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Relations among depressive symptoms, electrocardiographic hypertrophy, and cardiac events in non-ST elevation ACS patients

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Abstract

AIMS—Cardiac outcomes after acute coronary syndrome (ACS) are worse in patients with depression, but identifying which depressed patients are at increased risk, and by what means, remains difficult.

METHODS AND RESULTS—We analyzed inpatient ECGs from 955 patients admitted with non-ST elevation ACS (NSTE-ACS) in the Prescription Use, Lifestyle, and Stress Evaluation (PULSE) study. Patients with QRS duration ≥120 milliseconds or whose rhythm was not normal sinus were excluded (sample size=769). Depressive symptoms were measured by Beck Depression Inventory score ≥10. ECG markers of ventricular hypertrophy included Cornell voltage-duration product (CP-LVH) and strain pattern in the lateral leads. In multivariable logistic regression models, depressive symptoms were associated with increased odds of CP-LVH, ECG-strain, and the combination of the two (odds ratios 1.74 to 2.33, p values <0.01). The combination of both CP-LVH and ECG-strain was predictive of one-year risk of myocardial infarction (MI) or death among patients with depressive symptoms (hazard ratio 4.91, 95% CI 1.55–15.61, p=0.007), but not among those without depressive symptoms (p value for interaction 0.043).

CONCLUSION—In our NSTE-ACS cohort, ECG markers of hypertrophy were both more common, and more predictive of MI/mortality, among those with depressive symptoms. Cardiac hypertrophy is a potential target for therapy to improve outcomes among depressed NSTE-ACS patients.

Keywords

electrocardiography; hypertrophy; depression; acute coronary syndrome

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INTRODUCTION

Among patients with acute coronary syndrome (ACS), individuals with depressive symptoms are at higher risk for ACS recurrence and cardiac mortality compared with those without depressive symptoms.[1, 2] Several mechanisms have been postulated to explain this excess risk, including reduced adherence to medical treatments and behavioral recommendations among depressed patients,[3, 4] increased inflammation,[5] endothelial dysfunction,[6] abnormal platelet reactivity,[7] and reduced heart rate variability.[8]

Abnormal cardiac sympathetic activation has previously been observed in patients with major depression.[9, 10] A recent animal model has suggested that abnormal cardiac sympathetic activation may play an important role in the poor prognosis associated with depression after myocardial infarction.[11] To test this in humans, electrocardiographic markers may reflect the consequences for cardiac performance of the autonomic dysregulation commonly found in depressed patients,[12, 13] and so may also help to identify those patients with depression who are at particularly high risk for recurrent cardiac events. Electrocardiographic left ventricular hypertrophy (LVH) and strain pattern consisting of repolarization abnormalities in the lateral leads predict cardiac mortality in patients with hypertension,[14] aortic stenosis,[15] and ACS.[16] We assessed the relation between ECG markers of LVH and depression in patients with non ST elevation ACS (NSTE-ACS), expecting that depressed patients may more commonly have repolarization abnormalities. Also, we hypothesized that that depressed patients with these ECG abnormalities may be at particular risk for adverse outcomes after ACS.

METHODS

Patient selection and depression/anxiety symptom evaluation

Institutional Review Board approval for this study was obtained at Columbia University. The cohort was drawn from the Prescription Use, Lifestyle, and Stress Evaluation (PULSE). a prospective cohort study of depression after ACS,[17] Participants were recruited from patients admitted to Columbia University Medical Center between February 2009 and June 2010. ACS events were defined according to American Heart Association/American College of Cardiology criteria[18] as either acute MI or unstable angina. All patients had symptoms consistent with acute myocardial ischemia and at least one of the following: ischemic electrocardiographic changes (i.e. ST depression and/or T-wave abnormalities), an angiogram indicative of coronary artery disease on current admission, and/or documented history of coronary artery disease according to a stress test during the index admission or previous coronary angiogram. Patients who presented with an acute rise in serum troponin I levels >0.4 ng/ml were categorized as MI. A study cardiologist confirmed ACS eligibility for all patients.

A total of 1087 patients were enrolled in PULSE, and for this analysis we drew our sample from the 955 patients who were admitted with NSTE-ACS (87.9% of the total ACS sample). Participants were followed as long as one-year after their initial NSTE-ACS event. Cardiovascular events including myocardial infarction and/or death were identified by

participant report with medical record adjudication, electronic medical record search, and National Death Index search during follow-up.

The Beck Depression Inventory (BDI),[19] a 21-item self-report measure of depressive symptom severity, was administered within one week after the index ACS event, while patients were still hospitalized. The BDI is a well-validated questionnaire with the most extensive evidence of validity for predicting MI recurrence and death compared to all other self-report measures. Based on prior studies in cardiac patients, [20–23] the BDI score was dichotomized, and a score ≥10 was considered to indicate significant depressive symptoms.

Twelve-lead ECGs were acquired during hospitalization for ACS, analyzed by physician/medical student researchers and then over-read by a cardiologist (WW). All ECG interpretations were performed blinded to depression status. For this study we excluded patients with QRS duration ≥120 milliseconds or those whose rhythm was not normal sinus. Left ventricular hypertrophy (CP-LVH) was defined according to Cornell voltage-duration product {[RaVL+SV3+(6 mV in women)]*QRS duration} ≥2440 mV-ms.[24] ECG-strain was defined according to the presence of ≥1 mm ST depression and/or T-wave inversion in any lateral lead (I, aVL, V5, or V6).[16] Measurements were taken using CardioCalipers software (Iconico, Inc., New York, New York).

Statistical Analyses

All analyses were performed with SPSS Version 22 software (IBM Corp., Armonk, NY). Participants with and without depressive symptoms were compared using t-tests for continuous measures and chi-square tests for categorical measures. We estimated logistic regression models for odds ratios of the association of different ECG markers of hypertrophy with depressive symptoms. Three separate regression models were estimated, for CP-LVH, ECG-strain, and the presence of both CP-LVH and ECG-strain together. Full multivariable models included age, sex, black race, body mass index, ACS type, hypertension, diabetes, reduced estimated glomerular filtration rate <60 ml/min/1.73 m^2, left ventricular ejection fraction <0.40, heart rate by ECG, systolic blood pressure on admission, smoking, reported history of antidepressant use prior to admission, and history of beta blocker use.

We used proportional hazards models to estimate the risk of MI/death within one year of the index ACS event associated with different ECG markers. Then, we estimated models separately in patients with and without depressive symptoms. Finally, we estimated model in all patients together, with multiplicative interaction terms between depressive symptoms and each ECG marker to assess for effect modifiers.

RESULTS

Among 955 participants with NSTE-ACS in PULSE, 109 were excluded for QRS duration ≥120 msec, 67 for rhythm other than normal sinus, and 10 for poor quality/missing ECGs, resulting in a sample size of 769 for analysis. Participants with depression were more likely to be female and/or black, to have diabetes mellitus, and to report smoking. Average heart rate was higher among those who reported depressive symptoms. The proportion of

participants who presented with MI or left ventricular ejection fraction <0.40 were not significantly different between those with and without depression.

ECG markers of hypertrophy, including CP-LVH, ECG-strain, and the combination of CP-LVH and ECG-strain were each more frequent in the depressed group (Table 1). In separate age/sex/race- adjusted logistic regression models (Table 2), depressive symptoms were associated with increased odds of CP-LVH (odds ratio = 1.76, 95% CI 1.13–2.76), ECG-strain (OR 1.59, 95% CI 1.13–2.24), and the combination of CP-LVH and ECG-strain (OR 1.85, 95% CI 1.09–3.13). In full multivariable models, the association of depressive symptoms with these ECG markers was stronger (odds ratios 1.74 to 2.33, p values 0.005 to 0.008). The relation between depression and ECG markers did not differ between men and women (p value for interaction 0.898 to 0.943).

Nominal 1-year proportions for MI/death according to depressive symptoms and each ECG marker of hypertrophy are shown in Table 3. The presence of either depressive symptoms or an ECG marker alone was not associated with higher risk. However, when both depression and an ECG marker were present, the risk of MI/death was particularly high. Among patients with depressive symptoms and the combination of CP-LVH and ECG-strain (N=33, 4.3% of the sample), the nominal 1-year risk of MI/death was 24.2% (p for interaction 0.054). Adjusted hazard functions for MI/death also demonstrated evidence for an interaction between depressive symptoms and the combined presence of CP-LVH and ECG-strain (Figure). A proportional hazards model with full multivariable adjustment demonstrated a statistically significant interaction between depressive symptoms and the combination of CP-LVH and ECG-strain (p=0.043). In the subgroup of participants with depressive symptoms, the presence of CP-LVH and ECG-strain together was associated with elevated risk of MI/death in full multivariable models (hazard ratio 4.91, 95% CI 1.55–15.61, p value 0.007), whereas the risk was not elevated in the subgroup without depressive symptoms (hazard ratio 0.591, 95% CI 0.13–2.74, p value 0.502).

DISCUSSION

In this cohort of patients who presented with NSTE-ACS, ECG markers of cardiac hypertrophy including CP-LVH, ECG-strain, and the combination of the two were significantly more frequent among those with depressive symptoms. This relation held even with adjustment for multiple cardiovascular risk factors such as reduced left ventricular ejection fraction, reduced eGFR, and hypertension. The strength of association with depressive symptoms was greatest for the combination of CP-LVH and ECG-strain. Our findings are consistent with a prior cross-sectional study of 2420 subjects without known cardiovascular disease, among whom depressive symptoms measured by BDI score were associated with increased left ventricular mass index and with measures of diastolic dysfunction by echocardiography.[25] Our study extends this work to an ACS sample and with application of validated ECG criteria for detection of hypertrophy.

The ECG markers identified in our analysis suggest that depressive symptoms are associated with changes in cardiac structure that occur over the course of months or years. We cannot rule out the possibility that individuals with more severe cardiac disease develop depressive

symptoms as a consequence. However, it is also possible that depressive symptoms themselves contribute to hypertrophy, and a potential mechanism for the association involves elevated sympathetic activity. An investigation of cardiac norepinephrine spillover using coronary sinus sampling in 39 subjects with major depressive disorder and 76 healthy subjects demonstrated reduced norepinephrine reuptake among those with major depression.[26] Similarly, a recent rodent model of myocardial infarction demonstrated cardiac sympathetic hyperinnervation and left ventricular hypertrophy in rats with induced depression.[11] Our study is consistent with the idea that depression leads to subsequent hypertrophy, potentially due to increased cardiac sympathetic activation.

In this study, 33% of participants were depressed, and 12% showed the CP-LVH with ECG-strain pattern. Neither depression nor CP-LVH with ECG-strain pattern were independently associated with increased risk, but patients with the confluence of depressive symptoms and CP-LVH with ECG-strain pattern were at particularly high risk for death/MI by one year, compared with patients with depressive symptoms alone or ECG markers alone. In the Global Utilization of STrategies to Open occluded arteries (GUSTO IV) study of 7443 NSTE-ACS patients, there was a two-fold difference in mortality at one year among those with ECG-LVH with strain pattern (14.3%) compared with those without (7.1%), and this difference was significantly more pronounced in women than men.[16] In our study, the association of CP-LVH and ECG-strain with depressive symptoms was present regardless of sex. Women are known to report depressive symptoms more frequently than men after ACS,[27] and in our NSTE-ACS cohort, women comprised a greater proportion of patients with depressive symptoms. Thus, it is possible that differences in the burden of depressive symptoms may explain part of the sex interaction noted in GUSTO IV.

The risk of cardiac mortality associated with depressive symptoms in ACS is widely recognized, but trials of therapies to modify this risk have generally reported null findings [28] or have not been powered for cardiac mortality outcomes.[29–31] Our findings suggest that 12-lead ECGs may offer important markers of cardiac risk among ACS patients with depressive symptoms to guide future trials of therapy.

The ECG markers in our study may also serve as a potential target of therapy in depressed patients with cardiac disease. In the Losartan Intervention For Endpoint Reduction in Hypertension (LIFE) study, reduction in LVH according to CP-LVH and ECG-strain was associated with lower risk of cardiovascular mortality and sudden cardiac death.[32] Treatment that is focused on interruption of cardiac disease progression may offer more benefits, certainly in terms of cardiac events, than treatment of depressive symptoms per se.

Limitations of our study include the small number of endpoints, such that our findings may be the result of chance. However, the interaction between depressive symptoms and CP-LVH and ECG-strain was preserved with adjustment for numerous other predictive variables including hypertension, diabetes, and reduced left ventricular ejection fraction. Another limitation of our analysis is the single time point for ECG capture and analysis, such that we cannot assess for the impact of changes in depressive symptoms on ECG markers. Also, we did not collect magnetic resonance imaging or echocardiographic information regarding ventricular hypertrophy, such that we cannot correlate our ECG findings with other imaging

modalities. ECG measures of hypertrophy have generally been found to be specific, but not sensitive for ventricular hypertrophy based on these other methods.[33] In addition, we used a marker for depressive symptoms based on a self-report inventory, rather than an interview-based diagnosis for depression.

In conclusion, in our NSTE-ACS cohort, ECG markers of hypertrophy were both more common, and more predictive of MI/mortality, among those with depressive symptoms. Cardiac hypertrophy is a potential target for therapy to improve outcomes among depressed NSTE-ACS patients.

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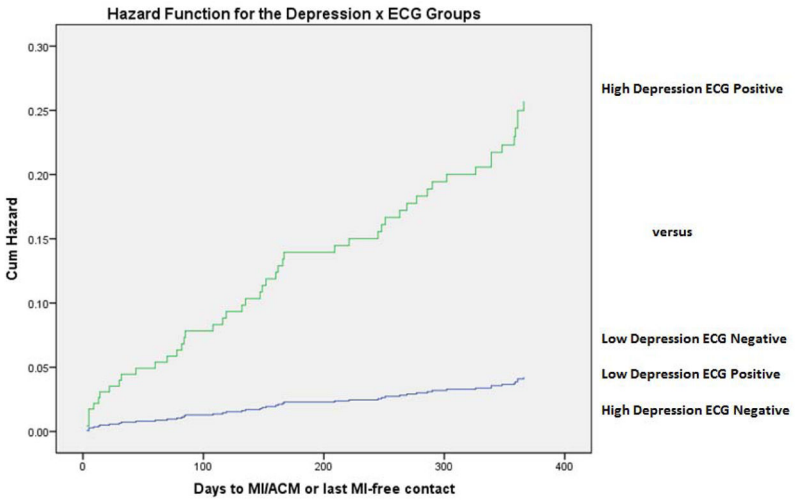

Figure.

Hazard functions for time to myocardial infarction or all-cause mortality, comparing subjects both with Beck Depression Inventory score ≥10 and left ventricular hypertrophy with strain (high depression, ECG positive) versus the average of the other 3 groups. Hazard functions are adjusted for age, sex, body mass index, ACS type, hypertension, diabetes mellitus, left ventricular ejection fraction <0.40, estimated glomerular filtration rate<60 ml/min/1.73 m^2, admission systolic blood pressure, ECG heart rate, smoking, antidepressant use, and beta blocker use.

Table 1

Characteristics of categories of participants based on cutoff values for Beck Depression Inventory (BDI) score

	BDI <10 (n=516)	BDI ≥10 (n=253)	P value
Age, years	63.3 (11.1)	61.7 (11.2)	0.07
Female, %	28.1	48.6	<0.01
Black %	17.0	27.6	<0.01
BMI >25	78.1	79.8	0.64
Hypertension, %	78.9	83.8	0.12
LVEF<0.40, %	7.2	9.1	0.39
ECG Heart rate, BPM	66.7 (11.4)	68.8 (12.2)	0.02
Diabetes mellitus, %	31.6	41.9	0.02
eGFR <60 ml/min/1.73 m^2	24.7	25.4	0.86
Smoking, %	48.4	56.5	0.04
Beta blocker, %	52.3	58.1	0.14
Antidepressant, %	11.0	31.2	<0.01
Non-STEMI, %	35.3	34.0	0.75
BDI score	4.2 (2.7)	15.8 (6.0)	<0.01
CP-LVH, %	9.9	19.0	<0.01
ECG-strain, %	23.8	36.0	<0.01
CP-LVH & ECG-strain, %	6.4	12.6	<0.01

Values are mean (standard deviation) or percentages. P values correspond to chi-square tests for categorical variables, analysis of variance for continuous variables.

BMI = body mass index; LVEF = left ventricular ejection fraction; BPM = beats per minute; BDI= Beck Depression Inventory; ECG=electrocardiogram; CP-LVH = left ventricular hypertrophy by Cornell product

Table 2

Odds ratios for different ECG indicators of hypertrophy associated with depressive symptoms (BDI ≥10).

	Age/sex/race adjusted	P value	Multivariable	P value
Model 1: CP-LVH				
BDI ≥10	1.76 (1.13, 2.76)	0.013	1.95 (1.19, 3.18)	0.008
Model 2: ECG-strain				
BDI ≥10	1.59 (1.13, 2.24)	0.008	1.74 (1.18, 2.56)	0.005
Model 3: CP-LVH & ECG-strain				
BDI ≥10	1.85 (1.09, 3.13)	0.023	2.33 (1.28, 4.25)	0.006

*All multivariable models also adjust for body mass index, ACS type, hypertension, diabetes mellitus, left ventricular ejection fraction <0.40, estimated glomerular filtration rate<60 ml/min/1.73 m^2, admission systolic blood pressure, ECG heart rate, smoking, antidepressant use, and beta blocker use.

Table 3

One-year nominal risk for MI/ACM according to depressive symptom category and absence or presence of ECG indicator of hypertrophy.

	BDI <10	BDI ≥10	P value for interaction
CP-LVH			
absent	27/465 (5.8%)	11/205 (5.4%)	
present	3/51 (5.9%)	8/48 (16.7%)	0.119
ECG-strain			
absent	20/393 (5.1%)	7/162 (4.3%)	
present	10/123 (8.1%)	12/91 (13.2%)	0.264
CP-LVH & ECG-strain			
absent	28/483 (5.8%)	11/220 (5.0%)	
present	2/33 (6.1%)	8/33 (24.2%)	0.054