

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

*pneumonia PE
pneumothorax*



Cardiac-type chest pain

Typical description of cardiac type chest pain

- vitals, ECG, glucose

Location	Duration	Character
<ul style="list-style-type: none">Central (Diffuse)RadiationVisceral type	<ul style="list-style-type: none">>15 minutes< 24 hours <p>Remember Risk factors: 1. Myocardium 2. Severe ATN</p>	<ul style="list-style-type: none">Not sharpNot stabbingAcheBurningPressureNot movement or breathing related

Most up to diffrentiate
Diffrentiate by $\left\{ \begin{array}{l} \text{ACS} \rightarrow \text{sudden very intense pain} \\ \text{AD} \rightarrow \text{most peaked at first then decreases} \end{array} \right.$
BP variance btw Rt & Lt

Heart Score for major cardiac event

The HEART Score for Chest Pain Patients in the ED		
History	<ul style="list-style-type: none"> Highly Suspicious Moderately Suspicious Slightly or Non-Suspicious 	<ul style="list-style-type: none"> 2 points 1 point 0 points
ECG	<ul style="list-style-type: none"> Significant ST-Depression Nonspecific Repolarization Normal 	<ul style="list-style-type: none"> 2 points 1 point 0 points
Age	<ul style="list-style-type: none"> ≥ 65 years $> 45 - < 65$ years ≤ 45 years 	<ul style="list-style-type: none"> 2 points 1 point 0 points
Risk Factors	<ul style="list-style-type: none"> ≥ 3 Risk Factors or History of CAD 1 or 2 Risk Factors No Risk Factors 	<ul style="list-style-type: none"> 2 points 1 point 0 points
Troponin	<ul style="list-style-type: none"> $\geq 3 \times$ Normal Limit $> 1 - < 3 \times$ Normal Limit \leq Normal Limit 	<ul style="list-style-type: none"> 2 points 1 point 0 points
Risk Factors: DM, current or recent (<one month) smoker, HTN, HLP, family history of CAD, & obesity <i>high lipid profile</i>		
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

STEMI

NSTEMI



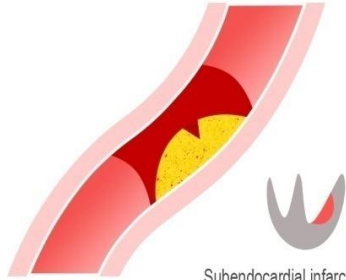
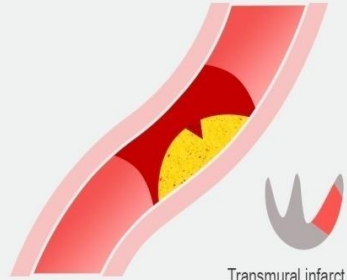




Unstable
angina

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.

unstable angina:-
↳ Duration
↳ Frequency
↳ Severity
↳ On rest
↳ No response for Nitro
First presentation with a coronary artery disease

ACS

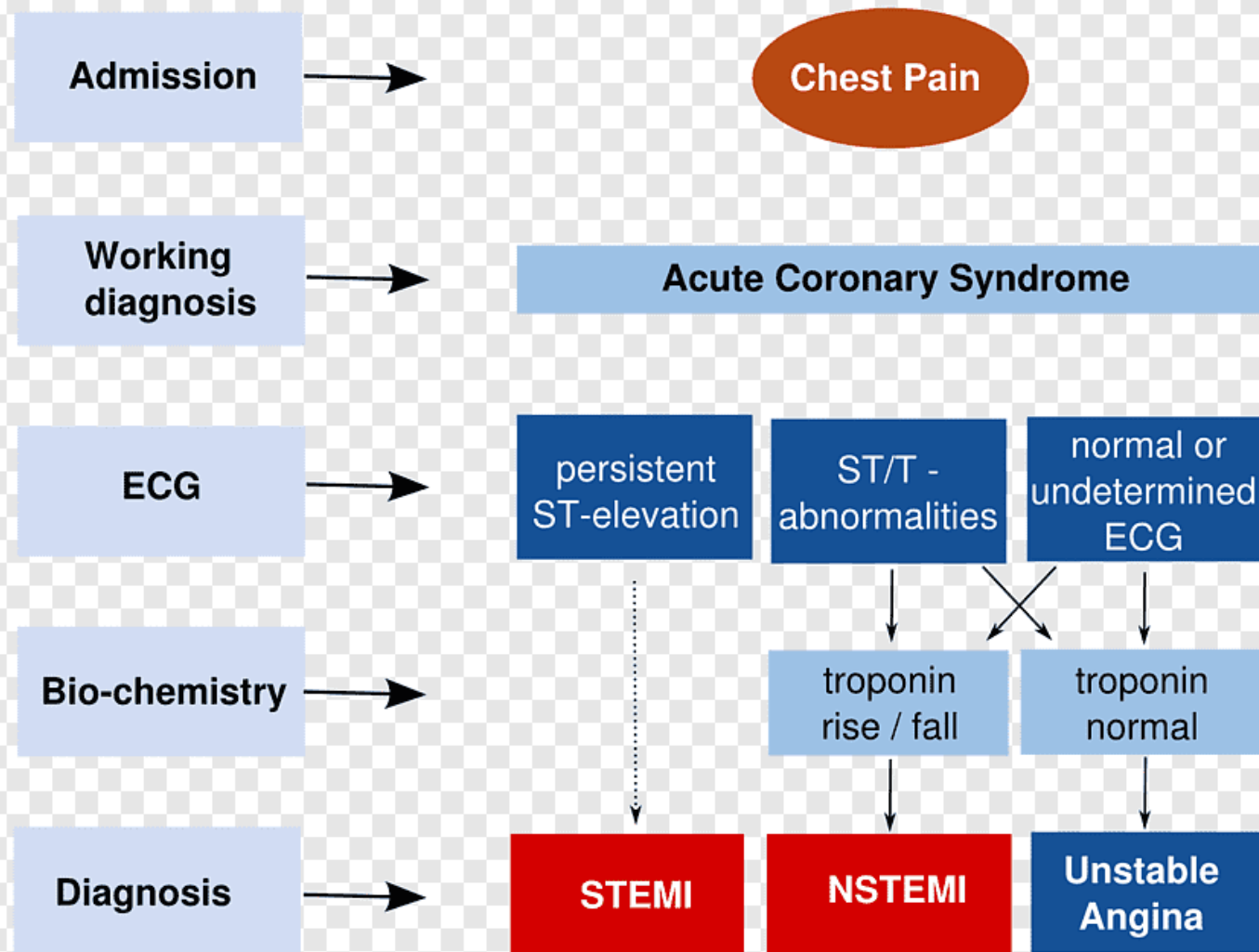
ACUTE CORONARY SYNDROME

	1 STABLE ANGINA	2 UNSTABLE ANGINA	3 NSTEMI	4 STEMI
	<p>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.</p>  <p>Demand ischemia, no infarct</p>	<p>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.</p>  <p>Supply ischemia, no infarct</p>	<p>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.</p>  <p>Subendocardial infarct</p>	<p>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.</p>  <p>Transmural infarct</p>
ECG	 <p>Normal</p>	 <p>Normal, Inverted T waves, or ST depression</p>	 <p>Normal, Inverted T waves, or ST depression</p>	 <p>Hyperacute T waves or ST elevation</p>
TROPONINS	Normal	Normal	Elevated	Elevated

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


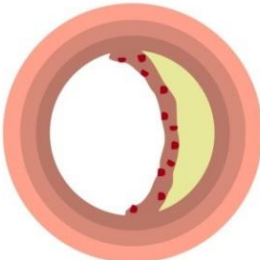
Acute coronary syndrome consists of : Unstable angina , NSTEMI and STEMI

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
<p>Ruptured plaque with non-occlusive thrombus*</p> <p><i>*Occlusive thrombus would typically cause a full STEMI.</i></p> 	<ul style="list-style-type: none"> • White • Platelet-rich 	<ul style="list-style-type: none"> • Acute chest pain • With activity and rest
<p>Progressive mechanical obstruction</p> 	<ul style="list-style-type: none"> • Red • Fibrin-rich <p><i>Same pathophysiology as stable angina.</i></p>	<ul style="list-style-type: none"> • "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!

Pre/In-hospital management of suspected ACS

Give the patient MONA

M: Morphine (pain management)

up to 5ml
some pt may have severe vomiting
so give with
an antiemetic

O : oxygen according to BTS protocol

↳ if $<94\%$ for normal, $<88\%$ COPD

N : Nitroglycerin for pain management

↳ if SBP <90 → don't give!

A : Anti-platelets (Aspirin)

give dose 325mg crushed
↳ for both effects
antiplatelet
anti-inflammatory

H :- Anti-thrombin
(Heparin)

if pt unconscious
insert 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. → 10 mins

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.

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
↳ after 12 hours
- 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 $\mu\text{mol/l}$)

STEMI management

If < 12 hours:

Aim for reperfusion as quickly as possible

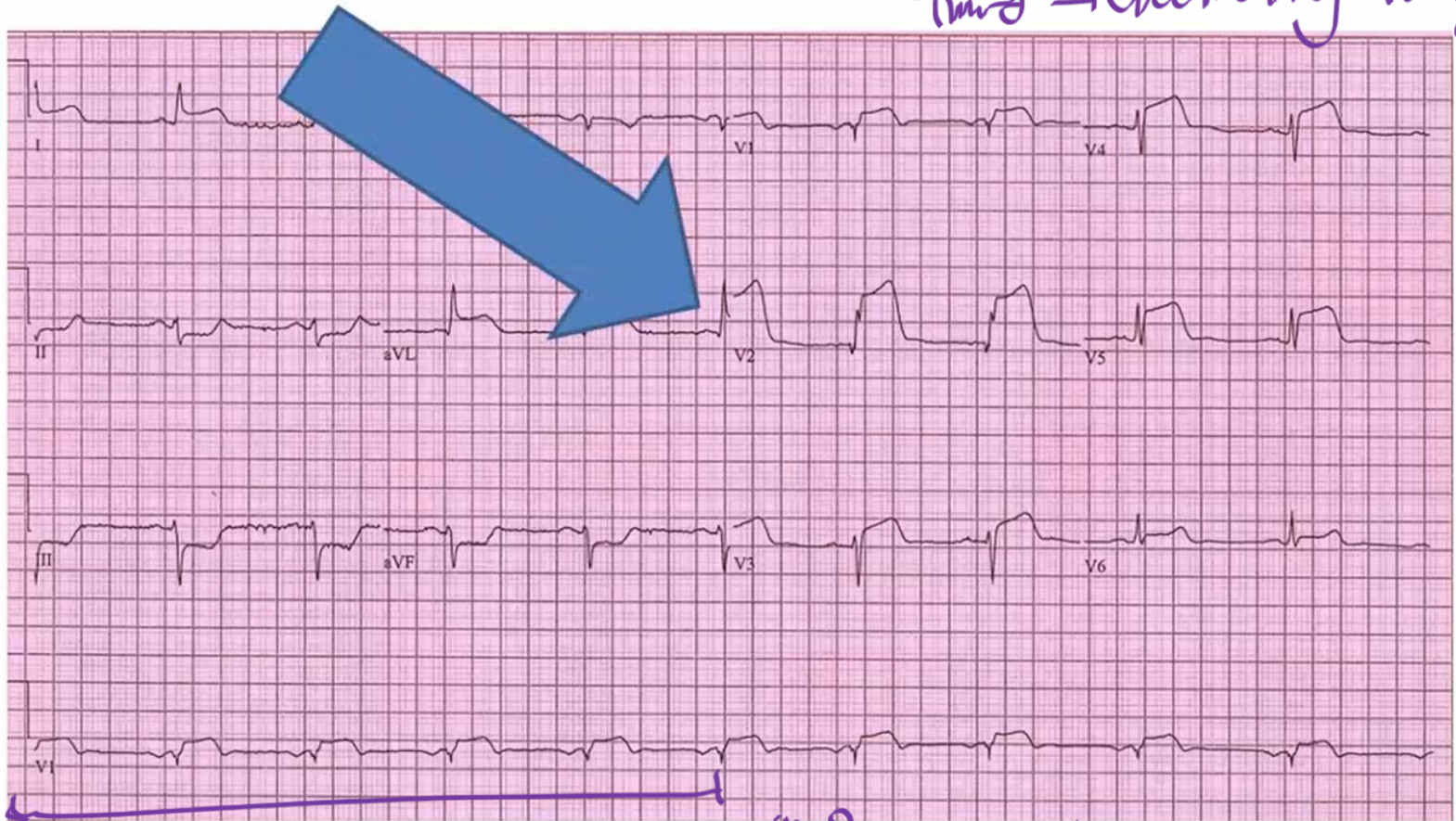
Primary PCI if possible

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

Give antithrombin with thrombolysis

STEMI ? ^{First rhythm}

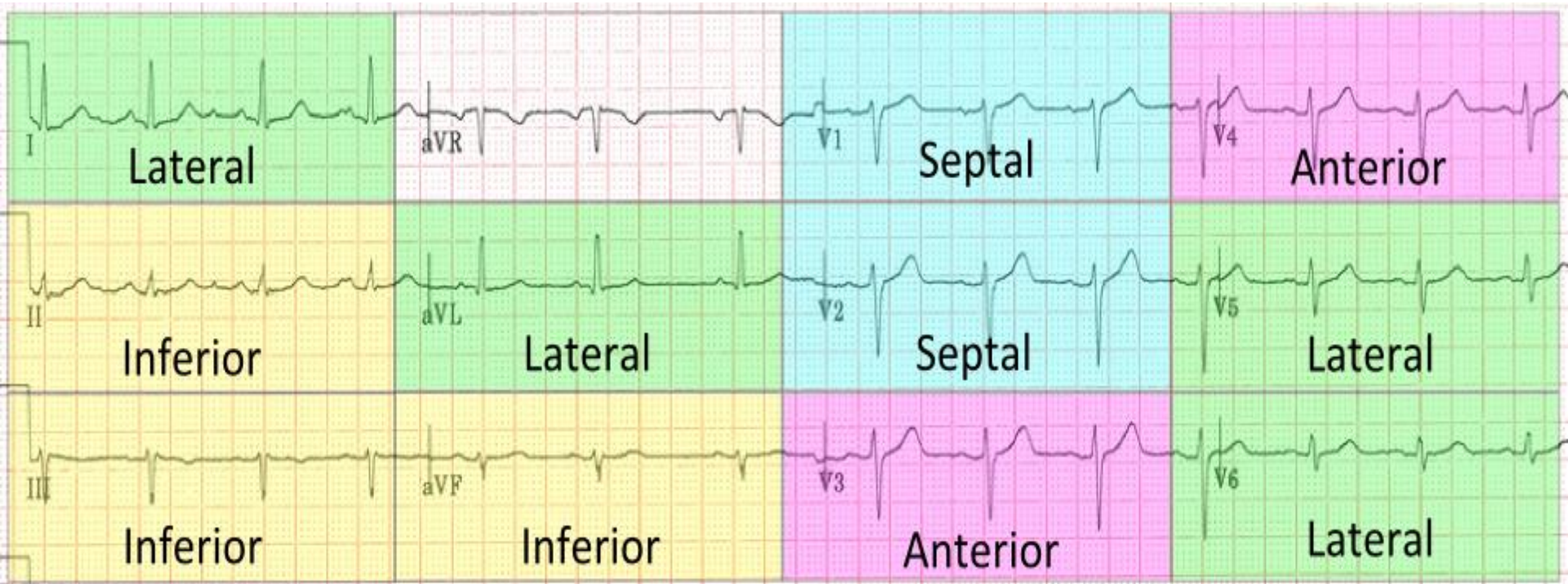
Second → ischemia / infarction
Third → electrolyte Fourth → other



Limb leads
Lyon test elevation = 1 small block in 2 consequent leads = equivalent ST elevation

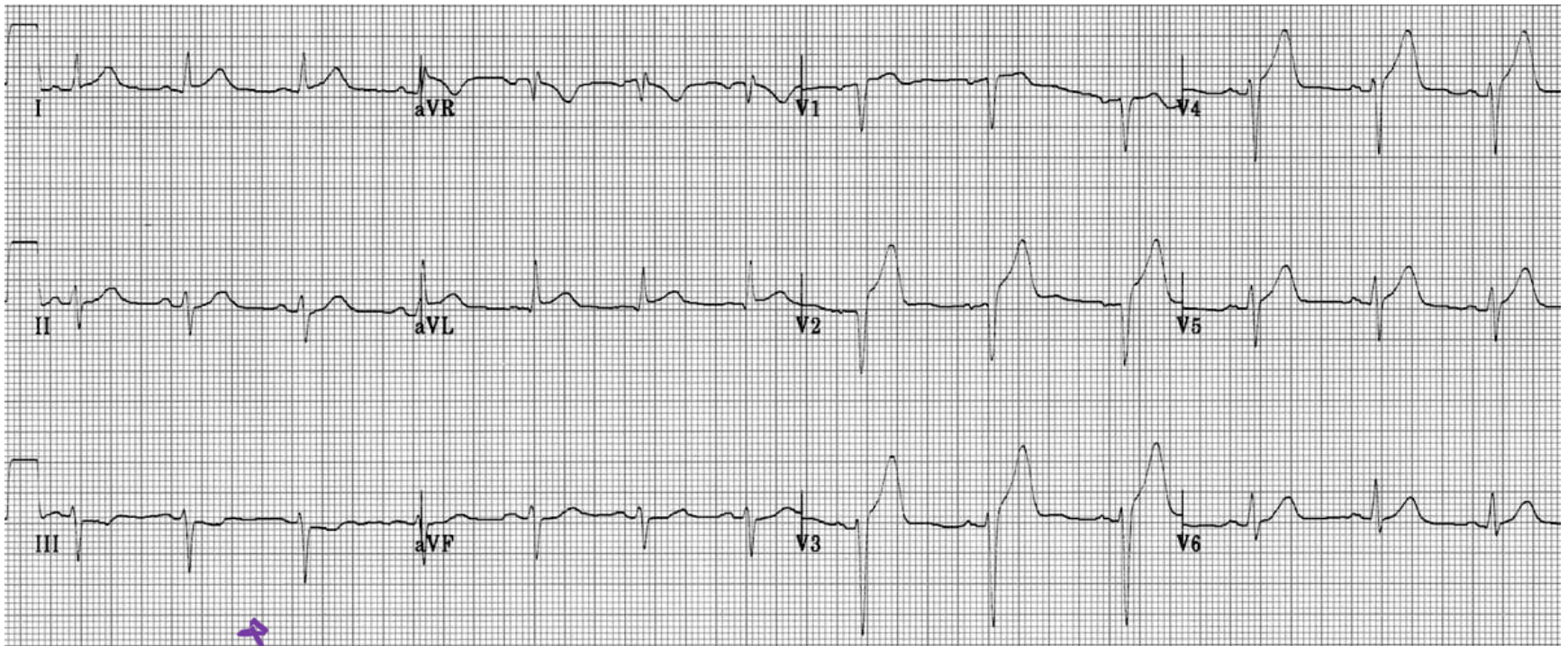
In chest leads → 2 small blocks in 2 consequent leads = =

Distribution of leads



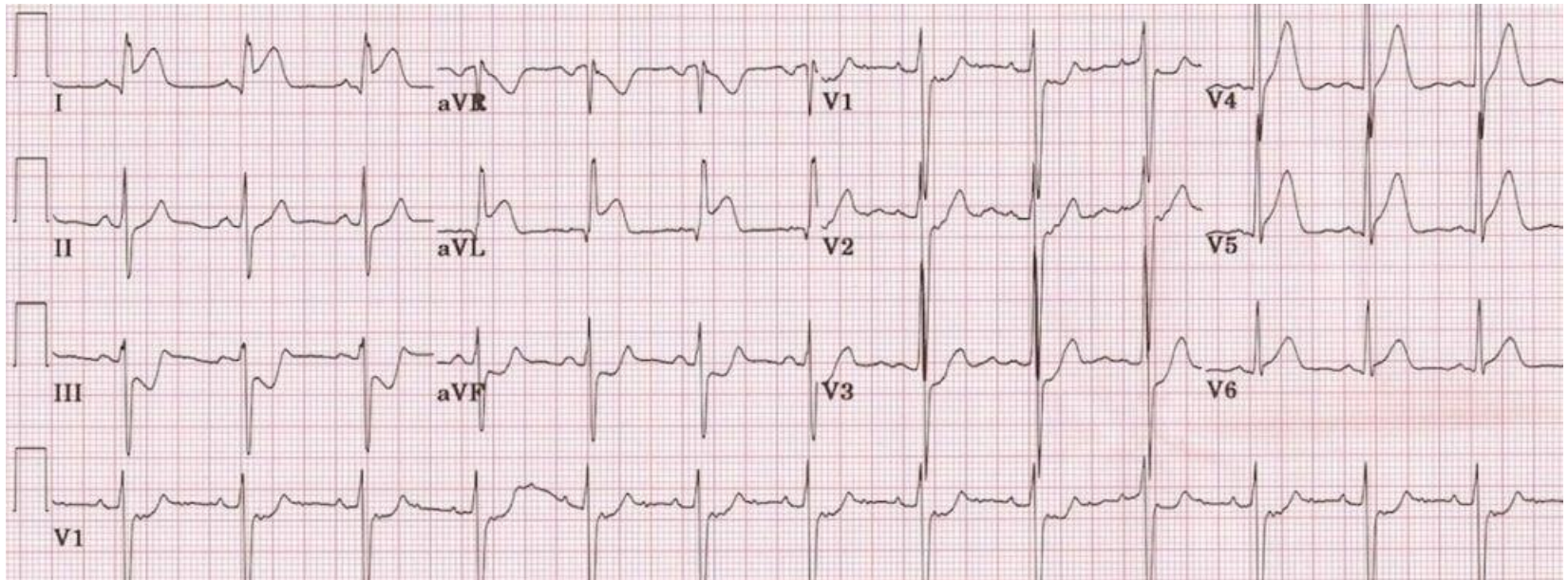
Anterior STEMI

Lateral → on the borderline so repeat ECG after 15 min



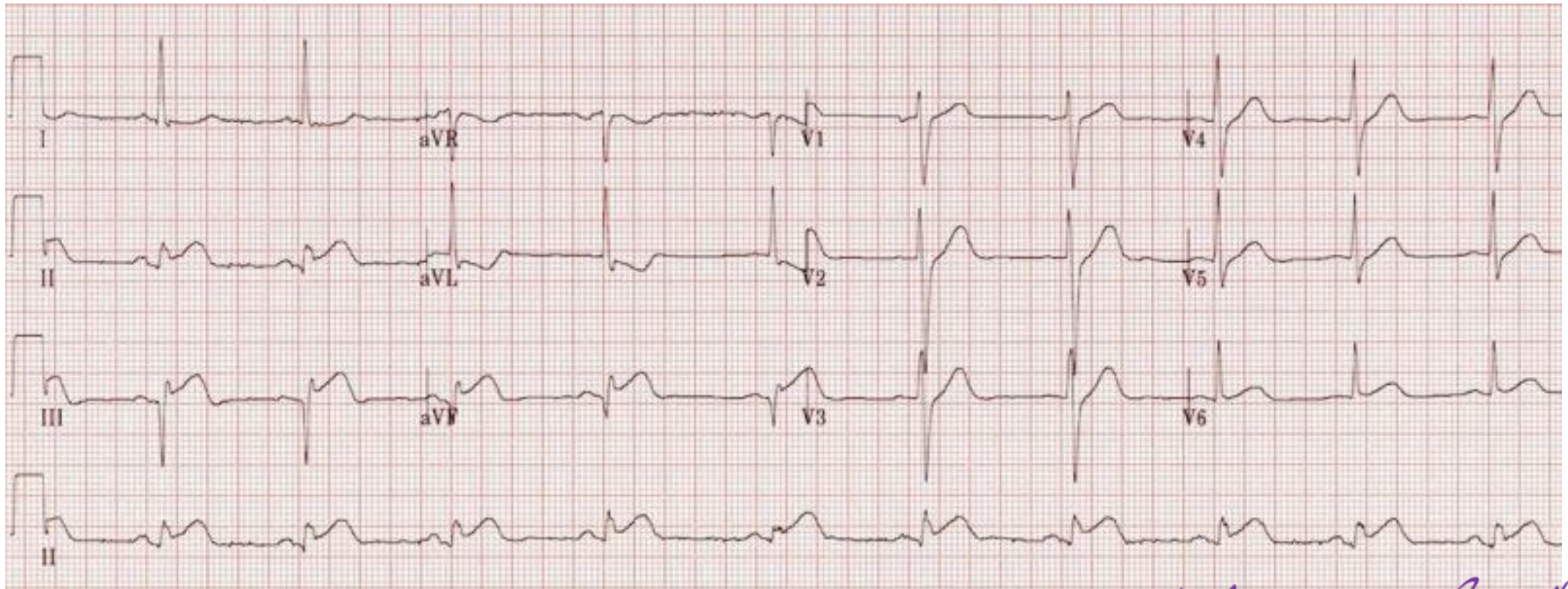
reciprocal changes
ST depression

Lateral MI



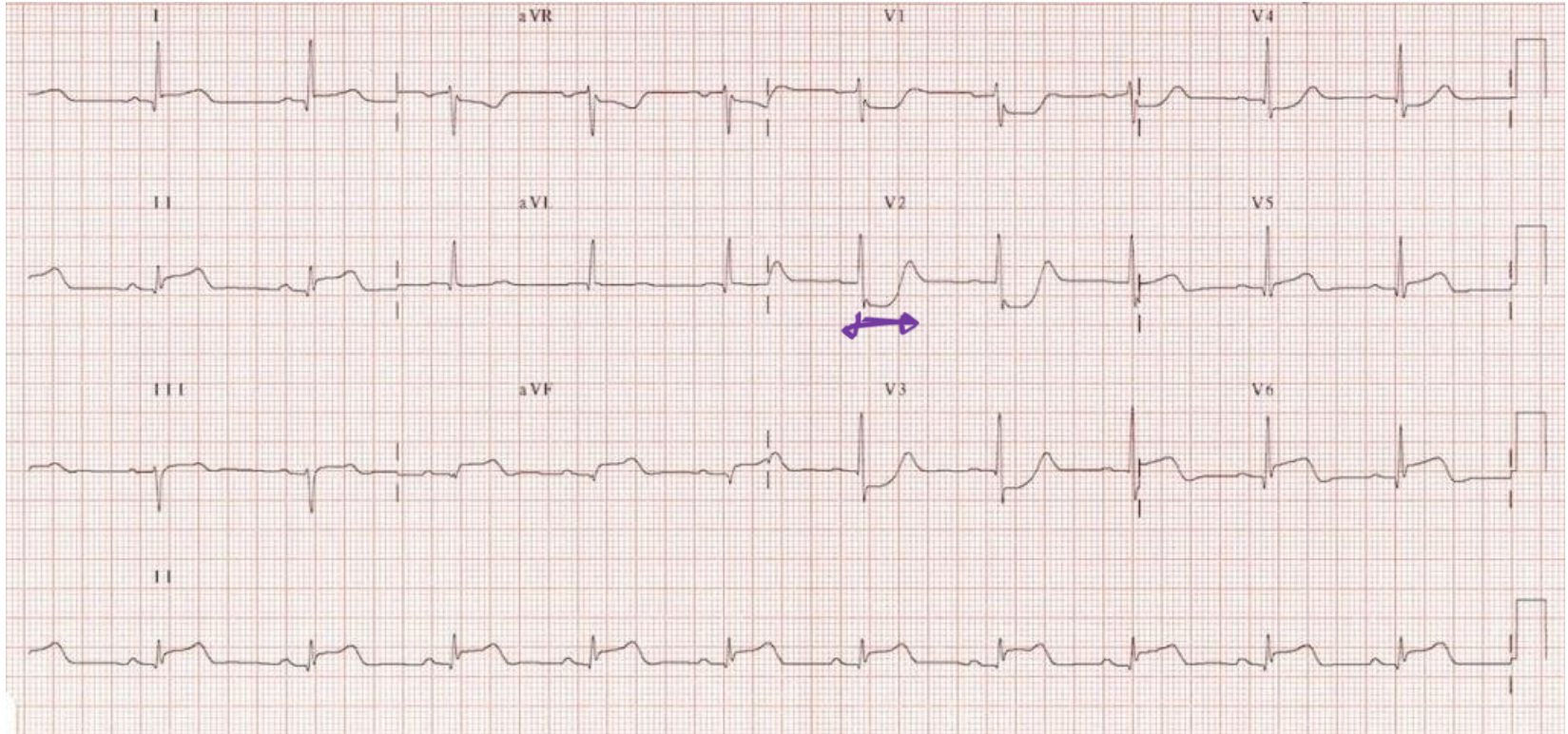
Inferior MI

Pathological Q wave
Easter Men 1 square
taller than 2 squares



Always when inferior MI → You have to check the posterior leads of the Rt ventricle

Posterior MI



Inferolateral STEMI. Posterior extension is suggested by:

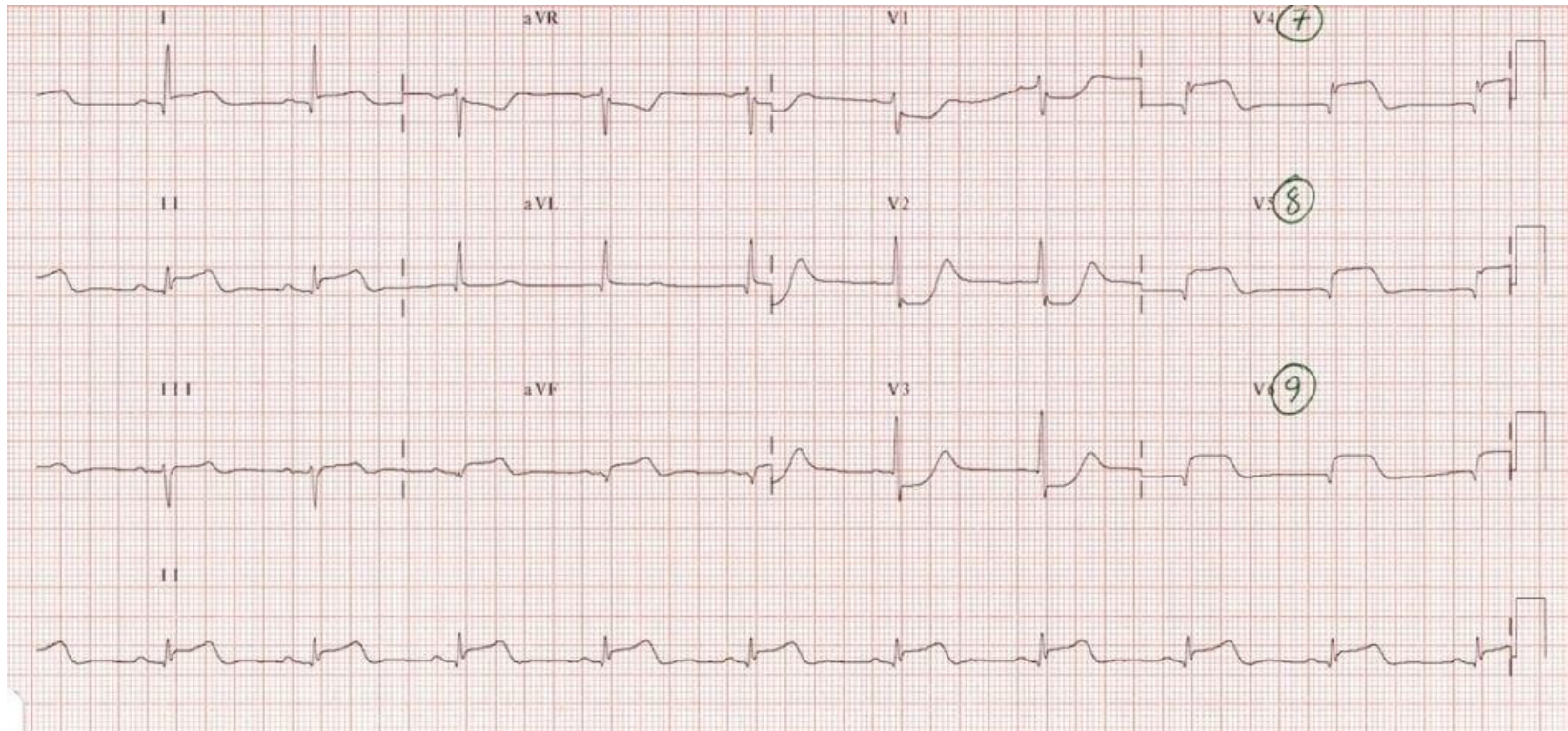
Horizontal ST depression in V1-3

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

Dominant R wave ($R/S \text{ ratio} > 1$) in V2

Upright T waves in V2-3

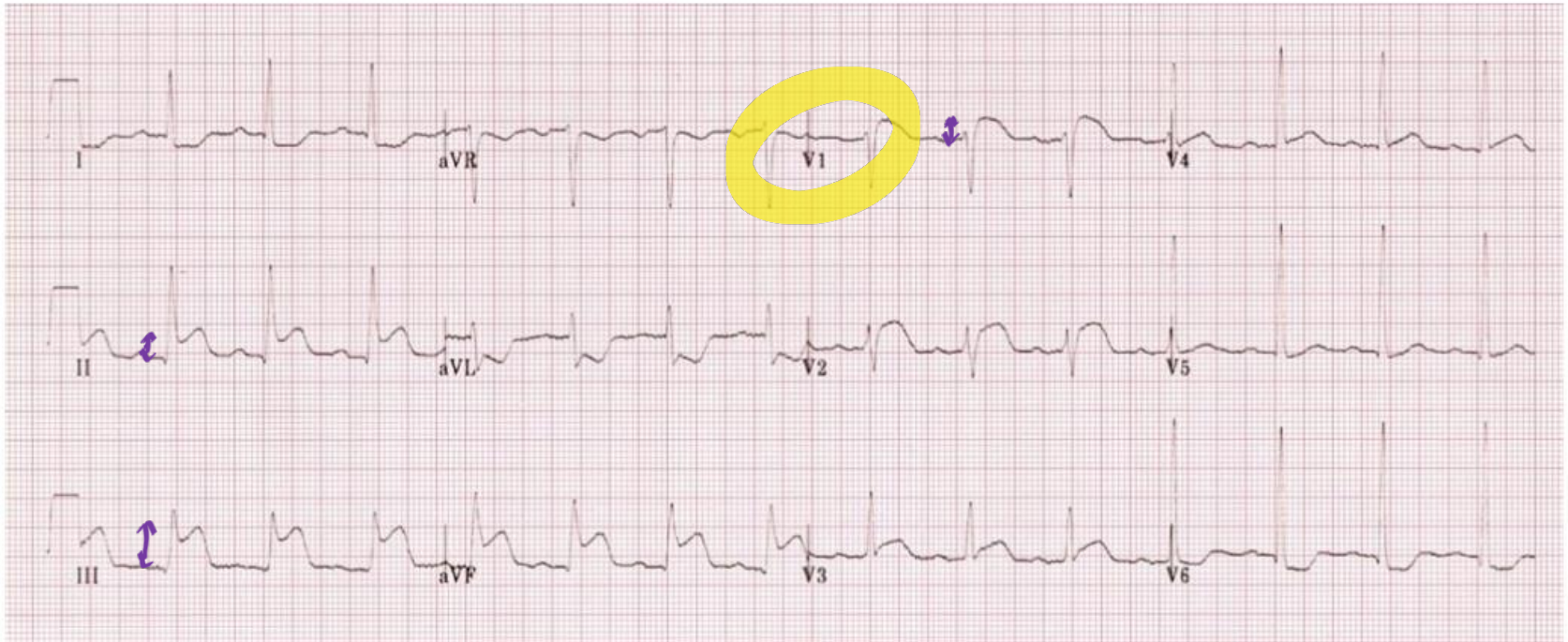
Posterior MI using posterior leads



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



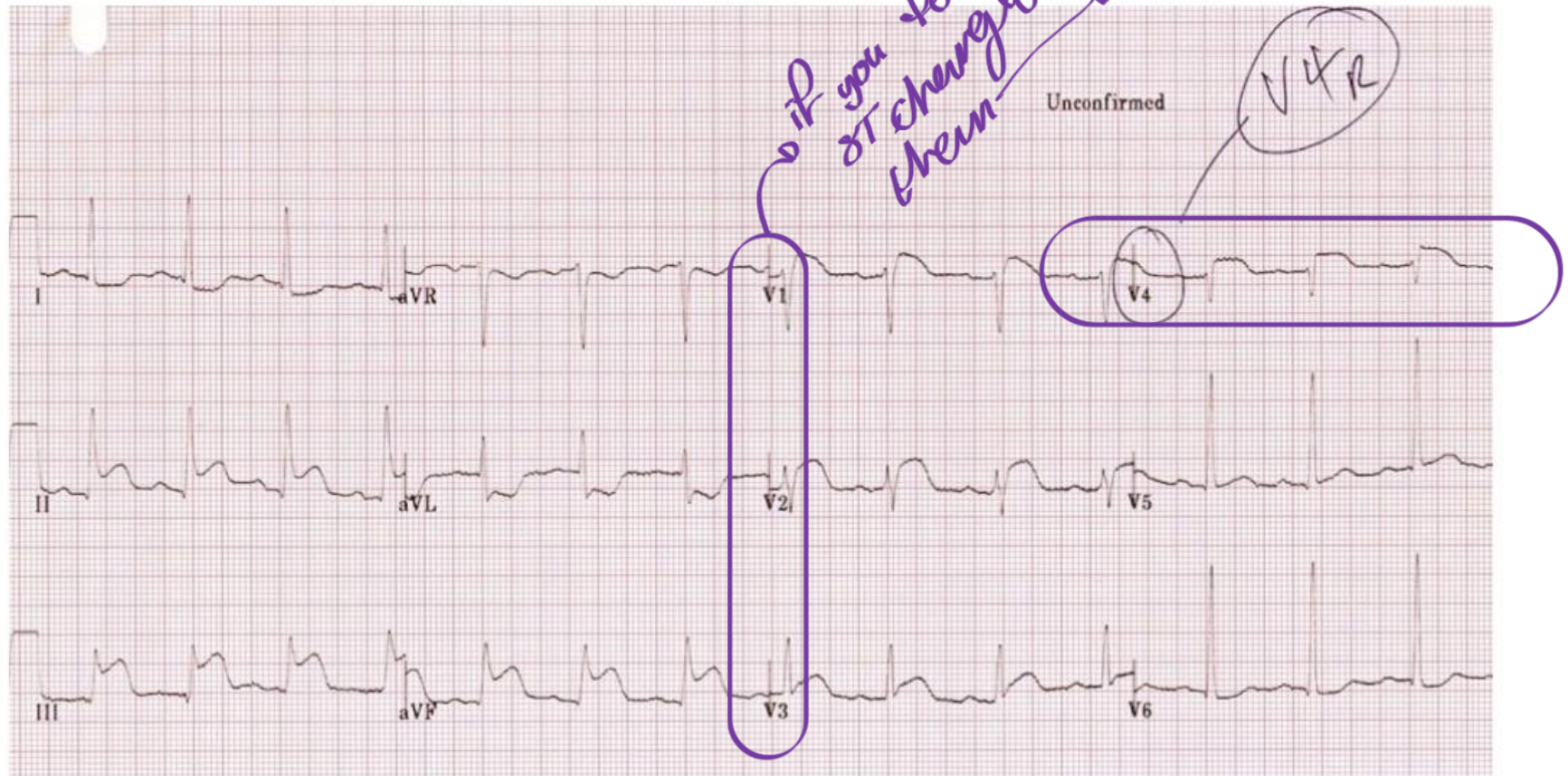
Inferior STEMI. Right ventricular infarction is suggested by:

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

Rt sided V4

Example 1b

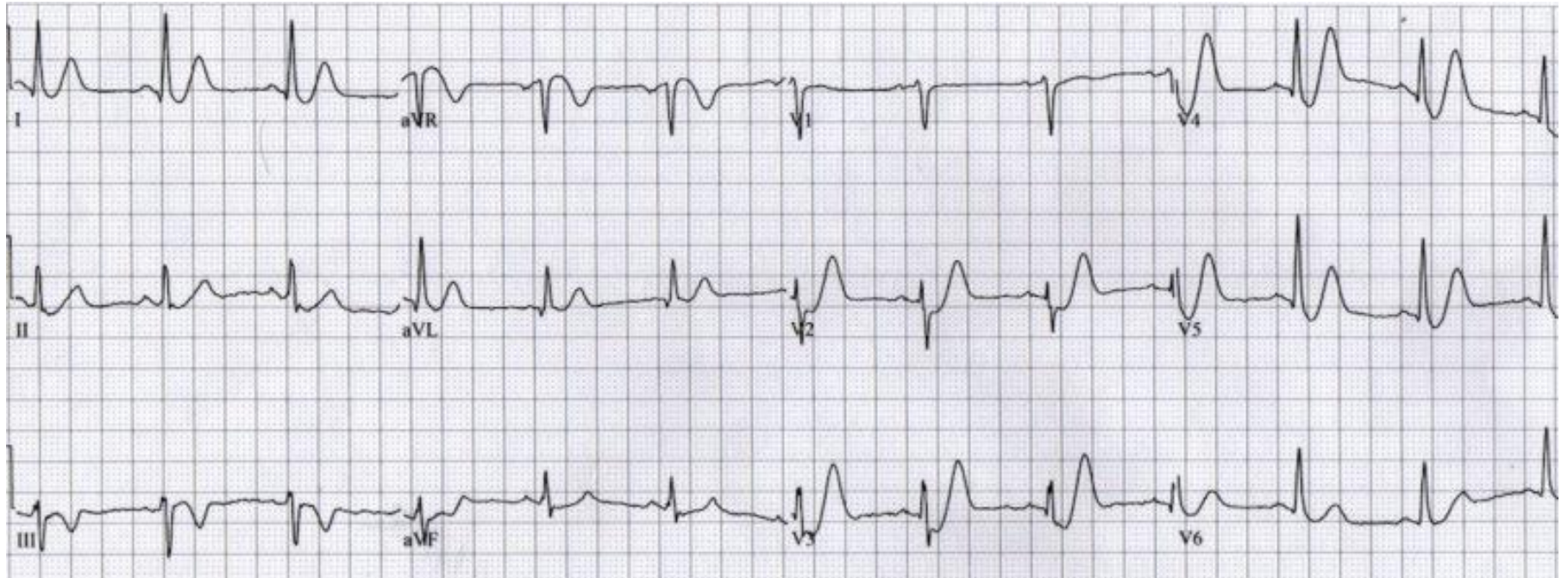
Repeat ECG of the same patient with V4R electrode position:



- There is ST elevation in V4R consistent with RV infarction

De Winter T Wave

happens [↳] before LAD occlusion



sky high T wave + ST depression in V₁/V₂/V₃

Note:

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

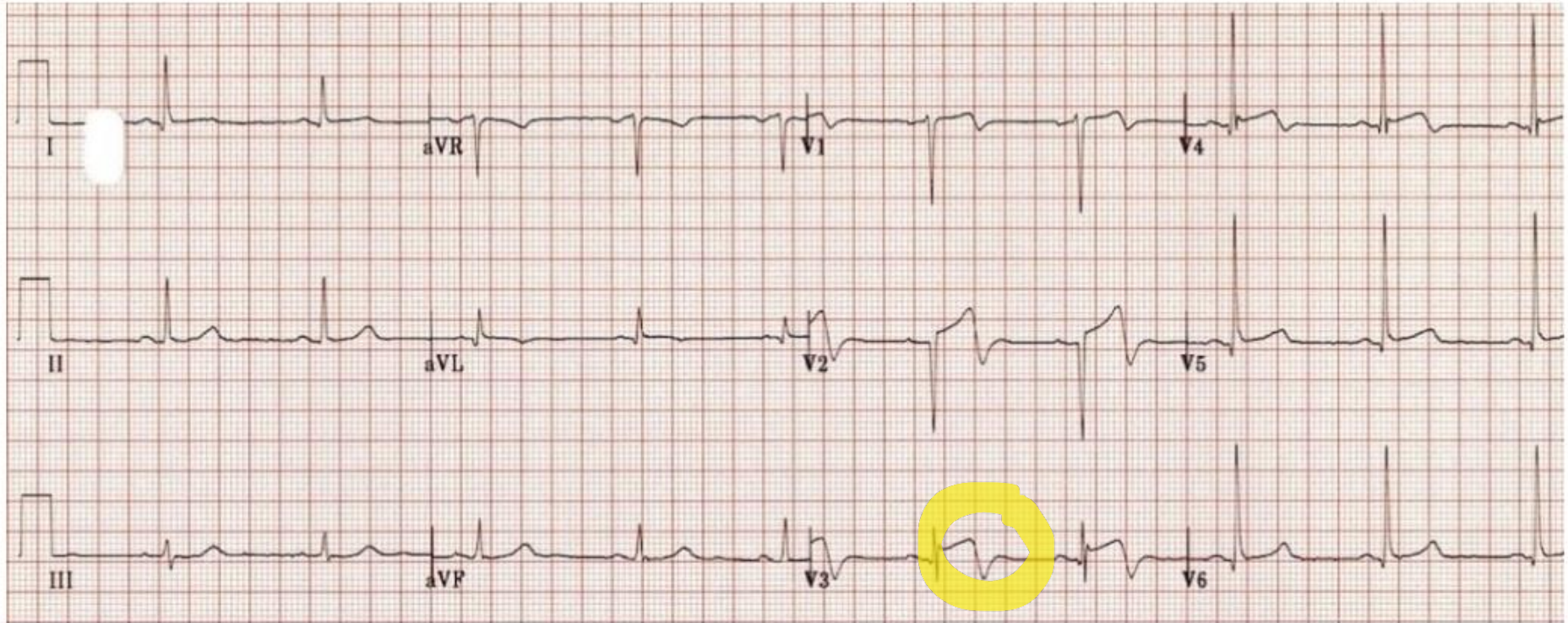
Diagnostic Criteria :

- 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.

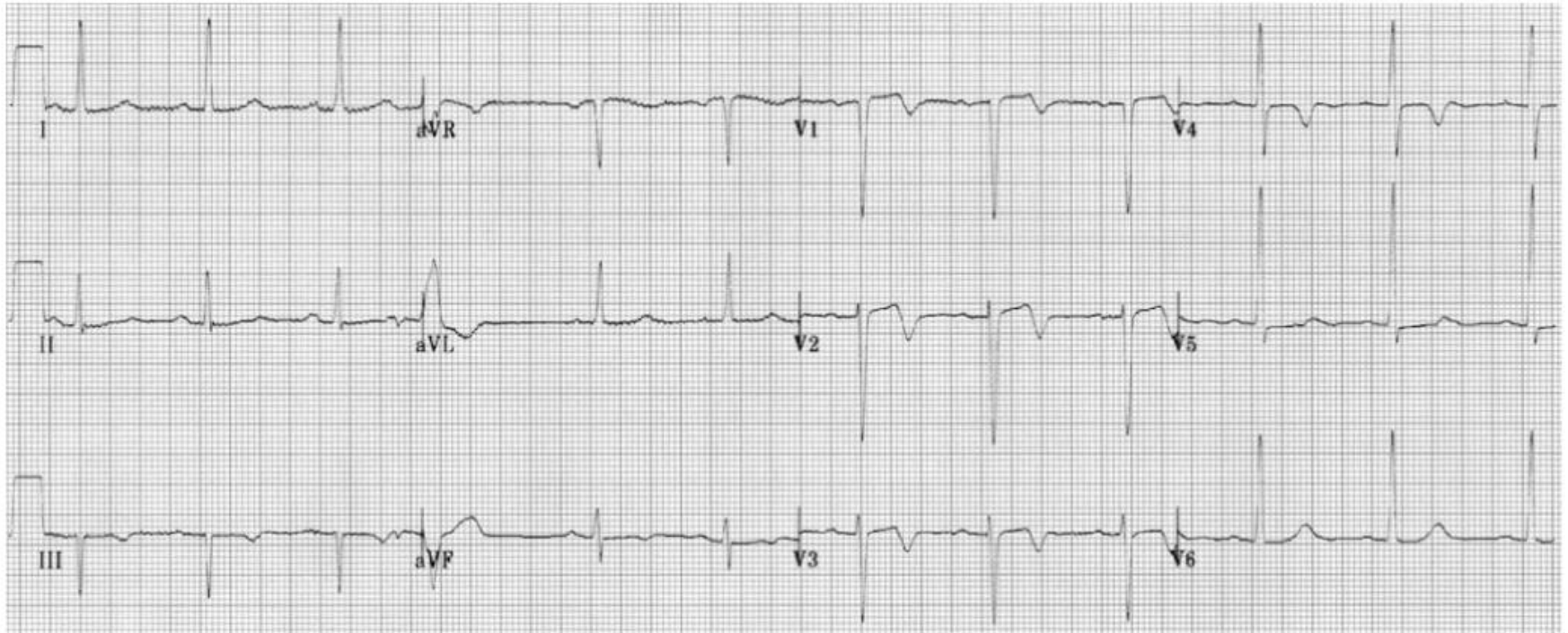
Example 1



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)

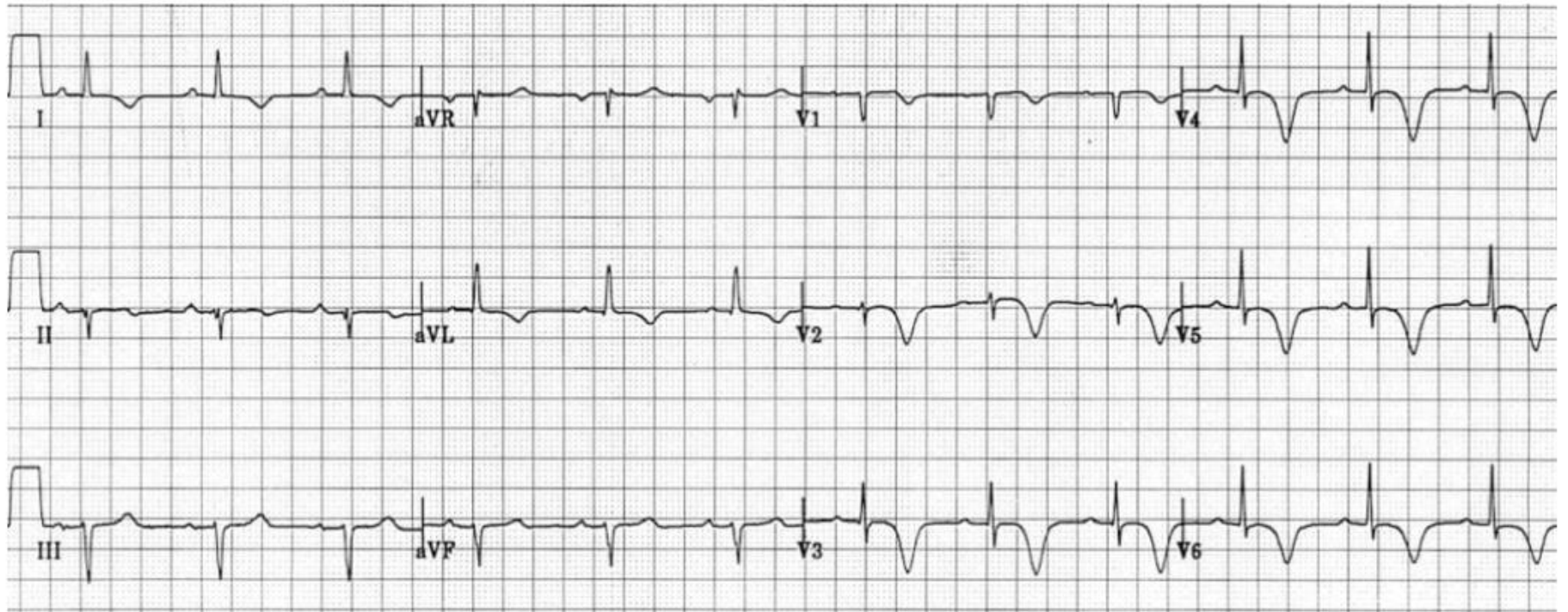
Example 2



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]). *, what we use in JUH → Timi score*

If NSTEMI is suspected

Antiplatelet and
antithrombin
therapy



Assess 6-month
mortality
(GRACE score)



PCI or medical
therapy alone

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

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

