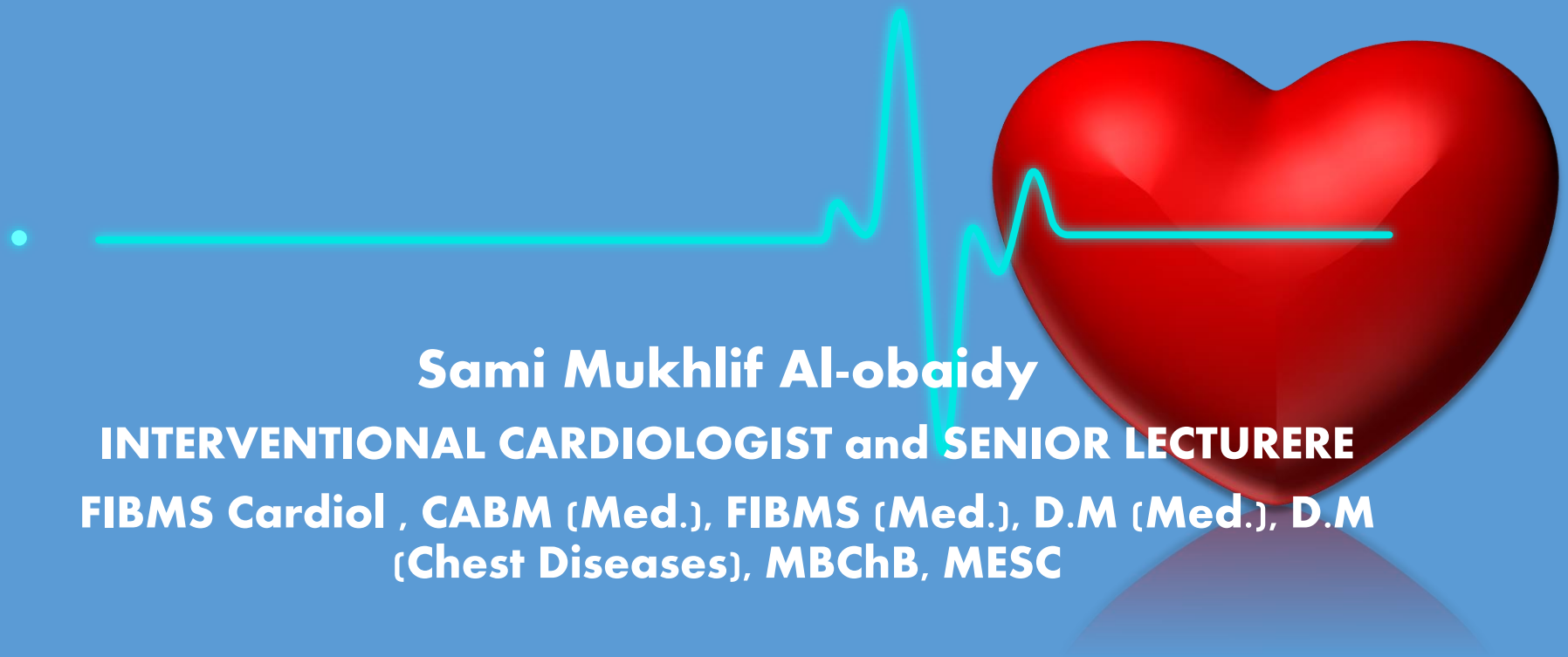
CORONARY ARTERY DISEASE
ACUTE CORONARY SYNDROMES



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Acute coronary syndrome is a term that encompasses both unstable angina and myocardial infarction.

Unstable angina is characterised by new-onset or rapidly worsening angina (crescendo angina), angina on minimal exertion or angina at rest in the absence of myocardial damage.

Myocardial infarction differs from unstable angina, since there is evidence of myocardial necrosis. The term acute myocardial infarction (MI) should be used when there is evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischaemia.

Acute coronary syndrome may present as a new phenomenon in patients with no previous history of heart disease or against a background of chronic stable angina.

The risk markers that are indicative of an adverse prognosis include recurrent ischaemia, extensive ECG changes at rest or during pain, raised troponin I or T levels, arrhythmias and haemodynamic complications (hypotension, mitral regurgitation) during episodes of ischaemia.

Careful assessment and risk stratification are important because these guide the use of more complex pharmacological and interventional treatments

Pathogenesis

Acute coronary syndrome almost always occurs in patients who have atherosclerosis.

The culprit lesion that precipitates the acute event is usually a complex ulcerated or fissured atheromatous plaque with adherent platelet-rich thrombus and local coronary artery spasm.

These vascular changes during an acute coronary syndrome are dynamic, such that the degree of obstruction may either increase, leading to complete vessel occlusion, or regress due to the effects of platelet disaggregation and endogenous fibrinolysis. In acute MI, occlusive thrombus is almost always present at the site of rupture or erosion of an atheromatous plaque.

The thrombus may undergo spontaneous lysis over the course of the next few days, although, by this time, irreversible myocardial damage has occurred. Without treatment, the artery responsible for the MI remains permanently occluded in 20–30% of patients.

Since the process of infarction progresses over several hours, most patients present when it is still possible to salvage myocardium and improve outcome.

Clinical features

The differential diagnosis of acute coronary syndrome is wide and includes most causes of central chest pain or collapse.

Chest pain at rest is the cardinal symptom but breathlessness, vomiting and collapse are also common features.

The pain occurs in the same sites as angina but is usually more severe and lasts longer; it is often described as a tightness, heaviness or constriction in the chest.

In acute MI the pain can be excruciating, and the patient's expression and pallor may vividly convey the seriousness of the situation.

Most patients are breathless and, in some, this is the only symptom. Painless or 'silent' MI may also occur and is particularly common in older patients or those with diabetes mellitus. If syncope occurs, it is usually caused by an arrhythmia or profound hypotension.

Vomiting and sinus bradycardia are often due to vagal stimulation and are particularly common in patients with inferior MI. Nausea and vomiting may also be caused or aggravated by opiates given for pain relief.

Sometimes infarction occurs in the absence of physical signs.

Sudden death, from ventricular fibrillation or asystole, may occur immediately and often within the first hour. If the patient survives this most critical stage, the liability to dangerous arrhythmias remains, but diminishes as each hour goes by.

It is vital that patients know not to delay calling for help if symptoms occur. Complications may occur in all forms of acute coronary syndrome but have become less frequent in the modern era of immediate or early coronary revascularisation

16.48 Criteria for diagnosis of a prior myocardial infarction

- Pathological Q waves with or without symptoms in the absence of non-ischaemic causes
- Imaging evidence of a region of loss of viable myocardium that is thinned and fails to contract, in the absence of a non-ischaemic cause
- Pathological findings of a prior myocardial infarction

Adapted from Thygesen K, Alpert JS, Jaffe AS et al. Third universal definition of myocardial infarction. Eur Heart J 2012; 33:2551–2267.

16.49 Clinical features of acute coronary syndromes

Symptoms

- Prolonged cardiac pain: chest, throat, arms, epigastrium or back
- Anxiety and fear of impending death
- Nausea and vomiting
- Breathlessness
- Collapse/syncope

Physical signs

Signs of sympathetic activation

- Pallor
- Sweating
- Tachycardia

Signs of vagal activation

- Vomiting
- Bradycardia

Signs of impaired myocardial function

- Hypotension, oliguria, cold peripheries
- Narrow pulse pressure
- Raised jugular venous pressure
- Third heart sound
- Quiet first heart sound
- Diffuse apical impulse
- Lung crepitations

Low-grade fever

Complications

- Mitral regurgitation
- Pericarditis

Complications:

- Arrhythmias
- Recurrent angina
- Acute heart failure
- Pericarditis
- Dressler’s syndrome
- Papillary muscle rupture
- Ventricular septum rupture
- Ventricular rupture
- Embolism
- Ventricular remodelling
- Ventricular aneurysm

Investigations:

Electrocardiogram:

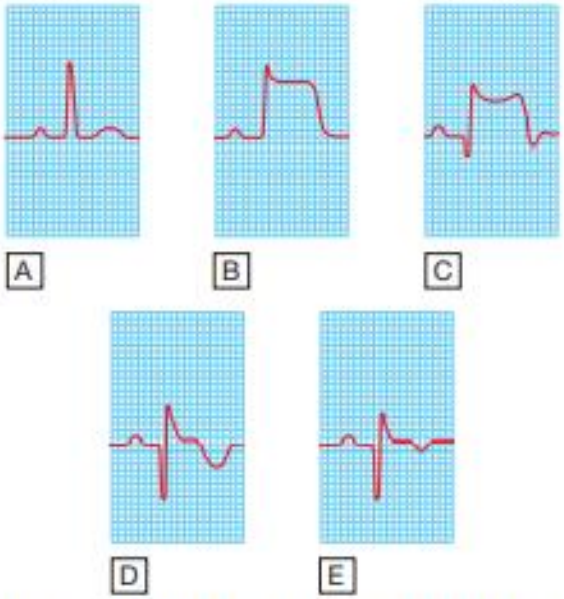


Fig. 16.65 The serial evolution of ECG changes in transmural myocardial infarction. A Normal ECG complex. B Acute ST elevation ('the current of injury'). C Progressive loss of the R wave, developing Q wave, resolution of the ST elevation and terminal T-wave inversion. D Deep Q wave and T-wave inversion. E Old or established infarct pattern; the Q wave tends to persist but the T-wave changes become less marked. The rate of evolution is very variable but, in general, stage B appears within minutes, stage C within hours, stage D within days and stage E after several weeks or months. This should be compared with the 12-lead ECGs in Figures 16.66–16.68.

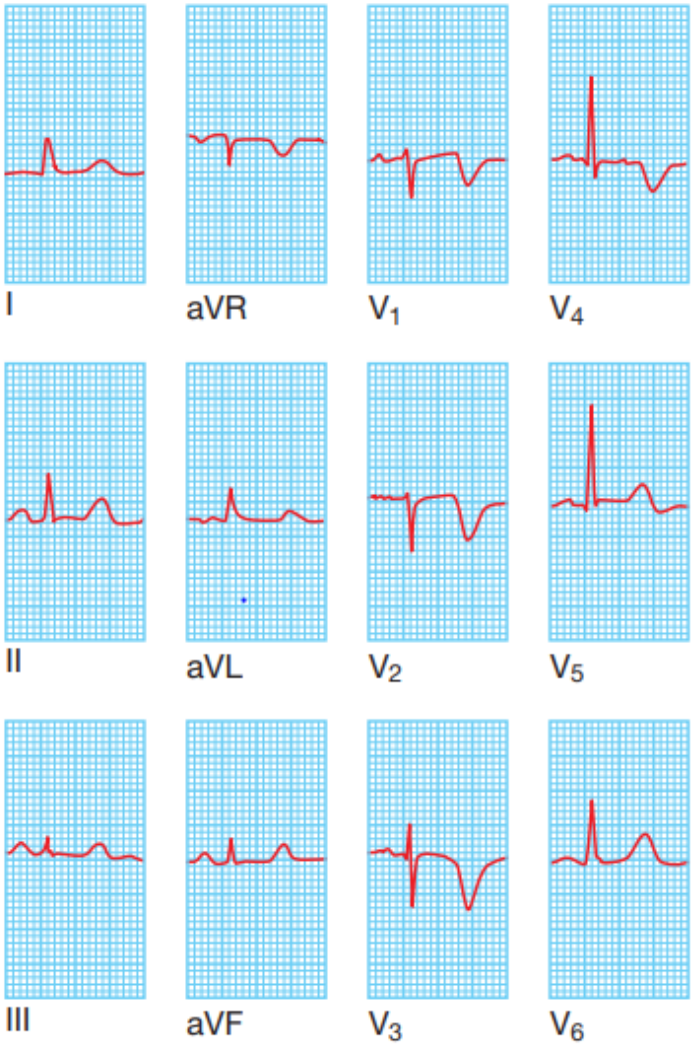


Fig. 16.66 Recent anterior non-ST elevation (subendocardial) myocardial infarction. This ECG demonstrates deep symmetrical T-wave inversion, together with a reduction in the height of the R wave in leads V_1, V_2, V_3 and V_4.

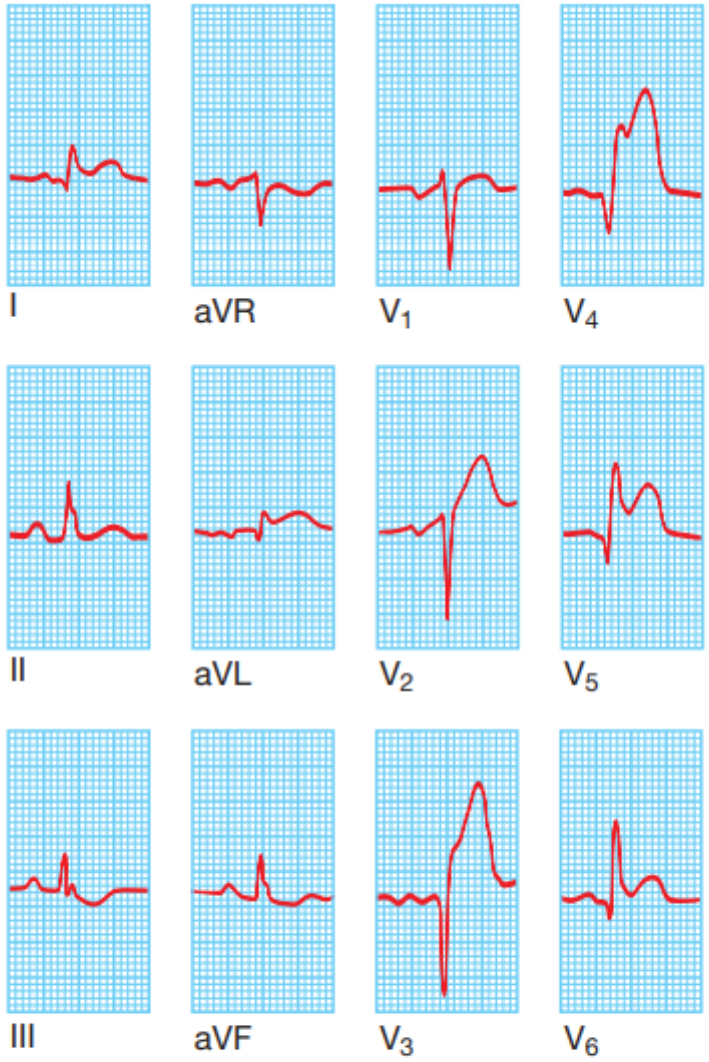


Fig. 16.67 Acute transmural anterior myocardial infarction. This ECG was recorded from a patient who had developed severe chest pain 6 hours earlier. There is ST elevation in leads I, aVL, V_2, V_3, V_4, V_5 and V_6, and there are Q waves in leads V_3, V_4 and V_5. Anterior infarcts with prominent changes in leads V_2, V_3 and V_4 are sometimes called 'anteroseptal' infarcts, as opposed to 'anterolateral' infarcts, in which the ECG changes are predominantly found in V_4, V_5 and V_6.

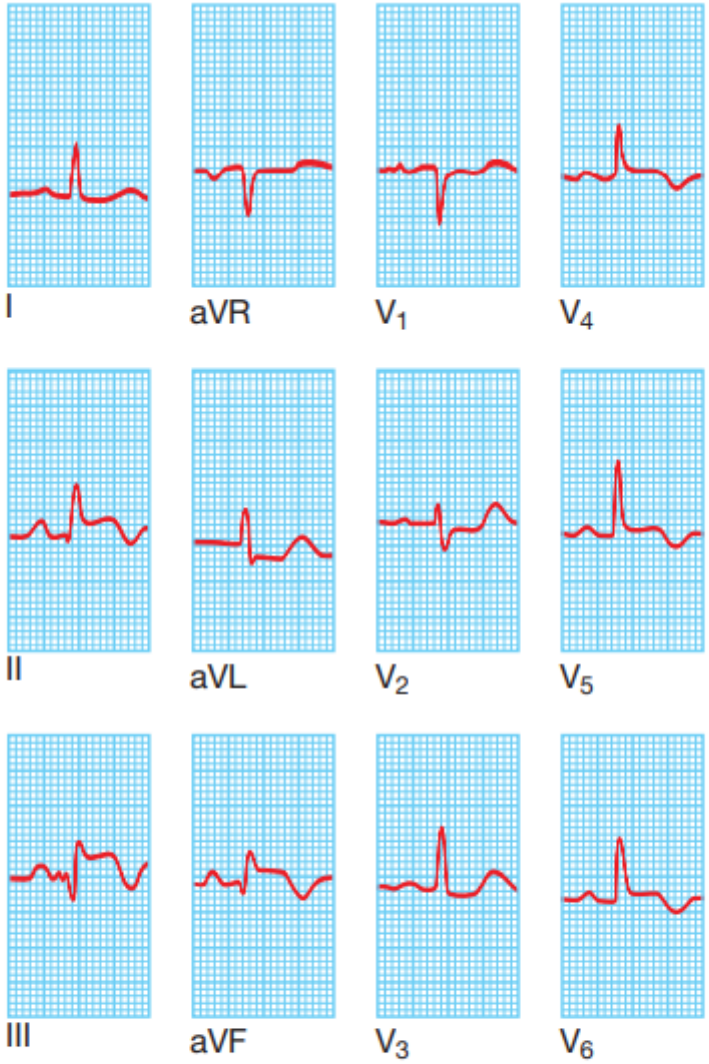


Fig. 16.68 Acute transmural inferolateral myocardial infarction. This ECG was recorded from a patient who had developed severe chest pain 4 hours earlier. There is ST elevation in inferior leads II, III and aVF and lateral leads V_4, V_5 and V_6. There is also 'reciprocal' ST depression in leads aVL and V_2.

IS THERE IS A TIMING FOR ECG CHANGES IN STEMI???

Cardiac Biomarkers:

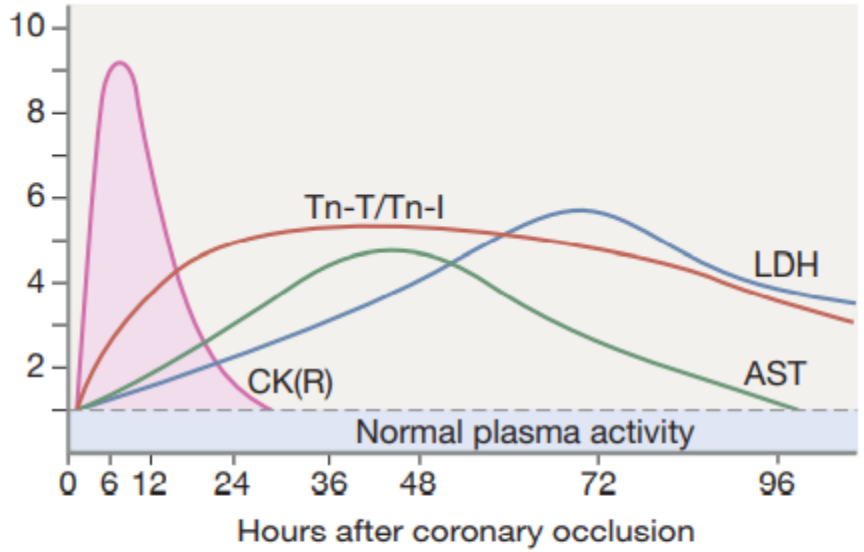


Fig. 16.69 Changes in plasma cardiac biomarker concentrations after myocardial infarction. Creatine kinase (CK) and troponins T (Tn-T) and I (Tn-I) are the first to rise, followed by aspartate aminotransferase (AST) and then lactate (hydroxybutyrate) dehydrogenase (LDH). In patients treated with repertusion therapy, a rapid rise in plasma creatine kinase (curve CK(R)) occurs, due to a washout effect.

WHAT ABOUT RECURRENT MI?

Echocardiography

RWMA

ventricular function

detecting important complications, such as mural thrombus, cardiac rupture, ventricular septal defect, mitral regurgitation and pericardial effusion

Coronary Angiography

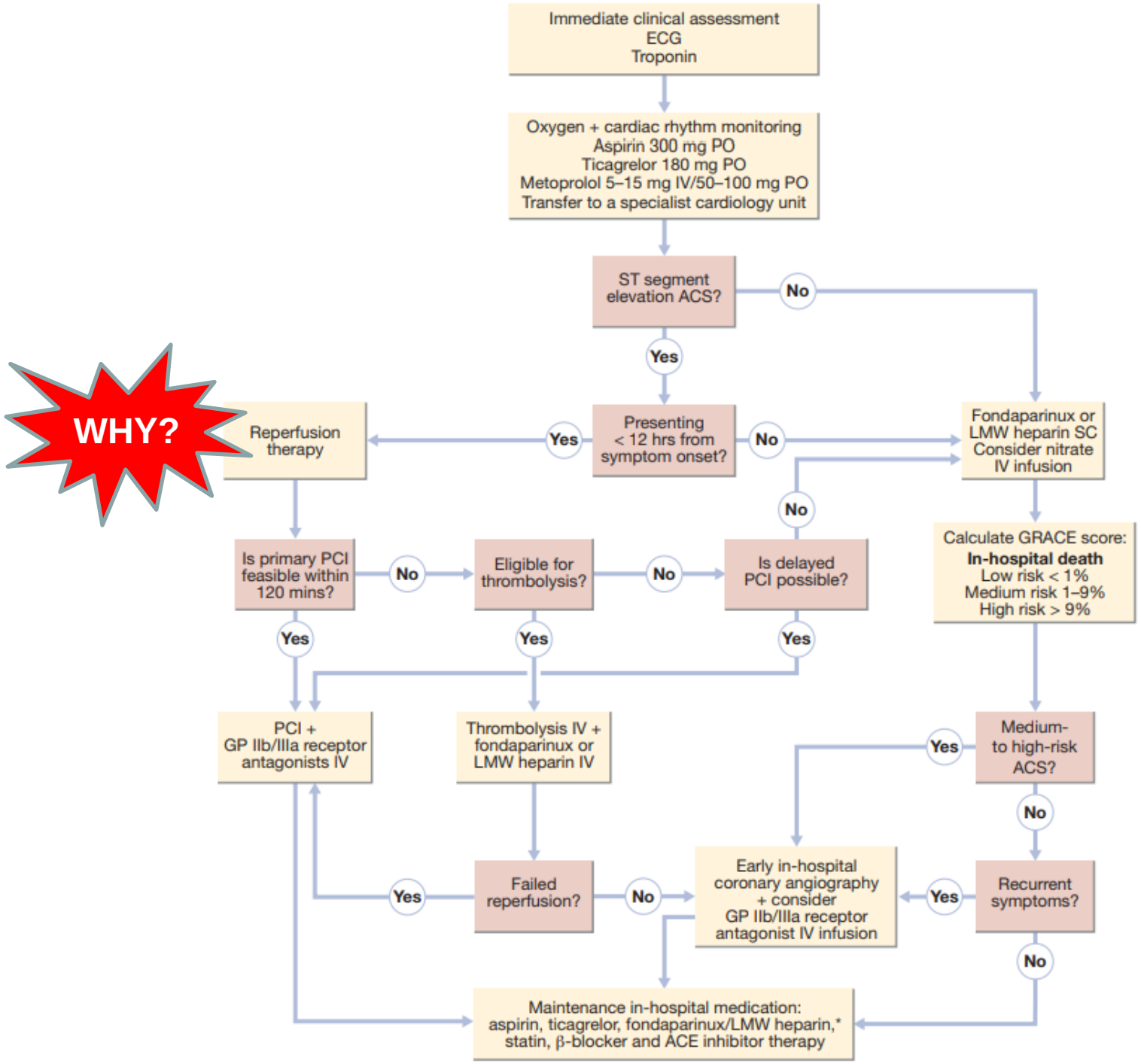
Treatment:

1-REVASCULARIZATION

2- MEDICAL THERAPY

ACCORDING TO TYPE OF ACS

STEMI VS NSTEMI VS UA


Immediate clinical assessment
ECG
Troponin
Oxygen + cardiac rhythm monitoring
Aspirin 300 mg PO
Ticagrelor 180 mg PO
Metoprolol 5–15 mg IV/50–100 mg PO
Transfer to a specialist cardiology unit
ST segment elevation ACS?
No
Yes
WHY?
Reperfusion therapy
Yes
Presenting < 12 hrs from symptom onset?
No
Fondaparinux or LMW heparin SC
Consider nitrate IV infusion
No
Is primary PCI feasible within 120 mins?
No
Eligible for thrombolysis?
No
Is delayed PCI possible?
Calculate GRACE score:
In-hospital death
Low risk < 1%
Medium risk 1–9%
High risk > 9%
Yes
Yes
Yes
PCI + GP IIb/IIIa receptor antagonists IV
Thrombolysis IV + fondaparinux or LMW heparin IV
Yes
Medium- to high-risk ACS?
No
Yes
Failed reperfusion?
No
Early in-hospital coronary angiography + consider GP IIb/IIIa receptor antagonist IV infusion
Yes
Recurrent symptoms?
No
Maintenance in-hospital medication:
aspirin, ticagrelor, fondaparinux/LMW heparin,*
statin, β-blocker and ACE inhibitor therapy

16.51 Late management of myocardial infarction

Risk stratification and further investigation (see text)

Lifestyle modification

- Diet (weight control, lipid-lowering, 'Mediterranean diet')
- Cessation of smoking
- Regular exercise

Secondary prevention drug therapy

- Antiplatelet therapy (aspirin and/or clopidogrel)
- β-blocker
- ACE inhibitor/ARB
- Statin
- Additional therapy for control of diabetes and hypertension
- Mineralocorticoid receptor antagonist

Rehabilitation

Devices

- Implantable cardiac defibrillator (high-risk patients)

(ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker)