CASE REPORT

Wellens' syndrome in a 24-year-old woman

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SUMMARY

Wellens' syndrome refers to specific ECG abnormalities in the precordial T-wave segment, which are associated with critical stenosis of the proximal left anterior descending (LAD) coronary artery culminating in an acute anterior wall myocardial infarction (MI) if the patient is not urgently revascularised. We describe the youngest reported presentation of Wellens' syndrome in a 24-year-old woman with unstable chest pain, characteristic ECG changes and slight troponin biomarker elevation. This was initially unrecognised by the emergency department as unstable coronary syndrome and she subsequently progressed to an anterior non-ST elevation MI (NSTEMI). Her coronary angiogram showed critical narrowing of the proximal LAD which was successfully treated with a drug-eluting stent.

Wellens' syndrome is also referred to as LAD coronary T-wave syndrome. Syndrome criteria include the following:

- ▶ Biphasic/inverted T-wave changes in precordial leads;
- ▶ History of anginal chest pain;
- ▶ Normal or minimally elevated cardiac enzyme levels;
- ▶ ECG without Q-waves, without significant ST-segment elevation, and with normal precordial R-wave progression.

The risk factors for the development of this condition are the same as for acute MI. They include: smoking, diabetes mellitus, hypertension, advanced age, hypercholesterolaemia, hyperlipidaemia, metabolic syndrome and family history of premature heart disease.

Although Wellens' syndrome is not infrequent (reported incidence of 10–15% of all acute coronary syndromes in the USA³), this case is unusual in describing the youngest reported woman to present with Wellens' syndrome.

BACKGROUND

Wellens' syndrome is a preinfarction stage of coronary artery disease. In 1982 Wellens *et al*¹ first published the clinical and ECG criteria of a subgroup of patients with myocardial ischaemia that later came to be known as Wellens' syndrome. Recognition of this ECG pattern allowed the identification of patients who had a critical stenosis of the proximal left anterior descending (LAD) coronary artery and hence were at risk of extensive anterior wall myocardial infarction (MI). T-wave changes in the syndrome usually occur during a pain-free interval. Although these patients may initially respond well to medical management, they ultimately fare poorly with a conservative therapy and require revascularisation strategies.

CASE PRESENTATION

A 24-year-old Asian woman presented to the emergency department of our District General Hospital with a recent onset of chest pain. She had initially presented to the emergency department 1 week earlier but was discharged home following a presumptive diagnosis of atypical chest pain with normal ECG (figure 1) and negative 6 h high-sensitivity cardiac troponin test. During the subsequent week, she reattended the same emergency department with crescendo deterioration in symptoms. Her pain was described as central, crushing and radiating to the left arm at rest.

Figures 1 Normal ECG on initial presentation with chest pain.

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Figures 2 ECG while pain free, showing newly biphasic T-wave inversion in septal leads V1–V2 with no R-wave loss or Q-waves.

She was a smoker, obese and had established diagnoses of type 2 diabetes mellitus and hypertension. She also had a family history of premature ischaemic heart disease on her paternal side. There was no prior history of illicit drug use.

The physical examination on admission was unremarkable except for a high body mass index. She was normotensive and oxygen saturations on air were normal.

INVESTIGATIONS

Her ECG on reattendance (carried out while pain free) demonstrated newly biphasic T-wave inversion in the septal leads, V1–V2 with no R-wave loss or Q-waves (figure 2). The cardiac biomarkers showed an initial high-sensitive cardiac troponin level of 0.62 ng/ml rising to 0.92 ng/ml after 12 h (normal level <0.006 ng/ml). She received the usual protocol-driven treatment for NSTEMI including loading doses of aspirin and clopidogrel followed by a subcutaneous injection of therapeutic fondaparinux anticoagulant. She was transferred to the coronary care unit for ongoing care and monitoring. Serial subsequent ECGs showed late evolution to T-wave inversion consistent with recent anterior NSTEMI, despite remaining pain free (figure 3). Her

random serum total cholesterol level on admission was elevated at 6.0 mmol/l. Her vasculitis and thrombophilia screens were negative.

Following a review by the cardiology team, she was further evaluated by echocardiography followed by diagnostic coronary angiography. The transthoracic echocardiogram demonstrated a structurally normal heart with no regional wall motion abnormality. The coronary angiogram revealed isolated critical stenosis of the proximal LAD (figure 4). She was transferred urgently to the regional tertiary cardiac centre for percutaneous coronary intervention.

DIFFERENTIAL DIAGNOSIS

- Myopericarditis
- Pulmonary embolism

TREATMENT

The LAD stenosis was successfully treated with a drug-eluting stent (DES). She was advised to continue dual antiplatelet therapy for 1 year and secondary prevention medications life-long. Smoking cessation advice and support was provided.

Figures 3 ECG showing later evolution to T-wave inversion, consistent with recent anterior non-ST elevation myocardial infarction.

Figures 4 Coronary angiogram demonstrating critical proximal left anterior descending stenosis.

OUTCOME AND FOLLOW-UP

She was discharged home symptom free and routine clinic follow-up arranged 6 weeks postmyocardial infarct.

DISCUSSION

The NSTEMI in this patient is likely to have been due to the rupture of a vulnerable atherosclerotic plaque in the proximal LAD coronary artery. The angiographic pattern of the disease was typical of the underlying atherosclerosis. She had multiple conventional cardiovascular risk factors (namely smoking, diabetes mellitus, hyperlipidaemia, obesity and family history of premature coronary disease) which we believe synergistically accelerated atherosclerosis. She is one of the youngest patients presenting with unstable coronary syndrome successfully treated by percutaneous coronary intervention. Premenopausal women have a much lower incidence of cardiovascular events than men—a benefit long attributed to natural oestrogen. Despite this, heart disease is the leading cause of death in women.

Wellens' syndrome presents with characteristic ECG findings that all physicians need to recognise due to the high risk of developing anterior wall MI if aggressive intervention is not

undertaken. Patients in whom Wellens' syndrome is suspected should undergo urgent cardiac catheterisation as medical management could lead to left ventricular dysfunction and death.³ Stress testing is contraindicated as increasing cardiac demand with a highly stenosed LAD may lead to myocardial infarction.

Wellens' syndrome is well described and frequently seen in cardiology centres worldwide; however, the case we describe is uniquely young and unusually a female.

Acute coronary syndrome (ACS) in young adults is more commonly attributable to familial hyperlipidaemia, spontaneous coronary artery dissection or cocaine-induced vaso-spastic ischaemia. Other unusual causes of ACS include Kawasaki's disease, embolism, vasculitis or hypercoagulability state (eg, nephrotic syndrome).

Learning points

- ▶ Early identification of characteristic ECG changes is crucial for the diagnosis of Wellens' syndrome.
- ▶ Prompt invasive treatment is essential to avoid evolution to acute anterior myocardial infarction, left ventricular dysfunction and possible sudden death.
- ▶ Acute coronary syndrome is becoming increasingly common in younger age groups with significant risk factors.
- ▶ The differential diagnosis for acute chest pain with similar ECG changes includes myopericarditis and pulmonary embolic disease.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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