

## The de Winter Pattern as Pre-Anterior ST-Elevation-Myocardial-Infarction : An Evolution Sequence

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### Abstract

**Background:** The de Winter pattern (dWP) was first described by de Winter and colleagues in 2008 as a static pattern associated with anterior myocardial infarction. A recent study showed the evolution sequence of this pattern into typical ST-elevation myocardial infarction (STEMI). This case discussed dWP who present as pre-anterior STEMI.

**Case Illustration:** A-56-year old Male arrived in the emergency room complained of chest pain for about 3 hours. The patient also complained of diaphoresis, nausea, and fatigue. The patient has a previous history of hypertension. The vital signs were stable with an unremarkable physical examination. The initial electrocardiogram (ECG) revealed sinus rhythm with j-point depression followed by prominent T wave in precordial leads, slight ST-segment elevation in aVR, and loss of precordial R-wave progression. The initial troponin T was 31 pg/mL. Follow-up 1-hour after initial ECG showed typical ST-segment-elevation in VI-V4. The patient undergoing thrombolytic, followed by angiography that showed subtotal occlusion in the proximal left anterior descending (LAD) artery, occlusion in the proximal circumflex artery, and stenosis in the proximal right coronary artery, echocardiography revealed regional wall motion abnormality in the septal and anterior segments and preserved ejection fraction 58%, the patient was discharged after 8-days treated in the intensive cardiac care unit.

**Conclusion:** dWP has been shown as a static and dynamic pattern in some conditions and is associated with acute LAD occlusion. In this case, we showed dWP as early anterior STEMI, recognition of this pattern leads to early reperfusion and better myocardial salvage as anterior STEMI has a poor outcome.

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## Background

**A**cute myocardial ischemia caused by coronary artery occlusion causes electrophysiological changes of the cardiac cell that lead to the appearance of a characteristic pattern in surface electrocardiogram (ECG). The de Winter pattern (dWP) was first described by de Winter and colleagues in 2008 as a static pattern of ECG that is associated with anterior myocardial infarction associated with left anterior descending (LAD) artery occlusion.<sup>1</sup> The ECG characteristics including upsloping ST-segment depression at the J-point in precordial leads followed by a prominent positive symmetrical T wave, other additional characteristics including slight ST-segment elevation in aVR and loss of precordial R wave progression.<sup>1</sup>

The prevalence of this pattern was 2% in the patient with anterior myocardial infarction with considerable loss of myocardium despite successful reperfusion procedure.<sup>1-4</sup> The mechanism of this ECG pattern was still unclear, and the early report state that the ECG pattern was static, but later, there are reports

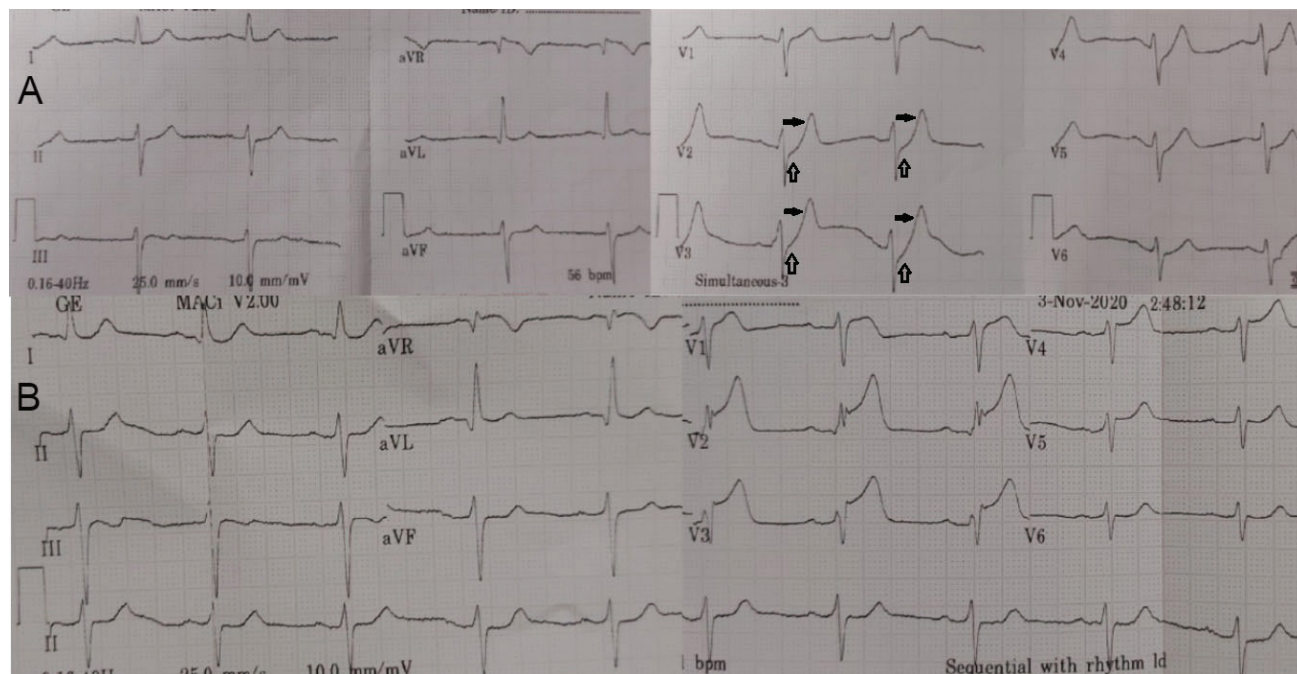
of the dynamic characteristics of this pattern into typical anterior ST-elevation myocardial infarction (STEMI).<sup>1,5-7</sup> Recognition of this ECG characteristic leads to early management and better prognosis.

In this paper, we discussed a case 56-year-old male with dWP ECG in first medical contact followed by a typical anterior STEMI pattern with a review of the mechanism.

## Case Illustrations

A 56-year old Male arrived in the emergency room complained of a heavy pressure sensation in the chest for about 3 hours. The patient also complained of diaphoresis, nausea, and fatigue. The patient has a previous history of hypertension about 3 years and was treated with amlodipine, and has no history of smoking and family history with premature coronary heart disease.

The vital signs revealed blood pressure 130/90 mmHg, heart rate 60x/m, respiration rate 18x/m, temperature 36.7°C, and SpO2 97% in room air with an unremarkable physical examination. The initial



**Figure 1.** Electrocardiogram (ECG) in Emergency Department. (A) Initial ECG showed sinus rhythm with j-point depression upsloping pattern (non-fill-arrow) followed by prominent T wave in precordial leads (black-fill-arrow), slight ST-segment elevation in aVR, and loss of precordial R-wave progression. (B) Follow-up ECG 1-hour after the initial ECG showed typical ST-segment elevation in V1-V4.

ECG that was recorded within 10 minutes of arrival showed sinus rhythm with j-point depression followed by prominent T wave in precordial leads, slight ST-segment elevation in aVR, and loss of precordial R-wave progression (Figure 1a). The initial laboratory showed troponin-T 31 pg/mL and elevated creatinine (Table 1). Chest X-ray showed cardiomegaly with clear lung. The point of care ultrasound showed regional wall motion abnormality in the septal and anterior segments. The patient was initially treated with oral aspirin 180 mg, clopidogrel 300 mg, and sublingual isosorbide dinitrate 5 mg.

Follow-up 1-hour after initial ECG, the serial ECG showed typical ST-segment-elevation in V1-V4 (Figure 1b). The patient received intravenous fondaparinux 2.5 mg followed with subcutaneous administration, nitroglycerin drip 20 mcg/minute, oral atorvastatin 40 mg, and although the patient arrives in percutaneous coronary intervention (PCI) center, because of the expected delay time to primary PCI, the patient undergoing streptokinase thrombolytic. The patient develops hypotension during thrombolytic therapy and is stabilized by dobutamine and norepinephrine drip. The chest pain was resolve after the thrombolytic therapy. The ECG 1-hour after thrombolytic therapy showed typical ST-segment elevation in the precordial area with marked loss of R wave progression (Figure 2).

The patient undergoing angiography that showed three-vessel disease including subtotal occlusion in the proximal left anterior descending artery, occlusion in the proximal left circumflex artery, and stenosis in the proximal right coronary artery (Figure 3). Follow-up 24 hours troponin-T showed marked elevation (10.000 pg/mL), echocardiography revealed regional wall motion abnormality in the septal and anterior segments and preserved ejection fraction 58%, the patient was stable during hospitalization and discharged after-8 day treated in the intensive cardiac care unit.

## Discussion

This case showed the evolution of dWP to a typical anterior STEMI pattern, leading this pattern may also have a dynamic sequence rather than only a static. From the early of the de Winter and colleagues introducing this pattern, some electrophysiological hypotheses were proposed, and the characteristic of this pattern now has

been studied.

A study from de Winter et. al. in 2008, recognize a static pattern of dWP in a minority (2%) of anterior myocardial infarction patients. The ECG was obtained with an average of 1.5 hours from the onset.<sup>1</sup> The ECG pattern was persisted from initial ECG to pre-procedural ECG. Although with a successful revascularization procedure, the peak of creatine kinase-myocardial band (CKMB) was still high.<sup>1,2,4</sup> In our case, we obtained the initial ECG about 3 hours from the onset, and the evolution was prominent 1 hour later. The follow-up of our cardiac marker (Troponin-T) was also high. A report from Barbati and colleagues also reporting the high peak of troponin-I despite adequate revascularization of proximal LAD occlusion, with the evolution of pathological Q waves and the appearance of cardiogenic shock during hospitalization.<sup>3</sup>

In contrast to the previous study, another study showed evolved cases into typical ST elevation patterns in the majority of patients with dWP.<sup>6-8</sup> In one study the prevalence of dWP was more common (3.4%) in anterior myocardial infarction, and 86.7% of the patient initially with dWP evolved into typical anterior STEMI pattern, with time from onset to ECG recording was 108 minutes, and time from dWP to ST-segment elevation evolution was 114 minutes, and mostly involved multi-vessel disease.<sup>6</sup> In a systematic review the dWP has a high positive predictive value (PPV) (95.2%, 100%, and 100% in the three respective studies) of acute LAD occlusion.<sup>9</sup> In our case the time from dWP to ST elevation was shorter and also involved LAD and multi-vessel disease.

Some possible mechanisms of this pattern were proposed. In 2008, de Winter and colleagues were proposed probable mechanisms that cause static patterns include myocardial conduction delay as a result of Purkinje fiber variance and lack of activation of sarcolemmal adenosine triphosphate (ATP) sensitive potassium channels that cause the absence of ST-segment elevation.<sup>1</sup> Later, a recent study showed the dynamic characteristic from dWP, that proposed some new probable mechanisms including subendocardial ischemia during the appearance of dWP that resulting from ischemic behavior of the subendocardial action potentials (AP) compared to the normal subepicardial AP, that progress to transmural ischemia that results in typical STEMI pattern in later.<sup>5,6</sup>

According to the theory of the transmembrane action potential (TAP) summation, the normal ECG can be explained by summing up the subendocardial plus the subepicardial TAP.<sup>10</sup> The QRS complex and T wave in the normal ECG is generated by voltage difference because the subendocardial AP starts earlier but ends later than the epicardial AP. The lack of voltage difference between subendocardial and subepicardial areas during the plateau phase of the AP resulting in a normal isoelectric ST segment. The mechanism of ST-segment depression in dWP would be related to the negative voltage difference between the ischemic subendocardial and the normal subepicardial action potentials during the plateau phase (phase 2 of AP) caused by subendocardial ischemia, this negative voltage difference is based on the theory of the TAP summation causing ST depression in the surface ECG. The peaked T waves in dWP were generated because of the shorter time duration between the ischemic subendocardial and normal subepicardial repolarization, causing both areas to repolarize within the almost same time, so generate more peaked T waves.<sup>5,10,11</sup> The change in the TAP shape would be resulting from repolarization delay in the subendocardial area because of hypoxia driven-alteration in ATP-dependent potassium channels.<sup>11</sup>

The repolarization abnormalities of the ischemic subendocardium area would be associated with interstitial accumulation of K<sup>+</sup> (ie. opening of ATP-sensitive K channels (iK-ATP)), catecholamines, and other metabolites caused by depletion of ATP and lactic acid accumulation. The Na<sup>+</sup>/H<sup>+</sup> exchanger was activated because of increased intracellular H<sup>+</sup>, resulting in H<sup>+</sup> extrusion in exchange for Na<sup>+</sup> entry, which in turn results in calcium overload because of activation of the Na<sup>+</sup>/Ca<sup>++</sup> exchanger. These processes causing the hallmark electrophysiological changes of ischemic myocardium include depolarization, inactivation of Na<sup>+</sup> channel (causing a reduction in peak inward Na<sup>+</sup>), and slow conduction.<sup>12</sup> If these ischemic cells are located in the subendocardium, based on the theory of the ischemic vector, this will produce the ST depression of the surface ECG because the ischemic vector toward the less electronegative area (ischemic area) or away from the surface lead during the systole (systolic current of injury), and elevation of TP segment causing ST depression because adjusted by AC amplifier because during diastole the vector toward

normal myocardium because the partially depolarized ischemic cell during diastole compared with normal cell (diastolic current of injury).<sup>10,13</sup> The shape of the ST depression may denote the difference of severity and extent of myocardial ischemia, anatomical different, and myocardial protection with the upsloping pattern was the most severe form than non-upsloping (horizontal and downsloping) for the dWP.<sup>14</sup>

Subendocardial ischemia in the early time results dWP to appear, when ischemia is progressed into transmural ischemia the STEMI pattern will appear with classic diastolic and systolic current of injury mechanism. It can be hypothesized that there was a time when the ischemia expanded from the subendocardium to the epicardium that showed the evolution of the ST-segment.<sup>6,13</sup>

## Conclusion

In this case, we showed the dWP in the presentation that evolves into typical anterior STEMI. The dWP has been shown as a static and dynamic pattern in some conditions and associated with acute LAD occlusion, and may involve multivessel lesions, some proposed mechanisms of this ECG pattern have been there to describe this phenomenon. The serial ECG is mandatory to show the evolution of dWP, and the most important, the recognition of this pattern in the “first look” ECG to avoid delay in the treatment and lead to early reperfusion and better myocardial salvage as anterior STEMI has a poor outcome in the short and longterm period.

## List of Abbreviations

ALT=Alanine transaminase  
 AP=action potential  
 AST=aspartate transaminase  
 ATP=adenosine triphosphate  
 CKMB=creatine kinase-myocardial band  
 dWP=de Winter pattern  
 ECG=electrocardiogram  
 LAD=left anterior descending  
 PCI=percutaneous coronary intervention  
 PPV= positive predictive value  
 STEMI=ST-elevation myocardial infarction  
 TAP=transmembrane action potential

## Conflict of Interest

None.

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