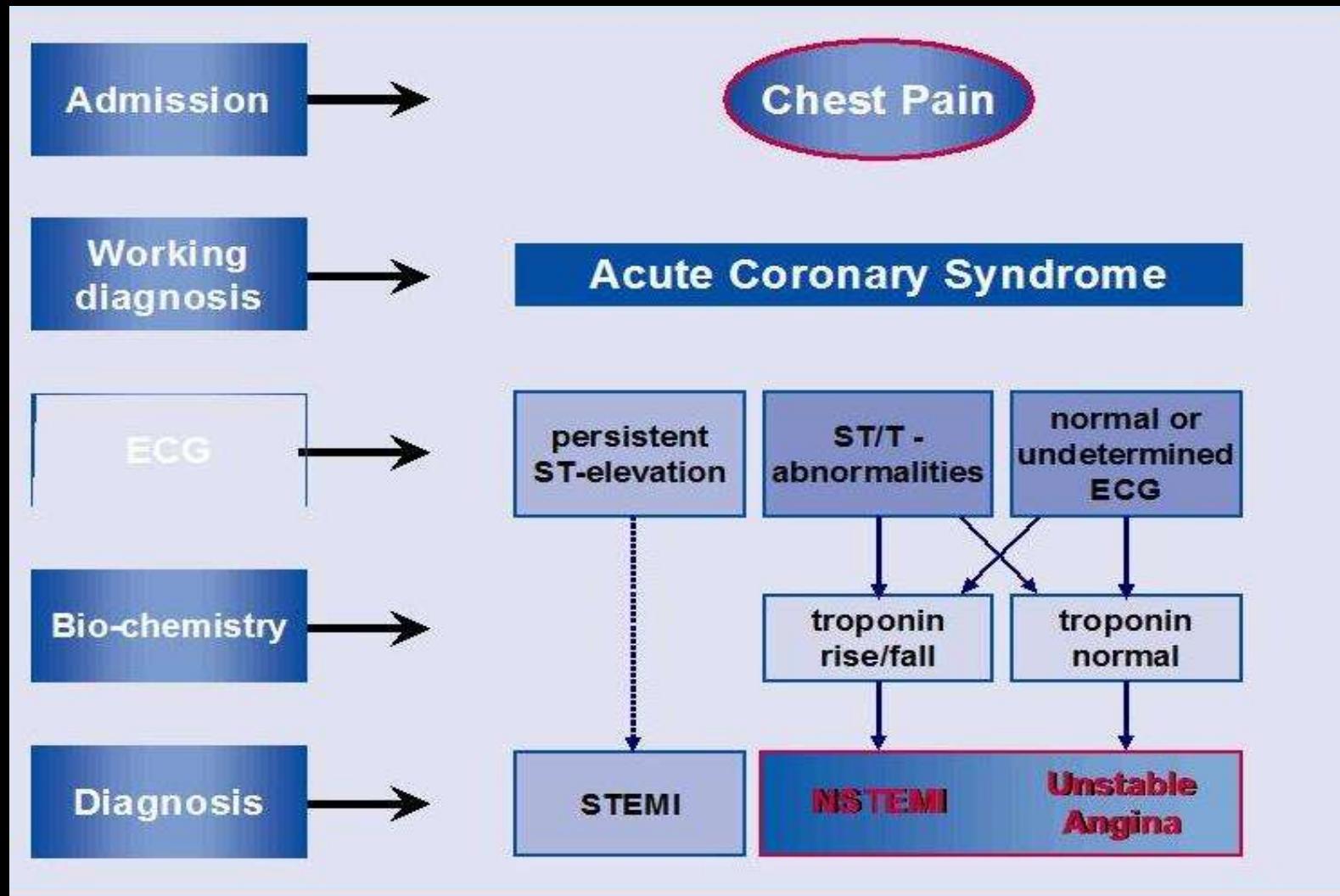
(V1-)V2-V3(-V4)

Marka Jantung



- Pada pasien dg SKA Peningkatan enzim Troponin terjadi **3-4 jam setelah onset gejala dan dapat bertahan 2 minggu**
- CKMB meningkat **4-6 jam** mencapai puncak **12 jam**, menetap 2 hari
- Pemeriksaan serial harus dilakukan dlm **6-12 jam** jika pemeriksaan pertama negative

Klasifikasi SKA



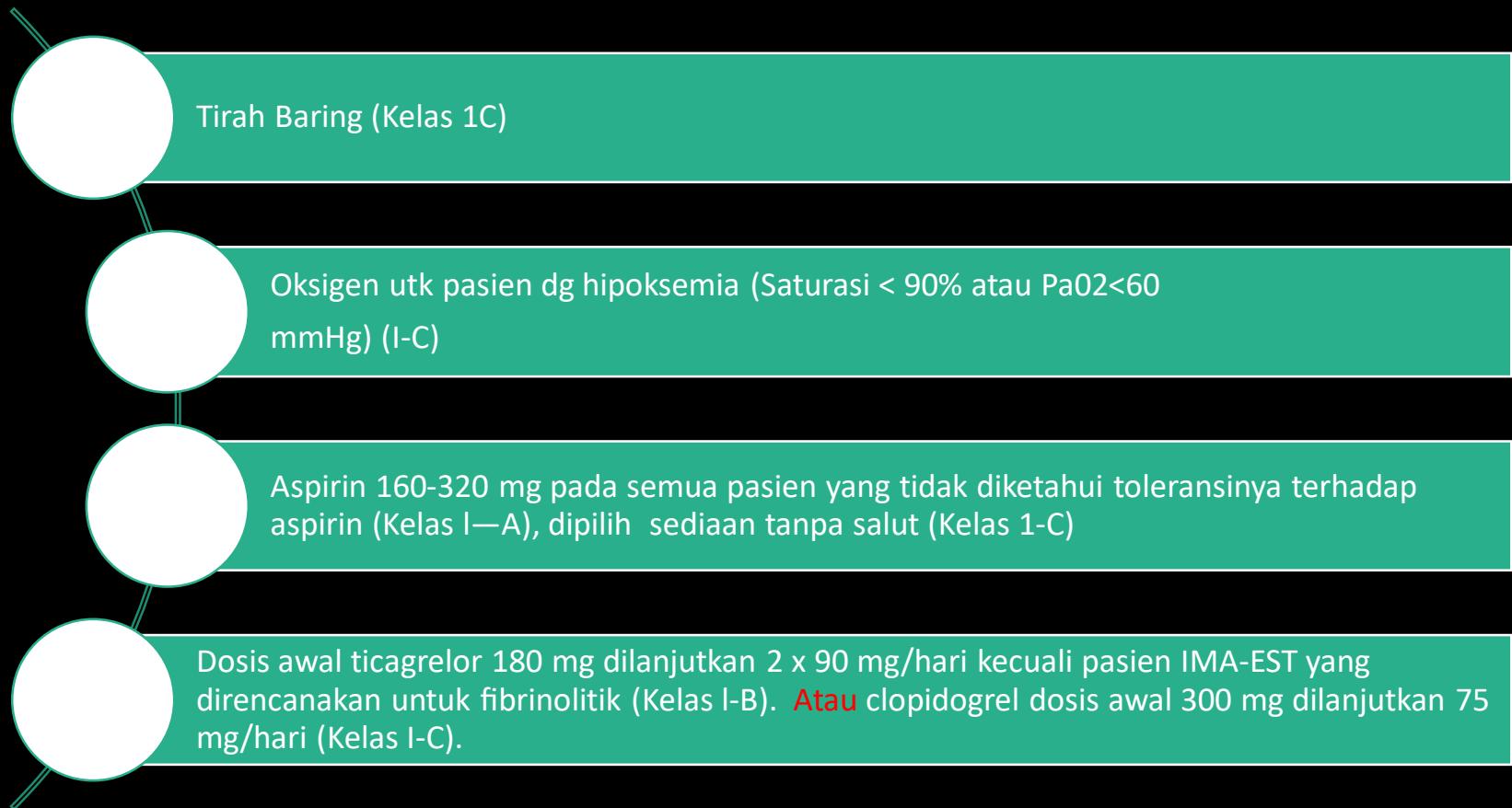
Klasifikasi Rekomendasi

Tabel 1.1. Klasifikasi rekomendasi tata laksana sindrom koroner akut

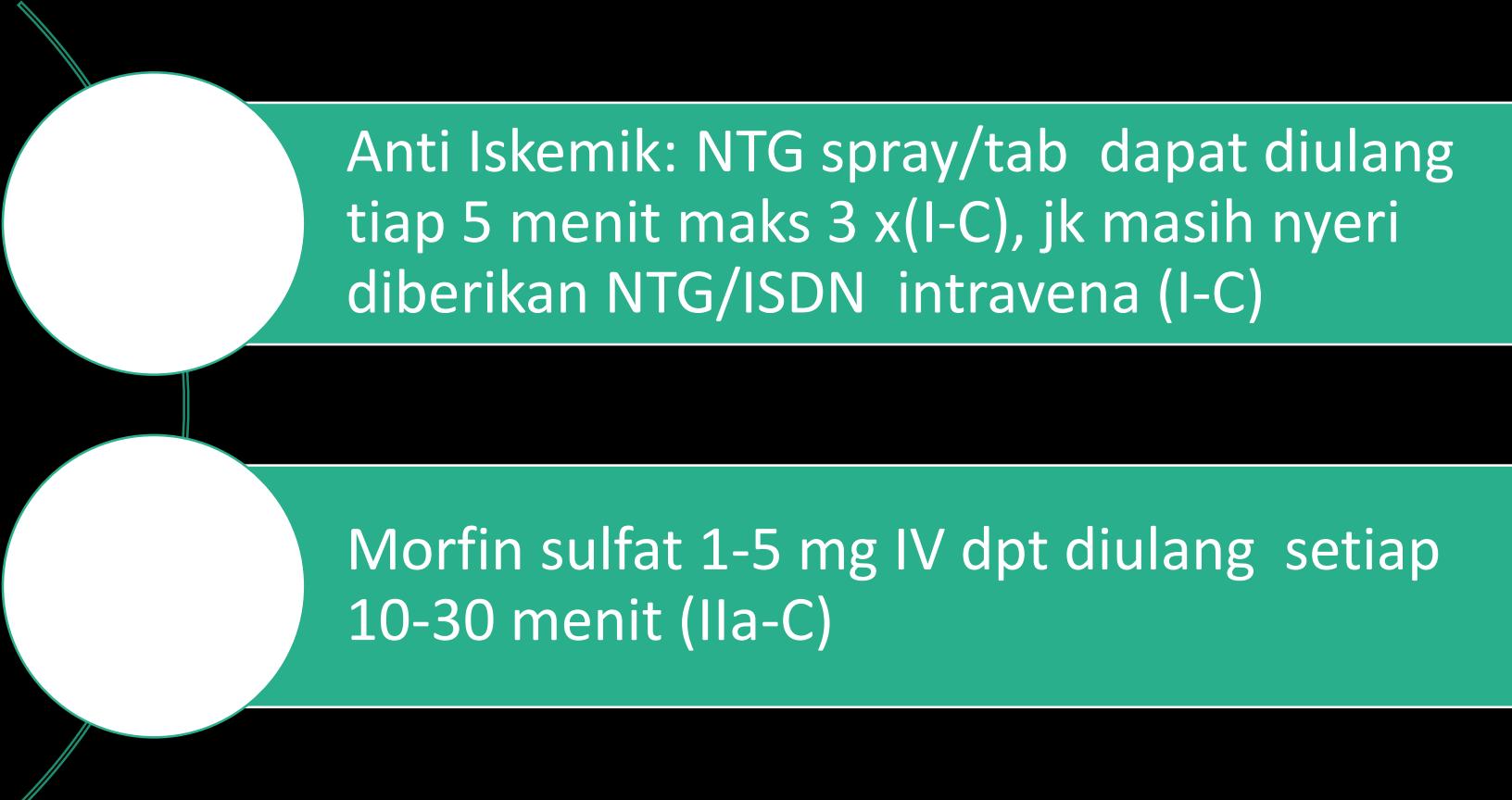
Kelas I	Bukti dan/atau kesepakatan bersama bahwa pengobatan tersebut bermanfaat dan efektif.
Kelas II	Bukti dan/atau pendapat yang berbeda tentang manfaat pengobatan tersebut.
Kelas IIa	Bukti dan pendapat lebih mengarah kepada manfaat atau kegunaan, sehingga beralasan untuk dilakukan.
Kelas IIb	Manfaat atau efektivitas kurang didukung oleh bukti atau pendapat, namun dapat dipertimbangkan untuk dilakukan.
Kelas III	Bukti atau kesepakatan bersama bahwa pengobatan tersebut tidak berguna atau tidak efektif, bahkan pada beberapa kasus kemungkinan membahayakan.
Tingkat bukti A	Data berasal dari beberapa penelitian klinik acak berganda atau meta-analisis
Tingkat bukti B	Tingkat Data berasal dari satu penelitian acak berganda atau beberapa penelitian tidak acak
Tingkat bukti C	Data berasal dari konsensus opini para ahli dan/atau penelitian kecil, bukti C studi retrospektif, atau registri

Manajemen

Tindakan Umum & Langkah Awal



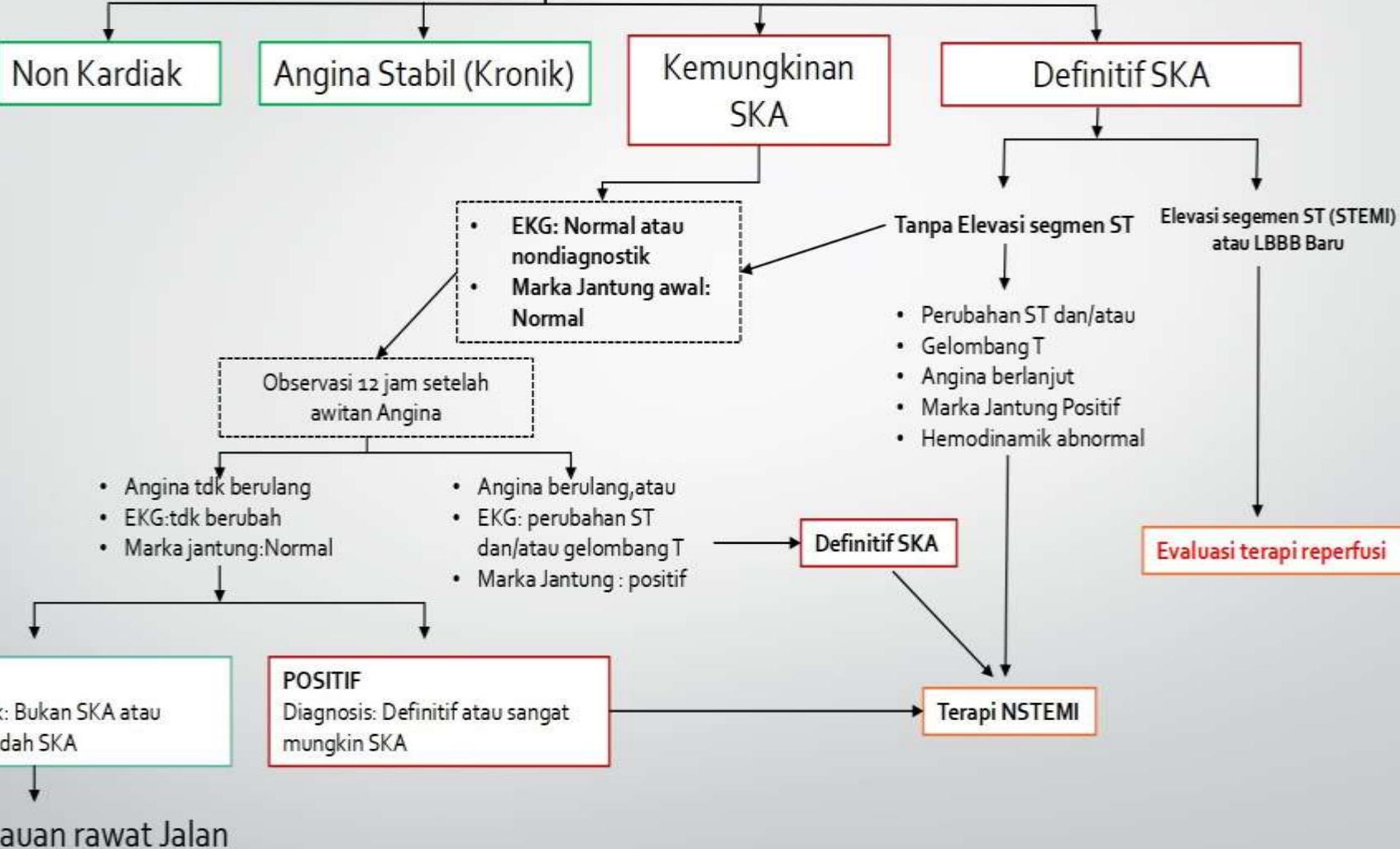
Tindakan Umum & Langkah Awal..Lanjutan..



Anti Iskemik: NTG spray/tab dapat diulang tiap 5 menit maks 3 x(I-C), jk masih nyeri diberikan NTG/ISDN intravena (I-C)

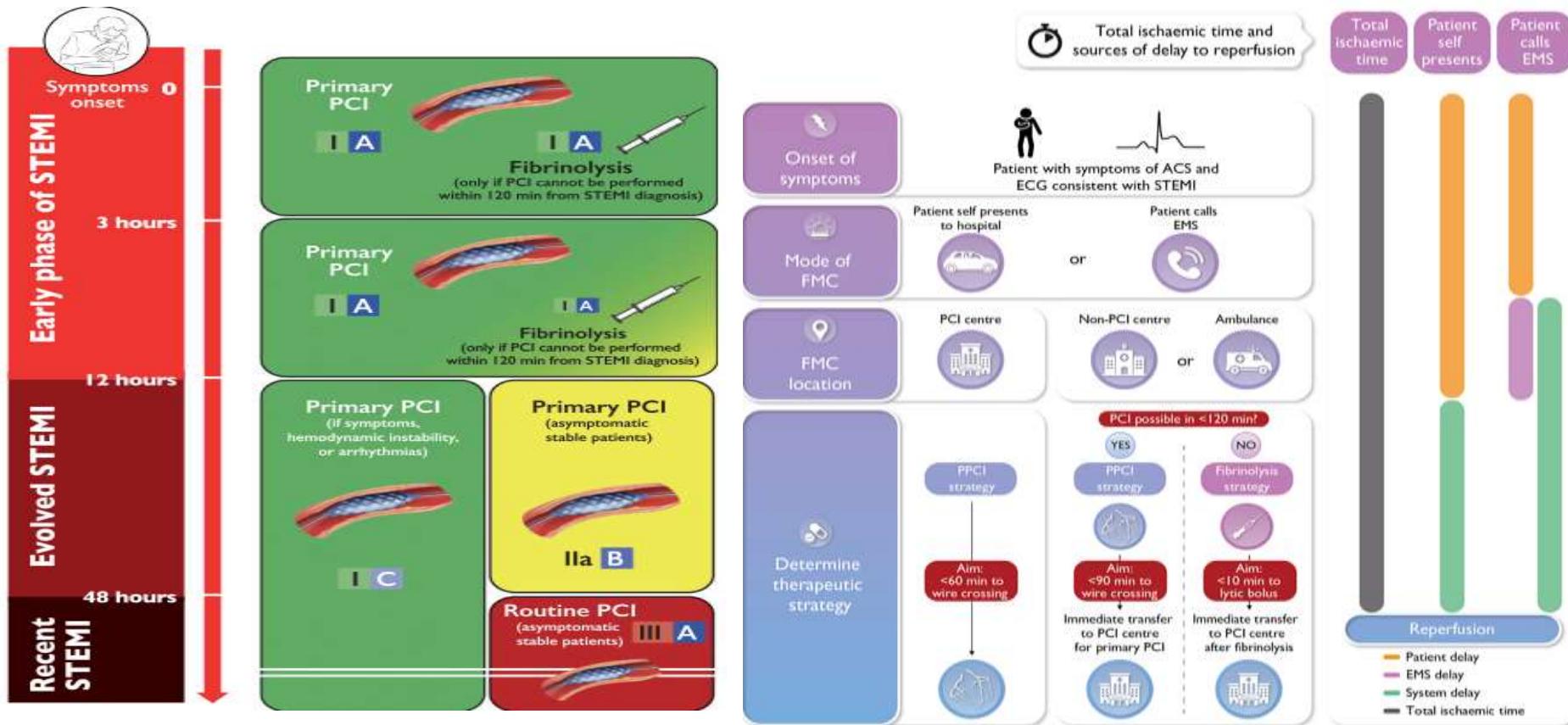
Morfin sulfat 1-5 mg IV dpt diulang setiap 10-30 menit (IIa-C)

Persangkaan SKA



STEMI

Strategi Reperfusi pada STEMI

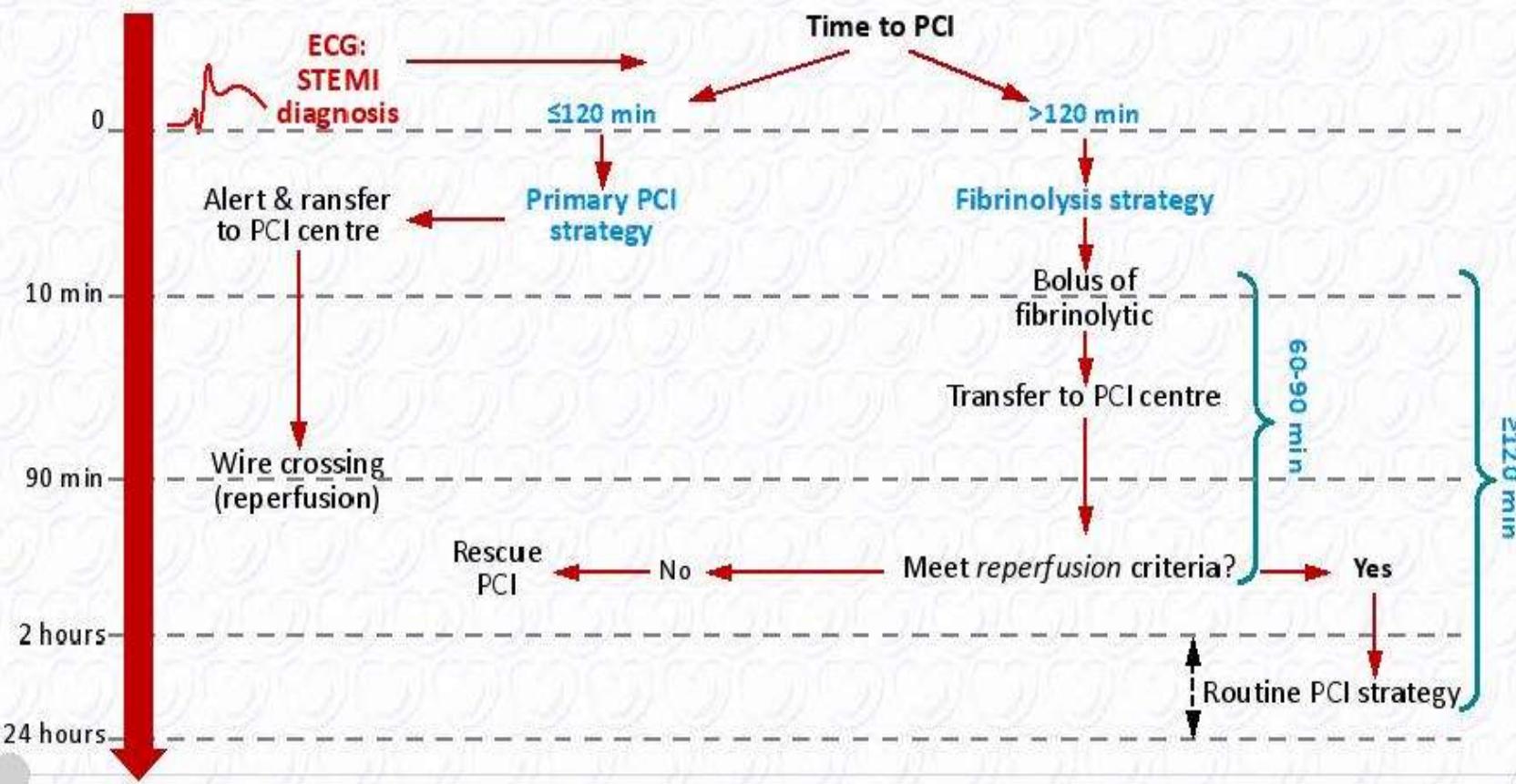


2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

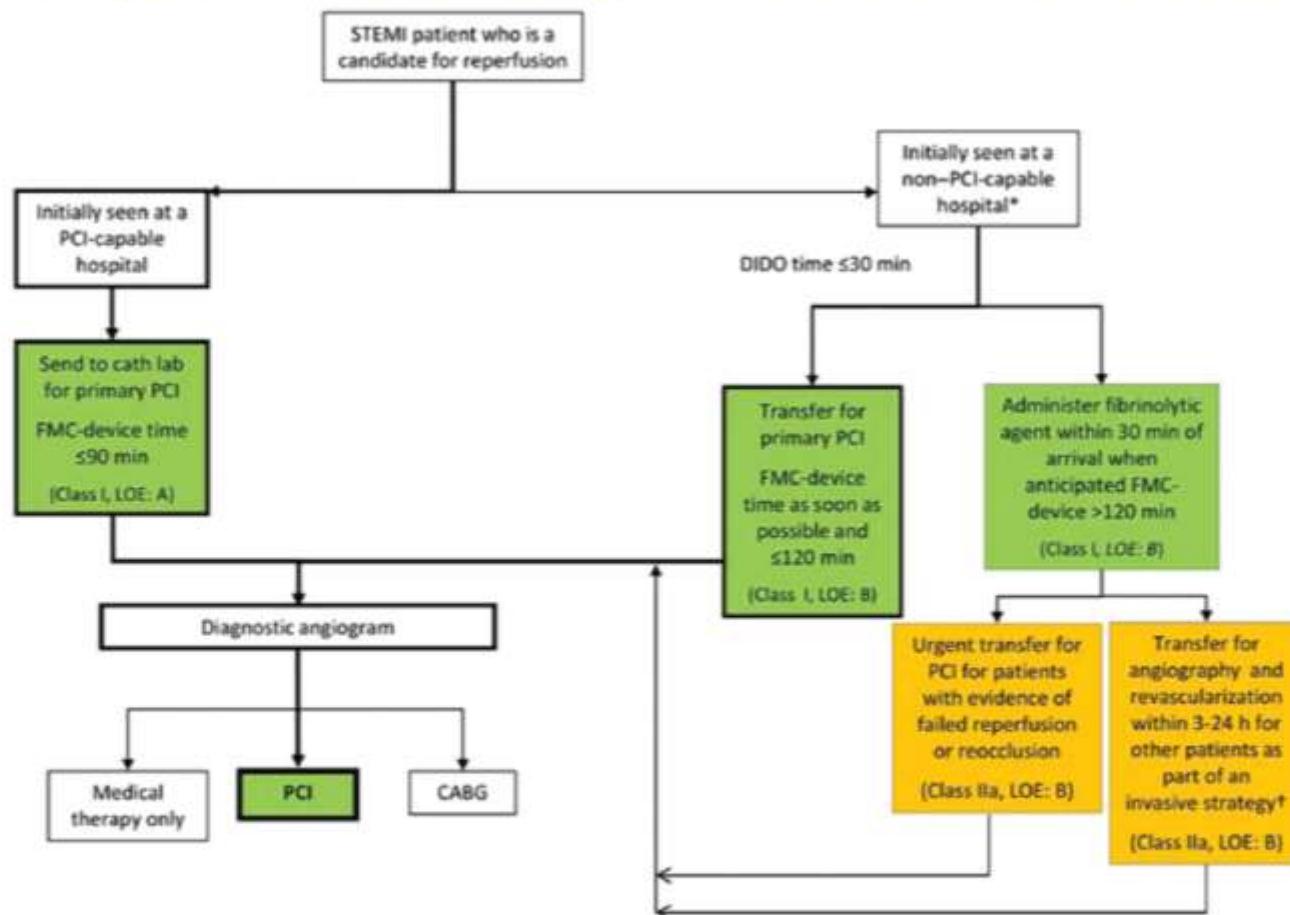
ESC Guideline of Management of Acute Coronary Syndromes, 2023

Maximum target times according to reperfusion strategy selection in patients presenting via EMS or in a non-PCI centre

Strategy clock



Reperfusion Therapy for Patients with STEMI



*Patients with cardiogenic shock or severe heart failure initially seen at a non-PCI-capable hospital should be transferred for cardiac catheterization and revascularization as soon as possible, irrespective of time delay from MI onset (Class I, LOE: B). †Angiography and revascularization should not be performed within the first 2 to 3 hours after administration of fibrinolytic therapy.



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Procedural aspects of PPCI strategy

- Primary PCI of the IRA =I A
- Stenting recommended over balloon angioplasty= I A
- New-generation DES over BMS for primary PCI= I A
- Radial over femoral access=I A
- Routine use of thrombus aspiration **No= III A**
- Routine use of deferred stenting **No = III B**
- Routine revascularization of non-IRA lesions in multivessel disease before hospital discharge Yes= IIa A
- Non-IRA PCI during the index procedure should be considered in patients with cardiogenic shock.= IIa C
- **CABG** should be considered in patients with ongoing ischaemia and large areas of jeopardized myocardium if PCI of the IRA cannot be performed. Otherwise after 3-7 days

Doses of antiplatelet and anticoagulant co-therapies in primary PCI

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	<p>Loading dose of 60 mg orally, followed by a maintenance dose of 10 mg/day.</p> <p>In patients with body weight \leq60 kg, a maintenance dose of 5 mg/day is recommended.</p> <p>Prasugrel is contra-indicated in patients with previous stroke. In patients \geq75 years, prasugrel is generally not recommended, but a dose of 5 mg/day should be used if treatment is deemed necessary.</p>

Doses of antiplatelet and anticoagulant co-therapies in primary PCI(*continued*)

Doses of antiplatelet and parenteral anticoagulant co-therapies in primary PCI	
Antiplatelet therapies (<i>continued</i>)	
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.

Doses of antiplatelet and anticoagulant co-therapies in primary PCI(*continued*)

Doses of antiplatelet and parenteral anticoagulant co-therapies in primary PCI	
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.

Fondaparinux is not recommended for primary PCI.



Regimen Fibrinolitik untuk Infark Miokard Akut

Agen	Dosis Awal	Ko Terapi Antitrombotik	Kontraindikasi spesifik
Streptokinase (Sk)	1,5 juta U dalam 100 ml dextrose 5% atau dlm larutan salin 0,9% dlm 30-60 menit	Heparin iv selama 24-48 jam	Sebelum SK atau Anistreplase
Alteplase (tPA)	Bolus 15mg IV 0,75 mg/kg selama 30 menit, kemudian 0,5 mg/kg selama 60 mrnit Dosis total tidak lebih dari 100 mg	Heparin IV selama 24-48 jam	
Tenecteplase*	Dosis tunggal bolus intravena sesuai berat badan, selama 5 detik: - <60 kg: 30 mg - 60—70 kg: 35 mg - 70—80 kg: 40 mg - 80-90 kg: 45 mg - >90 kg: 50 mg	Heparin i.v. selama diberikan 24—48jam	

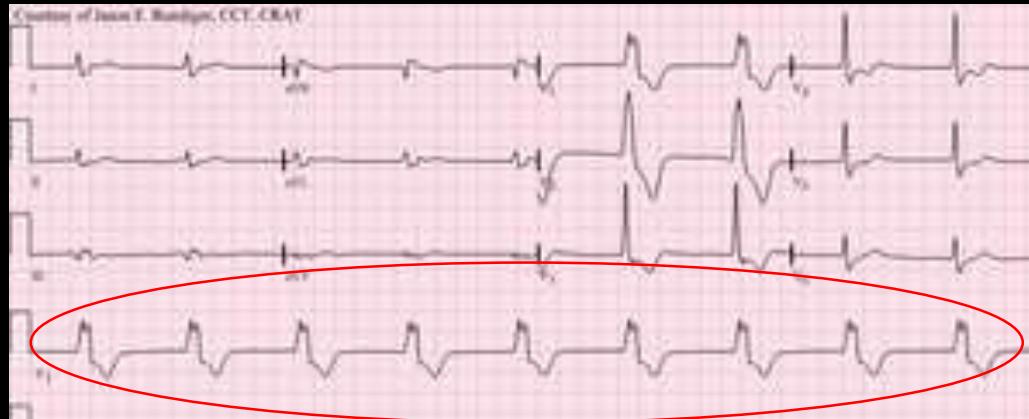
Kontra Indikasi Fibrinolitik

Kontraindikasi Absolut	Kontraindikasi Relatif
Stroke hemoragik atau stroke yg penyebabnya blm diketahui dg awitan kapanpun	Transient Ischaemic Attact(TIA) dlm 6 bulan terakhir
Stroke iskemik 6 bulan terakhir	Pemakaian antikoagulan oral
Kerusakan sistem syaraf sentral dan neoplasma	Kehamilan atau dalam 1 minggu post-partum
Trauma operasi/trauma kepala yg berat dalam 3 minggu terakhir	Resusitasi lama atau traumatisik
Penyakit perdarahan	Hipertensi refrakter (TDS >180 mmHg)
Diseksi aorta	Penyakit hati lanjut Infeksi endokartis
Non compressible puncture pada 24 jam (al biopsy liver, pungsi lumbar)	Ultus peptikum yang aktif

Terapi Reperfusi dengan Trombolitik

Dikatakan berhasil bila:

- Keluhan nyeri dada berkurang atau hilang
- Penurunan Segmen ST > 50% dalam 60-90 menit pemberian fibrinolitik
- Adanya reperfusi aritmia (Accelerated idioventricular rhythm → HR 40-120 x/mnt)



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

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

Doses of fibrinolytic agents and antithrombotic co-therapies (*continued*)

Drug	Initial treatment	Specific contra-indications
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 \geq 75 years of age: loading dose of 75 mg, followed by a maintenance dose of 75 mg/day.	

Doses of fibrinolytic agents and antithrombotic co-therapies (continued)

Drug	Initial treatment	Specific contra-indications
Doses of anticoagulant co-therapies		
Enoxaparin	<p>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.</p> <p>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 m², regardless of age, the s.c. doses are given once every 24 hours.</p>	

Doses of fibrinolytic agents and antithrombotic co-therapies (*continued*)

Drug	Initial treatment	Specific contra-indications
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.	

Maintenance antithrombotic strategy after ST-elevation myocardial infarction

Recommendations	Class	Level
Antiplatelet therapy with low-dose aspirin (75–100 mg) is indicated.	I	A
DAPT in the form of aspirin plus ticagrelor or prasugrel (or clopidogrel if ticagrelor or prasugrel is not available or is contra-indicated) <i>is recommended for 12 months after PCI unless there are contra-indications such as excessive risk of bleeding.</i>	I	A
A PPI in combination with DAPT is recommended in patients at high risk of gastrointestinal bleeding.	I	B
In patients with an indication for oral anticoagulation, oral anti-coagulants are indicated in addition to antiplatelet therapy.	I	C

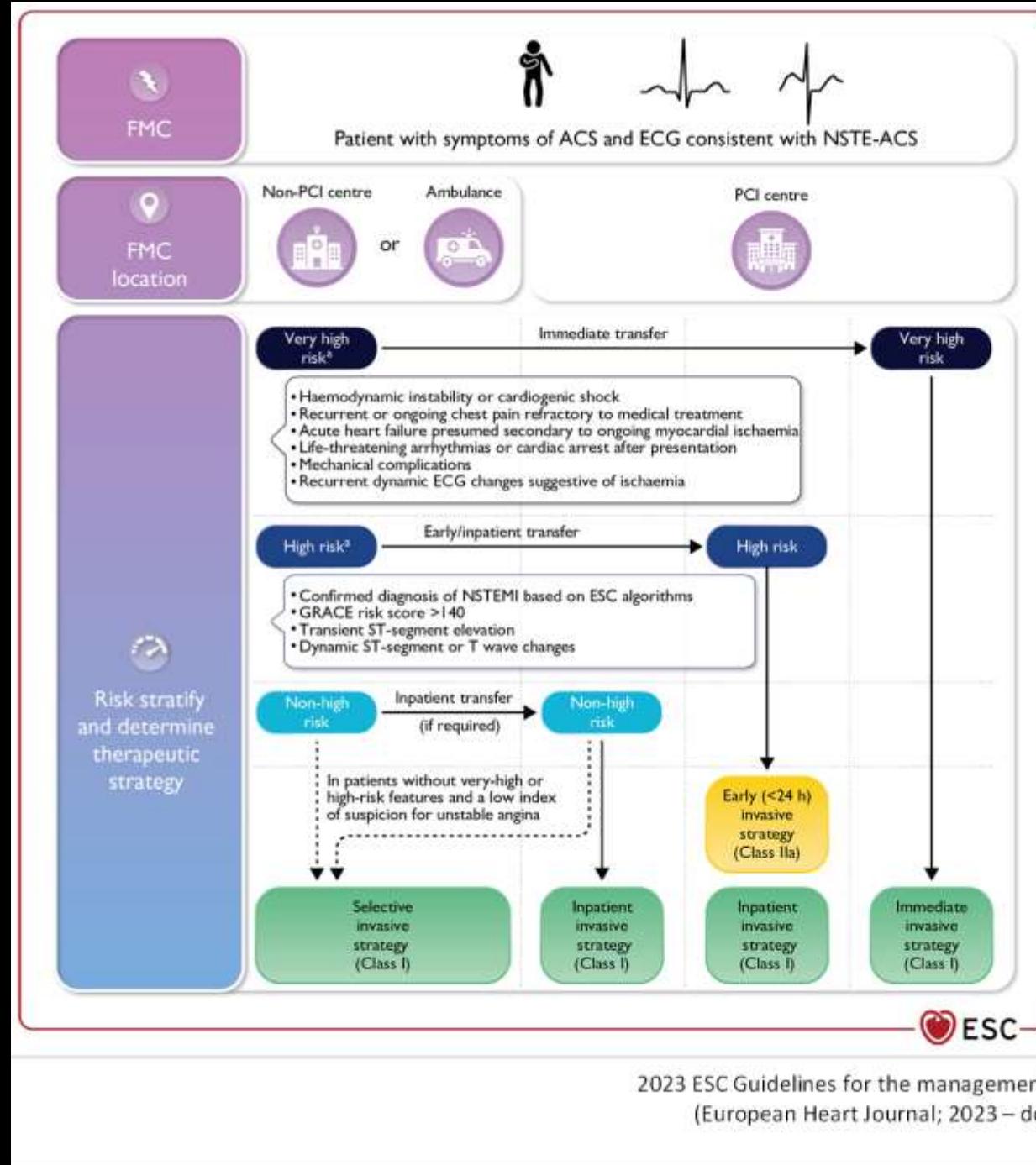
Maintenance antithrombotic strategy after ST-elevation myocardial infarction (continued)

Recommendations	Class	Level
In patients who are at high risk of severe bleeding complications, discontinuation of P2Y ₁₂ inhibitor therapy after 6 months should be considered.	IIa	B
In STEMI patients with stent implantation and an indication for oral anticoagulation, triple therapy should be considered for 1–6 months (according to a balance between the estimated risk of recurrent coronary events and bleeding).	IIa	C
DAPT for 12 months in patients who did not undergo PCI should be considered unless there are contra-indications 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.	IIa	C

Maintenance antithrombotic strategy after ST-elevation myocardial infarction (continued)

Recommendations	Class	Level
In high ischaemic risk patients 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.	IIb	B
In low bleeding risk patients who receive aspirin and clopidogrel, low-dose rivaroxaban (2.5 mg twice daily) may be considered.	IIb	B
The use of ticagrelor or prasugrel is not recommended as part of triple antithrombotic therapy with aspirin and oral anticoagulation.	III	C

NSTEACS



Grace Score

Medical History		Findings at Initial Hospital Presentation		Findings During Hospitalization	
① Age in Years	Points	④ Resting Heart Rate, beats/min	Points	⑦ Initial Serum Creatinine, mg/dL	Points
≤29	0	≤49.9	0	0-0.39	1
30-39	0	50-69.9	3	0.4-0.79	3
40-49	18	70-89.9	9	0.8-1.19	5
50-59	36	90-109.9	14	1.2-1.59	7
60-69	55	110-149.9	23	1.6-1.99	9
70-79	73	150-199.9	35	2-3.99	15
80-89	91	≥200	43	≥4	20
≥90	100				
② History of Congestive Heart Failure	24	⑤ Systolic Blood Pressure, mm HG		⑧ Elevated Cardiac Enzymes	15
③ History of Myocardial Infarction	12	≤79.9	24		
		80-99.9	22	⑨ No In-Hospital Percutaneous Coronary Intervention	14
		100-119.9	18		
		120-139.9	14		
		140-159.9	10		
		160-199.9	4		
		≥200	0		
		⑥ ST-Segment Depression	..11		

Platelet inhibition in NSTE-ACS

Recommendations	Class	Level
Oral antiplatelet therapy		
Aspirin is recommended for all patients without contraindications at an initial oral loading dose of 150–300 mg (in aspirin-naïve patients) and a maintenance dose of 75–100 mg daily long-term regardless of treatment strategy.	I	A
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
<ul style="list-style-type: none"> Ticagrelor (180 mg loading dose, 90 mg twice daily) is recommended, in the absence of contraindications, 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). Prasugrel (60 mg loading dose, 10 mg daily dose) is recommended in patients who are proceeding to PCI if no contraindication. 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
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
It is not recommended to administer prasugrel in patients in whom coronary anatomy is not known.	III	B

Anticoagulation in NSTE-ACS

Recommendations	Class	Level
Parenteral anticoagulation is recommended at the time of diagnosis according to both ischaemic and bleeding risks.	I	B
Fondaparinux (2.5 mg s.c. daily) is recommended as having the most favourable efficacy-safety profile regardless of the management strategy.	I	B
Bivalirudin (0.75 mg/kg i.v. bolus, followed by 1.75 mg/kg/hour for up to 4 hours after the procedure) is recommended as alternative to UFH plus GPIIb/IIIa inhibitors during PCI.	I	A
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
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
Enoxaparin (1 mg/kg s.c. twice daily) or UFH are recommended when fondaparinux is not available.	I	B
Crossover between UFH and LMWH is not recommended.	III	B
In NSTEMI patients with no prior stroke/TIA and at high ischaemic risk as well as low bleeding risk receiving aspirin and clopidogrel, low-dose rivaroxaban (2.5 mg twice daily for approximately one year) may be considered after discontinuation of parenteral anticoagulation.	IIb	B

Figure 17

Long-term management after acute coronary syndrome

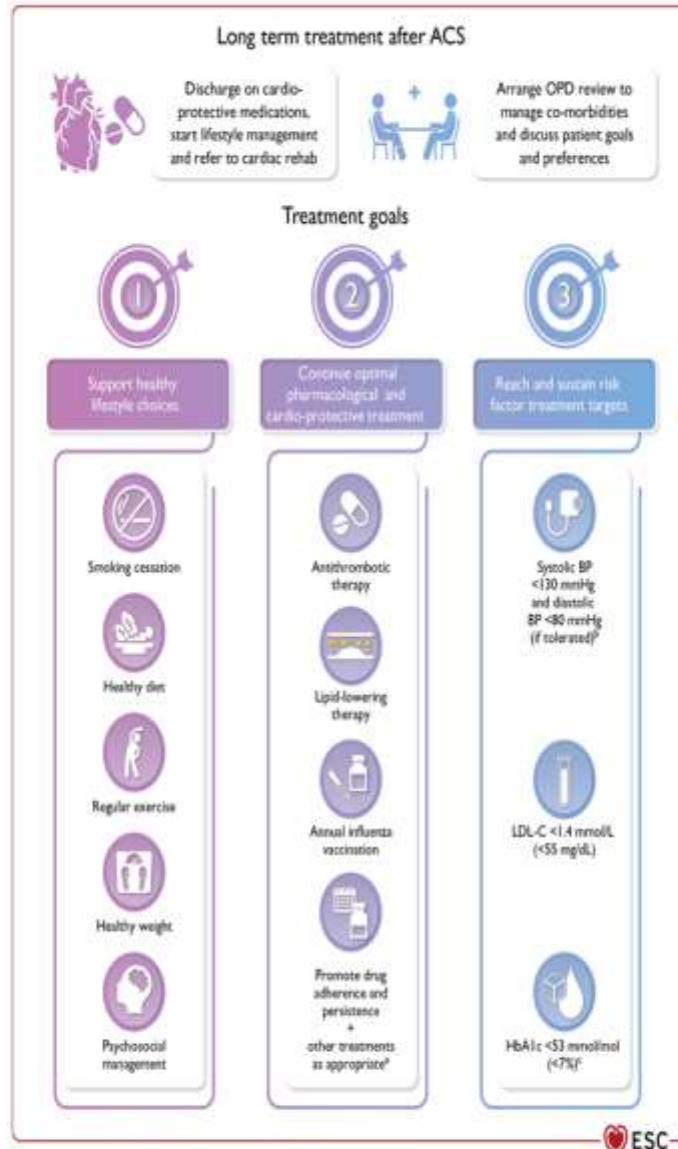
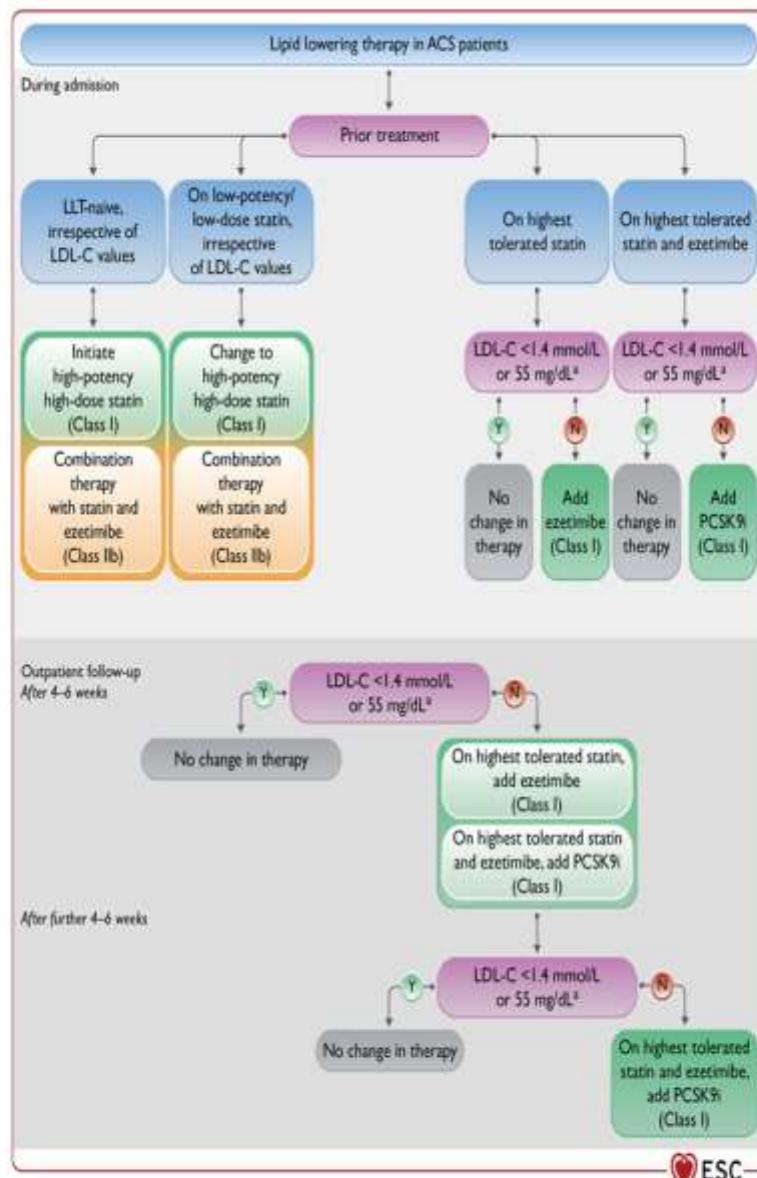


Figure 18

Lipid-lowering therapy in ACS patients



Recommendations for acute coronary syndrome comorbid conditions (2) ESC

Recommendations	Class	Level
Diabetes		
It is recommended to base the choice of long-term glucose-lowering treatment on the presence of comorbidities, including heart failure, CKD, and obesity.	I	A
It is recommended to assess glycaemic status at initial evaluation in all patients with ACS.	I	B
It is recommended to frequently monitor blood glucose levels in patients with known diabetes mellitus or hyperglycaemia (defined as glucose levels ≥ 11.1 mmol/L or ≥ 200 mg/dL).	I	C
Glucose-lowering therapy should be considered in patients with ACS with persistent hyperglycaemia, while episodes of hypoglycaemia should be avoided.	IIa	C

Recommendations for long-term management (3)

Recommendations	Class	Level
Pharmacological treatment		
<i>Lipid-lowering therapy (continued)</i>		
For patients with a recurrent atherothrombotic event (recurrence within 2 years of first ACS episode) while taking maximally tolerated statin-based therapy, an LDL-C goal of <1.0 mmol/L (<40 mg/dL) may be considered.	IIb	B
Combination therapy with high-dose statin plus ezetimibe may be considered during index hospitalization.	IIb	B
<i>Beta-blockers</i>		
Beta-blockers are recommended in ACS patients with LVEF ≤40% regardless of HF symptoms.	I	A
Routine beta-blockers for all ACS patients regardless of LVEF should be considered.	IIa	B

Recommendations for long-term management (4)

Recommendations	Class	Level
Pharmacological treatment		
<i>RAAS system inhibitors</i>		
Angiotensin-converting enzyme (ACE) inhibitors are recommended in ACS patients with HF symptoms, LVEF ≤40%, diabetes, hypertension, and/or CKD.	I	A
Mineralocorticoid receptor antagonists are recommended in ACS patients with an LVEF ≤40% and HF or diabetes.	I	A
Routine ACE inhibitors for all ACS patients regardless of LVEF should be considered.	IIa	A
Adherence to medication		
A polypill should be considered as an option to improve adherence and outcomes in secondary prevention after ACS.	IIa	B

Pengobatan Pasca Perawatan Pasien SKA

- ❖ Obat-obat untuk mengontrol keluhan iskemia harus dilanjutkan
- ❖ Antipaltelet (Aspirin dan atau clopidogrel)
- ❖ Beta-blocker
- ❖ ACE inhibitor

Modifikasi Faktor Risiko

- ❖ Berhenti merokok
- ❖ Hindari minuman beralkohol
- ❖ Pertahankan BB optimal
- ❖ Aktivitas fisik sesuai dengan hasil treadmill atau tanpa keluhan sesak dengan target 150 menit / minggu
- ❖ Diet
- ❖ Rendah lemak jenuh dengan kolesterol, bila perlu dengan target $LDL < 55 \text{ mg/dL}$
- ❖ Pengendalian hipertensi
- ❖ Pengendalian ketat gula darah pada penderita DM ($HbA1C < 7\%$)
- ❖ Kendali stress

REFERENSI

- Pedoman Tata Laksana Sindrom Koroner Akut. Perhimpunan Dokter Spesialis Kardiovaskular Indonesia. Pedoman Tatalaksana Sindrom Koroner Akut. 2018
- 2023 ESC Guidelines for the management of acute coronary syndromes
- 2020 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation
- 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation
- 2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction
- 2014 ACCF/AHA Guidelines for the Management of Patients With Unstable Angina/Non –ST-Elevation Myocardial Infarction



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YOU**

Travel and driving

	GROUP 1 ENTITLEMENT ODL – CAR, M/CYCLE	GROUP 2 ENTITLEMENT VOC – LGV/PCV
ACS PCI	If successfully treated by PCI driving may recommence after 1/52 If not successfully treated by PCI driving may recommence after 4/52	All ACS disqualify the license holder from driving for at least 6/52. Re/licensing may be permitted thereafter provided: The exercise or other functional test requirements can be met.
CABG	Driving must cease for at least 4/52.	Disqualifies from driving for at least 3/12. Re/licensing may be permitted thereafter
<u>AIR TRAVEL after ACS</u>		
	Low risk: <65, first event, successful reperfusion, EF>45%, no complications	Fly after 3 days
	Medium risk: EF>40%,no symptom of HF, No inducible ischemia or arrhythmia	Fly after 10 days
	High risk: EF<40%,HF +, Pending further investigation, revascularization or device therapy	Defer travel until condition stable