Heart, Lung and Circulation (2019) **28,** 1812–1818
1443-9506/04/$36.00
https://doi.org/10.1016/j.hlc.2018.11.013

ORIGINAL ARTICLE

Mild Depression Predicts Long-Term Mortality After Acute Myocardial Infarction: A 25-Year Follow-Up

Marian U. Worcester, PhD [a,b*], Alan J. Goble, MD [a], Peter C. Elliott, PhD [c], Erika S. Froelicher, PhD [d], Barbara M. Murphy, PhD [a,e,f], Alison J. Beauchamp, PhD [a,b,g,i], Michael V. Jelinek, MD [h,i], David L. Hare, MBBS [i,j]

[a]Australian Centre for Heart Health, Melbourne, Vic, Australia
[b]Department of Epidemiology and Preventive Medicine, Monash University, Melbourne, Vic, Australia
[c]Phoenix Australia Centre for Posttraumatic Mental Health, University of Melbourne, Melbourne, Vic, Australia
[d]Department of Physiological Nursing, Department of Epidemiology & Biostatistics, Schools of Nursing and Medicine, University of California San Francisco, CA, USA
[e]Department of Psychology, University of Melbourne, Melbourne, Vic, Australia
[f]Faculty of Health, Deakin University, Melbourne, Vic, Australia
[g]Australian Institute for Musculoskeletal Science (AIMSS), The University of Melbourne and Western Health, Melbourne, Vic, Australia
[h]Department of Cardiology, St Vincent's Hospital, Melbourne, Vic, Australia
[i]Department of Medicine, University of Melbourne, Melbourne, Vic, Australia
[j]Department of Cardiology, Austin Hospital, Melbourne, Vic, Australia

Received 9 April 2018; received in revised form 25 October 2018; accepted 26 November 2018; online published-ahead-of-print 5 December 2018

Background Past studies have found that depression is an independent predictor of death in patients after acute myocardial infarction (AMI). Our aim was to investigate whether the adverse effect upon mortality of depression, including mild levels, persisted up to 25 years.

Methods We used an historical design to study patients who had been consecutively admitted to hospital after transmural AMI during the 1980s and enrolled in an exercise training trial. The Beck Depression Inventory (BDI) was administered to 188 patients in the third week after hospital admission. Scores were trichotomised and classified as low (0–5), mild (6–9) or moderate to severe (≥10) depression. The Australian National Death Index was used to determine mortality status. Cox proportional-hazards modelling was undertaken to determine the relationship between the trichotomised BDI-I scores and all-cause mortality over five time periods up to 25 years.

Results The mean age of patients was 54.15 years. One hundred fourteen (114) (60.4%) had low or no depression, 47 (25.2%) mild depression and 27 (14.3%) moderate to severe depression. The mortality status of 185 (98.4%) patients was established. Depression was a significant predictor of death, independently of age and severity of myocardial infarction, at 5, 10 and 15 years but not at 20 or 25 years. Patients with mild depression had greater mortality than those with low or moderate to severe depression.

Conclusions Early identification of depression, including milder levels, is important since patients remain at increased risk for many years. They require ongoing monitoring and appropriate treatment.

Keywords Depression • Long-term mortality • Cardiovascular diseases prevention • Acute coronary syndrome • Cardiac rehabilitation • Secondary prevention

*Corresponding author. Tel.: +61 3 9326 8544. Email: marian.worcester@monash.edu

Introduction

Depression is common after acute myocardial infarction (AMI) and is associated with adverse outcomes, including mortality [1,2]. About 20% of patients experience major depression after acute coronary syndrome (ACS) or AMI [1,3]. Milder depressive symptoms are even more common [3,4]. Depression after AMI has been independently associated with mortality in several studies over a number of years [4–6], although not all [7,8]. The prognostic significance of milder depressive symptoms has also been recognised [6,9–11].

Despite the failure of past studies to adjust for potential confounders, a recent report concluded that there is convincing evidence of the adverse effect of depression upon prognosis following acute cardiac events, including mortality, independently of disease severity [2]. The American Heart Association has therefore recommended that depression should be elevated to the status of a risk factor for adverse medical outcomes in patients with ACS [2].

However, the mechanisms whereby depression is linked to poorer outcomes are unclear [2,12]. Non-adherence may be the mediator between depression and mortality in cardiac patients. Depression is commonly associated with non-adherence to advice regarding medication, smoking, physical activity [13] and cardiac rehabilitation (CR) attendance [12]. Thus, it is important to identify and manage depression early.

The Beck Depression Inventory (BDI) [14] has commonly been used in studies of depression after AMI [5–8,10,15–17], with most studies using a BDI cut-point of 10 to classify patients with clinically significant symptoms of depression [5,7,15–17]. However, even a BDI score of between 4 and 9 has been associated with an increased risk of death [6]. Moreover, when identifying depression in cardiac patients, cut-offs lower than those used in the general population are considered appropriate [18].

The present study involved a 25-year follow-up of men who were recruited after AMI to participate in a randomised controlled trial conducted during the 1980s. The aim of the trial was to determine whether high intensity exercise training achieved more favourable physical and psychological outcomes compared with moderate intensity exercise. Depression was one important endpoint of this trial [19,20]. The present follow-up study was undertaken to determine whether depression after AMI predicted mortality over a 25-year period. While the follow-up study was not planned at the time of the original study, the data we had collected in the 1980s provided an opportunity to investigate the prognostic value of depression over this extended period, longer than any previous mortality follow-ups. It was hypothesised that depressed patients, including those with mild symptoms, would have greater mortality risk at follow-up compared with those without depression. Further, it was anticipated that the effect of depression would persist for many years.

Methods

An historical prospective design was used to achieve the aims of this follow-up study.

Baseline Study Population

The baseline study population comprised men under 70 years of age who were consecutively admitted to the coronary care unit of a university teaching hospital following acute transmural (Q wave) myocardial infarction (AMI), historically prior to the introduction of routine early reperfusion strategies for ST elevation MI. Of the 339 eligible men, 224 were included and 115 were excluded (Figure 1). Details of the original study, which formed the baseline for the current follow-up, have been reported elsewhere [19,20]. Recruitment for the original study was confined to male patients to provide a uniform cohort for the trial of high intensity compared with moderate intensity exercise training.

Procedure

Baseline medical data were gathered in hospital by cardiology registrars. Interviews were conducted by the first author to document socio-demographic and occupational characteristics of patients. Physical working capacity, cardiovascular risk factors and depression were measured upon entry to the outpatient CR programs in the third week after AMI.

Measures

Medical data included the following: age, cardiac enzyme levels, site of AMI (anterior or lateral/inferior or posterior), clinical heart failure (no heart failure/heart failure or pulmonary oedema), radiographic heart size (normal/borderline/enlarged), cholesterol, previous infarction and/or ischaemia, smoking status, history of hypertension and diabetes status. Killip class was chosen as the measure of the severity of the event [21]. Occupation was classified according to a six-point scale [22] and dichotomised as non-manual and manual. The Beck Depression Inventory I (BDI-I) was used to assess depression [14]. It is a 21-item tool, with four possible responses ranging in intensity for each item. Scores range from 0 to 63.

Determining Mortality Status at Follow-Up

The Australian National Death Index (ANDI) was accessed to determine the mortality status of patients during the 25 years after the index event. The human research ethics committees of the Austin Hospital and the Australian Institute of Health and Welfare Institute approved the study to enable access to ANDI and surviving patients or their relatives.

Main Outcome

We selected all-cause mortality as the primary outcome of our study to be consistent with many other investigations [6,7,9,15–17].

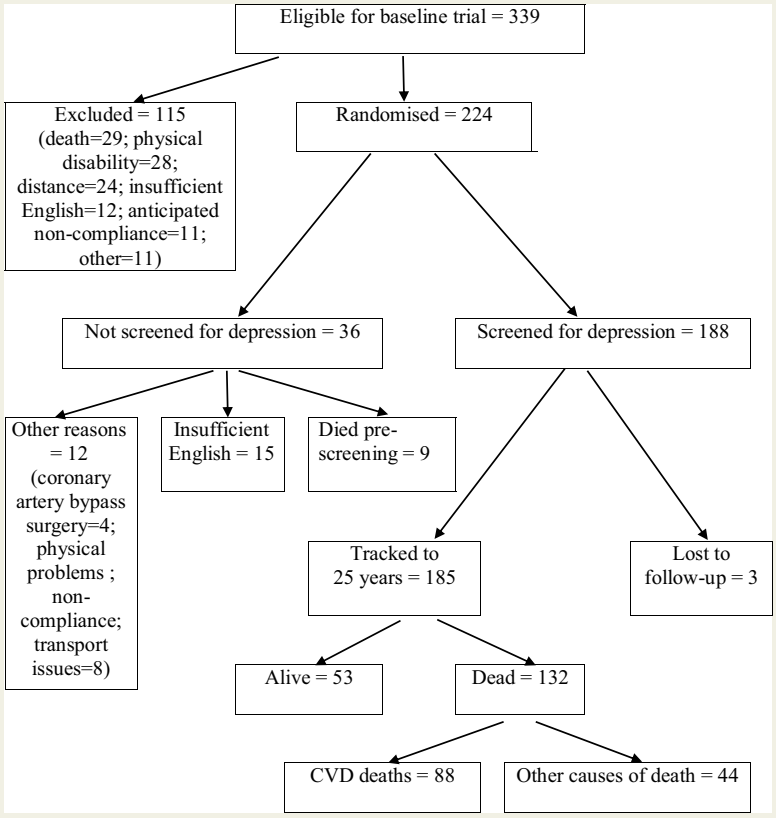


Figure 1 Flowchart of patient numbers at each time-point.

Data Analysis

All analyses were carried out using SPSS for Windows version 15 (SPSS Inc., 2006, Chicago, IL, USA). Beck Depression Inventory scores were trichotomised, with scores of 0–5 being classified as low or none, 6–9 as mild, and ≥10 as moderate to severe depression. These cut-offs were chosen on the basis of the distribution of deaths by BDI scores, a method used in a previous mortality study of cardiac patients [6], although the resulting cut-offs differed slightly from that study. Cross-tabulations were carried out to determine the percentage of patients who were dead by the end of each of the 5-year time periods. Cox proportional-hazards modelling was used to test the relationship between the BDI categories and all-cause mortality at 5, 10, 15, 20 and 25 years after AMI. For each period, time to death was predicted from the BDI category. Age and severity of infarction were included as control variables. For each proportional hazards model, the *p*-value for the overall relationship between BDI and mortality is reported, as well as the *p*-values for the three pair-wise comparisons between BDI categories and their ability to predict death, namely, comparisons between scores of 0-5 vs. 6-9; 0-5 vs. ≥10; and 6-9 vs. ≥10.

Results

The mean age of patients at entry to the baseline study was 54.15 (±8.54; range 34–69) years. Other socio-demographic and medical characteristics of patients are shown in Table 1.

The BDI was completed by 188 of the 224 patients randomised to the trial (Figure 1). At the 25-year follow-up, mortality data were available for 185 men. The median time when half the patients were alive and half were dead was 19.3 years.

Beck Depression Inventory scores were heavily skewed. Scores ranged from 0 to 23, with a mean score of 5.6 (SD = 4.5). There were 114 (60.4%) patients who had low or no depression, 47 (25.2%) with mild depression and 27 (14.3%) with moderate to severe depression.

Table 2 shows cumulative deaths within each BDI category over the 25-year period. The significance of the test across all categories of depression is shown in the ''Overall'' column. Post-hoc tests examining differences in death rates when comparing pairs of BDI categories are shown in the remaining three columns. There was a significant overall relationship between depression and death at 5, 10 and 15 years. At each of these three time-points, the significant difference was between the low and mild depression categories. At both 5 and 10 years, there was a trend ($p < 0.10$) for those with moderate to severe depression to have a higher percentage of deaths than those with low or no depression. There was no association between depression and all-cause mortality at 20 and 25 years.

Figure 2 shows the 25-year survival curves in the three BDI categories. There is a clear divergence in survival rates from 5 to 15 years, no longer observed by 20 years post-MI. Patients with low depression scores at three weeks had the best prognosis (mean survival time = 15.21 years, SD = 7.97;

Table 1 **Baseline characteristics of patients (n = 188).**

Participant characteristic	Mean	SD	N	%
Age (years)	54.15	8.54		
Born in Australia			122	64.9
Partnered			169	89.9
Left school ≤15 years			99	52.9
In workforce			141	75.0
Non-manual occupation			87	46.3
Anterior infarction			75	40.1
Killip class 2 or 3			75	39.9
Current smoker			101	53.7
History of hypertension			57	30.5
Cholesterol (mmol/litre)	6.04	1.37		
Systolic blood pressure (mmHg)	130.97	23.03		
Diabetes mellitus			9	4.9
Waist girth (cm)	93.33	10.19		
Body mass index (kg/m^2)	26.13	2.87		
Physical working capacity (METs)	6.76	2.56		

95% CI = 13.74–16.67), followed by patients with moderate to severe scores (11.59 years, SD = 10.05; 95% CI = 7.72–15.45). Patients with mild depression scores had the worst prognosis (10.18 years, SD = 9.29; 95% CI = 7.49–12.86).

Discussion

In this study, we tracked patients for 25 years, which, to our knowledge, is the longest reported follow-up investigation of the relationship between depression after AMI and long-term mortality. Follow-up periods of similar past studies ranged from 4 months [6,8], 6 months [4], 12 months [7,16]; 18 months [5]; 5 years [9,10,15] to 10 years [23]. We found that depression measured during early convalescence was a significant predictor of death, independently of age and AMI severity, at 5, 10 and 15 years but not at 20 and 25 years. These findings are testament to the strong prognostic role of depression up to 15 years post-event. It is not surprising that by 20 and 25 years, when the mean age of the cohort was 74 and 79 years respectively, depression no longer predicted death.

Importantly, our results demonstrate the prognostic significance of milder depressive symptoms. Whereas a BDI cut-point of 10 has commonly been used to denote the presence of at least mild depression [7,8,15–17], we used a cut-point of 6. Scores of 6 to 9, reflecting relatively low, sub-clinical levels of depression, were predictive of mortality for all the time periods up to 15 years. An earlier study similarly reported that low BDI scores between 4 and 9 were associated with an increased risk of death after AMI [6].

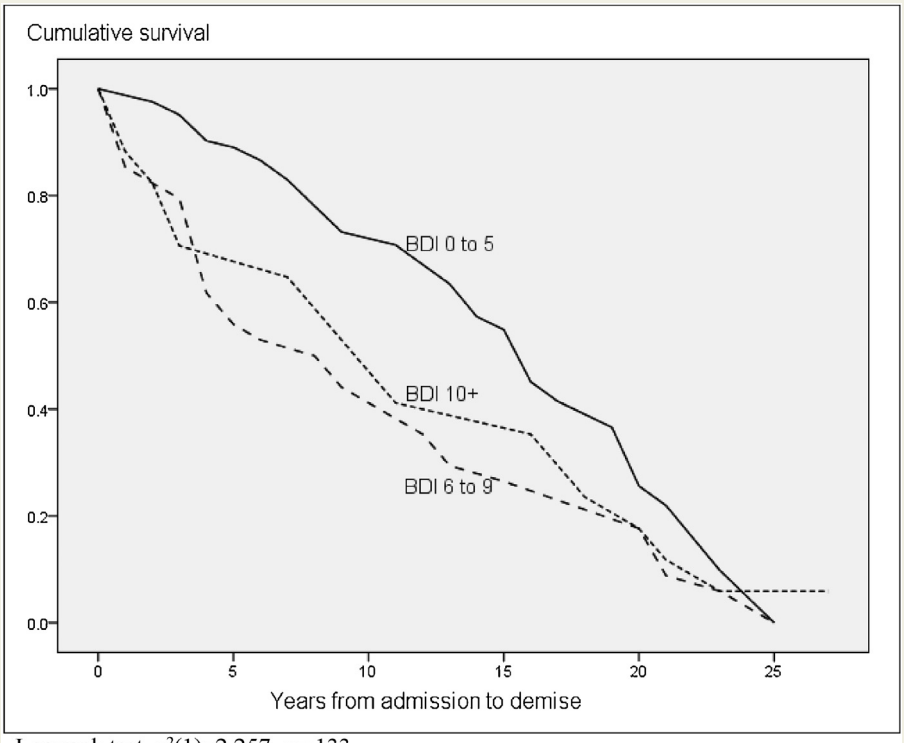
We found that patients with mild depression had greater mortality than patients with low or no depression and, although not statistically significant, possibly due to small sub-sample sizes, there appeared to be a tendency for those with mild depression to have a higher mortality than those with moderate to severe depression. This finding is in contrast to previous studies which found a dose response relationship between severity of depression and mortality [6,10]. However, the present finding is consistent with our study of a consecutive series of 170 female patients admitted to hospital after AMI or to undergo coronary artery bypass graft surgery where mild depression close to the cardiac event was a stronger predictor of death at 12 years post-event than was moderate to severe depression [11]. In the early post-event period, elevated depression scores in some patients resolve quickly and are indicative of a grief response which does not necessarily lead to poor outcomes, whereas milder depressive symptoms might suggest pre-existing depression and a worsening depression trajectory [11]. It may also be that severe depression is more often recognised and early treatment initiated, whereas mild depression is not so readily identified. This provides a hypothesis that is worthy of further study.

An unexpected finding of our study was the unusually low BDI scores of most patients. In previous studies, a significant number of AMI patients achieved BDI scores of 10 and above [6,15]. In our investigation, only one quarter of the patients had scores of 10 or more. The preponderance of low BDI scores might reflect the persistence of denial among our patients [24].

The timing of our depression measurements might also have accounted for the relatively low BDI scores. The exercise training trial involved numerous assessments at entry to CR in the third week after AMI. It was considered efficient to assess depression also at this time. Further, our decision to

Table 2 **Percentage of patients in each BDI category (n = 185).**

Year	Number	Percentage dead within BDI category			P-value			
	Dead	Low n = 113	Mild n = 46	Severe n = 26	Overall	Low vs. Mild	Low vs. Severe	Mild vs. Severe
5	29	8.0	32.6	19.2	0.004	0.001	0.069	0.431
10	51	19.5	43.5	34.6	0.014	0.004	0.071	0.639
15	72	32.7	54.3	38.5	0.022	0.006	0.271	0.355
20	103	54.0	60.9	53.8	0.406	0.184	0.561	0.670
25	132	72.6	73.9	61.5	0.517	0.278	0.941	0.405



Log rank test, $\chi^2(1)=2.257$, $p=.133$.

	0	5	10	15	20	25
Dead	0	29	51	72	103	132
Alive	185	156	134	113	82	53
Alive by BDI group						
Low	113	104	91	76	52	31
Mild	46	31	26	21	18	12
Severe	26	21	17	16	12	10

Figure 2 Twenty-five year survival curves for patients in each BDI category.
Abbreviation: BDI, Beck Depression Inventory.

measure depression during early convalescence rather than in hospital reflected the prevailing opinion that depression peaks soon after discharge from hospital [25]. It is likely that assessments of depression undertaken during convalescence are more reliable because there is a risk of misclassification if the identification of depressed patients is based purely on in-hospital assessments [11]. Depressive symptoms displayed in hospital might reflect a transient grief response [11,24] and resolve spontaneously [3] or be concealed by denial [24]. Later assessments enable the identification of patients with delayed onset or persistent depression [11].

There are several limitations to our investigation. First, it involved a relatively small number of participants and might, therefore, have been under-powered for a long-term mortality investigation. Nonetheless, analyses produced statistically significant associations between depression and death. Second, participants were enrolled to take part in a clinical trial. One-third of eligible patients were excluded or withdrawn from the trial, mostly because of death or physical disability. Excluded patients tended to be older and sicker than others [20]. Such patients might have had higher death rates. Moreover, the trial was confined to men aged less than 70 years from a single institution. Thus, our results might not be generalisable to a broader group of cardiac patients, including women, older patients and those who did not attend CR. As noted, though, a similar pattern of findings has been reported for women [11]. Importantly, excluded survivors were, nevertheless, invited to attend a standard physiotherapy-based CR program. Third, while we controlled for age and AMI severity, we did not control for other potential confounders or mediators. Later medical and other events occurring between the index AMI and follow-up at 25 years could have resulted in limitations and biases of unknown magnitude. Possible confounders include non-adherence [13], later comorbidities which exacerbated depression [17], worsening disease severity or poorer secondary prevention control of risk factors [26]. In particular, we have no data concerning resumption of smoking among our patients, given their high rate (53.7%) of smoking upon hospital admission. Continued smoking increases the risk of death after MI significantly [27]. Fourth, we had no information about a past history of depression or depression

treatment received during follow-up, both of which have been shown to have prognostic importance [28,29]. However, anti-depressants were infrequently prescribed for cardiac patients at the time of the baseline study, and even in much later studies [6,7]. Finally, improved treatments of coronary heart disease (CHD), with much lower cardiovascular disease (CVD) mortality rates [30], might also have reduced the negative physiological impact of depression.

Whether the course of patients' depression was related to mortality outcomes is also unknown. Depression is a chronic condition and might have persisted or recurred in some patients over the 25 years [26]. Further, patients who initially had mild depression might have developed major depression subsequently [5,17].

Despite these limitations, our study has several strengths. Our investigation of a consecutive series of patients to 25 years exceeds the length of previous follow-up studies of depression and mortality in post-MI patients. Participants recruited to the baseline trial were a consecutive series of patients with no physician selection bias and a low refusal rate. Rigorous tracking procedures ensured that only three patients were lost to follow-up. Importantly, our study has confirmed the significance of milder depressive symptoms in predicting mortality. Findings suggest that there could be value in shifting the focus away from the well-established BDI cut-point of 10 for predicting long-term mortality of post-MI patients to a lower cut-point of 6. However, additional research is needed to confirm these results.

Although there is not yet convincing evidence that treating depression reduces mortality [31], our findings underscore the importance of early identification and management of depression to prevent its adverse effects upon adherence and quality of life. Unfortunately, depression often has a subtle presentation and may be masked by denial [24]. It is not always recognised and is thus under-treated by clinicians [7]. Routine depression screening in hospital is therefore recommended [1,32] using a brief screening tool, such as the Patient Health Questionnaire-2 (PHQ-2) [33], to help identify patients who require further assessment, treatment or referral to a mental health specialist.

For routine screening to be beneficial, however, effective management protocols need to be in place and well-trained professionals available to provide appropriate care. Collaborative care offers an effective, multidisciplinary approach to the co-ordinated management of depressed patients [32,34]. Its strengths include systematic assessment of depression and the use of a non-physician care manager to monitor symptoms and to co-ordinate referrals, treatment interventions and specialist-provided stepped care recommendations [34].

Physical activity, cognitive behaviour therapy, selective serotonin reuptake inhibitors and collaborative care have been identified as effective treatments for depression in cardiac patients [32], with a combination of these treatments shown to yield better outcomes [35]. In addition to these treatment options, group CR and secondary prevention programs can also help to reduce depression [1,36,37]. Attendance is therefore strongly recommended [2,37]. Encouragement by cardiologists to attend has been shown to be a major predictor of CR participation [38]. Rescreening for depression can be readily incorporated into the routine of such programs. Further screening should be undertaken at intervals over time in various settings to identify new or persistent depression and to prevent worsening depression [1].

Conclusion

Patients with depression, including mild depressive symptoms, should be recognised as being at increased risk long after their ACS [9]. They require ongoing monitoring. More aggressive clinical management might also be needed to increase adherence to regimens for secondary prevention in patients who fail to respond to depression treatments [17].

Funding

This work was supported by *beyondblue*.

Author Contributions

MW, AG, PE, BM, MJ and DH contributed to the conception or design of the work. MW, EF, PE and AB contributed to the acquisition, analysis or interpretation of the data. MW, AG, PE and BM drafted the manuscript. EF, AB, MJ and DH critically revised the manuscript.

Declarations of Interest

None.

Acknowledgements

The assistance of Mr Michael Le Grande is gratefully acknowledged.

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