Chest pain and Acute Coronary Syndrome

Emergency Medicine lectures

The differential diagnosis of central chest pain other than Ischemic chest pain

Types of Chest Pain

- Musculo-skeletal
- Pleuritic
- Oesophageal
- Pericarditis
- Myocarditis
- Aortic dissection

Cardiac–type chest pain

Typical description of cardiac type chest pain

Location	Duration	Character
• Central • Radiation • Visceral type	• >15 minutes • < 24 hours	• Not sharp • Not stabbing • Ache • Burning • Pressure • Not movement or breathing related

ACS vs aortic dissection (severe HTN / marfan)
(why is it important to differentiate ? because we treat ACS with blood thinners and these if given to an aortic dissection patient might kill him)

1. how acute the onset is
2. duration and severity (ASC > progressive severity + lasts for >=30 mins / aortic dissection > peaks in severity in the 1st minute then the severity starts declining until it stabilizes)
3. PEx : aortic dissection > radio-femoral delay

Not required for the mini-osce

Heart Score for major cardiac event

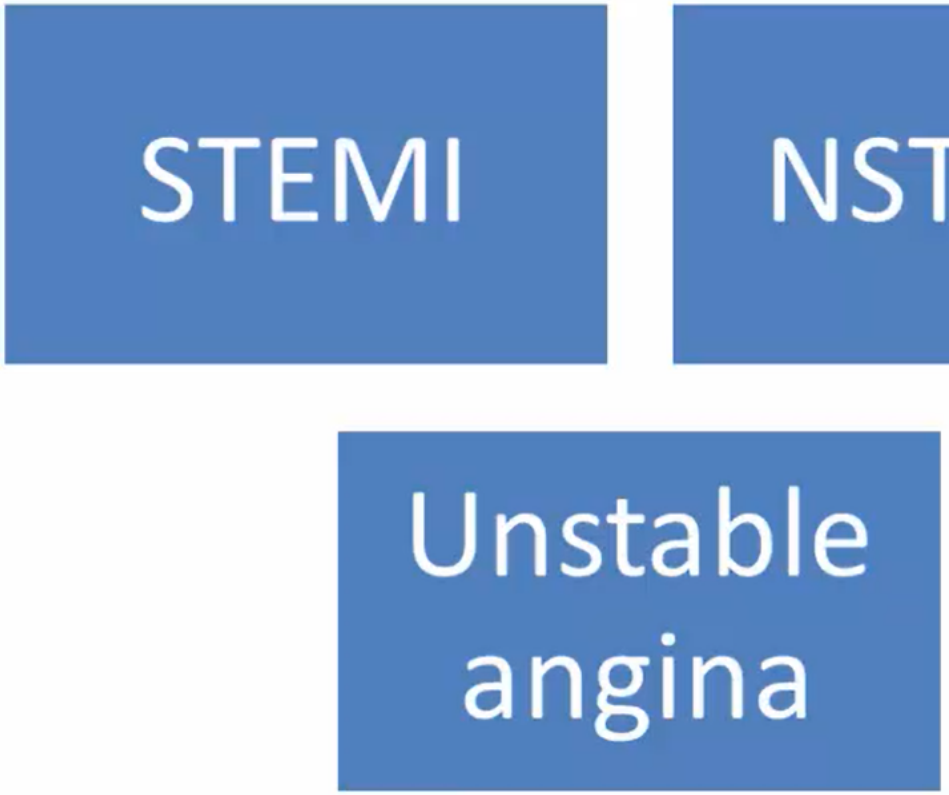
The HEART Score for Chest Pain Patients in the ED		
History	• Highly Suspicious • Moderately Suspicious • Slightly or Non-Suspicious	• 2 points • 1 point • 0 points
ECG	• Significant ST-Depression • Nonspecific Repolarization • Normal	• 2 points • 1 point • 0 points
Age	• ≥ 65 years • > 45 - < 65 years • ≤ 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		

Note:

The HEART score is a scoring system for patients presenting with chest pain at the emergency department.

With the HEART score it is immediately clear which patient is eligible for discharge without additional tests or emergency invasive procedures should be done .

Acute Coronary Syndromes

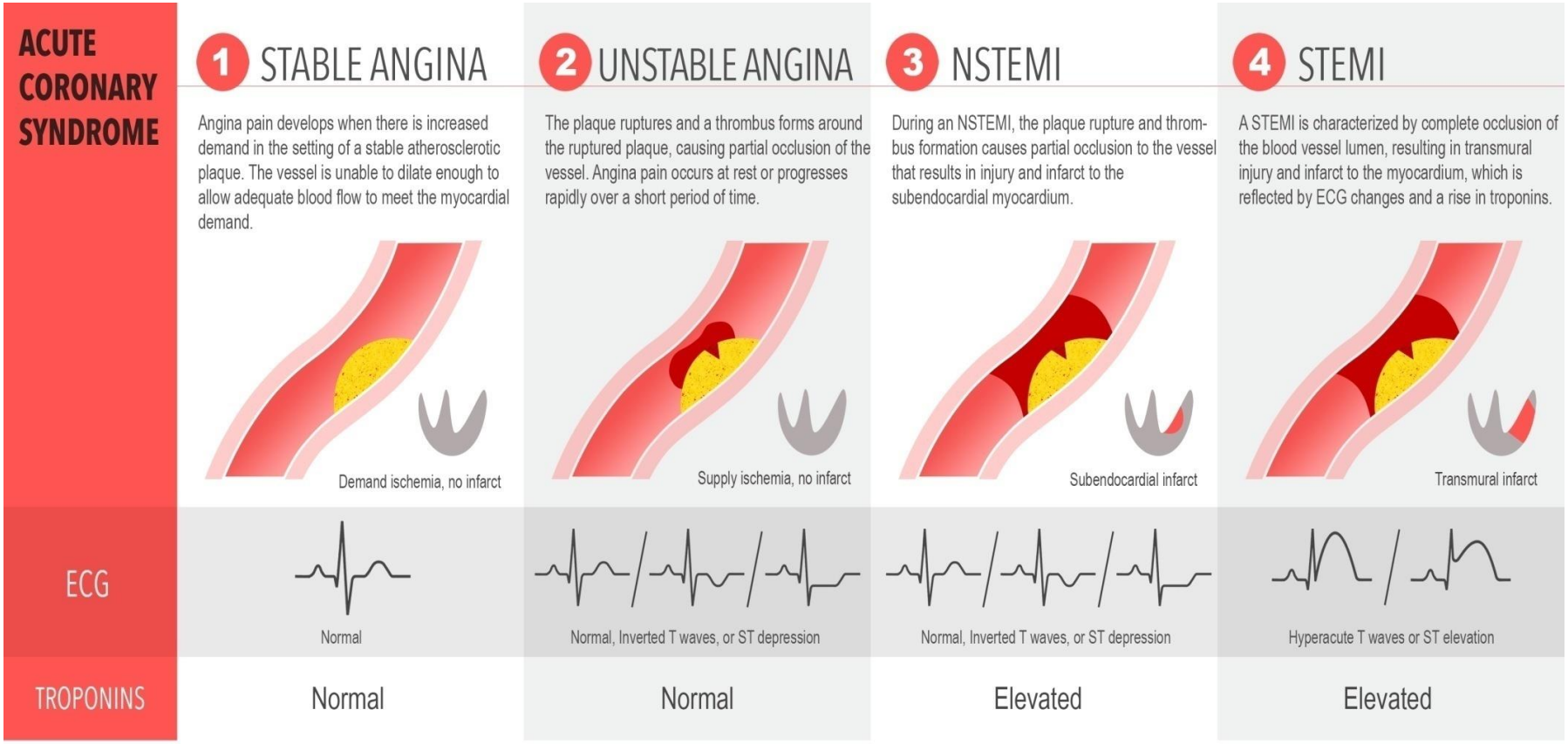


Acute coronary syndrome consists of : Unstable angina , NSTEMI and STEMI . It is part of Ischemic heart diseases that if left untreated it will lead to acute cardiac event and death.

Next step in management depends on history mainly (not ECG findings / not labs)

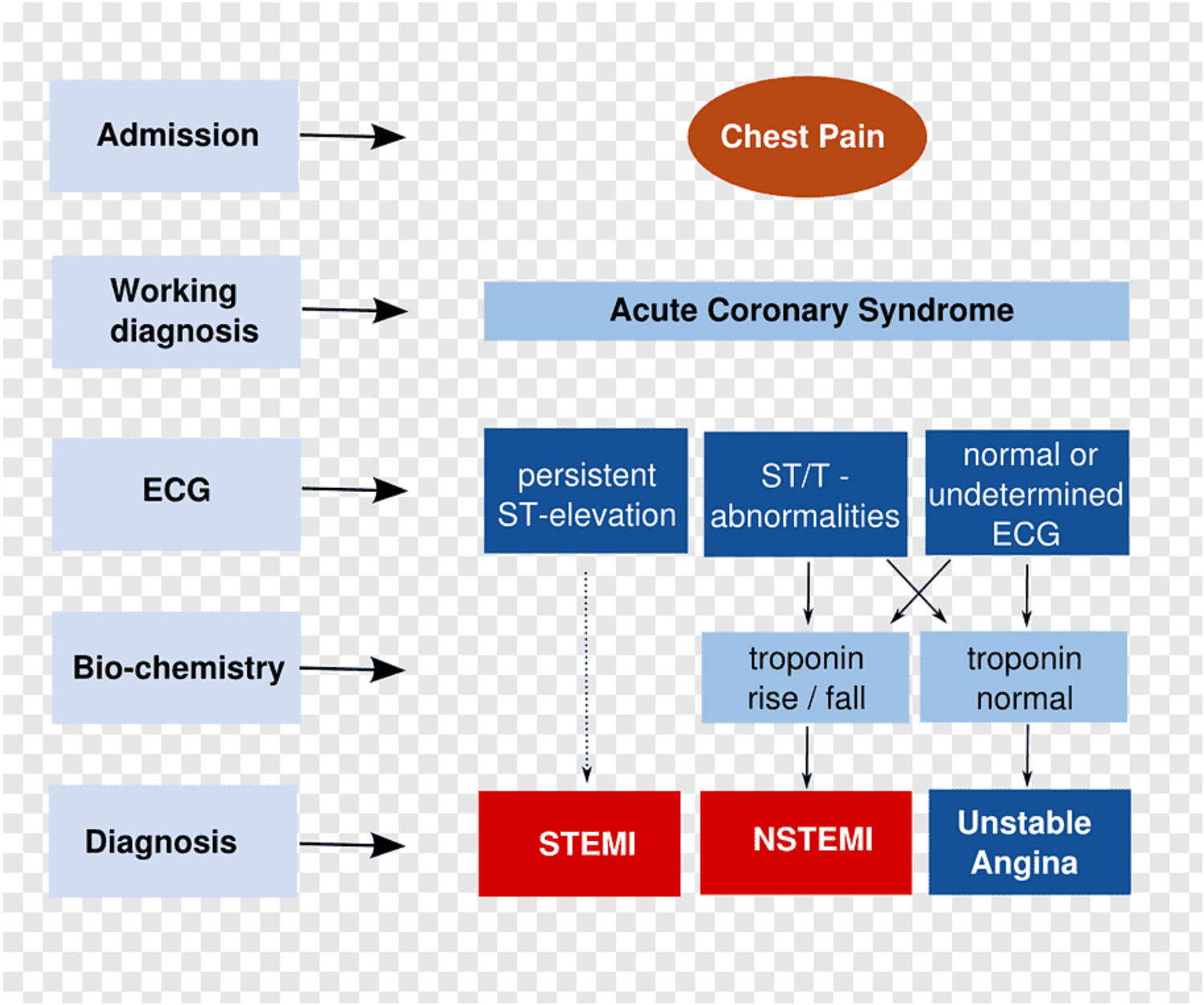
1. Increased frequency /duration
2. Increased severity (Not relieved by nitrates / at rest rather than exertional)
3. New onset angina (1 month)
4. Post PCI-managed MI angina (6 weeks)

ACS



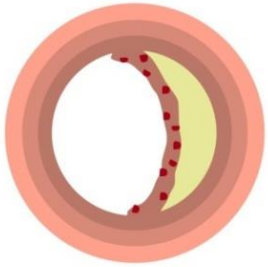
Acute coronary syndrome consists of : Unstable angina , NSTEMI and STEMI

Stable angina is part from Ischemic heart disease that is characterized by trivial central chest pain that last between 15-20 minutes , increased with exertion and relieved by rest or sublingual nitrates


Admission
Chest Pain
Working diagnosis
Acute Coronary Syndrome
ECG
persistent ST-elevation
ST/T - abnormalities
normal or undetermined ECG
Bio-chemistry
troponin rise / fall
troponin normal
Diagnosis
STEMI
NSTEMI
Unstable Angina

UNSTABLE ANGINA

CORE IM

PATHOPHYSIOLOGY	THROMBUS	CHEST PAIN SYMPTOMS
Ruptured plaque with non-occlusive thrombus* **Occlusive thrombus would typically cause a full STEMI.* 	• White • Platelet-rich	• **Acute** chest pain • With activity **and** rest
Progressive mechanical obstruction 	• Red • Fibrin-rich *Same pathophysiology as stable angina.*	• "Crescendoing angina" *Chest pain worsens over days to weeks.* • Should not occur at rest

REMINDER UNSTABLE ANGINA = TROPONIN NEGATIVE
NSTEMI = TROPONIN POSITIVE

The two share the same pathophysiology and symptomatology – the difference is in the cardiac biomarkers!

Pre/In-hospital management of suspected ACS

Give the patient MONA

M: Morphin (pain management)

O : oxygen according to BTS protocol

N : Nitroglycerin for pain management

A : Anti-platelets (Aspirin)

Anti-thrombin

M:
Has a vasodialtory effect along with its analgesic effect
Given up to 5 ml incrementally (not as a push)

** It induces vomiting in some people so we give anti-emetics
(metoclopramide / ondansetron (preferred in people under 20 as the risk of Metoclopramide-induced extrapyramidal effects is increased in people under 20 years of age))

O:
In trauma cases we always give oxygen
HOWEVER
in ACS the decision is based on O2 Sat
if <94% in normal people
if <88% in COPD patients
if <92% in asthma patients

N:
Not given when systolic BP is <90

A:
Aspirin: there's no IV aspirin
in conscious patients > oral pills
in unconscious patients > NG tube

If we suspect ACS

Do not routinely administer oxygen, but monitor oxygen saturation using pulse oximetry as soon as possible, ideally before hospital admission. Only offer supplemental oxygen to:

- people with oxygen saturation (SpO_2) of less than 94% who are not at risk of hypercapnic respiratory failure, aiming for SpO_2 of 94–98%
- people with chronic obstructive pulmonary disease who are at risk of hypercapnic respiratory failure, to achieve a target SpO_2 of 88–92% until blood gas analysis is available.

1.2.4 Assessment in hospital for people with a suspected acute coronary syndrome

1.2.4.1 Take a resting 12-lead ECG and a blood sample for troponin I or T measurement (see section 1.2.5) on arrival in hospital.

1.2.4.2 Carry out a physical examination to determine:

- haemodynamic status
- signs of complications, for example pulmonary oedema, cardiogenic shock **and**
- signs of non-coronary causes of acute chest pain, such as aortic dissection.

1.2.4.3 Take a detailed clinical history unless a STEMI is confirmed from the resting 12-lead ECG (that is, regional ST-segment elevation or presumed new LBBB). Record:

- the characteristics of the pain
- other associated symptoms
- any history of cardiovascular disease
- any cardiovascular risk factors **and**
- details of previous investigations or treatments for similar symptoms of chest pain.

1.2.5 Use of biochemical markers for diagnosis of an acute coronary syndrome

1.2.5.1 Take a blood sample for troponin I or T measurement on initial assessment in hospital. These are the preferred biochemical markers to diagnose acute MI.

1.2.5.2 Take a second blood sample for troponin I or T measurement 10–12 hours after the onset of symptoms.

** Q wave changes mean that it has been >=12 hrs since the onset of the event

** As we said we try to perform the cath within 12 hrs but if ST elevation persists beyond that or chest pain persists beyond that or ST changes do not improve beyond 50% following thrombolysis we do a revision cath

1.2.6 Making a diagnosis

1.2.6.1 When diagnosing MI, use the universal definition of myocardial infarction[2]. This is the detection of rise and/or fall of cardiac biomarkers (preferably troponin) with at least one value above the 99th percentile of the upper reference limit, together with evidence of myocardial ischaemia with at least one of the following:

- symptoms of ischaemia
- ECG changes indicative of new ischaemia (new ST-T changes or new LBBB)
- development of pathological Q wave changes in the ECG
- imaging evidence of new loss of viable myocardium or new regional wall motion abnormality[3].

Anti-platelet and antithrombin therapy

Antiplatelet

- Aspirin 300mg (unless allergic)
- Clopidogrel 300mg (unless very low risk)

Antithrombin

- Fondaparinux 2.5 mg sc
- Unfractionated heparin if PCI within 24 hours
- Reduce dose if significant bleeding risk
- Monitor clotting to guide dose if significant renal impairment (creatinine > 265 µmol/l)

** From door to ECG 10 mins
** From door to thrombolysis 60 mins
** From door to PCI 90 mins
AND if we can reach a PCI before 120 mins we always choose it over thrombolysis

STEMI management

If < 12 hours:

Aim for reperfusion as quickly as possible

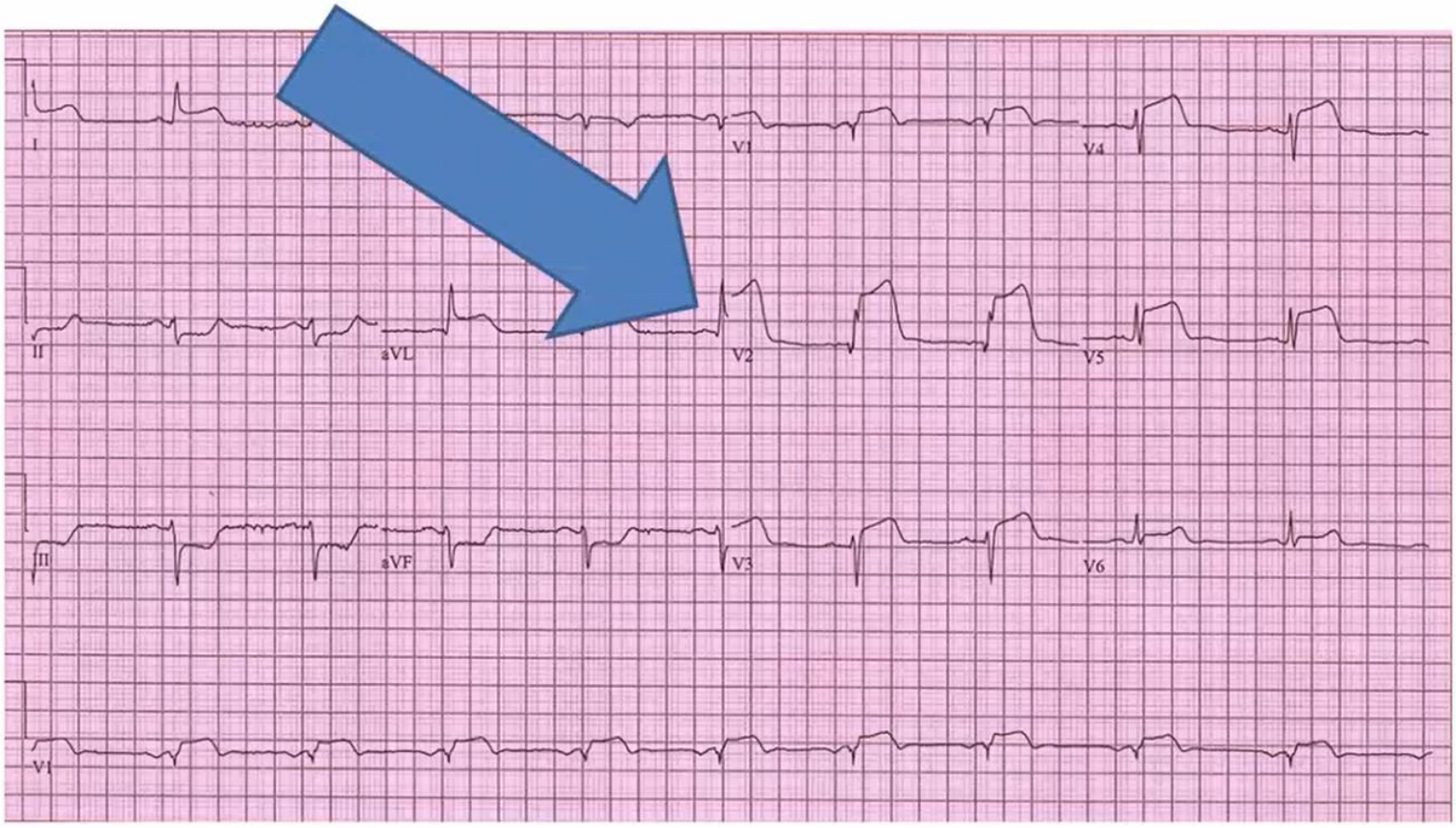
Primary PCI if possible

Use fibrinolysis if Primary PCI not within 2 hours of possible fibrinolysis time

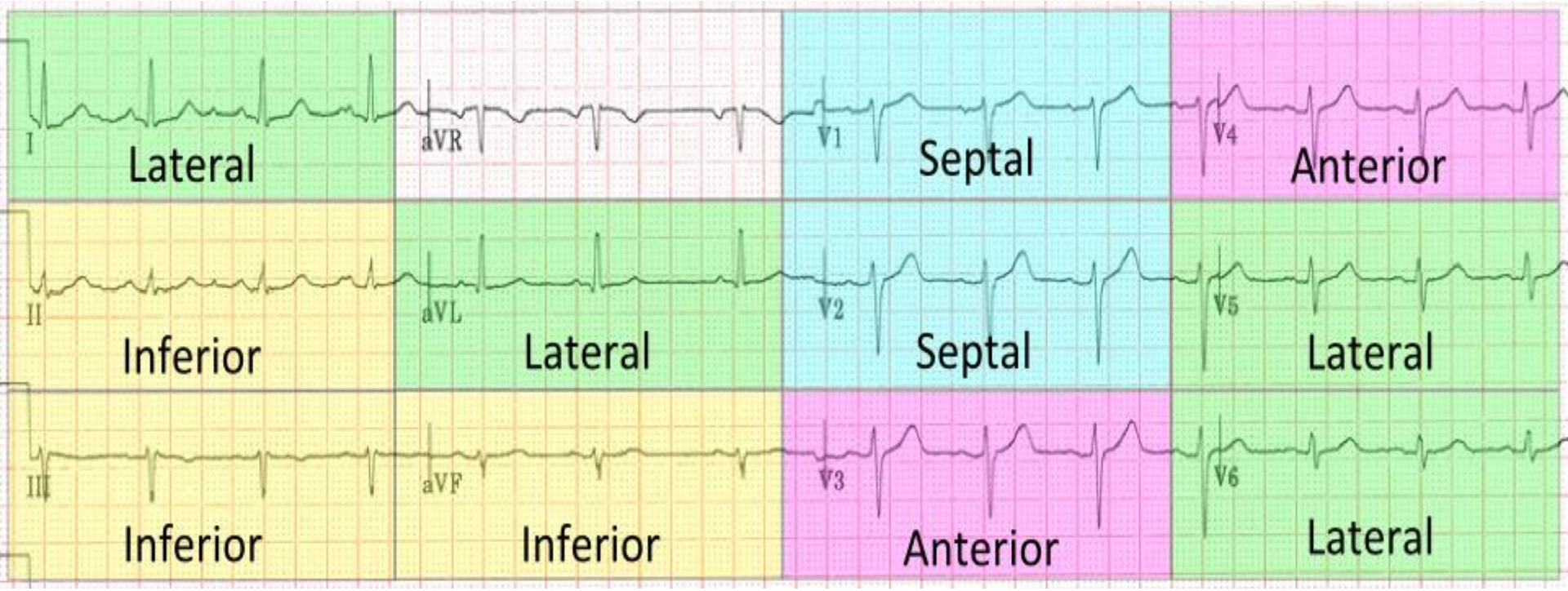
Give antithrombin with thrombolysis

In limb leads >> elevation of 1 small box or more is significant
In chest leads >> elevation of 2 small boxes or more is significant

STEMI ?

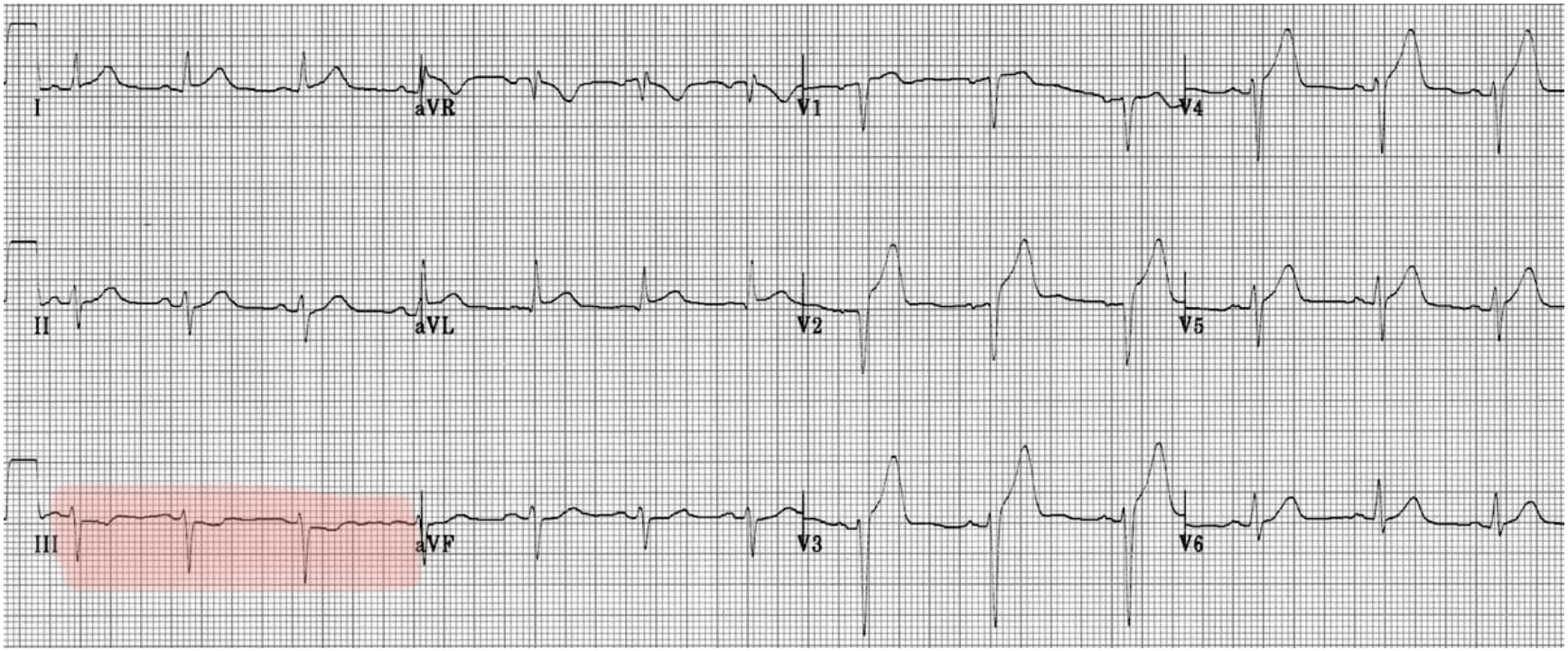


Distribution of leads



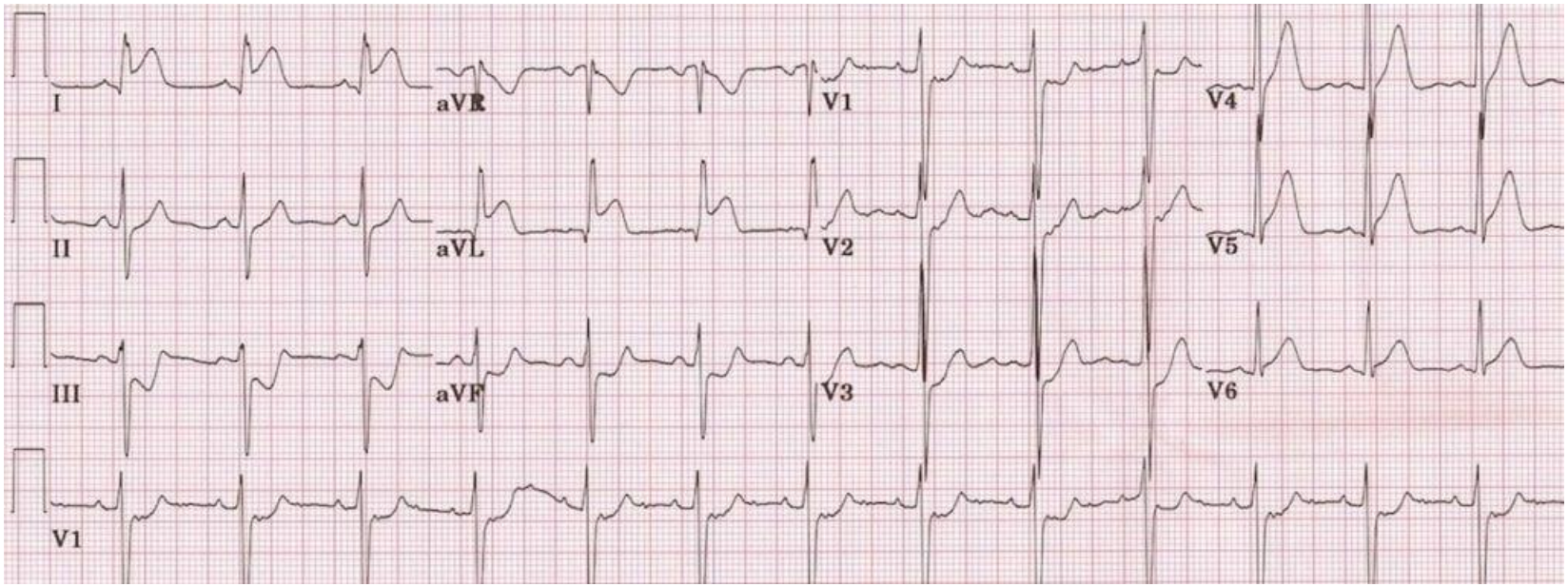
Anterior STEMI

** Here the lateral leads are in a borderline state so we repeat the ecg after 5 mins



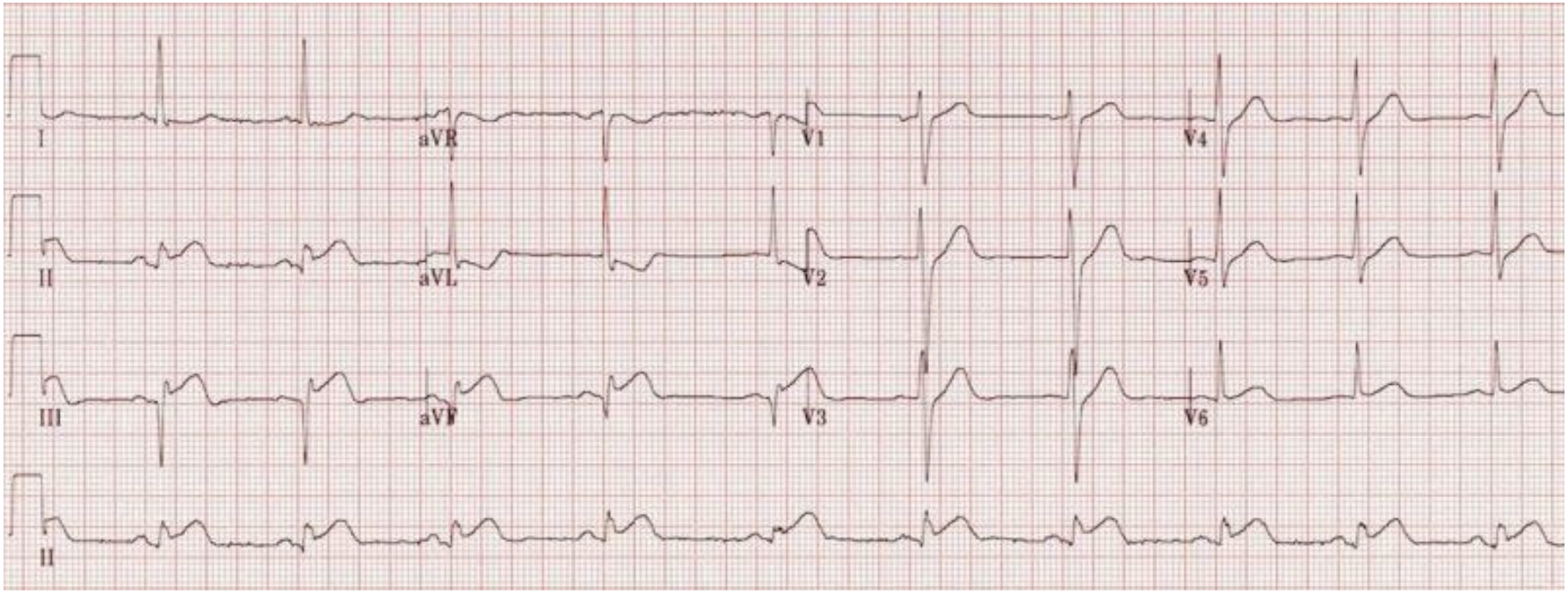
ST depression
Reciprocal changes (further confirm the presence of an MI)

Lateral MI



Inferior MI

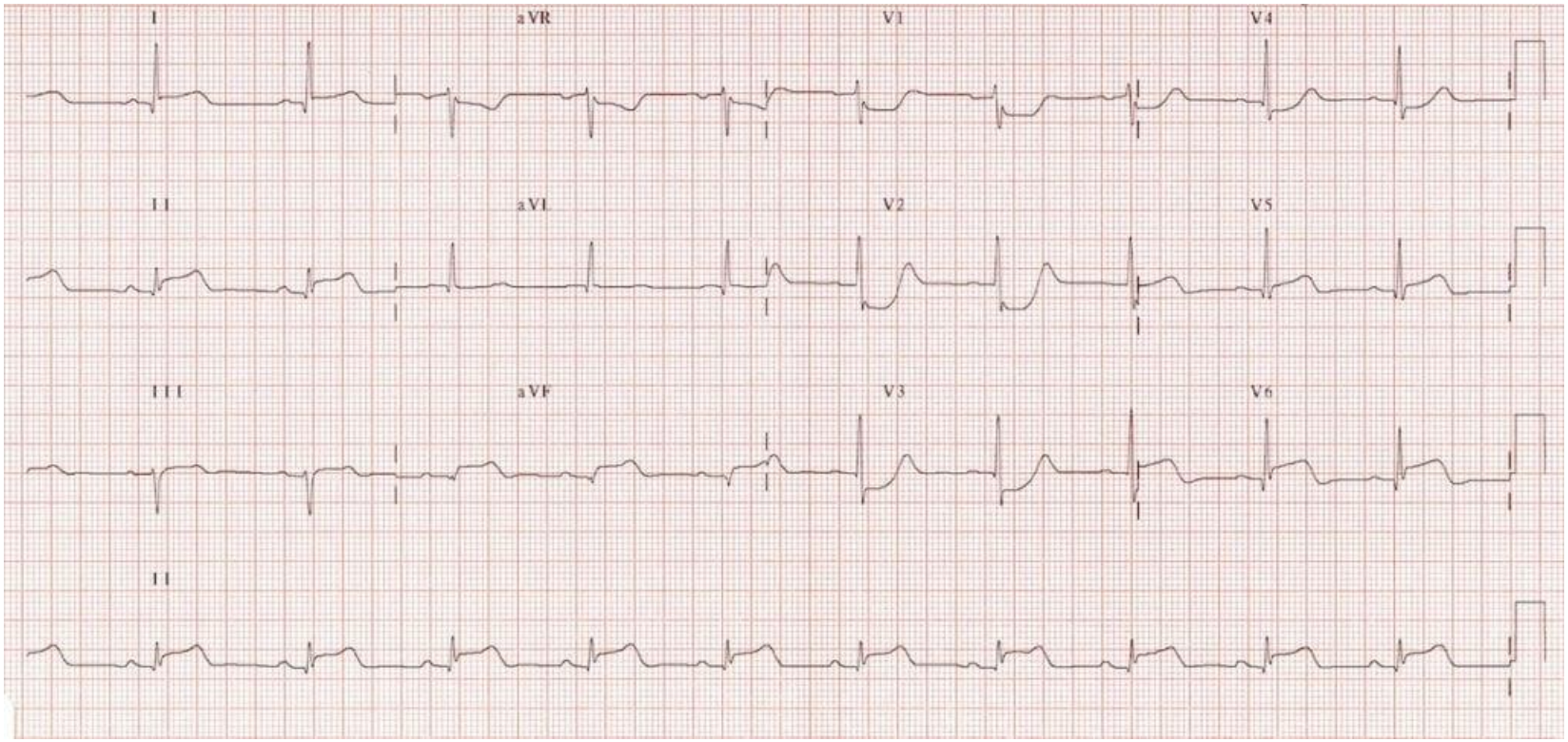
When we have an inferior MI we need to have high suspicion for posterior and RV MI



Pathological Q wave
1. Either wider than 1 small square or taller than 2 small squares
2. Present either in the inferior leads or the V1,2,3 leads

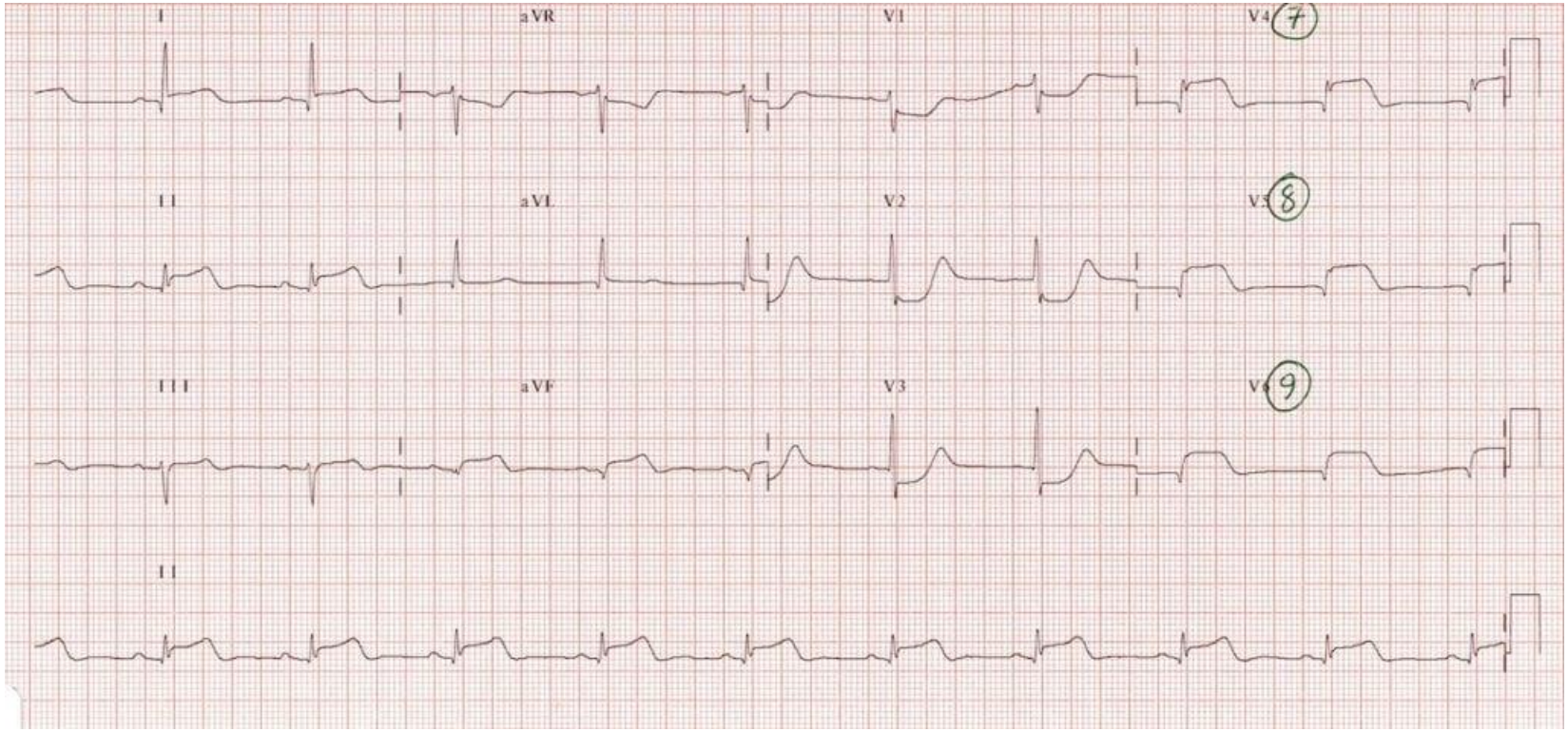
Not necessarily accompanied by an inferior MI

Posterior MI



Inferolateral STEMI. Posterior extension is suggested by:
Horizontal ST depression in V1-3
Tall, broad R waves (> 30ms) in V2-3
Dominant R wave (R/S ratio > 1) in V2
Upright T waves in V2-3

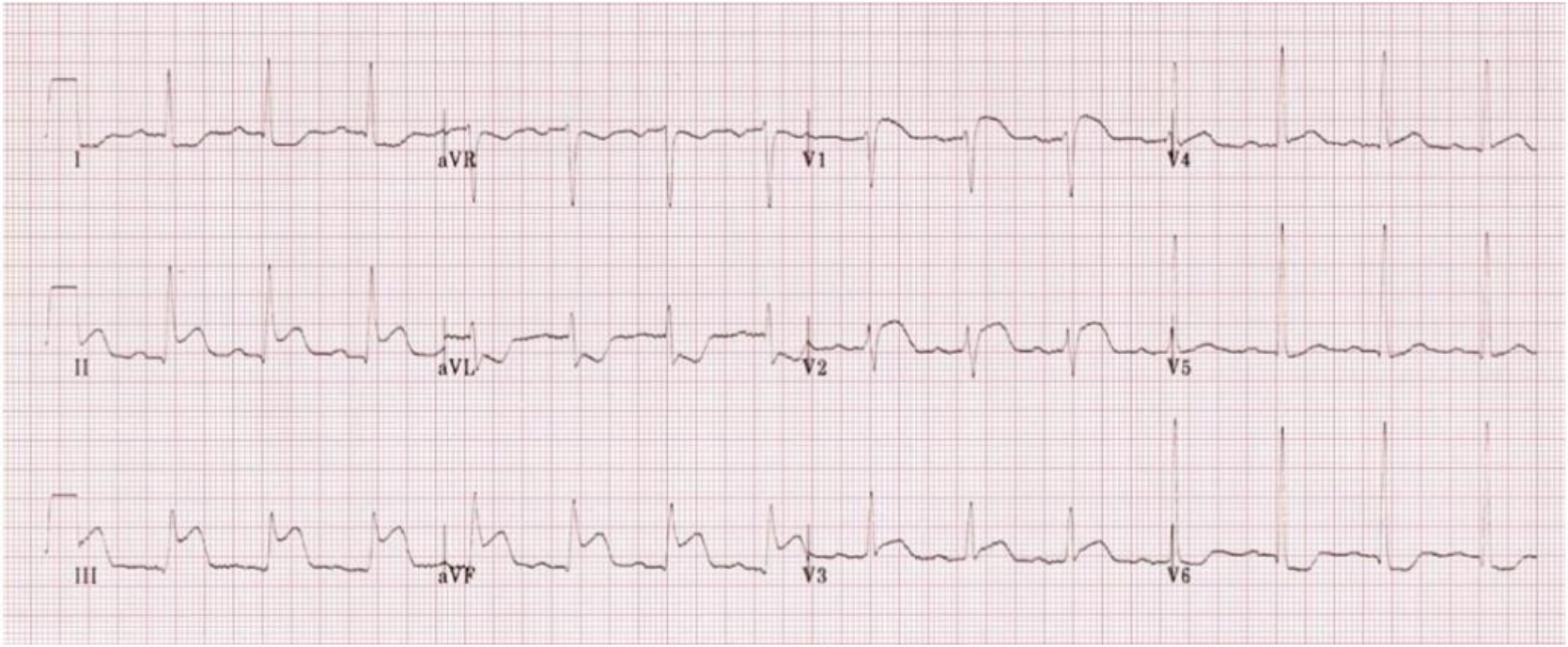
Posterior MI using posterior leads



Marked ST elevation in V7-9 with Q-wave formation confirms involvement of the posterior wall, making this an inferior-lateral-posterior STEMI (= big territory infarct!).

RV Wall MI

Example 1a



Inferior STEMI. Right ventricular infarction is suggested by:

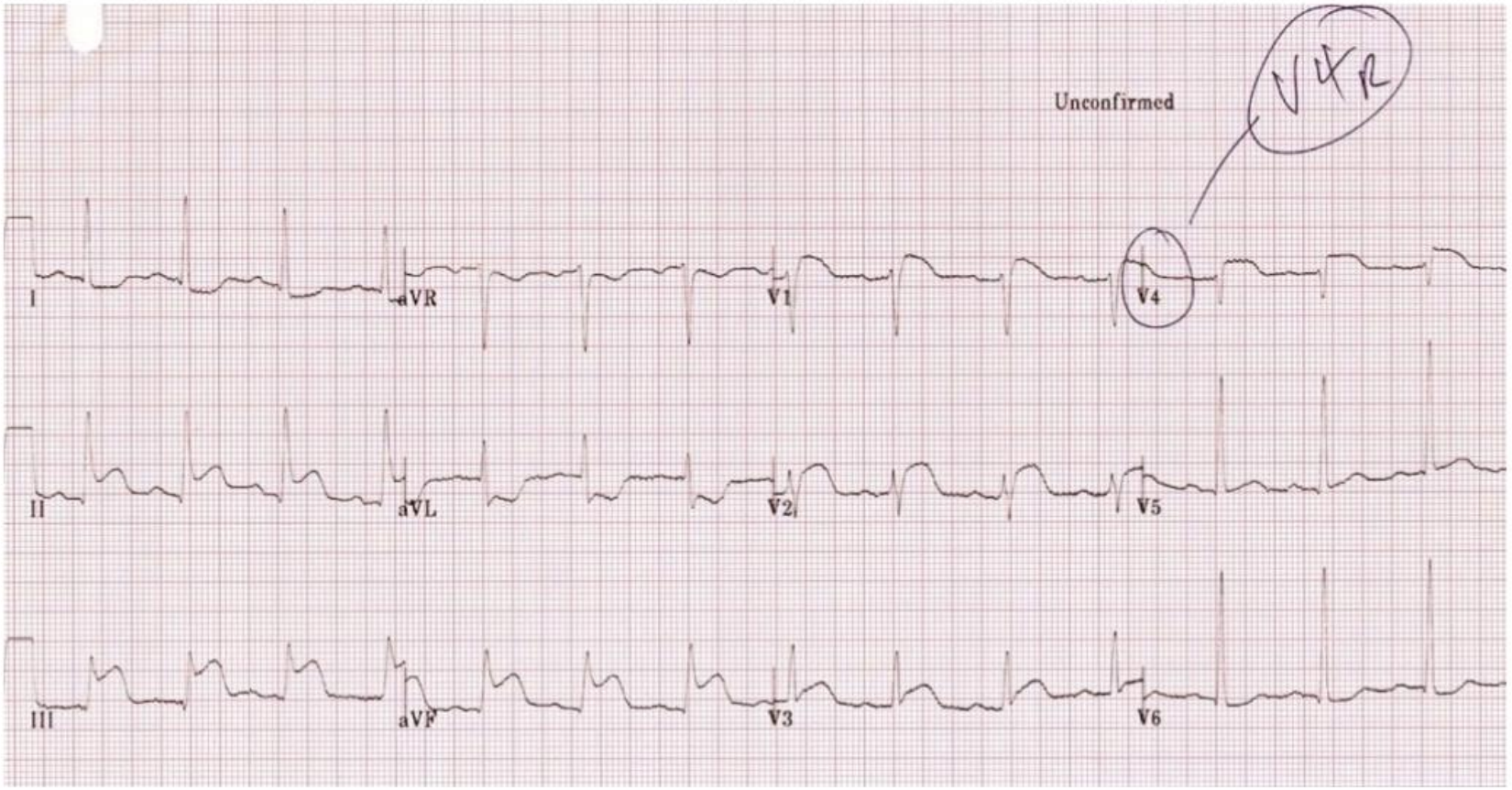
- ST elevation in V1
- ST elevation in lead III > lead II

How to confirm ? We do a right sided ECG

** We only move V4 from the left side to the right side while keeping it on the same level

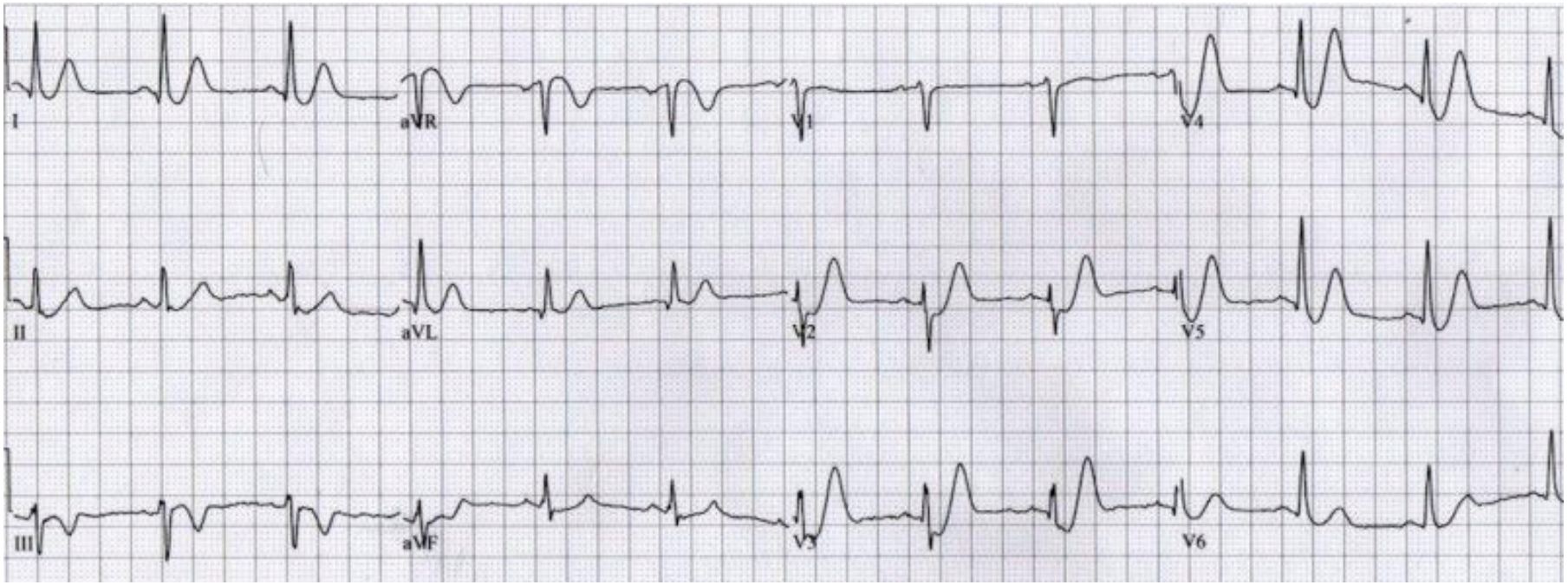
Example 1b

Repeat ECG of the same patient with V4R electrode position:



- There is ST elevation in V4R consistent with RV infarction

De Winter T Wave



Here we mainly care about V1,2,3

Note:

The de Winter ECG pattern is an **anterior STEMI equivalent** that presents *without* obvious ST segment elevation.

Diagnostic Criteria :

Tall, prominent, symmetric T waves in the precordial leads

Upsloping ST segment depression >1mm at the J-point in the precordial leads

Absence of ST elevation in the precordial leads

ST segment elevation (0.5mm-1mm) in aVR

“Normal” STEMI morphology may precede or follow the deWinter pattern

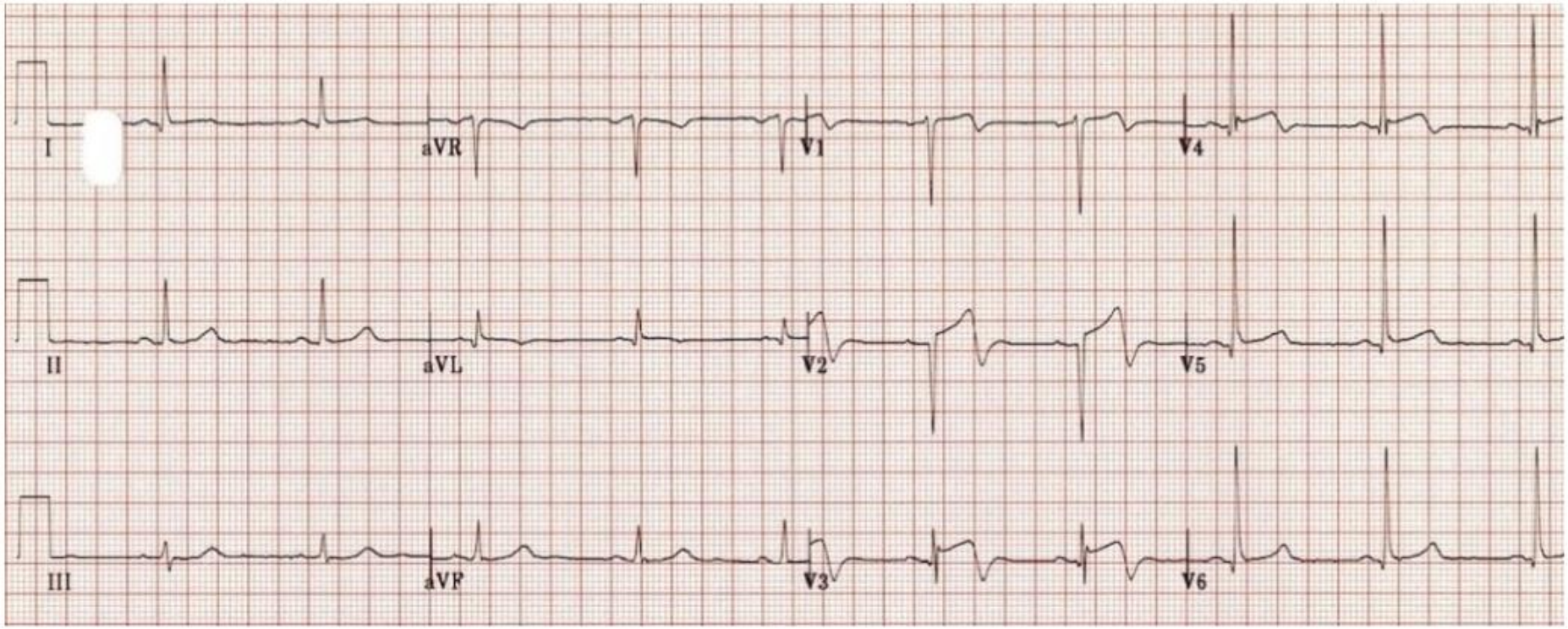
Wellens Syndrome

- Wellens syndrome is a pattern of **deeply inverted or biphasic T waves in V2-3**, which is highly specific for a **critical stenosis of the left anterior descending artery** (LAD).

- Patients may be pain free by the time the ECG is taken and have normally or minimally elevated cardiac enzymes; however, they are at extremely **high risk for extensive anterior wall MI** within the next few days to weeks.

- Due to the critical LAD stenosis, these patients usually require invasive therapy; do poorly with medical management; and may suffer MI or cardiac arrest if inappropriately stress tested.

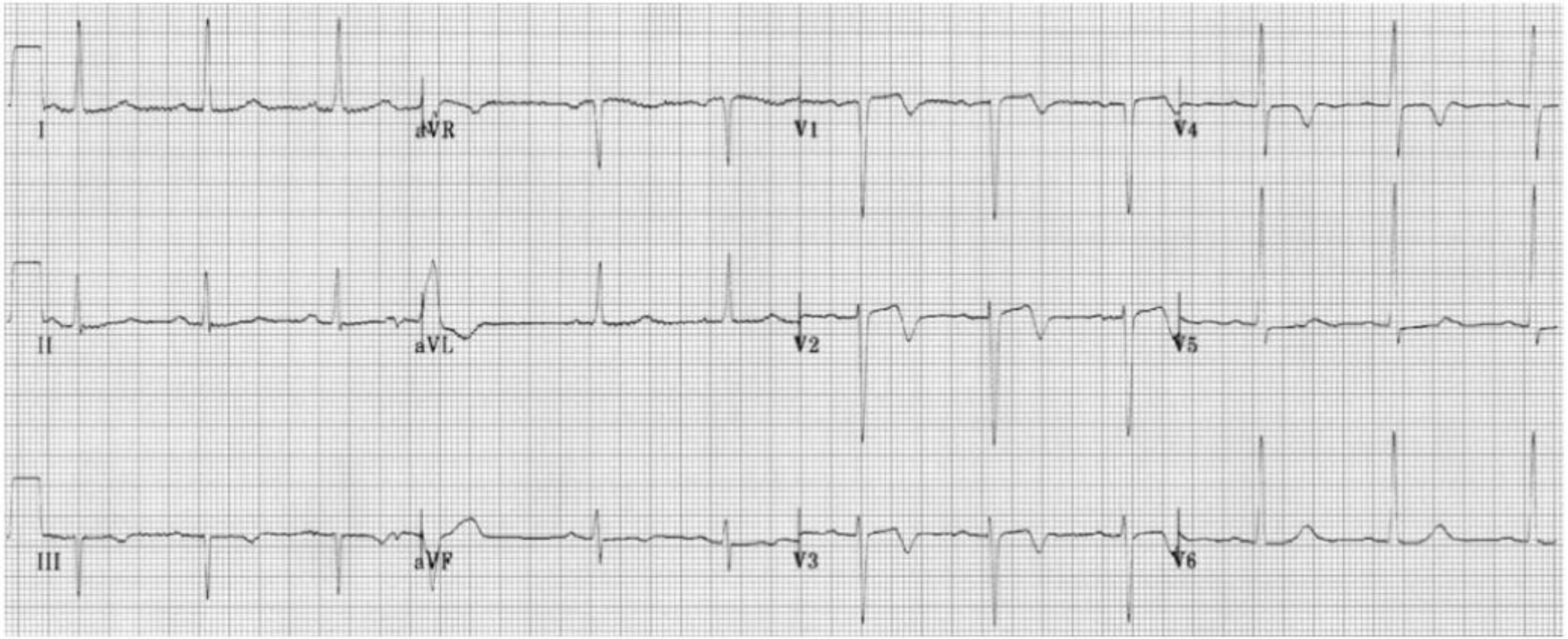
Example 1



Wellens Syndrome (Type A Pattern)

- Biphasic precordial T waves with terminal negativity, most prominent in V2-3.
- Minor precordial ST elevation.
- Preserved R wave progression (R wave in V3 > 3mm)

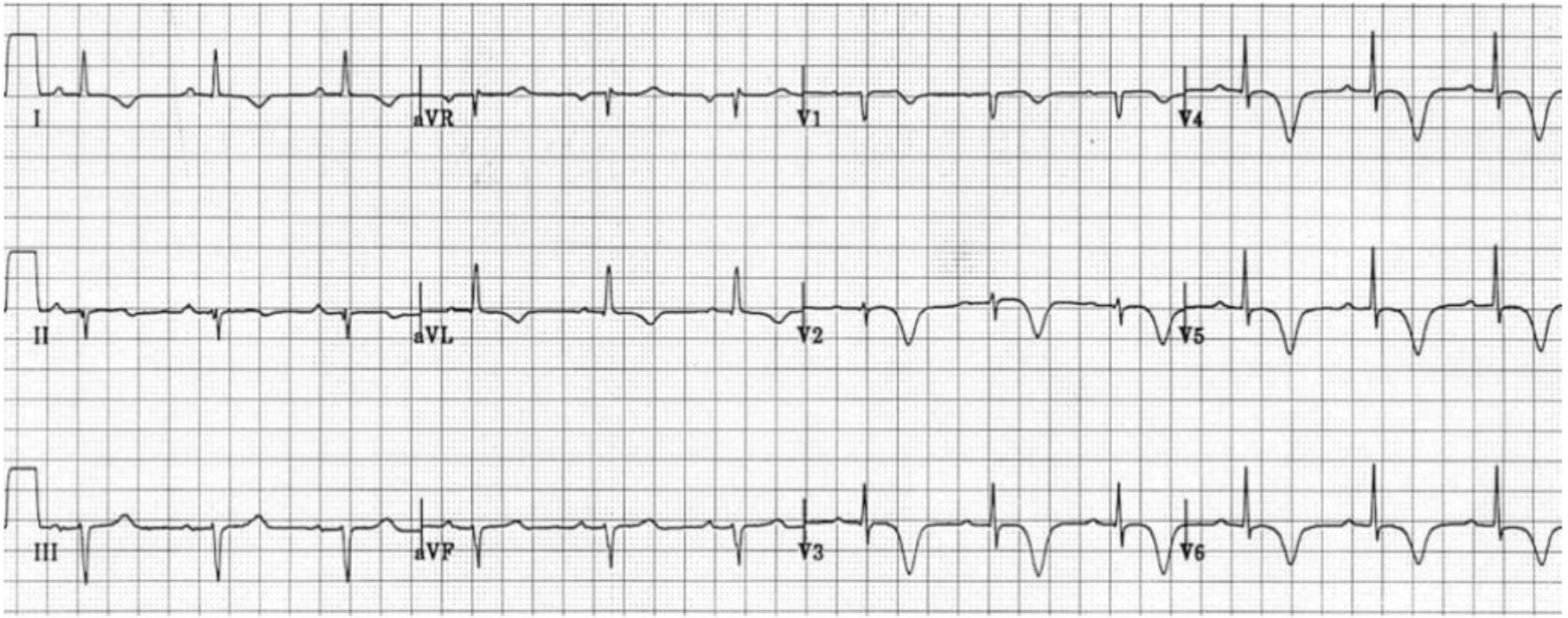
Example 2



Wellens Syndrome (Type A Pattern)

- The biphasic T waves in V2-3 are characteristic of Wellens syndrome.

Example 3



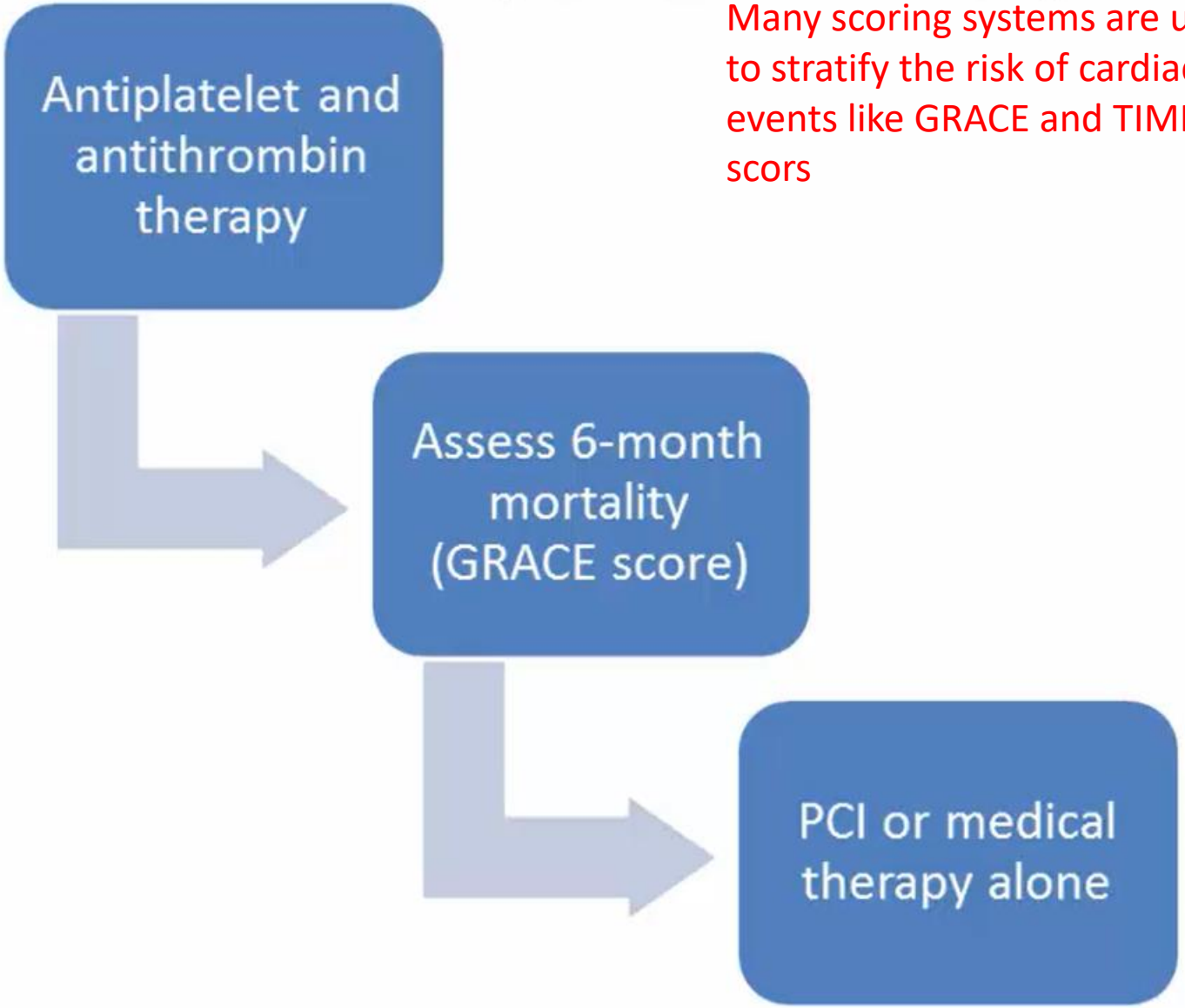
Wellens Syndrome (Type B Pattern)

- There are deep, symmetrical T wave inversions throughout the anterolateral leads (V1-6, I, aVL).

If NSTEMI is suspected

- As soon as the diagnosis of unstable angina or NSTEMI is made, and aspirin and antithrombin therapy have been offered, formally assess individual risk of future adverse cardiovascular events using an established risk scoring system that predicts 6-month mortality (for example, Global Registry of Acute Cardiac Events [GRACE]).

If NSTEMI is suspected



Many scoring systems are used to stratify the risk of cardiac events like GRACE and TIMI scors

Not required for the mini-osce

Risk assessment (GRACE Score)

- Age
- Heart Rate
- Systolic Blood Pressure
- Creatinine
- Heart failure
- Cardiac arrest at presentation
- Cardiac enzyme elevation
- ST deviation

Not required for the mini-osce

TIMI UA/NSTEMI RISK SCORE

1) Age ≥65	1 point
2) ≥3 risk factors for CAD	1 point
3) Use of ASA (last 7 days)	1 point
4) Known CAD (prior stenosis ≥50%)	1 point
5) >1 episode rest angina in <24 h	1 point
6) ST-segment deviation	1 point
7) Elevated cardiac markers	1 point

NSTEMI final managemant

Angiography

- Intermediate or higher risk
- Ischaemia returns
- Ischaemia on stress testing

Conservative

- Low risk