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Prevalence of Coronary Artery Disease in Patients with Peripheral Arterial Disease at the Hospital de Especialidades CMN La Raza"

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ABSTRACT

Introduction: 60-80% of patients with peripheral arterial disease (PAD) of the lower extremities present with coronary artery disease (CAD); there are no reports in Mexico of said relationship. PAD is undervalued; also secondary to this, patients do not wander, causing patients with CAD to go undetected; In Mexican population, a high percentage of coronary arteries calcification has been identified, which altogether with predominant cardiovascular risk factors, makes it a priority to identify this association.

Objective: To establish the prevalence of CAD in patients with PAD.

Methods: An ambispective, descriptive, observational study was conducted to determine the prevalence of CAD diagnosed by Cardiology in patients with PAD, as well as associated comorbidities; diagnosing patients with PAD using the Ankle-Brachial index (ABI) ≤ 0.70. The results were analyzed with the statistical program SPSS-27.0.

Results: We collected 120 patients with PAD, with a predominance on females (72%); of these, 40/120 (33.3%) met the selection criteria; with an average age of 67 ± 9 years and a predominance of the male sex (75%); overweight 41%; diabetes and systemic arterial hypertension 65%; 37.5% (15/40) had severe coronary artery disease.

Conclusion: Even with the advances in molecular and histochemical medicine, it hasn't been enough to reduce morbidity in cardiovascular diseases, so early detection of CAD is a priority.

Keywords: Coronary Artery Disease; Peripheral Arterial Disease; Prevalence; Cardiovascular Disease; Acute Coronary Syndrome

Abbreviations: PAD: Peripheral Arterial Disease; CAD: Coronary Artery Disease; ABI: Ankle-Brachial Index; AMI: Acute Myocardial Infraction; CAC: Coronary Artery Calcification; SMC: Smooth Muscle Cells

Introduction

Coronary artery disease (CAD) is mainly due to atherosclerosis; Atherosclerosis is a chronic and progressive process characterized by lipid and fibrous elements deposits in artery walls. It is the leading cause of morbidity and mortality in developed countries and is expected to be so in emerging countries. It manifests as a polyvascular disease (Figure 1) that affects more than one arterial territory, constituting an independent risk factor for mortality. It manifests itself more often in lower extremities (LE), so when we refer to peripheral arterial disease (PAD), we basically refer to this location. Location in LE is considered an independent risk marker for CAD [1]. CAD is common; some reports refer 40 to 60%, including up to 80% of patients with PAD; of these, 30% have severe CAD [2]. (Defined as one that affects three or more trunk coronary vessels or the left coronary artery.) The leading cause of death in patients with PAD is ischemic heart disease; 20% of patients with PAD are asymptomatic. Variations in Ancle brachial index (ABI) have been correlated with the severity and extent of CAD, as well as with the carotid mean intimate index (MIC); for every 0.1 decrease in ABI increases the risk of a major vascular event by 10% [3]. CAD etiology is considered multifactorial as in PAD, and it is associated with atherosclerosis, smoking, type 2 diabetes mellitus (DM2), systemic arterial hypertension (SAH) and dyslipidemia. The risk factors for PAD and CAD are similar, although their relative importance differs.

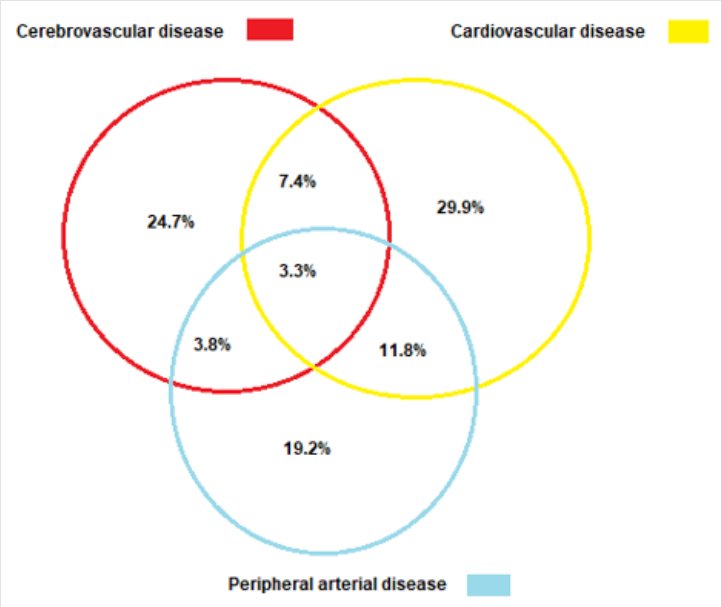

Figure 1: Polyvascular involvement; taken from: Martinez-Blanco Delio Felipe (2019) Grade [1] thesis.

Dyslipidemia is clearly associated with the development and progression of atherosclerosis; in particular, high serum total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) are considered important factors in the onset and progression of PAD and CAD, with lipid-lowering medical therapy being the cornerstone of treatment [4]. In addition to the predominant comorbidities in our population, coronary arterial calcification (CAC) has been identified in 27% by computed tomography in our population with higher prevalence and magnitude in males with 40% [4]. As already mentioned, a considerable percentage of patients with PAD, have CAD; this is of utmost importance, since a high percentage of these patients, is underdiagnosed. Fortunately, we have a simple tool such as the Ankle-Brachial Index (ABI), which is much more sensitive when the degree of stenosis is higher and predictive of adverse events: the lower the ABI, the greater the risk of cardiovascular events[4,5].The mortality rate in patients with PAD averages 2% per year; for non-fatal acute myocardial infarction (AMI), stroke or vascular death is 5-7% per year [6]. ABI is a simple, inexpensive, non-invasive marker for PAD; a low ABI (≤0.9) is associated with a two-fold increased risk of CAD (AMI or angina) in general population (normal ABI >0.9-1.3). According to international literature, the prevalence of low ABI in patients with auricular fibrillation is about 20%; the presence of asymptomatic PAD in this population was associated with a high risk of vascular events (defined as vascular death, fatal/nonfatal myocardial infarction, transient ischemic attack (TIA), fatal/nonfatal stroke) [7].

Coronary atherosclerosis is usually maintained for many years as a silent disease that does not cause any limitation or symptomatology during its development. When atherosclerotic lesions progress, they

can narrow the vascular lumen, erode or rupture abruptly, causing the formation of a thrombus or clot that obstructs the arterial lumen, leaving a part of the heart muscle without irrigation [8]. According to various anatomopathological studies carried out in patients who died after an acute coronary syndrome (ACS), it is known that, although the atheromatous plaques responsible for acute ischemic events usually have a heterogeneous architecture and cellular composition, there are certain well-defined characteristics that differentiate these lesions from those found in patients with stable coronary heart disease (CHD). Thus, vulnerable plaques (plaques with thin fibrous layer, broad lipid nucleus and high inflammatory cells content) with significant inflammatory activity can erode and induce thrombosis or rapid progression of coronary lesion, causing an acute coronary event. Therefore, the best treatment is prevention of the disease, acting early on cardiovascular risk factors, in order to avoid or delay as much as possible the development of lesions [8]. Coronary atherosclerosis is the most common form of cardiovascular disease. Its clinical manifestations, such as AMI, angina pectoris and sudden death, constitute the leading cause of mortality in the adult population in developed countries.

Atherosclerotic lesions will progress or not depending on cardiovascular risk factors, genetics, environmental and biomechanical factors, causing various clinical manifestations of ischemic heart disease or CHD [8]. Smoking is of utmost importance, especially in our country, because tobacco occupies the fourth place in addictive substances consumed, and as evidenced in the Framingham study, a consumption around ten cigarettes a day, increases by 20% the risk of suffering from CHD in men and 23% in women. Similarly, for every 10 mg/dl increased in blood cholesterol levels, cardiovascular mortality increases by 9%; elevated blood pressure levels above 160/95 mmHg double or triple the risk of CHD, just as DM2 increases the risk of cardiovascular events in the same proportion. Genetic predisposition and advanced age are also mentioned [8]. It is also important to mention that PAD represents a risk factor in patients undergoing percutaneous coronary intervention procedures, since these patients have a lower success rate of the procedure and a higher rate of cardiovascular complications, including greater blood loss with the possibility of requiring transfusion, or even the need for surgical repair at the puncture local level, with longer surgical time and greater anesthetic risk [9].

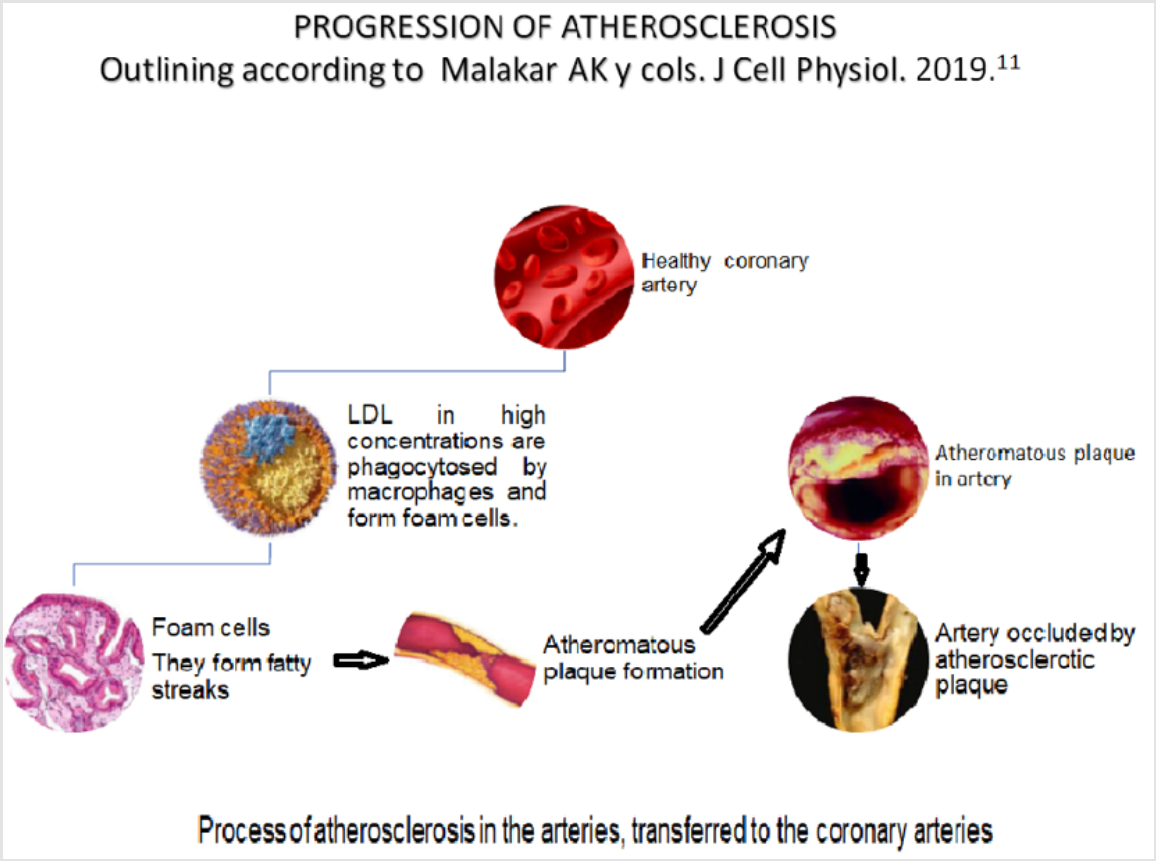

Figure 2.

Etiology and Risk Factors

CAD occurs due to atherosclerosis that conditions stenotic or occlusive lesions of the arteries. When the endothelial function of the arterial wall is disturbed, atherosclerosis is initiated due to the accumulation of lipoprotein droplets in the intimate layer of the coronary vessels. High concentrations of LDL can permeate the altered endothelium and undergo oxidation. This oxidized or modified LDL (endocytosis) attracts leukocytes to the intimate layer of the coronary vessels, which can be phagocytosed by macrophages, leading to the formation of foam cells. These cells replicate and form lesions called fatty streaks (streaks of fats), which are the earliest form of atherosclerosis. The formation of such lesions triggers signals that attract smooth muscle cells (SMC) to the fatty stria location, initiating the proliferation and production of extracellular matrix, mainly compound by collagen and proteoglycans. Atherosclerotic plaque begins to develop, accumulating a large volume of extracellular matrix produced by SMC, leading to progression of the lesion to fibrous plaque (Figure 2). The fibrous plaque invades coronary vessel lumen and form small blood vessels which can subsequently calcify the plaques. The final lesion formed is an advanced and complicated lesion consisting of a fibrous covering with a lipid-rich core with necrotic, highly thrombogenic material [10]. Proteoglycans produced by SMC can bind to lipoproteins and prolong their existence in the intimate layer, making them susceptible to oxidative modifications and non-enzymatic conjugation with sugars (glycation).

Modified lipoproteins, i.e., oxidized phospholipids and advanced glycation end products can prolong the inflammatory response [10]. In the diagram below, we exemplify the evolution of atheromatous plaque in an artery transpolated to the coronary arteries as described by Malakar, et al. [10]. Matrix metalloproteinases (MMPs) are secreted by endothelial cells in response to oxidation, hemodynamic, inflammatory, and autoimmune signals. These MMPs, are in balance with endogenous tissue inhibitors and modulate various functions of vascular cells, such as activation, migration, proliferation, cell death, geometric remodeling, formation of new vessels, arterial and myocardial extracellular matrix destruction, and finally, healing. Programmed cell death or apoptosis commonly occurs in atherosclerotic lesions, which eventually leads to tissue factors degradation into the form of particles [11]. Stary, et al. in 2000 classified atherosclerotic lesions (adopted and revised by the American Heart Association) into eight types of lesion; following this line of classification, Dalager, et al. in 2007, compared atheromatous plaques present in coronary, carotid and femoral arteries; They found that femoral arteries had less presence of atheromatous plaques (20%); Herisson, et al. in 2011, compared carotid and femoral endarterectomy specimens, and found that femoral artery plaques contains less calcium, less cholesterol, and it is less inflamed.

These differences could have an explanation from the hemodynamic point of view given the characteristics of the flow in different anatomical territories, conditioning that the plaque has different properties and behaves differently depending on its environment and location [12]. In addition to the risk factors mentioned above for CAD, we should mention PAD as part of an important risk factor (ABI ≤ 0.9) [4,13].

Clinical Presentation

CAD may present as angina; An estimated 10 million people in the United States have angina; more than 500,000 new cases are diagnosed each year. The main cause of angina is an imbalance between the oxygen heart supply and demand, usually resulting from an atherosclerotic plaque narrowing or occluding the vessel [9,14]. Regarding PAD, its prevalence directly proportional increases significantly with age and it is to be expected that it will increase more and more due to population longevity. Lower extremities PAD is diagnosed when ABI is ≤ 0.90, as we had already mentioned (ABI results from dividing ankle systolic blood pressure by arm systolic blood pressure); in 2010, an estimated 202 million people worldwide had an ABI ≤ 0.90; Of these, almost 70% live in low and middle-income countries [13]. Most PAD carriers are asymptomatic. Claudication (fatigue, burning or pain in leg muscles), reproduces when exerting or walking and is relieved by rest. The pain location guides us to the site of the disease. Claudication is often difficult to identify to such an extent that there are several questionnaires to interrogate it, such as the Edinburgh questionnaire; there are several differential diagnoses in patients with exercise related LE pain. Critical limb ischemia is often resistant to opioid analgesia and can be difficult to distinguish from neuropathy. The patient refers to hanging the leg over the edge of the bed to relieve pain [15]. In a series of patients with PAD who underwent intracoronary vascular ultrasound examinations, more severe and rapidly progressive coronary atherosclerosis was demonstrated [4,6] than in patients with CAD without PAD.

Patients with CAD and PAD are at increased risk of future cardiovascular events, as PAD, regardless of type, is a strong predictor of poor outcomes in patients with CAD. The presence of PAD is associated with increased coronary atherosclerotic progression; The greater the number of PAD affected sites, the greater the risk of death in patients with CAD. For all associations, the identification of PAD as a prognostic marker in CAD risk stratification is justified [1,15,16].

Diagnostic Evaluation

A proper history and complete physical examination are essential in the diagnosis of PAD. However, several conditions associated with leg symptoms, such as a narrow spinal canal, osteoarthritis, chronic venous insufficiency and neuropathy, may be present and mask PAD and therefore not evidence a probable coronary problem.

Risk of Cardiovascular Events in PAD

PAD is associated with a high risk of CAD or CVD. The rates of myocardial infarction, ischemic stroke and vascular death in patients with LE PAD without critical ischemia are between 5-7% per year, being the most common causes of death CAD in 40-60% and stroke with 10%-20%. In patients with critical limb ischemia, the risk of death is 25% at 1 year [9]. In the diagnosis of PAD, ABI leisurely is the initial test recommended by the American Heart Association/ American College of Cardiology (AHA/ACC) Guideline [9,15,16].

Treatment

Mainly is to modify lifestyle and risk factors to reduce cardiovascular events and amputations in patients with PAD, being the principal approach to quit smoking and methodical walking [9,15-17]. Statins reduce cardiovascular risk; simvastatin was associated with reduced mortality from peripheral cardiovascular and vascular events. Other studies report that statin therapy reduces the progression of claudication, critical ischemia and the need for revascularization or amputation (Level of evidence I in the AHA/ ACC guideline) proprotein convertase subtilisin/kexin type 9 (PCSK9, an enzyme involved in the degradation of LDL receptors in the hepatocyte) bind blockers which act in LDL receptors, demonstrated the effectiveness of lipid-lowering therapy in patients with PAD [16]. On the other hand, the decrease in BP in patients with PAD with the use of ramipril, an inhibitor of the enzyme converting an of angiotensin (ACE-I), as reported in the Heart Outcomes Prevention Evaluation (HOPE) trial, was associated with a significant reduction in the presentation of AMI, stroke or cardiovascular death, independent of symptoms and ABI values. A retrospective analysis of data from the University of California at Davis PAD Registry showed that the use of ACE-I receptor blockers was associated with a significantly lower risk of cardiovascular, cerebrovascular, and mortality events in patients with critical limb ischemia [16,17].

Antiplatelet therapy as evidenced in CAPRIE study, which evaluated clopidogrel versus aspirin, a dosage of clopidogrel 75 mg/day, was associated with a 24% relative risk reduction in the combined risk of AMI, ischemic stroke, or vascular death compared with aspirin 325 mg a day dosage. In other clinical trials, patients with documented prior AMI, ischemic stroke, or symptomatic PAD had a significantly lower rate of cardiovascular death, with low-dose dual antiplatelet therapy [16,17]. The COMPASS (Anticoagulant and Aspirin in Stable Cardiovascular Disease) study demonstrated a major advance in atherosclerotic cardiovascular disease treatment [15-17]. CAD is present in patients with PAD in a high percentage (60 to 80%) and of these patients, 30% have severe coronary disease, however there are no reports in the national literature of the prevalence of this relationship. Therefore we set as objectives, to investigate, the prevalence of coronary heart disease in patients with PAD in C. M. N. La Raza patients in a study period from April 2022 to March 2023 being the main risk factor for coronary heart disease, taking as a reference that in our medical unit, LE PAD has a prevalence of 33%; being a high percentage of PAD underdiagnosed and therefore, patients with coronaropathy underdiagnosed.

In addition, considering that the Organization for Economic Cooperation and Development (OECD) issued a statement that our country ranks first in cardiovascular lethality (with a percentage greater than 30% against an average of 7.9% worldwide), in patients over 45 years of age, and since our medical unit has a captive population of more than 12 million entitled. Since Mexicans suffer from predisposing factors for cardiovascular events such as DM2, SAH, obesity, coronary calcification [4,15,18]. Our research is justified by the high socioeconomic impact of cardiovascular morbidity and mortality [15,19].

Materials and Methods

In order to collect enough cases, an ambispective, descriptive, observational and analytical study was conducted to determine the prevalence of CAD in patients with PAD, as well as the associated comorbidities, in the Angiology and Vascular Surgery service of the "Dr. Antonio Fraga Mouret" hospital from April 2022 to March 2023; patients with PAD were identified by ABI ≤ 0.70. Beneficiaries were included; adults over 18 years of age of both sexes; with diagnosis of PAD and CAD diagnosed by the Cardiology service during admission, by echocardiogram and/or angiotomography. We did not include patients with comorbidities that would be confusing the origin or reason for the study, those with a high probability of adverse events when exposed to diagnostic methods, and patients whose hemodynamic status did not allow the staging of PAD. And we limit patients with poor diagnostic technique or death. The study variables were: Demographic and Anthropometric.

We carry out observational research and therefore without risk, attached to international and national regulations [20-22]. Statistical analysis was performed using SPSS-27 software for data description and analysis, using number and percentage for qualitative variables; minimum, maximum and average for quantitative; Descriptive statistics to determine measures of frequency and central, median, mode tendency of the population studied obtaining prevalence of both entities.

Results

From a total of 120 patients with PAD, we selected 40 (33.3%) who met the inclusion criteria; The average age was 67 ± 9 years; minimum 42, maximum 87. Females were predominant in patients with PAD 86 (72%), and males predominated 3:1 in CAD (Graph 1); normal body weight in 71 patients (59.2%); In 61 patients (50.8%), the most affected limb was the right limb (Table 1). Patients with CAD

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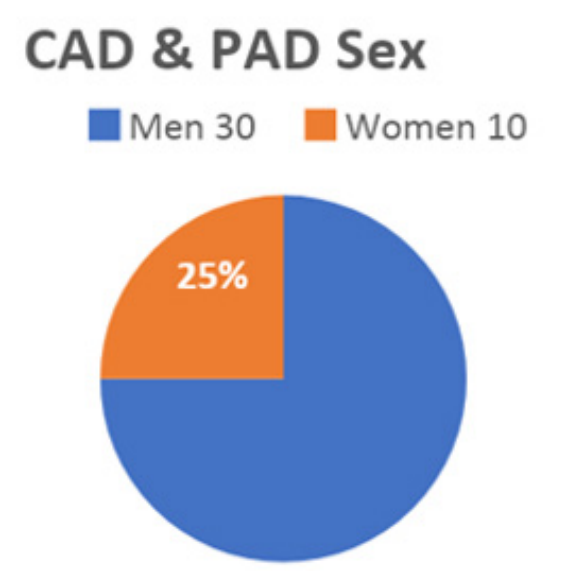
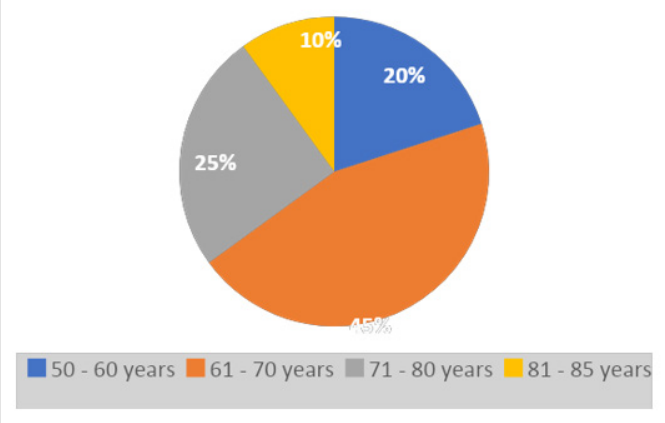
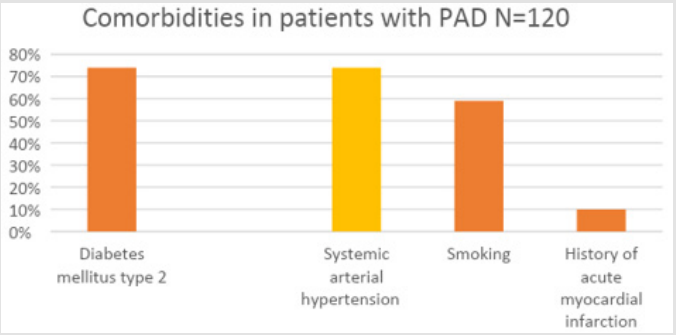
and PAD by age range (Graph 2). The predominant comorbidities in the 120 patients with PAD were: DM2 and SAH in 74%, smoking in 59%, (Graph 3). The comorbidities frequency in patients with CAD and PAD is shown in (Graph 4). The most frequent location of LE PAD was femoro-popliteal in 58 patients (48.3%) and tibial vessels in 21 (17.5%), both classified as Rutherford Category 5 (Table 2). The prevalence of CAD in our study in patients with PAD was 33.3% (40/120) (Graph 5). The clinical presentations of CAD in patients with PAD were: mild chronic ischemic heart disease in 11/40 (9.2%), and severe 15/40 (37.5%) (Tables 3 & 4) From 40 patients (33.3%) of the 120 patients with PAD, 37.5% (15/40) had severe coronary artery disease (Graph 6); of these, 73.3% (11/15) had multivessel disease and 26.6% (4/15) had left coronary artery disease.



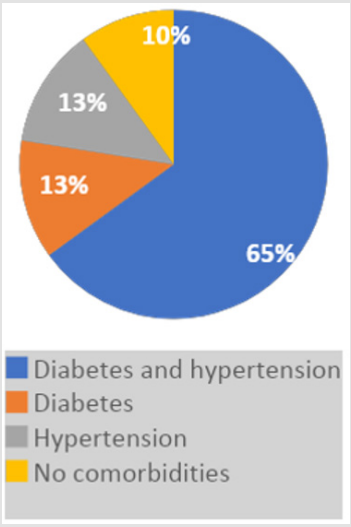
Graph 1: Frequency by gender in patients with CAD and PAD.



Graph 2: Source. - HECMNR Angiology Database.



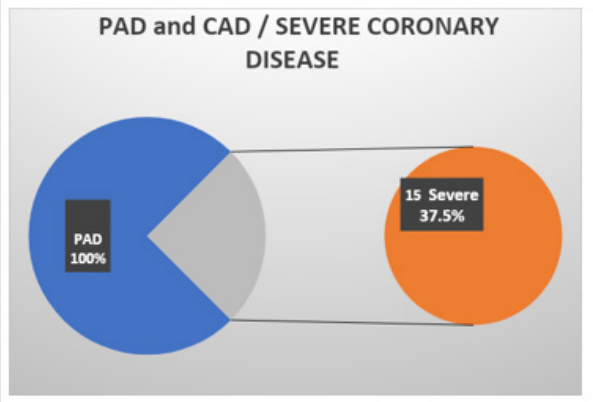
Graph 3: Comorbidities in patients with PAD N= 120.



Graph 4: Comorbidities in patients with CAD/PAD.

Graph 5: Ischemic heart disease. Patients with LE PAD (N= 120).

Note: Source: Angiology Database, La Raza National Medical Center.



Graph 6: Source: HECMNR clinical records.

Table 1: General characteristics of patients with CAD/PAD n=40 / N=120.

General characteristics	Frequency	Percentage
Gender	CAD/PAD	CAD/PAD
Male	30 / 34	75 / 28
Female	10 / 86	25 / 72
Physical constitution		
Low weight	3	2.5
Normal weight	71	59.2
Overweight	37	30.8
Obesity I	8	6.7
Obesity II	1	0.8
Affected member		
Right	61	50.8
Left	56	46.7
Both	3	2.5

Note: Source: Database of the Angiology Service; La Raza National Medical Center.

Table 2: Diagnosis of PAD and location. N= 120.

Diagnosis	Frequency	Percentage
FPD Rutherford category 5	58	48.3
TVD Rutherford category 5	21	17.5
AID type III Rutherford category 5	18	15.0
AID type III Rutherford category 4	6	5.0
FPD Rutherford category 3	3	2.5
FPD Rutherford category 4	3	2.5
TVD Rutherford category 6	2	1.7
AID type III Rutherford category 2	1	0.8
AID type I Rutherford category 5	1	0.8
AID type III Rutherford category 3	1	0.8
FPD Rutherford category 6	1	0.8
AID type III Rutherford category 5	1	0.8
AID type III Rutherford category 6	1	0.8

Note: Source: Database of the Angiology Service, La Raza National Medical Center.

Abbreviations: Aorto-iliac disease AID, Femoro-popliteal disease FPD, Tibial Vessel Disease TVD.

Table 3: Diagnostic of CAD in patients with LE PAD (N= 120).

Peripheral Arterial Disease N=120	Frequency	Percentage
No cardiac pathology	80	66.7
Coronary Artery Disease n=40 / n=120		
Coronary heart disease	40	33.3
Chronic ischemic heart disease	11	9.2
First-degree AV block	4	3.3
Chronic coronary syndrome	4	3.3
Paroxysmal atrial fibrillation	3	2.5
Right bundle branch lock	3	2.5
Severe coronary artery disease	15	37.5

Table 4: Laboratory tests of patients with LEPAD (N = 120).

Laboratory tests	Average	Minimal	Maximum
ABI	0.48 ± 0.12	0.27	0.78
Total cholesterol	150.4 ± 27.5	70	290
Triglycerides	226.1 ± 92.2	110	900
HDL	47.1 ± 6	38	67
LDL	174.8 ± 16.1	100	210

Note: Source: La Raza National Medical Center Clinical Archive.

Discussion

In a study conducted in our medical unit in 2019, to determine the prevalence of PAD in patients with ischemic heart disease, a prevalence of 36.9% was found in 225 patients; the most affected segment was the tibial vessels (53%), followed by the femoro-popliteus (27.7%); 24 In that study, patients admitted for CAD were looked for PAD; in our research, we studied patients who were hospitalized for PAD and found CAD. The above is very important, because it is described that in patients diagnosed with CAD and PAD, 30% have severe coronary heart disease, which CAD requires attention in the first place than PAD; and as we show in our research, the predominant lesions in more than 65%, are located in the femoro-popliteal segment and in the tibial vessels, most likely this implies that patients with PAD are underdiagnosed as well as patients with coronary artery disease, because they do not ambulate [23]. These locations surely influence the ambulation of patients, and with it, in cardiac manifestations.

There are no reports in Mexico of such a relationship; it is a priority to identify this association because of the high percentage CAC coupled with the predominant cardiovascular risk factors in our population.In relation to the population under 50 years of age, only 10% have coronary involvement; However, the diagnostic tools in these patients are deficient to assess the risks, and we must not forget that each time, atherosclerosis occurs in younger groups and in a more aggressive form [24,25]. Currently, the main problem is the necessity timely attention codes to detect and minimize the complications of pathologies that afflict developed and developing countries like ours; examples: heart attack code, brain code, to name a few. In our study, we detected CAD in 33.3% of patients with PAD who were admitted to our medical unit and who had no manifestations of this pathology. The importance of our research lies in the fact that it is described that ischemic cardiovascular diseases currently occupy the first places of morbidity and mortality in developed and developing countries, including, above oncological, traffic accidents and stroke, which is why the early diagnosis of CAD would allow us to achieve the prevention of acute ischemic attack and perform revascularization or appropriate medical management and thus reduce the morbidity and mortality and improve life quality of our population [26].

As we mentioned earlier, there are some unofficial reports that atherosclerosis behaves more aggressively in young people, so it would be very important to do more research to identify the role they play in this age population the miR-125a and miR-125b, identified in the arterial wall. Which play an important role in vascular pathogenesis that regulates vasoconstrictor gene expression and affects the synthesis and secretion of many vasoactive substances, which are associated with HDL (it is known that HDL has an important role in the innate and adaptive immune response, by modulating different components of the immune system); whereas the microRNA miR-125b promotes atherosclerosis in coronary patients and therefore, investigate the therapeutic potential of suppressing miR-125 in vivo [27]. Even though in our country the population pyramid is being inverted, there are reports of cardiovascular mortality since the 90'S of 33.4% in the population of 15-64 years, a fraction of the economically active population [28]. On the other hand, we must not forget that in our population it was found in 27% CAC, with predominance in the male sex (40%) and more severe [4]. In Latin America, mortality from coronary heart disease is increasing, and a 20% increase is expected for the 20's. Cardiovascular diseases represent 25% of all diseases aged 60 years and over, a prevalence that increases with age.

The fact that in our research we have found that the prevalence of CAD predominates mainly in patients with Rutherford category 5 (minor tissue loss), makes us think that we are detecting it late (in more advanced stages) then we must focus on preventive studies, practice preventive medicine and not only curative. In addition, our research showed that we are above international reports in terms of the severe coronary artery disease frequency in patients with PAD (37.5% vs 30%), surely, these results are influenced by the pathologies that afflict us (DM2, obesity, CAC). The frequency of presentation by gender in coronary heart disease was reversed with a predominance in male gender 3:1 (Graph 1).

Conclusion

Advances in molecular and histochemical medicine have not been sufficient to reduce the morbidity and mortality of cardiovascular diseases, so it is a priority to detect CAD early [29,30]. The diagnosis of coronary artery disease allows preventive measures to be taken to avoid complications and to establish a level of priority of the procedures to be carried out. The comorbidities that afflict our population influenced coronary disease.

Conflict of Interest

Authors declare no conflict interest.

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