J Behav Med (2012) 35:420–430
DOI 10.1007/s10865-011-9369-x

Symptom experience during acute coronary syndrome and the development of posttraumatic stress symptoms

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Received: May 5, 2011 / Accepted: June 29, 2011 / Published online: July 9, 2011

Abstract There is growing evidence for the development of posttraumatic stress symptoms as a consequence of acute cardiac events. Acute coronary syndrome (ACS) patients experience a range of acute cardiac symptoms, and these may cluster together in specific patterns. The objectives of this study were to establish distinct symptom clusters in ACS patients, and to investigate whether the experience of different types of symptom clusters are associated with posttraumatic symptom intensity at six months. ACS patients were interviewed in hospital within 48 h of admission, 294 patients provided information on symptoms before hospitalisation, and cluster analysis was used to identify patterns. Posttraumatic stress symptoms were assessed in 156 patients at six months. Three symptom clusters were identified; pain symptoms, diffuse symptoms and symptoms of dyspnea. In multiple regression analyses, adjusting for sociodemographic, clinical and psychological factors, the pain symptoms cluster ($\beta = .153$, $P = .044$) emerged as a significant predictor of posttraumatic symptom severity at six months. A marginally significant association was observed between symptoms of dyspnea and reduced intrusive symptoms at six months ($\beta = -.156$, $P = .061$). Findings suggest acute ACS symptoms occur in distinct clusters, which may have distinctive effects on intensity of subsequent posttraumatic symptoms. Since posttraumatic stress is associated with adverse outcomes, identifying patients at risk based on their symptom experience during ACS may be useful in targeting interventions.

Keywords Posttraumatic stress symptoms · Acute coronary syndrome · Cardiac symptoms · Cluster analysis

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Introduction

Acute coronary syndrome (ACS) patients experience a variety of symptoms on the onset of the coronary event. The term 'acute coronary syndrome' encompasses a spectrum of acute coronary heart disease manifestations that includes ST-elevation myocardial infarction (STEMI), non ST-elevation myocardial infarction (NSTEMI), and unstable angina (UA). Traditionally, chest pain has been seen as the primary indicator of ACS and many health-related texts and educational materials for the public focus on chest pain as the cardinal symptom (Canto et al., 2000). Yet, although chest pain is the most common symptom of ACS (Canto et al., 2000), patients experience a wide variety of other acute cardiac symptoms. The general public appears to be knowledgeable about the association of chest pain with ACS (Caldwell & Miaskowski, 2000; Goff et al., 1998;

Johnson & King, 1995; Zerwic, 1998), but there is a lack of awareness of other symptoms that are also associated with impending ACS (Caldwell & Miaskowski, 2000; Dempsey et al., 1995; Finnegan et al., 2000; Goff et al., 1998; Horne et al., 2000; Johnson & King, 1995; Ruston et al., 1998; Zerwic, 1998). Lindgren et al. (2008) reported that chest pain was not the primary symptom experienced by elderly cardiac patients (>65 years); rather, fatigue and shortness of breath were the most prevalent symptoms in this population (76.1% and 61.5%, respectively). There is also evidence suggesting that women are more likely to present with 'atypical' symptoms during acute cardiac events (Chen et al., 2005).

There is limited research on patterns of symptoms or clusters of symptoms in ACS patients, and studies vary in the numbers and labelling of clusters identified (Chen et al., 2005; Lindgren et al., 2008; Riegel et al., 2010; Ryan et al., 2007). Lindgren et al. (2008) described three symptom clusters based on pre-admission cardiac symptoms, and named these; classic ACS symptoms (defined as, severe ischemic pain and moderate fatigue), a diffuse symptom cluster (low intensity of all symptoms, except for mild shortness of breath and fatigue), and a weary symptom cluster (severe fatigue, sleep disturbance, and shortness of breath but only moderate ischemic pain). Patients who experienced the weary symptom cluster had the most disadvantageous risk profile (i.e. a history of hypertension, higher prevalence of heart failure and diabetes) and reported poorer quality of life and greater psychological distress. Riegel et al. (2010) reported four symptom clusters; classic ACS characterised predominantly by chest pain, other pain symptoms (neck, throat, jaw, back, shoulder, and arm pain), stress symptoms (including shortness of breath, sweating, nausea, indigestion, dread, anxiety), and a diffuse symptom cluster (low frequency of most symptoms). Patients in the diffuse cluster had significantly higher mortality rates at 2-year follow-up than did patients in the other clusters. The experience of different clusters of either premonitory or acute symptoms may have an influence on the levels of distress experienced by ACS patients in the aftermath of the acute event. Research in psychiatry and oncology has demonstrated that combinations or clusters of symptoms are of greater importance than individual symptoms because multiple concurrent symptom experiences may have synergistic effects on quality of life and psychological distress in comparison with individual symptoms (Dodd et al., 2001; Gift et al., 2004; Kim et al., 2005).

There is growing evidence for the development of symptoms of posttraumatic stress disorder (PTSD) as a consequence of acute cardiac events in patients with coronary heart disease (CHD) (Gander & von Känel, 2006; Spindler & Pedersen, 2005). PTSD classically occurs following traumatic experiences involving external stressors, but recently, attention has been drawn to the impact of stressful medical events on posttraumatic symptoms (Tedstone & Tarrier, 2003). Symptoms of PTSD fall into three broad categories: (1) intrusive symptoms (re-experiencing the event, intrusive thoughts, flashbacks, nightmares); (2) avoidance of reminders of the traumatic event and emotional numbing; and (3) physiological hyperarousal (difficulty staying or falling asleep, anger and irritability, hypervigilance, difficulty concentrating, and an exaggerated startle response). Experiencing different types or combinations of ACS symptoms may be associated with posttraumatic responses following acute cardiac events. One study of patients with implantable cardioverter-defibrillators, who had survived a cardiac arrest or myocardial infarction (MI), found that patients who subsequently developed PTSD reported significantly more cardiac symptoms compared with those who did not develop PTSD (Ladwig et al., 2008). The perceived severity rather than the objective severity of a cardiac condition as determined by cardiac criteria may be associated with PTSD. Patients may perceive some symptoms or clusters of symptoms as more frightening, confusing or vivid than others, leading to a greater likelihood of posttraumatic stress symptoms developing. However, patients' vulnerability to posttraumatic stress may not only be influenced by symptom type, but also their intensity. Some studies suggest that PTSD is associated less with objective clinical disease burden but more with subjective severity as indexed by chest pain (Cohen et al., 2000; Doerfler et al., 1994). Although previous studies suggest that objective severity of ACS is unrelated to subsequent posttraumatic symptoms (Doerfler et al., 2005; Ginzburg, 2006; Pedersen et al., 2003; Whitehead et al., 2006), a number of studies suggest that patients' subjective experience of pain during ACS is associated with subsequent posttraumatic reactions (Doerfler et al., 2005; Whitehead et al., 2006).

Previous research has shown that PTSD develops in 15% of MI patients on average (Spindler & Pedersen, 2005). However, the prevalence ranges from 0% to 32% (Jones et al., 2007; van Driel and Op den Velde 1995). Posttraumatic stress is associated with a number of adverse consequences following ACS. For example, PTSD is associated with non-adherence to cardiac medications in MI patients, which in turn is related to poor medical outcome (Shemesh et al., 2001). A study by Shemesh et al. (2004) found that PTSD was associated with almost a threefold increased risk of readmission for cardiovascular events 1.5 years post admission for MI. This increase in risk may relate to the increased non-adherence observed in the sample, or they may be related to biological correlates of PTSD, which may put an additional strain on an already ailing heart.

Although many risk factors have been associated with the development of PTSD following trauma, several of these appear not to be associated with the disorder in cardiac populations, suggesting that PTSD may have distinct correlates in this group of patients. Posttraumatic stress symptoms in ACS patients are predicted by subjective perceptions and emotional reactions in the immediate aftermath of the acute cardiac event. Greater levels of fear and helplessness (Guler et al., 2009; Wiedemar et al., 2008), acute stress or fear of dying (Whitehead et al., 2006), anxiety (Rocha et al., 2008), perceived impact of the illness (van Driel & Op den Velde, 1995), type D personality (Pedersen & Denollet, 2004; Whitehead et al., 2006), recurrent cardiac symptoms (Wikman et al., 2008) and female gender (Roberge et al., 2010) are variables that have been identified as increasing the likelihood of posttraumatic stress symptoms in cardiac patients. Previous studies also suggest that, when left untreated, posttraumatic symptoms are persistent in many cardiac patients (Abbas et al., 2009; Wikman et al., 2008).

Although previous studies of symptom clusters in ACS patients have found associations with poorer health outcomes, the possibility that posttraumatic stress symptoms are more likely to follow certain types of acute cardiac symptom experiences has not yet been studied. In ACS patients, the traumatic stressor is triggered by an internal cardiac event rather than an external threat, and so cannot be physically avoided in the same way as other traumas. Therefore, there may be ambiguity in the extent to which patients complete assessment instruments with respect to the index ACS, or take into account current health and symptomatology. For these reasons, the primary focus of this study was on severity of posttraumatic symptoms, rather than PTSD as a diagnosis.

Cluster analysis has proved useful in grouping patients based on their symptom experience in previous studies (Chen et al., 2005; Lindgren et al., 2008; Riegel et al., 2010; Ryan et al., 2007). The present analyses were largely exploratory in nature; however, the study had two main purposes. Firstly, we aimed to identify subgroups of patients with distinct ACS symptom experience using cluster analysis of acute symptoms reported, and secondly to investigate whether different symptom clusters are associated with the intensity of posttraumatic symptoms reported at six months. Symptoms of depression are also common following ACS and are predictors of subsequent cardiovascular morbidity (Nicholson et al., 2006; Thombs et al., 2008). The co-morbidity between PTSD and depression in the general trauma literature is well documented (McFarlane & Papay, 1992). A substantial proportion of PTSD sufferers also meet diagnostic criteria for depression, with prevalence rates ranging from 21% to 94% (Mollica et al., 1999; Salcioglu et al., 2003). Depressed mood is also a predictor of posttraumatic stress symptoms following ACS (Whitehead et al., 2006; Wikman et al., 2008). In addition to studying posttraumatic symptoms, we therefore assessed the relationship between acute cardiac symptom clusters and depressed mood three weeks after ACS.

Methods

Patients

The participants in this study were 294 patients (consecutive sample) admitted with ACS to one general hospital serving Southwest London between June 2007 and October 2008 as part of a larger study of psychosocial aspects of ACS (Steptoe et al., 2011; Wikman et al., 2011). A further four patients were recruited, but did not provide data on acute cardiac symptoms. Patients were included if they fulfilled the following criteria: a diagnosis of ACS based on the presence of relevant pain plus verification by diagnostic electrocardiographic (EKG) changes (new ST elevation >0.2 mV in 2 contiguous leads in leads V1, V2 or V3 and >0.1 mV in 2 contiguous other leads, ST depression >0.1 mV in 2 contiguous leads in the absence of any QRS confounders, new left bundle branch block or dynamic T wave inversion in more than one lead), troponin T or troponin I $\geq$ 99th percentile of the upper reference limit and/or a creatine kinase measurement more than twice the upper range of normal for the measuring laboratory (Thygesen et al., 2007). Additional inclusion criteria were age of 18 years or over, absence of co-morbid conditions that might influence either symptom presentation or mood, other conditions that might cause troponin positivity (Ammann et al., 2004), and ability to complete interviews and questionnaires in English. The study was approved by the Wandsworth Research Ethics Committee and written informed consent was obtained. In-hospital data were collected between June 2007 and October 2008. On the days that recruitment was conducted, 666 potentially eligible patients were admitted. Of these, 125 (19%) had been discharged or transferred to a different hospital before the patient could be recruited into the study, 90 (14%) were too clinically fragile (e.g. critical ischaemia, ventricular tachyarrhythmia) to take part, 58 (9%) patients declined to participate, 27 (4%) could not speak English, 23 (3%) were in confusional states, seven patients (1%) died in hospital and a further 38 (6%) were excluded for other reasons. Patients who were not included in the study were older on average than those who did take part (mean age 66.7, SD 14.0 vs. 60.2, SD 11.6, $P < .001$), and were more likely to be female (32.0% vs. 16.1%, $P < .001$).

Procedure

Patients were approached by a member of the research team as soon as possible after admission to hospital at which point the study was explained and written informed consent was obtained. Interviews were conducted in hospital within 48 h of admission (time 1), during which patients acute cardiac symptoms, fear responses, pain and distress during their ACS were measured. A follow-up home interview (time 2) took place on average 21 days (SD 8.54) following the original admission date. During the home interview patients completed a series of psychosocial measures including assessment of depressed mood. Six months following admission, patients completed a telephone interview and self-completion questionnaire (time 3), at which time posttraumatic stress symptoms were measured.

Measures

Clinical and sociodemographic variables

Clinical information was obtained from the hospital admission records. Admission ECGs and troponin T or creatine kinase levels were reviewed by a cardiologist in order to classify patients as presenting STEMI, NSTEMI or UA. This information was subsequently categorised as a binary variable (STEMI vs. NSTEMI/UA) for analyses purposes, since NSTEMI and UA are closely related clinical manifestations of acute coronary disease (ACC/AHA, 2007). Clinical risk was assessed using the algorithm developed in the Global Registry of the Acute Coronary Events (GRACE) study (Eagle et al., 2004). The GRACE index uses nine measures on hospital admission to define the risk of six-month post discharge death, and has been independently verified for both short- and long-term clinical morbidity (Tang et al., 2007). During the hospital interview patients were questioned in detail about acute symptoms experienced during ACS, and these were subsequently classified into 13 symptom categories; left arm pain, jaw pain, tooth/throat/neck pain, back pain, shoulder pain (including right arm pain), gastrointestinal discomfort (indigestion, stomach pain), sweating profusely, shortness of breath, numbness in hands/arms, nausea (including vomiting), dizziness (including collapsing/fainting), fatigue and chest pain. Information on the interval between symptom onset and hospital admission was obtained, and patients were classified as delayed in seeking care if the interval exceeded two hours (h). Subjective pain (general pain, regardless of location) experienced during the ACS was rated on a 10 point scale, with higher scores indicating more intense pain.

Sociodemographic information included age, gender, ethnicity, employment status at admission, and educational qualifications. A social deprivation index was computed based on three criteria: (a) living in a crowded household (defined as one or more persons per room); (b) not having access to a car or van and; (c) renting as opposed to owning a home (Wardle et al., 2002). Patients were categorised as low deprivation (negative on all items), medium deprivation (1 positive score) or high deprivation (2–3 positive items) on this index.

Psychological Variables

Depressed mood Depressed mood was assessed during the home interview three weeks following admission for ACS using the Beck Depression Inventory (BDI), a standard measure of depressive symptoms (Beck & Steer, 1987). The BDI is a 21-item self-report measure that assesses the severity of depressive symptoms over the past week. Patients rate symptoms from none (0) to severe (3). The scores can range from 0 to 63 with higher scores indicating more severe depressive symptoms. The Cronbach's alpha for the scale was .86.

Acute distress and fear of dying During the in-hospital interview, patients were asked to rate their experience of acute distress and fear at the time of their ACS. This was measured using three items; 'I was frightened when the symptoms came on', 'I thought I might be dying when the symptoms came on', and 'I found my cardiac event stressful'. Each item was rated on a five point scale; not at all true, slightly true, somewhat true, very true and extremely true. The Cronbach's alpha for the scale was .82.

Posttraumatic stress symptoms The presence and severity of posttraumatic stress symptoms was assessed at six months using the PTSD Symptom Scale-Self Report version (PSS-SR) (Foa et al., 1993), the precursor of the Posttraumatic Diagnostic Scale (Foa et al., 1997). Participants were asked specifically to answer the questions in relation to the heart symptoms which lead to their hospital admission six months earlier for ACS. The PSS-SR is a 17-item scale with items corresponding to the DSM-IV criteria (American Psychiatric Association, 1994) for diagnosis of PTSD for the three dimensions of intrusions/re-experiencing, avoidance and arousal. Each item is rated on a four point scale, ranging from 0 (not at all) to 3 (5 or more times per week) to indicate frequency of patients' experiencing symptoms during the past two weeks. In this study the PSS-SR was analysed as a continuous variable rather than as a diagnostic instrument. This measure

showed good internal reliability with a Cronbach's alpha of .91. The alpha's for the subscales were .71 for intrusion, .80 for avoidance, and .81 for arousal.

Statistical analyses

Attrition analyses

Of the 298 patients recruited at time 1, 40 patients (13%) declined to participate in the study any further, 12 (4%) patients were excluded because of extreme ill-health (patient too unwell to complete follow-up interview, readmission to hospital, memory problems), and 46 (16%) patients could not be re-contacted despite repeated efforts by telephone and mail. Two hundred and ninety four patients (99%) provided complete data on ACS symptoms at time 1. A total of 200 (67%) were re-interviewed at six month follow-up (time 3), 156 (52%) returned questionnaires with complete data on the assessment of posttraumatic symptoms. Patients included in the analyses of posttraumatic stress symptoms at six months did not differ from the remainder in gender, ethnicity, education, employment status, or in any of the clinical variables ($P > .05$). In addition, there was no difference in cluster membership ($\chi^2(2) = .11$, $P = .949$) or depressed mood at time 2 (H (1) = .389, $P = .533$) between those who participated at time 1 and time 3 compared with those who dropped out before time 3. However, those who did not complete the six-month follow-up were more likely to be younger ($H(1) = 7.665$, $P = .006$) and in the higher social deprivation categories than were completers ($\chi^2(2) = 14.72$, $P < .001$).

Main analyses

Statistical analyses were performed using SPSS software, version 17 (SPSS Inc, Chicago, IL). ACS symptoms from 294 patients were analysed using 2-step cluster analysis. The log-likelihood distance measure was used, with patients categorised under the cluster with the largest log-likelihood. No prescribed number of clusters was suggested. The Bayesian Information Criterion (BIC) was used to judge adequacy of the final solution. The 2-step method was chosen, and the log-likelihood distance was used, as this method accommodates categorical variables and does not require prior knowledge of the number of clusters. Therefore, this method is suitable for an exploratory analysis of the data. Examination of the composition of clusters was performed to determine the importance of each variable in shaping the clusters. For categorical variables this is achieved by calculating a χ^2 value that compares the observed distribution of values of a variable within a cluster to the overall distribution of values.

Analysis of variance (or independent samples Kruskal–Wallis test for non-normally distributed variables) and χ^2 statistics were used to examine between cluster differences on baseline clinical, sociodemographic and psychological variables for continuous and categorical variables, respectively. There were no significant differences between clusters in baseline characteristics among those who did not complete the six-month posttraumatic stress assessment compared with those who did. Therefore, multivariable regressions on posttraumatic symptoms at six months were conducted on data available from 156 patients to identify independent predictors of symptom levels. The association between symptom clusters and posttraumatic stress symptoms and the posttraumatic symptom sub-scales of intrusion, avoidance and arousal, were tested in 2-step hierarchical models. Model 1 included baseline sociodemographic, clinical and psychological factors. In Model 2, sociodemographic, clinical and psychological factors were entered at step 1, symptom clusters were added on the second step as two binary dummy variables (i.e. one fewer than the number of discrete categories) (Tabachnick & Fidell, 1996), using the diffuse symptoms cluster as the reference category. Standardised regression coefficients (β) with Standard Error (SE) are presented. None of the variables included in the final regression models showed multicollinearity according to variance inflation factor and tolerance values.

Results

Sample characteristics and cluster solution

The sample of 294 patients who provided data on ACS symptoms was aged 60 years (SD 11.57) on average, predominantly male (84%), of white ethnic origin (83%), and in employment at the time of the illness (57%). The majority of patients had experienced a STEMI (87%). ACS severity as defined by the GRACE score was moderate (mean 92.86, SD 27.62), 13% of patients had experienced a previous myocardial infarction (MI), and subjective pain ratings were high (mean 7.02, SD 2.50). The frequencies for each of the 13 ACS symptoms analysed are shown in Table 1. Chest pain was the commonest symptom (87%), followed by sweating (72%) and shortness of breath (51%). Patients reported experiencing five symptoms on average (mean 5.06, SD 2.33).

Characteristics of clusters

A three-cluster solution was identified based on the BIC and log-likelihood distance. For three clusters, the BIC

Table 1 Frequency of ACS symptoms for the total sample and the distribution of ACS symptoms within clusters

ACS symptom	Total sample % ($n = 294$)	Clusters		
		Pain symptoms % ($n = 121$)	Diffuse symptoms % ($n = 101$)	Symptoms of dyspnea % ($n = 72$)
Chest pain	87.4	95.0	80.2	84.7
Sweating	72.4	**88.4**	**39.6**	**91.7**
Shortness of breath	51.4	62.8	**18.8**	**77.8**
Shoulder pain	45.6	**72.7**	45.5	**.0**
Nausea	43.5	**60.3**	**13.9**	56.9
Fatigue	35.7	**57.9**	**14.9**	27.8
Dizzy or collapse	34.7	**51.2**	**5.9**	47.2
Left arm pain	31.3	**45.5**	22.8	19.4
Numbness hands/arms	30.6	**49.6**	**10.9**	26.4
Jaw pain	27.9	**58.7**	**10.9**	**.0**
Back pain	23.1	**37.2**	21.8	**1.4**
Gastro intestinal discomfort	13.6	8.2	9.9	11.1
Tooth/throat/neck pain	8.5	14.9	5.0	2.8

Numbers in bold represents those symptoms that significantly contributed to distinguishing cluster membership

ACS acute coronary syndrome, *n* number

ratio of change was .666, which was the maximum value for clusters ≥ 3. The ratio of distances value was 1.793, maximum of all solutions.[1] The first symptom cluster was characterised by a range of pain symptoms coupled with somatic reactions to pain, such as pain in the left arm, shoulders and back, jaw pain, numbness in hands and arms, sweating, nausea and dizziness, and fatigue. This cluster was named pain symptoms (n = 121). The second cluster (n = 101), contained a range of symptoms (jaw pain, sweating, shortness of breath, numbness, nausea, dizziness and fatigue), but since none were highly represented this cluster was named diffuse symptoms. The third, and final cluster was defined by an absence of jaw and shoulder pain, a negligible amount of back pain coupled with a high frequency of sweating and shortness of breath, and is therefore referred to as symptoms of dyspnea (n = 72). The composition of the three clusters is shown in Table 1. These numbers represent the frequency of symptoms within each cluster, and symptoms that significantly contributed to distinguishing between clusters are highlighted in bold. For example, although the symptom of chest pain was prevalent in all three clusters (95%, 80% and 85%, for the pain, diffuse and dyspnea symptom clusters, respectively), the distribution in all of the clusters was fairly similar to the overall distribution; therefore chest pain was not an important variable in forming the clusters or distinguishing cluster membership.

In order to assess the reliability of the clusters we re-ran the cluster analysis on a random selection (50%) of the sample. This analysis yielded a three-cluster solution similar to the original analysis presented above. The composition of the three clusters that emerged in this analysis was largely the same as the original, with a few exceptions. The symptoms of sweating and left arm pain were distinguishing symptoms in the pain symptoms cluster in the original analysis, but did not contribute to distinguishing between clusters in the reliability analysis. However, gastrointestinal symptoms did emerge as distinguishing between clusters, whereas in the original cluster analysis this was not the case. The second cluster that emerged was similar to the diffuse symptoms cluster with the exception of the symptom of pain in arms/shoulders. Whereas the diffuse symptoms cluster was characterised by the absence of this symptom, the second cluster that emerged from the reliability analysis was characterised by the presence of this symptom. Sweating profusely was highly represented in the dyspnea cluster, but did not emerge as a distinguishing symptom in the third cluster in the reliability analysis.

Differences between clusters on sociodemographic, clinical and psychological variables were assessed (Table 2). Patients in the dyspnea cluster were significantly older, and had significantly higher GRACE risk scores than

[1] 2-step cluster analysis, auto-clustering solution for first 5 solutions: 1 cluster; BIC 4419.161: 2 clusters; BIC 4138.803, BIC ratio of change 1.000, Ratio of distance 1.360: 3 clusters; BIC 3952.190, BIC ratio of change .666, Ratio of distance 1.793: 4 clusters; BIC 3880.778, BIC ratio of change .255, Ratio of distance 1.154: 5 clusters; BIC 3828.742, BIC ratio of change .186, Ratio of distance 1.047.

Table 2 Sociodemographic, clinical and psychological characteristics by ACS symptom clusters

	Mean (SD) or %			
	Pain symptoms ($n = 121$)	Diffuse symptoms ($n = 101$)	Symptoms of dyspnea ($n = 72$)	*P* value
Sociodemographic factors				
Age	58.05 (11.40)[a]	61.34 (11.61)[a,b]	62.15 (11.36)[b]	.03
Gender (male)	83.5	85.1	81.9	.85
Ethnicity (white)	80.2	81.2	90.3	.16
Educational attainment				
None	25.8	31.7	29.2	.61
Basic	25.0	28.7	19.4	
Secondary	35.0	24.8	34.7	
Degree	14.2	14.9	16.7	
Social deprivation				
Low	56.7[a]	74.7[b]	63.4[a,b]	.03
Medium	25.8	20.2	22.5	
High	17.5[a]	5.1[b]	14.1[a,b]	
Employed (yes)	58.3	59.4	50.7	.48
Clinical factors				
ACS type (STEMI)	89.3	83.2	88.9	.35
Number of vessels diseased	2.02 (.86)	1.74 (.84)	1.89 (.99)	.07
Cardiac arrest (yes)	5.8	5.9	12.5	.18
GRACE score	87.24 (27.37)[a]	96.57 (26.39)[b]	97.08 (28.44)[b]	.01
Previous MI (yes)	9.9	16.0	13.9	.39
Symptoms attributed to ACS (yes)	28.9	26.7	31.9	.76
Delay (yes)	34.5[a]	44.9[b]	23.6[a]	.02
Psychological factors				
Pain score (1–10 scale)	7.60 (2.18)[a]	6.88 (2.30)[b]	6.25 (3.12)[b]	.006[#]
Acute distress/fear of dying	1.98 (1.09)[a]	1.46 (1.07)[b]	1.96 (1.14)[a]	.002
Depressed mood	7.84 (7.78)	5.38 (4.51)	6.70 (7.42)	.13[#]
Posttraumatic stress symptoms (six months)	10.47 (9.53)[a]	6.76 (6.81)[b]	7.22 (7.25)[b]	.03[#]
Intrusive symptoms	1.82 (2.16)[a]	1.21 (1.69)[a,b]	1.06 (1.71)[b]	.05[#]
Avoidance symptoms	4.63 (3.97)[a]	3.05 (3.11)[b]	3.35 (3.42)[a,b]	.02[#]
Arousal symptoms	4.01 (4.15)	2.50 (2.78)	2.82 (3.07)	.09[#]

P values refer to univariate analysis of variance test (for continuous variables) or χ^2 test (for categorical variables). [#] Independent samples Kruskal–Wallis test (for non-normally distributed variables). Values on each line with a different superscript are significantly different from each other ($P < .05$)

ACS acute coronary syndrome, *MI* myocardial infarction, *GRACE* global registry of the acute coronary events index, *n* number

those in the pain symptoms cluster. Patients in the diffuse symptoms cluster also scored significantly higher on the GRACE risk index than those in the pain symptoms cluster. There were significantly more high social deprivation patients in the pain symptoms cluster than expected. Not surprisingly, those patients within the pain symptoms cluster reported significantly greater pain during ACS than did patients with diffuse symptoms or symptoms of dyspnea. A larger proportion of patients in the diffuse symptoms cluster delayed in seeking medical care than in the other two groups. Finally, patients in the pain symptoms and dyspnea clusters reported significantly higher acute distress and fear of dying than did patients in the diffuse symptoms cluster. There were no significant differences in depressed mood three weeks after ACS, although patients in the pain symptoms cluster reported the highest levels of depressive symptoms. Patients in the pain symptoms cluster reported significantly more intense posttraumatic stress symptoms at six months compared with the other clusters. Comparisons between clusters on the posttraumatic stress symptom sub-scales showed that patients in the pain symptoms cluster reported significantly more intense

intrusion symptoms than did the patients in the dyspnea cluster, and significantly greater avoidance symptoms than patients in the diffuse symptoms cluster. No other significant differences were observed between clusters on the clinical or sociodemographic variables.

Posttraumatic stress symptoms by cluster membership

Multiple regression models were run to test whether the effect of ACS symptom clusters on six-month posttraumatic stress symptoms were independent of clinical and sociodemographic factors (Table 3). These showed that age, gender, social deprivation, GRACE score, depressed mood, acute stress symptoms, subjective pain and pre-hospital delay together accounted for 42.4% of variance in six-month posttraumatic stress symptom intensity. In step 2, the pain symptoms cluster significantly predicted posttraumatic stress symptom severity six months following ACS ($\beta = .153$, $P = .044$), accounting for an additional 3.9% of variance in the model. Depressed mood three weeks after hospitalisation was also a strong predictor of greater posttraumatic symptoms at six months ($\beta = .645$, $P < .001$). Analyses of the posttraumatic stress symptom sub-scales revealed a marginally significant association between dyspnea symptoms and reduced posttraumatic intrusive symptoms at six months ($\beta = -.156$, $P = .061$).

Discussion

An exploratory 2-step cluster analysis identified a three-cluster solution based on 13 self-reported acute ACS symptoms in this sample of patients. The clusters identified were named pain symptoms (41%), diffuse symptoms (34%), and symptoms of dyspnea (24%). Cluster analysis proved to be useful for grouping patients based on their acute symptoms as it predicted differences in subsequent emotional experience. The primary difference among the clusters in levels of posttraumatic stress symptoms was between the pain symptoms cluster and the other two clusters, with the patients in the pain cluster having significantly greater symptoms of posttraumatic stress at six month follow-up. However, there was a marginally significant association between the dyspnea cluster and less intense intrusive posttraumatic symptoms at six months.

These findings may indicate that more distinctive or severe symptomatology (such as those experienced in the pain cluster) is more psychologically distressing in the

Table 3 Multivariable predictors of posttraumatic stress symptoms at six months post ACS

	Posttraumatic stress symptoms (total score)		Arousal symptoms		Intrusion symptoms		Avoidance symptoms	
	β (SE)	P	β (SE)	P	β (SE)	P	β (SE)	P
Step 1								
Age	.044 (.125)	.727	.032 (.130)	.805	.011 (.132)	.936	.095 (.131)	.471
Gender	.002 (.073)	.976	−.017 (.076)	.826	.018 (.077)	.818	−.015 (.077)	.847
Social deprivation	.002 (.077)	.980	−.011 (.079)	.891	.062 (.082)	.446	.023 (.081)	.780
GRACE	.011 (.122)	.931	.018 (.127)	.886	.023 (.129)	.859	−.027 (.128)	.833
Depressed mood (hospital)	.646 (.080)	.000	.635 (.082)	.000	.513 (.084)	.000	.606 (.084)	.000
Acute stress symptoms	.056 (.080)	.487	.008 (.083)	.919	.142 (.084)	.095	.027 (.084)	.743
Pain score	−.063 (.074)	.397	−.037 (.076)	.630	−.085 (.078)	.282	−.053 (.078)	.497
Pre-hospital delay (>2 h)	.012 (.071)	.864	.005 (.074)	.942	−.042 (.075)	.582	.032 (.075)	.672
R^2	.424	.000	.384	.000	.353	.000	.362	.000
Step 2								
Age	.076 (.123)	.538	.043 (.131)	.740	.057 (.130)	.660	.120 (.131)	.3632
Gender	.016 (.071)	.822	−.005 (.076)	.949	.035 (.075)	.640	−.004 (.076)	.963
Social deprivation	−.006 (.075)	.940	−.023 (.078)	.767	.057 (.080)	.473	.015 (.080)	.847
GRACE	−.024 (.120)	.839	.002 (.127)	.985	−.025 (.127)	.846	−.055 (.128)	.668
Depressed mood	.645 (.078)	.000	.624 (.081)	.000	.518 (.083)	.000	.602 (.083)	.000
Acute stress symptoms	.066 (.078)	.404	.013 (.083)	.873	.161 9.083)	.054	.034 (.083)	.681
Pain score	−.123 (.075)	.106	−.079 (.080)	.322	−.154 (.080)	.056	−.103 (.080)	.201
Pre-hospital delay (>2 h)	−.019 (.070)	.789	−.014 (.074)	.851	−.078 (.074)	.298	.006 (.075)	.938
Pain cluster (vs. reference)	.153 (.075)	.044	.137 (.079)	.074	.118 (.079)	.139	.141 (.080)	.079
Dyspnea cluster (vs. reference)	−.097 (.078)	.215	−.042 (.082)	.082	−.156 (.082)	.061	−.072 (.083)	.338
R^2	.462	.000	.406	.000	.398	.000	.391	.000

longer term than milder or more vague symptoms such as shortness of breath or perspiring. Previous research suggests that objective clinical severity of ACS does not predict intensity of subsequent posttraumatic stress symptoms (Doerfler et al., 2005; Ginzburg, 2006; Pedersen et al., 2003), but that patients' subjective experience and perceptions of severity and intensity of pain do (Whitehead et al., 2006). Depressed mood measured three weeks after hospitalisation also emerged as a significant predictor of posttraumatic stress symptoms six months later. This is consistent with previous studies of ACS patients which suggest that depression is a strong predictor of posttraumatic stress (Whitehead et al., 2006; Wikman et al., 2008). This is perhaps not surprising considering the high levels of co-morbidity of depressive and anxiety disorders (Beutel et al., 2010). Further, there is disagreement about whether the posttraumatic symptoms observed after health-related stressful events are a subtype of depressive illness or a distinct disorder. This may have implications for treatment, which may not affect subgroups equally (Bech, 2010). However, our results indicate that over and above depressed mood, experiencing particular clusters of acute ACS symptoms further increases vulnerability for developing posttraumatic symptoms.

Although our method of analysis was exploratory in nature, the clusters identified in this paper show some overlap with findings reported by Lindgren et al. (2008), Riegel et al. (2010) and Ryan et al. (2007). A 'diffuse symptoms' cluster was identified in all studies, where patients were characterised by the experience of low frequency or low intensity, diffuse or poorly defined multiple symptoms. The dyspnea cluster we identified is similar to the 'stress cluster' described by Riegel et al. (2010) in which shortness of breath, sweating, and nausea were predominant. The pain symptoms cluster observed in this study overlaps with the pain symptoms cluster described by Riegel et al. (2010) which contained primarily arm, back, shoulder, neck, throat, and jaw pain. In contrast to previous studies, our analyses did not identify a 'classic ACS cluster' characterised predominantly by chest pain alone. One reason for this may be that chest pain was one of the defining features of ACS, so was highly prevalent across patients in all three clusters.

There are a number of limitations to this study which must be acknowledged and should guide future work. Although the prospective design is a strength of the study, intervention studies are required to establish whether psychological interventions focusing on ACS symptoms and patients' interpretations of symptoms can reduce posttraumatic stress symptomatology in vulnerable individuals. Secondly, although participants were interviewed in hospital within 48 h of admission for ACS, the information about symptoms was collected after the primary treatment of the ACS, and this may have modified patients' ratings of their symptoms. A third limitation relates to the simple measures that were used to assess presence or absence of each symptom. A measure of intensity of symptoms would have been useful. Further, it is important to note that trait negative affectivity is a strong correlate of somatic symptom experience (Watson & Pennebaker, 1989) which may have influenced symptom reporting. Additionally, posttraumatic symptoms were assessed with self-report measures rather than gold standard clinical interviews. The results are also limited by the fact that the majority of patients were white European men attending a large urban hospital. Patients who were lost to follow-up tended to be more socially deprived. These factors may limit the generalisability of findings, since other populations of people with coronary disease might experience different patterns of acute symptoms. Finally, limitations related to the cluster solution must be acknowledged. Firstly, our purpose was to evaluate the relationship with acute ACS symptoms reported in-hospital with subsequent posttraumatic stress symptom severity at six months, therefore the cluster analysis was performed on the Time 1 sample only. We did not perform a stability analysis by resting the cluster solution on data collected at a different time point. Secondly, although the reliability analysis performed on a random selection (50%) of the sample produced a largely similar three-cluster solution to the original, suggesting adequate reliability, there were some minor deviations in the distribution of specific items. The characteristics of the second and third clusters that emerged from this analysis overlapped substantially with the diffuse symptoms and symptoms of dyspnea clusters, respectively, but there were greater differences between the first cluster emerging in the reliability analysis and the pain symptoms cluster. The significance of these differences is not known, and it should be noted that the reduced number of patients in the reliability analysis may also have influenced the solution.

This is the first study to describe a relationship in acute cardiac patients between presenting symptoms and subsequent experience of posttraumatic stress symptomatology. The findings may be of value to clinicians involved in treating patients who fail to adapt emotionally after an acute coronary event, and to research concerned with the relationship between acute health events and subsequent emotional responses. The findings suggest that acute ACS symptoms occur in distinct clusters, and that these have different effects on the experience of later posttraumatic stress symptoms. From the clinical perspective, targeting the symptoms in the pain cluster may be particularly important in reducing posttraumatic symptomatology, since individuals who experience these cardiac symptoms may be particularly vulnerable to developing later posttraumatic stress symptoms.

Acknowledgments This study was supported by the British Heart Foundation (RG/05/006) and the Medical Research Council. NMB was supported by the Swiss National Foundation (PBBE1-117004). The funding sources have not been involved in the submission of the manuscript or in the decision to publish the data.

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