Type D Personality Predicts Clinical Events After Myocardial Infarction, Above and Beyond Disease Severity and Depression

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Objective: To investigate the effect of Type D personality (high negative affectivity and social inhibition) on cardiac death and/or recurrent myocardial infarction (MI) in patients with acute MI, after adjustment for disease severity and depression. To explore the differential effect of Type D on early (≤ 6 months) versus late (> 6 months) events separately.

Method: Patients hospitalized for acute MI (N = 473) were recruited between May 2003 and May 2006. Patients were assessed on demographic and clinical variables and completed the Type D Personality Scale within the first week of hospital admission for acute MI; depression severity was assessed with the 17-item Hamilton Depression Rating Scale. The mean follow-up period was 1.8 years.

Results: There were 44 events attributable to cardiac death (n = 16) or recurrent MI (n = 28), with 26 early and 18 late events. Type D patients were at cumulative increased risk of death/recurrent MI compared with non–Type D patients (16.3% vs 7.8%; *P* = .012). Cardiac history, left ventricular ejection fraction, and use of statins were predictors of total and late death/recurrent MI, with statins showing a substantial protective effect. In addition, cardiac history and use of statins were significantly associated with early death/recurrent MI. Type D patients had a 2-fold increased risk of total death/recurrent MI after adjustment for disease severity and depression (HR = 2.23; 95% CI, 1.14–4.35; *P* = .019) and a more than 3-fold increased risk of late death/recurrent MI (HR = 3.57; 95% CI, 1.23–10.30; *P* = .019).

Conclusions: Type D was a strong predictor of adverse cardiac outcome after acute MI, above and beyond disease severity and depression severity, and the associated risk was similar to that of traditional cardiovascular risk factors. Type D may be an important psychosocial factor to assess in patients post-MI for risk stratification purposes.

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Worldwide, ample evidence exists demonstrating a strong and consistent association between psychological factors such as stress and both the development of coronary heart disease (CHD) and disease outcomes.[1,2] Furthermore, these factors are important contributors to health decrements in both initially healthy and chronic disease populations, including those with CHD and diabetes.[3] Efforts have been made to understand how these factors contribute to CHD onset, progression, and prognosis. Some of these efforts have been directed toward factors associated with personality,[4] such as hostility.

More recently, the Type D personality construct has become the focus of research attention. Type D personality is defined by the combination of 2 personality traits: the tendency to experience negative emotions (negative affectivity) and to inhibit self-expression in social interaction (social inhibition).[5] Hence, individuals with a Type D personality are inclined to experience emotional and interpersonal difficulties across time and situations. This personality construct can be easily assessed with the standardized and validated 14-item Type D Personality Scale (DS14), which measures negative affectivity as well as social inhibition[5]; Type D caseness is determined by a high score on both traits. The prevalence of Type D in cardiovascular patients largely ranges between 25%–35%. Recent studies have found that Type D is an important determinant of perceived health status[6–8] and clinical outcome[9–13] in patients with coronary artery disease (CAD), and the relative risk associated with Type D is comparable to that of left ventricular ejection fraction (LVEF).[14]

To date, these studies have only included selected patient groups, defined by percutaneous coronary intervention (PCI),[11] coronary artery bypass graft surgery (CABG),[7] and participation in cardiac rehabilitation programs.[9,10,12] The relative contribution of Type D to cardiovascular prognosis in a more general sample of myocardial infarction (MI) patients has not been investigated. In addition, there has been vigorous debate about whether Type D adds to the evidence concerning depression.[4]

We, therefore, prospectively evaluated the independent effect of Type D at time of the hospitalization for MI on cardiac death or recurrent MI. In addition, since early and late events post-MI may have a different pathological basis,[15] we explored the effect of Type D on early (≤ 6 months) and late (> 6 months) events separately. In these analyses, appropriate controls for factors such as CAD severity and depression known to affect post-MI prognosis were employed.

METHOD

Study Design and Patient Population

Patients hospitalized for acute MI (N = 473) were recruited between May 2003 and May 2006 from 4 teaching hospitals (Catharina Hospital, Eindhoven; St. Elisabeth Hospital, Tilburg; TweeSteden Hospital, Tilburg; and St. Anna Hospital, Geldrop) in The Netherlands. Inclusion

criteria were age > 30 and hospitalization due to acute MI. Criteria for diagnosis of MI were troponin I levels more than twice the upper limit, with typical ischemic symptoms (eg, chest pain) lasting for more than 10 minutes or electrocardiography (ECG) evidence of ST segment elevation or new pathological Q-waves. For patients without typical angina, the day of MI onset was identified as the day during hospitalization with peak troponin I levels > 1.0 and ECG evidence of ST segment elevation or new pathological Q-waves. Exclusion criteria were significant cognitive impairments (eg, dementia) and severe medical comorbidities that increased the likelihood of early death, such as malignant cancer, as verified by medical records and consulting the treating physician. The study protocol was approved by the institutional review boards of the participating hospitals, and written consent was obtained from all study participants.

Type D

Within the first week of hospital admission for acute MI (mean ± SD = 4.49 ± 3.11 days), patients completed the DS14.[5] Items on this scale are answered on a 5-point Likert scale from 0 to 4. The scale consists of two 7-item subscales assessing negative affectivity (eg, "I often feel unhappy"; "I am often irritated"; "I often find myself worrying about something") and social inhibition (eg, "I am a closed person"; "I would rather keep other people at a distance"; "I find it hard to start a conversation"). Patients were categorized as Type D using a standardized cut-off score ≥ 10 on both the negative affectivity and social inhibition subscales, following the protocol as previously established.[5] The DS14 is a valid and reliable scale with Cronbach α of 0.88/0.86 and a test-retest reliability over a 3-month period of $r = 0.72/0.82$ for the 2 subscales, respectively.[5] Type D has been found stable over an 18-month period in patients after acute MI.[16]

Depression Assessment

Patients were assessed on depression severity per the 17-item Hamilton Depression Rating Scale (HDRS),[17] which is an observer rating scale that is widely used to assess depression severity. The HDRS is a reliable and sensitive measure of post-MI depression severity[18] and has been previously used in research regarding the effect of pharmacologic[19] and cognitive-behavioral[20] treatment of post-MI depression. To account for the effect of depression as potential confounder in the relationship between Type D and cardiac prognosis, we used both the previously established cut-off of 17 as an index of depression severity[19] as well as continuous HDRS scores[20] in the present study.

Clinical Characteristics

Clinical variables associated with post-MI prognosis were obtained from the patients' medical records. These included cardiac history (prior MI, prior percutaneous [PCI] and/or surgical [CABG] revascularization), LVEF, multivessel disease, anterior location of index MI, invasive versus conservative treatment of index MI, participation in cardiac rehabilitation after index MI, smoking status (self-report), body mass index, hypertension (systolic blood pressure > 140, diastolic blood pressure > 90), hypercholesterolemia (total cholesterol > 6.50 mmol/L), systolic/diastolic blood pressure at the time of admission for index MI, and history of diabetes mellitus, renal insufficiency, chronic obstructive pulmonary disease, and arthritis. The following medications prescribed to the patient at discharge were also noted: β-blockers, angiotensin-converting enzyme inhibitors, anticoagulants, statins, diuretics, aspirin, and selective serotonin reuptake inhibitors (SSRIs). Demographic variables included age, gender, marital status, and classified educational level.

Endpoint

The primary endpoint was a composite of cardiac death and/or recurrent MI, as verified by medical records. Secondary endpoints were separated into early (≤ 6 months) and late events (> 6 months). Criteria for diagnosis of MI were those used for inclusion in the study. The mean follow-up period was 1.8 years (SD = 0.8 years), and follow-up data were complete for all patients (100%). There is variability in length of follow-up because the last follow-up on all patients was done at set points in time ("waves" of follow-up), while patients were enrolled continuously as a function of acute MI admission to hospital.

Statistical Analysis

Discrete variables were compared with the χ^2 test and are presented as numbers and percentages. Continuous variables were compared with the Student *t* test and are presented as means ± SDs. Univariate and multivariate Cox proportional hazard regression analyses (enter procedure) were performed to investigate the impact of Type D on cardiac death and recurrent MI at follow-up. Univariate analyses were used to test for the potentially confounding effect of biomedical and demographic factors on outcome. If significant at $P < .05$, the variables were included together in a regression model to remove redundant covariates (if any). Subsequently, the significant confounders were added as covariates to the multivariate analyses for death/MI. Depression and LVEF were included in all prediction models; LVEF was included to adjust for disease severity, and depression was included, since it is a known psychosocial risk factor, ruling out the possibility that the effect of Type D on outcome could be due to more severe cardiac disease or depression. Since 92 patients had no echocardiography, data imputation based on LVEF means was used to fill in missing LVEF data. Deleting cases based on missing data is not preferable, since missing data cannot safely be assumed to reflect randomness, and, therefore, deletion can introduce substantial bias into the study. Moreover, the loss in sample size can appreciably diminish the statistical power of the analysis. The cumulative incidence of death/recurrent MI in Type D patients was estimated according to the Kaplan-Meier method, comparing differences between groups with the log rank test. The zero time point indicates the time of hospitalization. A *P* value < .05 was used for all tests to indicate statistical significance.

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Table 1. Demographic and Clinical Predictors of Death and/or Recurrent MI (univariate analyses)[a]

Characteristic	All Patients (N = 466)	Death/MI (n = 44)	Event-Free (n = 422)	HR	95% CI	*P*
Age, mean (SD), y	59 (12)	64 (14)	59 (11)	1.04	1.00–1.06	.012
Sex, female	100 (22)	9 (21)	91 (22)	0.94	0.45–1.96	.871
Partner	382 (82)	31 (74)	351 (83)	0.58	0.29–1.15	.119
Educational level, high	259 (56)	20 (48)	239 (57)	0.76	0.41–1.39	.367
Disease severity						
Cardiac history[b]	76 (16)	19 (43)	57 (14)	4.25	2.34–7.72	<.0001
LVEF percentage, mean (SD)	50 (9)	46 (11)	50 (9)	0.96	0.94–0.99	.007
Multivessel disease	151 (38)	15 (46)	136 (38)	1.38	0.70–2.74	.359
Anterior MI location	172 (41)	18 (49)	154 (40)	1.34	0.70–2.55	.374
Comorbidity						
Diabetes mellitus	67 (14)	11 (25)	56 (13)	2.00	1.01–3.96	.046
Renal insufficiency	23 (5)	4 (9)	19 (5)	2.15	0.77–6.00	.146
COPD	46 (10)	6 (14)	40 (10)	1.44	0.61–3.40	.408
Arthritis	37 (8)	5 (11)	32 (8)	1.63	0.64–4.15	.302
Invasive treatment[c]	284 (61)	20 (46)	264 (63)	0.54	0.30–0.97	.038
Cardiac rehabilitation	283 (68)	22 (54)	262 (69)	0.60	0.33–1.12	.107
Medication use						
β-blockers	399 (86)	36 (82)	363 (86)	0.73	0.34–1.58	.425
ACE-inhibitors	173 (37)	17 (39)	156 (37)	1.03	0.56–1.88	.927
Anticoagulants	387 (83)	41 (93)	346 (82)	2.93	0.91–9.45	.073
Statins	422 (91)	32 (73)	390 (93)	0.24	0.13–0.48	<.0001
Aspirin	385 (83)	30 (68)	355 (85)	0.40	0.21–0.75	.004
Diuretics	89 (19)	20 (46)	69 (17)	3.76	2.08–6.81	<.0001
Smoking	179 (39)	18 (42)	161 (38)	1.12	0.61–2.06	.711
BMI, mean (SD), kg/m^2	27 (4)	26 (5)	27 (4)	0.92	0.84–1.01	.093
Hypertension	129 (29)	10 (23)	119 (30)	0.69	0.34–1.40	.304
Hypercholesterolemia	55 (12)	3 (8)	52 (13)	0.58	0.18–1.87	.359
Cardiac function						
Systolic BP, mean (SD)	140 (28)	135 (24)	141 (29)	0.99	0.98–1.00	.138
Diastolic BP, mean (SD)	82 (17)	79 (16)	82 (17)	0.98	0.97–1.00	.094
Depression						
HDRS score, mean (SD)	5.8 (5.5)	7.7 (6.6)	5.6 (5.3)	1.05	1.01–1.09	.029
HDRS score ≥ 17	25 (5)	5 (11)	20 (5)	2.27	0.89–5.76	.085
SSRIs	58 (13)	11 (25)	47 (11)	2.37	1.20–4.72	.013
Type D personality	92 (20)	15 (34)	77 (18)	2.18	1.17–4.07	.014

[a]Values are expressed as n (%) of patients unless otherwise indicated.
[b]Myocardial infarction, percutaneous coronary intervention, or coronary artery bypass graft surgery prior to the index MI.
[c]Percutaneous coronary intervention or coronary artery bypass graft surgery.
Abbreviations: ACE = angiotensin-converting enzyme, BMI = body mass index, BP = blood pressure, COPD = chronic obstructive pulmonary disease, HDRS = 17-item Hamilton Depression Rating Scale, LVEF = left ventricular ejection fraction, MI = myocardial infarction, SSRI = selective serotonin reuptake inhibitor.

Hazard ratios (HRs) with 95% confidence intervals (CIs) are reported. All statistical analyses were performed using SPSS version 14.0 (SPSS Inc, Chicago, Illinois).

RESULTS

Of the original 473 patients, 7 had no Type D assessment, leaving 466 patients (99%) to be included in final analyses. Of these 466 patients, 92 (19.7%) were classified as Type D.

Clinical Predictors of Death/MI

There were 44 events attributable to cardiac death (n = 16) or recurrent MI (n = 28), with 26 early (≤ 6 months) and 18 late (> 6 months) events. Patient characteristics stratified by death/recurrent MI are presented in Table 1 (columns 1–3). Patients experiencing a clinical event were older and more likely to have a previous cardiac history, to be treated with SSRIs and diuretics, and to have diabetes and lower mean LVEF than event-free patients. These patients were also less likely to be invasively treated at index MI, and to be treated with statins and aspirin, than event-free patients.

Age, cardiac history, LVEF, diabetes, invasive treatment, statins, aspirin, diuretics, and SSRIs were significant predictors for death/recurrent MI in univariate analyses (Table 1, columns 4–6). When entering all significant predictors into a multivariate analysis, only cardiac history, statins, and SSRIs remained significant. Hence, we adjusted for these covariates—in addition to LVEF and depression—in multivariate analyses.

Type D and Death/Recurrent MI (Univariate Analyses)

Type D patients were at a cumulative increased risk of total death/recurrent MI at 1.8 years compared with non–Type D patients (16.3% vs 7.8%; *P* = .012; Figure 1). No significant difference was found in the incidence of early death/recurrent MI for Type D patients versus non–Type D patients (8.7% vs 4.8%; *P* = .146); however, the occurrence of late death/recurrent MI was significantly higher in patients with versus without Type D (7.6% % vs 2.9%; *P* = .037).

Independent Predictors of Death/Recurrent MI

In multivariable analyses, Type D was found to be an independent predictor of death/recurrent MI and was associated

Figure 1. Cumulative Survival Stratified by Type D Personality (N = 466)

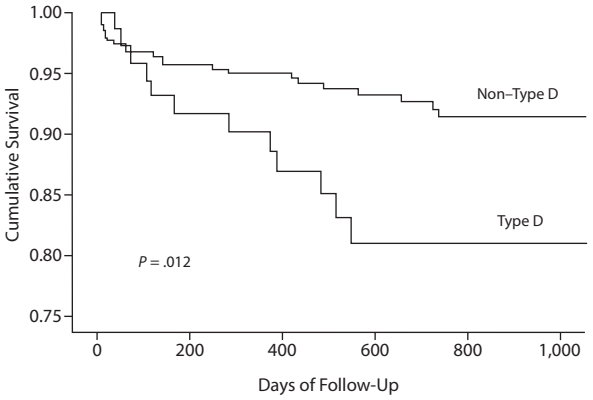

Table 2. Predictors of Total Death or Recurrent MI (n = 44; adjusted analyses)[a]

	Model 1			Model 2		
Predictor Variable	HR	95% CI	*P*	HR	95% CI	*P*
Type D personality[b]	2.26	1.19–4.29	.013	2.23	1.14–4.35	.019
Depression[c]	1.80	0.69–4.69	.228	1.37	0.50–3.72	.543
Cardiac history[d]	3.37	1.78–6.39	< .0001	3.50	1.83–6.64	< .0001
Age	1.02	0.99–1.05	.155	1.01	0.98–1.04	.603
LVEF percentage	0.97	0.95–1.00	.056	0.97	0.94–1.00	.024
Statins	…	…	…	0.28	0.14–0.56	< .0001
SSRIs	…	…	…	1.85	0.87–3.94	.112

[a]Enter procedure.
[b]Per the Type D Personality Scale (DS14).
[c]Per the 17-item Hamilton Depression Rating Scale (HDRS score ≥ 17).
[d]Myocardial infarction, percutaneous coronary intervention, or coronary artery bypass graft surgery prior to the index MI.
Abbreviations: LVEF = left ventricular ejection fraction, MI = myocardial infarction, SSRI = selective serotonin reuptake inhibitor.
Symbol: … = not applicable.

with a more than 2-fold increased risk (HR = 2.26; 95% CI, 1.19–4.29), adjusting for depression, age, cardiac history, and LVEF (Table 2; Model 1). When adding statins and SSRIs to the model, Type D remained predictive of death/recurrent MI (Table 2; Model 2). While Type D was not significantly associated with early events (HR = 1.70; 95% CI, 0.71–4.10), it was associated with a 3-fold increased risk of late events (HR = 3.57; 95% CI, 1.23–10.30), adjusting for disease severity and other confounders (Table 3). In these analyses, cardiac history, LVEF, and use of statins were also independent predictors of death/MI, with statins showing a substantial protective effect.

Depression severity was predictive of cardiac events in univariate analysis (Table 1), with continuous HDRS scores showing a trend toward late death/recurrent MI (HR = 1.07; 95% CI, 0.99–1.15; *P* = .078) in multivariate analysis. Type D remained an independent predictor of both total (Model 1: HR = 2.13; 95% CI, 1.11–4.09; *P* = .013; Model 2: HR = 2.19; 95% CI, 1.11–4.35; *P* = .024) and late death/MI (HR = 3.19; 95% CI, 1.12–9.08; *P* = .030) when entering continuous depression scores into the multivariate model.

Table 3. Predictors of Early (≤ 6 months) and Late (> 6 months) Death or Recurrent MI (adjusted analyses)[a]

	Early Death/MI (n = 26)			Late Death/MI (n = 18)		
Predictor Variable	HR	95% CI	*P*	HR	95% CI	*P*
Type D personality[b]	1.70	0.71–4.10	.237	3.57	1.23–10.30	.019
Depression[c]	0.86	0.19–3.92	.840	2.31	0.58–9.17	.235
Cardiac history[d]	3.34	1.44–7.73	.005	3.87	1.42–10.50	.008
Age	0.99	0.96–1.03	.896	1.02	0.98–1.07	.319
LVEF, %	0.97	0.94–1.01	.143	0.96	0.92–1.00	.064
Statins	0.28	0.11–0.71	.007	0.24	0.08–0.72	.011
SSRIs	2.42	0.94–6.24	.067	1.22	0.35–4.31	.753

[a]Enter procedure.
[b]Per the Type D Personality Scale (DS14).
[c]Per the 17-item Hamilton Depression Rating Scale (HDRS score ≥ 17).
[d]Myocardial infarction, percutaneous coronary intervention, or coronary artery bypass graft surgery prior to the index MI.
Abbreviations: LVEF = left ventricular ejection fraction, MI = myocardial infarction, SSRI = selective serotonin reuptake inhibitor.

Exploratory analyses concerning the increased risk associated with SSRI use revealed that Type D patients were more likely to be taking SSRIs than non–Type D patients (26% vs 10%).

DISCUSSION

This is the first prospective study to examine the effect of Type D personality on cardiac outcomes in patients after acute MI. Type D patients were at a cumulative increased risk of death or recurrent MI compared with non–Type D patients. Cardiac history, LVEF, and use of statins were predictors of death/recurrent MI, with statins showing a substantial protective effect. Type D patients had a more than 2-fold increased risk of total death/recurrent MI after adjustment for disease severity and a 3-fold increased risk of late death/recurrent MI.

The present findings, while consistent with those from previous studies showing that Type D independently predicts medical outcome,[9–13] expand upon the previous work, since those studies were based on more specific patient groups, thereby limiting the generalizability of the findings. Furthermore, the current study took a more nuanced look by distinguishing between early and late events. Only cardiac history and use of statins were associated with early death/recurrent MI, while Type D at the time of the index MI carried a 3-fold increased risk of late events. The fact that the effect of Type D was found for late events rather than early events may imply that Type D exerts a more chronic or persistent effect on the cardiovascular processes that underlie prognosis.

The Type D personality construct was developed in the 1990s to reflect a general vulnerability factor present in a subgroup of cardiac patients.[21] In the present study, Type D remained an independent predictor of cardiac events adjusting for depression severity and use of SSRIs. This finding confirms previous studies indicating that Type D is more than just a marker of depression. A recent 5-year follow-up study showed that Type D had unique prognostic value beyond that of depressive symptoms.[22] These findings do

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not refute depression as an important prognostic factor. In fact, depression severity was predictive of cardiac events in univariate analysis with continuous HDRS scores showing a trend toward late cardiac events in the multivariate model.

Also of note was a finding of greater than a 2-fold increased risk of death/recurrent MI associated with SSRI use. This finding was in univariate analyses and did not persist for either early or late events in multivariate analyses, with Type D accounting for its effect. Despite this, the finding is worth further consideration. SSRIs have largely been thought of as the antidepressant of choice after acute coronary syndrome, and, indeed, the Sertraline Antidepressant Heart Attack Randomized Trial (SADHART) Study found the cardiovascular effects of this class of agents benign in a study of 340 postacute coronary syndrome patients.[19] The finding of risk associated with these agents, however, is not the first,[23] and given the growing use of these agents in cardiac populations, further study is warranted.

We found a considerable protective effect of statins for adverse clinical events; 91% of patients were treated with this drug. These results give support to statin use as an effective therapy for improving cardiac prognosis in post-MI patients, but should be interpreted with caution given the low prevalence of those who were not taking these drugs. Of note, despite appropriate cholesterol management, Type D still emerged as a strong independent predictor of cardiac death and nonfatal MI. This raises questions as to the mechanisms by which Type D exerts its effect on post-MI prognosis.

There are several potential mechanisms that may help to explain the adverse effect of Type D personality on cardiac prognosis. One possible pathway is behavioral.[4] For example, in one study, Type D patients with chronic heart failure were less likely to consult clinical staff, even though their symptom reports were greater than those of other patients.[24] This is not surprising, given that Type D, by definition, includes a high degree of social inhibition—an unwillingness to or discomfort with utilizing available social and related network resources when in need. Behavioral factors may also influence adherence to medical recommendations, including the taking of prescription medications, and this may underlie the influence of Type D on prognosis even with patients prescribed statin medications. A second possible pathway involves immune activation and hypothalamic-pituitary-adrenocortical axis dysregulation,[25] as Type D patients evidence heightened levels of proinflammatory cytokines in heart failure[26,27] and disturbances in cortisol secretion after acute coronary syndrome.[28] A recent study also provides evidence of genetic influences in the determination of Type D,[29] raising the possibility that similar genetic pathways may be involved in the personality construct and the processes involved in the development and progression of CAD. Further research regarding the behavioral and pathophysiologic mechanisms responsible for the relationship between Type D and prognosis is warranted.

Our results indicate that subgroups of cardiac patients with a particular personality profile may not respond adequately to treatment, especially in the long run, and that for this subgroup, some form of psychosocial intervention may be warranted. Studies indicate that Type D is a stable construct not confounded by variability in mood status and/or disease severity.[16] This finding, however, does not imply that the patient's level of distress cannot be modified. It is the nature of the Type D construct that the confluence of emotional distress and social inhibition impair a person's ability to cope adequately with the stresses of daily life.[30] These patients could, therefore, likely benefit from psychological interventions that are targeted to improving their coping skills. Such interventions could, thereby, decrease the acute and chronic stress that patients experience with potential "downwind" effects on sympathetic and immune activity. Furthermore, these interventions could improve the patient's disease management skills. The extent to which these processes underlie the link between Type D and prognosis underscore the potential of these treatments. Future intervention trials are, therefore, needed in order to examine how the deleterious effects of Type D can be reduced.

Some limitations of the current study should be noted. First, the low number of women (22%) limits the generalizability of the results. Furthermore, patients were relatively healthy, with a mean LVEF of 50%. Third, prior to setting up a multivariate model to predict events, we evaluated a large number of predictors in univariate analysis. Although most of the variables have been associated with adverse cardiac outcome in previous studies, this procedure may have led to overfitting of our regression model. Finally, we had no information on the overall response rate of the study. However, we were able to look into a subsample of patients (n = 63). Of the 63 patients who met the inclusion criteria, 46 gave informed consent, leaving a response rate of 73%. The retention rate of this study has been reported. Despite these limitations, the present results represent an important contribution to the growing literature that demonstrates the importance of psychosocial factors for clinical outcome in acute MI patients. This study included an unselected group of acute MI patients and not, like studies in the past, specific patient subgroups (eg, PCI or CABG patients).[11,13] Moreover, this was a multicenter study, making generalization of our results to MI patients more justified. Furthermore, we evaluated a broad spectrum of possible confounding factors, including depression, disease severity, medical comorbidity, risk factors, and medication use. Finally, the prognostic effect of Type D in this study was measured both for early and late events.

In summary, Type D personality was identified as an independent predictor of adverse cardiac outcome in acute MI patients, above and beyond the effects of disease severity, depression, and several other related factors. The risk associated with Type D was similar to that of traditional cardiovascular risk factors. The results from this study indicate the need for future research directed to the identification of the underlying pathophysiologic processes by which Type D contributes to prognosis after MI and to the testing of interventions to alleviate the associated risk.

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