





The de Winter's pattern revisited: a case series

Thadathilankal-Jess John *, Alfonso Pecoraro , Hellmuth Weich ,
Lloyd Joubert , Bradley Griffiths, and Philip Herbst 

Division of Cardiology, Department of Medicine Stellenbosch University & Tygerberg Academic Hospital PO Box 241, Cape Town 8000, South Africa

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Background

The de Winter's electrocardiogram (ECG) pattern signifying proximal left anterior descending (LAD) artery occlusion was first described in 2008. The ECG changes were thought to be static and mechanisms for this were suggested. In addition, the optimal management of these patients was reported to be via a primary percutaneous coronary intervention (PCI) strategy.

Case summary

Case 1: A 48-year-old gentleman presented with a 2-h history of ischaemic chest pain with initial de Winter's pattern on ECG. This progressed to anterior ST-elevation myocardial infarction (STEMI) complicated by ventricular fibrillation. Emergency angiography revealed a mid-vessel LAD occlusion which was successfully reperfused. **Case 2:** A 34-year-old female presented with a 2-h history of ischaemic chest pain with initial ECG showing a de Winter's pattern. Due to concerns of performing PCI timeously, a pharmacoinvasive strategy of reperfusion was adopted with resolution of the de Winter's pattern. Urgent angiography revealed a proximal LAD lesion which was successfully stented.

Discussion

The two cases highlight that the de Winter's pattern may in fact not be static, but rather lie along the continuum of ischaemia and may evolve into STEMI. In addition, we provide further evidence that if primary PCI cannot be offered in a timeous manner, thrombolytic therapy may be considered in such patients. The de Winter's pattern remains a high-risk ECG pattern that requires early recognition and intervention.

Keywords

De Winter's ECG • STEMI • Pharmacoinvasive strategy • LAD occlusion • High-risk ECG • Case series

Learning points

- The de Winter's electrocardiogram (ECG) should be considered a high-risk ECG and should potentially be seen as an ST-elevation myocardial infarction equivalent with a strong association with an occluded artery.
- The de Winter's pattern may lie somewhere along the continuum of ischaemic ECG changes between subendocardial ischaemia and the transmural injury pattern associated with ST-elevation.
- Early reperfusion is paramount with good success noted with both a pharmacoinvasive or early percutaneous approach.

Introduction

De Winter *et al.*¹ first described the novel electrocardiogram (ECG) de Winter's pattern signifying proximal left anterior descending

(LAD) occlusion in 2008. The initial description identified 1–3 mm upsloping ST-segment depression at the J-point in leads V1–6 that continued into tall, positive symmetrical T-waves. In most cases, there was concomitant 1–2 mm ST-segment elevation in lead aVR.

* Corresponding author. Tel: +27 767702123, Email: jessjohnt@gmail.com

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The QRS complexes were usually not widened and in some cases, there was loss of praecordial R-wave progression.^{1,2} This pattern was described in 2% of cases of anterior myocardial infarction studied by the group. These features were described as static, with persistence of the ECG findings till the time of angiography which confirmed LAD occlusion.¹

Recent literature has however contested the pathophysiological mechanisms, static nature as well as the location of disease in patients with the de Winter's pattern on ECG.^{3–9} The optimal management of these patients is also not clearly defined. We describe two cases to highlight some of the described differences.

progression to an anterior ST-elevation myocardial infarction (STEMI) after 7 h (*Figure 1B*). This was complicated by ventricular fibrillation and cardiac arrest. The patient was successfully defibrillated without subsequent need for inotropic support or intubation and transferred for emergency angiography which revealed a mid-vessel LAD occlusion which was successfully reperfused (*Supplementary material online, Video S1*). Peak highly sensitive troponin I level was measured at 16 833 ng/L (normal < 14 ng/L) and echocardiography revealed a left ventricular ejection fraction (LVEF) of 30–35% with regional wall motion abnormalities (RWMA) within the LAD territory (*Supplementary material online, Video S2*). He had an uneventful stay

Timeline

Patient 1

2 h prior	Maximal ischaemic type chest pain
First medical contact	De Winter's pattern on electrocardiogram (ECG) noted—started on non-ST-elevation acute coronary syndrome protocol
7 h after admission	Progression of ECG to anterior ST-elevation myocardial infarction and subsequently developing ventricular fibrillation arrest (requiring defibrillation) Peak troponin: 16 833 ng/L Echocardiogram: left anterior descending (LAD) territory hypokinesia [ejection fraction (EF): 30–35%]
Emergency angiography	LAD occlusion (TIMI 0 Flow)—successfully reperfused
Day 2 post-percutaneous coronary intervention (PCI)	Discharged home
3 months follow-up	Clinically no failure, diuretic therapy stopped Echocardiogram: normalization of left ventricular ejection fraction (LVEF) (EF 50–55%)

Patient 2

2 h prior	Maximal ischaemic type chest pain
First medical contact	De Winter's pattern on ECG—Streptokinase infusion started
30 min into fibrinolytic therapy	Reperfusion rhythm on ECG Peak troponin: 2834 ng/L Echocardiogram: LAD territory hypokinesia (EF: 30–35%)
Urgent angiography	Severe proximal LAD lesion (TIMI II flow)—successfully stented
Day 2 post-PCI	Discharged home
3 months follow-up	Clinically no failure, diuretic therapy stopped Echocardiogram: normalization of LVEF (EF 50–55%)

Case presentation

Patient 1

A 48-year-old gentleman with dyslipidaemia, but not on any medical therapy, presented with a 2-h history of typical ischaemic chest pain to the emergency department. Initial ECG showed evidence of a de Winter's pattern (*Figure 1A*). This was managed by the treating physician as a non-ST-elevation acute coronary syndrome (NSTEMI-ACS) and was started on oral aspirin and clopidogrel, intravenous low-molecular weight heparin and medical therapy as recommended for NSTEMI-ACS.¹⁰ Initial clinical evaluation revealed normal auscultatory findings of the cardiovascular system, but with evidence of Killip Class II heart failure. The patient had ongoing chest pain with subsequent

in the ward and was discharged 2 days after percutaneous coronary intervention (PCI) on dual antiplatelet therapy (DAPT) [oral aspirin (lifelong), oral clopidogrel (6 months)], and lifelong simvastatin, enalapril, atenolol, and furosemide. Subsequent follow-up at 3 months revealed an improvement of LVEF to low-normal (50–55%) with discontinuation of diuretic therapy.

Patient 2

A 34-year-old female with a strong smoking history (>25 pack years), dyslipidaemia, and increased body mass index, but not on any medical therapy, presented with a 2-h history of typical ischaemic chest pain to the emergency department of a non-PCI centre. Initial ECG showed evidence of a de Winter's pattern (*Figure 2A*) with slight ST-



Figure 1 (A) Standard 12-lead electrocardiogram. Praecordial leads demonstrate the de Winter's pattern. (B) Progression to anterior ST-elevation myocardial infarction.

elevation in the septal leads V1, V2. Initial clinical evaluation revealed a normal cardiovascular examination with no clinical evidence of heart failure. Due to ongoing severe chest pain, the inability to transfer the patient to the closest PCI centre within 120 min and the close association between the de Winter's and an occluded artery, a pharmacoinvasive strategy for reperfusion was adopted. The patient was given 1.5 million units of streptokinase which was infused over a period of an hour. In addition, the patient was started on oral aspirin and clopidogrel, intravenous low-molecular weight heparin and medical therapy as recommended for the management of STEMI.¹¹ During the course of thrombolytic therapy, the patient developed a reperfusion rhythm in the form of an accelerated idioventricular rhythm with subsequent resolution of chest pain and disappearance of the de Winter's pattern on the ECG (Figure 2B). The patient was transferred to our PCI-capable centre with angiography confirming a severe proximal LAD lesion with evidence of plaque rupture which had reperfused and was successfully stented (Supplementary material online, Video S3). Peak highly sensitive troponin I level measured 2834 ng/L (normal < 14 ng/L) and echocardiography revealed an ejection fraction of 30–35% with RWMA within the LAD territory (Supplementary material online, Video S4). She had an uneventful stay in the ward and was discharged 2 days after PCI on DAPT [oral aspirin (lifelong), oral clopidogrel (6 months)], and lifelong simvastatin

enalapril, atenolol, and furosemide. Subsequent follow-up at 3 months revealed an improvement of LVEF to low-normal (50–55%) with discontinuation of diuretic therapy.

Discussion

The two cases presented here offer support for the status of de Winter's ECG pattern as an STEMI equivalent in terms of demonstrating a tight association with an occluded artery. The de Winter's pattern has classically been described in proximal LAD occlusion. This has been disputed with various reports and reviews describing the pattern in patients with isolated occlusions of the left circumflex or its branches, the right coronary artery (RCA), a large first diagonal branch or even without coronary occlusion at the time of coronary angiography.^{3–5} Wall et al.⁶ identified the de Winter's pattern in 8 of 1429 patients undergoing coronary angiography. They identified that the LCx was involved in 50% of cases and the RCA and LAD in only 25% of cases each.

The enigma surrounding the de Winter's pattern has been framed as the presence of a static ECG pattern that would typically not progress to ST elevation, despite the strong association with an occluded artery. Various pathogenic mechanisms for this lack of ST-elevation

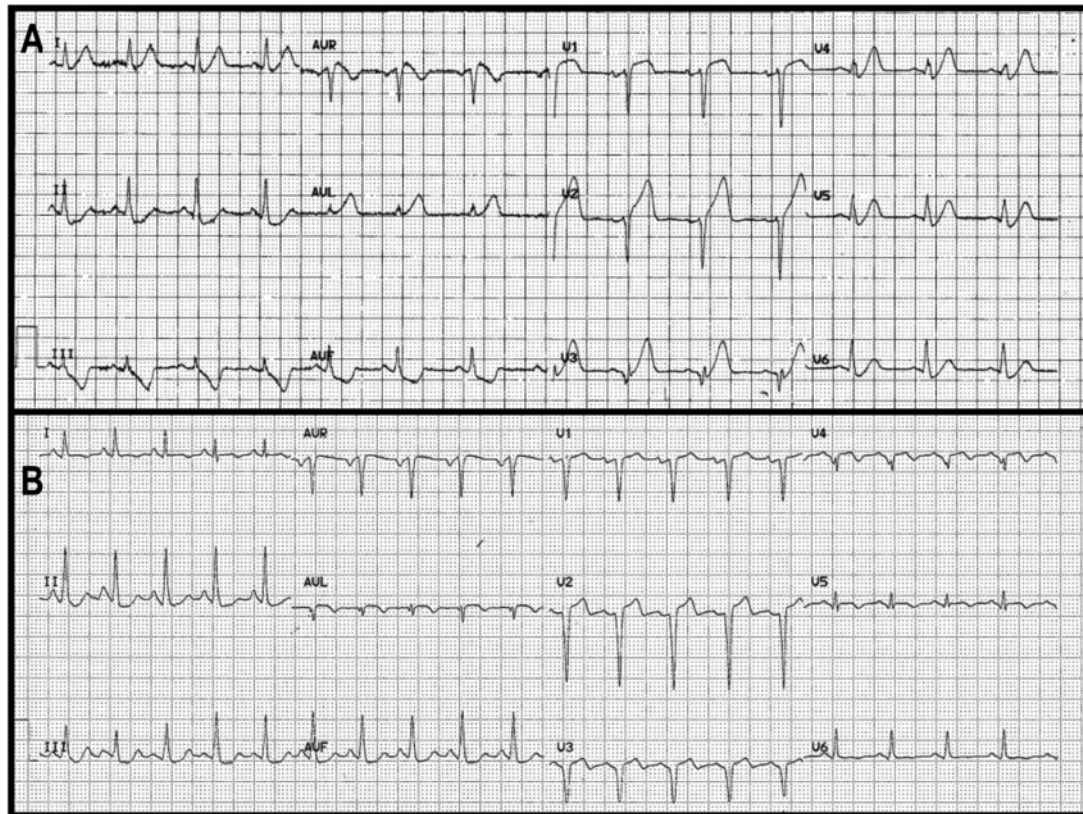


Figure 2 (A) Initial electrocardiogram demonstrating de Winter's pattern. (B) Resolution of the de Winter's pattern after thrombolytic therapy.

have been proposed and are discussed below. However, as with Case 1, the progression of the de Winter's pattern to overt STEMI in the absence of early reperfusion is well documented.⁷⁻⁹ It has been suggested that the static nature of the de Winter's pattern may be ascribed to its early identification and subsequent early invasive management.⁹ In the de Winter's series, the median time from first ECG to angiography was 60 min (range 42–101 min). This postulate, however, is debatable as ST-elevation typically develops rapidly in the setting of acute coronary artery occlusion. It has also been proposed that anatomical variants of Purkinje fibres with resultant endocardial conduction delay may result in the described changes. It has also been suggested that the lack of ST-elevation may be due to ischaemic adenosine triphosphate (ATP) depletion causing lack of activation of sarcolemmal ATP-sensitive potassium channels. This has been supported by similar lack of ST-elevation in knock-out mice lacking ATP-sensitive potassium channels.¹² Verouden et al.² also suggested that the area of transmural ischaemia was so large that the injury current was directed towards lead aVR and away from the precordial leads. The progression of this pattern to a STEMI in some patients, would however not support these pathophysiological mechanisms. It has also been postulated that collateral blood supply may preclude myocardial transmural ischaemia preventing ST-elevation. The loss of this collateral flow for various reasons may result in progression to STEMI.⁵

Our experience supports the fact that some patients exhibiting the de Winter's pattern who are not promptly reperfused, may go on to develop overt ST-elevation. Other patients continue to manifest this pattern until the time of reperfusion despite ongoing ischaemia for prolonged periods of time. This suggests that this pattern may lie somewhere along the continuum of ischaemic ECG changes between subendocardial ischaemia and the transmural injury pattern associated with ST-elevation. As to why some patients go on to develop ST-elevation and others maintain the de Winter's pattern until reperfusion occurs, remains poorly understood and should be the target for future research.

The optimal management of patients with the de Winter's variant ECG is not specifically addressed in current guidelines. The European Society of Cardiology (ESC) guideline on STEMI management however suggests a PCI strategy in patients with clinical concern of ongoing myocardial ischaemia with 'atypical ECG' patterns.¹¹ Based on angiographic evidence, this ECG pattern should most likely be treated as a STEMI equivalent. Case 2 highlights that there may be a role for thrombolytic therapy as a means of initial reperfusion in patients who exhibit a de Winter's pattern on their ECG where primary PCI cannot be offered timeously. Although others have reported poor response (50%) to thrombolytic therapy,⁷ the experience from our centre and MI referral network has been to the contrary with good reperfusion success rates. Initial thrombolytic

therapy should be coupled with early angiography (within 2–24 h) as part of a pharmacoinvasive approach to STEMI management.¹¹ Such an approach may be essential in a resource-limited setting where primary PCI cannot be offered in a timely manner.

Despite the deliberation regarding culprit vessel localization, the temporality thereof and the mechanisms behind the de Winter's pattern, it remains an important cardiological emergency where early recognition and swift action is paramount.

Lead author biography



Dr Thadathilankal-Jess John graduated from University of Cape Town in 2012 and completed his Physician Fellowship from the University of Stellenbosch in 2018. He is now working as a Cardiology Fellow at Tygerberg Hospital with an interest in Interventional Cardiology and Pericardial Disease.

Supplementary material

[Supplementary material](#) is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and

associated text has been obtained from the patients in line with COPE guidance.

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