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Case Report

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Presentation of a case with Wellens syndrome

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Acronyms
AMI: acute myocardial infarction

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ABSTRACT
This case report is about a 56-year-old male, farm worker with a history of being a smoker and suffering from high blood pressure, who was admitted at the Cardiology Care Department with the diagnosis of coronary artery disease –unstable angina–, because of chest pain related to physical effort and changes in the appearance threshold. Rest-electrocardiogram, painless, shows deep, symmetric negative T waves in anterior wall, without enzyme elevation; but during admission the patient evolves quickly, clinically and electrically, to an extensive anterior wall acute myocardial infarction, without responding to the fibrinolytic reperfusion therapy, and showing a ventricular tachycardia degenerating into ventricular fibrillation. There was no response to the maneuvers of cardiovascular resuscitation, thus, he dies. It is diagnosed postmortem as a Wellens syndrome, because necropsy showed severe atherosclerotic disease of the proximal segment of the left anterior descending coronary artery with extensive anterior transmural infarction.
Key words: Wellens syndrome, Left anterior descending artery, Acute myocardial infarction

Presentación de un caso con síndrome de Wellens

RESUMEN
Se presenta el caso de un varón de 56 años de edad, obrero agrícola, con antecedentes de ser fumador y padecer de hipertensión arterial; que ingresa en el Servicio de Cardiología con el diagnóstico de cardiopatía isquémica –angina inestable–, por presentar dolor precordial relacionado con el esfuerzo físico y cambios en su umbral de aparición. El electrocardiograma en reposo, sin dolor, muestra ondas T negativas profundas, simétricas, en cara anterior, sin elevación enzimática; y durante el ingreso evoluciona de forma rápida, clínica y eléctricamente, a un infarto agudo de miocardio anterior extenso, sin respuesta a la terapia de reperfusión con fibrinolíticos, y aparición de una taquicardia ventricular que degenera en fibrilación ventricular, sin respuesta a las maniobras de reanimación cardiovascular, por lo que fallece. Se diagnostica post mortem como un síndrome de Wellens, pues la necropsia demostró enfermedad aterosclerótica grave del segmento proximal de la arteria coronaria descendente anterior con infarto transmural anterior extenso.

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Palabras clave: Síndrome de Wellens, Arteria descendente anterior, Infarto agudo de miocardio

INTRODUCTION

Cardiovascular diseases are the leading cause of death in the developed and developing world; among them, the coronary artery disease is one of the main causes of death and disability[1], with epidemiological predictions which suggest that the morbidity and mortality will surpass cancer and infectious diseases in all countries of the world. The consequences of this disease on the global health originate a serious health problem[2]. Percutaneous coronary intervention is a valuable tool for revascularization of patients with ischemic heart disease[3].

The identification of certain typical electrocardiographic patterns allows selecting patients at high risk of evolving to an acute myocardial infarction (AMI) and death[4,5]. The interpretation of these drawnings, with suggestive changes of myocardial ischemia, is a necessary skill for doctors in the emergency service. Nevertheless, a recent study showed that from patients evaluated for chest pain and discharged from the emergency services, 2-13% presented an AMI that was not diagnosed[6].

First described in 1980, the Wellens syndrome is not common in medical practice. It represents a high risk acute coronary syndrome and it is associated with severe stenosis of the proximal segment of the left anterior descending coronary artery[4,5,7]. Such clinical condition, if not previously identified and treated, can evolve into an anterior wall AMI to death[8-10].

The appropriate conduct management in this subgroup of patients allows changing the natural history of their disease.

CASE REPORT

Personal history and physical examination

Male of 56-year-old, farm worker, high level of physical effort and pathological medical history of hypertension, smoker of a pack a day, occasional drinker, and family pathological history of deceased father by AMI. He was admitted in our center for an elective coronary angiography scheduled due to changes in the clinical characteristics of an exertional angina related to his regular work, which began to appear at lower thresholds. In the physical examination on admission, the patient was not afflicted with chest pain. Heart rhythmic sounds of good intensity without third sound, no cardiac murmurs, and absence of thermal gradient or edema in lower limbs. Blood pressure of 140/90 mmHg, and heart rate of 78 beats per minute. Respiratory system with vesicular murmur in lung fields, absence of rales and the rest of the physical examination normal.

Complementary tests

- Laboratory tests: blood count, coagulation and other blood profiles were normal. CPK when admitted of 156 units.
- Electrocardiogram when admitted: sinus rhythm, deep negative T waves across the anterior wall (V_1-V_6) (**Figure 1**).
- Chest X ray: Cardiac silhouette within normal limits, absence of congestive pattern or pleural effusion.
- Transthoracic echocardiogram when admitted: Evidence of mild hypokinesia of the upper segments of the left ventricle with preserved systolic function and structurally healthy valvular systems without pericardial effusion, or other alterations of interest.

Clinical evolution

While awaiting the coronary angiography, the patient presented intense midthoracic anchor, suddenly, with electrocardiogram compatible with large anterior AMI (important ST segment elevation in anterior wall [**Figura 2**]), with no response to fibrinolytic reperfusion therapy. The AMI became complicated with ventricular tachycardia that rapidly degenerated into ventricular fibrillation, which did not answer to cardiovascular resuscitation and the patient died a few hours after his admission.

Necropsy

In the necropsy analysis was confirmed the presence of severe atherosclerotic disease of the proximal segment of the left anterior descending coronary artery with superimposed thrombosis and extensive anterior transmural infarction.

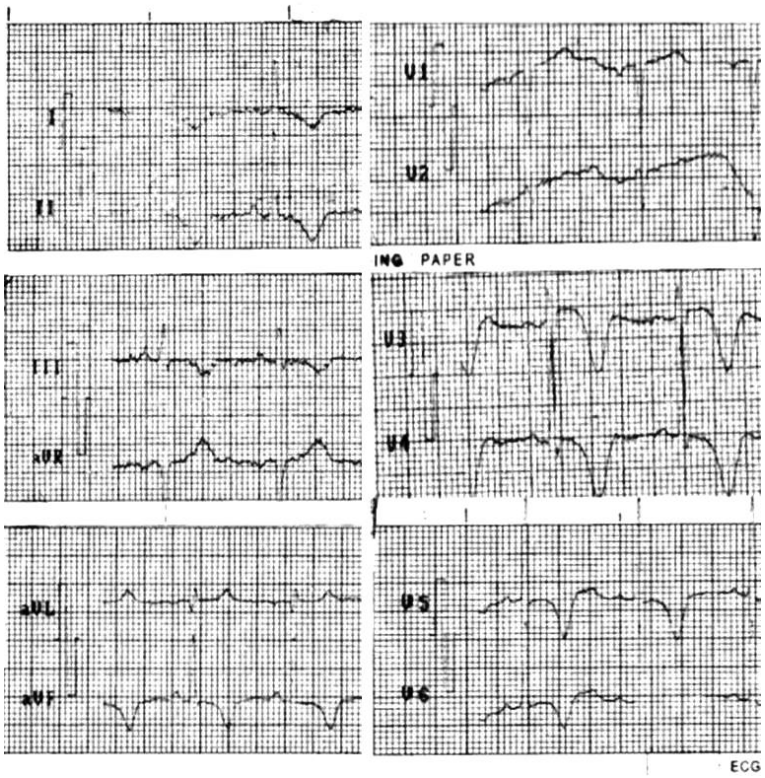

Figure 1. Electrocardiogram at admission, without chest pain, where repolarization abnormalities that can be found in the Wellens syndrome are observed.

COMMENT

The Wellens syndrome, also called the left anterior descending coronary artery syndrome, was first described in the 1980s, when a subgroup of patients with unstable angina, showing specific changes of the T wave in the precordial leads was observed, and which subsequently developed a large anterior wall AMI[4].

Clinical and electrocardiographic criteria for the diagnosis of Wellens syndrome are[4,5,7]:

1. Biphasic or deeply inverted T waves in V_2 and V_3 or, occasionally, V_1 and V_4-V_6,
2. Normal or minimally elevated cardiac enzymes,
3. Normal ST segment or slightly elevated (<1 mm),
4. No loss of R waves' progression in precordial leads,
5. Absence of pathological Q waves, and
6. Chest angina.

Two variants of the syndrome are recognized[4,6,8-10]:

- Type 1: Corresponds to the minority of cases (24%) and it is characterized by the finding of biphasic T wave (plus/minus) on the V_2 and V_3 leads, but may also include V_1-V_5/V_6.
- Type 2: Represents most cases (76%) and shows T wave with deep and symmetrical inversion, typically in the V_2 and V_3 leads, that may be present from V_1-V_4 and possibly in V_5 and V_6.

The most common way is the deep inversion of the T wave's segment in the precordial leads and it is the one present in our patient (Figure 1). It is considered a preinfarction stage of the coronary artery disease. Therefore, the causes of this syndrome are similar to any of the coronary artery disease: atherosclerotic plaques, superimposed vasospasm, increased cardiac demand of oxygen and hypoxia.

The Wellens syndrome, in particular, represents a critical stenosis of the left anterior descending artery that can lead to severe ventricular dysfunction and congestive cardiac failure, and if occludes, ventricular arrhythmias and death[11,12].

The electrocardiographic changes described in this syndrome have high specificity for the diagnosis of major atherosclerotic disease of the proximal left anterior descending coronary artery, at great risk of developing AMI and high mortality; thus, for these patients, it must be ideal to overlook the ergometry tests and to urgently indicate the coronary angiography to determine the extent of the disease and, if possible, to perform a percutaneous coronary intervention[2,11].

The diagnosis of the Wellens syndrome is electrocardiographic, as a variant of high-risk unstable angina, and it deserves early recognition to achieve better performance, from the therapeutic point of view, on the obstruction of the proximal left anterior descending coronary artery[13].

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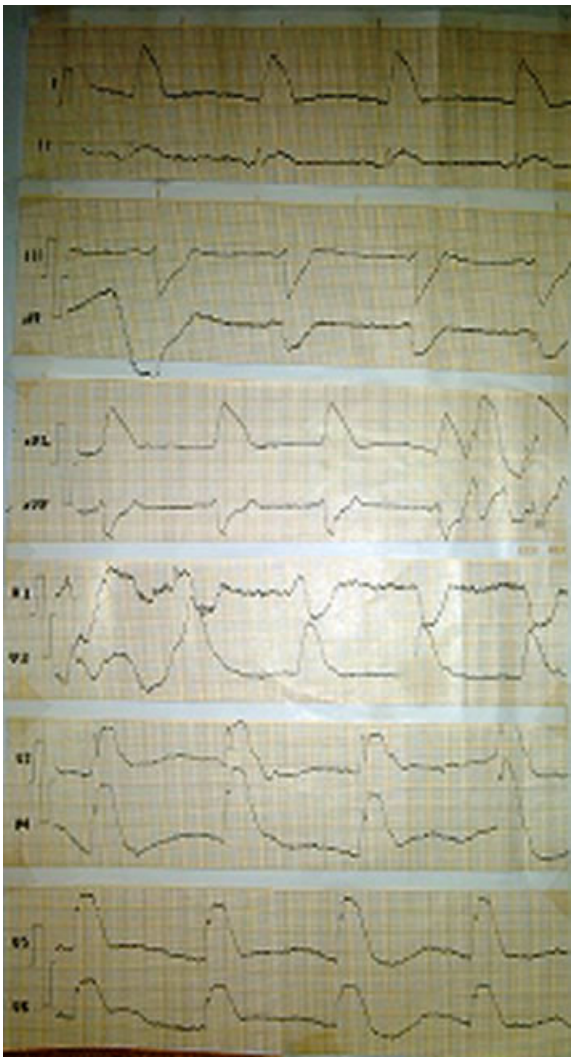

Figure 2. Electrocardiogram with large anterior wall AMI: ST segment elevation in D_I, aVL and V_2-V_6.

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