

## Case Report: Hyperacute Myocardial Infarction

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### Abstract

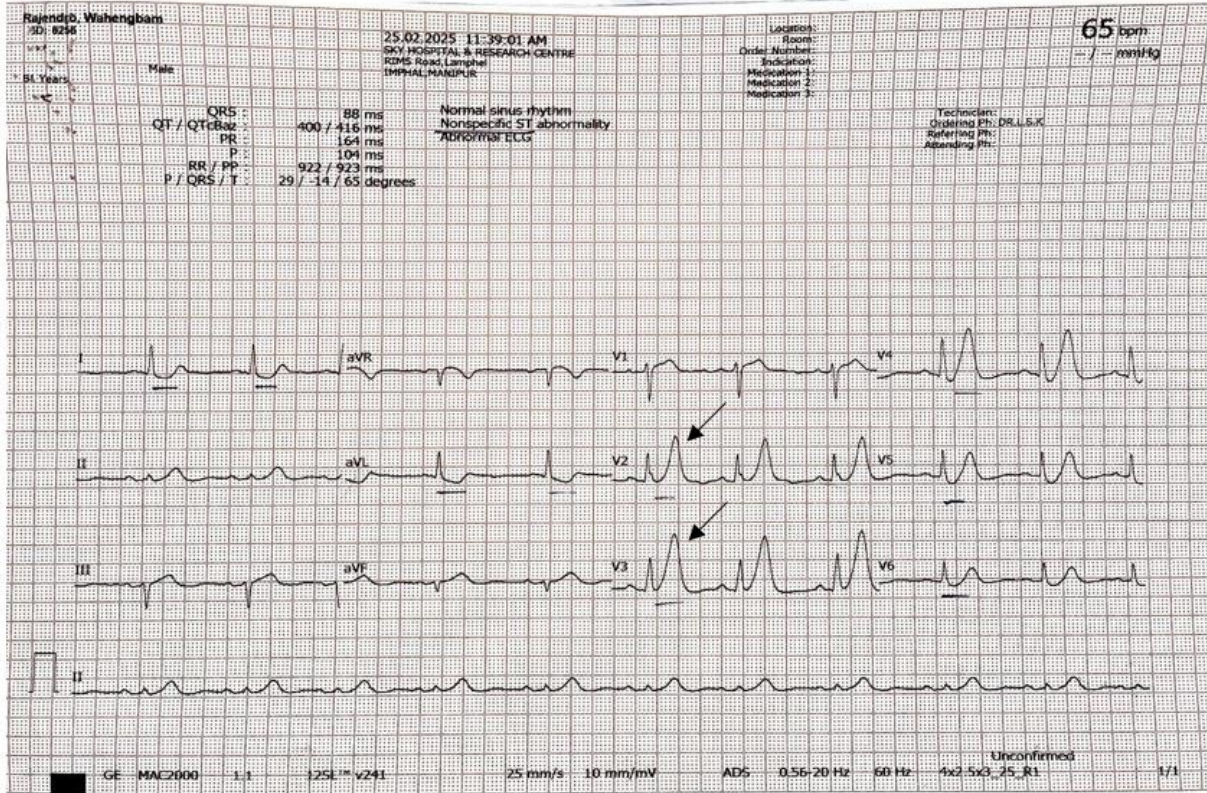
Among the various manifestations of electrocardiogram (ECG) for diagnosis of acute transmural myocardial infarction (ATMI), broad-based and tall tented T-waves, called hyperacute T-waves, may be the earliest and the only ECG sign of ATMI. As it is a rare ECG sign of ATMI, and because of its transient nature in the very early stage, quick identification of such ECG sign should trigger immediate evidence-based reperfusion therapeutic strategy to maximize myocardial salvage and survival of patients in the acute phase and in the long term. A male, 81 years of age with sudden onset typical ischemic chest pain, having history of chronic smoking, presented to the Heart Attack Centre (HAC) of SKY Hospital and Research Centre. The only ECG sign on presentation at the HAC was hyperacute T-wave changes in the chest leads, V<sub>2</sub>-V<sub>6</sub>. Cardiac injury marker (troponin T) done on presentation was normal. A simultaneously done bedside echocardiogram showed regional wall motion abnormality (RWMA) in the territory of left anterior descending (LAD) artery. Based on these findings, the patient was thrombolysed with intravenous (IV) tenecteplase with simultaneous administration of dual antiplatelet drugs and subcutaneous enoxaparin injection within 10 minutes of presentation to the HAC. His chest pain was completely relieved within minutes of the first-aid medication. Post-thrombolytic ECG showed near normalization of the T-wave changes. Subsequent coronary angiography showed 20% recanalization of proximal LAD artery obstruction which has probably helped save the myocardium in jeopardy and thereby reduced further damage of the myocardium. The culprit artery was stented with complete reperfusion of the affected myocardium.

**Key Words:** case report; hyperacute t-waves

### Introduction

Chest pain is one of the most common reasons for emergency department (ED) presentation. Ten to thirteen percent of these patients are diagnosed as acute coronary syndrome (ACS) [1]. Rapid identification of myocardial infarction as a life-threatening disorder is important for the early initiation of effective evidence-based therapy, including immediate reperfusion of the affected coronary artery [2,3,4,5]. ECG is a commonly used tool in the diagnosis of acute myocardial infarction [6]. The T-wave on an ECG typically represents ventricular repolarization [7,8]. Among ECG signs of acute cardiac ischemia, broad-based and tall tented T-waves, called hyperacute T-waves, may be the earliest and the only ECG sign of

transmural myocardial infarction<sup>6</sup>. ST-segment elevation is a marker of ongoing transmural ischemia secondary to acute coronary artery occlusion. On the contrary, ST-segment depression is usually a sign of subendocardial (non-transmural) ischemia [9]. We report herein a patient with ECG manifestations [Figure 1(a)] of hyperacute T-waves associated with severe transmural RWMA in the LAD artery territory detected on the simultaneously done bedside echocardiogram. The patient's ischemic chest pain completely subsided with near normalization of T-waves in V<sub>2</sub>-V<sub>6</sub> leads [Figure 1(b)] following emergency combo drug therapy (CDT) which includes oral dual antiplatelet drugs, IV tenecteplase and subcutaneous enoxaparin.



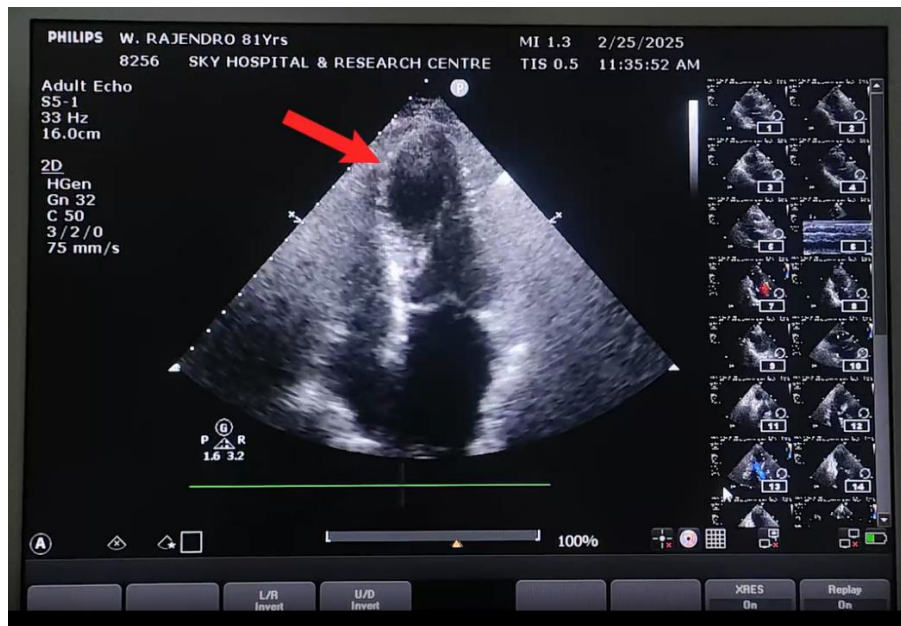
**Figure 1(a):** Patient’s first ECG on presentation to HAC, showing broad-based and tall tented T-waves inV<sub>2</sub>-V<sub>6</sub>, marked with arrow head.

**Case Presentation**

An 81 years old male with a history of chronic smoking was admitted to the HAC of SKY Hospital and Research Centre with sudden onset chest pain on February, 25, 2025 at 10:15am. Upon arrival to the HAC, physical examination was essentially normal.

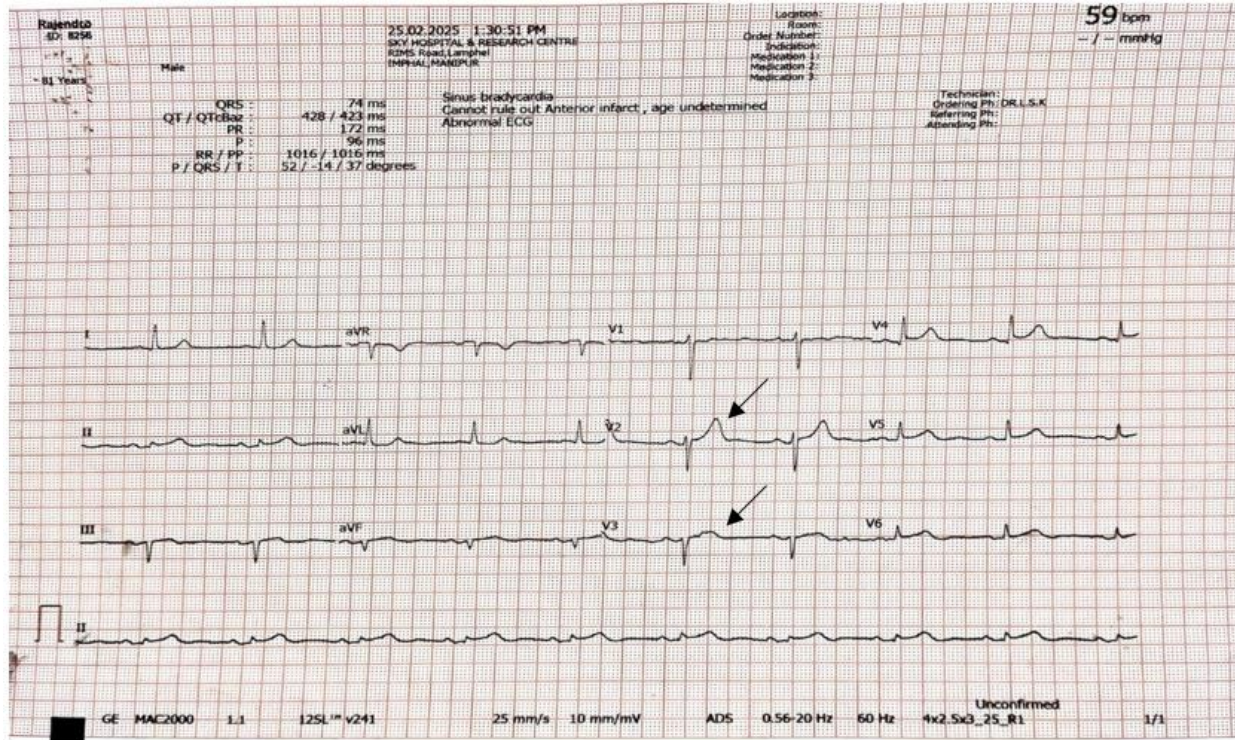
An ECG done simultaneously showed broad-based and tall tented T-waves, called hyperacute T-waves in V<sub>2</sub>-V<sub>6</sub> leads without ST-elevation. Blood sample was sent off for troponin T estimation.

A simultaneously done bedside echocardiogram revealed severe transmural wall motion abnormality in the LAD territory (Figure- 2a, the Echo video).



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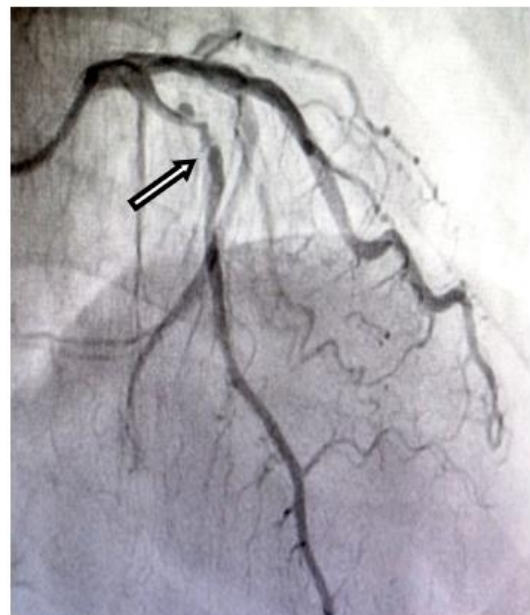
**Figure 2(a):** Echocardiogram performed at the time of presentation showing severe transmural wall motion abnormality in LAD territory (marked by an arrow head) with reduced LVEF (45%).



**Figure 1(b):** Patient's ECG done one hour after thrombolysis showing near normalization of T-waves and ST-segment in iso-electric line, marked with arrow head.

The echocardiogram also showed reduced left ventricular ejection fraction (LVEF) at 45%, moderate mitral regurgitation, mild aortic regurgitation, minimal tricuspid regurgitation, no pulmonary arterial hypertension and normal pericardium. Based on the symptom of ischemic chest pain, findings on ECG of hyperacute T-waves and severe transmural wall motion abnormality on bedside echocardiogram, immediately available pharmacological reperfusion regime consisting of dual antiplatelet drugs with intravenous tenecteplase and subcutaneous enoxaparin were administered to him immediately [10]. Intravenous bolus dose of enoxaparin was omitted considering his age of 81 years. His chest pain subsided within minutes of the CDT. Post-lysis ECG showed near normalization of T-wave changes, ST-segment remaining in iso-electric line (Figure 1b). The troponin T test done on presentation came back as normal although the treatment as for ATMI was already started before the result was available. The troponin T done 4 hours later has shown to be grossly elevated at 2000 ng/L. Further investigation revealed that he had dyslipidemia, hyperuricemia and thrombocytopenia (1,18,000/ $\mu$ l).

Subsequent coronary angiography showed 20% recanalisation of the proximal LAD obstruction (Figure. 3a, marked with arrow head). One drug-eluting stent (3.0 x 20.0 mm) was implanted across the lesion without complications (Figure. 3b, marked with arrow head). He was mobilized and discharged home in a stable condition. Echocardiogram at follow up two weeks later shows improvement of LVEF (50.6%).



**Figure 3 (a):** Patient's coronary angiogram after thrombolysis.

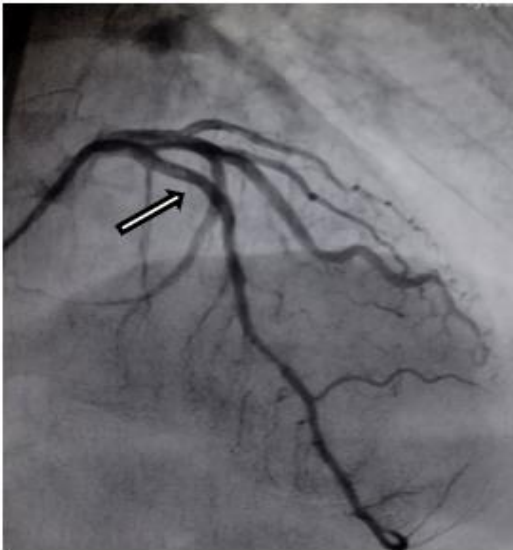


Figure 3 (b): Patient's Coronary Angiogram after stenting.

The ECG at 7-month follow up after discharge showed near normalization of T-waves and ST-segment in iso-electric line. Echocardiogram done on the same day showed improved LVEF at 53.4%. At 9-month follow up, the ECG showed normalization of T-waves (Figure. 1c, marked with arrow head) and ST-segment in iso-electric line. Echocardiogram on the same day showed normalization of LVEF at 56.9% (Figure 2b).

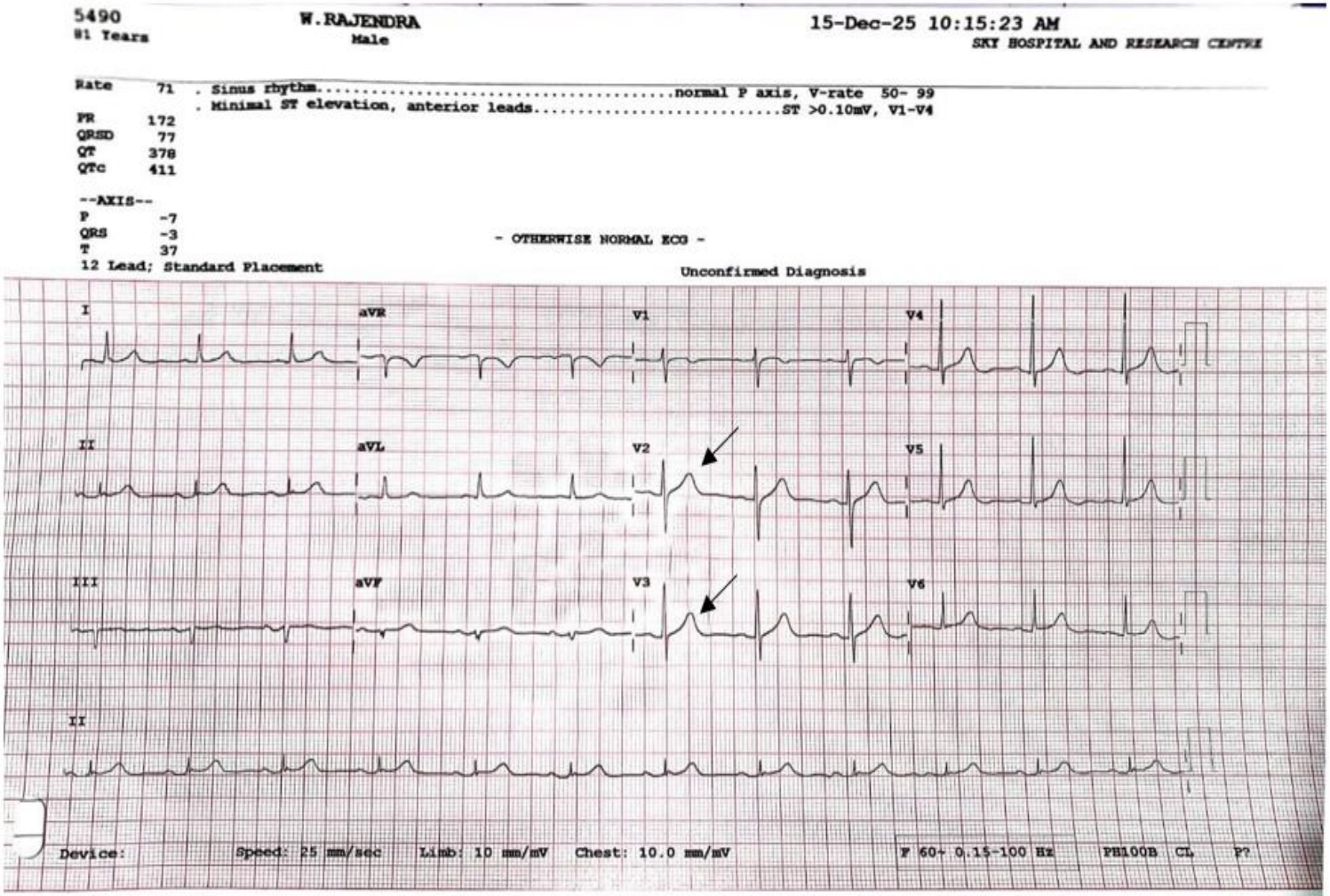
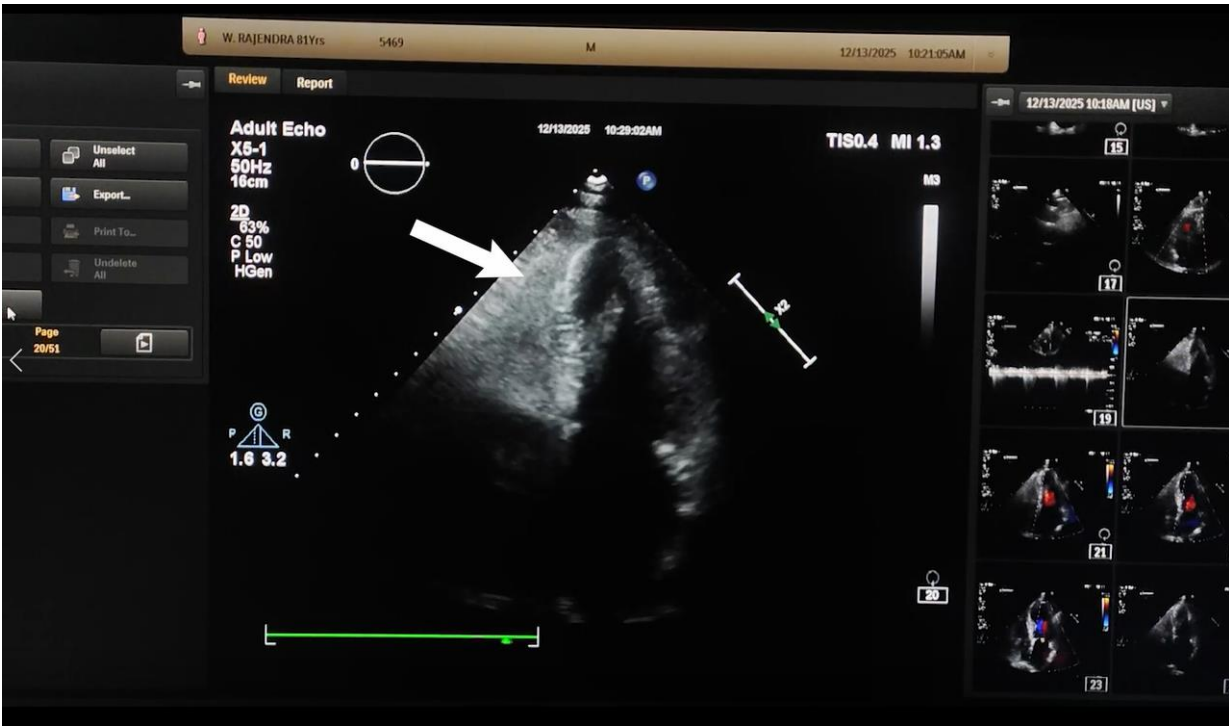


Figure 1(c): Patient's ECG at 9-month follow up after discharge showing normalization of T-waves and ST-segment in iso-electric line, marked with arrow head.



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**Figure 2(b):** Echocardiogram performed at 9-month follow up showed normalization of LVEF (56.9%).

## Discussion

The electrophysiological basis and clinical significance of the ECG pattern of persistent ‘hyperacute T-waves’ not evolving into ST-segment elevation in acute myocardial infarction are not completely known [11,12]. Some patients with an acute coronary occlusion may have an initial ECG without ST-segment elevation. Rarely, broad based and tall tented T-waves have been recognized as a transient early feature that changes into overt ST-elevation in the precordial leads [13]. Hyperacute T-waves have increased amplitude over the affected area and are more prominent, symmetric, and pointed (hyperacute) [13]. These changes in T-waves are usually present for only 5 to 30 minutes after the onset of the infarction and are followed by ST-segment changes [13]. The electrocardiographic differential diagnosis of the hyperacute T-waves includes transmural myocardial infarction, hyperkalemia as well as early repolarization, left ventricular hypertrophy, and acute myopericarditis [7]. ST-elevation myocardial infarction (STEMI) guidelines incorporated the knowledge, explaining that “in the very early phase of acute infarction, giant, hyperacute T-waves may be present with no ST-segment elevation,” and that “it is appropriate to administer thrombolytic therapy”. Thus, it should be clear that certain cases require experienced interpretation of the ECG before withholding reperfusion therapy at the earliest [14]. But most STEMI paradigms seem to exclusively focus on ST-elevation criteria and “displace” other ECG findings of acute coronary occlusion including hyperacute T-waves. The only mention in the current American STEMI guidelines is that “rarely, hyperacute T-waves changes may be observed in the very early phase of STEMI before the development of ST-elevation” [15]. Another review explained, “ST-elevation might be preceded by large ‘hyperacute’ T-waves, which might thus be a finding of early acute thrombotic occlusion.” [16] The European STEMI guidelines

consider hyperacute T-waves in the absence of ST-elevation as an indication for percutaneous coronary intervention (PCI) [4].

The diagnosis can sometimes become challenging when the patient is asymptomatic and has coincidental finding of hyperkalemia. Despite the well-known pseudoinfarction pattern of hyperkalemia, acute MI should be ruled out first. A high index of suspicion is needed, especially in high-risk patients. We think that in rare clinical situation when the diagnosis is in doubt, MI should be ruled in, as time has a high impact on patient mortality. An urgent bedside echocardiogram should be done to identify regional wall motion abnormality and prevent any delay in the initiation of appropriate therapy for ATMI[17]. One previous case report involving a patient who presented with hyperacute T-waves had transmural necrosis in the territory of LAD artery which was confirmed on cardiac magnetic resonance imaging performed during the same admission [18].

In our case the patient was given combo drug therapy within 10 minutes of ECG diagnosis of hyperacute T-wave changes and simultaneous echocardiogram finding of transmural wall motion abnormality in the LAD territory. This has led to complete relief of patient’s ischemic chest pain and near normalization of the ECG changes. As the facility was available, coronary angiogram was performed. A Drug-eluting stent (DES) across the affected segment of the LAD artery was implanted successfully. In-contrast, if the alternative means of mechanical reperfusion method i.e., PCI was chosen, it would have delayed at least 30 minutes to therapy during which time, hundreds and thousands of myocardial cells would have been irreversibly dead and LV function would have further deteriorated.

## Conclusión

Hyperacute T-waves on an ECG done for assessment of patients with suspected acute myocardial infarction is rare. However, when seen, it should be considered as the very first evidence of ATMI. The first ECG of our patient performed on presentation to the HAC of SKY Hospital and Research Centre showed typical hyperacute T-wave changes, accompanied by severe transmural myocardial wall motion abnormality in the LAD territory seen on the concurrent bedside echocardiogram. Prompt reperfusion therapy with combo drugs including oral dual antiplatelet drugs, IV tenecteplase and subcutaneous enoxaparin was thereby administered, leading to near normalization of the ECG changes within minutes of the reperfusion regime along with complete relief of patient's ischemic chest pain. Although it is the report of a rare case, lessons may be learned from it to not miss such ECG sign of acute transmural myocardial infarction.

This acute phase of ATMI is considered as the best time for initiation of any effective reperfusion therapy which can be initiated as soon as possible or within 10 minutes of diagnosis to achieve partial or full restoration of patency of the culprit coronary artery and to maximize salvage of myocardium in jeopardy with consequent increase of survival in the acute phase and in later years.

Our patient who is now 82 years of age, completed 12 months after the acute anterior transmural myocardial infarction and is fully back to his normal activity as on 28<sup>th</sup> February, 2026.

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