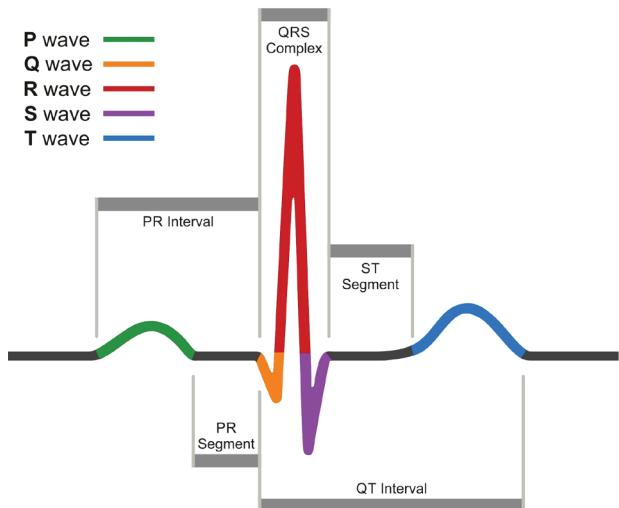


# Journal of Cardiac Rhythm

Detecting STEMI from fake STEMI



## CASE 1

A case of ECG showing ST elevation – Was it a STEMI?

## CASE 2

A case of anterior STEMI-equivalent (de Winter syndrome): Are we missing the STEMI?

## CASE 3

A case of acute coronary syndrome in a 61-year-old male presenting with tombstone ST-segment elevation

# PROTECTION COMES IN DIFFERENT FORMS

SOME ARE OUR  
**BELIEFS**



Hanging horse shoe brings good luck and **protection** from Evil

SOME ARE A  
**REALITY**

Patients Post PCI / Stroke are at high risk of CVD

For CVD prevention post PCI/CABG,

<sup>Rx</sup> **Rosukem Gold**  
Rosuvastatin 10mg + Clopidogrel 75 mg + Aspirin 75 mg



Confidence of Protection



# A case of ECG showing ST elevation – Was it a STEMI?

## Case summary

A 33-year-old male presented with sudden onset substernal chest pain that started while exercising on a treadmill one hour ago. Pain was left sided sharp, non-radiating, with associated nausea, diaphoresis, and shortness of breath. General physical and cardiovascular examinations were normal. Investigations revealed elevated troponin. A 12 lead ECG depicted ST-segment elevation in the inferior leads. An emergent left cardiac catheterization reported normal coronary anatomy with no obstructing coronary stenosis. He was started on a heparin drip and transferred to the coronary care unit for further investigations. Two days after presentation, the patient still reported continued chest pain and had an episode of nonsustained ventricular tachycardia. A cardiac MRI was done at this point and it demonstrated epicardial and midmyocardial enhancement in the inferior wall, sparing of the sub-endocardial region, and overlying focal pericardial enhancement, consistent with ECG changes. A diagnosis of focal myocarditis was made. He was started on indomethacin; his symptoms improved in the following 5 days, and he was discharged.

## Case presentation

- A previously healthy 33-year-old male presented with sudden onset substernal chest pain that started while exercising on a treadmill one hour ago
- He described a left sided sharp, non-radiating pain that persisted till he presented to the emergency department
- He had associated nausea, diaphoresis, and shortness of breath
- He denied heartburn, vomiting, cough, fever, and recent travel. He had no personal or family history of heart disease.



### Physical examination

- On physical examination, he was a young athletic male who appeared in distress from the pain
- His vitals were: Temperature- 98.6°F, pulse rate- 88/minute (with normal rhythm and tone), respiratory rate- 16 breaths/minute, and blood pressure (in arm at resting position)- 130/90 mmHg
- Pallor, icterus, and edema were absent.

### Systemic examination

- Jugular venous pressure (JVP) was not raised and carotid bruit could not be auscultated
- Cardiovascular examination was normal, with normal S<sub>1</sub> and S<sub>2</sub>
- S<sub>3</sub> and gallop rhythm could not be auscultated
- There were no murmurs or pericardial rubs
- Rest of the systemic examination was non-contributory.

### Investigations

- Complete blood count (CBC): Hb- 12.5 g/dL, TLC- 7,600 cells/mm<sup>3</sup>
- Fasting blood sugar- 100 mg/dL, post-prandial blood sugar- 130 mg/dL
- Lipid profile:
  - Total cholesterol: 140 mg/dL
  - High density lipoprotein cholesterol (HDL-C): 40 mg/dL
  - Low density lipoprotein cholesterol (LDL-C): 63 mg/dL
  - Triglycerides: 150 mg/dL
- LFT: AST-30 U/L (normal, 0-35 U/L), ALT- 30 U/L (normal, 3-36 U/L), alkaline phosphatase- 57 U/L (normal, 35-100 U/L)
- Troponin I -21.9 ng/mL (elevated)
- Serum creatinine: 0.9 mg/dL (normal, 0.6 to 1.2 mg/dL)
- Routine urine and microscopy were normal. Microalbuminuria was not detected.

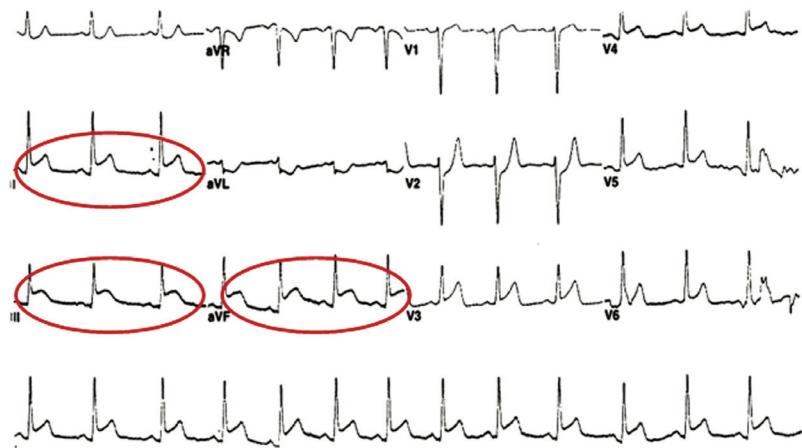
### ECG findings

A 12 lead ECG depicted ST-segment elevation in the inferior leads (Figure 1).



**FIGURE 1**

ECG showing ST-segment elevation in the inferior leads

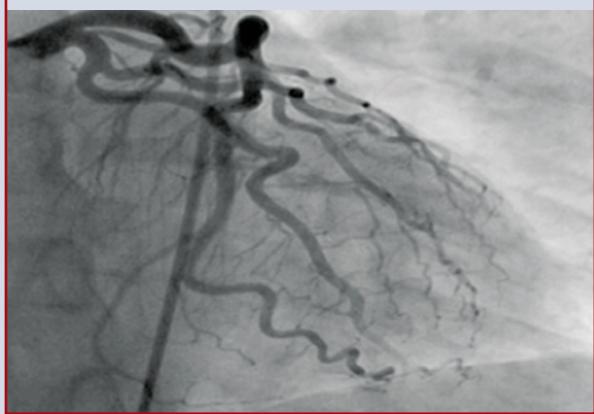


### Investigations continue...STEMI or Not?

- A STEMI alert was placed, and patient had an emergent left cardiac catheterization that reported normal coronary anatomy with no obstructing coronary stenosis (Figure 2, 3)
- A left ventriculogram was also normal
- He was started on a heparin drip and transferred to the coronary care unit
- A plain chest X-ray did not reveal any pulmonary lesions or consolidation, and a chest CT angiogram ruled out pulmonary embolism
- A transthoracic echocardiogram done reported a normal left ventricular ejection fraction (EF 50–55%) and a slight enlargement of the right ventricle without any wall motion abnormalities

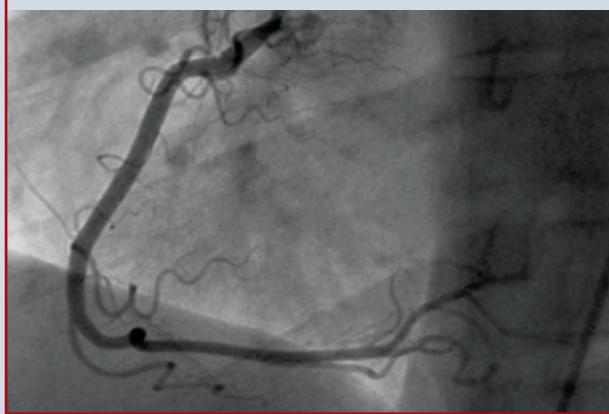
**FIGURE 2**

Left coronary angiogram with normal left coronary anatomy with no obstructing atheroma



**FIGURE 3**

Right coronary angiogram with normal right coronary anatomy with no obstructing atheroma



- Two days after presentation, the patient still reported continued chest pain and had an episode of nonsustained ventricular tachycardia (NSVT)
- At this point, a cardiac MRI was done that demonstrated epicardial and midmyocardial enhancement in the inferior wall, sparing of the sub-endocardial region, and overlying focal pericardial enhancement, consistent with ECG changes.

### Diagnosis

A diagnosis of focal myocarditis was made.

### Therapeutic intervention

He was started on indomethacin; his symptoms improved in the following 5 days, and he was discharged.

## Focal myocarditis

- Myocarditis is an inflammatory disease of the myocardium associated with cardiac dysfunction; it can be diffused or focal<sup>1</sup>
- The etiology of myocarditis might be infectious, post viral autoimmune-related, autoimmune-mediated (lupus myocarditis, giant cell myocarditis) or drug-associated (hypersensitivity myocarditis, toxic myocarditis)<sup>2</sup>
- Common manifestations of focal myocarditis are pleuritic chest pain, fatigue and decreased exercise capacity with a history of febrile syndrome.<sup>2</sup>

## STEMI and focal myocarditis: Identifying the ECG imposter

- STEMI is the most important differential diagnosis of focal myocarditis<sup>2</sup>
- Focal myocarditis commonly mimics acute STEMI in clinical symptoms as well as in its electrocardiogram (ECG) features and elevated levels of cardiac enzymes<sup>2</sup>
- However, in case of myocarditis, STE will be present in most leads, except for aVR and V1 where ST-segment depression can be seen due to its distant and opposite position of the normal heart axis<sup>3</sup>
- In order to differentiate between focal myocarditis and STEMI in a better way, cardiac magnetic resonance (CMR) with gadolinium must be performed<sup>2</sup>
- In cases of STEMI, the characteristic finding of CMR is sub-endothelial enhancement, while in cases of focal myocarditis, enhancement is noted to originate from epicardium with sub-endocardial sparing.<sup>2</sup>

## References

1. Calabrese F, Thiene G. Myocarditis and inflammatory cardiomyopathy: microbiological and molecular biological aspects. *Cardiovas Res*.2003;60(1):11–25.
2. Nozari Y, Tajdini M, Mehrani M, Ghaderpanah R. Focal Myopericarditis as a Rare but Important Differential Diagnosis of Myocardial Infarction; a Case Series. *Emerg (Tehran)*. 2016;4(3):159–162.
3. de Blieck EC. ST elevation: Differential diagnosis and caveats. A comprehensive review to help distinguish ST elevation myocardial infarction from nonischemic etiologies of ST elevation. *Turk J Emerg Med*. 2018;18(1):1–10.



# A case of anterior STEMI-equivalent (de Winter syndrome): Are we missing the STEMI?

## Case summary

A 36-year-old female presented to the emergency department with chest pain at rest since 3 hours. The pain commenced after she took her lunch; it was sharp and with a feeling of tightness in the chest. Past medical history revealed poorly controlled hypertension, for which patient was taking captopril. General physical examination and cardiopulmonary examination was within normal limits. ECG showed pattern characteristic of de Winter T-wave ECG pattern. The patient was diagnosed with acute coronary syndrome with suspected de Winter T-wave ECG pattern. Patient was administered aspirin 160 mg, clopidogrel 300 mg, sublingual isosorbide dinitrate, atorvastatin 20 mg, and enoxaparin 0.4 mg subcutaneous. She refused referral to the interventional cardiology capable hospital due to distance and socioeconomic causes. Repeat ECG done 3 hours later showed changes consistent with the final stages of acute STEMI and elevated troponin at 5<sup>th</sup> hour confirmed the presence of myocardial infarction. Streptokinase (1.5 million units) was administered, and chest pain subsided with a resolution of ST-segment elevation. The patient was discharged on the 5<sup>th</sup> day after admission.

### Case presentation

- A 36-year-old female presented to the emergency department with chest pain at rest since 3 hours
- The pain commenced after she took her lunch; it was sharp and with a feeling of tightness in the chest
- There was no history of shortness of breath or any previous episode of chest pain
- Past medical history revealed poorly controlled hypertension, for which patient was taking captopril
- There was no history of diabetes, smoking, previous stroke or myocardial infarction.



### Physical examination

- The patient was well-oriented to time, place and person
- Her vitals were: Blood pressure- 150/90 mmHg, heart rate- 57/minute, and respiratory rate- 20/minute
- Pallor, icterus, edema were absent.

### Systemic examination

- JVP was not raised and carotid bruit could not be auscultated
- Cardiopulmonary examination was within normal limits
- S<sub>1</sub> and S<sub>2</sub> were normal and S<sub>3</sub> and gallop rhythm could not be auscultated
- There were no murmurs or pericardial rubs
- Rest of the systemic examination was non-contributory.

### Investigations

- CBC: Hb- 12.0 g/dL, TLC- 7,500 cells/mm<sup>3</sup>
- Fasting blood sugar- 103 mg/dL, post-prandial blood sugar- 127 mg/dL
- Lipid profile: Normal
- LFT: AST-29 U/L (normal, 0-35 U/L), ALT- 28 U/L (normal, 3-36 U/L), alkaline phosphatase- 64 U/L (normal, 35-100 U/L)
- Troponin at 2<sup>nd</sup> hour onset was negative
- Serum creatinine: 1.0 mg/dL (normal range, 0.6 to 1.2 mg/dL)
- Serum sodium - 137 mmol/L (normal)
- Serum potassium - 3.4 mmol/L (normal)
- Routine urine and microscopy were normal. Microalbuminuria was not detected.

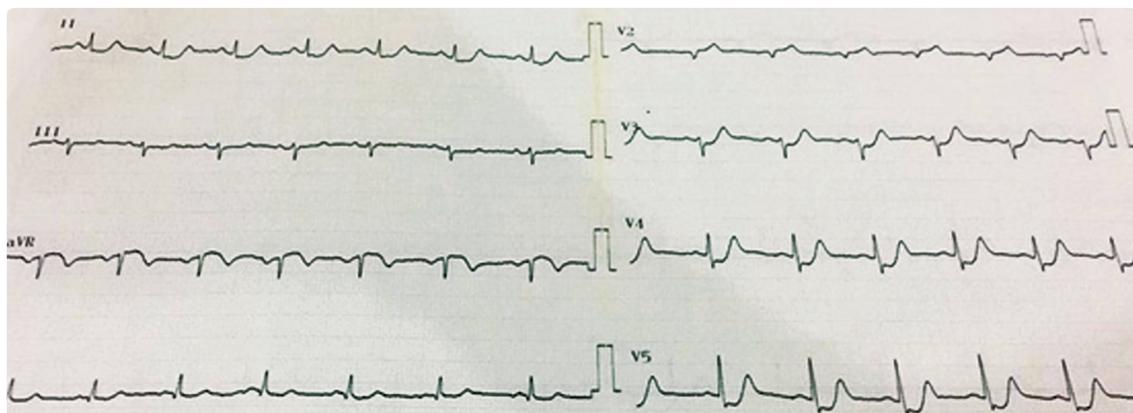
### ECG findings

- The 12-lead ECG obtained at admission revealed significant ST-segment depression (> 1 mm) at the J point in leads V<sub>3</sub>-V<sub>6</sub>, with tall, positively symmetrical T waves. Slight ST-segment elevation (0.5 mm) was evident in the aVR lead (Figure 1).



**FIGURE 1**

The 12-lead ECG taken at the time of admission showing upsloping ST-segment depression at the J point in leads V<sub>3</sub>-V<sub>6</sub>, with prominent T waves and slight ST-segment elevation evident in the aVR lead



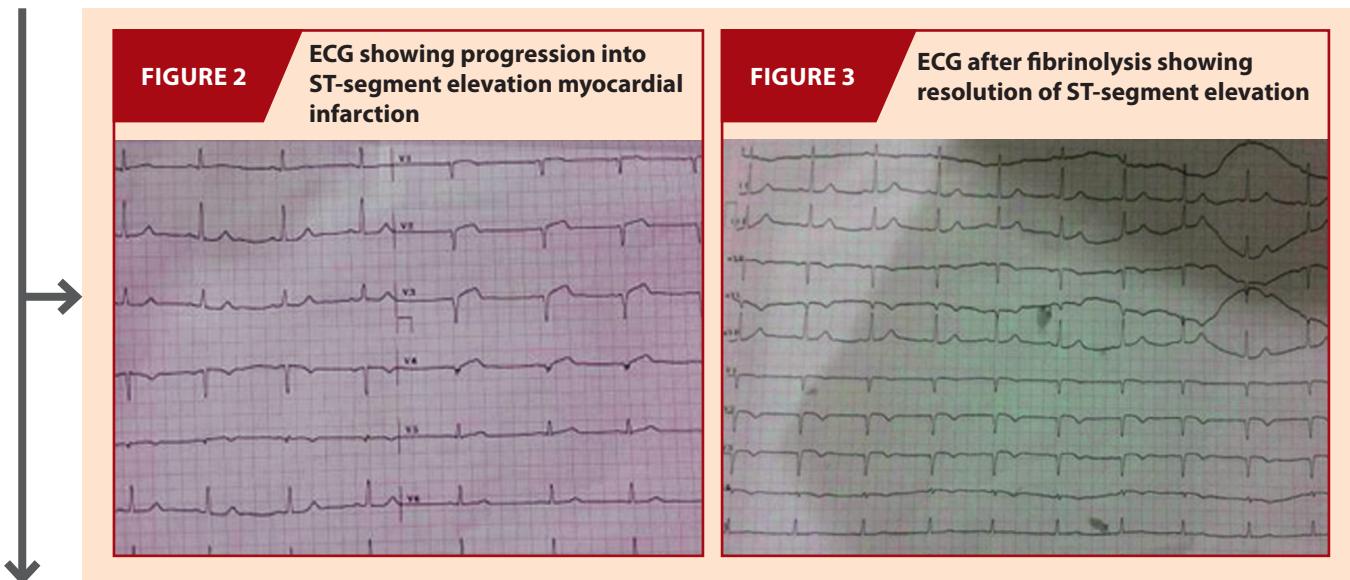
## Diagnosis

The patient was diagnosed with acute coronary syndrome with suspected de Winter T-wave ECG pattern.

## Therapeutic intervention

- Patient was administered aspirin 160 mg, clopidogrel 300 mg, sublingual isosorbide dinitrate, atorvastatin 20 mg, and enoxaparin 0.4 mg subcutaneous
- She refused referral to the interventional cardiology capable hospital due to distance and socioeconomic causes
- Repeat ECG was done 3 hours later showing Q-waves developing in V<sub>2-4</sub>, consistent with the final stages of acute STEMI accompanied with ST-segment elevation in lead V<sub>2-4</sub> (Figure 2)
- Elevated troponin at the 5<sup>th</sup> hour confirmed the presence of myocardial infarction
- There was still sustained and severe chest pain although not as intense as initial presentation
- Streptokinase (1.5 million units) was administered, and chest pain subsided with a resolution of ST-segment elevation (Figure 3)
- The patient was discharged on the 5<sup>th</sup> day after admission.





## de Winter syndrome: An ST-segment elevation myocardial infarction-equivalent

- As per the ESC/ACC/AHA/WHF expert consensus document 2018, ST-segment elevation in lead aVR with specific repolarization patterns should be regarded as a STEMI equivalent<sup>1</sup>
- STEMI equivalents are considered to be potentially devastating forms of STEMI that present a subtle ECG pattern without classical ST elevation<sup>2</sup>
- de Winter syndrome is an ECG pattern that is often unrecognized by physicians.<sup>3</sup>

## How to identify de Winter syndrome?

- Identification of the ECG patterns of de Winter syndrome should be done promptly for timely investigation and revascularization of the proximal left anterior descending coronary artery<sup>4</sup>
- The characteristic ECG features of de Winter syndrome are depicted in Table 1.<sup>4</sup>

## References

- Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ. ESC/ACC/AHA/WHF expert consensus document Fourth Universal Definition of Myocardial Infarction (2018). *Circulation.* 2018;138:e618–e651.
- Dastidar A. STEMI equivalent: Are we missing the STEMIs? Available at: [https://www.bcs.com/pages/news\\_full.asp?NewsID=19792165](https://www.bcs.com/pages/news_full.asp?NewsID=19792165) Accessed on: 2.4.19.
- Pranata R, Huang I, Damay V. Should de Winter T-Wave Electrocardiography Pattern Be Treated as ST-Segment Elevation Myocardial Infarction Equivalent with Consequent Reperfusion? A Dilemmatic Experience in Rural Area of Indonesia. *Case Rep Cardiol.* 2018;2018:6868204.
- Baranchuk A, Bayés-Genis A. Naming and classifying old and new ECG phenomena. *CMAJ.* 2016;188(7):485–486.

**Table 1: Characteristic ECG features of de Winter syndrome**

ST segment in precordial leads	Depressed
Preferential precordial leads	V2-V4
T-wave characteristics	Positive and symmetric (hyperacute T waves)
T-wave morphology	Ascending limb of the T wave commencing below the isoelectric baseline
Precordial R-wave progression	Normal
Pathologic Q waves	No
ST-segment elevation in lead aVR	Yes
Evolution of the ECG pattern	Stable or evolving into "classic" STEMI

**Abbreviations:** ECG = electrocardiography, LAD = left anterior descending artery, STEMI = ST-segment elevation myocardial infarction.

**Adapted from:** Baranchuk A, Bayés-Genis A. Naming and classifying old and new ECG phenomena. *CMAJ.* 2016;188(7):485–486.



# A case of acute coronary syndrome in a 61-year-old male presenting with tombstone ST- segment elevation

## Case summary

A 61-year-old male presented with a typical chest pain. He was a chronic smoker for the last 40 years. There was no history of diabetes mellitus, hypertension or ischemic heart disease. ECG was taken, which showed ST elevations in V1-V6, I and aVL leads. The ECG had typical tombstone pattern in V2-V5 leads. Since the patient came without any relatives, consent for cath-lab procedures could not be taken. However, considering the patient's condition, he was thrombolysed with streptokinase after ruling out the contraindications for thrombolytic therapy. Pain subsided and ECG was settled after 90 minutes post streptokinase. Echocardiography revealed anterior, antero-septal, apico-septal, and lateral wall hypokinesia with preserved thickness and an ejection fraction (EF) of 38%. The following day, relatives of the patient came and gave their consent for coronary angiography (CAG), which showed 80% thrombotic stenosis in proximal left anterior descending artery (LAD). Patient was taken up for percutaneous coronary intervention (PCI) with stenting to proximal LAD. He was given a loading dose of aspirin and clopidogrel. Procedural anticoagulation was achieved with heparin. Further, a glycoprotein IIb/IIIa inhibitor was given intraprocedurally. The PCI procedure was successful and the post procedural angiograph indicated thrombolysis in myocardial infarction (TIMI) grade III flow in the LAD. Improvement in patient's left ventricular EF (45%) was also noticed. Subsequently, the patient was discharged in good general condition.

### Case presentation

- A 61-year-old male presented with a typical chest pain since 3 hours
- The pain was described as crushing pain in the chest by the patient with nausea
- He was a chronic smoker for the last 40 years
- There was no history of diabetes mellitus, hypertension or ischemic heart disease.



### Physical examination

- On physical examination, the patient was in discomfort due to the pain
- His vitals were: Temperature- 98.4°F, pulse rate- 80/minute (with normal rhythm and tone), respiratory rate- 18 breaths/minute, and blood pressure- 126/84 mmHg
- Pallor, icterus, and edema were absent.

### Systemic examination

- JVP was not raised and carotid bruit could not be auscultated
- Cardiovascular examination was normal, with normal S<sub>1</sub> and S<sub>2</sub>
- S<sub>3</sub>, gallop rhythm and murmurs could not be auscultated
- Rest of the systemic examination was non-contributory.

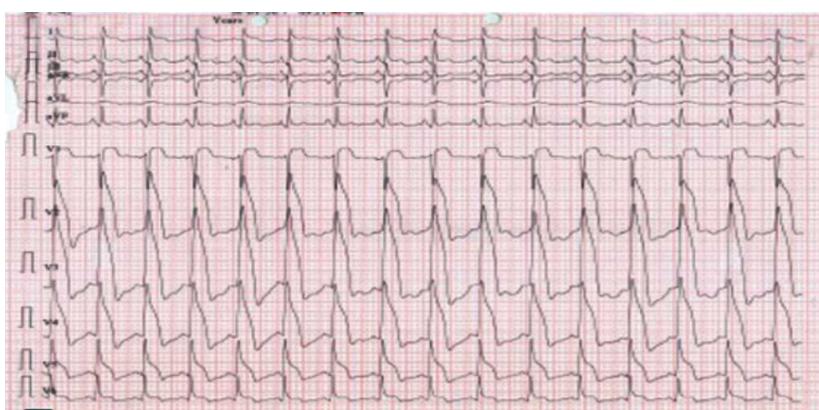
### Investigations

- Routine blood investigations: Normal
- Lipid profile: Normal
- Liver function test: Normal
- Serum creatinine: 1.8 mg/dL (normal, 0.6 to 1.2 mg/dL).

### ECG findings

The ECG had typical tombstone pattern in V<sub>2</sub>-V<sub>5</sub> leads (Figure 1).

**FIGURE 1** ECG showing typical tombstone pattern in V<sub>2</sub>-V<sub>5</sub> leads



## Diagnosis

Myocardial infarction with tombstone ST-segment elevation.

## Further investigations and interventions

- Since the patient came without any relatives, consent for cath-lab procedures could not be taken
- Hence, CAG of the patient at presentation could not be performed
- However, considering the patient's condition, he was thrombolysed with streptokinase after ruling out the contraindications for thrombolytic therapy
- Pain subsided and ECG was settled after 90 minutes post streptokinase
- Echocardiography revealed anterior, antero-septal, apico-septal, and lateral wall hypokinesia with preserved thickness and an EF of 38%
- The following day, relatives of the patient came and gave their consent for CAG, which showed 80% thrombotic stenosis in proximal LAD
- Patient was taken up for PCI with stenting to proximal LAD
- He was given a loading dose of aspirin and clopidogrel
- Procedural anticoagulation was achieved with heparin. Further, a glycoprotein IIb/IIIa inhibitor was given intraprocedurally
- The PCI procedure was successful and the post procedural angiograph indicated TIMI grade III flow in the LAD
- Improvement in patient's left ventricular EF (45%) was also noticed
- Subsequently, the patient was discharged in good general condition.

## STEMI and the tombstone pattern of ST segment

- Among patients with STEMI, many variants can be observed owing to differences in patterns such as amplitude and morphology of ST-segment elevation, T-wave variations, presence or absence of Q-wave and clinical course and prognosis<sup>1</sup>
- One such morphological variant is the tombstone ST-segment elevation, that is observed in the early phase of acute MI suggesting extensive myocardial damage and serious clinical results<sup>1</sup>
- In such cases, the ST segment is convexed upwards and the peak of the convexed ST segment is usually higher than the preceding R wave, which is less than 0.04 s and small in amplitude<sup>2</sup>
- The electrophysiological mechanisms that come into play in the formation of a tombstone appearance are delayed transmural conduction and intramyocardial conduction block.<sup>3</sup>



## ECG characteristics of tombstone STEMI

- Tombstoning in ECG is observed in about 10-26.1% of the patients<sup>3</sup>
- The ECG characteristics for tombstone STEMI are as follows:<sup>1</sup>
  - Absent R wave or an R wave duration of <0.04 second with minimal amplitude
  - Convex upward ST segment merging with descending R or the ascending QS/QR
  - Peak of ST segment is higher than the R wave
  - ST segment merges with the T wave
- A basic comparison of clinical profile of tombstone-STEMI with non-tombstone STEMI is given in Table 1.<sup>1</sup>

**Table 1. Clinical profile of tombstone-STEMI compared with non-tombstone STEMI**

Coronary risk factors	Similar
Symptom	Similar angina pectoris but less frequent preinfarction angina
Laboratory	Higher CK, higher BNP which means larger infarction area
Echocardiography	Lower left ventricle EF which means a heavier left ventricle dysfunction
Angiography	Similar epicardial coronary anatomy or more extensive. Higher TIMI frame count and lower TIMI myocardial perfusion grade which means more severe ischemia
Complication	More complications and poor prognosis
Reperfusion	Less efficient reperfusion therapy

**Abbreviations:** CK=creatinine kinase; BNP=brain natriuretic peptide; EF=ejection fraction; TIMI=thrombolysis in myocardial infarction.

**Adapted from:** Balci B. Tombstoning ST-Elevation Myocardial Infarction. *Curr Cardiol Rev.* 2009;5(4):273–278.

## References

1. Patil S, Shetty N, Hidayathulla M, Ramalingam R, Kasamsetty S, et al. Tombstone ST-segment elevation in acute anterior wall myocardial infarction. *IJH Cardiovascular Case Reports (CVCR)* 2018;2:S11eS13.
2. Sinha MK, Dasgupta D, Lyons JP. “Tombstone” ST segment elevation of acute myocardial infarction. *Postgraduate Medical Journal* 2004;80:276.
3. Balci B. Tombstoning ST-Elevation Myocardial Infarction. *Curr Cardiol Rev.* 2009;5(4):273–278.





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