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CASE REPORT

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De Winter syndrome in action: Captured on defibrillator

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Key Clinical Message

De Winter syndrome, though rare, demands heightened clinical suspicion. Recognizing its electrocardiographic subtleties is crucial, as prompt diagnosis allows for life-saving interventions such as percutaneous coronary intervention or thrombolysis.

KEYWORDS

De Winters syndrome, electrocardiography, primary percutaneous coronary intervention, ST-elevation myocardial infarction

1 | INTRODUCTION

In the realm of cardiovascular medicine, the vast spectrum of cardiac disorders continues to pose challenges to clinicians worldwide. One such intriguing entity that has gained recognition in recent years is De Winter syndrome, a unique electrocardiographic pattern associated with critical proximal left anterior descending (LAD) coronary artery occlusion. First described by Prof. Dr. R.J. de Winter in 2008, this syndrome represents a distinct electrocardiographic finding that can be easily misinterpreted, leading to potential diagnostic pitfalls and delayed intervention.[1,2] De Winter syndrome, also referred to as "upsloping ST-segment elevation at the J point," is characterized by a specific electrocardiographic pattern that mimics a benign early repolarization variant at first glance. However, this seemingly innocuous appearance belies the underlying ominous pathology of an acute myocardial infarction involving the proximal LAD coronary artery.[3] The hallmark of De Winter syndrome is the presence of prominent upsloping ST-segment elevation at the J point in the precordial leads (typically V1 to V6) during chest pain or other ischemic symptoms, often accompanied by reciprocal ST-segment. Clinically, patients with De Winter syndrome frequently present with symptoms suggestive of acute coronary syndrome, such as severe chest pain, diaphoresis, and shortness of breath. However, the electrocardiographic findings can be deceptively subtle, posing a diagnostic challenge for healthcare providers. The absence of classic ST-segment elevation in the conventional leads may lead to underestimation of the severity of the coronary artery occlusion and, consequently, delays in appropriate management.[4] As we delve into the case report that follows, it is imperative to recognize the diagnostic nuances and challenges associated with De Winter syndrome. Early identification of this syndrome is pivotal, as timely reperfusion therapy can significantly impact patient outcomes and reduce the risk of myocardial infarction-related complications.

2 | CASE PRESENTATION, HISTORY, AND EXAMINATION

A middle-aged male presented to the emergency room with shortness of breath persisting for over 5 h, accompanied

by chest tightness and pain. The patient, with no past medical history, had undergone a minor surgery (abscess evacuation in the gluteal region) the day before, under local anesthesia. He denied any substance abuse but was a current smoker with a 20-year history of smoking 30 cigarettes per day. On examination, he appeared diaphoretic and dyspnoeic. He was fully oriented, with vital signs revealing a body temperature of 36.9°C, pulse at 88 beats/min, respiratory rate at 22 breaths/min, and blood pressure at 110/77 mmHg. Regular heart rhythm with normal heart sounds was noted, and lung and abdomen examinations were unremarkable.

3 | DIFFERENTIAL DIAGNOSIS, INVESTIGATIONS, AND TREATMENT

A rapid ECG displayed hyperacute T wave elevation in the precordial leads (Figure 1) and blood tests revealed serum troponin significantly elevated at 9.3 ng/mL (Table 1).

One hour post-presentation, the patient suddenly collapsed, prompting CPR. Defibrillation was administered five times due to runs of ventricular tachycardia and fibrillation. Intravenous magnesium sulfate and lidocaine were given to suppress arrhythmias. CPR continued for 5 min before intubation. Up sloping ST depression and peaked T wave on the defibrillator monitor indicated ST-segment elevation myocardial infarction (STEMI) equivalent to De Winter syndrome (Figure 2). The patient, intubated and sedated, was transferred to the catheter laboratory where coronary angiography revealed a proximal total occlusion of the left anterior descending artery (LAD) (Video S1, Figure 3A).

A stent was successfully inserted and the blood flow returned as normal (Video S2, Figure 3B).

4 | OUTCOME AND FOLLOW-UP

An ECG, 30 min post-percutaneous coronary intervention showed normal ST segments and sinus rhythm, indicating successful revascularization (Figure 4).

The patient was admitted to the intensive care unit (ICU) under the care of a multidisciplinary team comprising an anesthesiologist, cardiologist, and specialist nurse for meticulous management and continuous monitoring. Throughout the ICU stay, the patient's vital signs remained within normal ranges.

The prescribed medications during hospitalization included aspirin 100 mg once daily, ticagrelor 90 mg twice daily, bisoprolol 5 mg once daily, lisinopril 5 mg once daily, and atorvastatin 80 mg once daily, aiming at comprehensive cardiac care and risk reduction.

After 2 days of admission, a decision was made to initiate the weaning process from the ventilator. The trial proved successful, leading to the discontinuation of ventilator support.

Neurological examinations revealed no focal neurological deficits, and the patient reported being free from pain. Subsequently, the patient was discharged from

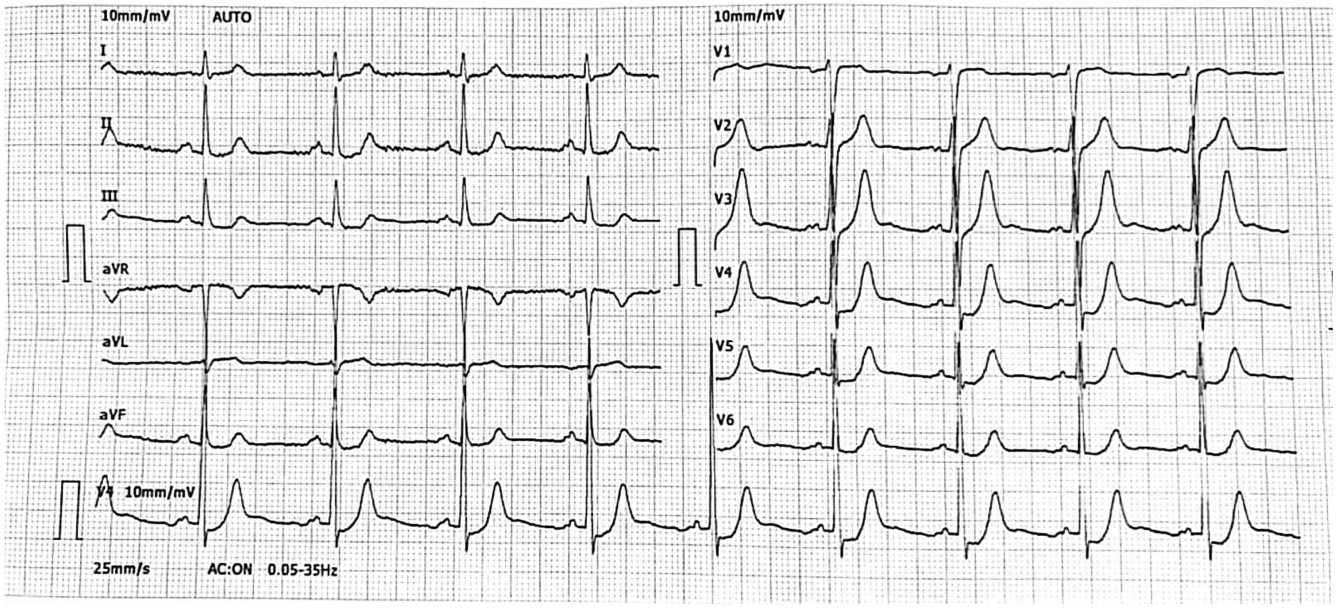

FIGURE 1 Emergency ECG showing hyperacute T wave.

TABLE 1 Blood tests revealing elevated s. troponin.

Test	Result	Normal value
Hemoglobin	15.7	12–15 g/dL
White blood cell	13.2	$4.3–9.5 \times 10^3/\mu L$
Platelets	330	$150–415 \times 10^3/\mu L$
Blood urea	33	10–45 mg/dL
Serum creatinine	1.1	0.4–1.1 mg/dL
Random Blood Sugar	122	140–200 mg/dL
Serum troponin I	9.3	0–0.04 ng/mL

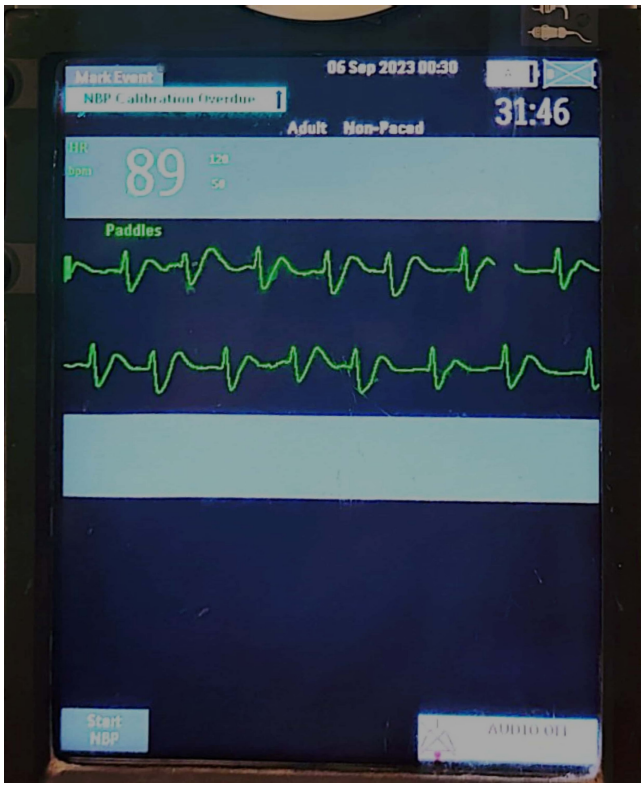

FIGURE 2 Up sloping ST depression and peaked T wave on defibrillator device.

the hospital with a full course of medical treatment and scheduled for weekly follow-up appointments to ensure continued recovery and address any potential concerns.

A month post-coronary intervention, the patient underwent an electrocardiogram (ECG), which demonstrated normal results (Figure 5), indicating positive progress in cardiac function and recovery.

The comprehensive approach taken by the healthcare team, including the coordinated efforts of specialists, meticulous medication management, successful weaning from the ventilator, and ongoing monitoring, contributed to the patient's positive outcome and subsequent discharge.

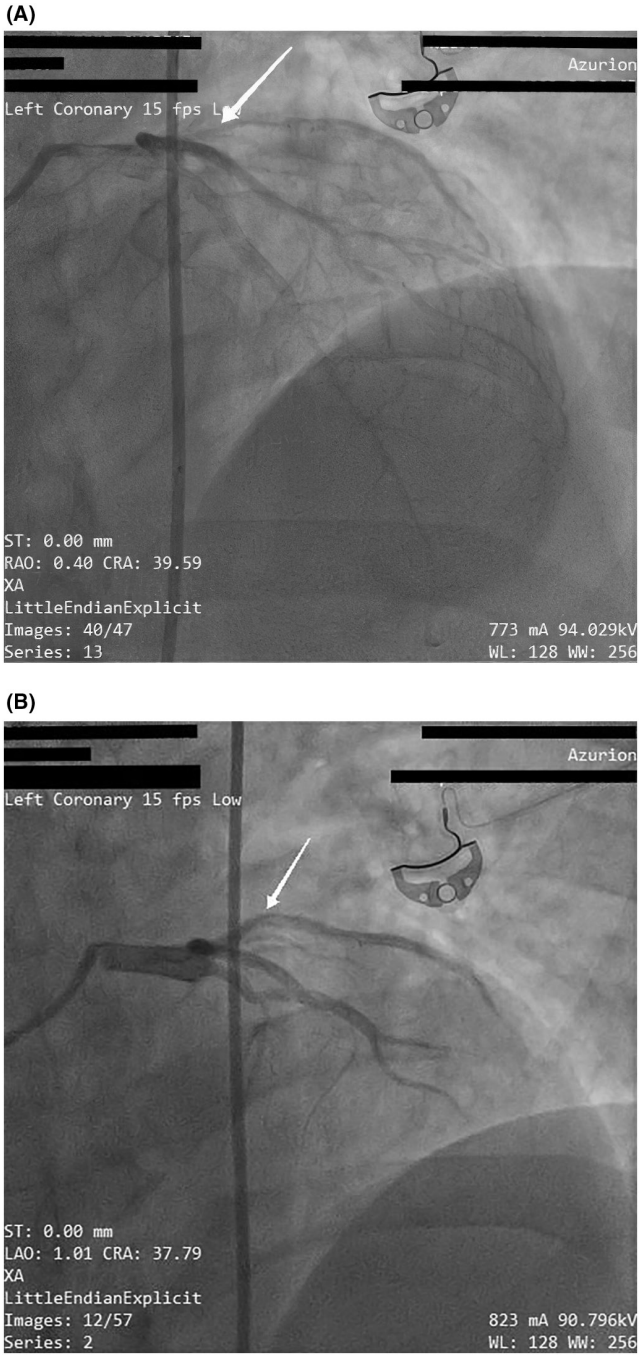

FIGURE 3 (A) proximal total LAD occlusion (white arrow). (B) Return of LAD blood flow after stent implantation (white arrow).

5 | DISCUSSION

De Winter syndrome, despite its increasing recognition, often remains underdiagnosed in clinical settings due to its relatively rare occurrence. The subtlety of its electrocardiographic manifestations poses a challenge, as it can mimic benign early repolarization, emphasizing the need for heightened clinical suspicion, particularly when

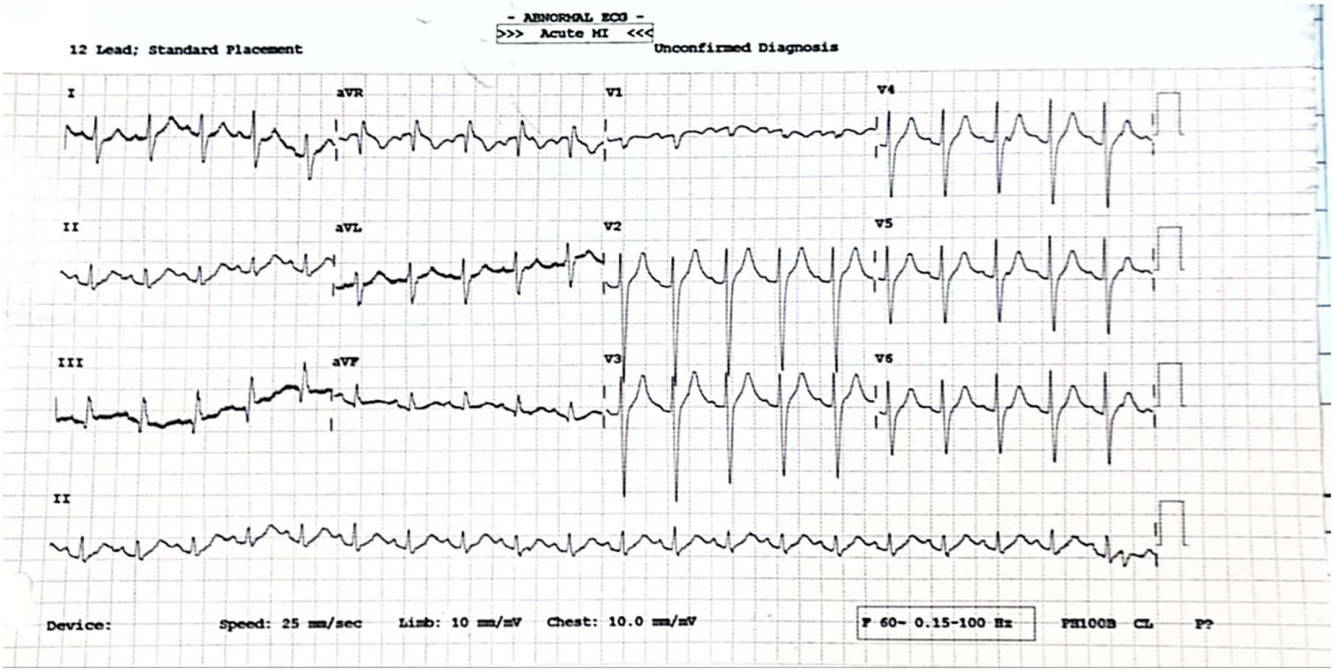

FIGURE 4 ECG 30 min after primary percutaneous intervention.

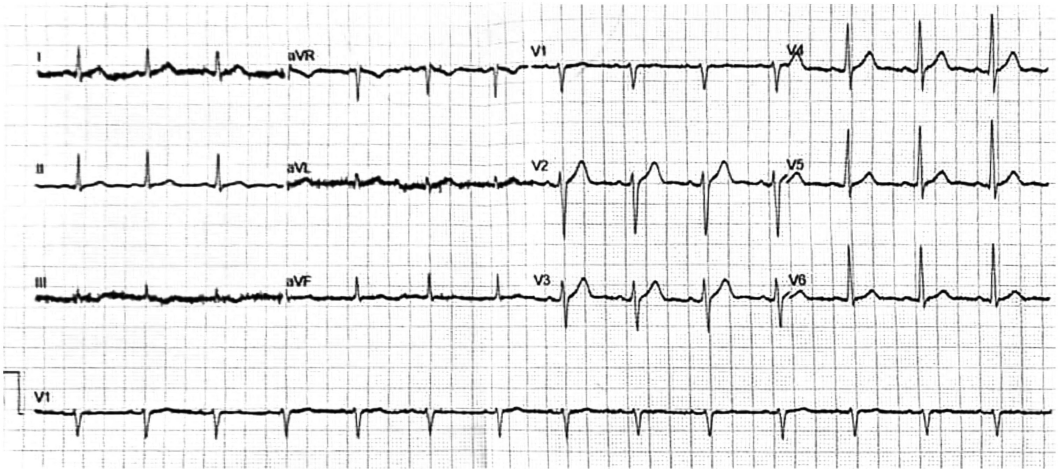

FIGURE 5 ECG 1 month after primary percutaneous intervention.

dealing with patients presenting atypical symptoms or electrocardiographic changes.[5]

The pathophysiology underlying De Winter syndrome is primarily associated with the critical occlusion of the proximal left anterior descending (LAD) coronary artery, a major vessel supplying the anterior portion of the heart.[6] This occlusion typically occurs during the early phases of acute myocardial infarction, leading to the distinctive electrocardiographic pattern observed in De Winter syndrome. The importance of understanding the pathophysiological basis of this syndrome cannot be overstated, as it is crucial for achieving a prompt diagnosis and initiating life-saving interventions.

Prompt recognition is essential for timely intervention, and this includes options such as percutaneous coronary intervention (PCI) or thrombolytic therapy.[7] PCI, in particular, involves the mechanical restoration of blood flow in the affected coronary artery, significantly improving the chances of myocardial salvage. Thrombolytic therapy, on the other hand, utilizes medications to dissolve the clot causing the occlusion, serving as an alternative in certain situations.

Beyond the acute phase, long-term management involves addressing underlying cardiovascular risk factors and instituting appropriate pharmacotherapy. Continuous monitoring and follow-up care are imperative to ensure the patient's recovery and to prevent potential complications.

6 | CONCLUSIONS

In conclusion, De Winter syndrome stands as a captivating entity within the spectrum of acute coronary syndromes, demanding vigilant attention from healthcare providers. The delicate balance between recognizing its subtle electrocardiographic features and the urgency for prompt intervention underscores the significance of understanding this syndrome. This case report aims to shed light on a clinical scenario involving De Winter syndrome, emphasizing the importance of astute clinical acumen and electrocardiographic interpretation in the timely management of this potentially life-threatening condition.

AUTHOR CONTRIBUTIONS

Ahmed Qasim Mohammed Alhatemi: Conceptualization; data curation; formal analysis; funding acquisition; investigation; methodology; project administration. **Hashim Talib Hashim:** Conceptualization; data curation; funding acquisition; methodology; project administration; resources; software; validation; visualization. **Ezzat Mohammed Hussain Aziz:** Conceptualization; data curation; formal analysis; funding acquisition; resources; software; supervision; validation. **Tiba Khaild Abdulhussain:** Conceptualization; formal analysis; funding acquisition; supervision; validation; visualization; writing – review and editing. **Ali Talib Hashim:** Conceptualization; data curation; investigation; software; supervision; writing – original draft.

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CONFLICT OF INTEREST STATEMENT

The authors declare that they have no competing interests.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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