characterise atherosclerotic plaque[1] (it must be kept in mind that it does not provide functional coronary information.) The feasibility and diagnostic accuracy of this study have improved due to widespread use of 64-slice multidetector CT scanners. Current systems provide a clear view of the main coronary arteries and their branches, with a spatial resolution similar to that of conventional angiography.[2] The usefulness of this technique is that it provides a maximum negative predictive value (99%-100%) to rule out coronary disease.[3] Thus, this technique significantly influences the stratification of selected patients with low or intermediate risk who come to the emergency department with chest pain.[2]

A triple rule-out study was performed after informing patients and obtaining their informed consent for inclusion in an institutional protocol. It was negative for pulmonary thromboembolism and acute aortic syndrome, while non-invasive coronary angiography (64-slice helical CT in prospective acquisition with volume reconstruction performed in sinus rhythm and calcium score 0) showed codominance; normal circumflex artery and left anterior descending artery and its branches; and an anomalous right coronary artery arising from the left sinus of Valsalva (Fig. 1A) with decreased calibre due to hypoplasia of the ostium and a sharp angled origin in the aorta. Furthermore, its course ran between the pulmonary artery and the ascending aorta (Figs. 1B and C). All these findings indicate a high risk anomalous origin of the right coronary artery.

Congenital anomalous origin of coronary arteries is seen in 1% to 1.2% of all coronary angiograms. 0.5% of them show high-risk lesions of the left main trunk or anterior descending branch with their origin in the opposite sinus of Valsalva. Coronary anomalies are the cause of 15% of sudden deaths in athletes. In 80% of the autopsies of athletes after sudden death with coronary arteries of anomalous origin, the affected coronary artery runs between the aorta and the pulmonary artery. Currently, the anatomical description of a coronary artery that passes between the aorta and pulmonary artery in a young person (under 50) is a major risk factor for an adverse event, with or without symptoms.[4] In this instance, after diagnostic confirmation by invasive coronary angiography, we opted for surgical revascularization by coronary reimplantation (AHA/ACC[3] class I indication). It is worth noting that we were able to confirm the anomalous origin and interarterial course of the coronary artery, and furthermore, we found a 2 cm intramural course beginning at the mid-surface of the right sinus. We reimplanted the right coronary artery in the non-coronary sinus, without complications and with a good outcome (Figs. 2A-C).

Cristina Goena,* Amaia Ibarra, Marta Campaña, Rubén Natividad, Koldobika García, and Pedro M. Montes

Servicio de Cardiología, Hospital de Cruces, Barakaldo, Bizkaia, Spain

* Corresponding author:
E-mail address: M.CRISTINA.GOENAVIVES@osakidetza.net (C. Goena).

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Parameters of Arterial Stiffness: Hypertensive and Diabetic Patients vs Controls

Parámetros de rigidez arterial en sujetos hipertensos y diabéticos comparados con controles

To the Editor,

Arterial stiffness predicts cardiovascular morbidity and mortality, and can be evaluated with: pulse wave velocity (PWV),[1] ambulatory arterial stiffness index (AASI),[2,3] and augmentation index (AI).[4]

There are studies in Spain on arterial stiffness in various subpopulations,[5] but there are no studies that have jointly analyzed PWV, AASI, and AI in diabetic and hypertensive patients. The aim of this study is to report average values for these parameters in diabetic and hypertensive patients compared to controls, and to analyze determining factors.

From 2006 to 2010, we recruited 373 patients from consultations in 2 health centers. Those who had medical histories of cardiovascular disease were excluded. The sample size was estimated for detecting differences of 1 m/s in PWV between groups. By assuming an alpha risk of 0.05, a beta risk of 0.2 and a standard deviation of 2.12 m/s, 95 subjects per group were needed.

The protocol was approved by the research ethics committee and all patients signed an informed consent form.

The PWV and AI were estimated with the SphymgoCor System. We performed the aortic pulse wave analysis with a sensor on the radial artery with the patient seated. Using mathematical transformation based on the radial wave, we estimated the central blood pressure and the central and peripheral AI. We evaluated reliability by intraclass correlation (r = 0.974; 95% confidence interval [CI], 0.936-0.989) in repeated measurements of 22 subjects. Carotid-femoral PWV was determined with the patient lying down, estimating the pulse wave delay at the carotid and femoral level with respect to the electrocardiogram. We defined AASI as 1 - the regression slope for the diastolic blood pressure (DBP) and the systolic blood pressure (SBP) of 24-hour readings, and the Sym-AASI as 1 - (1- AASI) / r.

We assessed the association between qualitative variables with the χ^2 test, and the difference of the means with ANOVA. We controlled for age with ANCOVA. We analyzed the variables related to the parameters of arterial stiffness with stepwise multiple regression analysis, adjusted for age and sex. PWV, AASI and central augmentation index (cAI) were used as dependent variables. The independent variables were smoking, SBP, DBP, heart rate (HR), body mass index, total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, baseline glycemia, C-reactive protein and fibrinogen. We used the SPSS/PC+18.0 statistical program.

Table 1 shows the cardiovascular risk factors, average values of the stiffness parameters, and average antihypertensive and lipid-lowering drugs per group. After adjusting for age, the differences between diabetic patients and controls were: PWV, 1.13m/s (95% CI, 0.51-1.74) ($P < .01$); AASI, 0.01 (95% CI, –0.01- 0.03); and cAI, 2.87 (95% CI, –0.82-6.56). Differences between hypertensive

patients and controls were: PWV, 0.67m/s (95% CI, 0.14-1.21) ($P < .05$); AASI, −0.01 (95% CI, −0.03-0.01); and cAI, 5.45 (95% CI, 2.26-8.66) ($P < .05$). In diabetic, hypertensive, and control patients, the correlation between PWV and AASI was 0.42 ($P < .01$), 0.38 ($P < .01$) and 0.19; between PWV and cAI, 0.15, −0.12 and 0.33 ($P < .01$) and between AASI and cAI, 0.1, 0.03 and 0.02, respectively.

The factors that best explain the arterial stiffness parameters in PWV, adjusted for age and sex, are SBP and waist circumference. They achieve the best fit ($r^2 = 0.6$) in diabetic patients. AASI is explained by SBP and DBP, with the best fit in diabetic patients ($r^2 = 0.61$). For cAI, the variable that is maintained in all cases is HR, with the best fit ($r^2 = 0.55$) in controls (Table 2).

The data obtained allow us to report the values and factors associated with PWV, AASI and AI in hypertensive, diabetic and control patients. PWV, but not AASI or AI, has a similar behavior in diabetic and hypertensive patients, after adjusting for age.

The values found for PWV, adjusted for age, are similar to those of the Framingham study.[1] When these values are compared with reference values from Europe, according to age and blood pressure readings, they are higher in hypertensive patients and the same in controls.[6] As in previous studies, the variability of PWV is greatly influenced by age and blood pressure.

AASI values were lower than those of the Dublin Outcome study[2] (AASI of 0.41, and 0.46 in diabetic patients) and the Ohasama study[3] (AASI of 0.45 in nonhypertensive patients, and 0.46 in hypertensive patients) with no differences between groups, as in our study. The differences may be due to the fact that neither age nor distribution of other risk factors is the same. AASI variability is influenced by age, sex, blood pressure, and HR.

cAI varies according to the subjects' characteristics, as shown in 11 studies that analyzed central blood pressure values.[6] Heart rate, age, sex, and blood pressure are some of the variables that are maintained in the multiple regression.

In conclusion, stiffness parameters do not behave uniformly. Thus, while PWV adjusted for age is greater in diabetic patients than in hypertensive ones and is greater in hypertensive patients than in controls, the same does not happen with the other evaluated parameters. These results would support systematic evaluation of PWV in diabetic and hypertensive patients.

Table 1
Clinical Characteristics, Cardiovascular Risk Factors, and Values for Arterial Stiffness Parameters

	Diabetic patients (n = 100)	Hypertensive patients (n = 174)	Control patients (n = 99)
Age (years) [a,b,c]	59.29 ± 10.56	55.09 ± 11.36	40.41 ± 12.12
Males	65 (65)	106 (60.9)	56 (56.6)
Risk factors			
Smoking	20 (20)	51 (29.3)	20 (20.2)
Clinical SBP (mm Hg)[b,c]	137.09 ± 18.68	142.7 ± 18.47	135.47 ± 12.03
Clinical DBP (mm Hg)[b,c]	83.09 ± 11.33	90.12 ± 11.3	85.64 ± 8.43
Clinical PP (mm Hg)[a]	54.61 ± 13.85	53.14 ± 13.22	50.25 ± 11.87
HR (bpm)[b]	72.36 ± 12.89	71.3 ± 12.24	74.74 ± 12.72
SBP OBPM 24h (mm Hg)[b,c]	123.08 ± 12.6	129.5 ± 14.24	121.78 ± 7.36
DBP OBPM 24h (mm Hg)[a,b,c]	72.61 ± 8.56	80.56 ± 10.85	75.49 ± 6.5
PP OBPM 24h (mm Hg)[a,b]	50.46 ± 10.12	48.93 ± 10.46	46.29 ± 6.92
HR OBPM 24h (bpm)	73.67 ± 9.94	71.45 ± 10.6	72.61 ± 9.91
Number of antihypertensive drugs[a,b,c]	1.32 ± 1.11	1.02 ± 1.02	0
BMI[a,c]	29.9 ± 5.24	28.1 ± 4	27.69 ± 3.68
Total cholesterol (mg/dl)	189.81 ± 38.33	208.43 ± 33.5	212.33 ± 41.92
LDL-C (mg/dl)[b,c]	111.19 ± 29.82	130.21 ± 31	133.24 ± 36.77
HDL-C (mg/dl)[b,c]	48.92 ± 11.13	53.03 ± 13.1	53.6 ± 12.71
Triglycerides (mg/dl)	143.28 ± 88.8	129.14 ± 78.27	124.61 ± 69.84
Number of lipid-lowering drugs[a,b,c]	0.58 ± 0.53	0.22 ± 0.43	0.11 ± 0.31
Baseline glycemia[a,b,c]	133.03 ± 41.25	88.69 ± 11.05	85.17 ± 9.84
Waist circumference (cm)[b,c]	102.32 ± 12.63	96.70 ± 11.49	94.06 ± 11.01
Insulin resistance (μU/ml) (HOMA index)[b,c]	3.69 ± 4.16	2.23 ± 1.88	1.88 ± 1.46
CRP (mg/dl)[a,b,c]	0.35 ± 0.47	0.31 ± 0.46	0.18 ± 0.18
Plasma fibrinogen (mg/dl)[a,b]	328.77 ± 67.28	323.05 ± 63.87	306.29 ± 53.04
Arterial stiffness parameters			
Pulse wave velocity (m/s)[a,b,c]	9.84 ± 2.35	9.09 ± 2.12	7.99 ± 1.82
Ambulatory arterial stiffness index[a,c]	0.41 ± 0.06	0.37 ± 0.06	0.37 ± 0.05
Sym-AASI[b,c]	0.37 ± 0.06	0.34 ± 0.06	0.34 ± 0.05
Central augmentation index[a,b]	30.77 ± 11.14	32.46 ± 10.47	26.03 ± 12.42
Peripheral augmentation index[a,b]	94.39 ± 22.54	96.35 ± 20.74	86.02 ± 20.25
Central PP[a,b]	44.38 ± 12.06	44.9 ± 14.21	39.24 ± 10.48

BMI, body mass index; CRP, C-reactive protein; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; HR, heart rate; LDL-C, low-density lipoprotein cholesterol; OBPM, outpatient blood pressure monitoring; PP, pulse pressure; SBP, systolic blood pressure; Sym-AASI, symmetrical ambulatory arterial stiffness index. Data is presented as mean ± standard deviation or no. (%).

[a] Differences between diabetic and control patients.

[b] Differences between hypertensive and control patients.

[c] Differences between diabetic and hypertensive patients ($P < .05$).

Table 2
Multiple Regression Analysis of Arterial Stiffness Parameters in Diabetic, Hypertensive, and Control Patients

Diabetic patients			Hypertensive patients			Control patients		
	β (95% CI)	*P*		β (95% CI)	*P*		β (95% CI)	*P*
PWV, $r^2 = 0.6$			PWV, $r^2 = 0.42$			PWV, $r^2 = 0.42$		
Constant	−8.82 (−12.975 to −4.663)	<.01	Constant	−6.546 (−10.037 to −3.054)	<.01	Constant	−3.113 (−7.359 to 1.133)	.149
Age	0.083 (0.051-0.116)	<.01	Age	0.088 (0.065-0.111)	<.01	Age	0.071 (0.042-0.1)	<.01
Sex	−0.311 (−1.044 to 0.422)	.402	Sex	0.34 (−0.243 to 0.924)	.251	Sex	−0.649 (−1.35 to 0.051)	.069
SBP	0.062 (0.043-0.082)	<.01	SBP	0.034 (0.02-0.048)	<.01	SBP	0.034 (0.007-0.061)	.013
Fibrinogen	−0.011 (−0.016 to −0.005)	<.01	HR	0.034 (0.012-0.056)	.003	Waist circumference	0.035 (0.004-0.065)	.025
HR	0.034 (0.006-0.061)	.016	Waist circumference	0.034 (0.01-0.058)	.006			
Waist circumference	0.045 (0.015-0.075)	.004						
Cholesterol	0.01 (0.001-0.019)	.037						
AASI, $r^2 = 0.61$			AASI, $r^2 = 0.58$			AASI, $r^2 = 0.5$		
Constant	0.349 (0.257-0.441)	<.01	Constant	0.245 (0.162-0.328)	<.01	Constant	0.409 (0.306-0.513)	<.01
Age	0.001 (<0.001-0.002)	.017	Age	0.001 (0.001-0.002)	<.01	Age	0.001 (<0.001-0.002)	.009
Sex	−0.036 (−0.054 to −0.017)	<.01	Sex	−0.023 (−0.037 to −0.009)	.001	Sex	−0.024 (−0.041 to −0.006)	.009
SBP	0.003 (0.002-0.003)	<.01	SBP	0.003 (0.002-0.004)	< .01	SBP	0.002 (0.001-0.003)	<.01
DBP	−0.004 (−0.006 to −0.003)	<.01	DBP	−0.005 (−0.006 to −0.003)	<.01	DBP	−0.004 (−0.006 to −0.003)	<.01
HR	−0.001 (−0.001 to <0.001)	.017	BMI	0.002 (0.001-0.004)	.01			
BMI	0.002 (<0.001-0.004)	.019						
cAI, $r^2 = 0.46$			cAI, $r^2 = 0.34$			cAI, $r^2 = 0.55$		
Constant	54.578 (37.912-71.243)	<.01	Constant	34.448 (17.3-51.596)	<.01	Constant	39.056 (18.487-59.624)	<.01
Age	0.263 (0.089-0.438)	.004	Age	0.208 (0.086-0.33)	.001	Age	0.358 (0.216-0.501)	<.01
Sex	−11.452 (−15.349 to −7.556)	<.01	Sex	−8.932 (−11.793 to −6.07)	<.01	Sex	−7.965 (−11.548 to −4.382)	<.01
HR	−0.439 (−0.58 to −0.298)	<.01	HR	−0.242 (−0.358 to −0.125)	<.01	HR	−0.356 (−0.487 to −0.224)	<.01
			Fibrinogen	−0.039 (−0.061 to −0.017)	.001	DBP	0.274 (0.067-0.481)	.01
			DBP	0.243 (0.118-0.368)	<.01	BMI	−0.838 (−1.282 to −0.394)	<.01

AASI, ambulatory arterial stiffness index; BMI, body mass index; cAI, central augmentation index; DBP, diastolic blood pressure; HR, heart rate; PWV, pulse wave velocity; SBP, systolic blood pressure.

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CONFLICTS OF INTEREST

None declared.

Manuel A. Gómez-Marcos,[a,*] José I. Recio-Rodríguez,[a] María C. Patino-Alonso,[b] Cristina Agudo-Conde,[a] Emiliano Rodríguez-Sánchez,[a] and Luis García-Ortiz[a]

[a]*Unidad de Investigación La Alamedilla, REDIAPP, Salamanca, Spain*
[b]*Departamento de Estadística, Universidad de Salamanca, Salamanca, Spain*

[*] Corresponding author:
E-mail address: magomez@usal.es (M.A. Gómez-Marcos).

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