Scientific letters

Oral Glucose Tolerance Test as a Tool for Patient Improvement After Percutaneous Coronary Intervention

Sobrecarga oral de glucosa como herramienta para la mejora tras intervencionismo coronario percutáneo

To the Editor,

High rates of unknown diabetes mellitus (DM) have been described in coronary patients.[1–3] Early diagnosis and treatment of DM is essential to reduce cardiovascular morbidity and mortality rates.[2] Bramlage et al.[4] demonstrated that secondary prevention in coronary patients with diabetes is only viable when optimal standards are met. Therefore, early diagnosis and optimal treatment would provide potential improvement for coronary patients with diabetes.

We analyzed 338 patients that underwent percutaneous coronary interventions (PCI) for revascularization, with systematic oral glucose tolerance tests (OGTT) and no known DM.[3] We divided the patients into a group treated with metabolic interventions and a control group. We attempted to determine whether differences could be observed after 1 year of follow-up in terms of: *a)* adequacy of secondary prevention; *b*) prevalence of metabolic syndrome (MS), and *c)* combined cardiovascular events (death, myocardial infarction, stroke, new coronary revascularization, and hospitalization for unstable angina).

The methodology of our study has already been described.[3] We performed a clinical interview, a patient examination, and laboratory analyses including OGTT and glycosylated hemoglobin at 15 days after hospital discharge and again at 1 year.

Our center serves as a PCI referral center for 8 hospitals. We offered our patients intensive multidisciplinary treatment. Patients from other hospitals received standard medical treatment and served as the control group. All patients and their responsible physicians were informed of the results of all the tests performed.

Intensive multidisciplinary treatment, based on clinical guidelines,[2] was provided during diabetic education nursing, cardiology, and endocrinology visits. All patients were informed as to the nature of the disease, lifestyle changes, tobacco abstinence, treatment (indefinite platelet therapy, beta blockers, statins), and target blood pressure (BP) values<140/90 mm Hg and low-density lipoprotein cholesterol (LDL-C) values<100 mg/dL. In addition, prediabetic and/or obese patients received personalized hypocaloric diet recommendations, and in patients with occult diabetes, recommendations included diet, metmorphin if there was glycosylated hemoglobin≥6.5% and/or associated obesity, target BP<130/80 mm Hg, LDL-C<70 mg/dL, and renin-angiotensin system inhibitors.

We described our study sample using traditional descriptive statistics. We compared continuous variables using Student's t-test, the Mann-Whitney U-test, and the Wilcoxon test, as

Table
Changes in the Various Parameters Measured for Secondary Prevention in the Study Population at 12 Months

	Treatment (n=98)			Control (n=220)			P^*
	Baseline	12 months	*P*	Baseline	12 months	*P*	
SBP, mm Hg	132.2±19.6	126.7±22.3	.036	135.9±18.8	135.8±18.8	.916	.035
Controlled BP (<140/90 without DM and <130/80 with DM)	42 (45.7)	65 (70.7)	<.001	68 (31.3)	116 (53.5)	<.001	.819
Weight, kg	79±14.9	77.4±15.4	<.001	78.7±10.8	78.5±12.6	.688	.045
Waist circumference, cm	98.8±9.7	96.4±9.7	<.001	97.4±7.7	96±12.2	.054	.001
BMI, kg/m^2	29.3±4.4	28.7±4.7	.595	28.8±3.3	28.8±4.1	.729	.042
Glucose, mg/dL	102.2±21.9	96.9±12.5	.005	99.03±13.5	96.4±12.9	.001	.130
HbA_{1c}, %	4.9±0.72	4.8±0.57	.035	4.8±0.57	4.8±0.051	.154	.080
LDL-C, mg/dL	90.7±29.2	87.2±32.9	.364	94.6±34.3	97.6±34.4	.198	.134
Controlled LDL-C (<100 without DM and <70 with DM)	56 (63.6)	56 (63.6)	1	112 (57.1)	101 (51.5)	.185	.401
HDL-C, mg/dL	43.7±11.1	48.7±12.7	<.001	47.6±13.1	52.1±13.3	<.001	.583
Triglycerides, mg/dL	141.6±64.6	118.9±70.7	<.001	130.9±72.8	116.4±61.2	<.001	.225
Microalbuminuria, mg/g creatinine	5.4±15.8	4.8±11.3	.133	4.2±12.6	4.3±10.9	.240	.419
MS	53 (54.1)	36 (36.7)	.005	99 (45)	88 (40)	.208	.066
Number of MS criteria met	2.4±1.1	2.1±1.2	.011	2.2±1.1	2±1.1	.001	.727
Statins	89 (90.8)	89 (90.8)	1	175 (79.5)	186 (84.5)	.029	.839
ACE inhibitors/ARB	50 (51)	68 (69.4)	<.001	75 (34.1)	75 (34.1)	1	<.001
Beta blockers	78 (82.6)	71 (79.6)	.092	156 (70.9)	148 (67.2)	.815	.125
Anti-platelets	97 (99)	97 (99)	1	219 (99.5)	219 (99.5)	1	1
Quadruple therapy	42 (40.4)	45 (48.9)	.180	45 (19.2)	48 (22.1)	.424	.710

ACE inhibitors/ARB, angiotensin-converting enzyme inhibitors/angiotensin II receptor blockers; BMI, body mass index; BP, blood pressure; DM, diabetes mellitus; HbA_{1c}, glycosylated hemoglobin; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure; MS, metabolic syndrome. Quadruple therapy: combined treatment with statins, angiotensin-converting enzyme inhibitors/angiotensin II receptor blockers, beta blockers, and antiplatelets.
Categorical variables are expressed as the absolute value (%), while numerical variables are expressed as mean±standard deviation.
* Test for heterogeneity of differences between treatment and control groups.

appropriate. We used chi-square tests to compare categorical variables. We performed log rank (Mantel-Cox) and Kaplan-Meier survival curve analyses to test for associations between events and patient groups. We also performed a multivariate Cox regression analysis, adjusted by age, sex, hypertension, MS, renal function, vascular disease, glycemia, acute coronary syndrome, previous infarction, ejection fraction, multivessel disease, and treatment.

There were no significant baseline differences between the treatment (n=104) and control (n=234) groups in terms of age, sex, hypertension, tobacco use, MS, vascular disease, history of infarction, multivessel disease, ejection fraction, BP, weight, lipids, renal function, or glycemia after performing the OGTT (occult DM: 24% vs 22.2%, *P*=.779; prediabetes: 39.4% vs 33.3%, *P*=.32; normal glycemia: 36.5% vs 44.4%; *P*=.190). The only significant differences were found in treatment upon discharge (Table).

Six patients (5.7%) were lost to follow-up in the intensive multidisciplinary care group vs 14 in the control group (6%).

The Table shows the changes observed in the secondary prevention group after 1 year. The prevalence of MS, as a paradigm of global secondary prevention, decreased in the treatment group (54.5% vs 36.7%; *P*=.005) but did not vary in the control group (45% vs 40%; *P*=.208).

After 1 year of follow-up (Figure), we observed a lower rate of cardiovascular events in the treatment group (9.2% vs 18.2%; *P*=.023). The decrease in cardiovascular events was primarily in rehospitalizations for unstable angina. Inclusion in the intensive multidisciplinary treatment program was associated with fewer events (odds ratio=0.36; 95% confidence interval, 0.15-0.86; *P*=.022).

In this study, we show that a multidisciplinary group of health care professionals that provide coordinated care to coronary patients with no known DM can achieve positive results in 3 distinct aspects: *a)* improved optimization of secondary prevention, with increased use of quadruple therapy (48.9% vs 22.1%), yielding a higher percentage of targets reached and lower BP and LDL-C levels; *b)* decreased prevalence of MS, and *c)* a decrease in combined cardiovascular events.

Our study's limitations are due to the sample size (e.g., absence of statistical significance for MS despite clinically relevant changes) and its observational, nonrandomized design. Since other inherent variables come into play with the intensive treatment protocol (tertiary hospital, shifts, and a coronary unit run by cardiologists), not all changes can be attributed to the intensive treatment.

At a time when European and American medical societies have not reached an agreement on the recommendations for using OGTT,[2,5] our study poses the following question: does the use of OGTT in coronary patients (even those selectively chosen[6]) aid in improving the health of our patients? Our data suggests that it does.

	Treatment (n=98)	Control (n=220)	*P*
Death	4 (4.1%)	6 (2.7%)	.575
New revascularization	4 (4.1%)	16 (7.3%)	.224
IS	2 (2%)	3 (1.4%)	.694
AMI	4 (4.1%)	13 (5.9%)	.459
Unstable angina	8 (8.2%)	37 (17.8%)	.031
Combined	9 (9%)	40 (18.2%)	.023

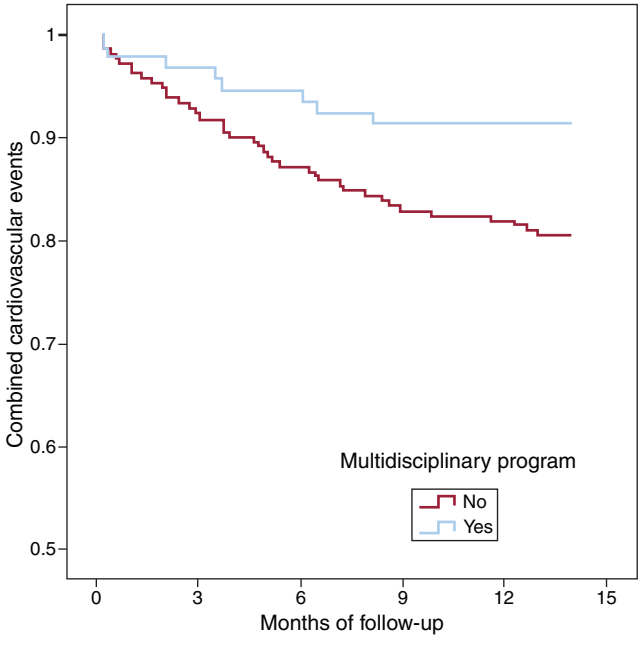

Figure. Kaplan-Meier curve for survival free of cardiovascular events. Event details. AMI, acute myocardial infarction; IS, ischemic stroke.

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REFERENCES

1. Norhammar A, Tenerz A, Nilsson G, Hamsten A, Efendic S, Ryden L, et al. Glucose metabolism in patients with acute myocardial infarction and no previous diagnosis of diabetes mellitus: a prospective study. Lancet. 2002;359:2140-4.
2. Ryden L, Standl E, Bartnik M, Van den BG, Betteridge J, De Boer MJ, et al. Guías de práctica clínica sobre diabetes, prediabetes y enfermedades cardiovasculares. Rev Esp Cardiol. 2007;60:525.e1–64.
3. De la Hera JM, Delgado E, Hernández E, García-Ruiz JM, Vegas JM, Avanzas P, et al. Prevalence and outcome of newly detected diabetes in patients who undergo percutaneous coronary intervention. Eur Heart J. 2009;30:2614–21.
4. Bramlage P, Messer C, Bitterlich N, Pohlmann C, Cuneo A, Stammwitz E, et al. The effect of optimal medical therapy on 1-year mortality after acute myocardial infarction. Heart. 2010;96:604–9.

5. Drozda Jr J, Messer JV, Spertus J, Abramowitz B, Alexander K, Beam CT, et al. ACCF/AHA/AMA-PCPI 2011 performance measures for adults with coronary artery disease and hypertension. A report of the American College of Cardiology Foundation/American Heart Association Task Force on Performance Measures and the American Medical Association-Physician Consortium for Performance Improvement. J Am Coll Cardiol. 2011;58:316–36.

6. De la Hera JM, Vegas JM, Hernández E, Lozano I, Garcia-Ruiz JM, Fernández-Cimadevilla OC, et al. Rendimiento de la glucohemoglobina y un modelo de riesgo para la detección de diabetes desconocida en pacientes coronarios. Rev Esp Cardiol. 2011;64:759–64.

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Ischemic Heart Disease in Women. Data From BARIHD Study

Cardiopatía isquémica en la mujer. Datos del estudio CIBAR

To the Editor,

The relationship between ischemic heart disease and sex is evidenced by continuing differences between men and women in treatment and secondary prevention strategies and their outcomes. Nevertheless, progress has been made over the last decade.[1–3] The aim of our study was to characterize differences by sex in the clinical features, diagnosis, treatment, and prognosis of an initial cohort of 1108 patients with chronic ischemic heart disease drawn from primary care (PC) practice, and with a minimum of 1 year of evolution after the first episode. Patients were from the CIBAR (Barbanza Ischemic Heart Disease) study, a prospective, multicenter, cohort study launched in 2007 in which, over a one month period,

Table 1
Clinical Features, Risk Factors, Comorbidities, Diagnostic Tests, and Patient Treatment in the CIBAR Study. Distribution by Sex

	Total	Men	Women	*P*
Patients	1108 (100)	798 (72)	310 (28)	
Age, years	69.2 ± 11.1	71.1 ± 9.8	68.5 ± 11.5	<.001
Stable angina	258 (23.3)	166 (20.9)	92 (29.3)	.003
Unstable angina	243 (21.9)	156 (19.6)	87 (27.7)	.004
Acute myocardial infarct	607 (54.8)	472 (59.4)	135 (43)	<.001
High blood pressure	726 (65.5)	478 (60.2)	248 (79.4)	<.001
Dyslipidemia	779 (70.3)	542 (67.9)	237 (76.5)	.009
Diabetes	318 (28.7)	212 (26.6)	106 (34.2)	.015
Current smoker	110 (9.9)	100 (12.5)	10 (3.2)	<.001
Obesity[a]	436 (39.4)	289 (36.2)	147 (47.4)	.001
Central obesity[b]	604 (54.5)	367 (46)	237 (76.5)	<.001
Metabolic syndrome[c]	490 (44.2)	309 (38.7)	181 (58.4)	<.001
Previous heart failure	120 (10.8)	81 (10.2)	39 (12.6)	.238
Atrial fibrillation	159 (14.4)	105 (13.2)	54 (17.4)	.071
Ictus	97 (8.8)	66 (8.3)	31 (10)	.407
Peripheral vascular disease	153 (13.8)	122 (15.3)	31 (10)	.025
Valve disease	175 (15.8)	108 (13.5)	67 (21.6)	.001
Echocardiogram	854 (77.1)	622 (77.9)	232 (74.8)	.266
Ergometry	617 (55.7)	453 (57.1)	160 (51.3)	.077
Coronary catheterization	827 (74.6)	628 (78.7)	199 (64.2)	<.001
Multivessel lesion	405 (49)	324 (51.6)	81 (40.3)	<.001
Coronary angioplasty	439 (39.6)	347 (43.5)	92 (29.7)	<.001
Coronary artery bypass surgery	195 (17.6)	161 (20.3)	34 (10.8)	<.001
Antiplatelet treatment	914 (82.5)	668 (83.7)	246 (79.4)	.094
Anticoagulants	184 (16.6)	127 (15.9)	57 (18.4)	.324
Nitrates	571 (51.5)	397 (49.7)	174 (56.1)	.061
Beta blockers	665 (60)	487 (61)	178 (57.4)	.275
ACE inhibitors and/or ARBs	674 (60.8)	466 (58.4)	208 (67.1)	.009
Calcium channel blockers	422 (38.1)	288 (36.1)	134 (43.2)	.033
Diuretics	367 (33.1)	237 (29.7)	130 (41.9)	<.001
Statins	967 (87.3)	695 (87.1)	272 (87.7)	.841
Physical exercise[d]	850 (76.7)	630 (79.3)	230 (70.1)	.001

ARB, angiotensin II receptor blockers; ACE, angiotensin-converting-enzyme.
Data are n (%) or mean ± standard deviation.
[a] Body mass index>30.
[b] Abdominal perimeter≥102 cm in men, ≥88 cm in women.
[c] Based on ATP-III 2001.
[d] Defined as any aerobic exercise lasting over 20 min, at least 3 times a week.