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Patient-reported symptoms improve prediction of acute coronary syndrome in the emergency department

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Abstract

Early diagnosis is critical in the management of patients with acute coronary syndrome (ACS), particularly ST-elevation myocardial infarction (STEMI), because effective therapies are time-dependent. Aims of this secondary analysis were to determine: (i) the prognostic value of symptoms for an ACS diagnosis in conjunction with electrocardiographic (ECG) and troponin results; and (ii) if any of 13 symptoms were associated with prehospital delay in those presenting to the emergency department (ED) with potential ACS. Patients receiving a cardiac evaluation in the ED were eligible for the study. Thirteen patient-reported symptoms were assessed in triage. Prehospital delay time was calculated as the time from symptom onset until registration in the ED. A total of 1,064 patients were enrolled in five EDs. The sample was 62% male, 70% white, and had a mean age of 60.2 years. Of 474 participants diagnosed with ACS, 118 (25%) had STEMI; 251 (53%) had non-ST elevation myocardial infarction (NSTEMI); and 105 (22%) had unstable angina. Sweating (OR = 1.42 CI [1.01, 2.00]) and shoulder pain (OR = 1.64 CI [1.13, 2.38]) added to the predictive value of an ACS diagnosis when combined with ECG and troponin results. Shortness of breath (OR = 0.71 CI [0.50, 1.00]) and unusual fatigue (OR = 0.60 CI [0.42, 0.84]) were predictive of a non-ACS diagnosis. Sweating predicted shorter prehospital delay (HR = 1.35, CI [1.10, 1.67]); shortness of breath (HR = 0.73 CI [0.60, 0.89]) and unusual fatigue (HR = 0.72, CI [0.57, 0.90]) were associated with longer prehospital delay. Patient-reported symptoms are significantly associated with ACS diagnoses and prehospital delay. Sweating and shoulder pain combined with ECG signs of ischemia may improve the timely detection of ACS in the ED.

Keywords

acute coronary syndrome; electrocardiography; emergency nursing; prehospital delay; symptoms

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CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

1 | INTRODUCTION

Patients presenting to the emergency department (ED) with acute coronary syndrome (ACS) are at risk for fatal arrhythmias, onset of heart failure, and death (Amsterdam, Wenger, Brindis, Ganiats, & Zieman, 2014; Cannon, Brindis, Chaitman, Cohen, & Weintraub, 2013; O’Gara et al., 2013). Of 1.2 million patients suffering from ACS or cardiac death each year, more than half die either before reaching the hospital or in the ED (Benjamin et al., 2017).

ACS is the acute manifestation of coronary heart disease and includes the diagnosis of ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), and unstable angina (UA). The two primary approaches for optimizing ACS patient outcomes are to reduce treatment delay and to minimize total ischemic time, defined as time of symptom onset to reperfusion of culprit arteries (Hicks et al., 2015; O’Gara et al., 2013). ACS symptoms trigger patient care seeking behaviors and inform providers’ choice of diagnostic testing; yet symptoms are often ambiguous (Rosenfeld et al., 2015). If symptoms are neither identified nor recognized, patients may be at risk for increased prehospital delay, delayed diagnosis, and increased mortality and morbidity (Doggen et al., 2016; Fanaroff, Rymer, Goldstein, Simel, & Newby, 2015; Menees et al., 2013; O’Gara et al., 2013; Wu, Moser, Riegel, McKinley, & Doering, 2011).

Accurate risk stratification and diagnostic testing are critical for time dependent therapies that restore blood flow to the compromised myocardium, thereby reducing morbidity and mortality and avoiding inappropriate hospital discharge (Hess et al., 2015; Pelter, 2010). To minimize total ischemic burden time, the American Heart Association and European Society of Cardiology (AHA/ESC) recommend that individuals with chest pain seek medical attention immediately and receive a 12-lead electrocardiogram (ECG) within 10 min of hospital arrival, based on evidence that longer delays are associated with adverse prognoses (O’Gara et al., 2013). ECG results should be analyzed in conjunction with patient history and clinical presentation to assess the likelihood of an ACS diagnosis (O’Gara et al., 2013; Savonitto et al., 1999).

ACS remains a diagnostic challenge because no single risk stratification model (e.g., Thrombolysis in Myocardial Infarction [TIMI] risk score; history, ECG, age, risk factors, Troponin [HEART] score) or diagnostic strategy has been shown to identify all ACS cases accurately and there is no clear reference standard (Cullen et al., 2013; Fanaroff et al., 2015; Worrall-Carter et al., 2015). Findings from prior studies suggest that patients presenting with chest symptoms (chest pain, chest pressure, or chest discomfort) have a high likelihood of an ACS diagnosis and certain clinical features (e.g., pulmonary basal crackles, hypotension) can predict ACS, but neither alone can confirm an ACS diagnosis (Body et al., 2010; DeVon, Rosenfeld, Steffen, & Daya, 2014). Little is known about the association between non-chest symptoms reported by patients in the ED, while they are occurring, with ECG and other clinical characteristics. The purpose of this study was to determine (i) the prognostic value of symptoms for an ACS diagnosis in conjunction with electrocardiographic (ECG) and troponin results; and (ii) if any of 13 symptoms were associated with prehospital delay in those presenting to the ED with potential ACS. These aims are important to better

characterize the early presentation of ACS for improved triage, risk stratification, diagnostic testing, and treatment.

2 | METHODS

This is a secondary analysis of data from the *Think Symptoms* study, a large prospective multicenter study, examining the influence of sex on symptom characteristics for suspected ACS (DeVon et al., 2017). Patients in the parent study were enrolled at five large medical centers across the United States (Midwest, Pacific Northwest, Southwest, and West regions). Clinically expert, registered nurse research associates were trained about the study procedures by the principal investigators. Research associates were present in each study ED where they worked 4-hr shifts, on a rotating schedule, 7 days a week. The research associates were present in each ED for approximately 20 hr per week. The project director was responsible for assisting research nurses in ethics training and management of records and data. Fidelity to the study procedures were assured in several ways: (i) The principal investigator (PI), site PIs, and the project director trained the research staff in all study protocols in two 2-hr didactic sessions; (ii) The project director was responsible for assisting research staff in collection, management, and verification of data; (iii) The research staff had printed detailed protocol manuals that contained all policies, procedures, and instruments for the study; (iv) The research staff were asked to role play data collection scenarios; (v) A random selection of 5% of the data were evaluated for completeness and accuracy by the project director. The project director reviewed the following procedures with the research nurses: recruitment, informed consent, administration of instruments, evaluation of instruments for completeness, conduct of phone call follow-ups, and maintenance of study brochures. The research nurses were then asked to role play each of these activities. Phone conferences or face-to-face meetings were conducted monthly with the PI, co-investigator, project director, and research nurses.

The institutional review boards (IRB) at the sponsoring institution and each site approved the study. An initial waiver of consent was granted by each IRB for the research associate to complete a symptom checklist within 15 min of the patient's presentation to the ED. The symptom data were destroyed if the patient declined to participate. Written consent was obtained when patients were admitted to an examination room and their condition was stable.

Patients >21 years of age, fluent in English, and with symptoms that triggered an evaluation for ACS were eligible for the study. Patients were enrolled regardless of their mode of arrival to the ED (walk in, private vehicle, or ambulance). Patients most likely to be ruled in for ACS were identified before enrollment based on standard ECG and troponin criteria (Thygesen et al., 2012). The targeted sampling plan included patients with any ECG changes suggestive of ischemia and/or with a troponin level outside the referenced norm for the institution. Ischemia was defined as new ST-elevation at the J point ≥ 0.1 mV in two contiguous leads and/or new horizontal or down-sloping ST-depression ≥ 0.05 mV in two contiguous leads (Thygesen et al., 2012).

2.1 | Measures

2.1.1 | Symptoms—The ACS Symptom Checklist is a 13-item validated instrument that measures symptoms dichotomously (yes/no).

The ACS Checklist has demonstrated reliability (Cronbach's $\alpha = .81$; DeVon, Hogan, Ochs, & Shapiro, 2010) and validity (content validity indexes of .88 and .94) in previous studies of ACS populations (DeVon & Zerwic, 2003; DeVon, Ryan, Ochs, & Shapiro, 2008). Symptom distress was measured separately on a numerical rating scale from 0 to 10 as an evaluation of the severity of a patient's entire symptom experience.

2.1.2 | ECG and prehospital delay time—Patients received an initial ECG on arrival to the ED, according to usual care at each institution, and ED physicians and/or cardiologists analyzed the ECGs. ECG results, past medical history, diagnosis, and treatments were abstracted from the medical record by the trained research associates. Prehospital delay time was determined by subtracting self-reported symptom onset time from ED registration time. Sample demographic data and clinical characteristics were collected via self-report.

2.2 | Statistical analyses

Data were analyzed with SAS (Version 9.4, SAS®, Cary, NC) and MPLUS (Version 7.4, Los Angeles, CA: Muthén & Muthén) software. A significance level of $p < .05$ was considered statistically significant for all models. Bivariate relationships between the demographic and symptom variables and the primary outcomes (diagnosis and prehospital delay time) were assessed. The amount of missing data were calculated for all variables. For key variables with more than 5% missing data, the demographic and clinical characteristics associated with missingness were determined. ECG signs of ischemia (ST-elevation, ST-depression, and/or T-wave inversion) and prehospital delay time had missing data that exceeded the above criterion, with 14.1% and 7.8%, respectively. Because ECG signs of ischemia and prehospital delay were primary variables of interest and exceeded the missing data threshold, Full Information Maximum Likelihood (FIML) regression was used for adjusted models. FIML allows all cases to be included despite missing data in predictor or outcome variables. FIML can be used when data are missing at random (versus missing completely at random), which assumes that the missingness of a variable does not depend on the value of the variable (e.g., patients who had longer prehospital delays were less likely to report delay time, or patients with a STEMI diagnosis were less likely to have their diagnosis documented compared to a patient with a NSTEMI diagnosis, are examples of data *not* missing at random). FIML requires that covariates predicting missingness be included in the model (Allison, 2001).

Unadjusted logistic regression was used to assess bivariate differences in symptoms, ECG, troponin levels, and prehospital delay time by the type of ACS diagnosis. For adjusted models, age, sex, and race were included as covariates due to their established relationship with the outcomes. Study site was also included as a covariate in adjusted models because the amount of missing data varied by site. Specifically, data were examined and the study site with the most missing data (ECG results or prehospital delay times) was adjusted for. The primary study site had the most missing data for ACS diagnosis and a secondary site

had the most missing data for prehospital delay time. Logistic regression FIML models were used to predict an ACS (UA, NSTEMI, or STEMI) versus non-ACS diagnosis, a UA versus non-ACS diagnosis, and a NSTEMI versus non-ACS diagnosis. A non-ACS diagnosis was defined as any diagnosis other than ACS. A STEMI versus non-ACS model could not be evaluated because ST-elevation is part of the criteria for a STEMI diagnosis. ST-elevation, therefore, is a constant that cannot add to the predictive value of a STEMI diagnosis (Thygesen et al., 2012). Regression cannot be conducted when there is no variability in a predictor (i.e., ECG signs of ischemia were present in all STEMI cases).

Proportional hazards FIML regression models were used to predict prehospital delay time from demographic and symptom variables (Asparouhov, 2014). We evaluated the assumptions of the proportional hazards model by graphing Schoenfeld residuals by time and by testing a non-zero slope of residuals by time for each predictor and the global model (Hosmer, Lemeshow, & May, 2011). Prehospital delay time was measured in hours and results are presented as hazard ratios (HR). The HR represents the increase/decrease in probability of arriving at the ED for participants with a given characteristic (e.g., female sex) compared to a reference group (e.g., male sex) for each 1 hr increase in prehospital delay time. The symptom distress scale (range 0–10) was dichotomized as eight or more (more distress) and seven or less (less distress) because eight was the median score for the sample. For the continuous predictor of age, the HR represents the increase/decrease in probability of arriving at the ED, given a 1-hr increase in time, for every additional 10 years in age (e.g., 70 vs. 60 years old or 50 vs. 40 years old). A hazard ratio greater than 1 equates to a greater likelihood of arriving at the ED, or shorter prehospital delay. A hazard ratio less than 1 equates to a decreased likelihood of arriving at the ED or longer prehospital delay.

3 | RESULTS

A total of 1,064 patients were included in the parent study. They had a mean age of 60.2 years, 62.4% were male, and 69.5% were White (Table 1). Of the 1,064 participants, 474 (44.5%) received a final diagnosis of ACS and 590 received a non-ACS diagnosis as determined by the admitting physician. Among patients diagnosed with ACS, 118 (24.9%) had STEMI; 251 (52.9%) had NSTEMI; and 105 (22.1%) had UA.

3.1 | Patient-reported symptoms, ECG signs of ischemia, and troponin by diagnosis

Patients with STEMI had significantly higher rates of sweating compared to non-ACS patients. Among individual ACS diagnoses, STEMI patients reported significantly higher rates of chest pain, sweating, and symptom distress scores of eight or higher compared to other diagnoses (Table 2). Chest pain, sweating, and higher symptom distress scores were more common, and unusual fatigue and palpitations were less common in patients with STEMI compared with other diagnoses. Shortness of breath and lightheadedness were more common and arm pain was less common in patients with non-ACS and UA diagnoses. Fewer non-ACS patients had ECG signs of ischemia, though ECG changes were observed in 29% of cases. Consistent with the accepted definition of acute myocardial infarction (Alpert, Thygesen, Antman, & Bassand, 2000; White, Thygesen, Alpert, & Jaffe, 2014), elevated troponin levels were most frequently noted in patients with NSTEMI followed by those with

STEMI. Elevated troponin levels were much less frequent in patients diagnosed with UA and non-ACS diagnoses.

ECG signs of ischemia and elevated troponins were the strongest predictors of an ACS diagnosis, followed by shoulder pain, sweating, greater symptom distress (8), and older age (Table 3). Shortness of breath, unusual fatigue, and female sex were predictive of a non-ACS diagnosis. In adjusted models predicting type of ACS diagnosis, ECG signs of ischemia, older age, and chest pressure predicted a diagnosis of UA, while female sex did not. NSTEMI was most strongly predicted by elevated troponin levels, followed by ECG signs of ischemia, shoulder pain, arm pain, older age, and male sex. Unusual fatigue, lightheadedness, and shortness of breath decreased the likelihood of a NSTEMI diagnosis.

3.2 | Prehospital delay

In ACS patients, sweating was the only symptom that predicted a shorter prehospital delay (Table 4) (OR = 1.35, CI [1.10–1.67]). A symptom distress score of eight or higher also predicted a shorter prehospital delay time. By contrast, shortness of breath and unusual fatigue were significant predictors of longer prehospital delay. In non-ACS patients, older age, chest pain, and nausea predicted shorter prehospital delay. Unusual fatigue and upper back pain predicted longer prehospital delay in patients with a non-ACS diagnosis.

Table 5 presents predictors of prehospital delay time by ACS type. In NSTEMI patients, lightheadedness and nausea predicted shorter prehospital delay. By contrast, shortness of breath and unusual fatigue predicted longer prehospital delay. Older age and white race predicted shorter prehospital delay for STEMI patients. No variables predicted prehospital delay in UA patients.

4 | DISCUSSION

To our knowledge, this is one of few prospective studies to evaluate comprehensive contextual variables (patient-reported symptoms, ECG, and troponin) and their association with ACS diagnoses and prehospital delay. In a large, contemporary population of patients presenting to the ED across multiple regions, we found specific symptoms (shoulder pain and sweating) and a higher symptom distress score (8) added to the predictive ability of ECG findings and troponin values for a diagnosis of ACS. Symptoms varied by type of ACS. STEMI patients reported higher rates of chest pain and an overall symptom distress score of eight or higher. NSTEMI patients had more nausea, arm pain, and shoulder pain. Patients with UA had higher rates of chest pressure. Prehospital delay times varied significantly by symptoms in our cohort. Sweating (or diaphoresis) and greater symptom distress predicted shorter prehospital delay times while shortness of breath and unusual fatigue predicted longer prehospital delay times.

A key finding was that sweating was more frequent for patients with STEMI compared to other ACS and non-ACS diagnoses. Sweating is associated with STEMI due to sympathetic nervous system stimulation. Sweating also predicted an overall ACS diagnosis and shorter prehospital delay time for patients diagnosed with ACS. Our results are consistent with previous investigations that reported sweating as highly predictive of acute myocardial

infarction (AMI; Body et al., 2010; Bruyninckx, Aertgeerts, Bruyninckx, & Buntinx, 2008). Body et al. (2010) examined patients presenting to the ED with chest pain and found those in whom ED physicians observed sweating were over five times as likely to be diagnosed with AMI and have adverse outcomes (e.g., death) compared to those without sweating. In a meta-analysis to ascertain the accuracy of signs and symptoms of ACS, Bruyninckx et al. (2008) determined sweating had the highest pooled positive likelihood ratio for AMI.

It is important to consider differences between our study and these prior studies. UA patients were not included in the Body et al. (2010) study sample. In addition, the treating ED physician collected symptom data in the Body study; this differed from our study where trained research associates collected symptom data with a validated instrument, while symptoms were occurring. Because there are no standardized clinical assessment tools for symptom assessment in the ED setting, results may vary by how clinicians assess symptoms. Studies included in the meta-analysis completed by Bruyninckx et al. (2008), involved patients who were in the ED and admitted to the hospital (e.g., chest observation units, coronary care units). There are inherent differences between admitted patients and ED patients. For example, admitted patients are likely to receive interventions (e.g., oxygen, morphine) that alleviate or alter symptoms and potentially influence their symptom recall. Furthermore, in studies used in the meta-analysis, symptom data were collected retrospectively from medical records (Bruyninckx et al., 2008); findings may be altered by the variations and omissions inherent with medical records and clinician documentation. Despite methodological differences between studies, sweating remained significantly associated with ACS and specifically, STEMI.

Contrary to our findings, results from a prior study showed that sweating was not significantly associated with an ACS diagnosis in patients returning to the ED after hospitalization for ACS (Pelter et al., 2012). Data for the study reporting these different findings were collected by research assistants who assessed symptoms using a scripted interview during follow-up telephone calls (Pelter et al., 2012). This difference could be related to the timing of symptom data collection after discharge, when patients may not be able to accurately recall all of their symptoms. Overall, our findings reinforce that sweating remains an important symptom in the context of STEMI.

Consistent with prior investigations, we found shoulder pain was predictive of overall ACS (Body et al., 2010; Pelter et al., 2012). NSTEMI patients reported significantly more shoulder and arm pain compared to other ACS diagnoses. Body et al. (2010) found that radiation of pain to the shoulder or right arm was a stronger predictor of myocardial infarction compared to pain in the left shoulder or arm. Pelter et al. (2012) found ED patients with arm pain were nearly twice as likely to have ACS, even though less than one-quarter experienced arm pain (Pelter et al., 2012). Findings suggest that patients need to be asked about shoulder and arm pain because of their diagnostic significance for ACS.

Recognition of ACS symptoms in the ED is important for accurate triage, especially in the absence of ECG signs of ischemia (Arora & Bittner, 2015). Our findings reinforce the importance of non-chest pain symptoms in ACS patients, because up to 30% of patients do not experience chest pain, leaving them vulnerable to a missed or delayed diagnosis and

delayed reperfusion therapy that contributes to adverse health outcomes (El-Menyar et al., 2011). The symptom differences by ACS diagnoses observed in our study may be attributed to variations among patients in collateral circulation, which partially mitigates the consequences of an abrupt coronary artery occlusion in the setting of STEMI (Karam et al., 2016). Patients with ACS may experience sporadic myocardial ischemia that results in both symptomatic and asymptomatic periods or intermittent ECG ST-segment changes (Pelter, 2010). New initiatives to evaluate non-chest pain symptoms (e.g., sweating, shoulder pain, and arm pain) in risk prediction models are needed.

Prehospital delay times varied significantly by symptoms in our cohort. Sweating and more symptom distress had shorter prehospital delay times whereas shortness of breath and unusual fatigue predicted longer prehospital delay times. Although we found shortness of breath and unusual fatigue predictive of non-ACS diagnoses, the associated longer prehospital delay suggests that adequate familiarity with the variety of ACS symptoms among adults remains low. Patients may not identify shortness of breath or fatigue as potentially life threatening or as being associated with ACS.

Our findings may be interpreted in the context of slow-onset and fast-onset symptoms of ACS and patient decision making behaviors (O'Donnell, McKee, Mooney, O'Brien, & Moser, 2014). O'Donnell and Moser (2014) examined patients presenting to the ED with ACS symptoms and found those with slow-onset presentations (e.g., fatigue) had significantly longer prehospital delay compared to patients with fast-onset presentations (e.g., chest pain). Patients with slow-onset presentations may have difficulty ascribing their symptoms to a potential cardiac cause due to expectations of what an ACS event should be (O'Donnell et al., 2014).

We also found prehospital delay was significantly lower in patients with STEMI, but did not differ between NSTEMI, UA, and non-ACS diagnoses. This is consistent with prior studies (DeVon, Ryan, Rankin, & Cooper, 2010; O'Donnell et al., 2014) and remains important because reducing the time from symptom onset to reperfusion therapy in STEMI patients decreases infarct size and improves survival (DeVon, Ryan, et al., 2010; Menees et al., 2013).

Prehospital delay remains a major barrier to optimizing patient outcomes despite initiatives to improve patients' ability to recognize early signs or symptoms of ACS. Although patient characteristics (e.g., older age, female sex, black race, low education level, and low socioeconomic status) have been well examined (Bugiardini et al., 2017; Ouellet et al., 2017), our study focuses on the impact of early symptoms and prehospital delay. This work is timely because of the recent emphasis on patient recognition of early symptoms for reducing prehospital delay to minimize total ischemic burden time and improve patient outcomes (Menees et al., 2013).

4.1 | Strengths and limitations

There have been numerous retrospective and population-based studies on the symptoms of ACS as well as predictors of prehospital delay (McKee et al., 2013; Moser et al., 2007; Ting & Bradley, 2009). We designed our study to address limitations of prior research, including

retrospective designs, focus on AMI patients only, data abstracted from the electronic health record, data collected from patients admitted with chest pain only, and oversampling of males. Our prospective clinical study using data collected by trained research associates, directly from the patient, during triage, using a validated instrument resulted in stronger internal validity and reduced potential recall bias by patients. We collected data across multiple study sites in different regions, which enhances the generalizability of our findings.

This study has limitations. The ACS Symptom Checklist only permitted a yes/no classification of symptoms. We are therefore unable to determine the strength or intensity of a specific symptom for an ACS diagnosis. We could not determine the interrater reliability of multiple ED clinicians who analyzed ECGs or if the most recent American Heart Association/American College of Cardiology criteria, which account for sex and age differences, were used consistently (Thygesen et al., 2012). Lastly, patients self-reported time of symptom onset. Prehospital delay time, therefore, may not always have been accurate due to recall bias.

5 | CONCLUSION

Differentiating patients with potential ACS in the ED remains challenging yet accurate detection of ACS, particularly STEMI, is essential to early treatment that directly improves patient outcomes. Findings from this study reinforce the importance of considering patient-reported symptoms in conjunction with ECG signs of ischemia and troponin results because ECG signs of ischemia and troponin levels alone are not sufficient to determine an accurate ACS diagnosis. Sweating combined with ECG signs of ischemia may improve the timely detection of ACS in the ED and is associated with shorter prehospital delay times. Future iterations of risk stratification models, such as TIMI which estimates mortality for patients with unstable angina and non-ST elevation MI or HEART score, which predicts 6-week risk of major adverse cardiac event, may benefit from inclusion of non-chest pain symptoms like sweating.

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TABLE 1

Sample demographic data and clinical characteristics

Age	
Mean (SD)	60.2 (14.0)
Unknown	0
Sex	
Male	664 (62.4)
Female	400 (37.6)
Unknown	0
Race/ethnicity	
White	739 (69.5)
Black	135 (12.7)
Hispanic	81 (7.6)
Other	104 (9.8)
Unknown	5 (0.5)
Insurance	
Private (employer)	332 (31.2)
Private (self)	106 (10.0)
Medicare	348 (32.7)
Government (other)	121 (11.4)
Not insured	139 (13.1)
Unknown	18 (16.9)
Education	
<High school diploma	125 (11.7)
High school diploma	235 (22.1)
Some college	344 (32.3)
College	216 (20.3)
Graduate degree	141 (13.3)
Unknown	3 (0.3)
Income	
Less than \$20,000	316 (29.7)
\$20,000–49,999	317 (29.8)
\$50,000–99,999	189 (17.8)
\$100,000 or more	137 (12.9)
Unknown	105 (9.9)
Hypertension	
Yes	687 (64.6)
No	369 (34.7)
Unknown	8 (7.5)
Diabetes	

Yes	307 (28.9)
No	754 (70.9)
Unknown	3 (2.8)
Hypercholesterolemia	
Yes	568 (53.4)
No	467 (43.9)
Unknown	29 (2.7)
Tobacco use	
Never smoked	567 (53.3)
Smoked previously	256 (24.1)
Current smoker	216 (20.3)
Unknown	25 (2.3)
Body mass index	
Mean (SD)	30.0 (7.2)
Unknown	19 (1.8)
Troponin level	
.03 ng/ml	484 (45.5)
>.03 ng/ml	572 (53.76)
Unknown	8 (7.5)
ECG signs of ischemia ^a	
Yes	436 (41.0)
No	478 (44.9)
Unknown	150 (14.1)

^aIncludes ST elevation, ST depression or T-wave inversion. Values are *n* (%) unless otherwise noted.

TABLE 2

Unadjusted differences in symptoms, diagnostic test results, and delay time by ACS diagnosis

Characteristic	a. Non-ACS (n = 590)		b. UA (n = 105)		c. NSTEMI (n = 251)		d. STEMI (n = 118)	
	n	%	n	%	n	%	n	%
Chest discomfort	413	70.00	78	74.29	170	67.73	80	68.38
Chest pain	389	65.93 ^d	73	69.52	173	68.92 ^d	94	80.34 ^{a,c}
Chest pressure	372	63.05 ^b	78	74.29 ^{a,c}	157	62.55 ^b	75	64.10
Short of breath	364	61.7 ^{c,d}	62	59.0 ^c	114	45.4 ^{a,b}	57	48.7 ^a
Unusual fatigue	316	53.56 ^{b,c,d}	45	42.86 ^{a,d}	111	44.22 ^{a,d}	32	27.35 ^{a,b,c}
Lightheadedness	294	49.83 ^{c,d}	52	49.52 ^{c,d}	89	35.46 ^{a,b}	36	30.77 ^{a,b}
Nausea	219	37.12	33	31.43 ^{c,d}	97	38.65 ^b	41	35.04 ^b
Arm pain	190	32.20 ^c	30	28.57 ^c	111	44.22 ^{a,b}	48	41.03
Sweating	198	33.56 ^d	31	29.52 ^d	85	33.86 ^d	58	49.57 ^{a,b,c}
Shoulder pain	202	34.24	36	34.29	102	40.64 ^d	31	26.50 ^c
Upper back pain	181	30.68 ^d	29	27.62	61	24.30	22	18.80 ^a
Palpitations	180	30.51 ^{c,d}	31	29.52 ^d	52	20.72 ^a	15	12.82 ^{a,b}
Indigestion	140	23.73	22	20.95	68	27.09	24	20.51
Symptom distress score	8 273	46.7 ^{c,d}	51	49.5 ^d	140	56.2 ^{a,d}	86	74.1 ^{a,b,c}
ECG signs of ischemia	144	28.80 ^{b,c,d}	47	54.65 ^{a,d}	127	60.48 ^{a,d}	118	100.0 ^{a,b,c}
Elevated troponin	167	28.55 ^{c,d}	26	25.00 ^{c,d}	211	84.74 ^{a,b,d}	80	67.80 ^{a,b,c}
Delay time > 3 hr	345	62.50 ^d	61	64.89 ^d	135	59.21 ^d	37	34.58 ^{a,b,c}

ACS, acute coronary syndrome; Non-ACS, all other diagnoses; NSTEMI, non-ST elevation myocardial infarction; STEMI, ST-elevation myocardial infarction; UA, unstable angina. Superscript letters represent statistically significant differences between diagnostic groups at $p < .05$.

TABLE 3

Prediction of ACS diagnoses from demographic traits, troponin levels, ECG sign of ischemia, and symptom characteristics

Characteristic	ACS vs. Non-ACS (N = 1,064)				UA vs. Non-ACS (n = 695)				NSTEMI vs. Non-ACS (n = 841)			
	OR	LL	UL	p	OR	LL	UL	p	OR	LL	UL	p
10 year increase in age	1.23	1.10	1.38	<.001	1.17	1.00	1.38	.048	1.24	1.08	1.43	.002
White race	1.30	0.92	1.84	.129	1.12	0.68	1.85	.644	1.33	0.86	2.06	.197
Female gender	0.48	0.34	0.66	<.001	0.40	0.25	0.64	<.001	0.61	0.40	0.93	.021
Study site	3.56	2.43	5.22	<.001	3.97	2.38	6.61	<.001	3.18	2.02	5.00	<.001
Elevated troponin	4.29	3.11	5.91	<.001	0.74	0.42	1.30	.292	12.91	8.26	20.17	<.001
ECG signs of ischemia	4.41	3.14	6.18	<.001	3.09	1.82	5.23	<.001	2.65	1.71	4.10	<.001
Distress score 8	1.53	1.11	2.11	.009	1.14	0.71	1.83	.581	1.32	0.88	1.98	.186
Chest discomfort	1.14	0.75	1.73	.532	1.14	0.61	2.14	.67	1.13	0.67	1.90	.649
Chest pain	1.29	0.87	1.91	.198	1.03	0.58	1.81	.927	1.24	0.76	2.04	.386
Chest pressure	1.39	0.97	2.00	.074	1.86	1.05	3.29	.032	1.16	0.73	1.83	.527
Short of breath	0.71	0.50	1.00	.049	0.88	0.53	1.47	.621	0.60	0.39	0.92	.020
Unusual fatigue	0.60	0.42	0.84	.003	0.61	0.36	1.03	.065	0.63	0.40	0.99	.045
Lightheadedness	0.79	0.55	1.11	.173	1.37	0.80	2.34	.252	0.61	0.39	0.96	.032
Nausea	1.14	0.80	1.64	.474	0.85	0.48	1.49	.565	1.30	0.82	2.06	.257
Arm pain	1.39	0.97	1.98	.072	0.80	0.45	1.41	.437	1.84	1.17	2.91	.009
Sweating	1.42	1.01	2.00	.046	0.88	0.51	1.51	.645	1.48	0.94	2.32	.091
Shoulder pain	1.64	1.13	2.38	.009	1.43	0.84	2.43	.192	2.23	1.37	3.65	.001
Upper back pain	0.82	0.57	1.17	.273	0.88	0.53	1.45	.611	0.73	0.46	1.17	.192

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Characteristic	ACS vs. Non-ACS (N = 1,064)				UA vs. Non-ACS (n = 695)				NSTEMI vs. Non-ACS (n = 841)			
	95% CI				95% CI				95% CI			
	OR	LL	UL	p	OR	LL	UL	p	OR	LL	UL	p
Palpitations	0.96	0.66	1.40	.839	1.46	0.83	2.57	.188	0.89	0.55	1.44	.633
Indigestion	1.10	0.75	1.60	.626	0.93	0.51	1.70	.807	1.13	0.71	1.78	.612

ACS, acute coronary syndrome; ECG, electrocardiographic; NSTEMI, non-ST elevation myocardial infarction; LL, lower limit; OR, odds ratio; UA, unstable angina; UL, upper limit.

TABLE 4

Demographic and symptom predictors of time from symptom onset to ED presentation for patients with and without ACS

	ACS (<i>n</i> = 474)				Non-ACS (<i>n</i> = 590)			
	95% CI				95% CI			
	HR	LL	UL	<i>p</i>	HR	LL	UL	<i>p</i>
10 year increase in age	1.06	0.98	1.14	.157	1.09	1.03	1.15	.002
White race	1.14	0.92	1.42	.214	0.94	0.77	1.15	.544
Female gender	1.02	0.80	1.29	.901	0.94	0.80	1.11	.490
Study site	1.05	0.86	1.29	.639	0.95	0.79	1.14	.553
Distress score of 8 or more	1.37	1.11	1.68	.003	0.98	0.82	1.16	.793
Chest discomfort	1.01	0.79	1.31	.919	0.96	0.77	1.19	.721
Chest pain	1.06	0.84	1.34	.610	1.30	1.07	1.58	.009
Chest pressure	1.04	0.83	1.30	.721	1.17	0.96	1.42	.112
Short of breath	0.73	0.60	0.89	.002	0.92	0.76	1.12	.398
Unusual fatigue	0.72	0.57	0.90	.004	0.79	0.66	0.94	.008
Lightheadedness	1.11	0.88	1.40	.363	1.09	0.90	1.31	.375
Nausea	1.18	0.97	1.43	.094	1.30	1.08	1.57	.006
Arm pain	1.01	0.79	1.28	.951	1.02	0.84	1.25	.817
Sweating	1.35	1.10	1.67	.005	1.05	0.87	1.26	.629
Shoulder pain	1.04	0.81	1.34	.754	1.00	0.80	1.24	.996
Upper back pain	0.99	0.76	1.28	.920	0.80	0.66	0.96	.019
Palpitations	0.95	0.75	1.21	.688	1.14	0.94	1.37	.194
Indigestion	0.92	0.74	1.15	.465	0.86	0.70	1.04	.123

ACS, acute coronary syndrome; ED, emergency department; HR, hazard ratio; LL, lower limit; UL, upper limit; HRs > 1.0 represent a greater likelihood of reaching the ED earlier. HRs < 1.0 represent a decreased likelihood of reaching the ED.

Prediction of time from symptom onset to ED from demographic and symptom characteristics stratified by type of ACS diagnosis

TABLE 5

	Unstable angina (<i>n</i> = 105)				NSTEMI (<i>n</i> = 251)				STEMI (<i>n</i> = 118)			
	95% CI				95% CI				95% CI			
	HR	LL	UL	<i>p</i>	HR	LL	UL	<i>p</i>	HR	LL	UL	<i>p</i>
10 year increase in age	1.12	0.94	1.33	.194	1.09	0.98	1.21	.117	1.27	1.05	1.53	.012
White race	0.85	0.49	1.48	.557	0.99	0.74	1.31	.927	1.99	1.22	3.25	.006
Female gender	0.68	0.33	1.38	.281	1.17	0.86	1.58	.324	1.20	0.71	2.03	.490
Study site	0.83	0.52	1.32	.436	1.10	0.83	1.46	.508	1.57	0.89	2.76	.119
Distress score of 8 or more	1.42	0.90	2.24	.134	1.23	0.94	1.60	.131	1.14	0.67	1.94	.636
Chest discomfort	1.13	0.57	2.22	.733	1.06	0.76	1.49	.718	1.15	0.67	1.98	.613
Chest pain	1.02	0.59	1.77	.944	0.97	0.70	1.36	.877	1.13	0.67	1.91	.636
Chest pressure	1.07	0.58	1.98	.830	1.11	0.81	1.51	.522	1.12	0.67	1.88	.663
Short of breath	0.70	0.42	1.17	.172	0.76	0.58	0.99	.044	0.68	0.38	1.22	.201
Unusual fatigue	0.74	0.46	1.20	.220	0.74	0.55	1.00	.046	0.65	0.33	1.29	.219
Lightheadedness	0.60	0.34	1.06	.080	1.72	1.25	2.37	.001	1.49	0.82	2.71	.193
Nausea	0.88	0.48	1.63	.695	1.47	1.12	1.93	.005	1.31	0.84	2.07	.237
Arm pain	0.85	0.41	1.75	.656	0.91	0.68	1.23	.553	1.11	0.61	2.00	.742
Sweating	1.34	0.79	2.27	.286	1.15	0.83	1.59	.411	1.00	0.67	1.49	.986
Shoulder pain	1.07	0.57	2.00	.840	1.17	0.84	1.64	.342	1.58	0.86	2.93	.144
Upper back pain	1.42	0.81	2.50	.221	0.89	0.64	1.23	.473	1.33	0.71	2.50	.367
Palpitations	1.31	0.80	2.14	.282	0.88	0.64	1.22	.437	0.68	0.32	1.47	.329
Indigestion	0.81	0.48	1.37	.433	0.88	0.66	1.17	.369	1.03	0.62	1.73	.903

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ACS, acute coronary syndrome; ED, emergency department; HR, hazard ratio; LL, lower limit; UL, upper limit; HRs > 1.0 represent a greater likelihood of reaching the ED earlier. HRs < 1.0 represent a decreased likelihood of reaching the ED. NSTEMI is non-ST elevated myocardial infarction. STEMI is ST elevated myocardial infarction.