Prediction of depressive disorder following myocardial infarction


Published in:
International Journal of Cardiology

Publication date:
2006

Citation for published version (APA):

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Prediction of depressive disorder following myocardial infarction
Data from the Myocardial Infarction and Depression–Intervention Trial (MIND-IT)

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Received 25 February 2005; received in revised form 24 May 2005; accepted 28 May 2005
Available online 5 July 2005

Abstract

Background: Depression following myocardial infarction (MI) is associated with complicated cardiac rehabilitation, non-compliance and poor prognosis. Whether depression following MI can be predicted from variables routinely assessed during hospitalization for MI is unknown.

Methods: Using data from the Myocardial Infarction and Depression–Intervention Trial (MIND-IT), we identified 2177 MI patients (mean age 63 years; 23% female). Patients were randomly divided into a derivation and a validation sample. In the derivation sample, we analyzed variables potentially associated with the development of post-MI depressive disorder, which were tested in the validation sample.

Results: In the year following MI, 18.5% suffered from depressive disorder (ICD-10 criteria). In a multivariate model, factors associated with depression were younger age (OR 1.94; CI 1.38–2.74), hypercholesterolemia (OR 1.68; CI 1.08–2.61), the use of calcium channel blockers at discharge (OR 1.80; CI 1.20–2.71), and left ventricular ejection fraction (LVEF) (OR 4.14 for patients with LVEF <30%; CI 2.42–7.10). The derived predictors were tested in the validation sample. The final model yielded two clinical predictors, i.e., younger age and severe LV-dysfunction, which correctly predicted post-discharge depression status in 82.9% of the MI patients. The model yielded a high negative predictive value (89%). A positive depression questionnaire (BDI) during hospitalization increased the positive predictive value of 23% to 52%.

Conclusions: During hospitalization for MI and using a two-step strategy with common clinical variables, i.e., younger age, severe LV-dysfunction and BDI score during hospitalization, it is possible to identify MI patients with a high risk for subsequent development of depression.

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Keywords: Myocardial infarction; Cardiac rehabilitation; Depression; Risk factors

1. Introduction

Depressive disorder following myocardial infarction (MI) is common, affecting 13–19% of all MI patients...
It is a major predictor of disability [3] and poor quality of life [4] in the year post-MI. Depressive disorder following MI is also associated with a delayed return to work [5], lower attendance to cardiac rehabilitation programs [6] and it complicates medical treatment due to its association with non-compliance [7]. Moreover, there is compelling evidence that post-MI depressive disorder has a negative influence on cardiac prognosis [8].

In view of the first positive experiences with antidepressant treatment for post-MI depressive disorder [9], it seems worthwhile to identify and subsequently treat post-MI depression. Specifically, it would be desirable to identify MI patients in the hospital wards who are at risk for a depressive disorder in the year following MI. Although previous studies reported clinical correlates of in-hospital depressive complaints, the predictors of a depressive disorder during the year following MI have not been investigated thoroughly.

We therefore prospectively studied a series of potential predictors of depressive disorder in the year following MI using standardized methods in a large cohort of MI patients. We considered only routinely available clinical variables to provide a practical tool to clinicians in predicting depressive disorder.

2. Methods

The present study is a predefined sub-study of a large, multicenter trial in The Netherlands: the Myocardial Infarction and Depression–Intervention Trial (MIND-IT) [10]. MIND-IT prospectively investigates the prognostic influence of antidepressive treatment for a depressive disorder following MI. The study was approved by the institutional review board of the University Medical Centre Groningen (coordinating centre) and the review boards of all other participating hospitals. Informed consent was obtained from each patient and the study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki. The Trial Coordination Centre in Groningen, The Netherlands, performed data management.

2.1. Patients

In MIND-IT, men and women, 18 years of age or older, who had had an acute MI between September 1999 and November 2002, admitted to one of the 10 participating hospitals, were eligible. MI was based on the occurrence of increased cardiac enzymes and either electrocardiographic changes and/or chest pain. Exclusion criteria were the occurrence of MI while the patient was hospitalized for another reason (except for unstable angina pectoris), inability to participate in study procedures (e.g., patients not able to communicate and patients not available for follow-up), any disease likely to influence short-term survival, patients already receiving psychiatric treatment for depression and participation in another clinical trial.

2.2. Design

We described the MIND-IT study design previously in detail [10]. In short, consenting, eligible patients were screened for depressive symptoms during hospitalization and at 3, 6, 9, and 12 months after MI with the Beck Depression Inventory (BDI) [11]. MI patients with depressive symptoms, as indicated by a BDI score ≥10 at 3, 6, 9 or 12 months post-MI underwent a standardized psychiatric interview. In this interview, the presence of a post-MI depressive disorder (ICD-10 criteria) was assessed. Whether the patient was referred for cardiac rehabilitation was left at the discretion of the patient’s cardiologist (who was blinded for the psychiatric screening results).

2.3. Predictor variables

As part of MIND-IT, demographics, medical history, clinical variables and risk factors for coronary artery disease were recorded at admission. Severity of infarction was assessed by the left ventricular ejection fraction (LVEF), as measured by either echocardiography or radio nuclide ventriculography. In-hospital treatment strategy (e.g., thrombolysis, coronary angioplasty (PTCA) or coronary bypass surgery (CABG)) and medication at discharge were extracted from patient charts. The cumulative burden of medical comorbidity was assessed with the Charlson Comorbidity Index, adapted for MI patients by Watkins et al. [12]. Higher scores correspond with more comorbidity.

2.4. Outcome variables

We used the 21-item BDI questionnaire [11] to screen for depressive symptoms. It yields a total score ranging from 0 to 63, with higher scores indicating higher levels of depressive symptomatology. BDI scores ≥10 are considered to indicate at least mild-levels of depression. Since the BDI does not provide a psychiatric diagnosis, patients with BDI scores ≥10 were interviewed using a standardized interview (the Composite International Diagnostic Interview [13] (CIDI; auto version 2.1). This psychiatric interview includes structured questions, allowing trained interviewers to assess criteria of the International Classification of Diseases, 10th revision [14] (ICD-10) systematically. The first CIDI interviews were performed not earlier than 3 months post-MI to allow natural recovery of depressive symptoms following a major life event. The interviewers were trained by skilled instructors. According to the outcome of the CIDI interview, we divided MI patients in two groups: (1) a group that met the ICD-10 criteria for a depressive disorder (further: “depression”) during the first post-MI year and (2) a group who...
remained free of depression. The antidepressant study intervention did not interfere with the present analysis, because the intervention was started after the identification of depression.

2.5. Statistical analysis

Our goal was to derive and validate clinical predictors according to methodological standards [15] to identify patients at risk for post-MI depression. Because of our large sample size, we used a split-sample design rather than a resampling technique to derive and internally validate our prediction model. For this goal, the patient group was randomly divided into a derivation sample (n=1073) and a validation sample (n=1104). All analyses were conducted with the use of SPSS software (version 12.0 for Windows).

2.6. Derivation of the prediction model

The bivariate associations between depression and potential predictors were assessed in the derivation sample by means of logistic regression analysis. Based on the bivariate regression model, it was decided which of the baseline variables were entered into the multivariate regression model (criterion of p \leq 0.10). We used a combined stepwise (backward and forward) procedure to obtain the final significant multivariate predictors. A p-value of 0.05 or less was considered to indicate statistical significance. The predictive value of the final set of predictors was evaluated by means of forced inclusion in the regression function, without interaction terms and without the non-significant predictors. The final logistic regression model yielded odds ratios (ORs) and 95% confidence intervals, estimating the odds of depression associated with a 1-unit change in the predictor variable, adjusted for other covariates in the model.

2.7. Validation of the prediction model

To assess the validity of our findings, we tested the accuracy of the prediction model by testing the derived predictors in the validation sample. The correctly predicted depression status was calculated from the proportion of patients that were correctly predicted as having a depression plus the proportion of patients that were correctly predicted as not having depression. Subsequently, we calculated the risk of depression for patient subgroups. This resulted in the predicted probability given the presence or absence of combinations of the observed predictors. Since depressive symptoms during hospitalization have already been considered as an important predictor of depressive disorder after discharge [16], we tested what this variable could add when included in the prediction model. For this purpose, we used the BDI score during hospitalization (categorized as a BDI score 0–9, 10–19, \geq 20).

3. Results

3.1. Baseline characteristics

In MIND-IT, 4780 MI patients were assessed for eligibility, of whom 1403 (29%) met exclusion criteria. Exclusion criteria were the occurrence of MI while the patient was hospitalized for another reason (n = 82), inability to participate in study procedures (e.g., decreased cognitive function, not able to communicate, transfer to other hospital, etcetera; n = 833), any disease likely to influence short-term survival (n = 87), patients already receiving psychiatric treatment for depression (n = 104) and participation in another clinical trial (n = 297). Of the 3377 remaining patients, 2177 were included (64%), while 1200 (36%) did not give informed consent. Table 1 gives a description of the baseline variables of the derivation sample (n=1073). The baseline variables in the derivation and validation sample (n=1104; data not shown) were comparable (all variables \textit{p}>0.05). About 78% of the included patients were men and the mean age was 61.2 (SD 11.9) years. More than one-third of the MI patients were treated with thrombolysis and a similar proportion underwent coronary angioplasty during hospitalization. Of the included patients, 7% had LVEF-scores below 30% and 9% was resuscitated during the transport to the hospital or during hospitalization. Mean BDI score during hospitalization was 6.7 (SD 6.2). Out of 1972 patients, 517 (24%) reported depressive symptoms (BDI \geq 10) during hospitalization.

3.2. Development of post-MI depression

During the follow-up period from 3 to 12 months, 33% (n = 722) of all MI patients reported once or more significant depressive symptoms (i.e., BDI \geq 10). A total of 876 CIDI interviews in these patients resulted in the identification of 375 patients with a post-MI depressive disorder according to ICD-10 criteria: \textit{n} = 199 (18.5%; 95% CI 16.2–20.9%) and \textit{n} = 176 (15.9%; 95% CI 13.8–18.1%) for the derivation and validation sample respectively; \textit{p}=0.11 for difference in proportion of depression in both samples. The distribution of the 375 positive CIDI interviews during the post-MI year was skewed, with the majority identified at 3 months (71%) and a minority identified at 6 months (16%), 9 months (8%) and 12 months (6%).

3.3. Predictors of depression in the derivation sample

Table 1 shows the results of bivariate analysis of clinical predictors for the occurrence of depression. Significant bivariate predictors (\textit{p} \leq 0.05) were younger age, hypercholesterolemia, smoking, PTCA during hospitalization for index MI and severe left ventricular dysfunction (LVEF <30%).

The results of multivariate modeling are shown in Table 2. MI patients younger than 60 years had almost a twofold
increased risk to develop depression (OR 1.94; CI 1.38–2.74). Patients with hypercholesterolemia (OR 1.68; CI 1.08–2.61) and patients using calcium channel blockers after discharge (OR 1.80; CI 1.20–2.71) had increased risk for depression post-MI. The risk for depression was more than quadrupled for MI patients with LVEF <30% (OR 4.14; CI 2.42–7.10). After adjustment, the effect of smoking did not reach statistical significance. In the derivation sample, the final model correctly predicted the depression status in 81.3% of the patients.

3.4. Validation of the prediction model

In the validation sample, relating the selected predictors to depression resulted in the following ORs: younger age (OR 1.99; CI 1.39–2.86), hypercholesterolemia (OR 1.25; CI 0.80–1.96), calcium channel blockers (OR 0.91; CI 0.56–1.46) and LVEF <30% (OR 2.31; CI 1.35–3.97). These variables correctly predicted the depression status in 84.6% of the patients. Although hypercholesterolemia and the use of calcium channel blockers were significantly associated with depression in the derivation sample, they lost their significance in the validation sample. Thus, two clinical predictors remained significant in the validation sample: younger age and LV-dysfunction. These two predictors correctly predicted depression status in 82.9% of the patients and were comparably predictive for early onset depression (≤3 months) as well as late onset.

Table 1
Results of bivariate analysis of clinical variables associated with post-MI depression (derivation sample)

<table>
<thead>
<tr>
<th>Baseline characteristic</th>
<th>Total (n=1073)</th>
<th>No depressive disorder (n=874)</th>
<th>Depressive disorder (n=199)</th>
<th>Odds ratio (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (female)</td>
<td>240</td>
<td>22.4</td>
<td>189</td>
<td>21.6</td>
<td>1.25 (0.87 to 1.78)</td>
</tr>
<tr>
<td>Age (&lt;60 years)</td>
<td>507</td>
<td>47.5</td>
<td>387</td>
<td>44.5</td>
<td>1.92 (1.40 to 2.63)</td>
</tr>
<tr>
<td>BMI (&lt;25)</td>
<td>399</td>
<td>38.4</td>
<td>320</td>
<td>38.0</td>
<td>0.92 (0.67 to 1.26)</td>
</tr>
<tr>
<td>DM</td>
<td>131</td>
<td>12.3</td>
<td>105</td>
<td>12.1</td>
<td>1.10 (0.69 to 1.74)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>375</td>
<td>35.3</td>
<td>308</td>
<td>35.6</td>
<td>0.92 (0.67 to 1.28)</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>812</td>
<td>76.4</td>
<td>645</td>
<td>74.6</td>
<td>1.84 (1.22 to 2.78)</td>
</tr>
<tr>
<td>Family history of CAD</td>
<td>473</td>
<td>45.3</td>
<td>376</td>
<td>44.2</td>
<td>0.97 (0.64 to 1.47)</td>
</tr>
<tr>
<td>Current smoker</td>
<td>516</td>
<td>48.6</td>
<td>407</td>
<td>47.2</td>
<td>1.09 (0.67 to 1.78)</td>
</tr>
<tr>
<td>Previous PTCA</td>
<td>66</td>
<td>6.2</td>
<td>48</td>
<td>5.6</td>
<td>1.70 (0.96 to 2.98)</td>
</tr>
<tr>
<td>Previous CABG</td>
<td>48</td>
<td>4.5</td>
<td>39</td>
<td>4.5</td>
<td>1.01 (0.48 to 2.11)</td>
</tr>
<tr>
<td>Previous MI</td>
<td>142</td>
<td>13.4</td>
<td>111</td>
<td>13.0</td>
<td>1.18 (0.77 to 1.82)</td>
</tr>
<tr>
<td>Killip class 2–4</td>
<td>118</td>
<td>11.1</td>
<td>88</td>
<td>10.2</td>
<td>1.57 (1.01 to 2.46)</td>
</tr>
<tr>
<td>Q-wave</td>
<td>695</td>
<td>66.6</td>
<td>569</td>
<td>67.0</td>
<td>0.90 (0.65 to 1.25)</td>
</tr>
<tr>
<td>Resuscitation</td>
<td>91</td>
<td>8.6</td>
<td>75</td>
<td>8.7</td>
<td>0.93 (0.53 to 1.63)</td>
</tr>
<tr>
<td>Thrombolysis</td>
<td>400</td>
<td>37.8</td>
<td>324</td>
<td>37.5</td>
<td>1.05 (0.77 to 1.45)</td>
</tr>
<tr>
<td>Charlson category</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cat. 0</td>
<td>599</td>
<td>57.1</td>
<td>497</td>
<td>58.3</td>
<td>1.0</td>
</tr>
<tr>
<td>Cat. 1</td>
<td>316</td>
<td>30.1</td>
<td>249</td>
<td>29.2</td>
<td>1.31 (0.93–1.85)</td>
</tr>
<tr>
<td>Cat. 2</td>
<td>101</td>
<td>9.6</td>
<td>84</td>
<td>9.9</td>
<td>0.99 (0.56–1.73)</td>
</tr>
<tr>
<td>Cat. 3</td>
<td>33</td>
<td>3.1</td>
<td>22</td>
<td>2.6</td>
<td>2.44 (1.15–5.18)</td>
</tr>
<tr>
<td>PTCA</td>
<td>400</td>
<td>37.6</td>
<td>309</td>
<td>35.7</td>
<td>1.53 (1.12–2.10)</td>
</tr>
<tr>
<td>CABG</td>
<td>51</td>
<td>4.8</td>
<td>43</td>
<td>5.0</td>
<td>0.80 (0.37–1.73)</td>
</tr>
<tr>
<td>Medication at discharge</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acetyl salicylic acid</td>
<td>910</td>
<td>85.4</td>
<td>748</td>
<td>86.3</td>
<td>0.70 (0.46–1.05)</td>
</tr>
<tr>
<td>Acenocoumarol</td>
<td>117</td>
<td>11.0</td>
<td>88</td>
<td>10.1</td>
<td>1.51 (0.96–2.37)</td>
</tr>
<tr>
<td>Nitrates</td>
<td>327</td>
<td>30.7</td>
<td>255</td>
<td>29.4</td>
<td>1.36 (0.98–1.88)</td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>917</td>
<td>86.0</td>
<td>745</td>
<td>85.9</td>
<td>1.04 (0.67–1.63)</td>
</tr>
<tr>
<td>Calcium channel blocker</td>
<td>186</td>
<td>17.4</td>
<td>142</td>
<td>16.4</td>
<td>1.45 (0.99–2.12)</td>
</tr>
<tr>
<td>Statin</td>
<td>802</td>
<td>75.2</td>
<td>643</td>
<td>74.2</td>
<td>1.39 (0.95–2.02)</td>
</tr>
<tr>
<td>Diuretics</td>
<td>162</td>
<td>15.2</td>
<td>129</td>
<td>14.9</td>
<td>1.22 (0.75–1.54)</td>
</tr>
<tr>
<td>Digoxin</td>
<td>34</td>
<td>3.2</td>
<td>25</td>
<td>2.9</td>
<td>0.72 (0.73–3.48)</td>
</tr>
<tr>
<td>ACE-inhibitor</td>
<td>447</td>
<td>41.9</td>
<td>363</td>
<td>41.9</td>
<td>0.74 (0.46–1.27)</td>
</tr>
<tr>
<td>LVEF &lt;30%</td>
<td>65</td>
<td>6.7</td>
<td>37</td>
<td>4.7</td>
<td>0.43 (0.23–0.80)</td>
</tr>
</tbody>
</table>

CI—confidence interval; BMI—body mass index, weight/(length)^2; DM—diabetes mellitus; CAD—coronary artery disease; PTCA—percutaneous transluminal coronary angioplasty; CABG—coronary artery bypass grafting; MI—myocardial infarction; LVEF—left ventricular ejection fraction.

Table 2
Results of multivariate analysis of clinical variables associated with post-MI depression (derivation sample)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Odds ratio (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypercholesterolemia</td>
<td>1.68 (1.08–2.61)</td>
<td>0.02</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>1.80 (1.20–2.71)</td>
<td>0.005</td>
</tr>
<tr>
<td>Age &lt;60</td>
<td>1.94 (1.38–2.74)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LVEF &lt;30%</td>
<td>4.14 (2.42–7.10)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

CI—confidence interval; LVEF—left ventricular ejection fraction.

*a Lost significance when tested in validation cohort.
depression (>3 to 12 months) \( (p > 0.05 \text{ for comparison between ORs}) \).

### 3.5. Assessing the risk of depression after MI

To determine how well the final model predicted outcome in the total sample, we assessed the associated risk of one or a combination of the two predictors for the development of depression (Table 3). Since no differences were found on the selected predictors of depression (i.e., age, LVEF and BDI score) between the derivation and the validation sample, we used data on risk assessment based on the whole sample. Younger MI patients with severely compromised LV-function had a 36% risk for post-MI depression. To assess the potential merit of baseline psychiatric data in the prediction of post-MI depression, we added the hospital BDI scores in the prediction model. Baseline BDI score was associated with an increased risk of depression (OR 1.18; 95% CI 1.16–1.21), while LVEF (OR 1.68; 95% CI 1.44–1.95) and age (OR 0.97; 95% CI 0.95–0.98) remained significant predictors. The presence of depressive symptoms at hospitalization (BDI score \( \geq 10 \)) improved the accuracy of the model markedly for all risk groups. In Table 4, these two steps in the prediction model are described in terms of sensitivity, specificity, negative predictive value and positive predictive value. The clinical predictor model had a high negative predictive value (89%), whereas a combination of the clinical predictor model and BDI-data resulted in a more than doubling of the positive predictive power (from 22.9% to 52.0%), while the negative predictive value did not deteriorate (from 89.1% to 87.4%).

### Results of prediction model of post-MI depression (total sample)

<table>
<thead>
<tr>
<th>Predictor model using age &lt;60 years or LVEF &lt;30% in all patients (n=1983)</th>
<th>Predictor model using BDI score in high-risk patients age &lt;60 years or LVEF &lt;30% (n=1002)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensitivity (%)</td>
<td>69.0 (234/339)</td>
</tr>
<tr>
<td>Specificity (%)</td>
<td>52.0 (855/1644)</td>
</tr>
<tr>
<td>Positive predictive value (%)</td>
<td>22.9 (234/1023)</td>
</tr>
<tr>
<td>Negative predictive value (%)</td>
<td>89.1 (855/960)</td>
</tr>
</tbody>
</table>

BDI—Beck’s Depression Inventory; LVEF—left ventricular ejection fraction.
increased risk to become depressed, although our assessment of severity of comorbidities and Killip class during hospitalization in this context did not provide useful prognostic information. It is tempting to speculate that neurohormonal activation or an increase in inflammatory cytokines, which both accompany left ventricular dysfunction, plays a role in the development of depression [20,21]. Of note, the ongoing discussion whether depression predicts clinical prognosis seems to concentrate on the issue if depression and cardiac disease severity are related [22,23]. Our finding that low LVEF predicts depression provides a rationale for more detailed examination of the role of LVEF in studies on the prognostic impact of depression. We found that younger patients were at increased risk of depression. These data are consistent with results from community-based studies [24] and studies in cardiac patients [25,26] reporting that depression is more prevalent among younger patients.

It is noteworthy that the use of beta-blockers was not associated with the development of depression, although beta-blockers are believed to potentially cause depression. Our data do not support this belief and may take away the reluctance and concerns to prescribe beta-blockers to post-MI patients for this reason. This finding is in line with results of a recent meta-analysis [27].

The results have to be considered in relation to the study limitations. Firstly, our analyses did not take into account history of depression as a potential predictor for post-MI depression. The accuracy of its assessment would have been biased by feelings of depression accompanying the MI. In addition, selection criteria (i.e., MI patients who were treated for a current depression at the time of the index MI were excluded) precluded entering this variable into the model. Further research is warranted to explore the merit of pre-MI depression in the predictor model. Secondly, only MI patients with BDI scores ≥10 had a psychiatric interview. Therefore, it could be possible that MI patients with low BDI scores had a depressive disorder if they had been interviewed. This chance however is relatively low, because of the high specificity of the BDI [2]. Thirdly, we were not informed about dosage and possible changes of the medication after discharge. Finally, both the SADHART [9] and ENRICHD [28] trial revealed that many depressed MI patients have spontaneous improvement in depression scores, especially shortly after MI. We partly accounted for this spontaneous improvement because the first interviews were performed not earlier than 3 months post-MI. Notwithstanding the spontaneous recovery in the early phase post-MI, Lespérance et al. [16] described that a substantial other part of the depressed MI patients showed a more chronic course of depression. From our study, it is not possible to make inferences on the natural course of post-MI depression, because no serial CIDI interviews were performed and because an antidepressant intervention was started in depressed MI patients.

We were able to correctly predict the post-discharge depression status in 82.9% of the patients. The positive predictive value of 22.9% is higher than the value reported in the study Lespérance et al. [16], although still modest. The strength of our model, however, lies in the high negative predictive value of 89.1%. In other words, almost 90% of the patients identified by our model as not having a high risk for developing depression did indeed not develop depression during follow up. When we take in at a glance our data and those of others [16,18], the best prediction may come from a model using both clinical and psychiatric data (e.g., depressive symptoms during hospitalization). Because the assessment of depressive symptoms during hospitalization is not part of today’s standard cardiac care, we propose a two-step strategy to identify patients at risk for depression. The first step would be to assess the risk of depression with 2 simple clinical variables (age and LV-function). After thus narrowing down the MI population at risk for depression, the second step would be to further explore the risk with psychiatric data using the BDI questionnaire. This strategy combines the strength of a high negative predictive value in the first step with the strength of a high positive predictive value in the second step. Although the procedure fails to identify a third of all cases of post-MI depression, we consider this acceptable. Firstly, because two-thirds of incident cases of depression in the entire post-MI year indeed is identified by taking into account two simple clinical variables and the administration of the BDI only in the high-risk subgroup. This is in contrast with the low identification rate in standard care [29]. In addition, given its high negative predictive value, this simple detection strategy can identify patients not at risk for post-MI depression with a fair certainty. Collectively, these data show that, prior to the rehabilitation phase, depression could be predicted using easy-to-obtain variables during hospitalization.

Acknowledgement

The MIND-IT is sponsored by The Netherlands Heart Foundation. Dr. Van Melle and Dr. De Jonge are supported by the same grant of The Netherlands Heart Foundation (97.016) to Prof. Ormel, Principal Investigator MIND-IT. The MIND-IT received educational grants from Organon (The Netherlands) and Lundbeck (Denmark).

Appendix A

The following investigators en institutions in The Netherlands participated in the MIND-IT study:

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