Scientific letters

Health Care Delay Times in Acute Myocardial Infarction: Differences by Gender

Tiempos de demora de atención sanitaria en el infarto agudo de miocardio: diferencias por sexos

To the Editor,

Several national[1] and international[2] studies have examined differences by gender in health care delay in patients with acute myocardial infarction (AMI). These studies have generally shown that delay times are longer in women than in men. Nevertheless, the results have been highly disparate due to differences in methodology and heterogeneity in the populations studied. In particular, different criteria were used to define AMI in studies carried out before 2001.[3]

We performed a prospective study to analyze delay times in the Cardiology Critical Care Unit of the *Marqués de Valdecilla* University Hospital between 2007 and 2009. We used a paired cohort design based on the age of the women included. A total of 203 patients were recruited consecutively into the study. Individuals diagnosed with the new diagnostic criteria for AMI[3] were eligible for inclusion in the study with no restriction on age. Patients who had a cardiac arrest before admission, those who did not speak Spanish or who had cognitive disorders were excluded from the study. Thirty-six patients were excluded due to the lack of reliable data. Data were obtained from clinical records and a standardized questionnaire completed by cardiology critical care unit nurses during a face-to-face interview with the patient performed after admission and within 48 h of the AMI. Contact delay time was defined as the time (in minutes) from symptom onset until the first medical contact. Transfer delay time was the time from the first request for assistance to arrival at the hospital, and total prehospital delay (TPD) was the time (in minutes) from symptom onset to arrival at the emergency department.
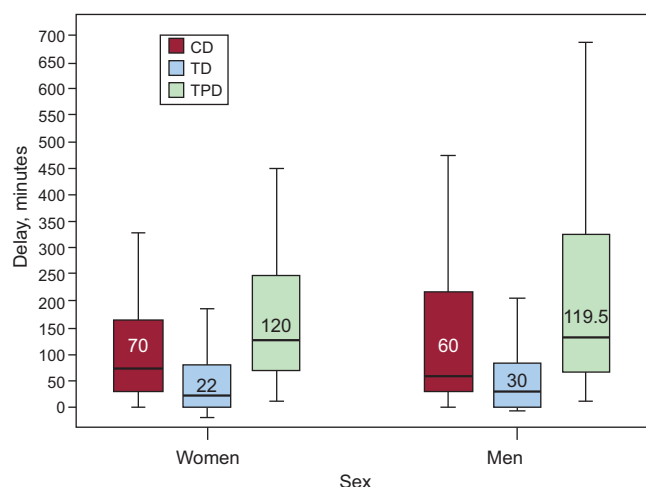
We used SPSS 15.0 for the statistical analysis. All delay times were expressed as medians [interquartile range]. To test for differences by gender we used the Wilcoxon and Mann-Whitney tests and logistic regression analysis using the 'enter' method.

A total of 370 patients were included in the study, of which 188 were women. Mean age was 67 (13.5) years. Median contact delay time was 65 [150] min. Median transfer delay time was 28 [81.75] min. Median TPD was 120 [226.3] min: women, 120 [180] min and men, 119.5 [277.5] min. There was greater variability in delay times among men, as indicated by the wider interquartile range. Other delay times are shown in the Figure, although no statistically significant differences were observed (*P*=.483). Likewise, there were no significant differences by gender when the data were examined by time bands (Table).

With respect to the type of AMI, TPDs were significantly higher in non-Q wave AMIs (median, 166 min vs 108.5 min; *P*<.05) than in Q wave AMIs, due to the longer contact delay time (median, 75 min vs 60 min; *P*=.067); the transfer delay time was almost identical (median, 27 min vs 28 min; *P*=.759) in both genders.

The multivariate analysis was performed with variables identified by earlier studies[2] as being associated with TPD and which were found to be significant, i.e. educational level (odds ratio [OR]=2.4; *P*=.001), pain intensity (OR=0.828; *P*=.003), history of ischemic heart disease (OR=1.871; *P*=.028) and diabetes at the limit of statistical significance (OR=1.686; *P*=.059).

In this study, in which patients were matched by age, we found no differences by gender in the time to seeking health care. Although sex matching is useful to control for a possible confounding effect of age, delay times in the overall sample were overstated for males. In contrast to our findings, most studies performed to date[1,2,4] have reported longer delays in women. We believe that the difference may be due to the effect of age and sex. As reported in other studies,[1,2,4] the difference by sex in delay times increases with age. The TPD in our study was 120 min, which is comparable to that in other international studies.[5] Surprisingly, despite technological advances in transportation over recent years, delay times have remained constant over the past 3 decades.[5] Only half of patients reach the hospital within 120 min after the onset of infarction pain, i.e. within the time considered appropriate in clinical practice guidelines[6] if maximum benefit is to be obtained from reperfusion therapies. Delays could be reduced by educational campaigns to help patients recognize the early signs and clinical symptoms of AMI, particularly in groups with longer delay times such as diabetics or patients with low education levels.

Figure. Median contact, transfer, and total prehospital delay times in both sexes. CD, contact delay; TD, transfer delay; TPD, total prehospital delay.

Table
Total Prehospital Delay Times in Acute Myocardial Infarction

Times	Total	Women	Men	P
<1 h	82 (22.2)	41 (21.8)	41 (22.5)	ns
1-2 h	100 (27)	50 (26.6)	50 (27.5)	ns
2-6 h	107 (28.9)	61 (32.4)	46 (25.3)	ns
>6 h	81 (21.9)	36 (19.1)	45 (24.7)	ns
Total	370 (100)	188 (100)	182 (100)	

ns, not statistically significant.
Data shown are no. (%).

Ana R. Alconero-Camarero,* Pedro Muñoz-Cacho,
and José M. Revuelta

Departamento de Enfermería, Universidad de Cantabria, Servicio de Cirugía Cardiovascular, Hospital Universitario Marqués de Valdecilla, Santander, Spain

* Corresponding author:
E-mail address: alconear@unican.es (A.R. Alconero-Camarero).

REFERENCES

1. Anguita M, Alonso J, Bertomeu V, Gómez-Doblas JJ, López-Palop R, Pedreira M, et al. Diseño general del proyecto de estudio sobre la situación de la enfermedad cardiovascular de la mujer en España. Rev Esp Cardiol. 2008;8 Supl D:4–7.
2. Nguyen HL, Saczynski JS, Gore JM, Goldberg RJ. Age and sex differences in duration of prehospital delay in patients with acute myocardial infarction: a systematic review. Circ Cardiovasc Qual Outcomes. 2010;3:82–92.
3. Thygesen K, Alpert JS, White HD. Universal definition of myocardial infarction. Eur Heart J. 2007;28:2525–38.
4. Cabadés A, Valls F, Echanove I, Francés M, Sanjuán R, Calabuig J, et al.; Estudio RICVAL. El infarto agudo de miocardio en la ciudad de Valencia. Datos de 1.124 pacientes en los primeros 12 meses del registro. Rev Esp Cardiol. 1997;50:383–96.
5. Goldberg RJ, Spencer FA, Fox KA, Brieger D, Steg PG, Gurfinkel E, et al. Prehospital delay in patients with acute coronary syndromes (from the Global Registry of Acute Coronary Events [GRACE]). Am J Cardiol. 2009;103:598–603.
6. Wijns W, Kolh P, Danchin N, Di Mario C, Falk V, Folliguet T, et al. Guía de práctica clínica sobre revascularización miocárdica. Rev Esp Cardiol. 2010;63:1485.e1–76.

http://dx.doi.org/10.1016/j.rec.2012.04.016

High-density Lipoproteins After Acute Coronary Syndromes: Evaluation of Residual Risk

Lipoproteínas de alta densidad tras un síndrome coronario agudo, evaluando el riesgo residual

To the Editor,

The reduction of low-density lipoprotein (LDL) concentrations through treatment with statins decreases the risk of cardiovascular events. However, in the studies carried out to examine this question, this decrease was never greater than 37%.[1] The remaining or residual risk encompasses not only the traditional risk factors but also the additional modification of lipids. In this respect, the study of low high-density lipoprotein (HDL) levels as a predictor of cardiovascular events is an emerging area of interest,[2] even in patients with low LDL concentrations, as they play a fundamental role in acute coronary syndrome.[3] In contrast, there are very few predictors of reinfarction over the long term, especially with regard to biochemical variables, and those identified to date are

Table

Baseline Characteristics of the Patients According to the Serum High-density Lipoprotein Concentration Divided Into Tertiles

	T1 (HDL<33 mg/dL)	T2 (HDL 33–40 mg/dL)	T3 (HDL>40 mg/dL)	P
Baseline characteristics and cardiovascular risk factors				
Age, years	62.1 (13)	64.7 (12.3)	66.4 (13.1)	.007
Men	86.4	78.1	62.7	<.001
Smokers	54.4	43.8	37.3	.002
Ex-smokers	20.1	23.7	16	ns
Hypertension	59.2	53.8	52.7	ns
Dyslipidemia	31.4	44.4	42	.031
Diabetes mellitus	36.1	34.9	33.7	ns
PHIHD	20.1	19.5	25.4	ns
Laboratory values on admission				
Cholesterol, mg/dL	169.6 (37.2)	186.4 (39.2)	199.6 (38.8)	<.0001
LDL, mg/dL	112.5 (30.9)	123.3 (34.6)	126.5 (33.1)	<.0001
HDL, mg/dL	28.2 (3.7)	36.2 (2.1)	47.4 (7.8)	<.0001
Triglycerides, mg/dL	153.3 (87.3)	136.6 (57.9)	128.3 (48.6)	.003
Glucose, mg/dL	132.8 (55.3)	129.2 (53.9)	127.8 (52.9)	ns
Features of the acute coronary syndrome				
Killip class on admission	1.43 (0.77)	1.28 (0.61)	1.33 (0.69)	ns
Troponin, ng/L	66.3 (38.7)	55.2 (44.1)	51.9 (39.8)	.003
Lymphocytes, %	18.5 (13.9)	20.9 (8.5)	18.5 (8.6)	.065
Treatment at discharge				
Statin at discharge	42.3	56.8	55.4	.013
Antiplatelet agent at discharge	95.9	95.3	98.8	ns
Beta blocker	49.7	52.7	49.1	ns
Long-term outcome				
Reinfarction	21.3	15.4	14.8	.03
Death	22.5	24.3	27.2	ns

HDL, high-density lipoproteins; LDL, low-density lipoproteins; NS, not significant; PHIHD, personal history of ischemic heart disease.
All of the continuous variables are expressed as mean (standard deviation) and the categorical variables, as percentages.