Wellens’ syndrome: a life-saving diagnosis

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Abstract
Wellens’ syndrome is a relatively common clinical entity; however, it is often missed, especially in young patients. Without prompt diagnosis and aggressive intervention, patients with Wellens’ syndrome may rapidly go on to develop extensive anterior wall myocardial infarction and possibly sudden death. In this case report, we present a 33-year-old male patient with atypical chest pain, and discuss the significance of a prompt recognition of Wellens’ syndrome.

Keywords: Wellens’ syndrome, ECG, electrocardiogram, young patient, medical education

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Wellens’ syndrome is a pattern of electrocardiographic T-wave changes associated with severe stenosis of the left anterior descending artery (LAD). The risk factors for Wellens’ syndrome are the same as for acute coronary syndrome, such as diabetes mellitus, hypertension, hyperlipidaemia, advanced age, and family history of premature coronary heart disease. However, it is different from other acute coronary syndromes in that an electrocardiogram (ECG) obtained during episodes of pain demonstrates normalisation, and T-wave changes are found during pain-free periods. Therefore it is inclined to be missed in young patients without obvious cardiovascular risk factors.

Without prompt diagnosis and aggressive intervention, patients with Wellens’ syndrome may rapidly go on to develop extensive anterior wall myocardial infarction and possibly sudden death. Immediate repetitive ECG evaluation after the chest pain subsides and timely recognition of this diagnostic ECG pattern are crucial to decrease cardiovascular risk. Here we report on a fortunate young man with Wellens’ syndrome who was correctly diagnosed and treated.

Case report
A 33-year old man was admitted to the hospital because of intermittent chest pain for seven days. The chest pain was substernal and ‘prickling’. It occurred in the morning and at night, and lasted for 10 minutes to a few hours. He had no history of diabetes, hypertension, hyperlipidaemia, drug abuse or family history of premature coronary heart disease. He had a sedentary lifestyle.

The physical examination was unremarkable. The initial ECG obtained after admission was normal. At 07:40 the next morning, his pain recurred. An immediate ECG was obtained and there were no obvious T-wave changes (Fig. 1A). Twenty minutes later, the pain was relieved with 0.5 mg sublingual nitroglycerin, and then a pain-free ECG was performed (Fig. 1B), which showed biphasic T waves in leads V2–V4.

The dynamic T-wave changes raised concerns about Wellens’ syndrome, which is associated with severe stenosis of the LAD. The patient underwent immediate coronary angiography, and the procedure showed 95% stenosis of the proximal LAD (Fig. 2A); the stenosis was treated with a drug-eluting stent (Fig. 2B). The troponin T level rose to a peak of 0.195 ng/ml (normal value < 0.1 ng/ml).

The patient was discharged home symptom free and referred to a cardiac rehabilitation programme. He has been in constant follow up and has not experienced angina again.

Discussion
Wellens’ syndrome is a pre-infarction stage of coronary artery disease. It comprises 10 to 15% of all acute coronary syndromes in the USA. However, it is often missed, especially in young patients.1–3

Khan reported Wellens’ syndrome in a 24-year-old woman with atypical chest pain and characteristic ECG changes. This was initially unrecognised and the young patient subsequently progressed to an anterior non-ST elevation myocardial infarction.4 Wang reported another Wellens’ syndrome in a 22-year-old man.5 Both young patients in these two cases had cardiovascular risk factors, namely diabetes and familial hypercholesterolaemia, respectively.5–6 Our case was different, as the young patient has no obvious cardiovascular risk factors or family history of premature coronary heart disease.

Wellens’ syndrome is prone to misdiagnosis. However the characteristic ECG pattern is specific for a differential diagnosis.

Wellens’ syndrome, first reported by de Zwaan in 1982, is a pattern of electrocardiographic T-wave changes associated with severe stenosis of the LAD.4 More specifically, Wellens’ syndrome can be classified into two types. Type 1 Wellens constitutes 24% of cases, is less common, poorly recognised, and
described as biphasic T waves in V2–V3, which was the finding in our patient (Fig. 1B). The more common type 2 Wellens’ accounts for the remaining 76% of cases and is identified by deep, symmetrically inverted T waves in V1–V4. This T-wave pattern is well recognised by junior doctors. It is important to emphasise that the T-wave changes of Wellens’ syndrome occur during pain-free periods, while during an episode of chest pain, the T waves normalise.

The criteria for Wellens’ syndrome are as follows: previous history of chest pain, no Q waves or loss of R waves, no significant ST-segment elevation, normal or minimally elevated cardiac markers, and biphasic/inverted T-wave changes in the precordial leads. Without prompt diagnosis and aggressive intervention, patients with Wellens’ syndrome may rapidly go on to develop extensive anterior wall myocardial infarction, with a mean time of 8.5 days. As a result, patients with Wellens’ syndrome should undergo immediate or rapid invasive coronary strategy.

Conclusion
We highlight three learning points about this case: (1) immediate repetitive ECG evaluation after the chest pain subsides, even in young patients without significant risk factors; (2) timely recognition of the diagnostic ECG pattern of Wellens’ syndrome; (3) emergency coronary angiography should be conducted if diagnosed.

References


