



YEDİTEPE UNIVERSITY
FACULTY OF MEDICINE

NSTEMI-USAP

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Definition of MI and Pathophysiology



What is Myocardial Infarction?

- **Pathological definition**
 - Myocardial cell death caused by prolonged myocardial ischaemia
 - Ischaemia → irreversible myocardial injury → myocyte death

What is Myocardial Infarction?

- **Clinical diagnosis**

- A diagnosis of MI requires:

- **Acute myocardial injury**

- +

- Evidence that the injury is caused by myocardial ischaemia**

- Clinical diagnosis is based on the integration of:

- **Clinical presentation + cardiac troponin + ECG + cardiac/coronary imaging**

- **No single finding alone establishes acute MI**

TROPONIN ELEVATION $\neq$ MI



Myocardial Injury vs Myocardial Infarction

- **cTn > 99th percentile URL**
→ **Myocardial injury**
- **Acute myocardial injury**
 - **Rise and/or fall in cTn**
with ≥ 1 value >99th percentile URL
- **Myocardial infarction**
 - **Acute myocardial injury**
+ **evidence of acute myocardial ischaemia**

Evidence of myocardial ischemia may include:

- Symptoms or other clinical evidence of acute ischaemia
- New ischaemic ECG changes
- New pathological Q waves
- Imaging evidence of acute coronary pathology
- New loss of viable myocardium
- New regional wall-motion abnormality consistent with ischaemia

Name:

ID

Age: 40

12-Lead 7

01 May 07

050107213625

Sex:

21:39:57

HR 77bpm

PR 0.162s

QT/QTc

P-QRS-T Axes:

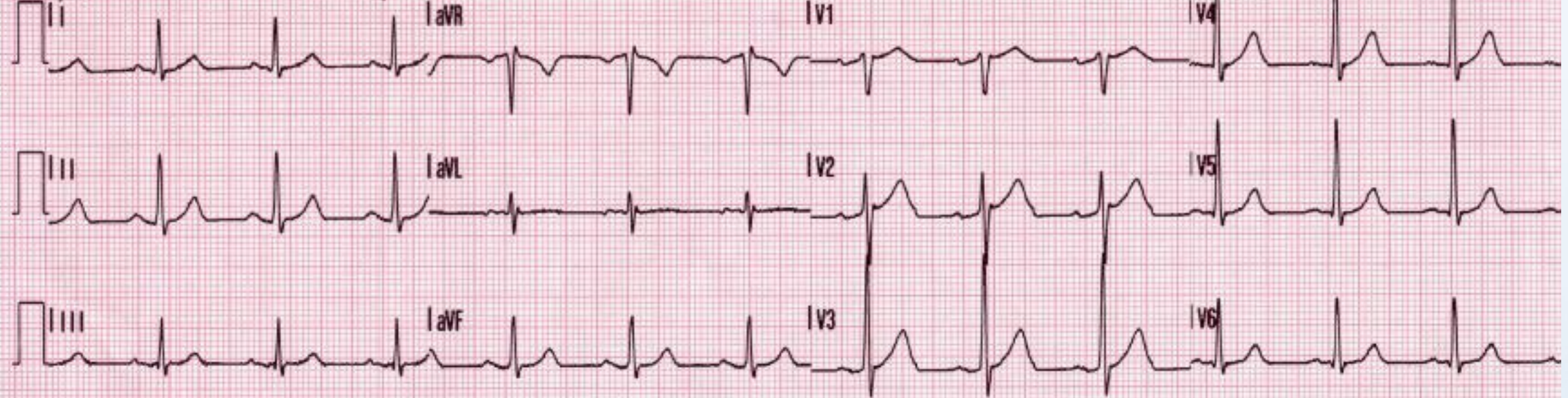
QRS 0.084s

0.360s/0.487s

48° 54° 49°

• Normal ECG ^{***}Unconfirmed^{***}

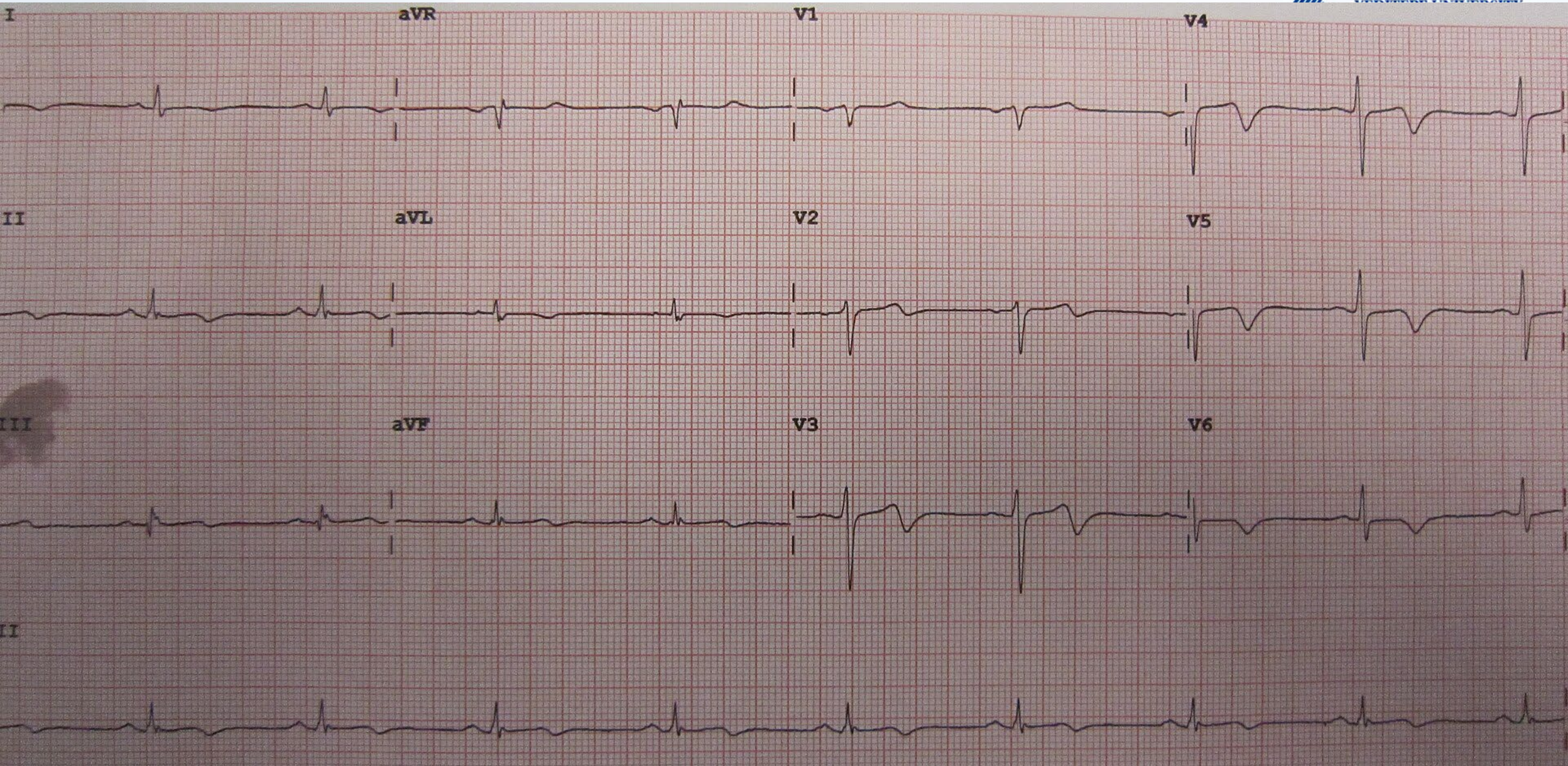
• Normal sinus rhythm



x1.0 .05-150Hz 25mm/sec

000 000 3011371-018 e3 7455574

MEDTRONIC PHYSIO-CONT



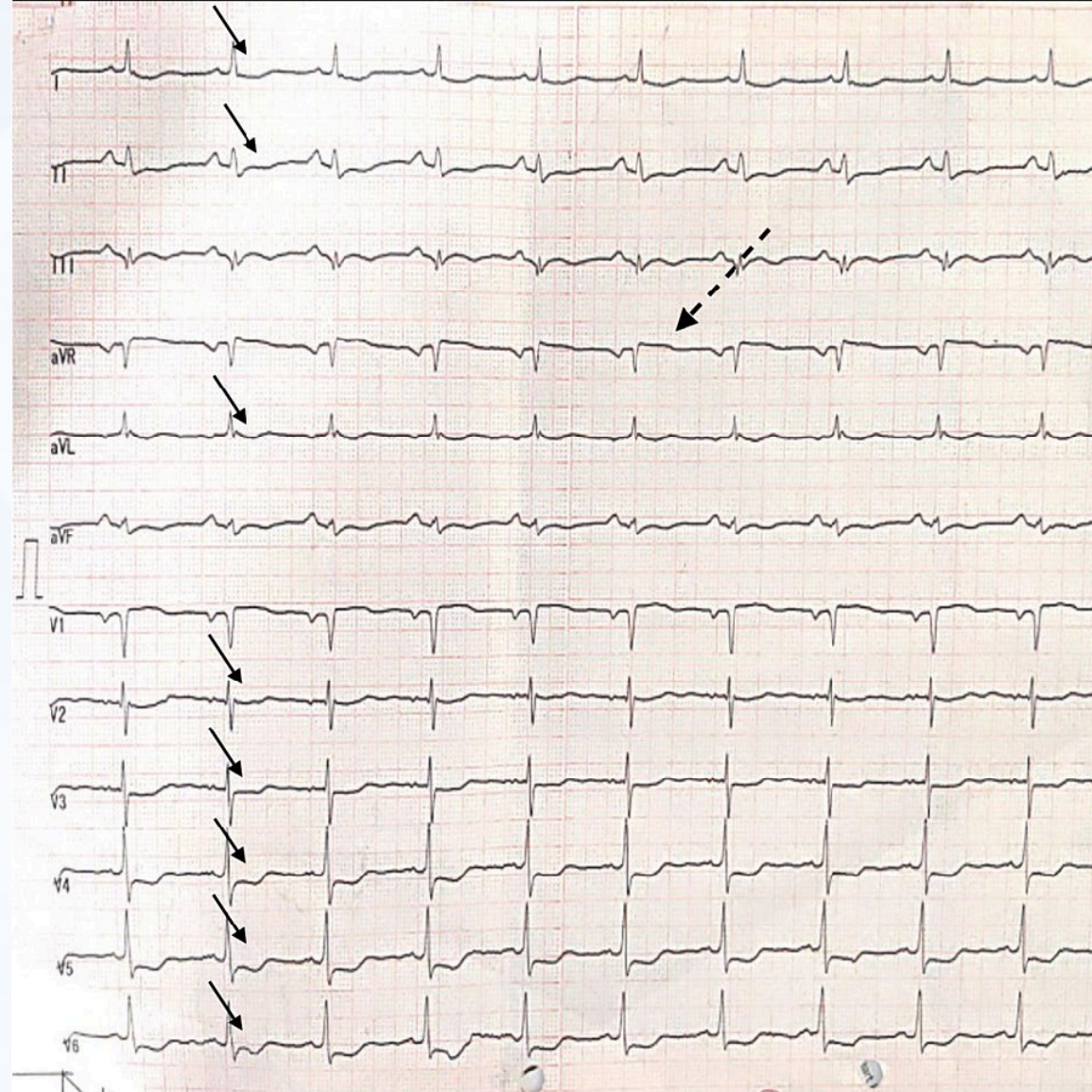


FIGURE 1: ST elevation in lead aVR and ST depression in leads V2-V6, leads I, II, and aVL

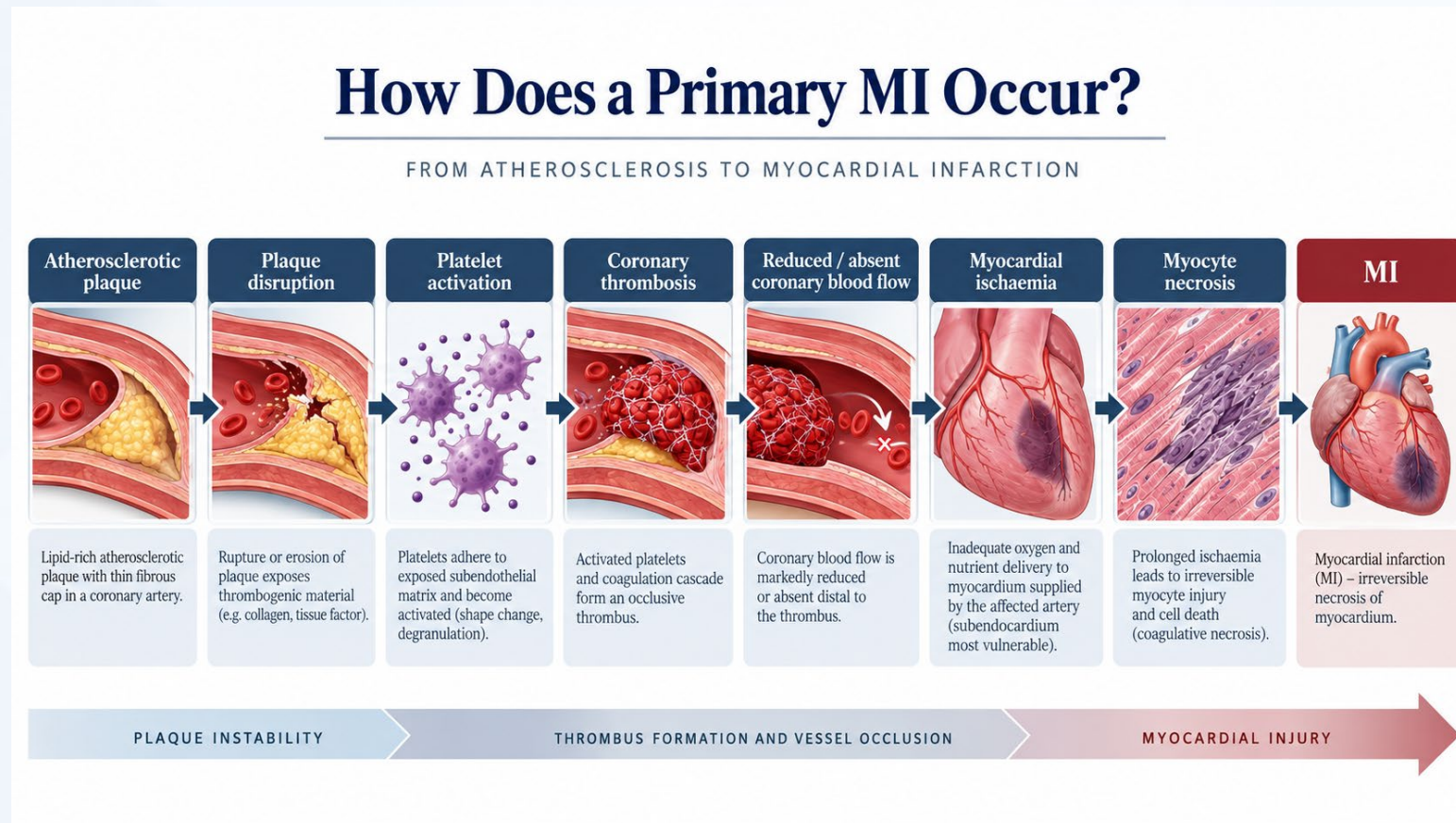
The dotted arrow shows ST elevation in lead aVR and the regular arrows show ST depression in leads V3-V6, leads I, II, and aVL.

Troponin tells us that the myocardium is injured.
It does not tell us why.



How Does a Primary MI Occur?

- Most commonly:



How Does a Primary MI Occur?

- **Atherothrombosis is the predominant mechanism**
 - However, primary MI may also result from other acute coronary pathologies:
 - **Spontaneous coronary artery dissection**
 - **Coronary embolism**
 - **Coronary vasospasm**

Why are platelets important?

- **Plaque disruption → platelet activation → thrombus formation**
 - This is the pathophysiological basis for **antiplatelet therapy** in ACS.



MI IS NOT A SINGLE PATHOPHYSIOLOGICAL ENTITY

The Fifth Universal Definition replaces the previous numerical classification with **three clinical categories:**



1. PRIMARY MI

- Spontaneous MI caused by a **primary acute coronary pathology**
- Most commonly:
atherothrombosis

2. SECONDARY MI

- Acute myocardial injury and ischaemia caused by:
 - **myocardial O₂ supply–demand imbalance due to another acute condition**
 - Examples of mechanisms include:
 - **↓ O₂ supply**
 - Hypoxaemia
 - Anaemia
 - Hypotension
 - **↑ O₂ demand**
 - Tachycardia
 - Severe hypertension

3. PROCEDURE-RELATED MI

- MI occurring as a complication of a:
 - **Percutaneous cardiac procedure**
 - **Surgical cardiac procedure**

MI CAN BE CLASSIFIED IN TWO DIFFERENT WAYS

- **BY UNDERLYING MECHANISM**

- Primary MI
- Secondary MI
- Procedure-related MI

- **BY PRESENTING ECG**

- STEMI
- NSTEMI

NSTE-ACS includes NSTEMI and unstable angina.

ECG classification

- Patients with suspected ACS are initially classified according to the presenting ECG
- **Persistent ST-segment elevation**
 - **STEMI pathway**
- **No persistent ST-segment elevation**
 - **NSTE-ACS pathway**
 - hs-cTn + clinical assessment
 - **NSTEMI or unstable angina**
- This classification is useful for **initial triage and acute management**, but:

STEMI / NSTEMI does NOT define the underlying mechanism of MI.



What does NSTEMI tell us?

- **NSTEMI describes the presenting ECG phenotype, not the underlying mechanism.**
- **Acute coronary occlusion may occur without ST-segment elevation on the standard 12-lead ECG.**
- **ECG findings must therefore be integrated with clinical assessment and cardiac troponin.**

- Fifth UDMI 2026; ESC ACS Guidelines 2023

A patient presents with chest pain but no persistent ST elevation.

How do we diagnose — or exclude — NSTEMI?





Diagnosis and Initial Management of NSTEMI



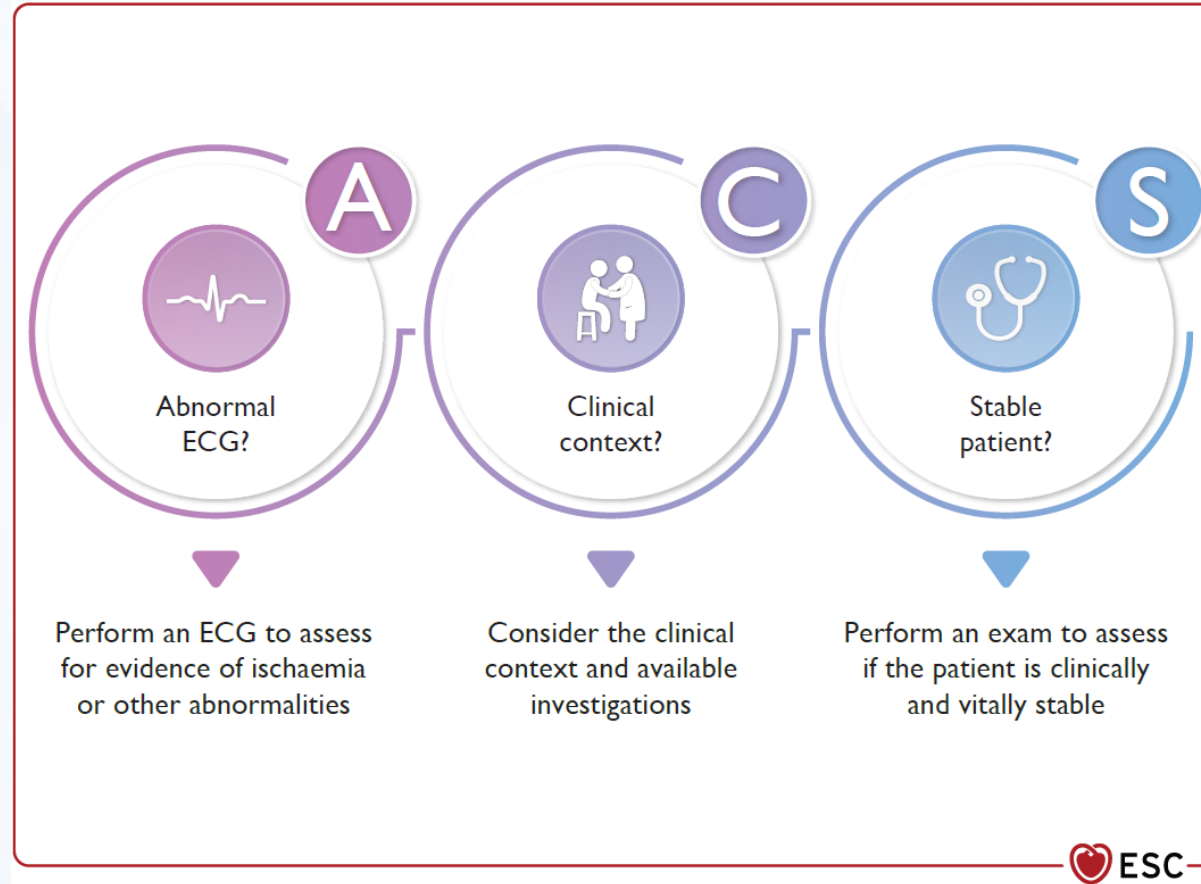
INITIAL ASSESSMENT OF SUSPECTED ACS

- **Diagnosis and initial risk assessment should integrate:**
- **Clinical history and symptoms**
- **Vital signs**
- **Physical examination**
- **12-lead ECG**
- **High-sensitivity cardiac troponin**

INITIAL ASSESSMENT OF SUSPECTED ACS

- **First medical contact**
 - Focused history + vital signs + physical examination + ECG should begin immediately.
 - 12-lead ECG: target <10 min
- **Physical examination should include:**
 - Major peripheral pulses
 - **Blood pressure in both arms**
 - Cardiac and pulmonary auscultation
 - Signs of heart failure or circulatory compromise

INITIAL ASSESSMENT OF SUSPECTED ACS



DO NOT SKIP THE PATIENT

- In suspected ACS, physical examination may:
- **Identify high-risk ACS features AND
Reveal an alternative life-threatening diagnosis**
- **Look for:**
 - **Haemodynamic stability**
 - Blood pressure
 - Heart rate
 - Oxygen saturation
 - **Circulation**
 - Major pulses
- **Blood pressure in both arms**
- **Heart & lungs**
- Murmurs
- Pulmonary congestion
- Signs of heart failure
- **Key message**
 - **ECG + troponin never replace history and physical examination.**

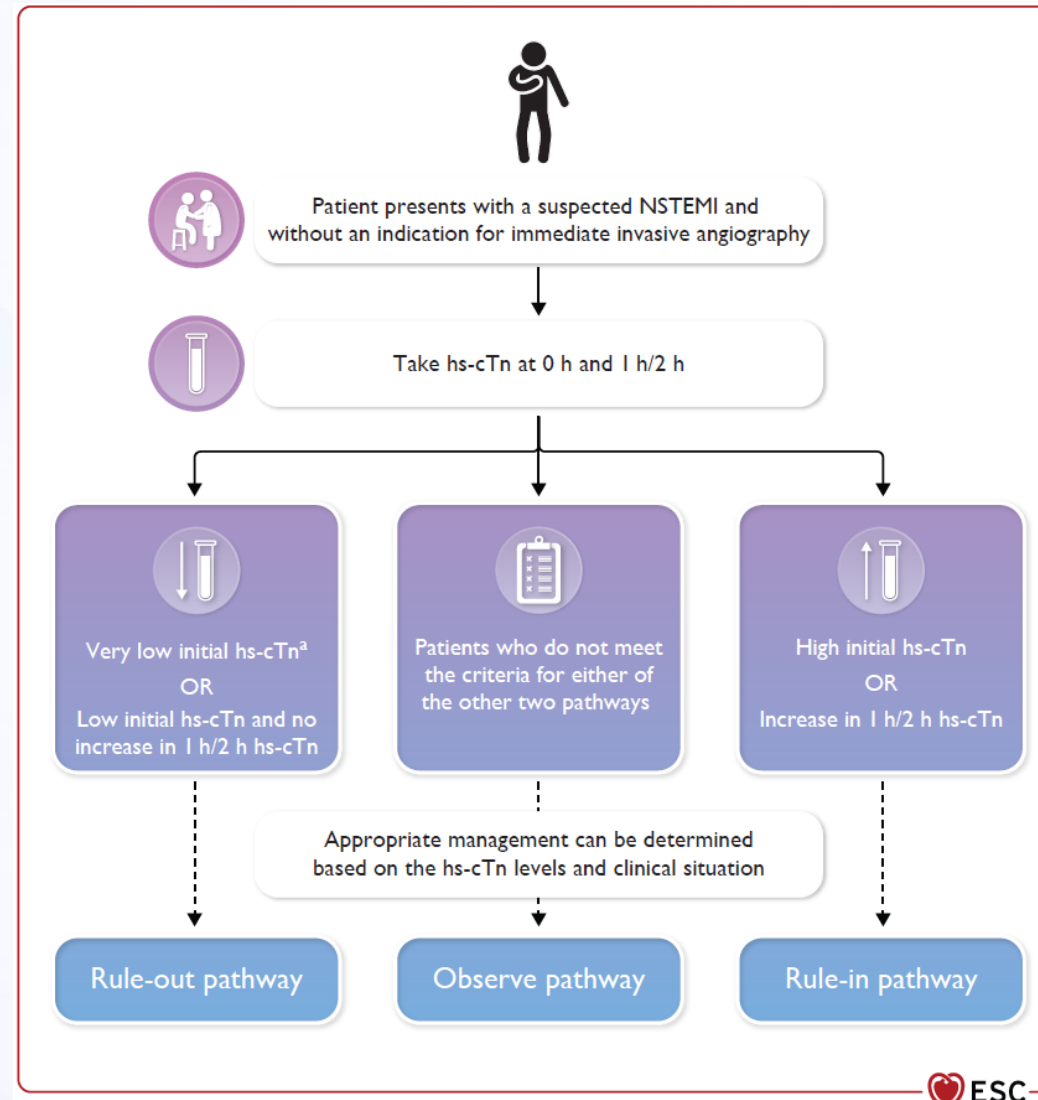
ECG IN NSTEMI-ACS

- **12-lead ECG is the first-line diagnostic test.**
- **Possible findings:**
 - **ST-segment depression**
 - T-wave inversion
 - Biphasic T waves
 - Transient ST-segment elevation
 - Other dynamic ST-T changes
- **But: The ECG may be normal in >1/3 of patients with NSTEMI-ACS**
- **Repeat ECG when:**
 - Symptoms recur
 - Symptoms persist
 - Diagnostic uncertainty remains
- **A normal ECG does NOT exclude NSTEMI-ACS.**

hs-cTn: ONE VALUE IS NOT THE WHOLE STORY

- **hs-cTn should be measured immediately in all patients with suspected ACS.**
- **Remember:**
 - **hs-cTn is a continuous variable**
 - Higher concentration
→ higher probability of MI
 - **Serial testing matters**
 - **Absolute change over time adds diagnostic information.**
- **Troponin concentration is also influenced by:**
 - Time from symptom onset
 - Age
 - Renal function
 - Sex
- **An initially non-elevated hs-cTn does NOT exclude an evolving NSTEMI.**

ESC RAPID RULE-IN / RULE-OUT ALGORITHM



Cut-offs are assay-specific — do not memorize a universal number.

RULE-OUT $\neq$ RULE-IN $\neq$ FINAL DIAGNOSIS

• RULE-OUT

- Very low initial hs-cTn
OR
Low initial hs-cTn + no significant change
- **Rule-out MI $\neq$ automatic discharge**
- Clinical context and ECG still matter.

• OBSERVE

- Does not fulfil rule-in or rule-out criteria
- $\rightarrow$ **Repeat hs-cTn at 3 h $\pm$ echocardiography**
- $\rightarrow$ Reassess the patient

• RULE-IN

- High initial hs-cTn
OR
Significant early rise
- $\rightarrow$ Hospital assessment
 $\rightarrow$ **Cardiology involvement**

The algorithm must always be integrated with clinical assessment and the 12-lead ECG.

- Rule-in PPV for MI $\approx 70-75\%$
- Rule-in does not mean “the computer has diagnosed Type 1 MI.”

WHEN SHOULD YOU ESCALATE?

- **Escalate immediately if:**
 - Haemodynamic instability / shock
 - Recurrent or refractory chest pain
 - Life-threatening arrhythmia
 - Mechanical complication
 - Heart failure clearly related to ACS
 - Recurrent dynamic ST/T changes, especially intermittent ST elevation
- **High-risk NSTEMI-ACS includes:**
 - **Rule-in / confirmed NSTEMI**
 - Dynamic ST/T changes
 - Transient ST elevation
 - GRACE score >140

SERIAL TROPONIN IS IMPORTANT — BUT...

- If the patient already has:
 - **Typical ischaemic presentation**
 - +
 - **Clear ischaemic ECG changes**
 - +
 - **Markedly elevated first hs-cTn / rule-in profile**
 - ↓
 - **CALL CARDIOLOGY NOW**
- **Continue serial hs-cTn measurement — but do not use the second result as an excuse to delay care.**
- Serial sampling continues. Clinical action does not have to wait.

DO NOT FORGET ASPIRIN

- **In ACS patients without contraindications:**
 - **ASPIRIN 150–300 mg PO LOADING DOSE**
- **In practice:**
 - **300 mg PO**
 - followed by:
 - **75–100 mg once daily**
- **Other immediate principles**
 - ECG monitoring when indicated
 - Treat ongoing symptoms and haemodynamic compromise
 - Antithrombotic therapy is a cornerstone of ACS treatment
 - Early cardiology involvement according to clinical risk

Failure to give indicated aspirin is not a trivial omission.



SUSPECTED NSTEMI-ACS

- **History + Clinical Context**
 - ↓
 - **Vitals + Physical Examination**
 - ↓
 - **12-lead ECG ≤10 min**
 - ↓
 - **hs-cTn at 0 h**
 - ↓
 - **Repeat ECG when needed + Serial hs-cTn**
 - ↓
 - **RULE-OUT — OBSERVE — RULE-IN**
 - ↓
 - **Diagnosis + Risk Assessment + Cardiology**
- **Never forget**
 - Patient stability
 - Alternative diagnoses
 - Dynamic ECG changes
 - Serial troponin
 - **ASA 300 mg when ACS is established and no contraindication exists**

The algorithm supports clinical judgement. It does not replace it.





Night Shift at Somewhere General Hospital ER

Six patients. Limited information. Your decisions matter.



SIMULATION RULES

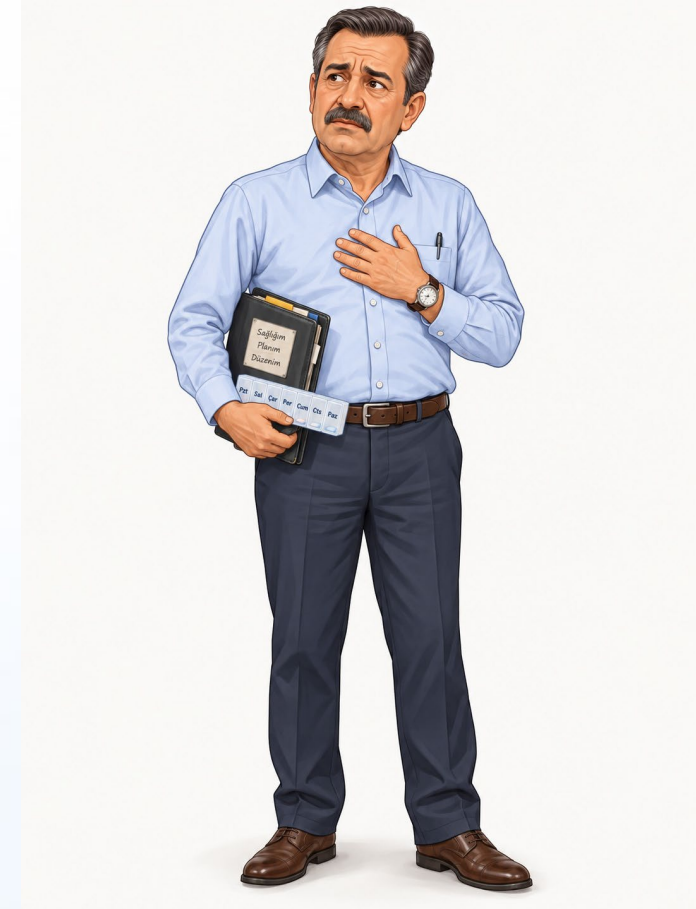
- You will receive **only the information you specifically request**.
- During Round 1, no vital sign or physical examination finding will be provided unless you ask for it. If you ask for “blood tests,” you must specify **which tests** you want.
- All six patients are managed **simultaneously** — not one by one.
- Record your decisions as you proceed:
working diagnosis – requested information/tests – immediate treatment – final decision
- Once a decision is made, **you cannot change it retrospectively** after new information appears.

SIMULATION RULES

- **ROUND 1 — HISTORY & PHYSICAL EXAMINATION | 10 MINUTES TOTAL**
 - You have **15 minutes for all six patients**.
Ask for any history, vital signs or physical examination findings you consider necessary.
When time is up, this round ends.
- **ROUND 2 — INVESTIGATIONS | 10 MINUTES TOTAL**
 - At the beginning of this round, you will receive **ECG findings and the first hs-cTn result for all six patients on a single slide**.
 - You then have **15 minutes for all six patients** to request any additional investigations you consider necessary.
- **Your case ends when you:**
 - 1. Make a working diagnosis
 - 2. Initiate immediate management
 - 3. Call the specialist
- **There will be no immediate feedback. Outcomes will be revealed at the end.**

Patient 1 — “By-the-Book” Kemal

- 56-year-old man presenting to the emergency department with chest pain at rest.



Patient 2 — “Sugar” Şükufe

- 74-year-old woman presenting to the emergency department with unusual fatigue and intermittent epigastric discomfort since this morning.



Patient 3 - “Yellow-Stache” Rıza

- 62-year-old man presenting to the emergency department with severe central chest pressure lasting for 90 minutes.



Patient 4 — “Necktie” Hakkı

- 61-year-old man presenting to the emergency department with chest pain associated with nausea.



Patient 5 — “Short-of-Breath” Necla

- 49-year-old woman presenting to the emergency department with shortness of breath and vague chest tightness.



Patient 6 — “Run-Down” Muzaffer

- 68-year-old man presenting to the emergency department with marked fatigue, shortness of breath on exertion, and vague chest pressure.





ROUND 1 — HISTORY & PHYSICAL EXAMINATION

15 MINUTES TOTAL



ROUND 2 — INVESTIGATIONS

15 MINUTES TOTAL



ROUND 2 — INVESTIGATIONS

hs-cTnT 99th percentile URL: 14 ng/L

Patient	ECG	First hs-cTnT
1. “By-the-Book” Kemal	Sinus rhythm, 72 bpm. No significant acute ST-T changes.	8 ng/L — 0.57× URL
2. “Sugar” Şükufe	Sinus rhythm, 78 bpm. 0.5–1 mm horizontal ST depression in V4–V6.	9 ng/L — 0.64× URL
3. “Yellow-Stache” Rıza	Sinus rhythm, 88 bpm. 1–2 mm horizontal ST depression in I, aVL, V4–V6.	126 ng/L — 9× URL
4. “Necktie” Hakkı	Sinus rhythm, 92 bpm. Non-specific ST-T changes; no clear ischaemic pattern.	7 ng/L — 0.50× URL
5. “Short-of-Breath” Necla	Sinus tachycardia, 112 bpm. Mild T-wave inversion in V1–V3.	8 ng/L — 0.57× URL
6. “Run-Down” Muzaffer	Sinus tachycardia, 108 bpm. ~1 mm horizontal ST depression in I, aVL, V4–V6.	28 ng/L — 2.0× URL



LONG-TERM MANAGEMENT AFTER NSTEMI

The event is over. The disease is not.

Discharge Is Not the End

- **Antiplatelet Therapy**
Prevent recurrent thrombosis
- **Lipid Lowering**
Treat the underlying atherosclerosis
- **Lifestyle Modification**
Smoking • Diet • Exercise
- **Cardiac Rehabilitation**
Exercise • Education • Adherence
- We treated the event. We did not cure atherosclerosis.

DAPT After ACS

- **Default strategy**
 - Aspirin + P2Y₁₂ inhibitor → 12 months
- **Aspirin**
 - 75–100 mg once daily

DAPT After ACS

P2Y₁₂ Inhibitors

Drug	Loading Dose	Maintenance Dose
Ticagrelor	180 mg	90 mg twice daily
Prasugrel	60 mg	10 mg once daily
Clopidogrel	300–600 mg	75 mg once daily

12 months is the default — not “one size fits all.”
Bleeding risk $\leftrightarrow$ Ischaemic risk



“Doctor, Can I Stop My Antiplatelet?”

- **Six months after ACS + PCI...**
- Patient needs:
 - **Dental extraction**
or
Colonoscopy
- **STOP IT?**

Before answering, ask:

- **Why** is the patient taking it?
- **When** was the ACS / PCI?
- Was a **stent** implanted?
- Which antiplatelet drug(s)?
- What is the **bleeding risk of the procedure?**
- What is the patient's **ischaemic / stent-thrombosis risk?**



Antiplatelet interruption is a clinical decision — not a routine instruction.



Treat the Atherosclerosis

- **High-dose statin**
 - Start or continue as early as possible
- **LDL-C <55 mg/dL AND $\geq 50\%$ reduction from baseline**
 - **High-dose statin**
 - ↓
 - Not at goal after 4–6 weeks**
 - ↓
 - + Ezetimibe**
 - ↓
 - Still not at goal**
 - ↓
 - + PCSK9 inhibitor**

The Best-Treated MI...

- ...is the second MI that never happens.
 - **Stop smoking**
 - **Mediterranean-style diet**
 - **Regular aerobic + resistance exercise**
 - **Reduce sedentary time**
 - **Cardiac rehabilitation**
 - **Medication adherence**

Additional medications

- **If LVEF $\leq 40\%$**
- **Beta-blocker**
- **ACE inhibitor if**
- HF • LVEF $\leq 40\%$ • Diabetes • Hypertension • CKD
- **MRA if**
- LVEF $\leq 40\%$ + HF or Diabetes

SO... WHAT HAPPENED TO YOUR SIX PATIENTS?



Five correct decisions cannot undo one critical mistake.



In the world, the most
genuine guide for everything,
civilization, life, and success
is knowledge, science.
Seeking a guide outside of
knowledge and science is
negligence, ignorance, and
deviation.

H. Atatürk

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Thank you for listening.