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REVIEW ARTICLE

THE DECEPTIVE PRESENTATION: DIAGNOSING STEMI BY SERIAL ECG IN A PATIENT WITH DYSPNEA AND SWEATING

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ABSTRACT

Background: Prompt diagnosis of acute ST-elevation myocardial infarction (STEMI) is crucial, but atypical presentations can delay recognition. This case highlights a 60-year-old male smoker presenting with dyspnea and diaphoresis, in whom serial electrocardiograms (ECGs) were essential for diagnosis. The electrocardiogram (ECG) is a fundamental tool for the rapid diagnosis of acute myocardial infarction (AMI). However, the initial ECG in patients experiencing AMI can be non-diagnostic, posing a significant challenge for timely intervention. Subsequent serial ECG recording over a short period revealed the progressive development of characteristic ST- segment elevation, ultimately leading to the diagnosis of ST- elevation myocardial infarction (STEMI). We discuss the timing of ECG changes, the role of bedside echocardiography, the significance of initial negative troponin, the risk of being an "apparently healthy" older individual, the impact of smoking with non-diagnostic ECGs, the diagnostic accuracy of ER physician-performed echocardiography, and the criteria for urgent catheterization in non-STEMI presentations. **Case Presentation:** A 60-year-old male smoker with no known comorbidities presented to the emergency department (ED) at 02:50 am on March 20, 2025, with a one-hour history of sudden onset shortness of breath and diaphoresis. His initial 12-lead ECG (02:50 am) was unremarkable, and the baseline troponin T was negative. Patient was given Bipap support to overcome the respiratory distress. No calf pain and intermittent claudication history. At 03:00 am, the patient developed chest discomfort. A second ECG showed subtle changes. By 03:42 am, a third ECG revealed a marked ST- segment elevation ("tombstone" pattern) in the same leads. Patient was shifted to Cathlab urgently. He underwent successful percutaneous coronary intervention (PCI) but developed cardiogenic shock in the cardiac care unit (CCU) and died. **Conclusion:** This case emphasizes that STEMI can occur with atypical symptoms and a non- diagnostic initial ECG. Serial ECGs are vital in such presentations. We address critical questions regarding the timing of ECG changes, the role of bedside echocardiography, the interpretation of early negative troponin, the risk in "apparently healthy" older smokers, the significance of smoking with non-diagnostic ECGs, the accuracy of ER-performed echocardiography, and the need for urgent catheterization in evolving non-STEMI cases.

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INTRODUCTION

Acute myocardial infarction (AMI) remains a major cause of mortality. While typical STEMI presents with chest pain and ST-segment elevation on ECG, atypical presentations are not uncommon, leading to diagnostic challenges. This case illustrates such a scenario and explores key diagnostic and management dilemmas.

Case Presentation: A 60-year-old male smoker with no known comorbidities presented to the emergency department (ED) at 02:50 am on March 20, 2025, with a one-hour history of sudden onset shortness of breath and profuse diaphoresis. He denied chest pain at this time.

Vitals on arrival: Pulse Rate: 137beats per minute Respiratory Rate: 30 cycles per minute Temperature: 96.8 F Blood Pressure: 160/100 mmHg Oxygen saturation: 85 % @ Room Air Random blood sugar: 153 mg/dl

ON PHYSICAL EXAMINATION: Conscious and Oriented to time, place and person GCS: E4 V5 M6 (15/15) CVS: S1 and S2 heard, no murmur, no added sounds

CHEST: Bilateral equal air entry, left side: wheeze and right side crepts. PER ABDOMEN: Soft, non-tender, bowel sound present.

Lab investigations

EDTA whole blood

Hb: 13.83 g/dL
TC: 13.83 10^9/L
Platelet: 236 10^3/ microL Serum sample:
S. Urea: 22 mg/dL
S. Creatinine: 1.01 mg/dL Sodium: 137g/mol Potassium: 3.7g/mol
Serology: HIV 1 and 2 : NON REACTIVE HBsAG: NON REACTIVE **HCV:** NON REACTIVE
Blood group and Rh typing: B Positive
Chest Xray: Apparent cardiomegaly with aortic arch unfolding seen. Bilateral lung lower lobe show few fluffy opacities ? cardiogenic pulmonary ? positional

The initial 12-lead ECG, recorded at 02:50 am, showed normal sinus rhythm without ST-segment elevation or depression, T-wave inversions, or other acute ischemic changes. Initial troponin T was negative. At 03:00 am, the patient reported the onset of chest discomfort. A second 12-lead ECG, performed at this time, revealed subtle changes.

By 03:42 am, a third 12-lead ECG demonstrated marked ST-segment elevation in the anterior leads, with a "tombstone" appearance, consistent with a large anterior STEMI. The patient was immediately transferred to the cardiac catheterization laboratory. Coronary angiography revealed

LMCA: Distal 50 % stenosis LAD: Proximal total occlusion

LCX: Non dominant, minor stenosis RCA: Dominant, mild **CTO**

Impression: Double Vessel Coronary Artery Disease (DVCAD) Primary PCI to LAD done

LAD-3.5/20 S Cruz DES LMCA to LAD- 4/24 Cruz DES POT done with 4.5/8mm NC Balloon.

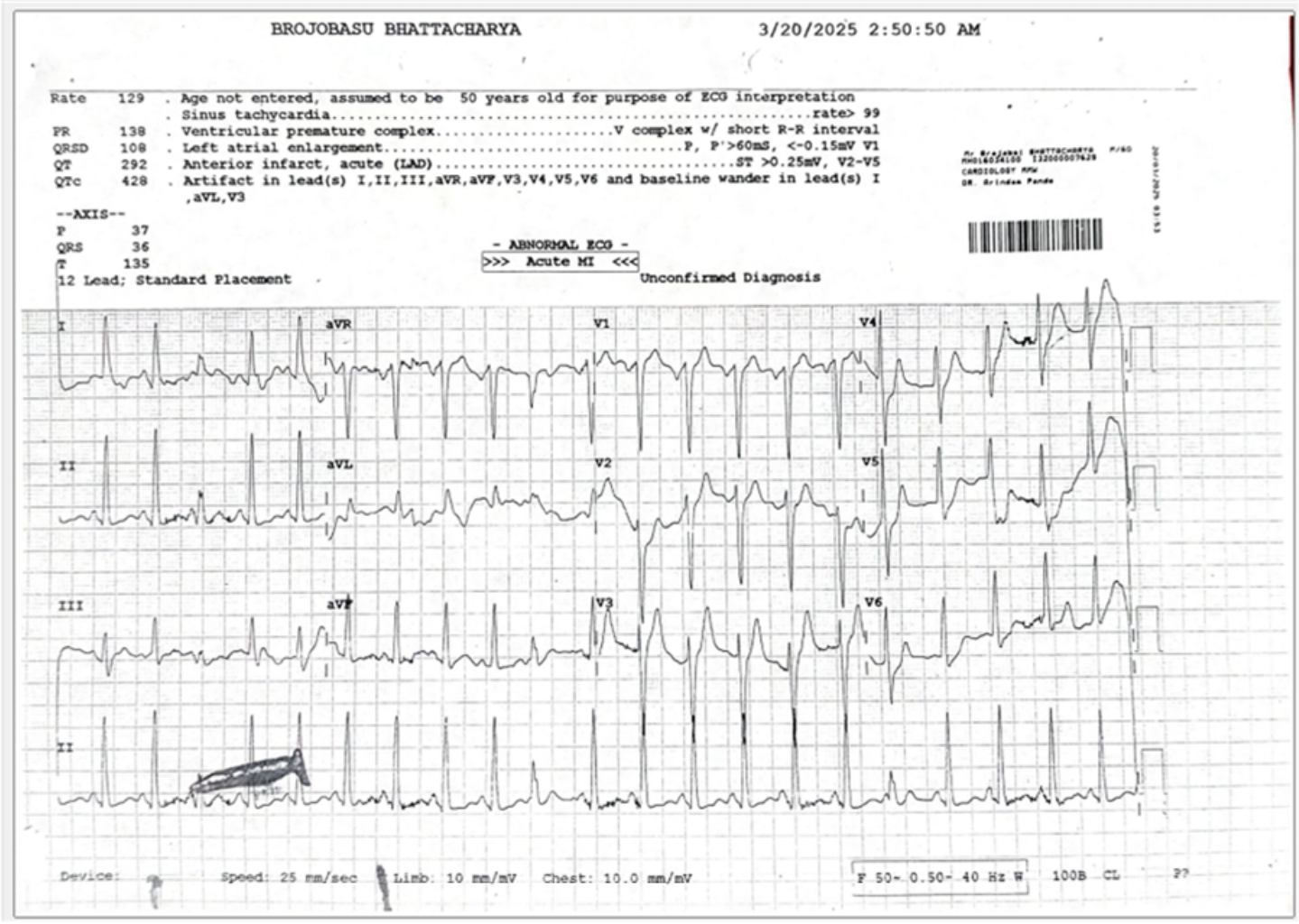
Following the procedure, the patient was transferred to the cardiac care unit (CCU) for close monitoring. However, within a few hours, he developed sudden onset of breathing difficulties, a decline in Glasgow Coma Scale (GCS) below 8, and hypotension. He progressed to cardiogenic shock, requiring intubation and vasopressor support. Despite aggressive medical management, the patient developed cardiac arrest and could not be resuscitated. He was pronounced dead later that day.

DISCUSSION

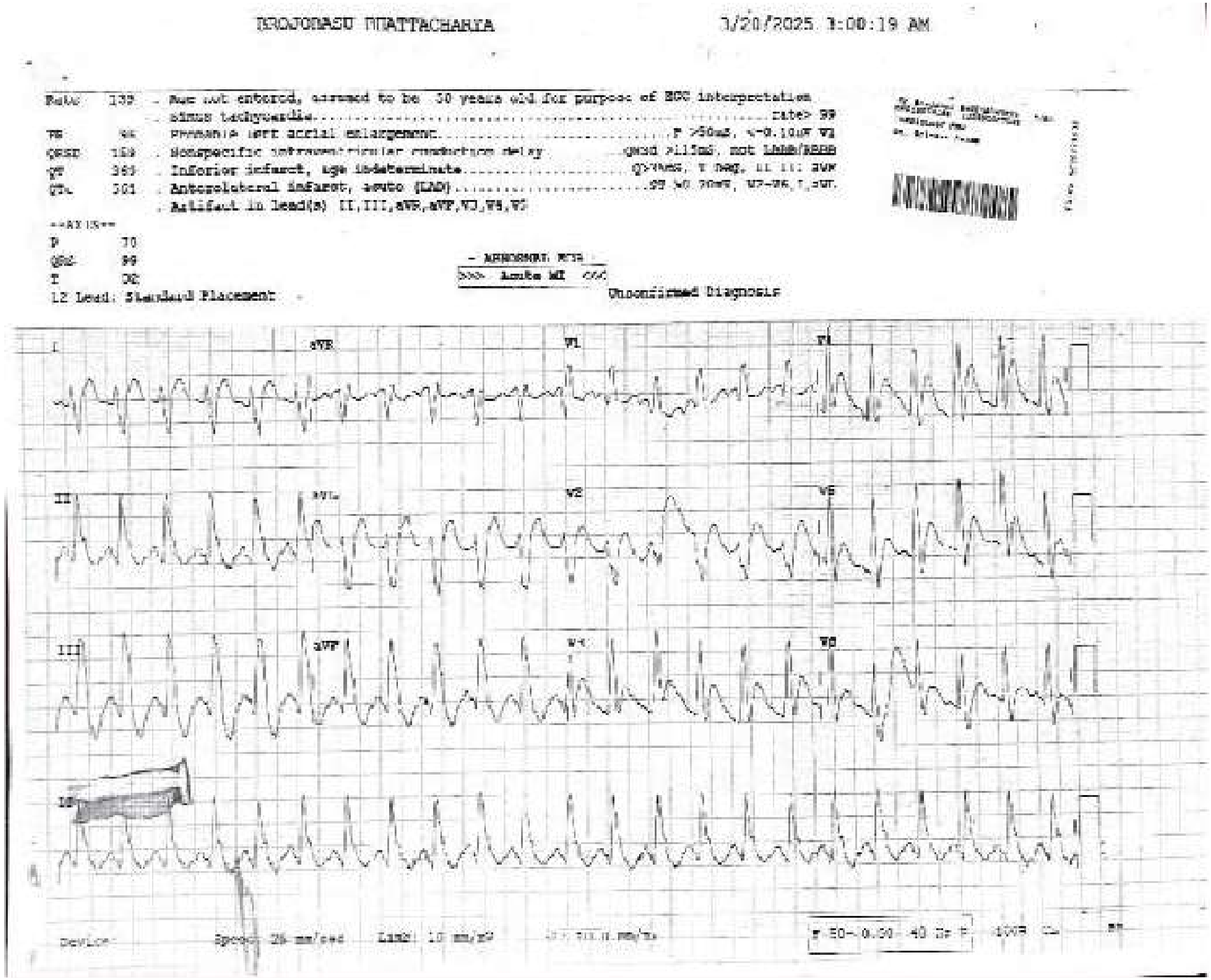
The case of this 60 years old male, who came to Emergency department on 20/3/2025 at 2:50 am with the atypical chest discomfort and an initially non diagnostic electrocardiogram (done at 2:50am), powerfully illustrates the critical importance of serial ECG monitoring in the early diagnosis of acute myocardial infarction (AMI). Despite the absence of immediate ischemic changes on the first ECG, the persistence of symptoms prompted a prudent clinical approach involving repeat electrocardiographic assessments. The subsequent ECG obtained approximately 10 minutes later (3:00am) revealed subtle but evolving changes that raised suspicion for underlying myocardial ischemia. This highlights a crucial point: early in the course of AMI, the ECG may not display the classic findings of ST segment elevation. The dynamic nature of thrombus formation and intermittent vessel occlusion can lead to fluctuating or initially absent ECG abnormalities. The decision to obtain a third ECG around 3:42 am, proved pivotal. This ECG demonstrated the clear evolution of ST- segment elevation meeting the criteria for STEMI protocol. This emphasizes that relying solely on a single, early ECG can be misleading and repeated assessments are essential in patients with ongoing or suspicious symptoms. The delay in diagnosis and subsequent reperfusion therapy had significant detrimental consequences. The rapid transfer to the catheterization laboratory (4:28am) for percutaneous coronary intervention (PCI) was appropriate next step following the diagnosis of STEMI. Patient was shifted to critical care unit. Post PCI patient was in critical care where he developed shortness of breath and decrease oxygen saturation, patient was given bag mask given with 100% oxygen and was intubated with 7.5 ETT tube (8:42 am) and was put on ventilator. Later patient progressed to cardiogenic shock necessitating inotropes. This underscore the severity of the underlying myocardial damage and the potential for significant complications even with guideline based treatment. The unfortunate subsequent development of cardiac arrest, CPR was started as per ACLS protocol and injection adrenaline was given every 3-4 minutes. 30 minutes of CPR was done but unfortunately Return of Spontaneous Circulation (ROSC) was not achieved. The patient's demise (9:10 am).

This case raises several important questions:

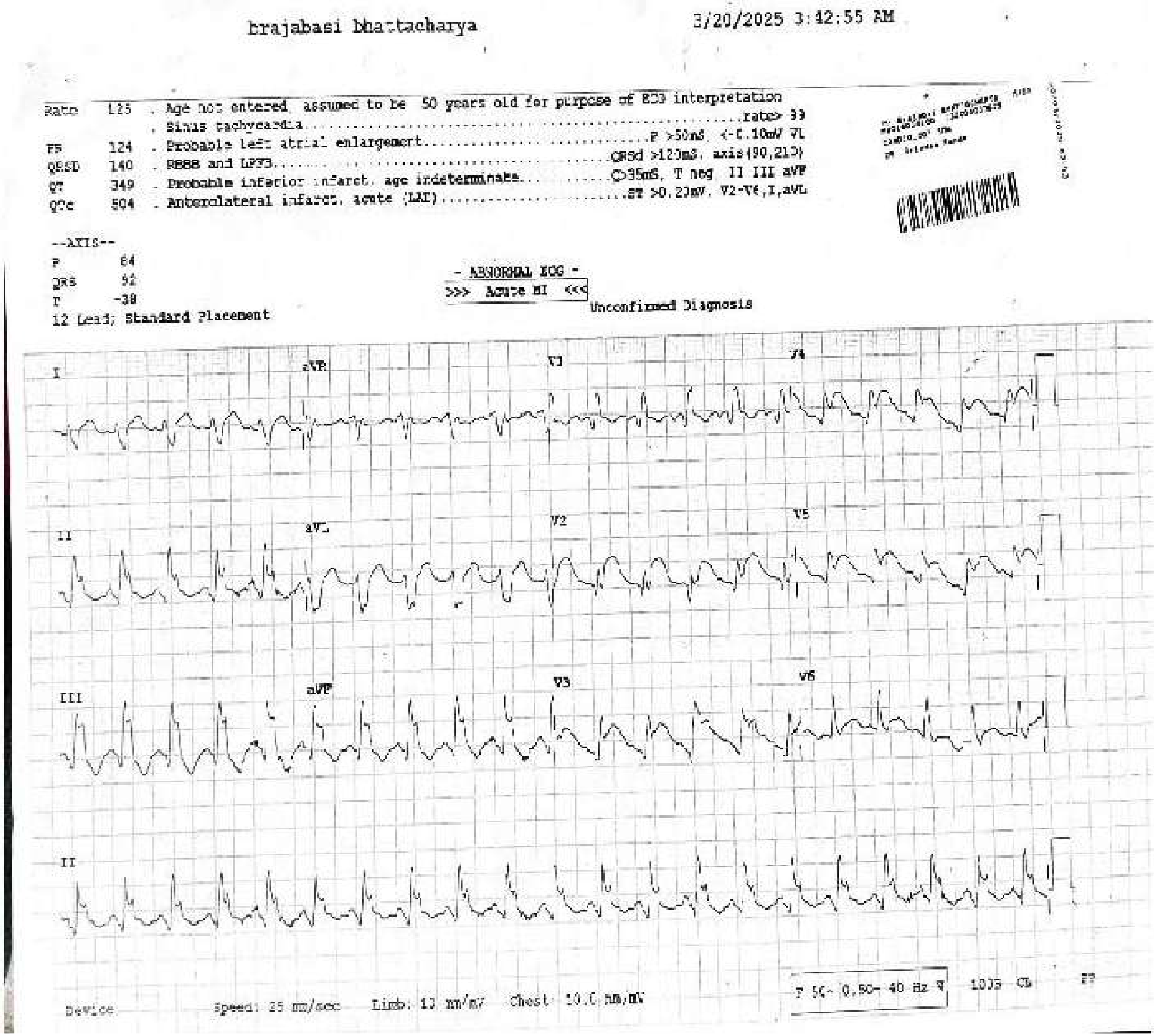
- **Timing of ECG Changes in Ischemia and MI, and Intervals for Repeat ECGs:** How quickly do ECG changes evolve in ischemia and MI, and what are the optimal intervals for serial ECGs in suspected ACS?
- **Role of Bedside Echocardiography in ACS in the ED:** What is the role of bedside echocardiography in evaluating ACS in the ED, what findings are sought, and how does its accuracy compare to formal cardiology echocardiography?
- **Significance of Negative Initial Troponin T:** Given the patient's one-hour history, the initial troponin T negative, when should serial troponins be measured, and when is it typically positive?
- **Risk in "Apparently Healthy" Older Smokers:** How does being an "apparently healthy" 60- year-old smoker with no medical check-ups increase the risk of ACS?
- **Role of Smoking with Non-Diagnostic ECGs:** What is the significance of a smoking history in a patient with shortness of breath, atypical chest discomfort, and a non-diagnostic ECG?
- **Diagnostic Accuracy of ER Physician-Performed Echocardiography:** How does the sensitivity and specificity of bedside echocardiography performed by an ER physician compare to that of a cardiologist?
- **Urgent Catheterization in Non-STEMI Presentations:** When should urgent catheterization be considered in a patient with new-onset chest discomfort but without ST-elevation on the initial ECG, and how should ER physician evaluation prioritize the need for urgent intervention?


BROJOBASU BHATTACHARYA
3/20/2025 2:50:50 AM
Rate 129
PR 138
QRSD 108
QT 292
QTc 428
. Age not entered, assumed to be 50 years old for purpose of ECG interpretation
. Sinus tachycardia.....rate> 99
. Ventricular premature complex.....V complex w/ short R-R interval
. Left atrial enlargement.....P, P'>60mS, <-0.15mV V1
. Anterior infarct, acute (LAD).....ST >0.25mV, V2-V5
. Artifact in lead(s) I,II,III,aVR,aVF,V3,V4,V5,V6 and baseline wander in lead(s) I ,aVL,V3
--AXIS--
P 37
QRS 36
T 135
12 Lead; Standard Placement
- ABNORMAL ECG -
>>> Acute MI <<<
Unconfirmed Diagnosis
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Device:
Speed: 25 mm/sec
Limb: 10 mm/mV
Chest: 10.0 mm/mV
F 50- 0.50- 40 Hz W
100B CL

ECG 1



ECG 2


Brajabasi Bhattacharya
3/20/2025 3:42:55 AM
Rate 125 . Age not entered, assumed to be 50 years old for purpose of ECG interpretation
. Sinus tachycardia
PR 124 . Probable left atrial enlargement
QRSD 140 . RBBB and LPFB
QT 349 . Probable inferior infarct, age indeterminate
QTc 504 . Anterolateral infarct, acute (LAD)
--AXIS--
P 64
QRS 92
T -38
12 Lead; Standard Placement
- ABNORMAL ECG -
>>> Acute MI <<<
Unconfirmed Diagnosis
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Device
Speed: 25 mm/sec
Chest: 10.0 mm/mV

ECG 3

CONCLUSION

This case underscores the need for a high index of suspicion for ACS, even with atypical presentations and initially non-diagnostic ECGs. The following points address the questions raised in the discussion:

Timing of ECG Changes

•ECG changes in ischemia can begin within minutes (hyperacute T waves) but may take hours to evolve into classic STEMI patterns. In this case, the patient's ECG evolved from normal at 02:50 am to a "tombstone" pattern by 03:42 am.
•Serial ECGs should be performed frequently: guidelines recommend within 5-10 minutes for ongoing symptoms, and then every 15-30 minutes if symptoms persist or new symptoms develop.

Research Summary: Guidelines emphasize repeated ECGs at intervals in patients with ongoing chest pain or other symptoms suggestive of NSTE-ACS. This approach aims to capture the dynamic evolution of ischemia, which may not be apparent on the initial ECG. (1,2,3)

Role of Bedside Echocardiography

•POCUS can rapidly detect regional wall motion abnormalities (RWMA) and assess left ventricular function.
•Findings suggestive of ACS: New or worsening RWMA.
•POCUS is a valuable adjunct, not a replacement for formal echocardiography.

Research Summary: A meta-analysis suggests that POCUS showing RWMA in patients with chest pain and non-diagnostic ECGs is associated with a higher risk of acute coronary syndrome. This indicates that POCUS can help identify patients with ACS who may be missed by initial ECG. (4,5)

Significance of Negative Initial Troponin T

•Troponin may be negative in the first few hours of AMI.
• High-sensitivity troponin I can be positive as early as 1-3 hours; peak is at 12-24 hours. In this case, the initial troponin at 02:50 am was negative, which is consistent with early presentation.
• Serial troponins should be measured at 0, 3, and 6 hours from presentation (or according to local protocol)

Cardiac biomarkers of acute coronary syndrome: from history to high-sensitivity cardiac troponin: The review article provides a good overview of the evolution of troponin assays and discusses the kinetics of troponin release in ACS. It highlights that troponins appear in the serum 4–10 hours after the onset of AMI, peak at 12–48 hours, and remain elevated for 4–10 days. It also emphasizes the utility of high-sensitivity assays for earlier diagnosis.(6)

High-Sensitivity Cardiac Troponin and the 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR Guidelines for the Evaluation and Diagnosis of Acute Chest Pain: The review article discusses the recommendations for using high-sensitivity cardiac troponin assays in the evaluation of acute chest pain, which is crucial in suspected ACS. It touches upon the timing of repeat testing within rule-out protocols.(7)

Early kinetics of cardiac troponin in suspected acute myocardial infarction: This review article specifically looks at the early release kinetics of high-sensitivity cardiac troponin T and I within the first 1-2 hours of presentation, which is relevant to the timing of repeat tests in rapid rule-out protocols.(8)

Cardiac Troponin Thresholds and Kinetics to Differentiate Myocardial Injury and Myocardial Infarction: This review examines troponin concentrations and their changes over time to differentiate between myocardial injury and infarction, providing insights into the significance of serial measurements.(9)

High-Sensitivity Troponin Assays: Evidence, Indications, and Reasonable Use: This review article discusses the advantages of high-sensitivity troponin assays, including their potential for earlier diagnosis and rule-out of MI, which directly impacts the timing of repeat testing strategies.(10,11)

Risk in "Apparently Healthy" Older Smokers

•Age >40 is an independent risk factor for atherosclerosis and ACS.
•Smoking compounds this risk, promoting plaque instability.
•Research Summary: Age is a non-modifiable risk factor for heart disease. The risk of heart disease increases with age due to the gradual buildup of plaque in the arteries.

Smoking is a Strong Independent Risk Factor: Multiple studies consistently identify smoking as a major and independent risk factor for ACS and Myocardial Infarction (MI) across various age groups. The odds ratio for smokers experiencing an MI compared to non-smokers is often cited around 2.0 to 2.5. Even low levels of smoking are associated with a significantly increased risk. Smoking contributes to endothelial dysfunction, inflammation, thrombosis, and the progression of atherosclerosis.(12)

Age Increases Risk: While smoking is a potent risk factor, age itself is also a non-modifiable risk factor for ACS. The incidence of ACS and cardiovascular-related deaths increases with age, particularly in men over 45 and women post-menopause.(13)

Interaction of Age and Smoking: Research indicates that the relative risk of cardiac death associated with smoking might be highest in middle-aged individuals (e.g., in their 40s) compared to never-smokers and may gradually decrease with very advanced age, although the absolute risk remains significant for older smokers.(14)

Lack of Comorbidities Doesn't Eliminate Risk: While the absence of comorbidities like hypertension, diabetes, and dyslipidemia is beneficial, smoking alone significantly elevates the risk of ACS. Smokers can develop ACS even without other traditional risk factors.

Smoking Cessation is Highly Beneficial: Quitting smoking at any age, even after 40, significantly reduces the risk of ACS and cardiovascular events. The risk of MI can be reduced by as much as 50% within one year of quitting.(15)

Role of Smoking with Non-Diagnostic ECGs

- Smoking increases ACS risk, even with a normal ECG.
- Suspicion should be high in smokers with suggestive symptoms, and serial ECGs are crucial.

Diagnostic Accuracy of ER Physician-Performed Echocardiography

- Sensitivity for RWMA in acute MI: 50-85%.
- Specificity for RWMA in acute MI: 75-95%.
- Cardiologist-performed echo is more comprehensive but less immediately available.

The Diagnostic Value of Echocardiography Performed by an Emergency Medicine Physician in the Diagnosis of Acute Coronary Syndrome: Review study found that emergency physicians could identify ACS based on wall motion defects with a sensitivity of 75% and a high negative predictive value (97%).(16)

Diagnostic accuracy of point-of-care cardiac ultrasound for acute coronary syndromes in patients presenting with chest pain to the emergency department: a single-center prospective study: This more recent study showed that regional wall motion abnormalities detected by FOCUS were an independent predictor of NSTE-ACS with high specificity but moderate sensitivity.(17)

Bedside Limited Echocardiography by the Emergency Physician Is Accurate During Evaluation of the Critically Ill Patient: While focusing on critically ill patients in general, this study supports the accuracy of focused echo by trained emergency physicians for assessing cardiac function.(18)

Urgent Catheterization in Non-STEMI Presentations

- Consider in high-risk NSTE-ACS: ongoing pain, dynamic ECG changes, elevated troponin, hemodynamic instability.
- ER physician evaluation must prioritize these high-risk features for urgent cath lab activation.

Risk Stratification is Crucial: The decision for and timing of catheterization in NSTEMI/NSTE- ACS heavily relies on risk stratification using clinical features, ECG findings, and cardiac biomarkers (like troponin).

Early Invasive Strategy for High-Risk: Current evidence and guidelines generally favor an early invasive strategy (coronary angiography within 24-72 hours) for patients deemed to be at moderate to high risk of adverse events.

Potential Benefits of Early Catheterization: Early catheterization allows for prompt identification of coronary artery lesions, risk assessment, and timely revascularization (PCI) when appropriate, potentially improving outcomes and reducing the risk of future events.

Ischemia-Guided Strategy for Low-Risk: A more conservative, ischemia-guided strategy (where catheterization is performed only if there is evidence of recurrent ischemia or high-risk features on non-invasive testing) may be appropriate for some low-risk patients.

This case underscores the need for a comprehensive and dynamic approach to patients with suspected acute coronary syndrome.

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