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Case Report

De Winter syndrome with occlusion of the proximal segment of the LAD coronary artery

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Abstract: We report a 57-year-old male that displayed a de Winter pattern on an electrocardiogram half an hour after chest pain. Emergency coronary angiography showed complete occlusion of the proximal segment of the left anterior descending branch (LAD), 50% stenosis in the left circumflex artery (LCA) and 95% stenosis in the middle of the right coronary artery (RCA). After emergency coronary intervention and drug treatment, the patient improved and was discharged.

Keywords: De Winter syndrome, acute myocardial infarction, primary PCI

Introduction

In 2008, de Winter et al. [1] described a novel electrocardiogram (ECG) pattern without classic ST-segment elevation myocardial infarction, which is now called de Winter syndrome. Specific criteria for de Winter ECG patterning include 1) ST depression 1-3 mm upsloping at the J-point in leads V1-V6 continuing into tall and positive symmetrical T-waves in the precordial leads with narrow or only slightly widened QRS complexes; 2) loss of precordial R-wave progression in some patients; and 3) slight ST-segment elevation in most patients with aVR, which has been described in association with ST depression in precordial leads [1]. de Winter syndrome is a special ECG pattern indicating acute occlusion of the proximal segment of LAD and is one of the most dangerous diseases in humans. A primary percutaneous coronary intervention (PCI) should be performed in these AMI patients as early as possible. We report the case of a patient who had a typical de Winter ECG pattern.

Case report

A 57-year-old male smoker without type 2 diabetes mellitus or previous cardiac history was admitted to the Emergency Department with complaints of severe precordial chest pain during rest. The chest pain started at 6 AM while he was still in bed. An electrocardiography (ECG) taken half an hour after the onset of pain showed sinus rhythm, J-point depression 1-3 mm, upsloping ST-segment depression, and tall and symmetrical T waves from leads V1 to V6 (**Figure 1A**). Laboratory examination results were as follows: cardiac troponin I (cTn I) 7.31 ng/ml; creatine kinase (CK) 1924 IU/L; creatine kinase-MB (CKMB) 222 IU/L; blood potassium (K^+) 3.95 mmol/L; and NT-proBNP 52 pg/mL. The patient had normal potassium levels, so high potassium was not responsible for the electrocardiographic changes. Blood pressure was 100/60 mmHg, pulse was 62 beats/min, respiration was 23 breaths/min, and axillary temperature was 36.3°C. Pulmonary examination revealed clear lungs without rales. Cardiac examination revealed a regular rate and rhythm with premature atrial contraction. There was no jugular vein engorgement and no pitting edema in lower extremities. One hour after the onset of pain, acetylsalicylic acid (300 mg) and ticagrelor (180 mg) were given to the patient. Interventional cardiology was immediately consulted, the catheterization laboratory was activated, and the patient was taken for emergent

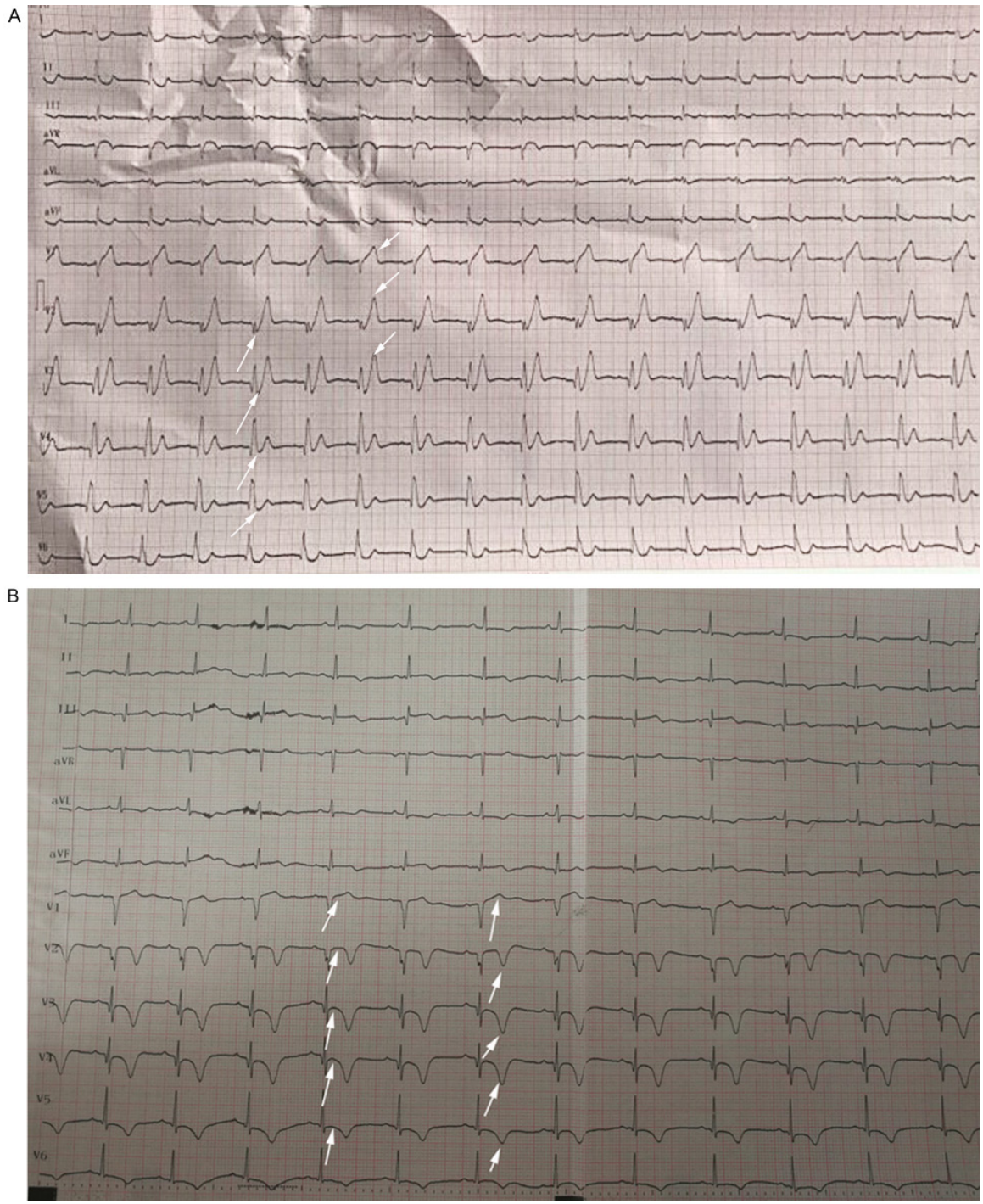

Figure 1. Comparison of ECGs between half an hour after the onset of pain and the next day after the onset of pain. A. The ECG showed sinus rhythm, the J-point depression 1-3 mm, upsloping ST segment depression and tall, symmetrical T waves from leads V1 to V6. B. The next day repeat ECG revealed sinus rhythm, the depressed ST segment returned to normal and the T wave reversed.

coronary angiography at 3 hours after the onset of chest pain. When he arrived in the catheterization laboratory, the patient's chest pain had already markedly relieved. The coronary angiography revealed complete occlusion of the proximal segment of LAD, 50% stenosis in LCA and 95% stenosis in the mid RCA. The patient was successfully treated with angioplasty and a drug-eluting stent in the proximal segment of the LAD (**Figure 2**). Coronary flow was com-

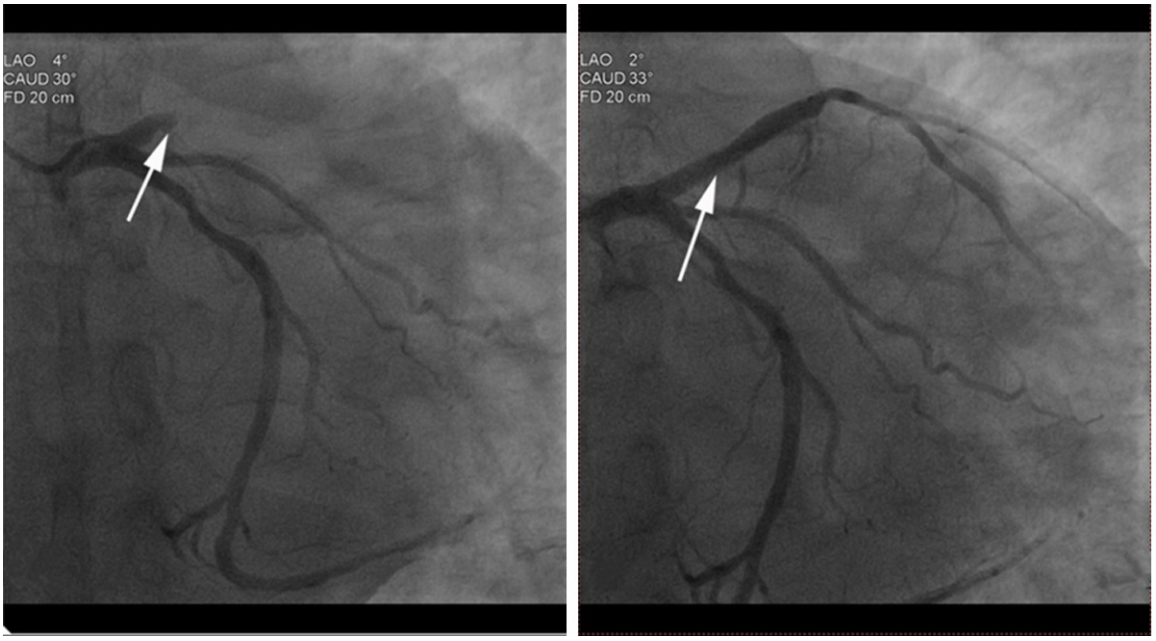

Figure 2. Comparison of results of coronary angiography between before PCI and after PCI. The left anterior descending artery (LAD) was completely occlusion (left). The blockage resolved after successful percutaneous coronary intervention (right).

pletely restored to Thrombolysis In Myocardial Infarction (TIMI) grade 3. The patient was treated with medicines, including aspirin, enoxaparin, clopidogrel, and statin, in the hospital. The next day, repeat ECG revealed sinus rhythm, the depressed ST-segment returned to normal, and the T wave reversed (**Figure 1B**). Repeat laboratory examinations were as follows: cTn I 8.52 ng/ml; CK 2761 IU/L; and CKMB 206 IU/L. The patient was discharged after improvement and persuaded to quit smoking and improve his lifestyle.

Discussion

The patient examined here displayed a typical de Winter ECG pattern. This ECG pattern may present in approximately 2% of patients with acute anterior myocardial infarction [2]. Patients with the de Winter ECG pattern are generally younger men with hypercholesterolemia [3]. This ECG pattern is always associated with a culprit vessel located in the proximal segment of the LAD artery. Therefore, these patients should be treated immediately with reperfusion therapy, preferably with primary PCI [4].

Although the exact electrophysiological mechanism underlying this ECG pattern remains elusive, it has been associated with ischemia and hypoxia of cardiomyocytes caused by a subtotal occlusion or a total occlusion of the LAD. de Winter syndrome may be related to a variation of sarcolemmal K_{ATP} channels, preventing the appearance of ST-segment elevation [5]. When cardiomyocytes are ischemic and anoxic, the permeability of the cell membrane will increase, the intracellular K^+ will flow out, and repolarization will be delayed. In this ECG, the tall and symmetrical T wave changes are observed in the ischemic region [6]. When the hypoxia and ischemia of cardiomyocytes are aggravated, necrosis will occur and cause potential differences between normal myocardium and damaged myocardium. In the ECG, the ST-segment vectors direct from ischemic myocardium to normal myocardium. The corresponding ST-segment leads will elevate in ischemia areas when subendocardial myocardial ischemia occurs. However, when endocardium myocardial ischemia occurs, the corresponding ST-segment leads will decrease in ischemia area. The potential difference between endocardium and epicardium can also lead to ST-segment depression. In addition, conduction delay in myocardium depolarization will occur through the damaged myocardium, and the ECG will show a wide QRS complex [7, 8].

Conclusion

The ST-segment depression and T wave change of de Winter syndrome are static phenomena. These symptoms tend to progress into transmural myocardial infarction. However, de Winter syndrome is under-recognized by clinicians, increasing patients' morbidity and mortality risk. This case highlights that the timely identification of this ECG pattern is critical, and the appropriate primary PCI is necessary.

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Disclosure of conflict of interest

None.

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