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Short communication

Associations of type-D personality and depression with somatic health in myocardial infarction patients

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Abstract

Objective: Depression and type-D personality have both been associated with worse cardiac prognosis in myocardial infarction (MI) patients. There is some debate, however, that the association between depression and cardiac prognosis is confounded by somatic health. We therefore compared to what extent depression and type-D personality are associated with somatic health. **Method:** We studied the association of depressive disorder and type-D with baseline somatic health in a subsample of 1205 post-MI patients from the Myocardial Infarction and Depression Intervention Trial study. Depressive disorder was assessed according to ICD-10 criteria with the Composite International Diagnostic Interview during the post-MI year and type-D with the DS14 at 1-year follow-up. Somatic health was operationalized by baseline LVEF, Charlson Comorbidity Index, previous MI, and CABG or

PTCA during hospital admission. **Results:** Prevalence rates were 17.1% for post-MI depression and 18.7% for type-D. After controlling for potential confounders, post-MI depression was associated with poorer baseline LVEF [odds ratio (OR)=3.17, 95% confidence interval (CI)=2.28–4.41] and greater comorbidity (OR=1.46, 95% CI=1.02–2.09), whereas type-D personality was not (LVEF: OR=1.31, 95% CI=0.93–1.87; comorbidity: OR=0.92, 95% CI=0.63–1.35). **Conclusion:** Post-MI depression during the post-MI year is more related to somatic health than type-D personality at 12 months post-MI and, specifically, somatic symptoms of depression. Confounding of cardiovascular effects of psychological distress by poor somatic health status is thus more likely to occur in post-MI depression than in type-D personality. © 2007 Elsevier Inc. All rights reserved.

Keywords: Depression; Myocardial infarction; Type-D

Introduction

Following myocardial infarction (MI), depression and type-D have both been associated with adverse cardiac prognosis [1,2]. However, in most studies that controlled for factors related to MI severity, these effects were attenuated, resulting in nonsignificant associations in some. In a meta-analysis we conducted some 2 years ago [1], we found that post-MI depression was associated with a 2- to 2.5-fold increased risk of poor outcomes (mortality; nonfatal cardiac

events). We identified 11 studies that included a multivariate adjustment by baseline MI severity or its consequences: in 4 studies, the univariate effects remained; in 4 studies, they were reduced; and in 3 studies, they were no longer statistically significant. More recently, Nicholson et al. [3] reported that half of the association between depression and progression of heart disease was explained by LVEF. Some authors have therefore argued that depression may be confounded by somatic health [4,5].

In several studies, the association between post-MI depression and LVEF has been examined, mostly using LVEF as a dichotomized variable. Four out of six studies (i.e., Refs. [6–9]) reported a higher risk of depression at lower levels of LVEF, approaching statistical significance in three studies (i.e., Refs. [7–9]). Two other studies (i.e., Refs. [10,11]) did

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not find an association. More recently, we have shown in a large sample that severity of post-MI ventricular dysfunction, operationalized in four categories, has a linear relation with severity of depressive symptoms during hospitalization and with the risk of major depression in the year following MI [12]. On the other hand, several studies have failed to demonstrate an association between depression and CAD severity [13–15].

Somatic symptoms of depression, such as fatigue and not being able to work, may be related to health status, whereas cognitive symptoms of depression, such as guilt and poor self-confidence, may be relatively independent [16]. To our knowledge, the possibility that type-D personality, a combination of negative affectivity and social inhibition [17], is confounded by somatic health is not yet studied. Since depression and type-D are both predictive of future cardiac events post-MI, we compared both risk factors on baseline somatic health and evaluated the associations of post-MI depression and type-D with left ventricular dysfunction, heart failure, and comorbidity in the same sample of patients. We hypothesized that type-D is less associated with somatic health, as it is defined as a trait rather than a state and as it does not contain somatic symptoms whereas depression does.

Methods

This study is a preplanned substudy of the Myocardial Infarction and Depression Intervention Trial (MIND-IT). Inclusion and exclusion criteria of MIND-IT have been described previously [18]. In short, we recruited consecutive patients (September 1999–November 2002), hospitalized for acute MI, in 10 hospitals in The Netherlands. Patients were enrolled if they met WHO MONICA criteria [19] for definite MI: increased cardiac enzymes as well as electrocardiographic changes and/or chest pain. Exclusion criteria were the occurrence of MI while the patient was hospitalized for another reason, inability to participate in study procedures, having received psychiatric treatment for depression, and participation in another clinical trial. All patients gave written informed consent before enrolment.

This substudy was conducted in 9 of the 10 hospitals that participated in the MIND-IT, representing 2083 of the 2177 patients enrolled in the MIND-IT. Due to limitations in funding, only the 1656 patients who were enrolled before February 1, 2002, were asked to complete the type-D assessment; 1267 returned the questionnaire. Of these patients, 14 had an incomplete depression follow-up and 49 had incomplete data on the type-D questionnaire (1 patient had missing data on both). This resulted in a sample of 1205 subjects on which the analyses were based.

We collected data on medical history, clinical variables, and medication use during hospitalization for the MI index. Medical comorbidity was assessed with a modified version of the Charlson Comorbidity Index following the sugges-

tions by Watkins et al. [20], such as excluding MI and conditions with a low prevalence as comorbid conditions. Scores were based on the presence of nine conditions and transformed into a four-level ordinal scale on which Categories 0, 1, 2, and 3 correspond to index scores of 0, 1–2, 3–4, and ≥ 5 . For the present analyses, we used a dichotomization of this variable, indicating at least three comorbid conditions. LVEF was measured by either echocardiography or radionuclide ventriculography and categorized as ≥ 45 or < 45 .

Patients were screened for depressive symptoms during hospitalization and at 3, 6, 9, and 12 months post-MI, using the Beck Depression Inventory (BDI) [21]. Those with depressive symptoms (i.e., BDI score ≥ 10) had a psychiatric evaluation using the WHO Composite International Diagnostic Interview (CIDI), auto version 2.1 [22]. The first CIDI interviews were performed not earlier than 3 months post-MI to allow natural recovery of depressive symptoms. In this article, we used the continuous BDI scores as a gauge for the presence and severity of depressive symptoms and the CIDI diagnosis for an estimation of the presence of depressive episode on at least one of the follow-up points. We assessed type-D personality at approximately 12 months post-MI with the DS14 [17]. The DS14 consists of 14 items with a response scale ranging from 0 (*false*) to 4 (*true*), of which 7 items refer to negative affectivity and 7 to social inhibition. The presence of type-D personality is defined as having a score of at least 10 points on both subscales.

We examined the associations of depression and type-D with five indicators of poor somatic health (i.e., Charlson comorbidity score > 2 , LVEF $< 45\%$, previous MI, previous CABG, previous PTCA) by means of odds ratios (ORs) and their 95% confidence intervals (CIs). For these analyses, we used the unadjusted ORs and ORs adjusted for age, sex, BMI, and current smoking. We used the *Z* test to evaluate whether the associations with these parameters were equal for type-D and depression. In addition, we analyzed the correlations between type-D dimensions (i.e., social inhibition and negative affect) and depression dimensions (somatic, cognitive, and appetitive symptoms) and the associations between depression dimensions and somatic health.

To evaluate the strength and stability of the associations between LVEF and the repeated BDI assessment, we applied a repeated measurements analysis using the SPSS mixed-model approach [23]. This analysis allows evaluating differential associations over time and makes optimal use of the available data at the repeated assessments that are clustered within subjects. For this analysis, we used the BDI scores as continuous variables.

Results

Table 1 shows the baseline characteristics of patients from the MIND-IT. Patients included in the present study tended to have a better somatic health status than those

Table 1
Comparison of total MIND-IT sample and patients included in the present study

	Total sample (N=2177)	Excluded from the present study (n=972)	Included in the present study (n=1205)	P
Demographic				
Age, mean (S.D.)	61.2 (11.9)	61.7 (12.6)	60.9 (11.4)	.13
Female sex (%)	22.6	23.9	21.5	.15
BMI>25 (%)	62.8	62.7	63.0	.89
Somatic health (%)				
LVEF<45	25.9	28.8	23.8	.01
Heart failure	12.6	14.0	11.5	.09
Killip class>1	11.2	12.6	10.1	.07
Charlson>2	10.3	11.5	9.4	.11
Previous MI	14.1	15.3	13.1	.14
PTCA during hospitalization	38.4	33.8	42.0	<.001
CABG during hospitalization	5.1	5.4	4.9	.63
Cardiac risk factors (%)				
Dyslipidemia	75.8	74.0	77.2	.09
Smoking	47.7	47.9	47.6	.87
Hypertension	33.9	34.8	33.1	.39
Diabetes	12.7	14.4	11.5	.03
Family history of CAD	45.1	44.3	45.8	.48

excluded. This is understandable from the perspective that patients had to undergo repeated depression assessments throughout the post-MI year and to fill in the DS14 at 12 months post-MI. The most severely ill were probably the ones that dropped out in the year following MI.

Of the 1205 patients, 206 patients had a post-MI depressive disorder (17.1%) and 224 were identified as having type-D personality (18.6%). We first evaluated the associations between depression dimensions and type-D personality dimensions by Spearman correlations. The highest correlations, although still rather modest ($r=.33$), were found between negative affect and the somatic-affective symptoms. No significant correlations were found between type-D and appetitive symptoms, and only very small correlations were seen between social inhibition and the depression dimensions ($r<.2$). The prevalence of post-MI depression during the post-MI year was significantly higher for patients with poor LVEF, heart failure, and having had a PTCA during hospitalization for the MI index (Table 2). For this latter group, the prevalence of type-D personality at 12 months post-MI was also increased. Comparison tests (Z scores) indicated that depression was more strongly associated with LVEF<45% ($P<.001$) as compared to type-D personality. Comparable results were found in the analyses

controlling for age, sex, BMI, and current smoking (Table 3). Of interest, in these analyses, the association between depression and medical comorbidity became significant.

To further explore the associations of post-MI depression with LVEF and comorbidity, we compared the associations for post-MI depressions with early (within 3 months post-MI) versus late onset (after 3 months post-MI). The risk of early-onset post-MI depression was significantly increased for patients with LVEF<45 (OR=2.48, 95% CI=1.75–3.53) but not for patients with somatic comorbidity (OR=1.26, 95% CI=0.87–1.83). Similarly, the risk of late-onset post-MI depression is significantly increased for patients with LVEF<45% (OR=3.34, 95% CI=1.68–6.64) but not for patients with somatic comorbidity (OR=0.91, 95% CI=0.41–2.01). Comparison tests showed no significantly differential associations between both types of post-MI depression and the two indicators of somatic health (LVEF<45: $Z=-0.76$, $P=.45$; comorbidity: $Z=0.73$, $P=.47$).

We explored whether specific symptom dimensions of depression were responsible for the associations with LVEF and comorbidity. For this goal, we used standardized factor scores (mean=0; S.D.=1) developed in a previous paper [16] distinguishing between cognitive, somatic, and appetitive symptoms based on the BDI assessed during

Table 2
Associations of somatic health with type-D and depression

	Post-MI depression			Type-D personality			Equality test	
	– (%)	+ (%)	OR (95% CI)	– (%)	+ (%)	OR (95% CI)	Z	P
LVEF<45	20.1	41.8	2.84 (2.05–3.94)	23.0	27.8	1.29 (0.92–1.76)	3.29	<.001
Charlson >2	22.7	26.0	1.20 (0.85–1.69)	24.0	19.8	0.78 (0.55–1.12)	1.72	.08
Previous MI	13.0	13.2	1.01 (0.65–1.58)	13.3	12.1	0.90 (0.58–1.40)	0.36	.72
PTCA	39.9	52.4	1.66 (1.23–2.25)	40.4	49.1	1.42 (1.06–1.90)	0.73	.46
CABG	5.1	3.9	0.75 (0.35–1.60)	5.1	4.0	0.78 (0.38–1.61)	–0.07	.94

Table 3
Associations of somatic health with type-D and depression controlling for potential confounders

	Post-MI depression, OR (95% CI)	Type-D personality, OR (95% CI)	Equality test	
			Z	P
LVEF<45	3.17 (2.28–4.41)	1.31 (0.93–1.87)	3.57	<.001
Charlson=2	1.46 (1.02–2.09)	0.92 (0.63–1.35)	1.72	.08
Previous MI	1.32 (0.86–2.03)	1.03 (0.66–1.62)	0.79	.64
PTCA	1.54 (1.14–2.08)	1.36 (1.01–1.83)	0.58	.56
CABG	0.83 (0.40–1.71)	0.78 (0.37–1.62)	0.12	.91

hospitalization. In Table 4, it is shown that while the somatic and appetitive symptoms of depression were associated with LVEF and comorbidity, the cognitive symptoms of depression were not.

Finally, we found that the difference in mean level of depressive symptoms (BDI) between patients with poor LVEF function and those with preserved LVEF function was stable throughout the first year post-MI (Fig. 1). Accordingly, a mixed-model analysis (using the repeatedly assessed BDI score as dependent variable and LVEF, timing of assessment, and LVEF×Timing as predictors) yielded a significant main effect of LVEF on BDI score of 0.91 (S.E.=0.17; $F=11.7$, $P=.001$) and a significant time effect ($F=21.6$, $P<.001$) but a nonsignificant interaction effect ($P=.18$).

Discussion

Using well-established methods to assess depression and type-D personality in a large sample of MI patients, our primary finding is that post-MI depression during the post-MI year is more strongly related to poor somatic status as compared to type-D personality assessed at 12 months post-MI. This finding is consistent with our hypothesis and suggests that confounding of the cardiovascular effects of psychological distress by poor somatic health status is more likely to occur in post-MI depression than in type-D personality. With respect to the magnitude of the association between depression and LVEF, we observed that poor LVEF was associated with a twofold increased rate of major depression and with about 1 point higher BDI score throughout the post-MI year.

Among the limitations of this study, we acknowledge that a minority of the patients received antidepressant treatment in the framework of the randomized trial. However, outcomes of the two interventions (usual care

vs. active antidepressant care) did not differ either in terms of depression status at 18 months post-MI or in terms of new cardiovascular events. Second, as the assessment of depression and type-D personality occurred at different times, there is a possibility that our findings merely reflect the unsurprising phenomenon that distress detected closer to the MI is more associated with somatic health. However, this possibility cannot explain our findings as, for example, LVEF is associated with early-onset post-MI depression (within 3 months) and has an even stronger association with late-onset post-MI depression (after 3 months). Moreover, the association between LVEF and depressive symptoms during the post-MI year remained stable across the different assessment points. In a previous study in 121 coronary artery patients, it was found that the type-D construct had a temporal stability as high as 0.82 for the negative affectivity and 0.72 for the social inhibition subscale over a 3-month period, which was higher than for two depression scales [17]. Therefore, we can conclude that type-D personality is not so much dependent on changes in mood status but rather reflects two stable personality traits: negative affectivity and social inhibition. Third, our findings may not apply to all MI patients since our data were based on an intervention trial that may have resulted in an underrepresentation of more severely ill MI patients. This underrepresentation was even stronger in our current sample, as patients had to undergo repeated psychiatric screenings throughout the post-MI year.

The associations of poor somatic health with depression were observed with respect to parameters related to heart failure and somatic comorbidity and, specifically, to left ventricular dysfunction. Moreover, the associations between poor somatic status and the presence of depression during the post-MI year were not restricted to early-onset post-MI depression but seemed to hold for the whole year following the MI. In fact, the effects of

Table 4
Associations between cognitive, somatic, and appetitive symptoms of depression (BDI during hospitalization) with LVEF and comorbidity (unstandardized β coefficients)

	Somatic symptoms		Cognitive symptoms		Appetitive symptoms	
	β (95% CI)	P	β (95% CI)	P	β (95% CI)	P
LVEF<45	0.20 (0.06 to 0.33)	<.001	-0.03 (-0.14 to 0.09)	.66	0.25 (0.12 to 0.38)	<.001
Charlson=2	0.26 (0.14 to 0.39)	<.001	0.07 (-0.04 to 0.18)	.21	0.28 (0.16 to 0.41)	<.001

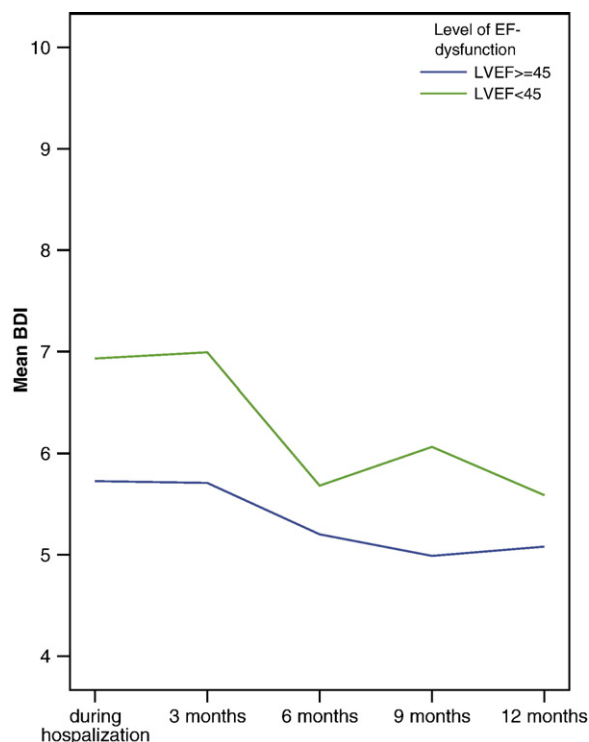


Fig. 1. Depressive symptoms (BDI) during the post-MI year as a function of LVEF.

poor LVEF on BDI scores indicate a relatively minor but consistent effect. This finding seems to suggest that, if any confounding is present, it is not so much the severity of the MI itself but rather the severity of the underlying coronary artery disease or somatic comorbidities that may be responsible. Studies evaluating cardiac effects of depression following MI should take this into account not only by controlling for severity of the MI itself but also by controlling for underlying cardiac disease and comorbidity. We can only speculate about potential mechanisms that account for the observed associations. Several of the hypothesized physiologic mechanisms that explain the cardiac effects of depression may be reversed: for instance, increased cytokine levels resulting from left ventricular dysfunction, such as interleukin-1, interleukin-6, and tumor necrosis factor- α , may play a role in the genesis of post-MI depression. Also, the social and physical limitations that arise from coronary artery disease or somatic comorbidities may be intertwined with post-MI depression.

Given the intrinsic difficulty of diagnosing depression in MI patients due to an extensive overlap of somatic symptoms, it is possible that symptoms attributable to depression in patients with severe LV dysfunction or somatic comorbidities may be, in fact, secondary to heart failure or other conditions. Depression may therefore be overestimated in this population. The presence of type-D personality following an MI seems to be less a function of somatic health. This suggests that a different form of

psychological distress is captured by the type-D personality construct that is relatively independent of somatic health but still predictive of its long-term consequences. Future studies should therefore focus on ways to reduce (aspects of) type-D personality characteristics, including negative affectivity and social inhibition.

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