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The Role of Emotional Competence in Tako-tsubo Cardiomyopathy

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Conflicts of Interest and Source of Funding: none declared

Abstract

OBJECTIVE: The current study examined the role of emotional competences and depression in Tako-tsubo Cardiomyopathy (TTC).

METHODS: We compared 37 TTC patients who experienced emotion triggers (TTC-t: mean age 66.4 \pm 12.8 years, 33 women) with 37 TTC patients who did not experience emotion triggers (TTC-nt: mean age 65.8 \pm 11.1 years, 33 women) and 37 patients with Acute Myocardial Infarction who experienced an emotion trigger (AMI-t: mean age 66.1 \pm 10.1 years, 33 women). Three aspects of emotional competence (emotional intelligence, metacognitive beliefs and emotional processing deficits) were assessed using the Trait Meta-Mood Scale (TMMS), the Meta-Cognitions Questionnaire-30 (MCQ-30) and the Emotional Processing Scale (EPS). Differences between group means were evaluated using MANCOVA, adjusting for depressive symptomatology (Hamilton Rating Scale for Depression; HAM-D).

RESULTS: Compared to the TTC-nt and AMI-t comparison groups, TTC-t patients had low scores on emotional intelligence (TMMS Attention: F [2, 184] = 23.10; p < .001. TMMS Repair: F [2, 184] = 11.98; p < .001), and high scores in metacognitive beliefs and emotional processing deficits (e.g. MCQ-30 Negative Beliefs about Thoughts: F [2, 184] = 56.93; p < .001), independent of the levels of depressive symptomatology. TTC-nt patients also had significantly lower scores on the HAM-D scale compared to AMI-t (p = 0.021) and TTC-t (p = 0.004) patients.

CONCLUSIONS: TTC-t patients showed a specific dysfunctional profile of emotional competence, even after adjusting for depressive symptomatology. These results provided a better understanding of the psychological factors that contribute to Tako-tsubo Cardiomyopathy.

Key words: Depression; Emotional Competence; Emotional Intelligence; Emotional Processing; Metacognitive Beliefs; Tako-tsubo Cardiomyopathy.

List of abbreviations:

AMI: Acute Myocardial Infarction ANOVA: analysis of variance CBT: cognitive-behavioural therapy CHD: Coronary Heart Disease EPS: Emotional Processing Scale HAM-D: Hamilton Rating Scale for Depression HPA: Hypothalamic-pituitary-adrenal axis IFG: Inferior frontal gyrus MANCOVA: multivariate analysis of covariance MCQ-30: Meta-Cognitions Questionnaire-30 MCT: metacognitive therapy TMMS: Trait Meta-Mood Scale

1. Introduction

Tako-tsubo syndrome (TTC) is a cardiomiopathy characterized by a left-ventricular dysfunction that resembles Acute Myocardial Infarction (AMI; 1, 2). Technically, it has been defined as a "transient apical hypokinesis with preserved basal systolic function in the setting of unobstructed coronary arteries" (1). Prognosis was previously considered favorable (1), but a recent study showed that early and late mortality is similar to AMI patients (3).

Interestingly, the syndrome seems to be triggered by chronic or intense physical or emotional stress (e.g. grief), which influences autonomic function through the dysregulation of the hypothalamic–pituitary–adrenal axis, catecholamine release and consequently a vascular dysfunction (1, 4). Even if there is an increasing knowledge on the basic pathophysiological mechanisms of the syndrome (5), the pathogenesis of Tako-tsubo syndrome is still unclear, and needs further research on the modulations of the physiological stress response (4). Moreover, TTC patients with and without emotion triggers have been previously described in the literature (6), leaving open the question as to the existence of psychological differences among these two groups. Few studies have focused on the emotional/cognitive factors predisposing to this form of cardiac disease.

Recently, Smeijers has found lower levels of openness to experience (7) and lower emotional arousal combined with higher catecholamine responsiveness to a mental stress task (8) of Tako-tsubo patients when compared to healthy individuals. It has also been suggested that emotional suppression could be a potential vulnerability condition of TTC patients' in reactions following emotion triggers, caused by the increased sympathetic activation of the cardiovascular system (9, 10). Based on the patient's history, the authors hypothesized that the elevated levels of suppression could have contributed to: (i) the maintenance of depression after the loss of the partner; and (ii) the vulnerability to TTC mediating specific physiologic mechanisms (i.e. decreased vagal tone and increased adrenomedullary response to stressful situations; 9). Therefore, even if Scantlebury and colleagues (11) recently found that Tako-tsubo patients had similar levels of vulnerability to stress and neuroticism (i.e. tendency to experience negative affects) compared to the general population, emotions and their regulation seem to play a significant role in the development of this syndrome.

Emotional competence is a basic skill useful in several aspects of everyday life like work, education, social and intimate relationships. Emotional competence has been conceptualized as the ability to regulate, clarify and attend to feelings, be aware of, and control, our emotions, as well as express them in an appropriate manner. Noticeably, Gross (12) developed a famous model of emotion regulation, with the latter defined as a set of processes by which positive or negative emotions are themselves regulated (e.g. increasing or decreasing them). According to Gross, there are several emotion regulatory processes (e.g. situation selection or situation modification) that could be conscious or unconscious, intrinsic or extrinsic, automatic or controlled (12). Literature has shown that lack of emotional competence is strongly related to mental disorders such as depression, generalized anxiety disorder, specific phobias, posttraumatic stress disorder and obsessive-compulsive disorder (13). Interestingly, it has also been argued that emotional competence may play a role in the association between mental disorders and individual predisposition to a physical illness (e.g. cardiac disease). The link between mental disorders (in particular anxiety and depression) and the onset of cardiac diseases is well-known, and has been widely explored (14, 15). One potential explanation of this link suggests that the lack of emotional competence can act as a non-specific risk factor for the development of mental disorders, which in turn may affect physical health through biological, physiological and behavioural mechanisms.

Three core psychological constructs arise from a review of the literature as potential transdiagnostic predictors of lack of emotional competence: emotional intelligence, metacognitive beliefs and emotional processing. Emotional intelligence refers to the individual ability to process and utilize emotional information (16). One of the most widespread definitions of emotional intelligence was provided by Salovey and Mayer (17), who considered it a "subset of social intelligence that involves the ability to monitor one's own and others' feelings and emotions, to discriminate among them and to use this information to guide one's thinking and actions".

Research has found that reduced emotional intelligence is associated with an increased occurrence of Coronary Heart Disease (CHD; 18). More specifically, Vlachaki and Maridaki-Kassotaki (18) demonstrated that the occurrence of CHD is related to decreased ability to regulate emotions in the form of the ability to control temper and emotions. These findings are consistent with previous research showing that specific negative emotion regulation strategies such as anger inhibition may increase the risk of CHD, whereas the control of emotions may be a protective factor (19).

Metacognitive beliefs refer to information individuals hold about their own cognitive experiences (e.g. "Having thought/sensation/emotion X is a sign of weakness") and ways to control such experiences ("I need to suppress thought/sensation/emotion X" or "If I worry about X I will be able to understand it better") (20). Metacognitive beliefs have been linked to the development and maintenance of a wide variety of mental disorders (for a review, see 21). It is believed that such metacognitive beliefs activate unhelpful forms of coping (such as thought suppression, rumination, worry and avoidance) which exacerbate and prolong psychological distress. No research, to date, has examined the role of metacognitive beliefs in the CHD sphere. However, research in other physical conditions (such as Chronic Fatigue Syndrome, Fibromyalgia and Parkinson's Disease) has indicated that metacognitive beliefs are associated with a worsening of presenting symptoms (22-27).

The third aspect of emotional competence involves emotional processing, defined by Rachman (28) as "a process whereby emotional disturbances are absorbed and decline to the extent that other experiences and behaviour can proceed without disruption". It has been postulated that emotional processing deficits (i.e. difficulties in the processing of emotions) may include lack of awareness of emotional experiences, difficulties in identifying and labeling emotions or distinguishing them from bodily sensations (sometimes defined as alexithymia), excessive awareness of emotional feelings, the active blocking or blunting of certain emotional experiences (commonly defined as emotional suppression), or an inability to link emotional feelings with the event(s) that triggered them (29). Research has shown that one particular form of emotional processing deficit, emotional suppression, is associated with measures of sympathetic nervous system activation (i.e. increased systolic and diastolic blood pressure; 10, 30, 31).

However, to the best of our knowledge current literature has not yet investigated the link between the full spectrum of emotional competence (i.e. emotional intelligence, metacognitions and emotional processing) and Tako-tsubo Cardiomyopathy (TTC). In order to gain a more comprehensive understanding of the role of emotional competence in predicting TTC we undertook a study comparing TTC patients who experienced emotion triggers (TTC-t) with TTC patients who did not experience emotion triggers (TTC-nt) and Acute Myocardial Infarction patients who experienced emotion triggers (AMI-t). Levels of depressive symptomatology (measured through HAM-D) were a covariate in the analyses, to address whether the supposed group differences remained significant when adjusting for this construct. Indeed, higher levels of depressive symptomatology are common after acute infarction (32); in addition, mood disorders are one of the most important risk factors of cardiovascular disease (33). We hypothesized that comparing the other two groups, TTC-t patients would present reduced levels of emotional intelligence, higher levels of metacognitive beliefs and higher levels of emotional processing deficits, over and above the depressive symptomatology.

2. Methods

2.1 Participants

Thirty-seven TTC patients who had experienced emotion triggers (TTC-t; 4 males, 33 females; mean age, 66.4 years, SD=12.8), thirty-seven TTC patients who had not experienced emotion triggers (TTC-nt; 4 males, 33 females; mean age, 65.8 years, SD=11.1) and thirty-seven acute

myocardial infarction patients who had also experienced emotion triggers (AMI-t; 4 males, 33 females; mean age, 66.1 years, SD=10.1) were enrolled in this cross-sectional retrospective study. TTC-nt patients experienced physical triggers (e.g. e.g. acute respiratory infection) or no trigger at all.

TTC and AMI-t patients were sampled starting from a previous study of our research group (6); as reported by Compare and colleagues (6) these patients were newly diagnosed and had not previously been treated at the time of psychological screening.

TTC-t patients were admitted consecutively to hospital-based specialized cardiovascular clinics in Milan (Italy) over a 3-year period (from February 2010 to May 2013). TTC-t was diagnosed according to the Mayo Clinic diagnostic criteria (34). At admission, careful history taking identified whether significant stressful events immediately preceded the manifestation of TTC. Preliminary assessment of trigger events was carried out by cardiologists during admission to the emergency unit. Information about trigger events was, where possible, confirmed with the companion who accompanied the patient to the emergency unit. In addition, formal assessment of emotional trigger events in a period of 48 hours prior to Tako-tsubo cardiomyopathy symptom onset was carried out using the Interview for Recent Life Events (35). The Interview for Recent Life Events was administrated by two clinical psychologists 8 hours after the initial Tako-tsubo cardiomyopathy assessment by the cardiologist. The Interview for Recent Life Events specifies 64 different life events in nine areas. For each life event, detailed information was recorded in order to assess the independence from the illness and the magnitude of the perceived adversity and to compute the standardized Paykel Stress Index (6). In the same period, the first consecutive 37 TTC-nt and acute myocardial infarction patients who reported acute emotion triggers and who 1:1 matched TTC-t cases on sociodemographic variables (age, sex, marital status, and living alone condition) using propensity score matching (36), were respectively selected from a larger population of more than 300 acute myocardial infarction cases and 103 TTC-nt patients admitted to the emergency units. Acute myocardial infarction, hypertension, diabetes, and hypercholesterolemia were assessed according to standard criteria (37).

The study was conducted in accordance with APA ethical standards for the treatment of human experimental volunteers; each participant provided written consent in compliance with the principles of the Declaration of Helsinki (2013). The research was approved by the Ethical Committee of the Lombard Healthcare Agency (ASL) - Milan 1.

2.2 Psychological Assessment

Psychological questionnaires were administered by two expert psychologists 3 months after initial screening and without knowledge of the clinical diagnosis of the patients. Psychologists were blind to the patients' group; information on emotional stressors experienced by AMI-t and TTC-t patients are reported in a previous paper (6). Because no validated Italian versions of the questionnaires were available at the time of the study, we adapted them using a back-translation procedure. No noteworthy inconsistencies between the original and the final back-translated versions emerged.

2.2.1 Emotional intelligence

The Trait Meta-Mood Scale (TMMS; 38) is a 30-item self-report questionnaire that measures aspects of emotional intelligence. Specifically, TMMS evaluates self-regulatory domains of emotional intelligence, such as the ability to regulate, clarify and attend to feelings. It consists of three subscales: Attention to Feelings, Clarity in Discrimination of Feelings, and Mood Repair. Items are rated on a 5-point Likert scale (1 = strongly disagree; 5 = strongly agree). Higher scores indicate higher levels of emotional intelligence. The TMMS has been shown to possess good internal consistency (Cronbach's alpha range = 0.82 - 0.88) and convergent validity (38, 39).

2.2.2 Metacognitive beliefs

The Meta-Cognitions Questionnaire-30 (MCQ-30; 40) is 30-item self-report questionnaire which measures individual differences in metacognitive beliefs, judgments and monitoring tendencies. The MCQ-30 consists of 5 subscales, each composed of 6 items: (i) Positive Beliefs about Worry; (ii) Negative Beliefs about Thoughts; (iii) Cognitive Confidence; (iv) Beliefs about the Need to Control Thoughts; and (v) Cognitive Self-Consciousness. Items are rated on a 4-point Likert scale (1 = do not agree; 4 = agree very much). Higher scores indicate higher levels of metacognitive beliefs. The MCQ-30 has evidenced good internal consistency (Cronbach's alpha range = 0.72 - 0.93) and convergent validity, and acceptable to good test–retest reliability (40).

2.2.3 Emotional Processing deficits

The Emotional Processing Scale (EPS; 29) is a 38-item self-report questionnaire designed to identify emotional processing deficits (i.e. difficulties in the processing of emotions). The EPS evaluates styles of emotional experience, mechanisms controlling the experience and expression of emotions, and signs of inadequate processing. It consists of 8 subscales: Intrusion (8 items), Suppression (4 items), Lack of Attunement (5 items), Uncontrolled (4 items), Dissociation (4 items), Avoidance (3 items), Discordant (7 items) and Externalized (3 items). Higher scores indicate higher levels of dysfunctional emotional processing. The EPS possesses good psychometric properties (Cronbach's alpha = Externalized: 0.42; Avoidance: 0.66; all other subscales: > 0.70) (29).

2.2.4 Depression

The Hamilton Rating Scale for Depression (HAM-D; 41) is a 17-item clinician-rated, semi-structured interview that evaluates depressive symptomatology. Items are rated on a 5-point (0 = absent; 4 = very severe) or on a 3-point (0 = absent; 2 = frequent) Likert type scale. Higher scores indicate higher levels of depressive symptomatology: scores between 8 and 16 suggest the presence of a mild depression, while scores between 17 and 23 suggest the presence severe depression (42). HAM-D has good psychometric properties (mean Cronbach's alpha across several studies; 0.74) (43).

2.3 Statistical Analyses

Preliminary analyses evidenced that few variables were positively skewed: a log-10 or a square-root transformation corrected the non-normality. However, analyses run with or without transformed variables led to similar results: as such, we reported only results with non-transformed data for ease of interpretation (44). Finally, no data were missing.

We tested differences between groups on emotional intelligence, metacognitive beliefs and emotional processing deficits using a multivariate analysis of covariance (MANCOVA) followed by Bonferroni-corrected post-hoc tests. Depressive symptomatology (i.e. mean scores of HAM-D) was added as a covariate in the model.

Correlations between HAM-D and psychological measures were investigated using Pearson's *r* coefficients. Finally, an analysis of variance (ANOVA) was performed in order to assess differences between groups as regards levels of depressive symptomatology, followed by Bonferroni-corrected post-hoc tests. Groups were inserted as independent factors, while HAM-D was the dependent variable. Effect sizes were computed using partial η^2 and interpreted according to guidelines (.02-.12 small; .13-.26 medium; > .26 large; 45).

We controlled for the inflation of Type-I error using a Holm-Bonferroni sequential correction on the *p*-values of the univariate tests (46). All statistical analyses were performed using Statistical Package for the Social Sciences (SPSS) version 23.0. All statistical tests were two-sided; a p value $\leq .05$ was considered significant.

3. Results

Between-group differences in socio-demographic and clinical characteristics (e.g. risk factors) are reported in Table 1.

We performed a MANCOVA to assess group differences on the psychological variables, adjusting for the levels of depressive symptomatology. The multivariate tests showed a statistically significant effect of the covariate (HAM-D; Wilk's Lambda = 0.61; F(16, 92) = 3.742; p < 0.001) and of the group (Wilk's Lambda = 0.103; F(32, 184) = 12.21; p < 0.001) on the linear combination of the dependent variables. Post-hoc (see Table 2) evidenced that the TTC-t group had significantly lower scores on the MCQ-30 Cognitive Self-consciousness factor and TMMS repair factor, compared to the other two groups. No differences were found between means of TTC-nt and AMI-t groups. Moreover, the TTC-t group had significantly higher scores on the MCQ-30 Negative Beliefs about Thoughts, Cognitive Confidence and Beliefs about the Need to Control Thoughts factors. Higher scores were also observed on the EPS Uncontrolled, Avoidance and Discordant factors when compared to the TTC-nt and AMI-t groups. No differences were found between the means of the latter groups. With regards to the TMMS Attention factor, the means of each group were significantly different from each other's, with the AMI-t group having the higher scores, and TTC-t group the lower ones. The AMI-t group also had significantly higher mean scores compared to the other two groups on the TMMS Clarity factor, with no significant differences between the TTC-t and TTC-nt groups. All estimated marginal means, standard errors, F values and significance levels are reported in Table 2. Figure 1 shows the between-group differences on the total scores of the three questionnaires.

We performed Pearson's *r* correlations to investigate the relationships between levels of depressive symptomatology and the other psychological variables. HAM-D correlated significantly and positively (p < 0.05) with MCQ-30 Negative Beliefs about Thoughts, and the EPS subscales Intrusion, Suppression, Uncontrolled, Dissociation, Avoidance, Discordant and Externalized. All other correlations were not significant. Table S1 with all correlations can be found in the Supplemental Digital Content, http://links.lww.com/PSYMED/A442.

Finally, we performed an ANOVA to assess differences between groups in levels of depressive symptomatology (see Table 1 for means and SD). ANOVA revealed a statistical significant effect of the factor group, F(2) = 6.17, p = .003, partial $\eta^2 = .102$. Post-hoc analyses showed significant differences in levels of depressive symptomatology between TTC-t and TTC-nt (p = .004), and between AMI-t and TTC-nt (p = .021); all other comparisons were not significant.

4. Discussion

This study found significant differences in emotional competence between TTC patients with emotion triggers (TTC-t), TTC patients without emotion triggers (TTC-nt) and AMI-t patients, even after adjusting for depressive symptomatology. TTC-nt patients had significantly lower levels in the mean scores of Hamilton Rating Scale for Depression (HAM-D), compared to AMIt and TTC-t. In addition, TTC-t patients showed the highest level of dysfunction in emotional competence across the three domains that were investigated: emotional intelligence, metacognitive beliefs and emotional processing deficits. This population seems to be specifically characterized by lower scores in emotional intelligence, as well as higher scores on metacognitive beliefs and emotional processing deficits.

According to clinical cut-off scores of HAM-D (42), we found that TTC-nt patients experienced mild levels of depression (i.e. scores between 8-16), while TTC-t and AMI patients experienced moderate levels (i.e. scores between 17-23). Therefore, Tako-tsubo patients with emotion triggers and AMI ones had the highest scores on depressive symptomatology. To the best of our knowledge, this is the first study that compared the three groups on this dimension. Previous findings showed that higher levels of depressive symptomatology are common after acute infarction (32). In addition, Tako-tsubo patients have a high prevalence of depression before the onset of the syndrome (47, 48).

With regards to emotional intelligence, we found that TTC-t patients showed specific deficits in giving attention to their own feelings, as well as concerning clarity in the discrimination of experienced feelings, and beliefs about terminating negative mood state or prolonging positive ones. Indeed, they declared to: (a) not pay a lot of attention to how they feel; (b) not be aware of their specific feelings on a matter and to not be able to tell how they feel; and (c) not be able to try to think to pleasant things when facing stressful events or have negative feelings.

In terms of metacognitive beliefs, the three established 'markers' of a dysfunctional metacognitive stance (Negative Beliefs about Thoughts, Cognitive Confidence and Beliefs about the Need to Control Thoughts) were found to be significantly higher in the TTC-t group. These

findings align themselves to those observed in many other studies on the role of metacognitive beliefs in both physical and mental ill health (see 21, for a review). It is plausible to assume that individuals who hold beliefs that their thoughts and inner experiences are uncontrollable (Negative Beliefs about Thoughts) and that thoughts need to be controlled (Beliefs about the Need to Control Thoughts) will engage in maladaptive coping strategies (e.g. perseverative thinking patterns, avoidance and thought suppression, threat monitoring). These strategies could lead to cycles of greater accessibility of threat concepts in processing and an escalation of stress responses and negative emotion, as well as taxing cognitive resources (20). This in turn may be associated to a tendency not to pay attention to what they feel (a facet of emotional intelligence) in order to avoid triggers for negative thoughts or worry they perceived as uncontrollable. Perceived levels of low Cognitive Confidence, reflecting low levels of cognitive esteem in the presence (or absence) of objective deficit, may be both a by-product of, and contributing factor to, emotional disturbance and contribute to greater difficulty in flexible and clear-headed problem-solving which is central to effective coping.

Moreover, we found that TTC-t patients have high levels of emotional processing deficits. In particular, they showed high levels of Intrusion, Avoidance, Discordance, and Sense of Uncontrollability in response to negative emotional stimuli. TTC-t patients were more likely to bottle up emotions, rarely think about feelings, present difficulties in controlling negative emotions and switching off feelings, not tolerate unpleasant emotions, and report confusing feelings. These results, in particular those concerning the high scores on suppression, confirm findings from a previous case study of Compare and colleagues (49) that showed high levels of

this emotional processing deficit in a patient with Tako-tsubo triggered by emotional stimulus (i.e. domestic argument).

To the best of our knowledge this is the first study to investigate three core components of emotional competence (i.e. emotional intelligence, metacognitive beliefs and emotional processing) in TTC patients triggered by emotion events. These results could have a significant role in increasing our understanding of this specific pathology. Lack of emotional intelligence, and the presence of both metacognitive beliefs and emotional processing deficits has been found to be strongly linked to anxiety and depression (50-52). In view of the link between emotional competence and mental distress, our results may help to shed light on previous findings that suggested a high prevalence of anxiety traits and anxiodepressive symptoms in Tako-tsubo patients (53, 54). Moreover, our findings may help to improve the knowledge about quality of life and distress in Tako-tsubo patients observed by Compare and colleagues (55).

Despite the caution required in interpreting these data it is possible to make some considerations about the potential causal mechanisms through which emotional intelligence might possibly cause, or be associated to, TTC. It is recognized that the stress response elicits changes in sympathetic-parasympathetic balance and the tone of the hypothalamic-pituitary-adrenal (HPA) axis, which might negatively and acutely affect the cardiovascular system (56). It is therefore plausible to suppose that emotional intelligence traits became "toxic" for TTC only when they are synergistically associated with metacognitive beliefs and emotional processing deficits. A recent study, investigating how the neural correlates of personality regulate emotion processing in the brain, showed that the inferior frontal gyrus (IFG) (Brodmann area 45), medial

prefrontal and right dorsolateral prefrontal area are associated with trait rumination (an emotional processing deficit) and regulates the top-down control processes increasing subcortical activation (57, 58). IFG, together with other cortical areas, has also been associated to emotional suppression traits (59). Considering that IFG is connected with areas that underlie empathy (60), it is cautiously plausible to hypothesize that marked emotional processing deficits (e.g. emotional suppression), driven by metacognitive beliefs about needing to control thinking (20), are not allowing to adaptively reason about negative emotions 'interrupting' the transition from acute adaptive to a chronic not adaptive stress response. Finally, even on the basis of findings from neural correlates of rumination (57), we can suppose that emotional processing deficits increase positive connectivity between subcortical limbic activation and the IFG forming a feedback loop heightening arousal and activation of HPA and Autonomic Nervous System. This explanation, on the potential causal mechanisms connecting an individual's personality and TTC (see Figure 2) clearly needs to be adequately investigated but throws a plausible new light on the potential causal mechanisms of TTC.

This study has some limitations. First, the small sample size could have affected the power of this study, reducing the number of significant comparisons. Second, the lack of a healthy control group limited the comparisons with normative data and the generalizability of our results. Finally, the cross-sectional design affected the possibility to make causal inferences. Future longitudinal studies could investigate, for example, if emotional competencies affect the prognosis of cardiovascular diseases, or if these psychological characteristics are a predisposing factor for the development of specific CVDs.

In conclusion, our results could be a starting point for developing a better understanding of the predisposing psychological factors in Tako-tsubo patients. Moreover our findings raise an important question: What form of psychological treatment targeting metacognitive beliefs and emotional processing deficits could be used to support TTC patients who have been triggered by emotional stimuli? We think that metacognitive therapy (MCT; 20), which focuses on modifying metacognitive beliefs and the interruption of rumination and suppression (emotional processing deficits) may be of use. Indeed, MCT has been found to be highly effective in the treatement of a variety mental disorders with data indicating that it may be more effective than traditional cognitive-behavioural therapy (CBT) protocols (61, 62).

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FIGURE CAPTIONS

Figure 1. Between group differences (TTC-t, TTC-nt and AMI) in total means of the Meta-Cognitions Questionnaire 30 (MCQ-30), Trait Meta-Mood Scale (TMMS) and Emotional Processing Scale (EPS). Error bars indicate 95% Confidence Intervals.

Figure 2: Potential causal mechanisms connecting emotional competence and TTC









Table 1. Sociodemographic and clinical characteristics of the three samples.

.	TTC-t TTC-nt		AMI-t	
Variables	(N = 37)	(N = 37)	(N = 37)	
Male, <i>n</i> (%)	4 (10.8)	4 (10.8)	4 (10.8)	
Age, mean (SD)	66.4 (12.8)	65.8 (11.1)	66.1 (10.1)	
Marital Status, <i>n</i> (%)				
Married	22 (59.5)	21 (54.1)	21 (56.8)	
Single	13 (35.1)	14 (37.8)	11 (29.7)	
Divorced	2 (5.4)	3 (8.1)	5 (13.5)	
Living alone	6 (16.2)	7 (18.9)	8 (21.6)	
Depression (HAM-D), mean (SD)	20.19 (14.4)	10.34 (9.3)	18.55 (14.5)	
Cardiac risk factors				
Smoking, <i>n</i> (%)	5 (13.5)	4 (10.8)	7 (18.9)	
Diabetes, n (%)	4 (10.8)	2 (5.4)	18 (48.6)	
History of CAD, <i>n</i> (%)	5 (13.5)	4 (10.8)	19 (51.4)	
Hypertension, n (%)	4 (10.8)	2 (5.4)	10 (27.0)	
BMI, mean (SD)	23.8 (3.3)	23.1 (2.4)	23.6 (3.1)	
Clinical Presentation				
Chest pain, n (%)	32 (86.0)	8 (1.9)	32.0 (86.0)	
EF, mean (SD)	24.0 (5.6)	26.3 (4.4)	27.0 (4.4)	
TT, mean (SD)	2.6 (3.7)	0 (0.0)	9.4 (4.1)	
CK-MB, mean (SD)	11.8 (5.9)	0 (0.0)	28.5 (6.3)	
WBCC, mean (SD)	7.3 (4.2)	4.7 (3.1)	12.4 (5.6)	
HR, mean (SD)	90.2 (5.3)	89.4 (3.4)	95.3 (2.3)	
SBP, mean (SD)	117.2 (2.1)	115.2 (2.3)	124.1 (3.8)	

Notes: TTC-t = Tako-tsubo cardiomyopathy patients with emotion triggers; TTC-nt = Tako-tsubo cardiomyopathy patients without emotion triggers; AMI-t = acute myocardial infarction patients with emotion triggers; CAD = coronary artery disease; BMI = body mass index (in kilograms per square meter); EF = ejection fraction (in percent); TT = troponin T (in nanograms per milliliter); CK-MB = Creatine kinase-MB fraction (in nanograms per milliliter);WBCC = white blood cell count (×10 3 /µL); HR = heart rate (in beats per minute); SBP = systolic blood pressure (in millimeters of mercury).

Table 2: Estimated Marginal Means, Standard Errors, F value and *p* values of univariate tests between patients with TTC-t, TTC-nt and AMI-t for all psychological characteristics. Depressive symptoms were a covariate in the analysis.

TTC-t	TTC-nt	AMI-t			
(N = 37)	(N = 37)	(N = 37)	F value	<i>p</i> value	
7.52 (0.69)	17.35 (0.7)	17.78 (0.68)	71.124	< 0.001*	a, b
21.01 (0.66)	13.5 (0.67)	11.71 (0.65)	56.932	< 0.001*	a, b
21.44 (0.55)	15.09 (0.56)	15.03 (0.54)	44.541	< 0.001*	a, b
13.22 (0.62)	13.48 (0.63)	14 (0.61)	.417	.660	
16.87 (0.88)	13.54 (0.9)	11.75 (0.88)	8.813	< 0.001*	a, b
2.68 (0.17)	3.69 (0.18)	4.3 (0.17)	23.100	< 0.001*	a, b, c
2.03 (0.18)	1.66 (0.19)	3.21 (0.18)	19.159	< 0.001*	b, c
2.47 (0.2)	3.62 (0.21)	3.75 (0.2)	11.981	< 0.001*	a, b
2.32 (0.12)	2.17 (0.12)	2.24 (0.11)	.385	.681	
2.4 (0.14)	1.82 (0.14)	2.19 (0.13)	4.297	.016	
2.4 (0.13)	2.27 (0.13)	2.18 (0.13)	.688	.505	
2.53 (0.15)	1.51 (0.15)	1.62 (0.15)	13.437	< 0.001*	a, b
1.93 (0.15)	1.52 (0.15)	1.44 (0.15)	3.208	.044	
2.49 (0.15)	1.65 (0.16)	1.89 (0.15)	7.636	.001*	a, b
2.17 (0.12)	1.51 (0.12)	1.67 (0.12)	7.567	.001*	a, b
1.39 (0.13)	1.14 (0.14)	1.07 (0.13)	1.618	.203	
	TTC-t $(N = 37)$ $7.52 (0.69)$ $21.01 (0.66)$ $21.44 (0.55)$ $13.22 (0.62)$ $16.87 (0.88)$ $2.68 (0.17)$ $2.03 (0.18)$ $2.47 (0.2)$ $2.32 (0.12)$ $2.4 (0.14)$ $2.4 (0.13)$ $2.53 (0.15)$ $1.93 (0.15)$ $2.49 (0.15)$ $2.17 (0.12)$ $1.39 (0.13)$	TTC-tTTC-nt $(N = 37)$ $(N = 37)$ 7.52 (0.69)17.35 (0.7)21.01 (0.66)13.5 (0.67)21.44 (0.55)15.09 (0.56)13.22 (0.62)13.48 (0.63)16.87 (0.88)13.54 (0.9)2.68 (0.17)3.69 (0.18)2.03 (0.18)1.66 (0.19)2.47 (0.2)3.62 (0.21)2.32 (0.12)2.17 (0.12)2.4 (0.14)1.82 (0.14)2.53 (0.15)1.51 (0.15)1.93 (0.15)1.52 (0.15)2.49 (0.15)1.65 (0.16)2.17 (0.12)1.51 (0.12)1.39 (0.13)1.14 (0.14)	TTC-tTTC-ntAMI-t $(N = 37)$ $(N = 37)$ $(N = 37)$ 7.52 (0.69)17.35 (0.7)17.78 (0.68)21.01 (0.66)13.5 (0.67)11.71 (0.65)21.44 (0.55)15.09 (0.56)15.03 (0.54)13.22 (0.62)13.48 (0.63)14 (0.61)16.87 (0.88)13.54 (0.9)11.75 (0.88)2.68 (0.17)3.69 (0.18)4.3 (0.17)2.03 (0.18)1.66 (0.19)3.21 (0.18)2.47 (0.2)3.62 (0.21)3.75 (0.2)2.32 (0.12)2.17 (0.12)2.24 (0.11)2.4 (0.14)1.82 (0.14)2.19 (0.13)2.53 (0.15)1.51 (0.15)1.62 (0.15)1.93 (0.15)1.65 (0.16)1.89 (0.15)2.17 (0.12)1.51 (0.12)1.67 (0.12)1.39 (0.13)1.14 (0.14)1.07 (0.13)	TTC-tTTC-ntAMI-tF value $(N = 37)$ $(N = 37)$ $(N = 37)$ $(N = 37)$ 7.52 (0.69)17.35 (0.7)17.78 (0.68)71.12421.01 (0.66)13.5 (0.67)11.71 (0.65)56.93221.44 (0.55)15.09 (0.56)15.03 (0.54)44.54113.22 (0.62)13.48 (0.63)14 (0.61).41716.87 (0.88)13.54 (0.9)11.75 (0.88)8.8132.68 (0.17)3.69 (0.18)4.3 (0.17)23.1002.03 (0.18)1.66 (0.19)3.21 (0.18)19.1592.47 (0.2)3.62 (0.21)3.75 (0.2)11.9812.32 (0.12)2.17 (0.12)2.24 (0.11).3852.4 (0.14)1.82 (0.14)2.19 (0.13)4.2972.4 (0.13)2.27 (0.13)2.18 (0.13).6882.53 (0.15)1.51 (0.15)1.62 (0.15)13.4371.93 (0.15)1.65 (0.16)1.89 (0.15)7.6362.17 (0.12)1.51 (0.12)1.67 (0.12)7.5671.39 (0.13)1.14 (0.14)1.07 (0.13)1.618	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$

TTC-t: Tako-tsubo Cardiomyopathy patients with emotion triggers; TTC-nt: Tako-tsubo Cardiomyopathy patients without emotion triggers; AMI-t: acute myocardial infarction with emotion triggers; ^a p < 0.05, TTC-t vs TTC-nt; ^b p < 0.05, TTC-t vs AMI; ^c p < 0.05, TTC-nt vs AMI.

* Comparison still significant after Holm-Bonferroni correction.