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Original Scientific Paper

Anxiety and depression after acute myocardial infarction: an 18-month follow-up study with repeated measures and comparison with a reference population

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Background **Recently, there has been substantial improvement in coronary care and a corresponding reduction in mortality after acute myocardial infarction (AMI). Some studies suggest that improved prognosis has led to reduced levels of anxiety and depression after AMI, in both the short and long term. The aims of this study were to assess symptoms of anxiety and depression from the acute event to 18 months following AMI, and to compare results with levels in the Norwegian reference population.**

Design and methods **The progress of 288 patients was monitored using self-reports 3, 6, 12 and 18 months after AMI. Anxiety and depression were measured by the Hospital Anxiety and Depression Scale. Reference population data were obtained from the Nord-Trøndelag Health Study 1995–1997 (the HUNT 2 Study).**

Results **At baseline, 19.7 and 13.6% of AMI patients reported high levels of anxiety and depressive symptoms, respectively. At baseline, AMI patients were more anxious, but not more depressed, when compared with the reference population ($P<0.001$ and $P=0.092$, respectively). After 3–18 months, AMI patients' levels of anxiety and depression were not higher than levels in the reference population. Anxiety and depression at baseline and after 3 months were the best predictors of anxiety and depression after 18 months, although complications, bed days and lifestyle improvement also significantly predicted depression after 18 months.**

Conclusion **Initially, AMI patients had higher levels of anxiety, but not depressive symptoms. After 3–18 months, these patients were not more anxious or depressed than the Norwegian reference population.** ***Eur J Cardiovasc Prev Rehabil*** **16:651–659 © 2009 The European Society of Cardiology**

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Introduction

There is increasing evidence that psychosocial factors, such as depression and anxiety, worsen the prognosis of coronary heart disease (CHD), as these factors may act as barriers in any effort to improve the lifestyle and promote the patients' health-related quality of life (HRQOL) [1,2].

Depression and depressive symptoms are common, both in the general population and in patients with acute myocardial infarction (AMI), although the prevalence varies somewhat depending on assessment method [3,4]. According to the pooled results of a recent meta-analysis, the prevalence of depression, assessed by a self-completed scaled questionnaire, a diagnostic interview, diagnosis by

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a physician, antidepressant medication or self-reported diagnosis, was 13% in healthy populations and 28% in populations with existing CHD [4]. Furthermore, it has been reported that 15–20% of such patients have major depression, and a similar proportion have minor depression, after an AMI [3,5–7]. In a longitudinal study of patients who had suffered an AMI, the occurrence of high levels of depressive symptoms, as measured by the Beck Depression Inventory, was 26, 38 and 37% during hospitalization, after 4 and 12 months, respectively [8].

In numerous studies, depression has been associated with mortality, physical disability and reduced HRQOL in CHD patients [6,7,9–11]. In addition, depressive symptoms are associated with use of healthcare resources and lack of lifestyle changes [12].

Compared with depression, few studies have investigated the relationship between symptoms of anxiety and outcomes after AMI [12–16]. High levels of anxiety symptoms, as measured by the Hospital Anxiety and Depression Scale (HADS), in a Norwegian general population, which excluded those with somatic disease, occurred in 11% of women and 7% of men [17]. Anxiety symptoms, as measured by the State Trait Anxiety Inventory or HADS, have been reported to be 24–37% in studies involving hospitalized AMI patients [8,12]. Other studies using the same instruments have reported the presence of anxiety symptoms in 27–41% of patients from 6 weeks to 12 months after discharge following an AMI [8,18].

Anxiety after AMI has been reported to predict poorer HRQOL [13,19], cardiac rehospitalization [14] and frequent visits to the cardiac outpatient clinic [14]. There is inconclusive evidence with regard to the effect of anxiety on mortality from CHD [19–21].

Recently, substantial improvements in medical treatment during coronary care, including revascularization and secondary prevention, may explain the reported improvements in coronary endpoints survival, cardiac event rates and CHD mortality [22]. To the best of our knowledge, there have been only a few recent studies reporting longitudinal assessments of symptoms of anxiety [8,12,18,21] and depression [8,12,18] following an AMI. More research is required to assess the extent to which changes in care and improved prognosis have affected the levels of depression and anxiety in AMI patients [7,15]. Thus, to increase knowledge about the symptom levels of anxiety and depression at several time points after an AMI, and to identify vulnerable groups with poor long-term psychological outcomes, the aims of this study were as follows:

(1) To compare the symptom levels of anxiety and depression longitudinally in AMI patients using repeated measures over a period of 18 months after discharge.
(2) To compare the symptom levels of anxiety and depression in AMI patients with those in the general Norwegian population.
(3) To determine the extent to which demographical and clinical variables, as well as changes in lifestyle, predict long-term symptoms of anxiety and depression in AMI patients.

Methods

Design and patients

This prospective cohort study was conducted using the sample from a randomized controlled trial conducted at the Haukeland University Hospital, Bergen, Norway, from September 2001 to July 2005 [23,24]. The aim of this trial was to evaluate the effect of a telephone follow-up intervention for AMI patients after their discharge from hospital. Briefly, all patients admitted to the hospital with a diagnosis of AMI, as confirmed from their medical records, were invited to participate in the study unless they had a coexisting severe chronic disabling disease, resided in a nursing home, or were unable to receive telephone calls or fill in questionnaires. Data from all participants were collected from medical records and by self-reports through mailed questionnaires at baseline and at 3, 6, 12 and 18 months after discharge, with at most one reminder at each of these time points. There were no significant effects of the intervention on the psychological dimensions of the HRQOL [23,24]. Furthermore, there were no baseline differences between the groups, or any effects of the intervention on the HADS subscales at each of the different measurement points 3, 6, 12 and 18 months after discharge (results not published). When analysing differences between the intervention and control groups, using a repeated-measures analysis of variance there were no significant main effects of group (results not published). Thus, we found it reasonable to include both the intervention and control groups and to analyse these groups together in this study. The study was approved by the Regional Committee for Medical Research Ethics and the Privacy Issues Unit at Norwegian Social Science Data Services.

In total, 413 patients fulfilled the inclusion criteria and were invited to participate in the study. Of these, 125 patients (28%) were excluded because of the following reasons: not willing (83); contraindications, for example, other serious disease (13); administrative failure (13) and participating in other study (16). Thus, 288 patients remained for analyses. Among the included patients there were fewer females than among the excluded (19 vs. 33%; $P = 0.003$) and those included were younger (mean age 60.2 vs. 65.7 years; $P < 0.001$).

During the period from baseline to 18 months after discharge, 89 (31%) patients were lost to follow-up [24]. Those lost to follow-up were characterized by having a longer hospital stay (median 6 vs. 5 days; Wilcoxon–Mann–Whitney U test: $P = 0.023$), being more likely to be nonsmokers or smokers than exsmokers (Fisher's exact test: $P = 0.016$) and having higher baseline anxiety scores on the HADS than those remaining in the study (t-test: $P = 0.011$). Both remaining and lost to follow-up patients were comparable with respect to their other characteristics.

The Norwegian reference population

Reference population data on the HADS were obtained from the Nord-Trøndelag Health Study 1995–1997 (the HUNT 2 Study) [25,26]. Nord-Trøndelag is one of 19 counties in Norway, comprising 3% of the national population. Except for a somewhat lower mean level of education, the county is representative of Norway as a whole. The HUNT 2 study was carried out from 1995 to 1997. On the basis of the official population register, all inhabitants in the county aged at least 20 years were invited to participate. Data collection was performed using mailed questionnaires and a clinical examination. From 92 936 eligible individuals, 66 140 (71.2%) from the general population, including both the healthy and those with chronic or acute illness, participated in the HUNT 2 study [25,26]. In our study, 54 867 individuals with complete data on the HADS, smoking and education variables, and no previous self-reported cardiovascular disease, were included.

Data collection

Baseline demographic and clinical assessment

The following demographic and clinical variables were assessed at baseline from patient interviews and medical records: age, sex, marital status/cohabitation, employment, highest level of education attained, smoking habits, exercise habits, diabetes type I/II, hypercholesterolaemia, hypertension, body mass index, family history of CHD, previous CHD and previous AMI (Table 1). Variables related to the acute event were treatment with thrombolysis, primary percutaneous coronary intervention (PCI), total PCI, type ST-segment elevation MI, length of hospital stay and creatinine kinase-myocardial band (Table 1).

Table 1 Demographic and clinical characteristics at baseline of the patients with AMI from the Hordaland county, Norway ($N = 288$)

Demographic characteristics	
Age (years) *N*, mean (SD)	288, 60.2 (12.0)
Men, *n*/*N* (%)	233/288 (80.9)
Married/cohabitating, *n*/*N* (%)	225/284 (79.2)
Living alone, *n*/*N* (%)	52/286 (18.2)
Employment, *n*/*N* (%)	
Full time	129/282 (45.7)
Part time	17/282 (6.0)
Not working	136/282 (48.2)
Highest level of education, *n*/*N* (%)	
Primary school	85/257 (33.1)
High school	91/257 (35.4)
College/University	81/257 (31.5)
Clinical characteristics	
Previous CHD, *n*/*N* (%)	69/285 (24.2)
Previous AMI, *n*/*N* (%)	33/ 287 (11.5)
Family history of CHD, *n*/*N* (%)	170/253 (67.2)
Smoking habits, *n*/*N* (%)	
Daily smokers	138/285 (48.4)
Never smoked	63/285 (22.1)
Exsmokers	84/285 (28.5)
Exercise habits, *n*/*N* (%)	
Not exercising	35/239 (14.6)
Once weekly	58/239 (24.3)
2–3 times weekly	93/239 (38.9)
At least 4 times weekly	53/239 (22.2)
Diabetes (type I/II), *n*/*N* (%)	28/284 (9.9)
Hypercholesterolemia, *n*/*N* (%)	122/286 (42.7)
Hypertension, *n*/*N* (%)	85/287 (29.6)
Body mass index in kg/m^2, *N*, mean (SD)	277, 26.2 (3.5)
Thrombolysis, *n*/*N* (%)	38/288 (13.2)
Primary PCI, *n*/*N* (%)	94/288 (32.6)
Total PCI, *n*/*N* (%)	215/288 (74.7)
Length of stay in days, *N*, median (IQR)	285, 5 (4–7)
STEMI, *n*/*N* (%)	182/287 (63.4)
Peak CK-MB in U/l, *N*, median (IQR)	285, 102.0 (35.0–216.5)

AMI, acute myocardial infarction; CHD, coronary heart disease; CK-MB, creatinine kinase-myocardial band; IQR, interquartile range; Primary PCI, percutaneous coronary intervention within 24 h; SD, standard deviation; STEMI, ST-segment elevation myocardial infarction; Total PCI, PCI during hospital stay.

Symptoms of anxiety and depression

The HADS, consisting of a seven-item subscale for anxiety (HADS-A) and a seven-item subscale for depression (HADS-D), was used to assess symptoms of anxiety and depression [27]. The HADS was developed so as not to be affected by physical symptoms. Each item has a four choice response with scores ranging from 0 for no symptoms to 3 for the maximum number of symptoms. The scores on each subscale range from 0 to 21. The HADS, which is extensively used, has optimal cut-off scores of at least 8 on both subscales for identifying possible cases of anxiety and depressive disorders [28]. The HADS has demonstrated good psychometric properties in AMI patients [18], in the normal population and in somatic, psychiatric and primary care patients [28].

Lifestyle changes

Lifestyle outcomes included the assessment of exercise and smoking habits. With respect to exercise, the question asked was 'How often on average do you exercise each week?' The response options were none, once, 2–3 times and four times or more. Changes in exercise habits from baseline to 3 months, and from baseline to 18 months after discharge were calculated. A change of plus or minus one step indicated that the person had changed up (increased exercise) or down (decreased exercise) one level. The maximum change could be plus or minus three levels. Total lifestyle change from baseline to 18 months after discharge was measured by a variable combining change in exercise and the termination of smoking, using the variable indicating change in exercise habits from baseline to 18 months and the termination of smoking variable. Patients who

managed to stop smoking during the 18-month follow-up were given a score of two levels of improvement and those who did not change their smoking habits were given no additional improvement credit to produce a change in the exercise habit variable. Thus, the theoretical range for the total lifestyle change variable was from −3 to 5.

Statistical analysis

Descriptive statistics for approximately normally distributed continuous variables are presented as means and standard deviations, and as median values and interquartile ranges for those continuous variables with non-normal distributions. Demographic and clinical variables at baseline were compared between those with HADS anxiety and depression scores less than 8 and at least equal to 8 using Fisher's exact test, Student's *t*-test or the Wilcoxon–Mann–Whitney *U* test, as appropriate. Using the same procedures, differences were also analysed between those participants who were included and excluded, and between those who remained in the study and those who were lost to follow-up from discharge to 18 months after discharge. To analyse relationship between the HADS anxiety and depression subscales at each measurement point, Pearson's correlation was used.

Expected scores for the clinical sample were calculated using the results from a linear regression analysis of the HADS depression and anxiety subscales in the reference population, controlling for age, sex, educational level and smoking habits (given in Table 2). Both datasets were analysed together to compare the clinical sample with the Norwegian reference population on the HADS. A categorical variable was defined to model subgroups (0 = reference population, 1 = clinical sample). Using linear regression analysis and controlling for age, sex, educational level and smoking habits at baseline, the clinical sample scores at each measurement point were compared with the reference population scores.

Table 2 Formulas for expected HADS scores based on Norwegian general reference population (the Nord-Trøndelag Health Study, HUNT 2 1995–1997) [25,26]

Expected HADS anxiety scores

$$Y = 4.036 - 0.017 \times \text{age} + 0.629 \times \text{sex} - 0.287 \times \text{educational level} + 0.319 \times \text{smoking habits}$$

Expected HADS depression scores

$$Y = 2.376 + 0.034 \times \text{age} - 0.256 \times \text{sex} - 0.348 \times \text{education level} + 0.227 \times \text{smoking habits}$$

Variable definitions

- Age (years)
- Sex
  - 1 = male
  - 2 = female
- Educational level
  - 1 = primary school
  - 2 = high school
  - 3 = college/university
- Smoking habits
  - 0 = never smoked
  - 1 = exsmoker
  - 2 = smoker

HADS, Hospital Anxiety and Depression Scale.

To analyse predictors of anxiety and depression after 18 months, multiple linear regression analyses were performed using the general linear model (GLM: UNIANOVA) module of SPSS (SPSS Inc., Chicago, Illinois, USA) [29]. The categorical variables were: sex, group, cohabitation, education, diabetes, complications, total PCI and smoking habits; and the continuous variables were: age, bed days, creatinine kinase-myocardial band level, HADS scores at baseline and after 3 months, change in exercise habits from baseline to 3 months after discharge and change in both exercise and smoking habits at 18 months. Models were reported as estimated regression coefficients (*B*) together with their associated *P* values, 95% confidence interval and unadjusted and adjusted coefficients of determination (R^2). Using a fully adjusted model, a backward stepwise elimination of predictors was performed until only significant predictors remained in the final model. All tests were two-tailed at the 5% significance level. Data were analysed using SPSS version 15.0. (SPSS Inc.)

Results

Characteristics of the acute myocardial infarction patients

The baseline and clinical characteristics for the total cohort ($n = 288$) are described in Table 1. The typical participant was male, 60 years old, married or cohabitating, with a family history of CHD. Typically, the patient was a daily smoker, had a ST-segment elevation MI, received PCI during hospitalization and stayed in hospital for 5 days after the AMI. Patients with anxiety scores at least equal to 8 were significantly younger (54.5 vs. 61.9 years) and exercised less often compared with those whose scores were less than 8. Patients with a depression score at least equal to 8 had significantly more diagnosis of diabetes (type I/II) (15.6% vs. 7.7%) compared with those whose score was less than 8. There were no significant differences for the other demographic and clinical characteristics.

Symptoms of anxiety and depression and comparison with the reference population

At baseline, 19.7% of the patients showed symptoms of HADS anxiety with scores that were at least equal to 8. During follow-up, the prevalence of anxiety symptoms decreased. After 3, 6, 12 and 18 months, the percentages of patients with such symptoms were 16.1, 16.5, 14.1 and 16.8%, respectively (Table 3). At baseline, 13.6% of patients had symptoms of HADS depression, as indicated by a score at least equal to 8. In addition, after 3, 6, 12 and 18 months, the percentages were 13.4, 14.7, 10.2 and 13.7%, respectively (Table 3). As expected there was a significant relationship between the HADS anxiety

Table 3 HADS anxiety and depression scores at baseline and 3, 6, 12 and 18 months after discharge following acute myocardial infarction in 244 patients from the Hordaland county, Norway and comparison with reference population

	Baseline	3 months	6 months	12 months	18 months
HADS anxiety, *N*	244	224	212	206	197
Mean score (SD)	4.71 (3.57)	4.20 (3.72)	4.23 (3.65)	3.93 (3.31)	4.09 (3.37)
<8 (normal)	80.3%	83.9%	83.5%	85.9%	83.2%
≥ 8 (cases)	19.7%	16.1%	16.5%	14.1%	16.8%
Mean (SD) expected scores[a]	3.57 (0.50)				
Difference clinical sample vs. reference[b]	0.828	0.315	0.286	0.004	0.278
P value[c]	<0.001	0.173	0.226	0.985	0.260
HADS depression, *N*	244	224	212	206	197
Mean score (SD)	3.66 (3.04)	3.29 (3.25)	3.65 (3.45)	3.47 (3.02)	3.50 (3.30)
<8 (normal)	86.4%	86.6%	85.3%	89.8%	86.3%
≥ 8 (cases)	13.6%	13.4%	14.7%	10.2%	13.7%
Mean (SD) score reference population[a]	3.69 (0.53)				
Difference clinical sample vs. reference[b]	−0.330	−0.623	−0.318	−0.481	−0.419
P value[c]	0.092	0.002	0.129	0.024	0.055

[a]Expected scores, formula in Table 2, based on Norwegian reference population of *N*=54 867 (the Nord-Trøndelag Health Study, HUNT 2 Study) [25,26] without previous CVD adjusted to the age, sex, education and smoking habits distribution at baseline in the total clinical cohort (*n*=257). [b]Unstandardized coefficient B from linear regression analysis adjusted for age, sex, education, and smoking habits. Positive value indicates that clinical sample score higher (more symptoms) and negative value indicates that clinical sample score is lower (less symptoms) than in the reference population. [c]*P* value from linear regression analysis on difference on Hospital Anxiety and Depression Scale (HADS) Anxiety/Depression scores between clinical cohort and reference population without CVD, at the different measurement points adjusted for age, sex, education and smoking habits.

and depression subscales at all measurement points (Pearson's correlation coefficient ranging from 0.56 to 0.68, $P < 0.001$).

The mean anxiety scores in the clinical cohort at each measurement point were compared with the mean score in the Norwegian reference population, adjusting for age, sex, educational levels and smoking habits at baseline (Table 3). At baseline, the clinical cohort had a mean HADS anxiety score that was 0.828 ($P < 0.001$) points higher than that for the reference population. No significant differences were found at the other measurement points. When comparing the HADS depression scores, the clinical cohort scored lower than the reference population at all measurements points and significantly lower at 3 and 12 months after discharge (Table 3).

Predictors of symptoms of anxiety and depression after 18 months

Unadjusted and adjusted models of predictors on HADS anxiety and depression scores, respectively, after 18 months are shown in Table 4.

The final model predicting HADS anxiety score after 18 months, which included HADS anxiety score at baseline (B: 0.31, $P < 0.001$) and after 3 months (B: 0.46, $P < 0.001$), explained 54% of the variance. Higher anxiety at baseline and at 3 months predicted higher anxiety scores at 18 months.

In the final model predicting HADS depression after 18 months, 62% of the variance was explained by the variables: complications (B: 1.36, $P = 0.001$); bed days (B: −0.11, $P = 0.014$); depression scores at baseline (B: 0.27, $P < 0.001$) and after 3 months (B: 0.59, $P < 0.001$) and improvement in lifestyle after 18 months (B: −0.35, $P < 0.005$). Higher depression scores at 18 months was thus predicted by more complications, shorter hospital stay, depression scores at 3 and 6 months and no improvement in lifestyle over 18 months.

Discussion

High levels of anxiety symptoms were reported in 20% of the AMI patients at baseline, the percentage decreasing to between 14 and 17% during the 18-month follow-up period. The percentage of patients with depressive symptoms was 14% at baseline and was between 10 and 15% during the follow-up period. The study's most unexpected finding was that AMI patients reported symptoms of anxiety and depression to a much smaller extent than anticipated when compared with earlier research. Compared with the Norwegian general reference population, AMI patients were more anxious 1 week after discharge. However, with respect to depression, they were comparable with or scored lower than the reference population during the 18-month follow-up period.

This surprising improvement in symptoms of anxiety and depression for the AMI patients, when compared with the results obtained in studies conducted a decade ago, might reflect the psychological effects of a reduction in physical morbidity and mortality that has been reported recently. This reduction is because of better medical and surgical treatments, an increase in the frequency of coronary interventions and improved secondary prevention measures [2,22,30,31]. According to Lazarus and Folkman's [32] theory of stress, appraisal and coping, the appraisal of threat when experiencing a stressful situation influences both coping in that situation and emotional reactions such as anxiety. Therefore, the possible mechanisms explaining the reductions in anxiety and depression found in this study might involve a change

Table 4 Multiple linear regression analysis of HADS anxiety and depression scores after 18 months on potential predictors

	HADS anxiety score						HADS depression score					
	Unadjusted model			Fully adjusted model			Unadjusted model			Fully adjusted model		
Predictors	*B*	95% CI	*P* value	*B*	95% CI	*P* value	*B*	95% CI	*P* value	*B*	95% CI	*P* value
Age/10 years	0.11	(−0.53 to 0.31)	0.603	0.03	(−0.39, 0.46)	0.883	0.40	(−0.00 to 0.81)	0.052	0.14	(−0.25 to 0.52)	0.493
Sex												
Male (1) vs. female (0)	−1.13	(−2.34 to 0.09)	0.070	0.08	(−1.05 to 1.22)	0.887	−0.34	(−1.54 to 0.87)	0.581	0.47	(−0.57 to 1.50)	0.376
Cohabiting												
No (1) vs. yes (0)	0.40	(−0.84 to 1.63)	0.528	0.01	(−1.10 to 1.12)	0.986	0.64	(−0.58 to 1.87)	0.300	0.24	(−0.77 to 1.25)	0.640
Education level			0.592			0.151			0.863			0.214
Primary school (1) vs. College/ University >4 years (0)	0.98	(−0.77 to 2.72)		0.49	(−0.79 to 1.77)		0.24	(−1.46 to 1.94)		0.47	(−0.70 to 1.64)	
High school (1) vs. College/ University >4 years (0)	1.00	(−0.74 to 2.73)		1.28	(0.00 to 2.56)		0.51	(−1.19 to 2.20)		1.03	(−0.14 to 2.20)	
College/University <4 years (1) vs. College/University >4 years (0)	0.41	(−1.46 to 2.28)		0.75	(−0.61 to 2.10)		−0.03	(−1.86 to 1.80)		0.31	(−0.93 to 1.55)	
Smoking habits			0.213			0.082			0.816			0.270
Never (1) vs. smoker (0)	−0.65	(−1.93 to 0.63)		−0.80	(−1.96 to 0.37)		−0.32	(−1.58 to 0.94)		−0.36	(−1.43 to 0.70)	
Exsmoker (1) vs. smoker (0)	−0.92	(−1.99 to 0.14)		−1.19	(−2.24 to −0.14)		−0.29	(−1.34 to 0.76)		−0.78	(−1.74 to 0.18)	
Diabetes												
No (1) vs. yes (0)	−0.55	(−2.20 to 1.11)	0.514	−0.30	(−1.48 to 0.88)	0.612	−0.39	(−2.01 to 1.23)	0.638	0.18	(−0.90 to 1.26)	0.745
No. of risk factors (0–5)	0.26	(−0.21 to 0.73)	0.272	−0.09	(−0.54 to 0.35)	0.677	−0.13	(−0.58 to 0.33)	0.590	−0.15	(−0.56 to 0.25)	0.451
Group												
Control (1) vs. intervention (0)	0.17	(−0.78 to 1.12)	0.729	−0.01	(−0.75 to 0.72)	0.971	−0.09	(−1.02 to 0.84)	0.855	−0.61	(−1.28 to 0.06)	0.075
CK-MB (U/l)	0.00	(−0.00 to 0.00)	0.852	0.00	(−0.00 to 0.00)	0.516	−0.00	(−0.00 to 0.00)	0.164	0.00	(−0.00 to 0.00)	0.987
No PCI during hospital stay (1) vs. PCI (0)	0.18	(−0.99 to 1.35)	0.764	0.54	(−0.42 to 1.50)	0.269	0.32	(0.83 to 1.47)	0.582	0.35	(−0.53 to 1.22)	0.438
No complications during stay (1) vs. complications (0)	−0.71	(−1.89 to 0.46)	0.233	−0.95	(−1.90 to 0.01)	0.052	−1.66	(−2.79 to −0.53)	0.004	−1.22	(−2.09 to −0.35)	0.007
Bed days (1–218)	0.04	(−0.09 to 0.18)	0.516	−0.08	(−0.18 to 0.03)	0.137	0.06	(−0.07 to 0.19)	0.369	−0.12	(−0.22 to −0.03)	0.013
HADS anxiety baseline	0.63	(0.52 to 0.73)	<0.001	0.33	(0.14 to 0.52)	0.001	0.38	(0.25 to 0.51)	<0.001	0.10	(−0.08 to 0.27)	0.275
HADS anxiety 3 months after discharge	0.67	(0.56 to 0.77)	<0.001	0.47	(0.27 to 0.66)	<0.001	0.42	(0.29 to 0.55)	<0.001	−0.02	(−0.20 to 0.16)	0.844
HADS depression baseline	0.53	(0.39 to 0.67)	<0.001	0.00	(−0.19 to 0.19)	0.996	0.66	(0.54 to 0.79)	<0.001	0.26	(0.08 to 0.43)	0.004
HADS depression 3 months after discharge	0.56	(0.43 to 0.38)	<0.001	0.04	(−0.16 to 0.24)	0.693	0.73	(0.63 to 0.84)	<0.001	0.58	(0.40 to 0.77)	<0.001
Exercise improvement from baseline to 3 months	−0.19	(−0.64 to 0.27)	0.417	0.01	(−0.38 to 0.39)	0.980	−0.49	(−0.93 to −0.04)	0.034	0.11	(−0.24 to 0.47)	0.536
Lifestyle improvement from baseline to 18 months	−0.04	(−0.43 to 0.34)	0.825	−0.32	(−0.69 to 0.05)	0.090	−0.29	(−0.67 to 0.09)	0.129	−0.50	(−0.83 to −0.16)	0.004
Constant				1.73	(−2.10 to 5.56)					1.22	(−2.28 to 4.71)	
Determination coefficients				R^2=0.64, adjusted R^2=0.58						R^2=0.70, adjusted R^2=0.65		

CK-MB, creatinine kinase-myocardial band; HADS, Hospital Anxiety and Depression Scale; PCI, percutaneous coronary intervention.

in the patients' appraisal of AMI as a life-threatening event compared with what was previously the case. This might be because of patients, families and friends knowledge about the present AMI treatment and course, based on input from healthcare workers and the media, and the patients' experience of less physical discomfort and chest pain after the AMI, compared with the experiences of previous AMI patients. This explanation accords with the findings of a qualitative study in which post-AMI anxiety was related to an impending threat to existential values, and additional symptoms such as angina reminded the informants about the AMI and its threat of possible reinfarction and sudden death [33]. With a more positive perception of the illness, including a positive outlook on future health and functioning after an AMI, the psychological sequelae of an AMI might be attenuated. Hence, patients report improved levels of mental HRQOL, as was found in our earlier studies in which AMI patients' mental HRQOL levels were similar to those of the normal population [23,24]. Less frequent occurrences of depressive symptoms, similar to the findings in our study, have been reported in recent studies [11,18,34]. As expected and in accordance with resent research, the HADS anxiety and depression subscales were highly correlated [35,36].

Anxiety scores at baseline and at 3 months were the best predictors of anxiety at 18 months. The best predictors of depression at 18 months were depression level at baseline and after 3 months, complications during hospital stay, lifestyle improvement and bed days. Thus, screening for symptoms of anxiety and depression both immediately after the event as well as after 3 months can be useful for identifying patients at risk for psychological sequel after AMI. Our results are consistent with another study in which the best predictor of distress at 12 months was distress at 3 months and where distressed patients exercised less and smoked more than nondistressed patients [12]. Positive psychological effects, such as reductions in anxiety and depression from increased physical activity, have been reported in earlier research for both cardiac and other populations, although the mechanisms explaining this effect have been less clearly identified [35,37]. There is some evidence that nicotine (smoking) is a self-administered antidepressant for some patients [38]. This may partly explain why depressed patients tend to smoke more than nondepressed patients. In a large Norwegian population study with follow-up, the relative impact on long-term anxiety and depression of having a single first AMI during the follow-up period was quite moderate and weaker than the effects of many preexisting sociodemographic, lifestyle and psychological risk factors [39]. The relationship between lifestyle changes and depression could be explained by Lazarus and Folkman's theory [32] and related theories. According to these theories, one's ability to succeed in adaptively coping with lifestyle changes affects and improves in turn one's perceived self-efficacy and appraisal of one's own coping resources and vice versa. Accordingly, behaviour as well as motivation, thinking patterns and well-being will continue to either improve or worsen [32,40].

After controlling for complications, patients with a longer hospital stay were less depressed than patients who had a shorter hospital stay. Although the results from a study published 8 years ago concluded that hospitalization of patients with an uncomplicated AMI for more than 3 days is economically unattractive by conventional standards [41], it has been claimed that a minimum hospital stay after an AMI is needed to properly prepare patients for dealing with the consequences of the disease [42]. With reference to stress and coping theory [32], higher levels of depression among those with the shortest hospital stays might be explained as a reaction to not having been prepared sufficiently in coping tasks that are necessary for successful rehabilitation. Another explanation is that too little time was spent in hospital assessing and addressing patients' psychosocial problems, especially when combined with a lack of services for AMI patients once they have been discharged from hospital.

Strengths and limitations

The strengths of this study are its relatively large sample size and the multiple follow-up assessments of anxiety and depression. Repeated measures of anxiety and depression capture initial anxiety and depression after discharge from hospital, short-term and long-term changes in anxiety and depression, as well as any long-term exposure to anxiety and depression. A second strength of our study is its use of participants recruited from the general population of AMI patients who were able to participate in a study where they had access to a telephone and could receive calls after discharge. In many other studies, the participants were recruited from more demanding cardiac rehabilitation services involving, for example, travel, staying away from home, costs, etc. As cardiac rehabilitation services are underutilized in most countries [43,44], those attending cardiac rehabilitation are probably not representative of AMI patients in general [45], and the results from our study can possibly be generalized to a wider population of AMI patients.

The main limitation of this study was the loss to follow-up, which provided a threat to its external validity. Thirty-one per cent of the patients were lost during the four follow-up assessments, compared with the 5–10% lost to follow-up between each of the different assessment points reported in similar studies [8,18,46–48]. Those not included in our study are similar to those who

are usually not well represented in clinical research, that is, people who are elderly or female [45,46]. Thus, our sample can be considered to be representative of those AMI patients who usually participate in research. Hence, our results can be generalized cautiously to AMI patients in general who receive similar AMI and post-AMI care. However, caution should be exercised when extrapolating the findings to those who are elderly or female. Another limitation is the 4–10 years time difference between collections of data in the clinical versus the reference population. However, we justify the use of this reference population as we are not aware of studies indicating that the normal population levels in symptoms of anxiety and depression have changed during this time, and thus this allowed us to control for age, sex, educational levels and smoking habits; all related to symptoms of anxiety and depression in previous research.

Conclusion

About 1 week after discharge many AMI patients experience symptoms of anxiety. However, 3–18 months after the event, AMI patients were not more anxious or depressed than the Norwegian general reference population. Psychological morbidity in AMI patients, as indicated by symptoms of anxiety and depression, seems to have declined compared with levels reported a decade ago. However, patients showing symptoms of anxiety and depression after discharge following an AMI are at risk for experiencing a persistence of the same symptoms. Assessment and treatment of anxiety and depression, and encouraging lifestyle changes after AMI, continue to be important in post-AMI care that maximizes the outcomes for AMI patients.

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