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Case Report

Atypical de Winter ECG: What is the Culprit?

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Abstract

Various ECG patterns can help determine the location of the occluded coronary artery and the extent of threatened myocardium. The reported positive predictive value for de Winter ECG in predicting acute left anterior descending artery occlusion is inconsistent. Additionally, the morphology of ST depression and other ECG findings may have varying degrees of severity and prognostic significance. This case demonstrates the importance of integrating multiple ECG findings, such as ST elevation, Q waves, and the R/S ratio, with the location of the de Winter pattern on various ECG leads to accurately predict the culprit artery and assess the anatomical extent of myocardial ischemia.

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Introduction

The de Winter ECG pattern is an ST-segment elevation myocardial infarction (STEMI) equivalent, characterized by upsloping ST-segment depression in leads V_1–V_6 and tall, peaked T waves. Although it has a high predictive value for acute occlusion of the left anterior descending artery (LAD), the reported positive predictive value for this pattern in predicting acute LAD occlusion is inconsistent.

Furthermore, relying solely on ST depression (STD) in the precordial leads to predict the culprit artery can be misleading since other reports have identified non-LAD culprits, such as the right coronary artery (RCA), the left circumflex artery (LCx), and the LAD diagonal branch, exhibiting ECG changes similar to the de Winter pattern.[1] Here, we present a case of posterolateral STEMI mimicking an atypical de Winter ECG pattern.

Case Report

A 40-year-old male heavy smoker presented to the emergency department with a 7-hour history of oppressive chest pain and diaphoresis. His past medical history was unremarkable, although he had a first-degree family history of coronary artery disease. Upon examination, his blood pressure was 118/70 mm Hg, and his heart rate was 98 bpm, with no other significant findings.

Troponin I levels were elevated at 4.46 ng/mL (reference range: 0-0.1 ng/mL), indicating myocardial injury. A 12-lead

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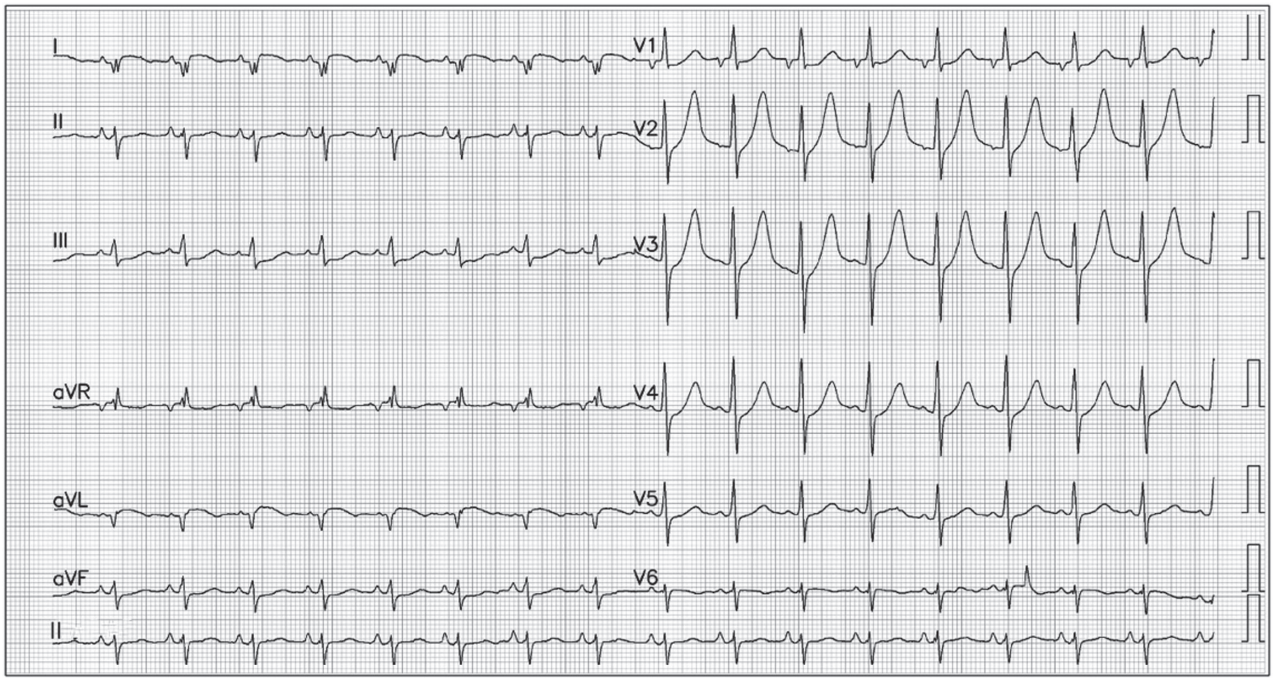

Figure 1. The image presents the patient's initial 12-lead ECG.

ECG was performed (Figure 1). The initial ECG showed abnormal Q waves and slight ST elevation (STE) in leads I and aVL, along with STD in leads II and aVF. In addition, an upsloping STD of up to 2.0 mm with tall, symmetrical T waves was observed in leads V_2–V_4.

Upsloping STD by ≥0.1 mV at the J point in leads V_1–V_6 and tall, symmetrical T waves are the principal criteria for the de Winter ECG changes.[2] We initially identified the patient's ECG as de Winter ECG, indicative of extensive anterior myocardial infarction. Based on this pattern, an acute occlusion of the LAD artery was suspected. An alternative explanation for these ECG changes could be a high lateral myocardial infarction with total occlusion in the first diagonal branch of the LAD. In such cases, STE may be observed in leads I and aVL, while the de Winter-like pattern may solely represent subendocardial ischemia in the anterior wall. However, the presence of prominent R waves in leads V_1–V_2 should raise suspicion that they might reflect the formation of Q waves in the posterior leads. In combination with STE in leads I and aVL, these findings suggested that posterolateral myocardial infarction was the most probable cause of this atypical de Winter pattern.

Coronary angiography (CAG) revealed a total occlusion of the proximal LCx (Figure 2A), while the RCA exhibited insignificant stenosis. Primary percutaneous coronary intervention was performed with drug-eluting stent implantation in the LCx (Figure 2B), leading to complete resolution of the patient's chest pain. A subsequent 15-lead posterior ECG showed complete resolution of the ST to baseline in leads V_2–V_4. Nonetheless, abnormal Q waves and STE were observed in posterior leads V_7–V_9, along with more prominent STE in leads V_5–V_6 (Figure 3). These findings further supported the diagnosis of posterolateral myocardial infarction mimicking an atypical de Winter ECG pattern.

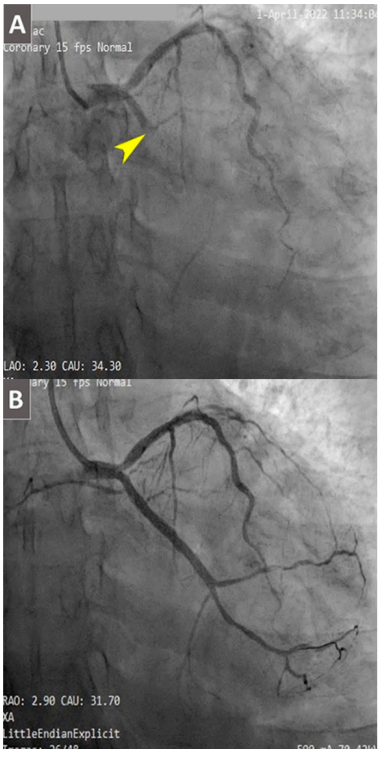

Figure 2. A) The patient's coronary angiogram reveals a total occlusion of the proximal left circumflex artery (yellow arrow).
B) The image illustrates primary percutaneous coronary intervention with thrombus aspiration, followed by the implantation of a drug-eluting stent (3.5×20 mm) in the proximal left circumflex artery.

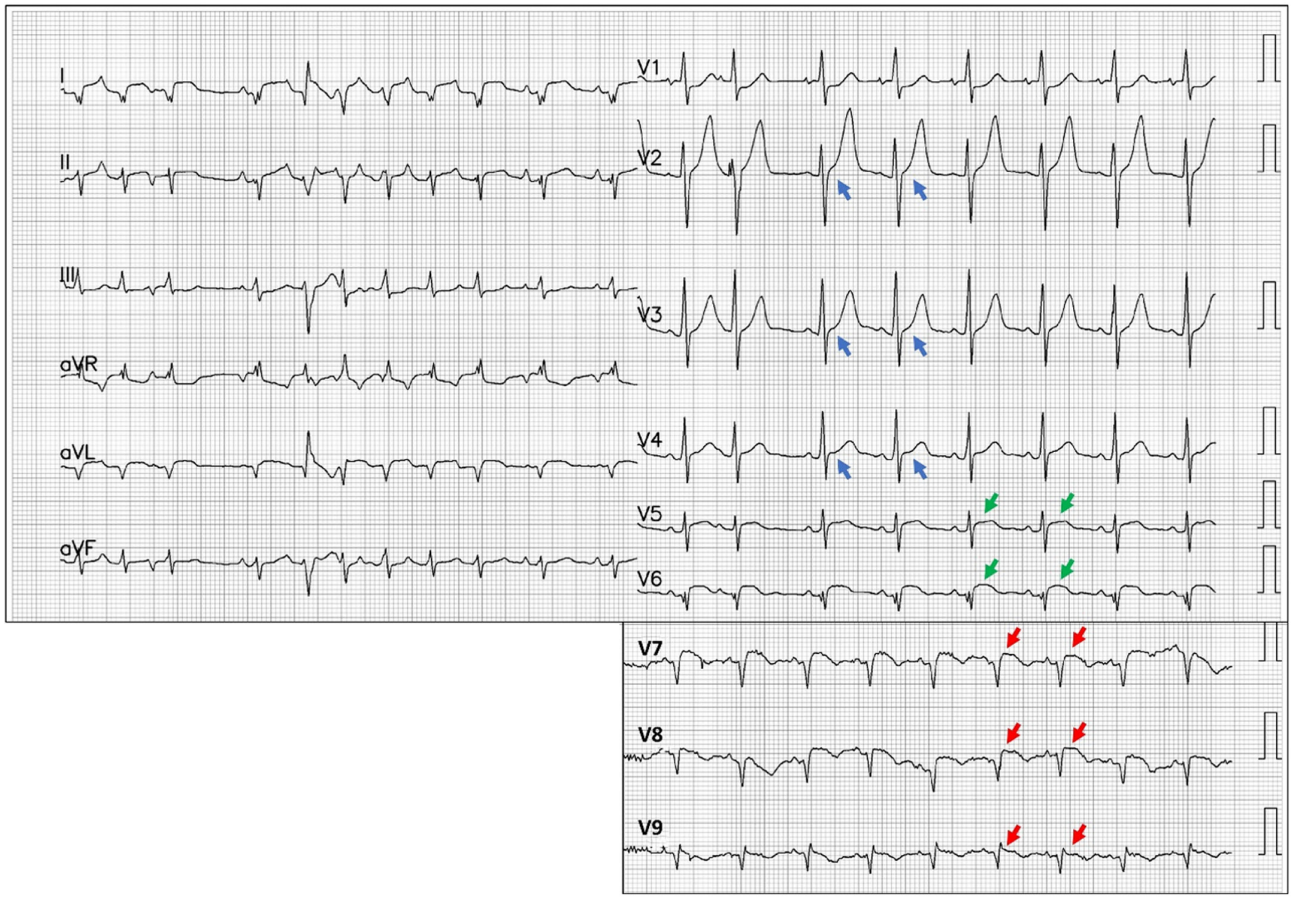

Figure 3. Post-percutaneous coronary intervention 15-lead posterior ECG shows complete resolution of the ST segment to baseline in V_2–V_4 (blue arrows). Still, abnormal Q wave and ST-segment elevation are detectable in posterior leads V_7–V_9 (red arrows) and more apparent ST-segment elevation in V_5–V_6 (green arrows).

Discussion

The de Winter ECG pattern is recognized as a STEMI equivalent, with a high predictive value for acute occlusion of the (LAD artery (range: 89%–98%). In most patients with LAD as the culprit artery, the core ischemia leads exhibiting maximal STD are typically located at V_2 or V_3. Nevertheless, relying solely on STD in the precordial leads to predict the culprit artery can be misleading since multiple reports have identified non-LAD culprits, such as the RCA, the LCx, and the LAD diagonal branch, exhibiting ECG changes similar to the de Winter pattern.[1] Consequently, in order to accurately predict the culprit artery and assess the anatomical extent of myocardial ischemia, it is essential to consider other ECG findings, such as STE, Q waves, and the R/S ratio, in conjunction with the location of the de Winter pattern in various ECG leads.

In addition to the classic de Winter pattern, our patient exhibited other ECG changes, including subtle STE in leads I and aVL, along with STD in leads II and aVF. As previously mentioned, several factors could contribute to these patterns. Abnormal Q waves and STE in leads I and aVL, coupled with the de Winter ECG pattern, initially raised suspicion of occlusion in the proximal LAD, potentially affecting a large anterior area of the left ventricle. Additionally, tall and prominent R waves (R/S ratio >1) in leads V_1–V_2 could indicate pathological R waves in the setting of posterior infarction (Q-wave equivalent), reflecting the formation of Q waves in the posterior leads. Furthermore, upsloping STD and tall, symmetrical T waves in leads V_2–V_4 may mirror STE and inverted T waves in the posterior leads.

Thygesen et al[3] suggested that isolated STD≥0.5 mm in leads V_1–V_3 could be indicative of inferobasal myocardial ischemia, potentially indicating LCx occlusion. This is particularly true when the terminal T wave is positive, which can be best observed using the posterior leads. Diagnosing posterior myocardial infarction can be challenging in the absence of traditional ECG infarct signs, such as STE, potentially leading to misdiagnosis or delayed treatment. Nonetheless, with a working diagnosis of STEMI equivalent in this patient, we conducted urgent CAG, which revealed a total occlusion in the proximal LCx.

The LCx is responsible for supplying blood to the posterior (inferobasal) and lateral aspects of the left

ventricle. Consequently, occlusion at this location can result in a posterolateral myocardial infarction presentation, which may explain the atypical ECG findings observed in this case.[4] The postprocedural ECG demonstrated STE in the posterior leads and more prominent STE in lateral leads V_5–V_6, confirming the diagnosis of posterolateral myocardial infarction mimicking a de Winter ECG pattern. Moreover, echocardiographic evidence of hypokinesis at the basal-mid-apical anterolateral and inferolateral segments further corroborated the anatomic location of this myocardial infarction.

The literature provides reports of different presentations of atypical de winter ECG patterns. Wang et al[5] reported a case of STEMI equivalent with ECG changes mimicking an atypical de Winter pattern. Their patient's initial ECG revealed slight STE in leads I and aVL and T-wave inversion in the inferior leads. A subsequent follow-up ECG showed an upsloping STD of up to 6 mm at the J point in leads V_1–V_6, I, and aVL, along with tall symmetrical T waves in leads V_2–V_5, a 1 mm J-point elevation in lead aVR, and J-point elevation in the inferior leads. CAG demonstrated a total occlusion of the proximal right coronary artery with collateral circulation from the left coronary artery and a subtotal occlusion of the LCx, with no significant lesions in the LAD or the left main artery. Other case reports have shown atypical de Winter presentations of LAD occlusion.[6, 7] Kainat et al[6] described an ECG with tall, prominent, and symmetrical T waves in the precordial leads accompanied by minor concave STE and STE<0.5 mm in lead aVR, without STD in the precordial leads. CAG revealed a subtotal occlusion of the LAD in this case, highlighting the variability in ECG findings associated with coronary artery occlusion. Yang et al[7] presented another case of atypical de Winter ECG pattern with a delayed presentation. The initial ECG demonstrated slight STE in lead aVR and STD in the inferior leads. Five hours after symptom onset, the ECG showed a significant upsloping STD at the J point in precordial leads V_3–V_6, slight STE in aVR, and depression in the inferior leads. CAG confirmed proximal LAD occlusion, emphasizing the importance of continuous ECG monitoring and considering the de Winter ECG pattern in the differential diagnosis of acute coronary syndromes, even when initial ECG changes are subtle.

Other ECG signs can help differentiate the atypical de Winter pattern from its typical form. For instance, ST-segment elevation in lead aVR is indicative of proximal LAD or left main coronary artery as the culprit vessel.[8, 9] A tall R wave in lead V_1 is a strong indicator against LAD involvement since it could signify pathological Q waves in posterior infarction resulting from occlusion of the RCA or the LCx.[3,10] Conversely, the absence of inverted T waves in the anterior leads during follow-up ECG typically does not support LAD reperfusion.[11] Additionally, the presence of a typical de Winter pattern in the inferolateral leads (leads II, III, aVF, and V_4–V_6), along with STE in lead aVR, can be indicative of RCA occlusion.[12]

Conclusion

The de Winter ECG pattern, a STEMI equivalent, has a high predictive value for acute LAD occlusion and necessitates urgent CAG. In some cases, posterolateral myocardial infarction may mimic the atypical de Winter ECG pattern, making accurate diagnosis challenging.

To enhance diagnostic accuracy, it is essential to evaluate other ECG findings, such as STE, Q waves, and the R/S ratio, along with the location of the de Winter pattern in various ECG leads. This comprehensive approach can help identify the culprit artery and determine the extent of myocardial ischemia. In patients with a high clinical suspicion of acute LCx occlusion, atypical de Winter ECG pattern, or inconclusive ECG findings regarding the anatomical location of myocardial infarction, recording the posterior leads is strongly recommended for improved diagnostic precision.

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