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A Case of Wellens Syndrome Combined with De Winter Syndrome

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Abstract: This paper reports a typical case of Wellens syndrome combined with De Winter syndrome. The male patient was admitted with "one day chest pain". He is 64 years old. He had chest pain without obvious inducement. Preliminary diagnosis: coronary heart disease unstable angina pectoris. The Electrocardiogram of this case is typical of Wellens syndrome. The patient's Electrocardiogram pseudonormalized during the onset of chest pain, and no significant ST-T changes were observed. After being admitted to hospital, chest pain was relieved. In the case of this case, when the chest pain occurred again, the electrocardiogram of the patient showed a 1-3mm depression of J point in lead V2-V6, followed by symmetrical high tip of T wave and normal ECG changes in QRS wave. Which was in line with the characteristics of electrocardiogram changes in De Winter syndrome. Coronary angiography results: right dominant type, no stenosis in LM, LCX and RCA, and 90%-95% stenosis in the middle part of LAD. The Electrocardiogram of the patient showed dynamic evolution of ST-T. Wellens syndrome and De Winter syndrome successively appeared. It suggests that severe proximal stenosis of the anterior descending artery is accompanied by coronary spasm and the possibility of spontaneous remission. Which will lead to changes of electrocardiograph in ST-T. For these patients, it is necessary to observe the changes of the condition closely and monitor the changes of electrocardiogram dynamically. It is likely to progress to acute extensive anterior wall myocardial infarction without further treatment. So it is necessary perform coronary intervention in time.

Keywords: Wellens Syndrome, De Winter Syndrome, Stenosis of Anterior Descending Artery

1. Introduction

In recent years the incidence of coronary heart disease is higher and higher. The mortality rate from acute coronary syndrome is also increasing year by year. Among acute coronary syndromes, Wellens syndrome and De Winter syndrome are relatively rare and often overlooked. Both of them had characteristic changes of electrocardiograph. Which were caused by severe proximal stenosis or occlusion of the anterior descending artery. Both are acute coronary syndromes that need urgent treatment. Early diagnosis is of great value for clinical treatment. The main difference between the two is the characteristic electrocardiograph changes. Wellens syndrome is characterized by deep symmetry inversion of T wave. Which no ST segment changes are involved in. The electrocardiogram of De Winter syndrome shows a symmetrical high tip of T wave with ST segment depression. This paper reports a typical case of Wellens syndrome combined with De Winter syndrome.

2. Case Report

The male patient was admitted with "one day chest pain". He is 64 years old. e had chest pain without obvious inducement, one day ago. Which was aggravated during

activity, and was relieved after taking Jiuxin pills. He had no discomforts such as shortness of breath, palpitation and syncope. He had a history of Grade three hypertension for 10 years and was treated with telmisartan. The result of physical examination: BP: 130/80mmHg, HR: 60 times/min, R: 20 times/min, T: 36.5°C. No wet and dry rales were heard in both lungs. No pathological murmurs were heard in the auscultation area of the heart valves. There was no edema in the lower limbs. Supplementary examination: ECG should be examined urgently when chest pain occurs, as shown in Figure 1 (Date: 16 March 2020 12:36): Sinus rhythm. T-wave upright in lead V2-6. No ST changes were observed in the limb lead and chest lead. The hypersensitive troponin was detected at 0.037ng/mL (Normal range < 0.05ng/mL). Preliminary diagnosis: coronary heart disease unstable angina pectoris. He was given ECG monitoring, oxygen inhalation, oral 300mg aspirin, 300mg Clopidogrel, heparin anticoagulant, and intravenous drip of isosorbide nitrate. Test results showed that blood routine: NT-proBNP: 865.8ng/L, myoglobin 15.96ug/L, troponin 0.05ug/L, D-dimer 0.157mg/L, low density lipoprotein cholesterol 1.25mmol/L. Blood routine, myocardial enzymes, blood glucose, thyroid function, kidney function and electrolytes were normal. Four hours later, myoglobin was 19.18ug/L and troponin was 0.04ug/L. Cardiac B ultrasound: EF: 58%, left atrial enlargement (36mm), left ventricular wall motion slightly weakened, left ventricular diastolic function decreased, LVDd45mm, LVDs31mm. Holter ECG: sinus rhythm, frequent ventricular premature beats 1788, ST-T ischemia changes. The chest pain was gradually relieved after symptomatic treatment. On the second day, the patient's chest pain was relieved and the ECG was reviewed. The ECG is shown in Figure 2 (Date: March 17, 2020 08:49): In sinus rhythm, the T waves were positive and negative in lead V2-3 and deep inverted in lead V4-5. The hypersensitive troponin was less than 0.02ng/mL. The patient was advised to complete coronary angiogram, but the patient did not agree. In the early morning of the third day, I had chest pain again. The ECG is shown in Figure 3 (Date: 02:35, March 18, 2020): Sinus rhythm, frequent ventricular premature beats, down J point in lead V2-6, low ST segment, and high peak T wave. The hypersensitive troponin was normal. The patient agreed to complete the coronary angiography. Coronary angiography results: right dominant type, no stenosis in LM, LCX and RCA, and 90%-95% stenosis in the middle part of LAD. LAD stenosis is shown by the arrow in Figure 4. Stent implantation was recommended, but the patient and his son refused. And the patient was treated conservatively with drugs. After the chest pain was relieved, the patient was discharged.

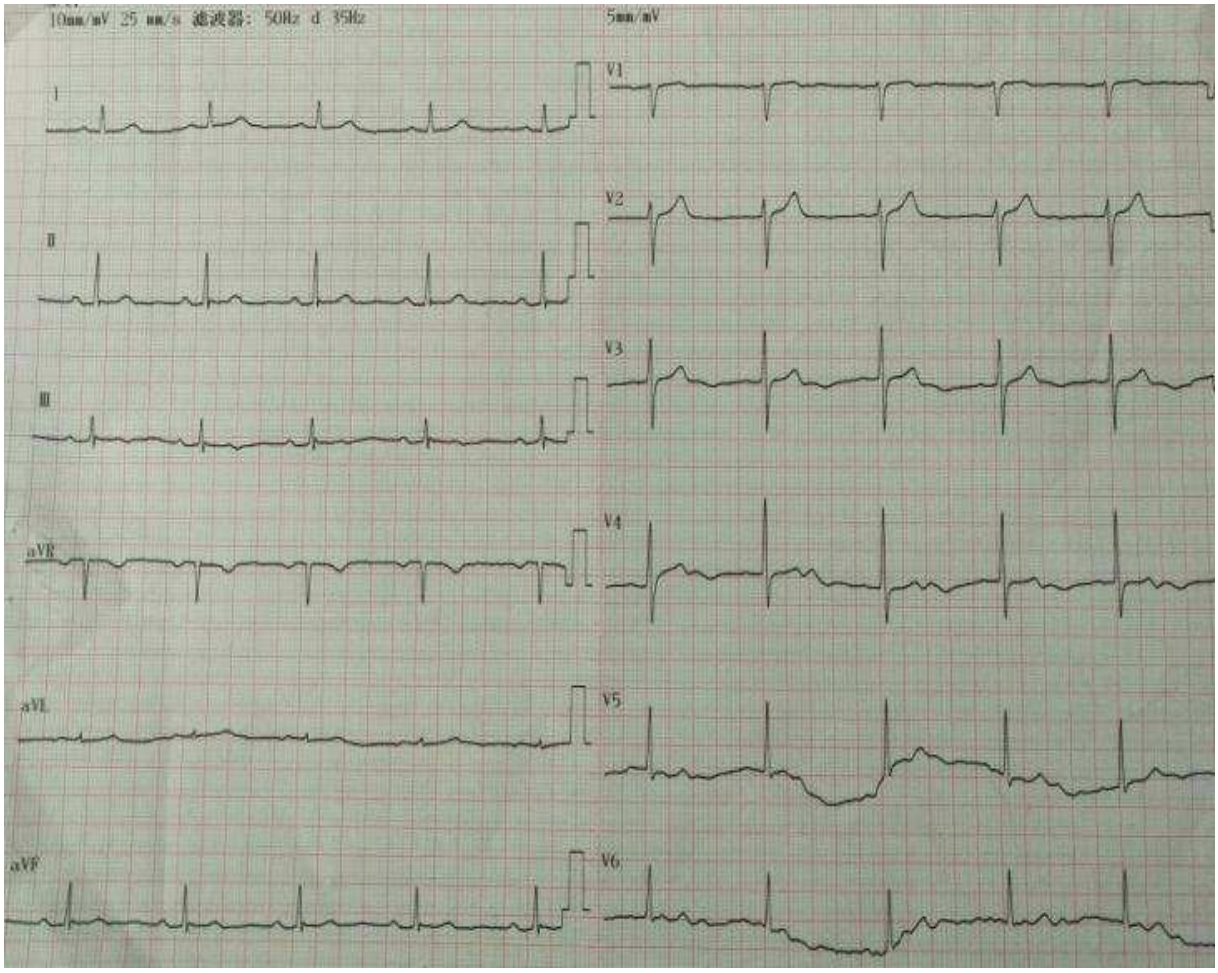

Figure 1. *Pseudonormalizing electrocardiogram at the onset of chest pain upon admission.*

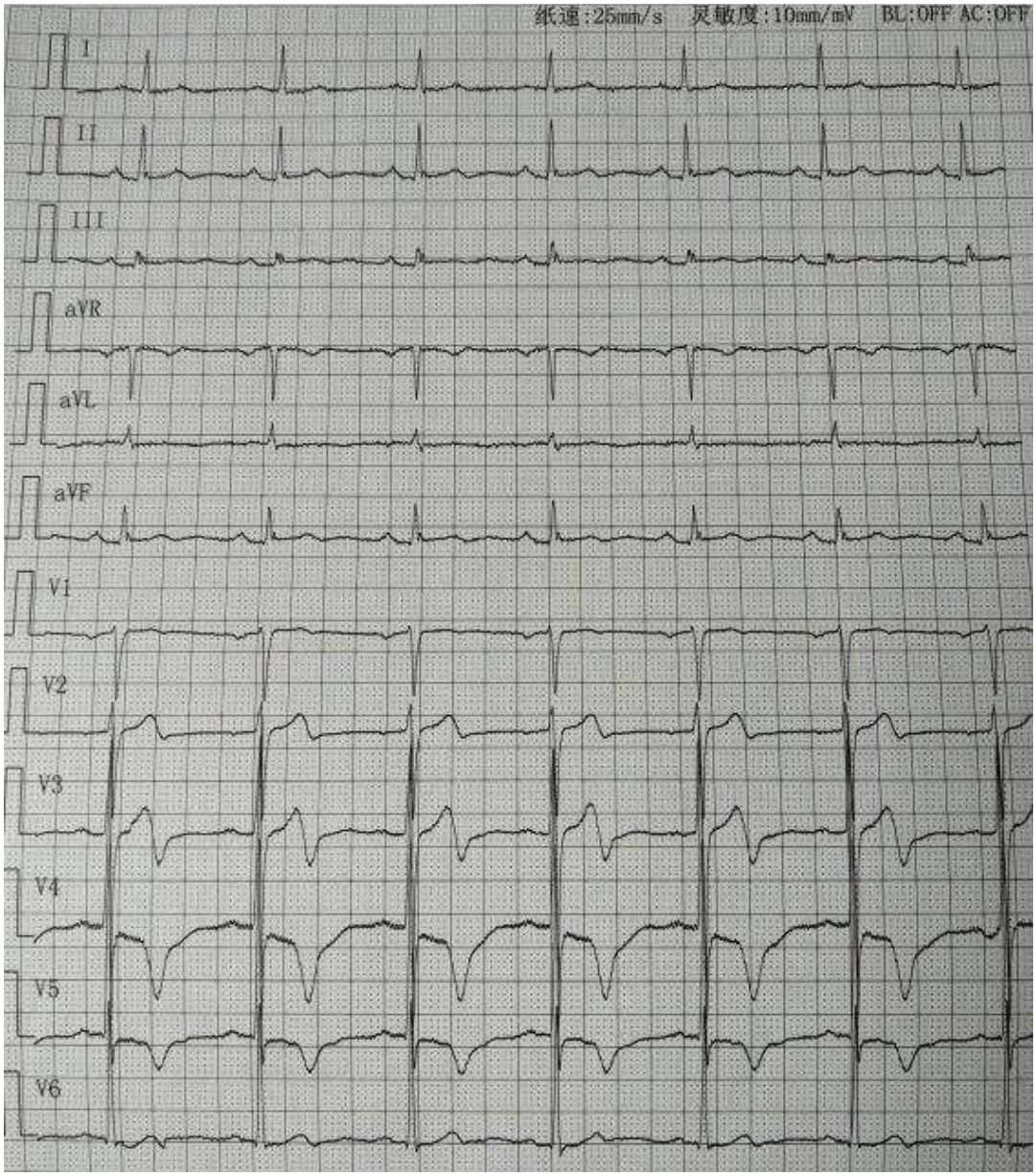

Figure 2. *The ECG changes of Wellens syndrome were found after the chest pain relief.*

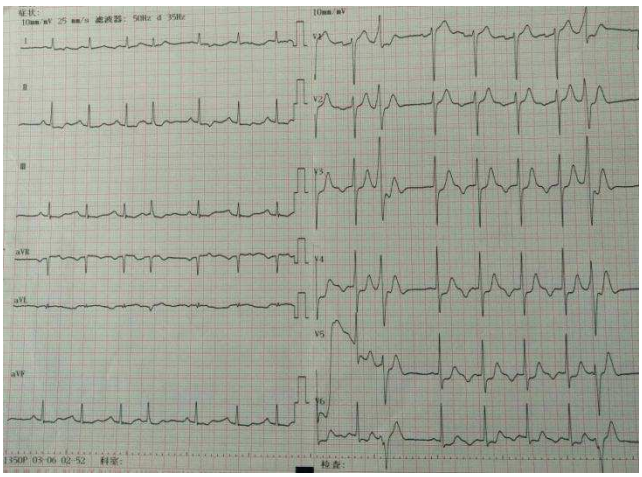

Figure 3. *The ECG changes of De Winter syndrome were found in the emergency ECG examination when chest pain reoccurred.*

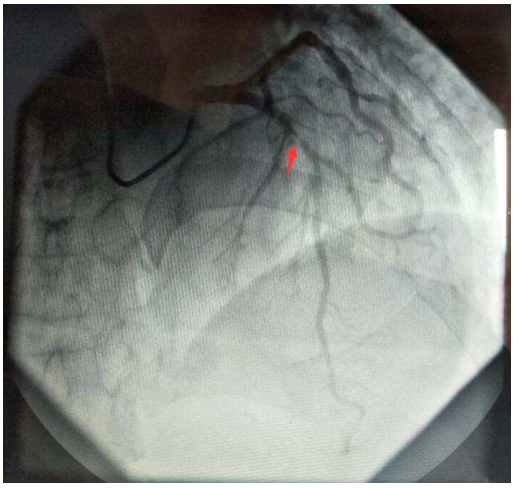

Figure 4. *Coronary angiography suggest severe anterior descending artery stenosis (AP+Cra35°).*

3. Discussion

In 1982, Wellens admitted that the Electrocardiogram was normal during angina attack and T wave changed during angina relief, firstly. The separation of ischemic symptoms and ECG changes was called Wellens syndrome [1]. which was also clinically known as left anterior descending T-wave syndrome (LAD). According to the characteristics of Electrocardiogram, it can be divided into two types. Type 1 shows a deep symmetry inversion of T wave, accounting for 75%. Type 2 is characterized by bidirectional T wave, which is positive first and then negative, accounting for 25%. Type 1 May convert to type 2 over time. In some patients, the T wave in lead V2-3 is inverted or upright during chest pain without ST segment changes. As the chest pain subsided, the T wave inverted further or became positive and negative, lasting for hours or weeks. The Electrocardiogram of this case is typical of Wellens syndrome type 2. The patient's Electrocardiogram pseudonormalized during the onset of chest pain, and no significant ST-T changes were observed. After being admitted to hospital, chest pain was relieved. Then positive and negative T waves appeared in leads V2 and V3, and T waves were inverted in leads V4 and V5. The myocardial enzymes and troponin were normal repeatedly. Unstable angina was considered. And coronary angiography suggested severe stenosis of the anterior descending artery. 90-95% stenosis in the middle part of the anterior descending artery. Sobnosky et al. proposed clinical and electrocardiographic diagnostic criteria for wellens syndrome: 1, history of chest pain; 2, V2 and V3 leads have bidirectional or deep inverted T waves. Sometimes V1-V4 and even V5 -V6 leads can also appear; 3, Myocardial enzymes were normal or slightly elevated; 4, ST segment of related lead was at equipotential line or slightly elevated (<0.1mV); 5, The amplitude of no R wave decreases or disappears; 6, No pathological Q wave. The specific mechanism of characteristic T wave changes in Wellens syndrome is not fully understood. The possible mechanism of Wellens syndrome is as follows: myocardial suppression and myocardial hibernation. The change of T wave reflects the improvement of myocardial ischemia. The inversion degree of T wave gradually becomes shallower, which may be due to the shallow degree of myocardial injury and necrosis. Meybe it is not enough to cause the changes of QRS and ST segment, but only the characteristic changes of T wave. T wave changes can be repeated during angina attacks. Normal coronary spasm is also seen in Wellens syndrome. In patients with angina pectoris, temporary complete or almost complete occlusion of the anterior descending artery leads to severe myocardial ischemia. Then the coronary arteries recanalize rapidly or get blood supply from collateral circulation. Then the myocardium is reperfusion and reperfusion injury occurs. This results in myocardial stagnation, which corresponds to significant repolarization abnormalities. In the ECG, it shows bidirectional or inverted T wave. The function of the suppressed myocardium can return to normal in a few days or weeks. And gradually return to normal in response to the morphological changes of T wave. Myocardial stagnation is a state of myocardium before it is on the verge of necrosis. It is easy to progress to myocardial necrosis due to the exacerbation of ischemia. In electrocardiogram, arched upward ST-segment elevation and pathological Q wave appear. Wellens syndrome accounts for 14-18% of patients with unstable angina, and can also be seen in patients with non-ST-segment elevation myocardial infarction or anterior descending coronary spasm [2]. Wellens syndrome is prone to progression to myocardial infarction. And conservative treatment with drugs is difficult to improve the prognosis of patients. So early coronary intervention is recommended. In ECG T wave changes usually occur within hours or days (mostly within 24 hours) after chest pain relief. The asymptomatic period following angina attack. Most of the patients had normal myocardial biochemical markers, while some had mildly elevated cTnT (I). So it will be diagnosed with acute non-ST-segment elevation myocardial infarction. Once diagnosed, exercise test and other cardiac stress test should be avoided. Becauce it will exacerbating the disease and causing acute myocardial infarction or even sudden death. The study of Wu Gaobo et al. [3] suggested that the stenosis degree of anterior descending artery in patients with type 2 Wellens syndrome was more serious than that in patients with Type 1 Wellens syndrome, and the risk level was higher. In this case, the positive and negative T waves in lead V2-3 belonged to Type 2 Wellens syndrome. The increase of BNP also suggested severe coronary stenosis. Coronary angiography after admission suggested severe anterior descending stenosis of 90-95%. The degree of T wave inversion in patients was correlated with myocardial reperfusion. The greater the degree of T wave inversion imply the greater the degree of reperfusion. Alsaab et al. [4] showed that the spontaneous recreness rate of the T-wave inversion group was significantly higher than that of the T-wave bidirectional group and the T-wave upright group before reperfusion treatment in anterior STEMI patients. Among which the spontaneous recreness rate of the T-wave upright group was the lowest. Studies have shown that T wave inversion usually occurs after patients' ischemia symptoms disappear [5].

In 2008, De Winter et al. [6] found that proximal occlusion of the left anterior descending artery (LAD) in the heart center by reviewing the electrocardiogram of 1532 patients with acute coronary syndrome. Thirty of these patients did not show the typical hyper acute ECG pattern of ST-segment elevation myocardial infarction (STEMI). In the form of Letter, they first reported the electrocardiogram findings of De Winter syndrome caused by proximal occlusion of the left anterior descending artery in the New England Journal of Medicine. Typical electrocardiogram characteristics of De Winter syndrome: 1, J point of prefrontal lead V1-6 was depressed by 1-3mm, ST segment was upward inclined downward, followed by a symmetrical high tip of T wave; 2, QRS wave was not generally widened; 3, Prefrontal lead R wave increased poorly in some patients; 4, ST segment of aVR was slightly elevated in most patients. In the case of this case, when the chest pain occurred again, the

electrocardiogram of the patient showed a 1-3mm depression of J point in lead V2-V6, followed by symmetrical high tip of T wave and normal ECG changes in QRS wave. Which was in line with the characteristics of electrocardiogram changes in De Winter syndrome. Subsequently, coronary angiography suggested 90-95% stenosis in the near middle part of the anterior descending artery. De Winter ST-T changes and Wellens syndrome may sometimes alternate [7]. Dynamic changes of electrocardiogram were observed in De Winter. This may be related to the degree of cardiovascular stenosis and collateral circulation. However, it is not absolutely suggestive of anterior descending occlusion and may be suggestive of other coronary artery stenosis. The exact mechanism of De Winter ST-T changes does not have a satisfactory explanation at present. Some scholars speculated that the changes of ST segment and T wave peak might be related to the changes of potassium ion levels in and out of cell membrane during myocardial injury and myocardial ischemia suppression. De Winter syndrome may be an early change in STEMI, or it may be an ECG manifestation of a particular type of ACS. The non-elevation of ST segment may be related to the failure to activate ATP-sensitive potassium channels on the cell membrane, and the mechanism may be the lack of ATP production caused by myocardial ischemia [8].

De Winter syndrome suggests subtotal occlusion but not complete occlusion of the anterior descending artery, severe endocardial ischemia and partial epicardial ischemia, resulting in T wave changes without ST segment elevation, which is consistent with the ECG characteristics of this case. De Winter syndrome can easily progress to ST-segment elevation myocardial infarction. Related studies have reported that De Winter syndrome can be divided into static type and ST-T dynamic evolution type. ST-elevation myocardial infarction and ST-T changes in De Winter syndrome are interconverted due to complete occlusion and spontaneous recitalization of coronary arteries [9]. Studies have shown that De Winter syndrome is not only the electrocardiogram of anterior descending artery occlusion, but also other main vascular lesions [10]. Xu Ning et al. [11] found that most of the patients with De Winter syndrome had lesions of the anterior descending artery. It could be lesions of the left main trunk or left circumflex coronary artery, and could also be coronary spasm. Patients with De Winter syndrome need timely reperfusion therapy to avoid more cardiomyocyte necrosis [12]. There is currently no indication for thrombolysis. It could be an early alteration of STEMI or a specific type of acute coronary syndrome. Initial studies have shown that ST-T does not change in De Winter syndrome. In this case, the electrocardiogram of the patient showed dynamic evolution of ST-T. Wellens syndrome and De Winter syndrome successively appeared. For these patients, it is necessary to observe the changes of the condition closely and monitor the changes of electrocardiogram dynamically. So it is necessary perform coronary intervention in time. De Winter et al. [13] even suggested that ST-T changes in patients with De Winter syndrome should be treated as ST-segment elevation myocardial infarction. The angina symptoms of Wellens syndrome and De Winter syndrome were not synchronized with the ECG changes.

4. Conclusion

To sum up, the above can improve the understanding of the electrocardiogram changes in Wellens and De Winter syndrome. And targeted treatment is very important. The presence of Wellens and De Winter syndrome suggests severe proximal stenosis of the left anterior descending coronary artery [13-14]. This unstable angina pectoris is a high-risk pectoris. It is likely to progress to acute extensive anterior wall myocardial infarction without further treatment [15]. Patients who underwent early coronary angioplasty or coronary artery bypass grafting may benefit. Because ECG is often poorly understood by clinicians, it is often ignored or misdiagnosed as reversible anterior wall ischemia, leading to prolonged reperfusion. Wellens and De Winter syndrome must be treated as STEMI [16]. Emergency interventional therapy is suitable for both of them. But thrombolytic therapy is not currently suitable.

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