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2020

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Review

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Missing heart attacks during confinement

Pocos infartos del miocardio en la pandemia de COVID-19

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Keywords:
Coronavirus, pandemic, heart attack, myocardial infarction, stroke, fear of contagion.

Palabras clave:
Coronavirus, pandemia, infarto de miocardio, infarto cerebral, ataque, miedo al contagio.

ABSTRACT

The current COVID-19 home isolation is perhaps a cause for a significant reduction in the presentation and late arrival of cases of myocardial and cerebral infarction. Several publications announce the significant drop of patient's volume with myocardial infarction in China, Italy, France, Spain and The United States, warning possible impact on global fatality and patient's disability, that may be higher than the COVID's related. This paper warns about similar behavior in Mexico with possible sequelae.

RESUMEN

El aislamiento domiciliario actual de COVID-19 es quizás una causa de reducción significativa en la presentación y llegada tardía de casos de infarto de miocardio y cerebral. Varias publicaciones anuncian la caída significativa del volumen de pacientes con infarto de miocardio en China, Italia, Francia, España y Estados Unidos, advirtiendo un posible impacto en la mortalidad global y discapacidad, que puede ser mayor que la relacionada con COVID. Este artículo advierte sobre un comportamiento similar en México con posibles secuelas graves.

INTRODUCTION

The current COVID-19 pandemic has caused a significant change in social behaviour, with the predominance of home isolation, due to the successful stay-at-home campaign. In several countries it has been noted that the fear of contagion in hospitals has caused a significant reduction in the presentation of cases of myocardial and cerebral infarction; not only are they fewer patients but those who finally arrive at the hospital do so much later than in the months corresponding to the last year.

One of the initial publications comes from the experience of a Hong Kong hospital, in which the delay between the onset of symptoms and arrival at the hospital, was four times longer. If we take into account that current protection strategies require more time to transfer patients to the Cath Lab, the total cost in time is even higher, with the consequent impact on mortality and complications.[1]

The Italian experience, based on a national and multicenter registry, observed a reduction in general admissions for heart attack of 48% and more than 50% in the case of heart attacks with ST-elevation. These changes have statistical significance concerning the same months of 2019 and have a definite impact on mortality, with a risk ratio of 3.3.[2] The impact of increasing home cardiac arrests can also, be very significant, and some of Italy's regions reach almost 200%.[3]

A multicenter registry from France compared admission to intensive cardiac units of 4.8 ± 1.6, before confinement, to 2.6 ± 1.5 patients per day, during confinement ($p = 0.0006$). The authors confirm a dramatic drop in acute cardiovascular cases and consider that it is time to sound the alarm that these patients may suffer from inattention and severe consequences, with an increase in ambulatory myocardial infarctions, mechanical complications of heart attack, heart failure, unexplained deaths, among others.[4]

A Spanish study, with the heart attack code, which involved all the autonomous communities reported reductions of 40 to 81% in diagnostic and therapeutic cardiovascular procedures; warning about the risk of increased morbidity and mortality, and suggested that

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scientific societies and health authorities should promote that patients with heart attack symptoms demand assistance from the health system in order to receive reperfusion treatment adequately.[5] In this regard, it is worth reviewing a case published by Saquib Masroor, from The United States, of a complication that is already unusual, of the ruptured interventricular septum.[6]

A letter to the editor, from the New England Journal of Medicine, reveals that a phenomenon similar to myocardial infarction occurs with imaging studies of stroke cases in the United States, which could have an impact on acute fibrinolytic treatment.[7]

All the phenomenon has generated alarm in several countries, because myocardial infarction and cerebral infarction are prominent causes of death and disability, with a severe possible impact on general mortality and viability, accompanied by potentially disabling sequelae and with enormous costs of secondary and tertiary prevention and rehabilitation. For this reason, the Society for Cardiac Angiography and Intervention (SCAI) conducted a questionnaire that helps answer questions about why people with these problems and other emergencies do not go to hospitals, finding fear of contagion. The society actively encourages people to go to the hospital in case of symptoms.[8,9]

Cardiovascular diseases are the primary cause of death in México, with strategies that have been ineffective in themselves in the treatment of acute coronary syndrome and stroke, with one of the highest fatality rates among the countries of the Organization for Economic Co-operation and Development (OECD).[10] Addressing this problem during the pandemic could help reduce the impact of these entities' mortality and complications and would help remedy the previously observed high case fatality because the consequences are even more severe than those of coronavirus infection.

The members of the Mexican College of Interventional Cardiology and Endovascular Therapy (COMECITE for Colegio Mexicano de Cardiología Intervencionista y Terapia Endovascular) have the same perception of a reduction in the care of patients with myocardial infarction. We consider it urgent to start a parallel campaign to confinement, with understanding and analyzing the phenomenon to inform the nation «stay home to avoid infection», but go to the hospital immediately, in case you have symptoms of myocardial infarction. This campaign must expose the consequences of the unattended heart attack and that the hospitals and all the health systems have reserved areas for the diagnosis and treatment of problems out of coronavirus contamination.

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Original Research

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Prevalence and clinical-therapeutic profile of atrial fibrillation in private cardiology offices in northeast Mexico

Prevalencia y perfil clínico-terapéutico de la fibrilación auricular en consultorios de cardiología privados del noreste de México

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Mario Alberto Carrillo Pérez,§ Juan Manuel Solís Soto‖

Keywords:
Atrial fibrillation, atrial fibrillation ambulatory prevalence, oral anticoagulant, new oral anticoagulant, vitamin k antagonist, mineralocorticoid receptor antagonist, angiotensin receptor blockers.

Palabras clave:
Fibrilación auricular, prevalencia ambulatoria de fibrilación auricular, anticoagulante oral, nuevos anticoagulantes orales, antagonistas de la vitamina K, antagonistas de los receptores de mineralocorticoides, antagonistas de los receptores de angiotensina II.

ABSTRACT

Introduction: The prevalence of atrial fibrillation (AF) in Mexico is unknown. **Objectives:** To document AF prevalence and a clinical-therapeutic profile. **Methods:** Cross-sectional study in three private cardiology offices in Northeast Mexico; in 337 patients from 8,999 clinical records, AF was documented. **Results:** AF prevalence was 3.74%, permanent 56.0%, paroxistic 28.4%, persistent 15.7%; non valvular atrial fibrillation (NVAF) 94.06%. Age 74 ± 12.89 years, women 52.22%, hypertension, 74.18%, smoking, 36.79%, alcoholism 35.01% and type 2 diabetes, 30.56%. The CHA_2DS_2-VASc ≥ 2 group vs 0-1, received more anticoagulant (OAC) (31.88 vs 8.33%) and new oral anticoagulant (NOAC) (53.31 vs 13.33%) $p < 0.01$ for both. In ages 60-75, the CHA_2DS_2-VASc ≥ 2 group vs 0-1, received more OAC (34.89 vs 6.25%) $p = 0.0004$, and NOAC (54.16 vs 0%) $p < 0.01$. The CHADS group ≥ 2 vs 0-1, used more OAC (33.6 vs 18.8%) and NOAC (55.17 vs 34.11%) $p < 0.01$ for both and vitamin K antagonist (VKA) (12.06 vs 3.5%) $p = 0.004$. The CHA_2DS_2-VASc group ≥ 2 vs 0-1, had more women than men (95.03% versus 85.9%), received more diuretics (57.49 vs 13.33), mineralocorticoid receptor antagonist (MRA) (27.52 vs 0%) and angiotensin receptor blockers (ARB) (55.40 vs 16.66%) $p < 0.01$ for all. **Conclusion:** In our patients, AF is characterized by having a clinical profile of high cardiovascular risk, alcoholism, senility and predominance of women, with a prevalence (3.74%) similar to that of other western countries.

RESUMEN

Introducción: *La prevalencia de fibrilación auricular (FA) en México es desconocida.* ***Objetivos:*** *Documentar la prevalencia de FA y su perfil clínico-terapéutico.* ***Métodos:*** *Estudio transversal, en tres consultorios privados de cardiología; en 337 de 8,999 expedientes clínicos, la FA fue documentada.* ***Resultados:*** *Prevalencia de FA, 3.74%; permanente 56.0%, paroxística 28.4%, persistente 15.7%. FANV 94.06%. Edad, 74 ± 12.89 años, mujeres 52.22%, hipertensión, 74.18%, tabaquismo, 36.79%, alcoholismo 35.01%, diabetes tipo 2, 30.56%. El grupo CHA_2DS_2-VASc ≥ 2, comparado al 0-1, recibió más anticoagulación oral ACO (31.88 vs 8.333%) y nuevos anticoagulantes orales (NAO) (53.31 vs 13.33%) $p < 0.01$ para ambos. En pacientes de 60-75 años, se utilizaron más ACO en el grupo CHA_2DS_2-VASc ≥ 2 respecto al 0-1 (34.89 vs 6.25%) $p = 0.0004$ y NOA (54.16 vs 0%) $p < 0.01$. El grupo CHADS ≥ 2 comparado al 0-1, utilizó más ACO (33.6 vs 18.8%) NAO (55.17 vs 34.11%) $p < 0.01$ para ambos y antagonistas de la vitamina K (AVK) (12.06 vs 3.5%) $p = 0.004$. El grupo CHA_2DS_2-VASc ≥ 2 vs 0-1 tuvo más mujeres que hombres (95.03 vs 85.9%), recibió más diuréticos (57.49 vs 13.33%), antagonistas de los receptores de mineralocorticoides (ARM) (27.52 vs 0%) y bloqueadores de los receptores de angiotensina 2 (BRA 2) (55.40 vs 16.66%) $p < 0.01$ para todos.* ***Conclusión:*** *En nuestros pacientes la FA se caracteriza por tener un perfil de alto riesgo cardiovascular, alcoholismo, senilidad y predominio de mujeres, con una prevalencia (3.74%) similar a la de otros países occidentales.*

INTRODUCTION

With the increase in the life expectancy of the population, in the last 20 years there has also been a notable increase in the diagnosis of atrial fibrillation (AF). This arrhythmia, the most frequent in cardiological practice, is of special importance because it increases the risk of dementia, heart failure (HF) and embolic cerebrovascular disease (ECVD).[1] In addition to causing a deterioration in the quality of life, it increases hospital mortality and health care costs.

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The prevalence of AF in western countries is 1.9 to 3.0% in the general population, predominates in men and increases in frequency of onset with advancing age, in both sexes.[2]

The evidence about the prevalence and consequences of AF, comes from studies conducted in Europe and North America, whose data contrast with those provided by studies conducted in African-American and Indo-Asian populations, observing significant differences between these ethnic groups and the population white, as reported in the Indo-Asian population of the United Kingdom, with a very low prevalence of AF of 0.6%.[3,4] We do not know if these ethnic epidemiological differences also occur in Hispanic patients, since the epidemiology of AF is scarce in Latin America and Mexico. The results of the CARMEN AF study (in process)[5] will surely expand this information.

On the other hand, given the known difficulties in achieving optimal oral with vitamin K antagonists (VKA), a new oral anticoagulants (NOAC), of simple dosage and predictable pharmacokinetics appeared a decade ago, observing a progressive increases in their prescription and actually are currently the first-line oral anticoagulant (OAC) in the cardiological guidelines, with similar efficacy or higher to VKA in the prevention of ECVD, in patients with non-valvular atrial fibrillation (NVAF).[6] Despite this, some experts have pointed out that these OAC are still underutilized, considering suboptimal percentages of utilization less than 70% observed in countries like Mexico, Germany, United Kingdom, Canada and even in the health system of France, with 66.3%.[7]

The present work reviews the experience in the care of Mexican patients with AF, from a group of private cardiologists in Northeast Mexico, seeking to document their clinical-therapeutic profile, with special focus on the frequency of use of OAC.

Objectives: Primary objective: to document the prevalence of AF in Mexican patients treated in private cardiology offices in Northeast Mexico. Secondary objective: to establish a clinical-therapeutic profile.

MATERIAL AND METHODS

Cross-sectional and observational study of patients treated during the last 5 years in 3 private cardiology offices. Two of the offices were located in the State of Nuevo León (one in the city of Monterrey and another in San Nicolás de los Garza) and the other in the city of Tampico, Tamaulipas. From 8,999 clinical records, 337 were diagnosed with AF. Inclusion criteria: both sexes, any age and etiology, NVAF and valve (VAF), regardless of their duration. Exclusion criteria: undocumented AF. Qualitative and quantitative variables of interest were obtained, such as main clinical history, etiologies, left ventricular ejection fraction (LVEF) and pharmacotherapy. The CHADS and CHA_2DS_2-VASc scales were used to calculate embolic risk. The paroxysmal AF was identified by its sudden onset and spontaneous end, within 7 days of initiation. The persistent form was defined as one in wich the episode last more than 7 days. This includes cases that could stop spontaneously and those that end with pharmacologicalor electrical cardioversion. The permanent AF was defined as one that is decided not to revert to sinus rhythm.

Statistic analysis: Descriptive statistics. Continuous variables expressed as mean ± standard deviation; categorical variables as percentages. For the association of 2 categorical variables, T of proportions was used. Statistical analyzes were performed using the SPSS software. The tentative level of significance was 0.05.

RESULTS

We document a prevalence of AF of 3.74% in patients from the Mexican Northeast. The predominant type of this arrhythmia was the NVAF n = 317. VAF occurred in only 20 patients. The predominant form was permanent AF, followed by paroxysmal and persistent. *Table 1* displays the main demographic variables of these patients. These were senile subjects (age of 74.97 ± 12.89 years) with 52% of women, with high prevalence of cardiovascular risk factors such as hypertension, smoking, diabetes mellitus 2 and alcoholism. The etiologies most associated with this arrhythmia were hypertensive heart disease (46.29%), ischemic heart disease (27.29%), sinus node disease (7.12%), rheumatic heart disease n (4.74%) and dilated cardiomyopathy (4.45%). Some patients presented more than one etiological association

Table 1: Demography and backgrounds in 337 patients with atrial fibrillation.

	n (%)
Age in years, media ± SD	74.97 ± 12.89
Sex, % M/H	52.22 / 47.77
Smoking	124 (36.79)
Alcoholism	118 (35.01)
Drugs	2 (0.59)
Type 2 diabetes	103 (30.56)
Systemic hypertension	250 (74.18)
CKD	32 (9.49)
CPOD	38 (11.27)
Hyperthyroidism	5 (1.48)
Hypothyroidism	42 (12.46)
Dyslipidemia	79 (23.44)
Prior myocardial infarction	31 (9.19)
Prior TIA	16 (4.74)
Stroke/thromboembolism	36 (10.68)
Prior coronary stent	16 (4.74)
Prior pacemaker	22 (6.52)
CABG	8 (2.37)
Associated etiology	
CAD	92 (27.29)
Hypertensive cardiopathy	156 (46.29)
Sinus node disease	24 (7.12)
Mitral prolapse	7 (2.07)
Hypertrophic cardiomyopathy	4 (1.18)
Dilated cardiomyopathy	15 (4.45)
Restrictive cardiomyopathy	2 (0.59)
Rheumatic cardiomyopathy	16 (4.74)
Others	64 (18.99)
AF type	
Valvular (VAF)	20 (5.93)
No valvular (NVAF)	317 (94.06)
Permanent	189 (56.00)
Persistent	53 (15.70)
Paroxistic	96 (28.40)
Score CHA_2DS_2-VASc, media ± SD	3.80 ± 1.70
Score CHADS, media ± SD	2.29 ± 1.28
LVEF, media ± SD	50.08 ± 15.96
Left atrial thrombus	0.01%
Treatment	
Digoxin	90 (26.70)
Propafenone	15 (4.45)
Type III antiarrhythmic	130 (38.57)
ARB	169 (50.14)
ACEI	35 (10.38)
BB	170 (50.44)
Diuretic	184 (54.59)

Continuation of Table 1: Demography and backgrounds in 337 patients with atrial fibrillation.

	n (%)
MRA	89 (26.40)
OAC in AF	207 (61.42)
VKA	48 (14.24)
NOAC	159 (47.18)
OAC in NVAF	187 (59.00)
Nitrates	58 (17.21)
Antiplatelet therapy	101 (29.97)
Calcium channel blockers	98 (29.08)

Quantitative values are expressed in numbers and percentages.
F = female, M = male, CKD = chronic kidney disease, CPOD = chronic pulmonary obstructive disease, CABG = coronary artery bypass grafting, CAD = coronary artery disease, LVEF = left ventricle ejection fraction, ARB = angiotensin receptor blocker, ACEI = angiotensin converting enzyme inhibitors, BB = beta blockers, MRA = mineralocorticoid receptor antagonist, OAC = oral anticoagulant, VKA = vitamin K anticoagulant, NOAC = new oral anticoagulant, AF = atrial fibrillation, NVAF = non valvular atrial fibrillation.

and in 64 (18.99%) it was not possible to determine any, because they were isolated episodes of arrhythmia, solitary AF or arrhythmia related to drug, alcohol or idiopathic cause. The CHA_2DS_2-VASc score was 3.80 ± 1.70 standard deviation (SD), that of CHADS was 2.29 ± 1.28 (SD) and the LVEF of 50.08 ± 15.96% (SD).

Of 317 patients with NVAF, 59.0%, n = 187, were anticoagulated (85% NOAC and 15% VKA). In *Table 2*, the relationship between pharmacological prescription and score on embolic risk scales was analyzed. The CHA_2DS_2-VASc ≥ 2 group, compared to 0-1, received higher OAC and NOAC prescriptions, although not VKA. The CHADS group ≥ 2 with respect to 0-1, had very similar results. With other drugs, in the CHA_2DS_2-VASc ≥ 2 group versus 0-1, the prescriptions were higher in diuretics, mineralocorticoid receptor antagonist (MRA), angiotensin receptor blockers (ARB), calcium channel blockers (CCB) and nitrates. Also, in the CHADS group ≥ 2, prescriptions were higher, the diuretics, MRA, ARB, CCB and nitrates. CHADS group 0-1 only the prescription of antiplatelet agents (AP) was greater than in the group ≥ 2.

By dividing patients by age groups and analyzing the OAC prescription in relation to the score on the scales, it can be seen in *Table 3* that, in patients < 60 years of age, the use of OAC was higher in the CHA_2DS_2-VASc group ≥ 2 with respect to the group 0-1, although not individually for VKA or NOAC. Using the CHADS scale in this age group, the prescription of OAC, VKA or NOAC did not show differences between group ≥ 2 and group 0-1. In the next segment of 60-75 years of age, using the CHA_2DS_2-VASc scale there was a higher prescription of OAC and NOAC in the group ≥ 2 compared to 0-1. With the CHADS scale, more OAC and VKA were used in the group ≥ 2 with respect to 0-1. In the age group ≥ 76 years, the CHADS group ≥ 2 exceeded the group 0-1 in OAC, VKA and NOAC prescriptions.

The *Table 4*, shows the relationship of the rest of medications with the risk scales by age groups, in < 60 years of age CHA_2DS_2-VASc ≥ 2 group exceeded the 0-1 group, in use of diuretics, MRA, digoxin, and ARB. With the CHADS scale, a higher prescription was observed in the group ≥ 2 with respect to 0-1 in diuretics, MRA and ARB. Antiplatelet therapy (AP) and Propafenone reached higher prescription within the CHA_2DS_2-VASc 0-1 group compared to ≥ 2. Propafenone was also used more in the CHADS group 0-1. In patients aged 60-75, the CHA_2DS_2-VASc ≥ 2 group also exceeded the 0-1 group in the use of diuretics, MRA and ARB. Using the CHADS scale, the group ≥ 2 compared to 0-1, had a greater use of beta blockers (BB), diuretics,

MRA and ARB. In CHA_2DS_2-VASc 0-1, AP were used more frequently in this age group, than in the group ≥ 2. In the age group > 76 years, a higher prescription was also observed in the CHADS group ≥ 2 with respect to 0-1, in diuretics, MRA, angiotensin converting enzyme inhibitors (ACEI), ARB, CCB and propafenone. In this age group CHA_2DS_2-VASc ≥ 76 years was not evaluated, due to insufficient simple, 0-1 n = 2, ≥ 2 n = 181.

Table 5 shows the relationship between the score obtained on the CHA_2DS_2-VASc and CHADS scales with the proportion of female or male patients in each category. In the CHA_2DS_2-VASc ≥ 2 group, a greater number of women than men can be observed (153, 95.03% versus 134, 85.9%) p < 0.01. In contrast the group 0-1 presented a greater proportion of men than women (14.1 vs 4.97%) p = 0.0363.

DISCUSSION

The results of the present study show in this large group of Mexican patients in Northeast Mexico a AF prevalence comparable to that of other western countries. This statistical data can be considered relevant, since as far as we know, there was no information on the AF prevalence in our country. As we know, in Mexico 60.7% of tachyarrhythmias are caused by AF, considering this arrhythmia as a determining factor that conditions high percentages of ECVD, a pathology that according to the Brain Attack Surveillance project in Durango presents prevalences of 18 cases per 1,000 inhabitants over 65 years of age, considering it a true public health problem.[8] Knowledge of the epidemilogical reality in our setting is relevant, since a large number of studies in epidemiology and treatment for this arrhythmia have frequently underrepresented some population groups. Our work evaluated Hispanic patients, an ethnic group with little representation in large population registries and in randomized clinical trials (RCTs). As can be seen when analyzing together the data of the GARFIELD-AF and ORBIT-AF I and II records[9] (n = 73,004 patients) 69.24% of the subjects were Caucasian, 6.0% Hispanic, 1.79% Black

Table 2: Relationship between pharmacological prescriptions and risk category in the CHA_2DS_2-VASc and CHADS scales.

	CHA_2DS_2-VASc			CHADS		
	0-1	≥ 2		0-1	≥ 2	
Drugs	n = 30	n = 287	p	n = 85	n = 232	p
OAC	8.33	31.88	< 0.01	18.82	33.62	< 0.01
VKA	3.33	10.45	0.0665	3.52	12.06	0.004
NOAC	13.33	53.31	< 0.01	34.11	55.17	< 0.01
BB	50	49.82	0.985	43.52	52.15	0.175
Type 3 antiarrhythmic	33.33	40.06	0.469	45.88	37.06	0.164
Diuretics	13.33	57.49	< 0.01	22.35	64.65	< 0.01
MRA	0	27.52	< 0.01	9.41	30.60	< 0.01
Antiplatelet	46.66	30.31	0.099	41.17	28.48	0.039
Propafenone	16.66	3.13	0.063	5.88	3.87	0.485
Digoxin	16.66	26.48	0.192	20	27.58	0.151
ACEI	13.33	10.45	0.663	9.41	11.20	0.637
ARB	16.66	55.40	< 0.01	23.52	62.06	< 0.01
CCB	20	31.35	0.159	17.65	34.91	< 0.01
Nitrates	3.33	19.51	< 0.01	10.58	20.68	0.019

OAC = oral anticoagulant, VKA = vitamin K antagonist, NOAC = new oral anticoagulant, BB = beta blockers, MRA = mineralocorticoids receptor antagonist, ACEI = angiotensin converting enzyme inhibitors, ARB = angiotensin receptor blockers, CCB= calcium channel blockers.

Table 3: Relationship of anticoagulant prescription by age group and category CHA_2DS_2-VASc and CHADS.

	CHA_2DS_2-VASc			CHADS		
Anticoagulants	0-1	≥ 2	p	0-1	≥ 2	p
< 60 years	n = 44	n = 22		n = 48	n = 18	
OAC	9.09	36.36	< 0.01	12.5	33.33	0.106
VKA	0	27.27	0.081	4.16	22.22	0.266
NOAC	18.18	45.45	0.1462	20.83	44.44	0.249
60-75 years	n = 16	n = 192		n = 82	n = 128	
OAC	6.25	34.89	0.0004696	24.39	37.5	0.0425
VKA	12.5	15.62	0.816	4.87	21.87	< 0.01
NOAC	0	54.16	< 0.01	43.90	53.12	0.18
≥ 76 years	n = 4	n = 181		n = 20	n = 159	
OAC	25	30.11	Insufficient	15	32.07	0.0087
VKA	0	6.62	Sample	0	7.54	0.0004
NOAC	50	53.59		30	56.60	0.026

OAC = oral anticoagulants, VKA = vitamin k antagonist, NOAC = new oral anticoagulants.

and 0.38%, Asians. In a meta-analysis of 30 RCTs with NOACS in the context of NVAF, venous thromboembolism and acute coronary syndromes, Jackson et al.[10] evaluated reports of race and ethnicity in 184,414 patients. They found that 75.2% of the patients were Caucasian, 14.3% Asian, 3.9% Hispanic and only 2.0% Black. On the other hand, in the ARISTOTLE[11] study in which apixaban versus warfarin was compared in 18,201 patients with NVAF, 65% of the patients were Whites with only 19% and 16% of Latin and Asian origin, respectively, without reporting Black patients. This lack of scientific statistical information in Hispanic and other race patients is important, due to the well-known racial differences in AF epidemiology and in the response to certain medications, including Warfarin, which requires higher doses of the drug in black patients than in whites, but lower doses in Asians, to maintain an INR between 2.0 and 3.0. The validity of extrapolating the conclusions of the RCTs to racial minorities has not been demonstrated.[12] Our group, interested in filling this epidemiological informative gap in Hispanics, obtained the AF prevalence in Mexican patients from the Northeast of the country, region with the highest presence of cardiovascular diseases in Mexico and documented a high-risk clinical profile for cardiovascular outcomes in our patients, with high percentages of patients with hypertension, type 2 diabetes, CAD, alcoholism, women and elders, meritorious of frequent pharmacological prescriptions of OACS, ARB, diuretics, MRA and nitrates. Regarding the prescriptions of antiarrhythmic drugs in our patients, it is relevant that these were directed more towards control of heart rate than to control of heart rhythm, with use of type III antiarrhythmics being observed in 38.57%, propafenone in 4.45%, BB in 50.44% and digoxin in 26.70%. The REMEFA study (The Mexican Registry of Atrial Fibrillation)[13] compared the rate-control strategy vs the rhythm control strategy in 1,201 patients, reporting better control of arrhythmia and lower incidence of cerebrovascular disease in the rhythm control group. If we analyze the rate-control group of that study, we can observe higher prescriptions than in our study of digoxin (69%), type III antiarrhythmics (59.0%) and BB (56.0%). However, since this was a prospective study and comparative with 1-year follow-up, between 2 intentional treatment strategies for AF, it clearly differs from our cross-sectional work, which evaluated only the daily practice of 3 cardiologists, without comparative purposes,

Table 4: Relationship of drug therapy, age group and category in the CHA_2DS_2-VASc and CHADS scales.

Age group	CHA_2DS_2-VASc						CHADS					
	< 60 years			60-75 years			< 60 years			60-75 years			≥ 76 years		
Drugs/Category	0-1 n = 22	≥ 2 n = 11	p	0-1 n = 8	≥ 2 n = 96	p	0-1 n = 24	≥ 2 n = 9	p	0-1 n = 41	≥ 2 n = 64	p	0-1 n = 20	≥ 2 n = 159	p
BB	45.45	63.64	0.342	62.5	54.17	0.672	45.83	66.67	0.305	43.9	60.93	0.046	40	47.79	0.519
Type 3 antiarrhythmic	31.82	54.55	0.24	37.5	56.25	0.352	29.17	66.67	0.071	60.97	51.56	0.174	35	29.55	0.641
Diuretics	13.64	72.73	0.0019	12.5	45.83	0.034	20.83	66.67	0.029	29.26	53.12	< 0.01	10	69.18	< 0.01
MRA	0	72.73	0.00042	0	30.21	< 0.01	8.33	66.67	0.007	12.19	37.5	< 0.01	5	25.78	0.0014
Antiplatelet	36.36	9.09	0.059	75	27.08	0.022	33.33	11.11	0.149	39.02	26.56	0.097	55	30.18	0.049
Propafenone	18.18	0	0.042	12.5	2.083	0.434	16.67	0	0.042	2.439	3.125	0.417	0	4.4	0.007
Digoxin	13.64	54.55	0.033	25	19.79	0.765	20.83	44.44	0.249	14.63	23.44	0.129	30	28.3	0.879
ACEI	4.55	0	0.3287	37.5	11.46	0.201	4.17	0	0.327	17.07	10.94	0.196	0	11.94	< 0.01
ARB	18.18	72.73	0.0039	12.5	57.29	0.0083	20.83	77.78	0.004	36.58	65.62	< 0.01	0	59.74	< 0.01
CCB	18.18	18.18	0.999	25	33.33	0.638	16.67	22.22	0.743	26.82	35.93	0.164	0	35.22	< 0.01
Nitrates	0	9.09	0.3409	12.5	19.79	0.593	0	11.11	0.346	14.63	21.87	0.173	15	20.75	0.5193

BB = beta blockers, MRA = mineralocorticoids receptor antagonist, ACEI = angiotensin converting enzyme inhibitor, ARB = angiotensin receptor blockers, CCB = calcium channel blockers.

only informative, possibly reflecting a practice closer to reality, such as it can be inferred by the significantly lower digoxin prescriptions used in our study. With this information, one would think that there is little probability of finding substantial differences in the prevalence and characteristics of AF between the Mexican Hispanic population and the white population of the United States. However, larger studies, of the type of population records in AF in Mexico, are required to confirm this presumption.

Globally, probably the only study with multiracial population was the XANTUS LE,[14] which prospectively assessed the safety of the use of rivaroxaban (bleeding and adverse events) and all-cause mortality, in 2,064 patients, reporting low incidence of bleeding and similar results in the incidence of ECVD. However, 75.8% of the patients included in this study were Caucasian and 13.7% had no race information. On the other hand, the short duration of the study, of one year, becomes insufficient time to demostrate substantive differences between multiracial populations.

Another underrepresented group in most clinical trials of AF are women. In our study, the percentage of women was higher than that of men, reaching 52.22% of the sample. This characteristic makes a difference with the majority of RCTs, main references of the cardiological practice guidelines in the anticoagulation of patients with NVAF, in which the inclusion of women has been suboptimal, with only 30% in studies with VKA and 35% with NOACS. Distributions similar to ours in terms of gender are observed in «real world» records of NVAF in the United States, which included up to 55% of women.[15,16] The women evaluated in our work even had a significantly greater numerical presence in relation to men in the highest risk categories, CHA_2DS_2-VASc ≥ 2, clinical behavior that is also common in other countries, as it has been documented in Chinese women in the recent AF Registry of China.[17] Considering these data and that cardiovascular diseases are the main cause of female mortality, causing 1 in 3 deaths,[18] RCTs would have to enter an equal proportion of men and women. Greater inclusion is necessary, since women with NVAF generally have worse symptoms, worse qual-

Table 5: Relationship between sex and location on embolic risk scales.

Embolic risk scale	Men (n = 156) n (%)	Women (n = 161) n (%)	p
CHA_2DS_2-VASc 0-1	22 (14.1)	8 (4.97)	0.0363
CHA_2DS_2-VASc ≥ 2	134 (85.9)	153 (95.03)	< 0.01
CHADS 0-1	51 (32.7)	34 (21.12)	0.9999
CHADS ≥ 2	105 (67.3)	127 (78.88)	0.2705

ity of life and a greater risk of cardiovascular disease and death than men, but at the same time they benefit from greater reductions in thromboembolic risk than men, when they receive OACS.[19,20]

Finally, given that one of the most important aspects of AF is preventive therapy with OACS for ECVD, it is worrying that there is a perception in the world that this drugs are actually being underused, underpinning this view of the reports of some European nations, with really high percentages of OACS prescription in NVAF. There are, however, certain considerations that cannot be omitted. In a study conducted by Volterrani et al. in Italy, although 88% of the patients categorized in CHA_2DS_2-VASc ≥ 2, received OAC, also in the category CHA_2DS_2-VASc = 0, 59% of the patients received them, in obvious indication «off-label».[21] In Belgium, in the GARFIELD-AF study, the use of OAC in 1,713 patients, was the highest in Europe, with 80.1% of patients receiving this therapy, increasing its use up to 84.3% in CHA_2DS_2-VASc ≥ 2 patients. However, also in 58.7% of the low-risk CHA_2DS_2-VASc 0-1 patients, OACS were used, without a clear indication.[22] In our study, the percentage of OACS prescription in NVAF was 59%, a percentage close to that obtained in the follow-up cohort 2-year prospective of 17,162 patients, from the GARFIELD-AF registry, which reported 60.8%.[23] However, in the present work the participating cardiologists were not inclined to prescrib OACS to patients with NVAF located in category 0-1 of the CHA_2DS_2-VASc scale, receiving them only 8.33%. These patients located in low-risk categories, had a higher prescription of AP therapy as well as antiarrhythmic drugs such as propafenone. In subjects < 60 years, OACS did not have a differential prescription among patients with greater or lesser cardiovascular risk, a situation that was totally different in older age groups, even in senile subjects ≥ 76 years, in wich a preferential use of OACS was evident in high cardiovascular risk groups. It is likely that this high OACS prescription in some European countries are due to a combination of factors. The first would be that in the records of the health system of the United States and Europe, the OACS were prescribed by doctors of different specialities, such a family doctors, internists, geriatricians, neurologists and cardiologists, unlike Mexico, where practically the only one specialist who initates and monitors OAC therapy in NVAF is the cardiologist. This medical practice would lead these countries to artificially «inflate» their OACS prescriptions, when administered in patients without indication, category 0-1 of the risk scales. Another potencial cause is that in some of the AF Registries, many patients were admitted to studies during hospitalizations of heart disease, so the attending physician would be more likely to initate on OAC.

Limitations of the study

The findings of our study portray the reality of AF in the Northeast region of Mexico and although their data are valid, its usefulness should be considered limited to residents of that region, because of the socioeconomic and health care differences between the different regions of the country.

CONCLUSIONS

The prevalence obtained from AF (3.74%) is similar to that of other western countries. Non-valvular AF, the predominant type of AF, was mainly associated with hypertensive and ischemic heart disease. In our patients, AF is characterized by having a clinical profile of high cardiovascular risk, senility and predominance of women, with a prevalence (3.74%) similar to that of other western countries. Greater inclusion in RCTs of patients with this profile, is necessary.

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The «de Winter» pattern as equivalent of acute ST segment elevation myocardial infarction

Patrón de «de Winter» como equivalente de infarto agudo de miocardio con elevación del ST

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Keywords:
The «de Winter» pattern, electrocardiogram, acute myocardial infarction, percutaneous coronary intervention, proximal left anterior descending artery, occlusion.

Palabras clave:
Patrón de «de Winter», electrocardiograma, infarto agudo de miocardio, intervención coronaria percutánea, segmento proximal de la arteria descendente anterior, oclusión.

ABSTRACT

We present a case of a young patient with anginal chest pain, electrocardiogram (ECG) showed «de Winter» pattern characteristics, which was first reported in 2008 by Winter et al, being so far, an atypical electrocardiographic presentation of ST elevation acute myocardial infarction (STEMI) little reported in the literature. The «de Winter» ECG pattern is characterized by high and symmetrical T waves along with J-point depression in all precordial leads, associated with proximal left anterior descending artery (LAD) occlusion. In this patient, the best treatment was emergency coronary angiography with primary percutaneous coronary intervention (PCI) placing a LAD drug-eluting stent, with successful result, improving patient's prognosis. Current guidelines do not take into account the «de Winter» ECG pattern as an equivalent of STEMI, however, literature reports an association with acute myocardial ischemia that endangers the patient's life; we consider necessary to include this ECG pattern in the STEMI guidelines, in order to allow doctors in charge of emergency services become familiar with this diagnosis and to be able to offer a timely and effective treatment to patients.

RESUMEN

Presentamos el caso de un paciente joven con dolor torácico anginoso, con electrocardiograma (ECG) que muestra características del patrón de «de Winter», reportado por primera vez en el 2008 por Winter y colaboradores. Hasta el momento es una presentación electrocardiográfica atípica de infarto agudo de miocardio con elevación del segmento ST (IAMCESST) poco reportada en la literatura. Las características de este patrón encontradas en el ECG son ondas T altas y simétricas, asociadas con depresión del punto J en todas las derivaciones precordiales; este tipo de patrón se asocia a oclusión del segmento proximal de la arteria descendente anterior (DA). El tratamiento correcto en este caso fue la realización de angioplastia coronaria percutánea con colocación de stent liberador de fármaco a la DA, con resultado exitoso, lo que mejoró la supervivencia del paciente. Las guías actuales no toman en cuenta este patrón electrocardiográfico como equivalente de IAMCESST; no obstante, los reportes de la literatura lo asocian a isquemia miocárdica aguda, que pone en peligro la vida del paciente; consideramos necesario incluir este patrón electrocardiográfico en las guías de IAMCESST y, de esta manera, permitir a los médicos a cargo de los servicios de urgencias familiarizarse con este diagnóstico y ser capaces de ofrecer un tratamiento oportuno y efectivo para los pacientes.

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INTRODUCTION

Despite the multiple programs implemented for detection and timely treatment of acute coronary syndromes, acute myocardial infarction (AMI) is the principal cause of death worldwide. Immediate coronary reperfusion therapy, through intravenous fibrinolysis or percutaneous coronary intervention, has allowed to reduce mortality and complications.[1] In addition to the characteristic electrocardiographic pattern in the STEMI, there are at least five patterns mentioned in the literature as equivalents of STEMI, potentially fatal without immediate diagnosis and treatment; within these patterns are described isolated posterior infarction, Wellens syndrome A and B, hyper-acute T waves, complete left bundle branch block, the sign of «shark fin» and the «de Winter» pattern.[2,3] In 2008, Winter et al described in 30 of 1532 (2.0%) young men patients with hypercholesterolemia and with

occlusion of the anterior descending artery, an electrocardiographic pattern that they called the «de Winter», characterized for 1) ascending depression of the ST segment > 1 mm at point J of the precordial leads (V1-V6), 2) high, prominent and symmetrical peak T waves, without classical variation of the ST segment in the precordial leads and, 3) slight ST segment elevation (0.5-1 mm) in the aVR lead.[4,5] The objective of this case is the early recognition of the «de Winter» pattern as equivalent of STEMI, allowing the timely treatment of acute coronary syndrome with atypical electrocardiographic characteristics.

CASE PRESENTATION

A 43-year-old obese male, with history of smoking, is presented to the emergency department because of suddenly thoracic oppressive pain, intensity 9/10 for more than 20 minutes, accompanied by nausea, palpitations and anxiety; the patient self-medicated with NSAIDs, with partial improvement. The physical examination revealed blood pressure of 130/80 mmHg, heart rate of 78 bpm, respiratory rate of 17 rpm, temperature 36.7 ºC and pulse oximetry saturation in 91%; a normal S1 and S2 cardiovascular examination, with no apparent murmurs. Laboratory tests reported TnI 0.025 µ/L (0.010-0.023). In the 12-lead electrocardiogram obtained at admission, sinus rhythm, significant depression of the ST segment (> 1 mm) at point J in leads V3-V6, and high and symmetrical T waves in leads V2-V6 were observed, with slight elevation of the ST segment in the aVR lead (0.05 mm) and moderate reciprocal depression of the ST segment in lower leads *(Figure 1)*. The «de Winter» electrocardiographic pattern was recognized and treated as equivalent to STEMI, coronary angiography was indicated. clopidogrel 600 mg, acetylsalicylic acid 300 mg and atorvastatin 80 mg were administered orally, as well as 5000 IU intravenous unfractionated heparin. Urgent coronary angiography was performed using right femoral access, which showed single-vessel disease with acute LAD occlusion *(Figure 2)*. PCI was performed with successful placement of a drug-eluting stent (everolimus) of 3.5 x 18 mm in LAD, restoring arterial blood flow *(Figure 3)*. In the follow-up studies, the post-procedure ECG showed an anteroseptal STEMI with Q waves in leads V1-V3, with extensive anterior subepicardial ischemia, lower leads and aVR returned to

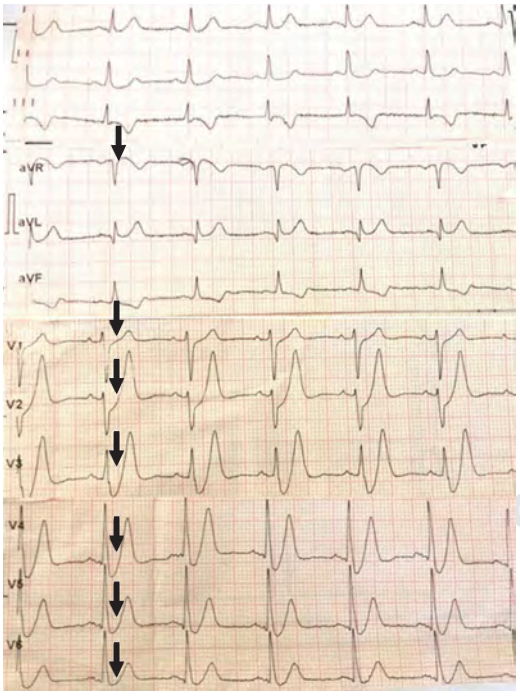

Figure 1: Initial ECG with the «de Winter» pattern.

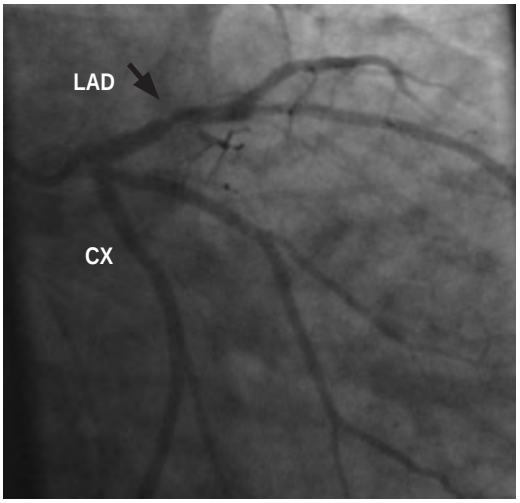

Figure 2: Diagnosis coronary angiography with subtotal occlusion of the proximal left anterior descending artery (LAD) (black arrow).

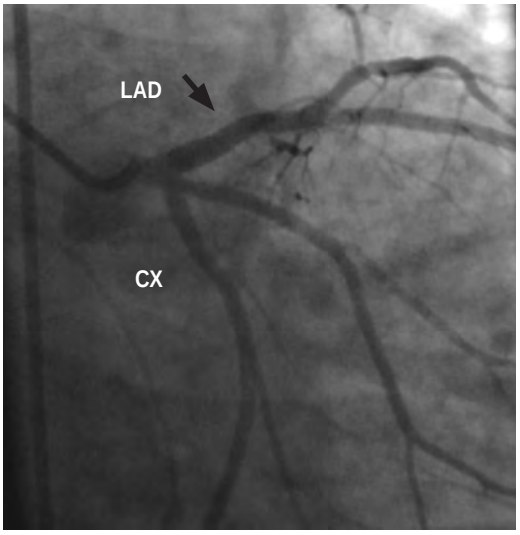

Figure 3: PCI with successful placement of a drug-eluting stent in LAD (black arrow).

normal *(Figure 4)*; patient remained stable in the coronary unit. Echocardiogram 24 hours after the procedure reported antero apical hypokinesia with 52% ejection fraction *(Figure 5)*. The patient evolved without complications and 72 hours later he was discharged at home with dual antiplatelet therapy, statin, beta blocker and angiotensin converting enzyme inhibitor.

DISCUSSION

The introduction of the electrocardiogram as a diagnostic tool in the medical area, has allowed the rapid identification of AMI and with it, timely treatment and better prognosis, however, up to 2% of patients with STEMI can present an atypical ECG pattern, which in inexperienced hands could go unnoticed, delaying the proper treatment established by guides.[6] The «de Winter» pattern is the atypical electrocardiographic expression of a LAD occlusion, which for reasons that to date are not clear, is not expressed with ST elevation as would be expected, so the condition can be confused with an AMI without ST elevation and thereby, delay the urgent treatment required, either intravenous thrombolysis (in the case of hospital centers that do not have a hemodynamic room) or PCI.[7,8]

The delay in the optimal management of patients with STEMI with typical or atypical presentation on the ECG, brings with it an increased risk of complications around the so-called major adverse cardiovascular events (MACE), however, until now there is no section in the current clinical guidelines of acute coronary syndromes that describe and indicate the management of these patients with the urgency that is required.[9]

The knowledge of the «de Winter» pattern will allow the first contact physician in the emergency department to familiarize themselves with this finding, identifying the cases and avoiding overlooking ECG particularities, that may have an impact on the treatment and prognosis of patients with STEMI.[10]

CONCLUSIONS

In addition to the typical electrocardiographic pattern of ST segment elevation in acute myocardial infarction, there are other electrocardiographic patterns reported in the literature as equivalents of STEMI, such as the «de Winter» pattern, the recognition of these patterns is very important to provide early revascularization therapy.

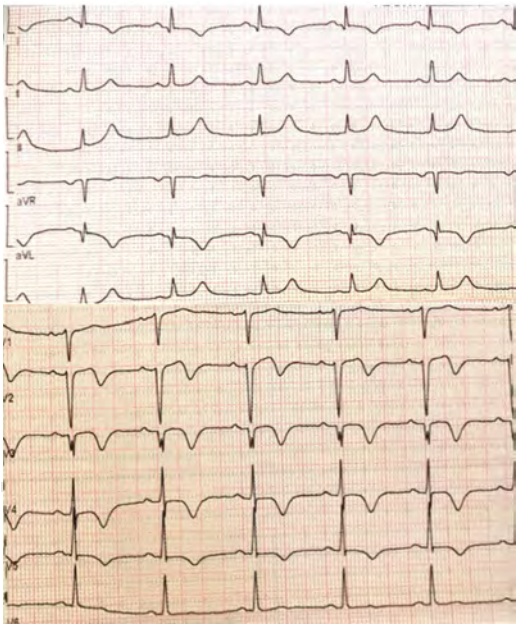

Figure 4: Post-procedure ECG with normalization ST segment and extensive anterior subepicardial ischemia.

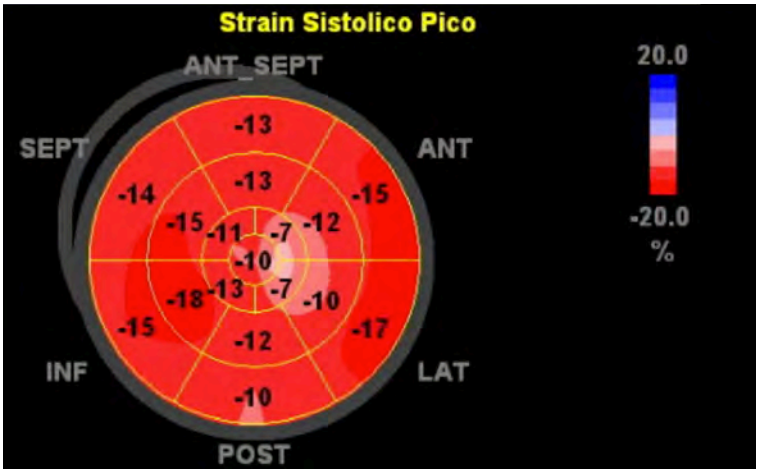

Figure 5: 24 hours post-procedure echocardiogram reported antero apical hypokinesia.

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Wellens' syndrome: report and review of a case

Síndrome de Wellens. Reporte y revisión de un caso

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Keywords:
Wellens syndrome, severe stenosis, percutaneous coronary intervention, stent, acute myocardial infarction.

Palabras clave:
Síndrome de Wellens, estenosis severa, intervención coronaria percutánea, stent, infarto agudo al miocardio.

ABSTRACT

Wellens' syndrome is a clinical entity characterized by an important occlusion of the left anterior descending artery (10-15% of incidence) in more than the 80% of the cases in the proximal segment, that is diagnosed with an electrocardiogram with the presence of precordial T wave inversion (V1-V5) in the 76% of the cases (type B) and in the 24% of the cases in V2-V3 leads (type 1). We present a patient evaluated in the emergency department for intermittent chest pain and T-wave inversion of the Wellens syndrome type B pattern, who was treated with percutaneous coronary intervention where a critical lesion was demonstrated in the medial anterior descending. This case shows how timely diagnosis prevents progression to extensive anterior acute myocardial infarction.

RESUMEN

El síndrome de Wellens es una entidad clínica caracterizada por una oclusión importante de la arteria descendente anterior izquierda (10-15% de incidencia) en más de 80% de los casos en el segmento proximal, que se diagnostica con electrocardiograma por la presencia de inversión de onda T precordial (V1-V5) en 76% de los casos (tipo B) y en 24% de los casos en derivaciones V2-V3 (tipo 1). Presentamos a un paciente evaluado en el Servicio de Urgencias por dolor torácico intermitente e inversión de la onda T del patrón del síndrome de Wellens tipo B, que fue tratado con intervención coronaria percutánea, en la que se demostró una lesión crítica en la descendente anterior media. Este caso muestra cómo el diagnóstico oportuno previene la progresión a infarto de miocardio agudo anterior extenso.

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INTRODUCTION

Chest pain is a common reason for seeking care in emergency departments. Cardiovascular disease can be present in up to 20%, but only 5.5% represents a life-threatening condition. The low to moderate risk stratification could lead to an early discharge, it has been shown that from 2% to 13% of patients discharged from the emergency services and who came for chest pain presented undiagnosed acute myocardial infarction.[1] The interpretation of the ECG usually follows the evaluation of patients with suspected myocardial ischemia after medical history and physical examination, so it is vitally important to recognize any pattern that suggests myocardial ischemia taking into account that 10 to 25% of patients with acute coronary syndrome requiring urgent reperfusion therapy, may have atypical ECG patterns.[2,3]

Wellens Syndrome (WS) is a preinfarction stage, characterized by an electrocardiographic pattern of changes in the T wave associated with severe stenosis of the anterior descending artery.[4] It is currently not considered an equivalent pattern of acute coronary syndrome,[5] despite that without timely diagnosis and intervention, extensive acute myocardial infarction of the anterior wall can occur in an average time of 8.5 days[6] and possibly sudden death, so they must undergo percutaneous coronary intervention. We present a clinical case from the Hemodynamics Service of our institution, in order to analyze the characteristics of Wellens syndrome, emphasizing timely and differential diagnosis.

CASE REPORT

A 62-year-old male, without a family history of cardiovascular disease. With a personal pathological history of positive smoking from 42 years of age with an 8 pack/year smoking history, type 2 diabetes mellitus and systemic

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hypertension of two years of diagnosis without adequate control and chronic kidney disease KDIGO 5, recently diagnosed, without renal function replacement treatment.

He was admitted to a second-level hospital with 6-hour history of sudden and intermittent oppressive chest pain at rest intensity 7/10, with irradiation to the left thoracic limb lasting two minutes, associated with dyspnea, improvement with nitrate intake. Braunwald class IIIA unstable angina was diagnosed and was referred to our hospital for evaluation.

At admission, the patient was asymptomatic with vital signs of blood pressure 150/80 mmHg, SO_2 94%, temperature 36 °C, heart rate 83 beats per minute. Neck without jugular venous distention, precordium without abnormal heart sounds, lung fields with vesicular murmur, painless abdomen, edema-free extremities with symmetrical pulses.

Initial electrocardiogram showed sinus rhythm, heart rate 75 beats per minute, P wave 80 ms, PR interval 190 ms, QRS complex 80 ms, electrical axis + 30°, no Q waves, symmetric and deep T wave inversion (up to 1 mV) from V2 to V6, and shallow (up to 0.2 mV) in I, II, aVL *(Figure 1)*.

The cardiac biomarkers were increased: troponin I 1.54 ng (0-0.40 ng/mL) and CK-MB 53 mg/dL (0-24 mg/dL). The enzymatic curve did not show any increase, while the electrocardiogram recorded during a period of pain revealed pseudo normalization of the T wave *(Figure 2)*. The patient was pharmacologically treated with atorvastatin, aspirin, clopidogrel and enoxaparin from admission.

The typical angina, the result of the highly sensitive troponin, as well as the high-risk electrocardiographic pattern are nosological characteristics of Wellens syndrome type B. Therefore, it was decided to submit the patient to coronary angiography that revealed a critical lesion in the medial portion of the anterior descending with distal TIMI 3 flow that required the placement of 2 drug-eluting stents. Angiography also showed a 90% lesion in the marginal obtuse and no significant disease in the right coronary artery *(Figure 3)*.

The patient was discharged stable and improved six days later, with a pharmacological indication of double antiplatelet therapy for 12 months. At 6 months follow-up in the outpatient clinic, he was clinically asymptomatic.

DISCUSSION

There are many elements described in the universal literature as case report for Wellens syndrome. Wellens syndrome is a clinical entity, didactic due to its acute symptoms and documented by means of electrocardiograms, but there is important evidence of ischemia in resting nuclear medicine, given that in many cases it is persistent angina.[7]

We present a male patient with multiple cardiovascular risk factors and chest pain; with a first diagnosis to be ruled out: acute coronary syndrome with or without ST segment

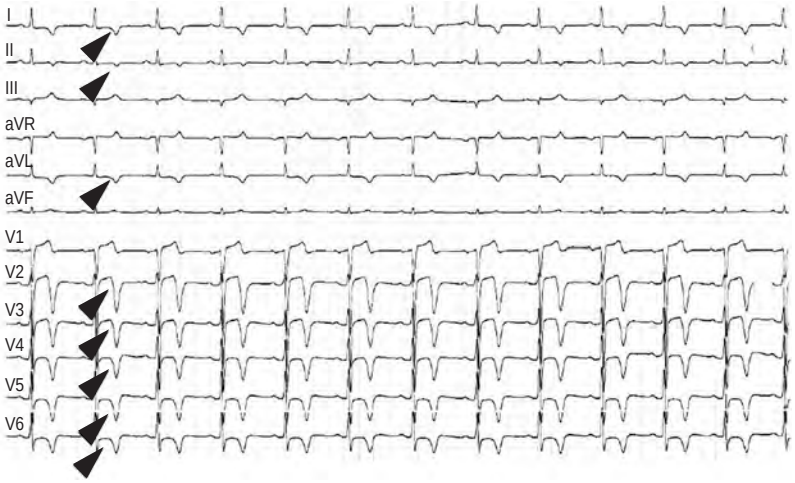

Figure 1:
12-lead electrocardiogram, shows symmetrical and deep T-wave inversion (up to 1 mV) from V2 to V6 and lower depth (up to 0.2 mV) in I, II, aVL.

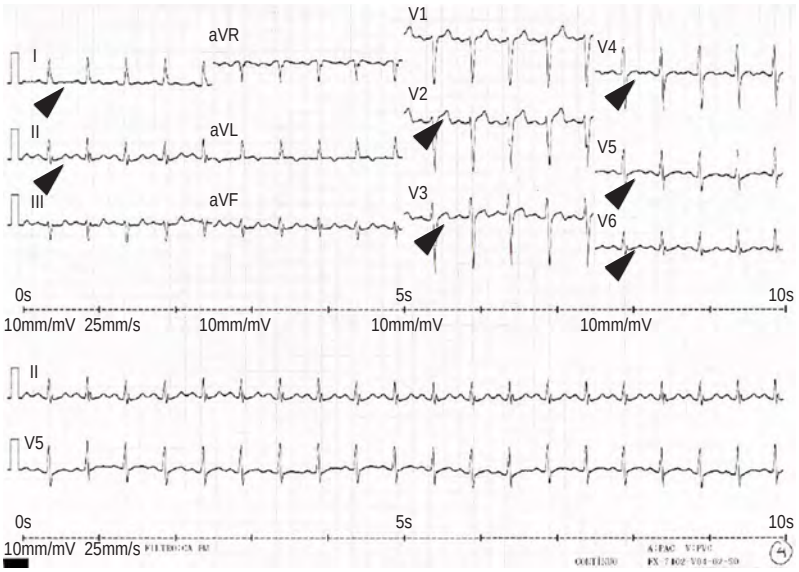

Figure 2:

Pseudo-normalization of T-waves.

elevation, being essential to establish risk, the measurement of highly sensitive troponins and the electrocardiogram. The latter was taken in a period without pain and showed a typical pattern of Wellens syndrome type B,[8] which allowed the patient to be defined as high risk,[9] conditioning an early invasive strategy[4] without performing other types of ischemia-inducing tests, reducing the morbidity and mortality.[10,11] The patient presented a critical obstruction in the middle portion of the anterior descending artery, although it has been controversial whether these patients should really be diagnosed as Wellens syndrome,[12,13] since the original study refers to the proximal portion of the descending anterior[4,9] we are convinced (based on multiple studies and case reports in the medical literature) that this may be a variant or subgroup of Wellens syndrome, but more studies and more debate on the subject will be required.

Wellens syndrome or anterior descending artery syndrome, in an electrocardiographic pattern with changes in the precordial T waves, was described by Zwaan and Wellens in 1982[4] in 14% to 18% of admitted patients with unstable angina and associated with critical stenosis of the proximal anterior descending. Kobayashi and collaborators, reported in 2019 a retrospective analysis of 274 patients with non-ST-segment elevation myocardial infarction, presenting Wellens pattern 24 (8.8%) of the patients, of these, 16 had a culprit lesion of the anterior descending (8 proximal, 8 mid), 2 patients had a culprit lesion not of the anterior descending artery and 6 patients had non-obstructive coronary artery disease.[14] Therefore, despite the association that was initially described, the same electrocardiographic pattern may be in critical obstructions of the mid anterior descending artery, also in normal coronary arteries with spasm induced by substances such as cocaine called pseudo Wellens syndrome,[15,16] coronary angiography being the method of choice to confirm this latter diagnosis, since precordial pain as well as elevation of cardiac enzymes is also present in cocaine users.

The incidence of WS is 10-15% of all acute coronary syndromes[17] with an etiology similar to any other condition that causes coronary heart disease: atherosclerotic plaque, coronary arterial vasospasm, hypoxia, and increased cardiac demand. Its risk factors include a family history of premature coronary heart disease, type II diabetes mellitus, metabolic syndrome, hypertension, smoking, hyperlipidemia, work stress, advanced age[18] (55 ± 9 years), cases associated with HIV, probably due to premature coronary disease[19] and also in patients without cardiovascular risk factors.[20]

It is classified into type A and B (1 and 2 respectively).[8] Type A constitutes 24% of cases,

it is less common but more specific and less recognized, it presents biphasic T waves in V2 and V3. Type B is the most common and least specific,[9] represents 76% of cases and is characterized by symmetrical and deep inverted T waves in V1 to V4.[17] Schears MR and collaborators, describe the evolution of these patterns starting with biphasic T waves (pattern A) that are deeply and symmetrically reversed (pattern B). Then it extends to V4, then to V5 and finally to V6, not documented in all cases by lack of serial electrocardiograms,[21] without defining the time in which one pattern can change to another. Atypical cases have been described where the type B pattern changes to type A.[22] It should be emphasized that changes in the T wave occur during periods without precordial pain and pseudo normalized during the precordial pain episode, and may persist for hours or weeks[7,23] leading to errors in diagnosis and inadequate risk stratification, so the ability to recognize these patterns is extremely important, since ECG changes can be subtle[8-24] There are atypical presentations in which there is no precordial pain, only syncope, but the electrocardiographic pattern meets WS criteria.[25] In the case presented, the fact that the patient was asymptomatic at the time of admission to the emergency department, and the taking of an initial electrocardiogram as part of the cardiology evaluation, showed the inversion of T waves in precordial leads from V2 to V6, leading to timely diagnosis confirming it with pseudonormalization during an episode of precordial pain, which occurred spontaneously without maneuvers to cause ischemia.

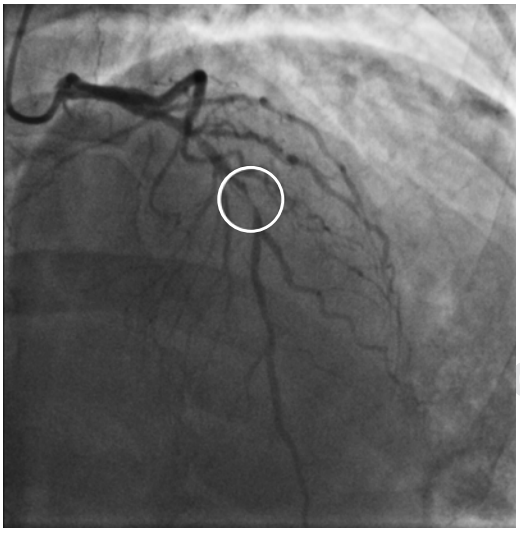

Figure 3: Coronary angiography showing critical lesion in the middle portion of the anterior descending.

The pathophysiology of ECG changes is unclear, local edema or lightheadedness due to intermittent or destabilized blood flow in the anterior descending coronary artery with critical obstruction has been proposed.[26] These changes have been demonstrated through cardiac magnetic resonance imaging.[27]

Although the patient's physical examination was normal, a protodiastolic murmur has been described in the third intercostal space above the left clavicular midline, probably caused by turbulence of blood flow immediately distal to the stenosed segment, known as Dock's murmur.[28]

The criteria for Wellens syndrome were described by Rhinehardt and collaborators, as follows:[8] deeply inverted T waves in leads V2 and V3 (can also be seen in leads V1, V4, V5 and V6) or biphasic T waves (with initial positivity and terminal negativity) in V2 and V3, recent history of precordial pain, absence of Q waves without loss of R waves in precordials, without significant ST segment elevation (< 1 mm), normal or minimally elevated cardiac markers, and changes in the T wave (biphasic or inverted) in the precordial leads in a pain-free state. The positive predictive value of WS to detect significant proximal anterior descending coronary artery stenosis is 86%, with a sensitivity of 69% and specificity of 89%.[2,29]

Differential diagnosis of precordial T-wave inversion includes pulmonary embolism,[30] cerebral hemorrhage, left ventricular hypertrophy, cocaine or morphine-induced coronary vasospasm, chronic thromboembolic pulmonary hypertension, interruption of transient left bundle branch block, or Wolff-Parkinson-White pattern, persistent juvenile T wave pattern, late stages of pericarditis, digitalis effect and Takotsubo cardiomyopathy.[31-33] Under a suitable clinical context, each one can be ruled out.

It is possible to confirm the diagnosis with coronary angiotomography, in hemodynamically stable patients[7,13] or with doubt in the initial approach since it is the most accurate

and reliable non-invasive modality to rule out significant coronary artery stenosis. It also helps to rule out conditions that cause the syndrome of pseudo Wellens such as pulmonary embolism, cannabis and phencyclidine poisoning, use of crack and vasospastic angina,[34] avoiding unwarranted cardiac catheterization.

Stress ischemia was not performed in this patient using a stress test since it is not indicated because it increases the demand for myocardial oxygen, producing adverse effects such as ST elevation and/or ventricular tachycardia with fatal results.[10,11]

Treatment with double antiplatelet (acetylsalicylic acid and clopidogrel), thrombolysis, control of blood pressure and glucose, as well as statin therapy alone do not decrease morbidity and mortality (left ventricular dysfunction, anterior myocardial infarction or sudden death),[8,35] 75% of patients progress to myocardial infarction only with medical treatment.[4] Therefore, immediate coronary intervention is highly recommended as a definitive therapeutic strategy.

CONCLUSIONS

The clinical presentation is the cornerstone for the suspected diagnosis in every chest pain to guide the correct diagnosis (typical and atypical angina). Wellens syndrome is characterized for electrocardiographic changes with right precordial T wave inversion V1-V5, with an occlusion of the proximal segment of the left anterior descending artery in the majority of cases, but in some cases the medial or distal segment of the left anterior descending coronary artery is occluded. Cardiac biomarkers are normal or may be slightly increased. The ischemic changes can be documented by myocardial perfusion gammagraphy or coronary angiography by computed tomography.

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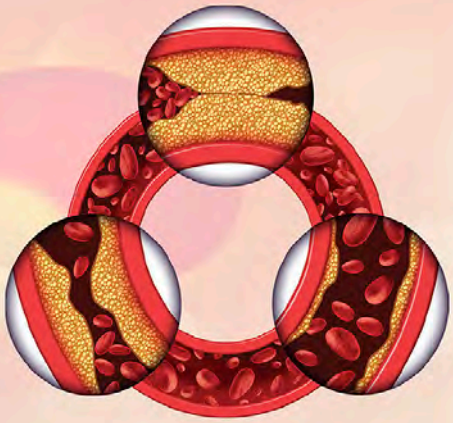

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