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Link to published article http://dx.doi.org/10.2174/2666082215666191113121558

http://hdl.handle.net/2440/126151

ARTICLE TYPE

Title: Post-traumatic stress disorder in heart failure patients: a test of the cardiac disease-induced PTSD hypothesis

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Abstract

Background: Post-traumatic stress disorder (PTSD) is prevalent in approximately 12% of patients with cardiovascular disease (CVD) and such patients are at risk of further CVD morbidity and mortality. It is unknown whether CVD patients with cardiac and non-cardiac traumatic events leading to PTSD differ in medical comorbidities and psychiatric vulnerabilities. Our objective was to compare heart failure (HF) patients with cardiac and non-cardiac PTSD.

Method: A population of HF patients from 3 hospitals underwent a two-step depression and anxiety screening process to identify potential mental health treatment needs. The post-traumatic stress disorder module of the Structured Clinical Interview for DSM-IV Axis-I disorders was used to classify trauma(s) exposure, and other disorders. The patients with PTSD were sub-divided by cardiac related traumas (e.g. myocardial infarction, sudden cardiac arrest) and non-cardiac related traumas (e.g. sexual abuse, interpersonal violence).

Results: 10 patients met criteria for non-cardiac trauma and 18 patients met criteria for cardiac-induced trauma. There were no significant differences in HF aetiology or severity nor cardiac comorbidities. Time since PTSD onset was significantly longer for those with non-cardiac PTSD. Among psychiatric comorbidities, alcohol and substance abuse disorders, as well as depression were more prevalent in the patients with non-cardiac PTSD.

Conclusion: Cardiac related PTSD was associated with less alcohol and substance abuse disorders, and depression by comparison to their non-cardiac induced PTSD counterparts. Ongoing research is required to establish if cardiac-induced PTSD truly reflects a unique subtype of PTSD, and whether there are different treatment needs and therapeutic approaches for this subtype.

Keywords: post-traumatic stress disorder; depression; anxiety disorder; heart failure; cardiomyopathy; trauma;

1. INTRODUCTION

Post-traumatic stress disorder (PTSD) is associated with a higher risk of developing incident cardiovascular disease (CVD) [1]. Moreover, among populations with known or prevalent CVD, PTSD is detected in approximately 12% (95% confidence interval [CI], 9%–16%) of patients [2] and portends a poorer cardiovascular prognosis [2, 3]. However, past estimates of PTSD prevalence have generally not differentiated between CVD-related and non-CVD traumas [2, 4]. Recently, Vilchinsky et al. [5] synthesized literature pertaining to PTSD following a CVD event or intervention (e.g. myocardial infarction [MI] or heart transplant), positing that cardiac-induced PTSD was a specific subtype of PTSD. The prevalence of PTSD induced by CVD ranges from 0 to 38% of patients [2, 5, 6]. These findings suggest high uncertainty in the prevalence of CVD-induced PTSD and perhaps raise questions as to the validity of a distinct PTSD phenotype. Moreover, the CVD-induced PTSD hypothesis relies largely on information gleaned from acute coronary syndrome patients, even though PTSD is relatively common in patients with cardiomyopathies [7], heart transplant [8], implantable cardioverter defibrillator [9], coronary revascularization [10], and sudden cardiac arrest [11].

Indeed, less is known about the ways in which cardiac-induced PTSD differs from other PTSD trauma onset in populations with CVD, such as variation in biomedical or psychiatric comorbidities or vulnerabilities. This is an

important limitation to reconcile given the public health importance of PTSD [6] and the preponderance to CVD in populations exposed to trauma including veterans [7, 12, 13]. Moreover, explicating differences between trauma sub-types may uncover discrete associations between PTSD subtypes and pertinent CVD risk factors. For example, prior research indicates PTSD post-acute coronary syndrome (ACS) is associated with lower CVD medication adherence [14]. Indeed, most information regarding PTSD and CVD pertains to ACS [2, 5], with less reported for heart transplant, and ventricular assist device populations, with no specific reports among heart failure (HF). This is an important limitation to overcome because of the burgeoning HF population worldwide [15], coupled with the adverse cardiovascular outcomes for HF patients with PTSD [7, 12, 13]. This study aims to compare the characteristics of HF patients with cardiac and non-cardiac PTSD.

2. METHODS

2.1 Population

This study reports data from HF patients participating in a HF self-management program (HFSMP) at 3 public hospitals in the North and Western urban areas of Adelaide, South Australia (Queen Elizabeth Hospital, Royal Adelaide Hospital, Lyell McEwin Hospital). HFSMP nurses initiated a standardized screening protocol for depression, anxiety, and panic attack [16]. Patients in this study provided written consent. This study complies with the Declaration of Helsinki and the study approved by the human research ethics committee of the Queen Elizabeth and Lyell McEwin hospital (#HREC/12/TQEHLMH/188).

2.2 Psychiatric assessments

In this HFSMP screening consisted of assessment for depression, (Patient Health Questionnaire-9 items, PHQ-9), anxiety (Generalized Anxiety Disorder-7 items, GAD-7) and panic-attack ("*In the last 4 weeks, have you had an anxiety attack – suddenly feeling fear or panic?*") [16] as previously described [17]. Total 81 HF patients with a positive screen for depression, anxiety, or panic attack were referred for further assessment from 404 eligible HF patients. 73 referred patients underwent structured psychiatric interview to determine disorder onset, comorbidities, and treatment needs. At this time, the PHQ-9, GAD-7 and panic-attack screening question were repeated.

The post-traumatic stress disorder module of the Structured Clinical Interview for DSM-IV Axis-I disorders [18] was used to classify trauma(s) exposure, and other disorders including personality disorders [18, 19]. Diagnoses were made for treatment purposes and verified by two senior clinical psychologists once per month. For the purposes of this research, PTSD was categorized according to the trauma type preceding PTSD onset, classified as cardiac or non-cardiac.

2.3 Medical comorbidities

HF etiology was determined from medical records and the HFSMP nurse practitioner who referred each patient (blinded to trauma exposure). The clinical presentation of HF at the most recent hospital admission preceding the psychiatric interview was classified consistent with the European Society of Cardiology [15]. Comorbidities and hospitalization data were classified according to International Classification of Diseases and Related Health Problems 10th Revision criteria [20] and extracted from electronic medical records and data linkage between hospitals, blinded to psychiatric history. The methods for audit of cardiac events is reported elsewhere [21].

2.4 Statistical analyses

Data analyses were performed with SPSS® 24.0 (IBM Corp. Armonk, NY). Descriptive comparisons between cardiac and non-cardiac PTSD employed the independent samples t-test, and the chi-square statistic with Fisher's exact test as appropriate. All statistical tests were two-tailed, an alpha value $p<.05$ was considered statistically significant. Since this study was exploratory, where Type II errors are more important to avoid, no adjustment was made for multiple comparisons [22].

3. RESULTS

From 73 HF patients assessed, 28 patients (38.4%) met criteria for PTSD or adjustment disorder. 10 patients meeting criteria for non-cardiac trauma were categorized as rape, sexual abuse, or intimate partner violence (n=7), witness a death or suicide (n=2), and witnessed inhumane slaughter of animals (n=1). 18 patients meeting criteria for cardiac-induced trauma were categorized according to HF-hospitalization (n=12), myocardial infarction (n=3), sudden cardiac arrest (n=2), and coronary artery bypass grafting (n=1).

There were no significant demographic differences between those with cardiac and non-cardiac PTSD (Table 1), nor differences in HF etiology, phenotype, or severity. Time since PTSD onset was significantly longer for those with non-cardiac PTSD. Other differences in psychiatric presentation included that the cardiac-PTSD patients were less likely to endorse the panic attack screening question during routine screening. Among psychiatric comorbidities, alcohol and substance abuse disorders, as well as depression were more prevalent in the patients with non-cardiac PTSD (Table 2). Otherwise, there were few differences between groups with regards to mental health including comorbid anxiety disorder or personality disorder, anxiety and depression symptom severity or mental health hospital admissions over 6 months. Longer term HF-related hospital admissions were similar in the cardiac and non-cardiac PTSD patients (Table 3).

4. DISCUSSION

This short report was among the first to compare cardiac and non-cardiac PTSD patients, suggesting few demographic and medical comorbidity differences between persons with cardiac-induced PTSD and their non-cardiac induced PTSD counterparts. Among psychiatric comorbidities, alcohol and substance abuse disorders, and depression were more prevalent in patients with non-cardiac induced PTSD. Cardiac trauma itself was more common than non-cardiac trauma in this HF population.

There is a limited body of literature suggesting that those with cardiac-induced PTSD are at greater risk for subsequent adverse CVD events and outcomes [1]. The present findings, however, indicate comparable CVD outcomes and severity regardless of trauma-type, suggesting that prognosis and course of HF may be similar. Further, no differences in cardiac admissions over 6 months were observed between groups. While it remains unclear whether cardiac-onset PTSD represents a distinct subtype of PTSD [5], our findings suggest that trauma type does not portend differences in CVD outcomes and thus may not represent different HF treatment needs.

Significant differences between PTSD groups was observed with regards to psychiatric comorbidity. Non-cardiac induced PTSD patients had higher rates of drug and alcohol disorders as well as depression, although depression symptom severity did not differ. Substance use is common as self-medication for PTSD symptoms, frequently resulting in comorbid substance use disorders [23, 24] and increasing CVD risk [25]. Time since PTSD onset was significantly longer for those with non-cardiac PTSD, increasing duration of possible self-medication coping strategies and likelihood of resultant disorders. In the case of cardiac PTSD, onset likely occurs during a period where patients are engaged in cardiac rehabilitation, including education, guidance and monitoring regarding health and lifestyle factors, such as drug/alcohol use [26]. Hence, this subgroup may be less likely to self-medicate with drugs or alcohol post-PTSD onset. Further, higher prevalence of depression disorders was noted in the non-cardiac PTSD group and substance use disorders are more prevalent in those with comorbid depression and PTSD, than those with PTSD only [27]. Among risk factors for comorbid depression in PTSD are younger age [28] and childhood adversity or sexual abuse [29]. Within our sample, PTSD onset occurred at a younger age among the non-cardiac PTSD group, and a large proportion of non-cardiac traumas were sexual-abuse related. Thus, the higher rates of comorbid depression may reflect the posited childhood trauma phenotype of PTSD [30]. Therefore, although there were differences between cardiac and non-cardiac PTSD groups with regards to psychiatric comorbidity, this may not necessarily reflect a distinct cardiac-PTSD subtype and may be explained by other sample characteristics.

Also evident from this study is that a proportion of cardiac patients experience PTSD only, with an absence of depression. Many cardiovascular guidelines recommend routine screening for depression [31-34], with PTSD not typically assessed in medical care [35]. Our findings indicate that the mental health needs of PTSD patients may not be identified or addressed in routine medical care if only depression screening is in place [36]. The HF nurse initiated HF screening results indicate that 10% of non-CVD induced and 40% of CVD-induced HF patients meeting criteria for PTSD would not be identified by depression-only screening. Such patients were likely identified by HFSMP nurses through anxiety and panic-attack screening. Further, the extant literature has largely focused on depression assessment and treatment in CVD, potentially to the neglect of other mental health needs such as PTSD and anxiety disorders [37]. Thus, how to best identify and support the mental health needs of the CVD population with PTSD (without depression) needs further exploration as do preventative efforts [38].

Despite that PTSD portends increased risk for poorer cardiac outcomes [2, 3] treatments for PTSD in CVD populations are poorly described [39] and treatment trials remain scant [35]. To date, trauma-focused cognitive behavioral therapy have shown reductions in PTSD symptoms [39-41], with one study addressing the safety of in vivo exposures for this population [39]. However, best practice in the treatment of PTSD in CVD populations and the suitability of existing treatments remains underdeveloped, with ongoing exploration needed to address cardiovascular response and safety [39, 42]. Further, whether a cardiac-induced PTSD subtype is supported by the evidence and represents differential PTSD treatment needs requires clarification. Considering that cardiac-related trauma was more common than non-cardiac trauma, our findings underscore the need for further clinical research in these populations.

This paper also extends understanding around PTSD in cardiac populations by highlighting the demographic, cardiac and psychiatric profile of HF patients with PTSD. The majority of extant literature has examined ACS, with course and outcomes of PTSD in other CVD presentations lesser reported. As many as 1 in 8 ACS patients are reported to develop PTSD [43], the present findings also highlight the presence of cardiac-related PTSD in a HF population with mixed CVD comorbidities. The CVD-induced PTSD group here had a range of precipitant factors including hospitalization, surgery, and sudden cardiac arrest. The findings here raise the possibility that hospitalization itself may be the triggering traumatic event for these patients and that the CVD cause is less important [44, 45].

4.1 Strengths and limitations

Strengths of this study include structured diagnostic interview, use of medical records for physical and cardiac health, and blinded assessment of HF etiology by qualified CVD practitioners. This study is presented with several limitations that limit the generalizability of this study. Chiefly, no comparison was made on PTSD severity between cardiac and non-cardiac induced PTSD. Though PTSD symptoms were quantified for therapeutic purposes, these data were unavailable for all patients and did not form the core battery of tests administered to patients in the initial HFSMP screen and psychiatric assessment. A related point is that identification of PTSD and related treatment needs is likely incomplete. Because screening for depression and anxiety any resultant identification of PTSD is contingent on patients attending HF appointments, highly avoidant patients with CVD-related trauma may not have attended appointments or participated in the HFSMP. Additionally, longer-term follow-up to ascertain cardiac outcomes and major adverse cardiac events beyond 6 months was not undertaken. Moreover, the sample was restricted to persons assessed for potential psychological treatment. It is possible that purely epidemiological studies in CVD patients, not associated with a mental health care component, would arrive at different findings related to CVD trauma sub-type and differences from non-CVD patients meeting criteria for

PTSD. A related point concerns the population referred for psychological treatment from 1 region in metropolitan Adelaide, which precludes generalization to other healthcare populations or diverse ethnic regions. Moreover, the size of the sample likely results in low statistical power to detect small to moderate differences, especially with regards to major adverse events and hospitalizations which require large samples [46].

5. CONCLUSION

The present study identified minimal differences between cardiac and non-cardiac induced PTSD, especially with regards to CVD phenotype, severity or course over 6 months; suggesting little difference in CVD treatment needs regardless of PTSD trauma type. Differences in psychiatric comorbidities may be explained by the childhood trauma phenotype, and timing of PTSD onset. Ongoing research is needed to further establish if cardiac-induced PTSD truly reflects a unique subtype of PTSD, and whether there are different treatment needs for this subtype.

Current & Future Developments

Cardiac-induced PTSD was recently proposed as a specific subtype of PTSD, posited to follow exposure to major cardiac events or cardiac diagnosis in some individuals. The ways in which PTSD symptoms and treatment needs differ between persons with and without cardiac trauma remain uncertain, questioning the need for a distinct subtype. Further research is required to validate whether cardiac-induced PTSD is a distinct subtype from non-cardiac PTSD. Likewise, further research is required to compare cardiac-induced PTSD with other types of medical trauma or major illness such as cancer.

Availability of Data and Materials

Because of the sensitive nature of the data collected for this study, requests to access the dataset from qualified researchers trained in human subject confidentiality protocols may be sent to Central Adelaide Local Health Network Human Research Ethics Committee at Health.CALHNResearchEthics@sa.gov.au.

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Table 1: Demographic, cardiac and biomedical characteristics of heart failure patients grouped according to cardiac and non-cardiac PTSD

Descriptive characteristic	Non-cardiac trauma N = 10	Cardiac trauma N = 18	P
Age in years M ± SD	54.3 ± 12.5	61.5 ± 16.7	0.24
Female sex	7 (70.0)	8 (44.4)	0.25
Live alone	3 (30.0)	5 (27.8)	0.99
Disability pension	3 (30.0)	3 (16.7)	0.63
Employed	1 (10.0)	3 (16.7)	0.99
Tobacco smoking	4 (40.0)	8 (44.4)	0.99
Median age of HF onset, years (IQR)	0.7 (0.5 – 0.9)	0.7 (0.3 – 0.7)	0.99
HF aetiology			0.99
Ischemic	7 (70.0)	10 (55.6)	
Valvular	-	2 (11.1)	
Viral	1 (10.0)	2 (11.1)	
Peripartum	-	1 (5.6)	
Alcohol	-	1 (5.6)	
Idiopathic	2 (20.0)	2 (11.1)	
HF phenotype			0.51
Pulmonary edema	1 (10.0)	4 (22.2)	
Cardiogenic shock	2 (20.0)	1 (5.6)	
Decompensated HF	5 (50.0)	7 (38.9)	
Hypertensive	2 (20.0)	2 (11.1)	
Right HF	-	2 (11.1)	
ACS-HF	-	2 (11.1)	
NYHA class			0.76
I	-	-	
II	3 (30.0)	7 (38.9)	
III	6 (60.0)	9 (50.0)	
IV	1 (10.0)	2 (11.1)	
Left ventricular ejection fraction % M ± SD	31.2 ± 12.0	34.3 ± 11.3	0.51
Previous myocardial infarction	6 (60.0)	5 (27.8)	0.125
Previous CABG or PCI	2 (20.0)	4 (22.2)	0.99
Atrial arrhythmia	3 (30.0)	6 (33.3)	0.99
Ventricular arrhythmia	1 (10.0)	1 (5.6)	0.99
Sudden cardiac arrest	1 (10.0)	2 (11.1)	0.99
Implantable cardioverter defibrillator	4 (40.0)	5 (27.8)	0.68
Pacemaker	1 (10.0)	2 (11.1)	0.99
Valvular regurgitation	-	2 (11.1)	0.52
Hypertension	5 (50.0)	12 (66.7)	0.44
Peripheral vascular disease	-	1 (5.6)	0.99
Chronic kidney disease	4 (40.0)	7 (38.9)	0.99
Diabetes	3 (30.0)	10 (55.6)	0.25
Lung disease	4 (40.0)	5 (27.8)	0.68
Cerebrovascular disease	1 (10.0)	4 (22.2)	0.63
Sleep apnea	1 (10.0)	3 (16.7)	0.99

Data presented as N (%) unless otherwise noted.
ACS, acute coronary syndrome; CABG, coronary artery bypass graft; HF, heart failure; IQR, interquartile range; NYHA, New York Heart Association heart failure class; LOS, length of stay; PCI, percutaneous coronary intervention;

Table 2: Psychiatric characteristics of heart failure patients grouped according to cardiac and non-cardiac PTSD

	Non-cardiac trauma N = 10	Cardiac trauma N = 18	P
Results from routine screening			
Depression, PHQ-9 ≥10	9 (90.0)	11 (61.1)	0.19
Anxiety, GAD-7 ≥ 7	9 (90.0)	11 (61.1)	0.19
Panic attack symptoms (yes)	9 (90.0)	6 (33.3)	0.006
Psychiatric interview and assessment			
Median PTSD duration in years, IQR	7 (2-34)	1 (0-1)	<0.001
Depression disorder[1]	10 (100.0)	6 (33.3)	0.001
Anxiety disorder[2]	9 (90.0)	13 (27.2)	0.28
Alcohol abuse/dependence	6 (60.0)	2 (11.1)	0.011
Substance abuse/dependence	4 (40.0)	1 (5.6)	0.041
Personality disorder	5 (50.0)	2 (11.1)	0.063
Depression symptoms (PHQ-9) M ± SD	15.1 ± 7.0	12.2 ± 8.1	0.35
Anxiety symptoms (GAD-7) M ± SD	13.8 ± 5.9	11.1 ± 7.6	0.34

1. Depression inclusive of major depression, dysthymia, bi-polar; 2. Anxiety disorder inclusive of generalized anxiety disorder, panic disorder, agoraphobia, social anxiety disorder, obsessive compulsive disorder (excluding post-traumatic stress disorder and adjustment disorder). GAD-7, Generalized Anxiety Disorder-7 items; IQR, inter-quartile range; PHQ-9, Patient Health Questionnaire-9 items; PTSD, post-traumatic stress disorder;

Table 3: Cardiac and biomedical characteristics of heart failure patients grouped according to cardiac and non-cardiac trauma

	Non-cardiac trauma N = 10	Cardiac trauma N = 18	P
Pre-psychiatric interview			
HF admissions over 6 months	1 (0-2)	2 (1-3)	0.43
HF admission LOS over 6 months	6 (0-18)	12 (1-15)	0.70
Mental health admissions over 6 months	0 (0-0)	0 (0-0)	0.99
Mental health admission LOS over 6 months	0 (0-0)	0 (0-0)	0.99
Post-psychiatric interview			
HF admissions over 6 months	0 (0-0)	0 (0-0)	0.99
HF admission LOS over 6 months	0 (0-2)	0 (0-1)	0.99
Mental health admissions over 6 months	0 (0-0)	0 (0-0)	0.52
Mental health admission LOS over 6 months	0 (0-0)	0 (0-0)	0.52

HF, heart failure; LOS, length of stay;