Somatic Versus Cognitive Symptoms of Depression as Predictors of All-Cause Mortality and Health Status in Chronic Heart Failure

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Objective: Depression is a predictor of adverse health outcomes in chronic heart failure (CHF), but it is not known whether specific symptoms drive this relationship. We examined the impact of somatic/affective, cognitive/affective, and total depressive symptoms on all-cause mortality and health status in CHF.

Method: Consecutive CHF outpatients (n = 366) completed the Beck Depression Inventory. The primary endpoint was all-cause mortality; the secondary endpoint was disease-specific health status, as measured by the Minnesota Living with Heart Failure Questionnaire (n = 285) at inclusion and 1-year follow-up. The study was conducted between October 2003 and March 2007.

Results: There were 68 (18.6%) deaths (mean ± SD follow-up, 37.2 ± 10.6 months). Patients high on somatic/affective depressive symptoms had a greater incidence of mortality compared to patients low on somatic/affective depressive symptoms (31% vs 15%; hazard ratio [HR] = 2.3; 95% CI, 1.38–3.69; $P = .001$). There was no significant difference in the incidence of mortality between patients high versus low on cognitive/affective depressive symptoms (23% vs 18%; HR = 1.4; 95% CI, 0.80–2.40; $P = .25$), but there was a significant difference between patients high versus low on total depressive symptoms (24% vs 16%; HR = 1.6; 95% CI, 1.01–2.63; $P < .05$). After adjusting for demographic and clinical characteristics, we found that somatic/affective depressive symptoms predicted all-cause mortality (HR = 1.8; 95% CI, 1.03–3.07; $P = .04$), while cognitive/affective and total depressive symptoms did not. Both dimensions of depressive symptoms predicted disease-specific health status at 1 year.

Conclusions: Only somatic/affective depressive symptoms significantly predicted all-cause mortality in CHF. In the context of diagnosing and intervening, awareness of subtypes of depressive symptoms is important.

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There is an ongoing debate as to whether depressive symptoms following myocardial infarction reflect psychological comorbidity or the underlying heart disease.[1,2] In this context, it is important to note that depression is not a homogeneous construct but consists of somatic/affective as well as of cognitive/affective symptoms.[2,3] In several studies focusing on patients with coronary heart disease (CHD), these 2 dimensions of depressive symptoms seem to be differently associated with important health outcomes. The Heart and Soul Study[3] found that the somatic but not the cognitive dimension of depression was associated with lower heart rate variability in patients with stable CHD. Similarly, in post–myocardial infarction patients enrolled in the Myocardial INfarction and Depression-Intervention Trial (MIND-IT) and the Depression after Myocardial Infarction study, only the somatic symptoms of depression were related to cardiovascular prognosis.[2] Thus, it seems that, in particular, somatic/affective symptoms of depression are associated with impaired prognosis in CHD.

Chronic heart failure (CHF) is often the end stage of CHD and has a poor prognosis.[4,5] The prevalence of depressive symptoms in CHF varies according to the sample characteristics, but depression and depressive symptoms are common in CHF.[6–11] However, the condition of CHF itself is characterized by symptoms that highly overlap with somatic/affective symptoms of depression, such as fatigue, loss of appetite, and sleep difficulties.[6,7,12] Previous studies have shown that the somatic/affective dimension of depression was strongly associated with left ventricular dysfunction in post–myocardial infarction patients.[2,13] A recent study,[12] however, showed that somatic/affective depressive symptoms did not differ between depressed patients with and without CHF and that the cognitive/affective symptoms of depression are essential in discriminating between these 2 groups of patients. Therefore, there is a risk of assigning symptoms to the wrong condition and thus the prevalence

of depression and depressive symptoms may be overestimated in heart disease in general,[13] and, in particular, in CHF, but there may also be a risk for low recognition of depression in CHF.[12] Furthermore, because of the association between somatic/affective symptoms of depression and left ventricular dysfunction,[2,13] the relationship between depression and mortality[8,14] and health status[15] in CHF may be limited to the somatic dimension.

Until now, no study has examined the impact of the different dimensions of depressive symptoms on health outcomes (ie, prognosis and health status) in CHF. Hence, we examined the predictive value of the somatic/affective versus the cognitive/affective dimension of depressive symptoms on (1) all-cause mortality and (2) disease-specific health status in patients with established CHF.

METHOD

Participants

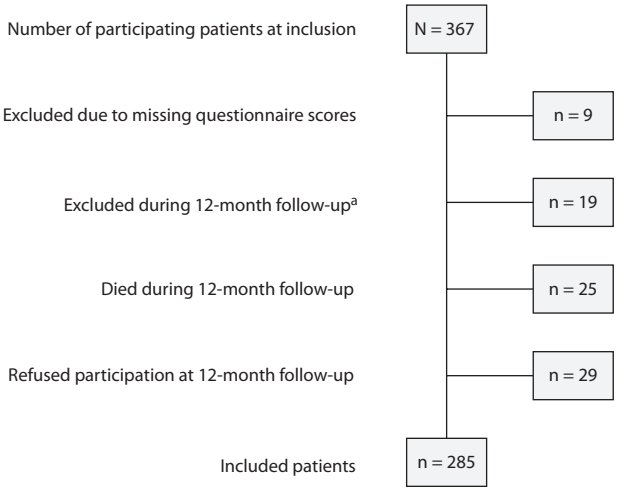
The sample comprised consecutive CHF outpatients visiting the Cardiology department of the TweeSteden teaching hospital in Tilburg and Waalwijk, the Netherlands. Inclusion criteria for the current study were (1) left ventricular ejection fraction (LVEF) ≤ 40%, (2) aged ≤ 80 years, and (3) pharmacologically stable for at least 1 month preceding inclusion. Patients who (1) were older than 80 years, (2) had diastolic heart failure, (3) were incapable of understanding and reading Dutch, (4) had evident cognitive impairments, or (5) had life-threatening comorbidities were excluded.

Of all patients approached for participation, 78.3% (367/469) agreed. We lost no patients to follow-up, but 1 patient had to be excluded from statistical analyses because of too many missing values on the Beck Depression Inventory (BDI)[16]; therefore, analyses on mortality are based on 366 patients. Final analyses on the association between depressive symptoms and health status are based on 285 patients for whom complete data were available at inclusion and 1-year follow-up (Figure 1). The study was conducted between October 2003 and March 2007.

All patients were treated following the most recent guidelines for CHF.[17,18] The study was approved by the hospital medical ethics committee and carried out according to policies to protect human subjects as formulated by the World Medical Association, described in the Helsinki Declaration (2004).

Patients were informed about the study and asked to participate by their cardiologist or specialized CHF nurse. If patients agreed to participate, they were called the same week to set up a first appointment for the study, written informed consent was obtained from all patients, and they were asked to complete the BDI[16] and the Minnesota Living with Heart Failure Questionnaire (MLHF)[19] at home and return them in a preaddressed, stamped envelope. One year later, the patients were contacted to complete the MLHF again. Patients who did not complete all of the items were contacted by phone in an attempt to obtain the answers, or they were mailed a copy of the items and asked to complete them. If the questionnaires were not returned within 2 weeks, patients received a reminder telephone call or letter.

Figure 1. Flowchart of Patient Selection (12-month follow-up)



[a]These patients were excluded due to medical (eg, cerebrovascular accident during the study) or logistic (eg, moving abroad) reasons.

Symptoms of Depression

Depressive symptoms were assessed with the BDI. The 21 items are rated on a scale from 0 to 3, with the total score ranging from 0 to 63.[16] The BDI has good psychometric properties, also in nonpsychiatric samples (Cronbach α = .81).[20] Various studies have reported on the factor structure of the BDI, and, in general, 2 or 3 dimensions are found.[2] In this study, the somatic/affective and cognitive/affective dimensions[2] and the total scale score[16] were used.

Endpoints

The primary endpoint of this study was all-cause mortality. Information on mortality (date and cause of death) was collected by checking the patients' medical records or by contacting the general practitioner on April 1, 2008.

The secondary endpoint was defined as disease-specific health status at 1-year follow-up, as measured by the MLHF.[19] The scale consists of 21 items that are answered on a 6-point Likert scale, ranging from 0 ("no") to 5 ("very much"); a higher score on the MLHF represents a poorer health status. The MLHF consists of 2 dimensions (ie, a physical and an emotional/mental dimension),[21,22] but the total score can also be used[19] as has been done in the current study. The MLHF has solid psychometric properties, with good internal consistency (Cronbach α = .91–.96).[21]

Sociodemographic and Clinical Variables

Sociodemographic variables (ie, age, gender, marital status, educational level, and working status) were measured by separate, purposed-designed questions in the questionnaire. Smoking was also assessed by means of self-report. Information on clinical variables, ie, disease characteristics (LVEF, New York Heart Association [NYHA] functional class, etiology), comorbidities and risk factors (hypertension, diabetes mellitus, hypercholesterolemia, previous cardiac history, kidney disease, liver disease, gastrointestinal problems, cerebrovascular problems, chronic obstructive pulmonary disease, and peripheral arterial disease), and commonly prescribed medications (angiotensin-converting enzyme [ACE] inhibitors, angiotensin receptor blockers, diuretics, spironolactone, digitalis, β-blockers, nitrates, aspirin, statins, and psychotropic medication) was obtained from the patients' medical records and the treating cardiologist/CHF nurse at the time of inclusion into the study.

Statistical Analyses

Discrete variables were compared by using the χ^2 test and continuous variables by using the Student *t* test for independent samples. The Kaplan-Meier method was used to calculate the cumulative mortality rate at follow-up; log rank tests were performed to compare the mortality rate between patients (1) high versus low on somatic/affective depressive symptoms, (2) high versus low on cognitive/affective depressive symptoms, and (3) high versus low on total depressive symptoms. BDI scores were dichotomized by using the predetermined cutoff of 10 for the total score on the BDI[16,20] and the highest 20% for the 2 dimensions of depressive symptoms, ie, the somatic/affective and cognitive/affective dimensions.[2]

Univariable Cox proportional hazards regression analyses were conducted to assess the impact of somatic/affective, cognitive/affective, and total depressive symptoms[2,16] on all-cause mortality. We also examined the predictive value of the clinical and demographic variables listed in Table 1. NYHA class (NYHA I/II versus III/IV), etiology of CHF (ischemic versus nonischemic), marital status (living with a partner versus living without partner), work status (working versus not working), and years of education (8 years or fewer versus more than 8 years) were recoded into dichotomous variables. In multivariable Cox proportional hazards regression analysis, we adjusted for all covariates that were statistically significant in the univariable analysis, ie, LVEF, NYHA classification, smoking, comorbid kidney disease, prescription of nitrates, older age, and not working. Univariable and multivariable logistic regression analyses were used to examine the impact of depressive symptoms on the secondary outcome measure, disease-specific health status at 1-year follow-up. With the aim of enhancing clinical interpretability, several authors advise logistic regression analysis above linear regression analysis.[23,24] Prior to the logistic regression analyses, the scores on the MLHF (secondary outcome measure) were recoded into dichotomous variables. The highest tertile on the MLHF indicated impaired disease-specific health status.[25]

All analyses were performed using SPSS 14.0 for Windows (SPSS Inc, Chicago, Illinois). A *P* value of .05 was used to indicate statistical significance.

RESULTS

Patient Characteristics

The prevalence of somatic/affective depressive symptoms was 22%; of cognitive/affective depressive symptoms, 21%; and of total depressive symptoms, 36%. The mean ± SD score on the somatic/affective dimensions of the BDI was 4.6 ± 2.9 (range, 0–15); on the cognitive/affective dimensions, it was 3.7 ± 4.6 (range, 0–28); and on the total BDI, it was 9.2 ± 7.2 (range, 0–45).

In Table 1, patient characteristics, stratified by high versus low scores on somatic/affective depressive symptoms (cutoff, highest 20%[2]) and by high versus low scores on cognitive/affective depressive symptoms (cutoff, highest 20%[2]), are provided. There were significant differences between patients scoring high on somatic/affective depressive symptoms as compared to patients scoring low on somatic/affective depressive symptoms on some patient characteristics. High scorers more often were living without a partner, were not working, were in NYHA class III/IV, and had diabetes mellitus and gastrointestinal problems; more often were prescribed spironolactone, nitrates, and psychotropic medication; and less often were prescribed ACE inhibitors. When comparing patients high on cognitive/affective depressive symptoms to patients low on cognitive/affective depressive symptoms, high scorers were significantly more often living without a partner, had a lower educational level, more often had a cardiac history, and were less often prescribed β-blockers but more often nitrates and psychotropic medication. Finally, they had less often had CHF with an ischemic etiology (Table 1).

Depressive Symptoms and Mortality in CHF

Of 366 CHF patients, 68 (18.6%) died during the mean follow-up period of 37.2 months (SD = 10.6 months).

Patients high on somatic/affective depressive symptoms had a significantly higher all-cause mortality rate as compared to patients low on somatic/affective depressive symptoms (31% [25/80] vs 15% [43/286]; hazard ratio [HR] = 2.3; 95% CI, 1.38–3.69; *P* = .001), with the associated risk being 2-fold (Figure 2, left). There was no significant difference between patients high versus low on cognitive/affective depressive symptoms (23% [17/75] versus 18% [51/291]; HR = 1.4; 95% CI, 0.80–2.40; *P* = .25) (Figure 2, center), but there was a significant difference in all-cause mortality between patients high versus low on

Table 1. Patient Characteristics Stratified By Somatic/Affective and Cognitive/Affective Depressive Symptoms[a]

Characteristic	Somatic/Affective Symptoms			Cognitive/Affective Symptoms		
	High (n = 80)	Low (n = 286)	*P*	High (n = 75)	Low (n = 291)	*P*
Sociodemographics						
Age, mean (SD), y	66 (10)	65 (10)	.51	65 (11)	66 (10)	.29
Male gender	64 (51)	74 (211)	.08	75 (56)	71 (206)	.51
Living without a partner	44 (35)	25 (72)	.001**	45 (34)	25 (73)	< .001**
< 8 Years of education	43 (34)	32 (90)	.07	44 (33)	31 (91)	.04*
Not working	94 (75)	83 (237)	.02*	85 (64)	85 (248)	.98
Disease characteristics						
LVEF, mean (SD)	31 (7)	31 (7)	.91	31 (7)	30 (7)	.46
NYHA class III/IV	53 (42)	37 (105)	.01*	44 (33)	39 (114)	.45
Ischemic etiology	58 (46)	52 (150)	.43	35 (26)	50 (144)	.02*
Comorbidities and risk factors						
Hypertension	46 (37)	37 (106)	.14	40 (30)	39 (113)	.85
Diabetes mellitus	38 (30)	22 (64)	.006**	27 (20)	25 (74)	.83
Smoking	28 (22)	22 (64)	.34	29 (22)	22 (64)	.18
Hypercholesterolemia	48 (38)	49 (140)	.81	47 (35)	49 (143)	.70
Previous cardiac history[b]	63 (50)	56 (159)	.27	68 (51)	54 (158)	.03*
Kidney disease	20 (16)	13 (36)	.09	13 (10)	14 (42)	.81
Liver disease	5 (4)	4 (12)	.76	7 (5)	4 (11)	.28
Gastrointestinal problems	14 (11)	5 (14)	.006**	9 (7)	6 (18)	.34
Cerebrovascular problems[c]	20 (16)	13 (36)	.09	17 (13)	13 (39)	.39
COPD	15 (12)	12 (34)	.46	11 (8)	13 (38)	.57
Peripheral arterial disease	15 (12)	13 (38)	.69	19 (14)	12 (36)	.16
Medication						
ACE inhibitors	63 (50)	76 (216)	.02*	76 (57)	72 (209)	.47
Angiotensin receptor blockers	21 (17)	18 (50)	.44	11 (8)	20 (59)	.06
Diuretics	85 (68)	75 (214)	.06	81 (61)	76 (221)	.32
Spironolactone	33 (26)	20 (56)	.01*	27 (20)	21 (62)	.32
Digitalis	24 (19)	26 (73)	.74	20 (15)	27 (77)	.25
β-blockers	54 (43)	71 (202)	.10	55 (41)	70 (204)	.01*
Nitrates	35 (28)	19 (54)	.002**	32 (24)	20 (58)	.03*
Aspirin	41 (33)	42 (120)	.91	41 (31)	42 (122)	.93
Statins	48 (38)	51 (146)	.58	56 (42)	49 (142)	.27
Psychotropic medication	24 (19)	9 (26)	< .001**	23 (17)	10 (28)	.002**

[a]Results are presented as % (n) unless otherwise stated.
[b]Myocardial infarction, percutaneous coronary intervention, coronary artery bypass graft surgery.
[c]Cerebrovascular accident, transient ischemic attack.
Abbreviations: ACE = angiotensin-converting enzyme, COPD = chronic obstructive pulmonary disease, LVEF = left ventricular ejection fraction, NYHA = New York Heart Association.
*$P < .05$.
**$P < .01$.

total depressive symptoms (24% [31/130] vs 16% [37/236]; HR = 1.6; 95% CI, 1.01–2.63; $P < .05$) (Figure 2, right). Kaplan-Meier curves for somatic/affective depressive symptoms, cognitive/affective depressive symptoms, and total depressive symptoms are presented in Figure 3.

In univariable analysis, we found older age, having no work, lower LVEF, higher NYHA class III/IV, smoking, comorbid kidney disease, and the prescription of nitrates to be significant predictors of all-cause mortality in CHF (Table 2). After adjusting for these covariates and cognitive/affective depressive symptoms, we found that somatic/affective depressive symptoms remained an independent predictor of all-cause mortality (Table 3, upper part). Total depressive symptoms were not predictive of all-cause mortality in the multivariable model ($P = .37$). Older age, lower LVEF, smoking, and comorbid kidney disease were significant independent predictors of all-cause mortality in the current study (Table 3, upper part).

Depressive Symptoms and Disease-Specific Health Status in CHF

We examined whether the somatic/affective and cognitive/affective dimensions of depressive symptoms and total depressive symptoms would predict disease-specific health status at 1-year follow-up. In univariable logistic regression analysis, somatic/affective depressive symptoms (OR = 3.5; 95% CI, 1.94–6.38; $P \leq .001$), cognitive/affective depressive symptoms (OR = 3.4; 95% CI, 1.97–5.84; $P \leq .001$), as well as total depressive symptoms (OR = 5.0; 95% CI, 2.93–8.47; $P \leq .001$) predicted impaired, disease-specific health status at 1 year.

When adjusting for confounders, we found that cognitive/affective (Table 3, lower part) and total depressive symptoms (OR = 3.68; 95% CI, 2.01–6.75; $P \leq .001$) still predicted impaired, disease-specific health status at 1-year follow-up. Although the relationship between somatic/affective depressive symptoms lost statistical significance

Figure 2. All-Cause Mortality Risk, Stratified by Somatic/Affective, Cognitive/Affective, and Total Depressive Symptoms

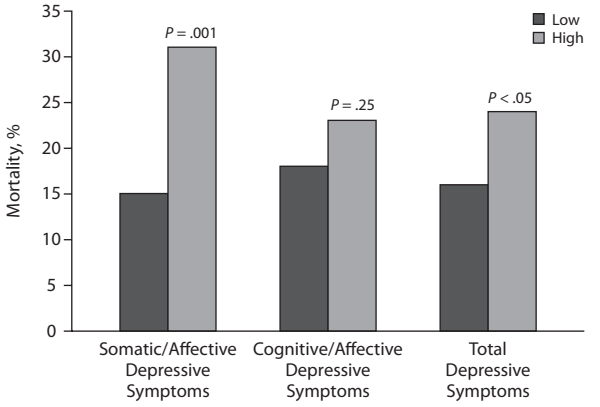


Figure 3. Kaplan-Meier Curves, Stratified by Somatic/Affective, Cognitive/Affective, and Total Depressive Symptoms

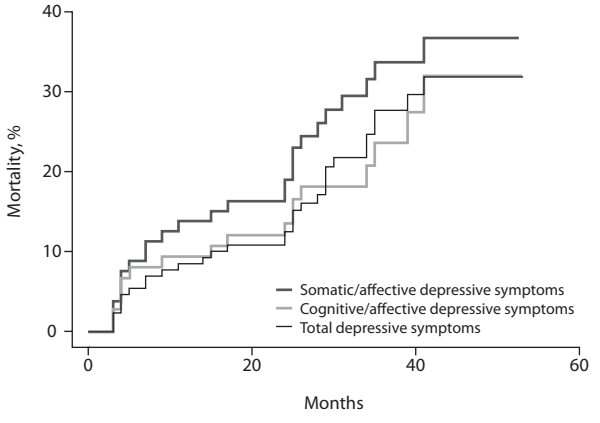


Table 2. Sociodemographic and Clinical Predictors of All-Cause Mortality (univariable analysis)

Predictor	HR	95% CI	*P*
Older age	1.1	1.02–1.09	< .001**
Not working	4.2	1.31–13.26	.016*
LVEF	0.9	0.89–0.96	< .001**
NYHA class III/IV	1.9	1.20–3.13	.007*
Smoking	1.9	1.15–3.10	.01*
Kidney disease	3.5	2.11–5.84	< .001**
Nitrates	1.7	1.02–2.81	.04*

Abbreviations: LVEF = left ventricular ejection fraction, NYHA = New York Heart Association.
*$P < .05$.
**$P < .01$.

Table 3. Depressive Symptom Clusters as Predictors of All-Cause Mortality and Disease-Specific Health Status (multivariable analysis)

Predictor	HR	95% CI	*P*
All-cause mortality			
Somatic/affective depressive symptoms	1.8	1.03–3.07	.04*
Cognitive/affective depressive symptoms	1.1	0.59–1.94	.83
Older age	1.0	1.00–1.01	.04*
Not working	1.9	0.55–6.72	.31
LVEF	0.9	0.91–0.97	≤ .001**
NYHA class III/IV	1.3	0.78–2.19	.32
Smoking	2.3	1.35–4.00	.002*
Kidney disease	2.8	1.65–4.87	≤ .001**
Nitrates	1.2	0.71–2.11	.47
	OR	95% CI	*P*
Disease-specific health status			
Somatic/affective depressive symptoms	2.0	0.95–4.07	.07
Cognitive/affective depressive symptoms	2.3	1.21–4.44	.01*
Older age	1.0	0.98–1.05	.48
Not working	2.0	0.78–5.25	.15
LVEF	1.0	1.00–1.09	.06
NYHA class III/IV	0.9	0.46–1.56	.59
Smoking	0.9	0.44–1.85	.79
Kidney disease	0.7	0.27–1.65	.39
Nitrates	1.5	0.74–2.84	.28
Health status at inclusion	3.3	1.75–6.05	≤ .001**

Abbreviations: LVEF = left ventricular ejection fraction, NYHA = New York Heart Association.
*$P < .05$.
**$P < .01$.

($P = .07$), there was still a trend for somatic/depressive symptoms to predict impaired health status (Table 3, lower part). In addition, lower LVEF and health status at inclusion were also important predictors of impaired, disease-specific health status at 1-year follow-up.

DISCUSSION

To our knowledge, this is the first study to examine whether somatic/affective versus cognitive/affective depressive symptoms have differential prognostic effects in CHF. Patients with a high total score on the BDI and on somatic/affective depressive symptoms were at a higher risk of all-cause mortality compared to patients low on these symptoms. There was no difference in all-cause mortality rates between patients high and low on cognitive/affective depressive symptoms. In adjusted analysis, the somatic/affective symptoms remained an independent predictor of all-cause mortality, with the associated risk being 2-fold, whereas the total BDI score fell short of significance.

Our results are not in line with a study by Frasure-Smith and Lespérance[26] who reported that both somatic and cognitive symptoms of depression were related to increased cardiac events over a period of 5 years, and, after adjustment for disease severity, in post–myocardial infarction patients. However, our results are consistent with those of another recent study on this topic.[2] De Jonge and colleagues[2] found somatic/affective, but not cognitive/affective, depressive symptoms to be predictive of cardiovascular death. Thus, in post–myocardial infarction as well as in CHF patients, depressive symptom clusters seem to exert differential prognostic effects. It has been speculated that

somatic depressive symptoms reflect underlying heart disease rather than psychological comorbidity.[3,13] However, the relationship between somatic/affective depressive symptoms and prognosis remained significant after adjusting for LVEF and NYHA class in the current study. In post–myocardial infarction patients, somatic/affective depressive symptoms also remained predictive of cardiovascular death after adjustment for baseline somatic health status (LVEF, Killip class, and previous myocardial infarction).[2] Furthermore, a recent study showed that there were no differences in the depressive profile between patients with and without CHF, apart from CHF patients reporting lower levels of depressed mood and worthlessness/guilt.[12] Therefore, it seems too premature to conclude that somatic/affective symptoms reflect the underlying heart disease rather than signs of a mood disturbance. Although the results of the current study do not provide a definitive answer to this intriguing question, they suggest that the relationship between depressive symptoms and prognosis in CHF may be limited to the somatic/affective dimension of depressive symptoms. Whether these symptoms are symptoms of depression or symptoms of CHF itself is, however, not clear.

The results have some implications for future research and clinical practice. First, it is important to acknowledge that different symptom clusters of depression may have differential prognostic effects in patients with CHF. Second, treatment of depression and depressive symptoms needs to target those clusters of depressive symptoms that seem to exert a detrimental effect. Until now, randomized controlled trials aimed at reducing the negative effects of depression on prognosis have produced mixed results. The Sertraline Antidepressant Heart Attack Randomized Trial (SADHART)[27] found sertraline to be a safe treatment of recurrent depression in post–myocardial infarction patients, and, although it was found that about 20% fewer cardiovascular events occurred in the sertraline group as compared to the placebo group, this difference was not significant. Furthermore, the study was not powered enough to investigate whether a reduction in depression following sertraline treatment would decrease mortality in post–myocardial infarction patients.[27,28] Subsequently, the MIND-IT found no effects of antidepressant medication on prognosis in coronary patients.[29] The Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD) Randomized Trial[30] found that cognitive-behavioral therapy did affect depression, but there were no effects on prognosis post–myocardial infarction. Finally, the Canadian Cardiac Randomized Evaluation of Antidepressant and Psychotherapy Efficacy (CREATE) trial[31] failed to document a benefit of interpersonal psychotherapy in comparison with clinical management of depression in patients with coronary artery disease. However, this latter study compared interpersonal psychotherapy with an intervention of clinical management given by an interpersonal psychotherapy–focused therapist. In the same study, an antidepressant effect of citalopram was documented.[31] Taken together, the findings of these intervention trials are disappointing. De Jonge and Ormel[32] very recently suggested that the identification of depression subtypes might enhance the diagnosing and treatment of depression in cardiac patients. The results of the current study and those in post–myocardial infarction patients[2] showed that different subtypes of depressive symptoms, namely somatic/affective and cognitive/affective depressive symptoms, are differently related to prognosis. Therefore, studies on pharmacologic and behavioral interventions aimed at reducing depression and depressive symptoms and its effects on prognosis should be aware of this distinction.

It is important to note, however, that the condition of depression itself has a negative impact on patients' health status and quality of life. In the current study, somatic/affective and cognitive/affective depressive symptoms were significant predictors of 1-year disease-specific health status in univariable analysis. The effect of somatic/affective depressive symptoms lost statistical significance when we adjusted for covariates, including health status at time of inclusion. However, we still found that patients reporting high levels of somatic/affective depressive symptoms were at a 2-fold increased risk of impaired disease-specific health status. Previous studies found depression to be an important independent predictor of health status in CHF,[15,33] and, as we previously reported, both dimensions of depression seem to be important in predicting health status over time in CHF.[33] Thus, regardless of whether treating depression or depressive symptoms can improve prognosis, depression should be treated in its own right.[28]

The following limitations of the current study must be acknowledged. First, depressive symptoms were measured with a self-report questionnaire, and, although the BDI is a reliable measure of depressive symptoms in patients with heart disease,[20] the possibility of socially desirable answers cannot be ruled out. Second, the multivariable Cox proportional regression analysis was somewhat overfitted. However, the overall results did not change when we removed working status and nitrates as covariates from the multivariable model. Third, the sample may be biased by mobility and younger age, since study participants were required to visit the outpatient clinic in order to be included in the study.

In conclusion, we found that, in particular, somatic/affective symptoms of depression were associated with an increased incidence of all-cause mortality in CHF patients, and the cognitive/affective symptoms primarily seemed to predict impaired health status. Clinicians who use pharmacologic and behavioral interventions aimed at reducing depression and depressive symptoms and their effects on health outcomes should be aware of these differential effects on outcomes.

Drug names: citalopram (Celexa and others), sertraline (Zoloft and others).

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