Wellens Syndrome

Marcelo Campos Appel-da-Silva[1], Gabriel Zago[1], Aníbal Pereira Abelin[2], Walter Otávio Pin[2], Oscar Pereira Dutra[2], Renato Vaz[2]

Hospital Nossa Senhora da Conceição (HNSC), Porto Alegre, RS[1]; Instituto de Cardiologia - Fundação Universitária de Cardiologia (IC-FUC)[2], Porto Alegre, RS - Brazil

The assessment of chest pain is a routine in emergency health services. Although this is a common complaint with a broad differential diagnosis, the major concern is the possibility of acute coronary syndrome (ACS). Electrocardiography and cardiac enzyme levels are important tools in the investigation of these patients; however, negative results may actually be overlooking the identification of some conditions. We report the case of a female patient with angina, whose baseline tests in the emergency room were negative for changes suggestive of ischemic heart disease. The Wellens syndrome, a recently described disease, is a variant of unstable angina which, if not identified, may result in significant morbidity and mortality.

Introduction

Chest pain - a frequent reason for visits to emergency health services, has a broad differential diagnosis in a setting where prompt actions are prioritized, and where resources are not always available to help elucidate the hypotheses[1]. The causes of acute non-traumatic chest pain, especially the acute coronary syndromes, play an important role in the investigation, since they represent diseases with high morbidity and mortality, and have a great potential for reversibility[2].

The ability to interpret electrocardiographic tracings and thus identify changes suggestive of myocardial ischemia is required from physicians in emergency health services. Nevertheless, a recent study showed that of the patients seen with a chief complaint of chest pain in emergency health services and discharged, the diagnosis of myocardial infarction was missed in 2% to 13%[3].

First described in 1982, Wellens syndrome is uncommon in the medical practice; it is a high-risk form of acute coronary syndrome (ACS) and is associated with critical stenosis of the proximal portion of the anterior descending coronary artery.

Key words

Myocardial ischemia; acute coronary syndrome; angina, unstable; electrocardiography.

Mailing address: Marcelo Campos Appel da Silva •
Rua Com. Caminha, 250/902 - M Vento - 90430-030 - Porto Alegre, RS - Brazil
E-mail: marceloappel@yahoo.com.br
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If this clinical condition is not identified and treated early, patients may develop anterior wall myocardial infarction, leading to death[4].

We report the case of a female patient seen in a cardiology emergency health service who presented with a chief complaint of acute chest pain and whose laboratory tests led to the diagnosis of Wellens syndrome.

Case report

A 70-year-old Caucasian female patient was seen in a cardiology emergency service with a chief complaint of crushing substernal chest pain; the pain was strong and irradiated to the neck and jaw, and had started at rest. Her past medical history revealed a diagnosis of hypertension for five years, for which she received captopril 25 mg tid; dyslipidemia; diabetes mellitus, controlled with diet; and family history positive for coronary artery disease. The patient reported to be a second-hand smoker and denied using or having used alcohol.

On the baseline physical examination, the patient showed a fair general state of health; she appeared in pain, was alert, oriented and coherent; her mucous membranes were moist and pink; she had no respiratory distress or fever. Cardiac and pulmonary auscultation were normal; BP was 100/60 mmHg and HR = 80 bpm. In face of the presumptive diagnosis of unstable angina, cardiac enzymes were determined, and an electrocardiogram (ECG) was performed during pain, but did not show unquestionable signs of acute ischemic lesion. The baseline laboratory test results revealed total CK = 47 mg/dl, CK-MB = 5 mg/dl and negative troponin T. The patient was given sublingual isosorbide dinitrate 5 mg, with a good analgesic response. ECG was then repeated (Figure 1) and showed biphasic T waves in leads V1 to V4. In face of the clinical and laboratory manifestations and typical electrocardiographic changes, the patient was diagnosed with Wellens syndrome.

The patient underwent coronary angiography which showed a severe lesion in the proximal third of the left anterior descending coronary artery (LADA), and a severe lesion in the right coronary artery; ventriculography showed anterior wall hypokinesia (Figure 2). Coronary angioplasty was then performed with stent implantation in LADA. The patient was transferred to the intensive care unit (ICU) of *Instituto de Cardiologia - Fundação Universitária de Cardiologia* (IC-FUC) for observation and reassessment.

The second series of cardiac enzymes repeated the baseline

pattern. During the brief ICU stay, there was no recurrence of chest pain or hemodynamic changes, so the patient was transferred to the ward in good general state of health; outpatient follow-up was planned.

Discussion

Ischemic heart disease (IHD) is the main cause of death in the United States, exceeding the cases of AIDS, cancer, and infectious diseases. Every year, 1.1 million Americans are estimated to develop a new or recurrent coronary episode, of which 45% have a fatal outcome. With the advances in medicine and the development of new medications for the treatment and control of ACS, the prevalence of individuals living with IHD is increasingly greater, as are the costs with hospitalization, tests, health professionals and rehabilitation, with a great impact on health systems[5,6].

ACS may be classified as: (a) unstable angina and non-ST segment elevation myocardial infarction (NSTEACS); and (b) ST-segment elevation myocardial infarction (STEACS); they are usually caused by thrombosis and/or vasospasm of a coronary artery on an atherosclerotic plaque or plaque rupture[7].

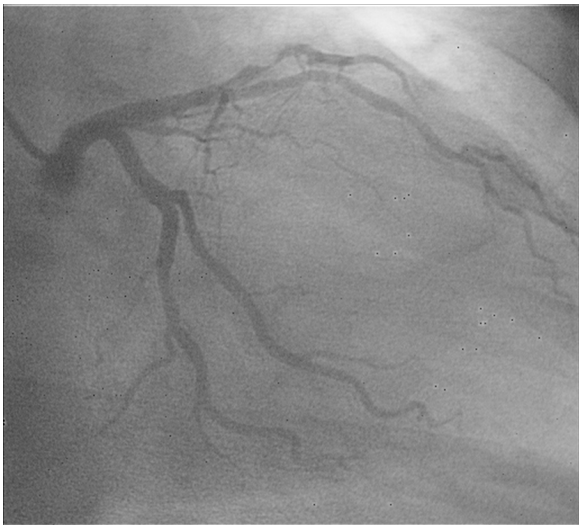

Figure 2 - *Coronary angiography image showing severe stenosis in the proximal segment of the left anterior descending coronary artery.*

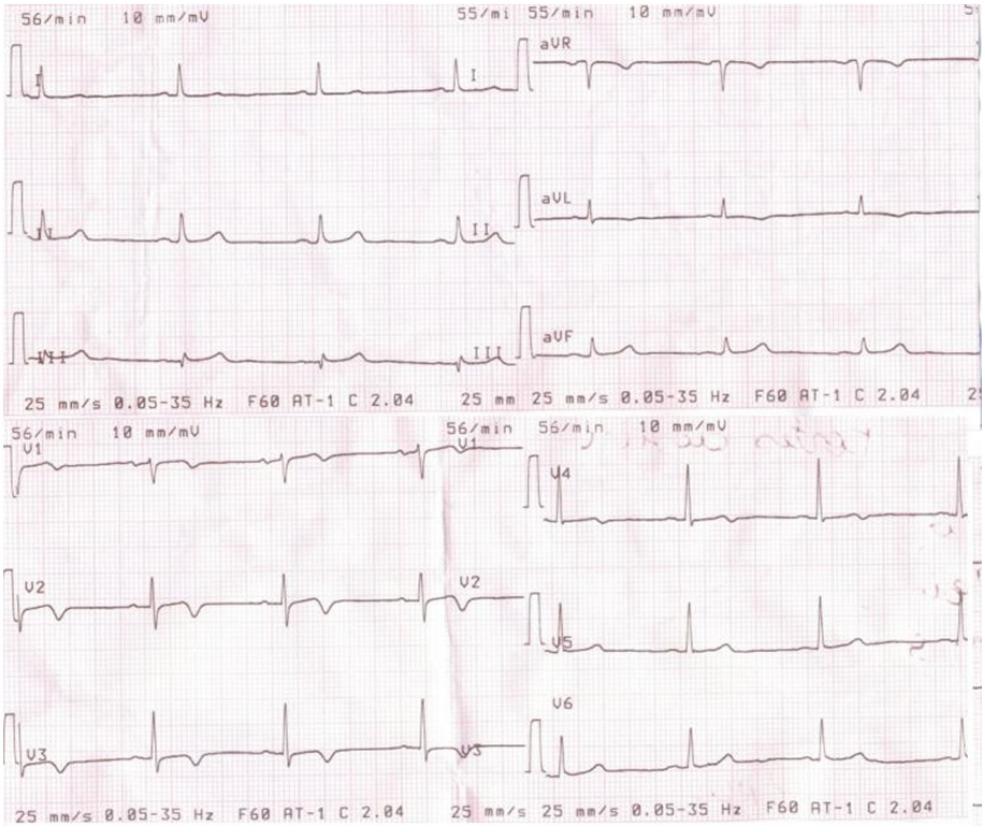

Figure 1 - *ECG performed in the absence of pain. Tracing shows plus/minus biphasic changes in V1-V4.*

The clinical presentation of ACS is not always typical, nor are the laboratory tests and so patients with ischemic heart disease may be underdiagnosed and not receive adequate management. In the present case, although the patient presented with a chest pain typical of angina, the initial investigation with ECG and cardiac enzymes was negative for ischemic heart disease. Among less experienced physicians, this could have resulted in discharge from the emergency unit, and the diagnosis of ACS would have been overlooked. Also, considering the TIMI risk score for NSTEACS, this patient's score would be three points (history of hypertension, dyslipidemia, diabetes mellitus, family history of coronary artery disease, and ST-segment elevation greater than 0.5 mm), which could result in moderate risk for MI or death[8].

Chest pain assessment protocols underline the importance of the early performance of ECG, within up to 10 minutes of arrival in the emergency unit, because this is a high-sensitivity method in the screening of ischemic heart disease. When changes suggestive of ischemia are not detected during pain, the risk of myocardial infarction (MI) is of approximately 4% and 2% in patients with or without previous history of coronary artery disease, respectively[8].

In order to identify patients with high-risk unstable angina, some decades ago some authors identified ECG changes that enabled a better management of these cases.

In 1982, De Zwaan and Wellens[4] described a subgroup of patients who had been admitted for unstable angina, because they had a high risk for the development of anterior MI. These patients had electrocardiographic changes similar to those described by Gerson et al[9]. These findings became known as Wellens syndrome (WS).

In two studies with hospitalized patients diagnosed with unstable angina, a 14%-18% prevalence of electrocardiographic changes consistent with WS was observed[4,10]. In one of these studies, 12 (75%) of the 16 patients with criteria for WS not undergoing myocardial revascularization developed extensive anterior MI within the first few weeks of hospital admission, thus showing the importance of early diagnosis of this syndrome.

Clinical and electrocardiographic criteria for the diagnosis of WS are the following: 1) biphasic or deeply inverted T waves in V2 and V3, or, occasionally, V1, V4, V5 and V6; 2) normal or minimally elevated cardiac enzymes; 3) normal or slightly elevated (< 1 mm) ST segment; 4) no loss of R wave progression in precordial leads; 5) absence of abnormal Q waves; and 6) anginal chest pain[4,10].

Two variants of WS are recognized:

• Type-1 WS, which corresponds to the minority of the cases (24%), and is characterized by the finding of biphasic T wave (plus/minus) in leads V2 and V3.

• Type-2 WS, which corresponds to the majority of the cases (76%), and is accompanied by deep and symmetrical T-wave inversion typically in leads V2 and V3, and, occasionally in V1-V4, V5 and V6[11,12].

Conclusion

Electrocardiographic changes of type-1 Wellens syndrome are highly specific for the diagnosis of severe atherosclerotic disease in proximal LADA, with a high risk for the development of anterior MI. Patients with clinical manifestations and tests suggestive of the syndrome should be promptly referred for angiographic study for diagnostic confirmation and intervention.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any post-graduation program.

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