

Chest pain and Acute Coronary Syndrome

Emergency Medicine lectures

Types of Chest Pain

Musculo-skeletal

Pleuritic

Oesophageal

Pericarditis

Myocarditis

Aortic dissection

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

Cardiac-type chest pain

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

Typical description of cardiac type chest pain

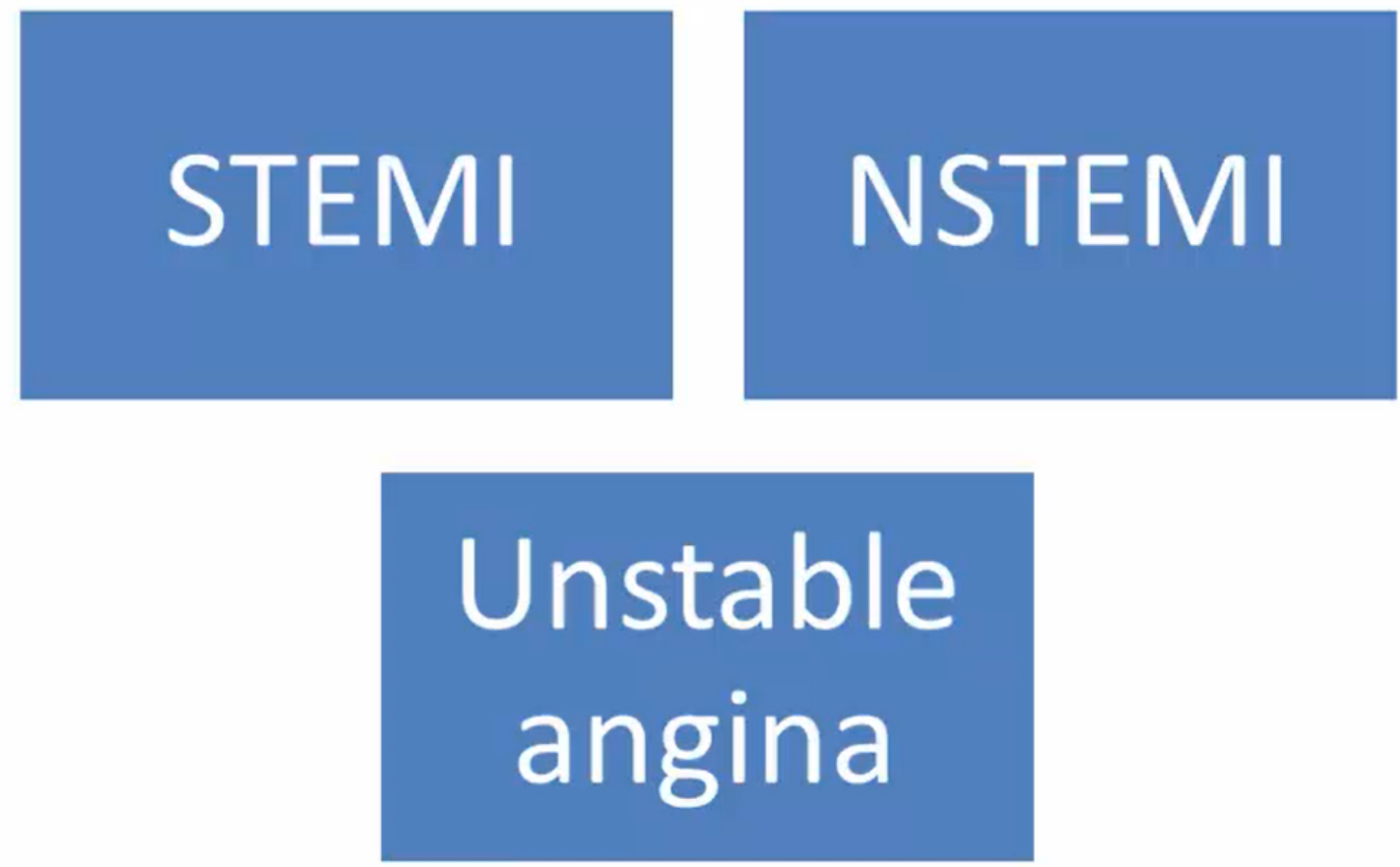
Heart Score for major cardiac event

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	• $\geq 3 \times$ Normal Limit • $> 1 - < 3 \times$ Normal Limit • $\leq$ 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		

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



A diagram showing the classification of Acute Coronary Syndromes. The title 'Acute Coronary Syndromes' is at the top. Below it, three blue boxes are arranged: 'STEMI' and 'NSTEMI' are side-by-side at the top, and 'Unstable angina' is centered below them.

STEMI

NSTEMI

Unstable
angina

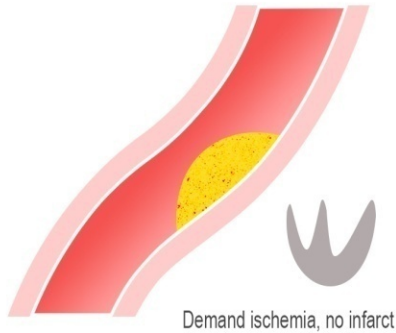
Acute coronary syndrome consists of : Unstable angina , NSTEMI and STEMI . It is part of Ischemic heart diseases that if left untreated it will lead to acute cardiac event and death

ACS

ACUTE CORONARY SYNDROME

1 STABLE ANGINA

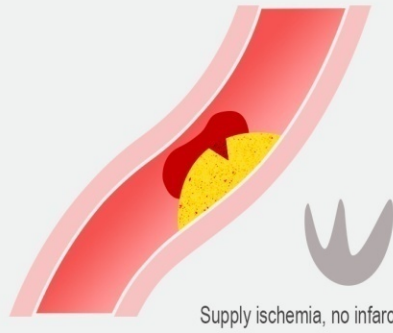
Angina pain develops when there is increased demand in the setting of a stable atherosclerotic plaque. The vessel is unable to dilate enough to allow adequate blood flow to meet the myocardial demand.



Normal

2 UNSTABLE ANGINA

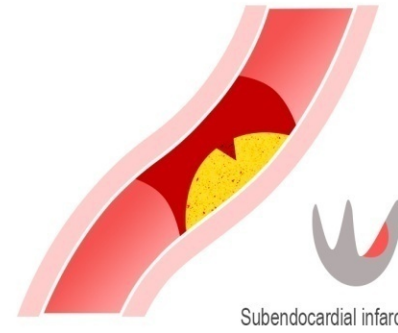
The plaque ruptures and a thrombus forms around the ruptured plaque, causing partial occlusion of the vessel. Angina pain occurs at rest or progresses rapidly over a short period of time.



Normal, Inverted T waves, or ST depression

3 NSTEMI

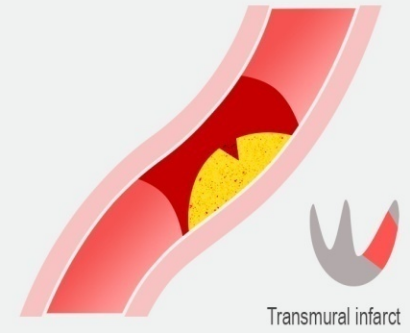
During an NSTEMI, the plaque rupture and thrombus formation causes partial occlusion to the vessel that results in injury and infarct to the subendocardial myocardium.



Normal, Inverted T waves, or ST depression

4 STEMI

A STEMI is characterized by complete occlusion of the blood vessel lumen, resulting in transmural injury and infarct to the myocardium, which is reflected by ECG changes and a rise in troponins.



Hyperacute T waves or ST elevation

ECG

TROPONINS

Normal

Normal

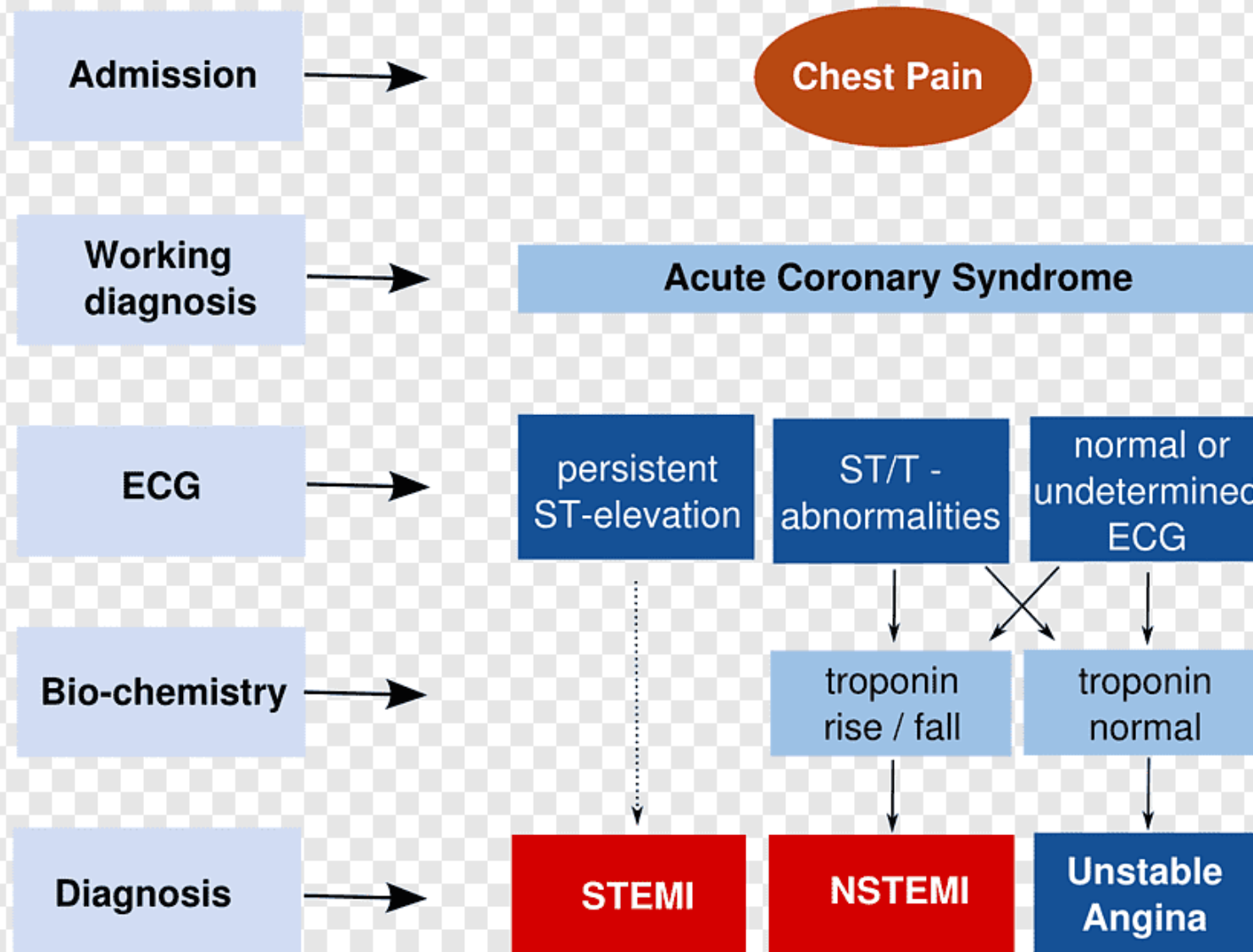
Elevated

Elevated

This infographic was created by Paula Sneath and Leah Zhao for the Sirens to Scrubs series of CanadiEM.org.

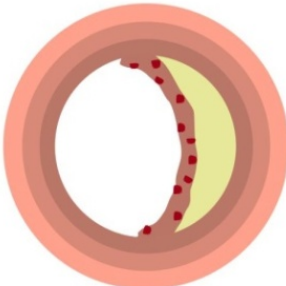
Acute coronary syndrome consists of : Unstable angina , NSTEMI and STEMI

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



UNSTABLE ANGINA

CORE
IM

PATHOPHYSIOLOGY	THROMBUS	CHEST PAIN SYMPTOMS
Ruptured plaque with non-occlusive thrombus* <i>*Occlusive thrombus would typically cause a full STEMI.</i> 	• White • Platelet-rich	• Acute chest pain • With activity and rest
Progressive mechanical obstruction 	• Red • Fibrin-rich <i>Same pathophysiology as stable angina.</i>	• "Crescendoing angina" <i>Chest pain worsens over days to weeks.</i> • 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!

2 MECHANISMS OF USA = RUPTURE , PROGRESSIVE MECHANICAL OBSTRUCTION

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 (Heparin)

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.

O₂ THERAPY

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.

Making Diagnosis

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 $> 265 \mu\text{mol/l}$)

Important timings

- Door to ECG : 10 minutes
- Door to needle (thrombolysis) : 60 minutes
- Door to PCI : 90 minutes
- Use fibrinolysis if primary PCI not available within 120 minutes of fibrynolysis

STEMI management

If < 12 hours:

Aim for reperfusion as quickly as possible

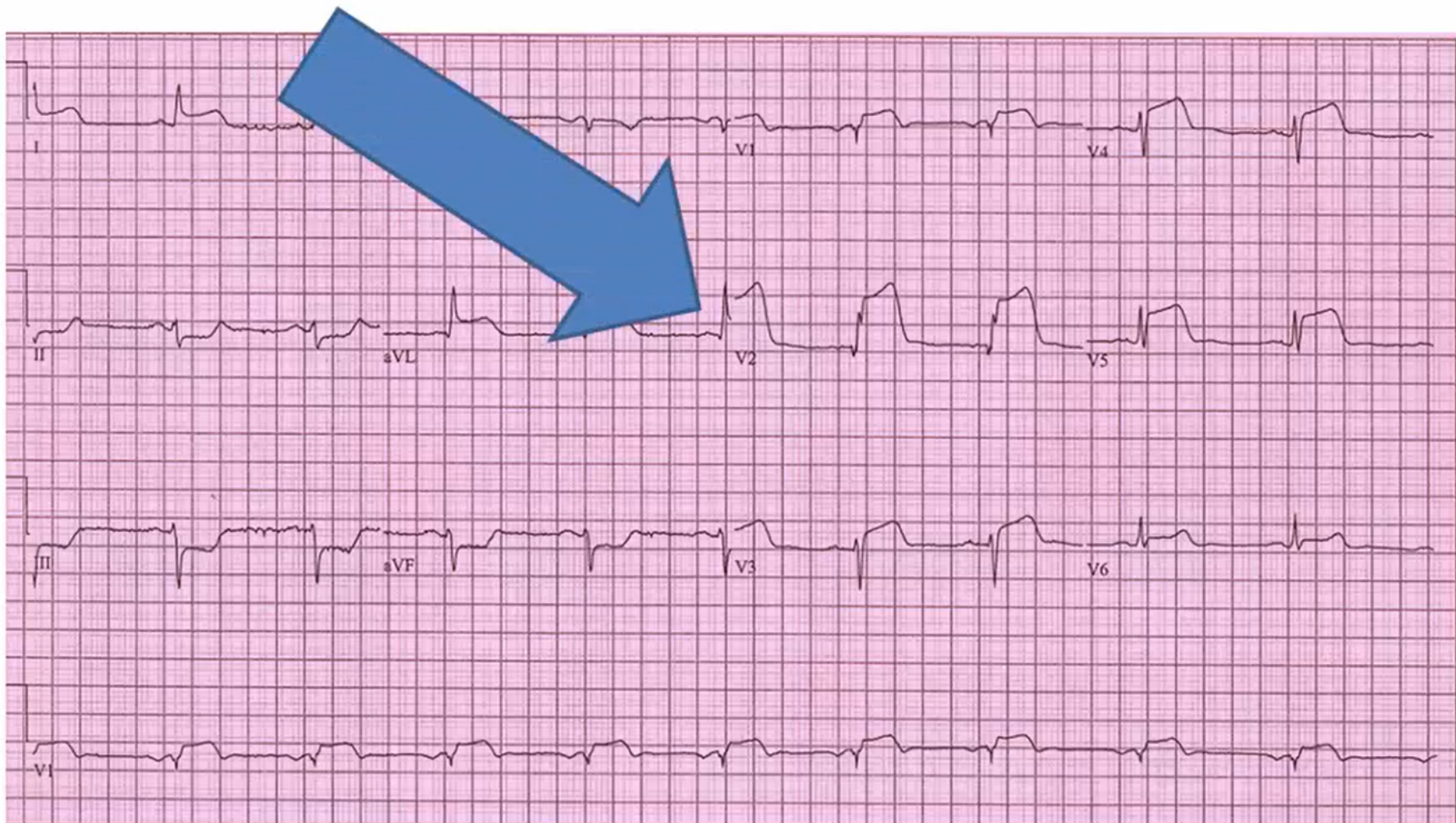
Primary PCI if possible

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

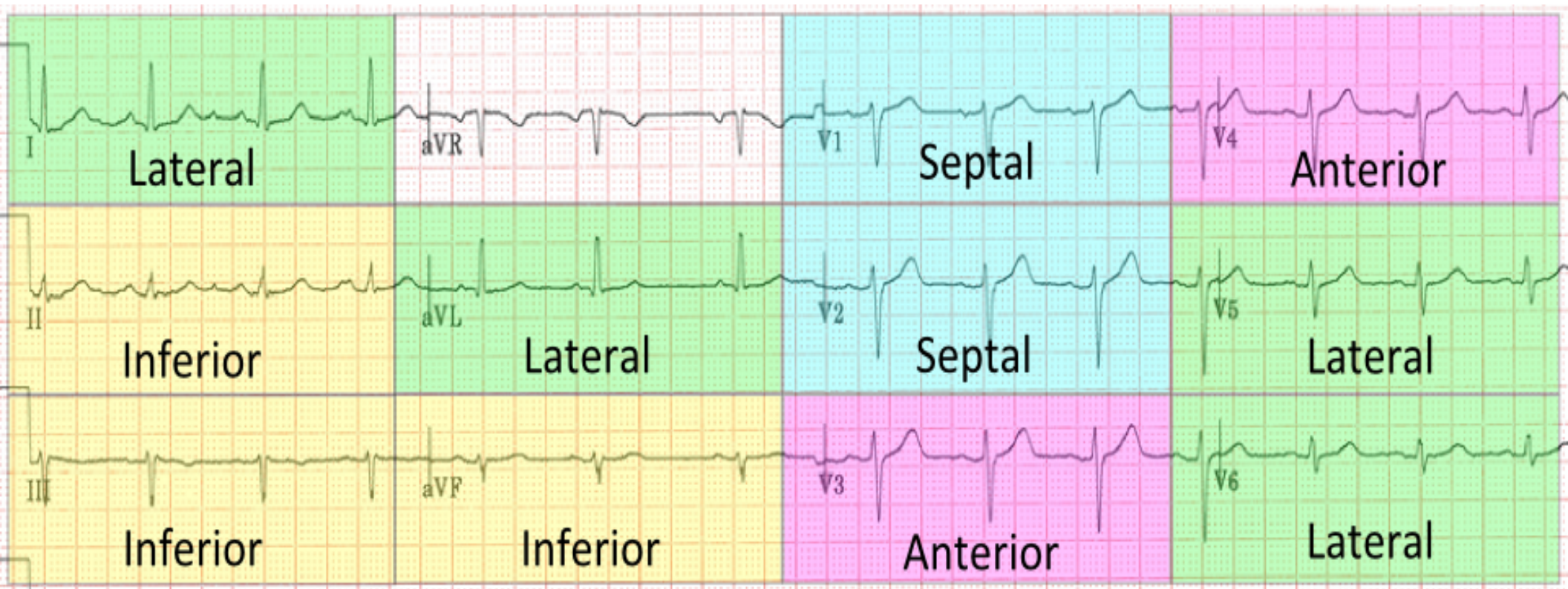
Give antithrombin with thrombolysis

TIME IS MUSCLE

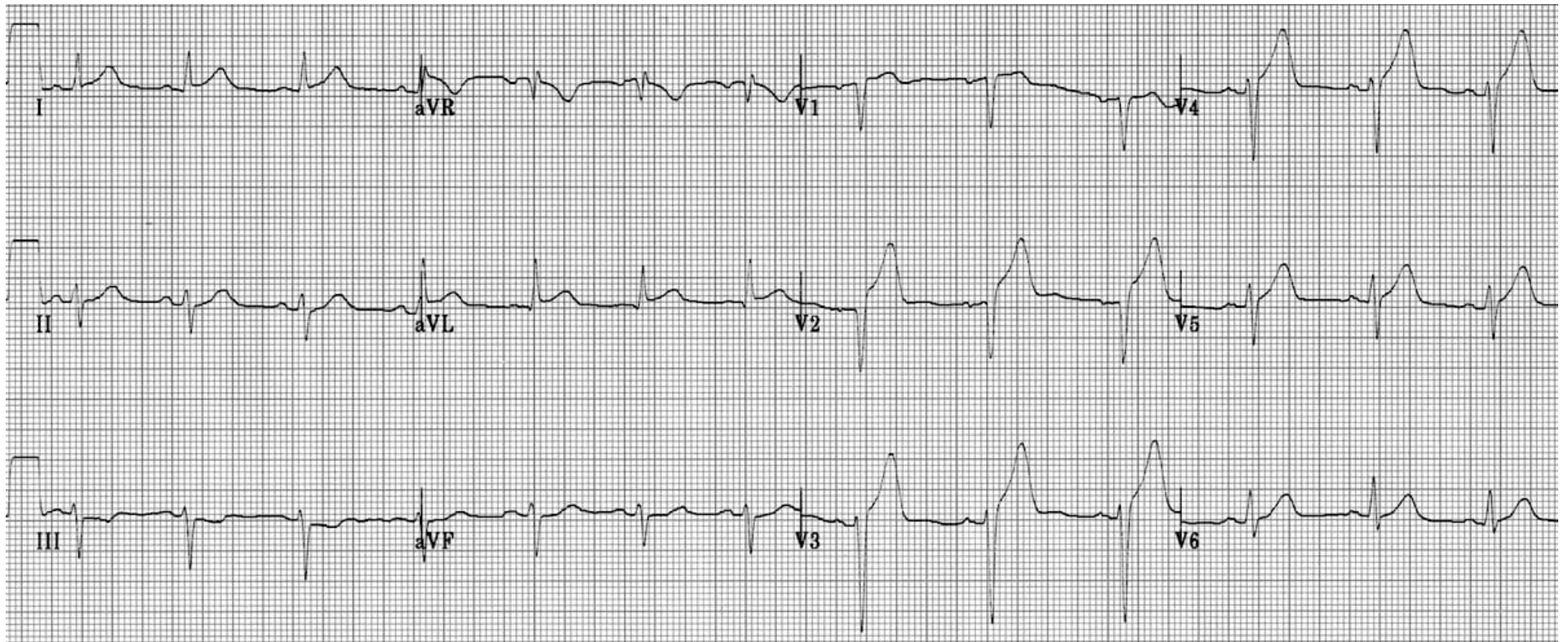
STEMI ?



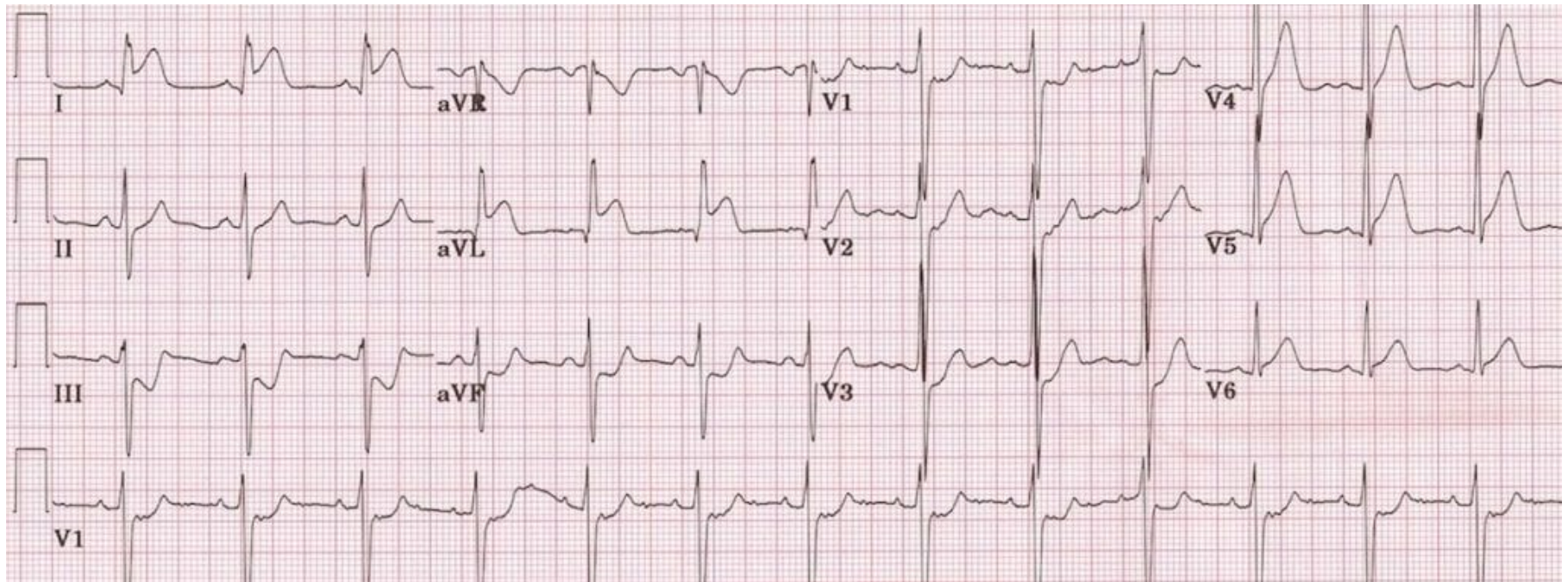
Distribution of leads



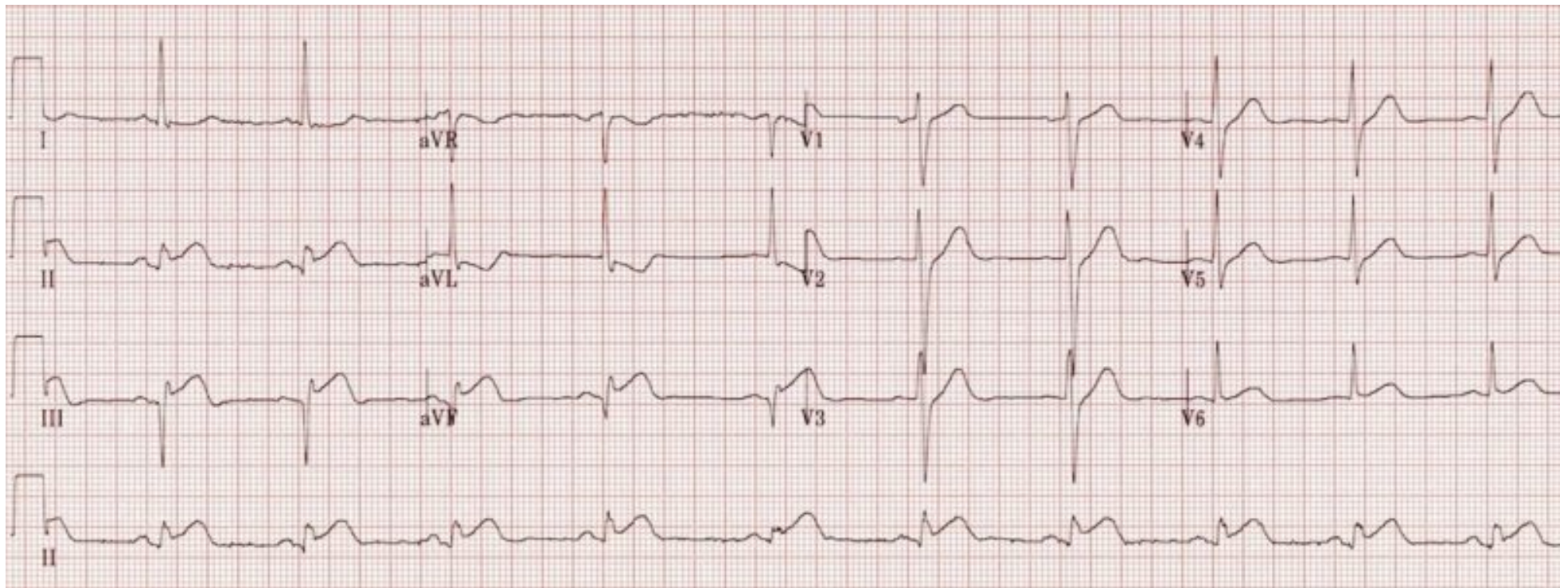
Anterior STEMI



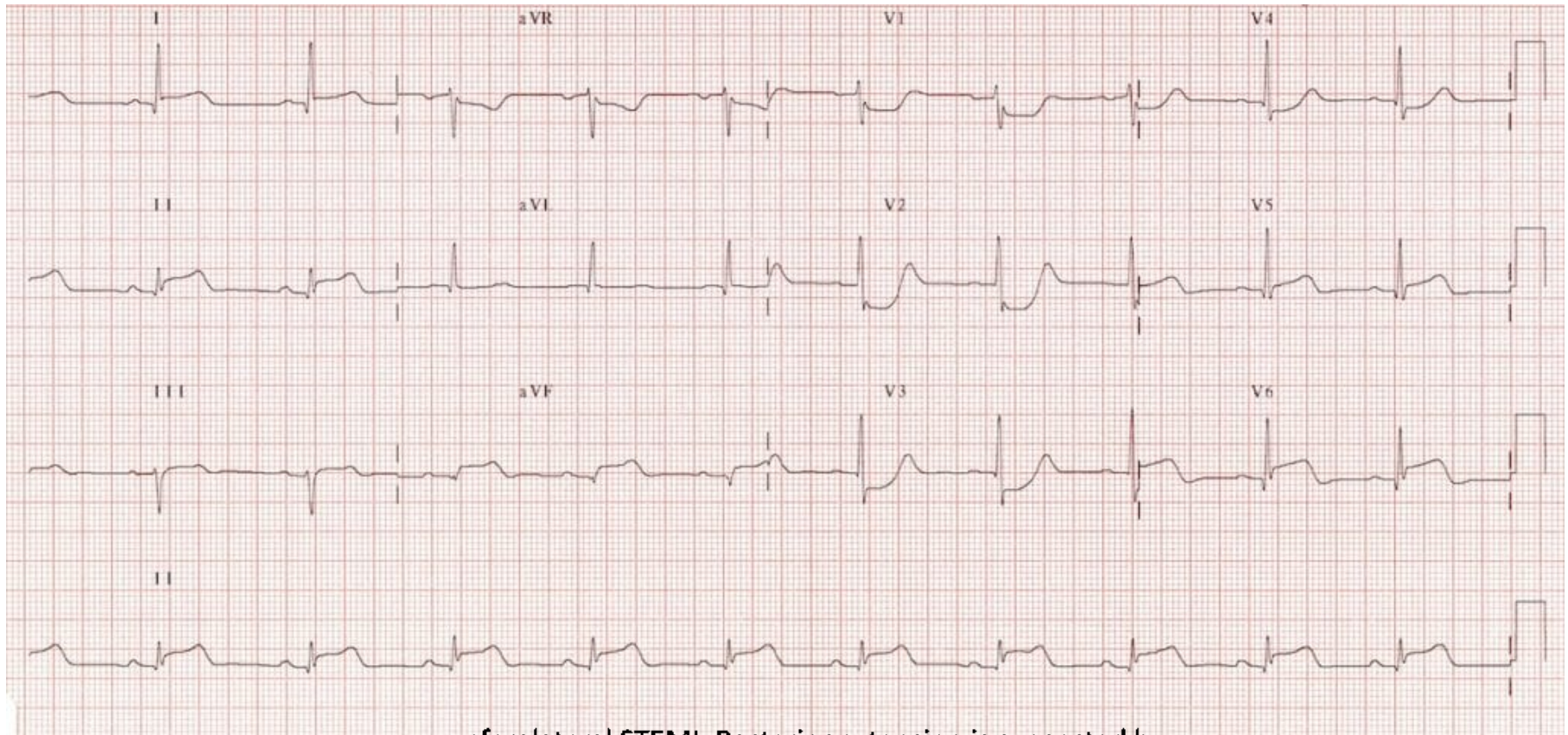
Lateral MI



Inferior MI



Posterior MI



inferolateral STEMI. Posterior extension is suggested by:

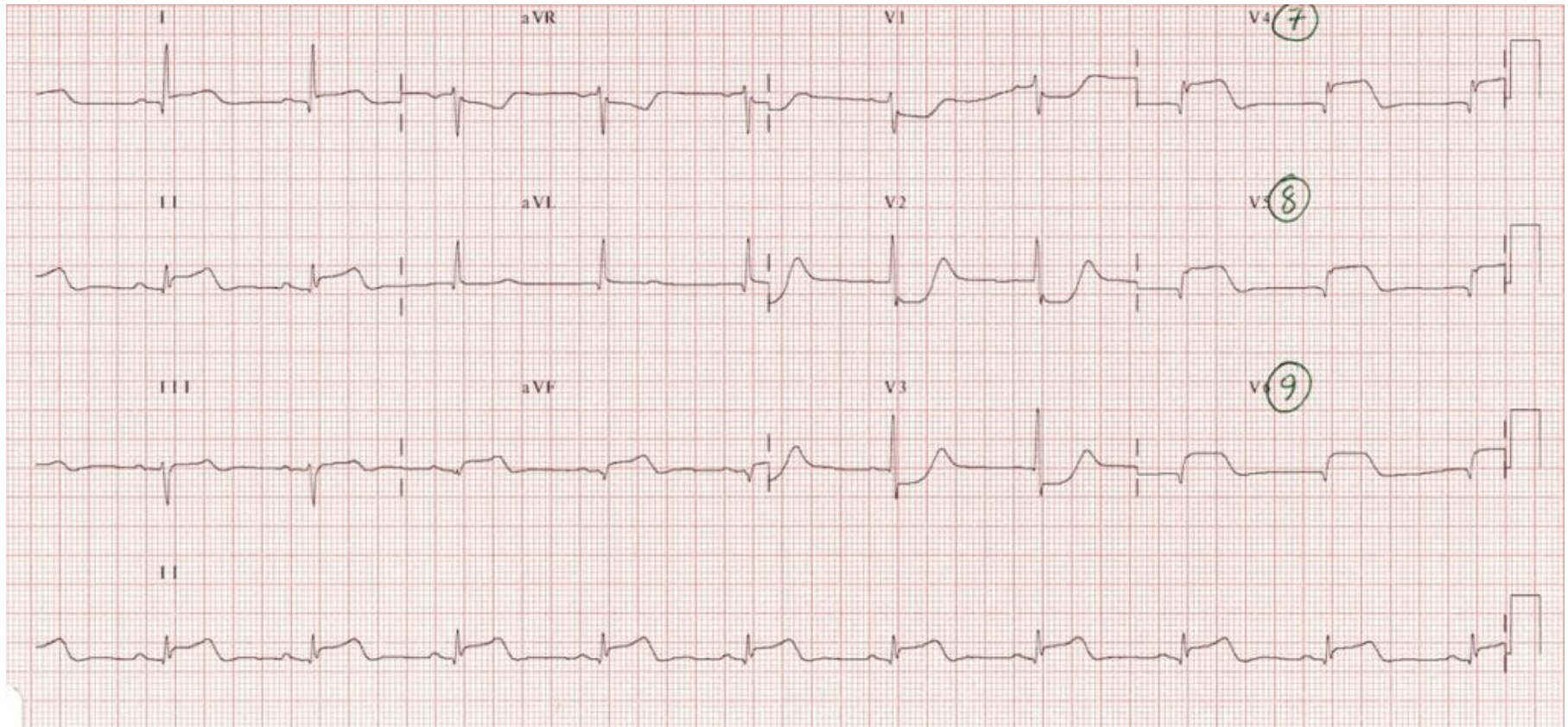
Horizontal ST depression in V1-3

Tall, broad R waves ($> 30\text{ms}$) in V2-3

Dominant R wave (R/S ratio > 1) in V2

Upright T waves in V2-3

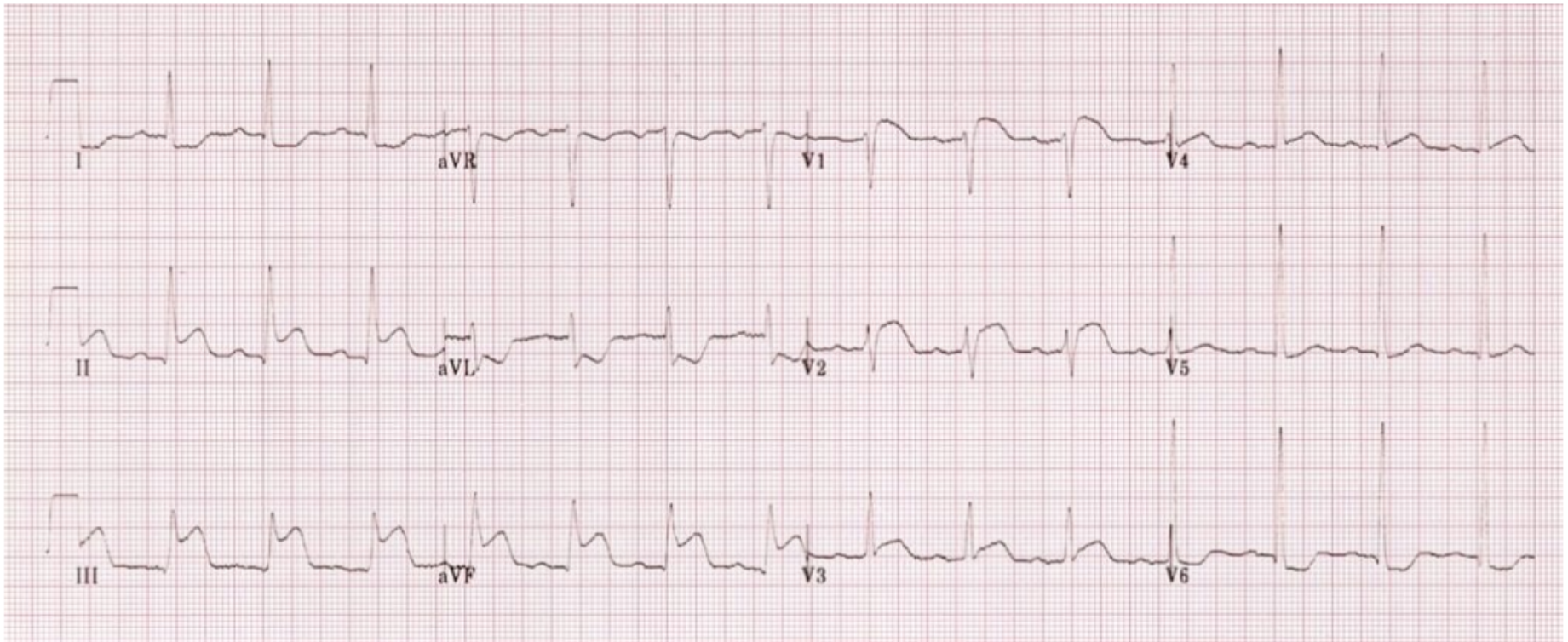
Posterior MI using posterior leads



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

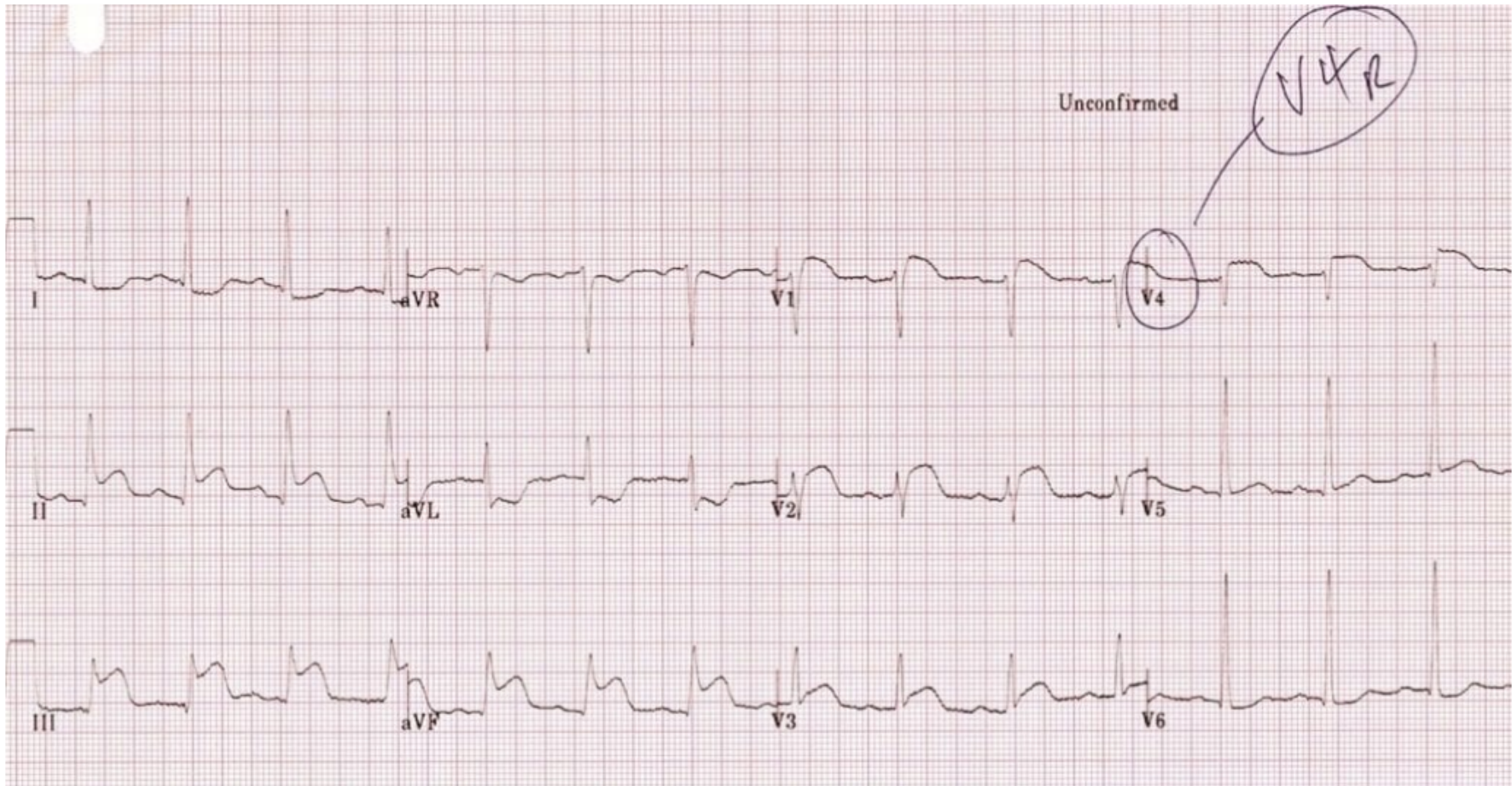


Inferior STEMI. Right ventricular infarction is suggested by:

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

Example 1b

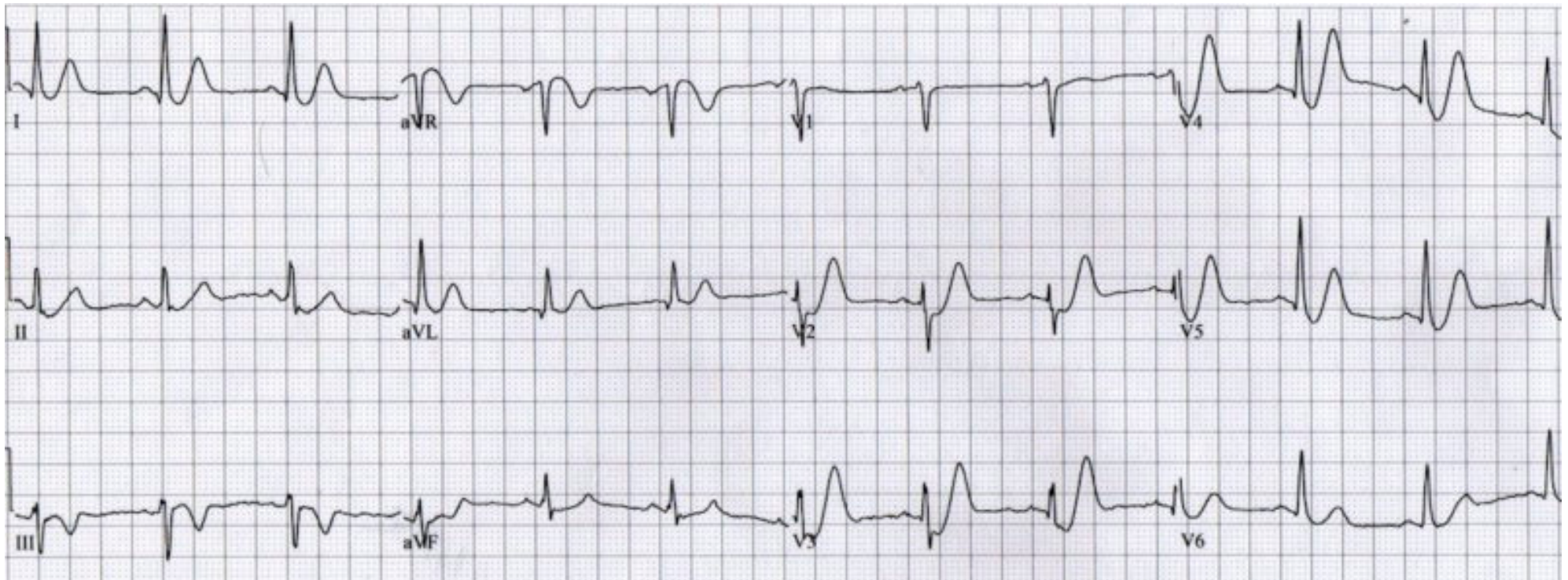
Repeat ECG of the same patient with V4R electrode position:



- There is ST elevation in V4R consistent with RV infarction

RT SIDED ECG

De Winter T Wave



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

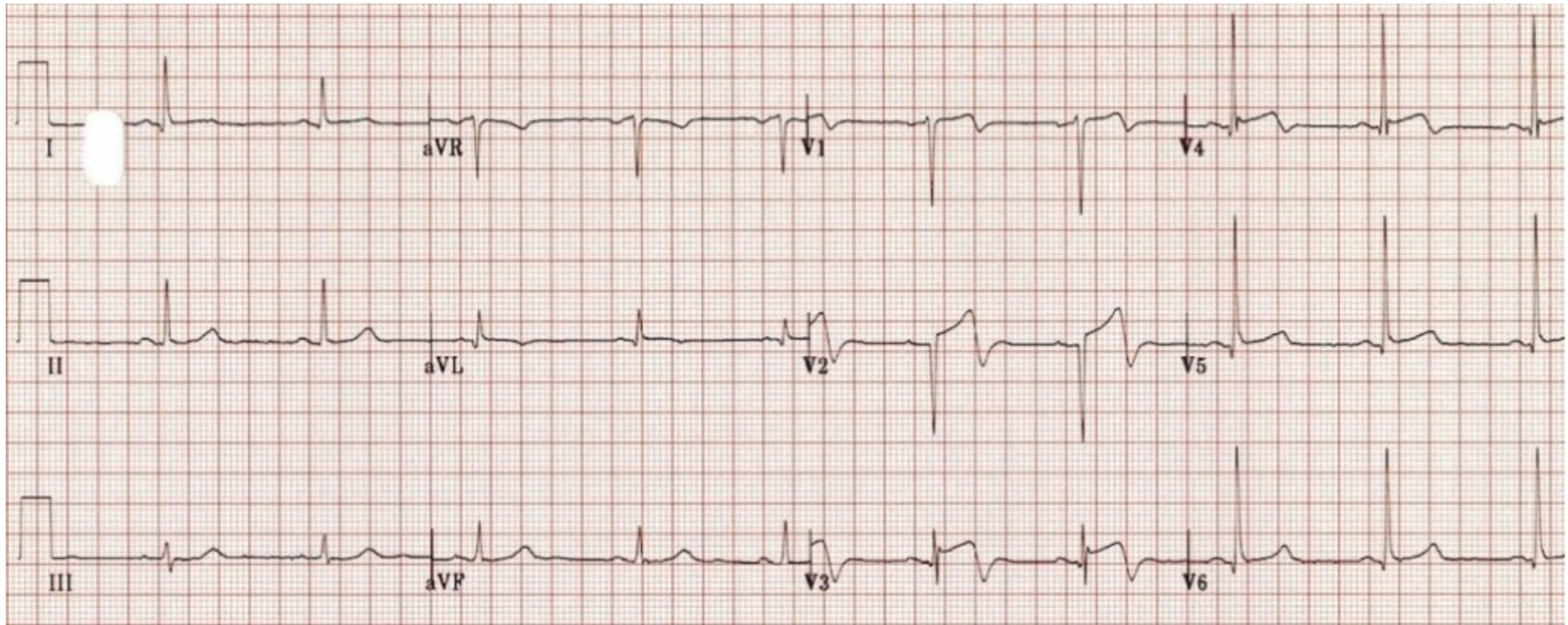
Diagnostic Criteria :

- Tall, prominent, symmetric T waves in the precordial leads
- Upsloping ST segment depression $>1\text{mm}$ 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.

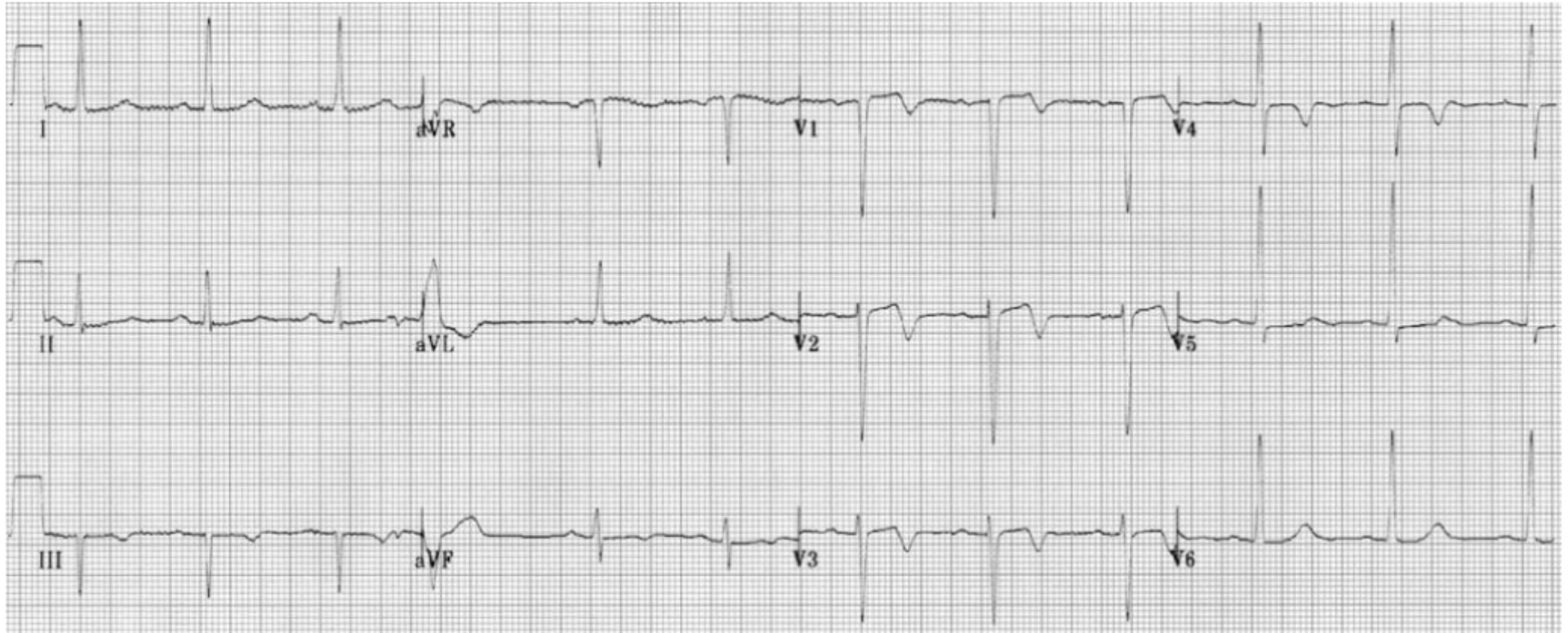
Example 1



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)

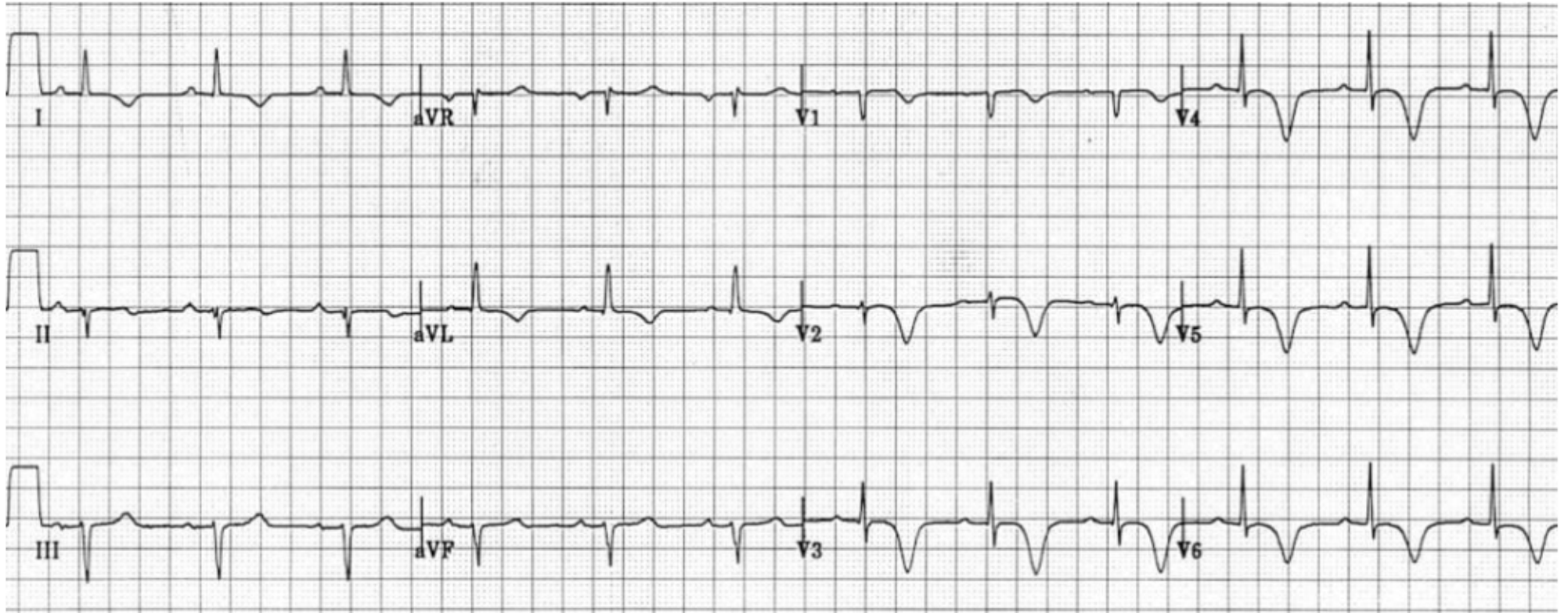
Example 2



Wellens Syndrome (Type A Pattern)

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

Example 3



Wellens Syndrome (Type B Pattern)

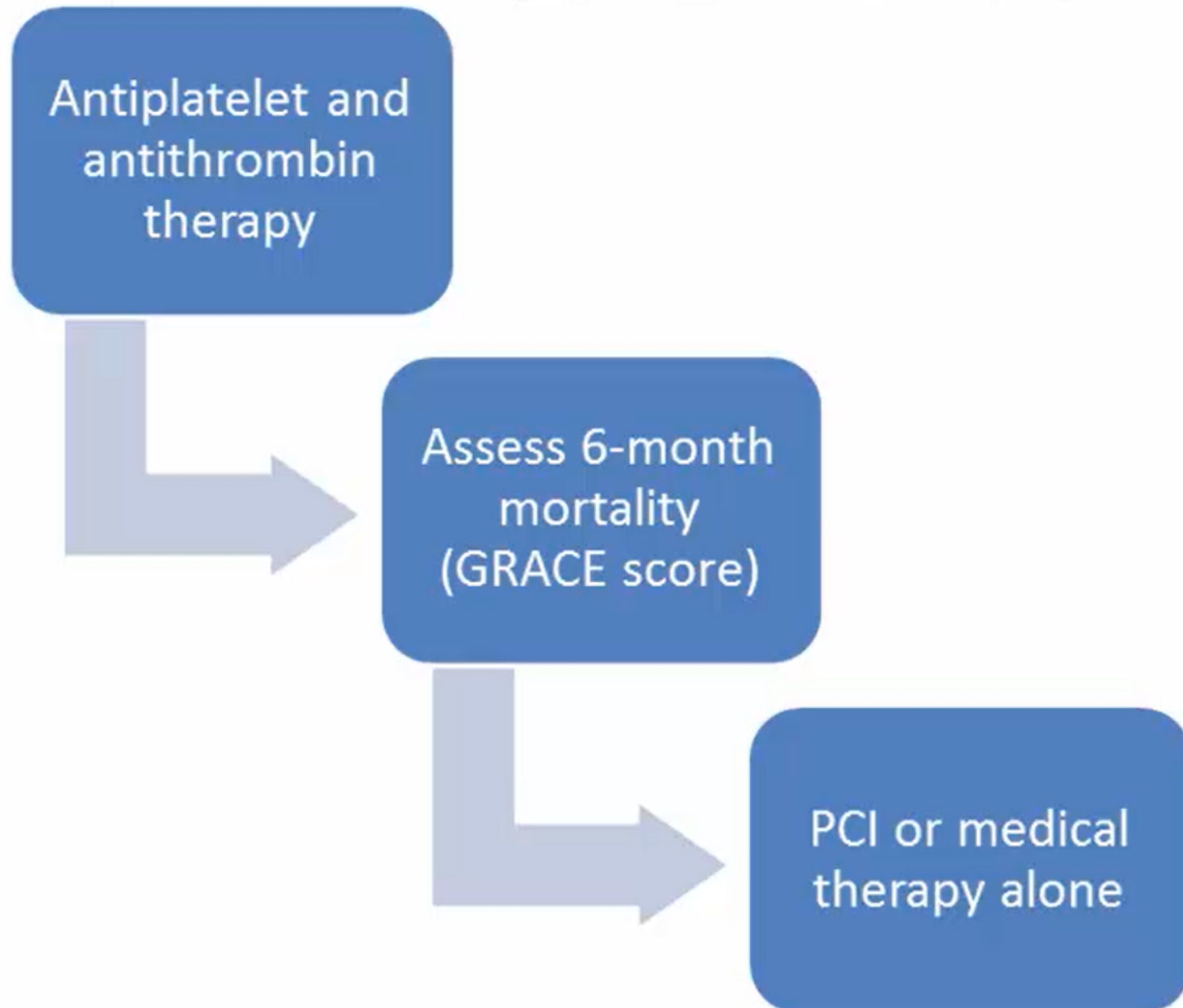
- There are deep, symmetrical T wave inversions throughout the anterolateral leads (V1-6, I, aVL).

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]).

PREDICT 6 MONTH MORTALITY , GRACE

If NSTEMI is suspected



Many scoring systems are used to stratify the risk of cardiac events like GRACE and **TIMI scores (thrombolysis in MI , 14 DAY RISK)**

Risk assessment (GRACE Score)

Age

Heart Rate

Systolic Blood Pressure

Creatinine

Heart failure

Cardiac arrest at presentation

Cardiac enzyme elevation

ST deviation

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 $\geq 50\%$)	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 management

Angiography

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

Conservative

- Low risk

