Akut Koroner Sendromlar ve Güncel Yaklaşım

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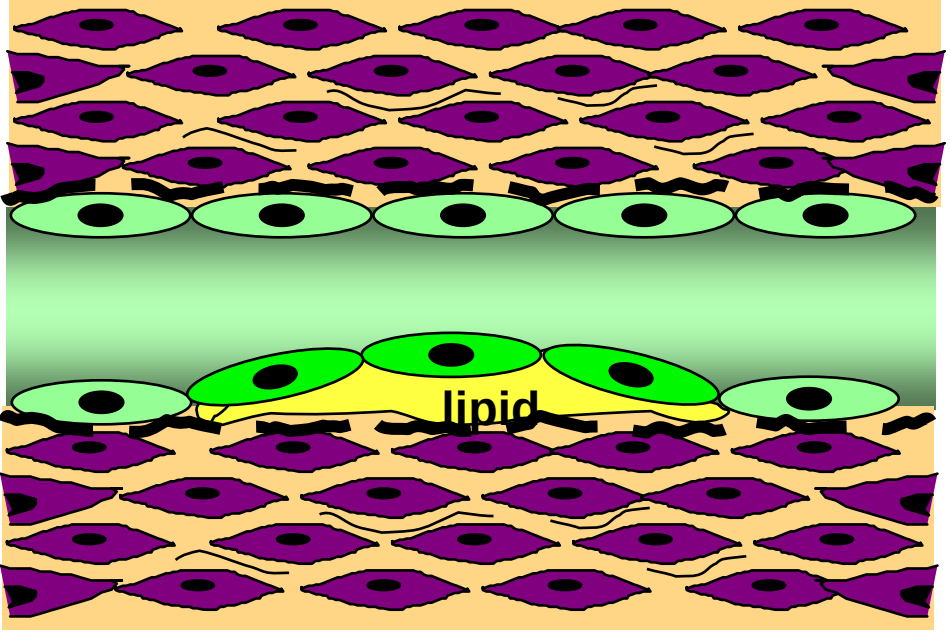
Kılavuzlar

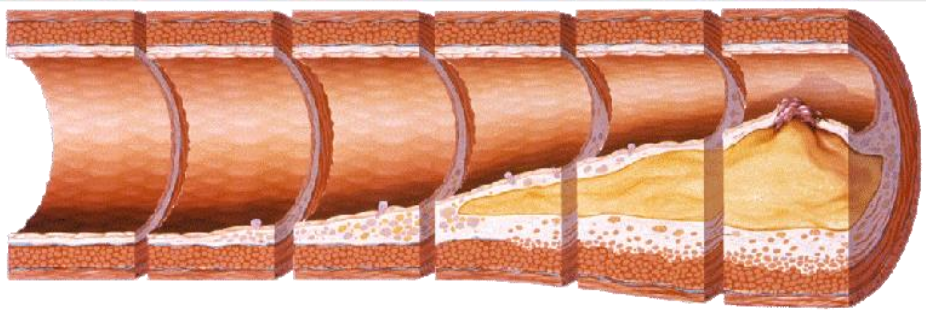
2011 Israrcı ST-segment yükselmesi belirtileri göstermeyen hastalarda Akut Koroner Sendromların (AKS) tedavi kılavuzları (ESC).

2012 Kardiyopulmoner Resüsitasyon ve Acil Kardiyak Bakım Bilimi için Amerikan Kalp Derneği (AHA) Kılavuzu & İlk Yardım için AHA ve Amerikan Kızıl Haç Kılavuzu.

2013 Kararlı Koroner Arter 2013 Kılavuzu (ESC).

2015 Acute Coronary Syndromes (ACS) in patients presenting without persistent ST-segment elevation (ESC).





Kararsız plak üzerinde, endotel hasarı (rüptüre, eroze plak) ve trombüs gelişmesi,

Vazokonstrüksiyon

Ateroskleroz hariç diğer nadir nedenler:

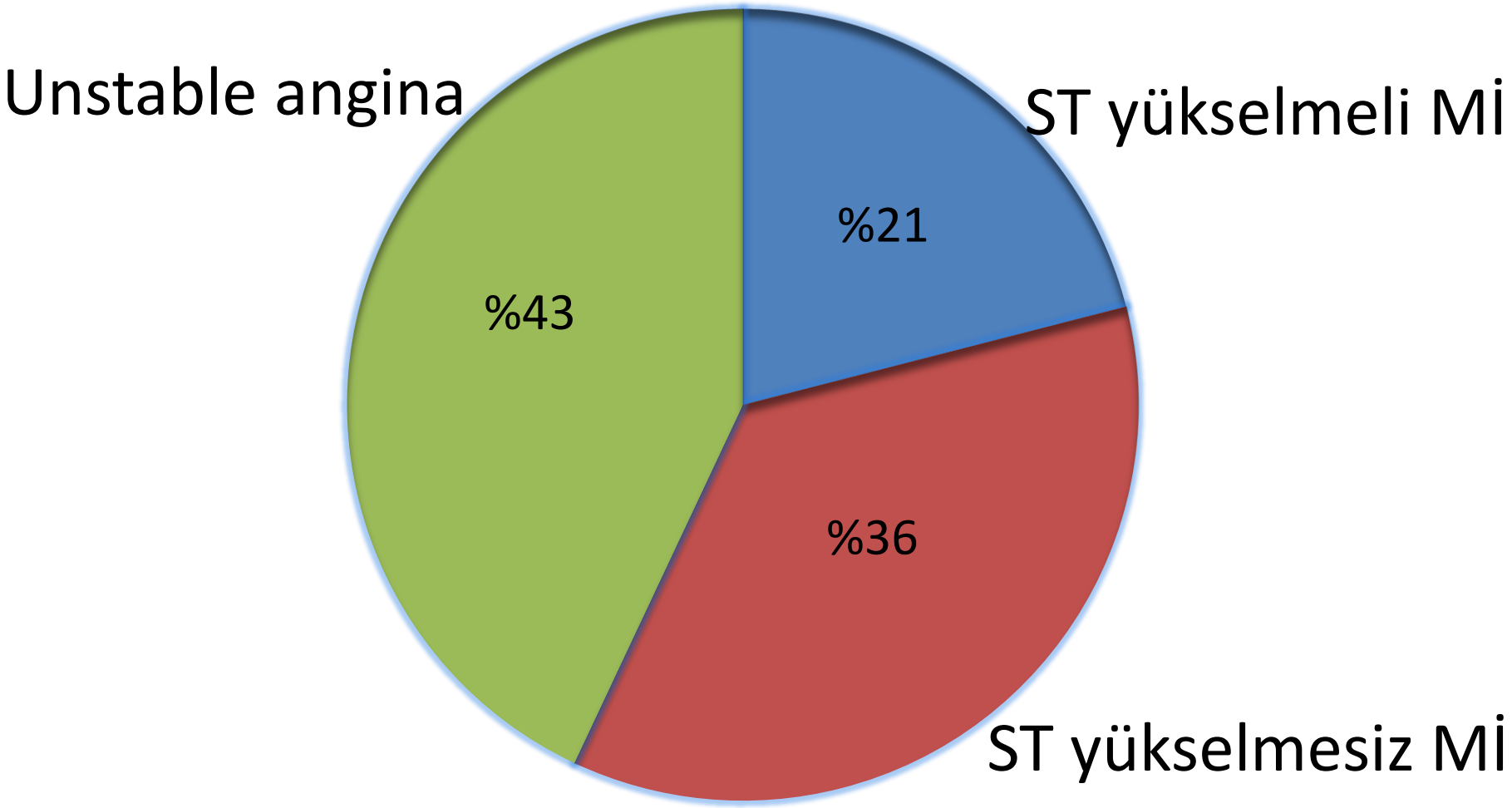
Travma, diseksiyon, emboli, anomaliler, kokain kullanımı

Sonuçta damar lümeninde;

- **Tam tıkanma ve fibrin eklenmesi → STEMI**
- **Tam olmayan tıkanma**
  - Nekroz (-) → **Unstable angina**
  - Distal embolilere bağlı nekroz → **NSTEMI**

Acile servise göğüs ağrısı başvuranların 1/3'ünü AKS oluşturmaktadır.

Akut Koroner Sendromlarda hastaneye yatışlar

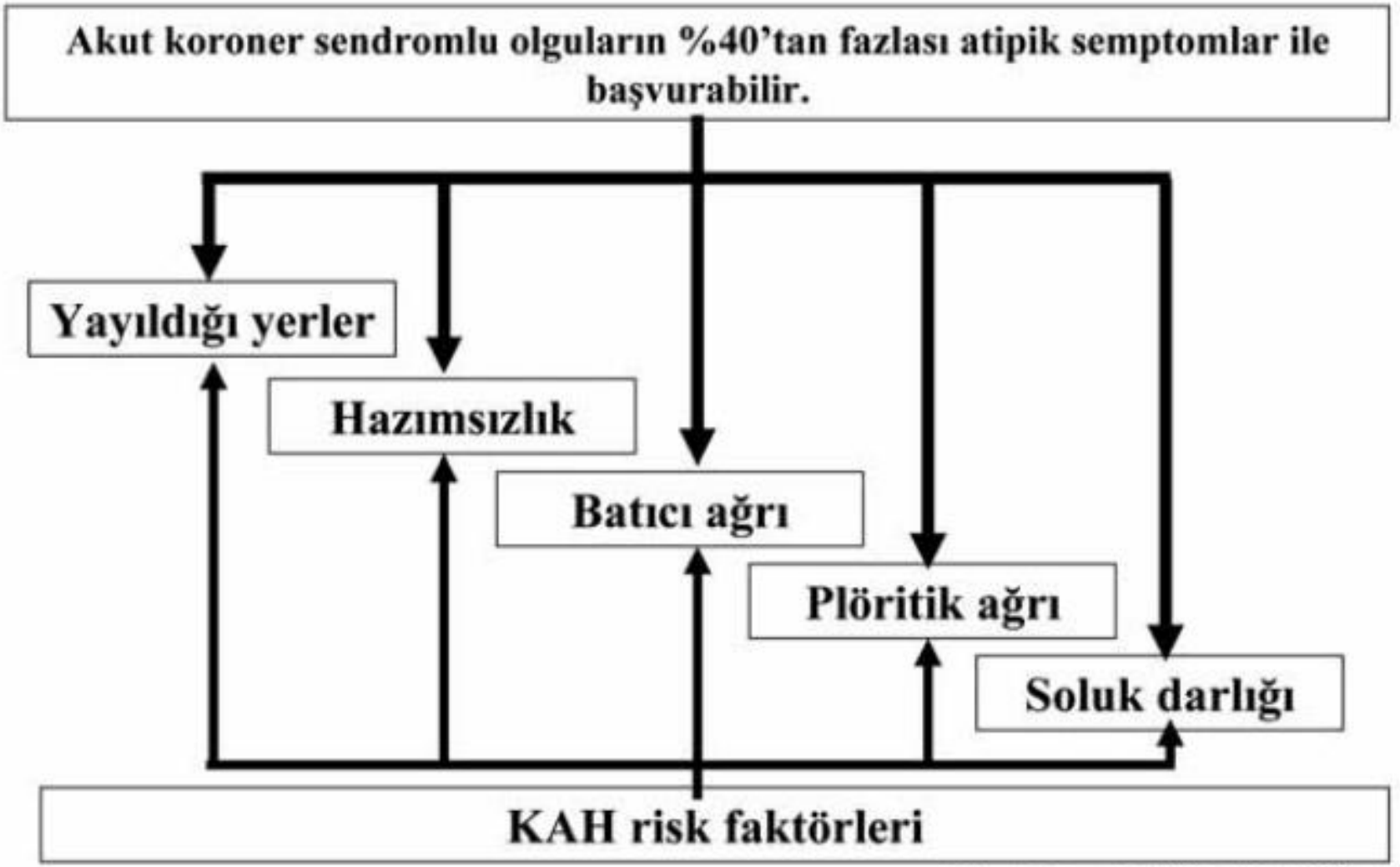


AKS

Başvuru → Göğüs ağrısı

- İstirahat anginası (> 20 dk) → **% 80**
- Yeni başlayan (<2 ay) efor angina
  - En az CCSC II veya III şiddetinde
- Stable anginanın sıklığı ve ciddiyetine artış olması
  - Akselere angina, CCSC class III
- Post MI angina

% 20

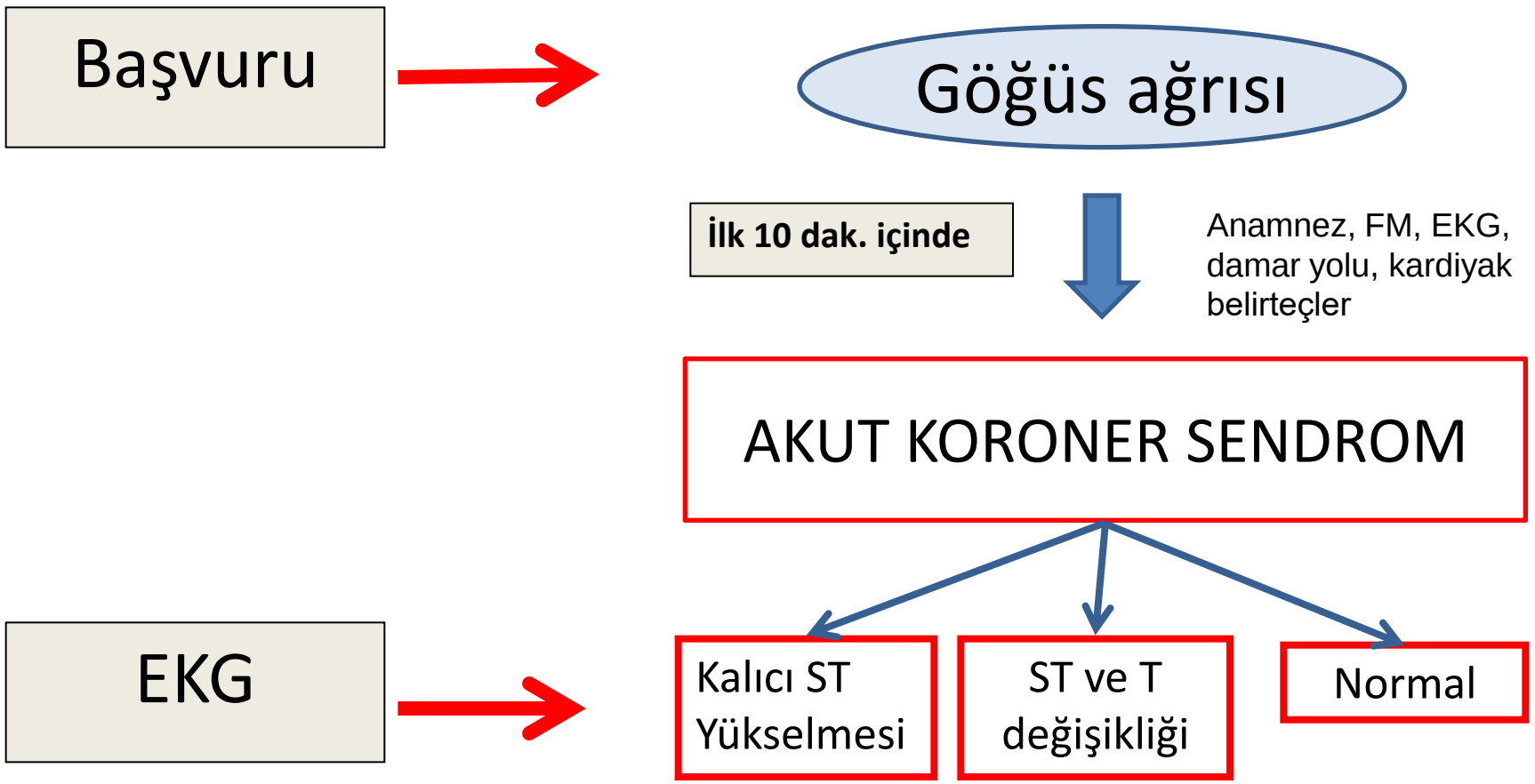


Eur Heart J 2002;23:1089-40

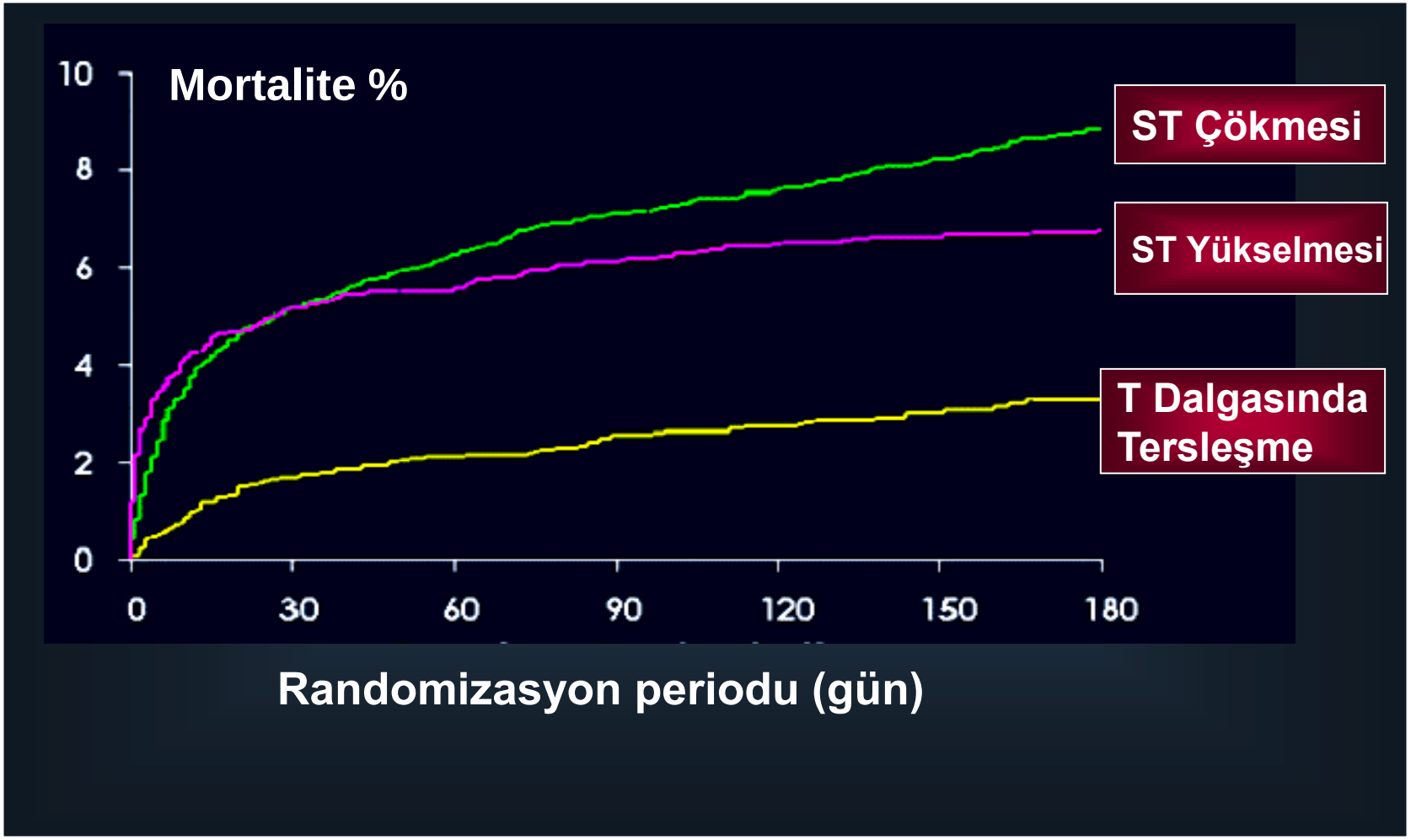
Başvuru → Göğüs ağrısı

- **10 dakika içinde EKG çekilmelidir.**
  - –İlk EKG tanısal değilse ancak hasta semptomatik ise 15-30 dk aralarla EKG çekmeye devam edilmelidir.
- **Kardiyak hasar markırları tayin edilmelidir.**

Öykü,
Fizik muayene,
Damar yolu,
Enzimler,

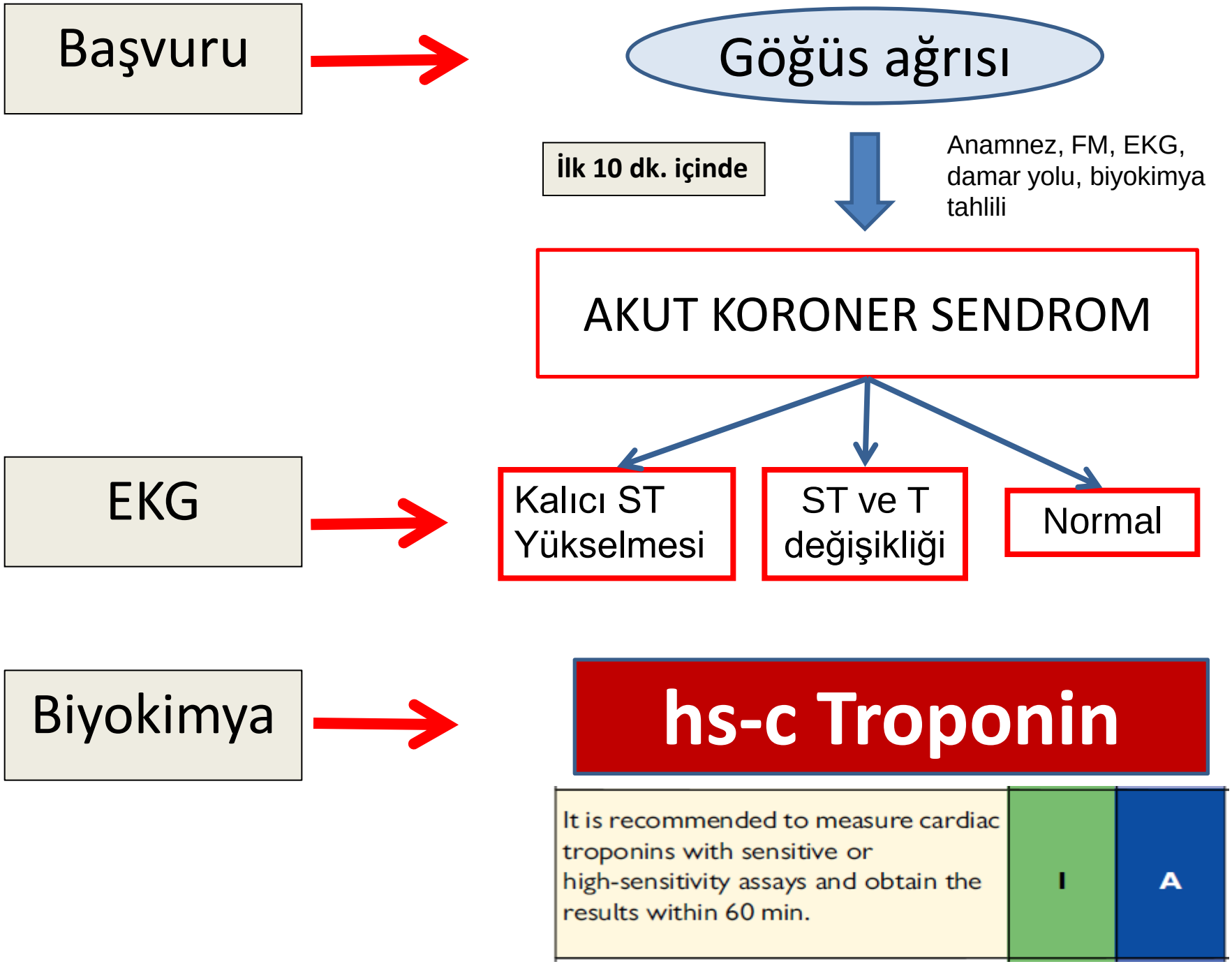

Başvuru
Göğüs ağrısı
İlk 10 dak. içinde
Anamnez, FM, EKG, damar yolu, kardiyak belirteçler
AKUT KORONER SENDROM
EKG
Kalıcı ST Yükselmesi
ST ve T değişikliği
Normal

EKG değişikliği & mortalite



Granger CB et al. J Am Coll Cardiol 31; 98 :71A

Recommendations	Class[a]	Level[b]
Diagnosis and risk stratification		
It is recommended to base diagnosis and initial short-term ischaemic and bleeding risk stratification on a combination of clinical history, symptoms, vital signs, other physical findings, ECG and laboratory results.	I	A
It is recommended to obtain a 12-lead ECG within 10 min after first medical contact and to have it immediately interpreted by an experienced physician. It is recommended to obtain an additional 12-lead ECG in case of recurrent symptoms or diagnostic uncertainty.	I	B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Göğüs ağrısı
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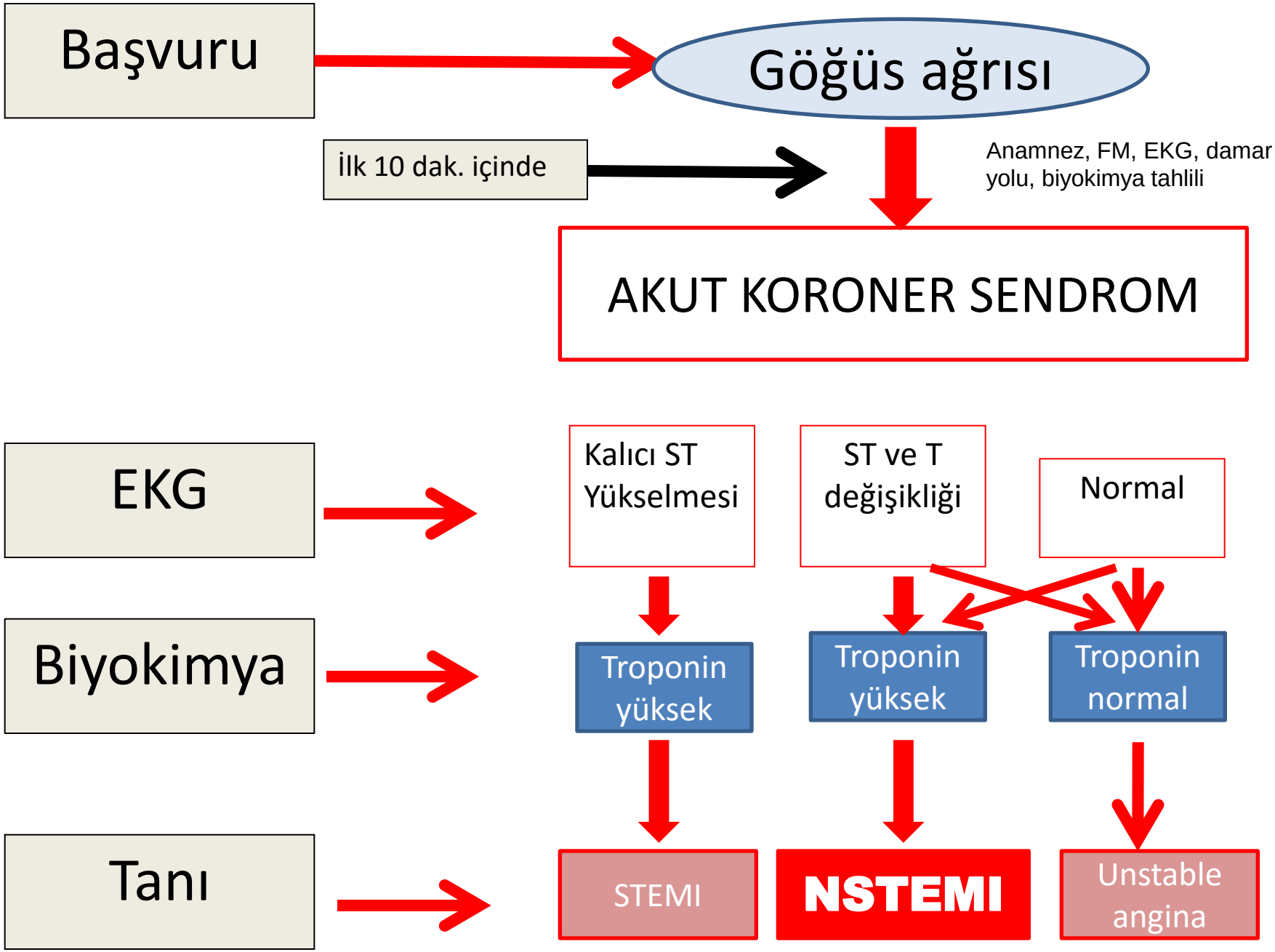
EKG
Kalıcı ST Yükselmesi
ST ve T değişikliği
Normal
Biyokimya
hs-c Troponin
It is recommended to measure cardiac troponins with sensitive or high-sensitivity assays and obtain the results within 60 min.
I
A

hsTn:

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).

Troponinin yükseldiği diğer durumlar

Tachyarrhythmias
Heart failure
Hypertensive emergencies
Critical illness (e.g. shock/ sepsis/ burns)
Myocarditis[a]
Tako-Tsubo cardiomyopathy
Structural heart disease (e.g. aortic stenosis)
Aortic dissection
Pulmonary embolism, pulmonary hypertension
Renal dysfunction and associated cardiac disease
Coronary spasm
Acute neurological event (e.g. stroke or subarachnoid haemorrhage)
Cardiac contusion or cardiac procedures (CABG, PCI, ablation, pacing, cardioversion, or endomyocardial biopsy)
Hypo- and hyperthyroidism
Infiltrative diseases (e.g. amyloidosis, haemochromatosis, sarcoidosis, scleroderma)
Myocardial drug toxicity or poisoning (e.g. doxorubicin, 5-fluorouracil, herceptin, snake venoms)
Extreme endurance efforts
Rhabdomyolysis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Tanı
Kalıcı ST Yükselmesi
ST ve T değişikliği
Normal
Troponin yüksek
Troponin yüksek
Troponin normal
STEMI
NSTEMI
Unstable angina

Ayırıcı tanı

Cardiac	Pulmonary	Vascular
Myopericarditis Cardiomyopathies[a]	**Pulmonary embolism**	**Aortic dissection**
Tachyarrhythmias	**(Tension)-Pneumothorax**	Symptomatic aortic aneurysm
Acute heart failure	Bronchitis, pneumonia	Stroke
Hypertensive emergencies	Pleuritis	
Aortic valve stenosis		
Tako-Tsubo cardiomyopathy		
Coronary spasm		
Cardiac trauma		

Gastro-intestinal	Orthopaedic	Other
Oesophagitis, reflus or spasm	**Musculoskeletal disorders**	**Anxiety disorders**
Peptic ulcer, gastritis	Chest trauma	Herpes zoster
Pancreatitis	Muscle injury/ inflammation	Anaemia
Cholecystitis	Costochondritis	
	Cervical spine pathologies	

Risk değerlendirmesi

It is recommended to use established risk scores for prognosis estimation.	I	B

Jneid et al. 2012 AHA Updata of the guidleine of NSTEMI. Circulation 2012;126:875-910/Eur Heart J 2011;32:2999-3054

Risk değerlendirmesi

- TIMI,

  (Thrombolysis In Myocardial Infarction)

  (Miyokart enfarktusunde tromboliz)

- **GRACE**

  (Global Registry of Acute Coronary Events)

  (Akut Koroner Olayların Global Kayıtları)

Eur Heart J 2011;32:2999-3054

http://www.timi.org/

TIMI Risk skorlaması

1. Yaş ≥ 65 yıl
2. Koroner arter hastalığı için ≥3 risk faktörü
3. Ciddi Koroner darlık
4. ST Segment değişimi
5. Ciddi anginal semptomlar
   (son 24 saat içinde ≥2 anginal olay)
6. Önceden aspirin kullanımı (son 7 gün içinde)
7. Artmış kardiyak markırlar

- Diyabet
- Sigara içimi
- Hipertansiyon
- Düşük HDL
- Aile hikayesi

- >3 Yüksek risk
- ≤2 Düşük risk

Antman EM. JAMA 2000;284:835-842

http://www.gracescore.org/WebSite/default.aspx?ReturnUrl=%2f

GRACE Risk 2.0 Hesaplaması

- Yaş,
- Kalp hızı,
- Sistolik kan basıncı,
- Serum kreatinin seviyesi,
- Killip sınıflaması,
- ST segment değişikliği,
- Yükselmiş kardiyak belirteçler,
- Kardiyak arrest

Hastane içi ve 6.ay mortaliteyi öngören risk skorlaması.

GRACE (0–258)	Age (years)	
	<40	0
	40–49	18
	50–59	36
	60–69	55
	70–79	73
	≥80	91
	Heart rate (bpm)	
	<70	0
	70–89	7
	90–109	13
	110–149	23
	150–199	36
	>200	46
	Systolic BP (mmHg)	
	<80	63
	80–99	58
	100–119	47
	120–139	37
	140–159	26
	160–199	11
	>200	0
	Creatinine (mg/dL)	
	0–0.39	2
	0.4–0.79	5
	0.8–1.19	8
	1.2–1.59	11
	1.6–1.99	14
	2–3.99	23
	>4	31
	Killip class	
	Class I	0
	Class II	21
	Class III	43
	Class IV	64
	Cardiac arrest at admission	43
	Elevated cardiac markers	15
	ST-segment deviation	30

✓mortality while
- ❑in hospital,
- ❑at 6 months,
- ❑at 1 year and
- ❑at 3 years.

✓The combined risk of death or MI at 1 year is also provided

Skor >140

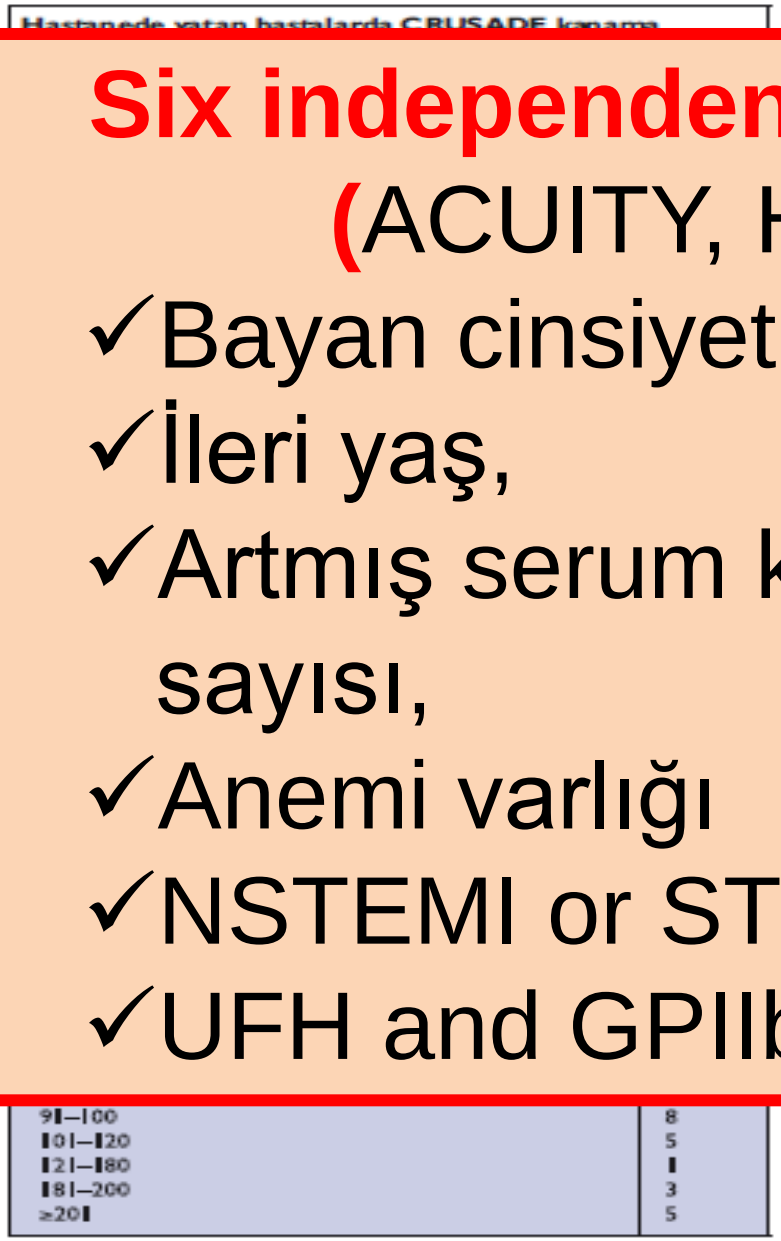
Hastane içi mortalite > %3

6 aylık mortalite > %8

Ritim Monitorizasyonu

Clinical Presentation	Unit	Rhythm monitoring
Unstable angina	Regular ward or discharge	None
NSTEMI at low risk for cardiac arrhythmias[a]	Intermediate care unit or coronary care unit	≤24 h
NSTEMI at intermediate to high risk for cardiac arrhythmias[b]	Intensive/coronary care units or intermediate care unit	>24 h

KANAMA RİSKİ (www.crusadebleedingscore.org/)



Six independent baseline predictors (ACUITY, HORIZONS-AMI)

- ✓Bayan cinsiyet,
- ✓İleri yaş,
- ✓Artmış serum kreatinin ve lökosit sayısı,
- ✓Anemi varlığı
- ✓NSTEMI or STEMI ile müracaat
- ✓UFH and GPIIb/IIIa inhibitör kullanımı

The use of the CRUSADE score may be considered in patients undergoing coronary angiography to quantify bleeding risk.	IIb	B

TEDAVİ-Medikal

- Miyokardial oksijen ihtiyacını azaltmak
  - HR, BP, preload ve kontraktilitenin azalması
- Miyokarda oksijen sunumunu artırmak
  - Koroner vazodilatasyon yada oksijen verilmesi(<%90)

AKUT KORONER SENDROMLARDA İLK TEDAVİ

ASA

O_2 sat <%90 ise: **oksijen**

KB>90 mmHg, İV veya SL: **nitrat**

Ağrı ciddi ise, 3-5 mgr **morfin** IV veya SC

Beta-bloker, tansiyon düşüklüğü ve blok yoksa

Jneid et al. 2012 AHA Updata of the guidleine of NSTEMI. Circulation 2012;126:875-910

Acute management STEMI:

- Select reperfusion strategy: Primary percutaneous coronary intervention (PCI) strongly preferred, especially for patients with cardiogenic shock, heart failure, late presentation, or contraindications to fibrinolysis. Activate cardiac catheterization team as indicated. For patients with symptoms of >12 hours, fibrinolytic therapy is not indicated, but emergent PCI may be considered, particularly for patients with evidence of ongoing ischemia or those at high risk of death.

- Treat with fibrinolysis if PCI unavailable within **120 minutes of first medical contact,** symptoms <12 hours, and no contraindications.*

- **Give oral antiplatelet therapy (in addition to aspirin) to all patients:**

1. **Patients treated with fibrinolytic therapy:** Give clopidogrel loading dose 300 mg if age 75 years or less; if age over 75 years, give loading dose of 75 mg.

2. **Patients treated with no reperfusion therapy:** Give ticagrelor loading dose 180 mg.

3. **Patients treated with primary percutaneous coronary intervention:** Give ticagrelor loading dose of 180 mg or prasugrel loading dose of 60 mg (if no contraindications: prior stroke or TIA, or relative contraindications for prasugrel such as those age 75 years or older, weight less than 60 kg). For patients at high risk of bleeding or those for whom prasugrel or ticagrelor cannot be used, we give clopidogrel 600 mg.

- **Give anticoagulant therapy to all patients:**

1. **For patients treated with primary PCI,** we prefer unfractionated heparin (UFH) to bivalirudin. This recommendation assumes that patients will receive a potent oral antiplatelet agent (ticagrelor or prasugrel), which we prefer to clopidogrel. For those patients who receive clopidogrel, we prefer bivalirudin.

   - **Dosing of UFH:** An initial intravenous (IV) bolus of 50 to 70 units/kg up to a maximum of 5000 units. Additional heparin may be given in the catheterization laboratory based on the results of activated clotting time (ACT) monitoring.
   - **Dosing of bivalirudin:** Initial bolus of 0.75 mg/kg IV followed by IV infusion of 1.75 mg/kg per hour; can be discontinued after PCI.

2. **For patients treated with fibrinolysis,** we prefer enoxaparin for patients not at high bleeding risk or fondaparinux for those at high bleeding risk. For those patients in whom PCI is possible or likely after fibrinolytic therapy, UFH is reasonable.

- **Dosing of enoxaparin**
  - Patients <75 years: Loading dose of 30 mg IV bolus followed by 1 mg/kg subcutaneously every 12 hours; maximum of 100 mg for the first two subcutaneous doses. The first subcutaneous dose should be administered with the IV bolus.
    - Dose adjustment for renal impairment (Crcl <30 mL/min)*: Loading dose of 30 mg IV followed by 1 mg/kg subcutaneously every 24 hours. The first subcutaneous dose should be administered with the IV bolus.
  - Patients ≥75 years: No IV loading dose. Administer 0.75 mg/kg subcutaneously every 12 hours; maximum of 75 mg for the first two doses.
    - Dose adjustment for renal impairment (Crcl <30 mL/min)*: No IV loading dose. Administer 1 mg/kg subcutaneously every 24 hours.
  - Supplemental IV bolus dose for patients who will receive PCI after >1 dose of therapeutic enoxaparin: 0.3 mg/kg if last enoxaparin dose was given 8 to 12 hours earlier; No supplemental IV dose if last enoxaparin dose was within 8 hours; Use UFH if last enoxaparin dose was more than 12 hours ago
- **Dosing of UFH:** IV bolus of 60 to 100 units/kg to a maximum of 4000 units, followed by an IV infusion of 12 units/kg per hour (maximum 1000 units per hour) adjusted to achieve a goal activated partial thromboplastin time (aPTT) of approximately 50 to 70 seconds (1.5 to 2 times control).
- **Dosing of fondaparinux:** 2.5 mg intravenously, followed by 2.5 mg subcutaneously every 24 hours. This drug should be avoided in creatinine clearance <30 mL/min.

3. **For patients not receiving reperfusion therapy,** we use enoxaparin or UFH.

- **Dosing of enoxaparin:** Dose same as for patients treated with fibrinolysis (refer to section 2 above)
- **Dosing of UFH:** IV bolus of 50 to 70 units/kg to a maximum of 5000 units, followed by an IV infusion of 12 units/kg per hour adjusted to achieve a goal aPTT of approximately 50 to 70 seconds (1.5 to 2 times control).

Acute management of unstable angina or non-STEMI:

- **Give antiplatelet therapy (in addition to aspirin) to all patients:**

1. **Patients not treated with an invasive approach:** Give ticagrelor loading dose 180 mg. For these patients who are at very high risk (eg, recurrent ischemic discomfort, dynamic ECG changes, or hemodynamic instability), consider adding a GP IIb/IIIa inhibitor (either eptifibatide or tirofiban).

2. **For patients managed with an invasive approach:** Give ticagrelor loading dose of 180 mg at presentation. Prasugrel loading dose of 60 mg may be used as an alternative if given after diagnostic coronary angiography.

For patients age 75 years or older, weight less than 60 kg, or past stroke or transient ischemic attack, ticagrelor or clopidogrel are preferred to prasugrel. Clopidogrel may be given in a dose of 300 to 600 mg, but we prefer 600 mg. For patients otherwise at high risk for bleeding due to prior hemorrhagic stroke, ongoing bleeding, bleeding diathesis, or clinically relevant anemia or thrombocytopenia, clopidogrel 300 to 600 mg is an option.

For patients treated with an invasive approach and who receive bivalirudin, we do not recommend routinely giving a GP IIb/IIIa inhibitor; for those patients treated with heparin and who are troponin positive, we suggest adding a GP IIb/IIIa inhibitor (either abciximab or eptifibatide) given after diagnostic angiography. For those undergoing an invasive approach who are at very high risk (eg, recurrent ischemic discomfort, dynamic ECG changes, or hemodynamic instability), we consider adding a GP IIb/IIIa inhibitor prior to diagnostic angiography (either eptifibatide or tirofiban) or after diagnostic angiography (abciximab or eptifibatide). See text for dosing.

- **Give anticoagulant therapy in all patients:**

1. **For patients undergoing urgent catheterization (within four hours) or those managed with an early invasive strategy (angiography within 4 to 48 hours),** we use either heparin or bivalirudin. We prefer initiation of heparin in the emergency department and a switch to bivalirudin in the catheterization laboratory.

- **Dosing of UFH:** IV bolus of 60 to 70 units/kg to a maximum of 5000 units, followed by an IV infusion of 12 units/kg per hour adjusted to achieve a goal aPTT of approximately 50 to 70 seconds (1.5 to 2 times control).
- **Dose of bivalirudin:** If bivalirudin is given in the emergency department, IV bolus of 0.1 mg/kg and an infusion of 0.25 mg/kg per hour before angiography. If PCI is performed, an additional 0.5 mg/kg bolus is given and the infusion rate is increased to 1.75 mg/kg per hour.

2. **For patients receiving a non-invasive approach,** we recommend either fondaparinux or enoxaparin.

- **Enoxaparin** is an alternative to UFH for patients not undergoing an early invasive approach. No loading dose is necessary. Dosing is 1 mg/kg subcutaneously every 12 hours. Dose adjustment for renal impairment (Crcl <30 mL/min)*: 1 mg/kg subcutaneously every 24 hours.
- **Fondaparinux:** 2.5 mg subcutaneously every 24 hours. This drug should be avoided in patients with a creatinine clearance <30 mL/min.

TEDAVİ-İnvaziv

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	An immediate invasive strategy (< 2 h)
High-risk criteria	
• Rise or fall in cardiac troponin compatible with MI • Dynamic ST- or T-wave changes (symptomatic or silent) • GRACE score >140	An early invasive strategy (<24 h)
Intermediate-risk criteria	
• Diabetes mellitus • Renal insufficiency (eGFR <60 mL/min/1.73 m²) • LVEF <40% or congestive heart failure • Early post-infarction angina • Prior PCI • Prior CABG • GRACE risk score >109 and <140	An invasive strategy (<72 h)
Low-risk criteria	
• Any characteristics not mentioned above	

Algorithm for management of patients with suspected acute myocardial infarction in the emergency department