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WELLENS SYNDROME: A SERIES OF CASES REGULATED BY THE SERGIPE TELECARDIOLOGY SERVICE

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Abstract: Introduction: Wellens Syndrome (WS), also known as "Anterior Descending Coronary T-wave Syndrome", presents the following characteristics: previous history of chest pain; absence of pathological Q waves; normal progression of precordial R waves; little or no elevation of cardiac markers; small or no ST elevation; biphasic or symmetric and inverted T waves in V2 and V3, occasionally in V1, V4, V5 and V6. WS is difficult to diagnose and requires high clinical suspicion, since negative T waves are not only present in it. Because it is a serious condition that is difficult to diagnose, the recognition of electrocardiographic changes is essential to allow better outcomes in the management of the disease. Therefore, this article has as main objective to report 5 cases of Wellens Syndrome regulated by the Telecardiology Service in Sergipe, correlating electrocardiographic findings with angiographic changes to highlight the relevance of early management in the disease. **Cases Reports:** There were reported 5 cases of patients treated at the emergency room with complaints of chest pain, different electrocardiographic presentation, cardiac necrosis markers not always altered and due interventionist conduct with catheterization. **Discussion:** Once the diagnosis of Wellens syndrome is made or suspected, a cardiologist must be consulted. Contact with an interventional cardiologist must be recommended because the definitive treatment is cardiac catheterization with percutaneous coronary intervention. It is important to note that Wellens patients are at increased risk of developing AMI even if treated with drug therapy alone. Therefore, definitive treatment is procedural. In the cases presented, the Telemedicine Services in Sergipe favored an early and accurate diagnosis through the analysis by a cardiologist of the changes present in the electrocardiogram suggestive of AMI. **Conclusions:** The patient

with WS benefits from an early invasive strategy to reduce their rate of infarction and death, but to achieve this goal it must be recognized early. One of the most insightful strategies in this regard is the Telecardiology service.

Keywords: Wellens Syndrome, Acute Coronary Syndrome, Telecardiology.

INTRODUCTION

Acute Coronary Syndrome (ACS) can be classified into (a) unstable angina and non-ST-segment elevation acute myocardial infarction (SCASSST) and (b) ST-segment elevation acute myocardial infarction (SCACSST), usually caused by thrombosis and/or vasospasm of a coronary artery over or ruptured atherosclerotic plaque.

The clinical picture of ACS is not always typical, as are the presentations of its exams, so patients with ischemic heart disease may be underdiagnosed and not receive adequate management.

Chest pain assessment protocols reinforce the importance of performing an electrocardiogram (ECG) early, within 10 minutes of arrival at the emergency room. It is a highly sensitive method for screening for ischemic heart disease. When changes suggestive of ischemia are not seen in the presence of pain, the risk of acute myocardial infarction (AMI) is approximately 4% in patients with a previous history of coronary artery disease and 2% in patients without a history.

Faced with an ACS without ST-segment elevation, it is important to apply risk stratification (Table 1) and the valued parameters to choose the best strategy in these cases (Table 2).

In 1982, De Zwaan and Wellens described a subgroup of patients who were hospitalized for unstable angina and who were at high risk for the development of previous AMI. These patients had similar electrocardiographic findings.

From this, Wellens Syndrome (WS), also known as "T-wave Syndrome of the Anterior Descending Coronary", was described in the same year by the Dutch physician who contributed to the characterization of the reentry mechanism in Wolf Parkinson White syndrome. WS has the following characteristics: previous history of chest pain; absence of pathological Q waves; normal progression of precordial R waves; little or no elevation of cardiac markers; small or no ST elevation; biphasic or symmetric and inverted T waves in V2 and V3, occasionally in V1, V4, V5 and V6.

The disease is classified into two types (Figures 1, 2 and 3): in the first, which affects 24% of cases, biphasic T waves are identified in leads V2 to V3; the second, more common, shows symmetrical inverted T waves in leads V2 and V3 (occasionally V1 to V6).

Such electrocardiographic findings are not very sensitive (69%), but highly specific (89%) for major obstructive disease of the proximal segment of the anterior descending coronary artery, which, if not adequately addressed, can determine extensive anterior infarction and a high risk of mortality.

The causes of electrocardiographic changes are not fully understood. They may represent a myocardium stunned by reperfusion due to complete obstruction of flow in the proximal anterior descending artery that recovers spontaneously, since this mechanism suggests the action of other alterations besides the coronary atherosclerotic lesion, such as, for example, coronary spasm, nonspecific repolarization, right ventricular overload, or microvascular disease.

Another occasion to be observed is the well-known "pseudo-Wellens syndrome", which has been documented with the use of cocaine causing a coronary vasospasm and

Each item = 1 point	Age ≥ 65 years
	≥ 3 risk factors for CAD
	Prior CATE with stenosis ≥ 50%
0-2 points: low risk *3-4 points: intermediate risk* *5 ≥ points: high risk*	Elevation of myocardial necrosis markers
	AAS use in the last 7 days
	ST infra ≥ 0.5mm
	≥ 2 episodes of angina in the last 24 hours

CAD: Coronary artery disease; CATE: catheterization; AAS: acetylsalicylic acid.

Table 1. Risk stratification TIMI risk for patients with non-ST elevation acute coronary syndrome. Source: modified SBC (2019).

Immediate invasive strategy (< 2 hours)	*Hemodynamic instability and cardiogenic shock*
	Electrical instability with sustained ventricular tachyarrhythmia or ventricular fibrillation
	Refractory angina despite clinical treatment
	Acute mitral regurgitation
Early invasive strategy (< 24 hours)	High risk score (GRACE > 140)
	Elevation of myocardial necrosis markers
	Dynamic ST changes
Invasive strategy (in the first 72 hours)	Score TIMI ≥ 2, GRACE 109-140
	Presence of diabetes or renal failure (creatine clearance < 60 ml/min)
	Left ventricular dysfunction (FE < 40%)
	Prior PCI or MRI
"Conservative" (selective invasive) strategy	Low risk scores (TIMI 0-1, GRACE < 109)
	Patient preference
	Doubts about the nature of symptoms

Table 2. Valued parameters for choosing the best strategy in patients with non-ST elevation acute coronary syndrome.

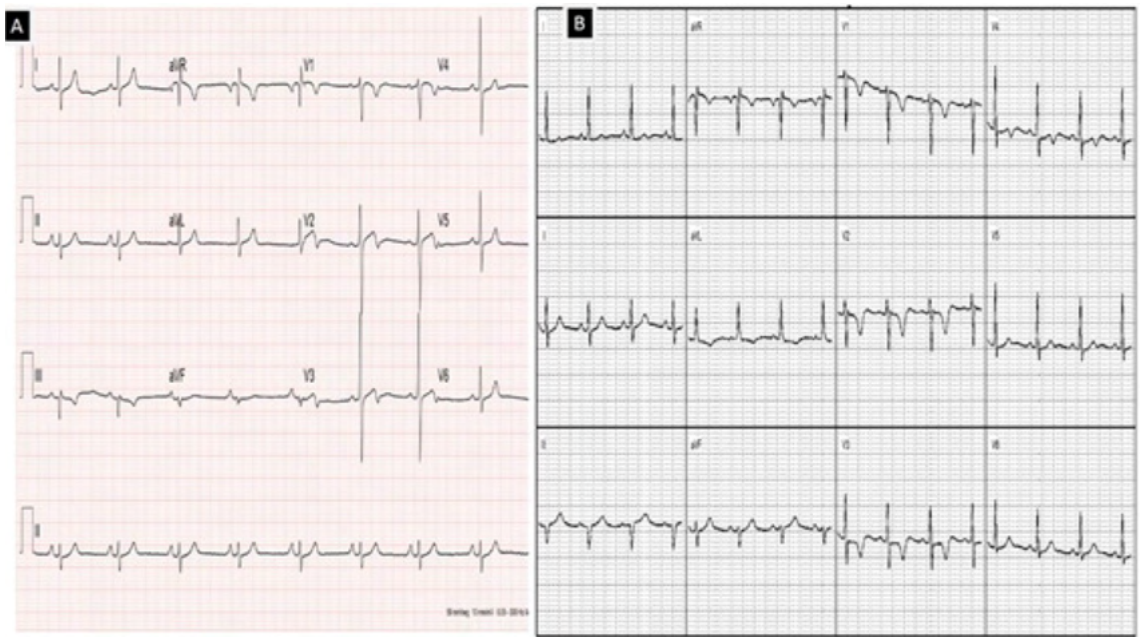

Figure 1. Electrocardiograms showing the patterns of Wellens syndrome (1st – Pattern A / 2nd – Pattern B).
Source: FONSECA, Eduardo et al. Arquivos Brasileiros de Cardiologia, v. 116, p. 363-366, 2021.

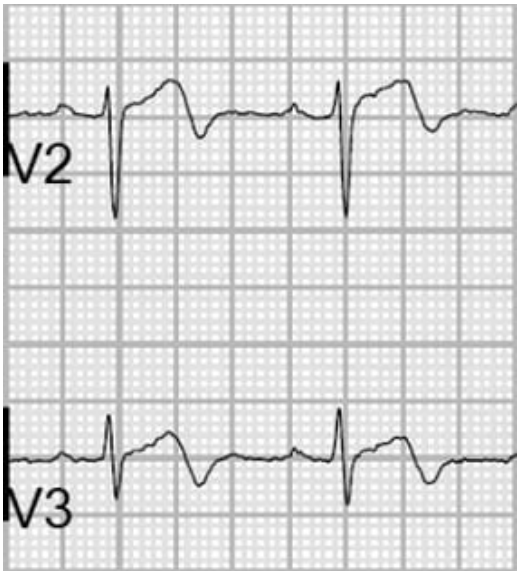

Figure 2. Biphasic T waves (Wellens type A).
Source: Alves (2020).

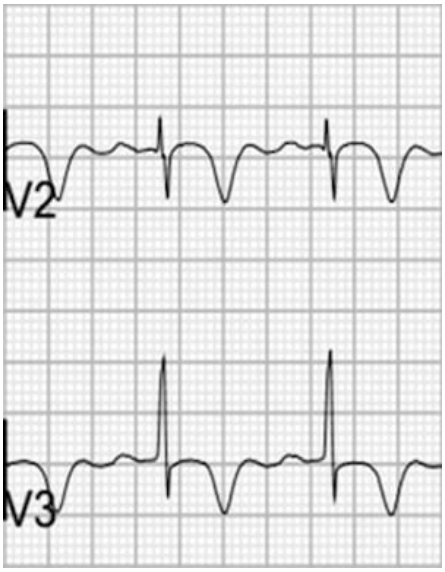

Figure 3. Symmetrical and deep T waves (Wellens type B).
Source: Alves (2020).

consequently altering the ECG, returning to normality after elimination of the substance by the body. The need for this recognition is due to the fact that these patients are harmed by the use of beta-blockers due to the potential stimulation of unopposed alpha receptors. Rarely, this T-wave negativity may also be present due to myocardial bridging or Takotsubo cardiomyopathy because of myocardial edema.

WS is difficult to diagnose and requires high clinical suspicion, since negative T waves are not only present in it, but also in other types of pathologies, such as: myocarditis, left ventricular hypertrophy, pulmonary embolism, Wolff Parkinson White syndrome, digitalis effects, ischemic events and juvenile T wave pattern. In addition, the patient may be asymptomatic at the time of the ECG, and cardiac necrosis markers are of little relevance in these cases, leading to the interpretation of electrocardiographic changes as nonspecific. In the presence of electrocardiographic findings of WS, the performance of provocative tests for ischemia is even discouraged.

As it is a serious condition that is difficult to diagnose, the Telemedicine coupling solution aimed at ACS was able to establish a more homogeneous relationship for the approach of this disease in the country and allow better outcomes. After all, Telemedicine plays a crucial role in supporting emergencies, particularly when the acute condition is life-threatening, requires prompt diagnosis and early treatment, and the patient and the specialist healthcare professional are physically separated by long distances.

Therefore, this article has as main objective to report 5 cases of Wellens Syndrome regulated by the Telecardiology Service in Sergipe, correlating electrocardiographic findings with angiographic changes to highlight the relevance of early management in the disease.

CASES REPORTS

CASE 1

RS, 62 years old, male, previous AMI, diabetic, hypertensive, previous heart disease, long-term smoker, goes to the emergency care unit with typical chest pain for 1 week that got worse in the morning of the day. On vital signs, blood pressure 60x44 mmHg, heart rate 49 bpm and 100% oxygen saturation. Complementary exams were requested (Figures 4, 5 and 6). Volume expansion, antiplatelet aggregation protocol, 4mg morphine and 5ml/h noradrenaline were performed. After stabilization, he had blood pressure of 105x79 mmHg, heart rate of 59 bpm and oxygen saturation of 100%, with slight improvement in pain. Negative cardiac necrosis markers.

CASE 2

J.A., 69 years old, 65 kg, female, hypertensive, was admitted to the Regional Hospital with chest pain that had started 24 hours before. The patient reported chest pain in retrosternal tightness, radiating to the back. She performed an ECG at admission (Figure 7) and had negative cardiac necrosis markers collected on admission. In the exams 3 hours later, troponin reacted and curved the CKMB from 23 to 31, with a new ECG (Figure 8). At that moment, she had already performed dual antiplatelet therapy with ASA and Clopidogrel. She underwent catheterization (Figure 9). She had a report of flu syndrome with resolution 8 days ago (no test for COVID-19).

For intervention, continuous cardiac monitoring and coronary angioplasty were performed with successful implantation of two drug-eluting stents in the proximal and middle portions of the circumflex artery (spasm was observed in the distal portions of the artery after stent release). The procedure

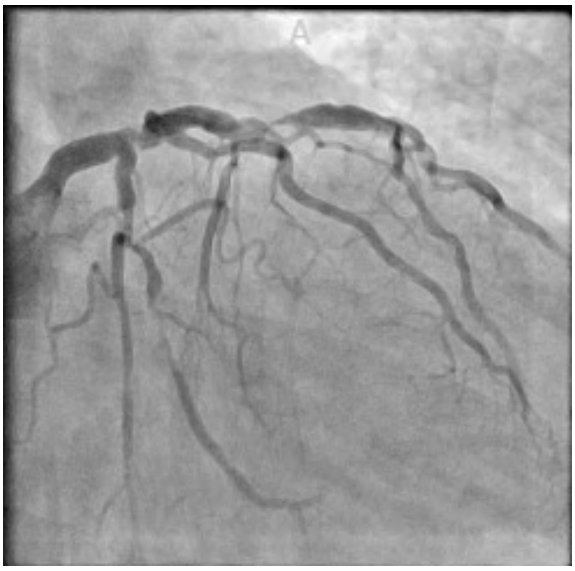

Figure 4. Catheterization showing mild to moderate diffuse atheromatosis in the Right Coronary Artery in the Posterior Descending Branch, Circumflex Artery, Anterior Descending Artery, in addition to issuing grade 2 collateral circulation to the Right Coronary.

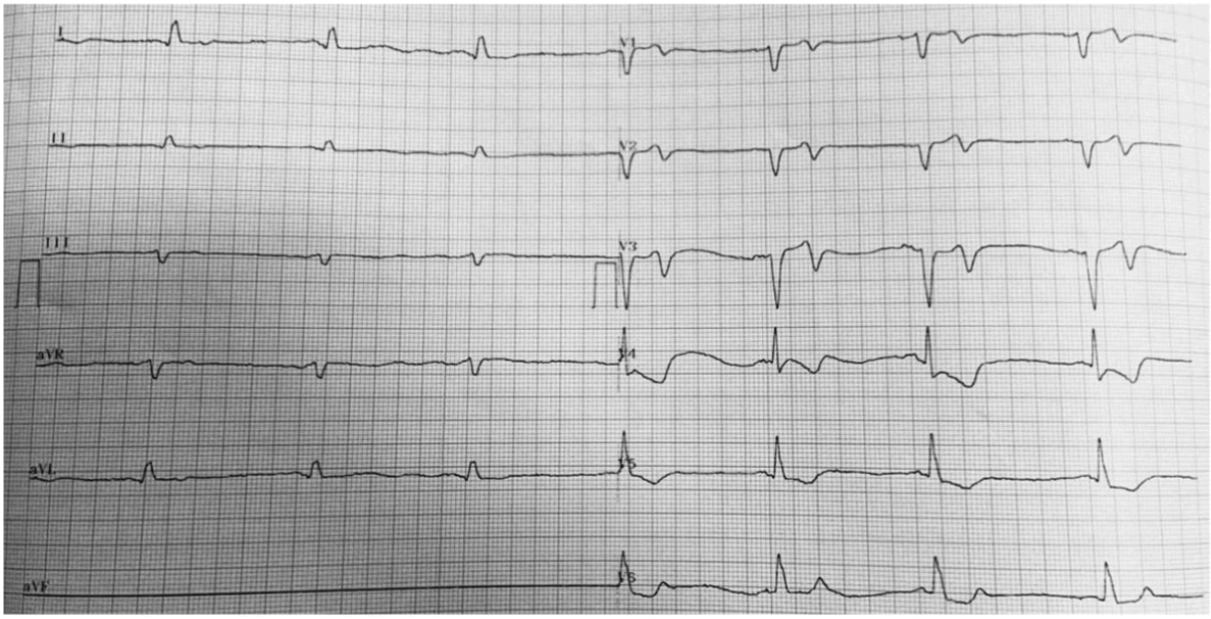

Figure 5. First electrocardiogram showing junctional rhythm, conduction delay by the left bundle branch, alteration of the ST segment in the anterior wall with biphasic T wave (plus-minus) in V1, V2 and V3 (Wellens A pattern) and diffuse alteration of ventricular repolarization.

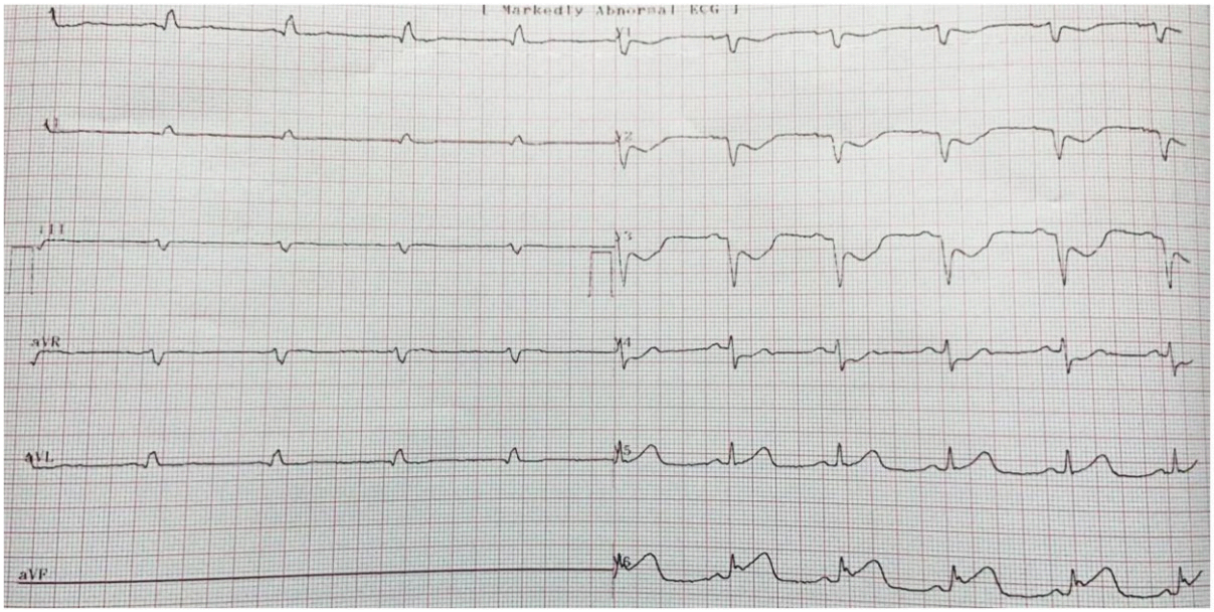

Figure 6. Second electrocardiogram showing sinus rhythm and lesion current in the laterodorsal wall compatible with evolving AMI.

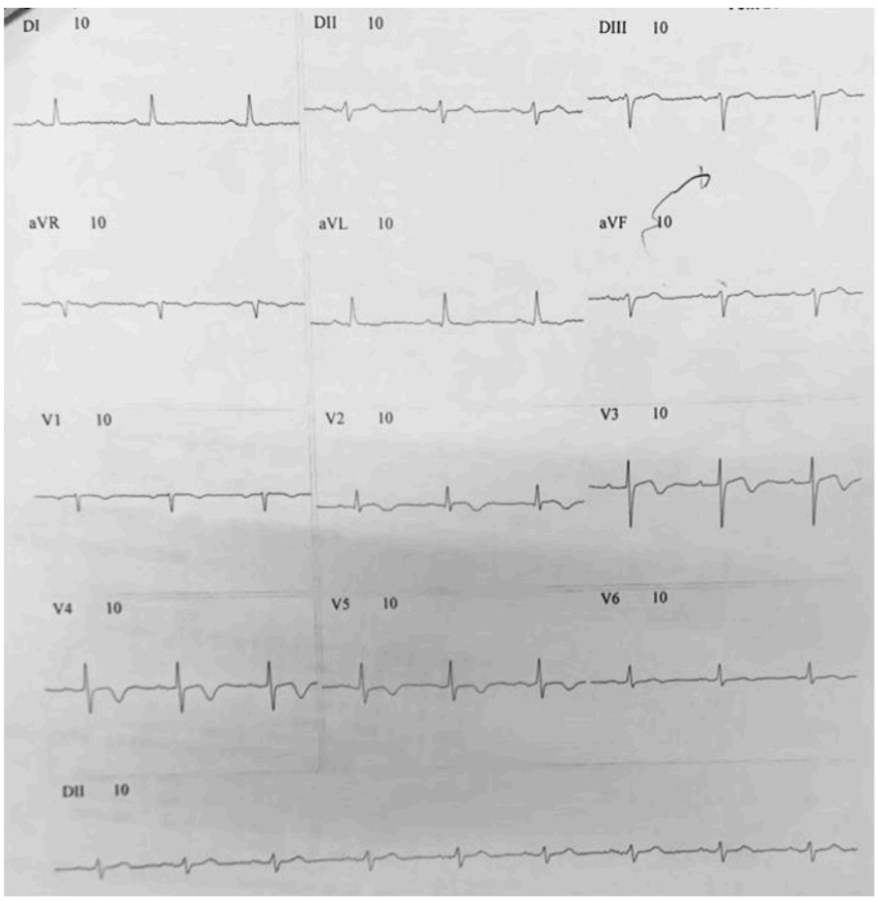

Figure 7. Admission ECG showing sinus rhythm with ST-segment change with a biphasic (plus-minus) T wave pattern in V2 and V3 (Wellens A pattern).

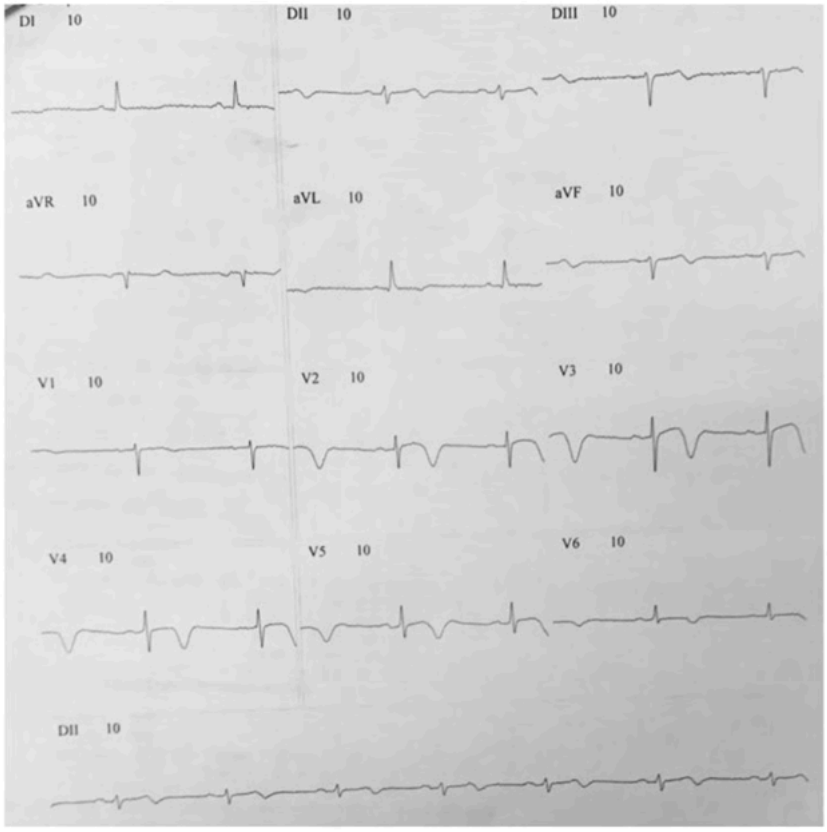

Figure 8. New ECG showing dynamic ST-segment change compared to the previous ECG. Presenting deep, symmetrical, inverted T wave in V2, V4 and V5 (Wellens's B pattern) and biphasic (plus-minus) T wave in V3.

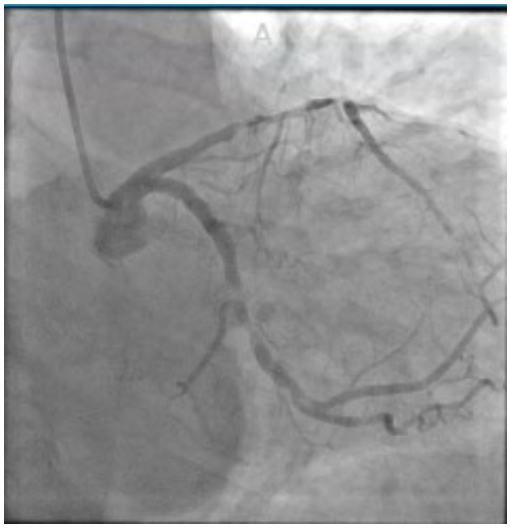

Figure 9. Catheterization showing segmental, subocclusive lesion, from mild to severe in the Anterior Descending Artery, Circumflex Artery, Third Marginal Branch, Fourth Marginal Branch, Right Coronary Artery, Posterior Ventricular Branch and Posterior Descending Branch.

followed with left control coronary angiography, removal of the sheath, local compression and dressing.

CASE 3

GDS, male, 64 years old, without comorbidities, sought a Private Hospital for 2 days of burning chest pain radiating to the back. He was referred to the Integrated Cardiology Unit. Cardiac necrosis markers also negative. Complementary tests were performed (Figures 10 and 11).

For intervention, direct implantation of the drug-eluting stent was successfully performed. Control cineangiography showed acute occlusion of the first diagonal branch, which is of medium caliber. Post-dilation of the previously implanted stent, a new control cineangiography showed the first diagonal branch with TIMI 1 distal flow. *kissing balloon* to open the stent mesh to the side branch and thus restore distal flow from the first diagonal branch. On angiographic control, there were no residual in-stent lesions or images of dissection and TIMI 3 distal flow in the anterior descending artery and in the first diagonal branch.

CASE 4

RSR, 72 years old, female, previous history of stroke. He sought the Regional Hospital for chest pain in the early hours of the same day, with improvement with the use of ASA and Clopidogrel after 4 hours of pain. He was stable at the time of admission, without pain since antiplatelet aggregation, with oxygen saturation of 99%, respiratory rate of 16 bpm, heart rate of 77 bpm and blood pressure of 138x90 mmHg. Complementary exams were also performed (Figures 11 and 12), with necrosis markers CPK 1260, CK-MB 182 and positive troponin.

CASE 5

MMBN, 45 years old, male, 80kg, without comorbidities and without continuous use of medication. He sought emergency care with a report of chest pain for the first time about 3 hours ago, with no triggering factor. At the time of consultation, asymptomatic. Vital signs: heart rate 80 bpm, respiratory rate 18 bpm, blood pressure 164x107, oxygen saturation 98% in room air, other physical examination without changes. Double antiplatelet aggregation was performed with AAS 300mg and Clopidogrel 300mg. No report of flu syndrome. ECG (Figure 13), catheterization (Figure 14) and myocardial necrosis markers were performed. Those with the following evolution: CK from 260 to 515, myoglobin from 66 to 91 and positive troponin.

For intervention, a successful coronary angioplasty with drug-eluting stent implantation was performed for the anterior descending artery.

DISCUSSION

The evaluation of the patient with ACS begins with the collection of a good anamnesis, mainly characterizing chest pain, the presence or absence of signs of severity such as syncope, dyspnea, persistent pain, potentially serious arrhythmias, and risk factors for complications. This pain can be classified according to its location, characteristics, duration and improvement and worsening factors.

Among the recommendations with good evidence in the initial care of ACS are: perform an ECG within a period of up to 10 minutes from arrival at the unit, in all patients with suspected ACS, repeat the ECG in cases without a definition of diagnosis after 6h, at least once, or recurrence of symptoms; perform a new ECG with leads V3R-V4R, V7-V9 in those patients with a 12-lead exam

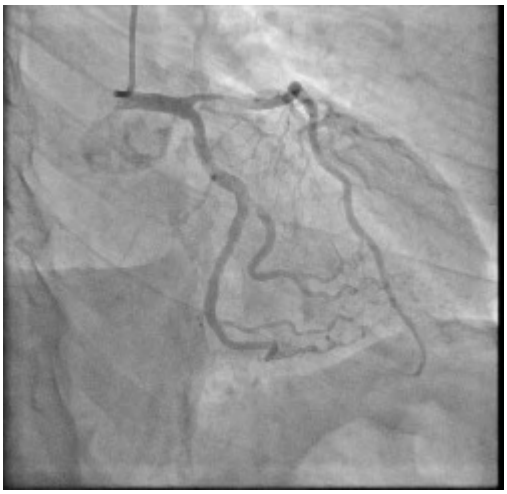

Figure 9. Catheterization showing severe lesion in the Anterior Descending Artery and first diagonal branch, in addition to a mild lesion in the Circumflex Artery and large marginal branch. The Right Coronary Artery exhibits discrete parietal irregularities along its course.

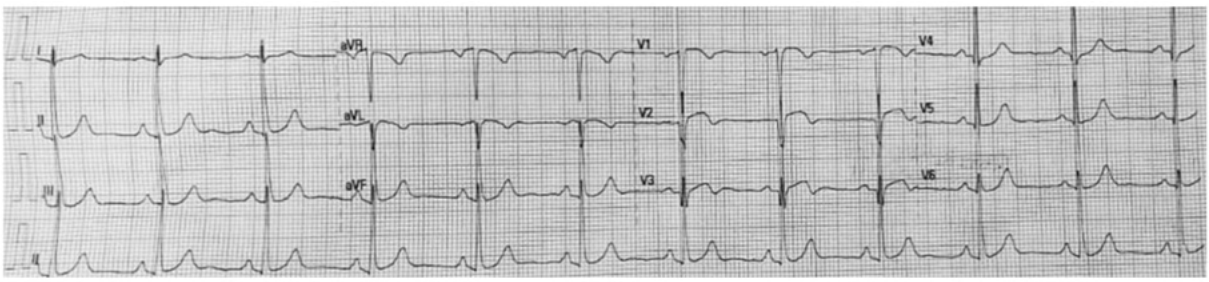

Figure 10. Admission ECG showing sinus rhythm with ST-segment change in the anterior wall with biphasic (plus-minus) T waves in V2-V3 (Wellens A pattern).

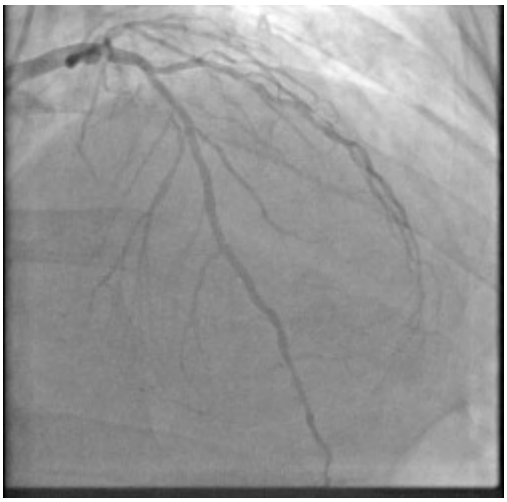

Figure 11. Catheterization showing severe obstructive coronary artery disease.

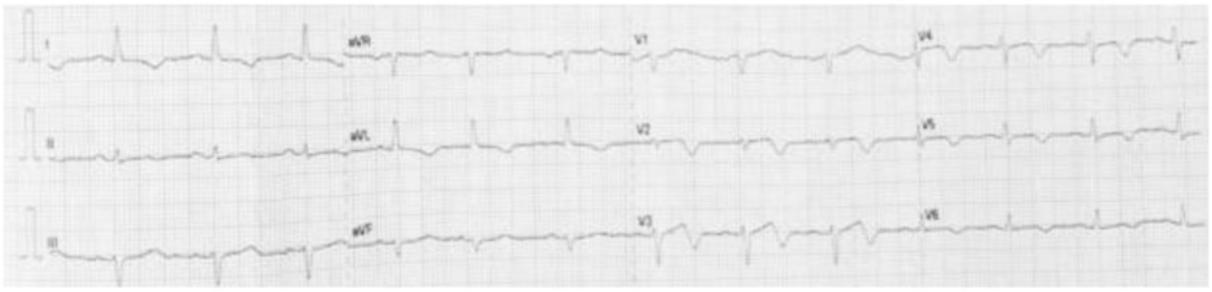

Figure 12. Admission ECG showing sinus rhythm with ST-segment change in the anterior wall with a biphasic (plus-minus) T wave in V2-V3 (Wellens A pattern). Diffuse change in ventricular repolarization.

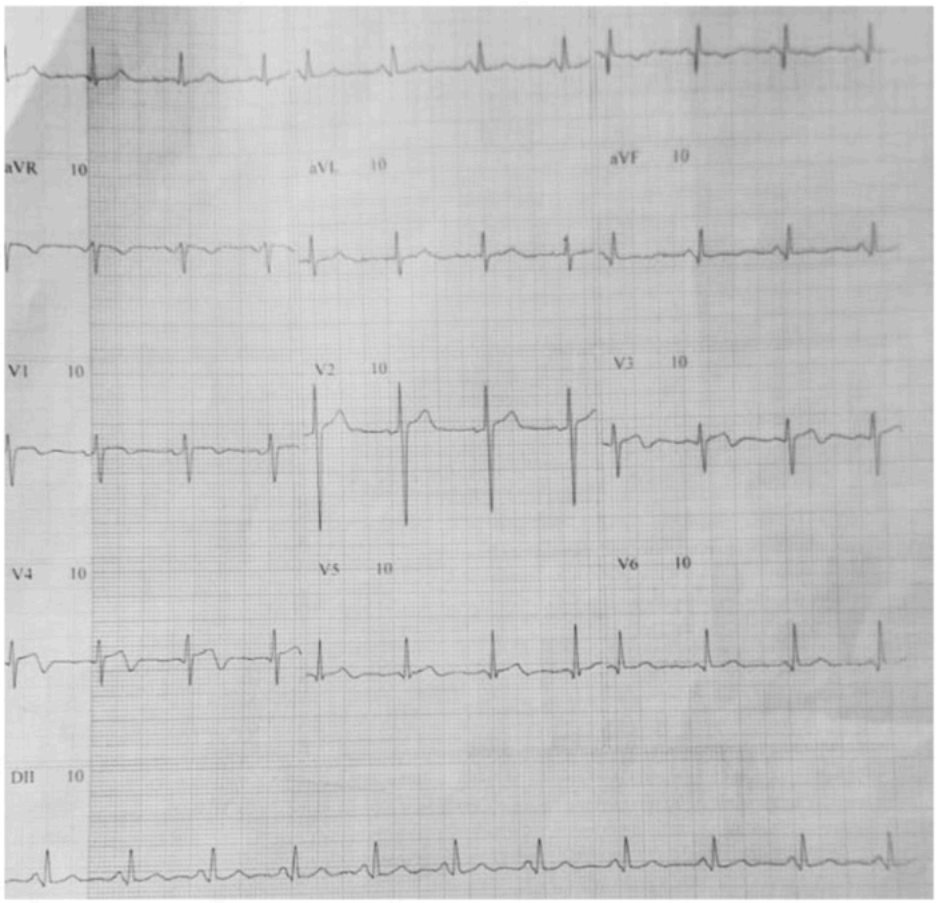

Figure 13. ECG showing sinus rhythm with ST-segment alteration in the anterior wall with biphasic T waves (plus-minus) in V3 (Wellens A pattern).

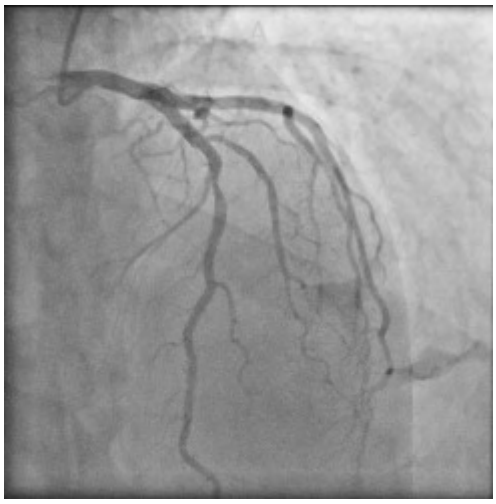

Figure 14. Catheterization showing parietal irregularities in Left Coronary Trunk, Diagonal Branches, Circumflex Artery, Marginal Branches, Right Coronary Artery, Ventricular Branches and Posterior Descending Branches. The Anterior Descending Artery with 70-80% lesion in the middle third associated with thrombus imaging.

without justifying changes and who remain with complaints.

The importance of recognizing this pattern is to perform early percutaneous coronary intervention in order to identify the occlusion and, if possible, treat it. In the aforementioned cases of Wellens Syndrome, for example, a frequent manifestation of chest pain is observed, but with an atypical aspect. In addition, in only 3 of the 5 cases there was an increase in cardiac necrosis markers at the first examination. The electrocardiographic changes were also subtle and, despite the absence of ST-segment elevation, they presented flow obstruction in the anterior descending artery associated with other changes.

Once the diagnosis of Wellens syndrome is made or suspected, a cardiologist must be consulted. Contact with an interventional cardiologist must be recommended because the definitive treatment is cardiac catheterization with percutaneous coronary intervention. Until this occurs, WS must be treated similarly to an acute myocardial infarction, including antiplatelet therapy with aspirin, anticoagulation with heparin and nitrates, and beta-blockers if the patient is not hypotensive. However, it is important to note that Wellens patients are at increased risk of developing AMI even if treated with drug therapy alone. Therefore, definitive treatment is procedural.

In the cases presented, the Telemedicine Services in Sergipe favored an early and accurate diagnosis through the analysis by a cardiologist of the alterations present in the electrocardiogram suggestive of AMI, the conduct of the treatment, whether or not the recommendation of thrombolysis and/or the administration of other medications, monitoring of vital signs and evolution of the patient's condition. All of this is provided through real-time communication between the medical team of the emergency care unit and the cardiologist at a distance from the pre-hospital service or hemodynamics center that will receive the patient. Therefore, with the competence to perform an essential function in the lines of care of the SCA.

CONCLUSIONS

Despite the existence of well-established criteria for the detection of WS, these electrocardiographic alterations are not properly valued and this is largely justified by the fact that their description is relatively recent.

The major issue of this syndrome is the evolution associated with AMI, but with subtle or absent clinical and laboratory manifestations, which induces the attending physician to adopt a generally conservative approach with little impact on a good prognosis. The patient with WS benefits from an early invasive strategy to reduce their rate of infarction and death, but to achieve this goal it must be recognized early.

One of the most insightful strategies in this regard is the Telecardiology service. Such a system can not only improve survival and reduce the costs of approaching these patients, but also reflect on the improvement of the urgency and emergency system in a global way, since acute coronary syndrome is an important cause of morbidity and mortality, in Brazil and in the United States.

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